COMPENSATORY RESPONSES IN NON-EXERCISE ACTIVITY THERMOGENESIS AND REPORTED ENERGY INTAKE IN COLLEGE-AGE FEMALES DURING A MODERATE-CONTINUOUS OR SPRINT-INTERVAL EXERCISE TRAINING PROGRAM

by

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ABSTRACT

The influence of exercise intensity while participating in a structured exercise program on a) energy expenditure outside of structured exercise (non-exercise activity thermogenesis (NEAT)), b) reported energy intake (REI), and c) cardiometabolic biomarkers have not been previously examined. This study aimed to: 1) examine how exercise intensity (continuous moderate-intensity exercise versus high-intensity interval training) influences compensatory responses (changes in NEAT and REI) to an exercise training program in overweight/obese college-age females, 2) explore whether compensatory changes in NEAT and EI explain inter-individual differences in cardiometabolic responses to moderate- and high-intensity exercise training, and 3) examine the effect of psychological constructs and view of exercise as commitment versus progress on compensatory changes in NEAT and EI. Overweight/obese college females were randomly assigned to continuous moderate-intensity cycling (MOD-C) or vigorous sprint-interval cycling (VIG-SIC) for 6 weeks of exercise training. Participants in the VIG-SIC group performed 5-7 repeated bouts of 30-second sprints at maximal effort, followed by 4 minutes of active recovery. Females in the MOD-C group completed 20-30 minutes of

moderate intensity with duration matched to maintain equal energy expenditure between groups. NEAT was measured with the Actiheart physical activity monitor, REI via the Automated Self-Administered 24-hour Recall (ASA24), and cardiometabolic biomarkers were determined using standard clinical procedures. Exercise views were recorded immediately following exercise training sessions via the wrist-worn PRO-Diary monitor.

No between or within group differences in NEAT were apparent on exercise or non-exercise days. Similarly, no between or within group differences were found in REI changes. When groups were combined, participants maintained NEAT ($p=0.99$) and decreased REI ($p=0.01$) throughout the study. Significant associations were found between NEAT responses and CRP and GLUC changes and while these associations were small in magnitude for GLUC, a 100 kcal/day in NEAT was associated with a 0.42 mg/dL decrease in CRP. Exercise views were not shown to be associated with compensatory responses, but higher self-control scores were associated with increased REI during the exercise training intervention. Future exercise training interventions should educate participants beginning a structured exercise program about potential adverse compensatory responses.

INDEX WORDS: compensation, non-exercise activity thermogenesis, energy intake, exercise training, females

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DEDICATION

Throughout this process, I have learned very few questions have a simple answer, instead answers usually involve an exasperated sigh followed by "it depends" or "it's complicated." I am confident that one answer remains the same, and actually I've become more certain of during this period, Jesus will always be the answer. I dedicate this dissertation, and my life, to my Heavenly Father, may He be glorified in all that I have done and will ever do. *Therefore, since we are surrounded by such a great cloud of witnesses, let us throw off everything that hinders and the sin that so easily entangles, and let us run with perseverance the race marked out for us.* (Hebrews 12:1)

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CHAPTER 1

INTRODUCTION

1.1 Significance

Traditionally researchers have focused on group mean changes in health parameters in response to exercise, but only reporting group changes leads to a masking of the heterogeneity in responses by individual participants. Recent studies have highlighted the less than predicted weight loss experienced by a subset of participants engaging in exercise training.[1-3] Weight changes are not the only health outcome that reveals heterogeneous results. Inter-individual variability also exists in cardiometabolic responses to exercise with adverse responses ranging from 8.3-13.3%.[4] These studies have shown that not all individuals adapt as favorably to exercise as predicted and more recently, focus has shifted to understanding the inter-individual variability in these responses to exercise. While genetic factors undoubtedly contribute to response variations,[5] behavioral changes also likely play a role. Potential behavioral responses to exercise include changes in diet or energy intake (EI) and non-exercise activity thermogenesis (NEAT) and several recent studies have focused on quantifying these behaviors.[2,3] Previous studies have compared mean changes in EI and NEAT in two groups based on achievement of expected weight loss ("responders") or non-achievement ("nonresponders"). For example, in the Midwest Trial 2, responders increased NEAT (116 ± 456 kcal/day) and average EI was 2696 ± 603 kcal/day while nonresponders decreased NEAT (-238 ± 502 kcal/day) and average EI was of 3076 ± 967 kcal/day.[6] Further, in a recent study composed of overweight/obese women, inter-individual variability in EI between an exercise and control condition ranged from -234.3 to

278.5 kcals/day highlighting the potential differences within individuals on exercise versus non-exercise days.[7] Future research should highlight not only the inter-individual variations in weight change, but also in EI and NEAT.

The extent that exercise characteristics (i.e., type, intensity, duration, etc.) influence the magnitude of non-exercise behavioral responses has yet to be fully explored. The effect of exercise dose on behavioral responses has been assessed by comparing actual versus predicted weight loss in different exercise energy expenditure groups (4 kcal/kg/wk, 8 kcal/kg/wk, or 12 kcal/kg/wk). Church et al.[1] reported only those in the 12 kcal/kg/wk group experienced less than predicted weight loss, implying changes in NEAT and/or EI occurred in this higher dose group. Accordingly, as different doses of exercise appear to have different effects on behavioral responses, it seems plausible that exercise intensity might also play a role, but mixed results have been reported. Among overweight/obese males (27 ± 3 y), an acute bout of either moderate- or high-intensity exercise did not alter NEAT on the exercise day or in the first two days after the exercise session, but NEAT was increased on the third day in the high intensity group by 25% compared to the exercise day.[8] Conversely, in post-menopausal women, a reduction in NEAT was found in both moderate- and vigorous-intensity exercise groups and the reduction was greater in women performing vigorous-intensity exercise.[9] Additional research is needed to further assess the effects of exercise intensity on NEAT.

While the effects of intensity on changes in EI have been studied, exercise training groups differed in exercise energy expenditure. No differences were found in EI between fifteen women randomized to either a four-week low-intensity exercise program (45 minutes at mean exercise heart rate of 132 beats per minute) or a four-week high-intensity program (45 minutes at mean exercise heart rate of 163 beats per minute) although exercise energy expenditure was not

equivalent.[10] Nor was a difference in EI reported between 22 obese females randomized to either a continuous group (30 minutes/once a day/3 times per week at 60-75% maximal aerobic capacity) or an intermittent group (15 minutes/twice a day/5 days per week at 50-65% heart rate reserve) for 18 months.[11] Again, exercise energy expenditure was not equivalent between groups highlighting the need for future studies to match exercise energy expenditure to determine if intensity influences EI responses.

The extent to which behavioral responses may explain inter-individual variability in cardiometabolic responses to exercise also warrants further attention. Cardiometabolic variables including high-sensitivity C-reactive protein (CRP), fasting plasma glucose (GLUC), high-density lipoprotein (HDL), fasting insulin (INS), low-density lipoprotein (LDL), systolic blood pressure (SBP), triglycerides (TG), and waist circumference (WC) are of significant health importance as they are risk factors for chronic diseases such as cardiovascular disease and type 2 diabetes. As previously mentioned, 8.3-13.3% of participants engaging in exercise experience an adverse cardiometabolic response in one or more risk factors and a great need exists to identify the predictors of such responses.[4] There is also a need to identify the mechanisms driving compensatory behavioral responses to exercise. While plausible mechanisms exist – including acute changes induced by exercise in fatigue, mood, hunger, stress, food preferences, and sleepiness – few studies have examined the relationships between these factors and compensatory changes in EI and NEAT.

In addition to the above factors, how an individual views exercise engagement may also play a role in behavioral responses. Two alternative hypotheses warrant assessment as possible drivers of compensatory behaviors induced by exercise. Actions toward goals (future events toward which committed endeavors are directed[12]) can be represented in two distinct ways:

(a) in terms of progress toward or (b) in terms of commitment to a desirable end state.[13,14] If a person interprets the movement toward the goal in terms of general level of goal commitment, that perception likely increases motivation to continue toward similar complementary actions and to hinder competing goals.[15-17] Alternatively, Fishbach's[18, 19] goal progress model proposes that if an individual views engaging in a behavior as progressing towards one's goal, the individual will be more likely to engage in pursuit of other goals, which might lead to incongruent actions. For example, if an individual has goals of becoming healthy and spending time with friends, engagement in exercise might make her feel that she has made progress towards becoming healthy and should now shift focus to spending time with friends - that might increase behaviors such as unhealthy eating and sedentary behavior.

1.2 Specific Aims

Primary Aim 1: To examine how exercise intensity (continuous moderate-intensity exercise versus high-intensity interval training) influences compensatory responses (changes in NEAT and EI) to an exercise training program in overweight college-age females.

Hypothesis: Overweight college-age females will exhibit greater compensatory responses in the high-intensity versus moderate-intensity exercise training program.

Primary Aim 2: To explore whether compensatory changes in NEAT and EI explain inter-individual differences in cardiometabolic responses (changes in CRP, GLUC, HDL, INS, LDL, SBP, TG, WC) to moderate- and high-intensity exercise training.

Hypothesis: Compensatory changes in NEAT and EI will be significantly associated with one or more adverse cardiometabolic responses (increased CRP, GLUC, INS, LDL, SBP, TG, WC, decreased HDL) to exercise training among individuals.

Primary Aim 3: To examine the effect of exercise view as commitment versus progress on compensatory changes in NEAT and EI.

Hypothesis: Viewing exercise as progress toward one's goal will lead to increased levels of compensatory responses to exercise training.

1.3 Public Health Related Significance

Currently, more than 1/3 of American adults (34.4% of women aged 20-39) are obese which leads to increased rates of obesity-related conditions including cardiovascular disease and type 2 diabetes.[20] Risk factors for Metabolic Syndrome, a cluster of risk factors that raises an individual's risk for obesity-related conditions, include a large waist circumference (WC), high blood pressure (SBP), high triglycerides (TG), high fasting glucose (GLUC), and low high-density lipoprotein cholesterol (HDL). Physically inactive individuals increase their risk of acquiring Metabolic Syndrome. To lessen the risk of developing Metabolic Syndrome, lifestyle changes including increasing exercise levels and losing excess weight are recommended for at-risk individuals. Unfortunately, on average, individuals engaging in an exercise program only lose ~30% of expected weight loss with a spectrum of responses ranging from more than expected weight loss to weight gain. Considerable heterogeneity in cardiometabolic responses to regular physical activity[21-23] have also been reported, including measures such as SBP, GLUC, and HDL-C. Given the substantial inter-individual variability that has been observed in responses to exercise, a better understanding of the predictors that cause this variability is greatly needed. Behavioral compensatory responses are one possible predictor to help explain the response variations. The data generated from the proposed study will advance understanding of the effects of intensity on behavioral responses to exercise and the mechanisms behind these compensatory behaviors. If intensity is shown to influence behavioral responses, this would

have important implications for exercise recommendations for those inclined to compensate. The generated data will also be used to explore whether compensatory behaviors help explain inter-individual differences in specific cardiometabolic responses to exercise. If found to be influential, those inclined to compensate would need to be provided with tailored exercise prescriptions designed to minimize compensatory behaviors in order to foster beneficial health outcomes from exercise engagement.

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CHAPTER 2

LITERATURE REVIEW

2.1 Introduction

Given the inter-individual variability in responses to exercise, a better understanding of the predictors that cause this variability is greatly needed. Behavioral compensatory responses are one possible predictor to help explain the response variations. This review summarizes the salient literature regarding this topic. First, the prevalence and magnitude of compensatory behaviors are discussed followed by measurement challenges in assessing compensatory changes. Potential modifiers of compensatory behaviors are next explored. Then, prevalence and magnitude of cardiometabolic risk factors are examined followed by the evidence supporting physical activity/exercise in prevention of cardiometabolic risk factors. Lastly, adverse cardiometabolic responses and potential causes are discussed.

2.2 Prevalence and Magnitude of Compensatory Behaviors

Increased energy expenditure through structured exercise creates a negative energy balance if outside behaviors are unchanged.[1] In exercise training studies with compliance monitored, individuals experienced 55-64% less weight loss than expected; often only showing 1-3 kg decreases.[2-4] A review by Riou et al.[5] recently reported individuals experienced only 82% of the body composition changes predicted by exercise energy expenditure (ExEE). These less than predicted results could be due to a lack of compliance to the prescribed exercise,[6] metabolic changes (e.g., reduction of resting metabolic rate[1]), and errors in the estimation of ExEE. Behavioral compensatory responses are a likely contributor and include increases in

energy intake (EI) or decreases in energy expenditure outside of structured exercise (known as non-exercise activity thermogenesis (NEAT)).[2,7]

Most studies to date have focused on changes in EI following exercise, with inconsistent findings reported. In Donnelly et al.'s recent review, half of studies examining changes in EI in response to a short exercise intervention (lasting less than 14 days) reported EI increases, however changes in EI in response to longer exercise interventions (3-72 weeks) were minimal.[8] Nine studies assessing the effects of an acute bout of exercise reported a significant EI increase of approximately 80-470 kcal/day following exercise,[9-17] while 23 studies found no differences,[9,18-40] and four studies reported a significant decrease in EI of approximately 125-240 kcal/day.[25,41-43] Five studies less than 14 days in duration reported increased EI of approximately 200-335 kcal/day.[44-48] Eleven exercise interventions averaging 12 weeks in duration reported no change in EI,[49-59] while one study found a significant increase in EI of approximately 84 kcal/day.[60] In studies involving youth engaged in exercise training programs, 60% showed a decrease in EI, while 40% showed no changes in EI.[61]

Fewer studies have examined compensatory changes in NEAT following exercise programs, with nine studies reporting decreases[62-70] and eight studies no change.[46,71-77] Decreases in NEAT with the onset of exercise have ranged from 7.7%[66] to 62%[63] in studies of aerobic exercise programs lasting 14 and 8 weeks, respectively, and conducted in older adult populations. In short-term studies (≤ 16 days), nine studies reported no significant effect of prescribed exercise on non-exercise physical activity or NEAT.[46,48,55,70,72,78-81] In non-randomized exercise trials that averaged 12 weeks, four studies reported a significant decrease in non-exercise physical activity or NEAT,[62,64-66] while increased NEAT ranging from 28-55% was reported in two trials.[73,82]

Only a few trials to date have included synchronized assessments of chronic changes in NEAT and EI over exercise periods lasting longer than four weeks.[60,83,84] In the 6-month DREW study with overweight/obese postmenopausal women, participants were randomized into one of four groups: a non-exercise control or exercise doses of 4 kcal/kg wk, 8 kcal/kg/wk, or 12 kcal/kg/wk. Individuals in all exercise conditions decreased EI at follow-up and increased daily steps by 558 to 615 steps/d.[83] Noticing the great inter-individual variability in weight loss responses, Herrmann et al.[84] separated adults (age range 18-30 y) from the Midwest Trial 2, which assessed the effects of a 10 month aerobic training program expending 400 or 600 kcal/session five days per week, into nonresponders (those with weight loss <5% of body weight) and responders (≥5% of body weight loss). Forty-six percent of participants were classified as nonresponders. While exercise volume was consistent between the two groups, NEAT increased in responders (116 ± 456 kcal/day) and decreased in nonresponders (NEAT = -238 ± 502 kcal/day). Similarly, nonresponder EI was higher (3076 ± 967 kcal/day) compared to responders (2696 ± 603 kcal/day). A separate 12-week aerobic exercise training study in which overweight/obese adults (39.6 ± 11.0 y) expended 500 kcal/session on five days per week revealed substantial individual variability: responders (based on actual weight loss) reduced EI by 130 ± 485 kcal/day while nonresponders (51% of participants) increased EI by 268.2 ± 455.4 kcal/day.[54] In an 8-week study in 34 overweight/obese females by Manthou et al.[60] where participants completed 150 minutes of aerobic exercise per week, responders (those that achieved reductions in adiposity) increased daily NEAT by 0.79 ± 0.50 MJ/day (188.69 ± 119.42 kcal/day) while non-responders (68% of participants) decreased by 0.62 ± 0.39 MJ/day (148.08 ± 93.15 kcal/day); while the exercise program induced a significant 9.7% increase in EI overall, no differences existed between responders and nonresponders.

Not only are there different behavioral responses between individuals but also potential differences within an individual on exercise versus non-exercise days. Recently, Hopkins et al.[85] reported substantial within-subject variability in EI differences between exercise and non-exercise days in overweight/obese women that ranged from -234.3 to 278.5 kcals/day. Differences may also exist in NEAT values within an individual on exercise versus non-exercise days. Meijer et al.[65] reported lower non-exercise accelerometer output on training days versus non-training days in older adults, whereas average NEAT values for the three days prior and three days after an acute bout of exercise did not differ from the exercise day in young, overweight/obese males.[86]

2.3 Measurement of Compensatory Changes in NEAT and EI

Assessing compensatory changes in NEAT and EI induced by exercise training requires accurate measurements in the free-living setting which is challenging.[87] Doubly labelled water (DLW) is considered the gold standard for measuring total daily energy expenditure.[88] When DLW is used in exercise training studies, NEAT is estimated by subtracting basal metabolic rate, thermic effect of food, and ExEE from total daily energy expenditure. Thermic effect of food is often not measured and assumed to be 10% of total daily energy expenditure; similarly, basal metabolic rate is not always directed measured but may be estimated. Three major limitations exist with using DLW to estimate compensatory changes in NEAT: (a) the cost (~$1000), (b) it provides an EE value over a period of days, making it difficult to detect changes in NEAT unless total ExEE is known, and (c) no information on physical activity patterns is provided.[87] Heart rate monitors are another option to measure EE in the free-living environment but estimates can be skewed due to confounding factors (e.g., ambient conditions, emotional state, hydration status, and fitness level).[89] Accelerometers are a commonly used

method to assess changes in NEAT as they can provide detailed information regarding patterns of physical activity throughout the day, but estimates of EE from accelerometers are less accurate than DLW.[88] In addition to this limitation, use of accelerometers introduces possible estimate inaccuracies due to differences in monitor wear times.[90] Additionally, waist-worn accelerometers may not accurately capture upper body movements or the extra energy cost of load-bearing activities, and are less sensitive to cycling.[91] Combining accelerometry with heart rate monitoring is promising to use in the free-living setting to estimate EE as estimates of EE utilizing both accelerometry and heart rate monitoring have been shown to correlate better with DLW in free-living settings compared to accelerometry or heart rate alone.[92] One such monitor, the Actiheart, records heart rate and body accelerations and uses branched equation modelling to estimate EE (CamNTech, USA). The Actiheart is water-proof enabling it to be worn continuously thus eliminating the influence of varying wear times on EE estimates. Additionally, the addition of heart rate allows better estimates of activities often underestimated by waist-worn accelerometers. The Actiheart produces minute-by-minute EE estimates in kilocalories.

