

Selections from current literature: effects of dieting and exercise on resting metabolic rate and implications for weight management

Josephine Connolly, Theresa Romano and Marisa Patruno

Introduction

The significance of the rising prevalence of obesity for morbidity and associated health care costs is clearly delineated by the United States National Institutes of Health's *Clinical Guidelines on the Identification, Evaluation and Treatment of Overweight and Obesity in Adults*.¹ The guidelines note the following: 55% of the adult population in the United States is overweight or obese, obesity is the second leading cause of preventable death after smoking in the United States, and the total cost to society attributable to obesity-related diseases approaching 100 billion US dollars annually. The expert panel that developed the guidelines, which are available on the World Wide Web (<http://www.nhlbi.nih.gov/nhlbi/>), define overweight as a body mass index (weight, kilograms/[height, meters]²) of 25 to 29.9 and obesity as a body mass index of 30 and above. Many countries use this criterion. The guidelines recommend weight loss to lower blood pressure, to lower high total cholesterol, to raise low levels of HDL and to lower elevated blood glucose. Calorie reduction, increased physical activity and behaviour therapy are recommended as the first-line treatment for obesity, with consideration of pharmacological therapies as a secondary alternative.

Despite years of research, the treatment of obesity continues to revolve around the seemingly simple concept of balancing calorie expenditure with calorie intake. However, the determinants of energy expenditure, specifically resting metabolic rate, are an active area of research with many debatable issues. This review will address the calorie expenditure side of the scale, with the examination of the effect of dieting and exercise on resting metabolic rate. Resting metabolic rate accounts for 60–75% of total energy expenditure in sedentary people.² Therefore, it is a major determinant of energy balance and changes in weight. Factors which decrease

resting metabolic rate would be associated with difficulty maintaining weight or weight loss, or frank weight gain. On the contrary, anything that increases resting metabolic rate would facilitate weight loss and maintenance of weight loss. Caloric restriction is known to produce a short-term reduction in resting metabolic rate. Issues that have not been resolved regarding such a reduction are as follows: is the reduction proportional to the reduction in body size or the degree of energy deficit, is the reduction permanent or self-limiting, and can exercise prevent the reduction? If this reduction is a permanent reduction in resting metabolic rate, and if it is above and beyond what would be predicted by the resulting smaller body size, then weight loss after calorie restriction will be very difficult to maintain. This paper reviews four articles that address these issues, three reports of primary research and one meta-analysis. Concluding remarks follow regarding actions that primary care physicians can take in the assessment and treatment of obesity.

Kraemer WJ, Volek JS, Clark KL *et al.* Physiological adaptations to a weight-loss dietary regimen and exercise programs in women. *J Appl Physiol* 1997; **83**: 270–279.

Summary

This study examined the effects of three interventions (diet; diet and aerobic exercise; diet, aerobic exercise and resistance training) on resting metabolic rate and body composition, as well as other physiological and metabolic parameters which are beyond the scope of this review. In this 12-week study, 31 overweight women (body mass index >27) were matched according to body mass index, and randomly assigned to one of the three treatment groups or the control group.

Measurements were taken at baseline, 6 weeks and 12 weeks in the same phase of each woman's menstrual cycle. RMR was determined by indirect calorimetry after a 10-hour fast. Body composition was measured using standard hydrodensitometry procedures and calculations.

Subjects in all three diet groups attended a weekly nutrition class on weight loss. Subjects kept diet records that were evaluated each week. Corrections were made

Received 13 August 1998; Accepted 19 November 1998.

Department of Family Medicine, University Hospital and Medical Center, SUNY Stony Brook, Stony Brook, New York 11794-8461, USA.

to facilitate a gradual and consistent weight loss of approximately one to two pounds per week. The nutrition intervention included use of a high-fibre, high-carbohydrate supplement. Based on participants' food records, there were no significant differences in nutrient intake among the three diet groups. Intakes approximated 1194 kilocalories per day, 70% from carbohydrate, 15% from protein and less than 15% from fat.

Subjects in the two groups involved in aerobic exercise exercised three times per week at 70–80% of their functional capacity for 30 to 50 minutes. Duration and intensity were progressively increased. Subjects in the aerobic plus resistance training group also completed 11 exercises following heavy resistance training principles three times per week.

