

# Effects of Performing Resistance Exercise Before Versus After Aerobic Exercise on Glycemia in Type 1 Diabetes

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**OBJECTIVE**—To determine the effects of exercise order on acute glycemic responses in individuals with type 1 diabetes performing both aerobic and resistance exercise in the same session.

**RESEARCH DESIGN AND METHODS**—Twelve physically active individuals with type 1 diabetes ( $HbA_{1c}$   $7.1 \pm 1.0\%$ ) performed aerobic exercise (45 min of running at  $60\% \dot{V}O_{2peak}$ ) before 45 min of resistance training (three sets of eight, seven different exercises) (AR) or performed the resistance exercise before aerobic exercise (RA). Plasma glucose was measured during exercise and for 60 min after exercise. Interstitial glucose was measured by continuous glucose monitoring 24 h before, during, and 24 h after exercise.

**RESULTS**—Significant declines in blood glucose levels were seen in AR but not in RA throughout the first exercise modality, resulting in higher glucose levels in RA (AR =  $5.5 \pm 0.7$ , RA =  $9.2 \pm 1.2$  mmol/L,  $P = 0.006$  after 45 min of exercise). Glucose subsequently decreased in RA and increased in AR over the course of the second 45-min exercise bout, resulting in levels that were not significantly different by the end of exercise (AR =  $7.5 \pm 0.8$ , RA =  $6.9 \pm 1.0$  mmol/L,  $P = 0.436$ ). Although there were no differences in frequency of postexercise hypoglycemia, the duration (105 vs. 48 min) and severity (area under the curve 112 vs. 59 units  $\cdot$  min) of hypoglycemia were nonsignificantly greater after AR compared with RA.

**CONCLUSIONS**—Performing resistance exercise before aerobic exercise improves glycemic stability throughout exercise and reduces the duration and severity of postexercise hypoglycemia for individuals with type 1 diabetes.

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Regular physical activity is associated with greater longevity and lower frequency and severity of diabetes complications in individuals with type 1 diabetes (1,2). The type of exercise to recommend for potential improvements in glycemia in this population is still uncertain. Intervention studies of aerobic exercise training have not shown consistent effects on blood glucose control, as

measured by  $HbA_{1c}$  (3). Two small ( $n = 8$ – $10$ ) studies examining the chronic effects of resistance exercise training have found  $\sim 1$  percentage point reductions in  $HbA_{1c}$  (4,5).

Including short bursts of intense activity, where anaerobic metabolism plays a major role in providing fuel, may assist in preventing hypoglycemia during and up to 2 h postexercise in individuals with type 1

diabetes (6–9). However, two studies using continuous glucose monitoring (CGM) systems suggested that the risk of nocturnal hypoglycemia after such exercise sessions is increased (10,11) and perhaps even more than after moderate aerobic activity (11). The effects of resistance training, another form of anaerobic exercise, on acute glycemia in type 1 diabetes is currently unclear. In one study, insulin sensitivity (measured by euglycemic clamp) was unchanged 12 and 36 h after resistance exercise, thereby suggesting that resistance exercise may not cause as much of a postexercise hypoglycemic response compared with aerobic exercise (12).

The American Diabetes Association Standards of Medical Care (13) encourages individuals with diabetes to follow the U.S. Department of Health and Human Services' Physical Activity Guidelines (14), which suggest including both aerobic and resistance exercise in fitness programs. Individuals who are actively engaged in training often wish to perform both types of exercise within the same session. We previously found that aerobic exercise causes a more rapid decrease in blood glucose and a greater need for carbohydrate supplementation during exercise than resistance exercise (15). We are unaware of previous research examining the acute effects in individuals with type 1 diabetes of combining these exercise modalities in a single session or whether there is an advantage related to the order in which they are undertaken. We sought to determine if the order of exercise in combined sessions has a differential effect on blood glucose during and postexercise (as measured by CGM) in this population.

In individuals without diabetes, performing aerobic exercise immediately after resistance exercise results in an increased reliance on lipids as a fuel source during activity (16). We therefore hypothesized that performing resistance exercise before aerobic exercise would lead to less of a decline in blood glucose during exercise in individuals with type 1 diabetes than when exercise is performed in the opposite order. Because performing resistance exercise first may result in a diminished

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reliance on carbohydrate for fuel during exercise, we anticipated that less nocturnal hypoglycemia would be found where aerobic exercise was preceded by resistance exercise.

