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ORIGINAL ARTICLE

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Effects of pre-exercise ingestion of carbohydrate on glycaemic and insulinaemic responses during subsequent exercise at differing intensities

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Abstract The development of rebound hypoglycaemia has been reported after pre-exercise carbohydrate (CHO) ingestion in some studies but not in others. Differences in the experimental design and factors such as the exercise intensity are likely to be responsible for the discrepancies between these studies. Exercise intensity might be a crucial factor since it affects both insulinaemia and glucose uptake. Therefore the aim of the present study was to compare the glycaemic and insulinaemic responses to exercise at different intensities after ingestion of a standardized pre-exercise CHO load. Eight moderately trained subjects consumed 75 g of glucose 45 min prior to 20 min of exercise at 40%, 65% or 80% maximal power output. Blood samples were collected before glucose ingestion, at 15 min intervals at rest and 5 min intervals during exercise. During exercise, measurements of heart rate and breath-by-breath analysis of expired gas were performed continuously. The trials were performed at [mean (SEM)] 55 (1), 77 (1) and 90 (1) percentages maximal oxygen uptake . At the onset of exercise, plasma glucose concentration returned to pre-ingestion levels, while the insulin concentration was more than three times higher than at rest [on average 57 (7) compared to 16 (1) $\mu U \cdot ml^{-1}$). During exercise, plasma glucose concentrations decreased during the first 5 min of exercise and then stabilized in all trials at concentrations that would not be considered to be hypoglycaemic. There were no significant differences in glucose or insulin concentrations between the three trials during exercise. These data suggest that the glycaemic response to ingestion of 75 g of CHO 45 min pre-exercise is similar during exercise of different intensities.

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Introduction

This study was one of a series designed to systematically investigate factors brought into play when carbohydrate (CHO) is ingested pre-exercise and how these affect exercise metabolism and subsequent time trial performance. The level of exercise intensity affects both the amount of glucose uptake by the muscle and the amount of endogenous glucose production. To maintain plasma glucose concentration during exercise, hepatic glucose output is increased (Marmy-Conus et al. 1996) to match the increased muscle glucose uptake (Wahren et al. 1971). Using a primed continuous infusion of ^{2}H-glucose, Romijn et al. (1993) showed that in the fasted state the rate of appearance (R_a) of glucose significantly increased when the exercise intensity was increased from 25% to 65% to 85% maximal oxygen uptake ($\dot{V}O_{2max}$). During low intensity exercise, the rate of disappearance of glucose equalled R_a and plasma glucose concentration remained constant. At moderate and high intensity exercise, the R_a glucose exceeded the uptake by the muscle, which resulted in a small increase in blood glucose concentrations at 65% and a substantial increase at 85% after 30 min of exercise (5.4 $mmol \cdot l^{-1}$ and 8.2 $mmol \cdot l^{-1}$ at 65% and 85% $\dot{V}O_{2max}$, respectively) (Romijn et al. 1993).

If exercise is commenced when plasma insulin concentrations are high, for example following a pre-exercise CHO load, the metabolic response to exercise is characterized by a rapid decline in blood glucose during the first 10–20 min of exercise (Ahlborg and Felig 1977; Costill et al. 1977; Coyle et al. 1985; Febbraio et al. 2000a; Febbraio and Stewart 1996; Fielding et al. 1987; Foster et al. 1979; Marmy-Conus et al. 1996; Sparks et al. 1998). The decrease in plasma glucose concentration is the result of the additional effects of the high insulin concentration and increased glucose uptake by the exercising muscle. In several studies (Coyle et al.

1985; Koivisto et al. 1981) these superimposed effects were of such a magnitude that the blood glucose concentration decreased to hypoglycaemic levels.

