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Review Article

Energy Balance and the association between energy expenditure and dietary intake

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Abstract

Energy balance, i.e., the relationship between energy expenditure and energy intake is an important concept for human biology and health. The increasing prevalence of overweight and obesity in various populations has been attributed to a positive energy balance due to an increased energy intake or reduced energy expenditure. Despite considerable efforts, intervention programs addressing the problem of increased body weight lack efficiency. The lack of success with intervention studies trying to attenuate weight gain or accomplish weight reduction may be partially explained by compensatory responses to increased energy expenditure (exercise, lifestyle intervention) or reduced energy intake (diet). Various studies have shown that dietary intake can affect physical activity or the other way around. A negative energy balance also affects energy expenditure. Even though components contributing to energy expenditure, such as resting metabolic rate, exercise energy expenditure, non-exercise activity thermogenesis, and thermic effect of food as well as energy intake underlie a biological regulation they are also influenced by behavioural patterns. In addition, there is a complex relationship between these components in an effort to maintain energy balance. Results on compensatory behaviours in response to exercise or dietary interventions have been inconsistent but overall an increase in energy expenditure rather than decreased dietary intake seems to be a more promising approach in addressing the obesity epidemic. The purpose of this review is to highlight the complex interaction between various components contributing to energy balance in order to increase the understanding of the regulation of energy balance and thus body weight. This should be beneficial in the development of new programs or the adjustment of existing programs to address the current obesity epidemic.

INTRODUCTION

Increased body weight has been shown to be related to various adverse health outcomes, such as cardiovascular disease, diabetes, or cancer [1, 2]. In addition, it has been associated with psychological problems and affects overall quality of life [3, 4]. The current obesity epidemic is often attributed to a reduction in physical activity due to lifestyle changes and/or an increase in energy intake due to increased consumption of energy dense foods which results in a reduced satiety index [5]. Currently, the average adult in the United States obtains less than 2 minutes of vigorous-intensity physical activity per day and only 6 to 10 minutes of moderate-intensity physical activity per day [6]. Furthermore, only 25-33% of the population meet current physical activity (PA) recommendations despite various intervention programs aimed at promoting PA [7]. On the other hand, 66% of US adults are classified as either overweight or obese [8], which matches the population with insufficient PA. Nevertheless, randomized controlled trials have shown that exercise energy expenditure (EEE) affects weight loss only modestly [9] and despite considerable efforts to increase PA levels and the promotion of healthy eating patterns overweight and obesity remain a problem in many industrialized countries.

The underlying cause for weight gain is a disruption of an individual's energy balance. Energy balance is defined as equilibrium between energy intake (EI), which is the sum of energy from foods, fluids, and supplement products, and energy expenditure [10]. Total daily energy expenditure (TDEE) is the sum of energy utilized at rest (basal/resting metabolic rate, BMR/RMR), the energy required for digestion of food (thermic effect of food, TEF), and activity energy expenditure [10]. Activity energy expenditure can be further divided into the energy expended in planned physical activity or exercise (EEE), and habitual physical activities of daily living or non-exercise activity thermogenesis (NEAT) (Figure 1). While dietary intake is mainly voluntary, only activity energy expenditure can be altered substantially with behavioural adaptations [5].