## RESEARCH DESIGN AND METHODS

The University of Ottawa and Ottawa Hospital Research Ethics Boards approved the experimental protocol. We recruited 12 nonobese adults with type 1 diabetes. Participants performed aerobic and resistance exercise at least three times per week and had been doing so for at least 6 months (Table 1).

### Experimental design

The research took place in the Human and Environmental Physiology Research Unit at the University of Ottawa. After being informed of the purpose, protocol, and possible risks of the study, participants gave written consent and completed physical activity readiness questionnaires (American Heart Association/American College of Sports Medicine Health/Fitness Facility Preparticipation Screening Questionnaire). Hand-held glucose meters (OneTouch Ultra, Lifescan, Johnson & Johnson, Milpitas, CA) and test strips (with identical code) were provided for capillary glucose tests.

On a separate visit, participants underwent an incremental workload running test on a treadmill with a monitored electrocardiogram (Quinton Q4500, Quinton, Bothell, WA) to determine peak oxygen consumption ( $\dot{V}O_{2\text{peak}}$ ).  $\dot{V}O_{2\text{peak}}$  was determined by measuring the volume and

concentration of expired oxygen and carbon dioxide (AMETEK model S-3A/1 and CD 3A, Applied Electrochemistry, Pittsburgh, PA). Muscular strength (eight repetition maximum [8-RM]) was recorded as the maximum weight that participants could lift eight times with good form for chest press (pectoralis major), leg press (quadriceps, biceps femoris, gluteus maximus), seated row (latissimus dorsi, rhomboids, trapezius), leg curl (biceps femoris), shoulder press (deltoids), and lat pull-down (latissimus dorsi). A venous blood sample was drawn for determination of HbA<sub>1c</sub>, which was measured by automated heterogeneous immunoassay with latex-enhanced turbidimetric detection on a Roche Cobas Integra 800 analyzer (Roche Diagnostics Corp., Indianapolis, IN).

### CGM

The CGMS System Gold (Medtronic, Northridge, CA) was used in this study. Participants were blinded to their glucose values and could not change their regular behavior patterns based on real-time glucose monitoring. Twenty-four hours before each experimental session, the CGM sensor was inserted subcutaneously in the abdomen or in the upper gluteal area. The same insertion site was used for both trials. Training on calibration and operation of the CGM units was provided. Participants performed capillary glucose tests and calibrated the CGM unit four times daily using the hand-held glucose meter provided. On the third day, 24 h postexercise, participants removed the sensors and the researchers retrieved the monitors. Data were downloaded using a Medtronic Com-Station and Minimed Solutions Software version 3.0 (Medtronic).

Participants maintained diaries of food intake and insulin administration while wearing the CGM sensor. They ate the same breakfast, same lunch, and same supper each day of sensor wear and kept their insulin doses the same each of these days to the greatest extent possible. Participants avoided exercise (apart from that performed in our laboratory) for 24 h before inserting the sensor (48 h before each study exercise session) as well as during the 3 days of sensor wear. They also avoided caffeine and alcohol during this time.

### Experimental sessions

Participants arrived at the laboratory at 1600 h. Intravenous catheters were inserted soon after arrival. Exercise started at 1700 h for all participants. Each participant

performed two experimental sessions in random order separated by at least 5 days:

1. Aerobic exercise before resistance exercise session (AR): A 45-min bout of moderate-intensity aerobic exercise (treadmill running at 60% of their predetermined  $\dot{V}O_{2\text{peak}}$ ), followed by a 45-min bout of resistance training (three sets of eight repetitions with 90 s of rest between sets).
2. Resistance exercise before aerobic exercise session (RA): The same exercises as above were performed, with the resistance exercise completed before the aerobic exercise.

Sessions were followed by 1 h of monitored recovery in a resting state. Women were using monophasic oral contraceptives and were tested during the active pill consumption phase.

