# The Effect of Pre-exercise Galactose and Glucose Ingestion on High-Intensity Endurance Cycling

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**ABSTRACT**

This study evaluated the effects of the pre-exercise (30 minutes) ingestion of galactose (Gal) or glucose (Glu) on endurance capacity as well as glycaemic and insulinaemic responses. Ten trained male cyclists completed three randomised high-intensity cycling endurance tests. Thirty minutes prior to each trial cyclists ingested 1 litre of either 40g of glucose, 40g of galactose, or a placebo in a double blind manner. The protocol comprised: 20 minutes of progressive incremental exercise (70% to 85% maximal power output ($W_{max}$)); ten 90 second bouts at 90% $W_{max}$, separated by 180 seconds at 55% $W_{max}$; 90% $W_{max}$ until exhaustion. Blood samples were drawn throughout the protocol. Times to exhaustion were longer with Gal (68.7±10.2 minutes, P=0.005) compared to Glu (58.5±24.9 minutes), with neither being different to placebo (63.9±16.2 minutes). Twenty-eight minutes following Glu consumption, plasma glucose and serum insulin concentrations were higher than with Gal and placebo (P<0.001). Following the initial 20 minutes of exercise, plasma glucose concentrations increased to a relative hyperglycaemia during the Gal and placebo, compared to Glu condition. Higher plasma glucose concentrations during exercise, and the attenuated serum insulin response at rest, may explain the significantly longer times to exhaustion produced by Gal compared to Glu. However, neither carbohydrate treatment produced significantly longer times to exhaustion than placebo, suggesting that the pre-exercise ingestion of galactose and glucose alone is not sufficient to support this type of endurance performance.

***Key Words:*** carbohydrate; galactose; glucose; endurance capacity; rebound hypoglycaemia; pre-exercise ingestion.

## INTRODUCTION

Pre-exercise muscle and liver glycogen concentrations are essential substrate sources during prolonged moderate to high-intensity exercise, as they are both directly associated with performance (1, 2). Athletes typically consume carbohydrates (CHO) prior to and/or during exercise as a means of improving performance and endurance capacity. The exogenous source of CHO is likely to maintain higher plasma glucose concentrations and a high rate of CHO oxidation, especially late in exercise when muscle and liver glycogen concentrations are becoming depleted (7). There is good evidence that actual blood glucose concentration and glucose flux into muscle to sustain energy demand during exercise is a primary determinant of capacity such that optimal rates are possible only at sufficient blood glucose concentration and adequate glycogen reserve (19). Pre-exercise strategies often include the consumption of CHO formulations that may include pure glucose or substances that are easily assimilated to glucose, within the hour before the commencement of exercise. The pre-exercise ingestion of glucose in comparison to placebo has been shown to have both negative (15, 28) and positive (40, 41) effects on endurance performance, as well as providing no additional benefit (3, 24, 43). Some of these findings may be reconciled since glucose ingestion can be associated with the occurrence of rebound hypoglycaemia during subsequent exercise (23, 29), which may limit the ability to enhance endurance performance.

There is evidence that not all individuals appear to be susceptible to rebound hypoglycaemia (23, 29) and some are even able to maintain endurance performance despite transient hypoglycaemic episodes (14). However, previous studies have variably defined hypoglycaemia, with thresholds of $<2.5$ mmol.l$^{-1}$ (14, 39), $\leq 3.0$ mmol.l$^{-1}$ (29, 33) and $\leq 3.5$

mmol.l$^{-1}$ (15, 23), making interpretation of susceptibility difficult. Defining rebound hypoglycaemia as a plasma glucose concentration ≤3.0 mmol.l$^{-1}$ is most appropriate in a continuous cycling context as it is consistent with definitions of symptomatic hypoglycaemia (12, 33).

Rebound hypoglycaemia is associated with the occurrence of hyperinsulinaemia, typically shown directly prior to exercise (6, 15). As a consequence of hyperinsulinaemia, hepatic glucose output (27) and fat oxidation (21) can be suppressed. These transient metabolic disturbances, at the start of exercise, as well as mechanisms that increase glycogen utilisation (6, 17), may explain why pre-exercise glucose does not always provide an additional benefit in comparison to placebo. Regardless, there now appears to be less concern about the efficacy and use of simple sugars in the hour prior to exercise (18, 22, 26) despite early reservations regarding rebound hypoglycaemia (6, 15). If rebound hypoglycaemia is relevant to endurance performance the use of other forms of CHO, such as galactose, with a low glycaemic index (GI: ~20), which has no primary insulin drive (37), may overcome the issue. Galactose consumption would therefore be unlikely to inhibit hepatic output, but maintain fat oxidation, protecting the absorbed CHO for later use. Further, the consumption of galactose within the hour before exercise may have the potential to pre-load the liver with newly synthesised glycogen, for subsequent release, as galactose has to be converted by the liver through the Leloir pathway. This is of particular importance as the liver is as important in sustaining high-intensity exercise as muscle glycogen (2).

