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# Relationships between metabolic markers and obesity measures in two populations that differ in stature—The SAMINOR Study

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## Summary

**Background:** The relationships between metabolic markers and obesity measures may differ by ethnicity, sex, and height. Questions have been posed whether these relationships differ by ethnicity in the population in Northern Norway, but this has not been explored yet.

**Objectives:** Investigate the relationships between metabolic markers and obesity measures in Sami and non-Sami and explore the impact of stature.

**Methods:** In total, 13 921 men and women aged 30 and 36 to 79 years (22.0% Sami) from a population-based cross-sectional survey in Norway, the SAMINOR 1 Survey (2003-2004, 57.2% attendance), were included. Relationships between triglycerides, high-density lipoprotein cholesterol, glucose, systolic/diastolic blood pressure (BP), metabolic syndrome and diabetes mellitus as outcomes, and body mass index (BMI), waist circumference (WC), and waist-to-height ratio (WHtR), respectively, were modelled using fractional polynomial regression. Appropriate interaction analyses and adjustments were made.

**Results:** The non-Sami were approximately 6 cm taller than the Sami. No interactions were found between ethnicity and obesity. At the same levels of WC, BMI, or WHtR, levels of lipids and BP differed marginally between Sami and non-Sami, but these were eliminated by height adjustment, with one exception: At any given WC, BMI, or WHtR, Sami had approximately 1.4 mmHg (95% CI, −2.1 to −0.7) lower systolic BP than non-Sami (*P* values < .001).

**Conclusions:** Height explained the marginal ethnic differences in metabolic markers at the same level of obesity, except for systolic BP, which was lower in Sami than in non-Sami at any given BMI, WC, or WHtR.

KEYWORDS

body mass index, ethnicity, metabolic syndrome, waist circumference

## 1 | INTRODUCTION

The relationships between obesity measures, body fat, and metabolic markers in various populations are a research priority of several health organizations.[1,2] In Asian populations, the World Health Organization has recommended lower body mass index (BMI)/waist circumference (WC) cut-offs because they are predisposed to disease at low levels of obesity.[3] In other ethnically diverse populations, such as in New Zealand, Greenland, Canada, and in the United States, findings diverge and implications for clinical practice are uncertain.[4-7]

The Sami is an ethnic minority and indigenous people living mainly in the northern parts of Norway, Sweden, and Finland and on the Kola Peninsula in Russia. In the last four decades, research from Norway has shown variations in obesity levels between people with and without Sami affiliation.[8-11] Sami women have repeatedly been shown to have higher BMI and/or larger WC than non-Sami women.[8-11] Yet researchers have observed differences concerning diabetes mellitus (DM) prevalence comparing the two groups with lower risks of DM in Sami than in non-Sami women in 1974–1975,[9] similar in 2003–2004,[12] and higher in 2012–2014.[11] In contrast, Sami men have previously been shown to have a lower WC than non-Sami men,[11,12] although recent reports show that Sami men have a higher prevalence of DM[11] and a higher severity score of metabolic syndrome (MetS) than non-Sami men.[13] However, no studies have explicitly examined the relationships between metabolic markers and obesity measures in this population.

As cut-offs for obesity should be population specific,[1] researchers have questioned the need for ethnic-specific cut-offs in Northern Norway.[8,12] On average, Sami populations have lower statures than non-Sami Norwegian populations.[9,11] Short people with a given WC are likely to be relatively fatter and have higher metabolic risk than tall people with the same WC.[14] Therefore, the aim of this study was to evaluate whether the relationships between metabolic markers and various obesity measures differ between Sami and non-Sami and to investigate the impact of stature on these relationships.

## 2 | METHODS

Data from the first survey of the population-based study on health and living conditions in regions with Sami and Norwegian populations—the SAMINOR Study—were used. The SAMINOR Study is run by the Centre for Sami Health Research at UiT The Arctic University of Norway. The first SAMINOR Survey was carried out in collaboration with the National Institute of Public Health during 2003 to 2004 in 24 rural municipalities in Northern and Central Norway.[15] Everyone who was 30 or 36 to 79 years old and registered in the National Registry as residents in the predefined areas was invited (27 987 individuals). In total, 16 014 (57%) attended the clinical examination and gave informed consent to participate in medical research. Trained personnel performed all clinical measurements and blood sampling. If pathologic measures were found, participants were encouraged to visit their primary physician. Researchers/health workers who are either Sami or work in Sami core areas have been consulted in order to meet the needs of the Sami community. This study has been approved by the SAMINOR Project Board and The Regional Committee for Medical and Health Research Ethics.

### 2.1 | Metabolic markers

Triglycerides, high-density lipoprotein (HDL) cholesterol, glucose, and systolic and diastolic blood pressure (BP) were included as dependent variables. BP was measured with a Dinamap-R automatic device (Critikon, Tampa, Florida, USA). Following at least 2-minute seated rest, three BP measurements with 1-minute intervals were recorded; the average of the second and third measurements was used in the analyses. Blood samples, taken nonfasting due to examination throughout the day, were drawn by venipuncture in a seated position. Samples were centrifuged within 1.5 hours, and serum was sent by overnight post to the laboratory at Ullevål University Hospital, Oslo. Lipids and glucose were measured by an enzymatic method (Hitachi 917 autoanalyzer, Roche Diagnostic, Switzerland). DM was based on self-report or current use of glucose-lowering drug (further details below). MetS was defined as having two or more of the following four metabolic abnormalities: hypertension (systolic BP ≥ 130 mmHg or diastolic BP ≥ 85 mmHg or use of BP-lowering drug), hypertriglyceridemia (triglycerides ≥ 1.7 mmol/L), reduced HDL cholesterol (HDL-C < 1.0 mmol/L in men and <1.3 mmol/L in women or use of cholesterol-lowering drug), or hyperglycaemia (glucose ≥ 11.1 mmol/L or DM). Although commonly included in the MetS definition,[2] WC was excluded from the criteria in order to avoid circular reasoning. Missing values in biochemical variables or BP measurements existed in less than 0.3% of cases.

### 2.2 | Obesity measures

WC was recorded to the nearest centimetre at the umbilicus with the participant breathing normally in a standing position. Height was measured to the nearest 0.1 cm, and weight was measured to the nearest 100 g, using an electronic height and weight scale with participants wearing light clothing and no shoes. BMI was calculated as weight in kilograms (kg) divided by height in metres raised to the second ($kg/m^2$), and waist-to-height ratio (WHtR) was calculated as WC divided by height measured in centimetres. Missing values in these measurements existed in less than 0.5% of cases.

### 2.3 | Lifestyle and drug use

Information on the following lifestyle factors were obtained from the questionnaire (answer options in parenthesis): education in years, alcohol consumption (never/not this year/a few times during this year/1 time per month/2-3 times per month/1 time per week/2-3

times per week/4-7 times per week), and smoking (currently/previously/never). Alcohol consumption was dichotomised into "weekly alcohol consumption" and "less than weekly alcohol consumption." Smoking was dichotomised into "current smoker" and "not current smoker."

Participants were asked about their leisure-time physical activity (PA) the last year through a question that has shown moderate validity.[16] One out of four categories were available: reading, watching television, or engaging in sedentary activities (sedentary); at least 4 hours a week of walking, bicycling, or other types of PA (light); at least 4 hours a week of participating in recreational athletics or heavy gardening (moderate); and regular, vigorous training or participating in competitive sports several times a week (hard). The latter two categories were merged into one, "medium/hard," because of low number in the "hard" category.

