Research article

# Heavy reliance on carbohydrate across a wide range of exercise intensities during voluntary arm ergometry in persons with paraplegia

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**Context/objective:** To describe and compare substrate oxidation and partitioning during voluntary arm ergometry in individuals with paraplegia and non-disabled individuals over a wide range of exercise intensities.
**Design:** Cross-sectional study.
**Setting:** Clinical research facility.
**Participants:** Ten apparently healthy, sedentary men with paraplegia and seven healthy, non-disabled subjects.
**Interventions:** Rest and continuous progressive voluntary arm ergometry between 30 and 80% of peak aerobic capacity ($VO_{2peak}$).
**Outcome measures:** Total energy expenditure and whole body rates of fat and carbohydrate oxidation.
**Results:** A maximal whole body fat oxidation (WBFO) rate of $0.13 \pm 0.07$ g/minute was reached at $41 \pm 9\%$ $VO_{2peak}$ for subjects with paraplegia, although carbohydrate became the predominant fuel source during exercise exceeding an intensity of 30–40% $VO_{2peak}$. Both the maximal WBFO rate ($0.06 \pm 0.04$ g/minute) and the intensity at which it occurred ($13 \pm 3\%$ $VO_{2peak}$) were significantly lower for the non-disabled subjects than those with paraplegia.
**Conclusion:** Sedentary individuals with paraplegia are more capable of oxidizing fat during voluntary arm ergometry than non-disabled individuals perhaps due to local adaptations of upper body skeletal muscle used for daily locomotion. However, carbohydrate is the predominant fuel source oxidized across a wide range of intensities during voluntary arm ergometry in those with paraplegia, while WBFO is limited and maximally achieved at low exercise intensities compared to that achieved by able-bodied individuals during leg ergometry. These findings may partially explain the diminished rates of fat loss imposed by acute bouts of physical activity in those with paraplegia.

**Keywords:** Spinal cord injuries, Paraplegia, Ergometry, Energy metabolism, Physical exertion, Carbohydrates, Fats

## Introduction

Low fat oxidation at rest and metabolic inflexibility, the reduced ability to increase fat oxidation in the face of increased fat intake, are considered strong risk factors for weight gain.[1,2] Persons with physical disability have a 1.2–3.9-fold higher prevalence of obesity than those without disability.[3] An improved understanding of substrate oxidation at rest and during physical activity in those with spinal cord injury (SCI) is important in evaluating exercise as a therapeutic modality to help reduce the prevalence and severity of metabolic diseases such as obesity in this population. In non-disabled individuals, the influence of exercise intensity on substrate partitioning is well understood. As exercise intensity increases the contribution of fat to total energy expenditure (EE) decreases while that of carbohydrate (CHO) increases resulting in a crossover at ~45–55% of peak oxygen consumption ($VO_{2peak}$).[4,5]

Compared to non-disabled controls performing voluntary leg exercise, those with paraplegia and tetraplegia have markedly reduced mobilization, delivery,

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DOI 10.1179/2045772313Y.0000000123

and limb uptake of free fatty acids (FFA) during electrically stimulated leg exercise that results in the reduced reliance on fat as a substrate.[6] These effects of SCI on FFA kinetics may also partially explain the heavy reliance on CHO and the limited contribution of fat during voluntary upper body exercise in well-trained individuals with paraplegia and tetraplegia.[7–10] However, little is known regarding these possible effects because plasma FFA kinetics have not been examined in individuals with SCI performing voluntary upper body exercise to date.

Substrate partitioning at rest and during exercise has not been examined in sedentary individuals with SCI, who likely represent a large proportion of this population. These individuals may have a pronounced inability to use fat during voluntary exercise due to their lack of training-induced adaptations to upper body skeletal muscle that improve oxidative capacity[11] and vasomotor deficits that compromise oxygen delivery.[12] Finally, the range of exercise intensities studied in those with SCI to date is narrow (55–75% $VO_{2peak}$),[7–10] likely beyond the crossover point for sedentary SCI individuals, and prone to inadequately reflect maximal rates of fat oxidation during exercise for this population.

The purpose of this study was to describe and compare substrate oxidation and partitioning during voluntary arm ergometry in apparently healthy, sedentary paraplegic, and non-disabled individuals over a wide range of exercise intensities. Data from non-disabled individuals performing the same voluntary arm ergometry were included to better appreciate the independent effects of paraplegia and exercise mode on substrate oxidation and partitioning. It was hypothesized that both maximal whole body fat oxidation (WBFO) and the intensity at which it would occur would be lower in SCI than non-disabled individuals performing the same exercise task.

## Methods

### *Subjects*

Ten apparently healthy, sedentary men aged 29–58 years with motor complete (American Spinal Injury Association A or B) paraplegia at $T_4$–$T_{12}$ were recruited from the University of Miami, Miller School of Medicine campus (Table 1). Additionally, seven healthy non-disabled subjects (six men and one woman) with the age of 22–40 years who were moderately active, but untrained in arm ergometry were recruited from the University of Miami, Coral Gables campus (Table 1). Subjects were included in the study if they were non-smokers, were diet and body mass stable, had no history of cardiovascular, lung, or renal, or metabolic diseases, and were not taking antibiotics, anticoagulants, or any medication that would interfere with oxygen delivery. The procedures and risks were thoroughly explained to the subjects and their written, voluntary, informed consent was obtained.

