The 10-s Maximal Sprint

A novel approach to counter an exercise-mediated fall in glycemia in individuals with type 1 diabetes

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OBJECTIVE — To investigate whether a short maximal sprint can provide another means to counter the rapid fall in glycemia associated with moderate-intensity exercise in individuals with type 1 diabetes and therefore decrease the risk of early postexercise hypoglycemia.

RESEARCH DESIGN AND METHODS — In the study, seven male subjects with type 1 diabetes injected their normal insulin dose and ate their usual breakfast. When their postprandial glycemia fell to ~11 mmol/l, they pedaled at 40% $\dot{V}O_{2peak}$ for 20 min on a cycle ergometer then immediately engaged in a maximal 10-s cycling sprint (sprint trial) or rested (control trial); the sprint and rest trials were administered in a counterbalanced order.

RESULTS — Moderate-intensity exercise resulted in a significant fall ($P < 0.05$) in glycemia in both trials (means ± SE: 3.6 ± 0.5 vs. 3.1 ± 0.5 mmol/l for sprint and control, respectively). The subsequent short cycling sprint opposed a further fall in glycemia for 120 min, whereas in the absence of a sprint, glycemia decreased further (3.6 ± 1.22 mmol/l; $P < 0.05$) after exercise. The stabilization of glycemia in the sprint trial was associated with elevated levels of catecholamines, growth hormone, and cortisol. In contrast, these hormones remained at stable or near-stable levels in the control trial. Changes in insulin and free fatty acid levels were similar in the sprint and control trials.

CONCLUSIONS — These results suggest that after moderate-intensity exercise, it is preferable for young individuals with insulin-treated, complication-free type 1 diabetes to engage in a 10-s maximal sprint to acutely oppose a further fall in glycemia than to only rest. The addition of the sprint after moderate-intensity exercise provides another means to reduce the risk of hypoglycemia in active individuals with type 1 diabetes.

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It is well established that exercise of moderate intensity increases the risk of hypoglycemia during and after exercise in type 1 diabetic individuals (1,2) due, in part, to a contraction-mediated activation of glucose utilization in skeletal muscle (3) and an increase in insulin sensitivity (4). In contrast, 10–15 min of high-intensity exercise (>80% of maximal rate of oxygen consumption [$\dot{V}O_{2peak}$]) causes an increase in postexercise blood glucose levels in insulin-treated individuals with type 1 diabetes, irrespective of their level of glycemic control (5–10). This hyperglycemic effect of prolonged, high-intensity exercise raises the intriguing possibility that this type of exercise might provide a means other than carbohydrate intake to counter a fall in postexercise glycemia in individuals with complication-free type 1 diabetes and thus acutely reduce their risk of hypoglycemia. However, 10–15 min of high-intensity exercise is unlikely to be well tolerated by most type 1 diabetic individuals due to the very intense nature of such exercise combined with its impractical duration.

A more practical way of using intense exercise as a means to prevent glycemia from falling might be to engage in a much shorter bout of exercise performed at maximal intensity. The main difficulty with this suggestion is that a maximal sprint effort that lasts >30 s is associated with unpleasant consequences such as nausea, vomiting, and dizziness (11,12). In contrast, a 10-s maximal sprint effort is well tolerated (13). This raises the question of whether such a short sprint could provide an alternative means other than carbohydrate intake to oppose a fall in glycemia. Although individuals at rest are unlikely to engage in a sprint to stabilize their glycemia, doing so might prove to be an effective and convenient way to counteract a rapid fall in blood glucose level in response to moderate-intensity exercise. Because this possibility has never been examined before, it was the primary goal of this study to determine whether a 10-s maximal sprint effort is preferable to only resting as a means to counter a further fall in glycemia during recovery from moderate-intensity exercise in individuals with type 1 diabetes.

