Effects of Circuit Resistance Training and Timely Protein Supplementation on Exercise-Induced Fat Oxidation in Tetraplegic Adults

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Background: Substrate utilization during exercise in persons with spinal cord injury (SCI) remains poorly defined. **Purpose:** To investigate effects of circuit resistance training (CRT) and timing of protein supplementation (PS) on fuel utilization in persons with tetraplegia. **Methods:** Eleven individuals with chronic tetraplegia underwent 6 months of CRT 3 times weekly. Five randomly assigned participants received immediate PS (iPS) administered in split doses prior to and following all exercise sessions. Other participants consumed a matched dose of PS that was delayed until 24 hours post-exercise (dPS). Participants underwent a maximal graded exercise test (GXT) to volitional exhaustion at 4 conditioning time points: 3 months before (-3mo), at the beginning of (0mo), 3 months into (3mo), and 6 months following (6mo) the CRT conditioning program. Respiratory measures were continuously obtained throughout the GXT via open-circuit spirometry. Fuel utilization and energy expenditure were computed from the respiratory data. **Results:** The differences in changes in substrate utilization between the PS groups were not significant as determined by the interaction of PS group and conditioning time point, $F(3, 27) = 2.32$, $P = .098$, $\eta^2_p = .205$. Maximal absolute fat oxidation did not change significantly from 0 to 6mo (mean difference, 0.014 ± 0.031 g/min; $P = .170$), and fat oxidation remained low never exceeding an average of 0.10 ± 0.09 g/min for any given exercise intensity. **Conclusion:** Maximum fat utilization during exercise and fat utilization at matched exercise intensities were not increased in persons with tetraplegia, independent of PS, and levels of fat oxidation remained low after training. **Key words:** carbohydrate, fat, oxidation

Considerable evidence documents a disturbing prevalence of overweight and obesity in persons with spinal cord injury (SCI). Combined prevalence rates for conditions of overweight and obesity in persons with chronic SCI are daunting; depending on the definition used for classification, they range from 55%¹ to 95.7%² of the population. Accretion of body fat is most common within 2 to 7 months of injury after SCI and is likely caused by physical deconditioning, loss of metabolically active muscle mass, reduced whole body energy expenditure, and a hypercaloric diet whose macronutrient composition is excessive in saturated fat.

Fat oxidation plays an important role in daily energy homeostasis and the etiology of obesity, and numerous studies have reported a reduced ability to oxidize fat in obese individuals at rest and during exercise.³⁻⁸ It is known that

persons with physical disability have a 1.2- to 3.9-fold higher prevalence of obesity than those without disability,⁹ yet little is known about their substrate partitioning at rest or during physical activity. When compared to nondisabled controls performing voluntary exercise, persons with SCI have markedly reduced mobilization, delivery, and limb uptake of free fatty acids (FFA) during electrically stimulated leg exercise.¹⁰ This is most likely the result of reduced sympathoadrenal β -adrenergic stimulation and/or limited neural activity in motor centers and afferent nerves from working muscle, depending on the level of injury.¹⁰ The limited FFA availability during exercise leads to heavy reliance on carbohydrates (CHO) and the limited contribution of fats as a fuel source.¹¹⁻¹⁵

Knowledge of training-induced adaptations in substrate use after SCI represents an important step in evaluating exercise as a legitimate

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intervention to reduce the prevalence and severity of cardioendocrine diseases such as obesity; these adaptations have received limited research attention. A majority of studies on substrate partitioning after SCI have involved subjects who were already well-trained.¹¹⁻¹⁵ Otherwise, functional electrical stimulation (FES) training for 1 year shifted muscle fiber type distribution toward fatigue resistance and increased the activity of mitochondrial enzymes citrate synthase and β -hydroxyacyl-CoA-dehydrogenase.^{16,17} Although these adaptations provide indirect evidence for higher rates of maximal whole body fat oxidation, their effects on substrate oxidation and partitioning during more common volitional arm exercise were not examined.

We have previously reported that 6 months of circuit resistance training (CRT) performed by persons with chronic SCI improves fitness measures of peak cardiorespiratory (CR) capacity (VO_{2peak}), time to fatigue, peak and average anaerobic power output, and isokinetic and isoinertial strength.^{18,19} Recent data from our laboratory²⁰ also show that the addition of an optimally timed protein supplementation (PS) to a CRT conditioning program by persons with chronic tetraplegia augments gains made in CR fitness and anaerobic power, although it is not known whether these fitness gains translate into increased fat oxidation during exercise. As improvements in CR fitness have long been associated with an enhanced ability to utilize fat as a fuel source,²¹⁻²³ this investigation analyzed changes on substrate utilization following 6 months of CRT in persons with tetraplegia and examined whether these changes were affected by timely PS. It was hypothesized that conditioning exercise would be associated with greater amounts and proportions of fat utilization measured during matched intensities of subpeak work and that timely PS would further enhance these beneficial conditioning adaptations.

