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THE EFFECT OF A SIX MONTH WALKING PROGRAM ON THE BODY COMPOSITION AND FAT DISTRIBUTION OF MILDLY HYPERCHOLESTEROLEMIC WOMEN POST MENOPAUSE

By

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THE EFFECT OF A SIX MONTH WALKING PROGRAM ON THE BODY COMPOSITION AND FAT DISTRIBUTION OF MILDLY HYPERCHOLESTEROLEMIC WOMEN POST MENOPAUSE

BY

DARREN G. BRERETON

A Thesis submitted to the Faculty of Graduate Studies of the University of Manitoba in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

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Absract

Cardiovascular disease is the leading cause of death in Canada for both sexes, accounting for 41 percent of all deaths. Women have an increased risk of developing coronary heart disease (CHD) following menopause due to the changes that occur in their serum lipids, body fat levels, and body fat distribution. As a result of these changes cardiovascular disease becomes the primary cause of death in women by their sixth decade. Regular physical exercise has been found to be an inexpensive intervention that has preventative effects on several risk factors for CHD including blood pressure, body composition, and serum lipid profile. Despite the increased risk of developing CHD in women post menopause, little research has been done on this group studying the effect of aerobic exercise on body composition and body fat distribution. As a result it is unknown whether aerobic exercise, at any intensity or duration, can positively alter the body composition and serum lipid profile of this group thereby reducing their risk of developing CHD.

The purpose of the study was to examine the effects of a six month brisk walking program on the total body fat, muscle mass, body fat distribution and fitness level of women post menopause. The subjects were women who were 55 years of age or older, nonsmokers, sedentary or minimally active and not on medications to control serum lipid levels. Their serum cholesterol levels had to be between 6.0 to 8.0 mmol/L. Women with triglyceride levels greater than 4.2 mmol/L were excluded as well as women receiving estrogen replacement therapy. Forty women met the screening criteria and were randomly assigned to either a walking (n=24) or control group (n=16). Participants in the walking group took part in a six month program of walking which progressed to five times per week, for 54.3 minutes, at an exercise intensity of 54% heart rate reserve. The controls remained sedentary or minimally active. Body composition, percent body fat, body fat distribution, maximal oxygen uptake and caloric intake and dietary makeup were assessed every 12 weeks. Data was analyzed on a Macintosh computer using the Data Desk program. Paired t-tests were used to analyze changes within the groups while unpaired t-tests of the change values were used to determine between group changes from the 0 to 24 week period. Correlation coefficients were used to test for covariance between independent variables while simple regression was used to test for significant relationships between independent and dependent (change) variables. If significant relationships were found to exist ANCOVA was used to control for the influence of the covariate so

that the true effect of the walking program on the dependent variable could be determined. The six month walking program was found to be well tolerated by the subjects and was of sufficient intensity to improve the fitness levels. The average weekly energy expenditure was sufficient to cause a significant decrease in weight and body mass index in the walkers and to cause fat to be lost equally from the upper and lower body regions. The walking program was unsuccessful in decreasing the walkers' percent body fat or increasing their muscle mass.

Acknowledgements

I would like to thank Dr. Elizabeth Ready and Dr. Don Drinkwater for all the time and support they gave during this project. I would also like to thank the Statistical Advisory Service of the University of Manitoba for the help and expertise they gave me in analyzing and interpreting my data.

Finally I would like to thank my wife Claire for all her patience and support during this time.

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CHAPTER ONE:

INTRODUCTION

Cardiovascular disease is the leading cause of death in Canada for both sexes, accounting for 41 percent of all deaths (Statistics Canada, 1990). In 1988 it was estimated that 16 billion dollars were spent on coronary health care in Canada alone, and about 25 percent of all disability pensions paid through the Canada Pension Plan in 1986 were for cardiovascular disease (Health & Welfare Canada, 1988). Women have an increased risk of developing coronary heart disease (CHD) following menopause and as a result, cardiovascular disease becomes the primary cause of death by their sixth decade (Statistics Canada, 1990). This increased risk is due to the changes that occur in serum lipids, body fat levels, and body fat distribution as a result of, or in conjunction with, menopause.

Menopause is associated with the development of a more adverse plasma lipoprotein profile. Decreased ovarian estrogen production, is associated with an increase in plasma low density lipoprotein cholesterol (LDL-C) levels and a decrease (Mathews et al., 1989) or no change (Krauss, 1987) in plasma high density lipoprotein cholesterol (HDL-C) levels. Elevated total cholesterol (hypercholesterolemia) and LDL-C as well as low levels of HDL-C are risk factors for CHD (Goldburt et al., 1979; Gordon et al., 1977).

As a result of, or in conjunction with, aging (Bray, 1989; Wing et al., 1991) or menopause, (Vague, 1956) women tend to gain weight and preferentially lay fat down in the upper body region.

These changes in body composition are associated with the development of an adverse lipoprotein profile. High levels of body fatness are associated with elevated levels of total cholesterol (hypercholesterolemia) and triglycerides (Després et al., 1988). An android or upper body fat distribution pattern, reflected by a waist to hip ratio (WHR) greater than 0.80, increases an individual's propensity to develop metabolic aberrations such as glucose intolerance, hyperinsulinemia and hyperlipidemia (Peiris et al., 1989). The metabolic aberrations are in turn associated with an adverse lipoprotein pattern characterized by elevated levels of plasma very low density lipoproteins (VLDL-C) (Olesky et al., 1974), triglycerides (Peiris et al., 1989; Seidell et al., 1989; Wing et al., 1991), total cholesterol (Wing et al., 1991),and LDL-C as well as an elevated TC/HDL-C ratio and decreased levels of HDL-C (Olefsky et al., 1974; Peiris et al., 1989; Seidell et al., 1989).

Regular physical activity is an inexpensive intervention that has preventative effects on several risk factors for CHD including blood pressure, body composition and serum lipid profile (Leon et al., 1987; Powell et al., 1987). In studies of men, aerobic exercise caused significant loss of total body fat (Wood et al., 1988; Tran & Weltman, 1985) and preferentially from the abdominal region, thereby reducing the WHR (Tremblay et al., 1988; Wood et al., 1988). These positive changes in men's body composition are in turn associated with a decrease in plasma total cholesterol and triglyceride levels (Lokey & Tran, 1988) and an increase in HDL-C levels (Wood et al., 1988) thereby, decreasing the risk of developing CHD.

Studies looking at the effect of aerobic exercise on body composition in premenopausal women have been contradictory and inconclusive. Some found that exercise did not cause women to lose significant amounts of fat (Després et al., 1984; Hill et al., 1987; Phinney et al., 1988; Thomas et al., 1986; Warwick & Garrow, 1981; Van Dale et al., 1987), while others reported that exercisers lost significant fat (Andersson et al., 1991; Baylor & Keesey, 1991;

Després et al., 1991) or preferentially lost fat from the abdominal region, thereby lowering the WHR (Després et al., 1991). Whether or not exercise results in fat loss seems to be related to the total energy expended. In studies where exercise was found to positively alter body composition, subjects exercised at intensities between 70 and 80% of VO_2 max (Andersson et al., 1991; Lewis et al., 1976; Schaberg-Lorei et al., 1990; Tremblay et al., 1991) or for a duration lasting 6 to 18 months (Després et al., 1991; Foss et al., 1980; Gwinup, 1975). These results suggest that a certain amount of energy must be expended in order for positive changes to occur in the body composition of premenopausal women. It has been suggested that energy expenditure must exceed 2000 kilocalories a week (kcal/wk) in order for positive changes to occur (Cauley et al., 1982; Cook et al., 1986).

Initial levels of body fatness and body fat distribution influence the effect aerobic exercise has on body composition changes. Women with high initial body fat levels have been found to lose significantly more weight and body fat than women with lower initial values (Andersson et al., 1991; Ballor & Keesey, 1991), while WHR has been found to decrease significantly more in women with upper body fat distribution than in women with a lower body fat distribution (Casimirri et al., 1989; Wadden et al., 1988).

Positive alterations in the body composition of premenopausal women are associated with favorable changes in serum lipid profile. Decreases in total body fat and WHR have been correlated to decreases in plasma total cholesterol and LDL-C (Després et al., 1991; Lokey & Tran, 1989) and in the TC/HDL-C ratio (Lokey & Tran, 1989). A loss of total or upper body fat in premenopausal women may therefore be associated with a decreased risk of developing CHD.

Despite the increased risk of developing CHD in women after menopause, little research has been done on this group looking at the effect of aerobic exercise on body composition and body fat

distribution changes. Rebuffé-Scrive et al., (1986) found that decreased ovarian estrogen production, which occurs as a result of, or in conjunction with menopause, decreased the lipolytic rate of the abdominal adipocytes and the activity of the LPL (lipoprotein lipase) enzyme in the gluteal/femoral region. As a result they concluded that fat loss would occur equally from the upper and lower body regions in women after menopause. This is contrary to the findings with men and premenopausal women that fat loss occurs preferentially from the abdominal region. Due to these contradictory findings and the lack of research it is unknown whether exercise, at any intensity or duration, can positively alter body composition and serum lipid profile. It is also unknown if any other factors, such as initial body fat levels or body fat distribution will influence these relationships.

1) Statement of the Problem

The purpose of this study was to examine the effects of a six month brisk walking program on: 1) total body fat; 2) body fat distribution; 3) muscle mass, and; 4) the relationship between initial fatness/body fat distribution and fat loss, in postmenopausal, mildly hypercholesterolemic women.

2) Hypotheses

The hypotheses were:

1) A six month program of brisk walking will lead to a significant loss in total body fat which will relate directly to the amount of energy expended during the study.

2) Those with the highest initial levels of body fatness will lose the most body fat and have the greatest change in body fat distribution.

3) Fat will be lost equally from the upper and lower body region as a result of a six month brisk walking program.

4) There will be a significant increase in muscle mass as a result of the six month brisk walking program.

5) The reduction in body fat, change in body fat distribution and increase in muscle mass will relate directly to the subject's increase in cardiovascular fitness levels.

3) Assumptions

Due to the fact that it was a community based study assumptions were made about the subjects' ability to accurately record and monitor physical activity and dietary intake. It was assumed that participants accurately completed their activity log books and three day food frequency records, and were able to accurately monitor their own pulse rates while exercising.

Assumptions also had to be made about the measurement techniques used in the study. With skinfold caliper measurements it was assumed that: a) the skinfold caliper was a valid and reliable instrument for measuring subcutaneous fat, b) that skinfold measurements taken at the body sites in this study were indicative of the subcutaneous fat stores in the limbs and trunks, c) that the sum of all skinfolds represented a valid indication of body fatness. It was also assumed that girth ratios were valid methods by which to assess regional adipose tissue distribution.

It was assumed that the formula developed by Martin, Drinkwater, Spenst, and Clarys, (1990) during their Brussels cadaver study, was an accurate and valid formula for determining the muscle mass of the postmenopausal women in the study.

The final assumption made was that participants kept additional endurance activity to a minimum and did not drastically alter their diet throughout the duration of the study.

4) Delimitations

The sample group was composed of 40 non smoking, clinically healthy females (no cardiovascular, pulmonary, or metabolic diseases) who were 55 years of age or older and were one year post menopause. All subjects consumed less than one ounce of alcohol per day, were not on medications that affected serum lipid levels, and were not receiving estrogen replacement therapy. Participants did not have triglyceride levels greater than 4.2 mmol/L and had serum total cholesterol levels between 6.0 to 8.0 mmol/L. All women were from Winnipeg or the immediate surrounding area.

5) Limitations

Whenever possible the same tester was used over the three test periods. One of the limitations of the study was that the body composition measurements (skinfolds and girths) at the zero, three, and six month measurement dates were not always taken by the same tester on each individual. This was impossible to arrange due to the length of the study.

The fact that the study was a community based study was the second limitation. This necessitated that energy intake be

calculated from self reported, three day food frequency records which were completed every 3 months, a method which is less accurate than more frequent, detailed records. Similarly, participants were only supervised for one out of the five weekly exercise sessions and were required to complete the other four on their own. This method is once again less accurate than if the participants were monitored during each walking session.

A third limitation of the study was that it only had a power level of 0.60 which is less than the accepted power level of 0.80 used in other studies. This limited the study's ability to determine if significant changes occurred in the subjects' body composition and body fat distribution as a result of the walking program. The decreased power level was due to the fact that the study was part of a larger study done by Professor A. E. Ready. As a result the sample size of the present study was dependent upon the number of subjects used in the larger study. The sample size used in the larger study was not large enough for the small treatment effect expected in the the present study which led to its lower than normal power level.

6) Definition of Terms

Adiposity:

Refers to the amount of adipose tissue present in a persons' body.

Body Fat:

Tissue whose primary function is lipid storage.

Cardiovascular Fitness:

The measurement of an individual's capacity for aerobic energy transfer (predicted Vo_2 max) as determined by the Balke treadmill test.

Hyperlipidemia:
A general term used to describe elevated concentrations of any or all plasma lipids.

Lower Body Adiposity:
A fat distribution pattern in which the adipose tissue is located peripherally in the femoral (hip/thigh) region. This is denoted by a WHR $\leq$ 0.80.

Mildly Hypercholesterolemic:
A medical condition where total blood cholesterol levels are between 6 and 8 mmol/L.

Muscle Mass:
The total weight of muscle mass measured in kg.

Postmenopausal:
Refers to women who are 55 years old or older and one year since their last period as indicated by self reported medical questionnaire.

Skinfold:
A double layer of skin and the underlying adipose tissue not including muscle.

Upper Body Adiposity:
A fat distribution pattern in which the adipose tissue is located centrally around the abdominal region. This is denoted by a WHR > 0.80.

CHAPTER TWO:

REVIEW OF LITERATURE

Little research has been done concerning the effects of aerobic exercise on body fat, fat distribution and serum lipid levels of women after menopause. As a result, it is unknown whether or not these factors can be positively altered in order to reduce the risk of developing CHD.

