

HOW EFFECTIVE IS EXERCISE IN PRODUCING FAT LOSS?

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Review

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Abstract:

Over the past quarter century obesity has reached epidemic proportions in many developed countries. Exercise is one of the behavioral approaches to curbing weight gain or losing body fat. We reviewed the data on (1) magnitude of weight and fat loss when exercise is implemented without any changes in food intake, (2) necessary volumes and intensities of exercise to accomplish and sustain weight and fat loss, (3) effects of obesity on spontaneous physical activity and other forms of energy expenditure, (4) effects of obesity and aerobic fitness on the capacity to oxidize fat, and (5) the effects of exercise on the appetite. When exercise is implemented without any changes in food intake, a daily expenditure of 400 Kcal produces a fat loss that is about one third of that expected on the basis of caloric deficit incurred by exercise. Oxidation of fat is negatively influenced by exercise intensity so that for an appreciable fat loss it would be necessary to exercise for long periods of time. Genetics and low aerobic fitness can also play a negative role. Obesity is associated with low levels of spontaneous physical activity and reduced capacity to oxidize fat. Aerobic training can enhance the capacity to oxidize fat by increasing the volume and function of mitochondria in skeletal muscles. Exercise is associated with short-term suppression of hunger and a failure to compensate for exercise energy expenditure during the hours to several days following exercise. Exercise is advisable as an approach toward fat loss, even more so when it is associated with dietary restraint, because it improves fat oxidation, saves lean body mass, and does not immediately stimulate hunger.

Key words: *exercise intensity, food intake reduction, energy expenditure, aerobic fitness, fat oxidation, genetics, short-term suppression of hunger, lean body mass*

Introduction

Over the past quarter century, obesity rates have increased dramatically in developed countries (Flegal, Carrol, Jogden, & Johnson, 2002; Mokdad et al., 1999; Popkin & Doak, 1998; Seidell, 1999). In the USA alone in 2005, about 60% of the adult population was overweight, about a quarter of the population was obese, and 3% was extremely obese (Centers for Disease Control and Prevention, 2008). Changes in our lifestyle are important contributors to the rapid rise in obesity, since our genetic make-up cannot modify so quickly. The obvious non-genetic reasons for the rise in obesity are an over-consumption of energy-rich food and a decrease in physical activity. While there are many social, environmental, and socio-economic reasons for these lifestyle shifts, this review will focus on information concerning exercise as a means of weight control and fat loss. Evidence-based research will be presented to examine the following issues: (1) magnitude of weight and fat loss when exercise is implemented without any changes in food intake,

(2) necessary volumes and intensities of exercise to accomplish and sustain weight and fat loss, (3) effects of obesity on spontaneous physical activity and other forms of energy expenditure, (4) effects of obesity on the capacity to oxidize fat, and (5) the effect of exercise on the appetite. These lines of research will be used to (6) generate a hypothesis of how the biology of human control of hunger and physical activity interact to produce weight plateaus and how we should modify our lifestyle to prevent weight problems or lower and maintain reduced body fat levels.

Effectiveness of exercise alone, without changes in food intake, to lower body weight and body fat levels

Because of the health risks associated with obesity, exercise as a means of maintaining body weight or lowering body weight and fat has attracted research attention for over a quarter of a century. Several previous reviews have been devoted to this topic (Ballor & Keeseey, 1991;

Catennachi & Wyatt, 2007; Epstein & Wing, 1980; Leon & Sanchez, 2001; Miller, Koceja, & Hamilton, 1997). To generate substantial fat loss, a considerable volume of exercise must be carried out over a period of time. Since different studies used different durations of exercise, in this review the data will be normalized to percent weight loss achieved over four weeks. Several prior authors did not measure energy expenditure directly and in some instances described the intensity of exercise in very general terms. Energy expenditure in these studies was estimated from the subjects' weight, described speed and duration of exercise, and the established value of the energy cost of walking or jogging over a distance of 1 km (1 Kcal/km kg body weight). The data from the available reviews (Ballor & Keeseey, 1991; Epstein & Wing, 1980; Leon & Sanchez, 2001) and a number of individual studies reviewed by the author (Donnelly et al., 2003; Duncan, Gordon, & Scott, 1991; Dunn et al., 1999; Fortmann, Haskell, & Wood, 1988; Franklin et al., 1979; Katznel et al., 1995; Leon, Conrad, Hunninghake, & Serfass, 1979; Ready et al., 1995; Rotkis et al., 1981; Wood et al., 1983; Wood et al., 1988), indicate that most exercise studies, carried out in the absence of dietary restraint, produce very modest weight and fat losses. Figure 1 shows that monthly body fat loss is a function of daily exercise energy expenditure.