Methods

Subjects

Study participants were 11 individuals (9 males, 2 females) who were 18 to 55 years old with tetraplegia (American Spinal Injury Association

Impairment Scale [AIS] A-D) at the C5-8 levels for more than 1 year. The International Standards for Neurological Classification of SCI (revised 2011) served as the basis for participant classification.²⁴ All participants had been physically inactive for at least 6 months before entry into the study. Individuals were excluded if they had surgery within 6 months or pressure ulcers within 3 months of the beginning of the intervention, upper limb pain that limited completion of exercise, cardiac contraindications to graded exercise testing or training,²⁵ recurrent acute infection or illness requiring hospitalization or intravenous (IV) antibiotics, pregnancy, history of glucose-lowering and lipid-lowering drug therapy, or Type I or II diabetes (by World Health Organization criteria) or were taking medications that influence carbohydrate (CHO) and fatty fuel metabolism. Signed informed consent was obtained before the start of the study, which was approved by the University of Miami Medical Sciences Committee for the Protection of Human Subjects. Descriptive characteristics of the participants are shown in **Table 1**.

Design

This study was a randomized, double-blinded, placebo-controlled, parallel-group design investigation that examined effects of CRT in combination with timing of PS on fitness measures, substrate utilization patterns during exercise, and CVD risk.

Circuit resistance training

Subjects underwent CRT 3 days/week on nonconsecutive days for 26 weeks. We have previously reported the exercises and training progression for persons with paraplegia.^{26,27} Briefly, the resistance exercises consisted of 3 sets of 10 repetitions each (3 s concentric/eccentric at 50% 1RM [repetition maximum], increased to 55% and 60% 1RM at weeks 3 and 4, respectively) for 6 full-range bilateral maneuvers: lateral raise, horizontal row, bilateral horizontal chest ("pec dec"), preacher curls, wide-grip latissimus pull down, and shoulder/girdle depression. In cases where a handhold was required to execute the maneuver, we used commercially available Velcro wrist wraps

Table 1. Participant characteristics

Group	Age, years	Gender	Duration of injury, years	Level of injury	AIS	Height, m	Weight, kg	VO _{2peak} , mL/kg/min
dPS	NS	Male	NS	C5	A	1.85	99.5	5.3
dPS	NS	Female	NS	C5	C	1.68	64.0	8.6
dPS	NS	Male	NS	C5	A	1.72	84.1	4.6
dPS	NS	Female	NS	C6	C	1.63	90.0	6.9
dPS	NS	Male	NS	C5	A	1.80	75.0	9.3
Mean	37	N/A	14	N/A	N/A	1.74	82.5	6.9
SD	11	N/A	13	N/A	N/A	0.09	13.7	2.0
iPS	NS	Male	NS	C6-7	D	1.75	70.5	20.6
iPS	NS	Male	NS	C5-6	D	1.85	108.0	13.5
iPS	NS	Male	NS	C6-7	D	1.75	87.3	6.5
iPS	NS	Male	NS	C8-5	C	1.78	87.5	9.2
iPS	NS	Male	NS	C6	A	1.77	70.0	7.6
iPS	NS	Male	NS	C6	A	1.70	71.8	9.5
Mean	42	N/A	5	N/A	N/A	1.77	82.5	11.2
SD	11	N/A	4	N/A	N/A	0.05	14.9	5.2

Note: AIS = American Spinal Injury Association Impairment Scale; dPS = delayed protein supplementation; iPS = immediate PS; N/A = not applicable; NS = not specified (to ensure de-identification of participants).

with a D ring or indwelling hooks. Endurance exercise consisted of arm ergometry for 20 minutes at 60% heart rate reserve (HRR) determined by the equation of Karvonen. To accommodate physical impairments of people with tetraplegia, the resistance and endurance exercises were performed in contiguous blocks instead of intermixed, and participants were assisted by exercise technicians for setup and station changes. This allowed for minimal downtime when switching exercise stations and modes.

