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The Role of Diet and Exercise for the Maintenance of Fat-Free Mass and Resting Metabolic Rate During Weight Loss

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Contents

Abstract

The incidence of obesity is increasing rapidly. Research efforts for effective treatment strategies still focus on diet and exercise programmes, the individual components of which have been investigated in intervention trials in order to determine the most effective recommendations for sustained changes in bodyweight. The foremost objective of a weight-loss trial has to be the reduction in body fat leading to a decrease in risk factors for metabolic syndrome. However, a concomitant decline in lean tissue can frequently be observed. Given that fat-free mass (FFM) represents a key determinant of the magnitude of resting metabolic rate (RMR), it follows that a decrease in lean tissue could hinder the progress of weight loss. Therefore, with respect to long-term effectiveness of weight-loss programmes, the loss of fat mass while maintaining FFM and RMR seems desirable.

Diet intervention studies suggest spontaneous losses in bodyweight following low-fat diets, and current data on a reduction of the carbohydrate-to-protein ratio of the diet show promising outcomes. Exercise training is associated with an increase in energy expenditure, thus promoting changes in body composition and bodyweight while keeping dietary intake constant. The advantages of strength training may have greater implications than initially proposed with respect to decreasing percentage body fat and sustaining FFM. Research to date suggests that the addition of exercise programmes to dietary restriction can promote more favourable changes in body composition than diet or physical activity on its own. Moreover, recent research indicates that the macronutrient content of the energy-restricted diet may influence body compositional alterations following exercise regimens. Protein emerges as an important factor for the maintenance of or increase in FFM induced by exercise training. Changes in RMR can only partly be accounted for by alterations in respiring tissues, and other yet-undefined mechanisms have to be explored. These outcomes provide the scientific rationale to justify further randomised intervention trials on the synergies between diet and exercise approaches to yield favourable modifications in body composition.

The prevalence of obesity has risen enormously over the past few decades. According to the *World Health Report 2002*,[1] obesity has increased 3-fold in some parts of North America, Eastern Europe, the Middle East, the Pacific Islands, Australasia and China since 1980. Defined by a body mass index >30 kg/m^2,[2] obesity has been associated with many diseases, including some forms of cancer,[3] type 2 diabetes mellitus,[4] stroke,[5] coronary heart disease, hypertension, dyslipidaemia, gallbladder disease, sleep apnoea[6] and osteoarthritis.[7]

Factors underlying this epidemic are complex. Although genetic susceptibility may play a role in the development of excessive adiposity,[8,9] diet factors, such as the availability of energy-dense food, and the widespread reduction in physical activity clearly make critical contributions.[10] Morbidity and mortality associated with obesity are substantial, but can be effectively reduced following weight reduction.

As early as 1975, a multi-factorial approach was used for the treatment of obesity, including dietary modification, exercise, psychotherapy and medication.[11] Over the past 2 decades, numerous intervention studies have examined strategies for the prevention and treatment of obesity. Diet and exercise are still the key variables for both men and women to imbalance the energy equation in the direction of weight loss. An abundance of studies provide evidence of successful bodyweight reductions following dietary restriction and physical activity.[12-15] However, in terms of the magnitude of change, reductions in bodyweight are often below expectations.[16-18] Moreover, the majority of people are not able to maintain the achieved losses and, over the long term, weight regain is usually the case.[19-21]

The reasons for the limited long-term effectiveness of conventional treatment strategies are diverse. The failure of changing lifestyle habits with respect to dietary intake and regular physical activity during follow-up is well established.[22,23] While behavioural issues are certainly the cornerstones, weight maintenance appears to be antagonised by a reduction in resting metabolic rate (RMR). As the largest component of daily energy expenditure, RMR comprises approximately 60–70%. Fat-free

mass (FFM) is the main factor that accounts for the magnitude of resting metabolism.[24-27] As a heterogeneous compartment, FFM consists of highly metabolically active muscle and organs and low-metabolic rate tissues such as bone and connective tissue.[28] [1] Therefore, any diet or exercise interventions, which are capable of maintaining FFM or at least attenuating its decline following weight loss, could have significant effects on total energy balance. The residual variation in RMR seems to be related to a diversity of physiological parameters, such as thyroid hormones, leptin levels and sympathetic nervous system activity.

The foremost objective of a weight-loss trial has to be the reduction in fat mass leading to a decrease in risk factors for metabolic syndrome. However, an accompanying loss in FFM can frequently be observed. Both with regard to a reduction in risk factors and long-term weight maintenance the content of adipose tissue in the weight lost has to be maximised, thus preserving FFM. It appears that some dietary regimens induce a higher loss in FFM than others, with the macronutrient composition and the energy content of the diets having a major impact on the composition of the bodyweight lost.[29,30] Another means by which a decline in bodyweight can be achieved while favourably modifying body composition with the maintenance of FFM is through physical activity.[31-33] The ability of exercise programmes to achieve these goals depends on the prescribed type and magnitude of exercise. Therefore, as numerous studies show, a reduction in RMR does not necessarily accompany a loss in FFM and alterations in RMR might occur independent of changes in muscle tissue.[34,35] Some studies have reported that a low RMR is a determinant of weight gain, thus attenuating the decline in RMR is desirable.

Accordingly, the purpose of this review is to document recent (1990–2005) results of diet intervention studies, exercise intervention studies and studies that have investigated the combined effects of diet and exercise for the treatment of obesity. The article covers well controlled, randomised clinical trials conducted in subjects with at least a minor degree of over-fatness or overweight and where data on body compositional changes were provided. The efficacy of the identified investigations was compared in terms of changes in bodyweight, fat mass, FFM and RMR. As only a limited number of research reports assessed energy expenditure, missing data on RMR were not considered a criterion for exclusion of the study. This article will conclude with an evaluation of treatment strategies for obesity, based on diet and exercise programmes and directions for future research will be discussed.

1. Diet Intervention Studies

1.1 Dietary Energy Restriction and Resting Metabolic Rate (RMR)

RMR is the energy expended by the active cell mass to maintain normal body functions at rest. There is evidence that RMR is largely dependent on FFM.[36-38] Several intrinsic factors, which are beyond the control of the individual, seem to account for the residual variation in RMR, including age, sex, thyroid status[39] and genetic factors.[40]

It is a well known fact that weight loss induced by dietary restriction is accompanied by a decline in RMR. A decrease in FFM mass most certainly has considerable contribution to this effect[41] (figure 1). Concomitant neuroendocrine disturbances, such as alterations in leptin level,[42,43] thyroid status[44] and sympathetic nervous system activity[45] may further contribute to the decrease in RMR. This raises the question as to whether the composition of the diet can modify physiological adaptations to energy restriction, thus blunting the fall in RMR. The reduc-

1 Throughout this article, the terms ‘fat-free mass’ and ‘lean body mass’ are used interchangeably.

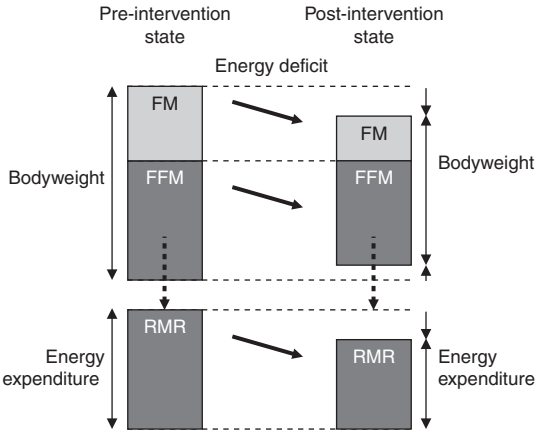

Fig. 1. Theoretical impact of energy restriction on fat mass (FM), fat-free mass (FFM) and resting metabolic rate (RMR). Energy deficit over a prolonged period of time induces a reduction in bodyweight, which is based on a decrease in FM and, possibly, FFM. These body compositional changes might be associated with a decline in RMR.

tion of adipose tissue during weight loss induces an associated decline in the secretion of leptin.[46] Changes in leptin level have been shown to predict changes in RMR. However, a positive association is still noticeable after adjusting for body composition,[42] indicating an effect of leptin on RMR independent of fat mass. Therefore, the maintenance of leptin appears to be desirable for weight stability in the post-obese state.[47] There is some evidence for a positive association between carbohydrate (CHO) consumption and leptin concentration during energy restriction,[48] suggesting an impact of the macronutrient content of the diet on energy expenditure.

