Original Article
CLINICAL TRIALS AND INVESTIGATIONS

Obesity

Baseline Habitual Physical Activity Predicts Weight Loss, Weight Compensation, and Energy Intake During Aerobic Exercise

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Objective: This study aimed to determine whether different measures of habitual physical activity (PA) at baseline predict weight change, weight compensation, and changes in energy intake (EI) during a 24-week supervised aerobic exercise intervention.

Methods: Data from 108 participants (78 women; 48.7 [SD: 11.6] years; BMI 31.4 [SD: 4.6] kg/m^2), randomly assigned to either the moderate-dose exercise group (8 kcal/kg of body weight per week) or the high-dose exercise group (20 kcal/kg of body weight per week) of the Examination of Mechanisms of Exercise-induced Weight Compensation (E-MECHANIC) trial, were analyzed. Moderate-to-vigorous PA (MVPA), steps per day, and PA energy expenditure (PAEE) were measured with SenseWear armbands (BodyMedia, Pittsburgh, Pennsylvania), and total activity energy expenditure and EI were estimated with doubly labeled water, all over 2 weeks, before and toward the end of the intervention. Multiple linear regression models, adjusted for sex, exercise group, and baseline value of the outcome, were used.

Results: Baseline habitual MVPA levels predicted weight change ($\beta = -0.275$; $P = 0.020$), weight compensation ($\beta = -0.238$; $P = 0.043$), and change in EI ($\beta = -0.318$; $P = 0.001$). Associations between baseline PAEE and outcomes were comparable, whereas steps per day and, importantly, total activity energy expenditure (via doubly labeled water) did not significantly predict change in weight-related outcomes.

Conclusions: While acknowledging substantial variability in the data, on average, lower baseline habitual MVPA and PAEE levels were associated with less weight loss from exercise, higher compensation, and increased EI.

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Study Importance

What is already known?

- Exercise is recommended for weight management.
- Exercise-induced weight loss often is less than expected based on measured energy expenditure (EE).
- This is called *weight compensation* and results primarily from increased energy intake (EI).

What does this study add?

- Moderate-to-vigorous physical activity (MVPA) and physical activity EE (PAEE) (≥3 metabolic equivalents) levels prior to engaging in a moderate- to high-dose aerobic exercise intervention predict weight loss, weight compensation, and changes in EI during the intervention.
- Prior MVPA and PAEE have a superior predictive value compared with steps per day and total activity-related EE, as estimated by doubly labeled water, regarding these outcomes.

How might these results change the direction of research?

- Further research is needed to understand why participants with lower baseline habitual MVPA and PAEE levels lose less weight from structured exercise, show higher weight compensation, and increase their EI more than those who are more active at baseline to develop strategies to mitigate this detrimental effect.

Introduction

The prevalence of overweight and obesity has grown into a worldwide epidemic in recent years (1), and excess body weight substantially increases the risk of adverse health conditions (2). Exercise has been shown to support the prevention and management of obesity (3); however, when used for weight loss, exercise interventions consistent with the physical activity (PA) guidelines for weight loss and weight loss maintenance (>225 min/wk of moderate-intensity PA) frequently produce less weight loss than expected based on energy

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expended in exercise (4-7). This discrepancy is called weight compensation (8), and it results primarily from exercise-induced increases in appetite and energy intake (EI) as opposed to changes in metabolism or activity (6,9).

It is unknown whether factors pertaining to one's lifestyle prior to starting an exercise program affect weight compensation and food intake (FI). An individual's habitual PA level at baseline might be such a determinant of the observed difference between actual weight change and predicted weight loss from the energy balance model. As suggested by Westerterp (10), it is possible that a lower habitual PA level at baseline allows an exercise-induced increase in energy expenditure (EE) without or with less compensatory increase in EI. Conversely, and based on previous research indicating that EI and energy balance are better regulated at higher levels of activity-related EE (11,12), lower habitual PA levels at baseline might be associated with larger compensatory increases in EI in response to an exercise-induced increase in EE (10).

To further elucidate the mechanisms for weight compensation in response to exercise, the aim of this analysis was to determine whether different measures of habitual PA at baseline predict weight change, weight compensation, and changes in EI during a 24-week, supervised, controlled aerobic exercise intervention. Specifically, we aimed to compare the predictive value of (1) minutes spent in moderate-to-vigorous PA (MVPA), (2) steps per day, and (3) EE through PA (PAEE), assessed via two validated methods, with regard to these outcomes. Based on previous work (11,12), we hypothesized that participants with lower PA levels at baseline would show greater weight compensation and larger exercise-induced increases in EI. Although PAEE is directly related to the energy balance model and a significant association with our outcomes might be expected, we aimed to additionally assess the association of MVPA and steps per day with our outcomes, as PA recommendations based on these parameters are commonly communicated to patients, and a predictive value of these parameters would consequently be of interest to many clinical and research settings.

Methods

Design and participants

This report is a secondary analysis of the Examination of Mechanisms of Exercise-induced Weight Compensation (E-MECHANIC) study (ClinicalTrials.gov identifier NCT01264406) that was approved by the institutional review board and conducted between November 2010 and December 2015 at Pennington Biomedical Research Center (Baton Rouge, Louisiana). The complete design, methods, and primary outcomes of the E-MECHANIC study have been previously published (6,13). In brief, this 24-week randomized controlled trial recruited 198 healthy men and women with overweight or obesity (BMI ≥ 25 kg/m^2 to ≤ 45 kg/m^2) and low levels of PA (≤ 20 minutes of structured exercise on ≤ 3 d/wk based on self-report; < 8,000 steps per day (14) assessed during 1 week of accelerometer data [SenseWear armband; BodyMedia, Pittsburgh, Pennsylvania]). Participants were randomly allocated in a 1:1:1 ratio to either a moderate-dose exercise group (8 kcal/kg of body weight per week [KKW]), a high-dose exercise group (20 KKW), or a nonexercise control group (13). The selected exercise doses reflect recommendations for general health (8 KKW) and for weight loss (20 KKW) (15). Exercise intensity during the supervised exercise sessions was self-selected between 65% and 85% peak oxygen uptake, and sessions varied in length to meet each participant's EE goal (16).

Participants were excluded if they were currently participating in a weight loss program (and/or had ≥ 4-kg weight change in the past 6 months), were currently pregnant or had been pregnant within the past 6 months, or were diagnosed with diabetes, cardiovascular disease, or arrhythmia. All participants provided written informed consent prior to inclusion in the study.

The primary aim of the E-MECHANIC study was to identify mechanisms of exercise-induced weight compensation (i.e., less than expected weight loss) by examining the effect of the two different doses of exercise training on EI over the 24-week intervention period. The study found significantly higher weight compensation in the high-dose exercise group compared with the moderate-dose exercise group, which resulted primarily from increased EI and concomitant increases in appetite (6).

