

A Single Bout of Upper-Body Exercise Has No Effect on Postprandial Metabolism in Persons with Chronic Paraplegia

MATTHEW T. FARROW^{1,2,3}, JENNIFER L. MAHER^{1,2}, TOM E. NIGHTINGALE^{4,5}, DYLAN THOMPSON^{1,2}, and JAMES L. J. BILZON^{1,2,3}

¹Department for Health, Centre for Clinical Rehabilitation and Exercise Medicine (CREM), University of Bath, Bath, UNITED KINGDOM; ²Department for Health, Centre for Nutrition and Exercise Metabolism (CNEM), University of Bath, Bath, UNITED KINGDOM; ³Centre for the Analysis of Motion, Entertainment Research and Applications (CAMERA), University of Bath, Bath, UNITED KINGDOM; ⁴School of Sport, Exercise and Rehabilitation Sciences, University of Birmingham, Edgbaston, Birmingham, UNITED KINGDOM; and ⁵International Collaboration on Repair Discoveries, University of British Columbia, Vancouver, BC, CANADA

ABSTRACT

FARROW, M. T., J. L. MAHER, T. E. NIGHTINGALE, D. THOMPSON, and J. L. J. BILZON. A Single Bout of Upper-Body Exercise Has No Effect on Postprandial Metabolism in Persons with Chronic Paraplegia. *Med. Sci. Sports Exerc.*, Vol. 53, No. 5, pp. 1041–1049, 2021.

Purpose: The acute effects of a single bout of upper-body exercise on postprandial metabolism in persons with spinal cord injury are currently not well understood. The primary aim of this study was to evaluate the effects of a single bout of upper-body high-intensity interval exercise (HIIE) and moderate-intensity continuous exercise (MICE) in comparison with a no-exercise control (REST) condition on postprandial metabolic responses in persons with chronic paraplegia. **Methods:** Ten participants (eight males, two females; age, 49 ± 10 yr; time since injury, 22 ± 13 yr) with chronic paraplegia took part in a randomized crossover study, consisting of three trials: HIIE (8×60 s at 70% peak power output [P_{PEAK}]), MICE (25 min at 45% P_{PEAK}), and REST, at least 3 d apart. Exercise was performed in the fasted state, and participants consumed a mixed-macronutrient liquid meal 1-h postexercise. Venous blood and expired gas samples were collected at regular intervals for 6-h postmeal consumption. **Results:** There were no significant differences in postprandial incremental area under the curve for triglycerides ($P = 0.59$) or glucose ($P = 0.56$) between conditions. Insulin incremental area under the curve tended to be lower after MICE (135 ± 85 nmol·L⁻¹ per 360 min) compared with REST (162 ± 93 nmol·L⁻¹ per 360 min), but this did not reach statistical significance ($P = 0.06$, $d = 0.30$). Participants reported a greater fondness ($P = 0.04$) and preference for HIIE over MICE. **Conclusions:** After an overnight fast, a single bout of upper-body exercise before eating has no effect on postprandial metabolism in persons with chronic paraplegia, irrespective of exercise intensity. This suggests that alternative exercise strategies may be required to stimulate postprandial substrate oxidation for this population. **Key Words:** EXERCISE INTENSITY, SPINAL CORD INJURY, INSULIN, GLUCOSE, TRIGLYCERIDES

Individuals with a spinal cord injury (SCI) are at an increased risk of developing cardiovascular disease (CVD) in comparison with the nondisabled population (1). It is therefore unsurprising that this population present a high prevalence of risk factors associated with CVD, including central adiposity (2), dyslipidemia (3), and impaired glucose tolerance (4). The

role of regular exercise training in the prevention of these CVD risk factors is well established in noninjured humans, and current SCI-specific exercise guidelines recommend that people with chronic SCI engage in at least 30 min of moderate- to vigorous-intensity aerobic exercise three times per week to improve cardiometabolic health (5). Specifically, there is consistent evidence that upper-body moderate-intensity continuous training improves fasting insulin sensitivity and reduces waist circumference in persons with chronic SCI (6). These chronic adaptations are a result of numerous individual bouts of exercise, but the metabolic responses to a single bout of upper-body exercise in this population are not well understood.

