Physical Activity, Energy Intake, and Obesity: The Links Between Exercise and Appetite

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Abstract Overweight and obesity are often theorized using the energy balance equation. When energy intake (EI) is greater than energy expended this creates a positive energy balance and leads to weight gain, conversely when energy expenditure (EE) exceeds EI this creates a negative energy balance and weight loss. Physiologists have been examining the relationship between EE and EI since the 1950s. Total daily EE is comprised of two main components; resting metabolic rate (RMR) and physical activity (PA). This review will examine and report the relationships between both major components of EE and EI, and therefore the impact of PA on body weight. The relationship between energy expended through PA and EI appears to be weak, especially within one day and over longer periods in overweight and obese adults. However, recent data have demonstrated a strong relationship between RMR, hunger and food intake. The differences observed in the relationships between PA EE and EI and metabolic EE and EI are not surprising. EE

from PA is highly variable and episodic whereas EE from RMR is very stable and tonic. These new data provide evidence for a fundamental link between EE and EI.

Keywords Physical activity · Appetite · Energy balance · Energy intake · Obesity · Exercise

Introduction

Exercise is frequently prescribed as a method of weight control and weight reduction. Exercise is prescribed with the primary aim of substantially increasing energy expenditure to produce an energy deficit and generating a number of health benefits such as increased cardiovascular fitness and reduced blood pressure. However, the weight loss observed following a program of exercise is often lower than would be expected. This lower than predicted weight loss could be caused by a number of different factors, such as; low exercise induced energy expenditure, compliance to the intervention and compensation for the increased energy expenditure through increasing food intake or reducing non-exercise activity (NEAT). Furthermore, in addition to the impact on energy balance, exercise also influences the control of food intake. Using recent evidence this review will provide an overview of the efficacy of exercise at producing a negative energy balance and resultant weight loss. It will discuss the evidence examining a gender difference in body weight loss in response to exercise and also highlight the importance of individual variability. In light of the adaptive nature of the energy balance system this review will also discuss the effects of increasing energy expenditure through exercise on hunger, food intake and appetite regulation both in the acute state and over a longer duration. Therefore this review will examine the fundamental relationship between energy expenditure and energy intake, it

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will include the relationship between metabolic energy expenditure and energy expended through exercise.

Exercise as a Weight Loss Tool

The overestimation of weight loss through the use of the 3500 kcal per pound rule has led to expectations of higher than achievable weight loss through exercise and dietary interventions. This rule which was derived from the energy content of a pound (Lb) of weight loss (including fat mass and fat free mass) ignores the adaptive physiological changes in metabolism that occur with weight loss. These metabolic changes include a decrease in resting metabolism and a decrease in the energy cost of physical activity [1•]. Furthermore, the 3500 kcal per pound rule does not take into account changes in body composition. The conversion of body fat and fat free tissue has very different energy values; the energy value of 1 kg of fat mass is 39.5 MJ and 7.6 MJ per kg of fat free mass [2]. Therefore to lose the same mass of fat as lean tissue would require a five times greater deficit in net energy. A new formulation that takes these factors into account has been developed [1•]. It is therefore important to assess the outcomes of weight loss interventions against more realistic changes in body composition and the overall improvement in health rather than weight loss.

The conventional position on the impact of exercise in reducing obesity is that exercise is ineffective for weight loss due to automatic compensation for the energy expended. Moreover, exercise regimes without an accompanying dietary intervention are often reported to produce only a modest weight loss. For example Thorogood and colleagues performed a systematic review which included 14 studies investigating the efficacy of exercise to produce weight loss. The review concluded that the value of exercise as a weight loss tool in overweight and obese participants was limited [3]. However, not one of the studies included in the review met the current guidelines for the amount of exercise required for weight loss (300 min per week [4]). In fact the mean exercise prescription was 166 minutes per week at a moderate to low intensity which is the recommendation for weight maintenance and improving cardiovascular fitness [5•]. This prescription of 166 min/wk at low to moderate intensity would only be expected to elicit a small energy expenditure and therefore the resultant anticipated weight loss would also be small. Furthermore, many of the studies investigating the impact of exercise on body weight are based on unsupervised exercise prescriptions and self report data. Therefore, it is likely that compliance to the exercise regime is a major factor in the success of most interventions. The efficacy of exercise to produce weight loss is based upon the energy deficit created by the exercise. A recent review concluded that the small magnitude of weight loss observed with exercise is primarily

due to low doses of prescribed exercise energy expenditure [6]. Randomized controlled trials have clearly demonstrated a positive dose response relationship between the amount of exercise completed and the amount of weight and fat lost [7, 8]. Therefore, if the exercise is supervised and a large amount of energy is expended exercise can produce a significant weight loss in overweight and obese individuals [9, 10, 11••].

