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ARTICLE

Antecedent hypoglycaemia does not diminish the glycaemia-increasing effect and glucoregulatory responses of a 10 s sprint in people with type 1 diabetes

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Abstract
Aims/hypothesis A 10 s sprint has been reported to provide a means to prevent acute post-exercise hypoglycaemia in young adults with type 1 diabetes because of its glycaemia-raising effect, but it is unclear whether this effect is impaired by antecedent hypoglycaemia. The purpose of this study was to investigate whether antecedent hypoglycaemia impairs the glycaemia-raising effect of a 10 s sprint in individuals with type 1 diabetes.
Methods Eight individuals underwent a hyperinsulinaemic–hypoglycaemic or hyperinsulinaemic–euglycaemic clamp on two separate mornings. Thereafter, the participants underwent a basal insulin–euglycaemic clamp before performing a 10 s sprint on a cycle ergometer. The levels of blood glucose and glucoregulatory hormones and rates of glucose appearance (Ra) and disappearance (Rd) were compared between conditions.
Results During the morning clamps, blood glucose levels were significantly different between conditions of hypoglycaemia (2.8±0.1 mmol/l) and euglycaemia (5.4±0.2 mmol/l; $p<0.001$). Mean glycaemia prior to sprinting was similar (5.6±0.4 and 5.5±0.3 mmol/l for hypoglycaemic and euglycaemic conditions, respectively; $p=0.83$). In response to the afternoon sprint, the pattern of increase in blood glucose levels did not differ between conditions, reaching similar maximal levels 45 min after exercise (6.5±0.4 and 6.6±0.3 mmol/l, respectively; $p=0.43$). The early post-exercise patterns in glucose Ra and Rd and increases in plasma adrenaline (epinephrine), growth hormone and cortisol levels did not differ between conditions.
Conclusions/interpretation Hypoglycaemia in the morning does not diminish the glycaemia-raising effect of an afternoon 10 s sprint in young adults with type 1 diabetes, suggesting that sprinting is a useful strategy for opposing hypoglycaemia, regardless of prior hypoglycaemia.

Keywords Exercise · Hypoglycaemia · Sprint · Type 1 diabetes mellitus

Abbreviations

CGMS	Continuous glucose monitoring system
Ra	Rate of appearance
Rd	Rate of disappearance
MDI	Multiple daily insulin injections
$\dot{V}O_{2max}$	Maximal rate of oxygen consumption

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Introduction

Moderate-intensity exercise increases the risk of hypoglycaemia in individuals with type 1 diabetes [1–5], and the associated fear of hypoglycaemia prevents many individuals from enjoying the numerous benefits of a physically active lifestyle [6, 7]. However, not all forms of exercise cause blood glucose levels to fall in individuals with type 1 diabetes. For instance, prolonged high-intensity exercise (10–15 min at >80% of the maximal rate of oxygen consumption [$\dot{V}O_{2max}$]) performed under basal insulin conditions results in a significant and sustained increase in blood glucose level in individuals with type 1 diabetes [8–13]. This raises the possibility that intense aerobic exercise may provide a means to correct or prevent hypoglycaemia.

Exercising at this intensity for an extended period would not, however, be practical for most individuals. For this reason, we have recently investigated whether a 10 s maximal sprint effort may be useful for hypoglycaemia prevention. We have shown that in young adults with type 1 diabetes a 10 s maximal sprint performed before or after moderate-intensity exercise (40% $\dot{V}O_{2max}$) while plasma insulin levels are elevated prevents blood glucose from falling after exercise, thereby reducing their risk of exercise-mediated hypoglycaemia [14, 15]. More recently, we have also shown that performing a 10 s sprint under basal insulin conditions causes a significant and sustained rise in blood glucose level in young adults with type 1 diabetes [16]. These findings suggest that a short sprint may provide a useful approach for reducing the risk of exercise-mediated hypoglycaemia [5]. For these reasons the use of sprinting for hypoglycaemia prevention has been included in recent clinical practice guidelines published by the International Society for Pediatric and Adolescent Diabetes and the National Health and Medical Research Council of Australia [17–19]. However, it is our view that before advocating sprinting as a reliable tool for hypoglycaemia prevention, it is important to determine whether there are factors that could impair the glucoregulatory benefits of this type of exercise.

