THE EFFECTS OF TRAINING ON TC, HDL AND TC/HDL RATIO IN MALE INTERCOLLEGIATE CROSS-COUNTRY RUNNERS

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ABSTRACT

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This study determined the effects a season of cross-country training had on body composition, max$\dot{V}O_2$, total cholesterol (TC), high-density lipoprotein (HDL) and the TC/HDL ratio of 43 runners ($\bar{x}$ = 19.8 yrs) from UW-La Crosse cross-country team. RV was determined by O_2 dilution technique and % body fat was ascertained by hydrostatic weighing. There was no sig ($p > .01$) diff in wt, lean body weight (LBW) or % body fat from pre- to post-season testing. Max$\dot{V}O_2$ was measured during a treadmill (TM) run using the Beckman Metabolic Measurement Cart. No sig ($p > .01$) diff was found in maxHR, TM time and max V_E. There was a sig ($p < .01$) diff in max$\dot{V}O_2$ measured in $ml \cdot kg \cdot min^{-1}$ and $L \cdot min^{-1}$ and in respiration exchange ratio (RER). Dietary intakes of meats, dairy products, fats, eggs and alcohol were analyzed using ANOVA with repeated measures at 3 times throughout the season. No sig ($p > .01$) diff were found in any of these variables. Blood variables were analyzed using the Data Medical Associates procedure. No sig ($p > .01$) diff were found in TC, HDL or TC/HDL ratio after the season of cross-country running. It was concluded that a season of cross-country running did sig increase max$\dot{V}O_2$, but did not sig change body composition, dietary intakes, TC, HDL's and TC/HDL ratio.

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We recommend acceptance of this thesis in partial fulfillment of this candidate's requirements for the degree: Master of Science, Adult Fitness/Cardiac Rehabilitation.

The candidate has completed his oral report.

Nancy Kay Butts — Thesis Committee Chairperson — 16 May 84 — Date

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DEDICATION

I will support you in all that you do.

I will help you in all that you need.

I will share with you in all that you experience.

I will encourage you in all that you try.

I will understand you in all that is in your heart.

I will love you in all that you are.

- Susan Polis Schultz -

I dedicate all my hours of thesis work and writing to my future wife, Mary, who has shared so much with me in this experience and who has given me all the support, help, encouragement, understanding and, most of all, love, that anyone could ever ask for throughout the year.

MTY
LTT

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CHAPTER I

INTRODUCTION

Need for the Study

The major plasma lipids, including cholesterol and triglyceride, do not circulate freely in solution in the blood, but rather are transported in the form of lipoprotein complexes. These complexes of lipid and protein impart solubility to otherwise insoluble lipids, and all lipids enter and travel through the blood stream as lipoprotein complexes (Levy & Rifkind, 1980). The major protein families are chylomicrons, very low-density lipoproteins, low-density lipoproteins and high-density lipoproteins. These lipoprotein classes can be categorized as types of cholesterol though each one possesses a different concentration (Davis, Gordon, LaRosa, Wood, & Halperin, 1980). The majority of past studies have been concerned with low-density lipid cholesterol because of its positive correlation to coronary heart disease. Recently, however, there has been more investigation into high-density lipid cholesterol because of its inverse association to incidence of coronary heart disease (CHD) and to the number and extent of arteries with atherosclerotic involvement (Hartung, Williams, & Gotto, 1981).

It has been well established that different intensities, durations and modes of aerobic training affect lipid concentrations (Adner & Castelli, 1980; Clarkson, Hintermister, Fillyaw, & Stylos, 1981; Haskell, Taylor, Wood, Schrott, & Heiss, 1980; Lehtonen & Viikari, 1978; Schnabel & Kinderman, 1982). Some researchers have found that exercise

significantly altered lipoprotein concentrations. Others have stated that exercise had little or no effect on these components. Most of the literature involved healthy middle-aged adults (Brownell, Bachorick & Ayerle, 1982; Kinsman, Weber & Anderson, 1980; Moore, Penford, Simpson, Mann & Turner, 1979); others observed changes in coronary heart patients (Castelli, Doyle, Gordon, Hames, Hjortland, Hulley, Kagen & Zukel, 1977a; Hartung et al., 1981; Miller, Thelle, Forde & Mjos, 1977). Few studies have been conducted on highly trained individuals. Thus, there is still a need to investigate the effect exercise has on lipoprotein concentrations in trained individuals. It was the intention of this researcher to utilize quantitative research methods to expose new and relevant information on lipoproteins in this population.

Purpose

The purpose of this study was to examine the effects of training on max$\dot{V}O_2$, body composition and concentrations of total cholesterol, high-density lipoproteins and total cholesterol/high-density lipoprotein ratio. This study utilized intercollegiate male cross-country runners, at the University of Wisconsin-La Crosse who participated in a training program as specified by their coach, Dr. Phillip Esten.

Hypothesis

In this study a null hypothesis was postulated: there would be no differences between pre-training and post-training levels of blood concentrations of total cholesterol, high-density lipid cholesterol, total cholesterol/high-density lipoprotein cholesterol ratio, physiological parameters of max$\dot{V}O_2$, maxV_E, maximal heart rate, treadmill time

and respiration quotient and body composition including weight, lean body weight and percent body fat. The basis for acceptance or rejection was based on the .01 level of confidence.

Assumptions

Within the limits of this study, the following assumptions were made:

1. All subjects had fasted for at least 12 hours prior to blood draws.

2. All subjects had abstained from alcohol consumption 24 hours prior to the blood draws.

3. The athletes remained in the realms of the practices, as they were set up and did not exercise or train outside the confines of the study.

4. Cross-country runners were healthy individuals throughout the season.

5. The dropout rate of the participants was due to injury, not from discontentment of the training regime.

6. The blood assays did, in fact, accurately measure the levels of total cholesterol, high-density cholesterol and total/high-density cholesterol ratio.

7. Maximum oxygen uptake, as measured by uphill treadmill running, is the single most accurate measure of cardiovascular fitness in cross-country runners.

8. Subjects performed the residual volume and hydrostatic weighing procedures to the best of their abilities.

Delimitations

The following situations were judged as delimitations to this study.

1. The subjects selected were all runners, regardless of ability, from the University of Wisconsin-La Crosse cross-country team.

2. The subjects were all males.

3. No control group was used, thus reducing the internal validity with regard to history and maturation.

4. There was no attempt to determine how long prior to this specific training program the subjects had been running.

5. Only total cholesterol, high-density cholesterol and total/high-density cholesterol ratio were analyzed.

Limitations

In reference to this study, the following situations were judged as limitations:

1. Alcohol may have had some effect on the composition of assays.

2. Motivational factors from one runner to another and pre- to post-test may have varied.

3. Time factor between pre- and post-season testing may have varied due to post-season competition (NCAA & NAIA National Championships).

Definition of Terms

Maximal Oxygen Uptake ($max\dot{V}O_2$) – oxygen consumption at maximal work, which represents the maximal circulatory transport of oxygen from the

lungs to the metabolically active tissue. In this study, max$\dot{V}O_2$ was obtained through uphill treadmill running (Strauss, 1979, p. 88).

Phospholipid - water soluble lipid-type molecule which increases the solubility of lipids for transport and absorption (Guthrie, 1983, p. 50).

Cholesterol - an alcohol lipid or sterol which can be synthesized in the body and which is present in animal fat (Guthrie, 1983, p. 50).

Chylomicron - the least dense of the lipoproteins (0.95 g/ml) which is composed of 98-99.5% lipid and 0.52% protein, with the major lipid component being triglyceride (Garman, 1978, p. 185).

Very Low-Density Lipoprotein (VLDL) - a substance which contains cholesterol and triglycerides: 19% and 50%, respectively. In addition, it contains approximately 10% protein and 18% phospholipid with a total density of 0.95-1.006 g/ml (Garman, 1978, p. 185).

Low-Density Lipoprotein (LDL) - the lipid class which has a density between 1.006-1.063 g/ml, composed of 21% protein, 47% cholesterol, and its ester, 23% phoshpolipid and 9% triglyceride (Albrink, 1962, p. 146).

High-Density Lipoprotein (HDL) - the lipoprotein which is composed of triglycerides, cholesterol, phospholipid and protein percentages of approximately 7, 19, 26 and 48, respectively. It has a density greater than 1.063 g/ml (Bennion, 1979, p. 428).

Total Cholesterol (TC) - total amount of cholesterol carried by the chylomicron, VLDL, LDL, and HDL complexes in circulation (Bennion, 1979, p. 427).

Lipoprotein Lipase (LPL) - an enzyme which causes or aids in the

splitting of triglyceride molecules of free fatty acid and consequent to stepwise removal of triglycerides in lipolytic fashion (Albrink, 1962, p. 151).

Apoprotein - protein components of lipoproteins having the abilities to bind lipids, solubilize and transport neutral fats (Levy & Rifkind, 1980, p. 5).

Cross-Country Season - a training and competitive program of the University of Wisconsin-La Crosse cross-country runners which varied from 9 to 12 weeks (August to October 29 or November 20), pending qualification for NCAA or NAIA National Championships.

Density - the weight of an object per unit volume.

Hydrostatic Weighing - the process of weighing a body under water to determine the volume of the body.

Percent Body Fat - the percentage of total body weight that consists of adipose tissue as measured by hydrostatic weighing.

Residual Volume (RV) - the volume of air remaining in the lungs following the greatest possible maximal expiration.

CHAPTER II

REVIEW OF RELATED LITERATURE

Introduction

"It is generally believed that atherosclerosis is a disease of multiple interacting causative factors, and its prevention or control requires intervention in several parameters" (Kuo, 1981, p. 949). Recently the discussion of high-density lipoproteins has been greatly intensified largely because of findings that high-density lipids are inversely related to atherogenisis and to the number and extent of arteries with atherosclerotic involvement (Hartung et al., 1981; Levy & Rifkind, 1980). Subsequent authors (Garman, 1978; Heiss, Johnson, Reiland, Davis, & Tyroler, 1980; Kuo, 1981) have tried to explain these findings by reference to the fact that the peripheral tissue cells are incapable of degrading cholesterol. To lighten the burden on the vessel walls, cholesterol has to be transported back to the liver for catabolism. Thus, high-density lipids are believed to promote this anti-atherogenic process (Castelli et al., 1977a; Gordon, Castelli, Hjortland, Kannel, & Dawber, 1977). From data that have been gathered it was found that HDL cholesterol made a threefold contribution to the discrimination between subjects who were likely or unlikely to suffer a coronary event, because of its inverse association (Miller et al., 1977). No concrete proof of any such relationship has yet been confirmed; however, researchers are hoping to demonstrate a link in these relationships so theory may be transposed to fact.

Since serum HDL cholesterol is inversely related to the incidence of CHD, it is considered the best single indicator of risk in apparently healthy individuals (Castelli et al., 1977a). According to Castelli and Levitas (1977) HDL cholesterol levels below 35mg/dl represent high risk, from 35-55mg/dl an intermediate risk and above 55mg/dl a low risk.

A general assessment can be established by determining the ratio of total cholesterol to high-density cholesterol (Castelli & Levitas, 1977). According to these authors a male will have half the risk of developing CHD if this ratio is below 3.43; average risk if the ratio is between 3.44-4.97; two times the average risk if between 4.98-9.55; and three times the average risk if between 9.56-23.39.

It appears that diet may have an important effect on the concentration of HDL's in the body. Albrink (1962) and Friedman and Rosenman (1957) suggested that the type of fat (i.e., saturated or unsaturated) may produce change in lipoprotein concentration. Quintao, Grundy and Ahrens (1971) have suggested that the amount of exogenous, dietary, or even endogenous cholesterol may affect high-density lipid levels. Still others have implied that the reduction in weight or the intake of dietary fiber beget alterations in concentration of these lipoprotein levels ("Dietary fiber, exercise and selected blood constituents," 1980; Liebman, Smith, Iverson, Thye, Hinkle, Herbert, Ritchy & Driskell, 1983).

The literature has further suggested other factors which may elicit change in levels of lipoproteins in the body. Phillips, Havel and Kane (1981) reported that alcohol may have such an effect. Others

suggested smoking as a factor which may affect blood concentration of high-density lipoproteins (Garrison, Kannel, Feinleib, Castelli, McNamera, & Padgett, 1978; Kanamura, 1981).

Extensive research has been conducted into the effects of exercise on blood lipid concentrations. Schnabel and Kinderman (1982) compared many different modes of exercise to determine the various effects on lipoprotein levels. Other authors compared exercising groups to control groups to identify differences in lipoprotein levels due to exercise (Adner & Castelli, 1980; Lehtonen & Viikari, 1978). Finally, some authors have investigated the effects of eight weeks of aerobic exercise on lipoprotein concentrations (Dufaux, Assman, Schachten, & Hollmann, 1982; Farrell & Barboriak, 1980), while another author was concerned with one intense exercise bout and changes it produced in high-density lipid levels (Haskell et al., 1980). The review of literature which follows will include a general background of lipids and lipoproteins and factors which may affect their concentrations; diet, body composition, alcohol, smoking and exercise. The relationship between max$\dot{V}O_2$ and lipoproteins will also be discussed.

General Background

Lipids, generally termed fats, are viewed as unsightly and the general public is always concerned with "losing those inches of fat." To the surprise of many people, fat is a necessary and vitally important component of the body's functions (Bennion, 1979; Guthrie, 1983; Krauss, 1982). It is primarily used as an energy source and gives insulation and protection to the internal organs and body parts. It is also a carrier

of the fat soluble vitamins, a source of essential fatty acid, a precursor of prostaglandins and adds palatibility and satiety to diet (Guthrie, 1983).

