fatty acids, elevated impairments in mitochondrial function, or inflammation associated with chronic over-nutrition [79, 107, 124, 125]. Weight loss via caloric restriction is known to attenuate or reverse insulin resistance [126, 127]. Caloric restriction combined with exercise has also been shown to improve insulin sensitivity [124, 128-131]. Reduced insulin resistance after weight loss may be primarily mediated by a reduction in fatty acid mobilization [124].

As previously mentioned, insulin resistance is associated with an atherogenic lipoprotein profile. The typical dyslipidemic profile of metabolic syndrome is characterized by elevated triglycerides, a low level of HDL-c, and a preponderance of small, dense LDL-c particles. Plasma levels of LCD-c may be normal or modestly elevated [119, 132, 133]. Atherogenic dyslipidemia has been shown to be associated with an increased risk of cardiovascular disease [134]. Furthermore, a large meta-analysis of 37 studies (172,573 patients) found that the presence of metabolic syndrome almost doubles the risk of cardiovascular events and death (odds ratio, 1.78; confidence interval 1.58-2.00) [135].

Diminished Quality of Life

Obesity is not only characterized by significant clinical repercussions, but is also has negative effects on levels of independence, psychological well-being and overall quality of life [136, 137]. Disability may be defined as a restriction in ability to perform an activity in any domain of life [138]. One tool extensively used for the assessment of health related quality of life (HRQL) is the Medical Outcome Survey Short-Form 36 (SF-36). This instrument measures the extent of HRQL in both physical and mental domains. The questionnaire consists of 36 multiple choice questions measuring 8 different

domains; 4 in the area of physical health (physical functioning, role limitation-physical, bodily pain, general health) and 4 in the area of mental health (role limitation-emotional, vitality, mental health, and social functioning) [139-141]. Increasing BMI is associated with progressively impaired HRQL, with physical status affected more profoundly than mental health [142-144]. Furthermore, moderate weight loss is associated with improved health related quality of life [142, 145].

Non-pharmacological Treatment of Obesity

Dietary Intervention

A great deal of research has been conducted in order to ascertain the macronutrient nutrient composition that elicits optimal weight reduction and superior improvements in cardiometabolic health factors. Very low carbohydrate (CHO) (< 20-50 g CHO/d) ketogenic diets, diets high in carbohydrate combined with low fat and moderate protein, and diets containing elevated protein combined with moderate amounts of carbohydrate and fat have been extensively investigated [146]. Several studies have shown that very low-carbohydrate diets can produce significantly greater weight loss than isocaloric low-fat diets and improve markers of metabolic syndrome and cardiovascular risk factors in both healthy persons and in persons with type 2 diabetes [146-152]. In a systematic review comparing low-carbohydrate/high protein diets (< 20-60 g CHO/d) to low-fat/high-carbohydrate diets (fat $\leq$ 30% daily intake) in 1,222 adults (BMI > 28 kg/m^2), low-carbohydrate/high protein diets were more effective at 6 months and as effective if not more effective in reducing weight and cardiovascular disease risk factors at 1 year. The review also revealed a higher attrition rate for the low fat/high

carbohydrate groups versus the low carbohydrate groups suggesting a participant preference for the later [153]. Though very low carbohydrate diets have been shown to be efficacious for weight reduction, they restrict fruit and vegetable intake. This may lead to vitamin and mineral deficiencies or constipation if the diet if followed for a prolonged period.

Diets with elevated protein content combined with moderate levels of fat and carbohydrate have also been extensively studied. The definition of a high protein diet varies considerably in the literature, with intakes ranging from 27-68% of daily energy intake or 90.5 to 284 g/d in absolute amounts. Most investigations have compared a high protein diet to a diet high in carbohydrate and low in fat. In a study by Noakes et al. [154], 100 women (mean$\pm$SD: age 49$\pm$9 years; BMI 32$\pm$6 kg/m^2) followed either a high protein diet (34% protein, 46% CHO, 20% fat) or a high carbohydrate diet (17% protein, 64% CHO, 20% fat) for 12 weeks. There were no significant diet composition effects on fasting LDL-c, HDL-c, glucose, insulin, FFA, and C-reactive protein concentrations. However, triacylglycerol (TAG) concentrations decreased more with the high protein diet. Furthermore, subjects with high serum TAG levels ($\geq$ 1.5 mmol/L) had significantly greater reductions in fat mass and TAG concentrations following the higher protein diet.

In a study conducted by Layman and associates [23, 29], women (n=24; 45 to 56 years; BMI $\geq$ 26 kg/m^2) were assigned to either a carbohydrate group consuming a isoenergetic diet with a CHO/protein ratio of 3.5 (68 g protein or ~.8 g/kg/d) or a protein group with CHO/protein ratio of 1.5 (125 g protein/d or ~1.6 g/kg/d) for 10 weeks. Both

diets had similar amounts of fat (~ 50 g/d or 30% energy intake). Subjects in the protein group had a significantly higher loss of fat/lean tissue compared to the carbohydrate group. The high protein group had significant reductions in triacylglycerol levels and the ratio of TAG/HDL cholesterol, whereas both groups had significant reductions in total cholesterol levels. In contrast to the protein group, the carbohydrate group had higher insulin responses to meals and postprandial hypoglycemia. Thus, the isocaloric higher protein diet had positive effects on body composition, blood lipids, glucose homeostasis and satiety.

Due and associates [24] studied 50 overweight and obese subjects (age 19-55; BMI 26-35 kg/m^2] who followed either a high protein diet (25% protein, 45% CHO, 30% fat) or a "medium protein" diet (12% protein, 58% CHO, 30% fat) for 6 months followed by a 6-12 month dietary counseling period and a subsequent 24 month follow-up. The high protein group had a significantly greater weight loss and fat mass reduction at 6 months, while at 12 months there was no difference between groups. The high protein group did have a 10% greater reduction in intra-abdominal adipose tissue than the medium protein group at 12 months. Both groups tended to maintain their 12 month weight loss at 24 months, although 50% of subjects were lost to follow-up. There were no differences between groups in serum markers of metabolic syndrome and cardiovascular risk factors.

In an investigation conducted by Skov et al. [25], 60 healthy, overweight and obese subjects followed either a high-carbohydrate (12% protein, 58% CHO, 30% fat) or high protein (25% protein, 45% CHO, 30% fat) ad libitum diet for 6 months. There was a

significantly greater reduction in weight, total body fat, and intra-abdominal adipose tissue in the high protein group compared to the high-carbohydrate group, whereas the control group experienced no body composition changes. The greater weight loss (8.9 vv. 5.1 kg) and fat loss (7.6 vs. 4.3 kg) most likely occurred secondary to lower energy intake [5.0 (1,194 kcals) vs. 6.2 Mj/d (1,480 kcals)] in the high protein group. Additionally, more subjects lost > 10 kg in the high protein group. Total cholesterol and HDL-C decreased in both diet intervention groups with no group differences, while no significant changes were observed in the control group. The high protein diet had a significantly greater reduction in fasting plasma TG and free fatty acids than the high carbohydrate group.

Sacks et al. [26] assigned 645 subjects [mean$\pm$SD; age 52$\pm$9 years; BMI 33$\pm$4 (kg/m^2)] to either a low-fat, average protein diet (15% protein, 65% CHO, 20% fat) to a low-fat, high protein diet (25% protein, 55% CHO, 20% fat), a high-fat average protein diet (15% protein, 45% CHO, 40% fat) or a high-fat high protein diet (25% protein, 35% CHO, 40% fat). Reductions in body weight, waist circumference, blood pressure, and serum triglyceride levels did not differ significantly between the 4 groups at either 6 or 12 months. All diets except the one with the highest carbohydrate content (low-fat, average protein diet) decreased fasting serum insulin levels. Additionally, at two years, the low-fat diets decreased LDL-C significantly more than the high fat diets. The investigators concluded that macronutrient composition of diets is less important than calories for weight loss over 2 years [26].

Few investigations have studied the effects of diets where > 40% of calories are derived from protein. In a 4 week study conducted by Luscombe-Marsh and colleagues [27], a low-fat, high protein diet (40% protein, 30 % CHO, 30% fat) was compared to a high–fat standard protein diet (20% protein, 30% CHO, 50% fat) in adults (n=57; mean BMI 33.8±0.9 kg/m^2). There were no differences between groups in weight loss, fat loss, REE, or in glucose and insulin response.

Advantages of Diets High in Protein

Proteins have been shown to be the most satiating macronutrient [20, 21, 155-159]. It has been hypothesized that protein-induced satiety is somehow related to relatively high increases in concentrations of anorexigenic hormones such as Glucagon-like peptide-1 (GLP-1), peptide YY (PYY), and Cholecystokinin (CCK), or decreases in orexigenic hormones such as Ghrelin. Some investigations have demonstrated significant changes in hormone concentration after consumption of a high protein meal for GLP-1 [160, 161], PYY [162], and CCK [161], while others have not [163]. Changes in GLP-1 may be dependent on the presence of carbohydrate, which stimulates GLP-1 release [21, 164]. Changes in hormones may also be dependent on the composition of exogenous peptides contained in diets [161] and the time frame of over which a diet is consumed [155]. Additional investigation is needed as changes in anorexigenic and orexigenc hormones have inconsistent relationships with satiety [161, 163].

High protein foods have also been shown to have a higher thermogenic effect than foods higher in other macronutrients [160, 165-168]. Protein has a gross energy value of 22-25 kJ · g $^{-1}$ and a net metabolizable energy of 13 kJ · g $^{-1}$, which is lower than either

fat or carbohydrate [22, 169]. Several trials have investigated the effect of diet composition on the thermic effect of food or diet-induced energy expenditure, a component of daily energy expenditure. High protein diets have consistently been shown to induce higher energy expenditure than diets higher in other macronutrients [170-172].

While there is a general consensus that protein increases satiety and stimulates dietary–induced thermogenesis above other macronutrients, the effect of elevated protein intake on body composition continues to be investigated. Several studies have shown that higher protein diets may increase total weight loss and increase the percentage of fat loss [23-25], while others have not [26-28]

Exercise Intervention

Exercise and exercise combined with caloric restriction have been shown to enhance weight loss and improve cardiometabolic risk factors. A Cochrane review examined 43 studies that included a total of 3,476 participants and found that exercise resulted in small weight losses across studies when compared with no exercise. Exercise combined with diet resulted in greater mean weight reduction than diet alone (-1.0 kg, 95% CI-1.3 to -0.7). Furthermore, increasing exercise intensity resulted in an increase in the magnitude of weight loss (-1.5 kg, 95% CI -2.3 to -0.7). Exercise alone resulted in significant reductions in triglycerides (-0.2 mmol/L; 95% CI -0.3 to -0.1), diastolic blood pressure (-2 mmHg; 95% CI -4 to -1), and fasting glucose (-0.2 mmol/L; 95% CI -0.3 to -0.1) [30]. Exercise induced weight loss has also been associated with physiological and biopsychological changes including an improved satiety response to meals and improved sensitivity to appetite control systems leading to reductions in hunger [31, 32].

The International Association for the Study of Obesity (AISO) has published a consensus statement on physical activity recommendations for obesity prevention and weight maintenance. For prevention, moderate intensity activity of approximately 45-60 minutes per day may be required to prevent the transition to overweight or obesity. In order to maintain significant weight loss, 60-90 min of moderate intensity physical activity may be required daily [173].

High Protein Diet Combined with Exercise

Investigations combining high protein diets with exercise are surprisingly rare. In a study conducted by Layman and associates [33], 48 women (aged 40-56y; BMI $\geq$ 26 kg/m^2) were assigned to either a high protein diet (30% protein, 40% CHO, 30% fat; carbohydrate/protein ratio < 1.5) with lifestyle activity group (n=12), a high protein diet combined with exercise group (n=12), a high carbohydrate diet (15% protein, 55% CHO, 30% fat; carbohydrate/protein ratio > 3.5) with lifestyle activity group (n=12), or a low high carbohydrate diet combined with exercise group (n=12) for 4 months. The high protein and high carbohydrate diet plus lifestyle activity groups walked for 30 minutes 5 d/wk. The "exercise" groups participated in walking for 30 minutes 5 d/wk combined with resistance training 2 d/wk (30 minutes of stretching and resistance exercise utilizing weight machines). All groups experience significant weight loss, however, weight changes were larger in the groups consuming the higher-protein diet (p=0.05). Exercise increased loss of body fat and preserved lean mass. Additionally, subjects in the high protein diet groups had greater reductions in triacylglycerol and maintained higher

concentrations of HDL-c, whereas, subjects in the high carbohydrate diet groups had greater reductions in total cholesterol and LDL-c.

Meckling and colleagues [34] randomized 44 overweight women (aged 20-62 years; BMI 25-30 kg/m^2) into a high protein diet group (n=10; protein/CHO ratio 1:1; 30% fat), a high protein combined with exercise group (n=14), a high carbohydrate group (n=8; protein/CHO ratio 1:3; 30% fat), or a high carbohydrate diet combined with exercise group (n=11) for 12 weeks. Exercise consisted of a supervised circuit training program 3 d/week. All groups experienced weight loss: -2.1 kg for the high carbohydrate group, -4.0 kg for the high carbohydrate combined with exercise group, -4.6 kg for the high protein group and -7.0 kg for the high protein/exercise group, with significant differences between all groups (p=0.05). The groups consuming a high protein diet had greater reductions in fat mass (determined by BIA) than those groups consuming a diet high in carbohydrate. The high protein group had a significant decrease in HDL-c, the high protein/exercise group had decreases in triglycerides, and both the high protein and high carbohydrate/exercise groups had decreases in total cholesterol.

Behavior Modification and Cognitive Therapy

Behavior modification is directed at changing behaviors that impede weight loss and disrupt weight maintenance. Behavioral modification techniques include stimulus control, goal setting and self-monitoring [38]. Cognitive therapy strategies aim at identifying and modifying aversive thinking patterns and mood states in order to facilitate weight loss [39]. Cognitive-behavioral therapy can occur individually or in a

group setting where treatment incorporates social support, problem solving, information dissemination, and encouragement to facilitate weight loss [40].

Behavioral and combined cognitive-behavioral therapies have been shown to facilitate weight loss. A Cochrane review of 36 studies and a total of 3,495 participants examined psychological interventions for the treatment of overweight or obese patients. Behavioral therapy resulted in greater weight reductions than placebo when assessed as a stand-alone weight-loss strategy (mean weight loss -2.5 kg, 95% CI -1.7 to -3.3). Furthermore, cognitive behavioral therapy combined with diet and exercise increased weight loss by 4.9 kg compared with diet and exercise alone (mean weight loss -4.5 kg, 95% CI -7.3 to -2.4) [36]. A systematic review conducted by brown and colleagues [37] also demonstrated that behavioral therapy techniques combined with diet and/or exercise can enhance weight loss.

Relevant Weight Watchers Research

The majority of Weight Watchers research has been conducted on the Weight Watcher's Pure POINTs program. In an investigation conducted by Heshka and colleagues [47], the Weight Watchers Pure POINTs program was compared to a self-help weight reduction program. Subjects enrolled in the Weight Watchers Pure POINTs program (n=171) had significantly greater reductions in body weight [mean+SD; -4.8$\pm$ 5.6 vs. -1.4$\pm$4.7 kg; p=0.001), body mass index (-1.7 $\pm$ 1.9 vs. -0.5 $\pm$ 1.6 kg $\cdot m^{-2}$: P=0.001), waist circumference (-4.3 $\pm$ 10.5 vs. -0.7$\pm$12.7 cm: p=0.05), and fat mass (-3.8 $\pm$7.0 vs. -1.5$\pm$7.6 kg: p=0.05) than the those in the self-help group (n=168) after 26 weeks of program participation. In a study comparing Weight Watchers to 3 other

commercially available weight loss programs available in the UK, subjects enrolled in the Weight Watchers Pure POINTs program for 6 months (n=16) experienced reductions in plasma LDL cholesterol (-12.2%: p= 0.01), and plasma triacylglycerol levels (-22.6%: p=0.01), in addition to a 9.0±5.6% reduction in body weight [45, 46].

Relevant Curves Research

In a study conducted by Kersick and colleagues [35], 161 obese, sedentary, premenstrual women (age 38.5±8.5 years; BMI 34.9±6.3 kg/m^2) were assigned to either a no diet control group (CON) (n=7), a no diet exercise group (ND) (n=17), a high energy, high carbohydrate low protein diet group (HED) (n=11; 2,600 kcalories; 15% protein, 55% CHO, 30% fat), a very low carbohydrate, high protein diet group (VLCHP) (n=48; 63% protein; 7% CHO, 30% fat), a low carbohydrate, moderate protein diet group (LCMP) (n=37; 20% protein; 50% CHO, 30% fat), or a high carbohydrate, low protein diet group (HCLP) (n=41; 15% protein; 55% CHO, 30% fat) for 14 weeks. The HED, VLCHP, LCMP and HCLP groups ingested 1,200 kcalories for 2 weeks, 1,600 kcalories for 8 weeks, and 2,600 kcalories for the remaining 4 weeks of the study. All groups except the control group performed circuit training for 30 minutes 3 times weekly. All groups excluding the CON group had significant reductions in waist circumference. The VLCHP, LCHP and LCMP groups had similar significant reductions in body mass ($P < 0.05 – 0.001$) in comparison to other groups. The VLCHP, LCMP and HCLP groups had significantly greater body fat percentage reductions when compared to the other groups. All exercise groups had analogous gains in muscular fitness ($P < 0.05$). Favorable but non-significant reductions occurred in lipid panels, glucose and HOMA-

IR levels, whereas, leptin levels significantly decrease in all groups excluding the CON group. The exercise groups also experienced significant improvements in quality of life and body image indices. Finally, resting energy expenditure lowered during the 1,200 kcalorie phase, but returned to baseline levels or above during the 1,600 kcalorie phase and further increased during the 2,600 kcalorie diet phase for the VLCHP, LCMP, and HCLP groups.

In another investigation, 43 overweight sedentary women (age 35±8 yrs; BMI 33±6 kg/m^2) were assigned to either a high carbohydrate diet (n=23; 30% protein, 45% CHO, 25% fat) or to a high protein diet (n=20; 45% protein, 30% CHO, 25% fat). Both groups ingested 1,200 kcalories for 1 week, 1,500 kcalories for 3 weeks, and 2,200 kcalories for the remaining 4 weeks. This cycle was repeated for the duration of the study. Both groups performed circuit training for 30 minutes 3 times weekly and walked briskly for 30 minutes 3 days/week. There were no significant differences between the two groups. Overall, training combined with caloric restriction resulted in significant reductions in body mass, fat mass, percent body fat waist and hip circumferences, resting systolic blood pressure, and serum triglyceride levels. Favorable but non-significant reductions occurred in total cholesterol and LDL-c levels. There were no significant changes in glucose or HDL-c levels. Absolute REE values modestly decreased over time, but were not significantly changed when weight loss was accounted for. Training also induced gains in cardiopulmonary fitness and muscular strength. Furthermore, training and dieting improved quality of life [50-53].

CHAPTER III

METHODS

Participants

A total of 149 sedentary overweight females (BMI > 25) between the ages of 18 and 50 years were recruited to participate in this study. Of these, 127 met phone interview entrance criteria and were invited to attend familiarizations sessions. Subjects had not been involved in an anaerobic or aerobic exercise training program for at least the last three months and had not taken ergogenic levels of nutritional supplements that could affect muscle mass or anaerobic exercise capacity (i.e. creatine, ergogenic levels of caffeine, HMB, etc.), anabolic/catabolic hormone levels (i.e. androstenedione, DHEA), or weight loss (i.e. ephedra, thermogenics) for at least three months prior to the start of the study. Participants with uncontrolled metabolic disorders, known electrolyte abnormalities, heart disease, arrhythmias, diabetes, or thyroid disease; a history of hypertension, hepatorenal, musculoskeletal, autoimmune, or neurological disease were not allowed to participate in the study. Participants with a controlled medical condition had their physicians complete and sign the physician's consent form prior to participation in the study. Subjects meeting eligibility criteria were informed of the study requirements and signed an informed consent statement in compliance with the Human Subjects Guidelines of the Texas A&M University and the American College of Sports Medicine.

Study Site

Familiarization and testing in addition to Curves circuit workouts took place in the Exercise and Sport Nutrition Laboratory in the Department of Health and Kinesiology at Texas A&M University in College Station, Texas.

Experimental Design

Table 1 shows the general research design and time course for assessments. The study included a baseline testing session followed by 5 additional testing sessions. Exercise testing occurred at the baseline and final testing session only. During testing sessions 2 through 5, only resting tests were conducted. The independent variable was weight loss program intervention. Dependent variables included: estimated dietary energy intake; estimated weekly physical activity (International Physical Activity Questionnaire – IPAQ); hip and waist anthropometric measurements; resting energy expenditure (REE); body composition; fasting clinical blood profiles (cholesterol, glucose, liver enzymes, red cells, white cells, insulin, leptin); maximal cardiopulmonary exercise capacity (peak VO_2); maximum repetition maximum (1RM) and 80% 1RM Isotonic testing; standardized quality of life (SF-36); and eating satisfaction inventory.

Familiarization Session

Participants who expressed interest in this study were interviewed on the phone to determine whether they qualified to participate. Participants who met the eligibility criteria were invited to attend a familiarization session. During this session, participants received written and verbal explanation of the study protocol and design, testing procedures and equipment, and blood procedures that would occur throughout the study.

