Endurance Training With Constant Energy Intake in Identical Twins: Changes Over Time in Energy Expenditure and Related Hormones

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The effects of exercise training and of its interaction with the genotype on components of energy expenditure and related hormones were examined in young male monozygotic twins. Energy intake was maintained at the pretraining level for a 93-day training period. The estimated net energy deficit induced by training was 244 MJ and was associated with a 5-kg body weight loss that was almost entirely explained by body fat loss. Resting metabolic rate (RMR) was significantly decreased by 8% after training despite the preservation of fat-free mass (FFM). Accordingly, plasma norepinephrine (NE) concentrations, NE appearance rate, and plasma levels of triiodothyronine (T_3), free T_3, and total thyroxine (T_4) were lower after training. The energy cost of standardized exercise was also reduced after the training program. A modest to significant within-twin-pair resemblance was observed for absolute changes in the RMR, thermic effect of food, energy cost of exercise, NE clearance, and plasma concentrations of thyroid hormones. These results suggest that when exercise training is associated with a substantial negative energy balance, energy expenditure and levels of related hormones are decreased, and this effect is partly accounted for by heredity.

VIGOROUS PHYSICAL ACTIVITY has the potential to induce a short-term energy deficit because of its energy cost and potential impact on other components of daily energy expenditure. The resting metabolic rate (RMR) usually explains the major part of daily energy expenditure and is sometimes increased by one bout of prolonged vigorous exercise.[1] The notion of an acute effect of exercise on RMR is corroborated by results suggesting that a significant decrease in this variable is observed after several days of inactivity in trained subjects.[2,3] Individuals performing aerobic exercise on a daily basis can also display an increased RMR.[4,5] This suggests that the enhanced RMR characterizing trained individuals is not mainly due to their fitness, but is instead the result of the repeated acute effects of exercise. Regular aerobic exercise in overweight subjects also increases RMR,[5,6] but there are contradictory results. Moreover, when training is accompanied by body weight and fat loss, the enhancing effect of exercise on RMR may be blunted. Indeed, we recently reported that a 100-day training program that was planned to induce a 4.2-MJ/d surplus in energy expenditure in overweight young men resulted in a small nonsignificant decrease in RMR.[7] Under severe energy restriction, regular aerobic exercise accentuated the reducing effect of a very-low-calorie diet on RMR in overweight individuals.[8,9] Moreover, experimental data show that the response of RMR to short-term endurance training may be influenced by undefined genetic factors.[10] However, uncertainty persists as to whether this genetic effect is still observed in response to a training program of longer duration and that induces a greater energy deficit. Thus, the first two aims of this study were to measure variations in RMR in response to a long-term energy deficit induced by exercise, and to determine the extent to which this variation is attributable to the genotype.

Alterations in sympathetic nervous system (SNS) activity have also been implicated in the regulation of resting energy expenditure in response to training.[11,12] However, this potential effect of SNS activity has not been investigated under training conditions imposing a large energy expenditure and deficit. Therefore, the third purpose of this study was to examine the effects of prolonged endurance training on RMR, norepinephrine (NE) kinetics, and fasting concentrations of thyroid hormones in monozygotic twins.

SUBJECTS AND METHODS

Subjects

Eleven pairs of sedentary male monozygotic twins aged 21 ± 0.8 years (mean ± SEM) were recruited to participate in this study, which was approved by the Laval University Medical Ethics Committee and the Office for Protection from Research Risks of the National Institutes of Health. Seven pairs of twins completed the total protocol. This report is based on values from these 14 subjects, whose age varied between 17 and 26 years. The monozygosity of these twins was established with several markers.[13]

None of the subjects had a history of recent illness related to the variables tested in this study. Criteria for subject selection were as follows: no clinical symptoms or signs of heart disease, resting blood pressure less than 140/90 mm Hg, normal resting electrocardiogram (ECG), normal ECG response to an exercise stress test, absence of any prescription or over-the-counter medication that could affect cardiovascular function, and no family medical history of diabetes.