Lipids and lipoproteins can be divided into four different classes: chylomicrons, very low-density lipoproteins (VLDL), low density lipoproteins (LDL), and high-density lipoproteins (HDL) (Bennion, 1979; Guthrie, 1983; Mahley, 1982; Levy & Rifkind, 1980). These lipoproteins composed of cholesterol, triglyceride, phospholipid and protein, thus have specific densities. Specific descriptions and density for each lipid are presented in Tables 1 and 2.

Table 1. Lipid Composition of Various Lipoproteins (%)

	Chylomicron	VLDL	LDL	HDL
Cholesterol	5	13	43	18
Triglyceride	90	65	10	2
Phospholipid	4	12	22	30
Protein	1	10	25	50

(Levy & Rifkind, 1983, p. 5)

Table 2. Operational Classification of Plasma Lipoproteins

Electrophoretic Mobility	Density (g/ml)
Chylomycron	< 0.950
Pre-Beta Lipid (VLDL)	0.950 - 1.006
Beta Lipid (LDL)	1.006 - 1.063
Alpha Lipid (HDL)	1.063 - 1.210 or >

(Levy & Rifkind, 1983, p. 5)

Lipoprotein Pattern

Bennion (1979), Garmon (1978), Heiss and associates (1980) and Levy and Rifkind (1983) stated that plasma lipids do not circulate freely, but are transported through the blood in lipoprotein complexes (VLDL, LDL, & HDL). The chylomicron, which originates in the intestines from exogenous or dietary fat, is acted upon by lipoprotein lipase (LPL) when metabolized. This separates the fat from the lipoprotein carrier. The triglyceride is hydrolized, thus, the fatty acid portion enters the cell and the water soluble glycerol portion stays in general circulation (Guthrie, 1983). Similarly, when the VLDL reaches the cell the LPL releases some of the triglyceride resulting in the formation of LDL. Once these fatty acids are taken up by the cell they can be oxidized and used or, if not used, make up the lipid portion of the cell wall (Guthrie, 1983).

Levy and Rifkind (1980) stated high-density lipids are polydispersed and heterogeneous with respect to size and lipid apoprotein content. They are spherically shaped and range from 70 to 120 angstrom in diameter. In metabolism both the liver and intestines are involved in the production of HDL, although the exact roles and relative importance are not fully understood (Krauss, 1982; Levy & Rifkind, 1980).

> Evidence indicated that the mature spherical plasma HDL particles are not secreted directly from either of these sources, but are derived from a discoidal shaped precursor form consisting of HDL, apoprotein, lecithin and free fatty cholesterol. Transformation of this discoidal form into spherical particles (HDL) involves the action of the enzyme lecithin-cholesterol acyl transferase (LCAT) (Levy & Rifkind, 1980, p. 7).

Another source of HDL component described by Mahley in 1982 appears to be in the glyceride-rich VLDL and chylomicrons. He suggested

that the lipolysis of chylomicrons and VLDL triglyceride by LPL also results in transfer of apolipoproteins and phosphates to high-density lipoprotein.

The exact function of the HDL remains uncertain. Levy and Rifkind (1980) suggested that it may play a role in cholesterol efflux from the ti-sue, therefore decreasing the amount of cholesterol stores. It has also been postulated that the HDL or at least some of its components may interfere competitively with the uptake of LDL cholesterol by the tissue (Carew, Koschinsky, Hayes, & Steinberg, 1976; Heiss et al., 1980). These authors have hypothesized that the role of the high-density lipid is as a scavenger during normal VLDL lipolysis, where HDL picks up cholesterol along with phospholipids and apoproteins. A final explanation was developed by Miller and associates (1977). They suggested that during exercise the enzymatic activity of LPL increases which in turn increases the breakdown of VLDL to form HDL. If the HDL levels are increased, there may be a greater efflux of cholesterol from the tissue and periphery to the liver where it can be catabolized (Levy & Rifkind, 1980). These processes have been linked to the antiatherogenic effect and coronary heart disease. Not enough is written or known to permit acceptance or rejection of these suppositions at this time.

Factors which Influence Lipoprotein Concentration

While the processes linking HDL function to CHD and antiatherogenesis remains uncertain, research is more concrete with respect to factors which influence lipoprotein levels. They include diet, body composition, alcohol, smoking and exercise.

Diet

Diet has been cited as a variable which may influence blood lipoprotein concentrations. The average present day diet consists of approximately 40% fat, of which approximately 15% to 17% is saturated. Meat, other animal sources and some plant sources fall into this category. Grundy, Blackburn, Brown, Kwiterovich, Mattson, Schonfeld and Weidman (1982) suggested that the average American should reduce their saturated fat intake to 10% of their total calories. Albrink (1962) suggested that there should be a decrease in saturated fat intake and an increase in unsaturated and polyunsaturated fat. Currently available data are insufficient to concretely establish whether any particular diet could alter plasma cholesterol levels ("Dietary fiber, exercise and selected blood constituents," 1980). However, Quintao and co-workers (1971) found that diets rich in saturated and animal fats elicited a hypercholesterolemic effect; that is, an increased total serum cholesterol. Albrink (1982) and Friedman and Rosenman (1957) reported that unsaturated or vegetable oils have the opposite effect of lowering serum cholesterol, but no mention was made of its effect on high-density lipid cholesterol. Another study (Grundy et al., 1982) indicated a diet low in saturated fat and high in unsaturated or polyunsaturated fat, the very type of diet recommended for prevention of CHD, may not only lower total serum cholesterol, but also increase high-density lipid portion as well.

Intestinal absorption determines the quantity of cholesterol that accumulates in the body pools. Further, the amount of dietary

cholesterol absorption increases with intake in a linear fashion (Quintao et al., 1971). Keys, as cited by Grundy and associates (1982), stated that the most controlled metabolic studies have shown dietary cholesterol to increase total plasma cholesterol, thus giving support to the previous statements of Quintao et al. (1971). This increase in exogenous cholesterol is associated with an increase in low-density lipid levels and a subsequent decrease in high-density lipid levels (Levy & Rifkind, 1980). Conversely, dietary restrictions of exogenous cholesterol will decrease endogenous or serum cholesterol (Friedman & Rosenman, 1957).

It has also been found that the amount of fiber or complex carbohydrates in the diet has some effect on HDL levels. Diets high in complex carbohydrates seem to help decrease total serum cholesterol (Albrink, 1980; Liebman et al., 1983). This same effect was found with the addition of guar gum and pectin into the diet ("Dietary fiber, exercise and selected blood constituents," 1980). Heiss and associates (1980) found increases in HDL cholesterol, as well as decreases in total serum cholesterol, with high intakes of complex carbohydrates. Ernst, Fischer, Gordon, Rifkind, Little, Mishkel, and Williams (1980) found complex carbohydrates decreased HDL as well as high starch and higher sucrose intakes.

If caloric intake is less than caloric expenditure, weight loss will occur. It has been found that an inverse relationship exists between wieght loss and HDL concentrations (i.e., weight reduction is accompanied by increased HDL levels) (Heiss et al., 1980). Conversely,

it has been reported that obese individuals had lower high-density lipid levels than their counterparts, though it remains unclear whether this is a function of adiposity or of energy balance ("Dietary intake, exercise and selected blood constituents," 1980; Liebman et al., 1983; Phillips et al., 1981).

Body Composition

Body weight is generally divided into three components: bone, muscle, and fat. Bone and muscle weight is classified as lean body mass and the remainder is termed fat weight, which consists of adipose tissue.

Recent advances in sports physiology have led to an interest in the development of physiological profiles to describe the qualities and characteristics of the elite athletes in their respective sports (Wilmore, 1983). These profiles can be used to better understand the athlete and provide a basis for comparison to other elite athletes of the same sports. In these profiles, prediction of body composition is a parameter which is desirable and useful and can be estimated in various ways.

The underwater weighing technique is the most accepted method to determine body composition because of its accuracy and reliability (Wilmore, 1969a). The principle of this technique is that the density of the whole body is simply the ratio of the total body weight to the total body volume; the latter being estimated by the weight of the volume of water displaced when the body is completely submerged in water (Wilmore, 1969a). From this density or specific gravity measure,

an estimation can be made of the percentage of body fat and lean body weight.

Behnke and Wilmore (1974) stated that a certain minimal level of fat is necessary to sustain life. They estimated this to be in the range of two to five percent of total lean body weight for males. These values are quite low but, in view of the literature, it was found that long-distance runners had relatively low fat percentages, usually below 12%. Sprynarova and Parizkova (1971) found a mean value of 6.3% for a group of cross-country runners, average age of 22 years. Three other authors found data similar to this value at 7.5%, 7.9%, and 8.4%, respectively (Costill, Bowers, & Kammer, 1970; Costill, Thomason, & Roberts, 1973; Rusko, Havu, & Karvinen, 1978). A final study conducted by Pollock, Miller, and Wilmore in 1974 attained a mean value of 10.9%, slightly higher than the other data.

Low percent body fat, such as those found in cross-country runners, has a significant effect on measures of high-density lipoprotein cholesterol. A negative correlation between body fat content or relative body weight and HDL concentrations has been confirmed by a number of studies (Gordon et al., 1977; Schnabel & Kinderman, 1982). Brownell and associates (1982) and Heiss and co-workers (1980) found that weight reduction during exercise was related to elevation of HDL levels. Another study comparing weight lifters and a control group to runners who had lower body fat, found that the runners had significantly higher HDL levels as opposed to the other two groups (Clarkson et al., 1981). Further support of this was documented by Ready and Quinney (1982) when

they reported a significant elevation of HDL cholesterol and a decline in percent fat in men following a 16 week aerobic bicycle training program. A 20 week study of sedentary men found similar changes in body weight and lipoproteins (Miller et al., 1979). These results suggest that change in body composition can account for lipoprotein alterations.

Alcohol

Investigation of triglyceride response to alcohol intake suggested that some people respond to heavy alcohol intake by a rise in triglyceride and some do not. Castelli, Gordon, Hjortland, Abraham, Doyle, Hames, Hulley, and Zukel (1977b) reported that a man who drank 5 to 6 ounces of alcohol per week had higher plasma triglycerides than did one who drank no alcohol. Ernst and co-workers (1980) reported similar findings. However, another study noted that weekly ethanol intake was unrelated to serum triglyceride levels (Phillips et al., 1981).

Many authors have reported that weekly ethanol consumption positively correlates to rises in HDL concentrations (Castelli et al., 1977b; Ernst et al., 1980; Phillips et al., 1981). Koga, as cited in Castelli and co-workers (1977b), was the only reference found that reported a decrease in HDL serum after high doses of alcohol. In their study of five major populations, Castelli and associates (1977b) reported that alcohol consumption was positively associated with HDL cholesterol levels and it appeared to be a graded response even over the lower range of ethanol intake. Ernst and co-workers (1980) reported that this increase was irrespective of alcohol type (i.e., beer, wine or mixed

drinks), but found that wine produced a higher gradient response to HDL's than beer or mixed drinks. Overall, the majority of research has found increases in HDL's with weekly alcohol consumption in amounts of 5 to 20 ounces per week. How alcohol exerts this effect is unknown (Ernst et al., 1980), but Castelli and associates (1977b) presumed that this lipid effect was a metabolic response to alcohol, since alcohol is known to influence lipid metabolism and transport of lipids in some way.

Smoking

Smoking has also been cited as a variable which may influence blood lipoprotein concentrations. It is still unclear how cigarette smoking acts biologically to influence lipoprotein levels, but some mechanisms have been established. Nicotine and carbon monoxide appear to affect serum lipids and a biologic action of some component of cigarette smoke on HDL cholesterol seems reasonable (Criqui, Wallace, Heiss, Mishkel, Schonfeld & Jones, 1980). A study conducted on heavy (> 20 per day) and light (5-15 per day) cigarette smokers reported that heavy cigarette smokers had significantly higher triglyceride levels than light cigarette smokers (Billimoria, Pozner, Metselaar, Best & James, 1975). These authors also found triglyceride levels to be 52 ml/dl higher in smokers than in non-smokers.

With regard to high-density lipid levels, "cigarette smoking was found to be associated with an average difference in HDL cholesterol of about four mg% in men...a significant association between number of cigarettes smoked and HDL concentrations was demonstrated in men" (Garrison et al., 1978, p. 17). Support for this was cited by Criqui

and associates (1980). They reported that persons who smoked 1 to 19 cigarettes per day had an HDL level significantly lower than non-smokers, but higher than those who smoked 20 or more cigarettes per day. Again this indicates a possible dose-response effect. Other authors (Phillips et al., 1981; Nakamara, 1981) reported similar findings. Skiers who were smokers had significantly lower HDL cholesterol and TC/HDL ratios than non-smokers (Enger, Herbjornsen, Erikssen, & Fretland, 1977). Thus the response of high-density concentration to amount of cigarettes smoked seems well established.

Exercise

It has been well established by many researchers that exercise exerts an effect on lipoproteins (Adner & Castelli, 1980; Haskell et al., 1980; Huttunen, 1982; Leghtonen & Viikari, 1978). There has been an array of studies documenting the effects of different modes of exercise on lipid concentrations. The effects of these different modes will be discussed later. Some researchers have indicated that exercise significantly alters these aspects and others stated that exercise had no effect on these components.