Only three studies have investigated the effects of galactose on endurance performance (24, 31, 42). Of these, only one (24) has specifically examined the pre-exercise ingestion (45 minutes prior to exercise) of galactose. This showed that there were no significant differences in

time trial performance between galactose (mean 42.04 ± 1.4 minutes) and glucose (mean 41.05 ± 2.9 minutes). The use of a time trial protocol is physiologically valid (9) and is likely to detect small but potentially crucial differences. However, with a small sample size (n=8) this study may have lacked adequate statistical power (calculated to be ~0.3). Therefore, further research is required to establish whether or not galactose may be a useful alternative pre-exercise substrate.

The purpose of the study was to compare the effects of the pre-exercise ingestion (30 minutes prior to exercise) of galactose and glucose on endurance capacity using a high-intensity endurance cycling test, as well as the glycaemic and insulinaemic responses at rest and during exercise. The pre-exercise ingestion of galactose has been shown to produce greater stability in plasma insulin and glucose concentrations (24), a more progressive glucose oxidation response during exercise, as well as sparing pre-existing liver glycogen stores (34), which may be beneficial for prolonged exercise performance. Therefore, we hypothesised that an initial bolus of galactose 30 minutes before exercise would sustain high-intensity endurance cycling capacity more effectively than glucose. In addition, we hypothesized that the pre-exercise ingestion of galactose would reduce the occurrence of rebound hypoglycaemia compare to glucose ingestion.

## METHODS

### Experimental Approach to the Problem

Cyclists completed three experimental trials of a variable high-intensity endurance cycling test to exhaustion (35); each test separated by 7 days. This exercise protocol was chosen as competitive endurance cycling is characterised by high-intensity efforts interspersed with sustained steady state exercise (36, 44) rather than constant load tests. Each trial involved the ingestion (30 minutes before exercise) of either 40g of galactose (Gal, (D-galactose, Inalco,

Milano, Italy), 40g of glucose (Glu, D-glucose, Cargill, Manchester, United Kingdom) or a placebo (water), as 1 litre formulations, using a randomised, double blind experimental design. Each cyclist was randomly assigned the order they would complete the experimental trials and followed their fixed sequence. All formulations contained 26 $mmol \cdot L^{-1}$ sodium chloride, as well as sweetener and flavouring to blind the participants to each condition. None of the cyclists reported that they recognised the placebo.

## Subjects

Ten trained male cyclists, aged: 31 ± 7 years (range 24-44 years), with a body mass of 76.2 ± 5.0 kg, body fat percentage of 9.7 ± 4.6% (BOD POD®, COSMED USA, Inc., Chicago, Illinois, USA), $\dot{V}O_{2\ max}$ of 58.1 ± 5.6 $ml \cdot kg^{-1} \cdot min^{-1}$ and maximal power output ($W_{max}$) of 364.3 ± 20.5 watts (W) participated in this study. The cyclists had trained for ≥15 hours per week, for at least the last 3 years, were competitive club level road racers, and in a maintenance phase of training throughout the duration of the study. The experimental procedures were fully explained to each cyclist prior to the study and all cyclists provided written informed consent. The protocol employed during this investigation received institutional ethical approval and was performed in accordance with the ethical standards specified by the Declaration of Helsinki (1964).

## Procedures

### *Preliminary Testing*

Cyclists completed a familiarization ride to allow habituation to the laboratory equipment and procedures employed (35). A week later the cyclists completed a maximal incremental cycle test to volitional exhaustion to determine their individual $W_{max}$ (30). This preceded the

experimental trials by at least one week. $W_{max}$ was used to determine the relative exercise intensities to be undertaken by each cyclist during the experimental trials (*viz*. power output (W) at a given % $W_{max}$). All exercise testing was performed on a standardized adjustable road bicycle fitted with SRM Powermeters (SRM, Germany) mounted on an air braked cycle ergometer (Kingcycle® Ltd, High Wycombe, U.K.). The Kingcycle® was calibrated as described by Schabort et al. (38). The SRM Powermeters, calibrated prior to the study, enabled high precision (manufacturers technical error <0.5 %) measurements of power output (W).

***Experimental Trials***

Cyclists were asked to record their food intakes and activity patterns during the 72-h prior to the first experimental trial. They were then instructed to repeat this diet and activity pattern for the remaining trials. Cyclists were also instructed to refrain from any strenuous activity, alcohol or caffeine consumption in the previous 24-h.