Participants were asked about DM (yes/no), use of BP-lowering drug (currently/previously, but not now/never), use of cholesterol-lowering drug (currently/previously, but not now/never), use of insulin (currently/previously, but not now/never), and use of glucose-lowering drug in tablet format (currently/previously, but not now/never). In addition to questions regarding specific medication, participants were asked to list any medication they had used within the last 4 weeks. These were later coded with ATC codes. Three drug variables were created—use of cholesterol-lowering drug, BP-lowering drug, and glucose-lowering drug—by combining responses to the drug-specific questions and the ATC codes that had cholesterol/BP/glucose-lowering (side) effects (see Supporting Information for details).

Responses were ad-hoc imputed by assuming that those who did not reply to questions concerning drug use (BP-lowering drug, n = 122; cholesterol-lowering drug, n = 288; glucose-lowering drug, n = 506) or DM (n = 477) were nonusers/did not have DM. Missing values existed for the following variables (percent missing in non-Sami men, Sami men, non-Sami women, and Sami women, respectively): leisure-time PA (7.3%, 9.1%, 10.4%, and 10.0%), alcohol consumption (2.0%, 3.4%, 3.5%, and 4.2%), and smoking (0.8%, 0.9%, 1.0%, and 0.7%).

## 2.4 | Ethnic categorisation

In Norway, it is by law illegal to register ethnicity in any registry or medical records, but for research purposes, it is permitted to ask about ethnic background. The questionnaire included three facets of ethnicity—language, ethnic background, and self-perceived ethnicity—making up in total eleven questions: *What language do/did you/your parents/your grandparents speak at home? What is your, your father's and your mother's ethnic background? What do you regard yourself as?* Alternatives were (more than one alternative was permitted) Norwegian, Sami, Kven (an ethnic minority of descendants of Finnish immigrants in the 1700s and 1800s), or other. Two criteria for Sami ethnicity were defined in this study. Participants had to answer Sami as

1. home language for at least one of their grandparents, parents, or themselves, and
2. their own ethnic background or self-perceived ethnicity.

All others were categorised as non-Sami.

## 2.5 | Final study sample

Participants were excluded if they failed to hand in the questionnaire (n = 213), did not answer any of the eleven ethnicity-related questions (n = 52) or questions regarding leisure-time PA (n = 1421), smoking (n = 80), or alcohol consumption (n = 240). Further, participants were excluded if they had missing information on any of the anthropometric measures (height, weight, or waist circumference, n = 59) or metabolic markers (triglycerides, HDL cholesterol, glucose, or systolic or diastolic BP, n = 28). A total of 13 921 subjects (7124 women and 6797 men, 50% of the invited population) were eligible for complete-case analysis (see Figure S1 for flow-chart).

## 2.6 | Statistical analyses

STATA version 15.1 (StataCorp, College Station, TX, USA) was used. Statistical code can be made available upon request. Sample characteristics are presented for each stratum of sex and ethnic group. Continuous variables are given as mean (standard deviation) or median (interquartile range) where appropriate; categorical variables are given as numbers (percentage). Because the relationships between metabolic markers and obesity may be non-linear, models were fitted using fractional polynomial regression, which is an extension of conventional polynomial regression.[17] It is implemented with the "fp" function in STATA and allows for $m$ degrees of the continuous predictor $X$ (the obesity measure in this case), with $p_1 \ldots p_m$ powers, which are chosen from {−2, −1, −0.5, 0, 0.5, 1, 2, 3}, where 0 means $\log(X)$.[17] In epidemiology, it is usually sufficient with $m = 2$.[18] Alpha ($\alpha$) was set to.05 for selection of powers. In a closed selection procedure using maximum likelihood, models with different $m$ are compared with a linear model; the linear fit is chosen unless a more complex model fits the data better.

Initially, interactions between sex and WC/BMI/WHtR and ethnicity and BMI/WC/WHtR were tested for using the "mfpigen" function.[19] Significant interactions ($P < .05$) were found between sex and obesity in models with HDL cholesterol and diastolic BP as outcomes; these models were therefore stratified by sex. No significant interactions were found between ethnicity and obesity. Ethnicity was therefore included as a covariate. All models were adjusted for age, age squared, smoking, alcohol consumption, leisure-time PA, and sex (except in sex-stratified models). In models with triglycerides and HDL cholesterol as dependent variables, additional adjustment was made for current use of cholesterol-lowering drugs. In models with glucose as dependent variable, adjustment was made for DM (including users of glucose-lowering drugs) and current use of cholesterol-lowering

drugs, because of its potential influence on glucose metabolism.[20] In models with systolic and diastolic BP, adjustment was made for current use of BP-lowering drugs.

Models were inspected visually for heteroscedasticity and nonnormality of residuals. All outcome variables were log-transformed because of nonnormality, and normality was confirmed. In models that still had heteroscedasticity, robust standard errors were computed. Results were back-transformed and plotted with the "marginscontplot2" function, which estimates average marginal effects with 95% confidence intervals by ethnic group (holding all other covariates constant). After plotting the models for visual presentation, all models were additionally adjusted for height.

## 2.7 | Sensitivity analyses

Several sensitivity analyses were conducted. First, the ethnicity variable was replaced with a variable indicating whether a subject was "short" or "tall," based on having a value below or above the sex-specific mean height in the sample (161 cm in women and 174 cm in men). Second, a three-level category of Sami ethnic markers was used. This was created by counting the number of "Sami answers": answered "Sami" on all 11 questions, 1 to 10 questions, or no questions. Third, the analyses were restricted to a presumably healthy sample, excluding individuals with DM (including those using glucose-lowering drugs), previous stroke, angina or myocardial infarction, and current use of cholesterol- or BP-lowering drugs. Fourth and finally, a multiply imputed data set was created, and all models were repeated using this data set. Multiple imputation is challenging when combined with fractional polynomials, mainly because of non-linearity in the models, and for not being able to use maximum likelihood in the model selection procedure.[21] Regarding the former, however, this was not viewed as an issue, as there was less than 0.5% missing in the fractional polynomial variables. Therefore, all missing data in the original sample, except the 52 individuals with missing ethnic information (N = 15 749), were imputed using multiple imputation chained equation. A total of 20 datasets were imputed using a "rich dataset" in order to make the missing-at-random assumption more likely. Fractional

**TABLE 1** Sex- and ethnicity-stratified sample characteristics in the SAMINOR 1 Survey (2003-2004, N = 13 921)

| | Women (N = 7124) | | Men (N = 6979) | |
|---|---|---|---|---|
| | Sami (N = 1538) | Non-Sami (N = 5586) | Sami (N = 1494) | Non-Sami (N = 5303) |
| Age, y | 52.5 (11.3) | 53.2 (11.4) | 54.1 (11.0) | 54.0 (11.2) |
| Education, y | 11.5 (4.6) | 11.7 (3.8) | 10.7 (4.1) | 11.3 (3.7) |
| Current smoker | 504 (32.8%) | 1747 (31.3%) | 490 (32.8%) | 1638 (30.9%) |
| Weekly alcohol consumption | 203 (13.2%) | 1211 (21.7%) | 389 (26.0%) | 1748 (33.0%) |
| Leisure-time PA | | | | |
| Sedentary | 437 (28.4%) | 1253 (22.4%) | 371 (24.8%) | 1229 (23.2%) |
| Light >4 h/w | 933 (60.7%) | 3686 (66.0%) | 795 (53.2%) | 2940 (55.4%) |
| Moderate-hard >4 h/w | 168 (10.9%) | 647 (11.6%) | 328 (22.0%) | 1134 (21.4%) |
| Diabetes mellitus | 68 (4.4%) | 258 (4.6%) | 66 (4.4%) | 225 (4.2%) |
| Metabolic syndrome | 597 (38.8%) | 2102 (37.6%) | 681 (45.6%) | 2460 (46.4%) |
| Cholesterol-lowering drug | 188 (12.2%) | 651 (11.7%) | 252 (16.9%) | 802 (15.1%) |
| BP-lowering drug | 328 (21.3%) | 1165 (20.9%) | 327 (21.9%) | 1179 (22.2%) |
| Glucose-lowering drug | 53 (3.4%) | 185 (3.3%) | 53 (3.5%) | 170 (3.2%) |
| Height, cm | 156.7 (6.0) | 162.4 (6.4) | 169.4 (6.4) | 175.4 (6.8) |
| Waist circumference, cm | 85.5 (12.2) | 85.2 (11.9) | 92.6 (10.7) | 94.6 (10.5) |
| Body mass index, kg/m$^2$ | 28.2 (5.0) | 27.3 (4.8) | 27.8 (4.0) | 27.5 (3.8) |
| Waist-to-height ratio | 0.547 (0.082) | 0.525 (0.076) | 0.547 (0.064) | 0.540 (0.060) |
| Triglycerides, mmol/L | 1.31 (0.97, 1.91) | 1.29 (0.93, 1.81) | 1.58 (1.10, 2.34) | 1.56 (1.09, 2.24) |
| HDL cholesterol, mmol/L | 1.45 (0.37) | 1.49 (0.39) | 1.26 (0.35) | 1.26 (0.33) |
| Glucose, mmol/L | 5.24 (4.81, 5.84) | 5.27 (4.87, 5.82) | 5.42 (4.99, 6.01) | 5.40 (4.97, 6.00) |
| Systolic BP, mmHg | 127.4 (20.2) | 129.2 (20.9) | 133.6 (19.5) | 134.1 (18.1) |
| Diastolic BP, mmHg | 71.8 (9.8) | 72.5 (10.2) | 77.5 (9.6) | 78.0 (10.0) |