Triglycerides. Many researchers have shown a negative correlation between aerobic training and triglyceride levels (Huttunen, 1982; Leghtonen & Viikari, 1978). Farrell and Barboriak (1980) cited significant decreases in triglyceride concentrations after eight weeks of endurance training. Brownell and associates (1982) and Hunt and White (1980) found similar results after ten weeks of aerobic training. However, these decreases in triglyceride levels were non-significant.

Only one study reported triglyceride levels to be unaffected after ten weeks of aerobic activity (Moore et al., 1979). A final study examined how one single exposure of bicycling affected serum lipids (Cullinane, Siconolf, Saritelli, & Thompson, 1982). They reported that there was no change in serum triglycerides four hours after the bout; however, at 24 hours after the two hour bout, triglyceride levels significantly decreased. By 48 hours after the exercise session serum levels were not significantly different from pre-exercising concentrations. These results demonstrate a delayed decrease in blood triglyceride response.

Total Cholesterol. An inverse correlation between exercise and TC has likewise been documented. Kinsman et al. (1980), Clarkson and associates (1981) and Dufaux and co-workers (1982) found that high and low intensities of aerobic training decreased total serum cholesterol. Except for long-distance runners, Schnabel and Kinderman (1982) found no significant decrease in TC levels of other sporting groups when compared to controls. Another author stated that no change in total cholesterol was found after eight weeks of running training (Moore et al., 1979).

In a study of marathon runners, average age 40 years, Adner and Castelli (1980) cited TC levels of 194 mg/dl in comparison to 199 mg/dl for their controls of comparable age. These TC levels were not significantly different. Another study investigating exercise and its effects on cholesterol reported TC concentrations of 186 mg% for male runners, average age 41.8 years (Brownell et al., 1982). A study

comparing marathon runners to controls (mean age 44 & 46 years, respectively) gave values of 187 mg% and 211 mg% for total cholesterol levels in these groups (Hartung, Foreyt, Mitchell, Vlasek & Gotto, 1980). Finally, in a study of ultra-marathon runners averaging 95.8 miles per week, Thompson, Nequin, Lesmis and Garfield (1982) reported average total cholesterol levels of 166.3 mg/dl.

High-Density Lipids. Positive changes in high-density lipid levels, when related to exercise, have been cited by many authors. When different durations of exercise were compared in training individuals, long-distance runners (40 miles per week) had significantly higher HDL levels than did short-distance runners of 15 miles per week (60 mg% to 47 mg%); controls in this study recorded values of 35 mg% (Rotkis, Cote, Coyle & Wilmore, 1980). An additional study on swimmers found linear increases up to mid-season with a plateau toward final exercise measurements (Gale, 1981). Brownell and his associates (1982) measured mean serum levels of 42 mg% after ten weeks of aerobic exercise, while Thompson et al. (1982) reported levels of 65.7 mg/dl for ultra-marathon runners. A final study recorded HDL values of 65 mg% and 43 mg% for marathon runners and inactive subjects, respectively (Hartung et al., 1980).

When comparing exercise groups to control or sedentary groups, similar findings were reported. Adner and Castelli (1980) compared 50 long-distance runners (500 miles/year) to controls from the Framingham study and found values of 54 mg/dl and 45 mg/dl for exercising and control groups, respectively. Other authors used weight lifters as a control group and found significant differences when they compared

these individuals to aerobically trained individuals (Clarkson et al., 1981). Finally, Lehtonen and Viikari (1978) found a significant difference in HDL levels of lumberjacks and a control group of electricians.

In reviewing the literature a few authors found no significant increase in HDL levels with aerobic exercise. Dufaux and associates (1982) found no significant changes in HDL's after eight weeks of aerobic training. Two other studies found no significant increases in high-density blood lipids after nine weeks of aerobic training (Hunt & White, 1980; Ready & Quinney, 1982). Finally, only one study was found to negate all results of increases in HDL's with training. Moore et al. (1979) found that after ten weeks of training eleven bank executives, HDL levels fell when expressed in absolute values and percentages.

A variety of modes were used for exercise training and their effects on serum lipoproteins in the studies that have been conducted. Dufaux et al. (1982), Clarkson and associates (1981) and Adner and Castelli (1980) used running as a mode of training or comparison. Ready and Quinney (1982) trained subjects for nine weeks on a bicycle ergometer, while Gale (1981) investigated effects of training on HDL's in collegiate swimmers. Finally, an extensive study conducted by Schnabel and Kinderman (1982) compared various modes of exercise (including long-distance running, biking, handball, soccer and walking) to determine the various effects these modes had on lipoprotein levels.

The previously cited studies reported results on subjects who trained at least three days per week for eight weeks. However, one

study reported that an acute exercise bout may increase HDL levels (Huttunen, 1982). Haskell et al. (1980) reported that HDL's were not affected by a short intense bout on the treadmill. Lastly, Enger, Stromme and Refsum (1980) suggested that one single exposure to heavy exercise immediately raises HDL's and lowers serum triglycerides. This elevation returned to pre-exercise levels four days after the exercising bout.

Total Cholesterol/High-Density Cholesterol Ratio. In addition to rises in HDL cholesterol concentrations consequent changes can be found in TC/HDL ratios when the effects of exercise are compared. Here a lower TC/HDL ratio is more beneficial than a higher one. Support of these changes in TC/HDL ratio are given by a few authors (Clarkson et al., 1981; Gale, 1981; Moffatt & Gilliam, 1979). In comparing high aerobic activities to low aerobic activities, Schnabel and Kinderman (1982) gave a clear explanation. Statistically significant differences in the TC/HDL ratios from control values were shown as a result of continuous training for at least eight weeks. The ratios between high and low aerobic activities correlated closely, though not perfectly. Correlation analysis within groups yield a coefficient between -0.80 and 0.98, indicating higher aerobic activities to lower TC/HDL ratios (i.e., long-distance runners 3.01 to controls 4.40). Hartung et al. (1980) found similar results with marathoners having a ratio of 2.9 compared to a 4.9 for controls. In another study comparing marathon runners and non-marathoners, TC/HDL ratios were examined. The non-marathon runners had a ratio of 4.6, while the marathoners' mean value

was 3.8. In an additional study, Clarkson et al. (1981) compared TC/HDL ratios in 20 to 23 year old runners, weight-lifters and controls and reported values of 2.32, 3.44, and 3.37, respectively.

Maximum Oxygen Consumption

Maximum oxygen uptake, maximum aerobic power or max$\dot{V}O_2$ has been defined by Fox and Mathews (1981) as a measure of maximal functional capacity of the cardiorespiratory system or oxygen transport system. It is considered by most exercise physiologists to be the single most accurate measure of endurance fitness (Fox & Mathews, 1981). The physiological factors which are involved in the oxygen transport system are: stroke volume (the amount of blood pumped by the left ventricle per minute), heart rate and arteriovenous difference (the difference between the oxygen content of arterial and mized venous blood).

> Thus, the increase in max$\dot{V}O_2$ is brought about by two main changes: one, an increased oxygen delivery to the working muscle through an increased cardiac output (heart rate times stroke volume) and two, increased oxygen extraction from blood by skeletal muscle (Fox & Mathews, 1981, p. 313).

Because body size affects oxygen consumption, max$\dot{V}O_2$ is usually expressed in milliliters of oxygen per kilogram of body weight per minute ($ml \cdot kg \cdot min^{-1}$) when measured in a laboratory (Weltman & Stamford, 1982).

There are many other factors which affect max$\dot{V}O_2$ of an individual. Some of these factors are physiological and others have to do with age, sex, body weight, genetics and training. The effect training has on the amount of oxygen that can be consumed per minute during maximal

exercise has been studied extensively. It has been shown that max$\dot{V}O_2$ increases with training (Astrand & Rodahl, 1977; Fox & Mathews, 1981; Weltman & Stamford, 1982). Although max$\dot{V}O_2$ is thought to be a product of training load, it is a remarkably stable feature in the trained individual (Pollock, 1973). Aerobic training can improve max$\dot{V}O_2$ by increasing blood volume and pumping capacity to the heart. This, in turn, makes more oxygen available to the muscles during exercise and increases their ability to use oxygen (Weltman & Stamford, 1982). Astrand and Rodahl (1977) and Weltman and Stamford (1982) have stated that maximum oxygen uptake can increase by 10% to 20% after a "regular" training period of ten weeks. Another author suggested a 5% to 20% improvement for college-age men following at least eight to twelve weeks of training (Fox & Mathews, 1981). "This increase is highest for athletes who compete and train for endurance types of activities" (Fox & Mathews, 1981, p. 313). Research conducted by Ready and Quinney (1982) found an increase of 40.5% (45.7 to 64.2 $ml \cdot kg \cdot min^{-1}$) in max$\dot{V}O_2$ after nine weeks of aerobic activity in untrained male subjects who exercised 30 minutes per day, four days per week, at 80% of maximum aerobic power.

As indicated earlier, a test for max$\dot{V}O_2$ is a standard measurement of cardiovascular endurance fitness. The higher the value for max$\dot{V}O_2$ the better the cardiorespiratory fitness level one has achieved. It has been noted by Boileau, Mayhew and Lussier (1982) that long-distance running necessitates a relatively great amount of aerobically derived energy. Furthermore, successful performance in distance running has been attributed primarily to the athlete's ability to consume oxygen

maximally. However, this alone may not be the predicting factor (Boileau et al., 1982; Conley, 1981; Costill et al., 1973; Costill, Fink, & Pollock, 1976; Pollock, 1977).

Many authors have classified the well trained, highly trained or elite athlete by maximum oxygen consumption values. "It appears that a high max$\dot{V}O_2$ (> 70 ml·kg·min^{-1}) is a requisite for distance running success" (Pollock, 1977, p. 319). This value was documented from a profile of 20 elite distance runners. The mean max$\dot{V}O_2$ in this study was 74.1 ml·kg·min^{-1}. In a study on muscle fiber composition of elite marathoners, Costill and co-workers (1976) reported a mean max$\dot{V}O_2$ value of 77.4 ml·kg·min^{-1} for these athletes. Weltman and Stamford (1982) stated that the elite marathon runner may consume as much as 80 ml·kg·min^{-1} or more of oxygen during maximal exercise compared with approximately 42 ml·kg·min^{-1} for the average college-age man. Other authors have classified "highly trained" athletes with max$\dot{V}O_2$ values of 71.7, 66.6, and 72.9 ml·kg·min^{-1} (Conley, 1981; Costill et al., 1973; Schnabel & Kinderman, 1982). Thompson and co-workers (1982), in a profile of ultra-marathoners (average age 31 years), cited these runners as having an average max$\dot{V}O_2$ of 60.5 ml·kg·min^{-1}.

Maximum heart rate tends to have a positive linear correlation to max$\dot{V}O_2$ values. Fox and Mathews (1981) have noted that heart rate increases approximately linearly with maximum oxygen uptake, and maximal heart rate of subjects during this type of exercise is about 195 beats per minute. This linear trend seems to break down toward maximal effort, where heart rate tends to plateau and max$\dot{V}O_2$ continues to increase

somewhat. In studies on elite long-distance runners Conley (1981), Costill et al. (1976) and Pollock (1977) reported maximum heart rate values of 184, 198, and 197 beats per minute (bpm), respectively. Two other authors reported maximum values of 191 bpm for highly trained distance runners (Costill et al., 1973; Fox & Mathews, 1981). Thompson and associates (1982) reported maximum attained heart rate of 175 bpm for ultra-marathoners, while Weltman and Stamford (1982) reported that highly trained athletes achieved an average maximum heart rate of 184 pbm.

The literature reviewed indicated variant results among studies when examining the relationship of HDL's and max$\dot{V}O_2$ in highly fit or elite runners. Brownell and associates (1982) found that positive changes in max$\dot{V}O_2$ did not correlate significantly with changes in lipoprotein measures. Huttunen (1982) obtained a weak positive relation between HDL's and maximum oxygen consumption values after four months of aerobic running training. However, research conducted by Hartung et al. (1980) found that highly trained athletes had higher levels of HDL's than sedentary counterparts of the same age and sex. Haskell and co-workers (1980) supported this and stated,

> ...endurance athletes with very high endurance capacities (max$\dot{V}O_2$), compared with less active persons who have average-to-low capacities, have higher high-density cholesterol levels and these two variables are positively and significantly associated. Furthermore, these levels can be 10-40% higher for active males and females, primarily the endurance runner (p. 56).

Data collected by Miller, Rao, Lewis, Bjorsvik, Myhe and Mjos (1977) also supported these findings. They found a strong positive correlation

between max$\dot{V}O_2$ and HDL cholesterol and stated, "these findings suggested that physical training itself raises the plasma HDL level through an effect on the synthesis and/or catabolism of the HDL subclass" (p. 111).

Summary

Research has suggested that the amount of blood lipids and lipoproteins can be altered by diet, body composition, alcohol, smoking and/or exercise. The majority of research has cited that diets low in saturated fats and higher in unsaturated and polyunsaturated fats, the very type of diet recommended for the prevention of CHD, may not only lower TC, but also increase the HDL portion as well (Albrink, 1982; Grundy et al., 1982; Quintao et al., 1971).

Behnke and Wilmore (1974) stated that a minimal amount of fat, from two to five percent, is necessary to sustain life. Low percent fats, such as those found in cross-country runners, have been correlated to high levels of HDL concentrations (Gordon et al., 1977). Other authors have found that weight reduction and/or decreases in percent fat were related to elevation in high-density cholesterol levels (Brownell et al., 1982; Ready & Quinney, 1982).

