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ORIGINAL ARTICLE

Optimal scaling of weight and waist circumference to height for adiposity and cardiovascular disease risk in individuals with spinal cord injury

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Study design: Observational cross-sectional study.
Objectives: Body mass index (BMI), measured as a ratio of weight (Wt) to the square of height (Wt/Ht^2), waist circumference (WC) and waist-to-height ratio (WHtR) are common surrogate measures of adiposity. It is not known whether alternate scaling powers for height might improve the relationships between these measures and indices of obesity or cardiovascular disease (CVD) risk in individuals with spinal cord injury (SCI). We aimed to estimate the values of '*x*' that render Wt/Ht^x and WC/Ht^x maximally correlated with dual energy x-ray absorptiometry (DEXA) total and abdominal body fat and Framingham Cardiovascular Risk Scores.
Setting: Canadian public research institution.
Methods: We studied 27 subjects with traumatic SCI. Height, Wt and body fat measurements were determined from DEXA whole-body scans. WC measurements were also obtained, and individual Framingham Risk Scores were calculated. For values of '*x*' ranging from 0.0 to 4.0, in increments of 0.1, correlations between Wt/Ht^x and WC/Ht^x with total and abdominal body fat (kg and percentages) and Framingham Risk Scores were computed.
Results: We found that BMI was a poor predictor of CVD risk, regardless of the scaling factor. Moreover, BMI was strongly correlated with measures of obesity, and modification of the scaling factor from the standard (Wt/Ht^2) is not recommended. WC was strongly correlated with both CVD risk and obesity, and standard measures (WC and WHtR) are of equal predictive power.
Conclusion: On the basis of our findings from this sample, alterations in scaling powers may not be necessary in individuals with SCI; however, these findings should be validated in a larger cohort.
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INTRODUCTION

With advances in the acute care and management of spinal cord injury (SCI), affected individuals have a longer life expectancy, and as a consequence, secondary complications such as cardiovascular disease (CVD) are becoming a priority for researchers, clinicians and those living with SCI.[1] CVD is now the leading cause of morbidity and mortality in this population.[2] In addition, SCI individuals experience an increased risk, earlier onset and faster rate of progression of CVD than in the general population.[3,4] For example, individuals with SCI exhibit a more than twofold increased risk of stroke, heart disease and type 2 diabetes compared with able-bodied individuals.[5–7]

Obesity is a well-known risk factor for CVD, and is particularly important to examine following SCI as adverse changes in body composition, metabolic rate and autonomic function are known consequences of injury.[8,9] These adaptations, in combination with reduced activity levels as a result of physical disability, may lead to a higher prevalence of obesity and greater CVD risk in this population.[10] Thus, accurate and practical measures of obesity, coupled with better understanding of their relationships with CVD risk, are essential for this population.

Body mass index (BMI), measured as a ratio of weight to the square of height (Wt/Ht^2), has been used worldwide, and is espoused by the World Health Organization, as a simple proxy for obesity in the general population.[11] Although BMI does not specifically measure fat mass, population studies have shown that it correlates well with measures of body fat.[11] However, we often take for granted that BMI is measured as the ratio of weight to the square of height; other scaling powers for height are reported to be more strongly correlated with measures of obesity in the able-bodied.[12]

Other measures that have been used as surrogate markers for obesity in the able-bodied population are waist circumference (WC), waist-to-hip ratio (WHR), waist-to-height ratio (WHtR) and neck circumference.[13] BMI is often considered to be a 'gold standard' measure, but it underestimates obesity in those with SCI, probably because of decreases in muscle mass below the injury level.[14] Moreover, we are particularly interested in measures that incorporate WC,

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because they are well correlated with visceral fat, which is thought to be a key player in determining CVD risk.[15]

We recently showed that WC was the best measure of obesity-related CVD risk after SCI.[16] In this study, BMI was strongly correlated with adiposity, but not with CVD risk. However, the impact of different scaling factors for height correction in individuals with SCI is unknown, and may improve the relationships between these measures, as has been shown in the able-bodied.[12] We, therefore, aimed to determine the power of the scaling factors '*x*' of weight and WC with respect to height (Wt/Ht^x and WC/Ht^x) that are maximally associated with total body fat, abdominal body fat and Framingham CVD risk scores.

