HHS Public Access

Author manuscript

Exerc Sport Mov. Author manuscript; available in PMC 2023 August 03.

Published in final edited form as:

Exerc Sport Mov. 2023 ; 1(2): . doi:10.1249/esm.0000000000000004.

The Effects of an Acute Bout of Aerobic or Resistance Exercise on Nonexercise Physical Activity

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Abstract

Introduction/Purpose: A reduction in nonexercise physical activity (NEPA) after exercise may reduce the effectiveness of exercise interventions on weight loss in adults with overweight or obesity. Aerobic exercise (AEx) and resistance exercise (REx) may have different effects on NEPA. The purpose of this secondary analysis was to examine the effect of a single bout of AEx or REx on NEPA and sedentary behavior in inactive adults with overweight or obesity.

Methods: Adults with overweight or obesity (n = 24; 50% male; age, 34.5 ± 1.5 yr; body mass index, 28.5 ± 0.9 kg·m^{-2}) not meeting current physical activity guidelines completed a single 45-min bout of AEx, REx, or a sedentary control on different days in random order.

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After each condition, participants' NEPA was recorded for 84 h by accelerometer. Time spent sedentary and in light, moderate, and vigorous physical activity; steps; metabolic equivalent of task (MET)-hours; and sit-to-stand transitions were calculated using activity count data.

Results: No differences were observed in the percent of waking time spent sedentary and in light, moderate, and vigorous activity between conditions ($P > 0.05$). No differences were observed in steps, MET-hours, or sit-to-stand transitions between conditions ($P > 0.05$). NEPA responses were variable among individuals, with approximately half of participants reducing and half increasing NEPA over the 84 h after each exercise condition.

Conclusion: NEPA was not reduced after an acute bout of AEx or REx in a sample of inactive adults with overweight or obesity.

Keywords

Nonexercise Physical Activity; Aerobic Exercise; Resistance Exercise; Obesity

INTRODUCTION

Overweight and obesity continue to be pervasive health concerns worldwide and are associated with increased risk for various diseases and all-cause mortality (1-4). Exercise interventions are commonly utilized to elicit weight loss and improve health; however, many exercise-based interventions do not result in as much weight loss as expected (5-7). This may be due to compensatory behaviors that follow exercise, such as increasing caloric intake or decreasing physical activity (PA) (8).

PA is separated into two unique components: exercise and nonexercise physical activity (NEPA). Exercise is planned, structured, and repetitive movement with the express intent to improve health and fitness (9). NEPA is all movement that is not exercise and encompasses movements, such as activities of daily living, general locomotion, fidgeting, postural control, and sit-to-stand transitions (9). In some cases, an increase in exercise is related to a reduction in NEPA and a corresponding relative increase in sedentary time (10). This movement compensation, or reduction in NEPA after exercise, may offset the intended energy deficit from exercise interventions.

Systematic reviews of human studies primarily conclude that no change in NEPA occurs during exercise training (11-13); however, approximately one-quarter of the studies examined by two systematic reviews reported reductions in NEPA after exercise (12,13). These findings may be due in part to high levels of variability among participants. Multiple studies have reported that approximately half of participants exhibit reduced levels of NEPA after exercise (termed "NEPA compensators" herein), whereas the other half does not reduce NEPA (termed "NEPA noncompensators" herein) (10,14-16). When NEPA reductions occur after exercise, it seems to be influenced by several factors, including weight status, age, and sex (17-19).

In addition, most research has examined the influence of aerobic exercise (AEx) on NEPA, but few studies have examined changes in NEPA after resistance exercise (REx) (20). Some studies report that NEPA may increase after long-term REx (21,22), whereas NEPA

is more likely to remain unchanged or decrease after AEx (23,24). However, few trials have directly compared the influence of REx versus AEx on NEPA, and it is unknown if the different exercise modalities elicit distinct responses (20). Additional gaps exist for identifying individual factors associated with compensatory behavior. Identifying factors linked to compensatory reductions in NEPA that occur at the initiation of AEx or REx training protocols could help generate personalized exercise interventions for long-term weight management.

The primary aim of this secondary analysis was to investigate if a single bout of AEx or REx affects short-term NEPA in inactive adults with overweight or obesity. We hypothesized that a reduction in NEPA would occur after a single bout of AEx but not REx. In addition, we conducted an exploratory analysis to identify unique groups within the sample that decrease NEPA (NEPA compensators) and increase or do not change NEPA (NEPA noncompensators) and predicted parameters for each group.

METHODS

The complete methods and primary findings of the study are detailed elsewhere (25,26).

Participants

Participants were initially recruited using the following inclusion criteria: age, 21–55 yr; body mass index (BMI), 25–35 $kg \cdot m^{-2}$; weight stable (±5% in the past 6 months); and physically inactive (not meeting the current American College of Sports Medicine PA guidelines of either 150 $min \cdot wk^{-1}$ of moderate-intensity activity or twice per week whole-body resistance training, as evaluated by a modified International Physical Activity Questionnaire-Short Form (27)) but otherwise healthy. During the trial, eligibility criteria were expanded to include individuals with a normal BMI (<25 $kg \cdot m^{-2}$) with high levels of body fat (>30% for women and >22% for men) to increase enrollment. This resulted in an additional six participants with BMIs <25 $kg \cdot m^{-2}$.