RESEARCH DESIGN AND METHODS

— For this study, seven young men with type 1 diabetes were recruited (ages 21.0 ± 3.5 years, BMI 26.9 ± 4.0 kg/m^2, $\dot{V}O_{2peak}$ 44.5 ± 4.2 ml · kg^{-1} · min^{-1}, diabetes duration 9.1 ± 3.6 years, and HbA_{1c} 7.4 ± 0.8% [range 6.6–9.0%]). All participants were free from any diabetes complications, had undetectable levels of C-peptide, and were not taking any prescribed medication other than insulin. They had all been treated with a stable insulin regimen composed of a combination of slow- or intermediate-acting insulin (e.g., NPH insulin) and fast-acting insulin analogs for ≥3 months before the study. All participants were required to attend our laboratory on three occasions. During the first visit they were familiarized with the study protocol and gave their informed consent; also during this visit, we obtained their anthropometric measurements and determined their maximal rate of oxygen consumption, as previously described

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A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

(14). The next two visits were the sprint and rest (control) trials administered in a random counterbalanced order. The institutional ethics committee approved all the procedures described in this study.

The participants were not permitted to exercise for 24 h before the experimental trial, and testing was rescheduled if they had experienced a hypoglycemic episode over the previous 48 h. Participants were also instructed to maintain a similar diet and avoid alcohol for 24 h before each test session. On the morning of testing, the participants were required to monitor their blood glucose regularly. They arrived in the laboratory at ~8:00 A.M. and were instructed to self-administer their usual dose of morning insulin and eat their breakfast. Afterward a catheter was inserted in the antecubital fossa. Participants' insulin dose and breakfast content (1,833 ± 224 kJ total energy; 57 ± 3% carbohydrate, 17 ± 1% protein, and 26 ± 4% fat) were kept identical for both trials. Glycemia was then measured every 15 min, and once blood glucose levels fell after peaking postprandially, no mid-morning snack was allowed so as not to counter the fall in glycemia. If participants' blood glucose levels were not decreasing at the commencement of exercise, the test session was cancelled because the purpose of this study was to determine whether a short sprint could counter an exercise-mediated fall in glycemia.

When participants' blood glucose levels reached ~11 mmol/l (~121 ± 15 and ~109 ± 10 min after insulin injection in the sprint and control trials), they engaged in 20 min of moderate-intensity exercise (40% $\dot{V}O_{2peak}$) on an air-braked Repco front-access cycle ergometer (Repco, Sydney, Australia), with the resistance to cycling increasing with the cycling rate. An intensity of 40% $\dot{V}O_{2peak}$ was adopted because it more closely mimics the intensity of most activity patterns performed under "real-life" conditions for the general population. Also, because all participants were tested at a time close to peak plasma insulin levels and insulin-treated individuals with type 1 diabetes are discouraged from engaging in any intense exercise during that time (15), the information gathered from exercising our participants at higher intensity would have been of little practical relevance to most individuals with type 1 diabetes. The 20-min duration was adopted because preliminary work in our laboratory revealed that had the exercise duration been longer, a large proportion of our participants would have reached hypoglycemic levels because of the rapid fall in glycemia when exercising at 40% $\dot{V}O_{2peak}$, and testing would have had to end prematurely to avoid a hypoglycemia-mediated counterregulatory response. On completion of this moderate-intensity cycling, participants were instructed to rest or perform a 10-s cycling sprint, depending on their experimental trial. All participants were instructed to cycle as hard as possible and not pace themselves for the whole duration of the 10-s sprint. Venous blood from the arm and arterialized capillary blood from the earlobe were sampled before the moderate-intensity exercise, immediately before the cycling sprint (in the sprint trial), and then at 0, 5, 10, 15, 30, 45, 60, 75, 90, 105, and 120 min of recovery or until blood glucose levels declined to 3.5 mmol/l, in which case the trial was ended and the participant was immediately given carbohydrates to prevent hypoglycemia.

At each sampling point, 35 μl of arterialized capillary blood was taken from the earlobe and assayed immediately for glucose and lactate levels using an ABL 625 blood gas system (Radiometer, Copenhagen, Denmark). A 15-ml sample of venous blood was removed from the catheter for measuring hormones. Some of the blood was combined with sodium metabisulfite, polyethylene glycol, or aprotinin (Trasylol) for the assay of catecholamines, insulin, and glucagon, respectively. All samples were then centrifuged at 720*g* for 5 min, and the plasma was stored at −80°C for later analysis of catecholamine, free fatty acid, insulin, glucagon, cortisol, growth hormone, and C-peptide levels.