In particular, the effect of a single bout of upper-body exercise on postprandial metabolism is important to determine as humans spend most of the waking day in a fed state, with elevated postprandial glucose and triglyceride responses, both independent risk factors for CVD (7,8). In addition, persons with SCI may have exaggerated postprandial lipemic and glycemic responses compared with the nondisabled population, which may partially explain their increased risk of developing CVD

Address for correspondence: James Bilzon, Ph.D., Centre for Clinical Rehabilitation and Exercise Medicine, University of Bath, Bath BA2 7AY, United Kingdom; E-mail: J.Bilzon@bath.ac.uk.
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(9,10). A single bout of moderate-intensity continuous exercise (MICE) (90 min at 50% maximal oxygen uptake) can decrease the postprandial triglyceride response to a high-fat meal consumed ~12–18 h postexercise in healthy nondisabled individuals (11). In people with type 2 diabetes, a single bout of MICE performed in the postprandial state can reduce short-term glucose area under the curve (AUC) and the prevalence of 24-h hyperglycemia (12). However, it is unclear how a single bout of upper-body MICE affects subsequent postprandial responses in persons with SCI.

There has been growing interest in high-intensity interval exercise (HIIE) as an alternative solution to MICE to improve cardiometabolic health outcomes in persons with SCI (13). HIIE can be generally characterized as repeated short intervals eliciting $\geq 80\%$ (but often 85%–95%) of maximum heart rate (14). This interest stems from a randomized controlled trial demonstrating that 180 min·wk⁻¹ of MICE is sufficient to improve cardiorespiratory fitness and fasting insulin sensitivity, but not fasting glucose, peripheral insulin sensitivity, or the lipid profile, suggesting that a higher exercise intensity is required (15). Training programs involving HIIE and MICE elicit comparable improvements, in insulin sensitivity, blood pressure, and body composition, in nondisabled overweight and obese individuals (16). Pilot work in individuals with SCI also indicates similar improvements in insulin sensitivity after training programs involving HIIE and MICE (17). HIIE is particularly appealing given the reduced time commitment and is often cited as more enjoyable than MICE (18). This finding has recently been replicated during upper-body exercise in persons with chronic SCI (19). It also appears that a single bout of HIIE can attenuate the postprandial glucose and triglyceride to meal to a similar extent as MICE in noninjured humans (20,21).

Bailey et al. (22) recently reported that postprandial glucose responses were attenuated by regularly breaking up sedentary time with short bouts of moderate-intensity arm-crank ergometry in persons with chronic paraplegia. However, to our knowledge, there are no published studies assessing the effect of a single bout of upper-body exercise on subsequent metabolic responses to a mixed-macronutrient test in this population. Therefore, the aim of this study was to evaluate the effects of both an acute bout of upper-body HIIE and MICE in comparison with a no-exercise control condition (REST) on postprandial metabolic responses to a mixed-macronutrient meal in persons with chronic paraplegia. We hypothesized that HIIE and MICE would be equally and more effective in reducing the total serum triglyceride response in comparison with the no-exercise condition.

METHODS

This study was approved by South West (Bristol) National Research Ethics Committee (REC reference number 19/SW/0021). All participants provided written informed consent, and the study conformed to the principles of the Declaration of Helsinki. The study was registered as a clinical trial at ClinicalTrials.gov (<https://clinicaltrials.gov/>) under the identifier NCT04011137.

Participants. We aimed to recruit 11 participants, based on an *a priori* sample size calculation (Cohen's $d = 0.97$, $\alpha = 0.05$, $\beta = 0.80$), to detect a significant difference in the total postprandial triglyceride response between the HIIE and the no-exercise control condition (20). A total of 13 individuals with chronic paraplegia agreed to take part in this randomized crossover study, two participants were withdrawn due to difficulties with venous cannulation, and one participant withdrew due to a lack of time, leaving a total of 10 participants (8 males and 2 females) completing all components of the study. Participant descriptive characteristics are presented in Table 1.

Participants were eligible to participate if they met all the following criteria: between 18 and 65 yr old, chronic (>1 yr postinjury) spinal cord lesion at or below the second thoracic level, self-reported wheelchair use of $>75\%$ of waking day in individuals with motor-incomplete injuries, and body mass not changed by $>3\%$ over the previous 3 months. Individuals who self-reported the use of lipid-lowering agents and/or antihyperglycemic drugs, type 2 diabetes mellitus medication, active medical issues (including pressure sores, urinary tract infection, and upper-body musculoskeletal issues), or contraindications to exercise testing were excluded.

Study design. Participants visited the laboratory on four separate occasions (one preexperimental visit and three experimental trials). The preexperimental procedures included basic anthropometric measurements, an assessment of resting metabolic rate (RMR) and peak aerobic capacity ($\dot{V}O_{2peak}$), and HIIE familiarization session. A submaximal exercise test was also performed to allow the individual calibration of a physical activity monitor, which participants wore for a 7-d period commencing immediately after this initial visit. The three experimental trials (MICE, HIIE, and REST) were then performed in a randomized order, at least 3 d apart. Participants arrived in a fasted state and performed one of the three conditions, which was followed by a 6-h mixed meal tolerance test (MMTT) (Fig. 1).

