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NARRATIVE REVIEW

Can Regular Physical Activity Prevent Obesity? Part 1: Potential factors reducing the efficacy of physical activity.

Roy J. Shephard^{1,*}

1 Faculty of Kinesiology and Physical Education, University of Toronto, Toronto, ON, Canada

*Corresponding Author: royjshep@shaw.ca

Abstract

Objective: The objective of this narrative review is to consider possible factors limiting the efficacy of regular physical activity as a means of preventing the development of obesity and/or treating established accumulations of body fat. **Methods:** Information obtained from Ovid/Medline and Google Scholar through to December 2018 was supplemented by a search of the author's extensive personal files. **Results:** In the early years of human history, humans probably evolved mechanisms to reduce energy expenditures during periods of severe food shortage. Adaptations could have included a decrease in resting energy expenditures and/or the temporary elimination of all except essential physical activities. However, it is less certain that such mechanisms operate when a person who has an excess of body fat conforms to rigid dietary restrictions and/or undertakes a rigorous exercise programme. Severe dieting may indeed lead to some reduction of resting metabolism, but such effects are commonly attenuated if not fully reversed by the inclusion of an exercise programmes that conserve lean tissue mass. There is also little evidence that exercise programmes stimulate appetite or reduce spontaneous leisure activity. **Conclusions:** An excess of adipose tissue is usually accumulated over several years. If one views the prevention and/or correction of obesity by an increase of physical activity as a similar long-term project, the required exercise is within the capacity of those who are overweight. There is no good reason why a well-designed exercise programme cannot optimize body composition, as well as offering other important health benefits; however, as with rigorous dieting, the challenge to health professionals is to sustain enthusiasm until this has been accomplished. **Health & Fitness Journal of Canada 2019;12(2):13-63.**

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Introduction

Many physical educators and kinesiologists have assumed, almost as a central component of their faith, that adequate and regular amounts of physical activity provide an effective means of preventing the development of obesity in their clients. Certainly, one finds few obese individuals in professional associations dedicated to kinesiology or sports medicine, and cross-sectional

comparisons between active and sedentary individuals generally support such an idea; however, there are as yet relatively few longitudinal studies to substantiate this notion. Investigators seeking the reasons behind a trim body figure face the perennial problem of disentangling the intertwined influences of a high level of physical activity, a well-regulated diet, other facets of a healthy lifestyle, and genetic predisposition

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among those achieving and maintaining an appropriate body build (Shephard, 2019b).

In this narrative review, we look critically at the potential contributions of a high level of habitual physical activity and of organized exercise programmes to the prevention of obesity, analyzing the various factors that could potentially limit the efficacy of such an approach. A more detailed discussion of the anticipated role of physical activity in the control of obesity can be found in a Cochrane Library (Shaw, Gennat, & O'Rourke, 2006).

We focus particularly on several issues that many nutritionists have raised concerning the likelihood that adequate and regular physical activity programme can contain and even reduce body fat content (Epstein & Wing, 1980; Jakicic, Clark, & Coleman, 2001; Malhotra & Noakes, 2015; Shaw et al., 2006). For example, Miller et al. (1997) argued that among 493 clinical trials which had continued for an average of 24 weeks, exercise programmes had typically yielded a weight loss of less than 3 kg, as compared with an immediate loss of 11 kg achieved over 15 weeks of dieting. Nevertheless, they admitted that after the elapse of one year, the dieters had lost their early advantage. Likewise, a review of programmes for the prevention of obesity in children and adolescents concluded that exercise had significant value in only 13 of 64 studies (Slice, Shaw, & Marti, 2006).

Those arguing against the value of

exercise as a means of preventing obesity have highlighted a number of factors potentially limiting the efficacy of such treatment (Table 1). Issues include the seemingly large total energy expenditure that is needed to reduce body fat content by a clinically significant amount, the likely stimulation of appetite and food intake when a person engages in a bout of vigorous exercise, possible reductions in both resting metabolic rate and spontaneous leisure activity that tend to cancel out a negative energy balance brought about by any combination of dieting and vigorous exercise, and a tendency of very active individuals to place a cap on their daily energy expenditures.

We will here address each of these issues in turn. A companion to the present review will consider the extent of empirical evidence demonstrating the effectiveness of regular physical activity in preventing and treating obesity.

Magnitude of the energy expenditure needed to induce a clinically significant loss of body fat

In addressing the question of the volume of physical activity needed to induce a clinically significant change in body fat content, we begin by considering some fundamentals of energetics in the context of obesity, and we then look at empirical relationships between exercise and fat loss, before determining whether an obese person can undertake a sufficient amount of physical activity to have a

Table 1. Reasons that have been suggested as to why regular physical activity has a limited efficacy in controlling obesity.

- A large expenditure of energy is needed to metabolize a small amount of fat
- Vigorous exercise is likely to stimulate appetite and thus food intake
- Potential energy deficits created by an exercise programme are largely compensated by a reduction of resting metabolic rate
- Involvement in an exercise programme may also be compensated by a reduction in various forms of voluntary leisure activity
- Very active populations seemingly place a cap on their daily energy expenditures

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clinically significant impact in the control of obesity.

Fundamentals of energetics in the context of obesity

How large a volume of daily physical activity is needed to prevent an age-related accumulation of body fat and/or to correct established obesity? Approximate answers to these questions can be derived from basic considerations of energetics. In essence, fat is lost if the daily energy expenditure exceeds the intake of food energy (although in the short-term there is also potential for some breakdown of lean tissue, particularly if severe dieting is unaccompanied by either exercise or protein supplements).

Many trials with an exercise component have sought to increase energy expenditures of obese clients and thus their negative energy balance by involving the individuals concerned in regular brief bouts of walking at a pace acceptable that is acceptable to them. The net energy expenditure needed to walk a distance of 1.6 km at a pace of 5 km/hr is about 370 kJ for a man and 310 kJ for a woman (Hall, Figueroa, & Fernhall, 2004). This is an intensity of physical activity that is commonly prescribed for participants in a modest obesity rehabilitation programme. Over the course of a 30-minute walk at a speed of 5 km/hr, the net increase of energy expenditure for a healthy person totals at least 578 kJ in a man and 484 kJ in a woman; moreover, because most obese people enter a programme with an above average body mass, their energy expenditures will likely be larger than those for an individual with an "ideal" body mass.

How much body fat is metabolized by an energy expenditure of this order? Classical studies by nutritionists such as

Rubner in Berlin and his pupils Atwater and Benedict at the Boston Nutrition Laboratory demonstrated that when one gram of pure fat is burnt in a bomb calorimeter, it yields around 9.3 kCal, (39.5 kJ) of energy (Table 2). However, because adipose tissue contains not only stored fat but also protein and water; thus, one gram of adipose tissue yields only about 7 kCal (29 kJ) of energy. If a person were to walk for 30 min per day at a pace of 5 km/hr, the gross energy cost would amount to about 5 kCal/min (21 kJ/min), and the total energy expenditure would be increased by some 90 kCal (375 kJ) over the course of the exercise session, equating to a loss of 13 g of adipose tissue. Other factors being equal, if an adult were to engage in 30 min of moderate walking on each of 77 days, the cumulative additional energy expenditure of 28.9 MJ would equate to the metabolism of 1 kg of fat. However, if such a programme were to be terminated after 20-30 weeks (as in many graduate thesis projects), the decrease of body mass would be unlikely to exceed 2-3 kg.

Hall (2008) suggested the generalization that a cumulative energy deficit of 32.2 MJ was required to reduce body mass by 1 kg. He further underlined that elements other than adipose tissue often contributed to the weight loss, particularly lean tissue protein. The lean tissue component of overall weight loss is greater in thin individuals than in those who are fat, and since lean tissue has a substantially smaller energy yield than an equivalent weight of fat, thin people can

Table 2: The energy yielded by the combustion of selected fats (Rubner, 1885; Widdowson, 1955).

Type of Fat	Heat of combustion (kCal/g)
Olive oil	9.184
Animal fat	9.372
Butter fat	9.179

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induce a 1 kg decrease of body mass by a smaller total energy expenditure than those who are obese. For the same reason, men lose somewhat more weight than women for any given energy expenditure.

Empirical data on successful weight-loss programmes

The U.S. National Weight Control Registry found that individuals who were successful in maintaining a substantial weight loss were walking an average of 45 km/week (Klein, Wing, & McGuire, 1997). Assuming they engaged in activity on a daily basis, this could be accomplished by covering 6.4 km/day at a pace of 5 km/hr over a 77-minute session.

However, others have suggested that a stable body mass can be maintained with a smaller daily volume of physical activity than this. A doubly-labeled water trial indicated that either 80 min per day of moderate physical activity or 35 min per day of vigorous exercise was sufficient to prevent women who had previously been obese from regaining an excessive body mass (Schoeller, Shay, & Kushner, 1997). Likewise, a consensus meeting that was held in Bangkok (Saris, Blair, & Van Baak, 2003) concluded that 45-60 min/day of moderate physical activity was likely enough to prevent the development of obesity in adults. Further, individuals who were formerly obese could maintain their weight loss by engaging in moderate physical activity for 60-90 min per day. However, greater volumes or intensities of physical activity were probably necessary to counter obesity in children.

On the basis of these reports, an official Position Statement of the American College of Sports Medicine (ACSM) recommended that in order to prevent obesity, adults should undertake at least 150-250 min of moderate to vigorous

exercise per week, equivalent to a total added energy expenditure of 4.8- 8 MJ per week (Donnelly, Blair, & Jakicic, 2009). Note that if the activity sessions were to be limited to 200 min per week, or 28.5 min per day, this would imply adopting a net exercise expenditure of 24-40 kJ/min, as compared with the 16-19 kJ/min that would have been anticipated if walking at a pace of 5 km/hr. On the other hand, even if the goal of 8 MJ/week was essential in order to ensure therapeutic success, this could still be realized by walking for a little over 60 min per day at a pace of 5 km/hr.

Can an obese person achieve a sufficient volume of activity to control body weight?

Despite the carefully considered conclusions of the consensus group, many nutritionists still argue that the daily volume of exercise needed to prevent weight gain and/or to induce a significant loss of body fat is larger than the average sedentary or inactive person is likely to accept, and in the case of someone who is already obese it may be an impossible assignment.

Such a viewpoint reflects the search for a treatment that will perform the miracle of correcting gross obesity within a few weeks. It ignores the fact that most obese people have accumulated an excess of body fat by maintaining a slight deficiency of physical activity or an equally small degree of over-eating for a period of 10 or even 20 years. Thus, the appropriate approach to both prevention and correction of obesity lies in an equally small but persistent change in the individual's energy balance. Certainly, the ACSM proposals seem reasonable and offer objectives that can be attained by the typical individual with moderate obesity.

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Many nutritionists have also thought about therapeutic options in terms of the treatment required by patients who have reached the Grade III level of obesity, people who are carrying 50 kg or more of excess fat. However, such individuals are in the minority. Most people who attend a health facility in order to reduce their weight are slightly rather than grossly obese, carrying perhaps 10-15 kg of excess body fat (Table 3). Moreover, in the absence of possible compensatory reductions in resting and non-exercise active energy expenditures (as discussed below), if 30 min of moderate physical activity were to be maintained 5 times per week for one year, there would be a cumulative fat loss of 5.0 kg in men and 4.2 kg in women. In keeping with these estimates, Epstein & Wing (1980) found a 1.15 kg weight loss over a typical 12-week moderate exercise programme, and Miller et al. (1997) saw a 2.9 kg decrease in body mass over a 24-week programme. Plainly, if such a programme were to be sustained for 2-3 years, the 26 kg excess of fat found in a person with Grade 1 obesity would be totally corrected.

Further, if a moderately obese person were able to undertake a larger volume or a more intensive type of exercise on a systematic basis, or if they were to add a modicum of dietary restraint, the fat loss would occur within less than 2-3 years. With an hour of moderately vigorous exercise per day (an *added* energy expenditure of 2.0-2.8 MJ/day), weight losses of 8 kg in men (Ross, Dagnone, & Jones, 2000) and 6 kg in women (Ross, Janssen, & Dawson, 2004) have been achieved over as little as 12 weeks.

Nevertheless, clinically significant benefit can only be anticipated if physical activity is pursued on a regular basis, and is sustained for longer than the typical

Master's Thesis experiment. Those critical of exercise programming are at least correct in their contention that the occasional bout of moderate walking is unlikely to restore a normal body composition in those who are currently grossly obese, even if the programme is accompanied by other therapeutic measures, including substantial dietary restrictions. Unfortunately, many of those who have become grossly obese are reluctant to engage in the requisite additional volume of physical activity on a daily basis. As with dietary programmes, the big challenge for health professionals is to maintain interest in a new, health-oriented lifestyle for several years.

Supposed stimulation of appetite by exercise programmes

It would plainly be possible to negate the benefits of a well-designed exercise programme by creating a positive energy balance through an increased intake of food. One review looked at an apparently disappointing response to some exercise interventions, and it concluded that the 2 dominant causes of failure to lose weight were an inadequate dose of exercise and an increased intake of food stimulated by

Table 3: Excess body fat content in relation to body mass index (BMI). Data for individuals with a standing height of 1.7 m.

Category of BMI	BMI (kg/m ²)	Total body mass (kg)	Excess fat mass (kg)
Normal weight	23.5	67.9	0
Overweight	27.5	79.5	11.6
Obesity Grade I	32.5	93.9	26.0
Obesity Grade II	37.5	108.4	40.5
Obesity Grade III	42.5	122.8	54.9

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the increase in physical activity (Thomas, Bouchard, & Church, 2012). However, any relationship between physical activity and appetite is not yet clearly established, in part because it is difficult to obtain accurate measures of food intake by the usual approach of 3-day or 7-day dietary records.

A brisk bout of exercise induces an increased secretion of adrenaline, and this should raise blood sugar, creating an immediate feeling of satiety rather than hunger. In support of this view, the majority of studies looking at subjective ratings of appetite, estimates of overall food intake, or changes in hormonal control mechanisms have all pointed towards an early decrease in appetite following a typical bout of moderate exercise, to the point that some authors have talked of an exercise-induced anorexia (King, Tremblay, & Blundell, 1997; Westerterp-Plantegna et al., 1997). However, the findings relative to food intake and blood levels of appetite-regulating hormone levels have been less consistent, and some long-term studies have found an increased intake of foods as body mass has decreased.

Subjective ratings of appetite

Analogue scales are commonly used to provide a subjective estimate of sensations such as hunger, appetite, satiety and "fullness." A number of authors have used this approach to assess possible changes in these sensations in response to an acute bout of physical activity (Table 4). Of the 14 reports listed, 10 show a decrease of hunger and appetite during and for up to 2 hr following a bout of exercise, in 4 there is no change; in only one study there is a possible increase of appetite in obese but not in lean individuals, and in another report appetite

is increased by 2 hr of athletics but not by 75 min of swimming.

