

A 10-s Sprint Performed After Moderate-Intensity Exercise Neither Increases nor Decreases the Glucose Requirement to Prevent Late-Onset Hypoglycemia in Individuals With Type 1 Diabetes

RAYMOND J. DAVEY, PHD^{1,2}
 VANESSA A. BUSSAU, BSC²
 NIRUBASINI PARAMALINGAM, BSC^{1,3}
 LUIS D. FERREIRA, PHD^{1,2}

EE MUN LIM, PHD^{4,5}
 ELIZABETH A. DAVIS, MD^{1,3,6}
 TIMOTHY W. JONES, MD^{1,3,6}
 PAUL A. FOURNIER, PHD²

OBJECTIVE—To determine whether performing a 10-s sprint after moderate-intensity exercise increases the amount of carbohydrate required to maintain euglycemia and prevent late-onset postexercise hypoglycemia relative to moderate-intensity exercise alone.

RESEARCH DESIGN AND METHODS—Seven individuals with type 1 diabetes underwent a hyperinsulinemic-euglycemic clamp and performed 30 min of moderate-intensity exercise on two separate occasions followed by either a 10-s maximal sprint effort or no sprint. During the following 8 h, glucose infusion rate to maintain euglycemia and rates of glucose appearance and disappearance were measured continuously.

RESULTS—In response to exercise and throughout the 8-h recovery period, there were no differences in glucose infusion rate, blood glucose levels, plasma insulin concentrations, and rates of glucose appearance and disappearance between the two experimental conditions ($P > 0.05$).

CONCLUSIONS—A 10-s sprint performed after 30 min of moderate-intensity exercise does not affect the amount of carbohydrate required to maintain euglycemia postexercise in individuals with type 1 diabetes.

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Recently, we have shown that a 10-s sprint carried out before (1) or after (2) moderate-intensity exercise performed under hyperinsulinemic conditions opposes the fall in glycemia during early recovery, with no carbohydrate intake required to prevent blood glucose from falling postexercise. These findings suggest that sprinting may offer a

novel approach for reducing the risk of exercise-mediated hypoglycemia in type 1 diabetes (T1D), and this approach has therefore been included in some recent guidelines for hypoglycemia prevention (3,4).

One possible limitation of combining sprinting with moderate-intensity exercise to prevent postexercise hypoglycemia

is that the reduced risk of early post-exercise hypoglycemia may be offset by an increased risk of late-onset postexercise hypoglycemia (LOPEH) (5,6). In support of this view, a short sprint not only can deplete muscle glycogen stores to the same extent as prolonged exercise of low intensity (7,8) but also may enhance insulin sensitivity later during recovery, as suggested by the recent finding that insulin sensitivity is increased 24 h after a prolonged sprint (9). For these reasons, the objective of this study was to determine whether performing a 10-s sprint after moderate-intensity exercise increases the amount of carbohydrate required to maintain stable glycemia late during recovery, thus providing an indirect assessment of the risk of LOPEH. This is an important issue to address before advocating the use of sprinting in hypoglycemia prevention.

RESEARCH DESIGN AND METHODS

Four male and three female participants aged a mean \pm SD of 18.9 ± 4.6 years with a duration of T1D of 10.4 ± 4.2 years, glycated hemoglobin of $7.9 \pm 0.7\%$ (63 mmol/mol), BMI of 26.3 ± 3.6 kg/m², and $\dot{V}O_{2peak}$ of 34.2 ± 8.4 mL · kg⁻¹ · min⁻¹ and who were hypoglycemia aware and free of complications gave informed consent to participate in this study in accordance with the local ethics committee.

After a familiarization session in which $\dot{V}O_{2peak}$ was determined, the participants attended the laboratory on two occasions during which they underwent two different experimental conditions administered according to a randomized, counterbalanced study design. On the morning of testing, the participants arrived at 7:00 A.M. and were given a standardized breakfast. After this meal, they were not allowed to eat for the remainder of the study. Thereafter, two cannulas

From the ¹Telethon Institute for Child Health Research, Centre for Child Health Research, The University of Western Australia, Perth, Western Australia, Australia; the ²School of Sport Science, Exercise and Health, The University of Western Australia, Perth, Western Australia, Australia; the ³Department of Endocrinology and Diabetes, Princess Margaret Hospital, Perth, Western Australia, Australia; the ⁴PathWest Laboratory Medicine, Queen Elizabeth II, Perth, Western Australia, Australia; the ⁵Department of Endocrinology and Diabetes, Sir Charles Gairdner Hospital, Perth, Western Australia, Australia; and the ⁶School of Paediatrics and Child Health, The University of Western Australia, Perth, Western Australia, Australia.

