

Body Composition and Physical Activity Maintenance One Year  
After a 12-Week Exercise Intervention in Women

by

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## ABSTRACT

**Purpose:** Exercise interventions often result in less than predicted weight loss or even weight gain in some individuals, with over half of the weight lost usually regained within one year. The current study hypothesized that one year following a 12-week supervised exercise intervention, women who continued to exercise regularly but initially gained weight would lose the weight gained reverting back to baseline, or continue to lose weight if weight was initially lost. Conversely, those who discontinued purposeful exercise at the conclusion of the study were expected to continue to gain or regain weight.

**Methods:** 24 women who completed the initial 12-week exercise intervention (90min/week of supervised treadmill walking at 70%  $\text{VO}_{2\text{peak}}$ ) participated in a follow-up study one year after the conclusion of the exercise intervention. Subjects underwent Dual-energy X-Ray Absorptiometry at baseline, 12-weeks, and 15 months, and filled out physical activity questionnaires at 15 months.

**Results:** A considerable amount of heterogeneity was observed in body weight and fat mass changes among subjects, but there was no significant overall change in weight or fat mass from baseline to follow-up. 15 women were categorized as compensators and as a group gained weight ( $+0.94 \pm 3.26\text{kg}$ ) and fat mass ( $+0.22 \pm 3.25\text{kg}$ ) compared to the nine non-compensators who lost body weight ( $-0.26 \pm 3.59\text{kg}$ ) and had essentially no change in fat mass ( $+0.01 \pm 2.61\text{kg}$ ) from 12-weeks to follow-up. There was a significant between group difference ( $p=.003$ ) in change in fat mass from 12-weeks to follow-up between subjects who continued regular vigorous exercise ( $-2.205 \pm 3.070\text{kg}$ ), and those who did not ( $+1.320 \pm 2.156\text{kg}$ ). Additionally, energy compensation from

baseline to 12-weeks and early body weight and composition changes during the intervention were moderate predictors of body weight and composition changes from baseline to follow-up.

Conclusion: The main finding of this study is that following a 12-week supervised exercise intervention, women displayed a net loss of fat mass during the follow-up period if regular vigorous exercise was continued, regardless of whether they were classified as compensators or non-compensators during the initial intervention.

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## CHAPTER 1

### INTRODUCTION

According to data from the 2007-2008 National Health and Nutrition Examination Survey (NHANES), 33.8% of adult Americans are obese, having a Body Mass Index (BMI) of greater than  $30\text{kg/m}^2$ . An additional 34.2% are considered overweight having a BMI of  $25\text{--}29.9\text{kg/m}^2$ . Based on these data, and alarming 68% of American adults are either overweight or obese, with less than one third of the adult population being considered “normal” weight (Flegal, Carroll, Kit, & Ogden, 2012).

BMI has been used as a tool to assess disease risk. A meta-analysis by Guh and colleagues showed evidence for the association of being classified as either overweight or obese and 18 different diseases, conditions, and co-morbidities including: Type II Diabetes Mellitus, multiple types of cancer, cardiovascular diseases including hypertension, Coronary Artery Disease, pulmonary embolism, and stroke, as well as asthma, gallbladder disease, osteoarthritis, and chronic back pain (Guh et al., 2009). However, because overweight and obese people tend to be less physically active than non-overweight and obese persons, and low physical activity level is also a strong predictor of many chronic diseases, the association between high BMI and many chronic diseases may not be causal (Wei et al., 1999).

The collective research indicates that maintenance of a healthy body weight, avoidance of excessive weight gain, and engaging in regular physical activity are important for maintaining physical health. With regard to body weight, multiple national organizations have designated goals and recommendations for reducing both adult and adolescent obesity in America. Healthy People 2020 set goals to try and decrease the

percentage of the population that is considered overweight and obese, and to increase the percentage of the population with a healthy weight (United States Department of Health and Human Services [HHS], Healthy People 2020, 2013). The National Heart, Lung, and Blood Institute has suggested that weight loss as little as five to ten percent of initial body weight may be sufficient to decrease the risk of Type II Diabetes Mellitus and heart disease despite whether or not the person was still classified as obese or overweight based on BMI after this recommended weight loss (National Heart, Lung, and Blood Institute, 2004). Additionally, many risk factors for chronic diseases can be improved or entirely resolved with regular physical activity independent of weight loss (King, Hopkins, Caudwell, Stubbs, & Blundell, 2009).

The primary prevention of obesity related diseases can be achieved when individuals who are not yet overweight successfully achieve weight maintenance by avoiding excessive weight gain. However, weight maintenance is not as simple as it may seem. People typically gain body weight throughout their adult years, with body weight usually peaking in the sixth decade of life for women. As age progresses, muscle mass is lost while fat mass typically continues to increase from the high school years through the end of life (Spirduso, 2010). For example, in a study by Donnelly et al., a non-exercising control group of women who were instructed to make no changes to their diet or physical activity levels gained 2.9kg, or almost six and a half pounds during the 16-month study (Donnelly et al., 2003).

In order to achieve weight maintenance and maintain a healthy weight, proper energy balance must be achieved. Energy balance is equal to energy intake (EI) minus energy expenditure (EE). EI are kilocalories (kcal) consumed from food and beverages.



EE consists of kcals burned by exercise, daily activities, and basal metabolism. Energy balance, and therefore weight maintenance, can be achieved when EE and EI values are equal (R. Stubbs, et al., 2002).

In order to achieve weight loss, a negative energy balance must be produced. A negative energy balance occurs when EE exceeds EI, creating a kilocalorie deficit. A negative energy balance can be produced when EI is decreased with dieting or caloric restriction, when EE is increased through purposeful exercise and physical activity, or a combination of the above methods (R. Stubbs, et al., 2002).

Purposeful exercise in particular has the ability to produce a significant negative energy balance, and has therefore been recognized as an important component of weight loss. The American College of Sports Medicine (ACSM) and multiple other health organizations have recommended 30 minutes of moderate intensity physical activity on most days of the week to improve health related outcomes (Garber et al., 2011), however significantly more physical activity may be necessary to maintain or lose body weight. It has been reported that 150 to 250 minutes per week of moderate-intensity exercise may prevent weight gain in most people or even produce modest weight loss in some. However, it has been suggested that significant weight loss may require in excess of 250 minutes per week of moderate intensity exercise (Donnelly et al., 2009).

It is sometimes recommended that in order to lose one pound of fat, a person must create a 3500 kcal energy deficit through diet and or exercise. Likewise, it is said that if a person consumes an excess 3500 kcal they will gain one pound of fat. However, a twin study in which monozygotic twins were confined and supervised throughout the study, demonstrated that the relationship may not be so simple. The participants were over-fed

1000 kcal per day for 84 days and not allowed to exercise. According to the equation, all participants were expected to gain 24 pounds. However, actual weight gain ranged from a gain of 9.5 to 29.2 pounds, with an average weight gain of 17.8 pounds. These results demonstrate significant heterogeneity in body weight in response to over-feeding. Additionally, the pairs of twins responded three times more similarly than non-twin individuals suggesting a strong genetic component as to how much of the excess kilocalories were stored as fat (Bouchard et al., 1990).

Similarly to the above study which demonstrated a heterogeneity of weight gain responses and an average less than expected weight gain in response to over-feeding, many exercise intervention studies have resulted in a heterogeneity of body weight and body composition responses and a less than expected average weight loss in response to exercise induced EE (Church et al., 2009; King et al., 2007b; Sawyer, Manuscript in Preparation). Individuals who lose less weight or body fat than expected in response to a negative energy balance have been described as “compensators”, while those who lose equal to or more than the expected value are referred to as “non-compensators”. It has been proposed that compensators may lose less weight than expected due to the activation of adaptive compensatory mechanisms which allow them to preserve the body’s energy stores in response to prolonged negative energy balance. These compensatory responses may either fully or partially offset negative energy balance making individuals more resistant to weight loss (R. Stubbs et al., 2002).

However, even if an individual is initially able to lose weight, weight regain must be avoided in order to reap the long term benefits of reduced disease risk. Successful weight loss has been defined as a weight loss of at least 10% of peak body weight that is

maintained for greater than one year. Predicting weight loss maintenance success is difficult as correlates of weight loss maintenance only explain approximately 20-30% of the observed variance in long term weight loss success with the source of the remaining variance being unaccounted for as of yet (J. Stubbs et al., 2011). However, the National Weight Control Registry, a prospective study of over 10,000 subjects who have lost in excess of 30 pounds and maintained the loss for at least one year, reports that a high level of physical activity is among the top two behaviors contributing to successful weight loss along with consuming a low fat, low energy diet. Stubbs et al. state that exercise “is of overwhelming importance in maintaining weight loss” and an even stronger predictor of weight maintenance than initial weight loss (J. Stubbs et al., 2011).

With successful weight loss maintenance correlates remaining largely unknown, approximately 80% of weight loss attempts result in either no initial weight loss, or the majority of lost weight being regained within one year (Wing & Hill, 2001). The ACSM has reported that perhaps even more than 250 minutes of moderate intensity exercise per week may be necessary to prevent weight regain following initial weight loss (Donnelly et al., 2009). Some may attribute this poor weight loss success rate and exceptionally high amount of exercise necessary to prevent weight regain as support for the Set-Point Theory of weight loss. The Set-Point Theory states that body weight and body fat stores are regulated by a homeostatic mechanism which maintains these variables within a narrow range around a genetically determined “set-point”. The theory holds that when EE is increased threatening fat stores, physiological adjustments take place to preserve body weight through a number of mechanisms possibly including increased EI, reduced efficiency of metabolism, and likely a number of other pathways (Harris, 1990).

Past research has demonstrated the heterogeneity of weight gain (Bouchard et al., 1990) and weight loss (Church et al., 2009; Donnelly et al., 2003; King et al., 2007b; Sawyer, Manuscript in Preparation) elicited by individuals in response to excess positive and negative energy balances. Studies have also documented the existence of energy compensation in supervised exercise interventions (Church et al., 2009; King et al., 2007b; Sawyer, Manuscript in Preparation). Investigators have aimed to identify the specific mechanisms contributing to compensation; however, this is still a work in progress. Compensatory responses have been placed into two different categories: behavioral and physiological. Behavioral compensatory responses generated as a result of increased EE from purposeful exercise may include a decrease in non-exercise activity thermogenesis (NEAT) during non-exercise hours, or an increase in EI resulting from increased appetite. Behavioral mechanisms also have a biological component, but are considered volitional in nature. For example, satiety and hunger have hormonally controlled feedback mechanisms, however a person can choose when or when not to eat. Physiological adaptations to negative energy balance are considered automatic as opposed to volitional and may include changes in Basal Metabolic Rate (BMR) (King et al., 2007b). Additionally, differing exercise doses were examined in relation to the prevalence and magnitude of compensatory responses that they generated (Church et al., 2009; Hollowell et al., 2009; Thomas et al., 2012).

Past research has also attempted to predict which individuals may be prone to compensation. Baseline demographic, body composition, exercise physiology responses to submaximal exercise, and changes in fat mass early on in exercise training were examined in relation to body weight and composition changes at the end of the

intervention in order to assess the existence of possible predictive relationships.

Investigators reported that baseline submaximal exercise ventilation rate (VE) and change in body weight after four weeks of training were moderate predictors of change in fat mass following a 12-week exercise intervention (Sawyer, Manuscript in Preparation).

In summary, past research has sought to understand how the body compensates, how to identify individuals likely to compensate, and exercise prescriptions that make people more likely to compensate. Additionally, there have been many studies that have attempted to determine the most effective method of weight loss maintenance (Caplan, Bowman, & Pronk, 2007; Curioni & Lourenco, 2005; Wadden et al., 1992). However, to my knowledge there has been no research examining the effects of continued exercise on the body composition of compensators long-term, or whether the weight gain experienced during the exercise intervention for these individuals caused a restoration of set point to the increased weight which could potentially carry long term negative consequences in terms of increased disease risk. This study aimed to investigate whether or not physical activity levels and body composition changes achieved during a 12-week exercise intervention were maintained post-intervention. We investigated whether weight loss and physical activity were maintained, and whether weight and fat mass gained during the exercise intervention was transitory or retained throughout follow-up. A secondary aim of this study was to examine whether changes in body composition during the 12-week exercise intervention could predict body composition changes at the one-year follow-up. I hypothesized that one year following a 12-week supervised exercise intervention, women who continued to exercise would lose weight reverting back to baseline if weight was initially gained, and producing further weight loss if weight was initially lost.

Conversely, those who discontinued purposeful exercise at the conclusion of the study were expected to continue to gain or regain weight.

This research is important because a large proportion of Americans are either overweight or obese and are therefore at an increased risk of obesity related chronic diseases and co-morbidities. Exercise is often prescribed to all people, especially those that are overweight and obese as a means of weight loss and weight maintenance. This research seeks to determine whether there may be long term negative consequences for over-compensators who actually gain weight during exercise interventions. If over-compensators and partial compensators who lose less weight than expected can be identified early on, perhaps other methods can be undertaken to help promote weight loss and disease risk reduction in these individuals.

## CHAPTER 2

### REVIEW OF LITERATURE

With less than one third of Americans being considered healthy weight (Flegal et al., 2012), a great deal of focus has been given to exercise as a method of weight loss to decrease chronic disease risk. However, weight loss, by whatever method has a notoriously low success rate, with individuals often losing no or less weight or fat mass than expected, or even gaining weight or fat mass in some cases. Thus, much research has been dedicated to investigating why individuals lose less weight or fat mass than expected in response to exercise, and how this phenomenon of compensation can be prevented.

It should be noted that a number of studies tracked only changes in body weight as measured on a scale, whereas other studies also measured body composition such as changes in lean and fat masses. Body weight is made up of a number of components which include both lean and fat mass but also bone mass and water content. When a study measures changes strictly in body weight, often referred to as just “weight”, the ratio of change in each of the four components of body composition cannot be determined. However, all changes in weight are not equal. An increase in body weight which is primarily a factor of increased body fat is detrimental to health whereas an increase in weight due primarily to increases in lean body mass is generally regarded as a beneficial outcome. Therefore, a number of more recent studies have begun tracking changes in body composition as well as body weight.

Past research has recognized a marked heterogeneity of weight and body composition changes in response to exercise (Church et al., 2009; Donnelly et al., 2003;

King et al., 2007b; Sawyer, Manuscript in Preparation) and documented compensation, a phenomenon in which individuals lose less weight or fat mass than expected in response to increased EE (Church et al., 2009; 2005; King et al., 2007b; Sawyer, Manuscript in Preparation). Multiple studies have examined the relationship between behavioral and physiological mechanisms and compensation (Church et. Al, 2009; Hollowell et al., 2009; King et al., 2007b; Manthou, Gill, Wright, & Malkova, 2010; McLaughlin, Malkova, & Nimmo, 2006; R. Stubbs et al., 2002; Westerterp, Meijer, Janssen, Saris, & Ten Hoor 1992; Woo, Garrow, and Pi-Sunyer, 1982; Woo and Pi-Sunyer, 1985), and doses of exercise which may cause induce a greater magnitude and prevalence of compensation (Church et al. 2009; Hollowell et al., 2009; R. Stubbs et al., 2002). Finally, taking this information, investigators have attempted to identify and predict which individuals may be more prone to compensation (Sawyer, Manuscript in Preparation). The following is a review of past literature on the topic of compensation.

