Resistance versus aerobic exercise: acute effects on glycemia in type 1 diabetes

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ABSTRACT

OBJECTIVES: In type 1 diabetes, small studies found that resistance exercise (weight lifting) reduced HbA_{1c}. In the present study, we examined the acute impacts of resistance exercise on glycemia during exercise and in the subsequent 24 hours, compared to aerobic exercise and no exercise.

RESEARCH DESIGN AND METHODS: 12 physically active individuals with type 1 diabetes (HbA_{1c}=7.1±1.0%) performed 45 minutes of resistance exercise (three sets of seven exercises at eight repetition maximum), 45 minutes of aerobic exercise (running at 60% of $\dot{V}O_{2peak}$) 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 following 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 four and a half to six hours post-exercise 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 post-exercise 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.

Key words: continuous glucose monitoring, hypoglycemia, glucose, resistance exercise, type 1 diabetes

The frequency and severity of complications in individuals with type 1 diabetes is 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 to 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, high intensity intermittent exercise), alone or combined with moderate intensity aerobic exercise, produce smaller declines in blood glucose during activity and up to 2 hours post-exercise 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 post-exercise 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 to that of aerobic exercise. The

risk of nocturnal hypoglycemia associated with restoration of muscle glycogen stores following 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 hours post-exercise compared to aerobic exercise or no exercise in individuals with type 1 diabetes. We hypothesized that, compared to 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 following the exercise, thereby potentially improving overall glucose stability.

RESEARCH DESIGN AND METHODS

The study was approved by the Research Ethics Boards of the University of Ottawa and the Ottawa Hospital. Non-obese, non-smoking adults with complication-free type 1 diabetes were recruited. Two of the participants were competitive athletes training six 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 six months. Participants were using either multiple daily injections (MDI) of insulin or continuous subcutaneous insulin infusion (CSII) 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 peak oxygen consumption ($\dot{V}O_{2peak}$), muscular strength [eight repetition maximum-(8RM)] and HbA_{1c} as described elsewhere (20).

Continuous Glucose Monitoring

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 behaviour based on real-time glucose monitoring. CGMS® sensors were inserted subcutaneously at 0830 h the day before the testing session. OneTouch® UltraSmart® handheld glucose meters (Lifescan, Johnson & Johnson, Milpitas, USA) and coded strips (same code throughout the study) were provided for capillary glucose tests. Participants tested capillary glucose for CGM calibration purposes 4 times daily. Twenty-four hours after the end of the exercise/no-exercise control session, CGM units were retrieved and data 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 hours prior to insertion of the sensor (48 hours prior to the experimental session), and avoided caffeine and alcohol during the monitoring period.

Experimental sessions

Participants arrived at the lab at 1600 h on the day following the sensor insertion. The following sessions were performed, separated by at least five days:

1) Resistance exercise: Three sets of 8-RM of seven different exercises with 90 seconds rest between sets (duration ~ 45 minutes);

2) Aerobic exercise: 45 minutes of treadmill exercise (60% of $\dot{V}O_{2peak}$.) and

3) No-exercise control: 45 minutes of seated rest.

Sessions were followed by 60 minutes 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 either making a 10% decrease in intermediate or long acting insulin (MDI), or a 50% decrease in basal rate starting an hour prior to 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 (Abbott Laboratories, Abbott Park, IL) – 150 calories, 25 g of carbohydrate] at 1600 h every day, including the exercise day, where the bar was consumed upon arrival at the laboratory.

Capillary glucose was checked 60 and 30 minutes before and immediately prior to exercise to ensure glucose levels >5.5 and <13.9 mmol/l. Glucose tablets were provided when necessary, and as described in detail elsewhere (20).

Blood sampling and analyses

Venous blood samples were collected through an intravenous catheter at baseline, 5, 10, 15, 30, and 45 minutes 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 minute time points during

recovery. Blood was immediately mixed by inversion, centrifuged (4000 rev/min for 4 minutes) and stored at -80ºC. The hexokinase timed endpoint method was used to determine plasma glucose levels using the Beckman Coulter Unicel ®DxC600 Synchron® Clinical Analyzer (Beckman Coulter Inc., Fullerton, CA) and SYNCHRON CX® Systems GLUCOSE reagent (Cat#442640).