In order to prevent the decline in RMR, sustaining FFM emerges as an important aim. Studies reveal that a protein intake sufficient to prevent negative nitrogen balance might be of great importance to lessen the decline in muscle mass[49] and energy expenditure (24-hour energy expenditure and sleeping metabolic rate).[50] Thus, it is apparent that various factors associated with dietary composition can modify the physiological adaptations to energy restriction. Although the potential effect of the macronutrient intake on the maintenance of FFM and RMR has received little attention to date, several intervention studies have included those variables in their outcome measurements.

1.2 High-Carbohydrate, Low-Fat Diets

There has been extensive research in the macronutrient composition of the diet and its effect on bodyweight reduction (table I). One popular approach within studies for the treatment of overweight entails altering the CHO to fat ratio in the diet. The CARMEN (Carbohydrate Ratio Management in European National diets) study, the largest and most recent multicentre trial, tested the impact of complex versus simple CHO in an *ad libitum*, fat-reduced (by 10% of total energy) regimen in 398 overweight individuals. After 6 months, a significant reduction in bodyweight and fat mass was observed on both diets while sustaining FFM.[12] This was further investigated in 45 overweight females. Similar to the CARMEN study, a low-fat, high-CHO diet led to a spontaneous decline in bodyweight, which was independent of the glycaemic index of the foods consumed.[51] Therefore, the removal of a substantial amount of fat from the diet leads to a spontaneous decline in bodyweight. As shown in several other studies, the mechanism appears to be due to the concomitant energy reduction.[51-54] Although some studies advocate the consumption of foods with a low glycaemic index, this was not confirmed in the two above-mentioned studies.[12,51] In a recent review on high-CHO diets and energy balance, Brand-Miller et al.[55] claimed that foods that promote a high glycaemic response, such as potatoes, breads and low-fat cereal products, tend to favour body fat gain. Another approach was explored by randomly assigning 40 overweight women to an *ad libitum*, low-CHO diet or an energy-restricted, low-fat diet. After 4 months, the loss in bodyweight and fat mass was greater in the low-CHO group and reductions in FFM and RMR were recorded. These results could not be accounted for

Table I. Diet intervention studies

Study	Dietary intervention	Subjects	BMI (kg/m^2)	Duration (wk)	Δ Bodyweight (%)	Δ Fat mass (%)	Δ FFM (%)	Δ RMR (%)
Saris et al.[12]	CC: ad lib, low-fat, high complex CHO	389	30.4	26	SC: −1.0[a]	SC: −3.8[a]	NS	
	SC: ad lib, low-fat, high simple CHO	F, M			CC: −2.0[a]	CC: −5.3[a]		
	C: control	M			C: NS[b]	C: NS[b]		
	N: seasonal control group							
Brehm et al.[56]	LC: ad lib, low CHO (20 g/day)	40 F	33.2	16	LC: −16.3[a]	LC: −6.7	LC: −6.8[a]	LC: −5.9
	LF: 30% fat, 55% CHO				LF: −8.7[b]	LF: −2.3	LF: −10.8[b]	LF: −5.5
Sloth et al.[51]	HGI: <30% fat, high GI	45 F	27.6	10	HGI: −1.6	HGI: −1.3	−1.6	
	LGI: <30% fat, low GI				LGI: −3.5	LGI: −3.5		
Skov et al.[57]	C: control diet (40% fat)	50 F, 15 M	30.4	26	HP: −10.0[a]	HP: −26.7[a]		
	HP: ad lib, 30% fat, 25% PRO				HC: −5.6[b]	HC: −14.1[b]		
	HC: ad lib, 30% fat, 12% PRO							
Baba et al.[58]	80% of RMR, 30% fat	13 M	35.8	4	HP: −7.3[a]	−6.7	−6.1	HP: −5.9[a]
	HP: 45% PRO	HI			HC: −5.7[b]			HC: −17.4[b]
	HC: 12% PRO							
Torbay et al.[35]	80% of RMR, 30% fat	14 HI	35.1	4	HPHI: −7.3[a]	−15.7	NS	HPHI: −5.9[a]
	HP: 45% PRO	13 NI			NI: −5.9[b]			NI: −4.0[a,c]
	HC: 12% PRO	M			HCHI: −5.7[b]			HCHI: −17.5[b,d]
					NI: −6.6			NI: −18.1[c,b]
Farnsworth et al.[30]	70% TEE, 4wk E balance (30% fat):	14 M	34.0	12	−8.5	−17.3	−2.5	
	HP: 27% PRO	43 F						
	SP: 16% PRO							
Luscombe-Marsh et al.[59]	70% of TEE, 4wk E balance	32 F	34.0	12	−9.5	−13.2	−5.9	−4.0
	HP: 29% fat, 34% PRO	15 M						
	SP: 45% fat, 18% PRO	HI						
Luscombe et al.[60]	70% TEE, 4wk E balance (30% fat)	10 M	34.1	12	−8.4	−16.5	−3.0	−8.8
	HP: 27% PRO	26 F						
	SP: 16% PRO	HI						

Continued next page

Table I. Contd

Study	Dietary intervention	Subjects	BMI (kg/m²)	Duration (wk)	Δ Bodyweight (%)	Δ Fat mass (%)	Δ FFM (%)	Δ RMR (%)
Layman et al.[29]	<1700 kcal/day	24 F	30.3	10	−8.4	−13.3	NS	
	HP: 30% PRO, 41% CHO							
	HC: 16% PRO, 58% CHO							

a Significantly different from b, c and d at $p < 0.05$.

b Significantly different from a, c and d at $p < 0.05$.

c Significantly different from a, b and d at $p < 0.05$.

d Significantly different from a, b and c at $p < 0.05$.

ad lib = *ad libitum*; **BMI** = body mass index; **C** = control group; **CC** = diet high in complex carbohydrates; **CHO** = carbohydrate; **E** = energy; **F** = females; **FFM** = fat-free mass; **GI** = glycaemic index; **HC** = high-carbohydrate diet; **HCHI** = high-carbohydrate diet, hyperinsulinaemic; **HGI** = high GI; **HI** = hyperinsulinaemic; **HP** = high-protein diet; **HPHI** = high-protein diet, hyperinsulinaemic; **LC** = low-carbohydrate diet; **LF** = low-fat diet; **LGI** = low GI; **M** = males; **N** = no intervention; **NI** = normoinsulinaemic; **NS** = not statistically significant ($p > 0.05$); **PRO** = protein; **RMR** = resting metabolic rate; **SC** = diet high in simple carbohydrates; **SP** = standard protein diet; **TEE** = total energy expenditure; Δ = change.

by changes in components of energy expenditure or intake through 3-day food records and most likely represent underreporting.[56] Thus, although a low-fat diet seems to induce a spontaneous loss in body fat, this may be accompanied by reductions in FFM. In addition, the current opinion on the impact of the type of CHO on weight loss seems inconclusive.