Overall, the majority of research has cited increases in HDL's with weekly alcohol consumption in amounts of 5 to 20 ounces per week (Ernst et al., 1980; Phillips et al., 1981). This alteration in lipoproteins seems to be related to the impingement of lipid metabolism and transport of lipids (Castelli et al., 1977b).

The effect smoking has on lipoproteins has been documented by many researchers. Their findings seem to be in agreement and suggest that cigarette smoking increases triglyceride levels and more importantly decreases the HDL concentrations (Criqui et al., 1980; Phillips et al., 1981).

Presently, exercise seems to be the most important factor involved in the alteration of lipoprotein measures. It has been enumerated by many researchers that serum triglycerides decrease with aerobic training or exercise. Some authors have found significant decreases in triglycerides (Cullinane et al., 1982; Farrell & Barboriak, 1980), while others have found non-significant decreases (Brownell et al., 1982; Hunt & White, 1980).

An inverse correlation between exercise and TC has been likewise documented, where decreases in serum cholesterol have been attained after aerobic exercise (Dufaux et al., 1982; Kinsman et al., 1980; Schnabel & Kinderman, 1982). In comparing active individuals to sedentary individuals it is apparent that the former possess lower TC levels than the latter, though these differences may not always be significant (Adner & Castelli, 1980; Hartung et al., 1980). It appears that these decreases in TC are related to increases in HDL's. Levy and Rifkind (1980) have suggested that high-density lipid cholesterol aids in the efflux of cholesterol from the periphery to the liver where it can be catabolized.

Positive changes in HDL's, when related to exercise, have been cited by many authors. Different modes of aerobic exercise such as

bicycling, swimming and running seem to elicit increases in these components (Ready & Quinney, 1982; Gale, 1981; Adner & Castelli, 1980). Furthermore, it has been documented that aerobically active individuals attained significantly higher HDL levels than their sedentary counterparts (Adner & Castelli, 1980; Clarkson et al., 1981; Hartung et al, 1980). A few authors have found non-significant increases in HDL's with exercise (Hunt & White, 1980; Ready & Quinney, 1982), while only one study was found to negate all results of increases in HDL's with aerobic training (Moore et al., 1979).

Exercise has thus been looked upon favorably because the positive changes elicited in lipoproteins seem to be negatively associated with the atherogenic process (Castelli et al., 1977a).

CHAPTER III

METHODS

Introduction

In this study, four basic variables were measured before and after a season of cross-country running. The first variable was maximum oxygen consumption which was used to measure cardiorespiratory fitness. The second variable was body composition, determined by hydrostatic weighing (Katch, 1969). Thirdly, blood draws were taken to determine TC, HDL, and TC/HDL ratio. The final component measured was dietary intake. This chapter will present the methods, experimental treatment and procedures employed for subject selection, max$\dot{V}O_2$ testing, body composition, blood assays and dietary intakes.

Subject Selection

Forty-five healthy male cross-country runners, ranging in age from 18 to 24 years, from the University of Wisconsin-La Crosse participated in this study. All subjects signed an informed consent (see Appendices A & B) prior to blood draws, max$\dot{V}O_2$ testing and hydrostatic weighing. Due to injury, two participants were dropped from the study, thus the final results included a total of forty-three individuals.

Experimental Treatment

The training program was set up for the intercollegiate cross-country runners by the coach at the University of Wisconsin-La Crosse. It was specified that the runners adhere to the training program.

Weekly running mileages were recorded for each subject and average weekly distances for the team were calculated (see Appendix C). Runners who did not qualify for the national championships trained and competed for nine weeks. Those who qualified for either the NAIA or the NCAA National Championships trained and competed for twelve weeks. Retesting of the previously described variables began immediately after the last regular season meet (October, 29) or post-season meet (November 20).

Maximum Oxygen Consumption

Max$\dot{V}O_2$ was measured during a treadmill run, using the Beckman Metabolic Measurement Cart (BMMC). Prior to the actual testing each individual participated in a practice session in order to become familiar with treadmill running (Quinton Pit Model). The test protocol was explained to the participant at this time. The treadmill test was a modification of the protocol set up by Butts (1982) (see Appendix D).

For a warm-up the subjects walked on the treadmill at 3.5 mph, 10% grade, for three minutes. At the conclusion of the warm-up the grade was decreased to zero degrees and the subjects began to run at 7 mph. Every two minutes the incline of the treadmill was increased by 2.5%, up to a maximum of 10%. After 10 minutes, the grade remained the same (10%) and the speed was increased by one half mile per hour every two minutes. The test was terminated at the subject's request or when the participant could no longer continue. At that time they straddled the treadmill and the speed was decreased to 3.5 mph with no incline, so that the subject could cool down for approximately five minutes. Following the cool down process, the subjects were assisted off the

treadmill and walked around the room.

During the run gas samples were collected every minute and analyzed using the BMMC. The cart was turned on at least one hour prior to the first scheduled appointment of the day. The gas analyzers (LB-2 & OM-11) were calibrated before and after every test using a control tank of known O_2 and CO_2 percentages, previously determined by the Scholander technique. A two way Rudolph valve, appropriate rubber mouth piece and adjustable head gear were also used for gas collection. Room temperature, barometric pressure and volume of air flow were also calibrated.

Subjects reported to the Human Performance Laboratory for their appointed times and changed into their exercising clothing. The BMMC was then programmed with its digital card and the weight of the subject, measured to the nearest 0.25 of a kilogram (kg), was entered. Recordings of each minute were taken of the subject's VE, VO_2, VCO_2, RER, $FECO_2$, and FEO_2. Relative perceived exertion (RPE), using the Borg scale (Borg, 1973), was recorded every two minutes. Prior to the start of the test instructions about the relative perceived exertion scale were given to the subjects as cited by Butts (1982). The BMMC and its calibrations were checked at the conclusion of each test and prior to the commencement of the next test. The highest $\dot{V}O_2$ observed, in conjunction with the highest RER (> 1.00) and VE, was considered the max $\dot{V}O_2$, if maintained for one complete minute.

Maximum Heart Rate (MHR)

During the treadmill test a modified CM-5 lead system, with "Quick

Prep" electrodes, was used to monitor heart rate. Each subject's chest was cleaned of oil with alcohol and dried with gauze. Chest hair that interfered with electrode placement was shaved off. Two leads were placed on the left and right midclavicular lines in the second intercostal space. The third lead was placed on the right midclavicular line at the edge of the ribs. The final electrode was placed in the fifth intercostal space at the left anterior axillary line. Monitoring was continuous via a Burdick EK-4 electrocardiograph and an electrocardiogram (EKG) was taken the last 15 seconds of each minute and multiplied by four to determine the heart rate for one minute. Heart rates were later recorded along with the BMMC data on an appropriate form (see Appendix D).

Body Composition

Body composition was determined by hydrostatic weighing. Residual volume (RV) was determined by the oxygen dilution technique (Wilmore, 1969a) outside the weighing tank while the subject was seated in front of the Collins Nitrogen Analyzer. While the procedure of the test was explained to the subject, the total system was flushed with oxygen three times to clear out the nitrogen. A noseclip was secured on the subject's nose and the mouth was comfortably positioned around the rubber mouth piece. For determination of RV (see Appendix E) the values for alveolar nitrogen (AN_2), impurity nitrogen (IN_2), equilibrium nitrogen (EN_2) and final nitrogen (FN_2) were used. Two trials were conducted to avoid error (Wilmore, 1969b) and the lowest RV value obtained was used in calculation of body density.

Dry weight was then determined prior to entering the immersion tank, using a Continental Health-O-Meter scale and recorded to the nearest 0.25 kg. Height was also recorded at this time to the nearest 0.25 centimeter. Subjects were required to fast 6 to 12 hours prior to the immersion procedure.

Subjects were then instructed to shower thoroughly, soaking the body and swimming suit. They entered the immersion tank via a ladder and assumed a comfortable position on the light-weight polypropylene chair which was suspended by a Chatillon Autopsy Scale accurate to 0.25 kgs. Participants then eliminated air bubbles from the skin and from within the swimming suit.

The procedure of hydrostatic weighing was explained and a noseclip was placed on the subject's nose. The subject then forcefully expired as much air as possible from their lungs in the way that RV was determined. As the subject's head was slowly drawn toward his knees and gradually submerged, he continued to expell as much air as possible. While the subject was completely immersed, the technician steadied the scale and recorded the underwater weight to the nearest 0.25 kg. The technician then tapped on the tank signalling the subject to surface.

The procedure was repeated up to ten times until three similar readings to the nearest 0.25 kg were obtained. The subject was then instructed to get out of the tank via the ladder and redress in the changing room. The weight of the apparatus and the water temperature were then recorded. Body density was then determined using the mass of the subject in the air (M_A), in the water (M_W) and the subject's RV

and gastrointestinal air volume (see Appendix E).

Once the body density was determined the percent body fat was calculated using the formula developed by Brozek, Grande, Anderson and Keys (1963) (see Appendix E). Percent fat was then expressed in kilograms of body weight and subtracted from the total body weight to derive lean body values. The hydrostatic weighing was completed by the same technician, not directly involved with this study.

Blood Assays

In this study blood samples were taken prior to training and after training for TC, HDL, and TC/HDL ratio analysis. All subjects fasted for at least twelve hours (overnight) prior to blood collection with a 24 hour abstention from alcohol. Adherence to these restrictions was checked by questioning each individual at the time blood was drawn. Those who failed to fast and/or abstain from alcohol were rescheduled for a later draw. Venipuncture was performed by a medical technologist at the Human Performance Laboratory of the University of Wisconsin-La Crosse. Draws of 10 ml were taken from the anticubital vein with the subject in a sitting or lying position. A tourniquit was used, then released before sampling to avoid artifactual increases in the concentration of plasma lipids.

All samples were placed to coagulate for approximately one hour and then centrifuged. Plasma was separated from the whole blood and immediately frozen in a refrigerator in the blood laboratory. After draws were taken from all subjects, duplicate samples were analyzed using the following procedure.

General Set-Up Procedure (DMA Cholesterol Kits)

Prior to analyzing the samples, the water bath was turned on to a temperature of 25° C and the Digi spectrometer was warmed up. Blood samples and controls were then thawed. If controls were not frozen new bottles of Data-trol Normal and Data-trol Abnormal were reconstituted according to the directions on the bottles. Likewise, Data-zyme Cholesterol Reagents were reconstituted with dionized water, as stated on the labels, and swirled until dissolved. Controls and reagents were allowed to stand at room temperature for ten minutes before using.

HDL Procedure

Duplicate 13x100 disposable test tubes were labeled with a red wax pencil for controls (Data-trol Normal and Data-trol Abnormal) and unknowns (samples). Next, five microliters (ul) of serum samples, controls and unknowns, were placed in their respective tubes. One hundred ul of Isopol Precipitating Reagent was then added to each tube. Following this, the tubes were vortexed and centrifuged for 10 minutes at 1650 rpm. The supernatant contained the HDL and the pellet contained the VLDL, LDL and protein.

Color Reaction

Duplicate 13x100 disposable test tubes of blanks (dionized H_2O), standards (HDL = 50mg%), controls and unknowns were labeled. Two ml of Data-zyme Cholesterol Reagent was then placed into each tube and warmed in a water bath for 10 minutes. After 10 minutes, 100 ul of HDL serum, blank and standard were added to their respective tubes. The

samples were then vortexed gently and incubated (25° C) in the water bath for 10 minutes, to develop the color. The digi spectrometer was then zeroed at 500 nm using the reagent blank. Optical density (absorbance) of each sample was then recorded from the Digi spectrometer. Values for HDL (mg%) were obtained by the following equation:

$$\text{HDL (mg\%)} = \frac{\text{absorbance of unknown (nm)}}{\text{absorbance of standard (nm)}} \text{ X HDL standard (50mg\%) X 1.2}$$

The 1.2 factor corrected for the sample dilution during the precipitation procedure (Data Medical Associates, 1983).

Total Cholesterol Procedure

For TC assays, duplicate 13x100 disposable test tubes were labeled: blanks, standard (TC = 200mg%), controls and unknowns. First, 2.0 ml of Data-zyme Cholesterol Reagent was dispensed in each tube and warmed for 10 minutes. Next, 20 ml of each sample was added to its respective tube. The assays were mixed gently and incubated in the water bath for 10 minutes. The Digi spectometer was then zeroed with the blank reagent. Optical density (absorbance) of each sample was then recorded from the Digi-spec. Finally, TC values (mg%) were ascertained by the following equation:

$$\text{TC (mg\%)} = \frac{\text{absorbance of unknown (nm)}}{\text{absorbance of standard (nm)}} \text{ X TC standard (200 mg\%)}$$

(Data Medical Associates, 1983)

Values for TC, measured in mg%, were determined by averaging the two samples if their difference was less than or equal to 10mg%. HDL values were averaged if the difference between the two samples was less

than or equal to 5 mg%. If differences were greater than the aforementioned margin, assays were reanalyzed. TC/HDL ratio was then determined by dividing the final HDL fraction into the final TC fraction. These data were then entered in the university computer for later tabulation.

Diet

Although runners' diets were not controlled, they were assessed at pre-, mid- and post-season testing to determine if any alterations had taken place or if diets had remained unchanged (see Appendix F). Food items which were thought to alter TC or HDL serum levels were analyzed. Thus, intakes of meats, such as beef, poultry, pork and fish; dairy products such as milk, cheese and ice cream; fats like mayonnaise, butter and salad dressing; eggs; and alcohol were recorded using one week dietary recall sheets. Analysis of variance with repeated measures ($p < .01$) was used to compare dietary intakes from pre- to mid-season, pre- to post-season, and mid- to post-season.

