Exercise and obesity

Tracy J. Horton* and James O. Hill

Center for Human Nutrition, Department of Pediatrics, University of Colorado Health Services Center, Denver, Colorado 80231, USA

Obesity is a condition of excess body fat. While genetic factors can influence the body fatness of an individual, environmental factors, promoting a high energy intake and low energy expenditure (EE), appear to be playing an important role in the development of obesity in many countries (Danforth, 1985; Brownell & Wadden, 1992; Van Itallie, 1996). A low level of physical activity can contribute to a low total EE and is thought to promote positive energy balance and weight gain (Flatt, 1987; James, 1995). This is supported by cross-sectional data which show that individuals and groups characterized by a low level of physical activity have a greater body weight and body fat content compared with their more active counterparts (Schulz & Schoeller, 1994; Roberts, 1995; Ryan et al. 1996). A low activity level, however, could be a consequence rather than a cause of obesity. Nevertheless, longitudinal data also suggest that a low level of physical activity is associated with an increased risk of weight gain (Roberts et al. 1988; Griffiths et al. 1990; Williamson et al. 1993). Thus, regular physical activity appears to be important in helping maintain a normal body weight and body composition.

Both metabolic and behavioural factors play a role in the development of obesity (Hill et al. 1994). The relative contributions of these factors to weight gain probably vary between individuals. Both food intake and physical activity have behavioural and metabolic components. With respect to physical activity, the behaviour relates to the quantity and type of physical activity an individual chooses to engage in, whereas the metabolic factors relate to the amount of energy expended, the pattern of fuels utilized and the hormone and metabolite changes resulting from the exercise. As yet, we have a poor understanding of how the metabolic effects of physical activity affect exercise and/or food intake behaviour. However, the metabolic response to exercise is more clearly understood. The present paper, therefore, will review the metabolic effects of exercise, specifically with respect to energy and fat metabolism, and how this may relate to the long-term control of body weight.

Energy expenditure and its components

Total daily EE (24 h EE) = resting metabolic rate (RMR) + energy cost of activity + thermic effect of food (TEF)

The major component of 24 h EE is the RMR. This generally accounts for approximately 60–75% of total expenditure (Danforth, 1985). TEF contributes about 10% to 24 h EE, while the contribution of arousal is probably very small. The energy cost of activity can be the most variable component of 24 h EE both from day to day and between individuals. Although the obvious consequence of planned exercise is an increase in the contribution of the energy cost of activity to daily EE, exercise may also affect other components of 24 h EE, e.g. RMR. Exercise may also have important effects on nutrient balance and, in particular, fat balance.

The effect of an acute bout of exercise on energy expenditure

The net energy cost of exercise is calculated as the increase in exercise EE above resting level. In athletes, the net energy cost of exercise can contribute as much as 3360–16 800kJ to 24 h EE (Brouns et al. 1989; Schulz et al. 1992; Horton et al. 1994). This requires moderate- to high-intensity activity sustained for 75 min to many hours. However, most individuals do not have the ability, the time or the desire to be so active. For the average person who wishes to incorporate exercise into their daily routine, the net energy cost of exercise is likely to be much lower. For moderate exercise of moderate duration, a net EE of 756–1890kJ can be predicted (Table 1).

In addition to the net energy cost of exercise during the activity itself, EE remains elevated for a certain time period post-exercise, i.e. the excess post-exercise O2 consumption (EPOC; Gaesser & Brooks, 1984). This is calculated as the elevation in O2 consumption, above resting level, during the post-exercise period. Much research has focused on this phenomenon as it has been suggested that EPOC could significantly increase daily EE and so help with weight loss and the prevention of weight gain (Cannon & Einzig, 1984). With aerobic exercise, it has been shown that the degree and duration of EPOC are dependent on the intensity and duration of the exercise (Bahr et al. 1987; Gore & Withers, 1990; Quinn et al. 1994). However, EPOC is unlikely to significantly increase 24 h EE for the average individual performing moderate exercise (Freedman-Aka-
Table 1. Energy cost of an acute moderate exercise bout

