Nonprescribed physical activity energy expenditure is maintained with structured exercise and implicates a compensatory increase in energy intake\(^1\)–\(^3\)

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**ABSTRACT**

**Background:** Exercise interventions elicit only modest weight loss, which might reflect a compensatory reduction in nonprescribed physical activity energy expenditure (PAEE).

**Objective:** The objective was to investigate whether there is a reduction in nonprescribed PAEE as a result of participation in a 6-mo structured exercise intervention in middle-aged men.

**Design:** Sedentary male participants [age: 54 ± 5 y; body mass index (in kg/m\(^2\)): 28 ± 3] were randomly assigned to a 6-mo progressive exercise (EX) or control (CON) group. Energy expenditure during structured exercise (prescribed PAEE) and nonprescribed PAEE were determined with the use of synchronized accelerometry and heart rate before the intervention, during the intervention (2, 9, and 18 wk), and within a 2-wk period of detraining after the intervention.

**Results:** Structured prescribed exercise increased total PAEE and had no detrimental effect on nonprescribed PAEE. Indeed, there was a trend for greater nonprescribed PAEE in the EX group \((P = 0.09)\). Weight loss in the EX group \((-1.8 ± 2.2 \text{ kg} \text{ compared with } +0.2 ± 2.2 \text{ kg in the CON group, } P < 0.02)\) reflected only \(\approx 40\%\) of the \(300–373 \text{ kcal/kg body mass potential energy deficit from prescribed exercise. Serum leptin concentration decreased by 24\% in the EX group (compared with 3\% in the CON group, } P < 0.03)\), and we estimate that this was accompanied by a compensatory increase in energy intake of \(\approx 100 \text{ kcal/d.}\)

**Conclusions:** The adoption of regular structured exercise in previously sedentary, middle-aged, and overweight men does not result in a negative compensatory reduction in nonprescribed physical activity. The less-than-predicted weight loss is likely to reflect a compensatory increase in energy intake in response to a perceived state of relative energy insufficiency. *Am J Clin Nutr* 2010;92:1009–16.

**INTRODUCTION**

Enrollment in some form of structured exercise program is often recommended as a therapeutic intervention in sedentary and overweight populations (1). However, it has been suggested that such prescribed exercise may not always create negative energy balance, and therefore weight loss, due to compensatory alterations in other behavioral aspects of energy expenditure (2–4). For example, it has been proposed that an increase in energy expenditure as a result of structured exercise may be accompanied by a decrease in spontaneous physical activity energy expenditure (PAEE), such as fidgeting (3, 5). In support of this contention, there are several reports that total energy expenditure (TEE) remains unchanged in middle-aged and elderly men and women in response to 8 wk of endurance training (6), 12 wk of aerobic and resistance exercise training (7), and 14 wk of interval training (8). In contrast, other studies show increases in TEE as a result of structured exercise, with no change in measures of spontaneous physical activity in obese boys (9) and young, lean men and women (10). Similarly, in middle-aged overweight and obese men and women, 8 mo of aerobic training was reported to increase energy expenditure without a decrease in nonexercise physical activity (11).

Therefore, it remains unclear whether a structured exercise program leads to a compensatory change in nonexercise energy expenditure (ie, a decrease in nonprescribed physical activity outside of the intervention). This is critically important to understand the potential role of prescribed exercise in manipulating energy balance. Previous research in this area has been problematic due to the limited availability of tools for the accurate assessment of both free-living energy expenditure and prescribed PAEE, which may therefore explain some of the inconsistency in the evidence base. For example, TEE has been estimated with use of accelerometers, doubly labeled water, or the combination of activity diaries and whole-body calorimetry (6–8). With the use of these techniques and deductive reasoning, the absence of an observed increase in TEE with prescribed exercise has led logically to the conclusion that compensation in other aspects of nonprescribed PAEE must have occurred (6–8). However, this is
only one possibility, especially within the context of a rather modest “dose" of prescribed exercise in many intervention studies (12). To illustrate this point, brisk walking at 4 miles/h for 3 times/wk for 30 min expends ≈190 kcal per exercise bout for a 72-kg man and therefore ≈570 kcal/wk (13). For an average man with a total daily energy expenditure of ≈2500 kcal, this amount of structured exercise (if prescribed) would increase TEE by only ≈3%. Thus, given the noise in measurement of TEE even with use of sophisticated techniques such as doubly labeled water (14), it is quite possible that these tools are not sufficiently sensitive to capture relatively small increases in TEE over the course of a typical exercise intervention. In this scenario, the deductive argument that this evidence supports the concept of compensation in nonexercise PAEE would be unsound.

