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## Nutrition and Behaviour Group Symposium on 'The relationship between physical activity patterns and patterns of food, energy and nutrient intake'

# Influence of intense physical activity on energy balance and body fatness

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The reduced contribution of physical activity to daily energy expenditure and the accessibility to high-fat foods have put an excessive burden on energy balance, resulting in an increase in the prevalence of obesity throughout the world. In this context, fat gain can be seen as a natural adaptation to deal with a fattening lifestyle, since the hormonal adaptations that accompany fat gain favour the readjustment of energy expenditure to energy intake. Intense physical activity would also seem to facilitate the regulation of energy balance, since it increases the energy cost of exercise, increases post-exercise energy expenditure and the potential of skeletal muscles to utilize lipids, and also favours a decrease in post-exercise intake. Moreover, the effects of intense exercise seem to be mediated by an activation of sympathetic nervous system activity that seems to be specific to skeletal muscle. It is also important to manipulate macronutrient composition in order to reduce fat intake, because unhealthy food habits can favour overfeeding and thus overcome the energy deficit caused by regular physical activity. Under free-living conditions, the combination of vigorous activity and healthy food practices can amount to a substantial weight loss which is comparable with that of other non-surgical approaches to treat obesity.

**Exercise: Energy balance: Fat intake: Obesity**

The mechanization and computerization of labour that have occurred over this century have considerably reduced the energy demand of daily chores. This effect has not been quantified in longitudinal studies, but some cross-sectional observations suggest that the impact of this lifestyle modification on daily energy needs is often underestimated by health professionals. In a study reported by Singh *et al.* (1989), the daily energy expenditure : resting metabolic rate ratio at the peak labour season in Gambian women was 2·35, which strongly contrasts with values of 1·4–1·9 (Livingstone *et al.* 1991; Meijer *et al.* 1992; Black *et al.* 1996; Prentice *et al.* 1996; Spurr *et al.* 1996) that are generally observed in women living in industrialized countries. At a given body weight, this observation may correspond to a difference in daily energy expenditure that may be as large as 3–4 MJ/d.

An increase in the fat content of the diet is another significant lifestyle change that has occurred in this century; available estimates reveal that this increase in dietary fat corresponded to 10–20 % (Danforth, 1985; Leaf & Weber, 1987). The impact of such an increase in relative fat intake on *ad libitum* energy intake has been the object of many studies which have demonstrated that overfeeding associated with a high-fat diet may reach 1–4 MJ/d (Lissner *et al.* 1987; Tremblay *et al.* 1989, 1991*b*; Stubbs *et al.* 1995). Overfeeding may also result from alcohol intake that is at best partly compensated by a decrease in other energy substrates (Foltin *et al.* 1993; Tremblay *et al.* 1995*c*; Poppitt *et al.* 1996; Tremblay & St-Pierre, 1996). Thus, a lifestyle characterized by sedentariness and high fat and alcohol intakes increases the risk of positive energy balance and body fat gain (Tremblay *et al.* 1995*a*). This issue has been

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documented in the UK, where the increases in both the fat content of diet and sedentariness have been associated with body-weight gain in the last decades (Prentice & Jebb, 1995), although sedentariness appears to be a better predictor of adiposity in more recent years.

The prevalence of obesity continues to increase in industrialized countries and the related comorbidities impose an increasing burden on health care systems (Wolf & Colditz, 1998). Accordingly, the so-called 'epidemic of obesity' (World Health Organization, 1998) draws attention to the health consequences of excess body fat deposition, whereas much less consideration is generally given to the physiological mechanisms that may make obesity a necessity in some individuals. In the present paper, questions are raised as to whether fat gain is the price which has to be paid for recovery of energy balance in a 'fattening' environment, and whether intense exercise can represent a valuable means of counteracting this undesired outcome.

## Fat gain: a natural adaptation to deal with a modern lifestyle

The mechanisms contributing to the recovery of energy balance when weight gain occurs have not been clearly established, but experimental evidence suggests that fat gain is the common feature driving many of the hormonal and metabolic adaptations which favour long-term regulation of energy and fat balance. According to Groop *et al.* (1992), an increase in the contribution of free fatty acids to metabolism promotes fat oxidation, which seems to be a strategy that helps the obese individual to recover fat balance. This fatty acid gradient might be particularly useful for obese individuals who display lower rates of fat oxidation than their lean controls when they are tested in a reduced-obese state (Lean & James, 1988; Buemann *et al.* 1992; Larson *et al.* 1995).