Corresponding author: Raymond J. Davey, raymond.davey@health.wa.gov.au.

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were inserted for blood sampling and the infusion of glucose, insulin and [6,6-²H] glucose (10). Insulin was infused at a constant rate of 20 mU · m⁻² · min⁻¹, and blood glucose was measured regularly with the glucose oxidase method (YSI Inc., Yellow Springs, OH) and maintained at 5–6 mmol/L for the duration of the studies by varying the infusion rate of a 20% (w/v) dextrose solution. This insulin infusion rate was chosen to approach the hyperinsulinemic conditions under which sprinting has been shown to prevent glycemia from falling early during recovery from moderate-intensity exercise (1,2).

At 12:30 P.M., the participants exercised for 30 min on a cycle ergometer (RepcO, Melbourne, Australia) at 40% of their previously measured $\dot{V}O_{2peak}$ before performing a 10-s maximal sprint effort or no sprint, as described previously (1,2). For the remainder of the 8-h recovery period, samples for the assessment of blood glucose level, plasma insulin concentration, and the enrichment of [6,6-²H]glucose were obtained from arterialized venous blood (10). Heparinized plasma was assayed for free insulin by means of a noncompetitive chemiluminescent immunoassay (Abbott Architect i2000; Abbott Laboratories, Abbott Park, IL), and the rates of glucose appearance (Ra) and disappearance (Rd) were calculated from [6,6-²H]glucose enrichment by applying the single-pool nonsteady-state equations of Steele (11).

Data were analyzed with a two-way repeated-measures ANOVA and Fisher least significant difference a posteriori test by means of SPSS 17.0 software. Statistical significance was accepted at $P < 0.05$.

RESULTS—Before exercise, there were no significant differences in glucose infusion rate (GIR), blood glucose levels, plasma insulin levels, glucose Ra (undetectable for both conditions), and glucose Rd between the no sprint and sprint conditions (Fig. 1A–E). During moderate-intensity exercise, the GIR necessary to maintain euglycemia increased significantly but did not significantly differ between conditions (Fig. 1A). Moreover, there were no significant differences in blood glucose levels (Fig. 1B), plasma insulin levels (Fig. 1C), or relative oxygen consumption ($42.5 \pm 2.9\% \dot{V}O_{2peak}$ and $41.8 \pm 2.9\% \dot{V}O_{2peak}$) during exercise between conditions. The increase in GIR during moderate-intensity exercise was associated with no changes in glucose