Measuring EI in the free-living setting is also challenging as it is universally recognized that most subjects misreport what and how much they consume.[93] Some foods which are deemed 'healthy' are commonly overreported while 'unhealthy' foods are often underreported; the net result is most individuals substantially underreport EI.[94] Social-desirability and motivation are also influencing factors in EI estimates.[95] Food frequency questionnaires, used to assess frequency of categorized food intake over a specified time period, while a practical and economical method, rely heavily on participants' episodic memories of eating behaviors.[96] 24-hour recalls and 24-hour multiple pass method recalls have gained popularity for providing

more accurate estimates of EI. For example, 7% of females (age range 40-69 y) underreported EI (assessed by DLW and urinary nitrogen) with a 24-hour dietary recall versus 23% of the same group underreporting with a food frequency questionnaire; a substantial improvement with the 24-hour dietary recall.[94]

Technological advances have also enabled improvements over traditional pen-and-paper recalls. Web-based technologies are self-administered instruments involving interactive help features that allow participants to complete diet assessments at a time and location that is most convenient.[97] For example, the National Cancer Institute's self-administered 24-hour dietary recall (ASA24) is an example of a web-based diet data collection tool modeled after the Automated Multiple Pass Method.[97] The ASA24 uses the five pass system to enhance accuracy and completeness of recalls, including a time and occasion pass, meal-based quick list, detail pass, forgotten foods pass, and final review.[98] This process has been shown to reduce bias in EI estimates.[99] The elimination of a trained interviewer with ASA24 substantially reduces costs while providing similar EI estimates to an interview-administered assessment.[100] ASA24 produces daily reported energy intakes in kilocalories along with macronutrient values in grams. Using accurate measurement tools to study changes in NEAT and EI are needed to better understand influencing variables of compensatory behaviors.

2.4 Potential Modifiers of Compensatory Behaviors

Exercise parameters (type, frequency, intensity, duration, time of day, intermittent vs. continuous) in addition to participant characteristics (age, gender, BMI, fitness or activity level) have the potential to modify compensatory behaviors induced by structured exercise.[8,101] For example, while decreased NEAT has been found with exercise intensities of 60-85% VO2max[66] and at 50% heart rate reserve,[65] no change was reported at intensities of 70-77%

maximum heart rate.[53] In a study looking specifically at acute bouts of exercise at different intensities, NEAT was observed to increase by 25% two days after a day of high-intensity aerobic exercise compared to moderate-intensity exercise but did not differ on any of the other seven study days, suggesting this might have been a chance difference.[86] Exercise intensity may also play a role in moderating EI responses, but results have been mixed. In acute studies assessing the effect of exercise intensity on post-exercise EI, four studies reported no effect of intensity,[9,31,36,42] while another study reported a significant increase in EI (295 ± 490 kcals) in college females following high-intensity but not low-intensity exercise.[13] Further, twelve males maintained EI (-5 ± 713 kcals) when exposed to an acute bout of sprint interval exercise and increased EI (145 ± 766 kcals) when exposed to an acute bout of continuous endurance exercise.[102] The effects of exercise intensity on compensatory responses deserves further exploration. Specifically, measuring changes in both NEAT and EI, in addition to matching ExEE between exercise intensity training groups, is needed to better understand the independent effects of intensity on behavioral responses.

Another possible influencer of compensatory behaviors is how the individual views exercise engagement. A goal is a future event toward which a committed endeavor is directed.[103] Actions toward goals can be represented in two distinct ways: (a) in terms of progress toward or (b) in terms of commitment to, a desirable end state.[104,105] If a person interprets the movement toward the goal in terms of general level of goal commitment, that perception likely increases motivation to continue toward similar complementary actions and hinder competing goals.[106-108] However, if the individual interprets the same movement toward a goal in terms of general level of goal progress,[109] it serves as a reason to move away from the original goal to pursue other goals. Perceiving exercise engagement as a commitment to

health may lead an individual to continue in similar positive behaviors (e.g. eating healthy). Alternatively, exercise behaviors perceived as progress towards being healthy may subsequently lead to the pursuit of alternative goals which may include opposing behaviors (e.g. eating and drinking while socializing with friends). These two dynamics—goal commitment versus goal progress—demonstrate some of the paradoxical effects of perceived goal progress on future behaviors.[104,110-112] Better understanding why some individuals are susceptible to increasing EI or decreasing NEAT, in the context of perceived goal progress, is warranted as these behaviors may negate the health benefits associated with exercise.[87]

2.5 Prevalence & Magnitude of Cardiometabolic Risk Factors in College-Age Females

In 2011, CVD accounted for 31.3% of all deaths in the United States, an average of 1 death every 40 seconds.[113] Cardiometabolic risk factor criteria for women include blood pressure (BP) ≥ 130/85, fasting glucose (GLUC) ≥ 100 mg/dL, high-density lipoprotein (HDL) ≤ 50 mg/dL, low-density lipoprotein (LDL) ≥ 130 mg/dL, triglycerides (TG) ≥ 150 mg/dL, and waist circumference (WC) ≥ 88 cm.[114,115] In 207 male and female college students, Dalleck & Kjelland[116] reported 16.4% exhibited elevated BP, 7.2% elevated GLUC, 47.3% low HDL, 13.5% elevated TG, and 5.8% elevated WC. In approximately 1500 college-females, 6.1% had BMI ≥ 30 kg/m2 (mean for group as whole = 23.7 kg/m2), 6.4% had elevated GLUC (group mean = 84.9), 23.7% had low HDL (group mean = 59.3), 45.5% had elevated LDL (group mean = 98.9), and 18.3% had elevated TG (group mean = 113.9).[117]

Prevalence of cardiometabolic risk factors in college students is dependent upon BMI status. College students falling within a normal BMI had lower prevalence of risk factors (0.7% BP, 5.7% GLUC, 18.4% HDL, 16.3% TG, 0.7% WC) compared to overweight (2.9% BP, 8.6% GLUC, 11.4% HDL, 20.0% TG, 14.3% WC) or obese students (15.4% BP, 23.1% GLUC, 61.5%

HDL, 23.1% TG, 61.5% WC).[118] A large sample of college females were surveyed in both 2005 (n=33,134) and 2008 (n=50,949) and 17.1% and 18.2% were classified as overweight and 7.6% and 9.6% classified as obese, respectively, highlighting the need for programs aimed at reducing the prevalence of elevated cardiometabolic values in overweight/obese college females.[119,120]

2.6 Physical Activity/Exercise in Prevention of Cardiometabolic Risk Factors

It is well established that exercise has a positive effect on cardiometabolic risk factors. Improvements in risk factors induced by exercise is consistently seen in diverse groups, including men,[121] children and adolescents,[122] overweight/obese adults,[123] older adults,[124] cardiovascular disease patients,[125] and type 2 diabetes patients.[126] Specifically in women, exercise training is associated with increased HDL (mean ± standard error, 1.8 ± 0.9 mg/dL) and decreased LDL (-4.4 ± 1.1 mg/dL) and TG (-4.2 ± 2.1 mg/dL).[127] Cai & Zou[128] reported in their recent meta-analysis that in exercise training studies lasting less than 2 months and including overweight/obese adults the induced changes were: GLUC (-2.6 mg/dL, 95% CI -8.9, 3.7), HDL (0.3 mg/dL, 95% CI -2.8, 3.4), LDL (0.8 mg/dL, 95% CI -4.1, 5.7), and TG (-9.2 mg/dL, 95% CI -18.2 to -0.2).[128] Further, reviews have shown decreases in C-reactive protein (CRP) levels (-0.53 mg/L, 95% CI -0.74, -0.33) induced by exercise training.[129] Specifically in young women, 16 weeks of aerobic exercise improved HDL and TG levels in 10 women,[130] as did 12 weeks of exercise training in 12 overweight females in addition to improved LDL levels as well.[131] Additionally, fasting insulin (INS) decreased by 33% following 12 weeks of exercise training as part of a lifestyle intervention in young women.[132] Previous research has shown that physical activity and exercise are effective for

prevention of adverse cardiometabolic outcomes in diverse groups, including young adults.[133,134]

In previous reviews, most of the evidence has been based on continuous moderate-intensity exercise, but sprint-interval exercise may offer similar health benefits.[135] Sprint-interval exercise has gained popularity, specifically one popular protocol is characterized by cycles of 30 second 'all out sprints' followed by 4-4.5 minutes of recovery, and is often performed on a cycle ergometer.[136] There is a growing body of evidence suggesting sprint-interval exercise is also effective in the prevention of cardiovascular risk factor development. In fact, in recent reviews sprint-interval exercise interventions performed as well or better than continuous moderate-intensity exercise in inducing SBP decreases,[137] GLUC decreases,[138] INS decreases,[139] TG decreases,[140] and VO2max gains.[136] Additional studies are needed to further compare cardiometabolic responses to sprint-interval versus continuous moderate-intensity exercise training.

2.7 Prevalence & Magnitude of Adverse Cardiometabolic Responses and Potential Causes

Inter-individual variability exists not only in less than predicted weight loss,[54,60,83] but also in cardiometabolic responses to exercise training. This variability can result in adverse responses in some individuals with the prevalence of adverse responses ranging from 8.3-13.3% [141]. Specifically, data from six major exercise studies were analyzed and researchers found that 13.3% of exercise participants experienced "adverse responses" in HDL, 10.3% in TG, 12.2% in SBP, and 8.3% in INS.[141] Adverse responses were defined as "an exercise-induced change that worsens a risk factor beyond measurement error and expected day-to-day variation" and were quantified from change scores as follows: HDL ≤ -4 mg/dL (-0.12 mmol/L), GLUC ≥ 14.4 mg/dL (0.8 mmol/L), INS ≥ 3.5 mU/L (24 pmol/L), SBP ≥ 10 mmHg, TG ≥ 37 mg/dL (0.42

mmol/L).[141,142] Thirty-one percent of participants in the six studies experienced one adverse response to exercise, 6% experienced two, and 0.8% experienced three or more.[141] Additionally, in a separate 14-week aerobic plus resistance exercise program with 332 adults (age range 28-88 y), 6.0% of participants experienced adverse responses in SBP, 3.6% for TG, and 5.1% for HDL.[143] The prevalence of multiple adverse cardiometabolic responses in this study population was 1.2%.

When studying those who experienced adverse cardiometabolic responses to exercise, Dalleck et al.[143] described the possibility that other factors (eg, dietary and sedentary behavior/sitting time) might have contributed to the prevalence of adverse responses. Recently, researchers have assessed cross-sectional associations between light intensity physical activity, which is characteristic of most NEAT, and cardiometabolic outcomes in groups with equivalent levels of moderate-to-vigorous intensity physical activity.[144,145] Loprinzi et al.[145] reported individuals with greater levels of light intensity physical activity had more desirable values for HDL, INS, and TG. Bakrania et al.[144] reported similar desirable results in HDL and WC values for those with higher levels of light intensity physical activity. These cross-sectional studies highlight the potential impact of NEAT, independent of exercise, in modulating cardiometabolic outcomes.

Energy intake is also a known influencer of cardiometabolic markers.[142] For example, after a 7-day fast (300 kcal/day) overweight females experienced SBP reductions of -16.2 mmHg in addition to decreased LDL and INS levels.[146] Individuals (age range 20-51 y) in a 2-year intervention designed to decrease caloric intake by 25% of baseline levels experienced decreases in CRP (-0.5 µg/mL), TG (-25.0 mg/dL), and SBP (-3.0 mmHG), all significantly different compared to the control group.[147] Similarly, in a 12-week energy-restriction study (1200

kcal/day) overweight/obese females (49.6 ± 11.0 y, 31.2 ± 2.1 kg/m^2) experienced significant decreases in SBP (-8.5 ± 23.5 mmHg), LDL (-9.1 ± 18.7 mg/dL) and non-significant decreases in GLUC (-2.7 ± 10.7 mg/dL), TG (-10.9 ± 52.9 mg/dL).[148] Since moderate diet changes have been observed to influence a range of cardiometabolic risk factors, it is plausible that changes in EI in response to exercise may influence observed changes in cardiometabolic risk factors. However, studies are needed which directly examine this issue.

2.8 Summary

A closer look at factors that may affect behavioral compensatory responses is needed in order to enhance the efficacy of exercise interventions for weight loss and metabolic health.[149] Simultaneous measurement of temporal changes in NEAT and EI during participation in structured exercise is needed to further understand these potential counter-productive behavioral responses. In addition, further evidence is needed to understand whether the intensity of exercise programs influences the behavioral compensatory response and to determine if these responses differ acutely on exercise versus non-exercise days. Also, associations between compensatory and adverse metabolic responses to exercise have yet to be explored. Nor have views of exercise engagement and compensatory responses been studied. Thus, the first aim of this study was to examine the effects of exercise intensity on compensatory responses (changes in NEAT and reported energy intake (REI)) upon initiation of an exercise training program in overweight/obese college-age females and to compare differences in these responses on exercise versus non-exercise days. The second aim of this study was to explore whether compensatory changes in NEAT and REI explain inter-individual differences in cardiometabolic responses (changes in CRP, HDL, GLUC, INS, LDL, SBP, TG, WC) to exercise training in college-age

females. Finally, the third aim of this study was to assess the independent associations of exercise view (i.e. commitment vs. progress) and compensatory behaviors.

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CHAPTER 3

THE EFFECT OF CONTINUOUS MODERATE OR SPRINT INTERVAL CYCLING ON COMPENSATORY RESPONSES IN COLLEGE-AGE FEMALES[1]

[1]Hathaway, E.D, Fedewa, M.V., Higgins, S., Evans, E.M., Schmidt, M.D. To be submitted to *Journal of Physical Activity and Health*.

3.1 Abstract

Background:

A better understanding of the factors that affect behavioral compensatory responses to exercise is needed in order to enhance the efficacy of interventions for weight loss. Therefore, this study examined whether the intensity of exercise programs influences behavioral responses and whether these responses differ acutely on exercise versus non-exercise days.

Methods:

Overweight/obese previously inactive females (n=63, 20.4 ± 1.6 y, 30.7 ± 5.0 kg/m^2, 67% Caucasian) were randomized to 6-weeks of a) continuous moderate-intensity exercise (MOD-C) or b) sprint-interval exercise (VIG-SIC). Non-exercise activity thermogenesis (NEAT) and reported energy intake (REI) were assessed over 4-day continuous periods at baseline and two time points during the program on exercise and non-exercise days.

Results:

Participants maintained NEAT throughout the study (-0.40 ± 180.2 kcals/day, p=0.99) and decreased REI (-177.1 ± 489.6 kcals/day, p=0.01). There were no significant changes in NEAT values found within or between groups on exercise or non-exercise days. No within group differences were found in REI changes although differences in the VIG-SIC group on exercise weekdays versus non-exercise weekends approached significance (p=0.06). No between group differences were found in REI although the differences between groups on non-exercise weekend days approached significance [328.1 kcals (95% CI = -658.6, 2.4, p=0.06)].

Conclusions:

The results of this study indicate no differences in weighted NEAT or REI compensatory responses to either continuous moderate-intensity or sprint-interval training in overweight/obese

college females during 6-weeks of exercise training. On average, neither exercise intensity group experienced adverse compensatory responses, but 50% of all participants decreased NEAT and 34% increased REI. Future research should assess compensatory responses to larger and longer exercise doses in more diverse populations.

3.2 Introduction

Exercise training has been shown to be an important factor for long-term weight management and is also a component of most weight loss programs – both to increase energy expenditure (EE) and to preserve lean body mass.[1, 2] However, there is substantial evidence that the average impact of exercise programs on weight loss is up to 70% less than predicted by the increase in exercise energy expenditure (ExEE).[3] These attenuated effects have been shown to persist when changes in body composition are examined, rather than changes in body weight which may be reduced by exercise induced increases in lean mass.[4]

Metabolic changes (e.g., reduction of resting metabolic rate[5]) and errors in the estimation of ExEE may account for some of the attenuated weight loss responses that have been observed. However, behavioral changes that accompany participation in structured exercise programs, such as increases in energy intake (EI) or decreases in energy expenditure outside of structured exercise (known as non-exercise activity thermogenesis (NEAT)),[6, 7] are more likely to explain these findings. Changes in behavior that occur due to exercise participation, such as eating a calorically dense food post-exercise or engaging in sedentary screen based leisure activities at night following exercise, have been labelled "compensatory" as they may compensate, at least in part, for the increases in ExEE. Recent evidence suggests that behavioral compensatory responses may collectively result in 55-64% less weight loss than predicted based on the level of exercise energy expenditure.[7]

While the influence of compensatory behavioral responses on weight changes following exercise participation is generally accepted, the extent to which changes in NEAT and/or EI contribute to the overall compensatory response remains unclear. Most studies to date have focused on changes in EI following exercise, with inconsistent findings reported. Over 50% of

studies examining changes in EI in response to a short exercise intervention (lasting less than 14 days) reported partial or full compensatory responses, however changes in EI in response to longer exercise interventions (3-72 weeks) were minimal.[8] Exercise intensity may play a role in moderating EI responses, but results have been equivocal. Seventeen college females increased daily EI by 295 ± 490 kcals ($p>0.05$) and 112 ± 334 kcals ($p>0.05$) when exposed to an acute bout of high-intensity and low-intensity exercise, respectively, compared to a control day.[9] In a separate study, daily EI was also increased in eleven college males exposed to differing intensities of an acute bout of exercise (7 kcals ($p>0.05$) with high-intensity exercise vs. 190 kcals ($p>0.05$) with moderate-intensity exercise).[10] Further, twelve males exposed to an acute bout of sprint interval exercise maintained EI (-5 ± 713 kcals; $p>0.05$) and increased EI (145 ± 766 kcals; $p>0.05$) when exposed to an acute bout of continuous endurance exercise compared to a control day.[11] These prior studies did not, however, equalize ExEE between groups which makes it difficult to differentiate the effects of intensity from overall exercise dose.

