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ORIGINAL ARTICLE

Effect of pre-exercise carbohydrate ingestion on substrate consumption in persons with spinal cord injury

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Objective: To investigate the influence of pre-exercise carbohydrate ingestion on fat and carbohydrate oxidation during prolonged arm cranking exercise in persons with spinal cord injury.
Subjects: Six male paraplegic subjects (PS, Ll–Th3, 46.3 ± 6.6 years) and seven able-bodied subjects (AB, 43.1 ± 4.6 years) were volunteered to participate in the present study.
Methods: The subjects were required to consume a glucose solution (1 g kg^{-1} body mass and 500 ml plain water; glucose experiment) or only plain water (water experiment) before the prolonged exercise. Then the subjects performed for 1-h arm cranking exercise at a moderate workload.
Results: In the water experiment, the carbohydrate oxidation slightly decreased and the fat oxidation increased continuously in AB. In contrast, the carbohydrate and fat oxidation of PS was constant during the exercise in the water experiment. In the glucose experiment, the fat oxidation did not rise and the carbohydrate oxidation was constant until the end of the exercise in PS and AB. PS oxidized more fat than AB in the glucose experiment ($P < 0.05$), but no significant difference was found between PS and AB in the water experiment.
Conclusion: Using a wheelchair in daily life regularly was regarded as an exercise training that disciplined PS indirectly and is considered to cause PS to have more percentage of type I fiber than AB in the anterior deltoid muscle. Thus, the distribution of muscle fiber type in anterior deltoid muscle might be one of the factors that impacted the fat oxidation of PS in glucose experiment.
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Keywords: paraplegic; fat oxidation; carbohydrate oxidation; arm cranking exercise

Introduction

Carbohydrates are important substrates for contracting muscle during strenuous, prolonged exercise. The reduction of body stores of carbohydrate and blood glucose (BG) is related to the perception of fatigue and the inability to maintain high-quality performance.[1] For this reason, many investigators examined the influence of carbohydrate ingestion before or during exercise. It has been reported that during moderate prolonged exercise, ingestion of a very large amount of glucose significantly reduced endogenous glucose oxidation, thus sparing glycogen stores of muscle and/or liver, presumably via enhanced carbohydrate oxidation in able bodied subjects (AB).[2] Moreover, Spendiff and Campbell (2005)[3] examined the effect of different concentrations of carbohydrate ingestion before moderately prolonged arm cranking exercise in paraplegic subjects (PS). They concluded that a higher concentration of carbohydrate in a sport drink might be a better choice for paraplegic athletes.[3]

Carbohydrate and free fatty acid (FFA) are the dominant fuels oxidized by the muscle for energy production during exercise[4] and the absolute and relative contribution of these fuels can be influenced by many factors, one of the most important factors of substrate oxidation is exercise intensity.[4] In healthy man and woman, Venables *et al.*[4] demonstrated that the crossover point of fat and carbohydrate oxidation occurred between 48 and 53% VO_{2max} in an incremental treadmill experiment. Recent studies revealed that well-trained athletes were able to oxidize more fat at high intensities than low intensities. Van Loon *et al.*[5] demonstrated the highly endurance-trained cyclists showed the highest fat oxidation at 57% VO_{2max} corresponding to a 55% $Workload_{max}$. In endurance-trained women, they showed the highest fat oxidation at a 75% VO_{2peak}.[6] We can recognize from these results that trained AB has higher exercise intensity of fat oxidation than untrained AB.

