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Complete List of Authors:	Siri, Susanna; UiT The Arctic University of Norway, Centre for Sami Health Research, Department of Community Medicine Eliassen, Bent Martin; Nord University, Faculty of Nursing and Health Sciences Jacobsen, Bjarne; UiT The Arctic University of Norway, Department of Community Medicine; UiT The Arctic University of Norway, Centre for Sami Health Research, Department of Community Medicine Melhus, Marita; UiT The Arctic University of Norway, Centre for Sami Health Research, Department of Community Medicine Broderstad, Ann Ragnhild; UiT The Arctic University of Norway, Centre for Sami Health Research, Department of Community Medicine Michalsen, Vilde; UiT The Arctic University of Norway, Department of Community Health Braaten, Tonje; UiT The Arctic University of Norway, Department of Community Medicine
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CHANGES IN CONVENTIONAL CARDIOVASCULAR RISK FACTORS AND THE ESTIMATED 10-YEAR RISK OF ACUTE MYOCARDIAL INFARCTION OR CEREBRAL STROKE OVER 8–11 YEARS IN SAMI AND NON-SAMI POPULATIONS -THE SAMINOR STUDY

Susanna Ragnhild A. Siri[1], Bent-Martin Eliassen[2], Bjarne K. Jacobsen[1,3], Marita Melhus[1], Ann Ragnhild Broderstad[1], Vilde Lehne Michalsen[1], Tonje Braaten[3]

[1]Centre for Sami Health Research, Department of Community Medicine, Faculty of Health Sciences, UiT The Arctic University of Norway, Tromsø, Norway. [2] Faculty of Nursing and Health Sciences, Nord University, Bodø, Norway. [3]Department of Community Medicine, Faculty of Health Sciences, UiT The Arctic University of Norway, Tromsø, Norway.

Corresponding author: Susanna Ragnhild A. Siri

Postal address: Centre for Sami Health Research, Department of Community Medicine, UiT The Arctic University of Norway. Postboks 6050, Langnes. 9037 Tromsø

E-mail: susanna.r.siri@uit.no

Telephone number: 0047-77646977

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Abstract

Objective: To describe changes in cardiovascular risk factors and in the estimated 10-year risk of acute myocardial infarction (AMI) or cerebral stroke (CS) between SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014), and explore if these changes differed between Sami and non-Sami.

Design and methods: This study included inhabitants of rural Northern Norway aged 40–79 years who participated in SAMINOR 1 (n=6417) and/or SAMINOR 2 (n=5956). Generalised estimating equation regression with interaction term was used to estimate and compare changes in cardiovascular risk factors and 10-year risk of AMI or CS between the two surveys and by ethnicity.

Results: Mean cholesterol declined by 0.50, 0.43, and 0.60 mmol/L in all women, Sami men, and non-Sami men, respectively (all $p<0.001$). The change in mean high-density lipoprotein (HDL) cholesterol differed by ethnicity in both sexes; the change in triglycerides differed only in men. In women and men, systolic blood pressure declined by 3.6 and 3.1, and diastolic blood pressure by 1.0 and 0.7 mmHg, respectively (all $p<0.01$). Use of anti-hypertensive drugs increased in men, whereas the prevalence of hypertension decreased in women. Mean waist circumference increased by 6.7 cm and 5.9 cm in women and men, respectively (both $p<0.001$). The odds of being a smoker declined by 35% in women and 46% in men (both $p<0.001$). Estimated 10-year risk of AMI or CS decreased in the overall study sample ($p<0.05$) with an indication of a different change in women.

Conclusions: Independent of ethnicity, there was a decline in mean cholesterol, blood pressure, proportion of smokers, hypertension (women only), and 10-year risk of AMI or CS, but waist circumference increased. Regarding ethnic differences in changes of cardiovascular risk factors, minor ethnic differences were observed in lipids (only HDL cholesterol in women) and in the 10-year risk of AMI or CS (women only).

ARTICLE SUMMARY

Strength and limitations:

- We used generalised estimating equation regression to account for overlapping samples.
- We used self-reported measures to categorise participants into ethnic groups, including questions on self-perceived ethnicity, ethnic background, and language use.
- Due to lack of ethnic identifiers in national registries, we do not know if participation differs by ethnicity.
- We have a conventional participation rate in both surveys.
- We lack information about the use of lipid-lowering drugs.

INTRODUCTION

Since the 1970s, a favourable decline in systolic blood pressure,[1-4] total cholesterol[5-7] and smoking[8] has been reported for the adult population across different regions of Western Europe. This decline is probably due to changes in diet and behaviour,[7, 9, 10] in addition to the use of medication.[1, 5] In Norway, this decline has coincided with a decrease in cardiovascular mortality and an increased prevalence of obesity and a sedentary lifestyle.[11]

The Sami is an Indigenous people living in Sápmi, i.e. the northern parts of Norway, Sweden, Finland, and the Kola Peninsula in the Russian Federation. There are no official population records on the Sami population, but data from the 1970 national census roughly estimated that there were 40,000 Sami in Norway,[12] whereas 55,000 is the population number that the Sami Parliament uses when considering subsidy schemes for business development.[13] In 2017, approximately 17,000 Sami adults were enrolled in the electoral register to the Sami Parliament in Norway, which gives them the right to vote and be elected.[14] The Sami people have unique cultures and languages, but these have partly vanished or at least declined in practice, due to structural assimilation that occurred from 1850–1960.[15] The Norwegian part of Sápmi is also inhabited by Norwegians and Kvens, the latter of whom are descendants of Finnish-speaking people that came from Sweden and Finland to Northern Norway in the 1700s and 1800s.[16]

Cross-sectional studies from Norway have concluded that there are no or only minor differences in cardiovascular risk factors and morbidity between the Sami and non-Sami in rural regions.[17-21] However, knowledge is lacking on changes in conventional cardiovascular risk factors. Thus, this study aimed to describe changes in cardiovascular risk factors and in the estimated 10-year risk of acute myocardial infarction (AMI) or cerebral stroke (CS)

between SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014), and explore if these changes differed between Sami and non-Sami.

METHODS

We used data from two cross-sectional surveys of the population-based Study on Health and Living Conditions in Regions with Sami and Norwegian Populations (The SAMINOR Study): the SAMINOR 1 Survey carried out in 2003–2004 (SAMINOR 1) and the SAMINOR 2 Clinical Survey carried out in 2012–2014 (SAMINOR 2). SAMINOR 1 was a collaboration between the Centre for Sami Health Research at UiT The Arctic University of Norway and the Norwegian National Institute of Public Health,[22] whereas SAMINOR 2 was performed by the former only. Our analyses were restricted to participants aged 40–79 years from 10 municipalities included in both SAMINOR 1 and SAMINOR 2 (figure 1), who attended clinical examinations, gave blood samples, and answered the self-administered questionnaires. Within the 10 selected municipalities, a total of 11,518 inhabitants were invited to SAMINOR 1 and 12,455 to SAMINOR 2.

Study sample

There were 6550 (56.9%) and 6004 (48.2%) from the 10 selected municipalities that attended the clinical examinations in SAMINOR 1 and 2, respectively. If information on ethnicity was lacking in one of the surveys, ethnicity information given in the other survey was used, as Sami ethnicity is found to be stable.[23] This strategy was valuable for the SAMINOR 1 sample, as ethnicity information was lacking for some participants due to the study design.[22] In SAMINOR 1, we categorised 69 out of 201 by using ethnicity information from SAMINOR 2: 7 non-Sami and 62 Sami. In SAMINOR 2, 96 had missing on ethnicity and we categorised 58: 37 non-Sami and 21 Sami. Furthermore, we excluded those that did not hand

in the main questionnaires (SAMINOR 1: n=1, SAMINOR 2: n=10). This left us with a final sample of 6417 and 5956 from SAMINOR 1 and 2, respectively, wherein 3249 participated in both surveys.

The Norwegian Data Inspectorate and the Regional Committee for Medical and Health Research Ethics for region North (REC North) have approved The SAMINOR Study. The REC North (2015/2204–11) and the SAMINOR Project Board have also approved this study. All participants included in this study gave written informed consent and consented to linkage between the surveys.

Information from questionnaires

Participants were categorised into ethnic groups based on information from the following 11 questions, which were identical in the two surveys: "What language(s) do/did you, your parents, and your grandparents use at home?"; "What is your, your father's, and your mother's ethnic background?"; "What [ethnicity] do you consider yourself to be?". The response options were "Norwegian", "Sami", "Kven" and "Other" and multiple answers were allowed. Participants were defined as Sami if they 1) considered themselves to be Sami, or reported a Sami ethnic background for themselves, and 2) spoke a Sami language themselves or had at least one parent or grandparent that used it at home. All others were categorised as non-Sami. Sensitivity analyses were performed, in which different ethnic categorisations were used.

Smoking status was determined by the following questions, in SAMINOR 1: "Are you currently, or were you previously a daily smoker?" (Yes, currently/Yes, previously/Never); in SAMINOR 2: "Have you ever smoked daily?" (Yes/No), and "Are you currently a daily smoker?" (Yes/No). Previous and never smokers were categorised as non-smokers.

Use of anti-hypertensive drugs was determined by the question: "Do you take medication for high blood pressure?" (Currently/Previously, but not now/Never used). Previous use, never-use and missing values were merged into non-use.

Clinical examination

Trained staff conducted the clinical examination. Waist circumference was measured at the umbilicus when the participant was standing. Blood pressure was measured with digital oscillometric devices (SAMINOR 1: DINAMAP-R, Criticon, Tampa, Florida, USA; SAMINOR 2: CARESCAPE^{TM}V100 monitor, GE Healthcare, Milwaukee, Wisconsin, USA), with the participant in a seated position. Following a 2-minute rest, three recordings were made at 1-minute intervals, and the average of the last two measurements was used in the analysis. Participants were considered to have hypertension if their systolic blood pressure was ≥140 mmHg, or diastolic blood pressure was ≥90 mmHg, or if they reported using anti-hypertensive drugs.

In both surveys, non-fasting blood samples were collected. The blood samples were left to coagulate for a minimum of 30 minutes, after which they were centrifuged and serum was separated within 2 hours. In SAMINOR 1, serum was sent by overnight post and analysed consecutively for lipids (total cholesterol, high-density lipoprotein (HDL) cholesterol and triglycerides) with an enzymatic method (Hitachi 917 auto analyser, Roche Diagnostics, Switzerland) at Ullevål University Hospital, Oslo, Norway. In SAMINOR 2, serum samples were kept at -20° C before they were sent to the biobank at UiT The Arctic University of Norway, for further storage at -70° C. The samples were analysed in batches during autumn 2014 at the University Hospital of North Norway, Tromsø, Norway. Lipids were measured

with an enzymatic colorimetric method (Cobas 8000B, Roche Diagnostics GmbH, Mannheim, Germany).

The 10-year absolute risk of fatal or non-fatal AMI or CS was estimated by the NORRISK 2 model[24] and determined separately in women and men based on age, total cholesterol, HDL cholesterol, smoking status, systolic blood pressure, and use of anti-hypertensive drugs.

Statistical analyses

Statistical analyses were done using STATA version 15.0 (StataCorp, College Station, Texas, USA). In order to account for the partly overlapping samples, changes in population average means and proportions of risk factors between SAMINOR 1 and SAMINOR 2 were estimated by sex- and ethnicity-specific linear or logistic generalised estimating equation regression models. Assumptions of normality and homoscedasticity were assessed by a visual inspection of residual plots. Changes in triglycerides and in the estimated 10-year risk of AMI or CS were log-transformed due to skewed distributions. All regression models were adjusted for age, and linear models were additionally adjusted for age squared. We assessed if changes in outcomes differed by ethnicity by including an interaction term between survey and ethnicity in sex-specific models. If the p-value for interaction was >0.05, the interaction term was excluded from the model and an overall sex-specific mean/prevalence was reported. In the opposite case, ethnicity-specific changes were reported. Marginal means/prevalences were estimated at age 57.5 years in women and at 58.2 years in men, i.e. the sex-specific mean ages in the overall sample. We considered a two-sided $p<0.05$ to be significant.

Sensitivity analyses were done with same sex-stratified generalised estimating equation models by

1. Dividing the study sample into three groups: 1) those who reported "Sami" on all 11 questions, 2) who reported Sami in one to 10 questions, and 3) those who did not report Sami on any of the questions (non-Sami).
2. Using the original ethnic categorisation, we adjusting for and stratified into three geographical regions: 1) Kautokeino and Karasjok, 2) Nesseby, Tana, and Porsanger, 3) Kåfjord, Lyngen, Storfjord, Skånland, and Evenes.

RESULTS

Of the total sample, 53.3% were women. In women and men, 37.8% and 39.5% were Sami, respectively.

Both non-Sami and Sami women had a decline in total cholesterol between SAMINOR 1 and SAMINOR 2 ($p<0.001$, table 1). The overall change in total cholesterol in women was -0.50 mmol/l. Sami women had lower HDL cholesterol and higher triglycerides than non-Sami at both surveys (visual inspection, table 1). The change in triglycerides did not differ by ethnicity (p for interaction=0.07), but the change in HDL did (p for interaction<0.001), with non-Sami showing a minor increase, and Sami showing no change.

Table 1. Age-adjusted predicted changes in means and prevalences of cardiovascular risk factors between the SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014) surveys in non-Sami and Sami women (n=6624).

	Non-Sami (n=4122)		Sami (n=2502)		Interaction ‡	Overall (n=6624)	
Linear regression	**β (95% CI)**	**p-value**	**β (95% CI)**	**p-value**	**p-value**	**β (95% CI)**	**p-value**
Total cholesterol, mmol/L	-0.51 (-0.57, -0.45)	<0.001	-0.47 (-0.55, -0.39)	<0.001	0.86	-0.50 (-0.54, -0.45)	<0.001
SAMINOR 1, mean †	6.24 (6.19, 6.30)		6.24 (6.16, 6.32)			6.25 (6.20, 6.29)	
SAMINOR 2, mean †	5.73 (5.67, 5.79)		5.77 (5.70, 5.85)			5.75 (5.70, 5.79)	
HDL cholesterol, mmol/L	0.05 (0.03, 0.07)	<0.001	-0.02 (-0.04, 0.01)	0.14	<0.001	*	
SAMINOR 1, mean †	1.51 (1.49, 1.53)		1.47 (1.45, 1.49)				
SAMINOR 2, mean †	1.56 (1.54, 1.58)		1.45 (1.43, 1.48)				
Triglycerides, mmol/L§	-0.02 (-0.05, 0.004)	0.10	0.03 (0.004, 0.07)	0.03	0.07	0.002 (-0.02, 0.02)	0.87
SAMINOR 1, mean †#	1.43 (1.40, 1.47)		1.48 (1.43, 1.53)			1.45 (1.43, 1.48)	
SAMINOR 2, mean †#	1.40 (1.37, 1.44)		1.53 (1.49, 1.58)			1.46 (1.43, 1.48)	
Systolic blood pressure, mm Hg	-3.9 (-4.86, -2.98)	<0.001	-3.0 (-4.23, -1.81)	<0.001	0.10	-3.6 (-4.36, -2.88)	<0.001
SAMINOR 1, mean †	134.7 (133.8, 135.6)		132.4 (131.2, 133.7)			133.9 (133.2, 134.6)	
SAMINOR 2, mean †	130.8 (129.9, 131.7)		129.4 (128.3, 130.6)			130.3 (129.6, 131.0)	
Diastolic blood pressure, mm Hg	-0.9 (-1.38, -0.33)	0.002	-1.1 (-1.77, -0.46)	0.001	0.39	-1.0 (-1.39, -0.57)	<0.001
SAMINOR 1, mean †	73.9 (73.4, 74.4)		73.4 (72.7, 74.1)			73.7 (73.3, 74.1)	
SAMINOR 2, mean †	73.0 (72.5, 73.6)		72.3 (71.6, 72.9)			72.7 (72.3, 73.2)	
Waist circumference, cm	7.0 (6.41, 7.49)	<0.001	6.1 (5.44, 6.76)	<0.001	0.26	6.7 (6.24, 7.07)	<0.001
SAMINOR 1, mean †	86.4 (85.83, 87.00)		88.0 (87.29, 88.78)			87.0 (86.55, 87.45)	
SAMINOR 2, mean †	93.3 (92.75, 93.94)		94.1 (93.43, 94.85)			93.7 (93.20, 94.11)	
10-year risk of AMI and CS, %§	-0.19 (-0.22, -0.17)	<0.001	-0.13 (-0.16, -0.09)	<0.001	0.011	*	
SAMINOR 1, mean †#	4.16 (4.05, 4.26)		3.91 (3.77, 4.05)				
SAMINOR 2, mean †#	3.43 (3.33, 3.52)		3.44 (3.32, 3.56)				
Logistic regression	**Odds ratio (95% CI)**	**p-value**	**Odds ratio (95% CI)**	**p-value**	**p-value**	**Odds ratio (95% CI)**	**p-value**
Anti-hypertensive treatment	0.96 (0.85, 1.09)	0.51	0.98 (0.83, 1.16)	0.82	0.77	0.96 (0.87, 1.07)	0.47
SAMINOR 1, prevalence % †	24.0 (22.10, 25.80)		22.9 (20.34, 25.36)			23.6 (22.11, 25.10)	
SAMINOR 2, prevalence % †	23.2 (21.17, 25.24)		22.5 (20.04, 24.98)			23.0 (21.38, 24.53)	
Hypertension	0.77 (0.68, 0.86)	<0.001	0.79 (0.68, 0.93)	0.003	0.56	0.77 (0.70, 0.85)	<0.001
SAMINOR 1, prevalence % †	47.4 (45.12, 49.72)		44.5 (41.37, 47.68)			46.3 (44.47, 48.20)	
SAMINOR 2, prevalence % †	40.8 (38.39, 43.25)		38.9 (35.90, 41.79)			40.1 (38.18, 41.94)	
Current smokers	0.59 (0.53, 0.66)	<0.001	0.74 (0.64, 0.85)	<0.001	0.10	0.65 (0.59, 0.71)	<0.001
SAMINOR 1, prevalence % †	31.0 (29.07, 32.90)		27.9 (25.28, 30.45)			29.8 (28.26, 31.34)	
SAMINOR 2, prevalence % †	21.0 (19.26, 22.76)		22.3 (20.07, 24.46)			21.6 (20.21, 22.94)	

β –coefficients are estimated by linear generalised estimating equation regression models and adjusted for age and age^2. Odds ratios are estimated by logistic generalised estimating equation regression models and adjusted for age. †Predicted means/prevalences in % at age 57.5 years, which is the mean age for women in the overall sample. §β –coefficients are log transformed. #Predicted geometric means at age 57.5 year. ‡ Test of interaction between survey and ethnicity in overall model. If p-value for interaction >0.05, interaction term was excluded from the overall model. *p-value for interaction <0.05, only ethnicity-specific estimations are reported. Number of missing values: total and HDL cholesterol and triglycerides were missing in 18 subjects; systolic and diastolic blood pressure were missing in four subjects; hypertension in three. NORRISK 2 score was missing for 193 subjects. Excluding missing values did not change the results. Abbreviations: CI, confidence interval; AMS, acute myocardial infarction; CS, cerebral stroke.

At both surveys, Sami women had somewhat lower blood pressure than non-Sami (visual inspection, table 1). The overall decline in systolic and diastolic blood pressure was 3.6 and 1.0 mmHg (both p<0.001), respectively; these changes did not differ by ethnicity (p for interaction for systolic and diastolic blood pressure was 0.10 and 0.39, respectively). Roughly 23% of women reported use of antihypertensive drugs; this did not change over time (p for interaction=0.77, table 1). The proportions of participants considered hypertensive declined in a similar magnitude in Sami and non-Sami (p for interaction=0.56, table 1), and by 6.2 percentage points in women (p<0.001).

Non-Sami and Sami women had an increase in mean waist circumference of 7.0 cm and 6.1 cm, respectively (both p<0.001, table 1), and this change did not differ by ethnicity (p for interaction=0.26). The proportion of smokers in non-Sami and Sami women declined by 10 and 5.6 percentage points, respectively (both p<0.001, table 1); this change did not differ by ethnicity (p for interaction=0.10). Overall, the odds of current smoking declined by 35%.

The estimated 10-year risk of AMI or CS declined between SAMINOR 1 and SAMINOR 2 in both Sami and non-Sami women (both p<0.001, table 1), however, more so in non-Sami (p for interaction=0.01).

Between SAMINOR 1 and SAMINOR 2, total cholesterol declined more in non-Sami than in Sami men (0.60 vs. 0.43 mmol/l; both p<0.001, table 2), and this change varied by ethnicity (p for interaction=0.03). Between the surveys, Sami men had a slight decline in HDL cholesterol (p<0.001) and a slight increase in triglycerides (p<0.001); whereas non-Sami men had no changes, hence, change in HDL and triglyceride differed for Sami and non-Sami, (p for interaction for HDL cholesterol and triglycerides: 0.005 and <0.001, respectively, table 2).

Table 2. Age-adjusted predicted changes in means and in prevalences of cardiovascular risk factors between the SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014) surveys in non-Sami and Sami men (n=5749).