A registered dietitian provided instruction on accurate dietary record completion and estimation of portion sizes. Participant height and weight were attained. Participants then signed informed consent statements and completed personal and medical histories. Participants with controlled metabolic disorders were required to obtain medical clearance from their personal physician prior to participating in the study. Participants

Table 1. Overview of Research Design and Testing Schedule

Familiarization	Baseline (T1)	4 Weeks (T2)	6 Weeks (T3)	10 Weeks (T4)	12 Weeks (T5)	16 Weeks (T6)
Complete Paperwork Review Medical history Randomized assignment	Diet Record Review IPAQ[a] Body Weight Hip and Waist Measurements Resting Energy Expenditure Resting BP[b] and HR[c] DEXA[d] Scan Fasting Blood Maximal Cardiopulmonary Exercise Test 1RM[e] and 80% 1RM Isotonic Leg Press and Bench Press Measures Survey Completion[f]	Diet Record Review IPAQ Body Weight Hip and Waist Measurements Resting Energy Expenditure Resting BP[a] and HR[b] DEXA[c] Scan Fasting Blood Survey Completion	Diet Record Review IPAQ Body Weight Hip and Waist Measurements Resting Energy Expenditure Resting BP[a] and HR[b] DEXA[c] Scan Fasting Blood Survey Completion	Diet Record Review IPAQ Body Weight Hip and Waist Measurements Resting Energy Expenditure Resting BP[a] and HR[b] DEXA[c] Scan Fasting Blood Survey Completion	Diet Record Review IPAQ Body Weight Hip and Waist Measurements Resting Energy Expenditure Resting BP[a] and HR[b] DEXA[c] Scan Fasting Blood Survey Completion	Diet Record Review IPAQ Body Weight Hip and Waist Measurements Resting Energy Expenditure Resting BP and HR DEXA Scan Fasting Blood Maximal Cardiopulmonary Exercise Test 1RM and 80% 1RM Isotonic Leg Press and Bench Press Measures Survey Completion

[a]International Physical Activity Questionnaire
[b]Blood Pressure
[c]Heat Rate
[d]Dual Energy X-ray Absorptiometry
[e]Repetition Maximum
[f]Standardized quality of life (SF-36) and eating satisfaction inventory

were then matched according to body mass index (BMI) and age into either the Curves (C) group or the Weight Watchers (W) group.

Curves Fitness and Weight Management Plan

Participants in the C group followed the Curves Fitness and Weight Management Plan. In concurrence with this plan, subjects followed the Phase 1 higher protein weight loss diet (1,200 kcals/day) for one week and the phase 2 high protein weight loss diet (1,500 kcals/day) for 3 weeks. Phase 3 consisted of a metabolic recovery phase. During phase 3, subjects followed a 2,000-2,500 kcals/d higher protein diet for two weeks. If subjects had a weight increase of 3 pounds during phase 3, they were instructed to return to Phase 1 for 2-3 days until their weight returned back down. The three dietary phases were cycled in this manner over the course of 16 weeks. A registered dietitian reviewed the Curves high protein diet (HPD) and exercise plan with subjects and provided them with the "Curves Fitness and Weight Management Plan" [49], "The Curves Food & Exercise Diary" [174], and "Curves Essentials 2 go" dietary supplements. Participants were encouraged to consume two multivitamins, 800mg of calcium, and 520mg of Omega-3 fatty acids daily. The dietitian also met with subjects weekly throughout the study to discuss any dietary challenges or concerns. The Curves Fitness and Weight Management Plan dietary intervention protocol is delineated in Table 2.

The Curves physical training protocol included one regular curves circuit work out and two Curves Zumba workouts each week for 16 weeks, while maintaining a greater than 80% compliance record. Attendance was recorded at each workout session in order to monitor compliance. The Curves circuit consisted of 13 bi-directional

Table 2. Curves Fitness and Weight Management Dietary Plan						
Dietary Phase	Kcalories	Group	Macronutrient	g/d	Kcals/d	Percentage Daily Diet (%)
Phase 1 (1 Week)	1,200 kcals/d[a]	HPD[b] + Exercise	PRO[c] CHO[d] FAT	135 90 33	540 360 300	45 30 25
Phase 2 (3 Weeks)	1,500 kcals/d	HPD + Exercis	PRO CHO FAT	169 113 42	675 450 375	45 30 25
Phase 3 (2 weeks)	2,000-2,500 kcals/d 14 Day Metabolic Recovery * Subjects can adjust caloric intake as needed to maintain weight (e.g., 2,000 – 2,500 kcals/d) with 2,200 kcals/d given as an example starting diet. * If subjects gain 3 lbs during the metabolic recovery period, they will follow the 1,200 kcal/d diet for 2-3 days to adjust weight back down	HPD + Exercise	PRO CHO FAT	165 248 61	660 990 550	30 45 25
[a]Kcalories per day [b]High protein diet. [c]Protein [d]Carbohydrate						

hydraulic resistance exercise machines that worked all major muscle groups. During the Curves circuit workout, subjects were instructed to complete as many repetitions as

possible during a 30 second time period on each resistance machine. Between machines, subjects performed floor-based aerobic exercises designed to maintain an elevated heart rate. Subjects performed the entire circuit twice during the 26 minute regular circuit workout. During the Curves Zumba workout, subjects performed 1 minute of Zumba dance moves in-between 1 minute of resistance exercise on each machine. All Zumba classes were taught by a certified Zumba instructor and all training sessions were monitored by trained fitness instructors. Subjects were assisted with self-monitoring of heart rate in order to maintain an exercise heart rate between 60-80% of target heart rate using age-predicted maximal heart rate (220-age). Subjects also performed 5 minutes of whole body stretching after all circuit workouts. In addition to circuit training, subjects were encouraged to walk for 30 minutes at a brisk pace [60-80% of heart rate reserve (maximum HR- resting HR)] on most days of the week. The Curves circuit was located in the Exercise and Sports Nutrition Laboratory.

Weight Watchers Momentum Program

Participants assigned to the W group took part in the Weight Watchers Momentum Program and were signed up for a Weight Watchers monthly pass subscription. Subjects were required to attend 1 meeting each week with an 80% compliance record. Subjects emailed the study coordinator each time they attended a meeting. Meetings consisted of group discussions and some individual interaction as members weighed in. The core program material was delivered over ten weeks and included: food plans based on the POINTs system, exercise recommendations, strategies

for dealing with hunger, successful habits, strategies for eating out, tracking intake and activity, eating with others, recipes, workout variation, and plateaus.

The momentum eating plan revolved around the POINTS system. On the POINTS plan, subjects were assigned a daily POINTS target that was based on weight, height, age, and activity level. Subjects were also allowed additional POINTS each week, which could be utilized as needed. The POINTS values assigned to foods were based on portion size, calories, fat and fiber content. POINTS values were also provided for activity, and were based on weight, time, and intensity. Subjects were encouraged to track their intake of foods and beverages. The Weight Watchers Momentum program also encouraged subjects to perform 30 minutes of activity on most days of the week. Study participants were encouraged to purchase the Weight Watchers 2010 Basic Member Kit, which included: "Complete Food Companion POINTS", "Dining Out Companion POINTS", "Fast Fixes", and the Weight Watchers 3 Month Journal. Subjects also had access to web-based Momentum Program information with the monthly pass subscription. All subjects attended meetings at the Weight Watchers site located in Bryan, Texas.

Medical Monitoring

Interested participants filled out medical histories at the familiarization session. Based on review of this information, the study coordinator determined whether the participant had met entry criteria in order to participate in the study. Participants with uncontrolled metabolic disorders, known electrolyte abnormalities, heart disease, arrhythmias, diabetes, or thyroid disease; a history of hypertension, hepatorenal,

musculoskeletal, autoimmune, or neurological disease were not allowed to participate in the study. Participants with a controlled medical condition had their physicians complete and sign the physician's consent form prior to participation in the study.

In preparation for a medical emergency, the ESNL laboratory was equipped with an automated electronic defibrillator. All ESNL personnel were certified in CPR and first aid and were trained on emergency procedures. No less than two researchers worked with each participant during testing sessions. In the event of any emergency, one researcher would have checked for vital signs and began any necessary interventions while the other researcher would have contacted the Texas A&M emergency services at extension 9-911. Participants were instructed to report any unexpected problems or adverse events they encountered during the course of the study to the study coordinator Michelle Mardock, MS, RD. If clinically significant side effects were reported, the participant would have been referred to their personal physician. No adverse events were reported during the course of the study.

Testing Sessions

Participants were instructed to refrain from exercise for 48 hours and fast for at least 12 hours prior to reporting to the ESNL for testing sessions. Baseline testing sessions and 16 week testing sessions were identical and consisted of dietary inventory review, weekly physical activity assessment [International Physical Activity Questionnaire (IPAQ)], anthropometric assessments (body mass, waist and hip circumference); resting energy expenditure, resting heart rate and blood pressure, body composition analysis (DEXA); blood collection (metabolic panels, blood lipids, white

defined light physical activity as walking level intensities (3.3 METs), moderate physical activity as activities at a 4.0 MET level, and vigorous physical activity as activities at an 8.0 MET level. The IPAQ has been identified as a valid indicator of general changes in physical activity patterns [175-178].

Anthropometric Measurements

Height was measured using standard anthropometry. Total body weight was measured using a Healthometer (Bridgeview, IL, USA) self-calibrating digital scale with a precision of +/-0.02 kg. Hip and waist measures were performed using a Gulick II tape measure per guidelines established by the American College of Sports Medicine [179].

Resting Energy Expenditure Assessment

Resting energy expenditure assessments were conducted according to standard protocols using the Parvo Medics TrueMax 2400 Metabolic Measurement System (ParvoMedics, Inc, Sandy, UT, USA). This test was conducted in a fasted state with the participants lying supine on an exam table. A clear metabolic canopy was placed over the subject's head and neck, so that resting oxygen uptake and energy expenditure could be determined. The participants remained motionless without falling asleep for approximately 20 minutes. Metabolic measurements were recorded after the first 10 minutes during a five minute period in which principle variables, such as oxygen uptake, changed less than 5% [180]. Mean test-retest reliability studies on 14 participants from a previous study revealed test-retest correlations (r) of collected oxygen uptake range from 0.315 to 0.901 (mean 0.638) and a coefficient of variation range from 8.2% to 12.0% (mean 9.9%) with a mean intraclass coefficient of 0.942; $p<0.001$ [35].

Resting Heart Rate and Blood Pressure

Heart rate was determined by palpitation of the radial artery using standard procedures [181]. Blood pressure was assessed in the supine position after resting for 5 minutes using a mercurial sphygmomanometer (American Diagnostic Corporation, model #AD-720, Hauppuage, NY, USA) using standard procedures [182].

Body Composition

Participants had bone density and body composition (excluding cranium) determined using a Hologic Discovery W QDR series Dual Energy X-ray Absorptiometry (DEXA) system (Hologic Inc, Waltham, MA, USA) equipped with APEX software (APEX Corporation Software, Pittsburgh, PA, USA). Dual-energy X-ray absorptiometry has been validated as an accurate method for body composition assessment [183-186]. Mean test-retest reliability studies performed on male athletes with this Hologic system have yielded mean coefficients of variation for total bone mineral content and total fat free/soft tissue mass of 0.31% to 0.45% with a mean intra-class correlation of 0.985 [187]. Participants were informed of any inherent risks that could present from radiation exposure and completed a radiation exposure questionnaire prior to all scans. Quality control (QC) calibration procedures were performed on a spine phantom (Discovery W-CALIBER Model DPA/QDR-1 anthropometric spine phantom) prior to each testing session. During DEXA testing, participants lay down on their back in a standardized position. A low dose of radiation scanned their entire body for approximately six minutes.

The DEXA segments regions of the body (right arm, left arm, trunk, right leg, and left leg) into three compartments for determination of fat, soft tissue (muscle), and bone mass. Radiation exposure from DEXA for the whole body scan is approximately 1.5 mR per scan. This is similar to the amount of natural background radiation a person would receive in one month while living in College Station, Texas. The maximal permissible x-ray dose for non-occupational exposure is 500 mR per year. Total radiation dose was estimated to be less than 9 mR for the entire study.

Blood Collection and Analysis Procedures

Fasted and whole blood and serum samples were collected using standard phlebotomy techniques. Whole blood samples were analyzed for complete blood counts with platelet differentials utilizing an Abbott Cell Dyn 3500 automated hematology analyzer (Abbott Laboratories, Abbott Park, IL, USA). Serum samples were analyzed for a complete metabolic panel by Quest Diagnostics (Quest Diagnostics, 5850 Rogerdale Road, Houston TX, USA 77072) using an Olympus AAU 5400 Chemistry Immuno Analyzer (Olympus America Inc., Center Valley, PA, USA). Serum leptin was determined in duplicate using a commercially available enzyme linked immunoabsorbent assay (ELISA) kit (No. 11-LEPHU-E01, ALPCO, Salem, NH, USA) using a BioTek ELX-808 Ultramicroplate reader (BioTek Instruments Inc, Winooski, VT, USA) at an optical density of 450 nm against a known standard curve utilizing standard procedures using BioTek Gen5 Analysis software (BioTek Instruments Inc, Winooski, VT, USA). Intra-assay coefficient of variation has been shown to range from 3.7% to 5.5%, whereas the inter-assay coefficient of variation of has a ranged from 5.8%

to 6.8% (ALPCO, Salem, NH, USA). Fasting insulin was also assayed in duplicate via a commercially available Enzyme Linked Immunosorbent assay (ELISA) kit (No. 80-INSHU-E10, ALPCO, Salem, NH, USA) using a BioTek ELX-808 Ultramicroplate reader (BioTek Instruments Inc, Winooski, VT, USA) at an optical density of 450 nm against a known standard curve using standard procedures with BioTek Gen5 Analysis software (BioTek Instruments Inc, Winooski, VT, USA). The intra-assay coefficient of variation has been shown to ranged from 2.9% to 6.2%, with an inter-assay coefficient of variation range of 5.4% to 8.6% (ALPCO, Salem, NH, USA). The homeostasis Model Assessment for insulin resistance (HOMA-IR) was calculated as the product of fasting insulin times fasting glucose expressed in standard units divided by 405 [188].

Maximal Cardiopulmonary Exercise Test

Cardiopulmonary exercise tests were performed at baseline and 16 weeks by ESNL exercise physiology graduate students in accordance to standard procedures described by the American College of Sports Medicine's (ACSM) *Guidelines for Exercise Testing and Prescription* [179]. The Nasiff Cardio Card electrocardiograph (Nasiff Associates, Inc, Central Square, NY, USA) was used to assess heart function using a standard 12-lead arrangement [179]. Electrode sites were cleansed with a sterile alcohol wipe using a circular motion. Once the site was dry, electrodes were placed on the right subclavicular fossa (RA), the left subclavicular fossa (LA), the 4th intercostals space at the right sternal border (V1), the 4th intercostal space at the left sternal border (V2), equidistant between V2 and V4 (V3), the 5th intercostal space at the mid-clavicular line (V4), the 5th intercostal space at the anterior axillary line (V5), the 5th

intercostals space at the midaxillary line (V6), and the right abdomen (RL) and left abdomen (LL) line. While the subject was in a supine position, resting blood pressure, heart rate, and a 12-lead ECG were obtained. The 12-lead ECG was reviewed to ensure that no contraindications for exercise testing were present based on the ACSM guidelines [181]. The participant was then asked to stand and step onto the treadmill. A standing blood pressure, heart rate, and a 12-lead ECG was obtained and reviewed. A sterile mouthpiece, attached to a head harness, was then secured on the participant and a nose clip placed on their nose. Expired gases were collected using a ParvoMedics 2400 TrueMax Metabolic Measurement System (ParvoMedics Inc, Sandy, UT, USA). Once the participant was ready to begin the test protocol, the participant was asked to straddle the treadmill with both legs while the treadmill was turned on to a speed of 2.0 mph and at a 0% grade. The participant then carefully stepped onto the belt while still gripping the handrails with both hands. Once comfortable walking on the treadmill, they let go of the handrail and began walking freely. The participant performed the Bruce treadmill protocol [189] following the speeds and grades delineated in Table 3. The mean coefficient of variation for assessing peak Vo_2 utilizing the Bruce protocol has been shown to be 6.5% (range, 2.0-14%) [190]. Heart rate (HR), ECG tracings, and expired gases were monitored continuously throughout the exercise test. Blood pressure (BP) and ratings of perceived exertion (RPE) were obtained toward the end of each stage. The participant was encouraged to exercise to their maximum unless they experienced clinical signs that required test termination as stated by the ACSM's *Guidelines for Exercise Testing and Prescription* [181]. These signs included: a decline in systolic blood pressure > 10 mmHg

from baseline, angina, ataxia, dizziness, syncopy, cyanosis, nausea, dangerous dysrhythmias (ventricular tachycardia, supraventricular tachycardia, new atrial fibrillation, or A-V block, increasing or multi-form premature ventricular contractions), an excessive rise in systolic blood pressure over 250 mmHg or diastolic over 115 mmHg, chronotropic impairment, technical difficulties of the monitoring system, or other signs or symptoms necessitating termination of the test. Once the exercise test was complete, the participant continued an active recovery period for three minutes followed by a three minute seated recovery period.

Table 3. Bruce Protocol

Stage	Speed (MPH)	Grade (%)	Duration (minutes)	METS
Warm-up	2.0	0	2	3.3
1	1.7	10	3	4.5
2	2.5	12	3	6.5
3	3.4	14	3	9.7
4	4.2	16	3	13.5
5	5.0	18	3	17
6	5.5	20	3	20.5

Isotonic Strength Tests

Participants had their 1RM determined using an isotonic Olympic bench press (Nebula Fitness, Versailles, OH, USA) and a standard hip sled/leg press (Nebula Fitness, Versailles, OH, USA) to determine changes in maximal strength according to standardized procedures. Hand positioning on the bench press and foot and seat position on the hip sled/leg press were standardized between trails. Muscular endurance was assessed by having participants perform as many repetitions as possible at 80% of their

predetermined 1RM on the bench press and leg press using standard lifting techniques and testing criteria [191]. Test-retest reliability of performing these strength tests on resistance-trained subjects in the ESNL have yielded low mean coefficients of variation and high reliability for the bench press (1.9%, intraclass $r = 0.94$) and leg press/hip sled (0.7%, intraclass $r = 0.91$) [192].

All strength/exercise tests were conducted using standard procedures and were supervised by certified lab assistants experienced in conducting strength/anaerobic exercise testing. In order to test for upper body strength and endurance, participants performed a one repetition maximum (1 RM) test on the isotonic bench press and the Nebula Fitness (Versailles, OH, USA) Olympic Power Station (#1005). Participants performed a warm-up (2 sets of 10 repetitions at approximately 50% of anticipated 1RM) followed by progressive lifts starting at about 70% of anticipated 1RM and increasing by 5 – 10 lbs until 1 RM was reached. Once the 1RM was attained, subjects performed as many repetitions as possible with 80% of their 1 RM effort. Participants rested for 10 minutes, then performed a warm up of 8-10 repetitions at approximately 50% of anticipated maximum on the Nebula 45° Leg press. Participants performed successive lifts on the leg press starting at about 70% of anticipated 1RM and increasing by 10 – 25 lbs until 1RM was attained. Once the 1RM was achieved, subjects performed as many repetitions as possible with 80% of their 1 RM effort.

Psychometric Assessments

Participants completed the SF-36 Health-Related Quality of life (QOL) inventory [140] and an appetite/eating satisfaction questionnaire. The SF-36 quality of life

questionnaire has been validated for the measurement of psychosocial dimensions that may be influenced by general improvement in health and/or weight loss [145, 193]. The SF 36 questionnaire assessed a number of physical and mental components including physical functioning (ie, the ability to perform most vigorous physical activities without limitation to health), role physical (ie, ability to work and perform daily activities), bodily pain (ie, limitations due to pain), general health (ie, assessment of personal health), vitality (ie, feeling of having energy), social functioning (ie, ability to perform normal social activities), role emotion (ie, problems with work or other daily activities), and mental health (state of feelings of peacefulness, happiness, and calm). Eating Satisfaction Questionnaires were performed for comparison to previous curves intervention studies. These questionnaires satisfied the requirements of the sponsor of this study and are listed as a limitation of the study as they have not been validated or tested for reliability. Following the final testing session, participants were asked to complete a post-study questionnaire to assess their impressions about the weight loss study.

Statistical Methods

Only subjects who completed the 16 week trial with greater than 80% compliance were included in the analyses. Missing data, if any, were replaced using the last observed value method or by replacing missing values with the group mean method. Baseline demographic data were analyzed by one-way Analysis of Variance (ANOVA). Data were normally distributed. Study data were analyzed by Multivariate Analysis of Variance (MANOVA) with repeated measures (PASW Statistics version 19, 2011, SPSS

Inc, Chicago, IL.). Overall MANOVA effects were examined using Wilks' Lamda time and group x time p-levels as well as MANOVA univariate ANOVA group effects. Greenhouse-Geisser univariate tests of within-subjects time and group x time effects and between-subjects univariate group effects were reported for each variable analyzed within the MANOVA model. In some instances, repeated measures ANOVA was run on variables not included in a MANOVA design with univariate group, time, and group x time interaction effects reported. Variables with baseline differences determined by ANOVA were analyzed using analysis of covariance (ANCOVA). Delta values or percent difference were calculated and analyzed on select variables by ANOVA for repeated measures to assess changes from baseline values. Delta values were calculated by subtracting the first testing session (T1) from later testing sessions (T6-T1). Percent differences were calculated by subtracting T1 from the later testing session, then performing division by T1 followed by multiplication by 100 [(T6-T1)/T1·100]. Data were considered statistically significant when the probability of type I error was 0.05 or less and statistical trends were considered when the probability error ranged between >0.05 to $p<0.10$. If a significant group, treatment and/or interaction alpha level was observed, Tukey's least significant difference (LSD) post hoc analyses were performed to determine where significance was obtained. Power analysis of previous studies using similar designs and subject populations indicated that a sample size of 30 subjects per group yielded high power (>0.8) for delta values of 0.75 to 1.25 for weight and fat loss. All data are presented as means ± standard deviation.

CHAPTER IV

RESULTS

Methods

Experimental Approach

This study was conducted as a randomized comparative effectiveness trial from June 2009 to June 2011. Participants were matched based on age and body mass index (BMI: calculated as kg/m^2) and randomized into one of two weight reduction interventions. Dietary intake and weekly physical activity, anthropometric measurements, resting energy expenditure (REE), resting heart rate (RHR) and systolic and diastolic blood pressure (BP), body composition, serum clinical chemistry panels, whole blood counts and hormone concentrations, and quality of life were assessed at 0, 4, 10, and 16 weeks to determine differences in weight reduction program effects. Maximal cardiopulmonary exercise capacity (Peak VO_2) and upper and lower body isotonic strength and endurance were assessed at 0 and 16 weeks to ascertain chronic program effects.

