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Original Research Article

A Comparative Assessment of the Effect of an Endurance Activity on Cardiac and Metabolic Markers in Persons with and Without Spinal Cord Injury: An Analytical Study

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Abstract:

Aim: The aim of the present study was to check effects of an endurance activity on cardiac and metabolic markers in persons with and without spinal cord injury.

Methods: The present study was conducted at Department of Physical medicine and Rehabilitation. Individuals were considered eligible if they met the following criteria: (a) diagnosed with a traumatic SCI at the lower cervical, thoracic, and upper lumbar level (C5-L2); (b) classified as A, B, C, D (motor and sensory complete or incomplete) on the American Spinal Injury Association (ASIA) Impairment Scale (AIS); and (c) >3 years post injury. Individuals with cardiovascular disease, renal disease, or orthopedic problems were considered ineligible. The study was approved by the Institutional Review Board at a large university medical center.

Results: There were no significant differences between groups on any baseline measures. With the exception of a significant time effect for QUICKI, there were no other significant differences between groups on body composition and cardiometabolic markers. However, there was a significant Group x Time interaction for arm fat percent. There was a significant time effect for chest press and lateral pulldown.

Conclusion: No differences between two groups were observed. Both conditions led to improvements in insulin sensitivity, aerobic capacity, muscle strength, and blood lipids in individuals with SCI. Future larger cohort studies are needed.

Keywords: Spinal Cord Injury, Cardiac Rehabilitation, Exercise Training, Cardiovascular, Autonomic.

Introduction

Spinal cord injury (SCI) is currently estimated to be prevalent in over two million individuals worldwide. [1] Spinal cord injuries (SCI) occur as a result of damage to the spinal cord, which has wide-ranging negative effects that depend on the severity of damage and level of lesion. [2] Individuals with chronic SCI often experience severe muscle atrophy and increased adiposity and have increased risk of developing cardiometabolic diseases, such as obesity, dyslipidemia, insulin resistance, and type 2 diabetes, compared to age- and weight-matched nondisabled individuals. [3] Symptoms can range from partial to complete loss of sensation or muscular control over the trunk, legs, and arms as well as impairments of autonomic functions (e.g., cardiovascular control, temperature regulation, bladder and bowel control) and/or breathing. [4,5]

Impairments in physical functioning, environmental and psychological barriers typically affect the engagement in physical activity after the injury and approximately 50% of individuals with SCI are engaged in inactive lifestyle (as compared to around a quarter of adults in general population) [6-9] Reduced physical activity coupled with loss of somatic and autonomic control in SCI cause reduced cardiorespiratory fitness, detrimental changes in body composition and metabolic profile and lead to worsening of cardiometabolic disease (CMD) risk profile following the SCI. [10-12]

The benefits of exercise and physical activity for delaying and in many instances reversing health conditions associated with cardiometabolic diseases are well known in nondisabled [13] and disabled persons, [14] however, two-thirds of the SCI population remain physically inactive. [15] Thus, it is important to identify novel exercise strategies that can not only improve health outcomes but also lead to greater exercise adherence in individuals with SCI. [16]

Hence, while SCI individuals may meet the advised weekly exercise hours some may not be able to reach the 40–59% of peak oxygen uptake that is considered a bare minimum to observe health benefits. [17] Similarly, individuals who meet the recommended relative exercise intensity may achieve up to fourfold lower energy expenditure as compared to able-bodied individuals. [18]

Hence the aim of the study was to check effects of an endurance activity on cardiac and metabolic markers in persons with and without spinal cord injury.

Materials & Methods

The present study was conducted at Department of Physical medicine and Rehabilitation, PMCH, Patna, Bihar, India for 48 months. Individuals were considered eligible if they met the following criteria: (a) diagnosed with a traumatic SCI at the lower cervical, thoracic, and upper lumbar level (C5-L2); (b) classified as A, B, C, D (motor and sensory complete or incomplete) on the American Spinal Injury Association (ASIA) Impairment Scale (AIS); and (c) >3 years post injury. Individuals with cardiovascular disease, renal disease, or orthopedic problems were considered ineligible. The study was approved by the Institutional Review Board at a large university medical center. Subjects were randomly assigned to two groups (Group I and Group II) i.e, with spinal cord injury and without spinal cord injury.