The substrate consumption of PS during exercise has also been investigated.[7,8] Knechtle *et al.*[7] pointed out that, during different intensities, 20 min arm cranking exercise, well-trained wheelchair athletes showed total energy expenditure and carbohydrate oxidation was highest at 75% VO_{2peak},

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while absolute fat oxidation was not significant different at the three tested intensities. In the next year, Knechtle *et al.*[8] reported that, during wheelchair exercise the highest absolute fat oxidation was found at 55% VO_{2peak} compared with cycling, where highest fat and carbohydrate oxidation was found at 75% VO_{2peak}. The different results in these studies may have been a consequence of the type of exercise[6] and duration.[2]

Although many studies were performed on substrate consumption during exercise in PS and AB, there was no study concerning the characteristics of fat and carbohydrate oxidation during prolonged arm cranking exercise using PS. Therefore, the purpose of this study was to clear the influence of glucose ingestion on fat and carbohydrate oxidation during prolonged arm cranking exercise in PS.

Methods

Subjects

Six male PS and seven male AB (mean ± standard error, s.e.: 43.1 ± 4.7 years, 72.6 ± 2.7 kg) were volunteered to participate in the present study. The general characteristics of PS are presented in Table 1. All subjects were physically active without any special upper body training, and had no evidence of metabolic or cardiovascular disorder nor were any of them taking prescription medications. All subjects gave their written informed consent. The present study was approved by the ethical committee of the Graduate School of Integrated Arts and Sciences, Hiroshima University.

Maximal exercise test

On the experiment day, subjects arrived at the laboratory (before 10.30 am) and performed a maximal exercise test on an arm cranking ergometer (Monark-881, Sweden). All subjects were asked to refrain from eating and drinking for 12 h preceding the experiment. The subjects were required to consume a glucose solution ($1\,g\,kg^{-1}$ body mass and 500 ml plain water; glucose experiment) or only 500 ml plain water (water experiment) immediately before the maximal exercise. The maximal exercise started at an intensity of 0 W with 50 r.p.m. The intensity then increased 5 W every 2 min until the subject was no longer able to maintain the required speed. Before the test and at the end of every 2 min, capillary blood was taken from earlobe (∼1 ml) to measure plasma lactate concentration (PLC) by enzymatic instruments (Lactate Pro, Japan and Medisafe GR-102 Terumo, Japan, respectively).

Table 1 Characteristics of all subjects

	Age (years)	Weight (kg)	Injury level	Injury duration (years)
PS				
1	64	65	Th10	29
2	60	74	Th6	33
3	30	51	Th12	11
4	38	65	L1	5
5	28	49	Th9	2
6	58	60	Th3	38
Mean ± s.e.	46.3 ± 6.6	60.7 ± 3.9	—	19.7 ± 6.3
AB				
1	53	73	—	—
2	53	65	—	—
3	50	66	—	—
4	28	82	—	—
5	28	75	—	—
6	35	81	—	—
7	55	66	—	—
Mean ± s.e.	43.1 ± 4.7	72.6 ± 2.7	—	—

Abbreviations: AB, able-bodied subjects; PS, paraplegic subjects.

Determination of lactate threshold

For each subject, PLC was plotted against exercise intensity using the data of the maximal exercise test. Piecewise linear regression analysis was applied to the data to determine the intensity at the inflection point of the lactate curve. The intensity at the inflection point was regarded as lactate threshold (LT).

Exercise protocol

Subjects were asked to refrain from eating and drinking for 12 h preceding the experiment. These subjects were required to consume a glucose solution ($1\,g\,kg^{-1}$ body mass and 500 ml plain water; glucose experiment) or only 500 ml plain water (water experiment) immediately before the prolonged exercise. The prolonged exercises consisted of arm cranking exercise for 60 min exercise with 50 r.p.m. at the workload of LT80%.

Physiological measurements

Before the exercise and at the end of every 5 min, capillary blood was taken from the earlobe (∼1 ml) to measure PLC by enzymatic instruments (Lactate Pro, Japan and Medisafe GR-102 Terumo, Japan, respectively). During the prolonged exercise, VO_2 and VCO_2 were continuously measured by an automatic apparatus (AE-300 s Minato Medical Science Co. Ltd, Japan) every 30 s, whose gas analyzers were calibrated before each test using standard O_2 (15%) and CO_2 (5%) gas and air. Before and at the end of the prolonged exercise, blood was taken from an arm vein by a nurse of the Hiroshima University Health Service Center to analyze for FFA and insulin. Heart rate was measured continuously during the exercise with a Heart Rate Monitor (Polar S610i, Japan) and recorded at every 1 min.