Six subjects were light smokers. Two members of two twin pairs were smokers, and the other two smokers were from different pairs. Smoking was discouraged during the study, and this resulted in a reduction of the frequency of smoking to a few cigarettes per day. Further details about this intervention study have been reported elsewhere.[13]

Experimental Protocol

Six to eight subjects were tested at a time over 2 years. The experimental protocol was exactly the same for each subgroup. For the duration of the study, subjects were housed in an experimental research station located in a wildlife reserve area approximately 80 km from the Laval University Campus. They were under 24-hour supervision by research assistants living with them. On average, each subject stayed in the unit for 117 consecutive days, which included the following three

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periods: a 17-day baseline observation period including initial measurements, 93 days for the exercise training program, and 7 days for testing at the end of the protocol.

Baseline Period

The 17-day observation period was mainly aimed at estimating the energy cost of body weight maintenance before the training program. Subjects were instructed to freely eat foods prepared by dietitians involved in the study. All foods were weighed before meals and reweighed after meals when not consumed to precisely determine daily food intake. The energy content and macronutrient composition of foods were calculated using the Canadian computerized nutrient file.[14]

Body weight was measured every day, whereas hydrostatic weighing was performed three times during the baseline period. In subjects who displayed changes in fat and fat-free mass (FFM) during this period, the energy equivalents of these changes were used to correct the energy intake of body weight maintenance. In such cases, corrections were performed by assuming that the energy equivalent of fat and lean tissues corresponds to 38.9 and 4.3 MJ/kg, respectively. The baseline energy cost of weight maintenance estimated from these calculations was used to establish the daily energy intake of each subject during the training program.

Testing Before and After the Training Program

RMR was measured after a 12-hour fast. Upon arrival at the laboratory, the subject was seated in a comfortable semireclined chair with his head inside a Beckman hood system (Schiller Park, IL). After a 30-minute rest period, the concentrations of oxygen and carbon dioxide were measured using paramagnetic and infrared analyzers (S-3A and CD-3A; Ametek, Pittsburgh, PA), and pulmonary ventilation was assessed with a Fleish respirometer. The energy equivalent of oxygen consumption ($\dot{V}O_2$) was calculated according to the method of Weir.[15] Following measurement of RMR, the subject consumed a 4.2-MJ meal with a macronutrient composition of 15%, 35%, and 50% energy as protein, lipid, and carbohydrate, respectively. The meal was consumed in 15 minutes, and calorimetric measurements were then continued for 240 minutes. These data were used for calculation of the thermic effect of the meal (TEM), which corresponded to the mean total energy expenditure above RMR over the postprandial measurement period.

On the following day, the energy cost of treadmill walking was measured approximately 2 hours after a standardized meal for 10 minutes at each of the following exercise intensities: 4.5, 5.5, and 6.5 km/h (0% slope). The subject wore a nose clip and a mouthpiece for expired-gas collection. Zirconia-cell and infrared analyzers (S-3A, Applied Electrochemistry, Sunnyvale, CA; and Anarad RI, Santa Barbara, CA) were used to assess oxygen and carbon dioxide concentrations, and pulmonary ventilation was measured with a Fleish respirometer. For resting measurements, the Weir[15] formula was used to determine the energy equivalent of $\dot{V}O_2$.

A series of measurements were also performed at the University of Vermont before and after the training program. Plasma NE kinetics (appearance and clearance rates) were determined under steady-state conditions using a modification of the tritiated dilution method of Esler et al.[16] The dose of infused ^{3}H-NE was 0.71 μCi/min for 60 minutes. Arterialized blood samples were drawn from a hand vein 50, 55, and 60 minutes later for determination of steady-state conditions, measurement of plasma NE levels, and calculation of plasma NE appearance and clearance rates. NE plasma clearance rates (liters per minute) were calculated as the infusion rate (cpm per minute) divided by cpm per liter of plasma (mean of three samples corrected for extraction recovery). Appearance rates (micrograms per minute) were calculated as clearance (liters per minute) times plasma NE concentration (micrograms per liter).