A limited number of studies have examined compensatory changes in NEAT following exercise training with some studies reporting decreases[12-20] and other studies no change.[21-28] Decreases in NEAT when beginning a structured exercise training program have ranged from 7.7%[16] to 62%[13] in studies of aerobic exercise programs lasting 14 and 8 weeks, respectively, and conducted in older adult populations. In contrast, NEAT was observed to increase by 25% two days after a day of high-intensity aerobic exercise in an acute study in overweight/obese males (26.5 ± 3.0 y).[29] Several factors may explain, in part, these discrepant findings.[30] For instance, compared to moderate-intensity exercise, vigorous exercise may cause a greater reduction in NEAT because of fatigue and discomfort.[31] Participant age may also modify behavioral responses to exercise as multiple studies have shown greater reductions

in non-exercise physical activity in response to training in older adults.[14-16] Differences in the methods used to measure changes in NEAT may also contribute to the divergent results. For example, while accelerometers provide an objective measure of physical activity, most subjects fail to wear the devices during all waking hours, thus not capturing all physical activity. Finally, variations in the timing of measurement may also contribute to inconsistent findings, as NEAT was observed to be significantly lower on exercise versus non-exercise days in older adults during a 12-week combined aerobic and resistance training program.[14]

In order to improve our understanding of the relative importance of changes in EI and NEAT in moderating the effectiveness of exercise as a tool for weight management, studies are needed which measure simultaneous behaviors using robust methodologies. However, only a few trials to date have included synchronized assessments of chronic changes in NEAT and EI over exercise periods lasting longer than four weeks.[4, 32, 33] In the DREW study, participants were randomized into one of four groups: a non-exercise control or exercise doses of 4 kcal/kg wk, 8 kcal/kg/wk, or 12 kcal/kg/wk. Individuals in all exercise conditions decreased EI at follow-up and increased daily steps by 558 to 615 steps/d.[32] Manthou et al.[4] also measured changes in EI and NEAT during an 8-week program. In response to 150-min/wk of moderate intensity exercise among women aged 31.7 ± 8.1 years, EI significantly increased by 9.7%. In addition, participants who lost the expected amount of weight increased NEAT by 0.79 ± 0.50 MJ/day (188.69 ± 119.42 kcal/day) while those who did not decreased NEAT by 0.62 ± 0.39 MJ/day (148.08 ± 93.15 kcal/day). Finally, a study by Herrman et al.[33] defined those with weight loss ≥ 5% of body weight as responders and < 5% as nonresponders. Responders increased NEAT by 116 ± 456 kcal/day and increased EI by 121 kcal/day while nonresponders decreased NEAT by -128 ± 502 kcal/day and increased EI by 38 kcal/day.

A closer look at factors that may affect behavioral compensatory responses is needed in order to enhance the efficacy of interventions for weight loss.[34] Simultaneous measurement of temporal changes in NEAT and EI during participation in structured exercise is needed to further understand these counter-productive behavioral responses. In addition, further evidence is needed to understand whether the intensity of exercise programs influences the behavioral compensatory response and to determine if these responses differ acutely on exercise versus non-exercise days. Thus, the aims of this study were to examine the effects of exercise intensity on compensatory responses (changes in NEAT and reported energy intake (REI)) upon initiation of an exercise training program in overweight/obese college-age females and to compare differences in these responses on exercise versus non-exercise days.

3.3 Methods

Participants

Female college students were recruited via email and print advertising between February 2014 and January 2015 with e-mail messages sent directly to the university e-mail account for each student, through a listserv created by the Office of the Registrar. Interested participants completed an online survey and were contacted by researchers if they met inclusion criteria. Inclusion criteria included: female gender, enrolled student at the University, age between 18-24 years, body mass index (BMI) > 25 kg/m2, waist circumference (WC) > 88 cm, and self-reported being inactive (< 30 minutes of moderate-to-vigorous PA, < 2 days per week). Exclusion criteria included being pregnant or recently (<12 months) pregnant females, a current smoker (<6 months), or a varsity athlete. Individuals with a health condition potentially exacerbated by moderate or vigorous exercise were also excluded. BMI, WC, and self-reported physical activity status were reassessed in person by researchers after the online screening. While 92 eligible

students completed the screening process, five did not have access to the recreational facility where training sessions were held, seventeen never enrolled, and seven withdrew during the study leaving a sample size of n=63. The University Institutional Review Board approved the study protocol and informed consent document, and written informed consent was obtained from each participant prior to enrollment.

Design

This study used a parallel-arm, non-crossover design, with participants randomized to a 6-week continuous moderate-intensity exercise treatment group (MOD-C) or to a 6-week sprint-interval exercise treatment group (VIG-SIC) after being stratified on BMI status (overweight vs. obese). Participants performed their assigned training protocol three times weekly (on Mondays, Wednesdays, Fridays, and - if needed – a makeup weekend day) in a group-training format under the supervision of trained research staff. Training groups were matched on exercise energy expenditure (ExEE) to isolate the effect of exercise intensity on the outcomes of interest. Heart rate during exercise, rating of perceived exertion, and estimated EE from the cycle ergometer were recorded during each exercise session to further ensure compliance with the training protocol. Participants were required to attend a minimum of 13 sessions (70%) to be included in the final analysis. Free-living energy expenditure, and reported energy intake were measured at baseline (Week 0), mid-study (Week 2 or 3), and end of study (Week 5 or 6) during two weekdays (one non-exercise and one exercise day) and two weekend days.

Exercise Interventions

A magnetic-resistance stationary cycle ergometer was used for all exercise sessions for both the VIG-SIC and MOD-C groups (Keiser, Keiser M3 Indoor Cycle, Fresno, California).

VIG-SIC

Participants assigned to the VIG-SIC group began each exercise session with a brief warm-up, followed by repeated bouts of 30-second sprints interspersed with four minutes of active recovery pedaling against minimal resistance at a low pedal frequency. During weeks one and two, participants repeated this procedure to complete 5 sprints, which equated to 2.5 minutes of near-maximal effort sprinting interspersed with 16 minutes of recovery and a subsequent cool-down period following each session. Training progression increased the number of sprint repetitions to 6 sprints during weeks three and four, and to 7 sprints during weeks five and six.

MOD-C

Participants assigned to the MOD-C group were instructed to cycle continuously at an intensity of 60-70% heart rate reserve (HRR) for 20-30 minutes to match energy expenditure (EE) of the VIG-SIC group, with the duration of each training session increased on a biweekly basis in order to maintain equal EE between groups. Individual HRR values were determined by a maximal graded exercise test prior to randomization. During the test, participants pedaled continuously on an electronically braked cycle ergometer (Lode Excalibur Sport 2000; Lode B.V., Groninger, Netherlands) while oxygen uptake was measured using an indirect calorimetry system (ParvoMedics True Max 2400; ParvoMedics, Sandy, UT) and heart rate was measured continuously throughout the test (Polar FT1; Polar Electro, Kempele, Finland).

Measures

Free-Living Energy Expenditure

Energy expenditure was measured objectively over a 4-day continuous period at baseline, mid-study (Week 2 or 3), and end of study (Week 5 or 6) using the Actiheart monitor (CamNtech, USA) positioned at the level of the third intercostal space using ECG electrodes

(Red Dot 2560, 3M). The Actiheart records heart rate and body accelerations and uses branched equation modelling to estimate Activity Energy Expenditure (AEE). The Actiheart has been shown to produce reliable estimates for walking and running activities[35] and is a valid and reliable measure for estimating total energy expenditure.[36, 37]

Reported Energy Intake

Reported energy intake was measured for continuous 4-day periods at baseline, mid-study (Week 2 or 3), and end of study (Week 5 or 6) using the Automated Self-Administered 24-hour Recall (ASA24), a Web-based tool modeled on the USDA's Automated Multiple Pass Method (AMPM). The ASA24 guides participants through the completion of a 24-hour dietary recall by using an online dynamic user interface. The program then estimates total caloric intake and provides details regarding macronutrient and micronutrient intake.[38] It performed well (3% difference) in comparison to an interviewer-administered assessment and is recommended for assessing the effect of interventions on diet.[39]

Anthropometric Measures

Body weight and height were collected pre and post the exercise intervention. Standing height was measured by a stadiometer (Seca 242, SECA Corp, Hamburg, Germany) to the nearest 0.1 cm. Body weight was measured with a digital scale (Tanita WB-110A class III, Tanita Corporation, Tokyo, Japan) to the nearest 0.1 kg. BMI was calculated as weight divided by height squared (kg/m^2).

Computation of Outcome Variables

Change scores for NEAT and REI were computed as intervention values minus baseline values where a positive number corresponds to an increase in the variable of interest during the intervention. Actiheart estimates of NEAT were excluded for days having ≥ 10% missing heart

rate values over the 24 hour-period. Based on this requirement 12.2% of daily NEAT values were excluded from analyses (8.7% on non-exercise weekdays, 19.0% on exercise weekdays, and 8.7% on weekend days). Participants failed to complete the online diet recalls 6.3% of the time (4.0% on non-exercise weekdays, 11.1% on exercise weekdays, and 4.0% on weekend days). No significant changes were seen between mid-study and end of study assessments so these values were averaged to compute one value for non-exercise weekdays, exercise weekdays, non-exercise weekend days, and exercise weekend days (if applicable, as participants were not required to attend the makeup weekend sessions). In order to calculate changes in NEAT on exercise days, the equivalent exercise time period was removed from baseline values. A weighted summary change score was also computed for each individual as [(Non-exercise weekday x 2) + (Exercise weekday x 3) + (Weekend average x 2)]/7. In order to calculate the weighted score, participants had to have data for all three days, leading to weighted change scores for 61 participants with REI and 58 participants with NEAT.

Data Analysis

Statistical analyses were performed using SAS 9.4 (SAS Institute Inc., Cary, NC). After testing for normality and homogeneity of variance using standard procedures, baseline group differences were assessed using independent T-Tests. Baseline weekday and weekend values were compared using dependent T-Tests. Changes in NEAT were analyzed using a 2-way ANCOVA (Group X Day) controlling for baseline NEAT where group was included as the between factor and day was included as the within factor. The equivalent procedure was performed for REI changes. Statistical significance was set at an α level of .05. All data are expressed as mean ± standard deviation (M ± SD), unless otherwise indicated.

3.4 Results

A total of 63 participants completed the study with no significant differences existed between groups at baseline for age or BMI (see Table 3.1). Baseline values for NEAT, REI, and macronutrient intake are shown in Table 3.2; no significant differences between the MOD-C and VIG-SIC groups were observed. During the exercise intervention, ExEE did not differ between the two groups (137.1 ± 35.1 kcals/session in MOD-C vs. 140.7 ± 39.4 kcals/session in VIG-SIC; p=0.72).

Changes in NEAT

Within groups, there were no significant differences in NEAT changes by day classification. As shown in Figure 1, the MOD-C group experienced little change in NEAT values (ranging from -7.1 to 3.7 kcals) for all days except exercise weekdays (-30.6 kcals). Alternatively, the VIG-SIC exhibited more deviation from baseline values with NEAT change scores increasing by 38.4-43.3 kcals on exercise weekdays and both weekend days regardless of exercise. Conversely, those in the VIG-SIC decreased NEAT values on non-exercise weekdays by 28.3 ± 192.6 kcals.

No differences were found between groups on exercise or non-exercise days when controlling for baseline NEAT values (Figure 3.1). Differences between groups were 32.0 kcals (95% CI = -67.1, 131.1, p=0.52) on non-exercise weekdays, 38.5 kcals (95% CI = -145.4, 68.4, p=0.47) on exercise weekdays, 50.4 kcals (95% CI = -160.8, 60.0, p=0.36) on non-exercise weekend days, and 69.2 kcals (95% CI = -259.4, 121.1, p=0.45) on exercise weekend days.

Weighted changes in NEAT did not differ from baseline values in either group (MOD-C p=0.82, VIG-SIC p=0.84) and were not different between groups [MOD-C: -6.2 (95% CI = -61.0, 48.6), VIG-SIC: 5.4 (95% CI = -49.4, 60.2); p=0.77]. Inter-individual variability in

weighted changes in NEAT is shown in Figure 3.2. Weighted NEAT values decreased in 29 participants (50%) with an average change of -0.40 ± 180.2 kcals (p=0.99) in the study sample.

Changes in REI

No significant differences were found within groups in REI changes in the MOD-C group by day classification, while the differences in the VIG-SIC group on exercise weekdays versus non-exercise weekend days approached significance. Significant changes from baseline REI values were seen in both groups on exercise weekdays regardless of exercise condition (Figure 3.3). Individuals in both the MOD-C and VIG-SIC groups reported significant decreases in energy intake on exercise weekdays (-300.9 and -270.7 kcals, respectively). The MOD-C group decreased REI on all other study days with mean changes ranging from -206.1 to -8.8 kcals. Alternatively, the VIG-SIC increased REI on non-exercise weekend days by 190.8 kcals but decreased REI on all other study days with mean changes ranging from -476.7 to -166.4 kcals.

No between group differences in REI were found although the differences between groups on non-exercise weekend days approached significance [328.1 kcals (95% CI = -658.6, 2.4, p=0.06)]. Differences between groups on other days were 21.1 kcals (95% CI = -215.8, 258.0, p=0.86) on non-exercise weekdays, 39.3 kcals on exercise weekdays (95% CI = -245.3, 323.9, p=0.78), and 223.3 kcals (95% CI = -577.0, 1023.6, p=0.57) on exercise weekends.

Weighted changes in REI did differ from baseline values in MOD-C (p<0.01), and a trend for decreased values existed in VIG-SIC (p=0.06). There were no differences between groups [MOD-C: -209.7 (95% CI = 349.1, -70.3), VIG-SIC: -141.1 (95% CI = 287.6, 5.3); p=0.50]. Inter-individual variability in weighted changes in REI is shown in Figure 3.4. Weighted REI changes were increased in 21 participants (34%) with an average change of -177.1 ± 489.6 kcals in the study sample (p=0.01).

3.5 Discussion

This study assessed the effects of exercise intensity on compensatory changes in NEAT and REI in overweight/obese college-age females and compared compensatory responses on exercise versus non-exercise days. The primary findings are 1) no differences were found in NEAT or REI compensatory responses between the two exercise intensities although REI differences on non-exercise weekend days approached statistical significance, 2) when all participants were combined, group mean NEAT levels were maintained and REI levels decreased, and 3) no differences were seen on exercise vs. non-exercise days within groups although REI differences in the VIG-SIC group on exercise weekdays versus non-exercise weekends approached statistical significance. The results of this study suggest that either moderate- or high-intensity exercise trainings can be prescribed in this population without affecting compensatory responses.

Few studies have simultaneously assessed compensatory responses in both NEAT and REI. Often NEAT is measured alone and assumptions about changes in EI are made. For example, NEAT was maintained during an 8-day period with every other day exercise sessions in young (24 ± 3 y) lean females expending ~500 kcal per session (NEAT over 8-day period: 19.4 vs. 21.1 MJ (4633.6 vs. 5039.7 kcal), during the exercise period versus control period, respectively; $p>0.05$).[22] The ExEE and changes in NEAT did not explain the reduced body mass of the eight females which led the authors to speculate that weight reduction was the result of a decrease in EI, although not directly measured in the study.[22] In the current study, NEAT levels were maintained and REI decreased suggesting overweight females of this age may be likely to maintain NEAT and decrease EI when beginning a structured exercise program. Similarly, Turner et al.[26] reported that NEAT was maintained over a 6-month exercise period

in previously inactive male participants, but the authors hypothesized the less-than-predicted weight loss was due to compensatory increases in EI in this older population (54 ± 5 y). This highlights the potential moderating effect of gender, age, and weight status on compensatory responses. While exercise intensity might not affect responses in younger participants, it appears that it may influence responses in older participants. Also, the decrease in REI observed in the current study may be due, in part, to the sample being overweight/obese and that many expressed a desire to lose weight.

Few studies to date have compared the effects of exercise intensity on changes in NEAT. Alahmadi et al.[29] reported no differences in NEAT on the exercise day or the following day to both moderate-intensity continuous and high-intensity interval walking in sixteen overweight/obese males (27 ± 3 y). Conversely, Kriemler et al.[40] reported decreased NEAT following an acute bout of 50-minutes high-intensity cycling (-30.1 kJ/h (-7.2 kcal/h), $p<0.05$) and increased NEAT following 30-minutes medium-intensity cycling (35.1 kJ/h (8.4 kcal/h), $p=0.05$) in fourteen moderately obese boys. The differences in these responses might be explained by differences in ExEE. Both Alahmadi et al.[29] and Kriemler et al.[40] utilized within-subject designs and assessed the effects of an acute bout of exercise on changes in NEAT. The current study allowed the assessment of chronic effects over the 6-week exercise intervention. The decreased REI reported in this study is consistent with the study by Martins et al.[41] which reported no difference in EI in twelve overweight/obese participants (33 ± 10 y) performing acute isocaloric bouts (250 kcals/session) of high-intensity intermittent cycling and moderate-intensity continuous cycling. Similarly, Ueda et al.[42] reported decreased EI in response to 30-minute acute bouts of both moderate- (50% VO2max) and high-intensity (75%

VO2max) cycling compared to a resting condition with no differences evident between exercise intensities in males (23 ± 4 y).