Calculation of oxidation rates

VO_2 ($l\,min^{-1}$) and VCO_2 ($l\,min^{-1}$) during the prolonged exercise were used to calculate the rate of the oxidized substrate. Total oxidation rates of fat and carbohydrate ($mg\,min^{-1}$) were calculated by the stoichiometric equations of Jeukendrup and Wallis[9] with the assumption that protein oxidation during exercise was negligible. The following calculations were used to assess the rates of oxidation during the exercise at every 1 min.

$$\text{Carbohydrate oxidation (mg/min)} = 4.21 \times VCO_2(\text{l/min}) - 2.962 \times VO_2(\text{l/min})$$

$$\text{Fat oxidation (mg/min)} = 1.695 \times VO_2(\text{l/min}) - 1.701 \times VCO_2(\text{l/min})$$

Statistical analysis
All data are expressed as mean value ± s.e. of mean. Analysis of variance (ANOVA) was employed to evaluate the differences in the mean values of the two conditions (water and glucose experiment) as well as the calculation of fat and carbohydrate oxidation rates during the prolonged arm cranking exercise. When ANOVA indicated the presence of a significant difference, *post hoc* comparisons using the Bonferroni's method were applied to determine pairwise differences. All analyses were performed using a statistical software package (SPSS Version 12.0) on a personal computer. A *P*-value less than 0.05 was considered significant.

Result

The results of the maximal exercise test were presented in Table 2. In the glucose experiment, AB showed higher maximal workload than water ingestion condition ($P<0.05$). There was no significant difference in peak oxygen uptake between PS and AB in the two conditions (Table 2). The FFA increased significantly after the exercise in both PS ($P<0.05$) and AB in the water experiment ($P<0.05$; Figure 1).

Table 2 Physiological measurements during the maximal exercise test

Variables	*PS*	*AB*
Water experiment		
$VO_{2\ peak}$ (l min^{-1})	1713 ± 202	1658 ± 105
$VO_{2\ peak}$ (l min^{1} kg^{-1})	28.8 ± 3.6	22.7 ± 0.9
$Workload_{max}$ (Watts)	71.7 ± 7.2	63.6 ± 4.2[a]
Glucose experiment		
$VO_{2\ peak}$ (l min)	1640 ± 224	1683 ± 154
$VO_{2\ peak}$ (l min kg^{-1})	27.8 ± 4.1	23.0 ± 1.8
$Workload_{max}$ (Watts)	73.3 ± 10.9	67.9 ± 4.6[a]

Abbreviations: AB, able-bodied subjects; PS, paraplegic subjects; VO_{2peak}, peak oxygen uptake; $Workload_{max}$, maximal workload.
Values are mean ± s.e.
[a]Water experiment vs glucose experiment ($P<0.05$).

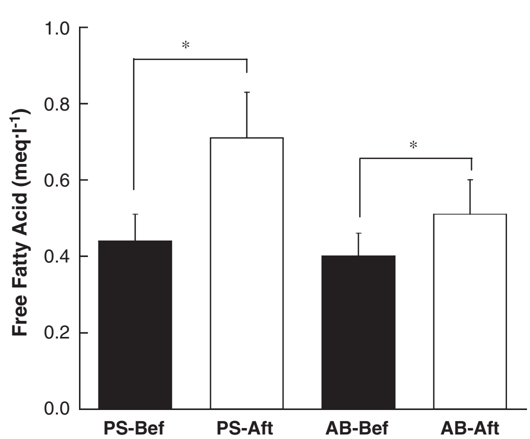

Figure 1 Free fatty acid before and after the prolonged exercise in water experiment. PS, paraplegic subjects; AB, able-bodied subjects; Bef, before the prolonged exercise; Aft, after the prolonged exercise. *PS vs AB ($P<0.05$)