STATISTICAL ANALYSES

Glucose levels were compared among sessions using two-way repeated-measures (Time and Condition) analysis of variance (ANOVA). Exercise and recovery periods were examined separately among the three sessions (aerobic, resistance, no-exercise control). The exercise period consisted of the 5, 10, 15, 30, and 45 minute time points, while the recovery period consisted of the remaining time points. Paired samples t-tests were used to perform pair-wise 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-minute averages in the following windows: 24 hours pre-exercise, overnight (2400 to 0600 h) pre-exercise, 1 to 6 hours post-exercise, overnight post-exercise and 24 hours post-exercise. A two-way (time and condition) repeated-measures ANOVA was used to compare among conditions in the one to six hour post-exercise period. Paired sample t-tests were then used to perform pair-wise post-hoc comparisons for each 15 minute 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 hypoglycemia and hyperglycemia, and areas under the curve (AUC**)** for time spent in hypo- and hyperglycemia

were determined for each window. Pre-exercise values were compared to post-exercise values within exercise conditions using related-samples Wilcoxon Signed Ranks tests. Differences among conditions were examined using related-samples Friedman's Two-Way ANOVA by Ranks. Agreement between CGM data and capillary glucose over the three days was determined by performing Pearson correlations between sensor glucose and self-recorded capillary glucose values.

Daily total insulin and carbohydrate intake were calculated based on the information provided in participant logs. Comparisons among conditions for each day were performed using related-samples Friedman's Two-Way ANOVA by Ranks. Where significant results were found, related-samples Wilcoxon Signed Ranks tests ensued to determine where the differences lay. Analyses were performed using SPSS 18.0 for Windows (SPSS Inc. Chicago, IL).

RESULTS

Twelve (10 male, 2 female), non-obese (BMI=25.3±3.0 $kg \cdot m^{-2}$), physically active (VO_{2peak}=51.2±10.8 $ml \cdot kg^{-1} \cdot min^{-1}$) individuals aged 17 to 62 years (mean age 31.8±15.3 years) took part in the study. Mean diabetes duration was 12.5 years (±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 CSII.

Plasma glucose

Exercise

Plasma glucose levels are plotted in Figure 1. Information regarding treadmill speeds/inclines as well as the workloads for the resistance exercise sessions are provided in

Supplementary Table S-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 (Figure 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 minute 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 minutes 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 minutes ($P=0.001$)], resulting in significant changes from baseline within 10 minutes. Glucose levels in the aerobic session were lower than the no-exercise session after 30 minutes 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 following 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 both no-exercise and resistance exercise at 60 minutes post-exercise.

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 hours following exercise (Table 1); however carbohydrate intake was higher on the exercise testing day in the aerobic exercise session when compared to the resistance exercise session (P=0.013), mostly due to 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 hand-held meters and interstitial glucose levels measured by CGM were 0.95, 0.90 and 0.94 during non-laboratory periods in the resistance exercise, aerobic, and no-exercise control sessions respectively. During the 24 hours before either exercise trials or no-exercise control, there were no significant differences among sessions in the total time spent in hypoglycaemia, AUC for hypoglycemia, number of hyperglycemic events, time spent in hyperglycemia, AUC for hyperglycemia, or mean blood glucose.

Post-exercise CGM data were only available for 11 and 10 out of 12 participants in the no-exercise and aerobic exercise sessions respectively, due to 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 hand held 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 one to six hours post-exercise. Higher mean interstitial glucose concentrations were found in the fourth and fifth hours following the aerobic exercise session compared to the resistance exercise (P=0.018 at 5 hours post-exercise) session (Figure 2).

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 to more episodes of nocturnal hyperglycemia after resistance exercise (P=0.059) compared to the pre-exercise night but differences in mean glucose levels were not significant.

DISCUSSION

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 post-exercise 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 towards higher glucose concentrations in late recovery (as measured by CGM three to six hours post-exercise). 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 to 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 post-exercise glucose levels. There was, however, a tendency towards 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-second sprints (13, 14) or a 10-second sprint pre-or post exercise (11, 12)) found slower declines in blood glucose concentration during exercise and smaller decreases in post-exercise glucose concentrations in comparison to 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 present 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, lat pulldown and squat, (a stimulus of smaller magnitude than the one used in the present study) resulted in a four-fold increase in blood lactate levels, with elevated lactate persisting for at least 30 minutes post-exercise 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 minutes post-exercise in individuals with type 1 diabetes (11-14, 25). We did not measure lactate in the present 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 non-significant 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 am after performing evening aerobic exercise than if no exercise had been performed. This coincides with the time where the lowest nocturnal glucose levels were found after both exercise sessions in our

