

ORIGINAL ARTICLE

Current coronary heart disease risk assessment tools may underestimate risk in community-dwelling persons with chronic spinal cord injury

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Study design: Cross-sectional, observational study.

Objectives: To quantify, in adults with chronic spinal cord injury (SCI): (1) presence of metabolic syndrome versus the general North American population (GP) and (2) 10-year coronary heart disease (CHD) risk using Framingham risk scoring (FRS).

Setting: Ontario, Canada.

Methods: Fasting anthropometric and biochemical data were collected from 75 adults with chronic SCI. Metabolic syndrome was determined using four internationally recognized definitions and FRS using the most recent (2001) algorithm.

Results: Prevalence of metabolic syndrome was up to 5.4 times lower in SCI participants compared to GP, and FRS categorized 3.1% of participants as being at high 10-year CHD risk. However, high-sensitivity C-reactive protein (CRP) values indicated 36.7% of participants as being at high CHD risk.

Conclusion: Current metabolic syndrome definitions and FRS may underestimate true CHD risk in people with SCI. Tools that better identify CHD risk require validation in the SCI population. CRP may be a potential factor to consider in the development of SCI-specific screening tools.

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Keywords: spinal cord injury; metabolic syndrome; heart disease; secondary complications; evidence-based practice; SHAPE SCI

Introduction

Medical advances in acute management of traumatic spinal cord injury (SCI) have resulted in an increased lifespan in this population. While encouraging, cardiovascular diseases (CVD) have become one of the leading causes of death in

long-term SCI¹ with increased rates of morbidity related to CVD, particularly coronary heart disease (CHD), relative to the able-bodied (AB) population.² Persons with SCI experience body composition changes (decreased lean body mass, increased fat mass) and more sedentary lifestyles than AB persons,³ as well as increased rates of impaired glucose tolerance, insulin resistance and diabetes and reduced high-density lipoprotein cholesterol (HDL-C).² All of these may explain the increased risk for CVD.

Despite these metabolic changes, there are currently no clinically relevant, validated tools to identify adults with SCI at risk for CHD. Practical screening tools such as Framingham Risk Scoring (FRS) or diagnosis of metabolic syndrome may simplify identification of persons with SCI at high CHD risk. Only two studies have identified the prevalence of metabolic syndrome in persons with SCI. Relative to AB populations, the prevalence in SCI was either lower⁴ or no different.⁵ However, despite the multitude of metabolic syndrome definitions in the literature, both Lee *et al.* and Liang *et al.* used only one definition; the National Cholesterol Education Panel (NCEP)⁶ and the National Heart Lung

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and Blood Institute/American Heart Association (NHLBI/AHA),⁷ respectively. In addition, neither research group reported FRS.

Should tools such as FRS and/or metabolic syndrome definitions help to identify CHD risk, health care interventions could be provided in a more timely manner, potentially leading to improved prevention and/or treatment in this high-risk population. The objectives of our study were to (1) quantify metabolic syndrome in adults with chronic SCI using four internationally recognized definitions and to compare these to metabolic syndrome in the general North American population (GP) and (2) quantify 10-year CHD risk using the most recent (2001) FRS algorithm.

Materials and methods

Participants

The Study of Health and Activity in People with Spinal Cord Injury (SHAPE-SCI) was a multicenter study in Ontario, Canada, the purpose of which was to determine the role of physical activity in the health and quality of life of people with SCI, and for which the complete methodology has been published.⁸ The chronic disease risk factor data reported herein were collected from a subset of SHAPE-SCI participants, all of whom lived within a 200 km radius of Hamilton, Ontario, Canada. Eligible participants were ≥ 18 years of age and had experienced a traumatic SCI at least 12 months prior to study enrollment. The SHAPE-SCI and substudy protocols were approved by the Research Ethics Boards of Hamilton Health Sciences and University of Guelph, Ontario, Canada. All study participants gave written, informed consent, and substudy participants were provided with a nominal honorarium for participation.