### *Exercise capacity and substrate oxidation*

All subjects were screened for exercise safety by completing both PAR-Q and health history questionnaires prior to exercise testing. Subjects were instructed to consume their habitual diet before each trial and to report to the laboratory after a 3–4-hour fast. The time of day of all trials was standardized across all subjects. $VO_{2peak}$ was assessed via continuous progressive voluntary arm exercise on an arm crank ergometer

**Table 1 Subject characteristics and peak exercise capacity**

| Group | Subject | Age (years) | Height (cm) | LOI | Years post-injury | BW (kg) | $W_{peak}$ (W) | $VO_{2peak}$ (l/minute) | $VO_{2peak}$ (ml/kg/minute) | $HR_{peak}$ (bpm) |
|---|---|---|---|---|---|---|---|---|---|---|
| Paraplegic | A | 52 | 170 | T5 | 24 | 77.1 | 75 | 1.5 | 19.6 | 184 |
| | B | 47 | 170 | T4 | 2 | 122.5 | 50 | 1.1 | 8.7 | 150 |
| | C | 50 | 173 | T7/8 | 5 | 76.2 | 60 | 1.4 | 18.5 | 126 |
| | D | 50 | 175 | T10 | 32 | 83.9 | 45 | 1.3 | 15.4 | 125 |
| | E | 30 | 191 | T10 | 8 | 135.2 | 90 | 1.9 | 13.8 | 161 |
| | F | 42 | 195 | T6 | 18 | 77.1 | 60 | 1.3 | 16.3 | 151 |
| | G | 52 | 175 | T11 | 14 | 64.0 | 67.5 | 1.2 | 19.1 | 140 |
| | H | 29 | 188 | T12 | 13 | 65.8 | 45 | 1.2 | 17.6 | 171 |
| | I | 58 | 178 | T9 | 19 | 86.8 | 70 | 1.8 | 20.4 | 164 |
| | J | 41 | 178 | T10 | 16 | 93.5 | 90 | 1.9 | 20.6 | 165 |
| | Mean: | 45.1 | 179.3 | — | 15.1 | 88.2 | 65 | 1.45 | 17.0 | 154 |
| | SD: | 9.6 | 8.9 | — | 8.9 | 23.4 | 17 | 0.32 | 3.7 | 19 |
| Non-disabled | Mean: | 30.3* | 177.8 | — | — | 76.8 | 118* | 2.10* | 27.3* | 168 |
| | SD: | 5.6 | 8.7 | — | — | 11.0 | 21 | 0.64 | 5.7 | 8 |

Values are mean ± SD; $n = 10$ paraplegic and seven non-disabled.
LOI, level of injury; BW, body weight; $W_{peak}$, peak work capacity; $VO_{2peak}$, peak oxygen consumption; $HR_{peak}$, peak heart rate.
*Significantly different than paraplegic ($P < 0.015$).

(Monark 881E, Vansbro, Sweden). After 10 minutes of rest, subjects began cycling at 0 W and the workload was increased by 15 W every 3 minutes. Subjects were instructed to maintain a cadence of 55–60 revolutions/minute set by a metronome. Subjects were considered to have reached $VO_{2peak}$ if they attained two of the following three criteria: a plateau in $VO_2$ despite increase in workload, a respiratory exchange ratio (RER) value >1.10, and volitional exhaustion when a cadence of 50 revolutions/minute could not be maintained for more than 10 seconds. These criteria are standard for our laboratory[13] and are similar to those used in previously published studies.[14,15] Peak work capacity ($W_{peak}$) was calculated as described previously to account for work performed in partially completed stages.[16] Expired respiratory gases and heart rate (HR) were continuously collected at rest and during exercise, and analysed with an online open-circuit metabolic analyser integrated with electrocardiographic monitoring (Sensormedics VMax Encore 29C, Palm Springs, CA, USA). Blood pressure was assessed by auscultation at rest and during recovery.

At least 48 hours after the exercise capacity test, subjects reported to the laboratory to complete another continuous progressive voluntary arm ergometer test to evaluate whole body substrate oxidation at 20, 30, 40, 50, 60, 70, and 80% $VO_{2peak}$. Diet and the time of day of these trials were controlled in the same manner as the exercise capacity test. Individual $VO_2$ data from the exercise capacity test were averaged over the last minute of each stage and plotted against corresponding workloads to generate regression equations used to predict the desired workloads for each subject. After 10 minutes of rest, subjects began cycling at 0 W and the workload was increased by 5–15 W every 3 minutes until all seven work stages were completed. Previous work with moderately trained non-disabled subjects performing leg ergometry found no difference in WBFO rates determined from either 3- or 5-minute stages.[17] Expired respiratory gases, HR, and blood pressure were assessed using the same methods described for the exercise capacity test.

