Resistance Versus Aerobic Exercise

Acute effects on glycemia in type 1 diabetes

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OBJECTIVE—In type 1 diabetes, small studies have found that resistance exercise (weight lifting) reduces HbA_{1c}. In the current study, we examined the acute impacts of resistance exercise on glycemia during exercise and in the subsequent 24 h compared with aerobic exercise and no exercise.

RESEARCH DESIGN AND METHODS—Twelve physically active individuals with type 1 diabetes (HbA_{1c} 7.1 ± 1.0%) performed 45 min of resistance exercise (three sets of seven exercises at eight repetitions maximum), 45 min of aerobic exercise (running at 60% of $V_{O_{2max}}$), or no exercise on separate days. Plasma glucose was measured during and for 60 min after exercise. Interstitial glucose was measured by continuous glucose monitoring 24 h before, during, and 24 h after exercise.

RESULTS—Treatment-by-time interactions ($P < 0.001$) were found for changes in plasma glucose during and after exercise. Plasma glucose decreased from 8.4 ± 2.7 to 6.8 ± 2.3 mmol/L ($P = 0.008$) during resistance exercise and from 9.2 ± 3.4 to 5.8 ± 2.0 mmol/L ($P = 0.001$) during aerobic exercise. No significant changes were seen during the no-exercise control session. During recovery, glucose levels did not change significantly after resistance exercise but increased by 2.2 ± 0.6 mmol/L ($P = 0.023$) after aerobic exercise. Mean interstitial glucose from 4.5 to 6.0 h postexercise was significantly lower after resistance exercise versus aerobic exercise.

CONCLUSIONS—Resistance exercise causes less initial decline in blood glucose during the activity but is associated with more prolonged reductions in postexercise glycemia than aerobic exercise. This might account for HbA_{1c} reductions found in studies of resistance exercise but not aerobic exercise in type 1 diabetes.

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The frequency and severity of complications in individuals with type 1 diabetes are greater among those reporting little leisure-time physical activity versus those with higher activity levels (1). However, it remains unclear whether exercise is beneficial for glycemic control in type 1 diabetes (2). Aerobic exercise interventions have generally shown little effect on blood glucose control as determined by HbA_{1c} (3). In contrast, several studies evaluating resistance exercise (weight lifting) alone (4), in comparison with aerobic exercise (5), as part of a circuit-training program (6) or in combined resistance and aerobic exercise sessions (7,8) showed HbA_{1c} reductions.

During prolonged mild- to moderate-intensity aerobic activities, blood glucose levels decrease rapidly in individuals with type 1 diabetes, increasing the risk of hypoglycemia (9,10). Conversely, short bursts of higher-intensity activities (short sprints and high-intensity intermittent exercise), alone or combined with moderate intensity aerobic exercise, produce smaller declines in blood glucose during activity and up to 2 h postexercise than moderate-intensity aerobic activity alone (11–14). Moderate aerobic exercise is also associated with an increased risk of nocturnal hypoglycemia (15,16), but small studies using continuous glucose monitoring (CGM) have yielded mixed results regarding the effects of high-intensity activity on the risk of late postexercise hypoglycemia (17–19).

Resistance exercise is a moderate- to high-intensity activity performed in relatively short-duration intervals that carries many potential benefits for individuals with type 1 diabetes including increases in muscular strength (4), improved lipid profile (4), decreased insulin dosage (4,5), and lower self-monitored blood glucose levels (4,5). The acute effects of resistance exercise in individuals with type 1 diabetes have not been examined; therefore, it is unknown whether the risk of exercise-induced hypoglycemia is comparable with that of aerobic exercise. The risk of nocturnal hypoglycemia associated with restoration of muscle glycogen stores after resistance exercise is equally unknown. The aim of this study was to evaluate the effects of resistance exercise on blood glucose levels during, immediately after, and for 24 h postexercise compared with aerobic exercise or no exercise in individuals with type 1 diabetes. We hypothesized that, compared with aerobic exercise, resistance exercise would be associated with less of a decline in blood glucose levels during the activity but more of a sustained reduction in glycemia after the exercise, thereby potentially improving overall glucose stability.