Methods

Substudy data collection took place in participants' homes. Demographics (sex, age, ethnicity), level and completeness of spinal cord lesion, and brief family and personal medical histories (including information on current medications) were collected from each participant by trained interviewers using a structured questionnaire. All anthropometric measurements were completed by two trained research assistants, after participants had fasted for at least 10 h. Each measure was taken twice and averaged if individual measures differed by $\leq 5\%$. If the difference was $>5\%$, a third determination was made and the two closest measures were averaged. Participants were weighed in light clothing and wearing no shoes, using a portable, digital, wheelchair scale (Health O Meter 2450KL, Brooklyn, NY, USA). Measures were taken in kg to one decimal place. At each visit, the accuracy of the scale was determined using two 6.8 kg weights. Participants then transferred from their wheelchairs to a spine board (National Lifesaving Society item EQ-10, Edmonton, Alberta, Canada) that lay on top of their beds to provide a hard, flat surface on which to measure length. Participants' feet were placed in dorsal flexion and head in the Frankfurt Plane. As in the general SCI population, leg contractures in our sample were common and prevented

some participants from straightening their legs, even with assistance. To maintain measurement consistency for the whole sample, length for all participants was measured to the nearest decimal place, on the right side of the body alongside the spine board, in segments from heel to crown using a flexible, nonelastic tape measure with a spring attachment for constant tension (Gulick II Tape Measure, Gays Mills, WI, USA).

Body mass index (BMI, in kg m^{-2}) was assessed using World Health Organization (WHO) classifications.⁹ Percent fat mass (%FM) was determined using whole-body bioelectrical impedance analysis (BIA; Bioelectrical Body Composition Analyzer Quantum II, RJL Systems, Clinton Twp., MI, USA) and electrode placement of Lukaski *et al.*¹⁰ Fat-free mass (kg) was calculated using NHANES III equations,¹¹ and FM by subtracting fat-free mass from weight. Lohman and Going's age-specific obesity cutoffs were used to assess %FM.¹²

Waist circumference (WC) was measured at the level of the iliac crest to the nearest decimal place using the same nonelastic, flexible measuring tape used for length. Participants were supine with arms abducted 30° from midline. The iliac crest was land-marked with a pen and the circumference recorded after normal expiration. Values were interpreted against National Institutes of Health cutoffs.¹³

Venous blood samples were collected in empty vacutainer tubes with the exception of those for glucose, in which there was anticoagulant. All tubes were immediately placed on ice in a lightfast cooler and were analyzed at the McMaster Medical Centre Department of Laboratory Medicine, Hamilton, on the day of collection. All specimens were centrifuged for 15 min at 3000 r.p.m. Samples for insulin were separated and frozen at -80°C until analysis, the next business day.

Insulin was measured using a solid-phase, two-site chemiluminescent immunometric assay (IMMULITE 2000/2500; Intermedico, Holliston, MA, USA). Insulin values of six participants were lower than detectable by assay ($<15 \text{ pmol l}^{-1}$); these were assigned a value of 14 pmol l^{-1} during statistical analyses. Glucose was measured using the hexokinase method (Roche Modular ISE 1800; Roche, Laval, Québec, Canada). Insulin resistance was determined using the homeostasis model assessment for insulin resistance (HOMA-IR),¹⁴ where $\text{HOMA-IR} = (\text{glucose mmol l}^{-1} \times \text{insulin mU l}^{-1})/22.5$.