Clearly, inaccuracies in assessment of TEE during an exercise intervention have the potential to generate erroneous conclusions regarding compensatory adjustments in nonprescribed physical activity. One potential solution to this problem is to simultaneously determine PAEE during (ie, prescribed exercise) and outside (ie, nonprescribed physical activity) structured exercise. In the present study, we use a novel technique to investigate the effect of a 6-mo exercise intervention on both prescribed and nonprescribed PAEE in formerly sedentary middle-aged men.

SUBJECTS AND METHODS

Participants

A total of 54 middle-aged men were initially recruited for this investigation and randomly assigned to either an exercise (EX) or a control (CON) group. Recruitment began in July 2007. Nine men dropped out for a variety of reasons, and therefore 41 men completed the study (15). A complete physical activity record was available for 29 men (15 in the EX group and 14 in the CON group; see below for details). Blood samples were available for 33 men (15 in the EX group, 18 in the CON group). Eligibility criteria included age 45–64 y, nonsmoker, free from diagnosed disease, and classed as sedentary according to age-specific physical activity recommendations (16). The nature and possible risks of participating in the study were explained both verbally and in writing to each participant before obtaining their full written and informed consent. This study was approved by the Bath NHS Research Ethics Committee (United Kingdom).

Baseline measures

At baseline, participants reported to the laboratory (pre-intervention) for various measurements (eg, height and body mass). They also completed a submaximal and maximal walking test to establish the relation between oxygen consumption (\( \text{VO}_2 \)) and heart rate, as well as maximal oxygen uptake (\( \text{VO}_2 \) max), to prescribe exercise as previously described (15).

Intervention

Subjects in the EX group completed a 24-wk individually prescribed, progressive, and monitored exercise program, whereas subjects in the CON group were asked to maintain their existing (sedentary) lifestyles. At the end of the 24-wk intervention, subjects in the EX group were asked to return (temporarily) to their previous sedentary lifestyles for a 2-wk period. This exercise intervention has been described in detail elsewhere (15). Briefly, the exercise intervention consisted primarily of walking or running, with some cycling or cross-training. The intervention progressed in terms of frequency, duration, and intensity over a 24-wk period. During weeks 1–2, subjects performed exercise 3 times/wk for 30 min at ≈50% \( \text{VO}_2 \) max, and by week 24 they performed exercise 4 times/wk for 60 min at 70% \( \text{VO}_2 \) max. Approximately 10% of sessions (once every 2 wk) were supervised in the laboratory. The remaining exercise sessions took place in the University of Bath fitness suite, and each nonlaboratory exercise session was recorded (attendance, time of day, and duration) with use of an electronic monitoring system (Fitronics, Bath, United Kingdom). In addition to collecting accurate information regarding each bout of structured prescribed exercise, this system also allowed subject-specific settings to be preprogrammed into exercise equipment before each exercise bout. Adherence to the intervention was maintained with regular telephone calls, e-mails, and written feedback. No dietary constraints were imposed, and participants in both groups were free to consume food and fluid ad libitum.

Physical activity assessment

Estimates of energy expenditure and time spent participating in physical activity above predetermined thresholds were assessed in both groups pre- (baseline) and postintervention (week 24–26 during a 2-wk detraining period) as well as during approximately weeks 2, 9, and 18 of the intervention. PAEE and the time engaged in physical activity above defined intensity thresholds were estimated with use of synchronized accelerometry and heart rate (AHR) over 7 full consecutive days (Actiheart; Cambridge Neurotechnology, Cambridge, United Kingdom). Briefly, AHR uses branched-equation modeling to estimate PAEE from synchronized AHR data, providing precise estimates of energy expenditure in minute-by-minute epochs during a range of physical activities (17–20). Importantly, this is a continuous measurement (ie, day and night). Subjects were blinded to the purpose of the monitor to minimize any potential effect on their behavior. The AHR monitor is worn on the chest, and subjects were told that this device was being used to determine heart rate variability. For a participant to be included in the present investigation, >90% of physical activity data had to be available over the course of the 6-mo intervention (mean ± SD for EX and CON; 96 ± 2% and 94 ± 4%, respectively).