Preexperimental visit. Participants arrived at the laboratory after an overnight fast (>10 h), having refrained from caffeine and alcohol (24 h prior) and strenuous physical activity (48 h prior). Body mass was measured using platform wheelchair scales (Decto® BRW1000, Webb City, MO). Supine length, waist circumference, and hip circumference were

TABLE 1. Participant characteristics ($n = 10$).

Age (yr)	49 ± 10
AIS classification	A: 7, B: 3
LOI	T3–T12 (T3: 1, T4: 1, T5: 1, T6: 2, T10: 1, T12: 4)
TSI (yr)	22 ± 13
BMI (kg·m ⁻²)	28.1 ± 6.2
Waist-hip ratio	0.94 ± 0.08
PAL	1.50 ± 0.17
$\dot{V}O_{2peak}$ (mL·min ⁻¹ ·kg ⁻¹)	20.3 ± 5.32
HOMA2-IR	1.29 ± 0.74
Fasting glucose (mmol·L ⁻¹)	6.53 ± 1.54
Fasting triglycerides (mmol·L ⁻¹)	1.73 ± 1.00

Data are presented as mean ± SD.

AIS, American Spinal Injury Association Impairment Scale; LOI, level of injury; TSI, time since injury; BMI, body mass index; PAL, physical activity level; $\dot{V}O_{2peak}$, peak oxygen uptake; HOMA2-IR, homeostatic model assessment for insulin resistance.

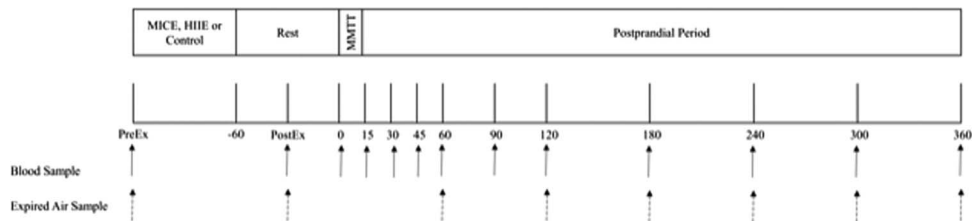


FIGURE 1—Schematic of experimental trial days (laboratory visits 2–4).

measured while the participant was lying flat on a medical bed. RMR was estimated via indirect calorimetry from four 5-min expired gas samples collected into Douglas Bags (Hans Rudolph, Kansas City, MO) through a mouthpiece. Ambient O_2 and CO_2 fractions, in addition to atmospheric pressure and temperature, were measured at close proximity to the participants to account for changes in an enclosed laboratory environment (23). Fractions of expired O_2 and CO_2 were measured using a paramagnetic O_2 and infrared CO_2 analyzer (miniMP 5200; Servomex, Crowborough, UK), calibrated with known concentrations of gas on the morning of testing. RMR was calculated using stoichiometric equations (24) and was recorded as the mean of three samples differing by $\leq 100 \text{ kcal}\cdot\text{d}^{-1}$.

A submaximal incremental exercise test was then performed on an electronically braked arm-crank ergometer (Lode Angio, Groningen, Netherlands), consisting of four 3-min stages, starting at 5 W, and increasing by either 10 or 15 W (depending on self-reported fitness). Energy expenditure (using the Douglas Bag method) and heart rate for each stage were used to perform an individual calibration of a chest-worn physical activity monitor (Actiheart™; Cambridge Neurotechnology Ltd., Papworth, UK) (25). Participants were instructed to wear the device for 7 d to monitor habitual physical activity patterns. Subsequently, physical activity energy expenditure and physical activity level were estimated (26).

After an adequate rest, participants then performed a $\dot{V}O_{2\text{peak}}$ test on an electronically braked arm-crank ergometer. The ramp-based protocol included a 2-min warm-up at 10 W before increasing by 1 W every 6 s. Before the test, participants were fitted with a rubber face mask connected to a two-way breathing valve; this was connected to a computerized metabolic system (TrueOne® 2400; ParvoMedics, Salt Lake City, UT). The system was calibrated with a known concentration of gas (20% O_2 , 8% CO_2) and a 3-L calibration syringe on the morning of testing. Heart rate and single-breath data were recorded simultaneously on the software throughout the test. A cadence of $\sim 75 \text{ rpm}$ was encouraged throughout, and the test was terminated at volitional fatigue or when cadence dropped below 50 rpm. $\dot{V}O_{2\text{peak}}$ was defined as the highest 15-breath rolling average for $\dot{V}O_2$. Peak power output (P_{PEAK}) was defined as the highest power output achieved before termination of the test. All participants achieved a valid $\dot{V}O_{2\text{peak}}$ according to the following criteria: peak HR $\geq 95\%$ age-predicted maximum for upper-body exercise (200 bpm – age),

RPE ≥ 19 , and a peak RER ≥ 1.10 . Participants then performed a shortened HIIE protocol on the electronically braked arm-crank ergometer, consisting of a 1-min warm-up at 10% P_{PEAK} , followed by four 60-s intervals at 70% P_{PEAK} , interspersed by 60-s recovery intervals at 10% P_{PEAK} . Participants were encouraged to reach a cadence of at least 75 rpm before the start of each high-intensity bout.