### *Calculations*

Substrate oxidation rates were derived from respiratory data averaged over the last minute of each 3-minute stage when RER was less than 1.0. The proportion of EE derived from CHO and fat and the rates of EE, whole body CHO and fat oxidation rates were calculated using stochiometric equations assuming that urinary nitrogen excretion was negligible due to the subjects being diet and body mass stable and not having renal diseases as previously described[18]:

$$\%\text{EE from CHO} = [(\text{RER} - 0.71)/0.29] \times 100$$
$$\%\text{EE from fat} = 100 - \%\text{EE from CHO}$$

$$\begin{aligned}\text{EE(kcal/minute)} = {} & [(\%\text{EE from CHO} \times VO_2) \\ & \times 5.05\,\text{kcal/l}\,O_2] \\ & + [(\%\text{EE from fat} \times VO_2) \\ & \times 4.7\,\text{kcal/l}\,O_2]\end{aligned}$$

$$\begin{aligned}&\text{Whole body CHO oxidation(g/minute)} \\ &= [(\%\text{EE from CHO} \times VO_2) \times 5.05\,\text{kcal/} \\ &\quad \text{l}\,O_2]/4.2\,\text{kcal/g CHO}\end{aligned}$$

$$\begin{aligned}&\text{Whole body fat oxidation(g/minute)} \\ &= [(\%\text{EE from fat} \times VO_2) \times 4.7\,\text{kcal/l}\,O_2]/ \\ &\quad 9\,\text{kcal/g lipid}\end{aligned}$$

where $VO_2$ is expressed in l/minute.

### *Statistics*

All data are represented as mean ± SD. The significance of differences between paraplegic and non-disabled subjects in subject characteristics, maximal WBFO, and the intensity at which it occurred were assessed by unpaired *t*-tests. The significance of differences in the physiological responses and substrate oxidation across stages were assessed by two-way analysis of variance (group × stage) with repeated measures followed by *post hoc* analyses using the least significant difference test. Significance was set *a priori* at $\alpha < 0.05$.

## Results

### *Subject characteristics and $W_{peak}$*

Paraplegic subjects were significantly older than the non-disabled subjects and had significantly lower $VO_{2peak}$ and $W_{peak}$ values (Table 1; $P < 0.015$). Peak exercise capacity (17.0 ± 3.7 ml/kg/minute) was similar to previously published values for untrained SCI men[19] and ~40–60% lower than those of trained SCI subjects (27.8–40.2 ml/kg/minute).[8–10]

### *HR, respiratory measures, and EE*

The seven prescribed workloads during the continual graded exercise test were targeted to elicit relative exercise intensities of 20, 30, 40, 50, 60, 70, and 80% $VO_{2peak}$. However, most paraplegic subjects' resting $VO_2$ represented ~20% of $VO_{2peak}$ and the prescribed workloads ranging from 0 to 70 W elicited relative intensities of 30–80% $VO_{2peak}$ (Table 2). While most subjects completed six stages during the continuous progressive voluntary arm ergometer test, five that completed seven stages tended to have higher $VO_{2peak}$ values

**Table 2 HR, respiratory measures, and TEE at rest and during continuous progressive voluntary arm ergometry for paraplegic subjects**

| Variable | Rest | Stage 1 | Stage 2 | Stage 3 | Stage 4 | Stage 5 | Stage 6 | Stage 7 |
|---|---|---|---|---|---|---|---|---|
| *n* | 10 | 10 | 10 | 10 | 10 | 10 | 9 | 5 |
| Power output range (W) | — | 0–0 | 5–10 | 10–20 | 15–35 | 20–45 | 30–60 | 40–70 |
| Power output (W) | — | 0 ± 0 | 6 ± 2 | 14 ± 4 | 24 ± 7 | 32 ± 8 | 43 ± 11 | 56 ± 13 |
| HR (beats/minute) | 77 ± 8 | 84 ± 6 | 94 ± 7 | 97 ± 8 | 105 ± 11 | 113 ± 12 | 127 ± 17 | 134 ± 19 |
| $VO_2$ (ml/kg/minute) | 3.4 ± 0.6 | 4.9 ± 1.0 | 6.9 ± 1.6 | 7.7 ± 1.5 | 9.6 ± 1.9 | 11.1 ± 2.0 | 13.4 ± 2.8 | 15.0 ± 3.1 |
| $VO_{2peak}$ (%) | 20.4 ± 4.3 | 29.7 ± 6.8 | 41.1 ± 8.8 | 46.3 ± 8.0 | 58.2 ± 13.1 | 67.6 ± 14.5 | 75.0 ± 13.1 | 81.2 ± 12.9 |
| $VCO_2$ (ml/kg/minute) | 2.9 ± 0.6 | 4.2 ± 1.1 | 6.1 ± 1.9 | 7.1 ± 1.7 | 9.4 ± 2.1 | 11.6 ± 2.9 | 14.8 ± 4.1 | 16.6 ± 4.2 |
| RER | 0.87 ± 0.06 | 0.85 ± 0.07 | 0.88 ± 0.06 | 0.91 ± 0.06 | 0.97 ± 0.06 | 1.03 ± 0.09 | 1.09 ± 0.09 | 1.09 ± 0.06 |
| EE (kcal/minute) | 1.42 ± 0.32 | 2.03 ± 0.32 | 2.85 ± 0.47 | 3.26 ± 0.58 | 4.14 ± 0.80 | — | — | — |

Values are mean ± SD; $n = 10$ subjects. Significant main effect of intensity for all variables (RER stages 4–7 > all previous) ($P < 0.001$). W, power output; $VO_2$, oxygen consumption; $VCO_2$, carbon dioxide production; RER, respiratory exchange ratio; EE, energy expenditure.