*Notes.* Numerical variables are given in mean (standard deviation), except triglycerides and glucose, which are given in median (1st quartile, 3rd quartile). Categorical variables are given in frequency (percent).

Abbreviations: BP, blood pressure; HDL, high-density lipoprotein; PA = physical activity; h/w = hours per week.

polynomial models were then fitted on the multiply imputed data using the "mfpmi" command in STATA, which utilises log-likelihood type tests.[21]

All statistical tests had a two-sided significance level of.05. Because of a large sample size and multiple testing, strong emphasis was put on effect sizes in the interpretation of the results.

## 3 | RESULTS

### 3.1 | Sample characteristics

Table 1 shows sample characteristics by ethnic group (22.0% were categorised as Sami). Non-Sami of both sexes were on average approximately 6 cm taller than Sami.

### 3.2 | Relationships between metabolic markers and obesity measures

The relationships between metabolic markers and obesity measures were the same in Sami and non-Sami (no significant interactions), but there were some differences in the levels of metabolic markers between Sami and non-Sami at the same level of the obesity measure.

Visualisations of the *estimated* relationships concerning the three measures of obesity (WC, BMI, and WHtR), and triglycerides, glucose, systolic BP, MetS, and DM are found in Figure 1, and sex-stratified models for HDL cholesterol and diastolic BP are found in Figure 2.

There were no ethnic differences in glucose levels or probabilities of DM with respect to any obesity measure.

At any given WC, Sami had higher levels of triglycerides (+0.04 mmol/L, 95% confidence interval [CI], 0.01-0.07) and, in

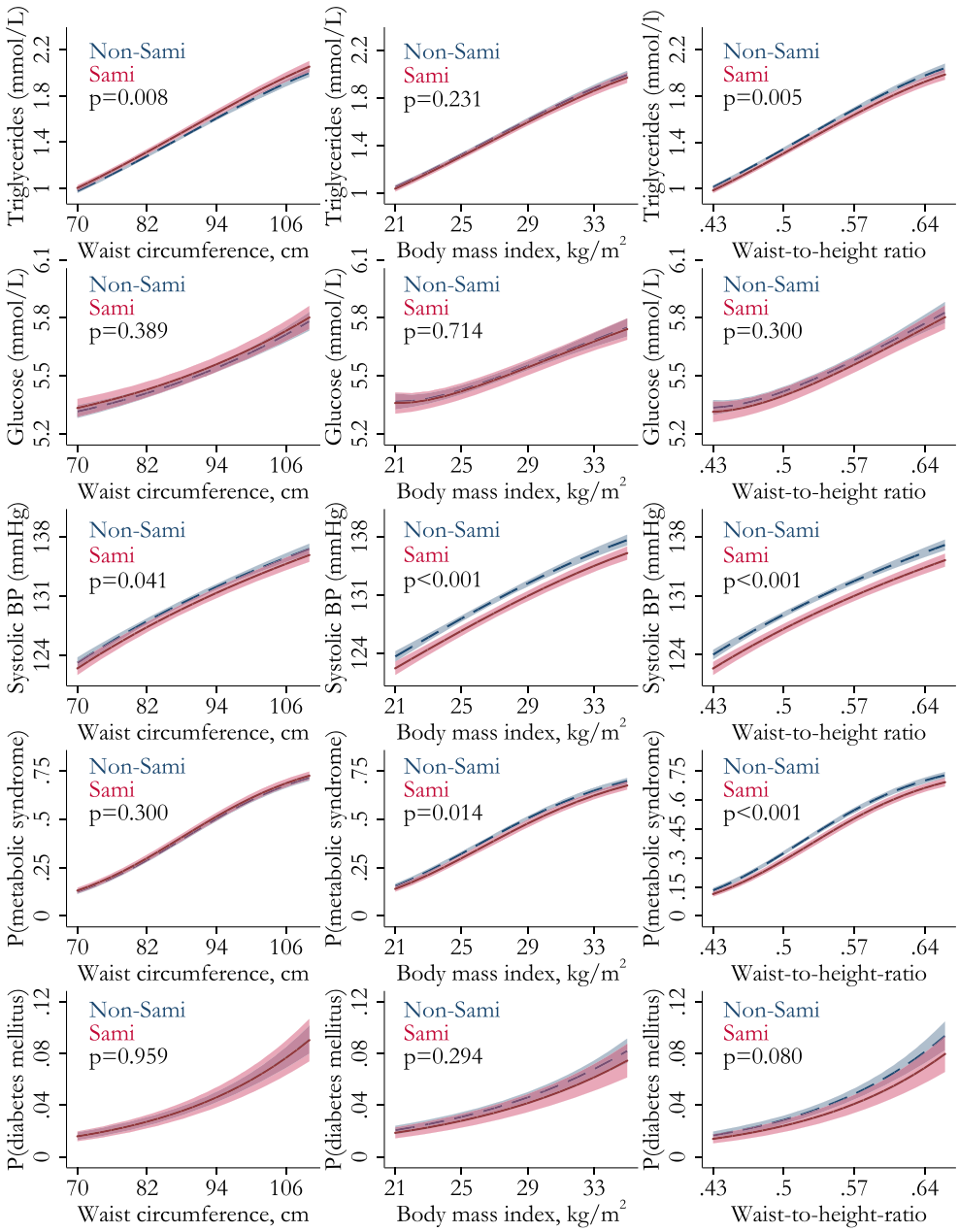


**FIGURE 1** Estimated relationships between metabolic markers and obesity measures in Sami vs non-Sami. All models were fitted with fractional polynomial regression and adjusted for age, age squared, leisure-time PA, smoking, alcohol consumption, sex, and relevant use of medication. Curves are drawn separate for Sami (red, solid line) and non-Sami (blue, dashed line). *P* values are for Sami vs non-Sami. Average marginal effects for each ethnic group were estimated, holding all other variables in the model constant, for the 5th to the 95th percentile of the obesity measure. BP, blood pressure