In the few instances where comparisons have been made, higher intensities of effort have been more likely than moderate physical activity to suppress appetite.

Looking at individual studies, Bilski et al. (2013) evaluated the effects of both moderate exercise (30 min of cycle ergometry at 30% of maximal oxygen intake) and a bout of high intensity exercise (completion of a firefighter fitness test with aerobic and strength items) in 12 healthy young men. Exercise diminished reported sensations of hunger and the motivation to eat, significantly so for 90 min following the high intensity effort (particularly if an ad-libitum meal was available after exercise).

Broom et al. (2007) noted that hunger ratings were reduced for 3 hr when a group of 9 lean young men ran on a treadmill for 60 min at a speed demanding 72% of their maximal oxygen intakes. Burns et al. (2007) also reported decreased sensations of hunger in healthy young adults (9 men and 9 women) for 2 hr following a similar pattern of exercise.

Hagobian et al. (2013) had lean and healthy young adults (11 men and 10 women) exercise at 70% of their peak oxygen intake until they had spent 30% of their daily energy expenditure (an effort amounting to a total of about 3 MJ in the women, and 4 MJ in the men). This volume of physical activity induced no change in ratings of hunger, satiety or fullness.

Holliday & Blannin (2017) had 8 overweight individuals perform a succession of 4 Wingate 30-second all-out sprints on a cycle ergometer. They observed a significant suppression of appetite ratings for at least 30 min

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following this period of exhausting exercise.

King et al. (1994) measured feelings of hunger both by using a visual analogue scale, and by timing the interval to voluntary eating post-exercise. They found a small suppression of appetite following moderate exercise (60 min of cycle ergometry at 30% of maximal aerobic power), and a much greater but also short-term (<30 min) suppression of appetite after a more intensive bout of exercise (30 min at 70% of the individual's maximal aerobic power).

The volunteers recruited by Kissileff et al. (1990) included both lean and obese women. Each group undertook both low intensity (30W) and high intensity (90W) cycle ergometry. In the lean women, hunger ratings were unchanged by either bout of exercise. Data were harder to interpret for the obese group; hunger ratings appeared to increase with vigorous exercise, but at the same time there was a significant reduction in the volume of a test meal that was ingested post-exercise.

Martins et al. (2007) had 12 lean young volunteers undertake an hour of moderate exercise at a heart rate of ~130 beats/min; they found that ratings of hunger were reduced during the period when they were actually exercising.

Pomerleau et al. (2004) tested 17 non-obese young women, finding no change in appetite ratings with the performance of a total of 350 kCal (1.46 MJ) of treadmill exercise, whether the energy expenditure was accumulated at an intensity of effort demanding 40% or 70% of the individual's peak oxygen intake.

Reger et al. (1984) worked with non-obese women, finding that 60 min of moderate treadmill exercise (an intensity 50% of maximal oxygen intake) reduced ratings of appetite in their subjects.

Reger et al. (1984) also observed a brief reduction of hunger after 30 min of exercise at 50% of maximal oxygen intake followed by 30 min of exercise at intensities alternating between 40% and 70% of maximal oxygen intake.

Verger et al. (1992; 1994) found no change in the hunger ratings of 13 young and healthy students with 75 min of swimming, but in contrast a 2-hour bout of athletics led to increased ratings of appetite that persisted for 60 min.

Westerterp-Plantegna et al. (1997) evaluated sensations of hunger in young adults, both obese and non-obese, following 2 hr of cycle ergometry at 60% of peak power output. Hunger was decreased relative to controls immediately following this bout of exercise.

In summary, with the exception of the findings of Verger et al. for prolonged athletics (1994), and the possible adverse effect observed by Kissileff et al. (1990) in obese subjects, a bout of moderate or vigorous aerobic exercise has left appetite either unchanged, or more commonly temporarily suppressed. More information is needed on the effects of exercise intensity, but in the few instances where comparisons have been made, higher intensities of effort seem to have increased rather than decreased the suppression of appetite. Often, the effect has been quite short-lived, and there remains a need to examine chronic responses, both for longer periods following a single bout of exercise, and in response to involvement in a continuing exercise programme. Further, more information is required on possible differences of response between lean and obese individuals. Finally, are there any differences in findings, depending upon the type of physical activity that is performed?

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Table 4: Influence of exercise upon sensations of hunger.		
Author	Subjects and exercise	Findings
Bilski et al. (2013)	12 healthy young men, moderate (30 min at 30% max. ox. intake) vs. high intensity effort (fire-fighter fitness test)	Decrease of hunger, significant after high intensity exercise
Broom et al. (2007)	9 lean young men, 60 min run at 72% of maximal oxygen intake	Lower ratings of hunger for 3 hr
Burns et al. (2007)	Young healthy adults (9 men, 9 women (60 min treadmill ex, at 73.5% maximal oxygen intake)	Sensations of hunger depressed for 2 hr post-exercise
Douglas et al. (2017)	47 lean and 25 obese adults, 60 min treadmill exercise at 59% maximal oxygen intake	Exercise suppressed appetite similarly in lean and obese individuals
Hagobian et al. (2013)	Lean and healthy young adults (11 men, 10 women) exercised at 70% of peak oxygen intake to total of 30% of daily energy expenditure	No significant effect on hunger, satisfaction or feelings of fullness
Holliday & Blannin (2017)	8 overweight individuals performed 4 Wingate test 30 sec sprints	Suppression of appetite ratings for 30 min following exercise
King et al. (1994)	23 lean males: 60 min of cycle ergometry at 30% of maximal aerobic power, or 30 min at 70% of maximal aerobic power	Feelings of hunger suppressed during and after exercise, subjects waited longer before eating; greater hunger suppression with higher effort intensity
Kissileff et al. (1990)	40 min of moderate (30W) or heavy (90W) cycle ergometer exercise in 9 obese, 9 non-obese women aged 18-35 yr	Lean subjects show no change of hunger ratings; possible increase in obese individuals (but less of test meal ingested)
Martins et al. (2007)	12 lean young adults, 60 min of cycle ergometry at heart rate of ~130/min	Hunger depressed during exercise
Pomerleau et al. (2004)	350 kCal total exercise at 40% or 70% of peak oxygen intake in 17 young non-obese women	Visual analogue scales show no change of appetite relative to control
Reger et al. (1984)	60 min treadmill at 50% maximal oxygen intake, or 30 min at 50% + 30 min alternating 40/70%, non-obese women	Brief depressions of hunger and appetite ratings
Thompson et al. (1988)	15 lean young men, Cycle ergometry, 35% or 70% maximal oxygen intake	Feelings of hunger suppressed briefly by higher intensity effort
Verger et al. (1992; 1994)	13 healthy young students 75 min of swimming, or 2-hour bout of athletics	No change of hunger ratings with swimming, but appetite increased by 2 hr of athletics
Westerterp-Pnategna et al. (1997)	30 young adults, both obese and non-obese, 2 hr cycling at 60% peak power output	Hunger decreased immediately post-exercise

Empirical data on food intake following exercise

The type and quantity of food ingested might seem a helpful indication of changes in appetite following a bout of exercise. Often, estimates have been based upon short-term dietary questionnaires, or investigators have watched selections made from a buffet table during the immediate post-exercise period, but a few

authors have made measurements of longer-term (7-14 day) changes in metabolism, using doubly-labeled water. This last approach is well suited to looking at the chronic rather than the acute effects of exercise upon total energy consumption.

Perhaps because estimates of food intake reflect relatively long-term reactions to exercise, whereas ratings of

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appetite are based on the more immediate responses, findings from the 2 approaches differ somewhat. Of 18 studies of food intake, only 4 showed a decrease following exercise, mostly with some qualifications. In 9 reports there was no change of food consumption, in 5 studies there was a clear increase, and in 5 other investigations there was an increase under some circumstances (Table 5). Differences in response seem linked to the sex of the subjects, their body fat content, and the intensity and type of effort that was performed.

Considering individual reports, Bilski et al. (2013) evaluated the effects of both moderate and of high intensity exercise bouts upon food intake during the 30 min following the activity. In contrast to the immediate decrease of appetite ratings (noted by the same subjects, above), food intake was increased, particularly following performance of the high intensity firefighters' physical fitness test.

A 6-month study of 94 obese post-menopausal women (average age 57 years, BMI 31.4 kg/m²) suggested that compensatory mechanisms such as an increased food intake were only likely to develop if the weekly volume of additional exercise exceeded a certain threshold (Church et al., 2009). The subjects of this experiment were divided among controls and 3 groups of exercisers who expended an additional 16, 32 or 48 kJ of energy per kg of body mass per week at an intensity of effort demanding about 50% of the individual's peak oxygen intake. With the lower 2 volumes of weekly exercise, the decrease in body mass over the 6-month programme closely matched the value predicted from the increase in daily energy expenditures, but in those who were spending an extra 48 kJ/kg per week, the decrease in body mass was only

1.5 kg, rather than the expected 2.7 kg. However, the discrepancy in weight reduction does not seem due to any stimulation of appetite. Indeed, the final daily food intake of all 3 subject groups, as estimated rather crudely from a simple questionnaire, was less than at baseline (-1.12 MJ/day, -1.65 MJ/day, and -1.32 MJ/day respectively). At least a part of the discrepancy in weight loss for the most active group could reflect the synthesis of additional lean tissue, rather than any failure to metabolize the expected amount of body fat. Mechanisms underlying any residual compensation for a large negative energy balance remain unclear, but other factors could include a reduction of resting metabolic rate or a decrease in other leisure activities (see further discussion below).

Cox et al. (2003) divided a sample of 60 sedentary overweight middle-aged men between light and vigorous exercise programmes, with or without associated dietary controls. Over a 16-week intervention, the self-selected food intake of the light exercise group decreased by 3.7%, but in contrast the heavier exercise group (who were performing three 30-minute sessions/week at 65-75% of maximal power output) increased their food intake by 10.7%.

Douglas et al. (2017) had 47 lean and 25 obese adults participate in 60 min of treadmill exercise at 59% of their maximal oxygen intakes. This substantial bout of exercise had no significant influence on the ad-libitum food intake in either lean or obese participants. Hagobian et al. (2013) also found that when lean and healthy young adults (11 men, 10 women) exercised at 70% of their peak oxygen intakes, accumulating a 30% increase in their daily energy expenditures, there was no significant increase in ad-libitum food

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Table 5: Influence of participation in exercise programmes upon daily food intake.		
Author	Subjects and exercise	Findings
Bilski et al. (2013)	12 healthy young men, moderate (30 min at 30% max. ox. intake) vs. high intensity effort (fire-fighter fitness test)	Increased intake of test meal 15 and 30 min post-exercise especially with high intensity ex.
Church et al. (2009)	411 obese post-menopausal women: controls & energy expenditures of 4, 8 or 12 kCal per kg each week	Two lower intensities led to predicted weight loss, but only 56% of expected loss at highest intensity-? synthesis of lean tissue (no increase of food intake)
Cox et al. (2003)	60 sedentary overweight middle-aged men; light or vigorous exercise, with or without dietary restriction	Food intake decreased by 3.7% with light exercise, but increased 10.7% with heavier exercise (30 min at 65-75% power output 3 times/week).
Douglas et al. (2017)	47 lean and 25 obese adults, 60 min treadmill exercise at 59% maximal oxygen intake	No significant change in <i>ad-libitum</i> food intake in either lean or obese.
Hagobian et al. (2013)	Lean and healthy young adults (11 men, 10 women) exercised at 70% of peak oxygen intake to total of 30% of daily energy expenditure	No significant increase in <i>ad-libitum</i> energy intake, exercise causes net expenditure of energy
Holliday & Blannin (2017)	8 overweight individuals performed 4 Wingate test 30-sec sprints	<i>Ad-libitum</i> food intake unchanged 2 hr after exercise
Kissileff et al. (1990)	40 min of moderate (30W) or heavy (90W) cycle ergometer exercise in 9 obese & 9 non-obese women aged 18-35 yr	134g/754g reduction of test meal intake in non-obese women after strenuous exercise, no change of intake in obese.
Martins et al. (2007)	12 lean young adults, 60 min of cycle ergometry at heart rate of ~130/min	Energy intake 16% greater than controls (+151 kCal, ~30.7% of added energy expenditure)
Pomerleau et al. (2004)	350 kCal exercise at 40% or 70% of peak oxygen intake in 17 young non-obese women	Food intake not increased by light exercise, but increased by 127 kCal with vigorous intensity exercise
Pritchard et al. (1997)	58 middle-aged overweight men- worksite programme of exercise (3 sessions/wk at 65-75% maximal heart rate), diet or control	2.6 kg weight loss in exercisers, 6.4 kg in dieters; but food intake of exercisers increased only 3.1% as against a 14.6% increase of energy expenditure
Staten (1991)	20 lean young adults, undertaking 60 min of exercise per day for 5 days at ~68% of maximal oxygen intake	Food intake unchanged in women, but in men increased by ~2900 kCal (about a third of the added energy expenditure)
Stubbs et al. (2002)	Six lean women, 7 day periods of control, moderate and vigorous exercise	33% of the added energy expenditure compensated by an increase of <i>ad-libitum</i> food intake
Thompson et al. (1988)	15 lean young men, Cycle ergometry, 35% or 70% maximal oxygen intake	Food intake unchanged by either exercise intensity
Verger et al. (1992; 1994)	28 non-obese males: 75 min of swimming or 2 hr of athletic activity	Swimming did not affect food intake, but athletics increased it by 440 kCal (about 50% of energy expended in exercise)
Westerterp-Plantegna et al. (1997)	30 young adults, both obese and non-obese, 2 hr cycling at 60% peak power output	Food intake decreased immediately following this exercise
Westerterp et al. (1992)	32 sedentary but not obese adults training 40 weeks for half-marathon	Doubly-labeled water estimates of food intake: decreased by 1.6 MJ/d in men, n.s. increase of 0.4 MJ/d in women
Woo et al. (1982)	3 obese women, 57 days of moderate exercise (125% increase of daily energy expenditure)	No compensatory increase of food intake with exercise
Woo et al. (1985)	5 non-obese women, energy expenditures increased to 114% and 122% of sedentary values, each for 17 days	In contrast to obese women, food intake increased by comparable amounts to added energy expenditure

intake; thus, the exercise bout was effective in causing a substantial net decrease in body energy reserves.