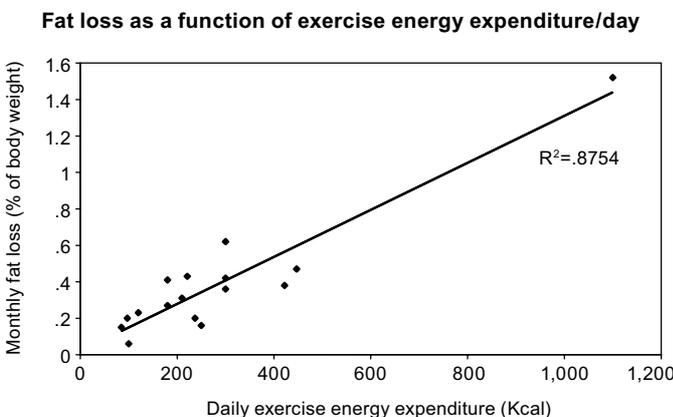


Figure 1. Monthly percent weight loss as a function of daily exercise energy expenditure.

Figure 1 also shows that in most studies, this exercise energy expenditure was too small to produce substantial fat losses. One outstanding data point was generated by a study reported by Leon et al. (1979) where male subjects expended 1,100 Kcal in daily exercise over a 16 week period to produce a substantial monthly fat loss.

Besides the direct energy cost of exercise, expected indirect effects of physical workouts are increases in resting metabolic rate and in diet-induced thermogenesis (DIT). These, too, could contribute

to total energy loss. Here the data are less clear. Several studies that involved substantial energy expenditure through severe exercise reported increases in metabolism over a period of hours. For instance, Edwards, Thorndike and Dill (1935) reported a 7% increase in sleep metabolic rate after a football practice in college-age students. This increased to 9.9% after a severe workout and to 20% after a very severe workout. Sleep metabolic rate was 23% higher 13 hours after a lacrosse game in college students, and 8% higher after 31 hours. Seven hours after walking 10 km at 4 km/h (at which time the measurements were discontinued), the metabolic rate in four men was 14 to 17% higher than before exercise (Passmore & Johnson, 1960). However, most studies without severe workouts in very young subjects show a rapid decline in metabolic rate after exercise. For instance, metabolic rate returned to pre-exercise levels within 90 minutes of three hours of walking at 50% VO_{2max} (Bielinski, Schutz, & Jequier, 1985), and within 1 hour of 20-min bouts of exercise carried out at 35 to 55% VO_{2max} during each of 4 consecutive hours (Pacy, Barton, Webster, & Garrow, 1985). It therefore does not appear that other than extremely strenuous and long exercise in young subjects, metabolic rate increases substantially to contribute to significant energy loss. The same conclusion can be made for

DIT. It increased 33% above the values seen after meals in a sedentary state in young men after 3 hours of exercise at 50% of maximal effort (Bielinski, Schutz, & Jequier, 1985), but no exercise-associated increases in DIT (or in the resting metabolic rate the next morning) were seen in studies with post-menopausal women after 2 hours of exercise that expended 435 Kcal during each of two exercise bouts in a day (Wuorinen, 2007).

Necessary volumes and intensities of exercise to accomplish and sustain weight and fat loss

About 7,830 Kcal needs to be expended to lose a kilogram of body fat which contains 87% lipid. Assuming that one wanted to achieve a quarter of a kg of fat loss per week, almost 2,000 additional Kcal of physical activity would have to be expended per week. Assuming 5 days of exercise per week, the necessary energy expenditure should be 400 Kcal per day. The Table 1 (modified after American College of Sports Medicine Position Stand, 2001) shows the duration of exercise that is necessary to expend 400 Kcal for people of different body weight participating in different forms of exercise. Table 1 indicates that high volumes of exercise are needed to generate

a 400 Kcal daily energy expenditure. Such daily exercise expenditure levels may be unrealistic for most sedentary and unfit subjects, although some individuals, particularly those who engage regularly in sport, do achieve them.

Table 1. Minutes of continuous exercise necessary to expend 400 kcal based on subject's body weight (modified data from ACSM, 2001)

Body weight (kg)	75	100
Bicycle ergometer	64.5	48
Walking 3.8 km/h	107	80
Walking 4.5 km/h	92	69
Walking 5.3 km/h	79.8	60
Swimming laps	40	31
Resistance exercise	53	40
Ballroom dancing (fast)	58	44
Raking the lawn	80	60

Will daily expenditure of 400 Kcalories through exercise assure a weekly loss of 250 g of body fat? The answer is complicated by the variations in exercise intensity, an individual's genetic endowment, and the fitness level necessary to sustain high levels of fat oxidation. These circumstances make it difficult to achieve this rate of fat loss by exercise alone.

Turning to exercise intensity, low intensity exercise does permit higher rates of fat oxidation than higher intensity exercise. For instance, at 25% of maximal effort, about 90% of energy cost of exercise comes from oxidation of fat derived from fat depots, and about 10% of energy is provided by glucose produced by the liver (Romijn et al., 1993). These high rates of fat oxidation are a consequence of unimpaired circulatory connection between adipose tissue and muscle, sympathetic stimulation of lipolysis, and sufficient oxygen supply to support the slow process of fat oxidation. At moderate intensity of 65% of maximal effort, about half of the energy cost of exercise is supplied by free fatty acids, but only half of those are derived from the fat depots and the other half are supplied by the intramuscular fat. The other half of energy comes from muscle glycogen and to a lesser extent from liver glycogen. As exercise intensity increases to 85% of maximal effort, only about 30% of the energy cost of exercise is supported by fat, half of it of fat depot origin and the other half from intramuscular deposits. This is due partly to a circulatory disconnection between adipose tissue and muscle during intense sympathetic activation at high exercise intensities, and even more so by metabolic impairments of fat oxidation in favor of carbohydrate utilization.