Strengths

The current study adds to the body of knowledge on compensatory responses and has multiple strengths. First, NEAT and REI changes were measured simultaneously, an improvement upon many previous studies which have only measured one of the two components. Two separate 4-day measurements were collected during the exercise intervention compared to some studies that have only assessed compensatory responses at one time point during the exercise program. Exercise energy expenditure between the two groups was also designed to be equivalent, which enabled the comparison of exercise intensity effects independent of differences in ExEE. Further, no group mean difference in energy expenditure was observed in the 1-hour period following the exercise sessions (MOD-C = 66.2 ± 39.7 kcals versus VIG-SIC = 76.2 ± 36.2 kcals, p=0.34), suggesting that differences in post-exercise energy expenditure were not induced by the two exercise conditions. In addition, the current study utilized a contemporary and accurate measure of NEAT over continuous 4-day periods, thus eliminating the potential issue of wear time differences which can arise when using other objective measures of NEAT. Finally, in order to compare NEAT values during the exercise intervention to baseline values, the time period spent in structured exercise during the intervention was removed from the baseline values. Correcting for time spent in structured exercise is warranted, as a failure to so would lead to a reduction in the time that can be dedicated to NEAT and thus introduce potential bias.[43]

Limitations

The present study is not without limitations. While an objective measure was used for the measurement of NEAT, self-report was used to assess energy intake. The ASA24 is an improvement upon previously used Food Frequency Questionnaires; 7% of females underreported EI (assessed by doubly labeled water and urinary nitrogen) with a 24-hour dietary recall versus 23% of the same group underreporting with a food frequency questionnaire, a substantial improvement with the 24-hour dietary recall.[44] Measuring EI during an intervention is challenging, as some individuals underreport their true intake or may intentionally change their diet during the assessment period.[45, 46] Second, individuals were assessed at only two time points during the exercise intervention period, in addition to the baseline period, which may have failed to capture true behavior changes throughout the 6-week program. Third, all activity energy expenditure at baseline was classified as NEAT based on the assumption that no intentional exercise was undertaken (according to self-report). Fourth, because the variability in NEAT and REI responses to the two exercise conditions was unknown, it was not possible to perform an *a priori* power calculation. A retrospective power calculation (two-tail, α=0.05, power=0.80) indicated that the study had sufficient power to detect an effect size of 0.75 in NEAT or REI between the MOD-C and VIG-SIC groups. This effect size is considered large and the present study is only powered to assess differences of more than 0.75 standard deviations between the two exercise groups.

Conclusions

In conclusion, the results of this study indicate no differences in weighted NEAT or REI compensatory responses to either continuous moderate-intensity or sprint-interval training in overweight/obese college females during 6-weeks of exercise training, as both groups maintained

baseline NEAT levels and decreased REI. These results are promising as they suggest that, in this study population, engaging in exercise may lead to sustained increases in energy expenditure that are not counteracted by negative compensatory responses. Future research should assess compensatory responses to larger and longer exercise doses in more diverse populations and monitor participants throughout the intervention to better capture any compensatory responses that may occur.

3.6 Acknowledgements

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Table 3.1. Demographics.

	All (n=63)	Study Groups: MOD-C (n=32)	Study Groups: VIG-SIC (n=31)
	Mean ± SD or N (%)	Mean ± SD or N (%)	Mean ± SD or N (%)
Age	20.4 ± 1.6	20.3 ± 1.4	20.5 ± 1.8
Race/Ethnicity			
Asian	3 (4.8)	2 (6.3)	1 (3.2)
Black	11 (17.5)	5 (15.6)	6 (19.4)
Hispanic	4 (6.3)	1 (3.1)	3 (9.7)
White	42 (66.7)	23 (71.9)	19 (61.3)
Other	3 (4.8)	1 (3.1)	2 (6.5)
Weight	84.1 ± 13.9	82.4 ± 13.1	85.9 ± 14.7
BMI	30.7 ± 5.0	30.6 ± 5.1	30.9 ± 4.8

MOD-C = moderate-intensity, continuous group; VIG-SIC = vigorous-intensity, sprint interval cycling group; SD = standard deviation; BMI = Body Mass Index reported in kg/m^2; Note: white and black are non-Hispanic

Table 3.2. Baseline Values of NEAT and REI.

	All		Study Groups: MOD-C		Study Groups: VIG-SIC		Between Group
	n	Mean ± SD	n	Mean ± SD	n	Mean ± SD	P value
NEAT (kcals)							
Weekday	62	627.7 ± 217.2	31	586.3 ± 203.9	31	669.0 ± 225.4	*0.13*
Weekend	61	545.1 ± 261.7	32	498.9 ± 220.4	29	596.0 ± 296.5	*0.15*
Within Group (P Value)	60	*0.01*	31	*0.09*	29	*0.07*	
REI - KCALS							
Weekday	63	1980.6 ± 684.8	32	1949.8 ± 710.8	31	2012.3 ± 667.0	*0.72*
Weekend	62	1882.4 ± 627.9	32	1890.5 ± 636.2	30	1873.7 ± 629.6	*0.92*
Within Group (P Value)	62	*0.29*	32	*0.66*	30	*0.3*	
REI - PROT (grams per 1,000 kcal)							
Weekday	63	36.6 ± 9.4	32	37.1 ± 9.8	31	36.2 ± 9.1	*0.71*
Weekend	62	35.6 ± 9.8	32	37.5 ± 11.9	30	33.5 ± 6.3	*0.10*
Within Group (P Value)	62	*0.52*	32	*0.85*	30	*0.17*	
REI - TFAT (grams per 1,000 kcal)							
Weekday	63	38.9 ± 8.8	32	40.5 ± 8.4	31	37.1 ± 9.0	*0.13*
Weekend	62	39.3 ± 7.8	32	40.0 ± 8.4	30	38.6 ± 7.3	*0.50*
Within Group (P Value)	62	*0.53*	32	*0.76*	30	*0.19*	
REI - CARB (grams per 1,000 kcal)							
Weekday	63	116.6 ± 24.6	32	112.5 ± 20.8	31	120.9 ± 27.7	*0.18*
Weekend	62	123.8 ± 23.6	32	120.5 ± 26.4	30	127.3 ± 19.9	*0.26*
Within Group (P Value)	62	*0.06*	32	*0.13*	30	*0.29*	
REI – ALC (grams per 1,000 kcal)							
Weekday	63	7.2 ± 14.2	32	7.2 ± 15.6	31	7.2 ± 12.8	*0.99*
Weekend	62	3.0 ± 8.3	32	3.0 ± 9.5	30	3.1 ± 6.9	*0.96*
Within Group (P Value)	62	*0.02*	32	*0.12*	30	*0.06*	

MOD-C = moderate-intensity, continuous group; VIG-SIC = vigorous-intensity, sprint interval cycling group; NEAT = non-exercise activity thermogenesis; REI = reported energy intake; PROT = protein; FAT = total fat; CARB = carbohydrate; ALC = alcohol

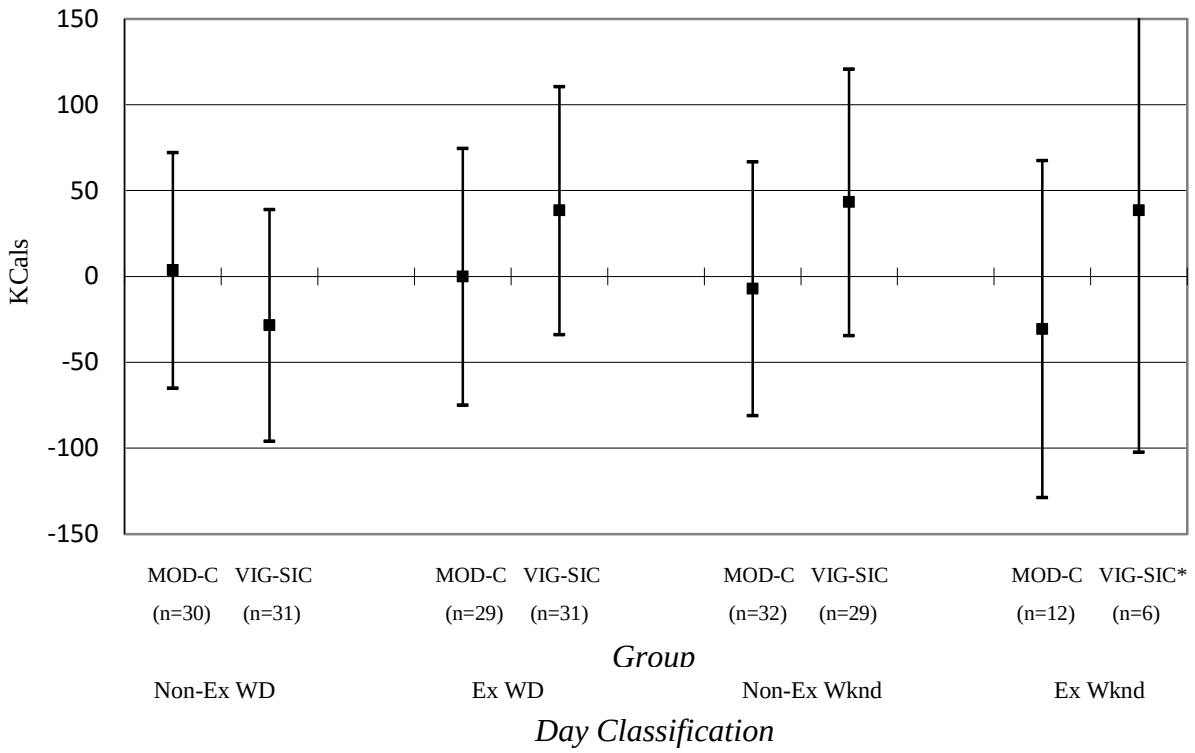

Figure 3.1. Changes in NEAT during exercise program. Adjusted for baseline values on all days and exercise periods on exercise days. NEAT = non-exercise activity thermogenesis; MOD-C = moderate-intensity, continuous group; VIG-SIC = vigorous-intensity, sprint interval cycling group; Non-Ex WD = non-exercise weekday; Ex WD = exercise weekday; Non-Ex Wknd = non-exercise weekend; Ex Wknd = exercise weekend. Bars represent 95% Confidence Interval. *95% CI for VIG-SIC Ex Wknd = (-102.23, 179.43).

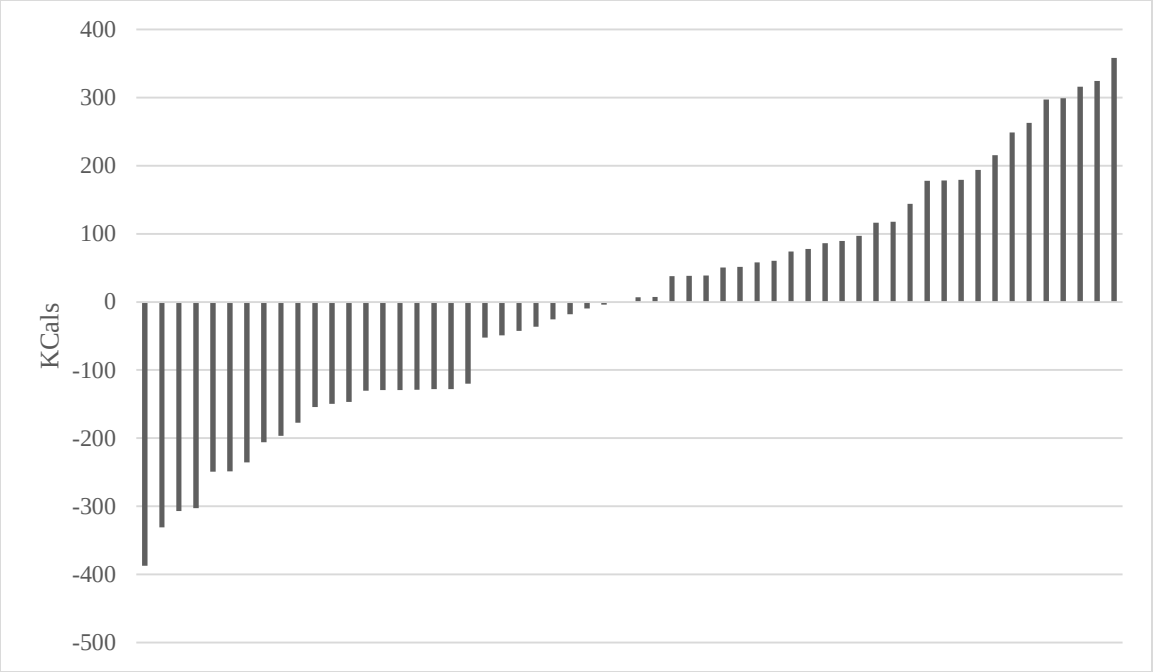

Figure 3.2. Individual weighted changes in NEAT during exercise program (n=58).

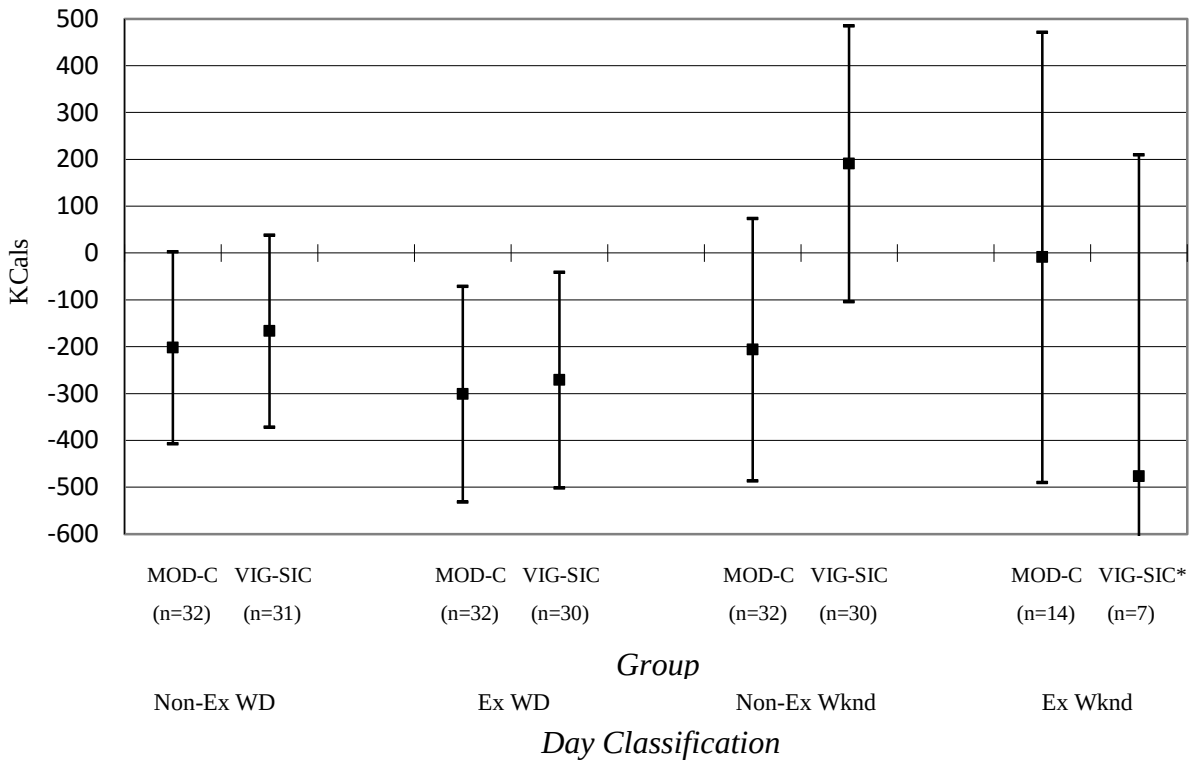

Figure 3.3. Changes in REI during exercise program. Adjusted for baseline values. REI = reported energy intake; MOD-C = moderate-intensity, continuous group; VIG-SIC = vigorous-intensity, sprint interval cycling group; Non-Ex WD = non-exercise weekday; Ex WD = exercise weekday; Non-Ex Wknd = non-exercise weekend; Ex Wknd = exercise weekend. Bars represent 95% Confidence Interval. *95% CI for VIG-SIC Ex Wknd = (-1163.7, 210.2).

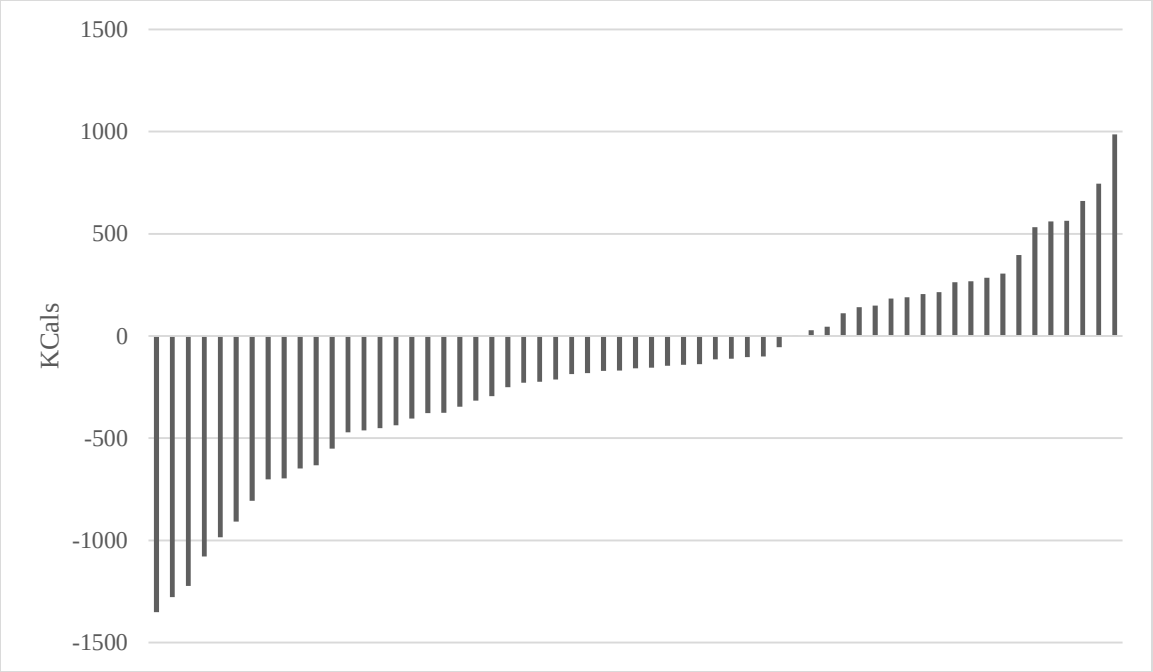

Figure 3.4. Individual weighted changes in REI during exercise program (n=61).