### Measurements

On the days that exercise was scheduled (day 2 of each 3-day monitoring period), participants were asked to decrease their insulin doses (a 10% decrease in long- or intermediate-acting for patients receiving multiple daily injections and a 50% decrease in basal rate 1 h pre-exercise for patients receiving a continuous subcutaneous insulin infusion). A further 25% basal rate decrease was made for continuous subcutaneous insulin infusion patients if their capillary glucose was  $\leq 5$  mmol/L upon arrival at the laboratory. Adjusted rates were maintained throughout exercise. Standardized snacks (Glucerna Chocolate Graham Snack Bars, 150 calories, 25 g of carbohydrate; Abbott Laboratories, Abbott Park, IL) were provided and consumed at 1600 h each day of monitoring.

Before starting exercise, participants were required to have blood glucose levels between 5.5 and 13.9 mmol/L. Capillary glucose tests were performed upon arrival at the laboratory, 30 min before and immediately before exercise. If capillary glucose levels were  $< 4.5$  mmol/L, participants were provided with 32 g of glucose (Dex4, AMG Medical, Montreal, QC, Canada) before levels were checked again 15 min later. If initial readings were between 4.5 and 5.4 mmol/L, participants were given 16 g of glucose. These steps were repeated until a level of  $\geq 5.5$  mmol/L was achieved.

Glucose concentrations during exercise were monitored by applying a drop of venous blood to a test strip inserted in the study hand-held glucose meter when

Table 1—Participant characteristics

|  | n or<br>mean $\pm$ SD |
|--|-----------------------|
| Patients   | 12                    |
| Sex  |                       |
| Male   | 10                    |
| Female   | 2                     |
| Age (years)  | 31.8 $\pm$ 15.3       |
| Height (m)   | 1.77 $\pm$ 0.07       |
| Weight (kg)  | 79.2 $\pm$ 10.4       |
| BMI (kg/m <sup>2</sup> )   | 25.3 $\pm$ 3.0        |
| $\dot{V}O_{2\text{peak}}$<br>(mL O <sub>2</sub> · kg <sup>-1</sup> · min <sup>-1</sup> ) | 51.2 $\pm$ 10.8       |
| HbA <sub>1c</sub> (%)  | 7.1 $\pm$ 1.1         |
| Diabetes duration (years)  | 12.5 $\pm$ 10.0       |
| Insulin delivery   |                       |
| Multiple daily injections  | 5                     |
| Continuous subcutaneous<br>insulin infusion  | 7                     |

venous blood samples were collected. When levels were  $<4.5$  mmol/L, exercise was interrupted and participants were provided with 16 g of glucose. Capillary glucose tests were then performed every 10 min, and an additional 16 g of glucose was provided when necessary until a level of  $\geq 5.5$  mmol/L was achieved and exercise resumed. Oxygen consumption was measured using a portable gas analysis system (Oxycon Mobile, Jaeger, Hoechberg, Germany). Energy expenditure was calculated as described elsewhere (17).

### Blood analyses

Venous blood samples were collected at baseline, 5, 10, 15, 30, 45, 50, 55, 60, 75, and 90 min during exercise and at 5, 10, 15, 20, 30, 40, 50, and 60 min after exercise. Blood was drawn using 5 mL sterile plastic syringes and transferred immediately into 5.4 mL plasma ( $K_2$ EDTA) BD Vacutainer tubes (BD, Franklin Lakes, NJ), mixed by inversion and centrifuged immediately. Plasma aliquots were transferred into 1.5 mL microcentrifuge tubes and stored at  $-80^\circ\text{C}$  until analyzed. Plasma glucose concentrations were determined using the hexokinase timed end point method on the Beckman Coulter Unicel DxC600 Synchron Clinical Analyzer (Beckman Coulter Inc., Fullerton, CA) with SYNCHRON CX Systems GLUCOSE reagent (Cat#442640).

### Statistical analyses

Exercise and recovery periods were examined separately. Plasma glucose concentration was compared between treatments using two-way repeated-measures ANOVA with the factors of time (exercise: 5, 10, 15, 30, 45, 50, 55, 60, 75, and 90 min; recovery: 5, 10, 15, 20, 30, 40, 50, and 60 min) and treatment (RA or AR). Paired sample *t* tests were used to perform pairwise post hoc comparisons between treatments for each time point to examine within-treatment changes from baseline and changes throughout recovery. The level of significance was set at 0.05. Energy expenditure during the exercise sessions (including the recovery) was compared using a paired sample *t* test.