Study Design

We conducted a secondary analysis of a randomized crossover trial with three conditions designed to investigate the effect of exercise modality on appetite and energy intake (25). Data were collected between Fall 2017 and Spring 2019. Participants completed baseline evaluations including weight, height, body composition by dual-energy x-ray absorptiometry (Hologic Discovery W, Bedford, MA, USA), and a 2-wk exercise familiarization period to support personalized prescriptions and learn the proper use of exercise equipment.

Block randomization, which was stratified by sex, was used to allocate participants to one of six session orders. Participants then completed a single bout of AEx, REx, or sedentary control (CON). AEx consisted of 45 min on a treadmill at 65%–70% of age-predicted heart rate maximum. REx consisted of one set to failure on 12 different exercises (leg press, leg extension, leg curl, hip abduction, hip adduction, chest press, overhead press, seated row, overheard pull-downs, assisted triceps dips, barbell calf raises, and dumbbell biceps curls). CON consisted of sitting quietly for 45 min. Each condition was separated by a washout period of 7 d for men and 1 month for women, to ensure testing occurred during

the follicular phase of the menstrual cycle (as identified by self-report). All interventions occurred at the University of Colorado Anschutz Medical Campus Clinical and Translational Research Center and were overseen by trained research staff. The study was approved by the Colorado Multiple Institutional Review Board (COMIRB No. 16-2697) and registered on ClinicalTrials.gov (NCT03143868). Participants provided written informed consent before participation.

Outcomes

Immediately after each study condition, participants were given an ActivPAL3™ (PAL Technologies, Glasgow, Scotland) triaxial accelerometer to wear on the anterior thigh, following manufacturer guidelines. Accelerometer recordings were collected for 84 h after each condition. Upon completion of the tracking period, ActivPAL data were downloaded using the PALBatch program (PAL Technologies) and cleaned using the CREA algorithm provided by the manufacturer.

Custom duration epochs of 60 s were exported from the PALBatch program. The activity score for each epoch in metabolic equivalent of task (MET)-seconds was converted into MET-minutes by manual division by 60. Each epoch was then classified into sedentary, light, moderate, and vigorous minutes based on the MET values of sedentary (≤1.5 METs), light (LPA; >1.5 to <3.0 METs), moderate (MPA; ≥3.0 to <6.0 METs), and vigorous (VPA; ≥6 METs) PA during the waking and nonexercise portions of the day (28). In combination with self-report logs, ActivPAL data were visually inspected to identify nonwear, time in bed, and exercise periods, which were manually removed before statistical analysis. To account for differences in wear time, time spent in each intensity category was expressed as a percent relative to total waking nonexercise wear time. The total number of sit-to-stand transitions, steps, and MET-hours recorded by the accelerometer was also included to assist in modeling categorical profiles for the exploratory analysis.

Statistical Analyses

Data were analyzed using Stata v17.0 (StataCorp LLC, College Station, TX, USA). General linear mixed-effects models were used for the primary analysis on all outcomes on the continuous measurement scale. Generalized linear mixed-effects Poisson models were used for count outcome variables. Random intercepts were used at the participant level to account for the nested data structure. If the assumption of no dispersion was violated, a mixed-effects negative binomial model was used to account for any dispersion by including an additional correction parameter. Significance was determined based on an α level of 0.05.

Exploratory Analysis

For the exploratory analysis, descriptive statistics and change scores were created for each participant by subtracting outcome variables relative to CON. Only participants with complete 84-h tracking periods in one or both exercise and CON conditions were used. Latent profile analyses identified latent groups in each condition. Latent profile analysis is a statistical approach that attempts to define profiles, or classes, based on shared characteristics. Two latent profiles of NEPA compensators, characterized by increased sedentary time and reduced NEPA, and NEPA noncompensators, characterized by reduced

sedentary time and increased NEPA, were hypothesized. Additional latent groups were tested to determine if they improved model fit. Akaike's information criterion and Bayesian information criterion scores were used to identify the best-fitting model.

RESULTS

Participant Characteristics

Twenty-four participants (50% men) completed this study. Baseline characteristics are presented in Table 1.

Data Analyzed

Data were available for 22 of 24 (91.6%) periods after both AEx and REx and 21 of 24 (87.5%) periods after CON. Data loss was due to hardware malfunctions and participant noncompliance with the wear protocol. Initially, the time and condition interaction were evaluated, but no day-to-day changes were observed (all $P > 0.05$; Fig. 1).

