Physiological aspects of exercise in weight cycling

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Introduction

One of the main problems in the treatment of obesity is the maintenance of a reduced body weight after the original weight loss. The desire to be thin has resulted in an enormous number of methods of weight control and subjects are very willing to try another method. However the long-term results are disappointing.

Sjöström (1) reviews this gloomy situation by stating that the relapsing patient can be identified in advance with high precision by predicting that everyone will relapse. Therefore repeated weight loss and regain (weight cycling) is becoming a field of interest especially with respect to the long-term effects on health. Recent epidemiological data from the Zutphen Study show that weight instability is positively related with a higher risk of coronary heart disease. Subjects with a high body mass index (BMI) and a high individual variability (SD > P30) in BMI during a period of 10 y had a 2.5-fold higher risk than those with a normal BMI and a low individual SD in BMI in the follow-up period of 15 y (D Kromhout, personal communication). Therefore more information about the physiological aspects of weight cycling is needed.

It is suggested that exercise plays a special role in weight control. It is one of the few factors positively correlated with successful long-term body weight maintenance (2). This review will focus on the role of daily physical activity as a mechanism involved in regulating energy balance especially in relation to repeated cycles of weight loss and regain.

Physical activity and the regulation of energy intake

The problem of weight cycling is a problem of keeping energy exchange in balance. It is plausible to suggest that increasing weight and inactivity may form a vicious circle.

In 1954 Mayer (3) had already suggested that the regulation of food intake was linked with the level of physical activity in rats. At sedentary levels energy intake was higher than at higher levels of daily energy expenditure, suggesting that regulation of the energy balance is not optimal at lower activity levels. This hypothesis was supported by a study of workers in a jute mill in West Bengal (4). However, the study had serious defects in both design and analysis (5).

Reviewing the literature on aerobic exercise and weight, Epstein and Wing (6) concluded that there is a reliable and directly related effect of exercise on weight loss. Thin persons exhibit a better energy balance than heavy persons. This was confirmed in a long-term study on obese women (7). Increasing exercise increases food intake but not sufficiently to compensate completely.

There is a large variability in daily energy intake and expenditure indicating that energy balance is best studied in individuals rather than groups. A study in British military cadets (8) where intake and expenditure were carefully measured does not show any sign of a relation between energy intake and expenditure on a daily basis. The results did show an interesting time lag of 4 d between intake and expenditure. However, this was not confirmed in a balance study in another group of cadets (9). On the basis of 69 subjects from six balance studies, Durnin (10) concluded that the results are not supportive of a precise short-term control mechanism. Perhaps the difficulties in measuring habitual daily activities and food intake over a period of time obscure an existing mechanism. If Mayer’s hypothesis is true one should expect a better relationship between energy intake and output over a shorter period of time at higher levels of exercise.

In the past years we (11) have had the opportunity to measure energy intake and expenditure during one of the world’s most demanding sport events, the Tour de France, over a period of 21 d. Energy expenditure was calculated from basal metabolic rate (BMR) and equations to predict expenditure from cycling speed, weight of cyclist and cycle, altitude, road pavement, etc. Energy intake was based on the food records kept by the athletes and checked weekly. In Figure 1 the results of one cyclist are depicted, showing a day-to-day regulation of food intake. The absolute mean divergence from energy balance over 21 d in five cyclists was only 14 MJ (3300 kcal) from a total energy expenditure of 558 MJ (132.8 Mcal). There was a close correspondence of daily intake and expenditure in all subjects with significant individual correlation coefficients all > r = 0.85. Analy-

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sis of moving averages revealed the best correlation value when calculated over a 3-d period. Although the number of subjects was small in this study, it nevertheless provides evidence that food intake is better regulated at higher levels of physical activity.

A possible route by which physical activity can lead to a better regulation of food intake is the observed change to higher carbohydrate (CHO) intake at the expense of fat in physically active subjects. In a nation-wide study in athletes in The Netherlands, information about food intake was systematically obtained in different sport groups of high national or international level. The actual mean intake CHO varied from 41 to 63 relative contribution (En%).

In Figure 2 the mean En% CHO and energy intake of groups of athletes in both sexes participating in the endurance-type sports, like cycling, running, swimming, and triathlon, are shown. With increasing energy expenditure the relative intake of CHO increases. Relative intake of protein remains constant (12–15 En%). Furthermore the figure shows that even in the sport groups with a relative low energy intake percentage CHO is higher than the CHO intake level of the Dutch population in general.

Why is this observation of interest in relation to the problem of relapse in obesity? There is increasing evidence that the CHO-fat ratio in the diet is of importance in relation to obesity (12). The conversion from dietary CHO to fat by de novo synthesis costs 23% of the original energy from CHO whereas for dietary fatty acids the cost of deposition as triglyceride is only 3%. Therefore the thermic effect of dietary fat is less than that of CHO. So high-fat diets may contribute to obesity through a lower diet-induced thermogenesis (DIT) as well as higher energy density.

