Eur J Appl Physiol (1997) 76: 197–202

ORIGINAL ARTICLE

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Metabolic and hormonal responses during repeated bouts of brief and intense exercise: effects of pre-exercise glucose ingestion

Accepted: 22 February 1997

Abstract We investigated metabolic and hormonal responses during repeated bouts of brief and intense exercise (a force-velocity test; *Fv* test) and examined the effect of glucose ingestion on these responses and on exercise performance. The test was performed twice by seven subjects [27 (2) years] according to a double-blind randomized crossover protocol. During the experimental trial (GLU), the subjects ingested 500 ml of glucose polymer solution containing 25 g glucose 15 min before starting the exercise. During the control trial (CON), the subjects received an equal volume of sweet placebo (aspartame). Exercise performance was assessed by calculating peak anaerobic power ($\dot{W}_{an,peak}$). Venous plasma lactate concentration increased significantly during the *Fv* test ($P < 0.001$), but no difference was found between CON and GLU. Blood glucose first decreased significantly from the beginning of exercise up to the 6-kg load ($P < 0.001$) and then increased significantly at $\dot{W}_{an,peak}$ and for up to 10 min during the recovery period ($P < 0.001$) in both CON and GLU. Insulin concentrations decreased significantly in both groups, but were higher at $\dot{W}_{an,peak}$ in GLU compared with CON ($P < 0.05$). Glucagon and epinephrine did not change significantly in either group, but epinephrine was significantly lower in GLU after glucose ingestion ($P < 0.05$) and at $\dot{W}_{an,peak}$ ($P < 0.05$). $\dot{W}_{an,peak}$ was not significantly different between CON and GLU. In conclusion, blood glucose and insulin concentrations decreased during repeated bouts of brief and intense exercise, while blood lactate concentration increased markedly without any significant change in glucagon and epinephrine concentrations. Glucose ingestion altered metabolic and hormonal responses during the *Fv* test, but the performance as measured by $\dot{W}_{an,peak}$ was not changed.

Key words Glucose · Insulin · Epinephrine · Lactate · Force-velocity test

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Introduction

Exercise is known to have effects on blood glucose levels as well as on concentrations of glucoregulatory hormones (Alborg and Felig 1976; Pruett 1970). The studies concerning the hormonal responses involved in glucoregulation during prolonged exercise are well known (Coyle et al. 1986; Marliss et al. 1991). There is evidence that carbohydrates are necessarily involved in the metabolism of active muscles, and their availability may be a limiting factor in some muscular performances and endurance events. It is also clear that the glucose concentration in blood is dependent on the level of power output (Coyle et al. 1986). However, to our knowledge no investigation has been carried out on the metabolic and hormonal responses that occur during repeated bouts of brief intense exercise such as during a force-velocity exercise test (*Fv* test). Some studies have shown that performance of intense exercise lasting a few seconds depends mainly on glycolytic processes (Hirvonen et al. 1987; Medbo and Tabata 1989). Furthermore, other studies have reported that repeated bouts of intense exercise (6 s) against increasing braking forces on a cycle ergometer induces a high venous lactate accumulation (Bedu et al. 1991; Mercier et al. 1991, 1994). Since repeated bouts of brief and intense exercise stimulate strongly glycolytic metabolism, we hypothesized that blood glucose and glucoregulatory hormones would be altered by this kind of exercise task. Because glucose is an important substrate for the glycolytic pathway, another testable hypothesis is that performance may be

improved by pre-exercise glucose ingestion. To our knowledge, this has never been investigated during an *Fv* test. Indeed, authors who have investigated the effect of carbohydrate ingestion on performance during high-intensity exercise used other types of exercise tasks (Hargreaves et al. 1997; Montain et al. 1991; Snyder et al. 1993), which may lead to differences in the metabolic and glucoregulatory hormonal responses observed.

We therefore investigated the changes in blood glucose and hormonal responses involved in glucoregulation during the *Fv* test which was used as a model of repeated bouts of brief and intense exercise. In addition, we studied the effect of pre-exercise glucose ingestion on these metabolic and hormonal responses and on the performance as measured by peak anaerobic power ($\dot{W}_{an,peak}$).

