

ORIGINAL ARTICLE

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Increase in energy cost of running at the end of a triathlon

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Abstract The purpose of the present study was to verify the increase in energy cost of running at the end of a triathlon. A group of 11 trained male subjects performed a triathlon (15-km swimming, 40-km cycling, 10-km running). At least 1 week later the subjects ran 10-km as a control at the same pace as the triathlon. Oxygen uptake ($\dot{V}O_2$), ventilation (\dot{V}_E) and heart rate (HR) were measured during both 10-km runs with a portable telemetry system. Blood samples were taken prior to the start of the triathlon and control run, after swimming, cycling, triathlon run and control run. Compared to the control values the results demonstrated that triathlon running elicited a significantly higher ($P < 0.005$) mean $\dot{V}O_2$ [51.2 (SEM 0.4) vs 47.8 (SEM 0.4) $\text{ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$], \dot{V}_E [86 (SEM 4.2) vs 74 (SEM 5.3) $\text{l} \cdot \text{min}^{-1}$], and HR [162 (SEM 2) vs 156 (SEM 1.9) $\text{beats} \cdot \text{min}^{-1}$]. The triathlon run induced a greater loss in body mass than the control run [2 (SEM 0.2) vs 0.6 (SEM 0.2) kg], and a greater decrease in plasma volume [14.4% (SEM 1.5) vs 6.7% (SEM 0.9)]. The lactate concentrations observed at the end of both 10-km runs did not differ [2.9 (SEM 0.2) vs 2.5 (SEM 0.2) $\text{m} \cdot \text{mol} \cdot \text{l}^{-1}$]. Plasma free fatty acids concentrations were higher ($P < 0.01$) after the triathlon than after the control run [1.53 (SEM 0.2) to 0.51 (SEM 0.07) $\text{mmol} \cdot \text{l}^{-1}$]. Plasma creatine kinase concentrations rose under both conditions from 58 (SEM 12) to 112 (SEM 14) $\text{UI} \cdot \text{l}^{-1}$ after the triathlon, and from 61 (SEM 7) to 80 (SEM 6) $\text{UI} \cdot \text{l}^{-1}$ after the control run. This outdoor study of running economy at the end of an Olympic distance triathlon demonstrated a decrease in running efficiency.

Key words Triathlon · Oxygen uptake · Ventilation · Biochemical variables · Plasma volume

Introduction

During a triathlon race, the running that follows swimming and cycling appears to be the most difficult segment to complete. There is evidence that running subsequent to cycling seems more stressful than running on its own. This subjective perception has motivated previous studies to investigate possible decreases in the energy efficiency of running after cycling. The first data obtained by Boone and Kreider (1986) has shown that bicycle exercise at 80% of maximal heart rate increases the metabolic energy cost of subsequent running. Kreider et al. (1988) have confirmed this decrease in metabolic efficiency during a simulated triathlon session. After an 0.8-km swim and 75-min of cycling on a laboratory cycle-ergometer a decrease in running economy was observed, expressed by a higher oxygen uptake ($\dot{V}O_2$) required to maintain the same average running speed on the treadmill as during the control trial. These results agree with other investigations that have demonstrated a slow rise in $\dot{V}O_2$ during constant intensity prolonged exercise (Whipp and Wasserman 1972; Hagberg et al. 1978; Casaburi et al. 1987; Bahr et al. 1991; Poole et al. 1991). This observation of a lesser running economy during running at the end of a triathlon raises two questions, one concerning the physiological and biomechanical factors responsible for the increase in the cost of running and the other concerning the consequences for performance.

As reviewed by Morgan et al. (1989) numerous investigations have demonstrated that a lengthy list of physiological and biomechanical variables might account for the variability in running economy. It has been proposed that muscle glycogen depletion, mechanical damage to muscle fibres, changes in stride length, altered substrate utilization, increased work of

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breathing and increased body temperature are responsible for the enhanced cost of running which occurs with the duration of exercise (Hagberg et al. 1978; Hanson et al. 1982; Casaburi et al. 1987; Dick and Cavanaugh 1987; Morgan et al. 1989; Bosch et al. 1990). But clear support for the respective importance of each variable has been said to be lacking (Poole et al. 1991) The influence of running economy on inter-individual differences in performance has attracted attention (Morgan et al. 1989). It has been suggested that differences in performance of runners with the same aerobic power could be attributed to variability in running economy (Costill and Winrow 1970), but the effect of a decreased running economy on performance at the end of a triathlon has not been documented.

