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The effect of a high fat and high carbohydrate diet on the plasma lipid and lipoprotein profiles of endurance trained athletes

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Abstract

Athletes training and competing for endurance events have very high energy requirements. They are advised to eat a high carbohydrate diet, to achieve maximum glycogen stores and prolong endurance performance. Diets higher in fat could provide an easier means of consuming the high energy intakes required. They are not recommended because of the potentially adverse effects on plasma lipids and lipoproteins and because a high carbohydrate diet is considered necessary for achieving maximum glycogen stores.

I have compared the effects of diets high in carbohydrate, HC, (60-65% total energy from carbohydrate, 15-20% from fat) or relatively high in fat, HF, (35-40% energy from carbohydrate, 45-50% from fat) on plasma lipids and lipoproteins in endurance cyclists during a 3-month and extended to four month training period. Thirty-five cyclists were studied for twelve weeks and twenty-two continued the study for an additional four weeks. There were no significant differences in blood lipid and lipoprotein levels between the two diet groups throughout the study. However, plasma TC (p=0.011), LDL-C (p=0.013) and apo B (p=0.024) significantly decreased and apo A1 increased significantly (p=0.015) in both groups from baseline to week twelve. The HDL-C/LDL-C (p=0.003) and HDL-C/TC (p=0.001) ratios increased significantly in both groups over the same period. A similar reduction in TC (p=0.007) and LDL-C (p=0.008) was present to week sixteen, as well as a significant increase in HDL-C (p=0.004) and HDL3-C (p=0.009) and a significant decrease in TG (p=0.006) in both groups. An increase in physical fitness and training throughout the study is likely to account for these favourable changes in plasma lipids and lipoproteins.

In addition, there were no significant differences in physical fitness, physical performance, body weight and total body fat between the two study groups throughout the study.

The findings suggest that exercise produces favourable effects on plasma lipids and lipoproteins and this effect persists over a wide range of
dietary intakes in endurance athletes involved in a high volume of training. Proscriptive dietary advise may be unnecessary for endurance athletes and a dietary regimen of their preference, within certain limits, may be more appropriate.
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1 Introduction

Both diet and exercise can influence plasma lipid and lipoprotein concentrations. Dietary factors are known to be important in determining lipoprotein profiles, however, dietary factors alone are unable to account for the lipoprotein profiles observed in physically active individuals.

Physically active individuals have a more favourable lipoprotein profile compared to their inactive counterparts (Powell, et al. 1987; Stray-Gundersen, et al. 1991). Physically active individuals have higher HDL-C, a normal or lower total and LDL cholesterol concentration, and lower levels of plasma triglycerides (Giada, et al. 1991; Martin, et al. 1977; Tyroler 1989).

Whether, diet or exercise exerts a dominate effect on plasma lipids and lipoproteins is largely dependent on the levels of physical activity.

A low fat/high carbohydrate diet is recommended as a means to achieve optimal cardiovascular health. It is yet to be determined whether such recommendations are applicable to endurance trained athletes.

High carbohydrate diets are widely recommended for endurance athletes since they are believed to enhance physical performance. The energy expenditure involved in training and competing for endurance and ultra-endurance events can be so great that many athletes find difficulty in complying with this recommendation unless large quantities of extrinsic sugars are consumed (Rantoyannis, et al. 1989). An alternative means of satisfying the large energy needs of endurance training is through the consumption of a diet relatively high in fat. However, such a dietary regimen is not usually recommended because of the potential adverse effects of a high fat diet on total and LDL cholesterol concentrations. Also, high carbohydrate intakes are believed to be necessary to achieve maximum glycogen stores to enhance endurance performance by delaying the onset of fatigue (Ahlborg, et al. 1967; Bergström, et al. 1967; Hermansen, et al. 1967; Hultman 1967).

However, although numerous studies have shown that endurance performance is superior on a high carbohydrate diet versus a high fat diet,
most studies have been of relatively short duration and not accounted for the prolonged period required for the adaptation to a carbohydrate restricted diet. Additionally, the complex interaction between diet and exercise on lipids and lipoproteins has not been adequately addressed.

It is of interest to determine whether the positive effects of exercise can blunt the negative influences of extreme diet intakes on plasma lipoprotein concentrations.

The aim of the current research was to investigate the effects of a high fat and high carbohydrate diet on the plasma lipid and lipoprotein profiles of endurance trained athletes. In addition, it is of interest to determine how a diet high in fat will effect endurance capacity for a prolonged duration.
2 Literature Review

2.1 Coronary heart disease and blood lipids

Serum cholesterol is one of the major modifiable risk factors for cardiovascular disease (Kannel, et al. 1971; Lipids Research Clinics Program 1984; Lipids Research Clinics Program 1984b; World Health Organisation European Collaborative Group 1986).

Lifestyle modifications can profoundly alter coronary heart disease risk factors. Many large clinical trials have shown favourable changes in lipid profiles with dietary intervention (Multiple Risk Factor Intervention Trial Research Group 1990; World Health Organisation European Collaborative Group 1986).

Longitudinal studies suggest coronary artery disease risk increases as levels of total serum cholesterol reach 4.7mmol/L. Above 5.7mmol/L, the risk increases sharply. At approximately 6.7mmol/L the risk is increased almost four-fold (Campbell and Sanson-Fisher 1991).

Clinical trials of cholesterol lowering, show a continuous reduction in coronary heart disease occurs with reductions in plasma cholesterol over a wide range of concentrations (Tyrroler 1989).

Meta-analyses have shown that a 1% reduction in total serum cholesterol levels translates to a 2-3% reduction in coronary heart disease risk (Peto, et al. 1985), and that the higher the total blood cholesterol level, the greater the risk of a coronary event. This association is when elevated blood cholesterol is due to an excess of LDL cholesterol. Conversely many studies have shown a negative correlation between HDL cholesterol levels and coronary heart disease risk (Gordon, et al. 1986; Gordon, et al. 1977; Kannel, et al. 1971; Lipids Research Clinics Program 1984; Miller 1975).
2.2 Diet and blood lipids


In the Seven Countries study (Keys, et al. 1986), Keys observed a strong positive relationship between saturated fat intake and serum cholesterol levels.

In a review of studies on dietary intervention and reduction in cardiovascular risk factors or mortality, Standberg suggests that studies failing to show a reduction in risk factors induced insufficient changes diet and lifestyle. (Standberg, et al. 1991).

A variety of dietary factors affect plasma lipids. Numerous studies suggest saturated fatty acids have a causal role in coronary heart disease. Saturated fatty acids, especially lauric (C12:0), myristic (C14:0) and possibly palmitic (C16:0), increase LDL cholesterol appreciably (Fehily, et al. 1988; Keys, et al. 1965). Other dietary factors known to increase LDL-C include dietary cholesterol consumed in large amounts, and particularly in association with saturated fat. This is believed to cause some increase in LDL cholesterol (Applebaum-Bowden, et al. 1984; National Diet-Heart Study Research Group 1968; Connor, et al. 1964; Hegsted, et al. 1965).

Conversely, polyunsaturated and mono unsaturated fatty acids fail to display a hypercholesterolemic effect and may in fact be protective (Grundy 1986; Hegsted, et al. 1965; Mattson and Grundy 1985; Mensink and Katan 1987). Although polyunsaturated fatty acids are known to cause a decrease in LDL cholesterol, large intakes of these fatty acids can also reduce HDL-C levels (Jackson, et al. 1984; Kohlmeier, et al. 1985; Shepard, et al. 1978).

High carbohydrate intakes have also been shown to decrease HDL-C levels and increase plasma triglycerides (Blum, et al. 1977; Mensink and Katan 1987; Schonfeld, et al. 1976). Fibre depleted complex carbohydrates (if comprising more than 60% of total energy) have also been shown to decrease HDL-C and increase triglycerides. Large amounts of sucrose in the diet have been associated with increased triglycerides and VLDL-C, especially when combined with a high saturated fatty acid intake. However, these effects may be transient (Antonis and Bersohn 1961).

The major dietary intervention employed to reduce serum cholesterol levels is the consumption of a diet low in fat and high in carbohydrate. Much evidence exists demonstrating how a diet low in fat, particularly

Dietary guidelines have been developed for the general population to reduce the risk of CHD. These guidelines recommend the quantities and types of fat that should be consumed. For example, the American Heart Association and the National Cholesterol Education Program have recommended the following dietary guidelines to reduce the risk of coronary heart disease (National Cholesterol Education Program Expert Panel 1988):

- less than 30% of calories derived from total fat
- less than 10% of calories derived from saturated fat
- 10%-15% of calories derived from mono unsaturated fat
- up to 10% of energy derived from polyunsaturated fat

The New Zealand Taskforce targets regarding fat intake by the year 1995 are as follows (Taskforce 1991):

- Total fat intake provide 30-35% of total energy
- saturated and trans fatty acids provide no more than 15% of total energy (range 8-15%)
- Polyunsaturated fat provide approximately 8% of total energy (range 6-10%)
- For monounsaturated fat to provide up to 20% of total energy (range 10-20%).

These guidelines are based on results from general population studies. For special groups who have very high energy intakes, for example growing children and the infirm, a higher fat intake is often recommended. It is yet to be determined whether these guidelines are also applicable to endurance trained athletes who are very physically active and whose energy requirements are large. Exercise per se has been shown to have an independent effect on coronary heart disease.

2.3 Exercise and coronary heart disease

Peters et al (1983) followed 2,779 healthy men for an average of 4.8 years. Physical work capacity (PWC), assessed on a cycle ergometer, was used as a measure of physical fitness. The relative risk of myocardial infarction (MI) in those with a below median PWC was 2.2. When associated with certain other risk factors (above median cholesterol level, smoking, above median systolic blood pressure) this R.R increased. When two of these risk factors were associated with below median PWC, the R.R = 6.6. Peters et al concluded that poor physical fitness was an important risk factor for heart disease, especially when associated with other risk factors.

Early work by Morris et al (1953), showed an association between coronary heart disease and physical activity at work, in a group of bus drivers and conductors. Conductors had a lower rate of coronary heart disease compared to the drivers; with the disease also appearing at a later age in the conductors. The conductors had a substantially lower early mortality rate, and if disease was present, it was less severe. Greater physical activity of the conductors was suggested as a protective factor, safeguarding them in middle-age from some of the manifestations of coronary heart disease suffered by less active workers.

Two reviews of the literature (Powell, et al. 1987; Berlin and Colditz 1990) have shown a causal relationship between physical inactivity and coronary heart disease morbidity and mortality. Powell and associates (Powell, et al. 1987) reviewed forty-three studies describing the effects of physical activity on the morbidity and mortality from coronary heart disease. The authors concluded that “the inverse relationship between physical activity and incidence of cardiovascular disease is consistently observed, especially in the better designed studies”. They found that the relative risk (R.R) of physical inactivity was similar in magnitude to the three traditional risk factors of elevated systolic blood pressure (R.R = 2.1), hypercholesterolemia (R.R = 2.4), and cigarette smoking (R.R = 2.5). The protective effect of physical exercise was reported to be “consistently observed, strong, biologically graded, correctly sequenced and coherent with existing evidence.” No studies reported detrimental effects of exercise on coronary heart disease. Also, in an overview of physical activity and chronic disease, Powell and associates (1989) have stated that “given the high prevalence of sedentary lifestyle in developed countries, the population attributable risk is probably greater for inactivity than for any of the traditional acknowledged risk factors.”

Berlin and Colditz (1990), reached a similar conclusion to that of Powell and associates in a meta-analysis on physical activity and CHD risk. They found that the relative risk for those who were sedentary
compared to those with an active occupation was 1.9 for mortality from coronary heart disease. Again, the better designed studies demonstrated a larger benefit from physical activity than the less well designed studies.

2.3.1 Exercise threshold required to reduce coronary heart disease risk

Physical activity and fitness are cardioprotective. Precisely how much physical activity, and intensity necessary to confer a cardioprotective effect are important considerations.

Morris et al (1980) investigated the relationship between vigorous exercise in leisure time and coronary heart disease risk in a group of 17,944 middle-aged men. Vigorous exercise was defined as activities likely to reach peaks of energy expenditure of 31.5KJ/min. It was found that men engaging in vigorous physical exercise in an initial survey in 1968-1970 had an incidence of coronary heart disease that was less than half that of their colleagues who recorded no vigorous exercise. This lowered rate of coronary heart disease was present for both fatal and non-fatal events and persisted in situations where family history, smoking, obesity, short stature, hypertension and subclinical angina were present. The authors stated that 'adequate' exercise is vigorous exercise, namely, dynamic aerobic activity involving free movement of large muscle groups and above the intensity that is required to elicit a training effect.

Further research has shown that both vigorous and light physical activity can reduce cardiac event risk independently (Magnus, et al. 1979; Scragg, et al. 1987). Results from a study of Harvard alumni showed participation in both vigorous (≥10kcal/min) and less vigorous sports reduced the incidence of coronary heart disease (Paffenberger, et al. 1978). It was concluded that all regular physical activity reduces the risk of coronary heart disease, however, participation in more vigorous activities provides some added benefit. This conclusion is similar to that of Powell et al (Powell, et al. 1987) whose review demonstrated that the protective effect of physical activity was biologically graded.

The Royal College of Physicians in a summary report (Royal College of Physicians 1991) suggest a threshold of physical activity of 2000kcal per week or twice weekly vigorous aerobic activity involving peaks of energy expenditure of about 420kcal/h.

Data from the Multiple Risk Factor Intervention trials indicates that on average, a threshold one-half hour daily of even predominantly light or moderate physical activity can reduce risk of cardiovascular disease mortality (Leon and Connett 1991).
How long an individual is required to be regularly physically active, to induce the cardioprotective effect remains unclear. Notable changes in cardiovascular fitness, lipid profiles and can be identified within a matter of months. However, studies have shown that alterations in the incidence in coronary heart disease may take longer to become detectable (Brand, et al. 1979; Scragg, et al. 1987).

2.4 Effects of exercise on serum lipids

The exact mechanism of how physical exercise reduces the risk of coronary heart disease is unclear. However one of the major contributing factors seems to be the effect of exercise on plasma lipids.

2.4.1 Mode of exercise for inducing blood lipid changes.

Different exercise modalities produce different effects on blood lipids and lipoproteins.

Physical exercise can be divided into three main classes on the basis of the predominant energy source utilised. Giada (1991) has described these as follows:
1. Aerobic or endurance exercise, in which energy is produced through the aerobic glycolysis of glucose and fatty acid oxidation.
2. Anaerobic or resistive exercise in which energy is freed from transformation of glucose to lactic acid and by the splitting of phosphocreatine.
3. Mixed activities in which both anaerobic and aerobic energy systems are utilised.

Cross-sectional studies among athletes indicate that strength trained athletes have a less desirable lipid profile than those of endurance athletes (Berg, et al. 1980; Clarkson, et al. 1981; Farrell, et al. 1982; Marti, et al. 1991). Hurely (1989) concluded that in individuals who participated in resistive training for twenty weeks, no alterations in lipoprotein-lipid levels were apparent in those at high risk of coronary heart disease. Studies involving strength trained athletes can be confounded by anabolic-androgenic steroid use and diet. However, endurance-type training produces the most favourable changes in blood lipid and lipoprotein levels.

Giada and colleagues (1991) investigated the effects of specialised physical training programs on serum lipids, lipoproteins, apoproteins and lipolytic enzymes in a cross-sectional study. Four groups of men were
recruited into the study. The groups comprised of elite male athletes engaged in either aerobic (n=13), anaerobic (n=17) or mixed training (n=9) programs. An additional group (n=15) was made up of sedentary controls. It was found that in the mixed and aerobic groups serum triglycerides were significantly lower compared to sedentary controls; whereas total serum and LDL cholesterol as well as apoprotein B were slightly lower. HDL and HDL2 cholesterol were slightly higher and the LDL/HDL cholesterol ratio and the total/HDL cholesterol ratio were higher only in the aerobic group compared to the sedentary group. Extrahepatic lipoprotein lipase activity was slightly higher in the aerobic group whereas hepatic triglyceride lipase was significantly lower in the aerobic and mixed training groups. It was concluded that specialised training programs have different effects on lipoprotein patterns and lipolytic enzyme activities, only aerobic exercise having a potentially anti-atherogenic effect. The authors suggest that high energy costs of aerobic exercise and the associated increase in metabolic flux may be linked to the favourable lipoprotein-lipid profiles seen with this type of training.

2.4.2 Total cholesterol and exercise

Studies suggest no difference in plasma cholesterol concentrations between those who are physically active and sedentary individuals (Haskell, et al. 1980; Keys 1970; Keys, et al. 1986). However, it has been suggested that a trend is present towards lower total plasma cholesterol concentrations with greater physical activity (Hoffman, et al. 1967; Holloszy, et al. 1964; Stray-Gundersen, et al. 1991). This is consistent with the graded response as suggested by Powell et al (1987).

Several longitudinal studies have shown reductions in total cholesterol with endurance training (Peltonen, et al. 1981; Goodyear, et al. 1990; Lipson, et al. 1980). A meta-analysis by Tran (1985) suggests significant reductions in cholesterol are present with exercise training and that these reductions are greatest when accompanied with weight loss. Wood and Haskell (1976) have concluded that exercise is most effective at reducing total cholesterol levels when initial values are in the normal to high range. In many studies indicating lower total cholesterol levels in endurance athletes, the athletes have tended to be very physically active. Haskell et al (1988) have suggested that these athletes are usually doing training equivalent in energy expenditure to 30km/week of running suggesting a threshold level of physical activity before significant decreases in serum cholesterol is observed.
Not all studies have demonstrated a relationship between total cholesterol and physical activity (Bannister and Cameron 1990). As suggested by Haskell et al. above, it is possible that the level of physical activity in many studies has not been sufficient to show a lipid lowering effect. This dose-response relationship for endurance exercise indicates a threshold of 8-15 miles/week running or similar to elicit changes in total cholesterol (Superko 1991; Wood, et al. 1983).

Serum or plasma TC measurements include the HDL-C fraction which may be elevated in endurance trained individuals (Haskell, et al. 1988; Nikkila, et al. 1978). High levels of HDL cholesterol associated with endurance training may contribute to this elevated total cholesterol and may account for discrepancies in elevated total cholesterol concentrations in athletes. Metabolism and composition of cholesterol may differ between physically active individuals and sedentary individuals.

2.4.3 LDL cholesterol and exercise


A Meta-analysis by Tran (1985) (involving ninety-five studies) showed that even when body weight remained unchanged, LDL cholesterol levels decreased significantly with endurance training (p<0.05).

Wirth et al (1985) in a cross sectional analysis of runners and non-runners indicated that runners had significantly lower levels of LDL cholesterol (p<0.004). However density gradient ultra-centrifugation demonstrated that only the “small” LDL were reduced, with no significant difference in the “large” LDL particles. Williams et al (Williams, et al. 1986) employing resting and sub-maximal heart rates as an indication of fitness, showed a significant positive association with “small” LDL but not “large” LDL. The evidence suggests a difference in LDL-C exists between
endurance athletes and a sedentary control group. Further to this, studies suggest that this difference can be affected in a population with training, even in the absence of weight loss and overall that exercise has a positive effect in reducing LDL-C levels in endurance athletes.

2.4.4 HDL cholesterol and exercise

Compared with sedentary individuals, athletes consistently have significantly higher concentrations of serum HDL cholesterol (Haskell, et al. 1988; Nikkila, et al. 1978; Wood, et al. 1976). Haskell et al report that endurance athletes show a 20 to 30% increase in mean HDL cholesterol concentrations compared to sedentary controls (Haskell, et al. 1988).

Endurance exercise generally results in an increase in HDL cholesterol concentrations (Myhre, et al. 1981; Baker, et al. 1986; Marti, et al. 1991; Marti, et al. 1990). Over half of the longitudinal studies have indicated a significant increase in HDL cholesterol with endurance training. The magnitude of the rise in HDL cholesterol appears to be related to exercise intensity (Tran and Weltman 1985; Williams, et al. 1986). Myhre and co-workers (1981) concluded from an eight month study involving cross-country skiers, that elevated HDL cholesterol concentrations associated with high levels of physical activity, were related to both amounts and intensity of the training. Lehtonen et al (1978) found that exercise increased serum HDL cholesterol, and that there was a positive correlation between the amount of weekly exercise in kilometres and plasma HDL cholesterol concentration. Exercising more than seventy kilometres per week (running or skiing) increased HDL concentration above normal levels.

Houmard et al (1991) investigated the effects of fitness level and regional distribution of fat on carbohydrate metabolism and plasma lipids in middle- to older-aged men. Sedentary (n=16) and exercised trained (n=30) men were involved in the study. Fitness level was assessed by time to exhaustion and maximal oxygen uptake achieved during an incremental treadmill test. Physical fitness was the only significant multiple regression predictor for high density lipoprotein cholesterol. Other factors involved in the multiple regression analysis that were not significantly related to HDL-C levels were abdomen to hip ratio (used as an index for regional adiposity) and percent body fat. It was suggested that the level of physical fitness is an important determinant total HDL cholesterol levels.

HDL-C decreases with age. Exercise may blunt this age-related effect on HDL-C. An American study, Owens et al (1992) demonstrated that
women who increased their activity during a three year interval, tended to have the smallest decrease in total HDL-C and HDL2-C levels associated with age. The changes in lipids were seen to be due to activity and were largely independent of changes in body weight.

Although many investigators claim that rises in the HDL fraction are due to changes in body weight or composition, evidence shows that exercise-induced changes in HDL cholesterol occur independently of weight changes. Schwartz et al (1992) demonstrated that potentially important improvements in the lipoprotein profiles, specifically HDL cholesterol levels were found following an intensive exercise training program in both healthy young and older men. None of the lipoprotein changes were significantly related to body composition or fat distribution.

The subfractions of HDL-C, namely HDL2-C and HDL3-C are of particular interest. The HDL2-C subfraction is negatively associated with coronary atherosclerosis, whereas HDL3-C has not been shown to be related to coronary heart disease. It is the HDL2-C subfraction which appears to be elevated in endurance athletes (Williams, et al. 1982). When sedentary individuals (Rauramaa, et al. 1984) and cardiac patients (Ballantyne, et al. 1982) have undertaken endurance training, the HDL2 subfraction has increased significantly.

Although most research suggests that HDL2-C is the subfraction to increase with endurance training, there is some evidence indicating that HDL3 cholesterol is also increased (Stampfer, et al. 1991).

Endurance exercise appears to increase HDL cholesterol concentrations which is important in view in the epidemiological relationship where a 1% increase in HDL cholesterol translates to a 3% decrease in coronary risk (Manninen, et al. 1988).

2.4.5 Triglycerides, VLDL and exercise


Since in the fasting state most of the plasma triglycerides are carried in VLDL, the major plasma triglyceride effects are seen in VLDL.

It appears that a relationship exists between VLDL-TG and HDL cholesterol. Research suggests that VLDL degradation, in part, contributes
to the increase in HDL cholesterol levels associated with exercise. The degradation of triglyceride particles is largely dependent on lipoprotein lipase, where surface material is transferred to HDL. Increased lipoprotein lipase activity resulting from endurance training may explain the lower serum and VLDL triglyceride levels and higher HDL cholesterol concentrations in well-trained subjects (Nikkila, et al. 1978).

Triglyceride rich particles have an important role in atherosclerosis (Zilversmit 1979). Physical exercise reduces fasting and post-prandial triglyceride rich lipoprotein levels and thus, CHD risk. Weintraub et al (1989) conducted a study where subjects underwent physical training for a seven week period. Physical conditioning significantly increased postheparin lipoprotein lipase activity and decreased fasting triglycerides. Significantly, physical exercise reduced chylomicron levels by 37%. It was concluded that physical exercise conditioning reduced fasting and post-prandial lipoprotein levels by increasing the catabolism of triglyceride rich particles.

Cohen (1989) showed similar findings when comparing post-prandial lipemia and chylomicron clearance in endurance athletes and sedentary controls. It was concluded that chronic exercise decreases post-prandial lipemia by reducing chylomicron-triglyceride half-life. It was hypothesised that this effect was due partly to the decreased fasting serum triglyceride pool size and partly to a direct effect of exercise on the serum triglyceride removal system.

2.4.6 Apolipoprotein A1 and exercise.

Apolipoprotein A1 is the major lipoprotein involved in the metabolism of HDL cholesterol, and is involved in the activation of the enzyme lecithin-cholesterol acetyl transferase (LCAT), which mediates the esterification of free cholesterol in HDL particles. There is a negative correlation between apo A1 levels and atherogenesis (Avogaro, et al. 1980; Kukita, et al. 1984; Maciejko, et al. 1983).

Cross-sectional comparisons between endurance athletes and sedentary controls have shown an increased concentration of apolipoprotein A1 in athletes (Hoffman, et al. 1967; Wood and Haskell 1979). Levels have been reported to be 30% higher in athletes (Haskell, et al. 1988).

Endurance training increases apolipoprotein A1 levels in most studies (Haskell, et al. 1988; Kiens, et al. 1980), but this is not a consistent finding (Wood, et al. 1988). Differing exercise levels may account for some of the discrepancies in apoproteins concentrations.
Schwartz (1987), studied the effects of either aerobic training or weight loss through caloric restriction on apolipoprotein A1 levels. Exercise appeared to directly affect the proposed HDL-mediated reverse cholesterol transport system by increasing both HDL cholesterol and apolipoprotein A1 proportionally. Conversely, dietary weight loss mainly affected the transport of cholesterol and triglyceride to cells as VLDL and LDL, and was not related to the observed changes in apo A1.

2.4.7 Apolipoprotein B and exercise.

The bioform of Apolipoprotein B, Apo B100, is important in lipoprotein metabolism in humans. It is produced in the liver and is required for the synthesis and secretion of VLDL. Apolipoprotein B100 is also virtually the only protein component of LDL. Elevated levels of apolipoprotein B in the plasma is a recognised risk factor for coronary artery disease (Consensus Conference 1985).

Inadequate data is available regarding apolipoprotein B and its relationship with endurance exercise. Several investigators have shown that apolipoprotein B is negatively related to endurance exercise (Wood, et al. 1983). Hoffman et al (1967) demonstrated lower B-lipoprotein levels in high exercising individuals compared to those who exercised less routinely.

A longitudinal study performed by Baker et al (1986) investigated the effects of twenty weeks of aerobic training on lipoprotein profiles of previously untrained men, and found that physical training resulted in a significant reduction (18.7%) in LDL-protein. The authors concluded that "the lowering effect of exercise on LDL-protein probably represents predominantly a reduction in apo B.

2.5 Diet, exercise and serum lipids

Although numerous studies have investigated the relationship between exercise and lipoprotein metabolism, the role of diet in this relationship has largely been overlooked.

The relationship between consumption of diets high in fat, particularly saturated fat, elevated serum cholesterol levels and the subsequent increased risk of coronary heart disease and atherogenesis is well-established. Few studies have adequately investigated this epidemiological relationship in very physically active individuals.
One of the major dilemmas in advocating a diet high in carbohydrate to reduce the risk of CHD is the apparent decrement in HDL-cholesterol levels and the increase in triglyceride concentrations (Blum, et al. 1977; Thompson, et al. 1984b). When carbohydrate is the primary source of energy in the diet, the liver synthesises lipids which are exported into the blood as VLDL. These particles undergo further degradation to LDL cholesterol. Several studies have demonstrated that the expected fall in HDL-C with the consumption of a high carbohydrate diet is countered by increased physical activity (Faber, et al. 1992; Thompson, et al. 1984b).

2.5.1 Descriptive and cross-sectional studies

Many cross-sectional studies have investigated the diets of athletes and sedentary controls in an attempt to determine the relative contributions of diet and exercise to the lipid/lipoprotein profiles of people involved in regular physical exercise.

Blair et al (1981) investigated the nutrient intakes of middle-aged men and women runners compared to controls in association with lipid and lipoprotein profiles. Three-day food diaries were completed by athletes and compared with twenty-four hour recalls completed by the controls (the use of differing nutritional assessments may effect the validity of the study as the 24-hour recall is known to under-estimate actual intake). Runners had significantly higher energy intakes (2959kcal/d vs 2361kcal/d for men; 2386kcal/d vs 1871kcal/d for women) (p<0.001) and were reported to eat more total fat and carbohydrate (but when expressed as percent of total E, no significant differences in carbohydrate and fat intake were present). Total cholesterol levels were significantly lower in female runners than controls (p≤0.002), and marginally, but not significantly so in male runners (p≤0.06). Triglycerides were considerably lower in runners than controls (p≤0.001), and HDL cholesterol was higher in runners. LDL-cholesterol was lower in runners compared to controls. When the diets were expressed as per 1000kcal, saturated fat, polyunsaturated fat, and dietary cholesterol were no different between the exercise and the control groups. The authors concluded that nutrient intake seemed unlikely to account for the observed differences in plasma lipids and lipoproteins between the control group and the runners. Therefore the differences in plasma lipoproteins were due to some other factor; possibly exercise.

Hagan et al (1983) investigated the effects of diet and exercise on lipids and lipoproteins in forty-five male runners over six months. Training involved 183.4 ± 166.1km per month of running. Each subject completed one food frequency questionnaire per month. Dietary components were
not important in the prediction of lipoprotein levels. Multiple regression analysis indicated that BMI was the best predictor of triglycerides, total and LDL cholesterol. While distance run was the best predictor of HDL and HDL/total cholesterol ratio, and was second best as a predictor of triglycerides. Some of these results contradict those of Schwartz et al (1992) who found that lipoprotein changes were not significantly related to body composition or fat distribution.

Some investigators have shown the importance of the influence of exercise in the diet/exercise interaction on HDL cholesterol. Faber et al (1992) studied eleven males participating in a hiking expedition over a period of six weeks, where the average distance covered was 15km/day. Subjects completed seven day food diaries prior to, and during the hiking expedition. It was evident that fat and carbohydrate intake changed from 36.9% and 40.6% to 14.0% and 76.4% respectively. HDL cholesterol did not change during the expedition, although the HDL to total cholesterol ratio increased significantly. It was concluded that the expected fall in HDL cholesterol due to the consumption of a high carbohydrate diet was counteracted by the increase in physical activity and weight loss. Schwartz et al (1992) did not consider body weight and composition an important determinant of HDL-C.

Moore et al (1983) studied the relationship of exercise and diet on high density lipoprotein cholesterol levels in women. This relationship was investigated in forty-five long distance runners, forty-nine joggers, and forty-seven inactive women. HDL-cholesterol levels in the long distance runners were significantly higher compared to the joggers and inactive women. Multiple regression analyses indicated that alterations in lipoprotein levels could not be attributed to differences in nutrient intake. It was found that distance run and percentage body fat were the strongest predictors of HDL cholesterol in women. Even when results were adjusted for percent body fat, significant differences remained between exercise groups and HDL cholesterol. There was a graded response between HDL-C and amount of exercise. HDL-C levels in women who ran on average 19.6km/wk were 8mg/dl (12.9%) higher than in their inactive counterparts, and in those averaging 49.9km/wk, HDL-C levels were even higher. This graded response was also present in males.

Thompson et al (1983) looked at exercise, diet and physical characteristics as determinants of HDL-C levels in endurance athletes. Serum lipids, lipoproteins, apolipoproteins, physical characteristics, and ten-day dietary records of twenty male distance runners were compared with those of fourteen controls. The runners had significantly higher levels of HDL cholesterol and apolipoprotein A1. Runners consumed 20% more
calories than the sedentary men and the additional calories consumed were derived from largely carbohydrate. Runners consumed 413g/day of carbohydrate compared to sedentary men who consumed 294g/day. It was evident that the consumption of high absolute quantities of carbohydrate did not result in a depression of HDL-C levels in lean individuals involved in exercise training. The authors concluded that the high energy intakes associated with physical activity may in part be responsible for the higher HDL cholesterol concentrations in endurance athletes. The authors suggest that dietary factors and the associated increase in caloric intake may be as important as exercise itself in producing the lipoprotein pattern characteristic of endurance athletes.

It is interesting to note that Tarahumara Indians of Mexico renown for their high levels of physical activity, have low HDL levels (26 ± 7mg/dl) (Connor, et al. 1978) It has been suggested that the high carbohydrate content (75% of calories) and lack of fat (12% of calories) in their diets may account for this phenomenon (Thompson, et al. 1983). Also genetic factors may play a more important role in the determination of HDL concentrations in this particular group of people. Conversely levels of physical activity in this group may not be as great as that seen in highly trained endurance athletes.

2.5.2 Intervention and experimental studies

Experimental studies have provided further insight into the interaction of diet and exercise on lipid and lipoprotein metabolism.

A study in the mid 1950's, looking at the relationship between diet, energy expenditure and serum cholesterol, concluded that young men initially consuming high fat diets (50% of energy from fat) were able to double their caloric supply (consuming a diet of 25% of energy from fat) without changing the levels of their serum lipids, as long as the excess energy was dissipated as exercise (Mann, et al. 1955). A limitation of this study however was the small number of subjects, namely three.

In a recent study, cross-country skiers were fed four different diets whilst participating in heavy cross-country mountain trips (Ekstedt, et al. 1991). Three of the diets provided of 3800kcal/d and differed in the fat content; 26%, 52% and 29% of energy as fat. Dietary cholesterol contributions were 260mg, 480mg and 410mg respectively. The fourth diet was a low energy diet of 2300kcal/d and consisted of 26% of energy from fat and 110mg of cholesterol per day. A significant decrease in the VLDL-LDL fraction was evident in all groups. Triglycerides decreased by 30%, with no significant differences between the diet groups. Only in the
high fat group was there an observed increase in HDL cholesterol and an increase in the HDL/total cholesterol ratio from 0.327 to 0.49. It was concluded that vigorous physical activity caused decreases in VLDL and LDL substantially, irrespective of diet used. But the changes in the HDL fraction seemed more dependent on diet. This study did have its limitations, namely the small sample size (n=7) and the duration of dietary change (eight days).

