REVIEW

Energy Compensation with Exercise and Dietary-Induced Weight Loss

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ABSTRACT

Obesity-related conditions are among the most expensive health care problems, accounting for $92 billion to $117 billion in annual health care costs. Obesity treatment has therefore emerged as a prime focus of health care. However, there is currently no reliable method to consistently attain and sustain weight loss as evidenced by the continually escalating prevalence of overweight/obese adults in the United States, now exceeding 70%. The difficulty many have in decreasing body weight and maintaining weight loss is largely because of a coordinated set of compensatory mechanisms the body uses to resist maintenance of an energy deficit induced by exercise or energy-restricted dieting. By working to maintain energy balance, these compensatory mechanisms represent an important barrier to many individuals' weight loss efforts. For this review, relevant publications were searched via PubMed database using the terms obesity treatment, exercise, energy compensation, compensatory mechanisms, and weight loss. This paper provides an overview of these specific compensatory mechanisms, the physiology driving them, and how different exercise modalities influence energy compensation. There are many sources of energy compensation including metabolic, hormonal, and behavioral, which may either drive eating behaviors or reduce energy expenditure when attempting weight loss. Exercise is a useful weight loss strategy only if these compensatory mechanisms do not completely abolish the negative energy balance produced by exercise. There is evidence that greater amounts of exercise can overcome these compensatory responses to produce significant weight loss without dietary intervention. *Journal of Clinical Exercise Physiology*. 2021;10(2):51–61.

Keywords: Obesity, adaptive thermogenesis, energy balance

INTRODUCTION

Obesity is one of the largest epidemics plaguing affluent societies today, with nearly 40% of the US adult population classified as obese (1). Obesity is a risk factor for some of the most serious health complications including cardiovascular disease, hypertension, certain cancers, and type 2 diabetes (2). Additionally, increased healthcare costs and loss of workplace productivity associated with obesity have placed a strain on the US economy (3,4). Obesity is attributed to a variety of factors, with lifestyle choices creating a positive energy balance (i.e., when energy consumed is greater than energy expended) cited as the largest contributor (5). In the 1950s, scientists demonstrated physical activity not only affects energy expenditure but is also the major modifiable determinant of energy intake (6). A large body of work bears proof of physical activity, often increased by leisure-time exercise training, as the main factor of nutrient energy partitioning (7–11). Energy partitioning simply refers to what becomes of macronutrients once they are absorbed. If one is engaged in regular physical activity, energy is delegated to repairing and refueling the body rather than storage as adipose tissue. (7–10,12,13). Additionally, most individuals can exercise for prolonged periods of time at intensities 2 to 16 times above resting rates of energy expenditure (14). As such, single bouts of exercise can result in 250 to 2,500 kilocalories (kcal) expended and, when repeated across days, can lead to the significant negative energy balance needed for weight loss (14–16). Therefore, energy expended through

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Conflicts of Interest and Source of Funding: The authors declare no conflicts of interest

greater physical activity or exercise and the accompanying metabolic flux are important and modifiable determinants of energy balance. This has led to many using exercise training as a cost-effective solution to reverse and prevent obesity and the resulting comorbidities. Unfortunately, weight loss in response to exercise is often much less than expected (17). Indeed, some report no changes in weight between an inactive control group and exercise group after 4 weeks of exercise (18), while others demonstrate similar weight loss between groups expending different amounts of energy through an intervention (19,20). The reason for these perplexing results is most likely caused by a phenomenon referred to energy compensation.

ENERGY COMPENSATION

One of the most important biological functions of the body is its ability to maintain homeostasis in an ever-changing environment. There are many examples of this, including maintaining acid/base balance, blood glucose, body water/electrolyte equilibrium, and hormonal regulation. Another often-overlooked regulatory process is energy homeostasis, where the human body is working to maintain energy balance. Like other acts of maintaining homeostasis, the ability to maintain energy balance can be viewed as an evolutionarily conserved mechanism, specifically in place to retain bodily energy stores to preserve reproductive function, a useful survival strategy in times of famine (21). Unfortunately, maintaining energy homeostasis is not advantageous for most individuals living in developed nations today as this process resists desired/needed weight loss of many. Compensatory responses provoked by a negative energy balance arising from exercise or prolonged energy restriction may be biological (reduced resting metabolic rate [RMR] and non-exercise activity thermogenesis) or behavioral (increased energy intake, decreased physical activity) (17,22–24). Figure 1 outlines the process of how compensatory mechanisms work to restore energy homeostasis (balance) when the human body is in an energy deficit, as is the case when attempting to lose weight.

