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Energy expenditure, physical activity and body-weight control

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Regular physical exercise and endurance training are associated with low body weight and low body fat mass. The relationship between exercise and body-weight control is complex and incompletely understood. Regular exercise may decrease energy balance through an increase in energy expenditure or an increase in fat oxidation. It may also contribute to weight loss by modulating nutrient intake. An intriguing question that remains unresolved is whether changes in nutrient intake or body composition secondarily affect spontaneous physical activity. If this were the case, physical activity would represent a major adaptative mechanism for body-weight control.

Energy expenditure: Physical activity: Body-weight control

Body-weight regulation

Maintenance of constant body weight and body composition requires that two conditions be met. First, an even energy balance must be attained, i.e. energy expenditure must on average be equal to energy intake. This state is found in weight-stable individuals when assessed over periods ranging from several days to several weeks, although large day-to-day variations are observed. Second, there must be an even balance for each individual substrate, i.e. protein, carbohydrate and fat oxidation must be equal to protein, carbohydrate and fat intakes respectively. If this state were not present, body composition would inevitably change, even during isoenergetic feeding (Jéquier, 1993).

If energy intake continuously exceeds energy expenditure, the excess energy ingested has to be deposited, thus increasing the body nutrient stores. There is clearly a 'hierarchy' in substrate oxidation during overfeeding. Any increase in protein intake will rapidly lead to stimulation of protein oxidation, restoring a steady protein balance. The same is true for carbohydrates, the oxidation of which increases over 1–3 d to match any increase in carbohydrate intake (Elwyn & Bursztein, 1993). The result of this hierarchy is that excess energy intake leads essentially to fat storage, mainly in subcutaneous and visceral adipose tissue. In contrast, a period of hypoenergetic feeding will lead to a negative fat balance and a loss of adipose tissue.

Energy expenditure has been extensively studied in lean and obese individuals. The 24 h energy expenditure, whether measured in a respiratory chamber (Ravussin *et al*. 1982) or by using doubly-labelled water (Schoeller & Fjeld, 1991), increases linearly with increasing body weight. Multivariate analysis shows that lean body mass is the major determinant of energy expenditure, with minor influences of fat mass, age and gender (Ravussin *et al*. 1986). However, the considerable inter-individual variation is currently unexplained. There is at the present time little evidence for major adaptations of energy expenditure during overfeeding, and changes in body weight and body composition appear to be the major factors that, by increasing energy expenditure, allow energy balance to be restored. The body-weight gain may therefore be seen as an adaptative change to overfeeding (Jéquier & Tappy, 1999).

Total energy expenditure can be partitioned into three main components: BMR; the thermic effect of food, or diet-induced thermogenesis; energy expended in physical activity. The first two components can be measured conveniently with reasonable accuracy and have been extensively studied in lean and obese subjects. There is presently no clear evidence that a low BMR is a factor in the development of obesity, although the issue continues to be debated (Ravussin *et al*. 1988). The thermic effect of food, and more specifically the thermic effect of carbohydrate, has been shown to decrease in obese individuals (Golay *et al*. 1989; Laville *et al*. 1993). This decrease may, however, be secondary to obesity-associated insulin resistance, and is of too small a magnitude to account for a major weight gain (Tappy *et al*. 1991).

Role of physical activity in body weight

During physical activity mechanical work associated with muscle contractions clearly requires energy. As a result of

