ORIGINAL ARTICLE

Glucose tolerance and physical activity level in people with spinal cord injury

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Study design: Cross-sectional, observational study.

Objectives: To evaluate the associations of physical activity and neurological lesion level with glucose tolerance in people with spinal cord injury (SCI).

Setting: New South Wales, Australia.

Methods: Twenty-five people (5 women, 20 men) with SCI (>6 months post-injury) aged between 18 and 65 years were recruited. Exclusion criteria included known coronary heart disease, stroke or diabetes. Participants underwent an oral glucose tolerance test. Fasting and 2-h plasma glucose concentrations were classified according to the World Health Organization categories of glycemia. Participants also completed the Physical Activity Scale for Individuals with Physical Disabilities and mean MET-hours day^{-1} was calculated. Associations with the 2-h plasma glucose concentration were calculated through multiple and stepwise regressions.

Results: Participants presented with complete or incomplete tetraplegia ($n=11$ TETRA) or complete or incomplete paraplegia ($n=14$ PARA) with neurological lesion levels ranging from C3/4 to T12. Mean 2-h plasma glucose was $7.13 \pm 2.32 \, \text{mmol l}^{-1}$. Nine participants had disordered glycemia ($n=6$ TETRA; $n=3$ PARA) and the remaining participants had normal glucose tolerance. Those participants with normal glucose tolerance participated in more moderate-vigorous and strength exercise and undertook more non-exercise-related mobility than those with disordered glycemia. Physical activity and age, but not lesion level were independent determinants of 2-h plasma glucose concentration ($r=0.683$, $P=0.001$), explaining 47% of the variance.

Conclusion: Physical activity level is independently associated with glucose tolerance in people with SCI. Non-exercise activity may also be important for maintaining normal glycemia.

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Introduction

Disordered carbohydrate metabolism, evidenced by impaired glucose tolerance (IGT), insulin resistance and type II diabetes mellitus, is more prevalent in people with spinal cord injury (SCI), and is thought to appear at a younger age than in able-bodied individuals.[1,2] It has been suggested that the extent of neurological impairment after SCI is an important determinant of these impairments; participants with complete tetraplegia (TETRA) had higher levels of serum glucose and plasma insulin during a 2-h oral glucose tolerance test (OGTT) and higher incidence of IGT or diabetes than participants with either incomplete TETRA or complete or incomplete paraplegia (PARA).[3]

The suggestion that greater neurological deficit is associated with poorer glucose tolerance may be explained by the consequences of denervation such as skeletal muscle atrophy[4,5] and altered fiber-type proportions,[6,7] with the volume of muscle affected related to level and completeness of the neurological lesion. Skeletal muscle is the major site for insulin-mediated glucose disposal and glucose transport is rate limiting for glucose utilization under most conditions.[8] Thus, the elevated glycemic response to an oral glucose load that is evident in some people with SCI[1,4] may be partially explained by fewer sites for glucose uptake consequent to muscle atrophy.

A second consequence of SCI that might also contribute to the prevalence of diabetes and its precursors is reduced level of physical activity. Manns et al. (2005)[9] found an inverse relationship between physical activity and fasting glucose, although there was no relationship been physical activity and 2-h glucose after an OGTT or markers of insulin

resistance, including fasting insulin and the homeostasis model assessment. In addition, Bauman and Spungen (1994)[1] found that aerobic capacity was related to insulin sensitivity in participants with PARA. However, it is unknown whether in these relationships, physical activity and fitness levels are simply surrogate measures of neurological impairment. That is, those with higher lesions are less physically active by nature of their impairment, and as such it is the neurological impairment, which underpins the physical activity/fitness relationships observed by others. Yet, it is important to establish the effect of physical activity independent of neurological impairment because physical activity and fitness are modifiable, either through voluntary or electrically stimulated exercise. The purpose of this study was to determine the independent roles of physical activity and neurological impairment on glucose tolerance in participants with SCI.

Materials and methods

Participants
Twenty-five participants, 5 women and 20 men, with SCI were recruited through website and newspaper advertisements or word-of-mouth. Participants were included if they were aged between 18 and 65 years, had a SCI, and were >6 months post-injury. Participants were excluded if they had known coronary heart disease, stroke or diabetes. There was no restriction on neurological lesion level or severity or on physical activity levels for this study as we sought representation across a broad range of injury and activity levels. The study protocol was approved by the University of Sydney human research ethics committee and informed consent was obtained from all participants.