Participants

This research protocol was reviewed and approved by the university Institutional Review Board before initiation. Participants were recruited through advertisements in local newspapers, campus flyers, radio and Internet advertisements. Interested participants were asked to contact the laboratory for an initial telephone prescreening interview. General entrance criteria included being an apparently healthy woman between ages of 18 and 50 years with a BMI greater than 25 and no recent participation in a diet or

exercise program. Individuals who met initial entrance criteria were invited to attend a familiarization session in which the details of the study were explained, human subject consent forms were signed and personal and medical history information was obtained. Participants were not allowed to participate in the study if the subjects reported the following at baseline: a recent weight change of (±3.2 kg or 7 lb) within 3 months; any uncontrolled metabolic or cardiovascular disorder, including known electrolyte abnormalities, heart disease, arrhythmias, diabetes, or thyroid disease, or a history of hypertension, hepatorenal, musculoskeletal, autoimmune, or neurological disease; taking any weight loss supplements and/or ergogenic levels of nutritional supplements that affect muscle mass, anaerobic exercise capacity, anabolic/catabolic hormone levels, or weight loss within 3 months; a history of pregnancy or lactation within the past 12 months or intentions to become pregnant during the next 12 months; participation in a regular exercise program within the past 3 months; and, any condition that is classified as high risk for cardiovascular disease according to American College of Sports Medicine criteria [194]. Information obtained during the familiarization session was reviewed by the research coordinator to determine eligibility to participate in the study. Those meeting eligibility criteria were scheduled to undergo baseline assessments. Participants with a controlled medical condition had their physicians complete and sign the physician's consent form prior to participation in the baseline assessment.

A total of 149 women responded to advertisements to participate in this study. Of these, 127 met phone interview entrance criteria and were invited to attend familiarizations sessions. One hundred and twenty women showed up for familiarization

sessions and met entrance criteria to participate in the study after evaluation of medical history and obtaining consent. Of these women, 98 women completed baseline testing and were cleared to participate in the study. Fifty one women completed the 16 week study. The primary reasons participants dropped out of the study were due to time constraints, job conflicts, transportation difficulties, and relocation.

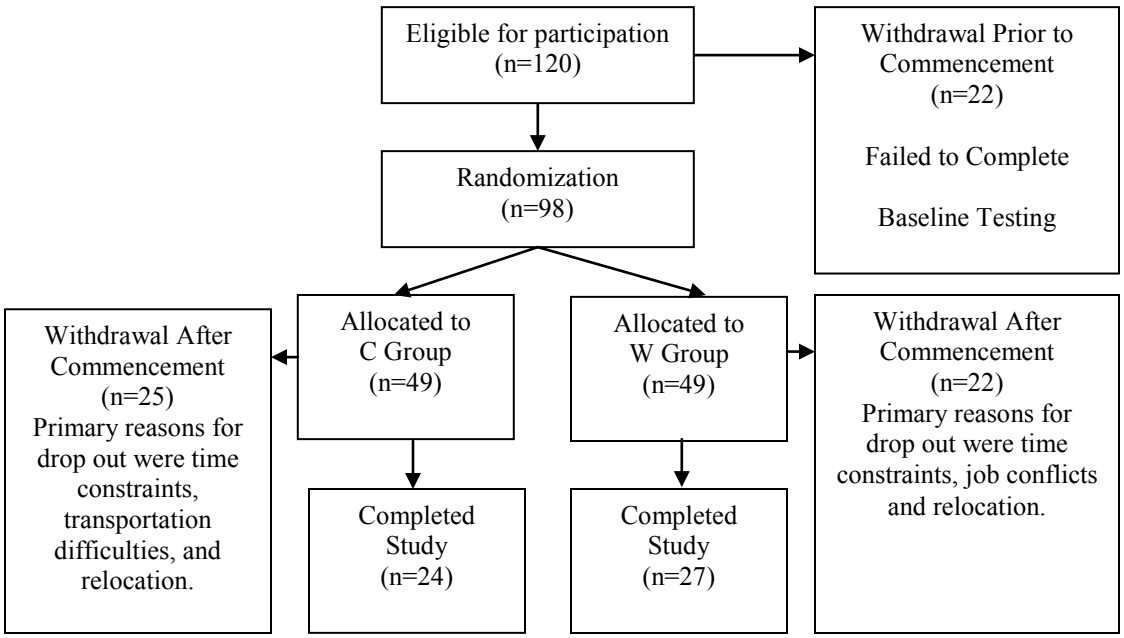

Figure 1. Consort diagram for participation.

Testing Sessions

Participants were instructed to refrain from exercise for 48 hours and fast for at least 12 hours prior to reporting to the ESNL for testing sessions. Baseline testing sessions and 16 week testing sessions were identical and consisted of dietary inventory review; weekly physical activity assessment [International Physical Activity Questionnaire

(IPAQ)]; anthropometric assessments (body mass, waist and hip circumference); resting energy expenditure; resting heart rate and systolic and diastolic blood pressure (BP); body composition analysis (DEXA); blood collection (metabolic panels, blood lipids, white and red blood cells); cardiorespiratory and muscular fitness assessments; psychosocial assessments [standardized quality of life (SF-36) and eating satisfaction inventory]. Additional testing sessions occurred during the fourth and tenth weeks and consisted of all baseline measures excluding cardiorespiratory and muscular fitness assessments.

Program Intervention

Participants were randomized into one of two popular weight loss program intervention groups. Participants in the Curves group (C) followed the Curves Fitness and Weight Management Plan (C program). In concurrence with this plan, subjects followed the phase 1 higher protein weight loss diet (1,200 kcals/day) for one week and the phase 2 higher protein weight loss diet (1,500 kcals/day) for 3 weeks. The macronutrient content for both phase 1 and 2 diets was (45% protein, 30% carbohydrate, 25% fat). Phase 3 consisted of a metabolic recovery phase, where subjects followed a 2,000-2,500 kcals/d diet with a macronutrient breakdown of (30% protein, 45% carbohydrate, 25% fat) for two weeks. Subjects experiencing a weight increase of 3 pounds during phase 3 were instructed to return to Phase 1 for 2-3 days until their weight returned back down. The three dietary phases were cycled over the course of 16 weeks. A registered dietitian reviewed the higher protein diet and exercise plan with subjects. Participants were encouraged to consume 2 multivitamins, 800 mg of calcium, and 520 mg of Omega-3

fatty acids daily. The dietitian also met with subjects weekly throughout the study to discuss any dietary challenges or concerns.

The physical training protocol included one regular curves circuit work out and two circuit combined with Zumba workouts each week for 16 weeks, while maintaining a greater than 80% compliance record (38 out of 48 workouts). The circuit utilized the computerized CurvesSmart system (curves International, Waco, TX, USA) equipped with software designed by MYTRAK (version 4.2.0.0, copyright 2004-2010, MYTRAK Health System, Mississauga, Ontario, Canada). The circuit consisted of 13 bi-directional hydraulic resistance exercise machines that worked all major muscle groups (ie, elbow flexion/extension, knee flexion/extension, shoulder press/lat pull, hip abductor/adductor, chest press/seated row, horizontal leg press, squat, abdominal crunch/back extension, chest flies, oblique, shoulder shrug/dip, hip extension, and side bends). During the circuit workouts, subjects were instructed to complete as many repetitions as possible during a 30 second time period on each resistance machine. Between machines, subjects performed floor-based aerobic exercises or stepping exercise designed to maintain an elevated heart rate. Subjects performed the entire circuit twice during the 26 minute regular circuit workout. During the circuit combined with Zumba workout, subjects performed 1 minute of Zumba dance moves in between 1 minute of resistance exercise on each machine. All Zumba classes were taught by a certified Zumba instructor. All circuit workouts were supervised by trained fitness instructors who assisted subjects with proper exercise technique and self-monitoring of heart rate in order to maintain an exercise heart rate between 60-80% of target heart rate using age-predicted maximal

heart rate (220-age). Subjects also performed 5 minutes of whole body stretching following circuit workouts. In addition to circuit training, subjects were encouraged to walk for 30 minutes at a brisk pace [60-80% of heart rate reserve (maximum HR-minimum HR)] on most days of the week. The circuit equipment was located in the Exercise and Sports Nutrition Laboratory at Texas A&M University.

Participants assigned to the Weight Watchers Group (W) took part in the Weight Watchers Momentum Program (W program) and were signed up for the monthly pass subscription. Subjects were required to attend 1 meeting each week with an 80% compliance record (13 out of 16 meetings). Subjects emailed the study coordinator each time they attend a meeting. Meetings consisted of group discussions and some individual interaction as members weighed in. The core program material was delivered over ten weeks and included: food plans based on a system of points, exercise recommendations, strategies for dealing with hunger, successful habits, strategies for eating out, tracking intake and activity, eating with others, recipes, workout variation, and plateaus. The program also encouraged subjects to perform 30 minutes of activity on most days of the week. All subjects attended meetings at the Bryan Texas site.

Methods and Procedures

Dietary Inventories

A registered dietitian instructed all subjects on precise documentation of food intake and accurate estimation of food portion size. Participants recorded all food and fluids consumed over a four day period (including one weekend day) prior to each testing session. Dietary inventories were reviewed with participants at each testing session to

ensure accuracy, completeness, and legibility. Dietary information was then analyzed to determine the average caloric intake and macronutrient content using Food Processor Nutrition Analysis Software Version 9.1.0 (ESHA Nutrition Research, Salem, OR, USA). A registered dietitian review all analyzed dietary information.

Weekly Physical Activity Assessment

Physical activity patterns were quantified by assessing responses to the 7 day version of the International Physical Activity Questionnaire (IPAQ) [175-177]. This assessment tool evaluated the frequency and intensity of job-related physical activity; transportation physical activity; housework, house maintenance, and caring for family related physical activity; and recreation, sports, and leisure-time physical activity based on established metabolic equivalent (MET) levels for common activities. The IPAQ defined light physical activity as walking level intensities (3.3 METs), moderate physical activity as activities at a 4.0 MET level, and vigorous physical activity as activities at an 8.0 MET level. The IPAQ has been identified as a valid indicator of general changes in physical activity patterns [175-178].

Anthropometrics and Body Composition

Height and total body weight were determined according to standard procedures using a Healthometer (Bridgeview, IL, USA) self-calibrating digital scale with a precision of +/- 0.02 kg. Hip and waist circumference were measured using a Gulick tension standardized measuring tape per guidelines established by the American College of Sports Medicine [181]. Bone density and body composition (excluding cranium) was assessed using a Hologic Discovery W QDR series Dual Energy X-ray Absorptiometry

(DEXA) system (Hologic Inc, Waltham, MA, USA) equipped with APEX software (APEX Corporation Software, Pittsburgh, PA, USA). Mean coefficients of variation for bone mineral content and bone mineral density measurements performed on the spine phantom ranged between 0.41% and 0.55%. Mean test-retest reliability studies performed on male athletes with this Hologic system have yielded mean coefficients of variation for total bone mineral content and total fat free/soft tissue mass of 0.31% to 0.45% with a mean intra-class correlation of 0.985 [187].

Resting Energy Expenditure

Resting energy expenditure assessments were conducted according to standard protocols using the Parvo Medics TrueMax 2400 Metabolic Measurement System (ParvoMedics, Inc, Sandy, UT, USA). This test was conducted in a fasted state with the participants lying supine on an exam table. A clear metabolic canopy was placed over the subject's head and neck, so that resting oxygen uptake and energy expenditure could be determined. The participants remained motionless without falling asleep for approximately 20 minutes. Metabolic measurements were recorded after the first 10 minutes during a five minute period in which principle variables, such as oxygen uptake, changed less than 5% [180]. Mean test-retest reliability studies on 14 participants from a previous study revealed test-retest correlations (r) of collected oxygen uptake range from 0.315 to 0.901 (mean 0.638) and coefficient of variation range from 8.2% to 12.0% (mean 9.9%) with a mean intraclass coefficient of 0.942; $p<0.001$ [35].

Blood Collection and Analysis Procedures

Fasted and whole blood and serum samples were collected using standard phlebotomy techniques. Whole blood samples were analyzed for complete blood counts with platelet differentials utilizing an Abbott Cell Dyn 3500 automated hematology analyzer (Abbott Laboratories, Abbott Park, IL, USA). Serum samples were analyzed for a complete metabolic panel by Quest Diagnostics (Quest Diagnostics, 5850 Rogerdale Road, Houston TX, USA 77072) using an Olympus AAU 5400 Chemistry Immuno Analyzer (Olympus America Inc., Center Valley, PA, USA). Serum leptin was determined in duplicate using a commercially available enzyme linked immunoabsorbent assay (ELISA) kit (No. 11-LEPHU-E01, ALPCO, Salem, NH) using a BioTek ELX-808 Ultramicroplate reader (BioTek Instruments Inc, Winooski, VT) at an optical density of 450 nm against a known standard curve utilizing standard procedures using BioTek Gen5 Analysis software (BioTek Instruments Inc, Winooski, VT). Intra-assay coefficient of variation has been shown to range from 3.7% to 5.5, whereas the inter-assay coefficient of variation of has a ranged from 5.8% to 6.8% (ALPCO, Salem, NH). Fasting insulin was also assayed in duplicate via a commercially available Enzyme Linked Immunosorbent assay (ELISA) kit (No. 80-INSHU-E10, ALPCO, Salem, NH) using a BioTek ELX-808 Ultramicroplate reader (BioTek Instruments Inc, Winooski, Vermont) at an optical density of 450 nm against a known standard curve using standard procedures with BioTek Gen5 Analysis software (BioTek Instruments Inc, Winooski, VT). The intra-assay coefficient of variation has been shown to ranged from 2.9% to 6.2%, with an inter-assay coefficient of variation range of 5.4% to 8.6% (ALPCO,

Salem, NH). The homeostasis Model Assessment for insulin resistance (HOMA-IR) was calculated as the product of fasting insulin times fasting glucose expressed in standard units divided by 405 [188].

Exercise Capacity

Resting heart rate was determined by palpitation of the radial artery and resting blood pressure was assessed in the supine position utilizing a mercurial sphygmomanometer (American Diagnostic Corporation, model #AD-720, Hauppuage, NY, USA) using standard procedures [181]. Maximal Cardiopulmonary exercise tests (peak VO_2) were performed utilizing the Bruce treadmill protocol [189]. Standard test termination criteria were utilized to assess maximal volitional fatigue [181]. The Nasiff Cardio Card electrocardiograph (Nasiff Associates, Inc, Central Square, NY, USA) was used to assess heart function using a standard 12-lead arrangement [181]. Expired gases were collected using a ParvoMedics 2400 TrueMax Metabolic Measurement System (ParvoMedics Inc, Sandy, UT, USA). Gas and flow sensors were calibrated before testing and were found to be within 3% of previous calibration points.

Participants had their 1RM determined using an isotonic Olympic bench press (Nebula Fitness, Versailles, OH, USA) and a standard hip sled/leg press (Nebula Fitness, Versailles, OH, USA) to determine changes in maximal strength. Hand positioning on the bench press and foot and seat position on the hip sled/leg press were standardized between trails. Muscular endurance was assessed by having participants performed as many repetitions as possible at 80% of their predetermined 1RM on the bench press and leg press using standard lifting techniques and testing criteria [191]. Test-retest reliability

of performing these strength tests on resistance-trained subjects in the ESNL have yielded low mean coefficients of variation and high reliability for the bench press (1.9%, intraclass $r = 0.94$) and leg press/hip sled (0.7%, intraclass $r = 0.91$) [192].

Psychometric Assessments

Participants completed the SF-36 Health-Related Quality of life (QOL) inventory [140] and an appetite/eating satisfaction questionnaire. The SF-36 quality of life questionnaire has been validated for the measurement of psychosocial dimensions that may be influenced by general improvement in health and/or weight loss [145, 193]. The SF 36 questionnaire assessed a number of physical and mental components including physical functioning (ie, the ability to perform most vigorous physical activities without limitation to health), role physical (ie, ability to work and perform daily activities), bodily pain (ie, limitations due to pain), general health (ie, assessment of personal health), vitality (ie, feeling of having energy), social functioning (ie, ability to perform normal social activities), role emotion (ie, problems with work or other daily activities), and mental health (state of feelings of peacefulness, happiness, and calm). Eating Satisfaction Questionnaires were performed for comparison to previous curves intervention studies. These questionnaires satisfied the requirements of the sponsor of this study and are listed as a limitation of the study as they have not been validated or tested for reliability. Following the final testing session, participants were asked to complete a post-study questionnaire to assess their impressions about the weight loss study.

Statistical Methods

Only subjects who completed the 16 week trial were included in the analyses. Missing data, if any, were replaced using the last observed value method or by replacing missing values with the group mean method. Baseline demographic data were analyzed by one-way Analysis of Variance (ANOVA). Data were normally distributed. Study data were analyzed by Multivariate Analysis of Variance (MANOVA) with repeated measures (PASW Statistics version 19, 2011, SPSS Inc, Chicago, IL.). Overall MANOVA effects were examined using Wilks' Lamda time and group x time p-levels as well as MANOVA univariate ANOVA group effects. Greenhouse-Geisser univariate tests of within of within-subjects time and group x time effects and between-subjects univariate group effects were reported for each variable analyzed within the MANOVA model. In some instances, repeated measures ANOVA was run on variables not included in a MANOVA design with univariate group, time, and group x time interaction effects reported. Variables with baseline differences determined by ANOVA were analyzed using analysis of covariance (ANCOVA). Delta values or percent difference were calculated and analyzed on select variables by ANOVA for repeated measures to assess changes from baseline values. Delta values were calculated by subtracting the first testing session (T1) from later testing sessions (T6-T1). Percent differences were calculated by subtracting T1 from the later testing session, then performing division by T1 followed by multiplication by 100 [(T6-T1)/T1·100]. Data were considered statistically significant when the probability of type I error was 0.05 or less and statistical trends were considered when the probability error ranged between >0.05 to

$p<0.10$. If a significant group, treatment and/or interaction alpha level was observed, Tukey's least significant difference (LSD) post hoc analyses were performed to determine where significance was obtained. Power analysis of previous studies using similar designs and subject populations indicated that a sample size of 30 subjects per group yielded high power (>0.8) for delta values of 0.75 to 1.25 for weight and fat loss. All data are presented as means ± standard deviation.

Results

Fifty-one apparently healthy but sedentary and obese women (age 35±8 yrs, height 163±7 cm; weight 90±1 kg; BMI 34±5 kg/m^2; 47±7 % body fat) completed the 16-week study (n=24 C group; n=27 W group) with greater than 80% compliance. No significant differences were observed between groups in baseline age, height, weight, BMI, or percent body fat as determined by ANOVA (Table 4).

Table 4. Baseline demographics for the Curves (C) and Weight Watchers (W) groups

	Group (n-51)	C group (n=24)	W group (n=27)	p-value
Age (years)	34.5±7.7	35.1±9.2	34.0±6.2	0.63
Height (cm)	162.8±7.0	163.0±7.4	162.6±6.7	0.84
Weight (kg)	90.1±14.5	90.8±15.7	89.5±13.6	0.77
BMI[a] (kg/m²)	34.0±5.1	34.1±5.1	33.9±5.2	0.90
DEXA[b] fat mass (%)	46.5±7.1	48.1±6.5	45.1±7.3	0.14

All data is presented as means ± SD at baseline. Significance level was set at 0.05.
[a]Body mass Index
[a]Dual Energy X-ray Absorptiometry

Dietary Intake

Table 5 presents changes in nutritional intake at 0, 4, 10, and 16 weeks of program intervention. Complete food records were obtained on all participants completing the

study. MANOVA analyses were run on energy intake (kcals/kg/d), macronutrient intake expressed in g/kg/day, and macronutrient intake expressed as a percentage of total calories consumed. MANOVA analysis of dietary intake data revealed an overall time

Table 5. Changes in nutritional intake obtained at 0, 4, 10 and 16 weeks of program participation for the Curves (C) and Weight Watchers (W) groups[a]

		Week				
Variable	Group	0	4	10	16	p-value
		← Mean±Standard Deviation →				
Energy Intake (kcals/kg/d)	C	19.17±5.99	16.53±4.23[b]	15.83±4.04[b]	17.24±4.54[b]	G[c] = 0.34
	W	22.32±6.80	15.58±4.52[b]	16.27±4.12[b]	15.67±4.22[b]	T[d] = 0.001
	Total	20.84±6.55	16.03±4.37[b]	16.06±4.05[b]	16.50±4.42[b]	I[e] = 0.04
CHO Intake[f] (g/kg/d)	C	1.94±0.89	1.58±0.66[b]	1.54±0.62[b]	1.51±0.68[b]	G = 0.12
	W	2.26±0.88	1.70±0.53[b]	1.69±0.55[b]	1.89±0.50[b]	T = 0.001
	Total	2.11±0.89	1.64±0.59[b]	1.62±0.58[b]	1.71±0.61[b]	I = 0.43
Protein Intake (g/kg/d)	C	0.89±0.27	1.07±0.28[gb]	0.94±0.31	1.00±0.35[g]	G = 0.02
	W	0.86±0.23	0.79±0.24	0.85±0.25	0.80±0.22	T = 0.44
	Total	0.88±0.25	0.93±0.29[b]	0.89±0.28	0.90±0.30	I= 0.005
Fat Intake (g/kg/d)	C	0.74±0.27	0.62±0.18[gb]	0.58±0.19[b]	0.59±0.25[b]	G = 0.64
	W	0.90±0.32	0.47±0.20[b]	0.48±0.19[b]	0.58±0.18[b]	T = 0.001
	Total	0.82±0.31	0.54±0.20[b]	0.53±0.20[b]	0.58±0.22[b]	I= 0.001
CHO Intake (%)	C	42±9	38±7[b]	41±11	40±10	G = 0. 003
	W	43±9	47±9	46±9	47±6	T = 0.90
	Mean	42±9	43±10	43±10	43±9	I = 0.06
Protein Intake (%)	C	21±5[g]	27±6[gb]	25±7	27±7[g]	G = 0.006
	W	17±3	22±4	23±5	20±3	T =0.75
	Mean	19±5	24±6	24±6	23±6	I= 0.02
Fat Intake (%)	C	37±8	35±6[h]	34±7[h]	33±6[b]	G = 0.11
	W	39±8	30±8[b]	30±9[b]	32±6[b]	T = 0.001
	Mean	38±8	32±8[b]	32±8[b]	33±6[b]	I = 0.02

[a]Complete food intake records were obtained at 0, 4, 10, and 16 weeks for 24 participants in the C group and 27 participants in the W group (n=51). Data were analyzed using ESHA Food Processor Nutritional Analysis Software (Version 8.6).
[b]p<0.05 difference from baseline.
[c]G=group alpha level.
[d]T=time alpha level.
[e]I=group x time interaction alpha level.
[f]Carbohydrate intake
[g]p<0.01 difference between C and W groups.
[h]p<0.05 difference between C and W groups.

