INTRODUCTION

Reduced physical activity represents one of the most significant changes in lifestyle that has been observed during the twentieth century. Our sedentary lifestyle and the reduced energy requirements of the majority of our jobs has been a source of comfort in a business world where efficiency and productivity are sought. The impact of the transition from a traditional to a modern lifestyle on daily energy needs can be estimated by various means. By using the doubly labelled water technique and indirect calorimetry, Singh et al. (1) showed that the energy cost of living at the peak labor season was as high as $2.35 \times$ resting metabolic rate (RMR) in Gambian women. When this value is compared to results usually obtained in women living in industrialized countries, 1.4 to 1.8 $\times$ RMR (2,3), it can be estimated that for a given body weight, a modern lifestyle may have reduced the energy cost of living by as much as 1 to 4 MJ/day. Accordingly, a recent analysis by Prentice and Jebb (4) has emphasised the contribution of sedentariness to the increased prevalence of overweight in the United Kingdom.

Despite these observations, the contribution of exercise to the prevention and treatment of obesity is still perceived as trivial by many health professionals. The perception of many of them was recently well summarized by Garrow (5) who stated that exercise is a remarkably ineffective means of achieving weight loss in obese people, mainly because their exercise tolerance is so low that the level of physical activity that they can sustain makes a negligible contribution to total energy expenditure. When one looks at the currently available literature, it is difficult to disagree with this statement. Indeed, numerous studies have demonstrated that when exercise is used alone to treat obesity, body weight loss is generally small (6). In addition, the further weight loss generated by adding an exercise program to a reduced-calorie diet is also often small if not insignificant (7).

Traditionally, the study of the impact of exercise on body weight control has focused on its energy cost and on the hope that the body energy loss will be equivalent to the cumulative energy cost of exercise sessions. In practical terms, this means for instance that if a physical activity program induces an excess of energy expenditure of 2000 kcal/week, a similar energy deficit should be expected in the active obese individual. Recent experimental data show that such a view is not realistic since it does not take into account the compensations in other components of energy balance which may either attenuate or amplify the impact of exercise on body energy stores. It thus appears preferable to consider exercise as a stimulus affecting regulatory processes which can ultimately affect all the components of energy balance instead of only focusing on its energy cost. The objective of this chapter is to
EXERCISE AND MACRONUTRIENT BALANCE

The maintenance of body weight stability depends on one’s ability to match energy intake to expenditure. This principle is one of the most accepted axioms of science and represents the main guideline for health professionals treating obesity. However, even if energy balance is a central issue in body weight control, it does not necessarily imply that matching energy intake to expenditure is the primary target of mechanisms involved in the regulation of body energy stores.

Flatt (8) reported convincing evidence showing that energy balance is linked to macronutrient balance. His research and that of other scientists have also clearly established that the regulation of the balance of each macronutrient is not performed with the same precision. Of particular interest for obesity research is the fact that fat balance is the component of the macronutrient balance that is the most prone to large variations. This is probably explained by some of the following factors:

- The weak potential of dietary fat to promote a short-term increase in its oxidation (9–11).
- The weak potential of high fat foods to favor satiety without overfeeding (12–15).
- The inhibiting effect of the intake of other energy substrates on fat oxidation (16,17).
- The absence of a metabolic pathway other than lipogenesis to buffer a significant fraction of an excess fat input (excess dietary fat intake and/or fat synthesized from other substrates).
- The greater dependence of fat oxidation on sympathoadrenal stimulation (18).

The fact that fat balance appears as the ‘Achilles tendon’ of the macronutrient balance system is probably compatible with the importance of maintaining body homeostasis. Indeed, it is probably less toxic and damaging for the body to store a large amount of triglycerides as opposed to an equicaloric storage of alcohol and glycogen. However, in the long run, a large body accumulation of fat causes metabolic complications which worsen health status. For the exercise physiologist, the question raised by this argument is whether the exercise stimulus can facilitate the regulation of fat balance, i.e. can favor fat balance without relying on body fat gain to promote macronutrient balance.