Methods

Subjects

Seven healthy male subjects participated in this study. Their mean (SEM) age, body mass and height were 27.6 (2.8) years, 72 (3.3) kg and 174 (4.0) cm, respectively. During the 3 days preceding each trial the subjects refrained from exercising and from drinking alcohol and caffeine. All of the subjects were judged to be free of cardiopulmonary diseases on the basis of a medical examination that included a resting electrocardiogram. All subjects gave their informed written consent and the study was approved by the local ethical committee.

Exercise test

The *Fv* test was conducted on a cycle ergometer (864, Monark-Crescent AB, Varberg, Sweden) and consisted of repeated maximal sprints of 6 s duration against increasing braking forces (*F*) with a 5-min between-bout recovery period (Vandewalle et al. 1987). The *Fv* relationships were calculated using an automatic system (Mercier et al. 1991) that allowed the determination of peak velocity (*v*) from the measurement of pedal frequency, and for each braking force, the power corresponding to the product *Fv*. The accuracy of the measurement of pedal revolution duration was 3.3 ms. During the test, the power corresponding to the product of the braking force and the maximal pedalling frequency was determined for each braking force. Given the linear *Fv* relationship, the power-velocity and power-force relationships were parabolic (Vandewalle et al. 1987). $\dot{W}_{an,peak}$ was defined as the highest power output value calculated for the different braking forces.

Blood samples and analysis

Venous blood samples were drawn from a Teflon catheter (32 mm cathlon IV 4426) inserted into a superficial forearm vein. Samples were placed into tubes containing heparin and sodium fluoride for glucose determination, fluoride/ethylene diamine tetra-acetic for lactate measurement, lithium and heparin for insulin determination, and lithium and heparin with 200 µl of a protease inhibitor for glucagon determination. For epinephrine determination approximately 3 ml of blood was placed into a tube (lithium heparin) containing reduced glutathione (1.2 mg · ml^{-1}) to control catecholamine oxidation. The sample was then centrifuged at 3,000 *g* for 10 min and the plasma was stored at −80°C. Plasma epinephrine concentrations were determined using high performance liquid chromatography (HPLC). Before the HPLC run the epinephrine was extracted by selective adsorption to aluminum oxide (Chromsystems-HPLC-Kit, Waters, Milford, Mass., USA). The aluminum oxide was shaken up briefly in extraction buffer (50 µl) and then 1 ml of plasma was added with 50 µl internal standard solution (600 pg dihydroxybenzylamine). The aluminium oxide was then washed three times, with a brief centrifugation between washes. The epinephrine was extracted with 120 µl elution buffer by shaking briefly and subjecting it to a final centrifugation at 1,500 *g* for 1 min. Then 50 µl of the sample eluant was injected into the HPLC column (Resolve TM 5 µl Sherical C 18, HPLC column, Waters) and eluted with a mobile phase. The flow rate was 1 ml · min^{-1} at 13.8 mPa and a potential of 0.60 V. The chromatogram was analyzed by computer integration (Baseline 815, Waters). Plasma lactate concentrations were measured using a fully enzymatic method (Lactate UV L76B, Boehringer, France). The different readings were taken by a spectrophotometer (Cobas-Bio, Roche, France). Plasma glucose was measured using the glucose oxidase method (Glucose Analyzer II, Beckman Instruments, Fullerton, Calif., USA). Plasma levels of insulin and glucagon were determined using radioimmunoassay reagent kits (CIS BioInternational, Gif-sur-Yvette, France and Biodata Rome, Italy respectively) with intra-assay coefficients of variation of 1.7 and 2.5%, respectively.