1.3 High-Protein Diets

Recently, renewed interest was shown in whether replacing some dietary CHO with protein may favourably affect bodyweight and body composition during energy restriction. Addressing this question, Skov et al.[57] randomly assigned 60 overweight subjects to a control diet or an *ad libitum* low-fat diet, which was either high in protein or CHO. After 6 months, a clinically relevant loss of bodyweight (−8.7 vs −5.0kg, $p = 0.0002$) and fat mass (−7.6 vs −4.3kg, $p < 0.0001$) was achieved on both low-fat diets, which was greater in the high-protein than the high-CHO group. The study design allowed the participants to collect the foods from a shop set up by the researchers. Therefore, between-group differences, such as a limited variety of foods shown to influence bodyweight,[61] may have biased the results. However, the observed tendency was confirmed by a well controlled study in 24 overweight women. The investigators examined the effects of low-fat meals with CHO/protein ratios of 3.5 and 1.4. After 10 weeks, declines in bodyweight and fat mass were similar across groups, but the participants in the high-protein group achieved an elevated fat/lean tissue loss ($p < 0.05$).[29] Moreover, a study in 13 hyperinsulinaemic obese men with normal fasting plasma glucose levels showed similar effects after 4 weeks on a hypo-energetic diet. A high intake of protein was associated with 28% more weight loss (−8.3 vs −6.0kg, $p < 0.05$) than the isocaloric consumption of a diet high in CHO. It is possible that this effect is partly derived from a significantly greater loss of total body water in the high-protein

compared with the high-CHO group.[58] In subjects with normal insulin levels following the same protocol, no major differences have been detected. However, despite the comparable decline in lean tissue, the high-protein diet was superior to the high-CHO diet in maintaining RMR ($p < 0.05$),[35] in both hyperinsulinaemic and normoinsulinaemic participants.[35]

In contrast, a study in 57 overweight subjects with slightly elevated insulin levels (>12 mU/L) failed to show an association between the protein content of the diet and weight reduction after 12 weeks of energy restriction and 4 weeks of energy balance. The results, however, indicated a preservation of lean mass during weight loss in the 43 female subjects ($p = 0.02$). The authors pointed out that the lack of an effect in the 14 male subjects may be due to the small number participating in the study and a reported protein intake, which was sufficient to prevent proteolysis in women, but not in men.[30] A similar reduction in RMR was observed after the two diets, presumably in a subgroup (36 subjects) of the above-mentioned study.[60] In a recent study investigating energy restricted diets high in protein or monounsaturated fat, no differences with respect to body composition and RMR were found.[59]

The studies reviewed in this section indicate that replacing some dietary CHO with protein might favourably affect body compositional changes during energy restriction compared with fat-restricted, high-CHO diets. The explanation for these observations, however, remains unclear. It is well established that the utilisation of ingested nutrients for energy is inversely related to the thermogenesis of food, a phenomenon associated with the energy cost of nutrient absorption, processing and storage.[62] This loss of energy is highest for protein consisting of 25–30% of the ingested energy, followed by CHO with 6–8% and fat with only 2–3%.[63,64] Therefore, a higher thermogenic response following the intake of protein compared with isocaloric preloads of either CHO or fat may make some contribution. This effect may also account for the reduced fall in 24-hour energy expenditure when maintaining the protein content of an energy-restricted diet.[50] Furthermore, compared with high-protein diets, hormonal responses associated with high-CHO diets may induce a series of physiological events favouring catabolism of FFM. A rise in insulin following a high intake of CHO promotes the uptake of glucose and triacylglycerol in the liver and adipose tissue, reduces glycogenolysis and lipolysis, thus suppressing the post-absorptive appearance of glucose and fatty acid. This triggers the release of counter-regulatory hormones inducing catabolism of lean mass. Following the intake of a meal high in protein, less reliance on peripheral insulin actions and a delayed postprandial rate of disposal for amino acids appear to stabilise the glycaemic environment[65] and may conserve lean tissue. Moreover, the metabolic role of leucine and the branched-chain amino acids for muscle protein synthesis seems consistent with the conservation of FFM on high-protein diets.[65]

In one study, high-protein diets were investigated under *ad libitum* conditions.[57] Subjects were required to cut out foods high in CHO. A greater reduction in caloric intake due to the limitation in food choice rather than an effect of macronutrient composition may have facilitated the observed loss of bodyweight. This theory is supported by the finding that the variety of sweets, snacks and CHO consumed is positively associated with body fatness.[66] Furthermore, more subtle aspects of palatability of foods high in protein cannot be discounted. Nevertheless, several studies suggest that protein promotes satiety and reduces appetite when compared with fat and CHO[29,67-71] and only a small number oppose this view.[72,73]

Therefore, in the light of recent data, high-protein hypocaloric diets may facilitate weight loss for overweight subjects while more lean tissue (and there-

fore RMR) might be preserved. Moreover, a diet high in protein was superior to conventional diets in improving insulin sensitivity[29,58,74,75] and no deleterious effects on blood pressure,[74] total cholesterol, triglycerides[29,30,58,74,75] and bone turnover[30,57] have been reported. However, according to Eisenstein et al.[76] detrimental impacts on bone health and renal disease can not be discounted as dietary protein increases urinary calcium excretion. Evidence from a study conducted by Kerstetter et al.[77] weighs against this hypothesis, as 80% of the protein-induced calciuria was compensated for by increased intestinal absorption of dietary calcium. In addition, when substituting foods high in protein (e.g. dairy products and nuts) for highly refined foods in the grain and starch groups (e.g. bread, rice, cereals and pasta) potential adverse effects of a high intake of protein may be reduced by other nutrients found in the protein source (e.g. high amounts of calcium in milk or high potassium levels of legumes).[78]

Based on the evidence noted in this section, the frequently recommended high-CHO, low-fat diets have not always been associated with the greatest loss in bodyweight. Complex and as yet incompletely understood physiological processes associated with a high intake of protein seem to facilitate reductions in body fat, with less declines in FFM and RMR. However, evidence is still scarce and further examinations are needed in order to clarify the relative merits of diets high in protein.

2. Exercise Intervention Studies

2.1 Exercise and RMR

Controlled trials on the effects of an increased physical activity level as the primary intervention without diet modification provide the best way to determine exercise effects on weight, body composition and RMR. An increase in physical activity without changing energy intake can successfully promote negative energy balance, thereby decreasing body fat. As importantly, weight loss due to exercise may be associated with a retention of lean body mass. Although weight reduction following physical activity is mainly brought about by the energy expended during the exercise bout, additional mechanisms may increase resting metabolism, thus further promoting energy imbalances. Given the association between lean tissue and RMR, the most apparent impact of exercise training on resting metabolism is the ability to initiate skeletal muscle growth.[79] In addition, increases in energy expenditure during the post-exercise recovery period (excess post-exercise oxygen consumption) may induce a short-term rise in metabolic rate for >24 hours.[80] However, as recently reviewed, this effect might only be noticeable following medium- to high-intensity exercise.[81] Despite a more rapid return to baseline levels in trained individuals,[82,83] regular exercise bouts may promote small elevations in RMR.[84,85] Further mechanisms, by which exercise may increase metabolic rate, have yet to be fully elucidated, but may include uncoupled respiration,[86,87] protein turnover[88] and sympathetic nervous system activity.[89] Therefore, the theoretical effects of physical activity on changes in body composition are apparent. Nonetheless, with regard to RMR, the literature to date is still inconclusive, as exercise training has also been associated with reductions in RMR.[90,91] As will be seen, the outcomes of exercise intervention studies with respect to body compositional alterations seem to be related to the type of exercise, with intensity, frequency and duration of the exercise bouts as limiting factors.