Most studies of the effects of pre-exercise ingestion of CHO have been performed at intensities eliciting 70% $\dot{V}O_{2max}$. While in some of these studies hypoglycaemia was reported (Coyle et al. 1985; Koivisto et al. 1981), in other studies glucose concentrations did not decrease markedly (Costill et al. 1977; Hargreaves et al. 1987; Sherman et al. 1989; Sparks et al. 1998). The results of studies where subjects were asked to exercise at higher intensities (more than 80% $\dot{V}O_{2max}$) are also inconclusive. In some studies, glucose concentration did not change or increased (Bonen et al. 1981; Neufer et al. 1987), while others have reported a decrease in glucose concentration during the first 15 min of exercise (Bonen et al. 1980; Foster et al. 1979; McMurray et al. 1983). The studies that investigated the glucose response during low intensity exercise did not describe the changes in glucose concentration during the first hours of exercise (Ahlborg and Felig 1977; Greenhaff et al. 1987). So, at present, it is not clear whether the occurrence of hypoglycaemia after the pre-exercise ingestion of CHO depends on the exercise intensity. Hence, the aim of the present study was to investigate the glycaemic and insulinaemic responses during exercise at different intensities, 45 min after ingestion of a 75 g CHO load.

Methods

Subjects

Eight healthy, moderately trained men participated in this study, which was approved by the local Ethics Committee. Prior to participation, the health of the subjects was assessed from the responses to a general health questionnaire. Each volunteer gave his written informed consent after explanations of the experiment procedures, and possible risks and benefits. The subjects were all club/county standard endurance athletes with a training background of at least 3 years. The characteristics of the subjects are shown in Table 1.

General design

All the studies in this series used the same basic protocol. A brief outline of that for the present study is given below with particular attention being given to the deviations from the basic protocol. For a detailed description of the basic protocol the reader is referred to Jentjens et al. (2002). Before the start of the experiments, the subjects were familiarized with the equipment and the procedures.

The subjects were asked to perform a graded exercise test until they were exhausted to determine their maximal power outputs ($\dot{W}_{max}$) and $\dot{V}O_{2max}$. In addition, during this visit, body mass, body fat and height were determined as previously described (Jentjens et al. 2002). The subjects were then asked to visit the laboratory on three more occasions. After arrival at the laboratory, the subjects consumed a drink containing 75 g of glucose. After 45 min of rest the subjects exercised for 20 min on a cycle ergometer at either 40%, 65% or 80% of their $\dot{W}_{max}$. The tests were performed at least 2, and not more than, 7 days apart. The order in which the exercise intensities were allotted was counterbalanced in six of the eight subjects; the remaining two subjects were randomly assigned an exercise intensity sequence that differed from one another. During exercise, blood samples were collected for plasma glucose, insulin and lactate concentration analyses and gas exchange measurements were performed.

Table 1 Subject characteristics

Age (years)	26.4 (2.9)
Height (cm)	179.2 (1.4)
Body mass (kg)	75.3 (1.6)
Percentage body fat (%)	14.3 (1.2)
Maximal power output (W)	360 (16)
Maximal oxygen uptake ($ml \cdot kg^{-1} \cdot min^{-1}$)	58.4 (1.9)

The trials

The subjects were asked to record their food intakes and activity patterns during the day prior to the first trial. The subjects were then instructed to repeat this diet and activity pattern during the day before all subsequent trials. In addition, subjects were asked not to perform any strenuous exercise on the day before each trial, and to abstain from drinking alcohol and using tobacco during this time. All experiments were performed in the morning (start of exercise between 8 and 10 a.m.) and for each subject at the same time of day to avoid the effects of variations in circadian rhythm.

Subjects reported to the Human Performance Laboratory in the morning (between 7–9 a.m.) after an overnight fast (10–12 h). After arrival at the laboratory, an initial blood sample was collected after which the subjects consumed a 15% CHO drink. The drink consisted of 75 g of glucose made up with distilled water to a volume of 500 ml. The subjects then began a 45 min rest period, during which three blood samples were collected (at $t = -30$ min, $t = -15$ min and $t = 0$). The subjects were then asked to mount an electromagnetically braked cycle ergometer and cycle for 20 min at either 40%, 65% or 80% $\dot{W}_{max}$. Blood samples were collected at 5 min intervals during this exercise. Heart rate was recorded continuously during the test using a radio telemetry heart rate monitor. Breath-by-breath measurements of expired gas were performed throughout exercise using an automated gas analysis system. The volume and gas analysers of the system were calibrated using a 3l calibration syringe and a calibration gas, respectively.

Analysis

Plasma glucose, insulin and lactate concentrations were determined as previously described (Jentjens et al. 2002). Hypoglycaemia was defined as a plasma glucose concentration below 3.5 $mmol \cdot l^{-1}$. The oxygen uptake ($\dot{V}O_2$), carbon dioxide production and minute ventilation data from the breath-by-breath analysis were averaged over 5 min intervals.