Experimental trials. Before all three conditions, participants refrained from strenuous physical activity in the 48 h prior and consuming alcohol or caffeine in the 24 h prior. Participants arrived at the laboratory at the same time each morning (between 8:00 and 10:00 AM) to minimize diurnal variation, after an overnight fast ($>10 \text{ h}$) and having consumed ~ 1 pint of water on waking. In the 2 d before the first experimental trial, participants completed a nonweighed food diary and asked to replicate this before each experimental trial. Trials were completed within the follicular phase of the menstrual cycle (3–10 d after onset of menses) for the eumenorrheic females taking part in the study.

Upon arrival, body mass was measured, a resting expired gas sample was obtained, and a fasting blood sample was collected via venipuncture (“PreEx”) from the antecubital vein. One of three conditions was then performed in a randomized order ($\geq 3 \text{ d}$ apart): (i) REST—a no-exercise control condition, (ii) MICE—25 min at 45% P_{PEAK} , and (iii) HIIE—eight 60-s intervals at 70% P_{PEAK} , interspersed with 60-s recovery intervals at 10% P_{PEAK} . Both exercise protocols began with a 5-min warm-up at 10% P_{PEAK} , and the HIIE condition included a 5-min cooldown at the same intensity. Participants wore a rubber face mask connected to a computerized metabolic system as previously described. Heart rate, RPE (global, local, and central), and affective valence were recorded at the end of the warm-up (0%), 25%, 50%, 75%, and 100% through each exercise condition. Affective valence was measured using the Feeling Scale, whereby participants are asked how they feel at the current moment using an 11-point scale, ranging from “very bad” (–5) to “very good” (+5) (27). Expired gases were averaged across 1-min intervals, and total exercise energy expenditure was calculated using published equations for high-intensity exercise (28). When RER exceeded 1.0, energy expenditure was calculated assuming a relationship of 5 kcal used for each 1 L of O_2 consumed (29).

Within 30 min of exercise completion, participants completed a modified Physical Activity Enjoyment Scale (30). Participants also completed a 5-item questionnaire relating to

exercise self-efficacy (31). This measure asked participants to consider how confident they were to be able to perform the exercise protocol (once to five times per week) over the next 4 wk, with responses ranging from “not at all” (0%) to “extremely confident” (100%), in increments of 10%. After the completion of both exercise trials, participants were asked which type of exercise they preferred, and their fondness of each, on a 7-point Likert scale ranging from “very much dislike” (1) to “extremely like” (7).

At 30 min postexercise, expired gas and blood samples were collected (“PostEx”), after a cannula was inserted into an antecubital vein. At 60 min postexercise, participants then consumed a mixed-macronutrient liquid meal that provided a total energy content of 65% RMR, chosen to meet resting energy requirements for the study hours in which no other food was consumed (i.e., ~17 h). The macronutrient composition (45% calories from carbohydrate, 37% calories from fat, and 18% calories from protein) was designed to reflect that of a typical meal in persons with SCI (32). Participants were given 10 min to consume the meal. Expired gas samples were collected at 60, 120, 180, 240, 300, and 360 min after drink consumption. Blood samples were collected at 0, 15, 30, 45, 60, 90, 120, 180, 240, and 360 min after meal consumption. All blood samples were collected with the participant’s hand in a heated hand box (55°C) (33).

All arterialized blood samples collected (10 mL) were dispensed into treated serum collection tubes and then centrifuged at 4000g for 10 min at 4°C. The serum was then apportioned into aliquots, cooled immediately on dry ice, and then stored in a -80°C freezer for long-term storage before analysis. Serum triglyceride, glucose, nonesterified fatty acid (NEFA), and glycerol concentrations were determined using an automated analyzer (Randox RX Daytona, Co., Antrim, UK). Serum insulin concentrations were determined by commercially available enzyme-linked immunosorbent assay (Mercodia AB, Uppsala, Sweden). Expired gas samples were used to estimate carbohydrate and fat oxidation rates, using previously published equations (24).

