Predictors of Fat Mass Changes in Response to Aerobic Exercise Training in Women

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Predictors of Fat Mass Changes in Response to Aerobic Exercise Training in Women

Brandon J. Sawyer, Dharini M. Bhammar, Siddhartha S. Angadi, Dana M. Ryan, Justin R. Ryder, Elizabeth J. Sussman, Farryl M.W. Bertmann, and Glenn A. Gaesser

School of Nutrition and Health Promotion, Healthy Lifestyles Research Center, Arizona State University, Phoenix, Arizona

Abstract

Sawyer, BJ, Bhammar, DM, Angadi, SS, Ryan, DM, Ryder, JR, Sussman, EJ, Bertmann, FMW, and Gaesser, GA. Predictors of fat mass changes in response to aerobic exercise training in women. J Strength Cond Res 29(2): 297–304, 2015—Aerobic exercise training in women typically results in minimal fat loss, with considerable individual variability. We hypothesized that women with higher baseline body fat would lose more body fat in response to exercise training and that early fat loss would predict final fat loss. Eighty-one sedentary premenopausal women (age: 30.7 ± 7.8 years; height: 164.5 ± 7.4 cm; weight: 68.2 ± 16.4 kg; fat percent: 38.1 ± 8.8%) underwent supervised treadmill walking 3 days per week for 30 minutes at 70% of VO2peak. Overall, women did not lose body weight or fat mass. However, considerable individual variability was observed for changes in body weight (−11.7 to +4.8 kg) and fat mass (−11.8 to +3.7 kg). Fifty-five women were classified as compensators and, as a group, gained fat mass (25.6 ± 11.1 kg to 26.1 ± 11.3 kg; p < 0.001). The strongest correlates of change in body fat at 12 weeks were change in body weight (r = 0.52) and fat mass (r = 0.48) at 4 weeks. Stepwise regression analysis that included change in body weight and body fat at 4 weeks and submaximal exercise energy expenditure yielded a prediction model that explained 37% of the variance in fat mass change (R2 = 0.37, p < 0.001). Change in body weight and fat mass at 4 weeks were moderate predictors of fat loss and may potentially be useful for identification of individuals who achieve less than expected weight loss or experience unintended fat gain in response to exercise training.

Key Words: weight loss, compensation, heterogeneity, walking, public health guidelines, vigorous-intensity exercise

Introduction

Exercise training without caloric restriction is not considered a very effective means of weight loss on a group level (8,18,34). After exercise training, the magnitude of weight loss typically is much less than that predicted from the cumulative energy expenditure of the exercise training sessions (34). Although mean weight or body fat loss is relatively small, considerable variability in weight change occurs at the individual level (1–3,6,7,18–22,24,28,31,33,34). In fact, a significant percentage of women may actually gain body weight or fat mass in response to the exercise training (6,8,22,24). The marked heterogeneity in individual responses to exercise training reveals responders (or noncompensators), who lose at least as much weight or body fat as expected from the energy expended during training sessions, and nonresponders (or compensators), who either lose less weight or body fat than expected or actually gain weight or body fat during exercise training (1,3,6,8,18–22,24,28,34).

Reasons for the heterogeneous weight loss response to exercise are largely unknown. Downregulation of free-living spontaneous physical activity during a structured exercise intervention has been reported in some studies (4,13,24,27,29), but not in others (15,25). Individual variability in energy intake in response to exercise training could also contribute to the heterogeneity of exercise training-induced weight loss (2,18,33,41,43,44). Variation in subject adherence to exercise training could also play a role (16).

Preintervention body weight and body fat are the only baseline characteristics that have been shown to be even moderately correlated with overall weight or fat change in response to exercise training (9,18,19,41) or caloric restriction (14,30,36,38–40), with higher baseline body weight generally associated with greater weight loss. During caloric restriction, early weight loss has been shown to predict subsequent weight loss (32). It is not known whether this holds true for exercise programs. Such information might be useful in exercise programs that have weight loss as a primary outcome and in interventions with the intention to improve overall health.