RESEARCH DESIGN AND METHODS

METHODS—The study was approved by the research ethics boards of the University of Ottawa and Ottawa Hospital.

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Nonobese, nonsmoking adults with complication-free type 1 diabetes were recruited. Two of the participants were competitive athletes training 6 days per week, while those remaining were recreationally active. All participants had been regularly performing both aerobic and resistance exercise at least three times weekly for a minimum of 6 months. Participants were using either multiple daily injections (MDIs) of insulin or continuous subcutaneous insulin infusion with an insulin pump. The same cohort of participants also took part in a previously published study from the same research group (20).

Experimental design

Testing took place in the Human and Environmental Physiology Research Unit at the University of Ottawa. Participants attended one preliminary visit and three experimental trials. During the preliminary visit, participants provided written informed consent prior to being tested for $V_{O_{2max}}$, muscular strength (eight repetition maximum), and HbA_{1c} as previously described (20).

CGM

The CGMS System Gold (Medtronic, Northridge, CA) was used in this study so that participants would be blinded to their glucose values and would not change their behavior based on real-time glucose monitoring. CGMS sensors were inserted subcutaneously at 8:30 A.M. the day before the testing session. OneTouch UltraSmart handheld glucose meters (LifeScan; Johnson & Johnson, Milpitas, CA) and coded strips (same code throughout the study) were provided for capillary glucose tests. Participants tested capillary glucose for CGM calibration purposes four times daily. Twenty-four hours after the end of the exercise/no-exercise control session, CGM units were retrieved and data were downloaded (Minimed Solutions v.3.0c; Medtronic, Northridge, CA).

Over each monitoring period, participants consumed the same self-selected breakfast, lunch, and dinner daily at the same times of day and recorded food and insulin intake on study log sheets. Participants refrained from exercise for 24 h before insertion of the sensor (48 h before the experimental session) and avoided caffeine and alcohol during the monitoring period.

Experimental sessions

Participants arrived at the laboratory at 4:00 P.M. on the day after the sensor insertion. The following sessions were performed, separated by at least 5 days: *1*) resistance exercise, three sets of eight repetitions maximum of seven different exercises with 90-s rest between sets (duration ~45 min); 2) aerobic exercise, 45 min of treadmill exercise (60% of $V_{O_{2max}}$); and 3) no-exercise control, 45 min of seated rest.

Sessions were followed by 60 min of monitored resting recovery. Testing sessions for the female participants, who were using monophasic oral contraceptives, took place during the active pill-consumption phase. No-exercise control sessions were performed first. The remaining sessions were randomly assigned.

Insulin adjustments and glucose supplementation

Participants reduced their insulin doses on exercise days by making either a 10% decrease in intermediate or long-acting insulin (MDI) or a 50% decrease in basal rate starting 1 h before exercise and maintained until the end of exercise for pump users. If blood glucose was <5 mmol/L upon arrival, those using insulin pumps decreased their basal rate a further 25%. Participants consumed a standard snack (Glucerna Chocolate Graham Snack Bars, 150 calories, 25 g carbohydrate; Abbott Laboratories, Abbott Park, IL) at 4:00 P.M. every day, including the exercise day, with the bar consumed upon arrival at the laboratory.

Capillary glucose was checked 60 and 30 min before exercise and immediately prior to exercise to ensure glucose levels ≥5.5 and ≤13.9 mmol/L. Glucose tablets were provided when necessary and as previously described (20).

Blood sampling and analyses

Venous blood samples were collected through an intravenous catheter at baseline and 5, 10, 15, 30, and 45 min during all three testing sessions (resistance exercise, aerobic exercise, and no-exercise control) and at the 50-, 55-, 60-, 65-, 75-, 85-, 95-, and 105-min time points during recovery. Blood was immediately mixed by inversion, centrifuged (4,000 revolutions/min for 4 min), and stored at −80°C. The hexokinase timed end point method was used to determine plasma glucose levels using the Beckman Coulter Unicel DxC600 Synchron Clinical Analyzer (Beckman Coulter, Fullerton, CA) and SYNCHRON CX Systems GLUCOSE reagent (cat. no. 442640).