The link between energy intake and energy balance appears to be much tighter for CHO and protein whereas the fat balance is directly influenced by fat intake (13). It is suggested that improvement of the CHO-fat intake ratio is therefore one of the factors that can prevent weight gain (14). To achieve a maximal level of performance, adaptation to a modified nutrient intake is necessary. The need for an enhanced intake of CHO is especially recognized in athletes (15). Although maximal performance is not the ultimate goal of the exercise therapy in obesity, the improvement of the CHO-fat intake ratio may be of benefit in the prevention of weight gain.

Exercise and the energy stores
One of the effects of physical training is a decrease in body fat and increase in fat-free mass (FFM). The relative changes in both variables have direct implications for metabolic rate. The magnitude of weight loss and change in body composition is proportional to the frequency and intensity of the exercise (6). However even with an intensive exercise program weight loss is relatively small (0.2 kg/wk) compared with energy intake restriction.

In addition, the individual response is highly variable. Björntorp et al (16) demonstrated that this difference in response has to do with the differences in the degree of
filling of the adipose tissue. Exercise may benefit individuals with enlarged fat cells but have less effect on those with excess number of fat cells. After emptying the fat cell to a certain size, regular physical exercise does produce weight stability.

This observation also partly explains the differences in outcome in the treatment of obese subjects with exercise in addition to energy restriction. In a review of the studies with a low-energy diet (D) or in combination with an exercise program (DE), it was found that there is no clear indication for better results in the DE groups with respect to weight loss and fat loss than in groups where diet only was prescribed (17). It was interesting that all studies with a significantly positive effect of exercise included male subjects.

In males a much greater fat loss is observed especially in individuals with larger adipocytes whereas in female this is not the case (18, 19). These observations indicate that the initial level of fatness is an important determinant of fat loss in men but not in women.

Differences in distribution of adipose tissue may be the key to this different response. Male subjects are characterized by a storage capacity in the abdominal region. This fat-cell type is more sensitive for release and storage of triglyceride. Leibel et al (20) found a higher number of beta receptors in abdominal fat cells that are more responsive to exercise. In women storage capacity is located especially in the femoral region where alpha receptors are predominant. In a study with obese men and women, physical training had more effect on body composition in the male type of adipose tissue distribution than in the female distribution (19). Therefore in a discussion about weight cycling these differences in response to exercise must be considered.

If weight gain accompanying relapse from energy restriction leads to higher levels of body fatness compared with the prediet situation, weight cycling can lead to a vicious circle where in fact the end result is a progressive increase in body fatness. During short-term energy restriction the loss of FFM is high compared with fat loss. Garrow (21) suggested an optimal FFM-fat loss ratio of 25:75 but often this ratio is much higher especially in the so-called crash diets often seen with athletes (15). One can speculate about this process during weight cycling. A positive energy balance can lead to a negative change in the ratio of FFM to fat mass gain. In theory physical exercise can prevent such deterioration by stimulating extra FFM gain.

In the Minnesota starvation study (22) the percentage body fat turned out to be increased after the refeeding period and the volunteers had attained base-line body weight again. Gray (23) suggested that especially in lean and/or male subjects relative fat mass gain could be greater. There are only a few animal studies available and no convincing evidence that weight cycling does initiate such a vicious circle (23–26). It was concluded that age, sex, and state of obesity interfered with the outcome of the study. Perhaps the fact that animals are not inactive can explain this negative finding.

Besides the relative increase in fat mass, the redistribution is also a point of concern. Although no data are yet available, migration from the femoral to the abdominal region is a possibility on basis of the characteristics of both fat depots. The results from the epidemiological data tending to a higher health risk for weight instability do suggest that this is the case. The role of exercise in this process is uncertain. If exercise would increase the energy drain both from abdominal and femoral sites and if in
the weight-gain phase fat is preferentially stored in the abdomen, exercise will contribute to this shift.

Exercise and metabolism

As a result of physical training several metabolic adaptations occur that can be considered advantageous in relation to obesity.

Training does increase sympathetic nervous activity not only during exercise but also during resting conditions. A situation is created where target organs are more sensitive. For instance, lipolytic response of adipose tissue to catecholamines is increased by training (16). Another important adaptation is the increase in insulin sensitivity. Insulin is considered one of the key hormones in regulation of the substrate flow to muscle and adipose tissue (27).

Insulin resistance, related to obesity, results both in diminished glucose oxidation and decreased glucose storage (ie, nonoxidative glucose disposal) in the muscle. This will lead to a blunting of the facultative thermogenic response to feeding. Newsholme et al (27) suggest that insulin resistance and decreased thermogenesis may be explained by a common mechanism: a decreased sensitivity of skeletal muscle and perhaps other tissues to catecholamines or a decreased activity of the sympathetic nervous system (SNS). This view supports the idea that the muscle compartment is of importance in relation to obesity.