MATERIALS AND METHODS

Participants

This study represents a retrospective analysis of data previously collected.[16] The study received ethical approval from the Research Ethics Committee at Simon Fraser University and the Vancouver Coastal Health Research Institute. Measurements were taken on individuals with chronic SCI (>1 year), who gave written informed consent, had no known pre-existing (prior to injury) CVD and were not taking any cardiovascular-related medications.

Motor and sensory assessment

Neurological classification was conducted according to the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) to determine American Spinal Injury Association (ASIA) Impairment Scale (AIS) severity and neurological level of injury (the last spinal cord segment with fully preserved sensory and motor function).[17]

Weight, height, WC and body composition measurements

Weight (Wt) in kilograms was determined using a dual energy x-ray absorptiometry (DEXA) whole-body scan (QDR 4500, Hologic Inc., Bedford, MA, USA). Height in meters was determined using an electronic ruler (Matlab 2012b, Math Works, MA, USA) on the DEXA images, as previously described.[16] In cases where participants could not fully straighten their legs because of contractures or spasticity, self-reported height was used ($n=4$). WC was measured in centimeters, using a stretch-resistant tensiometer measuring tape, at the narrowest part of the waist after a normal expiration, while lying supine on the DEXA scanner bed. Total body fat mass in kilograms and total body fat percentage, computed as (total body fat mass/total mass) × 100, were determined using the whole-body DEXA scan. Abdominal fat mass in kilograms was determined using standardized landmarks[16] to distinguish the trunk region, and abdominal fat percentage was computed as (abdominal fat mass/total mass in the defined region) × 100.

Fasting lipid plasma levels

High-density lipoprotein cholesterol and total cholesterol levels were obtained to compute Framingham cardiovascular disease risk scores (described below). Venous blood samples were collected following a 12-h overnight fast (excluding water). Samples were centrifuged immediately at 3 °C and 3000 r.p.m. for 10 min, and the plasma component was withdrawn for subsequent analysis. The plasma samples were sent to the clinical laboratory at Vancouver General Hospital where high-density lipoprotein cholesterol and total cholesterol levels were determined by enzymatic assays (Dimension Vista system, Siemens Healthcare Diagnostics Inc., Malvern, PA, USA).

Framingham 30-year risk for CVD score

We used the Framingham 30-year risk for CVD score as a measure of overall risk of CVD.[18] This risk score incorporates the following risk factors: high-density lipoprotein cholesterol, total cholesterol, age, sex, systolic arterial pressure (SAP) at rest, smoking status, diabetes and antihypertensive treatments. However, instead of including the measured SAP, we entered a SAP value of 120 mm Hg into the risk score formula for all participants. This decision was based on the fact that SCI can impair normal blood pressure control with lesions at or above the fifth thoracic level, leading to lower resting blood pressure.[19] The known relationship between SAP and CVD risk, might, therefore, not exist in the same way in this population. Entering a value of 120 mm Hg is neutral to the score, and therefore excludes any effect of SAP on the generated risk score.[16] As a sensitivity analysis, we re-ran the same analyses using the original Framingham scores, which included the measured SAP.

Statistics

R Statistical Software Version 2.15.3[20] was used for all analyses and creation of plots. For each value of '*x*' ranging from 0.0 to 4.0, in increments of 0.1, the correlations between Wt/Ht^x and WC/Ht^x with total body fat percentage, total body fat (kg), abdominal fat percentage, abdominal fat (kg) and Framingham risk scores were computed and plotted, using Pearson's correlation coefficient (r). We defined a 'meaningful change' with respect to the strength of a correlation as plus or minus 0.05 from the maximum correlation coefficient. As there are already generally accepted standards in place for scaling of Wt/Ht^x and WC/Ht^x, we wanted to only consider further evaluation of a new standard of measurement if there was a meaningful change.