The duration of dietary change may prove crucial in the investigation of serum lipid changes in trained athletes. Most previous studies have employed intervention periods ranging from four to six weeks (Phinney, et al. 1983b; Phinney, et al. 1983a; Phinney, et al. 1980). However, it has been suggested that true metabolic adaptation to a high fat diet may take in excess of twenty (Hammel, et al. 1977).

Thompson et al (1984b) studied the effects of a high carbohydrate diet and a high fat diet on the serum lipids and lipoproteins concentrations in runners who were actively training, averaging 16km/day. Prior to intervention, subjects consumed a diet of 15% protein, 32% fat and 53% carbohydrate. During a fourteen day intervention period, subjects consumed a diet comprising of 69% of energy from either carbohydrate or fat. A control group remained on the pre-study diet. It was evident that a diet of 69% fat, 111g of which was from saturated fat, had little effect on total and LDL cholesterol, and resulted in a 10-20% decrease in triglycerides. The high carbohydrate regimen resulted in a significant drop of 9% in HDL levels. This decrement, however, is lower than that observed in normal sedentary subjects, (Blum, et al. 1977; Falko, et al. 1980; Gonen, et al. 1981; Oster, et al. 1981; Schonfeld, et al. 1976), who have reported reductions in HDL-C of around 23% following the consumption of high carbohydrate diets. It was suggested by Thompson et al that although in athletes the changes in HDL were qualitatively similar to those of sedentary subjects, the athletes response to a high carbohydrate diet may well be blunted. In subjects consuming 69% of energy as fat, it was evident that HDL levels did not change greatly. However, on the last day of the fourteen day study, HDL cholesterol and apolipoprotein A1 levels were above initial values. The duration of dietary intervention was only fourteen days which may not have allowed for true adaptation to the diets. The authors concluded that the plasma lipoproteins of endurance runners are susceptible to changes in diet. However, diet alone was unlikely to account for the athletes high HDL levels. Exercise possibly limited diet-induced changes to lipoproteins.

Lukaski et al (1984) suggest that dietary factors exert a dominant effect over exercise with regards to lipoproteins. They investigated the influence...
of type and amount of dietary lipid on plasma lipid concentrations in endurance trained athletes. Three endurance trained male cyclists consumed isonenergetic diets of which carbohydrate, or fat (either predominantly polyunsaturated or saturated fat) contributed 50% of daily energy intake. The diets were consumed for periods of twenty-eight days. Maximal aerobic power was maintained at 62ml/min/kg and body weights were held within 3% of admission levels. Results showed that the polyunsaturated diet significantly (p<0.05) reduced mean fasting plasma total cholesterol in comparison to the saturated fat and carbohydrate diets (160 versus 254 and 243mg/dl, respectively). Likewise, the polyunsaturated fat diet reduced mean plasma triglycerides (p<0.05) relative to the saturated fat and carbohydrate diets (37 versus 62 and 79 mg/dl, respectively). No significant dietary effects were evident with regards to HDL cholesterol. It was evident that a potentially atherogenic diet resulted in rises in both LDL and HDL cholesterol. Whereas the high carbohydrate diet and a diet with a high P:S ratio, both of which are considered anti-atherogenic, lowered both LDL and HDL cholesterol. It was concluded by the authors that under controlled conditions where physical exercise was constant, dietary lipid differences influence fasting serum lipid and lipoprotein concentrations among men with high energy expenditures. In addition to this it was stated that the Keys’ equation 

\[
\Delta \text{Cholesterol} = 1.35 (2\Delta S - \Delta P) + 1.5 \Delta Z
\]

where, \(\Delta \text{Cholesterol}\) is the expected change in total plasma cholesterol, \(\Delta S\) and \(\Delta P\) are changes in the percentages of saturated and polyunsaturated fatty acids in the diet respectively, \(\Delta Z\) is the change in dietary cholesterol intake (mg/1000kcal) provides useful predictions of changes in plasma total cholesterol among men undertaking vigorous exercise and consuming different types and amounts of dietary lipid. The investigators concluded that the usefulness of the Key’s equation can be extended to encompass physically active men. This is despite the fact that subjects in the study were vigorously active and consuming almost twice the energy requirements as the population from whom the equation was developed. This research suggests that diet is a more powerful predictor of serum lipids and lipoproteins than is exercise in a group of trained individuals. However the small sample size of three males largely invalidates both the results and the assertion that the Keys’ equation can be extrapolated to include physically active men.

Griffin et al (1988) presented similar conclusions to that of Lukaski et al. Six physically fit men were observed during walks of 37km on 4 consecutive days, while consuming high-carbohydrate (85% of total energy derived from carbohydrate) and high fat diets (75% of total energy
derived from fat). The high carbohydrate diet elicited a significant increase in VLDL cholesterol and a significant decrease in HDL cholesterol. Conversely, the high fat diet resulted in a significant decrease in VLDL cholesterol and an equally significant increase in HDL cholesterol. However, because of the small sample size and duration of intervention, caution should be practiced when determining the reliability of such results.

Kiens et al (1981) presented results opposing those of Lukaski and Griffin. Kiens et al investigated the influence of alterations in fat intake on lipoproteins levels of twenty-three regularly active men (exercising for an average of fifty minutes per day). Thirteen subjects remained in a control group while ten participants comprised an experimental group. The latter group firstly consumed a ‘fat-rich’ (54% energy derived from fat, 29% of energy derived from carbohydrate) for four weeks and then a ‘fat-poor’ (29% of energy derived from fat, 53% of energy derived from carbohydrate) diet for four weeks. Surprisingly, such large dietary differences resulted in negligible changes in total, LDL, and HDL cholesterol. The only differences between the two diets was in apo A1 and AII which were 8% and 3% lower after the fat-poor diet compared to the fat-rich diet. Diet was not a major factor determining lipoprotein levels in this group of men. The design of this study makes it susceptible to an order effect.

Blum and associates reported that consuming a diet comprising 80% of it’s calories from carbohydrate, caused an increase in the catalytic rate of HDL apoproteins by 39% in several subjects (Blum, et al. 1977). Thompson and co-workers (1984b) found no changes in HDL apolipoproteins with the consumption of a high carbohydrate diet. Other investigators, however, have reported decreases in apolipoprotein A1 and AII (Cohen, et al. 1991; Schonfeld, et al. 1976).

The literature shows that diet cannot account for all the changes in lipids/lipoproteins, but due to limitations in study design, in particular intervention duration, order effect and small sample size, no significant conclusions can be drawn from the data at present.

2.5.3 Animal research

Animal research has provided further data in controlled environments on the effects of diet and exercise on plasma lipids/lipoproteins.

Kramasch et al (1981) investigated the effects of moderate conditioning exercise in monkeys fed an atherogenic diet. In the monkeys fed an atherogenic diet, those who underwent exercise conditioning had lower
levels of plasma triglycerides, LDL and VLDL cholesterol and significantly higher levels of HDL cholesterol compared to the non-exercising monkeys. In fact, exercise was associated with substantially reduced atherogenic involvement, lesion size, and collagen accumulation. In addition, larger hearts and wider coronary arteries were observed in the exercised monkeys, decreasing the degree of luminal narrowing. The authors concluded that moderate exercise may prevent or retard coronary heart disease in primates.

Exercise has been shown to reduce slightly, the degree of induced atherosclerosis in other animal models including geese, swine and rabbits (Gollnick, et al. 1985; Gollnick and Saltin 1982; Kukita, et al. 1984; Parry-Billings, et al. 1990)

In many species the major lipoprotein is not LDL as in man but is in fact HDL, and therefore animal studies must be viewed with caution and may not be directly applicable to man.

2.6 Fuel utilisation and Exercise

For centuries athletes and physiologists have investigated fuel utilisation during exercise in an attempt to discover the ideal diet for optimal physical performance. Numerous studies have been conducted over the last 150 years to determine the utilisation of the major macronutrients during exercise. The results from these studies have presented conflicting conclusions.

Early work by Von Liebig suggested that protein was the main energy source; whereas in 1896, Chauveau (1896) claimed that carbohydrate was the only fuel able to be oxidised by the working muscle. This concept was supported by many physiologists. Work from the Zank laboratory from 1896-1901, challenged this theory, claiming that both carbohydrate and fat could be utilised by the muscle. Additional evidence to support this new hypothesis eventuated from work on the respiratory exchange ratio (RER). Furthermore, Fritz and co-workers (1958), through experimentation on stimulated isolated muscle, demonstrated conclusively that muscle took up and oxidised fatty acids in the exercising condition.

Despite the fact that both carbohydrate and fat have been shown to be important fuels for endurance exercise, most attention has focused on carbohydrate as the preferred fuel in endurance exercise.
2.6.1 Carbohydrate as a fuel for endurance exercise

Over the past two to three decades, dietary carbohydrate and the relationship between dietary carbohydrate and muscle glycogen stores, has received most attention in the scientific literature. As a result of this research, athletes are recommended high carbohydrate diets and various carbohydrate loading regimes for endurance events.

Interest in the role of carbohydrate as a fuel for endurance exercise gained momentum with the work of Christensen and Hansen in the 1930s (Christensen and Hansen 1938). The investigators perfected the respiratory quotient (RQ) as a tool to determine fuel utilisation during physical exercise. RQ and endurance times were measured for three subjects following the consumption of a mixed diet, a high fat diet and a diet high in carbohydrate. Following the high fat diet, the respiratory exchange ratio (RER) was lower and the total work endurance time was 70% of that after following a mixed diet. Conversely, following the high carbohydrate diet, the RER was higher and total work endurance was approximately twice that of the mixed diet condition. This now classical study, however, has been criticised. The small subject number (three males) and the short duration for adaptation to the study diets (three days to one week) are the major criticisms. Some investigators have suggested that adaptation to a high fat diet may take in excess of six to twenty weeks (Phinney, et al. 1980; Hammel, et al. 1977).

In addition, the macronutrient composition of the study diets in the study of Christensen and Hansen (95% of calories derived from carbohydrate in the high carbohydrate regimen and 5% of calories derived from carbohydrate in the high fat diet) were extreme and impractical long-term.

Three decades on from Christensen and Hansen, Ahlborg et al (1967) perfected the needle biopsy technique to measure muscle glycogen stores in relation to physical exercise under different dietary conditions. They found a positive correlation between performance time and the initial glycogen content. Ahlborg concluded that “all these correlations indicate that local glycogen stores in the working muscle is a determining factor for the ability to perform long-term exercise ie. the higher the muscle glycogen content the longer the performance time.” The study subjects however were untrained and were not metabolically adapted to exercise conditions.

Hermansen et al (1967), studied muscle glycogen utilisation at high relative workloads, and concluded that primarily the glycogen stores in the exercising muscle will determine the capacity for prolonged work.
Bergström and colleagues (1967) manipulated the glycogen content of the muscle using differing diets and exercise regimes. They demonstrated that times to exhaustion for exercising subjects were 114, 57 and 167 minutes following the mixed diet, high fat-high protein diet, and high carbohydrate diet respectively. This led the authors to conclude that “the individuals ability to sustain prolonged exercise is highly dependent on the glycogen content of the muscle which in turn is dependent on the type of diet before exercise.” However, as with Christensen and Hansen this research has its short-comings in allowing only three days for adaptation to the particular diets.

In the classic review by Hultman (1967) the author states “we can conclude that the limiting factor in the performance of long term heavy muscular work is the preformed glycogen store in the working muscle.” This conclusion, as well as those reached by Hermansen, Bergström and other investigators have been universally accepted, forming the basis of the common “carbohydrate loading” practice used by endurance athletes world-wide to improve performance.

It must be noted that carbohydrate provides only a limited amount of fuel in the form of glycogen in the liver and the muscle. For example, in a 70kg non-obese man, stored carbohydrate can provide 2000kcal. Conversely, fat, the major storage fuel within the human body, comprising 80-85% of stored fuel, provides some 140,000kcal.

Evidence also exists indicating that a normal carbohydrate-rich diet is not sufficient to replenish glycogen stores within twenty-four hours following exhaustive exercise. Brouns et al conducted a series of controlled Tour de France simulation studies using highly trained cyclists (Brouns, et al. 1989b; Brouns, et al. 1989a; Saris, et al. 1989). They found a carbohydrate-rich diet, available ad libitum, was insufficient to fully restore glycogen within twenty-four hours following exhaustive cycling. Subjects also developed a negative energy balance (-9MJ/day) with the consumption of a normal high carbohydrate diet.

Thus, doubt arises about the efficacy of a high carbohydrate diet in situations where energy expenditure is large and time for recovery is limited.

2.6.2 Fat as a Fuel for endurance exercise

Fat has several characteristics which in some situations make it the preferred fuel. Firstly, an important property of fat is that carbon and hydrogen comprise about 90% of the free fatty acid molecule. In comparison this figure is only about 50% for the carbohydrate molecule.
This is important because the oxidation of the carbon and hydrogen of the molecule is responsible for energy release. Thus the total energy stored per unit of weight of fat is nearly twice that of carbohydrate. A further advantage of fat as a substrate is that it is not stored in an hydrated form in the body, as is carbohydrate. The extra water stored with glycogen can result in a feeling of heaviness and stiffness may become apparent due to deposition of water in the muscle.

It is surprising that few investigators have challenged the basic premise of Hultman and his associates regarding carbohydrate as the preferred fuel for endurance exercise. This is despite the fact that in parallel with research concluding that carbohydrate is the preferred fuel for endurance exercise, there is a great deal of research examining the effects of low carbohydrate - high fat diets on endurance performance.

Numerous human and animal studies support the hypothesis that both animals and humans adapt to high fat-low carbohydrate eucaloric diets in much the same way they as they do to endurance training. That is, they experience increased mitochondrial oxidative capacity of the Krebs cycle and beta-oxidative enzymes.

Phinney and co-workers in the 1980s conducted a series of studies involving ketogenic diets. The first of these measured the capacity for endurance exercise and changes in metabolic fuel utilisation with adaptation to a ketogenic diet in six moderately overweight subjects (Phinney, et al. 1980). The findings of this study were, that with the consumption of a ketogenic diet, RQ was low, and blood glucose and muscle glycogen was maintained during exhaustive exercise. Thus indicating that on adaptation to a ketogenic diet, lipid becomes the major fuel with net carbohydrate utilisation significantly reduced during exhaustive exercise. It was stated by the authors that “the most important finding of this study is that prolonged exercise can be sustained after virtual absence (<10g/d) of dietary carbohydrate for six weeks.”

A second study by Phinney (1983a) looked at the human metabolic response to chronic ketosis without caloric restriction in lean individuals. A ketogenic diet supplying less than 20g/d of carbohydrate was consumed for a period of four weeks. After the four week diet a ketogenic state had been induced which was well tolerated by lean individuals.

A third study by Phinney et al (1983b) used a similar dietary regime as previously mentioned, and five endurance trained cyclists. The protocol involved measuring times to exhaustion during continuous exercise on a cycle ergometer. From week one to week four of the ketogenic diet, RQ decreased from 0.83 to 0.72 and was accompanied by a three-fold drop in glucose oxidation and a four-fold decrease in muscle glycogen use.
time to exhaustion on a eucalorically balanced diet was 147 minutes compared to 151 minutes at week four of the ketogenic diet. It was concluded that after four weeks on a ketogenic diet, aerobic endurance exercise is not impaired in a group of highly trained cyclists.

Jansson and Kaijser (1982) studied the effects of a diet high in fat (69% of energy from fat) and a diet high in carbohydrate (75% and 8% of energy from carbohydrate and fat respectively). Muscle glycogen depletion, following a bout of exercise at 60% VO\textsubscript{2}max was 94mmol/L/kg d.w (dry weight) with the high fat diet compared to 144mmol/kg d.w. after following the high carbohydrate diet. Lactate production was significantly lower after following the high fat diet as compared to the high carbohydrate regimen. The R-value (respiratory quotient) was lower following the high fat dietary regime and was associated with an increase in free fatty acid extraction by the exercising muscle, thus indicating an adaptation to increased fat oxidisation. The contributions of fat and carbohydrate to oxidative metabolism were determined for the different dietary regimes. Following the high fat diet the contribution of fat and carbohydrate were 63% and 37% respectively. Whereas, following the carbohydrate-rich diet 27% and 73% were contributed from fat and carbohydrate respectively. It was concluded from this study that a diet high in fat increases the relative contributions of fat to oxidative metabolism; the major contributing factor being the increase in plasma free fatty acids. The reduction in carbohydrate to oxidative metabolism was attributed to a reduction in muscle glycogen breakdown.

2.6.2.1 Animal studies

Research was performed in the late 1970s to observe the metabolic responses to exhaustive exercise in racing sled dogs fed diets containing medium, low and zero amounts of carbohydrate (Hammel, et al. 1977). Eighteen dogs were assigned to three different dietary groups whose composition of protein, fat and carbohydrate were as follows: Diet A, 39:61:0; Diet B, 32:45:23; and Diet C, 28:34:38 (macronutrient proportions represent percentage of total energy). Dietary intervention lasted a duration of twenty-eight days, at the end of which an exhaustive bout of exercise were performed. Results showed that free fatty acid increment during exercise was highest in the dogs fed diet A. These dogs had a superior ability to mobilise body fat during exercise.

Miller et al (Miller, et al. 1984) studied adaptations to a high fat diet and exercise endurance in rats. Eighty-seven male rats were assigned to either a high fat - low carbohydrate diet or a normal diet. The rats were run to
exhaustion at weeks one or five of dietary intervention. It was evident that at both weeks one and five, rats fed the high fat-low carbohydrate diet ran significantly longer to exhaustion. Adaptations to the low carbohydrate diet involved lower liver and muscle glycogen, a decreased rate of glycogen breakdown, decreased lactate production and increased levels of blood ketones. Results of this study indicate that when following a high fat-low carbohydrate diet, rats are capable of prolonged, intense exercise despite the fact muscle glycogen levels are lower. This phenomenon seems attributable to muscular adaptation to a high fat diet, the increased ability to oxidise fat and thus spare glycogen.

2.6.3  Adaptation to endurance exercise

Highly trained individuals oxidise proportionally more fat and less carbohydrate at any given workload than non-trained individuals (Holloszy and Booth 1976). The major adaptation with endurance exercise is the slower utilisation of muscle glycogen and a greater reliance on fat oxidation with less lactate production at a given intensity. Hormonal and enzyme systems are involved in adaptation to endurance exercise (Bloom, et al. 1976; Holloszy and Coyle 1984; Holloszy and Booth 1976; Lithell, et al. 1981).

2.6.3.1  Hormonal adaptation to endurance exercise

The mobilisation of fuel is regulated by neural and hormonal response. The sympathetic nervous system is activated by the release of noradrenaline, and insulin levels decline. There is also an increase in glucose counter regulatory hormones, including glucagon, adrenaline, growth hormone and cortisol. The balance of insulin and glucagon play an important role in the regulation of glucose, and the opposing effects of insulin and adrenaline on lipolysis are important in the regulation of free fatty acid availability. Hormonal changes as physical exercise becomes prolonged, increases the availability of fat for the exercising muscle.

Hormonal changes during exercise differ between trained and untrained individuals. Bloom (1976) investigating hormonal changes during exercise in trained and untrained subjects, showed marked differences in blood-borne fat metabolites and a greater degree of lipolysis in well trained subjects, compared to untrained subjects. Plasma catecholamines increased significantly less in the trained cyclists compared to untrained subjects during strenuous physical exercise. Plasma insulin was depressed to a greater extent, and plasma glucagon and human growth hormone rose to a
greater extent in untrained subjects during exercise. Bloom suggested that there is a greater sensitivity of the tissues of trained athletes to catecholamines, glucagon and human growth hormone. These hormonal differences between trained and non-trained individuals persist when the subjects work at the same relative workload. Such differences allow the athlete to optimise fat utilisation during prolonged exercise.

2.6.3.2 Muscle, mitochondrial and enzymatic adaptation to endurance exercise

A notable adaptation to endurance training is the increase of skeletal muscle mitochondria reticulum (50 to 100%) (Davies, et al. 1981). Endurance training increases skeletal muscle mitochondrial density as demonstrated by the increase in respiratory chain enzymes and increases in total protein content (Holloszy and Coyle 1984). Increases in mitochondrial mass is thought to improve lipid metabolism during exercise and suppress glycogenolysis.

Physical training increases components of the electron transport chain required to oxidise fatty acids, and enzymes involved in fatty acid translocation, the beta-oxidation pathway, and the citric acid cycle, (Mole, et al. 1971). The latter is achieved by an increase in the concentration of mitochondria per unit of muscle tissue, providing greater potential for the movement of cytosolic metabolites.

Increased capillarisation of the muscle, enables a greater exchange of intra- and extracellular metabolites due to increased surface area. In this way, increased capillarisation can improve the transport of fatty acids into the fibres. In addition, there is greater translocation of fatty acids into the mitochondria via the carnitine palmitate transferase system in the trained state (Gollnick, et al. 1985; Gollnick and Saltin 1982).

Lithell and co-workers (1981) have demonstrated the importance of lipoprotein lipase in a study involving elite Swedish soldiers. Lipoprotein lipase activity increased with endurance training in the working muscle, thus increasing the access of fatty acids. The extent of elevation in LPL was related to the degree of physical effort.

Endurance training results in decreased glycolytic flux and increased lipid oxidation. An athlete's metabolism is adapted in such a manner as to allow for increased power output from fat oxidation.
Many researchers who believe that carbohydrate is the most important fuel for endurance exercise, argue that the limiting factor in endurance exercise is the preformed glycogen in the muscle (Ahlborg, et al. 1967; Bergström, et al. 1967; Hermansen, et al. 1967; Hultman 1967). It is of popular belief that fatigue is attributable to muscle glycogen depletion. However this view is an over-simplification of the phenomenon of fatigue. In the thirty years since muscle biopsy techniques were developed to measure glycogen levels, no causal relationship has been found between glycogen depletion and fatigue. No mechanism has been found for such a relationship.

Fatigue is defined physiologically as the inability to maintain power output. The aetiology of fatigue is multi factorial (Bannister and Cameron 1990; Parry-Billings, et al. 1990). The causes of fatigue include:
1. Depletion of ATP/phosphocreatine in muscle
2. Accumulation of protons in muscle
3. Depletion of glycogen in muscle
4. Decrease in blood glucose
5. Increased blood ammonia (from protein degradation)
6. Altered brain neurotransmitters (serotonin “exhaustion syndrome”)

The first three factors above relate to the muscle itself and the last three involve the central nervous system. It is thought that the brain is able to detect changes in the levels of normal constituents of the blood which can then act as a specific signals and increase the sensitivity of the athlete to fatigue, thus making the athlete give up more easily (Bannister and Cameron 1990).

The phenomenon of fatigue is thus very complex because it involves both physiological and psychological factors. The factors involved in the control of fatigue may interact. The view that fatigue is solely related to glycogen depletion is a limited one.

2.6.5 A high fat diet for the endurance athlete?

The physical endurance of an individual is determined by the capacity of the working muscle to cover its energy needs by aerobic metabolism of fatty acids and limited carbohydrate stores of the body (Davies, et al. 1981). Therefore the greater the aerobic fitness, the greater the contribution of fat metabolism to energy expenditure. Evidence discussed thus far suggests that a diet higher in fat, may be beneficial for endurance performance. A higher fat diet has been shown to potentiate the effects of a
system already adapted to the preferential use of fat as a fuel. However, firstly it must be determined whether it is irresponsible to advocate a high fat diet in view of the well established relationship between high fat diets, elevated serum cholesterol levels and the associated increased risk of coronary heart disease and atherosclerosis. These important epidemiological observations should not be disputed. However, such associations have been based on the general population which is largely sedentary. What of the minority of individuals within the population who are very active?

2.7 Definitions of physical activity, exercise and physical fitness.

In the literature, "physical activity", "exercise" and "physical fitness" are often not well defined. This can cause difficulties in comparisons of study results. Physical activity is defined as any bodily movement produced by skeletal muscle that results in energy expenditure (Caspersen, et al. 1985). Physical activity can be categorised into occupational, sports, conditioning, household or other activities (Caspersen, et al. 1985).

Exercise is a subset of physical activity which is planned, structural and repetitive with a final or intermediate objective of the improvement or maintenance of physical fitness (Caspersen, et al. 1985).

Physical fitness is a set of attributes that are either health or skill related. These attributes can be measured with specific tests (Caspersen, et al. 1985). Caspersen et al have defined five health related components of physical fitness; a major component being cardiorespiratory endurance which can be measured in the laboratory by a maximum oxygen uptake test.

It may be possible that these three categories of energy expenditure may result in different health benefits.
2.8 **Assessment of physical performance**

Two main approaches are generally used in the assessment of physical performance:

1. Physical fitness tests with the scoring of actual performance in situations representative of the basic performance demands.
2. Studies of cardiopulmonary function at rest and/or during exercise. (Astrand and Rodahl 1986).

### 2.8.1 Maximum aerobic power

Maximum aerobic power is an objective laboratory determinant of cardiovascular fitness. Maximum aerobic power can be defined as the highest oxygen uptake an individual can attain during exercise while breathing at sea level.

In a test of maximum aerobic power, an individual's VO\textsubscript{2max} (maximum oxygen intake) is calculated from the collected expired air. The test performed should be specific to an individual's given sport, i.e., a cyclist's VO\textsubscript{2max} should be measured on a cycle ergometer, whereas the VO\textsubscript{2max} of a runner should be determined on a treadmill. It should be noted that values depicted from a treadmill test are on average 4-8% higher compared to those on a cycle ergometer. A possible explanation for this is increased number of muscles engaged on a treadmill test. However, it is evident that elite cyclists can achieve VO\textsubscript{2max} values on a bicycle ergometer that equal treadmill values.

A maximum aerobic power test usually begins with a submaximal work rate which also serves as a warming-up activity. Workloads can then be increased in several ways:

1. The workload may be immediately increased to a level which has been found to represent the predicted maximal workload for the subject, as predicted from preliminary tests. This workload is maintained for three to six minutes.
2. The load may be increased in stepwise fashion with several submaximal, maximal, or "supermaximal" loads. The subject continues exercising for five to six minutes at each workload, with or without resting periods between each load.
3. The load may be increased stepwise every minute or so, until exhaustion (Astrand and Rodahl 1986).
When any one of the procedures has been carried out carefully, they result in the same maximal oxygen uptake (McArdle, et al. 1973; Stamford 1976).

Ideally a test of maximum oxygen uptake should meet the following general criteria:
1. Blood lactate levels of about 8-9mmol/L
2. The attainment of a maximum heart rate.
3. An R-value greater than 1.
4. Perceived exhaustion by the subject.

The maximum workload attained by an individual during a maximal oxygen uptake test can be a measure of assessing changes in exercise capacity over a period of time.

As a predictor of physical performance, maximum aerobic power is not necessarily an accurate measure. Performance is dependent on a number of factors including technique, tactics, motivation, state of training, experience and various other psychological factors. Therefore individuals with the highest VO\textsubscript{2}max levels are not necessarily the best performers. Performance ability can depend on how an individual utilises their energy and the percentage of VO\textsubscript{2}max they are able to perform at. High aerobic power is needed for the attainment of top exercise results, however a high level of aerobic power does not guarantee good physical performance.

A number of studies have been conducted to determine the predictability of an individual's performance in an all-out run taxing the oxygen transport system maximally, from the measurement of maximum oxygen uptake. Shepard (1984) reviewed thirty-seven such studies and the coefficients of correlation ranged from 0.04 to 0.90. It was evident that the higher correlations were found when a wide range of subjects were observed.

Measurement of maximum aerobic power does not reveal an individual's exact potential to perform well in aerobic power events. However it is a useful tool to measure changes over time for the assessment of training or some type of intervention (eg dietary). It can also be used to categorise individuals into broad categories of fitness levels.

### 2.9 Dietary Assessment

Dietary assessment of individuals or populations creates somewhat of a challenge for researchers.

Four methods of dietary assessment are recognised. These include diet records, dietary recall, diet history and food frequency questionnaires.
The diet record and the dietary recall methods attempt to measure food intake during a specific time with the values obtained assumed to be representative of the usual diet. Conversely, the diet history and food frequency questionnaire techniques attempt to assess habitual intake directly.

The use of any one method is highly dependent on the aims, resources and subjects of the research undertaken. The appropriate choice of method relies upon subject motivation and numbers, costs, available resources eg. trained personnel, and time limitations. A compromise is present between obtaining highly accurate results which may be time consuming and thus eventuating in low subject co-operation rates; and a less accurate measure which may result in high subject co-operation rates.

2.9.1 Diet Records

The diet record technique for dietary assessment is the most accurate of the measurements and the most time consuming. Subject motivation is therefore a priority.

There are variations present within the diet record technique. The time period for recording food intake can vary (eg. twenty-four hours, three, four five and seven days). The recording technique can vary from precise weighing to the use of household measures and the estimation of portion sizes from photographs and food models.

The number of recording days required is dependent on the aims, desired accuracy, and the subjects themselves. The twenty-four hour record is utilised most commonly in large scale studies because of its simplicity and time-saving aspects. However this method has serious short-comings. Wide fluctuations in daily intake are known to occur (Edholm, et al. 1955). Extreme fluctuations in dietary intake are present from day-to-day which makes this technique somewhat suspect.

The number of days required to classify 80% of subjects into tertiles of nutrient intake with 95% confidence is approximately three to seven days for energy; five to seven days for protein, five to nine days for fat; and two to four days for carbohydrate (Bingham 1987; Bingham, et al. 1981; Gardner and Heady 1973; Liu, et al. 1978; Marr and Heady 1986). Thus, if the objective is to determine the intake of the macronutrients, a time period of approximately five - seven days should prove to be sufficient to estimate habitual intake to the degree of accuracy that will not result in a tedious procedure and thus low compliance rates. The inclusion of weekend days in multi-day food records is important to account for weekend variability (Tarasuk and Beaton 1992).
Performing multiple five to seven day diet records over a period of time (eg dietary intervention period in research) will result in greater accuracy. Well-motivated subjects are at a premium in these circumstances.

A number of researchers have evaluated the validity of the diet record. Persson and Carlgren examined the internal validity of food records by comparison with chemical analysis of duplicate diet portions. Results obtained indicated the two methods were well correlated (Persson and Carlgren 1984).

2.10 Summary

It is evident that both diet and exercise affect plasma lipids and lipoproteins. Endurance trained athletes have lipoprotein profile associated with a decreased risk of coronary heart disease. It is unclear however, the extent to which diet and exercise play a role in this phenomenon.

The extent to which endurance exercise may off-set the adverse effects of extreme dietary intakes is not well defined in the literature. Previous research has indicated that in very physically active individuals, exercise can in part, counter the effects of extreme dietary intakes. This has predominantly been observed where high intakes of carbohydrate have failed to decrease HDL cholesterol concentrations to the same extent as observed in sedentary individuals. Less emphasis has been placed on the effects of high fat intakes in highly trained endurance athletes. Some investigators argue that athletes are as susceptible as their sedentary counterparts to the adverse effects of high fat (particularly saturated fat) intakes. However, evidence obtained from well-controlled intervention studies does not support this hypothesis. Some studies have shown that the consumption of higher amounts of fat in the diet does not adversely affect plasma lipids when individuals are partaking in large amounts of intense physical exercise. Many of these studies are limited with respect to subject numbers and duration of dietary intervention. An important factor determining whether exercise is a dominant factor influencing of lipoproteins, over that of diet, is the amount and intensity of exercise. It is not yet clear in the literature the amount and intensity of exercise required to exert a beneficial effect on the plasma lipoprotein profile. It appears that endurance type exercise, as opposed to largely anaerobic strength type exercise, induce lipoprotein changes.

Research indicating a relationship between high carbohydrate diets, muscle glycogen and enhanced endurance performance has led to the recommendation of carbohydrate-rich diets for endurance athletes. In
parallel with this research, other investigators have obtained less publicised results showing favourable effects of relatively high fat diets on endurance capacity. However, investigators caution against the recommendation of higher fat diets for athletes in view of the well-established relationship between high fat diets, particularly saturated fat, and an increased risk of coronary heart disease and certain cancers.

The logics of such a cautionary note need to be addressed. Lack of adequate well-controlled research in the area looking at the long-term effects of diet and exercise on plasma lipids and lipoproteins has necessitated this study.
3 Methods

3.1 Study design

The study was a parallel design of two dietary interventions in competitive road cyclists (see figure 3.1). The study was approved by the Ethical Committee of the Otago Area Health Board, and written informed consent obtained from each study subject.

The subjects were thirty-six competitive road cyclists recruited from the 120 eligible registered cyclists in the Dunedin area. Recruitment was by letter sent to all registered cyclists in July/August, 1992.

Baseline measurements were obtained from all subjects in early August, 1992. These variables included age, weight, height, body mass index (BMI), total body fat as estimated from the sum of six skinfolds, maximum aerobic power via a VO\(_2\)max exercise test and the measurement of plasma lipid and lipoprotein variables obtained from two fasting blood tests.

Subjects were randomised to receive one of two diets, HC (high CHO) or HF (high fat), after matching for TC at baseline. Sixteen subjects commenced diet HF and twenty diet HC. Two subjects were not randomised and commenced diet HC after expressing preference for this diet.

The duration of dietary intervention was twelve weeks. Participants were given the option of continuing in the study for an additional four weeks. Twenty-two subjects remained in the study for a total duration of sixteen weeks (n=15 for HC and n=7 for HC).

Final analyses were carried out on thirty-four subjects after a subject from HF withdrew at week nine due to work and cycling commitments and the results from a further HF subject were not included due to commencement of medication known to affect lipid metabolism.
Figure 3.1 Study Design

Baseline variables
- plasma lipid/lipoprotein profile
- weight
- height
- estimation of total body fat
- VO2max exercise test

Key
- Blood Test
- Exercise Test

n=15
n=20
n=7
n=15
16 Weeks
3.2 Blood tests

Blood samples were collected at baseline and at weeks four, eight, twelve and sixteen of dietary intervention. Two blood samples were taken within each blood testing week to account for intra-individual variation. Lipid and lipoproteins measured included total, LDL, HDL, HDL₂ and HDL₃ cholesterol and triglycerides. In addition to this, measurements of apolipoproteins A1 and B were performed on the first blood sample taken at each blood testing period. Ferritin and haemoglobin were measured at baseline.

