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Cardiovascular risk factors and incidence of acute myocardial infarction and cerebral stroke in Sami and non-Sami populations—The SAMINOR Study

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Susanna, 2020 Sep

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Summary

The overall aim of this thesis was to examine and compare cardiovascular risk factors, time-trends in these risk factors, and the incidence of cardiovascular disease in Sami and non-Sami populations living in the same rural geographical area.

For this thesis, we used information from the SAMINOR Study. This population-based study on health and living conditions in regions with Sami and non-Sami populations in Northern and Mid Norway consists of three separate surveys, conducted in two waves. Paper 1 was based on data from the SAMINOR 2 Clinical Survey (SAMINOR 2, 2012–2014), which was conducted in 10 municipalities of Northern Norway. In this paper, conventional cardiovascular risk factors (hereafter referred to as conventional risk factors) were compared and the 10-year risk of fatal or non-fatal acute myocardial infarction or cerebral stroke (hereafter referred to as 10-year risk) was estimated using the NORRISK 2 risk model in Sami and non-Sami women and men aged 40–79 years. In Paper 2, we examined the change in conventional risk factors and 10-year risk from the SAMINOR 1 Survey (SAMINOR 1, 2003–2004) to SAMINOR 2 in participants aged 40–79 years that lived in 10 municipalities in Northern Norway. In Paper 3, SAMINOR 1 participants aged 30 and 36–79 years were followed until the end of 2016. Baseline information from SAMNOR 1 was linked to hospital discharge data provided by the Cardiovascular Disease in Norway Project (1993–2009) or the Norwegian Patient Registry (2010–2016); to cause of death data from the Cause of Death Registry; and to emigration data from Statistics Norway. The aim of Paper 3 was to measure and compare the risk of fatal or non-fatal acute myocardial infarction, coronary heart disease, ischemic stroke, cerebral stroke and a composite endpoint (acute myocardial infarction or cerebral stroke) in Sami and non-Sami populations.

In Paper 1, we observed some small ethnic differences in conventional risk factors. The estimated and age-standardised 10-year risks were similar in Sami and non-Sami participants who were free of angina pectoris or myocardial infarction. Paper 2 showed an overall favourable change in conventional risk factors from SAMINOR 1 to SAMINOR 2 in both sexes and ethnic groups. Compared with non-Sami men, Sami men had a somewhat smaller change in lipids that most likely are of no clinical relevance in terms of different risk; and in women, Sami had a smaller decline in the estimated 10-year risk than non-Sami. Both Sami and non-Sami had an increase in waist circumference over time. Paper 3 showed that, during 13 years of follow-up in SAMINOR 1, the risk of ischemic stroke and cerebral stroke was approximately 36% and 31% higher, respectively, in Sami than in non-Sami participants. This higher risk was not explained by differences in conventional risk factors, which might not be surprising given the similar estimated 10-year risks observed in Paper 1, and the rather similar change in conventional risk factors over time observed in Paper 2. However, in Paper 3 we also observed that adjustment for height attenuated the ethnic differences in the risk of stroke somewhat. We observed no ethnic differences in the risk of acute myocardial infarction, coronary heart diseases, or in the composite endpoint in Paper 3.

The results of this thesis indicate only small ethnic differences in conventional risk factors and a somewhat higher risk of ischemic and cerebral stroke in Sami. Ethnic differences were not explained by conventional risk factors, but a substantial part was explained by height. The differences in the risk of stroke were small and may have been due to bias (residual confounding) or chance. Therefore, more studies are warranted to replicate the findings and to explore how height is associated with excess risk in this population.

Sammendrag
Formålet med avhandlingen var å undersøke og sammenligne konvensjonelle risikofaktorer til hjerte- og karsykdommer, trender i risikofaktorer og insidens av hjerte- og karsykdommer hos den samiske og ikke-samiske befolkningen som bor i de samme geografiske rurale områdene.

Vi har brukt data fra SAMINOR til artiklene som inngår i avhandlingen. SAMINOR-undersøkelsen (Helse- og levekårsundersøkelsen i områder med samisk og norsk bosetting) som er en befolkningsundersøkelse om helse og levekår i den samiske og ikke-samiske befolkingen i Nord- og Midt Norge. SAMINOR består av tre separate undersøkelser gjennomført over to innsamlingsperioder. I den første artikkelen brukte vi data fra SAMINOR 2 klinisk undersøkelse (SAMINOR 2, 2012–2014), hvor deltakerne var mellom 40–79 år og fra 10 kommuner i Nord-Norge. Vi sammenlignet konvensjonelle risikofaktorer for hjerte- og karsykdommer og målte 10-års risiko for fatalt og ikke-fatalt hjerteinfarkt eller hjerneslag (heretter 10-års risiko) hos samer og ikke-samer ved hjelp av NORRISK 2 risikokalkulator. I den andre artikkelen undersøkte vi om endringer i konvensjonelle risikofaktorer fra SAMINOR 1 undersøkelsen (SAMINOR 1, 2003–2004) til SAMINOR 2 var lik for samiske og ikke-samiske deltakere mellom 40 og 79 år, bosatt i 10 kommuner i Nord-Norge. I den tredje artikkelen ble deltakere i SAMINOR 1, som var i alderen 30 og 36–79 år, fulgt opp til slutten av 2016. Oppfølgingen skjedde ved at opplysninger gitt ved SAMINOR 1 ble koblet til Dødsårsaksregistret for opplysninger om underliggende dødsårsak, til Statistisk sentralbyrå for opplysninger om emigrasjon, og til CVDNOR prosjektet (Cardiovascular Disease in Norway Project) og til Norsk pasientregister for opplysninger om utskrivningsdiagnoser for henholdsvis 1993–2009 og 2010–2016. Risikoen for fatalt og ikke-fatalt akutt hjerteinfarkt, koronar hjertesykdom, iskemisk hjerneslag, cerebralt hjerneslag og et samlet endepunkt (akutt hjerteinfarkt eller cerebral hjerneslag) ble målt hos samer og ikke-samer, og gruppenes risiko ble sammenlignet.

I den første artikkel fant vi noen små etniske forskjeller i konvensjonelle risikofaktorer. Den estimerte og aldersstandardiserte 10-års risikoen var lik for samer og ikke-samer som ikke hadde angina pectoris eller gjennomgått hjerteinfarkt før oppfølgingen startet. Den andre artikkelen viste at begge kjønn og etniske grupper hadde en gunstig utvikling i konvensjonelle risikofaktorer fra SAMINOR 1 til SAMINOR 2. Sammenlignet med ikke-samiske menn, hadde samiske menn en litt mindre fordelaktig utvikling av lipider, mens samiske kvinner hadde en mindre nedgang i 10-års risiko enn ikke-samiske kvinner. Begge etniske grupper hadde en økning i midjeomkrets. Resultatene fra den tredje artikkelen viste at i en oppfølging på 13 år, hadde samiske deltakere omtrent 36% og 31% høyere risiko for henholdsvis iskemisk og cerebralt hjerneslag. Den økte risikoen ble ikke forklart av konvensjonelle risikofaktorer for hjerte- og karsykdommer, noe som ikke var uventet gitt at gruppene hadde lik 10-års risiko (artikkel 1) og relativt like endringer av risikofaktorer over tid (artikkel 2). Ved å justere for høyde, ble de etniske forskjellene redusert. Risikoen for akutt hjerteinfarkt, koronar hjertesykdom eller for det samlede endepunktet, var lik hos samer og ikke-samer.

Denne avhandlingen viser at det er små forskjeller i konvensjonelle risikofaktorer, og at samiske menn og kvinner har en noe forhøyet risiko for hjerneslag. Forskjellene i risiko var derimot små, og vi kan ikke utelukke at resultatene skyldes systematiske skjevheter eller tilfeldigheter. Derfor er det nødvendig med flere studier som kan bekrefte funnene og undersøke hvordan høyde er assosiert med risiko for hjerneslag i denne befolkningen.

Čoahkkáigeassu

Dán dutkamuša váldomihttu lea leamaš iskat ja buohtastahttit dábálaš riskafáktoriid váibmo- ja varrasuotnasivaide, movt riskafáktorat rivdet, ja mihtidit movt váibmo- ja varrasuotnasivat dihttojit sápmelaččain ja eará čearddalaš joavkkus geat orrot seamma doaresbeale guovlluin.

Dutkosa artihkkaliin leat geavahan dieđuid SAMINOR guorahallamis, mii lea dearvvašvuođa- ja eallindilleiskkadeapmi sámi álbmogis ja álbmogis geat eai leat sápmelaččat Davvi- ja Gaska Norggas. SAMINOR iskkadeapmái gullet golbma sierra iskosa mat leat čoggojuvvon guovtti áigodaga badjel. Vuosttaš artihkkalii geavaheimmet SAMINOR 2 klinalaš iskkadeami (SAMINOR 2, 2012–2014) mii čađahuvvui 10 suohkanis Davvi Norggas ja mas rávisolbmot gaskal 40 ja 79 jagi oassálaste. Mii buohtastahtiimet dábálaš riskafáktoriid váibmo- ja varrasuotnasivaide sápmelaččain ja eará čearddalaš joavkkus, ja mihtideimmet NORRISK 2 riskamodeallain sin 10-jagi riskadási jápmit dahje buohccát vuoiŋŋaščaskkástagain dahje fáhkka váibmodohppehagain (dás maŋás 10-jahkasaš riskadássi). Nuppi artihkkalis guorahalaimet rivdet go dábálaš riskafáktorat ja 10-jagi riskadássi seamma ládje sápmelaččain ja eará čearddalaš joavkkus geat ledje gaskal 40 ja 79 jagi, orro 10 suohkanis Davvi Norggas, ja serve SAMINOR 1 (SAMINOR 1, 2003–2004) ja SAMINOR 2 iskkadeapmái. Goalmmát artihkkalis mii čuovuimet SAMINOR 1 oassálastiid geat ledje 30, ja gaskal 36 ja 79 jagi, gitta 2016 loahpageahčai. Dieđut mat ledje čohkkejuvvon SAMINOR 1 iskkadeamis čadnojuvvojedje dieđuide Jápminsivvaregistaris diđoštit mainna sivain olbmot jápmet, Váibmo- ja varrasuotnaregistarii (CVDNOR) dahje Norgga pasieantaregistarii diđoštit mainna sivain buohccájit, ja Statistihkalaš guovddášdoaimmahaga registarii diđoštit leat go olbmot fárren eret riikkas. Mii mihtideimmet ja buohtastahtiimet dihttogo jápmin dahje fáhkka buohccán seamma dávjá sápmelaččain go eará čearddalaš joavkkus čuovvovaš dávddaide: fáhkka váibmodohppehahkii, vigit guoskevaččat váldováibmosuonaide, vuoiŋŋaščaskkástahkii, eará vuoiŋŋamaš vigiide ja muhtin seahkalas dávddaide.

Vuosttaš artihkkala gávdnosat čájehit ahte dábálaš riskafáktoriid dáfus ledje smávva erohusat čearddaid gaskkas. Vuosttaš artihkkalis lei dat merrojuvvon ja ahkeheivehuvvon 10-jagi riskadássi seammadássásaš sápmelaččain ja eará čearddalaš joavkkus go buohtastahtiimet oassálastiid geain ii lean ovdalaččas angina pectoris dahje váibmodohppehat. Nuppi artihkkalis oaidnit ahte dábálaš riskafáktorat leat njiedjan sihke sápmelaččain ja eará čearddalaš joavkkus SAMINOR 1 iskkadeamis SAMINOR 2 iskkadeami ektui, earetgo seakkášmihttu, mii lei sturron. Buohtastasttedettiin vuhtiimet ahte sámi dievdduin lei buoidemearri varas njiedjan veahá unnit go eará čearddalaš joavkkus. Nissonolbmuid gaskkas fas vuhttui ahte sámi nissoniin lei merrojuvvon 10-jagi riskadássi njiedjan veahá unnit go eará čearddalaš joavkkus. Goalmmát artihkkalis čuovuimet oassálastiid 13-jagi ja mihtut čájehedje ahte sápmelaččain dihttojedje vuoiŋŋamaš vigit 36% dávjjibut ja vuoiŋŋaščaskkástagat 31% dávjjibut go eará čearddalaš joavkkus. Dábálaš riskafáktorat eai čilgen manin sápmelaččain lei alibuš riska, mii ii lean nu imáš go eai han lean mearkkašeaddji erohusat 10-jagi riskadásis ja riskafáktoriin vuosttaš ja nuppi artihkkalis. Olbmo allodat čilgii daid čearddalaš erohusaid, muhto ii ollislaččat. Eat gávdnan čearddalaš erohusaid das man dávjá dihtto fáhkka váibmodohppehat, vigit guoskevaččat váldováibmosuonaide ja muhtin seahkalas dávddaide.

Dát dutkamuš čájeha ahte sápmelaččain ja eará čearddalaččain geat orrot seamma guovlluin leat ovttalágan dásit riskafáktoriiguin, muhto sápmelaččat dohppehallojit veahá dávjjibut vuoiŋŋamaš vigiide ja vuoiŋŋaščaskkástagaide. Dákkár dutkama ferte geardduhit go eat sáhte áibbas sihkkarit earuhit leat go meattáhusat dutkanvugiin dahje soahttáhat mat dagahit dákkár bohtosiid. Maiddái lea dárbu iskat movt olbmo allodat sáhttá váikkuhit vuoiŋŋamaš vigiide dáin čearddalaš joavkkuin.

List of papers

This thesis is based on the following papers, referred to in the text as Paper 1, Paper 2, and Paper 3.

Paper 1

Siri, Susanna Ragnhild Andersdatter; Braaten, Tonje; Jacobsen, Bjarne Koster; Melhus, Marita; Eliassen, Bent-Martin. *Distribution of risk factors for cardiovascular disease and the estimated 10-year risk of acute myocardial infarction or cerebral stroke in Sami and non-Sami populations: The SAMINOR 2 Clinical Survey*. Scandinavian Journal of Public Health 2018;46(6):638–646. Online ISSN 1651-1905. Doi: 10.1177/1403494818773534

Paper 2

Siri, Susanna Ragnhild Andersdatter; Eliassen, Bent Martin; Jacobsen, Bjarne Koster; Melhus, Marita; Broderstad, Ann Ragnhild; Michalsen, Vilde Lehne; Braaten, Tonje. *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*. BMJ Open 2019;9(7):e028939. Online ISSN 2044-6055. Doi: 10.1136/bmjopen-2019-028939

Paper 3

Siri, Susanna Ragnhild Andersdatter; Eliassen, Bent Martin; Broderstad, Ann Ragnhild; Melhus, Marita; Michalsen, Vilde Lehne; Jacobsen, Bjarne Koster; Burchill, Luke; Braaten, Tonje. *Coronary heart disease and stroke in the Sami and non-Sami population in rural Northern and Mid Norway–the SAMINOR Study*. Open Heart. 2020;7(1):e001213. Online ISSN: 2053-3624. Doi: 10.1136/openhrt-2019-001213.

Abbreviations and definitions

AMI	Acute myocardial infarction
BMI	Body mass index
CHD	Coronary heart disease
CI	Confidence interval
CS	Cerebral stroke
CVD	Cardiovascular disease
CVDNOR	the Cardiovascular Disease in Norway project
DBP	Diastolic blood pressure
ESC	European Society of Cardiology
HDL	High-density lipoprotein
ICD-10	International Statistical Classification of Diseases and Related Health Problems, the 10^{th} revision
IS	Ischemic stroke
LDL	Low-density lipoprotein
REC North	Regional Committee for Medical and Health Research Ethics for region North
SAMINOR 1	the first survey of the Population-based Study on Health and Living Conditions in Regions with Sami and Norwegian Populations
SAMINOR 2	the SAMINOR 2 Clinical Survey, a part of the second survey of the Population-based Study on Health and Living Conditions in Regions with Sami and Norwegian Populations
SBP	Systolic blood pressure

Definitions

Incident cases: the number of new cases that occur in a population of known size (person-years) within a defined time-period. [1] It may be measured as frequency, a rate or a proportion.[2]

Incidence rate: computed by dividing the number of new cases in a population in a given time-period, by the number of people who are at risk (person-years) of getting the disease within that same time-period.[2]

Prevalence: the number of cases (old and new) at a specific time-point in a population of known size, [1] also known as point prevalence.

1 Introduction

Cardiovascular disease (CVD) was the most common cause of death in 2013, accounting for about 32% of all deaths worldwide.[3, 4] CVD also accounted for most deaths across 52 member countries of the European Society of Cardiology (ESC) in 2007–2017.[5] Globally, among the different CVD related deaths, coronary heart disease (CHD) and cerebral stroke (CS) ranked as the two most common causes of death in 2013,[4, 5] except in some Balkan countries.[4] Although CVD has been a leading cause of death, age-standardised CVD mortality rates have decreased across Europe since 2003, and some Western European countries actually experience more deaths due to cancer than to CVD.[3, 5] In 1980 to 2016, overall CS mortality declined across Europe and Central Asia, with the largest decline observed in Western European countries, and a plateauing trend was observed for Western and Central Europe in the most recent period.[6]

Guidelines for the prevention of CVD recommend assessing the impact of several risk factors simultaneously, using risk models that estimate absolute risk.[7, 8] The risk of CVD is continuous and multifactorial, i.e. there are modifiable and non-modifiable risk factors that act both independently and together. Non-modifiable risk factors include, but are not limited to, age, sex, and family history of premature CHD. Modifiable CVD risk factors include behavioural factors such as tobacco use, alcohol consumption, diet, and physical activity, and markers in blood e.g. lipids, including low-density lipoprotein (LDL), high-density lipoprotein (HDL) cholesterol, non-HDL cholesterol, triglycerides, and total cholesterol; hypertension; obesity; and diabetes.[9] In high-income countries, more than 60% of population attributable risk for CVD were due to behavioural risk factors and markers in blood.[9] Hypertension, hyperlipidaemia, smoking, and diabetes are considered to be conventional cardiovascular risk factors,[10] or sometimes referred to as termed as traditional cardiovascular

risk factors together with age and sex.[11] In this thesis, the factors included in the NORRISK 2 model[12] (section 1.3) are termed 'conventional risk factors' and refers to triglycerides, HDL cholesterol, total cholesterol, systolic and diastolic blood pressure (SBP and DBP), use of antihypertensive medication, smoking, and family history of premature CHD disease.

The Sami are the indigenous population in Norway. However, the Sami people also live in Sweden, Finland, and the Kola Peninsula in the Russian Federation. Internationally, indigenous populations experience poorer health outcomes and have a shorter life expectancy than their reference populations, which is often the majority population in the country or region.[13, 14] The region of the Sami Parliament subsidy schemes for business development has been used as a surrogate measure for being Sami,[15] and in 2013, this region had 1.6 fewer years of life expectancy at birth than the rest of the Norwegian population.[13] However, the subsidy region comprise a mixed-ethnic population including Sami, Norwegians, and Kven, who are descendants of Finnish-speaking people that came from Sweden and Finland in the 1700s and 1800s.[16] There are no studies comparing life expectancy in Sami and their reference population (hereafter referred to as non-Sami) using individual-level data. There is still some uncertainty as to whether Sami in Norway have similar or poorer cardiovascular health than non-Sami, as previous studies show small to no differences [17-22] in conventional risk factors. The incidence of CHD has been found to be similar in Sami and non-Sami,[23] whereas studies on CVD mortality show contradicting results.[24, 25] Incidence of and mortality from CS have been reported to be similar [26] and higher,[25] in Sami compared to non-Sami populations in Norway. Therefore, there is still a need to monitor conventional risk factors in Sami and compare them to their non-Sami counterparts. Also, there is a need for a better understanding of the associations between Sami ethnicity and cardiovascular health that calls for more research on this topic.

1.1 Cardiovascular disease

CVD is a diseases of the circulatory system, which includes the heart and the blood vessels. Common to some CVD is the underlying pathological process known as atherosclerosis, and conventional risk factors contribute to the formation of atherosclerosis. This thesis concerns descriptive epidemiology of conventional risk factors and the risk, i.e. fatal or non-fatal, of two groups of CVD: CHD, which is defined by International Statistical Classification of Diseases and Related Health Problems, the 10th revision (ICD-10) [27] codes I20–I25, and include acute myocardial infarction (AMI) and subsequent myocardial infarction, defined by ICD-10 codes I21–I22; and CS, which in this thesis comprises bleedings defined by ICD-10 codes I60–I61, and infarctions and unspecified strokes, defined by ICD-10 codes I63–I64, but not ICD-10 code I63.6 that represents cerebral infraction due to cerebral venous thrombosis, non-pyogenic. The defined endpoint included in this thesis are the same as used in the NORRISK 2 model [12] (section 1.3).

Epidemiology of cardiovascular disease in Norway

Since the 1970s, there has been a constant decline in CVD mortality in Norway (Figure 1), which has been reflected by a decline in mortality from CHD and CS (Figures 2 and 3). Northern Norway have from 1970s and up till today consistently shown a higher CVD mortality, particularly in men, than the rest of Norway (Figure 1).

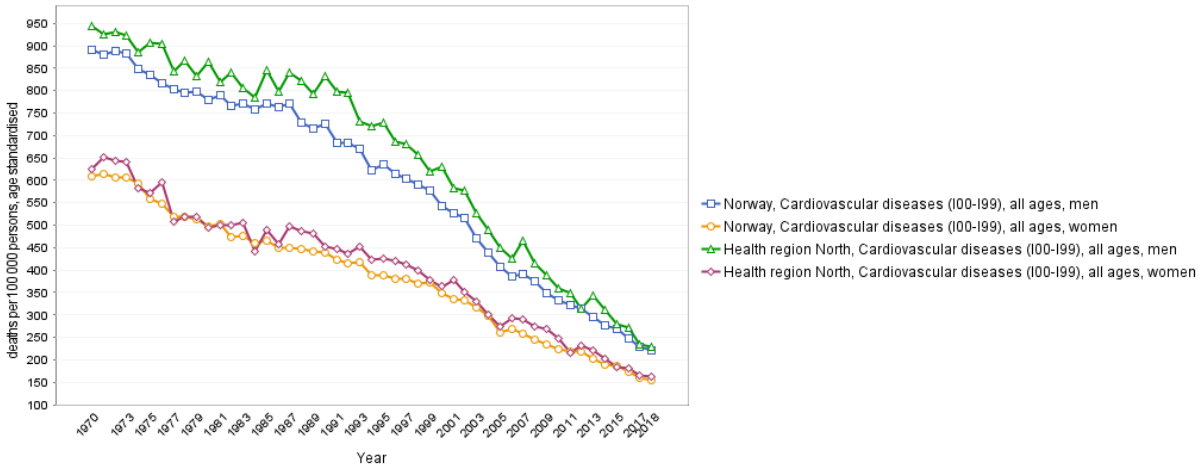

Figure 1. Age-standardised mortality rates per 100,000 of cardiovascular disease in men and women in Norway and in health region North (constituting the counties Finnmark and Troms, and Nordland) from 1970 to 2018. Source: Cause of Death Registry, Norwegian Institute of Public Health.

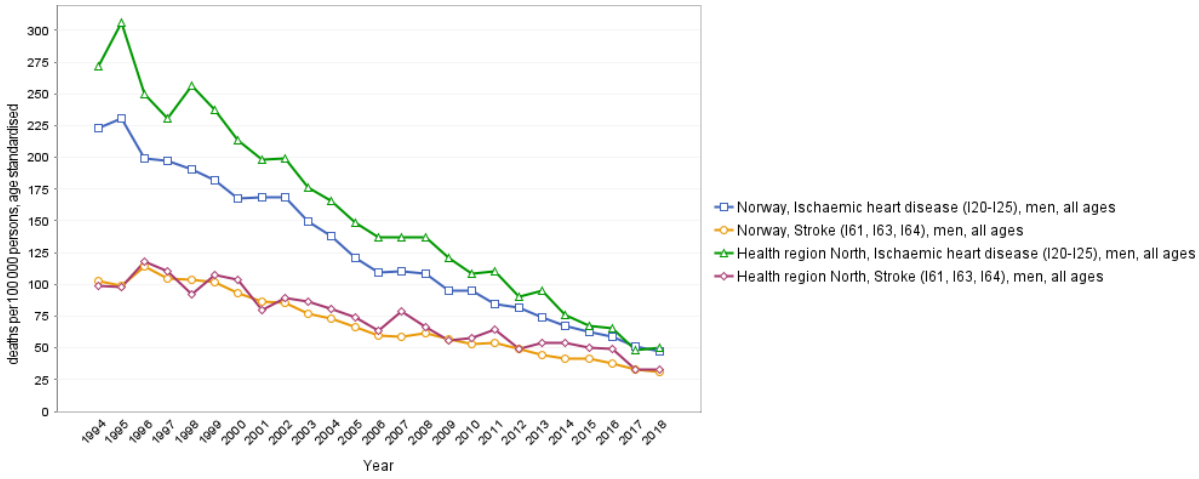

Figure 2. Age-standardised mortality rates per 100,000 of coronary heart disease and stroke in men in Norway and in health region North (constituting the counties Finnmark and Troms, and Nordland) from 1994 to 2018. Coronary heart disease include the International Statistical Classification of Diseases and Related Health Problems, the 10th Revision (ICD-10) codes: I20–I25, which corresponds to the definition used in this thesis. Strokes includes the ICD-10 codes: I61, I63, and I64, which is different from the definition of cerebral stroke in this thesis, as it does not include subarachnoid haemorrhage (ICD-10 code I60). Source: Cause of Death Registry, Norwegian Institute of Public Health.

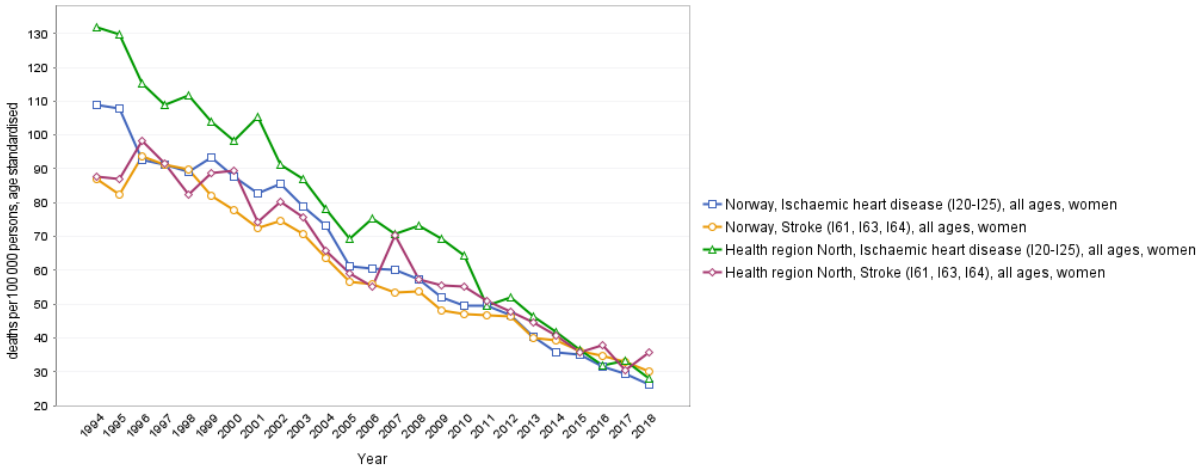

Figure 3. Age-standardised mortality rates per 100,000, of coronary heart disease and stroke in women in Norway and in health region North (constituting the counties Finnmark and Troms, and Nordland) from 1994 to 2018. Coronary heart diseases include the International Statistical Classification of Diseases and Related Health Problems, the 10th Revision (ICD-10) codes: I20–I25, which corresponds to the definition used in this thesis. Stroke includes the ICD-10 codes: I61,I63, and I64, which is different from the definition of cerebral stroke in this thesis, as it does not include subarachnoid haemorrhage (ICD-10 code I60). Source: Cause of Death Registry, Norwegian Institute of Public Health.

The aforementioned decline in mortality of CVD coincided with a decrease in conventional risk factors and an improvement in treatment, both medical and surgical. In 2001–2014, there was an annual decline in the incidence of AMI in those aged 25–85 years, which was attributable to a greater decline in CHD mortality than to AMI hospitalisations.[28] A similar decline was observed in hospitalisation rates of and mortality from CHD in 1995–2010 in the Tromsø Study, which is a population-based cohort study that includes the adult population of the municipality Tromsø in Northern Norway, and has been repeated seven times since 1974. [29] In the Tromsø Study, a favourable decline in modifiable risk factors accounted for 66% of the total decline in the incidence of CHD; total cholesterol, blood pressure, smoking, and physical inactivity contributed the most.[29] In 2001–2014, preliminary results in Norway suggested that there was a 20% decline in the age-standardised incidence rate of all CS in those aged >45 years, whereas an annual 2% increase in ischemic stroke (IS) and haemorrhagic stroke was observed in men aged 22–44 years and >85 years, respectively.[30] In 1995–2010 in the Tromsø Study,[31] an overall 24% absolute decline in the incidence of IS

was observed, and changes in modifiable risk factors accounted for 57% of this decline, wherein a decline in SBP and the prevalence of smoking contributed the most.[32]

Atherosclerosis

The underlying pathophysiology of CVD is complex. For CHD and many CS types, it is caused by atherosclerosis.[33, 34] Atherosclerosis is a chronic inflammatory condition, in which the arterial walls are stiffened and thickened due to a gradual accumulation of lipids and fibrous elements that creates a lesion known as plaque.[33] Atherosclerotic lesions lead to a narrowing of the arterial vessel lumen and a lower oxygen supply to the heart, brain, or extremities, which may cause an ischemic condition in the tissue. Atherosclerosis might be viewed as a biological response to an injury in the arterial endothelium, or as an endothelial dysfunction, to which the body reacts with complex mechanisms. Endothelial dysfunction leads to a destruction of the different layers in the vessel wall, which enables lipids and leucocytes to enter the sub-endothelium. This starts a cascade of reactions, including the formation of foam-cells (high content of lipids) and the migration of smooth muscle cells into the sub-endothelium, and the release of inflammation factors that further facilitates the passage of lipids and leucocytes into the sub-endothelium.[35] The damaged endothelia is coved by a fibrous cap that keeps the plaque content separate from the blood stream. New blood vessels supply the lesion with blood, but these immature vessels are fragile; if they rupture, bleeding will occur within the plaque and increase the size of the lesion, which can make it unstable. The death of macrophages within the plaque and necrosis of the plaque core can contribute to further instability.[35] The formation of plaque by atherosclerosis may take decades and may be accelerated by cycles of haemorrhage, erosion, or disruption of the plaque. Atherosclerosis can cause an acute condition if the plaque increases to the size of the arterial vessel lumen, due to erosion or rupture of the plaque that forms a thrombus (i.e., blood

clot) that flows with the blood stream and cause a blockage in small passages, or due to a bleeding in the plaque that activates thrombocytes and the coagulation system to create a blood clot that increases the plaque size (Figure 4).[36] Conventional risk factors such as cholesterol and smoking [37] initiate and contribute to the progression of injury or dysfunction. The associations between different conventional risk factors and CVD differ by CVD type. For example, total cholesterol seems to be more important in AMI than CS.[29, 32] However, the strength of association between a risk factor and CHD [38] or CS [39] is independent of whether these conditions lead to morbidity or mortality.

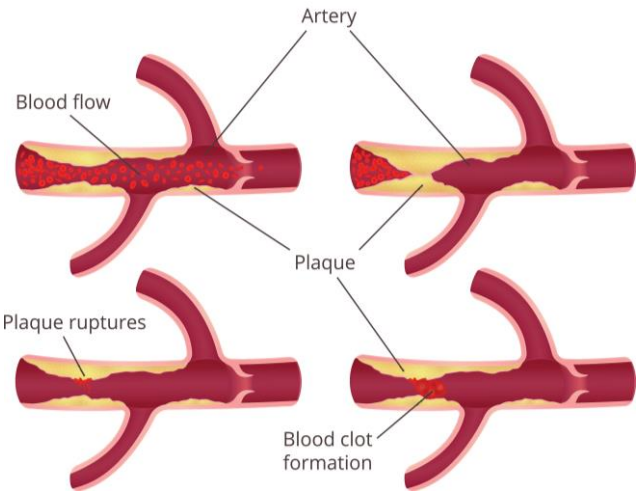

Figure 4. Atherosclerosis and acute ischemic event. Upper left, blood flow is reduced because of atherosclerosis, which forms a lesion known as plaque. The plaque causes a narrowing of the artery. Upper right: A complete densification of the artery, which causes an acute condition. Lower left: A rupture of a plaque, which creates a blood clot that enters the blood flow. The rupture also causes haemorrhage that enlarges the plaque. Lower right: a blood clot (due to erosion of a plaque) enters a small passage and cause a blockage, or the haemorrhage cause an enlargement of the plague that completely blocks the artery flow. Illustration: Colorbox

Coronary heart diseases

CHD is an umbrella term for diseases affecting the coronary arteries. The coronary arteries are the heart's own arteries; they supply heart muscle cells with blood and oxygen. The narrowing of the coronary arteries by atherosclerosis and subsequently plaque may be partial or complete and acute or chronic, giving rise to several coronary syndromes, such as angina pectoris and AMI. Angina pectoris is a condition caused by atherosclerosis in the coronary arteries and defined by episodic symptoms of myocardial ischemia (e.g. chest paint), often due to an increase in the heart's oxygen demand (e.g. walking uphill), but which subsides typically with rest or nitroglycerine administration and causes no permanent damage to the

tissue.[40] AMI is an acute, life-threatening condition caused by a sudden lack of oxygen supply to the heart muscle, most often due to plaque rupture and thrombus formation in one or several coronary arteries, causing tissue damage. CHD can be treated both medically (e.g. platelet inhibitors, statins, beta-blockers) and surgically (e.g. percutaneous coronary intervention, coronary artery bypass surgery), depending on the acuteness and severity of the condition.[41]

Cerebral stroke

CS is an umbrella term for cerebral conditions defined by lack of oxygen in the brain tissue, where the two main causes are thromboembolic ischemia (i.e. IS) and haemorrhage (i.e. haemorrhagic stroke). Among the different CS types, IS accounts for 80–85%. The rest are due to haemorrhagic strokes or bleedings,[42] which can either be intracerebral (10–12%) or subarachnoid (3%), depending on the location of the bleeding. Both bleedings and infarctions are acute conditions where the supply of oxygen to the brain is reduced, which causes a cellular hypoxia and ultimately, if left untreated, cell death. IS and haemorrhagic stroke have different aetiology. Haemorrhagic stroke occurs when there is a rupture of the vessel wall that causes bleeding, whereas IS is due to an occlusion of the arteries [34] as a consequence of atherosclerosis or formation of clot. As the aetiology of haemorrhagic and IS varies,[42] they have differing risk profiles and treatments. IS is most often a consequence of the atherosclerotic process and blood clot formation, with its conventional risk factors, whereas hypertension, aneurysms, and malformations are strong risk factors of haemorrhagic stroke.[34] The blood clot causing IS may be located in arteries within the brain or outside the brain. A blood clot may also travel with the blood stream up to the brain and cause blockage of the blood supply, causing ischemia. This is termed an embolus, and hence an embolic ischemic stroke. The embolus may be formed in arteries outside the brain or within the

chambers of the heart.[34] Atrial fibrillation is a common cause of clot formation in the heart, and is associated with a 2–5 fold increased risk of CS.[43] IS is treated with drugs that dissolve the blood clot or by platelet inhibitors; haemorrhagic stroke is an absolute contraindication of such treatment, and treatment relies mostly on hypertension control and supportive care.[34] Haemorrhagic strokes seems to have a higher mortality than ischemic strokes.[44]

1.2 Risk factors for cardiovascular disease

1.2.1 Modifiable risk factors

Lipids

Total cholesterol is positively associated with CHD incidence and mortality in both sexes, and this association decreases with age.[45, 46] An absolute reduction in total cholesterol is associated with a proportional decrease in the incidence of CHD, and this reduction may yield a larger decrease in incidence in younger than older adults.[46] Some studies have suggested that elevated total cholesterol is a more important risk factor for CHD [29] than for CS,[12, 45] whereas for CS, hypertension might be more important.[9, 47] However, the associations between total cholesterol and the risk of all CS types as a group might be less evident in studies because the aetiology of CS types differs.[48]

There is an inverse relationship between triglycerides and HDL cholesterol.[49] Low levels of HDL cholesterol is regarded as a risk factor for CVD, but how it is related to CVD remains uncertain.[49] The combination of low levels of HDL cholesterol and high levels of triglycerides are markers of a lipid profile (metabolic syndrome and insulin resistance) with an increased risk of CVD.[7, 8] The independent impact of triglycerides on CVD and their causal relationship is uncertain.[50, 51] Nevertheless, high levels of triglycerides are markers of

increased CVD risk, [51] because they are indicators of remnants that are rich in cholesterol, which contributes to plaque formation within the arterial endothelium.[50]

Blood pressure

Independent of age, SBP and DBP are positively associated with CHD and CS mortality, although different thresholds have been observed for when SBP or DBP starts to be positively associated.[52] Age interacts with blood pressure in fatal CS and all-cause mortality.[52] For example, in young adults (>19 years), elevated DBP seems to be a more important risk factor for CS mortality than elevated SBP, whereas with increasing age, SBP becomes more important than DBP.[52] With respect to the incidence of AMI, IS and CS, the risk increases with increasing SBP or DBP, although the association attenuates with increasing age.[47]

Hypertension

Hypertension is commonly defined as SBP ≥140 mmHg, DBP ≥90 mmHg, or current use of antihypertensive medications.[53] Hypertension is associated with an elevated risk of AMI, IS, and CS when compared to those with normal blood pressure,[47] and it is a more important risk factor for CS than for CHD.[9, 47] Use of antihypertensive medications that lower SBP by 10 mmHg is estimated to lower the risk of fatal or non-fatal CHD events by approximately 17%, CS by 27%, major CVD events by 20%, and all-cause mortality by 13%, irrespective of baseline SBP or comorbidities.[54] Although the goal of medical treatment of hypertension is often to achieve a SBP of <140 mmHg, some studies have suggested that the benefit or absolute risk reduction is larger if the target is <130 mmHg.[55] Also, the risk reduction in lowering SBP seems to be greater as the absolute risk for CVD increases, which implies that individuals at the highest risk (e.g. those with characteristics that are associated with elevated cardiovascular risk such as male sex, or diabetes) will benefit the most.[56]

Smoking

The risk of fatal and non-fatal AMI and most variants of CS is more than two-fold in current smokers compared to never smokers.[57, 58] Former smokers seem to have an elevated lifetime risk of CVD compared to never smokers, and this risk decreases with increasing number of years since cessation.[37, 39, 58] Second-hand smoking seems to increase the risk in a dose-dependent way,[59] where the risk rises with increasing exposure. Smoking is suggested to be more detrimental for women than men (RR=1.25 for women vs. men) in relation to fatal or non-fatal CHD.[60]

Obesity

Obesity is a condition of excess adipose tissue. Body mass index (BMI, a person's weight in kilograms divided by the square of height in meters) and waist circumference are measures of body fatness. General obesity is defined as BMI ≥30 kg/m^2,[61] whereas abdominal obesity is often defined as waist circumference >88 cm for women and >102 cm for men.[62, 63] Abdominal obesity is associated with an elevated risk of CHD and CS, but the association decreases with increasing age.[64] The effect of high body mass index on CHD and CS may be mediated by increases in total cholesterol, SBP, and glucose.[65] BMI or waist circumference do not seem to improve risk prediction when conventional risk factors are included in the risk models.[64]

Type 2 diabetes

Type 2 diabetes is a chronic lifestyle-related disease comprising peripheral tissue insulin resistance and progressively reduced insulin production in the pancreas, leading to increases in levels of blood glucose.[66] Type 2 diabetes is associated with end-organ damage in tissues in the heart, brain, kidney, nerves and retina, due to pathology in both large and small vessels.

Individuals with type 2 diabetes have an approximately 2-fold increase in the risk of CHD and CS compared to individuals without type 2 diabetes.[67] In Norway in 2009–2014, the incidence of type 2 diabetes declined by an average of 10.1% each year, whereas the prevalence have increased probably due to diagnosis at younger ages and increased longevity.[68] Type 2 diabetes is positively associated with obesity.[69]

1.2.2 Non-modifiable risk factors

Age and sex

Age is the single most important risk factor for CVD.[70] After the age of 55 years, for each 10-year increase in age, the incidence rate of CS more than doubles in both sexes.[70] Meta-analyses suggest that the risk of CS among men is 1.3 times that in women.[71] Data from the Global Burden of Disease Study showed that the incidence of IS was higher in men than in women in 2013, and the sex gap increased in 1990–2013 due to a deceasing trend of IS in women in this period.[72] Men also have a risk of AMI that is twice that in women, even after adjustment for risk factors that confound the relationship between sex and AMI. Still, the relative sex gap for AMI decreases with age.[73]

Family history of coronary heart disease

Family history of premature CHD or CS are risk factors for and predictors of future risk of CVD.[74] In this thesis, we focused on family history of premature CHD in first-degree relatives. The age at which CHD is considered premature varies by study and sex.[7, 75] According to the the Norwegian guidelines for the primary prevention of CVD, a family history of premature CHD is present if CHD occurred in a family member before the age of 60 years,[8] which is the threshold that we have applied in Paper 3. The risk of CHD or CS also seems to be dependent on the number of relatives affected, and it has been questioned

whether the increased risk is due to a genetic component or to shared lifestyles. A previous study have shown that the prevalence of CS was about four times higher in mono- than dizygotic twins,[76] which supports the hypothesis of a genetic component in the aetiology of CS.[42] In one study in Norway, self-reported history of premature AMI in one or both parents, or in one or more siblings was associated with elevated CHD mortality in men when compared to those with no family history.[77] In women, a history of premature AMI in siblings, or in both parents and siblings, was in that study associated with higher mortality of CHD.[77] The risk estimations were modestly attenuated when adjusting for conventional risk factors, and a subsequent adjustment for socioeconomic status had no impact.

Adult height

There are studies showing that height is inversely associated with the risk of CHD [78-81] and CS [78, 79] in both sexes; however, there are also studies that have found no association.[81, 82] Researchers have questioned whether the effect of height on CHD and CS is due to the association with certain genes or due to being a marker of unfavourable environment.

Approximately 60–90% [83, 84] of height is heritable, and the genetic contribution of height is the result of small contributions from many genes,[85] which is why it is a considered to be a polygenetic trait. Genetic studies have found a link between genes associated with adult height and genes that code for adverse lipid profiles,[80, 81] blood pressure [80] and hypertension,[78] and factors involved in the formation of atherosclerosis.[80] Others have suggested that the effect of height on CHD is mediated by lung capacity.[86] But despite this, height is considered to be a surrogate measure for environmental exposures like nutrition and diseases in childhood,[87, 88] socioeconomic conditions during childhood,[89] and conditions in foetal life.[90, 91] The average height in Europe increased from 1850 to 1980, most likely due to

improvements in environmental factors.[92] The average height of Norwegian conscripts increased from 1900 to 1980, but conscripts from Northern Norway have consistently had a lower average height than conscripts in Mid and Southern Norway.[93, 94] These studies suggest that, with respect to population height, environmental factors might be more important than genetics, as the average population height has increased in accordance with improvement in living conditions. It seems that genetics determine the *potential* for height in an individual, whereas environment factors determine the actual *attained* height.

1.3 Risk models

Risk models for predicting future risk of CVD have been developed because the risk of CVD is multifactorial and not only dependent on single risk factors, and each risk factor contribute to risk with different weights.[95] The levels of single risk factors might be rather low, but the cumulative contribution of several risk factors might put an individual at high absolute risk. The Norwegian guidelines for the primary prevention of CVD [8] recommend using the NORRISK 2 risk model to estimate the absolute 10-year risk of fatal or non-fatal AMI or CS combined. NORRISK 2 is a Norwegian risk model,[12] available at http://hjerterisiko.helsedirektoratet.no/. It estimates the absolute 10-year risk as a probability (percentage) based on sex, age, and the following conventional risk factors HDL cholesterol, total cholesterol, SBP, use of antihypertensive medications, smoking status, and history of CHD before the age of 60 in one or two family members.[12]

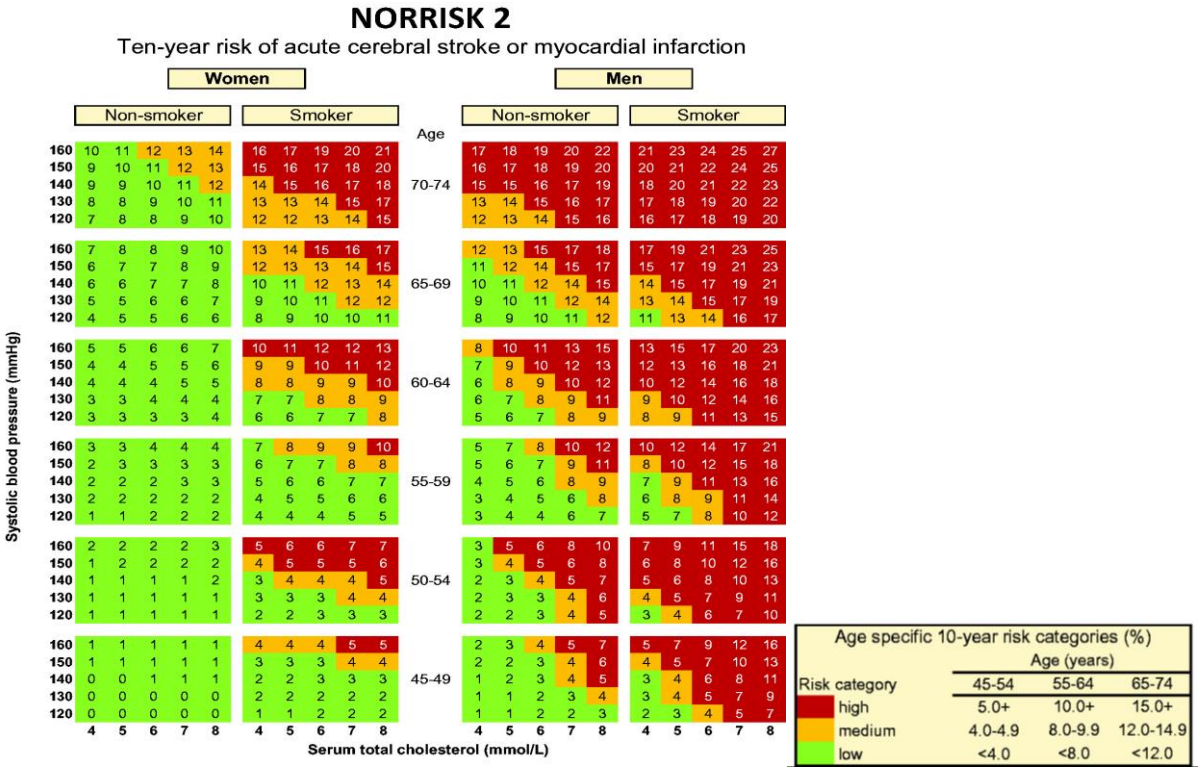

Figure 5. The NORRISK 2 risk chart. The chart show 10-year risk of acute myocardial infarction or cerebral stroke in percentages, in men and women with high-density lipoprotein (HDL)-cholesterol >1.0 mmol/L and 1.3 mmol/L, respectively, currently not taking antihypertensive medication, and with no family history of premature CHD (i.e., occurring before the age of 60 years).[12] Republished with permission of SAGE Publications, from Selmer R, Igland J, Ariansen I, Tverdal A, Njølstad I, Furu K, et al. NORRISK 2: A Norwegian risk model for acute cerebral stroke and myocardial infarction. Eur J Prev Cardiol 2017; 24: 773-782; permission conveyed through Copyright Clearance Center.

