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Differences in Glucose Metabolism Among Women With Spinal Cord Injury May Not Be Fully Explained by Variations in Body Composition

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Abstract

Objective: To investigate the differences in glucose metabolism among women with paraplegic, and tetraplegic spinal cord injury (SCI) in comparison to their able-bodied (AB) counterparts after adjusting for differences in body composition.

Design: Cross-sectional study. After an overnight fast, each participant consumed a 75-g glucose solution for oral glucose tolerance test (OGTT). Blood glucose, insulin, and C-peptide concentrations were analyzed before and 30, 60, 90, and 120 minutes after ingesting glucose solution. Insulin sensitivity index (ISI) was estimated using the Matsuda index. Percentage fat mass (%FM) and total body lean mass (TBLM) were estimated using data from dual-energy x-ray absorptiometry. Visceral fat (VF) was quantified using computed tomography. Outcome measures were compared among groups using analysis of covariance with %FM (or VF) and TBLM as covariates.

Setting: Research university.

Participants: Women (N=42) with SCI (tetraplegia: n=8; paraplegia: n=14) and their race-, body mass index-, and age-matched AB counterparts (n=20).

Interventions: Not applicable.

Results: At fasting, there was no difference in glucose homeostasis (glucose, insulin, C-peptide concentrations) among 3 groups of women. In contrast, glucose, insulin, and C-peptide concentrations at minute 120 during OGTT were higher in women with tetraplegia versus women with paraplegia and AB women ($P<.05$, adjusted for TBLM and %FM). In addition, women with tetraplegia had lower ISI ($P<.05$, adjusted for TBLM and %FM) versus AB women. These differences remained after adjusting for VF and TBLM.

Conclusion: Our study confirms that impaired glucose metabolism among women with tetraplegia may not be fully explained by changes in their body composition. Future studies exploring additional factors involved in glucose metabolism are warranted.

Keywords

Body composition; Insulin resistance; Rehabilitation; Spinal cord injury; Tetraplegia

Individuals with spinal cord injury (SCI) experience metabolic disorders including but not limited to glucose intolerance, dyslipidemia, and obesity.^{1,2} These disturbances occur at younger ages and more frequently relative to the general population.³ Lifestyle and physiological changes associated with SCI predispose individuals with SCI to metabolic diseases, which are among the leading causes of mortality for this population.^{4,5} Understanding the progression of metabolic diseases could help identify points of intervention to increase the life expectancy and quality of life of individuals with SCI.

Body composition deterioration after SCI is proposed as a key element in the development of metabolic disturbances.^{1,6,7} Neurologic and hormonal changes associated with SCI lead to skeletal muscle atrophy and increase in adiposity.⁸ As a major tissue for regulating energy balance as well as insulin-mediated glucose uptake, skeletal muscle plays an important role for maintaining metabolic health.⁹ Muscle loss after SCI is associated with a reduction of basal metabolic rate^{10,11} and glucose intolerance.¹² In addition, excess body fat is negatively related to whole-body carbohydrate metabolism. For example, total body percent fat is positively associated with peak glucose concentrations during oral glucose tolerance tests (OGTTs) among individuals with SCI.¹³ Emerging data further revealed that increased visceral adiposity is negatively correlated with insulin sensitivity in men with paraplegia and tetraplegia.¹⁴

Individuals with tetraplegia (mainly C5-C8) have significantly lower glucose tolerance, lower insulin sensitivity compared with able-bodied (AB) controls and individuals with paraplegia (T1 and below).^{3,13,15,16} Even though associations between muscle and body fat with indices of metabolic health are commonly studied^{13,17} in the current literature, it is unclear whether the exacerbated metabolic impairment associated with higher injury levels can be fully attributed to changes in body composition observed with SCI.