Total cholesterol (TC) and triglycerides (TG) were determined using an enzymatic colorimetric test and HDL-C using a homogeneous enzymatic colorimetric test (Roche). Low-density lipoprotein cholesterol (LDL-C) values were determined using the Friedewald equation.¹⁵ High-sensitivity C-reactive protein (CRP) was measured using particle enhanced immunonephelometry (CardioPhase hsCRP; Dade Behring, Mississauga, Ontario, Canada). CRP cut points of 3.1–9.9, 1.0–3.0 and $<1.0 \text{ mg l}^{-1}$ classified participants as high, average and low CHD risk, respectively.¹⁶ Those whose CRP values were indicative of acute inflammation ($\geq 10 \text{ mg l}^{-1}$)¹⁶ were excluded from CRP statistical analyses. CRP values between participants with versus without self-reported infections and taking versus not taking statins were

not significantly different (data not shown); these participants were therefore included in CRP statistical analyses. Blood pressure (BP) was measured on the right arm, in the sitting position after a 5 min rest, by auscultation using a stethoscope and sphygmomanometer.

All participants were categorized into those with >20%, 10–20% or <10% 10-year risk for CHD according to FRS using traditional risk factors including gender, age, TC, HDL-C and systolic BP.⁶ Participants were also classified as positive or negative for metabolic syndrome using four internationally recognized definitions (presented in the Appendix) including the World Health Organization (Metabolic Syndrome_{WHO});¹⁷ the original National Cholesterol Education Program (Metabolic Syndrome_{NCEP});⁶ the original National Heart, Lung, and Blood Institute/American Heart Association (Metabolic Syndrome_{NHLBI/AHA})⁷ and the original International Diabetes Federation (Metabolic Syndrome_{IDF}) definition¹⁸ (in our analysis, Europid cut points for WC were used for Caucasians, Native Canadians and those of unknown ethnic origin (≥ 94 cm in men and ≥ 80 cm in women; $n = 63$), while ethnic South and Central Canadian cut points were used for Hispanic Canadians (≥ 90 cm in men, ≥ 80 cm in women; $n = 1$)). The only modification we made was to Metabolic Syndrome_{WHO}. In the current study, and with respect to glucose regulation, only fasting glucose (FG) and self-reported diabetes mellitus data were collected. Therefore, these were the only two measures used to identify impaired glucose regulation as neither 2-h post-glucose load nor hyperinsulinemic euglycemic conditions were performed as suggested in Metabolic Syndrome_{WHO}.¹⁷ In addition, waist-to-hip ratio and urinary albumin excretion rate were not measured and were therefore excluded from Metabolic Syndrome_{WHO} diagnosis.

Statistical analyses

Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS version 14.0, Chicago, IL, USA). Only descriptive data are reported for both the percentage of participants with metabolic syndrome and the distribution of 10-year CHD risk according to FRS. The κ -values are calculated to determine interrater reliability and in this case were applied to test agreement between the four metabolic syndrome definitions where $\kappa = 0.40$ – 0.59 , $\kappa = 0.60$ – 0.79 and $\kappa \geq 0.80$ refer to moderate, substantial and good agreement, respectively.¹⁹ Correlations were computed between duration of injury and individual CVD risk factors using Pearson's and Spearman's correlation coefficients for normally and nonnormally distributed data, respectively.

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

A total of 75 participants participated in the substudy. Of these, 10 were excluded based on self-reported CHD and/or diabetes mellitus and/or were receiving pharmacological

Table 1 Background characteristics of participants

	All participants (n = 65)
Age (years) ^a	41.7 ± 11.7
Years living with SCI (years) ^a	15.0 ± 10.6
Sex ^b	
M	52 (80.0)
F	13 (20.0)
Level of lesion ^b	
Tetraplegia	33 (50.8)
Paraplegia	32 (49.2)
Completeness of lesion ^{b,c}	
Complete	22 (34.4)
Incomplete	42 (65.6)
Primary mode of mobility ^b	
Wheelchair	60 (92.3)
Braces	2 (3.1)
Walker	1 (1.5)
Cane	1 (1.5)
Crutches	1 (1.5)
Ethnicity ^b	
Caucasian	61 (93.8)
Other ^d	4 (6.2)

Abbreviations: F, female; M, male; SCI, spinal cord injury.

^aYears ± standard deviation.

^bFrequency (percentage).