The control group showed no change in body composition over the 12-week period. All three intervention groups had a significant decline in body mass at 6 weeks, and again at 12 weeks for an average total weight loss of 6.2 kg in the diet-only group, 6.8 kg for the diet plus aerobic exercise group, and 7.0 kg for the diet, aerobic and resistance training group (standard deviations only presented graphically). By 12 weeks there were also significant decreases in percentage body fat: 5.8, 8.0 and 4.3%, respectively. However, there were no significant differences between groups. There were no significant changes in fat-free mass in any of the groups at any time period. There were also no significant changes in resting metabolic rate (measured in absolute terms or relative to body mass) within groups over time or between groups over time. Linear regression models between resting metabolic rate and fat-free mass were also tested. These models demonstrated a significant relation only in the diet and aerobic exercise group, such that resting metabolic rate increased as fat-free mass increased (at baseline, $y = 442.74 + 23.00x$, $r = 0.89$, $P \leq 0.05$; at week 12, $y = 88.574 + 29.77x$, $r = 0.79$, $P \leq 0.05$).

Comment

The findings regarding no loss of fat-free mass in the diet-only group are surprising, as some degree of obligatory loss of fat-free mass is expected with significant weight loss. The authors state that the high-fibre nature of the participants' diets may have decreased the insulin response to the participants' meals and facilitated lipolytic pathways, thereby sparing fat-free mass from breakdown and oxidation. Although this concept is intriguing, a low insulin response would not be expected in obese, sedentary subjects consuming a diet that is 70% carbohydrate, very low in fat and only moderately high in fibre (28 grammes per day).

The calorie level may be of greater importance in explaining retention of fat-free mass. Much of the work regarding changes in fat-free mass and resting metabolic rate in response to hypocaloric diets have implemented diets containing 800–1200 kilocalories per day. Such low calorie diets result in a severe calorie deficit and

the need to oxidize protein. Information regarding the participants' dietary intake in this study is scant. Only mean intakes per group for the entire 12-week period are presented. These intakes are approximately 250–380 kilocalories less than mean baseline resting metabolic rates. In addition, dietary information is based on self-report, and there is a strong likelihood of underreporting of food intake in obese people.³ Systematic errors in this direction would lessen the actual calorie deficit. These relatively small calorie deficits may have enabled subjects to spare protein from oxidation. This rather limited attention and control of dietary intake in general in this area of research is a likely factor contributing to the inconsistency in reported results. Not only is the degree of calorie deficit important, but the distribution of macronutrients and amount of protein per kilogram body weight or fat-free mass is also of great importance in determining fuel substrate utilization. The calorie deficit, macronutrient distribution and rate of weight loss may be key factors in the retention of fat-free mass and resting metabolic rate. Dietary information should be prescribed and described on an individual basis, i.e. kilocalories or grammes of protein per kilogram body weight, rather than by group means, as in this study. Although there may have been enough carbohydrate calories to spare protein from oxidation, there may have been insufficient total grammes of protein per kilogram body mass to facilitate an increase in fat-free mass, despite the appropriate stimulus in the resistance training group. Since all subjects were able to retain fat-free mass, it follows that their resting metabolic rates would also be stable.

Ballor DL, Harvey-Berino JR, Ades PA *et al.* Decrease in fat oxidation following a meal in weight-reduced individuals: a possible mechanism for weight recidivism. *Metabolism* 1996; **45(2)**: 174–178.

Ballor DL, Harvey-Berino JR, Ades PA *et al.* Contrasting effects of resistance and aerobic training on body composition and metabolism after diet-induced weight loss. *Metabolism* 1996; **45(2)**: 179–183.

Summary

This two-part study is based on the assumption that a decrease in calorie intake and weight loss is associated with a decrease in resting metabolic rate and fat oxidation. The purposes of the study are as follows: (i) to determine if exercise training facilitates the maintenance of weight loss by attenuating the reductions in resting metabolic rate, resting fat oxidation and/or fat oxidation in the 5-hour post-prandial period; and (ii) to prospectively compare the maintenance of weight loss and changes in body composition and resting metabolic rate among subjects participating in aerobic or resistance training. All testing was done while subjects resided at a university clinical research centre.