2.2 Aerobic Exercise

The design of exercise intervention studies varies in terms of the mode, frequency, intensity and duration of exercise. Whereas total energy requirements rely on absolute exercise intensity, relative exercise intensity determines the contribution of fat and CHO as fuels. It is well established that fat is the predomi-

nant source of energy during physical activity at low to moderate intensities (<50% maximum oxygen consumption [$\dot{V}O_{2max}$]). A progressive decline of fat oxidation in favour of CHO occurs with increasing intensities (>50% $\dot{V}O_{2max}$).[92] As oxygen supply to the muscles is not sufficient at these intensities due to the limited capacity of the heart and lungs, glucose is used as the only fuel that can be oxidised anaerobically.[93] Despite the relative decline in fat oxidation with higher exercise intensities, the absolute quantity of fat oxidised may augment as the amount of work performed increases. Thus, with the aim of utilisation of lipid stores and a high caloric expenditure, high-intensity exercise might seem more beneficial. However, in the light of a low aerobic fitness and a bodily condition not capable of high-intensity training in overweight and obese patients, investigations into physical activity as a weight-loss tool have traditionally been based on low- to moderate-intensity exercise.

van Aggel-Leijssen et al.[94] conducted a study in 21 pre-menopausal women with either lower- or upper-body obesity (table II). All participants with lower-body obesity were assigned to 12 weeks of a low-intensity cycling programme and the women with upper-body obesity were randomly divided into an exercising and a control group. The study outcomes failed to demonstrate any changes in body composition and RMR across the groups. The same exercise modality was examined in the HERITAGE Family Study,[95] a highly controlled multicentre clinical trial, with a study population of 557 subjects of various races and ages. The 20-week training programme consisted of exercise sessions on 3 days per week with increasing intensity. The results suggested small, but statistically significant, reductions in total body mass and fat mass and an increase in FFM, with no changes in RMR. In women, bodyweight did not change as the loss of fat mass equalled the gain in FFM. This was further investigated in the Midwest Exercise Trial, a randomised 16-month supervised trial with 31 men and 43 women. Aerobic exercise on 5 days a week significantly decreased bodyweight (−2.9kg) and fat mass (−4.8kg) in men. Interestingly, despite the considerable amount of exercise performed, there were no body compositional changes from baseline in the female participants, but the control group gained a significant amount of weight ($p < 0.05$).[96,97] Thus, the results of these studies show small, but significant, reductions in body fat.

To determine, whether the intensity of exercise alters outcomes with respect to bodyweight and composition, Grediagin et al.[99] randomly assigned 12 untrained, moderately overweight women to either a high-intensity or a low-intensity exercise group. After 12 weeks of four-times weekly treadmill training (with a duration designed to expend 300 kcal), no statistically significant differences were detected between the groups. In accordance, a study set up by van Aggel-Leijssen et al.,[34] with a similar design including a control group, failed to show an effect on body composition. Twenty-four obese male volunteers trained according to a cycle instead of a treadmill test protocol on only three occasions per week. Furthermore, RMR was slightly lower after the 12-week intervention following the high-intensity training (−179 kcal/day, $p < 0.05$).

The impact of exercise frequency and duration was assessed in 22 sedentary, moderately obese females, who were randomly assigned to a continuous exercise group or an intermittent exercise group. After 18 months, reductions in body fat (−2.1kg, $p < 0.05$) and weight (−1.5kg, $p < 0.05$) were only seen in the continuous exercise group, whereas FFM was unchanged in both.[100] Disparity in outcomes for weight loss may have been a consequence of differences in total weekly energy expenditure between the groups. To further investigate this, Sykes et al.[101] conducted a study in 30 overweight Singaporean women, who exercised at moderate intensity on 2 and 5 days a week, respectively, with total

Table II. Exercise intervention studies

Study	Exercise intervention	Subjects	BMI (kg/m^2)	Duration (wk)	Δ Bodyweight (%)	Δ Fat mass (%)	Δ FFM (%)	Δ RMR
van Aggel-Leijssen et al.[94]	UB + LB: cycling, 40% $\dot{V}O_{2max}$ 3 d/wk, EE = ~250 kcal/d UB-C: no intervention	13 F UB 8 F LB	32.7	12	NS	NS	NS	NS
Wilmore et al.[95,98]	Cycling from 55% $\dot{V}O_{2max}$ 30 min to 75% $\dot{V}O_{2max}$ 50 min 3 d/wk	299 F 258 M	25.4	20	M: −0.5 F: NS	M: −4.8[a] F: −2.3[b]	+0.9	NS
Donnelly et al.[96] and Kirk et al.[97]	E: aerobic (primarily walking), from 55% to 70% $\dot{V}O_{2max}$ 20–45 min/d 5 d/wk C: no intervention	43 F 31 M	29.4	69	M: −5.1 F: NS[a] C: +3.6[b]	M: −11.1 F: NS[a] C: +7.1[b]	NS	
Grediagin et al.[99]	LI: 50% $\dot{V}O_{2max}$ 4 d/wk, EE = 300 kcal/d HI: 80% $\dot{V}O_{2max}$ 4 d/wk, EE = 300 kcal/d	12 F	25.0	12	−1.3	−1.1	−3.0	
van Aggel-Leijssen et al.[34]	LI: 40% $\dot{V}O_{2max}$ 3 d/wk, EE ≈350 kcal/d HI: 70% $\dot{V}O_{2max}$ 3 d/wk, EE ≈350 kcal/d C: no intervention	24 F	31.8	12	NS	NS	NS	HI: −7.8 LI: NS
Donnelly et al.[100]	CONT: walking 60–75% $\dot{V}O_{2max}$ 30 min 3 d/wk I: walking 2 × 15 min 5 d/wk	22 F	31.2	78	CONT: −1.8 I: NS	CONT: −6.2[a] I: NS[b]	NS	
Sykes et al.[101]	E1: treadmill + cycling 400 kcal 5 d/wk E2: treadmill + cycling 1000 kcal 2 d/wk	30 F	23.8	8	−3.1	−4.2	NS	
Schmitz et al.[102]	R: resistance 50 min 2 d/wk C: no intervention	60 F	26.6	15	NS	R: −3.9[a] C: NS[b]	R: +2.3[a] C: NS[b]	
Cullinen and Caldwell[103]	R: resistance 45 min 2 d/wk C: no intervention	30 F	23.0	12	NS	R: −8.1 C: NS	R: +4.5 C: NS	NS
Byrne and Wilmore[104]	R: resistance 4 d/wk RW: resistance 4 d/wk + walking 20–40 min 3 d/wk C: no intervention	19 F	27.0	20	NS	NS	+4.0	R: +3.0 RW: −3.8

a Significantly different from b at $p < 0.05$.

b Significantly different from a at $p < 0.05$.

