Visceral adipose tissue and the ratio of visceral to subcutaneous adipose tissue are greater in adults with than in those without spinal cord injury, despite matching waist circumferences[1–3]

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ABSTRACT

Background: Abdominal obesity, particularly visceral adipose tissue (VAT), is associated with an increased risk of coronary heart disease (CHD). Despite an elevated risk of CHD mortality in persons with spinal cord injury (SCI), neither abdominal adipose tissue accumulation nor the validity of waist circumference (WC) has been determined in persons with SCI.

Objectives: The objectives of this study were to compare total adipose tissue (TAT), visceral adipose tissue (VAT), subcutaneous adipose tissue (SAT), and the ratio of VAT to SAT (VAT:SAT) between adults with SCI and age-, sex-, and WC-matched able-bodied (AB) controls and to determine the relation between WC and VAT in both groups.

Design: Thirty-one men and women ($n = 15$ SCI and 16 AB) with a mean ($\pm$SD) age of 38.9 ± 7.9 y participated in this cross-sectional study. Abdominal adipose tissue was quantified by computed tomography at L_4-L_5. WC was measured at 3 sites: lowest rib, iliac crest, and the midpoint between the lowest rib and iliac crest.

Results: Persons with SCI had a 58% greater mean VAT ($P = 0.003$), 48% greater mean VAT:SAT ($P = 0.034$), and 26% greater mean TAT ($P = 0.055$) than did matched AB controls after differences in weight were accounted for. Mean SAT was not significantly different between groups. WC at all sites was correlated with VAT in both groups (SCI: $0.905 \leq r \leq 0.925$; AB: $0.838 \leq r \leq 0.877$; both $P < 0.001$).

Conclusions: High levels of VAT exist in young people with SCI who classify themselves as active and healthy. WC may be a valid surrogate measure of VAT in this population and serve as a tool for clinicians to identify those at risk of CHD. *Am J Clin Nutr* 2008; 87:600–7.

INTRODUCTION

Worldwide, coronary heart disease (CHD) and type 2 diabetes are associated with significant morbidity and mortality; however, in persons with long-standing spinal cord injury (SCI), the risk of mortality associated with diabetes and CHD is 1.4- and 2-fold higher, respectively, than that of able-bodied (AB) persons with diabetes and cardiovascular disease (1–3). Increased mortality risk may, in part, be due to SCI-specific changes in body composition. Persons with SCI have significantly less fat-free mass and greater fat mass than do those who are AB (4–6)

because of the muscle atrophy and physical inactivity associated with SCI (7, 8). Thus, body mass index (BMI) has been a poor and inconsistent predictor of CHD and diabetes risk factors in persons with SCI (9, 10), yet it remains a primary anthropometric assessment tool in the clinical context. Failure to accurately identify obesity may result in a failure to identify those at risk of diabetes and CHD. Abdominal obesity, specifically visceral adipose tissue (VAT), has been identified as an independent risk factor for diabetes and CHD in the AB population (11, 12), but has yet to be investigated in the SCI population. Although waist circumference (WC) is a good surrogate measure of VAT in the AB population (13–15), the relation between WC and VAT has not yet been examined in persons with SCI.

The primary objectives of the present study were to compare total abdominal adipose tissue (TAT), VAT, subcutaneous abdominal tissue (SAT), and the ratio of VAT to SAT (VAT:SAT) in adults with chronic SCI with those of age-, sex-, and WC-matched AB controls and to determine the relation between WC and VAT in both groups. The secondary objective was to examine the metabolic profiles of those with and those without SCI.

SUBJECTS AND METHODS

Subjects

Fifteen apparently healthy volunteers aged 19–54 y with chronic, traumatic SCI ($\geq$1 y after injury) were recruited for the study. Most of the SCI participants were recruited from a local fitness program (MacWheelers, Hamilton, Canada), whereas the remainder were active community members interested in research who attended the outpatient Spinal Cord Rehabilitation Program at Chedoke Hospital (Hamilton, Canada). By design, the group was heterogeneous with respect to BMI and level and completeness of lesion. The level of lesion was classified as