### Heterogeneity and Energy Compensation

Within this heterogeneity of weight and fat change seen above, there are a number of individuals whose weight and fat mass loss were less than expected given their ExEE, and even those who gained weight in response to exercise. Individuals who lose less weight than expected tend to exhibit an activation of compensatory mechanisms in response to an increase in ExEE which either fully, or partially, offsets the negative energy balance produced by exercise- making it more difficult for these individuals to lose weight. Sawyer et al. found that the cumulative ExEE generated by 12-weeks of vigorous exercise was not significantly correlated with changes in fat mass in women,



despite the fact that women on the high end of the ExEE range burned approximately two times more calories than those on the low end. This lack of a correlation between ExEE and change in body fat suggests that these compensatory mechanisms may be quite dominant (Sawyer, Manuscript in Preparation).

### Mechanisms of Compensation

The mechanisms of compensation can be examined within the context of energy balance. Energy balance is equal to total energy expenditure plus total energy intake. Energy intake is kilocalories consumed in the form of food or beverage. Energy expenditure are kilocalories burned through a number of processes including: ExEE, non-exercise activity thermogenesis (NEAT), basal metabolic rate (BMR) which is closely related to resting metabolic rate (RMR), and the thermic effect of food (TEF). ExEE is simply the number of calories expended through purposeful exercise. NEAT is the energy expenditure of the activities of daily living, and includes all non-purposeful exercise activity during the waking hours, with the exception of eating. Sitting, standing, fidgeting, working, and doing chores are all examples of NEAT. NEAT is the most variable component of EE in Americans and may account for 15 to 50% of total daily EE depending on personal habits (Levine, 2004). BMR is a measure of how many kilocalories per day are required to run essential autonomic functions of the body at rest such as breathing, circulation, and protein synthesis. When people lose weight, their BMR typically decreases. However, some evidence has suggested that certain individuals may have an overshoot of this decrease in BMR, where BMR is decreased disproportionately to their weight loss, resulting in a slower metabolism (Levine, 2004).

Approximately 75% of the variance in BMR between individuals is accounted for by volume of lean mass with greater lean masses resulting in greater BMRs. The TEF are kilocalories burned in the digestion and storage of food. There is little evidence to suggest that TEF is a factor in energy compensation in response to exercise (Levine, 2004).

Energy intake, like ExEE and NEAT, is a behavioral component of energy balance. EI has been shown by some studies to increase in response to increased ExEE, especially in leaner populations who have lesser fat stores at baseline. Some subjects have reported increases in appetite following an increase in ExEE (King et al., 2012; King et al., 2007a). Multiple investigators have reported that increased EI in response to increased ExEE results in partial compensation, and is likely the largest contributor to less than expected weight loss (King et al., 2007b; J. Stubbs et al., 2011; R. Stubbs et al., 2002; Westerterp et al., 1992; Woo & Pi-Sunyer, 1985).

In order to lose weight, one must increase one or more of the EE components, decrease EI, or a combination of these two methods to produce a negative energy balance. Behavioral components of compensation are generally regarded as having a greater magnitude of effect on compensation (King et al., 2007b). BMR is considered a physiological component of energy balance, and besides increasing lean mass through exercise, is primarily genetically determined and therefore not a common target of exercise interventions. ExEE, NEAT, and EI on the other hand are considered behavioral components of energy balance. These behavioral components are volitional in nature and therefore can be altered through lifestyle changes to produce a negative energy balance. The following articles investigate both the physiological and behavioral mechanisms of

energy balance that may lead to less than expected weight loss in response to exercise (King et al., 2007b).

In The Midwest Exercise Trial, Donnelly and colleagues sought to determine the effectiveness of exercise as a long term weight management strategy for overweight and obese populations (Donnelly et al., 2003). This was a randomized trial in which subjects were assigned to either an exercise, or a non-exercising control group. Subjects were overweight or moderately obese males and females between the ages of 17 to 35 who were previously sedentary. The intervention was a fully supervised 16 month aerobic exercise intervention. In the first six months participants ramped up to the desired intensity of 75% of heart rate reserve and duration of 45 minutes per day five days per week, at which level exercise was continued for the remainder of the 16 month study period. The 2000kcal/week energy expenditure elicited by the intervention was based upon the ACSM's recommendations for weight loss.

Both the exercise and control groups underwent the same measurements. Body weight was measured on an electric scale at baseline, 4, 9, 12, and 16 months. Body compositions determined by underwater weighing and visceral adiposity measure via CT were determined at baseline and follow-up. Doubly labeled water (DLW) was used at baseline and follow-up to measure total energy expenditure over a 12 day period. Energy intake was assessed by having participants eat all meals at the university cafeteria for two week periods at baseline and five other time points across the study period. During these two week periods snacks were recorded by recall on food questionnaires.

Analysis indicated that men expended an average of 667 kcal per exercise session and increased  $\text{VO}_{2\text{max}}$  by 20 % while women averaged 438 kcal per session and improved

VO<sub>2max</sub> by 16%. Males lost an average of 5.2kg while exercising women gained an average of 0.6kg and non-exercising women gained an average of 2.9kg over the length of the intervention. Male participants significantly decreased body weight, decreased body fat, and BMI as compared to controls. Males also significantly decreased visceral, subcutaneous, and total fat from their baseline measures. Females had significantly lower body weight, fat mass, and BMI as compared to controls. Females had small but insignificant decreases in visceral, subcutaneous, and total fat and small increases in lean mass from their baseline measures (Donnelly et al., 2003).

Data revealed that there was no significant difference in the number of kcals consumed or the proportion of kcals from each macronutrient between any of the groups at any of the time points. DLW data revealed that exercising males increased 24 hour energy expenditure by  $371 \pm 646$  kcals while control males decreased energy expenditure by  $34 \pm 999$  kcals. Female exercisers decreased energy expenditure by  $209 \pm 555$  kcals while control females decreased by  $141 \pm 431$  kcals. None of these differences were statistically significant, however changes in 24 hour EE was significantly correlated with ExEE.

Based on the results of this study, investigators reported that exercise had a significant effect on body weight even without dietary restriction. This intervention produced a negative energy balance in men of approximately 350kcals/day resulting in an average weight loss of 5.2kg. Exercising females experienced an average negative energy balance of 200kcals/day resulting in a small weight loss. However, this intervention did help exercising women avoid the 2.9kg weight gain that control women experienced over the 16 month period. In conclusion, exercise without dietary restriction is an effective method of weight loss in men and weight maintenance in women (Donnelly et al., 2003).

The following authors investigated the mechanisms of compensation in subjects who underwent exercise training without dietary restriction. King and colleagues published an original article in 2008 entitled “Individual variability following 12-weeks of supervised exercise: identification and characterization of compensation for exercise-induced weight loss” (King et al., 2007b). The purpose of this study was to document the variability of body weight in response to a supervised exercise intervention and to describe possible mechanisms of compensation. Authors hypothesized that individuals who lost less weight than expected in response to the imposed energy deficit would show differing magnitudes and mechanisms of compensation.

Thirty-five middle aged overweight and obese male (n=10) and female (n=25) subjects participated in this study. Participants exercised five times per week for 12-weeks, and burned 500 kilocalories at 70% of Heart Rate Max ( $HR_{max}$ ) on their choice of exercise machine.  $VO_{2max}$  was assessed by indirect calorimetry every four weeks in order to accurately prescribe exercise to meet the 500kcal target. The following measures were taken at baseline and immediately following the 12-week exercise intervention: resting metabolic rate (RMR) using a ventilated hood, body weight and body composition using the InBody brand bioelectrical impedance system, and height using a stadiometer. Additionally, on the baseline testing day participants were allowed to choose a meal to consume at the laboratory where the amount and macronutrient percentages of the meal were recorded. The same meal was prepared for the posttest and again the amount eaten and macronutrient contents were recorded. A subjective electronic 24-hour appetite survey was also filled out at baseline and 12-weeks.

Expected weight loss was calculated by summing the total exercise energy expenditure from each of the exercise sessions throughout the 12-week intervention period and dividing this figure by 7700 kcal which was the assumed energy composition of one kilogram of body weight. The number that resulted from this calculation for each participant was compared to actual weight change for each participant. Those participants who lost less weight than predicted were labeled as “compensators” (n=18) and those that lost equal to, or more weight than expected were labeled as “non-compensators” (n=17).

Mean weight loss was  $3.7 \pm 3.6$  kg and mean body fat loss was  $3.7 \text{ kg} \pm 2.6$  kg for the group which was statistically different from baseline values and highly similar to the expected weight loss for the group based upon total ExEE. However, there were large individual variances in bodyweight (-14.7 to +1.7 kg) and body fat (-9.5 to +2.6 kg) responses to the prescribed exercise.

On average change in body weight and fat mass were  $-6.3 \pm 3.2$  kg and  $-5.3 \pm 2.2$  kg respectively for non-compensators compared to  $-1.5 \pm 2.5$  kg and  $-2.1 \pm 2.3$  kg for compensators. Additionally, non-compensators displayed a decrease in lean mass of -0.89 kg while compensators experienced an increase in lean mass of +0.47 kg. All of the above body composition differences were significant between compensators and non-compensators despite no significant differences between the groups in baseline variables or energy expenditure during the exercise intervention (King et al., 2007b).

However, following 12-weeks of exercise the compensators increased EI consumed in the test meal by  $268.2 \pm 455$  kcal while the non-compensators decreased EI by  $-130.0 \pm 485$  kcal which was statistically significant. At post-testing, subjective hunger score was higher in compensators and this trend approached significance. RMR of non-

compensators showed a small increase of  $+14.2 \pm 242.4$  kcal per day whereas compensators had a non-significant decrease in RMR of  $-69.2 \pm 298.3$  kcal per day.

Based on these results, the authors concluded that compensation mechanisms that occurred in these participants, either partially or fully offsetting the energy deficit produced by increased exercise, were more behavioral than physiological in nature since compensators significantly increased their EI in test meals. Compensators also showed a trend of increased appetite. However, the authors do recognize that the test day meals may not be representative of the entire 12-week period. The authors also acknowledge that while the decreased RMR of compensators was not statistically significant, the effect may accumulate over time to play a role in offsetting negative energy balance (King et al., 2007b).

The purpose of the study by Manthou et al. was to investigate the contribution of NEAT to ExEE induced compensatory responses. 34 female subjects classified as overweight or obese who had been previously sedentary served as the subjects for this research. The study was an 8-week pretest posttest supervised exercise intervention in which subjects performed 150 minutes of aerobic exercise per week at approximately 72-77% age predicted  $HR_{max}$  (Manthou et al., 2010).

EI and total EE were measured the week prior to the start of the intervention and during the last week of the intervention. EI was self-reported via diary, as were all non-exercise activities. Subjects wore a heart rate monitor during all waking hours of the two one-week periods to assess total EE. On the first and last day of the intervention period body composition was assessed by leg to leg bioelectrical impedance, and various

physiological measures such as and RMR, lactate threshold, and indirect calorimetry in relation to heart rate during various activities were determined.

Results indicated that as a group, subjects had no weight loss despite a predicted weight loss of 0.8kg. There was a wide range of body fat responses (-3.2kg to +2.6kg) within the group with 11 being classified as non-compensators and 23 as compensators. Non-compensators reduced body fat percentage by 5.6% compared to only 1.9% in compensators despite no difference in ExEE. As a whole, the intervention increased total EE by an average 0.62MJ/day, however total daily EE was higher in non-compensators (+1.44MJ/day) than compensators (+0.29MJ/day). This difference in increased total EE despite no difference in ExEE between groups can be explained by a significant difference in NEAT EE between non-compensators (+0.79MJ) and compensators (-0.62MJ). There were no differences in RMR between groups. While EI did increase significantly from baseline in both groups, there were no significant between group differences in the number of kcals consumed or macronutrient composition (Manthou et al., 2010).

Authors found that the average group difference in NEAT from baseline to posttesting was a significant predictor of body fat loss during the intervention. They concluded that the compensatory response of decreasing NEAT EE in response to increased ExEE that was observed in compensators, but not in non-compensators, was at least partially responsible for the less than expected fat loss observed in compensators. This finding of no difference in EI between compensators and non-compensators in this study was in contrast to that found in the above article (King et al., 2007b) which reported that compensators significantly increased EI. Manthou et al., suggest that this



finding may either be a result of less accurate EI measures, or possibly of exercise dose. The King et al. intervention induced an ExEE of about 10MJ/week (King et al., 2007b) as compared to 4MJ/week in the current study. This difference may indicate a threshold for compensatory increase in EI (Manthou et al., 2010).

In a study by Woo and Pi-Sunyer, changes in EI in response to EE were investigated in a group of non-obese women (Woo & Pi-Sunyer, 1985). The subjects were five women between the ages of 21 and 51 with the average age of 37 who had been previously sedentary. These women were between 87% and 110% of ideal body weight based upon Metropolitan Life Insurance tables (equal to BMIs ranging between 19.07 and 23.79 with an average BMI of  $21.25\text{kg/m}^2$ ). Subjects were misled about the purpose of the study and were told that the research aimed to investigate the effect of exercise on protein metabolism.

The women spent the 62-day study period voluntarily confined in a hospital where voluntary food intake was discretely monitored and assigned exercise was fully supervised. EE was assessed every three to four days using indirect calorimetry. The first five days were dedicated as an observation phase used to assess sedentary EE and EI. The remainder of the study period was divided into 19 day phases in which exercise volume was manipulated. Voluntary food consumption was documented throughout the study. The order of treatments was determined randomly. During the sedentary EE phase no additional exercise was prescribed. During the mild EE phase an EE expenditure equal to 114% of sedentary which was equivalent to  $378 \pm 63\text{kcal/d}$  of EE was assigned. This assignment amounted to  $139 \pm 25\text{min/d}$  of treadmill walking at treadmill speeds between 2.5 and 3.5 miles per hour. During the moderate phase, an EE of 129% of sedentary EE

was prescribed which elicited an EE of  $772 \pm 40\text{kcal/d}$  and was equal to  $250 \pm 14 \text{ min/d}$  of exercise at the same walking speeds (Woo & Pi-Sunyer, 1985).

Results indicated that during the mild phase subject's EI was equal to 117% of sedentary EI, while EI during the moderate exercise phase was equal to 122% of sedentary EI. These values were both significantly greater than sedentary values but were not significantly different from each other. Analysis revealed that there was a significant increase in the percent of kcals coming from carbohydrates in the mild and moderate phases as compared to the sedentary phase, but that there was no difference between mild and moderate phases on this variable. Throughout all three phases of the study, subjects maintained an energy equilibrium where body weight and composition variables were unchanged. Energy balance was equal to  $+10 \pm 71\text{kcal/day}$  for sedentary,  $+64 \pm 43\text{kcal/day}$  in mild, and  $-116 \pm 92\text{kcal/day}$  in moderate exercise phases. Additionally, it was noted that there appeared to be no decrease in non-exercise EE during the course of the study based upon data from activity journals. From these data, authors concluded that lean women increase EI to compensate for increased EE imposed by exercise. They thought it likely that this increase in EI may be regulated by metabolic signals from fat stores which caused the females to consume more kilocalories in order to preserve their fat stores (Woo & Pi-Sunyer, 1985).