As far as possible, blood samples were taken at least twelve hours post-exercise and after an overnight fast of eight to twelve hours.

Blood pressure was measured at baseline and at week twelve by nurses with a random zero sphygmomanometer.

3.3 Dietary Design

Two diets differing in the proportions of fat and carbohydrate were designed.

The high carbohydrate diet (HC) consisted of the following proportions of macronutrients as a percentage of total energy:

- 60-65% carbohydrate
- 20-25% fat
- 15% protein

The high fat diet (HF) comprised of the following macronutrients as a percentage of total energy:

- 35-40% carbohydrate
- 45-50% fat
- 15% protein

The HF group were encouraged to eat foods high in saturated fats (SFA) eg. dairy products (cheese, cream, butter), coconut, chocolate. Protein intake was isocaloric for both diets, at a level of approximately
15% of total energy consumed. Both study diets were designed to be isocaloric.

3.3.1 Diet prescription

Prior to the intervention period each subject received dietary counselling from a dietitian on how to meet the requirements of the study diets. In addition, subjects were seen fortnightly and telephone contact was available throughout the study where the opportunity to discuss any questions regarding the practicality of the diets was made available.

Dietary prescription was based on the following well-known dietary principles:

1. Elimination - This involved the exclusion of foods, not appropriate to the particular study diets.
   eg. HC - The elimination of foods high in fat
   eg. HF - The avoidance of foods particularly high in simple carbohydrates

2. Substitution - This included the purchase of foods that are suitable to the study diets.
   eg. HC - The substitution of standard foods with low fat alternatives
   eg. HF - The use of higher fat alternatives

3. Modification - This encompassed the alteration of food preparation
   eg. HC - The modifications of recipes to increase the carbohydrate content and lower the fat content
   eg. HF - The addition of extra fat during food preparation

Each subject received a booklet providing dietary guidelines for HC and HF. This included lists of "foods to avoid", and "preferred foods" to be consumed, for each of the two diets. Examples of daily menu plans were provided as a guide and appropriate foods to be eaten during training were included. High fat and high carbohydrate recipes were provided for the HF and HC group respectively (see appendix).

3.3.2 Food provisions

Specific foods were supplied to subjects to enable them to meet the requirements of their prescribed diets.
Group HC received high carbohydrate confectionary eg fruit gums (97% CHO) and HF received chocolate bars (composition: fat 38%, CHO 50%, protein 12%). These foods were eaten mainly during training.

In addition, the high fat group received cream and specially manufactured bread which contained added quantities of butter or coconut oil (composition: 25% fat, 12% protein, 63% CHO; expressed a % E).

3.4 Dietary assessment

Five-day food diaries were completed at baseline and at weeks four, twelve and sixteen during the study. Nutrient intake was calculated using the computer program “Diet Entry and Storage / Diet Cruncher” (Marshall 1993) and data from the New Zealand Food Composition database (Ross and Ward 1985).

Subjects were asked to record three week days and two weekend days. Participants were instructed to record everything they consumed during the recording period. The importance of recording all dietary food intake including dietary supplements and training foods was stressed to the cyclists.

3.5 Anthropometry

3.5.1 Body weight and height

Height was measured at baseline and body weight at baseline and four weekly intervals. Weight was measured using electronic weight scales (Seca, model 770) and height on a standard stadiometer.

3.5.2 Estimation of total body fat

Total body fat, estimated by the sum of six skin folds, was measured at baseline and week twelve.

The six skinfold sites included, triceps, subscapular, supraspinale, abdominal, anterior thigh and medial calf. Each skin fold was measured once and then the sequence of measurements was repeated. Slimline skinfold calipers were used to measure to the nearest 0.5mm.
The sum of the six skinfolds was transferred to the following equation to estimate total body fat. Here, the individual's height is taken into account:

\[
\text{Total body fat} = \text{sum of six skinfolds} \times \frac{170.19}{\text{actual height (cm)}}
\]

All readings (except the abdominal skinfold) were taken on the right side of the body. The procedures used were those described by Ross and Ward (Ross and Ward 1985).

3.6 Physical fitness assessment

The maximum aerobic power (V02max) of each subject was measured as an indication of physical fitness. Maximum aerobic power was calculated from an exercise test eliciting the subject's maximum oxygen uptake (VO2max). VO2max exercise tests were performed at baseline and week twelve.

VO2max was measured by an incremental workload exercise test on a Rodby RE820 electromagnetically braked cycle ergometer. Subjects began the test on a set workload which was dependent on the cycle racing grade to which they belonged (A-grade, 200Watts; B-grade, 150Watts; and C-grade, 100Watts). Following a standard initial two minute warm-up on the set workload, the workload was increased at a rate of 25Watts per minute until the subject was exhausted and unable to continue.

The subjects breathed through a rubber mouth-piece attached to a 2-way Koegel “Y” valve. The inhalation section of the valve was open to room air, while the exhalation side was attached to an adjustable mixing chamber with a maximum volume of 6.5 litres. A noseclip was placed securely on the subject's nose to ensure that all inspired and expired air passed through the mouthpiece. The volume of inspired air and respiratory periods were measured, while mixed expired oxygen and carbon dioxide concentrations were monitored with an on-line gas analysis system developed at the School of Physical Education, University of Otago.

Mean values of the following variables were recorded at thirty second intervals: oxygen consumption, minute ventilation, respiratory exchange ratio, breathing frequency, tidal volume, inspiratory period, and expiratory period.

Apparatus used to determine the subject's maximum aerobic power were as follows: Applied Electrochemistry Inc. oxygen sensor N-22 and oxygen analyser S-3A (Sunnyvale, California); Datex Normocap carbon
oxygen and carbon dioxide gas analysers were calibrated with a gas mixture consisting of N₂, O₂, and CO₂ in concentrations typical of mixed expired air. Calibration gas was determined on an Airspec MGA 2000MKII mass spectrometer Kent, UK), which itself was calibrated against air and gravimetrically determined standard (NZ Industrial Gases, Wellington). The entire gas analysis system was controlled by a locally-built analogue-to-digital converter and a BBC 512 Master series micro-computer (Cambridge, UK).

Heart rate was monitored throughout the exercise test using electrodes connected to an electrocardiogram (ECG).

The maximum oxygen uptake of each subject was recorded. In addition, the maximum workload attained during the test and the time to exhaustion were recorded for all participants.

### 3.6.1 Training assessment

Training records were completed by each subject during the study period. This was included in the bound booklet each participant received. Athletes were asked to record cycling distance (km) covered, time spent bicycle training and non-bicycle training eg. weight training, running kayaking, swimming, etc. An additional section asked for training comments (see appendix).

### 3.6.2 Performance assessment

A detailed performance record was kept throughout the study. Participants recorded all competitive races, distances and rated their performance on a scale of one to ten (see appendix).

### 3.7 Sports nutrition information pamphlets

Sports nutrition information pamphlets were given to subjects during the study to increase subject interest and add potential benefit from the study. Subject matter was designed so not to interfere with any of the dietary requirements of the study. Topics included fluids, hydration
guidelines, information on sports drinks and the importance of iron for endurance athletes.

3.8 Questionnaires

3.8.1 General information questionnaire

Prior to dietary intervention, all subjects completed a general information questionnaire. This included demographic information, previous medical history's, previous dietary practices, dietary supplement usage, and information regarding exercise (see appendix).

3.8.2 Post-study questionnaire

A questionnaire was sent to all subjects three months following the completion of dietary intervention to determine subject reaction and feedback regarding dietary composition. Subject matter covered both the palatability of diets and subjective information regarding how the diets effected physical training and performance (see appendix).

3.9 Laboratory analysis

Blood lipid/lipoprotein tests were measured on a Cobas Fara Centrifugal Analyser. Cholesterol concentration in plasma and lipoprotein factions was measured enzymatically using the Boehringer Enzymatic colorimetric method (Siedel, et al. 1983). Plasma triglycerides were measured enzymatically using a Roche kit. The study period interassay coefficient of variation for cholesterol was 1.5% and 4% for triglycerides. Plasma low density lipoprotein cholesterol was calculated using the Friedwald equation (Friedwald, et al. 1972).

HDL-C was isolated through precipitation of apolipoprotein B-containing lipoproteins with phosphotungstate/magnesium chloride solution (Assmann, et al. 1983). The coefficient of variation for HDL cholesterol was 2%. HDL3 measurements were made using a Poly Ethylene Glycol (PEG) method (Demacker 1985). The coefficient of variation for HDL3 was 2.8%. HDL2 cholesterol was calculated from the difference of HDL3 cholesterol from total HDL cholesterol.
Apolipoprotein A1 and B measurements were determined using Boehringer (Tina-quant) test kits using Boehringer calibrators. The interassay coefficient of variation for apolipoprotein A1 and B were 4.3% and 3.6% respectively.

Calibration and quality control was maintained by participation in the New Zealand Analysis proficiency programme.

3.10 Statistical analysis

The data were analysed using a “Systat” statistical package and a “Statview” package.

Lipoprotein/lipid parameters and exercise variables for groups HC and HF were analysed for differences by independent t-tests. For the two groups, the dependent variable means were pooled and analysis of variance (ANOVA) with univariate repeated measures was employed to compare the time points pooled over the two groups, ie. the significance of change pooled over both diets.

Univariate repeated measures ANOVA was used to determine the significance of difference in change between the two groups at different sampling times, ie. to determine if the change in the two groups were statistically similar.

Paired one-tail t-tests were used to analyse the changes in the individual groups from baseline to weeks twelve and sixteen of dietary intervention.

Weekly averages for training variables were calculated for each group. ANOVA was employed to compare training variables between the two groups.

Dietary data for groups HC and HF were analysed for differences by independent two-tailed t-tests.

Pearson correlation coefficient matrix was employed to measure associations between plasma lipid/lipoproteins and exercise variables.
4 Results

4.1 Subjects

Table 4.1 shows baseline characteristics of study subjects. The subjects were thirty-six healthy, male endurance cyclists.

The age of study subjects ranged from 17 to 51 years. The mean age for HC and HF were (mean ± SD) 31.1 ± 9.9 and 24.7 ± 5.7 years respectively (p=0.031). Median age was 29 years and 23 years for groups HC and HF respectively. The group mean total cholesterol was 4.9 ± 0.7mmol/L, with a mean of 4.72 ± 0.72mmol/L for HC and 5.02 ± 0.63mmol/L for HF.

The average number of years subjects had been involved in competitive cycling was 7.5 years (range 2 to 22 years).
Table 4.1  Characteristics of subjects at entry to study according to group randomisation. Mean values (± SD).

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<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>20</td>
<td>15</td>
</tr>
<tr>
<td>Age (years)</td>
<td>31.1 ± 9.9</td>
<td>24.7 ± 5.7</td>
</tr>
<tr>
<td>Gender</td>
<td>all males</td>
<td>all males</td>
</tr>
<tr>
<td>Baseline Total Cholesterol (mmol/l)</td>
<td>4.72 ± 0.72</td>
<td>5.02 ± 0.63</td>
</tr>
<tr>
<td>Body Weight (kg)</td>
<td>76.51 ± 6.44</td>
<td>73.06 ± 6.22</td>
</tr>
<tr>
<td>Total body fat (S6SF)</td>
<td>59.4 ± 18.8</td>
<td>45.7 ± 13.4</td>
</tr>
<tr>
<td>Number of years cycling</td>
<td>9.3 ± 5.4</td>
<td>5.1 ± 1.9</td>
</tr>
</tbody>
</table>
4.2 Dietary data

4.2.1 Baseline

Table 4.2 (Figure 4.1) shows the baseline diets of HC and HF. Analysis of five-day food diaries revealed a total energy intake of 11657 ± 2695KJ/day for HC and 12584 ± 3060KJ/day (mean ± SD) for HF (p=0.37). Total fat intake (expressed as % of total energy) was greater in HF (34.9 ± 6.7%) compared to HC (28.3 ± 6.0%) (p=0.006). Fat intake as expressed in terms of absolute amounts was significantly higher in HF compared to HC (115.6 ± 25.1g vs 89.3 ± 29.1g, HF and HC respectively) (Figure 4.5). The P:S ratio was 0.3 ± 0.1 for both HC and HF. Saturated and monounsaturated fat contributed more energy to the HF group compared to the HC diet group at baseline (see Table 4.2 & Figure 4.6).

Reported intakes of carbohydrate, protein, dietary fibre and alcohol consumption were comparable for the two groups (see Table 4.2 & Figures 4.10 & 4.11).

4.2.2 Study periods

Tables 4.3, 4.4 & 4.5 (Figures 4.2 to 4.12) show the diets of both groups during the study.

4.2.2.1 Week twelve

Table 4.4 shows the diets of both groups to week twelve. Total fat intake (expressed as % of total energy) for HF was 44.9 ± 4.6% which was 26.1% greater than HC who consumed 18.1 ± 4.5% of energy from fat (p=0.0001) (Figure 4.3). Fat intake expressed in terms of absolute amount were significantly higher in HF compared to HC, (188.0 ± 44.2g vs 53.1 ± 18.8g, HF and HC respectively) p=0.0001 (Figure 4.5).

The percentage of energy derived from saturated fat, polyunsaturated fat and monounsaturated fat were significantly higher in the HF diet group compared to the HC group (see Table 4.4). SAFA intake (as expressed as % of total fat) was significantly higher in HF compared to HC; whereas PUFA intake (as expressed as % of total fat) was significantly higher in HC compared to HF (see Figure 4.8). The P:S ratio was 0.44 for HC compared to 0.23 for HF (p=0.0009).
HC consumed 62.3 ± 6.0% of energy from carbohydrate, 23.6% more than HF (38.7 ± 6.7%), p=0.0001.

Protein (expressed as % of total E) and dietary fibre intakes were similar in both groups (Figures 4.3 & 4.10). Alcohol consumption, although slightly higher in the HC group, was not significantly different from HF (see Table 4.4 & Figure 4.11).

Dietary cholesterol intake was significantly higher in HF compared to HC (541 ± 220mg vs 241 ± 105mg, HF and HC respectively), p=0.0001 (Figure 4.12).

There was a significant difference in reported energy intake between the two groups, with HC consuming significantly less energy than HF (mean ±SD: 10381 ± 2491KJ vs 15501 ±3278KJ, p=0.0001).

HF consumed approximately 11% more calories as fat during dietary intervention, compared to their previous habitual intakes. Conversely, the HC group, on average, consumed approximately 10% less calories derived from fat when compared to their prior usual intake.

### 4.2.2.2 Week sixteen

Table 4.5 illustrates dietary intakes of both groups to week sixteen of dietary intervention. Reported intakes of nutrients remained similar throughout the study.

HF consumed significantly more energy from fat (48.7 ± 6.2%) compared to HC (16.8 ± 4.9%) (p=0.0001). Absolute amounts of fat were greater in HF, 213 ± 35.8g compared to the 48.5 ± 18.0g consumed by HC (p=0.0001) (Figure 4.5). HF also consumed significantly more energy from saturated fat (HF=22.0%; HC=5.8%, p=0.0001), monounsaturated fat (HF=16.4%; HC=5.6%, p=0.0001), and polyunsaturated fat (HF=5.7%; HC=3.2%, p=0.0009). SAFA intake (as expressed as % of total fat) was significantly higher in HF compared to HC; whereas PUFA intake (as expressed as % total fat) was significantly higher in HC compared to HF (Figure 4.9). The P:S ratio was significantly higher for HC at 0.55 compared to 0.26 for HF (p=0.04).

Carbohydrate contributed 60.5% of total energy for HC and 34.4% for HF (p=0.0001), a difference of 26.1%.

Intakes of protein (as expressed as %E), alcohol and dietary fibre were comparable for the two diet groups (Figures 4.10 & 4.11).

Dietary cholesterol intake was significantly higher in HF compared to HC (602 ± 222mg vs 223 ± 70mg, HF and HC respectively) (Figure 4.12).
Again there was a significant difference in reported energy intake between HC (mean ± SD: 10607 ± 1888KJ) and HF (16547 ± 2782KJ) (p=0.0004).
Table 4.2 Baseline diet for HC and HF, n=20 for HC and n=15 for HF. All values are means ± SD

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (KJ)</td>
<td>11657 ± 2695</td>
<td>12584 ± 3060</td>
<td>0.37</td>
</tr>
<tr>
<td>Total fat (g)</td>
<td>89.3 ± 29.1</td>
<td>115.6 ± 25.1</td>
<td>0.012</td>
</tr>
<tr>
<td>Saturated fat (g)</td>
<td>37.3 ± 15.2</td>
<td>50.0 ± 10.3</td>
<td>0.012</td>
</tr>
<tr>
<td>Total fat (%E)</td>
<td>28.3 ± 6.0</td>
<td>34.9 ± 6.7</td>
<td>0.006</td>
</tr>
<tr>
<td>Carbohydrate (%E)</td>
<td>52.8 ± 8.0</td>
<td>46.0 ± 11.1</td>
<td>0.09</td>
</tr>
<tr>
<td>Protein (%E)</td>
<td>15.0 ± 2.5</td>
<td>16.0 ± 4.8</td>
<td>0.44</td>
</tr>
<tr>
<td>Saturated fat (%E)</td>
<td>11.7 ± 3.4</td>
<td>15.3 ± 3.6</td>
<td>0.007</td>
</tr>
<tr>
<td>Monounsaturated fat (%E)</td>
<td>9.3 ± 2.8</td>
<td>11.1 ± 2.3</td>
<td>0.014</td>
</tr>
<tr>
<td>Polyunsaturated fat (%E)</td>
<td>3.8 ± 1.2</td>
<td>4.2 ± 1.0</td>
<td>0.28</td>
</tr>
<tr>
<td>P:S ratio</td>
<td>0.3 ± 0.1</td>
<td>0.3 ± 0.1</td>
<td>0.23</td>
</tr>
<tr>
<td>Cholesterol (mg)</td>
<td>292 ± 130</td>
<td>425 ± 118</td>
<td>0.006</td>
</tr>
<tr>
<td>Alcohol (%E)</td>
<td>5.2 ± 7.3</td>
<td>3.3 ± 4.6</td>
<td>0.40</td>
</tr>
<tr>
<td>Dietary fibre (g)</td>
<td>33.1 ± 10.4</td>
<td>34.2 ± 15.4</td>
<td>0.81</td>
</tr>
</tbody>
</table>

p-values are for differences between HC and HF as determined by independent two-tailed t-tests.

Table 4.3 Dietary intakes for HC and HF to week four of dietary intervention, n=16 for HC and n=13 for HF. All values are means ± SD

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (KJ)</td>
<td>10153 ± 2372 (+1400 KJ of training foods)</td>
<td>15075 ± 2508</td>
<td>0.0001</td>
</tr>
<tr>
<td>Total fat (g)</td>
<td>53.1 ± 17.0</td>
<td>174.8 ± 27.1</td>
<td>0.0001</td>
</tr>
<tr>
<td>Saturated fat (g)</td>
<td>19.4 ± 7.9</td>
<td>78.4 ± 17.6</td>
<td>0.0001</td>
</tr>
<tr>
<td>Total fat (%E)</td>
<td>19.3 ± 4.1</td>
<td>43.2 ± 4.5</td>
<td>0.0001</td>
</tr>
<tr>
<td>Carbohydrate (%E)</td>
<td>60.8 ± 5.2</td>
<td>40.3 ± 8.5</td>
<td>0.0001</td>
</tr>
<tr>
<td>Protein (%E)</td>
<td>16.8 ± 3.0</td>
<td>14.6 ± 3.9</td>
<td>0.10</td>
</tr>
<tr>
<td>Saturated fat (%E)</td>
<td>7.0 ± 2.1</td>
<td>19.4 ± 3.8</td>
<td>0.0001</td>
</tr>
<tr>
<td>Monounsaturated fat (%E)</td>
<td>6.4 ± 1.5</td>
<td>14.3 ± 1.7</td>
<td>0.0001</td>
</tr>
<tr>
<td>Polyunsaturated fat (%E)</td>
<td>3.5 ± 0.8</td>
<td>5.4 ± 2.0</td>
<td>0.0018</td>
</tr>
<tr>
<td>P:S ratio</td>
<td>0.5 ± 0.2</td>
<td>0.3 ± 0.2</td>
<td>0.0009</td>
</tr>
<tr>
<td>Cholesterol (mg)</td>
<td>242 ± 93</td>
<td>518 ± 231</td>
<td>0.0002</td>
</tr>
<tr>
<td>Alcohol (%E)</td>
<td>3.9 ± 5.3</td>
<td>2.0 ± 2.5</td>
<td>0.23</td>
</tr>
<tr>
<td>Dietary fibre (g)</td>
<td>33.9 ± 7.8</td>
<td>32.1 ± 9.0</td>
<td>0.57</td>
</tr>
</tbody>
</table>

p-values are for differences between HC and HF as determined by independent two-tailed t-tests.
Table 4.4  Dietary intakes for HC and HF to week twelve of intervention, n=16 for HC and n=12 for HF. All values are means ± SD

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (KJ)</td>
<td>10381 ± 2491 ( + 1400KJ training foods)</td>
<td>15501 ± 3278</td>
<td>0.0001</td>
</tr>
<tr>
<td>Total fat (g)</td>
<td>51.4 ± 18.8</td>
<td>188.0 ± 44.2</td>
<td>0.0001</td>
</tr>
<tr>
<td>Saturated fat (g)</td>
<td>19.7 ± 8.8</td>
<td>89.0 ± 22.0</td>
<td>0.0001</td>
</tr>
<tr>
<td>Total fat (%E)</td>
<td>18.1 ± 4.5</td>
<td>44.9 ± 4.6</td>
<td>0.0001</td>
</tr>
<tr>
<td>Carbohydrate (%E)</td>
<td>62.3 ± 6.0</td>
<td>38.7 ± 6.7</td>
<td>0.0001</td>
</tr>
<tr>
<td>Protein (%E)</td>
<td>15.8 ± 2.4</td>
<td>14.1 ± 3.6</td>
<td>0.14</td>
</tr>
<tr>
<td>Saturated fat (%E)</td>
<td>6.8 ± 2.1</td>
<td>21.2 ± 2.2</td>
<td>0.0001</td>
</tr>
<tr>
<td>Monounsaturated fat (%E)</td>
<td>6.1 ± 1.7</td>
<td>14.6 ± 2.0</td>
<td>0.0001</td>
</tr>
<tr>
<td>Polyunsaturated fat (%E)</td>
<td>3.0 ± 1.0</td>
<td>4.9 ± 1.7</td>
<td>0.0009</td>
</tr>
<tr>
<td>P:S ratio</td>
<td>0.5 ± 0.2</td>
<td>0.2 ± 0.2</td>
<td>0.006</td>
</tr>
<tr>
<td>Cholesterol (mg)</td>
<td>241 ± 105</td>
<td>541 ± 220</td>
<td>0.0001</td>
</tr>
<tr>
<td>Alcohol (%E)</td>
<td>4.5 ± 5.4</td>
<td>2.2 ± 2.4</td>
<td>0.19</td>
</tr>
<tr>
<td>Dietary fibre (g)</td>
<td>35.4 ± 12.2</td>
<td>33.5 ± 9.6</td>
<td>0.65</td>
</tr>
</tbody>
</table>

p-values are for differences between HC and HF as determined by independent two-tailed t-tests.

Table 4.5  Dietary intakes for HC and HF to week sixteen of intervention, n=9 for HC and n=6 for HF. All values are means ± SD

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (KJ)</td>
<td>10607 ± 1888 (+1400KJ training foods)</td>
<td>16547 ± 2782</td>
<td>0.0004</td>
</tr>
<tr>
<td>Total fat (g)</td>
<td>48.5 ± 18.0</td>
<td>213.6 ± 35.8</td>
<td>0.0001</td>
</tr>
<tr>
<td>Saturated fat (g)</td>
<td>16.9 ± 8.0</td>
<td>97.4 ± 32.9</td>
<td>0.0001</td>
</tr>
<tr>
<td>Total fat (%E)</td>
<td>16.8 ± 4.9</td>
<td>48.7 ± 6.2</td>
<td>0.0001</td>
</tr>
<tr>
<td>Carbohydrate (%E)</td>
<td>60.5 ± 6.6</td>
<td>34.4 ± 7.4</td>
<td>0.0001</td>
</tr>
<tr>
<td>Protein (%E)</td>
<td>16.9 ± 2.7</td>
<td>14.2 ± 3.6</td>
<td>0.12</td>
</tr>
<tr>
<td>Saturated fat (%E)</td>
<td>5.8 ± 2.3</td>
<td>22.0 ± 6.1</td>
<td>0.0001</td>
</tr>
<tr>
<td>Monounsaturated fat (%E)</td>
<td>5.6 ± 1.8</td>
<td>16.4 ± 2.2</td>
<td>0.0001</td>
</tr>
<tr>
<td>Polyunsaturated fat (%E)</td>
<td>3.2 ± 0.9</td>
<td>5.7 ± 2.8</td>
<td>0.02</td>
</tr>
<tr>
<td>P:S ratio</td>
<td>0.6 ± 0.2</td>
<td>0.3 ± 0.2</td>
<td>0.04</td>
</tr>
<tr>
<td>Cholesterol (mg)</td>
<td>223 ± 70</td>
<td>602 ± 222</td>
<td>0.0001</td>
</tr>
<tr>
<td>Alcohol (%E)</td>
<td>6.5 ± 7.5</td>
<td>2.5 ± 2.0</td>
<td>0.24</td>
</tr>
<tr>
<td>Dietary fibre (g)</td>
<td>40.2 ± 13.1</td>
<td>33.4 ± 13.1</td>
<td>0.38</td>
</tr>
</tbody>
</table>

p-values are for differences between HC and HF as determined by independent two-tailed t-tests.
Figure 4.1  Mean macronutrient intakes as a percentage of total energy at baseline for HC and HF (±SD)

<table>
<thead>
<tr>
<th>Macronutrient</th>
<th>HC (n=20)</th>
<th>HF (n=14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat</td>
<td>*</td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*p=0.006
Figure 4.2  Mean macronutrient intakes as a percentage of total energy to week four of intervention for HC and HF (±SD)

Macronutrient

*p=0.0001
Figure 4.3  Mean macronutrient intakes as a percentage of total energy to week twelve of intervention for HC and HF (±SD)

Macronutrient

*p=0.0001
Figure 4.4 Mean macronutrient intakes as a percentage of total energy to week sixteen of intervention for HC and HF (±SD)

*p=0.0001
Figure 4.5  Mean fat intake as expressed in absolute amounts (g/day) for HC and HF at baseline and during the study period (± SD)

<table>
<thead>
<tr>
<th>Time (wks)</th>
<th>BL</th>
<th>4</th>
<th>12</th>
<th>16</th>
</tr>
</thead>
<tbody>
<tr>
<td>HC (n=20 to wk 12, n=15 at wk 16)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HF (n=14 to wk 12, n=7 at wk 16)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p=0.012  
**p=0.0001
Figure 4.6  Mean SAFA, PUFA, and MUFA intakes as percent of total fat for HC and HF at baseline (±SD)

<table>
<thead>
<tr>
<th>Type of fat</th>
<th>HC (n=20)</th>
<th>HF (n=14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAFA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PUFA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MUFA</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

% Total fat

Type of fat
Figure 4.7  Mean SAFA, PUFA, and MUFA intakes as a percentage of total fat for HC and HF at week four (±SD)

<table>
<thead>
<tr>
<th>Type of fat</th>
<th>HC (n=16)</th>
<th>HF (n=12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAFA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PUFA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MUFA</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p=0.0012
**p=0.0001
Figure 4.8  Mean SAFA, PUFA, and MUFA intakes as a percentage of total fat for HC and HF at week twelve (±SD)

Type of fat

* p=0.0008
** p=0.0001

HC (n=16)
HF (n=12)
Figure 4.9 Mean SAFA, PUFA, and MUFA intakes as a percentage of total fat for HC and HF at week sixteen (±SD)

* p=0.03
**p=0.011
Figure 4.10  Mean fibre intake (g) for HF and HC from baseline to week sixteen (±SD)
Figure 4.11  Mean alcohol intake (g) for HC and HF from baseline to week sixteen (±SD)

- HC (n= 20 to wk 12, n=15 at wk 16)
- HF (n= 14 to wk 12, n=7 at wk 16)
Figure 4.12  Mean dietary cholesterol intake for HC and HF from baseline to week sixteen (±SD)

* p<0.01  
**p<0.001
4.3 Laboratory Data

4.3.1 Total cholesterol

The mean total cholesterol (TC) (mean ± SD) at baseline was 4.72 ± 0.72mmol/L for HC and 5.02 ± 0.63mmol/L for HF (p=0.238) (Table 4.6).

The pooled changes for HC and HF show significant decreases in TC in both groups at week twelve (p=0.011) and week sixteen (p=0.0007) (Tables 4.13 & 4.14).

During the first twelve weeks of the study, TC in the HC group (n=20) decreased by 4.9% (0.23mmol/L), from 4.72 ± 0.72 to 4.49 ± 0.93mmol/L (p=0.050) (Table 4.7). A total decrease of 9.3% (0.44mmol/L), from 4.72 ± 0.72 to 4.28 ± 0.66mmol/L was present after sixteen weeks (n=15) (p=0.052) (Table 4.8).

After twelve weeks, mean TC concentrations for HF (n=14) decreased by 8.2% (0.41mmol/L), from 5.02 ± 0.63mmol/L to 4.61 ± 0.68mmol/L (p=0.033) (Table 4.9). After sixteen weeks of intervention (n=7), TC levels decreased 7.2% (0.36mmol/L) from 5.02 ± 0.63 to 4.65 ± 0.67mmol/L (p=0.046) (table 4.10).

No significant differences were present between the two diet groups throughout intervention (Figure 4.13). Changes in TC for HF did not differ from those in HC at week twelve (p=0.50) and week 16 (p=0.18) (Tables 4.11 & 4.12).

4.3.2 LDL cholesterol

LDL cholesterol at baseline was 2.84 ± 0.72mmol/L for HC and 3.01 ± 0.52mmol/L for HF (p=0.443) (Table 4.6).

Pooled changes for HC and HF show significant decreases in LDL-C for both groups at week twelve (p=0.013) and week sixteen (p=0.008) (Tables 4.13 & 4.14).

During the first twelve weeks of the study, LDL-C decreased 7.0% (0.20mmol/L) in HC, from 2.84 ± 0.72 to 2.64 ± 0.88mmol/L (p=0.064) (Table 4.7). At week sixteen LDL-C levels decreased 11.6% (0.33mmol/L) from 2.84 ± 0.72mmol/L at baseline to 2.51 ± 0.59mmol/L (p=0.071) (table 4.8).

In group HF there was a significant reduction in LDL-C of 9.3% (0.28mmol/L) from 3.01 ± 0.52 at baseline to 2.73 ± 0.50mmol/L at week
twelve (p=0.021) (Table 4.9). At week sixteen LDL-C remained significantly lower (9.0%) than baseline values at 2.74 ± 0.59mmol/L (p=0.028) (Table 4.10).

There were no significant differences in LDL-C between HC and HF throughout the study period (Figure 4.14). Changes in LDL-C for HF did not differ from HC at week twelve (p=0.669) or week sixteen (p=0.211) (Tables 4.11 & 4.12).

4.3.3 HDL cholesterol

Baseline concentrations of HDL-C were 1.32 ± 0.26mmol/L and 1.35 ± 0.22mmol/L for HC and HF respectively (p=0.702) (Table 4.6).

Pooled changes for HC and HF show significant increases in HDL-C for both groups at week sixteen (p=0.004), but not for week twelve (p=0.51) (Tables 4.13 & 4.14).

At week twelve there was a slight increase in HDL-C to 1.36 ± 0.34mmol/L in the HC group, however this was not statistically significant (Table 4.7). HDL concentration for HF at week twelve remained similar to baseline at 1.35 ± 0.35mmol/L (Table 4.9).

At week sixteen, significant rises in HDL-C occurred in both groups. In group HC, HDL-C increased 3% from 1.32 ± 0.26mmol/L to 1.36 ± 0.25mmol/L (p=0.026) (Table 4.8). HDL-C in HF increased 10.3% from a baseline value of 1.35 ± 0.22mmol/L to 1.50 ± 0.33mmol/L (p=0.029) (Table 4.10).

There were no significant differences in HDL cholesterol concentration between groups HC and HF during the study (Figure 4.15). Changes in HDL-C for HF did not differ from HC at week twelve (p=0.76) or week sixteen (p=0.45) (Tables 4.11 & 4.12).

4.3.4 HDL2 cholesterol

HDL2-C concentrations at baseline were 0.52 ± 0.15mmol/L and 0.57 ± 0.16mmol/L for HC and HF respectively (p=0.37) (Table 4.6).

Pooled changes for HC and HF show no changes in HDL2-C in either group during the study (Tables 4.13 &4.14).

In group HC, HDL2 levels increased slightly from baseline, 0.52 ± 0.15mmol/L to 0.55 ± 0.25mmol/L at week twelve. However, this difference was not statistically significant (p=0.33) (Table 4.7). In group HF, HDL2 concentrations at week twelve remained similar to baseline values (BL, 0.57 ± 0.16mmol/L; week twelve, 0.56 ± 0.16mmol/L) (p=0.46) (Table 4.9).
HDL\textsubscript{2} concentrations at week sixteen remained similar to baseline values for HC at 0.54 ± 0.17mmol/L (Table 4.8). HF concentrations increased slightly from baseline values of 0.57 ± 0.16mmol/L to 0.64 ± 0.20mmol/L, however, this difference was not statistically significant (p=0.26)(Table 4.10).

HDL\textsubscript{2} concentrations at weeks twelve and sixteen were not significantly different in the two diet groups (Figure 4.16). Changes in HDL\textsubscript{2}-C for HF did not differ from HC at week twelve (p=0.702) or week sixteen (p=0.983) (Tables 4.11 & 4.12).

### 4.3.5 HDL\textsubscript{3} cholesterol

HDL\textsubscript{3}-C levels at baseline for HC and HF were 0.80 ± 0.14 and 0.80 ± 0.11mmol/L respectively (Table 4.6).