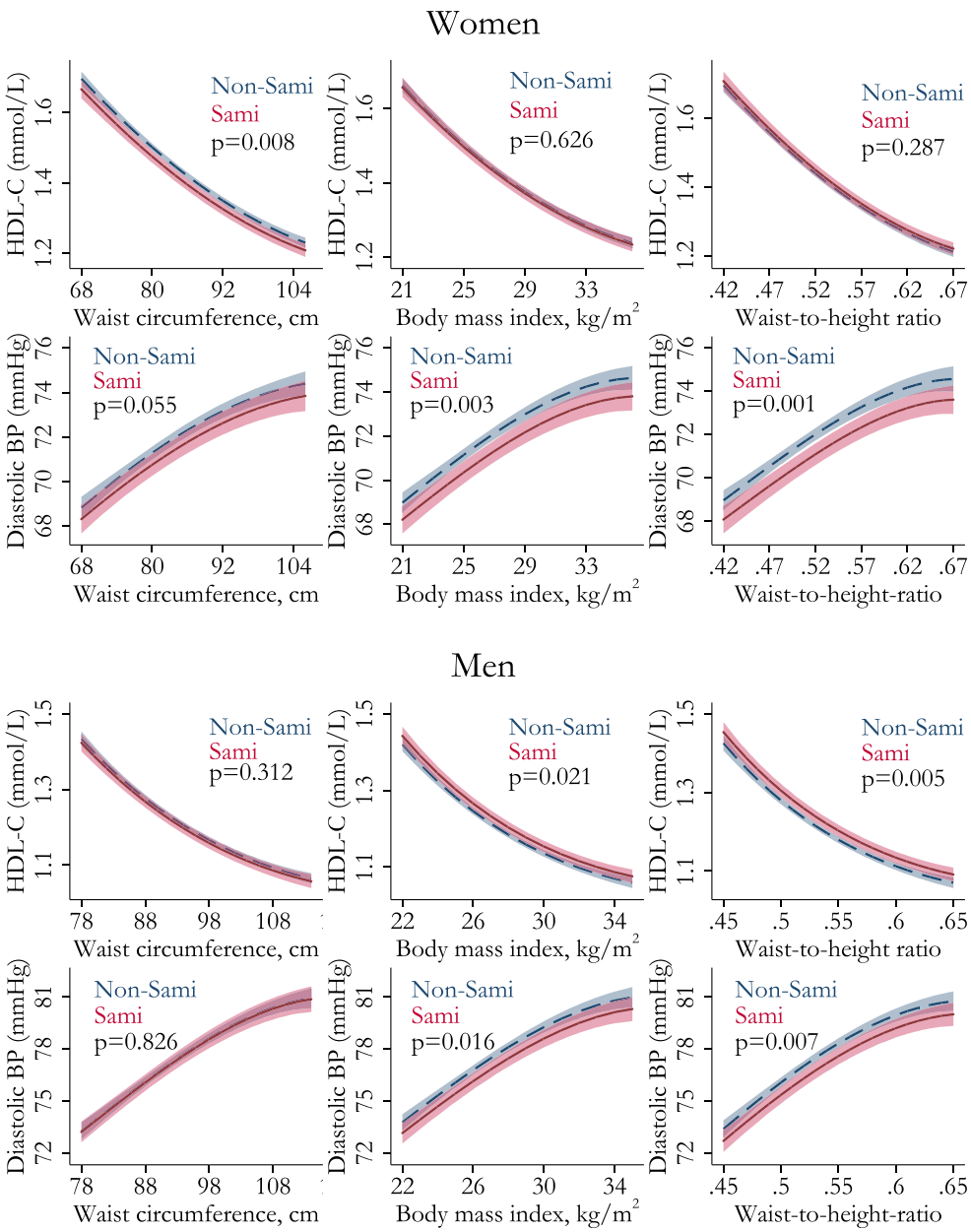


**FIGURE 2** Estimated sex-stratified relationships between metabolic markers and obesity measures in Sami vs non-Sami. All models were fitted with fractional polynomial regression and adjusted for age, age squared, leisure-time PA, smoking, alcohol consumption, and relevant use of medication. Curves are drawn separate for Sami (red, solid line) and non-Sami (blue, dashed line). *P* values are for Sami vs non-Sami. Average marginal effects for each ethnic group were estimated, holding all other variables in the model constant, for the 5th to the 95th percentile of the obesity measure. HDL-C, high-density lipoprotein cholesterol. BP, blood pressure

women, lower levels of HDL cholesterol (−0.03 mmol/L, 95% CI, −0.04 to −0.01) than non-Sami. However, at any given WC, Sami had more favourable levels of systolic BP than non-Sami (−0.70 mmHg, 95% CI, −1.37 to −0.03) (Table 2).

At any given BMI, Sami had more favourable levels of several metabolic markers than non-Sami. Levels of HDL cholesterol in men were higher (+0.02 mmol/L, 95% CI, 0.00 to 0.04). Levels of systolic (−1.50 mmHg, 95% CI, −2.16 to −0.83) and diastolic BP (in women, −0.81 mmHg, 95% CI, −1.34 to −0.27; in men, −0.64 mmHg, 95% CI, −1.17 to −0.12) and probability of MetS (−0.02, 95% CI, −0.04 to −0.00) were lower in Sami than in non-Sami at any given BMI (Table 2).

Models with WHtR showed similar ethnic differences as in models with BMI. Compared with non-Sami, Sami had lower levels of triglycerides (−0.04 mmol/L, 95% CI, −0.07 to −0.01), higher levels of HDL cholesterol in men (+0.02 mmol/L, 95% CI, 0.01 to 0.04), lower levels of systolic (−1.73 mmHg, 95% CI, −2.40 to −1.07) and diastolic BP (in women, −0.92 mmHg, 95% CI, −1.46 to −0.38; in men, −0.72 mmHg, 95% CI, −1.25 to −0.20), and probability of MetS (−0.04, 95% CI, −0.05 to −0.02) at the any given WHtR (Table 2).

When adjusting the models for height, most of the ethnic differences in metabolic markers were attenuated and lost statistical significance except in models with systolic BP or MetS as dependent variables ($Model_{heightadj}$ in Tables 3–5). Effect sizes concerning MetS were small, whereas effect sizes concerning systolic BP were substantial, and all *P* values were <.001: Compared with non-Sami, Sami had 1.37 mmHg (95% CI, −2.09 to −0.66) lower systolic BP at any given WC, 1.45 mmHg (95% CI, −2.16 to −0.73) lower at any given BMI, and 1.38 mmHg (95% CI, −2.10 to −0.67) lower at any given WHtR (results not shown).

**TABLE 2** Estimated average marginal effects with 95% confidence intervals (CI) for Sami vs non-Sami in main models

| | Waist Circumference | | | Body Mass Index | | | Waist-to-Height Ratio | | |
|---|---|---|---|---|---|---|---|---|---|
| Metabolic marker | AME | 95% CI | N | AME | 95% CI | N | AME | 95% CI | N |
| Triglycerides, mmol/L | 0.04 | 0.01, 0.07 | 13 921 | −0.02 | −0.05, 0.01 | 13 921 | −0.04 | −0.07, −0.01 | 13 921 |
| HDL-C, women, mmol/L | −0.03 | −0.04, −0.01 | 7124 | −0.00 | −0.02, 0.01 | 7124 | 0.01 | −0.01, 0.03 | 7124 |
| HDL-C, men, mmol/L | −0.01 | −0.02, 0.01 | 6797 | 0.02 | 0.00, 0.04 | 6797 | 0.02 | 0.01, 0.04 | 6797 |
| Glucose, mmol/L | 0.02 | −0.02, 0.06 | 13 921 | −0.01 | −0.05, 0.03 | 13 921 | −0.02 | −0.07, 0.02 | 13 921 |
| Systolic BP, mmHg | −0.70 | −1.37, −0.03 | 13 921 | −1.50 | −2.16, −0.83 | 13 921 | −1.73 | −2.40, −1.07 | 13 921 |
| Diastolic BP, women, mmHg | −0.53 | −1.07, 0.01 | 7124 | −0.81 | −1.34, −0.27 | 7124 | −0.92 | −1.46, −0.38 | 7124 |
| Diastolic BP, men, mmHg | −0.06 | −0.59, 0.47 | 6797 | −0.64 | −1.17, −0.12 | 6797 | −0.72 | −1.25, −0.20 | 6797 |
| Metabolic syndrome (probability) | 0.01 | −0.01, 0.03 | 13 921 | −0.02 | −0.04, −0.00 | 13 921 | −0.04 | −0.05, −0.02 | 13 921 |
| Diabetes mellitus (probability) | −0.00 | −0.01, 0.01 | 13 921 | −0.00 | −0.01, 0.00 | 13 921 | −0.01 | −0.01, 0.00 | 13 921 |