The continuous minute-by-minute physical activity data (kcal·kg\(^{-1}\)·min\(^{-1}\)) were used to determine daily overall PAEE, calculated as the mean of the 7-d record. As described previously (21), we used defined thresholds for intensity and in-house software to determine the amount of time (min) and energy (kcal) engaged in activity above predetermined thresholds and in defined bouts [eg, ≥3 metabolic equivalents (METs), ≥10-min bouts]. At baseline, these data were also used to confirm eligibility and that subjects were failing to meet age-specific recommended minimum levels of physical activity (16).

Energy expended during the exercise intervention (prescribed PAEE) was determined by using the date, time, and duration of each exercise session available from the electronic monitoring system (Fitronics). Nonprescribed PAEE (the energy expenditure...
The exercise intervention significantly increased overall PAEE (Figure 2; interaction: $F = 2.9, P = 0.03$). By week 18 of the intervention, overall PAEE in the EX group was significantly higher than in the CON group. The increase in PAEE was not only due to the prescribed exercise but also to the increased intensity or duration of physical activity during free-living conditions. This finding highlights the importance of considering both prescribed and nonprescribed PAEE when assessing the effects of exercise interventions.
greater than in the CON group \( (P = 0.004) \), with a nonsignificant trend apparent by week 9 \( (P = 0.08) \).

**Prescribed and nonprescribed PAEE**

Prescribed PAEE (ie, energy expenditure during structured exercise) was determined by synchronized AHR with use of information from the electronically recorded exercise sessions. Prescribed PAEE increased significantly over the course of the progressive exercise intervention (Figure 2; time: \( F = 15.7, P < 0.001 \)).

Nonprescribed PAEE was calculated by subtracting prescribed PAEE from overall PAEE. Whereas there was no interaction between treatment and time, the preplanned contrast between groups at week 18 was consistent with the visual trend for greater nonprescribed PAEE in the EX group \( (P = 0.09) \). This difference in nonprescribed PAEE was largely explained by a decrease in the CON group over this 6-mo period. Notably, this difference did not persist into the 2-wk detraining period (Figure 2).

The intervention had no effect on low-intensity nonprescribed PAEE (Table 3).

**Serum leptin and PYY**

As shown in Figure 3, the reduction in the serum leptin concentration in the EX group was \( -1999 \pm 2356 \text{ pg/mL} \), which was significantly greater than the change in CON \( (-177 \pm 1760 \text{ pg/mL}; P = 0.03) \). Structured exercise had no significant effect on the mean \( (\pm \text{SD}) \) serum PYY concentration (EX: baseline, 189 \pm 51 \text{ pg/mL}; week 24, 171 \pm 38 \text{ pg/mL}; week 26, 168 \pm 72 \text{ pg/mL}; CON: baseline, 192 \pm 93 \text{ pg/mL}; week 24, 199 \pm 72 \text{ pg/mL}; week 26, 187 \pm 67 \text{ pg/mL} ) .

**DISCUSSION**

We examined the effect of an individually prescribed and carefully monitored exercise program on free-living, nonprescribed physical activity thermogenesis in sedentary, overweight, middle-aged men.

It has been proposed that structured exercise might have a negative effect on nonprescribed PAEE (3, 5). However, we found no evidence that nonprescribed physical activity (including low-intensity nonprescribed physical activity) was negatively affected by participation in structured exercise. These findings support earlier observations in obese boys (9); lean, young adults (10); and middle-aged men and women (11). In contrast, some studies have reported a decline in physical activity behavior outside prescribed exercise in middle-aged participants (6–8). Unlike previous investigations, one of the strengths of the present study is that we used the same instrument to determine prescribed and nonprescribed PAEE. This notwithstanding, it is possible that