either paraplegia [injury to the sacral (S_1-S_5), lumbar (L_1-L_5), or thoracic (T_2-T_{12}) regions of the spine] or tetraplegia [injury to T_1 or the cervical spine (C_1-C_9)]. Completeness was classified as either complete [no sensory or motor function preserved in the sacral segments (S_4-S_5)] or incomplete (sensory but variable motor function preserved below the neurological level of injury). Sixteen apparently healthy AB volunteers were recruited from the University of Guelph (Guelph, Canada), McMaster University, and Hamilton Health Sciences (Hamilton, Canada). Participants were recruited by poster campaign and word of mouth. Participants with SCI were enrolled first, followed by AB controls matched for age (± 5 y), sex, and WC (± 5 cm). Exclusion criteria included pregnancy, lactation, amputation, nontrauma SCI etiology, a history of alcohol abuse, type 1 diabetes, bowel impaction, active decubitus ulcer, body weight >170 kg [maximum capacity of the computed tomography (CT) scanner], and thyroid, hepatic, or renal disorders. All participants completed an interviewer-administered health history questionnaire, including level, completeness, and etiology of lesion (only for participants with SCI), which was confirmed by clinical evaluation. The questionnaire also included information on medications, smoking status, recent illness, bowel and bladder function, and self-reported physical activity levels (included activities of daily living as well as recreational and structured physical activity). Participants were asked to categorize themselves as sedentary, mildly active, moderately active, or very active and describe the type, frequency, duration, and intensity of the activity. Women self-reported the first day of their last menstrual cycle and oral contraceptive use.

All data collection took place at McMaster University Medical Centre (MUMC) (Hamilton, Canada) during a 2-h visit. All participants emptied their bladders on the morning of the study and their bowels in the 24-h before data collection. Participants arrived in the morning or early afternoon after fasting for a minimum 12 h. The protocol was approved by the Research Ethics Boards of the University of Guelph and Hamilton Health Sciences, and written informed consent was obtained from all participants. Participants were provided with a nominal honorarium to cover travel and parking.

Anthropometric measures

All measurements were made with the participants wearing light clothing and no shoes. Those with SCI were weighed in their wheelchairs on a floor scale (Mettler Toledo Canada, Mississauga, Canada). Once they transferred from their wheelchair to the CT scanning table, their wheelchair weight was measured and subtracted from the total weight to determine body weight. The AB controls were also weighed with the same scale. The scale was calibrated with the use of 2×4.5 kg weights before each weight measurement; all weights were measured to the nearest 0.1 kg.

Length and WC measurements were made by using a flexible nonelastic Gulick II tape measure (Country Technology Inc, Gay Mills, WI), which was calibrated to provide consistent and adequate compression (4-oz, or 0.11-kg, tension) for each measurement. All measurements were taken on the right side of the body by the same investigator (LAE) while the participants were supine on the CT scanning table and immediately after the scan. Length was measured to the nearest centimeter. The tape measure was extended on the table beside the participant, from heel to crown, with the participants' feet stretched into dorsiflexion by the investigator. For SCI participants with permanent plantar flexion, length measures were made from the base of the heel. For participants with contractures preventing the straightening of their legs, length was measured in segments from heel to crown. WC was measured after normal expiration at 3 sites: immediately below the lowest rib, immediately above the iliac crest, and midpoint between the lowest rib and the iliac crest. The landmarking for all 3 sites was done while the participants were supine with their arms placed overhead. For all WC measurements, the tape measure was placed directly on the skin with the participants' arms by their sides. Measurements were taken in duplicate to the nearest 0.1 cm. If the difference between the first and second measure was >0.1 cm, a third measure was obtained. The reproducibility of the WC measurements was very high; the intraclass correlation coefficients (ICC) were $r = 0.999$ (95% CI: 0.998, 0.999) for all 3 sites. Because all measurements were conducted by the same investigator, we had no estimation of interrater reliability; however, published interrater reliability of WC measurements is high, with an ICC of $r = 0.988$ (95% CI: 0.982, 0.993) (16).

Adipose tissue

Total, subcutaneous, and visceral abdominal adipose tissue area was assessed by a single-slice abdominal CT scan of the intervertebral space between the fourth and fifth lumbar vertebrae (L_4-L_5). CT was performed with a GE CTI Single Slice scanner (General Electric Corp, Waukesha, WI) by using the procedures of Sjöström et al (17) and Kvist et al (18) as previously described (19). Each participant was examined while supine with their arms stretched overhead to minimize artifacts. A lateral CT scout image was used to obtain a radiograph of the spine to precisely localize the scan in the position between L_4 and L_5. The scan was performed with 140 kV and a slice thickness of 10 mm. Abdominal adipose tissue area was calculated by delineating the abdominal scan with a graph pen and then computing the adipose tissue surface by using an attenuation range from -190 to -30 Hounsfield units (HU). Abdominal VAT was measured by drawing a line within the muscle wall surrounding the abdominal cavity. VAT area corresponded to the surface inside the circumscription fulfilling the attenuation criteria of adipose tissue, whereas SAT corresponded to the area outside the abdominal muscle wall. TAT was calculated by adding VAT and SAT. The same radiation technologist performed and analyzed all scans in 1–2 sessions, with an intraindividual CV $\pm 5\%$.