*Notes.* The average marginal effects are estimated from the models, which were adjusted for age, age squared, smoking, alcohol consumption, leisure-time PA, relevant drug use, and sex (except in sex-stratified models). Average marginal effects are computed by fixing the value for ethnicity, but keeping the other variables in the models (those adjusted for) at their observed values in the sample. The probability/mean for each case is calculated, and then all estimates are averaged across the sample. This is done by fixing the ethnicity variable first at Sami, then at non-Sami. The average marginal effects for Sami and non-Sami are then compared. As all other variables except ethnicity are identical between the two hypothetical populations, the difference in the averaged mean/probability are attributed to the fixed variable: ethnicity.

Abbreviations: AME, average marginal effects; BP, blood pressure; CI, confidence interval; HDL, high-density lipoprotein cholesterol; N, sample size.

### 3.3 | Sensitivity analyses

Overall, sensitivity analyses agreed with the main analyses (Tables 3–5). In models evaluating stature, short people were found to have markedly less favourable levels of most markers at any given WC ($Model_{short/tall}$ in Table 3 and Figure 3), and somewhat better levels of most markers at any given WHtR ($Model_{short/tall}$ in Table 5), than tall people.

## 4 | DISCUSSION

In this population-based study from parts of rural Northern and Central Norway, the relationships between metabolic markers and WC, BMI, or WHtR were the same in Sami as in non-Sami. Sami and non-Sami had some differences in levels of metabolic markers, but these differences were only marginal in size. Adjusting the models for height eliminated practically all ethnic differences, but not regarding systolic BP, which was lower in Sami than in non-Sami at any given WC, BMI, or WHtR.

Two other findings with public health implications should be noted: First, short people had worse metabolic profile at any given WC compared with tall people; second, increases in obesity were associated with sharp increases in the probability of MetS.

Some results from studies on metabolic markers and obesity in other ethnically diverse Arctic populations are relevant for comparisons. At the same level of BMI, both the Greenlandic and Canadian Inuit had more favourable levels of BP and lipids, but not glucose and insulin, than their respective non-Inuit reference population.[22,23] On the other hand, the South Asian, Chinese, and Aboriginal descendant Canadians (from the Six Nation Reserve) had less favourable levels of cardiometabolic risk factors than European descendant Canadians at the same level of BMI.[24] An exception was for systolic BP, which was approximately 5 mmHg lower in Aboriginal than European descendant Canadians.[24] This resembles the findings in this study, although the effect sizes were much larger than in this study (approximately 5 vs 1.4 mmHg).

In a study comparing Pima Indians and White Americans, autonomic nervous system activation seemed to differ between the two groups, possibly explaining why Pima Indians have a lower prevalence of hypertension but a higher prevalence of obesity than Whites.[25] There is no reason to believe that the physiological response to obesity differ in Sami and non-Sami, but an intriguing question is whether they have different amounts/types of body fat at the same levels of obesity. For instance, a study found that Greenlandic Inuit and Kenyans had less adipose tissue at the same levels of obesity as Danes.[5] Currently, there are no such data available, but it is important to emphasise that throughout history, the Sami have lived side by side the majority Norwegian population and a large part of the population in Northern Norway have ethnically mixed ancestry. On the contrary, Pima Indians and Greenlandic Inuit have lived as isolated populations. Any physiologic difference in response to obesity or body composition between Norwegians with and without Sami affiliation therefore seems highly unlikely. The possibility of chance findings or residual confounding cannot be ruled out either.

The relationship between height and disease in a context with Sami ethnicity has previously been discussed: Ethnic differences in stroke were in general reduced when controlling for height,[26] and in women, height was inversely associated with both DM and myocardial infarction independently of ethnicity.[9] Height is largely determined by genetics, and whether individuals utilise their full genetic potential is considered to be influenced by environmental factors in utero[27] and in childhood.[28] Perhaps by being a marker of unfavourable environments, short stature is associated with an

**TABLE 3** Analyses for waist circumference

| | Triglycerides | HDL, Women | HDL, Men | Glucose | SBP | DBP, Women | DBP, Men | MetS | DM |
|---|---|---|---|---|---|---|---|---|---|
| **$Model_{main}$** | | | | | | | | | |
| Log-β/*OR | 0.03 | −0.02 | −0.01 | 0.00 | −0.01 | −0.01 | −0.00 | 1.05* | 0.99* |
| 95% CI | 0.01, 0.05 | −0.03, −0.00 | −0.02, 0.01 | −0.00, 0.01 | −0.01, −0.00 | −0.01, 0.00 | −0.01, 0.01 | 0.96, 1.15 | 0.81, 1.22 |
| *P* value | .008 | .008 | .312 | .389 | .041 | .055 | .826 | .300 | .959 |
| N | 13 921 | 7124 | 6797 | 13 921 | 13 921 | 7124 | 6797 | 13 921 | 13 921 |
| **$Model_{heightadj}$** | | | | | | | | | |
| Log-β/*OR | −0.02 | −0.00 | −0.00 | −0.01 | −0.01 | −0.01 | −0.00 | 0.89* | 0.83* |
| 95% CI | −0.04, 0.00 | −0.01, 0.01 | −0.02, 0.01 | −0.01, 0.00 | −0.02, −0.01 | −0.02, 0.00 | −0.01, 0.01 | 0.81, 0.98 | 0.67, 1.02 |
| *P* value | .117 | .891 | .870 | .201 | <.001 | .094 | .648 | .016 | .081 |
| N | 13 921 | 7124 | 6797 | 13 921 | 13 921 | 7124 | 6797 | 13 921 | 13 921 |
| **$Model_{short/tall}$** | | | | | | | | | |
| Log-β/*OR | 0.08 | −0.03 | −0.01 | 0.01 | 0.01 | −0.00 | 0.00 | 1.35* | 1.44* |
| 95% CI | 0.06, 0.09 | −0.04, −0.02 | −0.02, −0.00 | 0.01, 0.02 | 0.00, 0.01 | −0.01, 0.01 | −0.00, 0.01 | 1.25, 1.46 | 1.21, 1.71 |
| *P* value | <.001 | <.001 | .039 | <.001 | .005 | .655 | .532 | <.001 | <.001 |
| N | 13 921 | 7124 | 6797 | 13 921 | 13 921 | 7124 | 6797 | 13 921 | 13 921 |
| **$Model_{imputed}$** | | | | | | | | | |
| Log-β/*OR | 0.02 | −0.02 | −0.00 | 0.01 | −0.00 | −0.01 | 0.00 | 1.03* | 1.10* |
| 95% CI | 0.00, 0.04 | −0.03, −0.00 | −0.02, 0.01 | −0.00, 0.01 | −0.01, 0.00 | −0.01, −0.00 | −0.00, 0.01 | 0.94, 1.12 | 0.92, 1.31 |
| *P* value | .033 | .011 | .501 | .131 | .077 | .045 | .529 | .545 | .314 |
| N | 15 749 | 8233 | 7516 | 15 749 | 15 749 | 8233 | 7516 | 15 749 | 15 749 |
| **$Model_{healthy}$** | | | | | | | | | |
| Log-β/*OR | 0.04 | −0.01 | −0.01 | 0.00 | −0.01 | −0.00 | −0.00 | 1.01* | |
| 95% CI | 0.01, 0.06 | −0.03, 0.00 | −0.02, 0.01 | −0.01, 0.01 | −0.01, −0.00 | −0.01, 0.00 | −0.01, 0.01 | 0.90, 1.13 | |
| *P* value | .001 | .082 | .533 | .872 | .004 | .392 | .834 | .886 | |
| N | 10 040 | 5212 | 4828 | 10 040 | 10 040 | 5212 | 4828 | 10 040 | |

