Resistence to Exercise-Induced Weight Loss: Compensatory Behavioral Adaptations

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1Division of Endocrinology, Metabolism, and Diabetes and Division of Geriatric Medicine, School of Medicine, Anschutz Medical Campus, University of Colorado, Denver, CO; 2Cancer Prevention Fellowship Program, National Cancer Institute, Bethesda, MD; 3Center for Physical Activity and Weight Management, University of Kansas, Lawrence, KS; 4Department of Kinesiology, School of Public Health and Health Sciences, University of Massachusetts, Amherst, MA; and 5Institute of Health and Biomedical Innovation, School of Exercise and Nutrition Sciences, Queensland University of Technology, Brisbane, AUSTRALIA

ABSTRACT

MELANSON, E. L., S. K. KEADLE, J. E. DONNELLY, B. BRAUN, and N. A. KING. Resistance to Exercise-Induced Weight Loss: Compensatory Behavioral Adaptations. Med. Sci. Sports Exerc., Vol. 45, No. 8, pp. 1600–1609, 2013. In many interventions that are based on an exercise program intended to induce weight loss, the mean weight loss observed is modest and sometimes far less than what the individual expected. The individual responses are also widely variable, with some individuals losing a substantial amount of weight, others maintaining weight, and a few actually gaining weight. The media have focused on the subpopulation that loses little weight, contributing to a public perception that exercise has limited utility to cause weight loss. The purpose of the symposium was to present recent, novel data that help explain how compensatory behaviors contribute to a wide discrepancy in exercise-induced weight loss. The presentations provide evidence that some individuals adopt compensatory behaviors, that is, increased energy intake and/or reduced activity, that offset the exercise energy expenditure and limit weight loss. The challenge for both scientists and clinicians is to develop effective tools to identify which individuals are susceptible to such behaviors and to develop strategies to minimize their effect. Key Words: ENERGY EXPENDITURE, PHYSICAL ACTIVITY, NONEXERCISE ACTIVITY THERMOGENESIS, ENERGY INTAKE

The recent focus on exercise as medicine is predicated on a fundamental dose–response relationship; the application of exercise will confer benefits to health. On average, when previously sedentary individuals add exercise to their lifestyle, they become more physically fit, are at lower risk for many chronic diseases, and are better able to manage an appropriate body weight. This forms the basis for physical activity guidelines to enhance the health of the public. The average response obscures considerable variability, and the individual responses to habitual exercise deviate widely (Fig. 1). In addition to the fortunate subgroup who loses body weight/fat, there are the unfortunate people who do not, despite completing a similar volume of exercise. It is clear that, not only is there a large interindividual variability in weight change in response to exercise or diet is nonexercise activity or more EI in blunting the expected energy deficit generated by adding structured exercise. However, the observation that observed weight loss is modest or less than theoretically expected persists in studies where exercise is closely supervised and continuously adjusted over the course of the intervention (13). Therefore, people who lose little or no weight in response to adding structured exercise must be compensating for the increased EE of exercise by reducing their nonexercise physical activity (non-Ex PA) and/or increasing their EI (Fig. 2). Compensatory response will either attenuate or even reverse the energy deficit generated by adding structured exercise. However, data that support or refute the relative importance of less nonexercise activity or more EI in blunting the expected weight loss is modest or less than theoretically expected.

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benefits of exercise on energy balance and body weight are equivocal. Contributing factors to this ambiguity include not monitoring of the exercise intervention as well as accuracy and validity issues related to the measurement of EI and EE. The methodological issues are compounded by the fact that it is difficult to clamp EE during an exercise intervention because it is a behavioral adaptive response (i.e., non-Ex PA), which is also a dependent variable of interest.

The purpose of this review was to summarize the presentations of participants in a symposium entitled "Behavioral Compensation to Exercise: Do We Eat More and Do Less?" presented at the National American College of Sports Medicine Meeting in San Francisco, California, in 2012. The purpose of the symposium was to present recent, novel data related to the effects of exercise, specifically prescribed for weight loss, on compensatory eating and physical activity behaviors. The strategy was to feature speakers who could present data from each side of the energy balance equation. Dr. Melanson and Dr. Kozey Keadle discussed the effect of exercise on non-Ex PA and EE, and Dr. King and Dr. Donnelly discussed the effect of exercise on energy and macronutrient intake. The objectives of this review were to present the state of knowledge regarding the magnitude and direction of activity and diet compensation, to identify areas where more research is needed, and to provide guidance to improve exercise/diet recommendations, which in turn will improve the effectiveness of weight loss strategies.