Hormones and metabolite assays

Glucose and lactate were analyzed using an ABL 625 blood gas system (Radiometer). Heparinized plasma treated with sodium metabisulfate was used to determine catecholamine levels by reverse-phase high-performance liquid chromatography using a Waters Novapak C18 reverse-phase column and a model 5200A Coulochem detector (ESA Biosciences, Chelmsford, MA). Free fatty acid levels were measured in EDTA-treated plasma using the Roche Half-Micro Test Free Fatty Acids Assay kit (Mannheim, Germany). Heparinized plasma treated with polyethylene glycol was assayed for free insulin using the Coat-a-Count Insulin Kit (Diagnostic Products, Los Angeles, CA). Glucagon levels in plasma collected with aprotinin (Trasylol) were measured from EDTA-treated plasma by radioimmunoassay using a Linco glucagon radioimmunoassay kit (St. Charles, MO). Cortisol levels were assayed from venous serum by competitive immunoassay on an Immulite 2000 Analyser using the Immulite Cortisol Assay kit (Diagnostic Products). Growth hormone levels were determined from serum by immunometric assay on an Immulite 2000 Analyser using the Immulite Growth Hormone Assay kit (Diagnostic Products). C-peptide levels were determined by solid-phase competitive chemiluminescent enzyme immunoassay on an Immulite 2000 Analyser using the Immulite C-Peptide Assay kit (Diagnostic Products).

Statistical analyses

The results were analyzed using a two-way (time × trial) repeated-measures ANOVA and Fisher's least significant differences test for a posteriori analysis using SPSS 11.0 software. Statistical significance was accepted at $P < 0.05$. Participants' characteristics are expressed as means ± SD, whereas all other results are expressed as means ± SE.

RESULTS

Blood metabolite response

Before the bout of moderate-intensity exercise, blood glucose levels in both experimental trials fell significantly ($P < 0.05$) (Fig. 1). When glycemia reached ~11 mmol/l (11.2 ± 0.4 vs. 11.9 ± 0.4 mmol/l in the sprint and control trial, respectively), 20 min of cycling at 40% $\dot{V}O_{2peak}$ was initiated, with the total workload being identical between treatments for each subject (total work of 1,176 ± 105 and 1,178 ± 104 kJ/kg for sprint and control trials, respectively). This resulted in a further rapid significant decrease in glycemia in both experimental trials (sprint: 3.6 ± 0.5 mmol/l, $P < 0.05$; control: 3.1 ± 0.5 mmol/l, $P < 0.05$) (Fig. 1). When a 10-s maximal sprint effort was performed immediately after the moderate-intensity exercise, the sprint opposed a further fall in blood glucose levels for the next 120 min. In contrast, blood glucose levels decreased further (3.6 ± 1.22 mmol/l; $P < 0.05$) in the control trial (Fig. 1).

The response of free fatty acid levels to the sprint and control trials was similar, with stable levels observed during moderate-intensity exercise then marginally increasing later in recovery (Fig. 2). In response to the moderate-intensity exercise, lactate levels increased moderately

