

## METABOLIC EFFECTS

### Effect of a Small Dose of Alcohol on the Endurance Performance of Trained Cyclists

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**Abstract — Aim:** The aim of this study was to investigate the effect of an acute small ethanol (EtOH) dose (0.5 ml EtOH/kg fat-free mass, combined with carbohydrate) in a drink on endurance performance of trained cyclists. **Methods:** Thirteen well-trained male cyclists took part in this study. A 60-min cycling endurance performance test (time trial) was performed in a calorimetric chamber after drinking an EtOH (30 ± 1.8 ml) or a non-EtOH control (C) drink. **Results:** Overall, EtOH induced a significant decrease in the average cycling power output (PO) (EtOH: 233 ± 23 W versus C: 243 ± 24 W,  $P < 0.01$ ). The time course of mechanical PO showed an early decrease during the EtOH trial as compared to C ( $P < 0.01$ ). Due to the lower PO, oxygen consumption, carbon dioxide production and glucose oxidation were significantly lower ( $P < 0.05$ ) as compared to C. Relative to PO, heart rate response and ratings of perceived exertion (RPE) were increased by EtOH as compared to C ( $P < 0.05$ ). In contrast, EtOH did not influence gross work efficiency, glycaemia and blood lactate concentration. **Conclusions:** These results show that the acute low dose of EtOH decreased endurance performance. An increase of cardio-vascular strain and psychobiological mechanisms may explain this decrease of endurance performance.

## INTRODUCTION

Alcohol is often part of an everyday diet in western countries, represents 0–10% of the average daily energy intake and is mainly absorbed as ethanol (EtOH). Although it is not a nutrient, EtOH provides energy (~7 kcal/g) and has multiple effects on metabolic function (El-Sayed *et al.*, 2005; Maughan, 2006; Shirreffs and Maughan, 2006). Even though athletes are generally advised to refrain from consuming EtOH (American College of Sports Medicine, 1982), dietary surveys show that EtOH contributes 0–5% to the total daily energy intake of athletes (Maughan and Burke, 2002) and that their average daily intake of EtOH is similar to that of the general population (Gutgesell and Timmerman, 1998; Burke and Maughan, 2000). Moreover, athletes may be at a higher risk of hazardous EtOH consumption (O'Brien *et al.*, 2005), with binge drinking being linked to athletic participation (Gutgesell *et al.*, 1996; Nelson and Wechsler, 2001). Although EtOH is considered to be deleterious to endurance performance, anecdotal observations and some reports indicate that some athletes seldom consume EtOH prior to training sessions and/or competition (Maughan, 2006).

EtOH may be deleterious to high intensity endurance performance through different mechanisms. First, even at low levels, EtOH impairs psychomotor skills such as reaction time, hand–eye coordination, accuracy, balance and complex skilled tasks (Williams, 1991; Burke and Maughan, 2000). Also, and perhaps more importantly for endurance performance, EtOH has been shown to influence CHO metabolism: it inhibits liver glucose output during exercise (Jorfeldt and Juhlin-Dannfelt, 1977; Juhlin-Dannfelt *et al.*, 1977a; Kendrick *et al.*, 1993; Heikkonen *et al.*, 1998) and decreases muscle glucose uptake (Jorfeldt and Juhlin-Dannfelt, 1978). Muscle glycogen breakdown is also increased at rest and its pattern of utilization is modified during exercise according to the muscle-fibre-type composition (Juhlin-Dannfelt *et al.*, 1977b). Moreover, it inhibits the uptake of gluconeogenic precursors

(i.e. lactate (Jorfeldt and Juhlin-Dannfelt, 1978) and glycerol (Lundquist *et al.*, 1965)) by the liver, which subsequently decreases gluconeogenesis. In addition, it has been shown that EtOH increases cardio-vascular strain during exercise by increasing the heart rate (HR) (Borg *et al.*, 1990; Ferreira *et al.*, 2004) but without modification of stroke volume (Blomqvist *et al.*, 1970). Also, a decrease of skeletal muscle strength may be observed after acute EtOH consumption (Williams, 1991) and can be attributed to an alteration of nerve conduction, a modulation of membrane excitability and an impairment of muscle membrane transporter function (Nicolas *et al.*, 1998).