Sedentary Behavior and NEPA

There were no significant differences in the percentage of waking time spent in sedentary behavior, LPA, or MPA between AEx and CON, REx and CON, and AEx and REx (all $P > 0.05$). After removing exercise, no recorded activity ≥6.0 METs (VPA) occurred in any participant for any condition. There were no differences in total MET-hours, sit-to-stand transitions, or step counts between conditions ($P > 0.05$; Table 2).

Exploratory Analysis

The exploratory analysis was limited to participants who had tracking for the full 84 h for the control condition and at least one exercise condition. Data from 18 and 15 participants for AEx and REx, respectively, were used in the exploratory analysis. Latent profile analyses indicated two unique profiles after the AEx condition and two profiles after the REx condition (Table 3). Additional models testing for three and four latent profiles were compared, but the two-profile model had the smallest Akaike's information criterion and Bayesian information criterion values and was considered the best fit. After AEx, profile 1 displayed lower MET-hours, fewer steps, and fewer sit-to-stand transitions. This group was labeled NEPA compensators. Profile 2 was labeled NEPA noncompensators and primarily displayed minimal changes in each outcome measure with AEx compared with sedentary behavior. After REx, profile 1 displayed lower MET-hours, fewer steps, and fewer sit-to-stand transitions and was labeled NEPA compensators. Profile 2 was labeled NEPA noncompensators and displayed increased NEPA and reduced sedentary time. BMI, age, and sex did not significantly predict the probability of class membership within groups ($P > 0.05$ for all predictors and conditions). Individual responses to each condition are shown in Figure 2. Of the 12 subjects with complete 84-h tracking after both exercise conditions, compared with the CON condition, 5 subjects (41.6%) reduced MET-hours after each condition, 2 subjects (16.7%) increased MET-hours after each condition, 3 subjects (25.0%) reduced MET-hours after AEx but not REx, and 2 subjects (16.7%) reduced MET-hours after REx but not AEx. The outcomes associated with the best model fit are shown in Table 3.

DISCUSSION

This study examined the influence of an acute bout of AEx or REx on short-term NEPA in a sample of inactive adults with overweight or obesity. Contrary to our hypothesis, there were no differences in NEPA in response to AEx, REx, or CON. Although the primary outcomes do not show compensation in NEPA on a group level, exploratory analyses predicted two unique profiles, NEPA compensators, and NEPA noncompensators, after both acute AEx and REx. Interestingly, the REx NEPA noncompensator profile was uniquely characterized by increased NEPA and reduced sedentary time. In addition, change scores in NEPA compared with CON for each participant showed high variability, demonstrating that some participants reduced NEPA after both AEx and REx, whereas others reduced NEPA after one exercise condition but not the other.

Previous studies evaluating changes in NEPA after acute AEx and REx are limited. In a similarly designed crossover study, Cadieux et al. (29) reported that NEPA does not change 10 or 34 h after AEx and REx of equivalent energy expenditure. Similarly, Alahmadi et al. (30) reported that NEPA remained unchanged in overweight men for 2 d after a single AEx session and increased on the third day. Our statistical model initially included condition- and day-interaction terms, but no significant interactions were observed. Thus, in contrast to Alahmadi et al. (30), no increase in NEPA was observed in the days after exercise in this sample. Therefore, the day-by-day-interaction term was removed to make the model more parsimonious. The lack of evidence indicating NEPA changes after exercise in this sample is consistent with existing systematic reviews regarding similar exercise intensities and participant characteristics (11,20).

However, exercise dose may influence PA compensation. For example, a 24-wk AEx intervention demonstrated that objectively measured total energy expenditure was reduced to a greater degree in response to a higher dose of AEx than a moderate dose of AEx (7). In other words, although the higher dose of AEx resulted in weight loss over 24 wk, it was less than expected (7). Energy intake calculated by the intake-balance method remained unchanged, indicating the potential for NEPA to decrease the effectiveness of weight loss during higher-volume AEx interventions (7,31). Furthermore, other studies show that the intensity of AEx has no effect on PA compensation for both low versus moderate and moderate versus vigorous interventions (32,33). It is unknown if differing volumes and intensities of REx will have different effects on NEPA.

Changes in NEPA after REx also seem to vary across studies. Drenowatz et al. (24) conducted a 16-wk intervention and found that moderate-vigorous NEPA increased by approximately 216 $kcal \cdot d^{-1}$ on nonexercise days in the REx group. In comparison, no change in moderate-vigorous NEPA occurred on the nonexercise days in the AEx condition (24). Similarly, both Hunter et al. (34) and Halliday et al. (21) previously reported increased NEPA after longer-term resistance training. However, other studies have reported that NEPA does not change after resistance training (6,35). The current study does not reconcile these separate findings. Rather, the results suggest that if an individual reduces NEPA after exercise, the amount that NEPA is reduced may be lower after REx as compared with AEx, as seen in the exploratory analysis.