The intention of the risk model is to identify individuals at high 10-year risk who need to lower their risk by initiating use of medications or lifestyle changes.[12] The risk model identifies individuals at high risk defined according to the following age-specific thresholds: ≥5% risk in the age group 45–54 years, ≥10% risk in the age group 55–64 years, and ≥15% risk in the age groups 65–74 years.[12] Thus, the model is a tool for medical doctors to help them evaluate when to start an intervention. The risk chart (Figure 5) can also help medical doctors to convey information to patients about the benefits from risk reduction from for example smoking cessation.

Briefly, NORRISK 2 is designed by using information from the Cardiovascular Disease in Norway (CVDNOR) project.[12] The project have collected baseline information from population-based studies (questionnaires, clinical examinations and blood samples) conducted in Norway in 1994–1999. Participants' records were then linked to national hospital records and to the Cause of Death Registry for information on main or secondary discharge diagnoses and underlying cause of death, respectively.[96] To the development of NORRISK 2, participants aged 40–79 years that were free of angina pectoris, AMI, or CS, were followed from 1994 to 2009 for first occurrence of AMI or CS, which included either hospitalisations (non-fatal cases) or deaths (fatal cases). Fatal or non-fatal AMI was defined by the ICD-10 codes I20–I25 (all CHD) and I21–I22 (AMI and subsequent myocardial infarction), respectively. Fatal or non-fatal CS was defined by the ICD-10 codes I60–61 (subarachnoid and intracerebral haemorrhage) and I63–64 (cerebral infarction and unspecified stroke), except I63.6 (cerebral infarction due to cerebral venous thrombosis, non-pyogenic). NORRISK 2 was designed using the cumulative incidence function, which takes into consideration competing risks, in this case, deaths from other causes. NORRISK 2 was validated using regional health surveys from Norway, carried out in 2000–2003. The validation showed overall good agreement between the predicted and observed 10-year risk of AMI or CS, although somewhat poorer agreement with increasing age, possibly due to an increase of comorbidities with age.[12]

There are country and region-specific risk models that estimate the absolute 5- or 10-year risk of fatal or non-fatal CVD, or both, and models that are disease-specific or predict a composite endpoint. The risk models are based on national-specific CVD mortality and morbidity rates, sex and age distributions, and the prevalence of conventional risk factors.[12, 97-101] The risk models may differ in which age ranges they are applicable to, and in which additional risk

factors are included, such as HDL cholesterol, diabetes and family history of premature CHD.[12, 97, 98, 101] There are some conditions listed in the guidelines that elevate the estimated risk, and these should be taken into account: South-Asian ethnicity, rheumatoid arthritis, abdominal obesity, psychosocial stress, and depression/medically-treated psychotic disorders.[7, 8] South-Asian ethnicity is associated with a different phenotype for fat distribution that makes them more susceptible to insulin resistance and type 2 diabetes at lower BMI levels than other ethnic groups.[102] This, in turn, puts them at increased risk of CVD[103-105] and is the reason why their 10-year risk estimation has been suggested to be multiplied by 1.5. For rheumatoid arthritis, it is recommended to multiply the estimated 10-year risk by 1.4, whereas for the other conditions, no such factors are specified.[7, 8]

1.4 Cardiovascular disease in Sami compared to non-Sami

In this overview on the risk of CVD in Sami compared to their non-Sami reference population, I have included studies from Norway, Sweden, and Finland. Incidence refers to the first occurrence, whether the outcome is hospitalisation or death, thus studies including both outcomes are of relevance.

Acute myocardial infarction and coronary heart disease in Sami

A rather similar incidence of AMI have been observed in Sami and non-Sami populations in the previous Finnmark County (per date merged with Troms County into Troms and Finnmark County) in 1974/75–1989.[23] Using individual data from the previous Finnmark County collected in roughly the same period, Sami men had lower mortality rates for CHD, CVD, and somewhat lower for total deaths than non-Sami men in adjusted models, whereas similar risks were observed in women.[24] A study in 1970–1989 linked census data to Cause of Death Registry in Norway and found a somewhat higher CHD, CVD and total mortality in

Sami than in their non-Sami reference population.[25] An assumed higher consumption of reindeer meat was associated with lower CHD mortality in Sami men and CVD mortality in Sami women in Northern Norway in 1970–1998.[25]

In Sweden, a sample of Sami was constructed by linkage to registries, and individuals were followed in 1961–2000 [106] and in 1985–2002.[107] Similar incidence of AMI have been observed in Sami and their demographical matched reference population in Sweden in 1985–2002,[107] but a higher CHD mortality was observed in Sami women in Sweden in 1961–2000, except in reindeer herding women,[106, 107] who had lower incidence of AMI than their demographical matched reference population in 1985–2002.[107] Sami men had similar CHD mortality as their demographically-matched reference population.[106, 107] Sami in Finland had lower CHD mortality in 1961–1990 compared to a district representing the Finnish population,[108] whereas in 1974–2005, Sami men had similar and Sami women lower CHD mortality [109] than their non-Sami counterparts.

Cerebral stroke in Sami

A similar,[26] or possibly higher incidence of CS was found in Sami when compared to non-Sami living in the previous Finnmark County in 1974–1989,[23] and a higher incidence of CS was observed in men with a mixed Sami and Finnish background.[26] These studies were stratified into sex and ethnic groups, resulting in groups of small numbers and consequently low precision in the estimates. In Northern Norway, Sami men and women had higher CS mortality in 1970–1989 than their demographical matched reference population.[25] A trend of lower CS mortality was observed in Sami men as their assumed consumption of reindeer meat increased. A lower CVD mortality trend was observed in Sami women and men with an assumed higher consumption of reindeer meat.[25] In Sweden in 1961–2002, the incidence of

CS and subarachnoid haemorrhage among Sami was overall higher than in their demographically-matched reference population.[107] Mortality of CS in Sami and their non-Sami counterpart was found to be similar in Sweden in 1985–2002 [107] and Finland in 1979–2005,[109] whereas the mortality of subarachnoid haemorrhage were similar [107] or higher [106] in Sami in Sweden compared to their demographically-matched reference population in 1961–2002. However, lower incidence of CS was observed in Swedish Sami men with associations to reindeer herding compared with their demographically-matched reference population in 1985–2002.[107] Sami women in Sweden with an association to reindeer herding had a higher incidence of CS and subarachnoid haemorrhage in 1985–2002,[107] and had higher mortality of CS compared to the Swedish population in 1961–1985.[110] In Sweden in 1961–2000, CVD mortality was overall higher in Sami than non-Sami women.[106]

Incidence of acute myocardial infarction or cerebral stroke in Sami

The studies on risk of CHD and CS from Finland and Sweden are of more recent years compared to the studies conducted in Norway, which might makes them more relevant. It may be acceptable to compare incidence and mortality in Sami in Finland and Sweden with that in Norway, as all the countries have universal access to healthcare and higher education. Also, in Norway and Sweden, reindeer herding is a culturally specific trade engaged by only Sami individuals, which suggests that reindeer herding is a reliable marker of Sami ethnicity in these countries.[111] Moreover, all countries have their own Sami Parliaments, but only Norway has ratified the ILO Convention No.169 that imposes obligations and expectations on the government in terms of protection of indigenous rights.[112] Despite these similarities, careful comparisons are warranted as the studies vary by design, time-period, and study setting (different countries), and because Sami ethnicity is defined differently in these studies.

Also, the relative risks were small and some studies have low precision, which overall makes it uncertain if the risk of AMI and CS differ by ethnicity.

1.5 Risk factors in Sami compared to non-Sami

In this overview of conventional risk factors in Sami, we have included studies from Norway and Finland (only men), and one from Sweden. The studies compare risk factors in Sami to their non-Sami reference populations that live either in the same municipality or in a region close by. The associations between non-modifiable risk factors, such as age and sex, and CVD most likely do not differ by ethnicity; however, studies concerning family history of CHD and adult height have been included, as there has been speculation as to whether Sami are protected from CVD through their diet [113] or genetically.[24]

Lipids

Overall similar levels of triglycerides, HDL cholesterol, and LDL cholesterol, with the exception of some small differences in single studies, have been observed in Sami and non-Sami men in Northern Norway, in Sweden, and in Finland.[19, 113-117] In the SAMINOR 1 Survey (SAMINOR 1), which was conducted in 2003–2004 and included individuals aged 30 and 36–79 years, small differences in lipids that varied by age were observed. Sami men and women aged 36–49 years had higher mean levels of total cholesterol than Norwegians, whereas Sami women aged 65–79 years had lower mean levels than their non-Sami counterparts.[19] Sami women aged 36–49 years also had the highest mean levels of triglycerides, and Sami women aged 50–64 years had lowest mean levels of HDL cholesterol compared to Norwegians and Kven.[19] Use of lipid-lowering medications was similar in Sami and non-Sami in SAMINOR 1.[118]

Blood pressure and hypertension

In a study from previous Finnmark County carried out in the 1970s, Sami men aged 20–49 years had a statistically significantly lower SBP than non-Sami,[119] whereas no differences were observed in women. Later studies, including SAMINOR 1, have found a similar DBP, and a similar or somewhat lower SBP in Sami compared with non-Sami.[19, 116, 117] In the study from Finnmark County carried out in 1974–1975, use of antihypertensive medications was higher in Sami than in Finnish and Norwegian women, whereas the opposite was observed in men.[23]

Smoking

Some studies have observed no differences in smoking habits between Sami and non-Sami,[114, 116, 117] whereas others have observed that Sami women smoke less than non-Sami women.[19, 23]

Obesity

In SAMINOR 1, Sami women who reported that they themselves, both parents and all four grandparents spoke Sami at home had more often a BMI ≥30 kg/m^2, a waist circumference ≥88 cm, and a waist-to-hip ratio >0.85 than Norwegians, whereas fewer Sami than Norwegians men had a waist circumference ≥102 cm.[17] Other studies have also observed a tendency towards a higher prevalence of obesity in Sami women.[23, 114]

Type 2 diabetes

Similar prevalence of type 2 diabetes were observed in Sami and non-Sami in both SAMINOR 1 and the SAMINOR 2 Clinical Survey (SAMINOR 2), carried out in 2012–2014. when diabetes was defined based on self-report or random plasma glucose with cut-off of 7.5

mmol/L or above,[120] and when combining self-reported diabetes status and medication use (SAMINOR 1).[121] When using glycated haemoglobin with a cut-off of 6.5% in SAMINOR 2, a higher prevalence was observed in Sami than non-Sami, and waist-to-height ratio explained this in women.[20]

Family history of coronary heart diseases

A doctoral thesis used information collected in the previous Finnmark County in 1974–1988, and suggested that Sami and non-Sami populations had similar proportions of family members that had history of AMI and angina pectoris.[122] Later, a study including part of the same population, found a lower proportions of Sami with a family history of CHD.[114] In SAMINOR 1 [118] and in Sweden,[117] a somewhat higher proportion of Sami had a family history of AMI and CVD, respectively, when compared to their non-Sami counterparts.

Adult height

Studies have observed a lower average height in Sami across Norway, Sweden, and Finland when compared to their non-Sami reference populations.[17, 24, 116, 117] A possible inverse gradient has been observed between height and degree of Sami affiliation, where those with a strong Sami language connection (reporting Sami as home language for themselves, both parents and all four grandparents) had the lowest height.[17] In a study from 1974 including the population in the previous Finnmark County, height was inversely associated with CS, even after adjusting for other factors.[26]

Modifiable and non-modifiable risk factors for cardiovascular disease in Sami

The differences in the modifiable and non-modifiable risk factors listed above are small and caution must be applied when considering these results, as they are few, have small sample

sizes, are geographically specific, and define Sami ethnicity differently. The somewhat higher obesity level in Sami women and the lower stature in Sami seem to be a consistent observation.

1.6 Ethnicity as a determinant of cardiovascular disease

Ethnicity is a socially constructed variable based on shared characteristics like language, religion, traditions, diet, ancestry, and common history and origin;[2, 123] it is considered multi-dimensional and complex[124, 125] and is closely related to the concept of culture.[126] These markers of ethnicity might be considered as distinct to each ethnic group, and may be associated with different health outcomes. Ethnicity is commonly included as a study variable in epidemiological research, where the categorisation of ethnicity has been criticised for being imprecise, and self-identification for being fluid.[125] Ethnicity in epidemiology is not viewed as a risk factor in itself, but a marker for membership to a group with shared characteristics that may be associated with a given disease.[123, 125] That said, an ethnic category is not biologically based in contrast to the term race. The term race gives a notion of genetic, biological or physical differences that have previously been used to rank some people according to races or physical appearance,[127] which also the Sami and Kven populations were exposed to.[128-130] The terms race and ethnicity vary by place and time, and have been used interchangeable in social sciences.[131] The term race has, however, limited scientific values,[131, 132] as there are limited genetic differences between populations. In recent time, it has become more common to use the term ethnicity than race in epidemiology.[133]

Social determinants of health

In order to have an idea of how ethnicity can be related to health, conceptual diagrams for social determinants of health (Figure 6) such as the one suggested by Brunner and

Marmot,[134] might be applied. Shortly, social determinants of health constitutes the conditions or circumstances that people are born into, live in, and grow and age in, and the systems that deal with ill health.[135] Brunner and Marmot suggest that factors above and at the individual level determine health. Factors above the individual level are social structures, such as material factors, work, and the social environment. They influence health behaviours and individual psychological factors like stress. However, individual health is also determined by early life experiences, as well as genetic and cultural factors throughout one's life. Finally, the social structures vary by location and historical circumstances.[134]

Brunner and Marmot included culture in their conceptual diagram. Kohrt, Hadley and Hruschka describe culture to include a set of '*beliefs, values, norms and behaviour that are transmitted through social learning*'.[136] Cultural factors influences the way we do things and way of thinking, and in a broader perspective the ethnic-specific lifestyle of individuals. As for ethnicity,[125] the concept of culture as an explanatory factor for ethnic differences in health has been criticised for being crude and simplistic with regards to intra-group heterogeneity, and possible inaccurate as researchers often lack a concrete model for how culture influence health outcomes.[126, 137] Historical factors that may have influenced the ethnic groups differently. In the context of Sami and non-Sami, the governmental assimilation process in 1850–1960 known as 'norwegianisation' were directed towards the Sami and Kven [138] and had political and social consequences [139] in relation to use of Sami language and self-determination to own and use geographical regions. Moreover, the Second World War might have inflicted the ethnic populations or geographical regions differently.[140]

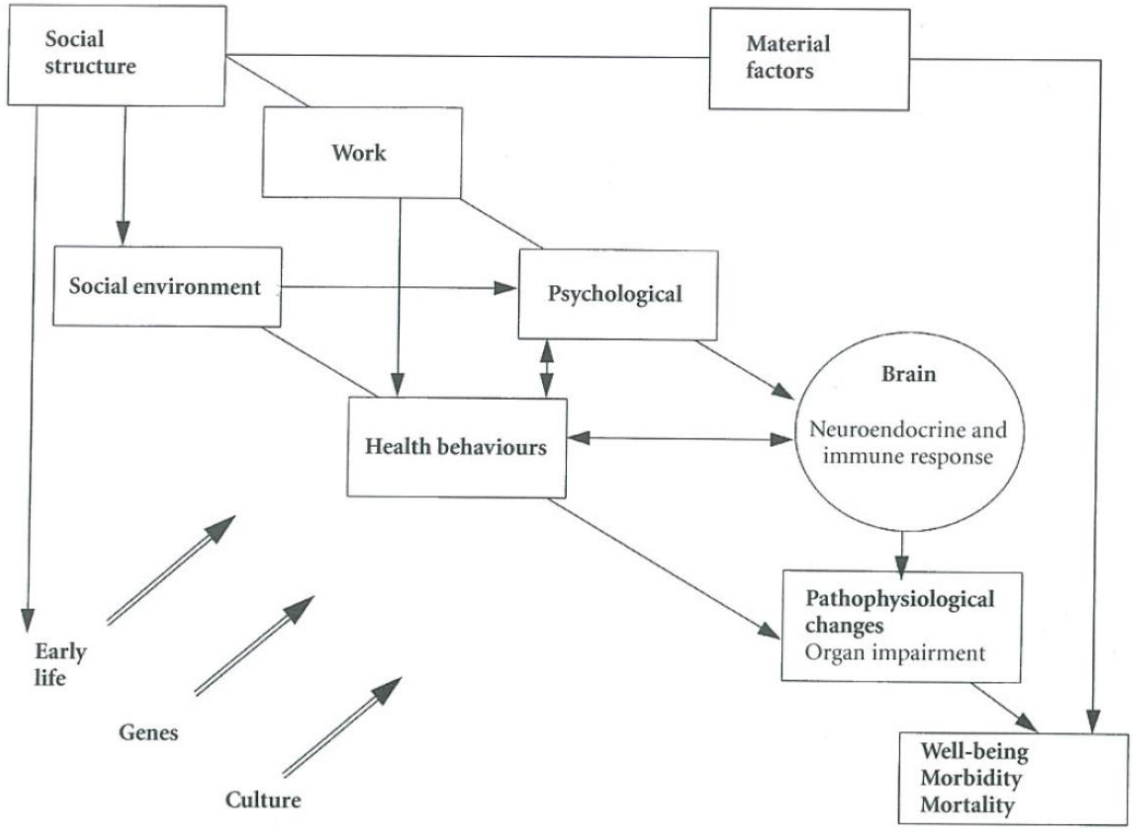

Figure 6. Social determinants of health.[134] Republished with permission from Oxford University Press, from Brunner E and Marmot M. Social determinants of health. 2nd. ed In: Marmot M and Wilkinson RG (eds.) Oxford: Oxford University Press, 2006. Chapter 2, Social organizations, stress, and health; p.6-30.; reproduced with permission of the Licensor through PLSclear.

In recent times, the revitalisation and establishing of Sami institutions, such as Sami University College, and the Sami Parliament might have strengthened the local Sami communities by improving the social structures and social environment in term of political acknowledgement and Sami self-determination.[141]

Ethnicity as a determinant of cardiovascular diseases in Arctic indigenous populations

Due to the lower CHD mortality observed in Sami men in Norway [24] and Finland,[113] particularly in with those with an affiliation to reindeer herding,[25] researchers have hypothesised that Sami people were protected from CHD due to their diet [113] or by their genetics.[24] However, the latter has not been supported by studies on the incidence of AMI

and CS in Sami.[23, 25] Also, a similar aetiology of these conditions is assumed in Sami and non-Sami, as the relative risks changed in similar directions for the incidence of CS and AMI when adjusting for the same variables.[23] There is only one study that have found genetic variations in Sami with association to lipid metabolism and consequently CVD. This study analysed allele frequencies of apolipoprotein A-IV (a protein associated with lipid metabolism) in 71 Sami vs. 177 Finns in northern Finland in 1989, and found a somewhat higher allele frequency of apolipoprotein A-IV-2 in Sami.[142] The Sami with this phenotype had higher HDL cholesterol levels than Finns with this phenotype, suggesting an effect of the lipoprotein allele on HDL cholesterol in Sami but not in Finns. However, overall levels of lipids did not differ statistically significantly between the groups.[142] In the 1950s to mid-1970s, lower CHD mortality was observed in the Greenland Inuit population compared to the population in Denmark, and it was suggested that the Inuit populations were protected against CHD either genetically,[143] or by their traditional diet, which had high levels of polyunsaturated fatty acids.[144] However, a subsequent systematic comparison of risk in Arctic Inuit populations showed a similar or lower incidence of and mortality from CHD and CVD compared to their reference populations,[145] which challenged the assumed protective effect of diet and differences in genetic susceptibility.[146] One recent study indicated a possible increased risk of type 2 diabetes in the Greenlandic Inuit population.[147] Moreover, the Greenland Inuit population has been found to be genetically distinct from other European and East Asian populations, possibly due to their isolation on an island, a generally low population size, and periods of high mortality (bottlenecks).[148] This is most likely not the case for Sami, although this has been suggested by some.[149] Sami in Norway have had close interaction with the Kven and Norwegians over centuries, which may have resulted in more genetic variation compared to the Greenland Inuit population.

It has also been suggested that the transition that indigenous populations have made from a traditional lifestyle to a modern or westernised lifestyle, have made them more susceptible for lifestyle diseases such as CVD, type 2 diabetes, obesity, and social, physical, and mental disorders.[150] Reindeer herding is assumed to represents a more traditional lifestyle with a different dietary pattern,[151] with an assumed higher intake of reindeer meat,[25] other nutrients (alpha-tocopherol, albumin and selenium) that may be protective against CHD,[113] and higher level of physical activity.[107] On the contrary, one study in Mid Sweden compared dietary habits to reindeer herding Sami, non-Sami and historical Sami in 1930s–50s (the latter by interview of decedents), and suggests that the traditional Sami diet is the diet consumed by present-day reindeer herding Sami and resembles the diet to historical Sami. The traditional Sami diet was suggested to consists of high amounts of meat, fatty fish, blood and organ dishes, berries, boiled coffee and high in total fat, and low amounts of vegetables, bread and fibres,[152] which is in contrast to the nutritional recommendation of low fat content and consumption of fibres and vegetables to prevent CVD.[7] However, reindeer herding Sami had a higher physical activity level during work than non-herding Sami and non-Sami.[152] In a Swedish non-Sami population, the traditional Sami diet was associated with a small non-beneficial effect on CVD mortality.[153] In SAMINOR 2, Sami ethnicity and inland residency rather than coastal residency has been associated with higher consumption of reindeer meat,[154] but no ethnic differences were observed in nutritional intake that were likely to give differences in health outcomes.[155] Others have suggested that Sami might be more susceptible to chronic lifestyle diseases due to ethnic discrimination,[156] acculturation processes,[157] and due to being more marginalised and possible chronic stress that accompanies this.[22]

1.7 Aim of the thesis

Due to the mentioned shortcomings in the studies on conventional risk factors and the risk of AMI or CS in Sami, there is a need for an update. A simplified conceptual framework for this thesis is illustrated in Figure 7. The main exposure is Sami ethnicity, which is assumed to be associated with a unique language, religion, traditions, diet, ancestry, and common history and origin, and a unique set of values, believes, and behaviour. These characteristics may translate to a different lifestyle that influence the risk of CVD through conventional risk factors and height. As such, adult height and conventional risk factors are intermediate factors between the lifestyle and risk of CVD.[158] These intermediate factors can be used to estimate the risk of CVD, and potentially to explain ethnic differences in risk.

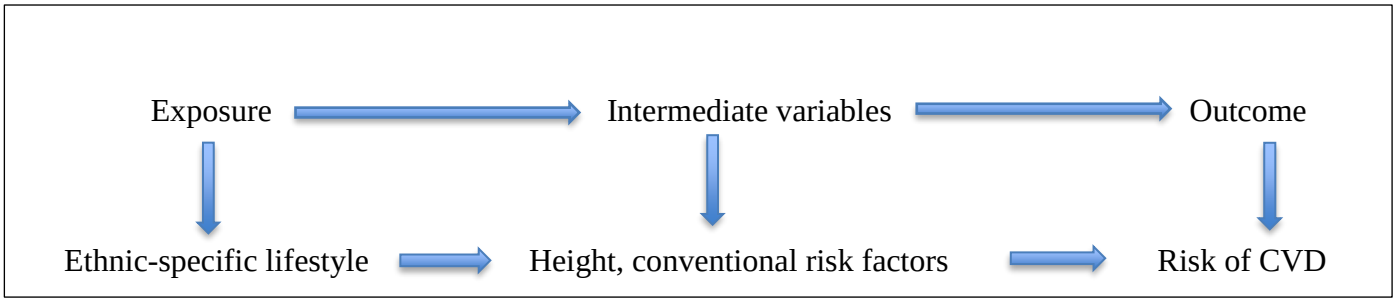

Figure 7. Conceptual framework for the thesis. Abbreviations: CVD: cardiovascular disease

The specific aims of this thesis were:

- To explore whether individual conventional risk factors and the estimated 10-year cardiovascular risk based on multiple conventional risk factors (the NORRISK 2 model) are similar in Sami and non-Sami populations.
- To estimate changes in conventional risk factors between two time-points and investigate whether these changes differ by ethnicity.
- To determine and compare the incidence of AMI, CHD, CS, IS, and a composite endpoint in Sami and non-Sami populations.
- If ethnic differences are observed, to identify intermediate factors that may explain differences in risks.

2 Methods

2.1 The SAMINOR Study

The SAMINOR Study (the Population-based Study on Health and Living Conditions in Regions with Sami and Norwegian Populations) is run by the Centre for Sami Heath Research at UiT the Arctic University of Norway. The study consists of three cross-sectional surveys: the SAMINOR 1 Survey (SAMINOR 1) conducted in 2003–2004,[159] the SAMINOR 2 Questionnaire Survey from 2012, and the SAMINOR 2 Clinical Survey (SAMINOR 2) conducted in 2012–2014.[160] Paper 1 used information from SAMINOR 2, Paper 2 used information from SAMINOR 1 and SAMINOR 2, and Paper 3 is a follow-up of participants in SAMINOR 1.

SAMINOR 1

All individuals aged 30 and 36–78/79 from 24 municipalities (in six municipalities, only the population in selected districts were invited), were invited (n=27,987) (see footnotes to Table 3, and www.saminor.no). In total, 16,865 completed at least one of three questionnaires (initial, main, or additional questionnaire) or attended the clinical examinations, which gave a response rate of 60.3%.[159] The sampling design of SAMINOR 1 changed after it was completed in the municipalities of Kautokeino, Karasjok, Tana, and Nesseby. In these municipalities, invitees received an initial questionnaire by mail. They could chose to participate in the study by completing the initial questionnaire only, or in addition, agree to be invited to the clinical examination. Those who chose to be invited to the clinical examination received their appointment information by mail, along with the main questionnaire. This two-stage invitation procedure led to low attendance to clinical examinations, as only people who had returned the initial questionnaire were invited to the clinical examination. Therefore, in

these four municipalities, all individuals were invited to the second round of clinical examinations, regardless of their response on the initial questionnaire. As a result, some attended the clinical examination without completing the initial questionnaire, which included questions on ethnic affiliation. In 2006, the initial questionnaire was mailed to those that participated in the clinical examination without completing the initial questionnaire, increasing the number of completed initial questionnaires. In the subsequent municipalities, the initial and main questionnaires were merged (hereafter referred to as screening questionnaire), and mailed to the invitees together with a scheduled time for clinical examination. At the clinical examination, participants handed in the screening questionnaire and completed the additional questionnaire. Four weeks after the examination, participants received the results of their clinical measurements. If abnormalities were present, participants were recommended to contact their local primary physician. The questionnaires, informational brochures, the consent form, and the reminder note (Appendix A), were prepared in Norwegian and translated into Northern Sami (available at www.saminor.no). The Northern Sami version of the questionnaires was available in the municipalities of Kautokeino, Karasjok, Porsanger, Tana, Nesseby, Lyngen, and Kåfjord, but 98% of all participants completed the Norwegian version. In Finnmark and Troms County, non-responders were offered a second chance to participate in the study, which led to a higher participation rate in Finnmark and Troms County compared to the counties of Nordland and Trøndelag.

SAMINOR 2

In SAMINOR 2, invitations were sent to all inhabitants aged 40–79 years residing in 10 municipalities (n=12,455) (see footnotes to Tables 1 and 2, and available at www.saminor.no) of the counties of Finnmark and Troms, and Nordland. Four weeks before clinical data

collection started, invitees received a pamphlet (Appendix B), with information on the study in Norwegian and in most municipalities also Northern Sami. In the municipalities of Porsanger and Storfjord, invitees also received information in the Kven language, whereas in the municipalities of Skånland and Evenes, invitees received only the Norwegian version. Questionnaires and informational brochures were sent (Appendix B), along with a scheduled date for the clinical examination, about 2 weeks before the data collection started in each municipality. The questionnaire and informational brochure were prepared in Norwegian and translated into Northern Sami. Residents of Porsanger and Storfjord received the informational brochure in all three languages; in Skånland and Evenes it was distributed only in Norwegian. All participants received the Norwegian version of the questionnaire. The Northern Sami translation was sent to participants in the municipalities of Kautokeino, Karasjok, Tana, and Nesseby. For the invitees in the municipalities of Kåfjord, Storfjord, and Porsanger, the Sami version was available upon request. Participants returned the questionnaire at the clinical examination. Less than 5% of all the participants used the Sami version of the questionnaires. When the collection was at the halfway point, a reminder was posted to the invitees who had not yet participated. In total of 6,004 agreed to participate, completed the questionnaires and attended the clinical examination, which gave a response rate of 48.2%.[160]

2.2 Clinical examination

The clinical examinations are described in detail in the papers. Briefly, in both surveys, the clinical examination included measures of blood pressure, weight, height, and waist circumference. Triglycerides, HDL cholesterol, total cholesterol, glucose, and glycated haemoglobin (the latter only in SAMINOR 2) were measured in non-fasting blood samples. In SAMINOR 1, the clinical examinations took place in buses placed at central locations in the

municipalities for 1–18 weeks, depending on the population size of the municipality (in the municipalities included in Paper 2 buses were placed for 1–6 weeks), and were conducted by trained personnel from the National Health Screening Service. In SAMINOR 2, rooms were rented in central locations in each municipality, and the clinical examinations were conducted within 2–7 weeks. Certified health workers primarily from the local community, were employed as temporarily fieldworkers and trained to perform the clinical examinations.

2.3 Ethnicity: main exposure

There is no registry of Sami ethnicity in Norway, as information about ethnicity is regarded as sensitive,[161] and it is prohibited to include in official registries. Therefore, in health surveys in Norway, information on ethnicity is usually obtained through self-reporting. Sami ethnicity was categorised similarly in Papers 1–3. Questions related to ethnicity (in total 11) were phrased identically in SAMINOR 1 and 2, with multiple options (Norwegian, Kven, Sami or 'other', specify) that could be ticked for each question. We used nine of the 11 questions related to ethnicity for our categorisations. To be categorised as Sami, two criteria had to be met: Self-definition as a Sami in addition to a Sami language connection. That is, participants had to 1) report either Sami ethnic background for themselves, or 2) consider themselves to be Sami. In addition, 3) Sami had to be spoken at home by the respondent him/herself, 4–5) by one of his/her parents, or 6–9) by one of his/her grandparents. Two additional question related to ethnicity, 10) ethnic background to father and 11) mother were not included in the main analyses, but used in sensitivity analyses regarding ethnic categorisation. All those that did not fulfil the two criteria for Sami ethnicity were categorised as non-Sami. That included: participants who reported Kven, Norwegian, or 'other' as their ethnic background, and considered themselves as any of these; and participants who reported Sami on some of the questions, but did not fulfil both criteria for Sami ethnicity. Moreover, those who were categorised as Sami may have reported multiple ethnic backgrounds or self-perceived

ethnicities, such as Kven, Norwegian, or other in addition to Sami. Participants with missing information on ethnicity were excluded in the analytical sample of Paper 1–3.

2.4 Paper 1

2.4.1 Study sample

The 10-year risk of fatal or non-fatal AMI or CS was estimated for participants in SAMINOR 2. The 10-year risk was based on sex, age, and the conventional risk factors included in the NORRISK 2 (HDL cholesterol, total cholesterol, SPB, use of antihypertensive medication, and smoking status) except family history of premature CHD, which was not available in SAMINOR 2. Figure 8 shows who were excluded from the analytical sample, and Table 1 shows some sample characteristics in those invited, in those who participated in clinical examination, and in final analytical sample. South-Asian ethnicity was not considered specifically in Paper 1, however, less than 6% of the study sample reported that they considered themselves as 'other' in addition to Sami, Kven or Norwegian. Thus, South-Asian ethnicity most likely does not influence the estimations in any of the ethnic groups. Additionally, rheumatoid arthritis, depression/medically-treated psychotic disorders, and psychosocial stress were not considered specifically in Paper 1.

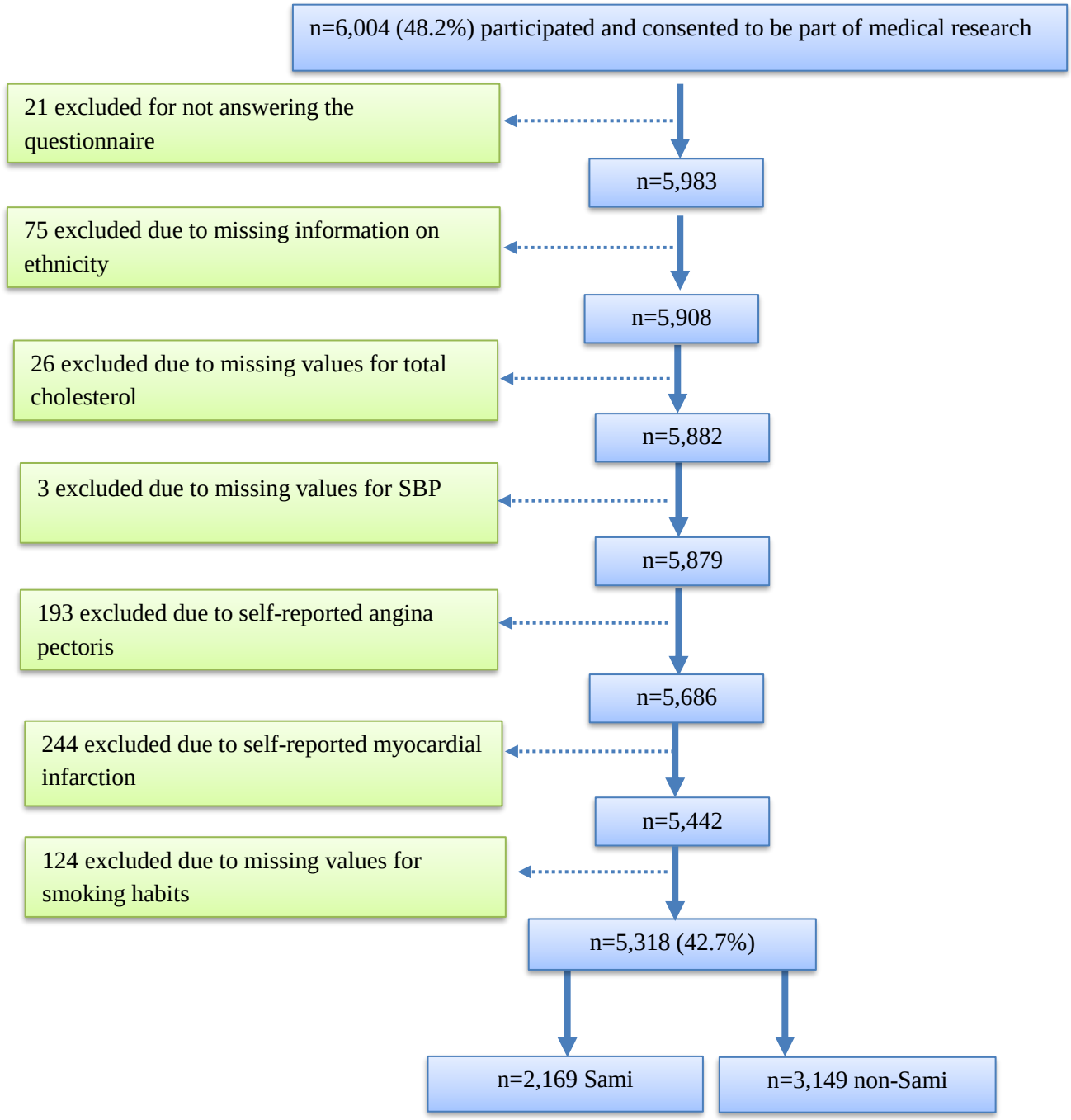

Figure 8. Flow chart for Paper 1 showing exclusions from the SAMINOR 2 sample. Abbreviations n: number, SBP: systolic blood pressure.

2.4.2 Sample characteristics

Table 1. Selected characteristics (frequency and percentages) of invitees, those who attended the clinical examination, and of the analytical sample of Paper 1.

	Invited	Clinical examination	Analytical sample
Total, (%)	12,455	6,004 (48)	5,318 (43)
Sex			
Men	6,469 (52)	2,747 (46)	2,346 (44)
Women	5,986 (48)	3,257 (54)	2,972 (56)
Age			
40–54 years	5,065 (41)	2,015 (34)	1,912 (36)
55–64 years	3,587 (29)	1,869 (31)	1,694 (32)
65–79 years	3,803 (30)	2,120 (35)	1,712 (32)
Region of residence			
Region 1†	2,616 (21)	1,289 (21)	1,138 (21)
Region 2†	4,034 (32)	2,011 (34)	1,793 (34)
Region 3†	5,805 (47)	2,704 (45)	2,387 (45)
Duration of education			
0–9 years		1,729 (29)	1,432 (27)
10–12 years		1,681 (28)	1,511 (28)
≥13 years		2,321 (39)	2,170 (41)
Missing		273 (4)	205 (4)
Ethnicity			
Sami		2,409 (40)	2,170 (41)
Non-Sami		3,499 (58)	3,148 (59)
Missing		96 (2)	0
Marital status			
Married		3,364 (56)	3,026 (57)
Cohabitant		850 (14)	791 (15)
Divorced		545 (9)	483 (9)
Unmarried		708 (12)	642 (12)
Widow(er)		385 (6)	325 (6)
Missing values		152 (3)	51 (1)

†Municipalities included in Region 1, Kautokeino, and Karasjok; Region 2, Tana, Nesseby, and Porsanger; Region 3, Skånland, Evenes, Storfjord, Lyngen, and Kåfjord.

2.4.3 Statistical analyses

Differences in crude sample characteristics between Sami and non-Sami were tested with two-sample t-test and Pearson's chi-square tests. Ethnic differences in ordered groups were tested with Wilcoxon's rank sum test. Sex- and age-specific, high-risk thresholds suggested by Selmer et al.[12] were used to identify individuals at high 10-year risk. The means and proportions in Sami versus non-Sami within these strata were compared using the two-sample t-test and Fisher's exact test, respectively. The overall crude mean 10-year risks in Sami and non-Sami were compared with the two-sample t-test, and proportions with Pearson's chi-square test. We used the direct method to age-standardise the overall means and proportions

according to the invited sex-specific SAMINOR 2 population, using 5-years intervals. When comparing the mean age-standardised 10-year risks by two-sample t-tests, we used the standard deviation estimated from the standard population, which increased the precision and lowered the p-values. The age-standardised proportions were tested with two-sample test of proportions.

2.5 Paper 2

2.5.1 Study sample

In Paper 2, we estimated changes in conventional risk factors, and in the 10-year risk of fatal or non-fatal AMI or CS from SAMINOR 1 to SAMINOR 2. The 10-year risk was based on sex, age, and the conventional risk factors included in NORRISK 2 (HDL cholesterol, total cholesterol, SPB, use of antihypertensive medication, and smoking status) except family history of premature CHD, which was not available in SAMINOR 2. Some SAMINOR 1 participants also participated in SAMINOR 2, and the samples were linked for the 16,778 SAMINOR 1 participants who accepted to have their information linked to other studies. To achieve a sample that was comparable to SAMINOR 2 with regard to residency (10 municipalities) and age (40–79 years), we excluded 9,513 participants from SAMINOR 1. In SAMINOR 2, all those who had attended the clinical examination (n=6,004) consented to linkage. From the combined sample (n=13,269, Figure 9), we excluded SAMINOR 1 participants who did not attend the clinical examination or did not complete the screening questionnaire. If information on ethnicity was missing in one of the surveys, we used information from the other survey. Those with missing information on ethnicity in both surveys, were excluded. The analytical sample consisted of 6,417 participants from SAMINOR 1 and 5,956 participants from SAMINOR 2 (Table 2), of whom 50.6% in SAMINOR 1 also participated in SAMINOR 2, constituting 54.6% of the SAMINOR 2

sample. The reproducibility of ethnicity by Cohen's kappa suggested a high agreement (0.93, p<0.001) [162] of Sami ethnicity from SAMINOR 1 to 2 among those that participated twice (n=3,723). In SAMINOR 1 and 2, 41% (n=1,538) and 42% (n=1,565) of participants were Sami, respectively.

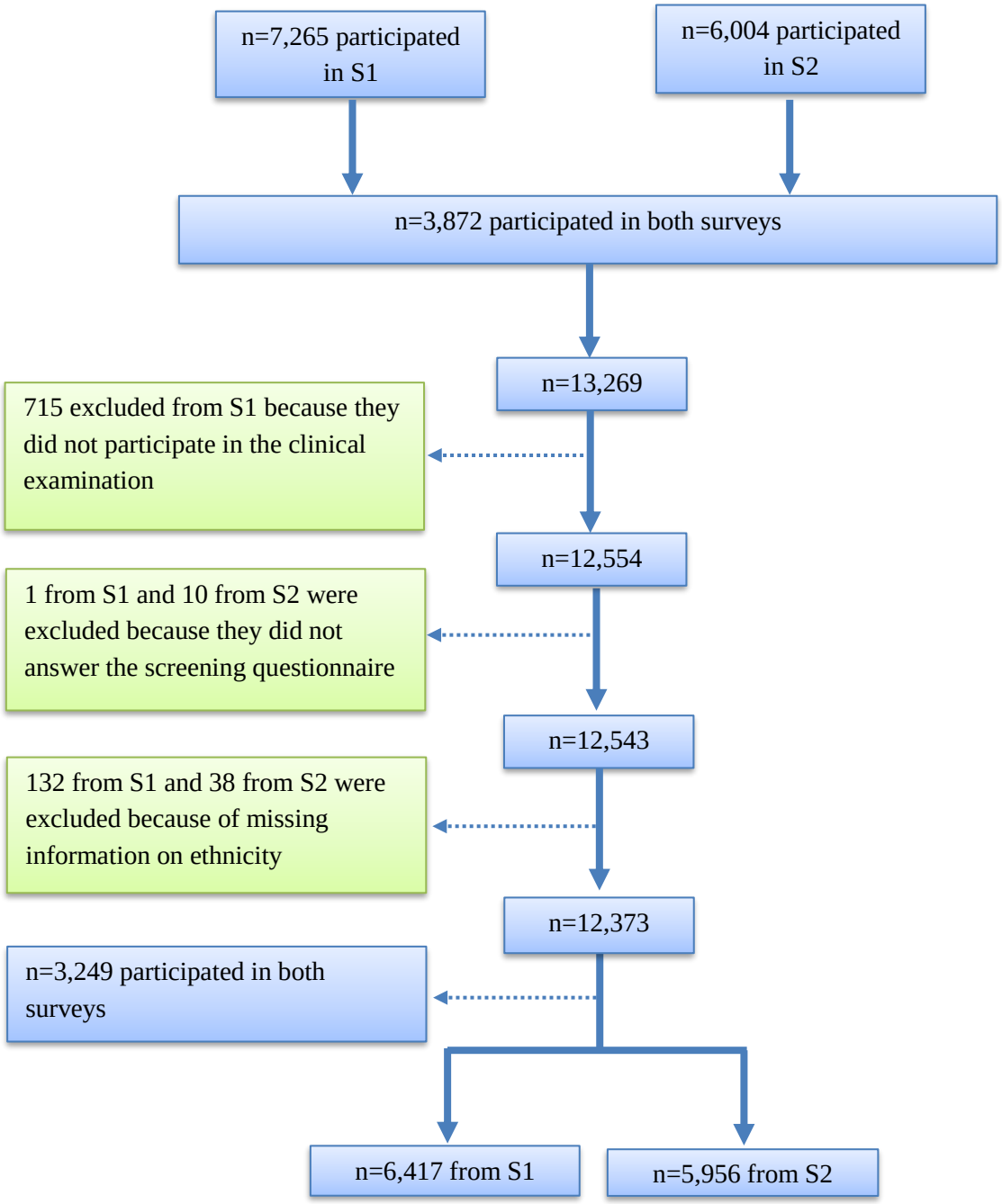

Figure 9. Flow chart for Paper 2 showing exclusions from the SAMINOR 1 and SAMINOR 2 sample. Abbreviations; S1: SAMINOR 1; S2: SAMINOR 2, n: number.

2.5.2 Sample characteristics

Table 2. Selected characteristics (frequency and percentages) of invitees, those who attended the clinical examination, and the analytical sample of Paper 2.