Statistical analyses

Glucose levels were compared among sessions using two-way repeated-measures (time and condition) ANOVA. Exercise and recovery periods were examined separately among the three sessions (aerobic, resistance, and no-exercise control). The exercise period consisted of the 5-, 10-, 15-, 30-, and 45-min time points, while the recovery period consisted of the remaining time points. Paired sample *t* tests were used to perform pairwise post hoc comparisons for each time point between conditions (aerobic, resistance, or no-exercise control) within exercise and recovery separately and to examine changes from baseline and changes from the end of exercise within each exercise condition. Significance was set at 0.05.

CGM data were examined as 15-min averages in the following windows: 24-h pre-exercise, overnight (12:00 A.M. to 6:00 A.M.) pre-exercise, 1–6 h postexercise, overnight postexercise, and 24 h postexercise. A two-way (time and condition) repeated-measures ANOVA was used to compare among conditions in the 1–6-h postexercise period. Paired sample *t* tests were then used to perform pairwise post hoc comparisons for each 15-min segment. Thresholds for hypo- and hyperglycemia were set at 3.5 and 10.9 mmol/L, respectively. The minimum, maximum, and mean blood glucose; amount of time spent in hypoglycemic and hyperglycemic states; and areas under the curve (AUCs) for time spent in hypo- and hyperglycemic states were determined for each window. Pre-exercise values were compared with postexercise values within exercise conditions using related-samples Wilcoxon signed rank tests. Differences among conditions were examined using related-samples Friedman two-way ANOVA by ranks. Agreement between CGM data and capillary glucose over the 3 days was determined by performing Pearson correlations between sensor glucose and self-recorded capillary glucose values.

Daily total insulin and carbohydrate intake was calculated based on the information provided in participant logs. Comparisons among conditions for each day were made using related-samples Friedman two-way ANOVA by ranks. Where significant results were found, related-samples Wilcoxon signed rank tests ensued for determination of where the differences lie. Analyses were performed using SPSS 18.0 for Windows (SPSS, Chicago, IL).

RESULTS—Twelve (10 male and 2 female) nonobese (BMI 25.3 ± 3.0 kg/m^2), physically active ($V_{O_{2max}}$ 51.2 ± 10.8 ml · kg^{-1} · min^{-1}) individuals aged 17–62 years (mean age 31.8 ± 15.3 years) took part in the study. Mean diabetes duration was 12.5 ± 10.0 years, and participants were in moderate to good control of their blood glucose levels (HbA_{1c} 7.1 ± 1.1%). Five participants were receiving insulin by MDI, while seven were using continuous subcutaneous insulin infusion.

Plasma glucose

Exercise. Plasma glucose levels are plotted in Fig. 1. Information regarding treadmill speeds/inclines as well as the workloads for the resistance exercise sessions is provided in Supplementary Table 1. A significant interaction between time and exercise modality was observed ($P < 0.001$) for mean exercise glucose levels indicating that the total declines and the rates of decline in plasma glucose levels differed among sessions (Fig. 1). There were no significant differences among sessions in pre-exercise baseline plasma glucose concentration. A gradual decline in plasma glucose concentration occurred with resistance exercise (from 8.4 ± 2.7 to 6.8 ± 2.3 mmol/L over the 45-min session), resulting in levels that were significantly lower than baseline by the end of exercise ($P = 0.008$). No changes from baseline were detected throughout the first 45 min of the no-exercise session (from 8.4 ± 3.5 to 8.6 ± 3.8 mmol/L [$P = 0.585$]). In contrast, during the aerobic exercise, plasma glucose levels declined rapidly and more dramatically (from 9.2 ± 3.4 to 5.8 ± 2.0 mmol/L over 45 min [$P = 0.001$]), resulting in significant changes from baseline within 10 min. Glucose levels in the aerobic session were lower than the no-exercise session after 30 min of the activity.