	Non-Sami (n=3478)		Sami (n=2271)		Interaction ‡	Overall (n=5749)	
Linear regression	**β (95% CI)**	**p-value**	**β (95% CI)**		**p-value**	**β (95% CI)**	**p-value**
Total cholesterol, mmol/L	-0.60 (-0.66, -0.53)	<0.001	-0.43 (-0.51, -0.35)	<0.001	0.03	*	
SAMINOR 1, mean †	6.00 (5.95, 6.07)		6.00 (5.92, 6.08)				
SAMINOR 2, mean †	5.41 (5.35, 5.48)		5.58 (5.50, 5.65)				
HDL cholesterol, mmol/L	-0.01 (-0.03, 0.01)	0.18	-0.06 (-0.08, -0.04)	<0.001	0.005	*	
SAMINOR 1, mean †	1.30 (1.28, 1.31)		1.28 (1.26, 1.31)				
SAMINOR 2, mean †	1.28 (1.26, 1.30)		1.22 (1.20, 1.25)				
Triglycerides, mmol/L§	0.001 (-0.03, 0.03)	0.96	0.09 (0.05, 0.13)	<0.001	0.001	*	
SAMINOR 1, mean †#	1.61 (1.56, 1.65)		1.58 (1.53, 1.64)				
SAMINOR 2, mean †#	1.61 (1.56, 1.66)		1.73 (1.67, 1.79)				
Systolic blood pressure, mm Hg	-3.2 (-4.19, -2.18)	<0.001	-2.8 (-4.11, -1.44)	<0.001	0.38	-3.1 (-3.87, -2.27)	<0.001
SAMINOR 1 mean †	137.0 (136.1, 137.9)		136.8 (135.5, 138.1)			136.9 (136.2, 137.7)	
SAMINOR 2, mean †	133.8 (132.8, 134.8)		134.0 (132.7, 135.3)			133.9 (133.1, 134.6)	
Diastolic blood pressure, mm Hg	-0.5 (-1.08, 0.11)	0.11	-1.1 (-1.82, -0.33)	0.004	0.08	-0.7 (-1.20, -0.28)	0.002
SAMINOR 1, mean †	79.7 (79.2, 80.2)		79.9 (79.2, 80.6)			79.8 (79.4, 80.2)	
SAMINOR 2, mean †	79.2 (78.6, 79.8)		78.9 (78.2, 79.5)			79.1 (78.6, 79.5)	
Waist circumference, cm	6.0 (5.45, 6.56)	<0.001	5.9 (5.22, 6.48)	<0.001	0.37	5.9 (5.50, 6.31)	<0.001
SAMINOR 1, mean †	94.4 (93.82, 94.90)		93.1 (92.45, 93.79)			93.9 (94.48, 94.32)	
SAMINOR 2, mean †	100.4 (99.78, 100.95)		99.0 (98.29, 99.64)			99.8 (99.36, 100.24)	
10-year risk of AMI and CS, %§	-0.19 (-0.22, -0.17)	<0.001	-0.16 (-0.19, -0.12)	<0.001	0.23	-0.18 (-0.20, -0.16)	<0.001
SAMINOR 1, mean †#	8.73 (8.52, 8.95)		8.75 (8.45, 9.04)			8.74 (8.57, 8.92)	
SAMINOR 2, mean †#	7.20 (7.00, 7.40)		7.48 (7.23, 7.73)			7.32 (7.17, 7.48)	
Logistic regression	**Odds ratio (95% CI)**	**p-value**	**Odds ratio (95% CI)**	**p-value**	**p-value**	**Odds ratio (95% CI)**	**p-value**
Anti-hypertensive treatment	1.19 (1.03, 1.36)	0.02	1.15 (0.97, 1.37)	0.11	0.37	1.17 (1.06, 1.31)	0.003
SAMINOR 1, prevalence % †	21.4 (19.48, 23.28)		20.7 (18.34, 23.11)			21.1 (19.60, 22.56)	
SAMINOR 2, prevalence % †	24.4 (22.15, 26.67)		23.1 (20.53, 25.74)			23.9 (22.18, 25.60)	
Hypertension	0.94 (0.83, 1.06)	0.32	0.89 (0.77, 1.04)	0.13	0.39	0.92 (0.83, 1.01)	0.08
SAMINOR 1, prevalence % †	51.0 (48.62, 53.42)		50.4 (47.43, 53.43)			50.7 (48.84, 52.63)	
SAMINOR 2, prevalence % †	49.4 (46.83, 52.01)		47.5 (44.39, 50.51)			48.6 (46.61, 50.57)	
Current smokers	0.51 (0.44, 0.58)	<0.001	0.59 (0.51, 0.69)	<0.001	0.27	0.54 (0.49, 0.60)	<0.001
SAMINOR 1, prevalence % †	30.4 (28.35, 32.50)		30.8 (28.16, 33.45)			30.7 (29.06, 32.35)	
SAMINOR 2, prevalence % †	18.1 (16.30, 19.97)		20.8 (18.55, 23.12)			19.4 (17.94, 20.81)	

β –coefficients are estimated by linear generalised estimating equation regression models and adjusted for age and age^2. Odds ratios are estimated by logistic generalised estimating equation regression models and adjusted for age. †Predicted means/prevalences in % at age 58.2 years, which is the mean age for men in the overall sample. §β –coefficients are log transformed. #Predicted geometric means at age 58.2 year. ‡ Test of interaction between survey and ethnicity in overall model. If p-value for interaction >0.05, interaction term is excluded from the overall model. *p-value for interaction <0.05, only ethnicity-specific estimations are reported. Number of missing values: total and HDL cholesterol were missing in 12 subjects; triglycerides were missing in 13 subjects, systolic and diastolic blood pressure and hypertension was missing in one subject. NORRISK 2 score was missing for 173 subjects. Excluding missing values did not change the results. Abbreviations: CI, confidence interval; AMS, acute myocardial infarction; CS, cerebral stroke.

In men, the decline in systolic and diastolic blood pressure did not differ by ethnicity (p for interaction: 0.38 and 0.08, respectively, table 2). The overall decline in systolic and diastolic blood pressure in men were 3.1 and 0.7 mmHg (both $p<0.05$), respectively. Overall, we found an increase in the prevalence of anti-hypertensive drug use, from 21.1% to 23.9% in men ($p=0.003$, table 2), which did not differ by ethnicity (p for interaction=0.37). The proportion of men categorised as hypertensive remained similar between SAMINOR 1 and SAMINOR 2, with roughly half of men being considered hypertensive (table 2).

Waist circumference increased similarly (p for interaction=0.37, table 2) in Sami and non-Sami men, with an overall increase of 5.9 cm ($p<0.001$) in all men. The proportion of smokers declined similarly (p for interaction=0.27, table 2) in non-Sami and Sami men, by 12.3 and 10 percentage points, respectively (both $p<0.001$). The overall decline in the odds of being a smoker was 46%.

The estimated 10-year risk of AMI or CS declined in non-Sami and Sami men (both $p<0.001$, table 2), but not differently in the two ethnic groups (p for interaction=0.23).

Sensitivity analyses

Overall, the sensitivity analyses were consistent with the main findings when adjusting for region and when using a different ethnic categorisation.

DISCUSSION

From SAMINOR 1 (2003–2004) to SAMINOR 2 (2012–2014), participants from the selected 10 municipalities in Northern Norway had a favourable decline in total cholesterol, blood pressure, proportion of smokers, and the estimated 10-year risk of AMI or CS, whereas waist

circumference increased. The changes in total cholesterol (men only), HDL cholesterol (both sexes), triglycerides (men only), and the estimated 10-year risk of AMS or CS (women only), were statistically significantly different between Sami and non-Sami. The odds of anti-hypertensive drug use increased only in men, whereas the proportion of participants categorised as hypertensive decreased only in women. To our knowledge, there are no other studies in Sápmi that explore whether changes in cardiovascular risk factors differ between Sami and non-Sami over time.

In both SAMINOR 1 and SAMINOR 2, participation was lowest among the youngest participants, especially young men. In both surveys, less than half of those invited participated, hence, selection bias might be an issue. Also, as there is no official registry on ethnicity, we do not know if non-participation differed by ethnicity. It might be expected that Sami would be less willing to participate, given the history of assimilation[15] and unethical research.[25] On the other hand, as the surveys were carried out by a Sami research centre, invitees with Sami affiliations might have been more motivated to participate. If that is the case, the slightly adverse pattern in Sami, might be due, in part, to a different selection of Sami compared to non-Sami participants.

Further, due to design issues of SAMINOR 1,[22] the study sample of that survey included a lower proportion of participants from Sami-dominated municipalities in Finnmark, while the same municipalities had an overall high response rate in SAMINOR 2. This influences the ethnic and regional compositions of the two samples, and makes comparisons between the surveys challenging. However, sensitivity analyses regarding regions and ethnicity were consistent with the main results and did not indicate large variations between regions or when a different ethnic categorisation was used. Moreover, generalisation to the entire Sami and

non-Sami populations in Northern Norway is not advised, as only 10 municipalities were included. However, assuming a similar response rate in Sami and non-Sami participants, we believe the findings are applicable to Sami and non-Sami women and men over 50 years of age living in the given geographical regions, and the sensitivity analyses confirm this.

The use of antihypertensive drugs increases with age[2] and during 1975–2010, the prevalence of treatment for hypertension increased by a factor of four in Norway.[26] However, the decline in systolic blood pressure most probably occurs independently of drug treatment for hypertension.[2] The proportion of women using anti-hypertensive drugs remained similar at both surveys, whereas the proportions of hypertensive women declined, which corresponds to a decline that is independent of treatment with anti-hypertensive drugs.[2] However, men showed an increase in the use of anti-hypertensive drugs, whereas the proportions of hypertensive men remained the same, which may indicate that treatment with anti-hypertensive drugs could have contributed to a decline in blood pressure.

The observed decrease in cholesterol, systolic blood pressure, and proportion of smokers, and the increase in waist circumference, corresponds well with evidence from studies in Western Europe[3, 4, 6, 7] and with national trends.[1, 2, 5, 8, 11, 27] Possible explanations are changes in behaviour and diet – in line with what is observed nationally[11] – decreases in smoking, less occupational physical activity, more frequent use of vehicles for transportation, higher consumption of fruits and vegetables, lower consumption of saturated fats, and an assumed smaller consumption of salt.[28] The decrease in systolic blood pressure may have been halted due to the increase in obesity over the last decades.[29]

In a cohort study in Finnmark (1987–2003), based on a follow-up of those participating in both the Finnmark 3 and SAMINOR 1 surveys, Hermansen et al[30] observed – using the same ethnicity definition as in our study – that changes in cardiovascular risk factors according to change in physical activity level occurred independently of ethnicity. Similarly, in our study, population average changes in cardiovascular risk factors did not differ substantially by ethnicity, but small differences were observed in total cholesterol and triglycerides in men, and in HDL in both sexes, which suggests that Sami and non-Sami populations have undergone similar lifestyle changes. This might be considered unexpected, as Sami may be perceived as distinct from non-Sami in terms of diet[31, 32] and physical activity.[30] A recent study from SAMINOR 2 found that participants who defined themselves solely as Sami had a lower consumption of vegetables, and a higher consumption of moose meat, reindeer meat, and fat spread on bread than non-Sami and those who regard themselves as both Sami and non-Sami.[31] In SAMINOR 1 (all 24 municipalities included), a higher consumption of unfiltered coffee was observed in Sami participants compared with non-Sami and Sami of mixed ethnic descent.[32] Furthermore, unpublished results from SAMINOR 2 (Borch, K., personal communication, 2018), show that, in women, Sami ethnicity was associated with lower total physical activity. In the cohort study by Hermansen et al, the proportion of leisure-time sedentary individuals in Finnmark decreased between 1987 and 2003; the proportions who were sedentary was higher in Sami than in non-Sami, at baseline and at the end of follow-up.[30] Nonetheless, evidence of relevant ethnic differences in changes in cardiovascular risk factors and estimated 10-year risk of AMI and CS, was not found in our study.

The observed decline in cardiovascular risk factors is likely to have a beneficial impact on the incidence of coronary heart diseases[33] and ischemic stroke[34] in this population, which is

also reflected by a decrease in the estimated 10-year risk of AMI or CS. The decrease in risk was smaller in Sami than non-Sami women, which might be due to the increase in HDL cholesterol in non-Sami women. However, the causal effect of low levels of HDL cholesterol on cardiovascular disease is debated.[35, 36]

Inuit populations are characterised by a rapid increase in obesity, diabetes, and hypertension in parallel with decreasing physical activity and deterioration of the lipid profile.[37] On the other side, decline in smoking and alcohol use have been observed.[37] Furthermore, there are still disparities in cardiovascular health between Indigenous peoples and their benchmark populations in high-income countries.[37, 38] Our study indicated that such disparities in cardiovascular risk factors are not present in Northern Norway. Previous studies have also shown similar burdens of cardiovascular risk factors and morbidity among Sami and non-Sami in Norway.[17-21] This might be due to the fact that the non-Sami reference population in these studies[17-21] lives side by side with the Sami in the same rural regions. This is a stark contrast to, for instance, the Inuit and reference Danish population, who live on different continents. If we had compared the Sami to the general Norwegian population, we might have found larger differences in cardiovascular risk factors, as there are disparities in health issues.[11] Second, the small or non-existent disparities in health between Sami and non-Sami are suggested to be due to similar access to health care and education,[39] whereas the lack of similar access has been put forward as a reason for health disparities between the Inuit and their reference population.[37] In summary, differences in settlement patterns and in the social determinants of health challenge our ability to compare our results with international data.

We were not able to adjust for lipid-lowering drugs, physical activity, coffee and alcohol consumption, affiliation with reindeer herding, or diet in our study, as questions relating to

these items in the two surveys were not comparable. Lipid-lowering drugs are estimated to account for approximately 20–30% of the decline in total cholesterol over time,[5, 6] and therefore it is likely that some of the decline in cholesterol is due to the use of these drugs.

In the future, we plan to link SAMINOR 1 data to mortality and morbidity registries to explore differences in the incidence of cardiovascular diseases and whether differences in risk factors at baseline can explain this.

CONCLUSION

From SAMINOR 1 (2003–2004) to SAMINOR 2 (2012–2014), the population of rural Northern Norway had a favourable decline in total cholesterol, blood pressure, hypertension (women only), proportion of smokers, and the estimated 10-year risk of AMI or CS; however, they had an increase in waist circumference. We found only minor differences between Sami and non-Sami subjects regarding change in cardiovascular risk factors during this period, which suggests that the population of Northern Norway have had similar changes in health behaviour and diet.

PARTICIPANT AND PUBLIC INVOLVEMENT

If participants had pathological findings from the clinical examination, they were recommended to contact their primary physician. In emergency situations, participants were sent directly to the local health centre or the nearest hospital.

The Centre for Sami Health Research had consultations with the Sami Parliament, Sami researchers, and health workers in Sami core areas to identify the needs of the Sami community. Results from the surveys were reported to decision makers at the municipal and

regional levels, and to the Sami Parliament and national health authorities. The population was informed through popular science forums, meetings, and lectures.

CONTRIBUTION FROM CO-AUTHORS

The study was conceived by BME and TB. SRAS performed all the data analyses, produced the tables and drafted the manuscript. MM produced the figure. TB guided and assisted with statistical analyses. All authors helped with the interpretation of the results, contributed to the revision of the manuscript, and approved the final version.

SHARING OF DATA

The data for this study are not available for the public, since the use of data is restricted by license. Data might, however, be available if a written request is sent to and accepted by the SAMINOR Project Board (www.saminor.no) and by the Regional Committee for Medical and Health Research Ethics.

DECLARATION OF CONFLICTING INTERESTS

There are no conflicts of interest.

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FIGURE LEGEND

Figure 1. Inhabitants aged 40–79 years living in these 10 municipalities in the Norwegian part of Sápmi were invited to the SAMINOR 1 and SAMINOR 2 surveys. Region 1 includes Kautokeino and Karasjok, region 2 includes Nesseby, Tana, and Porsanger, and region 3 includes Kåfjord, Lyngen, Storfjord, Skånland, and Evenes.

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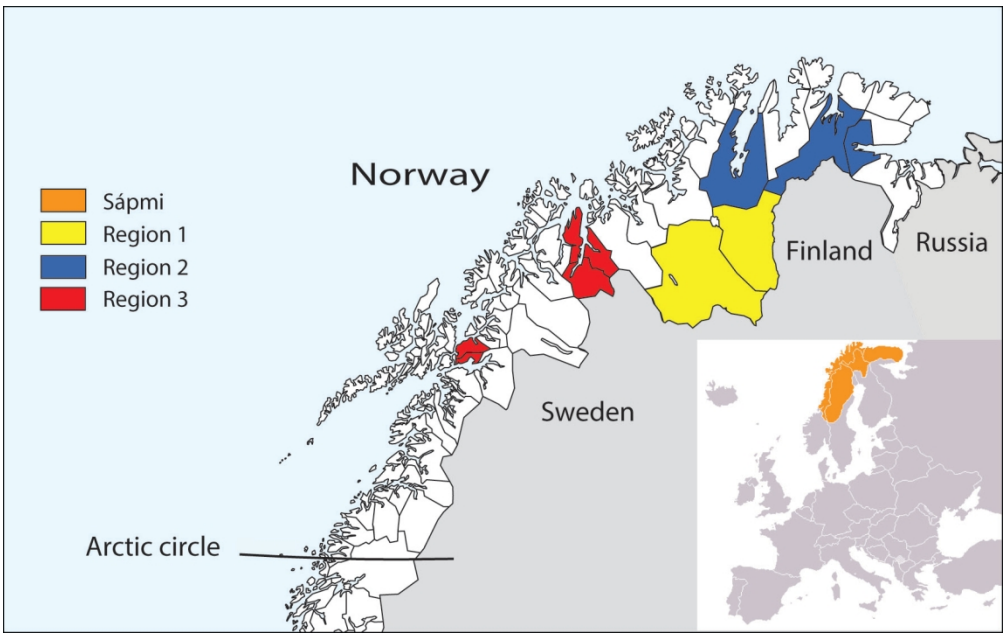

Inhabitants aged 40–79 years living in these 10 municipalities in the Norwegian part of Sápmi were invited to the SAMINOR 1 and SAMINOR 2 surveys. Region 1 includes Kautokeino and Karasjok, region 2 includes Nesseby, Tana, and Porsanger, and region 3 includes Kåfjord, Lyngen, Storfjord, Skånland, and Evenes.

STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of *cross-sectional studies*

Section/Topic	Item #	Recommendation	Reported on page #
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	
Objectives	3	State specific objectives, including any prespecified hypotheses	
Methods			
Study design	4	Present key elements of study design early in the paper	
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants	
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	
Bias	9	Describe any efforts to address potential sources of bias	
Study size	10	Explain how the study size was arrived at	
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	
		(*b*) Describe any methods used to examine subgroups and interactions	
		(*c*) Explain how missing data were addressed	
		(*d*) If applicable, describe analytical methods taking account of sampling strategy	
		(*e*) Describe any sensitivity analyses	
Results			

Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	
		(b) Give reasons for non-participation at each stage	
		(c) Consider use of a flow diagram	
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	
		(b) Indicate number of participants with missing data for each variable of interest	
Outcome data	15*	Report numbers of outcome events or summary measures	
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	
		(*b*) Report category boundaries when continuous variables were categorized	
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	
Discussion			
Key results	18	Summarise key results with reference to study objectives	
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	
Generalisability	21	Discuss the generalisability (external validity) of the study results	
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

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Changes in conventional cardiovascular risk factors and the estimated 10-year risk of acute myocardial infarction or cerebral stroke in Sami and non-Sami populations in two population-based cross sectional surveys—the SAMINOR Study

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CHANGES IN CONVENTIONAL CARDIOVASCULAR RISK FACTORS AND THE ESTIMATED 10-YEAR RISK OF ACUTE MYOCARDIAL INFARCTION OR CEREBRAL STROKE IN SAMI AND NON-SAMI POPULATIONS IN TWO POPULATION-BASED CROSS-SECTIONAL SURVEYS —THE SAMINOR STUDY

Susanna Ragnhild A. Siri[1], Bent Martin Eliassen[2], Bjarne K. Jacobsen[1,3], Marita Melhus[1], Ann Ragnhild Broderstad[1], Vilde Lehne Michalsen[1], Tonje Braaten[3]

[1]Centre for Sami Health Research, Department of Community Medicine, Faculty of Health Sciences, UiT The Arctic University of Norway, Tromsø, Norway. [2] Faculty of Nursing and Health Sciences, Nord University, Bodø, Norway. [3]Department of Community Medicine, Faculty of Health Sciences, UiT The Arctic University of Norway, Tromsø, Norway.

Corresponding author: Susanna Ragnhild A. Siri

Postal address: Centre for Sami Health Research, Department of Community Medicine, UiT The Arctic University of Norway. Postboks 6050, Langnes. 9037 Tromsø

E-mail: susanna.r.siri@uit.no

Telephone number: 0047-77646977

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Number of supplement tables: 3

Number of figures: 1

Number of supplement figures: 2

Abstract

Objective: To describe changes in cardiovascular risk factors and in the estimated 10-year risk of acute myocardial infarction (AMI) or cerebral stroke (CS) between SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014), and explore if these changes differed between Sami and non-Sami.

Design: Two cross-sectional surveys.

Setting: Inhabitants of rural Northern Norway.

Participants: Participants were aged 40–79 years and participated in SAMINOR 1 (n=6417) and/or SAMINOR 2 (n=5956).

Primary outcome measures: Generalised estimating equation regressions with an interaction term were used to estimate and compare changes in cardiovascular risk factors and 10-year risk of AMI or CS between the two surveys and by ethnicity.

Results: Mean cholesterol declined by 0.50, 0.43, and 0.60 mmol/L in women, Sami men, and non-Sami men, respectively (all p<0.001). In both sexes, changes in mean high-density lipoprotein (HDL) cholesterol and mean triglycerides were minor. Systolic and diastolic blood pressure declined by 3.6 and 1.0 mmHg in women, and 3.1 and 0.7 in men, respectively (all p<0.01). Mean waist circumference increased by 6.7 and 5.9 cm in women and men, respectively (both p<0.001). The odds of being a smoker declined by 35% in women and 46% in men (both p<0.001). Estimated 10-year risk of AMI or CS decreased in all strata of sex and ethnicity (p<0.001). For outcomes that varied by ethnicity, Sami had less favourable changes; however, the differences were minor. In men, ethnic differences were observed in total and HDL cholesterol, and in triglycerides, whereas in women, differences were observed in HDL cholesterol and in the 10-year risk of AMI or CS.

Conclusions: Independent of ethnicity, there was a decline in mean cholesterol, blood pressure, smoking, hypertension (women only), and 10-year risk of AMI or CS, but waist

circumference increased. Relatively minor ethnic differences were found in changes of cardiovascular risk factors.

ARTICLE SUMMARY

Strengths and limitations:

- We used generalised estimating equation regression to account for overlapping samples.
- We used self-reported measures to categorise participants into ethnic groups, including questions on self-perceived ethnicity, ethnic background, and language use.
- Due to lack of ethnic identifiers in national registries, we do not know if participation differs by ethnicity.
- We have an acceptable participation rate in both surveys.
- We lack information about the use of lipid-lowering drugs.

INTRODUCTION

Since the 1970s, a favourable decline in systolic blood pressure,[1-4] total cholesterol[5-7] and smoking[8] has been reported for the adult population across different regions of Western Europe. This decline is probably due to changes in lifestyle and diet,[7, 9, 10] in addition to use of medication.[1, 5] In Norway, this decline has coincided with a decrease in cardiovascular mortality and an increased prevalence of obesity and a sedentary lifestyle.[11]

The Sami is an Indigenous people living in Sápmi, i.e. the northern parts of Norway, Sweden, Finland, and the Kola Peninsula in the Russian Federation. There are no official population records on the Sami population, but data from the 1970 national census roughly estimated that there were 40,000 Sami in Norway,[12] whereas 55,000 is the population number that the Sami Parliament uses when considering subsidy schemes for business development.[13] In 2017, approximately 17,000 Sami adults were enrolled in the electoral register to the Sami Parliament in Norway, which gives them the right to vote and be elected.[14] The Sami people have unique cultures and languages, but these have partly vanished or at least declined in practice, due to structural assimilation that occurred from 1850–1960.[15] The Norwegian part of Sápmi is also inhabited by Norwegians and Kvens, the latter of whom are descendants of Finnish-speaking people that came from Sweden and Finland to Northern Norway in the 1700s and 1800s.[16]

Surveys from Norway have concluded that there are no or only minor differences in cardiovascular risk factors and morbidity between the Sami and non-Sami in rural regions.[17-21] However, knowledge is lacking on changes in conventional cardiovascular risk factors. Thus, this study aimed to describe changes in cardiovascular risk factors and in the estimated 10-year risk of acute myocardial infarction (AMI) or cerebral stroke (CS) between

SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014), and explore if these changes differed between Sami and non-Sami.