Research articles pertinent to this area of study are reviewed in order to establish the need for further research. The review of literature is organized into five sections: plasma lipids as a risk factor for CHD, sex hormones and their effect on body composition, effects of exercise on lipids and body composition, influence of initial levels of body fatness and fat distribution on changes in body composition and measurement of energy intake. In the first section, background information on the health risks associated with an adverse lipoprotein pattern, as well as factors that are known to influence, or be associated with plasma lipoproteins, are reviewed. A brief summary of male and female sex hormones, including how and where they are produced, how their levels vary over the years, and their developmental influence on body composition, is included in the next section. The third section looks at how lipids, body fat and body fat distribution are affected by exercise. The influence of other factors on the effect exercise has on changes in body composition is discussed in the fourth section. A comparison of different dietary measurement tools, their strengths and weaknesses and appropriateness for use in the present study are summarized in the final section.

1) Plasma Lipids as a Risk Factor for CHD

Levels of serum total cholesterol above 6.2 mmol/L have been found to directly or indirectly increase the risk of developing CHD (Connelly et al., 1992) and have long been acknowledged as a major risk factor in the development of this disease (Goldstein' et al., 1973; Kannel, et al., 1979; Keys, 1970). Numerous studies have shown that populations with high levels of total cholesterol or LDL-C and/or low levels of HDL-C have higher incidences of coronary artery disease (Goldburt et al., 1979; Gordon et al., 1977). Factors which have been found to influence or be associated with a women's plasma lipoprotein pattern include: body fat, body fat distribution, age and heredity.

In both males and females high levels of body fatness are associated with an adverse lipoprotein pattern which includes elevated levels of triglycerides, total cholesterol and LDL-C and decreased levels of HDL-C (Després et al., 1988), and a concomitant increased risk for CHD. The upper body fat distribution pattern, which is reflected by a WHR > 0.80, is correlated with an adverse lipoprotein pattern. The abdominal adipocytes of people with central type obesity are larger and have a higher rate of lipolysis (release and efflux of fatty acids from fat tissues) than the adipocytes located in the gluteal/femoral region (Rebuffé-Scrive et al., 1990). Because of this, increased levels of free fatty acids and glycerol are released from the abdominal adipocytes into the portal vein (Rebuffé-Scrive et al., 1990) thus increasing the risk of developing metabolic aberrations including glucose intolerance, hyperinsulinemia and hyperlipidemia. These aberrations are associated with elevated levels of VLDL-C and TG, and decreased levels of HDL-C (Olefsky, et al., 1974) thereby increasing the risk of development of CHD in individuals with a predominance of upper body fat, for example men and postmenopausal women.

Age also influences plasma lipoprotein pattern. A direct relationship exists between age and plasma total cholesterol levels (Deprés et al., 1988; Grundy et al., 1982). A cross sectional study of North American adults found that plasma triglycerides and LDL-C tend to rise in concentration with increasing age (Heiss, et al., 1980). Connelly et al, (1992) found that total cholesterol decreased slightly after the age of 54 in men while it gradually increased with age in women. As a result, total cholesterol values were higher after the age of 54 in women than in men. Based on the results of the above studies it can be seen that the risk for developing CHD increases with age and that this increase in risk is greater in women than men.

Genetic makeup has also been found to influence plasma lipoprotein profile. A study looking at the effect identical exercise programs had on six sets of monozygotic twins found that there was a similar decrease in plasma triglycerides, and oral glucose load, and an increase in HDL-C ratio, between each pair of twins. These results demonstrate that heredity plays a strong determining role as to how plasma lipids are altered due to a short-term training program (Després et al., 1988).

2) Sex Hormones and Their Effect on Body Composition

The sex hormones found in males and females have a major effect on body composition. This next section will describe the major hormones in males and females, their production, variation over the life span, and effect on body composition.

Sex Hormone Production

Both sexes use a common pathway of steroid hormone biosynthesis in gonadal tissue. The starting compound is cholesterol

which is either formed by de novo synthesis from acetyl CoA or taken up from the circulating plasma pool. The first step that occurs is a side chain cleave (20,22-desmolase step) of the cholesterol. This step occurs in the mitochondria and seems to be the rate limiting step for the synthesis of progesterone, of the androgens testosterone, dihydrotestosterone, and androstenediol, and of the estrogens estradiol and estrone.

From the initial side chain cleave of the cholesterol, two parallel pathways for testosterone production occur (Berne and Levy, 1988). The factors that determine which route will be taken are unknown.

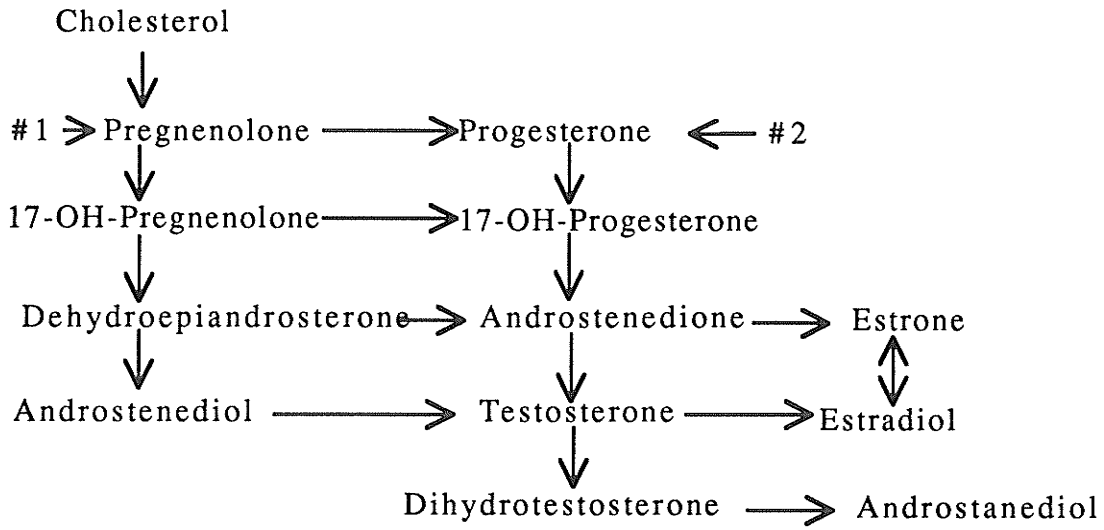

Figure 2-1 Pathways of synthesis of gonadal steroid hormones (Adopted from Berne & Levy (1988))

If the biosynthesis is occurring in the male gonadal tissue, the testis, the process leads to the production of testosterone. A small quantity of the testosterone produced would undergo further 5α reduction to dihydrotestosterone and a further α reduction to 5α–androstanediol.

If the biosynthesis is occurring in the female gonadal tissue, the ovary, there is further conversion of the androgens to estrogens: estrone, and estradiol. This conversion is controlled by the aromatase enzyme complex cytochrome P450. These series of steps occur in the endoplasmic reticulum and culminate with the production of estrone and estradiol from androstenedione and testosterone, respectively. The estrone produced by this process may also be interconverted to estradiol (see Fig. 2-1) (Berne & Levy, 1988).

Male Sex Hormones

In men, the major sex hormone is testosterone which is synthesized in the Leydig cells of the testis. It's synthesis and release by the Leydig cells is regulated by luteinizing hormone (LH). In men, LH is secreted in a pulsatile manner and therefore plasma testosterone levels also show small pulses throughout the day. Testosterone gives rise to two other potent androgens: dihydrotestosterone and 5α-androstanediol. The major fraction of circulating dihydrotestosterone and 5α-androstanediol comes from the reduction of testosterone by peripheral tissues. These two hormones are only secreted in small amounts by the testis.

Throughout life testosterone levels rise and fall. At about the age of 11, plasma testosterone begins a steep rise reaching an adult plateau at about age 17. This level of plasma testosterone is sustained for about 50 years. Of the circulating testosterone, approximately 50 to 60% is bound to sex steroid binding globulin while the rest is bound to albumin and other proteins. It is the free or loosely bound albumin fractions of testosterone and the other androgens that are biologically active. These hormones have a reproductive function as well as a major effect on secondary sexual characteristics. They also play an important role in the stimulation

of tissue growth and maturation. It is testosterone that first stimulates the pubertal growth spurt and then causes the cessation of linear growth by the closure of the epiphyseal growth centers. The testosterone and androgen hormones also cause the enlargement of muscle mass and the decrease in body fat that occurs during puberty in males. During the seventh or eighth decade of life plasma testosterone levels decline about 50 % due to the loss of Leydig cells' responsiveness to stimulation by LH (Berne & Levy, 1988).

Female Sex Hormones

The female sex hormones are the estrogens (estrone and estradiol), and the progestins. The estrogens are a family of hormones that can be produced in either the ovarian or extraovarian tissues. The most active and important naturally occurring ovarian hormone is estradiol. Estrone and estriol are also produced in the ovary but their biological activity is less than that of estradiol.

Estrogens are formed by the aromatization of the androgens by a complex process that uses the aromatase enzyme complex cytochrome P450. A more detailed description of the estrogen production pathway can be seen in Figure 2-1. As previously mentioned, estrone is formed from the aromatization of androstenedione while estradiol is formed from testosterone or estrone (Berne & Levy, 1988). In this way estrone is an important substrate for a series of reversible and irreversible reactions and a precursor for a series of irreversible reactions in the 16α pathway, leading to 16α-hydroxyesterone and then estriol; and alternate hydroxylation at C-2 which yields the catechol estrogens (see fig. 2-2) (Hershcopf & Bradlow, 1987). The catechol estrogens, 2-hydroxyestrone and 2-methoxyestrone have weak estrogenic activity but are potent agents in the mammalian central nervous systems (Berne & Levy, 1988). The other hormones produced in this

pathway, estrone and it's products, 16α-hydroxyestrone and estriol are nonactive.

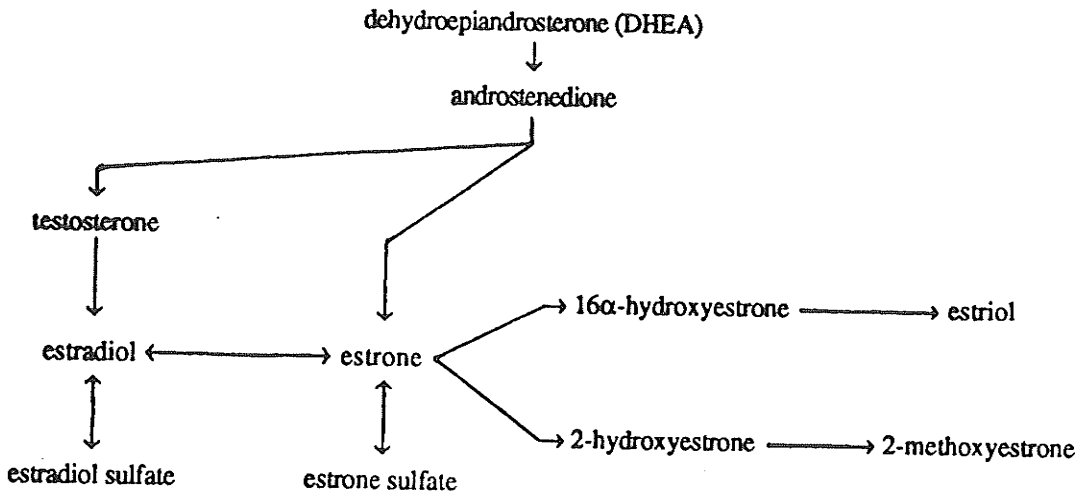

Figure 2-2. Pathways of estrogen productions and metabolism. (Adapted from Hershcopf, R. J., & Bradlow, H. L.(1987)).

Prior to menopause, most circulating estradiol is produced in the ovaries although a small amount of estradiol can also be formed by peripheral aromatization of testosterone. The estradiol formed in this manner is considered to be of little importance. Estrone, the other estrogen can be formed in three ways: in the ovaries, or via extraglandular oxidation of estradiol (Gurpide et al., 1962), or by the peripheral aromatization of androstenedione. The latter pathway is important in premenopausal women because it provides up to 50% of the daily production of estrone (Siiteri & MacDonald, 1973). The progestins are the other family of hormones produced and released by the ovaries. The most active of the progestins is progesterone which is produced and secreted by the corpus luteum (Berne & Levy, 1988).

The secretion of the steroid hormones fluctuates throughout the menstrual cycle. The amount of hormone secreted is directly related to the rate of synthesis in the ovaries. The hormones

are secreted when they are produced by the ovaries and as a result there is no storage of hormones (Berne & Levy, 1988). Once released from the ovaries, the hormones perform two functions. First they, function locally to modulate the complex events involved in the development and extrusion of the ova. Secondly, they are secreted into the plasma circulation and act on diverse target organs including the uterus, vagina, breasts, hypothalamus, pituitary glands, bones, liver, and adipose tissue. Upon secretion from the ovaries the estrogens and progestins enter the plasma and are circulated throughout the body bound in varying degrees to plasma transport proteins. About 98% of plasma estradiol and estrone is tightly bound to sex hormone binding globulin (SHBG). The remaining portion of these hormones are more loosely bound to albumin (Bruning, 1987). Free estradiol (non protein bound) is considered to be more available for biological activity than protein bound estradiol although estradiol bound to albumin may also be active (Bruning, 1987). The progestins circulate bound to corticosteroid-binding globulin (CBG).

The increase in sex hormones at puberty, especially estradiol of the estrogen family, leads to the development of secondary sex characteristics, skeletal maturation, and sex differentiation. It is this surge of hormones that is responsible for the preferential accumulation of fat in the gluteal/femoral region and the development of the lower body fat pattern. After the onset of puberty the hormonal levels of the estrogens and progestin remain unchanged until approximately age 50 at which time menopause occurs. During menopause the ovarian production of estrogen almost completely stops (Judd et al., 1974) although androgens continue to be produced. In fact, testosterone production is barely decreased while the ovaries secrete only minimal amounts of androstenedione. As a result the plasma levels of estradiol and estrone are greatly reduced. The peripheral aromatization of androstenedione to estrone becomes the major pathway of hormone production and estrone, the less biologically active estrogen, becomes the dominant hormone.