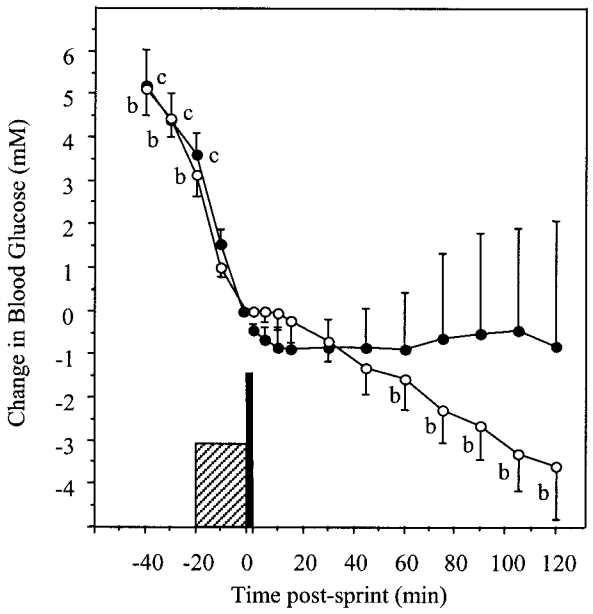

Figure 1—*Effect of a 10-s sprint on blood glucose after moderate-intensity exercise. The moderate-intensity exercise commenced at time point −20. Blood glucose levels are expressed relative to those immediately after the moderate-intensity exercise (time point = 0). All data are means ± SE. ▨, moderate-intensity exercise; vertical bar, sprint; ●, sprint trial; ○, control trial.* [b]*P < 0.05 vs. 0-min time point (after moderate-intensity exercise) in control trial;* [c]*P < 0.05 vs. 0-min time point (after moderate-intensity exercise) in sprint trial.*

and rose to a greater extent in response to the sprint, reaching maximal levels after 5 min ($P < 0.05$) before decreasing to basal levels within 45 min after the sprint. In contrast, in the absence of a short sprint, lactate levels remained at stable basal levels throughout the recovery period.

Hormonal response

In response to the 10-s maximal sprint effort initiated immediately after moderate-intensity exercise, epinephrine and norepinephrine reached maximal levels at the onset of recovery ($P < 0.05$ for each) and returned to pre-exercise levels within 5 min after the sprint (Fig. 2). In contrast, catecholamine levels remained stable in the control trial (Fig. 2). Likewise, the response of growth hormone differed between the sprint and control trials, with growth hormone levels increasing progressively after the sprint to reach maximal levels after 15 min of recovery (Fig. 2). The response of plasma cortisol levels in the sprint trial also differed from those during the control trial, with cortisol levels increasing significantly during recovery to reach maximal levels 30 min after the sprint ($P < 0.05$), but remained at stable levels in the control trial (Fig. 2). Glucagon increased early in the recovery period in the control trial ($P < 0.05$) but did not change significantly in the sprint trial. Finally, the pattern of insulin response to exercise was similar in the sprint and control trials, with insulin levels remaining relatively stable throughout exercise and recovery (Fig. 2). Both trials were performed at a time of the day when insulin levels were elevated (121 ± 15 and 109 ± 10 min after insulin injection in the sprint and control trials, respectively), and plasma insulin levels were similar between trials (Fig. 2).

CONCLUSIONS — Current guidelines for minimizing the risk of hypoglycemia associated with exercise in type 1 diabetes recommend a reduction in insulin dose or increased ingestion of carbohydrates before exercise based on an individual's previous glycemic responses to similar exercise (15). This study investigated the intriguing possibility of using a short bout of intense exercise as another means to counteract an exercise-mediated fall in glycemia. In particular, it examined whether a short 10-s cycling sprint could acutely oppose an exercise-mediated fall in glycemia in individuals with insulin-treated, complication-free type 1 diabetes. Our results suggested that to minimize the risk of a fall in glycemia after a bout of moderate-intensity exercise in young individuals with complication-free type 1 diabetes, it is preferable to engage in a 10-s maximal sprint effort before resting than to only rest during recovery. Such a sprint opposed a further fall in blood glucose levels for at least 120 min, whereas glycemia decreased significantly (by ~3.5 mmol/l; $P < 0.05$) when no sprint was performed (Fig. 1). Sprinting is likely to counter the exercise-mediated decrease in blood glucose levels through an increase in catecholamine, lactate, cortisol, and growth hormone levels. The ability of the sprint to oppose the fall in glycemia was more remarkable considering the sprint and control trials were performed when insulin levels were elevated, a time when exercise is not usually recommended (16).

It is likely that the marked rise in catecholamine levels after the sprint, at the onset of recovery, explain how such a sprint counters an exercise-mediated fall in glycemia, as the levels of the other counterregulatory hormones examined in this study did not change significantly during this time (Fig. 2). It is generally acknowledged that high catecholamine levels oppose an insulin-mediated fall in glycemia (10) via their activation of hepatic glucose production and inhibition of insulin-mediated glucose uptake in skeletal muscle (17). Likewise, elevated lactate levels (Fig. 2) may contribute to the stabilization of glycemia early in recovery by providing gluconeogenic precursors for hepatic glucose production (18).