In contrast, EtOH may in fact exert a small positive effect on exercise performance by decreasing pain and anxiety, which would in turn be beneficial to performance (Williams, 1991). Although it has been shown that acute doses of EtOH do not influence ratings of perception of effort (RPE) during short submaximal incremental exercise (Borg *et al.*, 1990), its effects the perceptual response during high intensity more prolonged endurance performance are not known.

It has been shown that acute EtOH consumption decreases middle-distance running (McNaughton and Preece, 1986) and endurance (Kendrick *et al.*, 1993) performance. In contrast, other studies did not show any effect of EtOH on the performance of runners (Bond *et al.*, 1983; Houmard *et al.*, 1987).

In this context, the aim of this study was to investigate the effect of an acute low dose of EtOH on the endurance performance of trained cyclists during a 1-h time trial (TT). It was hypothesized that EtOH would decrease endurance performance.

## METHODS

### Subjects

Thirteen male endurance-trained cyclists gave their informed consent to participate in this study, which was approved by the Ethics Committee of the Faculty of Biology and Medicine of

Table 1. Subjects' characteristics

	Mean	SD
Age (years)	31	10
Height (cm)	180	4
Weight (kg)	70.7	5.2
Body fat (%)	14.7	3.5
$W_{\max}$ (W)	349	29.1
$W_{\max}$ (W/kg)	4.9	0.6
$\dot{V}O_{2\max}$ (ml/kg/min)	60.9	6.0

$W_{\max}$ : maximal aerobic work output;  $\dot{V}O_{2\max}$ : maximal oxygen uptake.

the University of Lausanne (protocol no. 159/04). Their mean age, weight, height and maximal oxygen uptake ( $\dot{V}O_{2\max}$ ) are presented in Table 1. Subjects were irregular social alcohol drinkers or abstainers and had been involved in regular cycling training for at least 5–6 h per week for at least 3 years. The experimental sessions took place during the winter transition training season or the pre-competitive season. Subjects were familiar with road and/or off-road cycle racing and high-intensity training.

### Experimental design

The protocol consisted of four visits to the Department of Physiology. On the first visit,  $\dot{V}O_{2\max}$  and maximal aerobic power ( $W_{\max}$ ) were measured using an incremental test described below. The three subsequent visits were simulated TTs performed on a conventional road bike mounted on a magnetic resistance apparatus and which was equipped with a power measurement device. During each TT, subjects were asked to cycle as far as possible, or, in other words, to produce the highest workload as possible in 60 min (Coyle *et al.*, 1991; Bishop, 1997). All TTs were performed in a calorimetric chamber, in which room air temperature and humidity were controlled and maintained at 20°C and 60%, respectively. Visit 2 was a familiarization session to get accustomed to the cycling test and the general procedures. The experimental sessions (visits 3 and 4) were carried out in a semi-random order and in a balanced design. The exercise sessions were separated by ~7 days. Subjects ingested a solution containing either 0.5 ml of ethanol per kilogram of fat-free mass (FFM) and ~30 g of carbohydrate (CHO), or a control (C) solution of the same volume containing the same amount of CHO. The subjects were kept blind to the composition of the solution and were only asked after the last exercise session if they could distinguish the EtOH and C trials. For a given subject, all sessions occurred at the same time of the day and under standardized conditions when considering dietary intake and physical activity.