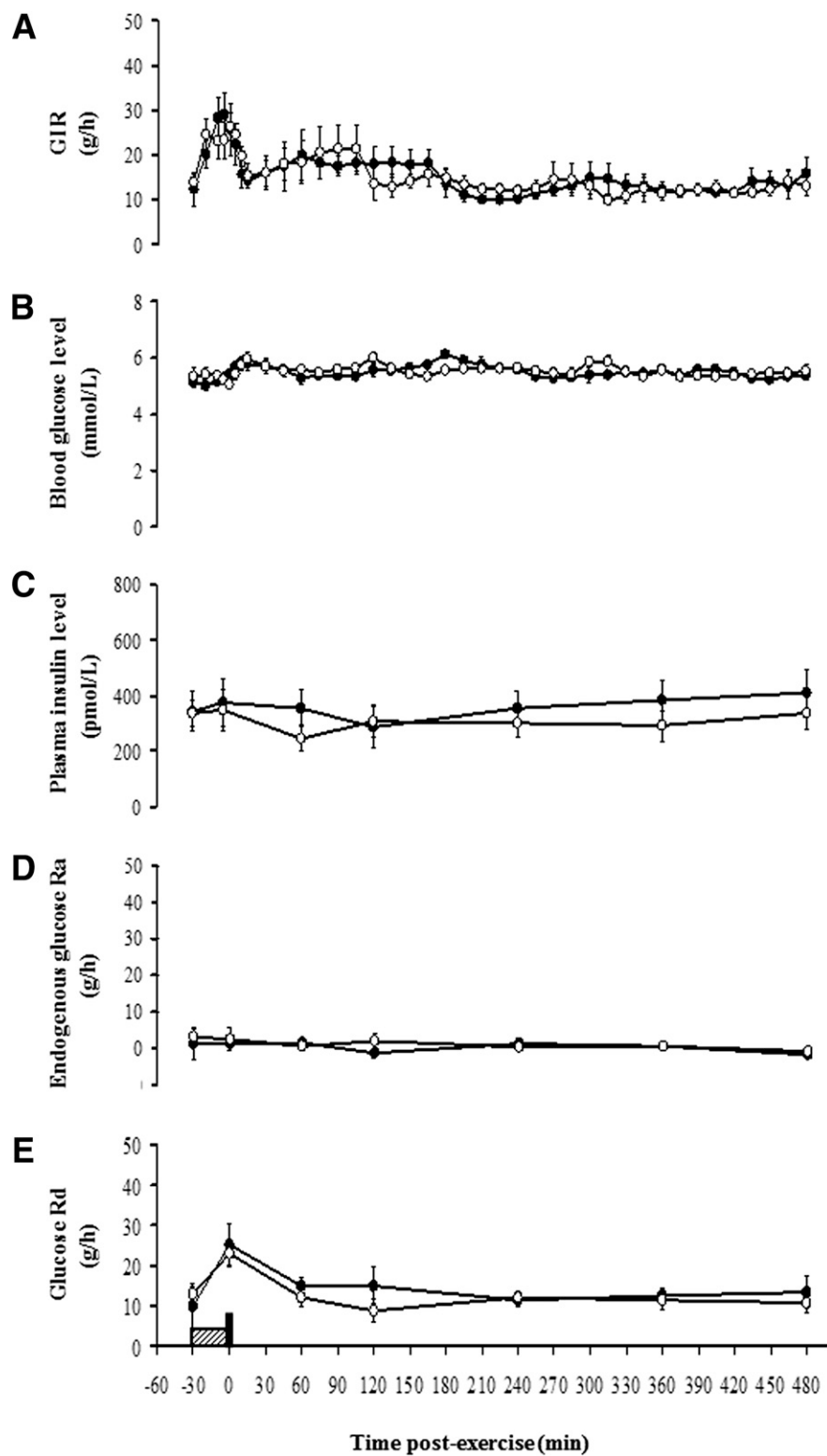


Figure 1—GIR to maintain euglycemia (A), blood glucose level (B), plasma insulin level (C), and glucose Ra (D) and Rd (E) in response to moderate-intensity exercise (●) and moderate-intensity exercise followed by a 10-s sprint (○). Results are expressed as mean ± SEM (n = 7). Black lines represent 10-s sprint periods; hatched boxes represent periods of moderate-intensity exercise.

Ra and significant increases in glucose Rd, with no differences between conditions (Fig. 1D and 1E). During the ensuing 8 h of recovery, GIR (Fig. 1A), blood

glucose levels (Fig. 1B), plasma insulin levels (Fig. 1C), glucose Ra (Fig. 1D), and glucose Rd (Fig. 1E) were similar for both conditions and did not change

for either condition for the duration of the recovery period.

CONCLUSIONS—This study shows that performing a 10-s sprint immediately after moderate-intensity exercise in hyperinsulinemic T1D individuals neither increases nor decreases the amount of glucose required to maintain euglycemia through 8 h of recovery and has no effect on glucose Ra and Rd. This suggests that sprinting, as performed under our experimental conditions, neither increases the risk of LOPEH nor reduces the risk of exercise-mediated hypoglycemia early and late during recovery, thus providing the first example of experimental conditions under which sprinting may not provide any glucoregulatory benefits against hypoglycemia. This is an important finding, because it is important to identify the conditions, such as those revealed here, under which the glucoregulatory benefits of sprinting are compromised before recommending the general adoption of sprinting as a means to reduce the risk of exercise-mediated hypoglycemia in T1D.

The absence of transient falls in both GIR and glucose Rd during early recovery from sprinting was unexpected in light of our previous findings that a sprint performed after moderate-intensity exercise prevents blood glucose from falling early postexercise (2) and that sprinting under basal insulinemic conditions both inhibits glucose Rd and results in a transient rise in blood glucose levels (10). It is unclear whether the absence of a fall in glucose Rd reported here, but not seen when sprinting is performed under basal insulin levels (10), is due to plasma insulin's maintenance at levels high enough to counter any inhibitory effect of sprinting on glucose Rd (12). In addition, our finding that glucose Ra was suppressed irrespective of whether sprinting was performed after moderate-intensity exercise is in agreement with the findings of others, who have shown that plasma insulin levels comparable to those maintained in this study are high enough to suppress hepatic glucose production (12). Taken with the elevated glucose Rd throughout recovery, this finding explains the high GIR required to maintain euglycemia before, during, and after exercise.