In addition, the current understanding of body composition and metabolic profile after SCI is predominantly based on studies of men, and their applications to women with SCI are virtually unknown. In a 17-year prospective study, women with SCI had an increased frequency of developing metabolic syndrome than their male counterparts (50% vs 35.5%).¹⁸ Given the pronounced differences in body composition among women and men with SCI,¹⁷ it is important to investigate the relation between body composition and metabolic health in women with SCI. Research focusing on women with SCI would help address unanswered questions in the area of metabolism, nutrition, and metabolic-related disease while offering insight to direct future studies as to the type of interventions (nutritional, lifestyle, etc) best suited to protect them from elevated metabolic-related disease risk. Thus, the current study aimed to investigate the differences in glucose homeostasis (at fasting and during OGTT)

among women with SCI (paraplegia and tetraplegia) relative to age-, race-, and body mass index (BMI)-matched AB women after adjusting for differences in body composition.

Methods

Participant recruitment

Study participants were recruited from the University of Alabama at Birmingham (UAB) via mailings and community fitness centers using advertisement fliers. Participants were considered eligible if they met the following inclusion criteria: (1) between the ages of 18 and 60 years; (2) have SCI and residual motor disability severe enough to require use of an assistive device for locomotion; and (3) be generally healthy without a known diagnosis of major diseases, including myocardial infarction, angina pectoris, diabetes mellitus, and cancers. Exclusion criteria included pregnancy and any medical condition that required hospital care. All measurements were taken during the follicular phase of the menstrual cycle if participants are premenopausal and under controlled laboratory conditions at the UAB Clinical Research Unit. The study was approved by the UAB Institutional Review Board, and participants provided written consent prior to the study initiation. Study participants stayed and fasted overnight in the Clinical Research Unit. On the test day, anthropometric measures (height and weight) for BMI, OGTT, dual-energy x-ray absorptiometry (DXA) for body composition estimates, and computed tomography (CT) scanning for visceral adiposity were performed.

Oral glucose tolerance test

After a 10- to 12-hour overnight fast, each participant consumed a 75-g glucose solution within 5 minutes. Blood samples were collected immediately before and 30, 60, 90, and 120 minutes after glucose ingestion for measurement of plasma glucose, insulin, and C-peptide. Blood was immediately centrifuged, separated for plasma and frozen at -80°C until analysis. Assays were performed in the UAB Metabolism Core facility. Plasma glucose was analyzed using an automated glucose analyzer,^a and plasma insulin and C-peptide were measured using an immunofluorescent method with an AIA-600 II analyzer^b per manufacturers' instructions. C-peptide was measured to reflect insulin production quantitatively. Whole-body insulin sensitivity index (ISI) was estimated from OGTT data (time point 0, 60, 90, 120) using Matsuda index. The formula for calculating ISI:

$$\frac{10,000}{\sqrt{\text{fasting glucose} \times \text{fasting insulin} \times \text{glucose mean concentrations} \times \text{insulin mean concentrations}}}$$

This index correlates strongly ($r=0.73$, $P=.0001$) with the direct measure of insulin sensitivity derived from the euglycemic insulin clamp.¹⁹

^aSirrus analyzer; Stanbio Laboratory.

^bAIA-600 II analyzer; TOSOH Bioscience.

Body composition

Recumbent height was measured on the right side of the body from the top of the participant's head to heels while the participant was in a supine position with a nonstretch tape. The weight (Wt) of wheel chair and participant ($W_{t\text{chair+participant}}$) and weight of wheel chair by itself ($W_{t\text{chair}}$) were measured on a wheelchair scale. Participants were in light clothing, and body weight was obtained by subtracting $W_{t\text{chair}}$ from $W_{t\text{chair+participant}}$. AB women were weighed standing on the same wheelchair scale. BMI was calculated by dividing the body weight (kg) by height² (m^2). Total body fat mass (FM) and lean mass were estimated by DXA scans (Hologic QDR-4500W^c). Percentage fat was calculated using DXA results. The scans were analyzed with the use of ADULT software, Lunar DPX-L version 1.35.^d Total body lean mass index (TBLMI) was calculated by dividing total body lean mass (TBLM) by m^2 . Total body FM and trunk and appendicular (sum of 4 limbs) lean mass were estimated using DXA results.