Protocol

Subjects performed two *Fv* tests 1 week apart in a double-blind randomized crossover: one with placebo (CON) and one after glucose ingestion (GLU). Subjects reported to the laboratory at 8 a.m. after an overnight fast and received a light standard breakfast including two wheat muffins and 300 ml of unsweetened orange juice, which represented a total energy intake of 1,200 kJ. A catheter was then inserted into a forearm vein for blood sampling and was kept patent by periodic flushing with 0.9% saline. Blood samples were collected for fast glucose determination. The subjects started the test 1 h 30 min after receiving a standard breakfast and either 500 ml of a glucose polymer solution containing 25 g of carbohydrate (GLU) or an equal volume of an aspartame-citric acid sweet placebo (CON). It was impossible for subjects to distinguish between the drink consumed in CON and GLU conditions. Indeed, the drinks had the same volume, temperature and taste because the placebo was flavored with aspartame. Furthermore, both drinks were presented to the subjects in the same colored containers. Subjects started the test against a braking force of 2 kg and they recovered for 5 min before repeating another intense exercise against an increased braking force (+2 kg). At the end of the test the braking force increase was 1 kg in order to obtain as precise a $\dot{W}_{an,peak}$ as possible. A three-lead electrocardiogram (D_{II}, V_2, V_5) (Quinton Q3000, Seattle, Wash., USA) was monitored continuously during the test for recording the heart rate. Blood samples were drawn for plasma lactate and glucose analysis with the subjects at rest before ingestion (BI), after ingestion immediately before starting the exercise (AI), during the *Fv* test for each load at the end of pedalling, and after the completion of exercise (recovery) at 5 and 10 min. In addition, plasma insulin, glucagon and epinephrine were measured before and after ingestion at rest and during exercise at $\dot{W}_{an,peak}$.

Statistical analysis

Data are presented as means (SEM). To test the effect of repeated bouts of brief and intense exercise on hormonal and metabolic responses we performed a one-way analysis of variance for repeated measures (ANOVA). A two-way ANOVA (group × load) was used to investigate the effect of GLU ingestion on exercise performance as well as metabolic and hormonal responses. When the *F*-ratio was significant, the location of the differences was identified using a Newman-Keuls post-hoc test. The level of statistical significance was set at $P < 0.05$ throughout the study.

Results

Control trial

Table 1 shows that during the *Fv* test the power output ($P < 0.05$) and heart rate ($P < 0.001$) values obtained at each load increased significantly. The time-course of plasma glucose and lactate changes during the test and recovery are shown in Fig. 1 a and b. Blood glucose first decreased significantly from the beginning of exercise up to the load of 6 kg ($P < 0.001$) and then increased significantly at $\dot{W}_{an,peak}$ and for up to 10 min during the recovery period ($P < 0.001$). Plasma lactate levels increased significantly during the test ($P < 0.001$). The values of plasma insulin, glucagon and epinephrine measured at rest and at $\dot{W}_{an,peak}$ are shown in Fig. 2a–c, respectively. During the *Fv* test insulin decreased significantly at $\dot{W}_{an,peak}$ ($P < 0.001$), while glucagon and epinephrine did not.

Glucose ingestion trial

At rest and before glucose ingestion, the plasma glucose, lactate, insulin, glucagon and epinephrine concentrations were not significantly different between the CON and GLU conditions. After glucose ingestion blood levels of glucose were significantly greater in the GLU than in the CON group ($P < 0.05$). During exercise glucose ingestion was not associated with significant changes in power or heart rate (Table 1). The onset of exercise induced a significant decrease in blood glucose levels ($P < 0.001$); however, blood glucose remained higher in GLU than in CON up to the power output corresponding to 4 kg braking force ($P < 0.05$). Above this power output the profile changed until the end of recovery so that blood glucose concentrations became lower in GLU compared with CON ($P < 0.05$). As in CON, blood lactate concentrations increased significantly during the *Fv* test after glucose ingestion ($P < 0.001$). There was no significant difference in blood lactate levels between CON and GLU. After completion of the test, blood lactate concentrations declined, with no differences being observed between CON and GLU (Fig. 1b). After glucose ingestion plasma insulin was