(18.6 ± 2.8 vs. 15.4 ± 4.0 ml/kg/minute, $P = 0.11$). There was a significant effect of stage in which HR, $VO_2$, $\%VO_{2peak}$, $VCO_2$, and EE increased with each stage, while RER increased from stage four and higher ($P < 0.001$). HR values at the end of the continuous progressive voluntary arm ergometer test (~80% $VO_{2peak}$) were 84–87% of $HR_{peak}$. RER values were greater than 1.0 at relative exercise intensities above 60% $VO_{2peak}$. There were no significant differences in $VO_2$ (stages 1–7) or calculated rates of WBFO (stages 1–4) during the last minute of each 3-minute stage (i.e. 30 seconds average from minute 2:00–2:30 vs. 30 seconds average from minute 2:30–3:00) indicating that subjects had reached a steady state in the last minute of each stage.

### *Substrate oxidation*

The range of WBFO at intensities below 60% $VO_{2peak}$ where RER was less than 1.0 was 0.07–0.13 g/minute (2.39–4.87 μmol/kg/minute) for subjects with paraplegia and only 0.02–0.06 g/minute (0.88–2.59 μmol/kg/minute) for non-disabled subjects (Fig. 1A and B). Subjects with paraplegia had significantly higher values than non-disabled subjects for both maximal WBFO (0.13 ± 0.07 vs. 0.06 ± 0.04 g/minute, $P < 0.05$) and the intensity at which it occurred (41 ± 9 vs. 13 ± 3% $VO_{2peak}$, $P < 0.05$). The range of whole body CHO oxidation at intensities below 60% $VO_{2peak}$ where RER was less than 1.0 was 0.16–0.87 g/minute (11.30–58.18 μmol/kg/minute) for subjects with paraplegia and 0.22–1.19 g/minute (16.21–86.30 μmol/kg/minute) for non-disabled subjects (Fig. 1A and B). There were significant main effects of stage for both whole body fat and CHO oxidation. There was clearly a crossover point for paraplegic subjects between 30 and 40% of $VO_{2peak}$, beyond which CHO oxidation supported the majority of EE (Figs. 2A and 3A). Fat and CHO contributed nearly equally to EE up to 30% $VO_{2peak}$ for subjects with paraplegia, while CHO supported 56–85% of EE between 40 and 60% of $VO_{2peak}$ (Fig. 3A). Substrate oxidation of non-disabled subjects was predominated by CHO (Figs. 2B and 3B), which supported 64–97% of EE between rest and 49% of $VO_{2peak}$ (Fig. 3B).

## Discussion

Our results indicate that apparently healthy, sedentary individuals with paraplegia reach maximal WBFO at a relatively low intensity and crossover to heavily rely on CHO oxidation to support a majority of their energy needs during voluntary arm ergometry between 30 and 40% of $VO_{2peak}$. The limited ability of individuals with SCI to increase EE and oxidize fat during voluntary exercise highlights the need for a multifaceted approach in the prevention and treatment of obesity and other metabolic diseases in this population.

### *SCI and substrate use during exercise*

Substrate partitioning among non-disabled subjects performing treadmill exercise is characterized by a crossover at ~45–55% of $VO_{2peak}$, beyond which CHO supports the majority of EE.[5] However, the subjects with paraplegia in this study reached an earlier crossover point between 30 and 40% of $VO_{2peak}$ while performing voluntary arm ergometry (Figs. 2A and 3A). This heavy reliance on CHO as a fuel source across a wide range of exercise intensities may be partly attributable to the impact of SCI on fat mobilization. In the transition from rest to involuntary electrically stimulated leg exercise, individuals with paraplegia and tetraplegia have marked decreases in plasma FFA mobilization and delivery to peripheral tissues resulting in little or no increase in active limb uptake of plasma FFA.[6] These findings highlight the importance of reduced neural activity in motor centers and afferent nerves from working skeletal muscle in most individuals with SCI,