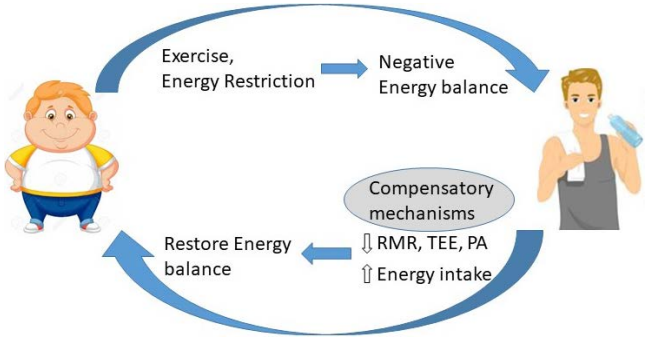

FIGURE 1. Schematic of how compensatory mechanisms, including decreases in RMR (resting metabolic rate), TEE (total energy expenditure), and PA (physical activity), and increases in energy intake, accompany an energy deficit and function to restore energy balance to resist further weight loss and subsequent weight regain.

Mechanisms for Metabolic Energy Compensation

Negative energy balance achieved through exercise or energy restriction can cause involuntary perturbations to metabolic processes that are at least partially sufficient to counter an exercise-induced or dietary-induced energy deficit. These involuntary metabolic changes include decreases in RMR, and increased skeletal muscle work efficiency resulting in less total energy expenditure (TEE) (21).

Resting Metabolic Rate

RMR is the rate at which the human body expends energy at complete rest, often conceptualized as kilocalories per 24 hours (25). RMR is the largest component (50–70%) of TEE, while fat-free mass (FFM) accounts for 60% to 70% of its variance (26). During prolonged periods of energy restriction and subsequent weight loss, the body responds by reducing RMR to conserve energy and regain energy balance (21). Decreases in serum catecholamine levels are one mechanism of RMR reduction, controlling the fraction of glucose oxidized for energy or stored in the body as glycogen or adipose tissue (27). Changes in RMR can also act as a mediating variable in the relationship between FFM and appetite (28). RMR is positively associated with FFM, meal size, and fasting levels of hunger, whereas a greater amount of FFM provokes greater energy expenditure and intake (29). This is observed in individuals with obesity who have greater amounts of FFM to support the large amounts of adipose tissue they contain, and thus are driven to consume more energy compared with nonobese individuals (29). Although the skeletal muscle of individuals with obesity is less metabolically active and contributes a different proportion to RMR than that of lean individuals (30), it has been repeatedly demonstrated that individuals with obesity have a higher RMR than their lean counterparts when controlling for FFM and thus require greater energy intakes to maintain FFM (31–34). This is a reason individuals with obesity have more difficulty tolerating energy restriction.

Skeletal Muscle Work Efficiency, Nonresting Energy Expenditure

Nonresting energy expenditure is that used as a result of physical activity or exercise. Nonvolitional reductions in nonresting energy expenditure during an energy deficit are accomplished by increasing skeletal muscle work efficiency (i.e., reducing the energy expended per unit of FFM for a given workload) (35,36). Improvements in skeletal muscle work efficiency can be the result of increasing hypothalamic-pituitary-adrenal axis activity and decreasing hypothalamic-pituitary-thyroid axis activity (21). Hypercortisolemia from increased hypothalamic-pituitary-adrenal axis activity results in reduction of FFM and more energy stored as adipose tissue (37). Attenuations in hypothalamic-pituitary-thyroid axis activity because of leptin reductions after weight loss reduce active thyroid hormone, T3 (27); these changes decrease energy expenditure by lowering heart rate, blood pressure, and muscle adenosine triphosphate (ATP)

consumption stimulated by the production of muscle adenosine triphosphatase (21).