Statistics

Experiment data are expressed as means (SEM). Before statistical analysis, the variables were tested for normality at all times. A two-way general linear model for repeated measurements (intensity×time) was used to identify differences between the three different trials. In the event that sphericity was violated, the analyses were adjusted using a Greenhouse-Geisser correction. When a significant F-ratio was obtained, the Tukey post-hoc test was used to locate the differences. For all statistical analyses, significance was accepted at $P < 0.05$. Data evaluation was performed using a SPSS for Windows version 10.0 software package (Chicago, USA).

Results

The average $\dot{V}O_2$ during the 40%, 65% and 80% $\dot{W}_{max}$ trials were equal to 55 (1), 77 (1) and 90 (1)% $\dot{V}O_{2max}$,

respectively. Average heart rate was significantly ($P < 0.01$) higher with increasing exercise intensity, from 122 (2) beats·min^{-1} at 40%, to 150 (2) beats·min^{-1} at 65%, to 174 (2) beats·min^{-1} at 80% $\dot{W}_{max}$.

In Fig. 1, the respiratory exchange ratio (R) during exercise is shown. The R did not change from 5 to 20 min and was approximately 0.94 (0.00) and 1.01 (0.01) in the 40% and 65% $\dot{W}_{max}$ trials, respectively. In the 80% $\dot{W}_{max}$ trial, R was 1.11 (0.01) after 5 min of exercise, which was significantly higher than the R in both other trials. Thereafter, the R in the 80% $\dot{W}_{max}$ trial decreased significantly to 1.04 (0.00) at $t = 10$, where it remained for the rest of the trial. At all times, R was significantly lower during the 40% $\dot{W}_{max}$ trial compared to the two other trials ($P < 0.01$). At $t = 10$ and $t = 20$, R was significantly higher at 80% $\dot{W}_{max}$ compared to 65% $\dot{W}_{max}$ ($P < 0.05$).

In agreement with the R data, plasma lactate concentration was significantly higher in the 80% $\dot{W}_{max}$ trial compared to the other two trials after 5 min of exercise (Fig. 2). At the highest intensity, plasma lactate concentration increased significantly to 8.6 (1.0) mmol·l^{-1} at $t = 10$, and remained steady for the rest of the exercise period. No increase was observed in the plasma lactate concentration during the 40% $\dot{W}_{max}$ trial whereas at 65% $\dot{W}_{max}$, the plasma lactate concentration increased during the first 5 min after which it levelled off. Lactate concentration was significantly higher at 65% compared to 40% $\dot{W}_{max}$ from $t = 5$ until $t = 20$ ($P < 0.05$).

Plasma glucose concentrations were similar during exercise at all three intensities (Fig. 3). However, there was a significant main effect of time for plasma glucose concentration ($P < 0.05$). The glucose concentration increased significantly during the first 15 min of rest. The glucose concentration remained elevated for another 15 min after which it had returned to baseline concentration at the onset of exercise. The glucose concentration decreased significantly from 4.7 (0.3), 5.1 (0.3) and 4.8 (0.3) to 4.1 (0.2), 4.3 (0.3) and 4.1 (0.2) mmol·l^{-1} in the first 5 min of exercise in the 40%, 65% and 80% $\dot{W}_{max}$ trials respectively, after which no more marked changes in the concentrations were observed.

No differences were observed in plasma insulin concentrations at the three exercise intensities (Fig. 4). In all trials, 30 min after the ingestion of the glucose drink, plasma insulin concentration increased to levels that were five times as high as the fasting values. Thereafter, the insulin concentration decreased significantly, but remained above the fasting concentration at the start of exercise. During exercise, the concentration decreased to fasting levels and after 10 min of exercise the concentration became steady.