What the Romijn et al. study shows (1993) is that exercise depends on a mixture of metabolic fuels so that fat lipids coming out of fat depots are uti-

lized during exercise to a variable extent. Choosing the lowest exercise intensity to maximize oxidation of fat from fat depots would not be realistic since total energy expenditure declines with reduction in exercise intensity. One would have to exercise for several hours a day at low exercise intensities to produce substantial fat losses (see Table 1). This metabolic design was perhaps a useful evolutionary feature for our ancestors who migrated on foot over many miles and for many days. It is not an appealing solution for 21st century individuals trying to shed rapidly unwanted body fat.

Effects of obesity and aerobic fitness on the capacity to oxidize fat

Additional complications in using exercise to achieve fat loss include genetics and fitness status. It is now clear that the propensity for fat gain has a genetic basis. Although the actual values are under discussion, most authors attribute to genetics about 60% of the tendency for fat gain (Bouchard, 1995). This genetic basis has been most thoroughly studied in Pima Indians who remain lean while living under physically and nutritionally more challenging conditions in Mexico, and who grow obese and diabetic in the USA (Schultz et al., 2006). Their genetic burden includes a tendency to be hypoactive, have lower metabolic rates, a lower capacity to oxidize fat, and increased capacity to store fat (Tataranni et al., 2003). The environment also encourages greater food consumption by Pima Indians in the USA than in Mexico (Schultz et al., 2006; Tataranni et al., 2003).

The molecular basis for the increased tendency of obese subjects to continue to store rather than reduce their fat mass is the lower proportion of oxidative fibers and reduced mitochondrial mass in their skeletal muscles. Mitochondria are subcellular sites of oxidative phosphorylation. In the obese state, the concentration of circulating fatty acids is elevated because of insulin resistance and reduced capacity of insulin to suppress adipose tissue lipolysis. High free fatty acid flux provides ample substrate for mitochondrial fat oxidation which cannot be effectively accomplished with the limited mitochondrial mass. The mitochondrial substrate overload leads to a dysfunction which includes a higher production of reactive oxygen species and a shift from fat to carbohydrate metabolism (Saris & Heymsfield, 2007). Obese people also have a lower expression of the adipose tissue hormone adiponectin which provides a potent stimulus for mitochondrial biogenesis (Civitarese et al., 2006). Thus obese people are handicapped in their effort to lose fat by reduced capacity to oxidize fat or to make more mitochondria and by a greater reliance on carbohydrate metabolism.

The other important variable that determines what proportion of exercise energy expenditure will

consist of fat is the aerobic fitness of an individual. Aerobic training has two important consequences. One is an increase in cardio-respiratory and circulatory capacities to supply oxygen to the working muscle. The other is an increase in the volume of mitochondria. A substantial body of knowledge has recently been amassed showing a direct connection between increased volume and function of the mitochondria in trained individuals and the capacity to oxidize fat (Saris & Heymsfield, 2007; Silva, 2006). A greater expression of adiponectin in adipose tissue contributes to increased mitochondrial biogenesis during exercise training (Citavirese et al., 2006). Aerobic training also favors a shift in the expression of oxidative fibers in the skeletal muscles. Collectively, all of these adaptive changes lead to the greater capacity of trained muscles to oxidize fat, a lower production of free radicals, and an overall reduction of the risk of all-cause death.

Effects of obesity on spontaneous physical activity

The final nail in the coffin is the non-homeostatic relationship between spontaneous physical activity and obesity. According to the tenets of most simplistic formulations of energy equation largely based on rodent research, exercise and food intake are connected to body fat stores through negative feedback loops. This energy regulatory view posits that when body fat stores decline, the urge to eat increases and energy expenditure of physical activity decreases to restore the fat depot balance. Conversely, when body fat stores increase, the appetite decreases and levels of physical activity increase to restore the body fat setpoint. This view is consistent with the results obtained with some models of genetically obese mice. The hormone leptin is considered to provide a negative feedback signal from the adipose tissue to the brain centers controlling feeding and physical activity (Schwartz, Woods, Porte Jr., Seeley, & Baskin, 2000). A leptin-deficient ob/ob mouse is hypoactive and can weigh three times more than the wild-type mouse due to increased food intake and accumulation of body fat (Friedman & Halaas, 1998). Injections of leptin into the ob/ob mouse suppress feeding, lead to loss of body fat, and, at a time when the weight is substantially reduced, result in increased levels of physical activity (Pellemounter et al., 1995).