Measurements
Participants attended the laboratory on one occasion, when the following measurements were made:

Glucose tolerance (dependent variable). Plasma glucose response to a 75 g oral glucose load was determined after a 12-h fast. Before commencing the OGTT, a flexible catheter was placed in a forearm vein and kept patent by periodic instillation of saline. Anhydrous glucose (75 g) was mixed with 300 ml water, and the participant consumed this drink within 2 min. Two-milliliter blood samples were drawn twice before glucose loading and then at 30, 60, 90 and 120 min after glucose ingestion. Blood samples were collected in heparinized syringes (Pico 50 syringes, Radiometer Medical, Copenhagen, Denmark) and stored briefly on ice before being assayed for glucose concentration using an automated analyzer (EML105, Radiometer Medical, Copenhagen, Denmark). Participants' fasting and 2-h plasma glucose concentrations were classified according to the World Health Organization's categories of glycemia.

Physical activity level (independent variable). Physical activity level was determined using the physical activity scale for individuals with physical disabilities (PASIPD).[10] The PASIPD examines both planned and incidental activity, level of intensity and work- and household-related tasks over the past 7 days. The number of days and the length of time the person was involved in each type of activity in the past week were used to calculate a metabolic equivalent, expressed as MET-hours day^{-1}.

The independent variables of gender, age, age at injury, neurological level and completeness of lesion, duration of injury, use of medications, family history of coronary heart disease and diabetes, number of standard alcoholic drinks consumed per day, number of cigarettes smoked per day, resting blood pressure, height (measured while supine) and body mass were also recorded. Participants were categorized according to lesion level into one of three categories: participants with TETRA, participants with thoracic lesions between T1 and T5 (HIGH PARA) and participants with lesions at T6 or lower (LOW PARA).

Data analysis
All data were visually inspected for normality of distribution. Non-normally distributed variables were log-transformed before analysis. One-way analysis of variance was used to examine the effect of lesion level category (TETRA vs HIGH PARA vs LOW PARA). Univariate regression analyses were used to examine relationships between the dependent variables (fasting plasma glucose and 2-h plasma glucose) and independent variables of interest. To identify overall and independent contributors to fasting plasma glucose and 2-h plasma glucose, multiple and stepwise regression models were constructed using those variables with a significant univariate association ($P < 0.15$) and age.

To further analyze the role of physical activity, we undertook a secondary analysis to examine the influence of different types of activity. First, we scored the average number of hours per day each participant was involved in moderate intensity exercise, vigorous intensity exercise, strength exercise, outdoor household work, housework and non-exercise mobility. The average number of hours per day was derived from the time estimates provided in the PASIPD.[10] For this secondary analysis, we performed regression modeling as well as compared participation in different types of activities between those participants with normal glucose metabolism (2-h plasma glucose < 7.8 mmol l^{-1}), and those with abnormal glucose metabolism (2-h plasma glucose $\geqslant 7.8$ mmol l^{-1}) using Mann–Whitney U-tests.

All data are presented as mean ± s.d. unless otherwise indicated. Statistical significance was set at $P < 0.05$ (except for univariate associations where $P < 0.15$). Statistical analyses were performed using SPSS (version 15).

We certify that all applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during the course of this research.

Results

Participants
Participants' lesion levels ranged from C3/4 to T12. In all, 4 participants were TETRA with complete lesions, 7 were

TETRA with incomplete lesions, 11 were PARA with complete lesions and 3 were PARA with incomplete lesions. Six participants were smokers (range 5–15 cigarettes per day) and 15 participants consumed alcohol (range 1–20 standard drinks each week). Six participants reported a family history of at least one close relative with either coronary heart disease or diabetes. One participant did not know his family history as he was adopted as a child. Participant characteristics are presented in Table 1.