REGULATION OF FAT BALANCE: FAT GAIN OR EXERCISE?

Many years ago, Kennedy (19) proposed a lipostatic theory stipulating that variables related to adipose tissue contribute to the long-term control of food intake. Accordingly, studies performed under different experimental conditions provided evidence suggesting that fat cell size (20), plasma glycerol (21), fat cell lipolysis (22), and fat oxidation (23) may be related to fat and energy balance and to the long-term stability of body weight. More recently, the discovery of leptin (24) represented an important step in the investigation of the role of adipose tissue on the regulation of fat and energy balance. As shown in Table 12.1, leptin exerts many functions and its most documented role has been to favor a negative energy balance or at least to promote the stabilization of body weight in a context of overfeeding by reducing food intake (25). This table also indicates that variations in plasma insulin without changes in glycemia produce effects which are similar to those of leptin. Since the clearance of insulin is modulated by the hepatic exposure to free fatty acid (FFA) flux (26), which itself partly depends on fat cell size, it is reasonable to associate changes in adiposity with the effects of changes in insulinemia on fat and energy balance.

To summarize, these observations demonstrate that adipose tissue is not passive when one experiences long-term underfeeding or overfeeding. It rather behaves like an organ actively involved in the

<table>
<thead>
<tr>
<th>Variables</th>
<th>Leptin</th>
<th>Insulin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy intake</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Energy expenditure</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Activity level</td>
<td>↑</td>
<td>?</td>
</tr>
<tr>
<td>Neuropeptide Y</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Sympathetic nervous system activity</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>
Table 12.2  Opposite (A) and concordant (B) effects of physical activity and metabolic cardiovascular syndrome related to fat gain

<table>
<thead>
<tr>
<th>Physical activity effect</th>
<th>Variables</th>
<th>Metabolic cardiovascular syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Blood pressure</td>
<td>↑</td>
</tr>
<tr>
<td></td>
<td>Plasma glucose</td>
<td>↑</td>
</tr>
<tr>
<td></td>
<td>Plasma insulin</td>
<td>↑</td>
</tr>
<tr>
<td></td>
<td>Plasma triacylglycerols</td>
<td>↑</td>
</tr>
<tr>
<td></td>
<td>Plasma total cholesterol</td>
<td>↑</td>
</tr>
<tr>
<td></td>
<td>Plasma HDL cholesterol</td>
<td>↓</td>
</tr>
<tr>
<td></td>
<td>Plasma apoB*</td>
<td>↑</td>
</tr>
<tr>
<td></td>
<td>Plasma cholesterol: HDL cholesterol</td>
<td>↑</td>
</tr>
<tr>
<td></td>
<td>LDL particle size*</td>
<td>↓</td>
</tr>
<tr>
<td>B</td>
<td>SNS activity</td>
<td>↑</td>
</tr>
<tr>
<td></td>
<td>Energy expenditure</td>
<td>↑</td>
</tr>
<tr>
<td></td>
<td>Fat oxidation</td>
<td>↑</td>
</tr>
</tbody>
</table>

*Additional atherogenic features of the metabolic cardiovascular syndrome (31).
HDL, high density lipoprotein; LDL, low density lipoprotein; SNS, sympathetic nervous system; apoB, apolipoprotein B.

recovery of fat and energy balance and of body weight stability.

Research conducted over the last decades has shown that exercise can also affect many of the above referenced variables. It has been demonstrated that exercise stimulates adipose tissue lipolysis and that trained individuals are more sensitive to the lipolytic effects of catecholamines (27,28). Furthermore, Turcotte et al. (29) reported that for any given plasma FFA concentration, trained individuals would utilize more fat during exercise than their untrained controls. With respect to leptinemia, recent data tend to show that for a given level of body fat, trained individuals display reduced plasma leptin levels compared to sedentary controls (30).