The plasma ratio of estrone to estradiol becomes 2:1 or 3:1 which is similar to the ratios found in men (Samoljik et al., 1977). It is this hormonal profile that is responsible for the similarity that exists in body fat distribution patterns between older men and women after menopause. Vague commented on this phenomena as early as 1956 when he discovered that there was a high percentage of overlap of fat distribution patterns between the sexes, especially at the two extremes in life (Vague, 1956).

Sex Hormone Binding Globulin

Estrogens are transported throughout the body in the blood bound to sex hormone binding globulin (SHBG). Studies have found that a relationship exists between plasma SHBG levels and the relative androgenic/estrogenic activity occurring in the body. An increase in plasma SHBG levels represents an increase in estrogenic activity while a decrease in plasma SHBG levels represents increased androgenic activity (Evans et al., 1983; Peirsis et al., 1989).

This interrelationship can be observed at puberty in both males and females. At puberty, males have increased androgenic activity due to increased plasma testosterone. This is reflected by a decrease in plasma SHBG levels. In females at puberty there is increased estrogenic activity due to increased plasma estrogen levels and as a result plasma SHBG levels are elevated. This relationship between SHBG levels and relative androgenic/estrogenic activity can also be observed in the elderly. At menopause there is increased androgenic activity due to the decrease in the synthesis of estradiol and the increase in testosterone levels. This increased androgenic activity is once again marked by a decrease in plasma SHBG levels. In men over 50 there is increased estrogenic activity, demonstrated by an increase in plasma SHBG levels. Based on these observations, researchers have stated that SHBG is a valid marker of relative androgenic/estrogenic activity (Wu et al., 1976).

Sex Hormones in Relation to Regional Adiposity

Plasma sex hormones and/or SHBG levels are related to level and pattern of fatness. Sex hormones have been found to affect both the size and number of fat cells developed, and the distribution of fat.

Adipose tissue mass is regulated by both the amount of triglyceride in each adipocyte and the number of adipocytes with the capacity to store triglycerides. In vitro studies have established that mammals, including adult humans, possess adipocyte precursors capable of replication and complete differentiation into mature fat cells (Van et al., 1976). This composition of adipocyte precursor pool is thought to be an important determinant of the growth of adipose tissue and appears to explain inter-regional and inter-individual differences (Dijan et al., 1983).

Male and female sex hormones have different effects on these adipocyte precursors and precursor pools. In vitro studies have shown that at puberty the female sex hormone 17β estradiol stimulates replication of human adipocytes in culture (Roncari & Van, 1978) and leads to an increase in overall fatness. Furthermore, research has found that the increase in estrogen level leads to the development of lower body fat distribution which is characterized by a decreased waist to hip ratio (WHR< 0.80). Researchers conclude that these changes are a result of the increased ovarian production and secretion of 17β estradiol and are representative of the pubertal endocrine activity in girls (Ridder et al., 1990). The male sex hormones testosterone and dihydrotestosterone do not influence growth or replication of the adipocyte precursors (Roncari, 1981) and, in fact, actually appear inhibitory. This is demonstrated by the

fact that at puberty males' body fat and adipocyte number decrease (Vague et al., 1984). Studies have also found that the male sex hormones cause fat to be centrally localized in adolescent males, resulting in the development of a central body fat distribution characterized by an increased waist to hip ratio (WHR > 0.80) (Baumgartner et al., 1987).

In summary, sex hormones play a role in the development of overall fatness as well as fat distribution. Female sex hormones increase overall adiposity and cause the development of lower body fat distribution. Male sex hormones play an inhibitory role on the adipocyte precursors and precursor pool leading to a decrease in the number of adipocytes and the amount of body fat. These hormones also cause fat to be preferentially distributed in the abdominal region which leads to the development of an upper body fat distribution.

Relative Androgenic/Estrogenic Balance

Much evidence suggests that body fat distribution is more a function of relative androgenic/estrogenic balance than of androgen or estrogen levels alone (Evans et al., 1983; Peirsis et al., 1987; Ridder et al., 1990). Androgen levels are elevated in adolescent girls with an upper body fat distribution (Ridder, et al., 1990). Both the onset of androgen secretion in males at puberty and the administration of exogenous testosterone to hypogonadal males leads to the development of an upper body fat distribution and a decrease in plasma SHBG levels (Evans et al., 1983). In nonobese and obese adult women central type adiposity (designated by a high WHR or waist to thigh ratio-WTR) is positively correlated with total serum testosterone (Kirschner et al., 1990), free testosterone (Evans et al., 1983), free testosterone to total testosterone ratio (Seidell et al., 1989), and the production rates of testosterone and

dihydrotestosterone (Kirshner et al., 1990). An inverse relationship exists between WHR and SHBG levels. Women with the highest WHR have the lowest levels of plasma SHBG because of the increased plasma androgen levels (Evans et al., 1983; Ridder et al., 1990; Kirschner et al., 1990).

Menopause is characterized by a more androgenic hormonal profile which alters the androgenic/estrogenic balance and results in the shift from a lower body fat distribution to an upper body fat distribution pattern (Vermeulen & Verdonck, 1972). With estrogen replacement therapy there is a decrease in upper body fat mass (Seidell et al., 1989) suggesting that the estrogens shift the androgenic/estrogenic balance back towards the premenopausal levels.

In both men and women, adipose tissue distribution is centrally located in the presence of an androgenic hormonal profile and located in the gluteal/femoral region in the presence of an estrogenic environment and/or when androgenic influences are weak. These observations support the hypothesis that body fat distribution is a result of the relative androgenic/estrogenic balance.

Body Fat Levels

High body fat levels confound the relationship of sex steroids with regional adiposity. This is because individuals with a high body fat percentage generally tend to have elevated sex steroid levels and decreased plasma SHBG levels.

Both plasma levels and metabolism of estrogens are elevated in conjunction with high body fat levels (Herschopf & Bradlow, 1987), as adipose tissue is capable of converting androstenedione to estrone via peripheral aromatization. Individuals with high levels of body fatness have greater amounts of adipose tissue and are therefore able to convert androstenedione to estrone

at an accelerated rate. The percentage conversion of androstenedione to estrone rises from 1 to 2% in normal weight subjects to 12 to 15% in people who are extremely overweight (Siiteri, 1987).

Most individuals with a high body fat percentage have an upper body fat distribution. This appears to be because of the well recognized inverse relationship of obesity with SHBG (Peirsis et al., 1989). Research has found that high body fat levels result in a decrease of SHBG in both men and women. The reason for this decrease, however, is not known. As a result of the decreased plasma SHBG levels, premenopausal women with a high body fat percentage have increased levels of biologically inactive estradiol and plasma free testosterone. These elevated levels result in an androgenic hormonal profile causing an upper body fat distribution to develop.

In men with high body fat levels the decreased SHBG levels are associated not only with elevated estrogens but also with decreased plasma testosterone levels (Glass et al., 1977; Schneider et al., 1979) and subnormal levels of free testosterone (Schneider et al., 1979). Therefore, men with a high body fat percentage develop an estrogenic hormonal profile as opposed to the expected androgenic hormonal profile. One would expect this estrogenic hormonal profile to lead to the development of a more lower body fat distribution, however, these men instead develop a more upper body fat distribution. The reason for this phenomena is unknown but it may be explained by the relationship that exists between person's body fat level and WHR. Studies have found that as an percent body fat increases so to does WHR (Després, 1991). This relationship explains why men with high body fat levels have an android body fat distribution even though they have a more estrogenic hormonal profile.

Regional Characteristics of Adipocytes

Sex steroids are able to influence body fat distribution by acting on the adipose tissue mass either in the abdominal or gluteal/femoral regions. Sex steroids are able to control adipocyte hypertrophy (increase in fat cell volume) and adipocyte hyperplasia (increase in fat cell number) in these two regions thereby influencing body fat distribution.

Before menopause women generally have a lower body fat distribution with a greater number of adipocyte cells in the gluteal/femoral region than the abdominal region. These cells are also larger than those in the abdominal region (Rebuffé-Scrive et al., 1985). The small number of women with an upper body fat distribution have significantly larger abdominal, but the same sized gluteal/femoral, adipocytes as women with lower body fat predominance (Hartz et al., 1984; Kissebah et al., 1982; Larsson et al., 1984). In this case SHBG and estrogen levels correlate inversely with abdominal cell volume (Evans et al., 1983). These findings indicate that large abdominal adipocytes and an android fat distribution are not estrogen dependent traits.

In contrast, after menopause women have larger abdominal but smaller gluteal/femoral adipocytes. Abdominal and gluteal/femoral adipocytes are also the same size (Rebuffé-Scrive et al., 1985) once again demonstrating that the lower body fat distribution is regulated by female sex steroids (Bjorntorp, 1991).

In summary, both premenopausal women with an upper body fat distribution and postmenopausal women have larger abdominal adipocytes as a result of decreased levels of estrogen. Most premenopausal women typically have a lower body fat distribution because of the effect of the female sex steroids. The elevated level of plasma estrogen causes adipocyte hypertrophy and hyperplasia in the gluteal/femoral region leading to lower body fat predominance.

Regional Differences in Adipocyte Metabolism

Sex steroids are not only able to affect differentiation and growth of adipocytes but are also able to affect the adaptation of these cells to new metabolic demands. In these two ways sex hormones are able to influence and control adipocyte size.

Sex steroids control the metabolism of fat cells and ultimately their size by exerting influence over two processes that control the uptake and release of triacylglycerol from adipose tissues. These two processes are lipolysis and lipogenesis. Lipolysis is the release and efflux of fatty acids from adipose tissue and is controlled by the action of hormone sensitive lipase. This enzyme is responsible for the hydrolysis of triacylglycerols to fatty acids and glycerol which can then be released from the adipose tissue into the bloodstream. Lipogenesis is the uptake of triacylglycerol into adipose tissue during periods of affluence and is controlled by the rate limiting enzyme lipoprotein lipase (LPL). This enzyme functions by hydrolyzing the triacylglycerols, which are transported in very low density lipoproteins (VLDL) and cholymicrons, into fatty acids which are then taken up by the adipose tissues. Both lipolysis and lipogenesis display sex and regional differences that are responsible for the common male and female fat distribution patterns (Berne & Levy, 1988).

Obese men and women with an android body fat distribution, have large abdominal adipocytes with a higher rate of lipolysis than those adipocytes located in the gluteal/femoral region (Rebuffé-Scrive et al., 1990; Wahrenberg et al., 1989) as well as decreased insulin sensitivity (Wahrenberg et al., 1989). This helps to explain why individuals with android obesity have a high frequency of impaired glucose tolerance. The higher rate of lipolysis in the large abdominal adipocytes leads to an increased rate of free fatty

acid release resulting in inhibition of the glucose uptake by the peripheral tissues and leading to peripheral insulin resistance.

The elevated rate of lipolysis found in the large abdominal adipocytes of people with android obesity is due to the interaction of adrenoreceptors located in the adipocytes. Adipose tissue cells have both α-adrenergic and β-adrenergic receptors that are sensitive to catecholamines. An increased number or sensitivity of β-adrenergic receptors in the adipocytes leads to an increase in the rate of lipolysis whereas an increased number or sensitivity of α-adrenergic receptors leads to a decreased rate of lipolysis. Research has found, in both males and females, that large abdominal fat cells are much more responsive than gluteal/femoral fat cells to the lipolytic effect of catecholamines. In both sexes there was a 10-to-20 fold increase in the rate of lipolysis in the abdominal fat cells when the β-agonist isoprenaline was added. There was no such increase in the gluteal/femoral fat cells indicating a much greater β-adrenergic sensitivity in the abdominal cells than in the gluteal/femoral cells in both sexes. This increase in β-adrenergic sensitivity in the abdominal adipocytes may be explained by either an increase in the number and/or an enhanced affinity of the β-receptors. Wahrenberg et al., (1989) found that there were twice as many β-adrenergic binding sites in the abdominal adipocytes than in the gluteal/femoral adipocytes. Thus the large abdominal fat cells, found in individuals with android obesity, have a higher rate of lipolysis due to an increased number of β-adrenergic binding sites.

Premenopausal women with lower body fat distribution have larger adipocytes in the gluteal/femoral region as compared to the abdominal region (Rebuffé-Scrive et al., 1985; Rebuffé-Scrive et al., 1986). The gluteal/femoral adipocytes are larger than the abdominal adipocytes because of their increased LPL activity and decreased catecholamine stimulated lipolysis (Rebuffé-Scrive et al., 1985; Rebuffé-Scrive et al., 1986). Studies have shown that by adding phentolamine, a known α-adrenergic blocker, to the

gluteal/femoral adipocytes the rate of lipolysis of these cells increases to a level equal to the rate of lipolysis found in the abdominal cells when they are stimulated by epinephrine. These results indicate that the decreased rate of lipolysis of the gluteal/femoral adipose tissue cells to catecholamine is due to an increased number or sensitivity of α-adrenergic receptors (Rebuffé-Scrive et al., 1985).

Post menopause women have larger abdominal and smaller gluteal/femoral adipocytes. This difference seems to be due to the decreased secretion of sex hormones that occurs as a result of menopause. These decreased levels of sex hormones eliminate the increased LPL activity of the gluteal/femoral region and blunt the increased lipolytic response of the abdominal cells. As a result postmenopausal women have abdominal and gluteal/femoral fat cells of the same size and lipolytic activity which leads to the development of the android fat distribution (Rebuffé-Scrive et al., 1986).

In conclusion, the distribution of body fat varies with both age and sex. At puberty there is a dramatic increase in the levels of circulating sex hormones in both males and females. In males this increase in production and release of testosterone by the testis leads to an overall decrease in body fat, increase in lean body mass, and the development of the android fat distribution. This body fat distribution dominates in men throughout their adult life. In females, at puberty there is an increase in the production and secretion of estrogens by the ovaries causing women to maintain virtually the same level of body fatness and to develop a lower body fat distribution which is maintained until menopause. At menopause the ovarian production of estrogen almost completely stops resulting in a more androgenic plasma hormone profile which leads to the development of an upper body fat distribution. Thus, after the age of approximately 55 the fat distribution patterns of males and females once again overlap.