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Holliday & Blannin (2017) had 8 overweight individuals perform a series of four 30-second all-out Wingate Test sprints on a cycle ergometer. Despite an initial suppression of appetite (as noted above), 2 hr after they had completed this high-intensity exercise, the ad-libitum food intake was unchanged relative to control data.

Kissileff et al. (1990) explored the influence of 40 min of moderate (30W) or vigorous (90W) cycle ergometer exercise on the fraction of a test meal that was ingested 15 min post-exercise. In the lean women, the vigorous exercise was followed by a significant decrease in the quantity of ingested fluid (620 vs. 754 g), but the intake of the obese women was unchanged following either of the two exercise bouts (532 vs. 581 g)

Martins et al. (2007) had 12 lean young adults undertake 60 min of moderate cycle ergometry. The energy content of the food that was subsequently eaten from a buffet table was 151 kCal (0.63 MJ) more for the exercisers than for the control subjects, accounting for 30.7% of the energy expenditure added by the daily exercise session.

Pomerleau et al. (2004) evaluated 17 non-obese young women. They performed 350 kCal of exercise on the treadmill at intensities demanding 40% and 70% of their peak oxygen intakes. The lower intensity of effort caused no significant increase of food intake (as determined by the items eaten from a finite supply), but following the more vigorous bout of exercise, food intake was increased by 127 kCal (0.53 MJ), thus compensating for about a third of the exercise energy expenditure.

Pritchard et al. (1997) examined responses to worksite health programmes in 58 middle-aged and overweight men.

The sample was divided between groups undertaking aerobic exercise (3 sessions/ of aerobic exercise at 65-75% of maximal heart rate), dieting and control groups. The dieters showed a greater weight loss than the exercisers (6.4 vs. 2.6 kg) over the 12-month programme, but the food intake of the exercisers increased by only 3.1% over the 12 months, as against a 14.6% increase in their daily energy expenditure, thus allowing them to metabolize a substantial quantity of excess fat.

Staten (1991) found a sex difference in her data; 20 lean young adults were exercised at ~68% of their individual maximal oxygen intakes, 1 hour per day for 5 days. The men increased their food intake by about a third of the added energy expenditure, but in the women the food intake remained unchanged over the 5 days of observation.

Stubbs et al. (2002) exposed six lean women to successive periods of control conditions, moderate (1.9 MJ/day) and heavy (3.4 MJ/day) exercise, finding that under conditions of ad-libitum food intake, about a third of the energy expenditure added by each form of exercise was compensated by increased eating.

Verger et al. (1992; 1994) examined the effects of 75 min of swimming and of 2 hr of athletics leading to an added energy expenditure of some 500 kCal (2 MJ) in a group of 13 lean young adults. The bout of swimming did not modify food intake, but there was a 440 kCal increase of food consumption after the athletic activity.

Westerterp-Plantegna et al. (1997) evaluated 30 young adults, both obese and non-obese, following 2 hr of cycle ergometry at an intensity demanding 60% of the individual's peak power output. Immediately following this bout of

activity, food intake was decreased. Another report (Westerterp et al., 1992) looked at longer-term findings in 16 men and 16 women, previously sedentary but not obese, who engaged in 40 weeks of hard physical training in preparation for a half-marathon run. Food intake, as determined by the doubly-labeled water technique, provided no strong support for the view that the strenuous training had caused any compensatory increase in appetite. Indeed, over the course of the 40 weeks of intense conditioning, daily energy intake tended to decrease in the men (at week 40, the intake was 1.6 MJ/day lower than initial values), whereas in the women there was a non-significant trend towards a small increase in intake (0.4 MJ/day) (Westerterp et al., 1992).

The response of the individual may depend in part on the extent of initial fat stores relative to the requirements of the imposed physical activity, either in terms of its intensity or its total energy demand. Thus, Woo et al. (1982) found that 57 days of walking at 125% of a person's daily sedentary energy expenditure had no effect on the food intake of obese females. On the other hand, 5 non-obese women who undertook the same activity showed increases in food intake that fully compensated for their added energy expenditures (Woo & Pi-Sunyer, 1985). But against these observations, Kissileff et al. (1990) found that shorter-term exercise reduced food intake in lean but not in obese women.

In summary, dietary studies have generally covered a longer post-exercise period than subjective ratings of appetite, and perhaps for this reason they have given a rather different indication of human responses to an increase of energy expenditures than the subjective data. In most studies, exercise has induced little

change in the ingestion of food, but in some situations (particularly when lean adults have engaged in high intensity exercise), increased eating has compensated for as much as 30% of the energy expenditure added by exercising.

Studies of control mechanisms

A third approach when assessing possible methods of compensating for exercise-induced energy expenditures is to examine changes in the secretion of some of the hormones that regulate appetite and sensations of hunger (Table 6). Dominant interests are levels of the gastric-secreted and appetite-stimulating "hunger hormone" ghrelin, and the satiety-inducing hormone leptin that is produced by the adipose tissue cells. Other relevant mechanisms include changes in blood levels of the polypeptide YY (secreted by the ileum and colon, and reducing appetite), pancreatic polypeptide (secreted by the PP or F cells of the pancreas, with functions that include acting as a satiety factor), the glucagon-like peptide GLP-1 (produced in the distal ileum and colon, and also increasing satiety), and an increase of sympathetic nerve activity.

In keeping with the evidence presented above, that exercise does not greatly modify appetite, blood levels of ghrelin and leptin show little change in response to bouts of moderate physical activity.

Ghrelin. An increase of blood ghrelin levels and a resulting increase of appetite are well-recognized consequences of dieting when this is not accompanied by exercise. In contrast, the majority of studies have reported that moderate to vigorous bouts of running have little impact on circulating levels of ghrelin (Dall, Kanaley, & Hansen, 2002; Douglas et al., 2017; Hagobian et al., 2013; Kallio,

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Pesonen, & Karvonen, 2001; Kraemer, Durand, & Acevedo, 2004; Martins et al., 2007, Schmidt et al., 2004). Indeed, ghrelin concentrations have sometimes actually fallen after a period of exercise, possibly due to the suppressant effect of increased sympathetic nerve activity as the intensity of effort rises (Shiia et al., 2011).

From a physiological point of view, it has been argued that the relevant information is not the total blood level of ghrelin, but rather its acylated fraction (since the latter can cross the blood-brain barrier, and thus act on the hypothalamic centres that regulate appetite). Some of the more recent studies have demonstrated that vigorous exercise can reduce the production of acylated ghrelin for several hours, discouraging excessive eating following a bout of exercise, although such a response is not always revealed by measurements of total ghrelin (Shiia et al., 2011). Two reports, apparently on a similar population, illustrate the potential for drawing opposite conclusions from determinations of total and acylated ghrelin (Broom et al., 2007; Burns et al., 2007). Looking at details of individual investigations, Bilski et al. (2013) reported a decrease of total ghrelin levels in 12 healthy young men for 30 min following a bout of vigorous exercise (the completion of an aerobic and muscular strength testing firefighters test), although they saw no response with more moderate exercise.

Broom et al. (2007) found reduced levels of acylated ghrelin for 3 hr after 9 lean young men had run for 60 min at a speed demanding 72% of their maximal oxygen intakes. In contrast, Burns et al. (2007) carried out a very similar study in healthy young adults (9 men and 9 women), but found no change in total ghrelin levels for 2 hr after 60 min of

treadmill exercise at 73.5% of maximal oxygen intake (this despite a substantial decrease in sensations of hunger, as noted above).

Dall et al. (2002) had eight healthy young males exercise at their lactate threshold for 45 min; this level of physical activity induced no change in total ghrelin levels.

Douglas et al. (2017) found that acylated ghrelin levels did not change from their control levels in either lean or obese individuals during the 8 hr following a substantial bout of exercise (60 min of treadmill running at 59% of maximal oxygen intake).

Hagobian et al. (2013), likewise, found no change of acylated ghrelin concentrations when lean and healthy young adults (11 men, 10 women) exercised at 70% of their peak oxygen intakes until they had accumulated 30% of their daily energy expenditure.

Holliday & Blannin (2017) noted that blood levels of acylated ghrelin were suppressed for as long as 2 hr post-exercise when 8 overweight individuals each performed a series of four 30-second all-out Wingate-type cycle ergometer sprints.

Jürimäe et al. (2007) carried out tests on 9 elite rowers, testing the effect of bouts of rowing just above and just below their anaerobic thresholds. No change of total ghrelin was seen at the immediate end of exercise, but 30 min later there was a non-significant trend to an increase of total ghrelin following the more intensive bout of exercise.

Kallio et al. (2001) were primarily interested in studying the effect of polymorphisms, but they saw no change in total ghrelin levels for 50 min following 30 min of cycle ergometry at intensities that

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increased progressively to 80% of the individual's maximal oxygen intake.

Kraemer et al. (2004a) had 6 well-trained young men perform a progressive treadmill test that required 10 min of running at 60% and 75% of maximal oxygen intakes, 5 min at 90%, and 2 min at 100% of maximal oxygen intakes. This protocol induced no change of total ghrelin concentration either immediately after or for 30 min following exercise.

Martins et al. (2007) exercised 12 lean young adults on a cycle ergometer for 60 min at a heart rate of about 130 beats per minute. No change of acylated ghrelin levels was seen for 60 min following this bout of physical activity.

Scheid et al. (2011) concluded that neither ghrelin nor leptin played an adaptive role in the response to a sustained exercise programme, provided that the individual's body weight remained unchanged. However, if food intake was constrained and the subject lost 3 kg or more of weight over a 3-month intervention they observed what seemed compensatory increases in levels of total ghrelin (acylated ghrelin unfortunately was not measured in this study).

Schmidt et al. (2004) had 8 healthy young men perform three bouts of cycle ergometry (20 min at 50 or 70% of maximal oxygen intake, and 10 min at 90% of maximal aerobic effort). None of these activities changed **total** ghrelin levels over the ensuing 80 min.

Shiia et al. (2011) measured both acylated and **total** ghrelin levels in 9 healthy young males during and for 30 min after a 60-minute bout of cycle ergometer exercise at 50% of the individual's maximal oxygen intake. They noted that acyl ghrelin levels were decreased by this activity, but that **total** ghrelin levels remained unchanged.

Zoladz et al. (2005) had 8 healthy non-smoking young men perform 2 progressive cycle ergometer tests (in both cases, a protocol requiring 30W incremental steps every 3 min to exhaustion). Some of the subjects were fed and others tested after an overnight fast but neither group showed any immediate change in **total** ghrelin levels.

Kraemer et al. (2004b) evaluated hormonal responses to resistance exercise in 9 well-trained young men. They observed no changes of **total** ghrelin levels for up to 15 min following eccentric exercise, but with concentric exercise there was a significant fall in ghrelin levels following the activity.

The study of Lopes et al. (2013) is relatively unique in that it examined long-term responses. Data were obtained on a small group of overweight women and men who had persisted in lifestyle programmes for 2-3 months, until their body mass had decreased by an average of 5%. Comparisons were drawn between the effects of dieting alone (a 2-4 MJ/day energy deficit) where acylated ghrelin levels were increased, and dieting plus exercise (40 min of cycle ergometry at 70% of maximal heart rate, 3 times/week), where acylated ghrelin levels were reduced by an average of 39%. The implication of this study seems that even in the long-term, exercise was helpful in countering the surge of appetite induced by serious dieting alone.

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Table 6: Influence of exercise upon hormones regulating appetite.		
Author	Subjects and exercise	Findings
Ghrelin		
Bilski et al. (2013)	12 healthy young men, moderate (30 min at 30% max. ox. intake) vs. high intensity effort (fire-fighter fitness test)	Decrease of total ghrelin 30 min following vigorous exercise
Broom et al. (2007)	9 lean young men, 60 min run at 72% of maximal oxygen intake	Acylated ghrelin levels reduced for 3 hr following exercise
Burns et al. (2007)	Young healthy adults (9 men, 9 women (60 min treadmill ex, at 73.5% max. ox. intake)	Total ghrelin levels unchanged for 2 hr post exercise
Dall et al. (2002)	Eight healthy males, 45 min exercise at lactate threshold	No change of total ghrelin levels post exercise.
Douglas et al. (2017)	47 lean and 25 obese adults, 60 min treadmill exercise at 59% maximal oxygen intake	No difference of acylated ghrelin levels for 8h post-exercise, in either lean or obese individuals
Foster-Schubert et al. (2005)	173 overweight post-menopausal women, aerobic exercise or stretching control for 1 yr.	Exercisers lost 1.4 kg & showed progressive increase of acylated ghrelin correlated with weight loss
Hagobian et al. (2013)	Lean and healthy young adults (11 men, 10 women) exercised at 70% of peak oxygen intake to 30% of daily energy expenditure	No significant effect on acylated ghrelin for up to 30 min post-exercise
Holliday & Blannin (2017)	8 overweight individuals performed 4 Wingate test 30 sec sprints	Reduction of acylated ghrelin for 2 hr,
Jürimäe et al. (2007)	9 elite male rowers, rowing just above and just below anaerobic threshold	Trend towards increase of total ghrelin 30 min after higher exercise intensity
Kallio et al. (2001)	18 young and healthy adults, 30 min cycle ergometer exercise peaking at 80% of max. ox. intake	No increase of total ghrelin over next 50 min
Kraemer et al. (2004a)	6 well-trained young men, treadmill exercise at 60, 75, 90 and 100% maximal oxygen intake (10, 10, 5, 2 min/bout)	No increase of total ghrelin over exercise and 30 min recovery
Kraemer et al. (2004b)	9 healthy young males performed bouts of concentric and eccentric resistance exercise	No change of total ghrelin (eccentric ex.), decrease 15 min post ex. (concentric ex.)
Lopes et al. (2013)	12 obese women, 6 obese men, diet plus exercise till 5% body mass lost (Over 2-3 months)	39% reduction of acylated ghrelin in exercise group over 74 days (in contrast with increase of ghrelin with diet alone)
Martins et al. (2007)	12 lean young adults, 60 min of cycle ergometry at heart rate of ~130/min	No change of acylated ghrelin for 60 min after exercise
Morpugo et al. (2003)	10 obese adults (3M, 7F), integrated (diet + ex.) 3-wk weight reduction programme.	5% weight loss did not normalize initially low total ghrelin levels
Scheid et al. (2011)	22 non-obese premenopausal women, 15 exercised for 90 min, 5/wk at 70-80% max. heart rate for 3 months, 10 with weight loss)	Total ghrelin increased by exercise only if weight lost
Schmidt et al. (2004)	8 healthy young males, cycle ergometry for 20 min at 50 or 70% max. ox. intake, or 10 min at 90%.	Total ghrelin unchanged for 80 min post-exercise.
Shiia et al. (2011).	9 healthy males exercised 60 min at 50% of maximal oxygen intake	Decrease of acyl ghrelin, but not total ghrelin during and 30 min post ex.
Zoladz et al. (2005)	8 healthy young non-smoking men, twp progressive cycle ergometer tests (30W/step), fed & fasting	No significant change of total ghrelin, fed or unfed

