【Short Communication】

CHANGES IN SUBSTRATE OXIDATION PERSIST OVERNIGHT AFTER A MARATHON RACE

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I . Introduction

After cessation of exercise, ones' energy requirements return back to resting levels, yet oxygen consumption remained elevated for several hours termed the excess post-exercise oxygen consumption (EPOC)¹⁾. Excess post-exercise oxygen consumption is consisted of a rapid (a large but short-lasting effect) and prolonged (a small but long-lasting effect) components²⁾.

The previous study has demonstrated that a significant elevation of resting metabolic rate (RMR) up to 48 h after an exercise session³⁾. The magnitude of the prolonged EPOC effect appears to be approximately 5-10% of the RMR⁴⁾. If one assumes from measurements at discrete time points that the effect is continuously protracted, it can be a sizeable percentage of the total energy cost of the exercise bout. In their review, Speakman and Selman⁴⁾ raised the possibility that more energy would be expended following the exercise than during the exercise event itself. However, little is known regarding the effect of exercise on energy metabolism during sleep, although the primitive but a monumental study published in 1910 reported an increase in sleeping metabolic rate after exercise⁵⁾.

Magnitude of EPOC after aerobic exercise is dependent on both the duration and intensity of the exercise²⁾. There is an exponential relationship between exercise intensity and the magnitude of

EPOC^{6,7)}. It appears that an intensity above 50-60% of maximal oxygen uptake is required to induce an EPOC lasting for several hours²⁾. The relationship between exercise duration and EPOC magnitude appears to be more linear, particularly at higher intensities²⁾. A marathon run (42.195 km) is one of the most extreme conditions for humans to stress physiological homeostasis. Total energy expenditure of marathon is estimated to be equal to or more than the energy expenditure during the whole day in most people⁸⁾. To our knowledge, the most exhaustive bout for any of the EPOC study was a 35 km run (70% of maximal oxygen uptake for 160 min)⁹⁾. However, no study is available regarding the effect of a marathon run on EPOC.

The purpose of the present study was, therefore, to examine the effect of a marathon run on EPOC during the post-exercise recovery period, including the sleep period using room-size human calorimeter.

II . Methods

A . Subjects

After approval from the University of Tsukuba Ethics Committee, eight trained athletes (7 males and 1 female; age 24.5 ± 0.7 yrs; height 168 ± 1.2 cm; weight 57.8 ± 1.6 kg; BMI 20.3 ± 0.4 kg/m², means $\pm$ SE.) gave their written informed consent to participate in this study. All individuals were free from cardiovascular diseases or abnormalities, and were not taking any medications or supplements known to influence lipid and carbohydrate metabolism.

B . Protocol

Each subject stayed twice in the room-size human calorimeter after completing a marathon race or sedentary day, at least 3 days apart in a randomised repeated-measures design. On the exercise trial (a marathon race) the subjects ran competitive marathon races in November–June, starting time of which ranged from 09:30–10:30. All subjects finished the race at 12:00–14:00. After the race, the subjects took shower and were instructed to avoid any additional exercise. Food intake before, during, and lunch after the race was according to individual habits, without any influence by the investigators. The subjects were transported to the laboratory at 18:00. On arrival, the subjects were asked to sit quietly in a chair and fitted with a telemetric heart rate monitor (LX-3230, Fukuda Denshi Co., Ltd., Tokyo, Japan). The subjects consumed an evening meal (3025 kJ with 16.3%, 22.1% and 61.6% energy provided from protein, fat and carbohydrate, respectively) in the laboratory at 19:00 and then the subjects entered the human calorimeter at 19:30. The subjects were instructed to remain quietly seated in a chair for remainder of the evening (sitting reading, writing, working at a computer or watching television) and the subjects went to bed at 23:00. On the following morning, the subjects awoke at 06:00 but they were asked to sit quietly in a chair. The subjects consumed a meal for breakfast (2390 kJ with 17.7%, 22.6% and 59.7% energy provided from protein, fat and carbohydrate, respectively) at 07:00 and thereafter the subjects were asked to remain quietly seated in a chair until 10:00. On the control trial (sedentary day), the subjects were reported to the laboratory at 18:00 and followed the same time-schedule as the day after the race. We did not standardise diet between the race day (i.e. prior to or during the race) and at an equivalent time points in the control trial since our study design was a randomised crossover study and controlling diet was practically impossible. However, the subjects consumed identical amounts of the same food and drink for an evening meal and breakfast on

both trials.