Statistical analysis. Paired *t*-tests were performed to compare total exercise energy expenditure, exercise enjoyment, exercise self-efficacy, and fondness between MICE and HIIE. The normality of the paired differences was checked using a Shapiro–Wilk test, and if significant, Wilcoxon tests were performed instead. A mixed-model ANOVA (condition × time) was performed to analyze serum blood analytes (glucose, insulin, triglycerides, NEFA, and glycerol) and indirect calorimetry derivatives (carbohydrate and fat oxidation rates, and RER) over time. A two-way ANOVA was used to compare %HR_{PEAK}, RPE, and affective valence over time between MICE and HIIE. One-way repeated-measures ANOVA were performed to compare the total AUC (TAUC) and the incremental AUC (iAUC) in the postprandial period (0 to 360 min) for glucose, insulin, and triglycerides, NEFA, and glycerol. Where significant interaction effects were found, Bonferroni comparisons were performed to identify the source of variation. Sphericity was determined with Greenhouse–Geisser

epsilon; all values <0.75 were corrected with Greenhouse–Geisser corrections. Statistical significance was accepted at $P \leq 0.05$. All data are presented as mean (lower 95% CI, upper 95% CI) unless otherwise noted. In addition, effect sizes (Cohen’s *d*) were calculated and interpreted as follows: small effect = 0.20–0.49, medium effect = 0.50–0.79, and large effect ≥ 0.80 .

RESULTS

Participant characteristics. The mean \pm SD $\dot{V}O_{2peak}$ values of the male ($n = 8$) and female ($n = 2$) participants were 21.4 ± 5.4 and 15.8 ± 0.6 mL·min⁻¹·kg⁻¹, respectively. Therefore, the fitness classifications of the male participants were poor ($n = 1$), average ($n = 2$), good ($n = 3$), and excellent ($n = 2$) (34). Eight participants (80%) could be classified as living a sedentary lifestyle (physical activity level, ≤ 1.60). Nine participants (90%) had a raised fasting glucose concentration (≥ 5.6 mmol·L⁻¹), and four participants (40%) could be classified as having hypertriglyceridemia (fasting triglycerides ≥ 1.7 mmol·L⁻¹) (35).

Mean \pm SD RMR was 1595 ± 227 kcal·d⁻¹; therefore, participants consumed a total 1037 ± 148 kcal for the MMTT, consisting of 129 ± 18 g of carbohydrate, 41 ± 6 g of fat, and 42 ± 6 g of protein.

Exercise characteristics. Mean \pm SD P_{PEAK} was 100 ± 28 W. Participants exercised at 45 ± 13 W for the MICE condition, which corresponded to an overall exercise intensity of $58\% \pm 7\%$ $\dot{V}O_{2peak}$. During the HIIE condition, participants exercised at 70 ± 20 and 10 ± 3 W for the “high” and “recovery” phases, respectively. This corresponded to an overall exercise intensity of $58\% \pm 8\%$ $\dot{V}O_{2peak}$ for the HIIE condition, $57\% \pm 10\%$ $\dot{V}O_{2peak}$ for the “high” intervals, and $59\% \pm 8\%$ $\dot{V}O_{2peak}$ for the “recovery” intervals.

The mean \pm SD total exercise energy expenditure was greater during MICE (128 ± 24 kcal) compared with HIIE (98 ± 15 kcal, $P < 0.01$). The %HR_{PEAK} was greater in HIIE compared with MICE at 75% ($P = 0.02$, $d = 0.42$) of exercise completion and tended to be greater at 50% ($P = 0.08$, $d = 0.34$) and 100% ($P = 0.09$, $d = 0.35$) of exercise completion (Fig. 2).

There were no significant interaction effects between conditions at any time point for global ($P = 0.75$), local ($P = 0.94$), and central ($P = 0.73$) RPE or affective valence ($P = 0.97$). There was no significant difference in enjoyment (mean \pm SD) between HIIE (93 ± 14) and MICE (82 ± 23) ($P = 0.13$). However, participants reported a greater fondness (mean \pm SD) for HIIE (5.5 ± 1.0) compared with MICE (4.1 ± 1.5) ($P = 0.04$, $d = 1.15$). Participants also reported a higher exercise self-efficacy at being able to perform four ($P = 0.03$, $d = 0.54$) and five ($P = 0.04$, $d = 0.33$) bouts per week of HIIE compared with MICE. Eight participants stated a preference for the HIIE and two participants for MICE.