Biochemical assays

Venous blood samples were collected by a phlebotomist. Serum insulin; C-reactive protein (CRP); total, LDL, and HDL cholesterol; triacylglycerol; and plasma glucose were all analyzed in the MUMC core lab, whereas plasma adiponectin and oxidized LDL (ox-LDL) were analyzed at the University of Guelph. All samples were centrifuged at $1000 \times g$ for 15 min at 4 °C. All variables except insulin, adiponectin, and ox-LDL were analyzed immediately. Plasma was separated into 0.5-mL aliquots into each of 2 microcentrifuge tubes, which were subsequently frozen at -80 °C at the MUMC until transferred to the University of Guelph on ice and again stored at -80 °C until analyzed.

Plasma glucose was measured with the glucose oxidase method on a Roche Modular ISE 1800 (Roche Diagnostics, Indianapolis, IN). Total cholesterol, HDL cholesterol, and triacylglycerol were measured with enzymatic colorimetric tests on a Roche Modular ISE 1800 (Roche Diagnostics). LDL was calculated by using the Friedewald equation (20). Serum blood samples for insulin analysis were frozen at $-80\ °C$ until measured with a solid-phase, two-site chemiluminescent immunometric assay on the Immulite 2000 Analyzer (Diagnostic Products Corp, Los Angeles, CA). Insulin resistance was calculated by the homeostasis model assessment (HOMA) method by using fasting glucose and insulin concentrations. Assuming that normal-weight participants have an insulin resistance of 1, the value for insulin resistance can be assessed by using the following equation (21):

Insulin resistance = fasting insulin (mU/L)

$$\times\ \text{fasting glucose (mmol/L)}/22.5 \quad (1)$$

For insulin concentrations <15 pmol/L (below the detection limit of the kit; $n = 1$ SCI and 2 AB), 14 pmol/L was used to calculate the mean values for insulin and HOMA (insulin resistance). Serum CRP was measured with a highly sensitive immunoassay by using a monoclonal antibody coated with polystyrene particles; the assay was performed by means of particle-enhanced immunonephelometry on a BN*II Systems analyzer (Dade Behring, Deerfield, IL). The high-sensitivity assay reliably measures low levels of inflammation (0–10 mg/L) associated with CVD (22). Participants with a CRP concentration >10 mg/L or who reported current infections ($n = 3$ AB and 6 SCI) were excluded from analysis (23).

The concentration of ox-LDL in plasma was measured with a sandwich enzyme-linked immunosorbent assay procedure by using the murine monoclonal antibody mAb-4E6 as capture antibody (bound to microtitration wells) and a peroxidase-conjugated antiapolipoprotein B antibody recognizing ox-LDL bound to the solid phase (Mercodia AB, Uppsala, Sweden). The resultant color reaction was read at 450 nm. The intra- and interassay CVs were 6.2% and 4.7%, respectively. Plasma adiponectin concentrations were also measured with a sandwich enzyme-linked immunosorbent assay procedure by using a murine monoclonal antibody specific for adiponectin (R & D Systems, Minneapolis, MN). The resultant color reaction was read at 450 nm, with a wavelength correction of 540 nm. The sensitivity of the kit was 0.25 ng/mL, and the intraassay and interassay CVs were 3.3% and 6.5%, respectively.

Dietary intake

At the end of the study visit, participants were given a 3-d food record and were instructed on how to complete it. Intake was recorded on 3 nonconsecutive days, including 2 weekdays and one weekend day. For those that preferred, food records were submitted electronically, instead of being handwritten, and returned in the stamped self-addressed envelope provided. Records include all food, beverages, vitamins, and supplements consumed on each day. Serving size pictures and a ruler were provided to aid participants with portion estimation. Food records were analyzed by using an ESHA Food Processor SQL (ESHA Research Inc, Salem, OR) updated with the 2005 Canadian Nutrient File.

Statistical analysis

All statistical analyses were performed by using SPSS version 14.0 (SPSS Inc, Chicago, IL) at a significance level of $P < 0.05$. Data are expressed as means ± SDs. Triacylglycerol, CRP, insulin, and HOMA values were normalized with the use of a logarithmic transformation. Because no study to our knowledge had previously measured VAT in persons with SCI, the sample size calculation was based on differences in truncal fat mass reported in studies of persons with and without chronic SCI (4, 6, 24). Assuming 80% power, 14 participants per group were required to detect a significant difference in truncal fat mass (3.4 kg).

