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Title: Energy Need, Nutrient Intake, Fitness, Body Composition, and Health Risk Factors in Women With Childhood and Adult-Onset Obesity Before and After a 9-Month Nutrition Education and Walking Program

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The effect of a 9-month nutrition education and walking program on energy need, body composition, nutrient intake, nutritional status, aerobic fitness, and health risk factors was determined in 7 women with childhood-onset obesity (CO) and 8 women with adult-onset obesity (AO). Data were collected before and after the 9-month program while particpants were on a 3-week controlled, weight-maintaining metabolic diet. Need for total calories increased by 2.9% for CO and 3.1% for AO from pre to post ($p=.09$) whereas calories for resting metabolic rate (RMR) decreased 3.6% for CO and 2.8% for AO ($p<.05$). Body weight decreased from 98 $\pm$ 12 to 93 $\pm$ 7 kg in CO and from 93 $\pm$ 14 to 90 $\pm$ 15 kg in AO ($p<.05$), with 93% of the loss as fat tissue. Time spent in light and moderate activity increased from pre to post ($p<.01$). The increase in activity more than compensated for the decrease in RMR, contributing to an overall increase in daily energy need. Over the 9 months % kcals as fat assessed from food records decreased from 46 $\pm$ 4 to 29 $\pm$ 2% for CO and from 42 $\pm$ 6 to 36 $\pm$ 6% for AO. Though total calorie need did not decrease pre to post, calorie intake decreased 38% for CO and 10% for AO. Despite reduced calorie intake, both groups

maintained or increased their intakes of iron and vitamin B6. Change in dietary fat was related to age of onset of obesity but weight change was not. However, weight change was correlated with body fat distribution ($r=-0.67$, $p<.01$). Those women with predominately upper body fat lost more weight than those with predominately lower body fat. Fitness improved in both groups, with VO2 max increasing by 8% for CO and 7% for AO ($p<.01$). Total plasma cholesterol (TC) decreased 11% for CO and 4% for AO ($p<.01$). LDL-C decreased similarly while no significant change occurred in HDL-C or triglycerides. Resting systolic blood pressure (SBP) did not change significantly, while resting diastolic blood pressure (DBP) decreased 4% and 5% for CO and AO, respectively ($p<.05$). During exercise at an intensity close to 50% of VO2 max, SBP decreased 3-4% ($p>.05$) while DBP decreased 9-11% ($p<.001$). Area under the glucose tolerance curve decreased 43% for CO and 21% for AO ($p=.07$). Change in TC was most highly correlated with change in % kcals from fat ($r=0.68$, $p<.01$) while change in resting SBP and DBP was most highly correlated with change in VO2 max ($r=0.64$, $p<.01$). Change in body weight was not significantly related to change in any of the risk factor variables. These results suggest that changes in dietary and activity habits will improve health risk factors in women with obesity without the need for drastic weight loss.

ENERGY NEED, NUTRIENT INTAKE, FITNESS, BODY COMPOSITION, AND HEALTH RISK FACTORS IN WOMEN WITH CHILDHOOD AND ADULT-ONSET OBESITY BEFORE AND AFTER A 9-MONTH NUTRITION EDUCATION AND WALKING PROGRAM

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CHAPTER I

INTRODUCTION AND PURPOSE

Current recommendations for prevention and treatment of elevated blood lipids, hypertension and glucose intolerance, all risk factors for coronary heart disease (CHD), call for decreases in dietary fat and cholesterol, participation in regular aerobic exercise, and attainment of ideal body weight (Berger & Berchtold, 1982; American Heart Association, 1984; Dietary Guidelines for Americans, 1985; Gotto et al, 1984; National Cholesterol Education Program Expert Panel, 1988). Recommendations for weight control in people with mild or moderate levels of obesity currently call for the combined use of exercise, moderate calorie restriction and behavior modification (American College of Sports Medicine, 1983; Weinsier et al, 1984; Stunkard, 1984; Brownell, 1984). Further, controlling body weight by permanent lifestyle change rather than temporary measures is highly recommended (Weinsier et al, 1984; Hirsch, 1985).

Consuming a diet low in fat with the quantity of food freely chosen results in consumption of fewer calories (Duncan et al, 1983). No calorie compensation occurs and weight loss results when consuming a

low-fat diet over time (Lissner et al, 1987). Because of this, a combination of the above recommendations could be that the best approach to weight control is one that combines an habitual low-fat dietary intake with regular aerobic exercise. Though this approach as a means for reducing and controlling body weight might find widespread agreement and support, there is considerable disagreement about the goal, eg. what is considered the ideal weight one should achieve by a program of weight reduction. Recommendations range from reducing weight to no more than 10% above the 1959 Metropolitan Relative Weight (Manson et al, 1987), to no more than 20% above desirable weight based on either the 1959 or 1983 Metropolitan Height/Weight Tables (Burton et al, 1985), to not treating obesity at all, especially in women (Wooley & Wooley, 1984). There is also an increasing number of researchers who are recommending that weight goals be determined individually depending on family, medical, and weight history and the presence of health risk factors (Knapp, 1983; Callaway, 1984; Van Itallie, 1985).

There is ample evidence to suggest that there are fewer health risks associated with obesity for women than for men (Montoye et al, 1966; Lewis et al, 1974; Keys, 1980; Taylor et al 1981; Bjorntorp, 1985) while at the same time, the social pressures to achieve thinness are much more strongly directed toward women than men, frequently going well beyond the concerns for health (Wooley & Wooley, 1984; Wadden & Stunkard, 1985). Accordingly, weight loss is attempted much more on the part of women than men (Wadden & Stunkard, 1985; Wooley & Wooley, 1984). Dieting is most often used, and it has a dismal rate of success even when combined with exercise and/or behavior modification (Stunkard

& McLaren-Hume, 1959; Wing & Jeffery, 1979). Lack of success may, at least in part, be due to the reduction in resting metabolic rate and total caloric need that results following weight loss (Schutz et al, 1984; Ravussin et al, 1985). In addition, weight loss diets are generally inadequate in nutrient intake (Fisher & Lachance, 1985; Smoller et al, 1988), and in their most extreme forms may cause death due to loss of tissue nitrogen and potassium (Van Itallie & Abraham, 1985).

Proponents of various theories of weight regulation suggest that drastic reduction of body weight below a certain point sets in motion strong physiological "restorative" forces which, in a sense, "compel the organism" to reestablish the pre-reduction body weight (Nisbett, 1972; Keesey, 1980; Hirsch & Leibel, 1984). Those people with obesity beginning in childhood may especially not be able to attain and maintain a weight anywhere near what might be considered ideal (Grinker & Hirsch, 1972; Bjorntorp et al, 1975; Krotkiewski et al, 1977). Because our dietary environment is calorically very rich and our level of physical activity very low, the weight we so easily maintain and revert to following drastic weight loss measures is higher than it might be with a less calorically-dense diet and more physical activity (Sclafani, 1980; Brownell, 1984).

Attainment of so-called "ideal or desirable" weight is not necessary to normalize CHD health risk factors. A moderate loss of body weight, on the order of 10-20 kg, normalizes blood pressure (Stamler et al, 1980; Eliahou et al, 1981), hypertriglyceridemia and

blood cholesterol (Olefsky et al, 1974; Stamler et al, 1980) while many subjects remain well above 120% of "ideal" body weight.

If one stands back and looks at all the pieces of evidence together, it appears that, at least for women who are mildly to moderately obese, a "best" body weight might be one that results from habitually consuming a low-fat diet, only moderately decreased in calories, and habitually participating in regular aerobic exercise. This may be true whether the weight attained fits the recommended weight for height or not. This approach would seem to maximize the opportunity to reduce or prevent health risk factors without the detrimental effects generally associated with drastic dieting for weight loss. Whether the body weight achieved would be socially acceptable or not is an issue beyond the scope of the present work.

The purpose of the research project to be described herein was to offer a nutrition education and walking program to women with moderate obesity over a period of 9 months. No goals were to be set for body weight change. The nutrition education program was designed to focus primarily on developing the habit of consuming a low-fat diet, the walking program was offered to help develop the habit of regular activity of moderate intensity. Women with childhood-onset of obesity were to comprise half the group studied, those with adult-onset obesity the other half, so as to compare the effects of this kind of program in women differing in age of onset of obesity. Data were collected on factors pertaining to nutrition, fitness, body composition, energy metabolism, and coronary heart disease risk.

The specific objectives of this project were the following:

1. Compare the effects of a 9-month nutrition education and exercise program on the following parameters in women with childhood and adult-onset obesity:

   a) Nutritional status: calories to maintain body weight, dietary intake of calories and nutrients, biochemical status of specific nutrients

   b) Aerobic fitness: maximal aerobic capacity, cardiovascular response during submaximal exercise

   c) Body composition: body weight, percent body fat, lean body weight

   d) Bioenergetics: resting metabolic rate

   e) Health risk: blood lipids, blood pressure, glucose tolerance

2. Identify clinical measures to evaluate health risk and "best" body weights for women with both childhood and adult-onset obesity.

For each of these individual factors the null hypothesis to be tested was two-fold: 1) there would be no difference after the program

compared to the beginning (no pre to post change), and 2) there would be no difference between the women with childhood-onset obesity and those with adult-onset obesity.

CHAPTER II

LITERATURE REVIEW

This review of the literature is specific to certain issues related to obesity and its treatment. First, the discussion of health risks associated with obesity is limited to those related to coronary heart disease (CHD). While other health risks are certainly associated with obesity (Bray, 1985), CHD is the leading cause of death in the United States for both men and women (Stamler, 1980) and obesity is closely associated with elevated blood lipids, hypertension and glucose intolerance, all major risk factors for CHD (Van Itallie & Abraham, 1985). Second, this discussion is directed to issues related to mild and moderate levels of obesity. Morbid obesity (more than 100% above relative body weight) carries with it greatly increased health risks (Kral, 1985; Bray, 1985) but a very small proportion of the obese population, probably less than 0.5%, falls into that category (Stunkard, 1984; Wadden & Stunkard, 1985). Third, literature related to treatment of obesity with diet and exercise are included, whereas information on drug and surgical treatment is not. Diet is by far the most used treatment for obesity (Wing & Jeffery, 1979) and a combination of diet and exercise the most highly recommended method for treatment of mild and moderate levels of obesity (Weinsier et al, 1984; Brownell, 1984). Fourth, although the literature using animal models to study obesity is extensive, with only a few exceptions (where human data are not available), the literature reviewed is that pertaining to

research done with human subjects.

With those constraints in mind, the following literature review covers incidence and definitions of obesity, health and social risks associated with obesity, dietary treatment of obesity, theories of weight regulation, and the effect of lifestyle change, eg. diet and exercise, on health risk factors and body weight.

Incidence and Definition of Overweight and Obesity

Based on information from the latest National Health and Nutrition Examination Survey (NHANES II), conducted in 1976–80, 14.5 million American men (22.8%) and 18.1 million women (25.8%) are estimated to be overweight. About 5.3 million men (8.4%) and 6.2 million women (8.8%) meet the criteria for being severely overweight (Van Itallie & Abraham, 1985).

The terms overweight and obesity are used interchangeably in the literature and there is no agreed upon definition for either term. The recent NIH Consensus Conference on Obesity described obesity as "an excess of body fat" (Burton et al, 1985) yet chose Body Mass Index (weight in kilograms divided by the square of height in meters) as the preferred indicator of weight status rather than some other more direct measure of body fat. Because weight in excess of a standard weight for height is considered to be due primarily to fat weight (Abraham et al, 1983; Van Itallie, 1985), and height and weight are more easily obtained than an estimate of body fat, obesity tends to be defined in

terms of a certain percentage overweight whether described as Body Mass Index (BMI) or compared to height/weight tables. However, in the NHANES surveys a separate definition of overweight and obesity was made.

In NHANES II, as well as NHANES I (conducted 1971-74), overweight was defined as having a BMI equal to or greater than that of the 85th percentile for the reference group of men and women aged 20-29 years. Severely overweight was designated when BMI was equal to or greater than that of the 95th percentile for the same reference group (Abraham et al, 1983; Van Itallie & Abraham, 1985). BMI was defined as the ratio of weight in kg to the power of height in meters, with the formula for men using meters squared and for women meters to the 1.5 power. Recently, recommendations for BMI for both men and women have been based on height in square meters (Burton et al, 1985). Using this formula for the NHANES II data, men with a BMI greater than 27.8 were designated overweight, a BMI greater than 31.1 indicated severe overweight. For women the designations were 27.3 and 32.3 for overweight and severely overweight, respectively (Van Itallie, 1985). When using the Metropolitan Life Insurance Tables as a standard for defining overweight, these BMI values correspond closely to 20% and 40% above the 1983 Metropolitan weight for height standard (Burton et al, 1985).

In NHANES I, the definition for obesity differed from that for overweight (Abraham et al, 1983). A person was considered obese if the sum of their triceps and subscapular skinfold measurements were equal

to or greater than the 85th percentile for the 20-29 year old reference group. Severe obesity was defined as the sum of triceps and subscapular skinfold measurements equal to or greater than the 95th percentile. Among the group designated overweight, a portion were found to be obese with others not meeting that criteria. An additional group was found to be obese but not overweight. Data on incidence of obesity are not yet available from NHANES II. Based on NHANES I information, however, an additional 6.6% of men and 6.1% of women were not overweight but were classified obese based on skinfold measurements.

Using the combined measurements of BMI and skinfold thickness, 5 categories were developed from NHANES I data based on cross-classifications of these indicators of overweight and obesity (Abraham et al, 1983; Van Itallie & Abraham, 1985). The 5 categories include: underweight and lean (Group 1), average weight and fatness (Group 2), obese and not overweight (Group 3), overweight and not obese (Group 4), and overweight and obese (Group 5). Using these categories and other NHANES I data, the relationship of overweight and obesity to systolic and diastolic blood pressure and to serum cholesterol, all of which are risk factors for premature coronary heart disease, was examined.

Relationship of Weight to Coronary Heart Disease (CHD) Risk Factors

Table 1 outlines the percentage of people participating in NHANES I who had elevated cholesterol and blood pressure (BP) measurements in each of the 5 weight categories described above (Van Itallie & Abraham,

Table II.1. Age adjusted percentage of NHANES I population with elevated serum cholesterol and blood pressure. (Values from Van Itallie & Abraham, 1985).

	Serum Cholesterol (≥ 260mg/dL)*	Diastolic BP (≥ 95mmHg)	Systolic BP (≥ 160mmHg)
Men			
Underweight & lean	7.1%	10.2%	7.2%
Ave weight & ave obese	21.2%	10.6%	6.0%
Obese not overweight	23.6%	8.7%	5.8%
Overweight not obese	24.0%	26.1%	9.9%
Overweight & obese	30.2%	27.7%	15.3%
Women			
Underweight & lean	15.8%	4.9%	6.6%
Ave weight & ave obese	23.4%	5.8%	5.5%
Obese not overweight	27.8%	8.8%	7.9%
Overweight not obese	34.4%	16.3%	12.5%
Overweight and obese	26.6%	23.5%	15.9%

* 260 mg/dL = 6.72 mmol/L (conversion factor: mg/dL x 0.02586 = mmol/L)

1985). Higher levels of serum cholesterol and blood pressure occurred more frequently in overweight and obese men and women. However, elevations also occurred more frequently in those who were overweight but not obese. This has raised at least two questions. One is whether or not the upper body skinfold measurements used to determine obesity (triceps and subscapular) included all who are obese, eg. what about those with predominately lower body fat? The other is whether larger body build and increased muscularity are risk factors for susceptibility to CHD independent of body fatness (Van Itallie & Abraham, 1985). These authors and others (Burton et al, 1985) have urged more research into these questions.

In data from NHANES II, relative risk of hypertension, elevated serum cholesterol, and diabetes have been related just to overweight as data on incidence of obesity by skinfold measurements are not yet available (Van Itallie, 1985). Relative risk is based on the ratio of the occurence of the risk factor in overweight persons compared to those who are non-overweight. For hypertension, the relative risk for those in the overweight category was 3 times that in the non-overweight. For elevated serum cholesterol (>250mg/dL), the relative risk for overweight was 1.5 times that for non-overweight. The relative risk for diabetes for overweight was 2.9 times that for non-overweight. Based on the NHANES I and II data it is clear that with overweight comes increased risk for hypertension, elevated serum cholesterol and diabetes. However, it is also clear that not every overweight or obese person has these symptoms. In fact, in NHANES I, two-thirds or more of those who were overweight and/or obese did not

have elevated serum cholesterol or blood pressure.

Other investigations have also found associations between weight and CHD risk factors. Blood lipid concentrations were found to increase with increasing relative weight in the Framingham Study (Kannel et al, 1979; Garrison et al, 1980). Blood cholesterol levels also increase with greater body fat (Matter et al, 1980). An exception to this is the high density lipoprotein cholesterol fraction (HDL-C) which tends to be negatively correlated with body weight (Kannel et al, 1979; Garrison et al, 1980) and body fat (Garrison et al, 1978; Matter et al, 1980). Blood pressure is generally found to be positively associated with body weight increasing fairly linearly with increasing body weight (Chiang et al, 1969; Kannel & Gordon, 1979; Berchtold et al,1981; Havlik et al, 1983). In studies specifically dealing with women, the incidence of hyertension is also positively correlated to body weight (Rimm et al, 1975) and to body fat (Noppa et al, 1980).

A strong association exists between obesity and diabetes, hyerinsulinemia and hyperglycemia as well (West, 1978). In the Framingham study the incidence of diabetes rose dramatically at relative weights higher than 130% for both men and women with the incidence being slightly higher in men than women at each relative weight (Kannel et al, 1979). In his survey of 73,532 women participating in TOPS (Take Off Pounds Sensibly) groups, Rimm et al (1975) found an association between obesity, age and adult-onset diabetes. In young adults the incidence of diabetes did not increase much until reaching weights above 150% of ideal. In the older adults

incidence of obesity was higher at all weights with the occurence of adult-onset diabetes increasing dramatically at weights above 120% of ideal. O'Sullivan (1982) longitudinally studied women with transient gestational glucose intolerance and found that after 16 years the incidence of diabetes was almost twice as high for those who were obese compared to non-obese. In contrast, a control group with normal glucose tolerance during and after pregnancy had no significant difference in diabetes incidence between obese and non-obese. His results suggest that obesity is a risk factor for diabetes for only a specific sub-set of the population.

Though the general trend with increasing body weight is to see a higher incidence of CHD risk factors, there is considerable disagreement as to whether body weight is independently associated (cholesterol, blood pressure, glucose tolerance corrected for) with the resultant CHD itself (Hubert et al, 1983; Keys et al, 1984; Brunzell, 1984; Van Itallie & Abraham, 1985). Most agree that severe overweight carries with it many health complications and increased mortality but controversy swirls around the issue of whether mild and moderate degrees of overweight are also associated with such risks (Keys, 1980; Knapp, 1983; Manson et al, 1987).

Relationship of Weight and Coronary Heart Disease Mortality

A wide variety of issues are involved in the controversy regarding body weight and health risks. Ideal weight for greatest longevity may not correspond with the best weight for health indicators like blood

pressure, serum lipids, and glucose tolerance. Age plays a role in the risk associated with body weight. People with certain subtypes of obesity based on fat cell number and size as well as pattern of fat distribution may be more at risk for health complications than others. The health risks associated with obesity appear to be substantially more detrimental for men than for women. These issues will be discussed below.

Interest in the effect of weight status on longevity has been long standing, coming initially from life insurance companies in the process of contructing actuarial tables (Simopoulos & Van Itallie, 1984). The Build and Blood Pressure Study (1959) and Build Study (1979) have together provided information on weight and mortality for over 8 million people in the United States and have been the basis for development of the 1959 Metropolitan Life Desirable Weight Tables (Metropolitan Life Insurance Company, 1959) and the 1983 Metropolitan Height and Weight Tables (Metropolitan Life Insurance Company, 1983), respectively. Mortality risk was found to be lowest for the groups with weights 5-15% below and 5-15% above the average weight for this insured population with mortality increasing for those in the lowest and highest weight categories. The resulting relationship was thus U- or J-shaped (Manson et al, 1987). In the 1979 Build study, average weights were higher than those in the 1959 study resulting in lowest mortality rates occurring in people weighing more than they had in the 1959 study. As a result, the 1983 Height and Weight Tables list weights from 0-13% higher than the 1959 tables (Metropolitan Life Insurnce Company, 1983). This increase in what is considered

"desirable" weight has stirred a good deal of controversy and initiated criticism of the methodology used to relate body weight to longevity (Knapp, 1983; Simopoulos & Van Itallie, 1984; Harrison, 1985).

In both Build Studies (1959; 1979) the relative mortality risk for women was considerable lower than for men. In the 1979 study for women between 15% underweight and 45% overweight, mortality risk ranged from 93% to 109%. For men within that same weight range, mortality risk ranged from 95 to 139%. Above 45% overweight risk increased for both but at a considerably higher rate for men than women.

Another study, conducted by the American Cancer Society, found associations between weight and mortality that were similar to those in the Build Studies (Lew & Garfinkel, 1979; Manson, 1987). Lowest mortality from all causes was found to occur in men with weights 90-109% of the average weight in this population. For women, lowest mortality was associated with weights 80-119% of average. At weights greater than 120% of average, relative mortality risk was similar in men and women. However, in all weight categories the death rate in women was half that of men up to age 70. Absolute mortality risk due to overweight is clearly less in women than men.

A number of other studies have investigated the relationship between body weight and mortality and found either no relationship at all or one in which the relationship is U- or J-shaped with increased mortality rising only at the extremes of under and overweight (Simopoulos & Van Itallie, 1984; Harrison, 1985; Feinleib, 1985).

These weight and mortality curves are especially flat for women. This evidence has prompted Ancel Keys to conclude "there is not acceptable evidence that relative body weight has any relevance to future health for women in the middle 80% of the relative weight distribution" (Keys, 1980).

Relationship of Weight Change to Decreased CHD Risk

Weight change has a positive correlation with change in CHD risk factors. In the Framingham Study, a 10 unit change in relative weight was associated with a change of 11.3 mg/dL in serum cholesterol, 6.6 mmHg in systolic blood pressure, and 2.5 mg/dL in glucose for men. The effects of weight change on these risk factors were less for women being 6.3 mg/dL for serum cholesterol, 4.5 mm Hg for systolic blood pressure, and 1.3 mg/dL for glucose (Ashley & Kannel, 1974). Noppa (1980) reported on weight change and corresponding risk factor change in a population of 1302 women in Sweden. In that study, a change of 10 kg in body weight corresponded to a change of 0.15 mmol/L (5.8 mg/dL) in serum cholesterol, 0.26 mmol/L (23 mg/dL) in triglycerides, 3.9 mm Hg for systolic blood pressure, 2.7 mmHg for diastolic blood pressure, and 0.16 mmol/L (2.9 mg/dL) for fasting glucose. In studies dealing specifically with weight reduction in the obese, accompanying decreases have been observed in serum cholesterol (Berkowitz, 1964; Galbraith et al, 1964) and blood glucose levels (Newburgh, 1942; Berkowitz, 1964).

When most of us think of needing to lose weight to improve these health risk factors, the assumption is that ideal or desirable body

weight for height must be reached. However, only moderate amounts of weight loss, in the order of 10-20 kg, have been found to normalize blood lipids, blood pressure, and glucose tolerance in overweight and obese subjects even though they remain at weights well above their so-called "ideal". Stamler et al (1980) found that with an average 12 kg weight loss, systolic and diastolic blood pressure were normalized in 67 hypertensive men, all of whom were 15% or more overweight. Serum cholesterol also fell 26 mg/dL, a 10% decrease. Relative weight decreased only 6%, ending at 122%, a level still considered overweight. Eliahou et al (1981) also found that achieving normal weight was not necessary for achieving normal blood pressure in 46 patients who were overweight and hypertensive. Of the 38 people in this study who achieved normal blood pressure following a weight loss of 5% or more, 82% were still >10% overweight, and 29% were still >30% overweight. Olefsky et al (1974) found similar results in men and women with relative weights ranging from 1.00 to 1.71, most of whom had hyperlipoproteinemias. Following a 11 kg weight loss, their subjects had a 44% decrease in fasting serum triglycrides, a 21% decrease in serum cholesterol and a 37% and 12% decrease in plasma insulin and glucose response, respectively, during the oral glucose tolerance test. Improvements in these risk factors were similar among the subjects regardless of their initial or final weight.

Van Itallie (1985) states that "longevity surely is not the most desirable criterion for assessing the health implications of overweight and obesity" and suggests that weights at which risk factors are within normal ranges might be considered optimal. If that were to actually be

considered a valid criteria, it would have to be recognized that most overweight and obese people already have normal values for CHD risk factors as seen in the NHANES I and II data. Many researchers are advocating an approach to desirable weight that is based on individual history and the actual presence of risk factors (Keys, 1980; Knapp, 1983; Callaway, 1984; Van Itallie & Abraham, 1985; Harrison, 1985; Feinleib, 1985). Recommendations of the NIH Consensus Conference on Obesity included that more liberal weight guidelines might be used in the absence of CHD risk factors and family history of risk (Burton et al, 1985) although the Consensus Panel felt anyone 20% or more overweight should be treated for weight loss.

Subtypes of Obesity More at Risk for CHD

The age of onset of obesity plays a role in CHD risk. Contrary to what we might first think, weight gain in adulthood carries more CHD risk. A much higher prevalence of hypertensive vascular disease and cardiovascular renal disease has been found in people who gain weight as adults than in those classified overweight as children who maintained that status as adults (Abraham et al, 1971). Obesity beginning in childhood is associated with high fat cell number (hyperplastic obesity) accompanied sometimes with enlarged fat cells, whereas adult-onset obesity is more associated with enlarged fat cells (hypertrophic obesity) but not increased fat cell number (Hirch & Knittle, 1970; Brook et al, 1972; Salans et al, 1973). The enlarged fat cells characteristic of hypertrophic obesity or weight gain in adulthood are associated with hypertriglyceridemia (Albrink et al,

1962; Bjorntorp & Sjostrom, 1971); hyperinsulinemia, insulin resistance, impaired glucose tolerance, and diagnosed adult-onset diabetes (Salans et al, 1968; Bjorntorp & Sjostrom, 1971; Bjorntorp et al, 1973; Sims et al, 1973); and autopsy-verified atherosclerosis (Bjurulf, 1959).

More recently, location of body fat has been confirmed as an important indicator of health risk associated with obesity (Bjorntorp, 1985). Abdominal obesity (also called upper body, android, and male-type obesity) is consistently related not only to CHD risk factors (Kissebah et al, 1982; Krotkiewski et al, 1983; Kalkhoff et al, 1983; Hartz et al, 1983; Foster et al, 1987) but to CHD and mortality as well (Larsson et al, 1984; Lapidus et al, 1984). Bjorntorp (1985a) has pointed out that this type of obesity is similar to adult-onset or hypertrophic obesity as the abdominal fat cells are greater in size rather than number. At the same relative degree of overweight Krotkiewski et al (1983) found significantly less body fat, but significantly higher blood pressure, triglycerides, fasting plasma insulin and glucose, and insulin and glucose response during an oral glucose tolerance test, in obese men than in obese women. The men carried their fat predominately at the waist while women's fat was more predominate around the hips and thighs. Among women in ths study, those with predominatly upper body fat (abdominal obesity) had higher values for blood pressure, triglycerides, insulin and glucose than those with predominately lower body fat (femoral-gluteal obesity). However, even those women with upper body fat did not have values for these CHD risk factors that were as high as those for the men. This

has led Bjorntorp to comment that when obesity is equal in men and women, the men "are sicker" (Bjorntorp, 1985a).

It appears that weight gain in early adulthood carries the highest risk for premature cardiovascular mortality (Van Itallie, 1979). The NHANES II data demonstrate much greater risk for hypertension and hypercholesterolemia in overweight adults aged 20–44 years than those 45–74 years (Van Itallie, 1985). Andres (1985; 1985a) has reviewed several weight and mortality studies and concludes that small amounts of gradual weight gain over the decades is associated with lowest mortality risk and that overweight is much less risky after age 40 and especially after age 50.

In summary, there are health risks associated with obesity. However, the majority of the population that has obesity does not, in fact, have elevated blood cholesterol, blood pressure, or glucose intolerance. It appears that the most at risk person is the male who gains considerable weight during the young adult years and sustains that increased weight through the 4th and 5th decades. Obese women are also at risk, but much less so than men. Of the obese population, women with predominately lower body fat have the lowest incidence of CHD risk factors. Weight loss decreases the health risks associated with obesity, but attainment of "ideal" weight for height is not necessary to achieve normal blood lipids, blood pressure, or glucose tolerance.

The Social Risk of Obesity

The discrimination aimed at obese individuals is almost universal (Astwood, 1962; Mayer, 1968; Wadden & Stunkard, 1985) and is so ingrained culturally that people of all ages including very young children demonstrate such attitudes (Lerner & Schroeder, 1971; Staffieri, 1972; Wooley & Wooley, 1984). A 1985 Gallup poll found that 90% of Americans would like to be thinner (Smoller et al, 1988), in spite of the fact that overweight affects only about 25% of the population (Van Itallie & Abraham, 1985). The pressure to be thin is particularly strong for women and far exceeds the concern for health (Garner et al, 1980; Wooley & Wooley, 1984; Wadden & Stunkard, 1985). In a nationwide survey, 56% of women aged 25-34 years were found to be dieting to lose weight (Fisher & Lachance, 1985). Women outnumber men 9 to 1 in weight loss programs and contribute a disproportionate amount of the millions of dollars spent on the diet and exercise industry (Wadden & Stunkard, 1985). This occurs in spite of the fact that the incidence of overweight and obesity is roughly equal in men and women (Van Itallie & Abraham, 1985) and that the health risks of obesity are less for women than for men (Van Itallie & Abraham, 1985; Harrison, 1985; Bjorntorp, 1985a).

Women's concerns about weight start early. In a study of 10th through 12th graders, half of the girls believed they were overweight and about 65% wanted to lose weight. The boys either were satisfied with their weights or thought they were too thin. Ironically, the

incidence of overweight was identical for both with 25% being only marginally overweight (Huenemann et al, 1966). The method of choice for weight loss is dieting, and for women it begins at an early age. Thompson and Schwartz (1982) found 40% of a sample of college women to demonstrate anorextic-like behaviors. Almost all of the 77 women in their study reported they were "always dieting" with the age of first diet occuring by 15 years. Another study done with Scandinavian women found that 80% had dieted by age 18 (Nylander, 1971).

Because social pressures toward thinness are so strong and because health risk factors do improve with weight loss, a great deal of time, effort and money has been directed toward weight loss by health professionals as well as those who are not so professional. The primary method used is dieting.

Dieting for Weight Loss

Dieting or some degree of caloric restriction is incorporated into almost all weight loss programs and attempts (Dwyer, 1980). The diets used range from the "sensible" (1200-1800 kcals/day) to those drastically restricting calories (400-1000 kcals/day) or containing no calories at all, in the case of fasting or starvation. Some restrict carbohydrate partially or completely to promote very rapid weight loss (Dwyer, 1980; Stern, 1983). Nutritional adequacy of these diets is very poor.

The Food and Nutrition Board (1980) states that it is "difficult

to assure nutritional adequacy of diets that are low in energy content (less than 1800-2000 kcal)". Nutritional analysis of 20 different popular weight loss diets has found levels of thiamin, vitamins B6 and B12, iron and zinc to be less than 70-80% of the RDA with calorie intakes ranging from 600-2000 kcal/day. No studies were located that have looked at actual nutrient intake of dieters. However, in nationwide surveys of women's diets, intakes of calcium, iron, magnesium, zinc, vitamin B6, and folacin are also found to be less than 70-80% of the RDA (Kurinij et al, 1986; Peterkin et al, 1986; Murphy & Calloway, 1986). With more than 50% of women in the US dieting, the low nutrient levels in weight loss diets may very well be responsible for the low intakes of several of these same nutrients in general population surveys.

In the study described in the present work, we were advocating a decrease in dietary fat rather than a decrease in calories per se. Because that kind of dietary change calls for eating more food of plant origin and fewer high-fat animal foods, the intake of two micronutrients, iron and vitamin B6, were of particular concern. These are nutrients which women already tend to consume in amounts lower than the RDA and they both are nutrients whose bioavailability is lower in plant foods than in animal foods (Kabir et al, 1983; Raper et al, 1984; Worthington-Roberts et al, 1988).

While low dietary intake may indicate a potential problem with nutritional status, biochemical indicators are also important to consider. Impaired iron status and iron deficiency anemia are problems

of particular concern for women prior to menopause. Iron status is assessed by measurement in blood of ferritin, percent saturation of transferrin, plasma protoporphyrin, hemoglobin, hematocrit, or mean corpuscular volume. When possible, a combination of 2 or more of these measurements is preferred. In the NHANES II survey, 9.8% of women aged 20–44 years had impaired iron status based on combined ferritin, % saturation of transferrin, and erythrocyte protoporphyrin measurements (Expert Scientific Working Group, 1985). Prevalence of anemia, defined as hemoglobin values below the 95% reference range, was 5.8% in women aged 25–44 years in the same NHANES II survey (Dallman et al, 1984). The biochemical status of vitamin B6 in women has not been determined on such a large scale as has iron. Plasma level of pyridoxal 5'–phosphate (PLP) is used as the primary vitamin B6 status indicator in blood, with urinary excretion of vitamin B6 metabolites also being used (Leklem & Reynolds, 1981; Shane, 1978). As with iron, a combination of methods to determine biochemicl status is preferred (Leklem & Reynolds, 1981). Plasma level of PLP has been found to be lower in women than men (Shultz & Leklem, 1981), perhaps reflecting the lower dietary intake of vitamin B6 in women compared to men (Chrisley & Driskell, 1979).

In spite of the wide variety of weight loss diets and the wide variety of dieters, there is only one goal: reaching ideal or desirable weight for height. The dismal failure of reaching that goal and being able to maintain it for any length of time (Stunkard & McLaren–Hume, 1959; Wing & Jeffery, 1979; Berchtold & Van Itallie, 1985) may lie in both the technique (temporary dieting) and the goal

(ideal weight for height). One problem inherent in going "on" a diet is that sooner or later one goes "off" the diet (Stunkard, 1984). Old habits are usually resumed and so is old weight. If for no other reason, dieting can be seen as ineffective because of this phenomenon. In addition, dieting in and of itself may physiologically set one up for regain of weight once the caloric restriction is ended. This may be due to the lowered calorie needs that result from weight loss and/or a biological response to semistarvation.

Calorie Need

Following weight loss, decreases in 24-hour energy need have been found to range from 9% to 40% in subjects who consumed very low-calorie diets in the range of 300-1100 kcals/day (Warnold et al, 1978; Bessard et al, 1983; Leibel & Hirsch, 1984; Ravussin et al, 1985; de Boer et al, 1986). These decreases in calorie need were found even with average weight losses in the range of 10 to 15 kg. All components of energy expenditure are affected by weight loss (Ravussin et al, 1985). These include resting metabolic rate (RMR), physical activity, and thermogenesis.

Resting metabolic rate comprises the largest component of total energy expenditure, about 70%, in people with and without obesity (Ravussin et al, 1982). People with obesity have greater RMRs than those without obesity (Hoffmans et al, 1979; Ravussin et al, 1982). This is because RMR is related primarily to fat-free mass and because people with obesity have a greater amount of fat-free mass than those

without obesity (James et al, 1978; Halliday et al, 1979; Hoffmans et al, 1979; Ravussin et al, 1982). Following mean weight losses of 10 to 15 kg, resting metabolic rate decreases by 9% to 29% in studies using diets containing less than 1100 kcal/day (Bray, 1969; Apfelbaum et al, 1971; Warwick & Garrow, 1981; Bessard et al, 1983; Welle et al, 1984; Ravussin et al, 1985; Hill et al, 1987; Van Dale et al, 1987). In studies using very low-calorie diets and exercise, decreases in RMR have ranged from 9% to 19% (van Dale et al, 1987; Hill et al, 1987). With higher calorie intakes, ranging from 1200-1800 kcal/day, and mean weight losses of 5 to 7.5 kg, others have observed no decreases in RMR when diet is combined with regular exercise, and non-significant decreases in RMR of 3-7% with diet alone programs (Lennon et al, 1985; Belko et al, 1987).