In addition to the belief that exercise alone produces a modest weight loss there is also a prevailing view that women do not lose as much weight as men in response to exercise [12]. The belief that women respond less successfully to exercise than men is based on previous research demonstrating that lean women lose less body weight in response to exercise than lean men [13]. Further studies have also supported this view [14]. In this meta-analysis the energy expenditure through exercise in men was twice that of women per exercise session. Therefore any difference observed in the changes in fat mass loss could be attributed to a difference in energy expenditure rather than sex. In fact when exercise is closely monitored so that the energy expenditure for men and women are similar, then no sex based differences in the body weight loss response to exercise are observed [7, 15–17]. These data indicate that women do not respond less well to exercise than men and they do not show a greater compensatory response to an increase in energy expenditure.

Exercise and Appetite Response

Compensation to an increase in energy expenditure through physical activity is often thought to account for the modest weight loss associated with exercise. However, the research examining the effects of exercise on appetite and food intake, especially over the short term, creates a different picture. The acute effects of exercise on appetite appear clear and well understood. The majority of research indicates that an acute bout of exercise does not create an increase in hunger, desire to eat or energy intake [18–21]. In fact if the exercise is high intensity a transient reduction in hunger is observed which may be mediated by the decrease in the active component of the orexigenic hormone ghrelin [22–26]. Although, when the exercise is extended over a few days and EI is monitored for a longer period partial compensation for the increase in energy expenditure has been documented [27, 28].

Long term studies examining exercise and EI which have monitored EI for between 16 weeks and 18 months in overweight men and women have also demonstrated no significant change in EI across the intervention [29–31]. However, the exercise sessions are typically unsupervised and the energy expended low, which may explain the lack of compensation. Nevertheless, even with supervised exercise

sessions and an energy deficit of approximately 2000 kcal/wk for 16 months, no compensatory increase in energy intake was observed [32]. These data suggest that even in the long term there appears to be no automatic increase in EI to compensate for an exercise induced energy deficit in overweight and obese individuals. In contrast increases in EI in response to long term exercise have been reported in lean participants [33–35]. Therefore, one hypothesis is that lean individuals may demonstrate a compensatory response to exercise in order to preserve their fat free mass.

It is also important to consider that the majority of research report mean changes in body weight, composition and appetite and food intake response to exercise. This may disguise other trends in the data such as individual variability. For example even when there is a fixed and large exercise induced energy expenditure and the exercise is supervised large individual variability in body composition response to exercise has been demonstrated [36]. This variability has been shown to be partly accounted for by appetite responses to exercise. Those individuals who did not lose as much weight as predicted experienced an increase in AUC hunger levels and objectively measured food intake, whereas, those individuals who lost the predicted amount of weight did not demonstrate a change in hunger levels or food intake [11•, 16, 36, 37]. It should be noted however, that even those individuals who achieved lower than anticipated changes in body composition and body weight still demonstrated significant and meaningful health benefits including reductions in waist circumference, blood pressure and resting heart rate and improvements in cardio vascular fitness [38]. Obesity and BMI are frequently associated with an increased risk of morbidity and mortality risk [39]. However, it has been documented that the higher risk of all cause mortality associated with obesity and metabolic syndrome can be explained by differences in cardiovascular fitness in both adults [40–42], and older adults [43]. Therefore, improvements in health markers including cardiovascular fitness may be more important than changes in body weight or BMI in reducing the risk factors for obesity co morbidities.

Physical Activity and Appetite Regulation

In addition to exercise influencing food intake in a compensatory manner there is emerging evidence that exercise can sensitize the appetite regulatory system. Regular exercisers are better able to compensate for a high energy pre load at subsequent meals than their sedentary counterparts [44, 45]. Moreover, when sedentary individuals take part in regular aerobic exercise they demonstrate an increase in post prandial satiety signaling in response to a fixed meal [11•, 46, 47]. However, this is not observed following 12 weeks of resistance training [47]. The explanations for the different

observed outcomes between aerobic and resistance exercise on appetite control are not understood. The aforementioned research is the first study to examine the impact of medium term resistance exercise on appetite. The authors speculate that the contrasting effects between aerobic and resistance exercise may arise from differences in insulin sensitivity. Aerobic exercise significantly improved insulin sensitivity whereas there was no significant increase in insulin sensitivity following the resistance training program. However, more research is required into the impact of resistance training on appetite control.

In addition to the increase in satiety response to food exercise also exerts an increase in fasting hunger as a compensatory response [11•, 16]. Therefore, the impact of aerobic exercise on appetite comprises two processes: an increase in the orexigenic drive that is counteracted by an increase in the satiety effects of food. The second process appears to be mediated through changes in either the release of or sensitivity to episodic satiety peptides [48, 49].