Human data do not support the homeostatic, negative-feedback, linkage between physical activity and body fat level. As mentioned previously, obese individuals are reported to be hypoactive (Schultz et al., 2006; Thorburn & Proietto, 2000). This includes lower levels of spontaneous organized physical activity and the un-structured body movements labeled NEAT (nonexercise activity thermogenesis) (Levine, Eberhardt, & Jensen, 1999).

Obese rodents are hypoactive, while underweight rodents are hyperactive (Borer, 1983) comparable to anorexic individuals who often exercise compulsively (Hebebrand et al., 2003). Obese individuals spend more time sitting than lean individuals and this produces an energy gain of about 350 NEAT Kcal/day. Inducing an 8 kg weight loss in obese individuals through dieting, and a 4 kg weight gain in lean individuals through overfeeding, produced no changes in their NEAT energy expenditure (Levine, Lanningham-Foster, McCrady, Krizan, Olson, Kane, Jensen, & Clark, 2005).

Experiments showing that obese rodents can run as much as lean rodents when they are externally motivated (Borer, Potter, & Fileccia, 1983) and that chemical lesions in the limbic forebrain normalize spontaneous running activity in rodents rendered obese by dietary manipulations (Borer, Bonna, & Kielb, 1989) suggest that hypoactivity is more likely a consequence of obesity rather than a genetically predetermined trait. Spontaneous physical activity is also often increased by sex hormones and by seasonal increases in the length of daylight (Borer, 1983). These lines of evidence suggest that spontaneous physical activity may not be a homeostatic component of the energy regulatory mechanism. Rather, it may serve the purpose of increasing the probability of finding food in times of famine, or sexual mates in seasonal breeders at the start of beneficial long-day season.

The effect of exercise on appetite

A number of studies have repeatedly shown that exercise transiently suppresses the sensation of hunger and increases the sensation of fullness (Imbeault, Saint-Pierre, Almeras, & Fremblay, 1997; King, Burley, & Blundell, 1994; King et al., 2007; King, Lluch, Stubbs, & Blundell, 1997; King, Snell, Smith, & Blundell, 1990; Kissileff et al., 1990; Moore, 2000; Reger, Allison, & Reid, 1998; Stubbs et al., 2002; Stubbs, Sepp, Hughes, Johnstone, King et al., 2002; Thompson, Wolfe, & Eikelboom, 1988). An example is shown in Figure 2, from one of the several studies by John Blundell and his collaborators (King, Burley, & Blundell, 1994).

As shown in the left panel of Figure 2, expenditure of 300 Kcal on a bicycle ergometer at low effort of 30% of VO_{2max} (triangles) had no effect on the ratings of hunger before the mid-day meal, while expenditure of the same number of calories at 70% of maximal effort led to a significant but transient suppression of hunger. In the right panel, the same authors show that expenditure of twice as many calories at 75% of maximal effort (500 Kcal) caused a greater suppression of appetite than expenditure of 250 Kcal at the same exercise intensity. From these studies it appears that exercise energy expenditure above some threshold caloric deficit and exercise

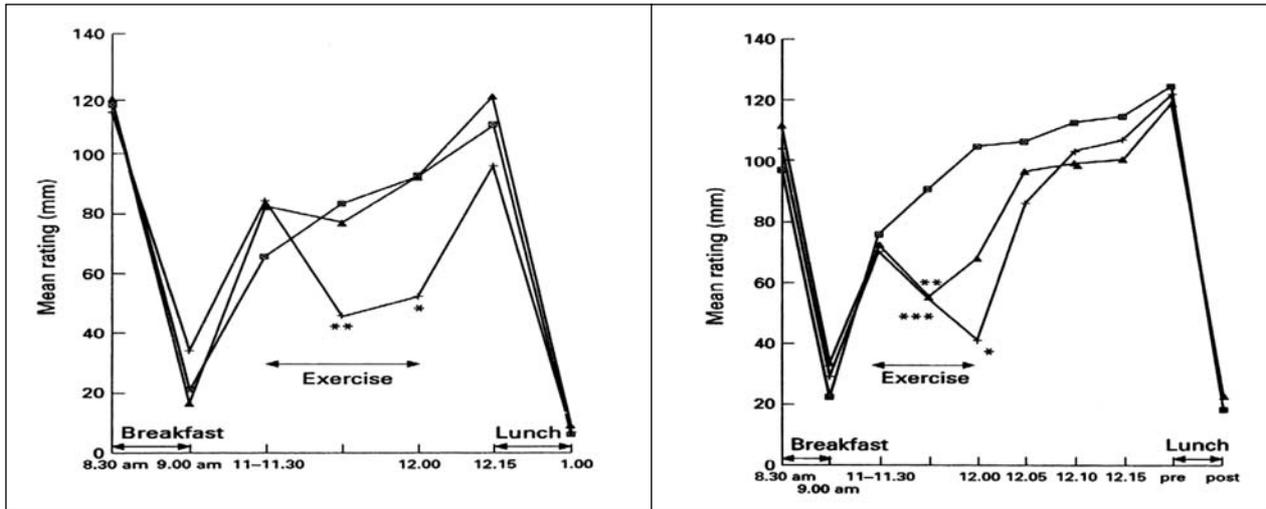


Figure 2. Expenditure of about 350 Kcal during one hour of cycling at 30% of maximal effort (solid triangles) did not alter hunger ratings while expenditure of the same number of calories over 30 minutes of cycling at 70% of VO_{2max} caused about 45 % lower hunger ratings (left). Expenditure of 540 Kcal during 52 min of cycling at about 75% of maximal effort produced about 60% lower hunger ratings, while expenditure of 300 Kcal at the same intensity over 30 minutes produced only 40% hunger suppression (right). (Figures from King, Burley, & Blundell, 1994).