Following an overnight fast (≥12-h) each cyclist commenced his experimental trials at the same time of day (between 0600 and 0900) to avoid any influence of circadian variability. All trials were performed under normal environmental conditions (temperature: 18 $^{o}$C, relative humidity; 60%). Upon arrival at the laboratory, a catheter was inserted into an antecubital vein for repeated blood sampling. Resting venous blood samples were drawn, at -5 minutes and -2 minutes prior to fluid consumption and analyzed for plasma glucose, plasma lactate and serum insulin concentrations.

Thirty minutes prior to exercise, participants consumed either the Gal, Glu or placebo formulation. Venous blood samples were drawn at 13, 18, 23 and 28 minutes following fluid consumption. Cyclists then completed four 5 minutes continuous progressive workload

increments corresponding to 70%, 75%, 80% and 85% $W_{max}$. This was followed by ten 90 second sprints at 90% $W_{max}$, separated by 180 seconds recovery at 55% $W_{max}$ (35). Venous blood samples were drawn and heart rate and ratings of perceived exertion (RPE) were recorded throughout the exercise period.

If a cyclist completed all 10 sprints, following a 180 seconds interval at 55% $W_{max}$, cycling to volitional exhaustion was undertaken at 90% $W_{max}$. Exhaustion was defined as an inability to maintain power output within 5 W of that required and an inability to restore this within 15 seconds despite verbal encouragement. The same criteria were applied constantly throughout the protocol to ensure the maintenance of the prescribed power outputs. No feedback on elapsed time or heart rate was provided to prevent potential bias from cyclists targeting previous times or heart rates.

***Blood Analyses***

Blood samples drawn for both plasma glucose and plasma lactate measurements were collected in fluoride oxalate containing tubes (Becton Dickinson, Oxford, UK), while those for serum insulin were collected in plain tubes (Becton Dickinson, UK). Blood samples were stored on ice and centrifuged with aliquots of plasma and serum then being analysed for selected metabolites. Plasma glucose (Glucose Oxidase kit, Siemens Healthcare Diagnostics Inc, New York, USA), plasma lactate (Lactate kit, Siemens and serum insulin (Insulin IRI kit, Seimens) concentrations, were analysed using a semiautomatic analyser (ADVIA Centaur® System, Bayer Diagnostics, Newbury, Berks, UK). The within-run precision (coefficient of variation) for plasma glucose, plasma lactate and serum insulin was 0.5 to 0.6%, 1.0 to 1.9% and 3.2 to 4.6%, respectively.

**Statistical Analysis**

Data were tested for normal distribution (Kolmogorov-Smirnov test) and are presented as mean ± SD. A Friedman two-way ANOVA by ranks was used for the analysis of differences in time to exhaustion between trials, as these data were not normally distributed. Where significance was detected *post hoc* analysis was performed using a Wilcoxon signed rank test with Bonferroni adjustment (alpha level of 0.0166 per test (0.05/3)). Statistical comparisons for time to exhaustion were made using Cohen's Effect size (ES) with threshold values for small (0.2), medium (0.6), large (1.2), very large (2.0) and extremely large (4.0) effects (20). A one-way ANOVA was used to assess whether there was an order effect of the randomization of the trials for time to exhaustion, as well as the mean of the total area under the curve (AUC) for plasma glucose and serum insulin. Two-way ANOVA for repeated measures was used to compare differences in blood related variables and heart rate over time and between conditions. Where significance was detected for both a one-way and two-way ANOVA *post hoc* analysis was performed using a paired t-test with Bonferroni adjustment to establish differences between conditions and condition and time interactions (alpha level of 0.0166 per test (0.05/3)). A Friedman two-way ANOVA by ranks was used to analyse differences in RPE over time as well as between conditions. A Wilcoxon signed rank test, with Bonferroni adjustment (alpha level of 0.0166 per test (0.05/3)) was used to analyse the interaction between time and condition. Only the eight participants who completed 65 minutes of the exercise protocol (period over which these variables are reported) had their blood related variables, heart rate and RPE evaluated using SPSS for Windows version 17 (Chicago, USA). A 0.95 level of confidence was predetermined to denote statistical significance ($P<0.05$).