The decrease in RMR following weight loss is felt to be primarily due to the loss of fat-free mass which occurs during weight loss (Ravussin et al, 1985). Analysis of the composition of tissue lost during weight reduction has shown that starvation and severe caloric restriction (intake <1000 kcals/day) result in 30-40% of the weight lost coming from fat-free tissue (Buskirk et al, 1963; Passmore, 1964; Yang & Van Itallie, 1976; Krotkiewski et al, 1981). This is true whether the severe caloric restriction is accompanied by exercise (Hill et al, 1987; van Dale et al, 1987; Belko et al, 1987) or achieved by diet alone (Warnold et al, 1978; Dore et al, 1982; Bessard et al, 1983; de Boer et al, 1986). The commonly held belief that exercise will in some way lessen the effect of severe caloric restriction on fat-free tissue has not been demonstrated. Even mild to moderate caloric

restriction (500-1000 kcal deficit/day) can result in a loss of fat-free tissue comprising 10-20% of the weight lost (Goldman, 1963 Zuti & Golding, 1976; Thompson et al, 1979).

Other components of energy expenditure have also been observed to decrease following weight loss. Apfelbaum et al (1971) found that the cost of cycling, walking, and stair climbing decreased from 12-18% in obese subjects consuming a diet of 500 kcals/day. Ravussin et al (1985) found a decrease in energy needed for both thermogenesis and physical activity following weight loss in obese subjects who had consumed 300-1100 kcals/day. Bessard et al (1983) observed a 20% lower thermogenic response in obese women compared to controls prior to weight loss which decreased further to a level 35% lower than that of controls following an average 12 kg weight loss. The decrease in thermogenesis and energy cost of physical activity may account for about half of the reduction in caloric need following weight loss with the other half due to a decrease in RMR (Ravussin et al, 1985).

Schutz et al (1984) have suggested that the decrease in caloric need after weight loss is a reason for the regain in weight that usually follows a program of weight loss. Others strongly suggest that retention of fat-free tissue be maximized in any weight loss program to help prevent the decrease in calorie need that is generally observed (Ravussin et al, 1982; American College of Sports Medicine, 1983). Based on the evidence cited above, greatest fat-free mass is retained when calorie intakes stay above 1200-1800 kcals/day and exercise is included.

The rapid regain in weight following weight loss by dieting is very similar to that seen following semistarvation. In the Minnesota semistarvation study (Keys et al, 1950) conscientious objectors agreed to lose 25% of their body weight by consuming a calorie-restricted diet. Following refeeding, regain of weight was very rapid and weight returned very nearly to initial or control levels. At 33 weeks of refeeding body weight was at 105-109% of control weight, by 58 weeks it was at 102% of control weight. The authors state that this post starvation "obesity" or tendency to gain to a level above initial weight is typical and found in experimental and natural starvation situations. With time however, weights return very close to initial levels. Of interest is what happened to body composition during the refeeding period. While body weight at 33 weeks of refeeding was 105-109% of control, body fat was at 139% of control level. By 58 weeks body fat had decreased to 110% of control. Regain of body fat was disproportionately larger than gain of fat-free tissue. This rapid regain especially of body fat has also been demonstrated in Brownell's well-known "yo-yo" rats (Brownell et al, 1986). Rats were subjected to repeated bouts of calorie restriction and ad-libitum feeding. Following each period of weight loss, a little more weight than control levels was regained, and a higher proportion was fat. During each succeeding period of calorie deprivation it took longer to lose weight. If such evidence can be applied to humans, the prognosis is not good for those people who have lost and regained weight several times.

Along with the tendency for people to return close to their

original body weight following weight loss, is the tendency to return to initial body weight following weight gain. The overfeeding studies done by Sims et al (1973) demonstrated the difficulty of increasing body weight by 25% and the rapid loss back to initial weight with a return to normal calorie intake. Because of this strong tendency to "defend" one's body weight against drastic change either up or down, several theories of weight regulation have been suggested. These include the set-point theory, the fat-cell theory, and dietary-induced obesity (Brownell, 1984)

Theories of Weight Regulation

Based on the experimental evidence in humans (Keys et al, 1950; Sims et al, 1973; Johnson & Drenick, 1977) and animals (Keesey, 1980) that drastic weight change either up or down is not easy to accomplish and maintain, a set-point theory of body weight regulation has been proposed (Nisbett, 1972; Keesey, 1980; Keesey & Corbett, 1984). This theory is based on the observation that though body weight itself varies widely among individuals, for each person the coefficient of variation of body weight is very small (Keesey, 1980). When body weight is radically changed, eg. with drastic weight reduction, powerful restorative forces seem to function to return weight to its initial level (Hirsch & Leibel, 1984). One of the possible "restorative forces" is the enzyme adipose tissue lipoprotein lipase. This enzyme catalyzes the breakdown of triglycerides from plasma chylomicrons and very low density lipoproteins to free-fatty acids which recombine with glycerol to form storage triglycerides in fat

cells (Brunzell & Greenwood, 1983). The activity of this enzyme per fat cell is higher in obese subjects compared to lean controls (Pykalisto et al, 1975) and has been found to increase further following stabilization of reduced weight (Schwartz & Brunzell, 1981). After regain of lost weight, enzyme activity returned toward the original baseline level, although in one of the 4 subjects enzyme activity stayed elevated (Schwartz & Brunzell, 1981). The authors have concluded that this enzyme activity may be a counterregulatory mechanism which predisposes one to reattain a "set point" for body fat mass or fat cell size.

That fat cells themselves may be regulated in size is the basis for the fat-cell theory of weight regulation. This theory holds that fat cell size may regulate how much a person's total fat mass can be decreased (Brownell, 1984). People with hyperplastic obesity have a greater fat cell number than those with hypertrophic obesity (Hirsch & Knittle, 1970). When fat cells are reduced to a normal size, the person with hyperplastic obesity will still have a much greater than normal fat mass and a body weight that is still much above "ideal". To reach an "ideal" body weight requires reducing the fat cell to a size below normal which is very difficult both to attain and to maintain. During weight reduction and maintenance of lowered body weight, people with juvenile-onset obesity (hyperplastic) report hunger, food obsession, and a variety of psychological symptoms (Grinker & Hirsch, 1972; Nisbett, 1972) which are similar to those experienced by normal weight individuals undergoing semistarvation (Keys et al, 1950). These symptoms were not reported by people with adult-onset obesity

(hypertrophic) during or following weight reduction (Grinker & Hirsch, 1972). Bjorntorp et al, 1975 found in a program of weight reduction by diet (1100 kcals/day) in hyperplastic and hypertrophic obese women that when weight loss no longer continued, fat cells in both groups had reached a size close to that of the control women. With weight reduction those women with hypertrophic obesity reached a body fat mass that was not different from that of the control women, while those with hyperplastic obesity had a remaining fat mass that was an average of 10 kg greater. In another study, Krotkiewski et al (1977) found that those women with hyperplastic obesity were able to maintain their weight loss for 12 weeks while women with hypertrophic obesity maintained it for 51 weeks. When the regain started, the women with hyperplastic obesity gained at a rate three times that found in the women with hypertrophic obesity. Fat cell size at the end of weight loss was not measured in this study, but in the hyperplastic group of women initial fat cell size was already the same as the normal weight reference group and much smaller than that found in the hypertrophic group. If fat cells can be described as having a "biological need" to maintain a certain minimum size, such a "need" could explain why people with hyperplastic obesity regain lost weight so readily.

Another explanation for the ease with which people gain and regain weight has to do with the composition of the diet consumed. Experiments with rats have demonstrated that when fed diets high in fat or sucrose or made up of high-fat, high-sugar foods (the "supermarket" diet) weight gain is greater and faster than when fed regular (and very low-fat) chow (Sclafani, 1980). Rats fed the supermarket diet gained

269% more weight than control animals fed regular chow (Sclafani & Springer, 1976). Experiments in humans have demonstrated a similar tendency. In the overfeeding studies by Sims et al (1973), weight gain was much more easily accomplished and maintained when the excess calories were fed as fat rather than as carbohydrate. Other studies in humans have compared spontaneous calorie intake when fed diets varying in fat content.

One study compared satiety, calorie intake and eating time when consuming a high-energy density (high-fat) diet vs a low-energy density (low-fat, high-fiber) diet in both lean and obese subjects (Duncan et al, 1983). Subjects were free to choose desired quantities of food at each meal. Calorie intake on the low-energy density diet was 52% of that on the high-energy density diet (1750 vs 3000 kcals/day) and did not change during the 5-day feeding period, eg. no compensation in calorie intake occurred. Subjects rated themselves as somewhat hungrier before meals on the low-energy density diet but reported no differences in acceptance between the two diets. Though weight was not followed in this study, one can speculate that if no calorie compensation occurred over time, the low-energy density diet could lead to weight loss, the high-energy density diet to weight gain.

In a more recent study Lissner et al (1987) fed 3 different diets varying in fat content, each for two weeks and monitored calorie intake as well as body weight change. Subjects included both lean and obese and food was available freely throughout the day. While consuming the medium-fat diet (30-35% kcals as fat) calorie intake averaged 2352

kcals/day and weight changed very little (-0.03 kg). On the low-fat diet (15-20% kcals as fat) calorie intake was lower than on the medium-fat diet by 11% and weight decreased significantly (-0.40 kg). With the high-fat diet (45-50% kcals as fat) calorie intake increased to 15% above the intake on the medium-fat diet and weight increased significantly (+0.32 kg). No calorie compensation took place during any of the 2 week feeding periods. Considering the fact that average fat intake of Americans is 38-40% of calories (Gordon et al, 1982; Kurinij et al, 1986) the dietary environment is certainly supportive of the direction of weight gain.

If the calorie input side of the energy balance equation plays a role in regulating weight it is likely that the calorie output side plays a role also. People who are vigorously physically active, eg. athletes and laborers, tend to be more slender and have less body fat than those who are more sedentary (Mayer et al, 1956; Oscai, 1973). Though it is possible that more slender people self-select themselves into these activities, it is well known that physical activity has the general effect of decreasing body fat and increasing lean tissue, with the extent of the change depending on the duration and intensity of the physical activity (Oscai, 1973). The data on whether people with obesity are less active than those without obesity are conflicting, but results generally suggest that obese and lean children have similar activity levels whereas obese adults tend to be less active than their non-obese peers (Brownell & Stunkard, 1980).

Exercise training in obese men and women reduces body fat mass

(Moody et al, 1969; Bjorntorp et al ,1973; Gwinup, 1975; Getchell & Moore; 1975; Franklin et al, 1979). This occurs without "official" accompanying dietary restriction. However, even though subjects were requested to not change their usual diets, food intake was not monitored in these studies. Therefore, the role of calorie intake is unknown. Whether increasing physical activity in people with obesity causes a spontaneous change in calorie intake is of interest. A few studies have looked at this. Leon et al (1979) exercised obese college-age men 5 times/week for 16 weeks. No dietary restrictions were imposed and 3-day food records were kept at intervals during the study. At the 4th week reported intake was about 300 kcals/day above baseline, thereafter reported intake gradually decreased, and by the 16th week intake was about 150 kcals/day less than baseline. This implies that calorie intake did not keep up with calorie expenditure. However, subjects might also not have kept accurate food records. This is suggested by the fact that their weight loss was not as great as would be expected if their reported calorie intake was accurate (Leon et al, 1979; Pi-Sunyer & Woo, 1985). In exercise studies done in a metabolic ward where spontaneous calorie change was monitored by actual weighing of food, obese women did not increase their calorie intake during 19-day periods of increasing physical activty (Woo et al, 1982) whereas women of normal weight did increase calorie intake to match expenditure (Woo & Pi-Sunyer, 1984). In a longer exercise period of 57 days done with obese women, calorie intake still did not increase to match energy expenditure and the women lost weight (Woo et al, 1982a). To test the possibility that palatability and variety of the food offered might influence the quantity of food selected, another exercise

study was conducted by these researchers using obese men and a dietary offering described as being "in a much more gourmet, tastier form" (Pi-Sunyer & Woo, 1985). These subjects, like the obese women described above, consumed a fairly constant level of calories no matter what the exercise regime, but the intake of calories was in excess of energy need in all periods except that of the greatest expenditure (140% of basal activity). The authors concluded that sensory characteristics of food affect spontaneous dietary intake in people with obesity more than exercise does (Pi-Sunyer & Woo, 1985).

The effect of exercise on weight stability is also of interest. Jette (1975) reports that 8 years after a physical training program, those men who were classified as regular exercisers had the lowest body weights compared to those classified as irregular exercisers and non-exercisers, even though body weights had not differed during the original training program. Bjorntorp et al (1972) found that body weight and body fat were similar among middle-aged physically active men and young physically active men. The middle-aged physically active men had very stable weight histories and at age 53 had average body weights that were 8 kg lower than a group of 55 year old men who were mainly sedentary. Thirty-five years earlier, just prior to military service, these sedentary men had weighed an average of 68 kg, the same body weight that the physically active men had been able to maintain throughout adulthood. Bjorntorp (1976) cites this as longitudinal evidence that exercise contributes to weight stability over time. Bjorntorp et al (1972a) also found that with physical training in men following myocardial infarction, body weight and body fat decreased but

eventually became constant. Loss of body fat varied from 0 to 13 kg and constant body weight occured within 0 to 8 months following initiation of training. Fat cell biopsies showed very little variation in cell size among the trained men or in comparison to other trained men whether young or middle-aged (Bjorntorp, 1976). However, body fat mass was highly correlated with fat cell number. Bjorntorp (1976) concludes that physical training decreases and then stabilizes body weight at a point where fat cell weight reaches a fairly constant level. He also observed that the fat cell size at weight constancy achieved by physical training appears to be somewhat smaller than the fat cell size reached at weight constancy accomplished by dieting.

Using all of the above described evidence and theories pertaining to weight regulation, one can speculate on the possibility that each individual person has their own unique body weight and body fat range that is determined to some degree by the number of fat cells they have and perhaps to other, unknown "set-point" mechanisms. Movement up or down within that range may depend on one's level of physical activity and the richness or caloric density of the diet consumed. Maintaining body weight and fat mass at the low end of the range may most easily and effectively be achieved by habitual physical activity and low-fat dietary intake. In those people with relatively higher numbers of fat cells, the body weight achieved at the low end of their range may still be higher than what is either socially or medically "desirable" based on weight for height guidelines. Evidence has already been presented that demonstrates achieving "ideal" weight for height is not necessary for normalizing the health risk factors of blood lipids, blood

pressure, or glucose tolerance. A next logical question is what effect does decreasing dietary fat and increasing physical activity have on these health risk factors independently of body weight change?

Effect of changes in dietary fat intake on CHD risk factors

The importance of decreasing dietary fat, especially saturated fat, to decrease blood lipids is evidenced by the numerous current public health guidelines (Dietary Guidelines for Americans, 1985; American Heart Association, 1986; Nat'l Cholesterol Education Program Expert Panel, 1988) that are based on results from many metabolic and intervention studies. The classic metabolic studies of Hegsted et al (1965) and Keys et al (1965) demonstrated the blood cholesterol lowering effect of decreasing saturated and increasing polyunsaturated fats in the diet. Current recommendations call for not only decreasing saturated fat but also decreasing total dietary fat. Grundy et al (1986) recently demonstrated that when saturated fats are decreased in the diet, replacement by polyunsaturated fat, moderate amounts of carbohydrate, or high amounts of carbohydrate all had an equal effect in lowering serum total cholesterol and LDL-cholesterol. In their group of normolipidemic men, lowering total fat intake from 40% kcals as fat to 30% or 20% kcals as fat resulted in serum total cholesterol levels that decreased 17% and 19%, respectively. Replacing the saturated fat calories with polyunsaturated fat calories had a similar 17% lowering effect on serum total cholestrol.

Dietary intervention studies have achieved reductions in serum

cholesterol in the range of 6% to 15% by reducing dietary fat. In the Oslo Heart Trial serum cholesterol fell 13% when total dietary fat intake was reduced from 44% of kcals to 28% and saturated fat reduced from 18% of kcals to 8% (Hjermann et al, 1981). In the MRFIT study a smaller reduction in serum cholesterol was seen, falling 6.3% after the first year of dietary intervention and achieving a 7.4% reduction by the end of four years of intervention (Caggiula et al, 1981). The smaller reduction was attributed to the fact that at baseline dietary fat intake in this population was already lower than that of the general population (38.3% vs 39.0% found in the Framingham study and 40.4% found in the National Diet-Heart Study at baseline). In this study those men with higher baseline serum cholesterol levels experienced greater reductions in cholesterol levels. Those who lost weight also had greater decreases in serum cholesterol. About 26% of the overall group lost 10 pounds or more and this subgroup experienced a 10% reduction in total serum cholesterol. The men who lost weight were found to be those who had the greatest decreases in dietary fat intake and increases in dietary carbohydrate (Caggiula et al, 1981).

Replacing dietary fat with dietary carbohydrate has raised concerns about a possible effect of elevating plasma triglyceride level (NIH Consensus Conference on Hypertriglyceridemia, 1984; Nationl Cholesterol Education Program Expert Panel, 1988) which is also considered a CHD risk factor (Carlson et al, 1979; Castelli et al, 1977). Ginsberg et al (1976) found that a change from a control diet (45% kcals as fat, 40% as carbohydrate, 15% as protein) to a low fat, and therefore high carbohydrate, diet (30% kcals as fat, 55% as

carbohydrate, 15% as protein) produced a 41% increase in fasting triglyceride level raising triglycerides from a mean of 155 mg/dL to 219 mg/dL. The experimental diet was a liquid formula and fed for a period of one week to subjects, one-third of whom had existing hypertriglyceridemia (fasting triglycrides greater than 150 mg/dL). The same research group repeated a similar experiment using real food rather than liquid formula on a group of subjects with normal triglyceride levels and found a 33% increase in fasting triglyceride from a mean of 90 mg/dL to 134 mg/dL (Coulston et al, 1983). Their diet was again fed for a short period, 10 days in this case. In the metabolic study by Grundy et al (1986) triglyceride levels did not change at all from baseline levels when subjects consumed a diet with 30% kcals as fat for a 2 month period. With the 20% kcals as fat diet eaten for a similar 2 month period, triglyceride levels increased for a few subjects but for most remained the same or decreased.

In the longer term dietary intervention studies where participants changed to a low-fat, high-carbohydrate diet over several years, fasting triglyceride levels decreased significantly from baseline. In the Oslo Heart Trial (% kcal as fat decreased from 44% to 28%), the triglyceride level fell 20% (Hjermann et al, 1981). In the MRFIT study (decrease of 28% in saturated fat intake, increase of 9% in carbohydrate intake) triglyceride levels fell overall by 4.3% (Caggiula et al, 1981). In the subgroup who lost 10 pounds of body weight or more and made the greatest dietary fat decreases and carbohydrate increases, triglycerides fell 20%. For long-term habitual dietary change, an increase in dietary carbohydrate does not appear to have a

deleterious effect on plasma triglyceride level. In addition, the report of the NIH Consensus Conference on Hypertriglycridemia (1984), indicated that triglyceride levels between 250 and 500 mg/dL were considered "borderline" and that a diagnosis of hypertriglyerdemia was not appropriate until levels above 500 mg/dL were observed. The elevations in triglycerides due to an increase in dietary carbohydrate found in the two studies cited above did not exceed 250 mg/dL except in a subgroup in the study by Ginsberg et al (1976) whose triglycerides increased from a mean of 252 mg/dL to 338 mg/dL with a change to the higher carbohydrate diet. The long-term effect of such a dietary change in those individuals is unknown. If a low-fat diet is followed habitually, weight reduction is likely to occur (Lissner et al, 1987; Caggiula et al, 1981) which will play an important role in correcting hypertriglyceridemia (NIH Consensus Conference of Hypertriglyceridemia, 1984; National Cholesterol Education Expert Panel, 1988).

Though one commonly thinks of sodium in relation to the effects of diet on blood pressure and more recently the possible effects of calcium intake (McCarron et al, 1984), dietary fat appears to also have an effect on blood pressure. Puska et al (1983) demonstrated this effect in middle-aged couples whose blood pressure was monitored during a 2 week baseline control diet (39% of kcals as fat, P/S ratio 0.1-0.2), a 6 week low fat diet (24% of kcals as fat, P/S ratio 1.0), then back to the control diet for 4 weeks. Both systolic and diastolic blood pressure decreased significantly during the 6 week low fat diet period then rose significantly back to baseline levels when the regular control diet was resumed. This was in contrast to another group of

couples who followed the same pattern but decreased dietary sodium rather than dietary fat. Their blood pressures did not change from baseline levels when consuming the lower sodium diets. Another group consuming the regular control diet throughout also experienced no changes in blood pressure. The effect of lowered dietary fat on blood pressure was confirmed in a subsequent study (Huttunen et al, 1985). The low-fat diet (24% of kcals as fat) decreased blood pressure equally whether the P/S ratio was 0.4 or 0.9. Brussaard et al (1981) saw an effect of lowering dietary fat on blood pressure in young, normotensive students, but the slight decrease seen was no greater than what the control group experienced. Huttunen et al (1985) suggest that the effect of dietary change may depend on initial levels of blood pressure. In the study by Puska et al (1983) blood pressure decreases were greatest in those whose diastolic blood pressures were >90 mmHg at baseline (Huttunen et al, 1985).

While decreases in dietary fat are known to improve blood lipid status and may have an effect on improving blood pressure, whether reducing fat intake and increasing carbohydrate intake has beneficial effects on glucose tolerance is somewhat controversial. The benefits may depend on the type of carbohydrate that is consumed and the effect of the dietary change on weight status.

Because people with diabetes have increased risk for CHD, current dietary recommendations in the US, Canada, and England call for decreasing dietary fat to 30% of kcals or less and increasing carbohydrate to 50%-60% of kcals to lower their risk (Committee of the

American Diabetes Association on Food and Nutrition, 1979; Jenkins et al, 1982). These recommendations created some controversy because of the potential of increased dietary carbohydrate worsening glucose tolerance. In addition to investigating the effect of increased dietary carbohydrate on triglyceride levels, Ginsberg et al (1976) and Coulston et al (1983) studied the effect of such a dietary change on plasma glucose and insulin. Both studies found non-significant post-meal increases in glucose and significant post-meal increases in plasma insulin when subjects consumed the low-fat, high-carbohydrate diets. In the earlier study (Ginsberg et al, 1976), the diet fed was a liquid formula with the carbohydrate consisting of dextrins, maltose, lactose, and sucrose with sucrose comprising the same absolute percentage of calories (24%) in both the high-fat and low-fat diet formulas. In the later study (Coulston et al, 1983), the carbohydrate came from real foods with sucrose comprising 22-25% of total calories in both the high and low-fat diets. In both studies sufficient calories were fed to maintain body weight.

In subsequent studies, the effect of dietary carbohydrate on plasma glucose and insulin response has been found to vary with the source of carbohydrate. Certain foods fed singly or in meals (potatoes, dextrose, and sucrose) elicited a significantly greater insulin response compared to other foods (rice and corn) in both normal and glucose-impaired subjects (Crapo et al,1977; Crapo et al, 1980; Coulston et al, 1980; Coulston et al, 1981). In the glucose-impaired subjects, potatoes, dextrose and sucrose also elicited a significantly greater glucose response than did rice and corn. Legumes have been

shown to elicit a 45% lower glucose response than other carbohydrate foods like grains, breads and pasta, breakfast cereals, biscuits and tubers (Jenkins et al, 1980). Several studies have demonstrated a lowered glucose response to high-carbohydrate high-fiber containing meals (Jenkins et al, 1976; Jenkins et al, 1977) and to high-carbohydrate, high-fiber diets fed for several weeks (Miranda & Horwitz, 1978; Kiehm et al, 1976; Anderson & Ward, 1978) in subjects with diabetes.

Current dietary guidelines call for an increased intake of carbohydrate to replace calories from fat (Dietary Guidelines for Americans, 1985; American Heart Association, 1986; National Cholesterol Education Program Expert Panel, 1988). All specifically highlight an increased consumption of complex carbohydrate and fiber. Evidence indicates increasing the amount of carbohydrate as starch and fiber should minimize the impact of increased dietary carbohydrate on glucose tolerance. In addition, if weight is lost by consuming a lower fat diet, glucose tolerance would likely improve even in the face of an increase in dietary carbohydrate, as weight loss is highly effective for improving glucose tolerance in overweight persons both with and without diabetes (Golay et al, 1985).

While weight loss has the effect of improving glucose tolerance physical training also improves glucose tolerance. This occurs in people of normal weight or those who are overweight whether weight is lost or not (Berger & Berchtold, 1982). We turn now to a discussion of the effect exercise has on glucose tolerance, blood pressure and blood

lipids.

Effect of exercise on CHD risk factors

That habitual physical activity influences glucose tolerance has been observed in both men and women. Bjorntorp et al (1972) compared glucose and insulin response to a 100 g glucose load in physically active and sedentary middle-aged men (mean ages 54 and 55 years, respectively). They found significantly lower plasma levels of insulin at all time points for 2 hours following the load and significantly lower plasma glucose levels at all time points except at 2 hours in the physically trained men compared to those who were sedentary. For the physically well-trained men, glucose levels were above fasting only at 30 minutes whereas glucose did not return to the fasting level until 120 minutes in the sedentary men. In a study done with men and women ranging in age from 22 to 60 years, the relationship between physical fitness (maximal aerobic capacity) and insulin-stimulated glucose disposal was highly significant ($r=0.63$, $p<.0001$). The relationship between maximal aerobic capacity and glucose and insulin response following a 75 g glucose load was lower but still significant for glucose and insulin (Rosenthal et al, 1983). Likewise, Hollenbeck et al (1984) found that insulin-stimulated glucose disposal was significantly higher in older men (aged 60 to 75 years) who habitually exercised than in men of the same age who did not exercise. They found a highly significant relationship ($r=0.75$, $p<.001$) between maximal aerobic capacity and the metabolic clearance rate of glucose in their group of subjects.

In contrast, Montoye et al (1977) did not observe a significant difference between habitual physical acivity and glucose tolerance in males aged 16 to 64 years in the Tecumseh Health Study. Because glucose tolerance in this study was related to body fatness, the data were reanalyzed with three subgroupings based on skinfold measurements. In the leanest third, glucose tolerance was significantly decreased in those who were sedentary, but no differences were seen in the other two body fat groupings. The authors note that the intensity of exercise in their "most active" group was less than that in the group studied by Bjorntorp et al (1972) and may have accounted for the difference in findings.

In a recent study of women, those who were former college athletes were found to have a lower incidence of diabetes than non-athletes (Frisch et al, 1986). The 5398 women surveyed ranged in age from 21 to 80 years and had graduated from college between the years 1925 and 1981. Former athletes had a diabetes rate occurring after age 20 years of 0.5% compared to 1.2% in non-athletes. Both groups had similar percentages of family history of diabetes, and a similar distribution between gestational, insulin-using, and non-insulin-using diabetes. Of the former athletes, 74% were currently exercising regularly compared to 57% of the nonathletes. Body weight and estimated body fat were slightly but significantly lower in the former athletes, while significantly more nonathletes reported they were currently restricting their diets (46.2%) compared to athletes (42.0%). Equal percentages reported being on low-fat diets (20.5% and 21.3%).

LeBlanc et al (1979) suggest that the better glucose tolerance in athletes is due to their lower levels of body fat rather than their increased physical capacity. In a study of college students varying in levels of maximal aerobic capacity (VO2 max), those with a VO2 max <60 $ml.kg^{-1}.min^{-1}$, had an average percent body fat of 14.8%, while those with a VO2 max >60$ml.kg^{-1}.min^{-1}$averaged 4.4%. Lean body mass was equal in the two groups. Following i-v administration of glucose, plasma glucose was higher in the less-trained group for only the first 20 minutes while plasma insulin rose to a level 3 times higher in the less-trained group and stayed significantly higher for the two hours of the test. C-peptide concentration followed a similar pattern indicating that the well-trained group secreted significantly less insulin. The well-trained group also had a greater percent insulin binding to monocytes than those less well trained. In this study both VO2 max and adiposity were significantly related to initial plasma glucose, insulin, and insulin binding. Adiposity and VO2 max were also significantly related to each other. With partial correlation analysis only the relationship between adiposity and these three factors remained statistically significant. The authors concluded that body fat was a more important regulator than physical training. However, a direct effect of exercise training on improved glucose tolerance has been observed in adults who are both normal weight and overweight when no change occurs in body weight or fat.

Soman et al (1979) trained normal weight non-diabetic subjects for 6 weeks. Maximal aerobic capacity (VO2 max) increased 16% and

insulin-mediated glucose uptake increased 30%. The change in VO2 max was highly correlated with the change in glucose uptake (r=0.81). Insulin binding to monocytes increased by 35% following training. Body weight did not change as a result of the training program. In an 8-week exercise training study with obese men and women, Bjorntorp et al (1970) demonstrated significant decreases in insulin response to a 100 g load during a 3 hour oral glucose tolerance test. Plasma glucose response during the glucose tolerance test was not significantly different after the training. Body weight increased an average of 3 kg with the increase due to body fat which was 3.3 kg higher at the end of the 8 weeks. Maximal aerobic capacity increased 7% during the training program. In a longer training program Bjorntorp et al (1973) found improvements in both fasting insulin and glucose and insulin and glucose response to a glucose load. In this study, physical training was conducted for 6 months. Body weight increased an average of 1 kg and body fat decreased 3 kg, both non-significant changes. Maximal aerobic capacity increased significantly by 17% over the six months. Fasting levels of glucose and insulin decreased significantly as did the sum of the glucose and insulin values during a 3-hour glucose tolerance test. The magnitude of the change was greatest for insulin, with fasting level decreasing 67% compared to 10% for glucose, and the sum of the values during the glucose tolerance test decreasing 38% for insulin and 16% for glucose. A recent exercise training study in obese women (DeFronzo et al, 1987) has demonstrated that 6 weeks of physical training of moderate intensity (65% of VO2 max) decreases the hyperinsulinemia associated with obesity with no accompanying body weight or fat change. Both an increase in insulin sensitivity and an

increase in tissue sensitivity to insulin were demonstrated to result from training, although neither reached the levels seen in the control subjects. Because the decrease in hepatic glucose production was less following training than the increase in tissue glucose uptake during the euglycemic clamp study, the authors concluded that peripheral tissues contributed the most to the improvement in total body glucose metabolism.

Because of the beneficial effects of physical training on insulin, habitual exercise is recommended for people with obesity both with and without non-insulin dependent diabetes mellitus (Berger & Berchtold, 1982; Wirth & Bjorntorp, 1981). The effect of physical training on insulin may have an additional effect beyond that on carbohydrate metabolism. Blood pressure has also been found to decrease in obese subjects after physical training with the effect being related not to changes in body weight or body fat but to decreases in plasma insulin, glucose, and triglyceride levels (Krotkiewski et al, 1979).

In the study by Krotkiewski et al (1979) 27 obese women participated in an exercise training program for 6 months while making no dietary changes. Mean weight and body fat did not change over the 6 months for the whole group, but those women with fewer fat cells did experience the greatest decreases in body weight whereas those with more fat cells gained weight. At the end of 6 months, small, insignificant decreases were seen in fasting plasma insulin and glucose, while significant decreases were observed in both resting systolic and diastolic blood pressure. When divided into groups based

on fasting plasma insulin values, those with the high initial plasma insulin levels had significantly greater decreases in fasting plasma insulin and glucose, triglycerides, and systolic and diastolic blood pressure following training. The authors speculate that the hypertension associated with obesity is not so much associated with body fat but rather follows the other metabolic alterations of elevated plasma insulin, glucose, and triglyceride. They suggest a possible mechanism may be the effect insulin has on increasing sodium reabsorption in the distal tubule of the kidney. Horton (1981) suggests that an additional possible mechanism for decreased blood pressure following exercise training may be the lowered sympathetic tone and decreased plasma norepinephrine and epinephrine found in trained individuals. This may in turn decrease peripheral vascular resistance and result in lowered blood pressure.

Whatever the mechanism(s), physical training appears to have variable effects on blood pressure. In general, decreases in blood pressure on the order of 6% for systolic and 7% for diastolic have been seen in exercise training studies of hypertensive adults (Seals & Hagberg, 1984). Of the 12 studies Seals & Hagberg reviewed, only three reported significant body weight loss (1.2 to 3.2 kg). Interestingly, in those three studies blood pressure at rest was not significantly reduced. Therefore, body weight change was not an apparent confounding factor in the effects of exercise on blood pressure change.

Few studies have been done to determine the effects of exercise alone on blood pressure change in obesity (Horton, 1981). In addition

to the study by Krotkiewski et al (1979) described above, Franklin et al (1979) conducted a 12-week exercise program in obese and normal weight women, requesting that the subjects make no dietary changes. Body weight decreased by 2.6 kg in the obese group but did not change in the normal weight group. Systolic blood pressure decreased significantly by 6% and diastolic by 4% in the obese group but did not decrease in the normal weight group. Though the blood pressure in the obese group was not in the hypertensive range, it was higher than that found in the normal weight group. Horton (1981) has commented that exercise training can be expected to result in decreases in blood pressure in those with mild and moderate hypertension but will have little or no effect on blood pressure in individuals who are normotensive. Kukkonen et al (1982) trained overweight men and women for 17 months with periodic "general dietary advice" offered. Mean weight change was -3.8 kg for the men and -2.7 kg for the women. Significant decreases of 5% were seen for both systolic and diastolic blood pressure for the men, but no change was seen for the women. Initial blood pressure was 138/90 mmHg for the men and 142/90 mmHg for the women. Leon et al (1979) exercised obese young men for 16 weeks with dietary instructions to "eat whatever they wanted". Body weight decreased an average of 5.7 kg and diastolic blood pressure decreased a significant 6%. No change was seen in systolic blood pressure. Dahlkoetter et al (1979) compared weight loss and blood pressure change in overweight women in dietary behavior modification only, exercise only, and a combination group in an 8 week program. Average weight change was -6, -8, and -12 pounds, respectively. Systolic and diastolic blood pressure decreased significantly 7-8% in all three

groups. A clear effect of exercise apart from weight change cannot be discerned from this study. Lewis et al (1976) found a slight but nonsignificant decrease in systolic and diastolic blood pressure in obese women following a 17 week exercise and eating awareness program. Weight change averaged −4.2 kg. In the last three cited studies (Leon et al, 1979; Dahlkoetter et al, 1979; Lewis et al, 1976) mean systolic blood pressure was less than 120 mmHg and mean diastolic blood pressure was less than 80 mmHg before exerise training began.

While some larger scale studies have demonstrated that habitually physically active people have less risk of developing hypertension than those who are not active (Pfaffenbarger et al, 1983; Blair et al, 1984), "official" recommendations are lacking for promoting the use of exercise for the control of hypertension (Seals & Hagberg, 1984).

With the exception of treatment for hypertriglyceridemia, "official" recommendations for control of blood lipids also do not include exercise. Both the American Heart Association guidelines for treatment of hypertriglyceridemia and the NIH Consensus Conference report of treatment of hypertriglyceridemia mention exercise as a useful part of therapy (NIH Consensus Conference on Hypertriglyceridemia, 1984; American Heart Association, 1986), whereas exercise is not included in the recommendations for the treatment of elevated serum cholesterol (Gotto et al, 1984; National Cholesterol Education Program Expert Panel, 1988). Reduction of dietary fat and weight loss appear to have the major impact on serum cholesterol levels.