Independent t tests were used to compare the amount of abdominal adipose tissue (VAT, TAT, SAT, and VAT:SAT) between the SCI and AB control groups. Because VAT, SAT, TAT, and VAT:SAT are closely related, a multivariate analysis of covariance was used to examine the influence of weight as a potential confounder in the differences between groups. In both groups, Pearson correlation coefficients were used to examine the relation between VAT and WC at all 3 sites.

To compare the metabolic profiles of the SCI and AB groups, differences in biochemical variables (glucose, insulin, HOMA, triacylglycerol, CRP, adiponectin, and total, HDL, LDL, and ox-LDL cholesterol) were determined by using independent t tests. Associations between WC, VAT, BMI, and biochemical variables were examined by using Pearson correlation coefficients. The SCI and AB participants were divided into 2 subgroups according to VAT: a participant with a VAT value greater than the mean of his or her respective group (SCI: >120 cm²; AB: >83 cm²) was determined to have a high VAT. A participant with a VAT lower than his or her group's mean was determined to have low VAT. Similarly, the SCI and AB participants were divided into 2 subgroups according to WC (lowest rib): a participant with a WC greater than the mean of his or her respective group (SCI: >92.3 cm; AB: >90.8 cm) was determined to have a high WC. A participant with a WC lower than his or her group's mean was determined to have a low WC. The differences between those with high and low VAT and WC, normal (≤1.70 mmol/L) and elevated (>1.70 mmol/L) triacylglycerol concentrations (25), paraplegia and tetraplegia, complete and incomplete injuries, males and females, and AB and SCI dietary intakes (total energy, kcal; fat, kcal; protein, kcal; carbohydrate, kcal; alcohol, kcal; % of energy as fat, % of energy as protein, % of energy as carbohydrate, fiber, and cholesterol) were determined by independent t tests.

Linear regression models were used to examine the ability of WC (lowest rib) to predict VAT in the SCI and AB groups and to determine the difference in the amount of VAT between the SCI and AB groups per centimeter of WC. Chi-square tests were used to examine the differences in physical activity level between study (SCI and AB) and VAT (low and high) groups as well as the relation between physical activity and study group in those with a high VAT. Physical activity levels were dichotomized as high active (self-identified as moderately or very active) and low active (self-identified as sedentary or mildly active).

RESULTS

Descriptive characteristics of the participants are presented in **Table 1**. The BMI for participants with SCI ranged from 15.2 to

TABLE 1
Descriptive characteristics of participants[1]

	AB controls (n = 12 M, 4 F)	SCI subjects (n = 12 M, 3 F)
Age (y)	38.1 ± 8.4 (24–53)[2]	39.8 ± 7.4 (28–49)
Weight (kg)	83.2 ± 19.7 (52.6–127.9)	79.4 ± 21.4 (42.6–135.2)
Length (m)	1.80 ± .09 (1.57–1.90)	1.77 ± .12 (1.48–1.98)
BMI (kg/m^2)	25.5 ± 5.8 (18.1–36.9)	25.2 ± 6.5 (15.2–39.0)
Ethnicity [n (%)]		
White	14 (88)	12 (80)
African American	1 (6)	1 (7)
Other	1 (6)	2 (13)
Dietary intake		
Total energy (kcal)	2090 ± 447 (1511–2826)	2079 ± 652 (1090–3279)
Fat (% of total energy)	28.6 ± 6.1 (20–38)	33.6 ± 8.3 (17–44)
Protein (% of total energy)	16.2 ± 3.9 (10–25)	15.4 ± 3.3 (10–22)
Carbohydrate (% of total energy)	52.6 ± 6.7 (43–68)	51.2 ± 12.8 (28–72)

[1] AB, able-bodied; SCI, spinal cord injury. There were no significant differences between groups, $P > 0.05$ (independent t test).

[2] $\bar{x} \pm$ SD; range in parentheses (all such values).

39.0. There were no significant differences in sex, age, weight, length, BMI, ethnicity, caloric intake, or macronutrient distribution between the SCI and AB groups. Within the SCI group, 40% had paraplegia, 60% tetraplegia, 73% were classified as having complete lesions, and 27% were classified as having incomplete lesions. Most injuries resulted from motor vehicle accidents (60%), followed by sports-related and diving accidents (20%), falls (13%), and surgery (7%). The mean age at injury was 23.3 ± 6.1 y (18–40 y), and the mean time after injury was 16.5 ± 8.7 y (1.1–28.7 y).

