



REVIEW

Effects of physical activity on food intake

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Summary The understanding of the effect of physical activity (PA) on food intake is imperative for considering PA as an additional tool for prevention and treatment of many diseases. Prolonged strenuous PA performed on a regular basis causes an increase in overall energy turnover, and leads either to loss of body weight, or to an increased food intake. When leading to loss of body weight, PA may be used as a therapeutic adjunct in the treatment of obesity. When increasing food intake to compensate for increased energy expenditure and maintaining body weight, PA, by increasing intake of other constituents of food like minerals and vitamins, may contribute to micronutrient deficiency coverage. However, the type of activity, as well as body composition of the individuals engaging in sport, play an important role in food intake regulation and its changes with time. Some studies indicate that short-term exercise does not have the same effect on food intake as long-term exercise and that eventual increase in food intake due to increased PA does not follow the same pattern in obese as in lean individuals. To better understand the relationship between PA on food intake, current findings on this topic will be summarized in this paper.

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Contents

Introduction	886
Relationship between food intake, body mass and physical activity	886
Effects of sedentarism on food intake	887
Energy intake in response to high-energy requirements	887
Effect of short-term exercise on food intake	888
Regulation of energy intake	889

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Effects of long-term exercise on food intake in lean and obese subjects.	890
Conclusion.	894
Acknowledgement.	894
References.	894

Introduction

Physical activity (PA) is closely interrelated with energy intake. The working body requires energy and nutrients in order to fuel activity and function. PA, on the other hand, manipulates energy expenditure and regulates the use of fuels. When prolonged strenuous PA is performed on a regular basis, it causes an increase in overall energy turnover¹ and leads either to loss of body weight, or to a need for an increase in food intake.² Nevertheless, many questions remain open in that regard. For example: is increased energy expenditure due to short-term PA automatically compensated for by changes in energy intake? Does short-term exercise have the same effect on energy intake as long-term exercise? Does the eventual increase in food intake due to increased PA follow the same pattern in obese as in lean individuals?

Thus, to which extent the energy intake is regulated through PA still remains unclear and further research is required that may provide useful information for public health guidelines. For example, vigorous PA on a daily basis not only increases the overall intake of energy,³ but also of other constituents of food, such as micronutrients. Therefore, in conjunction with low-energy food abundant in minerals and vitamins, PA may not only enhance human health in general,⁴ but also prevent micronutrient deficiencies.⁵

Therefore, reaching a clear understanding of the relationship between PA and food intake may prove valuable in choosing the most beneficial approach for individual and societal health management. The aim of this paper is thus to summarize the state-of-art in our understanding of the effects of PA on energy and nutrient intake.

Relationship between food intake, body mass and physical activity

Mayer et al.^{6,7} performed several of the earlier studies, on both animals and humans, examining the effect of exercise on body mass and food intake. To the extent that it is legitimate to draw an equivalence between purely experimental results and the results of population studies, a remarkable parallelism was found between the

two.⁷ In order to provide an accessible overview of these and other findings on the same topic, the relationship between food intake, exercise and body mass is conceptualized in Fig. 1.

The first study was performed in rats exercising on a treadmill.⁶ It was observed that an introduction of short periods of exercise (1 h or less/day) in the life of normal rats were not followed by an increase in food intake over the amount corresponding to absolute inactivity. In fact, a significant decrease in food intake occurred indicating a probable greater availability of endogenous fuel mobilization due to enhanced vascularization of body tissues.⁶ As a result, the mass of the animals