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Table 6: Influence of exercise upon hormones regulating appetite.		
Author	Subjects and exercise	Findings
Leptin		
Kraemer et al. (2002)	Review article	No change of plasma leptin levels with less than 60 min of exercise; may be reduced with bouts >60 min duration
Hulver & Houmard (2003)		Plasma leptin reduced by exercise >60 min, possibly also by very intense exercise
Jürimäe et al. (2007)	9 elite male rowers, rowing just above and just below anaerobic threshold	No change of leptin concentration with either intensity of exercise
Simsch et al. (2002)	6 health young rowers, 3 wk intensive resistance ex, vs. 3 wks endurance exercise	Resistance training decreased leptin levels
Zoladz et al. (2005)	8 healthy young non-smoking men, twp progressive cycle ergometer tests (30W/step), fed & fasting	No significant change of leptin with exercise, whether fed or unfed
Peptide YY & Pancreatic polypeptide		
Douglas et al. (2017)	47 lean and 25 obese adults, 60 min treadmill exercise at 59% maximal oxygen intake	Increase of polypeptide YY with exercise, greater in lean individuals
Hagobian et al. (2013)	Lean and healthy young adults (11 men, 10 women) exercised at 70% of peak oxygen intake to 30% of daily energy expenditure	No significant effect on polypeptide YY for up to 30 min post-exercise
Martins et al. (2007)	12 lean young adults, 60 min of cycle ergometry at heart rate of ~130/min	Significant increases of both polypeptides in 60 min after this exercise
Scheid et al. (2011)	22 non-obese premenopausal women, 15 exercised for 90 min, 5/wk at 70-80% max. heart rate for 3 months, 10 with weight loss)	No change of peptide YY in exercisers, even if weight lost
GLP-1		
Douglas et al. (2017)	47 lean and 25 obese adults, 60 min treadmill exercise at 59% maximal oxygen intake	Increase of GLP-1 with exercise, greater in obese individuals
Holliday & Blannin (2017)	8 overweight individuals performed 4 Wingate test 30 sec sprints	Increase of GLP-1 for 2 hr post exercise
Martins et al. (2007)	12 lean young adults, 60 min of cycle ergometry at heart rate of ~130/min	Significant increase of GLP-1 60 min after this exercise
Sympathetic nerve activity		
Schwartz et al. (1990)	31 overweight men, 4.8 MJ/d diet vs. jogging 70-85% max. HR 40 min 3-5 d/wk for 13 wk	Sympathetic nerve activity decreased more in diet group than in exercisers

Foster-Schubert et al. (2005) also looked at long-term responses, dividing a sample of 173 overweight post-menopausal women between aerobic exercise and a stretching control group. Over a period of 12 months, the exercise group lost an average of 1.4 kg in weight, and individual acylated ghrelin levels decreased in proportion to the decrease of body weight.

Morpugo et al. (2003) likewise looked at longer-term responses in 10 obese adults (3 men and 7 women). After a 3-week weight loss programme that included both various forms of aerobic exercise 5 days/week and dietary restriction (4.8-7.2 MJ/day), there was a 5% weight loss, but this did not appear to reverse initially low levels of **total** ghrelin. **Leptin.** Levels of leptin, the satiety-inducing hormone, are not generally

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affected by exercise bouts of 60 min or less (Hickey & Calsbeek, 2001; Hulver & Houmard, 2003; Kraemer, Chu, & Castracane, 2002), although they tend to be reduced by very prolonged bouts of physical activity, and possibly also by very intense exercise (Hulver & Houmard, 2003).

Jürimäe et al. (2007) compared the effect of two bouts of rowing, one just above and the other just below the anaerobic threshold in a group of 9 elite rowers. Neither intensity of exercise had any effect on their leptin levels.

A review article by Kraemer et al. (2002) concluded that there was no change of leptin levels, unless the duration of exercise was longer than 60 min; however, there might be some decrease of leptin concentrations with more prolonged exercise, particularly if it included resistance training. Further, no long-term changes were observed over as much as 12 weeks of aerobic training. A further review by Hulyer & Houmard (2003) equally concluded that leptin concentrations were decreased only if the duration of an exercise bout was > 60 min, or the exercise was very intense.

Simsch et al. (2002) compared the response of 6 healthy young rowers to 3 weeks of high intensity vs. 3 weeks of endurance rowing. The resistance activity induced a significant reduction of leptin levels, whereas the endurance rowing did not.

Zoladz et al. (2005) had 8 healthy non-smoking young men perform 2 progressive cycle ergometer tests (30W increments of loading every 3 min to exhaustion). There was no immediate change in plasma leptin levels, whether the subjects were fasting or had been fed.

Polypeptides. In most investigations, exercise has increased levels of peptide YY

and pancreatic polypeptide, hormones that suppress appetite.

Douglas et al. (2017) had 47 lean and 25 obese adults run on the treadmill for 60 min at 59% of their maximal oxygen intakes; an increase of polypeptide YY was seen following this exercise, the response being greater in the lean than in the obese individuals.

The subjects of Hagobian et al. (2013) were lean and healthy adults (11 men, 10 women). They were exercised at 70% of their peak oxygen intakes until they had accumulated 30% of their daily energy expenditure. This volume of physical activity had no effect upon plasma concentrations of polypeptide YY for up to 30 min post-exercise.

In contrast, Martins et al. (2007) saw a significant increase in the polypeptide concentrations of 12 lean young adults during the first 60 min after they had undertaken 60 min of cycle ergometry at a heart rate of ~130 beats/minute.

Contrary to their initial expectations, Schied et al. (2011) found no increase of polypeptide YY in lean individuals who had participated in a 3-month exercise programme, even in a sub-group where there was an average 3kg weight loss.

GLP-1. GLP-1 (the gut-produced incretin glucagon-like peptide) has shown exercise-induced increases in several studies (Douglas et al., 2017; Holliday and Blannin, 2017; Martins et al., 2007).

Sympathetic nerve activity. Schwartz et al. (1990) compared dietary and exercise-induced weight loss, finding that dieting led to a greater suppression of sympathetic nerve activity than involvement in an exercise programme.

Thus, most studies confirm that with exercise, the levels of the hormones regulating appetite are either unchanged

or modified in a manner that reduces hunger.

Conclusions

If a bout of exercise is taken immediately before a meal, it does not seem to stimulate appetite or immediate food intake. Rather, it may be helpful in limiting the amount of food that is ingested. The chronic effects of a long-term exercise programme upon appetite seem to be influenced by the extent of cumulative weight loss. Participation of a normal weight person in an exercise programme that causes a sustained negative energy balance and weight loss may increase fasting ghrelin levels (Ravussin, Tschöp, & Morales, 2001); this can cause an increase of background appetite in the period between individual bouts of exercise, reducing the potential loss of fat by as much as a third relative to the energy that has been expended. However, if a person still has excessive fat stores, such a response seems much less likely to occur, and many investigations have seen little change of either ghrelin or leptin levels in response to either moderate bouts of exercise or prolonged periods of physical conditioning.

Decrease of resting metabolism associated with an increase of habitual physical activity

A further potential challenge to the correction of obesity could come from a reduction in non-exercise energy expenditures, induced by a negative energy balance and/or a reduction of lean tissue mass. Given that these expenditures account for a large fraction of the total daily energy expenditures in sedentary individuals, if they were to decrease by even a small amount, this could have a

serious negative effect on programmes designed to correct obesity.

In considering this type of possible compensation, we will begin by looking at the components of daily energy expenditure other than deliberate bouts of programmed exercise; we will examine methods for estimation of their magnitude, and will assess their likely impact upon overall energy balance. We will then consider empirical data on exercise-induced changes in resting metabolic rate (Table 7) and the other components of non-exercise energy expenditure.

Components of daily energy expenditure other than programmed exercise

Much of the available literature divides the total daily human energy expenditure between deliberate, programmed bouts of exercise such as treadmill walking or cycle ergometry sessions, resting and basal metabolic components. However, in recent years, some investigators have made the alternative proposal of classifying an individual's overall daily energy expenditure into four components reflecting deliberate bouts of exercise, the thermic effects of food consumption, non-exercise activity thermogenesis (often indicated by the acronym NEAT)(Levine, 2002), and the basal metabolic rate.

The substantial energy costs of metabolizing foodstuffs are discussed in a subsequent section, below.

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Table 7: Impact of weight loss programmes with or without an exercise component upon resting metabolic rate (RMR).

Authors	Subjects	Intervention	weight and fat loss	RMR (absolute & relative, kJ/kg FFM per day)
Short-term effects (<12 weeks)				
Ballor et al. (1996a, b)	20 subjects aged 56-70 yr, 18 exercised after dieting	12 weeks, diet 3.6-4.4 kJ/day, then 12 wks aerobic or resistance training	Weight loss 9.0 kg (2.6 kg lean, 6.4 kg fat) No further changes of body mass with exercise	15% decrease of absolute RMR, 8% decrease of relative RMR, no further change of RMR with exercise
Bingham et al. (1989)	Young normal weight adults (3 men, 3 women)	9 weeks of progressive aerobic exercise, increasing to running 1 hr/day 5 days/wk	30% increase of peak oxygen intake	No change of <i>basal</i> metabolic rate
Broeder et al. (1992)	47 lean young men	12 wk high intensity resistance or endurance exercise	Both groups reduced body fat (-2.5%, - 1.5%), increased lean tissue mass (2.1, 0.3 kg)	No significant change of RMR
Bryner et al. (1999)	17 women, 3 men	12 wk diet (2 MJ/d), plus aerobic ex. 1h, 4 times/wk or resistance ex. 3 times/wk	Both groups had similar increase of max. aerobic power (~30%), 4.1 kg loss of lean tissue with aerobics, almost no change with resistance ex.	RMR decreased 13.4% in aerobic group, but increased in resistance group by 4%
Byrne et al. (2012)	16 obese men and women	Diet (2.4-2.7 kJ/day) plus 4 aerobic & 2 resistance ex. sessions/wk for 12 wks	Decrease of RMR correlated with less than expected weight loss	RMR decreased 5% in weeks 4-12
Cullinen and Caldwell (1998)	20 normal weight women	45 min resistance ex. twice/wk for 12 wks	Fat loss -2.6% Lean mass +2.0 kg in exercise group	Non significant 8.6% increase of RMR
Doi et al. (2001)	17 normal weight men	12 wk diet (15% reduction) plus 25 min light resistance ex. 7d/wk, half received protein supplement	14.2% loss of fat mass, 2.9% loss of lean mass	8.1% increase of RMR with supplement, no change of RMR in remainder of subjects
Donnelly et al. (1994)	115 obese females	6 combinations of dietary restriction, endurance and resistance exercise continued for 12 wk	No significant inter-group differences in weight loss	Endurance exercise after 4 weeks dieting gave least decrease of RMR (-6%)
Geliebter et al. (1997)	245 men, 40 women, aged 19-48 yr, moderately obese (>20% overweight)	Diet vs. diet + aerobic exercise vs. diet + strength training	All 3 groups lost 9 kg body mass over 8 wks. Strength training lost less fat-free mass.	No inter-group differences of decrease in RMR (decrease ~500 kJ/day)
Gornall & Villiani (1996)	22 overweight female subjects	Diet (3.4 MJ/d) or diet + strength training (3/week) for 4 weeks	Loss of 4.1 kg body mass, 1.4 kg of lean mass,	12.1%, 5.5% decrease of absolute RMR in 2 groups

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Table 7: Impact of weight loss programmes with or without an exercise component upon resting metabolic rate (RMR).				
Authors	Subjects	Intervention	weight and fat loss	RMR (absolute & relative, kJ/kg FFM per day)
Short-term effects (<12 weeks)				
Kempen et al. (1995)	20 obese women	8 wk diet 2 MJ/d, then 3.5 MJ/d or diet + 90 min aerobic ex. 3 d/wk, at 50-60% of max. ox. intake	wt. loss 7.1, 9.1 kg, fat loss 5.5, 7.8 kg, lean tissue loss 1.6, 1.3 kg	<i>sleeping</i> metabolic rate -0.7 MJ/d (10%) in both groups
Kramer et al. (1997)	31 overweight women (BMI > 27 kg/m ²)	12 wks diet, diet + aerobics, diet + aerobics + resistance, or control	Weight loss 6.2, 6.8, 7.0 kg Fat loss 5.8%, 8.0%, 4.3%	No significant change of RMR in any of 3 intervention groups
Lennon et al. (1984)	38 men, 40 women, 15-35% overweight	8 wks daily self-prescribed vs. tri-weekly prescribed exercise	Increase of Bruce predictions of max. ox. intake 9% and 12%	RMR increased 4% and 10%
Lopes et al. (2013)	12 obese women, 6 obese men	Diet, or diet plus exercise till 5% body mass lost (Over 2-3 months)	5% weight loss in both groups, 10.7% increase of max. ox. intake in exercise group	16.3% increase of RMR in exercise group, no signif. change with diet alone
Meredith et al. (1989)	10 young and 10 elderly normal weight adults	8 wks thrice weekly exercise rising to 85% of max. ox. intake, 300 kCal/session	No change of body fat, 10% increase of max. ox. intake	No change of <i>basal</i> metabolic rate
Molé et al. 1989 (1989)	5 obese adults (4 women)	2 weeks 2 MJ/d diet for 4 wks, 30 min/d at 60% max. ox. intake final 2 wk	Loss of 4.1 kg first 2 weeks, further 2.5 kg second two weeks (but 1.1 kg gain of lean tissue)	Initial 13% decrease of RMR reversed with exercise
Phinney et al. (1988)	12 overweight women	3 MJ/d diet with 1.5g/kg protein for 4 wks, then half of group undertook 27 hr of exercise at 50% max. ox. intake for 4 wk	Nitrogen balance achieved throughout	Both groups showed decrease of RMR
Poehlman & Danforth (1991)	Older adults (13 men, 6 women)	8 weeks of exercise, 3 times/wk at 60% rising to 85 % of max. ox. intake	15% increase of max. ox. intake, no signif. change of lean or fat weight	RMR increased 10% after training
Racette et al. (1995)	23 obese women	12 wk 4.9 MJ/d diet or diet + 45 min aerobic ex. 3 times/wk, 60-65% max. ox. intake		RMR decrease 0.54 MJ/d in both groups
Schlundt et al. (1993)	49 men and women	Low fat or low calorie diet for 10-20 wk, both supplemented by ex. >5d/wk	loss of body wt -5.3, -9.0%; 2.5% loss of lean mass	5.5% decrease of RMR
Schwartz et al. (Schwartz et al., 1990)	31 overweight men	4.8 MJ/d diet vs. jogging 70-85% max. HR 40 min 3-5 d/wk for 13 wk	fat mass -29.9 % -vs. -9.5% lean mass -5.8% vs. no change	RMR - 8.1% vs. + 6.7%