Visceral adipose tissue

Cross-sectional area of visceral adipose tissue was estimated by CT with the use of a HiLight/HTD Advantage scanner.^e Participants were in the supine position with their arms stretched above their heads. A scan was taken (5-mm) for 2 seconds at approximately the level of the fourth and fifth lumbar vertebrae. Visceral fat (VF) was measured as the cross-sectional area of intraabdominal adipose tissue. Briefly, a computerized fat tissue-highlighting technique was used, where attenuation range for adipose tissue between -30 and -190 Hounsfield units was used to characterize intra-abdominal adipose tissue.²⁰ Additional details about the CT scanning were described elsewhere.²¹

Statistical analyses

Data were analyzed using Statistical Analysis Software (SAS version 9.4).^f In our preliminary analysis, data normality was visually confirmed. Indices of body composition (FM, VF, TBLM, TBLMI) were compared between complete and incomplete SCI using *t* test and no significant differences were found ($P > .05$ for all). As a result, no additional adjustments for completeness of injury were performed in the following analyses.

One-way analysis of variance was used to compare participant characteristics (age, BMI, body composition-related parameters) across 3 groups. Repeated measures analysis of variance was used to test group, time, and group $\times$ time interaction effects for OGTT results (glucose, insulin, C-peptide). Post hoc comparisons using Bonferroni adjustment were performed at each time point during the OGTT.

Analysis of covariance (ANCOVA) was used to assess the effect of SCI status (AB, paraplegic, tetraplegic) on parameters for glucose homeostasis (glucose, insulin, C-peptide at fasting and at min 120 during OGTT, homeostatic model assessment-insulin resistance, ISI) with covariates reflecting body composition. OGTT values at min 120 were used for

^c.Hologic QDR-4500W; Hologic.

^d.ADULT software, Lunar DPX-L version 1.35; GE-Luncar Corp.

^e.HiLight/HTD Advantage scanner; General Electric Co.

^f.SAS version 9.4; Statistical Analysis Systems.

analysis because most individuals with glucose intolerance and Type II diabetes are diagnosed on the basis of plasma glucose concentrations at this time point.²² Because of the small sample size, we limited covariates to 2 at a time in the ANCOVA. In our preliminary analysis, we found high correlations among parameters reflecting body adiposity (FM, trunk FM, leg/arm FM, %FM, and VF; $R^2 > 0.5$, $P < .05$ for each pair), and among parameters reflecting lean body mass (TBLM, trunk lean mass, leg/arm lean mass, TBLMI; $R^2 > 0.5$, $P < .05$ for each pair). As a result, to avoid multicollinearity, percentage fat and TBLM were included to reflect adiposity and lean mass, respectively, in the ANCOVA model. Additional covariates (eg, age, family history of type 2 diabetes) were examined and removed because they were nonsignificant ($P > .05$) when included in the ANCOVA or correlated with percentage fat or TBLM to avoid multicollinearity ($P < .05$). To further assess the effect of VF, we also performed a second ANCOVA with VF and TBLM as covariates to evaluate their effects on the listed outcomes. Post hoc pairwise comparisons were performed using Bonferroni adjustment. Data are presented as means $\pm$ SD unless otherwise stated. Significance level was set a priori at $P < .05$.

Results

Participant characteristics

Twenty-two women with SCI (8 with tetraplegia [C4: 1, C5: 3, C6: 3, C8: 1 injury], 14 with paraplegia [11 thoracic and 3 lumbar injuries]) and 20 AB women were recruited and frequency-matched for race, age, and BMI. Fifteen women with SCI had a motor complete lesion (American Spinal Injury Association Impairment Scale A or B), whereas 7 women with SCI had a motor incomplete lesion (American Spinal Injury Association Impairment Scale C or D).²³

Women who were tetraplegic had lower TBLM, appendicular lean mass and trunk lean mass than AB controls (table 1), and lower TBLMI than those with paraplegia and AB controls ($P < .05$). No differences were observed for the remaining variables (age, weight, BMI, %FM, VF, absolute FM) among the 3 groups.

Responses to OGTT

Group responses without adjustment for covariates during the 2-hour OGTT are shown in fig 1. Post hoc comparisons showed that women with tetraplegia had higher glucose (60, 90, 120min), insulin (120min), and C-peptide (60, 90, 120min) than those with paraplegia and AB women ($P < .05$ for all) (for detailed values, see supplemental table S1, available online only at <http://www.archives-pmr.org/>).

