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Review

A review of body mass index and waist circumference as markers of obesity and coronary heart disease risk in persons with chronic spinal cord injury

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Study design: Literature review.
Background: Increased fat mass and coronary heart disease (CHD) are secondary complications of chronic spinal cord injury (SCI). In able-bodied populations, body mass index (BMI, body weight (kg)/height (m^2)) is a widely used surrogate marker of obesity and predictor of CHD risk. Waist circumference, an accurate and reproducible surrogate measure of abdominal visceral adipose tissue, is also associated with CHD risk (more so than BMI) in able-bodied populations.
Objective: To review the literature on the accuracy of BMI and waist circumference as surrogate measures of obesity and CHD risk in persons with chronic SCI.
Setting: Ontario, Canada.
Methods: Literature review.
Results: In the SCI population, BMI is an insensitive marker of obesity, explains less of the variance in measured percent fat mass than in the able-bodied, and is inconsistently related to CHD risk factors. This may be due to potential measurement error, and to the inability of BMI to distinguish between fat and fat-free mass and to measure body fat distribution. Waist circumference has not been validated as a surrogate measure of visceral adipose tissue, however preliminary evidence supports a relationship between waist circumference and CHD risk in the SCI population.
Conclusions: We recommend that SCI-specific BMI classifications be determined. We also recommend that accuracy and reliability of waist circumference as a surrogate measure of visceral adipose tissue and CHD risk be determined in men and women with long-standing paraplegia and tetraplegia.
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Body mass index (BMI) as a marker of obesity in chronic SCI

Obesity is defined as an excess accumulation of fat mass. Able-bodied men and women $\leqslant 40$ years are considered obese when fat mass exceeds 22–25 and 35% of body weight, respectively.[1,2] As individuals age, fat mass accrues at the expense of fat-free mass, so that at older ages percentage fat mass is higher, even in individuals who do not gain weight.[3] Thus, obesity in 41–60-year-old able-bodied men and women can be defined as a fat mass >25 and $>38\%$ of body weight, respectively.[2] Mean percent fat mass (measured by dual energy X-ray absorptiometry (DXA), isotope dilution or the three-compartment model) reported in cross-sectional studies of persons (mostly men) with chronic spinal cord injury (SCI) ranges from 23 to 35%.[4–11] The percentage of body weight as fat mass is 8–18% higher in SCI *versus* age-, height- and/or weight-matched able-bodied control subjects. These values are often consistent with the above definitions of obesity, and are summarized in Table 1.

Accurate classification of an individual as normal weight, overweight or obese requires measurement of body composition. However, measuring fat mass can be difficult and expensive, and no accurate method is easily available for routine clinical use. Therefore, the body mass index (BMI) is widely used. Expressed as weight (kg) divided by height (m^2), BMI allows classification of able-bodied adults as underweight, normal weight,

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Table 1 BMI and measured percent fat mass in cross-sectional studies of adults with chronic spinal cord injury

Reference	*Study population(s)*	*Body composition method*	*BMI (kg/m^2)*	*Percent fat mass*
Modlesky *et al*, 2004[9]	Eight men with complete SCI (tetraplegic and paraplegic), 35 ± 9[a] years, duration postinjury not reported; eight age-, height- and weight-matched able-bodied male controls	DXA[b]	SCI: 24.6 Controls: 25.0 (P not reported)	SCI: 33.8 ± 16.4 Controls: 16.2 ± 8.7 ($P<0.05$)
Buchholz *et al*, 2003[7]	28 adults with paraplegia (17 men, 11 women; 18 complete, 10 incomplete), 33.9 ± 9.2 years, $\geqslant1.5$ years postinjury; 34 BMI-matched able-bodied controls (24 men, 10 women), 29.1 ± 7.6 years	Deuterium dilution	Paraplegic: 24.3 ± 6.0 Controls: 23.5 ± 1.8 ($P=0.8258$)	Paraplegic: 30.8 ± 8.7 Controls: 22.8 ± 7.2 ($P=0.0002$)
Jeon *et al*, 2003[11]	Seven men with complete tetraplegia, 38.3 ± 3.1 years (mean $\pm$ SEM); seven age-, weight-, height-, BMI- and waist circumference-matched able-bodied male controls	DXA	SCI: 26.7 ± 1.5 Controls: 29.4 ± 1.6 (NS)	SCI: 34.6 ± 7 Controls: 24.4 ± 6.5 ($P=0.016$)
Jones *et al*, 2003[6]	20 men with SCI (13 tetraplegic, seven paraplegic; five ASIA A, six ASIA B, one ASIA C, one ASIA D), 16–52 years, $\geqslant1$ year postinjury; 20 age-, height- and weight-matched able-bodied male controls	DXA	SCI: 23.1 ± 3.9 Controls: 24.0 ± 1.8 ($P=0.34$)	SCI: 27.5 ± 10.4 Controls: 18.1 ± 6.5 ($P<0.02$)
Maggioni *et al*, 2003[8]	13 men with SCI (one tetraplegic, 12 paraplegic), 33.8 ± 5.4 years, $\geqslant4$ years postinjury; 13 age- and BMI-matched able-bodied male controls	DXA	SCI: 25.7 ± 4.3 Controls: 24.5 ± 2.4 (NS)	SCI: 31.1 ± 8.2 Controls: 20.8 ± 6.9 ($P<0.05$)
Spungen *et al*, 2003[5]	133 men with SCI (66 tetraplegic, 67 paraplegic; 94 motor complete), approximately 38–40 years, approximately 11–14 years postinjury (group means not reported)	DXA	Tetraplegic: 25.4 ± 0.66 (mean $\pm$ SEM) Paraplegic: 25.8 ± 0.56	Complete tetraplegic: 34 ± 2 Incomplete tetraplegic: 35 ± 2 Complete paraplegic: 33.1 ± 1 Incomplete paraplegic: 28 ± 3
Desport *et al*, 2000[10]	20 adults with SCI (tetraplegic and paraplegic; 15 men, five women), 45.2 ± 12.8 years, $\geqslant4$ months postinjury	Three-compartment model (^{18}O dilution, skinfold thickness, weight)	26.9 ± 4.4	32.8 ± 6.8
Monroe *et al*, 1998[4]	10 men with SCI (one tetraplegic, nine paraplegic; all Frankel Class A), 35.5 ± 8.0 years, $\geqslant2$ years postinjury	DXA	21.7	23 ± 12