In our study, catecholamines returned rapidly to basal levels after the sprint, thus raising the question of whether other hormones opposed the decrease in glycemia as recovery progressed in the sprint trial. Because insulin levels did not change significantly from pre-exercise levels over the 2 h of recovery (Fig. 2), they could not explain how sprinting opposed the exercise-mediated fall in glycemia. Likewise, the absence of an increase in plasma glucagon levels in the sprint trial makes it unlikely that glucagon was responsible for opposing the decrease in glycemia (Fig. 2). Although there is some evidence that the progressive rise in cortisol levels may play a role in stabilizing glycemia due to cortisol's potential acute inhibitory effect on glucose utilization in skeletal muscle (19), it is likely that this hormone played only a minor role because the effects of cortisol on hepatic glucose production and blood glucose levels have been shown by most

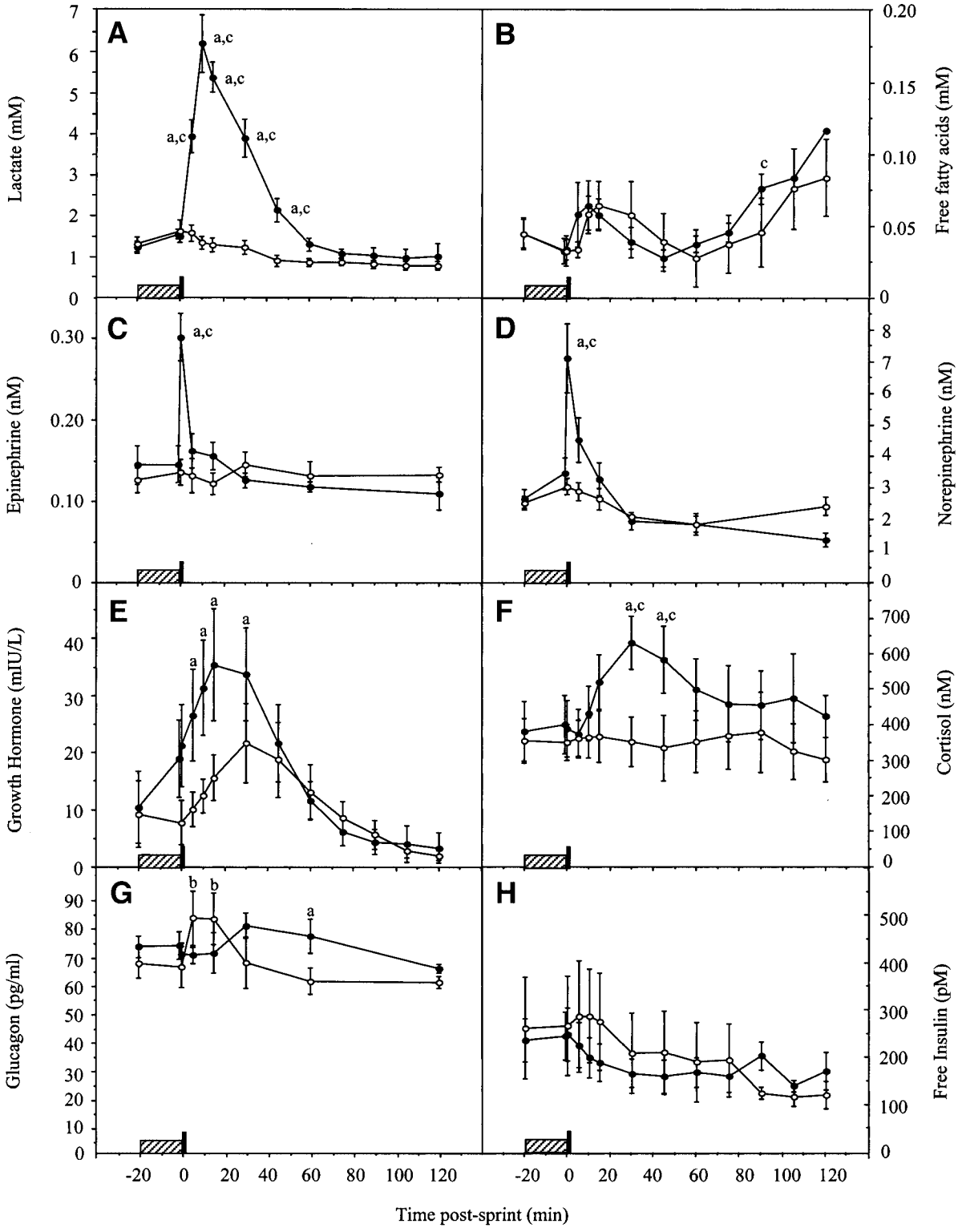

Figure 2—*Effect of a 10-s sprint on the levels of lactate (A), free fatty acid (B), epinephrine (C), norepinephrine (D), growth hormone (E), cortisol (F), glucagon (G), and free insulin (H) after moderate-intensity exercise. Data are means ± SE. ▨, moderate-intensity exercise; vertical bar, sprint; ●, sprint trial; ○, control trial.* $^{a}P < 0.05$ *for control vs. sprint trial;* $^{b}P < 0.05$ *vs. 0-min time point (after moderate-intensity exercise) in control trial;* $^{c}P < 0.05$ *vs. 0-min time point (after moderate-intensity exercise) in sprint trial.*

studies to require several hours to occur (20).

The elevated levels of growth hormone after exercise (Fig. 2) might play some role in opposing the decrease in glycemia later during recovery from sprinting. In support of this view, the administration of a physiological growth hormone pulse in nonexercised nondia-

betic individuals has been reported to result in a rapid fall in muscle glucose uptake (21–23) and a 1- to 2-h delayed increase in lipolysis, circulating free fatty acid levels, and fat oxidation rates (21–23), which could contribute further to lowering glucose utilization rates (22). However, the aforementioned fall in muscle glucose uptake in response to a growth hormone pulse does not occur in insulin-treated individuals with type 1 diabetes (24) and is not associated with a corresponding change in glycemia and glucose appearance (R_a) and disposal (R_d) rates (23), thereby making it unlikely that growth hormone had a