Statistical Treatment

Standard descriptive methods were employed to describe the subjects and their diets. Means and standard deviations were tabulated for each variable previously described. A dependent t-test was used to compare the pre- and post-training measures to determine if any significant ($p < .01$) changes in any variables occurred. Dietary analyses were assessed as previously discussed.

CHAPTER IV

RESULTS AND DISCUSSION

Introduction

The purpose of this study was to examine the effects of training on max$\dot{V}O_2$, body composition and blood concentrations of TC, HDL, and TC/HDL ratio of male intercollegiate cross-country runners. It should be noted that these runners from the University of Wisconsin-La Crosse had a very successful season and sent two teams, each consisting of seven men, to the National Championships. The second team placed thirteenth at the NCAA division III Championships, while the first team placed second at the NAIA Championships. This chapter presents the descriptive characteristics, max$\dot{V}O_2$ responses, dietary analyses and blood variables from pre-season and post-season testing.

Descriptive Characteristics

Forty-three male cross-country runners from the University of Wisconsin-La Crosse participated in this study and their descriptive characteristics are presented in Table 3. The average age for the runners was 19.8 years. Their mean height was 174.2 cm. Pre-season weight averaged 64.3 kgs, which was not significantly ($p > .01$) different from the average post-season weight of 64.5 kgs. There was no significant ($p > .01$) change in percent fat from pre- to post-training (8.7% and 8.8%, respectively). Lean body weight also was not significantly ($p > .01$) different from pre- to post-season training (58.7 and 58.9 kgs,

respectively). Therefore, there were no significant ($p > .01$) changes throughout the season in any of the descriptive parameters.

Table 3. Descriptive characteristics of the male cross-country team including means, standard deviations and ranges of age, height, and body composition (n = 43).

Variable	Mean		SD		Range	
	Pre	Post	Pre	Post	Pre	Post
Weight (kgs)	64.3*	64.5	5.15	5.09	55.2-76.7	56.1-77.7
% Body Fat	8.7	8.8	3.46	3.06	2.7-17.0	3.3-16.5
LBW (kgs)	58.7	58.9	5.49	5.05	49.5-70.7	50.7-71.5

* One subject, because of a broken eardrum, was unable to be hydrostatically weighed and his pre-season body fat and LBW calculations were excluded from these data.

In a study of elite runners Pollock (1977) and Costill et al. (1976) reported body weights of 63.0 and 64.0 kgs, respectively, and body heights of 176.8 and 177.8 cm, respectively. While the weight of the subjects in the present study were almost identical to the aforementioned studies, their heights were slightly less. However, they were similar to the value of 173.9 cm. reported for well trained long-distance runners by Costill et al. (1973). As expected, very little change was found between pre- and post-season weight since the athletes were highly trained when pre-season data were collected. Prior to pre-testing the team averaged 42.8 miles per week during the summer (see Appendix C).

Behnke and Wilmore (1974) stated that long-distance runners average 12% body fat or less. The results of this study showed that this cross-country team was much lower with values of 8.7% and 8.8% for pre- and

post-season testing, respectively. In an analysis of aerobic performance capacity Rusko and associates (1978) found average body fat to be 8.4% for long-distance runners. Costill et al. (1973) reported 7.9% body fat for long-distance runners which is similar to that found in the present study. The lower values and lack of significant change found in the present study can again be attributed to the high fitness level of the runners at the pre-testing time. Research conducted by Wilmore (1983) stated that exercise could result in changes in body composition. However, inspection of these present data suggested that alterations in body composition with exercise were minimal in the highly trained athlete who was already low in body fat composition. Since there was little change in total body weight and percent fat in this study, lean body weight showed little variation.

Physiological Characteristics

Physiological responses to the treadmill testing are recorded in Table 4. There was a significant ($p < .01$) difference in mean pre- to post-season max$\dot{V}O_2$ values when expressed in $ml \cdot kg \cdot min^{-1}$ as well as in $L \cdot min^{-1}$ (69.8 to 71.8 and 4.4 to 4.7, respectively). Although the average maximal heart rates tended to increase from 192.7 to 194.0 bpm this difference was not statistically ($p > .01$) significant. Likewise, the average treadmill time was not significantly ($p > .01$) different from pre- to post-season testing. The maximal ventilation volume was 144.6 $L \cdot min^{-1}$ in the pre-test and 144.1 $L \cdot min^{-1}$ in the post-test which was not significantly ($p > .01$) different. There was, however, a significant ($p < .01$) decrease in the respiratory exchange ratio from pre- to post-training (1.09 to 1.07, respectively).

Table 4. Physiological characteristics of 43 male cross-country runners including means, standard deviations and ranges of max$\dot{V}O_2$, maximum heart rate (MHR), treadmill time (TMT), maximum ventilation volume (V_E) and respiratory exchange ratio (RER).

Variable	Mean		SD		Range	
	Pre	Post	Pre	Post	Pre	Post
Max$\dot{V}O_2$ ($L \cdot min^{-1}$)	4.4	4.7*	0.37	0.45	3.6- 4.5	3.8- 5.5
Max$\dot{V}O_2$ ($ml \cdot kg \cdot min^{-1}$)	69.8	71.8*	5.03	4.49	54.2- 78.2	57.2- 80.3
MHR (bpm)	192.7	194.0	8.68	8.82	167.0-215.0	171.0-215.0
TMT (min)	15.5	15.7	2.32	2.08	8.0- 20.0	8.0- 20.5
V_E ($L \cdot min^{-1}$)	146.5	144.1	14.71	17.66	105.6-175.1	99.7-173.9
RER	1.09	1.07*	0.05	0.04	1.02-1.24	0.98-1.24

* $p < .01$

Maximum Oxygen Consumption

In discussion of physiological characteristics of long-distance and cross-country runners, Pollock (1977) stated that a high max$\dot{V}O_2$ of greater than 70 $ml \cdot kg \cdot min^{-1}$ was a requisite for distance running success. The pre-training max$\dot{V}O_2$ value for the present study fell slightly below this value, but the post-training value was above it. On this basis these athletes could be classified as being potentially successful runners. Evidence of this was provided by their second and thirteenth place finishes at the respective national meets.

The values for max$\dot{V}O_2$ in the present study can be compared to values attained by other elite class runners. Pollock (1977), Costill et al. (1976) and Weltman and Stamford (1976) conducted profile studies of elite cross-country runners and reported max$\dot{V}O_2$ values of 74.1, 77.4, and 80.0 ml·kg·min^{-1}, respectively. Values for other highly trained athletes have been previously documented at 71.7, 66.6, and 72.9 ml·kg·min^{-1} by Conley (1981), Costill et al. (1973), and Schnabel and Kinderman (1982), respectively. The values for pre- and post-training in the present study were below those cited for the elite athlete. However, they were very similar to those reported for the highly trained athlete.

When expressed in absolute terms (L·min^{-1}) the max$\dot{V}O_2$ values reported by Pollock (1977), and Costill and associates (1976) were 4.6 and 4.0 L·min^{-1}, respectively. Conley (1981), Costill and co-workers (1976) and Schnabel and Kinderman (1982), on the other hand, reported absolute max$\dot{V}O_2$ values of 4.6, 4.2 and 4.8 L·min^{-1}, respectively. In the present study there was an increase in the absolute max$\dot{V}O_2$ from pre- to post-training. The post-season value of 4.7 L·min^{-1} was above the values reported by Pollock (1977), Conley (1981), and Costill and associates (1973) and similar to the values documented by Costill et al. (1976) and Schnabel and Kinderman (1982).

The effect that training has on the amount of oxygen that can be consumed during maximal exercise has been studied extensively. Astrand and Rodahl (1977) and Weltman and Stamford (1982) documented increases of 10% and 20% after an aerobic training period of at least 10 weeks. Fox and Mathews (1981) suggested a 5% to 20% improvement in max$\dot{V}O_2$ following at least 8 to 12 weeks of training. These studies dealt with less

active individuals. Few studies have documented the effects of a competitive season on the max$\dot{V}O_2$ of highly trained athletes. The maximum oxygen consumption values in the present study, expressed in $ml \cdot kg \cdot min^{-1}$, increased by three percent from pre- to post-season training. This increase was small, but significant ($p < .01$). When expressed in absolute terms, a six percent increase was noted. In the highly trained individual, as training continues, further increases in max$\dot{V}O_2$ are difficult and slow in occurring because of approaching genetic limits (Weltman & Stamford, 1982). Even though the subjects in this study were considered highly trained at the beginning of the season, the training program successfully increased max$\dot{V}O_2$.

As Table 4 indicates, there was no significant change in maximum heart rate (MHR) from pre- to post-testing. Many studies in the literature have shown young runners to have MHR values of approximately 182 to 198 bpm (Conley, 1981; Costill et al., 1973; Costill et al., 1977; Fox & Mathews, 1981; Pollock, 1977; Thompson et al., 1982; Weltman & Stamford, 1982). The values for this study were similar to those found by Costill and associates (1977) and Fox and Mathews (1981). Further, the MHR's in the present study were higher than the values cited by Conley (1982), Thompson and associates (1982) and Weltman and Stamford (1982), but lower than those reported by Costill et al. (1976) and Pollock (1977).

With training, MHR is either unchanged or slightly decreased (Fox & Mathews, 1981). This decrease is particularly evident in athletes engaged in endurance training though it may take months or even years

to be manifested. If a decrease is found in MHR it is either related to an increased heart volume due to cardiac hypertrophy or to a decreased sympathetic drive and/or an increased parasympathetic drive (Fox & Mathews, 1981).

Treadmill Time

The actual running time on the treadmill test was recorded regardless of when the individual reached his max$\dot{V}O_2$. This time on the treadmill increased slightly, though not significantly ($p > .01$) over the course of the season. During the initial test the runners were highly motivated to attain the highest max$\dot{V}O_2$ value they possibly could. In this treadmill test most of the runners experienced a plateau or decrease in their O_2 consumption but were motivated to continue running. This indicated the runners reached their max$\dot{V}O_2$ prior to the actual finish of the treadmill test. Although the runners were able to take in, transport and utilize more O_2 than in pre-season testing, the treadmill time was not significantly ($p > .01$) higher in post-season testing. This would indicate that the increased cardiorespiratory fitness occurred without a concommitant increase in treadmill time, possibly due to a decrease in motivation of the runners in the post-season testing.

Maximum Ventilation Volume

Maximum ventilation volume (V_E) slightly increased, but not significantly, through the course of this study. The values of 141.1 and 144.7 $L \cdot min^{-1}$, for pre- and post-tests were similar to those reported

by Sprynarova and Parizkova (1971) of 142.6 $L \cdot min^{-1}$, but less than the volumes cited for elite long-distance runners by Pollock (1977).

Ventilatory volume at maximal exercise is related to physical size; so, theoretically, a taller and/or heavier individual, compared to a shorter and/or lighter individual, will have a greater ventilatory volume. Values of body height, body weight and ventilation volumes in this study were similar to those cited by Pollock (1977) and Sprynarova and Parizkova (1971).

Respiratory Exchange Ratio

Even though the respiratory exchange ratio (RER) was significantly ($p < .01$) lower in post-testing as compared to pre-training, the values were well above 1.00 indicating that a max$\dot{V}O_2$ was attained. RER is the ratio of CO_2 volume/O_2 volume utilized (Astrand & Rodahl, 1977). For the RER value to have significantly decreased, there had to be a decrease in CO_2 production, an increase in the amount of O_2 that was utilized or a combination of these factors.

For example, during short-term exhaustive exercise, the buffering of lactic acid causes large quantities of CO_2 to be produced. The point at which this increase in lactic acid, thus increase in CO_2 occurs, is referred to as the anaerobic threshold (Fox & Mathews, 1981). With training the anaerobic threshold is thought to increase (Fox & Mathews, 1981). If this occurs an individual could work at a higher intensity without producing as much lactic acid or CO_2. This decrease in CO_2 could, in turn, decrease the RER if O_2 utilization remains the same.

This increase in anaerobic threshold may be accompanied by an increase in A-VO_2 difference. This means that the body tissue would be able to extract and utilize more O_2 than prior to the training period. Thus, an increase in O_2 utilization could also help to decrease the RER after training. Although the actual anaerobic threshold was not measured in this study, the decrease in RER from pre- to post-training may have been due to the increased O_2 utilization, since there was a significant increase in O_2 consumption.

Diet

Although there was no attempt to control the diets of runners, their diets were assessed prior to, midway through and at the conclusion of the cross-country season. Average dietary intakes of the athletes for specific food groups are listed in Table 5. Dietary intakes were examined using analysis of variance with repeated measures. In these analyses comparisons were drawn between pre-season and mid-season, pre-season and post-season, and mid-season and post-season.

As can be seen, diets appeared to have remained fairly consistent throughout the cross-country season and no significant ($p > .01$) differences were found over time. The results were not unexpected since many of the runners live in university housing where cafeteria style meals were served, which could have contributed to the consistent dietary intakes. Another reason may have been that in order for the athletes to feel comfortable in practice and competition a conscious effort was made to maintain an adequate and balanced diet. Finally, established

eating patterns may have been developed by this age, keeping day to day intakes similar.