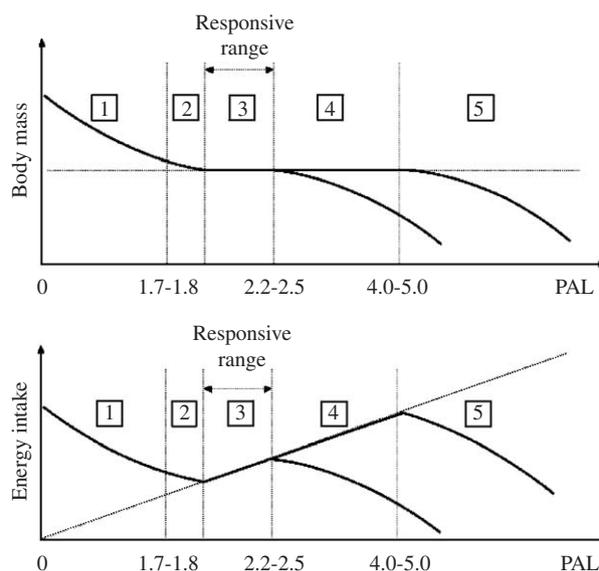


Figure 1 A model showing the relationship between Physical Activity Level (PAL), body mass and food intake; *zone 1*: decrease in PAL and increase in sedentariness does not induce compensatory reduction in food intake and leads to increase in body mass; *zone 2*: introduction of acute physical activity on a short-term basis suppresses food intake, due to mobilization of stored fuels, and leads to decrease in body mass; *zone 3 (responsive range)*: moderate to intense physical activity performed regularly and on a long-term basis by lean individuals increases food intake accordingly and maintains body mass; obese individuals, due to their excess energy storage, do not show significant changes in food intake; the PAL level in the general population has an upper limit around 2.2–2.5 (*zone 4*), and in highly trained subjects around 4.0–5.0 (*zone 5*). Above these values, the human body is not capable to cover the high-energy expenditure, and loss of body mass occurs as a consequence.

decreased. This range of activity was called “non-responsive” with respect to food consumption. When the duration of exercise increased more than 1 h/day, food intake increased linearly and the body mass was maintained. This range of activity was termed “normal” because of the proportional response of exercise on food intake. Once the exercise was prolonged to more than 6 h/day, the animals became exhausted, lost body mass, food intake decreased and appearance deteriorated. As the increase in the activity was not accompanied by a corresponding increase in food intake, this range of PA was also considered as “non-responsive”.

The second study of Mayer et al.⁷ used an indirect method of assessing PA based on job classification along with dietary intake records in Indian men. Although the study was not well controlled it was concluded that men employed in jobs requiring physical labour increased energy intakes with increased demands for PA while maintaining their lean body mass constant. On the other hand employees with a sedentary job had a disproportional larger energy intake compared to the PA levels. Thus, energy intake balanced with activity level only within a certain zone. By analogy with previously used terminology⁶ this zone was called “normal activity range”.

Effects of sedentarism on food intake

In mice, under laboratory conditions, the decrease in energy expenditure due to reduced activity is not accompanied by a corresponding decrease in energy intake.⁸ Thus, physical inertia in mice is considered to play an essential role in the development of obesity.

The influence of inactivity on energy balance was investigated in humans by Murgatroyd et al.⁹ Two-day measurements of energy expenditure in the respiration chamber, and ad libitum food intake were measured in eight normal-weight male subjects. The limited PA did not induce a decrease in food intake, and thus resulted in a positive energy balance. When total energy expenditure data was compiled from 22 studies that used the doubly labelled water method,¹⁰ a considerable variation in body fatness was present among sedentary individuals, again suggesting that a low rate of non-basal energy expenditure is a predisposing factor for obesity.

More recent studies with humans also examined the occurrence of positive energy balance as a result of decreased PA. Stubbs et al.¹¹ attempted to assess the effect of an imposed sedentary routine

on energy intake and nutrient balance in six lean men (Fig. 1, zone 1). Each subject was studied during a sedentary ($1.4 \times$ resting metabolic rate (RMR)) and an active ($1.8 \times$ RMR) regime, in protocols that involved 7 days of continuous monitoring in the respiration chamber. It was concluded that reducing the level of PA from 1.8 to $1.4 \times$ RMR did not induce a compensatory reduction of energy intake and led to a significant positive energy balance, most of which was stored as fat.

A review of cross-sectional data from double labelled water studies in adults¹² supports the latter. The resulting estimate of the threshold of PA associated with weight control is around $1.8 \times$ RMR. Physically active men ($>1.75 \times$ RMR) had lower body mass index than did those with lower energy expenditures. Previously obese women whose activity was above $1.75 \times$ RMR were generally able to maintain their reduced weight, whereas half of the less active women gained weight. Saris et al.¹³ concluded that, although definitive data are lacking, a physical activity level (PAL) of 1.7–1.8 is required to prevent transition to overweight or obesity.