<table>
<thead>
<tr>
<th>Body weight (kg)</th>
<th>60</th>
<th>80</th>
<th>100</th>
</tr>
</thead>
<tbody>
<tr>
<td>Estimated RMR* (kJ/min)</td>
<td>4.2</td>
<td>5.6</td>
<td>7.0</td>
</tr>
<tr>
<td>Net EE (kJ)</td>
<td>756</td>
<td>1008</td>
<td>1260</td>
</tr>
<tr>
<td>EPOC† (kJ)</td>
<td>113</td>
<td>151</td>
<td>189</td>
</tr>
<tr>
<td>Potentiation of TEF‡ (kJ)</td>
<td>63</td>
<td>63</td>
<td>63</td>
</tr>
<tr>
<td>Total energy (kJ)</td>
<td>932</td>
<td>1222</td>
<td>1512</td>
</tr>
<tr>
<td>Exercise at 10.0 MET for 30 min</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Net EE (kJ)</td>
<td>1008</td>
<td>1512</td>
<td>1890</td>
</tr>
<tr>
<td>EPOC† (kJ)</td>
<td>151</td>
<td>227</td>
<td>284</td>
</tr>
<tr>
<td>Potentiation of TEF‡ (kJ)</td>
<td>63</td>
<td>63</td>
<td>63</td>
</tr>
<tr>
<td>Total energy (kJ)</td>
<td>1222</td>
<td>1802</td>
<td>2237</td>
</tr>
</tbody>
</table>

RMR, resting metabolic rate; 1 MET, O₂ consumption at rest; EE, energy expenditure; EPOC, excess post-exercise O₂ consumption; TEF, thermic effect of food.

*Ainsworth et al. (1993).
† Estimated as 20% of the TEF from 3150 kJ meal (Segal et al. 1985).
‡ Estimated as 15% of net exercise EE (Bahr et al. 1987).

Bas et al. 1985; Maresh et al. 1992). Following resistance-type exercise, or high-intensity exercise, it has been suggested that the magnitude of EPOC is substantial (Bahr et al. 1992; Melby et al. 1993). However, the intensity and duration of exercise used in these studies is probably impractical for the average individual. Resistance exercise may produce a greater EPOC than aerobic exercise (Gillette et al. 1994), although it is difficult to equate similar exercise bouts for these two types of activity. Thus, the increase in 24 h EE that is due to EPOC in the average individual is likely to be small and to have minimal impact on weight loss or weight maintenance.

An interaction between exercise and TEF has also been pursued to determine if there is potentiation of TEF when preceded or followed by exercise. Results are equivocal, with some authors reporting an enhancement of TEF (Segal & Gutin, 1983; Young et al. 1986) and others finding no effect (Dallosso & James, 1984; Schutz et al. 1987). This lack of consistency is partly due to the poor reproducibility of both measures of TEF (Weststrate, 1993) and EPOC (Commerford et al. 1997). However, quantitatively, if exercise does potentiate TEF, the increase in 24 h EE resulting is probably very small (Segal et al. 1985).

Table 1 gives an estimate of the total EE that may result from a single bout of daily exercise (net exercise EE + EPOC + potentiation of TEF). For individuals who are active for health reasons and/or to maintain or lose weight, the additional energy that may be expended over 1 d due to planned exercise probably ranges from 932 to 2237 kJ/d depending on body weight and the duration and intensity of the moderate exercise.

Chronic exercise training and energy expenditure

As RMR constitutes a large proportion of 24 h EE, an increase in RMR could increase daily EE. The major determinant of RMR is fat-free mass (FFM; Ravussin et al. 1986). Habitual exercise, therefore, could mediate an increase in RMR indirectly through an increase in FFM. Whether regular activity also has a direct stimulatory effect on RMR, i.e. increasing metabolic rate per unit FFM, is controversial.