Tran & Weltman (1985) reviewed 95 studies dealing with the effects of exercise on blood lipids and analyzed the results based on changes in body weight. They found that with no change in body weight resulting from exercise training (change <1kg), serum total cholesterol and LDL-cholesterol (LDL-C) decreased significantly 7.3 and 3.3 mg/dL, respectively. When weight loss resulted from exercise training (mean loss -2.2 kg), serum total cholesterol and LDL-C decreased even further, by 13.2 and 11.1 mg/dL, respectively. With weight gain following exercise training (mean gain +1.3 kg), serum total cholesterol and LDL-C increased 2.9 and 3.0 mg/dL, respectively, though the change was not statistically significant. Changes in triglycerides followed a similar pattern with a 14.0 mg/dL decrease associated with no weight change, a 21.5 mg/dL decrease with weight loss and a 9.4 mg/dL increase with weight gain. In all three weight categories HDL-cholesterol (HDL-C) increased, although only the increases in the no weight change (+1.7 mg/dL) and weight loss (+2.3 mg/dL) categories were statistically significant. Exercise studies in obese women were not included in the analysis by Tran & Weltman. These studies have not found significant decreases in total cholesterol or LDL-C even with weight losses up to 5.7 kg (Lewis et al, 1976; Franklin et al, 1979; Kukkonen et al, 1982; Bjorntorp et al, 1973; Krotkiewski et al, 1979).

Recommendations for treatment of mild and moderate obesity

Current recommendations for weight loss/control programs for people with mild and moderate levels of obesity include the combined

use of exercise, moderate calorie restriction, and behavior modification (American College of Sports Medicine, 1983; Weinsier et al, 1984; Stunkard, 1984; Brownell, 1984). Intensity of exercise is recommended to be 60% to 85% of maximum heart rate (American College of Sports Medicine, 1983; Weinsier et al, 1984) although it is important to note that lower intensities of exercise will promote more fat loss than will exercise of higher intensity (Girandola, 1976), and that people with obesity who are also unaccustomed to physical activity will need to begin at very low intensities and durations (Brownell, 1984). Dietary management needs to be designed to meet the following criteria: 1) satisfy all nutrient needs except energy; 2) be acceptable to meet individual tastes and habits; 3) minimize hunger and fatigue; 4) be readily obtainable and socially acceptable; 5) favor establishment of a lasting pattern of eating; 6) be conducive to improvement of overall health (Weinsier et al, 1984). Hirsch (1985) further recommends that diets should do no physiologic harm and should be seen as "forever". He states that attention to diet cannot be relaxed after weight loss is achieved and that people should not change anything until they are ready to make permanent dietary changes. Changes in diet should be small at first, gradually adding new restrictions, rather than the more usual approach of radically new and different ways of eating that can rarely be followed for more than a short period of time (Hirsch, 1985). Diets are also recommended that maximize retention of lean body mass (American College of Sports Medicine, 1983; Isaksson, 1985) and will therefore require intakes above 1200 kcals/day. Behavior modification is recommended to include self-monitoring (records of food, activity, emotions), control of environmental factors surrounding eating,

learning to make "trade-offs" eg. eating more on special occasions and less at other times to achieve an overall energy balace, and learning to use postive, coping self-statements (cognitive restructuring). Additional psychological counseling may be recommended for selected people (Weinsier et al, 1984; Stunkard, 1984; Brownell, 1984).

While there is growing agreement on the methods of weight loss and control for people with mild and moderate obesity, there is a lack of consensus on what the appropriate goal for body weight should be. The NIH Consensus Conference on Obesity recommended that anyone weighing 20% above desirable weight for height or more should be treated for weight loss (Burton et al, 1985). Others feel that is too generous and anyone above 110% of desirable weight for height is at risk and should be treated (Manson et al, 1987). Because of the concern for the development of eating disorders among women who are under great social pressure to be thin and because evidence is fairly weak that mild and moderate obesity carry health risks for women, some are advocating that perhaps obesity in women should not be treated at all (Wooley & Wooley, 1984). However, a growing number of recommendations are being offered that weight goals for obesity should be determined on an individual basis taking into account family weight and medical history, personal weight and medical history, distribution of body fat, and presence of health risk factors (Knapp, 1983; Callaway, 1984; Van Itallie, 1985; Harrison, 1985; Feinleib, 1985; Bjorntorp, 1985).

Proposal for the "best" approach to weight control for mild to moderately obese women

Evidence presented in this literature review points to the following conclusions: 1) overweight/obesity in women is associated with less mortality risk than in men; 2) overweight/obesity in women is associated with a lower incidence of the cornonary heart disease risk factors of elevated blood lipids, hypertension, and glucose tolerance than in men unless body fat is carried predominately around the abdomen (upper body fat); 3) social pressures on women to be thin are very strong and go beyond the concern for health; 4) dieting as a means to lose weight has a very low success rate and because of the associated loss of lean body mass and potential for inadequate intake of required nutrients may do more harm than good; 5) individuals may have their own body weight "set points" and efforts to reduce weight below that point may meet with major physiological resistance; 6) individuals with large numbers of fat cells may remain well above ideal body weight when fat cells are of a normal size or smaller; 7) a high-fat diet may predispose to easier gain of body weight and its maintenance at higher levels; 8) habitual physical activity may contribute to stability of body weight at lower levels; 9) a decrease in intake of dietary fat reduces blood lipid levels, decreases overall calorie intake spontaneously without accompanying feelings of hunger, and may also reduce blood pressure; 10) increased physical activity increases insulin sensitivity and improves hyperinsulinemia, hyperglycemia and hypertriglyceridemia, and is associated with decreases in blood pressure and blood cholesterol with the effects being enhanced further

by accompanying weight loss; 11) attainment of an "ideal" body weight is not necessary to achieve normal levels of blood lipids, blood pressure or glucose tolerance.

Based on the above it seems reasonable to propose that the best approach to weight control in women with mild and moderate levels of obesity is one that focuses on learning to habitually consume a diet that is low in fat and to habitually participate in moderate intensity activity while in an environment where one's self-worth is not inversely proportional to one's body weight. The weight achieved by this approach might then be considered "best". As stated in the first chapter, the purpose of this research project was to test this possibility.

CHAPTER III

OVERVIEW AND EXPERIMENTAL DESIGN

This chapter will give a brief overview of the project, including the overall experimental design, and describe the role each of the authors of the subsequent papers has played in the research project.

Overview

This research project began in the fall of 1984 with the recruitment and orientations of subjects. The numbers of people involved in the recruitment process are included in the Appendix. Of the twenty women initially selected to be subjects, 17 completed baseline testing, two being rejected due to difficulty with blood draws, and one dropping out due to illness. During the 9-month program, one woman dropped out due to a change in job location and one woman was unable to participate in the end-point testing because of work-related injuries. Thus, complete pre and post data were collected on 15 women and they comprise the group whose results are reported in this dissertation.

After obtaining informed consent from the women selected to be subjects, a 12-lead EKG was done in the Human Performance Lab. Fasting blood was also drawn and sent to the lab at Good Samaritan Hospital for a general chemistry screen. Results of both the chem screen and EKG

were sent to each woman's physician and a signed permission form returned from the physician. On two occasions before baseline testing the women visited the Human Performance Lab to become accustomed to the metabolic rate testing, and the bicycle ergometer, treadmill, and underwater weighing procedures. On the second visit both a practice submaximal bicycle test and maximal treadmill test were conducted.

Figure 3-1 describes the experimental design of the study. The exercise and nutrition education program was proceeded and followed by a testing period with some of the tests repeated in the middle of the 9-month program. Figure 3-2 depicts the testing schedule used for both pre and post testing periods. The women were divided into groups of four based on similarities in timing of menstrual cycles. When the first group began week two of the testing schedule, the 2nd group began week 1. With each ensuing week a new group rotated through the 3-week schedule until all subjects were tested. As soon as a group completed their 3-week testing schedule at baseline testing they began participating in the walking program. When all five groups finished baseline testing, the nutrition education program began for all subjects. Prior to the baseline and endpoint testing periods, blood was obtained for analysis of blood lipids. Food records were also obtained prior to each testing period as were body weight and percent body fat measurements.

In May/June certain tests were repeated. These included resting metabolic rate (RMR), body composition, aerobic capacity (VO2 max and submax) blood pressure (BP) measurements, blood lipids, glucose

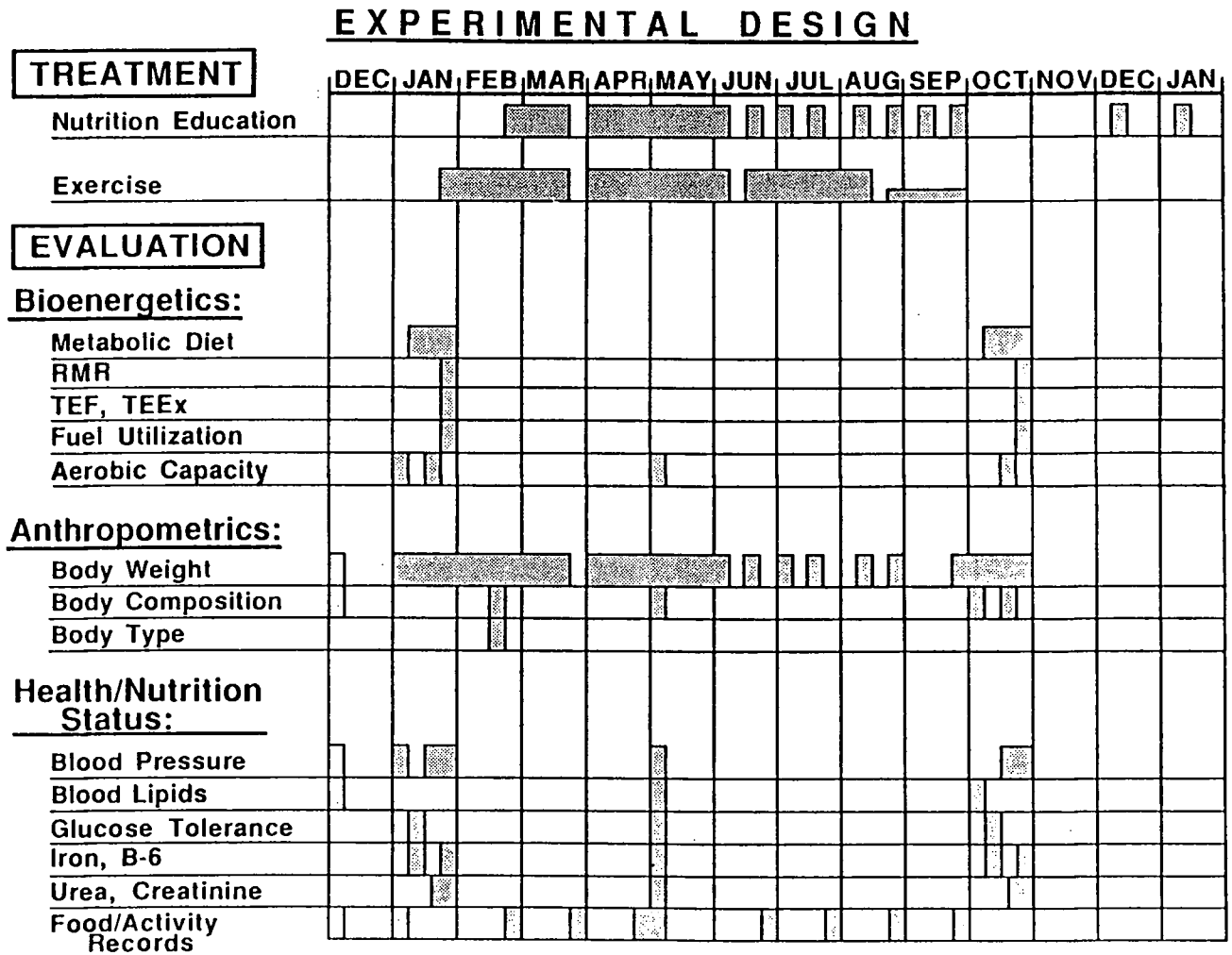

Figure III.1. Outline of experimental design of the project. Shaded areas indicate when treatment or evaluation occurred.

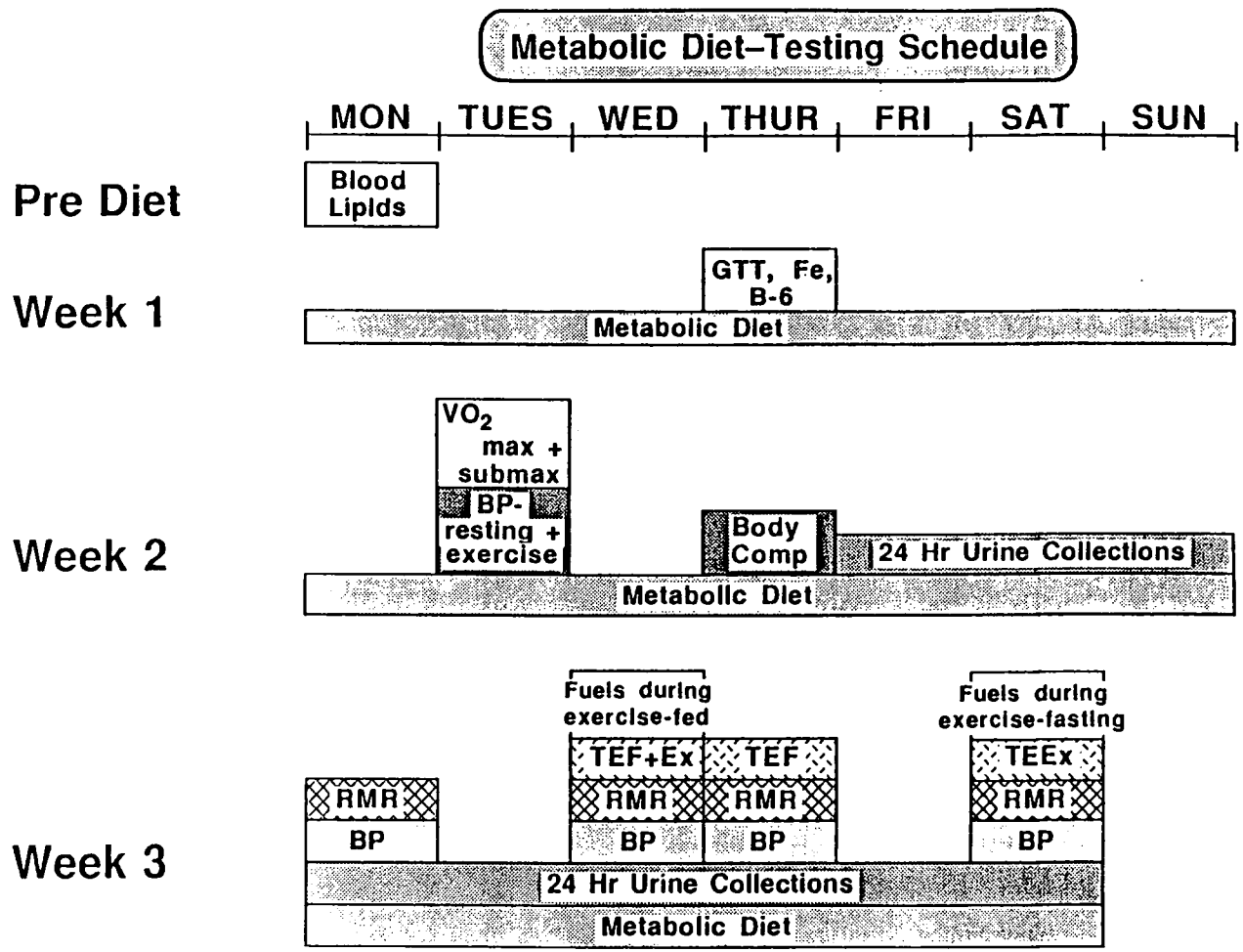

Figure III.2. Outline of 3-week testing schedule conducted before and after the 9-month program. (Abbreviations: GTT-glucose tolerance test, Fe-iron, VO_2-maximal aerobic capacity, RMR-resting metabolic rate, BP-blood pressure, TEF-thermic effect of food, TEEx-thermic effect of exercise.)

tolerance test (GTT), serum iron (Fe) and plasma pyridoxal 5'-phosphate (PLP) determinations, and 24-hour urine collections in conjunction with recording 3 days of food intake. It should be noted that the women were not on a controlled metabolic diet during this mid-point testing period. Data from the testing done at mid-point were used for making new exercise prescriptions and to monitor progress. Because the data collected at mid-point was not comparable to that obtained during the metabolic diet pre or post, it is not discussed in the following papers. The data are included in the tables in Appendix I.

The papers in the following 3 chapters will describe in detail the metabolic diet, glucose tolerance test, iron and PLP determinations, aerobic capacity, blood pressure measurements, body composition, 24-hour urine collections, resting metabolic rate, and blood lipid measurements conducted during the baseline and endpoint testing periods, and the nutrient analysis of food records collected during the 9-month program.

During the 3rd week of each testing period, additional data were collected which will not be reported in the 3 papers in this dissertation. The thermic effect of food (TEF), thermic effect of exercise (TEEx), and potentiation of the thermic effect of food with exercise (TEF+Ex) were measured in all of the women in conjunction with the measurements of RMR. Results from the baseline measurements of thermogenesis have been reported by Oddou (1985). Results from both the baseline and endpoint testing of thermogenesis are expected to be submitted for publication at a later date.

Contribution of Authors

A project of this magnitude could not be accomplished through the efforts of just one person. Many people contributed time and expertise. Dr. James Leklem served as my major professor and has been involved in this project since its inception in the fall of 1983. The roles of the other authors of the papers in the next three chapters are described below.

William Oddou was a fellow doctoral student in the Physical Education Department and participated in all aspects of this project except the end point testing. He collaborated in planning the details of the testing schedule and the testing protocols for the exercise tests, body composition determinations, resting metabolic rate and thermogenesis study. He participated in almost all aspects of baseline testing. He was responsible for planning and coordinating the walking program and shared teaching responsibilities in the nutrition education classes. Bill completed his doctoral dissertation in 1985 and since that time has been working on both the pre and post thermogenesis data and body compostion data for submission for publication.

Dr. Michael Maksud is currently serving as my minor professor and has been involved in the planning, implementation, and evaluation of this project since the summer of 1984. As Director of the Human Performance Lab, he played an essential role in making those facilities available and in developing protocols used for the exercise testing,

metabolic rate and thermogenesis studies, and body composition determinations. He has continued to contribute his expertise in evaluating the data obtained and in reviewing the papers which are being submitted for publication.

Dr. Donald Campbell served as my minor professor until his retirement in June, 1986. He was involved in the very beginning stages of this project, particularly in the planning, implementation, and evaluation of a pilot project on body composition determinations done in the fall of 1983. During the research project he was most involved in advising about the protocols for resting metabolic rate, thermogenesis measurements, and the fuel utilization during exercise study. Following completion of the year long project, he provided valuable assistance with statistical analysis of the data. Though retired, he continues to serve on my doctoral committee and provides his expertise in the evaluation and presentation of the data for publication.

CHAPTER IV

ENERGY NEED IN CHILDHOOD AND ADULT-ONSET OBESE WOMEN BEFORE AND AFTER A 9-MONTH NUTRITION EDUCATION AND WALKING PROGRAM

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ABSTRACT

The effect of a 9-month nutrition education and walking program on total calorie need, resting metabolic rate (RMR), body composition, and activity level was determined in 7 women with childhood-onset obesity (CO) and 8 women with adult-onset obesity (AO). A 3-week testing period was conducted before and after the 9-month program during which all subjects were on a controlled, weight-maintaining metabolic diet which was used to determine total calorie need. Body composition and RMR were measured during both 3-week testing periods. Body weight decreased significantly for CO (-5.7 $\pm$ 9.3 kg) and AO (-3.3 $\pm$ 6.2 kg), but fat-free mass (FFM) was unchanged for both groups over the 9 months. Need for total calories increased by 2.9% for CO and 3.1% for AO from pre to post whereas calories for RMR decreased 3.6% for CO and 2.8% for AO. Time spent in light and moderate level activity increased significantly from pre to post. The increase in activity more than compensated for the slight decrease in RMR, contributing to an overall increase in daily energy need at the end of the program, even for those women losing substantial body weight.

KEY WORDS: energy need, body composition, age of onset of obesity, diet, exercise

INTRODUCTION

A major problem associated with most approaches to weight loss is the reduction in total calories needed to maintain body weight after weight is lost (Bray, 1969; Bessard et al, 1983; Leibel & Hirsch, 1984). This decreased calorie need is thought to be due primarily to a loss of fat-free mass with its concomitant reduction in resting metabolic rate (Ravussin et al, 1985) and has been suggested as a possible reason for the typical regain in weight following weight loss (Schutz et al, 1984). We were interested in investigating whether it was possible to prevent that decrease in fat-free mass and caloric need by offering a program to obese women that promoted improved health and fitness and gradual body fat reduction achieved by a long-term moderate increase in physical activity and moderate decrease in calorie intake rather than emphasizing dieting or rapid and drastic weight loss (American College of Sports Medicine, 1983; Hirsch, 1985). Because treatment of childhood-onset obesity is considered more difficult and less successful than adult-onset obesity whether by diet or exercise (Krotkiewski et al, 1977; Krotkiewski et al, 1979), we were also interested in comparing body composition and energy need of women with these two types of obesity before and after such a program.

The purpose of this study, therefore, was to determine body composition, resting metabolic rate and total calorie need, and their interrelationships, prior to and following a program promoting moderate and long-term changes in calorie intake and physical activity in women differing in age of onset of obesity.

METHODS

Subjects

Fifteen premenopausal women participated in all aspects of this study. Approval for the study was granted by the Human Subjects Committee at Oregon State University. The women, ranging in age from 28 to 42 years, were healthy, non-smokers, sedentary, and taking no medications or vitamin/mineral supplements. All had body weights greater than 30% above the reference weight for their height (based on the midpoint of the weight range in the 1983 Metropolitan Weight Tables) and all had been weight stable ($\pm$ 2 kg) for at least 2 months prior to beginning the study. Blood chemistry screens were done by a local medical laboratory. All values, including those for thyroid status, fell within normal ranges. Medical clearance for participation in the study was obtained from all subjects' personal physicians, and all the women signed informed consent forms.

Participants were selected from an initial pool of over 200 women responding to information about the program via local newspapers and radio stations. Those meeting our criteria as described above were sent questionnaires detailing additional medical and weight history. From the questionnaires returned, pools based on childhood or adult-onset of obesity were used for the final selection of participants. Age of onset of obesity was determined by personal photographs, medical records, and/or school records. The two final groups, based on differences in age of onset of obesity and each consisting of 10 people, were matched according to age at the time of

the study and Metropolitan Relative Weight (MRW). Three people did not complete baseline testing, and 2 more were unable to participate in testing at the end of the program leaving 15 women on whom complete pre and post data were collected. This report is based on the results of these 15 women. In our final groups, 7 had obesity occurring prior to adolescence (childhood onset) and 8 had obesity occurring after age 18 (adult onset). Age of onset of obesity for CO was 8 $\pm$ 2 years (range 5-11) and for AO was 22 $\pm$ 3 years (range 19-26). Chronological age at the time of the study did not differ between the groups being 34 $\pm$ 5 and 33 $\pm$ 5 years for CO and AO, respectively.

Nutrition and Exercise Program

All subjects participated in a 9-month nutrition education and exercise program with an emphasis on forming new lifestyle habits that could be continued on a permanent basis. For the exercise portion, the women met in groups for 1 hour 3x/week. The groups walked for 40 minutes each session, preceded and followed by a 10 minute warmup/cool down period. Intensity of the exercise approximated 50% of each individuals' VO2 max which was determined during baseline testing and again at a point midway in the program by a graded treadmill test using a modified Balke protocol (Naughton & Haider, 1973). Nutrition education was provided during 1-1/2 hour classes held once each week during the first 4-1/2 months and two times per month the second 4-1/2 months. The classes emphasized permanent changes in eating habits and physical activity rather than dieting or weight loss. Information focused on developing a low-fat, high carbohydrate eating style and a regular practice of moderate physical activity. No more than a 500

kcal (2093 kJ) deficit per day from food was allowed during the 9 months and this was checked by collecting 3-day food records on a monthly basis. Weight was monitored on a weekly basis to ensure that the combination of a moderate calorie deficit and moderate increase in physical activity resulted in no more than 1 kg of weight loss per week. Attendance needed to be at least 80% for the nutrition education and walking classes to remain in the program, and all met that criteria.

Pre and Post Testing Periods

A 3-week testing period was conducted prior to and after the 9-month program to determine body composition, energy need, and activity level. The women were fed a controlled metabolic diet throughout the two testing periods which were used to estimate overall calorie need. Measures of fitness and body composition were made during the 2nd week and measures of resting metabolic rate during the 3rd week. The women were organized into groups based on similarities in menstrual cycle and began the 3 weeks of testing generally between days 3 and 7 of their menstrual cycle. With only 3 exceptions at baseline testing and 1 at the end-point testing, RMR's were measured between days 17 to 25 of their cycles.

Body density was determined by hydrostatic weighing (McArdle et al, 1981) and percent body fat calculated using the Siri formula (Siri, 1961). Residual lung volume was estimated at 28% of the largest of 3 repeated trials of forced vital capacity (McArdle et al, 1981; Astrand & Rodahl, 1977). Fat free mass (FFM) was calculated by subtracting fat

weight from total body weight.

To determine calories needed to maintain body weight, the women consumed the controlled metabolic diet for the entire 3-weeks of each testing period. The diet consisted of commercially available foods and was prepared in the metabolic kitchen in the Foods and Nutrition Department. The foods consumed were the same each day and are listed in Table 1. Foods and amounts were adjusted so that the percent distribution of calories remained consistent for each person at 26%, 14%, and 60% for fat, protein, and carbohydrate, respectively. Determination of calorie and nutrient content of the diet was made using the Ohio State Nutrient Data Base, 1984 Edition (Schaum et al, 1973). The diet, in addition, served an educational function in demonstrating to the women the types and proportions of food that could be consumed to achieve a low-fat, high-carbohydrate intake.

Foods were purchased in bulk at the beginning of each metabolic diet period with the exception of fluid milk which was purchased from the same vendor on a weekly basis. Foods and beverages were weighed to within 0.1 g and individually packaged. The women ate their dinner in the metabolic kitchen and took home their food for breakfast, lunch, and snacks. Water, tea, coffee, and diet soda were consumed as desired, although no caffeinated beverages were allowed during the third week when resting metabolic rate was measured. Body weight was measured each day before dinner on a balance beam scale within 0.1 kg. Calories were adjusted when a consistent trend in weight change was seen for 3 consecutive days. Table 1 indicates which foods were used

to adjust calorie intake while maintaining the percent calories as fat, protein, and carbohydrate as described above. Mean body weight of the group stabilized by the 10th day of both the pre and post feeding periods, with a mean change of only −0.5 kg ± 0.7 between the 10th and 20th days of the metabolic diet occuring during both testing periods. Calories needed to maintain body weight are the average intakes for the last 10 days of each metabolic diet period.

Values for resting metabolic rate (RMR) are the average of measurements taken on 4 separate mornings during the third week of each testing period. The women reported to the lab at 6:30am, 12−hours fasted. Following a 30−minute rest period, RMR was measured. The women breathed through a Daniels' Type low resistance two−way breathing valve. After a 2 minute adjustment period, expired air was collected for 5 minutes into a 120−liter meteorological balloon. A sample of the expired air was analyzed immediately following collecton for CO2 and O2 using a Beckman LB−2 infrared carbon dioxide analyzer and an Applied Electrochemistry S−3A oxygen analyzer. Both instruments were calibrated with gases of known concentration immediately before each measurement. Total volume of expired air was measured using a Parkinson−Cowan CD−4 spirometer at the pre testing and a 120 L Tisot at the post testing. Volume was corrected to STPD and RQ calculated. Kcals were calculated using Weir's formula (Weir, 1949). Measurements of resting metabolic rate, body composition, and VO2 max were conducted in the facilities of the Human Performance Lab in the College of Health and Physical Education.

During each of the 3-week testing periods the women kept 24-hour activity logs. Activities were divided into 4 general categories including sleeping, sitting, light activities and moderate activities. Light activities included standing, slow walking, driving, light house and yard work. Moderate activities were those equivalent to the program's exercise-intensity activities and included brisk walking and vigorous house and yard work.

Statistical Analysis

A repeated measures ANOVA procedure using the BMDP Statistical Package (1987) was used to analyze the data by the age of onset subgrouping. In this procedure, a significant interaction indicates a different pattern of change from pre to post for the two groups being compared (CO vs AO). A significant time effect indicates a change from pre to post that is similar in the two groups, a significant group effect denotes no significant change over time but a significant difference in the group means. For analyzing the data involving the entire group of 15 women, Pearson product-moment correlations were used (Steel & Torrie, 1980). Probability values were based on two-tailed tests and level of significance set at $p<.05$.

RESULTS

Body weight and body composition data before and after the program are presented in Table 2. The only significant difference between the CO and AO groups at baseline in these variables was height, with the CO group being taller. Though the other variables were not significantly different, the CO group weighed slightly more and had a higher fat-free mass (FFM) than the AO group. However, mean percent body fat was essentially identical in the two groups. Both groups lost weight over the 9 months with the pre to post decrease reaching significance ($p<.05$) for both groups. Mean weight change was -5.7 and -3.3 kg for the CO and AO groups, respectively. The CO group had a greater decrease in body weight than the AO group, however the difference between the groups was not significant. Most of the weight lost was fat weight with very little loss of lean tissue. Mean fat weight change was -5.8 and -2.8 kg for the CO and AO groups, respectively. BMI and MRW values decreased from pre to post by percentages similar to that of body weight change for both groups.

Table 3 contains the data for total calorie need prior to and following the 9-month program. At baseline the CO group needed about 200 kcals (837 kJ) per day more than the AO group to maintain body weight. When expressed on a relative basis as kcals per kg FFM per day, the CO group continued to have a slightly higher need for total calories. However, neither difference between the groups was stastically significant. At the end of the program total calories needed to maintain body weight were, on the average, 3% higher compared

to the beginning for both groups. This increase was not statistically significant, however, because of the wide variation in calorie need among individuals. Total calorie need expressed per kg FFM increased 3% and 4% for the CO and AO groups, respectively. This increase from pre to post was statistically significant for both groups because of the very small variation around the means.

Calories per day needed for resting metabolic rate (RMR) are shown in Table 4. At baseline, mean calories needed for RMR were, like total calories, higher for the CO group, although only by about 100 kcals (419 kJ), and the difference was not significant. When expressed per kg FFM, calorie needs for RMR were identical in the two groups. Unlike total calorie need, mean RMR decreased from pre to post in both the CO and AO groups by about 3% when expressed as calories per day. Calories per kg FFM needed for RMR decreased by 4% and 2% for the CO and AO groups, respectively. The decreases in calories needed for RMR pre to post were statistically significant for both groups but there were no significant differences between the two groups. At baseline testing, RMR accounted for an average of 70% of total calorie need; at the end of the program, 65% of the calories needed per day were used for RMR.

Hours spent per day in the four activity categories are presented in Table 5. The two groups spent similar amounts of time in each of the categories at baseline and had similar changes at the end of the program. From pre to post there was a decrease in time spent sitting and a substantial increase in time spent in both light and moderate

activities. These pre to post changes were statistically significant for both groups.

In essence, the CO and AO groups did not differ in body composition or energy need before or after this program. Both groups had significant decreases in mean body weight and body fat, with little change in mean FFM. Both groups had a higher average need for total calories to maintain body weight at the end of the program than the beginning, though the increase for both was only significant when expressed on a fat-free mass basis. Both groups had significantly lower mean RMRs at the end of the program when expressed on both an absolute and relative basis. At the same time, hours spent in sedentary activity decreased and time in non-sedentary activity increased similarly for both groups. Because the two groups responded similarly to the 9-month program, the age of onset distinction was dropped to consider a further question. That is, how did weight change affect the need for calories to maintain body weight?

In Table 6 are listed the pre to post changes in body composition and energy need. Data are arranged in descending order of weight lost in the group of 15 women. Body weight change over the 9 month program ranged from -18.7 to +8.7 kg, while the change in FFM covered a much more narrow range of from -2.9 to +2.3 kg. The correlation between change in body weight and change in FFM was relatively high with $r = 0.74$ ($p<.01$). The correlation between change in body weight and change in fat weight was even higher at $r = 0.99$ (individual data not shown) which would be expected since most of the weight lost in this group was

fat weight. In general, there was a need for more total calories per day to maintain body weight at the end of the program than at the beginning, with a mean increase from pre to post of +74 kcals/day. Those who gained weight from pre to post had the biggest increases in calorie need per day; however, the opposite was not necessarily true. Those who lost the most weight did not consistently have a reduced need or the smallest increase in need for calories to maintain body weight from pre to post. The correlation between change in body weight and change in need for total calories at $r = 0.65$, reflects a significant relationship but indicates that other factors must also be considered.

Changes in RMR followed the pattern of changes in body weight from pre to post much more closely than did changes in total calorie need. This was reflected in the highly significant correlation of $r = 0.85$ between change in body weight and change in RMR. For each 10 kg change in body weight in either direction, a corresponding change in RMR of about 100 kcals (419 kJ) occurred (regression equation: change in RMR = $-$ 9.40 + 10.50(change in kg body weight), S.E.E.= 52 kcals/day). The correlation between the change in FFM and the change in RMR was lower than that between changes in body weight and RMR, though both were statistically significant.

Changes in time spent in non-sedentary activity increased in all but 2 of the 15 women with a mean increase of slightly more than 1 hour per day. The correlation between change in activity and change in either body weight or FFM was very low and not significant.

DISCUSSION

An aim of this nutrition education and walking program was to promote gradual fat loss, maintain FFM, and avoid the decrease in energy need which commonly follows more drastic approaches to weight reduction. The minimal loss of FFM and slight increase in calories needed to maintain weight at the end of this study reflect the success of that effort.

The weight loss in this study was similar to that found in other studies which have included moderate changes in dietary intake, physical activity, and behavior management (Lewis et al, 1976; Zuti & Golding, 1976; Harris & Hallbauer, 1973; Stalonas et al, 1978; Dahlkoetter et al, 1979), and, as expected, less than that found when exercise was combined with very low calorie intakes (van Dale et al, 1987; Hill et al, 1987; Warwick & Garrow, 1981). Of the weight lost in this program, 93% was due to fat loss. This result is consistent with that found in similar programs stressing moderate changes in diet and exercise habits (Lewis et al, 1976; Zuti & Golding, 1976). Studies with greater calorie deficits and more rapid weight loss achieved either by diet and exercise (Hill et al, 1987; van Dale et al, 1987; Belko et al, 1987) or diet alone (Bessard et al, 1983; Dore et al, 1982; Warnold et al, 1978; de Boer et al, 1986) have demonstrated a loss of fat ranging from 58 to 84% of tissue lost. The loss of FFM in these studies was much greater than the 7% of tissue lost in the present study. Ravussin et al (1982) have stressed the importance of maintaining lean body mass during weight reduction since energy need is

primarily dependent on lean tissue.

The level of calories needed to maintain body weight at baseline in this group of women is similar to that needed by others of comparable body weight (de Boer et al, 1986; Blair & Buskirk, 1987; Ravussin et al, 1985). When expressed on a relative basis, calories needed approximated 25 kcal (105 kJ)/kg BW/day and 48 kcal (201 kJ)/kg FFM/day. This is within the range of 24-30 kcal (100-126 kJ)/kg BW and 40-49 kcal (167-205 kJ)/kg FFM found in obese subjects by others (Blair & Buskirk, 1987; Ravussin et al, 1982; Bessard et al, 1983; Leibel & Hirsch, 1984; Warnold et al, 1978; Bradfield & Jourdan, 1972; de Boer et al, 1986; Belko et al, 1987; Ravussin et al, 1985). Calorie need determined in this study can be considered a conservative estimate since a mean loss of 0.5 kg body weight occurred in the last 10 days of both the pre and post feeding periods. If the loss was due to adipose tissue rather than water weight flucuation, calorie need may be underestimated by as much as 385 kcal (1612 kJ)/day (assuming 1 kg = 7700 kcal). Blair & Buskirk (1987) found their adult-onset obese women needed significantly more calories than their child-onset obese women on a FFM basis. This difference was not found in the present study where the CO group had a slightly, but not significantly, higher calorie need than AO on a FFM basis.