Table 5. Means and standard deviations of dietary intakes of meats, dairy products, fats, eggs and alcohol, comparing pre-season, mid-season and post-season, of the male cross-country runners (n = 43).

Food Group	Time					
	Pre		Mid		Post	
	Mean	SD	Mean	SD	Mean	SD
Meat Intake (oz/wk)	45.2	26.6	49.6	24.8	47.2	24.3
Dairy Intake (cups/wk)	24.3	14.8	26.7	18.2	24.6	16.6
Fat Intake (Tbl/wk)	9.8	7.1	11.6	11.1	10.1	8.1
Egg Intake (#/wk)	4.5	3.9	4.5	4.3	4.0	3.6
Alcohol Intake (drinks/wk)	7.6	7.3	5.7	6.2	6.5	7.4

Quintao and co-workers (1971) suggested that intakes of saturated fats increased serum cholesterol levels. Conversely, another study maintained that a diet low in saturated fat and high in unsaturated and/or polyunsaturated fat lowered total serum cholesterol, but also increased high-density lipid portions (Grundy et al., 1982). For this reason those food items which were thought to alter total cholesterol or HDL cholesterol were statistically analyzed. It was thought by this author that increased intakes of meats (beef, poultry, pork & fish), dairy products (milk, cheese & ice cream), fats (mayonnaise, butter & salad dressing) and eggs may increase TC or decrease HDL cholesterol components. On the other hand, data from numerous studies indicated that weekly alcohol consumption positively correlated to rises in HDL

concentrations (Castelli et al., 1977b; Ernst et al., 1980; Phillips et al., 1981). Thus, alcohol intakes were also recorded at specified times, but as shown in Table 5, there were no significant differences over the course of the season in any dietary component.

Blood Variables

Means and standard deviations for TC, HDL and TC/HDL ratios are reported in Table 6. There were no significant ($p > .01$) changes in any of the parameters from pre- to post-season analysis.

Table 6. Blood variables of the male cross-country runners including means, standard deviations and ranges of TC, HDL, and TC/HDL ratio (n = 43).

Variable	Mean		SD		Range	
	Pre	Post	Pre	Post	Pre	Post
TC (mg%)	163.0	163.5	30.82	29.24	108-249	101-214
HDL (mg%)	53.5	54.4	11.67	10.86	35- 81	37- 84
TC/HDL	3.09	3.06	0.54	0.57	2.1-4.2	2.0-4.4

Total Cholesterol

The TC values of 163.0 and 163.5 mg% found in the present study are compared to other studies in Table 7 along with age, type of training and weekly running mileage of the subjects. It should be noted that the values in the present study fell below the value for total serum cholesterol of 200 mg% in the "average" untrained individual (Cooper, 1982; Grundy, 1982). This "normal" value could, however, vary from individual to individual. Grundy (1982) has suggested an

ideal range for total cholesterol to be between 130 and 190 mg%. As shown in Table 7, Adner and Castelli (1980), Cooper (1982), Brownell et al. (1982) and Hartung et al. (1980) reported higher TC levels than those cited in the present study. Thompson et al. (1982) reported TC values very similar to this study. Finally, Clarkson et al. (1981) reported TC values much lower than the present study.

Table 7. A comparison of various studies including age, type of training, TC, and weekly running mileage.

Name of Researcher	Age	Type of Training*	TC (mg%)	Miles/Wk
Cooper (1982)	30	UT	186.0	--
	40	UT	210.0	--
Adner & Castelli (1980)	40	LDR	194.0	25.0
Brownell et al. (1982)	42	NM	186.0	10.0
Clarkson et al. (1981)	20	LDR	126.4	70.0
Hartung et al. (1980)	44	M	187.0	40.0
Thompson et al. (1982)	32	UM	166.3	95.8
Klapperich (present)	20	LDR	163.0	51.5

* UM = ultra-marathoners, M = marathoners, LDR = long-distance runners, NM = non-marathoners, UT = untrained

From the data presented in Table 7, it could be postulated that there appears to be a relationship between age and TC. Runners in the studies conducted by Adner and Castelli (1980), Brownell et al. (1982) and Hartung and co-workers (1980) were approximately 20 years older than subjects in the present study. Research conducted on the older individual indicated TC values at least 20 to 30 mg% higher than those

for younger runners in the present study. While Thompson and colleagues (1982) reported total cholesterol values similar to those in the present study (average age of the former study being only 10 years older), Clarkson et al. (1981) reported mean TC values of 126.4 mg% for 20 year old long-distance runners. Further support of the age relation to total cholesterol was documented by Cooper (1982). He found that total serum cholesterol increased as age increased (i.e., the average TC for males 30 years and younger was 186 mg%, whereas the average 40 year old maintained a value of 210 mg%). Finally, in a meta analysis of studies Tran, Weltman, Glass and Mood (1983, p. 395) stated "clearly, older males had higher baseline levels of TC than younger males ($r = 0.53$)." Thus, age could have had some effect on the lower levels of total serum cholesterol in this study.

Another possible reason for the low TC levels in the present study may have been its relationship to running distances. As shown in Table 7, the runners who averaged greater than 50 miles per week had lower TC levels than those who ran less than 50 miles per week. Cooper (1982) stated that subjects who were more physically fit (i.e., higher max$\dot{V}O_2$ values and higher weekly running distances) had lower total cholesterol levels than the less fit individual. Since additional running miles result in an increased training time, the meta analysis study of Tran and co-workers in 1983 demonstrated that an increase in the number of hours of exercise was associated with lower serum cholesterol levels. Therefore, miles run per week may have been another reason for low TC levels found in the present study.

Decreases in total cholesterol after 16 weeks of high intensity aerobic training (85% of max$\dot{V}O_2$) was found by Kinsman and associates (1980) in previously sedentary male individuals. Another study on relatively active male subjects found that a 16 week exercise program significantly decreased total cholesterol levels (Dufaux et al., 1982). The results of the aforementioned studies indicated significant decreases in TC levels from pre- to post-training sessions. In the present study, no significant ($p > .01$) differences were found from pre- to post-season testing in TC of highly trained cross-country runners.

Tran and associates (1983) found an inverse correlation between hours of training and TC where increases in training time or distance were associated with decreases in total cholesterol. This supports the findings of Kinsman et al. (1980) and Dufaux et al. (1980) who reported 16 weeks of aerobic exercise decreased TC levels. The results of the present study suggest that the level of fitness prior to a training program may have prevented further decreases in total cholesterol. These subjects began their training season highly fit. As a team they were averaging 42.8 miles per week at the time of the pre-test and had a max$\dot{V}O_2$ of 68.8 $ml \cdot kg \cdot min^{-1}$. Since the TC values of the present study were low to start with (163.0 mg%), the training program did not reduce them further (163.5 mg%). It could be postulated that the cross-country runners reached a threshold in TC prior to their competitive season. Therefore, their training program could not further decrease the total cholesterol levels.

Quintao et al. (1971) and Grundy et al. (1982) also reported that alterations in dietary intake could affect TC levels. It is apparent from the results in Table 5 that the diets of the cross-country runners remained consistent throughout the training season. Therefore, two elements often considered to influence TC, diet and training, apparently did not influence TC in these subjects.

High Density Lipid Cholesterol

It has been reported by many authors that aerobically trained individuals have higher high-density lipid levels than non-trained individuals (Adner & Castelli, 1977; Cooper, 1982; Hartung et al., 1980; Huttunen, 1982; Schnabel & Kinderman, 1982). These authors cited values ranging from 42.5 to 48.7 mg% for HDL levels in non-active individuals compared to a range of 54.2 to 64.7 mg% for trained individuals. In the present study, the mean HDL cholesterol levels for pre- and post-testing of 53.5 and 54.4 mg%, respectively, were higher than all values reported for sedentary subjects. The values in the present study were 10 mg% lower than the HDL reported by Schnabel and Kinderman (1982) and Hartung and associates (1980) for highly trained male runners. However, the HDL values in the present study were similar to those reported by Adner and Castelli (1980), Cooper (1982) and Huttunen (1982) for aerobically trained individuals (54.8, 58.0 & 54.2 mg%, respectively). One could only speculate why these two studies reported higher serum levels. It could be postulated that adipose-tissue lipoprotein lipase activity of their runners was higher than in the subjects of the present study. The higher lipoprotein

lipase activity appears to be related to higher HDL's (Levy & Rifkind, 1980).

Research conducted on the effects of aerobic training on HDL levels seems to be in general agreement. Two authors found significant increases in HDL cholesterol after eight weeks of aerobic training (Farrell & Barboriak, 1980; Gale, 1981). Farrell and Barboriak reported increases in runners from 51.1-57.4 mg%, while Gale reported increases from 38.8-43.5 mg% in male swimmers.

Other researchers have found increases in HDL cholesterol after exercise, though these changes were not significant. Brownell et al. (1980) conducted a study using telephone employees to determine if ten weeks of aerobic activity affected HDL levels in the blood. Along with warm-up and cool-down, the subjects exercised aerobically for only 15 to 20 minutes three times per week. With this low level of activity, the authors found non-significant increases in HDL's. Similarly, Dufaux et al. (1982) found that 16 weeks of jogging training three times per week did not significantly affect HDL cholesterol levels. Finally, Ready and Quinney (1982) cited non-significant increases in HDL cholesterol after nine weeks of bicycle training.

The results of the present study supported the findings of many other researchers. Although HDL concentrations did rise after training, the increases were not significant. One possible explanation might be that the subjects, being highly trained (i.e., mean max$\dot{V}O_2$ = 69.8 ml·kg·min^{-1}) on entering the study, attained peak HDL levels before their training season. It may be postulated that the runners

maintained these HDL levels throughout the season. Cooper (1982), in discussion of high-density cholesterol in the highly trained, suggested that serum HDL's may reach a peak level of concentration after weeks or months of aerobic training.

If the athletes in the present study were, in fact, highly trained upon entering the study and their training or mileage throughout the season was similar to pre-season workouts, then the theory that peak HDL levels exist in training would be supported by these present findings.

It has been previously discussed that dietary intake could affect HDL cholesterol levels. Grundy et al. (1982) and Quintao et al. (1971) reported that decreased intakes of saturated fats and increased intakes of polyunsaturated fats resulted in increased HDL cholesterol levels. Analysis of variance, in the present study, comparing pre-, mid- and post-season intakes of meats, dairy products, fats and eggs revealed no significant ($p > .01$) differences in any of these components. Likewise, no significant changes were attained in alcohol consumption throughout the season. Finally, it should be noted that non-significant increases were found in HDL cholesterol after the training period.

Another factor which may have affected HDL concentrations was body composition. Brownell and associates (1982) and Heiss and co-workers (1980) found that weight reduction had been related to elevation of high-density cholesterol levels. Further support of this was documented by Ready and Quinney (1982) when they reported a significant elevation of HDL cholesterol and a decline in percent fat in men following nine

weeks of bicycle training. The results of the present study showed no significant changes in body weight, percent fat or lean body weight from pre- to post-testing. Likewise, no significant changes were attained in serum levels of HDL's. While these results appear to go hand in hand, there is minimal evidence in the literature that supports these parallel findings.

When HDL values are compared to max$\dot{V}O_2$ values, inconsistencies have been documented. Brownell et al. (1980) found that increases in max$\dot{V}O_2$ did not correlate significantly with changes in plasma HDL concentrations. Huttunen (1982) obtained a weak positive correlation between HDL cholesterol and max$\dot{V}O_2$ values. Finally, while Hartung et al. (1980) found that highly trained athletes had higher HDL's than their sedentary counterparts, Haskell et al. (1980) found that endurance athletes with very high maximal oxygen capacities had higher HDL's than individuals with average-to-low capacities.

The present study, as compared to the literature, reported relatively high mean HDL levels for trained long-distance runners (53.5 & 54.4 mg% for pre- and post-testing, respectively). Furthermore, these high HDL values corresponded to high max$\dot{V}O_2$ values (68.8 & 71.8 ml·kg·min^{-1} for pre- and post-training, respectively). Interestingly, Ready and Quinney (1982), like the present study, found significant increases in max$\dot{V}O_2$ after at least nine weeks of training, but found no significant increases in HDL levels after exercise. Explanation of this could be that those individuals with very high max$\dot{V}O_2$ values were less likely to show a change in serum lipids (Tran

et al., 1983). Ready and Quinney (1982) suggested that the max$\dot{V}O_2$ increases showed a cardiovascular training effect, whereas the non-significant changes in serum lipoproteins could have been associated with other factors.

Total Cholesterol/High-Density Cholesterol Ratio

Numerous authors have found that trained individuals maintained lower TC/HDL ratios than sedentary individuals. Schnabel and Kinderman (1982) reported values of 3.01 and 4.40 for long-distance runners and untrained controls, respectively. Clarkson et al. (1981) documented highly trained males (i.e., running 70 miles/week) with a TC/HDL ratio of 2.32, while their inactive counterparts had a ratio of 3.44. Finally, Hartung and co-workers (1980) reported that well trained males (i.e., running 40 miles/week) had a lower TC/HDL ratio than their sedentary counterparts (2.91 & 4.90, respectfully). In the present study the fractions of TC over HDL for pre- and post-testing (3.09 & 3.06, respectively) supported the literature in that these values were less than those reported for sedentary subjects.