CHAPTER 4

ASSOCIATIONS BETWEEN COMPENSATORY AND CARDIOMETABOLIC RESPONSES TO EXERCISE IN COLLEGE-AGE FEMALES[1]

[1]Hathaway, E.D, Fedewa, M.V., Higgins, S., Evans, E.M., Schmidt, M.D. To be submitted to *Medicine & Science in Sports & Exercise*.

4.1 Abstract

Background:

Substantial inter-individual variability exists in cardiometabolic responses to exercise and both genetic and behavioral factors likely influence these differences. Potential behavioral responses to exercise include changes in diet or energy intake and non-exercise activity thermogenesis (NEAT). The purpose of this study was to explore whether compensatory changes in NEAT and reported energy intake (REI) explain inter-individual differences in cardiometabolic responses to exercise training in college-age females.

Methods:

Overweight/obese previously inactive females (n=61, 20.4 ± 1.6 y, 30.6 ± 4.9 kg/m^2, 67% Caucasian) were randomized to 6-weeks of a) continuous moderate-intensity exercise (MOD-C) or b) sprint-interval exercise (VIG-SIC). NEAT via Actiheart and REI via ASA24 were assessed at baseline and two time points during the program. Cardiometabolic markers (C-reactive protein (CRP), high-density lipoprotein (HDL), fasting glucose (GLUC), fasting insulin (INS), low-density lipoprotein (LDL), systolic blood pressure (SBP), triglycerides (TG), and waist circumference (WC)) were measured at baseline and post-study.

Results:

On average, participants maintained NEAT levels (-0.4 ± 180.2 kcal/day) and decreased REI (-177.1 ± 489.6 kcal/day). NEAT increases of 100 kcal/day were associated with reductions of 0.14 mg/dL in CRP (p=0.05), 0.74 mg/dL in GLUC (p=0.05), and 1.86 mg/dL in LDL (p=0.06).

Conclusions:

These findings do not support the hypothesis that NEAT and REI compensatory responses have a strong influence on cardiometabolic responses to exercise in previously inactive

overweight/obese college-age females. However, these associations warrant further examination in higher risk groups and studies of longer duration.

4.2 Introduction

Conventionally researchers have focused on group mean changes in health parameters in response to exercise, but only reporting group changes leads to a masking of the heterogeneity in responses by individual participants. Inter-individual variability exists not only in less than predicted weight loss by some exercise participants,[1-3] but also in cardiometabolic responses to exercise.[4] For example, data from six major exercise studies were analyzed and researchers found that 13.3% of exercise participants experienced "adverse responses" in high-density lipoprotein (HDL), 10.3% in triglycerides (TG), 12.2% in systolic blood pressure (SBP), and 8.3% in fasting insulin (INS).[4] Adverse responses were defined as "an exercise-induced change that worsens a risk factor beyond measurement error and expected day-to-day variation".[4] Thirty-one percent of participants in the six studies experienced one adverse response to exercise, 6% experienced two, and 0.8% experienced three or more.[4] In a separate 14-week exercise program with 332 adults, 6.0% of participants experienced adverse responses in SBP, 3.6% for TG, and 5.1% for HDL.[5] The prevalence of multiple adverse cardiometabolic responses in this study population was 1.2%. A better understanding of the factors influencing inter-individual variability in cardiometabolic responses (HDL, INS, SBP, TG, C-reactive protein (CRP), fasting glucose (GLUC), low-density lipoprotein (LDL), and waist circumference (WC)) induced by exercise training is needed as exercise training is often prescribed as an essential part of lifestyle change for managing these risk factors.[6]

The mechanisms which influence individual differences in cardiometabolic responses to exercise have yet to be fully explored.[7] While genetic factors undoubtedly contribute to response variations,[8] behavioral changes also likely play a role. Potential behavioral responses to exercise include changes in diet or energy intake (EI) and non-exercise activity thermogenesis

(NEAT) and several recent studies have focused on quantifying these behaviors in relation to weight loss.[2, 3] Often in these studies, only group mean changes in EI and NEAT are reported for “nonresponders” (non-achievement of predicted weight loss) versus “responders” (achievement of predicted weight loss). Manthou et al.[3] reported differences in NEAT between these two groups (nonresponders, -0.62 ± 0.39 MJ/day (-148.08 ± 93.15 kcal/day) vs. responders, +0.79 ± 0.59 MJ/day (188.69 ± 119.42 kcal/day); $p<0.05$). Similarly, King et al.[2] reported significant differences in EI between nonresponders (+268.2 ± 455.4 kcal/day) and responders (-130.0 ± 485.0 kcal/day). Not only are there different behavioral responses to exercise between individuals but also potential differences within an individual on exercise versus non-exercise days. Recently, Hopkins et al.[9] provided evidence of substantial within-subject variability in EI differences between exercise and non-exercise days in overweight/obese women that ranged from -234.3 to 278.5 kcals/day. While previous research has highlighted the substantial variability in NEAT and EI responses between individuals in exercise programs, the extent to which these behavioral responses underlie the variability in cardiometabolic responses remains unclear.

When studying those who experienced adverse cardiometabolic responses to exercise, Dalleck et. al.[5] described the possibility that other factors (eg, dietary and sedentary behavior/sitting time) might have contributed to the prevalence of adverse responses. Recently, researchers have assessed cross-sectional associations between light intensity physical activity, which is characteristic of most NEAT, and cardiometabolic outcomes in groups with equivalent levels of moderate-to-vigorous intensity physical activity.[10, 11] Loprinzi et al.[11] reported individuals with greater levels of light intensity physical activity had more desirable values for HDL, INS, and TG. Bakrania et al.[10] reported similar beneficial differences in HDL and WC

values for those with higher levels of light intensity physical activity. These cross-sectional studies highlight the potential impact of NEAT, independent of exercise, in modulating cardiometabolic outcomes.

Energy intake is also known to influence cardiometabolic markers.[12] For example, after a 7-day fast (300 kcal/day) overweight females experienced SBP reductions of -16.2 mmHg in addition to decreased LDL and INS levels.[13] For instance, Lee et al.[14] reported total calorie consumption as positively associated with TG levels and negatively associated with HDL levels. Individuals in a 2-year intervention designed to decrease caloric intake by 25% of baseline levels experienced decreases in CRP (-0.5 µg/mL), TG (-25.0 mg/dL), and SBP (-3.0 mmHG), all significantly different compared to the control group.[15] Similarly, in a 12-week energy-restriction study (1200 kcal/day) overweight/obese females experienced significant decreases in SBP (-8.5 ± 23.5 mmHg), LDL (-9.1 ± 18.7 mg/dL) and non-significant decreases in GLUC (-2.7 ± 10.7 mg/dL), TG (-10.9 ± 52.9 mg/dL).[16]

To the authors' knowledge, associations between compensatory behaviors and adverse metabolic responses to exercise have not been previously examined. Thus, the purpose of this study was to explore whether compensatory changes in NEAT and reported energy intake (REI) explain inter-individual differences in cardiometabolic responses (changes in CRP, HDL, GLUC, INS, LDL, SBP, TG, WC) to exercise training in college-age females. Our hypothesis was that compensatory decreases in NEAT and increases in REI would be associated with adverse responses in cardiometabolic values to exercise training in a dose-dependent manner.

4.3 Methods

Participants

Female students were recruited via e-mail messages sent directly to the University e-mail account for each student, and through a listserv created by the Office of the Registrar between 2014 and 2015. After completing an online survey, interested participants were contacted by researchers if they met inclusion criteria. Inclusion criteria included: female gender, enrolled student at the University, age between 18-24 years, body mass index (BMI) > 25 kg/m2, waist circumference (WC) > 88 cm, and self-report of being inactive (< 60 minutes of moderate-to-vigorous PA per week). Exclusion criteria included being: pregnant or recently (<12 months) pregnant, a current smoker (<6 months), or a varsity athlete. Individuals with a health condition that would prevent safe participation in moderate or vigorous intensity exercise or that could be exacerbated by moderate or vigorous exercise were also excluded. BMI, WC, and self-reported physical activity status were reassessed in person by researchers after the online screening. While 92 eligible students completed the screening process, five did not have access to the recreational facility where training sessions were held, 17 never enrolled, seven withdrew during the study, and two did not have complete NEAT or REI data leaving a sample size of n=61. The University Institutional Review Board approved the study protocol and informed consent document, and written informed consent was obtained from each participant prior to enrollment.

Design

Data for this investigation came from a 6-week parallel-arm design study where participants were randomized to a continuous moderate-intensity (MOD-C) or sprint-interval (VIG-SIC) exercise group after being stratified on BMI status (overweight vs. obese). Participants performed their assigned training protocol three times weekly (on Mondays,

Wednesdays, Fridays, and - if needed – a makeup weekend day) in a group-training format under the supervision of trained research staff. Training groups were matched on exercise energy expenditure (ExEE). During each exercise session, heart rate, rating of perceived exertion, and estimated EE from the cycle ergometer were recorded to ensure compliance with the training protocol. Participants were required to attend a minimum of 13 sessions (70%) to be included in the final analysis. Non-exercise activity thermogenesis (NEAT) and reported energy intake (REI) were measured at baseline (Week 0), mid-study (Week 2 or 3), and end of study (Week 5 or 6) during two weekdays (one non-exercise and one exercise day) and two weekend days. Cardiometabolic markers were measured at baseline and post-study.

Exercise Interventions

The Keiser M3 Indoor Cycle was used for all exercise sessions for both the MOD-C and VIG-SIC groups (Keiser, Fresno, California).

VIG-SIC

In the VIG-SIC group, participants began each exercise session with a warm-up, followed by repeated bouts of 30-second sprints interspersed with four minutes of active recovery pedaling against minimal resistance at a low pedal frequency. During weeks one and two, participants repeated this process to complete five sprints, which equaled 2.5 minutes of near-maximal effort sprinting interspersed with 16 minutes of recovery and a subsequent cool-down period following each session. The number of sprint repetitions progressed to six sprints during weeks three and four, and to seven sprints during weeks five and six.

MOD-C

Participants assigned to the MOD-C group were instructed to cycle continuously at an intensity of 60-70% heart rate reserve (HRR) for 20-30 minutes to match ExEE of the VIG-SIC

group, with the duration of each training session increased on a biweekly basis in order to maintain equal ExEE between the two groups. Individual HRR values were determined by a maximal graded exercise test prior to randomization. During the test, participants pedaled continuously on an electronically braked cycle ergometer (Lode Excalibur Sport 2000; Lode B.V., Groninger, Netherlands) with oxygen uptake measured using an indirect calorimetry system (ParvoMedics True Max 2400; ParvoMedics, Sandy, UT) and heart rate measured continuously throughout the test (Polar FT1; Polar Electro, Kempele, Finland).

Measures

Free-Living Energy Expenditure

Free-living energy expenditure was measured continuously by the Actiheart physical activity monitor over three separate 4-day periods (2 weekdays, 2 weekend days) (CamNtech, USA) at baseline, mid-study, and end of study with the device positioned at the level of the third intercostal space using ECG electrodes (Red Dot 2560, 3M). The Actiheart uses branched-equation modeling to estimate EE from synchronized accelerometry and heart rate data, providing precise estimates of EE in minute-by-minute epochs during a range of physical activities.[17-20] Each participant completed an Actiheart individual heart rate calibration procedure at baseline consisting of an 8 minute step test where the stepping speed ramps linearly from 15 to 33 step cycles per minute to individually calibrate the heart rate-physical activity intensity relationship.

Reported Energy Intake

Reported energy intake (REI) was measured at baseline, mid-study, and end of study using the Automated Self-Administered 24-hour Recall (ASA24), a Web-based tool modeled on the USDA's Automated Multiple Pass Method (AMPM). The ASA24 guides participants

through the completion of a 24-hour dietary recall by using an online dynamic user interface. The program then estimates total caloric intake and provides details regarding macronutrient and micronutrient intake[21] and is used for assessing the effect of interventions on diet.[22]

Anthropometric Measures

Body weight and height were collected at baseline and post exercise intervention. Standing height was measured by a stadiometer (Seca 242, SECA Corp, Hamburg, Germany) to the nearest 0.1 cm. Body weight was measured with a digital scale (Tanita WB-110A class III, Tanita Corporation, Tokyo, Japan) to the nearest 0.1 kg. BMI was calculated as weight divided by height squared (kg/m2). Waist circumference (WC) was measured twice at the umbilical waist with a flexible, tension-sensitive, non-elastic vinyl tape measure to the nearest 0.1 cm and values were averaged (Gulick, Lafayette Instrument Co, Lafayette, IN). A third WC measurement was taken if the first two measures differed by 0.5 cm or greater. Relative adiposity (%Fat) was measured via dual x-ray absorptiometry (DXA; Lunar iDXA, v 11.30.062, GE Healthcare, Madison, Wisconsin).

Cardiometabolic Measures

Fasting blood samples were obtained via venipuncture following a 12-hour fast using standard clinical procedures (Quest Diagnostics, Atlanta, GA, USA). Fasting lipid measures of high-density lipoprotein (HDL), low-density lipoprotein (LDL), and triglyceride (TG) levels, as well as fasting glucose (GLU), insulin (INS), and serum CRP were obtained. Women reporting recent illness or antibiotic use were excluded from the analysis. Systolic blood pressure (SBP) was measured while seated according to standard procedures (SunTech 247, Morrisville, North Carolina).

Computation of Explanatory Variables

Continuous minute-by-minute estimated EE data (kcal/min) from the Actiheart were used to determine overall daily activity energy expenditure (AEE). On non-exercise days, NEAT was equivalent to AEE. On exercise days, energy expended during the exercise intervention (ExEE) was subtracted from daily AEE to determine NEAT values. ExEE was determined by using the date, time, and duration of each exercise session available from records. In order to calculate changes in NEAT on exercise days, the equivalent exercise time period was removed from baseline values.

Change scores for NEAT and REI were computed as intervention values minus baseline values where a positive number corresponds to an increase in the variable of interest during the intervention. Actiheart estimates of NEAT were excluded for days having greater than 10% missing heart rate values over the continuous 24 hour-period. Based on this requirement 12.2% of daily NEAT values were excluded from analyses (non-exercise weekdays: 8.7%; exercise weekdays: 19.0%; and weekend days: 8.7%). Participants failed to complete the online diet recalls 6.3% of the time (non-exercise weekdays: 4.0%; exercise weekdays: 11.1%; and weekend days: 4.0%). No significant changes were seen between mid-study and end of study assessments so these values were averaged to compute one value for non-exercise weekdays, exercise weekdays, non-exercise weekend days, and exercise weekend days (if applicable, as participants were not required to attend the makeup weekend sessions). There were no differences in changes in NEAT and REI between exercise groups, so data were combined for further analyses. The weighted summary change score was computed for each individual as [(Non-exercise weekday x 2) + (Exercise weekday x 3) + (Weekend average x 2)]/7. Of the 63

participants that completed the study, 61 had complete weighted average REI data and 58 had complete weighted average NEAT data.

Change scores for weight, BMI, %FAT, MetS risk factors, CRP, LDL-C, and INS were calculated as post-study values minus baseline values where negative scores show improvements for all variables except HDL-C. Adverse responses were defined from change scores as follows: HDL ≤ -4 mg/dL (-0.12 mmol/L), GLUC ≥ 14.4 mg/dL (0.8 mmol/L), INS ≥ 3.5 mU/L (24 pmol/L), SBP ≥ 10 mmHg, TG ≥ 37 mg/dL (0.42 mmol/L).[4, 12]

Data Analysis

Statistical analyses were performed using SAS 9.4 (SAS Institute Inc., Cary, NC). After testing for normality and homogeneity of variances using standard procedures, group baseline and post-study values were compared using dependent T-tests. Spearman correlations were performed to examine bivariate associations between compensatory and cardiometabolic variables. Multiple linear regression was used to evaluate associations between compensatory responses (predictor variables) and changes in cardiometabolic responses (outcome variables) while adjusting for baseline values, as appropriate. Groups were created based upon compensatory responses for both NEAT and REI. Adjusted means were calculated for each group to compare cardiometabolic changes between those that engaged in positive versus negative compensatory behaviors. Groups included: NEAT Level 1 (≤-100 kcal/day, 34% of participants), NEAT Level 2 (-100 to100 kcal/day, 40% of participants), NEAT Level 3 (≥100 kcal/day, 26% of participants), REI Level 1 (≤-200 kcal/day, 43% of participants), REI Level 2 (-200 to 200 kcal/day, 36% of participants), REI Level 3 (≥200 kcal/day, 21% of participants). Statistical significance was set at an α level of .05. All data are expressed as M ± SE, unless otherwise indicated.

4.4 Results

Baseline characteristics of participants (20.4 ± 1.6y, 67% Caucasian) are presented in Table 4.1. Baseline NEAT ranged from 315.9 to 1267.7 kcal/day and baseline REI ranged from 604.5 to 3581.0 kcal/day. No baseline differences existed in cardiometabolic values between exercise groups. During the intervention, exercise energy expenditure did not differ between the MOD-C (137.1 ± 35.1 kcal/session) and VIG-SIC (140.7 ± 39.4 kcal/session) (p=0.72). Change scores are presented in Table 4.2 and changes in NEAT and REI ranged from -387.3 to 358.1 kcal/day and -1276.7 to 986.2 kcal/day, respectively. The incidence of adverse responses to exercise varied widely depending upon the cardiometabolic variable: 1.7% for GLUC, 5.2% for TG, 10.2% for INS, 21.3% for SBP, and 23.7% for HDL. Half of participants experienced one adverse response, 8.5% experienced two, and 1.7% experienced three. No differences were seen between the moderate versus high intensity groups so groups were collapsed for further analyses.