Increasing the ability of skeletal muscle to oxidize fat over glucose is another mechanism that improves skeletal muscle work efficiency and results in weight loss (36,38). In accordance with this, maintenance of a 10% reduced body weight is associated with a roughly 20% increase in skeletal muscle efficiency, which coincides with a decrease in TEE of 9 kcal·kg^{-1} FFM per day, altering gene expression involved with lipid and carbohydrate metabolism to increase free fatty acid oxidation (35,36). The downregulation of phosphofructokinase 1 (PFK-1) and fructose-bisphosphate aldolase C (AldoC) are observed with energy-restricted weight loss, while genes involved in fatty acid oxidation such as 3-hydroxyacyl-CoA dehydrogenase (HADHsc) and fatty acid binding protein 4 (FABP4) are upregulated (38). These changes in gene expression reduce activity-induced energy expenditure attenuating further weight loss with skeletal muscle becoming efficient in using endogenous fat for fuel and less reliant on glucose (38). These skeletal muscle adaptations are beneficial, and these improvements in metabolic flexibility are especially important in alleviating insulin resistance. However, such increases in fatty acid oxidation and decreases in glucose use are an important source of metabolic compensation, making weight loss more difficult to sustain and maintain.

Mechanisms for Behavioral Energy Compensation

Behavioral compensatory mechanisms are volitional responses to an energy deficit influenced by certain neurobehavioral mechanisms (39). These compensatory mechanisms include increased energy intake and decreased voluntary physical activity, with the former being the primary mechanism responsible for maintaining energy homeostasis when exercising for weight control (24).

Increases in Energy Intake

An energy deficit influences the desire to eat through activation/deactivation of certain regions of the brain (40). With weight loss, greater energy intake in response to a negative energy balance can be caused by decreased satiation and changes in neuronal signaling in response to food (40). Brain areas that are more active in response to visual food vs nonfood cues following weight loss include areas of the limbic and reward system while parts of the brain associated with executive and decision-making functions have reduced activity (41,42). This causes the rewarding properties of food to take precedence over inhibitory control and drives eating behavior (43). Fluctuations in appetite-regulating hormones are attributed to increases in appetite during a negative energy balance, either from energy restriction or exercise (44). The *hunger hormones* are either orexigenic (ghrelin) or anorexigenic (leptin, insulin, GLP-1, pancreatic peptide, peptide YY) (45). A rise in ghrelin can cause greater appetite, whereas decreases in the anorexigenic hormones lower feelings of satiety after a meal, both of which can promote energy intake (46).

Reductions in Physical Activity

Limiting the amount of time spent doing unstructured physical activity may counter the energy expended during exercise or the negative energy balance created via energy restriction (47). Experiencing muscle soreness or fatigue after a rigorous bout of exercise or prolonged energy restriction may lead a person to engage in more sedentary behaviors such as taking the elevator instead of climbing the stairs, reducing TEE. Similarly, exercise that leaves individuals sore and fatigued may foster noncompliance to the exercise prescription and serve as another way TEE is reduced (48).

EXERCISE AND ENERGY BALANCE

Exercise Results

Exercise is a common therapy for weight loss. The American College of Sports Medicine recommends 225 minutes of moderate physical activity per week for adults seeking weight loss (49). However, exercise-induced weight loss is often much less than one would expect based on the energy expended from exercise because of the compensatory mechanisms working to maintain energy balance discussed above. Increased energy intake is commonly assumed to be the primary compensatory response when exercising to create a negative energy balance (24,50). Edholm et al. (51,52) was the first to establish a positive relationship between energy expenditure and energy intake, suggesting activity levels and energy intake form a J-shaped curved, where inactive and highly active individuals have the greatest energy intakes and where moderately active individuals have the lowest. Subsequent research has backed this claim, implying excessive exercise is a futile weight loss strategy (20,53). However, disagreement exists in the notion that greater amounts of exercise energy expenditure (ExEE) cause an equivalent increase in energy intake. A recent investigation determined groups expending 3,000 or 1,500 kcal·wk^{-1} compensate similarly (about 1,000 kcal·wk^{-1}), causing only the 3,000 kcal·wk^{-1} group to have significant weight loss after 12 weeks (54). This finding was replicated in another trial where overweight individuals exercising either 6 d·wk^{-1} (expending 2,753 kcal·wk^{-1}) or 2 d·wk^{-1} (1,490 kcal·wk^{-1}) compensated similarly, about 1,000 kcal·wk^{-1}, with only the group exercising at the greater dose experiencing significant body fat loss (55).