EFFECTS OF EXERCISE ON NON-EX PA

Total daily EE (TDEE) is the sum of resting metabolic rate, thermic effect of food, and physical activity EE (AEE) (Fig. 2). AEE can be further divided into exercise (i.e., planned, structured physical activity [EXEE]) or nonexercise physical thermogenesis (NEAT). In human studies, if the outcome measure is physical activity assessed via accelerometry, then the appropriate term to describe this is non-Ex PA. However, if the outcome is nonexercise EE measured using doubly labeled water (DLW) or estimated from accelerometry or other methods (see next paragraph), then the appropriate term to use is NEAT. We have used this terminology to properly distinguish these two outcomes throughout the manuscript. In some studies, particularly animal studies, the term spontaneous physical activity (SPA) is often used to indicate NEAT. Thus, in some instances, we have used the same terminology that was used by the authors. Garland et al. (23) recently addressed some challenges defining and measuring NEAT. For this review, we are interested in determining whether individuals "do less" when they start exercise training. Therefore, we will broadly define non-Ex PA as including all activities of daily living, which include fidgeting, maintaining posture, and ambulation, and NEAT is the term that defines the EE associated with these activities (23,37). In humans, NEAT is highly variable (37,56,75), a strong positive predictor of TDEE (56,72), and likely influenced by environmental (18) and biological factors (29,75). Studies in rodents suggest that multiple neuroregulators (e.g., dopamine, orexin A, leptin, ghrelin, agouti-related protein) play a role in regulating SPA/NEAT (23,35).
Determining whether behavioral compensation occurs in response to exercise training requires accurate measurement of all components of TDEE. However, obtaining accurate measures of TDEE in free-living EE in humans is challenging. The two most common approaches are the DLW method (24,49,69) or the use activity monitors (12,27,41). A complete review of the various methods to measure physical activity have been published elsewhere (69). DLW is the gold standard method for measuring TDEE (69). When DLW is used in exercise training trials, NEAT is typically estimated using the measured or estimated resting metabolic rate and EXEE. Few studies actually measure the thermic effect of food; most assume it is 10% of TDEE and that it does not change during the intervention period. NEAT is thus estimated as the difference between the TDEE and the sum of RMR and exercise EE (e.g., TDEE of 2500 kcal - (RMR of 1600 kcal + EXEE of 400 kcal) = 500 kcal NEAT). The major limitation of this method is the cost, so sample sizes are typically small or contain only a subset of participants. In addition, DLW provides one value of EE over a period of days; thus, unless total EXEE is accounted for, it is difficult to detect changes in NEAT. DLW water does not provide information on the type of non-Ex PA that is taking place (e.g., sitting and ambulatory movement) or the patterns of physical activity. This is an important issue because sedentary behavior has been shown to be predictive of health outcomes independent of total physical activity (47). These limitations can somewhat be overcome using activity monitors, but estimates of EE from accelerometry are less accurate than those from DLW (69).

Does NEAT contribute to body weight regulation? The strongest evidence that NEAT plays a role in regulating body weight comes from studies in animals. For example, Teske et al. (65) demonstrated that obesity-resistant rats had higher levels of SPA throughout their lifespan. Obviously, performing such a detailed longitudinal study in humans would not be feasible, and consequently, the evidence from human studies is not as convincing. The best evidence in humans comes from prospective studies performed in Pima Indians, which demonstrated that SPA measured in a respiration chamber was inversely correlated with fat mass change in men (75). Interestingly, there was no association between SPA and fat mass change in women. This study also demonstrated that family membership accounted for 57% of the variation in SPA, suggesting that NEAT may be genetically programmed. Additional evidence that NEAT contributes to body weight regulations comes from two long-term overfeeding studies. The seminal studies of Bouchard et al. (5) demonstrated that gains in fat mass were primarily determined by genetic factors (~50%), but given that physical activity levels were tightly controlled in this study, it is possible that differences in NEAT contributed to the unexplained variance in weight gain. The second study, by Levine et al. (38), demonstrated that changes in NEAT were the only component of EE that predicted fat gain. However, this was a small study (N = 16), and as reviewed by Westerterp (70), this is the only study in humans that demonstrated an increase in physical activity in response to overfeeding (38). In addition, how NEAT may regulate body weight during experimental overfeeding may be different from what is experienced in free-living individuals. For example, data from a recent large (N = 321) prospective study of free-living women demonstrated that AEE, determined using DLW, was not a predictor of weight change for the 3-yr of follow-up (40). Even at modest levels of AEE (i.e., <4 MJ d⁻¹), some women lost weight, some remained weight stable, and others actually gained weight. Consistent with this latter study, two recent cross-sectional studies of approximately 120 individuals demonstrated that “incidental PA,” determined using accelerometry, is positively associated with cardiorespiratory fitness but shows no association with abdominal fat mass (46,47). Thus, definitive studies demonstrating a clear link between NEAT and body weight regulation in humans are still lacking.