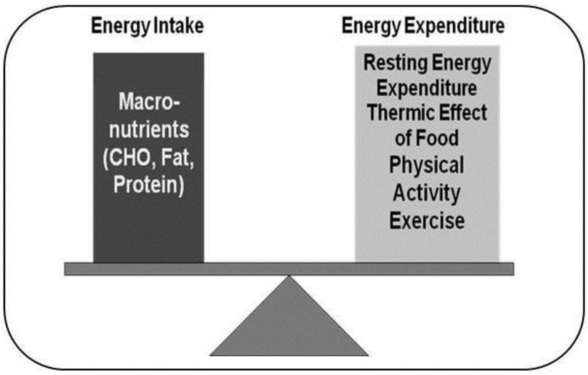

Figure 1. Components of Energy Expenditure and Energy Intake

One reason for the limited success of PA intervention studies on weight loss could be that TDEE does not necessarily increase with increased systematically planned exercise due to a compensatory reduction in habitual physical activity (NEAT) outside the intervention program [11]. Assuming such a constant TDEE, EI would be the component to consider when addressing obesity. It is also possible that increased TDEE due to physical exercise is compensated for by an increased EI, which would not result in a negative energy balance either. In addition to behavioural adaptations there is evidence of a central regulation involved in energy balance. The exact pathways through which changes in physical activity may interact with nutritional intake and energy balance, however, are not clearly understood [12]. Even though the central regulation of appetite and nutritional intake is well established [13, 14] there is a lack of understanding on the regulation of PA and TDEE and the interaction between various components contributing to energy balance. The focus of this review is primarily on behavioural aspects but the effect of endocrine and neural processes on the regulation of energy balance need to be considered as well due to their effect on behaviour.

INTERACTION OF COMPONENTS CONTRIBUTING TO TDEE

TDEE can vary considerably in a population, despite a suggested central regulation of energy expenditure [15]. For example, Wickel and Eisenmann [16] reported TDEE ranging between 33.7 kcal/kg/day and 68.9 kcal/kg/day in young adults. TDEE, however, consists of several components, which may contribute more or less to the overall variability. RMR is generally the major contributor to TDEE (60% and 75%) but the actual caloric values in relation to body weight do not differ significantly when measured under standard conditions [17]. It has also been shown that habitual PA 24 hours prior to testing does not explain the variability in RMR measurements. Prior food intake rather than habitual PA, excluding exercise, has been suggested to cause minor changes in RMR. Food intake, however, will most likely cause alterations in TEF rather than RMR, which contributes between 5% and 10% to TDEE. Based on dietary intake the variability of TEF is roughly 20% [17]. Especially protein consumption is related to changes in TEF. In addition, an increase in TEF was shown with lower temperatures [18]. Further, lean body mass influences TEF as an increase in TEF was observed during weight gain while TEF was decreased during weight loss (19). This, however, may also be attributed to changes in dietary intake. Recently, a genetic contribution to variations in TEF was found as well [17].

Another component influencing RMR is exercise (in contrast to habitual PA) due to EPOC (excess post-exercise oxygen consumption) [20]. Eliakim et al. [21] showed a 4% increase in energy expenditure with endurance training in adolescent females. In addition to EPOC the increase in lean body mass in response to exercise training potentially increases RMR and thus results in a TDEE higher than the energy expenditure attributed to exercise [22]. Interestingly TEF is also affected by exercise as Westrate & Hautvast [23] reported a 23% increase in TEF the day after a glycogen-depleting exercise. Nevertheless, this change was not significantly different compared to baseline. Increases in TDEE beyond that attributed to the exercise bout may also be do to a more active lifestyle resulting in an increase in NEAT [24]. Such an increase in NEAT in response to exercise might be of particular interest in the general population, in which NEAT contributes the majority of activity energy expenditure [17]. Considering the high variability in NEAT it has also been argued that lifestyle and cultural milieu are the prime predictors of TDEE [17] as a variety of studies showed that built environment as well as social environment influence PA [25]. Further, seasonal variability in activity energy expenditure (EEE and NEAT) needs to be considered [26] as higher activity levels are generally observed during the summer, while lower activity levels occur during the winter months [27]. Such behavioural changes and the resulting changes in TDEE may also contribute to the increased weight gain observed during the winter months [28]. Social constraints, such as holidays with increased EI, however, may contribute to this phenomenon as well. In athletes seasonal variability relates more to changes in exercise regimen due to competition or off-season, but in this population EEE, rather than alterations in NEAT are the major contributor to TDEE.