[a]Mean $\pm$ SD, unless otherwise indicated.
[b]Dual energy X-ray absorptiometry.

overweight or obese, using the World Health Organization criteria in Table 2.[12] Mean BMI values in studies of persons with long-standing SCI range from 20 to 27,[5,6,10,13–18] consistent with the classifications of normal and overweight, but inconsistent with classification based on measured percent fat mass.[4–11] One reason for the underestimation of obesity using BMI may be the potential measurement error associated with determination of weight and/or height in persons with SCI. While weight can be measured using a wheelchair scale, an accurate height is difficult to obtain in those who are wheelchair dependent. Ideally, height is measured with the subject standing against a stadiometer, with his/her head in the Frankfurt plane and shoulders, buttocks and heels pressed against the stadiometer.[19] This may not be possible in a significant proportion of persons with SCI. Subject recall of height is not recommended as recalled height and measured length have been found to disagree, independent of age or years since injury.[20] Despite this, recalled height has been used in a number of studies reporting BMI in subjects with chronic SCI.[5,16,17] An alternative is to measure length. Jones *et al*[21] reported the Pearson's correlation coefficient for height (measured by wall-mounted stadiometer) *versus* length (by the electronic ruler function of DXA) to be 0.996 ($P=0.0001$). Buchholz *et al*[22] found the

Table 2 World Health Organization[12] classification of adults according to the BMI

Classification	*BMI*	*Risk of comorbidities*
Underweight	<18.50	Low (but risk of other clinical problems increased)
Normal range	18.50–24.99	Average
Overweight/ preobese	25.00–29.99	Increased
Obese class I	30.00–34.99	Moderate
Obese class II	35.00–39.99	Severe
Obese class III	$\geqslant 40.00$	Very severe

coefficient of variation between height (by wall-mounted stadiometer) *versus* length (by length board) to be $0.8 \pm 0.02\%$ (mean $\pm$ SD, NS). Comparisons between methods in both studies were performed on able-bodied subjects; nonetheless, these data indicate good agreement between length and stadiometer-determined height.

A second potential reason for the underestimation of obesity using BMI is that persons with chronic SCI have greater fat mass and less fat-free mass per unit BMI than age-matched able-bodied control subjects.[6,22,23] Thus, despite greater fat mass in subjects with *versus* without SCI, body weight and BMI often do not differ.[5,6,8,9,11,22] Buchholz *et al*[22] found the $30\,kg/m^2$ BMI cutoff correctly identified only 20% of truly obese paraplegic subjects, as compared with published sensitivity values of 48–66% in able-bodied populations.[24–26] In the study of Buchholz *et al*, weight was measured using a wheelchair scale, length was measured using a length board, fat mass was determined by isotope dilution and obesity was defined by the method of Lohman.[2] Finally, BMI has been found to explain 46–79% of the variance in measured percent fat mass in able-bodied persons,[5,27–29] compared with only 35–36% in persons with SCI.[5,30]

Taken together, these findings suggest that BMI is a poor surrogate marker of obesity in the chronic SCI population. The $30\,kg/m^2$ cutoff recommended by the World Health Organization to define obesity in able-bodied persons is insensitive in persons with long-standing SCI. This may be due to potential measurement error and the inability of body weight to distinguish between fat mass and fat-free mass.