Therefore, the primary purposes of this study were to assess the effect of baseline body weight and composition on body fat change in response to a 12-week aerobic exercise...
program in premenopausal women and to determine whether changes in body weight or composition during the first 4 weeks of the program were predictive of changes at the end of the program. We hypothesized that women with higher baseline body fat percentage would lose more body fat and would be less likely to compensate than leaner women and that early fat loss would predict final fat loss.

A secondary aim was to determine if any baseline exercise testing variables predict energy compensation during exercise training. Although aerobic capacity has been reported to be similar in compensators and non-compensators (24), whether other physiologic responses to exercise (e.g., pulmonary ventilation, energy expenditure as an index of economy) are predictive of weight loss or energy compensation has not been reported. Such information could potentially be useful for identification of individuals who achieve less than expected weight loss or experience unintended fat gain in response to exercise training. A recent review recommended that identification of individuals who are susceptible to compensation should be a high priority for researchers and clinicians (28).

METHODS

Experimental Approach to the Problem
We conducted a 12-week supervised aerobic exercise training program. The study was designed as an exercise-only program to evaluate the body fat changes that occur in response to an aerobic exercise dose slightly above the current minimum public health recommendations for vigorous-intensity exercise (12). Although higher exercise doses may be necessary for significant weight loss (5), most U.S. adults do not meet even the minimum public health recommendation for either moderate- or vigorous-intensity activity (37). Therefore, we felt that it would be important to test our hypotheses with a more modest, and possibly more attainable, exercise dose. A priori power calculations using previous data (36) showed that to achieve statistical significance with a 3-predictor regression model that explained at least 20% of the variance in our dependent variable (change in fat mass), we would need 73 subjects to achieve 90% power. We overenrolled by 9 subjects to account for subject attrition. Similar to other studies (2,4,20–22,24), we chose aerobic exercise as our independent variable with no control group because significant fat change is not expected over a 12-week period without lifestyle change. Our primary dependent variable was change in body fat mass.

Subjects
Eighty-two women, aged 19-45 years, read and signed an informed consent form approved by the Arizona State University institutional review board. Of these 82 women, 81 completed all 36 exercise training sessions (described below) and all the requirements of the study and were therefore included in the analyses. Subjects were non-smoking, nonpregnant healthy women who were not currently, or within the previous 3 months, meeting the public health guidelines for physical activity (37) or trying to lose weight. We ensured through written questionnaire that all subjects were not engaged in any regular (more than 1–2 days per week) aerobic or resistance exercise training, not currently training for a specific athletic event, and not dieting. Furthermore, subjects were required to be willing to not change their current diet and eating habits for the course of the study. Other exclusion criteria included hypertension, history of cardiac arrhythmia, stroke, myocardial infarction, or current use of heart medications. All participants were asked to abstain from other activities outside of the study and to not make any changes to dietary intake. Participants were frequently reminded to not change diet or physical activity routines throughout the course of the study.

Procedures
Height was assessed by a standing wall-mounted stadiometer (Seca, Chino, CA, USA). Weight was assessed

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>30.7 ± 7.8</td>
<td>30.7 ± 7.8</td>
</tr>
<tr>
<td>BMI (kg·m⁻²)</td>
<td>25.2 ± 5.7</td>
<td>25.2 ± 5.6</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>68.2 ± 16.4</td>
<td>68.2 ± 16.3</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>38.1 ± 8.8</td>
<td>37.6 ± 8.8</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>27.0 ± 11.5</td>
<td>26.6 ± 11.4</td>
</tr>
<tr>
<td>Lean mass (kg)</td>
<td>41.2 ± 7.0</td>
<td>41.6 ± 7.0†</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>84.8 ± 13.1</td>
<td>84.4 ± 12.6</td>
</tr>
<tr>
<td>VO₂peak (ml·kg⁻¹·min⁻¹)</td>
<td>30.0 ± 5.7</td>
<td>32.4 ± 6.0†</td>
</tr>
</tbody>
</table>