However, only AB showed lower FFA after the exercise in the glucose experiment ($P<0.05$; Figure 2). The results of the PLC and insulin are presented in Table 3. In the water experiment, the PLC and insulin of AB significantly decreased after the exercise ($P<0.05$). In the glucose experiment, PS showed higher insulin ($P<0.05$), and AB showed higher PLC ($P<0.05$) and insulin ($P<0.05$) after the exercise.

Figure 3 presents the time course of the calculated fat and carbohydrate oxidation of AB during the prolonged exercise. In the water experiment, the carbohydrate oxidation decreased and the fat oxidation increased continuously during the exercise. However, the fat oxidation did not rise during the exercise, and the carbohydrate oxidation was constant until the end of the exercise in the glucose experiment. In contrast to AB, the fat and carbohydrate oxidation were almost constant during exercise with no influence of glucose ingestion during both experiments in PS

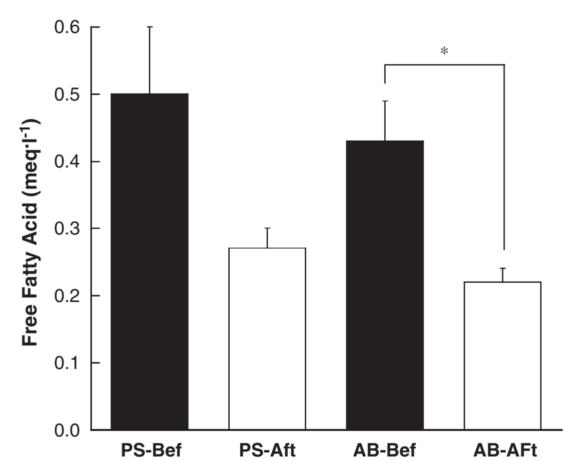

Figure 2 Free fatty acid before and after the prolonged exercise in glucose experiment. PS, paraplegic subjects; AB, able-bodied subjects; Bef, before the prolonged exercise; Aft, after the prolonged exercise. *PS vs AB ($P<0.05$).

Table 3 PLC and insulin before and after the prolonged exercise

Variables	*PS*	*AB*
Water experiment		
PLC-Bef (mmol l^{-1})	3.22 ± 0.49	2.14 ± 0.15[a]
PLC-Aft (mmol l^{-1})	2.38 ± 0.60	1.80 ± 0.26[a]
Insulin-Bef (μU ml^{-1})	5.82 ± 2.08	5.74 ± 1.23[a]
Insulin-Aft (μU ml^{-1})	5.77 ± 2.87	3.57 ± 0.33[a]
Glucose experiment		
PLC-Bef (mmol l^{-1})	2.68 ± 0.38	2.00 ± 0.10[b]
PLC-Aft (mmol l^{-1})	2.62 ± 0.49	2.80 ± 0.33[b]
Insulin-Bef (μU ml^{-1})	4.45 ± 1.70[b]	6.86 ± 1.73[b]
Insulin-Aft (μU ml^{-1})	17.83 ± 3.33[b]	17.81 ± 2.88[b]

Abbreviations: AB, able-bodied subjects; Aft, after the prolonged exercise; Bef, before the prolonged exercise; PLC, plasma lactate concentration; PS, paraplegic subjects.
Values are mean ± s.e.
[a]Aft vs Bef in water experiment ($P<0.05$).
[b]Aft vs Bef in glucose experiment ($P<0.05$).