THE EFFECT OF EXERCISE INTERVENTION ON TDEE

In addition to seasonal effects, exercise interventions have been shown to affect habitual PA and accordingly TDEE. Effects, however, depend on intensity and length of the exercise intervention [12]. Hunter et al. [29] suggest that relatively high intensity exercises are more successful in inducing a negative energy balance since this can partially offset the reduction in RMR that may occur in response to a negative energy balance. On the other hand, Westerterp [30] shows in a meta-analysis that due to the relatively short duration, high intensity exercise does not have much impact on PAL (Physical Activity Level) and TDEE. Rather an increased engagement in moderate activities via higher habitual PA is suggested as major determinant of PAL. Concerning duration a longer intervention program would intuitively lead to more pronounced results. In a review, Westerterp and Plasqui [31] reported no significant changes in PAL during a 12-week resistance training intervention (2 hours per week) while a similar program over 18 weeks resulted in significantly higher PAL. Similarly, changes in PAL in response to endurance training were more pronounced with longer intervention periods [31].

Very high volume training, as observed in endurance athletes, however, resulted in a compensatory reduction of NEAT [32] and amateur athletes training for a marathon displayed a lower increase in TDEE as was the energy expenditure attributed to exercise training [33]. Two explanations are provided for such compensatory responses. First, the extremely high EEE in athletes may limit the opportunity for dietary intake adaptations, which necessitates a reduction in energy expenditure in other components to maintain energy balance. Westerterp [34] suggests an upper limit of tolerable exercise at a PAL of roughly 2.5 and a potential decline in RMR at energy expenditures above this level. Second, it can be argued that higher training intensities induce higher fatigue post-exercise, which may lead to a reduction in NEAT [35].

When examining compensatory behaviour in non-athletic samples results are less clear. Based on a review of the current literature Westerterp [24] proposes that older adults compensate for increased EEE with a reduction in NEAT, and therefore, do not increase TDEE with the implementation of exercise programs. Younger participants, on the other hand, will increase TDEE with increased EEE. Hollowell et al. [36] also showed increased TDEE in older subjects but this study was conducted in a low-active sample including overweight and obese subjects where an increase in TDEE may have been tolerated more easily. Similar results, however, were reported in older lean individuals as well [22]. These results suggest that exercise interventions will generally increase TDEE even though small compensatory adjustments in NEAT may be observed. An important differentiation between these studies and research on TDEE in athletes is that in all studies examining non-athletic populations NEAT remained the major contributor to activity energy expenditure, despite an increase EEE. In athletes EEE was the major contributor, but with PAL levels below 2.5 compensatory adaptations in response to high volume training were limited as well [37]. Individual differences in compensatory responses to exercise intervention programs, however, need to be considered and may also explain the variability of success rates of such programs [38]. Further, not only activity levels may be adjusted in response to exercise but increased EE may also affect dietary behaviour.

RELATIONSHIP BETWEEN ENERGY EXPENDITURE AND ENERGY INTAKE

Levine and Kotz [39) addressed the critical relationship between EE and EI in their review and reported reductions in habitual PA in response to a restriction in EI in humans as well as in primates and rodents. In a review, Westerterp [24] also showed that dietary restriction is related to lower TDEE. Even though it has been acknowledged that this reduction in EE is partly due to a reduction in body weight, as a result of a negative energy balance, as well as a small reduction in TEF, the major reason (~60%) of the decreased TDEE with negative energy balance has been attributed to a reduction in PA [24]. Overfeeding, on the other hand, was not shown to increase PA in the general population [24]. Levine et al. [40], however, showed that an extra energy intake of 1000 kcal/day results in compensatory changes in NEAT, including increased fidgeting and other PA of daily life. In response to increased TDEE, due to an exercise intervention, no adjustment in dietary intake has been shown during the first 2 days

[41]. After this initial phase, a compensatory increase in EI occurred, but the adaptation was only partial and incomplete. Martins et al. [42] reported a reduced response to increased EE, resulting in a negative energy balance, for up to 16 days and even after an increase in EI the compensation accounted for only 30% of energy cost of exercise. On the other hand, forcing previously active people into a more sedentary lifestyle did not result in a reduced energy expenditure, and the positive energy balance lead to weight gain [41].