Although the group findings did not yield statistically significant results, the exploratory analysis indicates two unique profiles after AEx and REx conditions characterized by differences in NEPA. This split into NEPA compensators and NEPA noncompensators is similar to the categorization reported in other studies (10,14-16). The continued findings of compensating groups throughout multiple studies warrant further investigation. Sedentary time was not different between the NEPA compensators and NEPA noncompensators after the AEx conditions. However, MET-hours, sit-to-stand transitions, and total steps decreased in the NEPA compensator group. Interestingly, the REx NEPA noncompensator profile was characterized by increased NEPA and reduced sedentary time. These findings indicate that a reduction in the intensity of daily movement rather than a change in the allocation of time spent in sedentary behavior can follow AEx, whereas REx may lead to both reduced sedentary time and increased NEPA in those classified as noncompensators. However, these findings need to be interpreted with caution because of the exploratory nature of the analysis.

Although age, sex, and BMI were not associated with compensator status in the present study, these factors and others, such as cardiorespiratory fitness, should be directly examined in future larger-scale trials. When NEPA compensation does occur, it is most often associated with sedentary older adults with overweight/obesity (11). Larger trials specifically designed to identify common factors of NEPA compensators may be more successful in identifying characteristics and mechanisms associated with NEPA compensation. If factors identifying those likely to reduce NEPA during exercise interventions can be identified, personalized NEPA prescriptions that simultaneously target exercise and NEPA may increase the effectiveness of exercise interventions for weight management.

Variance in NEPA compensation may be explained by individual physiological and psychological responses to exercise, as identified by Gray et al. (36). Delayed onset muscle soreness (DOMS) can occur after novel exercise and last several days after starting an exercise routine (37). DOMS was not assessed throughout our intervention, and individual responses may have varied. The inclusion of exercise familiarization sessions for both AEx and REx conditions and the moderate-intensity protocol for each condition was selected to avoid the compounding effects of DOMS as much as possible. Other potential mechanisms identified by Gray et al. (36) for reduced movement in the hours and days after an exercise bout include doing so as a reward for exercise, fatigue, and time management. Additional psychological responses, such as increased compensatory health beliefs and smaller reductions in disinhibition, are more likely in NEPA compensators (38). No qualitative assessments were used during the present study, and it is unknown how these individual factors may have played a role in changing NEPA. In a weight management setting, if NEPA remains the same and weight loss attempts are blunted, then any compensatory changes disrupting the intended negative energy balance are more likely to be caused by increased energy intake.

Notably, the lack of group compensation across conditions in our sample may be due to the sample population. Based on the CON condition, participants spent ~3% of their day in MVPA and ~74% sedentary. It may be hard to become more sedentary, thus

limiting the opportunity to compensate by decreasing NEPA. Future studies examining NEPA compensation should include robust measurements of NEPA and energy intake to identify potential mechanisms that offset negative energy balance. In addition, future studies could identify and examine if the individuals that change NEPA at the start of a weight-loss-focused exercise intervention maintain that change throughout a long-term exercise intervention.

Limitations

The strengths of this study include a randomized crossover design and an objective measure of NEPA. However, this study is not without limitations. First, energy expenditure and exercise intensity were not matched between AEx and REx conditions, nor was energy expenditure directly measured throughout the study. Instead, we opted for a practical approach of matching exercise conditions by equivalent time to reflect standard exercise recommendations. Second, we acknowledge that the impact of our findings is limited by not having a record of energy intake for the 3 d after the exercise. Changes in energy intake and appetite hormone regulation may be a more likely mechanism leading to less than expected weight loss than NEPA compensation (39), and recording food intake would provide more robust insight into the compensatory behaviors after exercise. However, self-reported dietary intake data are highly prone to misreporting and often unrelated to actual energy intake (40). The acute energy intake data for this trial are published elsewhere (25,26). Third, waking wear time was identified from the first and last instance of steps registered on the ActivPAL device. Sedentary time may be underestimated by missing time spent lying in bed before and after waking. However, this limitation occurs in each condition and is likely to have an equal effect on all conditions. Lastly, this study was a secondary analysis of previously collected data, and as such, the results herein should be interpreted with caution because of the small sample size and evaluation of nonprimary outcomes.

Conclusions

The findings of this trial suggest that NEPA compensation by reducing NEPA after exercise does not consistently occur after acute bouts of AEx or REx in adults with overweight and obesity. However, subsets of individuals reduce or increase NEPA in response to AEx and REx. PA prescriptions that account for individual variability by including exercise and NEPA recommendations may provide more effective weight loss outcomes. More research is needed to identify common factors that associate and differentiate NEPA compensators and NEPA noncompensators.

ACKNOWLEDGMENTS

We acknowledge and thank the University of Colorado's Clinical and Translational Research Center for their support in conducting this trial.

The manuscript content is the authors' sole responsibility and do not necessarily represent official NIH views. The contents and results of the present study do not represent the views of the US Department of Veterans Affairs, or the US government. The results of the present study do not constitute endorsement by the American College of Sports Medicine.