Hyperinsulinaemia is not only a feature of the plurimetabolic syndrome (Reaven, 1988), but also seems to be part of a mechanism for restoring energy balance over time when body-weight gain occurs. Hyperinsulinaemia with euglycaemia promotes an increase in sympathetic nervous system activity, either measured by the level of circulating catecholamines (Rowe *et al.* 1981), muscle sympathetic nerve activity (Berne *et al.* 1992; Vollenweider *et al.* 1994) or heart rate variability (Bellavere *et al.* 1996). This effect of hyperinsulinaemia represents a potential explanation for the positive relationship that exists between muscle sympathetic nerve activity and body fat (Scherrer *et al.* 1994), particularly when fat is preferentially stored in the abdominal area (Parker Jones *et al.* 1996).

Since an increase in sympathetic activity favours thermogenesis and a decrease in energy intake (Rothwell & Stock, 1981; Himms-Hagen, 1984), hyperinsulinaemia appears to be a factor that may contribute to the long-term restabilization of energy balance in individuals experiencing weight gain. This is consistent with prospective data showing that higher baseline serum insulin is associated with a reduced long-term body-weight gain in obese subjects (Valdez *et al.* 1994; Folsom *et al.* 1998). However, other findings do not support this concept (Folsom *et al.* 1998). In this respect, it is of particular interest to emphasize results obtained by the Phoenix group, who reported that hyperinsulinaemia is associated with a reduced long-term weight gain in adults (Schwartz *et al.* 1995) whereas the opposite relationship is observed in children (Odeleye *et al.* 1997). This may indicate that the peripheral anabolic effects of hyperinsulinaemia may dominate its central catabolic effects in children in order to promote growth.

The study of overfed monozygotic twins in our laboratory showed that a high increase in postprandial insulinaemia predicts a greater increase in postprandial energy expenditure (Tremblay *et al.* 1995*b*). Since this effect and those described previously were documented in a context where glycaemia was relatively stable, it is important to make the distinction between this phenomenon and the impact of hyperinsulinaemia on energy balance when it is accompanied by hypoglycaemia. In the latter case, an increase in energy intake is expected (Campfield & Smith, 1986), which is concordant with the clinical observation that a reinforcement of insulin therapy in type 1 diabetics not only results in a decrease of glycaemia but also favours body-weight gain (Wing *et al.* 1990; Carlson & Campbell, 1993).

The discovery of leptin (Zhang *et al.* 1994) provided support for the idea that energy and fat balance are subject to regulation that involves lipostatic factors (Kennedy, 1952). Leptin is produced in adipose cells, and its circulating levels vary in proportion to body fatness (Considine *et al.* 1995; Lönnqvist *et al.* 1995). As shown for insulin, the increase in plasma leptin which accompanies obesity also seems to be a factor facilitating the recovery of energy balance in the individual experiencing weight gain. Indeed, leptin promotes thermogenesis (Pelleymounter *et al.* 1995), reduces energy intake (Campfield *et al.* 1995), favours an increase in spontaneous movement (Salbe *et al.* 1997), reduces neuropeptide Y gene expression and thus attenuates the orexigenic effects of this peptide (Schwartz *et al.* 1996), and is associated with an increase in muscle sympathetic nerve activity (Snitker *et al.* 1997). As illustrated in Table 1, both leptin and insulin promote central effects leading to a negative energy balance, or at least allow the recovery of energy balance on exposure to an energy surplus.

In summary, these observations clearly show that the body is not passive when there is chronic exposure to excess energy intake. Hormonal and metabolic changes are displayed that permit the body to re-equilibrate energy intake and expenditure over time. As mentioned previously, fat gain is a common feature of these changes, and thus represents the price to be paid in order to take advantage of this strategy of regulation of energy balance.

**Table 1.** Effects of leptin and insulin (euglycaemia) on energy balance

| | Leptin | Insulin |
|---|---|---|
| Energy intake | ↘ | ↘ |
| Energy expenditure | ↗ | ↗ |
| Activity level | ↗ | ? |
| Neuropeptide Y | ↘ | ↘ |
| SNS activity | ↗ | ↗ |

SNS, sympathetic nervous system; ↗, promote; ↘, suppress.

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### Is intense exercise a potential alternative?