One factor that has the potential to affect the glycaemia-increasing effect of sprinting is a prior episode of hypoglycaemia. Antecedent hypoglycaemia has been shown to attenuate the glucoregulatory responses to subsequent moderate-intensity exercise, with a marked dampening of the rise in catecholamine, glucagon and cortisol levels [20–22]. If an episode of hypoglycaemia were also to reduce the glucoregulatory response to a subsequent sprint, the glycaemia-increasing effect of a 10 s sprint may be diminished, thus reducing the efficacy of sprinting in hypoglycaemia prevention. For these reasons, the primary purpose of this study was to examine the effect of morning hypoglycaemia on both the glucoregulatory and blood glucose responses to an afternoon 10 s sprint in young adults with type 1 diabetes.

Methods

Participants Eight young adults with type 1 diabetes were recruited to the study. Their characteristics are described in Table 1. All participants were on stable insulin regimens prior to the study and were not taking any medications other than insulin. The participants on multiple daily insulin injection (MDI) regimens were using glargine (A21Gly,B31Arg, B32Arg human insulin) and aspart (B28Asp human insulin) insulin analogues. The participants on insulin pump regimens were using aspart or lispro (B28Lys,B29Pro human insulin) insulin analogues. None of the participants had complications of diabetes or hypoglycaemia unawareness. They each received information about all experimental procedures prior to the study and gave informed consent in accordance with the Princess Margaret Hospital Ethics Committee.

Experimental procedures Prior to testing, all participants attended a familiarisation session where they were introduced to the team members and all equipment and procedures, and their anthropometric characteristics were measured. Before each of the following two testing sessions, participants were required to visit the research unit to be fitted with a continuous glucose monitoring system (CGMS) (Guardian REAL-Time CGM System; Medtronic, Northridge, CA, USA) and an accelerometer (Activity Monitor GT1M; ActiGraph, Pensacola, FL, USA) to continuously monitor interstitial fluid glucose levels and physical activity levels, respectively. The CGMS and accelerometer were worn for 3 days prior to each testing session to ensure that the participants did not experience any episodes of hypoglycaemia and were not engaging in any vigorous activity prior to testing; both being important precautions to take, since the glucoregulatory response to exercise is affected by antecedent hypoglycaemia and antecedent exercise [21, 23]. All CGMS readings ≤3.5 mmol/l were confirmed with a blood glucose measurement; testing was rescheduled if blood glucose levels were ≤3.5 mmol/l or if the participants experienced any symptoms of hypoglycaemia at any stage over this 3 day period. To ensure that the diet was matched between studies, the participants were required to

Table 1 Clinical characteristics of the study participants

Characteristic	Mean ± SD
N	8 (6 men, 2 women)
Age, years	21.3±2.6
Duration of diabetes, years	6.7±3.1
HbA_{1c}, % (mmol/mol)	7.7±1.0 (61±11)
BMI, kg/m^2	25.9±5.1
Insulin regimen	Pump (*n*=4) MDI (*n*=4)

keep a food diary for 24 h prior to their first study and then consume a similar diet the day before their second study. The day before each study, the participants on MDI regimens administered their long-acting insulin in the morning to ensure that little residual therapeutic insulin was present during the study. Finally, to account for the influence of the menstrual cycle on glucoregulation, the female participants were studied during the follicular phase of their cycle.

On the morning of testing, the participants arrived at the research unit at 08:00 hours following an overnight fast and were subjected on two separate occasions, and at least 3 weeks apart, to one of two experimental conditions administered following a randomised counterbalanced study design: antecedent hypoglycaemia or euglycaemia. A cannula was inserted into a vein in the dorsum of one hand for blood sampling, with this hand placed in a hotbox (CN370; Omega, Sydney, NSW, Australia) at ~60°C to arterialise venous blood prior to sampling. Another cannula was inserted into a vein in the contralateral antecubital fossa for the infusion of glucose, insulin and [6,6-^{2}H]glucose to determine glucose kinetics. A blood sample was then collected to determine the background enrichment of [6,6-^{2}H]glucose. Following this, insulin was infused at a constant rate of 80 mU m^{-2} min^{-1} as described previously [24]. A blood glucose target of 5.5 mmol/l was achieved within 90 min by varying the infusion rate of a 20% (wt/vol.) glucose solution [24]. At this point, blood samples were drawn to measure baseline levels of glucoregulatory hormones including insulin, glucagon, adrenaline (epinephrine), noradrenaline (norepinephrine), growth hormone and cortisol. Thereafter, either euglycaemia was maintained or hypoglycaemia was induced. In the euglycaemic condition, blood glucose levels were maintained at ~5.5 mmol/l for 90 min by adjusting the infusion rate of glucose guided by blood glucose measurements obtained every 5 min and analysed using a YSI analyser (Yellow Springs Instruments, Yellow Springs, OH, USA). In the hypoglycaemic condition, blood glucose levels were lowered over 30 min to a nadir of 2.8 mmol/l, which was maintained for a further 60 min. Following this, euglycaemia was restored once more by decreasing the insulin infusion rate and gradually reducing the glucose infusion rate. The depth and duration of this hypoglycaemic episode were chosen on the basis that they were expected to attenuate glucoregulatory hormone responses to a subsequent stimulus such as moderate-intensity exercise or hypoglycaemia [22, 25, 26]. At time intervals before and during euglycaemia and hypoglycaemia, blood samples were collected for the assessment of glucoregulatory hormone levels.