Pooled changes for HC and HF show significant increases in HDL\textsubscript{3}-C concentrations in both groups at week sixteen (p=0.009) (Table 4.14), but not at week twelve.

HDL\textsubscript{3}-C concentrations in group HC at week twelve increased slightly from baseline to 0.83 ± 0.11mmol/L, however, this was not statistically significant. (p=0.12) (Table 4.7). HDL\textsubscript{3}-C concentrations for HF at week twelve did not change significantly from BL (BL, 0.80 ± 0.11mmol/L; week twelve, 0.80 ± 0.13) (Table 4.9).

At week sixteen there were significant increases in HDL\textsubscript{3} in both groups. HC concentrations increased 2.5%, from 0.80 ± 0.14mmol/L to 0.82 ± 0.12mmol/L (p=0.038) and HF concentrations also increased 2.5% from 0.80 ± 0.11mmol/L to 0.82 ± 0.15mmol/L (p=0.034) (Tables 4.8 & 4.10).

There were no significant differences in HDL\textsubscript{3} between HC and HF at any sampling period during the study (Figure 4.17). Changes in HDL\textsubscript{3}-C for HF were not significantly different from HC at week twelve (p=0.649) or week sixteen (p=0.497) (Tables 4.11 & 4.12).

### 4.3.6 Triglycerides

Plasma triglycerides (TG) concentrations at baseline were 1.26 ± 0.36mmol/L for HC and 1.27 ± 0.38mmol/L for HF (p=0.90) (Table 4.6).

Pooled changes for HC and HF show a significant reduction in TG in both groups at week sixteen (p=0.006) (Table 4.14), but not at week twelve.
At week twelve TG concentrations for HC decreased slightly from 1.26 ± 0.36mmol/L to 1.19 ± 0.45mmol/L, however this was not statistically significant (Table 4.7). HF concentrations decreased slightly from 1.27 ± 0.38mmol/L to 1.18 ± 0.48mmol/L, again this was not statistically significant (Table 4.9).

In group HC, TG concentrations at week sixteen decreased 23% (0.29mmol/L) to 0.97 ± 0.33mmol/L (p=0.032) (Table 4.8) and HF decreased 25% (0.32mmol/L) to 0.95 ± 0.26mmol/L (p=0.018) (Table 4.10).

There were no statistically significant differences in TG between HC and HF throughout the study (Figure 4.18). Changes in TG for HF did not differ from HC at week twelve (p=0.977) or week sixteen (p=0.495) (Tables 4.11 & 4.12).

Pearson correlation coefficient matrix showed a significant correlation between triglycerides concentrations at week twelve and total kilometres cycled/week to week twelve (p=0.004) and also week sixteen (p=0.010). There was also a significant correlation between triglycerides at week twelve and the average kilometres cycled/week to week twelve (p=0.004) and week sixteen (p=0.007).

### 4.3.7 Apolipoprotein A1

Apolipoprotein A1 (apo A1) levels at BL were 106.40 ± 17.94mg/dl and 106.33 ± 16.98mg/dl for HC and HF respectively (p=0.99) (Table 4.6).

Pooled changes for HC and HF show a significant increase in apo A1 concentrations in both groups at week twelve (p=0.015) (Table 4.13), but not at week sixteen.

Apo A1 levels in HC increased 8.7% (9.3mg/dl) from 106.40 ± 17.94mg/dl at baseline to 115.7 ± 16.4mg/dl at week twelve (p=0.023) (Table 4.7). HF apo A1 levels increased 5.6% (6mg/dl) from 106.33 ± 16.98mg/dl at baseline to 112.3 ± 12.9mg/dl at week twelve (p=0.07) (Table 4.9).

At week sixteen apo A1 levels for HC were slightly higher than baseline at 111.1 ± 13.4mg/dl, however this was not statistically significant (p=0.25) (Table 4.8). Apo A1 concentrations for HF at week sixteen were slightly higher than baseline levels at 115.1 ± 16.4mg/dl, however, this was not statistically significant (p=0.48) (Table 4.10).

There were no significant differences in apo A1 concentrations between groups HC and HF during the study (Figure 4.19). Changes in Apo A1
for HF did not differ from HC at week twelve (p=0.772) and week sixteen (p=0.673) (Tables 4.11 & 4.12).

There were significant positive correlations between apo A1 concentrations and average hours of endurance training per week at week twelve (0.032) and week sixteen (p=0.020).

4.3.8 Apolipoprotein B

Baseline concentrations of apolipoprotein B (apo B) were 76.80 ± 15.32mg/dl and 75.20 ± 17.94mg/dl for HC and HF respectively (p=0.78) (Table 4.6).

Pooled changes for HC and HF show a significant reduction in apo B concentration in both groups at week twelve (p=0.024) (Table 4.13).

At week twelve HC showed a significant reduction in apo B of 8.3% (6.4mg/dl) from 76.80 ± 15.32 to 70.4 ± 17.3mg/dl (p=0.013) (Table 4.7). HF levels decreased from 75.20 ± 17.94mg/dl to 73.1 ± 13.4mg/dl, which was not statistically significant (p=0.15) (Table 4.9).

Apo B levels at week sixteen were 75.8 ± 36.0mg/dl for HC which were not significantly different from baseline concentrations of 76.80 ± 15.32mg/dl (p=0.45) (Table 4.8). HF showed a significant reduction in apo B levels of 9.7% (7.3mg/dl) at week sixteen from 75.20 ± 17.94 to 67.9 ± 7.2mg/dl (p=0.020) (Table 4.10).

No significant differences in apo B concentrations were present between the two groups during the study (Figure 4.20). Changes in apo B concentrations for HF did not differ from HC at week twelve (p=0.540) or week sixteen (p=0.183) (Tables 4.11 & 4.12).

4.3.9 HDL/Total cholesterol ratio

At baseline the HDL/total cholesterol ratio was 0.29 ± 0.06 and 0.28 ± 0.06 for HC and HF respectively (p=0.63) (Table 4.6).

Pooled changes for HC and HF show significant increases in the HDL/TC ratio in both groups at week twelve (p=0.001) and week sixteen (p<0.0009) (Tables 4.13 & 4.14).

During the first twelve weeks of the study, the HDL/TC ratio increased in group HC from 0.29 ± 0.06 to 0.31 ± 0.07 (p=0.008) (Table 4.7). For HF this ratio increased significantly from 0.28 ± 0.06 to 0.30 ± 0.05 (p=0.01) (Table 4.9).

For HC, the ratio increased from 0.29 ± 0.06 to 0.32 ± 0.07 (p=0.003) at week sixteen (Table 4.8). For HF the ratio was increased to 0.31 ± 0.07 at week sixteen (p=0.001) (Table 4.10).
There were no statistically significant differences in the HDL-C/TC ratio between groups HC and HF during the study (Tables 4.11 & 4.12).

4.3.10 HDL/LDL cholesterol ratio

Baseline HDL/LDL cholesterol ratios were 0.49 ± 0.16 and 0.47 ± 0.12 for HC and HF respectively (p=0.56) (Table 4.6).

Pooled changes for HC and HF show significant increases in the HDL/LDL ratio in both groups at week twelve (p=0.003) and week sixteen (p=0.003) (Tables 4.13 & 4.14).

During the first twelve weeks of the study, the HDL/LDL ratio increased from 0.49 ± 0.16 to 0.57 ± 0.20 (p=0.01) in group HC (Table 4.7). The HDL/LDL ratio increased from 0.47 ± 0.12 to 0.51 ± 0.13 (p=0.017) in HF (Table 4.9).

At week sixteen the HDL/LDL cholesterol ratio for HC increased to 0.58 ± 0.20 (p=0.010) (Table 4.8). In HF, the ratio increased from 0.47 ± 0.12 to 0.54 ± 0.18 at week sixteen (p=0.002) (Table 4.10).

There were no differences in the HDL/LDL ratio between HC and HF during the study period. Changes in the ratio for HF did not differ from HC at week twelve (p=0.522) or week sixteen (p=0.91) (Tables 4.11 & 4.12).

Significant correlations were found between the HDL/LDL ratio at week sixteen and total hours of endurance training to week sixteen (p=0.037). A significant correlation was also present between this ratio and average hours of endurance training to week sixteen.
Table 4.6  Plasma lipid and lipoprotein profile of HC and HF at baseline, n=20 for HC and n=15 for HF. All values are means ± SD

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.72 ± 0.72</td>
<td>5.02 ± 0.63</td>
<td>0.24</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>2.84 ± 0.72</td>
<td>3.01 ± 0.52</td>
<td>0.44</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.32 ± 0.26</td>
<td>1.35 ± 0.22</td>
<td>0.70</td>
</tr>
<tr>
<td>HDL&lt;sub&gt;2&lt;/sub&gt; cholesterol (mmol/L)</td>
<td>0.52 ± 0.15</td>
<td>0.57 ± 0.16</td>
<td>0.37</td>
</tr>
<tr>
<td>HDL&lt;sub&gt;3&lt;/sub&gt; cholesterol (mmol/L)</td>
<td>0.80 ± 0.14</td>
<td>0.80 ± 0.11</td>
<td>0.89</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.26 ± 0.36</td>
<td>1.27 ± 0.38</td>
<td>0.90</td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>106.40 ± 17.94</td>
<td>106.33 ± 16.98</td>
<td>0.99</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>76.80 ± 15.32</td>
<td>75.20 ± 17.94</td>
<td>0.78</td>
</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.29 ± 0.06</td>
<td>0.28 ± 0.06</td>
<td>0.63</td>
</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.49 ± 0.16</td>
<td>0.47 ± 0.12</td>
<td>0.56</td>
</tr>
</tbody>
</table>

p-values are differences between HC and HF as determined by independent two-tailed t-tests.

Table 4.7  Plasma lipid and lipoprotein profile for HC at week twelve compared to baseline, n=20. All values are means ± SD

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>week 12</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.72 ± 0.79</td>
<td>4.48 ± 0.93</td>
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</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>2.84 ± 0.72</td>
<td>2.64 ± 0.88</td>
<td>0.064</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.32 ± 0.26</td>
<td>1.36 ± 0.34</td>
<td>0.25</td>
</tr>
<tr>
<td>HDL&lt;sub&gt;2&lt;/sub&gt; cholesterol (mmol/L)</td>
<td>0.52 ± 0.15</td>
<td>0.55 ± 0.25</td>
<td>0.33</td>
</tr>
<tr>
<td>HDL&lt;sub&gt;3&lt;/sub&gt; cholesterol (mmol/L)</td>
<td>0.80 ± 0.14</td>
<td>0.83 ± 0.11</td>
<td>0.12</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.26 ± 0.36</td>
<td>1.19 ± 0.45</td>
<td>0.28</td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>106.4 ± 17.9</td>
<td>115.7 ± 16.4</td>
<td>0.023</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>76.8 ± 15.3</td>
<td>70.4 ± 17.3</td>
<td>0.013</td>
</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.29 ± 0.06</td>
<td>0.31 ± 0.07</td>
<td>0.008</td>
</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.49 ± 0.16</td>
<td>0.58 ± 0.20</td>
<td>0.010</td>
</tr>
</tbody>
</table>

p-values are for differences between baseline and week 12 as determined by paired one tailed t-tests.
Table 4.8  Plasma lipid and lipoprotein profile for HC at week sixteen compared to baseline, n=15. All values are means ± SD

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>week 16</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.72 ± 0.79</td>
<td>4.28 ± 0.66</td>
<td>0.052</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>2.84 ± 0.72</td>
<td>2.51 ± 0.59</td>
<td>0.071</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.32 ± 0.26</td>
<td>1.36 ± 0.25</td>
<td>0.024</td>
</tr>
<tr>
<td>HDL&lt;sub&gt;2&lt;/sub&gt; cholesterol (mmol/L)</td>
<td>0.52 ± 0.15</td>
<td>0.54 ± 0.17</td>
<td>0.27</td>
</tr>
<tr>
<td>HDL&lt;sub&gt;3&lt;/sub&gt; cholesterol (mmol/L)</td>
<td>0.80 ± 0.14</td>
<td>0.82 ± 0.12</td>
<td>0.038</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.26 ± 0.36</td>
<td>0.97 ± 0.33</td>
<td>0.032</td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>106.4 ± 17.9</td>
<td>111.1 ± 13.4</td>
<td>0.25</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>76.8 ± 15.3</td>
<td>75.8 ± 36.0</td>
<td>0.45</td>
</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.29 ± 0.06</td>
<td>0.32 ± 0.07</td>
<td>0.003</td>
</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.49 ± 0.16</td>
<td>0.58 ± 0.20</td>
<td>0.010</td>
</tr>
</tbody>
</table>

p-values are for differences between baseline and week sixteen as determined by paired one-tailed t-tests.

Table 4.9  Plasma lipid and lipoprotein profile for HF at week twelve compared to baseline, n=14. All values are means ± SD

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>week 12</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>5.02 ± 0.63</td>
<td>4.61 ± 0.68</td>
<td>0.033</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>3.01 ± 0.52</td>
<td>2.73 ± 0.50</td>
<td>0.021</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.35 ± 0.52</td>
<td>1.35 ± 0.25</td>
<td>0.39</td>
</tr>
<tr>
<td>HDL&lt;sub&gt;2&lt;/sub&gt; cholesterol (mmol/L)</td>
<td>0.57 ± 0.16</td>
<td>0.56 ± 0.16</td>
<td>0.46</td>
</tr>
<tr>
<td>HDL&lt;sub&gt;3&lt;/sub&gt; cholesterol (mmol/L)</td>
<td>0.80 ± 0.11</td>
<td>0.80 ± 0.13</td>
<td>0.38</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.26 ± 0.36</td>
<td>1.18 ± 0.48</td>
<td>0.33</td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>106.3 ± 17.0</td>
<td>112.3 ± 12.9</td>
<td>0.07</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>75.2 ± 17.8</td>
<td>73.1 ± 13.4</td>
<td>0.15</td>
</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.28 ± 0.06</td>
<td>0.30 ± 0.05</td>
<td>0.010</td>
</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.47 ± 0.12</td>
<td>0.51 ± 0.13</td>
<td>0.017</td>
</tr>
</tbody>
</table>

p-values are for differences between baseline and week twelve as determined by paired one-tailed t-tests.
Table 4.10 Plasma lipid and lipoprotein profile for HF at week sixteen compared to baseline, n=7. All values are means ± SD

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>week 16</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>5.02 ± 0.63</td>
<td>4.64 ± 0.67</td>
<td>0.046</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>3.01 ± 0.52</td>
<td>2.74 ± 0.59</td>
<td>0.028</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.35 ± 0.52</td>
<td>1.50 ± 0.33</td>
<td>0.029</td>
</tr>
<tr>
<td>HDL2 cholesterol (mmol/L)</td>
<td>0.57 ± 0.16</td>
<td>0.64 ± 0.20</td>
<td>0.26</td>
</tr>
<tr>
<td>HDL3 cholesterol (mmol/L)</td>
<td>0.80 ± 0.11</td>
<td>0.86 ± 0.15</td>
<td>0.034</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.26 ± 0.36</td>
<td>0.95 ± 0.26</td>
<td>0.018</td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>106.3 ± 17.0</td>
<td>115.1 ± 8.9</td>
<td>0.48</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>75.2 ± 17.8</td>
<td>67.9 ± 7.2</td>
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</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.28 ± 0.06</td>
<td>0.31 ± 0.07</td>
<td>0.001</td>
</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.47 ± 0.12</td>
<td>0.54 ± 0.18</td>
<td>0.002</td>
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</table>

p-values are for differences between baseline and week sixteen as determined by paired one-tailed t-tests.
Table 4.11 Changes in plasma lipoproteins/lipids for HC and HF from baseline to week twelve of dietary intervention

<table>
<thead>
<tr>
<th></th>
<th>HC (Baseline)</th>
<th>HC (Week 12)</th>
<th>HF (Baseline)</th>
<th>HF (Week 12)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.72 ± 0.72</td>
<td>4.49 ± 0.93</td>
<td>5.02 ± 0.63</td>
<td>4.61 ± 0.68</td>
<td>0.50</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>2.84 ± 0.72</td>
<td>2.64 ± 0.88</td>
<td>3.01 ± 0.52</td>
<td>2.73 ± 0.50</td>
<td>0.67</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.32 ± 0.26</td>
<td>1.36 ± 0.34</td>
<td>1.35 ± 0.22</td>
<td>1.35 ± 0.35</td>
<td>0.76</td>
</tr>
<tr>
<td>HDL2 cholesterol (mmol/L)</td>
<td>0.52 ± 0.15</td>
<td>0.55 ± 0.25</td>
<td>0.57 ± 0.16</td>
<td>0.56 ± 0.16</td>
<td>0.71</td>
</tr>
<tr>
<td>HDL3 cholesterol (mmol/L)</td>
<td>0.80 ± 0.14</td>
<td>0.83 ± 0.11</td>
<td>0.80 ± 0.11</td>
<td>0.80 ± 0.13</td>
<td>0.65</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.26 ± 0.36</td>
<td>1.19 ± 0.45</td>
<td>1.27 ± 0.38</td>
<td>1.18 ± 0.48</td>
<td>0.98</td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>106.40 ± 17.94</td>
<td>115.7 ± 16.4</td>
<td>106.33 ± 16.98</td>
<td>112.3 ± 12.9</td>
<td>0.77</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>76.80 ± 15.32</td>
<td>70.4 ± 17.3</td>
<td>75.20 ± 17.94</td>
<td>73.1 ± 13.4</td>
<td>0.54</td>
</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.29 ± 0.06</td>
<td>0.31 ± 0.07</td>
<td>0.28 ± 0.06</td>
<td>0.30 ± 0.05</td>
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</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.49 ± 0.16</td>
<td>0.57 ± 0.20</td>
<td>0.47 ± 0.12</td>
<td>0.51 ± 0.13</td>
<td>0.52</td>
</tr>
</tbody>
</table>

p-values for significance of the difference in change between the two diet groups from baseline to week twelve.
Table 4.11  Changes in plasma lipoproteins/lipids for HC and HF from baseline to week twelve of dietary intervention

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th></th>
<th>HF</th>
<th></th>
<th>p-value</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>baseline</td>
<td>week 12</td>
<td>baseline</td>
<td>week 12</td>
<td></td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.72 ± 0.72</td>
<td>4.49 ± 0.93</td>
<td>5.02 ± 0.63</td>
<td>4.61 ± 0.68</td>
<td>0.50</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>2.84 ± 0.72</td>
<td>2.64 ± 0.88</td>
<td>3.01 ± 0.52</td>
<td>2.73 ± 0.50</td>
<td>0.67</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.32 ± 0.26</td>
<td>1.36 ± 0.34</td>
<td>1.35 ± 0.22</td>
<td>1.35 ± 0.35</td>
<td>0.76</td>
</tr>
<tr>
<td>HDL2 cholesterol (mmol/L)</td>
<td>0.52 ± 0.15</td>
<td>0.55 ± 0.25</td>
<td>0.57 ± 0.16</td>
<td>0.56 ± 0.16</td>
<td>0.71</td>
</tr>
<tr>
<td>HDL3 cholesterol (mmol/L)</td>
<td>0.80 ± 0.14</td>
<td>0.83 ± 0.11</td>
<td>0.80 ± 0.11</td>
<td>0.80 ± 0.13</td>
<td>0.65</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.26 ± 0.36</td>
<td>1.19 ± 0.45</td>
<td>1.27 ± 0.38</td>
<td>1.18 ± 0.48</td>
<td>0.98</td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>106.40 ± 17.94</td>
<td>115.7 ± 16.4</td>
<td>106.33 ± 16.98</td>
<td>112.3 ± 12.9</td>
<td>0.77</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>76.80 ± 15.32</td>
<td>70.4 ± 17.3</td>
<td>75.20 ± 17.94</td>
<td>73.1 ± 13.4</td>
<td>0.54</td>
</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.29 ± 0.06</td>
<td>0.31 ± 0.07</td>
<td>0.28 ± 0.06</td>
<td>0.30 ± 0.05</td>
<td>0.92</td>
</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.49 ± 0.16</td>
<td>0.57 ± 0.20</td>
<td>0.47 ± 0.12</td>
<td>0.51 ± 0.13</td>
<td>0.52</td>
</tr>
</tbody>
</table>

p-values for significance of the difference in change between the two diet groups from baseline to week twelve.
Table 4.12 Changes in plasma lipoproteins/lipids for HC and HF from baseline to week sixteen of dietary intervention

<table>
<thead>
<tr>
<th></th>
<th><strong>HC</strong></th>
<th></th>
<th></th>
<th><strong>HF</strong></th>
<th></th>
<th></th>
<th><strong>p-value</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>baseline</td>
<td>week 16</td>
<td>baseline</td>
<td>week 16</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.72 ± 0.72</td>
<td>4.28 ± 0.66</td>
<td>5.02 ± 0.63</td>
<td>4.65 ± 0.67</td>
<td>0.18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>2.84 ± 0.72</td>
<td>2.51 ± 0.59</td>
<td>3.01 ± 0.52</td>
<td>2.74 ± 0.59</td>
<td>0.21</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.32 ± 0.26</td>
<td>1.36 ± 0.25</td>
<td>1.35 ± 0.22</td>
<td>1.50 ± 0.33</td>
<td>0.45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL2 cholesterol (mmol/L)</td>
<td>0.52 ± 0.15</td>
<td>0.54 ± 0.17</td>
<td>0.57 ± 0.16</td>
<td>0.64 ± 0.20</td>
<td>0.98</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL3 cholesterol (mmol/L)</td>
<td>0.80 ± 0.14</td>
<td>0.82 ± 0.12</td>
<td>0.80 ± 0.11</td>
<td>0.82 ± 0.15</td>
<td>0.50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.26 ± 0.36</td>
<td>0.97 ± 0.33</td>
<td>1.27 ± 0.38</td>
<td>0.75 ± 0.26</td>
<td>0.50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>106.40 ± 17.94</td>
<td>111.1 ± 13.4</td>
<td>106.33 ± 16.98</td>
<td>115 ± 8.9</td>
<td>0.67</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>76.80 ± 15.32</td>
<td>75.8 ± 36.0</td>
<td>75.20 ± 17.94</td>
<td>67.9 ± 7.2</td>
<td>0.18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.29 ± 0.06</td>
<td>0.32 ± 0.07</td>
<td>0.28 ± 0.06</td>
<td>0.31 ± 0.07</td>
<td>0.49</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.49 ± 0.16</td>
<td>0.58 ± 0.20</td>
<td>0.47 ± 0.12</td>
<td>0.54 ± 0.18</td>
<td>0.91</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

p-values for significance of the difference in change between the two diet groups from baseline to week sixteen.
Table 4.13  Pooled plasma lipid and lipoprotein profiles for HC and HF demonstrating the change pooled over both groups from baseline to week twelve of intervention, n=34.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Week 12</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.84</td>
<td>4.54</td>
<td>0.011</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>2.91</td>
<td>2.67</td>
<td>0.013</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.33</td>
<td>1.35</td>
<td>0.508</td>
</tr>
<tr>
<td>HDL2 cholesterol (mmol/L)</td>
<td>0.54</td>
<td>0.55</td>
<td>0.791</td>
</tr>
<tr>
<td>HDL3 cholesterol (mmol/L)</td>
<td>0.80</td>
<td>0.82</td>
<td>0.333</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.25</td>
<td>1.19</td>
<td>0.479</td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>105.8</td>
<td>114.3</td>
<td>0.015</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>76.8</td>
<td>71.5</td>
<td>0.024</td>
</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.28</td>
<td>0.30</td>
<td>0.001</td>
</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.48</td>
<td>0.54</td>
<td>0.003</td>
</tr>
</tbody>
</table>

p-values for the significance of change pooled over both diets.

Table 4.14  Pooled plasma lipid and lipoprotein profiles for HC and HF demonstrating the change pooled over both groups from baseline to week sixteen of intervention, n=34.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Week 16</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.72</td>
<td>4.40</td>
<td>0.007</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>2.84</td>
<td>2.58</td>
<td>0.008</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.31</td>
<td>1.41</td>
<td>0.004</td>
</tr>
<tr>
<td>HDL2 cholesterol (mmol/L)</td>
<td>0.54</td>
<td>0.57</td>
<td>0.363</td>
</tr>
<tr>
<td>HDL3 cholesterol (mmol/L)</td>
<td>0.78</td>
<td>0.84</td>
<td>0.009</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.23</td>
<td>0.96</td>
<td>0.006</td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>109.5</td>
<td>112.3</td>
<td>0.650</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>77.4</td>
<td>73.4</td>
<td>0.233</td>
</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.28</td>
<td>0.32</td>
<td>0.000</td>
</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.49</td>
<td>0.57</td>
<td>0.003</td>
</tr>
</tbody>
</table>

p-values for the significance of change pooled over both diets.
Figure 4.13  Mean total cholesterol for HC and HF from baseline to week 16 of intervention (±SEM).
Figure 4.14  Mean LDL cholesterol for HC and HF from baseline to week 16 of intervention (±SEM)

HC (n=20 to wk12, n=15 at wk 16)

HF (n= 14 to wk 12, n=7 at wk 16)
Figure 4.15  Mean HDL cholesterol for HC and HF from baseline to week 16 of intervention (±SEM).
Figure 4.16 Mean HDL2 cholesterol for HC and HF from baseline to week 16 of intervention (±SEM)
Figure 4.17  Mean HDL3 cholesterol for HC and HF from baseline to week 16 of intervention (±SEM).
Figure 4.18  Mean plasma triglycerides for HC and HF form baseline to week 16 of intervention (±SEM).
Figure 4.19  Mean Apo A1 for HC and HF from baseline to week 16 of intervention (±SEM)

- HC (n=20 to wk 12, n=15 to wk 16)
- HF (n=14 to wk 12, n=7 at wk 16)
Figure 4.20 Mean apo B for HC and HF from baseline to week 16 of intervention (±SEM).
4.4 Anthropometry

4.4.1 Body weight

At baseline the mean (±SD) weight for HC was 76.51 ± 6.44kg and 73.06 ± 6.22 kg for HF (p=0.122) (Table 4.15).

Pooled changes for HC and HF showed no significant changes in body weight in either group at week twelve (p=0.097) and week sixteen (p=0.295).

At week twelve mean body weight had decreased 0.96kg (1.25%) from 76.51 ± 6.44kg to 75.55 ± 6.63kg in HC, however, this was not statistically significant (p=0.07) (Table 4.16). For HF, body weight decreased 0.20kg (0.27%) from 73.06 ± 6.22kg to 72.86 ± 6.81kg. This difference was not statistically significant (p=0.16) (Table 4.17).

At week sixteen mean body weight decreased 0.29kg, from 76.51 ± 6.44kg to 76.22 ± 6.52kg (p=0.18) in HC (n=15). In HF (n=7) body weight decreased 1.41kg, from 73.06 ± 6.22kg to 71.65 ± 7.53kg, however, again this difference was not statistically significant (p=0.22).

Changes in body weight were not significantly different between groups HC and HF at week twelve (p=0.525) or week sixteen (p=0.927) (Table 4.18).

4.4.3 Total body fat

Total body fat (as estimated by the sum six skinfolds, S6SF) was estimated at 59.35mm for HC and 45.73mm for HF (p=0.023) (Table 4.19).

Pooled changes for HC and HF show no significant changes in S6SF to week twelve (p=0.924).

At week twelve S6SF decreased slightly from 59.35mm to 55.44mm, however, this difference was not statistically significant (p=0.23) (Table 4.16). S6SF in HF increased slightly from 45.73mm to 48.46mm at week twelve, however, this difference was not statistically significant (p=0.12) (Table 4.17).

Changes in total body fat were not significantly different between groups HC and HF (p=0.28) (Table 4.18).
Table 4.15  Body weight (kg) for HC and HF at baseline and during intervention. All values are means ± SD.

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>76.51 ± 6.44</td>
<td>73.06 ± 6.22</td>
<td>0.12</td>
</tr>
<tr>
<td>Week 4</td>
<td>76.12 ± 6.68</td>
<td>73.26 ± 6.60</td>
<td>0.21</td>
</tr>
<tr>
<td>Week 8</td>
<td>76.38 ± 7.01</td>
<td>73.35 ± 6.57</td>
<td>0.20</td>
</tr>
<tr>
<td>Week 12</td>
<td>75.55 ± 6.63</td>
<td>72.86 ± 6.81</td>
<td>0.26</td>
</tr>
<tr>
<td>Week 16</td>
<td>76.22 ± 6.52</td>
<td>71.65 ± 7.53</td>
<td>0.16</td>
</tr>
</tbody>
</table>

P-values are for differences between HC and HF as determined by independent two-tailed t-tests.

Table 4.16  Mean values (± SD) of body weight, BMI, and total body fat as estimated by the sum of six skinfolds (S6SF) for HC after twelve weeks of dietary intervention compared to baseline.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Week 12</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight (kg)</td>
<td>76.51 ± 6.44</td>
<td>75.55 ± 6.63</td>
<td>0.07</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>24.8 ± 1.9</td>
<td>24.5 ± 2.2</td>
<td>0.05</td>
</tr>
<tr>
<td>Total body fat (S6SF mm)</td>
<td>59.4 ± 18.8</td>
<td>55.4 ± 14.5</td>
<td>0.23</td>
</tr>
</tbody>
</table>

P-values are for differences between baseline and week twelve as determined by paired one-tailed t-tests.

Table 4.17  Mean values (± SD) of body weight, BMI, and total body fat as estimated by the sum of six skinfolds (S6SF) for HF after twelve weeks of dietary intervention compared to baseline.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Week 12</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight (kg)</td>
<td>73.06 ± 6.22</td>
<td>72.86 ± 6.81</td>
<td>0.16</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.9 ± 1.7</td>
<td>23.8 ± 1.7</td>
<td>0.10</td>
</tr>
<tr>
<td>Total body fat (S6SF mm)</td>
<td>45.7 ± 13.4</td>
<td>48.5 ± 13.0</td>
<td>0.12</td>
</tr>
</tbody>
</table>

P-values are for differences between baseline and week twelve as determined by paired one-tailed t-tests.
Table 4.18  Changes in body weight and total body fat for HC and HF from baseline to week twelve of dietary intervention

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th></th>
<th>HF</th>
<th></th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>week 12</td>
<td>Baseline</td>
<td>week 12</td>
<td></td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>76.51 ± 6.44</td>
<td>75.55 ± 6.63</td>
<td>73.06 ± 6.22</td>
<td>72.86 ± 6.81</td>
<td>0.53</td>
</tr>
<tr>
<td>Total body fat (S6SF)</td>
<td>59.4 ± 18.8</td>
<td>55.4 ± 14.5</td>
<td>45.7 ± 13.4</td>
<td>48.5 ± 13.0</td>
<td>0.28</td>
</tr>
</tbody>
</table>

p-values for significance of the difference in change between the two diet groups from baseline to week twelve.
Table 4.19  Total body fat as estimated by the sum of six skinfolds (mm) for HC and HF at baseline and week 12 of intervention. All values are means ± SD.

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>59.4 ± 18.8</td>
<td>45.7 ± 13.4</td>
<td>0.023</td>
</tr>
<tr>
<td>week 12</td>
<td>55.4 ± 14.5</td>
<td>48.5 ± 13.0</td>
<td>0.213</td>
</tr>
</tbody>
</table>

p-values are for differences between HC and HF as determined by independent two-tailed t-tests.
4.5 Physical Fitness

4.5.1 Maximum Aerobic Power

At baseline, mean values for VO2max were 61.38 ± 8.96ml/min/kg (4.62 ± 0.51L/min) for HC and 66.65 ± 7.86ml/min/kg (4.83 ± 0.70L/min) for HF (p=0.079, p=0.326) (Table 4.20).

Pooled changes for HC and HF show no changes in VO2max (as expressed in ml/min/kg) to week twelve (p=0.293) in either group, but a slight increase in VO2max (as expressed in L/min) to week twelve (p=0.051) in both groups (Table 4.24).

At week twelve, VO2max levels for HC increased only slightly from 61.38 ± 8.96ml/min/kg (4.62 ± 0.51L/min) to 61.34 ± 8.48ml/min/kg (4.70 ± 0.49L/min), however these differences were no statistically significant (p=0.38 (when expressed as ml/min/kg), p=0.17 (when expressed as L/min)) (Table 4.22).

For HF the mean VO2max levels increased from 66.65 ± 7.85ml/min/kg (4.83 ± 0.70L/min) at baseline to 66.93 ± 8.34ml/min/kg (4.91 ± 0.84L/min) at week twelve (p=0.07 (when expressed as ml/min/kg), p=0.02 (when expressed as L/min) (Table 4.23).

Changes in VO2max were not significantly different between groups HC and HF (p=0.550 (when expressed as ml/min/kg), p=0.516 (when expressed as L/min) (Table 4.25).

4.5.2 Workload

The average workload attained at baseline was 358 ± 63Watts for HC and 383 ± 56Watts for HF (p=0.218) (Table 4.20).

Pooled changes for HC and HF show significant increases in maximum workload in both at week twelve (p=0.001) (Table 4.24).

The mean workload for HC increased 7.5% (27Watts) from 358 ± 63Watts to 385 ± 65Watts (p=0.006) (Table 4.22). For HF the mean workload increased from 383 ± 56Watts to 391 ± 68Watts (p=0.069) (Table 4.23).

Changes in maximum workload were not significantly different between groups HC and HF (p=0.245) (Table 4.25).
4.5.3 Time to exhaustion

At baseline the mean times to exhaustion for HC and HF were 9.1 ± 2.0 minutes and 9.6 ± 1.7 minutes respectively (p=0.48) (Table 4.20).

After twelve weeks of training both groups HC and HF were able to cycle for a greater duration of time before they became exhausted. Time to exhaustion during the VO2max test at week twelve for HC increased from 9.1 ± 2.0 min to 10.0 ± 1.8 min (p=0.0003) (Table 4.22). For HF time to exhaustion following twelve weeks of intervention increased from 9.6 ± 1.7 min to 10.3 ± 1.9 minutes (p=0.036) (Table 4.23).