	SAMINOR 1			SAMINOR 2			Participated twice	
	Invited	Clin. exam.	Analy. sample	Invited	Clin. exam.	Analy. sample	SAMINOR 1	SAMINOR 2
Total	11,518	6,550 (57)	6,417 (56)	12,455	6,004 (48)	5,956 (48)	3,249	
Sex								
Men	5,987 (52)	3,089 (47)	3,027 (47)	6,469 (52)	2,747 (46)	2,722 (46)	1,432 (44)	
Women	5,531 (48)	3,461 (53)	3,390 (53)	5,986 (48)	3,257 (54)	3,234 (54)	1,817 (56)	
Age								
40–54 years	5,529 (48)	3,086 (47)	3,034 (47)	5,065 (41)	2,015 (34)	1,990 (33)	1,747 (54)	447 (14)
55–64 years	3,176 (28)	1,921 (29)	1,893 (30)	3,587 (29)	1,869 (31)	1,853 (31)	1,160 (36)	1,289 (40)
65–79 years	2,813 (24)	1,543 (24)	1,490 (23)	3,803 (30)	2,120 (35)	2,113 (36)	342 (10)	1,513 (46)
Region of residence								
Region 1†	2,348 (20)	1,224 (18)	1,118 (17)	2,616 (21)	1,289 (21)	1,282 (21)	654 (20)	654 (20)
Region 2†	3,704 (32)	2,067 (32)	2,051 (32)	4,034 (32)	2,011 (34)	2,002 (34)	1,038 (32)	1,036 (32)
Region 3†	5,466 (48)	3,259 (50)	3,248 (51)	5,805 (47)	2,704 (45)	2,672 (45)	1,557 (48)	1,559 (48)
Duration of education								
0–9 years		2,606 (40)	2,533 (39)		1,729 (29)	1,722 (29)	1,131 (35)	1,145 (35)
10–12 years		1,768 (27)	1,744 (27)		1,681 (28)	1,677 (28)	1,043 (32)	971 (30)
≥13 years		1,730 (26)	1,710 (27)		2,321 (39)	2, 310 (39)	1,058 (32)	1,116 (34)
Missing		446 (7)	430 (7)		273 (4)	247 (4)	17 (1)	17 (1)
Ethnicity								
Sami		2,281 (35)	2,343* (37)		2,410 (40)	2,430** (41)	1,318 (41)	
Non-Sami		4,067 (62)	4,074* (63)		3,498 (58)	3,526** (59)	1,931 (59)	
Missing		202 (3)	0		96 (2)	0		
Marital status ‡								
Single/unmarried	2,597 (22)	1,171 (18)	1,145 (18)		708 (12)	714 (12)	542 (17)	305 (9)
Married	6,537 (57)	4,128 (63)	4,047 (63)		3364 (56)	3,391 (57)	2,192 (68)	2,026 (62)
Cohabitant					850 (14)	856 (15)		310 (10)
Widow(er)	897 (8)	488 (7)	468 (7)		385 (6)	389 (6)	145 (4)	290 (9)
Divorced	1,264 (11)	656 (10)	652 (10)		545 (9)	549 (9)	310 (10)	285 (9)
Separated	222 (2)	107 (2)	105 (2)				60 (1)	
Missing values	1 (0)	0	0		152 (3)	57 (1)	0	33 (1)

* Included imputed values from SAMINOR 2/**from SAMINOR 1. †Municipalities included in Region 1: Kautokeino, and Karasjok; Region 2: Tana, Nesseby, and Porsanger; Region 3: Skånland, Evenes, Storfjord, Lyngen, and Kåfjord. From SAMINOR 1 to 2, two moved from Region 1 to Region 3; seven moved from Region 2 to Region 1; five moved from Region 1 to Region 2. ‡Marital status in SAMINOR 2 is self-reported; in SAMINOR 1 it is retrieved from Statistics Norway; married and same-sex partnerships were merged. Abbreviations; Clin. exam.: clinical examination; Analy. sample: analytical sample

2.5.3 Statistical analyses

We assessed if the change in conventional risk factors and 10-year risk [163] from SAMINOR 1 to 2 differed in the ethnic groups. We tested this statistically by including an interaction term between survey and ethnicity using linear and logistic generalised estimating equations by Liang and Zeger.[164, 165] The generalised estimating equations give population-averaged estimates [165] and corrects for dependence between observations by adding a term for within-subject correlations. We used the exchangeable correlation matrix that assumes a similar correlation for the within-subject observations.[166] Generalised estimating equation analyses assume that missing values are missing completely at random,[167] i.e., that the pattern of missing values is independent of both observed and unobserved data.

2.6 Paper 3

2.6.1 Study sample

To estimate the risk of the specified CVD endpoints, we used baseline data SAMINOR 1. Among the invitees, 16,865 participated, which gave a participation rate of 60.3%. Among these, 1,149 individuals were excluded because they did not consent to linkage, did not complete the screening questionnaire, or did not attend the clinical examination, which gave a sample of 15,716 (Table 3). Moreover, we excluded those with missing information on ethnicity, those with self-reported myocardial infarction, angina pectoris or CS, and those that had a hospital discharge diagnosis of CHD or CS prior to participation in SAMINOR 1. To get a complete case sample with regard to the variables that might explain potential ethnic differences in risk: ethnicity, adult height, and conventional risk factors included in NORRISK 2, we excluded all participants with missing values for these variables, which gave a final sample size of 13,787 (Figure 10, Table 3).

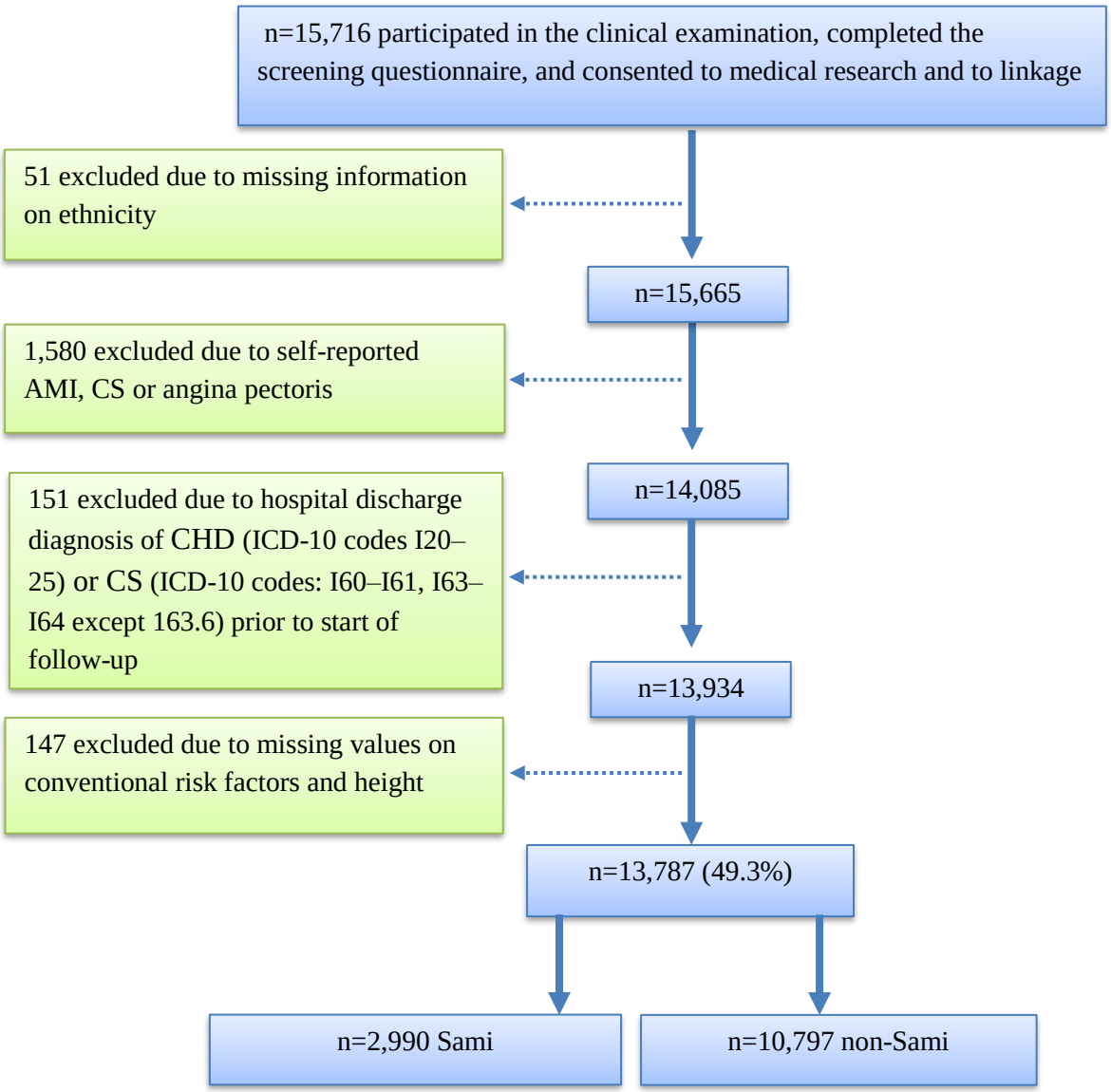

Figure 10. Flow chart for Paper 3 showing exclusions from the SAMINOR 1 sample. Abbreviations: n: number; AMI: acute myocardial infarction; CHD: coronary heart disease; CS: cerebral stroke: ICD-10: International Statistical Classification of Diseases and Related Health Problems, the 10th revision.

2.6.2 Sample characteristics

Table 3. Selected characteristics (frequency and percentages) of invitees, those who attended clinical examination, completed the screening questionnaire, and consented to medical research and linkage, and the analytical sample of Paper 3.

	Invited	Clinical examination, screening questionnaire, consent to medical research and linkage	Analytical sample
Total	27,987	15,716 (56)	13,787 (49)
Sex			
Men	14,541 (52)	7,523 (48)	6,379 (46)
Women	13,446 (48)	8,193 (52)	7,408 (54)
Age			
30, 36–39 years	4,028 (14)	1,740 (11)	1,702 (12)
40–54 years	11,500 (41)	6,558 (42)	6,257 (45)
55–64 years	6,590 (24)	4,123 (26)	3,537 (26)
65–79 years	5,869 (21)	3,295 (21)	2,291 (17)
Duration of education			
0–9 years		5,472 (35)	4,370 (32)
10–12 years		4,432 (28)	4,077 (30)
≥13 years		4,848 (31)	4,622 (33)
Missing		962 (6)	718 (5)
Ethnicity			
Sami		3,456 (22)	2,990 (22)
Non-Sami		12,209 (78)	10,797 (78)
Missing		51 (1)	0
Marital status*			
Single	7,057 (25)	3,066 (20)	2,819 (21)
Married	15,394 (55)	9,769 (62)	8,565 (62)
Widow(er)	1,826 (7)	989 (6)	749 (5)
Divorced	3,071 (11)	1,596 (10)	1,385 (10)
Separated	638 (2)	296 (2)	269 (2)
Missing values	1	0	0

*Marital status retrieved from Statistics Norway: married and same-sex partnerships were merged.

2.6.3 Linkage to national registries

The dates and discharge diagnosis to non-fatal cases of AMI, CHD, IS and CS were retrieved from CVDNOR and Norwegian Patient Registry for the periods 1993–2009 and 2010–2016, respectively. Both main and secondary discharge diagnosis were retrieved, and the first discharge diagnosis including one of the specified endpoints were used in the analyses. CVDNOR project is run by the University of Bergen together with the Norwegian Knowledge Centre for Health Services.[96] The CVDNOR have collected information of all hospitalisations with CVD (ICD-9 codes 390–459; ICD-10 codes I00–I99) or diabetes as main or secondary discharge diagnosis. This hospitals information is retrieved from the Patient Administrative System,[168] which seems to be of relative good quality as 99% of the

patients records on AMI, CS, and hip fractions had correct information on time, date of admission, and main diagnosis.[169] The CVDNOR has a 7-year look-back period starting in 1994–2001, which ensures that the incident cases in Paper 3 have no previous hospitalisations with the same discharge diagnosis.[170] The CVDNOR have observed few missing values for municipality and age, and have corrected for overlapping and multiple hospitalisations within 24 hours.[168] Death within 28 days after a hospitalisation was considered to be part of the hospitalisation.[96]

The Norwegian Patient Registry is used for reimbursement and management purposes, and have a high level of completeness, which indicates that all the necessary data have been registered.[171] Similar to CVDNOR, the patient information is retrieved from hospitals' Patient Administrative System.

The dates and underlying cause of deaths were retrieved from Cause of Death Registry for 2003 to 2016. The performance to the Cause of Death Registry has been compared with other national registries using an index based on six measures of quality, and Norway scored 87.6 out of the 100 points possible; Hungary obtained the highest score at 95.7 points.[172] Due to extensive use of non-meaningful or unspecified codes, the Norwegian Cause of Death Registry had a low index score.[173] Dates of emigration were retrieved from Statistics Norway for 2003–2016. The personal identification number that are assigned to all citizens in Norway enables a complete and unique linkage to different national registries.

2.6.4 Statistical analyses

Cox proportional hazards regression was used to explore whether the incidence of AMI, CHD, IS, CS, and a composite endpoint consisting of AMI or CS differed between Sami and

non-Sami. We ran separate models for each outcome, and adjusted for sex and age. Intermediate variables that could explain ethnic differences were included in a temporal order [174] and chosen based on previous studies of the Finnmark population that showed that height was inversely associated with CS and possibly also with AMI,[23, 26] and the conventional risk factors included in NORRISK 2 model (HDL cholesterol, total cholesterol, SPB, use of antihypertensive medication, smoking status, family history of premature CHD).[12] Participants were followed from the time of enrolment in SAMNOR 1, until occurrence of an event (fatal or non-fatal), moved out of the predefined municipalities, death from other causes, or end of follow-up (December 31st 2016). Attained age was used as the time-scale in the Cox models. Age at entry is preferable to time-on study as the time-scale variable when age is a strong risk factor for the outcome and covariates.[175] Intermediate variables were treated as covariates. Covariates that violated the proportional hazards assumption were included as time-varying covariates. The assumption in Cox regression is that censoring is independent, which means that the probability of being censored at any time does not depend on a participant's prognosis for failure at any given time.[176] Studies have suggested that Sami are at higher risk of violent death [25] and death from external causes [106] than non-Sami, which might hinders the events of interest from occurring,[177] leading to fewer person-years and possibly an underestimation of risk in Sami when comparing their risk to non-Sami. Therefore, we performed sensitivity analyses to account for competing events using the Fine and Gray cumulative incidence function.[178] The results from Fine and Gray analyses (results not shown) were similar to those from the Cox regression, indicating that we did not have issues with differences in mortality from other causes.[179]

2.7 Ethics

SAMINOR 1 and 2 are part of the SAMINOR Study, which has been approved by the Norwegian Data Protection Authority (02/01525-4), and by the Regional Committee for Medical and Health Research Ethics for region North (REC North) (2011/1840). This project has been accepted by REC North (2015/2204) and by the SAMINOR Project Board. All participants were invited by mail, volunteered to participate, and could withdraw from the studies at any time. Those included in the analytical samples of Papers 1–3 have given their written informed consent to medical research. In Papers 2 and 3, those that were included in the analytical sample also consented to have their information linked to registries. All statistical analyses were done using de-identified data files. For Paper 3, Statistics Norway linked SAMINOR 1 with emigration data, data from CVDNOR, the Norwegian Patient Registry, and the Cause of Death Registry. The complete data file was stored, and all the statistical analyses were conducted on a secure platform for sensitive data at the University of Oslo.

According to international guidelines, racial and ethnic minorities are considered as vulnerable populations.[180] This calls for special awareness when conducting research, so as not to inflict any harm. Vulnerability in the Sami context is suggested to be related to the preservation efforts that Sami communities have to make in order to maintain their existing culture in terms of public and governmental acknowledgment and acceptance to have an educational system that strengthen the use of the Sami languages, to have property rights, to have cultural diversity and avoiding stereotyping, and reliable statistics, and most of all, the acceptance of being Sami.[181] Interpreting vulnerability as such, our work may strengthen Sami communities, as we provide valuable health statistics on the Sami population.

3 Main results

3.1 Paper 1

In our analytical sample from SAMINOR 2, Sami men were somewhat younger than non-Sami men. Sami men had higher unadjusted mean levels of lipids, and a larger proportion had elevated levels of these than non-Sami men. No ethnic differences were observed in blood pressure, use of antihypertensive medication, or smoking status when comparing crude levels in men. Sami women were a bit younger than non-Sami women. Sami women had somewhat unfavourable levels of triglycerides, HDL cholesterol, and a larger waist circumference when comparing crude mean levels with non-Sami women. No ethnic differences were observed in total cholesterol, blood pressure, and similar proportions of Sami and non-Sami women used antihypertensive medication and were smokers.

The small ethnic differences in single risk factors were not reflected in the 10-year risk of AMI or CS in Sami and non-Sami men and women when using the NORRISK 2 model. The crude, mean 10-year risk and age-standardised estimates were similar in non-Sami and Sami men and women. In the age group 55–64 years, more Sami than non-Sami (36.1% vs. 26.7%, p-value 0.006) men had an elevated (>10% risk) 10-year risk. Overall, a similar proportion of non-Sami and Sami men and women were at high 10-year risk, and this did not change when comparing age-standardised proportions.

3.2 Paper 2

In our analysis of changes in conventional risk factors and 10-year risk of AMI or CS in Sami and non-Sami from SAMINOR 1 to SAMINOR 2, we observed that total cholesterol declined in women (by -0.50 mmol/l, 95% confidence interval (CI) -0.54, -0.45). Sami men had a

statistically significantly smaller decline (-0.43 mmol/l, 95% CI -0.51, -0.35) than non-Sami men (-0.60 mmol/l, 95% CI -0.66, -0.53). Small and probably negligible ethnic differences were observed in men for changes in triglycerides and HDL cholesterol, whereas women had similar changes in these variables. SBP and DBP declined by -3.1 (95% CI -3.89, -2.27) and -0.7 (95% CI -1.20, -0.28) mmHg in men, and -3.6 (95% CI -4.36, -2.88) and -1.0 (95% CI -1.39, -0.57) mmHg in women, respectively. Men were more likely to be using antihypertensive medications in SAMINOR 2 than in SAMINOR 1 (odds ratio 1.17, 95% CI 1.06, 1.31), but the likelihood of having hypertension did not change. In women, use of antihypertensive medications did not change, but the likelihood of having hypertension declined by 6.2 percentage points (odds ratio 0.77, 95% CI 0.70, 0.85). The likelihood of being a daily smoker was lower in SAMINOR 2 than in SAMINOR 1; in men, the odds ratio was 0.54 (95% CI 0.49, 0.60) and in women it was 0.65 (95% CI 0.59, 0.71), corresponding to a decline of 11.3 and 8.2 percentage points, respectively. Waist circumference increased in men by 5.9 cm (95% CI 5.50, 6.31), and in women by 6.7 cm (95% CI 6.24, 7.70). The 10-year risk decreased for similarly in men, whereas in women, Sami women had a somewhat smaller decline comped to non-Sami women.

3.3 Paper 3

Our investigation of the incidence of some CVD among Sami and non-Sami showed that Sami ethnicity was associated with a 36% (hazard ratio 1.36, 95% CI 1.10, 1.68) and 31% (hazard ratio 1.31, 95% CI 1.08, 1.58) increased risk of IS and CS, respectively, after adjustment for sex and age. Low adult height explained a considerable part of the higher risk of IS and CS in Sami, as this variable attenuated the hazard ratio to 1.26 (95% CI 1.00, 1.59) and 1.18 (95% CI 0.96, 1.44), respectively. Further adjustment for conventional risk factors changed the hazard ratio for IS and CS only slightly. The sex- and age-adjusted hazard ratio

indicated similar risks in Sami and non-Sami for AMI (hazard ratio 0.99, 95% CI 0.83, 1.17), CHD (hazard ratio 1.03, 95% CI 0.93, 1.15), and for the composite endpoint (hazard ratio 1.09, 95% CI 0.95, 1.24). When accounting for differences in height, the hazard ratio declined slightly for AMI, CHD, and the composite endpoint, and subsequent adjustment for conventional risk factors increased the hazard ratio slightly.

4 Discussion of methods

4.1 Study design used in Papers 1–3

We used a cross-sectional design in Paper 1, repeated cross-sectional design in Paper 2, and a prospective cohort design in Paper 3, which are all observational studies. A cross-sectional study includes a specific population in a specific place at one time-point, which gives an opportunity to observe the frequency of diseases and the distribution of factors that may cause disease. A prospective cohort study includes individuals at a specific time-point and follows them over time, usually to observe whether an event of interest occurs. Thus, compared to cohort studies, cross-sectional studies are less time-consuming and cost less to conduct, as there is no follow-up.[182] All observational designs enables the study of associations, but only prospective cohort studies enables researchers to ensure that the exposure comes before the effect, also known as temporality. In a cross-sectional study, it is more difficult to distinguish whether the outcome or the exposure came first, as both are observed at the same time.[183] Temporality is one of the Bradford Hill criteria`s used for determining if an association from an observational study might be causal, but it is not enough to claim causality.[184] Thus, cross-sectional studies are considered to be descriptive and suitable for estimation of prevalences and useful for generating hypotheses, whereas prospective cohort studies are more analytical, i.e., suggesting causation.

The prospective design in Paper 3 made it possible to exclude participants with CVD prior to follow-up, thus we avoided issues with temporal bias or reverse causality.[183] Temporal bias occurs when people with a disease, for example AMI, adopt a heathier lifestyle and improve their levels of total cholesterol as a consequence of the AMI event. This leads researchers to falsely underestimate the associations between cholesterol and AMI.[183] We used hospital discharge diagnoses from the CVDNOR and the Norwegian Patient Registry, in addition to

self-reported diagnoses, to exclude individuals with CVD prior to follow-up. Although we used both self-reported and registry information, there might have been prevalent cases of CVD that were counted as new cases. However, as both ethnic groups parish to the same hospitals and the hospitals most likely apply similar discharge codes to the same CVD independent of the patient`s ethnic belonging, we believe that the prevalent CVD cases were excluded in a similar way in both ethnic groups.

4.2 Internal validity

The validity of a study consists of internal and external validity, and the latter is dependent on the former. Internal validity describes how accurate and precise, or how true, the results are for the target population.[185] Therefore, an estimate has to be both accurate and precise to be valid. Internal validity requires the absence of random and systematic errors; the latter is known as bias and tends to give erroneous results.[183] Random errors do not necessarily give incorrect results, but they lead to variation that affects the precision and reproducibility of results, particularly when sample sizes are small.[183] Systematic errors in studies can occur at the design stage, during the collection of information, or during the process of data analysis.[183] Systematic errors that influence the groups compared to a similar extent, independent of other factors,[182] create a non-differential bias that, in most situations, underestimates the strength of the associations between the exposure and outcome.[182] Systematic errors that affect the comparison groups unequally creates differential bias, which either under- or overestimates the strength of the associations.

Observational studies are prone to errors, but it is difficult to know to what extent. Biases are often categorised into three main types: selection bias, information bias, and confounding,[182, 183, 186] although confounding is sometimes considered to be different from bias. The biases

can be related to each other and might overlap. Due to the possibility of bias and confounding, the results from observational studies need to be interpreted critically and cautiously.

4.2.1 Information bias

Information bias is also known as observation, classification, or measurement bias. These biases occur due to an incorrect measurement or classification of an outcome, an exposure, or both.[185] Standardisation in data collection can prevent information bias. In this thesis, the clinical examination, including blood sample, was standardised by following strict protocols and using trained personnel to conduct the examinations. We are not aware of any systematic biases that have influenced the clinical measurements. Also, the use of questionnaires might be regarded as a standardised way of collecting information. Biases related to difficulties in understanding the Norwegian language among Sami responders are likely small, as only a few (<5%) filled in the Sami version of the questionnaires in the municipalities with the largest Sami populations (results not shown). However, this might also indicate that the Sami questionnaires were difficult to understand, as primary and secondary schools did not allow the use of the Sami language until 1969. Moreover, the present writing style for the Northern Sami language was set in 1979, and for other Sami languages it was accepted even later.[187] Participants were, however, able to receive help from the staff at the research stations to complete the questionnaires.

The use of self-reported variables is associated with larger uncertainty than the use of standardised measurements. The following self-reported variables were included in the analyses: presence of CVD, use of antihypertensive medications, smoking status, family history of premature CHD, and ethnicity. According to Senior and Bhopal, a sound

epidemiological variable needs to fulfil three criteria: 1) it should be measured accurately, 2) it should differentiate populations in underlying relevant characteristics, and 3) the observed differences should generate testable aetiological hypotheses or be applicable to the planning or delivery of health care.[188] I will consider to what extend these criteria are present for ethnicity by describing issues related to heterogeneity and stability of Sami ethnicity, and challenges with lack of a golden standard. These conditions influence how well we might have been able to differentiate the populations. As ethnicity is multifaceted and a socially constructed trait, it may be inappropriate to use it for generating testable aetiology hypothesis. Moreover, I briefly mention how planning or delivery of health care to people with Sami ethnicity might be applicable. Lastly, I will discuss some limitation of other self-reported variables included in Paper 1–3, and strengths and limitations when using data from national registries.

Comparing ethnicity criteria with Sami electoral roll

The criteria used for the ethnic categorisation of Sami in Papers 1–3 resembles that of the Sami Parliament electoral roll, as both require that individuals perceive themselves as Sami and have a linguistic marker.[189] But the electoral roll differs from our categorisation in that they also include linguistic markers in great-grandparents, and the possibility to register themselves if they are descendants of someone already enrolled. Given that the legitimacy of the Sami Parliament is dependent on an electoral roll that is representative of the Sami people, one might argue that the ethnic categorisation used in the papers, on which this thesis is based, have high accuracy and represent the Sami population. On the other hand, the criteria for registration in the Sami electoral roll, have been constructed in relation to the political rights that Sami are granted due to their ethnicity. Such political rights, and hence the ethnic categorisation used herein, might be irrelevant for health research.

Intra-group heterogeneity in Sami

The criteria for ethnic categorisation might have determined whether or not we observed ethnic differences. Assuming that ethnic differences increase with the degree of 'Saminess', a strict definition of who are considered Sami might demonstrate ethnic differences, whereas a broad definition might dilute ethnic differences, which would make it more difficult to demonstrate important ethnic differences. The criteria used for ethnic categorisation, determine the sample size of each ethnic group, and hence, the precision of the estimates. A strict categorisation of Sami might give imprecise estimates due to a smaller sample size, but these estimated would be reliable if ethnic differences increase with 'Saminess'. A broad definition of Sami on the other hand, might give larger precision, but ethnic differences would more likely be diluted.

Participants who fulfilled only one of the criteria (either perceived themselves as Sami/had Sami ethnic background themselves, or had Sami as the home language of one of three generations of their family) were categorised as non-Sami although they clearly demonstrated a Sami background or connection. However, we treat the groups as mutually exclusive,[190] despite that many categorised as non-Sami have Sami ancestors. Likewise, participants were categorised as Sami even if, in addition to Sami, they chose Kven, Norwegian, or other, in their responses to questions on ethnic background, self-perceived ethnicity, and language use in three generations, which created a heterogeneous Sami group. The main limitation of heterogeneous groups is that associations can be hidden.[190] Heterogeneity might bias the results non-differentially if both groups have a similar level of heterogeneity, or differentially if the heterogeneity is present in only one of the groups. Potential heterogeneity within the Sami and non-Sami groups was assessed by repeating the analyses using an alternative ethnic categorisation, which consisted of one non-Sami group and two Sami groups that differed in

their number of Sami ethnicity markers. The non-Sami group consisted only of those who had no Sami ethnicity markers, i.e., Kven, Norwegians and those that had ticked 'other'. One of the Sami groups consisted of those who had ticked Sami for all 11 ethnicity-related questions, and the other consisted of those who had 1–10 Sami ethnicity markers. We did not observe a consistently different pattern when using the alternative ethnic categorisation in any of the papers, which suggests that our ethnic categorisation of the Sami group rendered robust results.

Stability of the Sami ethnicity variable – misclassification

Self-perceived ethnicity can change in relation to social and political contexts and time.[125] However, a comparison of the answers on self-reported Sami ethnicity (linguistic markers in three generations and self-perceived ethnicity) from the 1970 population census with the answers from SAMINOR 1, suggests that Sami ethnicity is stable.[191] We computed Cohen's kappa (section 2.5.1) to explore the level of agreement for reporting Sami ethnicity at both surveys compared to what would be expected by chance. The Cohen's kappa suggested that Sami ethnicity had high reproducibility, which supports the high stability of Sami ethnicity and supported our decision to impute ethnicity in those that participated in both SAMINOR 1 and 2, but had missing information for ethnicity in one of those surveys.

Evaluation of Sami ethnicity

We do not know how accurately we have measured ethnicity, as we do not have a registry of ethnicity that might be considered to provide a standard. But, even if there were a registry of ethnicity, the question of who would register themselves as Sami and non-Sami, and on which criteria, would still exist. Nonetheless, the criteria to the Sami electoral roll might be considered to be a standard for categorisation of Sami ethnicity. Given this, we might

consider our ethnicity criteria to be as accurate as possible given the available information. We might also assume that we have differentiate the populations correctly, as there was no indication of the contrary when doing sensitivity analyses using a different ethnic categorisation. In terms of generating testable aetiology hypothesis, Paper 3 suggests that height difference in Sami and non-Sami reflect an exposures that also determines their risk of CS. In terms of applicability of the ethnic categorisation for planning or delivery of health care, the resemblances between our ethnic categorisation and the Sami electoral roll suggests that targeted measures could be applied in a similar way as other targeted measures from the Sami Parliament directed towards the Sami people.

In recent time, Raj Bhopal has suggested, that the research question itself should determine how ethnicity is applied.[192] The ethnic categorisation chosen in Paper 1–3 might be in accordance with the research question, as we have included self-perceived ethnicity or ethnic background that captures the social dimension by ethnicity. The risk of CVD is also partly dependent of family history and inheritance, which we have embodied in the categorisation by including questions of Sami language use in one of three generations. It should be noted, that by including inheritance we only consider genetic susceptibility within families, and not that Sami are genetically different from non-Sami.

Self-reported smoking

Self-reported smoking can be underestimated, as smoking is perceived to be an undesirable behaviour in general, and in particular when smoking cessation is expected due to pregnancy, cancer, or other chronic diseases.[193] The precision of self-reported smoking has been found to be dependent on the study setting and population, how the question is posed, and the purpose of the study.[194] The smoking-related questions used in SAMINOR 1 and 2 have not

been validated in a Sami population, and they were posed differently in the surveys. In all of the papers in this thesis, we estimated the proportion of current smokers in Sami and non-Sami and included smoking status in the computation of 10-year risk. In Papers 1 and 3, we excluded all participants with missing values on smoking. In Paper 2, those with missing values on smoking (58 missing items in SAMINOR 1; 68 in SAMINOR 2) were categorised as non-smokers, which may not have been the correct assumption. Excluding those with missing items for smoking in Paper 2 (both SAMINOR 1 and SAMINOR 2) gave somewhat lower odds ratios and prevalences of smoking, but the conclusions regarding a significant decline in smoking in the overall and sex- and ethnicity stratified analyses did not change (results not shown).

Differentiating smokers from non-smokers might be regarded as crude and inaccurate, as the associations between smoking and CVD are dependent on duration (years) and intensity of smoking (number of cigarettes smoked per day), also known as pack-years.[195] However, as there were additional missing items for number of cigarettes smoked per day, and duration of smoking in years in both smokers and former smokers we chose not to include pack-years, as this would have given us an even smaller sample size. Moreover, we did not consider former smokers in any of the papers; we simply merged them with never smokers to create the category of non-smokers. Former smokers have higher mortality relative to never smokers,[196] and merging them with never smokers can underestimate the relative risk of smoking. Due to potential residual confounding from the imperfect operationalisation of variables in Paper 3, we included pack-years (number of cigarettes smoked per day divided by 20, multiply by years of smoking) of smoking instead of smoking as a dichotomous variable in the models for each endpoint. Using pack-years in a somewhat smaller sample did not change the relative risks of any of the investigated CVD endpoints (results not shown).

Use of antihypertensive medications and misclassification of item non-response

In all papers, missing items for self-reported use of antihypertensive medication were considered as negative responses, and merged with previous and never users to distinguish them from current users. Considering missing items as negative responses might be a correct assumption, as users are likely aware of their conditions, whereas those not taking any medication might find the question irrelevant and ignore it. In Paper 1, 171 participants had missing values on the use of antihypertensive medications; in Paper 2, this was the case for 105 participants in SAMINOR 1 and 299 in SAMINOR 2; and in Paper 3, there were 142 participants with missing items for use of antihypertensive medications, all of which were categorised as non-users. This is a strong assumption, however, and no literature supports this form of ad-hoc imputation. Therefore, as an additional sensitivity analysis, we excluded participants with missing items for antihypertensive medications in Papers 1–3 and repeated some of the analyses. The results for the sex-, ethnicity- and age-specific 10-year risks (n=5,147) in Paper 1, the sex- and ethnicity-specific GEE regression for use of antihypertensive medications and hypertension in Paper 2 (n=11,636), and the hazard ratio for ethnicity (model 3) in Paper 3 for the various CVD endpoints resembled the original results, thus the conclusions did not change.

Self-reported cardiovascular disease

We excluded those with self-reported angina pectoris and AMI in Paper 1, and in Paper 3, excluded those with self-reported CS. Self-reported AMI and CS in SAMINOR 1 had high to moderate agreement when validated against hospitalisation discharge codes, and no ethnic variation was observed in the validity of these.[197] Thus, ethnic differences in exclusion of prevalent cases based on self-report of AMI or CS, is most likely not an issue of concern.

Misclassification of endpoint in Paper 3

We compared fatal and non-fatal CVD cases in Sami and non-Sami, by using information from national registries. The main and secondary discharge diagnosis to CS in the Norwegian Patient Registry have been found to be adequate complete (i.e. to what extent all data that could have been registered is registered) and correct when compared to medical records from hospitals.[198, 199] The correctness to a registry is often measured by positive predictive value and describes to what extend the results confirms with the true. The correctness for CS in Norwegian Patient Registry varied from 68% to >90% and improved when restricting the comparison to main discharge diagnosis only.[198-200] The most common cause for incorrect diagnosis of acute CS was previous CS events that should have been coded as sequela and rehabilitation after CS.[198, 199]

The main and secondary discharge diagnosis to AMI in the Norwegian Myocardial Infarction Registry and the Norwegian Patient Registry were compared with the discharge diagnosis to patients admitted to a hospital in Mid Norway from July to December 2012 with a cardiac troponin T plasma value. Both registries were found to be adequately complete and correct. In terms of the latter, both registries had a positive predictive value >90% when compared to the hospital discharge diagnosis that was based on the comprehensive medical history.[201] As with CS, restricting the comparisons to only main diagnosis of AMI, improved the correctness for Norwegian Patient Registry.[201] These studies indicate that the Norwegian Patient Registry provide adequately complete and corrected data for both main and secondary discharge diagnosis of CS[198-200] and AMI,[201] which might eliminated potential differences in how the data were registered by the CVDNOR and Norwegian Patient Registry.

A study conducted in Norway in 1965–2005, compared the underlying cause of death among 1,140 individuals with autopsy records [202] to that in Cause of Death Registry, and found a somewhat lower agreement between CS deaths than CHD deaths, but an overall a substantial agreement between the two records. Assuming that these data hold for the CHD and CS deaths included in Paper 3, we might not have large issues with misclassification of deaths. Out-of-hospital deaths among individuals aged >80 years might be underreported, as comorbidities increase with age, and possible silent AMI or silent IS/CS [203] is not recognised when ICD-10 R96 (instantaneous death) and R98 (unattended deaths) are used as the underlying cause of death.[204] Using data from the Tromsø studies in 2001–2009, one study showed that age- and sex-specific trends in AMI did not change after the inclusion of cases with R96 and R98 as the underlying cause of death,[204] which suggests that excluding these codes from the AMI endpoint might not threaten our conclusions regarding AMI. How the use of R96 and R98 influence the incidence of CS is uncertain.

Use of specialist health care in geographical areas that represents Sami, i.e. municipalities included in the administrative area for Sami languages (varying from six to eight municipalities), have been compared with geographical areas (varying by study) with an assumed small proportion of Sami, representing non-Sami. The public expenditures to somatic hospitals and specialist services in 2002–2006 [205] and use of radiotherapy in cancer treatment in 1999–2008 [206] showed similar use in geographical areas representing Sami and non-Sami. Use of mammography screening for breast cancer was more common in areas representing Sami women than by their non-Sami counterparts in 2001–2010.[207] Use of conventional radiography, magnetic resonance, computerised tomography, and ultrasound in 2003–2009, showed smaller use of the two latter and more of magnetic resonance in areas representing Sami, which they assumed to be caused by a lower cancer risk in Sami.[208]

Overall, these studies [205-208] indicate no systematically different use of specialist health care in Sami and non-Sami areas. The coding of discharge diagnosis is assumed to be similar in all hospitals in Norway, as Norwegian hospitals have a similar governmental reimbursement system. Therefore, it is unlikely that main and secondary discharge diagnosis and the underlying cause of death differ systematically by ethnicity. Potential misclassification is probably similar for both of the comparison groups, which potentially underestimates the associations between ethnicity and risk of CVD.

4.2.2 Selection bias

Descriptive studies (Paper 1 and 2) and measures like incidence rates (Paper 3), are important as they reflect the health of the target population. However, the descriptive measures from a sample should be representative of the target population to be valid. If the descriptive measures are valid, it might be possible to extrapolating the figures to populations with comparable demographical characteristics.[209] If individuals have different probabilities of being included in a survey, there might be a selection of participants. A selection might cause systematic differences in participants and non-participants in relevant characteristics such as the exposure and outcome,[210] in which case we might have a selection bias.[183, 185]

Different participation by ethnic groups

A review investigated racial and ethnic minorities' willingness to participate in health research from 20 different health research studies conducted after 1984 mainly in US. The review showed that racial and ethnic minority groups were as willing as non-Hispanic white people to participate despite an assumed mistrust in medical research.[211] It is rather unlikely, however, that these results are generalizable to Northern Norway, as similar studies in Scandinavia with respect to Sami ethnicity and participation in health research do not exist.

Therefore, to what extent participants and non-participants in SAMINOR 1 and SAMINOR 2 differ in ethnicity, is unknown. It might be that Sami reframe from medical research as previous scientific research studies included scull measurements and physical appearance to place Sami as inferior to Norwegians.[128, 130] All invitees received the informational brochures where it was stated that information was collected in geographical regions where Sami and non-Sami live and that the survey was conducted by a Sami research centre. This could have increased participation of Sami as their presence in these regions were acknowledged, and could give lower participation among those that do not acknowledge Sami language and culture due to history of assimilation.[138] If this have induces a different selection of Sami and non-Sami participant into the surveys, and if such selection might bias the estimates, is however, unknown.

Non-participants in SAMINOR 1 and SAMINOR 2

As roughly half of those invited to SAMINOR 1 and SAMINOR 2 participated, there may have been a selection that makes the participants differ from non-participants (Tables 1–3). However, it is impossible to know to what extent if no research is done to compare participants with non-participants. We have limited information about the non-participants to SAMINOR 1 and 2, other than non-participants to both surveys were more often younger men,[160] and in SAMINOR 1, the non-participants were also more often single.[159] This is also reflected when comparing characteristics of the invitees with those included in the analytical samples in Tables 1–3, which suggests that the descriptive results from Paper 1–3 may be slightly prone to selection bias.

Surveys conducted in Norway with comparable participation rates to the papers included, have observed that non-participants were more often men, young, not married, were likely to

be receivers of disability pensions, and had lower education or income.[212-214] In the Tromsø study in 1994–1995 and 2007–2008, those with the highest level of education have better levels of conventional risk factors and smoke less than those with the lowest level of education.[215] If non-participants to SAMINOR 1 and 2 have lower education than those that participated, as observed by others[213, 214] and hence, have more adverse levels of conventional risk factors, then estimations of conventional risk factors in Paper 1 and 2 might be an underestimation of the true levels in the target population.

The third Nord-Trøndelag Health Study, also known as the HUNT 3 study, was conducted in 2006–2008 and 50,807 were invited, wherein 54% participated. The non-participants were more often younger, unmarried men,[213] comparable to the characteristic observed in non-participants to SAMINOR 1 and 2. Additionally, the non-participants to the HUNT 3 study had a higher prevalence of drug-treated hypertension, myocardial infarction, angina pectoris, CS, diabetes, psychiatric disorders, higher mortality, as well as being more inactive and having lower education and income.[213] Given that the HUNT 3 study and the SAMINOR 1 and SAMINOR 2 had resemblance in participation rate, and non-participants were more often younger and men, it might be that the non-participants to these surveys resemble each other in other characteristics as well. If that is the case, the incidence rates of CVD in Paper 3 might be somewhat underestimated as those with myocardial infarction, angina pectoris, CS, participated less. As no research is done among the non-participants to SAMINOR 1 and SAMINOR 2, we can only speculate how they may had influence our observations.

One study of non-participants to the Oslo health study that was conducted in 2000–2001, wherein 40,888 aged 30–76 were invited and 46% attended, explored how different prevalences in smoking, obesity and diabetes among non-participant changed the prevalences

of these conditions in the target population.[214] Their analyses showed that differences in prevalences in participants and non-participants, had only small effect on the overall results, which suggested that the sample was representative of the target population despite somewhat low response rate.[214] Similarly, the overall anthropometric and disease prevalence estimates in the HUNT 3 study did not changes substantially when merging the values to non-participants with those to participants, despite non-participants having poorer health and lower education.[213] It should be noted, the HUNT 3 study and the Oslo health study had substantial sample sizes. This suggests that despite only half of those invited participate in a survey, bias from selection might not be a problem, as long as the sample size is large. It is likely that the prevalences and mean values vary in participants and non-participants to SAMINOR 1 and SAMINOR 2, but our results in Paper 1 and Paper 2, and incidence rate in Paper 3 may still be valid for the target population as the sample sizes are relative large. It should be noted, that our assumption of negligible different selection of participants and valid estimates rest on an assumption that there are no different selection by ethnicity or by other variables related to ethnicity.

Estimation of change in risk factors in Paper 2

Table 2 show that the analytical sample in SAMINOR 2 tended to be older than the sample in SAMINOR 1. SAMINOR 1 had the fewest participants from Region 1, which included municipalities with that the highest proportions of individuals with Sami affiliation,[159, 160] whereas SAMINOR 2 had the fewest participants from Region 3, which included municipalities were Sami are in the minority. This was also reflected in the ethnic compositions of participants, with more participants of Sami ethnicity in SAMINOR 2 (40.4%) than in SAMINOR 1 (35.3%). However, as noted previously, this is most likely due to issues with the design of SAMINOR 1 (see section 2.1), not people's willingness to

participate in the surveys. However, if and how this issue might have influenced the ethnic comparisons, is unknown.

In Paper 2, we used a statistical method (generalised estimating equations) that assumes that missing values are missing completely at random.[167] However, the missing values in the surveys depended on the birth cohorts invited to SAMINOR 1 and 2, which might bias the regression coefficients when using this statistical method. We explored this, by excluding those who were not eligible for invitation to both surveys; we excluded those aged ≥70 years from SAMINOR 1 (n=820), and those aged 40–50 years from SAMINOR 2 (n=1,275). When the generalised estimating equations analyses were repeated in this sample (n=10,278), the results resembled those of the original analyses, except for SBP, which showed a different decline for non-Sami and Sami women (-5.2 and -3.7 mmHg, respectively). Overall, we consider the estimates from our study to be fairly robust.

Relative risk estimations in Paper 3

In order to have valid relative risk estimates from cohort studies (Paper 3), losses to follow-up should be independent of the outcome. In Paper 3, we had complete follow-up for all participants and 101 were lost to follow-up due to emigration. The losses to follow-up did not differ by ethnicity (results not shown), and assumed to be independent of the incidence of CVD, which was not explored.

Effect estimation that stem from aetiological relationships are less likely to be influenced by bias from selection, but the strength of association might vary by population.[182, 212, 214, 216] In Paper 3, height explained the excessive risk of CS and IS in Sami, which is supported by some studies [78, 79] whereas other studies show no associations.[81] A meta analyses estimated

a steeper risk reduction at lower height and suggested an inflection point at 170 and 160 cm for men and women, respectively, where height is no longer associated with CS.[217] The lack of an inverse association between height and CS between different studies might partially be due to variations in population average height, and due to a possible presence of an inflection point where height is no longer inversely associated with CS and IS. Therefore, it is likely that the association between height and CS and IS are valid for the target population, but the strength of association might vary between participant and non-participant as their average height differ.

4.2.3 Confounding and intermediate variables

Confounding is a phenomenon that can create a statistical association between an exposure and an outcome when the groups that are compared, in this case Sami and non-Sami, differ in other variables that are causally or non-causally associated with the exposure, and causally associated with the outcome.[183]

In an ideal comparison, the groups should be similar to each other in all aspects other than exposure status, which is why sample characteristics in the exposed and non-exposed groups are compared, with particular attention paid to factors that are thought to be causally associated with disease.[182] If the groups differ in factors that might be causally associated with the outcome, this can create a false association caused by the confounder and not by the exposure. In contrast to information and selection bias, it is possible to correct for confounding variables by adjusting for them in regression analyses or stratifying by these variables.

A confounder is different from an intermediate variable,[183] which is a variable in the causal pathway between an exposure and an outcome. Intermediate variables can cause variation in the outcome, and vary themselves by variation in the exposure.[35] In contrast to confounders, one should not adjust for intermediate variables if one is interested in the full effect of the exposure, unless this is done intentionally, as in Paper 3 when trying to explain what variables might explain differences in risks. We considered height and conventional risk factors as part of the causal pathway between ethnicity and risk of CVD. The risk of CVD is mediated through height as surrogate measure of genetic composition and environmental exposure, and conventional risk factors are considered mediators of a potential ethnic differences in risk.

The usefulness of an adjustment depends on how well the confounding variable is measured, if it is measured at all, and whether the researchers are aware of the confounders and actually adjust for them. Imperfect data collection of confounding variables, failing to take into account confounding variables, or the use of a suboptimal regression model can lead to biased estimates, and is known as residual confounding.[183] In Paper 3, we suggest that residual confounding might partly explain the observed association between Sami ethnicity, and CS and IS, as observational studies are prone to bias due to things like the imperfect collection of data.