Oral glucose tolerance test

Mean fasting plasma glucose was 4.76 ± 0.38 mmol l^{-1} and mean 2-h plasma glucose was 7.13 ± 2.32 mmol l^{-1}. According to the World Health Organization classification, 2 participants had diabetes mellitus on the basis of their OGTT, 7 had IGT and the remaining 16 participants had normal glucose tolerance. No participant showed impaired fasting glycemia. Of the participants classified as having either IGT or diabetes mellitus, six were TETRA and three were PARA. Figure 1 presents the 2-h plasma glucose data from each participant according to their lesion level.
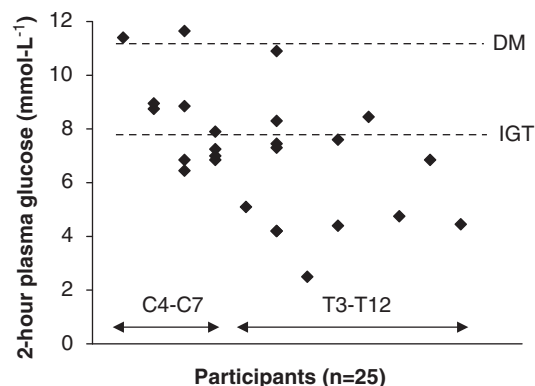
Physical activity levels

Mean PASIPD score was 12.2 ± 10.8 MET-hours day^{-1}, with the range being 0–46.9 MET-hours day^{-1}. There was no significant difference in PASIPD scores between the three lesion level categories (that is, TETRA vs HIGH PARA vs LOW PARA) (Table 1). Figure 2 presents the PASIPD score for each participant according to their lesion level. There were two outliers with respect to PASIPD score. One was a 35-year-old male with a T3-6 complete lesion, an unknown family history, a fasting plasma glucose of 4.90 mmol l^{-1} and a 2-h plasma glucose of 5.10 mmol l^{-1}. The other was a 30-year-old male with a T12 incomplete lesion, a negative family history, a fasting plasma glucose of 5.05 mmol l^{-1} and a 2-h plasma glucose of 4.45 mmol l^{-1}. The first participant was a national level wheelchair basketballer, undertook dance as part of a dance company, and also performed general exercise training. The second participant undertook both strength and endurance training three times each week and had a non-sedentary occupation. With these two outliers removed, there was still no significant

difference in PASIPD scores between the three lesion level categories.

Factors contributing to fasting and 2-h glucose

Physical activity level and lesion level category (TETRA vs HIGH PARA vs LOW PARA) were the only independent

Figure 1 Two-hour plasma glucose data for each participant according to their lesion level. Dotted lines represent diagnostic criteria for impaired glucose tolerance (IGT) and type II diabetes mellitus (DM).
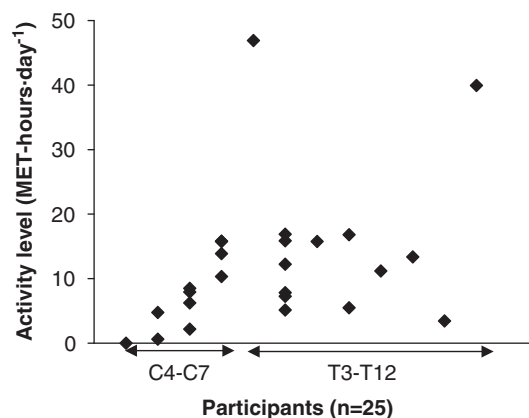
Figure 2 Physical activity levels for each participant according to their lesion level.

Table 1 Participant characteristics presented as a group (All) and on the basis of lesion level category (TETRA; HIGH PARA; LOW PARA)