Spearman correlations are presented in Table 4.3. Greater increases in NEAT, relative to the group, were associated with greater reductions in GLUC and LDL levels. Further, multiple linear regression revealed consistent results for GLUC and LDL, in addition to CRP, when controlling for baseline values (see Table 4.4). When change in %Fat was added to the model, CRP and GLUC remained significant. No significant Spearman correlations were found between REI and cardiometabolic changes, but REI and CRP changes approached significance in the multiple regression model (see Table 4.5).

Differences in cardiometabolic changes stratified by compensatory behavior group are shown in Figure 1. The expected positive to negative cardiometabolic change trend for NEAT groups occurred for CRP (p_{trend}=0.10), GLUC (p_{trend}=0.01), INS (p_{trend}=0.13), and WC (p_{trend}=0.78). Those who increased and maintained NEAT decreased LDL levels compared to

those who decreased NEAT, but no benefit in increasing or maintaining NEAT levels was seen for HDL, SBP, TG. For changes in cardiometabolic variables by REI groups, the expected trend only occurred for CRP (p_{trend}=0.12) and HDL (p_{trend}=0.49).

4.5 Discussion

This study explored whether compensatory changes in NEAT and REI explained inter-individual differences in cardiometabolic responses to exercise training in college-age females. Although these results provide further support for the inter-individual variability in both compensatory and cardiometabolic responses to exercise, the primary finding from this study is that compensatory responses in REI and NEAT have little influence on variations in cardiometabolic responses. While significant associations were found between NEAT responses and CRP, GLUC, and LDL changes, these associations were small in magnitude, ranging from a 0.42 mg/L decrease in CRP to a 0.74 mg/dL decrease in GLUC values for a 100 kcal/day increase in NEAT.

In their secondary analysis, Yates et al.[12] reported no significant correlation between total number of adverse responses in each individual and change in ambulatory activity measured by NL-800 pedometers. It was not possible in their study to tease out the effects of non-exercise ambulation as the ambulatory physical activity measure included both exercise related ambulation and non-exercise related ambulation. The current study allowed for the removal of structured, intentional exercise conducted as part of the training intervention to compute NEAT values. Although changes in NEAT were not shown to be strongly associated with cardiometabolic changes in the current study, modest associations between a 100 kcal/day NEAT increase and changes in CRP (β=-0.42, p=0.05), GLUC (β=-0.74, p=0.05), and LDL (β=-1.86, p=0.06) were observed. As the mechanisms leading to adverse compensatory responses have yet

to be identified, compensatory responses in NEAT and REI remain plausible causal factors deserving of further study.

In the present study, number of adverse responses were tallied using HDL, GLUC, INS, SBP, and TG; thus an opportunity to have five adverse responses for each participant compared to the possibility of four in Bouchard et al.'s study[4] (HDL, INS, SBP, TG), and four in Yates et al.'s study[12] (GLUC, 2-hour glucose, HDL, TG). Half of participants in the current study experienced at least 1 adverse response, higher than the 31% reported by Bouchard et al.[4] and 41% reported by Yates et al.[12] The 10.2% of participants in this study who experienced at least two adverse responses was similar to the 7% reported by Bouchard et al.[4] While the number of adverse responses to exercise programs may seem alarming, it is important to compare these values to a control group consisting of individuals not engaging in exercise over the same time period. Yates et al.[12] did this and reported 76% of participants in the control group experienced a response at one or more of the three follow-up measurements that would have been deemed adverse if participating in exercise (52% vs. 34% at 3 months, 41% vs. 24% at 6 months, 52% vs. 31% at 12 months, in the control group vs. exercise group, respectively). Relative to control groups, it appears that individuals in exercise programs less adverse cardiometabolic responses.

Strengths

To the authors' knowledge, this study is the first to test for associations between compensatory and adverse metabolic responses. As causes of adverse responses to exercise are currently unknown, exploration of possible mechanisms is warranted.[4, 12] Strengths of the study include simultaneous measurement of NEAT and REI during two separate 4-day periods including both exercise and non-exercise days. The current study utilized a contemporary

measure of NEAT which allowed continuous 24-hour measurements. In order to compute changes in NEAT on exercise days during the intervention, the equivalent time period during structured exercise bouts was removed from the baseline values. Correcting for time spent in structured exercise is necessary, as it leads to a reduction in the time that can be dedicated to NEAT and introduces potential bias.[23] Multiple linear regression allowed for inclusion of baseline cardiometabolic values as it has been shown that participants with higher baseline values are more likely to show improvements.[24] Lastly, structured exercise was supervised throughout the study, ensuring compliance.

Limitations

Although the current study was the first of its type to quantify associations between compensatory and cardiometabolic changes induced by an exercise program, it is not without limitations. First, while an objective measure was used for the measurement of NEAT, self-report was used to assess energy intake. Measuring EI during an intervention is challenging, as some individuals underreport their true intake or may change their diet during the assessment period.[25, 26] Further, obese individuals are more likely to underreport EI[27] and overweight/obese females were specifically recruited for this study. Second, individuals were assessed at only two time points during the exercise period, in addition to the baseline period, which may have failed to capture true behavioral compensatory responses throughout the 6-week exercise program. Third, all activity energy expenditure during baseline was classified as non-exercise activity thermogenesis as participants were self-described as physically inactive. If participants actively engaged in exercise during the baseline period, this would have led to an overestimation of NEAT baseline levels and influenced change scores. Fourth, the structured exercise energy expenditure was low due to the focus on the higher intensity interval protocol,

<500 kcal/wk (~140 kcal/session), and the intervention lasted only 6 weeks. While this might be considered a limitation, this was chosen so that significant weight loss would not be the driving force in changes in cardiometabolic variables. Longer exercise durations and intervention periods may lead to greater levels of compensation which may, in turn, be more likely to influence cardiometabolic responses.[28, 29] Fifth, young and healthy female volunteers participated in the study, possibly masking associations that may exist in more diverse populations. Lastly, the relatively small sample size of this study limited the precision of the associations between compensatory and adverse responses to exercise. Larger samples sizes will be needed in future studies as variability in cardiometabolic values are influenced by a number of factors beyond structured physical activity, illness, and life events that cause stress or alterations in normal behavior.[12]

Conclusions

In conclusion, the findings of this study do not support the hypothesis that NEAT and REI compensatory responses have a strong influence on cardiometabolic responses to exercise in previously inactive college-age females. However, significant associations were found between NEAT responses and CRP and GLUC changes and while these associations were small in magnitude for GLUC, a 100 kcal/day in NEAT was associated with a 0.42 mg/dL decrease in CRP. These associations warrant further examination in studies of longer duration with higher exercise energy expenditures and more accurate EI measures.

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Table 4.1. Baseline Values of Cardiometabolic Markers, NEAT, and REI.

	All		Study Groups: Moderate Intensity		Study Groups: Vigorous Intensity		Between Group
	n	Mean(SE)	n	Mean(SE)	n	Mean(SE)	P value
BMI (kg/m^2)	56	30.58 (0.66)	29	30.33 (0.92)	27	30.84 (0.96)	*0.70*
CRP (mg/dL)	58	4.28 (0.61)	30	4.10 (0.86)	28	4.46 (0.89)	*0.77*
%FAT	56	43.98 (7.59)	29	44.38 (1.06)	27	43.56 (1.10)	*0.59*
GLUC (mg/dL)	59	87.25 (0.79)	31	87.65 (1.10)	28	86.82 (1.15)	*0.61*
HDL (mg/dL)	59	56.17 (1.97)	31	57.39 (2.74)	28	54.82 (2.88)	*0.52*
INS (mU/L)	59	10.71 (0.78)	31	11.16 (1.08)	28	10.21 (1.13)	*0.55*
LDL (mg/dL)	59	96.39 (4.55)	31	97.94 (6.33)	28	94.68 (6.66)	*0.72*
SBP (mmHg)	61	130.15 (1.58)	32	128.2 (2.17)	29	132.3 (22.28)	*0.19*
TG (mg/dL)	58	86.90 (5.65)	31	91.90 (7.74)	27	81.15 (8.29)	*0.35*
WC (cm)	59	101.02 (1.61)	31	100.2 (2.23)	28	101.9 (2.35)	*0.60*
NEAT (kcals/day)	58	614.33 (26.78)	29	572.8 (37.39)	29	655.9 (37.39)	*0.12*
REI (kcals/day)	61	1947.20 (73.26)	32	1929.3 (102.73)	29	1967.0 (106.10)	*0.75*

Table 4.2. Change Values of Cardiometabolic Markers, NEAT, and REI.

	All		Study Groups: Moderate Intensity		Study Groups: Vigorous Intensity		Between Group
	n	Mean(SE)	n	Mean(SE)	n	Mean(SE)	P value
Δ BMI (kg/m^2)	56	-0.22 (0.13)	29	-0.10 (0.18)	27	-0.35 (0.18)	*0.33*
Δ CRP (mg/dL)	58	-0.49 (0.40)	30	-0.62 (0.49)	28	-0.35 (0.51)	*0.70*
Δ %FAT	56	-0.68 (0.18)	29	-0.58 (0.26)	27	-0.80 (0.27)	*0.56*
Δ GLUC (mg/dL)	59	0.68 (0.72)	31	0.25 (0.86)	28	1.15 (0.90)	*0.47*
Δ HDL (mg/dL)	59	1.64 (0.93)	31	1.39 (1.24)	28	1.93 (1.30)	*0.14*
Δ INS (mU/L)	59	-0.23 (0.53)	31	-0.15 (0.63)	28	-0.32 (0.66)	*0.86*
Δ LDL (mg/dL)	59	-2.80 (1.79)	31	-4.90 (2.22)	28	-0.47 (2.34)	*0.18*
Δ SBP (mmHg)	61	-0.33 (1.55)	32	0.02 (1.77)	29	-0.71 (1.86)	*0.78*
Δ TG (mg/dL)	58	1.12 (3.08)	31	0.43 (4.28)	27	1.91 (4.59)	*0.81*
Δ WC (cm)	59	-1.33 (0.53)	31	-0.84 (0.72)	28	-1.87 (0.76)	*0.33*
Δ NEAT (kcals/day)	58	-0.40 (23.66)	29	-6.19 (27.36)	29	5.41(27.36)	*0.77*
Δ REI (kcals/day)	61	-177.11 (62.69)	32	-209.71 (69.65)	29	-141.13 (73.17)	*0.50*

Adjusted for baseline values.

Table 4.3. Spearman Correlations between Compensatory and Cardiometabolic Responses (ρ (p-value)).

	ΔCRP	ΔGLUC	ΔHDL	ΔINS	ΔLDL	ΔSBP	ΔTG	ΔWC
ΔNEAT	-0.18 (0.20)	-0.29 (0.04)	0.04 (0.77)	-0.23 (0.09)	-0.29 (0.04)	0.06 (0.64)	0.13 (0.37)	-0.09 (0.53)
ΔREI	0.22 (0.11)	-0.15 (0.28)	-0.09 (0.52)	-0.04 (0.78)	-0.01 (0.95)	-0.02 (0.90)	-0.03 (0.83)	-0.07 (0.62)

Adjusted for baseline cardiometabolic values.

Table 4.4. Regression Coefficients for 100 kcal increases in NEAT.

	Model 1				Model 2			
	n	β	SE β	p	n	β	SE β	p
Δ CRP (mg/dL)	53	-0.42	0.20	0.05	52	-0.41	0.20	0.04
Δ GLUC (mg/dL)	54	-0.74	0.38	0.05	53	-0.78	0.39	0.05
Δ HDL (mg/dL)	54	0.23	0.54	0.68	53	0.24	0.54	0.66
Δ INS (mU/L)	54	-0.35	0.24	0.15	53	-0.4	0.25	0.11
Δ LDL (mg/dL)	54	-1.86	0.96	0.06	53	-1.67	0.96	0.09
Δ SBP (mmHg)	56	0.64	0.77	0.41	55	0.58	0.79	0.47
Δ TG (mg/dL)	53	2.06	1.57	0.20	52	2.03	1.61	0.21
Δ WC (cm)	54	-0.18	0.32	0.58	53	-0.19	0.33	0.57

Independent models presented where each model 1 adjusted for baseline cardiometabolic variable and model 2 adjusted for baseline cardiometabolic variable and change in %Fat. Unadjusted βs presented.

Table 4.5. Regression Coefficients for 100 kcal increases in REI.

	Model 1				Model 2			
	n	β	SE β	p	n	β	SE β	p
Δ CRP (mg/dL)	56	0.14	0.07	0.07	51	0.15	0.08	0.07
Δ GLUC (mg/dL)	57	-0.10	0.13	0.45	52	-0.08	0.15	0.59
Δ HDL (mg/dL)	57	-0.06	0.19	0.76	52	-0.03	0.21	0.90
Δ INS (mU/L)	57	-0.03	0.10	0.78	52	-0.02	0.09	0.85
Δ LDL (mg/dL)	57	0.04	0.33	0.90	52	0.18	0.37	0.64
Δ SBP (mmHg)	60	-0.10	0.26	0.73	55	-0.09	0.29	0.77
Δ TG (mg/dL)	56	-0.24	0.72	0.74	51	-0.10	0.68	0.88
Δ WC (cm)	57	-0.08	0.11	0.47	52	-0.04	0.12	0.77

Independent models presented where each model 1 adjusted for baseline cardiometabolic variable and model 2 adjusted for baseline cardiometabolic variable and change in %Fat. Unadjusted βs presented.

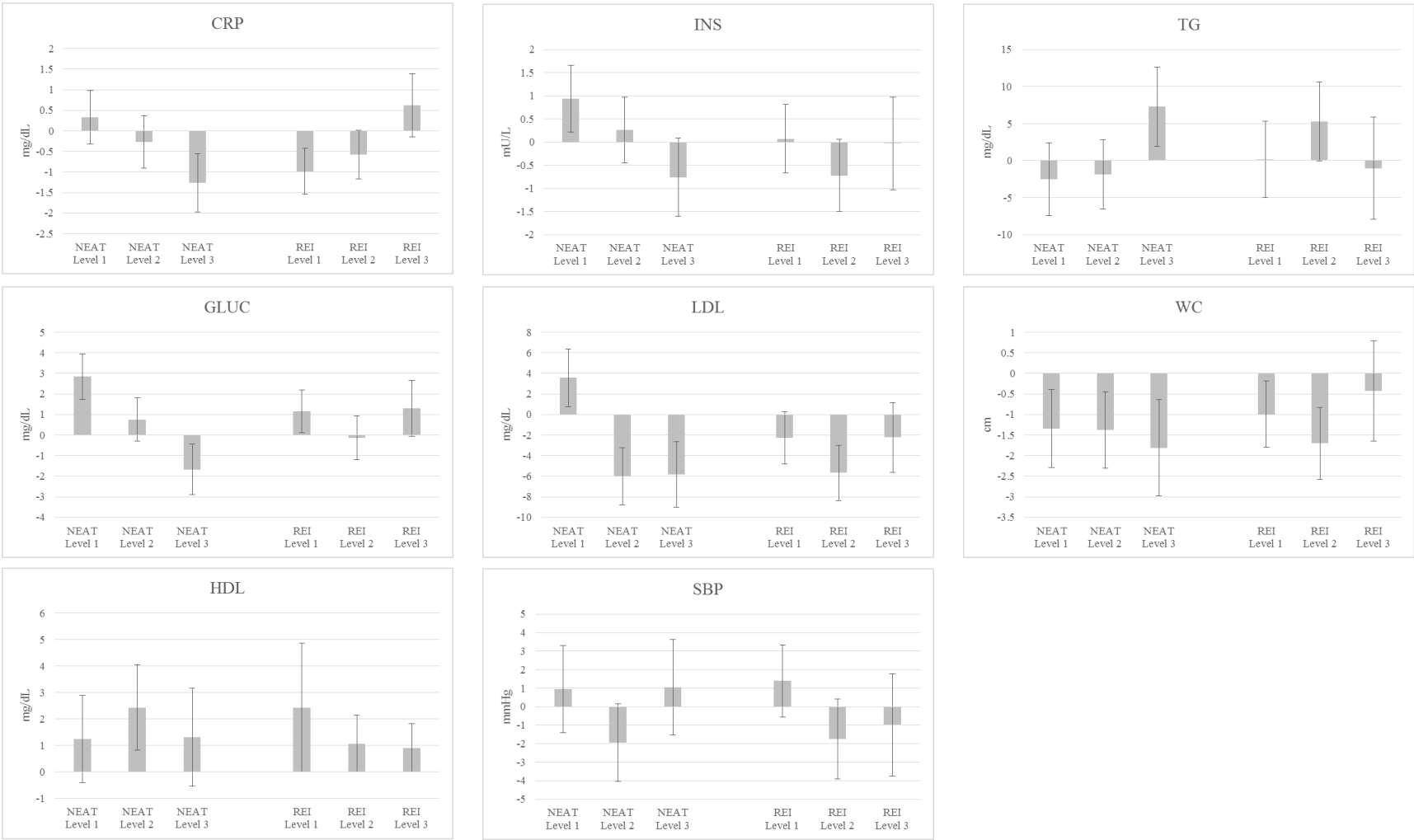

Figure 4.1. Cardiometabolic changes by compensatory grouping where NEAT Level 1 = decreases of 100 kcal/day or greater, NEAT Level 2 = within ±100 kcal/day, NEAT Level 3 = increases of 100 kcal/day or greater, REI Level 1 = decreases of 200 kcal/day or greater, REI Level 2 = within ±200 kcal/day, REI Level 3 = increases of 200 kcal/day or greater. Adjusted for baseline cardiometabolic values. Bars represent Standard Errors.

CHAPTER 5

ASSOCIATION BETWEEN EXERCISE VIEW AND CHANGES IN NON-EXERCISE ACTIVITY THERMOGENESIS AND REPORTED ENERGY INTAKE IN COLLEGE-AGE FEMALES

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5.1 Abstract

Background:

With a substantial proportion of exercise participants adopting counterproductive compensatory behaviors (i.e., increasing reported energy intake (REI) and decreasing non-exercise activity thermogenesis (NEAT)), more research is needed to better understand characteristics that distinguish compensators and non-compensators. The aim of this study was to assess the independent associations of exercise view (i.e. commitment vs. progress) and compensatory behaviors, specifically, changes in NEAT and REI. The secondary aim was to evaluate associations between psychological constructs (self-control, exercise self-efficacy, initiation, inhibition, and continuation) and changes in NEAT and REI.