After euglycaemia was maintained or hypoglycaemia was induced, the infusion of insulin was replaced with the infusion of a 1:10 dilution of insulin in saline (154 mmol/l NaCl) (6 U insulin lispro [Eli Lily, Indianapolis, IN, USA] in 60 ml 0.9% saline). The insulin was titrated to achieve euglycaemia without any exogenous glucose. This procedure has been previously described by us and others and allows the determination of each participant's physiological basal insulin infusion rate [13, 16, 22]. At this point, the infusion of [6,6-^{2}H]glucose commenced with a priming bolus of 3 mg/kg and a constant infusion of 2.4 mg kg^{-1} h^{-1} that continued for the remainder of the experiment. The infusion of [6,6-^{2}H]glucose continued for at least 150 min prior to the commencement of the exercise to allow for isotopic equilibrium.

At approximately 15:00 hours, the participants performed a 10 s maximal sprint on a front-access cycle ergometer (Repco, Melbourne, VIC, Australia) as described previously [14, 15]. They then rested in a seated position for the remainder of the 60 min recovery period. At time intervals before and after the sprint, blood samples were collected for the determination of glucoregulatory hormone levels and the enrichment of [6,6-^{2}H]glucose.

Analyses Plasma lactate was measured using a YSI analyser (Yellow Springs Instruments). Heparinised plasma was assayed for free insulin using a non-competitive chemiluminescent immunoassay (Architect i2000SR; Abbott Laboratories, Abbott Park, IL, USA). EDTA-treated plasma collected with benzamidine HCl was assayed for pancreatic-specific glucagon using a competitive RIA (Linco Research, St Charles, MO, USA). EGTA-treated plasma collected with glutathione was assayed for adrenaline and noradrenaline by HPLC (UltiMate 2000; Thermo Fisher Scientific, Melbourne, VIC, Australia). Serum was assayed for growth hormone using a non-competitive enzyme immunoassay with a chemiluminescent substrate (Siemens Immulite 2000XPi; Siemens Medical Solutions, Pleasanton, CA, USA), and cortisol using a competitive chemiluminescent immunoassay (Abbott). Finally, [6,6-^{2}H]glucose enrichment was determined by gas chromatography-mass spectrometry (Bioanalytical Mass Spectrometry Facility, The University of New South Wales, Sydney, NSW, Australia). The readings obtained were corrected for background enrichment of naturally occurring [6,6-^{2}H]glucose, and the resultant values were smoothed to minimise the effect of random error of measurements on calculations of glucose kinetics [27]. The rates of glucose appearance (Ra) and disappearance (Rd) were calculated from the changes in glucose enrichment using the single compartment, fixed-volume, non-steady-state model proposed by Steele [28], as modified for use with stable isotopes [29] and a pool fraction of 0.65 [30].

Data were analysed using two-way repeated-measures ANOVA and Fisher's least significant difference test for posterior analysis using SPSS software (version 20.0; SPSS, Chicago, IL, USA). Statistical significance was accepted at $p<0.05$. Unless otherwise stated, all results are expressed as mean ± SEM. Our sample size was calculated based on

published data on the effect of intense exercise on blood glucose levels, glucose Ra, glucose Rd and the levels of the glucoregulatory hormones [8–12, 14–16]. This sample size was calculated with a power of $1-\beta=0.8$ and statistical significance was set at $p<0.05$. Cohen's d was calculated to determine the effect size of the blood glucose response to the sprint between hypoglycaemic and euglycaemic conditions.