Plasma thyroxine (T_4), free T_4, and 3,5,3′-triiodothyronine (T_3) concentrations were measured using clinical assay kits (Baxter, Cambridge, MA) and free T_3 was assayed using an analog assay (Diagnostic Products, Los Angeles, CA), whereas NE concentrations were determined according procedures previously described.[11] We also took advantage of the testing at the University of Vermont to repeat the measurement of RMR, which was performed according to previously described procedures.[9] Ametek analyzers were also used for determination of oxygen and carbon dioxide concentrations.

Training Period

Each subject performed cycle ergometer exercise twice per day (57 ± 0.8 min/session) over a period of 93 ± 0.6 days at an intensity of 50% to 55% maximum $\dot{V}O_2$ ($\dot{V}O_2$max). A training day included one session in midmorning and one in midafternoon. Since 1 day of rest was planned every 10 days of exercise, a total of 160 sessions were completed by each subject over the 93-day training period. For each subject, exercise intensity was carefully controlled every session by monitoring the heart rate. The amount of work prescribed was calculated to induce an exercise energy expenditure of 4.2 MJ/d (1,000 kcal/d) above RMR. Since the estimated excess energy expenditure was standardized for each subject, body size and other correlates of resting and exercise energy expenditure were not expected to affect the results of this study.

As previously described,[13] this calculation was based on a preexperiment submaximal exercise test that allowed derivation of an individual regression line between $\dot{V}O_2$ and heart rate. The energy equivalent of $\dot{V}O_2$ at a target heart rate was derived using the Weir formula.[15] This procedure was repeated every 25 days, and the exercise prescription was then adjusted, if necessary, to maintain the predetermined training-induced energy deficit.

The estimated total energy cost of training above RMR was 354 ± 4 MJ. Considering that subjects expended more energy above the RMR level during daytime sedentary activities, this estimate was adjusted to derive a more realistic estimate of the net total energy deficit resulting from training. This was achieved by calculating the total energy cost of exercise above the postprandial awakened state, which gave a net estimated energy deficit of 244 ± 9.7 MJ. This energy deficit was also estimated for the first and second half of the protocol.

Statistical Analysis

The effect of training and the genotype-training interaction effect were assessed with a two-way ANOVA for repeated measures on one factor (time). The twins were considered nested within the pair, whereas the treatment effect was considered as a fixed variable. The intraclass correlation coefficient for changes induced by training provided a quantitative estimate of the resemblance within pairs in the response to the protocol. A paired t test was used to compare the energy deficit induced by the protocol and the body energy loss between the first and second half of the experimental treatment.

RESULTS

As previously reported,[13] the training protocol induced a mean decrease in body weight of 5.0 kg. This weight loss was almost entirely explained by fat loss, since the mean decrease in fat and FFM was 4.9 and 0.1 kg, respectively, as estimated from underwater weighing. As expected, training induced a significant increase in $\dot{V}O_2$max (3.12 v 3.54 L/min, $P \leq .001$).[13]

Table 1 shows that RMR was significantly decreased after the first half of the program. This reduction corresponded to 8% of the initial level and was maintained throughout the protocol. This table also compares values obtained at Laval University and the University of Vermont before and after the training protocol. The initial RMR and the change induced by training

Table 1. Effect of Exercise Training on RMR (kJ/min) Measured at Laval University and at the University of Vermont

Laboratory	Time: Before	Half	After
Laval	5.1 ± 0.2	4.7 ± 0.2*	4.7 ± 0.2*
Vermont	5.1 ± 0.2		4.7 ± 0.2*

NOTE. Values are the mean ± SEM. Statistical significance was established from a 2-way ANOVA with repeated measurements on 1 factor (time).

*Significantly different from the initial value, $P < .05$.

were identical in the two laboratories. Measurement of RMR at Laval University showed a significant within-pair resemblance for changes in RMR induced by training (Fig 1). The same trend was observed at the University of Vermont, but the effect was slightly less than standard statistical significance (F ratio = 2.11, intraclass correlation = .36).

Table 2 indicates that TEM was not significantly modified by the negative energy balance protocol. In addition, the twin resemblance for fluctuations in TEM was not statistically significant. The energy cost of treadmill walking at different speeds was reduced by the protocol, but the reduction did not always reach statistical significance (Table 2). However, individual variations in the response to the protocol were observed, and these did not occur at random, since a significant within-pair resemblance was observed for changes in the energy cost of walking. This resemblance was reduced, but remained statistically significant when the energy cost of walking was adjusted for body weight at 5.5 and 6.5 km/h (Table 2).