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Table 7: Impact of weight loss programmes with or without an exercise component upon resting metabolic rate (RMR).				
Authors	Subjects	Intervention	weight and fat loss	RMR (absolute & relative, kJ/kg FFM per day)
Short-term effects (<12 weeks)				
Svendsen et al. (1993)	121 post-menopausal women	Control or 4.2 MJ/d diet for 12 wks or diet + aerobic & resistance ex. 60-90 min 3 d/wk	wt loss diet 24.3%, diet + ex. 30.1%	Diet- no change of RMR, diet + ex. RMR +11.5%
Tagliaferro et al. (1986)	10 healthy young women	10 wk graded jogging exercise (to 5 MJ/wk)	7.0% increase of max. ox. intake, 10.4% fat loss	No signif. change of RMR
Thompson et al. (1996)	Meta-analysis of 22 studies, 68 M, 563 F aged 31-45 yr	Diets (usually less than 4.8 MJ/day) vs. diet + exercise		Decrease of absolute RMR 10.6%, 9.1%, decrease of relative RMR 6.8%, 5.9%
Tremblay et al. (1986)	8 moderately obese women	5 hr exercise per wk at 50% max. ox. intake for 11 wks		Increase of RMR 8% per unit of fat free mass
Tremblay et al. (1990)	5 overweight young men	100 days of cycle ergometry, 4.2 MJ/d		RMR and thermic effect of food unchanged
Van Aggel-Leijssen et al. (2001)	21 obese women	12 wks cycle ergometry at 40% max. ox. intake, 3 d/wk, 1 MJ/d	No signif. change of fat mass or fat-free mass	No signif. change of RMR
Van Dale et al; (1989)	12 obese women	2.9-3.5 MJ /day diet vs. diet plus 4h/wk moderate aerobic exercise	Similar loss of body mass and lean mass in 2 groups	Decrease of sleeping metabolism 20-27% vs.12-18% (ns)
Warwick & Garrow (1981)	3 obese women	3.3 MJ/d diet for 12-13 wk, alternating 3-4 wks of exercise (2 hr/d cycle ergometry, 6.8 MJ)		Basal metabolic rate not different during exercise periods
Wilmore et al. (1998)	40 men, 37 women, overweight	20 wks endurance training, 30 min/d 50% rising to 75% maximal oxygen intake	1% decrease of body fat	No change of RMR 24 and 72 hr after intervention
Long-term effects (40-52 weeks)				
Frey-Hewitt et al. (1990)	121 overweight men aged 30-59 years	1 year of diet vs. exercise (jog 10 miles/wk) vs. controls		Diet group had significant drop in absolute and relative RMR, no significant change in exercise group
Van Dale et al. (1990)	32 F, 12 M	Diet or diet + exercise for 18-40 months		Relative sleeping metabolic rate -15.8% (diet) vs. -3.2% (diet + exercise)
Wadden et al. (1990)	16 grossly obese women	Diet for 48 weeks, supplemented by walking	8.6% decrease of fat-free mass	Decrease of relative RMR 9.4%, 8.3% with 2 dietary choices

Basal and resting metabolic rates. The basal metabolic rate is the energy expenditure observed in the early morning, when a person is lying completely at rest, perhaps 12 hr after the most recent meal, and following a good night's sleep.

In a sedentary and/or inactive individual, basal metabolism accounts for as much as 60% of the total daily energy expenditure. As much as three quarters of the variability in this component of metabolism is attributable to inter-individual differences in lean tissue mass (Deriaz, Fournier, & Tremblay, 1992). Thus, changes of BMR can arise if prolonged dieting or a rigorous exercise programme leads to a decrease in lean tissue mass.

The resting metabolic rate is also usually determined early in the morning, with the subject well-rested and sitting, lying or semi-recumbent for 30 -45 min, breathing into a hood or spirometer. It is about 10% greater than the BMR.

Non-exercise activity thermogenesis (NEAT). The definition of non-exercise activity thermogenesis is broad and somewhat controversial. For Levine (2002), it embraces "*energy expended for everything we do that is not sleeping, eating or sports-like exercise...from walking to work, typing, performing yard work, undertaking agricultural tasks and fidgeting*". With such a broad definition, it is potentially the most variable component of total of daily energy expenditures (Chung et al., 2018). Some investigators have argued that (perhaps through a hypothalamic mechanism), NEAT shows a compensatory increase with over-feeding and a corresponding decrease with under-feeding. However, a recent meta-analysis of 10 randomized controlled trials

(Fedewa et al., 2017) demonstrated that in most studies the overall NEAT has not changed significantly in response to exercise training. A recent systematic review of NEAT drew similar conclusions (Silva et al., 2018).

Specific changes in the energy expended upon fidgeting, incidental movements and voluntary leisure activities associated with participation in exercise programmes are discussed further below.

Methods of determining the four components of daily energy expenditure

The energy costs of deliberate exercise, resting and basal metabolism can be determined by the standard methods of indirect calorimetry, and the thermic effect of foods can be obtained from tables. However, the estimation of NEAT is more challenging and the findings lack precision.

One option is to determine the total daily energy consumption by a method such as confining the subject within a whole body calorimeter or administering a dose of doubly labeled water. The NEAT component is then derived by the subtraction of appropriate figures for basal metabolism, the thermal cost of feeding and expenditures in any deliberate bouts of exercise.

An alternative approach is to log each of the non-exercise activities that a person performs during a typical day, along with their respective durations, and then to multiply each of these individual components by their anticipated metabolic costs. This method has the advantage that if exercise induces any change in total NEAT, it may be possible to see which of its components have changed,

There are many possible sources of error and misinterpretation in such calculations. Some authors have expressed metabolic rates in relative units (per unit of fat-free mass or total body mass), leading to changes in these indices if there has been a decrease in body mass as a result of the intervention. With repeated measurements, subjects may also become familiarized with the techniques of metabolic data collection, and as they become more relaxed, energy expenditures may decrease. In women, findings for the ovulatory phase may also differ from those for other phases of the menstrual cycle, with a significant impact upon energy expenditures. Finally, the most recent exercise bout here may have short- or medium-term residual effects upon energy expenditures, with some reports suggesting an increase of basal metabolic rate for as long as 24 hr post-exercise (Passmore, Johnson, & Shirling, 1960).

Potential impact of changes in RMR and NEAT upon overall energy balance

Changes in RMR and/or NEAT energy expenditures have a large potential impact upon the effectiveness of an exercise programme as a means of reducing body fat content. Let us suppose that as a planned weight-reduction tactic a person decides to undertake 30 min of brisk walking 3 times per week. The *net* energy cost of this activity is 100 kCal (0.42 MJ) per session, or a total of 300 kCal (1.25 MJ) per week. The RMR amounts to around 1.5 kCal/min, or a cumulative expenditure of 15,120 kCal (62.9 MJ) per week, and a decrease in RMR of only 2% would completely negate the benefits of the mild walking programme.

Empirical data

There seems general agreement that a restriction of food intake usually leads to some immediate decrease of RMR. However, such a response is generally less marked in obese than in lean subjects, and it is also less obvious if the treatment of obesity is based upon exercise rather than a programme of restriction. Moreover, RMR seems to be better sustained if the period of exercise training is prolonged rather than being limited to a few weeks (Wilmore, Stanforth, and Hudspeth, 1998), possibly because prolonged training often increases lean tissue mass.

Wilmore and associates (1998) saw no decrease in the RMR of 74 subjects 24 or 72 hr following a 20-week programme of endurance training that had decreased body fat content by 1%. Further, they cited several cross-sectional and longitudinal articles suggesting that exercise training without dietary restriction could increase rather than decrease the RMR.

In looking at the details of individual empirical studies, we will distinguish the effects of short-term (<12 weeks) and longer-term (>12 week) exercise interventions upon the resting metabolic rate.

RMR and short-term exercise programmes. One early review listed several small-scale studies where resting metabolic rate had been monitored over the course of short-term interventions (Molé, 1990). Of 6 reports not included in Table 7, three showed exercise as increasing RMR relative to dieting alone, and 3 did not.

Ballor et al. (1996a) had 20 older subjects (age 56-70 years) follow a 3.6-4.4 MJ/day diet for 12 weeks, finding at the end of this time a 15% decrease of their absolute RMR, and an 8% decrease of the RMR relative to lean tissue mass. 18 of the

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original subjects then participated in a further 12 weeks of aerobic or resistance training (probably with some continuing self-imposed dieting). There were no further changes in either the absolute or the relative RMR as compared with the findings seen at the end of the initial 12 weeks of dieting. The authors had hoped that the period of exercise would restore the initial RMR, removing this handicap to weight maintenance, but in general this did not occur with either type of exercise, despite some trend to an increase of lean tissue mass in those performing resistance training.

Bingham et al. (1989) recruited six normal weight young volunteers to a 9-week programme where a constant diet was maintained over 9 weeks of progressive aerobic exercise that peaked with subjects running 1 hour per day, 5 days per week, and developing overall a 30% increase in their maximal oxygen intake. Basal metabolic rate remained unchanged over the course of this study.

Broeder et al. (1992) compared the effects of 12 weeks of endurance training with a similar period of high intensity resistance training in a sample of 47 lean young men. Both groups reduced their body fat content (by - 1.5% and -2.5%, respectively), and they increased their lean tissue masses by 0.3 kg and 2.1kg, but neither group showed any significant change of RMR.

Bryner et al. (1999) had 17 obese women and 3 obese men undertake a 12-week programme of rigid dieting (2 MJ/day), combined with either 4 sessions of aerobic exercise or 3 sessions of resistance training per week. Both subject groups showed a similar increase of aerobic power. Those performing the aerobic exercise sustained a 4 kg loss of lean tissue, but in the resistance exercise

group, lean mass remained unchanged. The aerobic group also showed a 13.4% decrease of RMR, but probably because of the conservation of lean tissue, in the resistance group the RMR actually increased by about 4.0%.

Byrne et al. (2012) examined 16 obese men and women who had followed a very low energy diet (2.4-2.7 kJ/day) and who had also undertaken aerobics (4 times/wk) and resistance exercise (2 times/week) for 12 weeks. In response to this stringent regimen, the body mass had decreased by 18.6 kg (15.5 kg of this loss being fat, and 3.1 kg lean tissue). The measured weight loss in this study was only 67% of that predicted from the combination of dietary restriction and the energy expended in exercising. Resting metabolic rate (initially 7.5 kJ/day) decreased to 7.1 kJ/day by week 4 (a drop of 5%), and it remained at this lower level through week 12 of the study. Moreover, individual differences between the actual and the predicted decrease in body mass were significantly correlated with individual decreases in RMR ($r = 0.51$, $p < 0.01$). In all, the decrease in RMR accounted for about two thirds of the difference between observed and expected weight losses.

Cullinen & Caldwell (1998) enrolled 20 normal weight and previously untrained women in resistance exercise classes (45 min, twice per week for 12 weeks). This programme induced a fat loss of 2.6% and a 2.0 kg increase of lean tissue mass, with a non-significant trend to an 8.6% increase of RMR.

Doi et al. (2001) placed 17 slightly overweight men on a moderate diet (15% reduction of energy intake) for 12 weeks, supplementing this by 25 min of light resistance exercise 7 days per week. A half of the group also received a daily 10 g

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protein supplement. Those with the additional protein intake increased their RMR by 8.1%, but the other subjects showed no change of RMR.

Donnelly et al. (1994) compared 6 possible 12-week treatments in a sample of 115 obese women, dietary restriction being linked with endurance or resistance exercise either during or subsequent to a restriction of food intake. There were no significant differences of weight loss between the various subject-groups. However, the most favourable response was seen with endurance exercise that was initiated after 4 weeks of dietary restriction. This regimen increased maximal aerobic power by 6.2% and a strength index by 12.5%, also showing the smallest decrease of resting RMR (-6%).

Geliebter et al. (1997) compared the effects of dieting alone (a food intake sufficient to meet 70% of resting metabolic rate, i.e. 5.15 MJ/day) with dieting plus aerobic exercise or strength training performed 3 times per week in a sample of clients who were initially 20% overweight. All 3 subject-groups lost an average 9 kg of body mass over an 8-week study. The strength training group showing a lesser loss of lean mass than the other 2 groups, but there were no inter-group differences in the decrease of RMR, which amounted to around 500 kJ/day.

Gornall & Villani (1996) exposed 22 overweight women to 4 weeks of severe dieting (an energy intake of ~ 3.4 MJ per day). A half of their subjects also undertook strength training 3 times per week. The RMR was reduced significantly in both groups, although the decrease tended to be smaller in those receiving the strength training.

Kempen et al. (1995) had 20 obese women undertake 8 weeks of dieting (4 weeks at an energy intake of 2 MJ per day,

followed by 4 weeks at 3.5 MJ per day). A half of the group supplemented their dieting by performing 90 min of aerobic exercise at 50-60% of their maximal oxygen intakes 3 days per week. The cumulative weight loss (7.1 vs. 9.1 kg) and fat loss (5.5 vs. 7.8 kg) were somewhat greater for those adding exercise to their dietary programme, and losses of lean tissue (1.6 vs. 1.3 kg) tended to be slightly smaller for the exercisers, but both groups showed a 10% decrease of *sleeping* metabolic rate.

Kraemer et al. (1997) divided a sample of 31 overweight women between 4 groups. Over a 12-week intervention, one group served as controls. The remaining 3 groups met for nutritional counseling once per week, while embarking on a 5 MJ/day high fibre, high carbohydrate diet. One group supplemented the dietary intervention with bouts of aerobic exercise, performed 3 times per week at 70-80% of peak heart rate, sessions continuing for 30 rising to 50 min. Another group supplemented the dieting and the aerobics programme with a routine of 11 resistance exercises, performed 3 times per week. Responses were fairly similar with the 3 forms of intervention, with none showing any significant reduction in their RMR.

Lennon et al. (1984) compared the effects of self-selected daily exercise with tri-weekly prescribed exercise in a sample of 38 men and 40 women who were initially 15-35% overweight. Bruce test estimates of maximal aerobic power increased by 9 and 12% respectively, with 4% and 10% increases in RMR over the course of the intervention.