BMI = body mass index; **C** = control group; **CONT** = continuous exercise group; **E** = exercise group; **E1** = exercise group one; **E2** = exercise group two; **EE** = energy expenditure; **F** = females; **FFM** = fat-free mass; **HI** = high-intensity exercise; **I** = intermittent exercise group; **LB** = lower-body obesity; **LI** = low-intensity exercise; **M** = males; **NS** = not statistically significant ($p > 0.05$); **R** = resistance exercise group; **RMR** = resting metabolic rate; **RW** = resistance exercise and walking group; **UB** = upper-body obesity; **UB-C** = upper-body obesity control group; **$\dot{V}O_{2max}$** = maximal oxygen consumption; Δ = change.

energy expenditure being the same. After 8 weeks, a significant loss of bodyweight and body fat indicated beneficial effects regardless of the training frequency.

In summary, prolonged, sub-maximal exercise may result in a small decline in bodyweight and fat mass. However, the magnitude of the change appears to be less than expected and several exercise protocols failed to reduce body fat. The impact of exercise intensity on FFM and RMR warrants further evaluation.

2.3 Resistance Exercise and Combined Aerobic and Resistance Exercise

The relatively small muscle mass usually used during resistance exercise creates lower metabolic demands than aerobic exercise. Therefore, strength training was considered to provide only minimal stimulus to reduce body fat. However, as an important benefit of resistance exercise, Walberg[105] reported in her review preliminary evidence that suggests weight training in favour of aerobic exercise to preserve or increase FFM and RMR, while decreasing body fat. Referring to experimental data, in a recent study in 60 women aged between 30 and 50 years, 15 weeks of supervised strength training on 2 days per week resulted in a small but significant increase in FFM (+0.89kg) and a similar loss in fat mass (−0.98kg) when compared with a control group. These body compositional changes were maintained over 6 months through an unsupervised programme.[102] To investigate whether a combination of resistance training and walking was more beneficial than resistance exercise on its own, Byrne and Wilmore[104] set up a study in 19 moderately obese women. After 20 weeks, measurements of body composition indicated an increase in lean body mass in both exercise groups compared with a sedentary control group, which was associated with an elevation in RMR (+44 kcal/day) in the strength-trained subjects. The most striking results, however, were in those individuals that did a combination of resistance training and walking exercise, for whom RMR declined (−53 kcal/day), despite an increase in lean body mass. The authors suggested that the reduction in RMR was a result of heat acclimation as average monthly temperatures increased from the pre- to the post-training periods. Speakman and Selman[106] discussed in a recent review a down-regulation of uncoupling-protein 3 in muscle, enhancing mechanical efficacy during physical activity, as a possible reason for a decline in RMR.

In summary, considering the amount of physical activity prescribed in the above-mentioned exercise intervention studies, the magnitude of the changes in each of the variables was relatively small. With respect to those studies that included measurements of energy expenditure of the exercise bouts, theoretical losses in body fat could be calculated beforehand. The observed reductions were frequently below the expectations, and some exercise programmes failed to induce changes.[16,34] This raises the question of whether a compensation for the energy deficits has occurred, either as a compensatory rise in food intake, a decrease in spontaneous physical activity, or both. Whereas some studies dispute a reduction in spontaneous physical activity[107] and an increase in energy intake,[108-110] other studies provided strong evidence for at least partial compensatory trends with respect to habitual daily activities[111] and caloric consumption.[112] Some interesting patterns emerged from a recent study showing that negative energy balance is compensated for more readily than surfeits and that this compensation is more marked for changes in energy expenditure, i.e. decreases in nonexercise activity, than in energy intake.[113] Thus, in free-living populations, this may be a key factor counteracting the expected weight reduction. Furthermore, a less successful pattern of weight loss in women than in men has been reported in two studies.[95,96] An increase in energy intake may have provoked these results.

With a high rate of restraint eaters,[112,114] dieting women are more likely to allow for a greater food intake when exercise has been performed.[115] However, this notion remains controversial.[110] Some data also lend credence to the idea that abdominal adipose tissue, which is more pronounced in men, is better mobilised than gluteal adipose tissue as, for example, fat oxidation during exercise was only elevated in upper-body obese women when compared with participants with lower body obesity.[94] Nevertheless, there is insufficient evidence to generalise sex differences with respect to a loss in fat mass induced by physical activity.

The implications of the findings of strength training for changes in body composition underline its potential to increase FFM and this requires further evaluation.

In summary, for exercise to be successful at reducing body fat and increasing FFM, a considerable number of regular exercise bouts need to be performed. Regarding long-term effects of exercise on bodyweight and body composition, data are scarce and additional obesity intervention programmes are required. However, considerable controversy remains about the degree of the influence of exercise on RMR. While one study reported an increase in RMR following strength training,[104] most studies reported no changes[95] and a decline in RMR was detected in two intervention groups.[34,104] Thus, although exercise has the ability to increase FFM, an increase in RMR does not necessarily result.

3. Combined Diet and Exercise Intervention Studies

Inducing negative energy balance is the most important aim of weight-loss programmes. Physical activity in conjunction with dietary energy restriction has been promoted as an important component of successful weight-loss regimens. The results of many recent studies support the beneficial role of a combination of the two.

3.1 Dietary Energy Restriction and Aerobic Exercise

Over the past few decades, the focus of activity programmes for obesity treatment has been on aerobic exercise and the potential of this exercise type to improve outcomes has been repeatedly evaluated in diet-exercise intervention studies (table III).

As physical activity has been shown to promote favourable changes in body composition, the concept tested in some studies was as to whether exercise training may be a substitute for dietary restriction. Addressing this question, Frey-Hewitt et al.[116] randomly assigned 121 overweight men to 1 year of energy restriction or engagement in walking/jogging when compared with a control group. Although the reduction in bodyweight and fat mass in the dieters was greater ($p < 0.01$) than in the exercise group, exercise training was superior to dieting with respect to the maintenance of FFM. In the dieting subjects, FFM declined by 1.2kg and both absolute RMR (−149 kcal/day) and RMR per kilogram FFM (−1.68 kcal/kg FFM) decreased ($p < 0.05$). An even more striking result was found in a 3-month study, with a reduction in RMR (−247 kcal/day) after a hypocaloric diet and an increase (202 kcal/day) following jogging on 3–5 days per week. When both groups were evaluated simultaneously, alterations in RMR were related to the changes in FFM. As the loss of fat mass differed significantly between the groups, it was pointed out that exercise alone might not bring about the desired changes in bodyweight.[33] Several investigations have focused on the role of exercise for body compositional changes when added to a hypocaloric diet. In an investigation in 20 obese women, assigned to a very low-energy diet alone or combined with modest exercise sessions, a greater reduction of fat mass ($p < 0.05$) was observed following the exercise treatment, whereas declines in