Does initiation and adoption of exercise cause changes in NEAT? Several studies have been performed to determine whether NEAT changes during the initiation and adoption of regular exercise (Table 1). The acute effect of exercise (i.e., initiation) on non-Ex PA and NEAT has been studied in several studies. In a series of elegant studies, Stubbs et al. (61–63) studied the acute effects of different doses of exercise on EI, EE (measured using HR monitor), and energy balance for periods of 7–10 d. In two of these studies, TDEE tended to decrease over time when medium (1.6 MJ d⁻¹) and high (3.2–4.0 MJ d⁻¹) levels of exercise were performed (61,62), suggesting that NEAT decreased. However, in the latter study, dietary fat intake was also increased, so it is possible that the compensation was partly due to the dietary manipulation. More recently, Alahamdi et al. (1) compared the effects of single session of exercise performed at two different intensities on non-Ex PA in overweight and obese men. Non-Ex PA remained unchanged for the first 2 d but increased 3 d after the moderate-intensity (16%, not significant) and high-intensity (25%) sessions. The reasons for this delayed increase are not clear, but similar results were previously observed in obese boys (36). In contrast, NEAT (estimated from HR and physical activity diaries) remained unchanged in a group of lean men and women participating in every other day moderate-intensity exercise training for 8 d (48). Although a strength of these short-term studies is the within-subject design, they are limited by the short-term nature of the intervention. Longer-term studies aimed at understanding the effects of exercise adaptation on NEAT have also yielded equivocal results. Studies have shown reductions in non-Ex PA (12,41,49) and NEAT (13,24,49,51), whereas other studies have reported no changes in NEAT during the training period (27,71). It is difficult to reconcile these discrepant findings, but it is likely that differences in intensity and mode of exercise, the measurement tool used, and the age and sex of study participants were contributing factors. For example, it appears that older adults are more likely to exhibit compensatory changes
TABLE 1. Summary of experimental studies that directly measured TDEE in responses to exercise.