(Wilks' Lamda p=0.001) and overall time by diet interaction (Wilks' Lamda p=0.03) observed. MANOVA univariate analysis revealed the W group had a greater reduction in energy intake per kilogram body weight than the C group (C -2.6±5.4, -3.3±5.1, -3.5±6.0; W -6.7±5.8, -6.0±6.2, -5.1±6.2 g/kg/d) with time (p=0.001) and time by diet (p=0.04) effects observed. There was a greater reduction in fat intake in the W group (C -0.12±15, -0.16±0.31, -0.17±0.26; W -0.42±0.28,-0.41±0.33, -0.31± 0.34 g/kg/d) with time (p=0.001), time by diet (p=0.001) effects observed. Univariate ANOVA also revealed the W group had a greater reduction in the percentage of energy intake from fat (C -2.6±8.6, -3.0±8.9, -3.7±7.4; W -9.6±9.5, -9.4±11.2, -6.7±8.8 %) with effects seen for time (p=0.001) and time by diet (p=0.02).

Carbohydrate intake decreased over time in both groups (C -0.36±0.68, -0.40±0.61, -0.43±0.73; W -0.56±0.94, -0.57±0.87, -0.37±0.88 g/kg/d) with no significant difference between groups (p=0.43). However, the percentage of kcalories consumed as carbohydrate tended to increase with reduced energy intake in the W group (C -3.5±8.2, -1.0±10.2, -2.0±9.0; W 4.0±11.0, 2.7±12.1, 3.5±9.1 %) with a trend in time by diet (p=0.06) and diet (p=0.003) effects observed. The C group increased dietary protein intake (C 0.18±0.20, 0.05±0.30, 0.11±0.35; W -0.07±0.18, -0.01±0.26, -0.06±0.20 g/kg/d) with both time by diet (p=0.005) and diet (p=0.02) effects observed. One way ANOVA revealed the C group consumed a higher percentage of kcalories as protein at baseline (p=0.005). Univariate ANCOVA revealed the C group experienced a greater increase in the percentage of kcalories consumed as protein (C 6.4±7.1, 4.1±6.4, 6.1±7.7;

W 5.0±5.4, 6.2±6.5, 3.1±4.4 %) with significant time by diet (p=0.02) and group effects (p=0.006) observed.

Physical Activity

Table 6. Changes in physical activity patterns values observed at 0, 4, 10 and 16 weeks of program participation for the Curves (C) and Weight Watchers (W) groups[a]

Variable	Group	Week 0	Week 4	Week 10	Week 16	p-value
		← mean±standard deviation →				
Low PA[b]	C	1,097±1,301	1,240±1,222	1,146±1,637	1,370±1,722	G[e] = 0.80
(MET-min/week[c])	W	703±1,170	890±1,258	1,467±2,022[d]	1,428±1,868[d]	T[f] = 0.09
	Mean	889±1,237	1,054±1,241	1,316±1,840[d]	1401±1,783[d]	I[g]= 0.27
Moderate PA	C	1,295±1,606	1,081±876	1,625±1,691	1,446±1,787	G = 0.85
(MET-min/week)	W	1,037±1,372	1,432±2,406	1,427±1,839	1,205±1,544	T = 0.49
	Mean	1,158±1,477	1,267±1,842	1,573±1,754	1,318±1,650	I= 0.65
Vigorous PA	C	437±791	1,033±1,208[d]	1,281±937[d]	928±460[d]	G =0.002
(MET-min/week)	W	137±313	598±1,008[d]	637±943[d]	341±670	T=0.001
	Mean	278±602	803±1,117[d]	940±986[d]	617±647[d]	I = 0.61
Total PA	C	2,341±1,950	3,334±2,228[d]	4,053±2,671[d]	3,805±2,764[d]	G = 0.37
(MET-min/week)	W	1,825±2,028	2,879±3,228	3,703±3,687[d]	2,970±3,055	T = 0.001
	Mean	2,068±1,989	3,093±2,785[d]	3,868±3,222[d]	3,363±2,923[d]	I= 0.93
Job PA	C	1,230±1,713	1,157±1,999	1,465±1,936	1,526±2,334	G = 0.18
(MET-min/week)	W	460±1,132	751±1,332	1,341±2,504	640±1,443	T = 0.19
	Mean	822±1,472	942±1,674	1,400±2,233	1,057±1,946	I= 0.45
Transportation PA	C	274±389	311±450	419±636	523±1,003	G = 0.44
(MET-min/week)	W	129±294	183±545	477±878	820±1,528[d]	T = 0.05
	Mean	197±346	243±501	450±767[d]	680±1,303[d]	I = 0.59
House PA	C	594±882	627±532	1,133±1,500	670±601	G = 0.36
(MET-min/week)	W	876±1,251	1,262±2,279	873±1,124	950±1,388	T_q = 0.56
	Mean	743±1,092	963±1,712	996±1,308	818±1,090	I = 0.26
Recreation PA	C	439±577	1,259±593[d]	1,036±275[d]	995±492[d]	G = 0.01
(MET-min/week)	W	340±549	746±958	868±1,321[d]	421±481	T = 0.001
	Mean	387±559	987±840[d]	947±974[d]	691±561[d]	I = 0.20

[a]International Physical Activity Questionnaire (IPAQ) data were analyzed at 0, 4, 10, and 16 weeks of program participation for 24 participants in the C group and 27 participants in the W group (n=51).
[b]PA=physical activity.
[c]Met-min/week=MET level x minutes of activity per day x days per week.
[d]p<0.05 difference from baseline.
[e]G=group alpha level.
[f]T=time alpha level.
[g]I=group x time interaction alpha level.

Table 6 shows changes in physical activity observed at 0, 4, 10, and 16 weeks of program intervention. Overall MANOVA revealed a significant time effects (Wilks' Lamda p=0.001) with a trend in overall time by diet interaction observed (Wilks' Lamda p=0.08) in physical activity variables. Both groups increased total recreational activity (C 818±852, 597±549, 556±680; W 407±1052, 528±1082, 82±707 MET-min/week; p=0.20) with an effect of group observed (p=0.01) indicating total recreational activity was greater in the C group. Vigorous physical activity also increased in both groups (C 595±666, 845±1059, 491±943; W 462±1026, 500±1028, 204±763 MET-min/week, p=0.61) with the C group increasing vigorous activity to a greater degree than the W group (p=0.002). Both groups increased total physical activity (C 993±2148, 1712±2275, 1464±1775; W 1054±3519, 1878±2689, 1145±3215 MET-min/week; p=0.93) with no differences observed between groups. The W group increased low physical activity (C 143±1075, 49±1001, 273±1139; W 187±1501, 764±1454, 725±1806 MET-min/week) and transportation physical activity (C 37±278, 145±530, 249±948; W 54±558, 349±893, 691±1562 MET-min/week) with no significant difference observed between groups (p=0.27) and (p=0.33) respectively. Large standard deviations revealed considerable variability in the physical activity levels of study participants.

Body Composition, Anthropometry and Resting Energy Expenditure

Table 7 presents changes in body composition, anthropometry, and resting energy expenditure data observed at 0, 4, 10 and 16 weeks of program participation. MANOVA analysis of body composition data revealed overall time (Wilks' Lamda p=0.001) and time by diet effects (Wilks' Lamda p=0.003). MANOVA Univariate

analysis revealed that both groups lost a similar amount of total mass (C -2.4±2.0, -4.1±3.4, -5.1±3.9; W -2.3±2.3, -4.5±3.0, -5.5±4.6 kg, p=0.78). Lean tissue mass was

Table 7. Changes in body composition, resting energy expenditure, and fitness related data values observed at 0, 4, 10 and 16 weeks of program participation for the Curves (C) and Weight Watchers (W) groups[a]

		Week				
Variable	Group	0	4	10	16	p-value
		← mean±standard deviation →				
Total Mass (kg)	C	84.5±15.0	82.1±13.8[b]	80.4±13.0[b]	79.4±13.0[b]	G[c] =0.62
	W	82.8±12.7	80.5±13.2[b]	78.3±13.4[b]	77.3±14.1[b]	T[d]=0.001
	Mean	83.6±13.7	81.3±13.4[b]	79.3±13.1[b]	78.3±13.5[b]	I[e]= 0.78
Fat Mass (kg)	C	40.9±10.5	37.0±8.1[b]	36.2±8.4[b]	34.5±8.5[b]	G = 0.67
	W	37.4±9.1	37.0±8.7	35.3±10.5	34.5±10.7[b]	T =0.001
	Mean	39.1±9.9	37.0±8.4[b]	35.8±9.4[b]	34.5±9.6[b]	I = 0.09
Lean Mass (kg)	C	43.6±7.8	45.1±7.6	44.1±7.0	44.9±6.7	G =0.68
	W	45.3±8.7	43.5±7.2	42.9±6.5[b]	42.8±6.2[b]	T = 0.32
	Mean	44.5±8.2	44.3±7.4	43.5±6.7	43.8±6.5	I = 0.01
Body Fat (%)	C	48.1±6.5	44.8±4.7[b]	44.8±5.6[b]	43.4±5.4[b]	G = 0.74
	W	45.1±7.3	45.7±5.6	44.5±7.3	43.7±6.7	T= 0.007
	Mean	46.5±7.1	45.3±5.2	44.7±6.5	43.5±6.1[b]	I= 0.10
BMI[f] (kg/m^2)	C	34.1±5.1	33.2±4.8[b]	32.5±4.6[b]	32.2±4.5[b]	G = 0.75
	W	33.9±5.1	32.9±5.2[b]	31.9±5.2[b]	31.6±5.5[b]	T= 0.007
	Mean	34.0±5.1	33.0±5.0[b]	32.3±4.5[b]	31.9±5.0[b]	I = 0.24
Waist (cm)	C	96.7±12.3	92.9±11.4[b]	90.3±10.1[b]	89.6±10.2[b]	G = 0.64
	W	93.2±8.6	92.3±11.3[b]	89.8±10.6[b]	87.9±10.7[b]	T= 0.001
	Mean	94.4±10.5	92.6±11.2[b]	89.9±10.5[b]	88.7±10.4[b]	I = 0.46
Hip (cm)	C	120.8±10.2	118.4±10.3[b]	115.4±9.3[b]	114.7±9.3[b]	G = 0.98
	W	120.5±10.9	118.9±10.8[b]	115.7±11.1[b]	113.3±12.0[b]	T =0.001
	Mean	120.6±10.4	118.7±104[b]	115.6±10.2[b]	114.0±10.7[b]	I = 0.31
REE (kcals/d)	C	1,389.1±252.5	1,465.5±191.9	1,507.7±195.6[b]	1,507.1±223.2[b]	G = 0.34
	W	1,361.3±257.6	1,415.3±200.4	1,425.9±190.2[b]	1,418.4±244.2[b]	T =0.002
	Mean	1,374.4±253.0	1,438.9±196.1[b]	1,464.4±195.2[b]	1,460.1±236.5[b]	I = 0.22

[a]Data are from 24 participants in the C group and 27 participants in the W group (n=51) who completed the 16-week study.
[b]$p<0.05$ difference from baseline.
[c]G=group alpha level.
[d]T=time alpha level.
[e]I=group x time interaction alpha level.
[f]BMI = body mass index.
[g]Resting Energy Expenditure

maintained in the C group and decreased in the W group (C 1.5±4.3, 0.52±3.7, 1.3±4.0; W -1.8±5.4, -2.4±5.8, -2.5±5.1, p=0.01). The C group tended to have a greater reduction in percent body fat (C -3.3±5.2, -3.2±4.6, -4.7±5.4; W 0.6±6.7, -0.6±8.3, -1.4±8.1 %, p=0.10) and fat mass (C -3.9±5.5, -4.6±5.3, -6.4±5.9; W -0.4±5.7, -2.1±6.7, -2.9±7.8, p=0.09). Review of body composition data lead to acceptance H1 and a partial rejection of H01.

MANOVA analysis of anthropometry data revealed an overall time effect (Wilks' Lamda p=0.001) effect with no significant overall time by diet effect (p=0.18). After 16 weeks both groups had decreased BMI (C -0.9±0.8, -1.6±1.3, -1.8±1.4; W -1.0±0.5, -2.0±0.9, -2.3±1.5 kg/m^2; p=0.24), waist circumference(C -2.8±3.7, -5.4±5.4, -6.2±5.1; W -0.9±5.7, -3.8±6.0, -5.4±5.2 cm; p=0.46) and hip circumference (C -2.1±2.6, -5.0±4.2, -5.8±5.0; W -1.8±4.0, -5.1±3.7, -7.4±4.9 cm; p=0.31), with no differences observed. Univariate ANOVA revealed both groups had an increase in resting energy expenditure (REE) (C 76.4±195.7, 118.6±185.5, 118.0±162.5; W 53.9±187.9, 64.6±188.9, 57.0±216.6 kcals/d, p=0.54) with no difference observed between groups and lead to a rejection of H2.

Resting Heart Rate and Blood Pressure

Table 8 presents resting heart rate (RHR), resting systolic blood pressure (BP), and resting diastolic blood pressure observed at 0, 4, 10 and 16 weeks and changes in cardiorespiratory and muscular fitness after 16 weeks of program participation. MANOVA analysis of RHR and BP data revealed an overall time effect (Wilks' Lambda p=0.001) effect with no overall time by diet interaction effect (Wilks' Lambd p=0.22).

The C group experienced reductions in RHR (C -4.6±9.3, -5.9±10.7, -1.0±8.8; W -5.2±12.4, -1.0±12.1, -2.0±14.1 bpm, p=0.17) with no differences observed between groups. Both groups experienced similar changes in resting systolic BP (C -3.3±7.8, -3.8±11.1, -5.0±11.2; W -5.5±10.1, -4.9±11.9, -5.3±8.9 mmHg, p=0.86) and resting diastolic BP (C -4.1±8.0, -1.8±10.1, -1.5±10.1; W -4.3±8.8, -5.4±9.4, -4.7±7.9 mmHg, p=0.32) with no differences observed between groups. Review of resting cardiovascular data lead to an acceptance of H02.

Cardiorespiratory and Muscular Endurance and Fitness

MANOVA analysis of peak VO_2 and treadmill time revealed an overall time effect (p=0.001) with no overall time by diet (p=0.12) effect observed. Univariate ANOVA analysis revealed both groups experienced an increase in peak oxygen uptake in L/min (C 0.09±0.16; W 0.03±0.13 L/min, p=0.13), peak oxygen uptake in ml/kg/min (C 2.4±2.3; W 2.1±2.0 ml/kg/min, p=0.68) and treadmill time (C 0.61±0.76; W 0.72±1.22 min, p=0.71) with no differences observed between groups after 16 weeks of program participation. Consideration of peak $V0_2$ and treadmill time data lead to a rejection of H3.

MANOVA analysis of muscular fitness and endurance changes from 0 to 16 weeks revealed an overall time x diet effect (Wilks' Lambda p=0.01) with no overall time effect (Wilks' Lambda p=0.99). No significant changes were observed in either the C group or W group in leg press 1 RM (C 8.4±29.6; W -4.6±30.3 kg, p=0.13), bench press 1RM (C 1.1±4.7; W -0.7±3.8 kg; p=0.13), or leg press lifting volume at 80% 1 RM (C 131.3±925.3; W -56.1±1119.5 reps*kg; p=0.52). One way ANOVA revealed the C

Table 8. Changes in general health and fitness values obtained at 0, 4, 10 and 16 weeks of program participation for the Curves (C) and Weight Watchers (W) groups[a]

Variable	Group	Week 0	4	10	16	p-value
		mean±standard deviation				
Resting Heart Rate (bpm)	C	72.5±8.5	67.9±8.4[b]	66.6±9.2[b]	71.5±8.5	G[c] = 0.27
	W	70.0±9.1	65.8±8.1[b]	69.0±7.1	68.0±8.7	T[d]=0.008
	Mean	71.2±8.8	66.3±8.3[b]	67.9±8.1[b]	69.7±8.7	I[e]= 0.17
Resting Systolic BP[f] (mmHg)	C	120.5±11.4	117.2±8.5[b]	116.7±13.3	115.5±12.5[b]	G = 0.20
	W	118.4±9.6	112.9±9.4[b]	113.6±7.3[b]	113.2±8.7[b]	T= 0.003
	Mean	119.4±10.4	114.9±9.2[b]	115.1±10.6[b]	114.3±10.6[b]	I= 0.86
Resting Diastolic BP (mmHg)	C	75.5±8.5	71.4±9.0[b]	73.8±8.4	74.0±11.5	G = 0.72
	W	77.9±6.3	73.6±7.7[b]	72.5±7.0[b]	73.2±6.5[b]	T = 0.005
	Mean	76.8±7.5	72.6±8.3[b]	73.1±7.6[b]	73.6±9.1[b]	I= 0.32
Peak VO_2[g] (L/min)	C	2.0±0.4	———	———	2.0±0.4[b]	G = 0.42
	W	2.0±0.3	———	———	2.1±0.3[b]	T = 0.005
	Mean	2.0±0.3			2.1±0.4[b]	I = 0.13
Peak VO_2[g] (ml/kg/min)	C	23.0±4.2	———	———	25.3±4.4[b]	G = 0.74
	W	22.7±3.7	———	———	24.8±4.5[b]	T = 0.001
	Mean	22.8±3.9			25.1±4.4[b]	I = 0.68
Treadmill Time (min)	C	7.9±1.2	———	———	8.5±1.5[b]	G=0.72
	W	7.8±1.5	———	———	8.5±1.2[b]	T=0.001
	Mean	7.8±1.4			8.5±1.3[b]	I=0.71
Bench Press 1 RM[h] (kg)	C	34.4±7.6	———	———	35.5±70	G = 0.13
	W	34.5±9.4	———	———	33.8±8.4	T = 0.70
	Mean	34.5±8.5			34.6±7.8	I = 0.13
Bench Lifting Volume[i] (repetitions*kg)	C	156.0±77.2[j]	———	———	196.2±93.8[bj]	G = 0.002
	W	218.7±107.1	———	———	179.6±89.0[b]	T = 0.97
	Mean	189.2±98.5			187.4±90.7	I = 0.002
Leg Press 1 RM (kg)	C	196.0±57.3	———	———	204.4±56.4	G = 0.88
	W	200.0±67.7	———	———	195.3±65.6	T = 0.65
	Mean	198.1±61.9			199.6±61.0	I = 0.13
Leg Lifting Volume[k] (repetitions*kg)	C	1857.2±1018.7	———	———	1968.5±1040.4	G = 0.65
	W	2057.7±1103.3	———	———	2001.6±1235.5	T = 0.80
	Mean	1954.0±1059.6			1986.0±1136.6	I = 0.52

[a]Data are from 24 participants in the C group and 27 participants in the W group (n=51) who completed the 16-week study.
[b]$p<0.05$ difference from baseline.
[c]G=group alpha level
[d]T=time alpha level
[e]I=group x time interaction alpha level
[f]Blood Pressure
[g]Peak VO_2 = peak oxygen uptake.
[h]1 RM = 1 repetition maximum
[i]Bench press lifting volume at 80% 1 RM (kg)
[j]$p<0.01$ difference between C and W groups.
[k]Leg Press lifting volume at 80% 1 RM (kg)

group had a significantly lower baseline bench press lifting volume at 80% 1 RM (p=0.02). Univariate analysis of changes from baseline revealed participants in the C group had a moderate increase in bench press endurance at 16 weeks, while the W group displayed a decreased endurance (C 40.2±104.1; W -39.1±65.4 reps*kg) with effects of group (0.002) and time by diet (p=0.002) observed. Review of overall muscular strength and endurance data lead to a partial acceptance of H4.

Biochemical Markers of Health

Table 9 presents changes in Fasting blood lipid values observed at 0, 4, 10 and 16 weeks of program participation. An overall MANOVA of fasting lipids revealed a time effect (Wilks' Lamda p=0.001) with no significant overall MANOVA time x diet interactions (Wilks' Lamda p=0.21). MANOVA Univariate analyses were conducted on percent changes from baseline. Both groups experiences a decrease in CHL, with no differences observed between groups (C -6.1±11.0, -4.1±7.4, -2.3±9.5; W -6.8±9.4, -5.7±12.6, -6.3±13.0 %, p=0.49). LDL-c levels decreased in the W group with no differences observed between groups (C -6.9±17.3, -2.7±13.6, -4.6±17.2; W -5.6±14.5, -2.8±19.7, -11.4±15.9 %, p=0.31). The C group had a moderate increase in HDL-c, while the W group experienced a decrease in HDL-c at 4 and 10 weeks (C -2.1±12.5, 3.0±12.3, 5.9±18.3; W -9.5±11.5, -9.5±12.7, -1.6±14.6 %) with time by diet (p=0.03) and diet (p=0.005) effects observed leading to rejection of H04. The W group experienced an increase in the CHL:HDL ratio at 10 weeks (C -1.8±13.1, -4.0±10.1, -3.8±12.2; W 3.4±13.4, 5.3±12.5, -3.4±14.2 %) time by diet effect (p=0.04) and trend in diet effect (p=0.08) observed. The C group tended to experience greater reductions in TG levels,

with an effect of diet observed (p=0.06) (C -8.0±26.3, -11.7±18.0,-2.3±26.1; W 4.0± 25.2, 5.0±32.1, 7.8±52.4 %). Consideration of overall lipid biomarker data lead to a partial acceptance of H03.