CGM data were grouped and summarized as follows: 24 h and overnight (2400 to 0600 h) pre-exercise, as well as 24 h and overnight postexercise. Hypoglycemia was defined as any value  $<3.5$  mmol/L detected by CGM, and values  $>10.9$  mmol/L were categorized as hyperglycemic. Total time spent in hypoglycemia, euglycemia, and hyperglycemia for the predetermined periods and the area

under the curve (AUC, defined as the absolute distance from the described limits, multiplied by the time spent outside those limits) for time spent hypo- and hyperglycemic was determined along with the maximum, minimum, and mean interstitial glucose for each time period. Variables were compared between exercise treatments, and pre- and postexercise values were compared within treatments using related-samples Wilcoxon signed rank tests. These tests were also used to examine differences in insulin and carbohydrate intake (calculated from the participants' food and insulin diaries) between days within exercise treatments (day 1 vs. 2), and between exercise treatments (days 1 through 3). Pearson correlation analyses were performed comparing capillary glucose values recorded by the participants during nonexercise periods to CGM data to assess the accuracy of the sensors throughout each 3-day measuring period. Analyses were performed using SPSS 18.0 software (SPSS Inc., Chicago, IL). Data are presented as means  $\pm$  SD.

**RESULTS**—Energy expenditure was measured during exercise and recovery together for both sessions in 9 of the 12 participants. There were no differences in energy expenditure between AR ( $4,277 \pm 729$  kJ) and RA ( $4,247 \pm 589$  kJ).

### Plasma glucose

Plasma glucose levels for exercise and recovery (Supplementary Table 1) are plotted in Fig. 1. A significant effect of time ( $P = 0.001$ ) and an interaction of treatment and time ( $P = 0.004$ ) were found in examining plasma glucose levels during exercise (Fig. 1). Differences between treatments were not significant at baseline. The aerobic exercise performed in the AR treatment caused a substantial decline in blood glucose concentration, resulting in plasma glucose levels that were lower than baseline within the first 10 min of exercise, persisting until the end of aerobic exercise ( $9.1 \pm 2.4$  at baseline;  $5.5 \pm 2.4$  mmol/L at 45 min;  $P < 0.01$ ) and continuing into resistance exercise. Glucose then increased during resistance exercise, producing levels that were similar to baseline by the end of exercise. Conversely, the RA treatment did not produce significant changes from baseline during resistance exercise. After the change in exercise modality in RA, glucose levels were only significantly different from baseline after 75 ( $P = 0.044$ ) and 90 ( $P = 0.018$ ) min of exercise. Glucose

was lower in the AR treatment than in the RA treatment until the end of exercise, with differences achieving statistical significance between 30 and 60 min ( $P < 0.05$ ).

During the postexercise recovery period, there was a significant effect of time ( $P < 0.01$ ) for changes in plasma glucose, but no effect of treatment or interaction of treatment and time. Significant increases in plasma glucose from the end of exercise were seen throughout recovery after AR where none were observed after exercise in RA (Fig. 1).

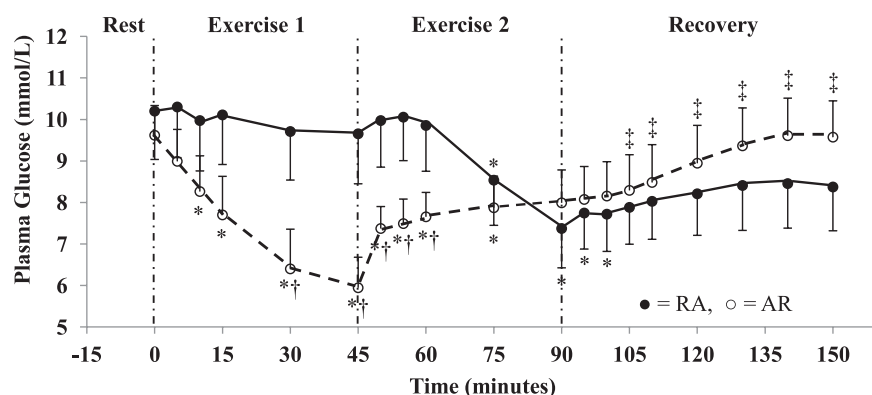
### Carbohydrate intake and insulin dosage