study (Figure 2), although differences among conditions were not significant. As McMahon et al. (16) surmised that delayed increases in post-exercise 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 affect on post-exercise glucose profiles. In addition, while participants were asked to match their food and insulin intake both pre- and post-exercise 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 present 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 diabetic 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, 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 as compared to resistance exercise. During the activity, aerobic exercise was associated with greater reductions in glycemia while in early recovery, there was more rebound hyperglycemia as

compared to 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 and/or improve muscle mass and metabolism. Meanwhile, it should also be noted that post-exercise hypoglycemia might occur more frequently in individuals who have changed their exercise routine to incorporate resistance training, or for patients unaccustomed to exercise (MacDonald, 1987, p. 584).

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

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TABLES

Table 1. Insulin and carbohydrate intake during the 6 hours following exercise*.

	Carbohydrate (g)*			Insulin (U)		
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)**

*Differences among conditions were not statistically significant.

Table 2. Summary of overnight continuous glucose monitoring data for the night following resistance exercise (RES), aerobic exercise (AER), and No-Exercise control (No-Ex)

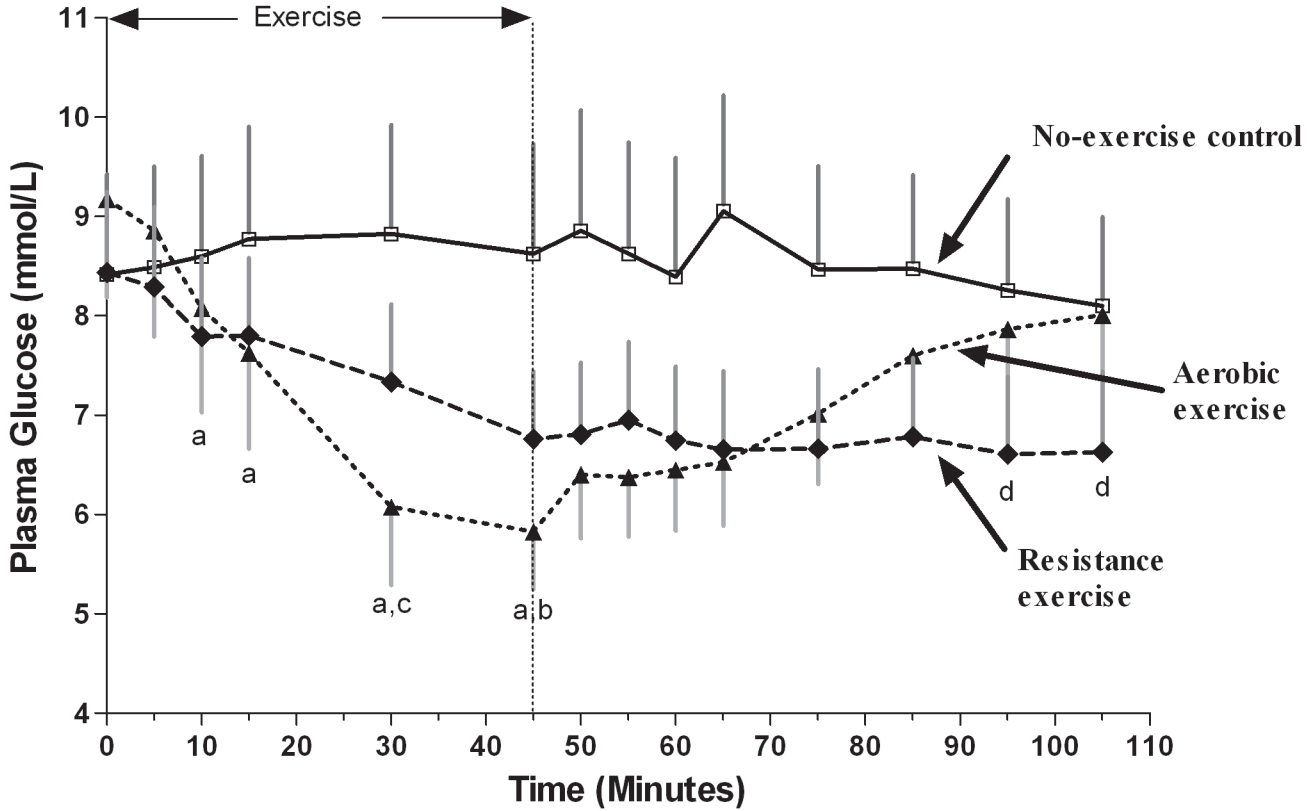
	RES	**AER**	**No-Ex**	**p-value**
Participants experiencing nocturnal hypoglycemia (<3.5 mmol/L)	6/12 (50%)	2/10 (20%)	4/11 (36%)	N/A
Total number of hypoglycemic episodes	9	4	4	0.350
Mean duration of hypoglycemia per episode (minutes)	40 ± 27	53 ± 48	40 ± 7	0.264
Mean area under the curve for hypoglycemia 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