BMI as a marker of coronary heart disease (CHD) risk in chronic SCI

CHD is now a major cause of morbidity and mortality in persons with SCI.[31–33] A higher prevalence of CHD has been reported in individuals with duration of SCI greater than 10 years compared with relatively healthy age-matched controls.[34] SCI is associated with a number of risk factors for CHD. High-density lipoprotein (HDL) cholesterol levels are 20–42% lower ($P<0.05$) in persons with SCI than in able-bodied persons.[14,15,35–37] Triglyceride levels are 6–60% higher in SCI, although not always significantly so.[15,35–38] Total and low-density lipoprotein (LDL) cholesterols are either higher, similar, or lower, than in able-bodied subjects;[15,16,37,39,40] the relatively small number of subjects and differences in subject characteristics studied may account for these discrepancies. Impaired glucose tolerance, insulin resistance and diabetes occur more frequently in SCI *versus* able-bodied persons.[15,38,41–45] Other potential CHD risk factors after SCI include decreased physical activity, psychosocial factors (depression, isolation), and elevated plasma homocysteine and C-reactive protein.[46]

Increased fat mass has also been identified as an important risk factor in chronic SCI and weight management is recommended as a key CHD prevention strategy.[46] Obesity, and its surrogate BMI, is associated with many CHD risk factors in able-bodied persons, including dyslipidemia (increased LDL cholesterol and triglycerides, and increased HDL cholesterol), hyperinsulinemia, glucose intolerance and hypertension.[47–50] The World Health Organization has recognized the largely linear relationship between body weight and these risk factors when BMI increases from 20 to $30\,kg/m^2$, and has identified $30\,kg/m^2$ as the threshold above which risk for CHD is high.[12] This has led to the widespread use of BMI as a simple, cost-effective marker of obesity and CHD risk in able-bodied populations.[12,51] However, the relationship between BMI or body weight and CHD risk factors in the SCI population is variable. Zlotolow *et al*[14] found no relationship between BMI and lipid levels in their study of 28 veterans with paraplegia, nor did Bauman *et al*[42] find a significant correlation between body weight and insulin sensitivity in 100 veterans with SCI. BMI in other studies of persons with SCI has been found to explain 5–29% of the variance in lipid parameters.[16–18]

The variable relationship between BMI and CHD risk factors in the SCI population may be due to the potential measurement error associated with BMI, the insensitivity of body weight in distinguishing fat mass from fat-free mass, and the lack of information conveyed by BMI regarding body fat distribution. Abdominal obesity, specifically visceral adipose tissue, is an independent risk factor for CHD in able-bodied populations. Visceral adipose tissue is intra-abdominal fat bound by the parietal peritoneum, and is measured using computed tomography or magnetic resonance imaging. A visceral fat depot of $>130\,cm^2$ is associated with significant proatherogenic changes in the plasma lipoprotein–lipid profile as well as in indices of glucose–insulin homeostasis.[52] In his comprehensive review, Després[53] notes that abdominal visceral adipose tissue is characterized by very active lipolysis. Since high free-fatty acid levels have been shown to reduce the binding and uptake of insulin by hepatocytes, an enlarged visceral fat depot may expose the liver to high free-fatty acid levels, leading to a reduced hepatic extraction of

insulin. Thus, visceral adipose tissue is associated with hyperinsulinemia, insulin resistance and glucose intolerance. Furthermore, the activity of lipoprotein lipase (a lipolytic enzyme which hydrolyzes triglycerides into lipoproteins) in skeletal muscle is negatively correlated with *in vivo* insulin resistance, which could contribute to the impaired catabolism of triglyceride-rich lipoproteins observed in insulin-resistant subjects. The hypertriglyceridemia of abdominal obesity is associated with triglyceride enrichment of LDL and HDL cholesterols, leading to the production of dense LDL cholesterol (with a reduced affinity for the cellular LDL receptor), increased levels of apolipoprotein B and reduced plasma HDL cholesterol levels. Thus, visceral adipose tissue creates a metabolic environment consistent with increased risk of CHD. However, to the best of our knowledge, no study has measured visceral adipose tissue in persons with chronic SCI, despite the finding that total truncal fat mass is significantly greater in men with SCI *versus* age- and height-, weight- and/or BMI-matched able-bodied men.[6,8]

Waist circumference: the missing link?