Serum blood analytes. There were no significant interaction effects between conditions at any time point for serum insulin ($P = 0.77$; Fig. 3), glucose ($P = 0.98$; Fig. 4), or triglycerides ($P > 0.99$; Fig. 5). However, there was a significant

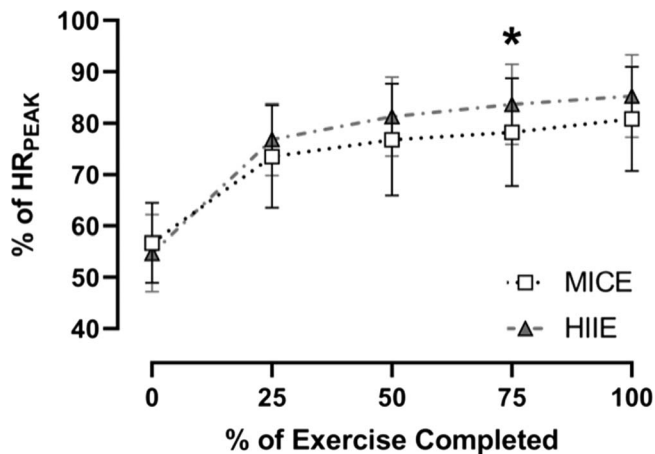


FIGURE 2—Heart rate (expressed as a % of HR_{PEAK}) at 0%, 25%, 50%, 75%, and 100% of exercise completion during MICE and HIIE. *Significant difference ($P \leq 0.05$) between conditions.

effect of condition for glucose ($P < 0.01$; Fig. 4), with the mean blood glucose concentration lower for the HIIE condition, in comparison with the MICE and REST (both $P < 0.01$). Serum insulin TAUC (data not shown, $d = 0.27$) and iAUC ($d = 0.30$) tended to be lower after MICE compared with REST (both $P = 0.06$) (Fig. 3). There were no significant differences between conditions for TAUC (data not shown) or iAUC for glucose ($P = 0.27$ and $P = 0.56$, respectively; Fig. 4) and triglycerides ($P = 0.74$ and $P = 0.59$, respectively; Fig. 5).

There were no significant interaction effects between trials at any time point for serum NEFA or glycerol, although there was a significant effect of condition for glycerol ($P = 0.01$), with mean glycerol concentration higher in the HIIE condition compared with the resting control condition ($P = 0.02$) (data not shown). In addition, serum NEFA ($P = 0.20$) and glycerol TAUC ($P = 0.37$) did not differ between conditions (data not shown).

Indirect calorimetry. There were no significant interaction effects between trials at any time point for fat ($P = 0.84$) and carbohydrate ($P = 0.71$) oxidation rates or RER ($P = 0.85$). However, there was a significant effect of condition for both fat

and carbohydrate oxidation and RER across the whole trial (all $P < 0.01$). RER was significantly lower in the MICE condition compared with both the HIIE ($P = 0.02$) and the resting control condition ($P < 0.01$).

DISCUSSION

The purpose of this study was to determine the effect of previous upper-body exercise (MICE and HIIE) on postprandial responses to a mixed-macronutrient meal in individuals with chronic paraplegia. Contrary to our hypothesis, a single bout of upper-body exercise was insufficient to reduce the subsequent postprandial triglyceride responses in comparison with the no-exercise condition. Despite no differences in postprandial glucose responses, the insulin response tended to be lower after MICE in comparison with the no-exercise REST condition. Participants reported a preference and a greater fondness and self-efficacy for HIIE compared with MICE.

Upper-body MICE and HIIE had no effect on the subsequent postprandial triglyceride response in comparison with the no-exercise REST condition. This contrasts findings from noninjured populations that a single acute bout of MICE or HIIE performed 12–18 h before a standardized meal attenuates the postprandial triglyceride response (11,36). Although studied less extensively, prior research indicates that this effect appears to still hold true when exercise is performed immediately (≤ 1 h) before the tolerance test (37,38). However, it appears that the magnitude of this effect is partially dependent on the energy expended during exercise, and there may be an exercise energy expenditure threshold needed to elicit changes in postprandial triglycerides (20,39). Therefore, an insufficient exercise energy expenditure (~ 100 – 130 kcal), which is a result of the limited active muscle mass involved in upper-body exercise, may partially explain the lack of change observed in the postprandial triglyceride response in the present study. It is also important to note that after the consumption of the liquid meal, participants were initially in a positive energy balance, which appears to diminish the beneficial effect of exercise on postprandial triglycerides (20).

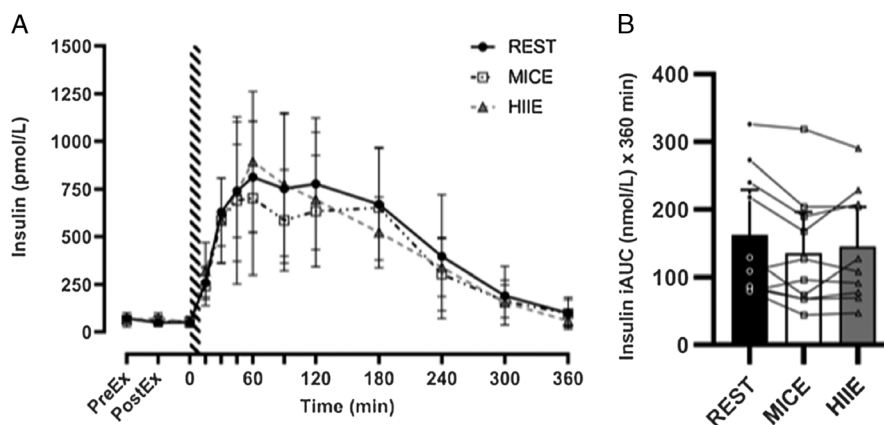


FIGURE 3—Serum concentrations of insulin (A) across each condition and iAUC (individual responses also denoted) for serum insulin (B) across the 6-h postprandial period after consumption of the MMTT. The hashed box represents consumption of the meal.

