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 EO1 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, glycemia 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; Heikonen 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
the University of Lausanne (protocol no. 159/04). Their mean age, weight, height and maximal oxygen uptake ($V\text{O}_{2\text{max}}$) 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, $V\text{O}_{2\text{max}}$ and maximal aerobic power ($W_{\text{max}}$) were measured using an incremental test described below. The three subsequent visits were simulated TTs 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.

**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 $W_{\text{max}}$ and $V\text{O}_{2\text{max}}$. The test was performed on a cycle ergometer (Ergoline eBike, GE Medical Systems, Freiburg, Germany) equipped with the subjects’ own clipless pedals. Saddle and handlebar positions were individually adjusted to the subjects’ preferences. The $V\text{O}_{2\text{max}}/W_{\text{max}}$ 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. $W_{\text{max}}$ 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% $O_2$, 5% $CO_2$). Oxygen uptake ($V\text{O}_2$) was considered maximal when at least two of the following criteria were met: (a) a levelling off of $V\text{O}_2$ with increasing workload (i.e. an increase of $<2 \text{ ml/min/kg}$); (b) a respiratory exchange ratio (RER) $>1.05$; (c) HR within 10 beats of the predicted maximal HR estimated by 220-age. $V\text{O}_{2\text{max}}$ was calculated as the average oxygen uptake of the last 30 s of the stage eliciting the highest $V\text{O}_2$.

**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 ± 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 warm-up. At this time, the instruction was repeated: subjects were instructed to begin with the same gear ratio 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 after the 15-min period, values of VO\(_2\) and VCO\(_2\) 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 ± 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 ± 0.06%\(_e\) at the start of exercise (i.e. exactly 30 min after drinking the solution) and subsequently reached 0.20 ± 0.06, 0.18 ± 0.05, 0.16 ± 0.05 and 0.14 ± 0.04%\(_e\) 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 ± 23 W) as compared to C (243 ± 24 W) (\( P < 0.01 \)), representing a decrease of 3.9% in performance over the EtOH trial. The mean VO\(_2\) were found to be 3640 ± 297 and 3513 ± 323 ml/min (\( P < 0.05 \)) in C and EtOH, respectively. Accordingly, the mean VCO\(_2\) were 3463 ± 305 and 3299 ± 393 ml/min (\( P < 0.01 \)). Therefore, subjects performed the TTs with an intensity corresponding to 83 ± 8 versus 80 ± 9% VO\(_2\)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.

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 CHO's 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% \( \dot{V}\text{O}_{2\text{max}} \)) 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 tissue, 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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REFERENCES