Total calories needed to maintain body weight at the end of this program increased an average of 3% for both the CO and AO groups. This finding is in contrast to most studies which report a decrease in 24 hour calorie need ranging from 9 to 40% following weight loss (Bessard

et al, 1983; Leibel & Hirsch, 1984; de Boer et al, 1986; Ravussin et al, 1985; Warnold et al, 1978). One reason for this difference may be that the subjects in these studies have had average weight losses in the range of 10 to 15 kg, with a mean loss in one study of 52 kg (Leibel & Hirsch, 1984), all of which are greater than the average weight loss in our study. However, 4 of the 5 women in the present study who lost more than 10 kg of body weight did not have reduced calorie needs at the end of the 9-months and the calorie need of the 5th woman decreased by only 4%.

Three components of energy expenditure can be considered when evaluating a change in calorie need following weight loss: resting metabolic rate, thermogenesis, and physical activity (Ravussin et al, 1985). In this paper we address two of these components, RMR and physical activity, with data on thermogenesis to be presented in a later publication.

In the present study, a decrease in RMR was observed even though a slightly increased need for total calories was found from pre to post. The mean decrease of 3% was very small, however, in comparison to the decreases of 9 to 19% found in very low calorie diet plus exercise studies (van Dale et al, 1987; Hill et al, 1987) and the decreases of 9 to 29% found in very low calorie diet only studies (van Dale et al, 1987; Ravussin et al, 1985; Hill et al, 1987; Apfelbaum et al, 1971; Bray, 1969; Welle et al, 1984; Warwick & Garrow, 1981; Bessard et al, 1983). With more moderate calorie deficits and mean weight losses from 5 to 7.5 kg, others (Belko et al, 1987, Lennon et al, 1985) have not

observed decreases in RMR in their diet and exercise groups and found only small or non-significant decreases in RMR in their diet only groups.

Comparison of the RMR determined in this study to those found in the studies cited above must be done cautiously. In the present study, RMR was measured under the same controlled, weight-maintaining dietary conditions both before and after the program. In addition, body weight had been stable for at least one month prior to both testing periods. Of all the studies cited above, only Bessard et al (1983) measured post weight loss RMR after a period (7 days) of weight maintenance, the other post measurements were done while the subjects were still consuming low-calorie diets. Other studies (van Dale et al, 1987, Lennon et al, 1985) did not have their subjects' diets controlled during RMR measurements so weight stability is not known.

Resting metabolic rate is believed to be determined primarily by FFM (Halliday et al, 1979; Ravussin et al, 1982; James et al, 1978; Hoffmans et al, 1979), and a loss of FFM to be primarily responsible for the decrease in RMR following weight loss (Ravussin et al, 1985). Because of the very small mean loss of FFM, we expected very little average change in RMR, and this was what we found. It was unexpected, however, that the change in RMR would be so very highly correlated with change in total body weight rather than with change in FFM. This suggests body weight, in addition to FFM, plays a role in determining RMR. Dore et al (1982) also found that body weight, total body potassium, and age together predicted RMR better than total body

potassium alone.

An increase in regular physical activity was a major goal in our program. The success of that effort is demonstrated by the significant increases in time spent both in light and moderate activities by the end of the program and the decrease in time spent sitting. However, even by the end of the present study, time spent sitting was 3 hours longer than that reported in child and adult-onset obese women by Blair and Buskirk (1987) and approximately 2 hours more than reported by Hoffmans et al (1979).

The energy cost of various daily activities was not directly measured in this study. However, energy cost above RMR averaged 757 kcals (3169 kJ)/day at baseline for the whole group and 886 kcals (3709 kJ)/day at the end of the program, an increase of 129 kcals (540 kJ)/day. The increase in calories used for activity more than compensated for the small decrease in RMR of 55 kcals (230 kJ)/day associated with a change in body weight and made possible a mean net increase in calories needed to maintain body weight at the end of the program compared to the beginning. This held true even for those women losing substantial amounts of body weight. While debate may still continue about whether exercise affects RMR during weight loss, what cannot be denied is the fact that an increase in regular physical activity will increase daily energy need.

In summary, results from this study indicate 1) RMR decreases with body weight loss even when up to 93% of weight is lost as fat, 2) the

decrease in RMR is very small, however, when the approach to weight loss involves a moderate decrease in calorie intake and a moderate increase in physical activity, 3) the calories needed to cover a moderate increase in physical activity can compensate for the slight decrease in RMR resulting from gradual weight loss so that calories needed to maintain body weight after weight loss can be equivalent to those needed prior to weight loss. In addition, those women with childhood-onset obesity in this study did not differ from those with adult-onset obesity in body composition or energy need before or after the nutrition education and exercise program.

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Table IV.1. Foods and quantities fed daily during the 3-week metabolic diets.

Breakfast	
Wheat flake cereal, fortified	45 g*
Raisins, seedless	28 g
Milk, 2%	245 g
Orange juice, frozen, reconstituted	249 g
Muffin, plain	80 g*
Margarine	10 g*
Lunch	
Bread, whole wheat	56 g*
Cheddar cheese	42 g
Dill pickle slices	15 g
Carrots, raw	72 g
Canned pears, light syrup	125 g
Apple juice, frozen, reconstituted	248 g
Vanilla wafers	40 g*
Snack	
Popcorn, air popped	24 g
Dinner	
Brown rice, uncooked weight	62 g*
Turkey breast	60 g
Corn, frozen	82 g
Peas, frozen	80 g*
Bread, whole wheat	56 g*
Margarine	10 g*
Salad:	
Lettuce, iceberg	55 g
Red cabbage	14 g
French dressing	32 g*
Ice milk	98 g*

Percent calories:

Protein	14%	Fat	26%	Carbohydrate	60%

*Quantity of these foods adjusted to meet individual caloric requirements.

Table IV.2. Data on height, weight, Metropolitan Relative Weight (MRW), body mass index (BMI), percent body fat, and fat-free weight (FFW) before and after the 9-month program (pre and post) grouped by age of onset of obesity.

S#	Height	Weight		MRW		BMI		Body Fat		FFW	
	cm	kg		%		W/H2		%		kg	
		pre	post	pre	post	pre	post	pre	post	pre	post
Childhood Onset (CO):											
1	169.2	112.6	93.9	179	149	39.3	32.8	50	42	56.3	54.4
2	170.8	99.9	87.4	156	137	34.2	30.0	46	39	54.4	53.3
3	166.7	81.2	83.7	131	135	29.2	30.1	38	40	50.0	50.2
4	171.1	107.9	97.3	169	152	36.9	33.2	49	43	54.9	55.6
5	175.6	91.4	85.7	138	130	29.6	27.8	43	39	52.4	52.1
6	166.4	88.3	97.0	142	156	31.9	35.0	45	47	48.9	51.2
7	168.0	107.2	104.2	173	168	38.0	36.9	50	48	53.4	54.1
Mean	169.7	98.4	92.7	155	147	34.2	32.2	46	43	52.9	53.0
± SD	3.2	11.7	7.4	19	13	4.1	3.2	4	4	2.7	1.9
% Change		- 5.8%*		- 5.2%*		- 5.8%*		- 6.5%*		+ 0.1%	
Adult Onset (AO):											
1	157.8	84.6	81.0	148	142	34.0	32.5	42	42	48.9	47.2
2	153.7	74.0	72.5	137	134	31.3	30.7	40	40	44.4	43.4
3	163.8	113.1	101.1	188	168	42.2	37.6	47	44	59.5	56.6
4	162.0	101.9	105.4	173	179	38.8	40.2	46	46	55.1	57.3
5	155.2	105.2	110.4	192	201	43.8	45.8	53	54	50.0	50.8
6	162.6	83.7	80.4	140	134	31.6	30.4	44	42	47.0	46.9
7	156.2	83.2	71.4	149	128	34.1	29.3	47	39	44.4	43.4
8	163.3	99.4	97.4	168	165	37.1	36.4	43	41	57.1	57.1
Mean	159.4	93.2	89.9	162	156	36.6	35.4	45	43	50.8	50.3
± SD	4.1	13.6	15.4	21	26	4.7	5.7	4	5	5.8	6.0
% Change		- 3.5%*		- 3.7%*		- 3.3%*		- 3.3%*		- 1.0%	

*Significant time effect, eg. significant change from pre to post ($p<.05$). No significant differences between CO and AO for any variables except height, which was significantly different at $p<.001$.

Table IV.3. Calories needed to maintain body weight expressed as total calories and as calories per kg fat-free weight (FFW) before and after the 9-month program (pre and post) grouped by age of onset of obesity.

S#	Kcals/day		KJ/day		Kcals/kg FFW/day		KJ/kg FFW/day	
	pre	post	pre	post	pre	post	pre	post
Childhood Onset (CO):								
1	2614	2641	10942	11055	46.4	48.5	194	203
2	2710	2734	11344	11444	49.8	51.3	208	215
3	2539	2743	10628	11482	50.8	54.6	213	229
4	2900	2769	12139	11591	52.8	49.8	221	208
5	2530	2537	10591	10620	48.3	48.7	202	204
6	2306	2607	9653	10913	47.2	50.9	198	213
7	2545	2641	10653	11055	47.7	48.8	200	204
Mean	2592	2667	10850	11164	49.0	50.4	205	211
± SD	182	84	762	352	2.3	2.2	10	9
% Change		+ 2.9%				+ 2.9%*		
Adult Onset (AO):								
1	2463	2281	10310	9548	50.4	48.3	211	202
2	2112	2216	8841	9276	47.6	51.1	199	214
3	2932	2916	12273	12206	49.3	51.5	206	216
4	2539	2717	10628	11373	46.1	47.4	193	198
5	2161	2537	9046	10629	43.2	49.9	181	209
6	2161	2281	9046	9548	46.0	48.6	193	203
7	2072	2131	8673	8920	46.4	49.1	194	206
8	2764	2713	11570	11357	48.4	47.5	203	199
Mean	2400	2474	10046	10356	47.2	49.2	198	206
± SD	326	286	1356	1197	2.3	1.5	10	6
% Change		+ 3.1%				+ 4.2%*		

*Significant time effect, eg. significant change from pre to post ($p<.05$). No significant differences between CO and AO for any variables.

Table IV.4. Resting metabolic rate expressed as total calories and as calories per kg fat-free weight (FFW) before and after the 9-month program (pre and post) grouped by age of onset of obesity.

S#	Kcals/day		KJ/day		Kcals/Kg FFW/day		KJ/Kg FFW/day	
	pre	post	pre	post	pre	post	pre	post
Childhood Onset (CO):								
1	1776	1613	7434	6752	31.5	29.5	132	124
2	1861	1670	7790	6991	34.2	31.2	143	131
3	1678	1786	7024	7476	33.5	35.5	140	149
4	1947	1814	8150	7593	35.5	32.6	149	136
5	1597	1498	6685	6271	30.5	28.8	128	121
6	1782	1872	7459	7836	36.4	36.5	152	152
7	1823	1757	7631	7355	34.1	32.6	143	136
Mean	1781	1716	7455	7183	33.7	32.4	141	136
± SD	116	130	486	544	2.1	2.9	9	12
% Change		- 3.6%*				- 3.9%*		
Adult Onset (AO):								
1	1609	1584	6735	6631	32.9	33.4	138	140
2	1572	1469	6580	6149	35.4	33.6	148	141
3	1990	1858	8330	7778	33.4	32.9	140	138
4	1700	1685	7116	7053	30.9	29.5	129	124
5	1597	1699	6685	7112	31.9	33.6	134	141
6	1623	1598	6794	6682	34.5	34.1	144	143
7	1557	1483	6518	6208	35.1	34.1	147	143
8	1885	1786	7891	7476	33.0	31.2	138	131
Mean	1692	1645	7083	6886	33.4	32.8	140	137
± SD	160	138	670	578	1.6	1.6	7	7
% Change		- 2.8%*				- 1.8%*		

*Significant time effect, eg. significant change from pre to post ($p<.05$). No significant differences between CO and AO for any variables.

Table IV.5. Average hours per day spent in 4 activity categories before and after the 9-month program (pre and post) grouped by age of onset of obesity.

S#	Sleeping		Sitting		Light activity		Moderate activity	
	pre	post	pre	post	pre	post	pre	post
Childhood Onset (CO):								
Mean	8.0	7.9	12.4	10.7	3.5	4.9	0.14	0.53
± SD	0.9	0.5	1.3	1.7	1.6	2.2	0.08	0.21
% Change	- 1.0%		- 14%*		+ 40%*		+ 280%*	
Adult Onset (AO):								
Mean	7.7	7.5	11.8	9.9	4.4	6.2	0.14	0.40
± SD	0.9	0.6	1.9	2.1	1.8	2.2	0.07	0.10
% Change	- 3%		- 16%*		+ 41%*		+ 186%*	

*Significant time effect, eg. significant change from pre to post ($p<.001$). No significant differences between CO and AO for any variable.

Table IV.6. Pre to post changes in body composition and energy need variables listed in descending order of weight lost over the 9-months, and correlations of each variable with change in body weight and change in FFW.

S#	Change in Body Weight kg	Change in FFW kg	Change in Total Calorie Need kcals/day	Change in RMR kcals/day	Change in Light plus Moderate Activity hours/day
CO-1	- 18.7	- 1.9	+ 27	- 163	+ 3.7
CO-2	- 12.5	- 1.1	+ 24	- 191	+ 1.3
AO-3	- 12.1	- 2.9	- 16	- 132	+ 3.6
AO-7	- 11.8	- 0.9	+ 59	- 74	+ 3.8
CO-4	- 10.6	+ 0.7	- 131	- 133	- 0.3
CO-5	- 5.7	- 0.4	+ 7	- 99	+ 1.9
AO-1	- 3.6	- 1.7	- 182	- 25	+ 0.9
AO-6	- 3.3	NC	+ 120	- 25	+ 1.8
CO-7	- 3.0	+ 0.7	+ 96	- 66	+ 2.0
AO-8	- 2.0	NC	- 51	- 99	- 0.3
AO-2	- 1.5	- 1.0	+ 104	- 103	+ 1.3
CO-3	+ 2.5	+ 0.2	+ 204	+ 108	+ 1.9
AO-4	+ 3.5	+ 1.4	+ 178	- 15	+ 2.2
AO-5	+ 5.0	+ 0.9	+ 376	+ 102	+ 3.9
CO-6	+ 8.7	+ 2.3	+ 301	+ 90	+ 2.1
Mean	- 4.3	- 0.3	+ 74	- 55	+ 1.2
± SD	7.6	1.4	150	94	1.3
Correlation with change in body weight:					
r =	--	0.74**	0.65**	0.85***	- 0.06
Correlation with change in FFW:					
r =	0.74**	--	0.60*	0.59*	- 0.20

*p<.05
**p<.01
***p<.001

CHAPTER V

NUTRIENT INTAKE, AEROBIC FITNESS AND BODY WEIGHT IN WOMEN WITH CHILDHOOD AND ADULT ONSET OBESITY BEFORE AND AFTER A 9-MONTH NUTRITION EDUCATION AND WALKING PROGRAM

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ABSTRACT

Seven women with childhood-onset obesity (CO) and eight with adult-onset obesity (AO) participated for 9 months in a nutrition education and walking program which emphasized a low-fat eating style, moderate and regular physical activity, and gradual body fat reduction. Nutrient intake, nutritional status, fitness, and weight were evaluated before and after the program. Over the 9 months % kcals as fat decreased from 46 $\pm$ 4 to 29 $\pm$ 2% for CO and from 42 $\pm$ 6 to 36 $\pm$ 6% for AO. Total calorie intake decreased from 2557 $\pm$ 572 to 1580 $\pm$ 354 kcals for CO and from 1867 $\pm$ 550 to 1689 $\pm$ 261 for AO. Despite significant decreases in calories both groups maintained or increased their intakes of iron and B6, two nutrients women generally consume in amounts well below the RDA. Fitness improved significantly in both groups with maximal aerobic capacity (VO2 max) increasing by 8% for CO and 7% for AO. Weight decreased significantly by 5.7 kg for CO and 3.3 kg for AO with 93% of the weight loss being due to fat loss. Change in dietary fat intake was related to age of onset of obesity but weight change was not. Weight change was, however, significantly correlated with body fat distribution. Those women with predominately upper body fat lost significantly more weight than those with predominately lower body fat.

KEY WORDS: low-fat diet, exercise, nutritional status, age of onset of obesity, waist/hip ratio, weight loss

INTRODUCTION

Current nutrition recommendations for health improvement and disease prevention include, among other things, decreasing dietary fat and attaining ideal body weight (Dietary Guidelines for Americans, 1985; American Heart Association, 1986). Recommendations for weight control in people with mild and moderate levels of obesity currently call for the combined use of aerobic exercise, moderate calorie restriction, and behavior modification (American College of Sports Medicine, 1983; Weinser et al, 1984; Stunkard, 1984; Brownell, 1984). Further, achieving this weight control by permanent lifestyle change rather than temporary measures is highly recommended (Weinsier et al, 1984; Hirsch, 1985). Because a dietary intake low in fat spontaneously decreases calorie intake to a moderate level (Duncan et al, 1983; Lissner et al, 1987) with accompanying weight loss (Lissner et al, 1987), a combination of the above recommendations could be that the best approach to weight control is one that combines an habitual low-fat dietary intake with regular aerobic exercise.

Though this approach for reducing and controlling body weight might find widespread agreement and support, there is considerable disagreement about the goal, eg. what is considered the ideal weight one should achieve. Recommendations range from reducing weight to no more than 10% above the 1959 Metropolitan Relative Weight (Manson et al, 1987) to not treating obesity at all, especially in women (Wooley & Wooley, 1984). However, other researchers are suggesting that weight goals be determined individually depending on medical and family weight

history (Callaway, 1984; Van Itallie, 1985).

Because conventional weight loss methods meet with very limited success (Stunkard & McLaren-Hume, 1959; Wing & Jeffery, 1979; Berchtold & Van Itallie, 1985), some have suggested that strong physiological forces resist loss of body weight below a certain point (Nisbett, 1972; Keesey, 1980; Hirsch & Leibel, 1984). Those people with obesity beginning childhood may especially not be able to attain and maintain a body weight anywhere near what might be considered "ideal" (Grinker & Hirsch, 1972; Bjorntorp et al, 1975; Krotkiewski et al, 1977). Additionally, Bjorntorp (1985a) has suggested that those women with predominately lower body fat may be less able to decrease body fat stores than those with predominately upper body fat.

A "best" body weight might result from habitually consuming a low-fat diet moderately decreased in calories, and habitually participating in regular, moderate-intensity aerobic exercise. This may be true whether or not the weight attained fits the various weight for height standards. To test this possibility, we offered a nutrition education and walking program to women with moderate obesity over a period of 9 months. Body weight change was observed rather than promoted. Women with childhood onset obesity comprised half the group studied, women with adult onset obesity made up the other half. Distribution of body fat was also determined and its relationship to body weight change investigated.

In addition to monitoring changes in dietary intake of calories

and fat, iron and vitamin B6 intake and status were followed. These two micronutrients were emphasized in the nutrition education classes and monitored for two reasons. Women consume both nutrients in amounts much lower than the RDA (Driskell & Chrisley, 1981; Raper et al, 1984). Intakes of both nutrients may be compromised by a low-fat, high-carbohydrate diet as bioavailability of both is lower in plant foods (Kabir et al, 1983; Raper et al, 1984).

The purpose of this project, therefore, was: 1) to evaluate the changes in dietary intake, nutrient status, aerobic fitness, and body weight in women with moderate obesity in response to a program emphasizing an habitual decrease in dietary fat intake and increase in aerobic physical activity, 2) to assess the effect age of onset of obesity has on the response to this type of program, and 3) to assess the effect body fat distribution has on weight change during a program of this type.

METHODS

Subject Selection and Participation

Over 200 women responded to information about the program which appeared in local newspapers and on radio. Initial screening was done by phone. Criteria for acceptance included being premenopausal, non-pregnant, free from chronic diseases, non-smoker, taking no medications or vitamin/mineral supplements, 25-40 years of age, 40-100% above the 1959 Metropolitan Standard for height, weight stable for at least two months, and sedentary. A total of 74 met the criteria and were sent a questionnaire asking for detailed medical, weight, and dieting history. Forty-one returned questionnaires and were divided into two groups based on age of onset of obesity. The childhood-onset obese group (CO), had obesity occurring prior to adolescence. Those comprising the adult-onset group (AO), developed obesity after attainment of adult stature. Determination of age of onset of obesity was made using photographs, medical records, and/or school records. From these two groups, 10 pairs matched for age and Metropolitan Relative Weight (MRW) were selected.

Prior to baseline testing a resting 12-lead EKG was done. Blood was drawn and sent to a local medical laboratory for a general chemistry screen. Results from the EKG and chem screens were forwarded to each person's personal physician for evaluation. A signed physician's approval form was required for each participant. Seventeen women completed baseline testing. During the 9-month progam one women dropped out due to change in job location and one woman was unable to

participate in the end-point testing because of work-related injuries. Thus, complete pre and post data were collected on 15 women and they comprise the group whose results are reported here. Approval for all aspects of the study was granted by the Human Subjects Committee at Oregon State University and all the women gave their informed consent.

Experimental Design

A nutrition education and walking program lasting nine months was preceded and followed by a three-week testing period. During both the pre and post testing periods, all subjects were fed a controlled, weight-maintaining diet (ref kcals paper). Prior to each testing period, food intake was recorded for three consecutive days. During the first week of each testing period blood was drawn in the fasting state for iron and vitamin B6 status measurements. Fitness and body composition determinations were done during the second week. During the last 10 days of each testing period 24-hour urine collections were made to determine urinary nitrogen excretion. Throughout the nine-month program all the women were in a free-living situation, making their own food selections. During the testing periods and 9-month program they were requested to take no vitamin or mineral supplements.

Nutrition Education and Walking Program

The women met weekly for a 1-1/2 hour nutrition education class during the first half of the program then twice a month for the second half. Topics in the classes included eating awareness; behavior modification; nutrient content of foods with a special emphasis on fat,

carbohydrate, iron, and vitamin B6; eating differently around family and friends; eating out; trying new foods; enhancement of self esteem; and the benefits of regular, moderate physical activity. Though all topics were considered important, a major emphasis was placed on encouraging a dietary change to a low-fat intake of between 25-30% kcals as fat, and a corresponding increase in percent calories as carbohydrate, primarily in the form of foods containing starch and fiber. The classes were team-taught by a Registered Dietitian and certified fitness instructor.

In the third month of the program, eating plans based on consuming primarily low-fat, high-complex carbohydrate foods were given to the women along with a recommended calorie intake that was about 500 kcals less than that determined during baseline testing to maintain their body weight (reference kcal paper). Following these plans was suggested but not required, and the women were encouraged to tailor the eating plans to meet their individual circumstances. Three-day food records were collected monthly and body weight was monitored weekly to insure that reductions in intake greater than 500 kcals/day were not being made.

Exercise consisted of walking. The women participated in groups 3x/week throughout the program for one hour. Ten minutes were spent in warm-up and cool-down activites and 40 minutes spent walking. Intensity of walking approximated 50% of each person's measured VO2 max and was monitored periodically by checking heart rate. Additional walking on other days of the week was encouraged. Weekly logs of time

spent in exercise activities were recorded throughout the program.

Data Collection

An overall compliance (attendance) of at least 80% was required to remain in the program. For the exercise component, 40 minutes of activity at an intensity equal to 50% of VO2 max three times per week (total 120 minutes) equalled 100% compliance. For the nutrition education portion, 100% compliance equalled attending all 20 classes. An overall program compliance score was determined in which a percent compliance no greater than 100% for the exercise component was combined with the nutrition education component so that equal weight was given to both parts of the program.

Intake of calories and nutrients was determined by three-day food records. These were obtained prior to baseline testing, monthly during the program, and just prior to the testing period at the end of the program. The dietary intake recorded prior to baseline was obtained in December. Because of concerns about holiday influences on food intake, an additional three-day food record was taken early in January. No significant difference was found in nutrient intake between the December and January records so those values were averaged and used as baseline dietary intake. A final 3-day food record was taken the following September for end-point intake. Seasonal differences between the December/January mean and September were not taken into account as several recent studies have found little or no seasonal variation in intakes of calories and nutrients (Windham et al, 1983; Kim et al, 1984; Van Staveren et al,1986). The three-day period for recording

food intake included both weekdays and weekends (Thompson et al, 1986). Food and beverage items were coded and computer analyzed using the Ohio State Nutrient Data Base, 1984 Edition (Schaum, 1973).

Because accuracy of recorded food intake is always a concern, an estimation of accuracy was done by comparing dietary nitrogen intake and urinary nitrogen output (Briones et al, 1973; Huse et al, 1974; Arvidsson Lenner et al, 1977; Warnold et al, 1978; Isaksson, 1980). Nitrogen intake and output data were collected both while the women were on the controlled weight-maintaining metabolic diet and during a 3-day period when free-living food intake was being recorded. Twenty-four hour urine collections were done both while on the metabolic diet and during the 3-day food recording period. Dietary nitrogen intake was estimated indirectly for both the food records and the metabolic diet by computer analysis (Schaum et al, 1973) to obtain the value for grams of protein. That value was then divided by 6.25 to obtain an estimate of grams of dietary nitrogen. This value was compared to a direct measurement of dietary nitrogen consumed during the metabolic diet done by Kjeldahl analysis. Urinary nitrogen in all cases was measured directly by the Kjeldahl method (Scales & Harrison, 1929).

Iron and vitamin B6 status were determined during both pre and post testing periods. Blood was obtained on the third day after beginning the controlled metabolic diets. Blood was collected in the morning after a 12-hour fast. Hematocrit, hemoglobin, and percent saturation of transferrin (Weissman & Pileggi, 1974) were determined to

assess iron status. Plasma pyridoxal 5'-phosphate (PLP) was measured as an indicator of vitamin B6 status (Chabner & Livingston, 1970).

Maximal aerobic capacity (VO2 max) was determined by a maximal treadmill stress test using a calibrated Quinton motorized treadmill. A modified Balke procedure was followed (Naughton & Haider, 1973). Each subject walked at 2.5 mph for a brief warmup, then at 3.0 mph for the duration of the test. Following 3 minutes at 0% grade, elevation was increased by 5% every 3 minutes in the early stages and by 2.5% every 2 minutes in the later stages. Heart rate was monitored continuously. Measurement of O2 consumption was made via open circuit spirometry. Fitness measurements were done in the Human Performance Lab in the College of Health and Physical Education. Orientation to the equipment and procedures was done in an initial visit to the Lab. In addition, a maximal stress test was done prior to baseline testing for familiarization purposes and to assess reliability. Test-retest reliability of VO2 max was high at $r = 0.95$.

Body weight was monitored daily during the pre and post testing periods and weekly throughout the program. Body density was determined by hydrostatic weighing (McArdle et al, 1981) and percent body fat calculated using the Siri formula (Siri, 1961). Residual lung volume was estimated at 28% of the largest of three repeated trials of forced vital capacity (McArdle et al, 1981, Astrand & Rodahl, 1977). Fat-free mass (FFM) was calculated by subtracting body fat weight from total body weight. The women were initially oriented to the equipment and procedures for hydrostatic weighing, also done in the Human Performance

Lab, then completed a practice trial prior to both pre and post testing periods. Test-retest reliability of the determination of percent body fat was high with r = 0.94. During testing, circumference measurements were taken and pattern of fat distribution determined based on waist/hip ratio (WHR). Measurements used to determine WHR were waist at the smallest circumference and hips at the largest (Evans et al, 1984).

Statistical Analysis

A repeated measures ANOVA procedure using the BMDP Statistical Package (BMDP/PC, 1987) was used to analyze the pre and post data by the age of onset groupings. In this procedure, a significant interaction (INT) indicates a different pattern of change from pre to post for the two groups being compared (CO vs AO). A significant time effect (T) indicates a change from pre to post that is similar in the two groups, a significant group effect (G) denotes no significant change over time but a significant difference in the group means. For analyzing the data involving the entire group of 15 women, paired t-tests and Pearson product-moment correlations were used. Significance of probability values was based on two-tailed tests and level of significance set at $p<.05$.

RESULTS

Table 1 contains baseline descriptive data. No significant difference was found between the CO and AO groups for body weight, Metropolitan Relative Weight (MRW), Body Mass Index (BMI), fat-free mass (FFM), or % body fat (%BF). A significant difference ($p<.001$) in height was seen.

Overall program compliance was 92 $\pm$ 5% for the group of 15 women and ranged from 84 to 100%. Mean compliance with the exercise portion of the program equalled 110 $\pm$ 20%, indicating that some of the women were exercising in addition to the 3 supervised days per week. Range of exercise compliance was 75 to 150%. Attendance at the nutrition education classes ranged from 70 to 100% with a mean of 86 $\pm$ 10%. When comparing the CO and AO groups, there was no significant difference in compliance to the program. The CO group did, however, average 146 minutes each week in exercise activities while the AO group averaged 119 minutes/week.

Dietary intake as reported on the 3-day food records is presented in Table 2. Calorie intake was highly variable for both groups especially prior to the study. Reported calorie intake at baseline ranged from 1766 to 3286 kcals/day for CO and from 861 to 2562 kcals/day for AO, with the CO group reporting a mean calorie intake almost 700 calories greater than the AO group. However, because of the high variability, this difference was not significant. At the end of the program the range of reported intake was 1153 to 2099 kcals/day for

CO and 1340 to 2115 kcals/day for AO, with a mean difference of only 100 kcals between the two groups. The pre to post decrease in calorie intake was significant for both groups. Though the CO group made a greater decrease in calorie intake pre to post, the difference between the two groups was not statistically significant.

The focus of the nutrition education portion of the program was primarily to effect decreases in % kcals from fat and to increase % kcals from carbohydrate (CHO). Both groups decreased their intake of fat. The CO group, however, started out with a higher fat intake and ended with a lower intake than the AO group. The CO group also started out with a lower CHO intake and ended with higher % kcals as CHO than the AO group. This difference in pattern of change between the two groups for both fat and CHO was statistically significant. Change in % kcals as protein was slight and not significant for either group. Percent calories from alcohol was 3 $\pm$ 4% and 1 $\pm$ 3% for CO pre and post, and 1 $\pm$ 2% and 3 $\pm$ 4% for AO pre and post. Neither the pre to post change nor the difference in groups was significant for alcohol intake.

Intakes of iron and vitamin B6 were higher in the CO group at baseline, reflecting their higher kcal intake. When expressed on a mg/1000 kcals basis there was no difference in intake between the two groups for either nutrient. A special effort was made in the classes to encourage an adequate intake of iron and vitamin B6 while simultaneously making changes in fat and carbohydrate intake. Despite a significantly decreased intake of calories and fat, reported intake

of iron decreased only slightly for CO and increased slightly for AO. The pre to post changes were not significant for the two groups, but there was a significant group difference with the CO group reporting a higher total iron intake than AO both at baseline and at the end. When iron and vitamin B6 are expressed per 1000 kcals, both groups had similar intakes and made significant pre to post improvements in selection of foods more dense in these two nutrients. The B6/protein ratio also improved increasing from 0.015 $\pm$ 0.005 to 0.019 $\pm$ 0.006 in CO pre to post and from 0.012 $\pm$ 0.002 to 0.017 $\pm$ 0.005 in AO.

Nitrogen (N) intake and output data which were used for estimating accuracy of recording food intake are presented in Table 3. The grams of urinary N excreted + 2g is estimated to equal dietary N (Isaksson, 1980). The values obtained directly by Kjeldahl analysis of the samples collected during the metabolic diet periods came very close to that estimation. Dietary N estimated indirectly by computer nutrient analysis on these samples was consistently 1 g higher than that determined by Kjeldahl analysis. An average of 74% of dietary N was excreted in urine during the metabolic diet periods. In contrast, 94% of reported dietary N was excreted as urinary N while subjects kept food records, indicating an underreporting of dietary N intake. Calculating the proportion of urinary N as a % of dietary N during the metabolic diet to that of urinary N as a % of dietary N during the food record period gives an average value of 79 $\pm$ 17% for the group. That means, using the metabolic diet nitrogen input/output as a reference, the subjects, on average, recorded 79% of their actual nitrogen (protein) intake, or, put another way, underestimated their nitrogen

intake by an average of 21% ($p<.001$). There was no difference between the CO and AO groups in underestimation, with CO reporting a mean of 80% and AO a mean of 78% of their actual nitrogen (protein) intake.

Pre and post values for hematocrit, hemoglobin, % saturation of transferrin (%ST), and plasma pyridoxal 5'-phosphate concentration are presented in Table 4. For hematocrit and hemoglobin there was no significant difference between the CO and AO groups or between pre and post values. For %ST the AO group had significantly higher values than CO both pre and post. The slight decrease in %ST in both groups from pre to post was not statistically significant. Plasma PLP did not differ significantly between the CO and AO groups at either baseline or the end of the program. For both groups plasma PLP increased about 20% from pre to post, but the increase was not statistically significant. Status of iron and vitamin B6 as measured by these biochemical indicators was not compromised by a change to a lower fat, higher carbohydrate diet observed in these women.

Table 5 contains data on measures of aerobic fitness. There were no differences between the CO and AO groups in the pattern of change from pre to post for maximal aerobic capacity (VO2 max) which increased significantly. Because VO2 max expressed in ml O2 kg body weight min can increase just by weight loss, aerobic capacity expressed in L/min or on the basis of FFM gives a better indication of the actual increase in fitness. On a FFM basis, aerobic fitness increased 7-8% in both groups.

Body weight change over the program year is graphically presented in Figure 1. The zero point represents body weight measured during baseline testing. Pre-study weights were within 3kg of weights measured at baseline. Weights reported during initial recruiting were quite close to measured weights with only one exception. At the end of the first month, body weights were again within 3kg. By that time the supervised walking program was well under way while topics in the nutrition education classes were focusing on eating awareness and behavior modification techniques and weight change was not anticipated. During the third month of the program, information on fat, CHO, and calories in food was presented and the low-fat eating plans were distributed. At that point body weight change in the group began to spread out and continued to increase in variation throughout the remainder of the program. Four people fairly continuously gained weight during the program, 6 people sustained a decrease in weight throughout. The remaining 5 decreased weight somewhat after the third month, then stabilized or slowly regained weight. At the end of the eighth month, as requested, everyone began maintaining their body weight so that at least a month of stable weight would be achieved prior to the end-point testing period. For 11 women, post-study weights were obtained 1-2 months following the end-point testing period. For these women, body weight was within 3 kg of that measured at the end of the study.

For the CO and AO groups the mean decreases in body weight and %BF were significant from pre to post ($p<.05$), but the pattern of change for the two groups did not differ. Mean body weight at the end of the

program was 92.7 ± 7.4 kg for CO, a decrease of 5.7 kg; for AO body weight was 89.9 ± 15.4 kg at the end, a decrease of 3.3 kg. Mean %BF decreased from 46 ± 4% to 43 ± 4% for CO pre to post, and from 45 ± 4% to 44 ± 5% for AO. There was no significant loss of FFM from pre to post, with a mean change of +0.1 ± 1.4 kg and −0.5 ± 1.6 kg for CO and AO, respectively.

A correlation analysis of compliance and other variables relating to changes resulting from the program was determined for the total group of 15 women. Program compliance was significantly correlated with a decrease in dietary kcals as fat ($r=0.72$, $p<.01$). Compliance was also significantly correlated with change in body weight ($r=0.65$, $p<.01$) and waist/hip ratio ($r=0.71$, $p<.01$). Change in body weight was significantly related to WHR ($r=0.71$, $p<.01$), e.g. those women with higher WHRs (more upper body fat) tended to lose more body weight. Age of onset of obesity was significantly correlated with change in dietary fat ($r=0.73$, $p<.01$) and change in total calorie intake ($r=0.57$, $p<.05$) but not with change in body weight ($r=0.14$).