On the health-history questionnaire, 7 participants (SCI) self-reported osteoporosis, 4 were smokers (n = 2 SCI and 2 AB), and 4 reported hyperlipidemia (n = 2 SCI and 2 AB); of the hyperlipidemic subjects, 2 reported taking lipid-lowering medication (n = 1 SCI and 1 AB) and 1 SCI participant reported taking an anticoagulant drug. Three AB participants reported elevated blood pressure; of these subjects, 2 were taking antihypertensive medication. Four SCI participants reported elevations in blood pressure; however, this may have been due to autonomic dysreflexia, because none of these participants were receiving pharmacologic treatment. All women (n = 7) were premenopausal; 4 were in the luteal phase of menstruation (n = 2 SCI and 2 AB),

2 were in the follicular phase of menstruation (n = 1 SCI and 1 AB), 1 had had a partial hysterectomy, and 4 reported oral contraceptive use (n = 2 SCI and 2 AB). One participant reported hyperlipidemia, osteoporosis, CHD, borderline diabetes type 2, and the use of anti-arrhythmic drugs. When this subject was excluded from the analyses, the results did not change; therefore, this participant's data were included in the final data set. No participants were taking oral hypoglycemic agents or any other medications known to influence glucose metabolism.

The mean VAT:SAT was 46% greater for the SCI group than for the AB controls ($P < 0.05$) (**Table 2**). Mean VAT (45%), TAT (16%), and SAT (6%) were also greater for the SCI group, but not significantly greater. After weight was adjusted for, the SCI group had a 58% greater VAT and a 48% greater VAT:SAT, which were statistically significant ($P < 0.05$). TAT was 26% greater in the SCI group ($P = 0.055$). The amount of SAT was not significantly different between groups. When differences in WC, age, or BMI were controlled for, the results did not change (data not shown).

The difference in VAT between a participant without (left) and with (right) SCI is shown in **Figure 1**. The pair was matched for age, WC, weight, BMI, and male sex (SCI subject: age, 47 y; WC,

TABLE 2
Abdominal obesity in able-bodied (AB) controls and spinal cord injury (SCI) participants

	AB controls (n = 12 M, 4 F)	SCI subjects (n = 12 M, 3 F)	P[1]	Adjusted P[2]
Waist circumference (cm)				
Lowest rib	90.9 ± 14.0 (64.2–117.5)[3]	92.3 ± 14.7 (66.8–123.3)	0.788	—
Midpoint	91.1 ± 14.2 (64.8–119.0)	93.2 ± 15.5 (67.0–125.7)	0.700	—
Iliac crest	91.9 ± 12.7 (72.4–119.5)	93.6 ± 14.5 (73.8–125.4)	0.727	—
Abdominal adipose tissue				
Total (cm^2)	335 ± 187 (68–697)	388 ± 211 (116–892)	0.463	0.055
Visceral (cm^2)	83 ± 50 (4–159)	120 ± 70 (27–257)	0.097	0.003
Subcutaneous (cm^2)	252 ± 146 (64–569)	268 ± 154 (83–635)	0.769	0.284
VAT:SAT[4]	0.32 ± 0.16 (0.06–0.55)	0.47 ± 0.22 (0.15–0.92)	0.039	0.034

[1] By independent t test.

[2] Abdominal adipose tissue compartments were adjusted for weight (kg) by using multivariate analysis of covariance.

[3] $\bar{x} \pm$ SD; range in parentheses (all such values).

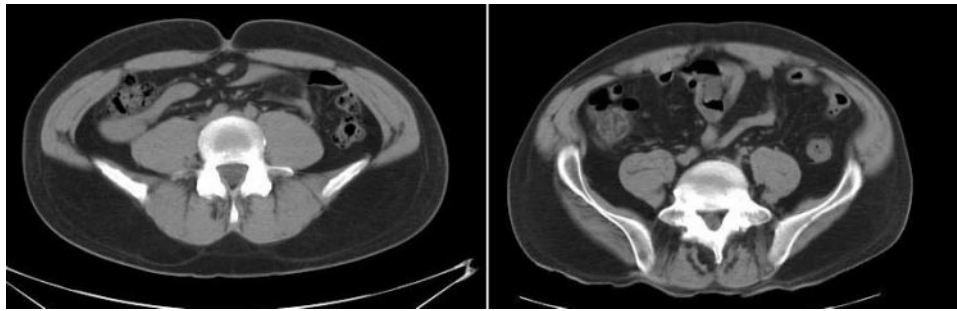[4] Ratio of visceral to subcutaneous adipose tissue.