A significant 23% decline in fasting plasma NE concentrations was observed after the protocol (Table 3). This decline was primarily due to a 35% decline in NE appearance rate, whereas no change was noted in NE clearance. Changes in NE concentrations and appearance rates were randomly distributed among twin pairs, whereas individual changes in NE clearance exhibited moderate within–twin-pair resemblance (Table 3). Indeed, there was 2.7 times (F ratio) more variance for changes in NE clearance between pairs than within pairs.

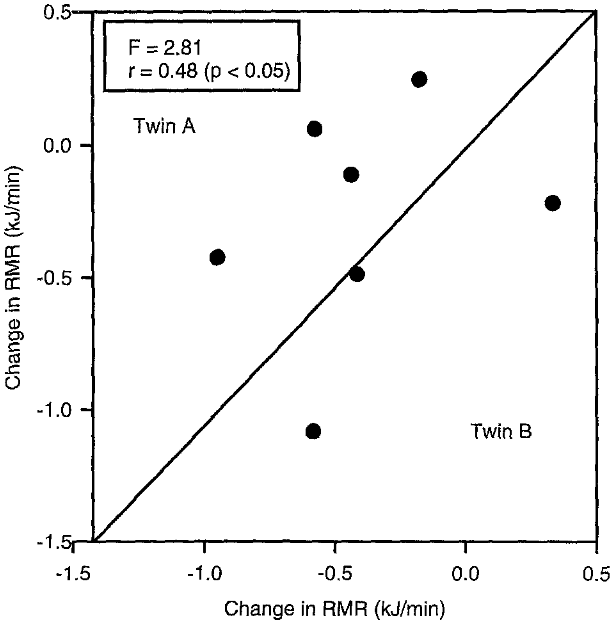

Fig 1. Intrapair resemblance in the response of RMR to training when subjects were tested at Laval University. Values are absolute changes in RMR in each member of twin pairs relative to the identity line.

Table 2. Effect of Exercise Training on the TEM and Energy Cost of Treadmill Walking at Different Speeds and Intrapair Resemblance in the Response to the Protocol

Parameter	Time: Before	Half	After	F Ratio	Intraclass Coefficient
TEM (kJ/4 h)	239 ± 26	248 ± 15	258 ± 16	1.95	.32
Energy cost					
4.5 km/h					
kJ/min	20.8 ± 1.3	18.8 ± 1.1*	17.7 ± 1.1*	2.39	.41‡
kJ/kg/min	0.26 ± 0.01	0.24 ± 0.01	0.23 ± 0.01	2.04	.34
5.5 km/h					
kJ/min	24.9 ± 1.5	24.2 ± 1.5	22.5 ± 1.3*	4.37	.69†
kJ/kg/min	0.31 ± 0.01	0.31 ± 0.01	0.29 ± 0.01	2.95	.49†
6.5 km/h					
kJ/min	32.6 ± 2.0	31.7 ± 2.0	30.1 ± 1.9*	4.21	.62†
kJ/kg/min	0.40 ± 0.01	0.40 ± 0.01	0.39 ± 0.01	2.80	.47†

NOTE. Values are the mean ± SEM. Statistical significance was established from a 2-way ANOVA for repeated measures on 1 factor (time).

*Significantly different from the initial value, $P < .05$.

†$P < .05$, ‡$P = .06$: significant intrapair resemblance in the response to training.

Table 4 shows the effects of training and the training-genotype interaction on plasma thyroid hormones. The protocol reduced levels of plasma T_3, free T_3, and total T_4, whereas no significant change was noted for free T_4. Significant variation was observed between twin pairs, with less response variation within twin pairs as evidenced by a significant intraclass correlation and F ratio for total T_4, whereas changes in total T_3, free T_3, and free T_4 showed a tendency for a significant intrapair resemblance (F ratio > 2.0).