Pre-training testing protocol

Eligible participants attended three baseline visits: Day 1, following an overnight fast, resting metabolic rate, body composition, and blood pressure were assessed; Day 2, an oral glucose tolerance test (OGTT) was performed and baseline blood samples were stored at -80ºC and analyzed for HDL, low-density lipoprotein (LDL), total cholesterol, and triglyceride levels; and Day 3, VO2peak was assessed using indirect calorimetry during a graded arm cycle ergometer test, and peak power was determined by the standard 30-second Wingate test on a Lode (The Netherlands) arm ergometer. Additionally, four one-repetition maximum (1RM) strength assessments were performed using the upper body.

Exercise Training

HIIT was performed on an electronically braked Lode arm ergometer. Participants performed a total of 20 minutes of exercise consisting of 4 minutes of arm crank exercise at 25% of HRR determined from the VO_2peak test, followed by 30 seconds at 50% of peak power obtained from the Wingate Test. This cycle was repeated four times ending with 2 minutes of recovery at 25% of HRR. Participants in the HIIT group exercised twice a week with at least 24 hours of rest between each training session. MIT was performed on a SCIFIT Arm Ergometer (SCIFIT; Tulsa, OK). MIT consisted of 30 minutes of continuous arm crank exercise at 55% of VO2peak as determined by the baseline VO2peak assessment. MIT participants exercised three times each week. Heart rate was recorded for each HIIT and MIT session. Heart rate was logged every 5 minutes during MIT and immediately after each 30-second work interval and 4-minute recovery period for HIIT.

Clinical Measures

Body Composition

Total body composition, including fat mass, lean mass, percent body fat, percent arm fat, and percent leg fat, were measured by dual-energy X-ray absorptiometry (DXA). Participants were lying supine with arms at their sides on a padded table. Scans were analyzed using ADULT software version 1.33 (Lunar Radiation).

Resting blood pressure

Resting blood pressure (systolic and diastolic) was taken by automatic auscultation (Omron Blood Pressure Monitor, model HEM-780; Omron Healthcare, Inc., Bannockburn, IL) while participants were seated in a wheelchair. Readings were taken after 12 hours of fasting between 7:00 and 9:00 a.m.

Resting energy expenditure

Resting energy expenditure (REE) was measured between 7:00 and 9:00 a.m. following a 12-hour overnight fast. Participants remained supine following the DXA measurement. Subjects remained awake in a quiet, dimly lit room while oxygen uptake and carbon dioxide production were measured continuously using a ventilated hood system. Oxygen uptake was measured using a computerized, open-circuit indirect calorimetry system (ParvoMedics). After a 10-minute equilibration period, the data from the remaining 20-minute steady state period was used for analysis. REE was generated from the ParvoMedics system from Weir equation 12. [19]

Oral glucose tolerance test and insulin sensitivity

An OGTT was performed on an in-patient basis at the Clinical Research Unit (CRU). Participants fasted overnight and arrived at the CRU between 7:00 and 9:00 a.m. The post training OGTT was performed at least 24 hours after the last exercise session. Prior to testing, a flexible intravenous catheter was inserted into the antecubital space of one arm. Within the first 20 minutes, two baseline blood samples were taken to establish basal glucose of the test, blood samples were immediately

centrifuged, separated for serum, and frozen at -80°C until analysis. Assays were performed in the Center for Clinical and Translational Sciences Metabolism Core. Serum glucose assays were performed on an automated glucose analyzer (Sirrus analyzer; Stanbio Laboratory, Boerne, TX), and serum insulin was measured using an immunofluorescent method with an AIA-600 II analyzer (TOSOH Bioscience, South San Francisco, CA) per manufacturers' instructions. Insulin sensitivity was calculated using Quantitative Insulin Sensitivity Check Index (QUICKI). [20] QUICKI was calculated as 1/[log glucose (mg/dL) + log insulin (μU/mL)]. Insulin resistance (IR) was assessed using the Homeostasis Model of Assessment of Insulin Resistance (HOMA-IR). HOMA-IR was calculated as [fasting insulin (μU/mL) x fasting glucose (mmol/L)]/22.5.