As was shown for responses in habitual PA in response to exercise interventions, there is also a differentiation between compensators and non-compensators with regards to changes in appetite and food intake in response to exercise [42]. In general, it has been shown that women increased their EI in response to a 3-day exercise program, while men did not show a change in EI [42]. Differences in exercise intensity, however, need to be considered when interpreting these findings as especially immediately after vigorous exercise a significant reduction in hunger was observed, which is known as “exercise induced anorexia” [43]. A direct regulation of appetite through PA via changes in satiety hormone secretion has been suggested, where exercise can trigger physiological changes in the secretion of satiety hormones resulting in a reduction in appetite post-exercise [42]. This reduction in hunger and the accompanying energy deficit, however, did not result in a prolonged reduction in body weight in response to exercise programs. One explanation for the lack of continued weight loss is that EE is reduced after the initial weight loss due to the reduced body mass. To maintain weight loss, EI would need to be further reduced, which is not feasible over a prolonged period of time. Another aspect is that movement economy and efficiency may increase with exercise training, potentially reducing net EEE. In addition to compensatory adjustments in other components of TDEE [42], the lack of exercise induced weight change could also be explained by a change in body composition. While exercise may induce a loss of fat mass, an increase in lean body mass could occur, attenuating a decrease in total body mass. Overall it has been suggested that behavioural and metabolic compensatory mechanisms are activated in response to exercise induced energy deficits to ensure long-term energy balance [28].

BIOLOGCIAL REGULATION OF ENERGY BALANCE

Despite the emphasis of this paper on behavioural adaptations with regards to energy balance biological constraints need to be addressed as well. Levine and Kotz [39] actually argue that biological regulatory mechanisms account for 75% of variability and susceptibility to fat gain, which is a result of a disruption in energy balance. Several neural pathways have been discussed to be involved in such regulatory mechanisms and the hypothalamus is thought to be the neural centre that integrates information from various sources and compares actual values of body fat with a reference value to determine necessary adjustments via EI and/or EE [44]. Shin et al. [44] further suggest a central mechanism to regulate PA according to EI and energy stores. These authors argue that PA, especially NEAT, may serve as a crucial thermoregulatory mechanism between energy storage and dissipation, but the exact pathways remain to be determined. In addition to the hypothalamus, the caudal brainstem and the cortico-limbic systems, have been shown to play an important role in appetite control and, therefore, potentially affect energy balance [44].

Besides a central regulation, the liver has been hypothesized to monitor and respond to changes in energy availability and control of food intake. The “depletion-repletion model” hypothesizes that dietary intake is initiated when immediately available energy such as blood glucose or fatty acids fall under a certain threshold and the intake is stopped once substrate levels are sufficiently replenished [45]. Such a model, however, only explains EI but does not provide an explanation for the matching of EI with EE since key parameters related to energy depletion and repletion correlate poorly with EE [45]. To maintain long-term stability in fat stores and body weight a proper response to increased energy expenditure is necessary. Interestingly satiety peptides have been shown to alter meal size but they do not seem to affect total caloric intake. For example the administration of CCK, a peptide released from the gut associated with a reduction in meal size, was also shown to increase meal frequency in order to maintain overall energy intake [46].

The long term regulation of energy balance also suggests that the onset of eating is not necessarily tied to immediate energy needs, which is supported by the previously mentioned exercise induced anorexia. Woods et al. [45], therefore, suggest that signals proportional to fat stores become integrated with other regulators of food intake. Meal termination, for example, was not shown to be correlated with a replenishment of depleted substrates and it seems that depletion signals such as leptin deficiency, food deprivation/restriction, or ghrelin administration stimulate EI much more than satiety signals (leptin or glucose) inhibit food intake [44]. Further, it should be considered that the previously discussed appetite control mechanisms were established during periods of high energy flux and Schoeller et al. [47] actually proposed the existence of a minimum threshold concerning the turnover rate of regulating substances to detect energy imbalances. This theory of a higher energy flux in order to regulate energy balance may also help to explain why active people are better able to maintain energy balance due to an increased sensitivity of physiological satiety signals, macronutrient preferences or food choices [41]. In particular, PA at intensity levels between 60% and 70% of VO2max have been shown to alter appetite control [48-50].