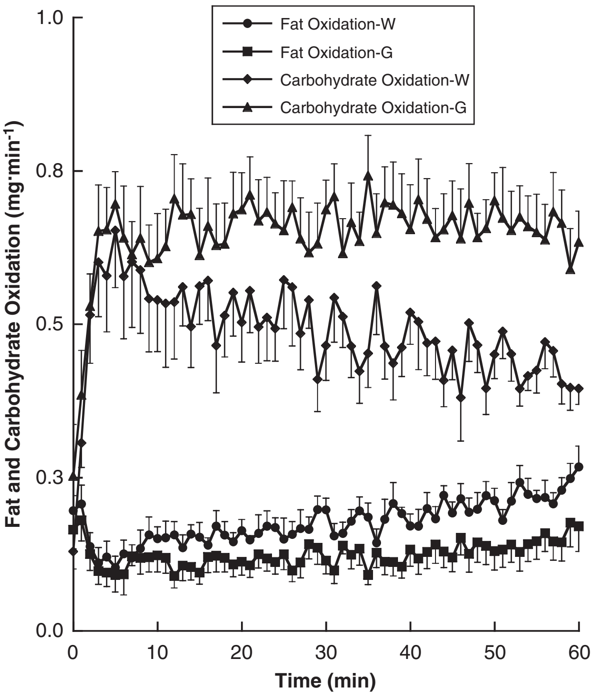

Figure 3 Fat and carbohydrate oxidation during the prolonged arm cranking exercise in able bodied subjects (AB). W, water experiment; G, glucose experiment.

(Figure 4). The amount of the fat oxidation per 1 kg body weight during the exercise is shown in Figure 5. Clearly, PS oxidized more fat than AB in the glucose experiment ($P<0.05$). Moreover, no significant difference was found between PS and AB in the water experiment. Figure 6 illustrates the carbohydrate oxidation per 1 kg body weight during the prolonged exercise. Although there was no significant difference in PS compared with AB, the consumption of the carbohydrate oxidation in PS was more than AB in the water experiment.

Discussion

During the prolonged exercise, the carbohydrate oxidation of PS was slightly higher than AB in the water experiment (Figure 6). It seems that using a wheelchair in daily life regularly might be a factor to influence carbohydrate utilization during the prolonged exercise that disciplined PS indirectly. Minoo *et al.*[10] compared carbohydrate oxidation using older AB males in three kinds of intensities (50, 60 and 70% VO_{2max}), for 30 min of cycling. They reported that carbohydrate oxidation was higher in trained than untrained older AB males. This finding was confirmed by Manetta *et al.*[11] who also investigated carbohydrate oxidation during 50 min of exercise at an exercise intensity above ventilatory threshold in middle-aged AB males, and they concluded that the carbohydrate oxidation in trained was higher than untrained man. These findings suggested, therefore, that using a wheelchair in daily life regularly might be considered as a factor that influenced the carbohydrate utilization during prolonged exercise and disciplined the physiological capacity of PS indirectly.

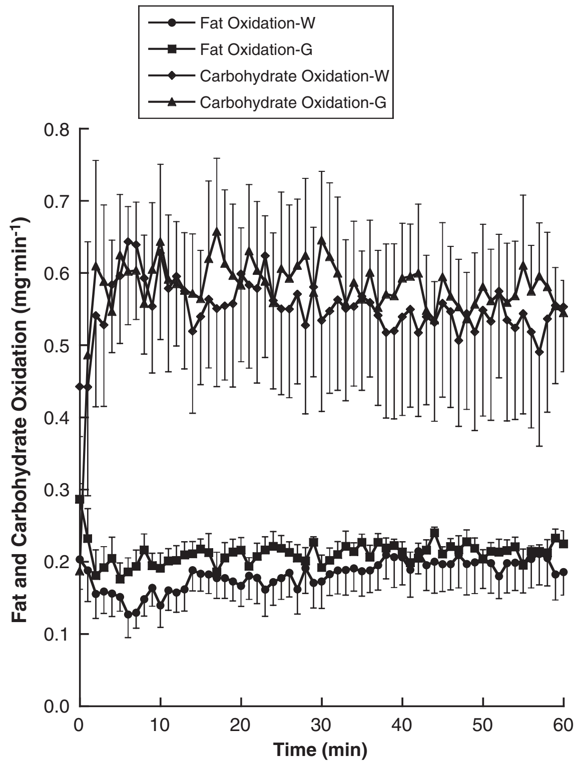

Figure 4 Fat and carbohydrate oxidation during the prolonged arm cranking exercise in paraplegic subjects (PS). W, water experiment; G, glucose experiment.