METHODS

We used data from two cross-sectional surveys of the Population-based Study on Health and Living Conditions in Regions with Sami and Norwegian Populations (The SAMINOR Study): the SAMINOR 1 Survey carried out in 2003–2004 (SAMINOR 1) and the SAMINOR 2 Clinical Survey carried out in 2012–2014 (SAMINOR 2). SAMINOR 1 was a collaboration between the Centre for Sami Health Research at UiT The Arctic University of Norway and the Norwegian National Institute of Public Health,[22] whereas SAMINOR 2 was performed by the former only. Participants were invited from 10 municipalities (Figure 1) that, according to the population census from 1970[12], had high proportions of Sami inhabitants. Invitations were mailed to all who were aged 40–79 and were registered as inhabitants in the 10 municipalities by the National Registry. In total, 11,518 and 12,455 received an invitation to SAMINOR 1 and SAMINOR 2, respectively. Participation was voluntarily and clinical examinations in each municipality were conducted within a period of one to seven weeks, depending on the population size. Our analyses were restricted to those who attended clinical examinations, gave blood samples, and answered the self-administered questionnaires.

Study sample

There were 6550 (56.9%) and 6004 (48.2%) individuals that attended the clinical examinations in SAMINOR 1 and 2, respectively. If information on ethnicity was lacking in one of the surveys, ethnicity information given in the other survey was used, as Sami ethnicity is found to be stable.[23] This strategy was valuable for the SAMINOR 1 sample, as ethnicity information was lacking for some participants due to the study design.[22] In SAMINOR 1, we

categorised 69 out of 201 by using ethnicity information from SAMINOR 2: 7 non-Sami and 62 Sami. In SAMINOR 2, 96 had missing data on ethnicity and we categorised 58: 37 non-Sami and 21 Sami. Furthermore, we excluded those that did not hand in the main questionnaires (SAMINOR 1: n=1; SAMINOR 2: n=10). This left us with a final sample of 6417 and 5956 from SAMINOR 1 and 2, respectively, wherein 3249 participated in both surveys.

The Norwegian Data Inspectorate and the Regional Committee for Medical and Health Research Ethics for region North (REC North) have approved The SAMINOR Study. The REC North (2015/2204–11) and the SAMINOR Project Board have also approved this study. All participants included in this study gave written informed consent and consented to linkage between the surveys.

Information from questionnaires

Participants were categorised into ethnic groups based on information from the following 11 questions, which were identical in the two surveys: "What language(s) do/did you, your parents, and your grandparents use at home?"; "What is your, your father's, and your mother's ethnic background?"; "What [ethnicity] do you consider yourself to be?" The response options were "Norwegian", "Sami", "Kven" and "Other" and multiple answers were allowed. Participants were defined as Sami if they 1) considered themselves to be Sami, or reported a Sami ethnic background for themselves, and 2) spoke a Sami language themselves or had at least one parent or grandparent that used it at home. All others were categorised as non-Sami. Sensitivity analyses were performed, in which different ethnic categorisations were used.

Smoking status was determined by the following questions, in SAMINOR 1: "Are you currently, or were you previously a daily smoker?" (Yes, currently/Yes, previously/Never); in SAMINOR 2: "Have you ever smoked daily?" (Yes/No), and "Are you currently a daily smoker?" (Yes/No). Previous and never smokers were categorised as non-smokers.

Use of anti-hypertensive drugs was determined by the following question: "Do you take medication for high blood pressure?" (Currently/Previously, but not now/Never used). Previous use, never-use and missing values were merged into non-use.

In both surveys, participates reported if they ever have had myocardial infarctions and age at first time. Positive responses to the former, or age reported for first time, were considered as having had a myocardial infarction.

Leisure time physical activity was measured in SAMINOR 1 by the "Saltin-Grimby" questionnaire.[24] Overall physical activity at current age was measured in SAMINOR 2 by a scale ranging from 1–10; an instrument validated in middle aged women living in Tromsø, Norway.[25]

Alcohol consumption was measured in SAMINOR 1 by asking: "How often during the last year have you consumed alcohol?" (Never/Not during the last year/A few times during the last year/1 time per month/2–3 times per month/1 time per week/2–3 times per week/4–7 times per week). To approximate the question in SAMINOR 2, we created two categories: never consumed alcohol and consumers of alcohol. In SAMINOR 2, alcohol consumption was asked as follows: "Do you practice total alcohol abstinence? " (Yes/no).

Education was measured similarly in both surveys by years of education. We categorised the item to match roughly primary and lower secondary school, upper secondary school and higher education: ≤ 9 years, 10–12 years and ≥13 years.

Clinical examination

Trained staff conducted the clinical examination. Waist circumference was measured at the umbilicus when the participant was standing. Blood pressure was measured with digital oscillometric devices (SAMINOR 1: DINAMAP-R, Criticon, Tampa, Florida, USA; SAMINOR 2: CARESCAPE^{TM}V100 monitor, GE Healthcare, Milwaukee, Wisconsin, USA), with the participant in a seated position. Following a 2-minute rest, three recordings were made at 1-minute intervals, and the average of the last two measurements was used in the analysis. Participants were considered to have hypertension if their systolic blood pressure was ≥140 mmHg, or their diastolic blood pressure was ≥90 mmHg, or if they reported using anti-hypertensive drugs.

In both surveys, non-fasting blood samples were collected. The blood samples were left to coagulate for a minimum of 30 minutes, after which they were centrifuged and serum was separated within 2 hours. In SAMINOR 1, serum was sent by overnight post and analysed consecutively for lipids (total cholesterol, high-density lipoprotein (HDL) cholesterol and triglycerides) with an enzymatic method (Hitachi 917 auto analyser, Roche Diagnostics, Switzerland) at Ullevål University Hospital, Oslo, Norway. In SAMINOR 2, serum samples were kept at -20° C before they were sent to the biobank at UiT The Arctic University of Norway, for further storage at -70° C. The samples were analysed in batches during autumn 2014 at the University Hospital of North Norway, Tromsø, Norway. Lipids were measured

with an enzymatic colorimetric method (Cobas 8000B, Roche Diagnostics GmbH, Mannheim, Germany).

The 10-year absolute risk of fatal or non-fatal AMI or CS was estimated by the NORRISK 2 model[26] and determined separately in women and men based on age, total cholesterol, HDL cholesterol, smoking status, systolic blood pressure, and use of anti-hypertensive drugs.

Statistical analyses

Statistical analyses were done using STATA version 15.0 (StataCorp, College Station, Texas, USA). Sample characteristics were given by sex for Sami and non-Sami in SAMINOR 1 and 2: means (standard deviations) of continuous variables and proportions (numbers) of categorical variables. In order to account for the partly overlapping samples, changes in population average means and prevalences of risk factors between SAMINOR 1 and SAMINOR 2 were estimated by sex- and ethnicity-specific linear or logistic generalised estimating equation regression models. Assumptions of normality and homoscedasticity were assessed by a visual inspection of residual plots. Changes in triglycerides and in the estimated 10-year risk of AMI or CS were log-transformed due to skewed distributions. All regression models were adjusted for age, and linear models were additionally adjusted for age squared. We assessed if changes in outcomes differed by ethnicity by including an interaction term between survey and ethnicity in sex-specific models. If the p-value for interaction was >0.05, the interaction term was excluded from the model and an overall sex-specific mean/prevalence was reported. In the opposite case, ethnicity-specific changes were reported. Marginal means/prevalences were estimated at age 57.5 years in women and at 58.2 years in men, i.e. the sex-specific mean ages in the overall sample. Two-way graphs illustrate how cardiovascular risk factors varied by age, ethnicity and survey (Supplementary Figures S1 and

S2). Potential heterogeneity by age in the overall models was assessed by comparing two strata divided at sex-specific mean age. The terms for interaction between ethnicity and survey remained non-significant across age strata for both sexes. Hence, we concluded that age did not modify the overall estimates of change in cardiovascular risk factors. We considered a two-sided $p<0.05$ to be significant.

Sensitivity analyses were done with same sex-stratified generalised estimating equation models by

1. Dividing the study sample into three groups: 1) those who reported "Sami" for all 11 questions, 2) who reported Sami in one to 10 questions, and 3) those who did not report Sami on any of the questions (non-Sami) (Supplementary Tables S1 and S2).
2. Using the original ethnic categorisation, we adjusting for geographical regions: 1) Kautokeino and Karasjok, 2) Nesseby, Tana, and Porsanger, 3) Kåfjord, Lyngen, Storfjord, Skånland, and Evenes (Supplementary Table S3).

RESULTS

Of the total sample, 53.5% were women. In women and men, 37.8% and 39.5% were Sami, respectively. The mean age was higher in both sexes in SAMINOR 2 than in SAMINOR 1. In both surveys, Sami women (Table 1) and men (Table 2) were less physically active, and Sami women reported more often to be non-consumers or abstainers of alcohol.

Both non-Sami and Sami women had a decline in total cholesterol between SAMINOR 1 and SAMINOR 2 ($p<0.001$, Table 3). The overall change in total cholesterol in women was -0.50 mmol/l. Sami women had lower HDL cholesterol and higher triglycerides than non-Sami in both surveys (Table 3). The change in triglycerides did not differ by ethnicity, but the change

in HDL cholesterol did, with non-Sami showing a minor increase, and Sami showing no change.

In both surveys, Sami women had somewhat lower blood pressure than non-Sami did (Table 3). The overall decline in systolic and diastolic blood pressure was 3.6 and 1.0 mmHg (both $p<0.001$), respectively; these changes did not differ by ethnicity. Roughly, 23% of women reported use of anti-hypertensive drugs, and this did not change over time. The prevalence of hypertension declined in a similar magnitude in Sami and non-Sami women; by 6.2 percentage points ($p<0.001$) (Table 3).

Non-Sami and Sami women had a similar increase of 6.7 cm in mean waist circumference. The prevalence of smoking in non-Sami and Sami women declined by 10.0 and 5.6 percentage points, respectively (both $p<0.001$); this change did not differ by ethnicity. Overall, the odds of current smoking declined by 35% (Table 3).

The estimated 10-year risk of AMI or CS declined between SAMINOR 1 and SAMINOR 2 in both Sami and non-Sami women (both $p<0.001$, Table 3), but more so in non-Sami.

Table 1. Unadjusted means and proportions of sample characteristics in women aged 40–79 years participating in SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014).

	SAMINOR 1 (n=3390)		**SAMINOR 2** (n=3234)	
Ethnicity	Non-Sami	Sami	Non-Sami	Sami
Proportions, % (n)	64.7 (2193)	35.3 (1197)	59.7 (1929)	40.4 (1305)
Age, mean (SD)	56.5 (10.1)	55.5 (10.2)	59.1 (10.3)	58.6 (10.4)
Self-reported myocardial infarction [a], % (n) †	2.6 (58)	1.9 (23)	3.2 (62)	1.8 (23)
Physical activity using "Saltin-Grimby" questionnaire†			‡	‡
Reading, watching TV or other sedentary activity, % (n)	21.3 (415)	27.7 (297)	-	-
Walking, bicycling or moving around in other ways at least 4 hour/week, % (n)	68.1 (1330)	61.9 (664)	-	-
Participation in recreational sports, heavy garden work etc. Duration at least 4 hours/week, % (n)	10.3 (200)	9.6 (103)	-	-
Participation in hard training or athletic competitions regularly and several times/week, % (n)	0.4 (7)	0.8 (8)	-	-
Level of physical activity on a scale from 1–10, mean (SD)†	‡	‡	5.6 (2.08)	5.2 (2.16)
Never consumed alcohol, % (n)†	14.8 (309)	24.5 (279)	‡	‡
Alcohol abstinence, yes % (n) †	‡	‡	18.4 (341)	27.0 (337)
Years of education, mean (SD)†	10.9 (3.8)	10.7 (4.6)	12.2 (4.0)	12.4 (4.5)
0–9 years education, % (n)	41.7 (864)	43.6 (497)	28.0 (530)	30.3 (385)
10–12 years education, % (n)	30.2 (626)	23.2 (265)	29.9 (565)	23.2 (295)
≥13 years of education, % (n)	28.1 (584)	33.1 (378)	42.1 (797)	46.5 (592)
Region 1: Kautokeino and Karasjok, % (n)	3.8 (84)	44.8 (537)	5.2 (101)	48.4 (631)
Region 2: Nesseby, Tana and Porsanger, % (n)	27.5 (603)	38.3 (458)	30.1 (580)	36.6 (478)
Region 3: Kåfjord, Lyngen, Storfjord, Skånland and Evenes, % (n)	68.7 (1506)	16.9 (202)	64.7 (1248)	15.0 (196)

† Based on a lower number due to missing values. [a] Measured differently in SAMINOR 1 and 2. ‡ Question not posed.

Table 2. Unadjusted means and proportions (%) of sample characteristics in men aged 40–79 years participating in SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014).

	SAMINOR 1 (n=3027)		**SAMINOR 2** (n=2722)	
Ethnicity	Non-Sami	Sami	Non-Sami	Sami
Proportions, % (n)	62.1 (1881)	37.9 (1146)	58.7 (1597)	41.3 (1125)
Age, mean (SD)	56.5 (9.8)	56.3 (10.1)	60.4 (10.2)	59.8 (10.3)
Self-reported myocardial infarction, % (n) †	6.9 (130)	6.5 (75)	8.8 (140)	8.1 (91)
Physical activity using "Saltin-Grimby" questionnaire†			‡	‡
Reading, watching TV or other sedentary activity, % (n)	20.2 (351)	24.3 (254)	-	-
Walking, bicycling or moving around in other ways at least 4 hour/week, % (n)	59.5 (1034)	53.0 (555)	-	-
Participation in recreational sports, heavy garden work etc. Duration at least 4 hours/week, % (n)	18.6 (324)	20.2 (212)	-	-
Participation in hard training or athletic competitions regularly and several times/week, % (n)	1.7 (29)	2.5 (26)	-	-
Level of physical activity on a scale from 1–10†, mean (SD)	‡	‡	5.2 (2.01)	5.12 (2.16)
Never consumed alcohol, % (n)†	5.4 (99)	4.5 (50)	‡	‡
Alcohol abstinence, yes % (n) †	‡	‡	10.6 (164)	13.4 (150)
Years of education, mean (SD) †	10.9 (3.7)	10.2 (4.1)	11.8 (3.6)	11.4 (3.8)
0–9 years education, % (n)	39.6 (719)	47.3 (519)	29.7 (467)	36.3 (400)
10–12 years education, % (n)	32.7 (594)	27.6 (303)	33.1 (520)	30.3 (333)
≥13 years of education, % (n)	27.7 (502)	25.1 (276)	37.2 (584)	33.4 (368)
Region 1: Kautokeino and Karasjok, % (n)	3.3 (63)	37.9 (434)	4.6 (73)	42.4 (477)
Region 2: Nesseby, Tana and Porsanger, % (n)	28.9 (543)	39.0 (447)	32.9 (525)	37.2 (419)
Region 3: Kåfjord, Lyngen, Storfjord, Skånland and Evenes, % (n)	67.8 (1275)	23.1 (265)	62.6 (999)	20.4 (229)

† Based on a lower number due to missing values. [a] Measured differently in SAMINOR 1 and 2.‡ Question not posed.

Table 3. Age-adjusted predicted changes in means and prevalences of cardiovascular risk factors between the SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014) in non-Sami and Sami women (n=6624).

	Non-Sami (n=4122)		Sami (n=2502)		Interaction ‡	Overall (n=6624)	
Linear regression	**β (95% CI)**	**p-value**	**β (95% CI)**	**p-value**	**p-value**	**β (95% CI)**	**p-value**
Total cholesterol, mmol/L	-0.51 (-0.57, -0.45)	<0.001	-0.47 (-0.55, -0.39)	<0.001	0.86	-0.50 (-0.54, -0.45)	<0.001
SAMINOR 1, mean †	6.24 (6.19, 6.30)		6.24 (6.16, 6.32)			6.25 (6.20, 6.29)	
SAMINOR 2, mean †	5.73 (5.67, 5.79)		5.77 (5.70, 5.85)			5.75 (5.70, 5.79)	
HDL cholesterol, mmol/L	0.05 (0.03, 0.07)	<0.001	-0.02 (-0.04, 0.01)	0.14	<0.001	*	
SAMINOR 1, mean †	1.51 (1.49, 1.53)		1.47 (1.45, 1.49)				
SAMINOR 2, mean †	1.56 (1.54, 1.58)		1.45 (1.43, 1.48)				
Triglycerides, mmol/L§	-0.02 (-0.05, 0.004)	0.10	0.03 (0.004, 0.07)	0.03	0.07	0.002 (-0.02, 0.02)	0.87
SAMINOR 1, mean †#	1.43 (1.40, 1.47)		1.48 (1.43, 1.53)			1.45 (1.43, 1.48)	
SAMINOR 2, mean †#	1.40 (1.37, 1.44)		1.53 (1.49, 1.58)			1.46 (1.43, 1.48)	
Systolic blood pressure, mm Hg	-3.9 (-4.86, -2.98)	<0.001	-3.0 (-4.23, -1.81)	<0.001	0.10	-3.6 (-4.36, -2.88)	<0.001
SAMINOR 1, mean †	134.7 (133.8, 135.6)		132.4 (131.2, 133.7)			133.9 (133.2, 134.6)	
SAMINOR 2, mean †	130.8 (129.9, 131.7)		129.4 (128.3, 130.6)			130.3 (129.6, 131.0)	
Diastolic blood pressure, mm Hg	-0.9 (-1.38, -0.33)	0.002	-1.1 (-1.77, -0.46)	0.001	0.39	-1.0 (-1.39, -0.57)	<0.001
SAMINOR 1, mean †	73.9 (73.4, 74.4)		73.4 (72.7, 74.1)			73.7 (73.3, 74.1)	
SAMINOR 2, mean †	73.0 (72.5, 73.6)		72.3 (71.6, 72.9)			72.7 (72.3, 73.2)	
Waist circumference, cm	7.0 (6.41, 7.49)	<0.001	6.1 (5.44, 6.76)	<0.001	0.26	6.7 (6.24, 7.07)	<0.001
SAMINOR 1, mean †	86.4 (85.83, 87.00)		88.0 (87.29, 88.78)			87.0 (86.55, 87.45)	
SAMINOR 2, mean †	93.3 (92.75, 93.94)		94.1 (93.43, 94.85)			93.7 (93.20, 94.11)	
10-year risk of AMI and CS, %§	-0.19 (-0.22, -0.17)	<0.001	-0.13 (-0.16, -0.09)	<0.001	0.011	*	
SAMINOR 1, mean †#	4.16 (4.05, 4.26)		3.91 (3.77, 4.05)				
SAMINOR 2, mean †#	3.43 (3.33, 3.52)		3.44 (3.32, 3.56)				
Logistic regression	**Odds ratio (95% CI)**	**p-value**	**Odds ratio (95% CI)**	**p-value**	**p-value**	**Odds ratio (95% CI)**	**p-value**
Anti-hypertensive treatment	0.96 (0.85, 1.09)	0.51	0.98 (0.83, 1.16)	0.82	0.77	0.96 (0.87, 1.07)	0.47
SAMINOR 1, prevalence % †	24.0 (22.10, 25.80)		22.9 (20.34, 25.36)			23.6 (22.11, 25.10)	
SAMINOR 2, prevalence % †	23.2 (21.17, 25.24)		22.5 (20.04, 24.98)			23.0 (21.38, 24.53)	
Hypertension	0.77 (0.68, 0.86)	<0.001	0.79 (0.68, 0.93)	0.003	0.56	0.77 (0.70, 0.85)	<0.001
SAMINOR 1, prevalence % †	47.4 (45.12, 49.72)		44.5 (41.37, 47.68)			46.3 (44.47, 48.20)	
SAMINOR 2, prevalence % †	40.8 (38.39, 43.25)		38.9 (35.90, 41.79)			40.1 (38.18, 41.94)	
Current smokers	0.59 (0.53, 0.66)	<0.001	0.74 (0.64, 0.85)	<0.001	0.10	0.65 (0.59, 0.71)	<0.001
SAMINOR 1, prevalence % †	31.0 (29.07, 32.90)		27.9 (25.28, 30.45)			29.8 (28.26, 31.34)	
SAMINOR 2, prevalence % †	21.0 (19.26, 22.76)		22.3 (20.07, 24.46)			21.6 (20.21, 22.94)	

β –coefficients are estimated by linear generalised estimating equation regression models and adjusted for age and age^2. Odds ratios are estimated by logistic generalised estimating equation regression models and adjusted for age. †Predicted means/prevalences at age 57.5 years, which is the mean age for women in the overall sample. § Outcome variables are log-transformed. #Predicted geometric means at age 57.5 year. ‡ Test of interaction between survey and ethnicity in overall model. If p-value for interaction >0.05, interaction term was excluded from the overall model. *p-value for interaction <0.05, only ethnicity-specific estimations are reported. Number of missing values: total and HDL cholesterol and triglycerides were missing in 18 subjects; systolic and diastolic blood pressure were missing in four subjects; hypertension in three. NORRISK 2 score was missing for 193 subjects. Excluding missing values did not change the results. Abbreviations: CI, confidence interval; AMS, acute myocardial infarction; CS, cerebral stroke.

Between SAMINOR 1 and SAMINOR 2, total cholesterol declined more in non-Sami than in Sami men (0.60 vs. 0.43 mmol/l; both p<0.001, Table 4), and this change varied by ethnicity. Between the surveys, Sami men had a slight decline in HDL cholesterol (p<0.001) and a slight increase in triglycerides (p<0.001); whereas non-Sami men had no changes, hence, changes in HDL cholesterol and triglyceride differed for Sami and non-Sami (Table 4).

In men, the decline in systolic and diastolic blood pressure did not differ by ethnicity (Table 4). The overall decline in systolic and diastolic blood pressure in men were 3.1 and 0.7 mmHg (both p<0.05), respectively. Overall, we found an increase in the prevalence of anti-hypertensive drug use, from 21.1% to 23.9% in men, which did not differ by ethnicity. The prevalence of hypertension remained similar in SAMINOR 1 and SAMINOR 2, with roughly half of men being considered hypertensive (Table 4).

Waist circumference increased similarly in Sami and non-Sami men, with an overall increase of 5.9 cm (p<0.001). The prevalence of smoking declined similarly in non-Sami and Sami men, by 12.3 and 10.0 percentage points (both p<0.001), respectively. The overall decline in the odds of being a smoker was 46%.

The estimated 10-year risk of AMI or CS declined in non-Sami and Sami men (both p<0.001, Table 4), but not differently in the two ethnic groups.

Sensitivity analyses

Overall, the sensitivity analyses were consistent with the main findings when using a different ethnic categorisation (Supplementary Tables S1 and S2) and when adjusting for region (Supplementary Table S3).

Table 4. Age-adjusted predicted changes in means and in prevalences of cardiovascular risk factors between the SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014) in non-Sami and Sami men (n=5749).