Longitudinal studies

Most studies report that following a period of exercise training, in previously sedentary individuals, RMR is not significantly elevated (Poehlman et al. 1986; Bingham et al. 1989) unless there is a concurrent increase in FFM (Broder et al. 1992; Campbell et al. 1994). Thus, if FFM is increased by exercise training, RMR can be increased. In this regard, resistance training has been shown to be more effective at increasing FFM (Ballor et al. 1988; Pratley et al. 1994; van Etten et al. 1997) compared with aerobic exercise (Bingham et al. 1989; Broder et al. 1992), although significant increases in FFM have been observed with prolonged endurance training (Westerterp et al. 1994). The probable magnitude of this increase in RMR through changes in FFM is, however, small (approximately 5% of initial RMR) (Broder et al. 1992).

Cross-sectional studies

Where trained individuals have a greater FFM relative to sedentary individuals, they not only have a higher RMR (Gilbert et al. 1991) but also a greater non-training 24 h EE (Horton & Geissler, 1994). Certain studies also report that trained subjects have a greater RMR, even relative to FFM, compared with their untrained counterparts (Tremblay et al. 1985; Poehlman et al. 1989). This suggests an increase in the metabolic activity of the FFM, at least for some time period, as a reduction in training leads to a decline in RMR (Tremblay et al. 1988; Herring et al. 1992). To explain the observation of a higher RMR per unit FFM in trained individuals, it has been suggested that the high state of energy flux (high EE and high energy intake) characteristic of trained subjects is required for an elevation in RMR to be observed (Goran et al. 1994; Bullough et al. 1995). However, this state of high energy flux appears to require an amount and intensity of exercise that is not practical for the average individual (> 5000 kJ/d). Consequently, for most individuals, the main effects of chronic exercise training on RMR are through any changes in FFM.

Does habitual exercise significantly increase daily energy expenditure?

Daily EE under free-living conditions can be measured using the doubly-labelled-water technique. Even in individuals who would not be considered trained athletes, 24 h EE is increased in relation to the amount of total active leisure time (Livingstone et al. 1991; Haggarty et al. 1994). With the introduction of an exercise training programme, significant increases in 24 h EE have been reported (Blaak et al. 1992; Westerterp et al. 1992; Van Etten et al. 1997). Goran & Poehlman (1992) observed no increase in 24 h EE following training in elderly subjects due to a decline in non-training activity. This emphasizes the need to maintain other spontaneous activity when commencing an exercise...
programme for weight control, otherwise the beneficial effect of exercise on 24 h EE may be negated.

**Effect of exercise on fat oxidation**

During exercise, energy is mainly supplied from the utilization of carbohydrate and lipid fuels, with a much smaller contribution from protein (Gollnick, 1985; Hood & Terjung, 1990). Energy production during sustained aerobic exercise is mainly derived from the oxidative metabolism of fat and carbohydrate. With high-intensity, intermittent exercise, and resistance exercise, there can be a large contribution of anaerobic metabolism to energy production which utilizes mainly carbohydrate (Saltin, 1973).

During aerobic exercise, as intensity increases, the contribution of fat to energy production decreases (Brooks & Mercier, 1994). Once exercise stops, fat oxidation is increased during the post-exercise period (Quinn et al. 1994; Horton et al. 1995). In addition, increased fat oxidation at rest has been observed on the day following aerobic exercise (Weststrate et al. 1990; Calles-Escandon et al. 1996). Little information is available on daily rates of fat oxidation with aerobic exercise, although Goldberg et al. (1990) observed no difference in 24 h RQ with three different levels of aerobic activity. Daily measurements of nutrient oxidation by indirect calorimetry need to be viewed critically and should be excluded from longer-term measurements (e.g. 24 h). However, well beyond the time of resistance or high-intensity exercise, reports are inconsistent as to whether fat oxidation is increased (Melby et al. 1993) or unchanged (Broeder et al. 1992; Treuth et al. 1996).

**Chronic exercise training effects on fuel utilization**

Adaptations in skeletal muscle following aerobic exercise training contribute to the increased ability to utilize lipid as a fuel during exercise (Hurley et al. 1986; Kiens et al. 1993). These changes include an increase in the proportion of oxidative muscle fibres, an increase in the muscle mitochondrial content and an increase in the activity of various enzymes involved in oxidative metabolism (Holloszy & Coyle, 1984). Aerobic exercise training also leads to an increased sensitivity of adipose tissue to the lipolytic action of catecholamines (Depres et al. 1984). This increases the ability to mobilize lipid. Resistance exercise and high-intensity exercise training tend to increase the number of glycolytic muscle fibres (Saltin, 1973) which may be less favourable to lipid oxidation. It could be argued, therefore, that aerobic training, rather than resistance and/or high-intensity exercise training, may be best for creating a metabolic environment geared towards fat utilization and decreasing fat mass.