**TABLE 1**

Subject characteristics^1^  

<table>
<thead>
<tr>
<th>Group</th>
<th>Baseline</th>
<th>Week 24^2^</th>
<th>Δ^2^</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>EX 55 ± 5</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>CON 53 ± 4</td>
<td>—</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Height (m)</td>
<td>EX 1.79 ± 0.04</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>CON 1.84 ± 0.07</td>
<td>—</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>EX 89.7 ± 9.6</td>
<td>87.9 ± 8.9^4^</td>
<td>-1.8 ± 2.2^5^</td>
</tr>
<tr>
<td>CON 93.7 ± 11.8</td>
<td>93.9 ± 11.6</td>
<td>+0.2 ± 2.2</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m^2^)</td>
<td>EX 28.1 ± 2.7</td>
<td>27.4 ± 2.4^4^</td>
<td>-0.7 ± 0.8^5^</td>
</tr>
<tr>
<td>CON 27.6 ± 3.0</td>
<td>27.7 ± 3.0</td>
<td>+0.1 ± 0.7</td>
<td></td>
</tr>
</tbody>
</table>

^1^ All values are means ± SDs. Exercise group (EX), \( n = 15 \); control group (CON), \( n = 14 \).

^2^ Data were collected on completion of the intervention.

^3^ Change relative to baseline.

^4^ \( P = 0.01 \) (paired-samples t-tests relative to baseline)

^5^ Denotes a group difference for change relative to baseline (independent-samples t-test, \( P \leq 0.02 \)).

**TABLE 2**

Time engaged in physical activity of \( \geq 3 \) metabolic equivalents accumulated in 10-min bouts^4^  

<table>
<thead>
<tr>
<th>Group</th>
<th>Baseline</th>
<th>Week 2</th>
<th>Week 9</th>
<th>Week 18</th>
<th>Weeks 24–26^3^ (detraining)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EX 37 ± 23</td>
<td>54 ± 33^4^</td>
<td>67 ± 45^4^</td>
<td>68 ± 42^4^</td>
<td>24 ± 24</td>
<td></td>
</tr>
<tr>
<td>CON 28 ± 14</td>
<td>26 ± 20</td>
<td>21 ± 16</td>
<td>24 ± 26</td>
<td>18 ± 13</td>
<td></td>
</tr>
</tbody>
</table>

^1^ All values are means ± SDs. Exercise group (EX), \( n = 15 \); control group (CON), \( n = 14 \).

^2^ Treatment × time interaction: \( F = 4.9, P = 0.006 \).

^3^ Data were collected on completion of the intervention during detraining.

^4^ \( P \leq 0.04 \) (independent-samples t tests); EX group compared with CON group at equivalent time point.
subtle differences in exercise prescription and/or the population under investigation may contribute to the discrepancies in the literature.

In contrast to a potential decrease in nonprescribed PAEE, there was some evidence that the reverse may be true (ie, there was a trend for nonprescribed PAEE to be maintained over time in the EX group relative to a temporal decrease in the CON group, \( P = 0.09 \)). The decrease in the CON group may reflect a seasonal effect, because 10 of these 14 participants started the intervention during the summer months (July to September), and there is well-documented seasonal effect for physical activity (25). This highlights the value of including a control group. Importantly, in support of an independent effect of the exercise intervention on nonprescribed PAEE, once structured exercise was removed (ie, during detraining), there was no longer any difference in nonprescribed PAEE between the EX and CON groups. Interestingly, a recent investigation reported a trend for an increase in nonprescribed physical activity in a small group of 8 men who performed the highest intensity and amount of exercise during an 8-mo structured exercise program (11). The reasons for relatively greater nonprescribed PAEE are unclear. This could reflect factors such as improved psychological state and mood or indeed pursuit of a pleasure-based reward with continued physical activity behavior (26, 27). This idea is more appealing following the discovery that the dopamine D2 receptor gene is associated with physical activity level (28). Alternatively, a small part of this enhanced nonexercise PAEE could be explained by the energy cost of necessary lifestyle changes associated with the exercise intervention (eg, preparing for and commuting to or from the fitness center).

In the present study, we derived nonprescribed PAEE by subtracting directly assessed energy expended during structured exercise from overall PAEE. Therefore, the time period available for nonprescribed PAEE in the EX group is reduced by the duration of the exercise prescription during a given week (eg, \( \approx 3.5 \) h in week 18). Whereas this represents only a small proportion of either total time (2%) or estimated waking time (3%; assuming a 16-h waking day), there is nonetheless a modest reduction in the time available in which to accumulate nonprescribed PAEE in the EX group. Had we sought to compensate for this “lost” time, based on their overall PAEE at baseline, daily nonprescribed PAEE in the EX group would increase to \( 12.4 \pm 3.3 \) kcal/kg at 18 wk, and this would be significantly different from the CON group (\( P = 0.03 \)). This observation has various implications, but the manner in which we present these data is perhaps of greatest practical relevance given that a structured exercise intervention will inevitably reduce the potential time that is available for nonprescribed PAEE. Therefore, our prescribed structured exercise intervention increased overall PAEE (the sum of prescribed plus nonprescribed PAEE), and this tended to have a positive (rather than negative) effect on nonprescribed PAEE, even if we do not correct for the modest loss of time that is available for such physical activity.