Some have demonstrated that exercise increases postexercise hunger and food intake (56,57). Although others have concluded single bouts of exercise do not alter circulating concentrations of hunger hormones (58–61), while chronic exercise may actually improve the satiety response to a meal (62,63), leading to reduced energy intake (64,65). Additionally, individuals with obesity often present with leptin and insulin resistance, causing lower and less-pronounced feelings of satiety. Exercise improves leptin sensitivity, promoting greater hormone/receptor binding to stimulate satiety even when decreasing concentrations of leptin (66,67). It therefore seems that additional mechanisms other than

changes in appetite-regulating hormones are responsible for greater energy intakes with exercise.

An emerging field of interest with regards to exercise and subsequent energy consumption involves investigating potential psychological mechanisms. Postexercise eating behavior can be influenced by the extent to which exercise is experienced as autonomous (enjoyable, valued) or controlled (forced, internal and external pressures) (68). Feelings about exercise have such a strong implication on food intake just reading about *tiring* physical activity leads to more snacking as opposed to reading about *fun* physical activity (69). Exercising because you *have* to rather than because you *want to* also influences eating behaviors; individuals who self-impose physical activity are more prone to consume a *food reward* after exercise compared with individuals who possess more self-determined regulation for exercise (70). In alliance with this, compared with individuals in a controlled exercise setting, individuals who have more choice over exercise mode, intensity, duration, time of session, and music played during exercise consume less energy after exercise (71). Exercise autonomy also leads to consuming less energy from *unhealthy* food choices after exercise (71). It therefore appears the notion that exercise causes compensatory increases in energy intake is multilayered and influenced by attitudes regarding the exercise bout itself.

The implications exercise has on metabolic energy expenditure is mixed. Many studies demonstrate greater postexercise oxygen consumption following single bouts of exercise can increase RMR for up to 48 hours (72–74). However, determining exercise's long-term effects on RMR is more mottled. It can be argued that a negative energy balance created from exercise would elicit reductions in RMR (metabolic compensation) if one is placed in a prolonged energy deficit. Couple this with homeostatic signals promoting overeating and you have a feedback loop primed to protect from losing body mass, abolishing the negative energy balance created through exercise (75). The change in one's RMR after aerobic and/or resistance exercise appear to depend on how long after the final exercise bout RMR is measured and if changes in FFM is statistically controlled (76). Long-term exercise studies consisting of predominantly aerobic interventions for maximizing fat loss have demonstrated significant decreases in RMR greater than would be expected from losses in FFM alone (77–79). Table 1 summarizes studies related to energy balance and weight loss.

Exercise Modalities

There are many different exercise modalities that may influence the degree of energy compensation. An investigation of how exercise mode may influence individual responses to exercise is of great interest to the health community in order to develop optimal exercise prescriptions resulting in a minimal compensatory response and therefore maximize weight loss.

High Intensity Interval Training and Compensation

High intensity interval training (HIIT) is characterized by brief, intense bouts of near maximal effort exercise performed at ≥ 80% of maximum heart rate or the equivalent

TABLE 1. Summary of research related to physical activity and energy balance.

Source	Study Design	Exercise Protocol	Study Population[a]	Primary Findings
Werle et al. (69)	Cross sectional analysis demonstrating compensatory eating after reading/thinking about engaging in physical activity	N/A	78 women, 45 men; healthy; age 38.7 ± 16 years; BMI 26.37 ± 4.78 $kg \cdot m^{-2}$	Reading about physical activity leads participants to compensate by eating more snacks and if the exercise is perceived as tiring
Fenzl et al. (70)	Randomized, 2-armed trial determining if labeling an exercise bout affects immediate postexercise food intake in individuals who self-impose exercise	20 min moderate intensity bicycle ergometer ride	45 women, 51 men; healthy; age 26.1 ± 9.4 y; recruited from a college campus	Self-imposed exercisers ate more food after exercise when the bout was labeled *fat-burning* compared with *endurance*
Beer et al. (71)	Randomized between subject yoked design investigating the role choice in exercise has on subsequent energy consumption.	30 min to 60 min aerobic training on either a bike or treadmill	38 men, 20 women; healthy; age 22 ± 4 y; BMI 23 ± 2.3 $kg \cdot m^{-2}$; VO_2 max 52.7 ± 6.4	Greater energy intake after exercise performed under the no-choice condition (2,456 ± 1,410 kj vs 1,668 ± 1,215 kj)
Johannsen et al. (78)	2-armed longitudinal design determining if diet restriction with exercise helps preserve FFM and maintain RMR.	90 $min \cdot d^{-1}$ of circuit or aerobic training for 40 wk	7 men, 9 females; obese; age 33 ± 10 y; BMI 49.4 ± 9.4 $kg \cdot m^{-2}$	% BW lost was 38 ± 8%, 83% of that being fat mass. RMR decreased out of proportion to decrease in body mass