In this report, to examine the impact of baseline levels of habitual PA on outcome measures during a supervised exercise intervention, only participants allocated to the two exercise groups ($n = 110$) who completed the trial per protocol were included in the main analyses. Demographics of those exercisers who did not complete the trial ($n = 25$) did not differ significantly from completers (all P values ≥ 0.093).

Outcome variables

Anthropometry and body composition. At baseline and follow-up, body weight was assessed under fasting conditions using a Tanita scale (Arlington Heights, Illinois), and waist circumference was determined using a nonextensible tape measure (Gulick II; Sammons Preston, Chicago, Illinois). Dual-energy x-ray absorptiometry (DXA) (Lunar iDXA and Encore software version 13.60; GE Healthcare, Madison, Wisconsin) was used to assess fat mass.

Weight compensation. Weight compensation is the difference between the amount of weight loss predicted from exercise-associated EE and observed weight loss from baseline to follow-up (actual – predicted weight change). Predicted weight loss was calculated using a validated dynamic energy balance model that overcomes the limitations of the conventional assumption that 1 kg of body weight equals 7,700 kcal/kg (7,17,18), accounting for adaptations that occur when body mass changes, including adaptations to resting metabolic rate (RMR), dietary-induced thermogenesis, and nonexercise activity thermogenesis (19).

EI. EI was estimated with doubly labeled water (DLW) and FI tests at baseline and follow-up. DLW data were collected over a 2-week period at both time points. DLW measures total daily EE (TDEE), which equals total daily EI during weight stability (20,21). The DLW period at baseline occurred before participants began exercising. During the DLW period at follow-up, participants exercised at their prescribed dose. During both DLW periods, participants were weight stable (≤ 0.15-kg change in weight during the 2-week period). Change in EI by DLW was calculated with and without adjusting for change in RMR. For participants who were weight stable or who gained weight during the 6-month trial, follow-up TDEE was subtracted from baseline TDEE to quantify the change in EI, as any changes in RMR from weight gain are reflected in the TDEE value from DLW. For participants who lost weight during the intervention, this calculation fails to consider decreased basal metabolic requirements;

therefore, the difference between RMR from baseline to follow-up was added to the difference in TDEE for these participants to quantify the change in EI during the intervention period.

In addition, at baseline and follow-up, validated laboratory-based FI tests were conducted at lunch and dinner. Following a standard breakfast between 0700 and 0800 consisting of a 190-kcal nutrition bar, participants returned to the center between 1100 and 1200 to complete their test lunch, which consisted of ad libitum sandwiches, potato chips, cookies, water, and choice of an artificially-sweetened soda or tea or sugar-sweetened soda or tea. At 5.5 hours after the start of their lunch, participants returned to the center again to complete their dinner meal, which consisted of a previously described 18-food-item buffet meal (22), presented to the participants all at once within arm's reach. At both test meals, participants were instructed to eat as much or as little of the presented food items as desired and to avoid distractions (e.g., mobile phone use), focusing completely on the meal. FI testing at follow-up occurred at least 24 hours after the last exercise session. We quantified FI at lunch and dinner by covertly weighing food provision and waste and combined EI (kilocalories) from both meals for the analyses presented in this paper.

RMR. We measured RMR with indirect calorimetry over 30 minutes after a 12-hour overnight fast with Max-II metabolic carts (AEI Technologies, Pittsburgh, Pennsylvania) at baseline and follow-up. The change in RMR was calculated as RMR at follow-up minus RMR at baseline. Calculations adjusted for change in body composition (i.e., lean mass measured with DXA), sex, and age did not differ meaningfully from the basic change scores; hence, the basic change scores are reported.

PA. SenseWear armbands measured the minutes per day spent in activities of different intensities, steps per day, and PAEE during a 2-week period at baseline and follow-up. In the MVPA-related analyses presented in this paper, only activities ≥3 metabolic equivalents (MET) are included (3), and, congruent with the most recent Physical Activity Gudelines for Americans (23), all MVPA was considered rather than only that accumulated in bouts of at least 10 minutes as recommended previously. The SenseWear software classifies any activity ≤3 MET as sedentary; hence, PAEE included only activities ≥3 MET (24). Participants were instructed to wear the armbands continuously and to take them off only during activities involving water. The SenseWear armbands detect and record wear time, and only full days of data, defined as a wear time of ≥95% (equating to 22 hours and 48 minutes or 1,368 minutes), were included in the analyses. During the monitoring period at follow-up, participants exercised at their prescribed dose; therefore, PA data collected by the SenseWear armbands during these sessions were removed before analysis. To account for differing wear times between participants caused by varying durations of the exercise sessions and different nonwear times within the time frame of 22 hours and 48 minutes, the total number of minutes of daily activity was divided by the total daily wear time (minutes) and then extrapolated out to a 24-hour day.

In addition to the PAEE estimates by the SenseWear armband, we calculated the gold standard of activity EE (AEE) based on the DLW-estimated TDEE (DLW-AEE = TDEE − [RMR + thermic effect of food]), which captures all PA-related EE. The thermic effect of food was estimated as 10% of TDEE.

Questionnaires. Retrospective visual analog scales assessed average ratings of appetite during the previous week (25) at baseline and follow-up. The Eating Inventory (26) was used to assess eating behavior, specifically restraint, disinhibition, and hunger at baseline and follow-up. Additional questionnaires included the Multifactorial Assessment of Eating Disorders Symptoms (27), Food Preference Questionnaire (28), and Food Craving Inventory (29).

Statistical analyses

The distribution of variables was verified using the Shapiro-Wilk test and by visual inspection of histograms and quantile-quantile plots of the residuals. The influence of outliers was estimated using studentized residuals, and multicollinearity was assessed via the variance inflation factor. Exclusion of outliers (≤2 for all models) did not change the results meaningfully; therefore, the models including outliers are reported. Descriptive data are reported as mean and SD. We used multiple linear regression models to estimate the effect of SenseWear-assessed habitual MVPA levels (minutes per day), steps per day, PAEE, and DLW-estimated AEE at baseline on weight change (kilograms) and weight compensation (kilograms), as well as on changes in waist circumference (centimeters), fat mass (kilograms), EI (by DLW in kilocalories per day), EI during FI testing (kilocalories at a test lunch and test dinner combined), RMR (kilocalories per day), and habitual MVPA levels (minutes per day), steps per day, and PAEE (kilocalories per day). Covariates in the models were sex, exercise group, and baseline value of the respective outcome. Results of analyses that included age, ethnicity, and baseline BMI did not differ meaningfully; therefore, the models without these additional covariates are reported. Similarly, interaction terms for sex and exercise group were nonsignificant; therefore, results are reported without the interaction terms in the models. Pearson product moment correlation analysis was used to assess the association between habitual MVPA levels and questionnaire-assessed eating behaviors at baseline. The analyses were conducted using SPSS Statistics for Windows, version 25 (IBM Corp., Armonk, New York), with the significance level set to 0.05 (two-sided).