Lopes et al. (2013) took a small sample of overweight women and men who persisted with their assigned programme for 2-3 months, until body mass had

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decreased by 5%. They compared the effects of dieting alone (a 2-4 MJ/day energy deficit), and dieting plus exercise (40 min of cycle ergometry at 70% of maximal heart rate, 3 times/week). The combined therapy yielded a 10.7% increase of maximal oxygen intake, and a 10.3% increase of RMR, with only a 1.0% decrease in lean tissue mass; in contrast, with dieting alone the lean tissue mass fell by 3.4%, and no increase of RMR was seen. Like Marzullo et al. (2004), Lopes et al. found an association between the increase of RMR and ghrelin concentrations.

Meredith et al. (1989) examined responses in 10 young and 10 elderly people of normal weight who undertook 8 weeks of thrice-weekly training at an intensity rising to 85% of peak oxygen intake, with a cumulative energy expenditure of 300 kcal per session. This volume of exercise did not change either body fat content or the *basal* metabolic rate, although maximal oxygen intake was increased by about 10%.

Molé et al. 1989 (1989) exposed 5 obese adults (4 of whom were women) to a low energy diet (2 MJ/day) for 4 weeks. At the end of the first 2 weeks, body mass had decreased by 4.1 kg, and there was a 13% average decrease in RMR. Over the following 2 weeks, daily exercise was added (30 min at 60% of maximal aerobic power). The total body mass decreased by a further 2.5 kg, but lean mass increased, and the RMR was fully restored to its initial level.

The study of Phinney et al. (1988) is unique in that the nitrogen balance of 12 obese women was maintained during the imposition of a rigorous 3 MJ/day diet by administering a protein supplement (1.5 g/protein per kg of ideal weight). After 4 weeks of dieting alone, a half of the group supplemented the dieting during the next

4 weeks by exercising for a total of 27 hr at 50% of maximal aerobic power. However, both 2 subsets of subjects showed a substantial decrease of RMR, to -10% in the diet group, and -17% in the exercise plus diet group.

Poehlman & Danforth (1991) tested older adults (13 men, 6 women, of normal weight, and an average age of 64 years). Subjects exercised for 8 weeks, 3 times/week, at an intensity rising from 60% to 85% of their maximal oxygen intakes. Following the intervention, the body fat content was reduced by an average of 0.5%, but the RMR was increased by an average of 10%.

Racette et al. (1995) compared the effects of 12 weeks of dieting (4.9 MJ/d) with dieting supplemented by 45 min of aerobic exercise, performed thrice weekly at 60-65% of maximal oxygen intake. Both groups showed similar (0.54 MJ/day) decreases in RMR.

Schlundt et al. (1993) studied 49 men and women who were involved in low fat or low calorie dieting programmes for 10-20 weeks. Fat loss was greater for the low calorie group (9.0%) than for the low fat diet (5.3%). Both groups showed small decreases in lean tissue (2.5%), with an associated 5.5% decrease of RMR.

Schwartz et al. (1990) compared the effects of 13 weeks of dieting (an energy intake of 4.8 MJ/day) with jogging at 70-85% of maximal heart rate for 40 min 3-5 times per week. The respective decreases in fat mass were -29.9% vs. -9.5%, and lean mass decreased by -5.8% vs. no change, while there was a sharp contrast in the effect of the two options upon RMR (-8.4% vs. +6.7%).

Svendsen et al. (1993) divided a sample of 121 post-menopausal women between control, diet (4.2 MJ/day) and diet plus exercise (60-90 min of resistance and

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aerobic exercise 3 times per week) groups. Over 12 weeks, there was a substantial loss of body mass with both interventions (for the dieters, 24.3%, for the diet + exercise group 30.1%). The diet group showed no change of RMR over the 12 weeks, but in the diet + exercise group there was an 11.5% increase of RMR.

Tagliaferro et al. (1986) had 10 healthy young women engage in 10 weeks of graded jogging exercises, to an energy expenditure of 5 MJ/week. This induced a 7.0% increase of maximal oxygen intake and a 10.4% decrease in body fat content, but there was no significant change of RMR.

Thomson et al. (1996) completed a meta-analysis on data for 68 men and 563 women aged 31-45 years. Most of the subjects had been placed on a diet of less than 4.8 MJ/day, and commonly studies had compared dieting alone versus dieting plus aerobic exercise. Both approaches had led to reductions in both absolute and relative RMR, but there was again a trend to a slightly smaller decrease of RMR in those who received a combination of exercise plus dieting. Nevertheless, the authors found difficulty in making generalizations, given differences in the length of study, the type, duration, frequency and intensity of exercise that had been undertaken, the overall degree of energy deficit imposed, and the relative proportions of carbohydrates, proteins and fats ingested. Moreover, few studies were of sufficient length to see whether any early reductions in RMR waned if the dieting and exercise programme continued for a longer period.

Tremblay et al. (1986) had 8 moderately obese women train for 11 weeks at 50% of their maximal aerobic power (30 minute sessions, 5 times per week). At the end of this period, the RMR

per unit of fat-free mass was increased by 8%. Further, a cross-sectional comparison between 20 trained and 39 untrained individuals showed that the RMR was 11% higher in those who were trained. In a further study, 5 overweight young men who were receiving a constant dietary intake performed daily cycle ergometry for 100 days, thus creating a 4.2 MJ/d energy deficit (1990). Neither the RMR nor the thermic effect of food was modified by this regimen. Van Aggel-Leissen et al. (2001) adopted a very light exercise programme (12 weeks of cycle ergometry at 40% of the individual's maximal oxygen intake, performed 3 days per week, with an average 1 MJ/day increase of energy expenditure). The 21 obese female subjects recruited to this programme showed no change of either body fat or fat free mass, and no significant change in their RMR.

van Dale et al. (1989) compared responses to a 2.9-3.5 MJ/day diet with the effects of the same diet when it was supplemented by 4 hr per week of moderate aerobic exercise (four 60 minute sessions/week at 50-60% of maximal oxygen intake). Over a 12-week trial, losses of body mass and lean mass in two groups of 6 obese women were similar, but the *sleeping* metabolic rate was reduced by 20-27% in those who undertook dieting alone, compared with a trend to a lesser 12-18% reduction in the combined dietary/exercise programme.

Warwick & Garrow (1981) placed 3 obese women on a 3.3 MJ/day diet for 12-13 weeks, with interspersed 3-4 week intervals of cycle ergometry (2 hr/day to an energy expenditure of 6.8 MJ). The BMR was not significantly changed by the introduction of the exercise sessions.

Wilmore et al. (1998) had 40 men and 37 women who were overweight undergo

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20 weeks of daily aerobic training (30 min sessions at an intensity increasing from 50 to 75% of maximal oxygen intake). Duplicate pre-test measurements agreed well with each other, and 24 and 72 hr after the intervention, the RMR was unchanged either as a gross figure or as a rate per unit of body mass.

RMR and longer-term exercise interventions. A number of cross-sectional studies have shown a higher RMR in active individuals, but inferences about long-term effects drawn from such observations must be limited, because of the many differences of lifestyle between sedentary and active individuals. Relatively few longitudinal investigations have looked at the long-term changes in RMR associated with involvement in a sustained exercise programme.

Frey-Hewitt et al. (1990) followed 121 overweight men aged 30-59 years for one year. Subjects were divided between diet, exercise (jogging 10 miles/week) and control groups. At the end of the year, the dieting group still showed a significant decrease in absolute and relative RMR, but in contrast the relative RMR of the exercised group showed no significant difference between initial and final values.

Van Dale et al. (1990) reported data for 32 females and 12 males after 18-40 months of dieting or dieting plus exercise. In those who added exercise to their dietary regimen, the relative sleeping metabolic rate had returned close to its initial values by the end of the study, whereas in those who were treated by dieting alone it remained decreased by a substantial 16%.

Wadden et al. (1990) observed 18 grossly obese women (average body weight 108 kg) for 48 weeks. A half of the group accepted a moderate limitation of

energy intake of 5.02 MJ/day throughout the study, while the other half adopted a very strict 1.76 MJ/day dietary regimen for the first 16 weeks. All subjects increased their physical activity, primarily by walking. Over the first 5 weeks, the reduction in RMR was more than double that expected simply from the reduction in body mass. However, by 48 weeks the RMR of the 2 sub-groups was reduced by only 9.4% and 8.3% respectively, whereas the body mass of the 2 groups had decreased by 16.6% and 19.5%, and fat-free mass had also decreased by 8.6% and 4.1%.

Conclusions. Summarizing the data presented in Table 7, there seems to be a short-term decrease in RMR of as much as 20-30% in response to a severe diet-induced negative energy balance, with a partial to complete reversal of this trend by the incorporation of exercise training into a weight-reduction programme.

The decrease of RMR seen with dieting alone is fairly consistent. There was a decrease of RMR in 13 trials in Table 7, and no change in 3 studies; no investigations saw an increase of RMR. In some reports, the decrease of RMR resulting from the dieting was as large as 10-15%.

When weight loss was brought about by exercise alone, or by a combination of dieting and exercise, the picture was rather different; 9 of 30 comparisons showed an increase of RMR of up to 10% (Cullinen & Caldwell, 1998; Lennon et al., 1984; Poehlman & Danforth, 1991; Tremblay et al., 1986), in a further 9 of 30 studies there was no change of BMR (Bingham et al., 1989; Broeder et al., 1992; Meredith et al., 1989; Tagliaferro et al., 1986; Tremblay et al., 1990; Van Aggel-Leijssen et al., 2001), and RMR was

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decreased in only 12 of 30 studies. Moreover, any decrease of RMR was typically smaller than with dieting alone (see, for example, (Ballor et al., 1996a, b; Donnelly et al., 1994; Frey-Hewitt, Vraizan, & Dreon, 1990; Gornall & Villani, 1996; Lopes et al., 2013; Schwartz et al., 1990; Svendsen, Hassager, & Christiansen, 1993; van Dale, Schoffelen, & ten Hoor, 1989). Comparisons of aerobic vs. resistance exercise found either no difference between the 2 modes of activity (Broeder et al., 1992; Kraemer et al., 1997) or a greater RMR with a resistance regimen (Bryner et al., 1999). Moreover, in at least one trial the maintenance of RMR was helped by the administration of a protein supplement (Doi et al., 2001). In 14 of 15 aerobic interventions, initially obese subjects showed little or no loss of fat-free mass during treatment (Thomas et al., 2012), probably because the energy demands of their programmes were met from the depletion of body fat stores.

There have been several trials where dieting was combined with exercise. In some, RMR then remained either unchanged (Doi et al., 2001; Frey-Hewitt et al., 1990; Kraemer et al., 1997; Molé et al., 1989; Tremblay et al., 1990; van Dale, Schoffelen, & ten Hoor, 1989; Warwick & Garrow, 1981) or decreased (Byrne et al., 2012; Donnelly et al., 1994; Geliebter et al., 1997; Gornall & Villani, 1996; Kempen et al., 1995; Phinney et al., 1988; Racette et al., 1995; Schlundt et al., 1993; Thomson et al., 1996; Van Dale et al., 1990; Wadden et al., 1990), and only very few authors found an increase of RMR (Lopes et al., 2013; Schwartz et al., 1990; Svendsen et al., 1993), in one series with resistance but not with aerobic exercise (Bryner et al., 1999), and in another only if a protein supplement was provided (Doi et al., 2001).

The difference in response between exercise alone and dieting plus exercise is striking, and it suggests that the combined treatment has often created too large a negative energy balance, perhaps with a substantial metabolism of lean tissue. The extent of lean tissue loss probably depends on the client's initial reserves of body fat relative to the imposed energy deficit, and the amount and quality of the amino acids that are made available during the period of weight loss. The substantial increase of RMR in subjects receiving a protein supplement, seen by Doi et al. (2001) but not by Phinney et al. (1988) offers some support to this idea. Unfortunately, few investigators have as yet examined how far the retention of lean tissue can be facilitated by providing essential amino acids in adequate amounts and at an appropriate time of day relative to exercise sessions (Shephard, 2012).

Possible mechanisms underlying maintenance of resting metabolic rate and/or NEAT when dieting is supplemented or replaced by exercise

A number of mechanisms have been suggested whereby regular vigorous exercise might help to sustain metabolism during a period of fat loss (Table 8). We will look briefly at each of these possibilities.

Table 8: Possible mechanisms whereby regular exercise might sustain or even augment resting and/or NEAT energy expenditures during a period of negative energy balance.

- Sustained post-exercise increase of oxygen consumption
- Increase of lean tissue mass
- Increase of fidgeting and incidental movements
- Shortened duration of sleep
- Increased energy expenditures on tissue growth and repair
- Hormonal adaptations
- Thermic effect of foods; change of metabolites
- Alteration of proton leakage

Sustained post-exercise increase of oxygen consumption

The repayment of any exercise-induced oxygen debt is usually completed within 60 min or less, but some authors have suggested that a bout of exercise can cause a more long-lasting stimulation of metabolism. Thus, Margaria et al. (1933) and Newsholme (1978) have both suggested that there is also an "ultra-slow" recovery process that keeps the RMR 10-20% above its initial level for 2 hr or longer. Such a process could give a short-term boost to the metabolism of a person who was seeking to reduce obesity by participation in an exercise programme. Further, some investigators have proposed an even longer-lasting post-exercise stimulation of metabolism. Such a concept seems quite plausible if the exercise is sufficiently severe to cause a need for the re-synthesis of energy stores, the repair of injured or metabolized tissues and muscle hypertrophy.

Atwater and Benedict first mooted this idea in 1903, and 2 years later Benedict and Carpenter demonstrated a 12.5% average increase of sleeping metabolic rate in 5 subjects during the 6 hr that followed a bout of exercise, with the extent of this response apparently determined by the intensity of effort. Edwards et al. (1935), likewise, reported that Harvard football players still showed a 23% increase of metabolism 13 hr after a game, and lacrosse players still had an 8% effect 31 hr after a contest. Passmore, Johnson, & Shirling (1960) again found a 16% increase of oxygen consumption 7 hr after completing a brisk 16 km walk.

Some more recent investigators have duplicated these findings (Table 9), but others have not, perhaps because their subjects undertook a less vigorous bout of exercise. Børsheim & Bahr (2003) made

an extensive review of the question, concluding that an exercise bout lasting at least 30-60 min with a minimum intensity of 50-60% of maximal oxygen intake was needed to generate any substantial post-exercise increase of oxygen consumption,

Parallel effects to those accompanying vigorous aerobic exercise can develop following a bout of heavy resistance exercise (Burleson et al., 1998; Elliot, Goldberg, & Kuehl, 1992). Causal factors probably include a shift from carbohydrate to fat metabolism as glycogen reserves are depleted, and the regeneration or synthesis of muscle protein in response to injuries and/or a training response.