There were no significant differences in times to exhaustion between the two diet groups at the exercise test at week twelve (p=0.62) (Table 2.21).
Table 4.20  Mean values (± SD) of maximal oxygen uptake (VO$_2$max),
maximum workload and time to exhaustion during exercise test
for HC and HF at baseline.

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO$_2$max (ml/min/kg)</td>
<td>61.38 ± 8.96</td>
<td>66.65 ± 7.86</td>
<td>0.079</td>
</tr>
<tr>
<td>VO$_2$max (L/min)</td>
<td>4.62 ± 0.51</td>
<td>4.83 ± 0.70</td>
<td>0.326</td>
</tr>
<tr>
<td>Maximum workload (Watts)</td>
<td>358 ± 63</td>
<td>385 ± 56</td>
<td>0.218</td>
</tr>
<tr>
<td>Time to exhaustion (min)</td>
<td>9.1 ± 2.0</td>
<td>9.6 ± 1.7</td>
<td>0.476</td>
</tr>
</tbody>
</table>

p-values for differences HC and HF as determined by independent two-tailed t-tests.

Table 4.21  Mean values (± SD) of maximal oxygen uptake (VO$_2$max),
maximum workload and time to exhaustion during exercise test
for HC and HF after twelve weeks of dietary intervention.

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO$_2$max (ml/min/kg)</td>
<td>61.34 ± 8.48</td>
<td>66.93 ± 8.34</td>
<td>0.12</td>
</tr>
<tr>
<td>VO$_2$max (L/min)</td>
<td>4.70 ± 0.49</td>
<td>4.91 ± 0.84</td>
<td>0.40</td>
</tr>
<tr>
<td>Maximum workload (Watts)</td>
<td>385 ± 65</td>
<td>391 ± 68</td>
<td>0.83</td>
</tr>
<tr>
<td>Time to exhaustion (min)</td>
<td>10.0 ± 1.8</td>
<td>10.3 ± 1.9</td>
<td>0.62</td>
</tr>
</tbody>
</table>

p-values for differences HC and HF as determined by independent two-tailed t-tests.

Table 4.22  Mean values (± SD) of maximal oxygen uptake (VO$_2$max),
maximum workload and time to exhaustion during exercise test
after twelve weeks of intervention compared to baseline for HC.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Week 12</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO$_2$max (ml/min/kg)</td>
<td>61.38 ± 8.96</td>
<td>61.34 ± 8.48</td>
<td>0.376</td>
</tr>
<tr>
<td>VO$_2$max (L/min)</td>
<td>4.62 ± 0.51</td>
<td>4.70 ± 0.49</td>
<td>0.171</td>
</tr>
<tr>
<td>Maximum workload (Watts)</td>
<td>358 ± 63</td>
<td>385 ± 65</td>
<td>0.0006</td>
</tr>
<tr>
<td>Time to exhaustion (min)</td>
<td>9.1 ± 2.0</td>
<td>10.0 ± 1.8</td>
<td>0.0003</td>
</tr>
</tbody>
</table>

p-values are for differences between baseline and week twelve as determined by paired one-tailed t-tests.
Table 4.23  Mean values (± SD) of maximal oxygen uptake (VO$_2$max), maximum workload and time to exhaustion during exercise test after twelve weeks of intervention compared to baseline for HF.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Week 12</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO$_2$max (ml/min/kg)</td>
<td>66.65 ± 7.86</td>
<td>66.93 ± 8.34</td>
<td>0.072</td>
</tr>
<tr>
<td>VO$_2$max (L/min)</td>
<td>4.83 ± 0.70</td>
<td>4.91 ± 0.84</td>
<td>0.021</td>
</tr>
<tr>
<td>Maximum workload (Watts)</td>
<td>385 ± 56</td>
<td>391 ± 68</td>
<td>0.069</td>
</tr>
<tr>
<td>Time to exhaustion (min)</td>
<td>9.6 ± 1.7</td>
<td>10.3 ± 1.9</td>
<td>0.036</td>
</tr>
</tbody>
</table>

p-values are for differences between baseline and week twelve as determined by paired one-tailed t-tests.

Table 4.24  Pooled values of maximal oxygen uptake (VO$_2$max), maximum workload and time to exhaustion during exercise test for HC and HF demonstrating the change pooled over both diet groups from baseline to week twelve.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Week 12</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO$_2$max (ml/min/kg)</td>
<td>62.93</td>
<td>63.78</td>
<td>0.293</td>
</tr>
<tr>
<td>VO$_2$max (L/min)</td>
<td>4.67</td>
<td>4.78</td>
<td>0.051</td>
</tr>
<tr>
<td>Maximum workload (Watts)</td>
<td>370</td>
<td>388</td>
<td>0.001</td>
</tr>
</tbody>
</table>

p-values are for the significance of change pooled over both diet groups.
Table 4.25  Changes in VO2max and maximum workload attained during exercise testing for HC and HF from baseline to week twelve of dietary intervention

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>week 12</td>
<td>Baseline</td>
</tr>
<tr>
<td>VO2max (ml/min/kg)</td>
<td>61.38 ± 8.96</td>
<td>61.34 ± 8.48</td>
<td>66.65 ± 7.86</td>
</tr>
<tr>
<td>VO2max (L/min)</td>
<td>4.62 ± 0.51</td>
<td>4.70 ± 0.49</td>
<td>4.83 ± 0.70</td>
</tr>
<tr>
<td>Max work load (Watts)</td>
<td>358 ± 63</td>
<td>385 ± 65</td>
<td>385 ± 56</td>
</tr>
</tbody>
</table>

p-values for significance of the difference in change between the two diet groups from baseline to week twelve.
4.5.4 Physical training

The amount of training performed by the two groups was similar. Group HF covered a greater distance cycle training per week compared to HC, however, group HC on average performed more hours of other endurance types of training.

4.5.4.1 Weekly kilometres cycled

Up to week twelve mean bicycle training was 178.9 ± 106.7km/wk (range: 0-287km/wk) for HC and 275.7 ± 186km/wk (range: 0-1120km/wk) for HF (p=0.132) (Table 2.26).

The average kilometres cycled per week from week thirteen to week sixteen were 179.8km/wk (range: 0-460km/wk) for HC and 274.1km/wk (range: 0-700km/wk) for HF (p=0.134) (Table 4.27).

A trend for an increase in km cycled per week was present during the study period in both groups.

4.5.4.2 Hours of other endurance training

Endurance training other than cycling was performed by a number of study subjects. Other training included running, kayaking and swimming.

Up to week twelve the average hours of “other endurance training” were 3.1 ± 4.15hrs/wk (range: 0-25hrs/wk) for HC and 1.5 ± 2.58hrs/wk (range: 0-30hrs/wk) for HF (p=0.269) (Table 4.26).

The average hours of “other endurance training” from week thirteen to week sixteen were 3.18 ± 4.18hrs/wk (range: 0-11.8hrs/wk) for HC and 1.50 ± 2.57hrs/wk (range: 0-3hrs/wk) for HF (p=0.249) (Table 4.27).
Table 4.26  Mean values (± SD) for cycle training kilometres and hours of other endurance training per week for HC and HF during the first twelve weeks of intervention. Ranges are shown in ( ).

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>km cycled/ week</td>
<td>178.9 ± 106.7 (0 - 287)</td>
<td>275.7 ± 186 (0 - 1120)</td>
<td>0.132</td>
</tr>
<tr>
<td>Hours of other endurance training</td>
<td>3.1 ± 4.15 (0 - 25)</td>
<td>1.5 ± 2.58 (0 - 30)</td>
<td>0.269</td>
</tr>
</tbody>
</table>

Table 4.27  Mean values (± SD) for cycle training kilometres and hours of other endurance training per week for HC and HF during the additional four weeks of the intervention period (weeks thirteen to sixteen). Ranges are shown in ( ).

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>km cycled/ week</td>
<td>179.8 ± 106.2 (0 - 460)</td>
<td>274.1 ± 181.1 (0 - 700)</td>
<td>0.134</td>
</tr>
<tr>
<td>Hours of other endurance training</td>
<td>3.18 ± 4.18 (0 - 11.8)</td>
<td>1.50 ± 2.57 (0 - 3)</td>
<td>0.249</td>
</tr>
</tbody>
</table>
5 Discussion

5.1 Dietary intervention

Few studies have reliably investigated the combined effects of diet and exercise on plasma lipids and lipoproteins.

The present study differs from previous research in duration, sample size and composition of dietary intervention. Many previous studies investigating both dietary and exercise effects on blood lipids have been descriptive or retrospective rather than involving a specific intervention (Blair, et al. 1981). Dietary assessment in some of the previous research have involved techniques less accurate than the multi-day food record method.

To allow for true adaptation to a high fat diet it has been suggested that a period in excess of six to twenty weeks is required (Hammel, et al. 1977; Phinney, et al. 1983a). Previous studies have involved periods of eight (Ekstedt, et al. 1991) to twenty-eight days (Lukaski, et al. 1984; Thompson, et al. 1984b). The duration of the present study was twelve to sixteen weeks as a compromise to maximise both dietary compliance and metabolic adaptation. A parallel study design was used because of the duration of dietary intervention.

Previous studies have been limited by the number of subjects studied and therefore unable to allow for strong statistical power. For example Mann et al (1955) employed three subjects, as did Lukaski et al (1984), and eight cross-country skiers were involved in the study by Ekstedt et al (1991). The present study employed thirty-five endurance trained cyclists.

Dietary intervention in the present study involved macronutrients proportions within a realistic range. The high carbohydrate diet comprised a daily intake of approximately 20% (51g fat) of energy from fat and 65% from carbohydrate. The high fat diet comprised approximately 45-50% (192g fat) of energy from fat and 35% from carbohydrate. Both diets consisted of approximately 15% of energy derived from protein.
The high carbohydrate diet was based on current recommendations for endurance athletes. The high fat diet, in contrast to other studies which have used extreme dietary proportions, was within a more realistic range and therefore able to be tolerated for longer periods of time. Other studies have used fat intakes as high as 69-90% of total energy (Christensen and Hansen 1938; Thompson, et al. 1984b). Such extreme amounts of fat may be poorly tolerated and unrealistic for any length of time.

The dietary proportions employed in the present study were within ranges that could be adequately met by free-living individuals. The diets were well tolerated for a duration of twelve-sixteen weeks.

It was noted that there was a significant difference in reported energy intake between the two groups, with HC consuming significantly less energy than HF. Since no significant differences in physical exercise were reported between the two groups and no apparent weight loss was evident in HC, I can conclude that HC under-reported their energy intakes and their energy intakes were more likely to be in the range consumed by those in the HF diet group. This under-reporting phenomenon could be attributed to several factors. Firstly, the high carbohydrate confectionary foods given to this group were less frequently recorded than were the high fat confectionary bars given to the HF diet group. This may be due to the nature of the foods ie. a chocolate bar (given to HF) seems more substantial and more likely to be recorded than a handful of sweets (given to HC). Furthermore, the HC confectionary was consumed more in a grazing manner ie. a handful, now and then during the day. Thus, subjects are more likely not to record this food to the same extent as the foods given to HF. Another factor that could have contributed to the lower reported intake of HC is the under-reporting of high energy-high carbohydrate supplements.

5.1.2 Dietary Assessment

Dietary intake in the present study was assessed by multiple five-day food diaries. The food record technique is most accurate of the dietary assessment techniques. It differs from the diet recall and food frequency questionnaire methods, in that it does not suffer from memory distortion.

The number of days required to classify 80% of subjects into tertiles of nutrient intake with 95% confidence is estimated to be approximately three to seven days for energy; five to seven days for protein, five to nine days for fat; and two to four days for carbohydrate (Bingham 1987; Bingham, et al. 1981; Gardner and Heady 1973; Liu, et al. 1978; Marr and Heady 1986). In the present study, where the objective was to determine the
intake of the macronutrients, a duration of approximately three to seven days was thought to be sufficient to estimate habitual intake to the degree of accuracy that will not result in a tedious procedure and thus low compliance rates. Many researchers employ a three or four day food record. A five day record (including three week days and two weekend days) was used in this study because the dietary intakes of athletes can vary significantly during the weekend days with competition and training commitments. Weekend eating patterns regarding fat intake has been observed to be different from weekday patterns (Maciejko, et al. 1983). It is important to measure both weekdays and weekend days to capture systematic variation associated with the day of the week (Tarasuk and Beaton 1992). A duration of five days recording (three week days and two weekend days) for the population I studied was believed to be a more accurate measure. Performing multiple five food records was believed to result in greater accuracy.

It was apparent that the athletes in this study underestimated their energy intakes. This was particularly evident in the high carbohydrate diet group. The subjects often estimated portion sizes by photographs supplied in the diet record booklets. This may not be an appropriate method of estimation of portion sizes for athletes whose energy intakes are large. The photos may have been misleading, in that the portion sizes did not extend to the amounts consumed by physically active individuals. A weighed inventory would be highly recommended for future research assessing the dietary intakes of endurance athletes. In addition, it should be emphasised to the athlete to include all training foods into the food records. It is believed that in the present study, training foods were often omitted from food diaries.

Previous studies examining the interaction between diet exercise and lipoproteins have been flawed with inappropriate dietary assessment. The twenty-four recall which involves past intakes is susceptible to memory distortion and has generally been shown to underestimate usual intake. Pekkarinen et al (1967) who studied the intakes of people in rural Finland found that the twenty-four hour recall gave calculations of nutrients which were one-third of that actually consumed, whereas others gave values three-times that ingested. Karvetti et al (1992) demonstrated that the twenty-four hour recall was considerably less accurate than the record measure. The percentage of foodstuffs omitted in the recall were 10-40% compared to 5-15% in the food diary. Correlation coefficients for nutrient intakes ranged from 0.58 to 0.74 for the recall versus observation, and from 0.75 to 0.92 for food diary versus observation.
Baghurst and Baghurst (1981) stated in a review of dietary assessment that with one or two exceptions, all studies of twenty-four hour recalls have been shown to grossly underestimate actual intake, making the twenty-four hour recall not only an unreliable method for the measurement of individual, but also for group intake.

Another form of dietary assessment that has been employed to determine habitual intake of athletes is the food frequency questionnaire. This is a recall technique and so suffers from memory distortion. The food frequency questionnaire usually involves a self-administered questionnaire or interviews to determine the frequency of consumption of a specified range of foods/drinks. This method is largely qualitative. However, quantitative aspects can be built into the technique. Quantitative aspects may involve comparisons with standardised serving sizes, the use of photographs or food models. This method of assessment was developed as a quick method that could be administered to large numbers of people with relatively high response rates. The food frequency questionnaire is an appropriate method for large scale studies where the aims are to assess mean group intake or to place individuals into broad categories of nutrient intake; or where descriptions of food patterns are needed. It is not an appropriate technique when the aim is to accurately assess absolute nutrient intakes of individuals. Here multiple food records are necessary.

Therefore, when assessing the habitual intakes of endurance athletes it is recommended that multiple five day food dairies are utilised. A weighed inventory is an important aspect of this assessment and emphasis should be placed on recording foods consumed while training.

5.2 Laboratory Data

5.2.1 Total cholesterol

The consumption of a high fat diet by the general population has been associated with an increase in plasma cholesterol levels and thus the subsequent increase in the risk of coronary heart disease and atherosclerosis in general (Lipids Research Clinics Program 1984; Lipids Research Clinics Program 1984b).

However, in the present study plasma total cholesterol levels of endurance cyclists consuming a high fat diet did not differ significantly from those consuming a high carbohydrate diet. In fact, total cholesterol
decreased significantly in both diet groups throughout the twelve to sixteen week study.

The effect of exercise on plasma total cholesterol concentration is unclear. Many studies indicate that no significant differences are present between those who are physically active and those who are sedentary (Haskell, et al. 1980; Keys 1970; Keys, et al. 1965).

The endurance cyclists in the present study had an average plasma total cholesterol concentration of 4.84mmol/l. This is 17% lower than that of the general population of New Zealand (Life in New Zealand Survey (LINZS)) where the mean cholesterol level for males overall was 5.8mmol/l (Mann, et al. 1991). This suggests that endurance athletes, or at least the endurance cyclists in this study, have lower total cholesterol levels than the general population.

Studies indicating no difference in total cholesterol concentrations with physical training may not have produced a level of physical exercise sufficient to demonstrate a lipid lowering affect. The cyclists in the present study were involved in large amounts of physical training. It has been suggested that a threshold of exercise in the vicinity of 10-15miles/week running is required to initiate a reduction in total cholesterol (Superko 1991). A report by the Royal College of Physicians quoted thresholds of an additional activity level of 2000kcal (which amounts to approximately five hours of brisk walking) per week or twice weekly vigorous aerobic activity involving peaks of energy expenditure of around 420kcal per hour (Royal College of Physicians 1991). The cyclists in this study averaged 230km/wk, with a maximum of 1120km/wk for one cyclist. Thus, the suggested threshold for producing beneficial changes to cholesterol concentrations, was exceeded by these cyclists. Physical training increased throughout the study period which could explain the decrease in total cholesterol apparent in both diet groups. Several longitudinal studies have demonstrated reductions in total cholesterol with endurance training (Goodyear, et al. 1990; Lipson, et al. 1980; Peltonen, et al. 1981).

Measurements of total cholesterol also include HDL cholesterol which has been found to be elevated in endurance athletes compared to non-athletes. Thus, higher concentrations of HDL cholesterol may lessen the reduction in total cholesterol levels. This may in part explain, why it is sometimes evident that total cholesterol is no lower in endurance athletes as compared to sedentary individuals.

There is limited research examining the effects of both diet and exercise on plasma total cholesterol. The results of this study indicate that exercise has a more important influence on plasma total cholesterol levels than diet in highly trained endurance cyclists.
Other researchers have reported similar results. Blair et al (1981) investigated nutrient intakes of male and female runners and controls. It was found that the runners had lower total cholesterol levels than the inactive controls, yet had a similar pattern of consumption, particularly of nutrients thought to effect plasma lipids. The data therefore was in agreement with the present, in that, the differences observed in plasma lipoproteins were likely to be due to exercise.

Hartung et al (1980) found similar results when investigating the relationship of diet and cholesterol levels in middle-aged marathon runners, joggers, and inactive men. It was evident that a negative correlation was present between the distance run and total cholesterol. The differences in cholesterol between the three groups was primarily due to distance run and not dietary factors.

Few intervention studies have looked at the effects of both diet and exercise on plasma lipids. Ekstedt et al (1991) investigated the effects of diets where either 26, 52, or 29% of energy were derived from fat in a group of highly trained cross-country skiers. Although limited in subject number (seven men) and duration of intervention (eight days), results were similar with regards to total cholesterol as those in the present study. It was found that total cholesterol was reduced by 20% with the consumption of the high fat, high cholesterol diet, which was in the same order of the standard diet (comprising of 26% of energy from fat). The present study similarly demonstrated a significant reduction in total cholesterol following the consumption of a high fat diet, and this was of the same magnitude as those consuming a diet high in carbohydrate.

Thompson et al (1984b), with feeding diets to runners where 69% of energy was derived from fat, also noted that the high fat diet had little effect on total plasma cholesterol.

Not all researchers have found that the role of diet in predicting total plasma cholesterol levels is reduced in highly trained, physically active individuals. Lukaski et al (1984) present results that conflict with those of the current study. They found that an atherogenic diet resulted in an increase in total cholesterol in endurance cyclists. Three diets were consumed where 50% of energy was derived from carbohydrate or fat. Two diets where 50% of calories were derived from fat differed in that one was predominantly saturated fat and the other was comprised of predominantly polyunsaturated fat. The diet high in saturated fat increased total cholesterol, whereas the polyunsaturated diet and the 50% carbohydrate diet decreased cholesterol levels.

In the present study the fat consumed was predominantly saturated fat (90g/day). However an increase in total cholesterol was not evident. The
small subject number in the Lukaski study, namely three, presents a severe limitation. Duration of dietary intervention was twenty-eight days in the Lukaski study compared to three-four months in the present study. Thus true adaptation to the diets may not have been evident in the former study. Lukaski concluded that the Keys' equation gives useful predictions of changes in plasma total cholesterol among vigorous men consuming different types and amounts of dietary lipid. This is somewhat surprising because the Keys' formula was developed from a population where the energy requirements were half that of the vigorous subjects used in the Lukaski study.

The Key's formula was developed to predict the effects of PUFA and SFA and dietary cholesterol on serum cholesterol (Antonis and Bersohn 1961). If the Keys' formula was used in the present study one would expect, for those consuming the high fat diet, an increase in total cholesterol of approximately 0.9mmol/l which would have transcribed to an 18% elevation of plasma cholesterol. The Key's formula is obviously not applicable to the endurance athletes in this study whose total cholesterol concentrations decreased approximately 8% with the consumption of a diet high in saturated fat. Exercise appears to have an important factor in predicting plasma cholesterol levels in endurance athletes. Exercise levels could become part of any equation used to predict plasma cholesterol.

In the present study, the consumption of a high fat diet did not increase plasma total cholesterol concentrations. Total cholesterol concentration did not differ in those consuming a diet high in fat from those consuming a diet high in carbohydrate. In fact it was evident that both dietary groups underwent a reduction in total plasma cholesterol during the study. It is likely that this decrease is due to the increased training of the subjects over the study period. Weight and body composition seem unlikely contributors as these indices did not change significantly in either of the diet groups during the intervention period.

Several plausible mechanisms may explain changes in total cholesterol with exercise. During prolonged exercise the increased energy demands of the working muscle exert a major influence on plasma lipoprotein metabolism. This is because the muscle relies on fat oxidation as the major fuel. The diversion of lipoproteins to the muscle in preference to the liver may explain the lower plasma cholesterol levels in endurance athletes. Exercisers use a considerable amount of saturated fatty acids for muscle energy before these fatty acids can exert their hypercholesterolemic action in the liver. Thus the consumption of a diet high in fat, even saturated fat, may not cause an increase in plasma total cholesterol.
It is possible that increased blood volume may in part account for the reduction of cholesterol. However since the subjects in this study were already highly trained and possessed a high level of physical fitness prior to the study, blood volume probably did not increase substantially, although additional measurements of haematocrit at each lipid/lipoprotein measurement would have been useful.

Tsopanakis et al (1989) have suggested that the mechanism by which exercise induces changes in cholesterol metabolism is via an increase in biliary excretion of cholesterol into the duodenum. Therefore cholesterol output from circulation is increased during prolonged exercise (again I did not measure this).

Saturated fatty acids may exert a hypercholesterolemic effect by retarding the catabolism of lipoproteins by influencing degradative enzymes such as lipoprotein lipase and LCAT. Exercise can increase the activity of LPL and LCAT. Therefore dietary induced reductions in these enzymes may be blunted by exercise. This would, in part, counter the hypercholesterolemic effect of saturated fat.

All metabolic changes that occur with endurance training aim at an accelerated turnover of fat in the muscle, adipose tissue and plasma. Changes in serum lipids are assumed to be a direct effect of utilisation of muscle, adipose and plasma triglycerides as a fuel.

5.2.2 LDL cholesterol

High plasma LDL cholesterol concentration is an important risk factor for coronary heart disease. LDL-C is regarded as an atherogenic lipoprotein. It is well established that a diet high in fat (particularly saturated fat) increases plasma LDL cholesterol concentrations in the general population (Fehily, et al. 1988; Keys 1970; Keys, et al. 1965; Keys, et al. 1986).

Cross-sectional studies have shown that LDL cholesterol levels in endurance athletes are lower than for non-athletes (Stray-Gundersen, et al. 1991; Williams, et al. 1986; Wood, et al. 1976). In the group of endurance cyclists used in the current study the mean baseline concentration of LDL-C was 2.91mmol/l. The LINZS has demonstrated that the mean plasma LDL-C concentration in the general New Zealand population is 4.0mmol/l (Mann, et al. 1991). This indicates that the endurance athletes in this study have average LDL levels 28% lower than the general New Zealand population. This is in agreement with other cross-sectional studies indicating lower plasma LDL-C in endurance trained athletes.
In the present study, LDL-C concentrations decreased despite the consumption of a high fat (predominantly SFA) diet. Furthermore, the decrease in LDL-C was not significantly different between the HF and HC groups. In fact, LDL-C in both diet groups was reduced significantly after twelve and sixteen weeks of dietary intervention. The 8% reduction in LDL-C with the consumption of a high fat diet is rather striking considering that untrained individuals may have been expected to show an increase in LDL-C of nearly 40% (Griffin, et al. 1988).

Ekstedt et al (Ekstedt, et al. 1991) have previously shown a significant decrease in VLDL-LDL-C in a group of cross country skiers despite the consumption of a high fat diet (fat supplying 52% of total energy). This decrease was similar in magnitude to the standard diets where 26% of energy was derived from fat. The authors suggested that physical activity is more important for the regulation of the lipoprotein fraction measured than dietary fat and cholesterol in endurance trained athletes undertaking very heavy physical exercise. This seems to be in agreement with the results obtained in the present study, where the cyclists were involved in high levels of physical exercise and the consumption of a high saturated fat diet did not increase LDL-C in the way anticipated for a sedentary group (Fehily, et al. 1988; Keys 1970; Keys, et al. 1965; Keys, et al. 1986).

Thompson et al (1984b), likewise, showed that when a diet where 69% of energy was derived from fat (111g of which was SFA), was consumed by long-distance runners (averaging 16km/d) for a period of fourteen days, little change was evident in the LDL cholesterol fraction compared to values prior to the study where a diet of 32% fat and 53% carbohydrate was consumed (pre-study LDL cholesterol were 173 ± 29mg/dl; compared to values of 162 ± 25mg/dl following the consumption of the high fat diet).

Kramasch et al (1981) also found that exercise reduced the adverse effects of an atherogenic diet. They investigated the effects or moderate exercise conditioning in monkeys fed an atherogenic diet. It was found that of monkeys fed an atherogenic diet, those who underwent exercise conditioning had lower levels of LDL and VLDL cholesterol. This study does involve an animal model and so should be regarded with caution.

Stray-Gundersen et al (1991) examined the influence of lifetime cross-country skiing on plasma lipids and lipoproteins in male and female skiers. They found LDL-C concentrations significantly lower than the general population. Analysis of the dietary data with the Keys equation suggested that dietary differences were unable to account for all of the change, again suggesting an effect of exercise on LDL-C.
The data from the present study would suggest exercise rather than diet is the predominant factor in determining LDL cholesterol in highly trained endurance athletes, since those consuming high amounts of saturated fat maintained low LDL-C concentrations. The increase in physical fitness and training throughout the study is likely to have contributed to the reduction in LDL-C concentrations in both groups of cyclists during intervention.

It has been suggested (Stray-Gundersen, et al. 1991) that a large volume of endurance exercise undertaken for many years is necessary for a significant reduction in total or LDL cholesterol. The cyclists in the present study have on average been partaking in endurance cycling for seven and a half years (range two to twenty-two years).

Exercise, thus, seems to be important for the determination of plasma LDL cholesterol levels in endurance athletes and it is interesting to note that Sutherland et al (1981b) and Thompson et al (1984a) found that LDL increased 15% and 14% respectively with the cessation of physical exercise. In part, such an increase could be due to a reduction in plasma volume due to cessation of exercise. Plasma volume is expanded with physical training (Oscai, et al. 1968). Changes in plasma volume can effect the measured concentrations of lipoproteins.

A mechanism suggested for lowered LDL cholesterol levels with endurance training is related to lower plasma triglycerides that are evident in endurance trained athletes. It is possible that low triglyceride levels may cause a decrease in the secretion or enhance the clearance of apo B containing lipoproteins (Stray-Gundersen, et al. 1991). Exercise may divert fat as a substrate away from the liver and thus retard the formation of apo B-containing lipoproteins. Trained individuals have an increase in oxidative capacity and therefore a decrease in the availability of free fatty acids for VLDL synthesis. In trained subjects the route of free fatty acid utilisation following mobilisation from fat cells would be more towards the muscle than the liver (Deprés, et al. 1985).

Aerobic athletes show a redistribution of plasma cholesterol with a decrease of the quota linked to the atherogenic VLDL and LDL and an increase in the protective lipoprotein, HDL (Giada, et al. 1991). This change in lipoprotein metabolism is due to the adaptation to physical training which increases the organism's capacity to increase fatty acid utilisation as a fuel during exercise.

The high fat diet may have failed to increase plasma LDL cholesterol because of the low carbohydrate content of the diet. This may have caused a subsequent decrease in VLDL synthesis, which in turn would decrease the formation of LDL cholesterol.
5.2.3 HDL cholesterol


There is strong evidence of a positive relationship between HDL cholesterol and physical activity (Baker, et al. 1986; Marti, et al. 1991; Marti, et al. 1990; Myhre, et al. 1981). Cross-sectional studies have consistently demonstrated that endurance athletes have higher HDL-C concentrations than sedentary individuals.

In the present study, at the end of the intervention period where training and physical fitness had increased, the mean HDL cholesterol level was 1.4mmol/l. This is slightly higher than for the general New Zealand population whose average plasma HDL cholesterol is 1.3mmol/l (Mann, et al. 1991).

One of the main concerns in advocating a high carbohydrate diet to reduce coronary heart disease risk, is the reduction in HDL-C following the consumption of a diet high in carbohydrate (Blum, et al. 1977). This reduction in plasma HDL-C concentration was not present amongst cyclists consuming a high carbohydrate diet in the present study. HDL cholesterol concentrations were not significantly different between subjects consuming a high fat diet and those consuming a high carbohydrate diet. It seems likely that the high level of physical exercise undertaken by the subjects in this study offset the expected reduction in HDL cholesterol.

Thompson et al (1984b) fed subjects a diet comprising of 69% of energy derived from carbohydrate. The reduction in HDL cholesterol in distance runners was only 9%, significantly lower than that observed in sedentary individuals where reductions of about 23% have been apparent (Blum, et al. 1977; Gonen, et al. 1981; Oster, et al. 1981; Schonfeld, et al. 1976). Thus, although changes in HDL-C were qualitatively similar in runners to non-athletes, the athletes response to a high carbohydrate diet may have been blunted.

Moore et al (1983) also showed that physical activity was more important than diet in determining HDL cholesterol concentrations. Long distance runners and joggers had higher plasma HDL-C (78mg/dl and 70mg/dl respectively) concentrations than sedentary controls (62mg/dl). The authors concluded that HDL-C differences between runners, joggers and sedentary controls could not be attributed to dietary intake. Distance run and percentage body fat were the strongest predictors of HDL-C. Even when the results were adjusted for percentage body fat, exercise remained a significant predictor of HDL-C.
Ekstedt et al (1991) in a study of highly trained cross-country skiers consuming diets high in fat or carbohydrate found that HDL cholesterol levels were increased significantly only when consuming a diet higher in fat. The authors concluded that the changes in HDL cholesterol seemed to be strongly influenced by diet. However, it must be noted that even when following a high carbohydrate diet, the skiers underwent a slight increase in plasma HDL concentrations. It is again possible that the expected decrease in HDL cholesterol was offset by the high level of physical activity undertaken by the athletes.

Thompson et al (1983) compared serum lipids and lipoproteins and ten day food records of twenty male distance runners to fourteen sedentary controls. Runners had higher plasma HDL cholesterol levels despite consuming significantly higher absolute levels of carbohydrate (413g/day compared to 294g/day). The cyclists in the current study consumed approximately 400 ± 110g/day of carbohydrate. This value is likely to be underestimated because of the failure of the HC diet group to record high carbohydrate supplements and training foods. This, again, suggests that consumption of a diet high in carbohydrate does not suppress HDL levels in individuals engaging in high intensity endurance training. However, in contrast to the present study, Thompson suggests that dietary factors may be as important as exercise itself in producing the lipoprotein pattern characteristic of endurance athletes. Diet did not appear to be a significant contributor to the plasma lipid profiles of the cyclists in the present study. The cyclists in this study were highly trained and were involved in large volumes of high intensity exercise. Average weekly training distances were 230 ± 150km, with racing distances ranging from 30 to 1120km. This level of exercise is higher than that reported in many previous studies, and thus, is likely to be a factor contributing to discrepancies between the present study and former research.

Significant increases in HDL cholesterol were not apparent in either group until week sixteen of intervention. There is a possibility that the increase in HDL cholesterol at week sixteen is due to the smaller subject number, namely twenty-two, who continued for this extended period of four weeks. The average HDL cholesterol at week sixteen was 1.36mmol/L for HC and 1.50mmol/L for HF. This significant increase from baseline (HC=1.32mmol/L; HF=1.35mmol/L at baseline) and week twelve (HC=1.36mmol/L; HF=1.35mmol/L at week 12) could possibly have been caused by increased adaptation over this extended period, that would have taken in excess of twelve weeks. A review of training studies showed that where training duration was ten weeks or less, many studies have failed to show an increase in HDL-C levels. However, in all studies
greater than twelve weeks duration, an increase in HDL cholesterol was observed (Haskell, et al. 1988). Thus duration of physical training may be an important factor for alterations in HDL cholesterol. Farrell et al (1980) showed that alterations in HDL cholesterol lagged behind changes in total cardiovascular fitness. However, the subjects in the present study were of a high level cardiovascular fitness prior to the commencement of the study.

There is evidence that the increase in HDL cholesterol in athletes becomes more marked with increasing age (Giada, et al. 1991). The relative youth of the cyclists in the present study, where the average age was 28.4 ± 8.9 years, may in part explain the non-significant rise in HDL cholesterol with increased physical training.