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the combined loss of energy as heat during ATP synthesis in the mitochondria and ATP hydrolysis during muscular contraction, the energetic efficiency of working muscles is approximately 25 %. As a consequence, physical exercise with a power output of 100 W will increase energy expenditure by about 400 W above the basal energy expenditure. Physical activity may therefore have a major impact on the total 24 h energy expenditure and energy balance. Based on the possible means by which alterations in physical activity can impact on 24 h energy expenditure and energy balance, the following hypotheses can be formulated. First, the energy efficiency of physical activity might be increased in some individuals. This response would lead to a lower energy expenditure for any given work load, and would possibly contribute to the achievement of a positive energy balance in affected individuals. There are presently no data to support this hypothesis. The energy efficiency of physical activity shows few inter-individual variations. It was further shown to be similar in both lean and post-obese women (Froidevaux *et al*. 1993). There is therefore little reason to suspect that this factor might be involved in body-weight gain. One interesting report, however, indicated that a common polymorphism of uncoupling protein 2, a protein with close homology to the uncoupling protein of brown adipose tissue ubiquitously expressed in man, was associated with a lower energetic efficiency of muscle contractions (Buemann *et al*. 2001). This observation, and the relationship between this polymorphism and body weight remain to be further evaluated. Second, physical activity may increase the BMR or the thermic effect of food. There are, however, no experimental data to support this hypothesis. Resting metabolic rate is higher in trained individuals than in sedentary individuals of the same weight (Matzinger *et al*. 2002), but the difference can be predominantly attributed to changes in body composition, with a lower fat mass and higher lean, metabolically-active, body mass in trained athletes. When training is associated with a negative energy balance, resting metabolic rate may even decrease (Tremblay *et al*. 1997). The increase in energy expenditure induced by a β-adrenergic stimulation, elicited by mental stress, was also found to be unchanged in obese individuals after a 6-week period of physical training (Seywert *et al*. 2002). Third, the total energy expended in physical activity, i.e. the period of time devoted daily to physical activity, may play a important role in determining energy balance. However, this component of daily energy expenditure is presently very difficult to assess accurately. Respiratory chambers inherently restrain physical activity and therefore are unsuitable for the assessment of physical activity. The doubly-labelled water method allows assessment of total energy expenditure under conditions of everyday life over several days and, therefore, is likely to include habitual physical activity. The difference between the total energy expenditure and the basal energy expenditure, the suprabasal energy expenditure, includes both the thermic effect of food and physical activity. This suprabasal energy expenditure was observed to be low in obese subjects (Schoeller & Fjeld, 1991). This finding strongly suggests that the amount of energy expended in physical activity is low in obese individuals. Whether a low physical activity exists pre-obesity, or is merely a consequence of it, remains an open question at the present time.

Physical activity and fat oxidation

Physical activity leads to increased substrate oxidation by the working muscle. The nature of the fuel mix oxidized depends on several factors. The conclusion from the substantial literature on this topic is that during an acute bout of exercise in untrained fed subjects carbohydrate oxidation supplies the major portion of the extra energy expended. In endurance-trained individuals the oxidative capacity of muscle increases (Romijn *et al*. 1993, 2000). In addition, there is an increased ability to oxidize lipids, due to up-regulation of the enzyme AMP-activated protein kinase in skeletal muscle (Winder & Hardie, 1999). The consequences are increased maximal O_2 consumption and a higher proportion of fat oxidized during exercise of low to moderate intensity. At high intensity, however, the reliance on carbohydrate increases (Romijn *et al*. 1993, 2000).

There is evidence that endurance-trained athletes consume a diet containing a high proportion of carbohydrate. Due to the hierarchy in substrate oxidation mentioned earlier, these dietary habits probably impact on substrate oxidation in everyday conditions. It can be expected, therefore, that athletes who maintain a constant body composition while consuming such a diet will have a high 24 h oxidation of carbohydrates.

In healthy lean untrained individuals studied in a respiratory chamber moderate physical activity increased total energy expenditure, essentially by increasing carbohydrate oxidation when the exercise took place after a meal. However, the same exercise performed in the fasting state, before breakfast, led to a marked increase in lipid oxidation (Schneiter *et al*. 1995). Since the subjects were fed the same isoenergetic diet under both conditions, exercising before breakfast *v*. after breakfast led to a negative lipid balance and to a positive carbohydrate balance. It might therefore be hypothesized that exercise in the fasting state may preferentially promote lipid utilization and favourably affect body composition by decreasing fat stores. This hypothetical scheme appears unlikely, however, if the diet consumed is not changed, since the positive carbohydrate balance will predictably increase carbohydrate oxidation.