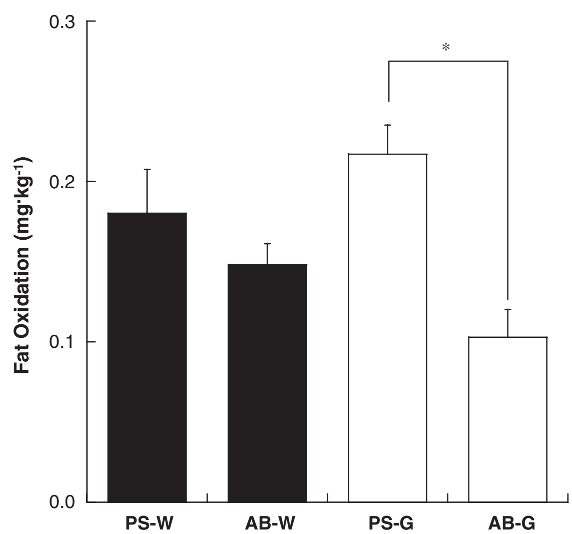

Figure 5 Comparison of fat oxidation in PS and AB. PS, paraplegic subjects; AB, able-bodied subjects; W, water experiment; G, glucose experiment. *$P<0.05$.

In the glucose experiment, the present study found no significant effect of glucose ingestion on the carbohydrate oxidation in PS (Figure 6). These findings have been supported by some investigators. Bosch *et al.*[12] used

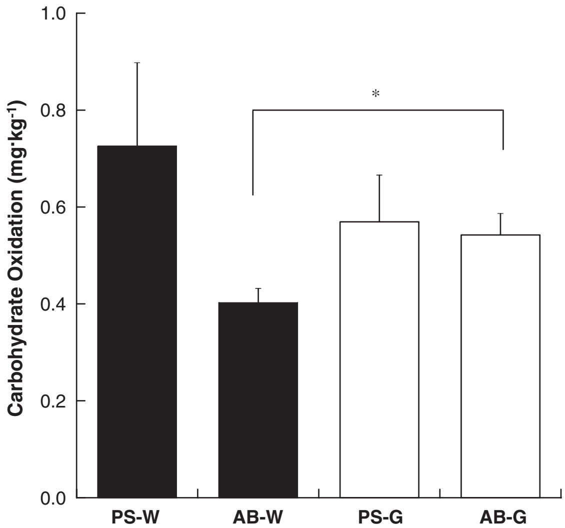

Figure 6 Comparison of carbohydrate oxidation in PS and AB. PS, paraplegic subjects; AB, able-bodied subjects; W, water experiment; G, glucose experiment. *$P<0.05$.

endurance-trained AB males who executed 180 min cycling at an intensity 70% VO_{2max}, and investigated the influence of ingestion of carbohydrate solution and plain water. They pointed out that muscle glycogen utilization was identical in both conditions.[12] McConell *et al.*[13] compared the influence of carbohydrate oxidation in glucose ingestion and placebo in well-trained AB males who participated in an endurance exercise lasting ~1 h at an intensity of 83 ± 1% VO_{2peak}. They concluded that glucose ingestion had no effect on carbohydrate oxidation.[13] Both Bosch *et al.* and McConell *et al.* did not observe increased carbohydrate oxidation rates with carbohydrate ingestion during prolonged exercise. Although using a wheelchair in daily life is considered to discipline the physiological capacity of PS, these studies mentioned above indicated no increase of carbohydrate oxidation during prolonged exercise in trained subjects. Thus, there are many discrepancies in the literature with regarding to the effect of carbohydrate ingestion on the metabolic substrate.