Results

Two participants were excluded from the analyses because they did not provide baseline accelerometer data. Baseline characteristics of all included 108 participants are shown in Table 1. Baseline characteristics of the control group (not included in main analyses) are provided in Supporting Information Table S1. At baseline, average wear time of the armbands was 1,415.2 min/d (SD: 9.1 min/d), equating to 98.3% (SD: 0.6%); at follow-up, average wear time (excluding study-related exercise sessions) was 1,393.1 min/d (SD: 31.7 min/d) or 97.8% (SD: 2.1%). Baseline habitual MVPA was 61.2 min/d (SD: 46.9 min/d) on average, with an average intensity of 3.7 MET (SD: 0.2 MET) and 99.2% (SD: 0.2%) of all MVPA below 6 MET. Total habitual PA, measured as steps per day, was 6,300 (SD: 2,301) at baseline. Total duration and intensity of daily habitual MVPA, habitual steps per day, and habitual PAEE (all outside of the structured exercise sessions) did not change significantly from baseline to follow-up (all P values ≥ 0.094). Average self-chosen exercise intensity during the intervention was 6.9 MET (SD: 1.0 MET), with no significant difference between the 20-KKW group and the 8-KKW group (P = 0.074). This average exercise intensity corresponds to vigorous PA (3).

Table 2 and Figure 1 (MVPA), Table 3 (steps per day), Table 4 (SenseWear PAEE), and Table 5 (DLW-estimated AEE) show the results of the multiple linear regression analyses. We found significant negative

TABLE 1 Baseline characteristics of 108 included participants

	All (*N* = 108)	8 KKW (*n* = 57)	20 KKW (*n* = 51)
Female, *n* (%)	78 (72.2)	42 (73.7)	36 (70.6)
Ethnicity, *n* (%)			
Caucasian	74 (68.5)	37 (64.9)	37 (72.5)
African American	32 (29.6)	20 (35.1)	12 (23.5)
Hispanic/other	2 (1.9)	0 (0.0)	2 (4.0)
Age (y)	48.7 (11.6)	48.3 (11.0)	49.1 (12.4)
Height (cm)	167.1 (8.2)	167.2 (8.7)	167.0 (7.6)
Weight (kg)	87.8 (15.5)	89.0 (16.0)	86.5 (15.1)
Waist circumference (cm)	97.8 (12.0)[a]	98.7 (12.1)[b]	97.0 (11.9)
BMI (kg/m^2)	31.4 (4.6)	31.8 (4.6)	30.9 (4.5)
Fat mass (kg)	36.8 (9.8)	37.3 (9.7)	36.2 (9.9)
EI, DLW (kcal/d)	2,497.7 (462.5)	2,530.1 (438.8)	2,461.5 (489.4)
EI, buffet (kcal at lunch and dinner combined)	1,795.5 (550.7)	1,820.1 (489.7)	1,768.1 (615.7)
RMR (kcal/d)	1,529.1 (297.1)[a]	1,525.8 (261.2)	1,532.8 (334.7)
MVPA (min/d)	61.2 (46.9)	63.9 (49.5)	58.2 (44.0)
Average intensity of MVPA (MET)	3.7 (0.2)	3.8 (0.2)	3.7 (0.2)
Steps per day	6,300 (2,301)	6,576 (2,613)	5,992 (1,870)
PAEE, SenseWear (kcal/d)	336.7 (257.8)	349.1 (257.6)	322.9 (259.8)
AEE, DLW (kcal/d)	717.5 (216.6)[a]	749.3 (201.3)[b]	682.6 (229.2)

Data are mean (SD) if not stated otherwise. ANOVA (continuous variables) and χ^2 test (categorical variables) used to test for baseline differences between two groups. The 8-KKW and 20-KKW groups did not differ significantly in any baseline measures presented in table.
[a]Data available in 107 of 108 participants.
[b]Data available in 56 of 57 participants.
AEE, activity energy expenditure; DLW, doubly labeled water; EI, energy intake; KKW, kcal/kg of body weight/wk; MET, metabolic equivalent; MVPA, moderate-to-vigorous physical activity; PAEE, physical AEE; RMR, resting metabolic rate.

associations between baseline habitual MVPA levels and weight change ($P = 0.020$; Figure 1A), weight compensation ($P = 0.043$; Figure 1B), and change in DLW-estimated EI both with ($P = 0.001$; Figure 1C) and without ($P = 0.001$; not shown in Figure 1) adjustment for change in RMR. The analyses further showed significant negative associations between baseline habitual MVPA levels and changes in waist circumference ($P = 0.030$), fat mass ($P = 0.025$), and habitual MVPA levels ($P < 0.001$; Figure 1D). Although there is substantial variability in the data (Figure 1), these results suggest that, on average, for every 15-minute decrease in habitual MVPA per day at baseline, participants lost 0.23 kg less weight, compensated 0.20 kg more, and increased DLW-estimated daily EI from baseline to follow-up by 21.5 kcal/d (adjusted for RMR of 23.2 kcal/d).

Compared with women, men lost 1.9 kg less weight, compensated 1.8 kg more, and increased DLW-estimated EI by 182.4 kcal/d (adjusted for RMR of 171.6 kcal/d) (Table 2). Further, compared with participants in the 8-KKW group, participants in the 20-KKW group lost 1.4 kg more weight but showed 1-kg-higher weight compensation (Table 2).

Baseline levels of habitual MVPA were significantly correlated with the disinhibition subscale of the Eating Inventory ($r = -0.229$; $P = 0.018$) and with the binge eating subscale of the Multifactorial Assessment of Eating Disorders Symptoms ($r = -0.230$; $P = 0.018$). No other correlations between baseline levels of habitual MVPA and eating behavior–related constructs, as assessed by questionnaires, were significant.

Baseline PA levels measured as steps per day significantly predicted change in steps per day ($\beta = -0.382$; $P < 0.001$); however, no associations between steps per day at baseline and change in any other of the outcome variables were significant (Table 3). Associations between average intensity (MET) of baseline habitual MVPA and all outcomes were nonsignificant (all $P > 0.1$, data not shown).

Associations between baseline habitual PAEE and outcomes were similar to those of baseline habitual MVPA, albeit slightly attenuated, as indicated by the regression coefficients (Table 4). DLW-estimated AEE only significantly predicted change in DLW-estimated EI (Table 5); all other associations were nonsignificant (all P values ≥ 0.709).