### Visit 1

Subjects arrived at the lab 3–4 h after eating a light meal. They were asked to repeat the same meal before each subsequent visit. Subjects were asked to refrain from strenuous physical activity and from drinking alcohol and caffeine during the preceding 24 h. Height and weight were recorded, and body composition was estimated using a four-skinfold measurement technique. Immediately after, they performed the incremental test to determine  $W_{\max}$  and  $\dot{V}O_{2\max}$ . The test was performed on a cycle ergometer (Ergoline eBike, GE Medical Systems, Freiburg, Germany) equipped with the subjects' own clipless

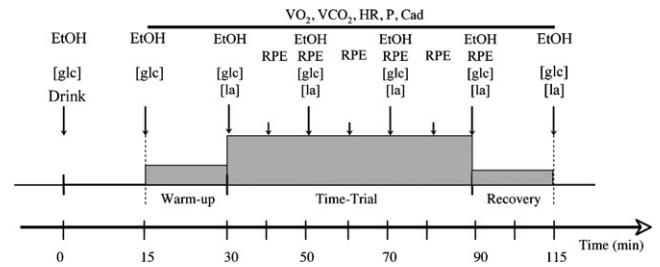


Fig. 1. Schematic representation of the experimental procedures. Following parameters were recorded at regular intervals, separated by 10 or 20 min: blood concentration of ethanol (EtOH), glucose (glc) and lactate (la). Oxygen consumption ( $\dot{V}O_2$ ), carbon dioxide production ( $\dot{V}CO_2$ ), heart rate (HR), power and pedalling cadence (Cad) were recorded continuously. The greyed areas represent the experimental procedures performed in the calorimetric chamber.

pedals. Saddle and handlebar positions were individually adjusted to the subjects' preferences. The  $\dot{V}O_{2\max}/W_{\max}$  test began with a 5-min warm-up at an initial power of 95 W. Thereafter, power was increased by 35 W every 3 min until volitional exhaustion.  $W_{\max}$  was determined as the last work rate completed plus the fraction of time spent in the unfinished stage multiplied by the work rate increment. The HR was recorded continuously by an automated 12-way electrocardiogram (Corina, GE Medical Systems, Freiburg, Germany). Breath-by-breath gas exchange measurements were recorded continuously during the test by an automated gas analysis system (Oxycon Pro, Jaeger, Hoechberg, Germany). The volume sensor was previously calibrated with an integrated automated flow calibration system and gas analysers were calibrated using a gas mixture of known concentrations (16% O<sub>2</sub>, 5% CO<sub>2</sub>). Oxygen uptake ( $\dot{V}O_2$ ) was considered maximal when at least two of the following criteria were met: (a) a levelling off of  $\dot{V}O_2$  with increasing workload (i.e. an increase of <2 ml/min/kg); (b) a respiratory exchange ratio (RER) >1.05; (c) HR within 10 beats of the predicted maximal HR estimated by 220-age.  $\dot{V}O_{2\max}$  was calculated as the average oxygen uptake of the last 30 s of the stage eliciting the highest  $\dot{V}O_2$ .

### Visits 2–4

Subjects reported to the lab after a 3- to 4-h fast and having abstained from alcohol, caffeine and strenuous physical activity for 24 h. The experimental procedures are summarized in Fig. 1. Upon their arrival at the Department of Physiology, blood glucose was measured using a fingertip capillary blood sample (FreeStyle, TheraSense Inc., Alameda, CA, USA). Then, they were asked to drink within 3 min a refrigerated (4°C) grapefruit juice solution containing 0.5 g CHO/kg of FFM and mixed with a commercially available ethanol-containing drink (Smirnoff Vodka 21, Smirnoff, Diageo, Switzerland) or the same volume of water. According to the manufacturer, the ethanol content of this drink was 40% vol. The amount of ingested EtOH was 0.5 ml/kg FFM, or  $30 \pm 1.8$  ml. The CHO contained in the solution were glucose (40%), fructose (40%) and sucrose (20%). Subjects had to wear a nose clip and rinse their mouth with water after ingestion in order to disguise the EtOH and C solution. Approximately one half of the subjects could distinguish both solutions, two inverted the treatments and the remainder were not able to distinguish them. Exactly 15 min after ingestion,