In studies of well trained runners, Schnabel and Kinderman (1982) and Hartung et al. (1980) reported TC/HDL ratios of 3.01 and 2.91, respectively. These values are similar to the TC/HDL values reported in the present study. Gale (1981), on the other hand, reported pre- and post-season TC/HDL ratios of 3.78 and 3.42, respectively, for male intercollegiate swimmers. Finally, Adner and Castelli (1980) reported that marathon runners had a ratio of 3.80. Both TC/HDL ratios from the studies of Gale and Adner and Castelli were found to be slightly

higher than the present study.

Since neither TC nor HDL were significantly altered over the season, there was no significant change in the ratio of TC to HDL. Studies which documented exercise's effects on TC/HDL ratios are few in number. Gale (1981) reported a non-significant decrease in male swimmers from pre- to post-season. Brownell and associates (1982) likewise found a non-significant decrease in TC/HDL ratio in men after a ten week aerobics program. Finally, Ready and Quinney (1982) found no significant change in TC/HDL ratio after nine weeks of bicycle training. In the present study no significant ($p > .01$) differences were reported in the TC/HDL after a season of cross-country running.

Two basic alterations would have taken place if TC/HDL ratios were to have decreased from pre- to post-season testing: (1) TC remained the same and HDL cholesterol increased, or (2) TC decreased and HDL cholesterol remained the same. The present study found no significant change in TC or HDL throughout the training season. The TC/HDL ratio was therefore not significantly different from pre- to post-season testing. Factors which could have affected the TC and HDL have been discussed previously. These included exercise, diet, running mileage and body composition. The present study found no significant changes in any of these parameters. Thus, the fact that TC/HDL ratio was not significantly different after a season of cross-country running was substantiated.

Blood Variables and Coronary Heart Disease

Since HDL serum cholesterol levels appear to be inversely associated to the incidence of coronary heart disease (Hartung et al., 1981), these levels seem to be the best indicators of risk in apparently healthy individuals (Castelli & Levitas, 1977). It has also been verified that HDL levels above 55 mg% represent a low risk for heart disease. General risk can also be established by assessment of TC/HDL ratio. Castelli and Levitas (1977) reported that a male with fractions below 3.43 will have half the risk of developing coronary heart disease than will a male with a ratio above this.

The data from the present study showed positive signs in both apsects. Pre- and post-season HDL levels were slightly below 55 mg%, at 53.5 and 54.4%. Similarly, the values for TC/HDL ratio were 3.09 and 3.06 for pre- and post-testing, respectively, corresponding to that of males with half the risk of developing heart disease. After analyzing the descriptive characteristics, physiological parameters and blood variables, the author could not guarantee these subjects free of developing coronary heart disease, but maintained that they were, in fact, at very low risk.

CHAPTER V

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Summary

This study was designed to examine the effects training would have on max$\dot{V}O_2$, body composition and the concentrations of blood lipids. The participants were 43 intercollegiate male cross-country runners who participated in a training program as specified by their coach, Dr. Phillip Esten.

Body composition, maximum oxygen uptake and blood samples were measured prior to the cross-country season and after the last competitive meet. Runners who did not qualify for the national championships (n = 29) trained and competed for nine weeks. Those who qualified for either the NAIA (n = 7) or the NCAA (n = 7) National Championships trained for twelve weeks. Body composition measurements included weight, LBW and percent fat. In addition, physiological parameters of max$\dot{V}O_2$, maxV_E, MHR, TMT and RER and blood concentrations of TC, HDL and TC/HDL ratio were determined. Dietary intakes of runners were obtained at three different intervals: pre-, mid- and post-season training.

Standard descriptive methods were employed to describe the physical characteristics of the subjects and their diets. Means and standard deviations were tabulated for each variable. A dependent t-test was used to determine if there was any significant change in the aforementioned variables from pre- to post-training measures.

A one way analysis of variance with repeated measures was used to compare the dietary intakes from pre- to post-season, to determine if there were any significant differences. All variables were tested at the 0.01 level of confidence.

Conclusions

Within the limitations of this study, several conclusions were drawn. Body weight, percent body fat and lean body weight did not significantly change from pre- to post-season. This lack of significant alteration in body composition was primarily due to the fact that the subjects were very lean and highly trained upon entrance to the training program.

After the season of cross-country training and competition, max$\dot{V}O_2$, as expressed in $ml \cdot kg \cdot min^{-1}$ and $L \cdot min^{-1}$ increased significantly. These post-season values for max$\dot{V}O_2$ were comparable to values attained by elite endurance athletes. When a significant increase in max$\dot{V}O_2$ is noted, this specifies a cardiovascular training effect. So in the present study the training program for these runners did, in fact, help to bring about an increase in cardiorespiratory fitness by the small but significant increase in max$\dot{V}O_2$. It appears that in the highly trained individuals, max$\dot{V}O_2$ may be increased, but not as dramatically as for untrained individuals who engage in a training program. Perhaps there is a genetic limit where little or no change may occur after a period of continuous training.

Although max$\dot{V}O_2$ showed a significant increase throughout the season, maximum heart rate, treadmill time and maximum ventilation

volumes did not significantly change. However, respiration exchange ratio did significantly decrease from pre- to post-testing. This decrease could have been attributed to a decrease in CO_2 production, an increase in O_2 utilization or a combination of both. Since there was a significant increase in O_2 consumption in the present study, the decrease in RER may have been primarily due to an increased O_2 utilization.

Dietary intakes of meats, fats, dairy products, eggs and alcohol did not significantly change from pre- to post-season analyses. This was probably due to some of the team eating their meals in the school cafeterias where the menu remained relatively consistent from week to week. For those who lived outside university housing, a conscious effort could have been made to keep the diet consistent in order to feel comfortable at practice as well as in competition. Whatever the reason, the data in the present study showed no significant difference over the course of the season in any dietary component.

There was no significant change in TC after the cross-country season. The lower total cholesterol levels in this study may have been due to the age, running mileage or possibly the diets of the subjects. One researcher documented increases in TC as age increased. Since the subjects in the present study were young ($\bar{x}$ = 19.8 years), it was not possible to determine if age affected the low serum cholesterol values. The present researcher found low TC levels related to higher running distances in the review of literature. The literature suggested that runners who averaged more than 50 miles per week had

lower TC levels than did runners who averaged less than 50 miles per week. The subjects in the present study averaged more than 50 miles per week and had lower TC, thus supporting this concept. Further, diets low in saturated fat intakes were associated with lower serum cholesterol levels. The low levels of total cholesterol which appeared in this study may have also been related to the lower percent fat in the runners. Finally, the high max$\dot{V}O_2$ values may also have been related to the low TC levels.

It was reported in the literature that the lower the initial level of TC before an exercise program, the less effective the physical training was in altering the TC concentrations. In the present study, there was no significant change in TC after a season of training, however the team was highly trained (i.e., running 42.8 miles per week & a max$\dot{V}O_2$ of 68.9 $ml \cdot kg \cdot min^{-1}$) at the time of the pre-test. Because the runners were highly fit on entering the training program, it appears that they may have reached a threshold in TC prior to their competitive season. Furthermore, no significant changes appeared in dietary intakes throughout the training season. Therefore, two elements often considered to influence TC, diet and training, apparently did not influence TC in these subjects. Changes in body composition have also been associated with altering TC concentrations. However, in this study, there was no significant difference in percent fat or lean body weight from pre- to post-season training. On this basis, it was not possible to ascertain if the changes in total cholesterol were related to alterations in body composition.

Over the course of the season, no significant changes were attained in HDL concentrations. Although this is the case, the pre- and post-season values were higher than all values found for sedantary individuals in the literature and comparable to other research on the highly trained individuals. High density lipoprotein changes caused by diet may have had equal but opposite effects as HDL changes caused by exercise. However, diet was not significantly altered, therefore, this is only speculative. There may be a peak level of HDL cholesterol which the highly trained can achieve or maintain. The results of this study support this concept in light of the fact that the high HDL levels did not significantly change from pre- to post-season testing. Changes in body composition have also been shown to affect HDL levels. The results of the present study, however, showed no significant changes in percent body fat or lean body weight after a season of cross-country running.

Since TC and HDL did not significantly change after a season of running training, there was no significant alteration in the TC/HDL ratio. Overall, the season of cross-country running did not alter lipoprotein concentrations. However, this training program did elicit a cardiorespiratory conditioning response where max$\dot{V}O_2$ increased from pre- to post-season testing. These results appear to demonstrate that the highly trained athlete can, in fact, increase cardiovascular fitness (though not dramatically), but that TC and HDL concentrations may reach a peak level at some other time in their training regime.

General risk of developing CHD can be ascertained by the level of HDL and/or by the ratio of TC to HDL that one possesses. The runners in

the present study obtained high HDL levels and low TC/HDL ratios, thus their chances of developing CHD appear to be very low.

Recommendations for Further Study

1. If economically possible, blood assays could be taken at two week intervals throughout the training season. This would enable the researcher to determine if fluctuations occur in lipoprotein concentrations at different times throughout the training season.

2. The effects of detraining on blood concentrations in the highly trained should be examined. Limited research has been conducted on this topic. Since exercise did not affect lipoproteins in this study, it would be interesting to see if detraining would elicit changes in lipoprotein concentrations.

3. A similar study could be conducted where correlation coefficients were used to compare relationships between variables of body composition, physiological parameters and blood constituents. This would enable the researcher to determine if relationships do exist between these variables.

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APPENDIX A

INFORMED CONSENT

INFORMED CONSENT

EFFECTS OF A SEASON'S CROSS COUNTRY TRAINING IN MALES

I, _____, volunteer to participate in the maximal treadmill, underwater weighing and blood tests to compare the effects of a season of cross-country training in males.

I understand the blood test will involve having approximately 10 ml of blood taken from a vein in my arm. The blood will be drawn by a trained Technician. Although there is a potential hazard of infection and other complications from a venipuncture no such complications have occurred with this procedure in the past.

I understand the maximal aerobic test consists of a run to voluntary exhaustion on a motor-driven treadmill. The speed and the incline of the treadmill will be gradually increased throughout the run. During this test heart rates will be monitored on a ECG and exhaled air will be collected. This test requires a maximal effort, however, I can terminate the run anytime I wish. As with any exercise, there exists the possibility of adverse changes (i.e., dizziness, etc.) occurring during the test. If any abnormal observations are noted the test will be immediately terminated. In addition, you will feel tired at the end of the exercise.

The body composition test will consist of having my residual lung volume measured on land and having my body weight determined while submerged in water. In working in a water environment, there are risks involved such as infection, accidents or possible drowning. However, there has never been an accident or report of infection as a result of this procedure at the UW-L Human Performance Laboratory.

To my knowledge I am not infected with any disease or have any limiting physical conditions or disabilities, especially with respect to my heart, that would preclude such a strenuous exercise.

I have read the foregoing and I understand it, and any questions which may have occurred to me have been fully answered to my satisfaction. The potential risks have been explained to me and I fully understand their implications. I hereby acknowledge that no representations, warranties, guarantees, or assurances of any kind pertaining to the procedures have been made to me by the University of Wisconsin-La Crosse, the officers, administrators, employees or by anyone acting on behalf of any of them.

Signed:_____ Date:_____

Witness:_____ Date:_____

APPENDIX B

BLOOD CONSENT FORM

NAME: _____ DATE: _____

SEX: _____ TIME: _____

Have you consumed any food within the last 12 hours? Yes___ No___

Have you consumed any alcohol within the last 24 hours? Yes___ No___

By my signature below I certify that the above statements are true.

Signed: _____

Witness: _____

APPENDIX C

WEEKLY RUNNING MILEAGE

Team Average Miles Per Week

1983	Average Miles Per Week
* June 1-August 28 (Summer)	42.80
August 29-September 4	45.25
September 5-September 11	57.75
September 12-September 18	59.97
September 19-September 25	59.97
September 26-October 2	57.28
October 3-October 9	55.09
October 10-October 16	52.81
October 17-October 23	50.68
October 24-October 30	46.56
** October 31-November 6	47.83
November 7-November 13	42.88
November 14-November 20	42.20
TOTAL SEASON	51.53

* June 1-October 31 N = 43

** October 31-November 14 N = 14

APPENDIX D

TREADMILL DATA FORM

NAME: _____ Date: /83 Time: _____ Temp: _____ Pbar _____

_____lbs _____kg _____%fat _____LBW _____Height _____age _____birthdate _____miles/week

	WORKLOAD	V_E	ml O_2	ml·kg	V_{CO2}	RER	$FeCO_2$	FeO_2	HR	RPE	ml.LBW
(1)	7 mph 0										
(2)	7 mph 0										
(3)	7 mph 2½										
(4)	7 mph 2½										
(5)	7 mph 5										
(6)	7 mph 5										
(7)	7 mph 7½										
(8)	7 mph 7½										
(9)	7 mph 10										
(10)	7 mph 10										
(11)	7½ mph 10										
(12)	7½ mph 10										
(13)	8 mph 10										
(14)	8 mph 10										
(15)	8½ mph 10										
(16)	8½ mph 10										
(17)	9 mph 10										
(18)	9 mph 10										
(19)	9½ mph 10										
(20)	9½ mph 10										
		O_2			CO_2						

APPENDIX E

BODY COMPOSITION

DATA FORM

La Crosse Exercise Program

RESIDUAL VOLUME DATA SHEET

Part 2

UNDERWATER WEIGHING

IMMERSION TANK TEMP _____ °C. DENSITY OF WATER (D_W) 0._____.
(from conversion chart)

RESIDUAL VOLUME (RV) = V.C. X CONVERSION FACTOR = _____ L.
(from conversion chart)

MASS IN AIR (M_A)_____kg.