Results

Over the 3 day period prior to each study, there were no reported hypoglycaemic events where sensor readings <3.5 mmol/l were confirmed with a blood glucose reading below this level or where the participants experienced symptoms of hypoglycaemia. Also, there was no difference between conditions in overall physical activity levels estimated from accelerometer counts ($39.4\pm2.4\times10^4$ and $46.0\pm4.9\times10^4$ prior to hypoglycaemia and euglycaemia, respectively; $p=0.89$).

During the morning clamps, blood glucose levels were significantly different between conditions (2.8±0.1 and 5.4 ±0.2 mmol/l during hypoglycaemia and euglycaemia, respectively; $p<0.001$; Fig. 1a). In response to 60 min of euglycaemia, there was a small but significant decrease in glucagon from baseline levels, but the levels of adrenaline, noradrenaline, cortisol and growth hormone did not change (Fig. 1b–f). By contrast, 60 min of hypoglycaemia resulted in significant increases in the levels of adrenaline, noradrenaline, cortisol and growth hormone from baseline ($p<0.05$; Fig. 1c–f).

During the 10 s sprint, there were no differences between conditions in the peak power output achieved (1,191.2±130.5 and 1,139.8±110.8 W following hypoglycaemia and euglycaemia, respectively; $p=0.39$) or the total work performed (8,244.3±780.2 and 8,134.9±773.3 J following hypoglycaemia and euglycaemia, respectively; $p=0.62$). Mean blood glucose levels prior to the afternoon 10 s sprint were similar (5.6±0.4 and 5.5±0.3 mmol/l following hypoglycaemia and euglycaemia, respectively; $p=0.83$; Fig. 2a). In response to the sprint, blood glucose levels increased significantly in both experimental groups, reaching similar maximal levels 43 min after exercise (6.5±0.4 and 6.6±0.3 mmol/l following hypoglycaemia and euglycaemia, respectively; $p=0.43$; Cohen's effect size $r=0.08$; Fig. 2a). There was no statistically significant interaction between the