The lower energy flux in modern societies may explain why biological regulation is not functioning as effectively any more to maintain energy balance, which is reflected by the increasing prevalence of overweight and obesity [51]. Shin et al. [44] also argue that a majority of the population gains less than 1 kg body weight/year and that these small disruptions in energy balance may not be detected through biological regulatory mechanisms. Another explanation is that regulatory pathways can be overridden by behavioural patterns. Additionally, it was shown that exposure to a high-energy diet will cause a change to a higher set point, which results in fat gain and an according progressive weight-gain [44]. These authors also argue that body weight or adiposity is regulated at different levels under different environmental conditions and, therefore, suggest that there is no fixed reference or threshold value set in the brain, which further undermines the role of environmental constraints on the regulation of energy balance.

The adaptability of regulatory processes in regards to energy balance is also referred to as “thrifty genotype” [52]. Chakravarthy and Booth [53] hypothesize that the cycling of fuel stores driven by cycles of feast and famine as well as PA and rest during the hunter-gatherer period, have molded the selection of “thrifty” genes. During this area PA was necessary to get food and, therefore, these two components have been linked together. Similarly Prentice and Jebb [5] argue that during the agrarian time an energy deficit occurred during planting, which was later compensated for by an energy surplus during harvest, resulting in an overall energy balance. In addition to genetic variability, the intra-uterine environment [54] as well as extrinsic factors like environmental conditions, peer influences and personal desires could result in a change in set points for EE or EI [55]. Therefore, it has been stated that genes and behaviour need to be incorporated in a single framework even though they function at different levels [56].

The behavioural or environmental influence on long-term coupling between EI and EE is also indicated by an improved regulation of energy balance with regular exercise [57]. The quicker adjustments in response to changes in energy expenditure due to exercise have been shown to be predominantly related to adjustments between meals [42]. If EI is reduced in one meal, there seems to be a compensatory increase in intake during the following meal in more active people. Long et al. [58] supported the role of exercise in ‘fine tuning’ physiological mechanisms to regulate dietary intake by showing a better compensatory response when manipulating preload energy content before an actual meal. An increased sensitivity was reported for up to 24 hours, but this coupling between EI and EE was only shown with higher energy expenditure levels.

CONCLUDING REMARKS

In summary, energy balance needs to be considered as a complex bio-behavioral phenomenon that is influenced by genetic, physiologic, early-life, and demographic-environmental constraints [5]. Energy homeostasis is accomplished through a highly integrated and redundant neurohumoral system that tries to minimize short-term fluctuations in energy balance and fat mass by proper adjustments of energy expenditure and energy intake. If these fluctuations, however, occur within certain thresholds no change may be sensed, and overall a gradual up-regulation of body fat stores is possible [45]. In more active people the sensitivity to changes in EE or EI seems to be improved and even though short, high-intensity exercise bouts may not result in a significant increase in TDEE such exercises may help in the regulation of energy balance. Nevertheless, the incorporation of moderate-intensity PA, such as active commuting to work, along with a reduction in snack consumption may have a stronger influence on longterm energy balance [59]. To address the growing problems associated with an energy dysbalance, more research on the complex relationships between biological and behavioural factors influencing EI and EE is needed. It needs to be emphasized that research on the problem of overweight or obesity should not simply consider how a particular intervention affects a single component of the energy balance equation, but rather address the overall outcome on the entire energy balance equation [28]. Only with an increased understanding of the interaction and regulation of overall energy balance successful interventions on weight management can be established.

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Appendix. List of abbreviations used.

Abbreviation	Open Spelling
BMR	Basal Metabolic Rate
EE	Energy Expenditure
EEE	Exercise Energy Expenditure
EI	Energy Intake
EPOC	Excess post-exercise oxygen consumption
NEAT	Non-Exercise Activity Thermogenesis (i.e. habitual physical activity)
PA	Physical Activity
PAL	Physical Activity Level (PAL = TDEE / RMR)
RMR	Resting Metabolic Rate
TDEE	Total Daily Energy Expenditure
TEF	Thermic Effect of Food (i.e. energy required for food processing)