The net energy deficit and the body energy loss induced by regular exercise during the first and second half of the protocol are depicted in Fig 2. Despite the fact that the energy deficit attributable to exercise remained constant during these two periods, body energy loss was reduced by 29% during the second half of the training program ($P = .08$). It is of interest that body energy loss corresponded to 91% of the estimated energy deficit during the first half of the program, whereas this

Table 3. Effects of Exercise Training on NE Kinetics and Intrapair Resemblance in the Response to the Protocol

Parameter	Before	After	F Ratio	Intraclass Coefficient
NE concentration (pg/mL)	228 ± 19	176 ± 14*	1.12	.06
NE appearance (μg/min)	0.28 ± 0.03	0.18 ± 0.02*	0.82	−.10
NE clearance (L/min)	2.67 ± 0.16	2.57 ± 0.25	2.72	.45

NOTE. Values are the mean ± SEM. Statistical significance was established from a 2-way ANOVA for repeated measures on 1 factor (time).

*Significantly different from the initial value, $P < .01$.

Table 4. Effects of Exercise Training on Plasma Thyroid Hormones and Intrapair Resemblance in the Response to the Protocol

Parameter	Before	After	F Ratio	Intraclass Coefficient
Total T_3 (ng/dL)	123 ± 4	105 ± 4*	2.62	.45
Free T_3 (ng/dL)	2.0 ± 0.09	1.81 ± 0.10*	2.59	.44
Total T_4 (μg/dL)	7.4 ± 0.25	6.2 ± 0.24*	6.17	.72†
Free T_4 (ng/dL)	1.42 ± 0.04	1.33 ± 0.05	2.07	.35

NOTE. Values are the mean ± SEM. Statistical significance was established from a 2-way ANOVA for repeated measures on 1 factor (time).

*Significantly different from the initial value, $P < .01$.

†Significant intrapair resemblance in the response to training, $P < .01$.

proportion decreased to 65% during the second part of the protocol.

DISCUSSION

This study was part of our ongoing effort to determine the impact of exercise training on resting components of energy expenditure and of the role of genetic factors in the heterogeneity of response that is often reported. Specifically, we investigated the effects of training in a context in which energy intake was maintained at the pretraining level and training resulted in a substantial net energy deficit. In a previous study where a similar training program was tested under the same nutritional conditions, a mean decrease in RMR of about 5% was noted.[7] This decrease is comparable to that observed in the present study (8%). In addition, we found that the training-induced change in RMR did not occur at random, since a moderate

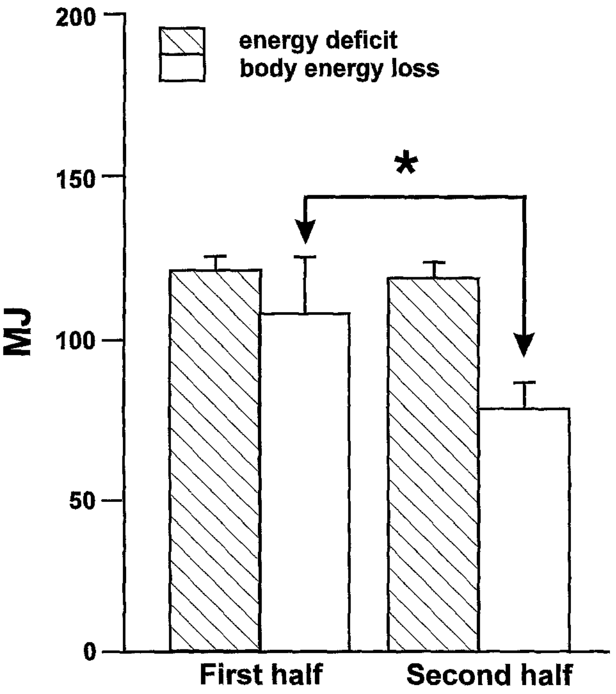

Fig 2. Estimated net energy deficit induced by regular exercise on a constant energy intake and body energy loss after the first and second half of the protocol. *$P = .08$. Values are the mean ± SEM.

within-pair resemblance was observed in the RMR response to training. This is concordant with previous data that we obtained in a training program of shorter duration.[10] This idea is also reinforced by the concordance between results obtained in the two laboratories involved in this study. Indeed, initial levels of RMR and changes induced by training were identical in the two laboratories, and the indicators of the genotype-training interaction effect on RMR, ie, the F ratio and intraclass correlation coefficient, were also comparable. In a context where the number of twin pairs was necessarily small because of the constraints of the protocol, the overall consistency between these observations reinforces the validity of this interaction effect on RMR.