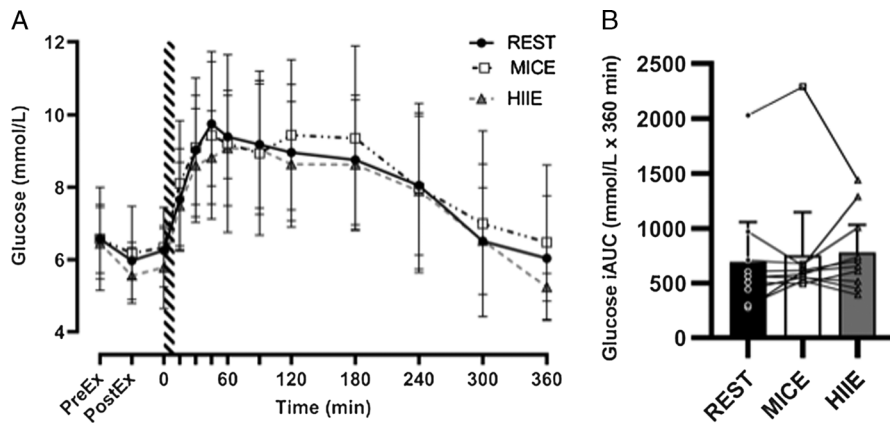


FIGURE 4—Serum concentrations of glucose (A) across each condition and iAUC (individual responses are also denoted) for serum glucose (B) across the 6-h postprandial period after consumption of the MMTT. The hashed box represents consumption of the meal.

There was also no beneficial effect of either exercise condition on postprandial glucose responses in comparison with the no-exercise condition. This is perhaps unsurprising, given that studies have demonstrated that 60 min of treadmill walking in the fasted state has no effect on glucose responses to a mixed-macronutrient meal in persons with obesity (37) and hyperglycemia (40). An increased rate of appearance of glucose from the liquid meal during the initial 3-h postexercise period is likely to be the reason for the lack of difference in postprandial glucose, offsetting the increased clearance rate (41). However, it is interesting to note that Short et al. (42) found that glucose clearance was increased after a 35-min moderate-to-vigorous-intensity handcycle exercise in adolescents with spina bifida or cerebral palsy. The reasons for this discrepancy with the present study are not immediately clear but may be related to the basal glucose tolerance of participants and/or differences in the quantity and macronutrient content of the oral tolerance test.

Despite the lack of differences in postprandial glucose after either exercise condition, the insulin iAUC tended (20%, $P = 0.06$) to be lower after MICE compared with the no-exercise REST

condition, with eight participants displaying a reduction. Comparatively, Farah and Gill (37) observed a 19% reduction in insulin AUC, but no change in glucose response, when 60 min of walking at 50% maximal O_2 uptake was performed in the fasted state, before an 8.5-h postprandial period, in overweight men. It is well established that even in individuals with insulin resistance, a single acute bout of aerobic exercise increases insulin sensitivity for up to 24 h (43). However, given the curvilinear relationship between exercise energy expenditure and ensuing improvements in insulin sensitivity, it is likely that the exercise conditions in the present study, which represent a realistic exercise stimulus for this population, were not sufficient to induce a change in insulin sensitivity (44).

Although there was no significant difference in exercise enjoyment between MICE and HIIE, participants did report a preference for HIIE, in addition to a greater fondness and exercise self-efficacy. Further, despite the higher %HR_{PEAK} achieved during the HIIE, levels of affective valence during exercise were similar compared with MICE. These findings largely support previous research in habitually active persons with chronic SCI who reported a greater preference and higher