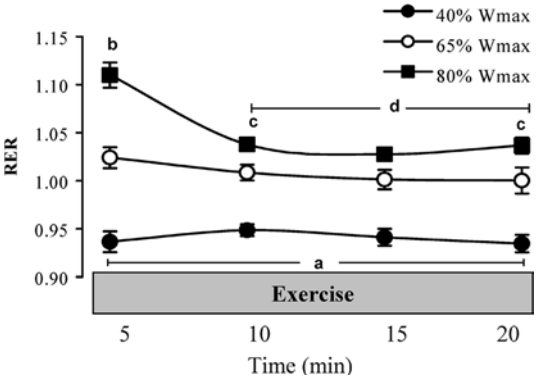

Fig. 1 Respiratory exchange ratio during exercise at 40%, 65% and 80% maximal power output ($\dot{W}_{max}$). Values are mean and SEM. [a]Significantly different from 65% $\dot{W}_{max}$ and 80% $\dot{W}_{max}$ ($P < 0.01$), [b]significantly different from 65% $\dot{W}_{max}$ ($P < 0.01$), [c]significantly different from 65% $\dot{W}_{max}$ ($P < 0.05$), [d]significantly different from values at five min ($P < 0.01$)

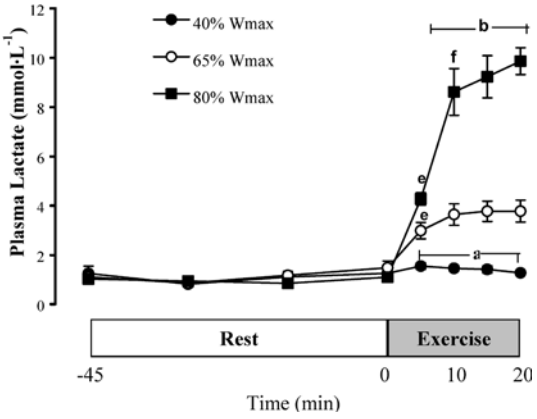

Fig. 2 Plasma lactate concentrations at rest and during exercise at 40%, 65% and 80% maximal power output ($\dot{W}_{max}$). Values are mean and SEM. [a]Significantly different from 65% $\dot{W}_{max}$ and 80% $\dot{W}_{max}$ ($P < 0.05$), [b]significantly different from 65% $\dot{W}_{max}$ ($P < 0.05$), [e]significantly different from values at 0 min ($P < 0.05$), [f]significantly different from values at five min ($P < 0.05$)

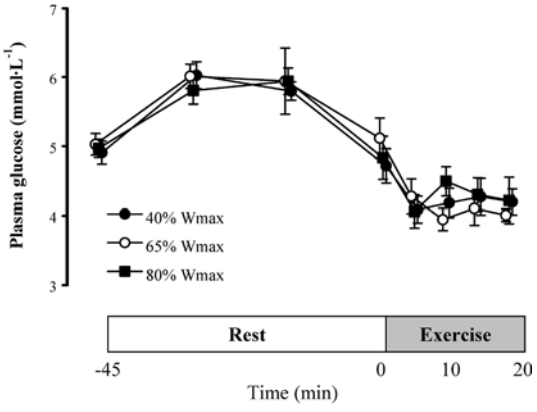

Fig. 3 Plasma glucose concentrations at rest and during exercise at 40%, 65% and 80% maximal power output ($\dot{W}_{max}$). Values are mean and SEM

Discussion

Over the last four decades numerous studies have been conducted that have investigated the effects of

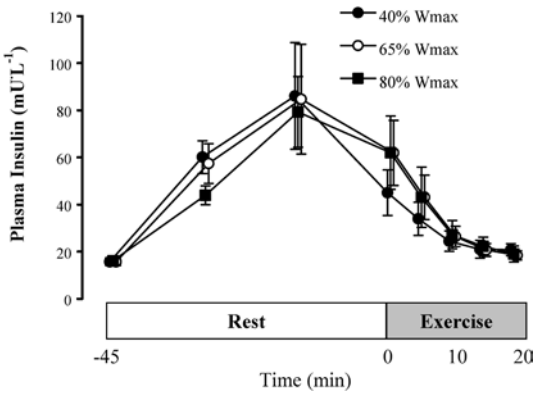

Fig. 4 Plasma insulin concentrations at rest and during exercise at 40%, 65% and 80% maximal power output ($\dot{W}_{max}$). Values are mean and SEM

pre-exercise ingestion of CHO on the occurrence of rebound hypoglycaemia and the effects this might have on performance. Unfortunately, the results of these studies have been inconclusive and no clear picture has emerged. It is likely that the discrepancies between these studies are caused by differences in the study designs, such as the type of subjects used (e.g. trained compared to untrained), the amount of CHO ingested, the timing of the ingestion, the type of CHO ingested, and the intensity of exercise. In a series of experiments in our laboratory, we have attempted to investigate systematically the effects of several of these factors on exercise metabolism and performance after ingestion of CHO. The present study was performed as one of this series and the aim was to investigate the effect of the exercise intensity on the changes that occur in insulin and glucose concentrations during subsequent exercise.