In a modern world where productivity with minimal energy cost is frequently perceived as the gold standard of living, exercise may be considered as a useless facultative investment. In fact, it is likely that with the rapid computerization of labour, sedentariness will further dominate physical activity participation than it does currently. This raises the question as to whether an increase in exercise intensity can compensate for the decrease in the time allocated to long-duration low-intensity physical activities. This issue has been of particular interest in our group over the last decade. Its investigation was initiated with the analysis of epidemiological data collected in the first Canada Fitness Survey (Stephens *et al.* 1986). This study revealed that for a given energy cost of leisure-time physical activities, subjects regularly practising vigorous activities were leaner than those not reporting participation in these activities (Tremblay *et al.* 1990). We further investigated this issue in the context of intervention studies by comparing the effect of two exercise training programmes differing in the intensity of exercise stimulus on body composition and skeletal muscle metabolism (Tremblay *et al.* 1994*b*). As illustrated in Fig. 1, the training programme of moderate intensity had a much greater energy cost than the high-intensity intermittent exercise programme. Despite its lower energy cost, the high-intensity intermittent exercise programme induced a greater loss in subcutaneous adiposity in comparison with the programme of moderate exercise intensity. When expressed per MJ expended during exercise, the fat loss was 9-fold greater after the high-intensity intermittent exercise programme. The enhancing effect of this programme on skeletal muscle oxidative potential was also more pronounced than that of the moderate-intensity programme (Fig. 1).