	Non-Sami (n=3478)		Sami (n=2271)		Interaction ‡	Overall (n=5749)	
Linear regression	**β (95% CI)**	**p-value**	**β (95% CI)**		**p-value**	**β (95% CI)**	**p-value**
Total cholesterol, mmol/L	-0.60 (-0.66, -0.53)	<0.001	-0.43 (-0.51, -0.35)	<0.001	0.03	*	
SAMINOR 1, mean †	6.00 (5.95, 6.07)		6.00 (5.92, 6.08)				
SAMINOR 2, mean †	5.41 (5.35, 5.48)		5.58 (5.50, 5.65)				
HDL cholesterol, mmol/L	-0.01 (-0.03, 0.01)	0.18	-0.06 (-0.08, -0.04)	<0.001	0.005	*	
SAMINOR 1, mean †	1.30 (1.28, 1.31)		1.28 (1.26, 1.31)				
SAMINOR 2, mean †	1.28 (1.26, 1.30)		1.22 (1.20, 1.25)				
Triglycerides, mmol/L§	0.001 (-0.03, 0.03)	0.96	0.09 (0.05, 0.13)	<0.001	0.001	*	
SAMINOR 1, mean †#	1.61 (1.56, 1.65)		1.58 (1.53, 1.64)				
SAMINOR 2, mean †#	1.61 (1.56, 1.66)		1.73 (1.67, 1.79)				
Systolic blood pressure, mm Hg	-3.2 (-4.19, -2.18)	<0.001	-2.8 (-4.11, -1.44)	<0.001	0.38	-3.1 (-3.87, -2.27)	<0.001
SAMINOR 1 mean †	137.0 (136.1, 137.9)		136.8 (135.5, 138.1)			136.9 (136.2, 137.7)	
SAMINOR 2, mean †	133.8 (132.8, 134.8)		134.0 (132.7, 135.3)			133.9 (133.1, 134.6)	
Diastolic blood pressure, mm Hg	-0.5 (-1.08, 0.11)	0.11	-1.1 (-1.82, -0.33)	0.004	0.08	-0.7 (-1.20, -0.28)	0.002
SAMINOR 1, mean †	79.7 (79.2, 80.2)		79.9 (79.2, 80.6)			79.8 (79.4, 80.2)	
SAMINOR 2, mean †	79.2 (78.6, 79.8)		78.9 (78.2, 79.5)			79.1 (78.6, 79.5)	
Waist circumference, cm	6.0 (5.45, 6.56)	<0.001	5.9 (5.22, 6.48)	<0.001	0.37	5.9 (5.50, 6.31)	<0.001
SAMINOR 1, mean †	94.4 (93.82, 94.90)		93.1 (92.45, 93.79)			93.9 (94.48, 94.32)	
SAMINOR 2, mean †	100.4 (99.78, 100.95)		99.0 (98.29, 99.64)			99.8 (99.36, 100.24)	
10-year risk of AMI and CS, %§	-0.19 (-0.22, -0.17)	<0.001	-0.16 (-0.19, -0.12)	<0.001	0.23	-0.18 (-0.20, -0.16)	<0.001
SAMINOR 1, mean †#	8.73 (8.52, 8.95)		8.75 (8.45, 9.04)			8.74 (8.57, 8.92)	
SAMINOR 2, mean †#	7.20 (7.00, 7.40)		7.48 (7.23, 7.73)			7.32 (7.17, 7.48)	
Logistic regression	**Odds ratio (95% CI)**	**p-value**	**Odds ratio (95% CI)**	**p-value**	**p-value**	**Odds ratio (95% CI)**	**p-value**
Anti-hypertensive treatment	1.19 (1.03, 1.36)	0.02	1.15 (0.97, 1.37)	0.11	0.37	1.17 (1.06, 1.31)	0.003
SAMINOR 1, prevalence % †	21.4 (19.48, 23.28)		20.7 (18.34, 23.11)			21.1 (19.60, 22.56)	
SAMINOR 2, prevalence % †	24.4 (22.15, 26.67)		23.1 (20.53, 25.74)			23.9 (22.18, 25.60)	
Hypertension	0.94 (0.83, 1.06)	0.32	0.89 (0.77, 1.04)	0.13	0.39	0.92 (0.83, 1.01)	0.08
SAMINOR 1, prevalence % †	51.0 (48.62, 53.42)		50.4 (47.43, 53.43)			50.7 (48.84, 52.63)	
SAMINOR 2, prevalence % †	49.4 (46.83, 52.01)		47.5 (44.39, 50.51)			48.6 (46.61, 50.57)	
Current smokers	0.51 (0.44, 0.58)	<0.001	0.59 (0.51, 0.69)	<0.001	0.27	0.54 (0.49, 0.60)	<0.001
SAMINOR 1, prevalence % †	30.4 (28.35, 32.50)		30.8 (28.16, 33.45)			30.7 (29.06, 32.35)	
SAMINOR 2, prevalence % †	18.1 (16.30, 19.97)		20.8 (18.55, 23.12)			19.4 (17.94, 20.81)	

β –coefficients are estimated by linear generalised estimating equation regression models and adjusted for age and age^2. Odds ratios are estimated by logistic generalised estimating equation regression models and adjusted for age. †Predicted means/prevalences at age 58.2 years, which is the mean age for men in the overall sample. §Outcome variables are log-transformed. #Predicted geometric means at age 58.2 year. ‡ Test of interaction between survey and ethnicity in overall model. If p-value for interaction >0.05, interaction term is excluded from the overall model. *p-value for interaction <0.05, only ethnicity-specific estimations are reported. Number of missing values: total and HDL cholesterol were missing in 12 subjects; triglycerides were missing in 13 subjects, systolic and diastolic blood pressure and hypertension was missing in one subject. NORRISK 2 score was missing for 173 subjects. Excluding missing values did not change the results. Abbreviations: CI, confidence interval; AMS, acute myocardial infarction; CS, cerebral stroke.

DISCUSSION

From SAMINOR 1 (2003–2004) to SAMINOR 2 (2012–2014), participants from the selected 10 municipalities in Northern Norway had a favourable decline in total cholesterol, blood pressure, proportion of smokers, and the estimated 10-year risk of AMI or CS, whereas waist circumference increased. The changes in total cholesterol (men only), HDL cholesterol (both sexes), triglycerides (men only), and the estimated 10-year risk of AMS or CS (women only), were statistically significantly different in Sami and non-Sami. The odds of anti-hypertensive drug use increased only in men, whereas the prevalence of hypertension decreased only in women. To our knowledge, there are no other studies in Sápmi that explore whether changes in cardiovascular risk factors differ between Sami and non-Sami over time.

In both SAMINOR 1 and SAMINOR 2, the participation rate was lowest among the youngest participants, especially young men. In both surveys, less than half of those invited participated, hence, selection bias might be an issue. Also, as there is no official registry on ethnicity, we do not know if non-participation differed by ethnicity. It might be expected that Sami would be less willing to participate, given the history of assimilation[15] and unethical research.[27] On the other hand, as the surveys were carried out by a Sami research centre, invitees with Sami affiliations might have been more motivated to participate. If that is the case, the slightly adverse pattern in Sami, might be partly due to a different selection of Sami compared to non-Sami participants.

Further, due to design issues of SAMINOR 1,[22] the study sample included a lower proportion of participants from Sami-dominated municipalities in Finnmark, while the same municipalities had an overall high response rate in SAMINOR 2. This influences the ethnic

and regional compositions of the two samples, and makes comparisons between the surveys challenging. However, when using a different categorisation of ethnicity or adjusting for region, the results remained consistent with the main results. Moreover, generalisation to the entire Sami and non-Sami populations in Northern Norway is not advised, as only 10 municipalities were included. However, assuming a similar response rate in Sami and non-Sami participants, we believe the findings are applicable to Sami and non-Sami women and men over 50 years of age living in the given geographical regions.

The use of antihypertensive drugs increases with age[2] and during 1975–2010, the prevalence of treatment for hypertension increased by a factor of four in Norway.[28] In our study, the use of anti-hypertensive drugs in women remained similar in the surveys, whereas the prevalence of hypertension in women declined, which corresponds to a decline that is independent of treatment with anti-hypertensive drugs.[2] In men, we observed an increase in the use of anti-hypertensive drugs, whereas the prevalence of hypertension remained the same, which may indicate that treatment with anti-hypertensive drugs could have contributed to a decline in blood pressure.

The observed decreases in cholesterol, systolic blood pressure, and proportion of smokers, and the increase in waist circumference, corresponds well with studies in Western Europe[3, 4, 6, 7] and with national trends.[1, 2, 5, 8, 11, 29] Possible explanations are changes in lifestyle and diet—in line with what is observed nationally[11] —decreases in smoking, less occupational physical activity, more frequent use of vehicles for transportation, higher consumption of fruits and vegetables, lower consumption of saturated fats, and an assumed lower consumption of salt.[30] The decrease in systolic blood pressure may have been halted due to the increase in obesity over the last decades.[31]

In a cohort study in Finnmark (1987–2003), based on a follow-up of those participating in both the Finnmark 3 and SAMINOR 1 surveys, Hermansen et al.[32] observed—using the same ethnicity definition as in our study—that changes in cardiovascular risk factors according to change in physical activity level occurred independently of ethnicity. Similarly, we observed that changes in cardiovascular risk factors did not differ substantially by ethnicity, only small and probably negligible differences were observed in total cholesterol and triglycerides in men, and in HDL cholesterol in both sexes, which suggests that Sami and non-Sami populations overall have undergone similar lifestyle changes. This might be considered unexpected, as Sami may be perceived as distinct from non-Sami in terms of diet[33, 34] and physical activity.[32] A recent study from SAMINOR 2 found that participants who defined themselves solely as Sami had a lower consumption of vegetables, and a higher consumption of moose meat, reindeer meat, and fat spread on bread than non-Sami and those who regard themselves as both Sami and non-Sami.[33] In SAMINOR 1 (24 municipalities included), a higher consumption of unfiltered coffee was observed in Sami participants compared with non-Sami and Sami of mixed ethnic descent.[34] Furthermore, unpublished results from SAMINOR 2 (Borch, K., personal communication, 2018), show that, in women, Sami ethnicity was associated with lower total physical activity. In the cohort study by Hermansen et al., the proportion of leisure-time sedentary individuals in Finnmark decreased between 1987 and 2003; however, the proportions who were sedentary was higher in Sami than in non-Sami, both at baseline and at the end of follow-up.[32] Nonetheless, evidence of relevant ethnic differences in changes in cardiovascular risk factors and estimated 10-year risk of AMI and CS, was not found in our study.

The observed decline in cardiovascular risk factors is likely to have a beneficial impact on the incidence of coronary heart diseases[35] and ischemic stroke[36] in this population, which is

also reflected by a decrease in the estimated 10-year risk of AMI or CS. The decrease in risk was smaller in Sami than non-Sami women, which might be due to the increase in HDL cholesterol in non-Sami women. However, the causal effect of low levels of HDL cholesterol on cardiovascular disease is debated.[37, 38]

Inuit populations are characterised by a rapid increase in obesity, diabetes, and hypertension in parallel with decreasing physical activity and deterioration of the lipid profile.[39] On the other hand, decline in smoking and alcohol use have been observed.[39] But still, there are disparities in cardiovascular health between Indigenous peoples and their benchmark populations in high-income countries.[39, 40] Our study indicated that such disparities in cardiovascular risk factors are not present in the 10 rural municipalities in Northern Norway. Previous studies have also shown similar burdens of cardiovascular risk factors and morbidity among Sami and non-Sami in Norway.[17-21] This might be due to the fact that the non-Sami reference population in these studies [17-21] lives side by side with the Sami in the same rural regions. This is a stark contrast to, for instance, the Inuit and reference Danish population, who live on different continents. If we had compared the Sami in this study to the general Norwegian population, we might have found larger differences in cardiovascular risk factors, as there are disparities in health issues across regions.[11] Second, the small or non-existent disparities in health between Sami and non-Sami are suggested to be due to similar access to health care and education,[41] whereas the lack of similar access has been put forward as a reason for health disparities between the Inuit and their reference population.[39] In summary, differences in settlement patterns and in the social determinants of health challenge our ability to compare our results with international data.

We were not able to adjust for lipid-lowering drugs, physical activity, coffee and alcohol consumption, affiliation with reindeer herding, or diet in our study, as questions relating to these items in the two surveys were not comparable. Lipid-lowering drugs are estimated to account for approximately 20–30% of the decline in total cholesterol over time,[5, 6] and therefore it is likely that some of the decline in cholesterol is due to the use of these drugs. The public health relevance of this study is that preventive measures aimed to reduce cardiovascular risk seem to have worked independent of ethnicity. Nevertheless, further surveillance of cardiovascular risk factors is advisable due to the adverse pattern—although minor—in Sami compared with non-Sami.

CONCLUSION

From SAMINOR 1 (2003–2004) to SAMINOR 2 (2012–2014), the population in rural Northern Norway had a favourable decline in total cholesterol, blood pressure, hypertension (women only), smoking, and the estimated 10-year risk of AMI or CS; however, they had an increase in waist circumference. We found only minor differences between Sami and non-Sami subjects regarding change in cardiovascular risk factors during this period, which suggests that the population of Northern Norway have had similar changes in lifestyle and diet.

PARTICIPANT AND PUBLIC INVOLVEMENT

If participants had pathological findings from the clinical examination, they were recommended to contact their primary physician. In emergency situations, participants were sent directly to the local health centre or the nearest hospital.

The Centre for Sami Health Research had consultations with the Sami Parliament, Sami researchers, and health workers in Sami core areas to identify the needs of the Sami community. Results from the surveys were reported to decision makers at the municipal and regional levels, and to the Sami Parliament and national health authorities. The population was informed through popular science forums, meetings, and lectures.

CONTRIBUTION FROM CO-AUTHORS

The study was conceived by BME and TB. SRAS performed all the data analyses, produced the tables and drafted the manuscript. MM produced the figure. TB guided and assisted with statistical analyses. BME, BKJ, MM, ARB, VLM and TB helped with the interpretation of the results, and contributed to the revision of the manuscript, and approved the final version.

SHARING OF DATA

The data for this study are not available for the public, since the use of data is restricted by license. Data might, however, be available if a written request is sent to and accepted by the SAMINOR Project Board (www.saminor.no) and by the Regional Committee for Medical and Health Research Ethics.

DECLARATION OF CONFLICTING INTERESTS

There are no conflicts of interest.

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FIGURE LEGEND

Figure 1. Inhabitants aged 40–79 years living in these 10 municipalities in the Norwegian part of Sápmi were invited to the SAMINOR 1 and SAMINOR 2 surveys. Region 1 includes Kautokeino and Karasjok, region 2 includes Nesseby, Tana, and Porsanger, and region 3 includes Kåfjord, Lyngen, Storfjord, Skånland, and Evenes.

Supplementary Figure S1: Cardiovascular risk factors in women according to ethnicity and mean age in 10-year age groups in SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014).

Supplementary Figure S2: Cardiovascular risk factors in men according to ethnicity and mean age in 10-year age groups in SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014).

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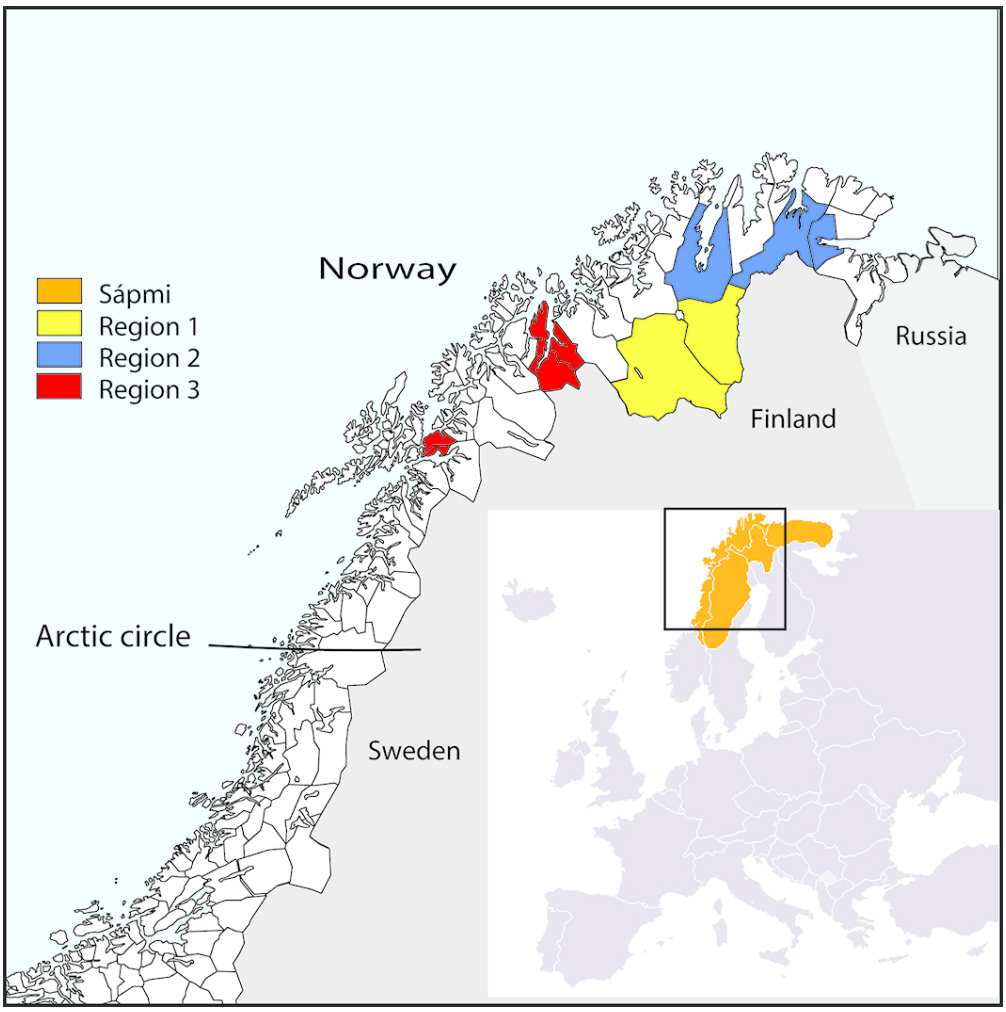
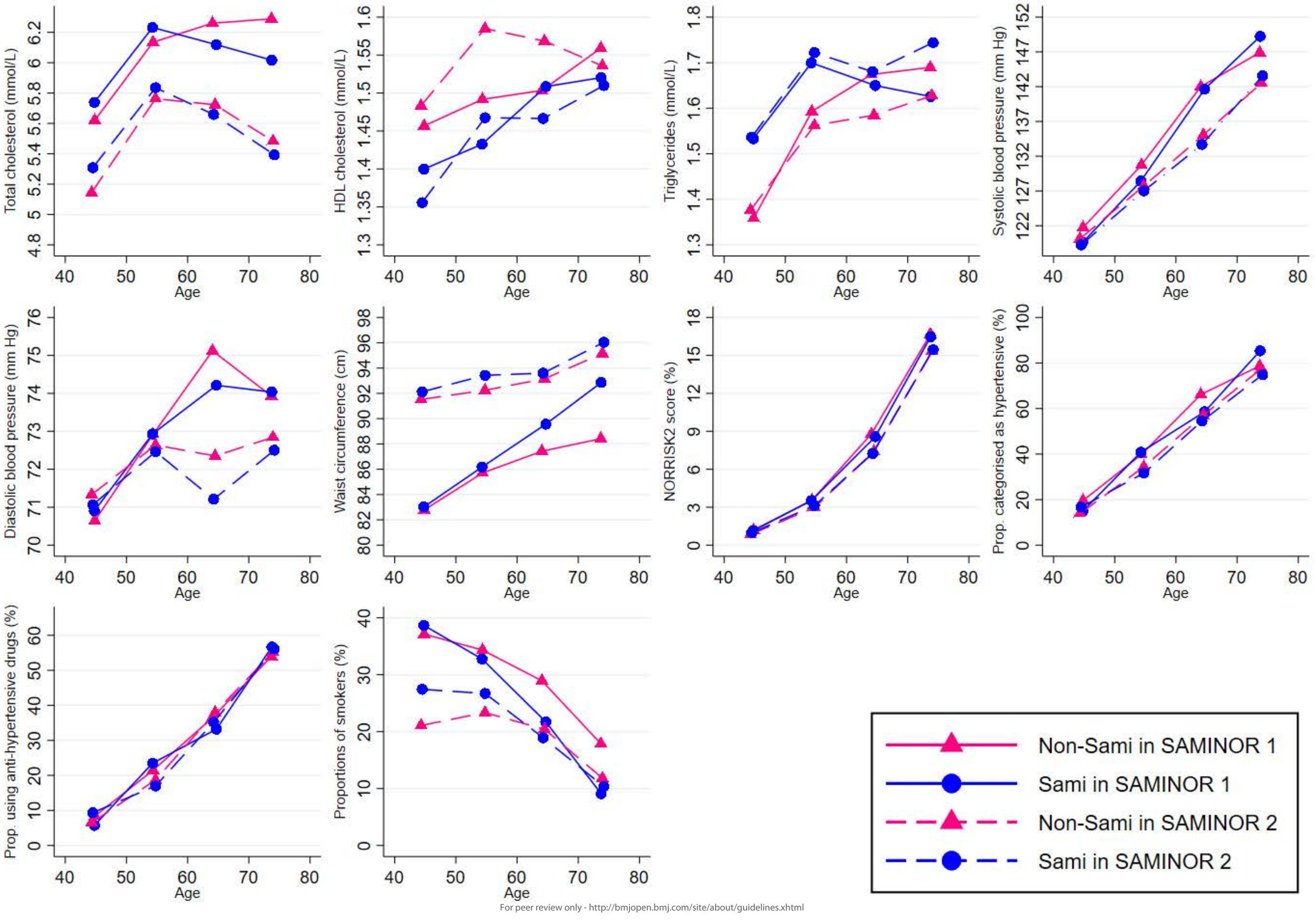


Figure 1. Inhabitants aged 40–79 years living in these 10 municipalities in the Norwegian part of Sápmi were invited to the SAMINOR 1 and SAMINOR 2 surveys. Region 1 includes Kautokeino and Karasjok, region 2 includes Nesseby, Tana, and Porsanger, and region 3 includes Kåfjord, Lyngen, Storfjord, Skånland, and Evenes.