intensity causes proportional reductions in hunger sensation.

What about consummatory behavior after exercise? Based on the energy regulatory models outlined above, energy expenditure should lead to either an increased appetite or increased food consumption. In the Blundell studies, energy intake was measured during a post-exercise meal and during two subsequent days (King, Burley, & Blundell, 1994). They found that, during 24 hours of the exercise day, and after the expenditure of 250 Kcal or 500 Kcal, food intake was indistinguishable from the intake on a sedentary day (Figure 3). No increases in food intake were detected during the two subsequent days either.

The above studies reveal a puzzling aspect of exercise in relation to energy regulation. Exercise energy expenditure causes appetite suppression in the short run, and no compensation for calories lost

over a longer period of time. The explanation for this paradox is at this time not available. But the phenomenon can be put to good use by the individual wanting to lose body fat through exercise. Blundell's studies and similar studies of others (Imbeault et al., 1997; King, Burley, & Blundell, 1994; King et al., 2007; King et al., 1990; Kissileff et al., 1990; Moore, 2000; Reger, Allison, & Kurucz, 1984; Staten, 1991; Stubbs et al., 2004; Stubbs et al., 1998; Stubbs et al., 2002; Stubbs, Sepp, Hughes, Johnstone, King et al., 2002; Thompson, Wolfe, & Eikelboom, 1988) further support the contention that exercise energy expenditure does not operate in a homeostatic negative-feedback manner to regulate short-term energy balance. Exercise studies that were carried out over several weeks indicate that energy intake can adjust to energy expenditure over a longer time period in lean (Woo & Pi-Sunyer, 1985) but apparently not in obese (Woo, Garrow, &

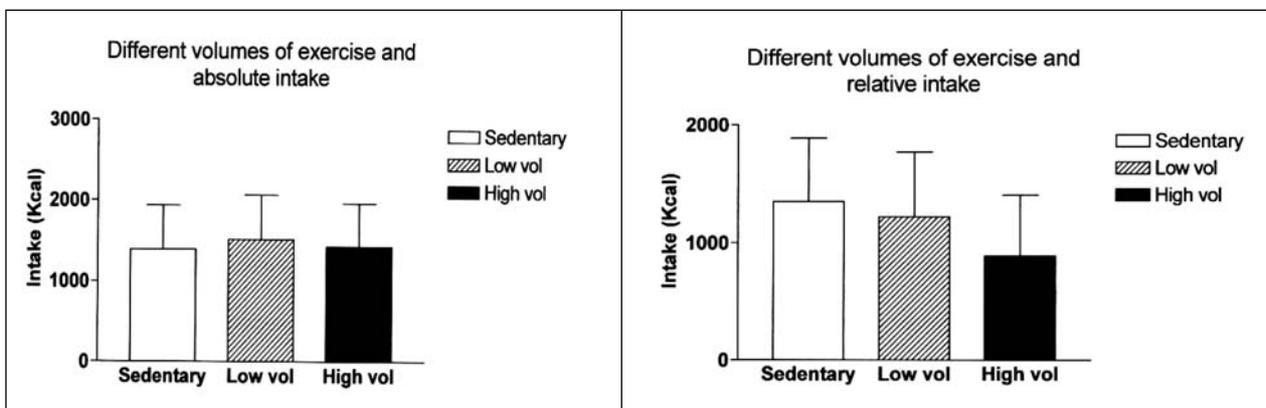


Figure 3. Daily energy intake during the exercise experiment described in the Figure 2 legend (left). Relative intake (right) reflects the difference between actual daily energy intake and the exercise energy expenditure in the same study (King, Burley, & Blundell, 1994).

Pi-Sunyer, 1982) subjects. The encouraging aspect of this fact is that subjects who chose to exercise regularly should not automatically experience increased hunger. In the aforementioned Leon et al. (1979) study, men who expended 1,100 Kcal daily over the period of 16 weeks, also reported progressive diminution of the sensation of hunger.