RESULTS

Participant characteristics

A total of 27 subjects with traumatic SCI (mean age ± standard deviation: 40 ± 11 years; mean time since injury: 14 ± 10 years; 70% male) participated in this study.

According to neurological levels, 59% had cervical injuries and 41% had thoracic injuries. The breakdown according to AIS severity was: 52% AIS A, 22% AIS B, 19% AIS C and 7% AIS D.

Summary statistics and bivariable relationships

Summary statistics (means, ranges and measures of variability) for all study measures are provided in Table 1. Figure 1 shows the correlation between BMI (Wt/Ht^2) with each of: Framingham risk scores (a); abdominal fat (b); and total fat (c). Figure 2 shows the correlation between WC with each of: Framingham risk scores (a); abdominal fat (b); and total fat (c). Figures 1, 2 and Table 1 reflect the ranges of BMI, WC, DEXA and Framingham risk measurements captured in the sample, as well as the varying correlations between the standard measures (BMI and WC) with Framingham scores and fat measures (abdominal and total), discussed in more detail below.

Comparisons between Wt/Ht^x and WC/Ht^x for CVD Risk

Figure 3 shows correlations of Wt/Ht^x at different scaling powers of '*x*' ranging from 0 to 4, with each of: Framingham risk scores (a); abdominal fat (b); and total fat (c). Figure 4 shows correlations of WC/Ht^x with these same measures. In Figures 3 and 4, when $x=2$, this is a typical BMI measurement; when $x=0$, this is the unscaled value for Wt. With respect to Wt/Ht^x and CVD risk, Wt/Ht^x appears to be a poor predictor of CVD risk, regardless of the scaling power: the maximum correlation coefficient is $r=0.29$, and correlation coefficients are not statistically significant at all scaling powers (Figure 3a).

Table 1 Summary statistics for study measures

Variable	*Range (minimum, maximum)*	*Mean*	*Standard deviation*
BMI (Wt/Ht^2; kg m^{-2})	(15.6, 34.8)	23.4	4.4
WC (cm)	(68, 111)	87.4	11.7
Framingham risk score	(2,29)	15.0	8.3
Abdominal body fat (kg)	(2.9, 18.2)	9.9	4.5
Total body fat (kg)	(7.1, 38.7)	20.3	8.2

Abbreviations: BMI, body mass index; WC, waist circumference.

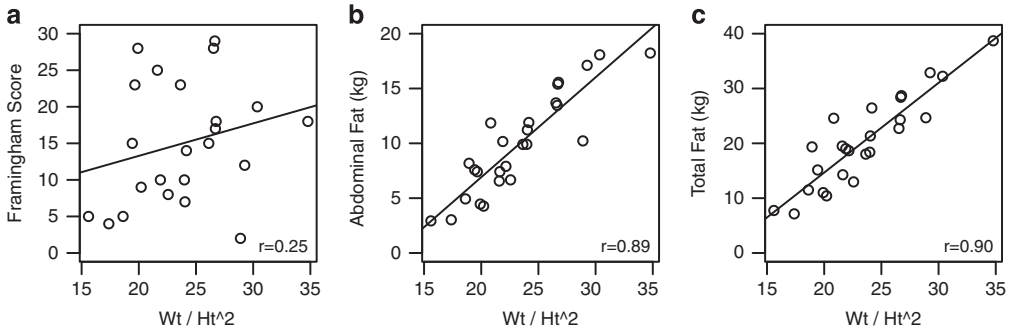

Figure 1 Bivariable relationships for BMI. Correlation between BMI (Wt/Ht^2) with each of: Framingham risk scores (**a**); abdominal fat (**b**); and total fat (**c**).