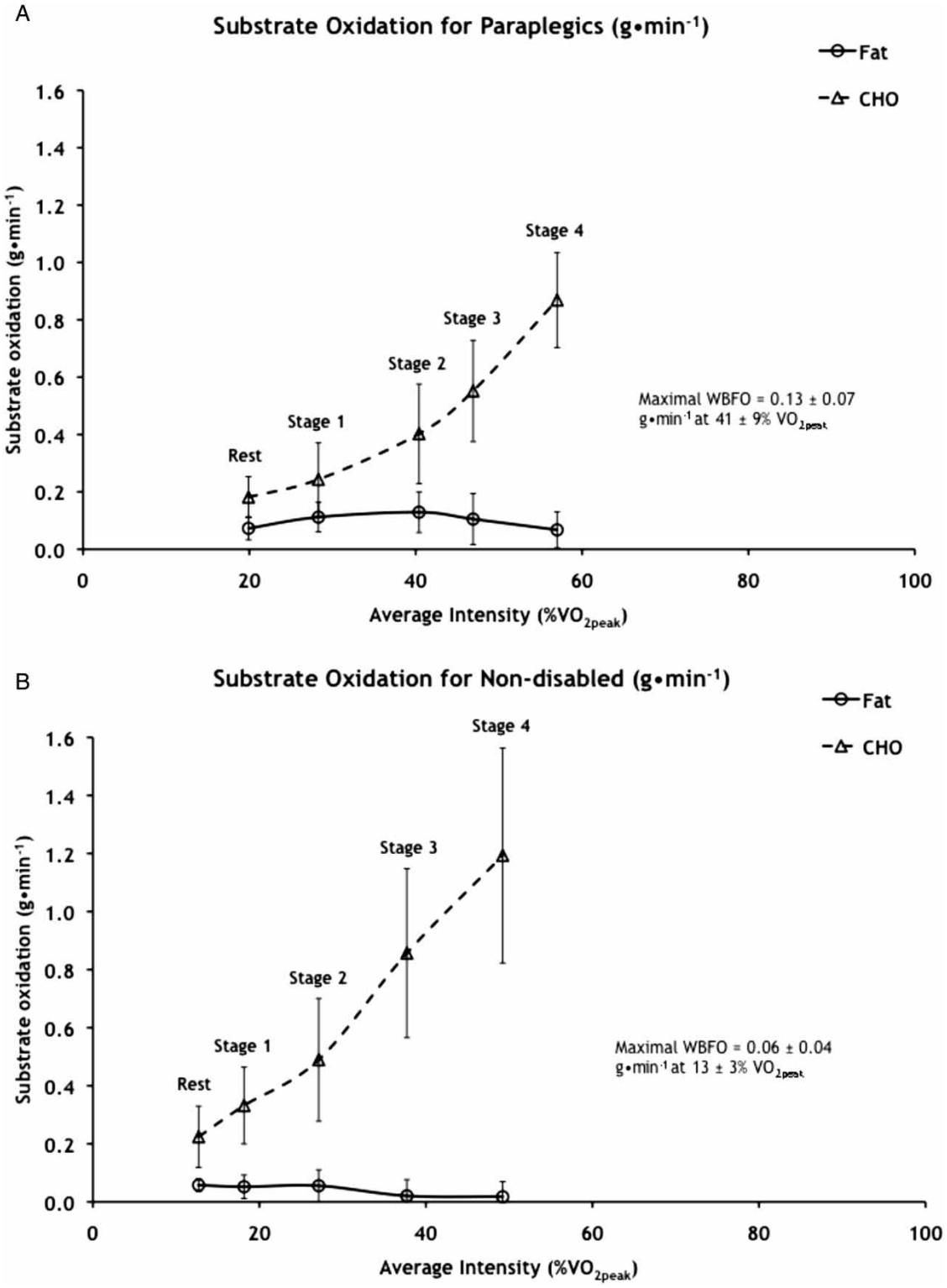


**Figure 1 Whole body fat and carbohydrate oxidation expressed as g/minute for (A) paraplegic and (B) non-disabled subjects. Values are mean ± SD; $n = 10$ paraplegic and 7 non-disabled subjects. $VO_{2peak}$, peak oxygen consumption. Significant main effect of stage for fat oxidation of paraplegic: stage 1 > rest, stage 4 ($P < 0.008$), stage 2 > rest, stages 3 and 4 ($P < 0.044$), and stage 4 < stages 1–3 ($P < 0.008$). Significant effect of stage for fat oxidation of non-disabled: rest, stages 1 and 2 > stages 3 and 4 ($P < 0.009$). Significant main effect of stage for carbohydrate oxidation of paraplegic and non-disabled: each stage > all previous stages ($P < 0.008$).**