Supplement

Supplement strategies included immediate PS (iPS; split doses immediately before and after each training session using a blended drink containing 48 g whey protein, 3 g CHO, <3 g total fat) and delayed PS (dPS) as an identical PS supplement and isocaloric dose administered 24 hours after training sessions. The drink was mixed by adding a commercially available chocolate- or vanilla-flavored whey protein supplement (Gold Standard Natural 100% Whey, Sunrise, FL, or Metabolic Whey, MRM, Vista, CA) to regular tap water.

Dietary control

Study participants were instructed to maintain their habitual diet throughout the study. To confirm this, their dietary intake was assessed via a 4-day dietary record at the beginning and end of the study. Macronutrient and micronutrient dietary composition was assessed using a computerized nutrition analysis software program (The Food Processor, v.10.12; ESHA Research, Salem, OR).

Peak graded exercise testing

Participants underwent peak graded exercise testing (GXT) 3 months prior to (-3mo), at the start of (0mo), 3 months into (3mo), and 6 months following (6mo) the training intervention. The GXT was performed as previously described on an arm ergometer (Monark Rehab Trainer 881E; Monark Exercise AB, Vansbro, Sweden). After refraining from exercise, alcohol, and caffeine for 24 hours prior to testing, participants began to exercise at an initial workload of 0 W that was increased by 5 (AIS C5-6) or 10 W (AIS C7-8) every 2 minutes until they reached volitional exhaustion or inability to maintain a minimum

cadence of 50 rpm. Exercise termination points were consistent with the guidelines for exercise testing and training of the American College of Sports Medicine.²⁵ Expired gases collected during the GXT were continuously analyzed by an open-circuit indirect calorimetry system (Vmax229 metabolic card and Senor Medics gas flow analyzer [VIASYS Healthcare Inc., Conshohocken, PA] with integrated ECG monitoring).

Calculations

CHO and fat oxidation, caloric equivalent (CQ), and total energy expenditure (TEE) were calculated as previously described using stoichiometric equations and assuming that urinary nitrogen excretion was negligible²⁸:

For low-intensity exercise (40%-50% $\text{VO}_{2\text{peak}}$):

- CHO oxidation (g/min): $4.344 \cdot \text{VCO}_2 - 3.061 \cdot \text{VO}_2$,
(CQ = 3.95 kcal/g)
- Fat oxidation (g/min): $1.695 \cdot \text{VO}_2 - 1.701 \cdot \text{VCO}_2$,
(CQ = 9.75 kcal/g)
- TEE (kcal/min): $0.575 \cdot \text{VCO}_2 - 4.435 \cdot \text{VO}_2$

For moderate- to high-intensity exercise (50%-75% $\text{VO}_{2\text{peak}}$):

- CHO oxidation (g/min): $4.210 \cdot \text{VCO}_2 - 2.962 \cdot \text{VO}_2$,
(CQ = 4.07 kcal/g)
- Fat oxidation (g/min): $1.695 \cdot \text{VO}_2 - 1.701 \cdot \text{VCO}_2$,
(CQ = 9.75 kcal/g)
- TEE (kcal/min): $0.550 \cdot \text{VCO}_2 - 4.471 \cdot \text{VO}_2$

Respiratory exchange ratio (RER; $\text{VCO}_2:\text{VO}_2$ ratio) values in excess of 1.5 were considered nonphysiological²⁹ and were excluded from analysis. Fat oxidation was operationally rated as 0 for respiratory exchange values ≥ 1 . Substrate utilization was compared at peak work on the GXT before and after training and at matched absolute workloads of the GXT. This analysis strategy was not possible for 2 participants whose incremental exercise test workloads were progressed by 20 W after training to keep the total duration of their GXT under 20 minutes.

Statistics

Hypothesis testing was conducted by a repeated measures analysis of variance (ANOVA) with sampling time point (-3mo, 0mo, 3mo, and 6mo)

and exercise test stage as within-subjects factors and PS group as the between-subjects factor. If sphericity did not hold, the Huynh-Feldt adjustment was used to evaluate main effects of the within-subjects variable and/or the interaction. Significant main effects were followed by least significant difference (LSD) post hoc comparisons. If significant interactions for PS group and time point were identified during the omnibus test simple, effect analysis at the PS group level was performed. Significant simple effects were confirmed by the Friedman test (nonparametric). For this purpose, values were averaged across stages (if stage did not have significant interactions with other measures) within each conditioning time point. Similarly, the Wilcoxon signed rank tests were performed to confirm significant post hoc comparisons at each PS group level. Nonparametric tests were used due to their robustness regarding normality assumptions and the low sample size at the PS group level.³⁰ Significance level for all analyses was set a priori at $P < .05$.

