Obesity risks among Inuit

ORIGINAL ARTICLE

Obesity risks: towards an emerging Inuit pattern

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ABSTRACT

Objectives. The aim of this study was to provide analytical overviews of anthropometric measurements and their relationships with type 2 diabetes and cardiovascular disease (CVD) risk factors within the Inuit population, given that few studies have focused on this issue.

Study design. Cross-sectional study.

Methods. Anthropometric and biological data were obtained from 867 Inuit participants from Nunavik (≥18 years).

Results. Obesity prevalence for men and women, respectively, was 25.1% and 31.3% according to body mass index (BMI: >30 kg/m²); 20.2% and 55.3% according to waist circumference (WC: >102 cm for men and >88 cm for women); 22.4% and 22.5% according to body fat percentage (%BF: ≥30 in men and ≥40 in women). There was substantial agreement between anthropometric obesity measurements, except for the waist-to-hip ratio (WHR) which showed the lowest agreement with the other measurements. All risk factors were significantly associated with anthropometry. The prevalence of abnormal values for risk factors increased across quartiles of BMI and WC. Among obese participants, as defined by the WC cut-off, 22% had metabolic syndrome based on the National Cholesterol Education Program in the Adult Treatment Panel III (NCEP-ATPIII) definition and 64.8% of them were also insulin resistant.

Conclusion. Obesity rates among Inuit are high, especially among women. Inuit women display especially high rates of abdominal obesity. Further longitudinal work is needed to evaluate the effects of central and global obesity among Inuit.

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Keywords: obesity, Inuit, sitting height, Cormic Index, anthropometry, body mass index
INTRODUCTION

There is evidence that type 2 diabetes and cardiovascular disease (CVD) are emerging problems among Inuit of circumpolar countries (1–3). While there are indications that obesity is becoming more prevalent among Inuit, there is also evidence that the effect of adiposity within Inuit populations has a less-than-average magnitude on obesity-related CVD and diabetes risk factors (4–6), raising questions concerning the validity of existing anthropometric cutoffs used for Inuit. For example, the widely used body mass index (BMI) may overestimate the prevalence of overweight and obesity in Inuit populations due to their shorter legs and higher sitting heights compared to other populations (7,8).

Moreover, the apparent differences in the impact of obesity among Inuit as compared with other populations are reminiscent of results obtained in other populations where a healthy metabolic profile was observed among obese people (9). Thus, there is a need to fully characterize the extent to which different adiposity measures are related to diabetes and CVD risk factors within an Inuit context.

The aims of the study were to provide analytical overviews of the anthropometric data obtained from Inuit of Nunavik randomly selected for a health screening, as well as the relationships between these data and risk factors for diabetes and CVD.

MATERIAL AND METHODS

The data were collected aboard the CCGS Amundsen research icebreaker from August to October 2004. Households were randomly selected and 67.5% of adults agreed to participate. Anthropometric data were obtained from 867 Inuit adults residing in randomly selected households across 14 communities for the Nunavik Health Survey Qanuippitaa – How are we? (http://www.qanuippitaa.com). The total sample was composed of 392 men and 475 women; 6.1% had incomplete data on either height, weight, sitting height, waist circumference (WC) or body fat percentage (%BF).

Approvals for the Nunavik Health Survey were obtained from the Ethics Committees of Laval University, l’Institut national de santé publique du Québec (INSPQ) and McGill University. All other appropriate regional reviews were also obtained along with written informed consent from each participant.

Anthropometry and blood pressure measurements

WC was measured using a graduated, inelastic tape over light clothing at the point midway between the iliac crest and the last floating rib at the end of a normal exhalation. Hip circumference (HC) was quantified by placing the measuring tape horizontal to the hips at the pubic symphysis and the most prominent part of the buttocks. Both a waist-to-hip ratio (WHR) and a waist-to-height ratio (W/H) were calculated. Height was quantified with a stadiometer. Sitting height (SH) was measured using a sitting height table equipped with a movable stadiometer and an adjustable foot support (10). Also, if the standing height, sitting height,
WC or HC measurements fell between 2 millimetres, the even millimetre was recorded. SH was measured to calculate sitting-height-to-height ratios (SH/H) (Cormic Index) in order to adjust the observed BMI and obtain a standardized body mass index (BMlstd) (10–15). This adjusted measure has been proposed for Inuit (10) and involves conducting a linear regression of the SH/H relationship to obtain the intercept and beta coefficient (slope) for predicting BMI. The intercept and beta coefficient are then used in a formula to adjust the BMI for relative sitting height (10,11,12,13). In an earlier study with a small sample size of Inuit, the externally derived beta coefficients and intercepts published to date (11,12,13) were used to adjust BMI for SH/H (6). However, in the current study, which has a larger sample size, the original methodology as recommended by Norgan and Collins (10–15) was applied to the data to obtain an Inuit-specific intercept and beta coefficients for the adjustment procedure.