According to the above suggestion, if structured exercise increases exercise energy expenditure (ie, the energy cost of the prescription; prescribed PAEE) and also increases or maintains nonprescribed PAEE, then exercise interventions should be very effective for weight loss. Unfortunately, in practice, this is not

![FIGURE 2. Mean (95% CI) physical activity energy expenditure (PAEE) before (baseline), during (weeks 2–18), and after (weeks 24–26; detraining) the intervention in control (CON; open bars) and exercise (EX; closed bars) groups. Energy expended during the prescribed exercise intervention is shown for the EX group (prescribed PAEE; hatched bars). EX group, \( n = 15 \); CON group, \( n = 14 \). There was a significant time \( \times \) group interaction effect for overall PAEE (\( F = 2.9, P = 0.03 \); 2-factor repeated-measures ANOVA). *Denotes that total PAEE in the EX group (prescribed and nonprescribed PAEE) is greater than in the CON group (\( P = 0.004 \), independent-samples \( t \) test).

### TABLE 3
Nonprescribed physical activity energy expenditure of low intensity [\(<3 \) metabolic equivalents (METs)] and moderate to vigorous intensity (\( \geq 3 \) METs)\(^1\)

<table>
<thead>
<tr>
<th>Energy expenditure of physical activity(^2)</th>
<th>Weeks 24–26(^3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>Baseline</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td><strong>Low intensity ((&lt;3 ) METs)</strong></td>
<td></td>
</tr>
<tr>
<td>EX</td>
<td>6.39 ± 1.26</td>
</tr>
<tr>
<td>CON</td>
<td>6.76 ± 2.02</td>
</tr>
<tr>
<td><strong>Moderate–vigorous intensity ((\geq 3 ) METs)</strong></td>
<td></td>
</tr>
<tr>
<td>EX</td>
<td>5.07 ± 2.29</td>
</tr>
<tr>
<td>CON</td>
<td>4.82 ± 1.71</td>
</tr>
</tbody>
</table>

\(^1\) All values are means ± SDs. Exercise group (EX), \( n = 15 \); control group (CON), \( n = 14 \).

\(^2\) There were no significant differences in these components of energy expenditure between groups or over time.

\(^3\) Data were collected on completion of the intervention during detraining.
FIGURE 3. Mean (95% CI) serum leptin concentrations before (baseline), after (week 24), and 2 wk after (week 26; detraining) the intervention in the control (CON; open bars) and exercise (EX; closed bars) groups. EX group, *n* = 15; CON group, *n* = 18. #Denotes a difference in the change scores from baseline between groups (*P* = 0.03, independent-samples *t* test).

Generally the case (29). According to our calculations, the total energy expended through exercise in the present intervention was between 300 ± 106 and 373 ± 50 kcal/kg body mass (see supplemental information under “Supplemental data” in the online issue for more details). If energy intake and resting metabolic rate (RMR) remained the same, then according to the laws of thermodynamics, this would translate into ∼3.8 to ∼4.7 kg weight loss if we assume that 1 g adipose tissue is equivalent to 7.1 kcal (30). In fact, the EX group lost only 1.8 ± 2.2 kg body mass. Whereas this degree of weight loss is meaningful and potentially important if it were to continue, these middle-aged men realized only 38–47% of the theoretical weight loss and deficit from the exercise intervention. This was not explained by a reduction in nonprescribed PAEE, because the trend was for the opposite (i.e., higher nonprescribed PAEE with structured exercise). We propose that the most likely explanation for the lack of predicted weight loss is that the increase in exercise energy expenditure (and associated energy deficit) is partially compensated for by an increase in appetite and energy intake (2–4). Based on the difference between actual weight loss and predicted weight loss from the energy expenditure of prescribed exercise, we estimate that such a compensatory increase in energy intake would be in the order of 84–123 kcal/d over the 24-wk intervention (depending on which estimate of prescribed exercise energy expenditure is used). The detection of such a small effect (an increase of 3–4%) would be challenging with the tools currently available for the assessment of energy intake.