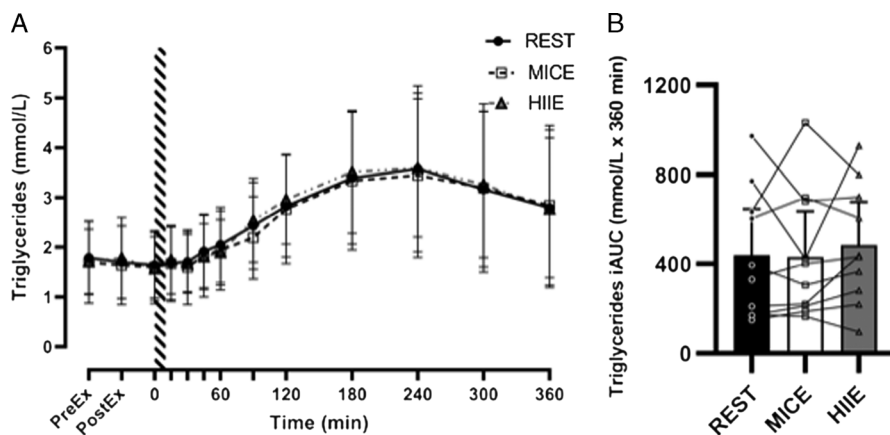


FIGURE 5—Serum concentrations of triglycerides (A) across each condition and iAUC (individual responses are also denoted) for serum triglycerides (B) across the 6-h postprandial period after consumption of the MMTT. The hashed box represents consumption of the meal.

enjoyment for HIIE compared with MICE and no differences in affective valence (19). Given that individuals are more likely to adhere to exercise that they enjoy and are confident they can perform (45), HIIE appears to be a viable training modality for persons with chronic SCI.

A significant strength of this study is that our sample of participants was representative of people with chronic paraplegia (i.e., physically inactive with poor metabolic health). It has been conservatively estimated that almost two thirds of individuals with chronic SCI have cardiometabolic syndrome (46), and in the current study, 9 of the 10 participants would be classified as having this condition. In addition, the macronutrient content of the MMTT reflected the habitual diet of persons with SCI (21) and allowed for triglyceride concentrations to peak at 4–5 h after meal consumption without participants being in a large energy deficit across the trial day. Finally, the exercise protocols were matched for total time commitment and represented realistic and achievable exercise sessions for this population, which closely match the SCI exercise guidelines (5,19). For example, in a free-living environment, persons with chronic paraplegia perform an average of just 17 min·d⁻¹ of moderate- to vigorous-intensity physical activity (47), which is less than both MICE and HIIE conditions. Therefore, we believe our findings have considerable real-world relevance.

The main limitation of this study is that despite the exercise protocols matching those previously characterized in this population (19), there was no difference in % $\dot{V}O_{2peak}$ between MICE and HIIE, and the %HR_{PEAK} achieved was only marginally higher for HIIE. It is possible that a more vigorous exercise intensity during the HIIE condition may have elicited changes in postprandial metabolism. In addition, we did not match the MICE and the HIIE conditions for energy expenditure, and therefore the total energy expenditure of MICE was ~30 kcal greater than HIIE. Third, because of participant dropout, we failed to reach our target sample size of 11; however, based on the observed effect size ($d = 0.04$) for our primary outcome (triglyceride TAUC) between HIIE and REST, one extra participant would not have meaningfully changed our findings regarding postprandial triglyceride responses. Finally, the MMTT contained a large bolus of calories (1037 ± 148 kcal), which is not typically consumed in a habitual diet. Although a more ecologically valid approach would have been to study responses to a typical breakfast and lunch meal, the total energy consumed ensured that participants' resting energy requirements were met.

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There remain large knowledge gaps with regard to the effect of a single bout of upper-body exercise on postprandial metabolism in persons with chronic SCI. To address this, future studies should assess the effect of exercise performed the evening before an MMTT, as the activity of the enzyme (lipoprotein lipase) believed to be primarily responsible for exercise-induced reductions in postprandial triglycerides peaks at 8 h postexercise in skeletal muscle (48). However, we speculate that any localized activation of lipoprotein lipase is unlikely to result in a reduction in the postprandial triglyceride response, given the limited active muscle mass involved in upper-body exercise. In addition, both HIIE and MICE performed in the postprandial state appear to improve 24-h glucose profiles in a free-living environment in noninjured populations (49,50), and it would be useful to understand if this effect is still present in persons with chronic SCI. Finally, we only recruited individuals with paraplegia, but this work should also be expanded to individuals with tetraplegia, who experience an early onset of exercise fatigue due to cardiovascular impairments.

CONCLUSIONS

After an overnight fast, acute upper-body exercise is not sufficient to improve subsequent postprandial responses to a large mixed-macronutrient meal in persons with SCI, irrespective of exercise intensity. This is likely due to the substantially lower active muscle mass and consequently reduced energy expenditure that can be achieved during upper-body exercise compared with whole and/or lower-body exercise. These findings highlight the need to identify alternative strategies to stimulate postprandial substrate oxidation in this population, including maximizing exercise energy expenditure (e.g., combining upper-body exercise with functional electrical stimulation cycling and/or resistance training), combining exercise with dietary restriction, or performing regular bouts of activity throughout the day.

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