Recovery. A significant interaction of time and exercise modality was also observed in mean plasma glucose levels during recovery ($P < 0.001$). Plasma glucose levels were stable after the resistance exercise and no-exercise sessions but increased by 2.2 ± 0.6 mmol/L during the recovery after aerobic activity ($P = 0.002$). Plasma glucose levels were not different from either no-exercise or resistance exercise at 60 min postexercise.

Carbohydrate intake and insulin dosage

The number of participants requiring glucose tablets during the testing session were two, nine, and three for the no-exercise control, aerobic, and resistance exercise sessions, respectively (Supplementary Table 2). Differences were significant between no-exercise control and aerobic exercise ($P = 0.007$). The P value for the comparison between resistance and aerobic exercise was 0.05. There were no significant differences in carbohydrate intake among conditions on the day before or the day after the laboratory session or in the 6 h after exercise (Table 1); however, carbohydrate intake was higher on the exercise testing day in the aerobic exercise session compared with the resistance exercise session ($P = 0.013$), mostly because of differences in supplementation during exercise. Two participants using insulin pumps chose to omit their usual insulin bolus with the Glucerna bar before exercise, and one insisted on suspending basal insulin (instead of a 50% reduction) when learning upon arrival at the laboratory that it was the day for aerobic activity. Daily insulin intake did not differ significantly among conditions on any day of sensor wear.

CGM data

Pearson correlations between capillary glucose levels measured on handheld meters and interstitial glucose levels measured by CGM were 0.95, 0.90, and 0.94 during nonlaboratory periods in the resistance exercise, aerobic, and no-exercise control sessions, respectively. During the 24 h before either exercise trial or no-exercise control, there were no significant differences among sessions in the total time spent in hypoglycemia, AUC for hypoglycemia, number of hyperglycemic events, time spent in a hyperglycemic state, AUC for hyperglycemia, or mean blood glucose.

Postexercise CGM data were only available for 11 and 10 of 12 participants in the no-exercise and aerobic exercise sessions, respectively, because of equipment malfunction in the remaining three sessions. Data were available for all 12 participants in the resistance exercise session. In total, there were 124 paired handheld meter and CGM values for the no-exercise control condition, 113 for the aerobic condition, and 115 for the resistance exercise condition. A marginal effect of time ($P = 0.073$) was found in the analysis of the CGM data from 1 to 6 h postexercise. Higher mean interstitial glucose concentrations were found in the fourth and fifth hours after the aerobic exercise session compared with the resistance exercise session ($P = 0.018$ at 5 h postexercise) (Fig. 2).