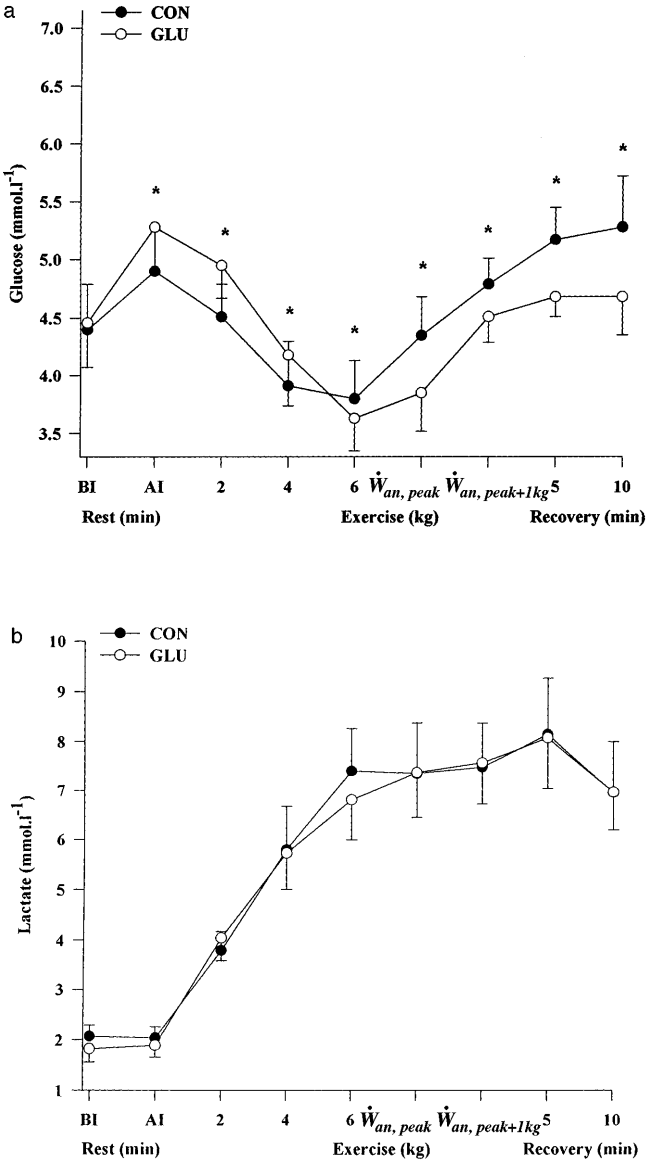

Fig. 1 Venous blood glucose levels (**a**) and venous blood lactate levels (**b**) before, during and after the force-velocity (*Fv*) test in the control condition (*CON*) and after glucose ingestion (*GLU*). Values are means (SEM), $n = 7$. (*BI* Before placebo or glucose ingestion, *AI* after placebo or glucose ingestion, $\dot{W}_{an,\,peak}$ peak anaerobic power, $\dot{W}_{an,\,peak+1kg}$ Peak anaerobic power measured after the addition of a further 1-kg load). * Significant difference between GLU and CON, $P < 0.05$

Table 1 Values of power and heart rate measured during exercise in control (*CON*) and after glucose ingestion (*GLU*). Values are means (SEM), $n = 7$. (*HR* Heart rate, $\dot{W}_{an,peak}$ peak anaerobic power, $\dot{W}_{an,peak+1kg}$ peak anaerobic power measured after further addition of a 1-kg braking force)

Parameter	Load (kg)						Power output			
	2		4		6		$\dot{W}_{an,peak}$		$\dot{W}_{an,peak+1kg}$	
	CON	GLU	CON	GLU	CON	GLU	CON	GLU	CON	GLU
HR	130	128	141	138	143	146	154	151	152	153
(beats · min^{-1})	(6)	(5)	(4)	(5)	(5)	(4)	(5)	(5)	(5)	(5)
Power	379	369	660	669	876	860	1018	990	966	928
(W)	(7)	(9)	(21)	(16)	(28)	(28)	(60)	(52)	(57)	(48)