Doses of exercise sufficient to cause a sustained post-exercise increase of oxygen consumption could be reached in some weight-reduction programmes, and could thus contribute to maintenance of effective levels of daily energy expenditure. In some instances, the cumulative post-exercise energy expenditures have been as large as 15% of the expenditures developed during a bout of exercise. However, many obese individuals would probably find that the necessary doses of exercise were excessive for them, and in consequence early optimism regarding the possible contribution of post-exercise oxygen consumption to weight-loss programmes has diminished (LaForgia, Withers, & Gore, 2006).

Exercise-induced increases of lean tissue mass

The fat-free mass is generally considered one of the main determinants of RMR (Sparti, DeLany, & de la Bretonne, 1997), and the loss of lean tissue during rigorous dieting is likely an important cause of the commonly observed decrease

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Table 9: Post-exercise stimulation of metabolism by a bout of vigorous exercise.		
Author	Exercise	Increase of resting metabolism & time post-exercise
Benedict & Carpenter (1910)	Bout of cycle ergometer exercise in a caloric chamber	12.5% increase of BMR during 6 subsequent hours of sleep
Bielinski et al. (1985)	3 hr treadmill walk at 52% max. ox. intake	9% increase of RMR 4h post ex. 4.7% increase at 18 hr
Brehm & Gutin (1986)	3.2 km treadmill run at 18% to 68% max. ox. intake	No post-ex. change of RMR
de Vries & Gray (1963)	Vigorous work-out	8-28% increase, 4 hr post ex.
Edwards et al. (1935)	Harvard football players	23% increase, 13 hr post ex.
Edwards et al. (1935)	Harvard lacrosse players	8% increase, 31 hr post ex.
Freedman-Akabas et al. (1985)	20 min exercise at anaerobic threshold	No change of RMR post-exercise
Maehlum et al. (1986)	90 min cycle ergometry at 71% max. ox. intake	14% increase, 12 hr post ex.
Passmore, Johnson & Shirling (1960)	16 km walk at 6 km/hr	16% increase, 7 hr post ex.
Poehlman et al. (1989)	90 min cycle ergometry at 50% max. ox. intake	No change of RMR post-exercise

in RMR as body mass falls. Conversely, if lean tissue mass is maintained or restored when dieting is supplemented by exercise, this does much to maintain RMR. As noted above, three quarters of inter-individual differences in RMR are attributable to

corresponding differences in lean tissue mass. In studies of twin pairs during periods of deliberate under- or over-feeding, Bouchard & Tremblay (1997) argued that changes in lean tissue mass were the most important factor behind metabolic adjustments associated with changes in the availability of nutrients.

Nevertheless, a 5-6 kg increase of lean tissue would be needed to counter a dieting-induced 10% decrease of RMR, and in many studies the observed changes in lean tissue mass are insufficient to account for the decrease in RMR (Table 10).

Modification of fidgeting and incidental movements.

A significant fraction of total body metabolism can be traced to minor fidgeting movements. Schoeller & Jefford (2002) estimated that the average energy cost of 5 minor sedentary movements such as crossing and uncrossing the legs or moving small bolts around on a table ranged from 1.8-5.1 kJ/min, or 0.1-0.3 MJ if practiced for only 1 hour during the day. Plainly, such movements could be reduced during a period of rigorous dieting, or following a bout of vigorous physical activity.

In the classic Minnesota starvation experiments (Keys et al., 1950), the physical activity index (the ratio of the average energy expenditure to basal expenditures) dropped from 2.1 to 1.6 as starvation progressed, suggesting that there had indeed been a decrease in voluntary and involuntary movements. Direct observation of the study participants confirmed that they spent more time sitting and less standing as their energy deficit accumulated. A 20% change in the metabolic cost of minor incidental activities could certainly arise if

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subjects decided to sit rather than to stand during most of their free time (Ravussin et al., 1986), and after rigorous dieting, less energy might be required to move a lighter chest wall during respiration (Leibel, Rosenbaum, & Hirsch, 1995). But behaviour during extreme starvation is hardly relevant to diet and exercise programmes for clients who still retain substantial reserves of adipose tissue.

One report found a decrease of doubly-labeled water estimates of total daily energy expenditures when non-obese subjects had pursued a rigid diet (3.7 MJ/day) for 6, 9 and 12 months, but accelerometry showed no detectable change of physical activity. The authors of this investigation concluded (apparently without examining the other factors listed in Table 8) that the energy deficit must have induced a reduction of minor fidgeting movements (Martin et al., 2011).

de Groot et al. (1989) collected direct evidence on the incidental movements of obese women during a rigorous dietary programme that reduced their body mass by 6.9-9.0 kg over the course of 8 weeks. Subjects were enclosed in a respiration chamber, and monitoring by a combination of radar and actometers revealed a significant decrease in minor activities.

In contrast, Ravussin et al. (1985) claimed that decreases in lean tissue mass, the thermic effect of food, and the energy cost of moving a lighter body accounted for almost all of the decrease in energy expenditures that were seen when a protein-supplemented 4 MJ/day diet was sustained for 14 weeks, with a cumulative 10.6 kg weight loss. Their data left little margin for any decrease of fidgeting, and moreover, they saw no change of radar-detected incidental movements during the period of dieting.

Meijer (1990) found that despite the high daily energy expenditure of men preparing themselves for participation in a marathon race, minor activities remained high, with total energy expenditures substantially exceeding the immediate metabolic cost of the training.

In contrast, a study of autistic children found clear evidence of a reduction in self-stimulatory movements immediately following 20 min of aerobic jogging (Rosenthal-Malek & Mitchell, 1997).

There is little other direct evidence from human experiments, but animal studies have demonstrated decreases in spontaneous activity following periods of heavy training (Richard & Arnold, 1987; Stevenson et al., 1966; Thomas & Miller, 1958).

Table 10: Relationship between changes in lean tissue mass and changes of RMR as seen during diet- and exercise-based weight-loss programmes.

Author	Change of lean mass	Change of RMR
Ballor et al. (1996a, b)	-2.6 kg	-15%
Bryner et al. (1999)	-4.1 kg	-13.4%
Cullinen & Caldwell (1998)	+2.0 kg	+8.6%
Doi et al. (2001)	-1.8 kg	+8.1%
Gornall & Villiani (1996)	-1.4 kg	-5.5%
Kempen et al. (1995)	-1.3 kg	-10%
Poehlman & Danforth (1991)	No significant change	+10%
Schlundt et al. (1993)	-2.5%	-5.5%
Schwartz et al. (1990)	-5.8%	-8.1%
Wadden et al. (1990)	-8.6%	-8.3%

Alterations of sleep duration

Since the sleeping metabolic rate is lower than the RMR, daily energy expenditures could be increased by a shortening of the average time that is allotted to sleeping. Incidental movements are greater when awake, and an increased ingestion of food is also likely if the hours of sleeping are shortened, in part because more time is available for eating, and in part because a lack of sleep appears to augment hunger and appetite (St-Onge, 2013). Some cross-sectional reports have also described lower leptin and higher ghrelin levels in short-duration sleepers (St-Onge, 2013).

The value of exercise training in correcting the sleep apnoea of obese individuals is well recognized (Iftikhar, Kline, & Youngstedt, 2014; Shephard, 2019a). But even in the absence of such a pathology, exercise training seems to improve the quality of sleep, leading to a shorter sleep latency without change of total sleep duration (Lira et al., 2011; Yang et al., 2012). However, there is little evidence that there are changes in sleep duration that could have a major impact upon energy balance.

Increased energy expenditures on tissue growth and repair.

A shorter than anticipated stature is one measure of childhood malnutrition (Delpeuch et al., 1994). Likewise, a severe training-imposed nutrient deficiency causes the athletic triad in women (De Souza et al., 2014; Nattiv et al., 2007), with a cessation of ovulation, and in otherwise healthy adults, excessive training can limit the potential for tissue hypertrophy and repair.

However, it is less clear that such effects occur when exercise is used to impose a moderate energy deficit upon a

person who still has substantial reserves of body fat. Regular exercise (if not pushed to excess) indeed seems likely to encourage the secretion of growth hormone, with greater expenditures of energy on tissue growth and repair. Nevertheless, an excessive volume of exercise, particularly if accompanied by severe dieting, can decrease the secretion of Insulin like growth factor 1 (IGF-1), with a retardation of growth. This is indeed one biological factor contributing to the small size of international-level gymnasts (Jahreis et al., 1991).

The difference of metabolic response between exercise training alone and dieting plus exercise training is quite striking, and it suggests that the combined treatment has the potential to create too large a negative energy balance, with a resulting metabolism of lean tissue. The extent of such lean tissue loss is likely to depend on both the client's initial reserves of energy relative to the imposed energy deficit, and the amount and quality of the amino acids that are made available as total energy intake is reduced. Containment of the decrease of RMR in subjects receiving a protein supplement, as seen by Doi et al. (2001) but not by Phinney et al. (1988), offers some support to this idea. Unfortunately, few investigators have as yet examined how far dieters can facilitate the retention of lean tissue by providing essential amino acids in adequate amounts and at an appropriate time of day relative to exercise sessions (Shephard, 2012).

Hormonal adaptations

The RMR is modified by changes in the blood levels of several hormones, including leptin, thyroid secretions and catecholamines. Moreover, the output of several of these hormones is modified by a

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negative energy balance, whether created by dieting or by exercising (Williams et al., 2001)

Doucet et al. (2000) studied 40 men and women during a 15 week weight loss programme. In the men, changes in RMR were associated most closely with leptin levels, whereas in women the dominant correlate was with the change in fat mass. RMR also appears to vary in parallel with serum T-3 concentrations (Pelletier et al., 2002); the output of this hormone may be depressed by bouts of intensive exercise (Jahreis et al., 1991; Mastorakos & Pavlatou, 2005), in company with a decreased output of leptin (Simsch et al., 2002), although Fisher (1996) suggested that values in healthy individuals are normally uninfluenced either by body weight or by physical training. Rosenbaum et al. (2000) also found correlations between thyroid hormone concentrations and changes in body mass; there were also associated changes in the excretion of noradrenaline.

Periods of overly intensive physical exercise lead to increases in cortisol and decreases in testosterone concentrations, with a resulting depression of anabolic activity (Adlercreutz et al., 1986; Kraemer & Ratamess, 2005).

Thermic effect of food and possible changes in metabolites with dieting and exercise.

The thermic effect of foods arises from the energy that is consumed during their ingestion, absorption and storage. It can amount to a substantial 10-15% of daily energy expenditures. Moreover, such costs vary with the type and volume of food that is consumed, and with the extent of protein breakdown that has been incurred in response to a negative energy balance induced by dieting and/or exercise.

In theory, a change in dietary composition could modify thermic effects, since the magnitude of the specific dynamic action differs between protein- and carbohydrate- or fat-based diets (Flatt, 1985) (Table 11). In one analysis, the total thermic cost of a 10MJ/day diet was estimated at 960 kJ/d for a low fat, and 830 kJ/d for a high fat diet, and if the food intake was restricted to 5 MJ/day, the corresponding figures dropped to 460 and 400 kJ/day (Hill, Drougas, & Peters, 1993).

However, in most circumstances, any exercise-related changes in the thermic effects of foods are relatively small. Tagliaferro et al. (1986) examined changes in specific dynamic action that occurred when 10 young and healthy women undertook 10 weeks of progressive jogging at an intensity sufficient to increase their maximal oxygen intake by 7.0%. On average, there was no significant change in the thermic effect of food, although there was a trend to an increase that was correlated with the individual's gain in maximal aerobic power.

Table 11: Specific dynamic action of foodstuffs.

- Carbohydrates: 5-15% of energy consumed
- Fats: 5-15% of energy consumed
- Proteins: 20-35% of energy consumed

Alterations of proton leakage

Harper et al. (Harper et al., 2002) pointed out that proton leakage in skeletal muscle has a major influence on RMR. Thus, exercise and/or dieting might modify the RMR by changing the extent of proton leakage. Chronic energy deficiency reduces noradrenaline-stimulated thermogenesis (Kurpad, Sheela, & Shetty, 1989), and by increasing noradrenaline levels, exercise might counter this effect. However, there is as yet no evidence on

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the possible magnitude of any changes in proton leakage.

Conclusions

Mechanisms underlying the decrease of RMR that is seen with rigorous dieting, and its partial or complete reversal by the inclusion of an exercise component to weight-loss programmes remain to be clarified. As yet, much of the information is derived from rather small groups of subjects who have been followed for only a limited time, and perhaps for this reason findings are conflicting.

Probably a substantial part of the diet-induced decrease or RMR and its correction by exercise are attributable to changes in lean tissue mass and effects of the late post-exercise increase of oxygen consumption. However, there may also be smaller contributions from altered patterns of hormone secretion, electron leakage, tissue hypertrophy and repair, and incidental fidgeting movements.

Compensation for a negative energy balance by reductions in spontaneous physical activity

The final possible method of compensating for a sustained negative energy balance, whether this has been created by an exercise regimen or by dieting, is to decrease spontaneous leisure activity, one of the items that is included in some definitions of NEAT. It is quite difficult to make accurate measurements of changes in incidental physical activity. Assessments have usually been based upon self-reports, and obese individuals are notorious in over-estimating their involvement in physical activity. However, there have been some more objective observations, based upon radar, accelerometry and doubly labeled water data (Jakicic, Polley, & Wing, 1998).

In the Minnesota starvation experiments, we have already noted that Keys et al. (1950) commented on the progressive decrease in the voluntary movements of their subjects; as the cumulative energy deficit grew More time was spent sitting and less standing. However, it is less clear whether a similar response occurs if the energy intake is reduced by a more moderate amount in humans who still retain a substantial excess of body fat. If an obese and sedentary person were to engage in a rigorous dietary programme, this might cause a depression of mood state, leading to a reduction of spontaneous physical activity. Likewise, the initiation of a vigorous exercise programme might initially cause a reduction of spontaneous leisure activity because of fatigue and/or a depression of mood state. However, such fatigue would likely diminish as exercise training improved the individual's physical condition.

Despite technical problems, there has been a substantial amount of research on the extent of compensatory reductions in voluntary physical activity during periods of negative energy balance (Table 12) (World health Organisation, 1985) and on the concept of an "activitystat" that can compensate for a bout of exercise by reducing physical activity at other times of the day (Rowland, 1998). The objective approach to this question has commonly been to compare the magnitude of any added energy expenditure with the total metabolic rate for the day as measured by doubly labeled water, making appropriate allowances for changes in RMR and dietary thermogenesis.