Blood Lipids

Laboratory analyses took place in the Core Laboratory of CRU, Nutrition Obesity Research Center, and Diabetes Research Center. Total cholesterol, HDL, and triglycerides were assessed in serum using an automated glucose analyzer (Sirrus analyzer; Stanbio Laboratory, Boerne, TX). The Friedwald method was used to calculate LDL values. [21]

Strength Assessment

Muscle strength was determined using a standard 1RM protocol. The maximum load that could be lifted one time was measured for chest press, overhead press, lateral pull down, and tricep extension (Cybex VR3 for chest press, overhead press, and lateral pulldown; and Free Motion EXT Dual Cable for tricep extension). 1RM was determined after subjects lifted progressively heavier loads through full range of motion until failure occurred. and insulin. At time 0, the patient was instructed to drink a 75-g oral glucose load within 5 minutes.

Peak oxygen uptake (VO2peak) After consumption, blood samples were taken at 10, 20, 30, 60, 90, and 120 minutes to assess plasma glucose and plasma insulin. Following completion Peak aerobic assessment, which determined aerobic capacity, was defined by a VO2peak test on a Lode arm cycle ergometer. Subjects were instructed to cycle on an arm crank ergometer at 10W for 2 minutes. Every 2 minutes thereafter, power output was increased by 10W until voluntary fatigue. Due to the variability of heart rates after SCI, VO2peak was determined by (a) volitional exhaustion, (b) failure to maintain 60-65 revolutions per minute (RPM), (c) RER ≥ 1.10, and (d) rate of perceived exertion (RPE) >18 using the 6-20 Borg scale. [22] Minute ventilation, oxygen uptake, and carbon dioxide production were continuously analyzed and recorded via open circuit spirometry (ParvoMedics).

Peak Anaerobic Power Assessment

Peak power was determined by the standard 30-second Wingate protocol on a Lode arm cycle ergometer. Participants were seated in front of the Lode ergometer and remained seated throughout the entire test. Prior to each test, participants completed a 5-minute warm up at 25 W, which included three short sprint efforts followed by a 5-minute recovery period. Following the warm up, participants were instructed to hand cycle as fast as possible. Verbal encouragement was given to all participants to maintain their highest possible cadence throughout the test. The resistance was determined by the body weight (0.075 kg/kg body weight), with data collected and analyzed using the Monark Anaerobic Test software. Peak power, mean anaerobic power, fatigue rate, and relative peak power were recorded following the test.

Statistical Analysis

Statistical analyses were performed using SAS, version 9.4 (SAS Institute, Inc., Cary, NC). Descriptive statistics were computed for each group at baseline and post exercise training. A mixed-model, repeated measures analysis of variance (ANOVA) was used to assess the effects of time (pre and post intervention), group, and Group x Time interactions for each variable of interest. A compound symmetry covariance matrix was assumed for these analyses. Due to the pilot nature of this study and the small sample size, post hoc comparisons were performed without adjustments for multiple pairwise comparisons. Statistical tests were two-sided, and $p < .05$ was deemed statistically significant.

Results

Table 1: Baseline descriptive data between groups

Parameters	Group I	Group	p-value
Age (years)	48.4 ± 12	52.4 ± 1.3	0.830
Height (cm)	175.5 ± 13.7	177.7 ± 12.8	0.982
Body weight (kg)	79.3 ± 22.8	88.4 ± 22.4	0.560
BMI (kg/m^2)	24 ± 4.5	28.2 ± 12.8	0.424
Lean mass (kg)	46.4 ± 14.6	48.2 ± 4.6	0.940
Fasting glucose (mg/dL)	106.4 ± 17.4	150 ± 18.4	0.314
Fasting insulin (mg/dL)	18.1 ± 11.8	21.6 ± 25.5	0.801
HOMA-IR	4.9 ± 3.3	12.5 ± 18.8	0.387