In all three trials, the ingestion of glucose before exercise significantly increased the plasma glucose concentration during the first 15 min after ingestion. The elevated glucose concentration then triggered an increase in plasma insulin concentration. At the onset of exercise the glucose concentration had returned to baseline levels in all three conditions, while the insulin concentration was still significantly elevated compared to pre-ingestion levels.

During all trials the subjects started exercise with similar insulin concentrations. The insulin concentration showed a significant drop during the first 10 min of exercise after which it levelled off at a concentration which was not significantly different from the values at rest. This pattern is also seen in other studies where exercise has been performed at moderate and high intensities (Bonen et al. 1980, 1981; Costill et al. 1977; Gleeson et al. 1986; Hargreaves et al. 1987; Koivisto et al. 1981; Sparks et al. 1998).

Plasma glucose concentration was also similar at the onset of exercise in all three trials. At all intensities, plasma glucose concentration decreased significantly during the first 5 min of exercise, after which the concentration became steady. During the 40% and 65% $\dot{W}_{max}$ trials, three subjects developed hypoglycaemia, while during the 80% $\dot{W}_{max}$ trial hypoglycaemia was found in four subjects. It appeared that two of the eight subjects were hypoglycaemic during exercise at all three intensities. On average, however, glucose concentration did not decrease to values below 3.5 mmol·l^{-1} during 20 min of exercise at either 40%, 65% or 80% $\dot{W}_{max}$.

In studies where exercise was performed at moderate intensities (67%–75% $\dot{V}O_{2max}$), a similar pattern in glucose concentration, with a drop in the first 10 min and steady concentrations thereafter, as in the present study, has been seen (Costill et al. 1977; Febbraio and Stewart 1996; Gleeson et al. 1986; Hargreaves et al. 1987; Koivisto et al. 1981; Sparks et al. 1998). The magnitude of the decrease in glucose concentration has not, however, been similar in all studies. In a study by Coyle et al. (1985) the subjects were asked to cycle at 70% $\dot{V}O_{2max}$ 4 h after they had consumed a high CHO breakfast. The glucose concentration had returned to baseline at the start of exercise and then dropped to 2.9 (0.2) mmol·l^{-1} after 20 min of exercise. The blood glucose concentration of the subjects in a study by Koivisto et al. (1981) decreased to a nadir of 2.5 (0.2) mmol·l^{-1} during exercise at 70% $\dot{V}O_{2max}$, which was started 45 min after the ingestion of 75 g of glucose. In contrast, in a study by Hargreaves et al. (1987) in which the subjects ingested 75 g of glucose 45 min before exercise, blood glucose concentration only decreased to 4.0 (0.3) mmol·l^{-1} during 15 min of exercise at 75% $\dot{V}O_{2max}$. Sherman et al. (1989) showed that blood glucose concentration decreased to 4.5 mmol·l^{-1} after 15 min of exercise when the subjects ingested 45 g of glucose before exercise, and it decreased to 4.2 mmol·l^{-1} after ingestion of 156 g of glucose. In studies where the exercise was performed at a higher intensity, hardly any change was seen in the plasma glucose concentration during the first 20 min of exercise (Bonen et al. 1980, 1981; Foster et al. 1979; McMurray et al. 1983; Neufer et al. 1987). The studies that investigated the effects of exercise intensities below 50% $\dot{V}O_{2max}$, did not report blood glucose concentration during the first few minutes of exercise (Ahlborg and Felig 1977; Greenhaff et al. 1987). So hypoglycaemia appears to occur in some studies (Coyle et al. 1985; Koivisto et al. 1981), but not in others (Ahlborg and Felig 1977; Costill et al. 1977; Febbraio et al. 2000b; Febbraio and Stewart 1996; Fielding et al. 1987; Foster et al. 1979; Marmy-Conus et al. 1996; Sparks et al. 1998). In the present study, hypoglycaemia was not found at any of the intensities. This suggests that the large variation found in the results of previously performed investigations cannot be explained by the different intensities used in those studies.