After adjusting for percentage fat and TBLM, indices of fasting glucose homeostasis (glucose, insulin, C-peptide, and homeostatic model assessment-insulin resistance) did not differ among groups (ANCOVA) (table 2A). At OGTT min 120, plasma glucose, insulin, and C-peptide were higher ($P < .05$) in women with tetraplegia versus women with paraplegia and their AB counterparts. Women with tetraplegia had lower ISI than AB women ($P < .05$).

Similarly, after adjusting for VF and TBLM, plasma glucose, insulin, and C-peptide concentrations at min 120 were higher ($P < .05$) in women with tetraplegia versus women

with paraplegia and AB women (table 2B). In addition, women with tetraplegia had lower ISI than AB women ($P<.05$).

Discussion

It is generally accepted that deterioration of body composition among individuals with SCI contributes significantly to the rapid decline of their metabolic health. However, our study shows that the impairment of glucose metabolism among women with tetraplegia in comparison to those with paraplegia and AB controls cannot be fully explained by differences in %FM (or VF) and TBLM, suggesting other factors must also be contributing to the impaired glucose metabolism found in women with tetraplegia.

A deterioration of body composition has been commonly reported among men with SCI; however, less is known about changes in body composition in women after SCI. In our study, women with tetraplegia have significant muscle atrophy, as evidenced by reduced TBLM, appendicular, and trunk lean mass than AB controls. In contrast, we did not observe differences in %FM or percentage lean mass (complementary of %FM) among women with paraplegia and AB controls. This is in contrast with a previous study of women and men with paraplegia (17 men/11 women; average age: 34 years; duration of injury: 11 years) which showed an 8% less percentage fat-free mass than the AB controls (77.2% vs 69.2%, $P<.05$).²⁴ The differences in the results may be due to several different demographics attributes (age, sex, time since injury) of the study cohorts. Furthermore, in contrast to a study using rat SCI model,²⁵ we observed no level-of-injury effect on visceral adiposity. Our result, however, is consistent with a human study, where the apparent differences in visceral adiposity between tetraplegic and paraplegic SCI disappeared after adjusting for age.¹⁴

Previously it was shown that men with SCI have a higher degree of insulin resistance compared to women with SCI.¹³ Our study further shows that women with tetraplegia also have worsened glucose tolerance as compared to those with paraplegia and their AB counterparts. This is evidenced by the pronounced elevation of glucose and insulin concentrations at min 120 during OGTT. In contrast to the OGTT results, normal fasting glucose profile was observed among women with SCI (regardless of the level of injury), similar to existing studies of men with SCI.^{3,26} These findings suggest a potential extrahepatic insulin resistance (eg, skeletal muscle) in the SCI population. However, the role of liver in fasting state glucose homeostasis cannot be dismissed, as a positive correlation between liver adiposity and fasting glucose concentration was previously found.²⁷ Furthermore, our results emphasize that fasting glucose concentration alone is not an adequate indicator for diabetes diagnosis in women with SCI, as previously reported in men with SCI.¹³ Thus, OGTT and/or hemoglobin A1c may be alternative options for diagnosis purpose.

Our data showed reduced lean body mass (−8.8kg), ~50% of which was attributed to reduced appendicular lean mass (−4.3kg), among women with tetraplegia compared to those with paraplegia and AB controls. Considering that skeletal muscle uses ~80% of insulin-mediated glucose uptake in fed state among lean healthy individuals,⁹ changes in skeletal muscle after SCI may explain part of the variability of glucose tolerance during OGTT.

However, our data suggested that the level-of-injury effect was still present after adjusting for lean body mass. It is probable that SCI-induced muscle quality deterioration may contribute to the impairment of carbohydrate metabolism.^{6,28} It was found that intramuscular fat (IMF) tissue is 3 times higher^{29,30} and abundance of glucose transporter 4 is significantly lower³¹ in individuals with SCI than in AB controls. Increased IMF is implicated in the development of glucose intolerance.^{6,29} In fact, Elder et al reported that IMF in thigh accounted for 70% of the glucose variability 120 minutes after ingesting glucose solution.²⁹ In addition, recent evidence points out that an increase in skeletal muscle mitochondria activity (citrate synthase activity) is associated with improved insulin sensitivity among individuals with SCI.³² Individuals with tetraplegia may have reduced mitochondrial activity due to skeletal muscle mass loss and increased IMF, which further contribute to glucose intolerance.³³ Thus, future investigations on tetraplegia SCI-related muscle quality adaptations, such as IMF content and mitochondria activity, may offer insight on their effects on metabolic disorders.