Supplementary Figure S1. Cardiovascular risk factors in women according to survey, ethnicity and mean age in 10-year age groups



Supplementary Figure S2. Cardiovascular risk factors in men according to survey, ethnicity and mean age in 10-year age groups

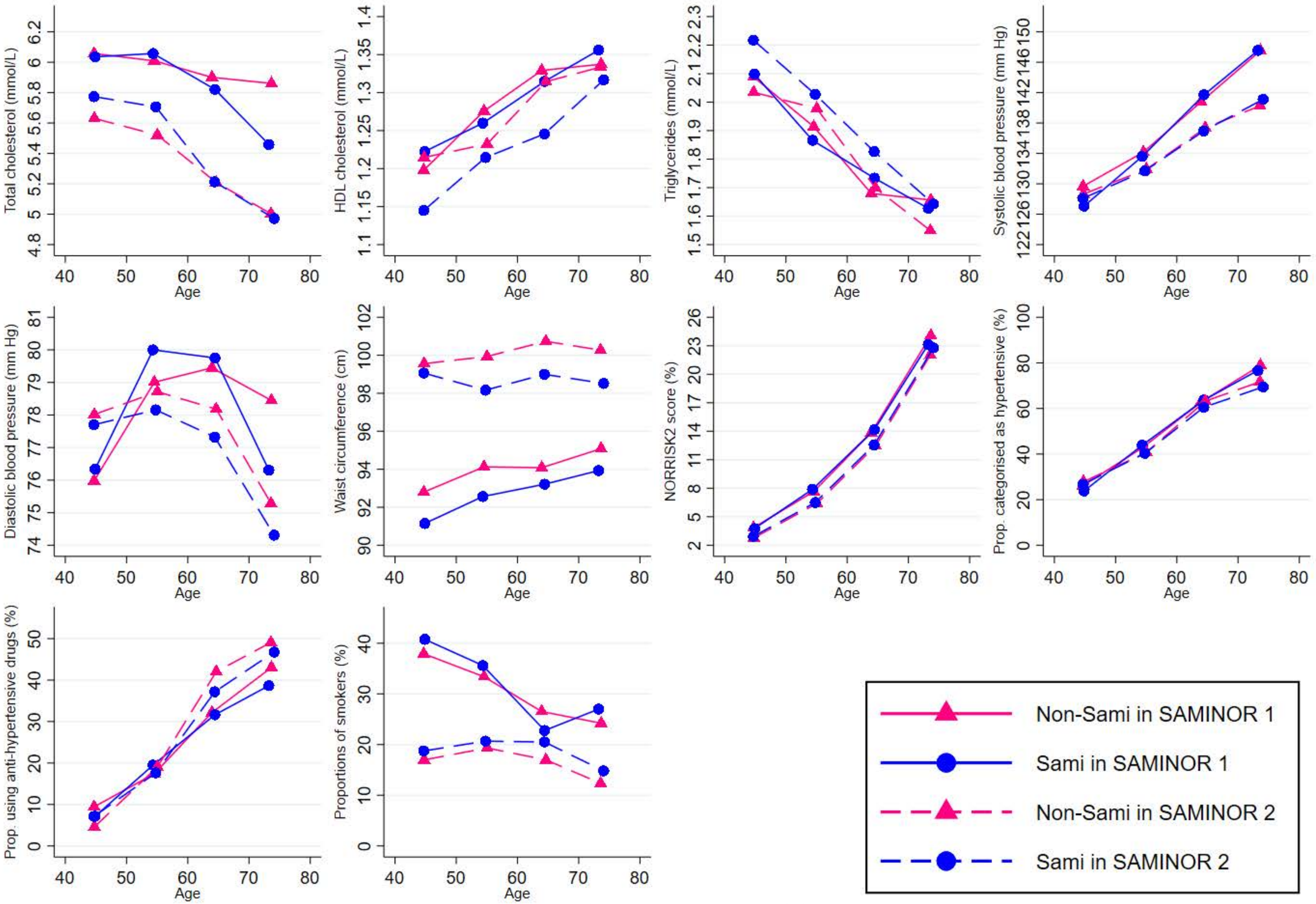


Table S1: Age-adjusted predicted changes in means and prevalences of cardiovascular risk factors between the SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014) in women (n=6624), stratified into three groups according to responses to ethnicity related questions.

Linear regression	**Non-Sami (n=3262)** β (95% Cl)	**p-value**	**Sami in all items (n=1453)** β (95% Cl)	**p-value**	**Sami in 1–10 items (n=1909)** β (95% Cl)	**p-value**	**Interaction‡**	**p-value**
Cholesterol, mmol/L	-0.55 (-0.61, -0.48)	<0.001	-0.44 (-0.55, -0.33)	<0.001	-0.46 (-0.55, -0.37)	<0.001	*	
SAMINOR 1, mean †	6.27 (6.21, 6.33)		6.23 (6.13, 6.34)		6.21 (6.12, 6.29)			
SAMINOR 2, mean †	5.72 (5.66, 5.79)		5.79 (5.70, 5.89)		5.75 (5.66, 5.83)			
HDL cholesterol, mmol/L	0.04 (0.02, 0.07)	<0.001	-0.03 (-0.06, -0.005)	0.02	0.03 (-0.002, 0.05)	0.07	Sami all items	<0.001
SAMINOR 1, mean †	1.53 (1.51 ,1.55)		1.47 (1.43, 1.50)		1.46 (1.43, 1.49)			
SAMINOR 2, mean †	1.58 (1.55, 1.60)		1.43 (1.40, 1.46)		1.48 (1.45, 1.51)			
Triglycerides, mmol/L§	-0.02 (-0.05, 0.004)	0.10	0.03 (-0.01, 0.07)	0.19	0.02 (-0.02, 0.05)	0.34	*	
SAMINOR 1, mean †#	2.21 (2.07, 2.34)		2.47 (2.23, 2.71)		2.59 (2.37, 2.80)			
SAMINOR 2, mean †#	2.09 (1.96, 2.23)		2.63 (2.39, 2.88)		2.69 (2.47, 2.92)			
Systolic blood pressure, mm Hg	-4.3 (-5.25, -3.13)	<0.001	-3.4 (-5.04, -1.83)	<0.001	-2.7 (-4.09, -1.23)	<0.001	*	
SAMINOR 1, mean †	134.6 (133.7, 135.6)		132.3 (130.7, 133.9)		133.7 (132.3, 135.1)			
SAMINOR 2, mean †	130.4 (129.4, 131.5)		128.8 (127.3, 130.4)		131.1 (129.7, 132.5)			
Diastolic blood pressure, mm Hg	-0.9 (-1.47, -0.28)	0.004	-1.4(-2.26, -0.57)	0.001	-0.8(-1.60, -0.02)	0.044	*	
SAMINOR 1, mean †	73.9 (73.34, 74.45)		73.2 (72.38, 74.06)		73.8 (73.00, 74.53)			
SAMINOR 2, mean †	73.0 (72.43, 73.62)		71.8 (71.00, 72.61)		73.0 (72.19, 73.71)			
Waist circumference, cm	7.1 (6.53,7.72)	<0.001	5.9 (4.97, 6.77)	<0.001	6.5 (5.75, 7.29)	<0.001	*	
SAMINOR 1, mean †	86.1 (85.48, 86.73)		88.6 (87.61, 89.59)		87.3 (86.43, 88.13)			
SAMINOR 2, mean †	93.2 (92.57, 93.90)		94.47 (93.52, 95.41)		93.8 (92.87, 94.63)			
10-year risk of AMI and CS, %§	-0.19 (-0.22, -0.16)	<0.001	-0.12 (-0.16,-0.07)	<0.001	-0.17 (-0.21,-0.12)	<0.001	Sami all items	0.02
SAMINOR 1, mean †#	4.09 (3.97, 4.21)		3.81 (3.63, 3.98)		4.22 (4.04, 4.40)			
SAMINOR 2, mean †#	3.37 (3.27, 3.48)		3.39 (3.24, 3.54)		3.58 (3.43, 3.73)			
Logistic regression	**Odds ratio (95% Cl)**	**p-value**	**Odds ratio (95% Cl)**	**p-value**	**Odds ratio (95% Cl)**	**p-value**		**p-value**
Antihypertensive drugs	0.96 (0.83, 1.11)	0.61	0.98 (0.77, 1.23)	0.84	0.95 (0.79, 1.14)	0.58	*	
SAMINOR 1, prevalences % †	22.9 (20.82, 24.94)		20.1 (16.96, 23.33)		27.2 (24.30, 30.15)			
SAMINOR 2, prevalences % †	22.2 (19.93, 24.50)		19.8 (16.60, 22.90)		26.2 (23.29, 29.16)			
Hypertension	0.78 (0.68, 0.90)	<0.001	0.76 (0.61, 0.94)	0.010	0.77 (0.65, 0.92)	0.004	*	
SAMINOR 1, prevalences % †	46.0 (43.45, 48.62)		42.9 (38.78, 47.05)		49.6 (46.06, 53.07)			
SAMINOR 2, prevalences % †	40.1 (37.29, 42.81)		36.3 (32.44, 40.20)		43.2 (39.76, 46.60)			
Current smoking	0.61 (0.53, 0.69)	<0.001	0.78 (0.64, 0.94)	0.011	0.63 (0.54, 0.74)	<0.001	*	
SAMINOR 1, prevalences % †	30.3 (28.15, 32.41)		26.3 (23.02, 29.61)		30.8 (27.94, 33.75)			
SAMINOR 2, prevalences % †	20.9 (18.91, 22.85)		21.7 (18.85, 24.61)		22.0 (19.50, 24.45)			

β –coefficients are estimated by linear generalised estimating equation regression models and adjusted for age and age^2. Odds ratios are estimated by logistic generalised estimating equation regression models and adjusted for age. †Predicted means/prevalences at age 57.5 years. §Outcome variables are log-transformed. # Predicted geometric means at age 57.5. ‡ Testing for interaction between survey and ethnicity using non-Sami as reference. If p-value for interaction <0.05, we have specified which group differed from non-Sami in SAMINOR 2. * If p-value for interaction between survey and ethnicity is >0.05, please refer to the overall estimation given in Table 3. Sami in all items (11 in total): reported use of Sami language in grandparents, parents and themselves; Sami ethnic background for parents and themselves; Sami as self-perceived ethnicity. Sami in 1–10 items: reported Sami for at least one item and maximum for 10 items. Non-Sami: all others. Total and HDL cholesterol and triglycerides were missing in 18 subjects; systolic and diastolic blood pressure were missing in four subjects; hypertension in three. NORRISK 2 score was missing for 193 subjects. Abbreviations: HDL, high density lipoprotein; CI, confidence intervals; AMS, acute myocardial infarction; CS, cerebral stroke.

Table S2: Age-adjusted change in means and prevalences of cardiovascular risk factors between the SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014) in men (n=5749), stratified into three categories according to responses to ethnicity related questions.

Linear regression	**Non-Sami (n=2678)** β (95% Cl)	p-value	**Sami in all items (n=1244)** β (95% Cl)	p-value	**Sami in 1–10 items (n=1827)** β (95% Cl)	p-value	**Interaction‡**	p-value
Cholesterol, mmol/L	-0.6 (-0.69, -0.53)	<0.001	-0.4 (-0.50, -0.28)	<0.001	-0.5 (-0.61, -0.41)	<0.001	Sami all items	0.023
SAMINOR 1, mean †	5.99 (5.92, 6.05)		6.01 (5.90, 6.11)		6.03 (5.94, 6.12)			
SAMINOR 2, mean †	5.38 (5.30, 5.45)		5.61 (5.51, 5.72)		5.52 (5.43, 5.61)			
HDL cholesterol, mmol/L	-0.01 (-0.03, 0.011)	0.33	-0.09(-0.12, -0.06)	<0.001	-0.03 (-0.05, -0.003)	0.03	Sami all items	0.001
SAMINOR 1, mean †	1.29 (1.27, 1.31)		1.29 (1.26, 1.32)		1.29 (1.27, 1.32)			
SAMINOR 2, mean †	1.28 (1.26, 1.30)		1.20 (1.17, 1.23)		1.27 (1.24, 1.29)			
Triglycerides, mmol/L§	0.003 (-0.03, 0.04)	0.89	0.10 (0.05, 0.15)	<0.001	0.03 (-0.02, 0.07)	0.22	Sami all items	0.005
SAMINOR 1, mean †#	2.91 (2.71, 3.14)		2.82 (2.50, 3.14)		3.04 (2.77, 3.31)			
SAMINOR 2, mean †#	2.94 (2.70, 3.18)		3.56 (3.17, 3.96)		3.22 (2.93, 3.53)			
Systolic blood pressure, mm Hg	-2.6 (-3.78, -1.50)	<0.001	-3.3 (-5.15, -1.54)	<0.001	-3.7 (-5.17, -2.17)	<0.001	*	
SAMINOR 1, mean †	136.8 (135.7, 137.8)		135.7 (134.0, 137.5)		138.0 (136.7, 139.4)			
SAMINOR 2, mean †	134.1 (133.0, 135.2)		132.4 (130.7, 134.1)		134.4 (132.9, 135.8)			
Diastolic blood pressure, mm Hg	-0.3 (-0.99, 0.36)	0.36	-1.2 (-2.19, -0.28)	0.011	-1.0 (-1.88, -0.17)	0.019	*	
SAMINOR 1, mean †	79.7 (79.13, 80.34)		79.2 (78.25, 80.04)		80.3 (79.51, 81.05)			
SAMINOR 2, mean †	79.4 (78.75, 80.09)		77.9 (77.02, 78.80)		79.3 (78.45, 80.06)			
Waist circumference, cm	6.3 (5.62, 6.87)	<0.001	5.9 (5.15, 6.69)	<0.001	5.61 (4.85, 6.38)	<0.001	Sami 1–10	0.04
SAMINOR 1, mean †	94.9 (94.25, 95.46)		92.6 (91.78, 93.49)		93.1 (92.37, 93.89)			
SAMINOR 2, mean †	101.1 (100.43, 101.77)		98.6 (97.68, 99.42)		98.8 (97.96, 99.53)			
10-year risk of AMI and CS, %§	-0.18 (-0.21, -0.15)	<0.001	-0.15 (-0.20, -0.11)	<0.001	-0.19 (-0.23, -0.15)	<0.001	*	
SAMINOR 1, mean †#	8.65 (8.41, 8.89)		8.54 (8.16, 8.92)		9.05 (8.72, 9.39)			
SAMINOR 2, mean †#	7.21 (6.99, 7.43)		7.34 (7.02, 7.66)		7.48 (7.19, 7.76)			
Logistic regression	**Odds ratios (95% Cl)**	**p-value**	**Odds ratios (95% Cl)**	**p-value**	**Odds ratio (95% Cl)**	**p-value**		**p-value**
Anti-hypertensive drugs	1.20 (1.03, 1.40)	0.022	1.15 (0.91, 1.46)	0.24	1.11 (0.91, 1.35)	0.30	*	
SAMINOR 1, prevalences % †	21.40 (19.25, 23.55)		18.43 (15.37, 21.49)		22.71 (19.99, 25.44)			
SAMINOR 2, prevalences % †	24.58 (22.00, 27.16)		20.66 (17.29, 24.04)		24.59 (21.57, 27.61)			
Hypertension	0.96 (0.83, 1.10)	0.51	0.87 (0.71, 1.07)	0.19	0.85 (0.72, 1.02)	0.07	*	
SAMINOR 1, prevalences % †	50.37 (47.64, 53.09)		45.39 (41.26, 49.51)		55.21 (51.87, 58.56)			
SAMINOR 2, prevalences % †	49.20 (46.26, 52.14)		42.02 (37.90, 46.15)		51.23 (47.75, 54.71)			
Current smoking	0.54 (0.46, 0.63)	<0.001	0.58 (0.47, 0.71)	<0.001	0.53 (0.44, 0.63)	<0.001	*	
SAMINOR 1, prevalences % †	29.79 (27.44, 32.14)		31.65 (28.05, 35.25)		31.63 (28.70, 34.57)			
SAMINOR 2, prevalences % †	18.49 (16.38, 20.60)		21.02 (17.91, 24.12)		19.54 (16.98, 22.10)			

β–coefficients are estimated by linear generalised estimating equation regression models and adjusted for age and age^2. Odds ratios are estimated by logistic generalised estimating equation regression models and adjusted for age. †Predicted means/prevalences at age 58.2 years. §Outcome variables are log-transformed. # Predicted geometric means at age 58.2. ‡ Testing for interaction between survey and ethnicity using non-Sami in SAMIMNOR 1 as reference. If p-value for interaction <0.05, we have specified which group differs from non-Sami in SAMINOR 2. *p-value for interaction between survey and ethnicity >0.05, please refer to the overall estimation given in Table 4. Sami in all items (11 in total): reported use of Sami language in grandparents, parents and themselves; Sami ethnic background for parents and themselves; Sami as self-perceived ethnicity. Sami in 1–10 items: reported Sami for at least one item and maximum for 10 items. Non-Sami: all others. Total and HDL cholesterol were missing in 12 subjects; triglycerides were missing in 13 subjects, systolic and diastolic blood pressure and hypertension was missing in one subject. NORRISK 2 score was missing for 173 subjects. Abbreviations: HDL, high density lipoprotein; CI, confidence intervals; AMS, acute myocardial infarction; CS, cerebral stroke.

Table S3: Age- and region adjusted predicted changes in beta coefficients and odds ratios of cardiovascular risk factors between SAMINOR 1 (2002–2004) and the SAMINOR 2 (2012–2017) in women and men, after testing for interaction between survey and ethnicity.

	Women (n=6624)		**Men** (n=5749)	
Linear regression	**β (95% Cl)**	**p-value**	**β (95% Cl)**	**p-value**
Total cholesterol, mmol/L	-0.50 (-0.54, -0.45)	<0.001	*	
	-		Non-Sami: -0.59 (-0.65, -0.52)	<0.001
	-		Sami: -0.43 (-0.51, -0.34)	<0.001
HDL cholesterol, mmol/L	*		*	
	Non-Sami: 0.05 (0.03, 0.07)	<0.001	Non-Sami: -0.01 (-0.03, 0.01)	0.31
	Sami: -0.01 (-0.04, 0.01)	0.24	Sami: -0.06 (-0.08, -0.03)	<0.001
Triglycerides, mmol/L †	-0.001 (-0.02, 0.02)	0.96	*	
	-		Non-Sami: -0.0002 (-0.03, 0.003)	0.99
	-		Sami: 0.09 (0.05, 0.12)	<0.001
Systolic blood pressure, mm Hg	-3.5 (-4.25, -2.77)	<0.001	-3.0 (-3.81, -2.21)	<0.001
Diastolic blood pressure, mm Hg	-1.0 (-1.37, -0.55)	<0.001	-0.8 (-1.22, -0.30)	0.001
Waist circumference, cm	6.6 (6.20, 7.03)	<0.001	5.9 (5.52, 6.33)	<0.001
10-year risk of AMI and CS, %†	*		-0.18 (-0.20, -0.16)	<0.001
	Non-Sami:-0.19 (-0.22, -0.17)	<0.001	-	
	Sami: -0.12 (-0.16, -0.09)	<0.001	-	
Logistic regression	**Odds ratio (95% Cl)**	**p-value**	**Odds ratio (95% Cl)**	**p-value**
Use of anti-hypertensive drugs	0.98 (0.89, 1.08)	0.68	1.20 (1.08, 1.33)	0.001
Hypertensive	0.79 (0.72, 0.87)	<0.001	0.93 (0.84, 1.02)	0.13
Current smoking	0.64 (0.59, 0.70)	<0.001	0.53 (0.48, 0.59)	<0.001

β–coefficients are estimated by linear generalised estimating equation regression models and adjusted for age, age^2 and region. Odds ratios are estimates by logistic generalised estimating equation regression models and adjusted for age and regions. Adjusting for three regions including the following municipalities: 1) Kautokeino and Karasjok. 2) Nesseby, Tana and Porsanger. 3) Kåfjord, Lyngen, Storfjord, Skånland and Evenes. When p-value for interaction between survey and ethnicity is >0.05, overall β –coefficients and odds ratios adjusted for region are reported, otherwise (indicated by *), ethnic specific β–coefficients are reported. †Outcome variables are log transformed.

STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of *cross-sectional studies*

Section/Topic	Item #	Recommendation	Reported on page #
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	1
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2–3
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	5
Objectives	3	State specific objectives, including any prespecified hypotheses	5–6
Methods			
Study design	4	Present key elements of study design early in the paper	6
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	6
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants	6–7
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	10–11, 15, 17
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	7–10
Bias	9	Describe any efforts to address potential sources of bias	N/A
Study size	10	Explain how the study size was arrived at	6–7
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	9
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	10–11
		(*b*) Describe any methods used to examine subgroups and interactions	10–11
		(*c*) Explain how missing data were addressed	7, 15, 17
		(*d*) If applicable, describe analytical methods taking account of sampling strategy	N/A
		(*e*) Describe any sensitivity analyses	11
Results			

Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	6–7, 11
		(b) Give reasons for non-participation at each stage	6–7
		(c) Consider use of a flow diagram	N/A
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	13, 14
		(b) Indicate number of participants with missing data for each variable of interest	15, 17
Outcome data	15*	Report numbers of outcome events or summary measures	15, 17
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	15, 17
		(*b*) Report category boundaries when continuous variables were categorized	15, 17
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	12, 16
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	16, supplementary
Discussion			
Key results	18	Summarise key results with reference to study objectives	18
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	18
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	18–22
Generalisability	21	Discuss the generalisability (external validity) of the study results	18–19
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	23

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

BMJ Open

Changes in conventional cardiovascular risk factors and the estimated 10-year risk of acute myocardial infarction or cerebral stroke in Sami and non-Sami populations in two population-based cross sectional surveys—the SAMINOR Study

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CHANGES IN CONVENTIONAL CARDIOVASCULAR RISK FACTORS AND THE ESTIMATED 10-YEAR RISK OF ACUTE MYOCARDIAL INFARCTION OR CEREBRAL STROKE IN SAMI AND NON-SAMI POPULATIONS IN TWO POPULATION-BASED CROSS-SECTIONAL SURVEYS —THE SAMINOR STUDY

Susanna Ragnhild A. Siri[1], Bent Martin Eliassen[2], Bjarne K. Jacobsen[1,3], Marita Melhus[1], Ann Ragnhild Broderstad[1], Vilde Lehne Michalsen[1], Tonje Braaten[3]

[1]Centre for Sami Health Research, Department of Community Medicine, Faculty of Health Sciences, UiT The Arctic University of Norway, Tromsø, Norway. [2] Faculty of Nursing and Health Sciences, Nord University, Bodø, Norway. [3]Department of Community Medicine, Faculty of Health Sciences, UiT The Arctic University of Norway, Tromsø, Norway.

Corresponding author: Susanna Ragnhild A. Siri

Postal address: Centre for Sami Health Research, Department of Community Medicine, UiT The Arctic University of Norway. Postboks 6050, Langnes. 9037 Tromsø

E-mail: susanna.r.siri@uit.no

Telephone number: 0047-77646977

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Abstract

Objective: To describe changes in cardiovascular risk factors and in the estimated 10-year risk of acute myocardial infarction (AMI) or cerebral stroke (CS) between SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014), and explore if these changes differed between Sami and non-Sami.

Design: Two cross-sectional surveys.

Setting: Inhabitants of rural Northern Norway.

Participants: Participants were aged 40–79 years and participated in SAMINOR 1 (n=6417) and/or SAMINOR 2 (n=5956).

Primary outcome measures: Generalised estimating equation regressions with an interaction term were used to estimate and compare changes in cardiovascular risk factors and 10-year risk of AMI or CS between the two surveys and by ethnicity.

Results: Mean cholesterol declined by 0.50, 0.43, and 0.60 mmol/L in women, Sami men, and non-Sami men, respectively (all p<0.001). Sami men had a small decline in mean high-density lipoprotein (HDL) cholesterol and an increase in mean triglycerides (both p<0.001), whereas non-Sami showed no change in these variables. Non-Sami women had an increase in mean HDL cholesterol (p<0.001) whereas Sami women had no change. Triglycerides did not change in non-Sami and Sami women. Systolic and diastolic blood pressure declined by 3.6 and 1.0 mmHg in women, and 3.1 and 0.7 in men, respectively (all p<0.01). Mean waist circumference increased by 6.7 and 5.9 cm in women and men, respectively (both p<0.001). The odds of being a smoker declined by 35% in women and 46% in men (both p<0.001). Estimated 10-year risk of AMI or CS decreased in all strata of sex and ethnicity (p<0.001), however, Sami women had a smaller decline than non-Sami did.