(Continues)

**TABLE 3** (Continued)

| | Triglycerides | HDL, Women | HDL, Men | Glucose | SBP | DBP, Women | DBP, Men | MetS | DM |
|---|---|---|---|---|---|---|---|---|---|
| **$Model_{altethnic}$** | | | | | | | | | |
| *1-10 Sami* | | | | | | | | | |
| Log-β/*OR | 0.02 | −0.02 | 0.00 | 0.00 | 0.01 | −0.01 | 0.00 | 1.14* | 1.08* |
| 95% CI | −0.00, 0.04 | −0.03, −0.00 | −0.01, 0.02 | −0.00, 0.01 | 0.00, 0.01 | −0.01, 0.00 | −0.00, 0.01 | 1.04, 1.25 | 0.88, 1.32 |
| *P* value | .096 | .011 | .800 | .221 | .047 | .081 | .526 | .004 | .454 |
| *11 Sami* | | | | | | | | | |
| Log-β/*OR | 0.04 | −0.03 | −0.01 | 0.00 | −0.01 | −0.01 | −0.01 | 1.09* | 1.12* |
| 95% CI | 0.01, 0.07 | −0.05, −0.01 | −0.03, 0.01 | −0.01, 0.01 | −0.02, −0.00 | −0.02, −0.00 | −0.02, 0.00 | 0.96, 1.23 | 0.86, 1.45 |
| *P* value | .005 | .001 | .239 | .692 | .015 | .041 | .183 | .195 | .419 |
| N | 13 921 | 7124 | 6797 | 13 921 | 13 921 | 7124 | 6797 | 13 921 | 13 921 |

*Notes.* Coefficient estimates are for Sami vs non-Sami ethnicity (short vs tall in $Model_{short/tall}$). The columns represent different dependent variables (indicated by the column names). The rows represent the different models (main models, height adjusted, short vs tall, imputed, healthy, and alternative ethnic categorisation). Models for HDL cholesterol and diastolic blood pressure were stratified by sex, hence the sex-specific columns for these variables.

Abbreviations: CI, confidence interval; DBP, diastolic blood pressure; DM, diabetes mellitus; HDL, high-density lipoprotein cholesterol; MetS, metabolic syndrome; N, sample size; OR, odds ratio; SBP, systolic blood pressure.

*Odds ratio (OR).

**TABLE 4** Analyses for body mass index

| | Triglycerides | HDL, Women | HDL, Men | Glucose | SBP | DBP, Women | DBP, Men | MetS | DM |
|---|---|---|---|---|---|---|---|---|---|
| **$Model_{main}$** | | | | | | | | | |
| Log-β/*OR | −0.01 | −0.00 | 0.02 | −0.00 | −0.01 | −0.01 | −0.01 | 0.89* | 0.90* |
| 95% CI | −0.03, 0.01 | −0.02, 0.01 | 0.00, 0.03 | −0.01, 0.01 | −0.02, −0.01 | −0.02, −0.00 | −0.02, −0.00 | 0.82, 0.98 | 0.74, 1.10 |
| *P* value | .231 | .626 | .021 | .714 | <.001 | .003 | .016 | .014 | .294 |
| N | 13 921 | 7124 | 6797 | 13 921 | 13 921 | 7124 | 6797 | 13 921 | 13 921 |
| **$Model_{heightadj}$** | | | | | | | | | |
| Log-β/*OR | −0.02 | −0.00 | 0.00 | −0.01 | −0.01 | −0.01 | −0.00 | 0.88* | 0.83* |
| 95% CI | −0.04, 0.00 | −0.02, 0.01 | −0.01, 0.02 | −0.01, 0.00 | −0.02, −0.01 | −0.02, 0.00 | −0.01, 0.00 | 0.80, 0.97 | 0.67, 1.03 |
| *P* value | .086 | .685 | .698 | .188 | <.001 | .099 | .400 | .011 | .091 |
| N | 13 921 | 7124 | 6797 | 13 921 | 13 921 | 7124 | 6797 | 13 921 | 13 921 |
| **$Model_{short/tall}$** | | | | | | | | | |
| Log-β/*OR | 0.01 | −0.00 | 0.02 | 0.00 | −0.00 | −0.01 | −0.01 | 1.01* | 1.19* |
| 95% CI | −0.01, 0.03 | −0.02, 0.01 | 0.01, 0.04 | −0.00, 0.01 | −0.01, 0.00 | −0.02, −0.00 | −0.02, −0.00 | 0.94, 1.09 | 1.00, 1.41 |
| *P* value | .293 | .414 | <.001 | .140 | .054 | .007 | .001 | .794 | .052 |
| N | 13 921 | 7124 | 6797 | 13 921 | 13 921 | 7124 | 6797 | 13 921 | 13 921 |
| **$Model_{imputed}$** | | | | | | | | | |
| Log-β/*OR | −0.02 | −0.00 | 0.02 | 0.00 | −0.01 | −0.01 | −0.01 | 0.88* | 0.99* |
| 95% CI | −0.03, 0.00 | −0.02, 0.01 | 0.00, 0.03 | −0.01, 0.01 | −0.02, −0.01 | −0.02, −0.00 | −0.01, 0.00 | 0.81, 0.96 | 0.83, 1.19 |
| *P* value | .067 | .679 | .007 | .770 | <.001 | .004 | .112 | .003 | .937 |
| N | 15 749 | 8233 | 7516 | 15 749 | 15 749 | 8233 | 7516 | 15 749 | 15 749 |
| **$Model_{healthy}$** | | | | | | | | | |
| Log-β/*OR | −0.00 | 0.00 | 0.02 | −0.00 | −0.02 | −0.01 | −0.01 | 0.86* | |
| 95% CI | −0.02, 0.02 | −0.01, 0.02 | 0.00, 0.03 | −0.01, 0.00 | −0.02, −0.01 | −0.02, 0.00 | −0.02, −0.00 | 0.77, 0.96 | |
| *P* value | .924 | .971 | .026 | .453 | <.001 | .067 | .013 | .009 | |
| N | 10 040 | 5212 | 4828 | 10 040 | 10 040 | 5212 | 4828 | 10 040 | |

(Continues)

**TABLE 4** (Continued)

| | Triglycerides | HDL, Women | HDL, Men | Glucose | SBP | DBP, Women | DBP, Men | MetS | DM |
|---|---|---|---|---|---|---|---|---|---|
| **$Model_{altmarker}$** | | | | | | | | | |
| *1-10 Sami* | | | | | | | | | |
| Log-β/*OR | −0.01 | −0.01 | 0.02 | 0.00 | 0.00 | −0.01 | −0.00 | 1.00* | 0.99* |
| 95% CI | −0.03, 0.01 | −0.02, 0.01 | 0.01, 0.03 | −0.01, 0.01 | −0.00, 0.01 | −0.02, −0.00 | −0.01, 0.00 | 0.92, 1.09 | 0.81, 1.20 |
| *P* value | .167 | .431 | .004 | .872 | .930 | .011 | .318 | .984 | .885 |
| *11 Sami* | | | | | | | | | |
| Log-β/*OR | −0.01 | −0.01 | 0.02 | −0.00 | −0.02 | −0.02 | −0.02 | 0.87* | 0.97* |
| 95% CI | −0.04, 0.01 | −0.03, 0.01 | 0.00, 0.04 | −0.02, 0.01 | −0.03, −0.01 | −0.03, −0.01 | −0.03, −0.01 | 0.77, 0.99 | 0.75, 1.27 |
| *P* value | .317 | .351 | .041 | .430 | <.001 | .002 | <.001 | .030 | .833 |
| N | 13 921 | 7124 | 6797 | 13 921 | 13 921 | 7124 | 6797 | 13 921 | 13 921 |

*Notes*. Coefficient estimates are for Sami vs non-Sami ethnicity (short vs tall in $Model_{short/tall}$). The columns represent different dependent variables (indicated by the column names). The rows represent the different models (main models, height adjusted, short vs tall, imputed, healthy, and alternative ethnic categorisation). Models for HDL cholesterol and diastolic blood pressure were stratified by sex, hence the sex-specific columns for these variables.