3) Effects of Exercise on Lipids and Body Composition

Elevated levels of plasma total cholesterol, triglycerides, and LDL-C as well as low levels of HDL-C are risk factors for CHD (Goldburt et al., 1979; Gordon et al., 1977). High levels of body fatness and an upper body fat distribution (Després et al., 1988; Olefsky et al., 1974; Peiris et al., 1989) are highly correlated with this adverse lipoprotein profile and an increased risk for developing CHD. Several different approaches have been used in an attempt to improve the lipid profile and reduce the risk for developing heart disease. Leon et al., (1987) found physical activity to be an inexpensive intervention that has beneficial effects on several risk factors for CHD including blood pressure, body composition and serum lipid profile.

A clear cut relationship has been found to exist between exercise intensity and improvements in cardio-vascular fitness (Wenger et al., 1986). The American College of Sports Medicine (1991) determined that training intensities between 40 and 85% functional capacity, depending on initial fitness level, are required to improve aerobic fitness. The optimal exercise prescription to achieve health benefits, such as positive changes in serum lipids and body composition, is less conclusive but appears to be dependent upon total energy expenditure (Superko, 1991; Cauley et al., 1982).

Aerobic exercise has been found to positively alter both the serum lipid profile and body composition of men. High intensity exercise programs (70 to 85% of VO_2 max) or programs of long duration (3 to 4 months) which cause large amounts of energy to be expended, have been reported to decrease plasma total cholesterol, triglycerides, LDL-C levels and TC/HDL-C ratio and to increase plasma HDL-C levels (Tran & Weltman, 1985; Brownell et al., 1982). Exercise of this intensity or duration has also been found to cause men to lose significant amounts of weight (Ballor & Keesey, 1991; Brownell et al.,

1982; Després et al., 1984; Wood et al., 1991) and total body fat (Andersson et al., 1991; Ballor & Keesey, 1991; Bouchard et al., 1990), to preferentially lose fat from the abdominal region, as indicated by a decreased WHR (Wood et al., 1991) and to maintain or increase fat free mass (Ballor & Keesey, 1991; Bouchard et al., 1990).

Controversy exists over the effect aerobic exercise has on the serum lipid profile of young women. Some studies have found that exercise favorably alters serum lipid profiles in women, as in men, by reducing their plasma total cholesterol (Després et al., 1991; Lokey & Tran, 1989; Owens et al., 1990), triglycerides (Gibbons et al., 1983; Owens et al., 1990), LDL-C (Després et al., 1991; Owens et al., 1990) and TC/HDL-C ratio (Gibbons et al., 1983; Lokey & Tran, 1989). Haskell (1984) concluded that it may be more difficult to bring about changes in the plasma lipoproteins of women than in men in response to exercise. He felt this was due to the elevated levels of HDL-C and lower levels of TC, TG, and LDL-C found in premenopausal women when compared to men.

Positive alterations in body composition appear to be related to total energy expenditure. Studies have reported that women lost significant amounts of body weight (Andersson et al., 1991; Ballor & Keesey, 1991; Brownell et al., 1982; Gwinup, 1975; Owens et al., 1990) and total body fat (Després et al., 1991; Ballor & Keesey., 1991; Owens et al., 1990; Schaberg-Lorei et al., 1990; Tremblay et al., 1990) when they participated in exercise programs of high intensity (70 to 85% of $V0_2$ max) or of long duration (6 to 18 months). Studies which have investigated the effects of volumes of exercise on body compositional changes have determined that energy expenditure must exceed 2000 kcal/wk in order for beneficial changes to occur (Cauley et al., 1982; Leon et al., 1979).

Exercise has also been found to positively alter the body fat distribution of women prior to menopause. The majority of studies reviewed found that vigorous aerobic activity caused women to preferentially lose fat from the abdominal region, thereby

decreasing the WHR and the risk for developing CHD (Després et al., 1991; Krotkiewski, 1988; Tremblay et al., 1990)

Despite the increased risk for developing CHD, few studies have analyzed the effects of aerobic exercise on the serum lipids or body composition of postmenopausal women. Only two studies were found that looked at the effect of aerobic exercise on the serum lipids of postmenopausal women. Owen et al., (1992) determined that an increase in physical activity by postmenopausal women was not related to a concomitant increase in plasma HDL-C levels, but rather the increase in exercise prevented a decrease in HDL-C levels. They also discovered that women who increased their activity levels at least 300 kcal per week over a three year period, which is equivalent to walking approximately three times per week for 20 minutes, displayed a smaller decrease in HDL-C levels. Cauley et al., (1982) reported that postmenopausal women must expend over 2000 kcal of energy per week in order to increase their serum HDL-C level and that low intensity activities were the main determinants of HDL-C levels. The results of these studies seem to indicate that aerobic exercise has a positive influence on serum lipids in women post menopause thereby reducing the risk of developing CHD.

Only one study could be found that looked at the effect aerobic exercise on the body composition in women after menopause. Schaberg-Lorei et al., (1990) found that aerobic exercise, in combination with a light resistance weight program, caused women after menopause to significantly decrease their weight and to preferentially lose abdominal fat. Other than this study no other research with this age group could be found. It is this lack of research that prompted the American Heart Society to state that future studies must include sufficient numbers of women and the elderly (Fletcher et al., 1992).

4) Influence of Initial Levels of Body Fatness and Fat Distribution on Changes in Body Composition

Initial level of fatness has also been found to affect the amount of weight or body fat lost by premenopausal women. Two studies reported that weight and fat loss were greatest in women who had the highest initial values (Andersson et al., 1991; Ballor & Keesey, 1991). Andersson et al., (1991) found that body fat loss was confined to the most obese subjects while the lean women did not lose significant amounts of fat.

Initial body fat distribution has also been found to affect the change in WHR and the amount of weight and body fat lost by premenopausal women. When placed on calorie reduced diets, women with an upper body fat distribution significantly decreased their WHR while the WHR of women with a gynoid fat distribution remained unchanged (Casimirri et al., 1989; Wadden et al., 1988). Casimirri et al., (1989) found that women with upper body obesity lost significantly greater amounts of weight and body fat than women with lower body obesity. In contrast, Wadden et al., (1988) found that women with lower body obesity lost significantly greater amounts of weight and body fat than women with an upper body fat distribution. The reason for this difference in results is unknown.

In conclusion, aerobic exercise of high intensity or of long duration is sufficient to positively alter the body composition and plasma lipid profile of men and premenopausal women. The effect aerobic exercise has on the body composition of premenopausal women is influenced by their initial weight, body fat levels and body fat distribution.

Most research showing a decrease in body fat and a beneficial change in body fat distribution has dealt with young adults

who were involved in vigorous or high intensity exercise programs of short duration. Few studies have systematically investigated the effect of chronic physical activity on the risk factors for CHD in the elderly population, particularly postmenopausal women. The high levels of activity, which have been shown to be beneficial in young adults, may not be achievable when dealing with the elderly. It seems clear more research is needed in order to determine the true effects of a moderate intensity program of extended duration on the body composition of postmenopausal women at risk for CHD.

5) Measurement of Energy Intake

Body composition and plasma lipid levels are strongly influenced by the content of the diet and the amount eaten. Hence estimates of food energy and nutrient intake are important to assess the influence of dietary changes. Nutritionists and epidemiologists have developed and utilized a variety of techniques to estimate the food intake of individuals.

Certain measurement techniques, such as direct observation, weighed food records and seven day diaries have been found to accurately record all nutrients consumed by individuals in free living populations. These techniques, however, may not accurately reflect usual dietary intake. With direct observation and weighed food records individuals are required to dissect, weigh and record all foods eaten over the predetermined measurement period. The need to weigh and record intake may lead to a reduced or more monotonous diet, which affects the accuracy of the measurement technique. With seven day food diaries individuals are required to write down or record everything they eat immediately after consumption. This can quickly become very tedious leading to problems with compliance which once again affects the accuracy of the measurement technique. Subject training and sustained

cooperation are also necessary to ensure the accuracy of these measurement techniques. Unfortunately these are very time consuming and expensive processes making direct observation, weighed food records and seven day food diaries impractical for use in large scale studies (Barrett-Connor, 1991; Block, 1982; Samet et al., 1984). Other techniques, such as diet history, which includes a 24-hour diet recall, a history of unusual foods and data on food preparation, require a 1-2 hour interview by a specially trained nutritionist. This once again makes them too costly and time consuming for use in large studies. Recall methods, such as the 24 hour dietary recall, are more readily used with a large number of subjects but may not provide sufficiently stable estimates of individual intake. This is not due to the accuracy of the measurement technique but to the fact that individual diets can vary greatly from day to day. A 24 hour dietary recall would therefore be unable to reflect this day to day dietary variability (Samet et al., 1984).

In order to meet the needs of large-scale epidemiological research alternative methods have been developed. One such method is the food frequency questionnaire which was devised in an attempt to obtain a self-administered, inexpensive and rapid estimate of usual dietary intake (Barrett-Connor, 1991). With this technique subjects are sent questionnaires that list a number of food items which are selected specifically for the subjects involved in the study. The individuals are then asked to record the number of times they remember eating the individual food items over a pre-determined number of days. The data obtained by the self-administered questionnaire is directly scanned into a computer data base designed specifically for the questionnaire. It is this scanning of data that greatly reduces the time and the cost of this technique. Research on this assessment tool has found that it provides an accurate measurement of relative intakes of specific foods and nutrients (Barrett-Connor, 1991; Mullen et al., 1984), even though its

validity needs to be assessed for specific responding groups (Samet et al., 1984). Therefore, due to its accuracy of estimating group and individual food intake, ease of administration and low cost, and despite its possible uncertain validity, this method has been deemed appropriate for use in large scale studies where the objective of data collection is to establish a subject's intake and dietary makeup (Barrett-Connor, 1991; Block, 1982; Mullen et al., 1984; Samet et al., 1984). Research with males and females has also determined that the minimum number of days required to estimate true average intake and proportion of carbohydrates, protein and fats in the diet to be three days (Basiotis et al., 1987).

CHAPTER THREE:

METHODS AND PROCEDURES

The purpose of this study was to examine the effects of a six month brisk walking program on total body fat, body fat distribution, muscle mass, and the relationship between initial fatness/body fat distribution and fat loss, in postmenopausal, mildly hypercholesterolemic women.

Data was collected as part of a larger study that examined the effects of a brisk walking program on the lipoprotein profile, body composition, and bone mineral density of the above study population.

1) Design of the Study

The study lasted for 24 weeks. Measurements of serum lipids, diet and body composition were taken at three month intervals throughout the study (zero, three and six months). Following screening, 41 women were considered eligible for the study. One of the participants withdrew from the study prior to assignment to a group leaving the number of participating subjects at 40. The subjects were then randomly assigned to a walking group (n= 24) or a control group (n= 16).

2) Calculation of Power Index

Since the sample size of the present study was already predetermined, as a result of being part of a larger study, it was important that its power level be calculated. The power of a study describes how likely the study is to detect a genuine treatment effect between the experimental and the control groups. A powerful study

will almost certainly find genuine differences if there are present while a weak test could very easily miss them. The power level is determined by 1- β. The power level of a study depends on several factors. It depends on the size of the α level. The smaller the α the larger the β and, hence, the smaller the power (1-β). The power level is also dependent on the sample size. The larger the sample size the smaller the β and the larger the power. It also depends on the true treatment effect. This is the size of the change that is expected to occur as a result of the intervention. The more dramatic the treatment effect the smaller the β and the larger the power.

Therefore in order to calculate the power level of the present study the α level was set at 0.05 and the sample size was set at 15 walkers and 10 controls. Literature provided information regarding the changes that can be expected to occur in percent body fat as a result of aerobic exercise. From this data it was possible to estimate the EFFECT SIZE based on the results of Lewis et al., (1976). They determined that premenopausal women, with an average age of 44 ± 6.8, would lose 4.9% body fat (Pretraining level 40.4 ± 6.2, Posttraining level 35.4 ± 5.8) as a result of a walking/jogging program. Based on these expected changes the following formula, as described by Hassard (1991), was used to determine the study's true treatment effect and power level.

$$\text{True Treatment Effect} = \frac{H1-H2}{\sqrt{\frac{\sigma_1^2}{n} + \frac{\sigma_2^2}{n}}} = \frac{4.9}{\sqrt{\frac{6.22}{15} + \frac{5.82}{15}}} = 2.23$$

True Treatment Effect = 2.23

The "α portion" of the true treatment effect at the 0.05 level equals 1.96.

The "β portion" of the true treatment effect = 2.23-1.96 = 0.27.

$P(z>0.27)= 0.3930$
$\beta = 0.3930$
Power= $1-\beta$
$= 1- 0.3930$
$= O.6070$

The power level of the present study is 0.6070. This is lower than the commonly accepted power level of 0.80. Therefore the study has an increased chance of missing a genuine treatment effect if one did exist between the experimental and the control groups.

3) Recruitment

Recruitment of subjects began in December, 1990. Bulletins were placed in the Winnipeg Free Press and other newspapers, and investigators involved in the study appeared on radio and television programs to recruit interested volunteers. Researchers received several hundred enquiries about the study. Approximately 300 women were sent medical questionnaires, and one hundred and five of them were asked to undergo further testing. Forty one were selected as eligible for the study.

4) Screening Procedure

Prior to being allowed into the study, women completed an informed consent form and medical questionnaire. Participants had to be non-smokers, 55 years of age and older, consume less than 1 ounce of alcohol per day, be clinically healthy (no cardiovascular, pulmonary or metabolic diseases that would preclude safe participation in an exercise program), and not on medications to

control their serum lipid levels (bile binding resins, statins, fibric acid derivatives, niacin, probucol). Serum cholesterol levels had to be between 6.0 to 8.0 mmol/L. Women with triglyceride levels greater than 4.2 mmol/l were excluded from the study as well as women who were receiving estrogen replacement therapy. Only sedentary or minimally active individuals who had not made recent changes in their activity patterns, and who were physically capable of exercising, were included in the study.