Conclusions: Independent of ethnicity, there was a decline in mean cholesterol, blood pressure, smoking, hypertension (women only), and 10-year risk of AMI or CS, but waist

circumference increased. Relatively minor ethnic differences were found in changes of cardiovascular risk factors.

ARTICLE SUMMARY

Strengths and limitations:

- We used generalised estimating equation regression to account for overlapping samples.
- We used self-reported measures to categorise participants into ethnic groups, including questions on self-perceived ethnicity, ethnic background, and language use.
- Due to lack of ethnic identifiers in national registries, we do not know if participation differs by ethnicity.
- We have an acceptable participation rate in both surveys.
- We lack information about the use of lipid-lowering drugs.

INTRODUCTION

Since the 1970s, a favourable decline in systolic blood pressure,[1-4] total cholesterol[5-7] and smoking[8] has been reported for the adult population across different regions of Western Europe. This decline is probably due to changes in lifestyle and diet,[7, 9, 10] in addition to use of medication.[1, 5] In Norway, this decline has coincided with a decrease in cardiovascular mortality and an increased prevalence of obesity and a sedentary lifestyle.[11]

The Sami is an Indigenous people living in Sápmi, i.e. the northern parts of Norway, Sweden, Finland, and the Kola Peninsula in the Russian Federation. There are no official population records on the Sami population, but data from the 1970 national census roughly estimated that there were 40,000 Sami in Norway,[12] whereas 55,000 is the population number that the Sami Parliament uses when considering subsidy schemes for business development.[13] In 2017, approximately 17,000 Sami adults were enrolled in the electoral register to the Sami Parliament in Norway, which gives them the right to vote and be elected.[14] The Sami people have unique cultures and languages, but these have partly vanished or at least declined in practice, due to structural assimilation that occurred from 1850–1960.[15] The Norwegian part of Sápmi is also inhabited by Norwegians and Kvens, the latter of whom are descendants of Finnish-speaking people that came from Sweden and Finland to Northern Norway in the 1700s and 1800s.[16]

Surveys from Norway have concluded that there are no or only minor differences in cardiovascular risk factors and morbidity between the Sami and non-Sami in rural regions.[17-21] However, knowledge is lacking on changes in conventional cardiovascular risk factors. Thus, this study aimed to describe changes in cardiovascular risk factors and in the estimated 10-year risk of acute myocardial infarction (AMI) or cerebral stroke (CS) between

SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014), and explore if these changes differed between Sami and non-Sami.

METHODS

We used data from two cross-sectional surveys of the Population-based Study on Health and Living Conditions in Regions with Sami and Norwegian Populations (The SAMINOR Study): the SAMINOR 1 Survey carried out in 2003–2004 (SAMINOR 1) and the SAMINOR 2 Clinical Survey carried out in 2012–2014 (SAMINOR 2). SAMINOR 1 was a collaboration between the Centre for Sami Health Research at UiT The Arctic University of Norway and the Norwegian National Institute of Public Health,[22] whereas SAMINOR 2 was performed by the former only[23]. Participants were invited from 10 municipalities (Figure 1) that, according to the population census from 1970[12], had high proportions of Sami inhabitants. Invitations were mailed to all who were aged 40–79 and were registered as inhabitants in the 10 municipalities by the National Registry. In total, 11,518 and 12,455 received an invitation to SAMINOR 1 and SAMINOR 2, respectively. Participation was voluntarily and clinical examinations in each municipality were conducted within a period of one to seven weeks, depending on the population size. Our analyses were restricted to those who attended clinical examinations, gave blood samples, and answered the self-administered questionnaires.

Participant and public involvement

Participants that had pathological findings from the clinical examination, were recommended to contact their primary physician. In emergency situations, participants were sent directly to the local health centre or the nearest hospital.

Before and after the surveys, the Centre for Sami Health Research had consultations with the Sami Parliament, Sami researchers, and health workers in Sami core areas to identify the needs of the Sami community. Results from the surveys were reported to decision makers at the municipal and regional levels, and to the Sami Parliament and national health authorities. The population was informed through popular science forums, meetings, and lectures.

Study sample

There were 6550 (56.9%) and 6004 (48.2%) individuals that attended the clinical examinations in SAMINOR 1 and 2, respectively. If information on ethnicity was lacking in one of the surveys, ethnicity information given in the other survey was used, as Sami ethnicity is found to be stable.[24] This strategy was valuable for the SAMINOR 1 sample, as ethnicity information was lacking for some participants due to the study design.[22] In SAMINOR 1, we categorised 69 out of 201 by using ethnicity information from SAMINOR 2: 7 non-Sami and 62 Sami. In SAMINOR 2, 96 had missing data on ethnicity and we categorised 58: 37 non-Sami and 21 Sami. Furthermore, we excluded those that did not hand in the main questionnaires (SAMINOR 1: n=1; SAMINOR 2: n=10). This left us with a final sample of 6417 and 5956 from SAMINOR 1 and 2, respectively, wherein 3249 participated in both surveys.

The Norwegian Data Inspectorate and the Regional Committee for Medical and Health Research Ethics for region North (REC North) have approved The SAMINOR Study. The REC North (2015/2204–11) and the SAMINOR Project Board have also approved this study. All participants included in this study gave written informed consent and consented to linkage between the surveys.

Information from questionnaires

Participants were categorised into ethnic groups based on information from the following 11 questions, which were identical in the two surveys: "What language(s) do/did you, your parents, and your grandparents use at home?"; "What is your, your father's, and your mother's ethnic background?"; "What [ethnicity] do you consider yourself to be?" The response options were "Norwegian", "Sami", "Kven" and "Other" and multiple answers were allowed. Participants were defined as Sami if they 1) considered themselves to be Sami, or reported a Sami ethnic background for themselves, and 2) spoke a Sami language themselves or had at least one parent or grandparent that used it at home. All others were categorised as non-Sami. Sensitivity analyses were performed, in which different ethnic categorisations were used.

Smoking status was determined by the following questions, in SAMINOR 1: "Are you currently, or were you previously a daily smoker?" (Yes, currently/Yes, previously/Never); in SAMINOR 2: "Have you ever smoked daily?" (Yes/No), and "Are you currently a daily smoker?" (Yes/No). Previous and never smokers were categorised as non-smokers.

Use of anti-hypertensive drugs was determined by the following question: "Do you take medication for high blood pressure?" (Currently/Previously, but not now/Never used). Previous use, never-use and missing values were merged into non-use.

In both surveys, participates reported if they ever have had myocardial infarctions and age at first time. Positive responses to the former, or age reported for first time, were considered as having had a myocardial infarction.

Leisure time physical activity was measured in SAMINOR 1 by the "Saltin-Grimby" questionnaire.[25] Overall physical activity at current age was measured in SAMINOR 2 by a scale ranging from 1–10; an instrument validated in middle aged women living in Tromsø, Norway.[26]

Alcohol consumption was measured in SAMINOR 1 by asking: "How often during the last year have you consumed alcohol?" (Never/Not during the last year/A few times during the last year/1 time per month/2–3 times per month/1 time per week/2–3 times per week/4–7 times per week). To approximate the question in SAMINOR 2, we created two categories: never consumed alcohol and consumers of alcohol. In SAMINOR 2, alcohol consumption was asked as follows: "Do you practice total alcohol abstinence? " (Yes/no).

Education was measured similarly in both surveys by years of education. We categorised the item to match roughly primary and lower secondary school, upper secondary school and higher education: ≤ 9 years, 10–12 years and ≥13 years.

Clinical examination

Trained staff conducted the clinical examination. Waist circumference was measured at the umbilicus when the participant was standing. Blood pressure was measured with digital oscillometric devices (SAMINOR 1: DINAMAP-R, Criticon, Tampa, Florida, USA; SAMINOR 2: CARESCAPE™V100 monitor, GE Healthcare, Milwaukee, Wisconsin, USA), with the participant in a seated position. Following a 2-minute rest, three recordings were made at 1-minute intervals, and the average of the last two measurements was used in the analysis. Participants were considered to have hypertension if their systolic blood pressure

was ≥140 mmHg, or their diastolic blood pressure was ≥90 mmHg, or if they reported using anti-hypertensive drugs.

In both surveys, non-fasting blood samples were collected. The blood samples were left to coagulate for a minimum of 30 minutes, after which they were centrifuged and serum was separated within 2 hours. In SAMINOR 1, serum was sent by overnight post and analysed consecutively for lipids (total cholesterol, high-density lipoprotein (HDL) cholesterol and triglycerides) with an enzymatic method (Hitachi 917 auto analyser, Roche Diagnostics, Switzerland) at Ullevål University Hospital, Oslo, Norway. In SAMINOR 2, serum samples were kept at -20° C before they were sent to the biobank at UiT The Arctic University of Norway, for further storage at -70° C. The samples were analysed in batches during autumn 2014 at the University Hospital of North Norway, Tromsø, Norway. Lipids were measured with an enzymatic colorimetric method (Cobas 8000B, Roche Diagnostics GmbH, Mannheim, Germany).

The 10-year absolute risk of fatal or non-fatal AMI or CS was estimated by the NORRISK 2 model[27] and determined separately in women and men based on age, total cholesterol, HDL cholesterol, smoking status, systolic blood pressure, and use of anti-hypertensive drugs.

Statistical analyses

Statistical analyses were done using STATA version 15.0 (StataCorp, College Station, Texas, USA). Sample characteristics were given by sex for Sami and non-Sami in SAMINOR 1 and 2: means (standard deviations) of continuous variables and proportions (numbers) of categorical variables. In order to account for the partly overlapping samples, changes in population average means and prevalences of risk factors between SAMINOR 1 and

SAMINOR 2 were estimated by sex- and ethnicity-specific linear or logistic generalised estimating equation regression models. Assumptions of normality and homoscedasticity were assessed by a visual inspection of residual plots. Changes in triglycerides and in the estimated 10-year risk of AMI or CS were log-transformed due to skewed distributions. All regression models were adjusted for age, and linear models were additionally adjusted for age squared. We assessed if changes in outcomes differed by ethnicity by including an interaction term between survey and ethnicity in sex-specific models. If the p-value for interaction was >0.05, the interaction term was excluded from the model and an overall sex-specific mean/prevalence was reported. In the opposite case, ethnicity-specific changes were reported. Marginal means/prevalences were estimated at age 57.5 years in women and at 58.2 years in men, i.e. the sex-specific mean ages in the overall sample. Two-way graphs illustrate how cardiovascular risk factors varied by age, ethnicity and survey (Supplementary Figures S1 and S2). Potential heterogeneity by age in the overall models was assessed by comparing two strata divided at sex-specific mean age. The terms for interaction between ethnicity and survey remained non-significant across age strata for both sexes. Hence, we concluded that age did not modify the overall estimates of change in cardiovascular risk factors. We considered a two-sided $p<0.05$ to be significant.

Sensitivity analyses were done with same sex-stratified generalised estimating equation models by

1. Dividing the study sample into three groups: 1) those who reported "Sami" for all 11 questions, 2) who reported Sami in one to 10 questions, and 3) those who did not report Sami on any of the questions (non-Sami) (Supplementary Tables S1 and S2).

2. Using the original ethnic categorisation, we adjusting for geographical regions: 1) Kautokeino and Karasjok, 2) Nesseby, Tana, and Porsanger, 3) Kåfjord, Lyngen, Storfjord, Skånland, and Evenes (Supplementary Table S3).

RESULTS

Of the total sample, 53.5% were women. In women and men, 37.8% and 39.5% were Sami, respectively. The mean age was higher in both sexes in SAMINOR 2 than in SAMINOR 1. In both surveys, Sami women (Table 1) and men (Table 2) were less physically active, and Sami women reported more often to be non-consumers or abstainers of alcohol.

Both non-Sami and Sami women had a decline in total cholesterol between SAMINOR 1 and SAMINOR 2 ($p<0.001$, Table 3). The overall change in total cholesterol in women was -0.50 mmol/l. Sami women had lower HDL cholesterol and higher triglycerides than non-Sami in both surveys (Table 3). The change in triglycerides did not differ by ethnicity, but the change in HDL cholesterol did, with non-Sami showing a minor increase, and Sami showing no change.

In both surveys, Sami women had somewhat lower blood pressure than non-Sami did (Table 3). The overall decline in systolic and diastolic blood pressure was 3.6 and 1.0 mmHg (both $p<0.001$), respectively; these changes did not differ by ethnicity. Roughly, 23% of women reported use of anti-hypertensive drugs, and this did not change over time. The prevalence of hypertension declined in a similar magnitude in Sami and non-Sami women; by 6.2 percentage points ($p<0.001$) (Table 3).

Non-Sami and Sami women had a similar increase of 6.7 cm in mean waist circumference. The prevalence of smoking in non-Sami and Sami women declined by 10.0 and 5.6 percentage points, respectively (both $p<0.001$); this change did not differ by ethnicity. Overall, the odds of current smoking declined by 35% (Table 3).

The estimated 10-year risk of AMI or CS declined between SAMINOR 1 and SAMINOR 2 in both Sami and non-Sami women (both $p<0.001$, Table 3), but more so in non-Sami.

Table 1. Unadjusted means (SD) and proportions (%) of sample characteristics in women aged 40–79 years participating in SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014).

	SAMINOR 1 (n=3390)		**SAMINOR 2** (n=3234)	
Ethnicity	Non-Sami	Sami	Non-Sami	Sami
Proportions, % (n)	64.7 (2193)	35.3 (1197)	59.7 (1929)	40.4 (1305)
Age, mean (SD)	56.5 (10.1)	55.5 (10.2)	59.1 (10.3)	58.6 (10.4)
Self-reported myocardial infarction , % (n)§†	2.6 (58)	1.9 (23)	3.2 (62)	1.8 (23)
Physical activity using "Saltin-Grimby" questionnaire†			‡	‡
Reading, watching TV or other sedentary activity, % (n)	21.3 (415)	27.7 (297)	-	-
Walking, bicycling or moving around in other ways at least 4 hour/week, % (n)	68.1 (1330)	61.9 (664)	-	-
Participation in recreational sports, heavy garden work etc. Duration at least 4 hours/week, % (n)	10.3 (200)	9.6 (103)	-	-
Participation in hard training or athletic competitions regularly and several times/week, % (n)	0.4 (7)	0.8 (8)	-	-
Level of physical activity on a scale from 1–10, mean (SD)†	‡	‡	5.6 (2.08)	5.2 (2.16)
Never consumed alcohol, % (n)†	14.8 (309)	24.5 (279)	‡	‡
Alcohol abstinence, yes % (n)†	‡	‡	18.4 (341)	27.0 (337)
Years of education, mean (SD)†	10.9 (3.8)	10.7 (4.6)	12.2 (4.0)	12.4 (4.5)
0–9 years education, % (n)	41.7 (864)	43.6 (497)	28.0 (530)	30.3 (385)
10–12 years education, % (n)	30.2 (626)	23.2 (265)	29.9 (565)	23.2 (295)
≥13 years of education, % (n)	28.1 (584)	33.1 (378)	42.1 (797)	46.5 (592)
Region 1: Kautokeino and Karasjok, % (n)	3.8 (84)	44.8 (537)	5.2 (101)	48.4 (631)
Region 2: Nesseby, Tana and Porsanger, % (n)	27.5 (603)	38.3 (458)	30.1 (580)	36.6 (478)
Region 3: Kåfjord, Lyngen, Storfjord, Skånland and Evenes, % (n)	68.7 (1506)	16.9 (202)	64.7 (1248)	15.0 (196)

†Based on a lower number due to missing values. §Measured differently in SAMINOR 1 and 2. ‡Question not posed.

Table 2. Unadjusted means (SD) and proportions (%) of sample characteristics in men aged 40–79 years participating in SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014).

	SAMINOR 1 (n=3027)		**SAMINOR 2** (n=2722)	
Ethnicity	Non-Sami	Sami	Non-Sami	Sami
Proportions, % (n)	62.1 (1881)	37.9 (1146)	58.7 (1597)	41.3 (1125)
Age, mean (SD)	56.5 (9.8)	56.3 (10.1)	60.4 (10.2)	59.8 (10.3)
Self-reported myocardial infarction, % (n)§ †	6.9 (130)	6.5 (75)	8.8 (140)	8.1 (91)
Physical activity using "Saltin-Grimby" questionnaire†			‡	‡
Reading, watching TV or other sedentary activity, % (n)	20.2 (351)	24.3 (254)	-	-
Walking, bicycling or moving around in other ways at least 4 hour/week, % (n)	59.5 (1034)	53.0 (555)	-	-
Participation in recreational sports, heavy garden work etc. Duration at least 4 hours/week, % (n)	18.6 (324)	20.2 (212)	-	-
Participation in hard training or athletic competitions regularly and several times/week, % (n)	1.7 (29)	2.5 (26)	-	-
Level of physical activity on a scale from 1–10, mean (SD)†	‡	‡	5.2 (2.01)	5.12 (2.16)
Never consumed alcohol, % (n)†	5.4 (99)	4.5 (50)	‡	‡
Alcohol abstinence, yes % (n)†	‡	‡	10.6 (164)	13.4 (150)
Years of education, mean (SD)†	10.9 (3.7)	10.2 (4.1)	11.8 (3.6)	11.4 (3.8)
0–9 years education, % (n)	39.6 (719)	47.3 (519)	29.7 (467)	36.3 (400)
10–12 years education, % (n)	32.7 (594)	27.6 (303)	33.1 (520)	30.3 (333)
≥13 years of education, % (n)	27.7 (502)	25.1 (276)	37.2 (584)	33.4 (368)
Region 1: Kautokeino and Karasjok, % (n)	3.3 (63)	37.9 (434)	4.6 (73)	42.4 (477)
Region 2: Nesseby, Tana and Porsanger, % (n)	28.9 (543)	39.0 (447)	32.9 (525)	37.2 (419)
Region 3: Kåfjord, Lyngen, Storfjord, Skånland and Evenes, % (n)	67.8 (1275)	23.1 (265)	62.6 (999)	20.4 (229)

†Based on a lower number due to missing values. §Measured differently in SAMINOR 1 and 2. ‡Question not posed.

Table 3. Age-adjusted predicted changes in means and prevalences of cardiovascular risk factors between the SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014) in non-Sami and Sami women (n=6624).

	Non-Sami (n=4122)		Sami (n=2502)		Interaction ‡	Overall (n=6624)	
Linear regression	**β (95% CI)**	**p-value**	**β (95% CI)**	**p-value**	**p-value**	**β (95% CI)**	**p-value**
Total cholesterol, mmol/L	-0.51 (-0.57, -0.45)	<0.001	-0.47 (-0.55, -0.39)	<0.001	0.86	-0.50 (-0.54, -0.45)	<0.001
SAMINOR 1, mean†	6.24 (6.19, 6.30)		6.24 (6.16, 6.32)			6.25 (6.20, 6.29)	
SAMINOR 2, mean†	5.73 (5.67, 5.79)		5.77 (5.70, 5.85)			5.75 (5.70, 5.79)	
HDL cholesterol, mmol/L	0.05 (0.03, 0.07)	<0.001	-0.02 (-0.04, 0.01)	0.14	<0.001	*	
SAMINOR 1, mean†	1.51 (1.49, 1.53)		1.47 (1.45, 1.49)				
SAMINOR 2, mean†	1.56 (1.54, 1.58)		1.45 (1.43, 1.48)				
Triglycerides, mmol/L§	-0.02 (-0.05, 0.004)	0.10	0.03 (0.004, 0.07)	0.03	0.07	0.002 (-0.02, 0.02)	0.87
SAMINOR 1, mean†#	1.43 (1.40, 1.47)		1.48 (1.43, 1.53)			1.45 (1.43, 1.48)	
SAMINOR 2, mean†#	1.40 (1.37, 1.44)		1.53 (1.49, 1.58)			1.46 (1.43, 1.48)	
Systolic blood pressure, mm Hg	-3.9 (-4.86, -2.98)	<0.001	-3.0 (-4.23, -1.81)	<0.001	0.10	-3.6 (-4.36, -2.88)	<0.001
SAMINOR 1, mean†	134.7 (133.8, 135.6)		132.4 (131.2, 133.7)			133.9 (133.2, 134.6)	
SAMINOR 2, mean†	130.8 (129.9, 131.7)		129.4 (128.3, 130.6)			130.3 (129.6, 131.0)	
Diastolic blood pressure, mm Hg	-0.9 (-1.38, -0.33)	0.002	-1.1 (-1.77, -0.46)	0.001	0.39	-1.0 (-1.39, -0.57)	<0.001
SAMINOR 1, mean†	73.9 (73.4, 74.4)		73.4 (72.7, 74.1)			73.7 (73.3, 74.1)	
SAMINOR 2, mean†	73.0 (72.5, 73.6)		72.3 (71.6, 72.9)			72.7 (72.3, 73.2)	
Waist circumference, cm	7.0 (6.41, 7.49)	<0.001	6.1 (5.44, 6.76)	<0.001	0.26	6.7 (6.24, 7.07)	<0.001
SAMINOR 1, mean†	86.4 (85.83, 87.00)		88.0 (87.29, 88.78)			87.0 (86.55, 87.45)	
SAMINOR 2, mean†	93.3 (92.75, 93.94)		94.1 (93.43, 94.85)			93.7 (93.20, 94.11)	
10-year risk of AMI and CS, %§	-0.19 (-0.22, -0.17)	<0.001	-0.13 (-0.16, -0.09)	<0.001	0.011	*	
SAMINOR 1, mean†#	4.16 (4.05, 4.26)		3.91 (3.77, 4.05)				
SAMINOR 2, mean†#	3.43 (3.33, 3.52)		3.44 (3.32, 3.56)				
Logistic regression	**Odds ratio (95% CI)**	**p-value**	**Odds ratio (95% CI)**	**p-value**	**p-value**	**Odds ratio (95% CI)**	**p-value**
Anti-hypertensive treatment	0.96 (0.85, 1.09)	0.51	0.98 (0.83, 1.16)	0.82	0.77	0.96 (0.87, 1.07)	0.47
SAMINOR 1, prevalence %†	24.0 (22.10, 25.80)		22.9 (20.34, 25.36)			23.6 (22.11, 25.10)	
SAMINOR 2, prevalence %†	23.2 (21.17, 25.24)		22.5 (20.04, 24.98)			23.0 (21.38, 24.53)	
Hypertension	0.77 (0.68, 0.86)	<0.001	0.79 (0.68, 0.93)	0.003	0.56	0.77 (0.70, 0.85)	<0.001
SAMINOR 1, prevalence %†	47.4 (45.12, 49.72)		44.5 (41.37, 47.68)			46.3 (44.47, 48.20)	
SAMINOR 2, prevalence %†	40.8 (38.39, 43.25)		38.9 (35.90, 41.79)			40.1 (38.18, 41.94)	
Current smokers	0.59 (0.53, 0.66)	<0.001	0.74 (0.64, 0.85)	<0.001	0.10	0.65 (0.59, 0.71)	<0.001
SAMINOR 1, prevalence %†	31.0 (29.07, 32.90)		27.9 (25.28, 30.45)			29.8 (28.26, 31.34)	
SAMINOR 2, prevalence %†	21.0 (19.26, 22.76)		22.3 (20.07, 24.46)			21.6 (20.21, 22.94)	

β coefficients are estimated by linear generalised estimating equation regression models and adjusted for age and age^2. Odds ratios are estimated by logistic generalised estimating equation regression models and adjusted for age. †Predicted means/prevalences at age 57.5 years, which is the mean age for women in the overall sample. §Outcome variables are log-transformed. #Predicted geometric means at age 57.5 year. ‡ Test of interaction between survey and ethnicity in overall model. If p-value for interaction >0.05, interaction term was excluded from the overall model. *p-value for interaction <0.05, only ethnicity-specific estimations are reported. Number of missing values: total and HDL cholesterol and triglycerides were missing in 18 subjects; systolic and diastolic blood pressure were missing in four subjects; hypertension in three. NORRISK 2 score was missing for 193 subjects. Excluding missing values did not change the results. Abbreviations: CI, confidence interval; AMS, acute myocardial infarction; CS, cerebral stroke.