4.2.4 Interaction

Interaction is present when the association between the exposure and an outcome change in the presence of a third variable, which is then considered to be an effect modifier.[183] An effect modifier is different from a confounder as the exposure–outcome association differ in levels or strata of the effect modifier, whereas the exposure–outcome association is similar in all levels of a confounder.[218]

In Paper 2, we formally tested whether continuous and discrete conventional risk factors changed differently by ethnicity from SAMINOR 1 to SAMINOR 2. This was tested on a multiplicative scale.[219] Moreover, the changes in risk factors were illustrated in figures in the supplementary figures (to Paper 2), which suggested that the interactions observed in men and women were most likely due to random variation, as the changes fluctuated by age group.

In Paper 3, for each variable that was entered, we tested if sex or ethnicity modified the associations with the endpoint. This was done by including a product term with ethnicity and a product term with sex in separate models. If the product term was statistically significant (p-value <0.05), the product term was included in the final model for each endpoint. The risk of CS and the composite endpoint were lower in women than men, and only in women, an inverse association was observed between height and both endpoints. For men, the association was also negative, but not statistically significant (Paper 3, Supplementary table 2). The product term between sex and height was not statistically significant (p=0.078) in the final model for composite endpoint. However, we chose to include it as it was statistical significant in the complete model for CS. Men had significantly higher risk of AMI than women, but only in women the use of antihypertensive drugs was associated with higher risk of AMI.

4.2.5 Statistical associations and chance findings

In all the papers included, we performed several statistical tests, like testing for interactions (Paper 3), which increases the probability of type 1 error, i.e., falsely-rejected null-hypotheses [220] and chance findings. We used the α-level of 0.05 for all tests in the papers, which means that the probability of type 1 error in a single test was 5%. However, when doing several tests on the same dataset, the probability of at least one falsely-rejected null-hypothesis is larger.

This is commented specifically in Paper 1 in relation to the significant difference in risks observed among men aged 55–64 years, but it applies to all papers.

A larger sample size increases the power of a study, and leads to smaller standard errors and narrower CI. Small differences between groups may be statistically significant simply because of a large size of the sample, but may not be clinically or practically meaningful, i.e., they may indicate a difference in health outcomes that is so small that is does not merit attention. Judging whether or not a difference is clinically significant is done on the basis of expert knowledge in the field of study. As mentioned, in Paper 2, we found that Sami and non-Sami had different changes in total cholesterol and triglycerides (men only), HDL cholesterol (both sexes), and in 10-year risk (women only), but the ethnic differences were small, the results fluctuated by age group, and the analyses included multiple tests, which suggests that the results might be partly due to random variation or chance, and might not be clinically relevant in terms of differences in health outcomes.

Conclusion regarding internal validity

We acknowledge that there are several potential biases and weaknesses in our study samples and analyses. Regarding potential misclassification of Sami and non-Sami ethnicity, this might be similar in both ethnic groups, which suggest a non-differential bias that might underestimate any ethnic differences. The repeating of the analyses after excluding missing values to self-reported variables (use of antihypertensive medication in all papers; smoking in Paper 2) and replacing dichotomous smoking variable with pack-years (Paper 3), suggest that we do not have serious misclassification bias as the results resembled the original results. Concerning selection bias, it might be differences in means and prevalence estimates between those that participated and non-participants to SAMINOR 1 and SAMINOR 2. But, as the

analytical samples were relative large the results from Paper 1–3 may still provide a true picture of the health status to the target population and of potential ethnic differences. In Paper 3, incidence were measured by linking SAMINOR 1 to information from national registries that are independent of ethnicity, which strengthens the estimates and comparisons. The association between height and IS and CS are likely to be applicable to the target population and other population, but the strength of association might vary by populations as the mean height vary in populations. In the papers included, confounding was not considered as Paper 1 and 2 were mainly descriptive studies, and in Paper 3, incidence rate and potential intermediate variables were identified. Despite these limitations, we believe that the internal validity of our findings was not compromised, and that the results reflect the health status and associations in the target population in a relatively unbiased way. Young, unmarried men participated less than older men, which suggests that the results might be less representative of the former population.

4.3 External validity

The external validity refers to whether the results from a study can be generalised to populations other than the target population,[2] and is also referred to as generalisability. As mentioned, in order to have external validity, the study has to be valid for the target population, i.e. have internal validity.

Although the internal validity of the papers in this thesis seems to be acceptable, the generalisability of the results from Papers 1 and 2 are limited to rural regions in Northern Norway where Sami and non-Sami co-exist. However, the applicability of the findings included in the Paper 1 and 2 might not be relevant to the Sami population today, as the results are from a survey in 2012–2014. These findings might on the other hand indicate that

Sami and non-Sami lifestyles do not contribute to different levels of conventional risk factors or differences in 10-year risks as the levels were rather similar in the time-period 2003–2004 to 2012–2014 (Paper 1 and 2).

The analytical sample in Paper 3 included the complete SAMINOR 1 sample, which covered a substantial part of the Sami population in rural Northern and Mid Norwegian. One might therefore argue that the results in Paper 3 can be generalised to all Sami populations in rural Norway, but not to those living in cities. The individuals in Paper 3 were followed to the end of 2016, and the ethnic comparison may still be valid to present day Sami and non-Sami aged 30 and 36–79 years, given that the change in conventional risk factors were rather similar from SAMINOR 1 to SAMINOR 2. However, the incidence rates might on the other hand be somewhat overestimated as conventional risk factors are expected to decline and treatment is expected to improve over time. Concerning the relative risk estimations between height and Sami ethnicity, similar associations are likely to be observed in populations that differ in height, although with varying strengths.

As for the non-Sami population included in Papers 1–3, the results might only be generalisable to Norwegians, and not to the Kven population living in rural areas of Northern and Mid Norway.

5 Discussion of main results

5.1 Paper 1

The sex-specific and age-stratified mean risks and proportions at high risk were overall similar in Sami and non-Sami men and women in 2012–2014. Studies up till today [19, 23, 113-117] have observed none or only small ethnic differences in conventional risk factors, which support our observation of no ethnic differences in 10-year risk.

Concerning abdominal obesity, Sami women had a larger mean waist circumference than non-Sami, as observed by others.[17, 23, 114] Together with the observation of higher prevalence of type 2 diabetes in Sami men and women in SAMINOR 2,[20] these characteristics may contribute to a higher 10-year risk of AMI or CS in mainly Sami women. However, the mechanism and aetiology of CS and IS are complex, and risk and protective factors for CS and IS might differ in time and in the ethnic groups.

The incidence of AMI and CS has most likely declined from the time when the regional health surveys (from 2000–2003) used in the validation of the NORRISK 2 model were conducted, and SAMINOR 2. As mentioned, in 1995–2012 decline in conventional risk factors accounted for roughly 60% of the decline in incidence of CHD [29] and CS.[32] As the decline in incidence of AMI and CS was not entirely caused by a decline in conventional risk factors, the 10-year risk might be overestimated. Paper 2 indicate no substantial ethnic differences in change of conventional risk from SAMNOR 1 to SAMINOR 2, which suggests that the observation of no ethnic differences in the risk of the composite endpoint observed in SAMINOR 2, might also be present after 10 years.

A study of a multi-ethnic cohort in the US suggested to include height in risk models of CVD to increase their predictive value,[221] as adding new predictive factors can help correctly classify those at borderline risk and avoid further laboratory testing.[95] If the NORRISK 2 is applied in populations that differ in height, including adult height might improve the 10-year risk estimation, as height provides information on the interaction between genetic and environmental factors. But, as risk models are seldom improved when adding new predictive factors beyond sex, age, and conventional factors,[11, 222] the addition of height might only help to correctly classify those at borderline risk.

5.2 Paper 2

From 2003–2004 to 2012–2014, Sami and non-Sami populations in the 10 rural municipalities had favourable declines in total cholesterol, blood pressure, and number of current smokers, which have been observed in studies in Norway [223-227] and in western high-income countries.[228-231] Small, but less favourable change were observed in some conventional risk factors in Sami compared to non-Sami men and women. However, the differences were small and possibly due to chance or random variation. The conventional risk factors in Sami and non-Sami are considered to be intermediate factors between ethnicity and risk of CVD, and they are expected to reflect an ethnic specific lifestyle.[158] As the changes in conventional risk factors were rather similar in the ethnic groups, Paper 2 suggest that Sami and non-Sami have had a similar changes in lifestyle from SAMINOR 1 to SAMINOR 2.

The SAMINOR 1 and 2 samples in Paper 2 included different birth cohorts. Compared to SAMINOR 1, SAMINOR 2 did not include those born in 1925–1934, but did include those born in 1964–1973, which increased the level of education in SAMINOR 2. Studies have shown an educational gradient in CHD mortality,[232, 233] where a low level of education is

associated with unfavourable levels of among other smoking. This suggests that the favourable decline in total cholesterol, blood pressure, and smoking observed in Paper 2 might be driven by a general improvement in education in rural Northern Norway.

5.3 Paper 3

Acute myocardial infarction and coronary heart diseases in Sami

We observed no ethnic differences in the incidence of AMI or CHD, which is supported by other studies that indicates a similar incidence of AMI in Sami and non-Sami,[23, 107] also when including studies on mortality from CHD despite variation in these. With regards to mortality from CHD, the studies show a higher,[25, 107] lower,[24, 108, 109] or similar mortality [106, 107] that vary by sex and reindeer herding, where the latter have lower incidence in women [107] and lower mortality in men.[25] In SAMINOR 1, Sami men and women reported more often to have angina pectoris and symptoms of these compared to non-Sami,[234] which is somewhat surprisingly give the observation of no differences in incidence of AMI or CHD. However, self-reported myocardial infarction has been reported to be similar in Sami and non-Sami in SAMINOR 1 [118] and the question posed in SAMINOR 1 is found to have high positive predictive value when compared to hospital discharge data.[197]

Cerebral stroke and ischemic stroke in Sami

Sami had a 36% higher risk of IS and a 31% higher risk for CS. In absolute terms, this suggest that while non-Sami have 2–3 persons affected by CS or IS per 1,000 person-years, Sami have 3–4, which may translate to a relative small excess risk. The study on incidence of CS in Norway indicate a possibly higher incidence in Sami.[23, 26] Together with a study from Sweden in 1985–2002, including incidence of subarachnoid haemorrhage,[107] these studies give support of higher incidence of CS in Sami that may vary by attachment to reindeer

herding. Including studies from Sweden, Finland and Norway, variation was observed in mortality of CS showing similar [107, 109] or higher,[25] where the latter was also observed in reindeer herding women [107] but not in reindeer herding men.[25, 107] Unfortunately, we did not conduct separate analyses on fatal and non-fatal cases nor in relation to reindeer herding, which could have provided important knowledge on mortality and variations among subgroups. In our study, the relative risks for IS and CS were rather small with large variations, as observed in comparable studies.[25, 26, 107] Due to the low strength of the associations, it is possible that the excess risk was caused by biases or residual confounding, as observational studies are prone to bias.[185]

The composite endpoint

We found no ethnic differences in the composite endpoint consisting of AMI or CS. When splitting the composite endpoint into AMI and CS, we observed ethnic differences in the incidence of CS, but not that of AMI or CHD. It might be that the small number of CS events that exhibited an ethnic difference were cancelled out when they were merged with AMI events that exhibited no ethnic differences. One study in Sweden in 1985–2002 [107] compared the risk of a composite endpoint consisting of AMI and/or CS, which resembles the composite endpoint in Paper 3. They observed no ethnic differences, except that non-herding Sami men had higher incidences, and reindeer herding Sami men had lower incidences of the composite endpoint than their demographically-matched counterparts.[107]

Reindeer herding was associated with lower mortality of CHD in Sami men,[25] lower incidence of AMI in women in women,[107] lower incidence of CS in men,[25, 107] and higher incidence of CS in Sami women.[107] The higher incidence of CS and subarachnoid haemorrhage observed in Sami women within reindeer herding was hypothesised to be related

to psychosocial factors, as income and education did not explain differences between reindeer herders, non-herding Sami and a demographical-matched reference population reference.[107] In the papers included in this thesis, we did not do separate analyses for Sami with affiliation to reindeer herding, as we only had information on those financially responsible for the husbandry unit, which might be incorrect due to the organisation of members of 'siida'.[111] According to the Norwegian government there are about 3000 people that have reindeer herding as their main occupation,[235] which suggests this that reindeer herding Sami might constitutes a small subgroup within the Sami population.

Population mean height

Height explained more of the ethnic differences in CS and IS than conventional risk factors did, but it is not clear how height is associated with risk of CS and IS. Also, what height represents in the context of Sami versus non-Sami is difficult to disentangle, as it is unlikely that physical appearance itself increase the risk of CS.

Anders Forsdahl, who was a medical doctor and professor, observed high CVD mortality in the municipality of South Varanger in Finnmark County in the 1970s. His ecological studies suggested that poor living conditions in childhood and prosperity in adulthood were risk factors for later CHD through conventional risk factors, and that adult height was an indicator of poor living conditions in childhood.[236] Barker and others have suggested that early life factors,[91, 237] already present in foetal life, can alter the risk of CS. Some suggest that epigenetic modification such as DNA methylation can occur in the fetus if maternal nutritional deficiency occur during important periods in the development to the fetus, which predispose individuals to future CVD.[238, 239] Researchers have suggested that living conditions were harder for the Sami and Kven populations as compared to Norwegians,[240]

and that the living conditions in the previous Finnmark County were poorer as compared to the rest of the Norway,[241] which could explain why Sami and conscripts from Finnmark have a lower mean height.[93, 94] Moreover, one study suggested that haemorrhagic CS is more related to childhood socioeconomic conditions than CHD.[242] Although the living conditions of Sami and non-Sami may be perceived as rather similar today given the similar level of education and conventional risk factors, it is possible that childhood living conditions were poorer for Sami than for non-Sami participants. This perspective suggests that exposure through lifetime might be relevant in the context of Sami versus non-Sami and risk of CVD. It might also be possible that poor living conditions in previous generations still influence present day population height, as it takes time for population mean height to increases.[92]

Figure 6 illustrates the complex interplay between social environment that influence the psychological conditions, and health behaviour, which together with genetic, culture and early life factors determine the health of individuals. When exploring ethnic differences, it has been recognised that socio-economic position, often measured by education, income or occupation, is a powerful determinant of health and CVD.[125, 243] In the papers included in this thesis we have not adjusted for socio-economic position as the sample characteristic and other studies [107] did not indicate large differences in education. In the context of Sami and non-Sami, other unknown socio-economic variables than education and income might be relevant in explaining differences in CVD risk, as suggested by Sjölander.[107] Adult mean height in Sami and non-Sami populations in SAMINOR 1 might actually represent childhood living conditions, i.e. childhood socio-economic conditions [89] to the included birth cohorts or to previous generations of these.

One of the main differences between Sami and non-Sami populations in Norway is that Sami were exposed to governmental assimilation,[138] similar to other Arctic indigenous populations. The structural assimilation policies [14] might be considered as one of the social structures that determines health (Figure 6). In SAMINOR 1, Sami and Kven report more ethnic discrimination and bullying [244] and stress [156] than Norwegian. Some have suggested that marginalisation induced chronic stress in Sami that make them more prone to ill health, i.e., higher proportions reported lifetime CVD.[22] Ethnic discrimination was associated with psychosocial stress in SAMINOR 1.[156] Psychosocial stress is a risk factor for CS,[245] and the excess risk of IS and CS in Sami might come from the psychosocial stress of assimilation and subsequent marginalisation,[22] which we did not measured nor adjusted for. The lower height in Sami can also be a marker of some ethnic-specific exposure, such as marginalisation.[22] However, the association between discrimination, stress and CS in this population is at this point only a speculation, but might serve as a foundation for further research on the topic.

5.4 Comparable indigenous populations

Alaska natives have found to have higher CS mortality than white populations in the US,[246] and the incidence of CS has been suggested to be higher in the Inuit population in Greenland than in Western populations.[247] The inferior cardiovascular health of American Indians and Alaska Natives relative to non-indigenous peoples has been suggested to be due to lower socioeconomic status and poorer access to quality healthcare among the indigenous population.[248, 249] It is challenging to compare the health of Sami to the Inuit, and Alaska natives, as the countries they inhabit differ in health care and education, which are determinants of health.[250] However, regarding the higher susceptibility to CS in Greenland Inuit populations, some have suggested that genetic, foetal, and early life factors, in addition to mental stress, should be of concern when considering Inuit health.[247] CVD is a complex

group of disease, and susceptibility to different types of CVD is a result of both genetic and environmental factors, which makes it difficult to disentangle which risk factors are relevant at a population level at any time.

6 Conclusion

1. Based on our findings, Sami and non-Sami populations living in 10 rural municipalities of Finnmark and Troms, and Nordland Counties had rather similar levels of estimated 10-year risk of AMI or CS, which reflected similar levels of conventional cardiovascular risk factors.
2. In these regions, we found that both Sami and non-Sami had substantial reductions in total cholesterol, blood pressure, and smoking from 2003–2004 to 2012–2014, that are likely to reduce the number of fatal and non-fatal CHD and CS cases in these populations. Small ethnic differences were observed mainly in total cholesterol (men only), HDL cholesterol, triglycerides (men only), and the 10-year risk of AMI or CS (women only), for which Sami had a slightly less favourable change than non-Sami. However, as the differences were small, it is likely that they are of little clinical value and may be due to random variation.
3. Comparing the incidence of CVD in Sami and non-Sami in 24 municipalities of Northern and Mid Norway revealed no ethnic differences the in risk of AMI and CHD, which is in accordance with previous studies. We found an approximately 31% higher risk of CS in Sami; this represents a relatively small excess risk, which might have been partly caused by residual confounding.
4. The excess risk of CS in Sami men and women was explained to some extent by adjustment for adult height. Height might be as relevant as conventional risk factors in explaining elevated risk between ethnic groups that differ in height. Low height was associated with Sami ethnicity. However, height might not be a causal factor in itself, nor is ethnicity;[125] they are both surrogate variables.

6.1 Public health relevance

The findings from the papers included in this thesis provide important knowledge on the CVD burden in Sami. A substantial proportion of fatal or non-fatal CVD cases are attributable to modifiable risk factors [9, 29, 32] that can be prevented by public health measures. The distribution of conventional risk factors and the 10-year risk were rather similar in Sami and non-Sami (Paper 1). This suggests that potential ethnic differences in Sami and non-Sami lifestyles might not lead to large differences in conventional risk factors and future risks. Moreover, it is likely that the favourable decline in conventional risk factors observed in Paper 2 will give fewer new cases of fatal or non-fatal CHD and CS in both ethnic groups. However, this will depend on the demographic structure of the population.

A substantial and favourable decline observed in total cholesterol, blood pressure, and smoking was observed in both ethnic group (Paper 2), which suggests that population-based preventive strategies are measures that work independent of Sami ethnicity. However, this does not exclude the possibility of other regional-specific, or ethnicity-specific protective or detrimental factors that influence the risk of CVD. Therefore, national health campaigns should also be promoted in the Sami languages to ensure similar health benefits in Sami and non-Sami communities.

The excess risk of IS and CS in Sami observed was small and possible due to residual confounding (Paper 3). The observed excess risk of CS was relatively small, which suggests that special clinical or public health measures in Sami communities are not necessary at the time. Moreover, as we do not know how low height is associated with risk of CS, it is difficult to know how to prevent new cases. On the other hand, as CS survivors can suffer significant physical and cognitive impairments, reduced capacity to perform daily activities, and might

become partially or fully dependent on caregivers, the observation of excess risk of CS in Sami calls for awareness. It is therefore important that Sami patients are offered care in culturally adapted nursing homes, and offered care in their own language.

6.2 Suggestions for further research

The findings of Paper 3 should be reproduced in a study including Sami from Norwegian cities and comparing reindeer herding Sami with Sami with other occupations, to see if the strength of associations changes overall and by these factors. Moreover, the endpoints included in Paper 3 should be divided into fatal and non-fatal cases to explore if the ethnic differences are more related to fatal than non-fatal events. Also, future research projects on cardiovascular health in Sami and non-Sami should try to disentangle what height actually represents as an intermediate variable between ethnicity and CS. This could be done with mediation analyses. Additionally, as one of the main differences between Sami and non-Sami is their exposure to governmental assimilation policies,[138] it would be interesting to investigate if Sami experience any long-term cardiovascular health consequences due to marginalisation and chronic stress.

Despite the findings of small or no ethnic differences in conventional risk factors and risk of CVD, it is necessary with a continuous health surveillance in rural Northern Norway, to make sure that health promotion campaigns have a national impact, and to avoid unintended ethnic or regional differences in health over time. Moreover, the studies that contribute to the present knowledge of small or no ethnic in risks, have used different ethnic categorisation, are from different time periods, and include different geographical population. These conditions make it challenging to draw a firm conclusion regarding ethnic differences in CVD health.

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Paper 1

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ORIGINAL ARTICLE

Distribution of risk factors for cardiovascular disease and the estimated 10-year risk of acute myocardial infarction or cerebral stroke in Sami and non-Sami populations: The SAMINOR 2 Clinical Survey

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Abstract

Objective: This study aimed to assess and compare the distribution of cardiovascular risk factors and the estimated 10-year risk of fatal or non-fatal acute myocardial infarction (AMI) or cerebral stroke (CS) among the Sami and non-Sami populations of Northern Norway. *Methods:* The SAMINOR 2 Clinical Survey is a cross-sectional survey conducted in 10 municipalities in the counties of Finnmark, Troms and Nordland in rural Northern Norway in 2012–2014. All inhabitants aged 40–79 years were invited to participate, and 6004 (48.2%) accepted. The NORRISK 2 model was used to estimate the 10-year risk of fatal or non-fatal AMI or CS. Sex and age were included in the model, as well as the following risk factors for cardiovascular disease (CVD): serum total cholesterol, serum high-density lipoprotein cholesterol, systolic blood pressure, smoking habits and anti-hypertensive treatment. *Results:* Only minor ethnic differences were observed between Sami and non-Sami populations in a number of individual risk factors for CVDs. Overall, the NORRISK 2 model revealed no ethnic differences in the 10-year risk of AMI or CS. ***Conclusions:*** **There were no differences in 10-year risk of AMI or CS between the Sami and non-Sami populations in 10 selected municipalities in Northern Norway.**

Keywords: *Cardiovascular mortality, cardiovascular morbidity, indigenous, native, aboriginal, ethnic, risk model, NORRISK, Norway*

Introduction

Indigenous populations often have poorer health than their respective majority reference populations [1], and recent reviews have indicated poorer cardiovascular health in Inuit populations and other indigenous populations in North America [2–4]. Population health is sculptured by many factors: level of education, wealth, environmental quality and protection, diet, behaviour traits such as physical activity and smoking, occupational and domestic stresses and genetics. Varying exposures to such factors over time can generate ethnic differences in health [5].

The Sami are an indigenous people whose traditional settlement area, Sápmi, stretches from the Kola Peninsula in the north to Engerdal and Idre in the south of Norway and Sweden, respectively [6]. Finnmark is the northernmost county in Norway and the region most influenced by Sami language and culture [6]. The Kven are descendants of Finnish-speaking settlers who emigrated from Sweden and Finland to the northern parts of Norway in the 1700s and 1800s [7].

In 1974–1975, a cardiovascular risk score among Sami and Kven/Finnish men aged 35–49 years in Finnmark was about 40% higher than that among Norwegian men [8]. The score only included systolic blood pressure, serum total cholesterol and smoking

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habits. However, later studies restricted to Finnmark County in the period 1974–1989 [9–12] and the SAMINOR 1 Survey from 2003–2004 [13] also including the southern regions of Sápmi showed none or only minor differences in the distribution of cardiovascular risk factors and in the burden of cardiovascular disease (CVD) between Sami and non-Sami populations in Norway. In terms of differences in CVD mortality between these ethnic groups, there have been conflicting results [14,15]. Furthermore, in Northern Norway, risk-factor levels and CVD mortality have declined over the last decades [16]. Whether this decline has been the same in Sami and non-Sami populations is unknown. Hence, updated knowledge concerning the distribution of CVD risk factors and a comprehensive CVD risk assessment of the Sami and non-Sami populations settled in Sápmi is needed.

CVD is multifactorial. Thus, guidelines for prevention recommend assessing the impact of several risk factors simultaneously [17]. The NORRISK 2 model is a cardiovascular risk model based on Norwegian data. It estimates the 10-year risk of fatal or non-fatal acute myocardial infarction (AMI) or cerebral stroke (CS) by combining information from several risk factors [18]. Using this model and data from the second survey of the Population-based Study on Health and Living Conditions in Regions with Sami and Norwegian Populations (the SAMINOR 2 Clinical Survey), the primary objective of the present study was to compare the distribution of risk factors included in the NORRISK 2 model and the estimated 10-year risk of AMI or CS in Sami and non-Sami populations. Furthermore, to give a comprehensive cardiovascular risk profile to Sami and non-Sami populations, we also present the distribution of other established risk factors and education attainment. The 10-year risk for AMI or CS has not previously been computed and compared for Sami and non-Sami populations.

Methods

The Population-based Study on Health and Living Conditions in Regions with Sami and Norwegian Populations – the SAMINOR Study – is run by the Centre for Sami Health Research at UiT – The Arctic University of Norway. The present analyses are based on cross-sectional data from the second survey, the SAMINOR 2 Clinical Survey (hereafter referred to as SAMINOR 2), conducted in 2012–2014. Invitation to SAMINOR 2 included all inhabitants (n=12,455) aged 40–79 years residing in the municipalities of Evenes in Nordland County; Skånland, Kåfjord, Storfjord and Lyngen in Troms County; and Karasjok, Kautokeino, Porsanger, Tana and Nesseby in Finnmark County. In total, 6004 inhabitants accepted. The overall response rate was 48.2% (i.e. 45.8% men and 54.2% women) and varied from 41% in Evenes to 56% in Kautokeino. The participants completed a self-administered questionnaire that was mailed with the invitation letter and returned at the clinical examination. Participants aged 40–69 years completed an eight-page questionnaire; those aged 70–79 years completed a four-page questionnaire, with fewer questions and larger fonts. Questionnaires were prepared in Norwegian and translated into Northern Sami. Both the Sami and the Norwegian versions of the questionnaire were distributed to participants in Kautokeino, Karasjok, Nesseby and Tana, and the Sami version was available upon request to participants in Kåfjord, Storfjord, Porsanger and Lyngen. Invitees in Skånland and Evenes received the Norwegian questionnaire only. The SAMINOR Study was accredited by the Norwegian Data Inspectorate and approved by the Regional Committee for Medical and Health Research Ethics. The committee also approved this study, for which all participants gave written informed consent.

Clinical examination and blood-sample collection

Waist circumference was measured at the umbilicus with the participant standing, and abdominal obesity was determined by using the thresholds >102 cm and >88 cm for men and women, respectively. At least 15 minutes after participant arrival, blood pressure was measured with an automatic device (CARESCAPE™ V100 monitor), with the participants in a seated position with their arms resting at heart level. Following a two-minute rest, three measurements were taken at one-minute intervals. The average of the last two measurements was used in the statistical analysis.

Non-fasting blood samples were collected with participants in a seated position. Tubes with anticoagulant were used for glycated haemoglobin (HbA1c) measurements conducted on-site within 15 minutes of blood collection using DCA Vantage™ (Siemens Medical Solutions Diagnostics, Tarrytown, NY), which uses an agglutination inhibition immunoassay method. Tubes for serum samples were centrifuged within two hours of blood collection. Serum was separated and stored at −20°C, transported for further storage at –70°C and later used to analyse high-density lipoprotein (HDL) cholesterol, triglycerides and total cholesterol at the University Hospital of North Norway using an enzymatic colorimetric

test run with Cobas 8000B (Roche Diagnostics GmbH, Mannheim, Germany). Thresholds for triglycerides and HbA1c were taken from European [17] and World Health Organization guidelines [19], respectively.

Questionnaire

Ethnicity was ascertained by the questions: '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?' and 'What ethnicity do you consider yourself to be?' On all items, the response options were 'Norwegian', 'Sami', 'Kven' and 'other'. The questions were answered separately for each relative (11 questions in total), and multiple answers were allowed. Participants were defined as Sami if they considered themselves to be Sami or reported a Sami ethnic background and at least one of their grandparents, parents or they themselves spoke a Sami language at home. All others were categorised as non-Sami.

We identified current smokers and non-smokers from the questions: 'Have you ever smoked daily?' (yes/no) and 'Are you currently a daily smoker?' (yes/no). Previous smokers were categorised as non-smokers.

Anti-hypertensive treatment was ascertained from the question: 'Are you taking medication for high blood pressure?' The response options were 'yes, currently', 'in the past, but not currently' and 'no'. Participants reporting former use were merged with non-users, and missing values were ad hoc imputed as non-users.

Both questionnaire information and HbA1c measurements were applied to identify participants with diabetes mellitus. The question was: 'Have you ever been diagnosed with diabetes (high blood sugar)?' (yes/no). Missing values were classified as 'no'. In addition, all participants with HbA1c values ≥6.5% (48 mmol/mol) were classified as having diabetes mellitus, regardless of their reply on the questionnaire.

Physical activity was measured by asking: 'Please indicate your levels of physical activity at the ages of 14, 30 and at your current age, on a scale from 1 to 10. "Physical activity" includes household chores and professional activities, as well as regular exercise and other physical activity, such as walking/hiking. Please mark (with an 'X') below the number that most accurately denotes your physical activity levels'. In this study, we used physical activity at current age, an instrument validated in middle-aged women living in Tromsø, Norway [20]. We recoded the 10-level physical activity scale into three categories: low (levels 1–3), moderate (levels 4–7) and high physical activity (levels 8–10).

Years of education was measured with the question: 'How many years of education have you completed? (Include any and all years in which you attended school or studied)'. We categorised this item into three levels: 0–9 years, 10–12 years and ≥13 years, which roughly corresponds to compulsory primary and lower secondary school, upper secondary school and higher education, respectively.

The NORRISK 2 model

The NORRISK 2 model is validated and intended to be used in primary prevention of CVD in the Norwegian population aged 40–79 years [18]. The model is used to estimate the 10-year risk (%) of hospitalisation with AMI (International Classification of Diseases [ICD-10] codes I21–22) as main or secondary diagnosis, death from ischaemic heart diseases (ICD-10 codes I20–25) as the underlying cause, or hospitalisation with CS (ICD-10 codes I60–61 and I63–64 except I63.6) as main or secondary diagnosis or death from CS as underlying cause [18]. The 10-year risk estimations are based on sex, age, total cholesterol, HDL-cholesterol, systolic blood pressure, smoking habits, anti-hypertensive treatment and family history of premature coronary heart disease [18]. All but family history of premature coronary heart disease were included in SAMINOR 2. In the model development, Selmer et al. treated death from other causes as competing risk. We used the age-specific, high-risk thresholds suggested by Selmer et al. [18] to identify individuals at high 10-year risk of fatal or non-fatal AMI or CS events who should be offered pharmacological treatment: ≥5% in the age group 45–54, ≥10% in the age group 55–64 and ≥15% in the age group 65–74 years. For those aged 40–44 and 75–79 years included in our study, we used the high risk threshold ≥5% and ≥15%, respectively.

Statistical analyses

Statistical analyses were performed using Stata v14.0 (StataCorp, College Station, TX). Ethnic differences in characteristics and age-specific cardiovascular risks were tested by two-sample *t*-tests with equal variance for continuous variables and Pearson's chi-square test (Tables I and II) and Fisher's exact test (Tables III and IV) for categorical variables. Test of trends across ordered groups (Tables I and II) was done with Wilcoxon's rank-sum test. Age standardisation of means and proportions was done separately in men and women using the direct method and having the sex-specific invited SAMINOR 2 sample in five-year age groups as the standard population.

Table I. Unadjusted sample characteristics of non-Sami and Sami men (*n*=2346): The SAMINOR 2 Clinical Survey (2012–2014).

Variables	Non-Sami, *n* = 1372	Sami, *n* = 974	*p*-value
Age (years), mean (*SD*)	59.4 (10.2)	58.8 (10.1)	0.11
40–54 years, % (*n*)	32.9 (451)	36.3 (353)	0.03
55–64 years, % (*n*)	31.4 (431)	32.4 (316)	
65–79 years, % (*n*)	35.7 (490)	31.3 (305)	
Total cholesterol (mmol/L), mean (*SD*)	5.42 (1.0)	5.52 (1.1)	0.03
Total cholesterol >5.0 mmol/L, % (*n*)	67.6 (928)	69.7 (679)	0.29
HDL-cholesterol (mmol/L), mean (*SD*)	1.29 (0.4)	1.24 (0.4)	<0.001
HDL-cholesterol <1.0 mmol/L, % (*n*)	17.9 (246)	21.6 (210)	0.03
Triglycerides (mmol/L), mean (*SD*)	1.80 (1.1)	1.94 (1.1)	0.002
Triglycerides >1.7 mmol/L, % (*n*)	44.1 (605)	51.2 (499)	0.001
Systolic blood pressure (mmHg), mean (*SD*)	134.9 (17.2)	134.3 (18.1)	0.39
Systolic blood pressure ≥140 mmHg, % (*n*)	34.4 (472)	33.8 (329)	0.75
Diastolic blood pressure (mmHg), mean (*SD*)	78.1 (9.4)	77.3 (9.9)	0.04
Diastolic blood pressure ≥90 mmHg, % (*n*)	11.0 (151)	11.4 (111)	0.77
Anti-hypertensive treatment (yes), % (*n*)	26.7 (366)	23.4 (228)	0.07
Current smoking (yes), % (*n*)	16.0 (220)	19.1 (186)	0.05
HbA1c[a] (%), mean (*SD*)	5.69 (0.6)	5.79 (0.8)	<0.001
Diabetes mellitus[b] (yes), % (*n*)	8.3 (114)	11.0 (107)	0.03
Waist circumference[c] (cm), mean (*SD*)	100.0 (10.6)	98.5 (10.8)	0.001
Waist circumference >102 cm, % (*n*)	35.0 (479)	31.8 (309)	0.11
Physical activity[d], mean (*SD*)	5.3 (2.0)	5.2 (2.2)	0.08
Low (1–3), % (*n*)	19.6 (263)	23.4 (220)	0.43
Moderate (4–7), % (*n*)	66.3 (892)	60.6 (569)	
High (8–10), % (*n*)	14.1 (190)	16.0 (150)	
Years of education[e], mean (*SD*)	12.0 (3.6)	11.7 (3.7)	0.14
0–9 years, % (*n*)	27.7 (369)	32.3 (302)	0.04
10–12 years, % (*n*)	33.1 (441)	31.0 (290)	
≥13 years, % (*n*)	39.2 (524)	36.7 (343)	

[a]Missing values: non-Sami, *n* = 3; Sami, *n* = 3.
[b]Diabetes mellitus: self-reported diabetes or HbA1c ≥6.5%.
[c]Missing values: non-Sami, *n* = 3; Sami, *n* = 2.
[d]Missing values: non-Sami, *n* = 27; Sami, *n* = 35.
[e]Missing values: non-Sami, *n* = 38; Sami, *n* = 39.
SD: standard deviation.

Comparisons of age-standardised means and proportions were done with a two-sample *t*-test with equal variance and a two-sample test for proportions, respectively, using the age-standardised estimates of means, proportions and standard deviations. We considered *p*-values <0.05 to be statistically significant.

In sensitivity analyses, three ethnic categories were compared with regard to mean risk scores and proportions at high risk: (1) those who reported 'Sami' on all 11 questions on ethnicity, (2) those who reported 'Sami' on at least one question and (3) those not reporting 'Sami' on any of the questions. We assessed whether region of residence modified mean risk scores and proportions at high risk (using the original ethnic categorisation) by merging municipalities that may be perceived as similar in terms of number of Sami inhabitants, Sami language users and geographical location: (1) Kautokeino and Karasjok; (2) Nesseby, Tana and Porsanger; and (3) Skånland, Evenes, Storfjord, Lyngen and Kåfjord. Additionally, in a separate set of analyses, we excluded participants on anti-hypertensive treatment (*n*=1382) to explore whether there were ethnic differences in non-users of anti-hypertensive medication.

Results

Of the 6004 SAMINOR 2 participants, 21 did not answer the questionnaire, 75 did not answer the questions on ethnicity, 26 had missing cholesterol values, three had missing systolic blood pressure, 193 reported angina pectoris, 244 reported AMI and 124 had missing information on smoking habits and were excluded. Thus, our analyses included 5318 individuals (44.1% of the sample were men and 40.8% were Sami), representing 42.7% of the invited sample.

In men, statistically significant differences were found in mean total cholesterol, the distribution of HDL-cholesterol and triglycerides, mean diastolic blood pressure, HbA1c, proportions with diabetes mellitus, mean waist circumference and the level of

Table II. Unadjusted sample characteristics of non-Sami and Sami women (*n*=2972): The SAMINOR 2 Clinical Survey (2012–2014).

Variables	Non-Sami, *n*=1777	Sami, *n*=1195	*p*-value
Age (years), mean (*SD*)	58.6 (10.6)	57.9 (10.2)	0.08
40–54 years, % (*n*)	37.0 (657)	37.7 (451)	0.10
55–64 years, % (*n*)	30.4 (541)	34.0 (406)	
65–79 years, % (*n*)	32.6 (579)	28.3 (338)	
Total cholesterol (mmol/L), mean (*SD*)	5.59 (1.0)	5.61 (1.1)	0.55
Total cholesterol >5.0 mmol/L, % (*n*)	71.1 (1264)	72.1 (861)	0.59
HDL-cholesterol (mmol/L), mean (*SD*)	1.55 (0.5)	1.45 (0.4)	<0.001
HDL-cholesterol <1.3 mmol/L, % (*n*)	25.3 (450)	32.7 (391)	<0.001
Triglycerides (mmol/L), mean (*SD*)	1.53 (0.9)	1.66 (0.9)	<0.001
Triglycerides >1.7 mmol/L, % (*n*)	33.6 (597)	39.7 (475)	0.001
Systolic blood pressure (mmHg), mean (*SD*)	130.6 (18.3)	129.6 (19.3)	0.14
Systolic blood pressure ≥140 mmHg, % (*n*)	27.0 (479)	26.5 (317)	0.80
Diastolic blood pressure (mmHg), mean (*SD*)	72.4 (9.0)	71.8 (9.3)	0.08
Diastolic blood pressure ≥90 mmHg, % (*n*)	3.6 (64)	3.8 (45)	0.82
Anti-hypertensive treatment (yes), % (*n*)	26.7 (475)	26.2 (313)	0.75
Current smoking (yes), % (*n*)	20.1 (357)	22.5 (269)	0.11
HbA1c[a] (%), mean (*SD*)	5.66 (0.6)	5.71 (0.5)	0.05
Diabetes mellitus[b] (yes), % (*n*)	8.3 (148)	10.0 (120)	0.11
Waist circumference[c] (cm), mean (*SD*)	92.7 (12.1)	93.4 (12.0)	0.13
Waist circumference >88 cm, % (*n*)	61.7 (1093)	66.1 (789)	0.01
Physical activity[d],mean (*SD*)	5.6 (2.1)	5.3 (2.1)	<0.001
Low (1–3), % (*n*)	15.5 (257)	22.8 (261)	<0.001
Moderate (4–7), % (*n*)	64.8 (1076)	60.7 (696)	
High (7–10), % (*n*)	19.7 (328)	16.5 (189)	
Years of education[e],mean (*SD*)	12.4 (4.0)	12.7 (4.4)	0.09
0–9 years, % (*n*)	26.0 (445)	27.8 (316)	0.28
10–12 years, % (*n*)	30.3 (517)	23.2 (263)	
≥13 years, % (*n*)	43.7 (747)	49.0 (556)	

[a]Missing values: non-Sami, *n*=1; Sami, *n*=6.
[b]Diabetes mellitus: self-reported diabetes or HbA1c ≥6.5%.
[c]Missing values: non-Sami, *n*=5; Sami, *n*=2.
[d]Missing values: non-Sami, *n*=116; Sami, *n*=49.
[e]Missing values: non-Sami, *n*=68; Sami, *n*=60.
SD: standard deviation.

Table III. Mean 10-year risk and proportion of participants at high risk of acute myocardial infarction (AMI) or cerebral stroke (CS) in non-Sami and Sami men aged 40–79 years, *n*=2346: The SAMINOR 2 Clinical Survey (2012–2014).

Age		Non-Sami (*n*=1372)			Sami (*n*=974)			
		n			*n*			*p*-value
40–54 years	Mean (*SD*)	451	3.6	(2.6)	353	4.0	(3.1)	0.06
	≥5% (*n*)		21.5%	(97)		23.2%	(82)	0.61
55–64 years	Mean (*SD*)	431	8.8	(4.2)	316	9.5	(5.0)	0.06
	≥10% (*n*)		26.7%	(115)		36.1%	(114)	0.006
65–79 years	Mean (*SD*)	490	18.0	(6.4)	305	18.1	(6.7)	0.81
	≥15% (*n*)		63.5%	(311)		61.3%	(187)	0.55
Total								
Crude mean (*SE*)		1372	10.4	(0.21)	974	10.2	(0.25)	0.55
Age-standardised mean (*SE*)		1372	9.5	(0.10)	974	9.8	(0.13)	0.05
% with high risk[a] (*n*)			38.1%	(523)		39.3%	(383)	0.56
Age-standardised % with high risk			35.6%			37.5%		0.35

[a]Proportions with 10-year risk of AMI or CS ≥5%, ≥10% and ≥15% in age groups 40–54, 55–64 and 65–79 years, respectively.
SD: standard deviation.

education (Table I). Compared with non-Sami men, Sami men had a more unfavourable distribution (except for waist circumference and diastolic blood pressure).

In women, Sami had statistically significantly lower HDL-cholesterol and physical activity level and higher triglycerides and higher proportions with waist circumference >88 cm (Table II).

Table IV. Mean 10-year risk and proportion of participants at high risk of acute myocardial infarction (AMI) or cerebral stroke (CS) in non-Sami and Sami women aged 40–79 years, *n*=2972: The SAMINOR 2 Clinical Survey (2012–2014).

Age		Non-Sami (*n*=1777)			Sami (*n*=1195)			
		n			*n*			*p*-value
40–54 years	Mean (*SD*)	657	1.3	(1.2)	451	1.4	(1.3)	0.24
	≥5% (*n*)		2.3%	(15)		2.7%	(12)	0.70
55–64 years	Mean (*SD*)	541	5.0	(3.0)	406	4.7	(2.8)	0.27
	≥10% (*n*)		5.2%	(28)		5.4%	(22)	0.88
65–79 years	Mean (*SD*)	579	12.0	(5.4)	338	12.2	(5.3)	0.68
	≥15% (*n*)		24.7%	(143)		28.1%	(95)	0.28
Total								
Crude mean (*SE*)		1777	5.9	(0.14)	1195	5.6	(0.16)	0.12
Age-standardised mean (*SE*)		1777	5.8	(0.07)	1195	5.9	(0.08)	0.44
% with high risk[a] (*n*)			10.5%	(186)		10.8%	(129)	0.78
Age-standardised % with high risk			11.0%			12.6%		0.19

[a]Proportions with 10-year risk of AMI or CS ≥5%, ≥10% and ≥15% in age groups 40–54, 55–64 and 65–79 years, respectively.
SD: standard deviation.

In the total analytical population, disregarding ethnicity, the age-standardised mean 10-year risk of AMI or CS and the proportion of participants at high risk were 9.6% and 36.4%, respectively, for men, and 5.8% and 11.6%, respectively, for women (results not shown).

The mean 10-year risk of AMI or CS by age for Sami and non-Sami men and women is displayed in Figure 1. Overall, there were no ethnic differences in the 10-year risk of AMI or CS (Tables III and IV, and Figure 1). This was due to only minor ethnic differences in the distribution of the cardiovascular risk factors included in the NORRISK 2 model. However, among men aged 55–64 years, more Sami than non-Sami had a high 10-year risk of AMI or CS (36.1% vs. 26.7%; p=0.006). Sami men in this age group had a somewhat unfavourable distribution in most of the risk factors included in the model (results not shown). Additionally, the age-standardised 10-year mean risk for non-Sami and Sami men was borderline significant (9.5 vs. 9.8; p=0.05).

Overall, the sensitivity analyses did not show markedly different results by ethnicity or region of residence. Among non-users of anti-hypertensive medication, Sami women had lower systolic (125.9 vs. 127.9 mmHg; p=0.008) and diastolic (71.0 vs. 71.9 mmHg; p=0.02) blood pressure than non-Sami women (results not shown).

Discussion

We observed no overall differences in the 10-year risk of AMI or CS, as estimated by the NORRISK 2 model, between Sami and non-Sami participants. This was due to minor ethnic differences in the distribution of the cardiovascular risk factors included in the model. Overall, 36.4% of men and 11.6% of women had a high 10-year risk of AMI or CS.

Our results of an overall similar distribution of cardiovascular risk factors between Sami and non-Sami participants agree with previous studies from Norway, Sweden and Finland [9-10, 13, 21-25]. However, we found a somewhat unfavourable distribution of triglycerides, physical activity (women only), waist circumference (women only) and diabetes mellitus (men only) in Sami compared to non-Sami participants. To what extent ethnic discrepancies in these risk factors may contribute to a different risk of AMI or CS in Sami relative to non-Sami populations, is uncertain.

A publication with data from 1974/1975–1989 found that Sami men in Finnmark had a similar risk of AMI to Norwegian men, but indicated a 50% increased incidence of cerebrovascular disease [12]. In 1970–1998, Sami men and women (based on census data) living north of the Arctic Circle in Norway had higher mortality from cerebrovascular diseases, that is, 14% and 28% higher mortality rates in men and women, respectively. However, male reindeer herders had a lower risk of death from ischaemic heart disease than other Sami and non-Sami populations, and this was also the case, to some extent, for cerebrovascular disease [14]. In a follow-up study from 1977/1978–1992, including men aged 35–52 years in Finnmark, 67% and 52% lower mortality rates were found for ischaemic heart disease and total CVD, respectively, in Sami compared to non-Sami men [15].