Methods:

Overweight/obese previously inactive females (n=59, 20.5 ± 1.6 y, 30.7 ± 4.9 kg/m^2, 70% Caucasian) were randomized to 6-weeks of a) continuous moderate-intensity exercise (MOD-C) or b) sprint-interval exercise (VIG-SIC). NEAT and REI were assessed at baseline and two time points during the intervention. Exercise view was measured at the two time points during the study, while psychological constructs were assessed at baseline.

Results:

During exercise training, participants marginally but not significantly increased NEAT (Δ=28.3 ± 236.2 kcal/day) and decreased REI (Δ=-233.0 ± 584.5 kcal/day). Exercise view, exercise self-efficacy, initiation, inhibition, and continuation were not associated with compensatory responses (all p>0.09). Self-control was positively associated with REI changes (r = 0.30, p=0.04).

Conclusions:

No associations were observed between exercise view and NEAT or REI changes over the course of an exercise training intervention in overweight/obese college-age females. Self-control and exercise self-efficacy were also not associated with NEAT changes. Self-control was the only psychological construct significantly associated with a compensatory response, although in the opposite direction than expected.

5.2 Introduction

A goal is a future event toward which a committed endeavor is directed.[1] Actions toward goals can be represented in two distinct ways: (a) in terms of progress toward or (b) in terms of commitment to a desirable end state.[2, 3] If a person interprets the movement toward the goal in terms of general level of goal commitment, that perception likely increases motivation to continue toward similar complementary actions and hinders competing goals.[4-6] However, if the individual interprets the same movement toward a goal in terms of general level of goal progress,[7] it may serve as a reason to move away from the original goal to pursue other goals. As an example, if an individual has a goal to be healthy, after a successful exercise session, they might feel as if they have made progress toward the health goal and this might temporarily lower the likelihood of consuming healthy food at the next meal. Alternatively, the individual might view a successful exercise session as evidence of their commitment to the goal of being healthy and they will be more likely to consume healthy food at the next meal. These two dynamics—goal commitment versus goal progress—demonstrate some of the paradoxical effects of perceived goal progress on future behaviors.[2, 8-10]

The wide range of individual responses to an exercise program has been a topic of increased interest as some individuals engaging in exercise programs to lose weight end up losing a substantial amount of weight, others maintain weight, and a few actually gain weight.[11] After engaging in exercise, an individual might "compensate" by ingesting a calorically dense food or spend more time in sedentary screen-based leisure behaviors. Some individuals are predisposed to compensatory responses that render them resistant to weight loss benefits associated with an exercise-induced increase in energy expenditure[12] and may be caused by behavioral changes that accompany participation in structured exercise programs, such

as increases in energy intake (EI) or decreases in energy expenditure outside of structured exercise (known as non-exercise activity thermogenesis (NEAT)).[13, 14]

For example, noticing the great inter-individual variability in responses, Herrmann et al.[15] separated participants from the Midwest Trial 2 into nonresponders (those with weight loss <5%) and responders (≥5% weight loss). Forty-six percent of participants were classified as nonresponders. While the exercise was consistent between the two groups, NEAT increased in responders (+116 ± 456 kcal/day) and decreased in nonresponders (NEAT = -238 ± 502 kcal/day). Similarly, nonresponder EI was higher (3076 ± 967 kcal/day) compared to responders (2696 ± 603 kcal/day). A separate 12-week exercise study further revealed substantial variability in behavioral responses following exercise: responders reduced EI by 130 ± 485 kcal/day and nonresponders (51% of participants) increased EI by 268.2 ± 455.4 kcal/day.[16] In an 8-week study by Manthou et al.,[17] responders increased daily NEAT by 0.79 ± 0.50 MJ/day (188.69 ± 119.42 kcal/day) while nonresponders (68% of participants) decreased by 0.62 ± 0.39 MJ/day (148.08 ± 93.15 kcal/day). While the exercise program induced a significant increase in EI by 9.7% in the group as a whole, no differences existed between responders and nonresponders. It is clear from current research that some individuals do compensate, while others do not. With such a substantial proportion of exercise participants adopting counterproductive outside behaviors (i.e., increasing EI and decreasing NEAT), more research is needed to better understand characteristics that separate compensators and non-compensators.[18]

One possible difference between people who do and do not engage in compensatory behaviors is how exercise engagement is viewed. Perceiving exercise engagement as a commitment to health may lead an individual to continue in similar positive behaviors (e.g. eating healthy). Alternatively, exercise behaviors perceived as progress towards being healthy

may subsequently lead to the pursuit of alternative goals which may include opposing behaviors (e.g. eating and drinking while socializing with friends). Another plausible difference between those who do and do not compensate are certain psychological constructs. Disinhibition and restraint are considered importing eating behavior traits that influence weight changes and the success of weight loss interventions.[19, 20] Data by Visona & George suggest people with high disinhibition may be more susceptible to overcompensate for energy expended during exercise.[21] A recently created Trait-Self-Control Scale by Hoyle & Davisson[22] assesses three factors, inhibition, initiation, and continuation, that might be associated with compensatory behaviors.

In this context, the aim of this study was to assess the independent associations of exercise view (i.e. commitment vs. progress) and compensatory behaviors, specifically, changes in non-exercise activity thermogenesis (NEAT) and reported energy intake (REI). The secondary aim was to evaluate associations between psychological constructs (self-control, exercise self-efficacy, initiation, inhibition, and continuation) and changes in NEAT and REI.

5.3 Methods

Participants

Recruitment targeted current female college students at a large state University between February 2014 and January 2015 with e-mail messages sent directly to the university e-mail account for each student, through a listserv created by the Office of the Registrar. Additionally, recruitment posters were placed around campus. Females interested in participating completed an online screening survey and were contacted by researchers if they met inclusion criteria. Eligible participants were female, enrolled students at the University, between 18-24 years of age, had a body mass index (BMI) > 25 kg/m^2, had a waist circumference (WC) > 88 cm, and

self-reported being inactive (less than or equal to 60 minutes of moderate-to-vigorous PA each week). Females were ineligible if pregnant or recently (<12 months) pregnant, a current smoker (<6 months), or a varsity athlete. In addition, those with a health condition for which moderate or vigorous intensity exercise was unsafe or could have worsened the condition were excluded. BMI, WC, and self-reported physical activity status were reassessed in person by researchers during the baseline visit to confirm eligibility. Of the ninety-two eligible students who completed the screening process, five did not have access to the recreational facility where training sessions were held, seventeen never enrolled, seven withdrew during the study, and four did not answer the exercise view question leaving a sample size of fifty-nine. The University Institutional Review Board approved all procedures and all participants signed an approved informed consent document prior to enrollment.

Study Design

After completing the online screening form, eligible participants were invited for an in-person screening visit where informed consent was signed, online screening information was confirmed, and baseline data, including demographics, non-exercise activity thermogenesis, and reported energy intake were collected. After baseline, participants were randomly assigned to either a sprint-interval cycling group or a continuous-moderate intensity cycling group. In the sprint-interval cycling group, participants alternated 30-second sprints with four minutes of active recovery pedaling. During weeks one and two, participants repeated this process to complete five sprints, which equaled 2.5 minutes of near-maximal effort sprinting interspersed with 16 minutes of recovery. The number of sprint repetitions progressed to six sprints during weeks three and four, and to seven sprints during weeks five and six. In the continuous-moderate group, participants cycled continuously at an intensity of 60-70% heart rate reserve

(HRR) for 20-30 minutes to match the exercise energy expenditure of the sprint-interval group, with the duration of each training session increased on a biweekly basis in order to maintain equal exercise energy expenditure between the two groups. Participants in both conditions met 3 days/week over 6 weeks in a group-training format under the supervision of trained research staff. Heart rate during exercise, rating of perceived exertion, and estimated exercise energy expenditure from the cycle ergometer (Keiser, Keiser M3 Indoor Cycle, Fresno, California) were recorded during each exercise session to ensure compliance with the training protocol. The exercise groups were combined for analyses in this study as no differences existed in exercise energy expenditure, NEAT changes, or REI changes between groups. Non-exercise activity thermogenesis and reported energy intake were measured at baseline (Week 0), mid-study (Week 2 or 3), and at the end of the study (Week 5 or 6) during two weekdays (one non-exercise and one exercise day) and two weekend days. Additionally, exercise view was recorded during the mid and end of study measurement periods immediately following the exercise session.

Measures

Exercise View during Intervention

Exercise view was assessed using the PRO-Diary monitor (CamNTech, USA) by the response to the following question using an analog scale ranging from 0 (total commitment) to 100 (total progress): "*Do you view your engagement in cycling today as more a sign of commitment towards being healthy or a sign of progress towards being healthy?"* The PRO-Diary is a compact wrist-worn electronic diary used to collect participant responses via a touch sensitive "slider". On all exercise days, participants were polled immediately after exercise regarding exercise view. Participants were surveyed on each exercise day during the mid and end of study measurement periods.

Non-Exercise Activity Thermogenesis

Estimates of free-living energy expenditure (EE) were measured continuously using the Actiheart monitor (CamNtech, USA) positioned at the level of the third intercostal space using ECG electrodes (Red Dot 2560, 3M) over three separate 4-day periods (2 weekdays, 2 weekend days) (baseline, mid, and end of study). The Actiheart uses branched-equation modeling to estimate EE from synchronized accelerometry and heart rate data, providing precise estimates of EE in minute-by-minute epochs during a range of physical activities.[23-26]

Reported Energy Intake

Participants reported energy intake at baseline, mid-study, and end of study using the Automated Self-Administered 24-hour Recall (ASA24), a Web-based tool that guides participants through the completion of a 24-hour dietary recall by using an online dynamic user interface. The program then estimates total caloric intake and provides details regarding macronutrient and micronutrient intake.[27] The ASA24 has been shown to be a valuable resource in assessing the effects of interventions on diet.[28]

Anthropometric Measures

Body weight and height were collected at baseline and after the conclusion of the 6-week cycling exercise interventions. Standing height was measured by a stadiometer (Seca 242, SECA Corp, Hamburg, Germany) to the nearest 0.1 cm. Body weight was measured with a calibrated digital scale (Tanita WB-110A class III, Tanita Corporation, Tokyo, Japan) to the nearest 0.1 kg. BMI was calculated as weight divided by height squared (kg/m^2).

Psychological Construct Questionnaires

The Brief Trait Self-Control Scale (self-control), Modified Exercise Self-Efficacy Scale for College-Age Women (exercise self-efficacy), and the Trait Self-Control Scale (inhibition,

initiation, continuation) were completed by participants at baseline. In the Brief Trait Self-Control Scale, participants responded to 13 questions such as "I am good at resisting temptation" by choosing the most appropriate response from 5 choices (ranging from (1) not at all like me to (5) very much like me). Higher scores correspond to greater self-control.[29] Individuals also completed the Modified Exercise Self-Efficacy Scale for College-Age Women, a 15-item questionnaire assessing the ability to exercise in different situations with responses ranging from 0 (I cannot do it at all) to 10 (certain I can do it) with higher scores signaling greater self-efficacy.[30] Lastly, study participants answered the Hoyle & Davisson measure[22] which assesses Inhibition, Initiation, and Continuation. Participants responded to questions by choosing from a five point scale from 1 (hardly ever) to 5 (nearly always). Example questions included: "I can deny myself something I want but don't need." (inhibition); "Even when the list of things to do is long, it is easy for me to get started." (initiation); and "After I have started a challenging task, I find it easy to stick with it." (continuation). Higher scores coincided with higher levels of the trait (e.g., inhibition, initiation, continuation) being measured.

Computation of Compensatory Variables

Change scores for NEAT and REI were computed as intervention values minus baseline values where a positive number signifies an increase in the variable of interest during the intervention. On the exercise days, energy expended during the exercise intervention, determined using the date, time, and duration of each exercise session available from records, was subtracted from daily free-living energy expenditure to determine NEAT values. In order to calculate changes in NEAT on exercise days, the equivalent exercise time period was removed from baseline values. Actiheart estimates of NEAT were excluded for days having ≥ 10% missing heart rate values over the 24 hour-period. Based on this requirement 12.2% of daily

NEAT values were excluded from analyses (8.7% on non-exercise weekdays, 19.0% on exercise weekdays, and 8.7% on weekend days). Participants failed to complete the online diet recalls 6.3% of the time (4.0% on non-exercise weekdays, 11.1% on exercise weekdays, and 4.0% on weekend days).

Data Analysis

Statistical analyses were performed using SAS 9.4 (SAS Institute Inc., Cary, NC). After testing for normality and homogeneity of variances using standard procedures, Pearson correlations were performed to examine bivariate associations. Scatterplots were inspected to make sure relationships were not non-linear and there were no influential outliers. We used multiple linear regression to quantify associations between exercise view score and compensatory responses. Mid-study and end of study time points were classified as categorical variables and exercise view scores were treated as continuous variables. NEAT and REI values were the dependent variables. We also used a repeated measures ANOVA to evaluate the effects of psychological construct scores on NEAT values at baseline, mid-study, and end of study NEAT. Baseline, mid-study, and end of study NEAT values were treated as within-subjects continuous variables, while psychological construct scores were treated as between-subjects and continuous. Equivalent models were constructed using REI values. For each psychological construct, participants were stratified into groups based on percentiles where "Low" corresponded to participants with scores ≤ 25th percentile, "Mid" corresponded to participants with scores between the 25th and 75th percentile, and "High" corresponded to participants with scores ≥75th percentile to explore trends in compensatory changes across the exercise training intervention. Statistical significance was set at an α level of .05. All data are expressed as M ± SD, unless otherwise indicated.

5.4 Results

Participant baseline characteristics, including self-control, self-efficacy, inhibition, initiation, and continuation scores, are presented in Table 5.1. Exercise view scores averaged 57.9 ± 27.4 on the 0 to 100 scale where higher numbers corresponded to greater view of goal progress. NEAT values during the intervention averaged 656.9 ± 209.1 kcal/day, a slight increase from baseline values (Δ=28.3 ± 236.2 kcal/day). REI values during the intervention averaged 1749.6 ± 579.3 kcal/day, lower compared to baseline values (Δ=-233.0 ± 584.5 kcal/day). NEAT and REI changes were modestly associated ($r = 0.25$, $p=0.06$) where a beneficial response in one component was associated with an adverse compensatory response in the other. Baseline REI was negatively associated with REI change ($r = -0.57$, $p<0.01$) and baseline NEAT was negatively associated with NEAT change ($r = -0.58$, $p<0.01$) indicating higher baseline levels were associated with greater reductions during the study.

NEAT Values

Bivariate Pearson correlations revealed no statistically significant associations between NEAT changes and exercise view, self-control, exercise self-efficacy, inhibition, initiation, and continuation scores when controlling for baseline NEAT values (Table 5.2). A graphical display of NEAT values stratified by exercise view is shown in Figure 5.1. The exercise view by time interaction was not significant ($F(1,53)=0.43$, $p=0.52$). The main effect of exercise view was not significant when Baseline NEAT values were included in the model (β=-1.00, SE=0.68, $p=0.15$), or when Baseline NEAT was left out (β=-0.81, SE=0.70, $p=0.25$). Changes in NEAT over the exercise training intervention were not statistically different across the different psychological construct score categories (Figure 5.2).

REI Values

Bivariate Pearson correlations revealed no statistically significant associations between REI changes with exercise view, exercise self-efficacy, inhibition, initiation, and continuation scores when controlling for baseline REI values (Table 5.3). Self-control was positively associated with REI changes ($r = 0.30$, $p=0.04$). A graphical display of REI values stratified by exercise view is shown in Figure 5.1. The exercise view by time interaction was not significant $F(1,55)=0.01$, $p=0.94$. Regardless of Baseline REI inclusion, exercise view was not a significant predictor for REI values during the intervention [without Baseline REI: ($\beta=-0.87$, $SE=2.24$, $p=0.70$), with Baseline REI: ($\beta=-1.76$, $SE=2.10$, $p=0.41$)]. Baseline REI was a significant predictor of REI during the study ($\beta=0.45$, $SE=0.11$, $p<0.01$). Changes in REI over the exercise training intervention were not statistically different across the different psychological construct score categories (Figure 5.3).

5.5 Discussion

This study assessed the relationships between exercise view (i.e. commitment vs. progress) and changes in NEAT and REI values in overweight/obese college-age females during a six-week exercise intervention. This study also sought to evaluate associations between psychological constructs (self-control, exercise self-efficacy, initiation, inhibition, and continuation) and compensatory responses. The primary findings do not support that exercise view, exercise self-efficacy, initiation, inhibition, or continuation are significantly associated with NEAT or REI changes induced by an exercise program. Self-control was positively associated with REI changes during the exercise training intervention, meaning those with higher self-control scores, signaling greater self-control, increased REI during the study.

The question: *"Do you view your engagement in cycling today as more a sign of commitment towards being healthy or a sign of progress towards being healthy?"* was used to assess exercise view in this study using an analog scale ranging from 0 (total commitment) to 100 (total progress) and could have led to nonsignificant findings. Fishbach & Dhar assessed the potential opposite effects of commitment versus goal progress in college students by separating them into two distinct conditions, one focused on commitment framing and the other centered on goal progress framing. Those in the commitment condition were asked to indicate whether they felt committed to academic tasks after studying whereas participants in the progress condition were asked to indicate whether they felt they had made progress on their academic tasks after studying. Students in the commitment condition were not asked any question regarding progress and students in the progress condition were not asked any question regarding commitment. Fishbach & Dhar reported students that felt progress towards a goal enhanced the pursuit of alternative goals whenever the progress was deemed satisfactory, but students focusing on goal commitment enhanced further goal pursuit.[2] Splitting participants into two distinctive groups and assessing views on either commitment or progress using a "strongly disagree – strongly agree" continuum versus putting the constructs at the end of a single question (as was done in this study), might have led to similar results reported by Fishach & Dhar.