Total daily insulin doses did not differ significantly between treatments on the first 2 days of CGM wear before the experimental exercise session, or between the first and second day within each treatment. Insulin adjustments for exercise were similar between treatments (Supplementary Table 2). On the day after the exercise testing session, insulin intake was lower after the AR session compared with RA ( $36.1 \pm 16.3$  vs.  $38.8 \pm 18.5$  units,  $P = 0.028$ ). Ten of 12 participants required carbohydrate supplementation during the AR session compared with only 6 of 12 during RA; however, there were no statistically significant differences between groups in total carbohydrate intake during exercise and recovery in the laboratory (Supplementary Table 3) and in the 6 h after exercise (Supplementary Table 4).

### Interstitial glucose levels

Pearson correlations between capillary glucose readings from nonexercise periods and sensor readings over the monitoring period were 0.95 for AR and 0.91 for RA ( $P < 0.001$ ). There were no significant differences between treatments with respect to hypoglycemia and hyperglycemia (number of excursions, time, AUC) as well as mean, maximum, and minimum glucose on the night before or 24 h before exercise.

Mean postexercise overnight CGM profiles are provided in Fig. 2. In the RA treatment, average maximum nocturnal glucose levels were significantly lower after exercise than the previous (nonexercise) night (pre-exercise =  $9.5 \pm 3.0$  mmol/L, postexercise maximum =  $8.8 \pm 4.0$  mmol/L;  $P = 0.04$ ). Within the AR treatment, there was a trend toward greater AUC postexercise for nocturnal hypoglycemia ( $P = 0.06$ ) compared with RA.



**Figure 1**—Mean  $\pm$  SE plasma glucose during exercise and recovery for aerobic exercise performed before resistance exercise (AR, dashed line with  $\circ$ ) and resistance exercise performed before aerobic exercise (RA, solid line with  $\bullet$ ) ( $n = 11$ ). \*Difference from baseline during exercise where  $P < 0.05$ . †Difference between conditions where  $P < 0.05$ . ‡Change throughout recovery from end-exercise level where  $P < 0.05$ .

Although the frequency of nocturnal hypoglycemic events did not differ between the two exercise sessions, the duration and depth of hypoglycemia tended to be longer and more severe after AR than after RA (Table 2).

**CONCLUSIONS**—This study evaluated, in the context of a combined resistance and aerobic exercise session, the effects of exercise order on blood glucose levels in individuals with type 1 diabetes. As we had anticipated, performing resistance exercise before aerobic exercise rather than the reverse resulted in attenuated declines in glucose concentration during exercise, fewer exercise-induced hypoglycemic events, and less need for carbohydrate supplementation. Furthermore, we observed beneficial effects from this sequence on subsequent 12-h glycemic trends where the duration and severity of

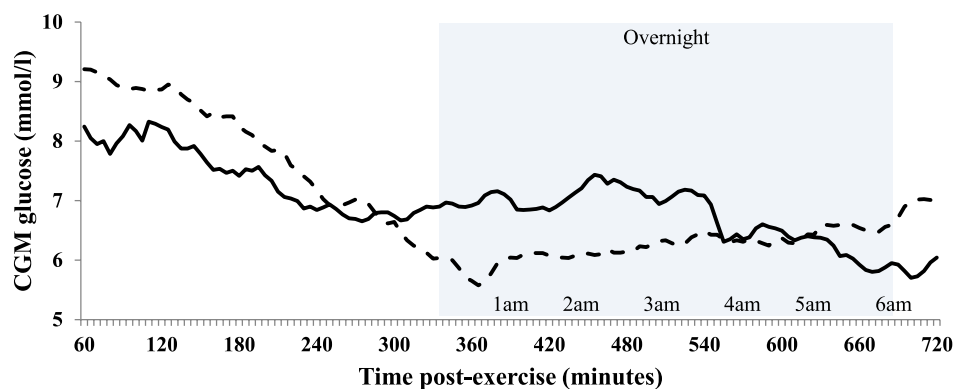
hypoglycemia was reduced. The benefits of performing resistance exercise before aerobic exercise instead of the reverse were observed despite overall energy expenditure being equal between experimental sessions.