Abbreviations: CI, confidence interval; DBP, diastolic blood pressure; DM, diabetes mellitus; HDL, high-density lipoprotein cholesterol; MetS, metabolic syndrome; N, sample size; OR, odds ratio; SBP, systolic blood pressure.

*Odds ratio (OR)

**TABLE 5** Analyses for waist-to-height ratio

| | Triglycerides | HDL, Women | HDL, Men | Glucose | SBP | DBP, Women | DBP, Men | MetS | DM |
|---|---|---|---|---|---|---|---|---|---|
| **$Model_{main}$** | | | | | | | | | |
| Log-β/*OR | −0.03 | 0.01 | 0.02 | −0.00 | −0.01 | −0.01 | −0.01 | 0.84* | 0.84* |
| 95% CI | −0.05, −0.01 | −0.01, 0.02 | 0.01, 0.03 | −0.01, 0.00 | −0.02, −0.01 | −0.02, −0.01 | −0.02, −0.00 | 0.76, 0.92 | 0.68, 1.02 |
| *P* value | .005 | .287 | .005 | .300 | <.001 | .001 | .007 | <.001 | .080 |
| N | 13 921 | 7124 | 6797 | 13 921 | 13 921 | 7124 | 6797 | 13 921 | 13 921 |
| **$Model_{heightadj}$** | | | | | | | | | |
| Log-β/*OR | −0.02 | −0.00 | −0.00 | −0.01 | −0.01 | −0.01 | −0.00 | 0.89* | 0.82* |
| 95% CI | −0.04, 0.00 | −0.02, 0.01 | −0.02, 0.01 | −0.01, 0.00 | −0.02, −0.01 | −0.02, 0.00 | −0.01, 0.01 | 0.81, 0.98 | 0.66, 1.02 |
| *P* value | .123 | .853 | .871 | .168 | <.001 | .106 | .651 | .016 | .073 |
| N | 13 921 | 7124 | 6797 | 13 921 | 13 921 | 7124 | 6797 | 13 921 | 13 921 |
| **$Model_{short/tall}$** | | | | | | | | | |
| Log-β/*OR | −0.03 | 0.01 | 0.04 | 0.00 | −0.01 | −0.01 | −0.01 | 0.88* | 1.07* |
| 95% CI | −0.04, −0.01 | 0.00, 0.02 | 0.03, 0.05 | −0.01, 0.01 | −0.01, −0.00 | −0.02, −0.01 | −0.02, −0.01 | 0.82, 0.95 | 0.90, 1.27 |
| *P* value | .003 | .028 | <.001 | .905 | <.001 | <.001 | <.001 | .001 | .460 |
| N | 13 921 | 7124 | 6797 | 13 921 | 13 921 | 7124 | 6797 | 13 921 | 13 921 |
| **$Model_{imputed}$** | | | | | | | | | |
| Log-β/*OR | −0.03 | 0.01 | 0.02 | −0.00 | −0.01 | −0.01 | −0.01 | 0.82* | 0.99* |
| 95% CI | −0.05, −0.02 | −0.00, 0.02 | 0.01, 0.03 | −0.01, 0.01 | −0.02, −0.01 | −0.02, −0.01 | −0.01, 0.00 | 0.76, 0.90 | 0.83, 1.19 |
| *P* value | <.001 | .183 | .001 | .657 | <.001 | .001 | .062 | <.001 | .937 |
| N | 15 749 | 8233 | 7516 | 15 749 | 15 749 | 8233 | 7516 | 15 749 | 15 749 |
| **$Model_{healthy}$** | | | | | | | | | |
| Log-β/*OR | −0.02 | 0.01 | 0.02 | −0.01 | −0.02 | −0.01 | −0.01 | 0.80* | |
| 95% CI | −0.04, 0.00 | −0.00, 0.03 | 0.01, 0.04 | −0.01, 0.00 | −0.02, −0.01 | −0.02, −0.00 | −0.02, −0.00 | 0.72, 0.90 | |
| *P* value | .108 | .181 | .005 | .180 | <.001 | .017 | .003 | <.001 | |
| N | 10 040 | 5212 | 4828 | 10 040 | 10 040 | 5212 | 4828 | 10 040 | |

(Continues)

**TABLE 5** (Continued)

| | Triglycerides | HDL, Women | HDL, Men | Glucose | SBP | DBP, Women | DBP, Men | MetS | DM |
|---|---|---|---|---|---|---|---|---|---|
| **Model$_{altmarker}$** | | | | | | | | | |
| *1-10 Sami* | | | | | | | | | |
| Log-β/*OR | −0.03 | 0.00 | 0.02 | −0.00 | −0.00 | −0.01 | −0.00 | 0.96* | 0.95* |
| 95% CI | −0.04, −0.01 | −0.01, 0.02 | 0.01, 0.04 | −0.01, 0.01 | −0.01, 0.00 | −0.02, −0.00 | −0.01, 0.00 | 0.88, 1.05 | 0.77, 1.16 |
| *P* value | .009 | .767 | .001 | .811 | .697 | .004 | .200 | .357 | .592 |
| *11 Sami* | | | | | | | | | |
| Log-β/*OR | −0.03 | 0.00 | 0.02 | −0.01 | −0.02 | −0.02 | −0.02 | 0.81* | 0.89* |
| 95% CI | −0.06, −0.00 | −0.02, 0.02 | 0.01, 0.04 | −0.02, 0.00 | −0.03, −0.01 | −0.03, −0.01 | −0.03, −0.01 | 0.71, 0.92 | 0.69, 1.17 |
| *P* value | .020 | .757 | .013 | .175 | <.001 | .001 | <.001 | .001 | .413 |
| N | 13 921 | 7124 | 6797 | 13 921 | 13 921 | 7124 | 6797 | 13 921 | 13 921 |

*Notes*. Coefficient estimates are for Sami vs non-Sami ethnicity (short vs tall in Model$_{short/tall}$). The columns represent different dependent variables (indicated by the column names). The rows represent the different models (main models, height adjusted, short vs tall, imputed, healthy, and alternative ethnic categorisation). Models for HDL cholesterol and diastolic blood pressure were stratified by sex, hence the sex-specific columns for these variables.

Abbreviations: CI, confidence interval; DBP, diastolic blood pressure; DM, diabetes mellitus; HDL, high-density lipoprotein cholesterol; MetS, metabolic syndrome; N, sample size; OR, odds ratio; SBP, systolic blood pressure.