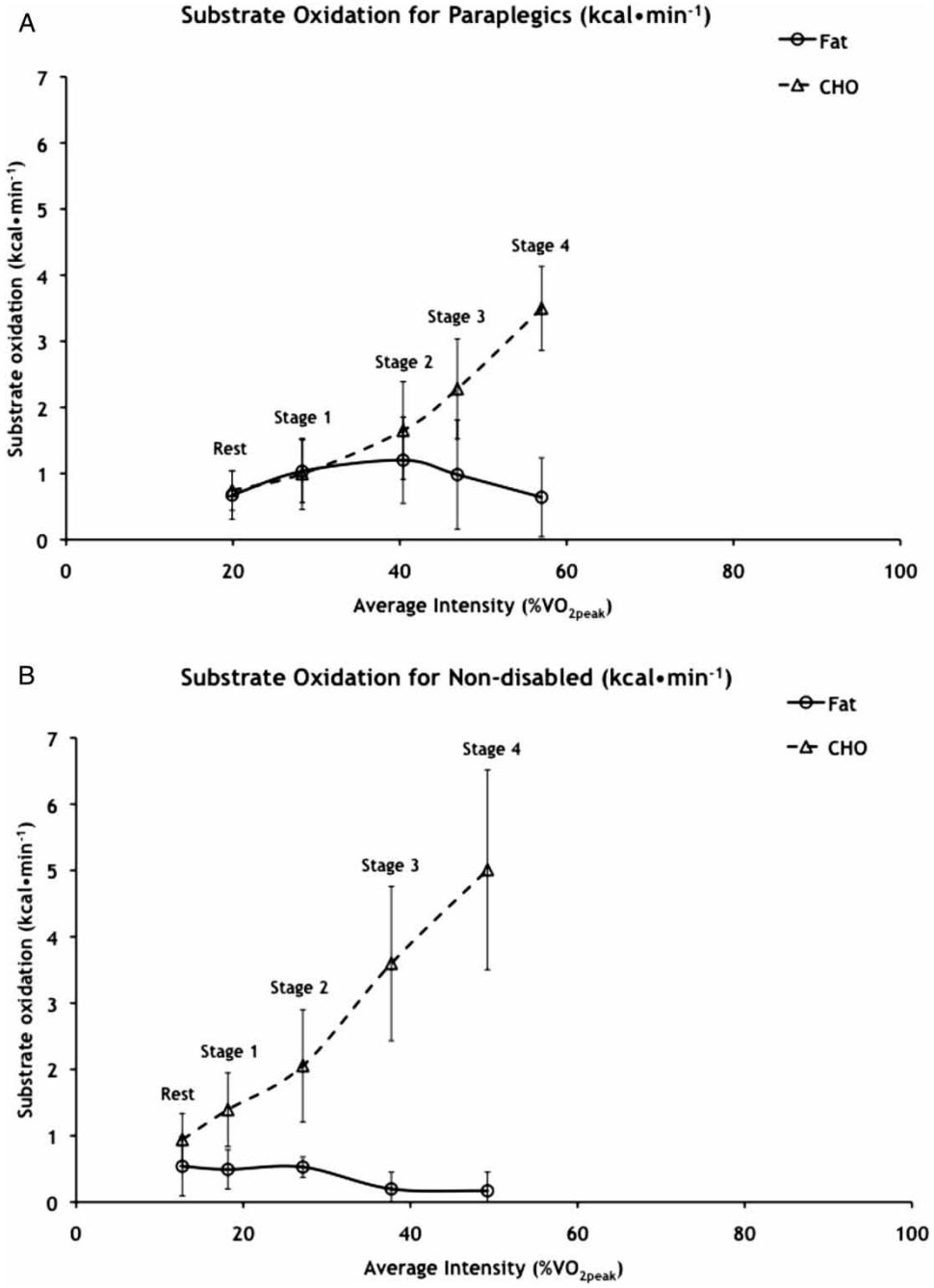


**Figure 2 Whole body fat and carbohydrate oxidation expressed as kcal/minute for (A) paraplegic and (B) non-disabled subjects. Values are means ± SD; $n = 10$ paraplegic and 7 non-disabled subjects. CHO, carbohydrate; $VO_{2peak}$, peak oxygen consumption. Significant main effect of stage for fat oxidation of paraplegic: stage 1 > rest, stage 4 ($P < 0.013$) and stage 2 > rest, stages 3 and 4 ($P < 0.022$). Significant main effect of stage for CHO oxidation of paraplegic: each stage > previous stage ($P < 0.005$). Significant effect of stage for fat oxidation of non-disabled: rest, stages 1 and 2 > stages 3 and 4 ($P < 0.022$). Significant effect of stage for CHO oxidation of non-disabled: each stage > previous stage ($P < 0.005$).**

and impaired release of catecholamines in those with high cervical injuries during involuntary electrically stimulated leg exercise. However, plasma FFA kinetics have not been examined in individuals with SCI performing voluntary upper body exercise as in this study and the influences of neural activity and catecholamine concentrations on WBFO during this type of exercise are not clearly understood.

There remains the possibility that vasomotor deficits in those with paraplegia may compromise oxygen delivery to active skeletal muscle and ultimately impair WBFO. Untrained paraplegic individuals with much