Table 9. Fasting blood lipid values obtained at 0, 4, 10 and 16 weeks of program participation for the Curves (C) and Weight Watchers (W) groups[a]

Variable	Group	Week 0	Week 4	Week 10	Week 16	p-value
		← mean±standard deviation →				
Total Cholesterol (CHL) (mg/dl)[b]	C	174.63±30.29	163.54±31.47[c]	167.22±31.35[c]	170.67±33.15	G[d]= 0.34
	W	186.07±29.51	172.40±26.50[c]	174.67±30.97[c]	173.47±30.22[c]	T[e]=0.001
	Mean	180.69±30.17	168.23±29.00[c]	171.16±31.06[c]	172.10±31.34[c]	I[f]= 0.42
LDL Cholesterol (mg/dl)	C	97.67±25.97	90.54±28.83	94.04±25.07	92.88±28.07	G = 0.36
	W	110.00±26.08	102.54±24.15[c]	105.07±25.48	95.72±21.86[c]	T= 0.001
	Mean	104.20±26.50	96.89±26.87[c]	99.88±25.65	94.38±24.80[c]	I = 0.15
HDL Cholesterol (mg/dl)	C	51.71±15.07	49.72±11.98	52.66±14.15[g]	54.25±16.38	G = 0.41
	W	52.11±14.05	46.80±12.78[c]	46.44±11.53[c]	50.78±13.98	T= 0.001
	Mean	51.92±14.39	48.18±12.37[c]	49.37±13.08[c]	52.41±15.10	I= 0.02
Triglycerides[h] (mg/dl)	C	125.58±58.51	113.00±58.15	108.85±49.36[c]	123.54±63.40	G = 0.70
	W	120.00±43.88	122.22±48.62	121.85±53.35	130.56±98.60	T = 0.32
	Mean	122.63±50.83	117.88±52.97	115.73±51.37	127.25±83.17	I= 0.51
CHL/HDL ratio	C	3.8±1.0	3.5±0.9	3.4±0.9[g]	3.4±1.0	G = 0.24
	W	3.6±1.1	3.9±1.0	4.0±1.1[c]	3.6±1.2	T = 0.19
	Mean	3.7±1.0	3.7±1.0	3.7±1.0	3.5±1.1	I= 0.08

[a]Lipid data were obtained at 0, 4, 10 and 16 weeks on 24 participants in the C group and 27 participants in the W group (n=51) completing the 16-week weight loss program.
[b]To convert mg/dL cholesterol to mmol/L, multiply mg/dL by 0.026. To convert mmol/L cholesterol to mg/dL, multiply mmol/L by 38.6. Cholesterol of 194 mg/dl=5.04 mmol/L.
[c]p<0.05 difference from baseline.
[d]G=group alpha level.
[e]T=time alpha level.
[f]I=group x time interaction alpha level.
[g]p<0.01 difference between C and W groups.
[h]To convert mg/dL triglyceride to mmol/L, multiply mg/dL by 0.0113. To convert mmol/L triglyceride to mg/dL, multiply mmol/L by 88.6. Triglyceride of 137 mg/dl=1.55 mmol/L.

Table 10 presents changes in fasting glucose, insulin, HOMA and leptin values observed at 0, 4, 10 and 16 weeks of program participation. MANOVA analysis of fasting glucose

and insulin related variables revealed no overall Wilks' Lamda significant diet (p=0.18), or time by diet (p=0.44) effects. Univariate MANOVA analyses of percent changes in

Table 10. Fasting glucose, insulin and leptin values obtained at 0, 4, 10 and 16 weeks of program participation for the Curves (C) and Weight Watchers (W) groups[a]

		Week				
Variable	Group	0	4	10	16	p-value
		← mean±standard deviation →				
Glucose[b] (mg/dl)	C	91.50±15.10	88.69±13.51	90.75±19.45	87.88±16.24	G[c] = 0.74
	W	89.07±9.35	89.23±7.81	87.37±5.52	89.04±6.79	T[d] = 0.59
	Mean	90.22±12.32	88.97±10.76	88.96±13.89	88.49±12.07	I[e] = 0.30
Insulin[f] (IU/ml)	C	20.48±12.14[g]	15.86±7.01	14.54±7.40	16.08±8.15	G = 0.15
	W	10.99±4.67	10.65±4.37	11.17±6.40	10.60±5.47	T=0.63
	Mean	15.46±10.10	13.10±6.28	12.76±7.03	13.18±7.33	I = 0.47
Glucose/Insulin Ratio	C	9.0±12.3	9.5±14.3	9.0±7.5	11.5±23.5	G = 0.94
	W	9.5±4.0	10.34±7.5	11.7±9.8	15.5±24.9	T= 0.69
	Mean	9.3±8.8	9.4±10.0	10.4±8.8	13.7±24.1	I = 0.22
HOMA[h]	C	4.71±3.04[g]	3.44±1.49	3.23±1.62	3.56±2.03	G = 0.12
	W	2.40±1.01	2.35±0.93	2.40±1.36	2.35±1.24	T = 0.81
	Mean	3.49±2.48	2.86±1.33	2.80±1.53	2.92±1.75	I= 0.79
Leptin[l] ng/ml	C	66.28±26.66[i]	53.05±26.30	42.11±26.68[j]	52.70±25.10	G = 0.45
	W	51.35±22.00	46.76±27.15	45.48±34.62	42.30±29.49	T = 0.37
	Mean	58.38±25.21	49.72±26.67	43.90±30.88	47.20±27.74	I = 0.08

[a]Glucose, insulin, and leptin data were obtained at 0, 4, 10 and 16 weeks on 24 participants in the C group and 27 participants in the W group (n=51) completing the 16-week weight loss program.
[b]To convert mg/dL glucose to mmol/L, multiply mg/dL by 0.0555. To convert mmol/L glucose to mg/dL, multiply mmol/L by 18.0. Glucose of 99 mg/dl=5.5 mmol/L.
[c]G=group alpha level.
[d]T=time alpha level.
[e]I=group x time interaction alpha level.
[f]To convert µIU/ml insulin to pmol/L, multiply µIU/ml by 6.945. To convert pmol/L to µIU/ml, multiply pmol/L by 0.144. Insulin of 13.8 µIU/ml=95.8 pmol/L.
[g]p<0.01 difference between C and W groups.
[h]Homeostatic Model Assessment
[i]p<0.05 difference between C and W groups.
[j]p<0.05 difference from baseline.

glucose and glucose-to-insulin ratios revealed neither group experienced significant changes in glucose (p=0.30) or glucose-to insulin ratios (p=0.30). One way ANOVA analysis revealed higher insulin and HOMA values in the C group at baseline (insulin,

p=0.001; HOMA, p=0.001). Univariate ANCOVA revealed neither group experienced significant changes in fasting insulin levels (p=0.47) or HOMA (p=0.79. Large standard deviations revealed considerable variability in study participant insulin levels. Review of glucose related variables resulted in acceptance of H05. One way ANOVA revealed higher leptin levels in the C group at baseline (p=0.03). Univariate ANCOVA revealed The C group tended to have greater reductions in leptin when compared to the W group (C -13.24±21.77, -24.17±31.00, -13.58±25.45; W -4.59±20.15, -5.86±27.60, -9.04±28.97 ng/ml, p=0.08).

Psychosocial

Table 11 presents changes in psychosocial values observed at 0, 4, 10 and 16 weeks of program participation as determined by the SF36 Quality of Life Inventory. MANOVA analysis of SF36 quality of life indices revealed an overall time effect (Wilks' Lamda p=0.001) with no significant overall time x diet interactions observed (p=0.11). Univariate MANOVA analysis revealed the C group tended to have a greater increase in vitality than the W group (C 8.96±8.84, 12.24±10.64, 10.00±11.33; W 8.06±9.9, 5.09±12.72, 9.35±14.99) with trends in time by diet (p=0.10) and diet effects (p=0.10) observed. Both programs increased ratings of emotion (C 5.50±7.47, 10.41±10.74, 10.58±8.95; W 7.26±10.11, 8.74±13.81, 6.07±14.35, p=0.18) bodily pain (C 8.58±18.23, 8.63±20.01, 11.38±20.39; W 6.96±13.25, 5.85±15.81, 7.37±14.64, p=0.72) and general health (C 6.67±10.72, 9.36±12.13, 7.54±12.83; W 4.37±10.63, 4.70±13.20, 6.11±12.50, p=0.41) with no differences observed between groups. The C group experienced

Table 11. Changes in SF36 Quality of Life Inventory values observed at 0, 4, 10 and 16 weeks of program participation for the Curves (C) and Weight Watchers (W) groups[a]

Variable	Group	Week 0	Week 4	Week 10	Week 16	p-value
		mean±standard deviation				
Physical Fitness	C	88.54±12.72[b]	91.67±9.05	94.05±6.38	94.58±8.59	G[c]=0.63
	W	78.15±21.53	88.33±13.16	90.56±12.66	90.00±14.48	T[d]=0.14
	Mean	83.04±18.52	89.90±11.42	92.20±10.25	92.16±12.18	I[e]=0.73
Role Physical	C	342.71±83.14	370.83±53.50	374.45±46.73	373.96±51.33	G=0.22
	W	337.04±87.26	344.44±72.50	346.30±76.81	354.63±66.88	T=0.06
	Mean	339.71±84.58	356.86±65.02	359.50±65.36	363.73±60.2[f]	I=0.55
Role Emotional	C	337.74±69.31	351.33±69.53	371.73±46.96[f]	377.75±44.6[f]	G=0.18
	W	329.51±74.20	341.91±61.76	345.64±81.21	344.42±83.74	T=0.06
	Mean	333.38±71.35	346.35±65.04	357.92±67.95[f]	360.10±69.6[f]	I = 0.51
Vital	C	51.88±12.75	60.83±11.86[bf]	64.12±9.88[gf]	61.88±10.82[bf]	G=0.10
	W	48.98±12.56	57.04±11.29[f]	54.07±15.51[f]	58.33±16.41[f]	T=0.001
	Mean	50.34±12.61	58.82±11.60[f]	58.80±11.60[f]	60.00±14.04[f]	I= 0.10
Emotion	C	56.08±13.14	61.58±11.24[f]	66.49±9.79[f]	66.67±8.80[f]	G=0.10
	W	52.74±11.74	60.00±9.67[f]	61.48±12.46[f]	58.81±13.49[f]	T=0.001
	Mean	54.31±12.41	60.75±10.36[f]	63.84±11.45[f]	62.51±12.08[f]	I= 0.18
Social	C	49.19±6.88	49.46±8.66	47.90±7.96	50.00±3.69	G=0.36
	W	49.52±7.37	50.00±4.90	50.93±3.34	49.54±6.45	T=0.96
	Mean	49.36±7.08	49.75±6.86	49.50±6.10	49.76±5.29	I=0.51
Bodily Pain	C	70.63±17.14	79.21±11.89[f]	79.26±11.32[f]	82.00±10.69[f]	G=0.28
	W	69.37±15.56	76.33±13.31	75.22±15.93[f]	76.74±14.61[f]	T=0.001
	Mean	69.96±16.17	77.69±12.62[c]	77.12±13.97[f]	79.22±13.06[f]	I =0.72
General Health	C	67.79±16.38	74.46±13.58[f]	77.15±12.81[f]	75.33±13.56[f]	G=0.26
	W	65.81±14.97	70.19±13.44	70.52±13.55[f]	71.93±15.68[f]	T=0.001
	Mean	66.75±15.52	72.20±13.54[f]	73.64±13.49[f]	73.53±14.68[f]	I =0.41

[a] SF36 Quality of Life Inventory data were analyzed at 0, 4, 10, and 16 weeks of program participation for 24 participants in the C group and 27 participants in the W group (n=51).
[b] $p<0.05$ difference between C and W groups.
[c] G=group alpha level.
[d] T=time alpha level.
[e] I=group x time interaction alpha level.
[f] $p<0.05$ difference from baseline.
[g] $p<0.01$ difference between C and W groups.

increases in the role emotional rating (C 13.60±75.8, 34.00±63.50, 40.01±69.99; W 12.41±69.31, 16.14±80.26, 14.01±69.99, p=0.24) with no differences observed between

groups. Neither group experienced changes in ratings of role physical (p=0.55) or social (p=0.51). One way ANOVA revealed the C group rated physical fitness higher than the W group at baseline (p=0.05). Univariate ANCOVA revealed neither group experienced significant changes in rating of physical fitness (p=0.85). Large standard deviations revealed considerable variability between study participant ratings of SF36 indices. Consideration of overall psychosocial data resulted in a partial rejection of H06.

Eating Satisfaction

Table 12 depicts changes in eating satisfaction observed at 0, 4, 10 and 16 weeks of program participation. Overall MANOVA analysis of eating satisfaction indices revealed a significant time effect (Wilks' Lamda p=0.001) with a trend in time by diet effect observed (Wilks' Lamda p=0.06). Feelings of fullness decreased in the W group (C 0.42±1.93, 0.01±1.75, 0.54±1.44; W -0.81±1.8, -0.67±1.9, -0.78±1.5, p=0.02). One way ANOVA revealed the W group rated food quality lower at baseline than the C group (ANOVA p=0.05). Univariate ANCOVA revealed neither group experienced significant changes in food quality (p=0.52). Neither the C group nor the W group experienced changes in hunger (p=0.51) or food satisfaction scores (p=0.68). Both the C group and W group experienced increases in energy (C 1.83±1.81, 1.23±2.0, 2.00±2.04; W 1.30±1.84, 1.63±1.78, 1.26±1.9, p=0.41) and decreases in appetite (C -0.83±1.49, -0.61±1.40, -0.50±1.50; W -0.48±1.45, -0.93±2.11, -0.44±1.45, p=0.72).

Table 12. Changes in eating satisfaction values observed at 0, 4, 10 and 16 weeks of program participation for Curves (C) and Weight Watchers (W) groups[a]

Variable	Group	Week 0	Week 4	Week 10	Week 16	p-value
		← mean+standard deviation →				
Appetite	C	6.17±1.20	5.33±1.17[b]	5.55±1.56[b]	5.67±1.43	G[c]= 0.62
	W	6.00±1.73	5.52±1.25	5.07±1.17[b]	5.56±1.25	T[d]=0.003
	Mean	6.08±1.94	5.43±1.20[b]	5.20±1.38[b]	5.61±1.33[b]	I[e]=0.45
Hunger	C	5.33±1.58	5.08±1.50	5.08±1.91	5.25 ±1.57	G = 0.70
	W	5.07±0.98	5.00±1.44	4.81±1.36	5.37±1.60	T= 0.38
	Mean	5.20±1.30	5.04±1.46	4.94±1.63	5.31±1.60	I= 0.80
Satisfied food	C	6.59±1.47	6.13±1.33	6.30±1.68	6.21±1.91	G =0.52
	W	6.19±1.62	6.07±1.69	6.07±1.60	6.04±1.40	T=0.64
	Mean	6.37±1.55	6.10±1.51	6.18±1.62	6.12±1.64	I= 0.90
Fullness	C	6.08±1.53	6.5±1.44[f]	6.08±1.53	6.63±1.53[f]	G =0.36
	W	6.63±1.18	5.81±1.69[b]	5.96±1.34	5.85±1.51[b]	T=0.58
	Mean	6.37±1.37	6.14±1.60	6.02±1.42	6.23±1.55	I = 0.04
Energy	C	5.13±1.73	6.96±1.33[b]	6.39±1.58[b]	7.13±1.65[b]	G = 0.02
	W	4.52±1.72	5.81±1.44[b]	6.15±1.59[b]	5.78±1.80[b]	T=0.001
	Mean	4.80±1.73	6.35±1.49[b]	6.26±1.57[b]	6.41±1.85[b]	I= 0.12
Quality	C	4.58±2.15[g]	6.48±1.25	6.39±1.31	7.08±1.59	G = 0.83
	W	3.56±1.50	6.48±1.34	6.41±1.40	6.11±1.45	T = 0.04
	Mean	4.04±1.89	6.48±1.28	6.40±1.34	6.57±1.58	I= 0.52

[a]Eating Satisfaction Questionnaire data were analyzed at 0, 4, 10, and 16 weeks of program participation for 24 participants in the C group and 27 participants in the W group (n=51).
[b]$p<0.05$ difference from baseline.
[c]G=group alpha level.
[d]T=time alpha level.
[e]I=group x time interaction alpha level.
[f]$p<0.05$ difference between C and W groups.

CHAPTER V

SUMMARY

The alarming rise in the prevalence of obesity, along with the direct and indirect costs of the disease, has caused much emphasis to be placed on finding efficacious weight loss interventions. A multitude of industry-sponsored commercial weight loss programs have emerged that are marketed as producing significant weight loss in overweight or obese individuals. Many of these programs are based on scientifically proven weight loss strategies such a following a nutritionally sound reduced kilocalorie diet plan, consuming nutritious prepared meals or meal replacements, providing physical activity recommendations or facilitating participation in a supervised exercise program, and/or behavior modification. Conducting comparative effectiveness trials on commercial programs that utilize proven weight loss strategies will reveal the relative efficacy, safety, and value of these programs [195, 196]. Our investigation compared the efficacy of the Curves® Fitness and Weight Management Plan to the Weight Watchers® Momentum™ Plan on body composition and markers of health and fitness in previously sedentary obese women.

Few studies have investigated the efficacy of commercial weight loss programs on weight loss and markers of fitness and health in randomized controlled trials. Truby et al.[45, 46] conducted a randomized controlled comparative effectiveness trial of 4 commercially available weight loss diets over a period of 6 months. A commercial program based on regular weighing, advice about diet and physical activity, motivation

and group support; a commercial program based on a low fat diet plan and weekly exercise; a meal replacement plan, and a self-monitored low carbohydrate eating plan were investigated. All diets resulted in clinically meaningful weight loss (4.9-7.3 %) and fat loss (2.1-3.6 %) compared with a no diet control, -0.6 and -0.1 % respectively, with no differences observed between diets. Both of the commercial weight loss programs reduced plasma LDL-c. Additionally, the group support based commercial program and the self-monitored low carbohydrate plan lead to reduced TG at 6 months; however, LDL particle size was also increased following these interventions [45, 46].

In our investigation, the C group followed a structured higher protein meal-plan-based cyclic diet plan while the W group followed a points centered diet plan that was based on weight, height, age, and activity level. The C group meal plan resulted in a similar average caloric intake as the W diet plan, with approximately 1,353±232 vs. 1,356±232 kcalories/d consumed on average throughout the study. The C group consumed a greater percentage of kcalories as protein (26±5 vs. 22±3 %), a higher percentage of kcalories as fat (34±5 vs. 30±5 %) and a lower percentage of kcalories as carbohydrate (39±8 vs. 47± 6%) in comparison to the W group. Though participants in both the C group and W group increased total physical activity 3,731±2,214 vs. 3,184±2,485 MET-min/week, participants in the C group increased recreational activity to a greater extent (1,097±273 vs. 678±646 MET-min/week) than the W group. Furthermore, the C group performed circuit resistance-training interspersed with a low-impact calisthenics three times each week, which resulted in a greater increase in vigorous activity 1,081±635 vs. 525±577 MET-min/week in comparison to the W group.

Both the C program and the W program resulted in similar levels of moderate physical activity 1,384±1,869 vs. 1,388±1,453 MET-min/week, job physical activity 1,382±1,869 vs. 911±1,332 MET-min/week, house physical activity 810±633 vs. 1,028±1285 MET-min/week, low physical activity 1,252±1,424 vs. 1,261±1,316 MET-min/week and transportation physical activity 417±619 vs. 493±737 MET-min/week; however, only the W group experienced an observable increases in low and transportation physical activity.

In our study, overall body composition changes were more favorable in the C group. Both groups lost a similar amount of total mass over the 16 week study with the C group experiencing a loss of 5.1±3.9 kg (5.8±4.0 % decrease in body mass) and the W group having a reduction of 5.5±4.6 kg (6.9±5.8 % decrease in body mass). However, the C group maintained lean tissue with a modest increase of 1.3±4.0 kg, while the W group had a reduction in lean mass of -2.5±5.1 kg. The C group also tended to have a greater reduction in fat mass -6.4±5.9 vs. -2.9±7.8 kg and percent body fat -4.7±5.4 vs. -1.4±8.1 % following the 16 week study. Both the C and W programs resulted in reductions in BMI -1.8±1.4 vs. -2.3±1.5 kg/m^2, waist circumference -6.2±5.1 vs. -5.4±5.2 cm and hip circumference -5.8±5.0 vs. -7.4 ±4.9 cm. Our results are in agreement with a previous study conducted by Kreider and colleagues[197], where the C program resulted in significant reductions in body mass, fat mass, BMI and waist circumference, while preserving muscle mass and other investigations where resistance based circuit training combined with calorically restricted high protein diets resulted in enhanced loss of body fat and preservation of lean body mass [33, 34]. As observed in our

investigation, previous trials have found the W program to be efficacious for the reduction of total body mass, body fat mass, BMI and waist circumference, though preservation of lean tissue mass has not been demonstrated [45, 198-200]. Our findings suggest adherence to a high protein meal-plan based diet and supervised resistance-training exercises interspersed with a low-impact calisthenics exercise program is more effective in promoting positive body composition changes than a points based diet plan combined with education and encouragement to increase physical activity.

Both the C program and W program resulted in improvements in cardiovascular capacity. The C group experienced a 5.2±10.1 % increase in peak VO_2 in L/min and 11.1±11.5 % increase in peak VO_2 in ml/kg/min, while the W group experienced a 1.3±6.6 % increase in peak VO_2 in L/min and 9.3±8.5 % increase in ml/kg/min. Both the C group and W group experienced decreases in RHR and diastolic BP during program participation; however, the W experienced a reduction in diastolic BP throughout the study and at 16 weeks (-1.5±10.1 vs. -4.7±7.9 mmHg). Both the C group and W group experienced a decrease in resting systolic BP at 16 weeks (-5.0±11.2 vs. -5.3±8.9 mmHg). Previous investigations have demonstrated reductions in blood pressure following participation in both the C program [35, 197] and W program [198, 200]. Both the C and W programs resulted in an increase in REE values by 9.7±12.8 vs. 5.7±18.0 % after 16 weeks of program participation. We did not observe any changes in upper or lower body 1RM strength or lower body endurance in either the C group or the W group during our investigation. However, the W group experienced a decrease in upper body muscular endurance, while participants in the C group had an increase in bench press

repetitions at 80 % 1RM, -12.3±35.5 vs. 49.1±89.0 %. The increase in upper body endurance observed in the C groups was most likely a result of biweekly Zumba workouts, which emphasizes muscular endurance. Other trials evaluating changes in muscular strength and cardiovascular fitness following participation in the C program have observed significant increases in upper and lower body 1 RM strength and peak oxygen uptake at 2.5, 3.5 and 8.5 months of program participation [35, 197]. Though we did not observe significant changes in 1RM strength in our investigation, this may be a result of greater upper and lower body 1RM strength at baseline in our study participants in comparison to other trials. Changes in muscular strength and fitness and cardiovascular endurance have not been evaluated in previous trials investigating the W program.