A second study by the same authors as above aimed to evaluate the effects of a moderate intensity exercise program on the EI of obese women (Woo et al., 1982). The subjects for this study were three previously sedentary women, who were classified as obese and were ages 17, 28, and 46. The women were between 169 and 206% of ideal

weight based upon Metropolitan Life Insurance tables (equivalent BMIs of 38.18, 34.36, and 42.65kg/m<sup>2</sup>).

Subjects were voluntarily hospitalized for 62 days during the course of this study. Subjects were observed for a five day period in which sedentary EI and EE were evaluated. During the subsequent 57 days the subjects were prescribed exercise at 125% of sedentary EE which was equivalent to approximately 111 minutes of treadmill walking on a 2.5% grade at 3 miles per hour. EE was assessed every three to four days by indirect calorimetry and voluntary EI was monitored discretely throughout the entire study. Body composition was measured using the total body water-nitrogen balance method at baseline and at the end of every 19 day period over the course of the study (Woo et al., 1982).

Results indicated that over the 57-day study period neither EE (2882kcal/day) nor EI (1903kcal/day) changed significantly. Subjects chose to eat an amount of kcal/day which was close to the amount of EE seen during the sedentary observation phase at the beginning of the study. These EEs and EIs resulted in a mean negative energy balance of 979kcal/day. There were statistically significant decreases in body weight (-6.75kg) and body fat (-6.09kg) over the course of the 57 day study. There was no significant change in lean mass, and weight loss was primarily from fat mass (89.3%). Additionally, actual weight loss of 6.75 kg closely matched calculated expected weight loss of 6.81kg in these subjects.

Based on the findings of this study, authors concluded that no compensatory increase in EI resulted to the prolonged negative energy balance produced by increased EE in the obese women. This finding was in contrast to the results demonstrated by the

same authors in a previous study in lean, non-obese women. Authors proposed that the decoupling of EI and EE observed in the obese women may have been due to a lack of activation of these compensatory mechanisms. They suggested that activation of compensatory mechanisms may only occur when the body's energy stores are running low as may have been the case in the lean women (28.5%) who had significantly less fat stores than the obese women (50.8%) (Woo et al., 1982).

The purpose of the study by McLaughlin and colleagues was to determine if an exercise program based on current exercise recommendations elicits compensatory responses on EI and NEAT, and to assess the resulting effect on energy balance. Eight males and eight females of Caucasian descent who were not regularly exercising participated in this study. They were between 20 and 25 years old and were considered lean, having body fat percentages of  $17 \pm 4.5\%$ . Subjects were not informed that EI was an outcome measure of the study in order to prevent bias in their eating patterns (McLaughlin et al., 2006).

Prior to the beginning of the intervention, participants underwent a graded exercise test to determine  $VO_{2peak}$ , exercise heart rate, and blood lactate threshold for exercise prescription purposes. The study period consisted of two eight day phases: the control phase where subjects performed their normal daily physical activity and the exercise phase where participants performed supervised exercise sessions. The order of the two phases was randomized, and separated by at least one week in males and by one menstrual cycle in females. Participants wore heart rate monitors during all non-sleeping hours during both the control and exercise phases and were also required to keep a

physical activity diary. BMR and weight were measured the day prior to and the day following each of the two phases of the intervention.

During the exercise phase of the intervention, participants exercised in the lab under supervision of a researcher every other day for a total of four sessions. Exercise was performed on a cycle ergometer at 60 revolutions per minute at a resistance which elicited 90% of lactate threshold (equivalent to 59%  $\text{VO}_{2\text{max}}$  in females and 48% in males). The length of exercise sessions was adjusted to burn 2092kJ plus BMR per session. During the control phase and the non-exercising portions of the exercise phase total EE was calculated from entries in the PA journal and EE values which were determined for each individual for a variety of activities prior to the start of the intervention (McLaughlin et al., 2006).

Results indicated that the prescribed exercise sessions led to a significant increase in total EE of  $9.6 \pm 3.3\text{MJ}$  in females and  $12.8 \pm 5.5\text{MJ}$  in males over the eight day exercise phase. Analysis revealed that there was no significant increase in sleeping EE (based on BMR), or NEAT EE in either gender between the control and exercise phases. Neither body mass nor BMR was significantly different between control and exercise phases for males. However, females experienced a small but significant decrease of 0.6kg in body mass during the exercise phase and a significant increase in BMR during the control phase.

From the results presented here, the authors concluded that there was no compensatory decrease in NEAT as a response to increased EE in either lean male or female subjects. The authors stated that the fact that women lost weight during the exercise phase suggests that women also did not increase EI in response to increased EE

whereas males who had no weight loss may have increased EI to offset the negative energy balance imposed by increased EE. However, the authors did note that the negative energy balance experienced by the women may not have persisted long enough in the eight day experimental period to induce adaptive compensatory mechanisms increasing EI, as a number of other studies have found that lean women do increase EI in response to long term negative energy balance (McLaughlin et al., 2006).

In summary, the majority of literature on mechanisms of compensation has found that the greatest contribution to less than expected weight loss stems primarily from behavioral rather than physiological mechanisms. As demonstrated, exercise, even the absence of caloric restriction can be a significant source of negative energy balance, as can dieting (Donnelly et al., 2003). The decrease of physical activity during non-exercise times of the day has been found to be a source of compensation in some (Manthou et al., 1992; Meijer, Westerterp, & Verstappen, 1999), but not other studies (Church et al., 2009; Hollowell et al., 2009; McLaughlin et al., 2006; R. Stubbs et al., 2002; Woo & Pi-Sunyer, 1985), and may be only transitory in younger adults with abruptly increased in ExEE and resulting fatigue (Hollowell et al., 2009), or a more lasting factor amongst older adults (Meijer et al., 1999). RMR may play a smaller role in compensation which may have a cumulative effect over the long term. One study described a decrease of 69kcal per day in compensators as opposed to non-compensators (King et al., 2007b), this decrease in EE per day would lead to a kcal surplus of 25,185kcal over a one year period which could lead to possibly significant weight loss if EI or EE were not adjusted accordingly. However, the majority of studies have found that increased energy intake as a result of increased appetite in response to exercise to be the biggest contributor to

compensation. Increased EI can offset the negative energy balance achieved by increased EE (King et al., 2007b; R. Stubbs et al., 2002; Thomas et al., 2012; Westerterp et al., 1992; Woo & Pi-Sunyer, 1985).

### Dose Response of Exercise and Compensation

Several studies have examined the effect of exercise dose and intensity on the degree and prevalence of compensation, as well as the mechanisms which may be responsible for this compensation. The Church et al. paper was based on data from the Dose Response to Exercise in Postmenopausal Women (DREW) study. The purpose of this study was to investigate changes in body composition and compensation in response to differing doses of exercise in overweight or obese sedentary postmenopausal women with elevated blood pressure (Church et al., 2009).

It was a randomized controlled study in which subjects were assigned to either a non-exercising control group or one of three exercising groups. The three exercising groups were assigned exercise doses which elicited increasing amounts of EE per kilogram of body weight per week. Prescribed doses were 4kcal/kg/week (KKW), 8KKW, and 12KKW corresponding to 50, 100, or 150 percent of the National Institute of Health (NIH) recommended dose for physical activity.

The exercise intervention spanned six months and participants exercised either three or four days per week on semi-recumbent cycles and treadmills, at a heart rate corresponding to an intensity of 50 percent  $VO_{2max}$ . All exercise sessions were supervised and monitored for intensity from which total exercise energy expenditure was calculated. Predicted weight loss was calculated taking total exercise energy expenditure divided by

7700. Body weight and waist circumference were measured at baseline and posttesting. Additionally, all participants wore a pedometer over the six month intervention period and filled out food intake questionnaires.

Results indicated that all four treatment groups, including the control group, experienced significant decreases in body weight from baseline to follow-up; however, there were no significant between group differences in weight change. Despite average weight loss in each group, there was a significant amount of heterogeneity in body weight response, and some individuals in every group actually gained weight during the intervention. Predicted weight loss corresponded closely with actual weight loss in the 4KKW and 8KKW exercise treatment groups. In fact, participants in these groups lost more weight than predicted. However, the 12KKW exercise group lost only 53% of the predicted weight loss. The prevalence of compensation in the exercise groups was as follows: 54.3% in the 4KKW group, 52.8% in the 8KKW group, and 72.6% in the 12KKW group. The mean compensation for the 12KKW group was 1.22kg whereas the other two exercise groups had no mean compensation. Additionally, all exercise groups had significant decreases in waist circumference, with the non-compensators decreasing approximately two times more than the compensators (Church et al., 2009).

Results on the behavioral mechanisms relating to energy expenditure indicated that during the first four weeks of the intervention, the control group took significantly more steps than any of the exercise groups, however by the end of the six month of the intervention there was no statistically significant difference in step counts between any of the groups. Additionally, all groups significantly reduced self-reported energy intake from baseline to posttesting, but there were no significant between group differences.



Based upon the results of this study, authors concluded that 150% of the current public health recommendation for physical activity produced both a greater prevalence and magnitude of compensation than lower doses of moderate intensity aerobic exercise. They noted that it was possible that higher intensity exercise may produce different levels and degrees of compensation and stressed the importance of finding effective exercise prescription protocols to help identified compensators effectively lose weight (Church et al., 2009).

Stubbs et al. also investigated the effects of differing exercise doses on mechanisms of compensation (R. Stubbs et al., 2002), but used lean rather than overweight and obese women as in the previous study (Church et al., 2009). Subjects were six women with an average age of 23 and an average BMI of  $21.4\text{kg/m}^2$ . This was a randomized repeated measures study design in which women performed each of the three nine-day treatment periods in random order at least one week apart. The treatments were: no additional exercise (NEX) in which subjects continued to perform habitual sedentary behavior expending an average total of  $9.2\text{Mj/day}$ , moderate exercise (MEX) consisting of two 40 minute exercise bouts per day expending an average total of  $11.0\text{Mj/day}$ , and high exercise (HEX) in which three 40 minute exercise bouts were performed per day resulting in an energy expenditure of  $12.1\text{Mj/day}$  (R. Stubbs et al., 2002).

Outcome measures included EE, EI, body weight, and subjective appetite and hunger scores. EE was calculated from heart rate data obtained from activity journals and heart rate monitors which were worn during all waking hours. All subjects were provided with a food scale and kept detailed records of EI. Body weight was measured

each morning under the same conditions. Appetite and hunger surveys were taken hourly and at the end of the day on a palm sized electronic device.

Analysis of data indicated that during the sedentary NEX condition EI was equal to 1.58xBMR. EI increased with exercise to 1.64 and 1.78xBMR in the MEX and HEX conditions respectively with additional calories coming from carbohydrate and fat sources. NEAT EE was calculated by taking total EE values obtained from HR data and subtracting ExEE measured during prescribed exercise. This calculation revealed no significant differences in NEAT EE between groups. Additionally, end of the day surveys showed a small but significant increase in subjective hunger.

Based on these results, the authors concluded that increased ExEE did not lead to any detectable compensatory decreases in NEAT EE. Additionally, the increase EI in response to increased hunger with greater ExEE provided only a partial compensation of approximately 33%. Therefore, the observed increase in EI did not completely offset the negative energy balance achieved by increased ExEE, and the women were able to achieve a negative energy balance (R. Stubbs et al., 2002).

A third paper by Hallowell et al. draws from subject data of the STRIIDE (Studies of a Targeted Risk Reduction Intervention Through Defined Exercise) trial to investigate the effect of both exercise duration and intensity on NEAT EE. Subjects included in this analysis were sedentary overweight or obese men and women with a mean age of 53.2 years. The intervention was 8-months in length with one non-exercising control group and three aerobic exercise groups: low amount/moderate intensity (40-55%  $VO_{2peak}$  expending 5023kJ/week), low amount/vigorous intensity (65-80% of  $VO_{2peak}$  expending

5023kJ/week), and high amount/vigorous intensity (65-80% of  $\text{VO}_{2\text{peak}}$  expending 8372kJ/week).

Outcome measures included total EE and NEAT EE. Total EE was computed from heart rate monitors worn during waking hours for seven consecutive days at both the beginning and the end of the intervention. NEAT EE was calculated by subtracting ExEE, obtained from HR monitor data taken during supervised exercise sessions, from total EE. Results showed that total EE increased with exercise dose as expected. The exercising group displayed small but non-significant increases in NEAT EE as compared to the control group.

Authors concluded that sedentary, middle aged overweight or obese men and women did not experience a compensatory decrease in NEAT EE as a result of increase ExEE. This finding is in contrast with several past studies (Manthou et al., 1992; Meijer et al., 1999) which have demonstrated a decrease in NEAT with increased ExEE. Authors suggest that compensatory decreases in NEAT EE seen in these short-duration studies may actually be a result of transitory fatigue due to the abrupt increase in ExEE. They proposed that this fatigue, and with it the down regulation of NEAT, may eventually subside as subjects become accustomed to their new exercise volumes (Hollowell et al., 2009).

### Predictors of Compensation

As more data became available on the mechanisms of compensation, investigators began searching for correlates that may possibly be used to predict compensation. Identification of possible non-responders early on in an exercise intervention, or even

before the intervention begins, may allow the prescription of different methods to those that do not respond to exercise. An experimental study by Sawyer et al. aimed to determine the effect of initial body weight and body composition on subsequent body weight and composition changes in response to an aerobic exercise intervention (Sawyer, Manuscript in Preparation). A secondary goal of the study was to determine if early body composition responses to exercise were predictive of changes at the conclusion of the study and whether any exercise testing variables could predict which subjects would be more prone to display compensation in response to exercise. Authors hypothesized that subjects with higher body fat percentages would lose more body fat than leaner women who would tend to compensate more. Additionally, authors predicted that fat loss within the first four weeks of the intervention would predict fat loss at 12-weeks (Sawyer, Manuscript in Preparation).

Forty previously sedentary premenopausal women served as the subjects for this research. The exercise intervention was 12-weeks in length with participants exercising three days per week for 30 minutes each session. Participants walked on a treadmill at 70% of  $VO_{2peak}$  which is approximately equivalent to 80% of  $HR_{max}$ . An incremental exercise test was issued at baseline and every four weeks thereafter to assess  $VO_2$  and adjust exercise intensity. Weight and waist circumference were measured at baseline, four weeks, and 12-weeks. Body composition was assessed by Dual-energy X-Ray Absorptiometry (DXA) at baseline and follow-up.

Total ExEE was calculated for the 36 exercise sessions in the intervention. Energy balance of the body was then actually calculated by multiplying the fat and lean mass values obtained from DXA scans by the accepted energy equivalents for fat and lean

tissue. Energy compensation was calculated by adding these two values. Subjects were then classified as compensators, if the energy compensation variable was positive, and non-compensators if the value was negative.