FIGURE 1. Axial abdominal computed tomography (CT) scans of L_4-L_5 for a pair of male participants (left: able-bodied control, right: spinal cord injury) matched (within $\pm5\%$) for age, waist circumference, weight, BMI, and total abdominal adipose tissue. Adipose tissue appears dark gray. Visceral adipose tissue (VAT) is the adipose area within the abdominal muscle wall, and subcutaneous adipose tissue (SAT) is the area outside the abdominal muscle wall. Left: VAT, 52 cm^2; SAT, 261 cm^2; VAT:SAT 0.20. Right: VAT, 145 cm^2; SAT, 185 cm^2; VAT:SAT, 0.78.

97.7 cm; weight, 88.0 kg; and BMI, 25.4; AB subject: age, 46 y; WC, 97.3 cm; weight, 91.2 kg; and BMI, 25.9), yet the amount of VAT is almost 3 times that in the participant with SCI.

Significant correlations were found between VAT and WC in both groups, at all 3 WC sites ($P < 0.0001$). Compared with the AB controls, the magnitude of correlation with VAT was slightly higher in the SCI group: lowest rib (SCI: $r = 0.925$, AB: $r = 0.873$), iliac crest (SCI: $r = 0.905$; AB: $r = 0.838$), and midpoint between the lowest rib and iliac crest (SCI: $r = 0.925$; AB: $r = 0.877$). Conversely, the correlation coefficient for the relation between VAT and BMI was slightly higher in the AB group ($r = 0.808$, $P < 0.0001$) than in the SCI group ($r = 0.757$, $P = 0.001$). In both groups, significant correlations were also found between WC at all 3 sites and TAT and SAT ($0.858 \leq r \leq 0.967$, $P < 0.0001$).

No significant differences in any of the biochemical variables measured were observed between the SCI and AB groups (**Table 3**). Also, the group means for glucose, insulin, CRP, triacylglycerol, and total, HDL, and LDL cholesterol were all within references ranges. There was, however, a trend toward higher adiponectin in the SCI than in the AB group ($P = 0.088$).

In the SCI group, significant correlations were found between VAT and $\log_{10}$ insulin ($r = 0.551$, $P < 0.05$) and $\log_{10}$ HOMA ($r = 0.589$, $P < 0.05$) and between WC at all 3 sites and $\log_{10}$ insulin ($0.561 \leq r \leq 0.649$, $P < 0.05$) and $\log_{10}$ HOMA (0.599

$\leq r \leq 0.684$, $P < 0.05$). A higher VAT:SAT was associated with lower HDL cholesterol ($r = -0.615$, $P < 0.05$) and higher $\log_{10}$ CRP ($r = 0.796$, $P = 0.01$). The relations between VAT, WC, and all other biochemical variables were not statistically significant in the SCI group.

There were no differences in any variable between persons with paraplegia ($n = 6$) and those with tetraplegia ($n = 9$), except for higher total cholesterol in those with paraplegia (5.2 ± 1.1 compared with 4.0 ± 0.7 mmol/L; $P = 0.031$). There was a trend toward higher insulin ($P = 0.055$), HOMA insulin resistance ($P = 0.053$), and VAT ($P = 0.076$) in the participants with tetraplegia than in those with paraplegia; however, after adjustment for differences in WC and BMI, the groups were not significantly different. Those SCI participants with incomplete injuries ($n = 4$) had greater weight, BMI, WC, VAT, and TAT ($P < 0.05$) than did those with complete injuries ($n = 11$). However, after adjustment for differences in WC and BMI, the differences in VAT and TAT became nonsignificant.

Men ($n = 24$) had a greater WC ($P < 0.05$), ox-LDL ($P < 0.01$), and triacylglycerol ($P < 0.01$) and lower HDL ($P < 0.01$) and adiponectin ($P < 0.05$) than did the women ($n = 7$). Men also had a greater VAT:SAT ($P < 0.01$) and a trend toward a greater VAT ($P = 0.058$) than did the women. After adjustment for differences in WC and BMI, the women had a greater SAT and TAT (both $P < 0.0001$), and the differences in triacylglycerol,

TABLE 3
Fasted biochemical variables in able-bodied (AB) controls and spinal cord injury (SCI) participants[1]