A medical history, resting 12 lead electrocardiogram (ECG), symptom limited exercise test (as described below), and an initial screening lipid measurement (as described below) were evaluated by a cardiologist in order to ensure that the volunteers could safely take part in the exercise program. Changes in medical status were monitored by a questionnaire which was given after the subjects completed the six month program.

5) Diet Analysis

Prior to the study the Nutrition Department of the University of Manitoba designed a food frequency questionnaire specifically for the women involved in this study, the majority of whom were Caucasian women from Winnipeg or the immediate surrounding region. A special mainframe database program, derived from the 1988 Canadian Nutrient File, was written to handle the data from this specific food frequency questionnaire. Also prior to the start of the study trained staff from the Nutrition Department of the University of Manitoba met individually and in groups with all subjects involved in the study and taught them how to properly and accurately fill out the three day food frequency questionnaires. After this training period the women recorded all foods consumed during a three day period, including two week days and one weekend day via the food frequency questionnaire. The data was

then entered into the mainframe database that was specifically designed for the study. Once entered the individual data was statistically analyzed using the SAS statistical program. The program determined a subject's total caloric intake (in kilojoules and kilocalories) and the proportion of the total caloric intake made up of carbohydrates, proteins and fats. Those people with high fat diets (greater than 40% caloric intake) were asked to complete a second three day food frequency record. All subjects who underwent this second analysis were kept in the study with the recommendation that they lower their intake of dietary fat. Subsequent three day food frequency records and computer analysis were completed at three month intervals throughout the investigation.

6) Exercise Tests

Symptom Limited Exercise Test:

A Balke treadmill test was used to determine whether subjects could safely participate in the exercise program and to predict VO_2 max (Pollock et al., 1976). A physician supervised each test and decided whether or not the subject could safely participate in the study. The subjects walked on the treadmill at a constant speed of 5.4 km/hr (3.3 miles/hr). Elevation began at 0% grade at 0 minutes, and increased by 1% per minute. Blood pressure measurements (sitting and supine) and a 12-lead ECG were taken prior to the test. ECG tracings and blood pressure were monitored each minute during the test. The test was terminated if ECG abnormalities were apparent, the systolic blood pressure increased above 260 mmHg, or the subjects became exhausted. A familiarization session was held with each subject prior to the treadmill test in order to decrease the subjects' anxiety and to eliminate a learning effect. To monitor alterations in cardiovascular

fitness level (VO_2 max), participants in the walking and control groups were tested at zero and six months.

Submaximal Steady State Test:

Each subject in the walking group completed a submaximal steady state treadmill test to determine the energy cost of walking at various velocities in order to predict the weekly and total energy costs of the walking program. The participants walked on the level treadmill (0% grade) for up to four ten minute stages and achieved steady state at each stage. Four different velocities were used. The initial 10 minute stage was used both as a warm up and to calculate energy costs. The subjects walked at a velocity equal to the one at which they had been walking at the end of week two (V1). The velocity used in stage three corresponded to the velocity at which the subjects were walking when their target heart rate was reached (V3). Stage two used the velocity corresponding to the velocity walked at the mid point between the end of week two and the time the target heart rate was attained (V2), while stage four used the velocity which corresponded to the end of the six month program (V4). If the subjects were found to walk at identical velocities during two or more of these measurement periods they completed only one steady state stage at this velocity. Velocities were preferably calculated using a time, taken to walk 5 laps recorded, on a weekly basis by a fitness monitor or by using distance/time (m/min) recorded in the participants' log books.

Expired gases were collected during the final minutes of each stage, using a Metabolic Measurement Cart (Sensormedics) so that the steady state oxygen uptake (VO_2) and the respiratory exchange ratio (R) could be determined. Heart rate was continually recorded throughout the test using a Sport Tester (Polar). Energy expenditure was calculated using the caloric equivalent for a liter of oxygen at the measured R value (Gardner et al., 1988).

A similar steady state treadmill protocol has been shown to be an accurate method of predicting energy cost, while walking on a track in this population (Brennan, 1990). The submaximal steady state test took place approximately six months after the initial start of each individual's walking program. Energy expenditure for each week was interpolated from the values obtained with this test.

7) Blood Lipid Assay

Subjects were asked to abstain from consuming alcoholic beverages for 72 hours and eating food 12 hours prior to blood sample collection. Two samples of 15 ml of blood were collected, from each subject on two different occasions, by venipuncture using vacutainer tubes (BD EDTA). The level of plasma cholesterol in each sample was determined enzymatically (Allain et al., 1974). The average of these measurements was used as an indicator of the subject's initial total cholesterol level. If this level was found to be between 6 to 8 mmol/L the subjects were included in the study.

8) Body Composition

Body composition measurements were taken at the beginning of the study and retests were done at time zero, three, and six months. Body composition was assessed by extensive anthropometry which included skinfold measurements (triceps, biceps, subscapular, iliac crest, ilio-spinale, abdominal, front thigh, medial calf), girths, breadths, lengths, height and weight in accord with procedures outlined by Ross, Marfell-Jones and Stirling, (1982). Measurements were made in triplicate. From the above anthropometric measurements waist to hip ratio (WHR), waist to thigh ratio (WTR), and subscapular to tricep skinfold thickness ratio

(STR) were calculated. Percent body fat was calculated from skinfold measurements according to the equation of Jackson, Pollock and Ward, (1980). Muscle mass was calculated from stature, thigh circumference corrected for the front thigh skinfold thickness, uncorrected forearm circumference, and calf circumference corrected for the medial calf skinfold thickness measurements according to the equation of Martin, Spenst, Drinkwater and Clarys, (1990).

9) Activity Record

A detailed activity record was completed by each subject prior to and following the exercise intervention. This allowed the activity levels of the exercise and control groups to be compared.

10) Training Program

Participants in the walking group took part in a six month program of walking which progressed to five times per week, for 60 minutes. Desired exercise intensity was 60% of heart rate reserve (HRR) = [0.60 (max heart rate - resting heart rate)] + resting heart rate. Maximum heart rate was recorded during the Balke Treadmill Test. Participants who were unable to reach a heart rate within 20% of the age predicted maximum were placed on a less intense program during the initial weeks. Similarly, participants who were initially unable to complete one hour of walking progressed in 20 minute increments.

The walking took place on an indoor track, on measured routes within local malls, or on measured routes outdoors during the summer months. Exercisers were monitored at the university by one of the investigators twice per week for the initial two weeks of the program, and then once per week. Although each participant usually

walked at her own prescribed pace, the weekly supervised sessions were conducted in groups. Average walking speeds, measured as the time to complete five laps of the track, were measured weekly. Subjects were taught to monitor their own heart rates either by the radial or carotid pulse and were encouraged to maintain the appropriate pace. Steady state heart rates, as measured at 15 minute intervals were reported for each walking session. A log book was provided in which average exercise heart rate, distance walked, attendance, and other physical activity were recorded. Participants were instructed to keep other endurance activities to a minimum. The energy cost of walking for each week was estimated based on the distance covered, assuming an energy expenditure equivalent to that calculated from submaximal treadmill tests described earlier.

11) Exercise Progression

Increased walking distance and speed was used to build progression into the exercise program. As subjects became more fit, the distance completed in the designated time increased, thereby ensuring progression. As resting heart rate declined in response to training, there was a decrease in target heart rate calculated as 60% heart rate reserve. For this reason, subjects continued to train at their initial target heart rate, or at a slightly elevated heart rate, while the distance walked increased.

12) Data Analysis

T-tests were used to test the effect of the 24 week walking program on fitness levels (VO_2 max), body composition (weight, percent body fat, body mass index and muscle mass), and body fat distribution (waist, hip, thigh and tricep girth, WHR, WTR and STR). Paired t-tests were used to compare zero and 24 week

measurement values in order to determine if there were any significant differences within groups. Two sample unpaired t-tests of the change scores (24 week-0 week values) were used to determine if the walking group values changed significantly more than did those of the control groups.

Correlation coefficients were used to test for covariance between independent variables. Only body mass index (BMI) and weight were found to have a high covariance.

A straight reqression was used to determine if a statistically significant relationship existed between any of the independent and dependent (outcome) variables:

a) initial body fat levels and a change in percent body fat (fig. 4.5) or change in body fat distribution (WHR, WTR and STR) (fig. 4.6-4.8)
b) initial WHR and the subjects' loss of body fat (% body fat)
c) walkers' energy expenditure and loss of body fat (fig. 4.2)
d) walkers' energy expenditure per kilogram of body weight and initial percent body fat levels (fig. 4.3)
e) change in VO_2 max and change in percent body fat (fig. 4.4), body fat distribution and muscle mass (Table 5).

If a statistically significant relationship was found to exist a three step procedure was used which controlled for the influence of the independent variable (covariate) so that the true affect of the walking program on the outcome (dependent) variables could be determined. The first step of the procedure, which must be done before undertaking a formal statistical analysis, was the development of a scatter diagram. The scatter diagram provided insight into the nature of the relationship exhibited by the data and if a linear relationship was found to be formed an analysis of covariance (ANCOVA) was then performed.

In the ANCOVA the influence the covariate had on the outcome variable was determined and controlled for so that the true affect the walking program had on the dependent variables could be

determined. The covariates were the independent variables that were regressed against the outcome or dependent variables in the previous analysis. The independent variable in all the ANCOVA analysis was the group variable. For example if a statistically significant relationship was found to exist between initial percent body fat (independent variable) and change in percent body fat (outcome variable) then an ANCOVA was done. In this particular ANCOVA initial percent body fat would be the covariate, change in percent body fat the outcome variable and group the independent variable. In this way the affect the walking program had on change in percent body fat could be determined while the influence initial percent body fat had on this relationship was controlled for.

If the ANCOVA determined that a significant relationship existed between the independent variable (group) and the dependent variable then another scatter diagram was plotted. On the second diagram the regression line or line of best fit was drawn in to give a visual representation of the relationship.

CHAPTER 4: RESULTS

The results of the 24 week walking study are reported for 25 of the original 40 subjects. Below is a flow chart (Figure 4-1) that explains how the final group of 25 was derived.

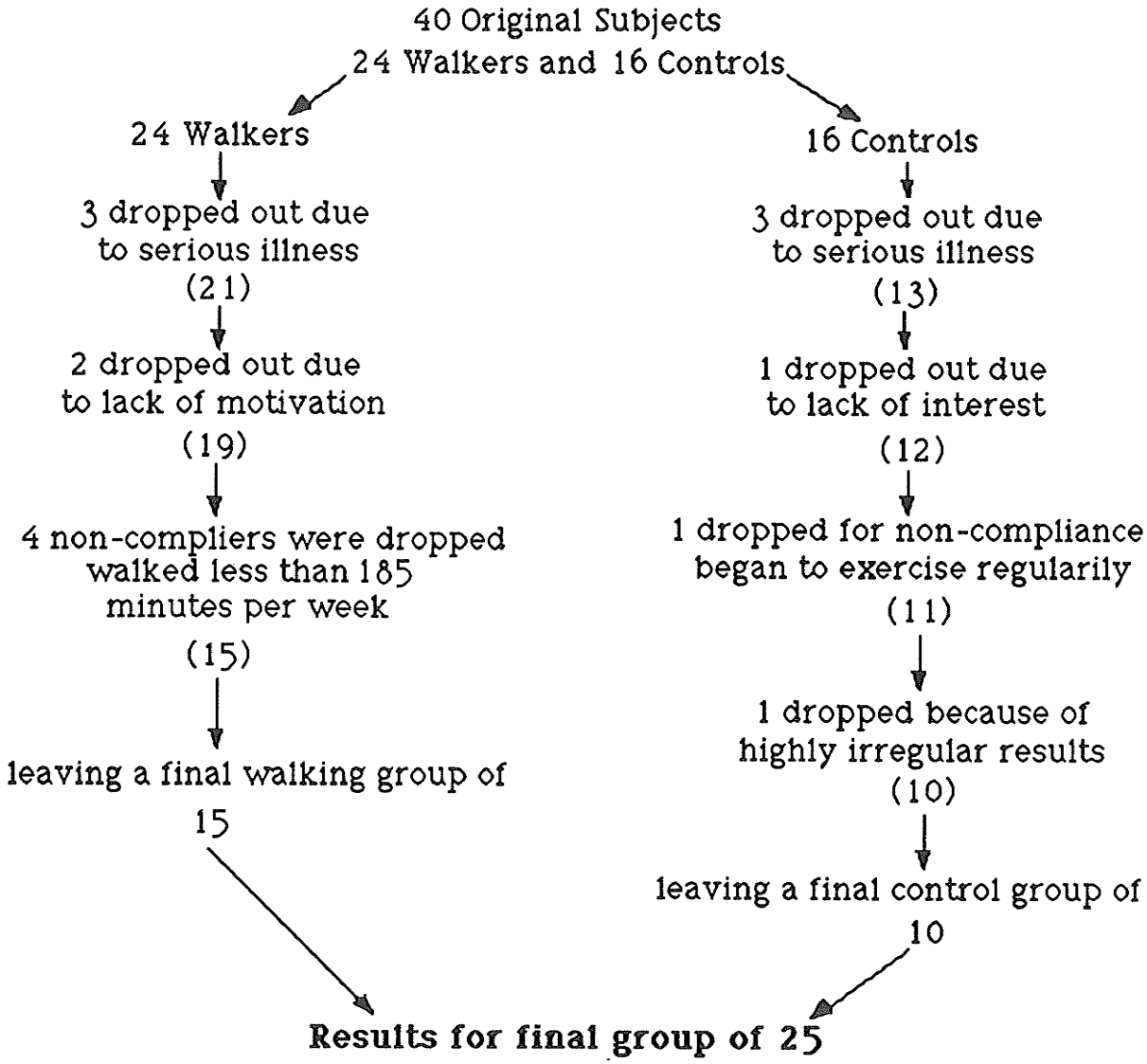

Figure 4-1 Derivation of final experimental groups.

1) Physical Characteristics of Subjects

Body composition variables (mean ± SD), for subjects completing the study, measured at the zero week period are presented below in Table 1. None of the initial body composition measurements were significantly different between the walking and control groups.