Results indicated that subjects displayed a heterogeneity of body composition responses, but that as a group, subjects had no significant weight loss, or changes in body fat, lean mass, fat mass, or waist circumference. The 29 subjects who were classified as compensators weighed less and had less body fat at baseline than non-compensators, despite no significant between group differences in total exercise energy expenditure during the intervention. Body weight, fat mass, and percent fat mass changed significantly from baseline in both compensators and non-compensators, however the direction of change was different in each group with compensators increasing these values and non-compensator decreasing them (Sawyer, Manuscript in Preparation).

Statistical analysis revealed that the only significant predictor of change in fat mass over the intervention period was submaximal exercise ventilation (VE) at baseline. Authors found that their data contradicted their hypothesis- initial body weight or composition did not predict changes in fat mass over the intervention period. Additionally, changes in fat mass were also not predicted by net exercise energy expenditure throughout the intervention or by baseline fitness level. However, the investigators did find that changes in body weight after the initial month of the intervention did moderately correlate with the change in fat mass and body weight at the conclusion of the intervention (Sawyer, Manuscript in Preparation).

## CHAPTER 3

### METHODS

The current study is a follow-up to a 12-week aerobic exercise training study in women. In the original study all participants completed a supervised 12-week aerobic exercise training program and had body composition analyses before and after training. A one year follow-up study using the original participants was conducted investigating changes in body composition and physical activity maintenance following the conclusion of the initial exercise intervention.

#### Participants and Study Design

Subjects for the 12-week exercise intervention were recruited from the community surrounding the Arizona State University Polytechnic Campus via email, posted flyers, and Craigslist advertisements. In order to participate in the original study subjects must have been females between the ages of 18 and 45 who were not engaging in habitual exercise for at least three months prior to the start of the study. Subjects must also have been willing to come to the laboratory to participate in 12-weeks of supervised exercise. Any potential subjects were excluded from participation if they answered “yes” to any of the questions on a PAR-Q survey taken during screening. Additionally, women who were pregnant or on a restrictive diet at the time of recruitment were also excluded. Women with orthopedic issues or injuries, heart conditions, and neuromuscular disorders were also excluded. 82 subjects meeting the inclusion criteria completed the original 12-week intervention.

In order to participate in the follow-up study, women must have been participants of, and have completed the entire 12-week exercise intervention including body composition testing. All subjects who had completed the initial study were contacted either by phone or email and invited to participate in the follow-up study. Potential subjects must have been willing to visit the laboratory on one occasion for approximately one hour to fill out two short physical activity surveys, have a urine test, DXA scan, and height and weight measurements taken.

Subjects who had reported being diagnosed with a thyroid problem, were currently pregnant, or had given birth since the completion of the initial study were excluded from participation in the follow-up. Additionally, any participants who had undergone any contrast procedures including treatments with iodine, barium, and or isotopes within the past seven days were not permitted to participate in the study until eight days had passed since the treatment was performed.

Women who were contacted that showed interest in participating were invited to the Healthy Lifestyles Research Center (HLRC) at the ASU Polytechnic Campus. The purpose and nature of the study as well as the procedures were explained in detail to the subjects. Questions and concerns were answered and written informed consent (Appendix A) was obtained from individuals wishing to participate in the study. 24 of the original 82 subjects who completed the exercise intervention passed initial screening and consented to participate in the follow-up study (n=24).

The study consisted of a single visit that was completed between October and December of 2012. Each subject received a ten dollar gift card to Starbucks upon

completion of the study. This follow-up study was approved by the Institutional Review Board at Arizona State University.

### Exercise Intervention Testing

Tests performed during the initial exercise intervention that are relevant to the follow-up are described here. All subjects underwent the following tests at baseline, and at four, eight, and 12-weeks:

#### VO<sub>2max</sub> Testing:

Gas exchange was monitored continuously through indirect calorimetry with a Parvo Truemax 2400TM (Parvomedics, Sandy, Utah) metabolic measurement device. Subjects were fitted with a mouthpiece and headset and allowed time to adjust to the measurement equipment before collecting resting gas exchange data. Subjects were then instructed to walk on a treadmill set at 3 miles per hour (mph) on a flat grade for one minute. After the initial minute, the treadmill grade was adjusted to 2%, and then increased another 1% each minute while treadmill speed was held constant. The test continued until the subject reached volitional fatigue and was unable to continue walking. All subjects received the same verbal encouragement from supervising research assistants for the whole duration of the test. Maximal oxygen consumption was recorded as the greatest VO<sub>2</sub> value achieved while respiratory exchange ratio was at least 1.15 and heart rate was greater or equal to 90% of the age predicted maximum calculated by subtracting the subject's age from 220.



#### Body Composition Assessment:

After a negative urine pregnancy test was confirmed, Dual-Energy X-Ray Absorptiometry (DXA, Lunar Prodigy, GE Medical Systems Lunar, Madison, WI) was used to determine fat mass and lean mass in kilograms. The DXA machine was calibrated at the beginning of each day of testing by the same certified radiology technician who performed all DXA scans. Subjects were asked to consume no food or liquid in the five hours preceding testing and wore no metal or jewelry during the scan. Subjects laid in a supine position on the DXA bed while the machine's arm performed a seven minute scan of the whole body. Analysis of DXA scans was performed using enCORE 2011 software version 13.6.

#### Anthropometric Measurements:

Weight and height were measured on a standard Beam scale and stadiometer respectively.

#### Exercise Training Program

All subjects participated in a 12-week exercise program where treadmill walking was performed three times per week on non-consecutive days. A total of 36 training sessions were completed throughout the course of the study. Each session began with a five minute warm-up period in which the subject walked at a speed which was approximately 15% below preferred walking speed on a 0% grade. Following the warm-up, subjects walked for 30 minutes at a speed which was equal to 10% below preferred walking speed. The incline was adjusted throughout the session to elicit a heart rate

which corresponded to 70% of the most recently tested  $VO_{2peak}$  (corresponding to approximately 80% of HRmax). A Polar<sup>TM</sup> heart rate monitor (Polar, Finland) was worn during each of the exercise sessions to confirm that target heart rate was being achieved. Each training session concluded with a cool-down performed at the same specifications as the warm-up. Every exercise session was supervised by a research assistant who was certified in cardiopulmonary resuscitation. If an exercise session was missed, the subject must have made up the session in the following week. If more than two exercise sessions were missed throughout the duration of the study, and not made up before the one week deadline, that subject was dismissed from the study.

#### Independent Variable

After the conclusion of the 12-week exercise intervention, participants were left to their own devices and contacted approximately one year later to participate in the follow-up study.

#### Dependent Variables

##### Physical Activity Questionnaires:

Subjects answered a questionnaire designed for this study (Appendix B) and filled out the validated (Booth et al., 2003) international physical activity questionnaire short form (IPAQ-SF) (Appendix B). These questionnaires inquired about subjects' physical activity and dieting habits presently, and since the conclusion of the exercise intervention.

## Anthropometric and Body Composition Measurements:

Weight and height were measured on a standard Beam scale and stadiometer. The body composition variables of fat mass and lean mass in kilograms were measured by DXA according to the same protocol as is seen above. However, the scans in the follow-up study were performed with the GE Lunar iDXA (DXA, Lunar iDXA, GE Medical Systems Lunar, Madison, WI) an updated model of the DXA machine used in the initial study scans. iDXA data was analyzed with CoreScan software.

## Data Treatment

### DXA Scans

GE Lunar Prodigy data from the initial exercise intervention were converted to GE Lunar iDXA values with the use of cross calibration regression equations for women as described by Hull et al. (Hull et al., 2009). The equations for lean and fat mass translations from Prodigy to iDXA are as follows:

Total Body iDXA Lean Mass (kg) = Total Body Prodigy Lean Mass (kg) x 1.005 + 0.405

Total Body iDXA Fat Mass (kg) = Total Body Prodigy Fat Mass (kg) x 0.944 + 1.171

### Compensation Calculations

ExEE for the 12-week exercise intervention was estimated separately in four-week blocks using the most recent  $\text{VO}_{2\text{peak}}$  measurement values obtained from testing. Total ExEE was estimated by summing the ExEE value obtained for each of the three four-week blocks. To estimate ExEE, resting  $\text{VO}_2$  (predicted using  $3.5 \text{ ml kg}^{-1} \text{ min}^{-1}$ ) was subtracted from 70% of the most recent  $\text{VO}_{2\text{peak}}$  measurement and multiplied by an

assumed respiratory exchange ratio of 0.85 (using 4.86 kcal/L of O<sub>2</sub>) and then multiplied by the duration and number of exercise sessions. The following equation represents ExEE for one four-week block of the 12-week intervention where VO<sub>2peak</sub> and VO<sub>2rest</sub> are expressed in the absolute units of L/min:

$$\text{ExEE} = (.7 \times \text{VO}_{2\text{peak}} - \text{VO}_{2\text{rest}}) \times 4.86 \text{ kcal/L} \times 30 \text{ min} \times 12 \text{ exercise sessions}$$

Changes in fat mass ( $\Delta\text{fat mass}$ ) and lean mass ( $\Delta\text{lean mass}$ ) from DXA data were calculated from baseline to 12-weeks using the following equations where masses are expressed in kilograms:

$$\Delta\text{fat mass} = 12\text{-week fat mass} - \text{baseline fat mass}$$

$$\Delta\text{lean mass} = 12\text{-week lean mass} - \text{baseline lean mass}$$

Delta fat and lean mass values were then multiplied by the accepted standard values for the energy equivalents of fat (9540kcal/kg) and lean (1100kcal/kg) mass (King et al., 2009) in order to estimate energy balance (EB) as is demonstrated by the following equation:

$$\text{EB} = (\Delta\text{lean mass (kg)} \times 1100 \text{ kcal/kg}) + (\Delta\text{fat mass (kg)} \times 9540 \text{ kcal/kg})$$

Total energy compensation (EEcomp) was then calculated by adding the values obtained from the ExEE and EB equations as is seen in the following equation:

$$\text{EEcomp} = \text{ExEE} + \text{EB}$$

Subjects who had positive EEcomp values were categorized as Compensators (or non-responders, and those with negative EEcomp values were categorized as Non-Compensators or responders.

## Statistical Analysis

All statistical analyses were performed using SPSS Statistics Version 21 (IBM, Armonk, NJ). Independent samples T-tests were used to determine statistical differences in various outcome variables between compensators and non-compensators. Paired samples T-tests were used to detect any significant differences in outcome variables over the following three time periods: baseline to 12-weeks (intervention period), 12-weeks to approximately one year (end of the intervention to the follow-up), and baseline to approximately 15 months (baseline to follow-up). Pearson's correlations were used to determine the relationships between body composition changes and other continuous variables such as EEcomp, ExEE, and IPAQ physical activity data. Spearman's correlations were used to correlate either continuous or dichotomous variables with other dichotomous variables such as compensator and non-compensator and yes/no survey questions. The level of significance for all tests was set at  $p=.05$ .

## CHAPTER 4

### RESULTS

#### Body Composition and Compensation

At baseline compensators and non-compensators were not significantly different on any demographic or body composition variable (Table 1). Subjects were  $31.0 \pm 7.6$  years old, weighed  $66.6 \pm 15.2$  kgs, stood  $161.6 \pm 6.3$  cms tall, had a BMI of  $25.5 \pm 5.5 \text{ kg/m}^2$ , with a fat mass of  $27.9 \pm 10.5$  kg and lean mass of  $38.9 \pm 5.4$  kg. Subjects were also not significantly different on the exercise physiology measures of submaximal exercise ventilation rate ( $\text{VE} = 27.6 \pm 8.0$  breaths per minute) and maximal oxygen uptake ( $\text{VO}_2 = 1.87 \pm 0.33 \text{ L/min}$ ) at baseline. During the intervention subjects expended a total of  $5810 \pm 1059$  kcal. While there was considerable variation in the amount of cumulative ExEE during the study (3754 to 8197 kcal) there was no between group difference for net ExEE between non-compensators ( $5810 \pm 1059$  kcal) and compensators ( $5839 \pm 1135$  kcal), however there was a significant between group difference in the EEcomp ( $p < .001$ ) which defines non-compensators (negative EEcomp) and compensators (positive EEcomp).

	All Subjects	Non-Compensator	Compensator
Age (years)	$31.0 \pm 7.6$	$29.9 \pm 9.4$	$31.7 \pm 6.6$
Weight (kg)	$66.6 \pm 15.2$	$68.6 \pm 15.7$	$65.5 \pm 15.4$
Height (cm)	$161.6 \pm 6.3$	$161.9 \pm 5.1$	$161.5 \pm 7.1$
BMI ( $\text{kg/m}^2$ )	$25.5 \pm 5.5$	$26.3 \pm 6.7$	$25.0 \pm 4.9$
Fat Mass (kg)	$27.9 \pm 10.5$	$28.6 \pm 10.9$	$27.5 \pm 10.6$
Lean Mass (kg)	$38.9 \pm 5.4$	$40.1 \pm 5.5$	$38.2 \pm 5.5$
Submax. Exercise VE (breaths/min)	$27.6 \pm 8.0$	$31.1 \pm 9.0$	$25.4 \pm 6.8$
$\text{VO}_{2\text{max}}$ (L/min)	$1.87 \pm 0.33$	$1.93 \pm 0.34$	$1.84 \pm 0.34$
Exercise EE (kcal)	$5810 \pm 1059$	$5840 \pm 1136$	$5793 \pm 1051$
Energy Compensation (kcal)	$5718 \pm 14,106$	$-7657 \pm 6398$	$13,743 \pm 10,932$

Table 1: Subject demographics at baseline. No significant ( $p < .05$ ) differences were found between groups on any variable with the exception of energy compensation ( $p < .001$ ).

### Baseline to 12-Weeks (Intervention Phase)

As a group, subjects experienced a non-significant body weight gain of  $+0.62 \pm 1.81$  kg during the 12-week exercise intervention (table 2). Fat mass decreased by a non-significant  $0.09 \pm 1.50$  kg and lean mass significantly increased by  $0.72 \pm 1.29$  kg ( $p=.001$ ). A substantial amount of heterogeneity was observed in the body weight ( $-3.01$  to  $+4.80$  kg), fat mass ( $-2.54$  to  $+3.44$  kg), and lean mass ( $-1.09$  to  $+3.46$  kg) changes in response to 12-weeks of supervised exercise. Based on EEcomp and the resulting body weight and fat mass changes, nine subjects ( $n=9$ ) were categorized as non-compensators and 15 subjects ( $n=15$ ) were classified as compensators. Non-compensators had an average EEcomp of  $-7657 \pm 6398$  kcals while compensators experienced  $+13,743 \pm 10,932$  kcals of EEcomp.

As a group, non-compensators lost  $-0.71 \pm 1.46$  kg of body weight compared to a gain of  $+1.42 \pm 1.54$  kg in compensators which represents a significant between group difference ( $p=.003$ ). Weight change was significantly different between baseline and 12-weeks for compensators ( $p=.003$ ) but not for non-compensators. There was also a significant between group difference in change of fat mass ( $p<.001$ ) with non-compensators losing  $-1.52 \pm 0.66$  kg of fat mass during 12-weeks of exercise training, compared to a gain of  $+0.76 \pm 1.14$  kg in compensators. This represents a significant within group change in fat mass from baseline to 12-weeks for both non-compensators ( $p<.001$ ) and compensators ( $p=.002$ ). Non-compensators gained  $+0.90 \pm 1.54$  kg of lean mass compared to a gain of  $+0.61 \pm 1.15$  kg in compensators which was not significantly different between the two groups or from baseline to 12-weeks within groups.