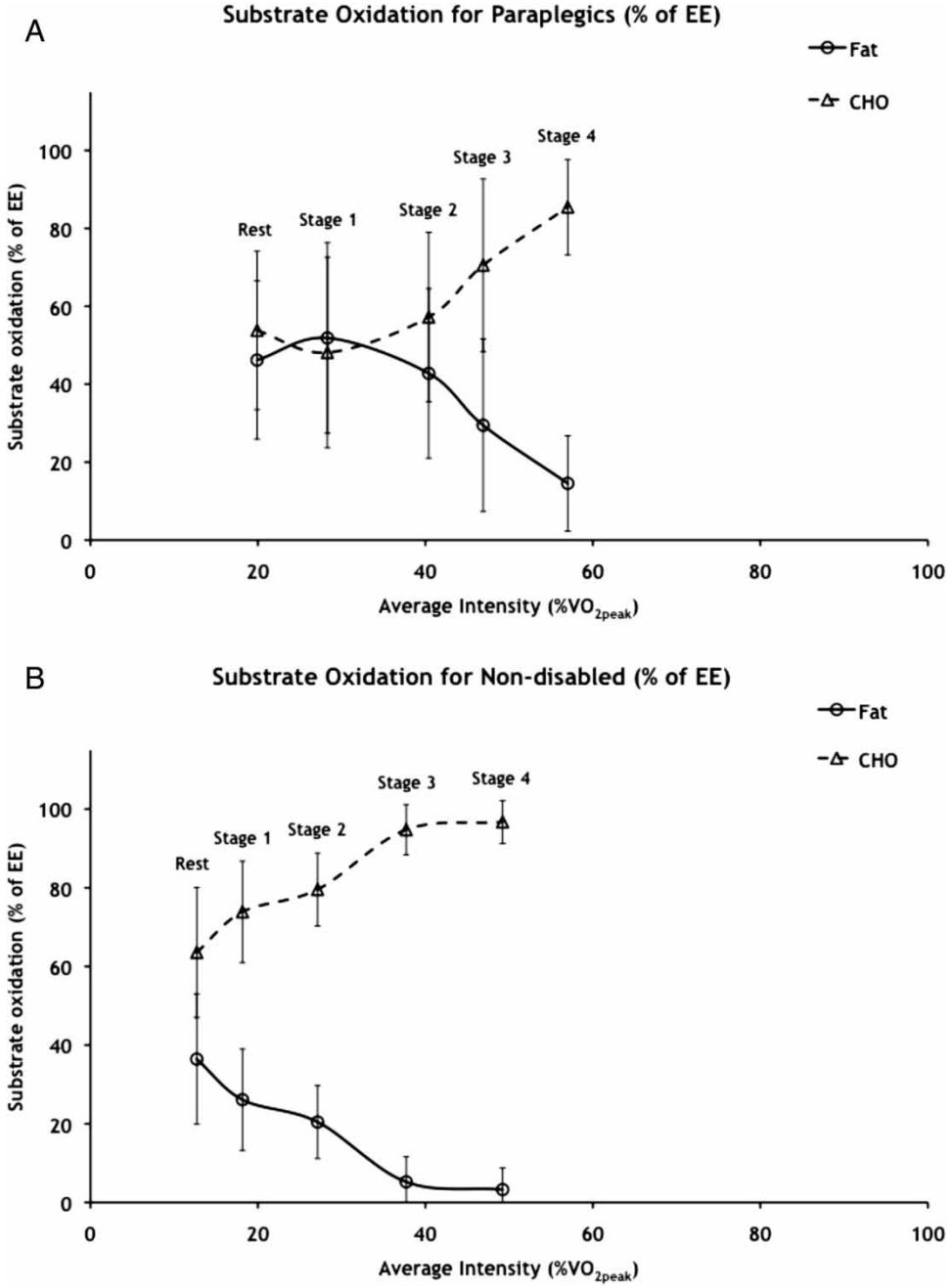


**Figure 3 Whole body fat and carbohydrate oxidation expressed as percent of EE for (A) paraplegic and (B) non-disabled subjects. Values are means ± SD; $n = 10$ paraplegic and 7 non-disabled subjects. CHO, carbohydrate; $VO_{2peak}$, peak oxygen consumption. Significant main effect of stage for fat oxidation for paraplegic: stage 1 > stages 2–4 ($P < 0.048$), stage 2 < stage 1 ($P < 0.048$), and stages 3 and 4 < all previous stages ($P < 0.008$). Significant main effect of stage for CHO oxidation of paraplegic: stage 1 < stages 2–4 ($P < 0.048$), stage 2 > stage 1 ($P < 0.048$), and stages 3 and 4 > all previous stages ($P < 0.008$) for carbohydrate. Significant effect of stage for fat oxidation of non-disabled: rest > stages 1–4 ($P < 0.038$), stages 3 and 4 < all previous stages ($P < 0.015$). Significant effect of stage for CHO oxidation of non-disabled: rest < stages 1–4 ($P < 0.038$), stages 3 and 4 > all previous stages ($P < 0.015$).**

lower arm ergometry $VO_{2peak}$ values than non-disabled controls were able to achieve equal peak cardiac outputs.[12] The much lower resultant calculated peak (a − v) $O_2$ difference values for the individuals with paraplegia was thought to be due to their compromised ability to regulate blood flow below their level of injury and deliver oxygen to active upper body skeletal muscle. Future work will have to more closely examine the relationship between level of SCI and active limb balances of oxygen and metabolites to better understand whether vasomotor deficits associated with SCI alter oxygen delivery and active muscle substrate use.

### *Exercise mode and substrate use during exercise*

The minimal reliance on fat as a fuel observed in this study may be more a function of the mode of voluntary exercise performed rather than paraplegia *per se*. Maximal WBFO rates of non-disabled individuals performing treadmill exercise ($0.46 \pm 0.1$ g/minute at $48 \pm 1\%$ $VO_{2peak}$)[5] and those of the paraplegic subjects from this study performing voluntary arm ergometry ($0.13 \pm 0.07$ g/minute at $41 \pm 9\%$ $VO_{2peak}$; Fig. 1A) differ widely. However, when the non-disabled subjects of this study performed the same continuous progressive voluntary arm ergometer test as the subjects with paraplegia, they only reached a maximal WBFO rate of $0.06 \pm 0.04$ g/minute at $13 \pm 3\%$ $VO_{2peak}$.

Differences in substrate partitioning between upper and lower body exercise have primarily been examined in non-disabled individuals[20] and are complicated by large differences in $VO_{2peak}$ between the two modes of exercise. Certainly, the smaller mass of musculature in the upper body and consequent 25–30% lower $VO_{2peak}$ achieved during upper vs. lower body exercise[21] would translate to significantly lower maximal rates of whole body fat and CHO oxidation. Additionally, lower metabolic efficiency[20] and lower oxygen extraction[22] may explain the heavy reliance on CHO found in this study.