Physical training is one of the most potent stimulators to increase glucose uptake and insulin sensitivity in the muscle, probably mediated by increased SNS activity. The emptying of the muscle CHO store (glycogen) during exercise is the logical explanation for this adaptation in muscle metabolism. At the same time physical training facilitates mobilization of free fatty acids (FFA) from the adipose tissue and a higher capacity to oxidize FFA in the muscle cell (28). Lipoproteinlipase (LPL) activity also increases with increasing level of physical training (29). As LPL can be considered as the gatekeeper enzyme for fat storage, it looks that exercise increases the capacity for delivery and storage of energy in the adipose tissue.

Therefore considering the integrated organism, it seems that physical training does lead to higher capacity to facilitate and to store energy both in muscle and adipose tissue as well as to increase in muscle the capacity to oxidize CHO, fat, and even protein to produce labor (16). In fact one can say that exercise produces a metabolic status where the body is more adapted to keep energy balance.

Exercise and thermogenesis

Recently Brownell (30) suggested that in athletes this ability to keep to a better energy balance leads to an increased food efficiency in order to save energy as a protective response to exercise. Looking for such an analogy in obese weight cyclers means the acceptance of an optimal thermogenic response instead of searching for factors leading to a defective thermogenesis.

Resting metabolic rate (RMR) accounts for ~75% of the daily energy expenditure in both lean and obese subjects. Recently Ravussin et al (31) demonstrated in a prospective study that preobese subjects with a lower-than-normal metabolic rate have a fourfold higher risk to gain 7.5 kg in a 2-y follow-up period. To validate this idea Garrow and Webster (32) compared RMR per unit FFM in subjects where body fat ranged from 6% to 60%. However, no differences were found. In a study with obese weight cyclers, no significant differences were found in sleeping metabolic rate (SMR) per kg FFM compared with obese individuals who do not repeatedly lose and regain weight (33). Therefore the controversy about a diminished RMR as an expression of genetic metabolic efficiency in weight gain still exists.

During energy restriction RMR drops 15–30%. These changes in metabolic rate are partly related to changes in FFM. However not all the decrease is attributable to a change in FFM; other factors, such as decrease in protein turnover, lead to a lower level of metabolic rate per kg FFM.

It is of interest to analyze whether weight cyclers adapt more quickly to a shortage in energy. In Figure 3 the weight change per week is shown for the previously mentioned weight cycling study (33). No difference in weight loss is found between those who do and do not repeatedly lose and regain weight. There was a significant decrease in SMR per kilogram FFM in the energy restricted group (D) compared with both groups with energy restriction.
and exercise (DE). Loss of body fat was significantly greater in the DE groups. Weight cyclers did not respond differently. This study also confirmed the observation as found in several other studies that during weight loss exercise can in part prevent the decrease in RMR per unit FFM (13.8% instead of 17.1% decrease) (34–36). The suggested return of RMR to predicting levels, however, is unlikely (37).

The literature is very divided on the effect of exercise on RMR in obese and lean subjects. It is suggested that there is a carry-over effect of the exercise of the previous days on RMR. A carefully controlled study in a respiration chamber showed such an effect (+4.7%) in an athletic population (38). In obese and lean inactive subjects no carry-over effect was found (39–43). The increased capacity for gluconeogenesis and storage of CHO in the athletic group can explain these differences. It demonstrates the thermogenic advantages of a better training status.

Two studies (44, 45) suggested that postobese subjects have enhanced metabolic efficiency (24% and 15% lower metabolic rates, respectively). The postobese and comparable lean subjects in one of these two studies followed subsequently different exercise regimes. However RMR was not affected. More than 90% of the difference in metabolic rate occurred during the day, indicating a reduced thermogenesis. Assuming a difference in diet-induced thermogenesis (DIT) response between obese and lean subjects of 8.7% and 14.8%, respectively, as was found by Jequier (46), 65% of the differences in metabolic rate can be explained. This called our attention to study whether this diminished response in DIT also occurred in weight cyclers and whether exercise can potentiate DIT. Miller et al. (47) suggested this hypothesis as a possibility for why extra energy is expended during periods of exercise. It is noteworthy that only two studies analyzed the extra effect of exercise on DIT in a situation of overfeeding as suggested by Miller (41, 48). However no positive effect was found in either study. It is difficult to explain the differences in outcome of all studies as reviewed by Horton (49). It is remarkable that all whole-body calorimetry studies did not reveal an extra effect. Another explanation is the observed relationship between DIT and aerobic capacity. Physically trained subjects showed a diminished DIT that is related to increased lipid oxidation (50, 51). This sparing effect of carbohydrates can be considered as another example of the adaptation of the body to exercise. Davis et al. (52) found a positive correlation between DIT and aerobic power (AP). However on the basis of their AP values, these subjects can be considered as nontrained. Therefore it appears that the described enhanced metabolic efficiency is related to highly trained subjects.

Comparing athletes with weight cyclers in relation to energy economy may be correct with respect to adipose tissue metabolism. However only the increased efficiency in muscle metabolism can explain the observed metabolic efficiency in athletes. It would be surprising to expect in weight cyclers a similar effect. Only physical exercise of considerable intensity will result in improved efficiency.

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