Energy intake in response to high-energy requirements

Animals and humans who engage regularly in moderate to intense PA are capable of maintaining their lean body mass.^{6,7} An important aspect of the maintenance of energy balance during high-intensity endurance exercise is an adjustment of food intake to high-energy requirements. An example of daily energy intake in endurance, strength, and team sport athletes are presented in Table 1.¹⁴ The nutritional information about habitual food intake was obtained from a 4 or 7-day food diary on athletes, who trained at least 1–2 h/day and competed mostly on an international level.

Food intake and energy expenditure was studied in four cyclists during the 22-day race of the Tour de France.¹⁵ The purpose of the study was to verify whether the energy intakes of the cyclists were in agreement with the energy expenditure on a race that could be considered as one of the most strenuous endurance activities that exist (4000 km long, including 30 mountain passages (up to 2700 m altitude)).¹⁶ Based on the double-labelled water technique, the cyclists reached an average daily metabolic rate of 4.3–5.3 times basal metabolic rate. The decrease in their body mass and body composition was not considered significant, and all

Table 1 Daily energy intake in endurance, strength, and team sport athletes.*

Type of sport	Sex	n	Age in years Mean \pm SD	Energy intake (kJ/kg ⁻¹)	
				Mean	Range
<i>Endurance</i>					
Cycling (Tour de France [†])	M	5	26 \pm 3	347	286–388
Cycling (Tour de l'Avenir)	M	4	24 \pm 2	316	247–378
Triathlon [†]	M	33	26 \pm 3	272	246–295
Cycling, amateur	M	14	20 \pm 1	253	207–314
Marathon skating [†]	M	5	33 \pm 5	222	175–294
Swimming [†]	M	20	18 \pm 3	221	119–300
Rowing [†]	M	18	22 \pm 2	189	167–225
Running [†]	M	56	30 \pm 5	193	127–311
Rowing	F	8	23 \pm 2	186	140–200
Cycling, amateur [†]	F	21	23 \pm 4	164	115–215
Running [†]	F	18	31 \pm 5	168	123–218
Sub-top swimming	F	50	12 \pm 2	200	92–338
<i>Strength</i>					
Body building [†]	M	8	30 \pm 7	157	106–183
Judo [†]	M	4	23 \pm 1	157	76–210
Weight lifting	M	7	27 \pm 4	167	99–203
Judo [†]	M	28	18 \pm 1	177	60–325
Top gymnastics	F	11	15 \pm 1	158	91–216
Sub-top gymnastics	F	41	13 \pm 1	206	113–334
Body building ^{c†}	F	4	25 \pm 5	110	91–133
<i>Team sport</i>					
Water polo [†]	M	30	24 \pm 3	194	92–299
Soccer [†]	M	20	20 \pm 3	192	118–287
Hockey [†]	M	8	27 \pm 2	181	167–217
Volleyball	F	9	23 \pm 3	140	101–229
Hockey [†]	F	9	24 \pm 4	145	91–199
Handball	F	8	22 \pm 2	142	78–271
Total		419			

*Adapted from: van Erp-Baart AM, et al., 1989. Nationwide survey on nutritional habits in elite athletes. Part I. Energy, carbohydrate, protein, and fat intake. *Int J Sports Med*, 10(Suppl. 1): S3–S10.

[†]Including World, European, and Olympic medal winners.

four subjects remained in energy balance (Fig. 1, zone 3).

Nevertheless, there is a limit to the performance of an organism set by energy intake and energy expenditure. The PA level in the general population was shown to have an upper limit of approximately 2.2–2.5 (Fig. 1, zone 4).¹⁷ The upper limit of sustainable metabolic rate is approximately twice as high in endurance athletes (Fig. 1, zone 5), as a result of long-term exercise training, increased fat-free mass and consumption of carbohydrate-rich food during exercise.^{16,17} Above these values, a human body is not capable to cover the high-energy expenditure with energy intake, and loss of body mass occurs as a consequence, which together with symptoms like fatigue, mood changes and sleep problems constitutes the overtraining syndrome.¹⁸

When regular PA is introduced in the otherwise sedentary population, one would thus expect that the increased energy expenditure accomplished through exercise would rapidly lead to increased dietary intake. However, as shown by the studies cited below, this reasoning is too simplistic. The type of activity, as well as body composition of the individuals engaging in sport, play an important role in food intake regulation and its changes with time.