DISCUSSION

Compliance with this program was high with an attrition rate of only 12%. Drop out rates in exercise programs are very high and can vary from 30-70% or higher (Dishman, 1981; Oldridge, 1982). Drop out rates in weight loss programs are reportedly lower, ranging from 5-24% with the programs involving exercise experiencing the highest attrition rates (Wing & Jeffery, 1979). Programs long in duration also have a higher drop out rate. Kukkonen et al (1982) reported a 44% dropout rate in a 17-month exercise program and Stunkard (1987) reported dropout rates in 6 weight control programs to be more than 50% after 10-20 weeks and greater than 80% by 52 weeks. Compliance in terms of attendance was also high in this program falling within the range of 70-93% reported in other diet and exercise programs (Stalonas et al, 1978; Gale et al, 1984; Moody et al, 1969; Lewis et al, 1976; Franklin et al, 1979).

Mean calorie intake reported at baseline in the present study was higher than that reported currently by women in the general US population. Various nationwide dietary surveys conducted between 1971-1985 report average intakes for women of 1550-1770 kcals/day (Peterkin, 1986; Murphy & Calloway, 1986; Kurinij et al, 1986). Intake was higher in the LRC Prevalence Study at 1829 kcals/day (Gordon et al, 1982) and at baseline in the recently completed Family Heart Study at 1879 kcals/day (SL Connor, personal communication, 1987). Those reported intakes are similar to that of the AO group but still much lower than that found in the CO group at baseline.

Obese women in general have not been found to report higher caloric intakes than non-obese (Braitman et al, 1985; Anonymous, 1983) and some have found inverse relationships between body fatness and caloric intake (Baecke et al, 1983). Curtis and Bradfield (1971), however, reported calorie intakes ranging from 1801 to 2252 kcals/day in their study of 6 obese women, an intake more similar to that found in the present study. To our knowledge, no studies have reported dietary intake in women with childhood vs adult-onset obesity. Without that comparison it is hard to know whether the higher-than-average calorie intake seen in the CO group is some artifact of this study or has some basis related to early onset of obesity.

By the end of the 9-month program, reported calorie intake in both groups had decreased to the level reported in the national surveys cited above. A similar change was seen for women in the Family Heart Study in which a mean intake of 1639 kcals/day after 60 months of intervention was found (Connor, 1987).

Assuming that an underreporting of dietary nitrogen can be generalized to an underreporting of dietary calories (Huse et al, 1974; Isaksson, 1980), the average calories consumed by the entire group of women in this study, corrected for a 21% underestimation, approximated 2770 kcal prior to the program and 2078 kcal at the end. Those values can be further compared to actual calories needed to maintain body weight which were determined while on the controlled metabolic diet during the pre and post testing periods. The estimated kcals needed to

maintain body weight were 2490 ± 278 and 2564 ± 232 kcals at baseline and endpoint, respectively for the group of 15 women (ref kcals paper). If these women were actually consuming prior to baseline testing almost 300 kcals/day more than they needed to maintain body weight (2770 vs 2490 kcals) this would surely contribute to a continual positive energy balance. By the end of the program, a deficit of approximately 500 kcals/day (2564 vs 2078 kcals) was being consumed, based on corrected food record intake, rather than the almost 1000 kcals based on reported intake. Such a deficit of 500 kcals/day is the level of calorie reduction that was being strongly recommended in the nutrition education program as a means to promote gradual, long-lasting reduction in body fat. No differences were found in the CO and AO groups in accuracy of recording food intake.

Obese women have been found to make considerable errors in estimating food quantities and in keeping accurate food records (Lansky & Brownell, 1982). Most likely, however, that is not a problem unique to persons with obesity. Stern et al (1984) reported on a study with students, not selected on the basis of body weight, which compared records of food intake to observations of food intake. They found agreement ranging from 65-91%.

Percent of calories as fat reported at baseline were very high in both the CO and AO groups compared to the 36-38% reported in US surveys (Kurinij et al, 1986; Murphy & Calloway, 1986; Peterkin, 1986). Slightly higher fat intakes of 40% of kcals were reported for women in the LRC Prevalence Study (Gordon et al, 1982) and at baseline in the

Family Heart Study (Connor, 1987). Curtis and Bradfield (1971) reported an intake of 42% of kcals as fat in their obese women which is similar to the baseline intake in the AO group but still lower than that reported by the CO group.

In the present study dietary fat intake was reduced to 33% of kcals for the total group of 15 women. This decrease is similar to that found in larger scale dietary intervention programs in men and women with hyperlipidemia (Mojonnier et al, 1980; Gorder et al, 1986). A decrease to 33% kcals as fat was also found for women in the Family Heart Study (Connor, 1987). In both the Family Heart Study and the MRFIT program, the decrease in % kcals as fat occurred within the first year of intervention, a time frame similar to our program (Gorder et al, 1986; Connor, 1987). It is difficult to offer an explanation for why the CO group in this study reported a significantly higher fat intake at baseline and a lower fat intake at the end of the program compared with the AO group. With no other studies for comparison we present it only as an observation at this point.

Percent kcals as carbohydrate were lower at baseline than that found in other studies (Gordon et al, 1982; Peterkin, 1986; Connor, 1987) reflecting the higher fat intake in our group. By the end of the 9 months, reported % kcals as CHO increased in a manner similar to that found in the Family Heart Study reflecting the replacement of high-fat foods with those higher in carbohydrate.

Intakes of iron, and vitamin B6 at baseline were very similar to

the intakes of women found in nationwide surveys both on a total intake basis (Kurinij et al, 1986; Peterkin, 1986; Murphy & Calloway, 1986) and on a per 1000 Kcal basis (Windham et al, 1981). Iron and vitamin B-6 intakes were 50-60% of the RDA, reflecting the typical low intake of these two nutrients by women (Driskell & Chrisley, 1981; Raper et al, 1984). In spite of a substantial mean decrease in reported calorie intake at the end of the program, the intake of these two nutrients either remained the same or increased, reflecting a selection of foods of a higher nutrient density. To our knowledge, no other studies have reported data on nutrient density changes resulting from a dietary intervention program aimed primarily at reducing fat intake and increasing carbohydrate intake. These results indicate it is possible to make dietary changes in macronutrient intake without compromising the intake of micronutrients whose level of consumption is already marginal. Maintenance of normal status for iron and vitamin B6 throughout the study is a further indication that intakes of these two nutrients were sufficient to support metabolic needs. Hematocrit, hemoglobin, and % ST were within normal ranges both pre and post (Beutler 1980; Weissman & Pileggi, 1974) as were plasma PLP levels (Shultz & Leklem, 1981).

There was no difference in fitness level between the CO and AO groups before the program or as a result of the program. Baseline VO2 max values expressed in L $O_2 \cdot min^{-1}$ are similar to those found in untrained obese women (Bjorntorp et al, 1970; Bjorntorp et al, 1973; Kukkonen et al, 1982) and higher than those found in women of average body weight (Franklin et al, 1979; Astrand, 1960; Kilbom, 1971).

Conversely, VO2 max values expressed in ml O2·kg $BW^{-1}\cdot min^{-1}$ are substantially lower than those seen in average weight women due to excess fat weight (Franklin et al, 1979; Davies et al, 1975; Drinkwater, 1973; Astrand, 1960). Comparisons of initial or final fitness measures for people with obesity are best based on fat-free weight rather than total body weight (Buskirk & Taylor, 1957), and on that basis VO2 max in the present study compares favorably with values found in sedentary women without obesity (Franklin et al, 1973).

Percent increases in VO2 max due to training are influenced by several factors including initial fitness level and intensity of training. Generally, the less fit the person and the higher the intensity of training the greater will be the increase in VO2 max (Pollock 1973). This program emphasized a moderate level of physical activity (50% of VO2 max) to maximize the utilization of fat for fuel during exercise. Increases in VO2 max, therefore, would not be expected to be as high as those found in studies with higher training intensities. The 7% improvement in VO2 max expressed independently of weight change in this study is less than the 11-15% increases seen in other studies of obese individuals who were trained at intensites of 60-75% of VO2 max (Franklin et al, 1979; Kukkonen et al, 1982), and substantially less than the 27% increase found in non-obese middle aged adults trained at 75-85% of VO2 max (Getchell & Moore, 1975). However, Bjorntorp et al (1970) found a similar 7% increase in VO2 max in a group of women and men described as extremely obese who did high intensity interval training for 8 weeks and Kilbom (1971a) found a 6% increase in VO2 max in a group of average weight women who trained,

like those in the present study, at 50% of VO2 max.

Though weight loss was more a "side-effect" in this program than a goal, average weight lost was slightly more than that found in exercise programs, similar to that found in behavior therapy programs, and less than that found in either diet or drug therapy programs (Wing & Jeffery 1979). A greater percentage of people in this study lost more than 20 lb, 5 of 15 women or 33%, compared to 16-26% in the studies reviewed by Wing and Jeffery (1979). In studies most similar to this one that have combined dietary instruction with physical activity and behavior management over several months, mean weight losses are similar ranging from 4.2-7.3 kg (Harris & Hallbauer, 1973; Lewis et al, 1976; Zuti & Golding 1976; Stalones et al, 1978; Dahlkoetter et al, 1979).

Decrease in percent body fat was similar to that found in other studies of obese women participating in combination diet and exercise programs (Lewis et al, 1976; Zuti & Golding, 1976). A mean of 93% of the weight that was lost in this study was fat weight, a proportion again similar to that found in similar programs (Lewis et al, 1976; Zuti & Golding, 1976). It is possible with a moderate increase in physical activity and a moderate decrease in caloric intake to gradually lose weight as fat while maintaining lean body mass.

The CO and AO groups did not differ in weight or body composition changes as a result of the program. Prognosis for weight and fat reduction is supposedly poorer for those with childhood onset of obesity whether by diet (Bjorntorp et al, 1975; Krotkiewski et al,

1977) or exercise (Bjorntorp et al, 1970; Bjorntorp et al, 1973) because of the association of childhood onset obesity with high fat cell number or a hyperplastic type of obesity (Hirsch & Knittle, 1970; Brook et al, 1972; Salans et al, 1973; Sjostrom & Bjorntorp, 1974). However, Gwinup (1975) found that an exercise program consisting of walking was equally effective at reducing body weight whether women had an early or late onset of obesity. The type of physical training program used by Bjorntorp et al in hyperplastic obese adults (Bjorntorp et al, 1970; Bjorntorp et al, 1973; Sullivan, 1976) was interval training of a very high intensity which does not promote fat loss (Girandola, 1976; American College of Sports Medicine, 1978). That may be a factor in their inability to demonstrate body weight or fat loss in their subjects. We do not know for a fact that the women in the CO group had hyperplastic obesity since adipose tissue biopsies were not performed. However, like Gwinup, no differences were seen in the ability of the women in the CO and AO groups to lose body weight or body fat.

A stable, reduced body weight was not reached by any of the women in this study by the time they were requested to begin maintaining body weight after 8 months of the program. An approach which promotes permanent change will, by its nature, produce slow, gradual change in habits and therefore, in body weight. Since low fat eating plans were not introduced until 3 months into the program only 5 months actually occurred during which weight reduction might be expected. Bjorntorp (1976) found it took up to 8 months for men after a myocardial infarction to reach a stable, reduced body weight with an exercise

training program. With a dieting approach, reduced body weights stabilized in women after 4 to 12 months of consuming 1100 kcals/day (Bjorntorp et al, 1975). Twelve months or longer may be needed for women to achieve a stable, reduced body weight using the approach employed in the present study.

Weight loss in this study was significantly correlated with WHR. Those women with a predominance of upper body fat (WHR >0.80) lost more weight (-7.6 kg) than those with predominantly lower body fat (+2.3 kg) despite similar changes in dietary and activity habits. Bjorntorp (1985a) has suggested that because of differences in lipid-storing and lipid-releasing characteristics abdominal obesity (upper body) is easier to treat than gluteal-femoral obesity (lower body). Recently, Krotkiewski and Bjorntorp (1986) demonstrated that women with android-type obesity (upper body) averaged a small amount of body weight loss after a 3 month high-intensity physical training program while women with gynoid-type obesity (lower body) averaged a significant weight gain. Both groups experienced a mean gain in fat weight, though the gain was only significant for the group with gynoid-type obesity. Results from the present study support the suggestion that those women with a predominance of upper body fat experience a greater weight loss than those with lower body fat predominance even though participating to the same extent in an exercise and nutrition education program.

In this study, those who participated to the greatest extent tended to make the greatest changes in dietary fat intake and to lose

the most weight. Adams et al (1986) looked at factors significantly related to weight loss in group vs individual programs and found that one of 7 significant variables was the number of sessions attended. The more sessions attended by participants the greater was the weight loss. Results from this study confirm that finding. Compliance in this study was also highly correlated with WHR. It is hard to imagine some physiological reason based on body fat distribution that would explain this relationship. It is more likely that, despite the deemphasis on weight loss in this program, losing weight was nonetheless a motivating factor for continued participation in the program. Since those women with upper body fat were more likely to be losing weight they were also the ones likely to be participating more regularly.

Considering the results from this study, the following conclusions can be made: 1) nutritional status need not be compromised by a shift to a lower fat, higher carbohydrate diet, even when calorie intake is reduced, 2) reduction in body fat and maintenance of fat-free weight results from a program involving a moderate increase in physical activity and a moderate decrease in calories primarily from dietary fat, 3) 9 months is not sufficient time to reach a stable, reduced body weight using this approach in women with moderate obesity, 4) age of onset of obesity is not an important factor in successful change in food intake, fitness level, body weight, or body fat, 5) those with predominately upper body fat tend to lose more body weight/body fat than those with predominately lower body fat even when the same dietary and fitness changes are made, 6) program participation is a significant

factor in dietary change and in reduction of body weight/body fat. In addition, this study confirmed that a program of regular walking of moderate intensity increases aerobic fitness.

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Table V.1. Baseline descriptive data grouped by differences in age of onset of obesity. There was no significant difference between CO and AO for any variable except height, which was significantly different at $p<.001$.

S#	Age	Age of Onset	Waist/ Hip Ratio	Height cm	Weight kg	Metropolitan Relative Weight* %	Body Mass Index w/h2	Body Fat %	Fat-Free Weight kg
Childhood Onset (CO):									
1	31	6	0.86	169.2	112.6	179 (185)	39.3	50	56.3
2	34	9	0.84	170.8	99.9	156 (161)	34.2	46	54.4
3	39	6	0.77	166.7	81.2	131 (138)	29.2	38	50.0
4	30	8	0.81	171.1	107.9	169 (174)	36.9	49	54.9
5	37	11	0.82	175.6	91.4	138 (141)	29.6	43	52.4
6	29	11	0.70	166.4	88.3	142 (150)	31.9	45	48.9
7	41	5	0.87	168.0	107.2	173 (179)	38.0	50	53.4
Mean	34	8	0.81	169.7	98.4	155 (161)	34.2	46	52.9
± SD	5	2	0.06	3.2	11.7	19 (19)	4.1	4	2.7
Adult Onset (AO):									
1	36	22	0.82	157.8	84.6	148 (160)	34.0	42	48.9
2	28	24	0.79	153.7	74.0	137 (148)	31.3	40	44.4
3	32	19	0.83	163.8	113.1	188 (198)	42.2	47	59.5
4	29	19	0.84	162.0	101.9	173 (182)	38.8	46	55.1
5	31	20	0.75	155.2	105.2	192 (207)	43.8	53	50.0
6	36	24	0.74	162.6	83.7	140 (149)	31.6	44	47.0
7	42	26	0.83	156.2	83.2	149 (160)	34.1	47	44.4
8	28	19	0.81	163.3	99.4	168 (174)	37.1	43	57.1
Mean	33	22	0.80	159.4	93.2	162 (172)	36.6	45	50.8
± SD	5	3	0.04	4.1	13.6	21 (22)	4.7	4	5.8

* Values in parentheses are based on 1959 weight standards, other values are vased on 1983 Metropolitan Tables.

Table V.2. Reported dietary intakes before and after the 9-month program (pre and post) grouped by age of onset of obesity. Values are mean ± standard deviation.

	Childhood Onset			Adult Onset			
	Pre	Post	Pre/Post Change	Pre	Post	Pre/Post Change	Statistical Significance*
Total Kcals	2557 ± 572	1580 ± 354	-38%	1867 ± 550	1689 ± 261	- 9%	T, p<.01
% Kcals							
as Fat	46 ± 4	29 ± 2	-37%	42 ± 6	36 ± 6	-14%	INT, p<.01
as CHO	38 ± 5	55 ± 5	+45%	42 ± 6	46 ± 8	+ 9%	INT, p<.01
as Protein	13 ± 2	15 ± 2	+15%	15 ± 2	16 ± 4	+ 7%	NS
Iron							
mg	13.8 ± 2.2	12.4 ± 4.4	-10%	10.0 ± 3.0	11.2 ± 2.7	+12%	G, p<.05
mg/1000 Kcals	5.7 ± 1.4	7.9 ± 2.7	+39%	5.4 ± 0.6	6.7 ± 1.5	+24%	T, p<.01
Vitamin B6							
mg	1.2 ± 0.4	1.2 ± 0.5	NC	0.8 ± 0.3	1.1 ± 0.3	+38%	NS
mg/1000 Kcals	0.5 ± 0.2	0.7 ± 0.3	+40%	0.5 ± 0.1	0.7 ± 0.2	+40%	T, p<.01

*T indicates a significant pre to post change for both groups, INT indicates a change from pre to post that differs significantly between the two groups, G indicates no change from pre to post but a significant difference between groups.

Table V.3. Dietary nitrogen intake and urinary nitrogen output during two controlled metabolic diet periods vs during a 3-day period of recorded food intake.

	Metabolic Diet			Food Records
	Pre	Post	Average	
Dietary N, g (Kjeldahl)	12.68 ± 0.98	12.82 ± 0.75	12.76 ± 0.83	--
Dietary N, g (Ohio State Data Base, g protein/ 6.25)	13.65 ± 1.05	13.77 ± 0.87	13.71 ± 0.94	11.34 ± 2.54
Urinary N, g (Kjeldahl)	9.94 ± 0.90	10.16 ± 0.93	10.03 ± 0.91	10.61 ± 1.80
Urinary N as a percent of Dietary N (as estimated by computer analysis)	73%	74%	74%	94%

Table V.4. Biochemical indicators of iron and vitamin B6 status before and after the 9-month program (pre and post) grouped by differences in age of onset of obesity. Values are mean ± standard deviation.

	Childhood Onset			Adult Onset			
	Pre	Post	Pre/Post Change	Pre	Post	Pre/Post Change	Statistical Significance*
Iron							
Hematocrit	0.41 ± 0.02	0.40 ± 0.03	- 2%	0.42 ± 0.02	0.42 ± 0.02	NC	NS
Hemoglobin g/L	141 ± 7	142 ± 17	< 1%	148 ± 12	149 ± 8	< 1%	NS
% Saturation Transferrin	20 ± 7	19 ± 6	- 5%	28 ± 7	26 ± 4	- 7%	G, $p<.01$
Vitamin B6							
Plasma PLP nmol/L	38.3 ± 13.3	45.9 ± 31.5	+20%	35.9 ± 12.1	43.8 ± 6.5	+22%	NS

* G indicates no change from pre to post but a significant difference between groups.

Table V.5. Maximal aerobic capacity (VO2 max) before and after the 9-month program (pre and post) grouped by age of onset of obesity. Values are mean ± standard deviation.

	Childhood Onset			Adult Onset			
	Pre	Post	Pre/Post Change	Pre	Post	Pre/Post Change	Statistical Significance*
L O2/min	2.56 ± 0.22	2.76 ± 0.34	+ 8%	2.40 ± 0.24	2.52 ± 0.32	+ 5%	T, p<.01
ml O2/kg BW/ minute	26.1 ± 3.1	29.6 ± 2.3	+13%	25.8 ± 2.6	28.4 ± 4.8	+10%	T, p<.01
ml O2/kg FFW/ minute	48.5 ± 4.9	52.4 ± 5.1	+ 8%	47.4 ± 3.1	50.7 ± 6.1	+ 7%	T, p<.01

*T indicates a significant pre to post change for both groups.

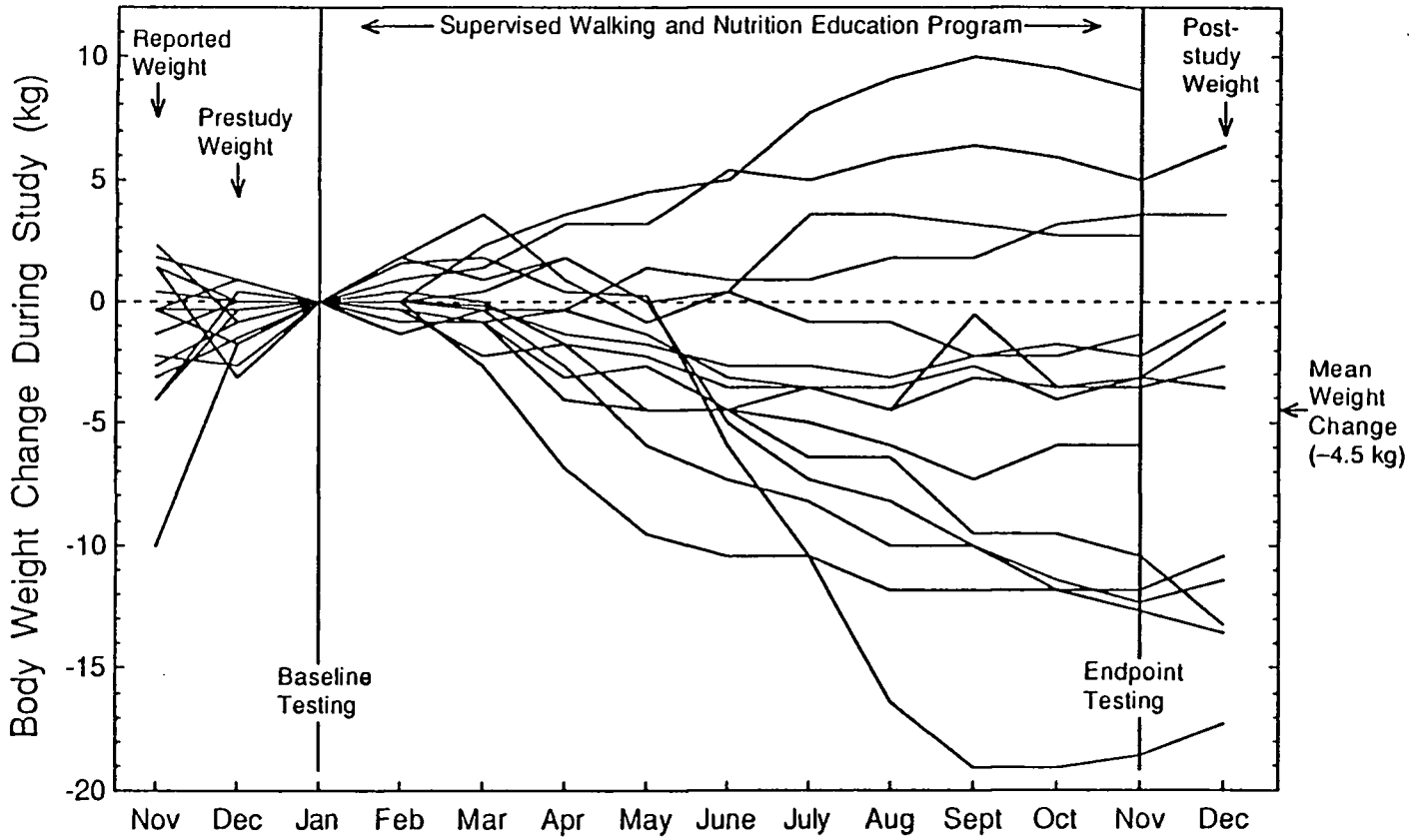

Figure V.1. Change in individual body weights during the study. Reported weight was obtained via questionnaire, prestudy weight measured during orientation procedures. Poststudy weight was measured on 11 subjects at a follow-up meeting.

CHAPTER VI

BLOOD LIPIDS, BLOOD PRESSURE AND GLUCOSE TOLERANCE IN WOMEN WITH CHILDHOOD AND ADULT-ONSET OBESITY BEFORE AND AFTER A 9-MONTH NUTRITION EDUCATION AND WALKING PROGRAM

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ABSTRACT

Seven women with childhood-onset obesity (CO) and 8 with adult-onset obesity (AO) participated for 9 months in a nutrition education and exercise program which promoted a low-fat eating style and regular walking. Nutrient intake, aerobic fitness (VO2 max), blood lipids, blood pressure, glucose tolerance, and weight were assessed before and after the program. Dietary fat intake decreased from 46 $\pm$ 4 to 29 $\pm$ 2% of kcals for CO and from 42 $\pm$ 6 to 36 $\pm$ 6% of kcals for AO. VO2 max increased 8% for CO and 7% for AO ($p<.05$). Total plasma cholesterol (TC) decreased for CO from 5.25 $\pm$ 0.83 to 4.63 $\pm$ 0.85 mmol/L and for AO from 4.55 $\pm$ 0.75 to 4.37 $\pm$ 0.78 mmol/L ($p<.01$). LDL-C decreased similarly with no significant change in HDL-C or triglycerides. Resting diastolic blood pressure (DBP) decreased 4% and 5% for CO and AO, respectively ($p<.05$). During exercise at 50% of VO2 max, SBP decreased 3-4% ($p>.05$) while DBP decreased 9-11% ($p<.001$). Area under the glucose tolerance curve decreased 43% for CO and 21% for AO ($p=.07$). Body weight decreased 5.7 kg in CO and 3.3 kg in AO ($p<.05$). Change in TC was most highly correlated with change in % kcals from fat ($r=0.68$, $p<.01$) while change in resting SBP and DBP was most highly correlated with change in VO2 max ($r=0.64$, $p<.01$). Change in body weight was not significantly related to change in any of the risk factor variables. These results suggest that changes in dietary and activity habits will improve health risk factors in women with obesity without the need for drastic weight loss.

KEY WORDS: blood lipids, blood pressure, glucose tolerance, nutrition education, walking, weight loss

INTRODUCTION

Decreasing dietary fat, increasing aerobic exercise and attaining ideal body weight are among the current recommendations for preventing and treating elevated blood lipids, hypertension and glucose intolerance, all risk factors for coronary heart disease (Berger & Berchtold, 1982; American Heart Association, 1984; Gotto et al, 1984; National Cholesterol Education Program Expert Panel, 1988). However, there is considerable disagreement as to what ideal body weight should actually be (Knapp, 1983; Burton et al, 1985; Manson et al, 1987). Because the strength of the association between obesity and occurence of these health risk factors and coronary heart disease (CHD) is less in women than men (Montoye et al, 1966; Lewis et al, 1974; Keys, 1980; Taylor et al, 1981; Krotkiewski et al, 1983) and because the social pressure on women to be thin goes well beyond health issues (Wooley & Wooley, 1984; Wadden & Stunkard, 1985), some suggest that obesity in women should not be treated at all (Wooley & Wooley, 1984). An alternative approach is that ideal body weight should be individually determined and based on the absence of important risk factors and on family weight history (Callaway, 1984; Van Itallie, 1985).

A moderate loss of body weight, on the order of 10-20 kg, normalizes blood pressure, blood lipids and glucose tolerance in people with obesity without the need to attain a so-called "ideal" body weight (Olefsky et al, 1974; Stamler et al, 1980; Eliahou et al, 1981). Decreases in dietary fat and/or increases in physical activity without accompanying weight loss are also associated with improvements in these

risk factors in people without obesity (Hegsted et al, 1965; Boyer & Kasch, 1970; Rosenthal et al, 1983; Blair et al, 1984; Bogardus et al, 1984; Tran & Weltman, 1985; Grundy et al, 1986) and in those with obesity (Bjorntorp et al, 1973; Krotkiewski et al, 1979; Caggiula et al, 1981).

In addition to improving health risk factors, a "side effect" of habitually consuming a lower-fat diet and increasing moderate-intensity physical activity could be a reduction in body weight since calorie intake spontaneously decreases when consuming a low-fat diet (Duncan et al, 1983; Lissner et al, 1987), and exercise has a well-known effect of decreasing body fat (American College of Sports Medicine, 1983). Because weight loss per se, especially to within the recommendations of weight for height, is not easily achieved and seldom maintained (Stunkard & McLaren-Hume, 1959; Wing & Jeffery, 1979; Berchtold & Van Itallie, 1985), and because "ideal" body weight does not need to be achieved for normalization of health risk factors, we were interested in offering a health improvement program to women with moderate obeity that focused on learning to habitually consume a low-fat diet and to increase moderate-intensity physical activity, with no preconceived notion of how much or whether weight would be lost. Though many weight control programs have been tested in women with obesity and some have included both diet and exercise, no programs have been done which specifically emphasized decreasing dietary fat and increasing physical activity and then observed the effect on blood lipids, blood pressure, glucose tolerance, and body weight.

Coronary heart disease risk factors tend to be associated more with weight gain during adulthood than with obesity beginning in childhood (Abraham et al, 1971; Albrink et al, 1962; Bjorntorp & Sjostrom, 1971) probably due to the increase in fat cell size associated with adult-onset of obesity (Salans et al, 1968). Reduction in weight is reportedly easier in those with adult-onset of obesity whether by diet (Bjorntorp et al, 1975; Krotkiewski et al, 1977) or exercise (Krotkiewski et al, 1979). Because we were offering a program with a focus toward improving health risk factors rather than losing weight, we were interested in comparing this type of approach in women with childhood vs adult-onset obesity.

Our purpose, therefore, was threefold: 1) to offer to women with moderate obesity a nutrition education program emphasizing reduction of dietary fat along with a moderate exercise program consisting of walking, 2) to evaluate changes in blood lipids, blood pressure, glucose tolerance, and body weight as a result of this program, and 3) to compare these changes in women with childhood vs adult-onset obesity.

METHODS

Subjects

Fifteen moderately obese women participated in this study. All were premenopausal, non-pregnant, free from chronic diseases, non-smokers, taking no medications or vitamin/mineral supplements, 25-40 years of age, 30-100% above the 1983 Metropolitan weight standard for their height, weight stable for at least 2 months prior to participation, and sedentary. Seven of the women had obesity whose onset occurred prior to adolescence (8 $\pm$ 2 years) and comprised the childhood-onset (CO) group. Eight had obesity occurring after attainment of adult stature (22 $\pm$ 3 years) and comprised the adult-onset (AO) group. Details of the selection process and participation in the program have been published previously (ref program results paper). The study was approved by the Human Subjects Committee at OSU and all the women gave their informed consent.

Program

All 15 women participated in a 9-month nutrition education and walking program. Nutrition education classes were held weekly during the first 4-1/2 months, then twice monthly for the second 4-1/2 months. The emphasis in the classes was on learning to make permanent dietary changes, specifically reducing fat intake and increasing complex carbohydrate intake while maintaining an adequate intake of other nutrients. In addition to nutrition information, issues related to behavior modification, self-esteem, and lifestyle change were included. The women also participated in supervised walking classes three times a

week throughout the program. The classes met for one hour and included 10 minute warm-up and cool-down activities and 40 minutes of walking. Intensity of walking was 50% of each person's measured VO2 max and was monitored by checking heart rate. More specific details of the 9-month program have been published elsewhere (ref program results paper).

Data Collection

Prior to and following the 9-month program a 3-week testing period was conducted during which the data pertaining to health risks factors was obtained. Throughout each 3-week test period the women were fed a weight-maintaining controlled diet prepared in the metabolic kitchen in the Foods and Nutrition Department. The diet was the same each day, consisted of commercially available foods, and provided 26% of kcals as fat, 60% as carbohydrate, and 14% as protein (ref kcals paper). Blood was obtained from subjects for lipid analysis prior to each 3-week controlled diet period. The glucose tolerance tests were done after 3 days of consuming the controlled diet. Measurements of body composition, aerobic fitness, and blood pressure were made in the Human Performance Lab in the College of Health and Physical Education during the 2nd and 3rd weeks of the testing periods.

Body weight was monitored daily during both pre and post testing periods and weekly during the 9-month program. Body density was measured by hydrostatic weighing (McArdle et al, 1981) and percent body fat (%BF) calculated using the Siri formula (Siri, 1961). Residual lung volume was estimated at 28% of the largest of 3 repeated trials of forced vital capacity (McArdle et al, 1981; Astrand & Rodahl, 1977).

Fat-free mass (FFM) was calculated by subtracting body fat mass from total body mass. Body mass index (BMI) was calculated using W/H2.

Maximal aerobic capacity (VO2 max) was measured on a calibrated Quinton motorized treadmill using a modified Balke procedure (Naughton & Haider, 1973). Heart rate was monitored continuously during this test. Oxygen consumption was measured via open circuit spirometry. Because VO2 max expressed as ml O2·kg body weight^{-1}.min^{-1} can increase with no fitness change by simply decreasing body weight, we have expressed values for VO2 max in this paper per kg FFM, as FFM did not change from pre to post in this group of women.

Prior to each 3-week testing period and while the women were consuming freely chosen diets, fasting blood was drawn for lipid analysis. In the morning following a 12-hour fast venous blood was drawn into tubes containing EDTA. Plasma was separated from red cells and sent to the Lipid Research Laboratory at the Oregon Health Sciences University for analysis. Total cholesterol (TC) and triglycerides were determined fluorometrically, HDL cholesterol (HDL-C) by heparin-manganese precipitation method, VLDL cholesterol by indirect estimation, and LDL cholesterol (LDL-C) by difference as described by Connor et al (1982).

Systolic (SBP) and diastolic (DBP) blood pressure were measured early in the morning at rest and during a 4-stage bicycle ergometer test conducted in the afternoon. All measurements were taken while in the seated position using an oversized cuff. Korotkoff phases 1 and 4

were used for SBP and DBP, respectively. The resting measurements are the mean of 4 measurements taken during the 3rd week of each testing period. The women came into the lab at 6:30am. Blood pressure measurements were taken after at least 5 minutes of seated rest. The submaximal bicycle ergometer test consisted of the following 4 stages, each 5 minutes in length: seated rest, 0 watts, 50 watts, and 100 watts, at a 60 rpm pedalling frequency. Blood pressure was measured during the 3rd minute of each stage.

The three hour glucose tolerance test (GTT) was conducted the first week of each testing period after 3 days on the high-carbohydrate metabolic diet. The women were organized to begin the 3-week testing periods generally between days 3 and 7 of their menstrual cycle. Consequently with only 3 exceptions at baseline testing and 1 at the end-point testing, the GTT was conducted during the first half of their menstrual cycles (Macdonald & Crossley, 1970). On the morning of the test a fasting blood sample was drawn after which the women consumed a 75g glucose load (National Diabetes Data Group, 1979). Subsequent blood draws were made at 1/2, 1, 2, and 3 hours. Plasma was separated and stored at -20°C until analysis. Each person's pre and post plasma samples were analyzed in the same assay to eliminate interassay variation within individuals. Glucose was determined by the glucose oxidase method (Pileggi & Szustkrewicz, 1974). Interassay coefficient of variation was 3%. Area under the GTT curve was calculated by integration of the glucose response above the level of fasting glucose.

Assessment of calorie, fat, and cholesterol intake was made from

3-day food records (ref program results paper). Prior to each 3-week testing period the women recorded food intake for 3 consecutive days. The records were coded and computer analyzed for nutrient content using the 1984 edition of the Ohio State Nutrient Data Base (Schaum et al, 1973). Oleic and linoleic acids were used as a measure of mono- and polyunsaturated fatty acids, respectively.

Statistical Analysis

A repeated measures ANOVA procedure using the BMDP Statistical Package (BMDP/PC, 1987) was used to analyze the pre and post data by the age of onset groupings. In this procedure, a significant interaction (INT) indicates a different pattern of change from pre to post for the two groups being compared (CO vs AO). A significant time effect (T) indicates a change from pre to post that is similar in the two groups, a significant group effect (G) denotes no significant change over time but a significant difference in the group means. For analyzing the data involving the entire group of 15 women, paired t-tests and Pearson product-moment correlations were used. Significance of probability values was based on two-tailed tests and level of significance set at $p<.05$.