Certainly, some articles supporting the idea of compensation for an increase of energy expenditures can be found in studies of both short term and sustained

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programmed exercise. However, the findings vary, depending at what stage of a programme observations are made; in the early stages of rehabilitation obese individuals may find an exercise programme very fatiguing, but this problem lessens as their physical condition improves (Donnelly & Smith, 2005). Since compensation is largely a behavioural phenomenon, there are also likely to be inter-individual differences, linked to personality, age and sex (King et al., 2007).

Baggett et al. (2010) examined correlations between moderate physical activity and compensatory periods of inactivity in 6916 Grade 8 schoolgirls; far from finding any compensation, for each minute of moderate physical activity on any given day, there were 1.85 min less of inactivity.

Blaak et al. (1992) had a small group of 10-11 year old obese boys undertake 4 weeks of cycle ergometry (45 min per day at 50-60% of their maximal oxygen intakes, 5 days per week). There was a 12% increase of daily energy expenditure in response to this intervention, and doubly-labeled water data provided no evidence that there had been any compensatory decrease of incidental energy expenditures.

Church et al. (2009) randomized a sample of 411 overweight or obese post-menopausal women between a control group and 3 intervention groups who undertook differing volumes of aerobic exercise (16, 32, or 48 kJ/kg of added activity per week) for a total of 6 months. With the 2 lowest volumes of added physical activity, the weight loss closely matched that predicted from the programmed increase of energy expenditures, but with the most vigorous regimen, the weight loss was only about a

half of that predicted, suggesting that some compensating reduction of leisure activity may have occurred.

Cooper et al. (2003) compared 10-year old children who walked to school with those who were driven. In the boys, accelerometer data showed that during their leisure time the walkers displayed greater physical activity than those who were driven to school, whereas leisure activities did not differ between the 2 categories of girls. Even in the boys it remains possible that those with a more active disposition chose to walk to school, rather than that the walk had a causal impact, increasing their active leisure behaviour.

Dale et al. (2000) examined the effects of restricting the physical activities of third and fourth grade students on 2 days, between the hours of 9 a.m. and 3 p.m. They found no evidence that this induced a compensatory increase of physical activity after school, between 3 p.m. and 7.30 p.m.

Goran & Poehlman (1992) had 11 elderly volunteers (aged 56-78 years) engage in 8 weeks of progressive cycle ergometry (3 sessions/week, rising to what was quite a high work rate for the elderly, a loading of 1.2 MJ/session). Although the exercise sessions increased their maximal oxygen intakes by an average of 9.1%, no increase in total daily energy intake was seen, and it was concluded that spontaneous activity had decreased by a substantial 0.96 MJ/day.

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Table 12: Influence of dieting and of exercise programmes upon voluntary leisure activity.		
Author	Observations	Conclusions
Baggett et al. (2010)	6916 Grade 8 schoolgirls	Each minute of added moderate physical activity decreased overall inactivity by 1.85 min
Blaak et al. (1992)	Ten 10-11 yr old obese boys, 4 wk cycling at 50-60% max. ox. intake, 45 min 5d/wk	No change of spontaneous activity
Church et al. (2009)	411 overweight or obese post-menopausal women, 6 months aerobic exercise at 16, 32 or 48 kJ/kg per week	With 2 lower volumes of exercise, weight loss matched energy expenditures, but at highest volume loss only 50% expected
Cooper et al. (2003)	114 children aged ~10 yr, accelerometer comparison of those walking and those driven to school	In boys, walkers also more active in their leisure time, girls no difference
Dale et al. (2000)	76 3rd & 4th grade students, activity restricted 9 a.m. to 3 p.m. for 2 days	No evidence of compensatory increase of phys. activity 3-70 p.m.
Goran & Poehlman (1992)	11 elderly volunteers, endurance training (8 wk cycle ergometry, 3 times/wk, rising to 1.2 MJ/session) increased max. ox. intake by 9%	No increase of total daily energy expenditure, due to 0.96 MJ/d decrease in voluntary activity
Kempen et al. (1995)	20 obese women, 8 weeks of severe dieting or severe dieting plus exercise (90 min 3/wk aerobic activities & dancing)	No decrease of activity with dieting, but daily energy expenditure not increased by dieting, ? some compensation
Keys et al. (1950)	Adults, prolonged starvation	Subjects observed to spend less time standing, more sitting
Kriemler et al. (1999)	14 10-15 yr old obese boys, one day of 50 min strenuous or 30 min of mod. cycle ergometry.	Heart-rate estimates of energy expenditure increased on day following mod. exercise, but decreased following intense ex.
Manthou et al. (2010)	34 overweight or obese women, 8 week exercise programme	11 responders, 23 non-responders; latter group reduced physical activity outside of programme
Meijer et al. (1991)	16 M, 16 F preparing for half-marathon over 5 months	Total activity increased 62-63%, but no compensatory change of phys. activity in non-exercise part of day.
Metcalf et al. (2004)	154 boys, 121 girls aged ~5 yr, questioned on walking to school	Walking accounted for only 2% of weekly activity, total weekly activity no lower in those driven to school
Paravidino et al. (2016)	24 obese boys aged 11-13 yr, single session of moderate or heavy exercise.	24 hr energy expenditure increased as expected; difference from control diminished over next 5 days
Shephard & Lavallée (1993).	Half of 546 primary school students performed 5 hr/wk supervised aerobic exercise for 6 yr	Diary records show no sign. change of leisure activity relative to controls
Thivel et al. (2013)	12-15 yr old boys, both obese and lean, high (75%) or low (40% max. ox. intake) exercise intensities	High intensity exercise produced compensatory decrease of voluntary exercise in obese but not in lean boys.
Van Dale et al. (1989)	12 obese women, 12 weeks of aerobic exercise, 4k/week at 55% of max. ox. intake	Actometer data shows final 27% increase of energy expenditure, compared with 10% increase in dieters
Washburn et al. (2003)	Young, overweight adults, 16 month exercise programme	Doubly labeled water shows no decrease of spontaneous activity

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Kempen et al. (1995) involved half of a sample of 20 obese women for 8 weeks in a severe dieting programme (2.0 MJ/day for 4 weeks, then 3.5 MJ/week for 4 weeks, with protein and micro-nutrient supplements), and the other half of the sample supplemented this regimen by 90 minute exercise sessions (aerobic activities and dancing, 3 times per week). The total daily energy expenditure as determined by doubly labeled water was similar for the 2 groups, suggesting that the exercisers had curtailed other aspects of their leisure activity.

Kriemler et al. (1999) examined the responses of 14 obese boys (with an average 36% body fat) after either a bout of 50 min of strenuous cycle ergometry, or 30 min of moderate cycling. On the following day, heart rate estimates of energy expenditures showed a decrease in those who had undertaken the strenuous activity, but an increase among those who had engaged in the moderate exercise.

Manthou et al. (2010) recruited 34 overweight or obese women to an 8 week exercise programme with a cumulative energy cost of 30 MJ. The other incidental activities of the subjects were recorded using a log where entries were made every 5 min. The cumulative weight loss over the programme ranged widely from -3.2 to +2.6 kg. The group was divided in terms of weight loss into responders (11) and non-responders (23). The responders showed a 1.4 MJ/day increase of daily energy expenditures, whereas in the non-responders it was only 0.3 MJ/day; moreover, active energy expenditures showed substantial differences between the two categories of subject (+0.8 MJ/day vs. -0.6 MJ/day).

Meijer et al. (1991) reported findings in 16 men and 16 women who were preparing themselves for participation in

a half-marathon run through a 5-month course of vigorous endurance training. The overall daily energy expenditure was increased by up to 63%, but there was no evidence that leisure activities outside of formal training sessions were curtailed; indeed, in the men there was a non-significant trend to an increase of leisure activity as training proceeded.

Metcalf et al. (2004) examined 154 boys and 121 girls aged ~5 years, focusing on their journey to and from school. For those who walked, this accounted for only 2% of their total weekly active energy expenditure, and this total was similar for walkers and for those who were driven to school by car, possibly providing some limited evidence for a centrally-determined cap on energy expenditures.

Paravidino et al. (2016) had 24 obese boys aged 11-13 years perform single sessions of moderate and heavy exercise, according to a cross-over design. Energy expenditures increased as expected on the first day, but over the following 5 days' differences from control sessions progressively decreased.

We conducted a quasi-experimental study where one half of a sample of 546 primary school students were required to undertake an additional hour of vigorous, supervised aerobic activity 5 days/week for six years. No dietary restrictions were imposed. Simple diary records suggested that the exercised students showed no reduction of leisure activity relative to the controls (Shephard & Lavallée, 1993).

Thivel et al. (2013) exposed 12-15 year old boys, both obese and lean, to single morning sessions of high (75%) and low (40% of maximal oxygen intake) exercise. In the obese individuals, actometer data showed that the high intensity exercise bout induced a decrease of leisure activity during the afternoon, but this

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compensatory response was not duplicated in the lean boys.

Van Dale et al. (1989) compared 12 weeks of aerobic exercise (4 hr per week at 55% of the individual's maximal oxygen intake) plus dieting with dieting alone. Actometer data showed that after 12 weeks, the physical activity level of the exercisers was increased by 27%, compared with a non-significant 10% increase in that of the dieters. No evidence of a compensatory decrease of leisure activity was seen in the exercised group.

Washburn et al. (2003) observed no changes in the spontaneous leisure activity of young overweight adults, as determined by the doubly-labeled water technique, over the course of a 16-month exercise programme.

Two systematic reviews illustrate the lack of a current consensus on this question. In a systematic review of 31 articles through 2013, all of 4 cross-sectional studies, 9 of 10 short-term studies, 5 of 10 non-randomized trials, and all of 7 randomized trials (Washburn et al., 2013) found no evidence of a compensatory reduction in incidental activity. However, the authors admitted the need for larger-scale studies, with a greater reliance upon objective techniques to determine both programmed energy expenditures and spontaneous leisure activities. In contrast, a second review of 28 articles, completed at about the same time, claimed that there was evidence of compensation in 15 of the 28 papers cited (Gomersall et al., 2013). Much of this discrepancy probably reflects problems of methodology. Subjective reports of incidental movement are necessarily imprecise and may exaggerate the extent of minor leisure activity and incidental movements. Actometers also fail to record important segments of physical activity

such as cycling or the energy expended in climbing hills, and doubly labeled water studies usually include in their estimates all sources of energy expenditure other than prescribed exercise and basal metabolism (including all the various sources of variations in energy expenditure discussed above). Findings are probably also influenced by the intensity of effort required, and/or the body build of subjects (obese vs. lean) (Thivel et al., 2013).

Discussion and conclusions

At first inspection, it seems plausible that evolutionary pressures will have caused humans to develop one or more biological mechanisms to defend themselves against a prolonged period of negative energy balance; this would offer some support to the claims of nutritionists that an increase of daily physical activity is unlikely to be very effective in correcting obesity, because the added energy costs of regular exercise are largely compensated by decreases in other components of daily energy expenditures. Phenomena such as the athletic triad, and the disturbance of ovarian function encountered when endurance athletes combine a heavy endurance training programme with a severe restriction of food intake have been cited as substantiating this idea. However, any compensatory mechanisms would likely have evolved to protect hunter-gatherers against an excessive energy expenditure during periods of extreme famine, and it is unlikely that such processes would be stimulated by a negative energy balance if a person still had a substantial reserve of body fat. Certainly, a detailed review of the possible methods whereby daily energy expenditures might be reduced suggests that their combined impact is small when

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individuals who have a substantial reserve of body fat engage in physical activity programmes that create only a modest negative energy balance.

Nevertheless, the volume of additional physical activity needed for an effective prevention or correction of obesity is greater than the minimal general public health recommendations for daily activity, probably amounting in adults to 150-250 min of moderate to vigorous aerobic activity per week. Further, if a client chooses to increase dietary intake in parallel with an increase of physical activity, this can sometimes lead to the failure of an otherwise well-designed exercise-based weight-loss programme. On the other hand, the required volume of daily physical activity seems within the capacity of a moderately obese individual who is prepared to accept a long-term weight management plan.

There is also little strong evidence that an exercise-based stimulation of appetite will inevitably counter the desired weight loss. Often, appetite is suppressed rather than increased. Indeed, in the few instances where comparisons have been made, higher intensities of effort seem to suppress appetite more than moderate physical activity, although more studies are needed, including an evaluation of appetite responses to a chronic increase of physical activity rather than a one-day exercise bout. There remains a need to look at possible differences of response between lean and obese individuals, and to consider effects related to the type of physical activity (for example swimming vs. athletics). Does the apparent difference of findings between subjective ratings of appetite and measures of dietary intake simply reflect the longer duration of observations in the latter type of investigation? Is there a point where the

decrease in reserves of stored fat triggers a hormonal response that increases food consumption? And is this threshold modified if protein supplements are provided to those who are attempting to lose weight? As yet, there has been little assessment of either the overall value of amino-acid supplements in dieters, or on the need to ingest such nutrients at an optimal time relative to exercise sessions.

There is good evidence that resting metabolism is depressed substantially by dietary restriction, but such compensation is much less apparent when a similar energy deficit is induced by exercising? Is this because lean tissue mass is conserved in the exercisers, and if so can this benefit be enhanced by a focus on resistance rather than aerobic exercise and/or the provision of protein supplements? Although the lean tissue hypothesis is tempting, a 5-6 kg increase of lean tissue would be needed to counter a 10% decrease of RMR, and in many studies the change of lean tissue mass is insufficient to account for all of the reported benefits of exercise relative to dieting. Other factors contributing to the maintenance of RMR in the exercisers may include the late post-exercise increase of oxygen consumption, altered patterns of hormone secretion, greater electron leakage, the consumption of energy in tissue hypertrophy and repair, and a lesser suppression of incidental fidgeting movements.

Reductions in more major components of leisure activity could also counter an exercise-based weight reduction programme, but to date evidence on the extent of any such changes is conflicting. Many of the current discrepancies probably reflect problems of methodology: subjective reports of incidental leisure activities are necessarily

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imprecise and may exaggerate the extent of such energy expenditures. On the other hand, actometers fail to record important segments of human activity, and calculations based on the metabolism of doubly labeled water studies usually include in their totals sources of energy expenditure other than prescribed exercise and other voluntary leisure activity.

In all, if the prevention and/or correction of obesity is viewed as a long-term project, there seems no good reason why a well-designed exercise programme should not achieve its primary objective of reducing excess body fat, while offering many other important health benefits.

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Author's Qualifications

The author's qualifications are as follows: Roy J. Shephard, C.M., Ph.D., M.B.B.S., M.D. [Lond.], D.P.E., LL.D., D.Sc., FACSM, FCSEP, FFIMS, FAAPE.

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