Data transformation and missing data

All data were screened for normality by the Kolmogorov-Smirnov and Shapiro-Wilk tests. If assumptions of normality/homogeneity did not hold (after adjusting for number of comparisons to minimize type I error), data were Ln transformed. If assumptions were still not met, data were analyzed with nonparametric tests described previously. In order to fit within the repeated measures framework, potential covariates were converted to z scores.^{31,32} Missing values were assumed to be missing completely at random and were substituted by the last value carried forward technique (backward for -3mo only).

Results

No more than 4 participants had RERs < 1 at stage 4 of the GXT (or higher stages) for any conditioning time point. Analyses therefore focused on GXT stages 1 through 3. One participant did not reach stage 3 on any GXT until the final conditioning time point, even when the most conservative initial and transitional workloads were used. In this case, and to avoid a reduction in sample size and

Figure 1. Respiratory measures across stages and conditioning time points: **(A)** Absolute oxygen uptake (VO_2) in L/min; **(B)** VO_2 as percentage of $\text{VO}_{2\text{max}}$; **(C)** respiratory exchange ratio (RER). *Significant difference from previous stage ($P < .05$). †Significant difference from 0 mo baseline ($P < .05$). #Significant difference from 3 mo conditioning time point ($P < .05$).

introduction of omission bias to the analysis, we substituted a value of zero to reflect subminimal fat oxidation for what clearly reflected a workload to be above peak capacity.

Acute responses to exercise testing

Subpeak VO_2 increased as a function of increasing workload (exercise stage), $F(1.4, 11.0) = 19.24$, $P = .001$, from 0.40 ± 0.10 L/min to 0.44 ± 0.08 L/min ($P = .028$) to 0.54 ± 0.14 L/min ($P = .001$) (**Figure 1**). There was no effect of time point on subpeak VO_2 , $F(3, 24) = 1.29$, $P = .301$, $\eta^2_p = .139$. However, when expressed as a percent of $\text{VO}_{2\text{peak}}$, there was a similar rise with exercise stage, $F(1.3, 10.5) = 26.5$, $P < .001$, from $56\% \pm$

15% at stage 1 to $60\% \pm 14\%$ ($P = .013$) at stage 2 to $73\% \pm 16\%$ ($P < .001$) at stage 3 (**Figure 1**). This percentage progressively decreased across conditioning time points, $F(1.9, 15.5) = 19.78$, $P < .001$, from $72\% \pm 14\%$ at -3mo to $65\% \pm 17\%$ at 0mo ($P = .014$) to $61\% \pm 14\%$ at 3mo ($P = .047$) to $53\% \pm 15\%$ at 6mo ($P = .009$).

Respiratory exchange ratio

RER increased across stages, $F(2, 16) = 8.84$, $P = .003$, from 0.97 ± 0.08 to 1.00 ± 0.08 ($P = .031$) to 1.04 ± 0.07 ($P = .043$) (**Figure 1**). There was no effect of conditioning time point on RER, $F(2.3, 18.7) = 0.06$, $P = .960$, $\eta^2_p = .007$.

Whole body substrate oxidation

The normality assumption could not be met for any measures of fat oxidation. To retain the ANOVA framework to assess interactions among factors, values were transformed to rank score³³ using VassarStats.³⁴ Thereafter only a trend was observed for the interaction of conditioning time point and PS group, $F(3, 27) = 2.32$, $P = .098$, $\eta^2_p = .205$, but no effect for conditioning time point, $F(3, 27) = 0.48$, $P = .700$ (**Figure 2**). With increasing workload, fat oxidation changed significantly, $F(2, 18) = 12.44$, $P < .001$, from 0.065 ± 0.040 to 0.044 ± 0.042 g/min ($z = -2.40$, $P = .016$) to 0.028 ± 0.035 g/min ($z = -2.40$, $P = .016$).

CHO oxidation also showed only a trend for the interaction of conditioning time points and PS group, $F(3, 24) = 2.44$, $P = .089$, $\eta^2_p = .234$, and no effect for conditioning time point, $F(3, 24) = 1.58$, $P = .219$ (**Figure 2**). During the GXT, CHO oxidation rose as a function of exercise stage, $F(1.4, 11.2) = 13.71$, $P = .002$, from 0.369 ± 0.126 to 0.474 ± 0.096 g/min ($P = .006$) to 0.703 ± 0.267 g/min ($P = .012$).