Overall, there appears to be a weak relationship between energy expended through physical activity and energy intake, especially within one day. This relationship is unsurprising as the energy expenditure from exercise is episodic and can vary greatly from day to day. This relationship between physical activity energy expenditure and appetite are likely to be quite different from the relationship between metabolic energy expenditure and appetite. Energy expenditure from resting metabolic rate is more uniform and tonic.

Impact of Body Composition on Appetite Control

The exact mechanisms that provide control over food intake and appetite are not clear. However, there has been a focus on the relationship between fat mass and appetite regulation. These investigations were initiated in the 1990s with the discovery of the adipose derived hormone leptin. Several adipocentric models of appetite regulation have been proposed. Adipose tissue was described as a major factor in influencing energy balance through the proportional release of leptin as a marker of the body's energy stores [50]. In addition to the adipostatic control over food intake, there is growing evidence to suggest a proteinostatic mechanism in the control of food intake [51]. This theory has been strengthened by the new link between fat free mass, appetite and energy intake. We have demonstrated that it is FFM that is associated with meal size and daily energy intake, and not fat mass or BMI in overweight and obese adults [52]. This relationship appears to be mediated through resting metabolic rate. FFM is the largest component of RMR and RMR is the largest component of daily energy expenditure. In addition to FFM influencing meal size and daily EI, it has also been shown that RMR significantly predicts meal size

and daily EI [53••]. It appears that RMR exerts its action through hunger. Hunger profiles and fasting hunger levels are influenced by RMR, those individuals in the highest tertile of resting metabolisms demonstrate greater hunger compared to individuals within the lowest tertile of RMR [53••]. The interpretation of these data are that RMR as a major component of energy expenditure is exerting a physiological drive for food intake [54••]. These data are consistent with Edholm's hypothesis that there is a fundamental relationship between energy expenditure and energy intake [55].

New Model of Appetite Control

New evidence demonstrating that FFM through RMR is strongly associated with hunger and food intake supports the notion that they contribute to the tonic drive to eat. Previous models of appetite have demonstrated that the GI peptides and adipose tissue through leptin are responsible for the inhibition of food intake. Taken together it can be proposed that the physiological control of appetite comprises three components: a tonic drive for energy driven by energy expenditure; tonic inhibition from energy stores and episodic signals generated from the mouth and GI tract in response to food (see Fig. 1). However, as fat mass increases and leptin resistance develops the tonic inhibition is diminished leading to poorer appetite control, overconsumption and further weight gain. Long term regular exercise can increase the sensitivity of appetite control. One mechanism

is through the action of post prandial satiety signaling [46]. However, the influence that exercise exerts over body composition is also likely to play an important role in the increased sensitivity of appetite control demonstrated with long term exercise. Exercise reduces body fat whilst maintaining FFM therefore reducing leptin levels but also improving leptin sensitivity [56]. Therefore exercise may also improve appetite control by increasing the inhibition of food intake through restoring leptin sensitivity via reducing fat mass and improving insulin sensitivity [57].

Conclusions

In conclusion, the link between exercise and appetite is not simple. In the short term, the impact of exercise on appetite and energy intake is weak. With regular exercise, partial energy compensation through an increase in food intake is observed in some individuals. These individual differences are not due to differences between men and women, but can be partly accounted for by appetitive mechanisms. Regular exercise has an impact on body composition and metabolic turnover, and a strong relationship exists between metabolic energy expenditure, appetite and food intake. Nevertheless, exercise may also improve the sensitivity of the appetite system in the long term. Therefore, exercise can be used as a successful method of weight control. The degree of success depends on whether the exercise is completed fully as prescribed; if the prescription involves a sufficient

Fig. 1 A new formulation for daily appetite control; influence on hunger profiles. There is a tonic drive to eat that reflects the body's basic energy requirements from FFM which exerts its effect through RMR. There is also a tonic inhibition of the drive to eat that reflects the body's energy stores which exerts its effect through leptin. Alongside

the tonic inhibition and drive there are also episodic inhibition and drive mainly from the GI peptides. Episodic satiety signaling following a meal dampens the expression of hunger. When the satiety effects have worn off the drive from RMR re-exerts its effect on hunger levels increasing the drive to eat and food is consumed

amount of energy expenditure; and whether the exercise program is implemented in the long term. Finally, it is important to note that the health benefits of regular exercise for moderately obese adults are not disputed and achieved independent of weight loss. Therefore, the current targets on reducing body weight should be replaced by a focus on changes in overall physical and psychological health.

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