Effect of short-term exercise on food intake

In highly physically trained persons, the enormous daily energy expenditure due to vigorous exercise is

thus generally matched by a high-energy intake (Fig. 1, zone 3).¹⁵ By contrast, under more normal conditions, increased energy expenditure due to short-term PA is not automatically compensated for by changes in energy intake in both, lean and obese individuals (Fig. 1, zone 2).¹⁹

King et al.²⁰ examined the effect of different types of short-term exercise on energy intake. Twenty-three healthy lean male subjects (21–27 years, mean BMI = 24.2 kg/m²) were randomly assigned to a control, low-intensity exercise treatment (cycling at 30% VO₂ max for ~60 min) and high-intensity exercise treatment (cycling on 70% VO₂ max for ~30 min) in the first study, and to a control, short (mean time = 26 min) and high duration (mean time = 52 min) exercise treatment of high intensity in the second study. For energy cost calculation, expired air was collected periodically during each of the exercise sessions using a modified Douglas bag system. Energy and macronutrient intakes were measured by recorded intakes for the next 2 days. It was concluded that short-term low-intensity exercise did not induce a suppression of hunger, and that hunger ratings at no time significantly deviated from the hunger in the session that included a rest period. Only high-intensity exercise induced a suppression of hunger. Long duration, high-intensity exercise had a stronger effect on energy intake than short duration exercise period. It was concluded that high-dose exercise suppresses appetite and induces an exercise-induced anorexia.

In another study, King et al.²¹ examined the effect of high doses of exercise (exercise-induced energy expenditure of ~1200 kcal) on energy intake within 48 h. Douglas bags were used to collect the expired air during the exercise in order to calculate the total energy cost. Energy intake was monitored using self-recorded food diaries. The study results suggested that a high dose of exercise occurring on a single day failed to have any effect on energy intake within the same day or on the day immediately after the exercise, compared with days of no exercise. The results demonstrated that a substantial but acute increase in energy expenditure due to intense exercise does not automatically increase hunger or energy intake within 48 h.

The effect on energy intake of two levels (30 vs. 90 W) of programmed exercise on a cycle ergometer for 40 min/d during a 3-day period were compared in nine obese and non-obese women.²² It was found that intake of liquid meals (1.04 kcal/g) 15 min after the exercise was significantly lower after the strenuous than after the moderate exercise in the non-obese women, but was not different after the two conditions in the obese women.

To study the effect of acute exercise on caloric intake in normal-weight young people, food intake was monitored in 10 men and 10 women during a period of 5 consecutive days.²³ All exercise was done on a treadmill and oxygen consumption was measured during the exercise period for energy cost evaluation. Throughout the protocol, subjects obtained all their food from a specially equipped metabolic kitchen in a research setting. All items not eaten were returned to the metabolic kitchen for weighing. Men responded to 5-day acute exercise with increased caloric intake (208 ± 64 kcal/d), which was insufficient to compensate for the caloric cost of exercise (596 kcal/d above RMR). Women did not change their caloric intake despite expending 382 kcal/d during exercise. Consequently, both normal-weight men and women were found to be in negative caloric balance during the exercise period.

These studies, as well as many others,^{24–26} reported a negative energy balance after a short-term increase of energy expenditure. The findings support the first results presented by Mayer et al.⁶ on suppressed dietary intake with introduction of acute PA (Fig. 1, zone 2). At that time, Mayer speculated that a reduction in food intake occurred because of the greater availability of body reserves due to changes of circulatory conditions. More recent studies performed on rats confirmed that blood glucose and free fatty acids levels, as well as plasma lactate levels are all increased during and after short-term exercise.²⁷ Thus, the mobilization of stored fuels into the blood may indeed play an important role in the inhibition of food intake. It was also found that exercise is accompanied by an increase in the release of glucagon, which in combination with other satiety factors such as cholecystokinin, cytokines and serotonin suppresses food intake. King et al.²⁸ reviewed studies reporting the effects of exercise on appetite control in humans and concluded that an elevation of body temperature, increased levels of lactic acid and an increase of tumor necrosis factor are all possible mechanisms that induce the suppression of hunger.