CONFLICTS OF INTEREST AND SOURCE OF FUNDING

The authors report no conflicts of interest. This work was supported by the National Institutes of Health (NIH) grants, such as NIH/National Center for Advancing Translational Sciences under award numbers UL1TR002535, 1UL01TR002538, KL2TR002539, and NIDDK P30DK048520, and NIH/National Institute of Diabetes and Digestive and Kidney Diseases grants T32DK07658 and T32DK007446. This work was also supported by a pilot grant award from the University of Colorado's Center for Women's Health Research. This trial was preregistered at ClinicalTrials.gov (NCT03143868). Drs. Melanson and Cornier are supported by resources from Geriatric Research, Education, and the Clinical Center at the Denver VA Medical Center.

REFERENCES

1. Chooi YC, Ding C, Magkos F. The epidemiology of obesity. Metabolism. 2019;92:6–10. doi: 10.1016/j.metabol.2018.09.005. [PubMed: 30253139]
2. Prospective Studies Collaboration, Whitlock G, Lewington S, et al. Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. Lancet. 2009;373(9669):1083–96. doi: 10.1016/S0140-6736(09)60318-4. [PubMed: 19299006]
3. Michaud DS, Giovannucci E, Willett WC, et al. Physical activity, obesity, height, and the risk of pancreatic cancer. JAMA. 2001;286(8):921–9. doi: 10.1001/jama.286.8.921. [PubMed: 11509056]
4. Apovian CM. Obesity: definition, comorbidities, causes, and burden. Am J Manag Care. 2016;22(7 Suppl):s176–85. [PubMed: 27356115]
5. Ross R, Janssen I. Physical activity, total and regional obesity: dose– response considerations. Med Sci Sports Exerc. 2001;33(6 Suppl):S521–7; discussion S528–9. doi: 10.1097/00005768-200106001-00023. [PubMed: 11427779]
6. Church TS, Martin CK, Thompson AM, et al. Changes in weight, waist circumference and compensatory responses with different doses of exercise among sedentary, overweight postmenopausal women. PLoS One. 2009;4(2):e4515. doi: 10.1371/journal.pone.0004515. [PubMed: 19223984]
7. Broskey NT, Martin CK, Burton JH, et al. Effect of aerobic exercise-induced weight loss on the components of daily energy expenditure. Med Sci Sports Exerc. 2021;53(10):2164–72. doi: 10.1249/MSS.0000000000002689. [PubMed: 34519717]
8. Melanson EL, Keadle SK, Donnelly JE, et al. Resistance to exercise-induced weight loss: compensatory behavioral adaptations. Med Sci Sports Exerc. 2013;45(8):1600–9. doi: 10.1249/MSS.0b013e31828ba942. [PubMed: 23470300]
9. 2018 Physical Activity Guidelines Advisory Committee. 2018 Physical Activity Guidelines Advisory Committee Scientific Report. Washington (DC): U.S. Department of Health and Human Services; 2018. 779 p.
10. Kozey-Keadle S, Staudenmayer J, Libertine A, et al. Changes in sedentary time and physical activity in response to an exercise training and/or lifestyle intervention. J Phys Act Health. 2014;11(7):1324–33. doi: 10.1123/jpah.2012-0340. [PubMed: 24184493]
11. Washburn RA, Lambourne K, Szabo AN, et al. Does increased prescribed exercise alter non-exercise physical activity/energy expenditure in healthy adults? A systematic review. Clin Obes. 2014;4(1):1–20. doi: 10.1111/cob.12040. [PubMed: 25425128]
12. Swelam BA, Verswijveren SJJM, Salmon J, et al. Exploring activity compensation amongst youth and adults: a systematic review. Int J Behav Nutr Phys Act. 2022;19(1):25. doi: 10.1186/s12966-022-01264-6. [PubMed: 35279187]
13. Silva AM, Judice PB, Carraca EV, et al. What is the effect of diet and/or exercise interventions on behavioural compensation in non-exercise physical activity and related energy expenditure of free-living adults? A systematic review. Br J Nutr. 2018;119(12):1327–45. doi: 10.1017/S000711451800096X. [PubMed: 29845903]
14. Manthou E, Gill JM, Wright A, et al. Behavioral compensatory adjustments to exercise training in overweight women. Med Sci Sports Exerc. 2010;42(6):1121–8. doi: 10.1249/MSS.0b013e3181c524b7. [PubMed: 19997033]
15. Di Blasio A, Ripari P, Bucci I, et al. Walking training in postmenopause: effects on both spontaneous physical activity and training-induced body adaptations. Menopause. 2012;19(1):23–32. doi: 10.1097/gme.0b013e318223e6b3. [PubMed: 21993080]