Among men in Sweden, the overall incidence of hospitalisations due to cerebrovascular disease was higher in Sami populations but lower in Sami

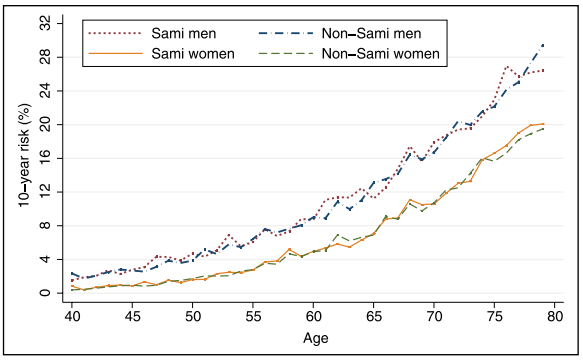

Figure 1. Mean 10-year risk of fatal or non-fatal acute myocardial infarction or cerebral stroke by age for non-Sami and Sami men (n=2346) and women (n=2972): The SAMINOR 2 Clinical Survey (2012–2014).

reindeer herders than in a regional non-Sami reference population [26]. Death from AMI was also higher in Sami women but not in female Sami reindeer herders [26,27], among whom the incidence of AMI was lower and cerebrovascular diseases higher [26]. In Finland, no difference in mortality from cerebrovascular disease between Sami and non-Sami populations have been reported [28], whereas a lower mortality from ischaemic heart disease has been observed in Sami women compared to a non-Sami female reference population [28,29].

Overall, it seems like Sami men and women may have a somewhat increased risk of ischaemic heart disease and cerebrovascular disease that does not necessarily apply to male reindeer herders, who might have reduced risk due to assumed higher physical activity levels [30]. This seems to contrast with what we found in our study, that is, no overall ethnic difference in 10-year risk of AMI and CS in men and women. We did find that Sami men aged 55–64 years had a higher 10-year risk of AMI or CS than their non-Sami counterparts. However, this might be a chance finding.

The inferior cardiovascular health observed among indigenous peoples relative to non-indigenous peoples in North America may be due to lower socio-economic status and poorer access to quality health care among the former [4]. A publication from Norway found that differences in smoking habits, systolic blood pressure, serum cholesterol and body mass index explained 72% and 56% of the absolute and relative educational gradients, respectively, in CVD mortality [31]. Assuming that the educational gradient in Sami and non-Sami populations is the same, the most plausible explanation for the similar levels of risk factors and risk of AMI or CS observed in our study may be that Sami and non-Sami populations in Norway differ little with regard to education levels (Tables I and II). However, Sjölander et al. found a somewhat higher incidence of cerebrovascular diseases and AMI mortality in Sami compared to non-Sami women in Sweden, which was not explained by education or income [26]. Thus, variables other than traditional socio-economic ones may also be relevant in explaining the disparities in the risk of CVD between Sami and non-Sami populations. Equal access to health care has also been put forward as a plausible explanation for the small differences in health and risk factors between Sami and non-Sami populations [32]. The fact that we observed no difference in anti-hypertensive treatment indicates that ethnic discrepancies in access to CVD treatment may not be an issue.

This study has some strengths. The relatively large sample of Sami and non-Sami participants from both coastal and inland regions, individual information on ethnic background and several biological markers and clinical measurements enabled an in-depth analysis of the total risk of AMI or CS. The NORRISK 2 model has a major advantage, as it takes into account competing risk and incorporates both fatal and non-fatal end points. Furthermore, the NORRISK 2 model was designed and validated for those aged 40–79 years in the general population of Norway [18], which is the same age range as that included in SAMINOR 2.

There are also some limitations. We assumed a similar CVD aetiology among Sami and non-Sami participants, which may not be correct [15]. Moreover, our risk estimations are most likely overestimated, as risk factor levels are expected to decrease in the future [16]. We did not have data on family history of premature ischaemic heart diseases and thus could not include it in the model. However, unpublished results from the SAMINOR 1 Survey, restricted to the same municipalities as SAMINOR 2, showed no ethnic differences in this regard (results not shown). We were not able to assess the 10-year risk of AMI or CS among Sami reindeer herders alone due to lack of information on occupation and association with reindeer husbandry. Finally, the NORRISK 2 model does not take into account all measured cardiovascular risk factors (e.g. triglycerides, physical activity, waist circumference and diabetes mellitus).

Only 10 municipalities were included in SAMINOR 2. Generalisations to the entire Sami or non-Sami populations in Norway are therefore not justified. Our analyses included 42.7% of the invited sample, with more female than male participants, though participation in both sexes increased with age, as in other studies [33]. Therefore, results for the youngest age group, particularly for men, are

uncertain. We do not know the response rate by ethnic group due to lack of ethnic information in national registries. However, participation was highest in Kautokeino, Nesseby and Tana, where Sami are in the majority, and low in some municipalities where they are in the minority. This may have led to an over-representation of Sami participants from majority areas compared to Sami from minority areas and non-Sami. Whether this affected the results of this study is, however, unknown.

This study provides updated knowledge about the cardiovascular health of Sami and non-Sami populations in rural Northern Norway, which is needed, as the majority of comparable studies were conducted in Finnmark during 1974–1989. Furthermore, a cardiovascular risk assessment that combines the impact of several risk factors simultaneous and estimates the 10-year risk of AMI or CS has not been conducted previously in Sami and non-Sami populations.

Conclusion

We observed only minor ethnic differences in several risk factors for CVDs. Based on results from the NORRISK 2 model, we observed no overall differences in the 10-year risk of AMI or CS between the Sami and non-Sami populations living in 10 rural municipalities in Northern Norway. Applying the thresholds of Selmer et al. [18], 36.4% of men and 11.6% of women in this population were at high risk of AMI or CS.

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Paper 2

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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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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 mm Hg 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.

Strengths and limitations of this study

- 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, that is, 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 to 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.

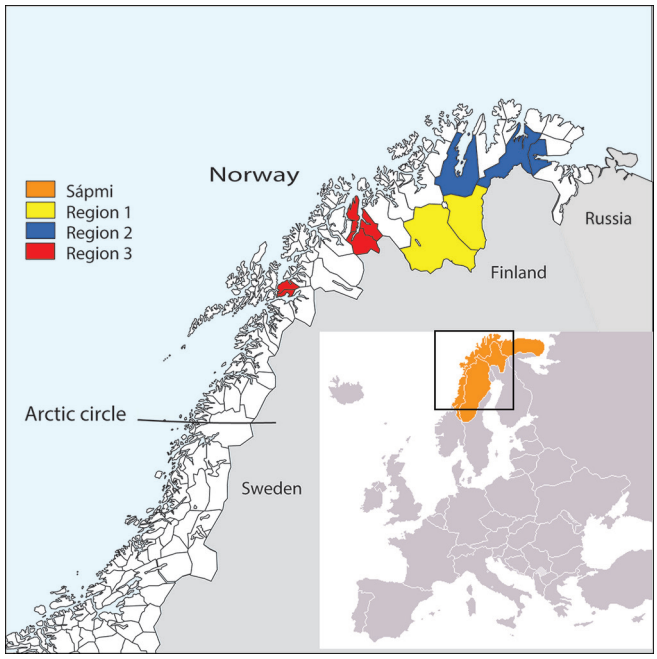

Figure 1 Inhabitants aged 40–79 years living in these 10 municipalities in the Norwegian part of Sápmi were invited to the SAMINOR 1 and SAMINOR 2 surveys. Region 1 includes Kautokeino and Karasjok, region 2 includes Nesseby, Tana and Porsanger, and region 3 includes Kåfjord, Lyngen, Storfjord, Skånland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

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 years 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 1–7 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 who attended the clinical examinations in SAMINOR 1 and SAMINOR 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 SAMINOR 2, respectively, wherein 3249 participated in both 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 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 to 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 min rest, three recordings were made at 1 min 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 mm Hg, or their diastolic blood pressure was ≥90 mm Hg, 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 min, 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 V.15.0. Sample characteristics were given by sex for Sami and non-Sami in SAMINOR 1 and SAMINOR 2: means (SD) 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, that is, the sex-specific mean ages in the overall sample. Two-way graphs illustrate how cardiovascular risk factors varied by age, ethnicity and survey (online 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 1–10 questions and (3) those who did not report Sami on any of the questions (non-Sami) (online supplementary tables S1 and S2).
2. Using the original ethnic categorisation, we adjusted for geographical regions: (1) Kautokeino and Karasjok, (2) Nesseby, Tana and Porsanger, (3) Kåfjord, Lyngen, Storfjord, Skånland and Evenes (online 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

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 television or other sedentary activity, % (n)	21.3 (415)	27.7 (297)	–	–
Walking, bicycling or moving around in other ways at least 4 hours/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 to 10, mean (SD)*	‡	‡	5.6 (2.1)	5.2 (2.2)
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 SAMINOR 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 television or other sedentary activity, % (n)	20.2 (351)	24.3 (254)	–	–
Walking, bicycling or moving around in other ways at least 4 hours/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 to 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 SAMINOR 2.
‡Question not posed.

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 mm Hg (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.

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 mm Hg (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).

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

*ß coefficients are estimated by linear generalised estimating equation regression models and adjusted for age and age^2. ORs are estimated by logistic generalised estimating equation regression models and adjusted for age.
*P value for interaction <0.05, only ethnicity-specific estimations are reported.
†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.
‡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 years.
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 subjects . NORRISK 2 score was missing for 193 subjects. Excluding missing values did not ange the results.
AMI, acute myocardial infarction; CS, cerebral stroke; HDL, high-density lipoprotein.

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

β coefficients are estimated by linear generalised estimating equation regression models and adjusted for age and age^2. ORs are estimated by logistic generalised estimating equation regression models and adjusted for age.
*P value for interaction <0.05, only ethnicity-specific estimations are reported.
†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.
‡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 years.
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.
AMI, acute myocardial infarction; CS, cerebral stroke; HDL, high-density lipoprotein.

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 (online supplementary tables S1 and S2) and when adjusting for region (online supplementary table S3).

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 with 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, 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 ischaemic 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 healthcare 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.

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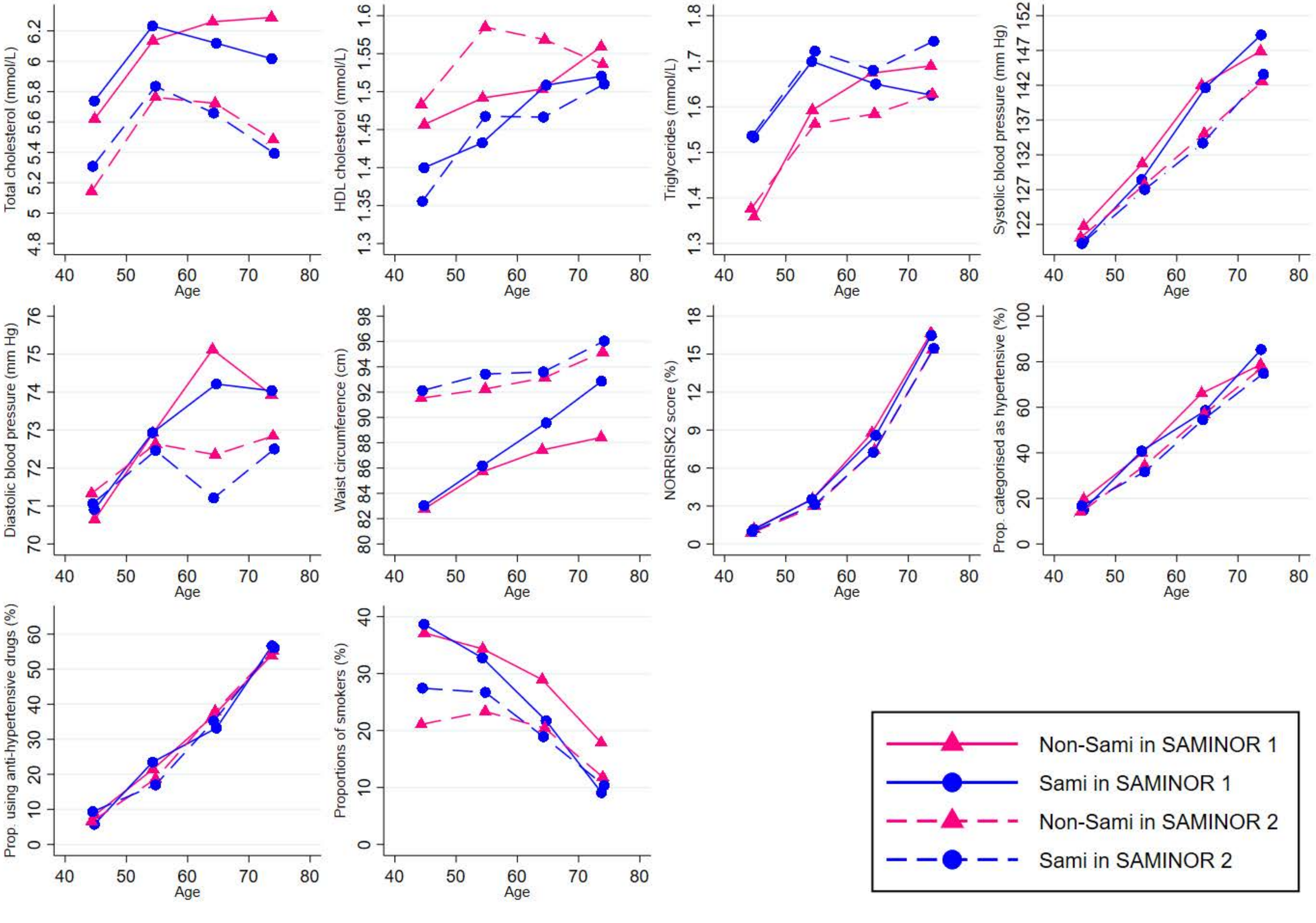
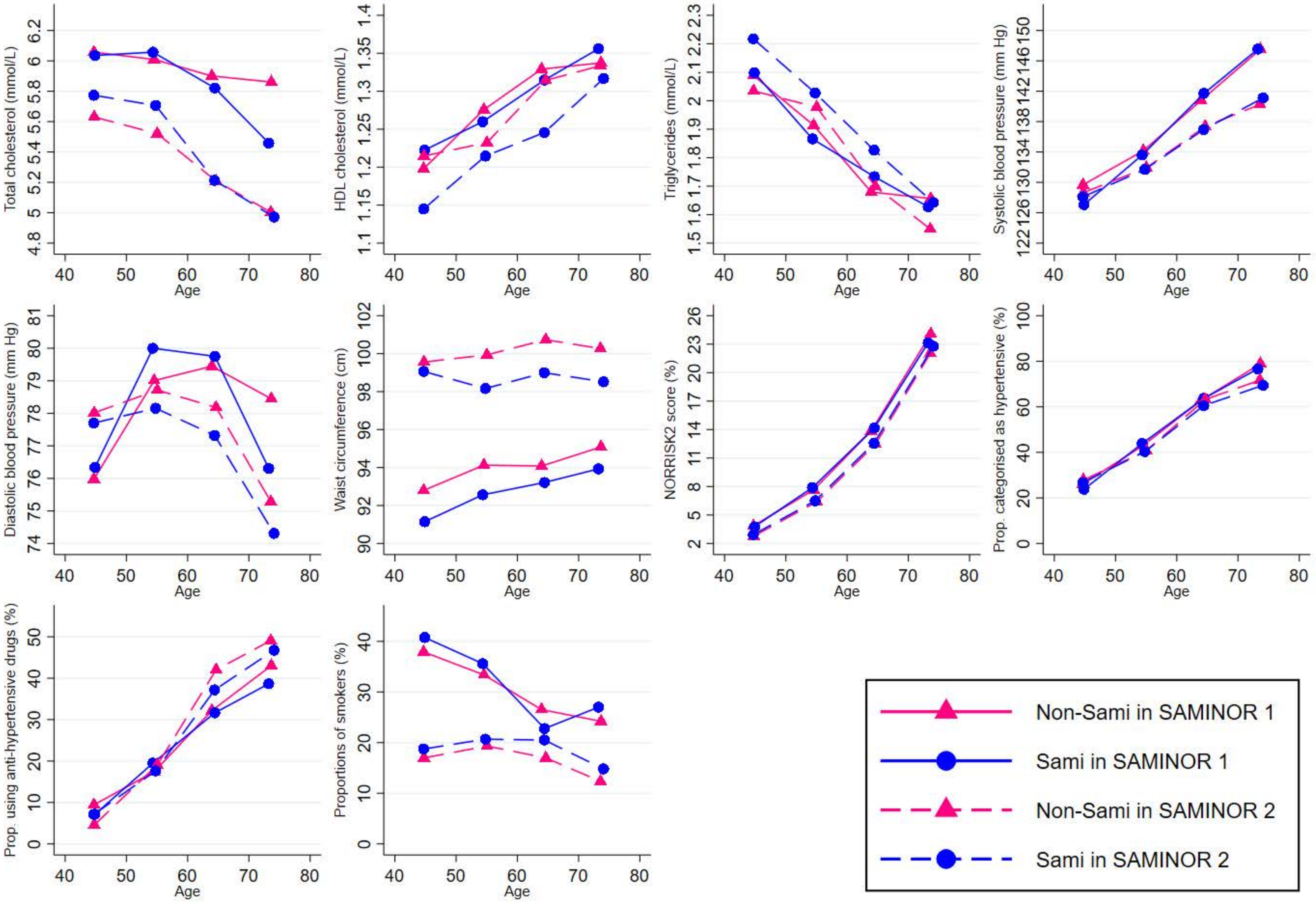
Supplementary Figure S1. Cardiovascular risk factors in women according to survey, ethnicity and mean age in 10-year age groups



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



Paper 3

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openheart

Coronary heart disease and stroke in the Sami and non-Sami populations in rural Northern and Mid Norway—the SAMINOR Study

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ABSTRACT

Background Previous studies have suggested that Sami have a similar risk of myocardial infarction and a possible higher risk of stroke compared with non-Sami living in the same geographical area.

Design Participants in the SAMINOR 1 Survey (2003–2004) aged 30 and 36–79 years were followed to the 31 December 2016 for observation of fatal or non-fatal events of acute myocardial infarction (AMI), coronary heart disease (CHD), ischaemic stroke (IS), stroke and a composite endpoint (fatal or non-fatal AMI or stroke).

Aim Compare the risk of AMI, CHD, IS, stroke and the composite endpoint in Sami and non-Sami populations, and identify intermediate factors if ethnic differences in risks are observed.

Methods Cox regression models.

Results The sex-adjusted and age-adjusted risks of AMI (HR for Sami versus non-Sami 0.99, 95% CI: 0.83 to 1.17), CHD (HR 1.03, 95% CI: 0.93 to 1.15) and of the composite endpoint (HR 1.09, 95% CI: 0.95 to 1.24) were similar in Sami and non-Sami populations. Sami ethnicity was, however, associated with increased risk of IS (HR 1.36, 95% CI: 1.10 to 1.68) and stroke (HR 1.31, 95% CI: 1.08 to 1.58). Height explained more of the excess risk observed in Sami than conventional risk factors.

Conclusions The risk of IS and stroke were higher in Sami and height was identified as an important intermediate factor as it explained a considerable proportion of the ethnic differences in IS and stroke. The risk of AMI, CHD and the composite endpoint was similar in Sami and non-Sami populations.

Key questions

What is already known about this subject?

► Previous studies have found similar risk of coronary heart disease and a possible higher risk of stroke in Sami compared to non-Sami populations.

What does this study add?

► In the SAMINOR 1 Survey (2003–2004), the Sami population has a higher risk of stroke and ischaemic stroke compared with non-Sami. Differences in height explained more than conventional risk factors.

How might this impact on clinical practice?

► Our findings have predominantly public health relevance. The clinical relevance depend on the interpretation of height and ethnicity as risk predictors of ischemic stroke and stroke.

INTRODUCTION

Through 2001 to 2014, the incidence of non-fatal acute myocardial infarction (AMI) and fatal coronary heart disease (CHD) declined in Norway in both sexes,[1] even among people aged 25–44 years.[1] Between 1994 and 2012, a decline was observed in the incidence of CHD[2] and stroke[3] in the largest city in Northern Norway, which is close to the regions included in the present study. The decline in the incidence of CHD and stroke was mainly driven by the improvement of cardiovascular risk factors[3]; for fatal CHD, fewer out-of-hospital sudden deaths and hospitalisations with severe myocardial infarction (MI) contributed to the decline.[2]

The Sami people live across Norway, Sweden, Finland and on the Kola Peninsula in the Russian Federation. In Norway, the Sami are acknowledged as indigenous people, and the majority live in Northern Norway, together with the Kven people and Norwegian majority population. The Kven people are descendants of Finnish-speaking immigrants who arrived in the 1700s and 1800s from northern Sweden and Finland.[4] As with other indigenous people, the Norwegian government imposed harsh assimilation policies on the Sami from 1850 until 1960,[5] when a revitalisation of Sami culture and languages began, but many had already given up using Sami languages. In the 1970 population census, information on Sami and Kven ethnicities was collected in selected areas of Northern Norway, and it was estimated that around 40 000 people in Norway were Sami.[6]

Unlike other indigenous populations,[7] the somatic health in Sami has been shown to be similar to that in the non-Sami population in the same geographical areas.[8]

Two studies regarding CHD mortality in Sami, covering roughly the same time period but different geographical areas, show contradicting mortality rates,[9 10] whereas similar incidence of MI was observed in Sami and non-Sami in 1973/1974 through 1989 in Finnmark County, in Northern Norway.[11] With regards to stroke, higher mortality[9] and possibly higher incidence[11 12] were observed in Sami compared with their reference populations in the years 1974–1998. Moreover, several studies observed lower average heights in Sami than in non-Sami populations,[10 13 14] and a possible inverse association between height and degree of Sami affiliation.[13] Moreover, an increase in height has been found to be inversely associated with CHD and stroke,[15 16] which has also been observed in Finnmark,[12] however, for MI, this was only found in women.[11]

We have previously reported overall similar levels of cardiovascular risk factors in Sami and non-Sami populations in rural Northern Norway.[17 18] However, it is not known whether the incidence of cardiovascular disease (CVD) differs in Sami and non-Sami in this population. Thus, the aim of this study was to compare the risk of fatal or non-fatal AMI, CHD, ischaemic stroke (IS), stroke and a composite endpoint (AMI or stroke) in Sami and non-Sami populations, and identify intermediate factors if ethnic differences in risks are observed.

METHODS

The SAMINOR 1 Survey (SAMINOR 1) was conducted in 2003–2004 and was the first of three population-based cross-sectional surveys, which together constitute the SAMINOR Study. The SAMINOR 1 was initiated due to limited knowledge about the health of the Sami population, and it was conducted by the Centre for Sami Health Research at UiT the Arctic University of Norway, together with the Norwegian Institute of Public Health. There is no registry of ethnicity in Norway; thus, SAMINOR 1 invited all inhabitants aged 30, 36–78/79 years (birth cohorts 1925–1967 and 1973 in 2003; birth cohorts 1925–1968 and 1974 in 2004) residing in 24 selected municipalities of Northern and Mid Norway, all of which had a considerable Sami population according to the 1970 census[6] and local knowledge. SAMINOR 1 included a clinical examination and self-administrated questionnaires, which included questions related to ethnicity. In total, 27 987 individuals were invited and 16 865 (60.3%) attended SAMINOR 1.[19]

Clinical examination

Trained staff conducted the clinical examinations, during which waist circumference was measured at the umbilicus to the nearest centimetre when participants were standing. Height in centimetres and weight in kilograms were measured with an electronic height and weight scale (DS-102, Dongsahn Jenix, Seoul, Korea) to the nearest 0.1 decimal, with the participant standing and wearing no shoes. A digital oscillometric device (DINAMAP-R, Critikon, Tampa, Florida, USA) was used to measure blood pressure three times at 1-minute intervals, and the average of the last two measurements was reported. Those who used antihypertensive medications or had systolic or diastolic blood pressure ≥140/90 mm Hg, respectively, were categorised as hypertensive. Non-fasting venous blood samples were taken while participants were resting. The samples were centrifuged within 30 min, separated within 2 hours and sent by overnight post to Ullevål University Hospital in Oslo, Norway, where total cholesterol, high-density lipoprotein (HDL) cholesterol, glucose and triglycerides were measured with an enzymatic method (Hitachi 917 autoanalyser, Roche Diagnostics, Switzerland).

Self-administered questionnaires

There is no registry on ethnicity in Norway and health surveys rely on self-reported ethnic information. There is no consensus on how ethnicity should be operationalised in health research except that it is recommended that several markers of ethnicity are applied, as ethnicity is a multifaceted concept.[20–22] The following 11 questions provided information about ethnicity: (1) *What language do/did you, (2) your mother, (3) your father, (4–7) your grandparents (all four) speak at home? (8) What is your, (9) your mother's, (10) your father's ethnic background? (11) What do you consider yourself to be?* To each of the questions, one or more of the following alternatives could be ticked: Sami, Kven, Norwegian and other (specify). Participants who (1) considered themselves as Sami or ticked 'Sami' as their own ethnic background, and (2) either spoke the Sami language themselves, or had at least one parent or grandparent who used it at home, were categorised as Sami. All others were categorised as non-Sami. Sensitivity analyses were performed using an alternative ethnic categorisation: (1) high Sami affiliation, that is, reported Sami to all 11 questions (n=1385), (2) some Sami affiliation, that is, reported Sami in 1–10 questions (n=3168), and (3) no Sami affiliation, that is, did not answer Sami in any of the questions (n=9234).

A family history of premature MI was considered present if first-degree relatives (parents, siblings or children) had MI before the age of 60 years. Disease status was obtained by asking if participants had MI, stroke/brain haemorrhage, angina pectoris and diabetes mellitus, and, if so, then age at which the condition first occurred. Participants with self-reported diabetes mellitus or glucose ≥11.1 mmol/L were categorised as having diabetes. Participants reported whether they were current, previous, or never users of antihypertensive or lipid-lowering medications. Previous and never users were merged with those who failed to reply to the question (missing values for antihypertension medication, n=142) and categorised as non-users.

Information on education and behaviour were collected through the questions: *How many years of*

education have you completed? Are you currently, or were you previously a daily smoker? (Response alternatives: currently, previously, or never – previous smokers and never-smokers were merged into non-smokers); *what was your alcohol consumption in the last year?* (Response alternatives: never consumed alcohol, not during the last year, a few times during the last year, one time per month, two to three times per month, one time per week, two to three times per week or four to seven times per week). We created a three-level category, consisting of (1) never consumers together with those who did not consume in the last year, (2) those who consumed less than weekly and (3) those who consumed at least weekly. Leisure-time physical activity in the last year was measured using the 'Saltin-Grimby' four-level scale.[23] Being sedentary during leisure time was defined as: reading, watching television or other sedentary activity, whereas the remaining three alternatives, all of which compromised some physical activity, were merged to describe those who were physically active during leisure time. The estimated 10-year risk of fatal or non-fatal AMI or stroke was computed using the NORRISK 2 score.[24]

Data from national registries

Linkage to the following national registries was done using each participant's unique, 11-digit national identity number: the Norwegian Cause of Death Registry provided dates and underlying causes of death for 2003–2016; the Cardiovascular Disease Project[25] provided information on all hospitalisations with CVD as the main or secondary discharge code for 1993–2009; the Norwegian Patient Registry provided records from all hospitalisations with discharge codes including CVD as the main or secondary diagnoses for 2010–2016; and Statistics Norway provided information on emigrations.

The five endpoints included the following codes from the International Classification of Disease, 10th Revision: (1) AMI: I21–22 as the hospital discharge code, or I20–25 as the underlying cause of death code. (2) CHD: I20–25 as the hospital discharge code or the underlying cause of death code. (3) IS: I63 as the hospital discharge code or the underlying cause of death code. (4) Stroke: I60–61 and I63–64 (except I63.6) as hospital discharge code or underlying cause of death code. (5) Composite endpoint, representing the NORRISK 2 endpoint: I21–22 and I60–61, I63–64 (except I63.6) as the hospital discharge code, or I20–25 and I60–61, I63–64 (except I63.6) as the underlying cause of death code.[24]

Study sample

Among the 16 865 individuals who attended the SAMINOR 1, we excluded 87 who did not consent to have their data linked to registries; 1027 who did not complete one or both of the questionnaires; 35 who did not attend the clinical examination; and 51 who had missing information on ethnicity. Furthermore, we excluded 1580 with self-reported angina pectoris, MI and stroke (the two latter have moderate to high agreement in this population when validated against hospital discharge diagnoses)[26] and 151 with CHD or stroke as main or secondary hospital discharge code prior to participation in SAMINOR 1. Due to missing values in conventional risk factors (n=128) and height (n=19), the final sample consisted of 13 787 individuals (49.3% of the invited sample).

Statistical analyses

Sample characteristics are given for each stratum of sex and ethnicity, as mean values with SD or as proportions with numbers. The instantaneous hazards in Sami and non-Sami were compared by Cox regression models using age as the time axis,[27 28] whereby each model was adjusted for age. In model 1, the risk estimates for ethnicity were adjusted for age and sex. We then adjusted for potential intermediate factors: first for height per 5 cm (model 2), and then for conventional risk factors included in the risk prediction model NORRISK 2 score,[24] systolic blood pressure, total cholesterol, HDL cholesterol, current smoking, use of antihypertensive medication and whether one or two family members had a history of premature MI (model 3). Due to the potential for non-linearity in risk for the continuous variables,[29] we re-ran model 3 (and model 4 in sensitivity analyses) while allowing for multiple fractional polynomials using the 'mfp' function in STATA, an extension of the conventional polynomial model allowing for flexible parametrisation of a continuous predictor. The results from these models showed that only systolic blood pressure could be transformed using different power and degree of freedom, but only in models with AMI, CHD and the composite endpoint. Importantly, the β-coefficients for ethnicity did not differ in the linear models compared with the models that included the transformed variable, thus, linear models were chosen for all endpoints.

For all endpoints, we assessed the presence of interaction by including product terms with ethnicity and sex, respectively, in separate models for each covariate included in models 2 and 3. In the presence of interaction (AMI: sex and antihypertensive medication; stroke and composite endpoint: sex and height), we included a product term in the models. The proportional hazard assumption was assessed by checking if the Schoenfeld residuals were independent of attained age at a 5% significance level, and if necessary, by visual inspection of log-minus-log graphs. If a covariate violated the proportional hazards assumption, then it was included as a time-varying covariate.

Participants in the study were followed from the date of enrolment in SAMINOR 1 until the date of the first non-fatal or fatal event of the specific CVD endpoint, death from other causes, emigration or end of follow-up (31 December 2016), whichever occurred first. A two-sided p-value <0.05 was considered statistically significant. All statistical analyses were performed using STATA V.15.1. Figure 1 was made using the open-source software R V.3.6.0 (Foundation for Statistical Computing, Vienna, Austria).

We conducted the following sensitivity analyses:

1. We additionally adjusted for waist circumference, diabetes, physical activity in leisure time, alcohol consumption and years of education (model 4), but in a smaller sample due to missing values in these covariates (n=12 078, results not shown).
2. We performed Fine & Gray competing risk survival analyses with deaths from other causes as competing events, as previous studies have found somewhat higher rates of total mortality and violent death in Sami than non-Sami populations.[9]
3. Models 1–3 were repeated using the alternative ethnic categorisations.
4. To account for potential geographical heterogeneity, we adjusted for regions in all three models, using region 1 (see table 1 for the definition of regions) as reference.

RESULTS

The final sample constituted of 21.7% Sami and more women (53.7%) than men. The age distribution was similar in Sami and non-Sami, both in men and women. The crude mean values for height showed that Sami men and women in this sample were 6.0 and 5.7 cm shorter than non-Sami men and women, respectively (both p<0.001, age-adjusted linear regression, results not shown). A higher proportion of Sami participants reported a family history of premature MI, and a higher proportion reported abstaining from alcohol or a seldom alcohol consumption. A higher proportion of Sami than non-Sami men reported being current smokers, and Sami men had a somewhat higher mean estimated 10-year risk of AMI or stroke. In this sample, Sami women had a somewhat lower mean systolic blood pressure than non-Sami women, whereas a higher proportion of non-Sami was categorised as hypertensive. Additionally, a lower proportion of Sami women reported being physically active in their leisure time (table 1).

The median follow-up time and age for the composite endpoints were 13.0 and 65.2 years, respectively. The number of fatal and non-fatal events by investigated endpoints were as follows: AMI, 102 and 672; CHD, 89 and 1882; IS, 9 and 423; stroke, 36 and 525; and the composite endpoint, 129 and 1130, respectively. Among censored cases, there were 101 participants who emigrated during follow-up.

For all endpoints considered, we found no statistically significant interactions between ethnicity and height, between ethnicity and sex, or between ethnicity and year of birth. Although the estimates for men and women were not different in Sami and non-Sami, we included the sex-specific and ethnicity specific incidence rates per 1000 person-years in a supplementary table (online supplementary table 1).

The number of cases, person-years and incidence rates (per 1000) by ethnic group are listed in figure 1. For each

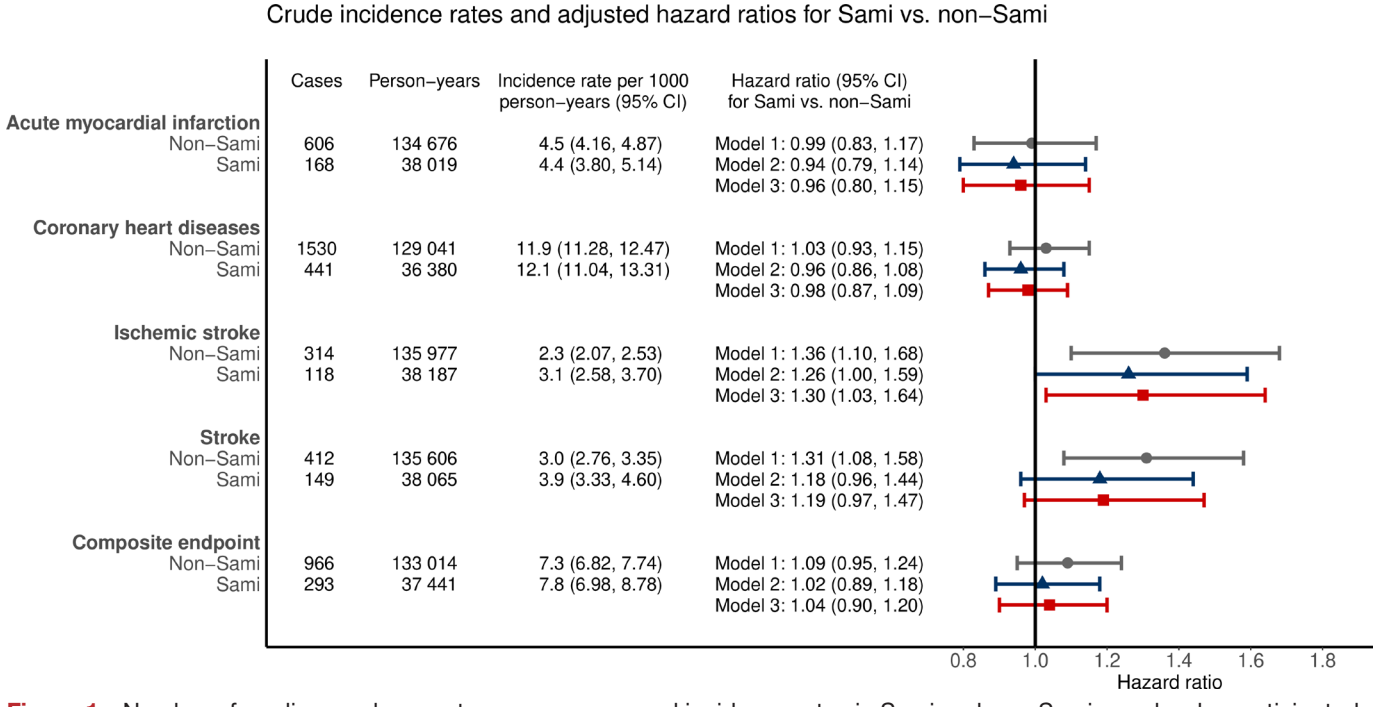

Figure 1 Number of cardiovascular events, person-years and incidence rates in Sami and non-Sami people who participated in the SAMINOR 1 Survey in 2003/2004 and were followed to the end of 2016. The forest plot gives the adjusted risk (HR with 95% CI) in Sami (n=2990) for different cardiovascular endpoints using non-Sami (n=10 797) as reference and age as the time axis in Cox regressions models, adjusting for the following covariates: model 1, sex; model 2, sex and height; model 3, sex, height, systolic blood pressure, total cholesterol, high-density lipoprotein cholesterol, current smoking (yes/no), use of antihypertensive medication (yes/no) and if one or two family members had a history of premature myocardial infarction (yes/no).

Table 1 Crude sample characteristics for the SAMINOR 1 population (n=13 787) given as means and SD or proportions and numbers

	Men		Women	
	Non-Sami (n=4971)	Sami (n=1408)	Non-Sami (n=5826)	Sami (n=1582)
Age (years)	52.8 (10.8)	52.9 (10.8)	52.8 (11.3)	52.1 (11.2)
Height (cm)	175.6 (6.9)	169.6 (6.4)	162.5 (6.4)	156.8 (6.0)
Systolic blood pressure (mm Hg)	134.0 (18.0)	133.3 (19.0)	129.2 (20.9)	127.2 (20.9)
Diastolic blood pressure (mm Hg)	78.3 (10.0)	77.9 (9.8)	72.7 (10.2)	72.1 (10.1)
Total cholesterol (mmol/L)	6.02 (1.1)	6.04 (1.1)	5.99 (1.2)	6.01 (1.1)
HDL cholesterol (mmol/L)	1.26 (0.33)	1.27 (0.35)	1.49 (0.38)	1.44 (0.37)
Smoking				
Current smoker	31.5 (1566)	34.7 (488)	32.3 (1882)	33.0 (522)
Previous smoker	37.7 (1876)	37.0 (522)	30.5 (1777)	28.1 (445)
Never smoker	30.8 (1529)	28.3 (398)	37.2 (2167)	38.9 (615)
Family history of premature MI*				
One family member	19.4 (960)	20.0 (281)	20.1 (1171)	21.4 (337)
Two family members	3.0 (147)	4.5 (63)	4.2 (242)	5.8 (91)
Users of antihypertensive medication†	13.7 (681)	13.5 (190)	16.6 (969)	16.1 (254)
Estimated 10-year risk of fatal or non-fatal AMI or stroke†‡	8.82 (7.9)	9.13 (8.1)	4.76 (6.0)	5.73 (4.4)
Triglycerides (mmol/L)†	1.87 (1.31)	1.87 (1.16)	1.50 (0.86)	1.55 (0.88)
Hypertensive	39.5 (1963)	38.6 (543)	35.7 (2078)	32.1 (508)
Users of lipid-lowering drugs†	7.1 (354)	8.6 (121)	8.7 (506)	8.7 (137)
Waist circumference (cm)†	94.3 (10.3)	92.3 (10.9)	85.1 (11.9)	85.3 (11.9)
Diabetes mellitus	3.4 (167)	4.1 (58)	3.5 (202)	3.4 (53)
Physically active in leisure time†	76.7 (3565)	75.7 (977)	78.4 (4 134)	72.1 (1037)
Alcohol consumption†				
Never/not last year	8.5 (416)	12.9 (177)	17.1 (966)	28.6 (436)
Less than weekly	58.2 (2843)	61.2 (838)	60.9 (3443)	58.3 (890)
Weekly or more often	33.3 (1626)	25.9 (354)	22.0 (1241)	13.1 (199)
Years of education†	11.5 (3.7)	11.0 (4.0)	11.7 (3.8)	11.6 (4.5)
Region 1: Alta, Loppa, Kvalsund, Lebesby, Lyngen, Storfjord, Kåfjord, Kvænangen§	62.7 (3117)	31.9 (449)	62.6 (3646)	28.2 (446)
Region 2: Kautokeino. Karasjok§	1.3 (66)	28.7 (404)	1.5 (89)	33.1 (524)
Region 3: Tana, Nesseby, Porsanger§	10.6 (527)	28.1 (396)	11.0 (639)	29.1 (460)
Region 4: Narvik, Evenes, Tysfjord, Skånland, Lavangen, Røros, Snåsa, Røyrvik, Namsskogan, Grane, Hattfjelldal§	25.4 (1261)	11.3 (159)	24.9 (1452)	9.6 (152)

*Parents, siblings or children with MI before the age of 60 years.
†Missing values: users of antihypertensive medication, n=142; estimated 10-year risk in men, n=13; triglycerides, n=12; waist circumference, n=47; education, n=770; users of lipid lowering drugs, n=300; physical activity, n=1185; alcohol consumption, n=358.
‡Estimated 10-year risk is measured with the NORRISK 2 risk score including sex, age, systolic blood pressure, total and HDL cholesterol, smoking (yes/no), use of antihypertensive medication (yes/no) and if one or two family members had a history of premature MI (yes/no).
§Included the listed municipalities.
HDL, high-density lipoprotein; MI, myocardial infarction.

endpoint, the HRs and 95% CIs in Sami versus non-Sami are visualised for model 1 to 3 with a forest plot (figure 1). We found no ethnic differences in the age-adjusted and sex-adjusted (model 1) or the multivariable-adjusted (model 2–3) risk of AMI, CHD or in the composite endpoint. Regarding IS and stroke, Sami had higher age-adjusted and sex-adjusted risks than non-Sami, with HR of 1.36 (95% CI: 1.10 to 1.68) and 1.31 (95% CI: 1.08 to 1.58), respectively. Adjustment for height attenuated the relationships between ethnicity and IS, and ethnicity and stroke to HR 1.26 (95% CI: 1.00 to 1.59) and 1.18 (95% CI: 0.96 to 1.44), respectively. Further adjustment for conventional risk factors (model 3) increased the risk of IS and stroke

somewhat (to HR 1.30 (95% CI: 1.03 to 1.64) and 1.19 (95% CI: 0.97 to 1.47), respectively) (figure 1).

A significant interaction between sex and height was present in the models for stroke and the composite endpoint. Sex-stratified analyses revealed that height was inversely associated with stroke (model 2, HR per 5 cm increase: 0.84, 95% CI: 0.76 to 0.94) and the composite endpoint (model 2, HR per 5 cm increase: 0.89, 95% CI: 0.82 to 0.96), only in women (online supplementary table 2).

The sensitivity analyses rendered results that were consistent with those reported above.

DISCUSSION

In this population-based study from Northern and Mid Norway with a median of 13 years of follow-up, we found that Sami had a higher risk of IS and stroke than non-Sami, and a considerable part was explained by differences in height. No ethnic differences were observed with regards to AMI, CHD and the composite endpoint consisting of fatal or non-fatal AMI or stroke.

Our findings of higher incidence of IS and stroke in Sami compared with non-Sami is supported by studies in Norway and Sweden that indicate a higher incidence[11 12 30] and higher mortality of stroke in Sami.[9] However, similar mortality of stroke was found between Sami and non-Sami in Sweden and Northern Finland.[30 31]

We observed a similar risk of CHD and AMI in Sami and non-Sami, which is consistent with the results regarding MI from a study based on data from 1974/1975 to 1989 that included the population in the Finnmark County.[11] A different study, covering the same population and time period, found lower total mortality, CVD mortality and CHD mortality in Sami men, whereas no ethnic difference was observed between Sami and non-Sami women.[10] Another study followed Sami identified through a population census in Northern Norway from 1970 to 1998 and found that Sami had higher total mortality, CVD mortality and CHD mortality than their benchmark population.[9] However, as we included both fatal and non-fatal events, direct comparisons of mortality rates are challenging. In Finland, lower and similar mortalities have been observed in Sami,[31 32] whereas in Sweden, no overall differences in incidence and mortality are observed when compared with reference non-Sami populations.[30] Being a member of a Sami reindeer herding household may, however, be protective.[9 30]

It seems likely that Sami people have a higher risk of IS and stroke than non-Sami, and similar risk of AMI and CHD, as this corresponds well with previous studies. We note that our age-adjusted and sex-adjusted effect estimates (model 1) are important from a public health perspective, as they reflect the actual distribution of risk in these populations. In our study, we were able to provide some explanation for the differences in risks, as the results for IS and stroke were attenuated, becoming only borderline significant after adjustment for height and conventional risk factors. However, the effect estimates for IS and stroke remained elevated after adjustments (models 2 and 3), which suggests that there might be other intermediate factors that we have not accounted for.

When adjusting for height, we most likely adjusted for factors that are related to health, nutrition and socioeconomic conditions, within and across generations,[33 34] and genetic factors,[15 35] which all influence the body height of an individual. Adjusting for baseline values of conventional risk factors (model 3) had only a small impact on the associations between ethnicity and IS, and ethnicity and stroke. This was as expected, given the similar baseline levels of conventional risk factors found in Sami and non-Sami at two time points.[17 18] The Emerging Risk Factors Collaboration included over 1 million people and found that after adjusting for height, further adjustments for long-term exposure to cardiovascular risk factors had little impact on the mortality-specific risk estimations.[16] They suggested that adult height is influenced by factors that also influence cardiovascular risk through the shared biological process between genes that determine adult height and atherosclerosis,[16] which has been considered by others.[36] For the risk of CHD, an increase in genetically determined height (6.5 cm) has been associated with the lower body mass index,[37] lower cholesterol[36 37] and better lung function,[37 38] whereas for stroke, the associations were less certain.[37] Some argue that height has a direct effect as short people have proportionally smaller coronary arteries that are occluded earlier than in tall persons by a similar burden of plaque.[39] Shorter people are found to have higher blood pressure[40] and increased heart rate due to a smaller arterial tree[41] than in taller people. The precise mechanism linking short stature with increased risk of CHD and stroke remains uncertain.