In our investigation, both the C group and W group had reductions in CHL after 16 weeks of program participation, -2.3±9.5 vs. -6.3±13 %. The W group also experienced reductions in LDL-c levels at 16 weeks, -11.4±15.9 vs. -4.6±17.2 %. While the C group maintained HDL-c levels throughout the investigation, the W group experienced a -9.5±11.5 % reduction in HDL-c at 4 and 10 weeks and a 5.3±12.5 % increase in CHL:HDL ratio at 10 weeks. The C group also experienced a -11.7±18.0 % reduction in TG levels at 10 weeks of program participation. Kersick and colleagues[35] examined changes in lipid panels after 3.5 months of participation in the C program's resistance-based circuit training combined with calorically reduced diets of varying macronutrient composition. A high carbohydrate, low protein intake resulted in reduced CHL, while a low carbohydrate moderate protein meal plan resulted in reduced TG after 14 weeks of program participation. All macronutrient combinations in combination with

the C program exercise plan maintained HDL-c levels [35]. Other investigations have also shown a superior affect of elevated protein ingestion [23, 25, 29] and elevated protein combined with a supervised circuit training based programs [34] for the reduction of triglycerides while maintaining HDL-C [33]. Previous investigations of the W program have shown varying lipid panel changes following program intervention. Bellise et al.[200] observed reductions in CHL and LDL-c with no changes in HDL-c, TG or in CHL:HDL-c ratio after 3 months of program participation, while Morgan et al.[46] observed decreased TG, LDL-c and HDL-c following 2-6 months of participation. In contrast, Jebb and coworkers[199] observed no significant changes in lipid panel values following 12 months of participation in the W program. We did not observe any changes in glucose, glucose to insulin ratio, insulin or HOMA following participation in either the C or W program. However, the C group tended to have greater reductions in leptin when compared to the W group -13.5±25.5 vs. -9.0±29.0 %. These results support previous findings that adherence to a meal plan based diet combined with weekly resistance-based circuit training can result in reductions in serum leptin levels[35]. The greater reduction in leptin may be directly related to the greater fat mass loss observed in the C group, as the concentration of leptin in plasma has been shown to parallel adipose tissue mass [99, 104].

Moderate weight loss has been associated with improved health related quality of life [142, 145]. Both the C program and W programs resulted in improved physical and mental health ratings following 16 weeks of program participation. Both the C and W groups experienced increases in ratings of emotion (23.3±23.6 vs. 15.5±34.3%), bodily pain (24.8±42.3 vs. 13.9±25.0%), and general health (14.7±22.3 vs. 11.0±24.8%) at 16

weeks. The C group tended to have a greater increase in vitality than the W group, 24.4±30.8% vs. 22.3±36.1%, and also experienced an improvement the role emotional rating, 16.4±28.7% vs. 7.7±30.9%. Other trials have found similar increases in vitality following participation in the C program [35, 197]. Feelings of fullness decreased in the W group following 16 weeks of program participation while remaining elevated in the C group, -11.1±27.9 vs. 13.2±30.5%. Feelings of fullness may have been maintained in the C program secondary to the higher protein intake consumed by the C group, as hypocaloric diets in combination with elevated protein intake have been shown to result in increased satiety[11, 20-22]. Moreover, exercise induced weight loss has also been associated with improved appetite control [31, 32]. Both the C group and W group experienced increases in energy, 34.0±55.1 vs. 46.1±85.3 % at 16 weeks.

There are several limitations in conducting weight loss clinical trials that should be taken into account when interpreting the results of this investigation. First, participants who volunteer to participate in weight loss trails are often more motivated to adhere to weight loss programs than the general population. Furthermore, study participants are continually monitored and encouraged to meet program requirements. In our trial, the C group had a compliance rate of 81 % or greater for exercise and the W group had 90 % compliance with attendance of weekly meetings. Although these are compliance rates for similar to those observed in other long term weight loss trials, results may not be generalizable to populations that do not receive similar monitoring and support. Second, both of the programs evaluated in this trial have required costs for participation in the program. Therefore, utilization of these programs may be limited to

populations that can afford to participate in them. Thirdly, the results observed in this trial are limited to the population studied, sedentary and obese women, and may not be applicable to other populations. Fourth, this trial lasted for only 4 months. Thus, this trial provides no information on maintenance of body composition changes and improvements in markers of health and fitness with long term participation in these programs. Finally, results are limited to the inherent difficulties in conducting clinical trials of this nature, compliance to diet and/or exercise protocols, and accuracy in data collection and analysis.

Conclusion

In our investigation, participants in both the C group and W groups experienced clinically significant weight loss, reductions in BMI, decreased waist and hip circumferences, increases in physical activity, improvements in cardiovascular endurance and positive changes in biomarkers of cardiovascular disease risk. However, adherence to a higher protein diet in combination with the resistance-based circuit training was found to be more efficacious in promoting fat mass loss, preserving fat free mass, increasing upper body endurance, and maintaining HDL-c levels. Participants in the C group engaged in a greater amount of vigorous recreational activity, while participants in the W group had increases in low physical activity. Present results support previous findings that participating in a supervised resistance-based exercise program may be more efficacious in promoting favorable changes in muscular fitness in sedentary obese women compared to a lifestyle based program with encouragement to increase physical activity. Both the C program and W program lead to improved ratings

of physical and mental health and eating satisfaction. However, the C group tended to experience a greater increase in vitality and emotional stability when dealing with daily challenges. Additionally, feelings of fullness were maintained in the C group, whereas the W group experienced a decrease. Feelings of fullness may have been maintained as a result of the higher protein intake consumed by the C group.

Our investigation supports the efficacy of both the C program and W program for promoting weight loss and improvement in markers of fitness and health. However, our findings suggest adherence to the C program's higher protein meal-plan based diet and supervised resistance-training exercises interspersed with a low-impact calisthenics exercise program is more effective in promoting positive body composition changes, enhancing muscular fitness, and improving ratings of physical and mental health and eating satisfaction than adherence to the W program points based diet plan combined with behavior modification advice, group support and encouragement to increase physical activity.

Commercial weight loss programs have the potential of reaching large numbers of overweight or obese individuals. Conducting randomized and controlled research trials on industry sponsored weight control programs will provide conclusions and results that health practitioners and governmental agencies can utilize when recommending and/or funding programs for overweight and obese populations. Future research on commercial weight loss programs should include long-term investigations that evaluate the costs of these programs relative to outcome benefits in terms of

prevention of obesity related illnesses, improvements in markers of health and enhancement of quality of life.

REFERENCES

1. World Health Organization 2010. Accessed from www.who.in on November 12, 2010.

2. Flegal KM, Carroll MD, Ogden CL, Curtin LR. Prevalence and trends in obesity among US adults, 1999-2008. *JAMA.* Jan 20;303(3):235-241.

3. Kenchaiah S, Evans JC, Levy D, et al. Obesity and the risk of heart failure. *N Engl J Med.* Aug 1 2002;347(5):305-313.

4. Ades PA, Savage PD, Harvey-Berino J. The treatment of obesity in cardiac rehabilitation. *J Cardiopulm Rehabil Prev.* Sep-Oct;30(5):289-298.

5. Calle EE, Thun MJ. Obesity and cancer. *Oncogene.* Aug 23 2004;23(38):6365-6378.

6. Calle EE, Kaaks R. Overweight, obesity and cancer: epidemiological evidence and proposed mechanisms. *Nat Rev Cancer.* Aug 2004;4(8):579-591.

7. El-Atat F, Aneja A, McFarlane S, Sowers J. Obesity and hypertension. *Endocrinol Metab Clin North Am.* Dec 2003;32(4):823-854.

8. Sowers JR. Obesity as a cardiovascular risk factor. *Am J Med.* Dec 8 2003;115 Suppl 8A:37S-41S.

9. Park YW, Zhu S, Palaniappan L, Heshka S, Carnethon MR, Heymsfield SB. The metabolic syndrome: prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988-1994. *Arch Intern Med.* Feb 24 2003;163(4):427-436.

10. Wysham CH. New perspectives in type 2 diabetes, cardiovascular risk, and treatment goals. *Postgrad Med.* May;122(3):52-60.

11. Paddon-Jones D, Westman E, Mattes RD, Wolfe RR, Astrup A, Westerterp-Plantenga M. Protein, weight management, and satiety. *Am J Clin Nutr.* May 2008;87(5):1558S-1561S.

12. Renzaho A, Wooden M, Houng B. Associations between body mass index and health-related quality of life among Australian adults. *Quality of Life Research.*19(4):515-520.

13. Tsai AG, Wadden TA, Sarwer DB, et al. Metabolic syndrome and health-related quality of life in obese individuals seeking weight reduction. *Obesity (Silver Spring).* Jan 2008;16(1):59-63.

14. Algul A, Ates MA, Semiz UB, et al. Evaluation of general psychopathology, subjective sleep quality, and health-related quality of life in patients with obesity. *Int J Psychiatry Med.* 2009;39(3):297-312.

15. Finkelstein EA, Trogdon JG, Cohen JW, Dietz W. Annual medical spending attributable to obesity: payer-and service-specific estimates. *Health Aff (Millwood).* Sep-Oct 2009;28(5):w822-831.

16. Mavri A, Stegnar M, Sentocnik JT, Videcnik V. Impact of weight reduction on early carotid atherosclerosis in obese premenopausal women. *Obes Res.* Sep 2001;9(9):511-516.

17. Giltay EJ, Lambert J, Elbers JM, Gooren LJ, Asscheman H, Stehouwer CD. Arterial compliance and distensibility are modulated by body composition in both men and women but by insulin sensitivity only in women. *Diabetologia.* Feb 1999;42(2):214-221.

18. Farnsworth E, Luscombe ND, Noakes M, Wittert G, Argyiou E, Clifton PM. Effect of a high protein, energy-restricted diet on body composition, glycemic control, and lipid concentrations in overweight and obese hyperinsulinemic men and women. *Am J Clin Nutr.* 2003;78:31-39.

19. Lien LF, Haqq AM, Arlotto M, et al. The STEDMAN project: biophysical, biochemical and metabolic effects of a behavioral weight loss intervention during weight loss, maintenance, and regain. *OMICS.* Feb 2009;13(1):21-35.

20. Soenen S, Westerterp-Pantenga MS. Protein and satiety: implications for weight management. *Curr Opin Clin Nutr Metab Car.* 2008;11(6):747-751.

21. Veldhorst M, Smeets A, Soenen S, et al. Protein-induced satiety: effects and mechanisms of different proteins. *Physiol Behav.* May 23 2008;94(2):300-307.

22. Westerterp-Plantenga MS, Nieuwenhuizen A, Tome D, Soenen S, Westerterp KR. Dietary protein, weight loss, and weight maintenance. *Annu Rev Nutr.* 2009;29:21-41.

23. Layman DK, Boileau RA, Erickson DJ, et al. A reduced ratio of dietary carbohydrate to protein improves body composition and blood lipid profiles during weight loss in adult women. *J Nutr.* Feb 2003;133(2):411-417.

24. Due A, Toubro S, Skov AR, Astrup A. Effect of normal-fat diets, either medium or high in protein, on body weight in overweight subjects: a randomised 1-year trial. *International Journal Of Obesity And Related Metabolic Disorders: Journal Of The International Association For The Study Of Obesity.* 2004;28(10):1283-1290.

25. Skov AR, Toubro S, Ronn B, Holm L, Astrup A. Randomized trial on protein vs carbohydrate in ad libitum fat reduced diet for the treatment of obesity. *Int J Obes Relat Metab Disord.* May 1999;23(5):528-536.

26. Sacks FM, Bray GA, Carey VJ, et al. Comparison of weight-loss diets with different compositions of fat, protein, and carbohydrates. *N Engl J Med.* Feb 26 2009;360(9):859-873.

27. Luscombe-Marsh ND, Noakes M, Wittert GA, Keogh JB, Foster P, Clifton PM. Carbohydrate-restricted diets high in either monounsaturated fat or protein are equally effective at promoting fat loss and improving blood lipids. *Am J Clin Nutr.* Apr 2005;81(4):762-772.

28. Farnsworth E, Luscombe ND, Noakes M, Wittert G, Argyiou E, Clifton PM. Effect of a high-protein, energy-restricted diet on body composition, glycemic control, and lipid concentrations in overweight and obese hyperinsulinemic men and women. *Am J Clin Nutr.* Jul 2003;78(1):31-39.

29. Layman DK, Shiue H, Sather C, Erickson DJ, Baum J. Increased dietary protein modifies glucose and insulin homeostasis in adult women during weight loss. *J Nutr.* Feb 2003;133(2):405-410.

30. Shaw K, Gennat H, O'Rourke P, Del Mar C. Exercise for overweight or obesity. *Cochrane Database Syst Rev.* 2006(4):CD003817.

31. Elder SJ, Roberts SB. The effects of exercise on food intake and body fatness: a summary of published studies. *Nutr Rev.* Jan 2007;65(1):1-19.

32. Martins C, Kulseng B, King NA, Holst JJ, Blundell JE. The effects of exercise-induced weight loss on appetite-related peptides and motivation to eat. *J Clin Endocrinol Metab.* Apr;95(4):1609-1616.

33. Layman DK, Evan E, Baum JI, Seyler J, Erickson DJ, Boileau RA. Dietary protein and exercise have additive effects on body composition during weight loss in adult women. *J Nutr.* 2005;135(8):1903-1910.

34. Meckling KS, Sherfey R. A randomized trial of a hypocaloric hihgh-protein diet, with and without exercise on weight loss, fitness, and markers of the MEtabolic Syndrom in overweight and obese women. *Appl Physiol Nutr Metab.* 2007;32:743-752.

35. Kerksick C, Thomas A, Campbell B, et al. Effects of a popular exercise and weight loss program on weight loss, body composition, energy expenditure and health in obese women. *Nutr Metab.* 2009;6(23).

36. Shaw K, O'Rourke P, Del Mar C, Kenardy J. Psychological interventions for overweight or obesity. *Cochrane Database Syst Rev.* 2005(2):CD003818.

37. Brown T, Avenell A, Edmunds LD, et al. Systematic review of long-term lifestyle interventions to prevent weight gain and morbidity in adults. *Obes Rev.* Nov 2009;10(6):627-638.

38. Wing RR, Greeno CG. Behavioural and psychosocial aspects of obesity and its treatment. *Baillieres Clin Endocrinol Metab.* Jul 1994;8(3):689-703.

39. Wilson GT. Cognitive behavior therapy for eating disorders: progress and problems. *Behav Res Ther.* Jul 1999;37 Suppl 1:S79-95.

40. Cooper Z, Fairburn CG. A new cognitive behavioural approach to the treatment of obesity. *Behav Res Ther.* May 2001;39(5):499-511.

41. Miller W. Weight Watchers WTW. *Morningstar StockInvestor.* 2010;9(7):8-8.

42. Jolly K, Daley A, Adab P, et al. A randomised controlled trial to compare a range of commercial or primary care led weight reduction programmes with a minimal intervention control for weight loss in obesity: the Lighten Up trial. *BMC Public Health.*10:439.

43. Weight Watchers: The POINTS system. 2010. Accessed from www.weightwatchers.com on November 12, 2010.

44. Truby H, Hiscutt R, Herriot AM, et al. Commercial weight loss diets meet nutrient requirements in free living adults over 8 weeks: a randomised controlled weight loss trial. *Nutr J.* 2008;7:25.

45. Truby H, Baic S, deLooy A, et al. Randomised controlled trial of four commercial weight loss programmes in the UK: initial findings from the BBC "diet trials". *BMJ.* Jun 3 2006;332(7553):1309-1314.

46. Morgan LM, Griffin BA, Millward DJ, et al. Comparison of the effects of four commercially available weight-loss programmes on lipid-based cardiovascular risk factors. *Public Health Nutr.* Jun 2009;12(6):799-807.

47. Heshka S, Greenway F, Anderson JW, et al. Self-help weight loss versus a structured commercial program after 26 weeks: a randomized controlled study. *Am J Med.* Sep 2000;109(4):282-287.

48. Curves: Curves for Women: Company Fact Sheet. 2010. Accessed from www.curves.com on November 12 , 2008.

49. Heavin G, Rodman N, Findley C. Curve Fitness and Weight Management Plan. Woodway, TX: Curves International, Inc.; 2008.

50. Kresta J, Byrd M, Oliver J, et al. Effects of intermittent dieting during resistance training in Women I: weight loss and energy expenditure. *Med Sci Sports Exerc.* May 2011;43(5):471.

51. Byrd M, Kresta J, Oliver J, et al. Effects of intermittent dieting during resistance training in women III: fitness. *Med Sci Sports Exerc.* May 2011;43(5):472.

52. Mardock M, Kresta J, Byrd M, et al. Effects of intermittent dieting during resistance training in women II: health markers. *Med Sci Sports Exerc.* May 2011;43(5):471-2..

53. Baetge C, Kresta J, Byrd M, et al. Effects of intermittent dieting during resistance training in women IV: quality of life. *Med Sci Sports Exerc.* May 2011;43(5):470-1.

54. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. *World Health Organ Tech Rep Ser.* 1995;854:1-452.

55. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults--The Evidence Report. National Institutes of Health. *Obes Res.* Sep 1998;6 Suppl 2:51S-209S.

56. Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath CW, Jr. Body-mass index and mortality in a prospective cohort of U.S. adults. *N Engl J Med.* Oct 7 1999;341(15):1097-1105.

57. Han TS, Seidell JC, Currall JE, Morrison CE, Deurenberg P, Lean ME. The influences of height and age on waist circumference as an index of adiposity in adults. *Int J Obes Relat Metab Disord.* Jan 1997;21(1):83-89.

58. Despres JP, Prud'homme D, Pouliot MC, Tremblay A, Bouchard C. Estimation of deep abdominal adipose-tissue accumulation from simple anthropometric measurements in men. *Am J Clin Nutr.* Sep 1991;54(3):471-477.

59. Bjorntorp P. The associations between obesity, adipose tissue distribution and disease. *Acta Med Scand Suppl.* 1988;723:121-134.

60. Yan RT, Yan AT, Anderson TJ, et al. The differential association between various anthropometric indices of obesity and subclinical atherosclerosis. *Atherosclerosis.* Nov 2009;207(1):232-238.

61. Chan RS, Woo J. Prevention of overweight and obesity: how effective is the current public health approach. *Int J Environ Res Public Health.* Mar;7(3):765-783.

62. Klein S, Allison DB, Heymsfield SB, et al. Waist circumference and cardiometabolic risk: a consensus statement from Shaping America's Health: Association for Weight Management and Obesity Prevention; NAASO, The Obesity Society; the American Society for Nutrition; and the American Diabetes Association. *Am J Clin Nutr.* May 2007;85(5):1197-1202.

63. Lean ME, Han TS, Morrison CE. Waist circumference as a measure for indicating need for weight management. *BMJ.* Jul 15 1995;311(6998):158-161.

64. Wang Y, Rimm EB, Stampfer MJ, Willett WC, Hu FB. Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. *Am J Clin Nutr.* Mar 2005;81(3):555-563.

65. Janiszewski PM, Janssen I, Ross R. Does waist circumference predict diabetes and cardiovascular disease beyond commonly evaluated cardiometabolic risk factors? *Diabetes Care.* Dec 2007;30(12):3105-3109.

66. Han TS, van Leer EM, Seidell JC, Lean ME. Waist circumference action levels in the identification of cardiovascular risk factors: prevalence study in a random sample. *BMJ.* Nov 25 1995;311(7017):1401-1405.

67. Despres JP. Health consequences of visceral obesity. *Ann Med.* Nov 2001;33(8):534-541.

68. Lakka HM, Lakka TA, Tuomilehto J, Salonen JT. Abdominal obesity is associated with increased risk of acute coronary events in men. *Eur Heart J.* May 2002;23(9):706-713.

69. Kanaya AM, Vittinghoff E, Shlipak MG, et al. Association of total and central obesity with mortality in postmenopausal women with coronary heart disease. *Am J Epidemiol.* Dec 15 2003;158(12):1161-1170.

70. Klein S, Allison DB, Heymsfield SB, et al. Waist circumference and cardiometabolic risk: a consensus statement from shaping America's health: Association for Weight Management and Obesity Prevention; NAASO, the Obesity Society; the American Society for Nutrition; and the American Diabetes Association. *Diabetes Care.* Jun 2007;30(6):1647-1652.

71. Janssen I, Katzmarzyk PT, Ross R. Waist circumference and not body mass index explains obesity-related health risk. *Am J Clin Nutr.* Mar 2004;79(3):379-384.

72. Ford ES, Mokdad AH, Giles WH. Trends in waist circumference among U.S. adults. *Obes Res.* Oct 2003;11(10):1223-1231.

73. O'Rahilly S, Farooqi IS. Human obesity as a heritable disorder of the central control of energy balance. *Int J Obes (Lond).* Dec 2008;32 Suppl 7:S55-61.

74. Hinney A, Vogel CI, Hebebrand J. From monogenic to polygenic obesity: recent advances. *Eur Child Adolesc Psychiatry.* Mar;19(3):297-310.

75. Viguerie N, Vidal H, Arner P, et al. Adipose tissue gene expression in obese subjects during low-fat and high-fat hypocaloric diets. *Diabetologia.* Jan 2005;48(1):123-131.

76. Gluckman PD, Hanson MA. Developmental and epigenetic pathways to obesity: an evolutionary-developmental perspective. *Int J Obes (Lond).* Dec 2008;32 Suppl 7:S62-71.

77. Rankinen T, Zuberi A, Chagnon YC, et al. The human obesity gene map: the 2005 update. *Obesity (Silver Spring).* Apr 2006;14(4):529-644.

78. Galgani J, Ravussin E. Energy metabolism, fuel selection and body weight regulation. *Int J Obes (Lond).* Dec 2008;32 Suppl 7:S109-119.

79. Capel F, Klimcakova E, Viguerie N, et al. Macrophages and adipocytes in human obesity: adipose tissue gene expression and insulin sensitivity during calorie restriction and weight stabilization. *Diabetes.* Jul 2009;58(7):1558-1567.

80. Stunkard AJ, Harris JR, Pedersen NL, McClearn GE. The body-mass index of twins who have been reared apart. *N Engl J Med.* May 24 1990;322(21):1483-1487.

81. Wardle J, Carnell S, Haworth CM, Plomin R. Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment. *Am J Clin Nutr.* Feb 2008;87(2):398-404.

82. Bluher M. Adipose tissue dysfunction in obesity. *Exp Clin Endocrinol Diabetes.* Jun 2009;117(6):241-250.

83. Loos RJ, Bouchard C. Obesity--is it a genetic disorder? *J Intern Med.* Nov 2003;254(5):401-425.

84. Hainer V, Zamrazilova H, Spalova J, et al. Role of hereditary factors in weight loss and its maintenance. *Physiol Res.* 2008;57 Suppl 1:S1-15.

85. Figlewicz DP, Sipols AJ. Energy regulatory signals and food reward. *Pharmacology Biochemistry and Behavior.*97(1):15-24.

86. Novak CM, Levine JA. Central neural and endocrine mechanisms of non-exercise activity thermogenesis and their potential impact on obesity. *J Neuroendocrinol.* Dec 2007;19(12):923-940.