In addition to muscle, the relations between adiposity and metabolic health were investigated. In our analyses, %FM is associated with several glucose-related parameters (see table 2), corroborating previous findings on the deleterious effect of adiposity on metabolic health. However, %FM, along with TBLM, did not fully explain the differences in glucose tolerance among AB women and women with tetraplegia or paraplegia. Potentially the differences in glucose tolerance among the groups may be attributed to alterations in adipose tissue distribution or type after SCI. Recent studies explored the effects of fat distribution, that is, VF versus subcutaneous fat in the arm, leg, and trunk areas, on metabolic outcomes.^{17,34} It was suggested that visceral adipose tissue was associated with a negative metabolic profile while trunk and leg subcutaneous adipose tissue is associated with reduced risk of glucose tolerance.^{34–36} However, our data suggest that the differences in glucose tolerance among groups may not be fully explained by visceral adiposity (total abdominal fat) either. Another preliminary study among men with SCI showed that neither visceral nor subcutaneous fat explained the differences in glucose tolerance and insulin sensitivity between different levels of injury.³⁷ Together, these results further suggest that other factors associated with injury level may contribute to the impaired glucose tolerance among women with tetraplegia.

Study limitations

First, FM was not distinguished by its distribution (eg, of intra- vs intermuscular vs subcutaneous; leg vs trunk vs arm). Because an increasing amount of research suggests the importance of fat distribution, sheer quantitative measure of body fat can only tell part of the story on body composition and metabolic health. Future studies using magnetic resonance imaging will provide additional insight on the effect of fat distribution on glucose intolerance among individuals with SCI. Second, because of the small sample size, we were limited to 2 covariates in the ANCOVA model. The effects of other potential contributing factors, such as duration of injury, physical activity, and menopausal status on metabolic profile in this population remains unclear. Last, when TBLM and %FM were included in the ANCOVA model, the TBLM was positively correlated with insulin or glucose concentrations at min 120 during OGTT and Matsuda index. Even though this relation is

unexpected physiologically, it is not uncommon when multiple linear regression analyses are performed. The coefficients for TBLM obtained in the current models are contingent on the presence of additional variables, that is, percentage body FM. However, we performed sensitivity analysis by adjusting for 1 covariate at a time, and the results are consistent with those obtained from current ANCOVA model and did not change our conclusions.

Conclusions

Our study provides initial data on the relation of body composition and level of injury on metabolic profiles in women with SCI. Our results suggest that although alterations in body composition may contribute to metabolic impairment in women with tetraplegia, it does not fully account for it. These results point to the need for identifying additional, alternative explanations for injury level-associated metabolic impairment in individuals with tetraplegia. Considering the alterations that occur in the autonomic nervous system (sympathetic and parasympathetic) after an SCI, significant dysfunction of brown adipose tissue function is expected. Accordingly, our studies are ongoing for investigating the potential effects of SCI on brown adipose tissue amount and function and the associations between brown adipose tissue function and metabolic health among individuals with tetraplegia and paraplegia. Research focusing on metabolism in this population may offer new clinical insight to direct future studies as to the type of interventions (nutritional, lifestyle, etc) best suited to protect women with SCI from metabolic diseases.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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List of abbreviations:

AB	able-bodied
ANCOVA	analysis of covariance
BMI	body mass index
CT	computed tomography
DXA	dual-energy x-ray absorptiometry
FM	fat mass
IMF	intramuscular fat

ISI	insulin sensitivity index
OGTT	oral glucose tolerance test
SCI	spinal cord injury
TBLM	total body lean mass
TBLMI	total body lean mass index
UAB	University of Alabama at Birmingham
VF	visceral fat
Wt	weight

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Fig 1.