	All (n = 25)	TETRA (n = 11)	HIGH PARA (n = 8)	LOW PARA (n = 6)
Age (years)	37 ± 9	34 ± 6	36 ± 7	43 ± 13
Height (m)	1.72 ± 0.15	1.77 ± 0.09	1.71 ± 0.06	1.65 ± 0.27
Weight (kg)	69.5 ± 16.1	71.0 ± 12.2	64.5 ± 15.7	73.4 ± 23.1
Body mass index (kg m^{-2})	23.5 ± 4.8	22.7 ± 3.6	22.0 ± 4.5	27.1 ± 5.8
Time since injury (years)	11.0 ± 7.6	12.9 ± 7.6	7.8 ± 4.9	11.9 ± 10.1
No. of medications	2.1 ± 1.7	2.5 ± 1.8	1.9 ± 1.8	1.7 ± 1.8
Resting systolic blood pressure (mm Hg)	111.7 ± 18.1	99.1 ± 15.9[a]	118.1 ± 12.9	126.3 ± 12.8
Resting diastolic blood pressure (mm Hg)	76.2 ± 11.6	69.7 ± 12.6	81.0 ± 10.0	82.0 ± 4.9
PASIPD (MET-hours day^{-1})	12.2 ± 10.8	7.8 ± 5.7	16.0 ± 13.3	15.0 ± 13.2
Fasting glucose (mmol l^{-1})	4.76 ± 0.38	4.78 ± 0.34	4.69 ± 0.29	4.83 ± 0.56
2-h glucose (mmol l^{-1})	7.13 ± 2.32	8.35 ± 1.80	6.24 ± 2.73	6.08 ± 1.78

Abbreviations: PARA, paraplegic; PASIPD, physical activity scale for individuals with physical disabilities; TETRA, tetraplegic.
[a]TETRA significantly different to HIGH PARA ($P = 0.024$) and LOW PARA ($P = 0.003$).

variables with significant ($P<0.15$) univariate associations with 2-h plasma glucose ($r=0.575$, $P=0.003$ for physical activity level; $r=0.483$, $P=0.054$ for lesion level category). A multiple regression model was constructed with these two independent variables and age. These three variables combined to explain 48% of the variance in 2-h plasma glucose ($r=0.691$, $P=0.003$). When these variables were entered in a stepwise multiple regression model, only physical activity level and age were independent contributors to 2-h plasma glucose, explaining 47% of the variance ($r=0.683$, $P=0.001$). To ascertain whether severity of lesion affected the outcome, we conducted the same analyses after categorizing participants according to level and severity of lesion. The outcomes of the regression modeling were similar when using 2-h plasma glucose as the dependent variable, with physical activity level and age being independent contributors ($r=0.686$, $P=0.001$). Figure 3 presents a graphical summary of the relationship between physical activity and 2-h plasma glucose across neurological lesion. This figure shows that among TETRA participants, those who are more active tend to have lower 2-h plasma glucose levels than those who are not (on average, 7.25 ± 0.46 mmol l^{-1} for more active TETRA vs 8.99 ± 2.00 mmol l^{-1} for less active TETRA). A similar observation exists for PARA participants (on average, 5.33 ± 1.90 mmol l^{-1} for more active PARA vs 7.58 ± 2.36 mmol l^{-1} for less active PARA).

We examined the components of physical activity further to determine whether certain volumes or intensities of activity were more important for determining glucose
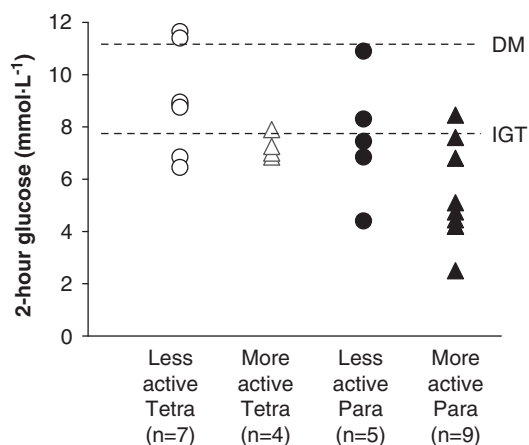
Figure 3 Two-hour plasma glucose data for each participant according to lesion level (TETRA, tetraplegic vs PARA, paraplegic) and activity level, in which activity level (more active vs less active) was determined using the median score from the PASIPD for all participants. Those classified as 'more active' (PASIPD score $\geqslant 10.32$ MET-hours day^{-1}) typically participated in more moderate-vigorous exercise (on average 9 h week^{-1} vs 2 h week^{-1}) and accumulated more mobility-related, non-exercise activity (11 h week^{-1} vs 4 h week^{-1}). Only three of the less active participants (PASIPD score $\leqslant 8.51$ MET-hours day^{-1}) reported engaging in any housework or home duties, whereas 12 out of 13 of those who were more active undertook such activities. Dotted lines represent diagnostic criteria for impaired glucose tolerance (IGT) and type II diabetes mellitus (DM). It is to be noted that some data points are superimposed. In the Less Active Tetra group, there were three participants with values of 8.75, 8.85 and 8.95, and in the More Active Para group, there were two participants with the same value of 4.2.