^cCompleteness of lesion unknown for one participant.

^dOther ethnicities include one Native Canadian, one Hispanic Canadian and two unknown.

treatment for these conditions. Of the remaining 65 participants, 4 had not fasted for at least 10 h; 3 were unable to provide blood samples; 1 was missing WC data and 1 was missing data to calculate BMI. Therefore complete data for 56 participants were available for determination of metabolic syndrome using Metabolic Syndrome_{NCEP}, Metabolic Syndrome_{NHLBI/AHA} and Metabolic Syndrome_{IDF} definitions, 57 for Metabolic Syndrome_{WHO} definition and 61 participants for determination of 10-year CHD risk using FRS.

Background characteristics are presented in Table 1. The group was predominantly male (80.0%) and Caucasian (93.8%). Mean age was 41.7 ± 11.7 years (range 21–79 years) and average duration of injury was 15.0 ± 10.6 years (range 1–47 years). Of the 33 (50.8%) participants with tetraplegia, 9 (27.3%) and 24 (72.7%) had complete and incomplete lesions, respectively. Of the 32 (49.2%) participants with paraplegia, 13 (41.9%) and 18 (58.1%) had complete and incomplete lesions, respectively; completeness of lesion unknown for one participant. The majority of participants (92.3%) ambulated with a wheelchair (manual or power), and the remainder with braces (3.1%), walker (1.5%), cane (1.5%) or crutches (1.5%). Participants with CHD and/or diabetes excluded from statistical analysis were older (47.1 ± 11.8 years; range 28–70 years), but had been living with SCI for a similar duration (14.9 ± 10.4 years; range 2–34 years) versus those without.

Anthropometric and biochemical data are presented in Table 2. All variables fell within normal limits, with the exception of CRP, %FM and WC (for female participants). Mean CRP ($3.4 \pm 3.0 \text{ mg l}^{-1}$) was consistent with high CHD risk. Mean %FM indicated that both male ($25.7 \pm 7.3\%$) and female participants ($39.4 \pm 7.6\%$) were obese. Mean WC values for female participants ($93.4 \pm 18.1 \text{ cm}$), but not for male ($89.7 \pm 13.3 \text{ cm}$), were also indicative of health risk. Duration of injury was not related to any of the CVD risk factors except for FG ($r = 0.272$, $P = 0.037$).

The percentage of metabolic syndrome in the study sample was 3.6, 12.3, 15.8 and 19.3% according to the criteria of Metabolic Syndrome_{WHO}, Metabolic Syndrome_{NCEP}, Metabolic Syndrome_{NHLBI/AHA} and Metabolic

Syndrome_{IDF}, respectively (Figure 1). The κ -agreement between definitions varied from no agreement beyond chance between Metabolic Syndrome_{WHO} and Metabolic Syndrome_{IDF} ($\kappa = 0.099$, $P > 0.05$) to moderate agreement between Metabolic Syndrome_{WHO} and Metabolic Syndrome_{NHLBI/AHA} ($\kappa = 0.324$, $P < 0.01$) and Metabolic Syndrome_{WHO} and Metabolic Syndrome_{NCEP} ($\kappa = 0.412$, $P < 0.001$) to substantial agreement between Metabolic Syndrome_{NCEP} and Metabolic Syndrome_{IDF} ($\kappa = 0.608$, $P < 0.001$) and Metabolic Syndrome_{NHLBI/AHA} and Metabolic Syndrome_{IDF} ($\kappa = 0.758$, $P < 0.001$) and finally to good agreement between Metabolic Syndrome_{NCEP} and Metabolic Syndrome_{NHLBI/AHA} ($\kappa = 0.855$, $P < 0.001$). The prevalence of metabolic syndrome in the GP is 19.4%,²⁰ 34.5%²¹ and 39.0%,²¹ according to Metabolic Syndrome_{WHO}, Metabolic Syndrome_{NCEP} and Metabolic Syndrome_{IDF}, respectively, 2.0–5.4 times higher than the SCI study sample (Figure 1).