<table>
<thead>
<tr>
<th>Reference</th>
<th>N</th>
<th>Subjects</th>
<th>Duration</th>
<th>Days per week</th>
<th>Intensity/Duration</th>
<th>Measurement</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Goran and Poehler (24)</td>
<td>11</td>
<td>NW/OW older men (56–78 yr)</td>
<td>8 wk</td>
<td>3</td>
<td>85% VOCmax, 300 kcal per session</td>
<td>DLW</td>
<td>Evidence for compensation, NEAT decreased by 62% (from 571 ± 386 to 340 ± 324 kcal d⁻¹)</td>
</tr>
<tr>
<td>Meijer et al. (49)</td>
<td>22</td>
<td>NW/OW older men and women (mean, 58 yr)</td>
<td>12 wk</td>
<td>2</td>
<td>60 and 90 min, weight training and aerobic</td>
<td>AM</td>
<td>Nonexercise activity significantly lower on training compared with nontraining days</td>
</tr>
<tr>
<td>Donnelly et al. (13)</td>
<td>74</td>
<td>OW/OB men and women (17–35 yr)</td>
<td>16 months</td>
<td>5</td>
<td>45 min, 75% HRR</td>
<td>DLW</td>
<td>Weight loss ~50% of predicted, suggests compensation</td>
</tr>
<tr>
<td>Mantou et al. (41)</td>
<td>34</td>
<td>OW/OB men and women (mean, 31 yr)</td>
<td>8 wk</td>
<td>2 or 5</td>
<td>150 min wk⁻¹, 90%–95% LT</td>
<td>HR</td>
<td>Predicted fat loss, 0.8 ± 0.2 kg; actual fat loss, 0.0 ± 0.2 kg; half of subjects compensated</td>
</tr>
<tr>
<td>Di Blasio et al. (12)</td>
<td>34</td>
<td>NW-OB postmenopausal women (mean, 56 yr)</td>
<td>13 wk</td>
<td>4</td>
<td>40 min, moderate walking</td>
<td>AM</td>
<td>Half of sample compensated, on average no change in TDEE</td>
</tr>
<tr>
<td>Hollowell et al. (27)</td>
<td>50</td>
<td>OW/OB men and women (mean, 53.2 yr)</td>
<td>8 months</td>
<td>3–5 times</td>
<td>Low 5055 kJ wk⁻¹ at 65%–80% or 40%–55% VOCmax, high 8372 kJ wk⁻¹ at 65%–80% VOCmax</td>
<td>AM</td>
<td>Both groups significantly increased TDEE, no change in nonexercise activity</td>
</tr>
<tr>
<td>Morio et al. (51)</td>
<td>13</td>
<td>NW/OW men and women (63 ± 2 yr)</td>
<td>14 wk</td>
<td>3</td>
<td>40 min total, 20 min moderate, 20 min interval training</td>
<td>Self-report diary</td>
<td>No change in TDEE, time walking decreased, energy expended during free-living activities significantly decreased by 7.7%</td>
</tr>
<tr>
<td>Alahmadi et al. (1)</td>
<td>16</td>
<td>OW/OB men (mean, 26.5 yr)</td>
<td>4 d</td>
<td>1</td>
<td>60 min; moderate</td>
<td>AM</td>
<td>No evidence for compensation, increase in TDEE</td>
</tr>
<tr>
<td>McLaughlin et al. (47)</td>
<td>16</td>
<td>NW men and women (20–25 yr)</td>
<td>8 d</td>
<td>4</td>
<td>2092 kJ; 59% VOCmax; 48% VOCmax in men</td>
<td>HR</td>
<td>Evidence for compensation, increase in TDEE and nonexercise activity</td>
</tr>
<tr>
<td>Stubbs et al. (60)</td>
<td>8</td>
<td>NW men (18–40 yr)</td>
<td>7 d</td>
<td>7</td>
<td>three 40-min exercise sessions per day at 65% VOCmax</td>
<td>HR</td>
<td>Evidence for compensation, 0.32 MJ d⁻¹ (or 2.2 MJ wk⁻¹)</td>
</tr>
<tr>
<td>Stubbs et al. (62)</td>
<td>6</td>
<td>NW women (18–40 yr)</td>
<td>7 d</td>
<td>6</td>
<td>Moderate: two 40-min exercise sessions per day; high: three 40-min exercise sessions per day</td>
<td>HR</td>
<td>Evidence for compensation, increase in TDEE and nonexercise activity</td>
</tr>
<tr>
<td>Stubbs et al. (61)</td>
<td>6</td>
<td>NM men (18–40 yr)</td>
<td>7 d</td>
<td>6</td>
<td>Moderate: two 40-min exercise sessions per day; high: three 40-min exercise sessions per day</td>
<td>HR</td>
<td>Evidence of modest compensation 0.3–0.6 MJ d⁻¹</td>
</tr>
<tr>
<td>Westerterp (70)</td>
<td>32</td>
<td>NW men and women, 28–41 yr</td>
<td>10 months</td>
<td>4</td>
<td>Half-marathon training, vigorous intensity, progressive increase to 90 min d⁻¹</td>
<td>DLW</td>
<td>No evidence for compensation, increase in TDEE</td>
</tr>
</tbody>
</table>

Values are presented as mean ± SD unless otherwise indicated.
NW, normal weight; OW, overweight; OB, obese; DLW, doubly labeled water; HR, heart rate; HRR, heart rate reserve; AM, activity monitor; NEAT, nonexercise activity thermogenesis; TDEE, total daily EE.

In NEAT (24,51) and non-Ex PA (50), although none of these studies compared younger with older adults. Moreover, because a variety of exercise intensities were used, it is not possible to determine the independent effects of age and exercise intensity. A recent study in overweight adults suggested that reductions in NEAT were dose dependent, with reductions occurring only in the group performing a high dose of exercise (600 kcal d⁻¹) but not in the group performing a moderate dose of exercise (300 kcal d⁻¹) (57). In contrast, no dose–response effect was observed in a secondary analysis in two large cohort studies that compared the effects of different doses of exercise on cardiovascular fitness and cardiovascular risk factors (10,27). However, these studies were not specifically designed to determine whether there is a dose–response effect of exercise on NEAT.