As described above, habitual MVPA, steps per day, and PAEE (all outside of structured exercise sessions) did not change significantly from baseline to follow-up on a group level. However, on an individual level, baseline habitual MVPA (Table 2, Figure 1D), steps per day (Table 3), and PAEE (Table 4) were significantly inversely associated with change in the respective measure.

Supporting Information Tables S2-S5 show the results of the multiple linear regression analyses for the control group. For habitual MVPA (Supporting Information Table S2), steps per day (Supporting Information Table S3), and PAEE (Supporting Information Table S4), only change in each PA measure was significantly associated with the respective baseline value. A Fisher r-to-z transformation revealed that the correlation coefficients for habitual MVPA did not differ between exercisers and the control

TABLE 2 Multiple linear regression analysis for association between baseline habitual MVPA levels and changes in body weight, fat mass, EI, and MVPA levels

	R^2	B	SE	β	*P*
Weight change (kg)	0.124				
Habitual MVPA at baseline (min/d)		−0.015	0.006	−0.275	**0.020**
Weight at baseline (kg)		−0.033	0.019	−0.197	0.095
Sex[a]		1.880	0.716	0.328	**0.010**
Group[b]		−1.442	0.483	−0.280	**0.004**
Waist circumference change (cm)	0.074				
Habitual MVPA at baseline (min/d)		−0.019	0.009	−0.267	**0.030**
Waist circumference at baseline (cm)		−0.034	0.034	−0.122	0.317
Sex[a]		1.357	0.947	0.185	0.155
Group[b]		−1.325	0.638	−0.201	**0.040**
Weight compensation (kg)	0.127				
Habitual MVPA at baseline (min/d)		−0.013	0.006	−0.238	**0.043**
Weight at baseline (kg)		−0.011	0.020	−0.064	0.585
Sex[a]		1.826	0.723	0.315	**0.013**
Group[b]		1.049	0.488	0.201	**0.034**
Fat mass change (kg)	0.153				
Habitual MVPA at baseline (min/d)		−0.014	0.006	−0.257	**0.025**
Fat mass at baseline (kg)		−0.070	0.027	−0.271	**0.011**
Sex[a]		1.173	0.562	0.209	**0.039**
Group[b]		−1.478	0.462	−0.293	**0.002**
Change in EI, DLW (kcal/d)	0.220				
Habitual MVPA at baseline (min/d)		−1.546	0.442	−0.336	**0.001**
EI, DLW at baseline (kcal/d)		−0.206	0.054	−0.443	**<0.001**
Sex[a]		182.400	57.888	0.381	**0.002**
Group[b]		−11.680	37.824	−0.027	0.758
Change in EI, adjusted DLW kcal/d)[c]	0.205				
Habitual MVPA at baseline (min/d)		−1.436	0.438	−0.318	**0.001**
EI, DLW at baseline (kcal/d)		−0.195	0.054	−0.427	**<0.001**
Sex[a]		171.594	57.370	0.365	**0.003**
Group[b]		3.388	37.486	0.008	0.928
Change EI, buffet (kcal at lunch and dinner combined)	0.158				
Habitual MVPA at baseline (min/d)		−0.938	0.951	−0.098	0.326
EI, buffet at baseline (kcal at lunch and dinner combined)		−0.287	0.077	−0.354	**<0.001**
Sex[a]		319.655	102.282	0.322	**0.002**
Group[b]		21.191	80.993	0.024	0.794
Change in RMR, indirect calorimetry (kcal/d)	0.067				
Habitual MVPA at baseline (min/d)		0.210	0.641	0.036	0.744
RMR at baseline (kcal/d)		−0.160	0.109	−0.195	0.146
Sex[a]		−44.702	77.817	−0.081	0.567
Group[b]		41.109	50.933	0.083	0.422
Change in habitual MVPA (min/d)	0.223				
Habitual MVPA at baseline (min/d)		−0.274	0.058	−0.450	**<0.001**
Sex[a]		−2.425	5.996	−0.039	0.687
Group[b]		7.963	4.992	0.141	0.114

Bold font indicates statistical significance ($P < 0.05$). Independent variable in all models: habitual MVPA levels (min/d) at baseline.
[a]Female = 0, male = 1.
[b]8 kcal/kg of body weight per week = 0, 20 kcal/kg of body weight per week = 1.
[c]Adjusted for change in RMR.
DLW, doubly labeled water; EI, energy intake; MVPA, moderate-to-vigorous physical activity; RMR, resting metabolic rate.

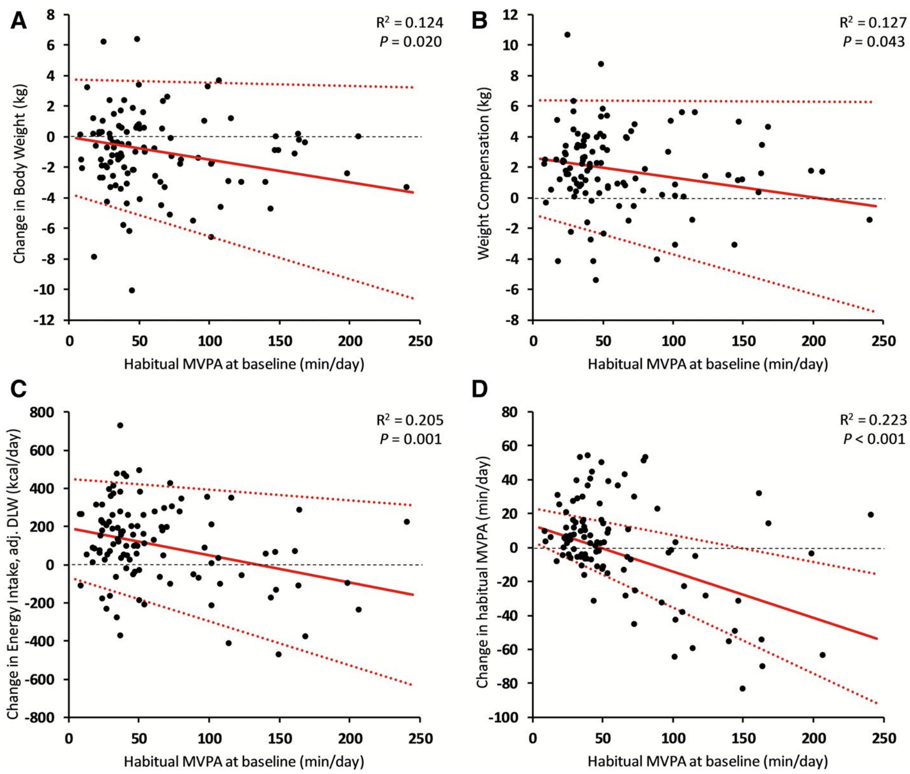

Figure 1 Association between habitual moderate-to-vigorous physical activity (MVPA) at baseline and change in (**A**) body weight, (**B**) weight compensation, (**C**) change in doubly labeled water (DLW)-measured energy intake, adjusted for change in resting metabolic rate, and (**D**) change in MVPA. Regression line (solid line) in each panel represents the relationship for the fully adjusted model with 95% confidence intervals (dotted lines). [Color figure can be viewed at wileyonlinelibrary.com]

group (data not shown). For habitual PAEE, the difference between exercisers and control participants was significant, with a markedly more pronounced association for the control participants.