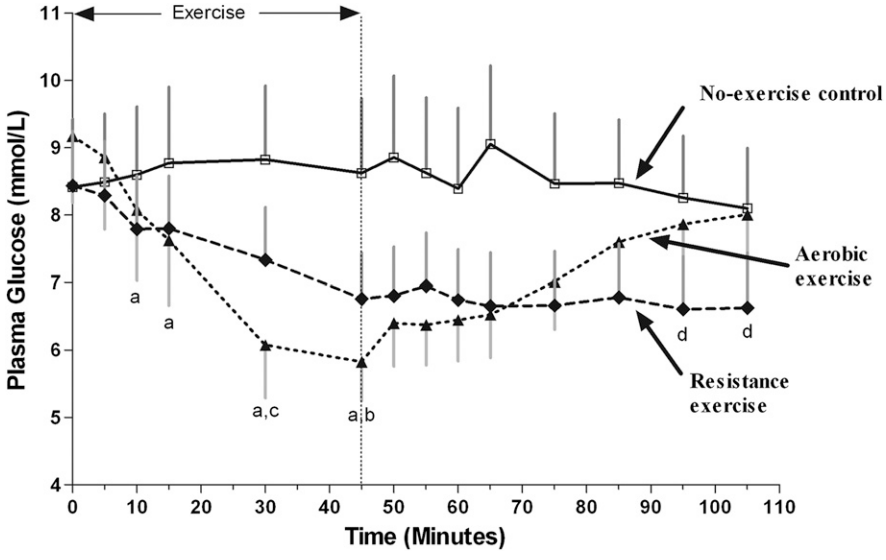

Figure 1—*Mean ± SE plasma glucose during the experimental sessions (represented by box) and 60 min of recovery* (n = *12 for aerobic exercise and no-exercise control;* n = *11 for resistance exercise).* □, *no-exercise control;* ◆, *resistance exercise,* ▲, *aerobic exercise.* [a]*Statistically significant change from baseline in aerobic exercise.* [b]*Statistically significant change from baseline in resistance exercise.* [c]*Statistically significant difference between no-exercise control session and aerobic session.* [d]*Statistically significant change throughout recovery after aerobic exercise. Differences were only considered statistically significant if still significant after Bonferroni corrections for multiple comparisons. During exercise, participants were provided with glucose tablets if blood glucose fell to <4.5 mmol/L.*

Table 1—*Insulin and carbohydrate intake during the 6 h after exercise

	Carbohydrate (g)*			Insulin (units)		
Participant	RES	AER	No-Ex	RES	AER	No-Ex
1	80	87	80	9.4	6.6	10.6
2	105	106	90	8	8	10
3	104	104	167	7.8	7.8	7.3
4	89	92	65	8	12	6
5	97	94	132	40	4	39
6	74	88	84	17	13	24
7	56	40	90	7	8.2	7
8	127	177	79	15.5	19.4	11.7
9	135	135	135	4.5	4.5	4.5
10	65	60	65	9.7	9.7	10.8
11	12	12	12	3.9	3.9	4.8
12	187	215	196	27	24.4	23.7
Mean ± SD	94 ± 44	101 ± 55	99 ± 50	13.2 ± 10.6	10.1 ± 6.3	13.3 ± 10.4

AER, aerobic exercise; No-Ex, no-exercise control; RES, resistance exercise. *Differences among conditions were not statistically significant.

Although there were twice as many nocturnal hypoglycemic excursions (Table 2) detected by CGM devices after resistance exercise (nine in total) versus aerobic exercise and no exercise (four for each), differences among conditions were not statistically significant. There was, however, a trend of more episodes of nocturnal hyperglycemia after resistance exercise ($P = 0.059$) compared with the pre-exercise night, but differences in mean glucose levels were not significant.

CONCLUSIONS—Resistance exercise resulted in much smaller declines in blood glucose during exercise than aerobic exercise or no exercise in individuals with type 1 diabetes. Resistance exercise was also associated with relatively stable early postexercise glucose concentration. Less carbohydrate supplementation was required during resistance exercise versus aerobic exercise, which would have attenuated some of the hypoglycemic effects of the aerobic activity. In contrast to resistance exercise and no exercise, aerobic exercise was associated with greater increases in glucose levels during early recovery, which resulted in a trend toward higher glucose concentrations in late recovery (as measured by CGM 3–6 h postexercise). These trends were observed in the absence of any significant differences in insulin dosage or carbohydrate intake during this time. Mean blood glucose levels after resistance exercise were similar to those when no exercise was performed: more stable during early recovery and within a healthier range (5–6 mmol/L) during late recovery. As such, performance of resistance exercise may represent an alternative strategy to prevent the acute decline in blood glucose levels observed with aerobic exercise while maintaining more favorable postexercise glucose levels. There was, however, a tendency toward more frequent, albeit mild, nocturnal hypoglycemia after resistance exercise sessions, which deserves further scrutiny.

The mechanisms for the more dramatic reduction in blood glucose levels during aerobic versus resistance exercise are unclear, but the reliance on anaerobic sources of fuel production during resistance exercise rather than aerobic sources (i.e., less reliance on blood glucose) (21,22) may have played a role. Previous studies involving anaerobic activity in individuals with type 1 diabetes (intermittent 4-s sprints [13,14] or a 10-s sprint pre- or postexercise [11,12]) found slower declines in blood glucose concentration during exercise and smaller decreases in postexercise glucose concentrations in comparison with low-intensity aerobic exercise alone. Insulin and cortisol levels were comparable across conditions in these studies and were therefore unlikely to be responsible for the differential patterns of blood glucose response (11–14). Growth hormone and catecholamines, meanwhile, were elevated after sprinting, potentially enhancing lipolysis and glycogenolysis, respectively, thereby potentially stabilizing blood glucose levels (11–14). It is undetermined whether these hormones are responsible for stabilizing blood glucose levels after resistance exercise in individuals with type 1 diabetes; however, both growth hormone and catecholamines are known to increase significantly in individuals without diabetes during resistance exercise protocols similar to the one used in the current study (23,24).