87. Hu FB, Li TY, Colditz GA, Willett WC, Manson JE. Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. *JAMA.* Apr 9 2003;289(14):1785-1791.

88. Wyatt HR, Peters JC, Reed GW, Barry M, Hill JO. A Colorado statewide survey of walking and its relation to excessive weight. *Med Sci Sports Exerc.* May 2005;37(5):724-730.

89. Weinsier RL, Hunter GR, Heini AF, Goran MI, Sell SM. The etiology of obesity: relative contribution of metabolic factors, diet, and physical activity. *The American Journal of Medicine.* 1998;105(2):145-150.

90. Wang Z, Heshka S, Zhang K, Boozer CN, Heymsfield SB. Resting energy expenditure: systematic organization and critique of prediction methods. *Obes Res.* May 2001;9(5):331-336.

91. Westerterp KR. Physical activity, food intake, and body weight regulation: insights from doubly labeled water studies. *Nutrition Reviews.* 2010;68(3):148-154.

92. Stubbs RJ, Hughes DA, Johnstone AM, Horgan GW, King N, Blundell JE. A decrease in physical activity affects appetite, energy, and nutrient balance in lean men feeding ad libitum. *Am J Clin Nutr.* Jan 2004;79(1):62-69.

93. Wiedmer P, Nogueiras R, Broglio F, D'Alessio D, Tschop MH. Ghrelin, obesity and diabetes. *Nat Clin Pract Endocrinol Metab.* Oct 2007;3(10):705-712.

94. Hall WL, Millward DJ, Long SJ, Morgan LM. Casein and whey exert different effects on plasma amino acid profiles, gastrointestinal hormone secretion and appetite. *Br J Nutr.* Feb 2003;89(2):239-248.

95. Lopez M, Lage R, Saha AK, et al. Hypothalamic fatty acid metabolism mediates the orexigenic action of ghrelin. *Cell Metab.* May 2008;7(5):389-399.

96. Figlewicz DP, Sipols AJ. Energy regulatory signals and food reward. *Pharmacol Biochem Behav.* Nov;97(1):15-24.

97. Sun Y, Asnicar M, Saha PK, Chan L, Smith RG. Ablation of ghrelin improves the diabetic but not obese phenotype of ob/ob mice. *Cell Metab.* May 2006;3(5):379-386.

98. Zigman JM, Nakano Y, Coppari R, et al. Mice lacking ghrelin receptors resist the development of diet-induced obesity. *Journal of Clinical Investigation.* 2005;115(12):3564.

99. Ahima RS, Antwi DA. Brain regulation of appetite and satiety. *Endocrinol Metab Clin North Am.* Dec 2008;37(4):811-823.

100. Cummings DE, Purnell JQ, Frayo RS, Schmidova K, Wisse BE, Weigle DS. A preprandial rise in plasma ghrelin levels suggests a role in meal initiation in humans. *Diabetes.* Aug 2001;50(8):1714-1719.

101. Tschop M, Weyer C, Tataranni PA, Devanarayan V, Ravussin E, Heiman ML. Circulating ghrelin levels are decreased in human obesity. *Diabetes.* Apr 2001;50(4):707-709.

102. Ukkola O. Ghrelin and metabolic disorders. *Curr Protein Pept Sci.* Feb 2009;10(1):2-7.

103. le Roux CW, Patterson M, Vincent RP, Hunt C, Ghatei MA, Bloom SR. Postprandial plasma ghrelin is suppressed proportional to meal calorie content in normal-weight but not obese subjects. *J Clin Endocrinol Metab.* Feb 2005;90(2):1068-1071.

104. Myers MG, Cowley MA, Munzberg H. Mechanisms of leptin action and leptin resistance. *Annu Rev Physiol.* 2008;70:537-556.

105. Rosenbaum M, Kissileff HR, Mayer LE, Hirsch J, Leibel RL. Energy intake in weight-reduced humans. *Brain Res.* Sep 2;1350:95-102.

106. Ahima RS, Saper CB, Flier JS, Elmquist JK. Leptin regulation of neuroendocrine systems. *Front Neuroendocrinol.* Jul 2000;21(3):263-307.

107. Gallagher EJ, Leroith D, Karnieli E. Insulin resistance in obesity as the underlying cause for the metabolic syndrome. *Mt Sinai J Med.* Sep-Oct;77(5):511-523.

108. Rosenbaum M, Leibel RL. Adaptive thermogenesis in humans. *Int J Obes (Lond).* Oct 2010;34 Suppl 1:S47-55.

109. Nelson KM, Weinsier RL, Long CL, Schutz Y. Prediction of resting energy expenditure from fat-free mass and fat mass. *Am J Clin Nutr.* Nov 1992;56(5):848-856.

110. Weinsier RL, Nelson KM, Hensrud DD, Darnell BE, Hunter GR, Schutz Y. Metabolic predictors of obesity. Contribution of resting energy expenditure, thermic effect of food, and fuel utilization to four-year weight gain of post-obese and never-obese women. *J Clin Invest.* Mar 1995;95(3):980-985.

111. Leibel RL, Rosenbaum M, Hirsch J. Changes in energy expenditure resulting from altered body weight. *N Engl J Med.* Mar 9 1995;332(10):621-628.

112. Weigle DS, Sande KJ, Iverius PH, Monsen ER, Brunzell JD. Weight loss leads to a marked decrease in nonresting energy expenditure in ambulatory human subjects. *Metabolism.* Oct 1988;37(10):930-936.

113. Astrup A, Gotzsche PC, van de Werken K, et al. Meta-analysis of resting metabolic rate in formerly obese subjects. *Am J Clin Nutr.* Jun 1999;69(6):1117-1122.

114. Amatruda JM, Statt MC, Welle SL. Total and resting energy expenditure in obese women reduced to ideal body weight. *J Clin Invest.* Sep 1993;92(3):1236-1242.

115. Weinsier RL, Hunter GR, Zuckerman PA, et al. Energy expenditure and free-living physical activity in black and white women: comparison before and after weight loss. *Am J Clin Nutr.* May 2000;71(5):1138-1146.

116. Rosenbaum M, Vandenborne K, Goldsmith R, et al. Effects of experimental weight perturbation on skeletal muscle work efficiency in human subjects. *Am J Physiol Regul Integr Comp Physiol.* Jul 2003;285(1):R183-192.

117. Goldsmith R, Joanisse DR, Gallagher D, et al. Effects of experimental weight perturbation on skeletal muscle work efficiency, fuel utilization, and biochemistry in human subjects. *Am J Physiol Regul Integr Comp Physiol.* Jan;298(1):R79-88.

118. Rosenson RS. New approaches in the intensive management of cardiovascular risk in the metabolic syndrome. *Curr Probl Cardiol.* May 2005;30(5):241-279.

119. Paraskevas KI, Karatzas G, Pantopoulou A, Iliopoulos DG, Perrea D. Targeting dyslipidemia in the metabolic syndrome: an update. *Curr Vasc Pharmacol.* Jul;8(4):450-463.

120. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). *JAMA.* May 16 2001;285(19):2486-2497.

121. Kakafika AI, Liberopoulos EN, Karagiannis A, Athyros VG, Mikhailidis DP. Dyslipidaemia, hypercoagulability and the metabolic syndrome. *Curr Vasc Pharmacol.* Jul 2006;4(3):175-183.

122. Reaven GM, Chen YD, Jeppesen J, Maheux P, Krauss RM. Insulin resistance and hyperinsulinemia in individuals with small, dense low density lipoprotein particles. *J Clin Invest.* Jul 1993;92(1):141-146.

123. Sorrentino MJ. Implications of the metabolic syndrome: the new epidemic. *Am J Cardiol.* Aug 22 2005;96(4A):3E-7E.

124. Schenk S, Saberi M, Olefsky JM. Insulin sensitivity: modulation by nutrients and inflammation. *J Clin Invest.* Sep 2008;118(9):2992-3002.

125. Zhuang XF, Zhao MM, Weng CL, Sun NL. Adipocytokines: a bridge connecting obesity and insulin resistance. *Med Hypotheses.* Dec 2009;73(6):981-985.

126. Assali AR, Ganor A, Beigel Y, Shafer Z, Hershcovici T, Fainaru M. Insulin resistance in obesity: body-weight or energy balance? *J Endocrinol.* Nov 2001;171(2):293-298.

127. Janssen I, Fortier A, Hudson R, Ross R. Effects of an energy-restrictive diet with or without exercise on abdominal fat, intermuscular fat, and metabolic risk factors in obese women. *Diabetes Care.* Mar 2002;25(3):431-438.

128. Tuomilehto J, Lindstrom J, Eriksson JG, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med.* May 3 2001;344(18):1343-1350.

129. Toledo FG, Menshikova EV, Azuma K, et al. Mitochondrial capacity in skeletal muscle is not stimulated by weight loss despite increases in insulin action and decreases in intramyocellular lipid content. *Diabetes.* Apr 2008;57(4):987-994.

130. Toledo FG, Menshikova EV, Ritov VB, et al. Effects of physical activity and weight loss on skeletal muscle mitochondria and relationship with glucose control in type 2 diabetes. *Diabetes.* Aug 2007;56(8):2142-2147.

131. Goodpaster BH, Katsiaras A, Kelley DE. Enhanced fat oxidation through physical activity is associated with improvements in insulin sensitivity in obesity. *Diabetes.* Sep 2003;52(9):2191-2197.

132. Zimmet P, Magliano D, Matsuzawa Y, Alberti G, Shaw J. The metabolic syndrome: a global public health problem and a new definition. *J Atheroscler Thromb.* 2005;12(6):295-300.

133. Daskalopoulou SS, Athyros VG, Kolovou GD, Anagnostopoulou KK, Mikhailidis DP. Definitions of metabolic syndrome: Where are we now? *Curr Vasc Pharmacol.* Jul 2006;4(3):185-197.

134. Ginsberg HN, Stalenhoef AF. The metabolic syndrome: targeting dyslipidaemia to reduce coronary risk. *J Cardiovasc Risk.* Apr 2003;10(2):121-128.

135. Gami AS, Witt BJ, Howard DE, et al. Metabolic syndrome and risk of incident cardiovascular events and death: a systematic review and meta-analysis of longitudinal studies. *J Am Coll Cardiol.* Jan 30 2007;49(4):403-414.

136. Corica F, Corsonello A, Lucchetti M, et al. Relationship between metabolic syndrome and platelet responsiveness to leptin in overweight and obese patients. *Int J Obes (Lond).* May 2007;31(5):842-849.

137. Banegas JR, Lopez-Garcia E, Graciani A, et al. Relationship between obesity, hypertension and diabetes, and health-related quality of life among the elderly. *Eur J Cardiovasc Prev Rehabil.* Jun 2007;14(3):456-462.

138. Ferraro KF, Su YP, Gretebeck RJ, Black DR, Badylak SF. Body mass index and disability in adulthood: a 20-year panel study. *Am J Public Health.* May 2002;92(5):834-840.

139. Ware JE. *SF-36 health survey: manual and interpretation guide*. Boston: Nimrod Press 1993.

140. Ware JE, Jr., Kosinski M, Bayliss MS, McHorney CA, Rogers WH, Raczek A. Comparison of methods for the scoring and statistical analysis of SF-36 health profile and summary measures: summary of results from the Medical Outcomes Study. *Med Care.* Apr 1995;33(4 Suppl):AS264-279.

141. Ware JE, Jr., Gandek B. Overview of the SF-36 Health Survey and the International Quality of Life Assessment (IQOLA) Project. *J Clin Epidemiol.* Nov 1998;51(11):903-912.

142. Samsa GP, Kolotkin RL, Williams GR, Nguyen MH, Mendel CM. Effect of moderate weight loss on health-related quality of life: an analysis of combined data from 4 randomized trials of sibutramine vs placebo. *Am J Manag Care.* Sep 2001;7(9):875-883.

143. Mannucci E, Petroni ML, Villanova N, Rotella CM, Apolone G, Marchesini G. Clinical and psychological correlates of health-related quality of life in obese patients. *Health Qual Life Outcomes.*8:90.

144. Kolotkin RL, Crosby RD, Williams GR. Health-related quality of life varies among obese subgroups. *Obes Res.* Aug 2002;10(8):748-756.

145. Fontaine KR, Barofsky I, Bartlett SJ, Franckowiak SC, Andersen RE. Weight loss and health-related quality of life: Results at 1-year follow-up. *Eating Behaviors.* 2004;5(1):85-88.

146. Westman EC, Feinman RD, Mavropoulos JC, et al. Low-carbohydrate nutrition and metabolism. *Am J Clin Nutr.* Aug 2007;86(2):276-284.

147. Keogh JB, Brinkworth GD, Noakes M, Belobrajdic DP, Buckley JD, Clifton PM. Effects of weight loss from a very-low-carbohydrate diet on endothelial function

and markers of cardiovascular disease risk in subjects with abdominal obesity. *Am J Clin Nutr.* Mar 2008;87(3):567-576.

148. Buscemi S, Verga S, Tranchina MR, Cottone S, Cerasola G. Effects of hypocaloric very-low-carbohydrate diet vs. Mediterranean diet on endothelial function in obese women*. *Eur J Clin Invest.* May 2009;39(5):339-347.

149. Volek J, Phinney S, Forsythe C, et al. Carbohydrate Restriction has a More Favorable Impact on the Metabolic Syndrome than a Low Fat Diet. *Lipids.* 2009;44(4):297-309.

150. Feinman RD, Volek JS. Low carbohydrate diets improve atherogenic dyslipidemia even in the absence of weight loss. *Nutr Metab (Lond).* 2006;3:24.

151. Wood RJ, Fernandez ML, Sharman MJ, et al. Effects of a carbohydrate-restricted diet with and without supplemental soluble fiber on plasma low-density lipoprotein cholesterol and other clinical markers of cardiovascular risk. *Metabolism.* 2007;56(1):58-67.

152. Walker C, Reamy BV. Diets for cardiovascular disease prevention: what is the evidence? *Am Fam Physician.* Apr 1 2009;79(7):571-578.

153. Hession M, Rolland C, Kulkarni U, Wise A, Broom J. Systematic review of randomized controlled trials of low-carbohydrate vs. low-fat/low-calorie diets in the management of obesity and its comorbidities. *Obesity Reviews.* 2009;10(1):36-50.

154. Noakes M, Keogh JB, Foster PR, Clifton PM. Effect of an energy-restricted, high-protein, low-fat diet relative to a conventional high-carbohydrate, low-fat diet on weight loss, body composition, nutritional status, and markers of cardiovascular health in obese women. *Am J Clin Nutr.* 2005;81:1298-1306.

155. Weigle DS, Breen PA, Matthys CC, et al. A high-protein diet induces sustained reductions in appetite, ad libitum caloric intake, and body weight despite compensatory changes in diurnal plasma leptin and ghrelin concentrations. *Am J Clin Nutr.* Jul 2005;82(1):41-48.

156. Astrup A. The satiating power of protein--a key to obesity prevention? *Am J Clin Nutr.* Jul 2005;82(1):1-2.

157. Halton TL, Hu FB. The effects of high protein diets on thermogenesis, satiety and weight loss: a critical review. *J Am Coll Nutr.* Oct 2004;23(5):373-385.

158. Westerterp-Plantenga MS. Protein intake and energy balance. *Regul Pept.* Aug 7 2008;149(1-3):67-69.

159. Johnston CS, Tjonn SL, Swan PD. High-protein, low-fat diets are effective for weight loss and favorably alter biomarkers in healthy adults. *J Nutr.* Mar 2004;134(3):586-591.

160. Lejeune MP, Westerterp KR, Adam TC, Luscombe-Marsh ND, Westerterp-Plantenga MS. Ghrelin and glucagon-like peptide 1 concentrations, 24-h satiety,

and energy and substrate metabolism during a high-protein diet and measured in a respiration chamber. *Am J Clin Nutr.* Jan 2006;83(1):89-94.

161. Diepvens K, HÃ¤berer D, Westerterp-Plantenga M. Different proteins and biopeptides differently affect satiety and anorexigenic/orexigenic hormones in healthy humans. *International Journal Of Obesity (2005).* 2008;32(3):510-518.

162. Batterham RL, Heffron H, Kapoor S, et al. Critical role for peptide YY in protein-mediated satiation and body-weight regulation. *Cell Metab.* Sep 2006;4(3):223-233.

163. Smeets A, Soenen S, Luscombe N, Westerterp-Plantenga M. Energy expenditure, plasma ghrelin, GLP-1, PYY concentrations, and satiety following a single high-protein lunch. *Appetite.* 2010;51:401.

164. van Loon LJ, Saris WH, Verhagen H, Wagenmakers AJ. Plasma insulin responses after ingestion of different amino acid or protein mixtures with carbohydrate. *Am J Clin Nutr.* Jul 2000;72(1):96-105.

165. Nair KS, Halliday D, Garrow JS. Thermic response to isoenergetic protein, carbohydrate or fat meals in lean and obese subjects. *Clin Sci (Lond).* Sep 1983;65(3):307-312.

166. Westerterp-Plantenga MS, Rolland V, Wilson SA, Westerterp KR. Satiety related to 24 h diet-induced thermogenesis during high protein/carbohydrate vs high fat

diets measured in a respiration chamber. *Eur J Clin Nutr.* Jun 1999;53(6):495-502.

167. Jequier E. Nutrient effects: post-absorptive interactions. *Proc Nutr Soc.* Mar 1995;54(1):253-265.

168. Raben A, Agerholm-Larsen L, Flint A, Holst JJ, Astrup A. Meals with similar energy densities but rich in protein, fat, carbohydrate, or alcohol have different effects on energy expenditure and substrate metabolism but not on appetite and energy intake. *Am J Clin Nutr.* Jan 2003;77(1):91-100.

169. Livesey G. A perspective on food energy standards for nutrition labelling. *Br J Nutr.* Mar 2001;85(3):271-287.

170. Westerterp KR, Wilson SA, Rolland V. Diet induced thermogenesis measured over 24h in a respiration chamber: effect of diet composition. *Int J Obes Relat Metab Disord.* Mar 1999;23(3):287-292.

171. Tentolouris N, Pavlatos S, Kokkinos A, Perrea D, Pagoni S, Katsilambros N. Diet-induced thermogenesis and substrate oxidation are not different between lean and obese women after two different isocaloric meals, one rich in protein and one rich in fat. *Metabolism.* Mar 2008;57(3):313-320.

172. Hochstenbach-Waelen A, Veldhorst MA, Nieuwenhuizen AG, Westerterp-Plantenga MS, Westerterp KR. Comparison of 2 diets with either 25% or 10% of

energy as casein on energy expenditure, substrate balance, and appetite profile. *Am J Clin Nutr.* Mar 2009;89(3):831-838.

173. Saris WH, Blair SN, van Baak MA, et al. How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. *Obes Rev.* May 2003;4(2):101-114.

174. Curves. *Curves Food & Exercise Diary* Woodway, TX: Curves International, Inc.; 2009.

175. Ainsworth BE, Bassett DR, Jr., Strath SJ, et al. Comparison of three methods for measuring the time spent in physical activity. *Med Sci Sports Exerc.* Sep 2000;32(9 Suppl):S457-464.

176. Ainsworth BE, Macera CA, Jones DA, et al. Comparison of the 2001 BRFSS and the IPAQ Physical Activity Questionnaires. *Med Sci Sports Exerc.* Sep 2006;38(9):1584-1592.

177. Booth M. Assessment of physical activity: an international perspective. *Res Q Exerc Sport.* Jun 2000;71(2 Suppl):S114-120.

178. Craig CL, Marshall AL, Sjostrom M, et al. International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc.* Aug 2003;35(8):1381-1395.

179. ACSM. *ACSM's Guildelines for Exercise Testing and Prescription*. 8th ed. Philadelphia, PA: Lippincott William & Wilkins; 2010.

180. Matarese LE. Indirect calorimetry: technical aspects. *J Am Diet Assoc.* Oct 1997;97(10 Suppl 2):S154-160.

181. ACSM. *ACSM's Guildelines for Exercise Testing and Prescription*. 7th ed. Philadelphia, PA: Lippincott William & Wilkins; 2006.

182. Pickering TG, Hall JE, Appel LJ, et al. Recommendations for blood pressure measurement in humans and experimental animals: Part 1: blood pressure measurement in humans: a statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Hypertension.* Jan 2005;45(1):142-161.

183. Glickman SG, Marn CS, Supiano MA, Dengel DR. Validity and reliability of dual-energy X-ray absorptiometry for the assessment of abdominal adiposity. *J Appl Physiol.* Aug 2004;97(2):509-514.

184. Kohrt WM. Preliminary evidence that DEXA provides an accurate assessment of body composition. *J Appl Physiol.* Jan 1998;84(1):372-377.

185. Mazess RB, Barden HS, Bisek JP, Hanson J. Dual-energy x-ray absorptiometry for total-body and regional bone-mineral and soft-tissue composition. *Am J Clin Nutr.* Jun 1990;51(6):1106-1112.

186. Svendsen OL, Haarbo J, Hassager C, Christiansen C. Accuracy of measurements of body composition by dual-energy x-ray absorptiometry in vivo. *Am J Clin Nutr.* May 1993;57(5):605-608.

187. Almada AL, Kreider RB. Comparison of the reliability of repeated whole body DEXA scans to repeated spine and hip scans. *J. Bone Miner Res.* 1999;14:S369.

188. McAuley KA, Williams SM, Mann JI, et al. Diagnosing insulin resistance in the general population. *Diabetes Care.* Mar 2001;24(3):460-464.

189. Svendsen OL, Hassager C, Christiansen C. Effect of an energy-restrictive diet, with or without exercise, on lean tissue mass, resting metabolic rate, cardiovascular risk factors, and bone in overweight postmenopausal women. *Am J Med.* Aug 1993;95(2):131-140.

190. Fielding RA, Frontera WR, Hughes VA, Fisher EC, Evans WJ. The reproducibility of the Bruce protocol exercise test for the determination of aerobic capacity in older women. *Med Sci Sports Exerc.* Aug 1997;29(8):1109-1113.

191. Baechle T, Earle R. *Essentials of Strength Training and Conditioning*. Champaign, IL: Human Kinetics; 2005.

192. Wilborn CD, Kerksick CM, Campbell BI, et al. Effects of Zinc Magnesium Aspartate (ZMA) Supplementation on Training Adaptations and Markers of Anabolism and Catabolism. *J Int Soc Sports Nutr.* 2004;1(2):12-20.