Discussion

To our knowledge, this is the first study to determine and compare the effect of prior habitual MVPA, steps per day, and PAEE on changes in weight, EI, RMR, and MVPA, steps per day, and PAEE in response to a moderate- to high-dose aerobic exercise intervention. The results show that, on average, lower levels of habitual MVPA and/or PAEE at baseline are related to less weight loss and greater weight compensation during the exercise intervention, supporting our hypothesis. Importantly, lower levels of habitual MVPA and/or PAEE at baseline were also associated with greater increases in EI, which likely contributed to the lower weight compensation in those with higher baseline levels of habitual MVPA and/or PAEE, particularly because changes in RMR were not associated with baseline habitual MVPA and/or PAEE levels. Interestingly, we found substantial heterogeneity in the weight loss/compensation response, which likely influenced the results of the regression analysis. Although many participants across all baseline MVPA levels successfully lost weight during the intervention, some participants with low MVPA at baseline actually gained weight, whereas no one with higher baseline MVPA gained weight.

In line with previous findings (30,31), participants with lower habitual MVPA levels showed higher tendencies for disinhibition and binge eating at baseline, factors that may have influenced the greater increases in EI and subsequent greater weight compensation in response to the exercise intervention. This assumption is supported by previous findings showing that individuals with lower levels of measured MVPA have weaker appetite control and satiety response to food and thus have an impaired regulation of energy balance compared with their more active counterparts (32-34). Consequently, in our study, participants with lower levels of habitual MVPA and/or PAEE at baseline may have had a more impaired regulation of energy balance than those with higher levels of habitual MVPA and/or PAEE, and this became particularly apparent with the onset of the exercise intervention. Although participants were weight stable during the 2-week baseline accelerometer assessment, suggesting an adequately regulated energy balance during that period, the exercise intervention and, subsequently, the substantial increase in daily EE disrupted this balance. This disruption may have revealed the potentially impaired energy balance regulation in participants with lower baseline levels of habitual MVPA and/or PAEE, as the intervention-related increases in MVPA and/or PAEE (i.e., structured exercise sessions) were

TABLE 3 Multiple linear regression analysis for association between habitual steps per day at baseline and changes in body weight, fat mass, EI, and steps per day

	R^2	B	SE	β	*P*
Weight change (kg)	0.091				
Habitual PA at baseline (steps/d)		−0.0001	0.0001	−0.1233	0.211
Weight at baseline (kg)		−0.0156	0.0177	−0.0945	0.379
Sex[a]		1.0394	0.6043	0.1812	0.088
Group[b]		−1.3705	0.4924	−0.2664	**0.006**
Waist circumference change (cm)	0.075				
Habitual PA at baseline (steps/d)		−0.0001	0.0001	−0.2324	0.057
Waist circumference at baseline (cm)		−0.0223	0.0311	−0.0807	0.476
Sex[a]		0.5600	0.7905	0.0763	0.480
Group[b]		−1.3730	0.6409	−0.2082	**0.035**
Weight compensation (kg)	0.098				
Habitual PA at baseline (steps/d)		−0.0001	0.0001	−0.0818	0.404
Weight at baseline (kg)		0.0052	0.0179	0.0310	0.772
Sex[a]		1.0611	0.6094	0.1828	0.085
Group[b]		1.1322	0.4965	0.2174	**0.025**
Fat mass change (kg)	0.124				
Habitual PA at baseline (steps/d)		−0.0001	0.0001	−0.1223	0.212
Fat mass at baseline (kg)		−0.0505	0.0252	−0.1954	**0.048**
Sex[a]		0.6968	0.5256	0.1238	0.188
Group[b]		−1.4435	0.4715	−0.2860	**0.003**
Change in EI, DLW (kcal/d)	0.147				
Habitual PA at baseline (steps/d)		−0.0138	0.0089	−0.1475	0.128
EI, DLW at baseline (kcal/d)		−0.1923	0.0589	−0.4127	**0.001**
Sex[a]		112.1006	58.4187	0.2340	0.058
Group[b]		−7.8955	39.6422	−0.0183	0.843
Change in EI, adjusted DLW kcal/d)[c]	0.140				
Habitual PA at baseline (steps/d)		−0.0128	0.0088	−0.1397	0.150
EI, DLW at baseline (kcal/d)		−0.1823	0.0580	−0.3985	**0.002**
Sex[a]		106.2766	57.5923	0.2260	0.068
Group[b]		6.8974	39.0800	0.0163	0.860
Change EI, buffet (kcal at lunch and dinner combined)	0.173				
Habitual PA at baseline (steps/d)		−0.0298	0.0177	−0.1537	0.095
EI, buffet at baseline (kcal at lunch and dinner combined)		−0.2771	0.0769	−0.3416	**<0.001**
Sex[a]		288.0988	93.6300	0.2901	**0.003**
Group[b]		10.5387	80.6567	0.0118	0.896
Change in RMR, indirect calorimetry (kcal/d)	0.066				
Habitual PA at baseline (steps/d)		−0.0006	0.0109	−0.0057	0.956
RMR at baseline (kcal/d)		−0.1629	0.1086	−0.1984	0.137
Sex[a]		−35.6318	72.7619	−0.0647	0.626
Group[b]		38.1756	51.0429	0.0774	0.456
Change in habitual PA (steps/d)	0.173				
Habitual PA at baseline (steps/d)		−0.3591	0.0870	−0.3823	**<0.001**
Sex[a]		74.8373	438.7446	0.0156	0.865
Group[b]		500.2422	399.3502	0.1159	0.213

Bold font indicates statistical significance ($P<0.05$). Independent variable in all models: habitual PA levels (steps/d) at baseline.
[a]Female = 0, male = 1.
[b]8 kcal/kg of body weight per week = 0, 20 kcal/kg of body weight per week = 1.
[c]Adjusted for change in RMR.
DLW, doubly labeled water; EI, energy intake; PA, physical activity; RMR, resting metabolic rate.