	Reference range	AB controls ($n = 12$ M, 4 F)	SCI subjects ($n = 12$ M, 3 F)
Glucose (mmol/L)	3.8–6.0	5.0 ± 0.3 (4.4–5.7)[2]	4.9 ± 0.4 (4.1–5.6)
Insulin (pmol/L)[3]	43–194	59 ± 66 (<15–271)	58 ± 36 (<15–128)
Insulin resistance (HOMA)[3]	>1.0	1.9 ± 2.2 (0.4–9.0)	1.8 ± 1.1 (0.4–3.6)
C-reactive protein (mg/L)[3,4]	<3.0	1.6 ± 1.3 (0.2–4.0)	2.5 ± 1.5 (0.6–5.1)
Triacylglycerol (mmol/L)[3]	<1.7	1.5 ± 0.8 (0.6–3.5)	1.2 ± 0.8 (0.6–3.7)
Total cholesterol (mmol/L)	<5.2	5.0 ± 1.1 (2.7–6.7)	4.5 ± 1.1 (2.9–6.7)
HDL (mmol/L)	>1.0	1.5 ± 0.5 (0.8–2.4)	1.3 ± 0.3 (1.0–1.9)
LDL (mmol/L)	<3.4	2.8 ± 0.9 (1.5–4.2)	2.6 ± 0.9 (1.4–4.8)
Oxidized LDL (U/L)	—	57 ± 16 (32–85)	58 ± 11 (47–81)
Adiponectin (μg/mL)[5]	—	4.6 ± 2.7 (1.1–11.0)	6.4 ± 2.9 (2.1–11.0)

[1] HOMA, homeostasis model assessment. There were no significant differences between groups, $P > 0.05$ (independent t test).

[2] $\bar{x} \pm$ SD; range in parentheses (all such values).

[3] t tests were done on log-transformed values (nontransformed values reported).

[4] $n = 13$ AB and 9 SCI.

[5] $n = 15$ AB and 15 SCI.

VAT, and VAT:SAT between men and women became nonsignificant.

Linear regression analysis showed $WC_{Lowest\ rib}$ to be the only significant predictor of VAT [SCI: $P < 0.0001$ (95% CI: 3.70, 9.48); AB: $P = 0.034$ (95% CI: 0.22, 4.80)] when age and BMI were included in the regression model. Sex was not a significant predictor of VAT in either group. Analysis by stepwise or hierarchical regression models did not change the results (data not shown).

More participants self-identified as high active (94%) in the low-VAT group, whereas 47% self-identified as low active and 53% as high active ($P = 0.01$) in the high-VAT group. Although 93% of SCI participants self-identified as high active, compared with 56% of AB participants ($P < 0.05$), the SCI group showed significantly greater VAT and TAT. Of the AB participants with high VAT, 75% self-identified as low active, whereas 86% of the SCI participants with high VAT self-identified as high active ($P < 0.05$).

To determine the best WC site, the relation between the biochemical variables and measurement of WC at all 3 sites was examined. WC at the lowest rib and midpoint differentiated normal (≤ 1.70 mmol/L) from elevated (> 1.70 mmol/L) triacylglycerol, whereas measurement at the iliac crest did not. When the sample was divided into high-and low-WC groups, a high WC at the lowest rib was associated with a higher glucose concentration (5.1 ± 0.3 compared with 4.8 ± 0.4 mmol/L; $P = 0.01$), whereas measurement at the midpoint or iliac crest showed no differences in fasting glucose.

DISCUSSION

The most important findings of this study were that *1*) adults with chronic SCI have significantly more TAT and VAT and a higher VAT:SAT than do AB adults matched for age, sex, and WC; and *2*) WC is highly correlated with VAT in persons with SCI.

In both SCI and AB persons, WC is an independent predictor of VAT. Our results suggest that persons with SCI have 42% more VAT per centimeter of WC than do their AB counterparts. Adjustment for BMI and age increased the difference between groups, such that persons with SCI had more than double (162%) the amount of VAT per centimeter WC. This was likely due to abdominal muscle atrophy and subsequent increases in the proportion of fat mass to fat-free mass.

The relation between VAT and WC at all 3 sites (lowest rib, iliac crest, and midpoint between the lowest rib and the iliac crest) in the SCI participants ($r > 0.90$) was strong and comparable with the relation observed in both the AB participants and the AB literature ($0.67 \leq r \leq 0.87$) (13–15, 19, 26, 27). The relation between WC and VAT was stronger than that of BMI and VAT, and, in general, the magnitude of the correlations between the biochemical variables and VAT and WC was stronger than that between the biochemical variables and BMI (data not shown). These results suggest that WC may be more sensitive in detecting CHD risk than BMI in the SCI population and complement preliminary evidence that current BMI cutoffs (established in AB population) are insensitive for identifying obesity in persons with chronic SCI (4, 6, 10, 28, 29).

A higher proportion of the SCI group than of the AB group reported an active lifestyle, in contrast with other cross-sectional studies reporting predominantly sedentary pursuits in the general SCI population (7, 30–32). However, this is not surprising given that most of the SCI participants were recruited from a community fitness program, and all but 2 were involved in a structured exercise program or organized sport. These results suggest that even those persons with SCI who partake in regular physical activity may have a high accumulation of VAT.