BMI = body mass index; BW = body weight; FFM = fat free mass; kj = kilojoules; RMR = resting metabolic rate
[a]Units are means ± SD

TABLE 2. Summary of research related to HIIT and compensation.

Source	Study Design	Exercise Protocol	Study Population[a]	Primary Findings
Schubert et al. (44)	RCT, Investigating the effects of different interval training on RMR	SIT or HIIT, 4 wk	30 healthy men and women; age 28.8 ± 7.6 y	SIT protocol significantly increased RMR after 4 wk
Rivest et al. (88)	RCT, Investigating the role CRF plays in the anorexia induced by exercise	40 min high intensity running	Male Wistar rats roughly 200 g in weight	Exercised rats ate less food when injected with saline than resting animals or CRF antagonist
Alkahtani et al. (89)	2-armed crossover design, Comparing the effect of moderate and high intensity interval training on eating behavior and compensation	4 wk of HIIT (3x per week) and 4 wk of MIIT (3x per week)	10 sedentary males; age 29 ± 3.7 y; BMI 30.7 ± 3.4 $kg \cdot m^{-2}$	HIIT decreased desire to eat, liking of high fat, nonsweet food, and overall fat consumption

BMI = body mass index; CRF = corticotropin-releasing factor; HIIT = high-intensity interval training; MIIT = moderate intensity interval training; RCT = randomized controlled trial; RMR = resting metabolic rate; SIT = sprint interval training
[a]Units listed as means ± SD

VO_2 max separated by recovery periods in a work-to-rest duration of ≥ 1:1 (80). Sprint interval training is another form of HIIT and is performed at intensities equal to or superior to one's VO_2 max (81). HIIT is equally effective, or superior to, moderate intensity continuous training for improving various health variables including increasing VO_2 max (82), increased capacity for oxidative phosphorylation in skeletal muscle (83), improving insulin resistance (84), and reducing body fat mass (81,83). HIIT is associated with increased nonexercise physical activity and thus an increase in total daily energy expenditure, which may lead to less energy compensation (85,86). HIIT may also reduce the compensatory response to exercise by reducing food intake and appetite sensations after exercise because of a rise in a potent anorectic peptide called corticotrophin releasing factor (CRF) (87,88). In rats infused with a CRF antagonist, hard exercise had no effect on food intake, nor did it reduce body weight, as opposed to exercised rats without the CRF antagonist who decreased food intake and body weight (88). Among humans, exercise-induced hunger and desire to eat decreases after HIIT when compared with moderate intensity interval training (MIIT), specifically causing less wanting and consumption of high fat foods after exercise (89). These changes in macronutrient preference may be one reason HIIT elicits greater reductions in fat mass even if energy expenditure is less than or equal to MIIT (89). HIIT may also preferentially influence metabolic compensation by provoking greater postexercise oxygen consumption and thus increasing TEE (90,91). It therefore appears HIIT has an advantage over traditional moderate intensity aerobic exercise by favoring less energy compensation. However, research on HIIT and weight loss is mixed possibly because of the variability of HIIT protocols (HIIT vs sprint interval training) (81). When compared with moderate intensity exercise, HIIT requires nearly half the exercise time to burn equivalent amounts of energy (89). With leisure time shrinking in modern societies, having the ability to shorten training time while maintaining increases in energy expenditure is of great value. Future research may investigate specific variables in a HIIT program that may be modified to attenuate the compensatory response to HIIT, such as different intensities of the work intervals, durations, frequency, and individual factors such as gender, age, and training status. Table 2 presents research related to HIIT and energy compensation.