We found no ethnic differences in the risk of AMI and CHD. CVD is multifactorial in origin, and comparison groups may differ with regards to unmeasured risk or protective factors. Poor socioeconomic circumstances during childhood are found to have a stronger impact on stroke mortality than CHD mortality,[42] which suggests some differences in the aetiology of stroke and CHD.

Education and healthcare are publicly financed in Norway, and this has been suggested as the underlying reason for the rather similar health observed previously in Sami and their majority population, as opposed to the situation in other indigenous populations.[43] Adjusting for baseline education and lifestyle factors (model 4 in sensitivity analyses) had a small impact in our study, and expenditures to secondary healthcare services are found to be similar in geographical areas that represent Sami and non-Sami populations.[44] Hence, differences in healthcare utilisation and education are most likely not an issue in this population.

All inhabitants, within the selected region and birth cohorts, were invited to the SAMINOR 1, regardless of ethnicity, which should help control some environmental confounding factors that can distort comparison between

ethnic groups. However, ethnicity itself is rarely the causal factor for diseases, but a risk marker,[20] representing certain 'exposures' that is determined by group membership. The height difference explained more of the risk of IS and stroke observed in Sami than conventional risk factors, which is interesting but challenging to interpret, as there are complex relationships between height, genetics, ethnicity and environmental factors across generations.[34 45] Moreover, we cannot rule out that the excess risk in Sami might be due to residual confounding.

Psychosocial stress has been found to be an important risk factor for stroke,[46] and the Sami people have been exposed to marginalisation[47] and ethnic discrimination.[48] If marginalisation and discrimination cause chronic psychosocial stress, they may lead to a higher risk of IS and stroke in Sami, as was previously suggested by Eliassen *et al*, who reported that marginalised Sami were more likely to report lifetime CVD.[47] Thus, further studies that clarify how height and possibly other intermediate factors lead to a higher risk of IS and stroke in Sami are warranted.

Strengths and limitations

The use of 11-digit national identity numbers to obtain complete follow-up to high-quality registries,[25 49] and the possibility to remove prevalent cases at baseline represents the strengths of this study. A limitation is that we have only adjusted for baseline values and cannot account for changes in risk factors or in the use of medication.

Potential misclassification of ethnicity is likely to be non-differential and would have led us to underestimate the associations. With regards to the reliability of ethnicity, Sami ethnicity was found to be stable when comparing information in the 1970 census, that is, language use in three generations and self-reported ethnicity, with corresponding information in SAMINOR 1.[50] However, we do not know if participation differed by ethnicity as there are no national registries that include this information. Moreover, participants in health surveys tend to be healthier than the underlying population,[51] which can lead to lower incidence rates.

In conclusion, over 13 years of follow-up, Sami in Northern and Mid Norway showed a higher risk of IS and stroke than non-Sami, and height was identified as an important intermediate factor, as it explained more of the excess risk observed in Sami than conventional risk factors did. Sami and non-Sami participants had similar risk of AMI, CHD and the composite endpoint (AMI or stroke).

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Contributors TB and BME: conceived the study. MM: linked the different data sources and prepared the research dataset for STATA. SRAS: performed the analyses, wrote the manuscript and made the tables. TB, BKJ and VLM: assisted with statistical analyses and the interpretations of the statistics. VLM: made the figure. All authors critically revised the manuscript, tables and figures, and contributed to drawing the final conclusions.

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Competing interests None declared.

Patient consent for publication Not required.

Ethics approval The present study (2015/2204–11) is approved by the Regional Committee for Medical and Health Research Ethics for region North (REC North) and by the SAMINOR Project Board. We included those who gave written informed consent to have their data linked to registries, and the data were deidentified before they were available for analyses. The SAMINOR is part of the SAMINOR Study that is approved by The Norwegian Data Inspectorate and the REC North.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement In this study, we have used de-identified participant data which is not available for the public, as it is restricted by licence. 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 for region North.

Correction notice This paper has been updated since first published to update the Key Questions box.

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Supplementary table 1. Number of cases, crude incidence rates (with 95% confidence intervals) and adjusted hazard ratios (with 95% confidence intervals) for cardiovascular diseases in Sami (n= 2 990) and non-Sami (n=10 797) men and women participating in the SAMINOR 1 Survey (2003/2004).

Ethnicity	Cases (n)	Person-years	Incidence rates per 1 000 person-years (95% CI)	Hazard ratio (95% CI)		
				Model 1	Model 2	Model 3
Acute myocardial infarction						
Men						
Non-Sami	394	60 713	6.5 (5.88, 7.16)	1	1	1
Sami	121	17 413	6.9 (5.82, 8.30)	1.06 (0.86, 1.30)	1.02 (0.82, 1.27)	1.03 (0.83, 1.28)
Women						
Non-Sami	212	73 962	2.9 (2.51, 3.28)	1	1	1
Sami	47	20 606	2.3 (1.71, 3.04)	0.85 (0.62, 1.17)	0.78 (0.56, 1.10)	0.82 (0.58, 1.15)
Coronary heart diseases						
Men						
Non-Sami	895	57 608	15.5 (14.55, 16.59)	1	1	1
Sami	282	16 453	17.1 (15.25, 19.26)	1.10 (0.96, 1.26)	1.04 (0.90, 1.20)	1.05 (0.91, 1.21)
Women						
Non-Sami	635	71 434	8.9 (8.22, 9.61)	1	1	1
Sami	159	19 927	8.0 (6.83, 9.32)	0.93 (0.79, 1.11)	0.86 (0.71, 1.03)	0.87 (0.72, 1.04)
Ischemic stroke						
Men						
Non-Sami	181	61 799	2.9 (2.53, 3.39)	1	1	1
Sami	70	17 593	4.0 (3.15, 5.03)	1.36 (1.03, 1.79)*	1.32 (0.98, 1.78)	1.34 (0.99, 1.82)
Women						
Non-Sami	133	74 178	1.8 (1.51, 2.13)	1	1	1
Sami	48	20 594	2.3 (1.76, 3.09)	1.38 (0.99, 1.92)	1.18 (0.82, 1.69)	1.20 (0.83, 1.72)
Stroke §						
Men						
Non-Sami	235	61 602	3.8 (3.36, 4.34)	1	1	1
Sami	87	17 511	5.0 (4.03, 6.13)	1.29 (1.01, 1.65)	1.26 (0.97, 1.65)	1.28 (0.98, 1.67)
Women						
Non-Sami	177	74 003	2.4 (2.06, 2.77)	1	1	1
Sami	62	20 554	3.0 (2.35, 3.87)	1.34 (1.00, 1.79)	1.08 (0.79, 1.48)	1.10 (0.80, 1.51)
Composite endpoint §						
Men						
Non-Sami	596	59 759	10.0 (9.20, 10.81)	1	1	1
Sami	194	17 057	11.4 (9.88, 13.09)	1.12 (0.96, 1.32)	1.10 (0.92, 1.31)	1.13 (0.95, 1.34)
Women						
Non-Sami	370	73 255	5.05 (4.56, 5.59)	1	1	1
Sami	99	20 384	4.86 (3.99, 5.91)	1.03 (0.82, 1.28)	0.88 (0.70, 1.13)	0.92 (0.72, 1.17)

* p-value <0.05, ** <0.01, ***<0.001

§ Interaction between sex and height.

Model 1, adjusted for age.

Model 2, adjusted for age and height.

Model 3, model 2 additionally adjusted for: systolic blood pressure, total cholesterol, high-density lipoprotein cholesterol, current smoking (yes/no), use of antihypertensive medication (yes/no) and if one or two family members had a history of premature myocardial infarction (yes/no).

Acute myocardial infarction: ICD10-codes I21–I22 as discharge code from hospitalisation, or I20–I25 as the underlying cause of death.

Coronary heart diseases: ICD10-codes I20–I25 as discharge code from hospitalisation, or as the underlying cause of death.

Ischemic stroke: ICD-10 code I63 as discharge code from hospitalisation, or as the underlying cause of death.

Stroke: ICD-10 codes I60–I61 or I63–I64 (except 163.6) as a discharge code from hospitalisation, or as the underlying cause of death.

Composition endpoint includes ICD-codes I21–I22, I60–I61 or I63–I64 (except 163.6) as discharge code from hospitalisation, or I21–I25, I60–I61 or I63–I64 (except 163.6) as the underlying cause of death.

Abbreviations: HR, hazard ratio; CI Confidence interval; ICD-10 International Classification of Diseases, 10th Revision.

Supplementary table 2. Overall and sex-specific hazard ratios (with 95% confidence intervals) for acute myocardial infarction, coronary heart diseases, ischemic stroke, cerebral stroke and the composite endpoint associated with a 5-cm increase in height. SAMINOR 1 Survey (n=13 787).

		Model 2	**Model 3**
		Hazard ratio (95% CI)	Hazard ratio (95% CI)
Acute myocardial infarction			
Overall		0.96 (0.91, 1.02)	1.00 (0.94, 1.05)
	Men	0.97 (0.91, 1.04)	1.00 (0.94, 1.08)
	Women	0.94 (0.84, 1.04)	0.97 (0.87, 1.08)
Coronary heart disease			
Overall		0.95 (0.91, 0.98)**	0.97 (0.94, 1.01)
	Men	0.96 (0.91, 1.00)*	0.98 (0.94, 1.02)
	Women	0.93 (0.88, 0.99)*	0.97 (0.91, 1.02)
Ischemic stroke			
Overall		0.94 (0.87, 1.02)	0.96 (0.89, 1.03)
	Men	0.98 (0.89, 1.08)	1.00 (0.90, 1.10)
	Women	0.88 (0.78, 1.00)*	0.90 (0.80, 1.02)
Stroke			
Overall		§	§
	Men	0.98 (0.90, 1.07)	1.00 (0.92, 1.08)
	Female	0.84 (0.76, 0.94)**	0.86 (0.77, 0.96)**
Composition endpoint			
Overall		§	§
	Men	0.98 (0.93, 1.03)	1.01 (0.96, 1.07)
	Women	0.89 (0.82, 0.96)**	0.92 (0.85, 0.99)*

* p-value <0.05, ** <0.01, ***<0.001

§ Interaction between height and sex.

Model 2, overall models adjusted for age, sex and ethnicity. Sex-specific models adjusted for age and ethnicity.

Model 3, as model 2 and additionally adjusted for: systolic blood pressure, total cholesterol, high-density lipoprotein cholesterol, current smoking (yes/no), use of antihypertensive medication (yes/no) and if one or two family members had a history of premature myocardial infarction (yes/no).

Acute myocardial infarction: ICD10-codes I21–I22 as discharge code from hospitalisation, or I20–I25 as the underlying cause of death.

Coronary heart diseases: ICD10-codes I20–I25 as discharge code from hospitalisation, or as the underlying cause of death.

Ischemic stroke: ICD-10 code I63 as discharge code from hospitalisation, or as the underlying cause of death.

Stroke: ICD-10 codes I60–I61 or I63–I64 (except 163.6) as a discharge code from hospitalisation, or as the underlying cause of death.

Composition endpoint includes ICD-codes I21–I22, I60–I61 or I63–I64 (except 163.6) as discharge code from hospitalisation, or I21–I25, I60–I61 or I63–I64 (except 163.6) as the underlying cause of death.

Abbreviations: CI Confidence interval; ICD-10 International Classification of Diseases, 10th Revision

Appendix A

SAMINOR 1 Survey

- Information brochure
  - Design 1
  - Design 2
- Invitation letter, design 1
- Informed written consent form
- Remainder card
- Screening questionnaire (english translation), design 2

All listed items and their Norwegian versions are available at www.saminor.no.

med opplysninger om deg i andre registre for forskningsformål slik som *Kreftregisteret, Dødsårsaksregisteret* og folketellingene. I alle disse tilfellene vil navn og personnummer bli fjernet. Forsikringsselskaper får ikke tilgang til dataene.

4) At blodprøven din kan lagres og brukes til medisinsk forskning og genetiske analyser for å finne årsak til sykdom. All bruk av denne prøven vil bare skje i samsvar med godkjenning fra *Datatilsynet* og etter at *Regional komité for medisinsk forskningsetikk i Nord-Norge* har vurdert og tilrådd prosjektet.

Selv om du sier ja til dette nå, kan du senere ombestemme deg og be om å bli slettet fra undersøkelsen uten at du må oppgi noen grunn for det. Dette gjøres ved skriftlig beskjed til **Institutt for samfunnsmedisin, UiTø, 9037 Tromsø**. Blodprøven din vil da bli tilintetgjort.

Vi ønsker å følge alle som møter til helseundersøkelsen i lang tid framover med hensyn til hjerteinfarkt, hjerneslag og andre aktuelle sykdommer. Derfor ønsker vi å lagre opplysningene du har gitt, frem til fylte 100 år, for å sammenholde disse med opplysninger fra sentrale registre slik som *Kreftregisteret* og *Dødsårsaksregisteret.*

Velkommen til helseundersøkelsen

Selv om du nettopp har vært hos lege eller selv om du føler deg frisk, kan du likevel delta i undersøkelsen. Da hjelper du oss til bedre kunnskap og riktigere oversikt over helsen i kommunen og fylket ditt.

Dødsårsaksregistret ja olmmošlohkamat. Visot dáid oktavuođain sihkko namma ja personnummar. Dáhkádusfitnodagat eai beasa dáid dieđuid oaidnit.

4) Ahte du varraiskkus sáhttá ráddjot ja adnot medisiinnalaš dutkamii ja genetalaš analysaide gávnnahit dávddaid árttaid. Dán iskosa juohke geavaheapmi geavvá dušše *Datatilsynet* dohkkeheami mielde ja maŋŋil go *Regional komite for medisinsk forskningsetikk i Nord-Norge* lea árvvoštallan ja rávven prošeavtta.

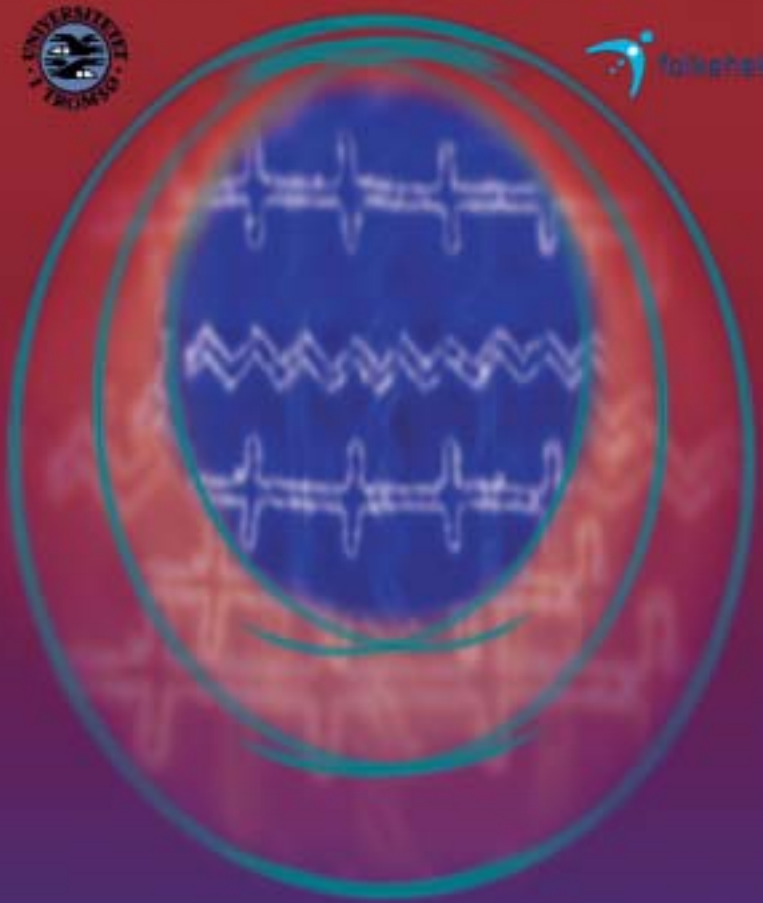
Vaikke dása dál mieđat, de sáhtát maŋŋil molsut oaivila ja bivdit sihkkot iskkadeamis dieđitkeahttá makkárge ákka dasa. Dán dagat čálalaččat Institutt for samfunnsmedisinii; **Institutt for samfunnsmedisin, UiTø, 9037 Tromsø**. Du varraiskkus dalle bálkestuvvo.

Mii dáhtošeimmet guhkit áiggi čuovvut juohkehačča gii boahtá dearvvasvuođaiskkadeapmái váibmodohppehaga, vuoiŋŋašgáldnanvigi ja eará vejolaš dávddaid hárrái. Danne dáhtošeimmet rádjat du addán dieđuid, gitta devdon 100 jahkái, vai daid beassá sulastahttit guovddáš registariid dieđuiguin, nugo *Kreftregistret* ja *Dødsårsaksregistret.*

Bures boahtin dearvvasvuođaiskkadeapmái

Vaikke leatge aiddo leamaš doaktára luhtte dahje dovddat iežat dearvvasin, de sáhtát liikká searvat iskkadeapmái. Dalle veahkehat min oažžut eanet máhtu ja riektasat dieđuid du gieldda ja fylkka dearvvasvuođas.

Gráfalaš barggus vástida/Grafisk utforming: Britt Hansen Biti – BHB Gráfalaš bálvalusat/Grafiske tjenester, Kárášjohka/Karasjok.
Deaddilan/Trykk: Bjørkmanns Trykkeri, Alaheadju/Alta.



Nå skal vi sette fokus på helsen i kommunen din.
Hvordan står det egentlig til? Hvordan fungerer helsetjenesten?
Er det store helseforskjeller i de ulike delene av fylket eller mellom de ulike etniske gruppene? Er kvinner friskere enn menn?
Hvorfor øker sukkersyke her i landet?

Dál áigut giddet fuomášumi dearvvasvuhtii din gielddas. Mo dat duođas lea?
Mo doaibmá dearvvasvuođabálvalus? Leatgo stuorra dearvvasvuođaerohusat fylkka iešguđet osiin dahje iešguđet čearddalaš joavkkuid gaskkas?
Leatgo nissonat dearvasat go albmát?
Manne lassána sohkardávda dán riikkas?

For mer informasjon, ring 78 46 89 04, Senter for samisk helseforskning, Karasjok.
E-post: helseus@fagmed.uit.no

Jus dárbbašat eambbo dieđuid, čuojahastte 78 46 89 04, Sámi dearvvašvuođadutkama guovddážii, Kárášjohka. E-poasta: helseus@fagmed.uit.no

Helseundersøkelsen har tre formål:

- Du som deltar i helseundersøkelsen får sjekket om du har bestemte sykdommer, eller om det er fare for at du kan få dem.
- Å få ny kunnskap om helse, sykdom og levekår i områder med samisk og norsk bosetting.
- Å lage en oversikt over folks helse – en «helseprofil» for fylket. Dette er viktig for å gi fylket og de enkelte kommunene et bedre grunnlag for å planlegge helsetjenesten i framtida.

Hvem kan delta?

Alle født 1925–1967 og i 1973 fra områder med samisk og norsk bosetting. Det er 9 kommuner i Finnmark, 6 i Troms, 4 i Nordland og 2 i Nord-Trøndelag med i undersøkelsen.

Hvordan får du time til helseundersøkelsen?

Dersom du ønsker å være med i helseundersøkelsen, krysser du av for det i vedlagte spørreskjema, besvarer det og sender det inn. Deretter får du time til helseundersøkelsen som vil foregå enten i buss eller i et fast lokale i kommunen. Hvis den oppsatte timen ikke passer, kan du møte når du vil innenfor åpningstiden vår som du finner i invitasjonsbrevet. Undersøkelsen er gratis. Du får tilsendt et spørreskjema sammen med innkallingen. Vi ber om at du fyller ut skjemaet hjemme og tar det med når du møter fram til helseundersøkelsen.

Dearvvasvuođaiskkadeami dieđuin leat golbma ulbmila:

- Dus gii searvvat iskkadeapmái iskat leatgo dus dihto dávddat, dahje leago dus várra daid oažžut.
- Oažžut ođđa máhtu dearvvasvuođa, dávddaid ja eallindili birra sámi ja dáža ássanguovlluin.
- Ráhkadit várdosa olbmuid dearvvasvuođas – fylkka «dearvvasvuođaprofiilla». Dát lea dehálaš vai fylkkas ja juohke gielddas lea buoret vuođđu plánet boahttevaš dearvvasvuođabálvalusa.

Gii sáhttá searvat?

Juohkehaš riegádan 1925–1967 ja 1973 guovlluin gos ásset sápmelaččat ja dážat. 9 gieldda Finnmárkkus, 6 Tromssas, 4 Nordlánddas ja 2 Davvi-Trøndelagas leat iskkadeamis mielde.

Mo oaččut diimmu dearvvasvuođaiskkadeapmái?

Jus dáhtut leat mielde dearvvasvuođaiskkadeamis, de russet dan čuovvu gažadanskovis, vástidat dan ja sáddet dan midjiide. Dasto oaččut diimmu iskadeapmái mii lea juogo busses dahje dihto lanjas gielddas. Jus biddjon áigi ii heive, de sáhtát boahtit vaikke goas min rahpanáiggis maid oainnát rávkanreivves. Iskkadeapmi lea nuvttá. Oaččut gažadanskovi oktan rávkamiin. Bivdit du deavdit skovi ruovttus ja váldit dan mielde go boađát iskkadeapmái.

Hvordan foregår helseundersøkelsen?

Det gjøres målinger av blodtrykk, høyde, vekt og livvidde, og det taes en blodprøve. Blodprøven kan senere bli analysert på fettstoffer i blodet, blodsukker, markører for betennelsesreaksjoner, kosthold, hormoner, lever- og nyrefunksjon samt beinmarkører. Genetiske analyser av blodet kan også bli aktuelt.

Omtrent fire uker etter helseundersøkelsen får du et brev i posten med opplysninger om ditt kolesterol, blodtrykk og blodsukker, og hvordan du ligger an i forhold til anbefalte verdier. De som har særlig høy risiko for å få hjerte- og kar sykdommer og sukkersyke, vil bli bedt om å ta kontakt med sin egen lege for videre oppfølging.

Alle som møter fram til helseundersøkelsen, får et tilleggsskjema, med spørsmål om blant annet kosthold og levekår.

Vi trenger din tillatelse

Når du møter fram til helseundersøkelsen, ber vi deg om å undertegne et samtykke der du sier deg enig i et eller flere av de fire punktene nedenfor. (Du vil få kopi av samtykke erklæringen).

1) At du kan bli kontaktet med anbefaling om oppfølging, behandling eller for å forebygge sykdom.

2) At opplysningene dine kan brukes til medisinsk forskning etter vurdering og tilråding fra *Regional komité for medisinsk forskningsetikk i Nord-Norge* og *Datatilsynet.*

3) At resultatene dine (etter godkjenning fra *Datatilsynet*) kan settes sammen

Mo iskkojuvvot?

Varradeaddu, allodat, lossodat ja seakkáš mihtiduvvojit, ja váldo varraiskkus. Varraiskosis sáhttá maŋŋil iskat vara buoideávdnasiid, varrasohkkara, infekšunreakšuvnnaid mearkkaid, biepmu, hormonaid, vuoivvas- ja monimušdoaimma ja dáktemearkkaid. Vara genetalaš analysat maid soitet šaddat áigeguovdilat.

Sullii njeallje vahku maŋŋil dearvvasvuođaiskkadeami oaččut poasttas reivve iežat kolestrola, varradeattu ja varrasohkkara birra, ja mo dat leat rávvejuvvon meriid ektui. Bivdit sin geain lea hui alla váibmo- ja suotnadávddavárra ja sohkardávda, váldit oktavuođa iežaset doaktáriin joatkka čuovvoleapmái.

Juohkehaš gii boahtá iskkadeapmái, ažžu lassiskovi, gažaldagaiguin ee. biepmu ja eallindili birra.

Mii dárbbašat du lobi

Go boađát iskkadeapmái, de bivdit du čállit vuollái miehtama, mas logat iežat leat ovttamielas ovtta dahje moatti dán njeallje čuoggás vulobealde (Miehtamis oaččut máŋgosa).

1) Ahte duinna sáhttá váldit oktavuođa go áigu rávvet čuovvoleami, dálkkodit dahje eastadit dávddaid.

2) Ahte visot du dieđut sáhttet adnot medisiinnalaš dutkamii *Regional komite for medisinsk forskningsetikk i Nord-Norge* ja *Datatilsynet* árvvoštallama ja rávvaga mielde.

3) Ahte du bohtosiid (*Datatilsynet* dohkkeheami mielde) sáhttá čohkket dieđuiguin du birra eará registariin dutkandoaimmaide nugo *Kreftregistret,*

Selv om du sier ja til dette nå, kan du senere ombestemme deg og be om å bli slettet fra undersøkelsen uten at du må oppgi noen grunn for det. Dette gjøres ved skriftlig beskjed til **Institutt for samfunnsmedisin, UiTø, 9037 Tromsø**. Blodprøven din vil da bli tilintetgjort.

Vi ønsker å følge alle som møter til helseundersøkelsen i lang tid framover med hensyn til hjerteinfarkt, hjerneslag og andre aktuelle sykdommer. Derfor ønsker vi å lagre opplysningene du har gitt, frem til fylte 100 år, for å sammenholde disse med opplysninger fra sentrale registre slik som *Kreft-* og *Dødsårsaksregisteret.*

Resultatene vil bli publisert i massemedia, og det utformes en rapport fra helse- og levekårsundersøkelsen når den er avsluttet.

Datatilsynet har gitt konsesjon for lagring av opplysninger fra undersøkelsen og forskningsprosjektet er tilrådd av *Regional komite for medisinsk forskningsetikk i Nord- Norge.*

Velkommen til helseundersøkelsen

Selv om du nettopp har vært hos lege eller selv om du føler deg frisk, kan du likevel delta i undersøkelsen. Da hjelper du oss til bedre kunnskap og riktigere oversikt over helsen i kommunen og fylket ditt.

Vaikke dása dál mieđat, de sáhtát maŋŋil molsut oaivila ja bivdit sihkkot iskkadeamis dieđitkeahttá makkárge ákka dasa. Dán dagat čálalaččat Institutt for samfunnsmedisinii; **Institutt for samfunnsmedisin, UiTø, 9037 Tromsø**. Du varraiskkus dalle bálkestuvvo.

Mii dáhtošeimmet guhkit áiggi čuovvut juohkehačča gii boahtá dearvvasvuođaiskkadeapmái váibmodohppehaga, vuoiŋŋašgáldnanvigi ja eará vejolaš dávddaid hárrái. Danne dáhtošeimmet rádjat du addán dieđuid, gitta devdon 100 jahkái, vai daid beassá sulastahttit guovddáš registariid dieđuiguin, nugo *Kreft-* ja *Dødsårsaksregistret.*

Bohtosiid almmuhat mediain, ja čállo raporta dearvvasvuođa- ja eallindilleiskkadeamis go dat lea loahpahuvvon.

Datatilsynet lea addán sierralobi rádjat iskkadeami dieđuid ja dutkanprošeavtta lea rávven *Regional komite for medisinsk forskningsetikk i Nord-Norge.*

Bures boahtin dearvvasvuođaiskkadeapmái

Vaikke leatge aiddo leamaš doaktára luhtte dahje dovddat iežat dearvvasin, de sáhtát liikká searvat iskkadeapmái. Dalle veahkehat min oažžut eanet máhtu ja riektasat dieđuid du gieldda ja fylkka dearvvasvuođas.

Dearvvuođaiguin / Med hilsen

Anne Kirsten Anti
Sámi dearvvašvuođadutkama guovddáš,
Senter for samisk helseforskning
Kárášjohka/Karasjok

Eiliv Lund
Institutt for samfunnsmedisin
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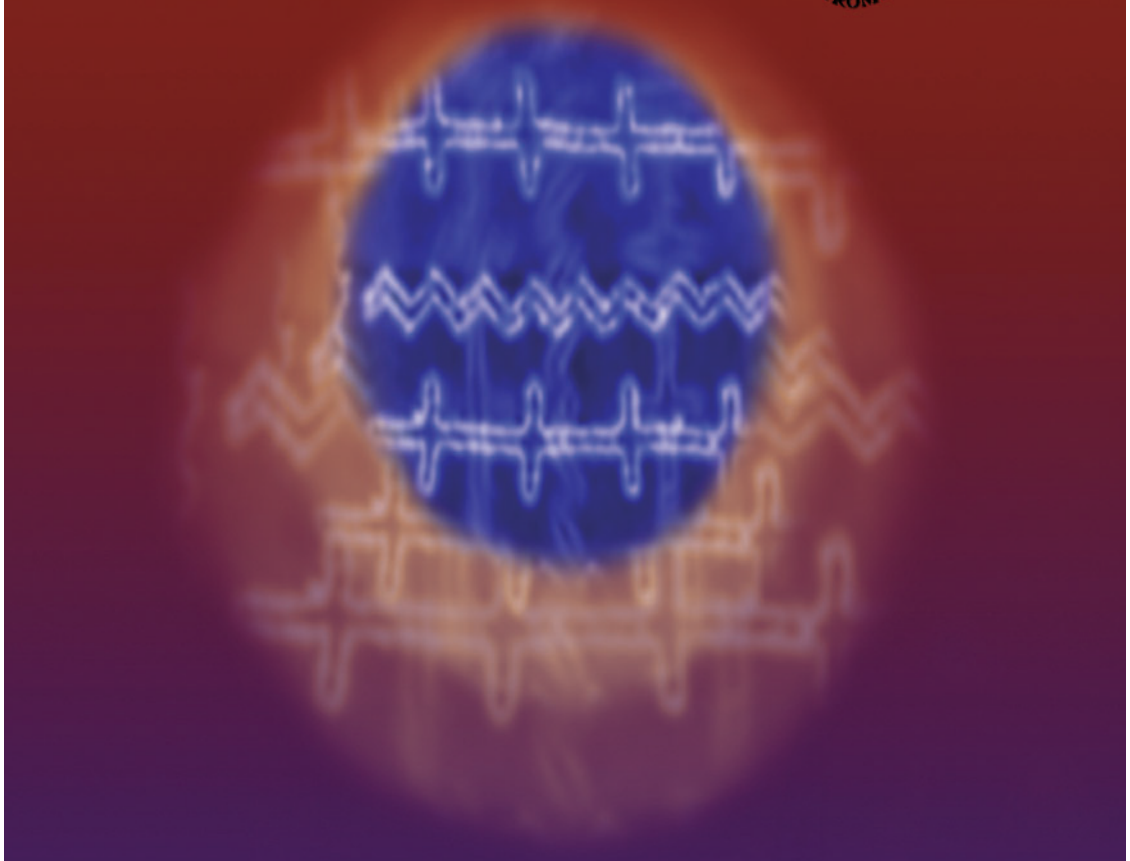
Gráfalaš barggus vástida/Grafisk utforming: Britt Hansen Biti – BHB Gráfalaš bálvalusat/Grafiske tjenester, Kárášjohka/Karasjok. Sámás: Davvi Girji OS.
Deaddilan/Trykk: Bjørkmanns Trykkeri, Alaheadju/Alta.

folkehelseinstituttet

UNIVERSITETET I TROMSØ



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- Å lage en oversikt over folks helse – en «helseprofil» for fylket. Dette er viktig for å gi fylket og de enkelte kommunene et bedre grunnlag for å planlegge helsetjenesten i framtida.

Hvem kan delta?

Alle født 1925–1967 og i 1973 fra områder med samisk og norsk bosetting. Det er 9 kommuner i Finnmark, 6 i Troms, 4 i Nordland og 2 i Nord-Trøndelag med i undersøkelsen.

Hvordan får du time til helseundersøkelsen?

Du får tilsendt et spørreskjema sammen med innkallingen. Vi ber om at du fyller ut skjemaet hjemme og tar det med når du møter fram til helseundersøkelsen. Helseundersøkelsen vil foregå enten i buss eller i et fast lokale i kommunen. Hvis den oppsatte timen ikke passer, kan du møte når du vil innenfor åpningstiden vår. Undersøkelsen er gratis.

Hvordan foregår helseundersøkelsen?

Det gjøres målinger av blodtrykk, høyde, vekt og livvidde, og det taes en blodprøve. Blodprøven kan senere bli analysert på fettstoffer i blodet, blodsukker, markører for betennelsesreaksjoner, kosthold, hormoner, lever- og nyrefunksjon samt beinmarkører. Genetiske analyser av blodet kan også bli aktuelt.

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Alle som møter fram til helseundersøkelsen, får et tilleggsskjema, med spørsmål om blant annet kosthold og levekår.

De som fullfører hele helse- og levekårsundersøkelsen vil være med i trekningen av 3 reisegavekort hver verdt kr. 10000,–. Vi regner med en deltakelse på ca. 15000 personer.

Dearvvasvuođaiskkadeami dieđuin leat golbma ulbmila:

- Dus gii searvvat iskkadeapmái iskat leatgo dus dihto dávddat, dahje leago dus várra daid oažžut.
- Oažžut ođđa máhtu dearvvasvuođa, dávddaid ja eallindili birra sámi ja dáža ássanguovlluin.
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Gii sáhttá searvat?

Juohkehaš riegádan 1925–1967 ja 1973 guovlluin gos ásset sápmelaččat ja dážat. 9 gieldda Finnmárkkus, 6 Tromssas, 4 Nordlánddas ja 2 Davvi-Trøndelagas leat iskkadeamis mielde.

Mo oaččut diimmu dearvvasvuođaiskkadeapmái?

Oaččut gažadanskovi oktan rávkamiin. Bivdit du deavdit skovi ruovttus ja váldit dan mielde go boađát iskkadeapmái. Iskadeapmi lea juogo busses dahje dihto lanjas gielddas. Jus biddjon áigi ii heive, de sáhtát boahtit vaikke goas min rahpanáiggis. Iskkadeapmi lea nuvttá.

Mo iskkojuvvot?

Varradeaddu, allodat, lossodat ja seakkáš mihtiduvvojit, ja váldo varraiskkus. Varraiskosis sáhttá maŋŋil iskat vara buoideávdnasiid, varrasohkkara, infekšunreakšuvnnaid mearkkaid, biepmu, hormonaid, vuoivvas- ja monimušdoaimma ja dáktemearkkaid. Vara genetalaš analysat maid soitet šaddat áigeguovdilat.

Sullii njeallje vahku maŋŋil dearvvasvuođaiskkadeami oaččut poasttas reivve iežat kolestrola, varradeattu ja varrasohkkara birra, ja mo dat leat rávvejuvvon meriid ektui.

Bivdit sin geain lea hui alla váibmo- ja suotnadávddavárra ja sohkardávda, váldit oktavuođa iežaset doaktáriin joatkka čuovvoleapmái.

Juohkehaš gii boahtá iskkadeapmái, oažžu lassiskovi, gažaldagaiguin ee. biepmu ja eallindili birra.

Sii geat čađahit olles dearvvasvuođa- ja eallindilleiskkadeami leat mielde vuorbádeamen 3 mátkeskeaŋkakoartta man árvu lea 10000,– ru. guđesge. Doaivut ahte su. 15000 olbmo servet.

Vi trenger din tillatelse

Når du møter fram til helseundersøkelsen, ber vi deg om å undertegne et samtykke der du sier deg enig i et eller flere av de fire punktene nedenfor. (Du vil få kopi av samtykke erklæringen).

1) At du kan bli kontaktet med anbefaling om oppfølging, behandling eller for å forebygge sykdom.

2) At opplysningene dine kan brukes til medisinsk forskning etter vurdering og tilråding fra *Regional komité for medisinsk forskningsetikk i Nord-Norge* og *Datatilsynet*.

3) At resultatene dine (etter godkjenning fra *Datatilsynet*) kan settes sammen med opplysninger om deg i andre registre for forskningsformål slik som *Kreftregisteret, Dødsårsaksregisteret* og folketellingene. I alle disse tilfellene vil navn og personnummer bli fjernet. Forsikringsselskaper får ikke tilgang til dataene.

4) At blodprøven din kan lagres og brukes til medisinsk forskning og genetiske analyser for å finne årsak til sykdom. All bruk av denne prøven vil bare skje i samsvar med godkjenning fra *Datatilsynet* og etter at *Regional komité for medisinsk forskningsetikk i Nord-Norge* har vurdert og tilrådd prosjektet.

Mii dárbbašat du lobi

Go boađát iskkadeapmái, de bivdit du čállit vuollái miehtama, mas logat iežat leat ovttamielas ovtta dahje moatti dán njeallje čuoggás vulobealde (Miehtamis oaččut máŋgosa).

1) Ahte duinna sáhttá váldit oktavuođa go áigu rávvet čuovvoleami, dálkkodit dahje eastadit dávddaid.

2) Ahte visot du dieđut sáhttet adnot medisiinnalaš dutkamii *Regional komite for medisinsk forskningsetikk i Nord-Norge* ja *Datatilsynet* árvvoštallama ja rávvaga mielde.

3) Ahte du bohtosiid (*Datatilsynet* dohkkeheami mielde) sáhttá čohkket dieđuiguin du birra eará registariin dutkandoaimmaide nugo *Kreftregistret, Dødsårsaksregistret* ja olmmošlohkamat. Visot dáid oktavuođain sihkko namma ja personnummar. Dáhkádusfitnodagat eai beasa dáid dieđuid oaidnit.

4) Ahte du varraiskkus sáhttá ráddjot ja adnot medisiinnalaš dutkamii ja genetalaš analysaide gávnnahit dávddaid árttaid. Dán iskosa juohke geavaheapmi geavvá dušše *Datatilsynet* dohkkeheami mielde ja maŋŋil go *Regional komite for medisinsk forskningsetikk i Nord-Norge* lea árvvoštallan ja rávven prošeavtta.

Helse- og levekårsundersøkelse

– et forskningsprosjekt

Helsedepartementet har bedt oss undersøke helse- og levekårsforhold hos alle født i 1925–1967 og i 1973 i utvalgte kommuner med samisk og norsk bosetting i Nord-Norge og Nord-Trøndelag. Formålet er å innhente opplysninger om hjerte- og karsykdommer, kreft, allergier, smerter og andre lidelser samt ulykker for å kunne forebygge dem. Videre er målet å få et bilde av folks oppfatning av helsetjenestetilbudet, deres levesett slik som kosthold og røyking, levekår og tilhørighet. De som ønsker å delta, blir med i et forskningsprosjekt som består av spørreskjemaer og helseundersøkelse. Alle opplysninger fra undersøkelsen vil bli behandlet konfidensielt.

Helse- og levekårsundersøkelsen er nærmere beskrevet i brosjyren, som ligger vedlagt. Dersom du er i tvil om noe, kan du kontakte oss på tlf. 78 46 89 04 eller på e-post: helseus@fagmed.uit.no

Du kan delta på følgende måter: (kryss av øverst på spørreskjema under «samtykke til deltakelse»)

A Dersom du ønsker å delta i helseundersøkelsen og forskningsprosjektet, krysser du av punkt **A**, fyller ut spørreskjemaet og returnerer det til oss i vedlagte konvolutt. Du vil senere få et brev med tid og sted for fremmøte sammen med et nytt spørreskjema.

B Dersom du bare ønsker å delta i en innledende del av forskningsprosjektet uten helseundersøkelse, krysser du av punkt **B** , fyller ut spørreskjemaet og returnerer det til oss i vedlagte konvolutt.

C Du kan unngå purring fra oss ved å krysse av punkt **C** og returnere spørreskjemaet til oss. Purring vil skje skriftlig.

Datatilsynet har gitt konsesjon for lagring av opplysninger fra undersøkelsen og forskningsprosjektet er tilrådd av *Regional komite for medisinsk forskningsetikk i Nord- Norge.*

For forskningen sin del vil det være av stor interesse at vi får inn så mange opplysninger som mulig. Du deltar frivillig og kan, etter å ha sagt ja til deltakelse, senere trekke deg uten å begrunne hvorfor og uten at det vil ha noen konsekvenser for deg. Det samme gjelder dersom man i utgangspunktet ikke ønsker å delta. Opplysninger du har gitt kan du be om å få slettet.

Resultatene vil bli publisert i massemedia, og det utformes en rapport fra helse- og levekårsundersøkelsen når den er avsluttet.

De som fullfører hele helse- og levekårsundersøkelsen vil være med i trekningen av 3 reisegavekort til en verdi av á kr. 10 000,–. Vi regner med en deltakelse på ca. 15000 personer.

Med hilsen

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Nasjonalt folkehelseinstitutt
Oslo

Dearvvasvuođa ja eallindilleiskkadeapmi

– dutkanprošeakta

Dearvvasvuođadepartementa lea min bivdán iskat dearvvasvuođa- ja eallindili juohkehaččas riegádan 1925–1967 ja 1973 dihto gielddain sámi ja dáža ássamiin Davvi-Norggas ja Davvi-Trøndelágas. Ulbmilin lea viežžat dieđuid váibmo- ja suotnadávddaid, borasdávdda, allergiaid, bákčasiid ja eará gillámušaid ja lihkohisvuođaid birra vai daid sáhtášii eastadit. Dasto lea ulbmilin diehtit olbmuid oaivila dearvvasvuođabálvalusa birra, sin eallinvuogi nugo biepmu ja borgguheami, eallindili ja gullevašvuođa birra. Geat háliidit searvat, leat mielde dutkanprošeavttas mas leat gažadanskovit ja dearvvasvuođaiskkadeapmi. Iskkadeami visot dieđut meannuduvvojit čiegusvuođas.

Dearvvasvuođa- ja eallindilleiskkadeapmi lea dárkilat válddahallon gihppagis mii čuovvu mielde. Jus eahpidat maidege, sáhtát gulahallat minguin tlf. 78 46 89 04 dahje e-poasta: helseus@fagmed.uit.no

Dán láhkai sáhtát searvat: (russe bajimučča gažadanskovis «mieđan searvamii» buohta)

A. Jus háliidat searvat dearvvasvuođaiskkadeapmái ja dutkanprošektii, de russet **A** čuoggá, deavddát gažadanskovi ja máhcahat dan midjiide čuovvu konfaluhtas. Maŋŋil oačču reivve mas čuožžu goas ja gosa boađát oktan ođđa gažadanskoviin.

B. Jus háliidat searvat dušše dutkanprošeavtta álgooasis almmá dearvvasvuođaiskkadeami haga, de russet **B** čuoggá, deavddát gažadanskovi ja máhcahat dan midjiide čuovvu konfaluhtas.

C. Eat rása jus russet **C** čuoggá ja máhcahat gažadanskovi midjiide. Rássan lea čálalaččat.

Datatilsynet lea addán sierralobi rádjat iskkadeami dieđuid ja dutkanprošeavtta lea rávven *Regional komite for medisinsk forskningsetikk i Nord-Norge.*

Dutkama dáfus lea hui miellagiddevaš ahte oažžut nu olu dieđuid go vejolaš. Don searvvat eaktodáhtolaččat ja sáhtát, maŋŋil go leat miehtan searvamii, geassádit vuođuškeahttá ja dutnje čuozakeahttá. Seamma guoská jus álggus juo ii hálit searvat. Dieđuid maid leat almmuhan sáhtát bivdit sihkkut.

Bohtosiid almmuhat mediain, ja čállo raporta dearvvasvuođa- ja eallindilleiskkadeamis go dat lea loahpahuvvon.

Sii geat čađahit olles dearvvasvuođa- ja eallindilleiskkadeami leat mielde vuorbádeamen 3 mátkeskeaŋkakoartta man árvu lea 10 000,– ru. guđesge. Doaivut ahte su. 15000 olbmo servet.

Dearvvuođaiguin

Anne Kirsten Anti
Sámi dearvvašvuođadutkama guovddáš, Kárášjohka

Eiliv Lund
Institutt for samfunnsmedisin
Romsa

Per G. Lund-Larsen
Nasjonalt folkehelseinstitutt
Oslo

INFORMERT SAMTYKKE

Jeg har lest informasjonen om undersøkelsen og samtykker i at (stryk det / de avsnitt du reserverer deg mot):

1. Jeg kan bli kontaktet med anbefaling om oppfølging, behandling eller for å forebygge sykdom.

2. Opplysningene mine kan brukes i medisinsk forskning til å kartlegge og finne årsaker til helse, sykdom og levekår. All bruk av opplysningene i eventuell framtidig medisinsk forskning vil bare bli brukt dersom Regional komité for medisinsk forskningsetikk og Datatilsynet ikke har noen innvendinger mot dette.

3. Etter godkjenning fra Datatilsynet kan opplysningene mine settes sammen med opplysninger om meg i andre registre for forskningsformål. I alle disse tilfellene blir navnet og personnummeret mitt fjernet. Det kan være registre om trygd, sykdom, inntekt, utdanning, yrke, og opplysninger fra de tidligere hjerte- og kar undersøkelsene. Eksempler på slike registre er Kreftregistret, Dødsårsaksregistret og folketellingene. Forsikringsselskaper vil ikke få tilgang til dataene.

4. Blodprøven min kan lagres og brukes til medisinsk forskning og genetiske analyser for å finne årsak til sykdom. All bruk av denne prøven vil bare skje i samsvar med godkjenning fra Datatilsynet og etter at Regional komite for medisinsk forskningsetikk i Nord- Norge har vurdert de etiske sidene ved gjennomføring av prosjektet.

....

sted og dato underskrift

DIEĐIHUVVON MIEHTAN

Lean lohkan dieđuid iskkadeami birra ja mieđan ahte (sihko dan / daid osiid maidda várašat):

1. Sáhttá muinna váldit oktavuođa go áigu rávvet čuovvoleami, dálkkodit dahje eastadit dávddaid.

2. Mu dieđuid sáhttá atnit medisiinnalaš dutkamii kártet ja gávdnat dearvvasvuođa, dávddaid ja eallindili árttaid. Visot dieđuid geavaheapmi soaiti boahttevaš medisiinnalaš dutkamii, adno dušše jus Regional komite for medisinsk forskningsetikk ja Datatilsynet eai vuosttal dan.