## RESULTS

### Time to Exhaustion

The pre-exercise ingestion of Gal produced longer times to exhaustion than Glu [68.7 ± 10.2 minutes vs. 58.5 ± 24.9 minutes, ES = 0.68, P=0.005 (figure 1)]. In fact, when ranking individual responses to Gal and Glu all 10 cyclists produced longer times to exhaustion on Gal, with these improvements ranging from 1.4 to 47.1 minutes. There were no differences in time to exhaustion between placebo (63.9 ± 16.2 minutes) and glucose (ES = 0.36, P=0.0214) or placebo and galactose (ES = 0.27, P=0.0214). There were no differences between results according to the randomized order in which the three trials were completed, showing that there was no order effect. Eight out of 10 cyclists produced longer times to exhaustion on placebo compared to Glu, whilst only two cyclists performed worse than placebo on galactose. Overall there was a range of endurance times of 18.5 minutes to 85.9 minutes. Following the ingestion of Gal only one cyclist did not complete all ten 90% $W_{max}$ sprints fatiguing at 44 minutes. Two cyclists whilst taking Glu (one of which was the cyclist reported above) and one of these during placebo did not complete any of the sprints, fatiguing during the initial 20 minutes of progressive increases in exercise intensity. All cyclists were included in the non-parametric analysis of these data. Further, none of the cyclists reported any gastro-intestinal problems prior to or during the exercise period.

### Blood Analyses

Plasma glucose concentrations increased to 8.0 ± 1.0 $mmol \cdot L^{-1}$ over the 28 minutes prior to exercise following Glu ingestion (P<0.001), which were higher than Gal (5.4 ± 0.5 $mmol \cdot L^{-1}$, P<0.001) and placebo (4.9 ± 0.3 $mmol \cdot L^{-1}$ (P<0.001, figure 2A)). Following Gal ingestion, there was a smaller increase in plasma glucose concentration (0.5 $mmol \cdot L^{-1}$). After the onset of exercise, plasma glucose concentrations fell rapidly following the ingestion of Glu to a nadir of 3.9 ± 1.2 $mmol \cdot L^{-1}$, at 15 minutes into exercise. The decrease in plasma glucose concentrations

was less evident following Gal ingestion (nadir 4.4 ± 0.8 mmol·L$^{-1}$) at 15 minutes into exercise. During the placebo condition plasma glucose concentrations remained stable. Plasma glucose concentrations fell below the hypoglycaemic threshold of 3.0 mmol·L$^{-1}$ (12, 33) during the initial 20 minutes of exercise for three cyclists during the Glu trial, though they did not report any symptoms and only one cyclist was unable to continue exercising. After the first 20 minutes of exercise, plasma glucose concentrations increased to slightly above baseline concentrations by 59 minutes (5.5 ± 1.3 mmol·L$^{-1}$) for Glu, with stability thereafter. During the galactose condition no cyclists had plasma glucose concentrations below the hypoglycaemic threshold. Following Gal and placebo ingestion plasma glucose concentrations increased to a relative hyperglycaemia by 59 minutes (6.7 ± 1.5 mmol·L$^{-1}$ and 6.9 ± 1.2 mmol·L$^{-1}$, respectively) and subsequently decreased to values comparable to the Glu condition. Even though there were different glycaemic patterns of response between conditions, there were no significant differences in the mean plasma glucose AUC between conditions (placebo: 486.8 ± 113.8 mmol·L$^{-1}$·min, Glu: 487.8 ± 119.2 mmol·L$^{-1}$·min, Gal: 509.0 ± 62.6 mmol·L$^{-1}$·min).

Figure 2B shows that following the ingestion of Glu, serum insulin concentrations significantly ($P<0.001$) increased to peak values of 29.6 ± 15.2 μU·mL$^{-1}$ at 28 minutes, which were higher than insulin concentration following Gal (12.5 ± 6.2 μU·mL$^{-1}$, $P<0.001$) and placebo (4.5 ± 1.9 μU·mL$^{-1}$, $P<0.001$). Concentrations following Gal were also higher than placebo ($P=0.001$). Throughout the initial 20 minutes of exercise serum insulin concentrations decreased towards basal values for Glu and Gal. Serum insulin was higher throughout this period during the Glu trial, though only significantly so at 35 minutes (19.0 ± 11.9 μU·mL$^{-1}$) in comparison to Gal (6.2 ± 3.5 μU·mL$^{-1}$, $P=0.003$) and placebo (3.6 ± 1.5 μU·mL$^{-1}$, $P=0.007$). Thereafter, values then converged and remained relatively stable until the end of exercise. The different insulinogenic

responses between conditions produced differences in the mean serum insulin AUC between conditions, with Glu (897.8 ± 3.15.0 $\mu U \cdot mL^{-1} \cdot min$) being significantly greater than placebo (339.4 ± 172.8 $\mu U \cdot mL^{-1} \cdot min$, $P<0.001$) and Gal (513.1 ± 170.4 $\mu U \cdot mL^{-1} \cdot min$, $P=0.011$) as well as Gal being significantly greater than placebo ($P=0.011$).