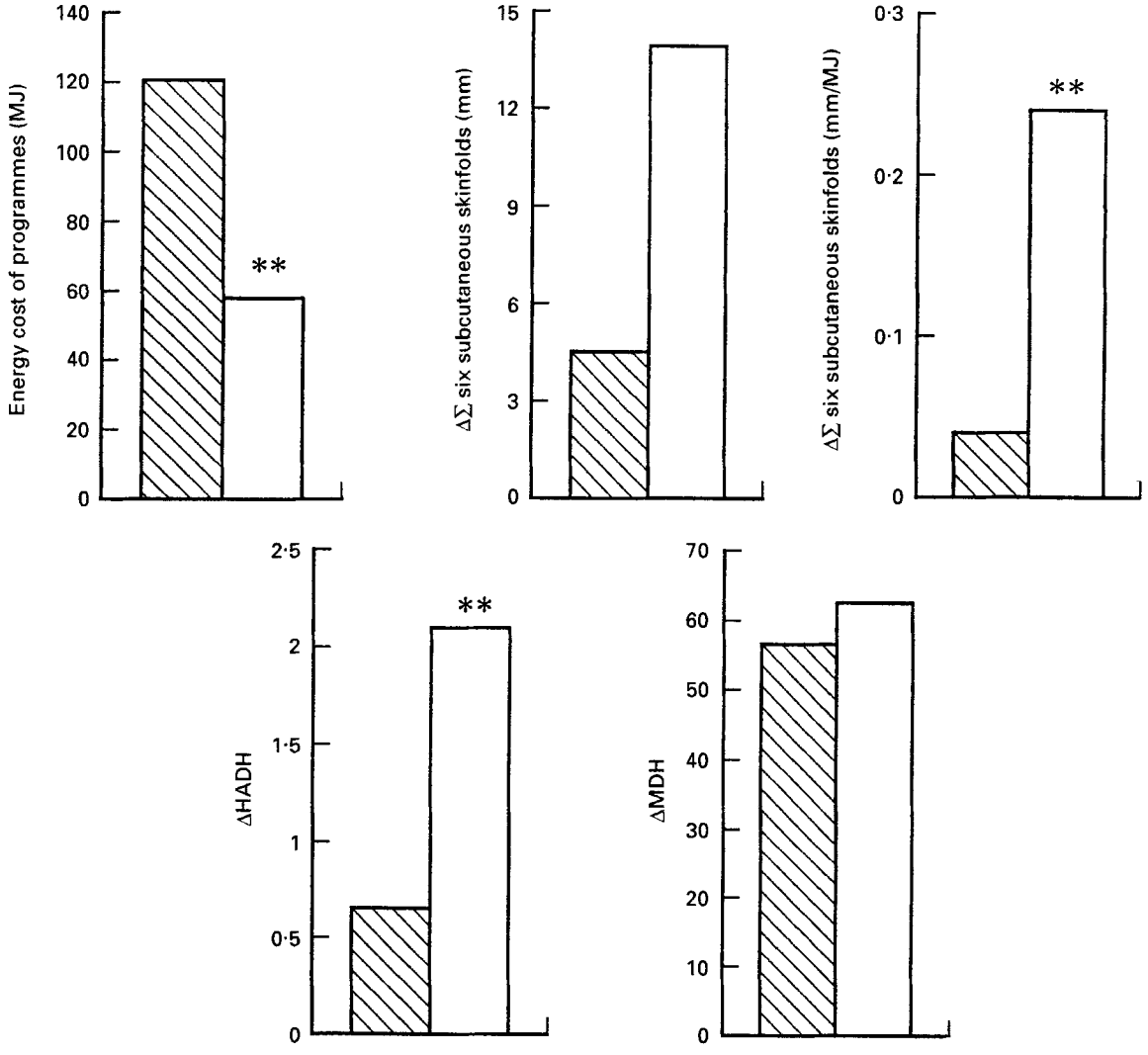


**Fig. 1.** Comparison of the effects of endurance training (▨) and high-intensity intermittent training (□) on subcutaneous adiposity and skeletal muscle metabolism. ΔHADH, change in 3-hydroxyacyl-Co A dehydrogenase (*EC* 1.1.1.36) activity; ΔMDH, change in malate dehydrogenase (*EC* 1.1.1.37) activity; ΔΣ, change in sum of. Mean value was significantly different from that for endurance training: $^{**}P \leq 0{\cdot}01$. (Adapted from Tremblay *et al.* 1994*b*.)

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Theoretically, there are three ways in which exercise intensity can modify energy balance: (1) an increase in the energy cost of exercise, (2) an increase in post-exercise energy expenditure, (3) a decrease in post-exercise energy intake. The first one is rather obvious, since it is well established that for a given individual the energy cost of exercise is proportional to its intensity. With respect to the other two effects, recent findings suggest that exercise intensity can modify both post-exercise energy expenditure and intake. We examined this issue by submitting subjects to two exercise sessions having a similar energy cost but differing in their intensity. In a first study, post-exercise resting $O_2$ uptake and fat oxidation were found to be greater after high-intensity exercise (70–75 % maximum $O_2$ uptake) than following low- to moderate-intensity (35–40 % maximum $O_2$ uptake) exercise (Yoshioka *et al.* 1997). The acute effects of exercise intensity on energy intake were tested in another study, which revealed that the post-exercise compensation in energy intake was less pronounced after a high-intensity exercise bout than a low- to moderate-intensity exercise bout (Imbeault *et al.* 1997).

Recent data obtained in the same context suggest that the enhancing effect of intense exercise on energy metabolism is mediated by β-adrenergic stimulation (Yoshioka *et al.* 1997). This is concordant with results demonstrating that the difference in resting metabolic rate and fat oxidation between trained and untrained subjects does not persist when determined after propranolol administration (Tremblay *et al.* 1992). This suggests that the thermogenic effects of intense exercise resemble the thermogenic effects of ephedrine (Astrup *et al.* 1986), capsaicin (Watanabe *et al.* 1987; Yoshioka *et al.* 1998), and caffeine (Bracco *et al.* 1995). However, the effects of exercise, unlike those of sympathomimetic compounds, are tissue specific, since it has been shown in animals that exercise training induces an increase in the concentration of β-adrenergic receptors in skeletal muscle, whereas the opposite adaptation is seen in the heart (Plourde *et al.* 1991, 1993). This finding is in agreement with the well-established fact that resting heart rate is generally reduced in trained individuals, even if they display an increase in resting metabolic rate mediated by β-adrenergic stimulation.

Taken together, these observations suggest that for a given energy cost, intense exercise is more effective in its ability to alter daily energy balance than low- to moderate-intensity exercise. This is not entirely attributable to the increased energy cost of exercise, but also depends on the potential of this type of exercise to increase post-exercise metabolic rate and to attenuate post-exercise compensation in energy intake.

## Healthy food habits: an important determinant of the impact of exercise on energy balance

As indicated previously, many studies have shown that high-fat feeding favours excess *ad libitum* energy intake (Lissner *et al.* 1987; Tremblay *et al.* 1989) and long-term body fat deposition (Dreon *et al.* 1988; Romieu *et al.* 1988; Tremblay *et al.* 1989; Tucker & Kano, 1992). However, this does not seem to be the case for a high polyunsaturated-fat intake, since this subclass of dietary lipids is a better promoter of its own utilization than saturated fat (Jones *et al.* 1985; Jones & Schoeller, 1988) and is not associated with excess body fat deposition (Doucet *et al.* 1998).