Cholesterol (mg/dL)	154 ± 37.3	174 ± 38.6	0.509
Triglycerides (mg/dL)	83.4 ± 23.4	97.3 ± 61.7	0.654
HDL (mg/L)	52 ± 9.4	57 ± 9.8	0.501
LDL (mg/dL)	88.2 ± 32.8	102.4 ± 38.2	0.668
SBP (mm Hg)	120 ± 14	125 ± 26.9	0.735
DBP (mm Hg)	67.7 ± 8.2	73.3 ± 15.5	0.516
VO_{2peak} (mL/kg/min)	13.5 ± 5.5	11.5 ± 2.6	0.588

There were no significant differences between groups on any baseline measures.

Table 2: Changes in body composition and cardiometabolic health markers by intervention group

	Group I		Group II		G	T	GxT
	Baseline	**6 weeks post**	**Baseline**	**6 weeks post**	***p***	***p***	***p***
Body weight (kg)	88.6 ± 24.3	85.5 ± 24.4	93.7 ± 23.4	93.8 ± 21.6	0.780	0.412	0.438
Fat (%)	26.6 ± 4.3	26.45± 4.8	32.2 ± 12.8	32 ± 12.8	0.542	0.365	0.589
Fat mass (kg)	33.7 ±10	32.7 ± 9.3	41.8 ± 24.6	41.8 ± 24.7	0.544	0.360	0.362
Lean mass (kg)	50.9 ± 13.7	50.8 ± 13.6	49.1 ± 4.4	47.5 ± 4.9	0.680	0.333	0.392
Arm fat (%)	25.9 ± 3.3	25.9 ± 3.4	37.9 ± 14.1	36.1 ± 13.9	0.175	0.061	0.043
Leg fat (%)	39.6 ± 8.3	39.7 ±8.7	45.4 ± 18.5	44.8 ± 19.8	0.634	0.657	0.540
QUICKI	0.3007 ± 0.02	0.3226 ± 0.01	0.3208 ±0.07	.3555 ± 0.04	0.374	0.044	0.560
HOMA-IR	5.9 ± 2.7	3.4 ± 0.63	12.5 ± 18.9	1.8 ±1.0	0.525	0.074	0.572
Glucose (mL/dL)	109.9 ±18.5	108.4 ±17.5	154 ±102.8	135.7 ±80.4	0.472	0.134	0.188
Insulin (mL/dL)	21.8 ±9.9	12.1 ±2.9	22.6 ±25.5	5.9 ±2.3	0.674	0.112	0.671
Cholesterol (mg/dL)	161 ±43	146.5 ±46.2	177.7 ±38.5	173.3 ±29.8	0.482	0.478	0.696
HDL (mg/dL)	48.5 ±6.0	46.8 ±3.4	57 ±9.8	57 ±10.8	0.134	0.740	0.737
LDL (mg/dL)	96.8±36.4	82 ±42.3	101.2 ±38.6	96.4 ±21.2	0.686	0.427	0.699
TRG (mg/dL)	88.2 ±22.4	94.5 ±20.8	97.3 ±61.7	99.7 ±45.6	0.816	0.648	0.893
SBP (mm Hg)	118.5 ±15.7	115 ±23.1	125 ±26.9	119.7 ±25.7	0.738	0.120	0.880
DBP (mm Hg)	66.4 ±8.6	67.3 ±15.4	73.3 ±15.5	71.2 ±15.4	0.572	0.775	0.724
REE (kcal/day)	1951 ±612	1951 ±879	1840 ±388	1718 ±462	0.723	0.745	0.752

With the exception of a significant time effect for QUICKI, there were no other significant differences between groups on body composition and cardiometabolic markers. However, there was a significant Group x Time interaction for arm fat percent.