Importantly, the absence of a difference in GIR between conditions does not exclude the possibility that sprinting after moderate-intensity exercise may increase the risk of LOPEH if the counterregulatory response to subsequent hypoglycemia is differentially affected by our interventions. Indeed, because moderate-intensity exercise reduces the counterregulatory responses to subsequent hypoglycemia (13), it is possible that sprinting performed after moderate exercise could further diminish these responses. Assessing the risk of hypoglycemia on the basis of the GIR response to exercise alone thus provides only a partial evaluation of this risk.

In summary, these findings indicate that a short sprint performed after moderate-intensity exercise neither increases nor decreases the glucose requirements to maintain euglycemia during 8 h of recovery from moderate-intensity exercise in T1D individuals. This study also uncovers for the first time experimental conditions under which the glucoregulatory benefits of sprinting in hypoglycemia prevention appear to be compromised.

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R.J.D. researched data and wrote the manuscript. V.A.B. and L.D.F. researched data. N.P. researched data and reviewed and edited the manuscript. E.M.L. contributed to data analysis. E.A.D., T.W.J., and P.A.F. supervised the study and reviewed and edited the manuscript. R.J.D. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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References

- Bussau VA, Ferreira LD, Jones TW, Fournier PA. A 10-s sprint performed prior to moderate-intensity exercise prevents early post-exercise fall in glycaemia in individuals with type 1 diabetes. *Diabetologia* 2007;50:1815–1818
- Bussau VA, Ferreira LD, Jones TW, Fournier PA. The 10-s maximal sprint: a novel approach to counter an exercise-mediated fall in glycemia in individuals with type 1 diabetes. *Diabetes Care* 2006;29:601–606
- Guelfi KJ, Jones TW, Fournier PA. New insights into managing the risk of hypoglycaemia associated with intermittent high-intensity exercise in individuals with type 1 diabetes mellitus: implications for existing guidelines. *Sports Med* 2007;37:937–946
- Robertson K, Adolfsson P, Riddell MC, Scheiner G, Hanas R. Exercise in children and adolescents with diabetes. *Pediatr Diabetes* 2008;9:65–77
- Tsalikian E, Mauras N, Beck RW, et al.; Diabetes Research In Children Network Dircenet Study Group. Impact of exercise on overnight glycemic control in children with type 1 diabetes mellitus. *J Pediatr* 2005;147:528–534
- Admon G, Weinstein Y, Falk B, et al. Exercise with and without an insulin pump among children and adolescents with type 1 diabetes mellitus. *Pediatrics* 2005;116:e348–e355
- Parolin ML, Chesley A, Matsos MP, Spriet LL, Jones NL, Heigenhauser GJ. Regulation of skeletal muscle glycogen phosphorylase and PDH during maximal intermittent exercise. *Am J Physiol* 1999;277:E890–E900
- Gollnick PD, Armstrong RB, Saubert CW 4th, Sembrowich WL, Shepherd RE, Saltin B. Glycogen depletion patterns in human skeletal muscle fibers during prolonged work. *Pflugers Arch* 1973;344:1–12
- Whyte LJ, Ferguson C, Wilson J, Scott RA, Gill JM. Effects of single bout of very high-intensity exercise on metabolic health biomarkers in overweight/obese sedentary men. *Metabolism* 2013;62:212–219
- Fahey AJ, Paramalingam N, Davey RJ, Davis EA, Jones TW, Fournier PA. The effect of a short sprint on postexercise whole-body glucose production and utilization rates in individuals with type 1 diabetes mellitus. *J Clin Endocrinol Metab* 2012;97:4193–4200
- Wolfe RR, Chinkes DL. *Isotope Tracers in Metabolic Research: Principles and Practice of Kinetic Analysis*. 2nd ed. New York, John Wiley & Sons, 2005
- Rizza RA, Mandarino LJ, Gerich JE. Dose-response characteristics for effects of insulin on production and utilization of glucose in man. *Am J Physiol* 1981;240:E630–E639
- Galassetti P, Mann S, Tate D, et al. Effects of antecedent prolonged exercise on subsequent counterregulatory responses to hypoglycemia. *Am J Physiol Endocrinol Metab* 2001;280:E908–E917