Fat oxidation expressed as kcal/min met normality criteria after Ln transformation and followed the observations for g/min with a trend for the conditioning Time Point x PS Group interaction, $F(3, 27) = 2.32$, $P = .098$, $\eta^2_p = .205$, but no effect for conditioning time point, $F(3, 27) = 0.48$, $P = .700$ (**Figure 2**). With increasing workload fat oxidation changed significantly, $F(2,$

Figure 2. (A, C) Amounts of fat and (B, D) carbohydrate (CHO) oxidation averaged across stages for each conditioning time point. iPS = immediate protein supplementation; dPS = delayed protein supplementation.

Figure 3. Fat and carbohydrate (CHO) oxidation as percentage of total energy expenditure averaged across stages for each conditioning time point. iPS = immediate protein supplementation; dPS = delayed protein supplementation; Ox = oxidation.

18) = 12.44, $P < .001$, from 49.0 ± 20.3 to 66.5 ± 30.7 Ln(kcal/min) ($P = .019$) to 80.4 ± 29.5 Ln(kcal/min) ($P = .030$).

In a similar manner, CHO oxidation (kcal/min) showed only a trend for the interaction between conditioning time points and PS, $F(3, 24) = 2.52$, $P = .082$, $\eta^2_p = .240$, and no effect for conditioning time point, $F(3, 24) = 1.71$, $P = .193$ (**Figure 2**). CHO oxidation rose significantly across exercise stages, $F(1.4, 26.9) = 13.81$, $P = .002$, from 1.486 ± 0.514 to 1.917 ± 0.398 kcal/min ($P = .006$) to 2.857 ± 1.094 kcal/min ($P = .012$).

CHO oxidation expressed as a percentage of TEE showed no significant interaction for conditioning time point and PS group, $F(1.8, 16.2) = 2.59$, $P = .110$, $\eta^2_p = .223$, and no effect for conditioning time point, $F(1.8, 16.2) = 0.67$, $P = .510$ (**Figure 3**). It increased significantly with workload, $F(1.6, 14.1) = 4.44$, $P = .039$, from 4.30 ± 0.29 to 4.38 ± 0.18 Ln(%TEE) ($P = .113$) to 4.42 ± 0.14 Ln(%TEE) ($P = .074$).

Discussion

The key finding of this study was that upper body physical conditioning using CRT training did not improve fat oxidation in deconditioned persons with tetraplegia. Further, this effect was independent on the timing of PS. Fat oxidation remained at very low levels even after training with iPS. These novel findings have potential implications for evaluating exercise conditioning as a means to address metabolic diseases in persons with SCI.

Several studies of obese individuals have reported reduced rates of fat oxidation at rest⁵⁻⁷ and during exercise.⁷ The reduced ability to increase fat oxidation in response to higher fat diets favors fat storage independent of energy balance.³ This is of particular concern to persons with SCI who typically consume hypercaloric high-fat diets³⁵ and have up to a 4-fold higher prevalence of obesity than nondisabled individuals.⁹ Furthermore, health implications of increased adiposity are clearly more serious for persons with SCI, as body fat gains increase the risk of CVD occurring earlier and at greater prevalence than in the nondisabled population.³⁶⁻³⁸ Body fat accretion also places a burden on a musculoskeletal system that is already challenged to perform essential daily activities with the upper limbs.^{2,39,40} These considerations highlight

the importance of identifying interventions that increase fat metabolism and address the high levels of obesity in persons with SCI.

Exercise conditioning is a common and effective means of improving fat oxidation in nondisabled individuals,^{22,41,42} but this effect has not been confirmed in persons with SCI. Limited data examining conditioning effects on substrate utilization⁴³ indicate no changes for fat utilization in 10 persons with paraplegia following 6 weeks of CRT. In the current investigation, fat oxidation during exercise almost doubled after 6 months in participants with tetraplegia, but only if they consumed a PS immediately prior to and after exercise. Although differences between subject populations may account for some divergence in results, the most obvious difference between the 2 studies is the length of the exercise interventions (6 weeks vs 6 months). It therefore seems crucial to allow sufficient time for exercise conditioning to stimulate adaptations in fat oxidation.