## 12-Weeks to Follow-Up

From the conclusion of the 12-week intervention to the follow-up at approximately one year, subject's body weight increased by an average of  $+0.49 \pm 3.36$  kg, fat mass increased by  $+0.15 \pm 2.96$  kg, and lean mass decreased by  $-2.00 \pm 1.88$  kg. During this time period an even greater degree of heterogeneity was observed in body weight ( $-6.41$  to  $+7.80$  kg), fat mass ( $-5.04$  to  $+5.56$  kg), and lean mass ( $-4.75$  to  $+1.71$  kg) as compared to during the 12-week intervention. Non-compensators lost  $-0.26 \pm 3.59$  kg of body weight as a group compared to a gain of  $+0.94 \pm 3.26$  kg in compensators, a difference that was non-significant between groups. Non-compensators gained essentially no body fat ( $+0.01 \pm 2.61$  kg) while compensators gained  $+0.22 \pm 3.25$  kg. Changes in weight and fat mass were not significantly different from week 12 to follow-up. However, changes in lean mass were significantly different from 12-weeks to follow-up for both non-compensators ( $p=.006$ ) and compensators ( $p=.003$ ). Non-compensators lost  $-2.42 \pm 1.93$  kg of lean mass and compensators lost  $-1.75 \pm 1.87$  kg which was not significantly different between groups.

## Baseline to Follow-Up

As a group subjects gained  $+1.11 \pm 3.90$  kg of body weight, gained  $+0.05 \pm 3.17$  kg of fat mass, and lost  $-1.28 \pm 1.54$  kg of lean mass while they were being observed over about a 15-month period (12-week intervention plus approximately one-year follow-up) (figure 1). Non-compensators lost body weight both during the intervention and during the one year follow-up period with weight loss totaling  $-0.98 \pm 2.86$  kg, which was not significantly different from baseline to follow-up. Net fat mass loss for non-



compensators over this period totaled  $-1.51 \pm 2.53$  kg and lean mass decreased by a significant  $-1.52 \pm 1.02$  kg ( $p=.002$ ). Compensators on the other hand, gained weight during each of the time points for a total weight gain of  $+2.36 \pm 3.98$  kg over the 15-month period of study which was significantly different from weight at baseline ( $p=.037$ ). Fat mass increased by  $+0.99 \pm 3.23$  kg, and lean mass decreased significantly ( $p=.028$ ) by  $-1.14 \pm 1.80$  kg in these individuals over the same period.

	Time Point	All Subjects	Non-Compensator	Compensator	Between Group Sig.
<b>Body Weight (kg)</b>	Baseline-12 weeks	$+0.62 \pm 1.81$	$-0.71 \pm 1.46$	$+1.42 \pm 1.54$ a ( $p=.003$ )	.003
	12 weeks- FU	$+0.49 \pm 3.36$	$-0.26 \pm 3.59$	$+0.94 \pm 3.26$	.410
	Baseline- FU	$+1.11 \pm 3.90$	$-0.98 \pm 2.86$	$+2.36 \pm 3.98$ c ( $p=.037$ )	.040
<b>Fat Mass (kg)</b>	Baseline-12 weeks	$-0.09 \pm 1.50$	$-1.52 \pm 0.66$ a ( $p<.001$ )	$+0.76 \pm 1.14$ a ( $p=.002$ )	<.001
	12 weeks- FU	$+0.15 \pm 2.96$	$+0.01 \pm 2.61$	$+0.22 \pm 3.25$	.868
	Baseline- FU	$+0.05 \pm 3.17$	$-1.51 \pm 2.50$	$+0.99 \pm 3.23$	.060
<b>Lean Mass (kg)</b>	Baseline-12 weeks	$+0.72 \pm 1.29$ a ( $p=.001$ )	$+0.90 \pm 1.54$	$+0.62 \pm 1.15$	.612
	12 weeks- FU	$-2.00 \pm 1.88$ b ( $p<.001$ )	$-2.42 \pm 1.93$ b ( $p=.006$ )	$-1.75 \pm 1.87$ b ( $p=.003$ )	.410
	Baseline- FU	$-1.28 \pm 1.54$ c ( $p<.001$ )	$-1.52 \pm 1.02$ c ( $p=.002$ )	$-1.14 \pm 1.80$ a ( $p=.028$ )	.564

Table 2: Summary of body weight and composition changes and within and between group significances over the 12-week intervention and one year follow-up period for all subjects, non-compensators, and compensators.

a= Significant within group change from baseline to 12-weeks,

b= Significant within group change from 12-weeks to FU,

c=Significant within group change from baseline to FU

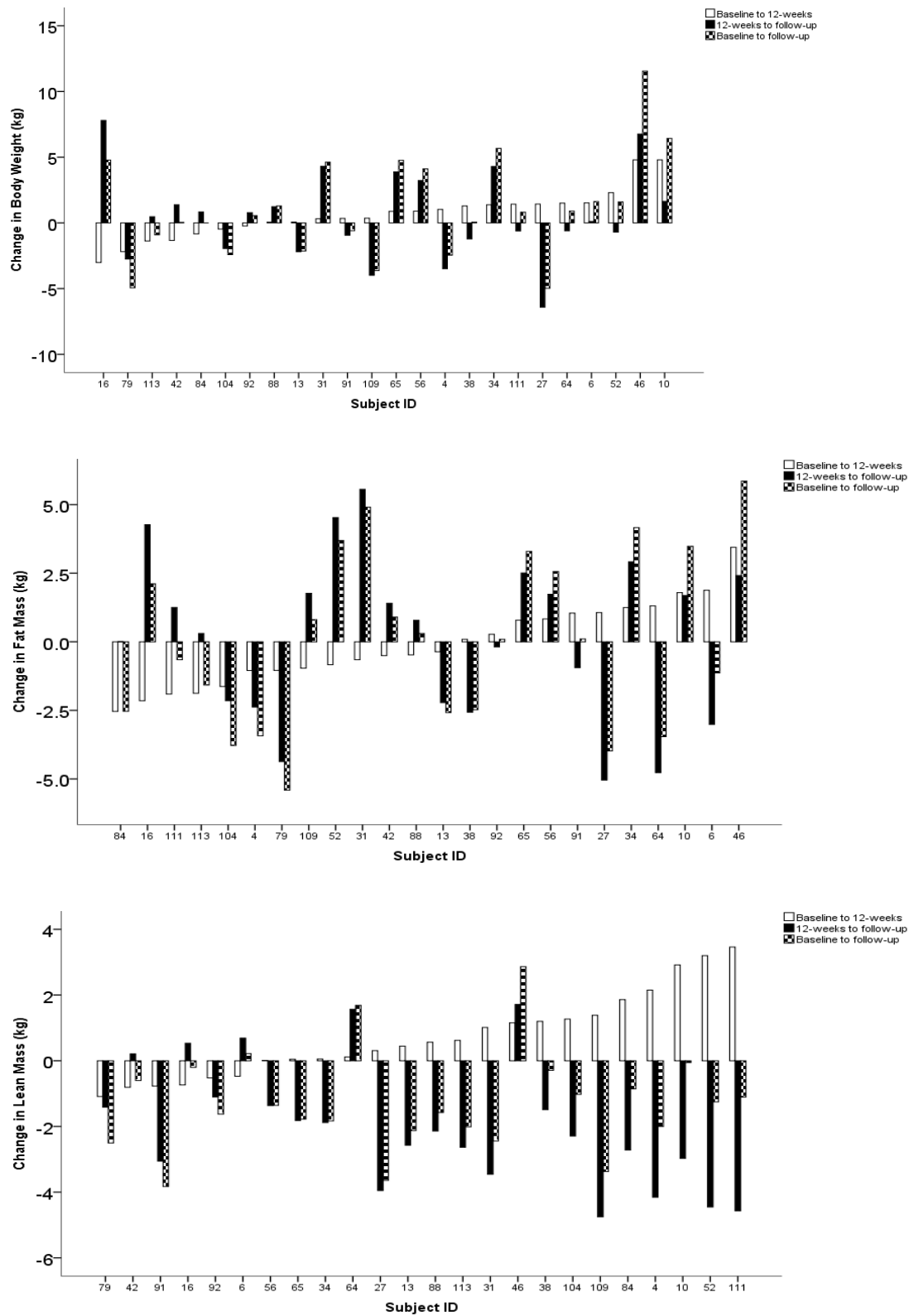


Figure 1: Changes in weight (top), fat (middle) and lean (bottom) masses in all subjects over 15 months.

## Patterns of Weight and Fat Mass Change

There are four possible combinations of patterns of weight and fat mass change over the course of this study. Participants could have lost+lost, lost+gained, gained+lost, or gained+gained weight or fat mass over the intervention period (0-12weeks) and the one year follow-up period (12weeks to 15 months) respectively. Weight changes over these periods are depicted graphically for non-compensators (figure 2a) and compensators (figure 2b).

Patterns of fat mass change over these same periods are displayed in (figure 3). Of the nine non-compensators, all nine initially lost fat mass during the 12-week exercise intervention. During the one year follow up period, three subjects continued to lose fat mass, one gained fat mass returning to initial fat mass (within 0.1kg), and five gained fat mass during the follow-up period. Of the five that gained fat during the follow-up, two gained less than they initially lost, resulting in a net fat mass loss over the 15 month period, while three regained more than was initially lost resulting in a net fat mass gain.

Four of 15 compensators initially lost fat mass, although fat mass loss was less than would be expected based upon net ExEE during training. One of these subjects continued to lose fat during the follow up period, while the other three gained more fat mass than was initially lost resulting in a net fat gain over the whole period. The remaining 11 compensators initially gained fat mass in response to the exercise intervention. Of these 11, six went on to lose weight during the follow up with two subjects ultimately returning to baseline fat mass (within 0.1kg) and four experiencing a net fat mass loss. The remaining five compensators gained fat mass both during the 12-week intervention and the one year follow-up.

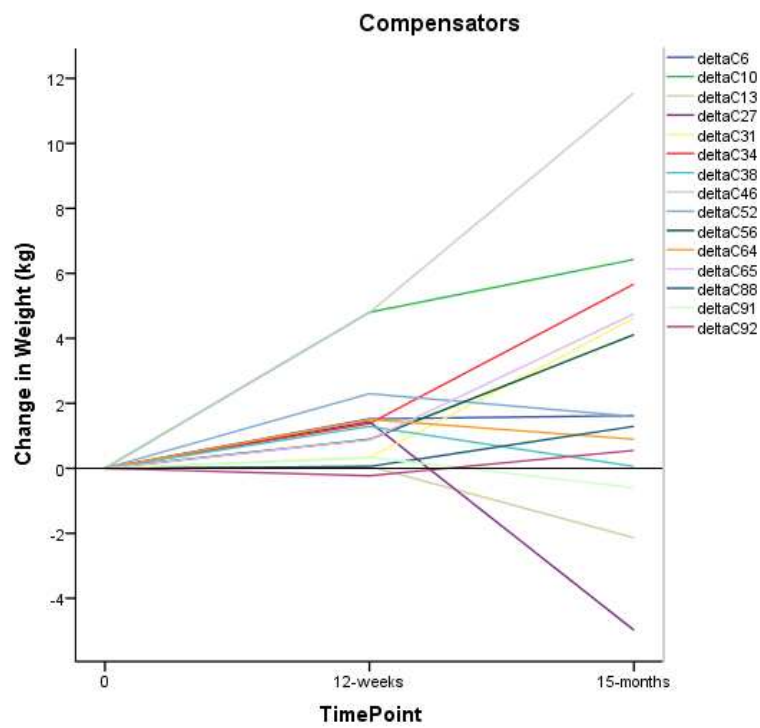
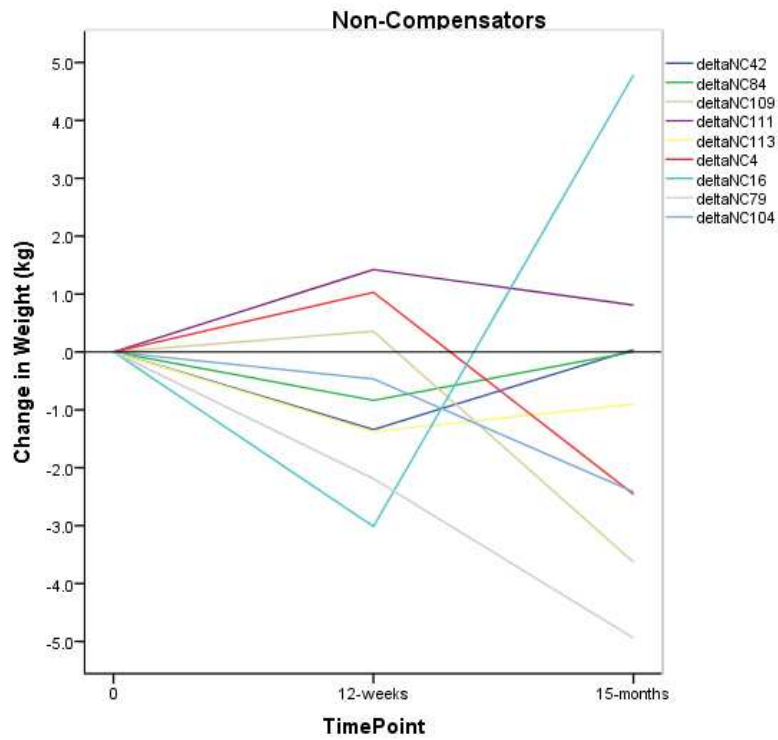


Figure 2: Weight changes over time in a) non-compensators (top) and b) compensators (bottom).