RESULTS

Data on changes in body weight and body composition over the 9 months are presented in Table 1. Body weight, MRW, BMI, and % BF all decreased significantly from pre to post for both CO and AO. There was no change in fat-free weight (FFW) during the program.

Dietary intake of calories, fat, and cholesterol is presented in Table 2. Both groups significantly decreased calorie intake from pre to post. However, the CO group made significantly greater pre to post changes in carbohydrate, total fat, saturated fat, and cholesterol intake than the AO group.

Blood lipid values before and after the program are listed in Table 3. Both the CO and AO groups had significantly lower total cholesterol and LDL-C at the end of the program. Pre to post changes in HDL-C and triglycerides were not significant for either group.

Maximal aerobic capacity increased significantly by 8% for CO and by 7% for AO from pre to post. VO2 max expressed in ml O2.kg FFW-1.min-1 increased from 48.5 $\pm$ 4.9 to 52.4 $\pm$ 5.1 for CO and from 47.4 $\pm$ 3.1 to 50.7 $\pm$ 6.1 for AO.

Values for SBP and DBP at rest and during the 4-stage exercise test are presented in Table 4. There was no significant difference in the CO and AO groups for any of the blood pressure measurements. Generally, greater pre to post decreases were seen in DBP than SBP for

both groups. This was especially true at the higher levels of exercise intensity where percent decreases in DBP were 2-3 times greater than in SBP.

Figure 1 shows the glucose tolerance curves for the CO and AO groups. Both groups had significantly lower plasma glucose levels at the end of the program for all time points ($p<.05$) except 3 hours which was not significantly different. The mean sum of the area under the curve decreased 43% for CO (from 1.79 $\pm$ 1.44 to 1.02 $\pm$ 1.35 mmol/L.hr) and 21% for AO (from 2.79 $\pm$ 1.94 to 2.21 $\pm$ 1.42 mmol/L.hr). However, because of the wide variation among individuals, the decrease in the area under the curve was not statistically significant ($p=.07$).

Correlation analysis was done on changes in diet, aerobic fitness, and weight, and changes in blood lipids, blood pressure, and glucose tolerance. Changes in blood lipids were most highly correlated with change in dietary fat intake. Significant correlations were found between change in % kcal as fat and change in total plasma cholesterol ($r=0.68$, $p<.01$) and LDL-C ($r=0.59$, $p<.01$). Changes in dietary cholesterol ($r=0.53$, $p<.05$) and body weight ($r=0.56$, $p<.05$) were also significantly correlated with change in LDL-C. Pre to post blood pressure at rest was highly correlated with the change in aerobic fitness. The greater the increase in fitness the greater the decrease in both resting SBP ($r=-0.68$, $p<.01$) and DBP ($r=-0.64$, $p<.01$). Surprisingly, that association did not carry over to blood pressure measured during the exercise test. No significant correlations were found between change in aerobic fitness and change in blood pressure

during any of the stages of the exercise test. Only two significant correlations were seen with the glucose tolerance data. Change in fasting plasma glucose concentration was significantly correlated with change in % kcal as fat ($r=0.57$, $p<.05$) and with change in body weight ($r=0.63$, $p<.01$). No significant correlations were found among any of the other glucose values at the various time points in the GTT or for change in the sum of the plasma glucose concentrations during the GTT.

DISCUSSION

This program for moderately obese women was unique in that its primary focus was on decreasing intake of dietary fat and increasing moderate intensity physical activity. The consequence of such a program is, of course, that body fat could be lost due to a change in calorie balance. However, of greater significance is the possibility that health risk factors such as blood lipids, blood pressure, and glucose tolerance might be improved whether or not a recommended weight for height was achieved.

Plasma cholesterol levels were significantly reduced in this study. Other studies have found weight reduction lowers cholesterol levels but that the effect may be temporary. Plasma cholesterol levels decrease drastically within one month with very-low-calorie weight reduction diets (Hill et al, 1987) but steadily increase, returning to baseline levels after 8-9 months once weight stabilizes at the reduced levels (Sorbris et al, 1981; Ellis et al, 1987; Friedman et al, 1982). Even with a more moderate approach to weight reduction involving behavior modification of eating habits, Thompson et al (1979) found significantly lowered plasma cholesterol levels at 10 weeks which returned to baseline levels at 8 months. This return to baseline occurred even though weight continued to decrease from 10 weeks to 8 months. Exercise alone has a cholesterol-lowering effect, although the effect is enhanced by weight reduction (Tran & Weltman, 1985). In other exercise studies in obese men and women with or without moderate calorie reduction, lowered plasma cholesterol levels have not been seen

even with mean body weight reductions up to -5.7 kg (Lewis et al, 1976; Franklin et al, 1979; Kukkonen et al, 1982; Bjorntorp et al 1973; Krotkiewski et al, 1979; Leon et al, 1979).

The reason for the significant decrease in cholesterol in this study may be due to the specific reduction in dietary fat rather than just calories in this group. The highly significant correlation between change in % kcal as fat and change in total plasma cholesterol supports that conclusion. The cholesterol-lowering effect of decreasing dietary fat is well-known. We calculated the predicted change in plasma cholesterol using the Keys and Hegsted equations (Keys et al, 1965; Hegsted et al, 1965). For CO the predictions were -0.47 mmol/L and -0.70 mmol/L for Keys and Hegsted, respectively. Observed change was -0.62 mmol/L (-24 mg/dL). For AO the predictions were -0.16 mmol/L and -0.23 mmol/L for Keys and Hegsted, respectively. Observed change for AO was -0.18 mmol/L (-7 mg/dL). This would seem to demonstrate the importance of stressing long-term dietary fat reduction as part of an overall program which emphasizes improvement of health risk factors rather than just reduction of body weight.

Change in LDL-C in this group followed the same pattern as change in total cholesterol and is consistent with results of other weight loss studies with obese subjects where changes in total cholesterol are reflected in LDL-C (Lewis et al, 1976; Leon et al, 1979; Thompson et al, 1979). The HDL-C levels in our group did not change significantly from pre to post. Since lower HDL-C levels are associated with obesity and sedentary lifestyles (Miller & Miller, 1975), it might be expected

that HDL-C levels would increase as a result of a program such as this. However, the results in the present study are similar to those found by Lewis et al (1976) and Thompson et al (1979) in their groups of obese women. In contrast, Leon et al (1979) found that a walking program with obese men resulted in significant increases in HDL-C. In weight loss programs using very-low-calorie diets, HDL-C levels in obese men and women have increased significantly and stayed elevated even into periods of weight maintainence (Sorbris et al, 1981; Friedman et al, 1982). It is possible that greater weight must be lost or exercise must be increased in intensity or duration beyond that used in this study to see significant increases in HDL-C. However, even though HDL-C did not increase, the ratios of total cholesterol and LDL-C to HDL-C either decreased slightly or stayed the same, demonstrating a trend in the direction of a lowered cardiovascular risk.

Of particular interest is the lack of significant change in plasma triglyceride levels in this group. Concern has been expressed about the possible increase in triglyceride levels accompanying the increase in dietary carbohydrate which results when dietary fat is reduced (Coulston et al, 1983). Of our 15 women, 6 had higher triglyceride levels at the end-point testing compared to baseline testing. However, at the end-point testing no one's level was greater than 1.62 mmol/L (144 mg/dl) and values for all but 4 people were less than 1.13 mmol/L (100 mg/dl). These values are all well within normal ranges.

Plasma triglyceride levels haven't generally been found to change significantly with exercise-related weight loss in obese subjects

(Lewis et al, 1979; Franklin et al, 1979; Bjorntorp et al, 1973; Krotkiewski et al, 1979; Leon et al, 1979; Thompson et al, 1979), although Kukkonen et al (1982) observed a significant decrease in triglycerides in the women they studied. Their exercise program was 17 months in duration, 11 months longer than any of the studies cited above. This may be a factor in seeing lowered triglyceride levels, although no significant change was seen for the men in their study. Weight loss achieved with very-low calorie diets also doesn't result in significantly lowered triglycride levels (Hill et al, 1987;, Ellis et al, 1987; Sorbris et al, 1981; Friedman et al, 1982). Triglyceride levels may decrease only if they are high at the onset of weight loss. Olefsky et al (1974) observed large decreases in triglyceride levels with moderate weight loss in hyperlipidemic patients and found the greatest reductions occurred in those with the highest initial values. In neither the present study nor any of the studies cited above were mean triglyceride values initially greater than 1.6 mmol/L (140 mg/dL).

The initial blood pressure level may also play a role in how much of a reduction is achieved in that risk factor by weight loss and/or exercise. There was only a slight reduction in resting blood pressure over the 9 months of this study. Early morning resting SBP at baseline ranged from 104-129 mmHg and DBP from 64-91 mmHg. Only one person in this group at baseline might have been considered mildly hypertensive with a DBP of 91 mmHg, though her SBP was well within the normal range at 129 mmHg. In people with both obesity and hypertension, weight reduction is associated with a reduction in blood pressures (Eliahou et al, 1981). We could find no documentation that weight reduction has a

lowering effect on blood pressure in women with moderate obesity who are already normotensive. Exercise training also tends to improve blood pressure in those with hypertension but not those with normal blood pressure, although results are conflicting (Horton, 1981).

Some exercise studies with obese women have demonstrated small but significant decreases in resting SBP and DBP (Franklin et al, 1979; Krotkiewski et al, 1979) whereas others have seen no change (Lewis et al, 1976; Kukkonen et al, 1982). In these studies, initial mean blood pressures were within normal ranges and still significant changes in resting blood pressure were seen in some groups and not others. Weight change accompanying exercise in these obese women does not seem to be a factor. Krotkiewski et al (1979) saw significant blood pressure reduction and no weight loss in his group of obese women whereas Lewis et al (1976) observed a mean 4.2 kg weight loss and no significant change in blood pressure. In the present study the correlation between change in body weight and change in resting blood pressure was very low and not significant, while a highly significant correlation was seen between change in aerobic fitness and change in both SBP and DBP. The interplay of exercise, weight change, and resting blood pressure in obese women obviously warrants further study.

Though Lewis et al (1976) did not observe a change in resting blood pressure, they did see a significant decrease in SBP and DBP during submaximal exercise. Franklin et al (1979) also observed significant decreases in exercise SBP and DBP. Their results are similar to those observed during exercise in the present study where

both SBP and DBP decreased significantly pre to post.

It is interesting that in the study by Krotkieski et al (1979) blood pressure change was not related to weight change but was significantly related to change in plasma insulin values. They proposed some possible mechanisms by which high blood pressure in obesity might be related to hyperinsulinemia including the effect of insulin on sodium reabsorption by the kidney. A lowering of insulin then would effect a lowering of resting blood pressure. Other studies have demonstrated the insulin-lowering effect of exercise in both lean (Rosenthal et al, 1983; Bogardus et al, 1984) and obese subjects (Bjorntorp et al, 1970) without the need for accompanying weight loss. We cannot report directly on insulin levels in this study but can speculate indirectly that insulin levels decreased because there was a significant pre to post decrease in plasma glucose response during the GTT. It is possible in our group of women that the exercise component of our program had a beneficial effect on both resting blood pressure and glucose tolerance through the effect exercise has on increasing insulin sensitivity and decreasing plasma insulin levels. Though the change in glucose response to the GTT was not correlated significantly with the change in fitness, the possibility remains that fitness change was related to change in insulin response to the GTT as has been found by others.

Weight reduction is well known as a therapy for improving glucose tolerance in people with obesity whether or not they meet the criteria for impaired glucose tolerance and non-insulin dependent diabetes

mellitus (Golay et al, 1985). We did see a significant correlation between change in fasting glucose and change in body weight, although change in body weight was not correlated with change in glucose at any other time point during the GTT or with change in the area under the curve. Within the group, individual variation in plasma glucose response to the GTT was very great. Thus, a lack of correlation with body weight change is not particularly surprising. Of the 15 women, 10 had reduced areas under the GT curve from pre to post. Of those 10, 5 had lost more than 4.5 kg of body weight, the remaining 5 had weight changes ranging from −3.6 to +5.0 kg. Four of the 15 women had slight increases in area under the GT curve, their weight change ranged from −10.4 to +8.6 kg. One person's area under the GT curve increased substantially (+88%) and her weight changed − 2.3 kg from pre to post.

We can say with certainty that glucose tolerance improved in this group of women following participation in a program emphasizing increased physical activity and decreased intake of dietary fat. This occurred even with initial GTT values for all individuals falling within the normal range. What we cannot say with certainty is whether one component of the program was more important than another for eliciting this response.

In none of the risk factors studied did we see any significant differences between the CO and AO groups. We cannot say for certain that these two groups had hyperplastic vs hypertrophic obesity as others have demonstrated (Brook et al, 1972; Salans et al, 1973; Sjostrom & Bjorntorp, 1974). However, we found no differences in blood

lipids, blood pressure or glucose tolerance, or in the ability to improve these parameters through changes in dietary fat and exercise in these women differing by a mean of 14 years in the age of onset of their obesity.

In summary, results from this study demonstrate that an habitual decrease in dietary fat and an increase in physical activity improve blood lipids, blood pressure, and glucose tolerance in both childhood and adult-onset obese women with only a small mean decrease in body weight. These results suggest that drastic weight loss for the purpose of attaining a "normal" or "ideal" weight for height is not necessary for achieving normalacy in terms of CHD risk factors for women. At least in terms of improving blood lipid values, an approach emphasizing decreased dietary fat and aerobic exercise may be more effective than conventional weight loss methods that stress calorie reduction with or without exercise.

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Table VI.1. Weight and other obesity indices before and after the program (pre and post) grouped by differences in age of onset of obesity. Values are mean ± standard deviation.

	Childhood Onset			Adult Onset			Statistical
	Pre	Post	% Change	Pre	Post	% Change	Significance*
Body weight (kg)	98.4 ± 11.7	92.7 ± 7.4	-5.7%	93.2 ± 13.6	89.9 ± 15.4	-3.5%	T, p<.05
MRW (1983)	155 ± 19	147 ± 13	-5.2%	162 ± 21	156 ± 26	-3.7%	T, p<.05
BMI (W/H2)	34.2 ± 4.1	32.2 ± 3.2	-5.8%	36.6 ± 4.7	35.4 ± 5.7	-3.3%	T, p<.05
Body fat (%)	46 ± 4	43 ± 4	-6.5%	45 ± 4	44 ± 5	-3.3%	T, p<.05
FFW (kg)	52.9 ± 2.7	53.0 ± 1.9	+0.1%	50.8 ± 5.8	50.3 ± 6.0	-1.0%	NS

* T indicates a significant pre to post change for both groups.

Table VI.2. Dietary intake of calories, fat, and cholesterol before and after the program (pre and post) grouped by differences in age of onset of obesity. Values are mean ± standard deviation.

	Childhood Onset			Adult Onset			Statistical
	Pre	Post	% Change	Pre	Post	% Change	Significance*
Total Kcals	2557 ± 572	1580 ± 354	-38%	1867 ± 550	1689 ± 261	-10%	T, $p<.01$
% Kcals as:							
Carbohydrate	38 ± 5	55 ± 5	+45%	42 ± 6	46 ± 8	+10%	INT, $p<.01$
Protein	13 ± 2	15 ± 2	+15%	15 ± 2	16 ± 4	+ 7%	NS
Fat	46 ± 4	29 ± 2	-37%	42 ± 6	36 ± 6	-14%	INT, $p<.01$
Saturated Fat	15 ± 4	9 ± 3	-40%	15 ± 3	12 ± 2	-20%	INT, $p<.01$
Monounsaturated Fat	15 ± 3	10 ± 6	-33%	14 ± 3	11 ± 3	-21%	T, $p<.01$
Polyunsaturated Fat	7 ± 3	6 ± 1	-14%	6 ± 2	5 ± 2	-17%	NS
Cholesterol (mg)	387 ± 121	158 ± 87	-59%	305 ± 154	233 ± 84	-24%	INT, $p<.05$

* T indicates a significant pre to post change for both groups, INT indicates a change from pre to post that differs significantly between the two groups.

Table VI.3. Plasma cholesterol and triglyceride concentrations before and after the program (pre and post) grouped by differences in age of onset of obesity. Values are mean ± standard deviation.

	Childhood Onset			Adult Onset			Statistical
	Pre	Post	% Change	Pre	Post	% Change	Significance*
Total Cholesterol (mmol/L)	5.25 ± 0.83	4.63 ± 0.85	-12%	4.55 ± 0.75	4.37 ± 0.78	- 4%	T, p<.01
LDL-C (mmol/L)	3.52 ± 0.59	3.03 ± 0.70	-14%	3.00 ± 0.67	2.74 ± 0.75	- 9%	T, p<.01
HDL-C (mmol/L)	1.32 ± 0.28	1.19 ± 0.18	- 6%	1.24 ± 0.13	1.24 ± 0.10	NC	NS
Triglyceride (mmol/L)	0.95 ± 0.33	0.94 ± 0.38	- 1%	0.69 ± 0.20	0.88 ± 0.38	+28%	NS
Chol/HDL-C	4.1 ± 0.6	4.0 ± 0.6	- 2%	3.6 ± 0.4	3.6 ± 0.6	NC	NS
LDL-C/HDL-C	2.7 ± 0.5	2.6 ± 0.5	- 4%	2.4 ± 0.5	2.2 ± 0.6	- 8%	NS

* T indicates a significant pre to post change for both groups.

Table VI.4. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) at rest and during exercise on a cycle ergometer before and after the program (pre and post) grouped by differences in age of onset of obesity. Values are mean ± standard deviation.

	Childhood Onset			Adult Onset			Statistical
	Pre	Post	% Change	Pre	Post	% Change	Significance*
	mmHg			mmHg			
Seated rest, early am							
SBP	115 ± 8	113 ± 4	- 2%	111 ± 10	110 ± 6	- 1%	NS
DBP	80 ± 7	77 ± 4	- 4%	76 ± 7	72 ± 5	- 5%	T, $p<.05$
Bicycle ergometer, seated rest, afternoon							
SBP	123 ± 8	115 ± 10	- 6%	121 ± 7	114 ± 8	- 6%	T, $p<.001$
DBP	83 ± 3	80 ± 6	- 4%	80 ± 6	78 ± 5	- 2%	T, $p<.01$
Bicycle ergometer, 0W workload							
SBP	134 ± 8	113 ± 12	-14%	129 ± 9	119 ± 8	- 8%	T, $p<.001$
DBP	87 ± 7	74 ± 8	-15%	81 ± 6	71 ± 6	-12%	T, $p<.001$
Bicycle ergometer, 50W workload							
SBP	137 ± 8	131 ± 19	- 4%	138 ± 15	134 ± 14	- 3%	NS
DBP	85 ± 6	76 ± 7	-11%	81 ± 6	74 ± 5	- 9%	T, $p<.001$
Bicycle ergometer, 100W workload							
SBP	151 ± 10	147 ± 21	- 3%	156 ± 13	146 ± 15	- 6%	T, $p<.05$
DBP	84 ± 8	76 ± 11	-10%	80 ± 8	72 ± 7	-10%	T, $p<.001$

* T indicates a significant pre to post change for both groups.

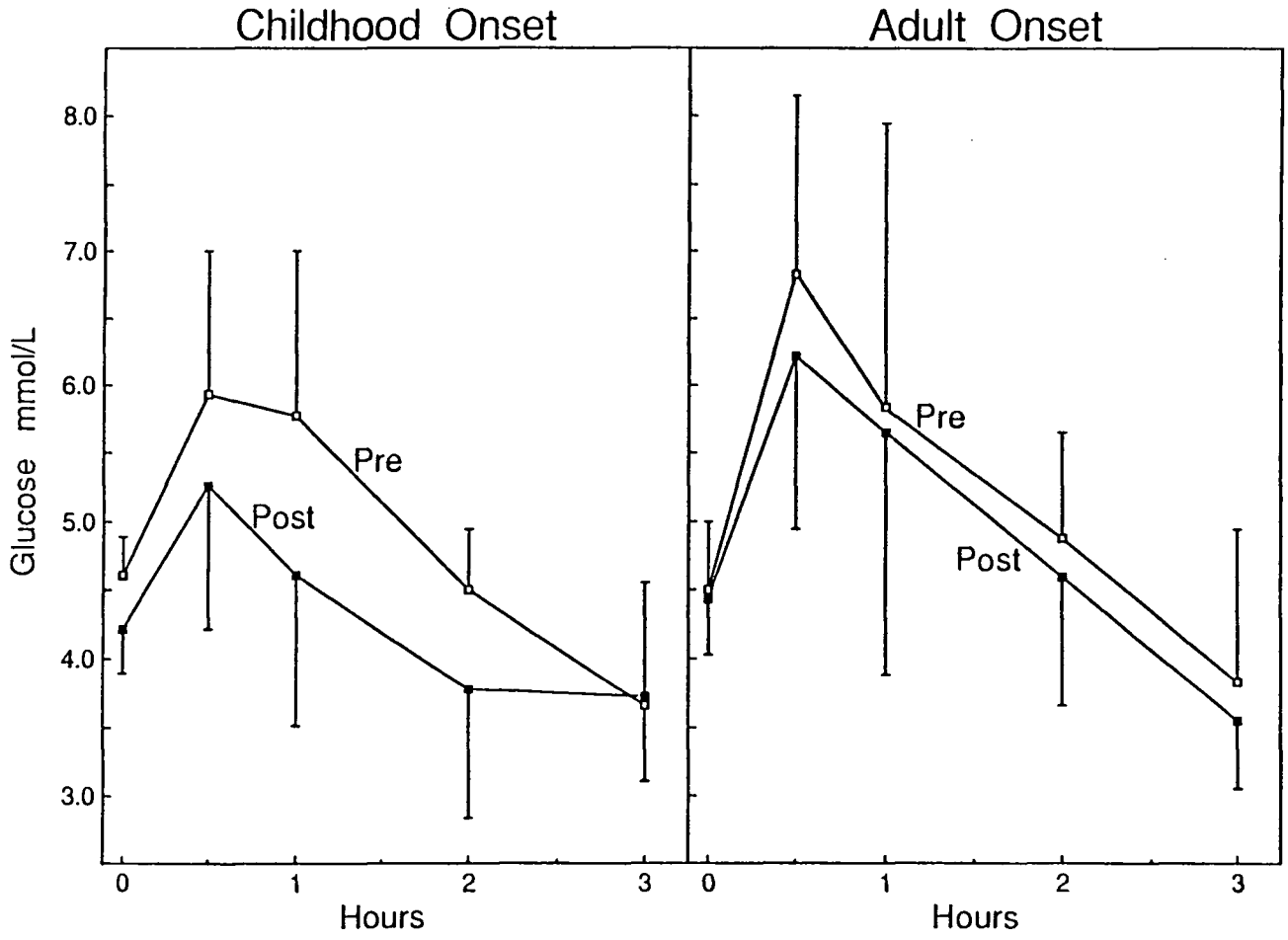

Figure VI.1. Plasma glucose levels during the GTT before and after the program (pre and post) for the CO and AO groups. A significant pre to post decrease (p .05) occurred for both groups at time points 0, 1/2, 1, 2 hours. The pre to post change at 3 hours was not significant.

CHAPTER VII

SUMMARY AND CONCLUSIONS

Summary

The effect of a 9-month nutrition education and walking program on energy need, body composition, nutrient intake, nutritional status, aerobic fitness and health risk factors was determined in 7 women with childhood-onset obesity (CO) and 8 women with adult-onset obesity (AO). A 3-week testing period was conducted before and after the 9-month program during which all subjects were on a controlled, weight-maintaining metabolic diet which was used to determine total calorie need and to provide dietary control during assessment of resting metabolic rate (RMR), fitness, blood pressure, and glucose tolerance. Three-day food records were obtained prior to each testing period as was fasting blood for analysis of plasma cholesterol and triglycerides.

During the 9-month program the women attended a nutrition education class once each week. The focus of the class was on learning to make permanent changes in dietary intake particularly by decreasing intake of dietary fat. Other topics in the classes included eating awareness; behavior modification; nutrient content of foods with a special emphasis not only on fat but also on carbohydrate, iron,

vitamin C, and vitamin B6; eating differently around family and friends; eating out; trying new foods; enhancement of self esteem; and the benefits of regular, moderate physical activity. In the exercise portion of the program the women participated in supervised walking classes three times each week. Walking was done at an intensity approximating 50% of each person's VO2 max.

Need for total calories increased by 2.9% for CO and 3.1% for AO from pre to post, a non-significant change ($p=0.9$), whereas calories for RMR decreased significantly ($p<.05$) by 3.6% for CO and 2.8% for AO. Body weight decreased significantly ($p<.05$) for CO from 98 $\pm$ 12 to 93 $\pm$ 7 kg (-5.7 $\pm$ 9.3 kg) and from 93 $\pm$ 14 to 90 $\pm$ 15 kg in AO (-3.3 $\pm$ 6.2 kg). Of the weight that was lost, 93% was from fat tissue with fat-free tissue not changing significantly over the 9 months. Time spent in light and moderate level activity increased significantly from pre to post ($p<.001$). The increase in activity more than compensated for the slight decrease in RMR, contributing to an overall increase in daily energy need at the end of the program, even for those women losing substantial body weight.

Over the 9 months, consumption of fat expressed as percent of kcal decreased from 46 $\pm$ 4 to 29 $\pm$ 2% for CO and from 42 $\pm$ 6 to 36 $\pm$ 6% for AO. A significant interaction ($p<.01$) between the two groups occurred for this variable with the CO group having a greater fat intake at the beginning and a lower intake at the end of the program compared to AO. Though total calorie need did not decrease pre to post, calorie intake decreased from 2557 $\pm$ 572 to 1580 $\pm$ 354 kcal for CO and from 1867 $\pm$ 550

to 1689 ± 261 for AO. The interaction for this variable was not statistically significant ($p=.07$) but the pre to post decrease was significant for both groups ($p<.01$).

Despite significant decreases in calories, both groups maintained or increased their intakes of iron and vitamin B6, two nutrients women generally consume in amounts less than the RDA. Iron status assessed by hematocrit, hemoglobin and % saturation of transferrin did not change significantly from pre to post nor did status of vitamin B6 assessed by plasma pyridoxal 5'-phosphate. Dietary change was related to age of onset of obesity but weight change was not. Weight change was, however, significantly correlated with body fat distribution. Those women with predominately upper body fat lost significantly more weight than those with predominately lower body fat. Fitness improved significantly in both groups with maximal aerobic capacity (VO2 max) increasing by 8% for CO and 7% for AO.

Total plasma cholesterol (TC) changed significantly ($p<.01$); for CO decreasing from 5.25 ± 0.83 to 4.63 ± 0.85 mmol/L and for AO from 4.55 ± 0.75 to 4.37 ± 0.78 mmol/L. LDL-C decreased similarly while no significant change occurred in HDL-C or triglycerides. Resting systolic blood pressure (SBP) did not change significantly, while resting diastolic blood pressure (DBP) decreased 4% and 5% for CO and AO, respectively ($p<.05$). During exercise at an intensity close to 50% of VO2 max, SBP decreased 3-4% ($p>.05$) while DBP decreased 9-11% ($p<.001$). Area under the glucose tolerance curve decreased 43% for CO and 21% for AO ($p=.07$). Change in TC was most highly correlated with

change in % kcals from fat ($r=0.68$, $p<.01$) while change in resting SBP and DBP was most highly correlated with change in VO2 max ($r=0.64$, $p<.01$). Change in body weight was not significantly related to change in any of the risk factor variables. These results suggest that changes in dietary and activity habits will improve health risk factors in women with obesity without the need for drastic weight loss.

Conclusions

Two overall conclusions can be made as a result of doing this research project. First, this is a very viable approach for promoting health improvement in women with moderate obesity no matter at what age their obesity began. Second, 9 months is not a long enough period of time to reach a point of stable reduced weight using this approach.

The viability of this approach is demonstrated by the fact that this group of women did reduce their intake of dietary fat to a level very close to the recommended 30% of kcals as fat, and they did increase their aerobic fitness with a walking program of moderate intensity. As a result of these changes, significant improvements occurred in blood lipids, blood pressure and glucose tolerance. These improvements occurred in spite of the fact that only two women had initial levels of these risk factors above the range of low risk, and at that, they were only mildly elevated. In addition, for those women who lost substantial amounts of body weight (5 to 18 kg), calorie need was not compromised nor was nutritional status.

Noting that the health risk factor improvements occurred before the process of weight reduction was complete is important in this study. None of the women had yet reached a reduced body weight that had begun to level out by the time they were requested in September to begin maintaining weight in preparation for end point testing. From the results obtained in this study, we do not know when such a point might be reached. None of these women reached a relative weight less than 130% or a BMI less than 28. At what percent above a relative body weight they might remain once a stable reduced body weight is achieved cannot be deduced from our data. The question of what body weight might be "best" for women with obesity remains to be answered.

A further question that begs to be answered is "How are they doing now?" The benefits of this approach, or any approach to health improvement or weight control for that matter, depends on maintaining over the long term the changes made in diet and physical activity. Whether these women have continued walking and consuming a low-fat diet is unknown.

Suggestions for future research in this area are: 1) an intervention period of longer duration to document the length of time necessary to achieve a reduced, stable weight and what that weight is, using a health improvement model, and 2) incorporation of a follow up assessment to evaluate whether the changes made with such a health improvement actually continue over the long term.

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APPENDICES

APPENDIX I. Tables of raw data.

Recruitment of subjects. Numbers of people participating in recruitment process.

Callers		216
Rejected		142
Reasons for rejection:		
Weight too low/high	37	
On medication	20	
Distance too far to travel	19	
Too old/young	17	
Postmenopausal/hysterectomy	17	
Too late	15	
Too much time/involvement	12	
Too physically active	2	
Weight not stable	1	
Chronic disease	1	
Smoker	1	
People sent questionnaires		74
Questionnaires returned		41
Subject pool:		
Childhood-onset	18	
Adult-onset	23	

Compliance. Percent participation in nutrition education classes and walking program.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
% attendance in nutrition education classes (20 meetings = 100%)	100	96	70	85	95	70	100	70	80	95	85	95	85	85	90
% participation in walking classes (120 minutes/week = 100%)	100	106	130	145	114	107	150	99	102	92	105	75	109	114	95
Combined compliance (exercise not more than 100%)	100	95	85	92	98	85	100	84	90	94	92	85	92	92	92

Chemistry Screen. Values from Good Samaritan Hospital biochemistry screen, done in December, 1985.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
Glucose (mg/dL), expected range: 62-128	96	94	90	96	109		105	112	90	111	89	93	104	98	93
Creatinine (mg/dL), expected range: 0.8-1.6	0.8	1.0	0.8	0.7	0.9		0.8	0.8	0.8	0.7	0.8	0.7	0.6	0.8	1.0
BUN (mg/dL), expected range: 5.7-26.8	15.5	17.0	14.8	8.2	11.8		10.8	22.7	12.7	11.8	10.8	19.2	9.6	13.1	11.1
Albumin (g/dL), expected range: 3.6-5.1	3.9	4.5	4.2	4.2	4.6		4.2	4.1	4.2	4.5	4.5	4.3	4.6	4.2	4.2
Cholesterol (mg/dL), expected range: 146-277	198	207	258	160	225		200	196	153	226	180	135	184	205	200
Triglycerides (mg/dL), expected range: 20-200, age dependent	100	122	64	70	90		140	84	78	100	60	92	46	60	60
rT3-Uptake, expected range: 34-44	38.6	35.2	37.3	34.1	36.4	36.4	35.8	35.9	38.2	34.6	38.0	34.1	35.2	35.0	36.7
T4 (mcg/dL), expected range: 5.0-12.0	6.9	10.8	7.4	10.2	8.7	9.4	8.5	9.2	7.8	9.3	7.4	8.2	8.3	8.2	9.2
Free T4 Index, expected range: 1.75-5.40	2.66	3.80	2.76	3.48	3.17	3.42	3.04	3.30	2.98	3.22	2.81	2.80	2.92	2.87	3.38

Metabolic Diet. Percent of calories as protein, fat, and carbohydrate, and weight change during the metabolic diet.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
% Kcal as Protein															
Pre	13	13	14	13	13	14	14	14	14	13	13	14	14	14	13
Post	13	13	13	13	13	13	13	14	14	13	13	13	14	14	13
% Kcal as Fat															
Pre	27	27	26	28	26	25	26	26	25	27	26	24	25	25	27
Post	27	27	27	27	26	26	27	24	24	26	27	26	24	24	27
% Kcal as Carbohydrate															
Pre	60	60	60	59	61	61	60	60	61	60	61	62	61	61	60
Post	60	60	60	60	61	61	60	62	62	61	60	61	62	62	60
Weight Change in Pounds (Days 10-20)															
Pre	-2.00	+0.25	NC	-0.25	+2.25	-2.25	-2.25	-1.50	NC	-0.25	-0.25	-2.00	-2.50	-2.25	-3.25
Post	-1.25	+2.75	+0.25	-0.50	-1.00	-1.25	-4.00	-0.75	-2.50	-2.00	-1.25	-1.25	NC	-2.25	-0.25

FOOD ANALYSIS FOR JANE MOORE

SUMMARY FOR THIS RUN:

RECORD NO.	CARDS READ	FOOD ITEMS PROCESSED	
1	24	24	METABOLIC DIET 2100
2	24	24	METABOLIC DIET 2550
3	24	24	METABOLIC DIET 2900