Synthesis of evidence and an integrative hypothesis regarding the use of exercise to promote fat loss

Clearly, all the presented facts do not provide a coherent picture of the role of exercise in energy regulation. If 400 Kcal daily exercise energy expenditure over 5 days should be sufficient to cause a loss of .25 kg fat per week, why does it not do so? As Figure 1 shows, for a person weighing 75 kg and not controlling food intake, expenditure of 400 Kcal/day for a month leads to a fat loss equivalent to .38 kg or .5% of body weight. Therefore, the actual fat loss is .09 kg/week or about one third the expected amount. Could all of this be accounted for by using inappropriate exercise intensities, poor genes, and lack of physical fitness? And furthermore, why is fat loss with exercise not greater given that exercise suppresses hunger in the short term and fails to produce compensatory caloric intake?

A contributing factor is very likely unrestrained eating which produces systematic overconsumption and preference for a higher than optimal content of fat in the diet. There is a demographic evidence for an increase in average food consumption in the USA over the last four decades from 3,100 in 1963 to 3,900 in 2004 (www.ers.usda.gov/Data/FoodConsumption/spreadsheets/nutrients.xls). This may be a consequence of many factors including an increased reliance on high-energy and high-palatability convenience foods (Paeratakul et al., 2003), larger serving sizes in restaurants provided on platters that have increased in size over time, the human tendency to eat more when more food is presented (Kral, Roe, & Rolls, 2004), and a significant prevalence of volumetric rather than energetic control of meal size (Rolls et al., 1999; Rolls, Bell, & Waugh, 2000; Rolls et al., 1998). For instance, both short-term studies (just mentioned) and studies carried out for as long as 11 weeks (Kendall, Levitsky, Strupp, & Lissner, 1991; Lissner et al., 1987) demonstrate that humans use volumetric cues in terminating their meals rather than the energy content of the meals.

The selection of macronutrients in the diet also affects the extent to which fat will be metabolized. Carbohydrate-rich meals elicit DIT largely because they stimulate insulin secretion and insulin-associated increase in carbohydrate metabolism. Fat-rich meals do not elicit as much insulin response or DIT and do not increase post-prandial fat oxida-

tion (Flatt, Ravussin, Acheson, & Jequier, 1985). Large mixed meals promote extra energy expenditure in the form of DIT, but the magnitude of DIT response is only 9% of the extra calories ingested (Weyer, Vojarova, Ravussin, & Tataranni, 2001). Thus fat-rich meals promote little energy expenditure and favor the deposition of fat within the adipose tissue.

One can conceptualize that human weight stabilizes at any number of different fatness plateaus as a result of two control systems operating in tandem but not closely interconnected. The first one is the feeding control system which is biased toward opportunistic excesses and thus tends to drive human body weight and body fat upward with a negative feedback provided more by the volume of food at individually satisfying satiation levels than by the calories ingested. The second system entails control of the habitual levels of physical activity that are influenced by occupational imperatives, genetic endowment, or voluntary choice. With changes in age, occupations, and even environmental factors that favor or hinder physical activity and allow greater access to energy-rich foods, body fat levels achieve different plateaus. This outcome is a consequence of insensitivity of feeding controls to both excess calories and exercise energy expenditure, and the non-homeostatic relationship between the physical activity and body fat. Fatness plateaus are a cumulative result of biological, social, and environmental variables that influence our eating and our levels of physical activity.

The corollary of this view is that exercise, particularly in the form of aerobic training, can be used for fat loss (Epstein & Wing, 1980; Forbes, 1985; Thompson, Wolfe, Eikelboom, 1988), but the process is more efficient when the exercise is coupled with dietary restraint or dietary restriction (Lewis et al., 1987; Svendsen, Hassager, & Christiansen, 1993). In most studies, a significantly faster rate of fat loss, but lower overall rate of weight loss, was achieved with exercise energy expenditure than with equicaloric dietary restriction (McMurray, Ben-Ezra, Forsythe, & Smith, 1985; Pavlou, Steffee, Lerman, & Burrows, 1985; Stefanick et al., 1998). However, in most of these studies with a few exceptions (Belko, Van Loan, Barbieri, & Mayclin, 1987; Strasser, Spreitzer, & Haber, 2007), exercise energy expenditure resulted in greater fat loss than when the weight loss is achieved by dieting alone (McMurray, Ben-Ezra, Forsythe, & Smith, 1985; Pavlou, Steffee, Lerman, & Burrows, 1985; Stefanick et al., 1998). Greater efficacy in maintaining weight loss after combined exercise-dietary restriction treatment (Pavlon, Krey, & Steffee, 1989) may be a consequence of the dampening effects of exercise on hunger and compensatory eating. A combination of exercise and dietary restriction as a means of weight loss has the added benefit of protection by

exercise of lean body mass and associated metabolic rate (Ballor & Poehlman, 1994; Leon et al., 1979; Pavlou et al., 1985; Svendsen, Hassager, & Christiansen, 1993; Wood et al., 1988), both of which decline during weight loss generated by dieting alone (Apfelbaum, 1978; Apfelbaum, Bostsarron, & Lucatis, 1971; Bray, 1969; Drenick & Dennin, 1973; Garrow et al., 1978; Krotkiewski, Toss Bjorntorp, & Holm, 1981; Weyer et al., 2001). The combined diet and exercise strategy has been successfully used by individuals listed in the National Weight Control Registry. They are entered in the registry if they have lost at least 13.5 kg and maintained this weight loss for at least one year. Their success in maintaining the fat loss resides in restricting their caloric intake to 1,400 Kcal per day, a low proportion of fat in their diet (25%), a tight monitoring of changes in body weight, and daily exercise for a minimum of 1 hour (McGuire et al., 1998).