*BMI = body mass index. Values represent mean ± SD. †Significant difference pre to post, p ≤ 0.05.
using a calibrated electronic scale (COSMED, Rome, Italy) with subjects in a bathing suit. Waist circumference was taken at the umbilicus using a standard Gulick tape measure. The same research assistant took all the circumference measurements. Body composition was determined at baseline and 12 weeks using dual-energy x-ray absorptiometry (DXA, Lunar Prodigy; GE Medical Systems Lunar, Madison, WI, USA). All DXA scans were analyzed using enCORE 2011 software, version 13.6, to determine total percentage of fat and lean tissue. A certified radiology technician calibrated the DXA before each day of testing and performed all DXA scans. Subjects wore no metal and removed all jewelry before each test. Dual-energy x-ray absorptiometry has been shown to be highly reliable (coefficient of variation ranging from 0.8 to 2.8%) and valid (1–2% difference from the 4-compartment model) for measuring body fat and for detecting small changes in body fat (35).

A modified Balke incremental exercise test was used at baseline and after 4, 8, and 12 weeks of exercise training to assess VO₂peak. A Parvomedics TrueMax 2400 metabolic cart (Parvomedics, Sandy, UT, USA) and Polar heart rate monitor (Polar, Lake Success, NY, USA) were used to measure gas exchange, pulmonary ventilation (VE), and heart rate continuously. The protocol consisted of 1 minute at 1.5 mph and 0% incline, followed by an increase in speed to 3.3 mph for 1 minute. After 1 minute at 3.3 mph and 0% incline, the treadmill grade increased to 2% and then increased again 1% each minute until volitional exhaustion was reached. Verbal encouragement was given throughout each test. The metabolic cart was calibrated before each test or every 4 hours per manufacturer recommendation. Peak oxygen uptake was calculated by taking average of the 2 highest consecutive 15-second VO₂ values. The VO₂-heart rate relationship was used to identify the heart rate that elicited 70% of VO₂peak at each testing period. The ventilation threshold was used to ensure that our VE and energy expenditure values were below the ventilation threshold. Body weight was also assessed at each of these visits.

The exercise program consisted of 12 weeks of treadmill walking at a heart rate that elicited approximately 70% of VO₂peak (~80% of HRmax) 3 days a week for 30 minutes. Each training session included a 5-minute warm-up and 5-minute cooldown. All subjects completed 36 supervised exercise sessions over the 12-week period. Exercise sessions were completed on nonconsecutive days. Treadmill speed stayed constant throughout the 12 weeks, and incline was increased to elicit the desired heart rate. Based on VO₂peak changes at 4 and 8 weeks, target HR was adjusted to elicit 70% of the new VO₂peak. To ensure that the target intensity was maintained during each training ventilation and gas exchange as previously described (11). Submaximal VE and energy expenditure were calculated using minutes 2–4 of the VO₂peak test. The ventilation threshold was used to ensure that our VE and energy expenditure values were below the ventilation threshold. Body weight was also assessed at each of these visits.

Figure 1. Individual changes in (A) total mass, (B) fat mass, and (C) lean mass (kg). Each subject is in the same position for each panel. Solid bars indicate compensators and hollow bars indicate noncompensators (see text for definitions).
session, subjects wore a Polar heart rate monitor and recorded their heart rate every 5 minutes under the supervision of research assistants.

**Statistical Analyses**

Total exercise energy expenditure (ExEE) for the 12-week study was estimated using 70% of $\text{VO}_2\text{peak}$, adjusted every 4 weeks, minus predicted resting $\text{VO}_2$ (using 3.5 ml·kg$^{-1}$·min$^{-1}$), and an assumed respiratory exchange ratio of 0.85 (i.e., 4.86 kcal·L$^{-1}$ of O$_2$) using the equation below:

$$\text{ExEE} = \left(0.7 \times \text{VO}_2\text{peak} \times \text{L}\cdot\text{min}^{-1} - \text{VO}_2\text{resting}\right) \times 4.86 \text{ kcal}\cdot\text{L}^{-1}\cdot\text{min}^{-30 \text{ min} \times 36 \text{ exercise sessions}}.$$

Based on body composition changes after the 12-week exercise program, standard values for the energy equivalents of fat (9,540 kcal·kg$^{-1}$) and lean tissue (1,100 kcal·kg$^{-1}$) (20) were used to determine energy balance (EB):

$$\text{EB} = \left(\Delta\text{lean mass} \times 1.100 \text{ kcal\cdotkg}^{-1}\right) + \left(\Delta\text{fat mass} \times 9.540 \text{ kcal\cdotkg}^{-1}\right).$$