	NUTRIENT	DAY1		DAY2		DAY3		AVERAGE
1	WEIGHT (GMS)	1678.21		1814.87		1896.96		1796.68
2	WATER (GMS)	1161.15	*	1188.56	*	1219.08	*	1189.60
3	FOOD ENERGY (CALS)	2064.87		2481.24		2701.12		2415.74
4	PROTEIN (GMS)	75.24		84.34		91.88		83.82
5	FAT (GMS)	58.36		75.26		80.56		71.40
6	CARBO. TOTAL (GMS)	325.26		384.25		421.24		376.92
7	FIBER (GMS)	8.10	*	9.02	*	9.73	*	8.95
8	ASH (GMS)	15.38	*	17.69	*	19.42	*	17.50
9	CALCIUM (MGS)	1090.52		1233.41		1316.68		1213.54
10	PHOSPHOROUS (MGS)	1522.20		1745.79		1902.15		1723.38
11	IRON (MGS)	18.00	*	21.11	*	23.25	*	20.79
12	SODIUM (MGS)	2368.46	*	3020.07	*	3418.29	*	2935.61
13	POTASSIUM (MGS)	2958.06	*	3149.48	*	3314.39	*	3140.64
14	VIT.A (IU)	12381.05		13001.36		13041.36		12807.92
15	THIAMINE (MGS)	1.98		2.31		2.51		2.27
16	RIBOFLAVIN (MGS)	2.07		2.41		2.58		2.36
17	NIACIN (MGS)	21.89		25.61		27.67		25.06
18	ASCORB. ACID (MGS)	156.71	*	161.13	*	161.26	*	159.69
19	PANTO. ACID (MGS)	3.99	*	4.49	*	4.81	*	4.43
20	PYRID. B6 (MCG)	2042.68	*	2342.89	*	2418.65	*	2268.08
21	VITAM. B12 (MCG)	4.10	*	4.88	*	4.88	*	4.62
22	FOLIC ACID (MCG)	383.11	*	406.49	*	432.58	*	407.39
23	TOTAL TOCO. (MGS)	4.68	*	5.73	*	6.66	*	5.69
24	ALPHA TOCO. (MGS)	2.92	*	4.22	*	4.41	*	3.85
25	NON-AL. TOCO. (MGS)	2.63	*	3.34	*	4.07	*	3.35
26	BIOTIN (MCG)	12.65	*	15.17	*	15.64	*	14.49
27	CHOLINE (MGS)	109.54	*	130.26	*	130.25	*	123.35
28	MAGNESIUM (MGS)	446.21	*	547.91	*	640.99	*	545.04
29	COPPER (MGS)	1.08	*	1.27	*	1.48	*	1.28
30	CHLORINE (MGS)	139.38	*	151.59	*	151.59	*	147.52
31	SULFUR (MGS)	89.00	*	111.39	*	111.39	*	103.93
32	IODINE (MGS)	.00	*	.00	*	.00	*	.00
33	HISTIDINE (MGS)	1310.91	*	1355.47	*	1355.47	*	1340.62
34	ISOLEUCINE (MGS)	3334.06	*	3648.59	*	4008.21	*	3663.62
35	LEUCINE (MGS)	5405.26	*	5920.95	*	6484.42	*	5936.88
36	LYSINE (MGS)	4216.14	*	4455.71	*	4716.54	*	4462.80
37	TYROSINE (MGS)	1901.75	*	1993.98	*	1993.98	*	1963.24
38	PHENYL. (MGS)	2987.53	*	3326.04	*	3707.85	*	3340.47
39	THREONINE (MGS)	2426.51	*	2657.40	*	2902.34	*	2662.08
40	TRYPTOPHAN (MGS)	772.83	*	866.21	*	954.44	*	862.49
41	VALINE (MGS)	3608.17	*	3972.60	*	4347.26	*	3976.01
42	CYSTINE (MGS)	465.51	*	501.24	*	501.24	*	489.33
43	CYSTEINE (MGS)	.00	*	.00	*	.00	*	.00
44	METHIONINE (MGS)	1423.57	*	1538.52	*	1663.06	*	1541.72
45	SAT. FAT (GMS)	22.08		26.67		27.95		25.57
46	OLEIC (GMS)	18.15		23.44		25.91		22.50
47	LINO. ACID (GMS)	10.56		15.85		17.10		14.51
48	UNSAT. FAT (GMS)	9.26	*	14.26	*	14.26	*	12.59
49	CHOLESTEROL (MGS)	162.19		192.55		215.12		189.99
50	LACTOSE (GMS)	1.51	*	2.01	*	2.77	*	2.10
51	SUCROSE (GMS)	32.25	*	38.15	*	38.16	*	36.19
52	MALTOSE (GMS)	.00	*	.00	*	.00	*	.00
53	GLUCOSE (GMS)	17.47	*	18.05	*	18.05	*	17.86
54	FRUCTOSE (GMS)	.00	*	.00	*	.00	*	.00
55	VITAM. D (IU)	100.00	*	100.00	*	100.00	*	100.00
56	SELENIUM (MGS)	.00	*	.00	*	.00	*	.00
57	COBALT (MCG)	19.22	*	21.07	*	21.07	*	20.46
58	ALCOHOL (GMS)	.00	*	.00	*	.00	*	.00
59	CAFFEINE (MGS)	.00	*	.00	*	.00	*	.00
60	REDUC. SUGAR (GMS)	63.00	*	65.65	*	69.17	*	65.83
61	ZINC (MGS)	9.34	*	10.46	*	11.22	*	10.34
62	MOLYBDENUM (MGS)	.01	*	.01	*	.01	*	.01
63	MANGANESE (MGS)	4.59	*	5.38	*	7.60	*	6.02
64	CHROMIUM (MCG)	157.16	*	174.69	*	201.21	*	177.69

Nitrogen Intake and Output. Dietary intake of nitrogen assessed by computer analysis and Kjeldahl analysis. Urinary nitrogen excretion assessed by Kjeldahl analysis.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
Dietary nitrogen intake in g/day by computer analysis (g protein/6.25)															
Metabolic Diet															
Pre	13.8	14.9	13.7	14.8	13.7	13.0	14.0	13.4	12.3	15.5	13.7	12.4	12.4	12.2	14.8
Post	14.0	14.5	14.5	14.6	13.5	13.9	14.0	12.7	12.5	15.2	14.4	13.5	12.8	12.2	14.3
Food Records															
Mid	15.8	5.7	9.8	13.6	13.6	11.2	11.6	13.4	8.7	11.1	11.7	13.8	10.4	8.6	11.2
Dietary nitrogen intake in g/day by Kjeldahl analysis															
Metabolic Diet															
Pre	12.8	13.9	12.7	13.7	12.7	12.1	13.0	12.5	11.4	14.4	12.8	11.5	11.5	11.3	13.7
Post	13.0	13.4	13.5	13.6	12.6	12.9	13.0	11.9	11.6	14.1	13.3	12.6	11.9	11.8	13.2
Urinary nitrogen output in g/day															
Metabolic Diet															
Pre	10.8	11.3	9.8	10.3	10.0	10.0	10.9	10.7	8.9	10.3	10.4	8.6	8.2	9.8	9.1
Post	10.3	9.4	11.1	9.1	9.1	11.0	10.7	9.9	9.3	11.8	10.3	9.8	10.5	8.7	11.4
Food Records															
Mid	12.6	8.4	9.0	11.1	9.6	11.9	10.9	9.3	9.6	12.8	11.1	14.0	7.9	9.1	11.9

Food Records-1. Reported intake of calories and % kcals as protein, carbohydrate, fat, and alcohol.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
Calories															
Dec	3614	1615	1604	2849	3355	2849	3214	2102	1376	2387	1792	1865	1185	997	1930
Jan	2958	2174	1929	1850	2542	2965	2238	--	--	2738	2128	2703	2075	725	2390
Pre	3286	1894	1766	2350	2948	2930	2726	2102	1376	2562	1960	2284	1630	861	2160
Mid	2308	1014	1258	2566	3157	2065	1770	1971	1591	1583	1786	2086	1669	1291	2461
Post	1332	2099	1296	1856	1153	--	1647	1540	1507	1573	2115	1665	1999	1340	1771
% Kcal as Protein															
Dec	12	15	16	11	9	11	11	17	16	18	11	15	13	19	12
Jan	19	15	11	18	11	13	11	--	--	15	13	19	15	14	17
Pre	16	15	14	14	10	12	11	17	16	16	12	17	14	16	14
Mid	16	14	19	13	10	13	16	17	13	17	16	16	15	16	11
Post	17	16	17	15	11	--	15	19	12	15	13	18	12	22	16
% Kcal as Carbohydrate															
Dec	41	36	45	34	44	36	33	42	54	48	48	40	45	36	42
Jan	24	43	39	35	47	39	39	--	--	40	40	38	38	31	38
Pre	32	40	42	34	46	38	36	42	54	44	44	39	42	34	40
Mid	49	53	56	46	41	50	56	38	57	50	50	49	49	53	41
Post	51	58	55	48	60	--	58	34	52	55	55	48	51	35	43
% Kcal as Fat															
Dec	45	49	39	50	47	33	53	41	30	47	41	42	42	40	46
Jan	57	42	45	42	42	46	50	--	--	46	47	40	40	55	45
Pre	51	46	42	46	44	40	52	41	30	46	44	41	41	48	46
Mid	34	33	25	40	49	35	28	45	30	36	34	35	32	31	44
Post	32	26	28	30	29	--	27	47	27	36	32	34	33	34	41
% Kcal as Alcohol															
Dec	2	0	0	5	0	20	3	0	0	0	0	3	0	5	0
Jan	0	0	5	5	0	2	0	--	--	0	0	3	7	0	0
Pre	1	0	2	5	0	11	2	0	0	0	0	3	4	2	0
Mid	1	0	0	1	0	2	0	0	0	0	0	0	4	0	4
Post	0	0	0	7	0	--	0	0	9	0	0	0	4	9	0

Food Records-2. Reported intake of % kcals as sat, mono and poly fat, cholesterol, and fiber.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
% Kcal as Sat Fat															
Dec	17	16	11	13	11	12	16	18	8	16	10	14	15	12	15
Jan	27	12	14	17	14	23	18	--	--	16	14	16	16	21	16
Pre	22	14	12	15	12	18	17	18	8	16	12	15	16	16	16
Mid	13	14	7	14	16	12	10	14	9	14	10	15	12	13	14
Post	14	8	9	9	6	--	9	15	9	15	11	11	11	16	12
% Kcal as Mono Fat															
Dec	15	13	13	13	19	8	21	12	8	19	8	16	11	14	15
Jan	18	15	10	15	12	16	18	--	--	14	20	14	15	18	16
Pre	16	14	12	14	16	12	20	12	8	16	14	15	13	16	16
Mid	10	9	8	11	27	11	11	16	12	12	10	12	11	10	14
Post	10	7	7	7	22	--	9	15	8	11	9	10	12	11	15
% Kcal as Poly Fat															
Dec	3	9	3	10	11	4	10	4	3	6	5	8	5	7	7
Jan	6	8	4	4	8	4	10	--	--	6	9	4	4	9	5
Pre	4	8	4	7	10	4	10	4	3	6	7	6	4	8	6
Mid	4	2	5	5	11	4	4	5	5	3	8	4	6	3	6
Post	5	5	7	4	7	--	5	7	3	3	5	4	4	2	9
Cholesterol, mg															
Dec	391	212	353	466	274	526	571	329	142	364	149	550	114	246	420
Jan	575	208	462	295	200	458	434	--	--	331	156	624	233	311	442
Pre	483	210	408	380	237	492	502	329	142	348	152	587	174	278	431
Mid	252	100	137	455	215	309	167	490	114	163	142	314	302	160	205
Post	156	179	133	186	15	--	284	229	191	164	170	420	276	223	193
Fiber, g															
Dec	7.9	2.7	1.0	6.6	7.8	3.1	3.4	2.3	1.2	2.7	2.6	2.6	3.0	1.4	2.0
Jan	1.4	4.5	2.4	2.6	4.6	5.1	5.5	--	--	1.8	3.7	2.4	3.5	1.9	2.8
Pre	4.6	3.6	1.7	4.6	6.2	4.1	4.4	2.3	1.2	2.2	3.2	2.5	3.2	1.6	2.4
Mid	9.1	4.1	5.0	3.8	5.2	3.3	4.4	3.7	2.7	4.1	4.8	4.1	5.0	8.5	4.1
Post	3.5	7.4	3.8	3.0	2.9	--	5.4	2.6	1.6	4.7	4.5	4.6	5.6	2.3	3.0

Food Records-3. Reported intake of sodium, potassium, calcium, iron, zinc.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
Sodium, mg															
Dec	3823	2246	2615	5642	3349	2474	3699	2480	2094	1137	3664	2830	1687	1464	2834
Jan	4162	3182	3418	3169	2960	3802	3048	--	--	3219	4158	4548	1825	908	4459
Pre	3992	2714	3016	4406	3154	3138	3374	2480	2094	2178	3911	3689	1756	1186	3646
Mid	2603	2180	1783	3578	2400	2468	1698	2484	2450	2394	3088	2706	2243	1767	2715
Post	1943	3370	2353	2347	3242	--	2115	1726	2011	2136	2675	2509	1742	2590	2660
Potassium, mg															
Dec	3587	1690	1315	3023	3151	2257	2136	1926	1766	1528	1988	1683	1302	1021	1422
Jan	2528	2810	2233	2021	1944	2668	2853	--	--	2799	2857	2332	2134	776	2999
Pre	3058	2250	1774	2522	2548	2462	2494	1926	1766	2164	2422	2008	1718	898	2210
Mid	3307	1250	1899	2581	2352	1618	2890	2059	1604	2345	2661	2340	2339	2853	2719
Post	1347	5570	1705	2248	1136	--	2021	1453	1686	2592	3402	2440	2518	1866	1866
Calcium, mg															
Dec	1260	600	646	750	686	1530	720	1058	1289	1137	602	503	450	285	495
Jan	1759	653	588	797	995	1714	820	--	--	1468	790	1446	986	291	1110
Pre	1510	626	617	774	840	1622	770	1058	1289	1302	696	974	718	288	802
Mid	1166	482	576	844	702	795	879	582	837	1022	598	1230	775	604	656
Post	500	657	761	697	426	--	826	915	937	1028	1024	944	767	641	613
Iron, mg															
Dec	19.4	10.3	16.7	13.5	14.9	11.3	12.7	10.9	6.6	14.4	10.7	11.7	6.0	6.8	9.1
Jan	16.0	17.9	9.6	11.2	14.2	16.6	8.5	--	--	11.9	9.9	16.5	11.4	4.4	12.1
Pre	17.6	14.1	13.2	12.4	14.6	14.0	10.6	10.9	6.6	13.2	10.3	14.1	8.7	5.6	10.6
Mid	22.0	7.4	11.2	13.1	15.4	10.2	17.3	14.1	16.2	8.8	10.0	15.9	13.9	11.5	14.2
Post	8.1	16.1	18.1	11.6	6.8	--	13.8	9.1	13.2	10.1	11.9	15.1	14.0	8.0	8.6
Zinc, mg															
Dec	7.1	3.5	5.0	7.8	2.0	8.4	9.9	5.3	5.2	7.3	6.3	5.0	3.3	3.0	3.3
Jan	11.7	4.6	6.4	7.9	6.7	8.8	8.8	--	--	9.7	3.5	10.2	5.8	2.3	5.7
Pre	9.4	4.0	5.7	7.9	4.4	8.6	9.4	5.3	5.2	8.5	4.9	7.6	4.6	2.6	4.5
Mid	10.2	3.6	4.7	7.9	8.8	6.9	5.8	6.7	3.8	7.9	3.8	7.1	6.0	7.3	5.3
Post	4.9	5.1	6.3	4.5	1.4	--	5.5	4.8	5.1	8.6	4.5	6.7	4.7	8.0	4.4

Food Records-4. Reported intake of vitamins A and C, thiamin, riboflavin, and niacin.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
Vitamin A, IU															
Dec	4071	6845	6590	6328	2601	4530	3739	2872	2312	3687	5501	3040	1978	1392	5770
Jan	4111	5479	3290	12693	3543	10380	9277	--	--	3329	2708	4570	5912	2617	10311
Pre	4091	6162	4940	9510	3072	7455	6508	2872	2312	3508	4104	3805	3945	2004	8040
Mid	5947	3362	13380	5494	7573	3522	4398	5269	3371	3330	3363	11885	4774	34458	6147
Post	1599	2977	6696	2889	5507	--	5811	5320	4711	5519	3775	10918	7659	1951	4579
Vitamin C, mg															
Dec	68	79	34	145	36	22	17	29	10	68	127	114	69	25	20
Jan	42	149	62	74	24	48	172	--	--	62	164	13	58	28	65
Pre	55	114	48	109	30	35	94	29	10	65	145	63	64	26	43
Mid	92	74	40	93	56	48	46	97	130	58	177	141	175	86	93
Post	48	340	99	134	49	--	31	40	37	121	275	108	133	86	155
Thiamin, mg															
Dec	1.9	1.1	1.2	1.2	1.7	0.8	1.3	1.0	0.9	1.3	0.9	1.0	0.5	0.4	0.9
Jan	1.4	2.3	0.9	0.9	1.2	1.3	0.9	--	--	1.4	1.0	1.4	0.8	0.2	1.6
Pre	1.7	1.7	1.0	1.1	1.5	1.0	1.1	1.0	0.9	1.3	1.0	1.2	0.7	0.3	1.2
Mid	2.0	0.8	1.1	1.6	1.8	1.1	1.4	1.2	1.4	1.3	0.9	1.6	1.2	0.8	1.6
Post	0.7	1.7	1.5	1.3	0.9	--	0.7	1.1	1.5	1.5	1.2	0.9	1.1		
Riboflavin, mg															
Dec	2.2	1.7	1.3	1.5	1.2	1.9	1.6	1.9	1.8	2.0	1.0	1.1	0.7	0.8	1.1
Jan	2.4	2.0	1.7	1.5	1.6	2.5	1.6	--	--	2.4	1.2	2.6	1.3	0.6	2.3
Pre	2.3	1.9	1.5	1.5	1.4	2.2	1.6	1.9	1.8	2.2	1.1	1.8	1.0	0.7	1.7
Mid	2.6	0.9	1.2	1.5	1.5	1.7	1.8	1.5	1.7	1.8	1.1	2.0	1.3	1.1	1.7
Post	1.1	1.8	1.7	1.7	0.5	--	1.5	1.3	1.8	2.0	1.6	2.3	1.5	1.1	1.2
Niacin, mg															
Dec	28	23	21	20	36	15	22	13	9	21	11	19	10	17	14
Jan	24	34	17	18	17	23	10	--	--	18	17	28	27	6	21
Pre	26	29	19	19	26	19	16	13	9	19	14	24	18	11	18
Mid	27	11	19	20	27	18	15	21	20	13	21	22	19	13	23
Post	17	31	20	27	12	--	14	18	11	16	17	23	18	21	22

Food Records-5. Reported intake of vitamins B6 and B12, and folacin.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
Vitamin B6, mg															
Dec	1.6	1.2	0.8	1.2	0.8	0.8	1.3	0.7	0.5	1.1	0.6	1.2	0.4	0.7	0.7
Jan	1.5	2.4	1.4	0.6	1.1	1.4	1.2	--	--	1.3	1.1	1.6	1.3	0.2	0.8
Pre	1.5	1.8	1.1	0.9	0.9	1.1	1.2	0.7	0.5	1.2	0.8	1.4	0.8	0.4	0.8
Mid	1.5	0.4	0.9	1.2	0.2	0.6	2.0	1.2	1.3	1.0	1.0	1.3	0.7	1.2	1.3
Post	0.7	1.6	1.6	1.7	0.5	--	0.8	1.2	1.0	1.2	1.2	1.7	1.2	0.5	0.9
Vitamin B12, mcg															
Dec	1.8	1.0	5.4	1.9	1.1	4.0	3.0	2.0	4.1	2.5	9.6	2.0	0.4	6.4	1.6
Jan	3.3	7.1	2.3	1.6	3.1	7.8	3.8	--	--	4.0	1.1	4.2	1.3	1.0	3.6
Pre	2.5	3.6	3.9	1.7	2.1	5.9	3.4	2.0	4.1	3.3	5.3	3.1	0.8	3.7	2.6
Mid	2.8	1.2	1.7	2.9	0.2	3.1	2.7	5.2	3.5	3.2	2.2	3.1	1.0	0.7	4.2
Post	1.8	1.6	7.0	3.4	0.6	--	3.6	1.7	4.1	2.0	2.5	4.6	1.8	2.2	1.3
Folacin, mcg															
Dec	101	168	47	217	170	117	109	69	75	116	130	156	61	60	80
Jan	79	268	101	86	146	174	327	--	--	110	115	116	125	76	83
Pre	90	218	74	151	158	146	218	69	75	113	123	136	93	68	82
Mid	324	64	163	99	61	135	149	190	61	148	191	126	162	189	75
Post	99	242	150	235	93	--	144	86	79	329	364	210	226	128	54

Nutritional Status. Hematocrit, hemoglobin, serum iron, iron binding capacity, % saturation of transferrin, and plasma pyridoxal 5′-phosphate (PLP).

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
Hematocrit, %															
Pre	41.5	41.5	41.0	43.5	41.5	39.5	38.5	39.8	40.7	39.0	45.5	42.8	42.0	45.0	44.0
Mid	36.0	45.5	39.2	40.2	37.7	41.2	40.0	38.0	44.0	40.7	45.7	42.5	42.3	43.3	44.0
Post	38.8	42.7	35.0	44.8	39.8	39.7	39.3	38.5	39.0	41.8	45.0	42.3	43.5	41.8	44.3
Hemoglobin, g/L															
Pre	135	150	142	139	147	131	133	130	140	139	157	157	142	165	150
Mid	123	156	128	139	130	142	138	130	133	144	150	150	147	149	152
Post	136	168	119	152	140	141	135	139	136	156	156	153	151	146	155
Serum Iron, mcg/100mL															
Pre	74	40	43	63	116	108	68	81	72	55	107	95	81	93	86
Mid	36	69	35	81	44	102	110	53	31	90	48	69	69	95	62
Post	65	65	33	72	60	95	89	52	90	82	97	69	97	57	84
Iron Binding Capacity, mcg/100mL															
Pre	368	273	398	306	358	343	398	321	280	302	291	291	324	251	346
Mid	358	290	416	304	321	370	362	328	295	304	328	297	316	254	351
Post	312	277	373	333	365	327	383	291	312	312	318	291	330	219	362
% Saturation of Transferrin															
Pre	20	15	11	22	32	32	17	25	26	18	37	32	25	37	25
Mid	10	24	8	27	14	27	30	16	10	30	14	23	22	37	18
Post	21	24	9	22	16	29	23	18	29	26	30	24	29	26	23
PLP, nmol/L															
Pre	30.5	65.2	31.8	27.9	42.9	41.8	28.3	21.8	39.0	23.8	39.4	57.4	25.4	35.7	34.2
Mid	24.7	110.1	33.6	16.6	64.8	32.9	67.9	20.8	28.8	26.6	30.7	30.6	20.4	24.1	23.4
Post - pre diet	25.8	85.1	22.6	24.2	60.8	25.0	37.0	18.6	26.9	28.4	--	48.3	33.1	22.7	46.9
Post - during metabolic diet	31.6	107.3	19.1	25.9	69.8	33.0	34.6	37.5	50.9	33.5	--	44.0	48.9	39.3	46.3

V02 Max. Maximal aerobic capacity assessed by treadmill stress test.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
02, L/min															
Prestudy	2.54	2.35	2.23	2.85	2.44	2.80	2.55	2.27	2.03	2.63	2.73	2.20	2.39	2.14	2.49
Pre	2.50	2.44	2.07	2.84	2.41	2.84	2.60	2.25	1.90	2.54	2.79	2.23	2.35	2.27	2.50
Mid	2.79	2.55	2.45	2.81	2.58	3.02	3.29	2.32	2.18	3.11	2.85	2.24	2.72	2.31	2.82
Post	2.73	2.59	2.23	3.30	2.60	2.88	2.97	2.42	2.28	2.50	3.10	2.21	2.87	2.22	2.53
02, ml/kg/min															
Pre	22.2	24.3	27.3	26.3	26.7	31.9	24.1	28.3	26.9	23.0	27.0	21.0	28.1	27.0	24.9
Mid	24.7	28.2	30.7	27.2	29.1	32.4	31.9	28.3	30.2	29.0	27.6	20.7	33.1	27.7	28.1
Post	29.0	29.6	26.4	33.9	30.0	29.6	28.4	29.7	31.2	24.7	29.3	20.0	35.9	31.0	25.7
02, ml/kg FFW/min															
Pre	45.1	44.8	44.6	51.7	46.6	58.1	48.7	49.1	45.7	44.2	50.6	44.6	50.0	51.1	43.6
Mid	48.8	48.8	49.1	48.6	49.2	58.6	61.3	49.3	50.0	54.5	48.5	43.7	57.2	51.9	49.4
Post	50.1	48.6	45.4	59.4	49.9	56.3	56.8	51.2	52.4	44.1	54.2	44.2	62.4	51.1	45.7

Body weight change in kg throughout the project year. Value in parentheses is baseline body weight (measured during second week of testing, body comp day).

S#	Re-ported Wt	Pre-Study Wt	Base-line Wt	Feb	Mar	Apr	May	June	July	Aug	Sept	Oct	End-Point Wt	Post-Study Wt
C81	- 3.2	- 1.5	0 (112.7)	+ 1.5	+ 1.8	+ 0.4	+ 0.2	- 5.9	-10.4	-16.4	-19.1	-19.1	-18.6	-17.3
CF5	- 0.4	- 0.4	0 (84.5)	- 0.4	- 2.3	- 1.8	- 2.3	- 3.6	- 3.6	- 4.5	- 3.2	- 3.6	- 3.6	- 2.7
TF6	+ 2.3	- 0.9	0 (100.0)	0	- 2.7	- 6.8	- 9.5	-10.4	-10.4	-11.8	-11.8	-11.8	-12.7	-13.6
CG7	+ 1.4	- 3.2	0 (80.9)	+ 1.8	+ 3.6	+ 0.9	- 0.9	+ 0.4	+ 3.6	+ 3.6	+ 3.2	+ 2.7	+ 2.7	--
MJ8	- 0.4	- 1.8	0 (74.1)	0	- 0.2	- 1.4	- 1.8	- 2.7	- 2.7	- 3.2	- 2.3	- 2.3	- 1.4	--
DJ9	- 0.4	+ 0.9	0 (113.2)	0	- 0.4	- 2.7	- 5.9	- 7.3	- 8.2	-10.0	-10.0	-11.4	-12.3	-11.4
KK11	- 4.1	+ 0.4	0 (101.8)	- 0.9	- 0.9	- 0.4	+ 1.4	+ 0.9	+ 0.9	+ 1.8	+ 1.8	+ 3.2	+ 3.6	+ 3.6
JL12	+ 1.4	0	0 (107.7)	- 0.9	- 0.9	- 4.1	- 4.5	- 4.5	- 6.4	- 6.4	- 9.5	- 9.5	-10.4	-13.2
SL13	-10.0	- 1.8	0 (105.4)	+ 0.9	+ 1.4	+ 3.2	+ 3.2	+ 5.4	+ 5.0	+ 5.9	+ 6.4	+ 5.9	+ 5.0	+ 6.4
JM14	- 2.7	- 0.9	0 (91.4)	- 0.4	- 0.9	- 3.2	- 2.7	- 4.5	- 5.0	- 5.9	- 7.3	- 5.9	- 5.9	--
PM15	- 2.3	- 2.7	0 (88.2)	0	+ 2.3	+ 3.6	+ 4.5	+ 5.0	+ 7.7	+ 9.1	+10.0	+ 9.5	+ 8.6	--
JS16	+ 1.8	+ 0.9	0 (83.6)	- 1.4	- 0.4	- 0.4	- 1.4	- 3.2	- 3.6	- 3.6	- 2.7	- 4.1	- 3.2	- 3.6
HS17	- 1.4	0	0 (83.2)	+ 1.8	+ 0.9	+ 1.8	0	- 5.0	- 7.3	- 8.2	-10.0	-11.8	-11.8	-10.4
BW19	- 4.1	- 0.4	0 (99.5)	0	+ 0.4	+ 1.8	0	+ 0.4	- 0.9	- 0.9	- 2.3	- 1.8	- 2.3	- 0.4
PW20	+ 0.4	0	0 (107.3)	+ 0.4	0	- 1.8	- 4.5	- 4.5	- 3.6	- 4.5	- 5.0	- 3.6	- 3.2	- 0.9

Body Composition. Body weight and percent body fat determined by underwater weighing.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
Body weight, kg															
Prestudy	111.2	99.0	77.7	107.7	90.3	85.3	107.2	84.0	72.3	113.7	102.2	103.7	84.4	83.0	99.1
Pre	112.6	99.9	81.2	107.9	91.4	88.3	107.2	84.6	74.0	113.1	101.9	105.4	83.7	83.2	99.4
Mid	113.0	90.5	79.9	103.4	88.4	92.9	102.8	82.1	72.4	107.3	103.3	108.6	82.3	83.3	100.4
Post	93.9	87.4	83.7	97.3	85.7	97.0	104.2	81.0	72.5	101.0	105.4	110.4	80.4	71.4	97.4
% Body Fat															
Prestudy	49	45	38	48	42	43	49	43	42	48	44	51	45	46	42
Pre	50	46	38	49	43	45	50	42	40	47	46	53	44	47	43
Mid	49	42	38	44	41	45	48	43	40	47	43	53	42	47	43
Post	42	39	40	43	39	47	48	42	40	44	46	54	42	39	41

Resting metabolic rate. Oxygen and RER values.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
O2, L/min															
Pre-1	.260	.269	.208	.288	.257	.246	.261	.249	.223	.299	.239	.235	.237	.214	.282
Pre-2	.251	.268	.234	.290	.220	.238	.266	.223	.229	.291	.262	.237	.233	.220	.278
Pre-3	.265	.271	.261	.291	.232	.239	.262	.237	.218	.282	.261	.233	.235	.218	.263
Pre-4	.264	.272	.263	.266	.236	.240	.276	.237	.252	.291	.240	.236	.241	.230	.283
Mid	.226	.225	.253	.260	.236	.239	.281	.227	.226	.318	.272	.266	.248	.243	.273
Post-1	.227	.234	.241	.266	.213	.265	.254	.228	.202	.258	.254	.241	.242	.206	.230
Post-2	.223	.249	.257	.263	.232	.261	.249	.223	.214	.269	.229	.248	.242	.215	.270
Post-3	.230	.229	.247	.254	.205	.258	.254	.236	.219	.266	.252	.267	.228	.208	.263
Post-4	.247	.240	.264	.253	.220	.283	.256	.215	.214	.270	.244	.266	.207	.210	.262
RER															
Pre-1	.81	.83	.79	.79	.74	.79	.76	.80	.75	.77	.72	.75	.81	1.00	.80
Pre-2	.75	.83	.87	.86	.75	.84	.75	.82	.76	.79	.77	.72	.87	.79	.75
Pre-3	.76	.74	.90	.74	.69	.79	.82	.84	.72	.78	.73	.76	.69	.83	.74
Pre-4	.73	.82	.81	.77	.71	.82	.79	.76	.81	.78	.72	.76	.70	1.03	.74
Mid	.61	.84	.50	.78	.73	.66	.64	.73	.77	.56	.63	.54	.81	.59	.77
Post-1	.86	.85	.89	.84	.87	.86	.92	.89	.79	.84	.83	.78	.85	.92	.79
Post-2	.80	.85	.90	.93	.78	.86	.90	.85	.80	.82	.80	.78	.79	.92	.86
Post-3	.82	.89	.87	.86	.77	.84	.81	.88	.86	.99	.82	.73	.84	.94	.84
Post-4	.84	.87	.98	.86	.78	.92	.81	.90	.78	.80	.77	.78	.85	.92	.87

Pre-1 = RMR, Pre-2 = RMR-F+Ex, Pre-3 = RMR-F, Pre-4 = RMR-Ex

Activity. Average hours per day spent in 4 activity categories during 3-week testing periods.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
Sleeping															
Pre	7.5	8.8	7.5	8.4	7.5	9.6	7.0	7.9	8.2	7.1	6.8	9.6	7.0	7.7	7.4
Post	7.4	8.8	7.5	8.2	7.9	8.0	7.4	8.1	7.6	7.1	6.6	7.8	7.2	8.4	7.2
Sitting															
Pre	12.8	12.6	9.6	12.2	13.4	12.5	13.4	9.6	11.9	12.5	9.4	10.0	13.4	14.4	12.8
Post	9.2	11.4	7.7	12.8	11.1	12.0	11.0	8.5	11.2	8.9	7.4	7.9	11.4	10.9	13.3
Light Activity															
Pre	3.4	2.5	6.7	3.3	3.0	1.8	3.5	6.4	3.8	4.3	7.7	4.2	3.3	1.8	3.7
Post	7.0	3.0	8.5	2.4	4.6	3.4	5.2	7.0	4.8	7.7	9.6	8.0	4.8	4.4	3.1
Moderate Activity															
Pre	0.3	0.1	0.2	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.2	0.3	0.1	0.1
Post	0.4	0.9	0.3	0.7	0.4	0.6	0.4	0.4	0.4	0.3	0.4	0.3	0.6	0.3	0.4

Blood lipids. Values for plasma cholesterol, cholesterol fractions, and triglycerides.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
Total Cholesterol, mg/dL (mg/dL x 0.02586 = mmol/L)															
Pre	196	203	254	148	211	218	193	189	139	212	183	127	173	203	181
Mid	173	196	246	153	205	236	161	210	155	193	233	138	184	226	158
Post	148	165	212	140	174	232	183	206	142	200	170	114	176	174	172
LDL-Cholesterol, mg/dL															
Pre	141	137	172	94	141	137	127	126	84	147	124	69	112	136	126
Mid	129	123	181	106	143	147	105	142	65	134	171	73	119	160	96
Post	103	101	142	79	120	159	116	129	75	139	115	52	113	118	105
HDL-Cholesterol, mg/dL															
Pre	38	46	71	44	57	57	43	48	41	48	49	43	54	56	46
Mid	30	48	54	36	54	66	38	48	49	44	49	44	54	52	48
Post	36	46	57	46	44	52	38	54	41	46	44	48	54	49	48
LDL/HDL															
Pre	3.7	3.0	2.4	2.1	2.5	2.4	3.0	2.6	2.0	3.1	2.5	1.6	2.1	2.4	2.7
Mid	4.3	2.6	3.4	2.9	2.6	2.2	2.8	3.0	1.3	3.1	3.5	1.7	2.2	3.1	2.0
Post	2.9	2.2	2.5	1.7	2.7	3.1	3.0	2.4	1.8	3.0	2.6	1.1	2.1	2.4	2.2
Total Cholesterol/HDL															
Pre	5.2	4.4	3.6	3.4	3.7	3.8	4.5	3.9	3.4	4.4	3.7	3.0	3.2	3.6	3.9
Mid	5.8	4.1	4.6	4.2	3.8	3.6	4.2	4.4	3.2	4.4	4.8	3.1	3.4	4.4	3.3
Post	4.1	3.6	3.7	3.0	4.0	4.5	4.8	3.8	3.5	4.4	3.9	2.4	3.3	3.6	3.6
Triglycerides, mg/dL (mg/dL x 0.01129 = mmol/L)															
Pre	83	101	53	50	67	119	116	77	69	83	49	77	35	57	43
Mid	70	125	53	55	42	115	92	102	205	73	63	104	55	68	68
Post	47	92	65	76	49	106	144	117	129	77	56	71	44	33	96

Resting Blood Pressure. Early morning measurements, seated.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
Systolic Blood Pressure, mmHg															
Pre-1	110	112	118	120	114	108	134	110	100	112	121	106	124	98	126
Pre-2	--	114	122	124	124	--	--	102	100	112	110	114	108	--	132
Pre-3	104	104	108	124	104	100	129	106	108	110	118	102	114	90	126
Pre-4	118	120	118	118	114	104	124	102	110	120	114	110	120	104	130
Mid	122	99	118	118	100	116	124	98	104	118	118	108	116	106	128
Post-1	112	108	106	116	112	106	120	114	104	106	114	114	104	102	122
Post-2	112	110	124	114	120	108	114	104	106	120	120	112	106	102	126
Post-3	118	106	124	116	106	110	118	116	112	108	110	110	114	94	128
Post-4	114	110	128	110	104	118	116	106	102	114	106	104	106	104	116
Diastolic Blood Pressure, mmHg															
Pre-1	70	80	72	90	82	68	94	68	80	82	84	76	82	70	82
Pre-2	--	84	86	86	86	--	--	68	76	80	80	74	68	--	82
Pre-3	76	76	84	78	72	70	90	62	78	80	78	80	74	68	86
Pre-4	74	80	86	82	74	68	88	60	72	86	82	74	78	72	90
Mid	82	67	82	80	72	76	89	60	80	80	80	78	72	76	80
Post-1	78	70	78	82	76	80	80	66	74	70	84	70	68	76	88
Post-2	78	70	84	80	74	68	88	64	70	76	76	74	60	70	78
Post-3	68	80	84	74	72	78	86	70	68	78	72	70	68	70	80
Post-4	72	76	86	70	72	70	82	64	68	76	72	68	66	66	78

Exercise Blood Pressure. Systolic and diastolic blood pressure during 4 stage bicycle ergometer test.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
S8P-Seated on Bike															
Pre	122	118	122	128	118	116	140	114	114	120	122	120	122	120	136
Mid	126	112	118	116	120	120	128	106	118	116	118	112	114	116	132
Post	124	106	124	114	110	100	124	106	112	122	122	114	106	104	122
S8P-0 Watts															
Pre	128	132	144	128	134	126	144	118	128	130	124	132	128	122	148
Mid	142	128	132	--	124	132	136	118	116	136	124	132	--	130	136
Post	130	114	124	110	100	102	--	116	116	120	134	116	110	110	128
S8P-50 Watts															
Pre	138	128	146	138	132	128	148	122	134	140	134	140	128	136	172
Mid	146	136	140	138	130	134	144	122	124	134	128	134	132	128	140
Post	144	126	144	130	104	112	156	140	118	138	136	130	128	120	164
S8P-100 Watts															
Pre	150	148	162	152	152	132	158	158	160	154	146	150	150	146	186
Mid	152	148	168	146	144	138	154	168	136	154	136	144	140	140	148
Post	154	148	168	150	130	110	168	170	138	148	148	--	140	122	158
D8P-Seated Rest															
Pre	84	80	86	84	84	78	88	68	78	84	86	80	78	80	86
Mid	84	80	80	74	86	78	90	62	80	84	76	82	74	78	82
Post	74	80	84	74	78	78	90	66	78	82	82	74	78	78	82
D8P-0 Watts															
Pre	88	84	88	98	84	74	90	68	82	84	80	86	78	84	88
Mid	82	82	90	--	78	76	88	76	72	84	80	86	--	80	82
Post	72	74	90	68	74	68	60	70	74	80	70	70	70	76	70
D8P-50 Watts															
Pre	82	80	90	92	82	78	92	68	84	82	78	86	78	84	88
Mid	84	86	94	78	82	70	84	72	76	80	78	80	82	78	78
Post	72	78	90	68	76	72	78	66	80	76	80	78	72	72	72
D8P-100 Watts															
Pre	82	82	98	84	82	72	90	62	86	80	84	84	76	84	86
Mid	82	88	98	80	84	74	90	76	86	78	84	82	78	76	82
Post	76	78	96	78	74	60	70	60	80	78	70	--	76	70	72

Glucose Tolerance Test. Glucose values in mg/dL during GTT.