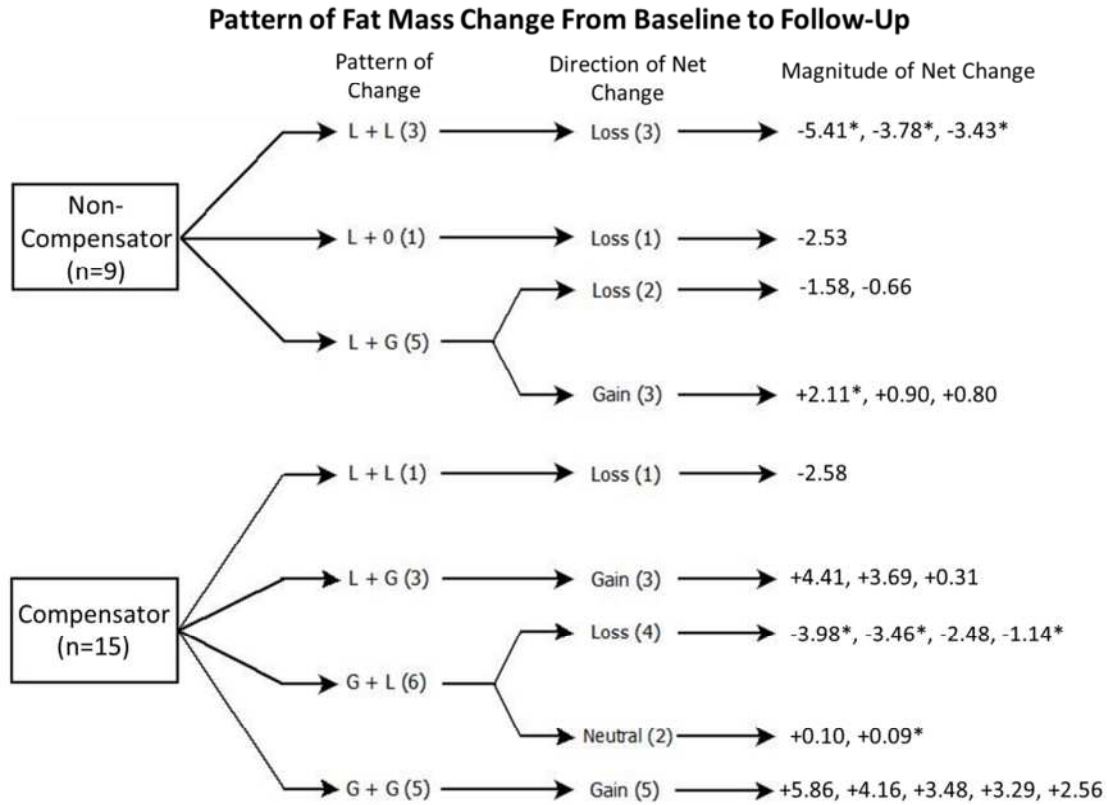


Figure 3: Patterns of fat mass change from baseline to follow-up in non-compensators and compensators. Pattern of change as indicated as the change from baseline to 12-weeks and 12-weeks to follow-up respectively, where L indicates a loss of fat mass during the period and G indicates a gain. The number of subjects in each category is indicated in parenthesis. Asterisks indicate that the subject continued to perform regular vigorous exercise on most days of the week after the conclusion of the 12-week intervention (as is discussed in chapter 5).

### Physical Activity Maintenance

Subjects filled out the IPAQ-short physical activity questionnaire (Appendix B) which quantifies sedentary time and allows the calculation of METmin/week for vigorous physical activity, moderate physical activity, and walking time. At the time of follow-up 14 of 24 subjects reported physical activity minutes and intensities that met the ACSM's recommendations of  $\geq 150$  min/week of moderate intensity exercise,  $\geq 75$  min/week of vigorous exercise, or some combination of the two (Garber et al., 2011). 10 of these

subjects reported that they met these requirements by achieving  $\geq 75$ min/week of vigorous exercise, three met requirements through a combination of vigorous and moderate exercise, and one through only moderate exercise. Conversely, 10 subjects reported physical activity habits that did not meet ACSM recommendations. Six performed no moderate or vigorous activity at all, while the remaining four performed insufficient amounts of physical activity. A total of 11 subjects reported performing no vigorous physical activity while 14 reported performing at least some vigorous activity. On the IPAQ-short questionnaire subjects reported the following averages which were not significantly different between compensators and non-compensators:  $5.93 \pm 3.60$  hours/day of sedentary behavior,  $1524.58 \pm 2007.29$  METmin/week of moderate and/or vigorous physical activity,  $195.94$ min/week of moderate intensity physical activity, and  $92.50 \pm 114.93$ min/week of vigorous physical activity. None of the physical activity or sedentary variables measured by the IPAQ-short form were significantly correlated with changes in weight, fat mass, or lean mass either from 12-weeks to follow-up or baseline to follow-up (table 3).

A second questionnaire (appendix B) which was designed to assess physical activity and dieting behaviors at the time of follow-up and in the year following the conclusion of the exercise intervention was also filled out during the follow-up visit. According to this survey only eight out of 24 subjects were currently performing habitual vigorous exercise programs on most days of the week. Seven subjects were currently trying to lose weight or dieting, and five had attempted diet or weight loss within the year following the exercise intervention. 12 subjects reported that they continued to exercise regularly after the initial intervention, seven did not continue to exercise, four reported

having exercised off and on in the last year, and one declined to answer the question.

Additionally, 11 subjects reported having returned to previous exercise habits after the conclusion of the exercise intervention. Currently dieting or attempting to lose weight was significantly correlated with changes in lean mass from 12-weeks to follow-up ( $r=.550$ ,  $p=.005$ ).

	<b>Body Weight (kg)</b>		<b>Fat Mass (kg)</b>		<b>Lean Mass (kg)</b>	
	<b>12wk-FU</b>	<b>Base-FU</b>	<b>12wk-FU</b>	<b>Base-FU</b>	<b>12wk-FU</b>	<b>Base-FU</b>
<b>Current vigorous exercise program</b>	-.319 ( $p=.128$ )	-.306 ( $p=.145$ )	-.562 ( $p=.004$ )*	-.587 ( $p=.003$ )*	.294 ( $p=.164$ )	.077 ( $p=.722$ )
<b>Continued to exercise regularly</b>	.008 ( $p=.971$ )	-.066 ( $p=.766$ )	-.042 ( $p=.848$ )	-.006 ( $p=.977$ )	.090 ( $p=.682$ )	-.145 ( $p=.510$ )
<b>Returned to prior exercise habits</b>	.260 ( $p=.220$ )	.199 ( $p=.350$ )	.344 ( $p=.099$ )	.272 ( $p=.199$ )	-.030 ( $p=.889$ )	-.079 ( $p=.715$ )
<b>Currently dieting or trying to lose weight</b>	.179 ( $p=.403$ )	.099 ( $p=.644$ )	-.033 ( $p=.878$ )	.007 ( $p=.976$ )	.550 ( $p=.005$ )*	.391 ( $p=.059$ )
<b>Diet and/or weight loss in last year</b>	.067 ( $p=.757$ )	.022 ( $p=.918$ )	-.052 ( $p=.810$ )	-.022 ( $p=.918$ )	.259 ( $p=.221$ )	.007 ( $p=.973$ )
<b>IPAQ Met-min/week</b>	-.057 ( $p=.791$ )	-.160 ( $p=.455$ )	-.151 ( $p=.481$ )	-.126 ( $p=.558$ )	.015 ( $p=.944$ )	-.297 ( $p=.158$ )
<b>Moderate exercise min/week</b>	.030 ( $p=.888$ )	-.014 ( $p=.949$ )	-.013 ( $p=.950$ )	.031 ( $p=.887$ )	.009 ( $p=.966$ )	-.186 ( $p=.383$ )
<b>Vigorous exercise min/week</b>	-.170 ( $p=.428$ )	-.331 ( $p=.114$ )	-.311 ( $p=.139$ )	-.320 ( $p=.127$ )	.020 ( $p=.925$ )	-.382 ( $p=.065$ )
<b>IPAQ sedentary hours/week</b>	.205 ( $p=.361$ )	.189 ( $p=.400$ )	.208 ( $p=.353$ )	.258 ( $p=.246$ )	-.041 ( $p=.855$ )	-1.51 ( $p=.503$ )

Table 3: Correlations of physical activity and dieting habits with body composition changes over time.

Additionally, there was a significant correlation between current vigorous exercise and fat mass changes from 12-weeks to follow-up ( $r=-.562$ ,  $p=.004$ ) and from baseline to follow-up for all subjects ( $r=-.587$ ,  $p=.003$ ). An independent T-test revealed between group differences from 12-weeks to follow-up in fat mass changes based on

whether or not subjects reported performing regular vigorous exercise on most days of the week (fig. 4). Subjects that did not report performing vigorous exercise gained  $+1.320 \pm 2.156$  kg of fat mass as compared to vigorous exercisers who lost  $-2.205 \pm 3.070$  kg of fat mass over the one year period ( $p=.003$ ).

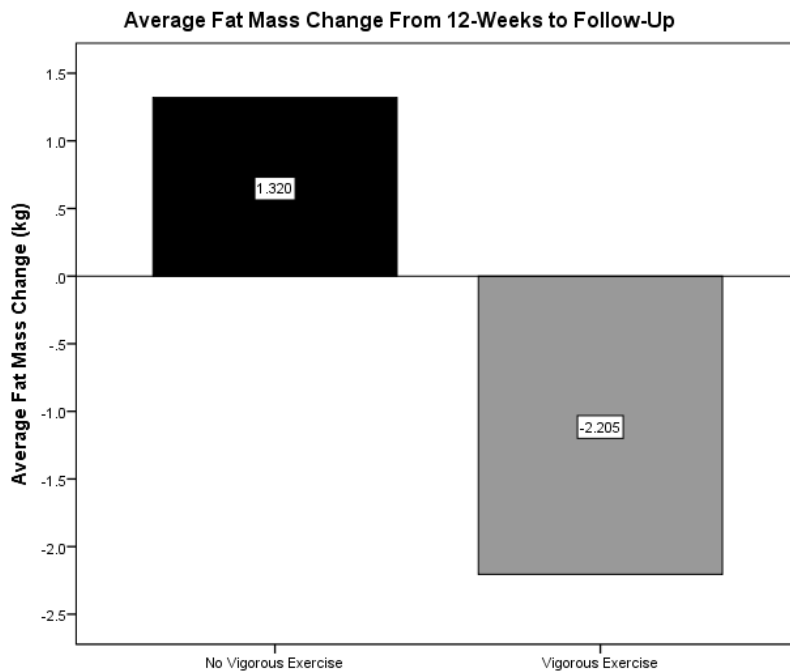


Figure 4: Fat mass change from 12-weeks to follow-up based on whether or not regular vigorous exercise was continued post-intervention.

### Predictors of Compensation

There were no significant correlations between either the dichotomous compensator/non-compensator or energy compensation (EEcomp) variables and any of the following measures: baseline age, weight, height, BMI, fat mass, lean mass, submaximal exercise ventilation rate, or net ExEE during the 12-week intervention (table 4). The degree of EEcomp observed during the 12-week exercise intervention was significantly correlated with change in weight from baseline to follow-up ( $r=.509$ ,



p=.011) while the correlation between EEcomp and change in fat mass from baseline to follow-up approached significance (r=.384, p=.064). None of these baseline variables were significantly correlated with weight or body composition changes from 12-weeks to follow-up.

		<b>Correlation</b>
<b>EEcomp (kcal) B-12wk</b>	<b>Change in weight (kg) B-FU</b>	r=.509 (p=.011)
<b>EEcomp (kcal) B-12wk</b>	<b>Change in fat mass (kg) B-FU</b>	r=.384 (p=.064)
<b>Change in weight (kg) B-4wk</b>	<b>Change in fat mass (kg) B-FU</b>	r=.421 (p=.040)
<b>Change in weight (kg) B-12wk</b>	<b>Change in weight (kg) B-FU</b>	r=.509 (p=.011)
<b>Change in fat mass (kg) B-12wk</b>	<b>Change in weight (kg) B-FU</b>	r=.504 (p=.012)
<b>Change in fat mass (kg) B-12wk</b>	<b>Change in lean mass (kg) 12wk-FU</b>	r=.346 (p=.033)
<b>Change in lean mass (kg) B-12wk</b>	<b>Change in lean mass (kg) 12wk-FU</b>	r=-.583 (p=.003)

Table 4: Correlations for predictors of body composition change during follow-up. B=baseline, FU= Follow-up.

However, several body weight and body composition changes that occurred during the 12-week exercise intervention were significantly correlated with body weight and composition changes during the one year follow-up or the entire 15-month study period. Weight change from baseline to four weeks was significantly correlated with the change in fat mass from baseline to follow-up (r=.421, p=.040). The change in weight from baseline to 12-weeks showed a moderate correlation with weight change from baseline to follow-up (r=.509, p=.001). Change in fat mass from baseline to 12-weeks correlated positively with both the change in weight from baseline to follow-up (r=.504, p=.012), and the change in lean mass from 12-weeks to follow-up (r=.346, p=.033). Finally, change in lean mass during the exercise intervention was moderately negatively

correlated with change in lean mass during the one year follow-up period ( $r=-.583$ ,  $p=.003$ ).

## CHAPTER 5

### DISCUSSION

As in other studies (Church et al., 2009; Donnelly et al., 2003; King et al., 2007b; Sawyer, Manuscript in Preparation), the population in this study displayed a substantial amount of heterogeneity in body weight and body composition changes in response to increased exercise energy expenditure. Based upon changes in lean and fat masses and energy expended during the 12-week intervention period, 62.5% of subjects were categorized as compensators while only 37.5% were classified as non-compensators. However, three of the nine non-compensators in our study actually gained weight, which has not previously been described. These subjects showed a pattern of having gained large amounts of lean mass (+1.38, +2.15, and + 3.46kg) and losing smaller amounts of fat mass (-0.96, -1.05, and -1.91kg) resulting in net weight gain (+0.356, +1.03, and +1.42kg) over the 12-week intervention. However, the above body weight and composition changes still lead to a negative EEcomp value ( $EE_{comp} = [(\Delta \text{lean mass (kg)} \times 1100 \text{ kcal/kg}) + (\Delta \text{fat mass (kg)} \times 9540 \text{ kcal/kg})] + ExEE$ ) given the observed ExEEs (5633.07, 6520.84, and 7461.82kcal) because the energy equivalent for fat mass is so much higher than the energy equivalent for lean mass.

In agreement with the hypothesis, we found that subjects that answered “yes” to the question, “Are you currently engaged in a regular vigorous exercise program, in which you perform intense aerobic or resistance exercise training on most days of the week?” had a net fat mass loss of  $-2.205 \pm 3.070$ kg over the follow-up year while those who answered “no” had a net fat mass gain of  $+1.320 \pm 2.156$ kg. Wadden et al. had a similar finding in a follow-up study to a 48-week clinical weight loss trial which had a

one year follow-up without contact with the subjects during this time (Wadden, Vogt, Foster, & Anderson, 1998). Subjects were 49 obese women with an average BMI of  $36.5 \pm 5.7 \text{ kg/m}^2$  and an average age of  $42.4 \pm 7.7$  years. The intervention involved dietary restriction, cognitive behavioral counseling, and either no exercise, or a supervised exercise treatment. At the conclusion of the treatment all subjects experienced substantial weight loss ( $13.5 \pm 9.1 \text{ kg}$  to  $17.3 \pm 10.3 \text{ kg}$ ) with no significant between group differences. At the one year follow-up subjects answered “yes” or “no” to the question, “Have you exercised regularly within the past four months?” 27 out of 49 subjects answered “yes” and had a greater net body weight loss of  $-12.1 \pm 8.9 \text{ kg}$  from baseline to follow-up as compared to those who answered “no” which had a net body weight loss of  $-6.1 \pm 6.2 \text{ kg}$ . Those that had engaged in a regular exercise program regained only  $+5.5 \pm 7.9 \text{ kg}$  from the end of the formal exercise intervention to the follow-up one year later, whereas those that did not regularly exercise regained  $+8.4 \pm 6.4 \text{ kg}$ . The most commonly reported physical activity of these subjects was walking of which an average of  $131 \pm 89.3 \text{ min/week}$  was performed. Investigators found that more minutes of walking per week significantly correlated with greater net weight loss from baseline to follow-up and less weight regain from the end of the formal intervention to follow-up (Wadden et al., 1998).