Reduced HDL-C levels (31.0%) followed by elevated TG (26.7%) and obesity (24.6%) were the most common metabolic syndrome risk factors in the study sample when averaged across all definitions, followed by hypertension (23.8%) and finally elevated FG (10.2%) (Figure 2).

FRS identified 80.3, 16.4 and 3.1% of participants to be at low (<10%), moderate (10–20%) and high (>20%) 10-year CHD risk, respectively, with a mean risk of $4.8 \pm 6.3\%$.

Table 2 Anthropometric and biochemical data of participants

	Participants (n = 49–65) ^a
BMI (kg m^{-2})	25.5 ± 5.3
Waist circumference (cm)	90.4 ± 14.2
% Fat Mass _{BIA} ^b	28.4 ± 9.1
Blood pressure (mm Hg)	
Systolic	111 ± 21
Diastolic	73 ± 13
Fasting glucose (mmol l^{-1})	5.1 ± 0.6
Fasting insulin (pmol l^{-1})	58 ± 56
HOMA-IR ^c	1.9 ± 1.9
C-reactive protein (mg l^{-1})	3.4 ± 3.0
Total cholesterol (mmol l^{-1})	4.64 ± 1.05
Triglycerides (mmol l^{-1})	1.45 ± 0.87
LDL cholesterol (mmol l^{-1})	2.73 ± 0.93
HDL cholesterol (mmol l^{-1})	1.22 ± 0.33

Abbreviations: BMI, body mass index; HDL, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment for insulin resistance; LDL, low-density lipoprotein cholesterol.

^aSample size ranged due to lack of blood samples and/or protocol violations (e.g., participant not fasted prior to blood draw, improper calibration of BIA in the case of fat mass). $n = 49$ for CRP as blood work was unavailable for four participants and an additional 12 participants had CRP levels indicative of acute phase response.

^bCalculated using formula derived from NHANES III data.¹¹

^cHOMA-IR, homeostasis model assessment for insulin resistance¹⁴. $\text{HOMA-IR} = (\text{glucose } \text{mmol l}^{-1} \times \text{insulin } \text{mU l}^{-1}) / 22.5$, where pmol l^{-1} were converted into mU l^{-1} by dividing by 7.217.

Discussion

This is the first study in community-dwelling persons with SCI to quantify metabolic syndrome using multiple definitions, and to calculate 10-year CHD risk using the most recent FRS algorithm. The most important finding was that persons with SCI are at seemingly low risk for CHD. However, rather than reflecting a truly decreased risk for CHD, this may be due in part to insensitivity of these tools in this population.

Although persons with SCI have increased morbidity (2) and mortality (1) related to CVD, metabolic syndrome was lower in this sample population compared to the larger GP. Similarly, FRS indicated relatively low CHD risk in our

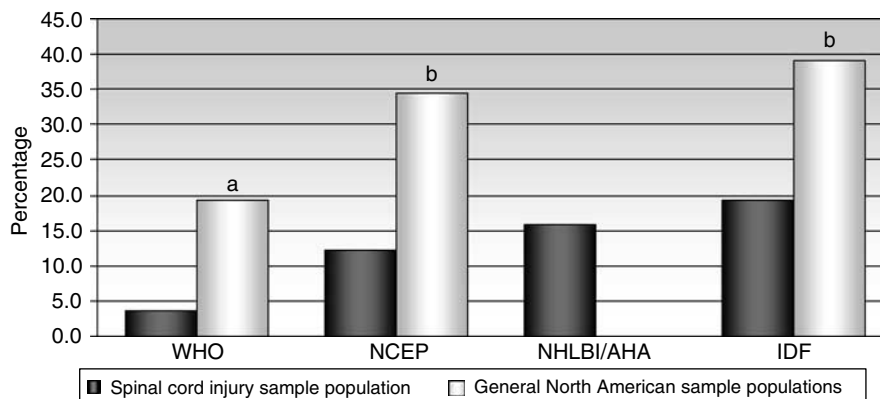


Figure 1 Metabolic syndrome in participants with spinal cord injury versus general North American population. ^a(20), ^b(21).