Attenuated declines in blood glucose concentration may also be related to increased lactate production during resistance exercise. In comparing the hormonal responses to various resistance exercise protocols, Smilios et al. (23) found that two sets of 10 repetitions of chest press, lateral pull down, and squat (a stimulus of smaller

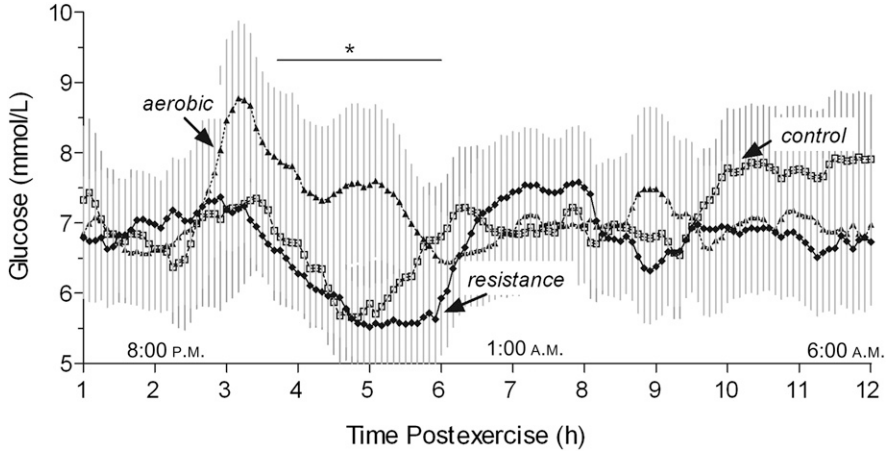

Figure 2—*Mean ± SE glucose as measured by CGM from 1 to 12 h postexercise. □, no-exercise control session; ▲, aerobic exercise session; ◆, resistance exercise session. The box represents the period of time where glucose was significantly higher after aerobic exercise compared with resistance exercise* ($P < 0.05$). n = *11 (no-exercise control),* n = *10 (aerobic), and* n = *12 (resistance).*

Table 2—*Summary of overnight CGM data for the night after resistance exercise, aerobic exercise, and no-exercise control*

	RES	AER	No-Ex	*P*
Participants experiencing nocturnal hypoglycemia (<3.5 mmol/L)	6/12 (50)	2/10 (20)	4/11 (36)	N/A
Total number of hypoglycemia episodes	9	4	4	0.350
Duration of hypoglycemia per episode (min)	40 ± 27	53 ± 48	40 ± 7	0.264
AUC for hypoglycemia per episode	31 ± 26	51 ± 55	35 ± 14	0.554
Mean overnight glucose (mmol/L)	6.8 ± 2.5	7.0 ± 2.8	7.2 ± 2.1	0.407

Data are *n*/*n* (%), *n*, or means ± SD. *P* values are for Friedman two-way ANOVA by ranks. AER, aerobic exercise; No-Ex, no-exercise control; RES, resistance exercise.

magnitude than the one used in the current study) resulted in a fourfold increase in blood lactate levels, with elevated lactate persisting for at least 30 min postexercise in individuals without diabetes (23). While we are unaware of published data on lactate production during resistance exercise in individuals with type 1 diabetes, there is no reason to believe that lactate production would be impaired in this population. Indeed, other anaerobic activity (high-intensity cycling) produced elevated lactate levels persisting up to 30 min postexercise in individuals with type 1 diabetes (11–14,25). We did not measure lactate in the current study but can surmise that blood lactate levels would have increased more during resistance exercise where glycolysis predominates (22) than during aerobic exercise where lipolysis generates much of the energy required (26), especially in physically fit individuals (21). Higher lactate levels could potentially attenuate declines in blood glucose by stimulating gluconeogenesis.