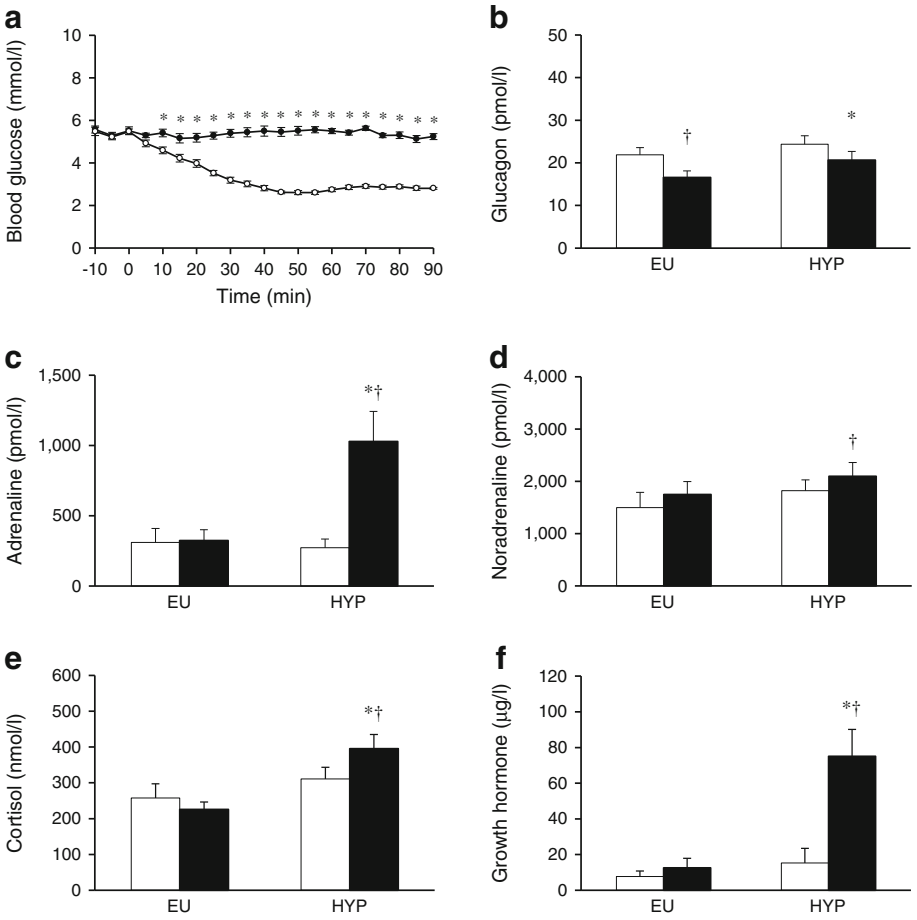

Fig. 1 Responses of blood glucose (**a**), plasma glucagon (**b**), adrenaline (**c**), noradrenaline (**d**), serum cortisol (**e**) and serum growth hormone (**f**) to morning euglycaemia (EU; black circles) and hypoglycaemia (HPY; white circles). White bars, baseline; black bars, after 60 min. *$p<0.05$ for euglycaemic vs hypoglycaemic conditions; †$p<0.05$ vs baseline levels

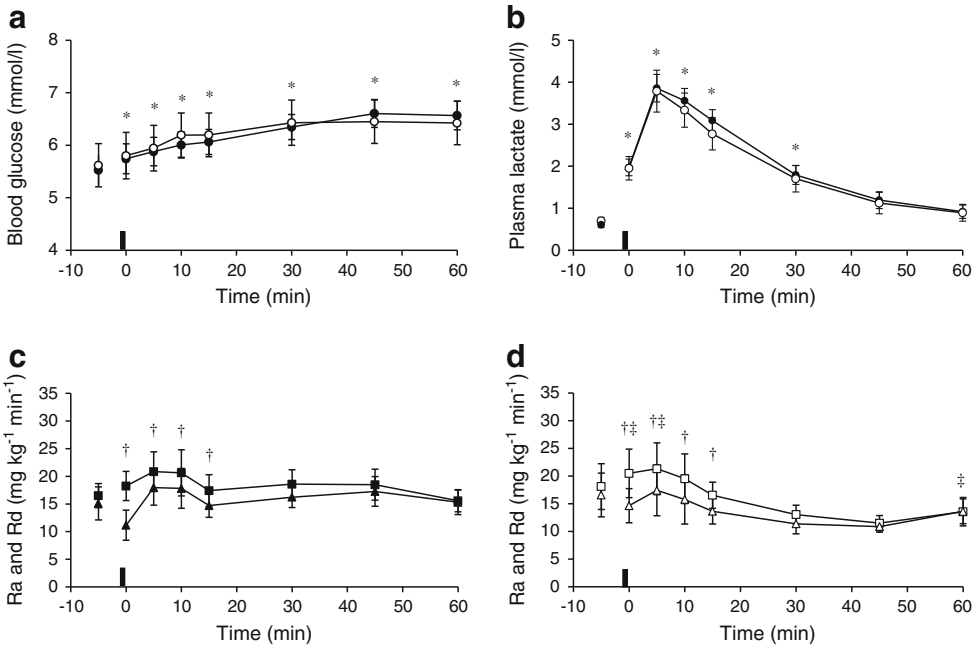

Fig. 2 Blood glucose (**a**) and plasma lactate (**b**) responses to a 10 s sprint following morning euglycaemia (black circles) and hypoglycaemia (white circles), and the effect of a 10 s sprint on glucose Ra (squares) and Rd (triangles) following euglycaemia (**c**) and hypoglycaemia (**d**). Vertical bar, sprint; $^{*}p<0.05$ vs baseline levels; $^{\dagger}p<0.05$ for Ra vs Rd; $^{\ddagger}p<0.05$ for Ra vs baseline levels

effects of time and treatment on blood glucose levels ($F=0.39$, $p=0.56$). Similarly, mean plasma lactate levels were comparable between conditions prior to the sprint (0.7±0.1 and 0.6±0.1 mmol/l following hypoglycaemia and euglycaemia, respectively; $p=0.37$; Fig. 2b). In response to the sprint, plasma lactate levels increased significantly in both experimental groups reaching similar peak levels 5 min after exercise (3.8±0.5 and 3.9±0.3 mmol/l following hypoglycaemia and euglycaemia, respectively; $p=0.85$), before returning to baseline levels after 45 min of recovery (Fig. 2b).