The present study was not designed to document the mechanisms by which RMR may adapt to endurance exercise training associated with an energy deficit. However, the fact that a significant reduction in RMR was observed while no change in FFM occurred suggests that the loss of lean tissue was not responsible for this decrease. This observation is of relevance considering that FFM has been frequently shown to be the main determinant of RMR. An alternative explanation for the decrease in RMR associated with the training and negative energy balance protocol involves a possible role of SNS activity. Experimental evidence suggests that the increased RMR characterizing endurance-trained individuals under some circumstances is due to increased SNS activity.[11,12] In contrast, a large body energy deficit induces a decrease in sympathetic tonus.[17] It is thus possible that the reduction in RMR observed in this study is attributable to a net decrease in sympathetic activity, suggesting that the reducing effect of body energy loss may have predominated over the enhancing effect of exercise. This suggestion is concordant with the observation that the training program was associated with a decrease in NE concentration and appearance rate despite involving a large volume of exercise. The decrease in RMR observed at the end of the training program also agrees with the decrease in thyroid hormones, a finding that was also documented in our previous short-term study.[10]

A decrease in the energy cost of treadmill walking was also observed in response to the protocol. Depending on the walking speed, this reduction ranged from 8% to 15% when expressed in absolute terms. However, these variations did not randomly occur, since the within-pair resemblance in response to training was significant (Table 2). Thus, as for RMR, the energy cost of standardized activity was decreased by the protocol and seemed to be partly influenced by the genotype. The fact that exercise testing was performed using a treadmill whereas training was performed on a cycle ergometer probably does not invalidate the observation of a genotype-training interaction effect on the energy cost of walking. Since the two activities mainly rely on muscles of the lower limbs and walking is generally part of daily activities, it is thus unlikely that a treadmill exercise test would not reflect the impact of exercise training on a cycle ergometer on the energy cost of exercise.

The net exercise-induced energy deficit was constant during the protocol and corresponded to 123 and 121 MJ in the first and second half of the training period, respectively. In the first half of the protocol, there was almost no energy compensation, since the estimated body energy loss represented 91% of the net energy deficit. This contrasts with the second part of the protocol, where the body energy loss was reduced to 65% of the energy deficit. This indicates that compensations aimed at preserving body energy progressively attenuated the ability of the protocol to induce a negative energy balance and to provoke weight loss.

The experimental controls that were applied in this study allowed maintenance of constant energy intake and energy cost of training throughout the duration of the protocol. Thus, these variables cannot be considered as factors potentially explaining the physiologic compensations to attenuate body energy loss. Moreover, since RMR decreased during the first half of the training period but remained stable thereafter, its variation alone cannot explain the decrease in body energy loss that was observed during the second part of the study. The argument is also relevant for the TEM and energy cost of standardized walking, since their variations were nearly equivalent in the two parts of the protocol. Therefore, it would seem that these compensations might be at least partly explained by changes in the energy cost of nonexercise daily activities. In a recent study, Goran and Poehlman[18] reported such a decrease in nonexercise energy expenditure in elderly subjects based on doubly labeled water assessment of energy expended. A change in nonexercise daily activities potentially represents a mechanism by which one can compensate for exercise energy expenditure when the exercise stimulus becomes too demanding. The net result would then be an apparent state of increasing resistance to weight loss.

In summary, the results of this study show that when exercise training is associated with a substantial body energy loss, the RMR and energy cost of standardized activity are also decreased despite no change in FFM. This decrease in energy expenditure is associated with a reduction in SNS activity and thyroid hormones. However, there are substantial individual variations in the response of these variables to training that seem to be partly explained by a training-genotype interaction effect. As the duration of the training program increases, its impact on body energy loss progressively decreases.

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