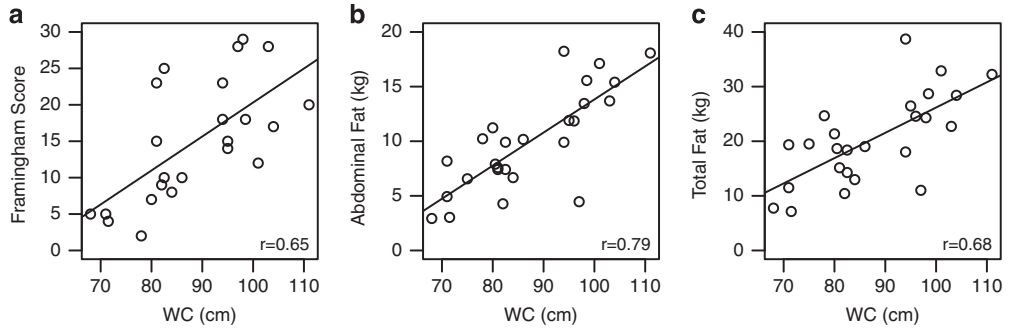

Figure 2 Bivariable relationships for WC. Correlation between WC with each of: Framingham risk scores (**a**); abdominal fat (**b**); and total fat (**c**).

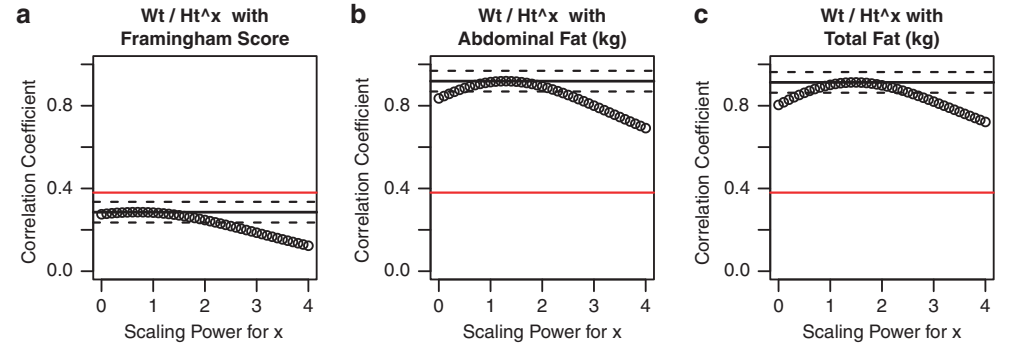

Figure 3 Correlations of Wt/Ht^x at different scaling powers of x ranging from 0 to 4, with each of: Framingham risk scores (**a**); abdominal fat (**b**); and total fat (**c**). The correlation coefficient at each value of x is indicated with overlapping open circles. The maximum correlation is indicated with a black line; dashed lines indicate the maximum correlation plus or minus 0.05, the limits of 'meaningful change'. The red line indicates the point above which the correlation coefficient is statistically significant ($P<0.05$).

In contrast, WC is more strongly and significantly correlated with CVD risk than Wt/Ht^x (Figure 4a). More specifically, WC is most strongly correlated with CVD risk when uncorrected for height, that is, when $x=0$, which is a commonly used standard; the maximum correlation coefficient is $r=0.66$ ($P<0.05$; Figure 4a). Moreover, the correlation coefficient is not 'meaningfully different' when $x=1$, another standard scaling power.

Comparisons between Wt/Ht^x and WC/Ht^x for obesity measures

Both Wt/Ht^x and WC/Ht^x generally showed strong and statistically significant correlations with measures of obesity (Figures 3b, c and Figures 4b, c, respectively). The maximum correlations with Wt/Ht^x for abdominal fat and total fat mass occurred at $r=0.92$ and $r=0.91$ at values of $x=1.3$ and $x=1.5$, respectively (Figures 3b, c). However, these maximum correlations were not meaningfully different than those obtained using the standard value of $x=2$. Thus, a typical BMI measurement remains a strong predictor of obesity.

The maximum correlations with WC/Ht^x for abdominal fat and total fat mass occurred at $r=0.82$ and $r=0.73$ at values of $x=0.60$ and $x=0.80$, respectively (Figures 4b, c). However, these maximum correlations were not significantly different compared with those at a value of $x=0$ or $x=1$ (standard scaling powers). Thus, typical WC and waist-to-height ratio measurements remain strong predictors of obesity.