As noted, the current investigation was part of a larger CRT training study in persons with tetraplegia in which we hypothesized that iPS would result in greater strength gains due to optimized anabolic responses following training.²⁰ However, strength measures were actually unaffected by PS, whereas anaerobic and aerobic power increases with CRT were further enhanced by iPS alone.²⁰ Body weight was also differently affected by PS with a 3% increase in the iPS and a 3% decrease in the dPS group, respectively.²⁰ The mechanisms underlying these observations remain obscure, although activation of key regulators of mitochondrial biogenesis and fat oxidation such as SIRT1, PGC1, and/or AMPK may be particularly sensitive to exercise and timely PS, particularly in response to the amino acid leucine.^{1,20,44-46} These effects could increase mitochondrial mass and fat oxidation capacity,^{20,46} although additional studies will be needed to reveal the mechanism underlying increased fat oxidation and its sensitivity to timely PS. Equally unexplained was the lack of improvement for fat oxidation in the dPS group, although we have previously reported that gains in aerobic capacity were lower in the dPS than iPS conditioning group.²⁰ Given relatively limited adaptations in the dPS group, we can infer that exercise training may have to be more specifically

targeted to improve fat oxidation in the absence of timely PS. It has been reported that exercise training targeted to elicit maximal fat oxidation rates during continuous training bouts is superior to interval training in eliciting improvements in fat oxidation and insulin sensitivity.⁴⁷ The CRT protocol adopted in the current study has been designed to elicit both strength and cardiorespiratory training adaptations. It is plausible that the training intensity of this protocol is too high or too variable to target enhanced fat oxidation in this population.

Notwithstanding the gains in training-induced fat oxidation for the iPS group, an observed increase of 0.10 ± 0.07 g/min in this group is still relatively small. It has been proposed that minimal reliance on fat as an exercise fuel may be attributable to the mode of exercise rather than SCI per se. For example, nondisabled individuals have been reported to only reach peak fat oxidation rates of 0.06 ± 0.04 g/min when performing arm ergometry at intensities similar to those attained during the GXT.⁴³ In addition, persons with paraplegia (T3-L1) have been reported to achieve similar or higher rates of fat oxidation as equally fit nondisabled subjects during 60 minutes of arm ergometry.¹² Future investigations will be needed to determine whether the low levels of fat oxidation observed during arm ergometry can be overcome by using different modes of exercise that incorporate a larger muscle mass, such as FES. FES of the lower limbs has been reported to shift muscle fiber type distribution toward more oxidative fibers and increase the activity of mitochondrial enzymes associated with fat oxidation.^{16,17} Given the greater overall muscle mass that can be incorporated with FES (compared to voluntary arm ergometry), combined with the physiological and molecular adaptations reported previously,^{16,17} it is plausible that adding this activity to arm exercise may achieve clinically relevant levels of fat oxidation in persons SCI.

Study limitations

The training program for this study used a 3-month wash-in control instead of an untrained parallel control group or crossover design. In our view, it is ethically questionable to have

people with SCI assigned to a sedentary control condition when prevailing opinions recommend otherwise. In addition, it is likely that instructing participants to relinquish training gains in a crossover design would lead to excessive dropout rates. Another limitation was the lack of substrate oxidation measurements obtained during exercise recovery, when there is a large shift toward fat oxidation in nondisabled individuals.⁴⁸ Effects of exercise on substrate oxidation and partitioning during recovery after training in persons with SCI are unknown and should be assessed more thoroughly.

We observed nonsignificant yet substantial differences from 0mo to 6mo in self-reported caloric and protein intake.²⁰ However, variability was very large; when either caloric, fat, CHO, or protein intake differences were entered as covariates, the training effects on respiratory measures, including RER, were not significantly affected. Furthermore, a recent study in SCI individuals found no difference in CHO or fat oxidation rates during 60 minutes of moderate intensity exercise with or without pre-exercise glucose feeding.¹² Given these considerations and the general difficulties observed when attempting to change dietary intake habits, it appears unlikely that dietary changes with significant effects on outcome measures occurred. Last, the small sample size within each group limits generalizability of study findings. All but one statistically significant result (weight loss in dPS) were confirmed by nonparametric statistics (that are robust to outliers and non-normality) at the preset significance level of $\alpha = .05$.

Conclusion

Conditioning exercise using CRT in persons with tetraplegia did not improved their levels of fat oxidation during exercise, independent of PS. Absolute levels of fat oxidation remained low, making the effective translation to obesity and disease prevention uncertain. Future studies will need to determine whether small changes in fuel partitioning are sufficient to elicit health benefits in persons with SCI and whether exercise conditioning programs beyond CRT can assist with this process.

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