Most early studies in this area have based their conclusions on mean data. As discussed earlier, there is large individual variability in the magnitude and even in the direction of weight lost as a result of exercise training. Recent evidence suggests there are individual differences in compensatory responses that may have important implications for weight loss and other important disease risk factors. Manthou et al. (42) measured NEAT using HR and diaries in overweight and obese women who completed an 8-wk exercise intervention. On average, the group increased TDEE by 0.62 MJ d⁻¹. However, there were large individual differences in weight loss. They classified 11 individuals as “responders” (those who lost as much weight as predicted) and 23 individuals as “nonresponders” (those who lost less weight than predicted). NEAT was the only variable that was significantly different between groups. Previous studies have reported individual variability in weight loss, but this was the first to demonstrate that changes in NEAT are associated with changes in body weight. Furthermore, change in NEAT was a significant predictor in fat mass in the group as a whole. Similarly, Di Blasio et al. (12) reported that half of the postmenopausal women who started training were compensators, decreasing NEAT by an average 233 kcal d⁻¹. In this study, those who decreased NEAT did not have improvements in blood lipids, suggesting behavioral compensation may have implications for changes in health outcomes as well as weight loss. These two recent studies illustrate an important shift from a group-based approach to an individual-level analysis. By considering individual difference in compensation, intervention strategies to reduce compensation and maximize weight loss strategies can be developed. Nonetheless, the results of these studies both demonstrate that on a group level, the trend was for non-Ex PA to decrease.
Do changes in NEAT offset exercise EE during weight loss? There is evidence that caloric restriction, without exercise, induces compensatory changes in non-EX PA and NEAT that can offset intended weight loss. The best evidence comes from a study in monkeys (64). In that study, when EI was decreased by 30% in the first month, the decrease in SPA was substantial enough that no significant decrease in body weight occurred. In the second month, when EI was decreased by 60%, significant weight loss occurred (−6.4% ± 1.7%), but further suppression of SPA was also observed. A similar effect has been observed in humans in the Comprehensive Assessment of Long-term Effects of Reducing Intake of Energy (CALERIE) study (43); NEAT decreased in the caloric restriction groups. However, because there were no changes in physical activity measured with accelerometry, the authors concluded that the decreases in AEE were due to increased muscle efficiency or decreased “fidgeting.”

The evidence that exercise, without caloric restriction, induces compensatory changes in NEAT is not as strong. Evidence can only be obtained from studies where either EI was controlled or measured intake did not change. The strongest evidence can be found in studies conducted by Donnelly et al. (13,16). In the first Midwest Exercise Trial (MET-1), the intervention produced weight loss in men (−5.2 ± 4.7 kg), but not women (13). There were no changes in measured EI during the intervention. At 16 months, EXEE was 668 ± 116 kcal·d⁻¹ in men and 438.9 ± 88 kcal·d⁻¹ in women, but TDEE (DLW) increased only by 371 ± 646 and 209 ± 555 kcal·d⁻¹ in men and women, respectively. TDEE increased by approximately 55% of EXEE in men and 48% of EXEE in women. In the second Midwest Exercise Trial (MET-2) conducted by the same group, EXEE was matched between men and women, and there were no differences in the change in TDEE (16). Thus, it does not appear that there were important sex differences in the degree of compensation. Additional evidence of the effects of exercise without caloric restriction on NEAT comes from the study of Tremblay et al. (67). Subjects were resident for a 12-wk exercise intervention, and EI was held constant at baseline levels. Change in body energy stores during the second half of the intervention was only 65% of the energy deficit. However, it is not possible to conclude that decreases in NEAT occurred not only because TDEE was not measured but also because a reduction in RMR was also observed. Conversely, an analysis of the carefully controlled studies performed by Janssen et al. (28) and Ross et al. (59) demonstrated that the estimated energy imbalance induced by exercise was not significantly different from the prescribed EXEE, suggesting that NEAT was preserved (66). However, there are several limitations to this approach. First, it cannot be determined to what extent changes in both EE and EI contributed to the estimated energy imbalance. Second, errors in body composition are incorporated in this calculation, and the changes in NEAT may be within the bounds of error and thus beyond detection, but they are still of great enough magnitude to have a meaningful effect on body weight (26). These examples illustrate the pitfalls of attempting to assess the degree of exercise compensation without accurate measurement of the individual components of EE.

When the magnitude of observed weight loss is less than the expected weight loss, this is often interpreted as evidence of compensation in non-EX PA and NEAT (13). It is worth noting that in every study we reviewed, the expected weight loss was calculated using the “3500-kcal·lb⁻¹ body weight rule,” that is, an energy deficit of 3500 kcal will induce a weight loss of 1 lb of body weight, which is based on the calculated energy content of body composition (70:30 fat mass:fat free mass [FM:FFM]) (74). However, a limitation to this static approach is that it erroneously predicts a linear change in body weight over time. Moreover, this model was derived from data obtained in short-term, low-calorie diets and thus is not directly applicable to changes in energy stores induced by exercise. As elegantly illustrated by Hall et al. (25), the rate of weight change over time will slow over time because of compensatory changes in EE, and it assumes that all people lose weight at a fixed ratio of 70:30 FM:FFM. Thus, the expected weight loss based on the 3500-kcal rule is likely an overestimate of the true theoretical weight loss.