Resistance Training and Compensation

Resistance training (RT) is a form of periodic exercise whereby external weights provide progressive overload to skeletal muscles in order to make them stronger often resulting in hypertrophy (92). Most individuals envision 3 sets of high loads (> 80% max) and low (5 to 9) repetitions per set are best for increasing muscle strength, whereas lower loads (50% to 70% max) and higher (9 to 20) repetitions are best for muscular endurance (92). RT lowers blood lipids and blood pressure, promotes skeletal muscle maintenance/growth, improves blood glucose levels and insulin sensitivity, and is effective for fat mass loss (93). Because RT acts to preserve FFM during weight loss, it may eliminate or attenuate metabolic compensatory responses such as the drop in RMR often seen with energy-restriction or aerobic exercise-induced weight loss (92). Indeed, RT and protocols using both RT and aerobic training increases RMR compared with aerobic exercise alone (94). When assessing differences in compensatory increases in energy intake between RT and aerobic exercise, there appears to be a sex effect, whereas only men are more prone to compensatory eating after RT, even when controlling for ExEE (95). RT does lead to different changes in body composition compared with aerobic exercises (96), and compensatory increases in energy intake in men may have to do with the anabolic nature of RT and the subsequent gains in lean muscle mass when combined with adequate protein intake (97,98). Therefore, increases in energy intake with RT may be caused by muscle growth and repair and less to do with replenishing energy stores to maintain energy balance. Therefore, this greater energy intake would not be viewed as a traditional compensatory mechanism in the context of RT and its

TABLE 3. Summary of research related to resistance training and compensation.

Source	Study Design	Exercise Protocol	Study Population[a]	Primary Findings
Dolezal et al. (72)	RCT, Comparing changes in RMR, body fat, max aerobic power, and strength between exercise modalities	10 wk, 3 times per week AT, CT, or RT	30 physically active, healthy men; age 20.1 ± 1.6 y	Greater increases in RMR in RT and CT compared with AT. Greater decreases in body fat in CT compared with RT and AT
Cadieux et al. (95)	3-armed crossover design to evaluate the effects of exercise modality on EI, TEE, NEAT	RT, AT, and control for 4 d·wk^{-1}, 6 weeks	8 men, 8 women; healthy, sedentary; age 21.9 ± 2.6 y	When controlling for ExEE, no differences in energy compensation except in males after resistance training (1,567 ± 469; 1,255 ± 409 kcal, respectively)

AT = aerobic training; CT = concurrent training; EI = energy intake; ExEE = exercising energy expenditure; kcal = kilocalories; NEAT = non-exercise activity thermogenesis; RCT = randomized-controlled trial; RMR = resting metabolic rate; RT = resistance training; TEE = total energy expenditure
[a]Units listed as means ± SD

anabolic nature. Table 3 presents research related to RT and energy compensation.

Aerobic Training and Compensation

Aerobic exercise is continuous exercise performed at submaximal intensity and involves large groups of skeletal muscles (22). Aerobic exercise has long been prescribed to combat obesity because of the large acute energy deficit it can elicit. Despite this, the magnitude to which aerobic exercise precisely impacts energy compensation and thus weight loss is debatable and highly individualistic, with some achieving drastic weight loss and others actually gaining weight (99,100). Some indicate an acute bout of aerobic exercise has little effect on immediate energy intake (60,61), while others demonstrate acute bouts impact *hunger hormones* and alter the substrate oxidation in muscle and liver that may be correlated to the postexercise change in hunger and food intake (56,57).