Between SAMINOR 1 and SAMINOR 2, total cholesterol declined more in non-Sami than in Sami men (0.60 vs. 0.43 mmol/l; both p<0.001, Table 4), and this change varied by ethnicity. Between the surveys, Sami men had a slight decline in HDL cholesterol (p<0.001) and a slight increase in triglycerides (p<0.001); whereas non-Sami men had no changes, hence, changes in HDL cholesterol and triglyceride differed for Sami and non-Sami (Table 4).

In men, the decline in systolic and diastolic blood pressure did not differ by ethnicity (Table 4). The overall decline in systolic and diastolic blood pressure in men were 3.1 and 0.7 mmHg (both p<0.05), respectively. Overall, we found an increase in the prevalence of anti-hypertensive drug use, from 21.1% to 23.9% in men, which did not differ by ethnicity. The prevalence of hypertension remained similar in SAMINOR 1 and SAMINOR 2, with roughly half of men being considered hypertensive (Table 4).

Waist circumference increased similarly in Sami and non-Sami men, with an overall increase of 5.9 cm (p<0.001). The prevalence of smoking declined similarly in non-Sami and Sami men, by 12.3 and 10.0 percentage points (both p<0.001), respectively. The overall decline in the odds of being a smoker was 46%.

The estimated 10-year risk of AMI or CS declined in non-Sami and Sami men (both p<0.001, Table 4), but not differently in the two ethnic groups.

Sensitivity analyses

Overall, the sensitivity analyses were consistent with the main findings when using a different ethnic categorisation (Supplementary Tables S1 and S2) and when adjusting for region (Supplementary Table S3).

Table 4. Age-adjusted predicted changes in means and in prevalences of cardiovascular risk factors between the SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014) in non-Sami and Sami men (n=5749).

	Non-Sami (n=3478)		Sami (n=2271)		Interaction ‡	Overall (n=5749)	
Linear regression	**β (95% CI)**	**p-value**	**β (95% CI)**	**p-value**	**p-value**	**β (95% CI)**	**p-value**
Total cholesterol, mmol/L	-0.60 (-0.66, -0.53)	<0.001	-0.43 (-0.51, -0.35)	<0.001	0.03	*	
SAMINOR 1, mean†	6.00 (5.95, 6.07)		6.00 (5.92, 6.08)				
SAMINOR 2, mean†	5.41 (5.35, 5.48)		5.58 (5.50, 5.65)				
HDL cholesterol, mmol/L	-0.01 (-0.03, 0.01)	0.18	-0.06 (-0.08, -0.04)	<0.001	0.005	*	
SAMINOR 1, mean†	1.30 (1.28, 1.31)		1.28 (1.26, 1.31)				
SAMINOR 2, mean†	1.28 (1.26, 1.30)		1.22 (1.20, 1.25)				
Triglycerides, mmol/L§	0.001 (-0.03, 0.03)	0.96	0.09 (0.05, 0.13)	<0.001	0.001	*	
SAMINOR 1, mean†#	1.61 (1.56, 1.65)		1.58 (1.53, 1.64)				
SAMINOR 2, mean†#	1.61 (1.56, 1.66)		1.73 (1.67, 1.79)				
Systolic blood pressure, mm Hg	-3.2 (-4.19, -2.18)	<0.001	-2.8 (-4.11, -1.44)	<0.001	0.38	-3.1 (-3.87, -2.27)	<0.001
SAMINOR 1 mean†	137.0 (136.1, 137.9)		136.8 (135.5, 138.1)			136.9 (136.2, 137.7)	
SAMINOR 2, mean†	133.8 (132.8, 134.8)		134.0 (132.7, 135.3)			133.9 (133.1, 134.6)	
Diastolic blood pressure, mm Hg	-0.5 (-1.08, 0.11)	0.11	-1.1 (-1.82, -0.33)	0.004	0.08	-0.7 (-1.20, -0.28)	0.002
SAMINOR 1, mean†	79.7 (79.2, 80.2)		79.9 (79.2, 80.6)			79.8 (79.4, 80.2)	
SAMINOR 2, mean†	79.2 (78.6, 79.8)		78.9 (78.2, 79.5)			79.1 (78.6, 79.5)	
Waist circumference, cm	6.0 (5.45, 6.56)	<0.001	5.9 (5.22, 6.48)	<0.001	0.37	5.9 (5.50, 6.31)	<0.001
SAMINOR 1, mean†	94.4 (93.82, 94.90)		93.1 (92.45, 93.79)			93.9 (94.48, 94.32)	
SAMINOR 2, mean†	100.4 (99.78, 100.95)		99.0 (98.29, 99.64)			99.8 (99.36, 100.24)	
10-year risk of AMI and CS, %§	-0.19 (-0.22, -0.17)	<0.001	-0.16 (-0.19, -0.12)	<0.001	0.23	-0.18 (-0.20, -0.16)	<0.001
SAMINOR 1, mean†#	8.73 (8.52, 8.95)		8.75 (8.45, 9.04)			8.74 (8.57, 8.92)	
SAMINOR 2, mean†#	7.20 (7.00, 7.40)		7.48 (7.23, 7.73)			7.32 (7.17, 7.48)	
Logistic regression	**Odds ratio (95% CI)**	**p-value**	**Odds ratio (95% CI)**	**p-value**	**p-value**	**Odds ratio (95% CI)**	**p-value**
Anti-hypertensive treatment	1.19 (1.03, 1.36)	0.02	1.15 (0.97, 1.37)	0.11	0.37	1.17 (1.06, 1.31)	0.003
SAMINOR 1, prevalence %†	21.4 (19.48, 23.28)		20.7 (18.34, 23.11)			21.1 (19.60, 22.56)	
SAMINOR 2, prevalence %†	24.4 (22.15, 26.67)		23.1 (20.53, 25.74)			23.9 (22.18, 25.60)	
Hypertension	0.94 (0.83, 1.06)	0.32	0.89 (0.77, 1.04)	0.13	0.39	0.92 (0.83, 1.01)	0.08
SAMINOR 1, prevalence %†	51.0 (48.62, 53.42)		50.4 (47.43, 53.43)			50.7 (48.84, 52.63)	
SAMINOR 2, prevalence %†	49.4 (46.83, 52.01)		47.5 (44.39, 50.51)			48.6 (46.61, 50.57)	
Current smokers	0.51 (0.44, 0.58)	<0.001	0.59 (0.51, 0.69)	<0.001	0.27	0.54 (0.49, 0.60)	<0.001
SAMINOR 1, prevalence %†	30.4 (28.35, 32.50)		30.8 (28.16, 33.45)			30.7 (29.06, 32.35)	
SAMINOR 2, prevalence %†	18.1 (16.30, 19.97)		20.8 (18.55, 23.12)			19.4 (17.94, 20.81)	

β coefficients are estimated by linear generalised estimating equation regression models and adjusted for age and age^2. Odds ratios are estimated by logistic generalised estimating equation regression models and adjusted for age. †Predicted means/prevalences at age 58.2 years, which is the mean age for men in the overall sample. §Outcome variables are log-transformed. #Predicted geometric means at age 58.2 year. ‡ Test of interaction between survey and ethnicity in overall model. If p-value for interaction >0.05, interaction term is excluded from the overall model. *p-value for interaction <0.05, only ethnicity-specific estimations are reported. Number of missing values: total and HDL cholesterol were missing in 12 subjects; triglycerides were missing in 13 subjects, systolic and diastolic blood pressure and hypertension was missing in one subject. NORRISK 2 score was missing for 173 subjects. Excluding missing values did not change the results. Abbreviations: CI, confidence interval; AMS, acute myocardial infarction; CS, cerebral stroke.

DISCUSSION

From SAMINOR 1 (2003–2004) to SAMINOR 2 (2012–2014), participants from the selected 10 municipalities in Northern Norway had a favourable decline in total cholesterol, blood pressure, proportion of smokers, and the estimated 10-year risk of AMI or CS, whereas waist circumference increased. The changes in total cholesterol (men only), HDL cholesterol (both sexes), triglycerides (men only), and the estimated 10-year risk of AMS or CS (women only), were statistically significantly different between Sami and non-Sami. The odds of anti-hypertensive drug use increased only in men, whereas the prevalence of hypertension decreased only in women. To our knowledge, there are no other studies in Sápmi that explore whether changes in cardiovascular risk factors differ between Sami and non-Sami over time.

In both SAMINOR 1 and SAMINOR 2, the participation rate was lowest among the youngest participants, especially young men. In both surveys, less than half of those invited participated, hence, selection bias might be an issue. Also, as there is no official registry on ethnicity, we do not know if non-participation differed by ethnicity. It might be expected that Sami would be less willing to participate, given the history of assimilation[15] and unethical research.[28] On the other hand, as the surveys were carried out by a Sami research centre, invitees with Sami affiliations might have been more motivated to participate. If that is the case, the slightly adverse pattern in Sami, might be partly due to a different selection of Sami compared to non-Sami participants.

Further, due to design issues of SAMINOR 1,[22] the study sample included a lower proportion of participants from Sami-dominated municipalities in Finnmark, while the same municipalities had an overall high response rate in SAMINOR 2. This influences the ethnic

and regional compositions of the two samples, and makes comparisons between the surveys challenging. However, when using a different categorisation of ethnicity or adjusting for region, the results remained consistent with the main results. Moreover, generalisation to the entire Sami and non-Sami populations in Northern Norway is not advised, as only 10 municipalities were included. However, assuming a similar response rate in Sami and non-Sami participants, we believe the findings are applicable to Sami and non-Sami women and men over 50 years of age living in the given geographical regions.

The use of antihypertensive drugs increases with age[2] and during 1975–2010, the prevalence of treatment for hypertension increased by a factor of four in Norway.[29] In our study, the use of anti-hypertensive drugs in women remained similar in the surveys, whereas the prevalence of hypertension in women declined, which corresponds to a decline that is independent of treatment with anti-hypertensive drugs.[2] In men, we observed an increase in the use of anti-hypertensive drugs, whereas the prevalence of hypertension remained the same, which may indicate that treatment with anti-hypertensive drugs could have contributed to a decline in blood pressure.

The observed decreases in cholesterol, systolic blood pressure, and proportion of smokers, and the increase in waist circumference, corresponds well with studies in Western Europe[3, 4, 6, 7] and with national trends.[1, 2, 5, 8, 11, 30] Possible explanations are changes in lifestyle and diet—in line with what is observed nationally[11] —decreases in smoking, less occupational physical activity, more frequent use of vehicles for transportation, higher consumption of fruits and vegetables, lower consumption of saturated fats, and an assumed lower consumption of salt.[31] The decrease in systolic blood pressure may have been halted due to the increase in obesity over the last decades.[32]

In a cohort study in Finnmark (1987–2003), based on a follow-up of those participating in both the Finnmark 3 and SAMINOR 1 surveys, Hermansen et al.[33] observed—using the same ethnicity definition as in our study—that changes in cardiovascular risk factors according to change in physical activity level occurred independently of ethnicity. Similarly, we observed that changes in cardiovascular risk factors did not differ substantially by ethnicity, only small and probably negligible differences were observed in total cholesterol and triglycerides in men, and in HDL cholesterol in both sexes, which suggests that Sami and non-Sami populations overall have undergone similar lifestyle changes. This might be considered unexpected, as Sami may be perceived as distinct from non-Sami in terms of diet[34, 35] and physical activity.[33] A recent study from SAMINOR 2 found that participants who defined themselves solely as Sami had a lower consumption of vegetables, and a higher consumption of moose meat, reindeer meat, and fat spread on bread than non-Sami and those who regard themselves as both Sami and non-Sami.[34] In SAMINOR 1 (24 municipalities included), a higher consumption of unfiltered coffee was observed in Sami participants compared with non-Sami and Sami of mixed ethnic descent.[35] Furthermore, unpublished results from SAMINOR 2 (Borch, K., personal communication, 2018), show that, in women, Sami ethnicity was associated with lower total physical activity. In the cohort study by Hermansen et al., the proportion of leisure-time sedentary individuals in Finnmark decreased between 1987 and 2003; however, the proportions who were sedentary was higher in Sami than in non-Sami, both at baseline and at the end of follow-up.[33] Nonetheless, evidence of relevant ethnic differences in changes in cardiovascular risk factors and estimated 10-year risk of AMI and CS, was not found in our study.

The observed decline in cardiovascular risk factors is likely to have a beneficial impact on the incidence of coronary heart diseases[36] and ischemic stroke[37] in this population, which is also reflected by a decrease in the estimated 10-year risk of AMI or CS. The decrease in risk was smaller in Sami than non-Sami women, which might be due to the increase in HDL cholesterol in non-Sami women. However, the causal effect of low levels of HDL cholesterol on cardiovascular disease is debated.[38, 39]

Inuit populations are characterised by a rapid increase in obesity, diabetes, and hypertension in parallel with decreasing physical activity and deterioration of the lipid profile.[40] On the other hand, decline in smoking and alcohol use have been observed.[40] But still, there are disparities in cardiovascular health between Indigenous peoples and their benchmark populations in high-income countries.[40, 41] Our study indicated that such disparities in cardiovascular risk factors are not present in the 10 rural municipalities in Northern Norway. Previous studies have also shown similar burdens of cardiovascular risk factors and morbidity among Sami and non-Sami in Norway.[17-21] This might be due to the fact that the non-Sami reference population in these studies [17-21] lives side by side with the Sami in the same rural regions. This is a stark contrast to, for instance, the Inuit and reference Danish population, who live on different continents. If we had compared the Sami in this study to the general Norwegian population, we might have found larger differences in cardiovascular risk factors, as there are disparities in health issues across regions.[11] Second, the small or non-existent disparities in health between Sami and non-Sami are suggested to be due to similar access to health care and education,[42] whereas the lack of similar access has been put forward as a reason for health disparities between the Inuit and their reference population.[40] In summary, differences in settlement patterns and in the social determinants of health challenge our ability to compare our results with international data.

We were not able to adjust for lipid-lowering drugs, physical activity, coffee and alcohol consumption, affiliation with reindeer herding, or diet in our study, as questions relating to these items in the two surveys were not comparable. Lipid-lowering drugs are estimated to account for approximately 20–30% of the decline in total cholesterol over time,[5, 6] and therefore it is likely that some of the decline in cholesterol is due to the use of these drugs. The public health relevance of this study is that preventive measures aimed to reduce cardiovascular risk seem to have worked independent of ethnicity. Nevertheless, further surveillance of cardiovascular risk factors is advisable due to the adverse pattern—although minor—in Sami compared with non-Sami.

CONCLUSION

From SAMINOR 1 (2003–2004) to SAMINOR 2 (2012–2014), the population in rural Northern Norway had a favourable decline in total cholesterol, blood pressure, hypertension (women only), smoking, and the estimated 10-year risk of AMI or CS; however, they had an increase in waist circumference. We found only minor differences between Sami and non-Sami subjects regarding change in cardiovascular risk factors during this period, which suggests that the population of Northern Norway have had similar changes in lifestyle and diet.

CONTRIBUTION FROM CO-AUTHORS

The study was conceived by BME and TB. SRAS performed all the data analyses, produced the tables and drafted the manuscript. MM produced the figure. TB guided and assisted with statistical analyses. BME, BKJ, MM, ARB, VLM and TB helped with the interpretation of the results, and contributed to the revision of the manuscript, and approved the final version.

SHARING OF DATA

In this study we have used deidentified participant data. The data are not available for the public, since the use of the data is restricted by license. Data might, however, be available if a written request is sent to and accepted by the SAMINOR Project Board (www.saminor.no) and by the Regional Committee for Medical and Health Research Ethics.

DECLARATION OF CONFLICTING INTERESTS

There are no conflicts of interest.

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FIGURE LEGEND

Figure 1. Inhabitants aged 40–79 years living in these 10 municipalities in the Norwegian part of Sápmi were invited to the SAMINOR 1 and SAMINOR 2 surveys. Region 1 includes Kautokeino and Karasjok, region 2 includes Nesseby, Tana, and Porsanger, and region 3 includes Kåfjord, Lyngen, Storfjord, Skånland, and Evenes. There are no copyrights attached to this figure. The figure is designed for this article by one of the co-authors, Marita Melhus,

Centre for Sami Health Research at UiT The Arctic University of Norway. The figure is based on a raw map of Norway made by the Norwegian Mapping Authority, merged with a map of Europe available to the public domain at Wikipedia.

Supplementary Figure S1: Cardiovascular risk factors in women according to ethnicity and mean age in 10-year age groups in SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014).

Supplementary Figure S2: Cardiovascular risk factors in men according to ethnicity and mean age in 10-year age groups in SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014).

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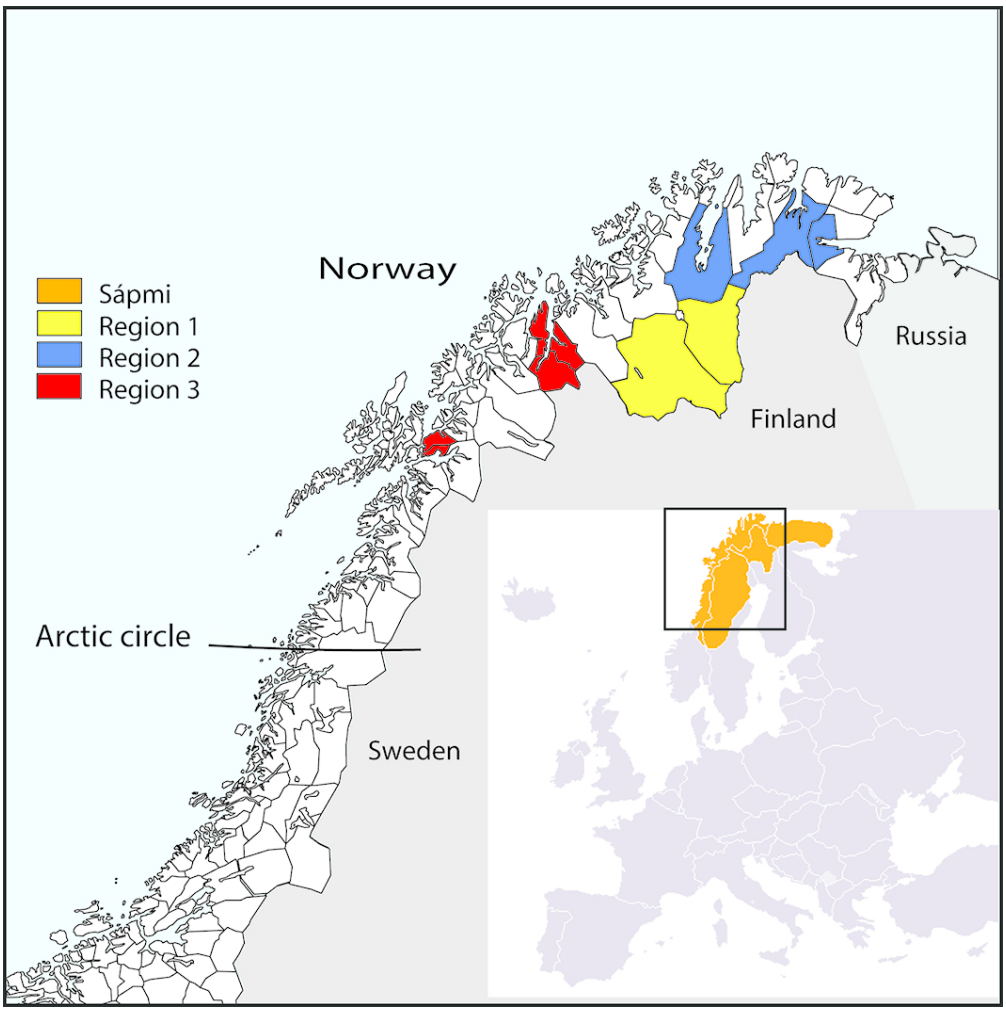
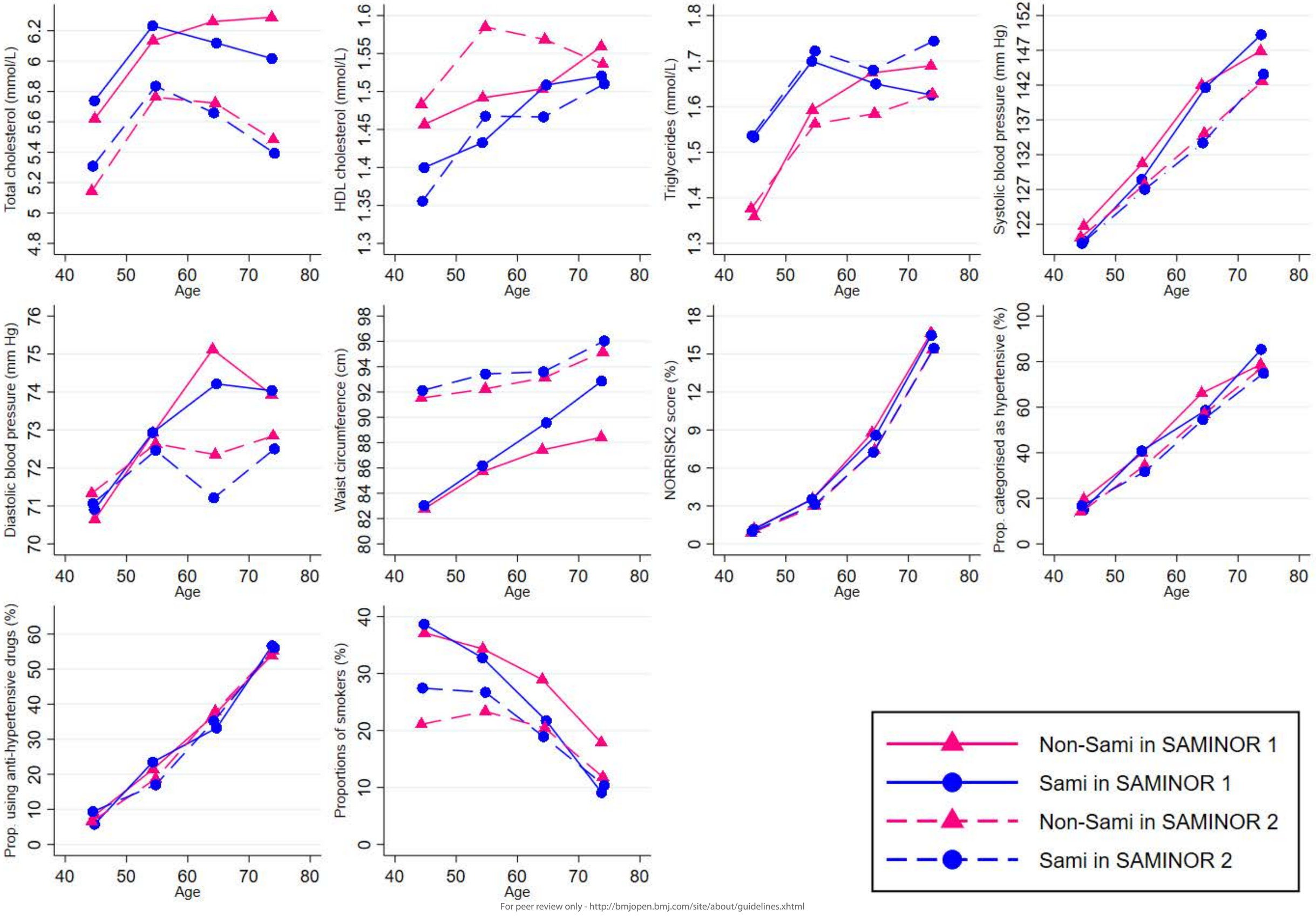


Figure 1. Inhabitants aged 40–79 years living in these 10 municipalities in the Norwegian part of Sápmi were invited to the SAMINOR 1 and SAMINOR 2 surveys. Region 1 includes Kautokeino and Karasjok, region 2 includes Nesseby, Tana, and Porsanger, and region 3 includes Kåfjord, Lyngen, Storfjord, Skånland, and Evenes.