Weight and body fat composition were measured using a Tanita leg-to-leg bioelectrical impedance instrument (Tanita TBF-300; Tanita Corporation of America, Arlington Heights, IL). Body fat percentage (%BF) was classified according to age-specific body fat ranges (11). The %BF was used as a continuous as well as a dichotomous variable (high %BF: ≥30 in men and ≥40 in women). Obesity cut-offs used were as follows: BMI: ≥30 kg/m²; WC: >102 cm for men and >88 cm for women; WHR: ≥0.90 in men and ≥0.85 in women (16,17).

Blood pressure (BP) was measured according to the Canadian Coalition for High Blood Pressure technique using mercury sphygmomanometers, 15-inch stethoscopes and cuffs sized to the subjects’ arms. Prior to the BP measurement, the subjects had to rest for 5 minutes and were not to have eaten or smoked for at least 30 minutes (18). Three BP readings were taken for each subject. An average BP was calculated from the last 2 measurements. An individual was defined as having HTN (hypertension) if his/her BP was ≥140/90 mmHg (19) or if he/she had a previous diagnosis of HTN with or without medication. Hypertension diagnosis was ascertained by individual medical file review as detailed elsewhere (20). The mean of pulse pressure was defined as the difference between systolic blood pressure (SBP) and diastolic blood pressure (DBP).

**Biological parameters**

After centrifugation, all tubes were labelled and stored at -80°C onboard the CCGS Amundsen. They were then sent to the Centre Hospitalier de l’Université Laval (CHUL) where they were analysed for complete lipid profile, as well as for glucose and insulin concentrations. Biochemical measurements were performed with the Auto-Analyzer II (Technicon Instruments Corporation, Tarrytown, NY) and reagents from Roche Diagnostics (Laval, QC, Canada). Blood glucose was assessed by a spectrophotometric assay (Vitros 950, Vitros Chemistry Station, Ortho-Clinical Diagnostics, Raritan, NJ). Fasting plasma glucose (FPG) levels were assessed (reference values [RV]: 3.6–5.8 mmol/L), along with total cholesterol (T-Chol) and triacylglycerols (TG). TG levels were assessed by standard enzymatic methods with a Vitros 950 Chemistry Station (Ortho-Clinical Diagnostics, Raritan, NJ), including the manufacturer’s reagents. Calibration was achieved using the Vitros Chemistry Proto-
type Calibrator Kit 2 (Ortho-Clinical Diagnostics) and Vitros Performance Verifier fluids. High-density lipoprotein cholesterol (HDL-C) concentrations were quantified by the Vitros direct HDL-C slide assay (Ortho-Clinical Diagnostics), based on the precipitation of apolipoprotein-B-containing lipoproteins by sulfate dextran/magnesium chloride and magnetic beads. The HDL-C fraction was obtained after precipitation of the other lipoproteins. Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald formula (21). Fasting plasma insulin was measured by immunnoassay with chemiluminescent detection using the Elecsys-2010 system from Roche Diagnostics (Laval, QC, Canada). Reference values for insulin were 0–150 pmol/L. Insulin resistance was evaluated by homeostasis model assessment (HOMA-IR). HOMA-IR was defined as FPI (mU/L) * FPG (mmol/L)/22.5 (22). All methods for clinical biochemistry measurements have been previously described in detail (23).