*Odds ratio (OR)

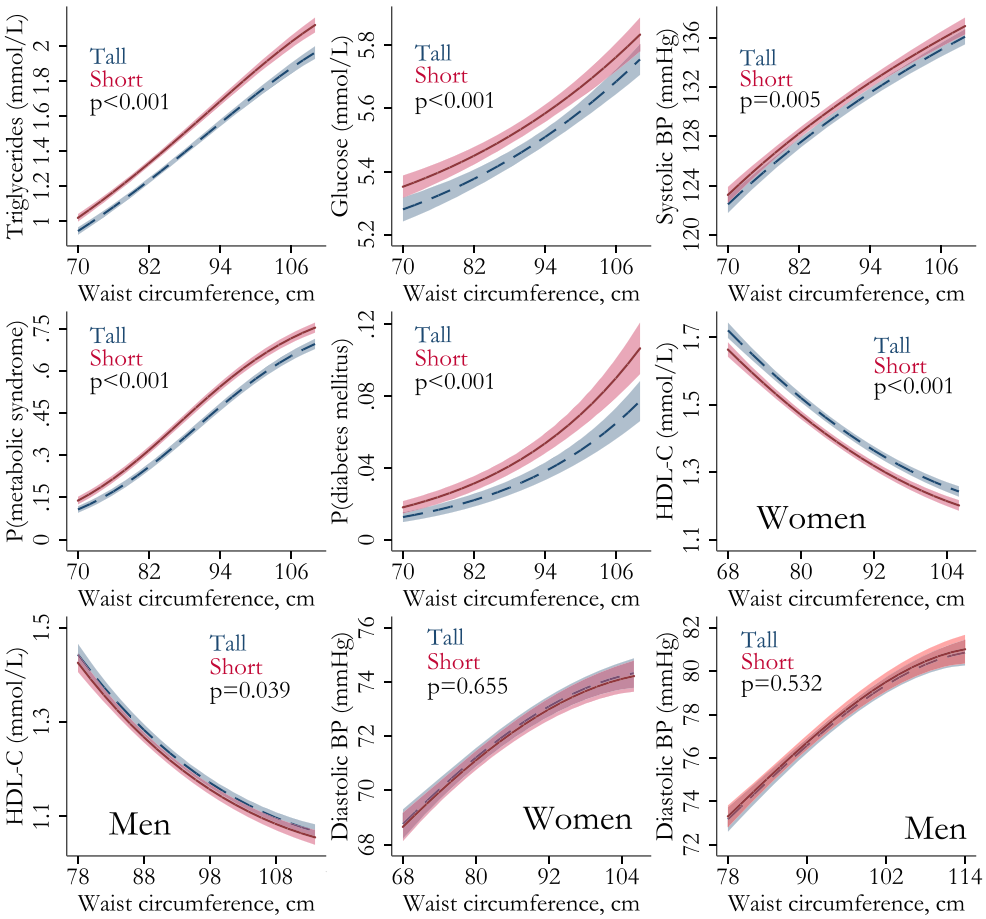


**FIGURE 3** Estimated relationships between metabolic markers and waist circumference in short vs tall people. All models were fitted with fractional polynomial regression and adjusted for age, age squared, leisure-time PA, smoking, alcohol consumption, sex (not in models with HDL-C and diastolic BP as dependent variables; these were sex stratified) and relevant use of medication. Curves are drawn separate for short (red, solid line) and tall (blue, dashed line) people. *P* values are for short vs tall people. Average marginal effects for each group were estimated, holding all other variables in the model constant, for the 5th to the 95th percentile of the obesity measure. HDL-C, high-density lipoprotein cholesterol. BP, blood pressure

increased risk of DM, cardiovascular disease, and mortality.[29,30] On the contrary, genetically determined height has been linked to cardiovascular disease perhaps trough shared biological pathways.[31] However, in contrast to previous studies on height, Sami ethnicity, and disease,[9,26] this study has examined the clinical implications when using various obesity measures, not the implication of height in itself. Hence, this topic will not be further elaborated on.

The findings regarding height, abdominal obesity, and metabolic markers support studies from Japan[14] and Germany[32]: Short people have worse metabolic profiles than tall people with the same WC but similar when having the same WHtR.[32] In a meta-analysis on a sample including a wide range of heights, WHtR was superior to WC with respect to cardiometabolic risk prediction.[33] In a recent review of anthropometric cut-offs and its impact on metabolic alterations, it was suggested that height differences could explain the different levels of metabolic markers at similar levels of obesity between various ethnic groups.[34] WHtR was suggested as a universal measure unaffected by ethnicity.[34] In our study, some metabolic markers were slightly more favourable at the same levels of BMI or WHtR in Sami than in non-Sami, despite height being integrated into both these measures. However, the differences were marginal and likely irrelevant clinically. Further, sensitivity analyses showed metabolic differences between short and tall people at the same level of WHtR, suggesting that WHtR does not capture the same level of metabolic markers along the entire range of height in this particular population.

Ethnicity is a complex concept and a challenging variable to define.[35] Depending on context, it can comprise language, culture, religion, skin colour, geography, diet, and genetics. In this study, an effort was made to tease the Sami ethnicity variable apart from other variables that may confound or mediate the relationships between metabolic markers and obesity, aiming to capture the "direct effect of ethnicity," whatever that entails.[36] The lack of such an effect is not surprising as Sami ethnicity is viewed first and foremost as a socio-cultural marker. Using various criteria for Sami ethnicity impacts both size and geographical distribution.[37] The residual "direct effect" of Sami ethnicity is—in this particular study—possibly a side-effect of dichotomising the sample into groups that differ substantially in height. Importantly, Sami ethnicity, defined in any way, is not deterministic with respect to short height. A person's stature seems to be a much more important predictor than a person's ethnic belonging, especially concerning WC.

The results do not support the need for ethnic-specific cut-offs of obesity to be used in rural Northern Norway. However, it may be suggested that researchers should evaluate whether some form of height adjustment is reasonable when studying obesity and its related disorders in two populations that differ in stature.

The large sample size is an obvious strength of the study. In addition, all measurements were performed by trained personnel and followed a protocol. Several markers of ethnicity were included such

that sensitivity (bias) analyses regarding the ethnic categorisation could be performed. Several factors comprising lifestyle and health status, such as leisure-time PA, smoking, and use of medication, were also possible to adjust for.

Limitations of the study include that it is cross-sectional, meaning that the temporality of the associations cannot be commented on. The response rate was moderately adequate: 57% overall attendance in the survey, but 50% in the final sample. Nonattendance with respect to ethnicity could not be evaluated, but it was more common in younger, unmarried men. The survey was conducted ~15 years ago, and extrapolation of the results beyond this sample is not advised. The results are exploratory and should be confirmed in other samples. Further limitations include nonfasting blood samples. Triglyceride levels have been found to vary around 20% between different fasting states,[38] but more importantly, random glucose is not a very valid measure of glucose metabolism nor diagnosing DM. Fasting blood samples on glucose, insulin, HbA1c, and an oral glucose tolerance test are necessary in order to evaluate the relationships between obesity and impaired glucose metabolism. Moreover, measurement error of self-reported variables cannot be excluded. However, if misclassification of these variables is of the same direction and magnitude in Sami and non-Sami, it is unlikely that it affects the confounding influence on the β-coefficient for ethnicity.

## 5 | CONCLUSION

The relationships between metabolic markers and obesity measures did not differ by ethnicity in Northern and Central Norway. The few marginal ethnic differences in levels of metabolic markers at the same levels of the obesity measure were eliminated by height adjustments. An exception was for systolic BP, which was lower in Sami than in non-Sami at any given level of obesity.

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### CONFLICT OF INTEREST STATEMENT

The authors declared no conflict of interest.

### AUTHOR CONTRIBUTIONS

VLM conceived the idea behind the study, performed all statistical analyses, and wrote the manuscript. TB aided with technical assistance in the statistical analyses. TB, ARB, KK, and MM contributed with planning of the design and analyses, and the interpretation of the results. All authors critically revised the manuscript and accepted the final draft for publication.

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## SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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