We can therefore suggest from the above observations that both fat gain and exercise represent strategies which may contribute to the regulation of fat and energy balance. However, these results also indicate that physically active individuals have a major advantage over sedentary individuals as they may regulate their fat balance more efficiently, i.e. with less substrate gradient and reduced hormone concentrations. In other words, trained persons are expected to rely to a lesser extent on variations in adiposity to maintain fat balance under free-living conditions. The main corollary of this phenomenon is depicted in Table 12.2, which reminds us there is also, unfortunately, a price to be paid in taking advantage of the regulatory impact of fat gain on fat and energy metabolism. Indeed, body fat gain, particularly in the visceral fat compartment, is associated with an increase in blood pressure and plasma glucose and insulin as well as with an atherogenic dyslipidemic plasma profile (32,33). This cluster of atherogenic and diabetogenic metabolic abnormalities is seldom formed among non-obese physically active individuals.

**EXERCISE, FAT BALANCE AND BODY WEIGHT CONTROL**

The evidence summarized above suggests that the exercise-trained individual can maintain a reduced level of adiposity because of an increased sensitivity and overall better performance of mechanisms involved in the regulation of fat balance. If this beneficial adaptation can be reproduced in the obese individual undertaking a physical activity program, this response would favor a metabolic context facilitating body weight loss. Accordingly, recent data demonstrate that the effects of exercise favorably influence components of fat and energy balance.

**Exercise and Fat Oxidation**

Exercise-trained individuals are characterized by an increased level of fat oxidation despite the fact that their adiposity is generally lower than that of untrained subjects (34–37). In the post-exercise state, the increase in fat oxidation is explained by an increase in resting metabolic rate and/or by an increased relative fat content of the substrate mix oxidized. Moreover, evidence suggests that the
enhanced fat oxidation characterizing trained individuals is at least partly explained by acute effects of exercise (38–40).

The mechanisms underlying the exercise-induced increase in fat oxidation are not clearly established but experimental data suggest that it is related to an increase in sympathetic nervous system activity (35) that seems to be mediated by beta adrenoreceptors (36). Other recent data emphasize the possibility that the impact of exercise on fat utilization is mainly determined by a change in glycogen stores and/or glucose availability (41,42). This observation is concordant with our recent finding that when exercise is immediately followed by a liquid supplementation compensating for carbohydrate and lipid oxidized during exercise, essentially no change in post-exercise fat oxidation is found (43).

For the obese individual who displays limitations in the ability to perform prolonged vigorous exercise, the above findings open new therapeutic perspectives. For instance, they raise the possibility that combining exercise and food-related sympathomimetic agents could produce a substantial increase in fat oxidation. One of these agents is capsaicin, which was recently found to significantly increase fat oxidation in the postprandial state (44).

In another recent study, we examined the impact of combining exercise and ad libitum intake of low fat foods on daily energy balance in heavy men (51). These subjects were tested twice in a respiratory chamber under either a sedentary condition with ad libitum intake of a mixed diet or an exercise condition with a low fat diet. As expected, daily energy balance was considerably reduced (1.6 MJ) under the latter condition. This finding and the evidence summarized above suggest that it is of primary importance to take into account diet composition to optimize the daily energy deficit which can be achieved with exercise.

Recent studies have been designed to test the hypothesis that exercise per se can modify macronutrient preferences. This has been examined by Verger et al. (52) who reported an increased preference for carbohydrate after prolonged exercise. In a subsequent study, these authors did not reproduce this finding but rather noted an increased preference for proteins after prolonged exercise (53). Another recent study performed in our laboratory revealed that vigorous exercise in untrained subjects did not selectively modify the preference for any macronutrient (54). On the other hand, Westerterp-Plantenga et al. (55) obtained results demonstrating

### Table 12.3
Energy intake, expenditure and balance over 2 days under high or low fat conditions following a moderate intensity exercise session

<table>
<thead>
<tr>
<th>Variables</th>
<th>Post-exercise period</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low fat diet</td>
</tr>
<tr>
<td>Energy intake (MJ)</td>
<td>25.7 ± 3.3</td>
</tr>
<tr>
<td>Energy expenditure (MJ)</td>
<td>29.9 ± 7.3</td>
</tr>
<tr>
<td>Energy balance (MJ)</td>
<td>-4.2</td>
</tr>
</tbody>
</table>

Adapted from Tremblay et al. (49).