Supplementary Figure S1. Cardiovascular risk factors in women according to survey, ethnicity and mean age in 10-year age groups



Supplementary Figure S2. Cardiovascular risk factors in men according to survey, ethnicity and mean age in 10-year age groups

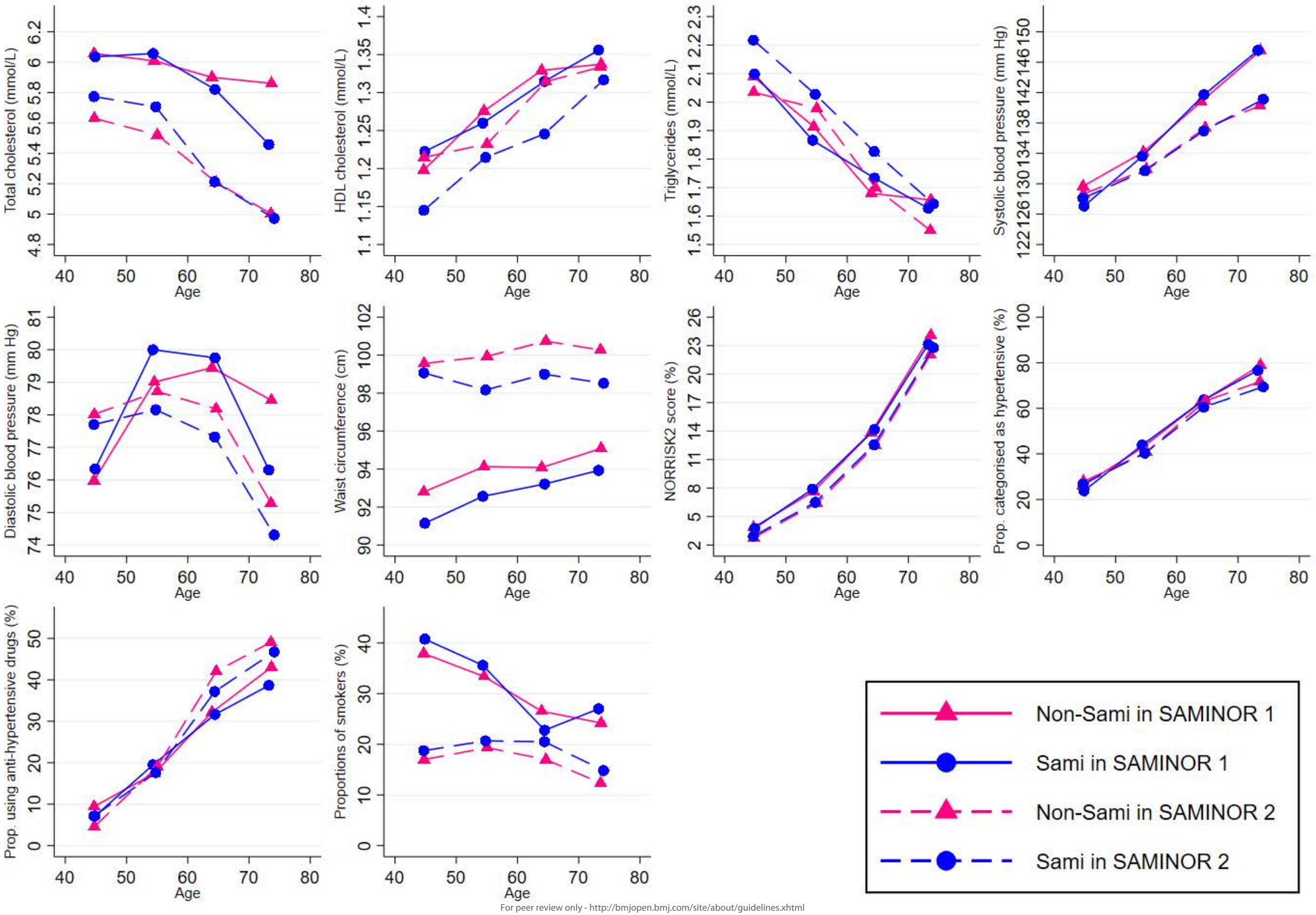


Table S1: Age-adjusted predicted changes in means and prevalences of cardiovascular risk factors between the SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014) in women (n=6624), stratified into three groups according to responses to ethnicity related questions.

Linear regression	**Non-Sami (n=3262)** **β (95% Cl)**	**p-value**	**Sami in all items (n=1453)** **β (95% Cl)**	**p-value**	**Sami in 1–10 items (n=1909)** **β (95% Cl)**	**p-value**	**Interaction‡**	**p-value**
Cholesterol, mmol/L	-0.55 (-0.61, -0.48)	<0.001	-0.44 (-0.55, -0.33)	<0.001	-0.46 (-0.55, -0.37)	<0.001	*	
SAMINOR 1, mean †	6.27 (6.21, 6.33)		6.23 (6.13, 6.34)		6.21 (6.12, 6.29)			
SAMINOR 2, mean †	5.72 (5.66, 5.79)		5.79 (5.70, 5.89)		5.75 (5.66, 5.83)			
HDL cholesterol, mmol/L	0.04 (0.02, 0.07)	<0.001	-0.03 (-0.06, -0.005)	0.02	0.03 (-0.002, 0.05)	0.07	Sami all items	<0.001
SAMINOR 1, mean †	1.53 (1.51 ,1.55)		1.47 (1.43, 1.50)		1.46 (1.43, 1.49)			
SAMINOR 2, mean †	1.58 (1.55, 1.60)		1.43 (1.40, 1.46)		1.48 (1.45, 1.51)			
Triglycerides, mmol/L§	-0.02 (-0.05, 0.004)	0.10	0.03 (-0.01, 0.07)	0.19	0.02 (-0.02, 0.05)	0.34	*	
SAMINOR 1, mean †#	2.21 (2.07, 2.34)		2.47 (2.23, 2.71)		2.59 (2.37, 2.80)			
SAMINOR 2, mean †#	2.09 (1.96, 2.23)		2.63 (2.39, 2.88)		2.69 (2.47, 2.92)			
Systolic blood pressure, mm Hg	-4.3 (-5.25, -3.13)	<0.001	-3.4 (-5.04, -1.83)	<0.001	-2.7 (-4.09, -1.23)	<0.001	*	
SAMINOR 1, mean †	134.6 (133.7, 135.6)		132.3 (130.7, 133.9)		133.7 (132.3, 135.1)			
SAMINOR 2, mean †	130.4 (129.4, 131.5)		128.8 (127.3, 130.4)		131.1 (129.7, 132.5)			
Diastolic blood pressure, mm Hg	-0.9 (-1.47, -0.28)	0.004	-1.4(-2.26, -0.57)	0.001	-0.8(-1.60, -0.02)	0.044	*	
SAMINOR 1, mean †	73.9 (73.34, 74.45)		73.2 (72.38, 74.06)		73.8 (73.00, 74.53)			
SAMINOR 2, mean †	73.0 (72.43, 73.62)		71.8 (71.00, 72.61)		73.0 (72.19, 73.71)			
Waist circumference, cm	7.1 (6.53,7.72)	<0.001	5.9 (4.97, 6.77)	<0.001	6.5 (5.75, 7.29)	<0.001	*	
SAMINOR 1, mean †	86.1 (85.48, 86.73)		88.6 (87.61, 89.59)		87.3 (86.43, 88.13)			
SAMINOR 2, mean †	93.2 (92.57, 93.90)		94.47 (93.52, 95.41)		93.8 (92.87, 94.63)			
10-year risk of AMI and CS, %§	-0.19 (-0.22, -0.16)	<0.001	-0.12 (-0.16,-0.07)	<0.001	-0.17 (-0.21,-0.12)	<0.001	Sami all items	0.02
SAMINOR 1, mean †#	4.09 (3.97, 4.21)		3.81 (3.63, 3.98)		4.22 (4.04, 4.40)			
SAMINOR 2, mean †#	3.37 (3.27, 3.48)		3.39 (3.24, 3.54)		3.58 (3.43, 3.73)			
Logistic regression	**Odds ratio (95% Cl)**	**p-value**	**Odds ratio (95% Cl)**	**p-value**	**Odds ratio (95% Cl)**	**p-value**		**p-value**
Antihypertensive drugs	0.96 (0.83, 1.11)	0.61	0.98 (0.77, 1.23)	0.84	0.95 (0.79, 1.14)	0.58	*	
SAMINOR 1, prevalences % †	22.9 (20.82, 24.94)		20.1 (16.96, 23.33)		27.2 (24.30, 30.15)			
SAMINOR 2, prevalences % †	22.2 (19.93, 24.50)		19.8 (16.60, 22.90)		26.2 (23.29, 29.16)			
Hypertension	0.78 (0.68, 0.90)	<0.001	0.76 (0.61, 0.94)	0.010	0.77 (0.65, 0.92)	0.004	*	
SAMINOR 1, prevalences % †	46.0 (43.45, 48.62)		42.9 (38.78, 47.05)		49.6 (46.06, 53.07)			
SAMINOR 2, prevalences % †	40.1 (37.29, 42.81)		36.3 (32.44, 40.20)		43.2 (39.76, 46.60)			
Current smoking	0.61 (0.53, 0.69)	<0.001	0.78 (0.64, 0.94)	0.011	0.63 (0.54, 0.74)	<0.001	*	
SAMINOR 1, prevalences % †	30.3 (28.15, 32.41)		26.3 (23.02, 29.61)		30.8 (27.94, 33.75)			
SAMINOR 2, prevalences % †	20.9 (18.91, 22.85)		21.7 (18.85, 24.61)		22.0 (19.50, 24.45)			

β –coefficients are estimated by linear generalised estimating equation regression models and adjusted for age and age^2. Odds ratios are estimated by logistic generalised estimating equation regression models and adjusted for age. †Predicted means/prevalences at age 57.5 years. §Outcome variables are log-transformed. # Predicted geometric means at age 57.5. ‡ Testing for interaction between survey and ethnicity using non-Sami as reference. If p-value for interaction <0.05, we have specified which group differed from non-Sami in SAMINOR 2. * If p-value for interaction between survey and ethnicity is >0.05, please refer to the overall estimation given in Table 3. Sami in all items (11 in total): reported use of Sami language in grandparents, parents and themselves; Sami ethnic background for parents and themselves; Sami as self-perceived ethnicity. Sami in 1–10 items: reported Sami for at least one item and maximum for 10 items. Non-Sami: all others. Total and HDL cholesterol and triglycerides were missing in 18 subjects; systolic and diastolic blood pressure were missing in four subjects; hypertension in three. NORRISK 2 score was missing for 193 subjects. Abbreviations: HDL, high density lipoprotein; CI, confidence intervals; AMS, acute myocardial infarction; CS, cerebral stroke.

Table S2: Age-adjusted change in means and prevalences of cardiovascular risk factors between the SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014) in men (n=5749), stratified into three categories according to responses to ethnicity related questions.

Linear regression	**Non-Sami (n=2678)** **β (95% Cl)**	**p-value**	**Sami in all items (n=1244)** **β (95% Cl)**	**p-value**	**Sami in 1–10 items (n=1827)** **β (95% Cl)**	**p-value**	**Interaction‡**	**p-value**
Cholesterol, mmol/L	-0.6 (-0.69, -0.53)	<0.001	-0.4 (-0.50, -0.28)	<0.001	-0.5 (-0.61, -0.41)	<0.001	Sami all items	0.023
SAMINOR 1, mean †	5.99 (5.92, 6.05)		6.01 (5.90, 6.11)		6.03 (5.94, 6.12)			
SAMINOR 2, mean †	5.38 (5.30, 5.45)		5.61 (5.51, 5.72)		5.52 (5.43, 5.61)			
HDL cholesterol, mmol/L	-0.01 (-0.03, 0.011)	0.33	-0.09(-0.12, -0.06)	<0.001	-0.03 (-0.05, -0.003)	0.03	Sami all items	0.001
SAMINOR 1, mean †	1.29 (1.27, 1.31)		1.29 (1.26, 1.32)		1.29 (1.27, 1.32)			
SAMINOR 2, mean †	1.28 (1.26, 1.30)		1.20 (1.17, 1.23)		1.27 (1.24, 1.29)			
Triglycerides, mmol/L§	0.003 (-0.03, 0.04)	0.89	0.10 (0.05, 0.15)	<0.001	0.03 (-0.02, 0.07)	0.22	Sami all items	0.005
SAMINOR 1, mean †#	2.91 (2.71, 3.14)		2.82 (2.50, 3.14)		3.04 (2.77, 3.31)			
SAMINOR 2, mean †#	2.94 (2.70, 3.18)		3.56 (3.17, 3.96)		3.22 (2.93, 3.53)			
Systolic blood pressure, mm Hg	-2.6 (-3.78, -1.50)	<0.001	-3.3 (-5.15, -1.54)	<0.001	-3.7 (-5.17, -2.17)	<0.001	*	
SAMINOR 1, mean †	136.8 (135.7, 137.8)		135.7 (134.0, 137.5)		138.0 (136.7, 139.4)			
SAMINOR 2, mean †	134.1 (133.0, 135.2)		132.4 (130.7, 134.1)		134.4 (132.9, 135.8)			
Diastolic blood pressure, mm Hg	-0.3 (-0.99, 0.36)	0.36	-1.2 (-2.19, -0.28)	0.011	-1.0 (-1.88, -0.17)	0.019	*	
SAMINOR 1, mean †	79.7 (79.13, 80.34)		79.2 (78.25, 80.04)		80.3 (79.51, 81.05)			
SAMINOR 2, mean †	79.4 (78.75, 80.09)		77.9 (77.02, 78.80)		79.3 (78.45, 80.06)			
Waist circumference, cm	6.3 (5.62, 6.87)	<0.001	5.9 (5.15, 6.69)	<0.001	5.61 (4.85, 6.38)	<0.001	Sami 1–10	0.04
SAMINOR 1, mean †	94.9 (94.25, 95.46)		92.6 (91.78, 93.49)		93.1 (92.37, 93.89)			
SAMINOR 2, mean †	101.1 (100.43, 101.77)		98.6 (97.68, 99.42)		98.8 (97.96, 99.53)			
10-year risk of AMI and CS, %§	-0.18 (-0.21, -0.15)	<0.001	-0.15 (-0.20, -0.11)	<0.001	-0.19 (-0.23, -0.15)	<0.001	*	
SAMINOR 1, mean †#	8.65 (8.41, 8.89)		8.54 (8.16, 8.92)		9.05 (8.72, 9.39)			
SAMINOR 2, mean †#	7.21 (6.99, 7.43)		7.34 (7.02, 7.66)		7.48 (7.19, 7.76)			
Logistic regression	**Odds ratios (95% Cl)**	**p-value**	**Odds ratios (95% Cl)**	**p-value**	**Odds ratio (95% Cl)**	**p-value**		**p-value**
Anti-hypertensive drugs	1.20 (1.03, 1.40)	0.022	1.15 (0.91, 1.46)	0.24	1.11 (0.91, 1.35)	0.30	*	
SAMINOR 1, prevalences % †	21.40 (19.25, 23.55)		18.43 (15.37, 21.49)		22.71 (19.99, 25.44)			
SAMINOR 2, prevalences % †	24.58 (22.00, 27.16)		20.66 (17.29, 24.04)		24.59 (21.57, 27.61)			
Hypertension	0.96 (0.83, 1.10)	0.51	0.87 (0.71, 1.07)	0.19	0.85 (0.72, 1.02)	0.07	*	
SAMINOR 1, prevalences % †	50.37 (47.64, 53.09)		45.39 (41.26, 49.51)		55.21 (51.87, 58.56)			
SAMINOR 2, prevalences % †	49.20 (46.26, 52.14)		42.02 (37.90, 46.15)		51.23 (47.75, 54.71)			
Current smoking	0.54 (0.46, 0.63)	<0.001	0.58 (0.47, 0.71)	<0.001	0.53 (0.44, 0.63)	<0.001	*	
SAMINOR 1, prevalences % †	29.79 (27.44, 32.14)		31.65 (28.05, 35.25)		31.63 (28.70, 34.57)			
SAMINOR 2, prevalences % †	18.49 (16.38, 20.60)		21.02 (17.91, 24.12)		19.54 (16.98, 22.10)			

β–coefficients are estimated by linear generalised estimating equation regression models and adjusted for age and age^2. Odds ratios are estimated by logistic generalised estimating equation regression models and adjusted for age. †Predicted means/prevalences at age 58.2 years. §Outcome variables are log-transformed. # Predicted geometric means at age 58.2. ‡ Testing for interaction between survey and ethnicity using non-Sami in SAMIMNOR 1 as reference. If p-value for interaction <0.05, we have specified which group differs from non-Sami in SAMINOR 2. *p-value for interaction between survey and ethnicity >0.05, please refer to the overall estimation given in Table 4. Sami in all items (11 in total): reported use of Sami language in grandparents, parents and themselves; Sami ethnic background for parents and themselves; Sami as self-perceived ethnicity. Sami in 1–10 items: reported Sami for at least one item and maximum for 10 items. Non-Sami: all others. Total and HDL cholesterol were missing in 12 subjects; triglycerides were missing in 13 subjects, systolic and diastolic blood pressure and hypertension was missing in one subject. NORRISK 2 score was missing for 173 subjects. Abbreviations: HDL, high density lipoprotein; CI, confidence intervals; AMS, acute myocardial infarction; CS, cerebral stroke.

Table S3: Age- and region adjusted predicted changes in beta coefficients and odds ratios of cardiovascular risk factors between SAMINOR 1 (2002–2004) and the SAMINOR 2 (2012–2017) in women and men, after testing for interaction between survey and ethnicity.

	Women (n=6624)		**Men** (n=5749)	
Linear regression	**β (95% Cl)**	**p-value**	**β (95% Cl)**	**p-value**
Total cholesterol, mmol/L	-0.50 (-0.54, -0.45)	<0.001	*	
	-		Non-Sami: -0.59 (-0.65, -0.52)	<0.001
	-		Sami: -0.43 (-0.51, -0.34)	<0.001
HDL cholesterol, mmol/L	*		*	
	Non-Sami: 0.05 (0.03, 0.07)	<0.001	Non-Sami: -0.01 (-0.03, 0.01)	0.31
	Sami: -0.01 (-0.04, 0.01)	0.24	Sami: -0.06 (-0.08, -0.03)	<0.001
Triglycerides, mmol/L †	-0.001 (-0.02, 0.02)	0.96	*	
	-		Non-Sami: -0.0002 (-0.03, 0.003)	0.99
	-		Sami: 0.09 (0.05, 0.12)	<0.001
Systolic blood pressure, mm Hg	-3.5 (-4.25, -2.77)	<0.001	-3.0 (-3.81, -2.21)	<0.001
Diastolic blood pressure, mm Hg	-1.0 (-1.37, -0.55)	<0.001	-0.8 (-1.22, -0.30)	0.001
Waist circumference, cm	6.6 (6.20, 7.03)	<0.001	5.9 (5.52, 6.33)	<0.001
10-year risk of AMI and CS, %†	*		-0.18 (-0.20, -0.16)	<0.001
	Non-Sami:-0.19 (-0.22, -0.17)	<0.001	-	
	Sami: -0.12 (-0.16, -0.09)	<0.001	-	
Logistic regression	**Odds ratio (95% Cl)**	**p-value**	**Odds ratio (95% Cl)**	**p-value**
Use of anti-hypertensive drugs	0.98 (0.89, 1.08)	0.68	1.20 (1.08, 1.33)	0.001
Hypertensive	0.79 (0.72, 0.87)	<0.001	0.93 (0.84, 1.02)	0.13
Current smoking	0.64 (0.59, 0.70)	<0.001	0.53 (0.48, 0.59)	<0.001

β–coefficients are estimated by linear generalised estimating equation regression models and adjusted for age, age^2 and region. Odds ratios are estimates by logistic generalised estimating equation regression models and adjusted for age and regions. Adjusting for three regions including the following municipalities: 1) Kautokeino and Karasjok. 2) Nesseby, Tana and Porsanger. 3) Kåfjord, Lyngen, Storfjord, Skånland and Evenes. When p-value for interaction between survey and ethnicity is >0.05, overall β –coefficients and odds ratios adjusted for region are reported, otherwise (indicated by *), ethnic specific β–coefficients are reported. †Outcome variables are log transformed.

STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of *cross-sectional studies*

Section/Topic	Item #	Recommendation	Reported on page #
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	1
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2–3
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	5
Objectives	3	State specific objectives, including any prespecified hypotheses	5–6
Methods			
Study design	4	Present key elements of study design early in the paper	6
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	6
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants	6–7
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	10–11, 16, 18
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	7–10
Bias	9	Describe any efforts to address potential sources of bias	N/A
Study size	10	Explain how the study size was arrived at	6–7
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	9
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	10–11
		(*b*) Describe any methods used to examine subgroups and interactions	10–11
		(*c*) Explain how missing data were addressed	7, 8, 16, 18
		(*d*) If applicable, describe analytical methods taking account of sampling strategy	N/A
		(*e*) Describe any sensitivity analyses	11
Results			

Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	6–7
		(b) Give reasons for non-participation at each stage	6–7
		(c) Consider use of a flow diagram	N/A
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	14, 15
		(b) Indicate number of participants with missing data for each variable of interest	16, 18
Outcome data	15*	Report numbers of outcome events or summary measures	16, 18
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	16, 18
		(*b*) Report category boundaries when continuous variables were categorized	16, 18
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	12, 16
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	17, Table S1, S2, S3
Discussion			
Key results	18	Summarise key results with reference to study objectives	19
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	19
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	19–23
Generalisability	21	Discuss the generalisability (external validity) of the study results	19–20
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	24

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.