Exercise Dose

Exercise dose may be another important variable influencing energy compensation and thus weight loss from an aerobic exercise intervention. A recent study demonstrated individuals expending 1,500 kcal·wk^{-1} or 3,000 kcal·wk^{-1} in aerobic exercise saw no differences in energy compensation (roughly an extra 1,000 kcal·wk^{-1}), indicating greater amounts of aerobic exercise do not produce more energy compensation. Rather, a large exercise dose is needed to overcome the compensatory response to promote significant loss in fat mass (54), which has been replicated in a separate trial where aerobic exercise expenditures of 2,753 kcal·wk^{-1} and 1,490 kcal·wk^{-1} resulted in similar energy compensation (55). This is at odds with Rosenkilde et al. (20), who demonstrated that expending either 1,800 kcal·wk^{-1} or 3,600 kcal·wk^{-1} during exercise produced nearly identical energy deficits after 12 weeks because of the greater energy compensation among the 3,600 kcal·wk^{-1} group. Results from the large Examination of Mechanisms of Exercise-Induced Weight Compensation (E-MECHANIC) study (101) offers additional insight with the high-volume group (ExEE of 20 kcal·kg^{-1} body weight) compensating significantly more than the low-volume group (eight kcal·kg^{-1} body weight); however, weight loss was greater in the 20 kcal·kg^{-1} group compared with the 8 kcal·kg^{-1} (−1.6 vs −0.4, respectively, P = 0.02). These results partially support both findings: that greater ExEEs are needed to produce weight loss, and that greater ExEE instigates greater compensation. The ExEE of E-MECANIC study participants was about 1,760 kcal·wk^{-1} and 700 kcal·wk^{-1} for the 20 kcal·kg^{-1} and 8 kcal·kg^{-1} groups respectively, much lower than the energy expenditures of participants reported in other studies (54,55). The larger dose (3,600 kcal·wk^{-1} vs 1,800 kcal·wk^{-1}) and larger differences in ExEE between groups (1,800 kcal) Rosenkilde et al. (20) used may explain some of the discrepancies. It is possible that there is a point at which greater levels of ExEE do not additionally contribute to weight loss, rather, disproportionately influence energy compensation. Future research may benefit from assessing the compensatory responses to 4,000 kcal·wk^{-1} to 5,000 kcal·wk^{-1}to investigate this possibility. Table 4 outlines the research related to aerobic exercise training and energy compensation.

CONCLUSION: TRANSLATION TO CLINICAL PRACTICE/GUIDELINES

In humans, homeostatic regulation of an energetic state is regulated by a sensory feedback system that attempts to preserve stability through the concerted amendment of both energy intake and energy expenditure. The disruption of this metabolic homeostasis is reflected by adaptations in body weight, with a positive or negative energy balance leading to weight gain or loss, respectively. Many Americans with obesity strive to maintain a negative energy balance needed for weight loss, yet the majority of these efforts lead to less-than-desirable outcomes. Unfortunately for these individuals, energy balance regulation favors defending against an energy deficit over surplus. It can be argued survival rather than sustainability is the evolutionary authority, where

TABLE 4. Summary of research related to aerobic training and compensation.

Source	Study Design	Exercise Protocol	Study Population[a]	Primary Findings
Rosenkilde et al. (20)	RCT examining effects of increasing doses of aerobic exercise on body composition, AEB, and compensation	moderate (300 kcal·d^{-1}) or high (600 kcal·d^{-1}) expenditure for 13 wk	61 males; age 20 y to 40 y; healthy, sedentary, moderately overweight	Similar body fat loss was obtained regardless of exercise dose, with the greater dose inducing a greater degree of compensation
Flack et al. (54)	2-arm randomized trial comparing compensation with ExEEs of 1,500 kcal·wk^{-1} and 3,000 kcal·wk^{-1}	Aerobic exercise expending 300 kcal or 600 kcal per exercise session, 5 d·wk^{-1}, 12 wk	10 males, 26 females; age 18 y to 49 y; sedentary; BMI 25 kg·m^{-2} to 35 kg·m^{-2}	Similar energy compensation occurs in response to both ExEEs rendering greater fat mass loss in 3,000 kcal·wk^{-1} group
Flack et al. (55)	3-arm RCT comparing compensation with exercise performed 6 d·wk^{-1} vs 2 d·wk^{-1}	Aerobic exercise performed 2 d·wk^{-1} or 6 d·wk^{-1}, 12 weeks, or sedentary control	12 males, 32 females; age 18 y to 49 y; BMI 25-35 kg·m^{-2}	Similar energy compensation occurs in response to both 6 d·wk^{-1} or 2 d·wk^{-1} protocol (2,753 kcal·wk^{-1} vs 1,490 kcal·wk^{-1}). Greater fat mass loss in 6 day, 2,753 kcal·wk^{-1} group
Martin et al. (101)	3-arm RCT comparing compensation with ExEEs of 8 kcal·kg^{-1} vs 20 kcal·kg^{-1} body weight per week	Aerobic exercise expending 8 kcal·kg^{-1} vs 20 kcal·kg^{-1} body weight per week, or sedentary control	47 males, 124 females; age 48.9 ± 15.4 y; BMI 31.5 ± 4.7 kg·m^{-2}	Significant compensation occurred in both groups, with the 20 kcal·kg^{-1} group compensating more than either 7 kcal·kg^{-1} or control. 20 kcal·wk^{-1} group lost more weight compared with 7 kcal·kg^{-1} or control groups.