subjects seated on the bike (Cannondale SC800, Cannondale Bicycling Corp., Bethel, NJ, USA) fit to their own preferences (saddle and handlebar height and forward position) and placed on a stationary magnetic resistance apparatus (Tacx Swing, Tacx, The Netherlands). The magnetic resistance apparatus was set to the same position allowing subjects to choose gear ratios corresponding to their preferred pedalling cadence with a wide range of power outputs (PO) (i.e. 50–600 W). Immediately before cycling, the tire was inflated to 700 psi. At this moment, the zero-power offset of the power measurement device (SRM 'Professional', Schoberer Rad Messtechnik, Welldorf, Germany) was set according to the manufacturer instructions and the handlebar unit of the SRM was set at a recording rate of 1 Hz. At this time, they warmed-up for 15 min at a light self-selected intensity. At the end of the warm-up period, the blood EtOH concentration was estimated in breath, by means of a portable breath analyser (Draeger 6510, Draeger Safety, Luebeck, Germany). At the same time, blood glucose and blood lactate concentrations (Accusport Lactate, Boeringer, Mannheim, Germany) in fingertip capillary blood were measured. The TT simulation began immediately after the 15-min warm-up. At this time, the instruction was repeated: subjects were asked to produce the maximal average power in the given time (60 min) as previously described (Coyle *et al.*, 1991; Bishop, 1997; Bishop *et al.*, 1998). The procedure was slightly modified: the initial PO was not set during the first 8 min. However, subjects were instructed to begin with the same gear ratio that was mainly used during the familiarization session. During the TTs, the subjects were informed only of elapsed time by a countdown timer placed in front of them. HR, pedalling cadence, power and distance were hidden. During the entire session, water was available *ad libitum*.

Every 10 min, subjects were asked to rate their perceived exertion (RPE) according to the 6–20 Borg scale, and every 20 min, blood lactate and glucose concentrations in fingertip capillary blood samples were measured and blood EtOH in expired air was estimated.

At the end of the TTs, subjects performed a light active recovery for 1 min, after which they rested in a seated position. After 15 min of passive recovery, blood glucose and lactate concentrations were measured and the blood EtOH concentration was estimated.

The four TTs were performed in a calorimetric chamber, the characteristics of which have been previously published (Jequier and Schutz, 1983). The analysers of the chamber were calibrated with a gas of known concentrations. Minute ventilation was set at a rate of 110 l/min in order to maintain the CO<sub>2</sub> concentration below 1%.  $\dot{V}O_2$  and carbon dioxide production ( $\dot{V}CO_2$ ) were continuously recorded and averaged over periods of 15 min.

During all TTs, the same investigator remained in the calorimetric chamber close to the subject. During each trial, the investigator followed the same pattern of activity, remaining seated except for measurements of RPE, blood glucose, blood lactate and EtOH every 10 min. Furthermore, he did not interact with the subjects during the TTs.

To offset the effect of the investigator's contribution to total  $\dot{V}O_2$  and  $\dot{V}CO_2$ , a pretest was performed with the investigator alone:  $\dot{V}O_2$  and  $\dot{V}CO_2$  were measured and averaged during ten 15-min periods of a simulation of the investigators activity performed in the calorimetric chamber. These sessions

were carried out on a single day. Mean  $\dot{V}O_2$  and  $\dot{V}CO_2$  were found to be  $325 \pm 43$  and  $271 \pm 4$  ml/min, respectively. These values were subsequently subtracted from the metabolic measurements obtained during the TTs. Considering the mean  $\dot{V}O_2$  and  $\dot{V}CO_2$  measured during the TTs, and considering a 10% variability in the investigators'  $\dot{V}O_2$  and  $\dot{V}CO_2$ , this would in turn induce <1% variability in the subjects'  $\dot{V}O_2$  and  $\dot{V}CO_2$ , which is lower than the accuracy of the indirect calorimeter used in the present study.

#### Calculations

From  $\dot{V}O_2$  (l/min) and  $\dot{V}CO_2$  (l/min) measures, glucose and lipid oxidation rates (g/min) were calculated using the stoichiometric equations proposed by Frayn *et al.* (Frayn, 1983), with protein oxidation during exercise considered negligible. Thus, the following equations were used:

$$\text{CHO oxidation (g/min): } 4.55 \dot{V}CO_2 - 3.21 \dot{V}O_2,$$

$$\text{Lipid oxidation (g/min): } 1.67 \dot{V}O_2 - 1.67 \dot{V}CO_2.$$

When the RER equalled or was superior to 1.0 over a 15-min period, values of  $\dot{V}O_2$  and  $\dot{V}CO_2$  were not used for the calculation of substrate oxidation.