3. Datatilsynet dohkkeheami vuođul, sáhttá mu dieđuid čohkket mu dieđuiguin eará registariin dutkandoaimmaide. Visot dáid oktavuođain sihkko mu namma ja personnummar. Sáhttet leat oaju, dávddaid, sisaboađu, oahpu ja fidnu birra registarat ja dieđut ovddeš váibmo- ja suotnaiskkademiin. Dákkár registariid ovdamearkkat leat Kreftregistret, Dødsårsaksregistret ja olmmošlohkamat. Dáhkádusfitnodagat eai beasa dáid dieđuid oaidnit.

4. Mu varraiskkus sáhttá ráddjot ja adnot medisiinnalaš dutkamii ja genetalaš analysaide gávnnahit dávddaid árttaid. Dán iskosa juohke geavaheapmi geavvá dušše Datatilsynet dohkkeheami mielde ja maŋŋil go Regional komite for medisinsk forskningsetikk i Nord- Norge lea árvvoštallan prošeavtta čađaheami ehtalaš beliid.

....

báiki ja beaivi vuolláičála

IN-25132027-3 3000 11.2002 e.s.trykk-Oslo

Muittuhus – Påminnelse

Dál leat mannan čađa dearvvasvuođa- ja eallindilleiskkadeami boahtán vástádusaid guovlluin gos ásset sámit ja dážat.

Váldit dál duinna oktavuođa gullan dihtii ahte leatgo muitán sáddet gažadanskovi maid mis ožžot duvle.

Jus gieskat leat máhcahan skovi, de it galgga dán muittuhusas beroštit.

Ii leat vel menddo maŋŋit sáddet skovi. Bija veaháš áiggi vástidit gažaldagaid. Gii diđeš, soaittát leat nu lihkoš ahte vuoittát 10000,– ru. árvosaš mátkeskeaŋkakoartta. Jus dus ii šat leat gažadanskovvi, de sáhtát oažžut ođđa go jearat: Institutt for samfunnsmedisin, Universitetet i Tromsø, 9037 TROMSØ, tlf. 77 64 66 38, Bente A. Augdalas.

Vi har nå gått igjennom innkomne svar fra helse- og levekårsundersøkelsen fra områder med samisk og norsk bosetting.

Vi tar kontakt med deg nå for å høre om du har glemt å sende inn spørreskjemaet som du mottok fra oss for en stund siden.

Dersom du nylig har returnert skjemaet, ber vi deg se bort fra denne henvendelsen.

Det er fremdeles ikke for sent å sende inn skjemaet. Sett av litt tid til å besvare spørsmålene. Hvem vet, kanskje blir du den heldige vinner av et reisegavekort til en verdi av kr. 10000,–. Dersom du ikke lenger har spørreskjemaet, kan du få et nytt ved å kontakte: Institutt for samfunnsmedisin, Universitetet i Tromsø, 9037 TROMSØ, tlf. 77 64 66 38 v/ Bente A. Augdal.

Ustitlaš dearvvuođaiguin / Med vennlig hilsen

Institutt for samfunnsmedisin, Universitetet i Tromsø
Sámi dearvvašvuođadutkama guovddáš /Senter for samisk helseforskning
Nasjonalt folkehelseinstitutt

Porto betalt.

UNIVERSITETET I TROMSØ

folkehelseinstituttet

1. YOUR OWN HEALTH

What is your current state of health? *(Mark only one)*
☐ Poor ☐ Not so good ☐ Good ☐ Very good

Do you have or have you had the following?	Yes	No	Age first time
Asthma	☐	☐	
Chronic bronchitis, emphysema, COPD	☐	☐	
Diabetes	☐	☐	
Fibromyalgia/chronic pain syndrome	☐	☐	
Psychological problems for which you have sought help	☐	☐	
Myocardial infarction (heart attack)	☐	☐	
Angina pectoris (heart cramp)	☐	☐	
Cerebral stroke/brain haemorrhage	☐	☐	
Multiple sclerosis	☐	☐	
Ulcerative colitis	☐	☐	

	Yes	No
Do you get chest pain or discomfort when walking up hills or stairs, or walking fast on level ground?	☐	☐
Do you get such pain or discomfort even if you are resting?	☐	☐

2. MUSCULAR AND SKELETAL PAIN

	Yes	No
Have you *during the last year* **suffered from pain and/or stiffness in muscles or joints that has lasted for** *at least 3 months***?**	☐	☐

Have you ever had the following?	Yes	No	Age last time
A wrist/forearm fracture?	☐	☐	
A hip fracture?	☐	☐	

3. STOMACH AND INTESTINAL SYMPTOMS

	Yes	No
Have you experienced pyrosis/heartburn almost daily for at least a week?	☐	☐
Have you ever had stomach pains/aches lasting for at least 2 weeks?	☐	☐

If yes, where in the stomach are the pains situated? *(Mark only one)*
☐ Upper part ☐ Lower part ☐ The whole stomach

Normally, for how long are the stomach pains present? *(Mark one)*
For periods of weeks in length ☐
For periods of months in length ☐
Always ☐

	Yes	No
Do you often suffer from flatulence, a rumbling stomach or much wind?	☐	☐

What consistency is your stool usually? *(Tick one or more boxes)*
☐ Normal ☐ Loose ☐ Hard and lumpy
☐ Alternating hard and loose ☐ Smelly

	Yes	No
Do you sometimes have three stools per day or more?	☐	☐
Have you had stomach/intestinal problems after consuming milk?	☐	☐

Are there others in your family with similar stomach symptoms?
☐ Mother ☐ Father ☐ Siblings ☐ Child ☐ None

4. OTHER PAINS/PROBLEMS

Listed below are some symptoms or problems. Have you experienced any of these *during the last week* **(including today)?**
(Tick one box for each item)

	Not affected	Slightly affected	Affected quite a lot	Severely affected
Suddenly scared for no reason	☐	☐	☐	☐
Feeling fearful or anxious	☐	☐	☐	☐
Faintness or dizziness	☐	☐	☐	☐
Feeling tense or keyed up	☐	☐	☐	☐
Blaming yourself for things	☐	☐	☐	☐
Insomnia/sleeplessness	☐	☐	☐	☐
Feeling blue/melancholic	☐	☐	☐	☐
Feeling of worthlessness/of little value	☐	☐	☐	☐
Feeling everything is an effort	☐	☐	☐	☐
Feeling hopeless about future	☐	☐	☐	☐
Thinking of ending your life	☐	☐	☐	☐

5. ILLNESS IN THE FAMILY

	Yes	No	Don't know
Have one or more of your parents or siblings had a heart attack or angina (heart cramp)?	☐	☐	☐

Tick off relatives who have, or have ever had, any of the following conditions, and report the age of when they got the illnesses.
(If several siblings were affected by a condition, report the one who got the illness at the youngest age)

	Mother	Father	Sister	Brother	Child	None	Age first time
Myocardial infarction before age 60	☐	☐	☐	☐	☐	☐	
Myocardial infarction after age 60	☐	☐	☐	☐		☐	
Diabetes	☐	☐	☐	☐	☐	☐	
Cerebral stroke or brain hemorrhage	☐	☐	☐	☐	☐	☐	
Asthma	☐	☐	☐	☐	☐	☐	
Colon cancer	☐	☐	☐	☐	☐	☐	
Breast cancer	☐		☐		☐	☐	
Ovarian cancer	☐		☐		☐	☐	

	Brothers	Sisters
How many siblings do you have?		

6. USE OF MEDICATION

Medicines, in this context, means medicines bought at a pharmacy. Food supplements and vitamins are not included here.

Do you take any of the following?	Currently	Previously, but not now	Never used
Medications for high blood pressure	☐	☐	☐
Cholesterol reducing medication	☐	☐	☐
Insulin	☐	☐	☐
Tablets for diabetes	☐	☐	☐

How often during the last 4 weeks have you used the following medications? *(Tick one box for each line)*

	Not used for the last 4 weeks	Less frequently than every week	Every week, but not daily	Daily
Painkillers without prescription	☐	☐	☐	☐
Painkillers with prescription	☐	☐	☐	☐
Sleeping pills	☐	☐	☐	☐
Tranquilizers	☐	☐	☐	☐
Antidepressants	☐	☐	☐	☐
Other prescribed medicines	☐	☐	☐	☐

For those medicines you have ticked off in the last two questions, and you have taken *during the last 4 weeks*:

State the name of the medicines and your reason for taking/ having taken them (disease, symptom): *(Tick one box on each line)*

Brand name of medicine *(one name per line)*	Reason for use of medicine	For how long? Up to one year	For how long? One year or more
		☐	☐
		☐	☐
		☐	☐
		☐	☐
		☐	☐

If there is not enough space here, continue on a separate page and enclose it with the form.

7. FOOD AND BEVERAGES

How often do you usually eat the following foods?

	Rarely/ never	1–3 per month	1–3 per week	4–6 per week	1–2 per day	3 or more per day
Fruit	☐	☐	☐	☐	☐	☐
Berries	☐	☐	☐	☐	☐	☐
Cheese (all types)	☐	☐	☐	☐	☐	☐
Potatoes	☐	☐	☐	☐	☐	☐
Boiled vegetables	☐	☐	☐	☐	☐	☐
Fresh vegetables/salad	☐	☐	☐	☐	☐	☐

What type of fat do you usually use? *(Tick one box for each line)*

	Do not use	Butter	Hard margarine	Soft/light margarine	Oils	Other
On bread	☐	☐	☐	☐	☐	☐
For cooking	☐	☐	☐	☐	☐	☐

Do you use the following food supplements?

	Yes, daily	Sometimes	No
Cod liver oil or cod liver oil capsules	☐	☐	☐
Fish oil capsules (omega 3)	☐	☐	☐
Vitamins and/or mineral supplements	☐	☐	☐

How much do you normally drink of the following?

(Tick one box on each line)

	Rarely/ never	1–6 glasses per week	1 glass per day	2–3 glasses per day	4 glasses a day or more
Full-fat milk, full-fat curdled milk or yoghurt	☐	☐	☐	☐	☐
Semi-skimmed milk, semi-skimmed curdled milk or low-fat yoghurt	☐	☐	☐	☐	☐
Skimmed milk or skimmed curdled milk	☐	☐	☐	☐	☐
Semi-skimmed milk	☐	☐	☐	☐	☐
Fruitjuice	☐	☐	☐	☐	☐
Water	☐	☐	☐	☐	☐
Soft drinks/cola drinks with sugar	☐	☐	☐	☐	☐
Soft drinks/cola drinks without sugar	☐	☐	☐	☐	☐

How many cups of coffee and tea do you usually drink per day?

(Write 0 for the types you do not drink daily)

	Number of cups
Filtered coffee	☐☐
Boiled coffee (coarsely ground coffee for brewing)	☐☐
Other coffee	☐☐
Tea	☐☐

How often during the last year have you consumed alcohol?

(Low-alcohol beer and non-alcoholic beer are not included)

- Never consumed alcohol ☐
- 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 those who have consumed alcohol during the past year:

When you drink alcohol, how many glasses or drinks do you normally drink? *Number of glasses or drinks* ☐☐

Approximately how many times during the last year have you consumed alcohol equivalent to 5 glasses or drinks within 24 hours? *Number of times* ☐☐

Which of the following types of alcohol do you normally drink?

(Tick one or more boxes)

☐ Beer ☐ Wine ☐ Spirits

8. SMOKING AND SNUFF USE

How many hours a day do you normally spend in smoke-filled rooms? *(Number of whole hours)* ☐☐

Did any adults living at home with you while you were growing up smoke?	Yes	No
	☐	☐

Do you currently, or did you previously live with a daily smoker after your 20th birthday? ☐ Yes ☐ No

Are you currently, or were you previously a daily smoker? ☐ Yes, currently ☐ Yes, previously ☐ Never

If you are current a daily smoker, do you smoke the following?

	Yes	No
Cigarettes	☐	☐
Cigars/cigarillos/pipe	☐	☐
Rolling tobacco	☐	☐

If you previously smoked daily, how many years is it since you stopped smoking? ☐☐ *(Number of years)*

If you currently smoke, or have smoked before, how many cigarettes do/did you smoke per day? ☐☐ *(Number of cigarettes)*

If you currently smoke, or have smoked before, how old were you when you began smoking daily? ☐☐ *(Age in years)*

If you currently smoke, or have smoked before, how many years in all have you smoked daily? ☐☐ *(Number of years)*

Do you take or have you been taking snuff daily? ☐ Yes, currently ☐ Yes, previously ☐ Never

If you have been taking snuff, for how many years in total have you been taking snuff? ☐☐ *(Number of years)*

9. EXERCISE AND PHYSICAL ACTIVITY

How has your physical activity *in leisure time* **been** *during this last year***?** *(Think of your weekly average for the year. Time spent going to work counts as leisure time. Answer both questions)*

	Hours per week			
	None	Less than 1 hour	1–2 hours	3 hours or more
Light activity (not sweating or out of breath)	☐	☐	☐	☐
Hard physical activity (sweating/out of breath)	☐	☐	☐	☐

Describe your exercise and physical exertion *in leisure time***. If your activity varies much, for example between summer and winter, then give an average. The question refers** *only to the last twelve months***.** *(Tick the box that is most appropriate)*

Reading, watching TV, or other sedentary activity ☐

Walking, cycling, or other forms of exercise *at least 4 hours a week* *(This should include walking or cycling to work, Sunday stroll/walk, etc.)* ☐

Participation in recreational sports, heavy gardening, etc. *(Note: duration of activity at least 4 hours a week)* ☐

Participation in hard training or sports competitions regularly and *several times a week* ☐

10. EDUCATION AND WORK

How many years of schooling/education have you completed? *(Count all years you have attended school or been studying)* ☐☐ *(Number of years)*

How content are you with your job?
☐ Very content ☐ Content ☐ Discontent ☐ Very discontent

Do you believe that you are in danger of losing your current work or income within the next 2 years? ☐ Yes ☐ No

Do you receive any of the following benefits?

	Yes	No
Sickness benefit/Sick pay	☐	☐
Rehabilitation benefit	☐	☐
Social welfare benefits	☐	☐
Transition benefit for single parents	☐	☐

11. THE REST OF THE QUESTIONNAIRE IS TO BE ANSWERED BY WOMEN ONLY

How old were you when you started menstruating? ☐☐ (Age in years)

If you no longer menstruate, how old were you when you stopped menstruating? ☐☐ (Age in years)

Are you pregnant at the moment?
☐ Yes ☐ No ☐ Uncertain ☐ Past fertile age

How many children have you given birth to? ☐☐ *(Number of children)*

If you have given birth, enter what year each child was born and how many months you did breastfeed after the birth?
(If you didn't breastfeed, write 0)

Children	Year of birth	Breastfed number of months
1st child	☐☐☐☐	☐☐
2nd child	☐☐☐☐	☐☐
3rd child	☐☐☐☐	☐☐
4th child	☐☐☐☐	☐☐
5th child	☐☐☐☐	☐☐

(If you have had more children, use an extra sheet of paper)

Do you use or have you ever used the following? *(Tick one box on each line)*

	Currently	Previously, but not now	Never used
Contraceptive pills/minipill/ contraceptive injection	☐	☐	☐
Hormonal intrauterine device	☐	☐	☐
Estrogen (tablets or patches)	☐	☐	☐
Estrogen (cream or suppositories)	☐	☐	☐

If you use/have used prescribed estrogen, for how many years have you used it? ☐☐ *(Number of years)*

If you use contraceptive pills, a hormonal intrauterine device, or estrogen, what brand do you currently use? *(Specify)*

USE OF HEALTH SERVICES

How many times *during the past year* **have you personally used the following?** *(Tick one box on each line)*

	None	1–3 times	4+
GP (general practitioner)	☐	☐	☐
Medical specialist	☐	☐	☐
Emergency GP	☐	☐	☐
Admission to a hospital	☐	☐	☐
Home nursing care	☐	☐	☐

	None	1–3 times	4+
Home aid, organized by the municipality	☐	☐	☐
Physiotherapist	☐	☐	☐
Chiropractor	☐	☐	☐
Dentist	☐	☐	☐
Alternative medical practitioner	☐	☐	☐

How many doctors have you seen in the last 12 months? ☐☐ *(Number)*

Have you been given a regular GP, whose name you know? ☐ Yes ☐ No

When you are being examined, which language do you and your doctor communicate in? *(Tick one or more boxes)*

☐ Norwegian ☐ Sami ☐ Use an interpreter
☐ Other language

Do you and your doctor sometimes misunderstand each other due to linguistic problems?

☐ Never ☐ Rarely ☐ Sometimes ☐ Often ☐ Not sure

If an interpreter is needed, is your doctor good enough to request one?

☐ Yes, always ☐ Yes, most of the time ☐ No, not always
☐ No, never ☐ Don't like to use interpreter

How satisfied/dissatisfied are you with the following aspects of the municipal health service in your municipality?
(Tick one box on each line)

	Very satisfied	Satisfied	Dis-satisfied	Don't know
The distance to your doctor	☐	☐	☐	☐
Your doctor's availability by telephone	☐	☐	☐	☐
How soon you can get an appointment with your doctor	☐	☐	☐	☐
How long you are allowed with your doctor	☐	☐	☐	☐
The chance you get to describe your pains and problems	☐	☐	☐	☐
Your doctor's understanding of your cultural background	☐	☐	☐	☐
The information your doctor gives about your health and the examination and treatment you get	☐	☐	☐	☐
Your doctor's language skills (Sami or Norwegian)	☐	☐	☐	☐
The local health services in your municipality as a whole	☐	☐	☐	☐
On the whole, how satisfied/ dissatisfied are you with the local health services in your municipality	☐	☐	☐	☐

How long is it since you last went to see a doctor? *(Report whole numbers)* Years ☐☐ Months ☐☐

If you have ever used an alternative practitioner, which did you use? *(Tick one or more boxes)*

A traditional healer *(guvllar, reader, "blåser", laying on of hands)* ☐
A (modern) healer ☐
An acupuncture practitioner ☐
A zone therapist, homeopath, kinesiologist etc. ☐

How long is it since you last used an alternative practitioner? *(Report whole numbers)* Years ☐☐ Months ☐☐

Suppose you need help/assistance from the local health- and social services *(home nursing care, home assistance services, social services, physiotherapy, etc.)***:**

	Yes	No	Uncertain
Do you know where to go (who to contact)?	☐	☐	☐
Do you feel confident you will receive help if you need it?	☐	☐	☐
If you already receive help from local health and social services, are you satisfied with the help they offer?	☐	☐	☐

INJURIES/ACCIDENTS

Have you been in accidents that resulted in treatment by a doctor and/or hospital admission?

	Yes	No	Number of times
Doctor	☐	☐	☐☐
Hospital admission	☐	☐	☐☐

If yes, what kind of accidents have you been treated for?

	At work	At home	During leisure time	No
Car accident	☐	☐	☐	☐
Motor cycle accident	☐	☐	☐	☐
Snowmobile accident	☐	☐	☐	☐
Quadbike accident	☐	☐	☐	☐
Tractor accident	☐	☐	☐	☐
Accident caused by falling	☐	☐	☐	☐
Cutting injury	☐	☐	☐	☐
Other	☐	☐	☐	☐

Has/have the accident(s) led to reduced ability to work?

☐ Completely ☐ Partly ☐ Not at all

FAMILY AND LINGUISTIC BACKGROUND

People of different ethnic backgrounds live in Northern Norway. That is, they speak different languages and have different cultures. Examples of ethnic background, or ethnic group, are Norwegian, Sami and Kven.

Which language did/do you, your parents, and your grand parents speak at home? *(Tick one or more boxes)*

	Norwegian	Sami	Kven	Other, specify
Mother's father	☐	☐	☐	☐
Mother's mother	☐	☐	☐	☐
Father's father	☐	☐	☐	☐
Father's mother	☐	☐	☐	☐

	Norwegian	Sami	Kven	Other, specify
Father.....	☐	☐	☐	☐
Mother.....	☐	☐	☐	☐
Myself.....	☐	☐	☐	☐

What are your, your father's, and your mother's ethnic backgrounds? *(Tick one or more boxes)*

	Norwegian	Sami	Kven	Other, specify
My ethnic background ...	☐	☐	☐	☐
My father's ethnic background ...	☐	☐	☐	☐
My mother's ethnic background ...	☐	☐	☐	☐

What do you consider yourself to be? *(Tick one or more boxes)*

☐ Norwegian ☐ Sami ☐ Kven
☐ Other, specify:

EMPLOYMENT/ECONOMY

What type of work/livelihood do you have? *(Tick one or more boxes)*

☐ Full time job with a fixed salary
☐ Part time job with a fixed salary
☐ Seasonal work ☐ Self-employed
☐ Unemployed ☐ Homemaker (fulltime housework)
☐ Old-age pension ☐ Disability pension
☐ Other, specify:

Would you be willing to move if you were offered work somewhere else?

☐ Yes ☐ No ☐ Parts of the year ☐ Uncertain

If you are out of work, for how long have you been seeking employment? *(Report whole numbers)*

Years ☐☐ Months ☐☐

If you are self-employed, what work do you do?

(Tick one or more boxes)

☐ Reindeer herding ☐ Fishing ☐ Farming
☐ Forestry ☐ Business
☐ Other, specify:

How many persons are living in your household?..... *(Number of persons)* ☐☐

What is your family's/household's gross income each year?

☐ Less than 150 000 NOK ☐ 150 000–300 000 NOK
☐ 301 000–450 000 NOK ☐ 451 000–600 000 NOK
☐ 601 000–750 000 NOK ☐ More than 750 000 NOK

How often do you participate in gambling (national lottery, football betting, gambling machines, etc.)?

☐ Never/rarely ☐ 1–3 times a month ☐ Once a week
☐ 2–6 times a week ☐ Daily

For how much money do you gamble per week on average?

☐ Less than 100 NOK ☐ 100–500 NOK
☐ 501–1000 NOK ☐ More than 1000 NOK

BULLYING

By bullying we mean when one or more persons *systematically and over time* **say or do bad things against you, and you have difficulty in defending yourself against them.**

Have you experienced bullying?

☐ Yes, in the last 12 months ☐ Yes, previously ☐ No

If you have been bullied, what kind of bullying did you experience?

(Tick one or more boxes)

☐ Talking behind your back/gossip ☐ Being ignored
☐ Discriminating remarks
☐ Other, specify:

Can you state where the bullying takes/took place?

☐ At school ☐ At boarding school/dormitory
☐ At work ☐ In local community
☐ Other, specify:

Appendix B

SAMINOR 2 Clinical Survey

- Pamphlet
- Information brochure
- Invitation letter (example from the municipality of Evenes)
- Informed written consent form
- Questionnaires (english translation):
    - 40–69 years
    - 70–79 years

All listed items and their Norwegian versions are available at www.saminor.no.

Foto: Bjørn-Kåre Iversen, helsefak. uit.no

VI KOMMER NÅ TIL DIN KOMMUNE

Du vil i løpet av noen uker motta en forespørsel i posten fra Universitetet i Tromsø om å delta i en helseundersøkelse. Resultatene vil kunne bidra til å fremme folkehelse og forbedre velferdstilbud i nord.

HVORFOR SPØR VI DEG?

Alle mellom 40 – 79 år i din kommune vil bli invitert. Hver deltaker er like viktig, enten du er ung eller gammel, kvinne eller mann, frisk eller syk. Godt oppmøte er viktig for gode forskningsresultater.

UNDERSØKELSER AV DEG

- Høyde og vekt
- Liv- og hoftevidde
- Blodtrykk og puls
- Blodprøve

Vi ber deg også om å fylle ut et spørreskjema.

TILBAKEMELDING PÅ RESULTATER

Dersom du ønsker det, vil du ved undersøkelsen få dine egne resultater på høyde, vekt, liv- og hoftemål, blodtrykk, puls, blodprosent og langtidsblodsukker.

DIN SIKKERHET

Det er frivillig å delta.

Din sikkerhet er høyt ivaretatt. All behandling av helseopplysninger eller prøvemateriale skjer i tråd med helseforskningsloven. Alle opplysninger og prøver anonymiseres og blir da behandlet uten navn og fødselsnummer eller andre direkte gjenkjennbare opplysninger.

Undersøkelsen er godkjent av Datatilsynet og REK Nord – Regional komite for medisinsk og helsefaglig forskningsetikk.

Foto: Bjørn Erik Rygg Lunde/ Nordlandssykehuset

VI VIL HA ØKT KUNNSKAP OM

- Kosthold
- Diabetes
- Hjerte-karsykdommer
- Miljøgifter
- Tannhelse
- Søvn

REISEGAVEKORT

Alle som deltar vil være med i trekning av to reisegavekort verdt kr 10 000,- hver. I tillegg vil det trekkes to ekstra reisegavekort i den kommunen som har best deltagelse. Ut over dette gis det ingen økonomisk kompensasjon for deltakelse i studien.

VI KOMMER NÅ TIL DIN KOMMUNE

Du vi i løpet av noen uker motta et brev om sted og tid for undersøkelsen. Ved å delta, bidrar du til spennende og samfunnsnyttig forskning på helse og livsstil i nord.

DERSOM DU HAR SPØRSMÅL

ta gjerne kontakt med oss på telefon eller via e-post.

Senter for samisk helseforskning
Institutt for samfunnsmedisin
Universitetet i Tromsø
9037 Tromsø
http://site.uit.no/helseoglivsstil/
E-post: saminor@ism.uit.no
Telefon: 404 90 467

SAMINOR 2

UNIVERSITETET I TROMSØ UIT
DET HELSEVITENSKAPELIGE FAKULTET
uit.no/helsefak

GRAFISK FORM: BJØRN-KÅRE IVERSEN, FORMIDLINGSTJENESTEN, UIT.NO/HELSEFAK

UNIVERSITETET I TROMSØ UIT
DET HELSEVITENSKAPELIGE FAKULTET

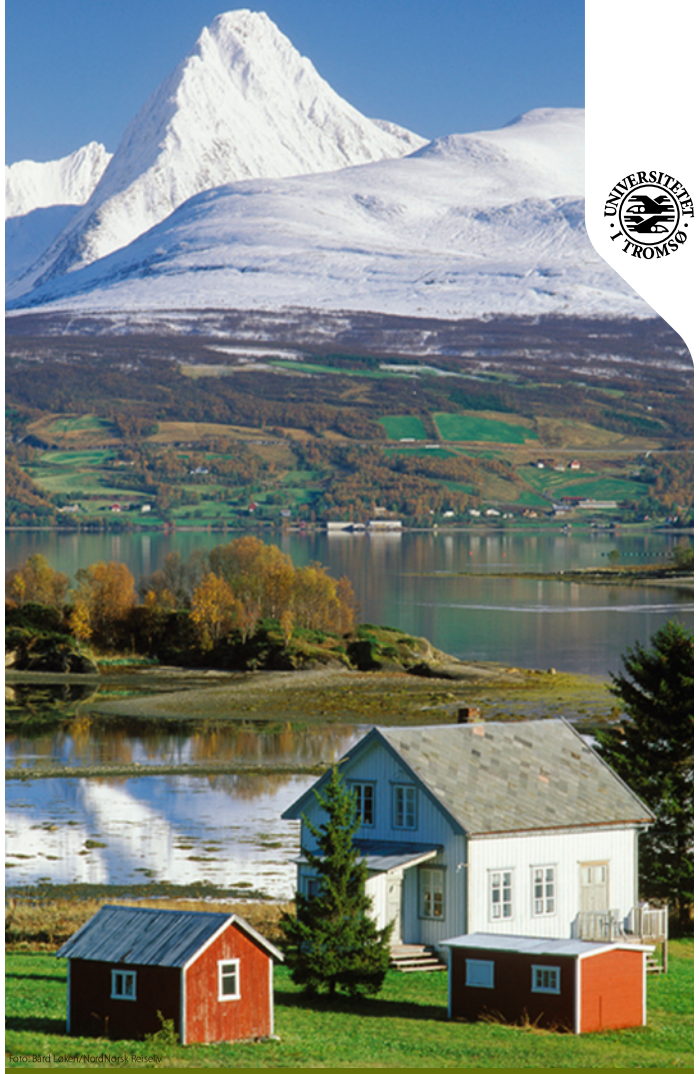

Foto: Bård Løken/NordNorsk Reiseliv

Helse- og livsstils-undersøkelse

BAKGRUNN OG HENSIKT

Dette er et spørsmål til deg om å delta i et forskningsprosjekt for å få mer kunnskap om helse, sykdom og levekår i områder med samisk og norsk bosetting. Du som deltar i denne undersøkelsen får sjekket om du har bestemte såkalte livsstilssykdommer eller om det er fare for at du kan få dem.

Du er invitert til å være med i denne studien fordi du er i alderen 40-79 år og tilhører en av de utvalgte kommuner. Studien utføres av Senter for samisk helseforskning, Institutt for samfunnsmedisin ved Universitetet i Tromsø.

HVA INNEBÆRER STUDIEN?

Du inviteres til å svare på vedlagte spørreskjema og ta det med når du møter opp på anvist forskningsstasjon i din kommune. Her vil det gjøres målinger av blodtrykk, puls, høyde, vekt og liv-hoftevidde, og det blir også tatt blodprøve.

Blodprøvene kan senere bli analysert for næringsstoffer, miljøgifter, fettstoffer og markører som kan knyttes til livsstilssykdommer eller tilstander som for eksempel diabetes (sukkersyke), hjerte-karsykdommer og søvnforstyrrelser. Genetiske analyser av blodet for å finne mulige årsaker til nevnte livsstilssykdommer/tilstander kan også bli aktuelt.

All bruk av blodprøvene krever godkjenning av Regional komité for medisinsk og helsefaglig forskningsetikk – REK nord.

Vedlagt følger informasjon om tid og sted for undersøkelsen. Hvis den foreslåtte tiden ikke passer, kan du møte opp uten å melde fra på forhånd.

MULIGE FORDELER OG ULEMPER

Det forventes ingen risiko forbundet med deltagelse i denne undersøkelsen. Blodprøven blir tatt ved stikk i blodåre i underarmen. Selve undersøkelsen vil ta om lag en halv time. Du vil på stedet få tilbud om resultater på egne målinger som blodtrykk, puls, høyde, vekt og liv-hoftevidde, blodprosent og HbA1c (gjennomsnittlig blodsukker de siste 6-8 ukene). Du kan reservere deg mot å få vite resultatene av prøvene dine. Men hvis et av disse prøveresultatene er slik at det er nødvendig med rask legebehandling, vil du uansett umiddelbart få tilbakemelding. Deltagelse i denne studien erstatter ingen legeundersøkelse. Dersom du har mistanke om noe galt med din helse, må du derfor i tillegg oppsøke din egen fastlege.

Foto: Bjørn-Kåre Iversen, helsefak. uit.no

HVA SKJER MED PRØVENE OG INFORMASJONEN OM DEG?

Prøvene tatt av deg og informasjonen som registreres om deg skal kun brukes slik som beskrevet i hensikten med studien. Videre behandling av helseopplysninger eller prøvemateriale skjer i tråd med helseforskningsloven og eventuell annen aktuell lovgivning. Alle opplysninger og prøver vil bli behandlet uten navn og fødselsnummer eller andre direkte gjenkjennende opplysninger. En kode knytter deg til dine opplysninger og prøver gjennom en navneliste. Det betyr at opplysningene er avidentifisert. Det er kun autorisert personell knyttet til prosjektet som har adgang til navnelisten og som kan finne tilbake til deg. Det vil heller ikke være mulig å identifisere deg i resultatene av studien når disse publiseres. Du kan seinere bli kontaktet med forespørsel om du vil svare på tilleggsspørreskjema.

Opplysninger som registreres om deg er basert på spørreskjemaopplysninger, mål fra helseundersøkelsen og blodprøveanalyser. Etter godkjenning fra Datatilsynet og/eller REK kan opplysningene dine settes sammen med opplysninger om deg i andre registre for forskningsformål. Dette kan være registre om trygd, sykdom, inntekt, utdanning, yrke og opplysninger fra andre helseundersøkelser som du har deltatt i. Aktuelle registre er Kreftregisteret, Dødsårsaksregisteret, Folkeregisteret, Reseptregisteret, Medisinsk fødselsregister, Hjerte- og karregisteret og andre nasjonale registre over sykdommer som det forskes på i denne undersøkelsen samt registre i Statistisk sentralbyrå og folketellinger. I alle disse tilfellene blir navnet og personnummeret fjernet. Forsikringsselskaper eller andre kommersielle institusjoner vil ikke få tilgang til dataene.

Prosjektslutt er satt til 31. desember 2067. Etter dette anonymiseres alle dataene.

BIOBANK

Blodprøvene vil bli lagret i en såkalt forskningsbiobank ved Universitetet i Tromsø eller eventuelt ved et annet nasjonalt lager for biobank med høyeste grad av sikkerhet i forhold til prøvens kvalitet og personvern som er godkjent av aktuelle instanser. Hvis du sier ja til å delta i studien, gir du også samtykke til at blodprøvene inngår i denne biobanken. Universitetet i Tromsø er ansvarshavende for forskningsbiobanken.

BEHANDLINGSANSVARLIG

Universitetet i Tromsø ved administrerende direktør er databehandlingsansvarlig.

RETT TIL INNSYN OG SLETTING AV OPPLYSNINGER OG PRØVER

Hvis du sier ja til å delta i studien, har du rett til å få innsyn i hvilke opplysninger som er registrert om deg. Du har videre rett til å få korrigert eventuelle feil i de opplysningene vi har registrert. Dersom du trekker deg fra studien, kan du kreve å få slettet innsamlede prøver og opplysninger, med mindre opplysningene allerede er inngått i analyser eller brukt i vitenskapelige publikasjoner.

KOMPENSASJON

Det gis ingen økonomisk kompensasjon for deltakelse i studien bortsett fra at alle som deltar vil være med i trekning av to reisegavekort hver verdt kr 10 000,-.
I tillegg vil det trekkes to ekstra reisegavekort i den kommunen som har best deltagelse.

ØKONOMI

Studien og biobanken er finansiert gjennom forskningsmidler fra det Regionale forskningsfond Nord-Norge, de tre nordligste fylkeskommunene, Helse Nord, Sametinget, Universitetet i Tromsø og Helse og omsorgsdepartementet. Ingen av disse instansene har interessekonflikter i undersøkelsen.

FORSIKRING

Deltakerne er dekket gjennom pasientskadeerstatningsloven.

HELSE OG LIVSSTIL

Kosthold – diabetes – hjerte-karsykdommer – miljøgifter – tannhelse – søvn

INFORMASJON OM UTFALLET AV STUDIEN

Resultater av undersøkelsen vil publiseres i internasjonale og nasjonale vitenskapelige tidsskrifter i tillegg til ulike populærvitenskapelige kanaler og media.

FRIVILLIG DELTAKELSE

Det er frivillig å delta i studien. Dersom du ønsker å delta, møter du opp til angitt sted og tidspunkt. Her vil du bli bedt om å signere et samtykke på deltakelse. Du kan når som helst og uten å oppgi noen grunn trekke ditt samtykke til å delta i studien.

Dersom du senere ønsker å trekke deg eller har spørsmål til studien, kan du kontakte oss på vår prosjektelefon: **404 90 467** eller på e-post: saminor@ism.uit.no

Du finner ytterligere informasjon om studien på vår nettside
http://site.uit.no/helseoglivsstil/

VELKOMMEN TIL UNDERSØKELSEN

Magritt Brustad
Prosjektleder
Professor

Ann Ragnhild Broderstad
Forsker
Overlege Dr. med.

SAMINOR 2

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DET HELSEVITENSKAPELIGE FAKULTET
uit.no/helsefak

GRAFISK FORM: BJØRN-KÅRE IVERSEN, FORMIDLINGSTJENESTEN, UIT HSV/HELSEFAK

UNIVERSITETET I TROMSØ UIT
DET HELSEVITENSKAPELIGE FAKULTET

FORESPØRSEL OM DELTAKELSE I HELSE- OG LIVSSTILS-UNDERSØKELSE

Foto: Bård Løken/NordNorsk Reiseliv

UNIVERSITETET I TROMSØ UiT

Helse og livsstil

Kosthold – diabetes – hjerte-karsykdommer – miljøgifter – tannhelse – søvn

Forespørsel om deltakelse i forskingsprosjekt

Vi spør deg om å delta i en helse- og livsstilsundersøkelse som Universitetet i Tromsø nå gjennomfører. Hele befolkningen i alderen 40-79 år i utvalgte distriktskommuner i Nord-Norge får tilbud om undersøkelsen. Skånland og Evenes kommune er først ut.

Vi inviterer deg til å møte opp på denne undersøkelsen som vil finne sted i tidsrommet **17. september til 25. oktober 2012** ved:

Helse- og sosialsenteret på Evenskjer, inngang v/NAV.

For å avvikle undersøkelsen raskest mulig, setter vi opp et visst antall personer i timen.

Du har fått tildelt frammøtetid:

Dato:
Tid:

Om du ikke kan møte opp til avtalt time, er du velkommen til å møte opp når som helst i åpningstiden for drop-in som skissert under. Merk at åpningsdagen åpner vi klokken **12:30**, og vi har lunsj i tidsrommet **12:00 -12:30**.

	Mandag	Tirsdag	Onsdag	Torsdag	Fredag	Lørdag 29.sept og 20.okt
Uke 38, 40, 42	09:30-15:45	09:30-19:30	09:30-15:45	09:30-19:30	09:30-15:15	10:15-14:30
Uke 39, 41, 43	09:30-19:30	09:30-15:45	09:30-19:30	09:30-15:45	09:30-15:15	

Senter for samisk helseforskning / Sámi dearvvašvuođadutkama guovddáš
Det helsevitenskapelige fakultet, Institutt for samfunnsmedisin,
Universitetet i Tromsø, NO-9037 Tromsø
http://saminor.uit.no • E-post: saminor@ism.uit.no
Sentralbord: 77 64 40 00 • Faks: 77 64 48 31• Mobil: 404 90 467

Hva undersøkes?

På stedet undersøker vi ditt blodtrykk, din puls, høyde, vekt og liv-hoftevidde, samt at vi tar en blodprøve av deg.

Ta med ditt utfylte spørreskjema til undersøkelsen

Vi ber deg om å svare på vedlagte spørreskjema og ta dette med for levering på undersøkelsesdagen. Her kan du også få hjelp til utfylling av skjemaet om du trenger det. Du kan la være å svare på enkelte spørsmål. Spørreskjemaet omhandler i hovedsak spørsmål vedrørende hjerte-karsykdommer, diabetes og kosthold. For å kunne beregne næringsinntak (kalorier, næringsstoffer o.l.) er det nødvendig med en grundig kartlegging av hva du normalt spiser.

Forberedelser til undersøkelsen

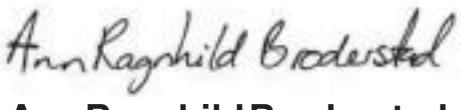
Ha gjerne på et kortermet plagg innerst som ikke strammer da det letter blodtrykksmålingen. Vekt og liv-hoftevidde måles også med lett påkledning og vekt uten sko. Ingen andre forberedelser som fasting o.l. er nødvendig.

Det er frivillig å delta. For mer informasjon om undersøkelsen, vennligst se vedlagte informasjonsfolder. Vi viser også til vår nettside http://site.uit.no/helseoglivsstil/

Har du spørsmål om undersøkelsen, kan du ringe Institutt for samfunnsmedisin ved Universitetet i Tromsø på telefon 77 64 48 36 eller mobil 404 90 467.

Med vennlig hilsen

Magritt Brustad
Prosjektleder
Professor

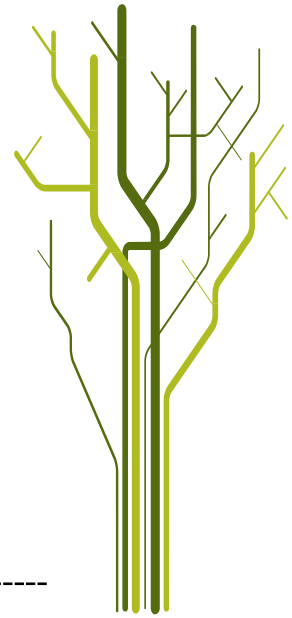


Ann Ragnhild Broderstad
Forsker
Overlege Dr. med.

Senter for samisk helseforskning / Sámi dearvvašvuođadutkama guovddáš
Det helsevitenskapelige fakultet, Institutt for samfunnsmedisin,
Universitetet i Tromsø, NO-9037 Tromsø
http://saminor.uit.no • E-post: saminor@ism.uit.no
Sentralbord: 77 64 40 00 • Faks: 77 64 48 31• Mobil: 404 90 467

UNIVERSITETET I TROMSØ UiT

Helse- og livsstilsundersøkelse



Samtykke til deltakelse i studien

Jeg er villig til å delta i studien

(Signert av prosjektdeltaker, dato)

☐ **Jeg ønsker ikke tilbakemelding på utvalgte prøvesvar**

Senter for samisk helseforskning / Sámi dearvvašvuođadutkama guovddáš
Det helsevitenskapelige fakultet, Institutt for samfunnsmedisin, Universitetet i Tromsø, NO-9037 Tromsø
http://site.uit.no/helseoglivsstil/• E-post: saminor@ism.uit.no
Sentralbord: 77 64 40 00 • Faks: 77 64 48 31 • Mobil: 404 90 467

Survey on health and lifestyle

We kindly request that you fill in the form as thoroughly and accurately as possible, and bring it with you to your scheduled physical examination. The form will be optically scanned. Please use blue or black ink. Use capital letters. Do not use decimals; for example, "0.5" should be rounded off to "1".

1. **In what year were you born?** ..... Year

2. **What is your gender?** ..... Female ☐ Male ☐

3. **What is your marital status?**

☐ Married ☐ Cohabiting ☐ Divorced
☐ Unmarried ☐ Widow/widower

4. **How many people live in your household?** ..... Number of persons

5. **How many years of education have you completed?** ..... Number of years
(Include all years you have attended school or studied)

6. **What is your family's/household's gross income per year?**

☐ Less than NOK 150,000 ☐ NOK 150,000–300,000
☐ NOK 301,000–450,000 ☐ NOK 451,000–600,000
☐ NOK 601,000–750,000 ☐ NOK 751,000–900,000
☐ More than NOK 900,000

Cardiovascular disease

7. **Are you taking medication for high blood pressure?** ..... Yes, currently ☐ Previously, but not now ☐ Never used ☐

8. **If you are taking high blood pressure medication, or have taken high blood pressure medication in the past, at what age did you start taking this type of medicine?** Age

9. **Have you ever had one or more heart attacks?**

☐ No, never ☐ One heart attack ☐ Two heart attacks ☐ Three or more heart attacks

10. **If yes, at what age did you have your first heart attack?** ..... Age

11. **Do you suffer from angina pectoris (heart cramp)?** ..... ☐ Yes ☐ No

12. **If yes, at what age did your symptoms of angina pectoris first emerge?** ..... Age

13. **If yes, how often have you experienced such pain in the past month?**

Rarely ☐ Once a week ☐ 2-3 times a week ☐ 4-6 times a week ☐ 7 times a week or more ☐

14. **Have you had heart (bypass) surgery?** ☐ Yes ☐ No

15. **Have you had your arteries unblocked/ had stent(s) placed** ☐ Yes ☐ No

16. **Has your doctor told you that you have atrial fibrillation?** ☐ Yes ☐ No

Physical activity

17. **We will now ask you to state your physical activity at the ages of 14, 30 and at your current age, on a scale from very low to very high.** The scale below runs from 1 to 10. Physical activity includes both housework and activity at work, as well as exercise and other physical activities such as walking/hiking, etc. Mark the number that best matches your level of activity:

	Very low									Very high
Age	1	2	3	4	5	6	7	8	9	10
14 years	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
30 years	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Current age	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Diabetes

18. **Have you ever been diagnosed with diabetes (elevated blood sugar levels)?** ..... ☐ Yes ☐ No
If no, please skip to question 28 regarding eating habits

19. **If yes, please specify your diabetes diagnosis:**
(chose one or more options)

Gestational diabetes ..... ☐
Type 1 diabetes ..... ☐
Type 2 diabetes ..... ☐

20. **How was your diabetes discovered?**

I consulted my doctor/physician because of symptoms ..... ☐ Yes ☐ No

It was discovered without the appearance of symptoms (medical certificate, work-related medical examination, pregnancy health examination, medical consultation for illness other than diabetes, etc.) ☐ Yes ☐ No

21. **At what age was your diabetes discovered/ diagnosed?** ..... Age

INSULIN

22. **Are you taking insulin for your diabetes?** ..... Yes, currently ☐ Previously, but not now ☐ Never used ☐

If you are taking (or have taken) insulin:

23. **At what age did you start your insulin treatment?** ..... Age ☐☐

24. **How many times per day do you/did you usually take insulin?** ..... ☐☐ times

25. **In total, how many units of insulin do you/did you take on an average day?** ..... ☐☐ units (E)

ORAL MEDICATION

26. **Are you taking oral medication for diabetes?** ..... Yes, currently ☐ Previously, but not now ☐ Never used ☐

If you are taking or have taken oral medication:

27. **At what age did you start taking oral medication for diabetes?** ..... Age ☐☐

Eating habits

Mark the square below the number that best describes your eating habits, taking the past four weeks into consideration:

28. **How satisfied are you with your eating habits?** (Choose only one option)

	1	2	3	4	5	6	7	
Very dissatisfied	☐	☐	☐	☐	☐	☐	☐	Very satisfied

29. **Have you resorted to 'comfort food' or excessive eating due to sadness or feelings of discontentment?** (Choose only one option)

	1	2	3	4	5	6	7	
Never	☐	☐	☐	☐	☐	☐	☐	Every day

30. **Have you ever felt guilty about eating/food?** (Choose only one option)

	1	2	3	4	5	6	7	
Never	☐	☐	☐	☐	☐	☐	☐	Every day

31. **Have you felt that strict diets (or other food-related rituals) are necessary for controlling the amount of food that you eat?** (Choose only one option)

	1	2	3	4	5	6	7	
Never	☐	☐	☐	☐	☐	☐	☐	Every day

32. **Have you felt that you are too fat?** (Choose only one option)

	1	2	3	4	5	6	7	
Never	☐	☐	☐	☐	☐	☐	☐	Every day

Smoking habits

33. **Have you ever smoked daily?** ..... ☐ Yes ☐ No

If you have **never smoked daily**, please skip to question 38.