Is there an effect of food intake on physical activity? It has been recognized for several decades that severe underfeeding leads to behavioural adaptations that result in a decreased spontaneous physical activity (Owen *et al*. 1990). More recently, there have been reports suggesting that overfeeding increases energy expenditure more than would be predicted on the basis of body weight and lean body mass changes (Leibel *et al*. 1995; Levine *et al*. 1999). Moreover, this increase in energy expenditure showed marked inter-individual variations, and was inversely correlated with body-weight gain (Levine *et al*. 1999). Since changes in BMR and in the thermic effect of food were essentially accounted for by alterations in body composition and dietary intakes, this increase in 'suprabasal' energy expenditure was attributed to a stimulation of spontaneous physical activity. Such physical activity was unrelated to exercise and hence was termed 'non-exercise activity thermogenesis'.

There is currently little understanding of the mechanisms that may possibly be responsible for stimulation of non-exercise activity thermogenesis during overfeeding.

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Calorimetric studies indicate that 24 h energy expenditure and suprabasal energy expenditure increase in healthy volunteers after short-term carbohydrate, but not fat, overfeeding (Dirlewanger *et al*. 2000). This effect was unrelated to the increase in plasma leptin concentrations observed after carbohydrate overfeeding. Several animal studies indicate that stimulation of the melanocortin-4 receptor is associated with an increase in spontaneous physical activity (Ste Marie *et al*. 2000; Adage *et al*. 2001). How the intake of specific macronutrients impact on this pathway and the mechanisms involved remain to be elucidated.

Physical activity and spontaneous food intake

Finally, the hypothesis that physical activity exerts beneficial effects on body-weight status by mechanisms unrelated to an increase in energy expenditure should be considered. Few studies have addressed the effects of exercise on food intake. However, data on energy expenditure and body composition do allow some conclusions to be drawn. If an endurance-trained athlete consistently expends a substantial amount of energy in physical activity and at the same time maintains a constant weight and body composition, energy intake must have increased appropriately, most probably spontaneously. The mechanisms responsible for this increase in energy intake have not been extensively studied. As a result of the complex network in the central nervous system that regulates energy expenditure and the known effects of physical activity on neuro-endocrine regulations, it is very likely that the relationship between energy expended in physical activity and food intake are quite complex. However, endurance-trained athletes are characterized by a low fat mass, which may be the result of previous periods of negative energy and fat balances. They also have low leptin levels, which are essentially attributable to the low fat mass, since comparable leptin levels are observed in very lean sedentary individuals. Numerous studies have searched for a direct inhibition of leptin release by acute or chronic exercise. Except for a decrease in plasma leptin levels following exceptionally intense exercises such as an ultra-marathon (Landt *et al*. 1997), these studies have failed to detect a long-lasting reduction in leptin levels after exercise (Hickey *et al*. 1996; Pérusse *et al*. 1997; Racette *et al*. 1997*a,b*; Dirlewanger *et al*. 1999). This factor may be very relevant to the effect of exercise on body weight. It may indicate that a substantial decrease in fat-free mass induced by exercise is required to decrease leptin secretion before low leptin in turn increases food intake. Interestingly, this absence of effects of exercise contrasts with the effect of severe energy restriction, which rapidly decreases leptin levels before any marked changes in body composition occur (Boden *et al*. 1996). It may indicate that exercise is more efficient than severe energy restriction in the promotion of weight loss without undue rebound hyperphagia.

Conclusions

The effects of physical activity on energy metabolism and body-weight control remain incompletely understood. There is ample evidence that physical training is associated with low body weight and low fat mass. This relationship unambiguously indicates that negative energy and fat balances are associated with physical training. The negative energy balance is likely to be directly secondary to the amount of energy expended while exercising, since there is no evidence that exercise affects other components of energy expenditure. The negative fat balance is probably secondary to this negative energy balance. In obese individuals the amount of energy expended in physical activity appears to be small, which certainly represents a factor that could prevent weight loss. Two issues that remain to be resolved are whether pre-obese individuals have a low physical activity level that contributes to weight gain and, if so, what are the biological determinants of this low physical activity.

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