Even at rest, increased fat oxidation has been observed following a period of aerobic exercise training (Poehlman & Danforth, 1991; Poehlman et al. 1994) and in trained compared with untrained individuals (Tremblay et al. 1992; Romijn et al. 1993). Part of this effect of chronic training on resting fat oxidation may be due to the previous exercise bout (Calles-Escandon & Driscoll, 1994). Resistance exercise training does not appear to increase fat oxidation at rest (Ryan et al. 1995; Toth & Poehlman, 1995). Treuth et al. (1995) reported a lower 24 h and resting RQ on a non-exercise day following a strength training programme, but subjects were also in greater negative energy balance during the post-training measurement, which would also lower RQ. Consequently, the effects of resistance exercise training on chronic fat oxidation need further clarification.

**The impact of exercise alone on weight loss**

Although exercise can increase 24 h EE in individuals for whom the goal is weight loss, the energy deficit created by exercise alone (1148–2475 kJ/d) is small relative to the

---

Table 2. Absolute amount of fat oxidized by a 65 kg recreationally-active individual with a maximum oxygen uptake of 40 ml/kg body weight per min exercising for 40 min at either 50% or 70% of their maximum oxygen uptake

<table>
<thead>
<tr>
<th>% Maximum O2 uptake</th>
<th>Total energy expended (kJ)</th>
<th>% Energy from fat oxidation</th>
<th>Fat oxidized</th>
</tr>
</thead>
<tbody>
<tr>
<td>50</td>
<td>1092</td>
<td>50</td>
<td>546</td>
</tr>
<tr>
<td>70</td>
<td>1530</td>
<td>30</td>
<td>459</td>
</tr>
<tr>
<td>Difference</td>
<td>438</td>
<td>87</td>
<td>2-6</td>
</tr>
</tbody>
</table>

The exact effects of resistance or very-high-intensity intermittent exercise on nutrient oxidation during and post-exercise is difficult to determine accurately. This is due to the anaerobic contribution to energy production during these types of activities, leading to inaccuracies in the indirect calorimetry calculations of nutrient oxidation (Brooks & Fahey, 1984). During exercise, non-metabolic CO2 production can occur leading to RQ of > 1.0 (Broeder et al. 1992). In the post-exercise period, there is CO2 retention as the bicarbonate pool is replenished and this can produce RQ < 0.7. Observations on short-term nutrient oxidation with these types of exercise, therefore, need to be viewed critically and should be excluded from longer-term measurements (e.g. 24 h). However, well beyond the time of resistance or high-intensity exercise, reports are inconsistent as to whether fat oxidation is increased (Melby et al. 1993) or unchanged (Broeder et al. 1992; Treuth et al. 1996).
amount of body energy they need to lose (approximately 31130 kJ/kg fat tissue). Even in theory, therefore, exercise alone would be expected to produce a very slow rate of weight loss. Not surprisingly, the effectiveness of exercise (aerobic and/or resistance) at decreasing body weight has been shown to be limited, with the magnitude of weight loss averaging 0.08–0.12 kg/week in men and women (Epstein & Wing, 1980; Garrow & Summerbell, 1995; Wilmore, 1995). Such a slow rate of weight loss is likely to be discouraging for an individual.

In general, exercise does have favourable effects on body composition (Wilmore, 1995). Aerobic exercise can significantly decrease fat mass (Bouchard et al. 1990; Westerterp et al. 1994), whereas resistance exercise appears to be less effective (Ballor et al. 1988; Broeder et al. 1992). This is probably due to the greater promotion of fat oxidation by aerobic v. resistance exercise. As previously discussed, resistance exercise can increase FFM, more so than aerobic activity, but this may lead to an even slower rate of weight loss. Exercise, therefore, can promote maintenance of a lower relative fat mass irrespective of degree of weight loss. However, exercise alone is of limited use as a primary treatment for obesity.