It has become clear over the past decade that leptin works primarily as an energy insufficiency signal (31, 32). Serum leptin concentration decreases with even very modest weight loss (33), and it has been proposed that this is an attempt to protect fat stores (34). In the present study, leptin concentration decreased 24% in response to exercise training. A decrease in leptin reduces both the repression of orexigenic pathways and the stimulation of anorexigenic pathways (35). Note that a decrease in leptin is associated with increased sensations of hunger (36), an increase in reward-related behaviors (31, 37), and altered neural activity in brain regions affecting regulatory and hedonic aspects of energy homeostasis (38). A decrease in leptin decreases sensitivity to other regulators of appetite such as cholecystokinin (39). There have been numerous reports of a decrease in leptin with exercise training (40–43) but can be prevented if additional food is provided to avoid an energy deficit (44). Collectively, the decrease in leptin confirms that in response to our exercise training intervention there was a challenge on energy stores that was sufficient to provoke a physiologic response within adipose tissue. Given the importance of leptin in this context, it is possible that the decrease in leptin could be a key biological signal involved in a compensatory increase in energy intake with exercise training. Notably, the replacement of leptin during weight loss (leptin administration) reversed the changes seen in neural activity responses to food stimuli (38). Leptin works in concert with other hormones such as PYY to inform feeding behavior (45), and, although we found no change in fasting PYY, it is feasible that the postprandial response of PYY and other mediators could have been more revealing.

One of the strengths of the present study is the relevant population of overweight, sedentary, middle-aged men who might typically be advised to take up exercise. From week 2 of the intervention, participants in the EX group spent more time engaged in the type of physical activity that is recommended for general health (16, 46). The 6-mo exercise intervention was carefully monitored and achieved excellent compliance (94%). Our repeated 7-d estimates of 24-h minute-by-minute energy expenditure coupled with the electronic monitoring of prescribed exercise allowed us to use the same instrument to determine the energy expenditure of both prescribed and nonprescribed physical activity. The technique has been validated against indirect calorimetry in a number of settings and has a small bias similar to other estimates of energy expenditure, such as doubly labeled water (18, 20). This allows us to deduce that energy intake increased on the assumption that RMR does not decrease. If there had been an increase in RMR with training, then our calculations would underestimate the increase in energy intake. However, an assumed lack of change in RMR with this kind of exercise intervention seems reasonable based on previous reports (47). It is also important to highlight that our calculated net energy deficit assumes that all weight loss is from adipose tissue. An increase in muscle mass could confound these estimates, although muscle hypertrophy is very unlikely with this type of exercise intervention, and fat loss is broadly equivalent to weight loss (48–55). Whereas our secondary calculations are well grounded, of course these estimates are inferred and less robust than our direct observations of prescribed and nonprescribed PAEE.

The most important finding of the present study is that prescribed exercise did not reduce nonprescribed PAEE in sedentary, overweight, middle-aged men. Indeed, in contrast, there was a trend for nonprescribed PAEE to be maintained and higher than in a CON group who continued with their normal (sedentary) behavior. We report a decrease in serum leptin with exercise training and this perhaps indicates adipocyte sensing of an energy insufficiency. Based on our calculations, the current exercise intervention triggered an increase in energy intake of ∼100 kcal/d without any behavioral compensation in PAEE. This supports subjective observations such as an increased drive to eat with exercise training (56) and increased fasting hunger (55). This makes a compelling case that an exercise-induced challenge on energy homeostasis increases energy intake so that a smaller...
proportion of the potential energy deficit from participation in structured exercise is realized.

The author’s responsibilities were as follows—JET: contributed to developing the experimental design, acquisition, analysis, and interpretation of the data collected and to drafting the manuscript; DM: was involved in the experimental design and the acquisition and analysis of data collected; JAB: contributed to the analysis and interpretation of data collected and drafting the manuscript; and DT: was responsible for the experimental design, analysis, and interpretation of results and drafting the manuscript. The use of commercial names in this manuscript is for information purposes only and does not imply endorsement. None of the authors had a conflict of interest.

REFERENCES