34. **Are you currently a daily smoker?** ..... ☐ Yes ☐ No

35. **If you are no longer a daily smoker, at what age did you quit?** ..... Age ☐☐

36. **In total, for how many years have you smoked daily?** ..... Years ☐☐

37. **Considering all the years in which you smoked regularly (daily), how many cigarettes/rolling tobacco did you smoke per day, on average?** ..... ☐☐

38. **Do you live with someone who smokes?** ..... ☐ Yes ☐ No

Chronic pain

39. **Are you experiencing pain that has lasted three months or longer?** ..... ☐ Yes ☐ No

40. **If yes, please indicate the intensity of your pain in the past week:** (Choose only one option)

No pain										Most severe pain
0	1	2	3	4	5	6	7	8	9	10
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

41. **Please indicate where your pain is most severe:** (Choose only one option)

☐ Neck ☐ Lower back ☐ Other

Diet

We would like to know more about your **usual** diet. For each of the following foods and beverages, please indicate **how often (the number of times)** you have consumed the food item in question **on average in the past year**, and the amount you usually eat/drink each time.

BEVERAGES

42. **How many glasses of milk do you normally drink?** (Choose only one option for each variety)

	Never/ rarely	1–4 per week	5–6 per week	1 per day	2–3 per day	4+ per day
Whole/full fat milk ("*Hel*") *(regular, sour/fermented)*	☐	☐	☐	☐	☐	☐
Semi-skimmed milk ("*Lett*") *(regular/sour/fermented)*	☐	☐	☐	☐	☐	☐
Low fat milk ("*Extra lett*")	☐	☐	☐	☐	☐	☐
Skimmed *(regular, sour/fermented)*	☐	☐	☐	☐	☐	☐

43. **How many cups of coffee/tea do you normally drink?** Choose only one option for each variety

	Never/ rarely	1–6 per week	1 per day	2–3 per day	4–5 per day	6–7 per day	8+ per day
Unfiltered or plunger/ steeped coffee	☐	☐	☐	☐	☐	☐	☐
Filtered coffee	☐	☐	☐	☐	☐	☐	☐
Espresso	☐	☐	☐	☐	☐	☐	☐
Latte	☐	☐	☐	☐	☐	☐	☐
Instant coffee	☐	☐	☐	☐	☐	☐	☐
Black tea	☐	☐	☐	☐	☐	☐	☐
Green tea	☐	☐	☐	☐	☐	☐	☐

44. **Do you take any of the following in your coffee?**

Sugar *(not including artificial sweeteners)* ..... ☐ Yes ☐ No

Milk or cream ..... ☐ Yes ☐ No

45. **Do you take any of the following in your tea?**

Sugar *(not including artificial sweeteners)* ..... ☐ Yes ☐ No

Milk or cream ..... ☐ Yes ☐ No

46. How many glasses of water do you drink on average?
(Choose only one option for each line)

	Never/ rarely	1–6 per week	1 per day	2–3 per day	4–5 per day	6–7 per day	8+ per day
Tap water	☐	☐	☐	☐	☐	☐	☐
Bottled water	☐	☐	☐	☐	☐	☐	☐

47. How many glasses of juice, squash/lemonade, and carbonated/soft drinks do you drink on a typical day?

(Choose only one option for each line)	Never/ rarely	1–3 per week	4–6 per week	1 per day	2–3 per day	4+ per day
Orange juice	☐	☐	☐	☐	☐	☐
Other juice	☐	☐	☐	☐	☐	☐
Squash/lemonade/soft drink containing sugar	☐	☐	☐	☐	☐	☐
Squash/lemonade/soft drink without sugar	☐	☐	☐	☐	☐	☐

YOGHURT/CEREAL

48. How often do you eat yoghurt (1 tub)? (Choose only one option)

☐ Never/rarely ☐ 1-3 per week
☐ 4-6 per week ☐ 1 or more per day

49. How often do you eat (breakfast) cereal, oatmeal or muesli?
(Choose only one option)

☐ Never/rarely ☐ 1-3 per week
☐ 4-6 per week ☐ 1 or more per day

BREAD/SANDWICHES

50. How many slices of bread (or equivalent; bread rolls, buns, crispbread, rye bread) do you normally eat? (1/2 bread roll = 1 slice of bread) (Choose only one option for each variety listed)

	Never/ rarely	1–4 per week	5–7 per week	2–3 per day	4–5 per day	6+ per day
Whole grain bread	☐	☐	☐	☐	☐	☐
Semi-whole grain bread	☐	☐	☐	☐	☐	☐
White bread (baguette)	☐	☐	☐	☐	☐	☐
Crispbread, etc.	☐	☐	☐	☐	☐	☐

The following questions are in regards to various sandwich spreads/ fillings. For each of the following sandwich spreads, we would like to know how many slices of bread/crispbread you normally eat with these spreads/fillings. If you regularly eat the given sandwich spreads with items other than bread (i.e., waffles, breakfast cereal, porridge) please include such use when answering the questions.

51. Please indicate how many slices of bread/crispbread you normally eat with the following sandwich spreads?

(Choose only one option for each line)	Never/ rarely	1–3 per week	4–6 per week	1 per day	2–3 per day	4+ per day
Jam	☐	☐	☐	☐	☐	☐
Brown (charamelised) whey cheese *(full fat)*	☐	☐	☐	☐	☐	☐
Brown cheese *(reduced fat)*	☐	☐	☐	☐	☐	☐
Cheese *(full fat)*	☐	☐	☐	☐	☐	☐
Cheese *(reduced fat)*	☐	☐	☐	☐	☐	☐
Mayonnaise based salads (*prawn salad, italian salad, etc.*)	☐	☐	☐	☐	☐	☐
Liver pâté	☐	☐	☐	☐	☐	☐
Preserved meats, low fat *(boiled ham, etc.)*	☐	☐	☐	☐	☐	☐
Preserved meats, high fat *(salami, cured mutton, etc.)*	☐	☐	☐	☐	☐	☐

52. Please indicate how many slices of bread/crispbread you have eaten on average per week in the past year with: (Choose one option for each line)

	Never/ rarely	1 per week	2–3 per week	4–6 per week	7–9 per week	10+ per week
Mackerel in tomato sauce; smoked mackerel	☐	☐	☐	☐	☐	☐
Caviar	☐	☐	☐	☐	☐	☐
Herring/anchovies	☐	☐	☐	☐	☐	☐
Salmon *(gravlax/smoked)*	☐	☐	☐	☐	☐	☐
Other types of fish	☐	☐	☐	☐	☐	☐

53. If you use butter/margarine on your sandwich/bread, how thick a layer do you normally spread onto it? (A single portion packet weighs 12 grams) (Choose only one option)

☐ Extra thin layer *(3 grams)* ☐ Thin layer *(5 grams)*
☐ Thick layer *(8 grams)* ☐ Extra thick layer *(12 grams)*

54. What type of butter/margarine do you normally put on your bread? (You may choose several options)

☐ I do not use butter/margarine on bread
☐ Butter
☐ Hard margarine *(e.g. Melange)*
☐ Soft margarine *(e.g. Soft, Vita)*
☐ Butter and margarine blends *(e.g. Bremyk)*
☐ *Brelett* (*fat reduced butter and margarine blend*)
☐ Reduced fat margarine *(e.g. Soft light, Vita Lett)*
☐ Olive oil margarine *(e.g. Brelett oliven, Soft oliven)*

FRUITS AND VEGETABLES

55. How often do you eat fruit? (Choose only one option for each line)

	Never/ rarely	1–3 per month	1 per week	2–4 per week	5–6 per week	1 per day	2+ per day
Apple/pear	☐	☐	☐	☐	☐	☐	☐
Orange/citrus fruit	☐	☐	☐	☐	☐	☐	☐
Banana	☐	☐	☐	☐	☐	☐	☐
Other fruit	☐	☐	☐	☐	☐	☐	☐

56. How often do you eat potatoes? (Choose only one option for each line)

	1-4 times per month	2-4 times per week	5-6 times per week	Once daily	Twice daily
Boiled	☐	☐	☐	☐	☐
Mashed	☐	☐	☐	☐	☐
Pan-fried/fried	☐	☐	☐	☐	☐

57. How often do you eat the following types of vegetables?
(Choose only one option for each line)

	Never/ rarely	1–3 per month	1 per week	2 per week	3 per week	4–5 per week	6–7 per week
Carrot	☐	☐	☐	☐	☐	☐	☐
Cabbage	☐	☐	☐	☐	☐	☐	☐
Swede	☐	☐	☐	☐	☐	☐	☐
Broccoli/cauliflower	☐	☐	☐	☐	☐	☐	☐

	Never/ rarely	1–3 per month	1 per week	2 per week	3 per week	4–5 per week	6–7 per week
Mixed salad	☐	☐	☐	☐	☐	☐	☐
Tomato	☐	☐	☐	☐	☐	☐	☐
Mixed vegetables (*frozen*)	☐	☐	☐	☐	☐	☐	☐
Onion	☐	☐	☐	☐	☐	☐	☐
Beans	☐	☐	☐	☐	☐	☐	☐
Peas	☐	☐	☐	☐	☐	☐	☐
Other vegetables	☐	☐	☐	☐	☐	☐	☐

58. **For the following vegetables in your diet, please indicate how much you typically eat each time:** (Choose only one option for each vegetable type)

Carrot ☐ 1/2 a carrot ☐ 1 carrot ☐ 1 1/2 carrot ☐ 2+ carrots

Potato ☐ 1-2 potatoes ☐ 3-4 potatoes ☐ 5-6 potatoes ☐ 7+ potatoes

Cabbage ☐ 1/2 dl ☐ 1 dl ☐ 1 1/2 dl ☐ 2+ dl

Swede ☐ 1/2 dl ☐ 1 dl ☐ 1 1/2 dl ☐ 2+ dl

Broccoli/cauliflower ☐ 1–2 pieces (bouquets) ☐ 3–4 pieces ☐ 5 + pieces

Mixed salad ☐ 1 dl ☐ 2 dl ☐ 3 dl ☐ 4+ dl

Tomato ☐ 1/4 of a tomato ☐ 1/2 a tomato ☐ 1 tomato ☐ 2+ tomatoes

Mixed vegetables (frozen) ☐ 1/2 dl ☐ 1 dl ☐ 2 dl ☐ 3+ dl

Beans ☐ 1–2 tbsp ☐ 3–4 tbsp ☐ 5–6 tbsp ☐ 7+ tbsp

Peas ☐ 1–2 tbsp ☐ 3–4 tbsp ☐ 5–6 tbsp ☐ 7+ tbsp

RICE, PASTA, PORRIDGE AND SOUP

59. **How often do you eat rice and pasta (spaghetti, macaroni)?** Choose only one option for each food)

	Never/ rarely	1-3 times per month	Once a week	Twice a week	3+ times per week
Rice	☐	☐	☐	☐	☐
Pasta (*spaghetti, macaroni, noodles*)	☐	☐	☐	☐	☐

60. **How often do you eat porridge?** (Choose only one option for each porridge type)

	Never/ rarely	Once a month	2-3 times per month	Once a week	2-6 times per week	1+ per day
Rice porridge	☐	☐	☐	☐	☐	☐
Other porridge *(oatmeal, etc.)*	☐	☐	☐	☐	☐	☐

61. **How often do you eat soup?** (Choose only one option per line)

	Never/ rarely	1-3 times per month	Once a week	Twice a week	3+ times per week
As a main course	☐	☐	☐	☐	☐
As appetizer, lunch or supper	☐	☐	☐	☐	☐

FISH

62. **We would like to know how often you eat fish, and kindly ask you to indicate your fish consumption below, as accurately as possible.** The availability of fish products may be seasonal; please indicate at which season you eat the various types of fish listed.

	Never/ rarely	Same amount all year	Winter	Spring	Summer	Autumn
Cod, saithe/coalfish, haddock, pollack	☐	☐	☐	☐	☐	☐
(Atlantic) wolf fish, flounder, redfish	☐	☐	☐	☐	☐	☐
Salmon, sea trout	☐	☐	☐	☐	☐	☐
Halibut	☐	☐	☐	☐	☐	☐
Mackerel	☐	☐	☐	☐	☐	☐
Herring	☐	☐	☐	☐	☐	☐
Freshwater fish (*perch, pike, grayling, charr, lavaret and trout)*	☐	☐	☐	☐	☐	☐
Other fish	☐	☐	☐	☐	☐	☐

63. **Considering the season(s) in which you eat fish, how often do you normally eat the following for dinner (main meal/course)?** (Choose only one option per line)

	Never/ rarely	Once a month	2–3 times per month	Once a week	2+ times per week
Boiled cod, saithe, pollack, haddock	☐	☐	☐	☐	☐
Pan-fried cod, saithe, pollack, haddocktorsk, sei, hyse, lyr	☐	☐	☐	☐	☐
Wolf fish, founder, redfish	☐	☐	☐	☐	☐
Salmon, sea trout	☐	☐	☐	☐	☐
Halibut	☐	☐	☐	☐	☐
Mackerel	☐	☐	☐	☐	☐
Herring	☐	☐	☐	☐	☐
Freshwater fish *(perch, pike, grayling, charr, lavaret, trout)*	☐	☐	☐	☐	☐
Other fish	☐	☐	☐	☐	☐

64. **If you eat fish, how much do you normally eat each time?** (1 piece/serving = 150 grams)

Boiled fish *(piece(s)/servings)* ☐ 1 ☐ 1 ½ ☐ 2 ☐ 3+

Pan-fried/oven-baked *(piece(s)/servings)* ☐ 1 ☐ 1 ½ ☐ 2 ☐ 3+

65. **How many times per year do you eat fish roe and fish liver?** (Choose only one option for each food)

	0	1–3	4–6	7–9	10+
Fish roe	☐	☐	☐	☐	☐
Fish liver	☐	☐	☐	☐	☐

66. **If you eat fish liver, how many tablespoons do you eat each time?** (Choose only one option)

☐ 1 ☐ 2 ☐ 3–4 ☐ 5–6 ☐ 7+

67. **How often do you eat the following fish products?** (Choose only one option per line)

	Never/ rarely	Once a month	2–3 times per month	Once a week	2+ times per week
Fishcakes/fish pudding/fish balls	☐	☐	☐	☐	☐
Fish stew/fish gratin	☐	☐	☐	☐	☐
Fried fish/fish fingers	☐	☐	☐	☐	☐
Other fish products/dishes	☐	☐	☐	☐	☐

68. **In which amounts do you normally eat the various following dishes?** (Choose only one option per line)

Fishcakes/fish pudding/fish balls *(pcs) (2 fish balls=1 fishcake)* ☐ 1 ☐ 2 ☐ 3 ☐ 4+

Fish stew, fish gratin *(dl)* ☐ 1–2 ☐ 3–4 ☐ 5+

Fried fish/fish fingers *(pcs)* ☐ 1–2 ☐ 3–4 ☐ 5–6 ☐ 7+

In addition to information regarding fish consumption, it is important to detail the sauces/fat that accompany fish meals.

69. How often do you eat the following as part of fish meals/ dishes?

	Never/ rarely	Once a month	2–3 times per month	Once a week	2+ times per week
Melted/solid butter	☐	☐	☐	☐	☐
Melted/solid margarine	☐	☐	☐	☐	☐
Sour cream, full fat *(35% fat)*	☐	☐	☐	☐	☐
Sour cream, reduced fat *(20% fat)*	☐	☐	☐	☐	☐
Sauce, high fat *(white/brown)*	☐	☐	☐	☐	☐
Sauce, fat free *(white/brown)*	☐	☐	☐	☐	☐

70. For the various types of fat/sauces that you regularly eat <u>with your fish</u>, please indicate how much you normally eat:

Melted/solid butter *(tbsp)*	☐ ½	☐ 1	☐ 2	☐ 3	☐ 4+
Melted/solid margarine *(tbsp)*	☐ ½	☐ 1	☐ 2	☐ 3	☐ 4+
Sour cream full fat *(tbsp)*	☐ ½	☐ 1	☐ 2	☐ 3	☐ 4+
Sour cream, red. fat *(tbsp)*	☐ ½	☐ 1	☐ 2	☐ 3	☐ 4+
Sauce, high fat *(dl)*	☐ ¼	☐ ½	☐ ¾	☐ 1	☐ 2+
Sauce, fat free *(dl)*	☐ ¼	☐ ½	☐ ¾	☐ 1	☐ 2+

71. How often do you eat shellfish? (i.e., prawns/shrimp, crabs, molluscs) (Choose only one option)

☐ Never/rarely ☐ Once a month
☐ 2-3 times per month ☐ Once a week or more

72. How many seagull eggs or eggs of other seabirds do you eat during the course of one year? (Choose only one option)

Never	1-3	4-6	7-9	10-15	16+
☐	☐	☐	☐	☐	☐

73. How often have you eaten freshwater fish? (perch, pike, grayling, charr, lavaret, trout) (Choose only one option per line)

	Never/ rarely	Once a month	2-3 times per month	Once a week	2-3 times per week	4+ per week
Childhood	☐	☐	☐	☐	☐	☐
Adolescence (13-19 yrs)	☐	☐	☐	☐	☐	☐
Adulthood (past year excl.)	☐	☐	☐	☐	☐	☐

MEATS

74. How often do you eat the following meat dishes? (Choose only one option for each meat type)

	Never/ rarely	1-2 times per month	3-4 times per month	2-3 times per week	4-6 times per week	7+ times per week
Reindeer meat	☐	☐	☐	☐	☐	☐
Moose/elk meat	☐	☐	☐	☐	☐	☐

75. How often do you eat the following meat and poultry dishes? (Choose only one option for each dish)

	Never/ rarely	Once a month	2–3 times per month	Once a week	2+ times per week
Roast *(beef, pork, mutton)*	☐	☐	☐	☐	☐
Cutlets *(beef, pork, mutton)*	☐	☐	☐	☐	☐
Steak *(beef, pork, mutton)*	☐	☐	☐	☐	☐
Hamburger/meat patties	☐	☐	☐	☐	☐
Sausages/hot dogs	☐	☐	☐	☐	☐
Grouse, other game birds	☐	☐	☐	☐	☐
Meat casserole, stew	☐	☐	☐	☐	☐
Pizza with meat toppings	☐	☐	☐	☐	☐
Chicken, unskinned	☐	☐	☐	☐	☐
Chicken, skinned	☐	☐	☐	☐	☐
Bacon	☐	☐	☐	☐	☐
Other meat dishes	☐	☐	☐	☐	☐
Blood-based dishes *(lamb/sheep, cattle, reindeer, moose)*	☐	☐	☐	☐	☐

76. If any of the following dishes are in your diet, please indicate your typical serving sizes: (Choose only one option for each dish)

Roast *(slices)*	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5+
Cutlets *(pcs)*	☐ ½	☐ 1	☐ 1½	☐ 2+	
Hamburgers, meat patties *(pcs)*	☐ 1	☐ 2	☐ 3	☐ 4+	
Sausages *(pcs; 1=150g)*	☐ ½	☐ 1	☐ 1½	☐ 2+	
Casserole/stew *(dl)*	☐ 1–2	☐ 3	☐ 4	☐ 5+	
Pizza with meat toppings *(slices of 100 grams each)*	☐ 1	☐ 2	☐ 3	☐ 4+	

77. Which of the following sauces do you have with your meat and pasta dishes? (Choose only one option for each sauce variety)

	Never/ rarely	Once a month	2–3 times per month	Once a week	2+ times per week
Brown sauce	☐	☐	☐	☐	☐
Gravy	☐	☐	☐	☐	☐
Tomato-based sauce	☐	☐	☐	☐	☐
Sauce with cream/sour cream	☐	☐	☐	☐	☐

78. For the various sauces listed, what amounts do you normally apply to your meals?

Brown sauce *(dl)*	☐ ¼	☐ ½	☐ ¾	☐ 1	☐ 2+
Gravy *(dl)*	☐ ¼	☐ ½	☐ ¾	☐ 1	☐ 2+
Tomato-based sauce *(dl)*	☐ ¼	☐ ½	☐ ¾	☐ 1	☐ 2+
Sauce with cream/sour cream *(dl)*	☐ ¼	☐ ½	☐ ¾	☐ 1	☐ 2+

OTHER FOODS

79. How many eggs do you normally eat in the course of <u>one week</u>? (pan-fried, boiled, scrambled, omelette) (Choose only one option)

☐ 0 ☐ 1 ☐ 2 ☐ 3–4 ☐ 5–7 ☐ 8-14 ☐ 15+

80. How often do you eat ice cream? (for dessert, Cornetto, etc.) (Choose one option for your ice cream consumption in summer, and one for the remainder of the calendar year)

	Never/ rarely	Once a month	2–3 times per month	Once a week	2+ times per week
In the summer	☐	☐	☐	☐	☐
The rest of the year	☐	☐	☐	☐	☐

81. How much ice cream do you normally eat each time? (Choose only one option)

☐ 1 dl ☐ 2 dl ☐ 3 dl ☐ 4+ dl

82. How often do you eat bakery goods, such as buns, cakes, danishes/pastries and cookies? (Choose only one option for each line)

	Never/ rarely	1–3 per month	1 per week	2–3 per week	4–6 per week	1+ per day
Yeasted bakery goods *(buns, etc.)*	☐	☐	☐	☐	☐	☐
Danish pastries	☐	☐	☐	☐	☐	☐
Cakes	☐	☐	☐	☐	☐	☐

	Never/ rarely	1–3 per month	1 per week	2–3 per week	4–6 per week	1+ per day
Pancakes	☐	☐	☐	☐	☐	☐
Waffles	☐	☐	☐	☐	☐	☐
Cookies, biscuits	☐	☐	☐	☐	☐	☐
Lefse, potato pancake	☐	☐	☐	☐	☐	☐

83. **How often do you have dessert?** (Choose only one option for each food)

	Never/ rarely	1 per month	2–3 per month	1 per week	2–3 per week	4+ per week
Pudding *(eg. chocolate/ caramel pudding)*	☐	☐	☐	☐	☐	☐
Creamed rice, mousse/ fromage	☐	☐	☐	☐	☐	☐
Compote, stewed fruit, canned fruit	☐	☐	☐	☐	☐	☐
Strawberries *(fresh, frozen)*	☐	☐	☐	☐	☐	☐
Other berries *(fresh, frozen)*	☐	☐	☐	☐	☐	☐

84. **How often do you eat chocolate?** (Sett ett kryss pr. linje)

	Never/ rarely	1–3 times per month	Once a week	2–3 times per week	4–6 times per week	Once a day or more
Dark chocolate	☐	☐	☐	☐	☐	☐
Milk chocolate	☐	☐	☐	☐	☐	☐

85. **If you eat chocolate, how much do you normally eat each time?** Imagine the size of a Kvikk Lunsj chocolate bar (47g), and indicate your serving size according to that.

☐ ¼ ☐ ½ ☐ ¾ ☐ 1 ☐ 1½ ☐ 2+

86. **How often do you eat other sweets/candy?** (Choose only one option)

Never/ rarely	1–3 times per month	Once a week	2–3 times per week	4–6 times per week	Once a day or more
☐	☐	☐	☐	☐	☐

87. **How often do you eat salty snacks?** (Choose only one option for each line)

	Never/ rarely	1–3 times per month	Once a week	2–3 times per week	4–6 times per week	Once a day or more
Potato crisps	☐	☐	☐	☐	☐	☐
Peanuts	☐	☐	☐	☐	☐	☐
Other nuts	☐	☐	☐	☐	☐	☐
Other snacks	☐	☐	☐	☐	☐	☐

COD LIVER OIL AND FISH OIL CAPSULES

88. **Do you take bottled cod liver oil supplements?** ☐ Yes ☐ No

89. **If yes, how often do you take bottled cod liver oil?** (Choose only one option per line)

	Never/ rarely	1–3 times per month	Once a week	2–6 times per week	Daily
In the winter	☐	☐	☐	☐	☐
Other seasons	☐	☐	☐	☐	☐

90. **If you take bottled cod liver oil, what amounts do you take each time?**

☐1 teaspoon ☐½ tablespoon ☐1+ tablespoons

91. **Do you take cod liver oil capsules/fish oil capsules?**

☐ Yes ☐ No

92. **If yes, how often do you take cod liver oil capsules/fish oil capsules?** (Choose only one option for each line)

	Never/ rarely	1–3 times per month	Once a week	2–6 times per week	Daily
In the winter	☐	☐	☐	☐	☐
Other seasons	☐	☐	☐	☐	☐

93. **What brand/type of cod liver oil/fish oil capsules do you take, and how many capsules do you take each time?**

Product/brand name:_____

Number of capsules: ☐ 1 ☐ 2 ☐ 3+

Other dietary supplements

94. **Do you take other dietary supplements?** (vitamins/minerals) ☐ Yes ☐ No

Alcohol

95. **Do you practice total alcohol abstinence?** ☐ Yes ☐ No

96. **If no, how often and how much did you drink, on average, in the past year?** (Choose only one option for each line)

	Never/ rarely	1 per month	2-3 per month	1 per week	2-4 per week	5-6 per week	1 per day	2+ per day
Beer/alcopops *(½ l)*	☐	☐	☐	☐	☐	☐	☐	☐
Wine *(glass)*	☐	☐	☐	☐	☐	☐	☐	☐
Liquor/distilled spirits *(drink/shot)*	☐	☐	☐	☐	☐	☐	☐	☐
Liqueur/fortified wine *(glass)*	☐	☐	☐	☐	☐	☐	☐	☐

Dental health

97. **In your most recent visit to the dentist, did you see a dentist/ dental hygienist in private practice or a dentist/dental hygienist employed in the public dental health service?** (Mark with an "X")

☐ Dentist in private practice
☐ Dental specialist in private practice
☐ Dental hygienist in private practice
☐ Dentist employed in public dental health service
☐ Dental specialist employed in public dental health service
☐ Dental hygienist employed in public dental health service
☐ Dentist abroad (outside of Norway)

98. **When did you last see a dentist or dental nurse?**(Choose only one option)

☐ Less than a year ago ☐ 1-2 years ago
☐ 3-5 years ago ☐ More than 5 years ago

99. **If your most recent dental appointment was more than two years ago, please supply the reason for not going more frequently to the dentist:** (Choose only one option)

☐ I have not been scheduled for a regular appointment
☐ Long waiting time for appointment
☐ I have not had the time
☐ Economic/financial reasons
☐ I have not required dental care
☐ I am afraid or anxious about seeing the dentist
☐ Other reasons: _____

100. **In the past 12 months, how much money have you spent on dental care (dentist, dental specialist, dental hygienist)?** (Choose only one option)

- ☐ Nothing (I have not had dental appointments)
- ☐ Nothing (I have had my costs covered)
- ☐ Less than NOK 1000
- ☐ NOK 1000-5000
- ☐ NOK 5001-10,000
- ☐ NOK 10,001-20,000
- ☐ More than NOK 20,000

101. **Please mark the two aspects that are most important to you in regards to your teeth/oral health:**

- That my teeth are nice-looking when I talk and smile ☐
- That my teeth are pain-free (do not hurt) ☐
- That I can chew/eat without any trouble ☐
- That I have fresh breath ☐
- That I keep my teeth for the rest of my life ☐

102. **How would you rate your dental health?** (Choose only one option)

☐ Poor ☐ Not so good ☐ Good ☐ Very good

103. **Do you have dentures/a dental bridge?** ☐ Yes ☐ No

Sunlight exposure/Tanning

104. **Have you been on holiday in southern countries or other beach/sunbathing holiday in the past month?** ☐ Yes ☐ No

105. **Please estimate the total number of hours during which you have been outside (during daylight hours) in the past seven days?** ☐☐ hours

106. **Have you used a solarium in the past month?**

☐ No ☐ 1 - 2 times ☐ 3+ times

Skin care products/Cosmetics

107. **How often (number of times) do you use the following cosmetic products?** (Choose only one option per product)

	Never/ rarely	1–3 per month	1 per week	2–4 per week	5–6 per week	1 per day	2+ per day
Face cream	☐	☐	☐	☐	☐	☐	☐
Hand cream	☐	☐	☐	☐	☐	☐	☐
Body lotion	☐	☐	☐	☐	☐	☐	☐
Perfume/aftershave	☐	☐	☐	☐	☐	☐	☐
Deodorant	☐	☐	☐	☐	☐	☐	☐
Hair products *(not incl. shampoo/conditioner)*	☐	☐	☐	☐	☐	☐	☐

Children and breastfeeding

108. **This question applies to mothers only: What is the birth year of your child(ren), and what was the approximate number of months during which the child(ren) was/were breastfed?**

	Birth year	Number of months during which the child was breastfed	Not breastfed
Firstborn	☐☐☐☐	☐☐	☐
Second child	☐☐☐☐	☐☐	☐
Third child	☐☐☐☐	☐☐	☐
Fourth child	☐☐☐☐	☐☐	☐
Fifth child	☐☐☐☐	☐☐	☐

If you had more than five children, please continue on a separate sheet.

Family and linguistic background

109. **How would you describe your family's financial situation when you were growing up?** (Choose only one option)

☐ Very good ☐ Good ☐ Challenging ☐ Extremely challenging

People of different ethnic backgrounds live in Northern Norway. That is, they have different languages and cultures. Examples of ethnic backgrounds, or ethnic groups, are Norwegian, Sami and Kven.

110. **What language(s) do/did you, your parents and your grandparents speak at home?** (Put one or more crosses for each line)

	Norwegian	Sami	Kven	Other, describe:
Mother's father	☐	☐	☐	☐ .....
Mother's mother	☐	☐	☐	☐ .....
Father's father	☐	☐	☐	☐ .....
Father's mother	☐	☐	☐	☐ .....
Father	☐	☐	☐	☐ .....
Mother	☐	☐	☐	☐ .....
Myself	☐	☐	☐	☐ .....

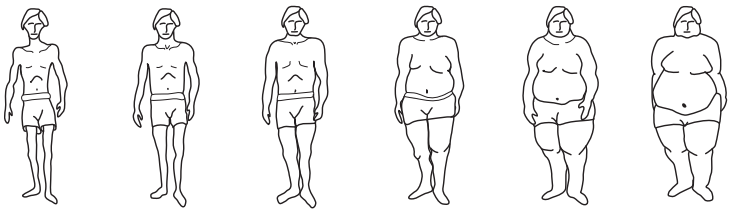
111. **What is your, your father's and your mother's ethnic backgrounds?** (Put one or more crosses for each line)

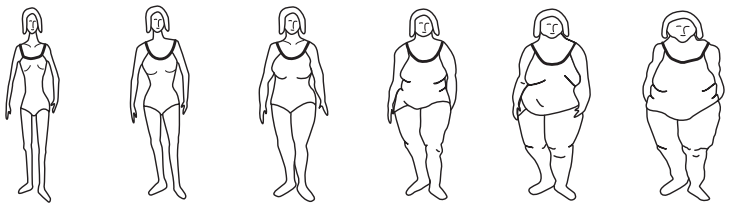
	Norwegian	Sami	Kven	Other, describe:
My ethnic background is	☐	☐	☐	☐ .....
My father's ethnic background is	☐	☐	☐	☐ .....
My mother's ethnic background is	☐	☐	☐	☐ .....

112. **What do you consider yourself to be?** (Put one or more crosses)

Norwegian	Sami	Kven	Other, describe:
☐	☐	☐	☐ .....

Body type/size





113. **Which of the above figures/illustrations most closely resembles your own body type/size?** ..... Figure number ☐

	Male figure number	Female figure number
114. **In your opinion, which figure corresponds to a healthy body type/size ?**	☐	☐
115. **Which figure is the first (in ascending numerical order) that you think of as representing a fat person?**	☐	☐
116. **Which figure/illustration is the first (in descending numerical order) that you think of as representing a skinny person?**	☐	☐

117. **How would you describe yourself?** (Choose only one response)

Extremely fat	Too fat	Average/Just right	Too thin/skinny	Extremely skinny
☐	☐	☐	☐	☐

118. **Have you attempted to lose weight (diet) in the past six months?** ..... ☐ Yes ☐ No

119. **If yes, how many kilograms have you lost in the past six months ?** ..... ☐☐ Kg

120. **Please indicate the methods used in order to lose weight?**
(You may choose one or more alternatives)

- ☐ Eating less
- ☐ Healthier diet
- ☐ Other dietary changes
- ☐ Exercise
- ☐ Weightloss drugs prescribed by doctor/physician
- ☐ Weightloss shakes/ powders
- ☐ Other, please describe: .....

Other health issues

121. **Below you will find a number of common health issues. Please consider each one carefully and individually, and then indicate the extent to which each individual health issue has affected you in the past four weeks.** (Choose only one option for each health issue)

	Not affected	Slightly affected	Affected quite a lot	Severely affected
Nervousness or shakiness inside	☐	☐	☐	☐
Feeling fearful	☐	☐	☐	☐
Feeling hopeless about the future	☐	☐	☐	☐
Worrying too much about things	☐	☐	☐	☐
Feeling blue/melancholic	☐	☐	☐	☐

Sleep/Sleeping habits

We would like to ask some questions concerning your sleeping habits. Please use the 24-hour time format, in which 11:00 corresponds to eleven o'clock in the morning and 23:00 corresponds to eleven o'clock at night.

122. **Have you taken part in shift work (worked night/evening shifts) in the past three months ?** ..... ☐ Yes ☐ No

123. **Please indicate the number of days a week in which you do not have the opportunity to choose freely when to go to sleep and when to get out of bed?** (This may apply, for instance, to any days in which you have to go to work, attend school, etc.) (Choose only one option)

0	1	2	3	4	5	6	7
☐	☐	☐	☐	☐	☐	☐	☐

124. **On the days that I do not have the opportunity to choose freely when to go to sleep/get out of bed,**

	Hours	Minutes
I go to bed at	☐☐	☐☐
I get ready to fall asleep at	☐☐	☐☐
Number of minutes that it normally takes before I fall asleep (fully):		☐☐
I wake up at	☐☐	☐☐

I wake up due to/using:

- ☐ Alarm clock
- ☐ External circumstances (i.e., noise caused by family members or others)
- ☐ I wake up naturally

Number of minutes normally passing from I wake up till I get out of bed: ..... ☐☐

On such days, do you sleep in other hours of the day? (i.e., afternoon nap) ..... ☐ Yes ☐ No

	Hours	Minutes
When (what hour) does this normally occur?	☐☐	☐☐
Provide the number of minutes of daytime sleeping:		☐☐

125. **On days in which I can freely choose my rising/waking/ sleeping hours:**

	Hours	Minutes
I go to bed at	☐☐	☐☐
I get ready to sleep at	☐☐	☐☐
Number of minutes that it normally takes before I fall asleep (fully):		☐☐
I wake up at	☐☐	☐☐

I wake up due to/using:

- ☐ Alarm clock
- ☐ External circumstances (i.e., noise caused by family members or others)
- ☐ I wake up naturally

Number of minutes normally passing from I wake up till I get out of bed: ..... ☐☐

On such days, do you sleep in other hours of the day (i.e., afternoon nap) ..... ☐ Yes ☐ No

Thank you for participating in the survey!

LUNDBLAD MEDIA AS – SVANEGODKJENT TRYKKSAK – 241 762 | 0-122027 – NORSK

Survey on health and lifestyle

We kindly request that you fill in the form as thoroughly and accurately as possible, and bring it with you to your scheduled physical examination. The form will be optically scanned. Please use blue or black ink. Use capital letters. Do not use decimals; for example, "0.5" should be rounded off to "1".

1. **In what year were you born?** ..... Year ☐☐☐☐

2. **What is your gender?** ..... Female ☐ Male ☐

3. **What is your marital status?**
☐ Married ☐ Cohabiting ☐ Divorced
☐ Unmarried ☐ Widow/widower

4. **How many years of education have you completed?** (Include any and all years in which you attended school or studied) ..... Number of years ☐☐

5. **If you are a woman: How many children have you given birth to?** ..... Number of children ☐☐

6. **If you are a woman: How many children have you breastfed?** ..... Number of children ☐☐

Personal health

7. **How is you state of health?** (Put one cross only)
☐ Poor ☐ Good
☐ Not so good ☐ Very good

8. **How is your dental health?** (Put one cross only)
☐ Poor ☐ Good
☐ Not so good ☐ Very good

9. **Do you have dentures/ a dental bridge?** ..... ☐ Yes ☐ No

10. **When did you last see a dentist or dental nurse?**
☐ Less than a year ago ☐ 1–2 years ago
☐ 3–5 years ago ☐ More than 5 years ago

11. **How satisfied are you with the dental health care offered in your municipality?** (Put one cross only)
Very dissatisfied ☐ ☐ ☐ ☐ ☐ ☐ Very satisfied ☐ Don't know

Cardiovascular disease

12. **Do you have, or have you ever had, high blood pressure?** ..... ☐ Yes ☐ No

13. **If yes, how old were you when you developed high blood pressure?** ..... Age ☐☐

14. **Are you taking medication for high blood pressure?** Yes, currently ☐ Previously, but not now ☐ Never ☐

15. **If you are taking high blood pressure medication, or have taken high blood pressure medication in the past, at what age did you start taking this type of medicine?** ..... Age ☐☐

16. **Have you ever had one or more heart attacks?**
☐ No, never ☐ One heart attack ☐ Two heart attacks ☐ Three or more heart attacks

17. **If yes, at what age did you have your first heart attack?** ..... Age ☐☐

18. **Do you suffer from angina pectoris (heart cramp)?** ..... ☐ Yes ☐ No

19. **If yes, how often have you experienced such pain in the past month?** .....
Rarely ☐ Once a week ☐ 2-3 times a week ☐ 4-6 times a week ☐ 7 times a week or more ☐

20. **How old were you when you had your first attack of angina pectoris?** ..... Age ☐☐

21. **Have you had heart (bypass) surgery?** ..... ☐ Yes ☐ No

22. **Have you had your arteries unblocked/had stent(s) placed?** ..... ☐ Yes ☐ No

23. **Has your doctor told you that you have atrial fibrillation?** ..... ☐ Yes ☐ No

24. **How old were you when you first experienced atrial fibrillation?** ..... Age ☐☐

Diabetes

25. **Have you ever been diagnosed with diabetes (elevated blood sugar levels)?** ☐ Yes ☐ No

If no, please skip to question 35.

26. **If yes, please specify your diabetes diagnosis:** (chose one or more options)

Gestational diabetes ☐

Type 1 diabetes ☐

Type 2 diabetes ☐

27. **How was your diabetes discovered?**

I consulted my doctor/physician because of symptoms ☐ Yes ☐ No

It was discovered without the appearance of symptoms (medical certificate, work-related medical examination, pregnancy health examination, medical consultation for illness other than diabetes, etc.) ☐ Yes ☐ No

28. **At what age was your diabetes discovered/diagnosed?** Age ☐☐

INSULIN

29. **Are you taking insulin for your diabetes?**	Yes, currently	Previously, but not now	Never used
	☐	☐	☐

If you are taking (or have taken) insulin:

30. **At what age did you start your insulin treatment?** Age ☐☐

31. **How many times per day do you/ did you usually take insulin?** ☐☐ times

32. **In total, how many units of insulin do you/did you take on an average day?** ☐☐ units (E)

ORAL MEDICATION

33. **Are you taking oral medication for diabetes?**	Yes, currently	Previously, but not now	Never used
	☐	☐	☐

If you are taking or have taken oral medication:

34. **At what age did you start taking oral medication for diabetes?** Age ☐☐

Other illnesses

35. **Do you have, or have you ever had, any of the following?**

	Yes	No	Age at onset
Asthma	☐	☐	☐☐
Eczema	☐	☐	☐☐
Chronic bronchitis, emphysema, COPD	☐	☐	☐☐
Multiple sclerosis (MS)	☐	☐	☐☐
Psoriasis	☐	☐	☐☐
Bechterew's disease	☐	☐	☐☐

Chronic pain

36. **Are you experiencing pain that has lasted three months or longer?** ☐ Yes ☐ No

37. **If yes, please indicate the intensity of your pain in the past week:** (Choose only one option)

No pain										Most severe pain
0	1	2	3	4	5	6	7	8	9	10
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

38. **Please indicate where your pain is most severe:** (Choose only one option)

☐ Neck ☐ Lower back ☐ Other

Physical activity

39. **We will now ask you to state your physical activity at the ages of 14, 30 and at your current age, on a scale from very low to very high.** The scale below runs from 1 to 10. Physical activity includes both housework and activity at work, as well as exercise and other physical activities such as walking/hiking, etc. Mark the number that best matches your level of activity:

	Very low									Very high
Age	1	2	3	4	5	6	7	8	9	10
14 years	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
30 years	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Current age	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Alcohol

40. **Do you practice total alcohol abstinence?** ☐ Yes ☐ No

41. **If no, how often and how much did you drink, on average, in the past year?** (Put one cross per line)

	Never/ rarely	1 per month	2–3 per month	1 per week	2–4 per week	5–6 per week	1 per day	2+ per day
Beer/alcopops *(½ l.)*	☐	☐	☐	☐	☐	☐	☐	☐
Wine *(glass)*	☐	☐	☐	☐	☐	☐	☐	☐
Liquor/distilled spirits *(drink/shot)*	☐	☐	☐	☐	☐	☐	☐	☐
Liqueur/fortified wine *(glass)*	☐	☐	☐	☐	☐	☐	☐	☐

Smoking habits

42. **Have you ever smoked daily?** ☐ Yes ☐ No

If you have never smoked daily, please skip to question 47.

43. **Are you currently a daily smoker?** ☐ Yes ☐ No

44. **If you are no longer a daily smoker, at which age did you quit?** Age ☐☐

45. **In total, for how many years have you smoked daily?** Years ☐☐

46. **Considering all the years in which you smoked regularly (daily), how many cigarettes/rolling tobacco did you smoke per day, on average?** Number of cigarettes ☐☐

47. **Do you live with someone who smokes?** ☐ Yes ☐ No

Language and use of interpreter

48. **In what language(s) do you primarily want to talk to health personnel?** (Put one or more crosses)

Norwegian	Sami	Other, describe:
☐	☐	☐ .....

49. **If you have answered "Sami" but were not offered a Sami-speaking doctor at your last doctors visit, did they offer you an interpreter?**

With your general practitioner:

☐ Yes ☐ No
☐ I do not want an interpreter ☐ Not relevant

In the hospital/with a specialist:

☐ Yes ☐ No
☐ I do not want an interpreter ☐ Not relevant

Family and linguistic background

50. **How would you describe your family's financial situation when you were growing up?** (Choose only one option)

☐ Very good ☐ Good ☐ Challenging ☐ Extremely challenging

People of different ethnic backgrounds live in Northern Norway. That is, they have different languages and cultures. Examples of ethnic backgrounds, or ethnic groups, are Norwegian, Sami and Kven.

51. **What language(s) do/did you, your parents and your grandparents speak at home?** (Put one or more crosses)

	Norwegian	Sami	Kven	Other, describe:
Mother's father	☐	☐	☐	☐ .....
Mother's mother	☐	☐	☐	☐ .....
Father's father	☐	☐	☐	☐ .....
Father's mother	☐	☐	☐	☐ .....
Father	☐	☐	☐	☐ .....
Mother	☐	☐	☐	☐ .....
Myself	☐	☐	☐	☐ .....

52. **What is your, your father's and your mother's ethnic backgrounds?** (Put one or more crosses)

	Norwegian	Sami	Kven	Other, describe:
My ethnic background is	☐	☐	☐	☐ .....
My father's ethnic background is	☐	☐	☐	☐ .....
My mother's ethnic background is	☐	☐	☐	☐ .....

53. **What do you consider yourself to be?** (Put one or more crosses)

Norwegian	Sami	Kven	Other, describe:
☐	☐	☐	☐ .....

Experience and use of health services

54. **Who is the doctor you normally use?**

☐ Your GP ☐ Another doctor

55. **How long have you had your current GP?**

☐ Less than 6 months ☐ 6 to 11 months

☐ 12 to 24 months ☐ More than two years

56. **In the last 12 months, have you contacted your doctor for help or advice for yourself?** ☐ Yes ☐ No

If yes, did you get the help you asked for?

☐ Never ☐ Sometimes ☐ Usually ☐ Always

57. **How satisfied are you with the following aspects of the doctor's service (regular GP scheme)?**
(Put one cross only)

	Very satisfied	Satisfied	Dissatisfied	Very dissatisfied	Don't know
The doctor's accessibility on the phone	☐	☐	☐	☐	☐
The waiting time for an appointment	☐	☐	☐	☐	☐
Time with the doctor	☐	☐	☐	☐	☐
The doctor's understanding of your problems	☐	☐	☐	☐	☐
Their information about your health issues, examination and treatment plan	☐	☐	☐	☐	☐
In total, how satisfied are you with the municipal health service?	☐	☐	☐	☐	☐

The next questions are about the specialized health service.

Specialized health service refers to hospitals, district psychiatric centers (DPS), specialized doctors services and individual specialists.

58. **In the last 12 months, have you been for examination or treatment for *physical problems* to the following?**

☐ The hospital ☐ Specialist medical center

☐ Private specialist ☐ None of these

59. **In the last 12 months, have you been for examination or treatment for *psychological problems* to the following?**

☐ Psychiatric hospital ☐ District psychiatric center

☐ Private specialist ☐ None of these

60. **If you have been for treatment with a specialist for physical or psychological problems in the last 12 months, answer the following questions:**
(Put one cross only) Answer on a scale from 0 to 10, where 0 = to a small extent, 10 = to a large extent.

Did you get a chance to say what you felt was important about your condition?

	0	1	2	3	4	5	6	7	8	9	10	Not relevant
Physical issues	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Psychological issues	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Did the doctors speak to you in a way you understood?

	0	1	2	3	4	5	6	7	8	9	10	Not relevant
Physical issues	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Psychological issues	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

All in all, do you trust the hospital or specialist who saw you?

	0	1	2	3	4	5	6	7	8	9	10	Not relevant
Physical issues	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Psychological issues	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

All in all, how satisfied are you with the care and treatment you eventually received?

	0	1	2	3	4	5	6	7	8	9	10	Not relevant
Physical issues	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Psychological issues	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Thank you for participating in the survey!