Table 3: Changes in aerobic capacity, anaerobic power, and muscular strength by intervention group

	Group I		Group II		G	T	GxT
	Baseline	**6 weeks post**	**Baseline**	**6 weeks post**	***p***	***p***	***p***
$VO_{2\ peak}$ (mL/kg/min)	14.2 ± 6	15.3 ± 7.3	11.5 ± 2.6	12.8 ± 1.3	0.634	0.048	0.348
Peak power (watts)	348 ± 132	376 ± 74	278 ± 71	308 ± 77	0.316	0.456	0.996
Relative PP (watts)	4.0 ±1.6	4.4 ± 0.87	2.9 ± 0.35	3.3 ± 0.78	0.190	0.408	0.918
Overhead press (kg)	46.4 ± 17.8	48.5 ± 22.2	37.9 ± 9.2	39.4 ± 4.7	0.541	0.268	0.680
Tricep extension (kg)	23.4 ± 7.6	31.8 ± 5.7	34.1 ± 16.1	38.6 ± 9.6	0.299	0.166	0.454
Chest press (kg)	75.4 ± 27.8	75.3 ± 29.7	56.1 ± 21.8	63.6 ± 20.2	0.473	0.036	0.036
Lat pulldown (kg)	44.8 ± 12.6	54.2 ± 18	36.4 ±8.2	47.3 ± 13.8	0.514	0.023	0.510

There was a significant time effect for chest press and lateral pulldown.

Discussion

Individuals with chronic SCI often experience severe muscle atrophy and increased adiposity and have increased risk of developing cardiometabolic diseases, such as obesity, dyslipidemia, insulin resistance, and type 2 diabetes, compared to age- and weight-matched nondisabled individuals. [23-26] The benefits of exercise and physical activity for delaying and in many instances reversing health conditions associated with cardiometabolic diseases are well known in nondisabled [14] and disabled persons, [15] however, two- thirds of the SCI population remain physically inactive. [16] Thus, it is important to identify novel exercise strategies that can not only improve health outcomes but also lead to greater exercise adherence in individuals with SCI.

There were no significant differences between groups on any baseline measures. With the exception of a significant time effect for QUICKI, there were no other significant differences between groups on body composition and cardiometabolic markers. However, there was a significant Group x Time interaction for arm fat percent. There was a significant time effect for chest press and lateral

pulldown. One study involving nondisabled individuals demonstrated similar improvements in glucose tolerance between traditional moderate-intensity exercise and low-volume HIIT training. [27] In addition, HIIT has been shown to significantly improve insulin sensitivity in as little as 2 weeks in healthy adults [28] and potentially has similar long- term (>24 hours) benefits as MIT. [29] In research on HIIT in SCI populations, Hasnan et al showed no improvements in oral glucose tolerance after 6 weeks of hybrid arm and leg exercise suggesting that 6 weeks of training may not be a sufficient dose (ie, duration) to improve glucose tolerance in individuals with SCI. [30] However, Jeon et al showed favorable results for both glucose tolerance and insulin sensitivity using electrical stimulated-assisted cycling in individuals with SCI. Glucose tolerance improved for all seven subjects while insulin sensitivity improved in two out of three subjects tested on this measure. [31]

SCI generally leads to significant skeletal muscle atrophy below the level of injury. Skeletal muscle atrophy of 27% to 56% has been observed in SCI participants from 6 to 24 weeks post injury with a cross-sectional area of only one-third that of able-bodied controls. [32] Electrical stimulation has been shown to improve muscular strength. Both resistance and endurance training with the assistance of NMES or FES in individuals with chronic SCI have demonstrated significant increases in muscle hypertrophy in the lower body [33] and overall muscular strength in the upper body, specifically the shoulder flexors, extensors, abductors, and adductors. [34] Additionally, high-intensity aerobic arm cycling of 90 minutes per week is enough to improve upper extremity muscular strength in individuals with tetraplegia. [35] Regular moderate-to-vigorous physical activity is associated with less visceral adipose tissue in persons with SCI. [36] Additionally, endurance training has been shown to improve body composition in this population. [37,38] While the present study observed no significant changes in fat mass, fat-free mass, or percent body fat, we did obtain a greater reduction of arm fat % following MIT compared to HIIT. Overall, 6 weeks is a relatively short training duration to see significant changes in body composition.

Conclusion

No differences between two groups were observed. Both conditions led to improvements in insulin sensitivity, aerobic capacity, muscle strength, and blood lipids in individuals with SCI. Future larger cohort studies are needed.

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