Regulation of energy intake

The absence of a compensatory effect in response to an exercise-induced energy deficit would not be expected to continue indefinitely; otherwise a considerable loss of body mass would occur. At some stage, a regulatory mechanism must trigger an increase in energy intake in order to match

energy expenditure. This mechanism reflects an active regulatory process within a human body, termed energy homeostasis.²⁹ Its variation is associated with changes in body weight and more specifically, with variations in protein and fat reserves.³⁰ Since fat reserves represent the most unstable depot of the body,³⁰ the main role of energy homeostasis is to promote stability in a body fuel stored as adipose tissue.³¹

The ingestion of food and expenditure of energy are regulated by a large number of mechanisms within the central nervous and hormonal system. Different signals reflecting the metabolic state arise from different peripheral tissues, including white adipose tissue, gastrointestinal tract, thyroid, adrenals, muscle, and reproductive organs.³² The signals with an anabolic effect stimulate food intake and promote weight gain (e.g. neuropeptide Y, ghrelin); those with a catabolic effect decrease food intake and promote depletion of body fat (e.g. insulin, leptin, corticotropin-releasing hormone, proopiomelanocortin).³³ Weight loss induced by, for example, prolonged exercise lowers insulin and leptin levels, which in turn activate anabolic and inhibits catabolic effectors, thereby promoting the recovery of lost weight.³⁴

Although previous evidence suggests that there is a weak coupling between energy intake and energy expenditure in the short term (1–3 days), Edholm et al.³⁵ confirmed that over longer periods of time, 7 days and more, the correlation between energy intake and expenditure improves. They studied this regulation between energy intake and output in military cadets with a relative high level of daily exercise. However, in well-trained athletes this mechanism must be very well regulated in order to avoid negative energy balance and thus fuel shortage to perform physically. It is suggested in some of the studies that intake follows expenditure with a 2-day interval, although this lag has not been confirmed by others. Saris³⁶ studied this phenomenon in professional cyclists during the Tour de France where expenditure levels are extremely high but also vary variably over the days ranging from 4302 to 7648 kcal/day. The correlation coefficient was calculated between the individual moving averages for energy expenditure and energy intake over 2–5 days in four athletes. The data clearly showed that the correlation coefficients did not improve further taken a period of 3 days into account. (Range 0.80–0.95).

The observed 3-day period is interesting and indicates that the 2-day period suggested by Edholm³⁷ seems to represent a physiological phenomenon under the conditions of hard physical work. Many of the compensating mechanisms

previously suggested like body temperature,^{27,38,39} blood lipids,^{40,41} glucose or insulin concentrations,^{40,42} might well come into play on a short-term basis. Long-term regulatory mechanisms like adipose tissue-related factors such as leptin^{43,44} are also known now. But for periods around 3–7 days no good physiological mechanisms are yet available.

Effects of long-term exercise on food intake in lean and obese subjects

It is clear that, as energy output is increased through exercise, in order to maintain body mass, an individual must increase energy intake to match expenditure. The lean marathon-runner eats much more calories than his sedentary counterpart.⁴⁵ Many reports in the literature confirm the existence of this balancing act over the long term. Even though the present obesity epidemic needs more insight into the effects of increasing energy expenditure in obese and lean individuals, several studies suggest that the increase of PA is less accurately matched by a corresponding increase of energy intake in obese than in lean individuals (Table 2).

Leon et al.⁴⁶ studied six sedentary obese men aged 16–31 years who completed 16 weeks of 90 min vigorous walking, 5 days/week, on a treadmill at up to 5.2 km/h on a 10% grade, expending about 1100 kcal per session. Physical work capacity was assessed by means of the modified Balke multistage treadmill exercise test protocol. There was no attempt to influence their dietary habits during this period of time, and eating patterns were monitored by 3-day dietary records before exercise and at 4, 8, 12, and 16 weeks of training. Monitored food intake initially increased and then progressively decreased below pretraining level. As a result the obese men lost an average 5.7 kg of body mass, representing the sum of 5.9 kg lost as fat and 0.2 kg gained as lean tissue.