Gross external cycling efficiency (GE) was calculated as the ratio between mechanical PO and metabolic energy expenditure (EE), as previously described (Moseley and Jeukendrup, 2001).

#### Statistical analysis

Data are reported as mean  $\pm$  standard deviation.

Performance and physiological parameters (mechanical power, pedalling cadence, HR) were averaged in six 10-min periods in order to evaluate the effect of time and condition on these parameters and were then analysed by a two-way ANOVA with repeated measures. A Bonferroni *post hoc* test was applied when significant differences were found in means. Mean substrate oxidation rates over the TTs were compared using a Student's paired *t*-test. Blood EtOH levels across time were compared using a one-way ANOVA. Statistical significance was set at  $P < 0.05$ . All calculations were performed using a computerized statistical software (SigmaStat for Windows, version 2.03, SPSS, Chicago, IL, USA).

## RESULTS

Blood EtOH levels reached  $0.18 \pm 0.06\%$  at the start of exercise (i.e. exactly 30 min after drinking the solution) and subsequently reached  $0.20 \pm 0.06$ ,  $0.18 \pm 0.05$ ,  $0.16 \pm 0.05$  and  $0.14 \pm 0.04\%$  after 20, 40 and 60 min of exercise and at the end of recovery, respectively. No significant difference was found throughout the test. Performance, assessed by the mean mechanical PO, was significantly lower under EtOH condition ( $233 \pm 23$  W) as compared to C ( $243 \pm 24$  W) ( $P < 0.01$ ), representing a decrease of 3.9% in performance over the EtOH trial. The mean  $\dot{V}O_2$  were found to be  $3640 \pm 297$  and  $3513 \pm 323$  ml/min ( $P < 0.05$ ) in C and EtOH, respectively. Accordingly, the mean  $\dot{V}CO_2$  were  $3463 \pm 305$  and  $3299 \pm 393$  ml/min ( $P < 0.01$ ). Therefore, subjects performed the TTs with an intensity corresponding to  $83 \pm 8$  versus  $80 \pm 9\%$   $\dot{V}O_{2\text{max}}$  in C and EtOH, respectively ( $P < 0.05$ ). The time course of PO was significantly different (time  $\times$  condition) during the EtOH trial

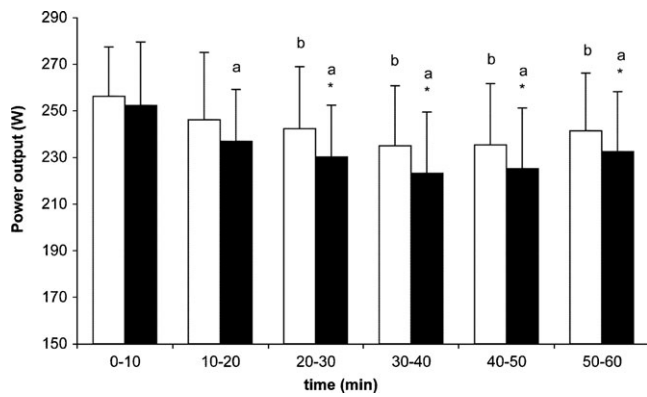


Fig. 2. The time course of power output during the 60-min time trial after ingestion of ethanol (EtOH) or control (C). Open bars and black bars represent the C and EtOH trials, respectively. \*A significant difference ( $P < 0.05$ ) with C; 'a' indicates a significant difference ( $P < 0.05$ ) with the first 10-min period (0–10) in EtOH; 'b' denotes a significant ( $P < 0.05$ ) difference with 0–10 in C.