The present study was performed as one of a series of experiments investigating the effects of the pre-exercise ingestion of CHO on the metabolic responses during steady-state exercise and subsequent cycling performance. The main motive for performing these studies was the apparent lack of consistency in the results of

previous studies investigating the effects of pre-exercise ingestion of CHO. Differences in designs (type and timing of CHO ingestion, training status of subjects) make it difficult to compare the results of the studies and are possibly the causes for parts of the discrepancy. In an attempt to elucidate the effect of the pre-exercise ingestion of CHO on CHO metabolism and performance, the following aspects were systematically investigated; the amount (Jentjens et al. 2002) and type (Jentjens and Jeukendrup 2002b) of CHO ingested, the timing of the CHO ingestion (Moseley et al. 2002) and the intensity of the subsequent exercise. In addition, the relationship between insulin sensitivity and the occurrence of rebound hypoglycaemia was investigated (Jentjens and Jeukendrup 2002a). The studies all used the same control trial (75 g of glucose ingested 45 min before submaximal exercise at 65% $\dot{W}_{max}$ followed by a performance trial) and the characteristics of the subjects were very similar. This approach is unique in that it allowed us to make direct comparisons between these different studies.

Interestingly, on average, the intake of 25–200 g of CHO 45 min before exercise did not result in rebound hypoglycaemia (Jentjens et al. 2002). However, rebound hypoglycaemia was observed in some individuals. The amount of CHO ingested did not seem to influence the incidence of hypoglycaemia as the number of individuals showing hypoglycaemia was fairly evenly distributed amongst the different trials (six subjects after 25 g, four subjects after 75 g, six subjects after 200 g of glucose; Jentjens et al. 2002). Also, in the present study, which looked at the prevalence of hypoglycaemia at different exercise intensities, hypoglycaemia occurred in a similar number of subjects in all three trials (three subjects at 40%. three subjects at 65%, four subjects at 80%).

The occurrence of rebound hypoglycaemia seems to be related to the glycaemic index of the ingested CHO. When the ingestion of glucose was compared with galactose and trehalose (both low-glycaemic index CHO) the number of subjects that developed hypoglycaemia tended to be different (four subjects in the glucose trial compared to one subject in the trehalose trial and none in the galactose trial, Jentjens and Jeukendrup 2002b). In addition, when the timing of the ingestion was increased from 15, to 45 and even 75 min before the commencement of exercise in another study, more subjects appeared to develop hypoglycaemia (two subjects after 15 min, three after 45 min and five after 75 min; Moseley et al. 2002). So, although the group results indicate that none of the investigated variables had a significant influence on the development of hypoglycaemia, individual results seem to suggest that both the timing of the ingestion and the glycaemic index of the CHO may be important factors.

A general observation from all the studies was that some individuals were more prone to develop rebound hypoglycaemia than others. For example, in the study by Moseley et al. (2002) the subjects who became hypoglycaemic when CHO was ingested 15 min before exercise, also became hypoglycaemic when CHO was ingested 45 and 75 min before the onset of exercise. Differing degrees of insulin sensitivity among individuals has been suggested as a possible explanation for the selective occurrence of rebound hypoglycaemia (Kuipers et al. 1999). However, in a study recently performed by Jentjens and Jeukendrup (2002a), it was shown that there appears to be no correlation between the insulin sensitivity of the subject and the prevalence of hypoglycaemia. It therefore remains to be determined what factors are associated with the occurrence of rebound hypoglycaemia.

The second important finding of this series of studies is the fact that none of the interventions affected cycling time-trial performance, which was measured immediately after the 20 min of steady-state exercise (Jentjens et al. 2002; Moseley et al. 2002; Jentjens and Jeukendrup 2002b). Even in the subjects that developed hypoglycaemia no change in performance could be detected.

In summary, this study showed no differences in plasma glucose or insulin concentrations during exercise at intensities ranging from 55% to 90% $\dot{V}O_{2max}$. Furthermore, an equal number of subjects developed rebound hypoglycaemia during low, moderate and high intensity exercise.

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