The principal purpose of this investigation was to verify the decrease in running economy during triathlon run, using a portable telemetry system for the measurement of $\dot{V}O_2$. A second objective was to examine the relationship with several physiological variables such as \dot{V}_E heart rate, hydration, blood substrates, and muscle enzyme release.

Methods

Subjects

Ten male triathletes and one endurance trained subject participated in this research study. All the subjects were familiar with the experimental protocol and signed informed consent statements. The mean age was 29 (SD 3) years with a range of 20–44 years. The mean body mass was 67 (SD 2.6) kg. The mean maximal oxygen uptake measured on a cycle-ergometer with an automated open circuit system (Sensors Medics Horizon, U.S.A) was 65.3 (SEM 6) $\text{ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$. The subjects had been training for triathlons for an average of 2.5 (SEM 1.3) years. The subjects completion times for a middle distance triathlon (1.5-km swimming, 40-km cycling, 10-km running) ranged from 2 h 10 min to 2 h 43 min. At 1 month before the beginning of the experiment, nine subjects among the ten involved in the study ran a 10-km road race with a mean time of 37 min 40 s (SD 1 min) 40 s. Physiological and training data demonstrated that these subjects were representative of previously reported triathlon populations (Kreider et al. 1988; O'Toole et al. 1989).

Experiment design

The subjects performed two tests, separated by at least 1 week during which moderate training, with only one session a day was done.

The first test was a simulated triathlon including 1.5-km swimming, 40-km cycling and 10-km of running completed at a racing pace. The swimming was in a swimming pool with a water temperature of 24°C. The cycling was on a road circuit previously used for triathlon competition. The foot race was on a 400-m tartan paved circular track. Running time was measured every 400 m with a manual chronometer. The main difference between real competition and this test was the increased transition times: 3 min were needed for blood sampling between swimming and cycling, and 5 min for the installation of telemetry systems between cycling and running. Before the start, at each transition and at the end of the run the subjects were weighed on an electronic balance.

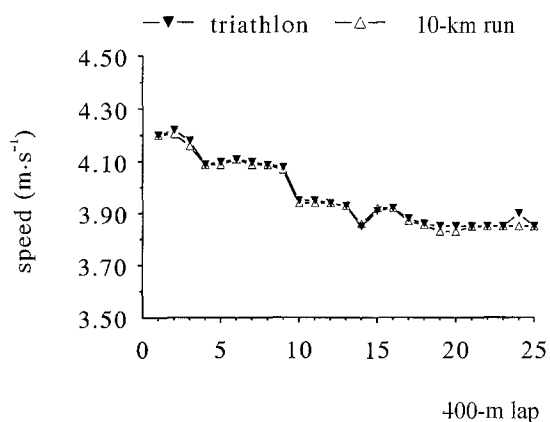


Fig. 1 Mean changes in speed of running ($\text{m} \cdot \text{s}^{-1}$) for the 10-km control run and 10-km triathlon run measured for each 400-m lap

The second test was a control test in the form of 10-km run between 7 and 10 days after the simulated triathlon. The same running pace as in the triathlon run was indicated by a sound signal every 400 m, at the time achieved by each subject during the triathlon run. As the subjects were trained runners, they were able to monitor running pace with a maximal error of 1 each 400 m. The start was preceded by 20 min of warm-up consisting of alternately running and stretching. The level of body hydration was standardised by drinking 250-ml water before swimming and during cycling and 150 ml before running during the triathlon. Only 250-ml water was ingested before the 10-km control race.

All the tests were performed between April and June in France. The range of outdoor temperature was 17°C–24°C. The mean completion times for all subjects were 24 min 15 s (SD 1) min for swimming, 73 (SD 4) min for cycling and of 42 min 12 s (SD 2) min for running (the mean time for the nine subjects who had previously completed a 10-km road run was 42 min 17 s) The mean total time to perform the simulated triathlon without the transitions was 2 h 19 min 25 s. The mean total time for the control run was of 42 min 18 s (SD 2) min. Figure 1 indicates the mean speed for each 400-m lap. A gradual decrease in speed was observed from the first lap to the last with less than 0.01% difference between the triathlon and control run.