Therefore, total energy compensation (EEcomp, in kilocalories) was calculated by the sum of ExEE and EB. Subjects who had positive values for EEcomp were considered compensators and those with negative values were considered noncompensators. All statistical analyses were performed using SPSS Statistics 20 (IBM, Armonk, NJ, USA). Independent samples t-tests were used to compare baseline characteristics between groups. A 2-way repeated-measures analysis of variance was conducted to detect group, time, and group × time differences in each outcome variable. Paired samples t-tests were used to detect significant changes in the outcome variables in the groups separately. We conducted a Holm-Bonferroni sequential correction on the p values from t-tests to correct for p value inflation. Pearson’s correlations were used to determine the relationships between fat mass change and other variables. Stepwise multiple linear regression analysis was used to determine predictors of fat mass change. All dependent variables were checked for normality using the Shapiro-Wilk test. An alpha level of p ≤ 0.05 was considered statistically significant.

**RESULTS**

**Changes in Body Composition**

Results of the Shapiro-Wilk test showed that all dependent variables were normally distributed, therefore meeting the assumptions of the parametric statistics used. Overall, there were no significant reductions in body weight, body fat, or waist circumference. Conversely, there was a significant

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**Table 2. Outcome variables comparing the compensators and the noncompensators.**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Compensators (N = 55)</th>
<th>Noncompensators (N = 26)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Age (y)</td>
<td>30.7 ± 7.6</td>
<td>30.7 ± 7.6</td>
</tr>
<tr>
<td>BMI (kg·m$^{-2}$)</td>
<td>24.3 ± 5.2</td>
<td>24.8 ± 5.2†</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>66.3 ± 16.5</td>
<td>67.1 ± 16.5†</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>37.3 ± 8.3</td>
<td>37.7 ± 8.3†</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>26.6 ± 11.1</td>
<td>26.1 ± 11.3†</td>
</tr>
<tr>
<td>Lean mass (kg)</td>
<td>40.7 ± 7.1</td>
<td>40.9 ± 7.0</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>83.1 ± 11.9</td>
<td>83.7 ± 11.9</td>
</tr>
<tr>
<td>$\text{VO}_2\text{peak}$ (ml·kg$^{-1}$·min$^{-1}$)</td>
<td>30.8 ± 6.01</td>
<td>33.4 ± 6.4†</td>
</tr>
</tbody>
</table>

*BM = body mass index. Values represent mean ± SD.
†Significant change pre to post (p ≤ 0.05).
‡Significant group × time interaction.
increase in lean body mass (Table 1). More importantly, there was considerable individual variability in the changes in body weight (−11.7 to +4.8 kg), fat mass (−11.8 to +3.7 kg), and lean mass (−3.6 to +4.0 kg) (Figure 1).

Fifty-five (68%) of the 81 women were classified as compensators, i.e., having an energy value of fat and lean body mass changes that was less than the net ExEE (Table 2 and Figure 1). There were no significant differences between compensators and noncompensators for baseline body weight, body mass index, fat mass, body fat percentage, lean mass, waist circumference, or \( V_{\text{O}_2} \text{peak} \) (Table 2). Net ExEE did not differ between the 2 groups. Peak oxygen uptake increased in both compensators and noncompensators (\( p, 0.01 \)), but there was no group \( \times \) time interaction in the \( V_{\text{O}_2} \text{peak} \) change (\( p = 0.65 \)). There were significant group \( \times \) time interactions for body weight, body mass index, fat mass, and body fat percentage because of decreases in noncompensators and increases in compensators (Table 2).