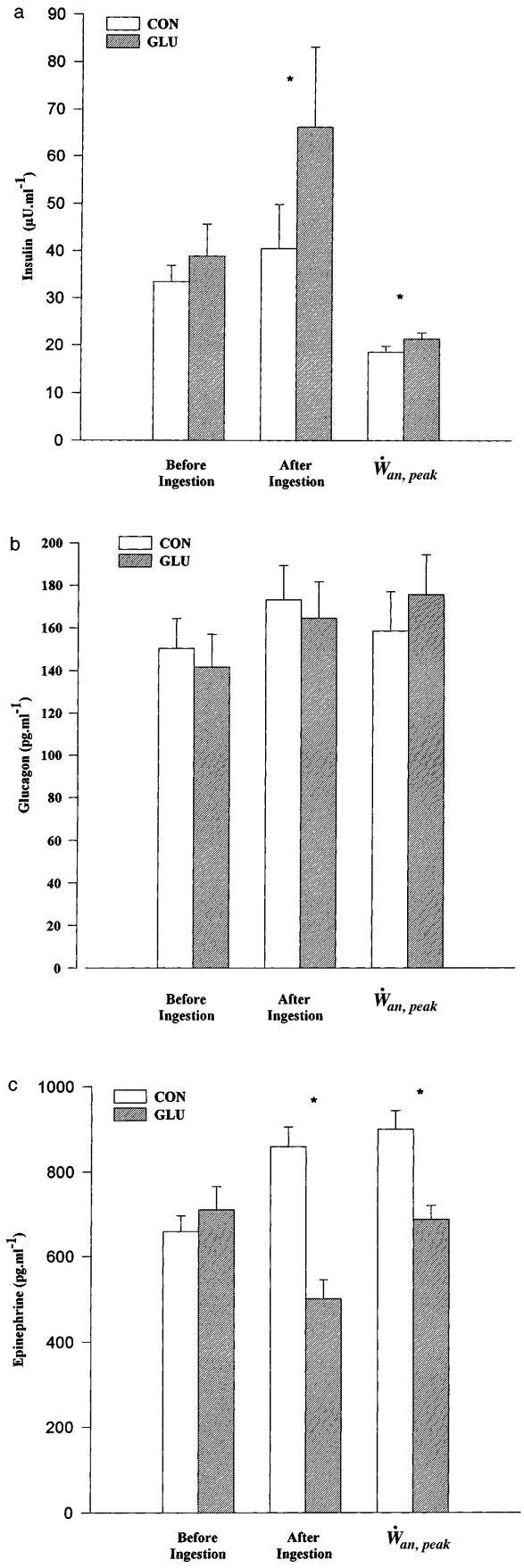

Fig. 2a–c Plasma insulin (**a**), glucagon (**b**) and epinephrine (**c**) concentrations before (*BI*) and after ingestion (*AI*) of either glucose or placebo, and at peak exercise during the *Fv* test in the control condition (*CON*) and after glucose ingestion (*GLU*). Values are means (SEM), $n = 7$. * Significant difference between GLU and CON, $P < 0.05$

greater in the GLU than in the CON group ($P < 0.05$). During the test it decreased significantly ($P < 0.001$) but the values were greater in GLU at $\dot{W}_{an,peak}$ ($P < 0.05$) (Fig. 2a). No significant difference was found between CON and GLU for glucagon (Fig. 2b). Plasma epinephrine levels were significantly lower after glucose ingestion at rest before starting exercise ($P < 0.05$) and at $\dot{W}_{an,peak}$ ($P < 0.05$, Fig. 2c).

Discussion

This study is the first investigation on glucoregulation during repeated bouts of brief and intense exercise, such as during the *Fv* test. We observed a decrease in glycemia and insulin, whereas epinephrine and glucagon were not significantly changed. Pre-exercise glucose ingestion resulted in higher insulin levels associated with a greater decrease in glycemia and a lower epinephrine response, but performance, blood lactate and glucagon responses were unchanged.

To calculate the anaerobic power we used the early definition of Vandewalle et al. (1987), i.e., the product of braking force and peak velocity without taking into account the flywheel inertia. In these conditions the anaerobic power is unlikely to represent the true $\dot{W}_{an,peak}$ (Lakomy 1986). However, since the calculation of power output was the same for CON and GLU conditions, $\dot{W}_{an,peak}$ represents a valid index for investigating the effect of GLU ingestion on performance as well as on metabolic and hormonal responses. Furthermore, because the order of testing was randomized and all subjects were familiarized with the *Fv* test before experimentation, the changes in performance and the metabolic and hormonal responses cannot be attributed to the effect of day-to-day variation or learning. Before the ingestion of 500 ml of glucose or placebo the subjects received an identical standard breakfast at the same hour in the morning. This was done in order to fulfill the conditions necessary for subjects performing an intense exercise (Galbo 1981; Péquinot et al. 1980). Coffee and tea were not included in the breakfast because of the potential pharmacological effects of their methylxantine component on anaerobic performance and the metabolic and hormonal responses to exercise. Indeed, our group has shown that caffeine increases $\dot{W}_{an,peak}$ and blood lactate concentrations (Anselme et al. 1992). We also took great care to standardize the timing during the experiment. All of the subjects were thus exercised at the same time, i.e., at 9:30 a.m., because it is well known that circadian rhythms change exercise performance and plasma hormone concentrations as well as glucose tolerance (Simon et al. 1987; Van Cauter et al. 1989).