Table 1 Subject's characteristics prior to the start of the study (x ± SD).

Parameters	Walkers	Controls
Number (n)	15	10
Age (yrs)	60.6 ± 4.0	62.3 ± 5.4
Height (cm)	162.0± 5.6	159.9 ± 6.1
Weight (kg)	77.2 ± 16.4	82.5 ± 11.3
BMI§	29.4 ± 6.1	32.2 ± 3.9
% Fat†	36.8 ± 7.0	40.4 ± 6.5
WHR	0.80 ± 0.04	0.83 ± 0.06

§ Derived by dividing weight in kilograms by height in meters squared.

† Predicted from skinfolds, Jackson et al, 1980.

2) Energy Expenditure

The total energy expended during the 24 week walking program is listed in Table 2. The table only includes 14 values as opposed to the expected 15 because one of the walkers did not complete the submaximal steady state test, making it impossible to calculate their total energy expenditure. On average the walkers (n=15) exercised for 54.3 minutes per session, 4.9 times per week, at an intensity of 54% of HRR. An average of 6366 kj were expended per week.

Contrary to expectations, the change in percent body fat of the walkers was not significantly related to the amount of energy expended (Figure 4-2). An additional regression was done to determine whether this was due to variation in energy expended in association with initial fatness, as it was possible that women with more fat were unable to expend as much energy during the program. Initial percent body fat was not significantly related to energy expenditure per kilogram of body weight (Figure 4-3), suggesting that variation in initial fatness was not responsible for the significant relationship in Figure 4-2.

3) Changes in Body Composition

The body composition and fitness levels of the walkers and controls prior to and following the 24 week study are listed in Table 3. In comparison to the control group, the weight and BMI of the walkers decreased significantly while their $V0_2$ max increased significantly. Neither the controls' nor the walkers' muscle mass or percent body fat changed significantly.

Table 2. Total energy expenditure for walkers (n=14) during 24 week program.

Walker	Energy Expenditure (Kilojoules)
Subject 1	247,043
Subject 2	* n/a
Subject 3	154,298
Subject 4	180,510
Subject 5	149,055
Subject 6	192,789
Subject 7	185,526
Subject 8	165,209
Subject 9	143,272
Subject 10	102,887
Subject 11	170,512
Subject 12	148,234
Subject 13	132,663
Subject 14	109,185
Subject 15	57,822
Average Expenditure (x ± SD)	152,786 ± 45,349

* subject did not complete steady state test so value could not be determined.

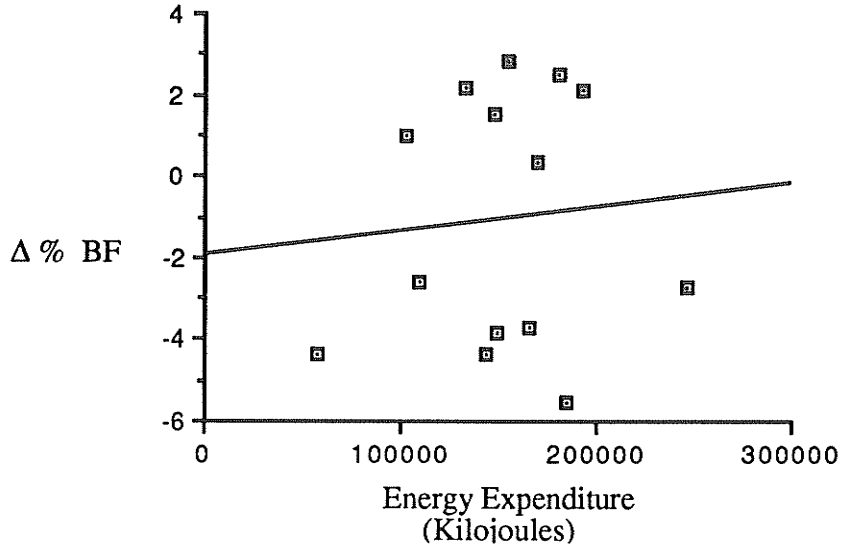

Figure 4-2. Energy expenditure for the walkers versus change in percent body fat (Δ %BF).

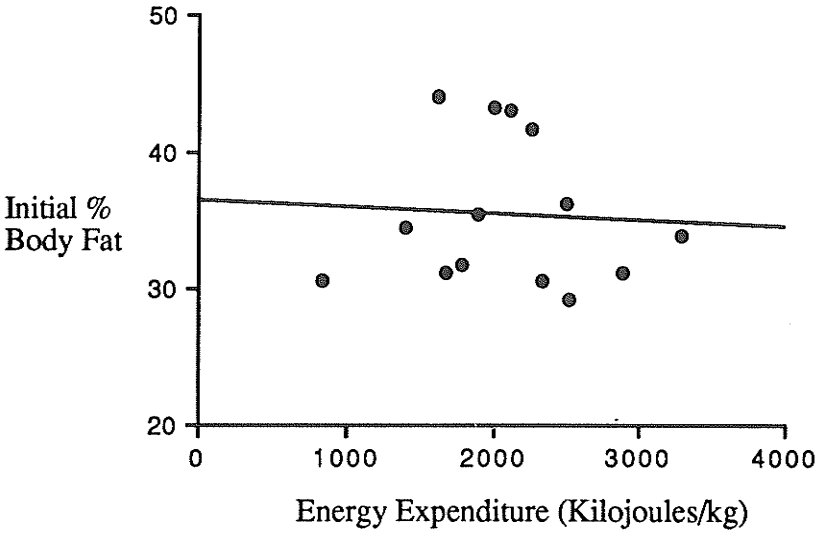

Figure 4-3 Energy expenditure (kilojoules/kg) versus initial percent body fat (%BF).

Table 3 Body composition and $V0_2$ max prior to and following a 24 week program in walkers (15) and controls (10) (X ± SD).

	Walkers			Controls		
Parameters	Pre	Post	Δ(Post-Pre)	Pre	Post	Δ(Post-Pre)
Weight (kg)	77.2 ± 16.4	75.2 ± 15.8	-2.0*	82.5 ± 11.3	82.7 ± 11.8	0.2
BMI	29.4 ± 6.1	28.5 ± 5.9	-0.9*	32.2 ± 3.9	32.4 ± 4.1	0.2
% Fat	36.8 ± 7.0	35.5 ± 7.9	-1.3	40.4 ± 6.5	40.0 ± 6.6	-0.4
M.M. (kg)	28.1 ± 5.7	27.1 ± 5.3	-1.0	27.4 ± 3.6	27.9 ± 5.2	0.5
$V0_2$ max § (ml/kg/min)	30.0 ± 4.9	33.5 ± 5.8	3.5*	25.6 ± 2.7	25.9 ± 2.7	0.3
$V0_2$ max § (l/min)	2.25 ± 0.24	2.42 ± 0.30	0.17*	2.10 ± 0.27	2.14 ± 0.04	0.04

BMI Body mass index derived by dividing weight in kilograms by height in meters squared.
M.M. Muscle mass in kilograms.
* Change values significantly different ($p < 0.05$) Walkers vs Controls.
§ Significant difference Walkers vs Controls at pre test ($p < 0.05$).

4) Changes in Body Fat Distribution

The walkers' waist girth was found to decrease significantly more than that of the controls, while the waist to hip ratio hip, tricep and mid thigh girth in both groups remained unchanged (Table 4).

5) Relationship of Changes in Fitness Levels with Changes in Body Composition

The change in VO_2 max was correlated with a change in percent body fat (Table 5). Upon further analysis the increase in VO_2 max was found to be primarily due to decreased body weight.

6) Relationship of Initial Percent Body Fat and WHR with Changes in Percent Body Fat

Contrary to what was hypothesized a significant relationship was not found to exist between initial percent body fat and change in body fat distribution. This is demonstrated by the lack of significant relationship between the subjects' initial percent body fat and change in waist to hip ratio, waist to thigh ratio and subscapular to tricep ratio (Figure 4-6, 4-7 and 4-8). An ANCOVA analysis also found that the walkers' ability to lose body fat or change body fat distribution was not related to their initial waist to hip ratio.

7) Dietary Intake and Composition

The dietary intake and composition of the controls and walkers were not significantly different at the zero week measurement period. The walkers' caloric intake and percentage of diet made up of carbohydrates and fats did not change significantly as compared to that of the controls over the 24 week study period (Table 6).

Table 4 Body fat distribution prior to and following 24 week program in walkers (15) and controls (10) (x ± SD).

	Walkers			Controls		
Paramaters	Pre	Post	Δ(Post-Pre)	Pre	Post	Δ(Post-Pre)
WHR§	0.80 ± 0.04	0.79 ± 0.05	-0.01	0.83 ± 0.06	0.84 ± 0.05	0.01
Waist Girth	87.6 ± 10.7	84.9 ± 10.5	-2.7*	93.8 ± 8.4	93.7 ± 9.1	-0.1
Hip Girth	110.2 ± 13.8	107.5 ± 12.3	-2.7	113.0 ± 11.4	112.5 ± 13.1	-0.5
Tricep Girth	32.6 ± 3.8	31.8 ± 4.0	-0.8	34.5 ± 2.8	34.3 ± 2.6	-0.2
Mid Thigh Girth	53.1 ± 6.3	51.0 ± 6.1	-2.1	54.0 ± 3.1	52.8 ± 3.3	-1.2
Abdominal SF	38.6 ± 11.4	41.9 ± 13.3	3.3	46.6 ± 11.2	50.5 ± 9.5	3.9
Tricep SF	27.9 ± 6.7	25.6 ± 7.1	-2.3	30.9 ± 6.7	31.0 ± 7.1	0.1
Mid Thigh SF	43.6 ± 14.0	36.6 ± 16.7	-7.0	49.2 ± 11.5	43.0 ± 14.5	-6.2
Calf SF	27.9 ± 11.0	27.1 ± 12.5	-0.8	33.1 ± 7.3	30.4 ± 8.6	-2.7

§ Waist to hip ratio. Derived by dividing the waist girth values by the hip girth values.
* Change values significantly different ($p < 0.05$) Walkers vs Controls.

Table 5 Correlation of changes(Δ) in $V0_2$ max with changes in body composition (R^2 values).

	Δ WTR	Δ WHR	Δ STR	Δ MM	Δ % BF	Δ FFM
Δ $V0_2$ max (ml/kg/min)	0.007	0.05	0.16	0.01	0.24*	0.002
(l/min)	0.05	0.05	0.16	0.07	0.37*	0.02

* Significantly related ($p<0.05$).
Δ WTR Change in waist to thigh ratio.
Δ WHR Change in waist to hip ratio.
Δ STR Change in subscapular to tricep skinfold thickness ratio.
Δ MM Change in muscle mass.
Δ % BF Change in percent body fat.
Δ FFM Change in fat free mass.

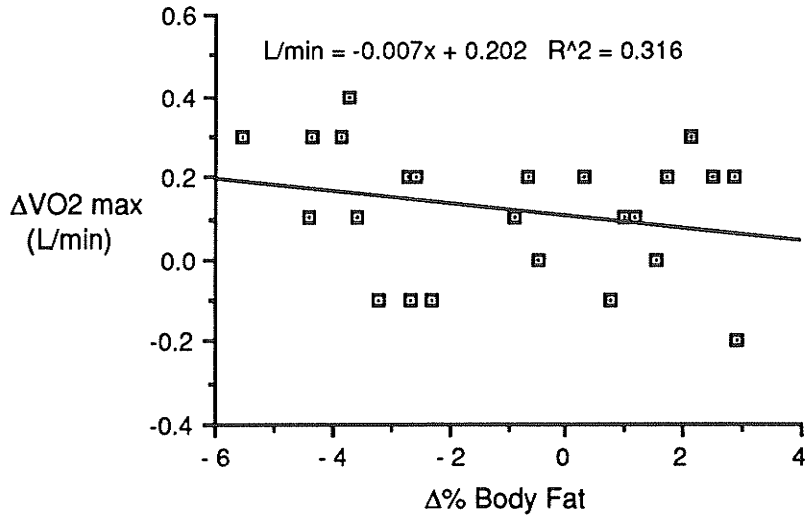

Figure 4-4 Change in percent body fat (Δ %BF) versus change in maximal oxygen uptake (Δ VO_2 max).

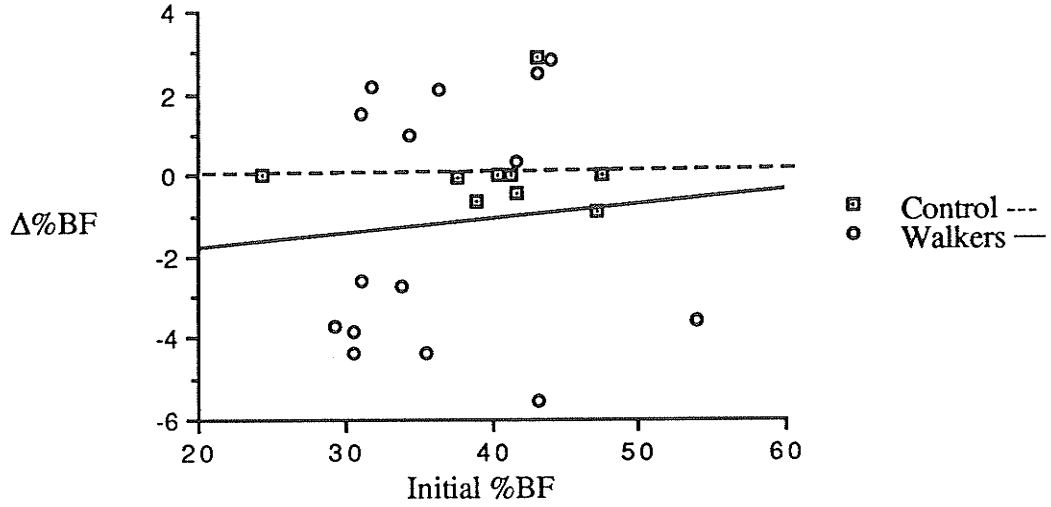

Figure 4-5. Initial percent body fat (%BF) versus change in percent body fat (Δ %BF).