### *WBFO in paraplegic vs. non-disabled subjects*

The paraplegic subjects of this study had WBFO values that were twice those of non-disabled subjects. One other study has shown that subjects with paraplegia ($T_3$–$L_1$) have similar or higher rates of WBFO than non-disabled subjects during 60 minutes of arm ergometry after consuming water or glucose, respectively.[8] These authors suggested that the daily use of the upper body by those with SCI for locomotion and other common tasks may be sufficient to produce improvements in WBFO during exercise by inducing a shift towards Type I muscle fiber. While no longitudinal studies to date have examined training-induced adaptations in substrate oxidation in those with SCI, cross-sectional evidence does suggest that that long-term voluntary upper body exercise training may induce adaptations that increase WBFO during exercise in those with SCI. Examination of the anterior portion of the deltoid of trained and untrained individuals with SCI indicated that training resulted in higher levels of mitochondrial enzymes and greater capillary density.[11] Additionally, trained subjects with SCI with similar levels of injury (C7–L1), but much higher $VO_{2peak}$ values than the current subjects (35.0–37.5 ml/kg/minute) have been shown to have maximal WBFO rates during voluntary arm ergometry (0.22–0.28 g/minute) that exceed those of this study ($0.13 \pm 0.07$ g/minute).[9,10] However, these studies only measured substrate oxidation at intensities ranging from 55 to 75% $VO_{2peak}$, and given that maximal WBFO occurred at $41 \pm 9\%$ $VO_{2peak}$ in this study (Figs. 1A and 2A), they may have missed the true maximal WBFO rates of their subjects. Therefore, the greater ability of the paraplegic subjects to oxidize fat compared to the non-disabled subjects of this study may have been due to some degree of adaptation within the upper body skeletal muscle of the subjects with paraplegia induced by daily locomotion coupled with the unfamiliarity of arm ergometry exercise to the non-disabled and their likely lower metabolic efficiency.

### *Implications for the use of exercise as a therapeutic modality*

Prescribing exercise for those with SCI that coincides with maximal rates of WBFO observed in our study (~40% $VO_{2peak}$) is likely impractical, as this intensity is below the training threshold needed to induce significant increases in $VO_{2peak}$. Regardless of the chosen exercise intensity, individuals with SCI may lack the ability to oxidize significant amounts of fat during voluntary exercise. The addition of involuntary FES training of the lower body holds the promise of involving a greater volume of contracting musculature, thus maximizing fat oxidation and total EE during exercise. Exercise programs that promote maximal EE, regardless of substrate source, are likely to be the most beneficial in reducing the prevalence and severity of obesity and other related metabolic disorders in those with SCI. The limited ability of persons with SCI to increase EE during voluntary exercise highlights the necessity of a multifaceted approach in the prevention and treatment of obesity and other metabolic diseases in this population, in which exercise is accompanied by nutritional and behavioral interventions.

### *Study limitations*

As with many studies of those with SCI, our subject pool was small and heterogeneous. A limitation of this study was that the subjects with paraplegia had significantly lower $W_{peak}$ and $VO_{2peak}$ values compared with the non-disabled subjects. This is a common limitation of studies that compare SCI and non-disabled subjects and others have reported similar differences in $VO_{2peak}$ between untrained paraplegic (1.12 l/minute, 17.5 ml/kg/minute) and non-disabled subjects (2.53 l/minute, 38.7 ml/kg/minute) performing voluntary arm ergometry.[12] Matching these subjects on arm ergometry $VO_{2peak}$ requires the recruitment of highly trained

subjects with paraplegia,[14,23] significantly narrowing the scope and applicability of the findings. It also should be noted that the workloads of the paraplegic and non-disabled subjects were matched for relative intensity, the most important variable in the determination of substrate partitioning during exercise.[4]

Subjects were instructed to maintain their habitual food intake prior to each trial and reported to the laboratory after a 3–4-hour fast, but diet was not rigorously controlled. However, a recent study in individuals with SCI found no difference in CHO or fat oxidation rates during 60 minutes of moderate intensity exercise with or without pre-exercise glucose feeding.[8]

## Conclusions

Use of their upper body on a daily basis may improve the ability of sedentary individuals with paraplegia to oxidize fat during exercise compared to non-disabled individuals. However, an early crossover from fat to CHO, low maximal rates of WBFO compared to what can be achieved by non-disabled individuals during leg ergometry, and a heavy reliance on CHO characterizes substrate oxidation during voluntary arm ergometry among those with paraplegia across a wide range of exercise intensities. This limited ability to oxidize fat may largely be due to the mode of voluntary exercise available to those with paraplegia rather than their injury *per se*. This highlights the need for the incorporation of other forms of exercise such as involuntary FES training of the lower body as part of a multifaceted approach in the prevention and treatment of obesity and other metabolic diseases in this population.

## Acknowledgements

The authors wish to thank the participants for their time and efforts. Additionally, we would like to thank Jennifer Raeburn and Elizabeth Edwards for their assistance in the preparation of this manuscript.

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