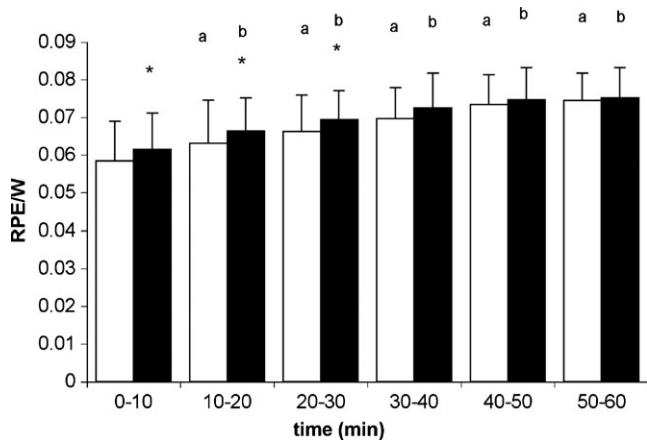


Fig. 3. The time course of the ratio between ratings of perceived exertion and work output (RPE/W) during the 60-min time trial after ingestion of ethanol (EtOH) or control (C). Open bars and black bars represent the C and EtOH trials, respectively. \*A significant difference ( $P < 0.05$ ) with C; 'a' indicates a significant difference ( $P < 0.05$ ) with the first 10-min period (0–10) in C; 'b' denotes a significant ( $P < 0.05$ ) difference with 0–10 in EtOH.

(Fig. 2) and PO was significantly lower from the second 10-min period to the end ( $P < 0.05$ ).

HR increased significantly over time from 158 bpm (end of the first 10-min period) and reached 171 bpm (last period). However, no significant difference was found between the two conditions. When normalized to the PO, the mean ratio between HR and PO was significantly higher during the EtOH trial ( $P < 0.05$ ).

The mean RPE was not different between C and EtOH but increased significantly with time in both conditions from the fourth period of the TTs to the end ( $P < 0.01$ ). No significant difference was found between conditions for RPE over time. However, the evolution of RPE normalized for work output (RPE/W) was significantly different during the EtOH trial compared to C: during the first three 10-min periods, RPE/W was significantly higher in EtOH as compared to C ( $P < 0.05$ ) (Fig. 3).

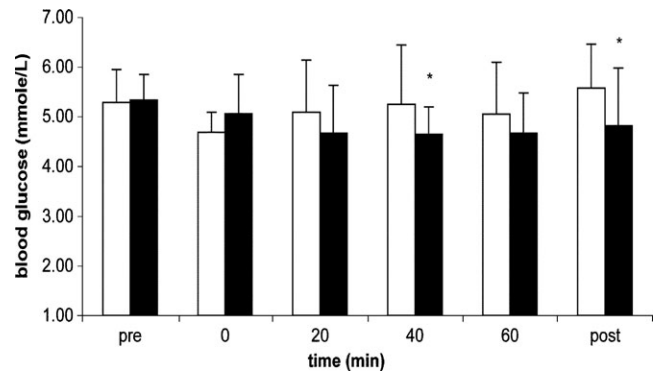


Fig. 4. Evolution of the blood glucose concentration during both the control (C) and the ethanol (EtOH) trial. \*A significant difference with C ( $P < 0.05$ ).

The mean blood glucose concentration was similar during both trials ( $5.1 \pm 0.9$  versus  $4.8 \pm 0.5$  mmol/l in C and EtOH, respectively). However, a slight decrease was observed in the EtOH condition (Fig. 4) compared to C at 40 min of exercise and at the end of the recovery period ( $P < 0.05$ ). Blood lactate concentrations were similar between conditions ( $5.1 \pm 1.4$  versus  $4.9 \pm 1.1$  mmol/l for C and EtOH, respectively) and over time.

Gross cycling efficiency was not different between both conditions ( $19.1 \pm 1.1\%$  versus  $19.0 \pm 1$  in C and EtOH, respectively).

Glucose oxidation rate was significantly ( $P < 0.05$ ) lower in the EtOH condition ( $3.1 \pm 0.6$  g/min (EtOH) versus  $3.5 \pm 0.5$  g/min (C)), whereas lipid oxidation rate tended to be greater ( $0.5 \pm 0.1$  g/min (EtOH) versus  $0.4 \pm 0.1$  g/min (C)), although it did not reach statistical significance ( $P = 0.099$ ).