Plasma lactate concentrations changed over time and increased from typical resting values after the initial period of exercise ($P<0.001$), and then decreased for the remainder of the prescribed part of the exercise protocol (figure 2C). Plasma lactate concentrations were higher for Gal (1.8 ± 0.7 $mmol \cdot L^{-1}$) in comparison to Glu (1.2 ± 0.4 $mmol \cdot L^{-1}$, $P=0.002$) and placebo (1.1 ± 0.3 $mmol \cdot L^{-1}$, $P=0.013$) at 28 minutes. Glu produced higher plasma lactate concentrations in comparison to placebo at 72.5 minutes (9.4 ± 3.7 vs. 7.7 ± 3.4 $mmol \cdot L^{-1}$, $P=0.009$).

**Heart Rate and Ratings of Perceived Exertion.**

Both heart rate (figure 3A) and RPE (figure 3B) showed a predictable pattern of physiological responses consistent with exercise at different intensities, with no significant main effect of condition or interactions between conditions and time.

## DISCUSSION

The present study demonstrates that the chance of improving performance following the ingestion of carbohydrate within the hour of exercise commencing was small in comparison to placebo. However, the pre-exercise ingestion of galactose was more effective at maintaining endurance capacity in comparison to glucose, producing greater times to exhaustion on all occasions. Galactose ingestion produced similar glycaemic responses to placebo during exercise, maintaining superior plasma glucose concentrations during the initial phase of the sprints

compared to glucose. In contrast, 30% of the cyclists were susceptible to rebound hypoglycaemia during the initial 20 minutes of exercise following glucose ingestion, however, only one cyclist was sensitive to this and was unable to continue exercising.

Pre-exercise ingestion of Gal and Glu did not produce a significant endurance capacity advantage in comparison to placebo. The latter is in agreement with (22, 43) and also in contrast with previous literature (28, 41). The variation in time to exhaustion within conditions was relatively large (CV: placebo 25.4%, Glu 13.3%, Gal 14.8%), which may explain why there are no significant differences from the placebo condition for either CHO condition. Nevertheless, Gal had a mean difference of +4.7 minutes and Glu -5.5 minutes compared to placebo which could be considered physiologically significant. Accordingly, this study supports previous concerns over the consumption of glucose as a pre-exercise formulation in comparison to a placebo (15, 28) for some individuals.

The performance advantage of Gal compared to Glu is contrary to the findings of Jentjens and Jeukendrup (24). However, that study used a different carbohydrate dose (75g *vs*. 40g) and exercise protocol in comparison to the present study (time trial *vs.* time to exhaustion). The high-intensity time to exhaustion protocol used in the present study, though less ecologically valid, was deliberately designed to test differences between metabolic aspects of fatigue, which is not possible with a time trial design. Time to exhaustion protocols have the advantage of providing a controlled environment for the comparison of metabolic variables (10), such as plasma glucose concentrations, compared to time trials, which are likely to produce variability in exercise intensity. Furthermore, though time trial designs are likely to detect small and potentially crucial differences, when combined with small sample sizes may lack adequate statistical power. The

present study produced statistical power of 0.5, which is higher than the 0.3 reported by Jentjens and Jeukendrup (24).

The distinct peak in plasma glucose concentration following Glu ingestion, during the pre-exercise period (~8 mmol·$L^{-1}$), was higher in comparison to plasma glucose concentration attained following Gal. This is to be expected since plasma glucose concentrations should rise following ingestion and absorption of just glucose, but galactose, after absorption, can only be a precursor for plasma glucose upon specific metabolism. Circulating glucose enters the muscle cell directly where it is converted to glucose-6-phosphate. This may at rest provide sufficient substrate for glycogen synthesis but also is "demand led" during exercise to support glycolytic flux for energy production. In contrast, on entering the circulation, ingested galactose is preferentially taken up by the liver (45), prior to conversion to glucose-1-phosphate through the Leloir pathway. Glucose-1-phosphate is then available for the formation of glycogen in the liver or is released as free glucose. This may explain the lower postprandial plasma glucose concentrations in the pre-exercise period, which is likely to be almost exclusively the controlled release of glucose, formed from galactose by the liver, into the circulation (16). This is consistent with increases in plasma glucose concentrations of no more than 1 mmol·$L^{-1}$ reported within a galactose tolerance test (50g (16)) or 75g of galactose consumed 45 minutes prior to exercise (24).