Finally, even if exercise does not produce the desired rapid levels of fat loss, we should be reminded that increased fitness conveys many health benefits. Thus an aerobically fit obese individual has a lower risk of dying from cardiovascular and all causes than a lean aerobically unfit individual (Lee, Blair, & Jackson, 1999; Lee, Jackson, & Blair, 1998). Aerobic fitness leads to adaptive increases in cardio-respiratory function and in the mitochondrial mass in the muscles (Holloszy & Coyle, 1984; Hood, Irrcher, Ljubicic, & Joseph, 2006). An increase in the mitochondrial volume improves fat oxidation, reduces the production of reactive oxygen species that have been implicated in the etiology of type 2 diabetes and other co-morbidities associated with obesity (Saris & Heymsfield, 2007). Engaging in aerobic training accompanied with restrained eating is the road toward a leaner and healthier body and a healthier and longer life.

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KOLIKO JE TJELOVJEŽBA UČINKOVITA U POTICANJU GUBITKA MASNOG TKIVA?

Sažetak

Uvod

Posljednjih 25 godina razina pretilosti u razvijenim zemljama dramatično je porasla. Iako postoje mnogobrojni razlozi koji su doveli do promjena suvremenog životnog stila odgovornog za razvoj pretilosti, ovaj je pregledni članak usmjeren na podatke koji se tiču tjelovježbe kao sredstva za kontrolu tjelesne težine i gubitka masnog tkiva. Prikazana su istraživanja koja obrađuju sljedeće teme: (1) gubitak težine i količina izgubljenog masnog tkiva kada se primjenjuje tjelovježba, ali bez istodobnih promjena u unosu hrane, (2) volumen i intenzitet tjelovježbe koji su potrebni da bi se izgubila tjelesna masa i masno tkivo, ali i da bi se održala nova tjelesna masa, (3) utjecaj pretilosti na razinu spontane tjelesne aktivnosti i druge oblike potrošnje energije, (4) utjecaj pretilosti na sposobnost oksidacije masti i (5) utjecaj tjelovježbe na apetit.

Učinkovitost samog vježbanja u smanjenju tjelesne mase i razine tjelesne masti bez promjena unosa hrane

Da bi se postigao znatan gubitak masnog tkiva, potrebno je tijekom određena vremenskog perioda provoditi tjelovježbu znatnog volumena. Podaci iz dostupnih preglednih članaka, kao i čitav niz pojedinačnih studija, ukazuju na to da najveći dio istraživanja u kojima se tjelovježba primjenjuje bez istovremenih restrikcija prehrane dovodi do vrlo skromnih smanjenja tjelesne mase i masnog tkiva.

Kada se govori o učinku tjelovježbe, osim izravne energetske potrošnje izazvane vježbanjem, očekivani neizravni učinci tjelovježbe jesu porast metaboličke aktivnosti u mirovanju i porast prehranom izazvane termogeneze (DIT, prema engl. diet-induced thermogenesis). I ti učinci mogu pridonijeti ukupnom gubitku energije.

Volumeni i intenziteti tjelovježbe potrebni da bi se postigao i održao gubitak težine i masnog tkiva

Da bi se izgubio kilogram tjelesne masti, koja sadrži 87% lipida, potrebno je potrošiti oko 7830 kcal. Uz pretpostavku da osoba želi postići gubitak četvrtine kilograma masti tjedno, morala bi, u istom periodu, tjelovježbom potrošiti gotovo 2000 dodatnih kalorija. Uz pretpostavku da se tjelesna aktivnost provodi 5 dana u tjednu, potrebna potrošnja energije iznosila bi 400 kcal dnevno.

No, ako se vježbanje primijeni bez ikakvih promjena u prehrani, dnevna potrošnja od 400 kcal dovest će do gubitka masti tek u iznosu od oko jedne trećine vrijednosti očekivane na temelju kalorijskog deficita izazvanog tjelovježbom.

Utjecaj pretilosti i aerobnog fitnesa na sposobnost oksidacije masti

Dodatne komplikacije u primjeni vježbanja radi gubitka masnog tkiva čini genski i kondicijski status osobe. Danas je jasno da sklonost nakupljanju masnog tkiva ima gensku podlogu. Iako su konkretne brojke još uvijek predmet diskusije, većina autora genima pripisuje gotovo 60% tendencije nakupljanja masnog tkiva.

Molekularna podloga pojačane sklonosti pretilih osoba daljnjem nakupljanju, umjesto smanjenju, masne mase jest niži udio oksidativnih vlakana i smanjena mitohondrijska masa njihovih skeletnih mišića. U pretilih osoba je također manje izražen hormon masnog tkiva adiponektin, koji snažno stimulira mitohondrijsku biogenezu. Stoga su pretile osobe u svom pokušaju gubitka masnog tkiva hendikepirane smanjenom sposobnošću oksidacije masti, odnosno smanjenom sposobnošću stvaranja mitohondrija te većim oslanjanjem na metabolizam ugljikohidrata.