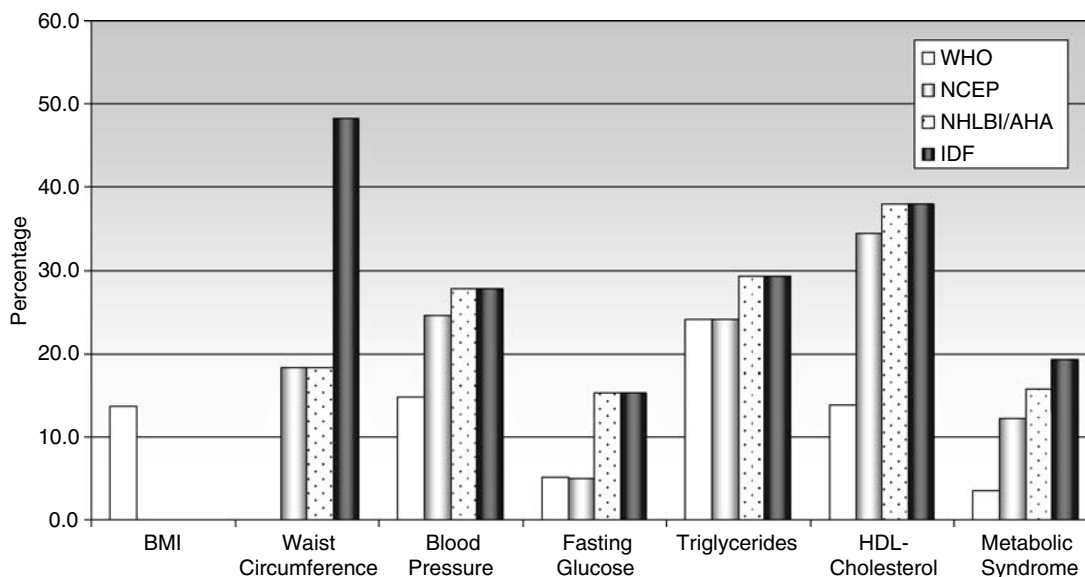


Figure 2 Presence of metabolic syndrome risk factors using four definitions in participants with spinal cord injury (SCI).

sample population. Rather than reflecting a truly decreased risk of CHD, this may be due to the fact that current metabolic syndrome definitions and FRS, all derived in AB populations, include metabolic risk factors that preclude accurate CHD risk assessment in SCI. Similar concerns regarding the application of common CVD risk assessment tools derived in the AB population to persons with SCI have been expressed by others.^{4,5,22} For example, impaired glucose tolerance, but not elevated FG, is common in the SCI population.² Metabolic syndrome definitions include FG and thus not surprisingly fail to identify persons for whom glucose-related aberrations occur in the nonfasted state. Similarly, mean %FM of our participants was consistent with the definition of obese;¹² however metabolic syndrome definitions include BMI or WC, both of which have questionable sensitivity in the SCI population when using AB cutoffs.^{23,24} Finally, mean BP of our study sample was low-to-normal. Although BP is a strong predictor of CHD risk in the AB population, an inverse relationship has been described between the level of spinal cord lesion and reduced BP²⁵ such that BP may not be predictive of CHD risk. Clearly, the use of AB derived metabolic risk factors and/or their associated cutoffs require reevaluation in the SCI population. This is supported by our finding of varying agreement across metabolic syndrome definitions, likely due to the various risk factors and corresponding threshold limits included in each definition. Inclusion of novel CHD risk factors, such as hsCRP, may help identify persons with SCI at risk for CHD. This work is currently underway in our group.