Our group (Tremblay *et al.* 1994*a*) and King & Blundell (1995) investigated the effect of a high-fat diet on the potential of exercise to alter energy balance. The main finding of these studies was that the overfeeding resulting from a high-fat diet was sufficient to compensate for the surplus of energy expended both during exercise and in the post-exercise period. In a subsequent study, we used a whole-body indirect calorimeter to examine the impact of the combination of exercise and a low-fat diet on daily energy balance in heavy men (Dionne *et al.* 1997). As shown in Table 2, this treatment induced a considerable change in daily energy balance that was mainly explained by variations in the energy cost of exercise and in post-exercise energy intake.

Experimental evidence suggests that satiety may be achieved with a reduced energy intake if there is an increase in the content of proteins (Hill & Blundell, 1986, 1990), low-glycaemic-index foods (Holt & Miller, 1995), dietary fibres (Turconi *et al.* 1995), caffeine (Tremblay *et al.* 1988; Racotta *et al.* 1994), and capsaicin (M Yoshioka, S St-Pierre, V Drapeau, I Dionne, E Doucet, M Suzuki and A Tremblay, unpublished results) in the diet. However, these dietary manipulations have not been tested in conjunction with exercise participation. It is thus relevant to undertake further research to determine whether some of these nutritional factors can increase the effect of combination of exercise and a low-fat diet on energy balance.

## Implications for the treatment of obesity

The exercise–low-fat-diet combination should theoretically permit satiety while an obese individual experiences a body energy deficit. In our first clinical trial to test this hypothesis, this treatment induced a mean body weight loss of 14·6 kg (about 16 % of initial body weight) before a resistance to further body fat loss was reached in obese women (Tremblay *et al.* 1991*a*). More recently, the exercise–low-fat-diet combination was used as a follow-up strategy after obese subjects were submitted to an initial phase of a low-energy diet with fenfluramine or a placebo. In this situation, the exercise–low-fat-diet follow-up accentuated initial weight and fat loss up to a threshold of resistance to further fat loss that corresponded with 10 and 14 % of initial body weight in obese women and men respectively (E Doucet, P Imbeault, N Alméras and A Tremblay, unpublished results).

**Table 2.** Difference in energy balance between time periods (d) characterized by either exercise and a low-fat diet or sedentariness and a mixed diet as measured by a whole-body indirect calorimeter (Adapted from Dionne *et al.* 1997)

| | |
|---|---|
| (a) Surplus of energy expenditure above resting level due to the energy cost of exercise | 3·8 MJ |
| (b) Difference in post-exercise daily energy expenditure (in a whole body calorimeter: post-exercise–rest) | 0·5 MJ |
| (c) Difference in daily energy intake between the two conditions (rest – post-exercise) | 3·0 MJ |
| Difference in energy balance between exercise day and sedentary day (a+b+c) | 7·3 MJ |

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This level of weight loss is comparable with that generally found with the use of other non-surgical approaches to treat obesity. Thus, if exercise could be combined with a diet having a greater satiety potential than the traditional low-fat diet, the impact on body-weight control in obese individuals might be greater than that achieved up to now.

## Summary

An increase in the intensity of exercise may accentuate the negative energy balance that is normally favoured by regular participation in low- to moderate-intensity activities. This effect does not seem to be entirely due to the increased energy cost of exercise, but also to an increase in post-exercise energy expenditure and a decrease in the post-exercise compensation in energy intake. Experimental data also demonstrate that the nutritional context surrounding exercise participation is an important determinant of the potential of exercise to induce a negative energy balance. Up to now, it has been shown that a decrease in dietary fat intake facilitates the occurrence of a negative energy balance in response to exercise. Moreover, it is possible to perform dietary manipulations that may improve the satiating power of low-fat diets. This could be done by some adjustments in the protein and fibre content of the diet and/or by the intake of food-related sympathomimetic agents. Accordingly, people who regularly perform intense exercise and who adhere to healthy food habits generally maintain energy balance without relying on fat gain to support the mechanisms involved in the long-term regulation of energy and fat balance.

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