TABLE 4 Multiple linear regression analysis for association between baseline habitual PAEE as assessed by SenseWear armband and changes in body weight, fat mass, EI, and SenseWear-assessed PAEE

	R^2	B	SE	β	*P*
Weight change (kg)	0.121				
Habitual PAEE at baseline (kcal/d)		−0.003	0.001	−0.260	**0.024**
Weight at baseline (kg)		−0.023	0.018	−0.136	0.208
Sex[a]		1.856	0.718	0.324	**0.011**
Group[b]		−1.400	0.481	−0.272	**0.004**
Waist circumference change (cm)	0.059				
Habitual PAEE at baseline (kcal/d)		−0.003	0.002	−0.220	0.074
Waist circumference at baseline (cm)		−0.019	0.032	−0.068	0.553
Sex[a]		1.222	0.980	0.167	0.215
Group[b]		−1.265	0.641	−0.192	0.051
Weight compensation (kg)	0.121				
Habitual PAEE at baseline (kcal/d)		−0.002	0.001	−0.212	0.065
Weight at baseline (kg)		−0.001	0.018	−0.008	0.944
Sex[a]		1.756	0.727	0.303	**0.017**
Group[b]		1.093	0.487	0.210	**0.027**
Fat mass change (kg)	0.156				
Habitual PAEE at baseline (kcal/d)		−0.003	0.001	−0.261	**0.021**
Fat mass at baseline (kg)		−0.059	0.025	−0.230	**0.019**
Sex[a]		1.376	0.597	0.245	**0.023**
Group[b]		−1.462	0.460	−0.290	**0.002**
Change in EI, DLW (kcal/d)	0.228				
Habitual PAEE at baseline (kcal/d)		−0.322	0.088	−0.385	**<0.001**
EI, DLW at baseline (kcal/d)		−0.169	0.055	−0.362	**0.003**
Sex[a]		186.132	57.668	0.389	**0.002**
Group[b]		−8.899	37.567	−0.021	0.813
Change in EI, adjusted DLW kcal/d)[c]	0.212				
Habitual PAEE at baseline (kcal/d)		−0.298	0.087	−0.363	**0.001**
EI, DLW at baseline (kcal/d)		−0.160	0.055	−0.351	**0.004**
Sex[a]		174.869	57.211	0.372	**0.003**
Group[b]		5.993	37.269	0.014	0.873
Change EI, buffet (kcal at lunch and dinner combined)	0.164				
Habitual PAEE at baseline (kcal/d)		−0.246	0.184	−0.142	0.184
EI, buffet at baseline (kcal at lunch and dinner combined)		−0.284	0.077	−0.350	**<0.001**
Sex[a]		351.316	107.794	0.354	**0.002**
Group[b]		19.233	80.663	0.022	0.812
Change in RMR, indirect calorimetry (kcal/d)	0.066				
Habitual PAEE at baseline (kcal/d)		−0.025	0.127	−0.023	0.846
RMR at baseline (kcal/d)		−0.163	0.108	−0.198	0.136
Sex[a]		−29.457	79.345	−0.053	0.711
Group[b]		37.075	51.013	0.075	0.469
Change in habitual PAEE (kcal/d)	0.289				
Habitual PAEE at baseline (kcal/d)		−0.400	0.069	−0.564	**<0.001**
Sex[a]		52.423	38.970	0.131	0.182
Group[b]		43.443	30.461	0.121	0.157

Bold font indicates statistical significance ($P < 0.05$). Independent variable in all models: habitual PAEE (kcal/d) at baseline as assessed by SenseWear armband.
[a]Female = 0, male = 1.
[b]8 kcal/kg of body weight per week = 0, 20 kcal/kg of body weight per week = 1.
[c]Adjusted for change in RMR.
DLW, doubly labeled water; EI, energy intake; PAEE, physical activity energy expenditure; RMR, resting metabolic rate.

TABLE 5 Multiple linear regression analysis for association between baseline habitual AEE as assessed by DLW and changes in body weight, fat mass, and EI

	R^2	B	SE	β	*P*
Weight change (kg)	0.076				
Habitual AEE at baseline (kcal/d)		−0.001	0.001	−0.041	0.709
Weight at baseline (kg)		−0.009	0.018	−0.053	0.630
Sex[a]		1.009	0.620	0.177	0.107
Group[b]		−1.250	0.496	−0.244	**0.013**
Waist circumference change (cm)	0.027				
Habitual AEE at baseline (kcal/d)		0.001	0.002	0.035	0.749
Waist circumference at baseline (cm)		0.003	0.030	0.012	0.911
Sex[a]		0.098	0.829	0.014	0.906
Group[b]		−0.999	0.654	−0.153	0.130
Weight compensation (kg)	0.098				
Habitual AEE at baseline (kcal/d)		−0.001	0.001	−0.013	0.906
Weight at baseline (kg)		0.009	0.018	0.053	0.626
Sex[a]		1.031	0.624	0.178	0.101
Group[b]		1.237	0.499	0.237	**0.015**
Fat mass change (kg)	0.109				
Habitual AEE at baseline (kcal/d)		−0.001	0.001	−0.019	0.854
Fat mass at baseline (kg)		−0.040	0.025	−0.155	0.111
Sex[a]		0.749	0.583	0.134	0.202
Group[b]		−1.324	0.479	−0.263	**0.007**
Change in EI, DLW (kcal/d)	0.171				
Habitual AEE at baseline (kcal/d)		−0.317	0.137	−0.317	**0.023**
EI, DLW at baseline (kcal/d)		−0.095	0.078	−0.204	0.223
Sex[a]		99.726	58.661	0.208	0.092
Group[b]		−13.740	39.562	−0.032	0.729
Change in EI, adjusted DLW kcal/d)[c]	0.167				
Habitual AEE at baseline (kcal/d)		−0.320	0.135	−0.326	**0.020**
EI, DLW at baseline (kcal/d)		−0.082	0.076	−0.179	0.287
Sex[a]		92.237	57.688	0.196	0.113
Group[b]		0.181	38.906	0.001	0.996
Change in EI, buffet (kcal at lunch and dinner combined)	0.149				
Habitual AEE at baseline (kcal/d)		0.070	0.210	0.034	0.741
EI, buffet at baseline (kcal at lunch and dinner combined)		−0.295	0.079	−0.366	**<0.001**
Sex[a]		264.367	101.080	0.267	**0.010**
Group[b]		25.578	82.706	0.029	0.758
Change in RMR, indirect calorimetry (kcal/d)	0.067				
Habitual AEE at baseline (kcal/d)		0.043	0.128	0.038	0.736
RMR at baseline (kcal/d)		−0.165	0.108	−0.201	0.132
Sex[a]		−42.818	75.767	−0.078	0.573
Group[b]		42.681	51.771	0.087	0.412

Bold font indicates statistical significance ($P < 0.05$). Independent variable in all models: habitual AEE (kcal/d) at baseline as assessed by DLW (total daily energy expenditure − [RMR + thermic effect of eating]) Thermic effect of food estimated as 10% of total daily energy expenditure.
[a]Female = 0, male = 1.
[b]8 kcal/kg of body weight per week = 0, 20 kcal/kg of body weight per week = 1.
[c]Adjusted for change in RMR.
AEE, activity energy expenditure; DLW, doubly labeled water; EI, energy intake; RMR, resting metabolic rate.

met by increases in EI, leading to the observed weight compensation. Participants with higher MVPA and/or PAEE levels at baseline might have already experienced this compensatory effort before the start of the intervention, explaining, at least partially, the observed results. In addition to being driven by homeostatic mechanisms such as the aforementioned changes in appetite and satiety, the observed increases in EI may also be related to hedonic processes such as food reward behaviors (35).