### Predictors of Fat Loss

Baseline body weight, fat mass, body fat percentage, lean mass, \( V_{\text{O}_2} \text{peak} \) and net ExEE were not significantly correlated with change in fat mass (Table 3). However, baseline submaximal exercise \( V_{\text{E}} \) (\( r = -0.25 \)) and energy expenditure (in kilocalories per minute) (\( r = -0.23 \)) during minutes 2–4 of the baseline \( V_{\text{O}_2} \text{peak} \) test were significantly inversely correlated with the change in fat mass at 12 weeks (Table 3). Changes in body weight (\( r = 0.52 \)), fat mass (\( r = 0.48 \)), and body fat percentage (\( r = 0.27 \)) at 4 weeks were also significantly correlated with change in fat mass at 12 weeks. Stepwise regression analysis that included all variables that were significantly correlated with change in fat mass at 12 weeks yielded a prediction model that explained 37% of the

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**Table 3. Correlations between change in body fat mass (kg) at 12 weeks and other variables.**

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Change in fat (kg), Pearson’s ( R )</th>
<th>Significance (( p ))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.14</td>
<td>0.21</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>-0.16</td>
<td>0.15</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>-0.15</td>
<td>0.18</td>
</tr>
<tr>
<td>Body fat (kg)</td>
<td>-0.17</td>
<td>0.13</td>
</tr>
<tr>
<td>Lean mass (kg)</td>
<td>-0.10</td>
<td>0.37</td>
</tr>
<tr>
<td>( V_{\text{O}_2} \text{peak (ml kg}^{-1} \text{ min}^{-1}) )</td>
<td>0.15</td>
<td>0.18</td>
</tr>
<tr>
<td>Submaximal exercise ventilation (L min^{-1})</td>
<td>-0.25*</td>
<td>0.03</td>
</tr>
<tr>
<td>Submaximal exercise energy expenditure (kcal min^{-1})</td>
<td>-0.23*</td>
<td>0.04</td>
</tr>
<tr>
<td>Change in body weight (kg) at 4 wk</td>
<td>0.52*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Change in body fat (%) at 4 wk</td>
<td>0.27*</td>
<td>0.01</td>
</tr>
<tr>
<td>Change in body fat (kg) at 4 wk</td>
<td>0.48*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Change in lean mass (kg) at 4 wk</td>
<td>0.10</td>
<td>0.39</td>
</tr>
<tr>
<td>Net exercise training energy expenditure (kcal)</td>
<td>-0.03</td>
<td>0.81</td>
</tr>
</tbody>
</table>

\( * \)Significant correlation, \( p \leq 0.05 \).

---

**Table 4. Predictors of change in body fat mass (kg) at 12 weeks.**

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Coefficient (( \beta ))</th>
<th>SE</th>
<th>( R^2 ) change</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stepwise regression (( r = 0.61, R^2 = 0.37, p &lt; 0.001 ))</td>
<td>1.566</td>
<td>0.927</td>
<td>0.10</td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Variables included in final model</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change in body weight at 4 wk</td>
<td>0.493</td>
<td>0.172</td>
<td>0.27</td>
<td>0.01</td>
</tr>
<tr>
<td>Change in fat mass at 4 wk</td>
<td>0.480</td>
<td>0.187</td>
<td>0.05</td>
<td>0.01</td>
</tr>
<tr>
<td>Submaximal exercise energy expenditure</td>
<td>-0.438</td>
<td>0.177</td>
<td>0.05</td>
<td>0.02</td>
</tr>
<tr>
<td>Variables excluded</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Submaximal exercise ventilation</td>
<td>-0.750</td>
<td></td>
<td></td>
<td>0.68</td>
</tr>
<tr>
<td>Change in body fat percentage</td>
<td>-0.629</td>
<td></td>
<td></td>
<td>0.10</td>
</tr>
</tbody>
</table>
variance in fat mass change ($R^2 = 0.37, p < 0.001$) (Table 4). Changes in body weight and body fat mass at 4 weeks, as well as submaximal ExEE, were included in the final model, whereas submaximal exercise ventilation and change in body fat percentage at 4 weeks were excluded (Table 4).