Prior to the sprint, there were no differences between glucose Ra and Rd in either condition nor were there any differences in these variables between conditions ($p>0.05$; Fig. 2c, d). In response to the sprint, glucose Ra increased above baseline levels in both conditions before returning to pre-exercise levels within 15 min. During this early post-exercise period, glucose Ra exceeded glucose Rd in both conditions ($p<0.05$; Fig. 2c, d). Later during recovery, there were no differences between glucose Ra and Rd in either condition; however, both glucose Ra and Rd were higher following euglycaemia (Fig. 2c, d).

Plasma insulin levels prior to the sprint were 95.6±22.7 and 99.7±23.0 pmol/l following hypoglycaemia and euglycaemia, respectively ($p=0.59$), and did not change in either condition following the sprint (Fig. 3a). There was a small but significant increase in glucagon above pre-exercise levels following euglycaemia ($p=0.02$) that returned to pre-exercise levels during early recovery. There was no change in glucagon from pre-exercise levels following hypoglycaemia ($p>0.05$; Fig. 3b).

In response to the 10 s sprint, adrenaline, noradrenaline and cortisol increased above baseline levels in both conditions, and growth hormone increased above baseline levels in the hypoglycaemic condition before returning to pre-exercise levels during early recovery ($p<0.05$). However, there were no significant differences between conditions except for a difference in noradrenaline levels immediately after the sprint (Fig. 3c–f).

Discussion

Although there is evidence that sprinting may be beneficial for hypoglycaemia prevention in young adults with type 1 diabetes [5, 14–16, 31, 32], factors that may impair the glucoregulatory benefits of sprinting must be identified before recommending this type of exercise in clinical practice. Since an episode of hypoglycaemia can reduce the glucoregulatory response to a subsequent bout of moderate-intensity exercise [21, 22], the purpose of this study was to investigate the possibility that antecedent hypoglycaemia impairs both the glucoregulatory and glycaemia-increasing responses to a 10 s sprint. This study shows that morning hypoglycaemia does not attenuate the glycaemia-increasing effect of a 10 s sprint performed in the afternoon and has little or no effect on the glucoregulatory response to this type of exercise in young adults with type 1 diabetes. Although we acknowledge that the small sample size described here is a limitation of this study, it is important to note that even if the small differences in our blood glucose readings after sprinting had been statistically significant, the associated Cohen's effect size was far too low (0.08) to be clinically significant.

The finding that antecedent hypoglycaemia has little or no effect on the glucoregulatory response to sprinting was