It is noteworthy that although habitual MVPA and/or PAEE levels did not change on a group level, on an individual level, these parameters were significantly inversely associated with change in the respective measure, indicating the substitution of habitual PA with prescribed PA (i.e., structured exercise session) in some participants (36,37). As shown by the results of a Fisher *r*-to-*z* transformation, however, the correlation coefficients for habitual MVPA did not differ between exercisers and the control group, suggesting that any decrease in MVPA in the exercisers was likely not caused by the structured exercise sessions but instead ocurred more likely because of regression to the mean. For habitual PAEE, the difference between exercisers and control participants was significant, with a substantially more pronounced association for the control participants, suggesting that the structured exercise sessions actually protected against decreases in habitual PAEE. It is further noteworthy that participants with greater prior habitual MVPA and/or PAEE remained more active compared with those with lower levels ($B = -0.274$ [MVPA] and $B = -0.400$ [PAEE]). Therefore, considering the magnitude of the change in habitual MVPA and/or PAEE levels and, more importantly, the opposite directionality compared with weight change, it is unlikely that the decrease in habitual MVPA and/or PAEE affected participants' weight compensation. Rather, higher absolute levels of MVPA and/or PAEE at follow-up, along with the reduced increase in EI during the intervention, contributed to the lower weight compensation in those who were more active at baseline.

The identification of baseline habitual MVPA and/or PAEE levels as predictors of weight loss, weight compensation, and changes in EI in this study may have important ramifications for future exercise interventions targeting weight loss. Less than expected weight loss from exercise likely leads to frustration and possibly causes discontinuation of the newly started exercise regimen because of the perceived lack of benefit. Assessing prior habitual PA levels may help determine when the exercise prescription should be combined with a concomitant lifestyle, dietary, or possibly pharmacological intervention to counteract weight compensation and increase the weight loss intervention–related health benefits.

Although habitual MVPA and PAEE predicted our outcomes quite comparably, daily step counts at baseline did not have the same predictive value with regard to weight loss, weight compensation, or EI during the intervention as habitual MVPA and/or PAEE. The better predictive value of PAEE compared with steps per day was expected because of the fact that PAEE is directly related to the energy balance model. The better predictive value of habitual MVPA compared with steps per day is likely because of the fact that MVPA includes an intensity component, whereas steps per day does not. Therefore, to identify individuals with a higher risk for exercise-induced weight compensation, baseline levels of habitual MVPA or PAEE should be considered. It should be noted that AEE, as estimated by DLW, did not predict most of our outcomes, with a substantial discrepancy compared with the associations from SenseWear-assessed PAEE. This suggests that the intensity component included in PAEE (and MVPA) made these parameters better predictors with regard to our outcomes. Therefore, total AEE seems to be less important than EE at an intensity ≥3 MET, which is different from Mayer et al.'s original suggestion (11,12). The use of MVPA as a predictor offers the advantages of being accurately assessable via most current accelerometers and of accelerometer data being more straightforward compared with PAEE data such as that of the SenseWear armband, which is based on a complex pattern-recognition algorithm consisting of heat flux, skin temperature, near-body ambient temperature, and galvanic skin response, in addition to the accelerometer-recorded activity counts.

The present study has several strengths. E-MECHANIC was a large randomized controlled trial, in which exercise dose was strictly monitored and supervised. Habitual PA was measured with validated accelerometers that allow an estimation of the intensity and EE of habitual PA. Additionally, EE and EI (via DLW) and RMR (via indirect calorimetry) were measured with the gold-standard methods to comprehensively assess all aspects of energy balance. The assessment of EI via validated laboratory-based FI tests and via DLW over 2 weeks is a particularly major strength, as self-reported EI, which is still commonly used in many trials today, has been found to be fundamentally inaccurate (38,39). A limitation of this analysis is that although PA assessment at follow-up was performed while participants were still exercising at their prescribed dose, we did not measure habitual MVPA, steps per day, and PAEE continuously throughout the intervention period and thus have no record of the effect of the exercise training on these outcomes over the course of the intervention.

In conclusion, taking into account the substantial variability in the data, our results show that habitual MVPA and/or PAEE levels before engaging in a structured exercise intervention predict weight loss, weight compensation, and changes in EI during that intervention. Importantly, habitual MVPA and/or PAEE (≥3 MET) at baseline showed a superior predictive value with regard to these outcome measures compared with steps per day and total AEE, suggesting that time spent and energy expended during MVPA rather than total activity-related EE before an exercise intervention targeting weight loss are protective against weight compensation. In this regard, habitual MVPA may be the preferable parameter compared with PAEE because of its easier, more economical, and (likely) more accurate assessment. Future studies are needed to elucidate the observed heterogeneous relationship between baseline habitual MVPA and/or PAEE levels and weight loss and compensation to develop individualized strategies to mitigate the detrimental compensatory increase in EI in response to an exercise-induced increase in EE in some individuals.**O**