As shown in Figure 1, the FFA significantly increased after the prolonged exercise in the water experiment in both PS and AB. It has been demonstrated that during low intensity exercise, FFA provides fuel sources for metabolism in contracting muscles.[14] Minoo *et al.*[10] used trained and untrained older healthy male subjects to explore the interrelations of exercise intensity and exercise duration for the same total energy expenditure on fat utilization during moderate cycling training. They indicated that the increased FFA and glycerol concentration was not related to the intensity or duration of exercise and higher in the trained than the untrained subjects.[10] It is considered that using a wheelchair frequently can increase the fat utilization during prolonged exercise.

In the glucose experiment, the FFA significantly decreased after the prolonged exercise only in AB ($P<0.05$) and in PS the decrease was observed but not significant ($P<0.08$) as presented in Figure 2. There was accumulating evidence to support an inhibitor role of increased exogenous glucose availability on fat utilization during exercise. Arkinstall *et al.*[15] compared the results of FFA in glucose ingestion and water ingestion during running and observed that FFA was significantly lower in glucose ingestion than water ingestion after 60 min of running. This study indicates that the reduction in fat metabolism following glucose ingestion is because of the coordinated effects of decreased fatty acid availability, secondary to decreased adipose lipolysis and fatty acid oxidation in the muscle.[16] All these things make it clear that, in the present study, PS had significantly higher fat oxidation when compared with AB in the glucose experiment (Figure 5). Therefore, pre-exercise glucose ingestion has less influence on the fat utilization in PS than AB during prolonged arm cranking exercise.

Fiber distribution might be one of the factors that can explain why PS showed higher fat oxidation during the glucose experiment compared with AB ($P<0.05$; Figure 5). Human muscle consists of different muscle fiber types. The distribution of type of muscle fiber and the change of muscle fiber recruitment during endurance exercise is considered to play a role in substrate metabolism.[16] During a prolonged arm cranking exercise, shoulder is the most important part of the body for propulsion of PS and the anterior part of deltoid muscle consists of more type I fiber than type II fiber.[17] It has been well documented that after spinal cord injury, paralyzed skeletal muscle of PS generally becomes atrophic, possesses lower tension generating capacity and is less fatigue resistant and as a result muscle histochemical and metabolic profiles shift toward type II fiber.[18] However, fiber type transportation from type II to type I[19] can occur in response to endurance training.[20] The highest percentage of type I fiber in anterior deltoid muscle was found in tetraplegia with 74%, followed in PS with 57% and in AB with 42%.[18] In untrained PS, Schantz *et al.*[18] pointed out that in anterior deltoid muscle, lower type IIB fiber and higher type I fiber percentages were noted when untrained PS were compared with untrained AB.[18] Furthermore, Taylor *et al.*[20] reported that in anterior deltoid muscle, well-trained PS had greater fiber areas, but lower levels of glycolytic and oxidative marker enzymes than highly-trained AB. Reflection on these results will make clear that because PS have higher percentage of type I fiber in anterior deltoid muscle, it is a good possibility of PS who could oxidized more fat than AB during the prolonged arm cranking exercise even though the present study only found significant difference in the glucose experiment.

In conclusion, during the prolonged arm cranking exercise, PS showed higher fat oxidation than AB both in the two conditions (water and glucose experiment), though the significant difference was only found in the glucose experiment. Using a wheelchair in daily life regularly could be regarded as an exercise training that disciplined PS indirectly. The daily regular wheelchair propulsion is considered to cause PS to have more percentage of type I fiber than AB in the anterior deltoid muscle. Thus, the distribution of muscle fiber type in anterior deltoid muscle might be one of the factors that impacted the fat oxidation of PS in glucose experiment.

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