Physiological measurements

Expired gas was measured continuously during the two 10-km runs (triathlon and control) with a Telemetric System K₂ (Cosmed Milan, Italy). The cosmed K₂ is a portable telemetry system for $\dot{V}O_2$ measurement in the field. This system has been previously described (Dal Monte et al. 1989; Kawakami et al. 1992; Lucia et al. 1993; Bigard and Guezennec 1995). It consists of a photo-electric turbine flowmeter attached to a face mask which calculated the \dot{V}_E to body temperature and pressure, saturated conditions. The expired air has analysed by a polarographic oxygen sensor for measurement of the oxygen fraction in expired gas (F_{EO_2}). The $\dot{V}O_2$ was calculated using \dot{V}_E and F_{EO_2} assuming that the respiratory exchange ratio (R) = 1 using the equation $\dot{V}O_2 = K \cdot \dot{V}_E(F_{I}O_2 - F_{E}O_2)$, where K is a constant, $F_{I}O_2$ is the oxygen fraction in inspired gas.

The major methodological problem was the error introduced by a fixed R value. Several validations have been conducted (Kawakami et al. 1992; Lucia et al. 1993; Peel and Utsey 1993; Bigard and Guezennec 1995). From a comparison with the direct measurement of carbondioxide fraction in expired gas (F_{ECO_2}), the maximal error introduced into the calculation of $\dot{V}O_2$ by an R change from 0.80 to 1.20 is of 4%. The K₂ system has been shown to be accurate for $\dot{V}O_2$ measurement during exercise intensities up to 85% of maximal values.

Preliminary experiments conducted outdoors under the conditions of the study presented here have demonstrated other methodological limitations to this method. The polarographic electrode was sensitive to temperature. The gain of the electrode remained stable only with outdoor temperatures between 14°C and 30°C, without rain or wind for temperatures near 14°C.

Heart rate was monitored by a Sport Tester PE 3000 (Polar Electro, Finland).

Installation of the K₂ system, heart rate monitoring and calibration procedures took a mean time of 5 min before each 10-km run.

Blood sampling

A 10 ml sample of blood was taken from the antecubital vein in the sitting position at the following times, the subjects staying only 2 min in this position in order not to induce a posture related shift in plasma volume (PV). (a) prior to the start of the triathlon, (b) after the swimming stage, (c) after the bicycle stage, (d) at the end of the running stage, (e) prior to the start of the 10-km control race, (f) at the end of the 10-km control race.

Analysis

Haematocrit (Hct) was determined with a micro-haematocrit centrifuge and corrected for 4% plasma trapped with packed cell. Percentage changes in plasma volume (% ΔPV) were calculated from values of Hct according to the method of Van Beaumont (1973):

$$\% \Delta PV = \frac{100}{100 Hct_{pre}} \times \frac{100(Hct_{pre} - Hct_{post})}{Hct_{post}}$$

where Hct_{pre} represents the pre-exercise Hct ratio and Hct_{post} represents the post-exercise value.

Blood lactate, and creatine kinase (CK) concentrations were assayed using a standard kit (Boehringer Mannheim, Meylan, France). Plasma free fatty acid (FFA) concentrations were determined using a commercial kit (Biomerieux Lyon, France).

Statistical analysis

Statistical variations between the control run and triathlon physiological responses were determined by ANOVA for repeated measures. A post hoc Tukey test was used to identify specific mean differences when a significant ratio was observed. Data were considered significantly different when the probability of error was 0.05 or less.

Results

Ventilation parameters

Figures 2 and 3 give the mean $\dot{V}O_2$ and ventilation responses observed during the 10-km run of the triathlon and of the control session. Each point indicates the mean value obtained for each 400-m lap. The mean $\dot{V}O_2$ for triathlon running [51.2 (SEM 0.4) ml · min⁻¹ · kg⁻¹, i.e. 3.43 l · min⁻¹] was significantly higher than the $\dot{V}O_2$ for mean control running [47.8 (SEM 0.4) ml · min⁻¹ · kg⁻¹, i.e. 3.18 l · min⁻¹ ($P < 0.05$). The mean oxygen cost (OC) was calculated from these

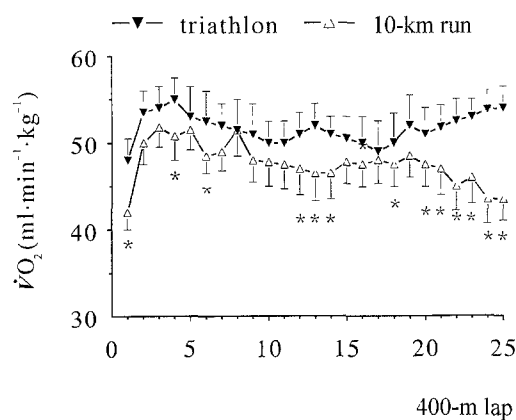


Fig. 2 Oxygen consumption ($\dot{V}O_2$) during the 10-km control run and 10-km triathlon run.