Table III. Diet and exercise intervention studies

Study	Dietary restriction	Exercise	Subjects	BMI (kg/m^2)	Duration (wk)	Δ Bodyweight (%)	Δ Fat mass (%)	Δ FFM (%)	Δ RMR (%)
Frey-Hewitt et al.[116]	D: −300 to −500 kcal/d E: no dietary restriction C: no dietary restriction	D, C: no exercise E: jogged 25 min 3 d/wk to 50 min 5 d/wk	121 M	27.3% BF	52	D: −7.1[a] E: −4.3[b] C: NS[c]	D: −21.6[a] E: −16.3[a] C: NS[b]	D: −1.7[a] E: NS[b] C: NS[bc]	D: −8.1[a] E: NS[b] C: NS[b]
Schwartz et al.[33]	D: 1200 kcal/d E: no dietary restriction	D: no exercise E: walking/jogging 70–85% HRR 40 min 3–5 d/wk	31 M	29.4% BF	13	D: −12.9[a] E: −2.3b	D: −29.8[a] E: −9.5[b]	D: −5.8[a] E: NS[b]	D: −8.1[a] E: +6.7[b]
Kempen et al.[111]	Wk 1–4: formula, 478 kcal/d Wk 5–8: formula + food, 956 kcal/d	D: no exercise DE: aerobic 90 min 3 d/wk	20 F	32.0	8	−9.1%	D: −14.9[a] DE: −20.5[b]	D, DE: −2.8	SMR: −10
Hays et al.[117]	C: no dietary restriction D, DE: ad lib, 18% fat, 63% CHO	C: no exercise D: no exercise DE: aerobic 45 min 4 d/wk	20 F, 14 M	30.9	12	D: −3.6[a] DE: −5.8[a] C: NS[b]	C: NS[b] D: −2.2[a] DE: −3.5[a]	In the thigh: NS	
Utter et al.[18]	C: no dietary restriction E: no dietary restriction D, DE: 1200–1300 kcal/d	C: no exercise D: no exercise DE, E: walking 60–80% HR_{max} 45 min 5 d/wk	91 F	33.0	12	C, E: NS[b] D, DE: −8.8[a]	C, E: NS[b] D, DE: −17.6[a]	NS	
van Aggel-Leijssen et al.[16]	Wk 1–6: formula, 500 kcal/d Wk 7–10: formula + self-selected foods	DE: cycling, walking, aqua jogging 40% $\dot{V}O_{2max}$, 60 min 4 d/wk D: no exercise	40 M	32.3	10	−14.6	−35.9	−3.5	
Brill et al.[17]	1200–1400 kcal/d, <35g fat/d, ad lib high-fibre, low-fat, low-calorie foods	D: no exercise DE1: walking 30 min 5 d/wk DE2: walking 60 min 5 d/wk	56 F	34.0	12	−5.8	−9.4	−3.3	
Okura et al.[118]	Approx. 1130 kcal/d	D: no exercise DW: walking 30 min 7 d/wk DA: aerobic dance 45 min 3 d/wk, EE = 1050 kcal/wk	90 F	29.3	14	D: −10.8[a] DW: −12.8 DA: −7.2[b]	D: −18.7[a] DW: −24.6[b] DA: −29.3[b]	FFM + BFM: D: −5.2[a] DW: −6.2[b] DA: −3.8[b]	

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Table III. Contd

Study	Dietary restriction	Exercise	Subjects	BMI (kg/m^2)	Duration (wk)	Δ Bodyweight (%)	Δ Fat mass (%)	Δ FFM (%)	Δ RMR (%)
Racette et al.[119]	~75% of RMR, compensated for EE of exercise LF, LFE: 60% CHO, 15% fat LC, LCE: 25% CHO, 50% fat	LF, LC: no exercise LFE, LCE: aerobic 60–65% $\dot{V}O_{2max}$ 45 min 3 d/wk	23 F	34.1	12	LC: −11.6[a] LF: −8.6[b] LFE, LCE: 11.3 LF, LC: 8.9	−7.9 LFE, LCE > LF, LC	−2.2	−129 kcal/d
Gornall and Villani[120]	812 kcal/day	D: no exercise DR: resistance 55 min 3 d/wk	20 F	28.8	4	−6.6	−11.2	−2.9	−8.8
Doi et al.[121]	C: −17% of EI S: −17% of EI + PRO	Light resistance 25 min 7 d/wk	17 M	25.9	12	C, S: −5.5	−14.2	C: −2.1 S: NS	S: +8.1 C: NS
Demling and DeSanti[49]	D: 80% of TEE DRC: 80% of TEE + casein DRW: 80% of TEE + whey	D: no exercise DRC: resistance 4 d/wk DRW: resistance 4 d/wk	38 M	27% BF	12	−2.6	D: −10.4[a] DRC: −39.5[b] DRW: −22.5[b]	D: +0.6[a] DRC: +5.1[b] DRW: +2.6[b]	
Rice et al.[122]	−1000 kcal/d, fat <30%	D: no exercise DA: 50–85% HR_{max} 19–60 min 5 d/wk DR: resistance 30 min 3 d/wk	29 M	32.7	16	−12.0	−27.4	D: −7.0[a] DA: NS[b] DR: NS[b]	
Janssen et al.[123]	−1000 kcal/d, fat <30%	D: no exercise DA: aerobic 5 d/wk DR: resistance 3 d/wk	38 F	33.6	16	−11.3	−20.9	D: −4.8 DA, DR: NS	
Geliebter et al.[124]	70% of RMR	D: no exercise DA: cycling 30 min 3 d/wk DR: resistance 60 min 3 d/wk	40 F, 25 M	41.1% BF	8	−9.2	−16.9	D: −4.7[a] DA: −4.0[a] DR: −1.9[b]	−6.9
Bryner et al.[125]	800 kcal/d liquid formula, 40% protein, 49% CHO	D: walking, biking or stair climbing 1h 4 d/wk DR: resistance 3 d/wk	17 F, 3M	35.4	12	D: −19.3[a] DR: −14.7[b]	−31.8	D: −8.0 DR: NS	D: −13.4[a] DR: +3.6[b]

Continued next page

Table III. Contd

Study	Dietary restriction	Exercise	Subjects	BMI (kg/m^2)	Duration (wk)	Δ Bodyweight (%)	Δ Fat mass (%)	Δ FFM (%)	Δ RMR (%)
Marks et al.[126]	C: no dietary restriction D, DA, DR, DAR: ~1237 kcal/d	D: no exercise DA: cycling DR: resistance DAR: both, 30 min 3 d/wk	44 F	29.4	20	C: 2.0[a] D, DA, DR, DAR: 5.2[b]	C: +2.2[a] D, DA, DR, DAR: −13.3[b]	NS	
Wadden et al.[127]	900–925 kcal/d (+ formula) Wk 18–19: + normal foods Wk 20: 1250 kcal/d Wk 22–48: 1500 kcal/d	D: no exercise DA: aerobic (stepping) DR: resistance DAR: both, 20–40 min 3 d/wk	128 F	36.4	48	−15.7	−28.2	−5.2	−2.6
Kraemer et al.[128]	D, DA, DAR: ~1200 kcal/d C: no dietary restriction	C: no exercise D: no exercise DA: aerobic 3 d/wk DAR: aerobic + resistance 30–50 min 3 d/wk	31 F	28.6	12	−8.9	Approx. −23	NS	NS
Svendsen et al.[129]	C: no dietary restriction D and DE: formula 1000 kcal/d	C: no exercise D: no exercise DE: aerobic + resistance 1–1.5h 3 d/wk	118 F	29.7	12	C: NS D, DE: −12.6	C: NS D: −24.3[b] DE: −30.1[a]	C: NS[b] D: −2.6[a] DE: NS[b]	C: NS[a] DE: +11.5[b] D: NS
Schlundt et al.[53]	LF: ad lib, high CHO Lkcal: low-fat, low-calorie	Exercise 5 d/wk	49 M + F	31.8	10–20	LF: −5.3[a] Lkcal: −9.0[b]	LF: −7.8[a] Lkcal: −17.0[b]	−2.5	−5.5

a Significantly different from b and c at $p < 0.05$.

b Significantly different from a and c at $p < 0.05$.

c Significantly different from a and b at $p < 0.05$.

ad lib = *ad libitum*; **approx.** = approximately; **BF** = body fat; **BFM** = bone-free mass; **BMI** = body mass index; **C** = control group; **CHO** = carbohydrate; **D** = diet group; **DA** = diet and aerobic exercise group; **DAR** = group following diet in combination with aerobic and resistance exercise; **DE** = diet and exercise group; **DE1** = diet and exercise group one; **DE2** = diet and exercise group two; **DR** = diet and resistance exercise group; **DRC** = diet and resistance exercise group receiving casein supplement; **DRW** = diet and resistance exercise group receiving whey supplement; **DW** = diet and walking exercise group; **E** = exercise group; **EE** = energy expenditure; **EI** = energy intake; **F** = females; **FFM** = fat-free mass; $\mathbf{HR_{max}}$ = maximum heart rate; **HRR** = heart rate reserve; **LC** = low-carbohydrate diet group; **LCE** = low-carbohydrate diet and exercise group; **LF** = low-fat diet group; **LFE** = low-fat diet and exercise group; **Lkcal** = low-calorie diet group; **M** = males; **NS** = not statistically significant ($p > 0.05$); **PRO** = protein; **RMR** = resting metabolic rate; **S** = diet group receiving protein supplement; **SMR** = sleeping metabolic rate; **TEE** = total energy expenditure; $\mathbf{\dot{V}O_{2max}}$ = maximal oxygen consumption; Δ = change.