16. Willis EA, Herrmann SD, Honas JJ, et al. Nonexercise energy expenditure and physical activity in the Midwest Exercise Trial 2. Med Sci Sports Exerc. 2014;46(12):2286–94. doi: 10.1249/MSS.0000000000000354. [PubMed: 24694746]
17. Morio B, Montaurier C, Pickering G, et al. Effects of 14 weeks of progressive endurance training on energy expenditure in elderly people. Br J Nutr. 1998;80(6):511–9. doi: 10.1017/s0007114598001603. [PubMed: 10211049]
18. Meijer EP, Westerterp KR, Verstappen FT. Effect of exercise training on total daily physical activity in elderly humans. Eur J Appl Physiol Occup Physiol. 1999;80(1):16–21. doi: 10.1007/s004210050552. [PubMed: 10367718]
19. Riou ME, Jomphe-Tremblay S, Lamothe G, et al. Predictors of energy compensation during exercise interventions: a systematic review. Nutrients. 2015;7(5):3677–704. doi: 10.3390/nu7053677. [PubMed: 25988763]
20. Fedewa MV, Hathaway ED, Williams TD, et al. Effect of exercise training on non-exercise physical activity: a systematic review and meta-analysis of randomized controlled trials. Sports Med. 2017;47(6):1171–82. doi: 10.1007/s40279-016-0649-z. [PubMed: 27873191]
21. Halliday TM, Savla J, Marinik EL, et al. Resistance training is associated with spontaneous changes in aerobic physical activity but not overall diet quality in adults with prediabetes. Physiol Behav. 2017;177:49–56. doi: 10.1016/j.physbeh.2017.04.013. [PubMed: 28414072]
22. Hunter GR, Wetzstein CJ, Fields DA, et al. Resistance training increases total energy expenditure and free-living physical activity in older adults. J Appl Physiol (1985). 2000;89(3):977–84. doi: 10.1152/jappl.2000.89.3.977. [PubMed: 10956341]
23. Paravidino VB, Mediano MFF, Silva ICM, et al. Effect of physical exercise on spontaneous physical activity energy expenditure and energy intake in overweight adults (the EFECT study): a study protocol for a randomized controlled trial. Trials. 2018;19(1):167. doi: 10.1186/s13063-018-2445-6. [PubMed: 29514690]
24. Drenowatz C, Grieve GL, DeMello MM. Change in energy expenditure and physical activity in response to aerobic and resistance exercise programs. Springerplus. 2015;4(1):798. doi: 10.1186/s40064-015-1594-2. [PubMed: 26702387]
25. Halliday TM, White MH, Hild AK, et al. Appetite and energy intake regulation in response to acute exercise. Med Sci Sports Exerc. 2021;53(10):2173–81. doi: 10.1249/MSS.0000000000002678. [PubMed: 33831896]
26. Tobin SY, Cornier MA, White MH, et al. The effects of acute exercise on appetite and energy intake in men and women. Physiol Behav. 2021;241:113562. doi: 10.1016/j.physbeh.2021.113562. [PubMed: 34516956]
27. Booth M. Assessment of physical activity: an international perspective. Res Q Exerc Sport. 2000;71(2 Suppl):S114–20. doi: 10.1080/02701367.2000.11082794. [PubMed: 10925833]
28. Ainsworth BE, Haskell WL, Herrmann SD, et al. 2011 compendium of physical activities: a second update of codes and MET values. Med Sci Sports Exerc. 2011;43(8):1575–81. doi: 10.1249/MSS.0b013e31821ece12. [PubMed: 21681120]
29. Cadieux S, McNeil J, Lapierre MP, et al. Resistance and aerobic exercises do not affect post-exercise energy compensation in normal weight men and women. Physiol Behav. 2014;130:113–9. doi: 10.1016/j.physbeh.2014.03.031. [PubMed: 24726390]
30. Alahmadi MA, Hills AP, King NA, et al. Exercise intensity influences nonexercise activity thermogenesis in overweight and obese adults. Med Sci Sports Exerc. 2011;43(4):624–31. doi: 10.1249/MSS.0b013e3181f7a0cb. [PubMed: 21412111]
31. Heymsfield SB, Peterson CM, Thomas DM, et al. Establishing energy requirements for body weight maintenance: validation of an intake-balance method. BMC Res Notes. 2017;10(1):220. doi: 10.1186/s13104-017-2546-4. [PubMed: 28651559]
32. Riou ME, Jomphe-Tremblay S, Lamothe G, et al. Energy compensation following a supervised exercise intervention in women living with overweight/obesity is accompanied by an early and sustained decrease in non-structured physical activity. Front Physiol. 2019;10:1048. doi: 10.3389/fphys.2019.01048. [PubMed: 31507431]

33. Paravidino VB, Mediano MFF, Hoffman DJ, et al. Effect of exercise intensity on spontaneous physical activity energy expenditure in overweight boys: a crossover study. PLoS One. 2016;11(1):e0147141. doi: 10.1371/journal.pone.0147141. [PubMed: 26771742]

34. Hunter GR, Fisher G, Neumeier WH, et al. Exercise training and energy expenditure following weight loss. Med Sci Sports Exerc. 2015;47(9):1950–7. doi: 10.1249/MSS.0000000000000622. [PubMed: 25606816]