## DISCUSSION

Information on the effect of acute EtOH consumption on endurance performance during cycling is almost non-existent, unlike running (McNaughton and Preece, 1986; Houmard *et al.*, 1987; Kendrick *et al.*, 1993), although cycling and running can be compared in terms of intensity and duration of exercise and physiological requirements.

The results of the present study are consistent with a previous study (McNaughton and Preece, 1986) that showed a decrease in the performance of runners in short- and middle-distance events after ingestion of different EtOH doses, inducing blood alcohol concentrations similar to those measured in the present study. These running events were of a shorter duration (i.e. between 11 s and 5 min) as compared with a 1-h TT, probably involving a larger contribution of the 'anaerobic' metabolism. Another study (Kendrick *et al.*, 1993) reported a significant negative effect of EtOH on the ability of well-trained runners to complete a 60-min running event. Some variables associated with performance (i.e. blood glucose concentration,  $\dot{V}O_2$ , HR) were affected, but running performance was not measured. In contrast, other studies did not report any significant change in running performance after EtOH ingestion (Bond *et al.*, 1983; Houmard *et al.*, 1987), assessed by a TT protocol or an incremental test. The discrepancy between the different studies can be explained by the differences in the experimental designs

and the exercise duration, intensity and type of performance measured. Running may be considered as a complex motor skill, and the decrease in performance during running under EtOH influence may be partially attributed to the alteration of coordination skills (McNaughton and Preece, 1986). In contrast, because cycling on an ergometer may not require high psychomotor or coordination skills, it seems reasonable that our results focus on the disturbances induced by EtOH on the metabolic aspects of performance. This is confirmed by the fact that our results show no difference in gross efficiency between the trials, which probably means that the coordination skills necessary to cycling ergometry were not altered after EtOH ingestion.

During the EtOH trial, time course of PO was significantly modified. A significant drop of PO occurred after 20 minutes of exercise, whereas PO was maintained during the C trial at this time. Afterwards, PO was significantly decreased in both conditions as compared to the initial 10-min period but to a greater extent during the EtOH trial ( $P < 0.05$ ). Overall, EtOH induced a 4% decrease in average PO. In terms of outdoor performance an early decrease of PO may lead to a greater decrease of TT performance, since pacing strategy is known to be an important factor for TT performance (Swain, 1997).

The early decrease of PO was not correlated to changes occurring in other metabolic parameters (i.e. blood glucose or lactate concentration) or the perceptual response (i.e. RPE). In fact, EtOH would induce psychobiological and/or metabolic effects that interfere with exercise performance but that were not measured in the present study. One explanation would be a cognitive effect of EtOH, inducing a misjudgement of the exercise capacity of the athletes, leading them to start exercise at a too high intensity. As a result, the athletes would fail to maintain the initial PO throughout the TTs. This may be linked with the comments of the subjects who generally reported a state of well-being at the beginning of the EtOH trials (personal communication). However, in both conditions, subjects started the TT at the same PO, meaning that EtOH probably did not lead them to overestimate their endurance capacity but altered the metabolic aspects of endurance performance discussed below.

Alcohol has previously been shown to induce an early appearance of hypoglycaemia, through a decrease of the hepatic glucose production occurring within 1 h of light-to-moderate intensity exercise (Jorfeldt and Juhlin-Dannfelt, 1977, 1978; Juhlin-Dannfelt *et al.*, 1977a; Kendrick *et al.*, 1993). In contrast, our results (Fig. 4) showed no differences in blood glucose concentrations throughout the trials. The small doses of EtOH used in our study and the small amount of CHOs contained in both the C and EtOH drink may explain the maintenance of euglycaemia throughout the TTs. As outlined by Jorfeldt and Juhlin-Dannfelt (Jorfeldt and Juhlin-Dannfelt, 1978), the prerequisite for an EtOH-induced hypoglycaemia during exercise would be a fasting state. In this study, our subjects performed the exercise several hours after eating their last meal, but not under fasting conditions. Moreover, since EtOH decreases muscle glucose uptake during exercise (Juhlin-Dannfelt *et al.*, 1977a; Jorfeldt and Juhlin-Dannfelt, 1978), the EtOH-induced decrease in hepatic glucose production may be counterbalanced by the former mechanism. Furthermore, Massicotte *et al.* (1993) showed no effect of EtOH on endogenous substrate oxidation during exercise. Taken together, this would

explain the maintenance of glycaemia throughout the trials. Our results also show that total CHO oxidation was significantly lower during EtOH than during C. Because POs were significantly lower during EtOH than during C, it is likely that the lower glucose oxidation during EtOH is the consequence of a lower exercise intensity and that CHO metabolism was not altered.