The effect of exercise on spontaneous energy intake in obesity was studied by Woo et al.⁴⁷ as well. In their study, six obese women were hospitalized as a part of a metabolic experiment. They underwent three 19-day treatments—one sedentary (no exercise), the second with treadmill exercise increasing daily expenditure to 110% (mild) of sedentary expenditure, and the third with the treadmill exercise increasing daily energy expenditure to 125% (moderate) of sedentary expenditure. Energy expenditure was monitored with daily activity diaries and indirect calorimetry

determinations of all the activities recorded, while voluntary intake was covertly examined by weighing all the items before and after serving. Although the expenditure increased significantly from 2221 to 2419 kcal/day during mild treatment and to 2714 kcal/day during moderate treatment, the results suggested that mean daily energy intake was not different between the three treatments. It was concluded that moderate realistic levels of PA, which required 39 min/day of brisk walking at mild and 96 min/day at moderate levels, was not compensated by increased energy intake in these obese women, at least not within the time frame of the study.

In order to study if this effect would be sustained over a longer time period, a second group of three obese female volunteers was maintained for all three 19-day periods at 125% of their sedentary expenditures.⁴⁸ Mean daily intake of 1903 kcal/day and expenditure of 2882 kcal/day did not change during these 57 days, resulting in lipolysis and consistent body mass loss of 0.12 kg/day. The study showed that an increase in expenditure during a prolonged period of time did not induce any changes in energy intake. This suggests that in a state of a positive energy/fat reserve there is a possible uncoupling of the intake of energy from the expenditure of energy such as in these obese women, at least in the settings and durations of the studies cited.

A much longer exercise program, lasting 8 months, was performed by 120 sedentary, overweight or obese men and women (BMI = 25–35) aged 40–65 years.⁴⁹ The subjects were randomly assigned to 1 of 3 exercise training groups: high amounts/vigorous intensity (32 km/week of jogging at VO_2 max = 65–80%), low amount/vigorous intensity (19.2 km/week of jogging at VO_2 max = 65–80%) and low amount/moderate intensity (19.2 km/week of walking at VO_2 max = 40–55%). The subjects were consuming ad libitum food intake. Even so, the high-amount/vigorous-intensity group lost 2.9 ± 2.8 kg body mass and 4.8 ± 3.0 kg fat mass; the low-amount/moderate-intensity group lost 0.9 ± 1.8 kg body mass and 2.0 ± 2.6 kg fat mass; and the low-amount/vigorous intensity group lost 0.6 ± 2.0 kg body mass and 2.5 ± 3.4 kg fat mass. The controls gained $+1.0 \pm 2.1$ kg and $+0.4 \pm 3.0$ kg body mass and fat mass, respectively. There were no significant changes in energy intake for any group. These findings again suggest an uncoupling of energy intake from expenditure in subjects with an excess of body energy reserves.

In order to clarify the effect of exercise-related factors on the energy intake of normal-weight

individuals, non-obese women were studied using the same protocol⁴⁷ as previously in comparable environments.³ Thus, five non-obese women underwent three 19-day treatment periods in which PA was modified. Total daily expenditure was increased to $114 \pm 4\%$ through mild exercise and to $129 \pm 3\%$ through moderate exercise. Intake was estimated by weighing the platters before and after serving and expenditure was monitored with daily activity diaries and indirect calorimetry determinations of the recorded activities. The corresponding voluntary intake during mild (mean expenditure 378 ± 63 kcal/d) and moderate (mean expenditure 772 ± 40 kcal/d) exercise was $117 \pm 5\%$ and $122 \pm 6\%$ of sedentary treatment. The resulting energy balance was $+10 \pm 71$ kcal/d for sedentary, $+64 \pm 43$ kcal/d for mild and -116 ± 92 kcal/d for moderate treatments. Thus with normal body energy reserves as in these normal weight women, the exercise-induced metabolic signals played a hyperphagic role in the regulation of energy intake, as the stability in body mass during sedentary (55.1 kg), mild (55.9 kg), and moderate (55.7 kg) was observed.