Although the concentrations of insulin, triacylglycerol, CRP, glucose, and total, HDL, and LDL cholesterol fell within the reference ranges for healthy adults and were not different between the groups, other researchers have found a high prevalence of metabolic risk factors, including low HDL, high CRP, glucose intolerance, and insulin resistance in large SCI samples (33–41). Two comments can be made regarding this finding. First, our participants with SCI were younger and more active than the general SCI population. This may provide one explanation for the discrepancy between our findings and the literature (ie, a higher metabolic risk in SCI than in AB groups), despite a higher VAT in our SCI group. Second, the biochemical reference ranges were established in AB populations; the utility of these cutoffs for persons with SCI is not known. Given that lower reference ranges for lipid concentrations have been recommended for primary and secondary CHD prevention in AB adults with multiple risk factors (42, 43), and that increased risk has been associated with large accumulations of VAT, lower blood lipid ranges may be appropriate for persons with SCI. For example, if the LDL cutoff suggested by the Canadian Cardiovascular Society of < 2.0 mmol/L for high-risk patients (42) was extended to our group of SCI participants (2.6 mmol/L), then 73% would have been at increased risk compared with 25% in the AB group.

Given the antiatherogenic and antiinflammatory effects of adiponectin, we expected higher adiponectin in the AB group. However, similar to the findings of Wang et al (44), there was a trend toward higher adiponectin in the SCI group. One possible explanation is that various forms of adiponectin are secreted by adipose tissue and measured by enzyme-linked immunosorbent assay, not all of which are biologically active (45, 46). Furthermore, persons with SCI may produce biologically inactive forms (45–47). Second, SCI may protect against hypoadiponectinemia. Ruige et al (48) have reported higher concentrations of adiponectin in men and women with the lowest resting metabolic rate (RMR), even after adjustment for age, VAT, and HDL. Given that persons with SCI have a reduced RMR because of a reduced fat-free mass (5), those with low RMR who are theoretically at greater risk of obesity-related disorders may be especially protected by adiponectin.

Although we cannot make any conclusions about the relation between abdominal obesity and the biochemical profile of persons with SCI, our finding of a positive relation between VAT, WC, and fasting insulin as well as insulin resistance is consistent with previous reports of an association of impaired carbohydrate metabolism with trunk fat mass and intramuscular fat in persons with SCI (35–39).

We recommend that WC be measured at the lowest rib in persons with SCI, for the following practical and physiologic reasons: *1*) the lowest rib is relatively easy to landmark; *2*) measurement at the lowest rib is likely associated with less error than is measurement at the midpoint, which requires the landmarking of 2 sites; *3*) measurements at the lowest rib are less likely to be affected than are measurements at the midpoint or iliac crest, by abdominal distension and/or bowel impaction, both of which occur frequently in the SCI population; and *4*) a high WC at the

lowest rib was associated with a higher glucose concentration, and WC measured at the lowest rib differentiated normal from elevated triacylglycerol.

We showed that WC provides a surrogate measure of visceral adiposity in persons with SCI. Our sample of 15 relatively young and active persons with SCI was not adequately powered for subanalyses by sex or level or completeness of injury. The assessment of physical activity, dietary intake, and medical history by self report alone, without confirmation through health records or other independent means, limits the ability of this study to evaluate the subjects' risk factor profiles and to examine the relation between risk factors and WC.

Given the ability of VAT to predict metabolic risk in the AB population, future research should examine this relation in a larger more representative sample of persons with SCI. Longitudinal assessment of VAT and WC may further elucidate the relation between abdominal obesity and chronic disease risk in the SCI population.

In conclusion, TAT, VAT, and the VAT:SAT in this group of relatively young, active and apparently healthy adults with SCI was clinically and significantly higher than age-, sex-, and WC-matched AB adults. Despite this, the metabolic profile that may be expected to accompany a high-VAT depot was not apparent, which suggests 1 of 2 things: *1*) despite high accumulations of VAT, an active lifestyle may significantly reduce biochemical risk factors for CHD and diabetes; and/or *2*) even in young, active individuals with SCI, high levels of VAT are present, which may eventually manifest as atherogenic dyslipidemia and aberrations in glucose-insulin homeostasis because this cohort ages into their 50s and beyond. Given the current recommendations for measurement of WC in the AB population (49, 50), the insensitivity of BMI in the SCI population and the finding that WC was highly correlated with VAT in persons with SCI, we encourage clinicians to incorporate WC as a measure of abdominal obesity and recommend that it be measured at the level of the lowest rib.

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