**Statistical analyses**

Analyses were run separately for men and women except when specified. Descriptive statistics were used to provide an anthropometric overview of the Inuit from Nunavik. The parameters presented are accompanied by their corresponding standard error (SE). Numbers of participants are included for information only. All data were analysed by the bootstrap technique in order to account for the complex sampling strategy employed and to correct for related sampling errors. Analyses were also weighted to achieve population representativeness. Weights were adapted to the non-response rate of each measurement instrument. For normally distributed variables, arithmetic means were calculated and accompanied by their corresponding SE. Medians within the interquartile range (25–75%) were presented for variables with a log-normal distribution. Descriptive analyses based on gender comparisons were performed by classical ANOVA for normally distributed variables while quantile regressions (24) were performed for non-normally distributed variables. Furthermore, BMI was stratified into age groups to provide a comparison with the general Canadian population.

An analysis of agreement between anthropometric obesity measures for men and women combined was also made using the percentage of agreement and the kappa statistic. The proportion of agreement between obesity measures can be misleading because it is heavily influenced by the proportion of people with the obesity characteristics being compared. Therefore, the kappa statistic and the 95% confidence interval (CI) were used to adjust for the amount of agreement expected by chance alone (25). The kappa statistic gives values between 0 and 1, where values the nearest to 1 indicate a near-perfect agreement and where a value equal to 0 indicates an agreement totally due to chance (25). The relationship between anthropometric measures and diabetes or CVD indicators was measured using age- and gender-adjusted multiple linear regression. Furthermore, the same relationship was measured between age-adjusted quartiles of BMI and WC and the percentage of the sample with an abnormal risk factor. The cut-points used were: fasting glucose (≥5.6 mmol/L) (26); fasting insulin (≥107 pmol/L) (which is the 90th percentile value of the whole population) (27); insulin resistance (defined as HOMA-IR
in the top quartile of the distribution of the population without diabetes) (9); LDL-C (≥3.5 mmol/L) (28); HDL-C (<1.03 for men and <1.29 for women) (29); Chol/HDL ratio (≥6.0) (30); total cholesterol (≥5.17 mmol/L); TG (≥1.7 mmol/L) (29); elevated SBP (≥130 mm Hg); elevated DBP (≥85 mm Hg) (29); HTN (SBP ≥140 mm Hg or DBP ≥90 mm Hg). To avoid any misclassification, medications for diabetes, hypertension and lipid lowering were appropriately taken into account in the previously mentioned categories. Statistical significance was set at a p-value of ≤0.05. Statistical analyses were performed using SAS version 9.2 and SUDAAN 9.03.

**RESULTS**

Table I provides a descriptive overview of the anthropometric measurements and selected risk factors of CVD for the population under study. Participants’ ages ranged from 18 to 74 years with a mean of 36.0±0.3 (SE) years in men and 37.0±0.3 years in women. Most anthropometric parameters were statistically different between men and women, except for WC and WHR (Table I). However, when WC was categorized according to elevated WC (>102 cm and >88 cm) vs. low WC, we detected significant gender differences in visceral obesity (p<0.0001), the propor-