C . Human calorimeter

Human calorimeter was used so as to measure oxygen consumption continuously during the post-exercise period, inducing the sleep period. The air-tight chamber measures $2.00 \times 3.45 \times 2.10$ m having an internal volume of 14.49 m^3 (FHC-15S, Fuji Medical Science Co., Ltd., Chiba, Japan). It has one entrance door and an air lock for food supply. Air in the chamber was pumped out at a rate of 70 L/min. Temperature and relative humidity of in-coming fresh air were controlled at $25.0 \pm 0.5^\circ\text{C}$ and $55.0 \pm 3.0\%$, respectively. The room air was mixed sufficiently by fans. The chamber was furnished with an adjustable hospital bed, a small desk, a chair, a wash basin and a toilet.

Concentration of oxygen and carbon dioxide in out-going air were measured with high precision by online process mass spectrometry (VG Prima δ B, Thermo Electron Co., Cheshire, UK). At every minute, oxygen consumption and carbon dioxide production rates were calculated based on noise suppression and trend identification algorithm by Henning et al.¹⁰⁾. Energy expenditure, carbohydrate oxidation and fat oxidation were calculated according to the equations described by Weir¹¹⁾ and Frayn¹²⁾, respectively.

D . Statistical analysis

Data are presented as means $\pm$ SE. Values shown are one-hour averages based on data recorded at one-minute interval. Two-factor (trial $\times$ time) ANOVA with repeated measures was used to examine differences the two trials over time for oxygen consumption, carbon dioxide production, energy expenditure, respiratory quotient (RQ), cumulative carbohydrate oxidation and cumulative fat oxidation. The changes in energy expenditure and RQ were calculated by subtracting energy expenditure/RQ on the exercise trial from the value on the control trial. Statistical significance was accepted at the 5%

level.

III . Results

All subjects completed the marathon ($2^{\circ}30'06''$ – $3^{\circ}21'48''$ for male and $3^{\circ}49'49''$ for female ; the average time to complete the marathon distance was $2^{\circ}55'51''$ for all subjects). Six male subjects completed the race under 3 h.

There was no difference in oxygen consumption from 20:00 (=7 h post-exercise) to 10:00 the following day (=21 h post-exercise) between the exercise (total oxygen consumption 185.5 ± 3.5 L) and control (total oxygen consumption 173.0 ± 5.6 L) trials. However, oxygen consumption, particularly

until bed time, tended to be higher in the exercise trial compared with the control trial but this difference was not statistically significant (Fig. 1a). Total energy expenditure from 20:00 (=7 h post-exercise) to 10:00 the following day (=21 h post-exercise) did not differ between the exercise (3877 ± 151 kJ) and control (3713 ± 175 kJ) trials. Heart rate from 20:00 (=7 h post-exercise) to 10:00 the following day (=21 h post-exercise) was higher (main effect of trial, $P=0.002$, main effect of time, $P<0.0005$, trial x time interaction, $P=0.002$) in the exercise trial (time-averaged values 56 ± 2 beats/min) compared with the control trial (time-averaged values 51 ± 2 beats/min) (Fig. 1b). Respiratory quotient from 19:30 to 10:00 was lower (main effect of trial, $P=0.001$, main effect of time, $P<0.0005$) in the exercise trial (time-averaged values 0.840 ± 0.009) compared with the control trial (time-averaged values 0.907 ± 0.006).

The cumulative carbohydrate oxidation were lower (main effect of trial, $P=0.008$, main effect of time, $P<0.0005$) in the exercise trial (124.6 ± 9.8 g/14 h) compared with the control trial (173.6 ± 9.8 g/14 h). The cumulative fat oxidation were higher (main effect of trial, $P=0.001$, main effect of time, $P=0.005$) in the exercise trial (50.5 ± 2.8 g/14 h) compared with the control trial (27.2 ± 1.4 g/14 h).

IV . Discussion

Excess post-exercise oxygen consumption estimated by subtracting baseline measurement over recovery period, practiced in some previous studies^{13,14}), suffers limitation due to the circadian variation in RMR. Withers et al.⁹) compared RMR with or without a preceding 35 km run and accounted for the circadian variation in RMR. Since RMR was measured using mouthpiece and nose-clip in the study by Withers et al.⁹), their measurements were at discrete time points (0.25, 0.5, 0.75, 1, 2, 3, 4, 5, 6, 7, 8, 24 and 48 h after the exercise) and could not continuously monitor RMR during sleep.