The results of this study confirm those of previous reports that showed a marked lactate increase during repeated bouts of brief and intense exercise of 6 s duration against increasing braking forces (Mercier et al. 1991, 1994). By contrast, an unexpected result was the hypoglycemia that occurred concomitantly with the marked decrease in insulin. However, the glucagon and catecholamine responses induced by the exercise task might provide an explanation for this apparently surprising result. Indeed, these two major short-term hyperglycemic hormones were not increased during the *Fv* test, probably because of the relative short duration of the exercise. Marliss et al. (1991) reported a hyperglycemia associated with a marked increase in epinephrine in subjects performing two identical bouts of intense exhaustive exercise of 15–20 min duration separated by 1 h of recovery, whereas in our study subjects performed repeated bouts of brief and intense exercise of 6 s duration against increasing forces. We can hypothesize that the consequence of the hormonal profile observed during the *Fv* test was an increase in hepatic glucose that was insufficient to compensate for the high glucose consumption through the anaerobic pathway. This waste of glucose was probably the main explanation for the marked decrease in glycaemia observed during the test.

These findings prompted us to study the effect of pre-exercise glucose ingestion on performance as measured by $\dot{W}_{an,peak}$, as well as metabolic and hormonal responses, during this kind of exercise. In contrast to the numerous studies in which the effects of carbohydrate ingestion on prolonged exercise performance and metabolic responses have been investigated (Hargreaves et al. 1984; Coggan and Coyle 1988; Coyle 1992; Murray et al. 1989), there are very few studies in which these effects have been investigated during repeated bouts of brief and intense exercise. Our experiment does not show any performance improvement, but we observed that pre-exercise glucose ingestion altered both metabolic and hormonal profiles. This lack of improvement in performance was also observed by Snyder et al. (1993) and Hargreaves et al. (1997), who also investigated performance during high-intensity cycling exercise after carbohydrate ingestion. From our results and those of Hargreaves et al. (1997), who reported that increased muscle glycogen availability has no direct effect on performance during high-intensity exercise, we can hypothesize that the main source of energy during repeated bouts of intense exercise is high-energy phosphate (Casey et al. 1996a, b) and not blood glucose or muscle glycogen content. The more marked hypoglycemia during exercise after glucose ingestion seems somewhat paradoxical, but this has also been observed by Montain et al. (1991). Montain et al. (1991) observed an enhanced susceptibility to hypoglycemia during exercise in the early period after carbohydrate feeding and reported that this phenomenon is not modified by muscle glycogen content. This hypoglycemia could be explained largely by the higher insulin and lower epinephrine responses observed in this condition, but also by a higher muscle glucose uptake. Indeed, very recently Ljungqvist and Horton (1996) reported that the stimulation of glucose uptake induced by oral ingestion of glucose may be explained by the increase in the rate of glucose transport across the plasma membrane and the Glut 4 translocation in human skeletal muscle. In fact, the beneficial effects of glucose ingestion, if any, are counteracted by the effects of glucose on insulin and epinephrine levels, which are likely to blunt hepatic glucose production, as well as by the molecular mechanisms underlying glucose disposal in skeletal muscle. Considering the important role of glucagon during exercise (Wasserman et al. 1989), it is surprising that plasma levels, although increased modestly by the *Fv* test, were not affected by glucose ingestion. This can be explained by the fact that plasma glucagon concentrations do not reflect changes in portal blood (Sirek et al. 1979) and that the main glucoregulatory factors during exercise seem to be the insulin and catecholamine responses.

In summary, repeated bouts of brief and intense exercise induced a decrease in blood glucose and insulin that was associated with a marked increase in blood lactate, whereas plasma levels of glucagon and epinephrine did not change. Pre-exercise glucose ingestion alters metabolic and hormonal responses during the *Fv* exercise test, but does not enhance the performance as measured by this test.

Acknowledgements We thank all participants for their enthusiastic cooperation and Jacqueline Coll (Cies) for making this study possible. We also thank Emmanuel Bitanga for his thorough discussions and comments. This work was supported in part by a grant from the Ministère de la Coopération Française.

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