Many mechanisms have been proposed to explain the higher HDL-C concentrations in endurance athletes compared to their less active counterparts. The changes in lipoprotein proportions seem to be related to adaptation to endurance exercise. Endurance training is associated with increased muscle capillarisation, increased mitochondrial size and number, and changes in enzyme systems aimed at an accelerated turnover of fat in adipose tissue, blood and muscle (Davies, et al. 1981; Mole, et al. 1971; Persson and Carlgren 1984). One of the key enzymes that is known to increase with endurance training is lipoprotein lipase (LPL) (Nikkilä, et al. 1978). This enzyme is thought to play a major role in the increase in HDL cholesterol with endurance type training. Lipoprotein lipase is responsible for the degradation of triglyceride-rich particles, with part of the cholesterol, phospholipid and apoproteins being transferred to the HDL class. An increase in muscle and adipose tissue lipoprotein lipase represents one of the many adaptive phenomena that occur with endurance training which aims to increase the capacity of the body to utilise fat as a fuel. Thus, it is likely that one of the major mechanisms for an increase in HDL levels with endurance training is the increased breakdown of VLDL-TG by LPL with the constituents being repackaged into HDL cholesterol. LPL may be increased during exercise through stimulation by increased circulating catecholamines associated with exercise (Holloszy, et al. 1964). Furthermore, it is speculated that rises in LPL may be related to the decrease in insulin secretion, caused by increased insulin sensitivity of tissue as a result endurance training (Hartung, et al. 1980; Nikkilä, et al. 1978).

A decrease in lipoprotein lipase activity of 56% has been reported in sedentary individuals consuming a 70% carbohydrate diet (Lithell, et al. 1982). This observation is likely to account in part for a reduction in HDL concentrations. In endurance trained individuals, exercise-induced rises in
lipoprotein lipase concentrations could blunt the reduction of this enzyme as a result of a high carbohydrate intake.

In the present study lipoprotein lipase activity was not measured. However, high levels of LPL may account for the reasonably high levels of HDL-C found in the cyclists, and LPL activity may have increased in participants whose HDL-C increased. LPL has been shown to be correlated with VO2max and physical fitness (Weintraub, et al. 1989). The physical fitness of subjects in this study increased. LPL activity may also have increased. Increased activity of LPL may account for prevention of the expected fall in HDL cholesterol with the consumption of a high carbohydrate diet.

Another enzyme that has been shown to correlate positively with aerobic capacity is lecithin:cholesterol acyltransferase (LCAT) (Marneimi, et al. 1982; Williams, et al. 1990). Increased LCAT activity may produce a rise in HDL in endurance trained athletes. LCAT is thought to play an essential role in cholesterol removal. LCAT is responsible for the removal of free cholesterol from the lipoprotein surface into the core which creates a concentration gradient that favours the net transfer of cholesterol from membranes to the lipoprotein surface.

An increase in survival of the HDL cholesterol could explain higher levels of this fraction in endurance athletes. It has been hypothesised that decreased levels of hepatic lipase increases HDL cholesterol levels by enhancing it's survival (Kuusi, et al. 1980; Kuusi, et al. 1982). Hepatic lipase has a physiological role in the degradation and removal of HDL particles from circulation. It has been proposed that hepatic lipase, by it's phospholipase action degrades the surface phospholipids of the HDL particle and this is followed by the release of cholesterol esters to the liver (Kuusi, et al. 1980). Lower activity levels of hepatic lipase as a result of physical exercise could well, in part, explain the higher levels of HDL cholesterol found in endurance athletes.

Hepatic lipase increases with the consumption a high carbohydrate diet. Increased hepatic lipase with a diet high in carbohydrate may be blunted with exercise (Thompson, et al. 1984b). Thus, it is possible that in the present study, low levels of hepatic lipase activity in the cyclists may have offset the expected decrease in HDL amongst those consuming a high carbohydrate diet.

Blum et al (1977) demonstrated that the decrement in HDL-C with the consumption of a high carbohydrate diet in sedentary individuals was a result of increased catabolism of the HDL cholesterol fraction, with the mean synthetic rate of HDL not affected by a high carbohydrate diet.
Therefore, it appears that exercise may counter the degradation of HDL cholesterol by reducing the catalytic rate of the HDL fraction.

Modification in the enzymes involved in HDL metabolism may mediate the exercise-induced changes in lipoproteins. The high rate of energy turnover in skeletal muscle and an increase in adrenergic drive during endurance exercise may be responsible for the changes in the activity of these enzymes (Haskell, et al. 1988).

5.2.4 HDL\(_2\) cholesterol

HDL\(_2\) cholesterol is thought to be the subfraction of HDL that is important for reducing the risk of coronary heart disease (Williams, et al. 1982). The HDL\(_2\) has been shown to be increased with endurance training (Ballantyne, et al. 1982; Rauramaa, et al. 1984). In the present study there were no significant differences in HDL\(_2\) between the two diet groups.

HDL\(_2\) cholesterol was not reduced with the consumption of a high carbohydrate diet (62% of total energy from carbohydrates). Again it is likely that physical exercise was a more important determinant of the HDL\(_2\) subfraction than was diet. Thompson and associates demonstrated in distance runners that the consumption of a high carbohydrate diet (69% of total energy from carbohydrate), resulted in a reduction in the HDL\(_2\) subfraction. Despite the high carbohydrate intakes, HDL\(_2\) concentrations remained above those of sedentary controls. This suggests that physical exercise may blunt the reduction in HDL\(_2\) caused by a high carbohydrate diet.

Several mechanisms may explain the higher plasma HDL\(_2\)-C concentrations of trained individuals. HDL\(_2\) is generated during the hydrolysis of triglyceride-rich lipoproteins by lipoprotein lipase. An adaptive increase in lipoprotein lipase activity in adipose tissue and muscle in response to endurance training has been observed. HDL\(_2\) levels have been shown to be positively correlated with lipoprotein lipase (Nikkilä, et al. 1978).

Another mechanism suggested for the higher levels of HDL\(_2\) in endurance athletes is decreased catabolism of this subfraction (Myhre, et al. 1981). Experimental studies have indicated that HDL\(_2\) is metabolised in the liver and that heparin-releasable hepatic lipase is the active enzyme involved in HDL\(_2\) cholesterol degradation. It has been reported that HDL and HDL\(_2\) cholesterol are inversely related to hepatic lipase activity (Kuusi, et al. 1980). Hepatic lipase activity seems lower in physically active individuals compared to those who are sedentary.
HDL₂ cholesterol did not increase significantly in either diet group during the intervention period even though increases in physical fitness and training were evident. It is possible that the athletes in the present study were physically trained to the extent that their HDL₂ concentrations were at an optimum level prior to the study.

5.2.5 HDL₃ cholesterol

Although HDL₂ cholesterol is believed to be the subfraction most markedly effected by endurance exercise, HDL₃ cholesterol may also increase with aerobic exercise (Stampfer, et al. 1991).

In the cyclists in the present study, slight increases in HDL₃ were noted to week twelve of intervention. At week sixteen, HDL₃ cholesterol increased significantly in both diet groups.

As previously mentioned, evidence does exist suggesting that a high carbohydrate diet decreases HDL cholesterol levels. However several investigators have demonstrated that this effect is blunted by physical activity. Thompson et al (1984b), showed a smaller decrease in HDL-C (9%) in distance runners than would be expected in inactive individuals (23%) (Blum, et al. 1977; Gonen, et al. 1981; Oster, et al. 1981; Schonfeld, et al. 1976), after the consumption of a high carbohydrate diet (69% of total energy from carbohydrate). The reduction in HDL cholesterol was mainly due to the reduction in the HDL₂ subfraction. Dietary carbohydrate seemed to have little effect on the HDL₃ subfraction. In the present study dietary carbohydrate demonstrated no effect on HDL₃ concentrations of the endurance trained cyclists. In fact, at the completion of dietary intervention the HDL₃ subfraction was increased in both diet groups, suggesting that increased training and physical fitness may actually increase HDL₃.

5.2.6 Apolipoprotein A1

Apolipoprotein A1 is the major protein associated with HDL cholesterol. Cross-sectional studies have shown higher apolipoprotein A1 concentrations in endurance trained athletes compared to inactive individuals (Thompson, et al. 1983). Endurance training has also been shown to produce an increase in apo A1 concentrations (Kiens, et al. 1980).

In the present study, apo A1 levels increased (from 106mg/dl to 113mg/dl at week 12 for both groups; and to 111mg/dl for HC and
115mg/dl for HF at week 16) in cyclists consuming high fat and high carbohydrate diets.

Apo A1 particles are synthesised and secreted as nascent HDL-particles. Intestinal synthesis of this apolipoprotein is stimulated by fat (Schonfeld, et al. 1978). Therefore a higher concentration of apo A1 would have been expected in the group consuming the high fat diet, compared to those consuming a carbohydrate-rich diet. However, such a difference was not observed. No differences were present between the two diet groups. Other mechanisms, other than the intake of dietary fat must be of greater importance in the regulation of apo A1 in endurance trained men. In the present study, high levels of endurance exercise are likely to have influenced apo A1 concentrations, as significant correlations were present between hours of endurance training and apo A1 concentrations.

Thompson et al found that in distance runners, consumption of a diet high in carbohydrate (69% of energy derived from carbohydrate), apo A1 levels did not decrease as would be expected in inactive individuals.

Kiens et al (1981) similarly found that in men partaking in regular physical training (average of 50 minutes aerobic exercise daily), apo A1 was unaffected by a high fat diet (54% fat). However, when the same subjects consumed a ‘fat-poor’ diet (29% fat), apo A1 decreased by 8%. This was 8% lower than after the consumption of the fat-rich diet (p<0.05).

Contrary to the results of the present study, Blum et al (1977) demonstrated that the consumption of a diet where 80% of calories where derived from carbohydrate, resulted in an increase in the catalytic rate of the HDL apoprotein of 39% in several subjects. In the present study, diet did not seem an important factor influencing Apo A1 concentrations. However subjects in the present study consuming a high carbohydrate diet, approximately 63% of calories were derived from carbohydrate. This is substantially less than the 80% carbohydrate diet consumed by the subjects in the Blum study, although the absolute amounts of carbohydrate may have higher in the present study. The major difference between the present study and the latter, is that in the present study, subjects were highly trained endurance athletes. The subjects of Blum et al were sedentary individuals.

Thompson et al (1984b) demonstrated that with the consumption of a diet where 69% of energy was derived from fat, apo A1 levels on the final day of a fourteen day intervention period were slightly higher than initial values. In the present study apo A1 values were in fact significantly higher with the consumption of a high fat diet. However my intervention period
was three to four months and is likely to allow for greater adaptation to the diets.

Apo A1 increased significantly to week twelve of dietary intervention in the present study, but this was not accompanied by a significant increase in HDL cholesterol. The protein component of HDL increased which is responsible for the activation of LCAT, which as previously mentioned, increases HDL concentrations. This increase in apo A1 concentration to week twelve may in part contribute to the significant increase in HDL cholesterol at week sixteen. Additionally, apo A1 may be a more sensitive measure of HDL than the precipitation technique used for the measurement of HDL cholesterol.

A further mechanism that may explain the increase in apo A1 levels observed in this study is the prolonged survival of HDL proteins that has been previously reported in runners compared to sedentary men (Herbert, et al. 1984), suggesting that reduced catabolism of apo A1 may be responsible for the increase of apo A1 with endurance training.

5.2.7 Apolipoprotein B

Apolipoprotein B100 is in essence the only protein component of LDL. Elevated levels of apo B in the blood is a recognised risk factor for coronary heart disease (Consensus Conference 1985).

In the present study apo B levels decreased significantly over the study period in both diet groups.

This finding is in agreement with the finding of Baker et al (1986) who showed that twenty weeks of endurance training resulted in a decrease in LDL-protein levels by 18.7%. This was presumably a decrease in apo B levels.

The increase in physical fitness and training in the present study seems likely to have resulted in the decrease in apo B levels observed. Apo B levels decreased in both the HC and HF groups and there was no significant difference between groups HC and HF. Surprisingly consumption of a diet rich in fat, did not result in an increment of apo B in this group of highly trained athletes.

An increase in the delivery of metabolites to the muscle is likely to have diverted dietary derived fat from the liver to the muscle. Thus, reduced delivery to the liver and the reduction of lipoprotein precursors could result in the observed reduction of plasma apo B and LDL cholesterol.

The reduction in apo B is likely to be related to the significant reduction in LDL cholesterol observed in the present study.
5.2.8 Triglycerides

Endurance athletes have lower plasma triglyceride levels compared to sedentary controls (Giada, et al. 1991; Martin, et al. 1977; Tyroler 1989; Wood, et al. 1976). The triglyceride levels of the cyclists in the present study were $1.26 \pm 0.36 \text{ mmol/L}$ for HC and $1.27 \pm 0.38 \text{ mmol/L}$ for HF. This is slightly lower than for the general New Zealand male population who have fasting triglyceride concentrations of $1.3 \pm 1.0 \text{ mmol/l}$ (LINZS) (Mann, et al. 1991). With increased physical training during the study period triglyceride levels dropped to $1.19 \pm 0.45 \text{ mmol/l}$ and $0.97 \pm 0.33 \text{ mmol/l}$ at weeks twelve and sixteen respectively for HC. In HF, TG levels decreased to $1.18 \pm 0.48 \text{ mmol/L}$ and $0.95 \pm 0.26 \text{ mmol/L}$ at weeks twelve and sixteen respectively. These values are substantially lower than observed in the general New Zealand population.

In this study triglyceride levels decreased significantly in both diet groups. There were no significant differences in TG levels between the HF and HC groups. It therefore seems that exercise was a more important determinant of triglycerides levels than was diet. Significant correlations were present between average and total kilometres cycled/week and TG concentrations at week twelve and sixteen.

These results are in agreement with those of Ekstedt et al (1991) who studied the effects of the consumption of diets where 26%, 29% or 52% of energy was derived from fat. It was found that serum triglycerides decreased more than 30% in all diet groups, and no significant differences between the diets were present.

Other investigators have demonstrated that physical training can reduce plasma triglyceride concentrations. Weintraub (1989) showed that a seven week exercise conditioning programme resulted in a decrease in both post-prandial and fasting triglyceride concentrations. These findings were present in the absence of weight loss. In the present study there were also no significant change in body weight or composition.

Increased exercise conditioning throughout the present study (as shown by increases in physical fitness variables (increases in VO2max, increased time to exhaustion and higher maximum wattage attained during exercise test) and an increase in training volume (according to training diaries)), are likely to be major contributors to the decrease in triglyceride concentrations since other variables such as weight and total body fat did not change throughout the study.

The importance of low plasma triglyceride levels should not be underestimated. Even in normolipidemic individuals, the variability in postprandial lipemia is thought to be possibly pathophysiological, since a
significant lipemia may persist throughout most of the day (Moreton 1950). Much time is spent in the postprandial state. Work by Zilversmit (1979) suggests that the postprandial metabolism of triglyceride-rich lipoproteins may attribute to an atherogenic process in those who chronically consume a diet high in fat.

It is therefore important in view of CHD risk, to decrease plasma triglyceride levels and the magnitude of post-prandial lipemia. This could provide a mechanism for protecting against atherosclerotic plaque formation. The consumption of a high fat diet in the present study would be expected to be associated with an increased risk of atherosclerosis. However fasting triglycerides did not increase with the consumption of a diet high in fat over a sixteen week period. Post-prandial lipemia was not measured in the present study. However, previous research has demonstrated that endurance training is associated with a diminished postprandial lipemia after a high fat diet (Merrill, et al. 1989; Weintraub, et al. 1989). Cohen et al (1989) have shown that chylomicron clearance and post-prandial lipemia are directly related to fasting triglyceride concentrations. The high level of endurance training undertaken by the cyclists in this study is likely to be responsible for the reduction in plasma triglyceride concentrations in those consuming high fat and high carbohydrate diets.

There is direct competition for the common clearance pathway between hepatic derived and intestinal derived triglyceride-rich lipoproteins. Thus, decreased post-prandial lipemia and accelerated chylomicron-triglyceride clearance would be expected to occur in association with fasting hypotriglyceridemia in endurance trained men. These athletes are therefore at a decreased risk of cardiovascular disease. Merrill et al (1989) hypothesised that if postprandial lipemia places individuals at an increased risk of coronary artery disease (CAD), then even in individuals of whom are traditionally considered to be of low CAD risk, but who chronically consume a diet high in fat, regular endurance exercise may decrease this risk.

Several mechanisms may explain the decrease in plasma triglycerides observed in the present study. These mechanisms are related to adaptations occurring with endurance training and are mediated through exercise-induced changes in enzymes involved in cholesterol and triglyceride synthesis, transport, and catabolism. Lipoprotein lipase is the rate limiting enzyme in the removal of triglycerides from the plasma. Extensive research has demonstrated that this enzyme is increased in adipose tissue and muscle with endurance training (Lithell, et al. 1981; Marti, et al. 1990; Nikkila, et al. 1978). The increase in LPL results in increased catabolism.
of triglyceride-rich lipoproteins, thus decreasing the concentration of plasma triglycerides.

Increased LPL activity facilitates the restoration of muscle and adipose tissue triglyceride stores during the recovery period following exercise, thus promoting plasma clearance of VLDL following exercise and therefore a decrease in plasma triglycerides. Increased capillarisation of skeletal muscle enables larger amounts of the substrate in the form of VLDL to come into contact with capillary-bound lipoprotein lipase (Haskell, et al. 1988).

The VLDL fraction may serve as an available energy source during endurance exercise. Kiens et al (1993) observed a net uptake of circulating VLDL-TG across the thigh during two hours of knee extension exercise, suggesting that this fraction may be an available source of lipid for the muscle during endurance exercise.

Cohen et al (1989) suggest that chronic exercise decreases postprandial lipemia by decreasing the chylomicron-triglyceride half-life, due partly to a decrease in the fasting serum triglyceride pool and partly due to the direct effect of exercise on the serum triglyceride removal system.

Diets high in carbohydrates have been shown to increase the rates of hepatic secretion of VLDL in sedentary individuals (Adams, et al. 1974; Quarfordt, et al. 1970). In the present study the consumption of a diet high in carbohydrate failed to increase plasma triglyceride levels as previously noted (Blum, et al. 1977; Schonfeld, et al. 1976; Wilson and Lee 1972). A possible explanation for this phenomenon is an increase in insulin sensitivity that occurs with physical training (Bloom, et al. 1976). A decrease in postprandial insulin response to a high carbohydrate diet decreases hepatic VLDL-triglyceride secretion and thus decreases plasma triglyceride concentrations. This exercise-induced increase in insulin sensitivity could protect against the possible negative effects of a high carbohydrate diet.

Furthermore, the stress associated with endurance training and competition is associated with an increase in the release of catecholamines causing an increase in lipolysis and favouring a decrease in triglyceride concentrations (Caballero, et al. 1992).

Sutherland et al (1980) have suggested that VLDL and HDL fluxes are significantly related. An increase in the catabolism of VLDL by lipoprotein lipase may contribute to the increase in HDL levels evident after physical training. Marti et al (1990) have also suggested that high HDL cholesterol levels induced by exercise training are caused in part to the increased catabolism of triglyceride-rich lipoproteins. This relationship may account for both the increase in HDL concentrations and the decrease in plasma
triglyceride concentrations observed after a period of endurance training in the athletes in the present study.

One of the major protective effects of exercise against CHD is via the reduction in triglyceride-rich lipoproteins because VLDL, IDL and postprandial lipoproteins have atherogenic properties. Thus decreased exposure of these to vessel walls would be beneficial (Weintraub, et al. 1989).

Chronic exercise improves fat tolerance, suggesting that a high fat diet does not affect a highly trained endurance athlete in the same way it would affect a sedentary individual. This, in part, may explain why the high fat diet consumed in the present study did not result in an atherogenic lipid/lipoprotein profile in the endurance trained cyclists.

5.3 Body weight and composition

The subjects in the present study were relatively lean when compared to the general New Zealand population. Data from the Life in NZ Survey (Mann, et al. 1991) showed that total body fat as estimated by the sum of six skinfolds is 85.5 ± 36.4mm. This is over 35% higher than the total body fat estimated in the present study where the average sum of six skinfolds for subjects at baseline was 59.4 ±18.8mm for HC and 45.7 ±13.4mm for HF.

Alterations in body weight and composition can affect lipoprotein metabolism. Many investigators argue that changes in body weight are largely responsible for the exercise-induced alterations in lipoprotein profiles (Drinkwater, et al. 1989; Marti, et al. 1991). Reduction in adiposity with long term endurance training has been suggested to be strongly related to the subsequent increase in HDL cholesterol and the reduction in VLDL concentration. Some previous research has indicated that without the concomitant weight loss with exercise, cholesterol levels would not be expected to change (Marti, et al. 1991; Williams, et al. 1982; Wood, et al. 1988).

Other studies have shown favourable changes in plasma lipids and lipoproteins with the onset of physical exercise, and in the absence of weight loss (Schwartz, et al. 1992; Owens, et al. 1992; Weintraub, 1989; Northcote, et al. 1988; Thompson, et al. 1988).

In the present study changes in the lipoprotein profiles of the endurance cyclists occurred independently of weight loss and alterations in body composition. There were no significant weight changes in either dietary group during the intervention period. In addition there were no significant
changes in body composition (estimated from S6SF) in either diet group over the study duration. It seems unlikely that body weight and/or composition change account for the alterations in lipoproteins observed in this study.

Weight gain was not observed in those consuming a high fat diet, indicating that endurance athletes are able to maintain energy balance whilst following such a regimen. Body weight did not differ significantly from those consuming a diet high in carbohydrate.

5.4 Exercise Performance

A high carbohydrate diet is recommended for endurance athletes to enhance physical performance. Current dietary recommendations advocate a diet of 65-70% carbohydrate or intakes of 10g/kg/day of carbohydrate in preparation for an endurance event (Simopoulos 1992). Such dietary recommendations formed the basis of the high carbohydrate diet in the present study. I compared exercise capacity and performance of those consuming a high carbohydrate diet to those consuming a diet relatively high in fat.

Many researchers anticipate decreased endurance with the consumption of a high fat diet. However, in the present study I have shown otherwise. There was no difference in work capacity, endurance capacity or performance as measured by a VO2max test including, time to exhaustion and maximum workload attained, between those consuming a diet high in carbohydrate and those consuming a high fat diet. In fact, both dietary groups underwent an increase in physical fitness throughout the intervention period. No differences in performance in weekly cycle racing were evident between the two groups.

The increase in training throughout the intervention period is likely to have resulted in the increase in physical fitness observed. Increased training was evidenced in the participants’ training diaries.

Previous investigators have demonstrated a reduction in work capacity with the consumption of high fat diets (Bergström, et al. 1967; Christensen and Hansen 1938). However, these studies differ from that of the present in that the duration of dietary intervention was short, not allowing for true adaptation to the diets. Christensen and Hansen allowed subjects to consume the study diets for a period of only three days to a week. It has been suggested that adaptation to high fat diets may take in excess of six to twenty weeks (Hammel, et al. 1977; Phinney, et al. 1980). In addition to this, the macronutrient proportions in much of the
previous research were more extreme than in the present study, and thus unrealistic to consume for prolonged periods. In the research by Christensen and Hansen the high fat diet comprised of approximately 95% of energy from fat and 5% from carbohydrate; and the high carbohydrate diet was comprised of approximately 95% of energy from carbohydrate and 3% from fat. This is in marked contrast to the present study where 60-65% and 20% of energy was derived from carbohydrate and fat respectively, on the high carbohydrate diet. The high fat diet comprised of 35% and 45-50% of energy derived from carbohydrate and fat respectively.

Drastic dietary changes such as that used by Christensen and Hansen for a period of three days is not likely to provide an adequate indication for potential physical performance on such diets. The less extreme fat intake and duration of the present study is likely to provide a superior indication of exercise capacity with the consumption of a diet high in fat.

Previous investigators have concluded that the limiting factor in performance of endurance exercise is the preformed muscular glycogen stores (Ahlborg, et al. 1967; Bergström, et al. 1967; Hermansen, et al. 1967; Hultman 1967). Current dietary recommendations for endurance athletes are based on research correlating fatigue with muscle glycogen depletion; and preformed muscle glycogen stores with endurance capacity. Carbohydrate loading regimes have resulted from such research. The subjects in the present study consuming a high fat diet were unlikely to have maximised their glycogen stores. However endurance capacity was not compromised. This finding suggests that factors other than availability of muscle glycogen are important exercise determinants.

Previous researchers have found similar results. Phinney et al (1983b) demonstrated that trained cyclists were able to consume a eucaloric ketogenic diet (low carbohydrate diet that induces ketosis) without compromising endurance capacity. Animal evidence also exists supporting the findings of the present study. Miller and colleagues (1984) demonstrated that rats exposed to high fat diets were capable of prolonged intense exercise.

In a recent study Sherman et al (1993) investigated the effects of a high carbohydrate diet (10g CHO/kg/day) and a diet containing moderate amounts of carbohydrate (5g/kg/day), on muscle glycogen and performance in runners and cyclists. Subjects (n=36) initially consumed a diet containing 8g CHO/kg/day (67% CHO, 15% protein, 18% fat), for a control period of five days, prior to consuming either a high or moderate carbohydrate diet for seven days. Subjects completed training sessions on all seven days with exercise, on average, eliciting 79% maximum heart
rate and 75% peak VO\textsubscript{2}max, through a one hour training session. This was followed by five, one-minute sprints eliciting, on average, 90% maximum heart rate. Following the training session on day seven, subjects ran or cycled to exhaustion at 80% peak VO\textsubscript{2}. Muscle glycogen was maintained on the high carbohydrate diet, but was reduced by 30-36% with the moderate carbohydrate diet. However, no differences in training capacity or times to exhaustion were evident between the two dietary regimens. Therefore, even though the cyclists and runners who were consuming a diet of a moderate carbohydrate content had a reduced muscle glycogen content, there were no deleterious effects on training capacity or high intensity exercise performance. Sherman et al concluded that a high carbohydrate diet maintains muscle glycogen, but this has no apparent benefit on training or high intensity exercise performance.

It must be noted that although muscle glycogen stores can be increased in trained subjects after the consumption of a carbohydrate rich diet, there are limitations to the amount of glycogen which can be stored in muscle (approximately 0.5kg).

The current study, along with previous research encompassing low-carbohydrate, high-fat diets supports the belief that both animals and humans can adapt to a high fat diet and endure endurance exercise. It is possible that a diet relatively higher in fat complements exercise induced metabolic changes allowing for increased fat oxidation during exercise.

5.4.1 Adaptation

Endurance training changes the regulation and mobilisation of fuels. Metabolic adaptations to endurance training allow a trained individual to oxidise proportionally more fat and less carbohydrate at a given workload (Holloszy and Coyle 1984). The altered neuro-hormonal environments during strenuous exercise strongly favours lipolysis. Catecholamines, cortisol and growth hormone increase, result in enhanced lipolysis. Enzyme systems also undergo alterations which allow for an increased free fatty acid release from adipose tissue. With prolonged exercise the regulation of substrate utilisation by the muscle is influenced by both intra- and extra-muscular factors. The inhibition of a massive uptake of glucose in favour of fat by the muscle allows for conservation of glucose in the blood and liver. If the uptake of glucose by the muscle was allowed to proceed unabated, this would have a deleterious effect on the nervous system which is dependent on glucose as it’s major energy source. Therefore, the increased reliance on fat as a fuel during prolonged exercise
has important implications. If the contribution of plasma free fatty acids is restricted, work capacity is severely inhibited.

There are similarities in the adaptation to a high fat diet and the adaptation to endurance exercise.

Jansson and Kaijser (1982) investigated the contributions of the different fuels to oxidative metabolism during endurance exercise in those consuming a high fat diet and those consuming a diet high in carbohydrate. It was found that glycogen depletion and lactate production were significantly reduced with the consumption of a high fat diet. In addition to this, the RQ (the ratio of the amount of carbon dioxide produced to the amount of oxygen consumed) was lower and an associated increase in fatty acid extraction by the exercising muscle was apparent following the high fat dietary regimen. With the consumption of the high carbohydrate diet, the relative contributions of fat and carbohydrate to oxidative metabolism was 27% and 73% respectively, and whilst following the high fat diet this changed to 63% and 37%. Such results indicate that adaptation to a high fat diet increases the relative contribution of fat to oxidative metabolism. An increase in plasma free fatty acids was present and the reduction in carbohydrate utilisation was attributed to a decrease in muscle glycogen breakdown.

The cyclists in the present study are likely to have adapted to the high fat diet in a similar manner. Such adaptations are likely to have allowed the subjects in this study to maintain the high level and intensity of endurance training which was evident during the intervention.

Research on rats by Simi et al (1991) has shown that training and high fat diets have an additive effect on energy metabolism during exercise. Prolonged adaptation to a high fat diet increased maximal oxygen uptake and submaximal running endurance. It was found that the diet-induced effects were cumulative with the well-known training effects on VO_{2\text{max}}, exercise endurance, oxidative capacity of red muscle and metabolic responses to exercise. In addition, there was a further reduction in liver glycogen breakdown. Such adaptations could also have occurred in the present study with enzymatic changes in skeletal muscle associated with endurance training enhanced by the superimposition of a high fat diet.

Several mechanisms are likely to be responsible for the maintenance of endurance capacity with the consumption of a diet relatively higher in fat. It seems that an increase in the capacity for exercise is partially the result of muscle adaptation which increases the ability to oxidise fat and concomitantly spare glycogen. Miller et al (1984) demonstrated that a high fat diet enhances exercise performance by increasing fat availability which both spared glycogen and delayed fatigue.
Glycogen sparing could partially be due to increased citrate concentrations. Jansson and Kaijser (1982) postulated that glycogen sparing caused by a high fat diet is due to an increase in citrate levels. Citrate inhibits the enzyme phosphofructokinase which primarily controls the rate of glycolysis. The increased supply of fatty acids after a high fat diet could result in the inhibition of glycolysis at the phosphofructokinase step.

Cyclists in the current study may have had increased intramuscular triglyceride stores with the consumption of a high fat diet. Holloszy et al (1984) have shown that in trained cyclists, the capacity of the quadriceps to incorporate fatty acids into triglycerides was increased. Morgan et al (1969) have also demonstrated higher levels of triglycerides in the trained muscle. In the trained state the utilisation of intramuscular triglyceride stores is increased. Hurley et al (1986) observed a 20% decrease in intramuscular triglycerides after two hours of bicycle exercise at 65% of VO2max in the untrained state. Following twelve weeks of endurance training the decrease was twice that as in the untrained state. It is possible that the ability to store intramuscular triglycerides is increased with the consumption of a high fat diet.

Conlee (1990) found that rats adapted to a high fat diet did not have a reduced capacity for endurance exercise, even after recovery from previous exhausting work bouts. They suggested this was in part due to an increase in storage and utilisation of intramuscular triglyceride stores.

A possible mechanism for increased intramuscular triglyceride stores is an increase in the enzyme lipoprotein lipase. Lipoprotein lipase is increased in the muscle of trained individuals (Nikkilä, et al. 1978). Intramuscular lipoprotein lipase activity may function as a regulator of endogenous triglyceride in muscle and exercise can activate endogenous lipoprotein lipase (Oscai and Palmer, 1983). An increase in lipoprotein lipase also leads to a rapid replenishment of triglyceride stores in the muscle. Lipoprotein lipase may also favour the release of fatty acids directly from the plasma free fatty acid pool and ultimately to oxidation in the muscle (Nikkilä, et al. 1978).

Lipoprotein lipase is increased by both exercise conditioning and with the consumption of a high fat diet (Jacobs, et al. 1982). It is thus possible that a high fat diet complements the exercise-induced elevation of lipoprotein lipase, which in part accounts for the capacity to perform endurance exercise whilst following a high fat diet.

In trained subjects it is also possible that the route of free fatty acid utilisation following mobilisation from fat cells could be more towards the
muscle than the liver. This in part could explain the greater reliance of fat in the trained state.

Research has suggested that the supply of fatty acids from the adipose tissue is the major factor limiting fat oxidation during sustained exercise (Hodgetts, et al. 1991). At higher work intensities a larger proportion of energy is derived anaerobically and the mobilisation of free fatty acids is depressed as long as blood lactate levels remain high. However, up to workloads of 70-80% VO2max, exercise activates an increase plasma free fatty acids which increases substrate availability, as exercise is prolonged. Even at workloads as high as 88.5% the energy derived from fat is still greater than 10% (Pruett 1970). It is possible that the oxidation of intramuscular lipids may, in part, be responsible for this effect. Increased intramuscular triglyceride stores may thus provide an advantage, allowing increased fat utilisation during heavy exercise and the ability to continue exercising for longer duration.

5.4.2 Perceived performance

In order to evaluate the perceived exercise performance of the subjects whilst following the intervention diets in the present study a questionnaire was sent to each participant. This questionnaire was sent after the completion of dietary intervention and required the participants to rate their individual cycling performance whilst following the study diets. Subjects rated their performance on a scale of one to ten. The high fat diet group scored a slightly higher rating with an average of 7 ± 2 compared to that of the high carbohydrate diet group who averaged 6 ± 2. This demonstrates that those following a high fat diet did not consider their physical performance to be compromised by such a dietary regimen. Other investigators have noted that the consumption of a diet high in fat is not only detrimental to performance, but is not well tolerated (Christensen and Hansen 1938). It is possible that in such studies the proportion of fat was too high or sufficient adaptation to the diets were not allowed. There was no evidence in the present study that the high fat diet was not well tolerated. In fact the post-study questionnaire indicated that many participants preferred the high fat diet.
5.5 Implications

One of the major findings of the present study is that the consumption of a high carbohydrate may not be necessary to optimise endurance performance.

The current study, along with previous research, has demonstrated that humans can perform endurance exercise despite the consumption of a low carbohydrate-high fat diet, and presumably low muscle glycogen concentrations. In this study, endurance capacity was no different in cyclists consuming either a HF or HC diet.

The present research shows that the need for a low fat diet for the reduction of CHD risk, is less applicable to endurance athletes than the population in general. Exercise was an important determinant of the lipid and lipoprotein profiles in these highly trained individuals.

Previous researchers have stated that the athlete is as susceptible to the adverse cardiovascular effects of dietary cholesterol and saturated fat as are the general population. Leaf et al (1989) suggested that the fat intakes of athletes should be kept within 20-30% of total calories, with saturated fat supplying no more than one-third of this amount. This, however, seems to be unnecessary in view of the findings of the present research. Here, 45-50% of dietary calories were provided from fat, of which was predominantly saturated fat. Such a dietary regime did not have a detrimental effect on lipid or lipoprotein profiles.