| Table I. Characteristics of a random sample of the Nunavik Inuit population. |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
|                                 | Men (n=392)     | Median IQR (25%-75%) | Women (n=475)   | Median IQR (25%-75%) | P-value |
| Age (years)                     | 36.0 (0.29)     | 33.0 (25.0–45.0)   | 37.0 (0.28)     | 35.0 (25.0–46.0)   | 0.16   |
| Height (cm)                     | 165.7 (0.29)    | 166.0 (162.0–170.0) | 153.9 (0.22)    | 154.0 (151.0–157.0) | <0.0001 |
| Sit height (SH) (cm)            | 88.7 (0.16)     | 88.6 (86.2–91.4)   | 83.1 (0.13)     | 83.2 (81.4–85.2)   | <0.0001 |
| Weight (kg)                     | 73.7 (0.80)     | 70.4 (62.0–83.0)   | 65.8 (0.67)     | 63.2 (54.4–75.2)   | <0.0001 |
| Waist (cm)                      | 91.0 (0.63)     | 88.0 (81.0–99.0)   | 91.5 (0.61)     | 90.0 (80.0–100.0)  | 0.47   |
| BMI (kg/m²)                     | 26.8 (0.26)     | 25.5 (22.9–29.8)   | 27.7 (0.27)     | 26.7 (23.2–31.1)   | 0.03   |
| Sitting-height-to-height ratio  | 0.54 (0.0006)   | 0.54 (0.53–0.54)   | 0.54 (0.0005)   | 0.54 (0.53–0.55)   | <0.0001 |
| Waist-to-hip ratio              | 0.89 (0.004)    | 0.89 (0.84–0.94)   | 0.89 (0.003)    | 0.90 (0.80–0.90)   | 0.53   |
| Waist-to-height ratio           | 0.55 (0.004)    | 0.54 (0.49–0.59)   | 0.59 (0.004)    | 0.58 (0.53–0.65)   | <0.0001 |
| Body fat (%)                    | 20.8 (0.37)     | 19.0 (14.9–26.3)   | 31.9 (0.39)     | 32.2 (24.4–39.1)   | <0.0001 |
| Insulin (pmol/L)                | 58.5 (3.40)     | 42.0 (34.0–57.0)   | 59.6 (1.9)      | 46.0 (36.0–69.0)   | 0.03   |
| Glucose (mmol/L)                | 4.9 (0.06)      | 4.5 (4.1–4.8)      | 4.5 (0.05)      | 4.3 (3.9–4.7)      | <0.001 |
| Homa-IR (n=328/396)             | 1.9 (0.18)      | 1.2 (0.9–1.7)      | 1.8 (0.07)      | 1.3 (0.9–2.1)      | 0.17   |
| Triacylglycerols (mmol/L)       | 1.2 (0.04)      | 1.0 (0.8–1.4)      | 1.1 (0.03)      | 1.0 (0.7–1.4)      | 0.04   |
| HDL-C (mmol/L)                  | 1.5 (0.02)      | 1.4 (1.2–1.7)      | 1.8 (0.02)      | 1.7 (1.5–2.1)      | <0.0001 |
| LDL-C (mmol/L)                  | 2.9 (0.05)      | 2.7 (2.2–3.6)      | 2.8 (0.04)      | 2.7 (2.2–3.3)      | 0.79   |
| Cholesterol (mmol/L)            | 4.8 (0.05)      | 4.8 (4.1–5.5)      | 5.1 (0.04)      | 5.0 (4.3–5.7)      | <0.01  |
| Cholesterol/HDL ratio           | 3.4 (0.05)      | 3.17 (2.6–4.1)     | 3.0 (0.04)      | 2.8 (2.3–3.5)      | <0.0001 |
| Systolic blood pressure (mmHg)  | 120 (0.77)      | 121 (111–128)      | 112 (0.66)      | 110 (106–120)      | <0.0001 |
| Diastolic blood pressure (mmHg) | 76 (0.57)       | 75 (70–80)         | 73 (0.48)       | 72 (66–78)         | <0.05  |

IQR: interquartile range.

*P-value based on quantile regression analysis.

**Analysis restricted to fasting individuals (n=730).**

(n=X/X): corresponds to the number of participants included in the analysis; fluctuations in values are due to the exclusion of participants with specific medications which could interfere with the measurement.

*Values have been rounded up for clinical meaning.

*P-value based on the median.

*HOMA-IR=(FPI mU/L * FPG mmol/L)/22.5.
Obesity risks among Inuit

The prevalence of obesity among men (25.1%) was lower than among women (31.3%) based on BMI ($\geq 30$ kg/m$^2$) ($p<0.0001$). Comparisons with data from the CCHS were performed (32). We stratified people with BMI $\geq 30$ kg/m$^2$ into age groups by gender and found that older Inuit men and women were on average more obese than the general Canadian population (men 55–74 years: Inuit 50.4% vs. CCHS participants 27.4%; women 55–74 years: Inuit 47.4% vs. CCHS participants 28.8%).

Analyses of the BMIstd (Norgan’s equation: $p>0.05$) (cf. “Methods” section) showed that the regression criteria for calculating the adjusted BMI were not met in the current population, and these measures were therefore not included in the presentation of data.

There was overall a good agreement between the obesity measures; the rate of agreement ranged from 65.8% to 90.5% (Table II). However, when agreement was assessed with the kappa statistic, the rate of agreement varied considerably (0.21–0.75). There was substantial agreement between BMI and %BF, whereas BMI and WC showed moderate agreement and WHR showed low agreement with both BMI and %BF.