Glucose, insulin, and C-peptide concentrations during OGTT. # $P < .05$, women with tetraplegia were different from women with paraplegia and AB women.

Table 1

Participants' characteristics

	AB (n = 20)	Paraplegic (n = 14)	Tetraplegic (n = 8)	P Value
Age (y)	43.6±11.9	43.4±10.1	40.6±12.3	.81
Injury duration (y)	NA	14.7±15.1	12.6±8.5	
Weight (kg)	71.4±19.0	69.8±21.9	61.8±9.2	.47
Height (cm)	165.2±4.8	161.4±7.1	167.8±7.5	.06
BMI (kg/m ²)	26.7±6.5	27.9±8.4	22.1±2.3	.16
FM (kg)	26.6±11.5	28.2±14.8	25.8±8.9	.89
% FM	35.9±6.4	38.5±9.1	41.0±9.2	.30
VF (cm ²)	143.0 ±113.8	134.1±78.4	140.0±79.1	.97
TBLM (kg)	44.8±8.2	45.6±8.7	36.0±4.4 [*]	.04
TBLMI (kg/m ²)	16.4±2.6	15.9±3.0	12.8±1.7 ^{*,†}	.01
LM _{appendicular} (kg)	18.6±3.8	16.0±4.2	14.3±2.2 [*]	.02
LM _{trunk} (kg)	20.8±4.3	21.9±4.6	18.4±2.3 [*]	.05

NOTE. Data are mean ± SD.

Abbreviation: LM, lean mass.

^{*} Different from able-bodied group.[†] Different from paraplegic group, $P < .05$.