From the previous discussion, it is clear that more sophisticated studies are required to more completely understand the effects of exercise, prescribed for weight loss, on non-EX PA. Such studies should encompass the simultaneous measurement of EI and EE, including all components of EE, objective measurement of physical activity, and accurate measurement of changes in body energy stores. There are several outstanding questions related to how exercise modifies non-EX PA. To our knowledge, studies examining the effects of type (endurance vs resistance), mode (swimming, cycling, or running), or intensity of exercise on non-EX PA and NEAT (or EI) have not been performed. As discussed earlier, the effects of age, sex, and obesity have not been well studied. Finally, given the equivocal findings from both acute and chronic studies, it is not possible to draw conclusions about differences in compensation that may occur during the initiation and adaption to exercise. Thus, more studies are needed to determine the effects of these potentially important factors on non-EX PA and NEAT.

EFFECTS OF EXERCISE ON ENERGY AND MACRONUTRIENT INTAKE

Compensatory increases in EI are thought to be at least partially responsible for the small magnitude of mean weight loss induced by aerobic exercise training without energy restriction (66). For example, King et al. (31,32) have demonstrated significant increases in EI among participants who did not reduce weight or fat mass in response to aerobic exercise training (12 wk, 5 d·wk⁻¹, 500 kcal per session, 70% max HR). However, evidence for an effect of exercise on EI or macronutrient composition is not compelling. Acute exercise has been shown to have no effect (33,34,39,45) or result in
only partial compensatory increases in EI after a bout of acute exercise (54,61,73). Most studies in this area have shown no change in EI or macronutrient intake in response to aerobic exercise training (2,8,11,13,14,16,44,55,60). However, the literature on change in EI and macronutrient intake in response to exercise training should be interpreted cautiously. EI typically has been assessed by self-reported 3- or 7-d food records, which have been demonstrated to underestimate EI when compared with EE assessed by DLW (2). For example, Donnelly et al. (16) compared EI measured by DLW and diet records and found underreporting for diet records of 20%–30%. Advanced technology can reduce this error considerably. For example, in MET-2 (described in the following paragraph), EI and macronutrient content was assessed across 10 months during 1-wk periods at four time points with ad libitum eating at the University of Kansas cafeteria (16). Digital photographs were obtained before and after consumption, and the type and the quantity of foods and beverages were quantified by trained research staff. Food and beverages consumed outside the cafeteria were assessed using multiple pass 24-h recall procedures using food models and standardized, neutral probing questions. These procedures reduced error of EI compared with DLW to approximately 3%.

Data from the MET-2 afforded a unique opportunity not only to examine the effect of exercise training but also to examine if a dose effect existed at two levels of EE and if there were gender differences for energy and macronutrient intake in a sample of previously sedentary, overweight/obese young adults. Moreover, data for individual variation for weight loss and energy intake were available. A detailed description of the design and methods for MET-2 has been previously published (16). Briefly, MET-2 randomized 141 young adults age 18–30 yr, with body mass index of 25–40 kg·m⁻² to a 10-month, 5-d week, 4 beats·min⁻¹ supervised exercise intervention at two levels of EXEE (400 or 600 kcal per session) or a nonexercise control group. All participants continued their typical patterns of daily physical activity and dietary intake for the duration of the 10-month intervention.

Exercise consisted primarily of walking/jogging on motor-driven treadmills and was supervised by trained research staff in a dedicated exercise facility. The exercise protocol gradually increased EXEE from baseline to the end of month 3 and then remained at 400 or 600 kcal per session for the remainder of the study, as previously described (16). Compliance to the exercise protocol, an essential element of an efficacy study, was defined as successfully completing >90% of scheduled exercise sessions. Successful completion was considered as maintaining the target exercise HR ±4 beats·min⁻¹ for the prescribed duration of the exercise session. Participants who were noncompliant during any 3-month interval (months 0–3, 3–6, and 6–9) or during the final month (month 10) were dismissed from the study. Participants assigned to the nonexercise control group were instructed to continue their typical patterns for physical activity and dietary intake for the duration of the 10-month study. With the exception of assessment of EXEE, the same outcome assessments were completed with both the exercise and control groups.