Age- and gender-adjusted linear regressions showed that all anthropometric measures were significantly associated with all CVD risk factors. As expected, the anthropometric measures were inversely related to HDL-C and positively associated with all other CVD parameters (Table III). Interestingly, adiposity explained a higher percentage of variance for HDL-C than for other CVD risk factors. Furthermore, models using WC explained a higher proportion of variance in the risk factors while the WHR showed the weakest $R^2$ and slopes (Table III). The log transformation of dependent and independent variables allowed for comparison of the slopes of each model since coefficients are interpreted as percentages of changes (33). WC presented higher slopes than BMI. On the other hand, WC, waist-to-height ratio and WHR showed very similar beta coefficients. However, waist-to-height ratio captured a morphometric gender difference that was not detected using the WHR or WC alone (Table I).

When CVD risk factors were presented as the percentage of participants with an abnormal value according to quartiles of BMI and WC (as described in Table IV), a strictly monotone trend of participants with abnormal values was observed across the WC quartiles (Fig. 1). Interestingly, the trend for insulin and glucose had a flat segment followed by a drastic increase at the 4th quartile in both genders. Similar patterns were observed for BMI (data not shown).

Finally, we determined the metabolic profile of the surveyed population. According to the definition of the metabolic syndrome used by the National Cholesterol Education Program in the Adult Treatment Panel III (NCEP-ATPIII) (29), 9.7% (SE) of participants met the criteria. Among the participants classified as obese by the WC cut-off, 78% were free of metabolic syndrome. To further investigate this result, we stratified the sample population according to the insulin resistance threshold (top quartile of HOMA-IR). We thus observed that most participants who met the ATPIII metabolic syndrome criteria were also insulin resistant (64.8%, $p<0.0001$).
### Table II. Agreement between anthropometric obesity measures.

| Obesity Measures                  | % Agreement | Kappa (95% CI*) |
|-----------------------------------|-------------|-----------------|
| BMI (kg/m²) and waist (cm)        | 83.45       | 0.63 (0.60–0.65) |
| BMI (kg/m²) and body fat (%)      | 90.51       | 0.75 (0.73–0.78) |
| BMI (kg/m²) and waist-to-hip ratio| 69.43       | 0.31 (0.29–0.34) |
| Body fat (%) and waist (cm)       | 78.15       | 0.49 (0.46–0.51) |
| Body fat (%) and waist-to-hip ratio| 65.83       | 0.21 (0.18–0.24) |

* CI=Confidence interval.

### Table III. Associations between anthropometric measures and cardiovascular disease parameters adjusted for age and gender.

| Dependent variables# | Model with BMI (kg/m²) R² | Beta coefficient | SE | Model with body fat (%) R² | Beta coefficient | SE | Model with waist (cm) R² | Beta coefficient | SE | Model with waist-to-hip ratio R² | Beta coefficient | SE | Model with waist-to-height ratio R² | Beta coefficient | SE |
|----------------------|---------------------------|------------------|----|---------------------------|------------------|----|--------------------------|------------------|----|----------------------------------|------------------|----|----------------------------------|------------------|----|
| Insulin (pmol/L)     | 0.22                      | 1.15***          | 0.1 | 0.18                      | 0.55**           | 0.07 | 0.23                     | 1.6**            | 0.13 | 0.13                             | 1.9**            | 0.28 | 0.21                             | 1.63**           | 0.01 |
| Glucose (mmol/L)     | 0.37                      | 0.15***          | 0.02 | 0.37                      | 0.07**           | 0.01 | 0.39                      | 0.19**           | 0.04 | 0.37                             | 0.16**           | 0.08 | 0.39                             | 0.22**           | 0.04 |
| HOMA-IR#             | 0.27                      | 1.31***          | 0.11 | 0.23                      | 0.62**           | 0.07 | 0.28                      | 1.88**           | 0.07 | 0.18                             | 1.89**           | 0.15 | 0.27                             | 1.86**           | 0.15 |
| Triacylglycerols (mmol/L) | 0.18                      | 0.97***          | 0.08 | 0.16                      | 0.50**           | 0.05 | 0.19                      | 1.35**           | 0.11 | 0.10                             | 1.8**            | 0.24 | 0.17                             | 1.32**           | 0.11 |
| HDL-C (mmol/L)       | 0.33                      | -0.55**          | 0.05 | 0.30                      | -0.28**          | 0.03 | 0.33                      | -0.77**          | 0.06 | 0.26                             | -0.97**          | 0.13 | 0.31                             | -0.74**          | 0.07 |
| LDL-C (mmol/L)       | 0.17                      | 0.51**           | 0.06 | 0.21                      | 0.32**           | 0.03 | 0.15                      | 0.62**           | 0.08 | 0.10                             | 0.68**           | 0.15 | 0.15                             | 0.62**           | 0.08 |
| Cholesterol (mmol/L) | 0.22                      | 0.20**           | 0.03 | 0.24                      | 0.14**           | 0.02 | 0.20                      | 0.24**           | 0.05 | 0.18                             | 0.26**           | 0.10 | 0.19                             | 0.24**           | 0.05 |
| Cholesterol/HDL ratio| 0.28                      | 0.75**           | 0.05 | 0.27                      | 0.42**           | 0.02 | 0.27                      | 1.10**           | 0.07 | 0.16                             | 1.23**           | 0.15 | 0.25                             | 0.99**           | 0.07 |
| Systolic blood pressure (mmHg) | 0.28                      | 0.13**           | 0.01 | 0.26                      | 0.06**           | 0.01 | 0.28                      | 0.19**           | 0.02 | 0.25                             | 0.24**           | 0.05 | 0.27                             | 0.18**           | 0.03 |
| Diastolic blood pressure (mmHg) | 0.12                      | 0.19**           | 0.02 | 0.12                      | 0.11**           | 0.02 | 0.13                      | 0.27**           | 0.03 | 0.09                             | 0.40**           | 0.06 | 0.12                             | 0.27**           | 0.03 |