In the first study, 20 older subjects (aged 56–70 years) underwent an 11-week weight-loss program. Subjects were educated on self-selecting diets meeting the following criteria: 900–1100 kilocalories per day, 60% carbohydrate, 15% protein and 25% fat. Subjects kept food diaries which were reviewed by a registered dietitian at weekly meetings. During the twelfth week, subjects were requested to increase their intake to allow for weight maintenance and stabilization of weight for post-diet measurements.

Subjects lost a significant amount of weight, approximately 9% of baseline weight (95.2 ± 3.2 kg), a significant amount of fat-free mass, approximately 5% of baseline (52.6 ± 2.7 kg), and a significant amount of fat mass, approximately 15% of baseline (42.6 ± 1.8 kg). In addition to losing mass, the following significant changes from baseline measurements were reported in the post-diet assessment period: a 15% decrease in absolute resting metabolic rate (1789.8 ± 80.2 kcal/24 hours, baseline), and an 8% decrease in resting metabolic rate relative to fat-free mass (33.8 ± 0.7 kcal/24 hours/kg fat-free mass, baseline).

In the second study, 18 of the 20 weight-reduced subjects began a 12-week exercise regimen, consisting of either aerobic training or weight training. Subjects followed progressive training programmes to elicit 80% of one-repetition maximum strength for weight trainers, and a minimum of 50% of peak oxygen uptake for 60 minutes for aerobic trainers. All subjects attended supervised exercise sessions three times per week.

After the 12-week training period following the initial diet intervention, the weight-training group did not experience further weight loss, but maintained the weight lost during the initial 11-week diet period. Nor did they experience any significant changes in fat-free mass or fat mass, although the trend for increased fat-free mass approached significance ($P = 0.071$). The aerobic trainers experienced a significant further decrease in weight (2.5 ± 0.6 kg) and a significant further decrease in fat mass (1.8 ± 0.8 kg), and their fat-free mass remained unchanged.

In addition, there were between-group differences in body composition such that the aerobic trainers lost weight and the resistance trainers' weight remained unchanged. Trends in fat-free mass were also significantly different in that the weight trainers experienced a trend toward increasing fat-free mass and the aerobic trainers experienced no change in fat-free mass. Although changes in absolute resting metabolic rate in the weight training group approached significance ($P = 0.068$), there were no significant changes in resting metabolic rate, absolute or per kilogram mass, within the two groups or between the two groups.

Comments

In the first part of the study, subjects' resting metabolic rate decreased to a greater extent than their weight or

fat-free mass. This excessive reduction is most likely attributable to the degree of calorie restriction, and therefore cannot be completely explained by the reduction in fat-free mass. Wadden and colleagues have concluded that short-term changes in resting metabolic rate are best predicted by baseline resting metabolic rate and degree of calorie restriction, whereas long-term changes in resting metabolic rate are best predicted by baseline resting metabolic rate and fat-free mass.⁴ Therefore, during and immediately after a hypocaloric period, resting metabolic rate is likely to be suppressed.

It is not clear how soon after the initial study participants began the second study, or what their dietary intake was during this time. The mean weights at the start of the second study are 2 kilograms less than at the end of the first study, so it is reasonable to believe that these subjects continued to consume a hypocaloric diet. As in the first study, diets were not prescribed individually or controlled for adequately in the data analyses. Therefore, it is difficult to assess the degree of calorie and protein restriction, and the effect these variables may have on the initial reduction in metabolic rate and subsequent maintenance of it. According to the description of recommended dietary intake during the first phase of the study, protein intakes may have been as low as 0.43 grammes per kilogram body weight. This level of restriction may partially explain why fat-free mass and resting metabolic rate did not increase in the resistance training group.

The researchers of this study have concluded that attenuating the reductions in resting metabolic rate and increasing fat oxidation rates after weight loss are not the mechanisms by which exercise prevents weight recidivism. However, until dietary factors are controlled for, these types of conclusions are premature.