The mean blood lactate concentration during both trials was relatively high and above the so-called 4 mmol anaerobic threshold, confirming the high intensity at which the subjects performed the exercise trials. However, the values measured in this study are lower than those reported by Coyle *et al.* (1991) (i.e. 7.1–7.4 mmol/l) with elite cyclists. The higher relative intensity of exercise (86–90%  $\dot{V}O_{2\max}$ ) reported in the latter study as compared to the present study may explain this difference. In our experiment, the blood lactate concentrations were not different between conditions, although the exercise intensity was significantly lower during the EtOH condition. Under EtOH influence, lactate production and oxidation in both muscle and liver and its conversion into glucose by gluconeogenesis are modified (Jorfeldt and Juhlin-Dannfelt, 1977, 1978; Juhlin-Dannfelt *et al.*, 1977a, 1977b). Although lactate fluxes were not measured in the present study, our results support previous studies showing EtOH disturbances of lactate metabolism (Jorfeldt and Juhlin-Dannfelt, 1977, 1978; Juhlin-Dannfelt *et al.*, 1977a, 1977b).

Our results show no difference of mean HR and time course of HR during the trials between C and EtOH. Previous studies showed that HR was elevated during exercise in response to EtOH ingestion, at least during submaximal exercise (Blomqvist *et al.*, 1970; Juhlin-Dannfelt *et al.*, 1977b; Borg *et al.*, 1990; Kendrick *et al.*, 1993; Ferreira *et al.*, 2004). During high-intensity exercise, however, HR was not influenced by EtOH (Blomqvist *et al.*, 1970; Borg *et al.*, 1990). In the present study, however, the ratio between HR and PO was increased by EtOH, which means that the HR response to a given exercise intensity was elevated. As outlined by Blomqvist *et al.* (1970), the elevated HR response during exercise after EtOH ingestion may be due to a shift in blood flow towards other tissues, such as an increase of skin perfusion due to the cutaneous vasodilation effect of EtOH. In addition, this study reported no difference in stroke volume during exercise during an EtOH trial. It seems therefore that during high intensity but submaximal exercise, EtOH increases cardio-vascular strain, which may account for the reduced performance measured in our study.

In contrast to previous work (Borg *et al.*, 1990), the relationship between RPE and exercise intensity was increased by EtOH. Considering that pacing strategy would partially depend on the actual RPE (Hampson *et al.*, 2001), it is reasonable to postulate that the decrease of performance could be partly due to a change in the perception of effort and thus through a psychobiological mechanism. Interestingly, a recent study showed that a mouth rinse with glucose had a similar, but stimulating, psychobiological effect on cycling performance (Carter *et al.*, 2004). Moreover, according to recent models of fatigue during exercise, pacing strategy is regulated within the brain, which is directly influenced by peripheral feedbacks (Abbiss and Laursen, 2008). The elevated RPE response during the EtOH trial may have thus indirectly influenced pacing strategy and, as a consequence, performance.

In conclusion, acute consumption of EtOH decreased endurance exercise performance as previously shown (American College of Sports Medicine, 1982; McNaughton and Preece, 1986; Kendrick *et al.*, 1993). Its effects were not correlated with any modifications of the gross metabolic or physiologic variables measured in this study. Our results suggest that EtOH worsens cycling performance by a concomitant alteration of physiological and psychobiological mechanisms. In practice and as previously stated (American College of Sports Medicine, 1982), athletes should avoid drinking EtOH, even at low doses, before training sessions or competition.

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