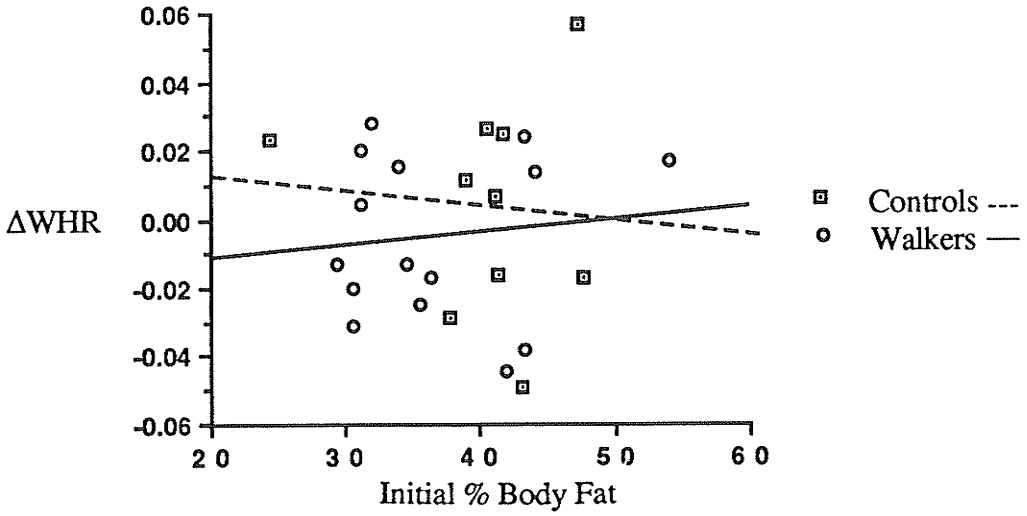

Figure 4-6. Initial percent body fat (In %BF) versus change in waist to hip ratio (Δ WHR)

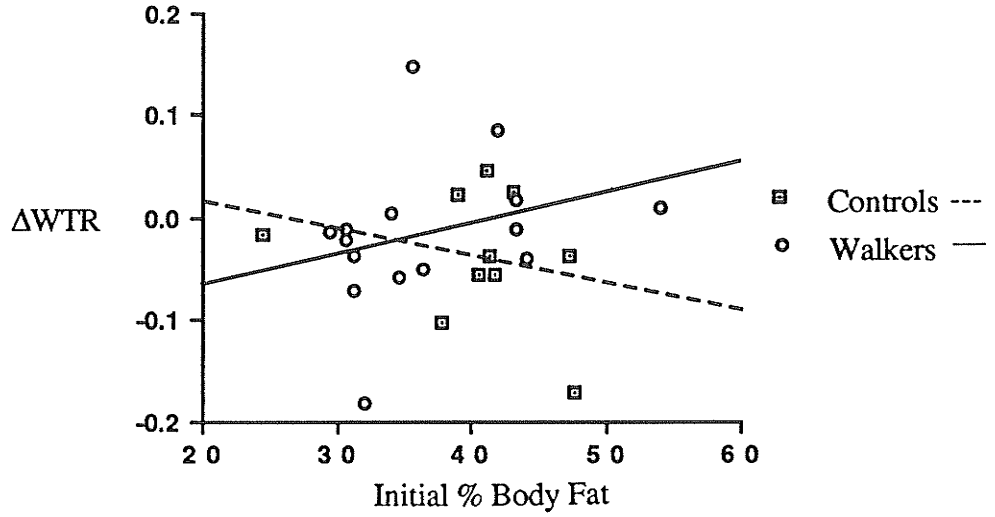

Figure 4-7. Initial percent body (In %BF) fat versus change in waist to thigh ratio (Δ WTR).

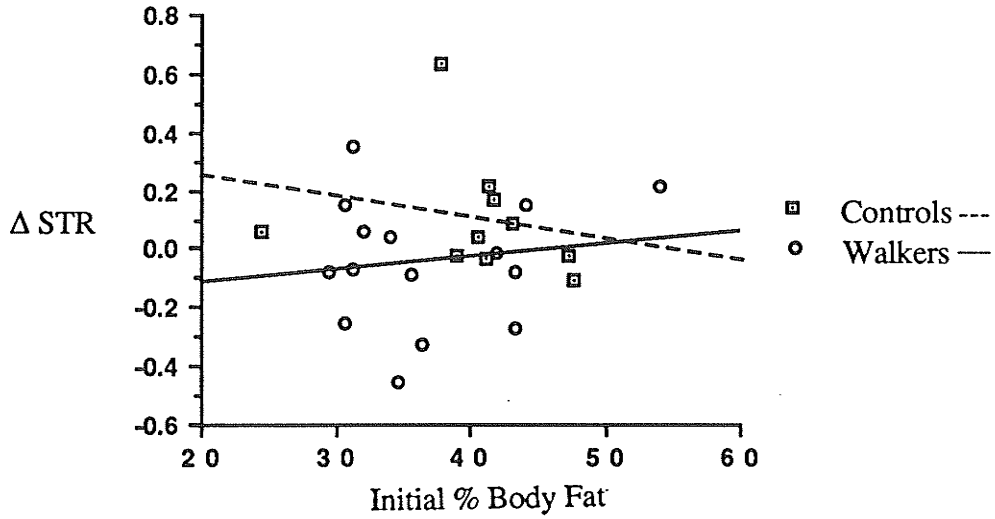

Figure 4-8 Initial percent body (In %BF) fat versus change in subscapular to tricep ratio (Δ STR).

Table 6 Daily energy intake and composition prior to and following 24 week program in walkers (15) and controls (10) (X ± SD).

	Walkers		Controls	
Parameters	Pre	Post	Pre	Post
Total Kilojoules	6869 ± 2120	7146 ± 3484	7454 ± 1668	6305 ± 1195
% Carbohydrates	51.8 ± 6.8	53.7 ± 5.8	50.8 ± 7.5	53.6 ± 5.3
% Protein*	18.7 ± 2.3	18.0 ± 2.0	16.4 ± 2.4	17.1 ± 2.2
%Fat	29.6 ± 5.7	29.0 ± 4.2	32.3 ± 4.3	30.8 ± 5.1

* Significant difference Walkers vs Controls at pre test ($p < 0.05$).

CHAPTER 5:

DISCUSSION

1) Introduction

Cardiovascular disease is the leading cause of death in Canada for both sexes, accounting for 41 percent of all deaths (Statistics Canada, 1990). After menopause, women have an increased risk of developing coronary heart disease, so much so that cardiovascular disease becomes the primary cause of death by the sixth decade (Statistics Canada, 1990). The increased risk is due to changes that occur in serum lipids, body fat levels and body fat distribution as a result of, or in conjunction with, menopause. Despite the increased risk of development of CHD in women after menopause little research has been done on this group. This study was designed to determine whether a six month program of walking could positively alter the body fat levels and body fat distribution of mildly hypercholesterolemic women post menopause thereby reducing their risk of developing CHD.

2) Compliance

The study began with 40 subjects randomly assigned to either a walking group (n=24) or a control group (n=16). A greater proportion of subjects were assigned to the walking group because approximately 30 percent of the exercisers were expected to drop out or withdraw before the completion of the study (Gwinup, 1975). During the 24 week study, three walkers dropped out due to serious illness and two dropped out due to lack of motivation. Four other

walkers were left out of the final analysis due to non-compliance having walked less than 185 minutes per week. This meant they had exercised only three times per week for 60.0 minutes. The rest of the walkers exercised on average 4.9 days a week for 54.3 minutes (average of 266 minutes per week) which was almost equal to the duration of the exercise program prescribed at the start of the study (5 days a week for 60 minutes). Three of the controls dropped out due to serious illness, another due to lack of interest and one was omitted for non-compliance. In this case the control subject began to exercise regularly. One other control was dropped from the final analysis because of highly irregular results. During preliminary statistical analyses it was found that her body composition measurements were consistently three to four standard deviations outside the mean values recorded for the rest of the controls. Therefore her results were dropped so that they did not abnormally skew the final results, interpretations and conclusions. Her results were also dropped in order to ensure that a normal distribution of results was maintained so that certain statistical analyses (t-test, correlation coefficients, regression analysis, and ANCOVA), which can only be used with a normal distribution of data, could be done. This left the study with a final walking group of 15 subjects and a final control group of 10.

On average, 25 to 50 percent of participants can be expected to drop out during a six month exercise program (Dishman, 1988; Gwinup, 1975). In our study 31 percent of the controls and only 21 percent of the walkers dropped out before the end of the six month study period. These attrition rates demonstrate that the exercise program was easily tolerated by the women and confirms the hypothesis that low to moderate activities, such as walking, are best suited to the abilities of older individuals (Procari et al., 1989).

3) Fitness Level Changes

Improvements in physical fitness are directly related to the intensity of an exercise program (Wenger et al., 1986). The women involved in our study walked, on average, at an exercise intensity equal to 54 percent of heart rate reserve. VO_2 max increased by 10% ($p<0.05$) demonstrating that the walking program was of sufficient intensity to improve the fitness levels of women after menopause.

Our findings are in agreement with the recommendations of the American College of Sports Medicine (1991) which states that an individual must exercise at a minimum intensity of 50 to 85% functional capacity or heart rate reserve in order to elicit improvements in maximal aerobic power. Wenger & Bell, (1986) determined that when exercise exceeds 35 minutes, a lower intensity of training (50 to 70% of $V0_2$ max) is sufficient to cause an increase in fitness levels comparable to those achieved when individuals work out for a short duration at 90 to 100% of their $V0_2$ max. Previous research has determined that aerobic exercise programs of 50 to 85 % heart rate reserve are of sufficient intensity to elicit a nine to thirteen percent increase in the fitness levels of women who have gone through menopause (Probart et al., 1991; Schaberg-Lorei, 1990; Steinhaus et al., 1990; Warren et al., 1993).

The change in VO_2 max was significantly and inversely related to the walkers' change in percent body fat. This result is consistent with the findings of Thomas et al., (1986) who calculated the contribution of fat reduction to the improvement in fitness levels to be about 20 to 33 percent in premenopausal women. The postmenopausal women's significant increase in VO_2 max in the present study can, therefore, be partially attributed to the decrease that occurred in percent body fat. The fact that this change in VO_2 max is not significantly related to a change in lean body mass further

indicates that the increase in VO_2 max is dependent on body fat loss. Other physiological improvements, such as improved ability to extract and transport oxygen, increased stroke volume and cardiac output (Steinhaus et al., 1990) are probably responsible for the remainder of the increase in VO_2 max.

4) Body Composition Changes

Body Weight and BMI

The optimal exercise prescription to achieve health benefits, such as positive changes in serum lipids and body composition, appears to be dependent upon total energy expenditure (Superko., 1991; Cauley et al., 1982). The walkers total energy expenditure was on average 152,787 kilojoules (36,504 kilocalories) or 6,366 kilojoules (1,521 kilocalories) per week throughout the 24 week study. This average weekly energy expenditure was sufficient to cause a significant decrease in body weight and body mass index.

These results are consistent with the findings of Schaberg-Lorei et al, (1990) who found that an exercise program consisting of 20 minutes of light resistance weights and 25-35 minutes of walking or jogging, was sufficient to cause both premenopausal and postmenopausal women to significantly reduce body weight. Other studies have also found that premenopausal women lost significant amounts of body weight when they participated in exercise programs of high intensity (70 to 85% VO_2 max) or of long duration (6 to 18 months) (Andersson et al., 1991; Ballor & Keesey, 1991; Brownell et al., 1982).

Although the average weight loss of two kilograms was a significant decrease, it is less than what would be expected from the estimated total energy cost of the program. The women in our study expended 6,366 kilojoules (1,521 kilocalories) per week or 152,787

kilojoules (36,504 kilocalories) in total over the 24 week period. If one assumes an energy equivalent of 25,113 kj/kg of adipose tissue (Després et al., 1991) one would have expected the women to have lost an average of six kilograms of body weight. Després et al., (1991) also found that women lost less body weight than what could be expected from the energy expended during exercise. They hypothesized that this resistance to fat loss was due to increased energy intake, or a compensatory decrease in energy expenditure the rest of the day.

Previous research involving animals (Rodin et al., 1990) and humans (Andersson et al., 1991; Tremblay et al., 1988) concluded that females react to exercise by increasing caloric intake more so than males. The women in the present study may have actually expended enough energy to lose the expected six kilograms of body weight but, because of energy overcompensation, some of the effects of the extra energy expenditure may have been negated. Energy overcompensation, therefore, may have been responsible for the less than expected weight loss. In the present study caloric intake and composition were monitored by three day food frequency records taken at the zero, twelve, and twenty-four week periods. Based on these records neither the walkers' caloric intake nor dietary composition changed significantly, suggesting that energy overcompensation was not responsible for the resistance to weight loss.

Gonan & Poehlman, (1992) discovered that vigorous endurance training did not increase total energy expenditure in older men or women after menopause because of a compensatory reduction in physical activity during the rest of the day. Because we did not keep track of energy expenditure outside the walking program this cannot be ruled out as a contributing factor.

One other possibility is that the two kilogram loss of body weight may not actually be less than what should be expected if we consider the different components that add together to produce the

total energy expenditure in the study. A six kilogram loss of body weight would have only occurred if all the 152,787 kilojoules of total energy expenditure were used for weight loss. This does not occur. A portion of each individual's total energy expenditure is made up of their resting metabolic rate.while walking. This works out to be 37,343 kilojoules of energy (344 kilojoules/hr x 120 hrs). Approximately another 20% of the total energy expenditure would be used by each individual to do the work involved in the actual walking. This works out to be another 20,000 kilojoules of energy that could not be used for weight loss. Thus, out of the 152,787 kilojoules of total energy expenditure only 95,444 kilojoules (152,787 - 37,343 - 20,000) are available to be used towards weight loss. If one assumes an energy equivalent of 25,113 kj/kg of adipose tissue (Després et al., 1991) then the women would be expected to lose 3.8 kilograms of body weight. This is much closer to the 2 kilogram loss of body weight that was found to occur in this study.