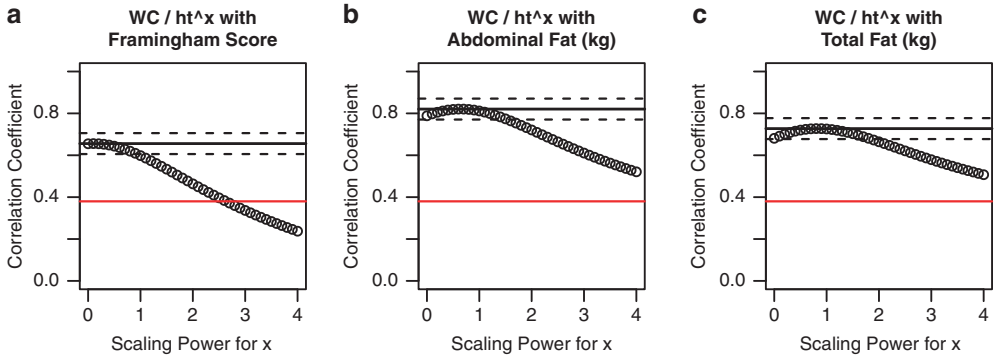

Figure 4 Correlations of WC/Ht^x at different scaling powers of *x* ranging from 0 to 4, with each of: Framingham risk scores (**a**); abdominal fat (**b**); and total fat (**c**). The correlation coefficient at each value of *x* is indicated with overlapping open circles. The maximum correlation is indicated with a black line; dashed lines indicate the maximum correlation plus or minus 0.05, the limits of 'meaningful change'. The red line indicates the point above which the correlation coefficient is statistically significant ($P<0.05$).

Table 2 Correlations at optimal scaling powers

Independent variable	*Dependent variable*	*Correlation at optimal scaling power*	*Meaningful benefit to optimized scaling*
Wt/Ht^x	Abdominal body fat (kg)	0.92	No
	Total body fat (kg)	0.91	No
	Abdominal body fat (%)	0.80	No
	Total body fat (%)	0.77	No
WC/Ht^x	Abdominal body fat (kg)	0.82	No
	Total body fat (kg)	0.73	No
	Abdominal body fat (%)	0.76	No
	Total body fat (%)	0.70	No

Correlations at optimal scaling powers are shown, for different combinations of dependent and independent variables. There was no meaningful benefit to the optimized scaling power for any of the variables tested.

Table 2 summarizes the maximum correlations for the absolute obesity measures, and also shows the maximum correlations for abdominal fat and total body fat percentages. As seen in the table, scaling of WC and Wt yielded stronger correlations with body fat measures in kg versus percentages. There was no meaningful benefit to the optimized scaling power for any of the variables tested (Table 2).

Multivariable analyses

As a follow-up analysis, we also performed multivariable regression with both WC and BMI as explanatory variables (covariates). These results were consistent with the bivariable results in that only WC/Ht^x measures were significant components of the model in relation to CVD risk, but both measures of WC/Ht^x and Wt/Ht^x were important in models for obesity. However, because of the limited sample size, the confidence intervals for the effect sizes were wide (results not shown).

Sensitivity analyses

We used a neutral value for SAP when calculating the Framingham risk score because of the known impact of high level SCI upon blood pressure control, whereby those with the most severe cardiovascular dysfunction tend to have lower resting blood pressure,[19] contrary to the case in the able-bodied. As a sensitivity analysis, we re-ran our analyses using the original Framingham scores, which included the measured SAP; the overall findings were the same as reported here (results not shown).

The majority of our participants were male; therefore, we performed an additional sensitivity analysis excluding females from the analytic sample. This also did not affect our findings (results not shown).

DISCUSSION

In this study, we aimed to evaluate whether alternate scaling powers for height might improve the relationships between WC/Ht and Wt/Ht with indices of obesity or CVD risk in individuals with SCI. Overall, we conclude that these standard measures were not improved by employing alternate scaling factors, and we reaffirm WC as a more valid and practical measure for obesity-related CVD risk in the SCI population. More specifically, our findings indicate that BMI is a poor predictor of CVD risk, regardless of the scaling factor. However, BMI is a strong predictor of obesity. Conversely, we showed that WC and waist-to-height ratio are strong predictors of both CVD risk and obesity.