35. Laussen JC, Chalé A, Hau C, et al. Does physical activity change after progressive resistance exercise in functionally limited older adults? J Am Geriatr Soc. 2015;63(2):392–3. doi: 10.1111/jgs.13270. [PubMed: 25688616]

36. Gray P, Murphy M, Gallagher A, et al. A qualitative investigation of physical activity compensation among older adults. Br J Health Psychol. 2018;23(1):208–24. doi: 10.1111/bjhp.12282. [PubMed: 29171704]

37. Cheung K, Hume P, Maxwell L. Delayed onset muscle soreness: treatment strategies and performance factors. Sports Med. 2003;33(2):145–64. doi: 10.2165/00007256-200333020-00005. [PubMed: 12617692]

38. 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(3):583–92. doi: 10.1093/ajcn/nqz054. [PubMed: 31172175]

39. Flack KD, Hays HM, Moreland J, et al. Exercise for weight loss: further evaluating energy compensation with exercise. Med Sci Sports Exerc. 2020;52(11):2466–75. doi: 10.1249/MSS.0000000000002376. [PubMed: 33064415]

40. Hill RJ, Davies PS. The validity of self-reported energy intake as determined using the doubly labelled water technique. Br J Nutr. 2001;85(4):415–30. doi: 10.1079/bjn2000281. [PubMed: 11348556]

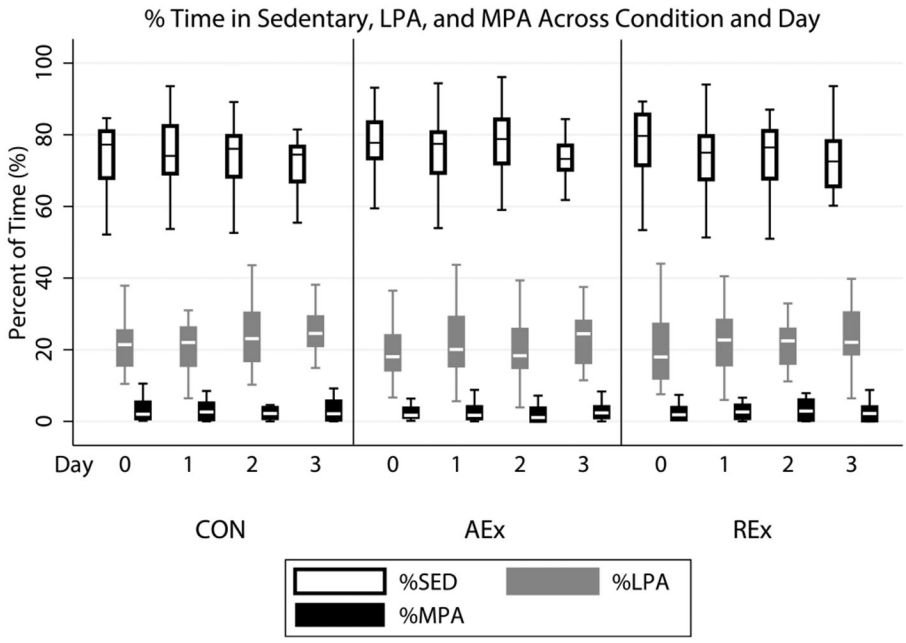

Figure 1.

Time spent sedentary (SED) and in light physical activity (LPA) and moderate physical activity (MPA) after exercise bouts. Comparison of percent waking, nonexercise time spent in SED (≤1.5 metabolic equivalents of task (METs)), LPA (>1.5 to <3.0 METs), and MPA (≥3.0 to <6.0 METs) between conditions. Box components indicate median (middle bar of box), interquartile range (outer edges of box), and most extreme values that are not outliers (ends of lines extending from box). AEx, aerobic exercise condition; CON, sedentary control condition; REx, resistance exercise condition.

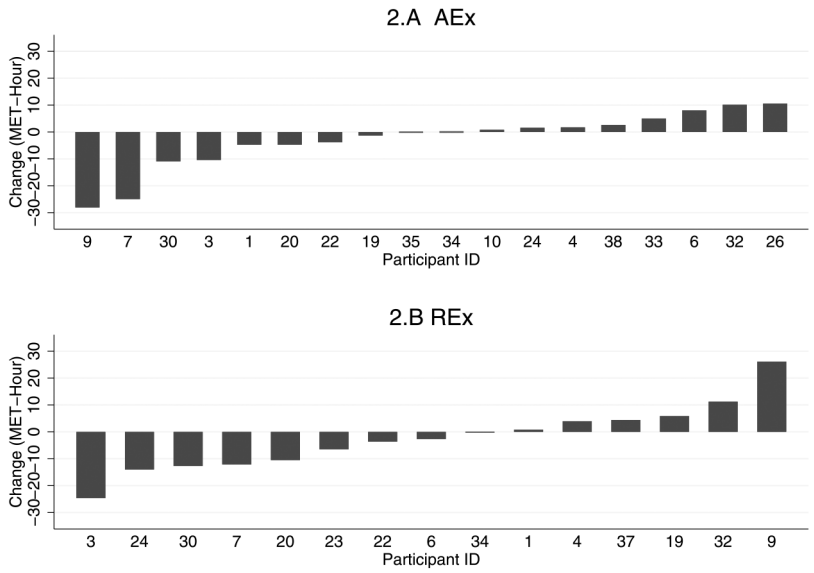

Figure 2.