**DISCUSSION**

Contrary to our hypothesis, fat mass change after 12 weeks of exercise training in premenopausal women was not related to initial body weight or fat mass. A number of exercise and caloric restriction studies reported that loss of body weight or fat was positively correlated with baseline body fat (9,14,17–19,40). Our results show that no baseline anthropometric measure was a significant predictor of fat loss. One potentially important finding was that the changes in body weight and fat mass at 4 weeks were moderately correlated ($r = 0.52$ and 0.48, respectively) with change in body fat mass at 12 weeks. During energy-restricted weight loss programs, early weight loss has been reported to be a significant predictor of subsequent weight loss (32). Our results suggest that this may also be true for exercise training. If so, monitoring weight or fat changes during the initial weeks of an exercise program might enable identification of potential compensators. Additional research may be able to demonstrate feasibility of using data on changes in body weight and body fat to address compensation and perhaps reduce overcompensation (i.e., unintended gain in body fat) with exercise training. In our study, compensators comprised approximately two-thirds of our study sample and, as a group, actually gained fat mass during exercise training (Table 2). As suggested in a recent review on resistance to exercise-induced weight loss (e.g., 7,700 kcal·kg$^{-1}$·wk$^{-1}$) does not reflect the considerable individual variability in changes in both fat mass and lean body mass after exercise training (Figure 1). Although higher levels of net ExEE can produce greater weight loss and reduce the number of individuals who actually gain body weight or body fat (2,7), considerable individual variability remains.

The strengths of our study include the fact that exercise training sessions were supervised with intensity monitored continuously by heart rate monitors, aerobic capacity was assessed every 4 weeks to adjust training HR accordingly, and there was 100% adherence to exercise sessions. Additionally, energy compensation was determined by using actual changes in fat and lean mass, rather than estimated on the basis of assumed composition of weight loss (10,42).

Our study also has limitations. We did not assess energy intake, although we instructed our subjects throughout the study not intentionally change their diet. Compensators may have increased energy intake in response to exercise training, as has been reported by King et al. (19). However, most studies show that marked individual variability in body weight or body fat changes in response to exercise training is not predicted by changes in energy intake (2,3,6,7,22,24).
Even studies using very detailed and rigorous 7-day food records (24) and multiple dietary assessments throughout the intervention (7,22) did not show any difference in dietary intake between compensators and noncompensators. These findings may reflect the fact that self-report dietary instruments have been shown to be unreliable for accurate quantitative assessment of energy intake (23). We also did not monitor free-living physical activity outside our formal exercise training. The exercise dose in our study was below the recommended level for weight loss (5), resulting in a relatively low net ExEE. However, our exercise dose of 90 minutes per week of vigorous-intensity exercise is higher than the minimum recommendation for adults of 75 minutes per week of vigorous-intensity activity (12). Furthermore, because our exercise dose produced considerable heterogeneity in weight change in response to exercise training, our study design was sufficient to test our hypotheses.

In conclusion, our results demonstrate considerable individual variability in body weight and composition changes in response to aerobic exercise training in premenopausal women and that changes in fat mass were not predicted by baseline body weight or body fat, aerobic fitness level, or net ExEE. Changes in fat mass after 12 weeks of exercise training were, however, moderately correlated with changes in body weight and fat mass at 4 weeks and less so with change in body fat percentage at 4 weeks and with baseline submaximal energy V̇\textsubscript{E} and energy expenditure. Further research is necessary to determine whether knowledge of these predictors can be used to enhance effectiveness of aerobic exercise for weight control. The fact that two-thirds of the women demonstrated compensation, and as a group gained fat mass after an exercise program consistent with current public health recommendations (12), suggests that this should be a high priority.

**Practical Applications**

Gain in body weight or body fat with aerobic exercise training is not considered a desirable outcome, yet is a consistent observation in controlled research settings and thus may be a common occurrence in clinical and professional practice. Since the change in body weight at 4 weeks was the strongest single correlate of body fat change after 12 weeks of exercise training, monitoring of body weight changes during the initial weeks of exercise training could prove to be useful in helping prevent unintended weight/fat gain in women who initiate an aerobic exercise program. Body weight is easy to measure and has the advantage over other predictors that may not be feasible outside a research setting (e.g., body composition testing, indirect calorimetry for assessment of energy expenditure) and therefore could potentially be used by coaches, personal trainers, and other professionals to assist individuals in achieving their weight loss goals and avoid unintended fat gain. Early identification of compensators through monitoring of body weight changes could lead to individualization of exercise programs to specifically fit the needs of each individual and prevent exercise-induced fat gain.

**Acknowledgments**

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**References**