In both the current study, and that by Wadden et al., data suggest that continuing to perform regular exercise after the conclusion of a formal exercise intervention or weight loss program helps to maintain weight lost during the training period. The difference in the direction of weight change during the same one year follow-up period in these studies may be an effect of exercise intensity. In the Wadden study (Wadden et al., 1998), those that answered “yes” to having performed regular exercise still regained

+5.5±7.9kg during the follow-up, while in the current study, participants engaging in regular exercise actually lost -2.205±3.070kg of fat mass. This finding suggests that while walking may decrease the rate of weight regain regular vigorous exercise may actually continue to produce weight loss rather than just maintain it.

In a study on weight loss maintenance in overweight to obese women aged 21 to 45 years authors. investigated the effects of four different exercise prescriptions on weight loss maintenance over a 24-month period. This study did advise a restricted calorie diet and offered behavioral counseling with all four of the following exercise prescriptions: moderate intensity/moderate duration, moderate intensity/high duration, vigorous intensity/moderate duration, and vigorous intensity/high duration. Weight loss did not significantly differ between groups at either six months or 24 months. Overall, subjects lost 9.3±5.6% of initial body weight at six months and only retained a loss of 5.0±8.5% of initial body weight at 24-months. However, the vigorous intensity/moderate duration exercise prescription showed a trend of producing more weight loss at six months and of superior maintenance at 12, 18, and 24 months. It should be noted that the exercise in this intervention was not supervised and therefore adherence may play a factor in the magnitude of weight loss. Perhaps if exercise was supervised this trend may have become significant (Jakicic, Marcus, Lang, & Janney, 2008). To date, there are not many long-term weight maintenance studies investigating vigorous exercise, however, Jakicic et al. and the current study provide promising evidence for the effectiveness of vigorous exercise and the long term maintenance of weight loss.

The difference in the magnitude of weight regain during the same one year follow-up period in subjects who answered “no” to having performed regular exercise

( $+1.320 \pm 2.156$  kg in the current study versus  $+8.4 \pm 6.4$  kg in Wadden et al.) is likely due to the magnitude of weight change achieved during the active intervention. Subjects in the Wadden study lost substantially more weight than did those in the current study, and therefore had much more weight to regain in order to return to baseline weight and once again achieve set-point. Length of the initial intervention also plays a role on the magnitude of initial weight loss, and therefore possibly the magnitude of weight regain. The Wadden study was four times the length of the current study and involved not only exercise but also dietary restriction and counseling on changing behaviors associated with weight gain (Wadden et al., 1998). Therefore, due to the lesser initial weight loss of subjects in the current study, they had less weight to regain in order to return to their set-point weight.

It should be noted that while the yes/no question about regular vigorous exercise produced significant differences between groups in changes in fat mass from 12-weeks to follow-up, that no information from the IPAQ-SF including self-reported total MET-min/week of moderate and vigorous exercise, total minutes of moderate exercise per week, or total minutes of vigorous exercise per week, had significant relationships either in the form of correlations between exercise amount or intensity and changes in body composition for all subjects, or in between group differences for physical activity levels and changes in weight or fat mass from 12-weeks to follow-up.

Despite validation (Booth et al., 2003), the accuracy of the IPAQ-SF has been questioned. Lee et al. reported in a recent review of studies which had concurrently used objective measures of physical activity and exercise with the IPAQ-SF, that the correlation between the questionnaire and objective measures ranged from 0.09 to 0.39,

which does not meet acceptable standard of 0.50. The studies reviewed found that actual physical activity was over-reported on the IPAQ-SF by 36% to 173% with an average over-reporting on the IPAQ-SF of 84% as compared to concurrently used objective measures (Lee, Macfarlane, Lam, & Stewart, 2011). The lack of relationship between IPAQ-SF data (especially on overlapping vigorous activity data) and fat mass changes from 12-weeks to follow-up despite significant findings from the second survey in which a simple yes or no question was asked, may be a result of this tendency of subjects to over-report on the IPAQ-SF.

Many studies have performed follow-ups after formal weight loss programs or exercise interventions to determine if weight loss and body fat losses were maintained over time. However, to date, none of the studies examining compensation have had follow-ups to determine what happened to compensators who actually gained weight during an exercise intervention.

Of these eight compensators who had net fat mass gain, five gained fat mass over both time periods. For these subjects, the fat mass gained during the one year follow-up period (12-weeks to follow-up) was divided by four in order to determine the rate of weight gain for 12-weeks (the length of the exercise intervention) when the subjects were unsupervised. This 12-week rate of fat mass gain that occurred when subjects were not involved in a formal exercise intervention or performing regular vigorous exercise was then compared to the rate of fat mass gain that was observed during exercise training to determine if the exercise may have caused an increased rate of fat mass gain in these subjects as compared to when they were left to perform their regular physical activity habits. Two of these five subjects gained more fat mass during the 12-week exercise

intervention than they did in the entire one year follow-up period, while the remaining three gained fat mass at an accelerated rate during the exercise intervention as compared to the follow-up. As a group these five women gained an average of  $3.87 \pm 1.25$  kg of fat mass and  $6.50 \pm 2.96$  kg of body weight over the 15 month observation period. These women experienced over-compensation, an unintentional gain in weight or fat mass due to the activation of compensatory mechanisms that completely offset the negative energy balance achieved by increased ExEE (Sawyer, Manuscript in Preparation).

The question is, whether or not these women who gained weight at both time points suffer any long term negative consequences as a result of this over-compensation. These five subjects had a group percent weight gain of  $11.4 \pm 5.0\%$  with individual percent gains ranging from 6.9% to 18.62% of their initial weight from baseline to follow-up. It is suggested that a weight loss of 5-10% of initial body weight is sufficient to decrease the risk of type II Diabetes Mellitus and heart disease (National Heart, Lung, and Blood Institute, 2004). By the same logic, one might expect that a weight gain of the same magnitude would increase the risk of these diseases. In fact, of these five women, three went from having BMIs classified as normal weight to overweight and one from overweight to obese within the 15-month observation period. These observations may invite the conclusion that the exercise did have a potentially lasting long-term negative effect on this subset of subjects with respect to chronic disease risk. However, it cannot be determined how much weight the women may have gained if they had not performed the exercise intervention because this study did not have a control group.

Before the conclusion is drawn that exercise is bad for individuals who are classified as compensators, several of the patterns of fat mass change deserve a closer



look in relation to physical activity maintenance. We hypothesized that for women who gained weight during the intervention but continued to exercise post-intervention, weight gain would be only transitory, while those who discontinued exercise at the end of the intervention would continue to gain weight at a rate consistent with that of normal aging (Spirduso, 2010). However, this did not seem to be the case, at least not with respect to body weight, which was likely partially due to the large changes observed in lean masses from baseline to follow-up. Body fat, however, showed a much clearer pattern in relation to physical activity as was demonstrated by the significant correlation between regular vigorous exercise and fat mass change from 12-weeks to follow-up.

Overall, there were only eight subjects out of 24 (33.3%) that reported that they maintained a regular vigorous exercise program after the conclusion of the initial exercise intervention. All but one of the individuals who reported having maintained vigorous exercise lost fat mass during the follow-up period with an average fat mass loss of  $-2.205 \pm 3.070$  kg for those that continued vigorous exercise, and an average fat mass gain of  $+1.320 \pm 2.156$  kg for those that discontinued vigorous exercise.

This pattern becomes even more telling when we look at compensators and non-compensators individually. Of the eight subjects that reported that they continued vigorous exercise, half were compensators (4/15) and half were non-compensators (4/9). All three non-compensators who lost fat mass at both time points reported having continued regular vigorous exercise. Four of the six compensators who initially gained fat mass during the exercise intervention but lost fat mass during the follow-up resulting in a net fat mass loss or reversion to baseline reported having continued regular vigorous exercise. Additionally, of the eight compensators who had a net fat mass gain from

baseline to follow-up, not a single one reported having continued vigorous exercise after the conclusion of the exercise intervention.

Therefore, these patterns of fat mass loss in relation to continued physical activity paired with the significantly different between group mean fat mass changes for vigorous exercisers and non-vigorous exercisers, strongly suggest that continuing a regular vigorous exercise program after an exercise intervention produces fat mass loss, regardless of whether subjects were categorized as compensators or non-compensators.

There were four compensators who initially gained fat mass during the exercise intervention and then lost either as much or more fat mass during the one year follow-up period in which they reported that they continued to exercise vigorously. This pattern would suggest that the gain in fat mass was transitory for these subjects who continued regular vigorous exercise. It may be possible that if the five subjects who gained fat mass at both time points (resulting in an average percentage weight gain of  $11.4 \pm 5.0\%$ ) had continued to exercise vigorously post-intervention, that their initial fat mass gain may have been transitory as well, and that they too may have had a net weight and fat mass loss from baseline to follow-up. However further research would need to be performed to confirm this.

The literature has shown some support of transitory mechanisms of compensation. While metabolic factors do contribute to compensation, it is generally accepted that the greatest magnitude and therefore majority of compensation is due to behavioral rather than metabolic factors (King et al., 2007b). Multiple studies have found that increased energy intake substantially contributes to compensation during exercise interventions (King et al., 2007b; J. Stubbs et al., 2011; R. Stubbs et al., 2002; Westerterp et al., 1992;

Woo & Pi-Sunyer, 1985). Stubbs et al. suggest that it would take “a number of weeks” for subjects to become accustomed to increases in ExEE and for increased appetite and EI to reach equilibrium with ExEE (Stubbs, R., et al., 2002).

Additionally, a number of studies have found that a compensatory decrease in NEAT occurs in response to increased ExEE (Church et al. 2009; Manthou et al., 1992) whereas other studies have not made this observation (Hollowell et al., 2009; R. Stubbs et al., 2002; Woo & Pi-Sunyer, 1985). However, Manthou et al. found that compensators, and not non-compensators, had a significant decrease in NEAT during an eight week study, and that this decrease in NEAT was a significant predictor of fat mass changes during the exercise intervention (Manthou et al., 2010). Additionally, Church et al. found that exercising and control groups had no significant step count differences at baseline, but that the control group had a significantly higher step count as compared to all exercise groups during the first four weeks of an exercise intervention. At six month posttesting there was once again no significant difference between the control group nor any of the exercising groups. This pattern suggests that the exercisers had a transitory compensatory decrease in NEAT during the first month of exercise which then normalized by posttesting (Church et al., 2009). Hollowell et al. found no compensatory decrease in NEAT at eight months as compared to baseline testing and had no intermediate time points. These authors suggested that their study (and perhaps a number of other studies) may have been too long to observe this transitory decrease in NEAT. Authors suggested that the decrease in NEAT that was observed during short to mid-length exercise interventions, was perhaps due to a high level of fatigue experienced by previously sedentary individuals whose ExEE was abruptly increased during exercise

interventions. They hypothesized that as subjects adapted to increased exercise volumes over the course of the longer exercise interventions, fatigue, and therefore the compensatory decreases in NEAT would subside and subjects would return to their normal NEAT EEs (Hollowell et al., 2009).

Therefore, the subjects who initially gained weight and fat mass with exercise, but then lost weight and fat mass as they continued to exercise over the follow-up period, may have experienced these transitory compensation mechanisms during the exercise intervention, and then become accustomed to the exercise volumes as they continued to exercise over the follow-up period. As they became accustomed to the new exercise volumes over time, the magnitude of compensation may have decreased allowing them to successfully achieve a negative energy balance through exercise. The subjects that gained fat mass at an accelerated rate during the exercise intervention and then continued to gain weight at a slower rate during the follow-up period (in which they did not continue to vigorously exercise), also experienced compensatory mechanisms during the intervention. However, it is possible that they never became accustomed to the new exercise volume as they did not continue to exercise post-intervention and therefore retained the weight and fat mass that was gained during the intervention, and continued to gain weight and fat mass during the follow-up as would be expected over time in sedentary individuals (Spiriduso, 2010).

If compensators and over-compensators can be identified before the exercise intervention starts, or early on in exercise training, it may be possible to try different methods that may be more effective to combat compensation, possibly preventing excessive weight or fat gain in the early stages of exercise interventions. A number of

reviews have indicated that increasing caloric intake is a significant mechanism by which negative energy balance is counteracted (King et al., 2007b; J. Stubbs et al., 2011; R. Stubbs et al., 2002; Westerterp et al., 1992; Woo & Pi-Sunyer, 1985). While exercise has been found to be effective at producing weight loss while maintaining an ad libitum diet (Donnelly et al., 2003), it has been found that exercise plus caloric restriction produces more significant initial weight loss than either diet or exercise alone (Caplan et al., 2007; Curioni & Lourenco, 2005) and that weight loss is maintained better over one year with both diet and exercise than with either one alone (Caplan et al., 2007). It has also been found that diet and counseling in behavioral modification in conjunction with exercise produces significant initial weight loss which is also sustained better than the counseling or diet and exercise alone (Skender et al., 1996). It may be that compensators need a more multi-factorial approach to initial weight loss, especially an approach that addresses EI which is a significant source of compensation (King et al., 2007b; J. Stubbs et al., 2011; R. Stubbs et al., 2002; Westerterp et al., 1992; Woo & Pi-Sunyer, 1985).

The current study had a secondary aim of determining whether the baseline variables, the compensation variable EEcomp, or early intervention body weight and composition changes could predict body weight and composition changes at the one-year follow-up. There were no baseline variables that significantly correlated with compensation or changes in body weight or composition throughout any time point during the current study. The current study's findings were in contrast to those of Wadden and colleagues who found that baseline body weight predicted weight loss both at the end of a dietary weight loss intervention and at the end of a one year follow-up

period with individuals having higher baseline body weights displaying more weight loss (Wadden et al., 1992).

The magnitude of energy compensation during the 12-week exercise trial was positively correlated with weight change and fat change from baseline to follow-up suggesting that compensators are more likely to gain body weight and fat mass over the 15-month period than non-compensators. Change in weight during the first month of the exercise intervention showed a positive correlation with change in fat mass from baseline to follow-up. Sawyer et al. found that changes in the first month of an exercise intervention correlated with fat mass changes at three months (Sawyer, Manuscript in Preparation), therefore, this study adds that these early changes in weight in response to exercise are indicative not only of short term fat mass change, but also of longer term changes in fat mass of up to at least 15 months.

Change in weight over the whole 12-week exercise intervention period in the current study also positively correlated with change in weight from baseline to follow-up at 15 months. This finding is in agreement with another study which found predictive relationships between changes in weight during the first month of a very low calorie diet and overall weight changes over a one year follow-up period (Wadden et al., 1992). Based upon the results from these two studies, it would appear that early intervention changes in weight predict later body weight changes following both dietary and exercise interventions.

Fat mass changes during the intervention were positively correlated with changes in lean mass from 12-weeks to follow-up indicating that if fat mass is gained, there is an increase in lean mass and vice versa. Additionally, change in lean mass during the

intervention was significantly negatively correlated with changes in lean mass from 12-weeks to follow-up indicating that the significant mean lean mass gain during the intervention was transitory and thus lost during the follow-up period.