AEB = accumulated energy balance; BMI = body mass index; ExEE = exercising energy expenditure; kcal = kilocalories; RCT = randomized-controlled trial

[a]Units listed as means ± SD

extended periods of energy deficit are protected by a hard-wired system that prevents starvation to promote species continuation. For most of human existence this was an instrumental system that ensured our survival; however, in the modern age of convenience, abundant energy-dense food and sedentary lifestyles, the once necessitous and rigid compensatory mechanisms are playing a role in the rising obesity trend. To be fair, our current biological makeup took millions of years to evolve, and expecting it to change in response to half a century of living in an obesogenic society is outlandish. Obesity has reached epidemic proportions along with the related comorbidities, thus identification of novel, applicable therapies to remedy the situation is imperative and will likely involve individual, environmental, and societal interventions.

Increasing energy expenditure is an intuitive way to attain a negative energy balance, commonly accomplished by increasing physical activity through exercise. Exercise comes in many forms and intensities, and it appears, at least for aerobic exercise, that total energy compensation reaches about 1,000 kcal·wk^{-1} when expending as much as 3,000 kcal·wk^{-1}, indicating greater expenditures may be needed to overcome this compensatory limit to reduce fat mass (54,55). Although there is some evidence that greater energy expenditures, beyond that of 3,000 kcal·wk^{-1}, elicit a greater compensatory response than a low dose (20). It therefore appears additional research is needed to determine the optimal dose of weekly or per session energy expenditure needed to best produce weight loss without instigating a greater compensatory response. Future research may focus on HIIT, as it appears HIIT favors less energy compensation, attenuating postexercise desire to eat, shifting macronutrient preferences, and increasing total energy expenditure by both metabolic mechanisms and nonexercise physical activity.

An interesting psychological aspect to exercise and subsequent food consumption comes in the role *choice* and *implied exertion* may play. If you give people structured choices in exercise modality, music, intensity, and duration, they are more inclined to view exercise as enjoyable and not seek food rewards after physical activity (71). The same is true when initial thoughts about an exercise protocol are more positive than negative. If someone thinks a workout will be hard and gruesome, then they are more likely to seek a reward for completing such a task. The opposite happens when they view a workout as beneficial and enjoyable (70). Future exercise for weight loss may therefore benefit from

incorporating a type of behavioral or outcome expectancy training to attenuate energy compensation and thus increase weight loss.

These findings could be of great use to clinical exercise physiologists, physicians, or other healthcare fields specializing in weight loss and using exercise as medicine. The evidence presented in this review can be used to revise specific guidelines relating to exercise dose and intensity that has not been considered as criteria to include in weight loss guidelines prior. Specifically, to overcome the ~1,000 kcal·wk^{-1} compensatory response to a new exercise program, individuals should expend roughly 3,000 kcal·wk^{-1} if weight loss is the goal. Greater energy expenditures may lead to a greater compensatory response, while lower expenditures will be more easily compensated for, thereby stalling weight loss. Higher intensity training, such as HIIT, also appears to be advantageous, as the compensatory response to this type of training is not as great. Therefore, we propose exercise intensity be considered when making weight loss recommendations. There also appears to be a few behavioral aspects that can be targeted to lessen the compensatory response and make exercise more effective for weight loss, such as how one perceives exercise (i.e., gruesome, difficult, pleasurable). It therefore appears that future exercise-for-weight-loss recommendations need not to only consider energy metabolism while exercising, but also the behavioral implications that may result from an exercise program and how they may affect various compensatory mechanisms to influence weight loss.

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