role in opposing the postsprint fall in glycemia. More importantly, the administration of this hormone after a bout of moderate-intensity exercise in growth hormone–deficient individuals has no effect on glucose R_a and R_d (25), and the administration of octreotide (a somatostatin analog) in nondiabetic individuals has no acute effects on the magnitude of the hyperglycemic effect of high-intensity exercise (26). It is important to stress, however, that no study so far has evaluated whether glucose metabolism in response to a short sprint is affected by growth hormone levels. The identity of the counterregulatory hormone(s) responsible for opposing the fall in glycemia when a sprint is performed after a bout of moderate-intensity exercise remains to be established.

In conclusion, this study provided the first evidence that a short maximal sprint effort performed immediately after moderate-intensity exercise is preferable to only resting as a means to counter a further fall in glycemia after exercise, thus decreasing the risk of early postexercise hypoglycemia in individuals with type 1 diabetes. On this basis, one might tentatively recommend that after exercise of moderate intensity, young individuals with complication-free type 1 diabetes consider performing a short 10-s sprint to counter a further fall in their blood glucose levels rather than only resting, particularly if a source of dietary carbohydrate is not readily available. This recommendation does not extend to intermittent high-intensity exercise, as we have shown recently that blood glucose remains at stable levels for at least 1 h after this type of exercise (27,28). Although the long-term health benefits of regular exercise are generally recognized, to the best of our knowledge these findings provide the first example of a bout of exercise offering immediate short-term benefits (stabilization of glycemia). It is important to stress, however, that different results might have been obtained had sprinting been initiated after exercise of higher intensity or longer duration, in younger or older individuals with reduced sprinting capacity, or in individuals with impaired counterregulatory responses. For these reasons, more studies of the kind described here will be required to identify the subpopulation of type 1 diabetic individuals for whom a short maximal sprint effort can be recommended as a safe approach for the short-term stabilization of blood glucose levels.

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