	CO-1	CO-2	CO-3	CO-4	CO-5	CO-6	CO-7	AO-1	AO-2	AO-3	AO-4	AO-5	AO-6	AO-7	AO-8
0 min															
Pre	87	77	76	85	85	87	82	77	85	99	77	70	74	86	81
Mid	83	74	71	76	79	86	85	77	85	89	71	78	74	85	73
Post	75	74	68	74	78	86	80	82	83	92	74	78	69	76	82
30 min															
Pre	106	88	119	108	122	76	128	146	132	169	105	98	103	118	114
Mid	68	98	93	102	138	124	136	139	129	162	120	98	110	122	103
Post	62	94	82	104	110	90	120	123	148	128	92	90	81	104	125
60 min															
Pre	85	123	113	86	98	83	142	119	109	189	--	86	68	73	98
Mid	68	98	93	102	138	125	136	139	129	162	120	98	84	79	110
Post	69	83	74	74	73	80	127	98	120	157	90	108	60	63	123
120 min															
Pre	74	84	85	69	93	82	79	98	98	105	76	104	71	75	79
Mid	85	67	74	74	79	59	143	101	131	104	77	105	82	86	109
Post	63	68	57	48	71	66	101	82	85	98	74	112	57	71	84
180 min															
Pre	67	82	67	68	64	68	48	65	78	99	48	95	62	54	48
Mid	74	80	44	67	47	74	84	63	105	90	49	90	74	64	81
Post	44	66	58	73	93	72	62	60	65	72	--	79	56	63	53
Integrated area under the curve above fasting, mg/dL/hr															
Pre	9.0	49.5	56.0	11.8	33.4	0	66.6	83.2	52.2	108.5	30.8	72.5	13.3	13.7	28.4
Mid	0	16.8	20.2	12.1	42.2	18.6	119.8	89.6	130.5	123.5	55.9	57.2	33.5	17.2	82.8
Post	0	15.0	10.7	15.0	14.9	1.4	71.4	33.0	61.4	70.4	21.0	63.0	4.7	11.8	53.3

(Conversion factor: mg/dL x 0.05551 = mmol/L)

APPENDIX II. Forms used in study.

SUBJECT EXPECTATIONS

Prior to beginning the study:

1. Complete health and physical activity forms.

2. Have a blood chemistry screen done through our lab. Our staff will draw the blood sample and send it to Good Samaritan Hospital in Corvallis for analysis.

3. Have a resting 12-lead electrocardiogram (ECG) done in the Human Performance Lab. This requires only that the subject lie quietly while electrodes are attached and ECG monitored. The procedure takes about 30 minutes.

2. Take the blood chemistry and 12-lead ECG information to personal physician, have a routine physical exam, and obtain physician's permission to participate. (We will pay for the routine physical exams up to $35.)

4. Provide some evidence of age of onset of obesity — medical or school records, or photographs.

5. Keep a record of menstrual cycle for two months.

During the study:

1. Attend three (3) 1-hour exercise (walking) sessions per week for 12 months with week long breaks at the end of fall, winter, spring, and summer terms.

2. Attend a nutrition education class 4x/month during months 1-4, 2x/mo. during months 5-8, and 1x/mo. during months 9-12.

3. Begin a walking routine the other 4 days of the week as physical ability improves due to training.

4. Keep records of food consumed and physical activity throughout the study. No mineral, vitamin, or other food supplements will be allowed during the study.

5. Consume a metabolic diet, eating all food in the metabolic kitchen in the Foods & Nutrition Dept. for 14-18 days at the beginning and again at the end of the study, and for a 8-10 day period during the 4th month testing period.

6. Participate in the following tests three (3) times during the study (1st, 4th, 11th months):

   a) Three-hour glucose tolerance test - This test involves coming to the nutrition lab early in the morning following a 12 hour overnight fast. A butterfly catheter will be inserted in an arm vein and an initial blood sample drawn. The subject then consumes a glucose drink and blood samples will be drawn 30 minutes, 1,2, and 3 hours after the glucose load. Ten mls of blood will be drawn each time. Subjects must remain in the lab sitting or lying quietly throughout the test.

b) Body composition determinations - This involves skinfold measurements, girths, and body diameters as well as underwater weighing. The procedures will require about 1-1/2 hours of the subjects' time.

c) Pulmonary capacity and function - These will be determined using standard procedures by breathing into a spirometer. This testing will be done during the same visit as the body composition determinations.

d) Exercise test to determine functional capacity - The test involves riding a bicycle ergometer at 3 separate submaximal workloads for 5 minutes at each workload. ECG and blood pressure is monitored each minute. Subjects will also be hooked up to a mouthpiece and breathing valve so inspired and expired air can be analyzed for oxygen, carbon dioxide, and total volume. After a 5 minute rest period, subjects will walk on a motor-driven treadmill at 3.0 mph at increasing grades until volitional fatigue is reached or end-point criteria as defined by the American College of Sports Medicine. ECG, blood pressure and ventilation will also be monitored each minute during this phase of the test.

e) Energy metabolism tests - Each subject will do all four tests described below. Each test will involve 4-6 hours of the subjects' time. They will arrive at the Human Performance Lab early in the morning and sit quietly for 30 minutes. Resting metabolic rate (RMR) will be measured via the mouthpiece and breathing valve for ten minutes. A butterfly catheter will then be inserted into an arm vein. Subject will sit quietly again for 30-60 minutes until their RMR returns to the initial level. At that point subjects will do one of the following:

1) sit quietly for a 4-hour period with RMR measured via the breathing valve for 10 minutes each half hour.

2) consume a 700 kcal meal then sit quietly for a 4-hour period with RMR measured for 10 minutes each half hour.

3) consume a 700 kcal meal, then ride a bicycle ergometer at 50% of their VO2 max for 40 minutes. For the following 4 hours they will sit quietly having RMR measured for 10 minutes each half hour.

4) no meal will be consumed but subjects will ride the bicycle ergometer and then sit quietly having RMR measured all in the same manner described in 3).

During tests 3) and 4), 20 mls of blood will be drawn through the butterfly catheter at 0, 20, 40, and 85 minutes. During the time subjects sit quietly following a meal or bicycle ride, they will be able to listen to the radio or read a book or magazine.

g) Blood lipid, iron, and B6 assessment - During each of the three test periods subjects will have 30 mls of blood drawn following an overnight fast for analysis of blood lipids, iron and B6 status.

h) Urine collections - Subjects will collect 24-hour urine specimens during the metabolic diet periods and all test periods. They will bring urine

to Milam Hall each morning and pick up new collection bottles.

i) Adipose tissue biopsies - At the beginning and end of the study subjects will have fat tissue biopsies done. Biopsies will be taken from the abdomen and buttock region. Dr. Robert Schwartz of the Seattle Veterans Administration Medical Center and the University of Washington will perform the biopsies at the Corvallis Clinic.

Qualified personnel will be conducting all of these tests. Medical Technologists will be involved in all blood drawing. People trained in doing graded exercise testing and CPR will conduct all the exercise and metabolic tests. Emergency procedures will be rehearsed and instructions posted at each testing site. Appropriate emergency facilities will be notified each time that testing is being conducted.

BENEFITS TO SUBJECTS

1. Extensive health, fitness, and metabolic evaluation will be done three times
   in a 12 month period. This kind of evaluation would cost subjects well over
   $500 if done on their own just one time.
2. Reduction in health risk factors associated with obesity through supervised exercise and nutrition education program.
3. Opportunity through the education program to develop lifestyle habits that will minimize health risk factors and allow maintenance of a recommended body weight over the long term.

Subject Consent Form

I, _____, give my consent to participate in this study. The study has been explained to me and all my questions have been answered. I understand that I will be expected to have a routine physical exam prior to beginning the study and have read the description of this in my "Subject Expectations" paper. I understand that I shall be expected to attend the exercise classes three times a week throughout the study, and the nutrition education classes on a weekly basis. I understand that I will be expected to keep records of my food intake and physical activity throughout the study, to abstain from taking any vitamin, mineral, or food supplements during the study, to consume all my food in the metabolic kitchen during the three testing periods, to do 24-hour urine collections during all testing periods, and to participate in the tests outlined in my "Subject Expectations" paper at months 1,4, and 11. These tests include a glucose tolerance test, body composition and pulmonary function tests, exercise test to determine functional capacity, four energy metabolism tests, blood lipid and nutritional status tests. I also understand that I am expected to have adipose tissue biopsies done at two sites (abdominal and buttocks), once at the beginning and again at the end of the study. I have read the attached description of the adipose tissue biopsy procedure to be used in this study. Any questions I have concerning this procedure have been answered. This procedure will be carried out at the Corvalis Clinic.

I understand the physical stress the exercise tests will place on me. Any symptoms such as chest pain, excessive shortness of breath, muscular cramps, etc. will be cause for discontinuing a test procedure. I understand that I may terminate the test on request at any time. There exists the possibility of certain changes occurring during an exercise test. They include abnormal

blood pressure, rapid or very slow heart beat, and very rare instances of heart attack. Constant surveillance by supervising personnel will help to anticipate such possibilities, and tests can thus be terminated where warranted. Emergency resusciation equipment and personnel are on constant standby if required to deal with an unusual situation that might occur.

I agree to allow blood to be drawn for the various tests as outlined in the "Subject Expectations" paper. I understand that there is a minimal risk of infection when blood is drawn, that sterile procedures will be followed to minimize this risk, and that a trained medical technologist will draw the blood samples. I understand there is a minimal risk of infection associated with each of the incisions made during the adipose tissue biopsy. I understand that sterile procedures will be followed to minimize the risk and I agree to follow the instructions for care of the two incision sites. I further understand that a local physician will be available should there be any problems following this biopsy procedure.

I understand that I am free to withdraw from the study at any time. I also understand that the investigators reserve the right to withdraw me from this study at any time.

I am assured that all information concerning me will be kept confidential. My name and address will not be on the medical questionnaire. All data will be coded and medical and exercise history forms will be kept in one place under the supervision of the principal investigator.

Name _____ Date _____

Witness _____ Date _____

Dr. Leklem
Dept. Foods & Nutrition
Oregon State University

Code_____

CONFIDENTIAL
Nutrition Project

Age:_____ Birth Date:_____

Predominant State of Residence:_____ City:_____ No. of Years:____

Present Employment:_____

Race (circle one): a. American Indian
b. Black
c. Caucasian
d. Latin American
e. Asian
f. Other (specify)_____

Marital Status (circle one): a. single
b. married
c. divorced/separated
d. widowed

*** *** *** *** *** *** *** *** *** *** *** ***

HEIGHT/WEIGHT: Height (feet and inches) _____ Present Weight _____

Length of time you have maintained your present weight _____

What is the most you have ever weighed? _____ What age? _____

How long did you maintain your heaviest weight? _____

What is the least you have weighed as an adult? _____ What age? _____

How long did you maintain your lightest weight? _____

How many times have you lost more than 10 pounds of body weight? _____

List below your age, the means used to lost weight each time (e.g. dieting, illness--please specify, exercise program, diet pills, etc.), and how long you maintained the reduced weight.

Age	How much lost	How lost weight	How long maintained the lost weight

Compared to others your age, describe your weight as:

an infant_____

a school-age child_____

a teen-ager_____

*** *** *** *** *** *** *** *** *** *** *** ***

MEDICAL HISTORY (check any conditions for which you have been diagnosed and give the age at diagnosis:

_____a. diabetes
_____b. hypothyroidism
_____c. hyperthyroidism
_____d. goiter
_____e. hypoadrenalism (Addison's disease)
_____f. osteoporosis
_____g. hepatitis
_____h. cirrhosis
_____j. kidney stones
_____k. nephritis
_____l. cystitis
_____m. high blood pressure
_____n. angina
_____o. mental depression requiring medication
_____p. insomnia requiring frequent medication
_____q. ulcers
_____r. pancreatitis
_____s. ulcerative colitis
_____t. spastic colon/diverticulitis
_____u. recurring gastritis
_____v. allergies
_____w. heart problems (specify)
_____
_____x. cancer (specify type)
_____

Have you ever had a glucose tolerance test? _____ yes _____no

If yes, please explain the reason and the results:

_____

Do any of your close relatives have diabetes? _____yes _____no

If yes, please check who of the relatives listed below had diabetes:

_____a. mother _____c. sister _____e. cousin _____g. uncle

_____b. father _____d. brother _____f. aunt _____h. grandmother

_____i. grandfather

*** *** *** *** *** *** *** *** *** *** *** ***

MEDICATION HISTORY (Check any which you take on a regular basis):

_____ a. sleeping tablets
_____ b. barbituates
_____ c. tranquilizers
_____ d. blood pressure tablets
_____ e. diuretics
_____ f. antibiotics
_____ g. oral contraceptives
_____ h. estrogens (female hormones)
_____ i. thyroid (thyroxin)
_____ j. insulin
_____ k. cortisone
_____ l. isoniazid
_____ m. other steroids (specify)
_____

*** *** *** *** *** *** *** *** *** *** *** ***

MENSTRUAL CYCLES/PREGNANCIES

What was the date you started your last menstrual period?_____

How many days are in your menstrual cycle (from beginning of one period to beginning of the next period)? _____

How regular are your menstrual cycles?

_____ Very regular (No more than 2 days variation in length of cycle)

_____ Fairly regular (2-5 days variation in length of menstrual cycle)

_____ Irregular (More than 5 days variation in length of menstrual cycle)

At what age did you begin menstruating? _____

Other than pregnancy, has there been a time when you stopped menstruating? _____ yes _____ no If yes, please describe_____

_____

How many times have you been pregnant? _____ How many full-term pregnancies? _____ At what age(s)? _____

*** *** *** *** *** *** *** *** *** *** *** ***

SURGICAL HISTORY (Please specify any type of surgery which you have had and the date and age when it occurred):

Surgery	Date	Age
_____	_____	_____
_____	_____	_____
_____	_____	_____

*** *** *** *** *** *** *** *** *** *** *** ***

DIETARY HISTORY:

Are you a vegetarian? _____ yes _____ no

If yes, circle the type of vegetarian diet that you follow:

a. ovo-lacto b. ovo c. lacto d. vegan

Do you take vitamins? (Circle one)

a. yes, daily b. yes, frequently c. never

If yes, what type, amount and how long have you taken them?

Type _____ Amount _____ How long? _____

Type _____ Amount _____ How long? _____

Type _____ Amount _____ How long? _____

Are you presently taking vitamins? _____ yes _____ no

If yes, what type, what amount and how often?

_____

Please list all foods which you refuse to eat, cannot eat or prefer not to eat.

_____

_____

*** *** *** *** *** *** *** *** *** *** *** ***

EXERCISE LEVEL

Do you have a daily fitness program? _____ yes _____ no

Please describe:

Dr. Leklem
Diet Study 1985
Food and Nutrition Dept.
Oregon State University

Name _____

Date _____

DAILY ACTIVITY SHEET

1. Record all activity for the previous day and length spent at each.

Activity	Length of Time (fraction of hours)	Time of Day*
Sleep		
Sitting		
Walking		
Physical work		
Other activities		
Other sports or activities (indicate type)		

* M - morning; A - after noon; E - evening; L - late night/early morning

2. Record all "free" foods in exact amounts used. Indicate type also used, decaf, etc.

Coffee (cups) _____

Tea (cups) _____

Diet Pop _____

3. How do you feel today? Excellent ____
Good ____
Fair ____
Poor ____

4. Any medications? (i.e., aspirin, etc.)

5. Other unusual events, exams, injuries, etc.

6. Did you turn your urine bottles in and pick up clean ones? _____

7. Your weight today _____

8. Other comments.

Guidelines for the Exercise Program.

1. The Type of Exercise to be engaged in. (Which Exercise)

2. The Frequency of the Exercise. (How Often)

3. The Intensity of the Exercise. (How Hard)

4. The Duration of the Exercise. (How Long)

5. The Supplemental Exercise Program.

The TYPE OF EXERCISE recommended is one which builds cardiorespiratory endurance and that leads to a gradual weight loss. This is done by increasing the rate of your energy expenditure to a level which is approximately 50% of your maximum aerobic capacity. We believe that cardiorespiratory endurance is the most essential physical fitness component, since our life depends on the capacity of the heart, lungs, and vessels to deliver nutrients and oxygen to the tissues and to remove wastes.

Efficient functioning of the heart and lungs is made possible through a progressive, consistant exercise program composed mainly of aerobic type activities such as walking, jogging, swimming and cycling. These exercise forms involve sustained rhythmic big muscle activity which call for a greater oxygen (aerobic) demand and caloric expenditure. This program will de-emphasize exercises involving intense bursts of activity lasting only a few minutes and will instead focus on mild exercise forms with increasing duration. Long, relatively fast walks will promote cardiovascular fitness and aid in weight loss because they are know to burn fats as the predominate fuel for the muscular activity.

PROGRAM CONCEPT

The goal of the Exercise Program is to participate in aerobic exercise at a moderate intensity (50 % of Max), a minumum of three (3) times per week. This activity should expend approximately 1000 calories per week by the 5th week. An optimal caloric expenditure is about 2000 calories per week. As your cardiorespiratory fitness improves the number of calories expended in exercise each week will gradually increase toward this optimal level. Please check the enclosed tables for the exact caloric values for a variety of exercise alternatives.

DETERMINING EXERCISE INTENSITY (HOW HARD).

Exercise should be done while within your TARGET ZONE for it to be most beneficial.

Your MAXIMUM AEROBIC CAPACITY IS _____ml/kg/min

This value is often describe in terms of METS. One MET is simply the amount of oxygen required to sustain all bodily functions at rest. Currently, you are able to increase your Met Level by a factor of _____. Therefore:

Your MAXIMUM AEROBIC CAPACITY IS _____METS.

The Training Intensity will range between 50 and 60 % of this MAXIMUM MET LEVEL.

Range_____METS.

To achieve this MET LEVEL you will have to walk on the level at approximately _____mph.

To assure that you are at your assigned target level you will need to monitor your heart rate during the activity.

YOUR TARGET HEART RATE FOR TRAINING WILL RANGE BETWEEN

_____BEATS PER MINUTE.

THE NUMBER OF PULSE COUNTS IN 6 SECONDS IS_____.

Counting your heart rate during exercise.

To determine whether your physical activity is eliciting a proper training stimulus (Target Zone) simply stop activity briefly to check your pulse. An accurate exercise HR can be estimated by taking your pulse for six seconds immediately after cessation of the activity. Be sure to skip the first beat that you feel. For example:

"COUNT" 0 1 2 3 4 5 6 7 8 9 10 11 12 13 "STOP"

[———————— 6 SECONDS————————]

The 6 second count was 13. By adding a 0 to the 13, we get a rate per minute equal to 130 beats. Try this.

Body Talk

a. Perceived exertion- Do you feel good while exercising? Breathe deeply, perspire freely, but feel good.

b. Do you feel good after exercise, or are you overly tired for the remainder of the day ?

c. Your 5-minute recovery pulse rate should be near 100 beats per minute, if it is over 120 you pushed yourself too hard.

d. Pain- Pain is the body's early warning system for impending problems. Excessive muscle or joint pain should not be ignored. Chest pain or discomfort should be reported immediately.

e. Morning heart rate- Elevated heart rate upon waking may be a sign of overtraining.

DURATION OF EXERCISE (HOW LONG).

The total exercise period will be 50 minutes in length for the first eight (8) weeks. It will include a 10 minute warm-up period of stretching and mild calisthenics. The "Aerobic" exercise period will last for thirty (30) minutues and an additional 10 minutes will be spent in cool-down and relaxation activities.

FREQUENCY OF EXERCISE (HOW OFTEN).

In the first few weeks of the program, it is recommended that exercise be limited to three (3) days per week. Each exercise day should be followed by a day of rest. As you get into better condition you may gradually extend your frequency to 4, 5, or 6 days per week. A varied program always seems to be more motivating and we will encourage the development of an active lifestyle. Three exercise sessions per week is considered minimal, however.

CALORIC EXPENDITURE

Researchers have found that individuals who burn 1000 calories per week in aerobic exercise are healthier than those who don't. By extending this energy output to 2000 calories per week we can make further improvements in our health. While burning more than 2000 calories per week in aerobic activities may be enjoyable and beneficial to some it is unlikely that additional health benefits are recieved. For these reasons we have determined that the expenditure of 1000 calories per week is good and that a progression of about 150 calories per month until we reach the 2000 calorie level be recommended.

Walking at _____mph for _____minutes will burn approximately _____calories. If we do this 3 times per week in the initial weeks of the program we will reach the goal of 1000 calories per week. The caloric expenditure values for other activities can be found by referring to Table 1.

HAVING FUN. We feel that an important component of our program is having fun while we participate. It is our belief that involvement in physical activity should be a joyful experience.

WEEKLY EXERCISE PLAN

Name _____ Week/Dates _____ / _____

Goals for the Week: 1.

2.

Target Zone_____Mets

Target Heart Rate_____beats per minute.

*to achieve this target I need to walk on the level at _____mph.

**this rate of walking will burn _____kcal/mile of walking

ACTIVITY	TIME	OBSERVATIONS

MONDAY : DAY ONE

1.

2.

3.

Pulse Check_____/_____
Calories Expended in Ex._____

TUESDAY : DAY TWO

1.

2.

3.

Pulse Check_____/_____
Calories Expended in Ex._____

WEDNESDAY : DAY THREE

1.

2.

3.

Pulse Check_____/_____
Calories Expended in Ex._____

Comments:

THURSDAY : DAY FOUR

1.

2.

3.

Pulse Check_____/_____
Calories Expended in Ex._____

FRIDAY : DAY FIVE

1.

2.

3.

Pulse Check_____/_____
Calories Expended in Ex._____

SATURDAY : DAY SIX

1.

2.

3.

Pulse Check_____/_____
Calories Expended in Ex._____

SUNDAY : DAY SEVEN

1.

2.

3.

Pulse Check_____/_____
Calories Expended in Ex._____

Number of miles walked _____miles

Number of calories expended in exercise_____kcal

Weight recorded on Friday_____lbs.

Blood Pressure recorded on Friday_____/_____mm/Hg

_____ _____
Signature (Participant) Signature (Instructor)

Comments:

FOOD TALK

A program aimed at decreasing body fat while increasing fitness levels and overall health is best done with a combination of increased physical activity and only a moderate decrease in caloric intake. You have already embarked on your walking program and are active right now improving your fitness level and ability to utilize body fat for fuel.

With regard to decreasing caloric intake, we are recommending reducing calories by only 500 per day. This will assure a loss of body fat of about one pound per week while at the same time preventing the decrease in metabolic rate that occurs when calories are decreased very much below that. This means that you should be able to continue losing one pound of body fat each week without the usual slowing down of the rate of weight loss as weeks go by. The number of calories you expend in walking and other physical activities each week will add to your rate of body fat loss.

The most desirable way to decrease caloric intake is to consume foods that are lower in fat and -- SURPRISE -- higher in carbohydrate, namely starch. Now, that may sound a little far-fetched to entrenched "dieters", but as you experienced during your three weeks on the metabolic diet, <u>a lot</u> of "starchy" foods could be consumed without gaining weight. Because gram for gram fat contains twice the calories that carbohydrate does, a little fat goes a long way calorie-wise while lots more carbohydrate can be eaten at a lower calorie cost.

The food pattern we would like you to learn to adopt is quite similar to the one you were eating during the metabolic diet. Only about 25% of the calories in this food pattern come from fat, in contrast to the 40-45% of calories coming from fat in the typical American diet. This food pattern is pictured on the next page.

Notice the predominance of foods of plant origin in comparison to those of animal origin. That is probably the simplest summary of this food pattern -- EAT FEWER FOODS OF ANIMAL ORIGIN AND MORE FOODS OF PLANT ORIGIN. Animal foods tend to be high in fat and low in complex carbohydrate (starches and fiber) while plant foods contain more complex carbohydrate and less fat.

An added bonus of selecting predominately low-fat foods is the decreased risk of developing diseases like cancer, heart disease, and diabetes. While we are interested in promoting habits that will decrease body fat we are also interested in your overall well-being. Low fat is obviously the way to go!

Below is an outline of the foods you consumed during the three weeks on the metabolic diet. The food groupings are listed on the left and the actual foods you consumed are listed in the middle. On the right are the quantities of each food that you consumed during the three weeks. The total amount of food for each group of foods is also listed.

Food Group	Foods Consumed	Amount	Total
Grains/Legumes	Wheaties	_____	
	Muffins	_____	
	Bread	_____	
	Rice	_____	
	Popcorn	4 c	_____
Vegetables			
Dk green/deep yellow	Carrots	1/2 c	
Starchy	Peas	_____	
	Corn	_____	
Other	Lettuce/Cabbage	1 c	_____
Fruits			
Citrus	Orange juice	1 c	
Other	Apple juice	1 c	
	Pears	1/2 c	
	Raisins	1 oz	2-3/4c
Meat or Alternate	Cheese	1-1/2 oz	
	Turkey	2 oz	3-1/2oz
Milk	Milk	1 c	
	Ice Milk	_____	_____

Added Fats	Margarine	_____	
	Dressing	_____	_____
Extra Calories	Cookies	_____	_____

* *

Based on the amount of food you ate over the three weeks on the metabolic diet, we estimate the number of calories needed to maintain your current body weight is _____. To achieve a one-pound per week loss of body fat, your daily caloric intake should approximate _____ calories.

* *

EATING PLANS

Listed below are several low-fat eating plans varying in calorie content. Each indicates the amount of food for the various food groupings that can be eaten in a day to equal the number of calories specified. Use the eating plan nearest your recommended calorie intake as a guide for planning foods to consume each day.

	Amounts of Food per Day				
	1500 Kcal	1750 Kcal	2000 Kcal	2250 Kcal	2500 Kcal
Food Groups *					
Grains/Legumes	2-1/2 c	3 c	3-1/2 c	4 c	4-1/2 c
Starchy vegetables	1 c	1 c	1-1/2 c	1-1/2 c	2 c
Vegetables	1 c	1-1/2 c	1-1/2 c	2 c	2-1/2 c
Fruit	1-1/2 c	1-1/2 c	2 c	2-1/2 c	3 c
Meat (lean) or alternate	3 oz	4 oz	4 oz	4 oz	4 oz
Milk	2 c	2 c	2 c	2 c	2 c
Fats, oils	2 tsp	3 tsp	4 tsp	5 tsp	6 tsp
Extra calories	1 serv	1 serv	1 serv	1 serv	1 serv
% Calories:					
as Fat	25%	26%	26%	25%	24%
as Carbohydrate	57%	55%	57%	58%	60%
as Protein	18%	19%	17%	17%	16%
Grams Protein	69	82	87	93	101

* See Food Group Lists for food equivalents

For comparison purposes, the amounts of food in each food group that you ate during the time you recorded food intake in December, January, and February is listed below.

	December	January	February
Grains/Legumes	_____	_____	_____
Starchy Vegetable	_____	_____	_____
Vegetable	_____	_____	_____
Fruit	_____	_____	_____
Meat or alternate	_____	_____	_____
Milk	_____	_____	_____
Fats, oils	_____	_____	_____
Extra calories	_____	_____	_____

FOOD GROUP LISTS

Grains/Legumes

"Cups" of grains/legumes refers to foods such as the following eaten in the cooked state.

Grains

Barley
Bulgur
Corn grits
Corn meal or polenta
Cous cous
Farina (cream of wheat)
Macaroni
Millet
Mixed grain cereals
Noodles
Oatmeal
Other pastas
Spaghetti
Rice
Rolled wheat
Wheat berries

Legumes (dried then cooked)

Blackeyed peas
Fava beans
Garbanzo beans
Kidney beans
Lentils
Lima beans
Navy beans
Pinto beans
Refried beans
Split peas
White beans

Other grain products are equivalent to "cups" of grains/legumes as outlined below.

This food	In this amount	Equals this amount of grains/legumes
Bagel	1 - 3-1/2" diam	1 cup
Biscuit	1 - 2" diam	1/2 cup + 1 tsp fat
Bread	1 slice	1/2 cup
Breakfast cereal:		
flakes, puffs, chex	1 cup	1/2 cup
Grape Nuts	1/4 cup	1/2 cup
Cake, see Extras		
Cookies, see Extras		
Corn bread	1 - 2" sq	1/2 cup + 1 tsp fat
Crackers:		
Akmak	5 - 2"x4"	1/2 cup
Graham	4 - 2-1/2" sq	1/2 cup
Oyster	20	1/4 cup
Rye	4	1/2 cup
Saltines	8	1/2 cup
Snack crackers	10	1/2 cup + 1 tsp fat
Donuts, see Extras		
Hamburger, hot dog buns	1 whole bun	1/2 cup
Muffins	1 - 2-1/2" diam	1/2 cup + 1 tsp fat
Pancakes	1 - 6" diam	1/2 cup + 1 tsp fat
Pocket or Mid-Eastern bread	1 - 6" diam	1/2 cup
Popcorn	4 cups	1/2 cup
Rolls:		
Brown & Serve or homemade type	1 - 2-1/2" diam	1/2 cup
Hard	1 - 3" diam	1/2 cup
Sweet, see Extras		
Tea breads like banana, zucchini	1 slice	1/2 cup + 1 tsp fat
Tortillas:		
Corn	1 - 6"	1/4 cup
Flour	1 - 8"	3/4 cup
Flour	1 - 12" (burrito size)	1 cup
Waffle	1 - 7" diam	1 cup + 1 tsp fat

Starchy Vegetables

Foods in this group include: corn, fresh lima beans, parsnips, peas (fresh, frozen, canned), potatoes both white and sweet, pumpkin, winter squash, yams. One "regular order" of french fried potatoes equals 1/2 cup of starchy vegetable plus 2 tsp of fat.

Vegetables and Fruits

These foods are listed according to the seasons when they are most available fresh.

Spring Apr May June	Summer July Aug Sept	Fall Oct Nov Dec	Winter Jan Feb Mar
Vegetables			
Artichokes	Beans	Broccoli*+	Artichoke
Asparagus*	Beets	Brussels sprouts*	Broccoli*+
Beans	Cabbage*	Cabbage*	Brussels sprouts*+
Cabbage*	Carrots+	Carrots+	Cabbage*
Carrots+	Celery	Cauliflower*	Carrots+
Celery	Corn	Celery	Cauliflower*
Cucumber	Cucumber	Corn	Celery
Greens*+	Eggplant	Eggplant	Greens*+
Lettuce	Lettuce	Lettuce	Lettuce
Mushrooms	Okra	Mushrooms	Mushrooms
Okra	Onions	Onions	Parsnips
Onions	Peas	Parsnips	Potatoes*
Parsnips	Peppers*	Peppers*	Rutabagas*
Peas	Potatoes*	Potatoes*	Squash, winter+
Potatoes*	Radishes	Pumpkin+	Sweet potatoes*+
Radishes	Squash, summer	Rutabagas*	Turnips*
	Tomatoes*	Squash, summer	
		Squash, winter+	
		Sweet potatoes*+	
		Tomatoes*	
		Turnips*	
Fruits			
Banana	Apricots+	Apples	Apples
Cantaloupe*+	Banana	Banana	Banana
Cherries	Berries*	Cranberries	Grapefruit*
Grapefruit*	Cantaloupe*+	Grapefruit*	Oranges*
Honeydew melon*	Cherries	Grapes	Pears
Oranges*	Grapes	Pears	Pineapple
Papaya*+	Honeydew melon*	Tangerines*	Tangerines*
Pineapple	Nectarines+		
	Papaya*+		
	Peaches+		
	Plums, prunes+		
	Watermelon +		

* High Vitamin C veggies/fruits
\+ High Vitamin A veggies/fruits

Lean Meat and Alternates

Meats and alternates to meat are arranged according to fat content per one ounce of the food unless otherwise indicated.

	Lean	+1/2 tsp fat/oz	+1 tsp fat/oz
Beef	Flank steak Hamburger (15% fat) Round, top	Hamburger (20% fat) Rib eye cuts Round steak	Chuck roast, steak Corned beef Hamburger (25% fat) Rib roasts Short ribs Steaks
Pork	Ham, trimmed Rump roast	Loin cuts Shoulder cuts Canadian bacon	Deviled ham Ground pork Spare ribs
Lamb	Leg Shoulder Loin chops & roasts	Arm, blade, or rib chops	Breast
Poultry	Meat without skin	Meat with skin	Duck, goose
Egg		1 whole egg or yolk	
Fish	Any fresh or frozen	Fried fish	
Processed Meats			Lunch meats Hot dogs Sausage
Cheese	Low-fat cottage cheese, 1/4 c Ricotta, part skim	Creamed cottage cheese, 1/4 c Ricotta, whole milk Farmers	American Cheddar Jack Parmesan Swiss Velveeta Cheese food, spread
Peanut butter			Peanut butter, 1 Tbs

Milk and Milk Products

Milk products equivalent to "cups" of low-fat (2%) milk are listed below.

This food	In this amount	Equals this amount of 2% milk
Milk, skim	1 cup	1 cup (you can use 1 tsp. of fat somewhere else!)
Milk, low-fat (2%)	1 cup	1 cup
Milk, whole (3.8%)	1 cup	1 cup + 1 tsp. fat
Milk Products:		
Ice milk	1/2 cup	1 cup
Yogurt, low-fat, plain	1 cup	1 cup
Yogurt, low-fat, fruit flavored	1 cup	1 cup + 1/2 serving "extra"
Yogurt, whole milk, plain	1 cup	1 cup + 1 tsp fat
Yogurt, whole milk, fruit flavored	1 cup	1 cup + 1 tsp fat + 1/2 serving "extra"

Fats and Oils

The quantity of each high-fat food that equals 1 teaspoon of fat or oil is listed below.

Food	Amount equal to 1 tsp of fat
Avocado, 3-1/2" diam	1/8
Butter	1 tsp
Bacon fat	1 tsp
Bacon	1 strip
Cream, light	2 Tbs
Cream, heavy	1 Tbs
Cream, sour	2 Tbs
Cream cheese	1 Tbs
French dressing	1 Tbs
Italiam dressing	1-1/2 tsp
Lard	1 tsp
Margarine	1 tsp
Mayonnaise	1-1/2 tsp
Nuts:	
Almonds	10 whole
Peanuts	10 whole
Pecans	2 whole
Walnuts	6 small
Other	6 small
Oils (corn, cottonseed, olive, peanut, safflower, soy, sunflower)	1 tsp
Roquefort dressing	1-1/2 tsp
Salad dressing (like mayonnaise)	1 Tbs
Salt pork	3/4" cube
Thousand Island dressing	1-1/2 tsp

Extras

Approximate sizes of one "serving" of foods and beverages in this group are listed below.

Food and Amount
Beer, 12 oz
Brownie, 2" square
Cake, frosted 2-layer, 1/16 of cake
Chips, 30 average size
Chocolate candy, 2 oz
Cookies, 2 average size
Cupcake, frosted, 2-1/2 " diameter
Donut, 1 average size
Ice cream, 1/2 cup
Pie, 1/8 of 9" pie
Pudding, 1/2 cup
Soft drink, 12 oz
Sweet roll, 1 average size
Wine, 4 oz

"The Beginning"