Resistance exercise is a primarily anaerobic activity. Other types of high-intensity exercise combining aerobic and anaerobic metabolism (e.g., high intensity cycling) can increase the rate of glucose appearance to a greater extent than the rate of glucose utilization (seven and four times, respectively) during exercise in type 1 diabetes (18). This may cause glucose levels to increase during exercise, producing postexercise hyperglycemia if intense exercise is sustained for 12 or more minutes (19). Shorter anaerobic exercise bouts (intermittent 4-s sprints or 10-s sprints before or after low-intensity aerobic exercise) attenuated declines in

blood glucose both during and after exercise when combined with low-intensity (40%  $\dot{V}O_{2peak}$ ) cycling (6–8). Elevated glucose production from very high-intensity exercise is generally attributed to increased levels of circulating epinephrine [known to triple with short sprints (6,8,9) and increase up to 14 times its resting value (18) after 12 min of exhaustive cycling] and norepinephrine, which augment glycogenolysis throughout exercise and early recovery (18,19).

Although we did not measure catecholamines during the sessions, responses to high-intensity exercise are known to be comparable (18,20) or slightly attenuated (19,21) in individuals with type 1 diabetes compared with nondiabetic counterparts. Catecholamines can increase to three or four times resting values during moderate-intensity resistance exercise in individuals without diabetes (22), with responses increasing in proportion to exercise intensity (23). If our participants experienced similar responses to resistance exercise as individuals without diabetes, then increases in epinephrine may have contributed to the attenuated rate of decline in blood glucose during the first 15 min of aerobic exercise in RA, and to the increase in glucose during resistance exercise in AR (Fig. 1). The latter should be interpreted with caution, because most participants needed glucose supplements to prevent hypoglycemia during aerobic exercise in this session.

It is also possible that exercise-related growth hormone (GH) secretion differed between treatments, potentially affecting fuel selection during exercise. Goto et al. (24) found that in nondiabetic individuals, endurance exercise performed before resistance exercise produced lower GH



**Figure 2**—Mean glucose ( $n = 12$ ) as measured by continuous glucose monitoring from 1 to 12 h after exercise following aerobic exercise performed before resistance exercise (AR, dashed line) and resistance exercise performed before aerobic exercise (RA, solid line). (A high-quality color representation of this figure is available in the online issue.)

**Table 2—Summary of overnight (2400 to 0600 h) continuous glucose monitoring data for the night before and the night after exercise\***

|   | AR<br>(n = 12) | RA<br>(n = 12) |
|---|----------------|----------------|
| Night before exercise session                     |                |                |
| Participants experiencing nocturnal hypoglycemia† | 4 (30)         | 5 (42)         |
| Total hypoglycemic episodes                       | 7              | 4              |
| Duration of hypoglycemia per episode (min)        | 97.5 ± 84.9    | 47.1 ± 32.8    |
| AUC for hypoglycemia per episode (mmol · min)     | 112.3 ± 97.6   | 42.3 ± 41.9    |
| Overnight glucose (mmol/L)                        | 6.7 ± 3.2      | 6.9 ± 2.7      |
| Night after exercise session                      |                |                |
| Participants experiencing nocturnal hypoglycemia† | 3 (25)         | 4 (30)         |
| Total hypoglycemic episodes                       | 5              | 6              |
| Duration of hypoglycemia per episode (min)        | 105 ± 116      | 48 ± 68        |
| AUC for hypoglycemia per episode (mmol · min)     | 110 ± 146      | 59 ± 110       |
| Mean overnight glucose (mmol/L)                   | 6.3 ± 2.4      | 6.7 ± 3.1      |

Categoric data are expressed as *n* or *n* (%), and continuous data are presented as mean ± SD. \*No significant differences between pre- and postexercise, or between exercise conditions. †Defined as glucose <3.5 mmol/L.

secretion than resistance exercise alone. They also found that resistance exercise performed 20 min or less before endurance exercise produced elevated levels of GH and greater rates of lipolysis during the subsequent aerobic activity compared with endurance exercise alone (16). Because higher GH levels are known to decrease muscle glucose uptake and increase lipolysis in nondiabetic individuals (25), this may have been a factor in the attenuated declines in blood glucose during aerobic activity in RA.