Changes in Percent Body Fat

Despite the significant decrease in body weight and body mass index the walking program was unsuccessful in significantly decreasing percent body fat. The walkers did, however, display an increased tendency to lose body fat when compared to the controls.

One possible explanation for the lack of significant change in percent body fat is that the walkers lost a significant amount of deep visceral fat as opposed to subcutaneous fat. The skinfold measurements, which register changes in subcutaneous fat, would therefore, be unable to detect the changes that occurred in the visceral fat mass. As a result a decrease in percent body fat would not be found to occur. Després et al., (1991), determined that in premenopausal women the greater the decrease in body weight with

training, the greater the reduction in the amount of deep abdominal fat. Therefore, it is possible that the women in the present study decreased visceral fat as opposed to subcutaneous fat and it was this fat loss that was responsible for the decrease in body weight.

The change in percent body fat was also not found to be significantly related to the amount of energy expended by the walkers. This is surprising as one would expect to see that the people who expended the most energy while walking would have lost the most fat. A compensatory decrease in physical activity is one way of explaining this, as total daily energy expenditure would not be significantly different from that prior to the walking program and there would be no relationship between energy expended while walking and change in body fat. Although activity for the remainder of the day was not monitored, the women did record all exercise in a log book. Based on these entries and conversations with the women, it did not appear as though they were inactive. However, daily energy expenditure outside of the walking program cannot be ruled out as a contributing factor for the lack of significant relationship between energy expenditure and change in percent body fat.

Change in Muscle Mass

The hypothesis that muscle mass would increase significantly as a result of a six month program of brisk walking was disproved. This is consistent with the findings of previous research which determined that aerobic exercise programs alone are unable to increase the muscle mass of premenopausal (Després et al., 1991; Kanaley et al., 1993; Schaberg-Lorei et al., 1990) or postmenopausal (Schaberg-Lorei et al., 1990) women. Research has found that aerobic exercise programs must be combined with, or interspersed with, strength training or weight bearing activities in order for significant changes to occur in the muscle mass of premenopausal

(Andersson et al., 1991) and postmenopausal women (Schaberg-Lorei et al., 1990) women. Based on our results it is apparent that our walking program did not provide enough weight bearing or strength training activity to increase the muscle mass of the women after menopause.

5) Change in Body Fat Distribution

Change in Upper and Lower Body Fat

The walkers experienced a significant decrease in waist girth while the hip girths of both groups remained unchanged. This suggests that the walking program caused a preferential loss of fat from the abdominal region. However, a closer inspection of the body fat distribution data and changes listed in Table 4, tends to contradict this conclusion.

The walkers' average waist and hip girths decreased the exact same amount (2.7 cm) as a result of the walking program. This seems to indicate that body fat loss occurred equally from the upper and lower body regions. The reason the walkers' hip girth was not found to decrease significantly more than did that of the controls is because of the wide individual variability of the controls' hip girth measurements. This coupled with the small experimental group sizes makes it difficult for any significant changes to be found. The initial group sizes were calculated based of the likelihood of observing significant changes in total cholesterol values as opposed to body fat distribution measurements. The group sizes were, however, not large enough to overcome the individual variability of the hip girth measurements and as a result significant results could not be found. Individual variability and its ability to conceal significant findings is one of the major problems that must be overcome when working with human subjects outside a clinical setting. Whenever possible

large group sizes should be used in the attempt to alleviate or control this problem.

The walkers' waist to hip ratio also did not change significantly more than did that of the controls. This once again disputes the statistical conclusion that preferential fat loss occurred from the abdominal region. Instead the lack of change in waist to hip ratio indicates that either no fat loss occurred or that fat loss occurred equally from the upper and lower body regions. The walkers' tricep and mid thigh girths, as well as their tricep, mid thigh and calf skinfolds also tended to decrease slightly as a result of the walking program. This finding further strengthens the argument that fat loss occurred equally from the upper and lower body regions.

Based on the overall changes in the walkers' body fat distribution measurements it safe to conclude that fat loss occurred equally from the abdominal and gluteal/femoral regions. The nonsignificant change in waist to hip ratio, the similar decrease in the waist and hip girths, and the trend towards decreasing girth and skinfold measurements indicate that fat loss occurred equally from the upper and lower body regions as opposed to preferentially from the abdominal region. This is one example where the statistically significant findings may not give an accurate overall picture as to what actually happened in the study.

Change in Abdominal Skinfold

There was a nonsignificant trend for the participants' abdominal skinfold to increase over the duration of the study. This is opposite to the fat loss trends that were found to occur in the other body fat distribution measurements. The waist to hip ratio and the other girth and skinfold values all show that fat loss occurred equally from the abdominal and gluteal/femoral regions. It is, therefore,

highly unlikely that the abdominal skinfolds of the subjects actually increased.

Large amounts of fat in the abdominal region make it very difficult for accurate abdominal skinfolds measurements to be taken. A large number of the subjects in the present study had a high percentage of body fat and therefore, large amounts of abdominal fat. As a result it was difficult to get accurate abdominal skinfold measurements. This difficulty in measuring this site may have led to inaccurate measurements and large fluctuations in values from one measurement period to the next and therefore, been responsible for the apparent increase in abdominal skinfolds.

One of the limitations of the study was that the body composition measurements (skinfolds and girths) at the zero, twelve and twenty-four week measurement periods were not always taken by the same tester on each individual. This decreased the reliability of the skinfold measurements from one measurement period to the next. Because the abdominal skinfold site was difficult to measure, this reduced reliability may be further magnified. The combination of the two may have been responsible for the slight increase that was found to occur in the subjects' abdominal skinfolds.

Even though the abdominal skinfolds were found to increase over the duration of the study it is highly unlikely that fat was actually gained in this area. Instead measurement error and tester variability are probably responsible for this questionable increase.

6) Influence of Initial Levels of Body Fatness and WHR on Changes in Percent Body Fat

Influence of Initial Levels of Body Fatness on Changes in Percent Body Fat

Initial levels of body fatness have been found to affect the amount of body fat lost by premenopausal women. Research has determined that weight and fat loss were greatest in women who had the highest initial values (Andersson et al., 1991; Ballor & Keesey, 1991). Based on these findings it was hypothesized that the women with the highest initial levels of body fatness would lose the most body fat in response to the six month program of brisk walking.

This was not the case however, as initial percent body fat was not associated with a change in percent body fat levels. It was thought that this lack of relationship was possibly due to the fact that women with high initial body fat exercised less and therefore expended less energy than the women with lower initial values. A regression analysis of initial percent body fat versus energy expenditure per kilogram of body weight clearly shows that this was not so (Figure 4-3).

It is possible that initial body fat levels were not related to change in percent body fat because women in this study were not obese. Andersson et al, (1991) found that body fat loss was confined to the most obese subjects while lean women did not lose significant amounts of fat. Obesity is denoted by a body mass index greater than 30 (Bray, 1989). The average body mass index of the women involved in the present study was 29.4 $\pm$ 6.1, which demonstrates that on average they were not obese. This lack of obesity may,

therefore, explain why the women's initial body fat levels were not related to change in percent body fat.

Influence of Initial Waist to Hip Ratio on Changes in Percent Body Fat

Initial body fat distribution has been found to be associated with a change in percent body fat. Women with high initial waist to hip ratios have been found to lose a greater amount of weight and percent body fat than women with lower initial values (Casimirri et al., 1989).

This was not the case with the women in our study. Initial waist to hip ratio was not found to be associated with a change in percent body fat. A comparison between the two studies show that the women involved in the Casimirri et al, (1989) study were more obese than the women involved in the present study. The average body mass index of the women involved in the Casimirri et al, (1989) study was 33.9 ± 4.5 while the average of the women in our study was 29.4 ± 6.1. The fact that our women were not obese may be the reason why the initial waist to hip ratio was not related to a change in percent body fat.

7) Summary

The purpose of this study was to examine the effects of a six month brisk walking program on the total body fat, body fat distribution, muscle mass and the relationship between initial fatness/body fat distribution and fat loss, in postmenopausal, mildly hypercholestrerolemic women. It was hypothesized that the walking program would lead to a significant loss in total body fat and

increase in muscle mass which would relate directly to the amount of energy expended by the subjects during the study. It was thought that the women with the highest initial levels of body fatness and waist to hip ratio would lose the most body fat and have the greatest change in body fat distribution. It was also hypothesized that the subjects would lose equal amounts of upper and lower body fat as a result of the walking program.

Participants had to be 55 years of age or older, nonsmokers, clinically healthy, sedentary or minimally active and not on medications to control serum lipid levels. Serum cholesterol levels had to be between 6.0 to 8.0 mmol/L. Women with triglyceride levels greater than 4.2 mmol/L were excluded from the study as well as women receiving estrogen replacement therapy. Forty women met the screening criteria and were included in the study. The forty subjects were randomly assigned to either a walking group (n=24) or a control group (n=16). Participants in the walking group took part in a six month program of walking which progressed to five times per week, for 53.4 minutes, at an exercise intensity of 54% heart rate reserve. The controls were asked to remain sedentary or minimally active for the same time period. Body composition measurements were taken at the zero, twelve and twenty-four week time intervals. Body composition was assessed by extensive anthropometry which included skinfold measurements, girths, breadths, lengths, height and weight in accordance with procedures outlined by Ross, Marfell-Jones and Stirling, (1982). The anthropometric measurements were used to determine the waist to thigh, waist to hip and subscapular to tricep skinfold thickness ratios. Percent body fat was calculated from skinfold measurements according to the equation of Jackson, Pollock and Ward, (1980). Three day diet records were also completed at twelve week intervals throughout the investigation so that dietary makeup and intake could be monitored and changes determined. At the end of the study each of the walkers completed a submaximal steady state test on the

treadmill to determine the energy cost of walking in order to predict the weekly total energy costs of the walking program. The relative change in each of the dependent variables was calculated by subtracting week zero value from the week twenty-four value.

At the end of the 24 week study paired t-tests were used to analyze changes within the groups while unpaired t-tests of the change values were used to determine between group changes from the 0 to 24 week period. Correlation coefficients were used to test for covariance between independent variables while simple regression was used to test for significant relationships between independent and dependent (change) variables. If significant relationships were found to exist ANCOVA was used to control for the influence of the covariate so that the true effect of the walking program on the dependent variable could be determined.

8) Conclusions

As a result of this investigation which studied the effect of a six month brisk walking program on the body composition and body fat distribution of mildly hypercholesterolemic women after menopause a number of conclusions are drawn and stated below.

1) The six month program of brisk walking was well tolerated and well suited to the abilities of the older women as demonstrated by the low attrition rate.

2) The walking program was of sufficient intensity (average of 54% heart rate reserve) to improve the fitness levels of the participants. The majority of the 10% increase in VO2 max can be accounted for by a loss of fat, while the remainder of the increase can be attributed to other physiological improvements which occurred as a result of the walking program.

3) The average weekly energy expenditure of 6366 kilojoules was sufficient to cause a small but significant decrease in the body weight and body mass index of the walkers. The average weight loss by the walkers was less than what would be expected based on the total energy cost of the program. Energy overcompensation or a compensatory decrease in energy expenditure the rest of the day may explain this discrepancy.

4) The first hypothesis that a six month program of walking could lead to a significant loss in total body fat relating directly to the amount of energy expended was rejected based on the findings of the present study. Although the walking program positively altered weight and body mass index it was unsuccessful in decreasing percent body fat. Possible explanations for this include wide variability in body fat measurements and small group sizes, and deep visceral fat loss as opposed to subcutaneous fat loss. A lack of significant relationship between energy expended by the walkers and change in percent body fat was also found to occur. A compensatory decline in the physical activity during the rest of the day of the walkers was considered the best possible explanation for this lack of relationship.

5) The hypothesis that a significant increase in muscle mass would occur as a result of the six month walking program was not supported. Muscle mass did not increase significantly as a result of the walking program perhaps due to the lack of sufficient weight bearing or strength training activities.

6) Based on the identical decrease in the walkers' waist and hip girth measurements, the nonsignificant change in waist to hip ratio and the trend toward fat loss in the other girth and skinfold measurements it was concluded that fat loss occurred equally from

the upper and lower body regions. Therefore, despite the statistical finding that the walkers' waist girth decreased significantly more than the hip girth, the original hypothesis that fat loss would occur equally from the upper and lower body regions was accepted.

7) The hypothesis that the women with the highest initial percent body fat and waist to hip ratio would lose the most body fat and have the greatest change in body fat distribution was rejected based on the findings of the present study. Initial percent body fat and waist to hip ratio were not significantly associated with the changes in percent body fat.

8) The final hypothesis that the reduction in body fat, change in body fat distribution and increase in muscle mass would relate directly to the subjects' increase in cardiovascular fitness levels was only partially proven The walkers' 10% increase in VO_2 max was only found to be significantly related to their decrease in percent body fat.

9) Recommendations

The following general recommendations are made on the basis of this current study:

1) A more exact measurement of additional activities should be included in future studies in order to determine whether or not compensatory reduction of physical activity during the remainder of the day is a confounding variable which influences weight loss or decreased percent body fat.

2) Larger group sizes or more homogeneous subjects should be used in order to overcome or avoid the problems associated with individual variability and its ability to conceal significant results.

3) Programs of a longer exercise duration should be used in the future in order to increase the likelihood that positive alterations in body composition and body fat distributions would occur.

4) There is the need to conduct a study incorporating an exercise program for obese women with a wide variability in waist to hip ratios, to determine whether different body fat distributions affect the ability of individuals to change their weight and percent body fat.

5) A longitudinal study incorporating a diet and exercise program should be designed in order to determine the effects a large energy deficit has on body composition and body fat distribution of women after menopause.

6) Walking programs which last one hour a day, five days a week, for at least six months should be prescribed by Fitness professionals in order to help women who are sedentary and post menopause to lose a small, but significant amount of body weight and to significantly increase their aerobic fitness. If the women are initially unable to walk for one hour they can gradually progress to this duration by walking for 20 minute increments.

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