The impact of exercise plus dietary restriction on weight loss

Dietary restriction alone can produce a much greater daily energy deficit than exercise. Much research, therefore, has focused on the effect of dietary restriction with and without exercise on weight loss. The rationale behind this is first, that the exercise may increase the rapidity and degree of weight loss due to the creation of a greater energy deficit. Second, exercise may reduce the decline in FFM that occurs with weight reduction but increase fat loss. The lesser reduction in FFM would help decrease the fall in RMR and diminish the decline in energy requirements that results from a decreased body weight. Finally, exercise may help maintain better dietary compliance.

For weight loss alone, the majority of data suggests that this is not significantly increased when regular exercise is combined with an energy-restricted diet (Bogardus et al. 1984; Van Dale et al. 1987; Ballor et al. 1988; Heymsfield et al. 1989; Svendsen et al. 1993). Exercise, however, does have favourable effects on the composition of the weight that is lost. Garrow & Summerbell (1995), in a meta-analysis of diet and diet plus exercise studies, reported that for a given weight loss, active men and women on a low-energy diet had a lesser decrease in FFM than their sedentary counterparts (41 and 23 % less respectively). Aerobic exercise also increases fat loss relative to diet alone (Heymsfield et al. 1989; Racette et al. 1995), whereas resistance exercise, in combination with dietary restriction, better maintains FFM (Ballor & Keesy, 1991; Ryan et al. 1995). Although in theory it may be predicted that a combination of aerobic and resistance exercise would lead to better maintenance of FFM during dietary restriction and greater loss of body fat, this has not generally been found (Donnelly et al. 1991; Sweeney et al. 1993). Where a greater maintenance of FFM occurs with diet plus exercise compared with diet alone, the decline in RMR is reduced in proportion to the change in FFM (Kiern et al. 1990; Ryan et al. 1995).

Exercise may also improve adherence to an energy-restricted diet. Racette et al. (1995) reported that subjects who participated in a diet plus exercise programme lost more weight than the diet-only group due to a greater energy deficit resulting from both the daily exercise and better dietary compliance. In conclusion, therefore, exercise has beneficial effects on weight loss with dietary restriction mainly due to favourable effects on body composition changes and it may also improve dietary compliance. However, exercise appears not to increase the extent or rapidity of weight reduction.

Effect of exercise on weight maintenance after weight reduction

One of the major issues in the treatment of obesity is the long-term maintenance of weight loss. Successful weight reduction is not that difficult to achieve, but it is well established that weight regain occurs in most individuals (NIH, Technology Assessment Conference Panel, 1992). In a review of studies addressing long-term weight maintenance, Pronk & Wing (1994) reported that individuals who were most successful at maintaining weight loss had incorporated regular exercise into their lifestyle. This was observed despite a large variation in the types of populations studied, and diet and exercise interventions used. Exercise, therefore, appears to be important in the post-obese individual for prevention of weight regain.

How does exercise enable a more appropriate regulation of energy balance?

Habitual, planned exercise has been shown to play a role in the maintenance of an appropriate body weight and body composition and to be beneficial in weight loss. Most of the present review has focused on how this may be mediated by the metabolic effects of physical activity, specifically in relation to EE and fat oxidation. However, the metabolic effects of exercise may also affect behaviour. With respect to food intake, physical activity appears to facilitate appropriate regulation of energy balance. Classically, this was shown by Mayer et al. (1954), who demonstrated that when animals exercised below or above a certain level they inappropriately adjusted their food intake to match EE such that weight gain and weight loss occurred respectively. In addition, there may be metabolic effects of exercise that determine subsequent exercise behaviour and why certain individuals are more inclined to exercise than others. In the future, elucidation of the link between exercise metabolism and behaviour may help in the design of exercise programmes that offer the most benefit with respect to the control of food intake and maintenance of an active lifestyle. This may then help in the long-term control of body weight and energy balance.
References