These results have important implications regarding the dietary recommendations given to endurance athletes. High carbohydrate diets are extensively recommended as the correct dietary practice to optimise endurance performance. However there is a suggestion, from this study as well as previous research, that increasing the relative amounts of fat in the diet is certainly not detrimental to endurance performance.

There are several ways in which a diet relatively higher in fat may prove to be advantageous. Firstly, with the high energy requirements of endurance athletes, a higher fat diet, which is more energy dense, provides an easier means of consuming such high energy requirements. Some reported energy expenditure values during endurance and ultra-endurance events range from 22.9-75.2MJ/day (Kreider 1991). It is difficult to consume large energy intakes with the consumption of a 60-70% carbohydrate diet. In such situations it is necessary to complement with large quantities of simple sugars (mainly as commercial high carbohydrate fluids).
Glycogen stores require several days for repletion (i.e., after depletion). During a multi-day event, if consuming a high carbohydrate diet, glycogen stores are utilised at a faster rate and time for repletion is limited. However, even in very lean individuals there is an appreciable caloric store as fat, and when adapted to a high fat diet there is an increased contribution of fat to oxidative metabolism. Thus, with adaptation to a higher fat diet, there is a potential benefit for athletes who are participating in prolonged endurance exercise over several days. There is an induced reliance on the body's extensive fat stores which in turn will spare glycogen stores and enhance endurance.

A question arises out of such research regarding the type of food to consume during training. Although many researchers support the concept of the consumption of carbohydrate during prolonged exercise, evidence exists to the contrary. Arieli et al. (1985) investigated the effects of food intake on fatigue. It was discovered that fat, rather than sugar may delay exhaustion. This is compatible with the theory of training induced reliance on fat metabolism. Carbohydrate ingestion prior to and during exercise impedes the mobilisation of free fatty acids and this may reduce time to exhaustion. The consumption of fat during exercise in place of carbohydrate to enhance endurance is an interesting concept and warrants further investigation.

In the present study physical performance was similar with the consumption of a high carbohydrate diet and a diet relatively high in fat. The beneficial effects of training on lipids and lipoproteins outweighed the adverse effects of extreme dietary intakes. Endurance athletes should feel confident from the results of this study that a diet relatively high in fat will not be associated with adverse effects. Thus it seems that strict fat restrictive advice is unnecessary for endurance athletes. A dietary regime of the athlete's preference appears to be more appropriate.
6 Conclusion

Diet and exercise both effect blood lipids and lipoproteins in humans. Results from the present study indicate that a high level of endurance activity exerts an effect on plasma lipids/lipoprotein that outweighs the adverse effects of extreme dietary intakes. Factors associated with physical training are largely responsible for the lipoprotein pattern evident in the endurance cyclists in the present study. This was characterised by relatively low levels of total and LDL cholesterol and triglycerides, with high concentrations of the HDL fraction.

A high fat diet is not generally recommended for endurance athletes because of the potential adverse effects on plasma lipid and lipoprotein profiles, and because it is believed that the consumption of a high carbohydrate diet is essential in maximising glycogen stores and enhancing endurance performance.

The results of this study challenge these classical beliefs. Lipoprotein profiles of the endurance athletes in the present study, consuming a relatively high fat (particularly saturated fat) diet (45 to 50% of energy derived from total fat; 35 to 40% of energy from CHO), were not significantly different from cyclists consuming a carbohydrate rich diet (60 to 65% of energy derived from CHO; 15 to 20% of energy from total fat). Throughout dietary intervention, both groups underwent favourable alterations in lipid profiles. Total and LDL cholesterol, along with apolipoprotein B were significantly reduced and apolipoprotein A1 increased significantly throughout the study period.

Increases in physical fitness and training, observed throughout the intervention period are likely to have resulted in the alterations in the lipoprotein profiles. No significant changes in body weight or composition were evident throughout the duration of intervention and so can not account for the changes in the lipid/lipoprotein profiles.

Therefore, in endurance athletes involved in large amounts of high intensity exercise, physical exercise is a very important determinant of plasma lipids and lipoproteins.
Of interest is the finding that the consumption of a high fat diet for a period of twelve to sixteen weeks did not adversely affect physical performance capacity. Physical fitness and endurance capacity was similar between the two diet groups.

The results found in this study, along with previous research, contribute to the growing acceptance that humans can adapt to a diet high in fat without compromising endurance capacity. Adaptation to a high fat diet appears to be similar to the adaptation to endurance training. That is, individuals experience an increased mitochondrial oxidative capacity of both the Krebs cycle and beta-oxidative enzymes. Such changes increase the relative contribution of fat to oxidative metabolism.

A diet relatively high in fat may offer certain advantages for endurance athletes, in that it is energy dense, thus, providing an easier means of consuming the high energy demands of endurance athletes. Such a diet may be efficacious in a situation where energy expenditure is high and time for recovery is limited.

Subjects in the present study were highly trained endurance athletes, involved in large amounts of high intensity exercise and therefore the results obtained in this study cannot be extrapolated to the general population. They do however, have considerable relevance to endurance athletes.

This study has provided the basis for research investigating the prolonged interactive effects of diet and exercise on plasma lipids and physical performance.

It seems reasonable to suggest that strict dietary advice during periods of high intensity training is unnecessary for endurance athletes. Such athletes should be advised to choose a dietary regimen of their choice within the range tested in this study. Increasing fat intake in order to achieve the high energy demands of endurance training is a reasonable proposal.

To conclude, it is evident that both diet and exercise influence plasma lipid and lipoprotein concentrations. In highly physically active individuals, such as those in the present study, exercise appears to blunt the adverse effects of extreme dietary intakes on plasma lipids and lipoproteins.
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Appendices
A Laboratory Data Appendix
Table 1  Plasma lipid and lipoprotein profile of HC and HF following four weeks of intervention, *n=20* and *n=15*. All values are means ± SD

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.54 ± 0.91</td>
<td>5.04 ± 0.86</td>
<td>0.11</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>2.66 ± 0.91</td>
<td>3.06 ± 0.70</td>
<td>0.16</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.33 ± 0.29</td>
<td>1.41 ± 0.29</td>
<td>0.42</td>
</tr>
<tr>
<td>HDL2 cholesterol (mmol/L)</td>
<td>0.60 ± 0.17</td>
<td>0.67 ± 0.24</td>
<td>0.37</td>
</tr>
<tr>
<td>HDL3 cholesterol (mmol/L)</td>
<td>0.76 ± 0.17</td>
<td>0.75 ± 0.15</td>
<td>0.83</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.13 ± 0.44</td>
<td>1.15 ± 0.39</td>
<td>0.90</td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>106.2 ± 17.4</td>
<td>111.3 ± 8.4</td>
<td>0.30</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>66.4 ± 19.4</td>
<td>72.7 ± 17.1</td>
<td>0.32</td>
</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.29 ± 0.09</td>
<td>0.29 ± 0.08</td>
<td>0.97</td>
</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.54 ± 0.26</td>
<td>0.49 ± 0.19</td>
<td>0.52</td>
</tr>
</tbody>
</table>

P-values for differences between HC and HF as determined by independent two-tailed *t*-tests.

Table 2  Plasma lipid and lipoprotein profile of HC and HF following eight weeks of intervention, *n=20* for HC and *n=15* for HF. All values are means ± SD

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.69 ± 0.89</td>
<td>5.04 ± 0.70</td>
<td>0.22</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>2.78 ± 0.77</td>
<td>3.06 ± 0.56</td>
<td>0.25</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.36 ± 0.30</td>
<td>1.36 ± 0.27</td>
<td>0.97</td>
</tr>
<tr>
<td>HDL2 cholesterol (mmol/L)</td>
<td>0.58 ± 0.19</td>
<td>0.59 ± 0.16</td>
<td>0.87</td>
</tr>
<tr>
<td>HDL3 cholesterol (mmol/L)</td>
<td>0.78 ± 0.15</td>
<td>0.77 ± 0.13</td>
<td>0.96</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.18 ± 0.38</td>
<td>1.38 ± 0.72</td>
<td>0.31</td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>113.8 ± 15.4</td>
<td>113.2 ± 10.3</td>
<td>0.90</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>71.6 ± 16.9</td>
<td>77.9 ± 13.3</td>
<td>0.27</td>
</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.31 ± 0.09</td>
<td>0.27 ± 0.06</td>
<td>0.27</td>
</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.54 ± 0.22</td>
<td>0.46 ± 0.13</td>
<td>0.21</td>
</tr>
</tbody>
</table>

P-values for differences between HC and HF as determined by independent two-tailed *t*-tests.
Table 3  Plasma lipid and lipoprotein profile of HC and HF following twelve weeks of intervention, n=20 for HC and n=14 for HF. All values are means ± SD

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.49 ± 0.93</td>
<td>4.61 ± 0.68</td>
<td>0.69</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>2.64 ± 0.88</td>
<td>2.73 ± 0.50</td>
<td>0.73</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.36 ± 0.34</td>
<td>1.35 ± 0.35</td>
<td>0.91</td>
</tr>
<tr>
<td>HDL2 cholesterol (mmol/L)</td>
<td>0.55 ± 0.25</td>
<td>0.56 ± 0.16</td>
<td>0.89</td>
</tr>
<tr>
<td>HDL3 cholesterol (mmol/L)</td>
<td>0.83 ± 0.11</td>
<td>0.80 ± 0.13</td>
<td>0.41</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.19 ± 0.45</td>
<td>1.18 ± 0.48</td>
<td>0.99</td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>115.7 ± 16.4</td>
<td>112.3 ± 12.9</td>
<td>0.53</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>70.4 ± 17.3</td>
<td>73.1 ± 13.4</td>
<td>0.62</td>
</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.31 ± 0.07</td>
<td>0.30 ± 0.05</td>
<td>0.53</td>
</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.57 ± 0.20</td>
<td>0.51 ± 0.13</td>
<td>0.33</td>
</tr>
</tbody>
</table>

p-values for differences between HC and HF as determined by independent two-tailed t-tests.

Table 4  Plasma lipid and lipoprotein profile of HC and HF following sixteen weeks of intervention, n=15 for HC and n=7 for HF. All values are means ± SD

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.28 ± 0.66</td>
<td>4.65 ± 0.67</td>
<td>0.24</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>2.51 ± 0.59</td>
<td>2.74 ± 0.59</td>
<td>0.39</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.36 ± 0.25</td>
<td>1.50 ± 0.33</td>
<td>0.30</td>
</tr>
<tr>
<td>HDL2 cholesterol (mmol/L)</td>
<td>0.54 ± 0.17</td>
<td>0.64 ± 0.20</td>
<td>0.24</td>
</tr>
<tr>
<td>HDL3 cholesterol (mmol/L)</td>
<td>0.82 ± 0.12</td>
<td>0.82 ± 0.15</td>
<td>0.51</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>0.97 ± 0.33</td>
<td>0.75 ± 0.26</td>
<td>0.93</td>
</tr>
<tr>
<td>Apo A1 (mg/dl)</td>
<td>111.1 ± 13.4</td>
<td>115 ± 8.9</td>
<td>0.47</td>
</tr>
<tr>
<td>Apo B (mg/dl)</td>
<td>75.8 ± 36.0</td>
<td>67.9 ± 7.2</td>
<td>0.58</td>
</tr>
<tr>
<td>HDL/Total cholesterol ratio</td>
<td>0.32 ± 0.07</td>
<td>0.31 ± 0.07</td>
<td>0.75</td>
</tr>
<tr>
<td>HDL/LDL cholesterol ratio</td>
<td>0.58 ± 0.20</td>
<td>0.54 ± 0.18</td>
<td>0.64</td>
</tr>
</tbody>
</table>

p-values are for differences between HC and HF as determined by independent two-tailed t-tests.

Table 5  Iron status at baseline for HC and HF. All values are means ± SD. Ranges are shown in ( ).

<table>
<thead>
<tr>
<th></th>
<th>HC</th>
<th>HF</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin (g/l)</td>
<td>156.9 ± 7.8 (141.3 - 175.7)</td>
<td>156.5 ± 7.0 (145.2 - 171.1)</td>
<td>0.86</td>
</tr>
<tr>
<td>Ferritin (µg/l)</td>
<td>121.7 ± 68.8 (24 - 324)</td>
<td>102.1 ± 66.0 (16 - 222)</td>
<td>0.41</td>
</tr>
</tbody>
</table>

p-values are for differences between HC and HF as determined by independent two-tailed t-tests.
We would like to thank you for your interest in this Sports Nutrition Study. Outlined below is information regarding the study.

I am sure you are all aware of the relationship between cholesterol levels and heart disease. Numerous studies have shown that regular physical exercise can decrease the risk of heart disease. There are many explanations for the protective effect of exercise on heart disease, including the beneficial changes in cholesterol levels. For most of the population, high cholesterol levels are due to a high intake of fat (particularly saturated fat). However, of interest is a finding that people who are very fit e.g. endurance trained athletes, tend to have lower cholesterol levels than people who do not do any exercise - even if the athlete's diet is relatively high in fat.

We aim to investigate this observation. Our aim is therefore to investigate the effects of different diets (varying in the amounts of fat and carbohydrate) on blood cholesterol levels in endurance trained cyclists.

The results of this research could provide important information as to future nutritional guidelines for endurance athletes.

For this study we plan to recruit around 30 Otago cyclists of whom are endurance trained. The definition of an endurance trained athlete is one who undergoes at least 1 hour of training for 5 days of the week.

Subjects will be divided randomly into 2 groups. Group 1 will receive a high carbohydrate diet and group 2 will receive an energy dense diet. The diets should not be too different from your current diet and are within the current recommended intakes for endurance athletes. They consist of normal foods and will provide all essential nutrients. We would ask you to follow the diet guidelines for 12-16 weeks. Cholesterol levels will be monitored throughout this period.
BENEFITS FOR YOU

1. **Exercise testing:**
   During this 12-16 week study you will have 2 VO2max tests performed at the performance laboratory, University of Otago. This will provide you with excellent feedback as to the effectiveness of your training and level of physical fitness throughout the cycling season. We will monitor body composition change also, including % body fat and body weight.

2. **Iron status:**
   Iron deficiency anaemia may also be an explanation for poor racing performance. We will take blood tests which will provide information about your iron levels.

3. **Cholesterol levels:**
   Blood tests on 4 occasions will monitor your cholesterol levels.

4. **Free training food:**
   Energy bars will be provided throughout the study. During diet (HC) high carbohydrate bars will be provided and during the Energy Dense diet, high energy bars will be provided.
YOUR INVOLVEMENT IN THE STUDY

We plan to start the diet period on 17th August. It will run for 12-16 weeks and therefore end in early November / December

Prior to the diet period we will collect baseline information from you. This will involve blood cholesterol tests, an exercise test and we would ask you to complete a 5-day food diary. This record of your usual intake will enable us to modify your current diet to meet the requirements of the study diets.

1. STUDY DIETS:
The study diets will consist of 2 diets which will differ in the amounts of carbohydrates and fat.

1. High carbohydrate diet (HC) - this will be 65-70% carbohydrate. Therefore you will have to restrict your fat intake.

2. Energy Dense diet (ED) - this will be 45-50% fat. Therefore you will be required to achieve a specific fat intake.

Dietary advice and guidelines will be provided for you throughout the study. Both diets will be designed, based on your current diet so that total calories will be matched. Therefore the amount of calories on the HC and ED diets will be the same as what you would usually consume.

Remember the study diets are not extreme. The diets use ordinary foods and contain all essential nutrients.

The diet period will last 12-16 weeks to allow for adaptation to each diet.

We would ask you to complete three, 5-day food diaries. One as a baseline measure, as mentioned previously. A further 2 diaries will be required throughout the 16 weeks of the study period so we can assess your actual intake.
2. **BLOOD TESTS:**
Baseline blood samples will be taken whilst you are on your current diet during the week leading up to the commencement of the study diet period.

During the study period we will take blood samples at weeks 5,9, and 13. So we can monitor any changes in your blood cholesterol levels, accurate values will be needed. Therefore we will take 2 blood samples a each measurement period, on 2 different days.

**Therefore** - total number of blood tests = 8 over a 12 week period

**Fasting** blood samples are needed. Therefore please do not eat anything after 9pm the night before your blood sample is to be taken.(water is allowed).

**Also** please do not do any exercise on the morning the blood sample is to be taken.

From these blood samples we will also be able to provide you with information on your iron status.

3. **EXERCISE TESTING:**
Exercise testing will involve the measurement of your VO2max (measure of exercise performance based on high intensity cycling). Testing will be done at the Human Performance Laboratory. Each visit will last about an hour.

Testing will be done during the baseline period before the study diet begins, and at weeks 5, 9 and 13 of the diet study period.

4. **BODY MEASUREMENTS:**
Measurements of your body weight, height, and skin folds (for total body fat) will be recorded during the baseline period before the commencement of the study diets. Additional body measurements will be made at weeks 5, 9 and 13.

5. **TRAINING DIARY:**
We would ask you to keep a training diary in which you will record your weekly milage and total hours of training.

Once again, thankyou for your interest in this study which will enable us to obtain information on the effects of diet on blood cholesterol levels in endurance athletes.
ENERGY DENSE

WELCOME TO THE SPORTS NUTRITION STUDY

THANK YOU FOR PARTICIPATING.

IN THIS BOOKLET YOU WILL FIND:

1. Appropriate foods / foods to avoid.

2. Example of a menu plan.

3. Foods to eat / drink while training.


5. Recipes.

6. Training record.

7. Performance record.
HIGH CARBOHYDRATE DIET

WELCOME TO THE SPORTS NUTRITION STUDY

THANKYOU FOR PARTICIPATING.

IN THIS BOOKLET YOU WILL FIND:

1. Appropriate foods / foods to avoid.

2. Example of a menu plan.

3. Foods to eat / drink whilst training.


5. Recipes.

6. Training record.

7. Performance record.
D Sample of dietary guidelines

ED

FOODS

PLEASE USE:-

- Toasted muesli and cereals
- High fat breads, eg. croissants, cheese bread, pastry, commercial baked products
- Potato, rice, pasta - prepared with extra fat eg. added butter
- Peanut butter, chocolate spread, lemon honey, tahini
- High fat cheeses - cheddar, cream cheese, blue cheeses
- Cream, full cream milk, ice cream, butter
- Chocolates, toffee, high fat baking
- Meat fat, fatty fish, fried foods, chicken skin, pork crackling
- Nuts, seeds
- Add butter to vegetables eg. stir fry
- Salad dressing
- avocado
- fresh fruit

PLEASE DO NOT USE:-

- Trim milk, cottage cheese
- Honey, jam, boiled sweets
- Low fat varieties of regular products eg. yogurt
- Fruit - dried or tinned in syrup
- High carbohydrate drinks eg. exceed
- Soft drinks
HC

FOODS

PLEASE USE:
- untoasted muesli - cereals
- low fat breads, rolls, pita bread, crumpets
- potato, rice, pasta (cooked without fat)
- honey, jam, syrup, treacle
- Beans - baked, harricot, lima, lentils, split peas
- Root vegetables - especially corn, kumera, yams, parsnips
- Fruit - especially dried, tinned in syrup
- trim milk, low fat yogurt (especially flavoured)
- cottage cheese, mozzarella, Edam
- White fish, low fat meat, white chicken meat
- water ices
- High carbohydrate drinks eg. exceed
- less than 1/2 teaspoon of margarine (preferably reduced fat eg. Vital, Slimarine) per slice of bread or 1/2 roll.
- boiled sweets, wine gums, barely sugars, jelly beans

PLEASE DO NOT USE:-

- Toasted cereals, pastry, high fat breads, croissants, Commercial baked products with high fat content.
- Fried foods, meat fat, high fat cheeses (eg. cheddar cheese
  cream cheese, blue cheese), chicken skin
- Toffees, chocolate
- Cream, ice cream (except weight watchers)
- Full cream milk
- peanut butter
E Sample of daily menu plan

ED

EXAMPLE OF MENU

BREAKFAST:
High fat muesli
banana
bread
butter
peanut butter
cream
Full cream milk

MID-MORNING SNACK
High fat baking eg. short bread

LUNCH
Bread or crispbread
butter or cream cheese
high fat cheese
salami
green salad
apple
full cream milk

MID-AFTERNOON SNACK:
chocolate bar

DINNER
Roast beef
potato (roasted or mashed with butter)
silverbeet
peas
full cream milk
Dessert - cheese cake

SUPPER
Hot chocolate drink
Baking eg. shortbread
cream
EXAMPLE OF MENU

BREAKFAST
Plain muesli
toast
margarine
jam or honey
low fat yogurt
trim milk
fruit - banana
sultanas

MID-MORNING SNACK
Low fat plain biscuit OR
fruit juice

LUNCH
Roll
meat or cheese
salad
margarine
fruit juice
sultanas

MID-AFTERNOON SNACK
fruit - orange
high carbohydrate snack eg. dates

DINNER
lean beef steak
potato or pasta
root vegetable eg. yams
green vegetables or salad
High carbohydrate dessert eg. meringues (no cream)
tinned fruit salad

SUPPER
Low fat baking, fruit OR
juice
boiled sweets
Sample of recommended training foods for subjects

TRAINING FOODS

Examples of what you can eat/drink while training:

- water
- chocolate
- milk flavoured eg. chocolate
- muesli bars

Please do not use:

- High carbohydrate drinks eg. Exceed
- Fruit juices
- Soft drinks

FLUID INTAKE

It is very important for you to drink plenty of fluids.
TRAINING FOODS

EXAMPLES OF WHAT YOU CAN EAT / DRINK DURING TRAINING:

- High carbohydrate drinks eg. exceed
- Fruit juices
- bananas
- water

PLEASE DO NOT USE:

- chocolate
- muesli bars

FLUID INTAKE:

It is very important for you to drink plenty of fluids.
G Calorie exchange list for training foods

**CALORIE CHART**

<table>
<thead>
<tr>
<th>FOOD</th>
<th>CALORIES</th>
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<tbody>
<tr>
<td>Power Bar</td>
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<td>Banana</td>
<td>105</td>
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<td>Muesli Bar</td>
<td>130</td>
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<td>1 chocolate bar</td>
<td>300</td>
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<tr>
<td>1 slice bread</td>
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<td>Pear (medium)</td>
<td>55</td>
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<tr>
<td>Carbohydrate drinks:</td>
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<tr>
<td>Exceed</td>
<td>167cal / 600mls</td>
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<tr>
<td>Gatorade</td>
<td>127cal / 600mls</td>
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<tr>
<td>Replace</td>
<td>170cal / 600mls</td>
</tr>
<tr>
<td>Fresh-up</td>
<td>108cal / 600mls</td>
</tr>
<tr>
<td>Dried fruit eg. raisins</td>
<td>126cal / 50g</td>
</tr>
</tbody>
</table>

1. It is important **not** to eat more than you normally would; had we not supplied you with confectionery bars. Therefore please eat the confectionery in place of what you normally would during training. If you normally eat very little or nothing during training then give the confectionery to your friends.
Please estimate from the calorie chart provided, the calories you would normally eat during training and replace this with confectionery supplied.

**FOR EXAMPLE:**
If you normally would eat:
1. One Power Bar per training ride (225 cal) - this could be replaced with 3/4 of a chocolate bar
2. Two bananas per training ride could be replaced by 3/4 of a chocolate bar.
3. 600 mls exceed = 1/2 chocolate bars
4. 600 mls gatorade = 1/2 chocolate bar

If you usually eat confectionery at other times, then feel free to use the confectionery supplied.

2. When you collect your training foods every 2 weeks (Mondays), please weigh yourself. We do not want your weight to change too much ie. not significantly increased or decreased.
# Calorie Chart

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Please estimate from the calorie chart provided, the calories you would normally eat during training and replace this with confectionery supplied.

**FOR EXAMPLE:**

If you normally would eat:

1. One Power bar during training this is = 60g Jet Planes/Jubes/eskimos/Jelly Beans
2. One banana = 30g Jet planes/Jubes/ekimos/jelly Beans
3.600ml exceed = 50g Jet Planes/Jubes/Eskimos/Jelly Beans
4.600ml gatorade = 36g Jet Planes/Jubes/Eskimos/Jelly Beans

[ 1 jet plane = 7 1/2g]
[ 1 fruit jube = 5g]
[ 1eskimo = 8 1/2g]
[ 1 jelly bean = 2g]

If you usually eat confectionery at other times, then feel free to use the confectionery supplied.

2. When you collect your training foods every 2 weeks (Mondays), please weigh yourself. We do not want your weight to change too much, i.e., not significantly increased or decreased.
Sample recipes

Recipes for high carbohydrate diet group

RECIPEs

MUESLI

1 cup rolled oats  
1 cup wheatgerm  
1/2 cup bran  
1/2 cup raisins  
1/2 cup dried fruits  
(chopped apricots, red dates etc)

Mix all ingredients together and store in an airtight jar. Serve with trim milk, low fat yogurt and fruit.

HERBY BEANS

1 medium onion, roughly chopped  
400g peeled tomatoes, roughly chopped  
450g tin three bean mix, rinsed and drained  
1 tsp oregano  
1 Tbsp tomato paste  
freshly ground black pepper

Saute the onion until browned. Add rest of the ingredients. Simmer for 5-10 minutes. This filling is suitable for pasta, rice, potato pile up, pita bread.

BEAN BUSTER

1 Tbsp polyunsaturated oil  
440g tin baked beans in tomato sauce  
1 cup roughly chopped celery  
2 Tbsp natural low fat yogurt  
2 medium onions finely chopped  
freshly ground black pepper

Heat oil in saucepan. Saute the celery and onions until lightly browned and softened. Add baked beans and yogurt. Heat gently until warmed through. Season with pepper. This filling is suitable for potato pile up, kumara split, pita bread.
LEMON AND BROCCOLI FISH
1 small head of broccoli (150g) 4 fish fillets (500g)
2 Tbsp water
LEMON SAUCE
1 Tbsp polyunsaturated margarine 1 Tbsp lemon juice
1 Tbsp flour freshly ground black pepper
3/4 cup chicken stock

Slice broccoli stalk thinly and break the heads into florets. Roll the fillets. Place the broccoli, fish and water in an oven proof dish. Cover and bake at 180 C for 15 minutes. Drain off the cooking liquid. Serve with lemon sauce.

LEMON: Melt the margarine in a small saucepan and stir in the flour. Gradually add the chicken stock, lemon juice and brown sugar. Heat gently stirring continuously for 3-4 minutes or until the sauce thickens. Season with pepper.

FISH CRUNCH
500g fish fillets eg. lemon fish 1 Tbsp grated parmesan cheese
1 cup roughly chopped celery 1/4 cup grated edam cheese
3 medium tomatoes, roughly chopped
1 Tbsp finely chopped fresh thyme or 1/2 tsp dried thyme
1 Tbsp lemon juice
1 Tbsp polyunsaturated oil
freshly ground black pepper
1 cup flaked cereal eg. bran flakes, cornflakes, weetbix

Cut the fish into bite six pieces. Place the fish, celery, tomato, lemon juice and pepper in a greased baking dish. Mix the remaining ingredients together and sprinkle over the fish. Bake a 180 C for 25-30 minutes or until the is white and flakes with a fork.

ORANGE AND YAM SURPRISE
1 kg chicken pieces 1/2 cup chicken stock
8-10 medium yams freshly ground black pepper
2 Tbsp marmalade 1 Tbsp cornflour
1 orange 1 Tbsp water

Remove the skin and all visible fat from the chicken. Place the chicken, yams and the marmalade in a casserole dish. Mix the juice and the grated rind of orange, stock and pepper together and pour over the chicken and yams. Cover and bake at 180 C for 25-30 minutes or until the chicken is tender. Mix cornflour and water to a paste and add to the casserole. Return to the oven for 5 minutes or until the sauce thickens.
CARROT AND ORANGE LOAF
2 medium carrots (200g) peeled and roughly chopped
2 eggs
1 3/4 cups wholemeal flour
2 tsp baking powder
1 tsp mixed spice
1 cup raisins
TOPPING
1 Tbsp brown sugar
Place the carrots, orange rind, sugar, yogurt and eggs in a food processor fitted with metal blade. Process 2-3 minutes or until smooth. Lightly mix in the remaining ingredients. Turn into a lightly greased 21cm by 10cm loaf tin. Sprinkle with the topping. Bake at 180 C for 1- 1 1/4 hours or until an inserted skewer comes out clean.

MERINGUES
4 egg whites
1 tsp vanilla extract
1 cup sugar
dash of salt
Preheat oven to 250 F. Line 2 baking sheets with parchment or waxed paper In a small mixing bowl, beat the egg whites until stiff but not dry. Add the sugar, vanilla and salt; mix well. Drop by the tsp full, about 1 inch apart onto the baking sheets. Bake for 1 1/2 hours.

APPLE CRISP
6-7 cooking apples peeled and sliced
1 Tbsp lemon juice
1 1/4 tsp cinnamon
3/4 cup rolled oats
1 cup brown sugar
1/2 cup white sugar
1/4 tsp nutmeg
1/2 cup plain flour
2 Tbs safflower oil
Preheat the oven to 350 F. Place the apples in a 9 inch deep-dish pie plate. Sprinkle the apples with lemon juice, white sugar, cinnamon and nutmeg. In a small mixing bowl, stir together the oats, flour, brown sugar, and safflower oil; sprinkle over the apples. Bake uncovered for 40 minutes.
CRISPY BAKED RHUBARB
225g rhubarb cut into 1 cm lengths  100g granulated sugar
2.5 ml ground ginger
FOR TOPPING:
25g margarine  50g white flour
25g rolled oats  50g light brown sugar

Place the rhubarb in an ovenproof pie dish and sprinkle with the sugar and cinnamon.
For the topping, rub margarine into the flour and stir in the oats and sugar. Sprinkle the topping over the rhubarb and bake in the oven at 180 C for 40-45 minutes until the topping is golden brown and the rhubarb is tender.

BLACKCURRENT SORBET
300ml water  100g sugar
225g blackcurrents  5ml lemon juice
2 egg whites

Heat water and sugar together in a saucepan, stirring until the sugar has dissolved. Bring to the boil and simmer gently for 10 minutes. Cool. Meanwhile, simmer the blackcurrents in a little water for 10 minutes until tender. Rub through a sieve and if necessary make up the puree to 300ml with water. Cool, add the lemon juice and sugar syrup and pour into an ice tray. Freeze for about 1 hour until nearly firm.
Whisk the egg whites until stiff. Turn the half-frozen mixture into a chilled bowl and whisk until smooth. Fold in the eggs whites, return to the ice tray and freeze until firm.
Recipes for the high fat diet group.

RECIPE

CARROT SALAD
2 large carrots, grated 1/4 cup coconut
1/4 cup sunflower seeds

1. Grate the carrots into a bowl
2. Mix in sunflower seeds
3. A few minutes before serving mix in coconut

STIR FRY VEGETABLES
Chop vegetables of your choice into small pieces. Heat pan or wok on moderate heat. Use 2 Tbl butter for 2 people. Use herbs to flavour. Cook until hot but still crisp.

COCONUT CREAM CHICKEN
1.5 kg chicken 50g desiccated coconut
600 ml cream 3 spring onions
2 cloves garlic, crushed 3 Tbs peanut butter
finely grated rind of 1 lemon 1 tsp coriander
1/2 tsp chilli powder 50g butter
1 Tbs soy sauce

Chop the chicken into 8 portions. Put the coconut into a bowl, heat the cream until it bubbles begin to rise and pour on the coconut. Put aside until cold then strain, pressing the coconut well to extract all the liquid.
Grind the spring onions, garlic, peanut butter, lemon rind, coriander, chilli together with a pestle and mortar or an electric blender.
Coat the chicken pieces in the ground mixture. Heat the butter in a large frying pan and fry chicken until browned. Add the coconut flavoured cream and soy sauce. Bring to the boil and simmer for 30 minutes or until chicken is tender.

TUNA FISH PIE
1 can condensed creme of mushroom, celery or chicken soup.
3-6 Tbl water
1 can tuna fish
packet of potato crisps
1 small can of peas
pinch pepper
2 tsp lemon juice
1. Heat soup with water
2. Add flaked tuna fish
3. Crush potato crisps and add to soup with drained peas, lemon juice and pepper.
4. Pour into a casserole
5. Edge with remaining crisps and bake in moderate oven for 25 minutes.

**POTATO SCALLOPS**
1. Peel medium sized potatoes
2. Cut into slices approx. 3/8 inch thick and drop into cold water.
3. Drain and dry.
4. Dust all the sides with seasoned flour.
5. Fry gently in hot shallow fat until golden brown and tender.

**SAUSAGE HOTPOT**
- 450g pork sausages
- 1 packet of frozen mixed vegetable
- 1 can tomatoes
- 1 Tbs worcestershire pepper
- 750 mls water
- 450 potatoes

Grill sausages quickly until browned all over and put into a casserole. Add the mixed vegetables, tomatoes, tomato puree, Worcestershire sauce and water or stock and season to taste. Parboil the potatoes in boiling water and cut them into 5mm slices. Overlap the potatoes on top of the casserole and dot with the butter. Bake in moderate oven (180 C), for 1 hour.

**SHORTCRUST PASTRY**
- 200g plain flour
- pinch salt
- 100 g butter
- about 2 Tbs cold water

Sift flour and salt into a mixing bowl. Add the butter and lard, cut into small pieces. Rub the fat into the flour using your fingertips until the mixture resembles fine breadcrumbs. Add cold water and mix with a fork to make a crumbly dough. Add a little more water if necessary. Using your hand, knead dough to make a firm ball.
SWISS CHEESE FLAN

175g shortcrust pastry 350g chopped bacon
1 onion, chopped 100g cheese, grated
150 ml full cream milk 6 Tbs yogurt
25g plain flour 2 egg yolks
pepper pinch of grated nutmeg
1 egg white

Make pastry and use to line a 20 cm flan ring.
Fry the bacon. Add onion and cook until softened. Spread over the prepared flan case and spread the cheese on top.
Beat together the milk, yogurt, flour, egg yolks, seasoning and nutmeg. Whisk the egg white until stiff and fold into the mixture. Pour into the flan case and bake straightaway in a moderately hot oven (190 C 375 F, Gas Mark 5) for 20 minutes. Reduce the temperature to moderate (180C, 350F, Gas Mark 4) and cook for a further 30-40 minutes, or until set. Serve straight from the oven.