* Significant differences ($P < 0.05$)

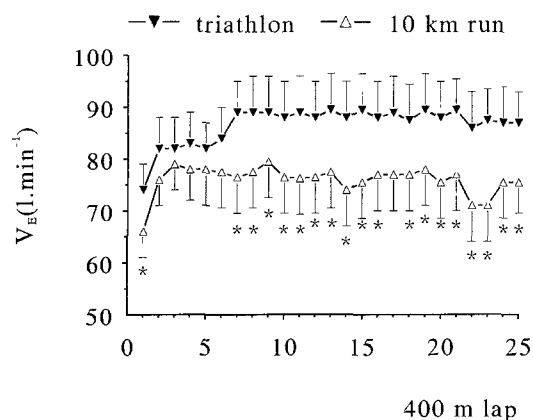


Fig. 3 Ventilation (\dot{V}_E) during the 10-km control run and 10-km triathlon run.

* Significant differences ($P < 0.05$)

values using the equation:

$$OC = \frac{\dot{V}O_2 \text{ (ml}^{-1} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) \times 60}{\text{Speed (km} \cdot \text{h}^{-1})}$$

The OC was 215 ml O₂ · kg⁻¹ · km⁻¹ during the triathlon run, and 199 ml O₂ · kg⁻¹ · km⁻¹ during the control run.

Triathlon running elicited a significantly higher mean \dot{V}_E [86 (SEM 4.2) l · min⁻¹] than the control run [74 (SEM 5.3) l · min⁻¹ ($P < 0.05$). The analysis for each lap indicated a significantly higher \dot{V}_E during the triathlon run at the start, and from the 7th lap to the end.

The mean heart rates measured for each lap (Fig. 4) only show statistical differences for laps 11, 12, 16, 20, 22, 23, 25. The mean heart rate during the triathlon [162 (SEM 2.1) beats · min⁻¹] was significantly higher than the mean heart rate [156 (SEM 1.9) beats · min⁻¹] resulting from the 10-km run.

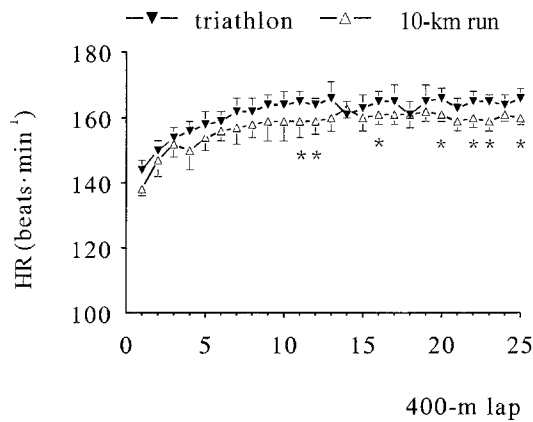


Fig. 4 Heart rate (HR) during the 10-km control run and 10-km triathlon run

* Significant differences ($P < 0.05$)

Hydration parameters

Table 1 shows that the triathlon run induced a substantially greater body mass loss [2 (SEM 0.2) kg] than the 10-km control run [0.6 (SEM 0.2) kg] ($P < 0.01$). The analysis of the triathlon indicated that the major part of body mass loss occurred during running.

The Hct changes indicated a progressive haemoconcentration throughout the triathlon, with a significantly higher mean value at the end compared to the 10-km run ($P < 0.05$). The difference in pre-post-stage Hct was used to calculate ΔPV . The largest decrease in PV had occurred after the first stage of the triathlon, the swimming stage. During this stage PV decreased by 9.15% from its resting value. It had only decreased from rest by 6.5% after cycling, so that a slight haemodilution occurred after cycling, relative to post-swimming. Compared to rest PV had decreased by 14.43% at the end of running. The control 10-km run only induced a 6.7% decrease in PV.