Of the 141 participants randomized at baseline, 92 (65.2%) complied with the study protocol and completed all outcome assessments. There were no significant between group differences in EI (kcal·d⁻¹) at baseline. During the exercise intervention, EI was 121 kcal·d⁻¹ (4.5%) and 285 kcal·d⁻¹ (10.7%) higher in the 400- and 600-kcal per session compared with control; however, these differences were not significant. Across the duration of the intervention, there were no significant changes for macronutrients for the 400- or 600-kcal per session groups. The control group had significant decreases in percentage and grams of fat. When EI and macronutrients were analyzed by sex, no significant differences were found for men between groups and no significant changes were found across the duration of the intervention. Significant differences were found in women for EI during the intervention. Women in the control group decreased EI by an average of 352 kcal·d⁻¹ compared with women in the 600-kcal per session group who increased by an average of 45 kcal·d⁻¹ (P < 0.05). Women in the control group had a significant increase in the percentage of carbohydrate (4.0%) and a significant decrease in the percentage of fat (4.2%) across the duration of the study.

Despite the supervision of exercise and tight control of EXEE, a wide variation was shown for weight loss (Fig. 1), suggesting compensation in components of energy balance. EI may be the largest source for compensation, and the individual differences for change across the intervention were considerable. The individual variation for EI may diminish weight loss and thus the effect of exercise alone as a primary weight loss strategy. These individuals may be considered “nonresponders” with respect to exercise for weight loss and if identified early during a weight loss program may represent and opportunity for more targeted interventions using diet counseling or energy restriction.

**What are the causes of variability?** There are a range of behavioral (increased food intake, decreased activity, and noncompliance with the exercise) and metabolic (decrease in resting metabolic rate) adaptations that could occur in response to increased EXEE. However, there is strong evidence to suggest that overeating is a pernicious and potent contributor to weight gain and obesity (4). Therefore, increases in EI are likely a strong contributing factor to the modest weight loss often observed in exercise interventions. The processes underpinning any compensation in EI need to be better understood.

**What are the drivers of exercise-induced compensatory eating?** With respect to biological needs and energy balance regulation, the homeostatic processes of appetite control are associated with changes in the orexigenic drive to eat (e.g., hunger), whereas the hedonic processes are associated with reward and the pleasure of eating. Individuals who are more susceptible to exercise-induced compensation could be characterized by an enhanced hunger...
Homeostatic processes or hyperresponsivity to the pleasurable components of food (hedonic processes) or both.

Homeostatic processes. It is important to determine how the homeostatic processes of satiation and satiety are adjusted in response to increased EXEE. Blundell et al. (9,30,31) at the University of Leeds in the UK have demonstrated that exercise can be used as a tool to better understand appetite regulation. The researchers used a 12-wk supervised exercise intervention model to characterize the drivers of compensatory eating. The exercise intervention consisted of five moderately high-intensity (70% VO\textsubscript{2max}) exercise sessions per week, with fixed intensity and duration for all individuals. The EE of each exercise session was approximately 2 MJ. An assay of appetite measures was used to objectively monitor EI and appetite sensations in the research unit. On each test day, after a fixed breakfast, participants were provided with lunch and dinner test meals and an evening snack box ad libitum, each separated by 4 h. This methodological platform and approach is based on several conceptual principles and is designed to provide a comprehensive and flexible model for the study of EE and EI. The key features of the approach are that the exercise is supervised, and the measurements of EI and appetite are conducted under carefully controlled conditions. The 12-wk exercise intervention of Blundell et al. serves as an ideal model of resistance to weight loss and provides an opportunity to help explain why exercise does not work for everyone.

The series of studies by Blundell et al. revealed that 12 wk of exercise exerted different effects on fasting and postprandial appetite sensations. Compensators (i.e., nonresponders) were defined as losing less weight after the exercise intervention than noncompensators (i.e., responders). Compared with their own baseline, compensators experienced marked and significant increases in EI and fasting hunger in response to the exercise intervention (Fig. 3). However, both compensators and noncompensators experienced an increase in satiety immediately after the fixed breakfast meal (Fig. 4). This dual response effect demonstrates that although some people might experience an orexigenic response to supervised exercise, exercise also has the capacity to improve satiety.

Hedonic processes. The hedonic aspects of food (e.g., the pleasure of eating) also influences appetite control (3). Reward plays an important role in the initiation, maintenance, and cessation of eating. Therefore, it is plausible that changes in food and macronutrient preferences might contribute to compensatory increases in EI (17).

Finlayson et al. (21,22) have worked extensively on the theoretical and methodological understanding of behavior related to “liking” and “wanting” food in humans to develop a novel methodology to detect changes or differences in liking and wanting hedonic responses. This procedure was used to demonstrate that individuals identified as compensators because they exhibit an acute compensatory increase in food intake after a single bout of exercise also experience a significant exercise-induced increase in hedonic preference for a range of foods (19). The liking and wanting procedure was also used in the 12-wk exercise intervention.
to assess changes in taste and nutrient preferences under more chronic energy balance conditions. Those who experienced an immediate postexercise increase in liking for food in general and an increased wanting for high-fat sweet foods also experienced less weight loss (20). Interestingly, this response to acute exercise was not influenced by chronic exercise. Therefore, it is possible that some people are more vulnerable to the acute effects of exercise on reward. An enhanced motivational drive or wanting for food after exercise may help to explain why some people overcompensate when given access to food after exercise.