tolerance. In the group with normal glucose tolerance, there were five TETRA, six HIGH PARA and five LOW PARA. In the group with abnormal glucose tolerance, there were six TETRA, two HIGH PARA and one LOW PARA. Using χ^2 analysis, the association between participants with normal vs abnormal glucose tolerance and the three lesion level categories was not significant ($\chi^2 = 3.04$; $P=0.219$).

As a group, participants with normal glucose tolerance spent significantly more time undertaking outdoor household activities, housework and non-exercise-related mobility tasks (Table 2). In total, 9, 9 and 16 participants in the normal glucose tolerance subgroup ($n=16$) reported undertaking outdoor household activities, housework and non-exercise-related mobility tasks, respectively, in the previous week. This compares with zero, one and seven participants, respectively, in the abnormal glucose tolerance group ($n=9$). Participants with normal glucose tolerance also spent significantly more time engaged in moderate or vigorous intensity or strength-related exercise (Table 2). In all, 15 out of 16 participants had participated in moderate-vigorous exercise or strength-related exercise in the previous week compared with 6 out of 9 participants in the group with abnormal glucose tolerance. Ten participants in the group with normal glucose tolerance (63%) spent at least 30 min day^{-1} on average engaged in exercise of at least moderate intensity, compared with three participants in the group with abnormal glucose tolerance (33%).

Discussion

This study shows that physical activity level is a stronger determinant of 2-h glucose concentrations than neurological lesion level in individuals with SCI. This is an exciting finding given that physical activity level is modifiable, and that disordered glucose metabolism is disproportionately prevalent in people with SCI compared with the able-bodied.[1,2] Previous studies in people with SCI have shown relationships between glycemia and neurological lesion level[3] and between fasting glucose and physical activity levels.[9] However, because lesion level may be associated with physical activity level,[11] it was not clear in these studies whether the influence of physical activity on disordered carbohydrate metabolism was independent of lesion level. The findings from this study show that physical activity level is a determinant of glucose tolerance, independent of the extent of neurological impairment.

Although 36% of our participants had either IGT or diabetes based on their 2-h OGTT, all participants had normal fasting plasma glucose. This discordance between indices of glucose metabolism has been observed by others,[1,4,12] is well documented in the able-bodied population,[13,14] and is likely because of the different metabolic determinants underlying impaired fasting glycemia and IGT. That is, IGT is thought to reflect peripheral insulin resistance, predominantly within skeletal muscle, whereas impaired fasting glycemia is thought to reflect hepatic insulin resistance and β-cell deficiency.[13]

The source of peripheral insulin resistance in our participants with abnormal glucose metabolism cannot be

Table 2 Comparison of time engaged in various types of activity between participants with normal glucose tolerance and participants with impaired glucose tolerance or diabetes

	Normal glucose tolerance (n = 16)	Abnormal glucose tolerance (n = 9)	P-value
Age (years)	39 (26–61)	37 (26–45)	0.609
Neurological level and completeness			
TETRA	n = 5 (2 inc, 3 com)	n = 6 (5 inc, 1 com)	
HIGH PARA	n = 6 (6 com)	n = 2 (1 inc, 1 com)	
LOW PARA	n = 5 (2 inc, 3 com)	n = 1 (1 inc)	
Time since injury (years)	9.5 (2.5–20.0)	12 (0.6–27.0)	0.977
No. of females	3	2	
Body mass index (kg m^{-2})	23.7 (17.3–32.1)	22.3 (16.2–33.5)	0.718
No. of smokers	3	3	
No. with positive family history	4 (and 1 unknown)	2	
Types of activities as recorded in the PASIPD			
Moderate intensity activities (h day^{-1})	0 (0–0.75)	0 (0–0.32)	0.369
Vigorous intensity activities (h day^{-1})	0.25 (0–4.29)	0 (0–0.75)	0.109
Strength activities (h day^{-1})	0.18 (0–1.5)	0 (0–0.43)	0.086
Moderate + vigorous + strength activities (h day^{-1})	0.72 (0–5.36)	0.25 (0–1.0)	0.023
Non-exercise mobility activities (h day^{-1})	1.29 (0.25–2.57)	0.32 (0–2.57)	0.044
Outdoor household activities (h day^{-1})	0.11 (0–1.07)	0 (0–0)	0.008
Housework activities (h day^{-1})	0.7 (0–6.97)	0 (0–0.86)	0.002