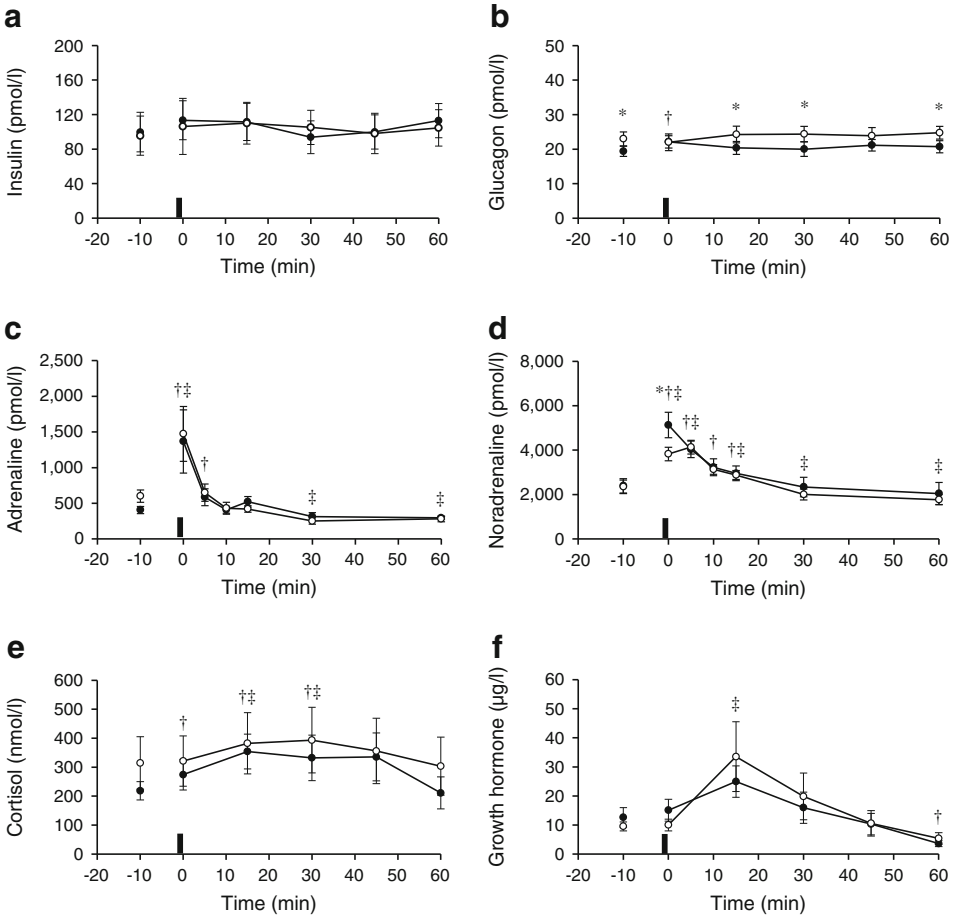

Fig. 3 Responses of plasma insulin (**a**), plasma glucagon (**b**), adrenaline (**c**), noradrenaline (**d**), serum cortisol (**e**) and serum growth hormone (**f**) to a 10 s sprint following morning euglycaemia (black circles) and hypoglycaemia (white circles). Vertical bar, sprint; $^{*}p<0.05$ for euglycaemic vs hypoglycaemic conditions; $^{\dagger}p<0.05$ for euglycaemia vs baseline levels; $^{\ddagger}p<0.05$ for hypoglycaemia vs baseline levels

unexpected considering the findings of Galassetti and colleagues [21, 22], who reported that antecedent hypoglycaemia reduces the glucoregulatory response to moderate-intensity exercise in individuals with type 1 diabetes. In particular, they showed that although antecedent hypoglycaemia has no effect on growth hormone responses to moderate-intensity exercise it results in diminished adrenaline, noradrenaline, glucagon and cortisol responses [21, 22]. In the present study, contrary to these findings, antecedent hypoglycaemia did not affect the post-exercise responses of adrenaline, growth hormone, glucagon and cortisol levels to sprinting, with the exception of noradrenaline levels, which increased to a lesser extent at the onset of recovery. However, there are several important differences between this study and the studies by Galassetti and colleagues [21, 22] that might have contributed to the differences in the glucoregulatory responses to exercise. First, Galassetti et al investigated the effect of antecedent hypoglycaemia on the glucoregulatory response during and not after moderate-intensity exercise. Second, the participants in these earlier studies were exposed to two consecutive episodes of antecedent hypoglycaemia and were exercised the following day, whereas our participants were subjected to a single hypoglycaemic episode and were exercise-tested on the same day. Although others have shown that antecedent hypoglycaemia reduces counterregulatory responses to subsequent hypoglycaemia as little as 2 h later [26], our finding that antecedent hypoglycaemia does not result in a short-term deficit in glucoregulatory and blood glucose responses to sprinting does not exclude the possibility that hypoglycaemia may impact these responses to sprinting later on. More research is required to address this possibility.

The absence of any effect of antecedent hypoglycaemia on the pattern of post-exercise rise in glycaemia in response to sprinting is unlikely to be due to the hypoglycaemic stimulus being insufficient to induce a deficit in subsequent glucoregulatory hormone responses. This is because the magnitude of the counterregulatory responses to hypoglycaemia reported here was comparable to that found in previous studies [24, 33]. Furthermore, the severity of hypoglycaemia in this study was chosen to match that of earlier studies which have reported a hypoglycaemia-mediated attenuation of the glucoregulatory response to moderate-intensity exercise [21, 22].

Perhaps our findings could be explained on the grounds that the intensity of the sprint was sufficient to override any detrimental impact antecedent hypoglycaemia may have on the glucoregulatory response to sprinting. Consistent with this interpretation is the observation that the increases in

adrenaline and noradrenaline levels were more pronounced after sprinting than in response to moderate-intensity exercise [21, 22]. It is also possible that the low insulin levels reported here during and after the sprint permit a glucose response irrespective of whether the sprint is preceded by hypoglycaemia.