* Units are presented here only for reference, as all dependent and independent variables were log-transformed in the model.

** HOMA-IR=(FPI mU/L * FPG mmol/L)/22.5.

** p≤0.0001.

Models were adjusted for age, gender and specific medication when appropriate (lipid-lowering drugs for all lipids; blood pressure medication for blood pressure; diabetes drugs for insulin and glucose). Because variables were log-transformed, the beta coefficients reflect the percentage change in each dependent variable as predicted by a 1% change in each adiposity variable.

### Table IV. Waist circumference and body mass index quartile ranges.

| Waist (cm) | BMI (kg/m²) |
|-----------|-------------|
| Men Min.–max. | Women Min.–max. | Men Min.–max. | Women Min.–max. |
| Q1 66.00–81.00 | 63.00–80.00 | 17.50–22.90 | 17.20–23.30 |
| Q2 81.50–89.00 | 81.00–90.00 | 23.00–25.70 | 23.40–26.80 |
| Q3 89.50–99.00 | 90.00–101.00 | 25.80–30.20 | 26.90–31.30 |
| Q4 99.50–136.00 | 102.00–136.00 | 30.20–45.80 | 31.50–48.00 |
Figure 1. Cardiovascular risk factors among people with large waist circumference (WC ≥102 or 88 cm) adjusted for age and stratified by gender and specific medication when appropriate. (Quartile values are presented in Table IV.)
DISCUSSION

When compared to the general Canadian population, the prevalence of obesity among Inuit adults in Nunavik is alarming (32,34). This finding is of particular concern among women. This statement, which has been affirmed in previous studies, was underlined by the present results and was the starting hypothesis for our analytical overviews of the Inuit anthropometry.

Based on previous observations among different populations, using a population’s sitting-height-to-height ratio to standardize the observed BMI may better represent the prevalence of obesity and subsequent risk for chronic disease among that population (10). However, in the present case, the regression criteria required to formulate a population-based BMIstd were not met, which raises questions regarding the use and validity of the adjusted values obtained from these equations. We suspect that the narrow distribution of the sitting height in this population is responsible for this aberrant result. In future studies, the possibility of using external standards and/or additional regressions of SH/H vs. BMI with pooled data from the Inuit health surveys will be explored.

Many authors debate on the best indicator of cardio-metabolic risk or mortality in the literature (35,36). In the present analysis we examined 5 simple anthropometric measures, but none of them stood out from the others regarding their relationship with traditional cardio-metabolic risk, even after age and gender adjustments.