NEVER-FAIL CHEESE SAUCE

1 can condensed creme of chicken, mushroom or celery soup
8oz grated cheese
3-6 Tbsp full cream milk

1. Warm condensed soup; add grated cheese and stir till smooth. Add 3-6 Tbl milk to bring the sauce to the consistency you need.

GOLDEN MEAT LOAF

200g shortcrust pastry beaten egg to glaze
450g minced steak 225g pork sausagemeat
1 onion, finely chopped 100g fresh breadcrumbs
1 tsp thyme pepper
1 egg
4 Tbl tomato sauce

Roll out pastry into a large oblong 5mm thick.
Pull all the meat loaf ingredients into a bowl and mix together very well. Press into a loaf shape and put in the centre of the pastry. Damp edges of the pastry with cold water and encase the meat pressing the edges firmly together to seal. Fold the edges under the meat loaf to enclose it completely. Put on a baking sheet and glaze with the beaten egg. Bake in a moderate oven (180 C) for 1 1/2 hours.
SHORTBREAD
100g butter 50g caster sugar
150g plain flour 25 semolina or rice flour

Grease an 18cm shallow cake tin
Cream the butter and sugar together until light and fluffy. Sift in the flour with the semolina or rice flour. Stir in, mixing with your hand, and knead to a smooth dough.
Press the dough into the cake tin. Prick the top with a fork, flute the edges using a thumb and mark into 8 wedges with a knife
Bake in a moderate oven (160 C) for 35-40 minutes.

BAKED CHOCOLATE ALMOND CHEESECAKE
225g biscuits, crushed 75g butter, melted
filling:
2 eggs separated 75g castor sugar
225g cream cheese 25 g ground almonds
150ml whipping cream 25g cocoa powder

First prepare the crumbcrust. Mix the ingredients together and press over the base and up the sides of a deep 20-cm round cake tin
To make filling, whisk the egg yolks and sugar together until light and fluffy. Beat in the cream cheese and ground almonds with the cream and cocoa. Whisk egg whites until stiff and fold in. Put the filling into the crumbcrust and bake in a moderate oven (160 C) for 30-40 minutes, or until firm.
I Weekly training record completed by subjects

**WEEKLY TRAINING RECORD**

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### PERFORMANCE RECORD

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* PERFORMANCE SCALE:-

1 5 10

awful average excellent

RATE YOU PERFORMANCE ON A SCALE OF 1-10 IN THE ABOVE TABLE
Sports nutrition information pamphlets supplied to subjects
FLUIDS

Water is the single most important food for physical performance.

WHY IS WATER SO IMPORTANT?

- Every living cell requires water to function
- Water is needed for:
  - Blood circulation
  - Removal of waste products
  - Maintaining body temperature control and sweating
  - The transport of nutrients

A man is composed of about 55% water and a woman is composed of about 50% water. The average man contains approximately 42 litres of water.

FLUID BALANCE

![Fluid Balance Diagram]

Water balance in humans.
FLUID LOSS

Excessive fluid loss results in dehydration which hinders performance.

During heavy exercise water loss can be up to a litre per hour. Athletes can lose up to 2-8% of body weight in fluid during heavy exercise. This fluid must be replaced to prevent dehydration which will prevent peak performance.

Dehydration occurs with a loss of about 1% body weight. A loss of 2% or more in body weight leads to a decrease in blood volume, an increase in heart rate and impairs body temperature control, and thus decreases work capacity.

MAINTAINING FLUID BALANCE

You should drink prior to, during and after an event.

Make sure you drink plenty of water on the 2 days leading up to the race.

Fluids should be consumed up to 30 minutes before exercise (400 - 600 mls depending on the individual).

During the event small amounts of fluid should be taken often eg. 100-150mls (1/2 cup) every 10 to 15 minutes.
**Do not wait until you are thirsty!** This is too late. During exercise the thirst mechanism is too slow in indicating fluid loss.

TYPE OF FLUID

- **Water** is best for replacing fluid loss

- **Cold water** is the best because it empties from the stomach quicker and so can be used faster.

- **Sports drinks** are popular, but **not essential**. Some Sports drinks are poorly designed, containing too much sodium and potassium (electrolytes). Therefore if you use the products make sure they are low in electrolytes i.e. no more than 230mg/L of sodium (Na) or 195mg/L of potassium (K).
- Some glucose drinks are too concentrated and slow the rate of water emptying from the stomach. Therefore you should dilute these Sports drinks and other commercial drinks. They should be diluted to about a 2-5% glucose concentration.

- Glucose polymer drinks contain about 5% glucose and is in a form which is able to empty from the stomach as fast as plain water.

SALT
Salt is not necessary in the replacement fluid. It is present in plenty of foods so can be easily replaced. In fact contrary to popular belief, sweat is quite dilute and contains only about 1/3 as much salt as other body fluids. The relative concentration of salt (sodium) in the blood does not fall as a result of sweating. Taking salt tablets during exercise can dehydrate you further. Therefore salt tablets are not recommended at all.

HINTS:
- After training or race events, make sure you drink water before starting into beers. Drinking alcohol at the completion of exercise can dehydrate you even more, leaving you feeling very sick. Therefore drink water first to rehydrate yourself, then alcohol.

- Drinking alcohol the night before a race is not recommended as it dehydrates you even more.

- If you don't like the taste of plain water, squeezing a few drops of lemon juice into water can make it more palatable.
A SPORTS NUTRITION MESSAGE

IRON

What is Iron needed for?
Iron has many functions in the body:
- In red blood cells iron is a part of haemoglobin which carries oxygen to all body tissues.
- Iron is also an essential part of the chain of chemical reactions which produce energy in the body

What happens when Iron Levels are low?
- A reduction of iron levels leads to a lowered oxygen supply to muscles
- Low iron levels can impair endurance. Iron deficiency decreases work capacity by influencing oxygen uptake and muscle metabolism. Hence during exercise the heart finds it difficult to pump the amount of blood required.

Anaemia is a problem to which athletes are susceptible. It can be difficult to detect. Often the only symptom is an otherwise unexplained falling away in performance.

STAGES OF ANAEMIA

IRON DEPLETION - The body has small stores of iron in the liver, spleen and bone marrow. The first step in the development of anaemia is the depletion of these stores. (This is assessed by a blood test measuring serum ferritin (stored iron))

IRON DEFICIENCY - Once iron stores are depleted, the level of iron in the blood falls. This stage is known as iron deficiency.

ANAEMIA - When iron levels drop to the point where haemoglobin is less than 140g/L, iron deficiency anaemia occurs.
SYMPTOMS
- First symptoms of iron deficiency are a 'washed-out' feeling, weakness, fatigue, a reduced ability for physical activity, headaches, cramps, breathing difficulties and impaired performance
- A full blown iron deficiency leads to anaemia, with symptoms of weakness, shortness of breath, coldness, palpitations and 'pins and needles' in the feet.

IRON LOSS
Iron is lost through cells in the gut, skin and hair
The total loss of iron from the body per day = 1mg

The following contribute to a decrease in iron stores:-
- Normal or abnormal blood loss
- Inadequate nutrition eg. poorly planned vegetarian diet or unbalanced food intake over a period of time.
- Poor absorption of iron from the gut and increased loss associated with heavy sweating
- Loss in urine
- Destruction of red blood cells

In addition athletes can lose iron in the following ways:-
- Athletes can sweat 2-3 litres per day and thus can double the rate of iron loss through sweat
- Exercise leads to an increase loss of iron through urine. It seems the stress of exercise damages the wall of the bladder resulting in small amounts of bleeding
- Direct trauma to muscles eg. foot muscles from the pounding they receive during running, also leads to some destruction of blood cells.
- Endurance athletes have a relatively large blood volume which may result in 'relative anaemia'.

TYPES OF IRON
THERE ARE TWO TYPES OF IRON:

Haem Iron - This is found in animal foods and is absorbed better than that found in plant sources. (10-40% is absorbed)

Non-Haem Iron - This is found in plant foods and is not as well absorbed as haem iron (5-10% is absorbed)

In general, the redder the meat the higher the iron content.
HOW TO REDUCE THE RISK OF IRON DEFICIENCY
- Eat a variety of cuts of meat eg. beef, lamb, pork, liver, poultry and fish
- Choose wholegrain breads, pasta, rice and cereals
- Vitamin C enhances iron absorption. Therefore serve foods which are high in Vitamin C with meals eg. kiwifruit, oranges, strawberries, rockmelon, green peppers, tomatoes, cauliflower, puha and potatoes. Another helpful hint is to drink a glass of orange juice with your meals. This can increase your iron absorption up to 250%.
- Tannic acid in tea and polyphenols in coffee decrease iron absorption. Therefore do not drink these beverages with your meals.
- Iron in vegetable protein (non-haem iron) can be absorbed better if combined with animal protein. Some good combinations are spinach and liver; spinach and egg; chillibeans and beef; lentil soup and chicken.
- Cooking in cast-iron pots or skillets increase iron content of foods.

SUPPLEMENTS
With careful attention to diet, iron deficiency can be prevented. Endurance athletes usually consume large amounts of calories and so will usually receive adequate amounts of iron.

Iron supplementation should be reserved only for those whom anaemia has been diagnosed and who cannot get adequate iron from their diet to correct the deficiency.

Iron tablets should be taken on an empty stomach, about half an hour before a meal with water or orange juice, but not milk. Iron tablets are not without some side-effects, the most common is constipation. Iron supplements should not be taken routinely.

RECOMMENDED DAILY INTAKES OF IRON
MEN - 10mg
TEENAGERS - 12mg
WOMEN - 12mg
CHILDREN - 10mg
MEASUREMENTS OF IRON STATUS

- Iron depletion can be assessed by measuring iron stores. This is done via a blood test, measuring serum ferritin (stored iron). Low ferritin in the blood is the first sign of iron deficiency.

Normal ranges of serum ferritin for males is = 20-350ug/L
Less than 10ug/L ferritin = deficiency

- Haemoglobin measures iron in the blood.

Normal Haemoglobin levels = 140 - 175g/L
Haemoglobin levels less than 140g/L indicate anaemia

Your ferritin level = __________________
Your Haemoglobin = __________________

<table>
<thead>
<tr>
<th>Iron per 100g (cooked unless stated)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lamb kidneys</td>
</tr>
<tr>
<td>11.4 mg</td>
</tr>
<tr>
<td>Beef - steak or mince, lean</td>
</tr>
<tr>
<td>3.2 mg</td>
</tr>
<tr>
<td>Pork, lean</td>
</tr>
<tr>
<td>1.3 mg</td>
</tr>
<tr>
<td>Wholemeal bread, 1 thin slice</td>
</tr>
<tr>
<td>0.7 mg</td>
</tr>
<tr>
<td>Baked beans, ½ cup</td>
</tr>
<tr>
<td>1.2 mg</td>
</tr>
<tr>
<td>Spinach, cooked drained ¼ cup</td>
</tr>
<tr>
<td>1.3 mg</td>
</tr>
<tr>
<td>Peas, cooked ½ cup</td>
</tr>
<tr>
<td>1.1 mg</td>
</tr>
<tr>
<td>Egg, 1 boiled</td>
</tr>
<tr>
<td>0.7 mg</td>
</tr>
<tr>
<td>Silverbeet, cooked, drained ¼ cup</td>
</tr>
<tr>
<td>1.0 mg</td>
</tr>
<tr>
<td>Raisins, 2 Tbsp</td>
</tr>
<tr>
<td>0.3 mg</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Iron per serving</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 cup porridge 1.4</td>
</tr>
<tr>
<td>0.7 mg</td>
</tr>
<tr>
<td>1 weetbix 0.8</td>
</tr>
<tr>
<td>0.7 mg</td>
</tr>
<tr>
<td>1 tablespoon raisins 0.3</td>
</tr>
<tr>
<td>0.7 mg</td>
</tr>
<tr>
<td>Teaspoon brewers yeast 0.3</td>
</tr>
<tr>
<td>0.3 mg</td>
</tr>
<tr>
<td>1 tablespoon raisins 0.3</td>
</tr>
<tr>
<td>0.7 mg</td>
</tr>
<tr>
<td>Sower: Australian Food Composition Tables</td>
</tr>
<tr>
<td>0.7 mg</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Iron per content of some foods</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lamb's kidney 8.5</td>
</tr>
<tr>
<td>3 slices cooked topside beef (75g) 3.5</td>
</tr>
<tr>
<td>4-5 sardines in oil</td>
</tr>
<tr>
<td>2.2 mg</td>
</tr>
<tr>
<td>½ cup cooked kidney beans 2.8</td>
</tr>
<tr>
<td>100g tofu 1.9</td>
</tr>
<tr>
<td>1.9 mg</td>
</tr>
<tr>
<td>chicken meat (75g)</td>
</tr>
<tr>
<td>1.6 mg</td>
</tr>
<tr>
<td>⅛ cup peas boiled</td>
</tr>
<tr>
<td>1.5 mg</td>
</tr>
<tr>
<td>1 cup porridge</td>
</tr>
<tr>
<td>1.4 mg</td>
</tr>
<tr>
<td>5 dried apricots</td>
</tr>
<tr>
<td>1.1 mg</td>
</tr>
<tr>
<td>1 weetbix</td>
</tr>
<tr>
<td>0.8 mg</td>
</tr>
<tr>
<td>1 slice wholemeal</td>
</tr>
<tr>
<td>0.3 mg</td>
</tr>
<tr>
<td>Teaspoon raisins</td>
</tr>
<tr>
<td>0.3 mg</td>
</tr>
<tr>
<td>Sower: Australian Food Composition Tables</td>
</tr>
<tr>
<td>0.7 mg</td>
</tr>
</tbody>
</table>
Example of five day food diary
INSTRUCTIONS FOR KEEPING A DIET RECORD

NAME: ___________________________ I.D. ___________________________

RECORD SHEET

PLEASE READ THESE IMPORTANT INSTRUCTIONS CAREFULLY

* Please record ALL food and drinks consumed
* Please record the food at the time of eating and NOT from memory at the end of the day
* You should include all meals & snacks, plus sweets, drinks (including water) etc.
* Remember to include any additions to foods already recorded such as: sauces, dressings or extras e.g. gravy, salad dressings, stuffings, sugar, honey, syrups etc., butter or margarine (e.g. added to bread, crackers, vegetables).
* If you do not eat a particular meal or snack, simply draw a line across the page at this point. This will show that you definitely have not eaten anything.

DESCRIBING FOOD AND DRINK – GUIDELINES

1. Please give details of the method of cooking all foods (e.g. fried, grilled, boiled, roasted, steamed, poached, stewed).

2. Give as many details as possible about the type of food that you eat e.g. brand name of food where applicable (e.g. Miracle margarine);
   type of: Breakfast cereal (e.g. Weetbix)
   milk (e.g. whole milk or 'trim milk')
   cake or biscuit (e.g. fruit cake, wheatmeal biscuit)
   fruit (e.g. fresh, canned, dried, stewed)
   soft drink (e.g. regular or low calorie)

3. Name the type of cheese, fish or meat (e.g. cheddar, cod fillet, loin of pork)

   e.g. EGGS
   Are they fried, boiled, poached or scrambled?
RECORDING THE AMOUNTS OF FOODS YOU EAT

It is also very important to record the quantity of each food and drink you consume.

Here are some suggestions on how to record amounts:

• IN HOUSEHOLD MEASUREMENTS

For many foods such as vegetables, cereals and canned or stewed fruit, a household measurement is adequate.

e.g. STATE THE NUMBER OF TEASPOONS (t), TABLESPOONS (T), CUPS etc. State whether spoons are level, rounded or heaped.

- level
- rounded
- heaped

Butter and margarine can be measured in teaspoons or tablespoons if you find this an easy method.

• WEIGHTS MARKED ON PACKAGES

All convenience foods have their weight marked on the packaging and this can be quoted e.g. half a 425g can of baked beans.

• BREAD - indicate the size of the slices (e.g. sandwich, medium, toaster).

• CHEESE, MEAT & FISH

If at all possible, it would be very helpful to weigh your portions of these foods.

If this is not possible, please use the pictures on the attached sheets to indicate what sort of portion sizes you eat e.g. you might have 1 portion of spaghetti size A, 1 portion of meat size B or 2 slices of cheese size C.

• USE COMPARISONS for describing portion sizes where this is easier e.g. potato - size of a hen’s egg, cheese - size of a matchbox.

IT IS VERY IMPORTANT THAT YOU DO NOT ADJUST WHAT YOU EAT AND DRINK BECAUSE YOU ARE KEEPING A RECORD. THIS IS VERY EASY TO DO, BUT REMEMBER, WE ARE INTERESTED IN YOUR EATING HABITS, NOT THE PERFECT DIET!!!
**DAY 1 - Date ..................**

- Record ALL food and drink consumed during the day including sweets, snacks, 'nibbles', sauces and dressings.
- Please record:  
  - METHOD OF COOKING (e.g. boiled pasta)
  - TYPE OF FOOD (e.g. boiled wholegrain pasta)
  - QUANTITY OF FOOD (e.g. 6 heaped T boiled wholegrain pasta)

<table>
<thead>
<tr>
<th>MEAL/ SNACK</th>
<th>QUANTITY EATEN</th>
<th>DETAILS OF FOOD AND DRINK</th>
</tr>
</thead>
<tbody>
<tr>
<td>EARLY MORNING</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BREAKFAST</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DURING MORNING</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MIDDAY</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MEAL/SNACK</td>
<td>QUANTITY EATEN</td>
<td>DETAILS OF FOOD AND DRINK</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>----------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td><strong>DURING ANY TIME</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>EVENING MEAL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>DURING EVENING/BEFORE BEDTIME</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**DAY 1 - continued**
PHOTOGRAPH 5  VEGETABLE PIE

PHOTOGRAPH 6  SPAGHETTI
CONSENT FORM

I agree to participate in this diet study looking at the effects of different diets on blood cholesterol levels in endurance trained athletes.

I have read the information sheet detailing my involvement in the study and understand I will be required to alter the amounts of fat/carbohydrate in my diet.

I understand that my involvement will include having blood samples taken on 8 separate occasions, completion of 5-day food diaries, exercise testing on 2 occasions and the monitoring of my weight and percentage body fat.

I am satisfied that the nature of the study and its relevance have been explained to me, and all of my questions answered.

I understand that my participation in this study is voluntary and I may withdraw at any time.

I understand information collected from me will be treated confidentially and dealt with only by those directly involved in the study.

I understand that no material which could personally identify me will be used in any future publications.

Signature: _______________________________
Date: ________________________________
GENERAL INFORMATION QUESTIONNAIRE

Name: ................................ Phone: Home: ........................................
Address: ........................................ Work: .................................
Date of Birth: ................................

1. Occupation: ................................

2. Marital status: .............................

3. How many years of secondary school education did you have? ............
   What was the highest certificate or qualification you attained at
   secondary school or at a tertiary institution?

4. What is your ethnic origin? ........................................

5. Do you currently smoke cigarettes? YES / NO (Please circle)
   If “yes” number per day..............
   number of years....................

6. Alcohol intake: 1 glass of beer = 1 unit
   1 glass of wine = 1 unit
   1 Nip of spirit = 1 unit

   UNITS of alcohol during week days = ......................................
   UNITS of alcohol during weekends = ......................................

7. Do you take any pills/medicines? YES / NO (Please circle)
   If “yes”, please include names of medicines and/or reasons for taking
   them
   ................................................................................................
8. Do you take any oral contraceptives? YES / NO (Please circle)

9. Medical History
   Do you currently suffer from, or have you previously been diagnosed as having:
   (Please Circle where appropriate)
   a. Diabetes
   b. High blood cholesterol
   c. High Blood Pressure
   d. Angina
   e. Asthma
   f. Anaemia
   g. Epilepsy
   h. Other (Please specify) .........................................

10. Do you currently take any dietary supplements? (Circle where appropriate)
    Multivitamins
    Vitamin C
    Calcium
    Iron
    B complex (B6 / B12)
    Folate / folic acid
    Caffeine tablets
    Salt tablets
    Protein supplement
    Other (Please specify) .................................
11. Have you ever taken dietary supplements in the past? Yes / No (please circle)

If "yes" (please specify) ..........................................................
Reason for taking supplement (eg. Doctors advice)..........................

12. Are you currently on a special diet? YES / NO (please circle)
If "yes" please specify..........................................................

13. Are you happy with your current weight? YES / NO (Please circle)

14. Do you have an ideal competitive weight? YES / NO (Please circle)
If "yes" what is it?.................kg

15. During the cycling season do you usually:-

- gain weight? YES / NO (Please circle)
- lose weight? YES / NO (Please circle)

16. a. How many years have you been cycling? .........................

- How many years have you been a competitive cyclist? ..............

17. What cycling grade do you currently cycle at?..........................

18. Do you train/participate regularly in other sports or training activities?
            eg. weight training YES / NO (Please circle)
            if "yes" please specify ..............................................

19. How many hours of training do you usually do a week during the cycling season? .........................

            hours of cycling .........................
            hours of other training .................
20. What are your main reasons for cycling? Please circle the one(s) most important to you

a. fitness
b. weight control
c. enjoyment/recreation
d. competition
e. general health
f. all of the above

21. Would you classify yourself as a vegetarian? YES / NO (Please circle)

Please circle the answer that best describes you:

a. semi-vegetarian (fish and chicken only i.e. no red meat)
b. lacto-ovo vegetarian (i.e. no fish, chicken, red meat but includes eggs and milk products)
c. vegan (i.e. no animal products at all)

22. Do you follow any other diets? YES / NO (Please circle)
e.g.-macrobiotic / low salt / low fat / high protein / high carbohydrate

if “yes” please specify: ....................................................

................................................................................

.................................................................................

23. Do you prescribe to a particular diet in preparation for race day? YES / NO (Please circle)

eg. carbohydrate loading/other.

if “yes” please specify ....................................................

................................................................................
24. Any further comments about your diet/nutritional status, or this questionnaire?

-------------------------------------------------------------------------------------
-------------------------------------------------------------------------------------
-------------------------------------------------------------------------------------

Thank you for your time.
0  Post-study questionnaire

POST-SPORTS NUTRITION STUDY QUESTIONNAIRE

1. Name ____________________________________________________________

2. Did you enjoy following the diet you were on during the Sports Nutrition Study? YES / NO (circle one)

3. Could you have followed your study diet for a longer period of time? YES / NO (circle one)

4. Was the study diet very different from your normal diet? YES / NO Comments : ____________________________________________________________

5. Would you have preferred to remain on your usual diet instead of the study diet? YES / NO (circle one)

6. Have you now reverted back to your pre-study diet? YES / NO (circle one)
   If ‘YES’- Why? ____________________________________________________________

   If ‘NO’ - Why? ____________________________________________________________

7. On a scale of 1-10 how do you rate your overall cycling performance whilst following the study diets -

   |   |   |
   1 5 10
   awful average excellent

   Number : __________

8. Did you find any problems with the study diet? YES / NO
   If ‘YES’, what were they : ____________________________________________________________
10. Any additional comments would be appreciated:
Samples of study correspondence
Initial letter inviting participation in the study.

Human Nutrition Department

Dear Cyclist,

With the cycling season due to recommence on the 25th July, we thought it would be an ideal opportunity to invite you to take part in our Sports Nutrition Study to be undertaken by the Department of Human Nutrition, University of Otago.

Our aim is to investigate the effects of two different diets on blood cholesterol levels in endurance trained cyclists. The results of this research could provide important information as to future nutritional guidelines for endurance athletes.

I am sure you are all aware of the relationship between cholesterol levels and Heart Disease. Numerous studies have shown that regular physical exercise can decrease the risk for coronary heart disease. There may be many explanations for the protective effect of exercise on heart disease, including the beneficial changes in cholesterol levels.

For most of the people in the population, high cholesterol levels are due to the high intake of fat in the diet (particularly saturated fat). However, an interesting research finding is that people who are very fit, eg endurance trained athletes, tend to have lower cholesterol levels than people who do not do any exercise - even if the athlete's diet is relatively high in fat.

**We aim to investigate this observation.**

In this study we aim to recruit 30 Otago cyclists, who will be divided into 2 groups. Group 1 will receive a high carbohydrate diet and group 2 an energy dense diet. The diets should not be too different from your current diet and are within the current recommended dietary intakes for endurance athletes. They consist of normal foods and will provide all essential nutrients. We would ask you to follow the dietary guidelines for 12-16 weeks. Cholesterol will be monitored throughout this period.

**What are the Benefits for you?**

1. **Exercise testing:**
   During this 12-16 week study you will have three VO2 max tests performed at the Human performance Laboratory, University of Otago. This will provide you with excellent feedback as to the effectiveness of your training and level of physical fitness throughout the cycle season. We will monitor body composition change also, including % body fat and body weight.

2. **Iron status:**
   Iron deficiency anaemia may be an explanation for poor racing and training performance. We will take blood tests which will provide information about your iron levels.

3. **Cholesterol levels:**
   Blood tests on 4 occasions will monitor your cholesterol levels.

4. **Free training food:**
   Energy bars will be provided throughout the study. During diet (HC) you will be provided with high carbohydrate energy bars and during the energy dense diet with high Energy bars.
Your involvement in the study:
The study duration will be 12-16 weeks, commencing 17th August and finishing early December.

1. Diets:
   We would ask you to alter the amounts of carbohydrate and fat in your diet. A trained sports dietitian will provide you with information and supervise throughout the study.

2. Food diaries:
   We would ask you to complete three 5-day food diaries throughout the study.

3. Training diary:
   We would ask you to keep a weekly record of training hours and mileage. We would also ask you to record your assessment as to how you think you performed.

4. Blood tests:
   There will be 4 blood tests throughout the study; before the study commences, at weeks 4, 8, 12 and 16.

5. Exercise testing:
   2 approximately 1 hour exercise tests.

If you are interested in taking part in this study, please fill in the section below the dotted line and post in the stamped-addressed envelop enclosed.

If you have any questions about the study, please do not hesitate to contact me (Rachel Brown) or Dr Charlotte Cox (evenings 474-1512).

We look forward to hearing from you. Best wishes for the up-coming cycling season. Thank you.

Yours sincerely,

Rachel Brown (BSc),
Masters student
Department of Human Nutrition
University of Otago.

I am interested in taking part in this study.

Name: ________________________________

Signed: ______________________________
Letter to subjects informing randomisation and diet group assigned to.

Human Nutrition Department
14/8/92

Dear

Regarding the Sports Nutrition Study, you have been allocated to the “Energy Dense Diet” group. The dietary period will start on Monday 17th August.

Please find enclosed a booklet detailing the diet. Remember during this diet to use full cream dairy products and generous helpings of margarine/butter. If you have any questions regarding the diet please do not hesitate to contact us.

In addition you will also find a weekly training and performance record in the booklet. Please fill this out weekly.

I will notify you in the near future regarding times for your next blood tests. Remember you can collect the Cadbury foods from us fortnightly.

If you have any further queries, do not hesitate to contact us. Phone numbers are on the booklet.

Best of luck with training. Thankyou.

Yours sincerely

Rachel Brown
Dear

Regarding the Sports Nutrition Study, you have been allocated to the "High Carbohydrate Diet" group. The dietary period will start on Monday 17th August.

Please find enclosed a booklet detailing the diet. Remember this diet is very high in carbohydrate and so the emphasis is on fat restriction. If you have any questions regarding the diet please do not hesitate to contact us.

In addition you will also find a weekly training and performance record in the booklet. Please fill this out weekly.

I will notify you in the near future regarding times for your next blood tests. Remember you can collect the Cadbury foods from us fortnightly.

If you have any further queries, do not hesitate to contact us. Phone numbers are on the booklet.

Best of luck with training. Thankyou.

Yours sincerely,

Rachel Brown
Letter to subjects regarding individual study results.

Human Nutrition Department
University of Otago
Dunedin

2/3/93

Dear

I am writing to you regarding the Sports Nutrition study you were involved in last year. I would like to take the opportunity to personally thank you for your participation. It was very much appreciated.

The results thus far are promising to be most interesting; making this research very worthwhile.

I have enclosed your own personal results from the study. These include your blood cholesterol levels as monitored throughout the study. Your weights and VO2max exercise test results; as well as some dietary information.

Your blood cholesterol levels throughout the study were as follows:
Baseline (beginning of study) mmol/L
Week 5 mmol/L
Week 9 mmol/L
Week 13 mmol/L
Week 17 mmol/L

Your weight throughout the study was:
Baseline (beginning of study) kg
Week 5 kg
Week 9 kg
Week 13 kg
Week 17 kg

Your initial VO2max exercise test result was mls/min/kg (L/min) and you attained Watts
Your final VO2max exercise test result was mls/min/kg (L/min) and you attained Watts.

The following are some examples of VO2max results obtained from various competitive cyclists:

- New Zealand Juniors - 60.2 - 67.0 ml/min/kg
- New Zealand Road team - 74.6 - 84.3 ml/min/kg
- New Zealand Track Team - 66.2 - 83.3 ml/min/kg
- American Junior team - 64.8 ± 5.5 ml/min/kg
- American Category 1 (A graders) 70.6 ± 9.5 ml/min/kg
- American National Team 74.0 ± 8.3 ml/min/kg
- Olympic Cyclists 75.0 ml/min/kg

Regarding dietary information obtained from your 5-day food diaries; the percentage of fat and carbohydrate in your diet was as follows:

Baseline (beginning of study)

<table>
<thead>
<tr>
<th>% fat</th>
<th>% carbohydrate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Throughout study</td>
<td></td>
</tr>
</tbody>
</table>

The results given thus far are your own personal results. In the near future when the final analyses have been completed, you will receive a more extensive account of the overall results of the study.

I would once again like to thank you for your willing participation in this research. If you have any questions do not hesitate to contact me at the above address, or ph 479 7940.

Yours sincerely,

Rachel Brown
Letter to subjects regarding study results.

Human Nutrition Department,

Dear

I am writing to you regarding the Sports Nutrition study you were involved in last year. I am pleased to say that we have now completed the final analyses and can provide you with the overall findings of the study.

The first important finding of the study is that both the high carbohydrate diet and the high fat diet affected blood cholesterol levels in a similar way. It was evident that both diet groups underwent a reduction in total blood cholesterol over the study period. In addition to this LDL cholesterol (the bad part of cholesterol in the blood) also was reduced in both diet groups.

The following table shows the average blood cholesterol (mmol/l) levels for both diet groups before the start of the study and after week 12 and sixteen.

<table>
<thead>
<tr>
<th></th>
<th>Pre-study</th>
<th>week 12</th>
<th>week 16</th>
</tr>
</thead>
<tbody>
<tr>
<td>High carbohydrate diet</td>
<td>4.7</td>
<td>4.5</td>
<td>4.3</td>
</tr>
<tr>
<td>High fat diet</td>
<td>5.0</td>
<td>4.6</td>
<td>4.6</td>
</tr>
</tbody>
</table>

In the general population the consumption of a high fat diet (particularly saturated fat) is known to increase blood cholesterol levels and thus the risk of heart disease. This does not seem to be the case in a group of healthy, highly trained athletes like yourselves.

We propose that the physiology and metabolism of trained endurance athletes is very different from those who are inactive. Endurance athletes are able to use fat as an important fuel source in exercise to a greater extent than non-trained individuals.

Physical exercise is known to independently cause beneficial effects to blood cholesterol. The findings of our study suggest that the beneficial effects of endurance exercise on blood cholesterol outweigh the adverse effects of a diet high in fat.

A second interesting finding of the study is that the consumption of a high fat diet did not cause any detrimental effects on training and
performance capacity. Both groups showed increases in physical fitness throughout the study. This increase was of the same magnitude in both groups. This indicates that a high carbohydrate diet which is usually recommended for endurance athletes may not be necessary to enhance performance.

Other results from the study show that no significant weight changes were evident in either of the two groups. No differences were evident in body weight between the two groups at any point during the study. Similarly, no changes in total body fat (as measured by skinfolds taken at the exercise tests) were evident in either group during the study.

The major conclusions from the study are as follows:

1. A diet high in fat did not have an adverse effect on blood cholesterol levels in a group of endurance trained athletes.

2. Exercise and performance capacity was not impaired with the consumption of a diet higher in fat.

3. Exercise seems to be a more important determinant of blood cholesterol than diet in endurance trained athletes.

**Implications:**
It seems that a high carbohydrate diet that is recommended for endurance athletes to improve performance is not necessary. A diet relatively higher in fat, which is more energy dense, may provide an easier means of consuming the increased energy demands of endurance exercise. The study shows that this will not adversely effect the blood cholesterol levels of individuals taking part in high levels of physical exercise.

As endurance athletes you should be able to choose a diet of your preference. You need not stick to strict dietary regimes that can prove to be very restrictive. As long as your diet is well balanced you should be fine.

An important point to remember is that the most important determinant of exercise performance is training.

If you have any questions regarding the results of the study do not hesitate to contact me.
ph 479-7940 - work
ph 477-6206 - evenings
I would like to thank you all for your participation in this important research. I have recently presented the results at a seminar where they were received with much interest. A paper is currently being written for a scientific journal on the results of our study, so much interest is being generated from this research.

We are currently considering undertaking a further study in this area of sports nutrition next year. If you would be interested in participating in this we would very much appreciate it.

Thankyou, and best wishes for the next cycling season!

Yours sincerely,

Rachel Brown
Post-study letter regarding post-study questionnaire.

Human Nutrition Department  
University of Otago  
Dunedin

Dear

During the analysis of your results from the Sports Nutrition Study we thought it to be important to obtain some feedback on how you found the study diets.

I have thus enclosed a brief questionnaire to enable us to complete our analyses.

It would be much appreciated if you could complete the questionnaire and return it to me in the stamped envelop provided.

If you have any additional queries do not hesitate to contact me at the above address or the following phone numbers:
479 7940 (work)  
477 6206 (evenings)

Thank you.

Yours sincerely,

Rachel Brown