193. Kosinski M, Keller SD, Hatoum HT, Kong SX, Ware JE, Jr. The SF-36 Health Survey as a generic outcome measure in clinical trials of patients with osteoarthritis and rheumatoid arthritis: tests of data quality, scaling assumptions and score reliability. *Med Care.* May 1999;37(5 Suppl):MS10-22.

194. ACSM. *ACSM's Guildelines for Exercise Testing and Presription*. 6th ed. Philadelphia, PA: Lippincott William & Wilkins; 2000.

195. Rock CL, Flatt SW, Sherwood NE, Karanja N, Pakiz B, Thomson CA. Effect of a free prepared meal and incentivized weight loss program on weight loss and weight loss maintenance in obese and overweight women: a randomized controlled trial. *JAMA.* Oct 27 2010;304(16):1803-1810.

196. Wing RR. Treatment options for obesity: do commercial weight loss programs have a role? *JAMA.* Oct 27 2010;304(16):1837-1838.

197. Kreider RB, Serra M, Beavers KM, et al. A structured diet and exercise program promotes favorable changes in weight loss, body composition, and weight maintenance. *J Am Diet Assoc.* Jun;111(6):828-843.

198. Dansinger ML, Gleason JA, Griffith JL, Selker HP, Schaefer EJ. Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. *JAMA.* Jan 5 2005;293(1):43-53.

199. Jebb SA, Ahern AL, Olson AD, et al. Primary care referral to a commercial provider for weight loss treatment versus standard care: a randomised controlled trial. *Lancet.* Oct 22;378(9801):1485-1492.

200. Bellisle F, Dalix AM, De Assis MA, et al. Motivational effects of 12-week moderately restrictive diets with or without special attention to the Glycaemic Index of foods. *Br J Nutr.* Apr 2007;97(4):790-798.

APPENDIX A

CONSENT FORM

CONSENT FORM
Comparison of Two Diet Approaches on Weight Loss and Health Outcomes in Women II

Introduction

The purpose of this form is to provide you information that may affect your decision as to whether or not to participate in this research study. If you decide to participate in this study, this form will also be used to record your consent.

You have been asked to participate in a research project studying the Curves International fitness and weight loss program and the Weight Watchers program. The purpose of this study is to determine whether following a Curves high protein/low fat diet for 30-days several times during 16 weeks of participating in the Curves fitness program promotes stepwise reductions in body weight, improvements in body composition, and/or improvements in markers of fitness and health better than following the Weight Watchers Momentum Program for the same time period. You were selected to be a possible participant because you met all entrance criteria for this study. This study is being sponsored/funded by Curves International.

What will I be asked to do?

If you agree to participate in this study, you will first be asked to sign an Informed Consent statement in compliance with the Human Subject's Protection Program (HSPP) at Texas A&M University and the American College of Sports Medicine. You will then be familiarized to the study requirements, food log recording and tests to be conducted during the study. This session will take approximately one hour to complete. Prior to reporting to the lab for baseline testing, you will record all food that you eat on dietary record forms for four days (including one weekend day). You will not exercise for 48 hours nor eat for 12 hours prior to reporting to the lab for baseline testing. You will then undergo a battery of tests as described in Table 1. You will fill out a Demographic Form, a Health History Form, A Radiation Safety Form, a Quality of Life Questionnaire, a Body Image Questionnaire and an eating Satisfaction Questionnaire. You will also be required to report any adverse side effects that you may experience on a weekly basis. .

You will then continue with the tests as described in Table 1. You will first be weighed and have your resting energy expenditure (REE) determined. This will involve lying down on an exam table and having a light blanket placed over you to keep you warm and placing ear plugs in your ears to reduce distractions. A see through plastic canopy will then be placed over your neck and head so that the air that you breathe can be measured for oxygen and carbon dioxide. You should stay motionless without going to sleep for 15-minutes so that your resting energy expenditure can be calculated. You will then donate about 20 milliliters (4 teaspoons) of venous blood from a vein in your arm. Blood samples will be obtained by standard/sterile procedures using a needle inserted into a vein in your arm. Personnel who will be taking your blood are experienced in phlebotomy

(procedures to take blood samples) and are qualified to do so under guidelines established by the Texas Department of Health and Human Services. This will take about 5-minutes. You will then have your total body water determined using a bioelectrical impedance analyzer (BIA). The BIA analysis will involve lying down on your back on a table and having two small electrodes placed on your right hand and your right foot. The analyzer wires will be attached and a small and safe current (500 micro-amps at a frequency of 5- kHz) will pass through your body so that the amount of water can be measured. This analyzer is commercially available and has been used in the health care/fitness industry as a means to assess body composition and body water for over 20 years. The use of this device has been approved by the Food and Drug Administration (FDA) to assess total body water and the current to be used has been deemed safe. Your body composition and bone density will then be determined by using a Discovery W dual energy x-ray absorptiometer (DEXA). This will involve lying down on your back on the DEXA exam table in a pair of shorts or a gown for about 6 minutes. A low dose of radiation will scan your entire body to determine the amount of fat weight, muscle weight, and bone weight. You will be exposed to an x-ray dose that is similar to the amount of natural background radiation a person would receive in one month while living in College Station. After this test, you will have resting blood pressure determined using a blood pressure cuff and stethoscope and heart rate determined by taking your pulse. You will then be prepared to perform a maximal treadmill test. You will have your right and left shoulder, right and left part of your stomach, and several places around your upper chest and below your bra line rubbed with alcohol gauze. Ten (10) electrocardiograph (ECG) electrodes will then be placed on your shoulders, chest, and stomach and you will be attached to an ECG to evaluate your heart. You will then be positioned on the treadmill and a sterile mouthpiece will be placed in your mouth and a mouthpiece holder will be placed on your head. A nose clip will be placed on your nose and that the air you breathe will be measured for oxygen and carbon dioxide content. Once the equipment is attached, you will be given instructions to begin walking on the treadmill. You will then perform an exercise test that involves increasing the speed and grade you are walking on the treadmill until you reach your maximal effort. Heart rate, ECG tracings, blood pressure and your ratings of exertion will be monitored throughout the test. Once you reach your maximum, you will undergo a slow walking and seated recovery period. This test will take about 30 minutes to complete. You will then perform a one repetition maximum (1RM) and 80% of 1RM endurance repetition test on the bench press and hip/leg sled using standard procedures. This will involve warming up and performing successive one repetition lifts on the bench press until you determine your 1 RM. You will then rest for 5-minutes and lift 80% of your 1 RM as many times as you can. You will then rest for 10-minutes and follow the same procedure in determining your 1 RM and 80% of 1 RM on the hip/leg sled. These tests will take about 20 minutes to complete. The same battery of tests will be performed at the post-study assessment 16 weeks into the study protocol. All the assessments minus the exercise tests will also be performed at 4, 6 10 and 12 weeks into the study protocol. Each testing session will take between 1.5 and 3 hours to complete. In the event of an emergency during an exercise test proper emergency response protocols (calling 9-911 for serious injury or a medical emergency, calling Biosafety/EHS for cleanup assistance or spill team response, calling UPD for incidents in public areas, retrieving AED located in the lab, performing CPR or other First Aid techniques, etc.) will be followed by the Exercise & Sport Nutrition Laboratory (ESNL) Staff depending on the severity of the emergency.

After baseline testing, you will be matched based on age and BMI and randomized into one of three intervention groups as described in Table 2. The first group (n=40) will follow the Curves high protein/low fat diet group (30% CHO, 45% PRO, 25% FAT) for 7-days at 1,200 kcals/day and then 1,500 kcals/day for 21-days. If you are assigned to this group you will then consume a 2,200 kcal/day diet consisting of 45% CHO, 30% PRO, 25% FAT for 14-days during a metabolic recovery and weight maintenance phase. You will diet for 2-days at 1,200 kcals/day only if you gain 3 pounds of weight during the maintenance phase. You will then repeat this entire cycle twice and then end with the 30-day weight loss and weight maintenance intervention to determine if this is an effective step-wise weight loss plan. If you are randomized into the second group (n=40) you will follow the Weight Watchers Momentum Program that is based on their four pillar approach (food, exercise, behavior and support). Every food has a POINTS value, based on its calories, fat and fiber. The Momentum program uses POINTS values to help keep track of what you eat. A POINTS "budget" will be personalized for you at the weekly meetings. You will be required to attend at least one meeting per week at the local Weight Watchers facility located at 4001 E. 29th Street, Suite 112 in the Carter Creek Center in Bryan, Texas. Membership dues/passes to the Weight Watchers program/facility will be covered for you during the duration of the study. The third group (n=40) will act as a control group. If you are assigned to this group you will not follow a prescribed nutrition program but will continue with your normal daily habits. Everyone, regardless of group assignment, will keep a food record and food frequency log to monitor dietary compliance.

If you are randomized to participate in the first group (n=40) you will participate in the Curves 30-minute fitness program three times per week throughout the investigation. The Curves program involves performing thirteen hydraulic resistance exercise machines that utilize bidirectional resistance that work all major muscle groups. These are interspersed with floor-based calisthenics exercises designed to maintain an elevated heart rate. Research has shown that exercise intensity averages 65% of maximal aerobic capacity and that participants generally perform 50 – 75% of 1 repetition maximum on the main exercise machines. The new Curves equipment includes the attached force measurement and feedback system. You will be instructed to push hard enough to generate a green light on the feedback panel for each repetition. All exercise sessions will be held in the ESNL. Research Assistants will monitor your exercise sessions and record your attendance. You will also be encouraged to walk for 30-minutes at a brisk pace (60 – 80% of heart rate reserve) on days they do use the Curves equipment. If you are randomized to participate in second group (n=40) you will start by focusing on the food plan and then incorporate the specifics of activity a week alter once you have had the chance to get conformable with the eating plan. After a week of reducing sedentary behavior, the POINTS Activity System is introduced. In a way that complements the POINTS values of food, a formula that calculates the POINTS values for activity is used. The formula is based on body weight, the amount of time the activity is done, and the level of intensity. This method enables you to do any exercise or activity that is enjoyable and fits within your lifestyle. If you are assigned to the third group (n=40) you will not follow a prescribed exercise program but will continue your normal daily habits. Everyone, regardless of group assignment, will be required to complete activity logs to monitor exercise frequency and intensity.

Please do your best to: 1) follow the instructions outline by the investigators; 2) show up to all scheduled testing and training sessions; and 3) follow the diet prescribed and do not take any other nutritional supplements or performance enhancing aids during this study (i.e., vitamins/minerals, creatine, HMB, androstenedione, DHEA, etc). In addition, please do not take any non-medically prescribed medications and report any medication that is prescribed for you to take during this study. If you take any other nutritional supplements or medications during the course of the study that may affect vitamin/mineral status, body composition, or strength you may be removed from the study.

What are the risks involved in this study?

The risks associated with this study are: You will be exposed to a low level of radiation during the DEXA body composition tests, which is similar to the amount of natural background radiation you would receive in one month while living in College Station. In addition, a very low level of electrical current will be passed through your body using a bioelectrical impedance analyzer (BIA). This analyzer is commercially available and has been used in the health care/fitness industry as a means to assess body composition and body water for over 20 years. The use of the BIA and DEXA analyzers have been shown to be safe methods of assessing body composition and total body water and are approved by the FDA. You will donate about 4 teaspoons (20 milliliters) of venous blood six (6) times during the study using standard phlebotomy procedures. This procedure may cause a small amount of pain when the needle is inserted into the vein as well as some bleeding and bruising. You may also experience some dizziness, nausea, and/or faint if you are unaccustomed to having blood drawn. The exercise tests that will be performed may cause symptoms of fatigue, shortness of breath, and/or muscular fatigue/discomfort. The exercise tests may also cause short-term muscle soreness and moderate fatigue for several days following the tests. You may also experience muscle strains/pulls during the exercise testing and/or training program. However, exercise sessions will be conducted by trained personnel and monitored to ensure you follow appropriate exercise guidelines. You will follow a prescribed dietary regimen involving consuming 1,200, 1,500 or 2,200 calories per day during various phases of the program. In addition, one group will ingest a high percentage of calories in the form of protein. Although the total amount of total protein is not excessive (100-220 grams/day or 1.1 - 2.3 grams/kg/day for a 95 kg female) it may be higher than you are accustomed to ingesting and may exceed recommended protein intake for active individuals (i.e., 1-2 grams/kg/day). As a result, you may experience weight loss or gain, feelings of hunger or fullness, and/or changes in appetite and/or mood during various phases of the dietary intervention.

What are the possible benefits of this study?

The possible benefit you may receive from participation in this study is increased physical fitness and improvements in body composition. You may also gain insight about your health and fitness status from the assessments that will be performed.

Do I have to participate?

No. Your participation is voluntary. You may decide not to participate or to withdraw at any time without your current or future relations with Texas A&M University being affected.

Will I be compensated?

You will receive $175 (i.e., $25 for each familiarization and experimental session) upon completion of the study. Disbursement will occur upon completion of all sessions and after all study related materials (food logs, training logs, etc.) are turned in. Participants who discontinue, or are dropped prior to completion will be compensated on a pro-rated basis depending on the total number of sessions completed (i.e., $25 for each familiarization and experimental session). Participants will be dropped if they are non-compliant with any portion of the study requirements at the discretion of the Principal Investigator, Dr. Richard Kreider.

Who will know about my participation in this research study?

The records of this study will be kept private. No identifiers linking you to this study will be included in any sort of report that might be published. Research records will be stored securely and only Mr. Christopher Rasmussen and Dr. Richard Kreider will have access to the records.

Whom do I contact with questions about the research?

If you have questions regarding this study, you may contact Dr. Richard Kreider, 945-1333, rkreider@hlkn.tamu.edu or Mr. Christopher Rasmussen, crasmussen@hlkn.tamu.edu.

Whom do I contact about my rights as a research participant?

This research study has been reviewed by the Human Subjects' Protection Program and/or the Institutional Review Board at Texas A&M University. For research-related problems or questions regarding your rights as a research participant, you can contact these offices at (979)458-4067 or irb@tamu.edu.

Signature

Please be sure you have read the above information, asked questions and received answers to your satisfaction. You will be given a copy of the consent form for your records. By signing this document, you consent to participate in this study.

Signature of Participant: _____ Date: _____

Printed Name:_____

Signature of Person Obtaining Consent: _____ Date: _____

Printed Name: _____

APPENDIX B

INFORMATION SHEET

INFORMATION SHEET
Comparison of Two diet Approaches on Weight Loss and Health Outcomes in Women II

Introduction

The purpose of this form is to provide you (as a prospective research study participant) information that may affect your decision as to whether or not to participate in this research.

You have been asked to participate in a research study involving the Curves International fitness and weight loss program and the popular Weight Watchers program. The purpose of this study is to compare the Curves fitness and weight loss program to the Weight Watchers Momentum Program.

What will I be asked to do?

If you agree to participate in this study, you will be asked to complete each of the following:

- Read and sign an Informed Consent statement
- Attend a Familiarization session detailing the requirements of the study
- Complete a series of 6 forms/questionnaires
- Complete a body weight assessment
- Complete a resting energy expenditure (REE) assessment
- Donate approximately 20 milliliters (4 teaspoons) of venous blood
- Complete a total body water assessment using a bioelectrical impedance analyzer (BIA)
- Complete a body composition/ bone density assessment using a dual energy x-ray absorbtiometer (DEXA)
- Complete a resting blood pressure (BP) assessment
- Complete a resting heart rate (HR) assessment
- Complete a graded exercise test (GXT)
- Complete a 1 repetition maximum (1RM) & 80% of 1RM test on the bench press and leg press
- Complete an exercise and diet intervention program lasting a total of 16 weeks
- Call a Research Assistant on a weekly basis to report progress in the study
- Complete some or all of these same testing procedures following 4, 6, 10, 12 and 16 weeks of training and dietary intervention as described in Table 1

What are the risks involved in this study?

The possible risks associated with this study are:

- Exposure to a low level of radiation during the DEXA body composition assessment
- Exposure to a very low level of electrical current during the BIA assessment

- Possible pain, bleeding, bruising, and/or dizziness or nausea during the phlebotomy procedure
- Possible fatigue, shortness of breath, and/or muscular fatigue during the exercise tests
- Possible weight loss/gain, feelings of hunger/fullness and/or changes in appetite and/or mood during different phases of the dietary intervention

What are the possible benefits of this study?

The possible benefits associated with this study are:

- Increased physical fitness and improvements in body composition
- Insight into personal health and fitness status from the assessments performed

Do I have to participate?

No. Your participation is voluntary. You may decide not to participate or to withdraw at any time without your current or future relations with Texas A&M University being affected.

Will I be compensated?

You will receive $275 (i.e., $25 for each familiarization and experimental session). Disbursement will occur upon completion of all sessions and after all study related material (food logs, training logs, etc.) are turned in.

Who will know about my participation in this research study?

The records of this study will be kept private. No identifiers linking you to this study will be included in any sort of report that might be published. Research records will be stored securely and only Mr. Christopher Rasmussen and Dr. Richard Kreider will have access to these records.

Whom do I contact with questions about the research?

If you have questions regarding this study, you may contact Dr. Richard Kreider, 945-1333, rkreider@hlkn.tamu.edu or Mr. Christopher Rasmussen, crasmussen@hlkn.tamu.edu.

Whom do I contact about my rights as a research participant?

This research study has been reviewed by the Human Subjects' Protection Program and/or the Institutional Review Board at Texas A&M University. For research-related problems or questions regarding your rights as a research participant, you can contact these offices at (979)458-4067 or irb@tamu.edu.

Participation

Please be sure you have read the above information, asked questions and received answers to your satisfaction. If you would like to be in the study you will now be scheduled for a familiarization session.

APPENDIX C

MEDICAL HISTORY QUESTIONNAIRE

Texas A&M UNIVERSITY

EXERCISE & SPORT NUTRITION LABORATORY

Medical History Inventory

Directions. The purpose of this questionnaire is to enable the staff of the Exercise and Sport Sciences Laboratory to evaluate your health and fitness status. Please answer the following questions to the best of your knowledge. All information given is **CONFIDENTIAL** as described in the **Informed Consent Statement.**

Name:_____ Age _____
Date of Birth_____

Name and Address of Your Physician:_____
_____

MEDICAL HISTORY

Do you have or have you ever had any of the following conditions? (Please write the date when you had the condition in the blank).

____Heart murmur, clicks, other cardiac findings?
____Frequent extra, skipped, or rapid heartbeats?
____Chest Pain of Angina (with or without exertion)?
____High cholesterol?
____Heart attack or any cardiac surgery?
____Leg cramps (during exercise)?
____Chronic swollen ankles?
____Varicose veins?
____Frequent dizziness/fainting?
____Muscle or joint problems?
____High blood sugar/diabetes?
____Thyroid Disease?
____Low testosterone/hypogonadism?
____Glaucoma?
____ Asthma/breathing difficulty?
____Bronchitis/Chest Cold?
____Cancer, Melanoma, or Suspected Skin Lesions
____ Emphysema/lung disease
____ Epilepsy/seizures?
____ Rheumatic fever?
____ Scarlet fever?
____ Ulcers?
____ Pneumonia?
____ Anemias?
____ Liver or kidney disease
____ Autoimmune disease?
____ Nerve disease?
____ Psychological Disorders?

Do you have or have you been diagnosed with any other medical condition not listed?
_____
_____
_____

Please provide any additional comments/explanations of your current or past medical history.

_____
_____
_____

Please list any recent surgery (i.e., type, dates etc.).

_____
_____
_____

List all prescribed/non-prescription medications and nutritional supplements you have taken in the last 3 months.

_____
_____

What was the date of your last complete medical exam?

_____
_____

Do you know of any medical problem that might make it dangerous or unwise for you to participate in this study? (including strength and maximal exercise tests) ____ If yes, please expain._____
_____

Recommendation for Participation (for ESNL use only):

____ No exclusion criteria presented. Subject is ***cleared*** to participate in the study.
____ Exclusion criteria is/are present. Subject is ***not cleared*** to participate in the study.

Signed: _____ Date: _____

APPENDIX D

PERSONAL INFORMATION WORKSHEET

Texas A&M University
EXERCISE & SPORT NUTRITION LABORATORY

Personal Information

Name:
_____

Address:
_____

City: _____ State: _____ Zip Code _____

Home Phone: (___) _____ Work Phone: (___) _____

Beeper: (___) _____ Cell Phone: (___) _____

Fax: (___) _____ E-mail address:_____

Birth date: _____ / _____ / _____ Age: _____ Height: _____Weight: _____

Exercise History/Activity Questionnaire

1. Describe your typical occupational activities.

2. Describe your typical recreational activities

3. Describe any exercise training that you routinely participate.

4. How many days per week do you exercise/participate in these activities?

5. How many hours per week do you train?

6. How long (years/months) have you been consistently training?

APPENDIX E

DATA COLLECTION FORM

Texas A&M University: Exercise & Sport Nutrition Laboratory
Trial: Comparison of Two Diet Approaches on Weight Loss and Health Outcomes in Women II

Demographics ESNL Staff Initials: _____
Name: _____Testing Session: _____ Group: _____
Date: _____ D.O.B.: _____ Age: _____

Resting Measures ESNL Staff Initials: _____

Psychological Questionnaires/Informed Consent:

HLKN Informed consent: _____ Body Image: _____
Radiation consent: _____ MOS SF-36: ____
Activity Log: _____ Eating Satisfaction: _____
Food Log: _____ Post-Study Questionnaire (T6 only): _____

Physiological Parameters: ESNL Staff Initials: _____

Height: _____ in. Waist: _____in.
Weight: _____ lb. Hip: _____in.
REE #1: _____ Resting H.R.: _____bpm.
Time: _____am Resting B.P.:_____/_____mmHg
Last Meal: _____am/pm BIA: _____
Hrs Fasted: _____hr. Handheld BIA: _____ #1 or #2
Last Wkout: _____ DEXA #2: _____
Lab (EBNL): _____ (2) SST Tubes/ (1) EDTA ECG (Rest): _____ #1 or #2
Time: _____am Max Test: _____ #1 or #2
Yr. /Menopause _____ Notes: _____

Exercise Measures: Strength Testing: ESNL Staff Initials: ____

Leg Press: Foot Position: _____ Sled Position: _____
Preceding Weights/Reps:
_____x_____: _____x_____: _____x_____: _____x_____: _____x_____
1 RM: _____ 80% 1RM: _____ 80% 1RM repetitions: _____

Bench Press: Hand Position: _____
Preceding Weights/Reps:
_____x_____: _____x_____: _____x_____: _____x_____: _____x_____
1 RM: _____ 80% 1RM: _____ 80% 1RM repetitions: _____