MASS IN WATER (M_X)_____kg.

MASS OF WEIGHING APPARATUS (M_Y)_____kg. (in water)

MASS OF WATER (M_W) = $M_X - M_Y$ = _____KG.

AIR IN GASTRO-INTESTINAL TRACT = 100 ml. = 0.1 L.

$$\text{RESIDUAL VOLUME} = \frac{VO_2\ (EN_2 - IN_2)}{(AN_2 - FN_2)} - DS = \_\_\_\_\_ L.$$

$$\text{BODY DENSITY } (D_B) = \frac{M_A}{\left(\frac{M_A - M_W}{D_W}\right) - RV - 0.1\ L.}$$

$$\% \text{ FAT} = \left[\frac{4.570}{D_B} - 4.142\right] \times 100 = \_\_\_\_\_ \%$$

Trial #1_____
2_____
3_____
4_____
5_____
6_____
7_____
8_____
9_____
10_____

APPENDIX F

DIETARY ANALYSIS FORM

Name:_____ Date:_____

Sex:_____ Time:_____

Please indicate the amount of units of the following foods you consumed last week.

Meats (one unit = 4 oz. or one serving)

beef_____
poultry_____
pork_____
veal_____
fish_____
cold cuts_____

Dairy Products (one unit = one cup; one slice = one oz.)

whole milk_____
low fat (1% 2% ½%)_____
cream (½ & ½)_____
ice cream_____
cheese_____
yogurt_____
cottage cheese_____

Fats (one unit = one tablespoon)

mayonnaise_____
butter/marg._____
salad dressing_____
peanut butter_____
sour cream_____

Alcohol (one unit = one beer; one mixed drink; one glass of wine)

beer_____
mixed drink_____
wine_____

Eggs (one unit = one egg) _____

APPENDIX G

PRE & POST-TRAINING

DATA FORM

Subject	LBW (1) Kgs	LBW (2) Kgs	TC (1) mg%	TC (2) mg%	HDL (1) mg%	HDL (2) mg %	TC/HDL Ratio	TC/HDL Ratio	$\dot{V}_E$ (1) L·min	$\dot{V}_E$ (2) L·min	RER (1)	RER (2)	Height cm.	Yrs of
1	59.7	61.8	235	214	69	71	3.4	3.4	170.4	163.4	1.14	1.08	172.1	21
2	61.2	59.7	150	156	45	42	3.3	3.7	118.0	118.2	1.18	1.12	173.4	20
3	58.2	58.0	150	207	52	50	2.9	4.1	105.6	99.7	1.08	1.06	181.0	19
4	57.5	58.4	145	161	44	53	3.3	3.0	150.3	140.8	1.13	1.04	179.1	19
5		58.0	145	144	64	48	2.3	3.0	157.9	159.0	1.05	1.09	175.3	19
6	58.0	59.3	163	187	40	43	4.1	4.5	157.8	135.4	1.24	1.11	163.8	21
7	57.2	58.2	171	157	66	58	2.6	2.7	137.5	124.3	1.07	1.06	165.7	20
8	57.0	58.5	208	204	64	60	3.3	4.4	127.8	134.4	1.05	1.03	175.3	20
9	56.0	58.0	189	203	56	51	3.4	4.0	136.0	130.1	1.13	1.11	171.5	19
10	55.0	55.8	184	186	70	72	2.6	2.6	136.1	140.6	1.06	0.98	168.7	21
11	55.8	56.9	131	176	52	57	2.5	3.0	150.4	163.2	1.12	1.04	167.6	20
12	65.9	65.9	146	148	60	55	2.4	2.7	145.5	147.9	1.15	1.08	178.4	22
13	54.7	52.0	148	146	57	55	2.6	2.6	142.6	126.2	1.07	1.00	173.4	21
14	58.4	58.4	176	186	43	54	4.0	3.4	135.4	129.5	1.09	1.06	173.4	20
15	50.1	52.6	157	155	44	52	3.6	2.9	136.0	125.9	1.09	1.07	162.6	19
16	49.5	51.1	160	143	47	45	3.4	3.2	129.8	132.7	1.07	1.05	178.4	18
17	53.5	51.8	155	151	58	58	2.7	2.6	131.0	132.8	1.08	1.09	172.7	19
18	68.8	69.9	120	117	41	37	2.9	3.1	158.0	156.4	1.16	1.09	183.5	20
19	55.6	56.5	159	178	40	45	4.0	4.4	155.6	141.6	1.10	1.12	173.4	22
20	68.4	66.8	180	178	60	70	3.0	2.5	154.3	158.4	1.06	1.06	182.2	18
21	55.8	54.7	148	208	64	62	3.0	3.3	129.2	133.0	1.10	1.08	173.4	18
22	49.5	50.7	176	163	71	81	2.5	2.0	147.0	122.2	1.04	1.13	160.0	23
23	51.9	55.5	152	156	44	52	3.5	3.0	124.0	107.0	1.08	1.13	170.8	18
24	61.9	61.5	167	144	59	56	2.8	2.6	140.4	149.2	1.11	1.12	181.6	20
25	70.7	71.5	130	152	49	51	2.7	3.0	175.1	169.0	1.13	1.14	188.6	19
26	67.7	66.0	168	129	81	66	2.1	2.0	154.2	166.3	1.02	1.10	189.2	22
27	56.4	57.3	214	185	81	84	2.7	2.2	146.9	173.9	1.06	1.17	170.8	24
28	51.0	51.9	173	173	46	53	3.2	3.3	157.1	154.1	1.17	1.07	170.2	20
29	56.3	55.8	140	147	60	54	2.3	2.7	157.1	168.1	1.11	1.13	170.8	2;
30	54.2	55.4	249	239	63	65	3.9	3.7	140.7	147.8	1.05	1.07	167.6	21
31	53.0	54.0	140	135	46	41	3.0	3.3	143.7	149.2	1.08	1.02	171.5	19
32	56.5	55.7	140	158	53	65	2.6	2.4	150.4	144.9	1.08	1.03	179.1	18
33	60.3	60.0	116	101	44	41	2.6	2.5	147.1	139.3	1.19	1.06	178.4	19
34	59.3	60.0	149	158	47	43	3.2	3.7	177.0	166.0	1.13	1.09	170.8	19
35	61.3	61.0	130	146	38	44	3.4	3.3	156.8	162.7	1.07	1.09	175.9	19
36	58.1	58.1	176	179	48	45	3.7	3.5	158.5	158.8	1.09	1.05	171.5	18
37	60.8	60.1	147	146	35	51	4.2	2.9	145.7	155.5	1.05	1.11	170.2	19
38	64.5	64.6	201	161	55	49	3.7	3.3	163.3	162.9	1.09	1.16	174.0	21
39	66.1	65.9	103	134	35	41	3.1	3.3	137.7	137.4	1.07	1.10	178.4	19
30	61.4	61.3	164	146	65	63	2.5	2.3	139.7	160.9	1.11	1.11	177.8	19
41	69.7	69.2	123	101	41	41	3.0	2.5	129.3	161.4	1.06	1.08	177.2	22
42	55.8	57.6	177	171	46	54	3.6	3.2	140.6	115.4	1.12	1.05	172.1	21
43	59.1	58.7	210	180	59	59	3.6	3.2	141.7	145.9	1.11	1.03	177.8	18
MEAN	58.7	58.9	163.0	163.5	53.5	54.4	3.09	3.06	144.6	144.1	1.09	1.07	174.2	19.8
SD	5.49	5.05	30.82	29.24	11.67	10.86	0.54	0.57	14.71	17.66	.045	.041	5.94	1.49
RANGE	49.5-70.7	50.7-71.5	108-249	101-214	35-81	37-84	2.1-4.2	2.0-4.4	105.6-175.1	99.7-173.9	1.02-1.24	0.98-1.24	160.0-189.2	18-24

Subject	MaxVO2 (1) L·min-1	MaxVO2 (2) L·min-1	Max VO2 (1) ml·kg·min-	Max VO2 (2) ml·kg·min-	MHR (1) bpm	MHR (2) bpm	TMT (1) min	TMT (2) min	Weight (1) kgs	Weight (2) kgs	% Fat (1)	% Fat (2)
1	4.9	5.3	65.7	75.7	192	197	15.0	18.0	68.8	67.5	13.3	8.4
2	4.9	4.8	73.4	71.9	192	194	18.5	17.0	66.3	64.4	7.6	7.3
3	3.6	3.9	54.2	57.2	186	193	8.0	8.0	65.2	66.6	10.7	13.0
4	4.5	4.6	65.4	70.5	185	188	13.0	14.0	65.1	64.3	11.7	9.2
5	5.2	5.2	76.6	76.7	189	188	16.0	16.75	67.8	54.9		10.8
6	4.2	4.2	65.1	63.5	215	210	14.75	11.75	65.2	65.8	11.3	9.9
7	4.5	4.2	72.1	68.1	201	206	17.0	15.25	61.7	61.6	7.3	5.5
8	4.3	4.5	66.3	72.0	191	198	15.0	16.5	64.7	64.5	11.9	9.4
9	4.5	4.6	74.1	72.7	200	200	16.25	18.0	59.2	61.3	5.3	5.4
10	4.1	4.8	64.4	72.2	180	187	12.0	14.0	63.4	61.2	13.3	8.8
11	4.0	5.2	63.3	74.4	167	171	12.0	14.0	63.4	63.4	8.3	10.5
12	4.7	4.1	66.6	72.4	200	202	15.0	15.0	68.6	69.9	4.0	5.6
13	4.3	4.7	74.4	71.7	208	198	16.0	14.0	56.3	56.4	2.7	7.8
14	4.6	4.7	66.8	68.7	182	181	14.0	15.0	68.0	68.1	14.1	14.2
15	4.7	4.4	74.6	72.0	188	184	19.0	18.25	57.3	56.5	12.5	6.9
16	4.4	4.2	72.6	73.1	197	199	19.0	16.0	55.2	57.3	10.3	10.7
17	4.0	4.1	67.7	69.3	197	200	14.25	15.5	59.4	58.3	10.0	11.2
18	5.1	5.2	69.9	68.9	183	184	17.0	16.0	71.5	74.4	3.7	6.1
19	4.2	4.3	71.4	73.3	202	196	16.0	16.5	58.5	59.1	5.0	4.3
20	5.1	5.2	70.7	71.1	194	190	16.0	16.25	72.2	72.3	5.2	7.6
21	4.5	4.9	74.2	78.7	193	194	18.0	17.5	58.9	60.7	5.2	9.8
22	4.3	3.8	74.5	63.0	189	184	17.0	14.0	60.2	60.7	17.8	16.5
23	4.2	4.3	67.3	67.5	193	194	14.0	13.0	60.3	62.8	14.1	11.6
24	4.8	5.0	71.1	73.1	201	199	17.0	17.25	66.6	65.7	7.0	7.9
25	5.4	5.4	69.9	69.1	186	200	17.5	18.0	76.7	77.7	7.8	8.0
26	5.0	4.8	69.8	69.8	167	193	14.0	16.0	71.7	69.5	5.6	6.6
27	4.7	4.9	75.3	76.3	190	184	16.0	17.5	61.8	62.5	8.8	8.4
28	4.2	4.6	73.3	79.0	187	204	17.0	17.0	54.7	56.1	6.7	7.6
29	4.4	4.5	66.8	67.7	184	184	13.0	14.0	63.9	65.8	16.7	15.2
30	4.3	4.5	70.6	74.9	188	181	16.0	16.0	59.5	58.9	8.9	5.9
31	4.5	4.6	76.4	76.0	184	186	16.0	17.0	59.1	59.2	10.2	8.7
32	4.7	5.0	72.5	77.6	192	192	15.0	18.0	64.7	63.5	12.7	12.3
33	4.7	5.0	66.8	71.8	187	184	16.0	15.0	69.2	69.3	12.9	13.1
34	4.7	4.7	73.0	72.9	199	200	16.0	15.0	63.6	62.8	6.9	5.2
35	4.6	4.9	70.9	72.0	204	212	16.0	17.0	66.3	68.3	7.0	10.7
36	4.9	5.0	78.2	80.3	201	200	16.0	16.25	62.5	62.5	7.2	7.0
37	5.0	5.1	75.0	75.3	194	192	17.0	18.0	65.1	64.9	6.6	7.4
38	5.1	5.3	73.9	77.1	191	192	20.0	20.5	62.4	66.8	4.2	3.3
39	4.7	4.8	64.2	65.7	184	180	12.0	14.0	71.3	71.6	7.2	8.0
40	4.0	4.7	54.2	67.5	206	215	14.0	15.0	66.7	68.5	8.0	10.5
41	4.9	5.4	64.1	73.9	180	185	14.0	17.0	73.6	73.4	5.4	5.6
42	4.6	4.4	75.2	72.3	175	190	18.0	15.0	55.1	59.7	5.0	3.4
43	4.3	5.0	64.7	72.9	199	205	14.0	15.0	65.8	66.0	9.5	11.1
MEAN	4.4	4.7	69.8	71.8	192.7	194.0	15.5	15.7	64.3	64.5	8.7	8.8
SD	.37	.45	5.03	4.99	8.68	8.62	2.32	2.08	5.15	5.09	3.46	3.05
RANGE	3.6-5.4	3.8-5.5	54.2-78.2	57.2-80.3	167-215	171-215	8.0-20.0	8.0-20.5	55.2 76.7	56.1 77.7	2.7-17.0	3.3-16.5