Given the strong relationships between BMI and adiposity measures (abdominal and total body fat) after SCI, it is perhaps surprising that BMI is not a strong predictor of obesity-related CVD risk in this population. This may be because, despite being correlated with abdominal fat, BMI does not have the ability to differentiate between subcutaneous and visceral fat, the latter of which is thought to be the main contributor to CVD risk.[16] Indeed, the Framingham Risk Score incorporates measures of dyslipidemia, diabetes and blood pressure,[18] all of which are influenced more by visceral adiposity than abdominal or total body fat.[21] As WC is reported to better reflect visceral adiposity than BMI,[22] this perhaps explains why the risk score was better correlated with WC than with BMI. After SCI, there is increased visceral fat for a given weight compared with the able-bodied.[23] In

addition, SCI-specific changes below the lesion level, such as a reduced muscle mass, differentially affect BMI and WC, and thus the corresponding relationships with adiposity and CVD risk.

In the general population, Heo *et al.*[12] found the optimal scaling (for BMI) was $x=1.0$ in men and $x=0.8$ in women for maximal correlation with total body fat. They also found the optimal scaling for WC was zero, that is, no scaling for height.[12] Their findings with respect to BMI are quite different to the results of the present study, in which we found no meaningful benefit to scaling BMI from the standard $x=2$; this may reflect the unique anthropometric alterations that occur after SCI. In addition, the alternate scaling powers suggested by Heo *et al.*[12] may not reflect a 'meaningful change' from standard measures as we have described here.

Unfortunately, no prior studies have examined the differences in able-bodied populations with respect to absolute fat measures in kg versus body fat percentages in relation to scaling powers for Wt and WC. This might be of interest given our results in which stronger correlations were obtained with absolute than percentage fat data.

Given that obesity is associated with increased morbidity and mortality, the present findings may reinforce the need for accurate proxy anthropometric measures for adiposity and CVD risk. The development of obesity and CVD risk classification criteria based on optimal Wt/Ht indices and WC/Ht indices stratified for subgroups, such as individuals with SCI, will provide a more accurate assessment of the true burden of obesity. As such, we will be able to better understand the implications for obesity-related morbidity and mortality among these individuals. Indeed, individuals with SCI exhibit a more than twofold increased risk of heart disease, stroke and type 2 diabetes compared with the able-bodied, all of which are highly associated with obesity.[5,6] Furthermore, having accurate estimates for obesity and obesity-related CVD risk is an important consideration for statistical regression models where BMI and WC might be used to control for confounding.

Study limitations

The main limitation of this study is the relatively small sample size in this population, which limits the generalizability of these findings. With this limitation in mind, we were not able to differentiate optimal scaling powers for specific age categories, sexes, neurological levels of SCI and completeness of injury. We did, however, perform a sensitivity analysis excluding females from the analytic sample; this did not affect the overall results of the study. We also interpret the results of our multivariable models cautiously in light of the limited sample size. In addition, because of the limited power, we were not able to statistically compare correlation coefficients at different values of 'x', and instead used a criterion of 0.05 for a 'meaningful change'. These issues will be important to address in larger studies in the future.

Finally, the Framingham 30-year risk for CVD risk score was not designed or validated for use in an SCI population, but rather for the population as a whole, which may include individuals with many comorbidities. It is possible that some aspects of the risk score, most notably the resting blood pressure, should be modified for use in SCI where those with the most severe cardiovascular dysfunction tend to have lower resting blood pressure,[19] in contrast to the norm. Accordingly, we conducted our analyses using both a neutral blood pressure, and the participant's actual blood pressure; our findings were unchanged. Therefore, regardless of how the risk score is utilized, we are confident that at least in this small cohort, there was no benefit to CVD risk prediction with the use of alternate scaling measures for obesity.

DATA ARCHIVING

There were no data to deposit.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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