High-intensity cycling increases blood lactate levels during and up to 40 min after exercise in individuals with type 1 diabetes (6,7,9,18,19). We are unaware of published data describing lactate responses to resistance exercise in this population. Resistance exercise protocols similar to the one we used have produced lactate concentrations up to four times those measured at rest, with levels remaining significantly higher than baseline until 30 min postexercise in trained nondiabetic individuals (26). Because elevated lactate could serve to increase gluconeogenesis (7), it could be a contributing factor in the attenuated decline in glucose during the first 15 min of aerobic exercise in RA as well as in the increases in postexercise glucose levels in AR.

Studies suggest that high-intensity exercise may be associated with a greater frequency of nocturnal hypoglycemia in type 1 diabetic individuals (10,11). Our participants experienced nocturnal hypoglycemia

as frequently postexercise as on non-exercise nights. Nocturnal hypoglycemia has been identified as a risk inherent with intensive insulin therapy (27), and it is possible that overnight hypoglycemia in our study was more related to insulin therapy than to exercise. It is noteworthy that hypoglycemic events occurring after AR tended to be longer and more severe than those experienced in RA, as demonstrated by a greater AUC. Studies using glucose clamp found that counter-regulatory responses to subsequent hypoglycemia were blunted after exercise, even in the absence of significant changes in glucose levels during exercise (28). In addition, even mild hypoglycemia (3.9 mmol/L) in nondiabetic individuals is sufficient to elicit counter-regulatory reactions that can blunt neuroendocrine responses to subsequent hypoglycemia within 24 h (29). Because decreases in blood glucose were greater during AR (reaching a mean of 5.5 ± 2.4 vs. 6.9 ± 3.1 mmol/L in RA), it is plausible that subsequent responses to declining blood glucose could have been subject to impairment after exercise.

Although there are advantages to admitting study subjects the night before testing to control participant activity and food intake, we chose a study design more reflective of real-life conditions. Participants controlled their meals and insulin but were asked to eat the same breakfast, lunch, and dinner at the same time for every day of sensor wear and to match their insulin intake as closely as possible. Exercise took place at 1700 h when many

individuals who work during the day opt to exercise, unlike several other studies where midmorning exercise was performed (6–9,18,19).

Several aspects of resistance training in type 1 diabetic individuals require further scrutiny. Glucose responses may be different if exercise is performed at another time of day because hormone and exogenous insulin concentrations are both likely to be different. Our participants were fit, habitual exercisers, and the effects of exercise may be less pronounced in unfit individuals exercising at the same relative intensity because the activity would be at a lower absolute intensity. In nondiabetic subjects running at very high relative intensity, glucose production and catecholamine concentrations increase more in athletes than in physically untrained individuals, resulting in hyperglycemia after exercise in the former group because glucose production falls more slowly than glucose utilization when exercise ends (30). Further research on different subpopulations of type 1 diabetic individuals, including those with lower fitness levels and poorer glycemic control, is warranted.

This study is limited by its small sample size (*n* = 12), which may have prevented us from finding all of the significant differences in plasma glucose levels during exercise. To examine our participants in a real-life scenario, we compromised a certain amount of experimental control such as having complete control over all food and insulin intake. The ability to interpret the data would have been improved by having catecholamine, lactate, and GH measurements. Finally, having a relatively fit sample with moderate to good control of their diabetes makes the applicability of the outcomes to individuals who are inactive or have poor glycemic control uncertain.

In summary, our findings suggest that trained individuals with type 1 diabetes who perform both resistance and moderate aerobic exercise should consider performing their resistance exercise first if they tend to develop exercise-associated hypoglycemia because doing so may attenuate declines in glucose levels during subsequent aerobic exercise. This order of exercise could lead to a lower reliance on glucose supplementation during exercise and might also decrease the severity of potential nocturnal hypoglycemia. Conversely, individuals having exercise-associated hyperglycemia may wish to perform aerobic exercise before resistance training.

Both approaches should still be accompanied by careful monitoring of blood glucose levels, both during and after exercise.

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J.E.Y. contributed to the conception and design of the project, contributed to discussion, collected and analyzed the data, and drafted, reviewed, and edited the manuscript. G.P.K., B.A.P., M.C.R., and R.J.S. contributed to the conception and design of the project, researched data, contributed to discussion, and reviewed and edited the manuscript. 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. 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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