The moderate (80%) agreement between the BMI and the WC confirms that both measures provide different information on body fat distribution; for example, BMI informs about an index of general adiposity and WC about central fat accumulation. Among this Inuit population, BMI and %BF were closely related. The strength of the association varies widely according to ethnic origin (35,37) and to the technique used (35). However, only comparisons with a gold standard technique will allow for a firm conclusion on the distribution of adiposity. To our knowledge, this has not yet been realized. Interestingly, WHR showed the lowest agreement with global adiposity indicators (i.e., BMI and %BF). This result is reminiscent of an analysis of a large case control study in which WHR showed the highest odds for myocardial infarction worldwide, as compared to BMI (36). Yusuf et al. have therefore argued for the need to redefine obesity indicators according to ethnic origin (36). The latter results point to the fact that, to our knowledge, no longitudinal study has examined the potential impact of obesity on metabolic or cardiovascular events among the Inuit population. This implies that the true ability of adiposity indicators to predict the risk of CVD in this population is still unknown.

However, from a cross-sectional perspective, all anthropometric measures in this study population had similar associations with CVD parameters found in other populations. In general, the increasing trends of the abnormal clinical parameters across the BMI and WC quartiles were similar to findings derived from other populations (38). However, the insulin resistance pattern observed through adiposity quartiles is noteworthy, and raises questions on how remarkably low diabetes rates can be observed despite the prevalent obesity rate, independently of the definition used for the latter.

In the present study, women in particular had a tendency towards abdominal fat patterning, which would increase the risk for obesity-related health complications (31,39,40). Susceptibility of women to central fat patterning has been observed in other northern populations (41), the Canadian Cree First Nation (42) and southern
American Indians (41). However, there is some evidence that Inuit women might be less affected by abdominal fat. For instance, elevated TG (≥1.7 or 150 mg/dl) was less common among Inuit Nunavik women (12.9±1.6%) and Yup'ik Alaskan women (18±1.4%) (41) than among U.S. white women (25.0±1.1%) and Navajo Indian women (47.0±0.9%) (41). Similarly, low HDL-C (<1.29 mmol/L or 50 mg/dl for women) was less prevalent among Nunavik Inuit women (12.8±1.6%) than Yup'ik Alaskan women (22.0±1.5%), U.S. white women (39.0±1.9%) or Navajo Indian women (50.0±0.9%). The prevalence of high blood pressure was also lower among Nunavik Inuit (data not shown) than in other groups (41).

Taken together, these various cases from study populations comparable to the Nunavik Inuit suggest that the paradox of a high prevalence of central fat patterning and a low prevalence of obesity-related CVD risk factors among Inuit clearly deserves further investigation.

One hypothesis is that women in the present study population may accumulate more subcutaneous fat than visceral fat, which in turn would confer some protection against obesity-related complications, although such a possibility cannot presently be evaluated in the current study.

While this report is limited by its lack of a non-Inuit reference group, our findings corroborate earlier studies that suggested that high BMI among Canadian and Greenlandic Inuit was associated with fewer metabolic consequences than among Caucasian Canadians and Danes (5,43,44). In this study, 78% of obese individuals (based on WC or BMI cut-points) were free of metabolic syndrome according to the ATPIII definition (29). Based on the same definition, 37% of obese individuals from a community-based study in United States did not present metabolic syndrome or significantly increased related risks (9).

To further explore this aspect, we stratified the population sample according to insulin resistance status (HOMA-IR top quartile). We observed that 64.8% of abdominally obese Inuit participants were insulin resistant. The latter observation is consistent with a recent analysis which proposes insulin resistance as a better prognostic factor than WC among first-degree relatives with type 2 diabetes (45).

However, results presented above should be considered with caution in light of the limitations of the study design. Indeed, its cross-sectional nature precludes any etiological relationship to be drawn between anthropometric measures and risk factors. Furthermore, the relatively young age of the population might be of great importance for the results obtained herein, particularly with regard to the absence of metabolic consequences. Nevertheless, the population-based status of the study confers an important external validity to the study.

The metabolically healthy obese (MOH) or MOH-like concept is relatively new and its definition varies from one study to another (46). Current estimates of its prevalence within the global population of those with obesity vary from 12 to 37% (9,47,48). In the Inuit population, firm conclusions about the exact rate of MOH or MOH-like phenotype are still premature. However, our results clearly stress the need for an accurate description of the health impact of obesity via a follow-up.

In conclusion, obesity is highly prevalent among Nunavik Inuit, especially among women. Furthermore, even though all anthropometric measures had similar associations with CVD parameters, the agreements between several anthropometric measures suggest that a measure of central obesity is important for characterizing risk among Inuit. Further longitudinal work is
needed to re-evaluate obesity as a risk factor and, more importantly, to determine the effects of central obesity among Inuit populations.

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