Fig. 1. Oxygen consumption (a) and heart rate (b) after the exercise (a marathon run) and control (sedentary day) trials. Data are presented as means $\pm$ SE. Note. Since all runners finished the race at 12:00–14:00, mean value from 20:00 to 21:00 was plotted at 7–8 h post-exercise.

The present study continuously monitored RMR with or without a preceding marathon run by adopting room-size human calorimeter. Our findings demonstrate that oxygen consumption was similar throughout the day including the sleep period between trials. However, the present study had a limitation. We did not measure oxygen consumption immediately after the marathon run (i.e. a practical reason) or at an equivalent time during the control trial, limiting our ability to examine the immediate effect of the marathon run on post-exercise energy metabolism. Nonetheless, EPOC during immediate 8 h after completion of 35 km running was reported to be $32.3 \text{ L} (+23.7\% \text{ of RMR})^9$. Our continuous measurement began 6–8 h after the race, and accumulated EPOC during the next 14 h including sleep and breakfast was $11.0 \pm 19.4 \text{ L} (+4.7\% \text{ of RMR})$. Thus, the EPOC is greatest immediately following the termination of exercise, and progressively declines thereafter. Speakman and Selman⁴) therefore usefully separated EPOC into two phases, the short-term effect ($<2 \text{ h}$) and the more long-lasting but a smaller effect.

To get a whole picture of enhanced energy expenditure after a long distance running, energy expenditure and RQ after the 35 km run presented in table 2 of the previous report by Withers et al.⁹) were superimposed on the present results (Figs. 2a and 2b). Combined data from the previous study by Withers et al.⁹) and the present findings indicate that the energy expenditure became indistinguishable within 24 h after the running. Enhanced energy expenditure during the 8 h immediately after the race (465 kJ) and the next 14 h (172 kJ) totaled $\sim 637 \text{ kJ}$.

If a marathon run is performed at an intensity close to 65% of maximal oxygen uptake, the estimated total energy expenditure would be 10,000–12,000 kJ ($=2400\text{--}2800 \text{ kcal}$)⁸). Touminen et al.¹⁵) reported 6% increase in basal metabolic rate on the morning after the marathon ($\sim 12 \text{ h}$ post-exercise).

Fig. 2. The changes in energy expenditure (a) and respiratory quotient (RQ) (b) after a marathon run. Note. Since all runners finished the race at 12:00–14:00, mean value from 20:00 to 21:00 was plotted at 7–8 h post-exercise. The changes in energy expenditure and RQ were calculated by subtracting energy expenditure/RQ on the exercise trial from the value on the control trial. For comparison, the data were superimposed on reported values⁹) after 35 km running ($\circ$).

Taken together, the available evidence does not support the notion that more energy would be expended following the exercise than during the exercise event itself.

In the present study, reduction of RQ after the race continued during the night. The cumulative carbohydrate oxidation was suppressed, while that of the cumulative fat oxidation was enhanced during the night after the race. If all the data from the previous study by Withers et al.⁹) (a 35 km run) and

the present study (a marathon run) were combined and linearly regressed against time after exercise, it would be speculated that RQ returned to resting values ~ 55 h after the exercise. Therefore, the present study suggests that energy expenditure returned ~ 24 h after the marathon run but changes in substrate oxidation seemed to continue one more day. It has been reported that a significant fraction of muscle glycogen ($56 \pm 4\%$) is utilised during the marathon, and it takes more than 2 days to replenish it¹⁶⁾. Decrease in carbohydrate oxidation on the exercise trial in the present study may permit more of the exogenous glucose to be used for repletion of glycogen.

EPOC is the sum of many underlying mechanisms, some of which are still unknown. In addition to glycogen repletion, elevated post-exercise concentration of free fatty acid (0–2 h), β -hydroxybutyrate (0–1 h and 8 h) and acetoacetate (0–3 h) favour lipid over glucose oxidation⁹⁾. Newsholme¹⁷⁾ suggested slow component of EPOC observed several hours after prolonged exercise is attributable to substrate cycling. Indirect calorimetry used in studies on EPOC estimates net oxidation of substrate, i.e. rate of gluconeogenesis and lipogenesis were subtracted from carbohydrate and fat oxidation. Nature of EPOC remains to be dissected with more analytical techniques such as stable isotope and functional magnetic resonance imaging.

In conclusion, the present study demonstrates that oxygen consumption continuously measured overnight did not differ between the exercise (a marathon race) and control (sedentary day) trials. Our findings also show that changes in substrate oxidation persist overnight after the marathon race.

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