The strengths of this study were that all exercise sessions during the 12-week intervention were supervised by laboratory assistants, and subjects achieved 100% adherence to exercise training sessions. Subjects wore a heart rate monitor continuously throughout all training sessions to ensure that exercise intensity goals were met, and exercise testing was performed every four weeks during the intervention in order to adjust target training heart rates as training adaptations occurred. In addition to measuring body weight, the current study also measured lean and fat mass changes via DXA at baseline, 12-weeks, and follow-up, producing a more complete picture of body weight changes. Quantifying the ratio of lean to fat mass change in weight for each individual allowed for energy compensation to be calculated based upon actual changes in body composition on an individual basis. A number of previous studies calculated energy compensation based upon an estimated ratio of lean to fat mass per kilogram of body weight loss which was assumed to be the same for every individual in the sample and ignores established heterogeneity among subjects (Church et al. 2009; King et al., 2007b).

The study also had limitations. Physical activity was measured by self-report via the IPAQ-SF and a second questionnaire specifically written for this study, and not through objective measures. The IPAQ-SF was chosen because it has been reported to be at least as good as other physical activity questionnaires, and has been validated and demonstrated to produce repeatable results (Booth et al., 2003). Also, energy intake was

not monitored, however research has shown that self-reports of EI, like physical activity, are not accurate (Lichtman et al., 1992).

Additionally, between the initial exercise intervention, and the follow-up study, the lab in which the research was performed upgraded from the Prodigy DXA to a new iDXA machine. Hull et al. recommended that cross-calibration equations should be used to translate the older Prodigy DXA values into iDXA values in order to ensure consistency and comparability of data taken from two different machines (Hull et al., 2009). Unfortunately, equations were produced for only fat and lean mass and not percent fat and percent lean mass, which makes it difficult to relate body weight with fat mass and lean mass measures. However, taken independently of each other both weight and lean and fat mass data can be considered consistent with readings from baseline to follow-up after conversion (Hull et al., 2009).

An additional limitation is that only 24 of the original 82 women that completed the 12-week intervention came back to complete the follow-up study despite all women being contacted. It is unknown if this may be a truly representative sample of the original subjects or if subjects may have decided whether or not to participate based on body composition and or physical activity maintenance since the conclusion of the exercise intervention. This smaller sample made the n, especially for non-compensators (n=9) and those who continued vigorous exercise post-intervention (n=8) relatively small. At the completion of the exercise intervention, the follow-up study had not yet been planned, so subjects were not informed about it, and therefore did not agree to participate in the follow-up study in advance.



It should be pointed out that the initial exercise intervention was not intended as a weight loss trial. The study involved 90 minutes per week of vigorous exercise, which is above the ACSM recommendations to improve health outcomes in apparently healthy adults (Garber et al., 2011), but did not meet ACSM recommendations for weight loss (Donnelly et al., 2009). It is likely that if a higher amount of exercise were prescribed, a greater magnitude of weight loss would have been induced, but because the purpose of this study was to investigate compensation, the exercise prescription was sufficient since it produced a prevalence of 62.5% compensation in the study population.

In conclusion, the main finding of this study is that following a 12-week supervised exercise intervention, slightly overweight, previously sedentary, young to middle aged women displayed a net loss of fat mass during the follow-up period if regular vigorous exercise was continued upon the conclusion of the exercise intervention, regardless of whether or not they were compensators or non-compensators. However, compensators were less likely to continue regular vigorous physical activity post-intervention. Additionally, energy compensation from baseline to 12-weeks and early body weight and composition changes during the intervention were moderate predictors of body weight and composition changes from baseline to follow-up.

In future research a larger scale study with a control group in which the follow-up study is a planned part of the initial exercise intervention should be performed. The study should use objective measures of NEAT such as accelerometers or pedometers, and constantly worn heart rate monitors to determine ExEE at regular intervals throughout the one year follow-up to determine maintenance of physical activity throughout this period. Additionally, it may be useful to monitor EI intake, to determine the magnitude of

increased EI in the over-compensation of subjects who gain weight in response to exercise. Future researchers may also wish to choose an exercise volume that is consistent with recommendations for weight loss (Donnelly et al., 2009), and examine the effects of exercise intensity, especially vigorous exercise, on weight loss maintenance and physical activity maintenance post-intervention.

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APPENDIX A  
CONSENT FORM

## **CONSENT FORM**

### **Body Composition and Physical Activity Level Maintenance One Year After a 12-week Exercise Intervention in Women**

#### **INTRODUCTION**

The purposes of this form are 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 and to record the consent of those who agree to be involved in the study.

#### **RESEARCHERS**

Glenn Gaesser, PhD, a professor in the Exercise Wellness Program, Healthy Lifestyles Research Center, Clarissa Cabbage, Brandon Sawyer, Dharini Bhammar, and Wesley Tucker, graduate students, and Lindsey Pilbeam, an x-ray technician, at Arizona State University, have requested your participation in a research study.

#### **STUDY PURPOSE**

The purpose of this research study is to evaluate whether changes in body composition and physical activity levels achieved during a supervised 12-week exercise intervention were maintained one year following the conclusion of the intervention.

#### **DESCRIPTION OF THE STUDY**

If you decide to participate, then as a study participant you will be required to come to the ASU Polytechnic campus on one occasion for approximately one hour.

You will be asked eight questions about your current physical activity and dieting habits and about these habits within the past year.

You will also fill out a seven question International Physical Activity Questionnaire (IPAQ) regarding your current physical activity status.

Following the completion of the questionnaires the following measurements will be taken:

#### **Anthropometric Measurements**

Your height and weight will be measured using a standard scale and stadiometer.

#### **Dual X-ray Absorptiometry (DEXA)**

You will report to the ISTB3 building on the Polytechnic Campus to complete your pre testing. Your body composition (relative amounts of fat and lean tissue) will be determined by using an FDA-approved bone density measurement machine. The procedure is called Dual Energy X-ray Absorptiometry (DEXA). You will be asked to lie face up, on a padded table for 7 minutes while the scanner arm of the DEXA machine passes over your entire body. The scanner will not enclose you or touch you, and you can wear your regular clothing (no metal allowed).



You will be exposed to minimal radiation (1-4 microSieverts) that is within an acceptable range as provided by the US FDA. Anytime you are exposed to radiation there is potential risk. The amount of radiation (1-4 microSieverts) that you would be exposed to is quite minimal. For example, you would receive radiation exposure of approximately 80 microSieverts on a transatlantic airline flight of 8 hours, 50 microSieverts living in Denver, Colorado, at an elevation of 5,000 feet for approximately 4 weeks, or 30 to 40 microSieverts during a typical chest x-ray.

If there is ANY chance of being pregnant then you should not undergo DEXA scanning. Also if you have undergone any contrast procedures within the past 7 days you will not be able to participate at this time. You will need to complete a urine pregnancy test when you arrive to the laboratory before you undergo the DEXA scan. You will only be allowed to undergo the DEXA scan if the pregnancy test result is negative. A certified X-ray technician will complete all DEXA scans. This test takes about 15 minutes.

### **POSSIBLE RISKS**

There is a very small risk of a false negative pregnancy test resulting in radiation to your fetus. This risk is minimized by confirmation of a negative pregnancy test before you undergo the DEXA scan. Furthermore, the urine pregnancy tests used in this study are ~99% accurate, therefore the risk of a false negative is very small.

### **BENEFITS**

There may be no direct benefits to you for your participation in this study. However, you will receive a free DEXA scan informing you of your body composition.

### **NEW INFORMATION**

If the researchers find new information during the study that would reasonably change your decision about participating, then they will provide this information to you.

### **CONFIDENTIALITY**

All information obtained in this study is strictly confidential unless disclosure is required by law. The results of this research study may be used in a Masters thesis, reports, presentations, and publications, but your name or identity will not be disclosed. In order to maintain confidentiality of your records, Dr. Gaesser will use subject codes on all data collected, maintain a master list separate and secure from all data collected, and limit access to all confidential information to the study investigators. All data will be secured in locked filing cabinets in the ISTB3 building within the testing laboratory.

### **WITHDRAWAL PRIVILEGE**

It is ok for you to say no. Even if you say yes now, you are free to say no later, and withdraw from the study at any time.

Your decision will not affect your relationship with Arizona State University or otherwise cause a loss of benefits to which you might otherwise be entitled.

Your participation is voluntary and if you decide not to participate or decide to withdraw from the study it will not affect your grade, treatment, care, or employment status.

### **COSTS AND PAYMENTS**

All study procedures will be provided to you at no personal cost.

On completion of your one visit to the Polytechnic Campus you will be given a ten dollar gift certificate to Starbucks.

### **VOLUNTARY CONSENT**

Any questions you have concerning the research study or your participation in the study, before or after your consent, will be answered by Dr. Glenn Gaesser (480-727-1944). If you have questions about your rights as a subject/participant in this research, or if you feel you have been placed at risk, you can contact the Chair of the Human Subjects Institutional Review Board, through the ASU Research Compliance Office, at 480-965 6788.

This form explains the nature, demands, benefits and any risks of the project. By signing this form you agree knowingly to assume any risks involved. Remember, your participation is voluntary. You may choose not to participate or to withdraw your consent and discontinue participation at any time without penalty or loss of benefit. In signing this consent form you are not waiving any legal claims, rights, or remedies. A copy of this consent form will be given to you.

Your signature below indicates that you consent to participate in the above study.

\_\_\_\_\_  
Subject's Signature

\_\_\_\_\_  
Printed Name

\_\_\_\_\_  
Date

### **INVESTIGATOR'S STATEMENT**

"I certify that I have explained to the above individual the nature and purpose, the potential benefits and possible risks associated with participation in this research study, have answered any questions that have been raised, and have witnessed the above signature. These elements of Informed Consent conform to the Assurance given by Arizona State University to the Office for Human Research Protections to protect the rights of human subjects. I have provided the subject/participant a copy of this signed consent document."

Signature of Investigator \_\_\_\_\_ Date \_\_\_\_\_

APPENDIX B

PHYSICAL ACTIVITY AND DIET QUESTIONNAIRES

**INTERNATIONAL PHYSICAL ACTIVITY QUESTIONNAIRE**  
**(August 2002)**  
**SHORT LAST 7 DAYS SELF-ADMINISTERED FORMAT**

FOR USE WITH YOUNG AND MIDDLE-AGED ADULTS (15-69 years)

The International Physical Activity Questionnaires (IPAQ) comprises a set of 4 questionnaires. Long (5 activity domains asked independently) and short (4 generic items) versions for use by either telephone or self-administered methods are available. The purpose of the questionnaires is to provide common instruments that can be used to obtain internationally comparable data on health-related physical activity.

***Background on IPAQ***

The development of an international measure for physical activity commenced in Geneva in 1998 and was followed by extensive reliability and validity testing undertaken across 12 countries (14 sites) during 2000. The final results suggest that these measures have acceptable measurement properties for use in many settings and in different languages, and are suitable for national population-based prevalence studies of participation in physical activity.

***Using IPAQ***

Use of the IPAQ instruments for monitoring and research purposes is encouraged. It is recommended that no changes be made to the order or wording of the questions as this will affect the psychometric properties of the instruments.

***Translation from English and Cultural Adaptation***

Translation from English is supported to facilitate worldwide use of IPAQ. Information on the availability of IPAQ in different languages can be obtained at [www.ipaq.ki.se](http://www.ipaq.ki.se). If a new translation is undertaken we highly recommend using the prescribed back translation methods available on the IPAQ website. If possible please consider making your translated version of IPAQ available to others by contributing it to the IPAQ website. Further details on translation and cultural adaptation can be downloaded from the website.

***Further Developments of IPAQ***

International collaboration on IPAQ is on-going and an ***International Physical Activity Prevalence Study*** is in progress. For further information see the IPAQ website.

***More Information***

More detailed information on the IPAQ process and the research methods used in the development of IPAQ instruments is available at [www.ipaq.ki.se](http://www.ipaq.ki.se) and Booth, M.L. (2000). *Assessment of Physical Activity: An International Perspective*. Research Quarterly for Exercise and Sport, 71 (2): s114-20. Other scientific publications and presentations on the use of IPAQ are summarized on the website.

## INTERNATIONAL PHYSICAL ACTIVITY QUESTIONNAIRE

We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the **last 7 days**. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the **vigorous** activities that you did in the **last 7 days**. **Vigorous** physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Think *only* about those physical activities that you did for at least 10 minutes at a time.

1. During the **last 7 days**, on how many days did you do **vigorous** physical activities like heavy lifting, digging, aerobics, or fast bicycling?

\_\_\_\_\_ **days per week**

☐

No vigorous physical activities



*Skip to question 3*

2. How much time did you usually spend doing **vigorous** physical activities on one of those days?

\_\_\_\_\_ **hours per day**

\_\_\_\_\_ **minutes per day**

☐

Don't know/Not sure

Think about all the **moderate** activities that you did in the **last 7 days**. **Moderate** activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal. Think *only* about those physical activities that you did for at least 10 minutes at a time.

3. During the **last 7 days**, on how many days did you do **moderate** physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

\_\_\_\_\_ **days per week**

☐

No moderate physical activities



*Skip to question 5*

4. How much time did you usually spend doing **moderate** physical activities on one of those days?

\_\_\_\_\_ **hours per day**  
\_\_\_\_\_ **minutes per day**

☐ Don't know/Not sure

Think about the time you spent **walking** in the **last 7 days**. This includes at work and at home, walking to travel from place to place, and any other walking that you might do solely for recreation, sport, exercise, or leisure.

5. During the **last 7 days**, on how many days did you **walk** for at least 10 minutes at a time?

\_\_\_\_\_ **days per week**

☐ No walking      ➔ *Skip to question 7*

6. How much time did you usually spend **walking** on one of those days?

\_\_\_\_\_ **hours per day**  
\_\_\_\_\_ **minutes per day**

☐ Don't know/Not sure

The last question is about the time you spent **sitting** on weekdays during the **last 7 days**. Include time spent at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.

7. During the **last 7 days**, how much time did you spend **sitting** on a **week day**?

\_\_\_\_\_ **hours per day**  
\_\_\_\_\_ **minutes per day**

☐ Don't know/Not sure

**This is the end of the questionnaire, thank you for participating.**

EasyTone Follow-Up Study  
Additional Questions

1. Are you currently engaged in a regular vigorous exercise program, in which you perform intense aerobic or resistance exercise training on most days of the week?
2. Are you currently training for a specific athletic event, such as an endurance race?
3. Do you classify yourself as a complete couch potato, with absolutely no leisure-time physical activity, and no interest in engaging in regular exercise?
4. Are you currently dieting or trying to lose weight?
5. After the conclusion of the initial study, did you continue to exercise on a regular basis?
6. Have you been on a diet, or any weight loss program, within the last year?
7. Have you started/continued to wear exercise “toning” shoes of any type since the conclusion of the study?
8. After the conclusion of the initial study, would you say that you returned to the exercise/physical activity habits that you had prior to starting the initial study?