Changes in metabolic equivalent of task (MET)-hours over 84 h after aerobic exercise (AEx) and resistance exercise (REx) compared with the control (CON) condition. A, Change in MET-hours over 84 h after AEx compared with CON. Each bar represents an individual participant's change compared with the MET-hours they obtained during the CON condition. B, Change in MET-hours over 84 h after REx compared with CON. Each bar represents an individual participant's change compared with the MET-hours they obtained during the CON condition.

Table 1

Participant Characteristics.

	Total Sample (*n* = 24)	**Women (*n* = 12)**	**Men (*n* = 12)**	***P***
Age, yr	34.6 ± 7.4	36.8 ± 7.7	31.1 ± 5.3	0.14
BMI, $kg \cdot m^{-2}$	28.5 ± 4.7	29.0 ± 5.1	28.1 ± 4.4	0.64
Body fat, %	35.1 ± 8.4	40.3 ± 7.2	30.3 ± 6.5	0.01

Data are presented as mean ± standard deviation.

BMI, body mass index.

Table 2

Average Daily Outcomes of Time Spent Sedentary and Engaging in NEPA after Exercise Bouts.

	AEx[a]	REx[a]	CON[a]	AEx vs CON[b]	REx vs CON[b]	AEx vs REx[b]
Time sedentary (≤1.5 METs), %	75.2 ± 11.0	74.4 ± 10.2	73.7 ± 8.9	*P* = 0.22 (95% CI, −0.01 to 0.04)	*P* = 0.61 (95% CI, −0.02 to 0.33)	*P* = 0.48 (95% CI, −0.02 to 0.04)
Time in LPA (>1.5 to < 3.0 METs), %	21.8 ± 9.8	22.5 ± 9.3	23.2 ± 8.3	*P* = 0.13 (95% CI, −0.04 to 0.01)	*P* = 0.51 (95% CI, −0.03 to 0.02)	*P* = 0.41 (95% CI, −0.03 to 0.01)
Time in MPA (≥3.0 to 6.0 METs), %	3.0 ± 3.7	3.1 ± 3.2	3.1 ± 2.8	*P* = 0.85 (95% CI, −0.01 to 0.01)	*P* = 0.82 (95% CI, −0.01 to 0.01)	*P* = 0.97 (95% CI, −0.01 to 0.01)
MET-hours	20.0 ± 4.8	20.0 ± 4.9	20.4 ± 5.2	*P* = 0.61 (95% CI, −1.88 to 1.10)	*P* = 0.56 (95% CI, −1.96 to 1.06)	*P* = 0.94 (95% CI, −1.43 to 1.54)
Sit-to-stand transitions	42.3 ± 16.7	42.7 ± 15.1	46.6 ± 17.0	*P* = 0.06 (95% CI, −9.13 to 0.20)	*P* = 0.09 (95% CI, −8.78 to 0.65)	*P* = 0.86 (95% CI, −5.05 to 4.24)
Steps	7043 ± 4538	7492 ± 4388	7506 ± 3822	*P* = 0.48 (95% CI, −1521 to 722)	*P* = 0.91 (95% CI, −1198 to 1068)	*P* = 0.34 (95% CI, −1451 to 783)

[a]Data are presented as mean ± standard deviation.

[b]Data presented as *P* value and 95% confidence intervals (CI).

AEx, aerobic exercise condition; CON, sedentary control condition; LPA, light physical activity; MET, metabolic equivalent of task; MPA, moderate physical activity; REx, resistance exercise condition.

Table 3

Latent Group Characteristics During 84 h after Each Exercise Condition.

	After AEx		After REx	
	NEPA Compensators	**NEPA Noncompensators**	**NEPA Compensators**	**NEPA Noncompensators**
Probability of class membership, %	27.8	72.2	67.6	32.3
Change in % sedentary time[a], %	0.2 ± 2.0	1.3 ± 1.2	1.3 ± 1.4	−4.2 ± 1.4
Change in MET-hours	−13.9 ± 3.4	1.6 ± 2.1	−8.0 ± 2.8	9.6 ± 4.8
Sit-to-stand transitions	−73.6 ± 10.4	5.3 ± 6.4	−29.9 ± 13.3	13.6 ± 14.8
Steps	−6235.8 ± 2146.3	−512.1 ± 1328.6	−4025.5 ± 2792.7	11,720.2 ± 5746.6

[a]Waking nonexercise time spent in sedentary behavior (<1.5 METs).

Data are presented as mean ± standard deviation.

AEx, aerobic exercise condition; MET, metabolic equivalent of task; NEPA, nonexercise physical activity; REx, resistance exercise condition.