Table 2

Comparison of indices of glucose homeostasis among participants

A. ANCOVA with %FM and TBLM as covariates									
Parameter	AB (n = 20) *	PARA (n = 14) *	TETRA (n = 8) *	Coefficient: β (95% Confident Interval); <i>P</i>			Group Effect, <i>P</i> [‡]	Difference Between Groups (95% Confidence Interval)	
				TBLM	% Fat			PARA-AB	TETRA-AB TETRA-PARA
Glucose _{Baseline} (mg/dL)	88.3±1.9	91.5±2.2	87.9±3.2	0.08 (-0.27, 0.44); 0.65	41.2 (5.9, 76.4); 0.02		.46	-3.3 (-10.6, 4.1)	-0.4 (-10.3, 9.6) -3.6 (-13.1, 5.9)
Glucose ₁₂₀ (mg/dL)	108.9±7.0	115.9±7.9	162.3±11.6 ^{‡, §}	1.34 (0.04, 2.6); 0.04	68.5 (-59.7, 196.7); 0.29		.00	-7.0 (-33.9, 19.9)	53.4 (17.2, 89.5) 46.4 (11.7, 81.0)
Insulin _{Baseline} (μIU/mL)	11.3±1.1	14.1±1.3	14.1±1.9	0.3 (0.1, 0.5); 0.00	23.0 (2.2, 43.9); 0.03		.25	-2.8 (-7.2, 1.6)	2.8 (-3.1, 8.6) 0.0 (-5.7, 5.6)
Insulin ₁₂₀ (μIU/mL)	58.6±12.4	65.6±14.0	134.5±20.4 ^{‡, §}	1.9 (-0.4, 4.2); 0.10	246.0 (20.1, 474.9); 0.03		.01	-7.0 (-54.4, 40.4)	75.8 (12.1, 139.6) 68.9 (7.8, 129.9)
ISI	4.8±0.3	4.1±0.4	2.9±0.6 [‡]	-0.08 (-0.15, -0.02); 0.01	-7.4 (-13.6, -1.2); 0.02		.04	0.8 (-0.5, 2.1)	-2.0 (-3.7, -0.2) -1.2 (-2.9, 0.4)
HOMA-IR	2.6±0.3	3.3±0.3	3.2±0.5	0.07 (0.03, 0.13); 0.03	5.4 (0.4, 10.4); 0.00		.16	-0.8 (-1.9, 0.2)	0.7 (-0.7, 2.1) 0.1 (-1.5, 1.2)
C-peptide _{Baseline} (nmol/L)	1.8±0.2	1.9±0.2	2.3±0.3	0.04 (0.00, 0.08); 0.02	4.1 (0.4, 7.8); 0.03		.48	-0.2 (-0.9, 0.6)	0.5 (-0.5, 1.6) 0.3 (-0.7, 1.3)
C-peptide ₁₂₀ (nmol/L)	8.2±0.8	8.3±1.0	13.3±1.4 ^{‡, §}	0.02 (-0.13, 0.18); 0.75	14.8 (-0.6, 30.3); 0.06		.01	-0.1 (3.3, 3.1)	5.1 (0.8, 9.5) 5.0 (0.9, 9.29)
B. ANCOVA with VF and TBLM as covariates									
Parameter	AB (n = 20) *	PARA (n = 14) *	TETRA (n = 8) *	Coefficient: β (95% Confident Interval); <i>P</i>			Group Effect, <i>P</i> [‡]	Difference Between Groups (95% Confidence Interval)	
				TBLM	VF			PARA-AB	TETRA-AB TETRA-PARA
Glucose _{Baseline} (mg/dL)	87.6±2.2	92.0±2.6	89.0±3.8	0.0 (-0.5, 0.6); 0.91	0.02 (-0.02, 0.06); 0.28		.44	4.3 (-4.2, 12.9)	1.4 (-10.3, 13.1) -2.9 (-14.5, 8.6)
Glucose ₁₂₀ (mg/dL)	105.6±6.6	107.2±7.9	169.9±11.6 ^{‡, §}	1.4 (-0.1, 3.0); 0.07	-0.00 (-0.07, 0.12); 0.95		.00	1.6 (-24.6, 27.7)	64.2 (28.5, 100.0) 62.6 (27.5, 97.8)
Insulin _{Baseline} (μIU/mL)	11.3±1.1	14.5±1.3	14.5±1.9	0.2 (-0.1, 0.4); 0.20	0.03 (0.01, 0.05); 0.00		.14	3.2 (-1.0, 7.5)	3.2 (-2.6, 9.1) 0.03 (-5.7, 5.7)
Insulin ₁₂₀ (μIU/mL)	54.4±12.4	58.3±14.8	151.8±21.8 ^{‡, §}	1.4 (-1.6, 4.4); 0.34	0.14 (-0.10, 0.37); 0.24		.00	3.9 (-45.3, 53.0)	97.3 (30.2, 164.5) 93.4 (27.4, 159)

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ISI	4.9±0.3	4.0±0.4	2.4±0.6 [‡]	-0.06 (-0.15, 0.02); 0.12	-0.01 (-0.01, 0.00); 0.06	.06	-0.9 (-2.3, 0.4)	-2.5 (-4.4, -0.7)	-1.6 (-3.4, 0.3)
HOMA-IR	2.5±0.3	3.3±0.3	3.3±0.5	0.05 (-0.02, 0.11); 0.14	0.007 (0.002, 0.012); 0.01	.12	0.8 (-0.2, 1.9)	0.9 (-0.6, 2.3)	0.01 (-1.4, 1.4)
C-peptide _{Baseline} (nmol/L)	1.7±0.2	1.8±0.2	2.5±0.3	0.03 (-0.02, 0.07); 0.23	0.003 (-0.001, .0006); 0.15	.17	-0.1 (-0.8, 0.7)	0.77 (-0.3, 1.8)	0.69 (-0.3, 1.7)
C-peptide ₂₀ (nmol/L)	8.0±0.8	7.8±1.0	14.7±1.4 [‡] , §	-0.01 (-0.20, 0.19); 0.95	0.01 (-0.01, 0.02); 0.22	.00	0.18 (-3.0, 3.4)	6.7 (2.3, 11.1)	6.9 (2.6, 11.2)

Abbreviations: HOMA-IR: homeostatic model assessment-insulin resistance; PARA, individuals with paraplegia; TETRA, individuals with tetraplegia.

* Data are least square mean ± standard error from analysis of variance analysis.

[‡] P-value for group effect from analysis of variance analysis.

[‡] Different from AB group.

[§] Different from PARA group, $P<.05$.