The decline in plasma glucose concentrations, albeit from different absolute concentrations, following the appearance of exogenous glucose or galactose reflects a change in glucose flux into the muscle during the initial 20 minutes of exercise but the prevalence and change was different for each substrate. Hyperinsulinaemia following glucose prior to exercise

combined with an effect of increased contractile activity upon exercise on muscle glucose uptake would combine to accelerate disposal of plasma glucose (13, 32) at a time when glucose production from the liver would be inhibited. During exercise such imbalance between production and disposal, if continued, would lead to low glucose concentrations that are insufficient to support muscle glucose uptake (27). There is also potentially, as a consequence of hyperinsuliaemia, inhibition of lipolysis (21) and also increased reliance on muscle glycogen stores (6, 17). All of these likely mechanisms theoretically may explain the significantly shorter times to exhaustion following pre-exercise ingestion of Glu compared to Gal, but do not seem to influence endurance capacity in comparison to placebo. This suggests that low plasma glucose concentrations overall may not be a concern with respect to matching performance on placebo, but would be a concern in that it did not improve performance. Even though plasma glucose concentrations might be sufficient in the early stages of moderate to high intensity exercise, some individuals may be more sensitive than others to this situation(8). Plasma glucose concentrations following galactose were far less sensitive to reduction and in contrast the decline was also not associated with hyperinsulinaemia. Therefore, the small decline was more likely a reflection of increased disposal of plasma glucose into the working muscle upon the initiation of exercise (32). Absence of hyperinsulinaemia is unlikely to have suppressed lipolysis and fat oxidation. Furthermore, as plasma glucose concentrations were very similar to those produced by placebo, Gal ingestion was unlikely to have affected hepatic glucose output during this period. Thus, the flux of glucose into the muscle is unlikely to be compromised following galactose ingestion. These differences between glucose and galactose in homeostatic balance may underlie differences in exercise capacity. Yet differences in individual sensitivity to plasma glucose concentration related to insulin concentration and receptor sensitivity may confound simple

interpretation and explanation of differences between glucose and galactose conditions. However, note that serum insulin AUC was far greater during glucose and under this condition exhaustion was likely to occur earlier than under galactose.

The mean plasma glucose concentrations following the pre-exercise ingestion of Glu (3.9 ± 1.2 mmol·$L^{-1}$) and Gal (4.4 ± 0.8 mmol·$L^{-1}$) did not fall below 3.0 mmol·$L^{-1}$, which has previously been defined as hypoglycaemic (12, 33), during the initial 30 minutes of exercise. However, 30% of the participants were shown to be below the threshold (3.0 mmol·$L^{-1}$) and thus susceptible to this rebound hypoglycaemia, following the pre-exercise ingestion of Glu. This adds to the evidence that rebound hypoglycaemia occurs, but not all individuals appear to be susceptible (23, 29). Individual responses appear very relevant. One cyclist in the present study only managed to complete 18.5 minutes, following the ingestion of Glu. In this case premature fatigue coincided with rebound hypoglycaemia. The cyclist was able to perform for 66.8 minutes following the ingestion of placebo. The other two cyclists were able to perform for 75.0 and 66.7 minutes despite rebound hypoglycaemia, demonstrating some individuals are more sensitive to low plasma glucose concentrations than others, as previously suggested (8). Therefore, even though research unequivocally supports the consumption of CHO during endurance exercise to sustain endurance capacity (25), it may not be to some individuals advantage to consume a glucose formulation 30 minutes prior to exercise. Additional research is warranted to evaluate those susceptible to rebound hypoglycaemia and whether this has a true effect on endurance performance, as well as gauging how many athletes are affected by this phenomenon.

A feature of the present study is the difference in the recovery of plasma glucose concentrations between the three conditions, following the initial 20 minutes of exercise. By the third repeat sprint, plasma glucose concentrations increased to a relative hyperglycaemia

following the ingestion of placebo and Gal, which was associated with a small increase in plasma insulin concentrations. The maintenance of higher plasma glucose concentrations following Gal and placebo during the repeated sprints could be explained by either a reduction in plasma glucose disposal compared to the Glu condition (unlikely due to the intensity of the exercise), or a relative increase in hepatic glucose production/or an adequate hepatic production of glucose. The latter hypothesis is particularly attractive for galactose since it is known to be an excellent precursor of liver glycogen compared to glucose (11). Therefore, it is possible that the pre-exercise ingestion of galactose produces during exercise, a situation where the liver is more able to maintain plasma glucose concentration a key determinant of muscle glucose uptake, as it is not associated with the effects of hyperinsulinaemia or other homeostatic imbalances. The lower plasma glucose concentrations, though not below basal concentrations, during the use of Glu are likely to be a consequence of the continued inhibition of hepatic glucose output due to the enduring effects of hyperinsulinaemia (27, 32). The differences in plasma glucose concentrations following galactose and glucose ingestion are unlikely to be related to intestinal absorption, as they both share the same transport mechanism. Both galactose and glucose are 'actively' transported across the brush border membrane into the cell primarily by the same sodium co-transport system (46). Therefore, the greater times to exhaustion shown for Gal may in part be related to the more effective availability of plasma glucose from subsequent hepatic production, especially as plasma glucose concentrations have been shown to be important with regards to endurance capacity (4, 5). The higher plasma glucose concentrations are likely to reflect more effective maintenance of glucose oxidation (19), especially as there is evidence that galactose can produce higher exogenous glucose oxidation rates during the latter stages of exercise in comparison to glucose (34). This by itself may postpone fatigue (7), alternatively it may reduce