The early post-sprint rise in blood glucose levels associated with the euglycaemic and hypoglycaemic conditions is similar to that recently described in a study investigating the effect of sprinting on blood glucose level in individuals with type 1 diabetes [16], and it results from a mismatch between glucose Ra and Rd. For both experimental conditions, sprinting resulted in a comparable post-exercise rise in glucose Ra in conjunction with a trend for a transient fall in glucose Rd early post sprint. The resulting mismatch between glucose Ra and Rd accounted for the rise in blood glucose levels. As recovery progresses, the eventual stabilisation of blood glucose levels under both experimental conditions was associated with the matching of glucose Ra and Rd. It is noteworthy, however, that glucose Ra and Rd later during recovery were significantly higher in the euglycaemic than in the hypoglycaemic condition. Although the effect of antecedent hypoglycaemia on the patterns of glucose Ra and Rd during recovery from other exercise modalities has not been investigated in type 1 diabetes, Galassetti and colleagues [21, 22] showed that endogenous glucose Ra increases to a lesser extent during exercise performed after a hypoglycaemic episode.

The pronounced rise in plasma catecholamines immediately after sprinting and the subsequent increase in growth hormone levels suggest that these hormones may play some role in mediating the increase in glycaemia and glucose Ra at the onset of recovery. This increase in plasma catecholamines may contribute to the rise in blood glucose via stimulation of glucose Ra [9, 34], with increases in both adrenaline and noradrenaline associated with increased rates of hepatic glucose production [10, 35, 36]. However, the observation that a lesser rise in noradrenaline level immediately after the sprint in the hypoglycaemic condition did not affect the increase in blood glucose levels after sprinting suggests that this hormone may play a less significant role in the increase in both glucose Ra and blood glucose levels. It is also possible that growth hormone contributed to the glycaemia-increasing effect of sprinting, at least in the hypoglycaemic group. This is because growth hormone increased above basal levels in response to sprinting and because this hormone has been reported to increase glucose Ra and acutely inhibit glucose Rd in non-diabetic individuals [37]. However, in opposition to this interpretation, glucose Rd has been shown not to fall in response to growth hormone in insulin-treated individuals with type 1 diabetes [38]. Moreover, it is unlikely that growth hormone contributes to the early post-sprint increase in both blood glucose levels and glucose Ra, since growth hormone levels are not significantly higher than baseline levels at that time. Finally, since insulin levels were maintained at basal and stable levels before and after the sprint, and plasma glucagon levels remained unchanged after exercise, it is unlikely that these hormones play a role in the post-sprint increase in endogenous glucose Ra, unless the portal levels of these hormones are affected by a 10 s sprint.

The sustained post-sprint increase in blood glucose levels in both experimental groups is likely to be due to the absence of a post-exercise increase in plasma insulin levels. In support of this interpretation, after a prolonged bout of intense exercise in individuals with type 1 diabetes, both plasma insulin and blood glucose remain at stable levels unless insulin is administered to decrease blood glucose levels [10]. By contrast, the post-exercise rise in blood glucose levels in non-diabetic individuals results in an increase in plasma insulin levels, which in turn causes a subsequent fall in blood glucose levels [8–10]. Since, in the current study, plasma insulin levels in both experimental groups remained at a low and stable level throughout recovery, this would explain the absence of a post-exercise fall in blood glucose level.

In summary, this study shows that hypoglycaemia in the morning does not reduce the capacity of an afternoon 10 s sprint to increase blood glucose level and has little or no effect on the glucoregulatory response to this type of exercise in young adults with type 1 diabetes. These findings thus suggest that antecedent hypoglycaemia does not affect the efficacy of a 10 s sprint at preventing acute post-exercise hypoglycaemia; an interpretation, however, which remains to be tested. To the best of our knowledge, this study provides the first description of a condition where hypoglycaemia has no or little effect on the glucoregulatory response to a subsequent bout of exercise. Finally, this study suggests that the glycaemia-increasing effect of sprinting is robust, thus providing further support for incorporating sprinting as a clinical tool for hypoglycaemia prevention in healthy young individuals with type 1 diabetes.

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Duality of interest The authors declare that there is no duality of interest associated with this manuscript.

Contribution statement RJD, EAD, TWJ and PAF contributed to the conception and design of the study and the interpretation of data. RJD, NP, AJR and EML contributed to the acquisition, analysis and interpretation of data. RJD drafted the article and all authors revised it critically for important intellectual content. All authors approved the final version of the manuscript. PAF is responsible for the integrity of this work.

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