Lastly, a third non-exercise group in the post-diet period would have strengthened the study. It would have been interesting to compare the resting metabolic rates and fat oxidation rates of weight-reduced exercisers versus non-exercisers.

Gornall J, Villani, RG. Short-term changes in body composition and metabolism with severe dieting and resistance exercise. *Int J Sport Nutr* 1996; **6**: 285–294.

Summary

The authors sought to examine the potential of strength training as a means to prevent the decline in fat-free mass and resting metabolic rate associated with very-low calorie diets. They randomly placed 22 female subjects in one of two groups, a diet-only group and a diet plus strength training group. Subjects were matched on body surface area. In addition, the authors controlled for two other factors: fluctuations in metabolic rate due to hormonal changes and losses in total body water. Women were tested at approximately the same time of

the month in their menstrual cycle. Body composition was analysed using a dual X-ray absorptiometry technique which is sensitive to changes in fat-free mass associated with fluctuations in water, minerals and protein.⁵ The contribution of total body water changes to changes in fat-free mass were calculated based on total body water measurements using bioelectrical impedance analysis. The treatment period was 4 weeks long, during which time subjects consumed 800 kilocalories per day. Sixty per cent of the calories were from carbohydrate, 20% from fat and 20% from protein. All pre-packaged meals were provided to subjects free of charge. Post-intervention tests were completed while participants were still on the very-low-calorie diet. They met with the research staff two times per week for support and weigh-ins. Those in the diet-plus-exercise group also participated in supervised strength training activities three times per week. They completed three sets of 10 free weight exercises each training session, and resistance was progressively increased. Post-intervention testing was conducted 2 days after the last exercise session. An analysis of variance with repeated measures revealed a significant time effect, such that those in the diet-only group and the diet plus strength training group experienced a significant decrease in kilograms body mass (74.44 ± 2.78 to 69.36 ± 2.78 , 78.51 ± 2.74 to 73.54 ± 2.30 , respectively), kilograms fat-free mass (32.77 ± 2.27 to 28.73 ± 2.31 , 34.72 ± 1.87 to 31.19 ± 1.61 , respectively), kilograms total body water (34.67 ± 0.94 to 33.32 ± 1.11 , 36.35 ± 0.93 to 34.50 ± 0.87 , respectively) and absolute resting metabolic rate in kJ/hour (273.1 ± 6.7 to 240.14 ± 7.38 , 286.9 ± 12.2 to 271.1 ± 12.4 , respectively). There were no significant group differences, indicating that strength training did not attenuate the reduction in resting metabolic rate or fat-free mass. In addition, an analysis of changes in absolute resting metabolic rate, controlling for fat-free mass as a covariate, again reveals a significant decrease in resting metabolic rate with no statistically significant differences between groups. In other words, for both groups there is a significant loss in absolute resting metabolic rates above and beyond what can be explained by loss of fat-free mass. The authors conclude that resistance training cannot reverse the negative effects of severe energy restriction on resting metabolic rate or fat-free mass. In addition, the authors conclude that the majority of fat-free mass lost could be accounted for by loss of body water. Since carbohydrate is stored in the muscle with water, the loss in body water is expected due to glycogen depletion associated with the hypocaloric diet. Strength training draws largely on locally stored glycogen for energy substrate, and can therefore further decrease the glycogen and water component of fat-free mass. The authors note that the short-term decrease in resting metabolic rate may be due to a decrease in sympathetic tone associated with a diet-induced decrease in circulating insulin levels.

Comment

Dietary factors are addressed in this study in that all meals were provided to patients. Patients were consuming approximately 0.5 grams of protein per kilogram body weight, which is again inadequate for a response to the strength training stimulus. Resting metabolic rate was measured while subjects were on the hypocaloric diets, and therefore is reflective of the stress of dieting itself and not simply of the loss of fat-free mass. The authors calculate that all of the loss in fat-free mass can be attributed to water losses. However, it should be noted that this is likely to be an oversimplification, and measurement errors are probably masking the loss of actual protein or muscle mass. Therefore, if water losses are not accounted for, the relationship between fat-free mass and resting metabolic rate may not be accurately and completely described.