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References

1. NCD Risk Factor Collaboration (NCD-RisC). Trends in adult body-mass index in 200 countries from 1975 to 2014: a pooled analysis of 1698 population-based measurement studies with 19·2 million participants. *Lancet* 2016;387:1377-1396.
2. Heymsfield SB, Wadden TA. Mechanisms, pathophysiology, and management of obesity. *N Engl J Med* 2017;376:254-266.
3. Garber CE, Blissmer B, Deschenes MR, et al. American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. *Med Sci Sports Exerc* 2011;43:1334-1359.
4. Swift DL, McGee JE, Earnest CP, Carlisle E, Nygard M, Johannsen NM. The effects of exercise and physical activity on weight loss and maintenance. *Prog Cardiovasc Dis* 2018;61:206-213.
5. Church TS, Martin CK, Thompson AM, Earnest CP, Mikus CR, Blair SN. Changes in weight, waist circumference and compensatory responses with different doses of exercise among sedentary, overweight postmenopausal women. *PLoS One* 2009;4:e4515. doi:10.1371/journal.pone.0004515
6. Martin CK, Johnson WD, Myers CA, et al. Effect of different doses of supervised exercise on food intake, metabolism, and non-exercise physical activity: The E-MECHANIC randomized controlled trial. *Am J Clin Nutr* 2019;110:583-592.
7. Thomas DM, Bouchard C, Church T, et al. Why do individuals not lose more weight from an exercise intervention at a defined dose? An energy balance analysis. *Obes Rev* 2012;13:835-847.
8. Blundell JE, Stubbs RJ, Hughes DA, Whybrow S, King NA. Cross talk between physical activity and appetite control: does physical activity stimulate appetite? *Proc Nutr Soc* 2003;62:651-661.
9. King NA, Hopkins M, Caudwell P, Stubbs RJ, Blundell JE. Individual variability following 12 weeks of supervised exercise: identification and characterization of compensation for exercise-induced weight loss. *Int J Obes (Lond)* 2008;32:177-184.
10. Westerterp KR. Exercise for weight loss. *Am J Clin Nutr* 2019;110:540-541.
11. Mayer J, Roy P, Mitra KP. Relation between caloric intake, body weight, and physical work: studies in an industrial male population in West Bengal. *Am J Clin Nutr* 1956;4:169-175.
12. Church T, Martin CK. The obesity epidemic: a consequence of reduced energy expenditure and the uncoupling of energy intake? *Obesity (Silver Spring)* 2018;26:14-16.
13. Myers CA, Johnson WD, Earnest CP, et al. Examination of mechanisms (E-MECHANIC) of exercise-induced weight compensation: study protocol for a randomized controlled trial. *Trials* 2014;15:212. doi:10.1186/1745-6215-15-212
14. Tudor-Locke C, Leonardi C, Johnson WD, Katzmarzyk PT, Church TS. Accelerometer steps/day translation of moderate-to-vigorous activity. *Prev Med* 2011;53:31-33.
15. West Suitor C, Kraak VI; Institute of Medicine. *Adequacy of Evidence for Physical Activity Guidelines Development: Workshop Summary*. Washington, DC: The National Academies Press; 2007.
16. American College of Sports Medicine. *ACSM's Guidelines for Exercise Testing and Prescription*. 9th ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins Health; 2014.
17. Thomas DM, Martin CK, Lettieri S, et al. Can a weight loss of one pound a week be achieved with a 3500-kcal deficit? Commentary on a commonly accepted rule. *Int J Obes (Lond)* 2013;37:1611-1613.
18. Heymsfield SB, Thomas D, Nguyen AM, et al. Voluntary weight loss: systematic review of early phase body composition changes. *Obes Rev* 2011;12:e348-e361. doi:10.1111/j.1467-789X.2010.00767.x
19. Thomas DM, Ciesla A, Levine JA, Stevens JG, Martin CK. A mathematical model of weight change with adaptation. *Math Biosci Eng* 2009;6:873-887.
20. Livingstone MBE, Black AE. Markers of the validity of reported energy intake. *J Nutr* 2003;133(suppl 3):895S-920S.
21. Schoeller DA. How accurate is self-reported dietary energy intake? *Nutr Rev* 1990; 48:373-379.
22. Martin CK, Coulon SM, Markward N, Greenway FL, Anton SD. Association between energy intake and viewing television, distractibility, and memory for advertisements. *Am J Clin Nutr* 2009;89:37-44.
23. Piercy KL, Troiano RP, Ballard RM, et al. The Physical Activity Guidelines for Americans. *JAMA* 2018;320:2020-2028.
24. Jones V, Bults R, de Wijk R, Widya I, Batista R, Hermens H. Experience with using the SenseWear BMS sensor system in the context of a health and wellbeing application. *Int J Telemed Appl* 2011:671040. doi:10.1155/2011/671040
25. Womble LG, Wadden TA, Chandler JM, Martin AR. Agreement between weekly vs. daily assessment of appetite. *Appetite* 2003;40:131-135.
26. Stunkard AJ, Messick S. *Eating Inventory Manual (The Psychological Corporation)*. San Antonio, TX: Harcourt Brace; 1988.
27. Anderson DA, Williamson DA, Duchmann EG, Gleaves DH, Barbin JM. Development and validation of a multifactorial treatment outcome measure for eating disorders. *Assessment* 1999;6:7-20.
28. Geiselman PJ, Anderson AM, Dowdy ML, West DB, Redmann SM, Smith SR. Reliability and validity of a macronutrient self-selection paradigm and a food preference questionnaire. *Physiol Behav* 1998;63:919-928.
29. White MA, Whisenhunt BL, Williamson DA, Greenway FL, Netemeyer RG. Development and validation of the food-craving inventory. *Obes Res* 2002;10:107-114.
30. Shook RP, Hand GA, Drenowatz C, et al. Low levels of physical activity are associated with dysregulation of energy intake and fat mass gain over 1 year. *Am J Clin Nutr* 2015;102:1332-1338.
31. Myers A, Gibbons C, Finlayson G, Blundell J. Associations among sedentary and active behaviours, body fat and appetite dysregulation: investigating the myth of physical inactivity and obesity. *Br J Sports Med* 2017;51:1540-1544.
32. Beaulieu K, Hopkins M, Long C, Blundell J, Finlayson G. High habitual physical activity improves acute energy compensation in nonobese adults. *Med Sci Sports Exerc* 2017;49:2268-2275.
33. Beaulieu K, Hopkins M, Blundell J, Finlayson G. Homeostatic and non-homeostatic appetite control along the spectrum of physical activity levels: an updated perspective. *Physiol Behav* 2018;192:23-29.
34. Beaulieu K, Hopkins M, Blundell J, Finlayson G. Does habitual physical activity increase the sensitivity of the appetite control system? A systematic review. *Sports Med* 2016;46:1897-1919.
35. Dalton M, Finlayson G, Esdaile E, King N. Appetite, satiety, and food reward in obese individuals: a behavioral phenotype approach. *Curr Nutr Rep* 2013;2:207-215.
36. Thompson D, Peacock OJ, Betts JA. Substitution and compensation erode the energy deficit from exercise interventions. *Med Sci Sports Exerc* 2014;46:423. doi:10.1249/MSS.0000000000000164
37. Thompson D, Karpe F, Lafontan M, Frayn K. Physical activity and exercise in the regulation of human adipose tissue physiology. *Physiol Rev* 2012;92:157-191.
38. Dhurandhar NV, Schoeller D, Brown AW, et al. Energy balance measurement: when something is not better than nothing. *Int J Obes (Lond)* 2015;39:1109-1113.
39. Dorling J, Broom DR, Burns SF, et al. Acute and chronic effects of exercise on appetite, energy intake, and appetite-related hormones: the modulating effect of adiposity, sex, and habitual physical activity. *Nutrients* 2018;10:E1140. doi:10.3390/nu10091140