FFM and sleeping metabolic rate were similar across groups.[111]

In contrast to the above-mentioned outcomes, other investigators have been unable to confirm an increased fat loss induced by exercise training. In 91 obese women, brisk walking on its own, or as a diet (1200–1500 kcal/day) plus exercise treatment showed no major impact on body mass and fat loss when compared with a diet-only and a control group.[18] These findings were confirmed in a recent investigation by van Aggel-Leijssen et al.[16] following a similar protocol in 40 obese men.

Some recent studies used an experimental approach to delineate the relationship between exercise intensity and duration on bodyweight modification during caloric restriction. Brill et al.[17] failed to show a positive effect of daily walking for 30 or 60 minutes with regard to alterations in body composition over a diet-only intervention in 56 overweight women. However, the results of Okura et al.[118] oppose these findings. Daily walking for 30 minutes was found to be associated with a greater decline in fat mass (−6.6kg) over diet modification alone (−5.0kg). A third group performed aerobic dance. This approach was superior to walking, due to a greater loss of fat mass (−8.0kg) and the preservation of fat- and bone-free mass.

To assess both the effects of macronutrient composition and aerobic exercise on body composition, 23 obese women were randomly assigned to an energy restrictive diet either low in fat or CHO and aerobic exercise or no exercise intervention in a 2 × 2 factorial design. After 12 weeks, the decrease in bodyweight was higher in the low-CHO group and the loss in fat mass tended to be enhanced following exercise training despite food intake being adjusted to the energy costs of the exercise sessions. No protective effect of exercise on RMR was found and RMR values decreased (−129 kcal/day) in all groups to values lower than predicted from reductions in FFM.[119]

In summary, weight loss, and specifically fat loss, may be promoted by aerobic exercise alone and during decreased energy intake, with a potential attenuation of the depletion of FFM being of major benefit. However, engagement in a considerable amount of physical activity of at least moderate intensity is recommended, when exercise is performed as a strategy to lessen body fat. The addition of aerobic exercise during the reducing diet proved to be effective in preserving FFM in some studies. Changes in RMR did not necessarily appear to be attributable to changes in lean tissue mass.

3.2 Dietary Energy Restriction and Resistance Exercise

Recent findings have revealed a new perspective on physical activity as part of obesity intervention. Resistance training shows promise as a means of losing bodyweight, while increasing or maintaining muscle mass. With respect to lean body mass as a major factor influencing RMR,[106] it is important to look closely at the impact of strength training on a successful pattern of weight reduction. A recent study investigating the effect of resistance exercise on weight loss when added to a very-low calorie diet (812 kcal/day) failed to indicate a suppressed decline in FFM and RMR after 4 weeks of intervention.[120] It is likely that 40g of protein per day, provided during severe energy restriction, was not sufficient to limit losses of skeletal muscle mass. Dietary protein is necessary to synthesise skeletal muscle and an increased intake may enhance nitrogen retention and muscle hypertrophy. Therefore protein supplements are frequently used by strength athletes to allow for accumulation and maintenance of lean tissue.[130]

Doi et al.[131] investigated whether the ingestion of a protein supplement could prevent losses of FFM and, as a result, reductions in RMR in 17 overweight men participating in a 12-week weight-reduction programme that consisted of both mild energy re-

striction (−17% of energy intake) and resistance exercise. The findings showed an increase in RMR expressed per kilogram bodyweight following the supplement intake, whereas the increase was not significant ($p = 0.07$) when RMR was adjusted for changes in FFM. Recent research has also demonstrated that the type of protein can markedly affect body compositional changes induced by a hypocaloric diet (80% of total daily energy expenditure) plus resistance training. When a casein protein hydrolysate was used, gains in lean mass were doubled (+4.1 vs +2.0kg) with a greater reduction in fat mass (−7.0 vs −4.2kg) compared with a whey protein hydrolysate.[49] Weight loss was similar across groups (−2.5kg) and in the diet control group, this was only from body fat (−2.5kg) with no change in FFM.

3.3 Dietary Energy Restriction Combined with Aerobic and Resistance Exercise

In the light of potential positive impacts of both aerobic and strength programmes on body composition, some recent data provide insight into whether the effects are comparable. Rice et al.[122] randomly assigned 29 obese men to one of three 16-week treatments, which consisted of a hypocaloric diet alone or in combination with resistance exercise or aerobic exercise, respectively. Whereas the reductions in weight (−12.4kg) and total adipose tissue (−9.7kg) were not significantly different between the three groups, skeletal muscle was only preserved after the exercise training, independent of the mode, compared with the diet-only group (−2.5kg). In women following the same protocol, the effect of exercise on maintaining lean body mass was less clear.[123] In this study, the recorded changes in body composition were comparable between the two exercise groups. In contrast, earlier work by Geliebter et al.[124] demonstrated that only strength training significantly attenuated the loss of lean tissue accompanying an energy-reduced formula diet (70% of RMR) when compared with aerobic exercise in moderately obese subjects. These findings are in agreement with those in a study of 20 subjects by Bryner et al.[125] that incorporated progressive intensive resistance training of high-volume and aerobic exercise of various modalities in conjunction with a very low-calorie diet. However, Geliebter et al.[124] were unable to show a blunting in the decline in RMR associated with a significant preservation of lean tissue following resistance exercise. Bryner et al.[125] showed impressive results with a decrease in lean body mass (−4.1kg) and RMR (−211 kcal/day) in the group performing aerobic exercise, which was not found when strength training was prescribed. These outcomes could be explained by the high protein content of the diet of approximately 80 g/day. Given the above-mentioned studies, convincing data in support of either aerobic or resistance training for weight loss and maintenance of muscle mass are rare, but there was a tendency for a preservation of lean tissue and RMR following strength exercise.

Several studies have examined whether a combination of the two exercise modalities may contribute to a more successful pattern of weight loss. Marks et al.[126] investigated the effect of a hypocaloric diet alone and together with resistance training, cycling or a combination of resistance training and cycling in a 20-week study in 44 overweight, inactive women. Mean reductions in fat mass and bodyweight were comparable in the exercise groups to those achieved by the diet controls and minimal changes were observed in the non-diet control group. No major differences in FFM were detected from baseline and between the groups. Nevertheless, only the combination of diet, resistance training and aerobic exercise significantly lowered percentage body fat (−4.6%). Following a similar study design, Wadden et al.[127] failed to illustrate any positive impact of exercise (strength training alone, aerobic training alone and as a combination) on body composition in 120 obese women when added to dietary restriction.