Thompson JL, Manore MM, Thomas JR. Effects of diet and diet-plus-exercise programs on resting metabolic rate: a meta-analysis. *Int J Sport Nutr* 1996; **6**: 41–61.

Summary

It is difficult to summarize the results of studies examining the effect of exercise on resting metabolic rate during a hypocaloric dieting period due to the number of variables that are involved (type, duration, frequency and intensity of exercise, degree of energy deficit, total daily calorie intake, and distribution of calories among carbohydrates, proteins and fats). Therefore, Thompson and colleagues suggest caution regarding narrative reviews of this body of literature. Rather, they have conducted a meta-analysis to quantify treatment effectiveness, specifically the effects of diet alone and diet-plus-exercise on resting metabolic rate.

The authors searched the literature and found 22 studies between 1984 and 1995 that documented resting metabolic rate in humans placed in either diet-only groups or diet-plus-exercise groups. The studies represent data from 631 subjects, 68 males and 563 females, 31–45 years of age. The majority of studies placed subjects on low-fat, high-carbohydrate diets of less than 1200 kilocalories per day. Most subjects were involved in aerobic exercise for 31 to 60 minutes, 4 or 5 days per week at an intensity of about 50–70% of maximum aerobic capacity. Intervention programmes lasted approximately 10 weeks. Effect sizes for differences in resting metabolic rate before and after diet and before and after diet-plus-exercise were calculated. Positive effect sizes indicate that resting metabolic rate increased due to the intervention, and negative effect sizes indicate that resting metabolic rate decreased as a result of the intervention.

The authors calculate effect size in absolute terms (kJ/hr) and relative terms (kJ/kg fat-free mass). When expressed in absolute terms, there was a significant

decrease in resting metabolic rate in diet only (283.4 ± 32.2 to 253.5 ± 34.5) and diet-plus-exercise groups (279.6 ± 36.4 to 255.5 ± 34.2). However, the drop is classified as small for the dieters who exercised and large for those who just dieted. This difference is also statistically significant. Similarly, when expressed per kilogram of fat-free mass per hour, the drops in resting metabolic rate for the dieters (5.17 ± 0.55 to 4.82 ± 0.62) and dieter-exercisers (5.24 ± 0.60 to 4.93 ± 0.65) were significantly greater than zero. The decrease in the dieters is classified as moderate, while the decrease with dieter-exercisers is considered small. The difference between groups is not significantly different. The authors were also able to establish that neither diet-related variables (number of calories, distribution of calories among macronutrients or duration of diet), exercise-related variables (type of exercise, intensity, duration or frequency) nor subject-related variables (age, gender, body composition) correlated significantly with changes in resting metabolic rate. There has been some attention given in the literature to the appropriateness of calculating relative metabolic rate by dividing resting metabolic rate by body weight or fat-free mass, since the line defining the relationship between these two variables does not intercept the y-axis at zero.⁶ An alternative method of examining a relative relationship is to plot resting metabolic rate as a function of mass and perform a linear regression to determine whether the slope and intercept of this relation change as a function of treatment. The results of this manipulation do reveal a decrease in resting metabolic rate due to diet alone and diet-plus-exercise. However, the slopes of the regression lines pre- and post-treatment are not significantly different, and, therefore, the relationship between mass and metabolic rate is the same and independent of treatment. In other words, the drop in resting metabolic rate is expected due to the decrease in body size. The authors conclude that resting metabolic rate does decrease significantly in response to a diet of less than 1000 kilocalories per day, and that the addition of moderate intensity aerobic exercise (50–70% maximum aerobic capacity) performed for 31 to 60 minutes, 4 or 5 days per week, decreases this response, but does not return resting metabolic rate to baseline.

Comment

The use of meta-analysis in this area of research is useful because it allows for a systematic examination of the many variables involved. It is, of course, limited by the range of studies available. Only seven studies looked at combining aerobic exercise with strength training, none looked at strength training alone and no studies looked at high-intensity aerobic exercise (over 70% maximum aerobic capacity). Half of the studies examined exercise conducted 4 or 5 days per week, 18% for three or fewer times per week and 32% at 6 or 7 days per week. Fifty-four per cent had subjects consume 800 kilocalories

per day, 27% had subjects consume 800 to 1200 kilocalories per day, and only three studies or 14% looked at the consumption of greater than 1200 kilocalories per day. In addition, the calorie level is rarely adjusted according to individual needs; therefore the actual calorie deficit per individual is an important confounding variable.

