

## Invited commentary

### Physical activity and metabolic cardiovascular syndrome

Industrialization and computerization have provided comfort and well-being to communities living in industrialized countries but have also been responsible for a considerable decrease in daily energy needs. This issue has not been investigated in longitudinal studies but cross-sectional observations suggest that the transition from traditional to modern lifestyle may have induced a decrease in daily energy expenditure of 1–2 MJ/d, or even more (Singh *et al.* 1989). Since a regular participation in physical activity is known to facilitate the control of body weight, to reduce glycogen stores, and to increase sympathetic nervous system (SNS) activity, at least in skeletal muscle, a shift towards a sedentary lifestyle also necessarily involves a loss in the potential to regulate efficiently the energy–carbohydrate–lipid metabolism. This means that active people can maintain energy and macronutrient balance without excess plasma glucose and non-esterified fatty acid concentrations and their related mass-action effect to sustain substrate flux. In more clinical terms, regular physical activity thus reduces the likelihood to develop diseases associated with excess circulating substrates that are characterizing metabolic cardiovascular syndrome.

In this month's issue, Wareham and colleagues (Wareham *et al.* 1998) report a study that examined the relationship between physical activity participation and the risk to develop metabolic cardiovascular syndrome. As expected, the less-active, less-fit subjects were more prone to display the features of the syndrome, i.e. an increase in fasting and post-glucose glycaemia, hyperinsulinaemia and blood pressure. They were also more predisposed to dyslipidaemias.

The original aspect of this study design was to allow a dissociation of the effects of fitness and energy expenditure in the aetiology of metabolic cardiovascular syndrome. The

fitness component reflects long-term adaptations to exercise such as change in adiposity and the cumulative impact of exercise on mechanisms involved in biological work production. The energy expenditure component rather integrates the effect of variations in spontaneous movement (fidgeting) energy cost of activities, and voluntary participation in physical activities. This component also reflects the contribution of acute changes in variables involved in the regulation of energy and macronutrient balance such as glycogen stores, SNS activity, and other less-characterized factors. In addition, the two components are probably subjected to a different effect of heredity, sex, and measurement error.

The calculation of univariate odds ratio revealed that the association with metabolic cardiovascular syndrome was stronger for the fitness component. However, after adjustment for obesity, sex, and exposure measurement error, results suggested that the syndrome was rather strongly related to reduced habitual energy expenditure.

Beyond these specific conclusions, the question that arises from the Wareham *et al.* paper is 'What is the biological meaning of their results?' An attempt to answer this question is illustrated in Table 1 which first draws attention to the opposite effects of physical activity and metabolic syndrome on blood pressure, plasma glucose and insulin, and plasma lipids–lipoproteins. Table 1 also emphasizes that both physical activity and the syndrome can increase SNS activity and energy expenditure. Indeed, exercise has been shown to increase post-exercise resting energy expenditure via an increase in SNS activity (Poehlman & Danforth, 1991) mediated by  $\beta$ -adrenoreceptors (Tremblay *et al.* 1992). This effect seems to be tissue-specific since an increased concentration of  $\beta$ -adrenoreceptors was observed in skeletal muscle but not in heart of

**Table 1.** Opposite (A) and concordant (B) effects of physical activity and metabolic cardiovascular syndrome

	Physical activity effect	Variable	Metabolic syndrome effect
A	↘	Blood pressure	↗
	↘	Plasma glucose	↗
	↘	Plasma insulin	↗
	↘	Plasma triacylglycerols	↗
	↘	Plasma total cholesterol	↗
	↗	Plasma HDL-cholesterol	↘
	↘	Plasma apo-B*	↗
	↘	Plasma cholesterol:HDL-cholesterol	↗
B	↗	LDL particle size*	↘
	↗	SNS activity	↗
	↗	Energy expenditure	↗

\* Additional atherogenic features of the syndrome (Lamarche *et al.* 1998).

trained animals (Plourde *et al.* 1991, 1993). With respect to metabolic cardiovascular syndrome, its related state of hyperinsulinaemia was found to activate SNS activity (Rowe *et al.* 1981; Berne *et al.*). In addition, studies have also demonstrated that hyperinsulinaemia is associated with an increase in postprandial energy expenditure (Tremblay *et al.* 1995) and attenuation of long-term body weight gain (Schwartz *et al.* 1995) in adults.

The resemblance of the effects of physical activity and metabolic cardiovascular syndrome on the regulation of energy balance suggests that the syndrome might well be an alternative to physical activity to stimulate energy metabolism. However, Table 1 and the data of Wareham *et al.* (1998) remind us that taking advantage of this regulatory strategy is associated with an increased risk of developing metabolic and cardiovascular complications. Thus, this paper seems to hit right on target by providing evidence that suggests that metabolic cardiovascular syndrome is the price to be paid to deal with an environment where physical activity appears as the fossil of industrialization and computerization.

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