Overall, there were no significant differences among the conditions with respect to any measures of hypoglycemia or mean nocturnal blood glucose levels (Table 2), although resistance exercise was associated with a nonsignificant trend for more nocturnal hypoglycemia. While we are unaware of any study examining nocturnal blood glucose levels after resistance exercise in type 1 diabetic subjects, McMahon et al. (16) found that adolescents with type 1 diabetes had a higher glucose infusion requirement to maintain euglycemia between midnight and 4:00 A.M. after performing evening aerobic exercise than if no exercise had been performed. This coincides with the time when the lowest nocturnal glucose levels were found after both exercise sessions in our study (Fig. 2), although differences among conditions were not significant. As McMahon et al. (16) surmised that delayed increases in postexercise glucose needs relate to replenishment of glycogen stores, a higher frequency of low blood glucose after resistance exercise (which relies more on glycogen for fuel) (22) might be expected.

It is also possible that differences in food and insulin intake (Table 2), while not statistically significant, could have had a minor effect on postexercise glucose profiles. In addition, while participants were asked to match their food and insulin intake both pre- and postexercise as closely as possible among the sessions, some differences may not have been reported. This does not, however, detract from the findings, as patient decisions regarding insulin dosage and carbohydrate intake play an essential role in diabetes management. As there is currently very little information available with respect to insulin adjustments for resistance exercise, participants in the current study were relying to a great extent on personal experience and judgment.

These findings have important clinical implications. Higher physical activity levels in individuals with type 1 diabetes have been associated with lower frequency and severity of diabetes complications (1); however, fear of hypoglycemia is generally the strongest barrier to physical activity for this population (27). Resistance exercise is associated with improvements in muscular strength (4), improved lipid profiles (4), lower insulin needs (4,5), and lower self-monitored blood glucose levels (4,5) in individuals with type 1 diabetes. It also carries many of the same benefits as aerobic exercise (higher bone mineral density, increased insulin sensitivity, and improved cardiovascular function) (28) and may therefore be a safe and effective option for this population. Interestingly, we observed more exercise-associated glycemic fluctuation with aerobic exercise compared with resistance exercise. During the activity, aerobic exercise was associated with greater reductions in glycemia, while in early recovery there was more rebound hyperglycemia compared with resistance exercise. Thus, one could conclude that resistance exercise may be more beneficial as far as glucose stability is concerned. Moreover, as individuals with type 1 diabetes may also have an increased risk of myopathy (29) and complications associated with insulin resistance (29,30), performing regular resistance exercise may help maintain or improve muscle mass and metabolism. Meanwhile, it should also be noted that postexercise hypoglycemia might occur more frequently in individuals who have changed their exercise routine to incorporate resistance training or for patients unaccustomed to exercise (15).

In summary, our findings suggest that in trained individuals with type 1 diabetes who habitually practice both aerobic and resistance exercise, resistance exercise may result in more stable glucose levels both during and after exercise than aerobic exercise, which may explain the beneficial effects on HbA_{1c} found in previous intervention studies involving resistance exercise. The trend toward more frequent, albeit mild, nocturnal hypoglycemia after resistance exercise reported in our study, however, indicates the possible need to develop more effective clinical management protocols for different forms of exercise.

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potential conflicts of interest relevant to this article were reported.

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J.E.Y. contributed to the conception and design of the project, contributed to the discussion, collected and analyzed data, and drafted, reviewed, and edited the manuscript. G.P.K., B.A.P., and M.C.R. contributed to the conception and design of the project, researched data, contributed to the discussion, and reviewed and edited the manuscript. N.B. contributed substantially to the acquisition of data. J.M. and P.B. contributed to the discussion and reviewed and edited the manuscript. F.K. took the lead in data analysis, contributed to the discussion, and reviewed and edited the manuscript. R.J.S contributed to the conception and design of the project, researched data, contributed to the discussion, and reviewed and edited the manuscript. R.J.S. 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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