Waist circumference may offer the clinician the most practical bedside measurement of visceral adipose tissue. A waist circumference >100 cm has been reported to be a good surrogate for a visceral adipose depot $>130\,cm^2$ in able-bodied men and women.[54] Waist circumference has been shown in large epidemiological studies of able-bodied populations to be strongly, significantly and independently correlated with blood pressure, dyslipidemia, fasting plasma glucose, 2-h plasma glucose and/or diabetes, even after adjusting for age and other confounding variables, and even among normal-weight subjects.[49,55,56] Furthermore, waist circumference was found to correlate more strongly than BMI with three of four obesity-related risk factors (LDL cholesterol, blood pressure, glucose, but not HDL cholesterol) in over 9000 participants of the third National Health and Nutrition Examination Survey,[50] similar to findings of other studies in the able-bodied literature.[57–60]

Waist circumference is measured in standing able-bodied adults using a measuring tape placed around the abdomen in a horizontal plane, with the subjects' arms hanging freely, after normal expiration.[61] Exact location of the measuring tape continues to be debated. A recent study[62] of 111 able-bodied males and females 7–83 years determined that waist circumference values measured at four sites (immediately below the lowest rib, at the narrowest waist, midpoint between the lowest rib and the iliac crest and immediately above the iliac crest) had equally high reproducibility and were almost equally associated with total body fat and trunk fat in each sex. The authors noted that of the 14 anatomical locations commonly reported in the literature, the narrowest waist is the most frequently recommended. In many subjects in their study, the narrowest waist was found to be at the lowest rib, a site the authors found easy to identify in most subjects, even in obese persons. The umbilicus may not be an appropriate landmark in obese persons because its position changes with increasing fat mass.

Three studies[11,15,63] have measured waist circumference in subjects with chronic SCI. Jeon *et al*[11] did not report values, nor was the method of obtaining waist circumference described. Maki *et al*[63] measured waist circumference in 46 men with paraplegia and tetraplegia of >6 months duration. Measures were carried out in duplicate at the level of the umbilicus after normal expiration, with subjects supine. If values differed by >1 cm, a third measurement was taken and the results of the two or three trials were averaged. Weight was determined by wheelchair scale. Height was either self-reported or measured in a supine position using a metal measuring tape. Fat mass was determined using near infrared interactance. Waist circumference explained 15–34% of the variance in HDL cholesterol, log 10 triglyceride as well as ratios of total:HDL cholesterols and LDL:HDL cholesterols, more so than BMI and percent fat mass (8–19 and 8–15%, respectively). Only waist circumference was significantly associated with HDL cholesterol. While these findings may indicate waist circumference to be a better indicator of CHD risk than BMI or percent fat mass, they need to be interpreted with caution. Self-reported height may have introduced measurement error in the calculation of BMI; as well, near infrared interactance has not been validated in the SCI population. This may have obscured the relationships between BMI, fat mass and CHD risk. Nonetheless, waist circumference was strongly and significantly associated with CHD risk. Demirel *et al*[15] measured waist circumference in 69 men and women with paraplegia and tetraplegia and 52 age- and sex-matched able-bodied controls. Measurements were made at the level of the umbilicus after normal expiration with subjects supine; measurement error was not reported. Waist circumference did not differ between the two groups (SCI: 84.8 ± 10 cm *versus* able-bodied: 85.7 ± 11 cm, mean $\pm$ SD), but the authors did report higher glucose, uric acid, total and LDL cholesterols and lower HDL cholesterol, and higher ratios of total/HDL cholesterols and LDL/HDL cholesterols in the subjects with SCI (all $P<0.001$). These proatherogenic changes may have been due to greater visceral *versus* subcutaneous adipose tissue in the SCI group, although this requires confirmation. Taken together, evidence from able-bodied populations suggests that waist circumference is strongly and independently associated, more so than BMI, with various CHD risk factors. Preliminary evidence in the SCI population supports an association between waist circumference and CHD risk factors. However, a number of issues pertaining to waist circumference have yet to be addressed in this population. These include (1) the accuracy and reliability of waist circumference as a surrogate measure of visceral adipose tissue, (2) identification of the most appropriate measurement site, (3) examination of the effects of positioning (supine, sitting, standing) and (4) effects of potential confounding variables unique to this popula-

tion, including spasticity, loss of muscle tone and abdominal distension.

Conclusion

In the SCI population, BMI may be prone to measurement error, does not adequately discriminate between the obese *versus* nonobese, explains less of the variance in measured percent fat mass than in able-bodied populations, and is inconsistently associated to CHD risk factors. However, BMI continues to be widely reported in the spinal cord literature. We recommend that future research efforts determine SCI-specific BMI obesity classifications.

Waist circumference, a reproducible surrogate measure of visceral abdominal adiposity, is associated with many CHD risk factors, more so than BMI, in able-bodied populations. Preliminary evidence supports a relationship between waist circumference and CHD risk factors in the SCI population. We recommend that accuracy and reliability of waist circumference as a surrogate measure of visceral adipose tissue, and the relationship between waist circumference and CHD risk factors, be determined in men and women with long-standing paraplegia and tetraplegia.

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