Metabolic parameters

During the triathlon, the maximal blood lactate concentration increase was observed after swimming, with a mean increase of $2.8 \text{ mmol} \cdot \text{l}^{-1}$ above the resting value (Table 1). The same lactate values were observed after both running trials [2.9 (SEM 0.2) vs 2.5 (SEM 0.2) $\text{mmol} \cdot \text{l}^{-1}$]

Blood glucose significantly increased after swimming ($P < 0.05$) and remained slightly higher than the rest value at the end of the triathlon.

Discussion

The main finding of this study was the confirmation of the increase in OC during the last stage of a triathlon run in a natural environment. The initial study of Kreider et al. (1988) performed during a simulated triathlon with treadmill running as the last stage has reported a mean increase of $440 \text{ ml} \cdot \text{min}^{-1}$ or $6 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ for $\dot{V}O_2$ and of 12.91 for \dot{V}_E at the end of the triathlon run compared to the control run. The data reported here showed an increase in OC of $250 \text{ ml} \cdot \text{min}^{-1}$ or $3.5 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ with a 12 l increase in \dot{V}_E . The fractions of maximal $\dot{V}O_2$ used during the triathlon and control runs were 78% and 69% in the study of Kreider et al. (1988) and 78% and 73% in the present study. This slight difference between track and treadmill running would indicate that under the latter conditions the wind resistance would be so small that it would have little effect on running economy as has been shown by Pugh (1971).

The phenomenon of the increased cost of running at the end of a triathlon is related to the mechanisms influencing running economy. Numerous investigations have shown an upward $\dot{V}O_2$ drift during long duration whole body exercise at higher intensities (Whipp and Wasserman 1972; Hagberg et al. 1978; Casaburi et al. 1987; Brueckner et al. 1991). A number

Table 1 Changes in body mass, haematocrit, blood lactate, free fatty acid (FFA) and creatine kinase (CK) concentration observed after a triathlon or a 10-km control run. PV Plasma Volume

	Mass (kg)		Haematocrit		PV (%)	Lactate ($\text{mmol} \cdot \text{l}^{-1}$)		FFA ($\text{mmol} \cdot \text{l}^{-1}$)		CK ($\text{UI} \cdot \text{l}^{-1}$)		
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Before triathlon	67	2.6	46.1	0.6		1.2	0.1	0.15	0.03	58	12	
After swimming	66.2	2.6	48.5	0.8 ^a	9.15	1.2	4	0.4 ^a	0.31	0.05 ^a	88	12 ^a
After cycling	66	2	47.8	0.7 ^a	6.5	0.8	2.8	0.4 ^a	0.97	0.15 ^a	87	12 ^a
After triathlon	65	1.5	50	1 ^a	14.43	1.5	2.9	0.2 ^a	1.53	0.20 ^a	112	14 ^a
Before 10-km run	67.2	2.6	46.3	0.8			1.2	0.16	0.16	0.04	61	7
After 10-km run	66.6	2.6	47	1 ^a b	6.7	0.9	2.5	0.2 ^a	0.51	0.07 ^{a, b}	80	6 ^{a, b}

^a Indicates that the result differs significantly from resting values, $P < 0.05$

^b Indicates that the result differs significantly between post-triathlon and 10-km run, $P < 0.05$

of mechanisms have been hypothesized to account for this rise. It has been suggested that a first group of factors could influence running economy by its action upon biomechanical variables such as stride length (Morgan et al. 1989). By using a comparative approach between mammals of different size Kram and Taylor (1990) have shown that the cost of running is determined by the cost of supporting mass and by the time course of force application. This data underline the fact that it is the muscles which generate the force that determines the body's metabolic rate. An increased metabolic rate has been associated with muscle fatigue (Sejersted and Vollestad 1992). Dick and Cavanaugh (1987) have proposed that an upward drift in $\dot{V}O_2$ during low intensity downhill running is related to skeletal muscle damage that occurs with this form of exercise (Guezennec et al. 1986b; Iwane 1987). These authors have suggested that damaged fibres are replaced by additional motor units for force generation but the damaged fibres continue to use oxygen.

The higher elevation in plasma CK concentration observed at the end of the triathlon run compared to the control run would suggest that CK release could be an indirect index of muscle damage. Fibres that are not generally used during an isolated running event may be used in a triathlon run and become damaged because they are unaccustomed to the stress placed on them.