Concluding remarks

Based on the above reviews, we can revisit the controversial issues delineated in the introduction of this paper, and apply these issues to a family physician's practice. One of the main points to be made is the potential impact of dietary intake, especially total calories, calorie deficits and grammes of protein per kilogram body weight. Further work is necessary to determine whether milder calorie deficits with adequate protein in combination with strength training can positively affect resting metabolic rate.

In contrast to Kraemer and colleagues' work, the majority of the studies point to a reduction in short-term resting metabolic rates that is greater than can be explained by the loss of body mass or fat-free mass over the same time period. Unfortunately, there has been very little work done over the last few years regarding the duration of this phenomenon. Wadden and colleagues' work indicates that this disproportionate reduction reflects metabolic processes associated with the hypocaloric dieting itself. When calorie balance is resumed, the resting metabolic rate is dependent on the new body mass, especially fat-free mass.⁴ This is relevant for motivated patients who adhere to severe hypocaloric diets to achieve rather large weight losses. When they get to goal weight their metabolic rate is severely depressed, and they can experience almost immediate weight gain if they resume their prior higher calorie intakes. Recent studies have not continued to measure changes in resting metabolic rate for extended periods to determine whether the reductions are self-limiting. Again, the work of Wadden and colleagues supports a self-limiting hypothesis.⁴ More research is needed on this critical issue. Lastly, exercise does not appear to negate this reduction in resting metabolic rate or fat-free mass. This may have been due to insufficient calories, protein or exercise stimulus in terms of frequency.

Family practice physicians can facilitate healthy and successful weight management among their patient populations by heeding the following tips: (i) determine long-term weight goals based on obtaining a body mass index under 27, if possible 25; (ii) determine short-term weight goals based on a reduction of 1 to 2 body mass index units (approximately 4.5–7.3 kilograms or 10–16 pounds); (iii) encourage patients to maintain this interim goal for 6 months to allow for readjustment of resting metabolic rate for new body mass (as well

as for psychosocial adjustments and commitment to necessary behaviour changes); and (iv) determine the next short-term weight goal. Continue to support patient through this cyclical process until body mass index is at least under 27, if not at 25.⁷ The physician will also need to explore appropriate nutrition and exercise education and counselling options with the patients, based on the patient's resources, education level and learning style. Based on patients' medical history and preferences, appropriate individualized diet and exercise prescriptions should be developed. This is best approached with a health care team including a physician, registered dietitian and exercise physiologist. Through this slow and thoughtful process of cycles of weight loss and weight maintenance it is thought that patients will be able to prevent the more debilitating cycles of rapid weight loss, short-term reductions in metabolic rate and rapid weight gain.

References

- ¹ National Heart Lung and Blood Institute. *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults*. Bethesda: National Institutes of Health, 1998.
- ² Apfelbaum MJ, Bestsarron J, Lacatis D. Effect of caloric restriction and excessive caloric intake on energy expenditure. *Am J Clin Nutr* 1971; **24**: 1405–1409.
- ³ Lansky D, Brownell KD. Estimates of food quantity and calories: errors in self-reporting. *Am J Clin Nutr* 1982; **35**: 727–732.
- ⁴ Wadden T, Foster GD, Letizia KA, Mullen JL. Long-term effects of dieting on resting metabolic rate in obese outpatients. *JAMA* 1990; **264**(6): 707–711.
- ⁵ Forbes G. *Human Body Composition*. New York: Springer-Verlag, 1987.
- ⁶ Ravussin E, Bogardus C. Relation of genetics, age, and physical fitness to daily energy expenditure and fuel utilization. *Am J Clin Nutr* 1989; **49**: 968–975.
- ⁷ American Dietetic Association. Position of the American Dietetic Association on weight management. *J Am Dietetic Assoc* 1997; **97**(1): 71–74.