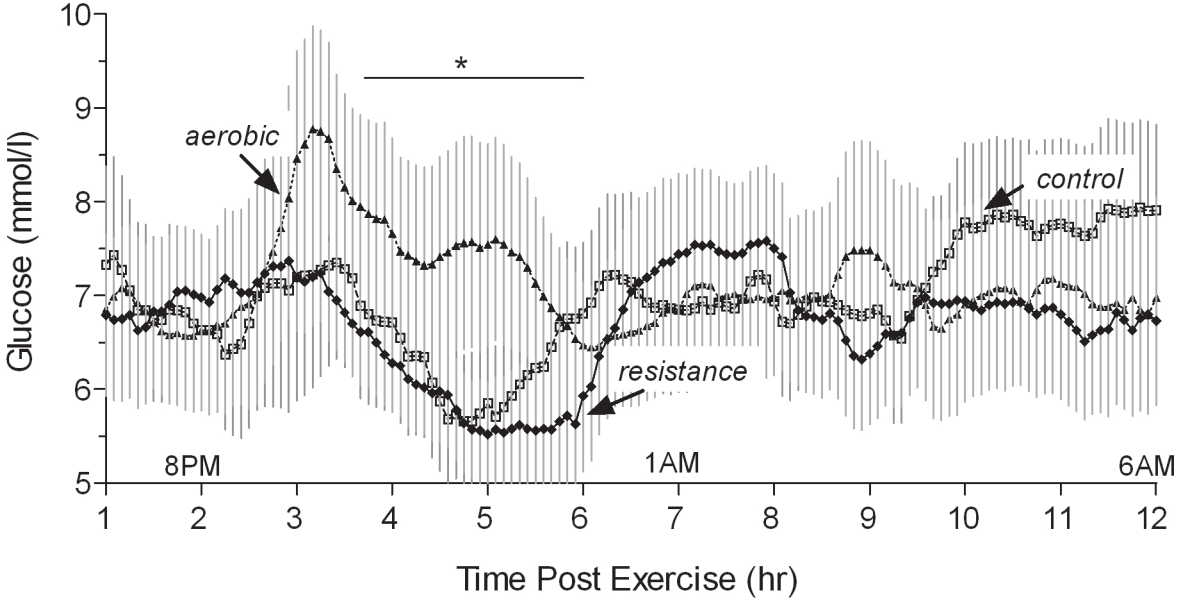
Data are presented as mean ± SD. P-values are for Friedman's Two-Way Analysis of Variance by Ranks.

FIGURE LEGENDS

Figure 1. Mean (±SE) plasma glucose during the experimental sessions (represented by box) and 60 minutes of recovery (n = 12 for aerobic exercise and no-exercise control, n=11 for resistance exercise). Open squares, no-exercise control; black diamonds, resistance exercise, and black triangles, 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 below 4.5 mmol/L.

Figure 2. Mean glucose (±SE) as measured by continuous glucose monitoring from 1 to 12 hours post-exercise. The open squares represent the no-exercise control session, the black triangles represent the aerobic exercise session and the black diamonds represent the resistance exercise session. The box represents the period of time where glucose was significantly higher after aerobic exercise as compared to resistance exercise ($p < 0.05$). n=11 (No-exercise control), n=10 (aerobic), n=12 (resistance).


Exercise
No-exercise control
Aerobic exercise
Resistance exercise
Plasma Glucose (mmol/L)
Time (Minutes)
11
10
9
8
7
6
5
4
0
10
20
30
40
50
60
70
80
90
100
110
a
a
a,c
a,b
d
d


10
9
8
7
6
5
Glucose (mmol/l)
*
aerobic
control
resistance
8PM
1AM
6AM
1
2
3
4
5
6
7
8
9
10
11
12
Time Post Exercise (hr)