reliance on endogenous glycogen stores which could sustain endurance performance more effectively than glucose in this scenario. However, these metabolic differences between glucose and galactose, do not explain why neither was able to produce greater performances to placebo, particularly galactose. Galactose was as effective as placebo at maintaining relatively high plasma glucose concentration during the initial repeated sprints. Therefore, with the accompanying rise in serum insulin concentrations similar to placebo, it might be logical to assume that the rate of glucose uptake by the exercise muscles was similar. This might explain why there were no performance differences between these two conditions, with galactose only able to match the normal 'situation'.

In conclusion, the present study showed that the pre-exercise consumption of galactose 30 minutes prior to exercise provided stable and higher plasma glucose concentrations throughout exercise for metabolism by the exercising muscles, as well as lower insulin responses. Further, this study has also highlighted that following pre-exercise ingestion of CHO 30% of the trained male cyclists were susceptible to rebound hypoglycaemia from Glu in comparison to Gal. The hypothesis that pre-exercise galactose ingestion would reduce the occurrence of rebound hypoglycaemia compared to glucose ingestion can then be accepted. Additionally, the hypothesis that pre-exercise galactose ingestion would produce significantly longer times to exhaustion during a high-intensity endurance capacity test in comparison to glucose can also be accepted. However, neither CHO treatment enhanced performance in comparison to placebo, which remains to be explained.

## PRACTICAL APPLICATIONS

The results of the present study show that the ingestion of CHO within the hour before exercise alone does not provide a performance capacity benefit. Therefore, if glycogen reserves are optimal then the consumption of CHO within the hour before exercise may not be required for such an endurance performance scenario.

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**Figures**

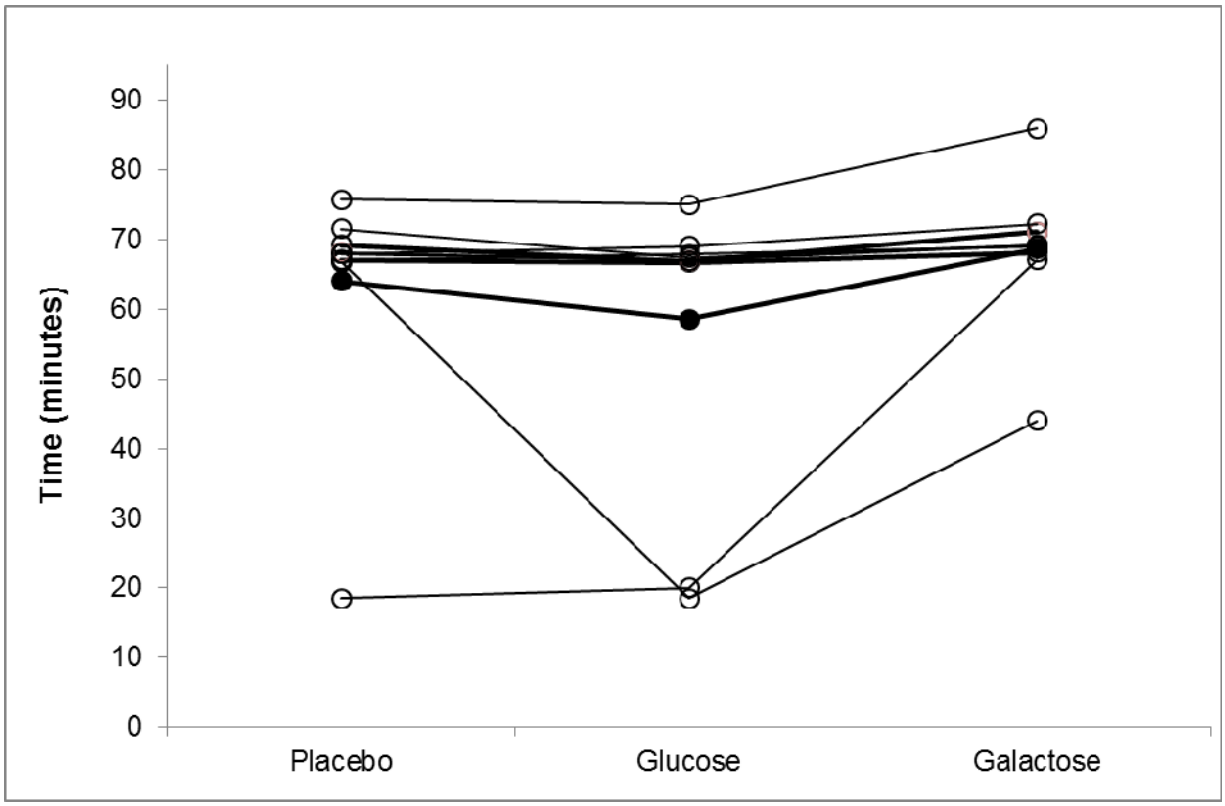


**Figure 1.** Individual time to exhaustion (n=10). *Solid Circles* represent the mean for each condition. *Gal significantly longer than Glu (P=0.005).