The insensitivity of metabolic syndrome definitions was mirrored in our FRS-related findings. The FRS algorithm includes both smoking status and TC as CVD risk factors; however, Krum *et al.*²⁶ found that the increased risk of CVD in SCI compared to the AB population cannot be explained

by differences in TC or smoking. This would therefore make it difficult for FRS to capture the increased risk of CHD in those with SCI.

Aside from potentially insensitive metabolic markers and/or cutoffs, other factors may have contributed to seemingly lower metabolic syndrome in the current study. First, given the lack of recent Canadian metabolic syndrome prevalence data, prevalence in the GP was based on data from the United States with Canadian obesity rates lower than those of the United States,²⁷ it is most likely that metabolic syndrome would also be elevated in the United States, offering a partial explanation for metabolic syndrome being lower in the current sample population. Second, given that prevalence increases with age, metabolic syndrome was higher in the older GP, potentially contributing to the disparity.

While our study contributes to the literature on chronic diseases in the SCI population, we recognize that it is limited by the cross-sectional nature and small sample size, particularly of women, which precluded a subanalysis by gender. Prospective research that investigates CHD risk factors in men and women against hard end points, such as coronary events, is required in the SCI population to help improve the identification of those at high risk of CHD. We also recognize that some medications may have confounded values for selected biochemical markers, however in excluding participants taking medications for CHD and/or diabetes-related risk factors, we have likely accounted for the most important confounders. Finally, we suggest that future studies investigate the effect of different bladder regimens on CRP values in this population.

In summary, current metabolic syndrome definitions and FRS may underestimate true CHD risk in persons with chronic SCI. We suggest that alternative biological markers be used and/or that cutoffs for current markers—all

established in AB populations—be revised for persons with SCI. CRP may be a potential factor to consider in the development of SCI-specific screening tools. CRP is explored more fully in our second article included in this issue of *Spinal Cord* (Gibson, Buchholz, Martin Ginis, SHAPE SCI Research Group).

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Appendix

World Health Organization metabolic syndrome diagnostic criteria (1988)

<i>Clinical measure</i>	<i>Cut points</i>
Impaired glucose regulation ^a (any of T2DM, IGT, IFG or lowered insulin sensitivity) plus any of two factors mentioned below for diagnosis	T2DM: Fasting plasma glucose: $\geq 7.0 \text{ mmol l}^{-1}$ or 2-h post-glucose load: $\geq 11.1 \text{ mmol l}^{-1}$ or both: IGT: Fasting plasma glucose (if measured): $< 7.0 \text{ mmol l}^{-1}$ and 2-h post-glucose load: $\geq 7.8 \text{ mmol l}^{-1} < 11.1 \text{ mmol l}^{-1}$ IFG: Fasting plasma glucose: $\geq 6.1 \text{ mmol l}^{-1} < 7.0 \text{ mmol l}^{-1}$ 2-h post-glucose load (if measured): $< 7.8 \text{ mmol l}^{-1}$ Men: waist-to-hip ratio $> 0.90^b$ Women: waist-to-hip ratio $> 0.85^b$ and/or BMI $> 30 \text{ kg m}^{-2}$ $\geq 1.69 \text{ mmol l}^{-1}$ $< 0.91 \text{ mmol l}^{-1}$ in men $< 1.01 \text{ mmol l}^{-1}$ in women $\geq 160/90 \text{ mm Hg}$ Microalbuminuria Urinary albumin excretion rate $\geq 20 \mu\text{g min}^{-1b}$ and/or Albumin:creatinine ratio $\geq 20 \text{ mg g}^{-1b}$
Body weight	
Elevated triglycerides	
Reduced HDL cholesterol	
Elevated blood pressure	
Other ^b	

Abbreviations: BMI, body mass index; HDL, high-density lipoprotein; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; T2DM, type II diabetes mellitus.

Adapted from Alberti and Zimmet.¹⁷

^aFasting glucose and self-reported diabetes mellitus were the only two measures of glucose regulation in the current study and were therefore the only measures used to identify impaired glucose regulation.