For participants in the aerobic condition, the reduction in RMR was significantly blunted in week 24, but not at the end of the 48-week study period when compared with the strength condition. These results are in accordance with those of Kraemer et al.,[128] who examined the effects of diet alone and diet combined with endurance exercise or endurance plus heavy-resistance exercise in 31 overweight women. After 12 weeks, similar reductions in body mass (between −6.2 and −7.0kg) were demonstrated across the three treatment conditions, but without changes in FFM and RMR.

However, in another 12-week study in 118 overweight postmenopausal women, the addition of combined aerobic and resistance exercise to an energy-restrictive diet (1000 kcal/day) led to a greater reduction in body fat (−9.6 vs −7.8kg) and a preservation of FFM in the exercising group compared with the diet-alone treatment. Furthermore, an increase in RMR per kilogram bodyweight reached statistical significance in the diet-plus-exercise group when compared with the control group.[129] However, this would not be surprising given that the decrease in bodyweight consisted of fat, not lean mass. From these studies in overweight and obese populations, it seems obvious that exercise training can reduce the loss in FFM during energy restriction. Another study showed that an individualised, more concentrated exercise protocol offers better overall results in terms of physical fitness and motivation to subsequent physical activity (despite similar results in terms of weight reduction).[132]

Only one study has addressed the question as to whether the effectiveness of a physical activity programme with respect to changes in body composition depends on the type of the prescribed, energy-restricted diet. In a 16- to 20-week programme with at least five exercise sessions per week, 60 subjects consuming a low-calorie diet lost significantly more weight, with a greater decline in body fat (−6.6 vs −2.7kg) [and similar reductions in FFM and RMR], than did subjects consuming a low-fat, *ad libitum* CHO diet. However, the authors pointed out that these results were related to a more reduced energy intake in the low-calorie group.[53]

From the above-mentioned studies it becomes clear that a combination of modest caloric restriction and physical training of different modes is preferable over dietary modification alone to induce favourable changes in body composition accompanying weight loss. However, the reduction in bodyweight and the changes in body composition induced by exercise training seem in many cases to be less than expected, and several studies failed to report significant benefits of exercise. The lack of an impact of exercise training on body composition may be the prescribed amount of exercise not being sufficient with respect to the frequency and duration of the exercise bout and from the investigated studies, no clear line of evidence seems to emerge in terms of the minimum amount of exercise required for successful outcomes. In addition, to date no intervention studies in overweight subjects have been done providing insight into the long-term effects of exercise programmes. Nevertheless, several lines of evidence suggest that resistance exercise may be preferable in terms of preserving FFM over aerobic training and this type of exercise should be explored, especially in patients that did not succeed with aerobic exercise programmes. In addition, although training at higher intensities seemed to produce better results, long-term compliance needs to be evaluated and constant monitoring of the exercising patient has to be ensured. With respect to the energy-restricted diet, there seems to be evidence for protein intake as a limiting factor for maintenance and/or augmentation of FFM. Furthermore, although only well controlled studies have been included in this review, compensatory behaviour, such as reductions in non-exercise physical activity and increases in food intake cannot be completely ruled out in free-living subjects. A discrepancy be-

tween self-reported and actual energy intake and self-reported and actual physical activity in obese subjects is well established,[133] with the degree of under-/over-reporting being related to bodyweight. As several studies required the subjects to keep daily diet records for the duration of the study, while regular dietary advice was provided,[121,123,128,134] inaccuracies of dietary histories may also have biased some study outcomes. Moreover, although all the assessed studies had recruited subjects with at least a minor degree of over-fatness, body fat of participants differed between studies. This appears to be important, as a role of the body fat content for changes in body composition induced by nutrition and exercise has been reported. Forbes[135] provided evidence that lean body mass was a function of body fat. Thus, the loss of FFM per unit of weight loss will be higher in those subjects with a lower initial relative body fat content. These differences could confound the results when comparing studies with participants with different body fatness.

Another purpose of this review was to compare intervention studies with respect to changes in RMR. However, only a few studies have examined RMR and these have produced equivocal findings. Despite the well known association between FFM and RMR, an offset in the decline in FFM following weight loss appears not to necessarily parallel a reduction in RMR and vice versa. A possible explanation is that length and intensity of the exercise programme were not sufficient to cause a continued disruption of metabolism or a growth of lean tissue. Metabolically active compounds include FFM and internal organs. Small changes in FFM my not be sufficient to impact RMR. On the other hand, exercise training may influence RMR independent of changes in body composition.[136-138] Increases in protein turnover associated with resistance training regimens may contribute to elevations of metabolic rate.[130] However, the negative energy balance induced by dietary limitation may offset this effect. Therefore, the juxtaposition of the energy restriction and exercise treatment adds a layer of considerable complexity to measuring and understanding the responses recorded in RMR. Furthermore, the time of measuring of RMR relative to the termination of the last exercise bout is important, because long-term excess post-exercise oxygen consumption events may last for up to 36–48 hours.[106,139,140] Hence, there is no clear evidence for the alteration of RMR by exercise. However, dietary restriction results in loss of lean tissue; the findings of several studies have demonstrated that the reduction in RMR can be blunted via physical activity, specifically resistance exercise.[33,120,127]

Apart from changes in body composition, exercise has been shown to increase insulin sensitivity,[122,141-143] cardiovascular fitness[128] and fat oxidation.[16,144,145] In addition, abdominal fat can be reduced independent of significant changes in body composition.[146] Moreover, increases in lifestyle activity may also be promoted[147] and exercise is associated with improvements in mood.[124] Therefore (for a successful study outcome), exercise training appears to be a beneficial if not substantial addition to diet modification.

4. Conclusion

A rapidly growing body of evidence supports the opinion that dietary recommendations beyond the generally advised reduction in fat intake should be considered for weight loss and long-term weight sustenance. Several studies lend credence to the idea that diets high in fibre and with a low glycaemic index are successful at producing and, more importantly, maintaining weight loss, which is due to a reduction in energy intake and hormonal responses associated with a blunted rise in blood glucose. Furthermore, potential benefits of diets high in protein (between 25% and 30% of total energy intake) are emerging, although scientific data with respect to enhanced losses of fat mass and sustained muscle

tissue are still inconclusive. Long-term interventions are required to clarify these issues.

Evaluation of both aerobic and strength training for treatment of overweight indicates that higher intensities may bring about more favourable changes in body composition. However, this may be difficult to realise with respect to low cardiovascular fitness in severely obese subjects. While the findings for resistance exercise show promising outcomes with respect to changes in body composition and maintaining RMR, many studies have methodological limitations and/or results that are somewhat equivocal. It seems, therefore, that further research in this area is required to clarify the most efficacious type of intervention with respect to exercise modality and dietary modification. Furthermore, the relationship between such interventions, body composition and energy balance requires a firm quantitative base from which advice may be given. Regarding the serious problem of poor patient compliance with exercise programmes, physical activity prescriptions, which would require the least effort while still producing the desired reductions in total body fat warrant further evaluation. Recent research, however, indicates that diet-exercise synergistic effects may be significant in inducing negative energy balance. Thus, clearly the macronutrient composition of the diet is important and in this respect the role of dietary protein is of considerable interest. For example, in addition to its relatively high thermogenic effect, some research suggests protein may attenuate the muscle decline, which usually accompanies energy restriction. Therefore, in the light of the recent data, the debate remains open and further well controlled studies are required with respect to the combined effects and the synergies and antagonisms that exist between diet and exercise in relation to producing desired changes in body composition and maintaining elevations of metabolic rate.

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