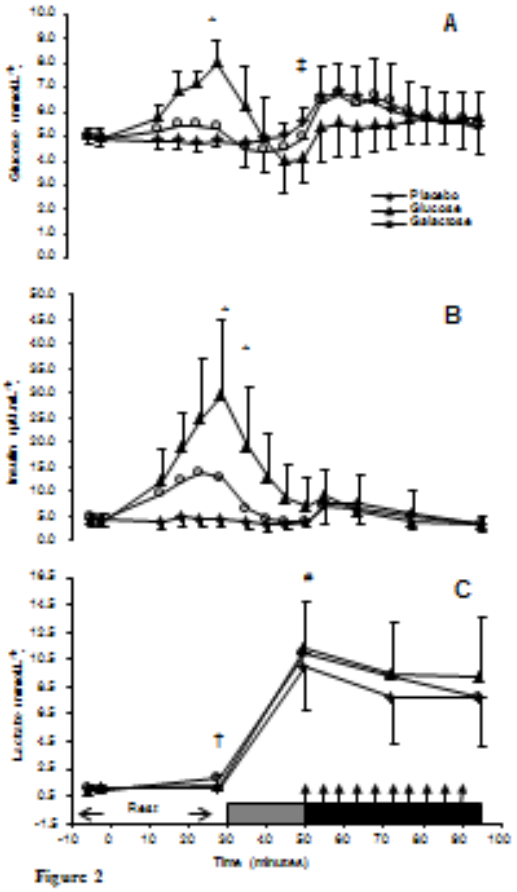


**Figure 2.** Plasma glucose (A) serum insulin (B) and plasma lactate (C) concentrations during the 30 min prior to exercise and during the initial 65 minutes of high-intensity cycling (n=10). *Grey rectangle* indicates 20 minutes of progressive intensity cycling from 70% to 85% $W_{max}$; *black rectangle* with arrows indicates the ten 90 s sprints at 90% $W_{max}$ interspersed with 180 s of active recovery at 55% $W_{max}$. * Glu significantly higher than Gal and placebo ($P<0.001$). † Gal significantly higher than Glu and placebo ($P=0.002$ and $P=0.013$, respectively). ‡ Placebo significantly higher than Gal and Glu ($P<0.001$). # Glu significantly higher than placebo ($P=0.009$).

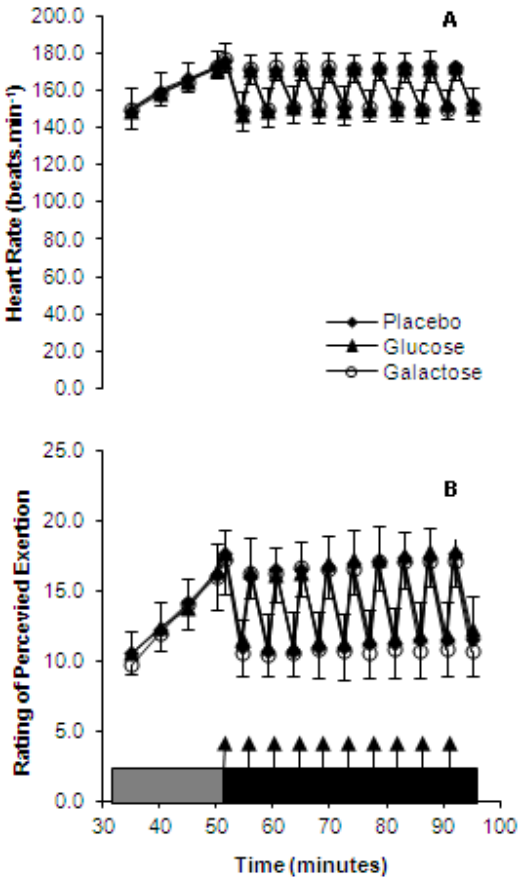


**Figure 3.** Heart rate (A) and ratings of perceived exertion (B) during the initial 65 minutes of high-intensity cycling (n=10). *Grey rectangle* indicates 20 minutes of progressive intensity cycling from 70% to 85% $W_{max}$; *black rectangle* with arrows indicates the ten 90 s sprints at 90% $W_{max}$ interspersed with 180 s of active recovery at 55% $W_{max.}$