No studies to the authors' knowledge have assessed psychological characteristics of those who compensate versus those who do not, while the findings of Myers et al.[31] have yet to be published. It is plausible that just as weight maintenance and loss are associated with certain psychological constructs, compensatory behaviors would be as well. Successful weight maintenance has been associated with control of over-eating, self-monitoring of behaviors, social support, and self-efficacy whereas factors that pose a risk for weight regain are greater hunger

and disinhibited eating.[32] While disinhibition is associated with obesity, less healthy food choices, poorer success at weight loss, and weight regain after weight loss regimes,[33] inhibition was not found to be significantly associated with compensatory responses in this study and may be explained by the use of the Hoyle & Davisson survey in the present study versus the Three Factor Eating Questionnaire used in other studies. The small sample size and variability in REI/NEAT limited the ability to find associations between compensatory and psychological constructs and could have led to null results. Also, the surveys used in the present study might have failed to capture true self-control, exercise self-efficacy, initiation, inhibition, and continuation. Alternatively, there may truly be no association between these psychological constructs and compensatory responses.

Interestingly, NEAT and REI were positively correlated indicating that a positive behavior change was associated with a negative behavior change (e.g., an increase in NEAT with an increase in REI). The results from the present study are in contrast to Carraca et al.[34] who reported that exercise may serve as a 'gateway behavior' for improved eating self-regulation. Important to note though, individuals that burn more calories via NEAT could afford to increase EI compared to those who decreased NEAT. As only total calories were assessed in this study, changes in macronutrient or food types were not known. Annesi et al.[35, 36] found positive associations between changes in exercise volume and fruit and vegetable intake. It is possible that this also occurred in the study. Also of note, most participants decreased REI during the intervention. The decrease in REI observed in the current study may be due, in part, to the sample being overweight/obese and that many expressed a desire to lose weight.

Strengths

To the authors' knowledge, this is the first study to look for associations between compensatory responses and exercise view, self-control, exercise self-efficacy, inhibition, initiation, and continuation. Strengths of the study include the simultaneous measurement of NEAT, REI, and exercise view at two periods during the intervention. The current study utilized a contemporary and accurate measure of NEAT over continuous 24-hour periods, thus eliminating the potential issue of wear time differences which can arise when using other objective measures of NEAT. The PRO-Diary was also used in the present study and allowed for programming of the timing of the survey by the researcher. This allowed participants to be surveyed immediately following the exercise training bout eliminating the possibility of participants completing the assessment at a later point. Also, baseline NEAT & REI were included in models as they can strongly influence the magnitude of changes which occur during an intervention.

Limitations

The present study is not without limitations. First, the question used to assess exercise view was created for this study and has not been previously validated. While an objective measure was used to assess NEAT, self-report was used to assess energy intake. Measuring EI during an intervention is difficult, as participants may underreport their true intake or change their diet during the assessment period.[37, 38] Another limitation of the present study is that individuals were not assessed on all exercise days which could have been problematic if participants changed their behaviors when being monitored. Additionally, all activity energy expenditure at baseline was classified as NEAT based on the assumption that no intentional exercise was undertaken. If participants engaged in structured exercise at baseline then baseline

NEAT levels would have been inflated affecting change score values during the exercise training intervention. Lastly, the high degree of variability in NEAT and REI plus the relatively small sample size of this study limited the precision of the associations between compensatory and psychological constructs.

Conclusions

No associations were observed between exercise view and NEAT or REI changes over the course of an exercise training intervention in overweight/obese college-age females. Self-control and exercise self-efficacy were also not associated with NEAT changes. Self-control was the only psychological construct significantly associated with a compensatory response, although in the opposite direction than expected. Future research should increase sample size, monitor participants throughout the intervention, and further explore the relationship between self-control and compensatory responses.

5.6 Acknowledgements

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Table 5.1. Subject Baseline Characteristics.

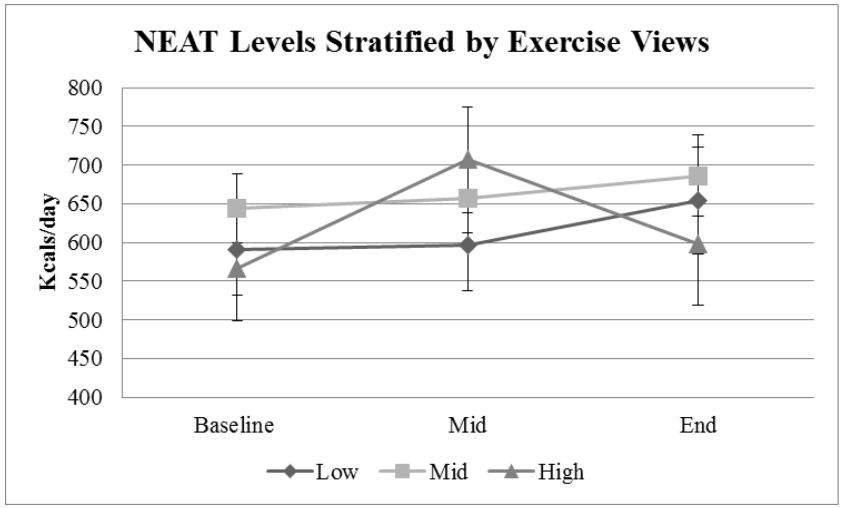
	N	Mean or %	SD
Age	59	20.5	1.6
Ethnicity			
Asian	3	5.1	
Black	9	15.3	
Hispanic	3	5.1	
White	41	69.5	
Other	3	5.1	
BMI	59	30.7	4.9
Weight (kg)	59	84.1	13.8
NEAT (kcals/day)	57	631.8	220.6
REI (kcals/day)	58	1963.6	655.7
Self-control score	48	40.6	8.4
Exercise self-efficacy score	48	5.5	1.9
Inhibition score	48	3.1	0.7
Initiation score	48	3.0	0.9
Continuation score	48	3.4	0.7

BMI = Body mass index; NEAT = non-exercise activity thermogenesis; REI = reported energy intake; Self-control score possible range from 13 to 65; Exercise self-efficacy score possible range from 0 to 10; Inhibition score possible range from 1 to 5; Initiation score possible range from 1 to 5; Continuation score possible range from 1 to 5. Higher scores for the five psychological constructs represent higher levels of that factor.

Table 5.2. Pearson Correlations between Compensatory Changes and Psychological Constructs (r (p-value))

	ΔNEAT	ΔREI
Exercise view	-0.08 (0.57)	-0.12 (0.37)
Self-control	0.07 (0.64)	0.30 (0.04)
Exercise self-efficacy	0.06 (0.72)	0.11 (0.45)
Inhibition	0.15 (0.33)	0.02 (0.91)
Initiation	0.10 (0.51)	0.25 (0.09)
Continuation	-0.02 (0.88)	0.25 (0.09)

Controlled for baseline NEAT or REI values as appropriate. NEAT = Non-exercise activity thermogenesis; REI = Reported energy intake. Higher scores for exercise view corresponds to greater view of goal progress. Higher scores for the five psychological constructs represent higher levels of that factor.



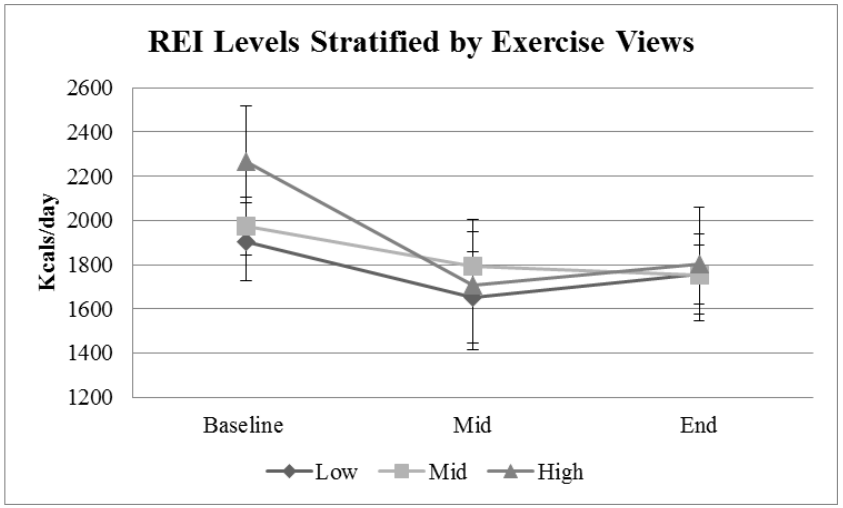


Figure 5.1. NEAT and REI values across the exercise training intervention. Participants were stratified into groups based on percentiles where "Low" corresponds to participants with scores ≤ 25th percentile, "Mid" corresponds to participants with scores between the 25th and 75th percentile, and "High" corresponds to participants with scores ≥75th percentile.

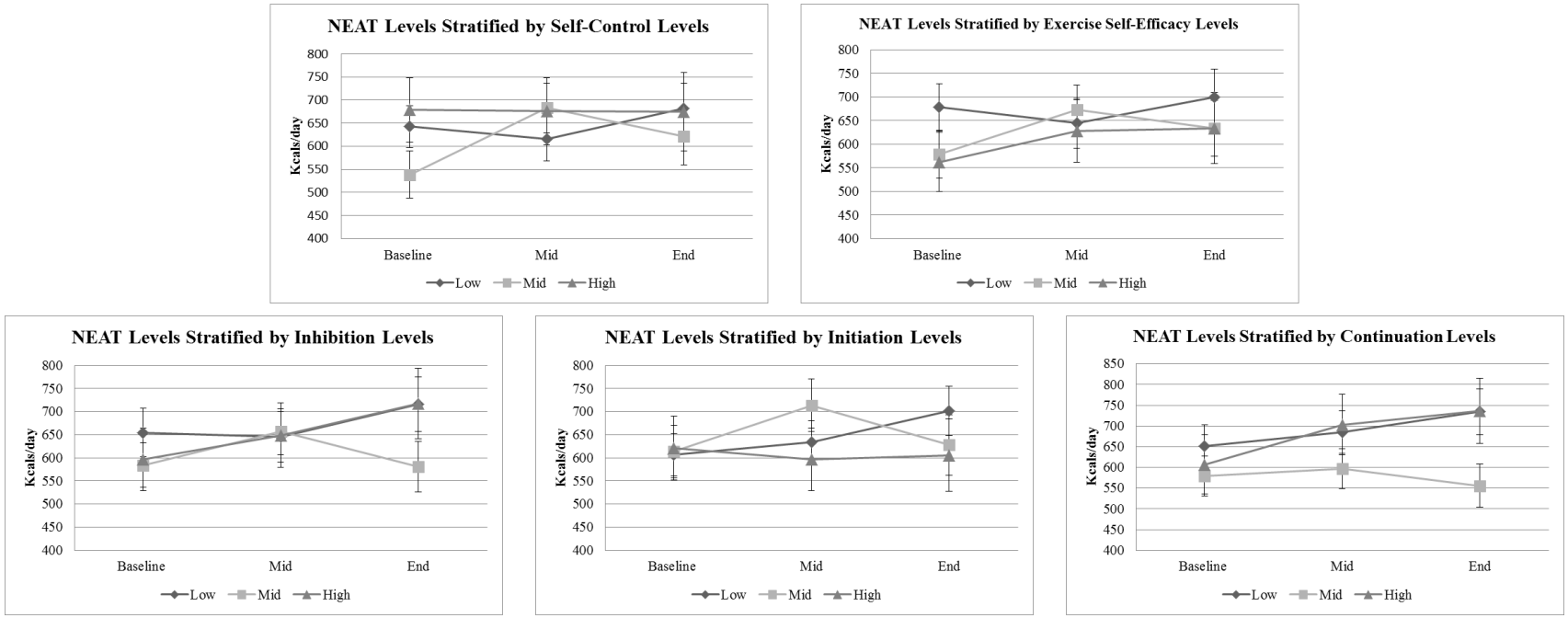


Figure 5.2. NEAT values across the exercise training intervention. For each psychological construct, participants were stratified into groups based on percentiles where "Low" corresponds to participants with scores ≤ 25th percentile, "Mid" corresponds to participants with scores between the 25th and 75th percentile, and "High" corresponds to participants with scores ≥75th percentile.

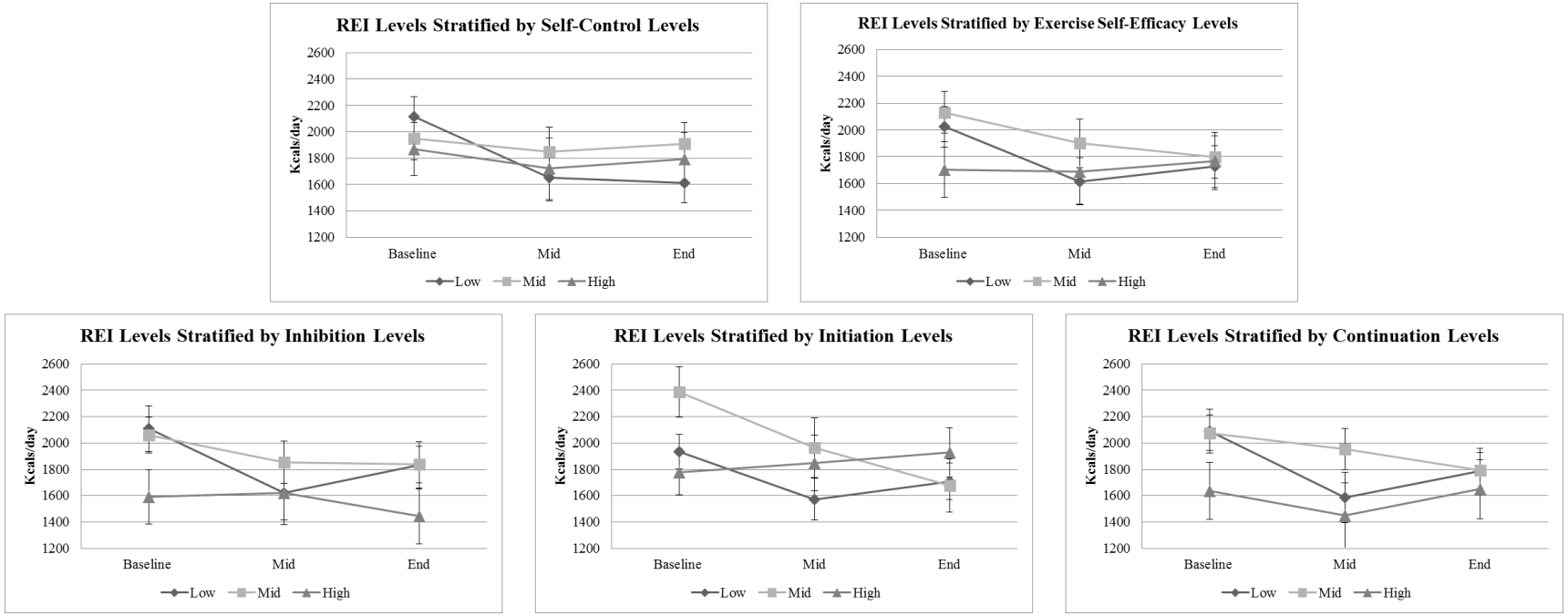


Figure 5.3. REI values across the exercise training intervention. For each psychological construct, participants were stratified into groups based on percentiles where "Low" corresponds to participants with scores ≤ 25th percentile, "Mid" corresponds to participants with scores between the 25th and 75th percentile, and "High" corresponds to participants with scores ≥75th percentile.

CHAPTER 6

SUMMARY AND CONCLUSIONS

The results from the present study add to the growing body of literature examining compensatory behavioral responses in EI and NEAT in response to structured exercise. Specifically, the effects of exercise intensity and exercise views on compensatory responses and associations between compensatory responses and cardiometabolic outcomes were examined. Strengths of the study included matched ExEE between exercise training groups, simultaneous measurement of REI and NEAT on exercise and non-exercise days at two measurement points within the intervention, and the use of objective, continuous 24-hour NEAT measurements.

Our findings indicate that exercise intensity does not influence adverse compensatory responses in overweight/obese college females. During the 6-week exercise training intervention, participants, on average, maintained NEAT values and reduced REI. No differences were found between exercise intensity training groups in NEAT or REI changes except on non-exercise weekend days where the high-intensity group increased REI while the moderate-intensity group decreased REI. These results are promising as they suggest that, in this study population, engaging in structured exercise may lead to positive health changes such as increased overall energy expenditure and cardiometabolic improvements that are not counteracted by adverse compensatory responses, although substantial variability within individuals still occurred. The results of this study suggest that either moderate- or high-intensity exercise trainings can be prescribed in this population without affecting the magnitude of negative compensatory responses.

Our results from the 6-week exercise training intervention align with previous research and provide further support for the inter-individual variability in both compensatory and cardiometabolic responses to exercise. Half of participants decreased NEAT during the exercise training intervention with 14% decreasing more than 200 kcal/day, while 34% of participants increased REI with 10% increasing more than 500 kcal/day. Further, half of participants experienced one adverse cardiometabolic response, 8.5% experienced two, and 1.7% experienced three. We found that in this study population compensatory responses in REI and NEAT have little influence on variations in cardiometabolic responses. While significant associations were found between NEAT responses and CRP and GLUC, these associations were small in magnitude.

While view of exercise as commitment versus progress and other psychological constructs were not associated with compensatory responses, higher self-control scores were associated with increased REI during the exercise training intervention, which is opposite of the expected direction. Our results will help inform the development of future interventions for overweight/obese college-age women. Specifically, educating participants about potential adverse compensatory responses is warranted as substantial inter-individual variability in responses was seen in this population. The college-setting provides a new environment away from the family-unit and its influence on health behaviors and is an ideal time to intervene to promote healthy lifestyle changes. Our findings highlight another reason to target young overweight/obese females in the college-setting as it appears that, on average, they maintain NEAT levels while decreasing EI during a short-term exercise training intervention.