---

**Exercise and Fat Intake**

Excess dietary fat is known to affect spontaneous energy intake considerably. In humans tested under conditions mimicking free-living conditions, the intake of high fat foods is associated with a large increase in daily energy intake (12–15). This is concordant with studies demonstrating a significant positive relationship between habitual dietary fat intake and adiposity (15,46–48). When the enhancing effect of a high fat diet on energy intake is considered in the context of exercise practice, high fat feeding is expected to inhibit the impact of exercise on energy balance. As shown in Table 12.3, we found that when subjects have free access to high fat foods after having performed a 60-minute vigorous exercise, they overfeed to a level that does not permit exercise to induce a negative energy balance (49). In contrast, a substantial energy deficit is achieved when exercise is followed by free access to low fat foods. This is in agreement with other recently reported data showing that high fat feeding favors an increase in the post-exercise compensation in energy intake (50).

In another recent study, we examined the impact of combining exercise and ad libitum intake of low fat foods on daily energy balance in heavy men (51). These subjects were tested twice in a respiratory chamber under either a sedentary condition with ad libitum intake of a mixed diet or an exercise condition with a low fat diet. As expected, daily energy balance was considerably reduced (1.6 MJ) under the latter condition. This finding and the evidence summarized above suggest that it is of primary importance to take into account diet composition to optimize the daily energy deficit which can be achieved with exercise.
that exercise may increase the preference for carbohydrates.

In summary, diet composition seems to be an important determinant of the potential of exercise to induce an overall negative energy balance. However, it remains uncertain whether a change in macronutrient preferences can be spontaneously driven by exercise or should be the result of a voluntary change in food selection.

**CLINICAL IMPLICATIONS**

The literature summarized above suggests that combining exercise and a reduced dietary fat intake should favor spontaneous body weight loss in obese individuals. In obese women, this combination was found to induce a mean decrease in body weight of 16% that was associated with a normalization of the metabolic risk profile (7). In a more recent study, we used the exercise–low fat diet combination as a follow-up of a treatment of obesity consisting of drug therapy and low calorie diet (56). In this context, exercise and low fat diet accentuated the fat loss induced by the first phase of treatment up to a mean cumulative weight loss of 14% and 10% of initial values in men and women, respectively. In addition, the exercise–low fat diet follow-up was again associated with a normalization of the metabolic risk profile. As shown in Table 12.4, these observations are consistent with a recent study demonstrating that the regular physical activity and adherence to a low fat dietary regimen are the main features of the lifestyle of ex-obese individuals maintaining a large weight loss on a long-term basis (57).

Even if the combination of exercise and low fat diet can induce a considerable body energy deficit under free-living conditions, it is likely that adipose tissue-related regulatory factors of energy and fat balance will over time favor the restabilization of body weight. These factors, which are associated with resistance to further loss of weight in the reduced-obese individual, are probably the same ones that promote the achievement of a new body weight plateau in the context of overfeeding. Thus, as discussed above, the decrease in sympathetic nervous system activity and in plasma FFA, leptin, and insulin probably contributes to resistance to losing more fat after having experienced success with exercise and a low fat diet. In this context of increased vulnerability towards a fattening lifestyle, the ex-obese person obviously must maintain his/her new exercise–low fat diet lifestyle to prevent further weight regain.

**CONCLUSIONS**

The combination of exercise and a low fat diet is an effective way to induce a spontaneous negative energy and fat balance. In the context of a weight-reducing program, this represents a strategy that focuses on lifestyle changes instead of directly targeting caloric restriction. The amount of body fat loss expected under these conditions probably corresponds to what the body does not need anymore to regulate macronutrient balance. This model considers adipose tissue as an active organ whose impact on energy balance can be at least partly replaced by a healthy lifestyle characterized by healthy food habits and regular exercise.

**REFERENCES**

717–726.