A second group of factors has been suggested as being related to metabolic events. It has been shown that muscle glycogen depletion which occurs in active muscle fibres during prolonged exercise is able to modify muscle metabolism and efficiency (Armstrong et al. 1977). Fat oxidation has been found to replace progressively carbohydrate fuel for muscle metabolism during prolonged exercise (Bulow 1988). Higher FFA concentration at the end of the triathlon compared to the control has been shown to be directly related to enhanced lipid mobilization (Van Rensburg et al. 1986). Carlson (1967) has shown that most of the energy demand during prolonged exercise in humans is supplied by the increased mobilization of fatty acids, so one could assume that at the end of the triathlon the proportion of lipid being used is higher than at the end of the control run. In theory, lipid oxidation produces less energy than equimolecular carbohydrate oxidation, so part of the $\dot{V}O_2$ drift observed here at the end of the triathlon could have been related to increased dependency on fat combustion.

The last group of factors which could influence running economy has been related to thermal stress and dehydration. The extent of dehydration has been found to be more severe in the triathlon than in comparable endurance events (Van Rensburg et al. 1986). The total 14.4% decrease in PV is consistent with the 14.3% decrease reported by MacNaughton (1989) after a sprint triathlon, and slightly higher than the 9.57% measured by Long et al. (1990) after running an Olympic triathlon distance.

Most of the dehydration occurs during running, as has been previously shown (Guezennec et al. 1986a), and is related to sweating and thermal stress; this could influence running economy.

On the one hand, thermal stress could be involved in the upward drift in \dot{V}_E observed here (Hagberg et al. 1978), since it has been found that one third of the rise in $\dot{V}O_2$ during constant intensity exercise can be attributed to hyperventilation (Hagberg et al. 1978; Casaburi et al. 1987). The increase in \dot{V}_E of 16% was of greater magnitude than $\dot{V}O_2$ (7%). The \dot{V}_E drive was higher than the need for O_2 delivery to the alveoli. The determinant of hyperventilation during prolonged exercise has been attributed to the elevation in core temperature, lactacidosis and circulating catecholamines (Hanson et al. 1982). The lactate changes observed here would indicate that there was no contribution of anaerobic metabolism during the relatively steady-state cycling or swimming as reported by O'Toole et al. (1989) and all the energy would have been provided aerobically. In these conditions metabolic acidosis is not a factor in hyperventilation. Casaburi et al. (1987) have demonstrated a significant relationship between the size of change in the \dot{V}_E and the $\dot{V}O_2$ drift.

On other hand, dehydration is responsible for enhanced cardiac work. The increase in heart rate observed here would have compensated for the decrease in stroke volume and maintained the same cardiac output (Kreider et al. 1988). The $\dot{V}O_2$ of heart has been shown to account for only 3% of the whole body metabolic requirement during maximal exercise (Armstrong et al. 1987), so that a small change in cardiac function could not have been responsible for a marked effect on running economy.

Considering both the higher heart rate and the ventilatory level for the same speed of running, there is a reasonable possibility that these factors contributed to the increased $\dot{V}O_2$ measured during the triathlon run (Shephard 1966). The question is how to evaluate that contribution. Voluntary hyperpnoea, which increases \dot{V}_E from 70 to 100 l·min⁻¹, has been found to enhance $\dot{V}O_2$ by 122 ml·min⁻¹ (Coast et al. 1993). In the conditions studied here, the increase in mean \dot{V}_E was probably on its own not able to explain the 250 ml·min⁻¹ increase in $\dot{V}O_2$ during the triathlon run. The increased cardiac and ventilatory work would not have been sufficient to produce the 8% higher energy cost of whole body exercise observed here.

The last point to discuss is the influence of the enhanced cost of running on performance during a triathlon. Data obtained on runners differing in running economy after a training period which influenced the cost of running have shown that running economy is a strong determinant of the performance (Costill and Winrow 1970; Daniels et al. 1978). In the conditions studied here, one could obtain an indirect estimation of the effect on performance by comparing the mean performance achieved by the nine subjects in the study

during a 10-km road race with the 10-km time completed during the experimental triathlon. The difference was 5 min. But as psychological motivation is higher during a real competition and the respiratory system is able to impede maximal performance during an experimental triathlon, it is probable that the differences between a competitive 10-km run and a triathlon run would be smaller.

Conclusion

This outdoor study of running economy at the end of middle distance triathlon confirmed the increase in energy cost during running. The simultaneous effect of a triathlon run on muscle enzyme release, dehydration, \dot{V}_E , and heart rate would indicate that the lower running economy is the result of several factors. Further studies are needed to measure each contribution and to examine whether inadequate water and glucose supplies could reduce mechanical efficiency at the end of a triathlon.

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