It should be noted that with the above moderate levels of PA, voluntary energy intake did not increase as much as expenditure. If the study were prolonged over a longer period of time, it would possibly result in negative energy balance. It was noted that the women had struggled to complete their treadmill assignments and that, possibly, a fatigue effect came into play. This situation is comparable with the one presented by Mayer et al.,⁶ where exhaustion exerted an anorexic effect on the intake of heavily exercising rats. For the particular subjects of this study, exercise costing more than 700 kcal/d may be close to the maximum PA level that could be accomplished on a habitual basis.⁴⁷

Nevertheless, this study indicates that unlike obese subjects, who do not match their energy intakes to energy expended through PA, lean subjects demonstrate an increase in energy intake and insignificant changes in body mass when exercised on a long-term basis. It is hypothesized that the absence of an increase in energy intake when increasing expenditure in obese subjects is based on their excess energy stores in the form of adipose tissue.

Adipose tissue is not only a lipid storing tissue but consists of functionally specialized tissues able to produce heat (in brown adipose tissue) and to produce or release a vast number of adipocytokines.⁵⁰ The adipocytokines, under normal weight conditions, guarantee homeostasis of glucose and lipid metabolism, but their dysregulated production in the obese state is associated with

Table 2 Effects of long-term exercise on ad libitum food intake in overweight and obese subjects.

Study	Subjects	Energy expenditure through exercise	Duration of the study	Significant change in food intake	Observations
Leon et al. ⁴⁶	6 obese men BMI = 38.2 kg/m ²	Walking 90 min, 5 d/week	16 weeks	No	Slightly decreased food intake
Woo et al. ⁴⁷	6 obese women BMI = 34.6 kg/m ²	Treadmill exercise (281 kcal/d and 694 kcal/d)	57 days	No	Slightly increased food intake
Woo et al. ⁴⁸	3 obese women BMI = 38.3 kg/m ²	Moderate exercise (125% of sedentary expenditure)	57 days	No	Slightly increased food intake
Durrant et al. ²⁶	12 obese subjects (1 male, 11 female) BMI = 37.6 kg/m ²	Bicycle ergometer (100 kcal/d)	6 days	No	Slightly decreased food intake
Wood et al. ⁵³	47 overweight men	Jogging (individual prescription necessary for 30% body fat decrease over 9 months)	1 year	No	Slightly decreased food intake
Keim et al. ⁵⁴	12 overweight women BMI = 27.4 kg/m ²	Walking 7 d/week (12.5% and 25% increase in E expenditure)	36 days	No	Slightly increased food intake

Andersson et al. ⁵⁵	22 overweight women 9 overweight men BMI = 25.15 kg/m ²	Exercise three times/week, 60 min/ session	3 months	No (men) Yes (women)	Slightly decreased food intake in men Significant decrease food intake in women
Snyder et al. ⁵⁶	13 obese women BMI = 32.5 kg/m ²	Moderate exercise 30 min/d, 5 d/week	32 weeks	No	Slightly increased food intake
Mertens et al. ⁵⁷	12 overweight subjects (8 males, 4 females) BMI = 29.5 kg/m ²	Walking 1 h/d (6.4 km/h)	12 months	No	Slightly increased food intake
Donnelly et al. ⁵⁸	11 obese women BMI = 30.12 kg/m ²	Exercise (559 kcal/week)	18 months	No	Slightly decreased food intake
Donnelly et al. ⁵⁸	11 obese women BMI = 32.33 kg/m ²	Walking (808 kcal/week)	18 months	No	Slightly decreased food intake
Donnelly et al. ⁵⁹	74 overweight subjects BMI = 29.7 kg/m ²	Moderate intensity exercise 45 min/d, 5 d/week	16 months	No	Slightly decreased food intake in women Slightly increased food intake in men

altered metabolic pathways, vascular pathologies and inflammatory processes.⁵¹ Thus, adipose tissue plays an active role in energy balance, and the responses of energy intake to exercise induced changes in energy expenditure may depend on the amount of body fat. It is likely that, in the obese state, fat mass acts as an energy buffer⁵² and that compensatory responses in intake to altered levels of exercise may not begin to act until the excess energy stores become depleted and the energy homeostasis is at risk.³

Conclusion

Increased energy expenditure due to short-term PA is not immediately compensated for by changes in energy intake. Once moderate to intense PA is performed regularly and on the long-term basis, however, a distinction has to be drawn between lean and obese subjects. While the lean show a tendency to balance the extra PA energy expenditure by adapting their energy intake accordingly within a period, of about 3 days, the obese, probably due to their excess energy storage, do not show such a compensatory mechanisms.

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