^bWaist-to-hip ratio and urinary albumin excretion rate were not performed in the current study and were therefore excluded from Metabolic Syndrome_{WHO}.

National Cholesterol Education Program Adult Treatment Panel III metabolic syndrome diagnostic criteria (2001)

<i>Clinical measure (diagnosis requires at least three of criteria)</i>	<i>Cut points</i>
Abdominal obesity (waist circumference)	$> 102 \text{ cm}$ in men $> 88 \text{ cm}$ in women
Elevated triglycerides	$\geq 1.69 \text{ mmol l}^{-1}$
Reduced HDL cholesterol	$< 1.01 \text{ mmol l}^{-1}$ in men $< 1.29 \text{ mmol l}^{-1}$ in women
Elevated blood pressure	$\geq 130 \text{ mm Hg}$ systolic blood pressure or $\geq 85 \text{ mm Hg}$ diastolic blood pressure
Elevated fasting glucose	$\geq 6.1 \text{ mmol l}^{-1}$ Includes diabetes mellitus ^a

Abbreviation: HDL, high-density lipoprotein.

Adapted from Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults.⁶

^aIn the current study, participants self-reported diabetes mellitus.

National Heart, Lung, and Blood Institute in collaboration with the American Heart Association metabolic syndrome diagnostic criteria (2005)

<i>Clinical Measure (diagnosis requires at least three of criteria^a)</i>	<i>Cut points</i>
Elevated waist circumference	≥ 102 cm in men ≥ 88 cm in women
Elevated triglycerides	≥ 1.69 mmol l ⁻¹ and/or On medication for elevated triglycerides
Reduced HDL cholesterol	< 1.01 mmol l ⁻¹ in men < 1.29 mmol l ⁻¹ in women and/or On medication for reduced HDL cholesterol
Elevated blood pressure	≥ 130 mm Hg systolic blood pressure and/or ≥ 85 mm Hg diastolic blood pressure and/or On antihypertensive medication in a patient with a history of hypertension
Elevated fasting glucose	≥ 5.6 mmol l ⁻¹ and/or On medication for elevated plasma glucose

Abbreviation: HDL, high-density lipoprotein.

Adapted from Grundy *et al.*⁷

^aIn our analysis, this definition was followed as reported.

International Diabetes Federation metabolic syndrome diagnostic criteria (2005)

<i>Clinical Measure</i>	<i>Cut points</i>
Elevated waist circumference ^a plus any of two factors below for diagnosis	≥ 94 cm in Europid men ≥ 80 cm in Europid women ≥ 90 cm in South Asian men ≥ 80 cm in South Asian women ≥ 90 cm in Chinese men ≥ 80 cm in Chinese women ≥ 85 cm in Japanese men ≥ 90 cm in Japanese women
Elevated triglycerides	≥ 1.69 mmol l ⁻¹ or On medication for elevated triglycerides
Reduced HDL cholesterol	< 1.01 mmol l ⁻¹ in men < 1.29 mmol l ⁻¹ in women or On medication for reduced HDL cholesterol
Elevated blood pressure	≥ 130 mm Hg systolic blood pressure or ≥ 85 mm Hg diastolic blood pressure or On antihypertensive medication in a patient with previously diagnosed hypertension
Elevated fasting glucose	≥ 5.6 mmol l ⁻¹ Includes diabetes mellitus ^b

Abbreviation: HDL, high-density lipoprotein.

Adapted from International Diabetes Federation.¹⁸

^aIn our analysis, Europid cut points for waist circumference were used for Caucasians, Native Canadians and for those of unknown ethnic origin (≥ 94 cm in men and ≥ 80 cm in women), while ethnic South and Central Canadian cut points were used for Hispanic Canadians (≥ 90 cm in men, ≥ 80 cm in women; *n* = 1).

^bIn the current study, participants self-reported diabetes mellitus.