What are the potential homeostatic and non-homeostatic mechanisms underpinning compensation? Obvious homeostatic candidates include the peptides strongly associated with appetite regulation. For example, orexigenic peptides such as ghrelin and anorectic peptides such as GLP-1 could partly explain some of the homeostatic and hedonic responses to exercise-induced EE (45). There is evidence that nonhomeostatic factors are associated with changes in food intake responses to exercise (7) such that individuals with a high level of disinhibition are more susceptible to overcompensation for the EXEE (68). Individuals who are identified as compensators—hence experience lower weight loss compared with noncompensators—could be characterized by a portfolio of homeostatic and/or nonhomeostatic characteristics that partially explain the resistance.

Implications: How Can the Evidence Be Used to Improved the Effectiveness of Weight Management Strategies?

Unless there is a better understanding of why some people fail to lose weight with exercise, the increase in the prevalence of obesity and the associated comorbidities will be unmanageable and unsustainable. Although we acknowledge that people aspire to unrealistically high rates of weight loss, it is futile to continue prescribing exercise and/or dietary interventions using a one-size-fits-all approach and expect people to be content when their efforts produce little or no weight loss. The evidence is suggestive, but not conclusive, that when initiating an exercise program with the intent of losing weight, some individuals compensate by decreasing their non-Ex PA and NEAT. This is likely mediated by subject factors (e.g., age, sex, and body weight) as well as factors related to the exercise program itself (mode, duration, intensity, and frequency), but how each of these factors contribute to the overall effect has not been well studied. Surprisingly, some data suggest that there are individuals who respond to an exercise program by increasing their non-Ex PA and NEAT. Understanding how exercise affects non-Ex PA has health implications far beyond regulating energy balance, as evidence suggests that limiting sedentary behavior has positive effects on many health outcomes, independent of exercise (52). Moreover, it is not clear from the existent literature, when compensation occurs, whether this is intentional (i.e., “I exercised today, so I will take the elevator”) or not. Clearly, there is much more to be learned in this area.

The evidence also suggests that a compensatory increase in EI could also account for variability and offset the expected weight loss. Preliminary evidence suggests that some people experience an orexigenic response to exercise, making them more resistant to exercise-induced weight loss. The potential underlying mechanisms underpinning this compensatory response include appetite peptide response and nonhomeostatic eating behavior traits. Collectively, the evidence indicates that compensatory adaptive responses in EI and NEAT offset the effects of exercise and result in some individuals, achieving little or no weight loss. However, to our knowledge, compensatory changes in non-EX PA and EI have not been comprehensively examined within the same study, and this is a high priority for future research. The model of resistance to exercise-induced weight loss needs to be used strategically in future studies. Identifying the resistance to weight loss and characterizing the adaptive compensatory responses will produce better strategies on how to individually tailor weight management programs. Indeed, energy restriction studies can also benefit from this approach.

Although a compensatory increase in food intake is disappointing to the people directly affected, it serves as an ideal model of resistance to weight loss and provides an opportunity to help explain why exercise does not work for everyone. It could also be used to inform strategies to help obese individuals who may avoid exercise based on their experience of disappointing weight loss. We propose that it is possible to pool the current evidence and use it strategically in the form of evidence-based screening procedures to identify resistance to weight loss due to a compensatory increase in food intake. This approach is novel because it targets resistance to weight loss and individuals susceptible to compensation during exercise interventions. The identification and the characterization of behavioral and physiological characteristics will provide evidence-based screening information that will facilitate the identification of individuals vulnerable to compensation and resistant to weight loss. The early identification of weight loss resistance will eventually permit tailoring of obesity prevention and treatment strategies to suit individuals who are more susceptible to compensatory eating. We also need to better educate people that weight loss is not the only health benefit of exercising. Indeed, there is strong evidence that people experience other health benefits (e.g., reduced blood pressure and waist circumference) despite not attaining the expected, or any, weight loss (32). Our concern is that efforts to increase physical activity among the general population have been undercut by negative media reports portraying exercise as ineffective for weight loss, which may be interpreted by some as therefore ineffective for improving health. We all have a responsibility to eradicate this “bad spin” and to educate the public weight loss not being the sole benefit of exercising; indeed, people can experience health benefits in the absence of weight loss (32).
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