Drugi važan čimbenik koji određuje koji će udio tjelovježbom izazvane potrošnje energije činiti masti jest aerobni fitnes pojedinca. Aerobni trening ima dvije važne posljedice. Jedna je povećanje kardiorespiracijske i cirkulacijske sposobnosti dopremanja kisika do radnog mišićja. Druga je porast volumena mitohondrija. Aerobni trening također potiče porast ekspresije oksidativnih vlakana u skeletnim mišićima. Ukupno gledano, sve te adaptacijske promjene dovode do povećane sposobnosti treniranih mišića da oksidiraju masti, smanjenog stvaranja slobodnih radikala i cjelokupnog smanjenja rizika od raznih uzroka smrti.

Utjecaj pretilosti na spontanu tjelesnu aktivnost

Podaci iz istraživanja provedenih na ljudima ne podržavaju teoriju mehanizma povratne sprege između tjelesne aktivnosti i razine tjelesne masti te sugeriraju da spontana tjelesna aktivnost nije homeostatska komponenta mehanizma energetske regulacije. Studije pokazuju da su pretile osobe hipoaktivne. Eksperimenti sugeriraju da je hipoaktivnost vjerojatnije posljedica pretilosti nego genski predodređena osobina.

Utjecaj tjelovježbe na apetit

Brojne su studije opetovano pokazale da tjelovježba prolazno potiskuje osjećaj gladi i pojačava osjećaj sitosti. Potrošnja energije vježbanjem uzrokuje kratkotrajno obuzdavanje apetita, a u dužem vremenskom periodu ne dolazi ni do kompenzacije izgubljenih kalorija. Ovaj paradoks za sada još nije objašnjen. Ali to je fenomen koji može biti od velike koristi osobama koje tjelovježbom žele smanjiti količinu masnoga tkiva.

Sinteza dokaza i integrativna hipoteza o primjeni vježbanja u poticanju gubitka masti

Jasno je da prikazane činjenice ne pružaju koherentnu sliku o ulozi vježbanja u regulaciji energije. Koji razlozi leže u pozadini činjenice da bi 400 kcal energije, dnevno potrošene vježbanjem, tijekom 5 dana, bez ikakvih promjena u unosu hrane, trebalo biti dovoljno da dovede do gubitka od 0,25 kg masnog tkiva tjedno, a stvarni gubitak masti zapravo iznosi 0,09 kg/tjedno ili oko jednu trećinu očekivane vrijednosti? Čimbenik koji tome pridonosi jesu vrlo vjerojatno prehrambene navike, tj. sustavna pretjerana potrošnja hrane i sklonost prehrani čiji je sadržaj masti veći od optimalnog.

Odabir makronutrijenata u prehrani također utječe na mjeru do koje će mast biti metabolizirana. Obroci bogati ugljikohidratima izazivaju DIT najvećim dijelom jer potiču lučenje inzulina. Obroci bogati mastima ne izazivaju toliki inzulinski odgovor ni DIT te ne pojačavaju postprandijalnu oksidaciju masti. Obilni miješani obroci potiču dodatnu potrošnju energije u obliku DIT-a, ali veličina DIT odgovora iznosi samo 9% dodatno unesenih kalorija. Stoga obroci bogati mastima potiču malu potrošnju energije i pogoduju odlaganju masti u masnom tkivu.

Logičan zaključak svega prikazanog jest da se tjelovježba, osobito u obliku aerobnog treninga, može koristiti za smanjenje masnoga tkiva, ali da je taj proces učinkovitiji kada je vježbanje udruženo s ograničenjem unosa hrane. Kombinacija tjelovježbe i restrikcije prehrane, kao načina gubitka težine, ima dodatnu vrijednost u zaštiti i održanju nemasne tjelesne mase i povišenja metaboličke stope vježbanjem, jer tijekom gubitka težine izazvanog samo restrikcijskom dijetom, dolazi do pada obiju varijabli.

Konačno, čak i ako vježbanje ne dovodi do željenih brzih gubitaka masti i tjelesne mase, moramo se podsjetiti da povećani fitnes donosi mnoge zdravstvene dobrobiti. Tako preti loj osobi visoke aerobne sposobnosti prijeti niži rizik smrtnosti od kardiovaskularnih bolesti, kao i smrtnosti od drugih uzroka, nego vitkoj osobi niske aerobne sposobnosti. Aerobni fitnes izaziva adaptacijska poboljšanja kardiorespiracijske funkcije i povećanja mitohondrijske mase u mišićima, koja, pak, poboljšavaju oksidaciju masti. Bavljenje aerobnim vježbanjem, uz adekvatna ograničenja u prehrani, predstavlja pravi put prema vitkijem i zdravijem tijelu te zdravijem i dužem životu.