Abbreviations: PARA, paraplegic; PASIPD, physical activity scale for individuals with physical disabilities; TETRA, tetraplegic.
Continuous data are presented as median and (range). Non-continuous data are presented as counts, and in the case of neurological level and completeness, the number of participants within each lesion level category followed by the number of participants within that category who had either incomplete (inc) or complete (com) injuries. Statistical comparison between the two subgroups were conducted for continuous variables only. P-values are provided for these comparisons.

determined with our limited measurements. However, from a point of speculation, it is possible that insulin resistance may be present in muscles both below and above the lesion. In paralyzed muscle, characteristic changes include a marked decrease in cross-sectional area of both slow- and fast-twitch muscle fibers[6] and reduction in slow myosin heavy chain proportion, and concomitantly, a marked shift to fast myosin heavy chain isoforms;[7] and increased intramuscular fat accumulation.[4,5] No studies have compared GLUT4 content in those with and without SCI. However, in rodents, denervation reduces GLUT4 and contraction-stimulated glucose transport, and more potently reduces insulin-stimulated glucose uptake than severe (streptozotocin-induced) diabetes.[15] Innervated muscle above the level of the neurological lesion may potentially become insulin resistant because daily energy expenditure is lower in those with SCI than in able-bodied individuals.[16] Thus, both denervation and deconditioning may contribute to IGT and insulin resistance in those with SCI.

Deconditioning of innervated muscle is reversible and denervated muscle may be stimulated. Therefore, increasing daily energy expenditure by increasing physical activity has the potential to enhance glucose tolerance and lessen insulin resistance in people with SCI. This notion is supported by the cross-sectional data in this study, and by longitudinal studies of exercise training.[7,12,17] For example, electrical stimulation-induced leg cycling training increased expression of muscle fiber types I and IIa,[7] increased GLUT4 content[12] improved glucose tolerance and insulin sensitivity[17] and increased oxidative capacity.[7,12]

Interestingly, with regard to physical activity, our data showed that it was not only participation in exercise, but also in non-exercise-related activity that differed between those with normal glucose tolerance and those with disordered glycemia. Those with normal glucose tolerance engaged in more housework (median 0.7 h day^{-1}, range 0–6.97 h day^{-1}), with participants citing specific tasks, such as doing the washing, looking after young children and vacuuming. This subgroup also undertook more non-exercise-related mobility activities (median 1.29 h day^{-1}, range 0.25–2.57 h day^{-1}), with participants citing-specific activities, such as doing errands (for example, post office, supermarket) and pushing outside their home either with (for example, to go to a park) or without an explicit purpose. Non-exercise activity is important in preventing obesity[18] and the accumulation of intramuscular lipid is linked with insulin resistance and disordered glycemia[19]. Individuals with SCI characteristically evidence elevated intramuscular lipid[4,5] and intramuscular lipid is a good predictor of OGTT glycemia in this group.[4] Although our patients were not obese according to their respective body mass indexes, body fat mass is greater for any given body mass index in those with SCI.[20] Therefore, strategies that encourage non-exercise activity in addition to exercise training may also be important to improve glycemia and health in people with SCI.

In conclusion, while previous studies have shown a relationship between neurological level of lesion and glucose tolerance, the role of physical activity in this relationship had not been established. Yet, the independent role of physical activity level is important because physical activity is modifiable and therefore may have a role in prevention of diabetes. The main finding from this study is that physical activity level is an independent predictor of glucose response to a 2-h OGTT in people with SCI. Furthermore, our findings suggest that non-exercise activity may be an important factor in maintaining normal glycemia in those with SCI.

Conflict of interest

Acknowledgements

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