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The prevalence and incidence of diabetes mellitus among Sami and non-Sami inhabitants of Northern Norway

The SAMINOR Study

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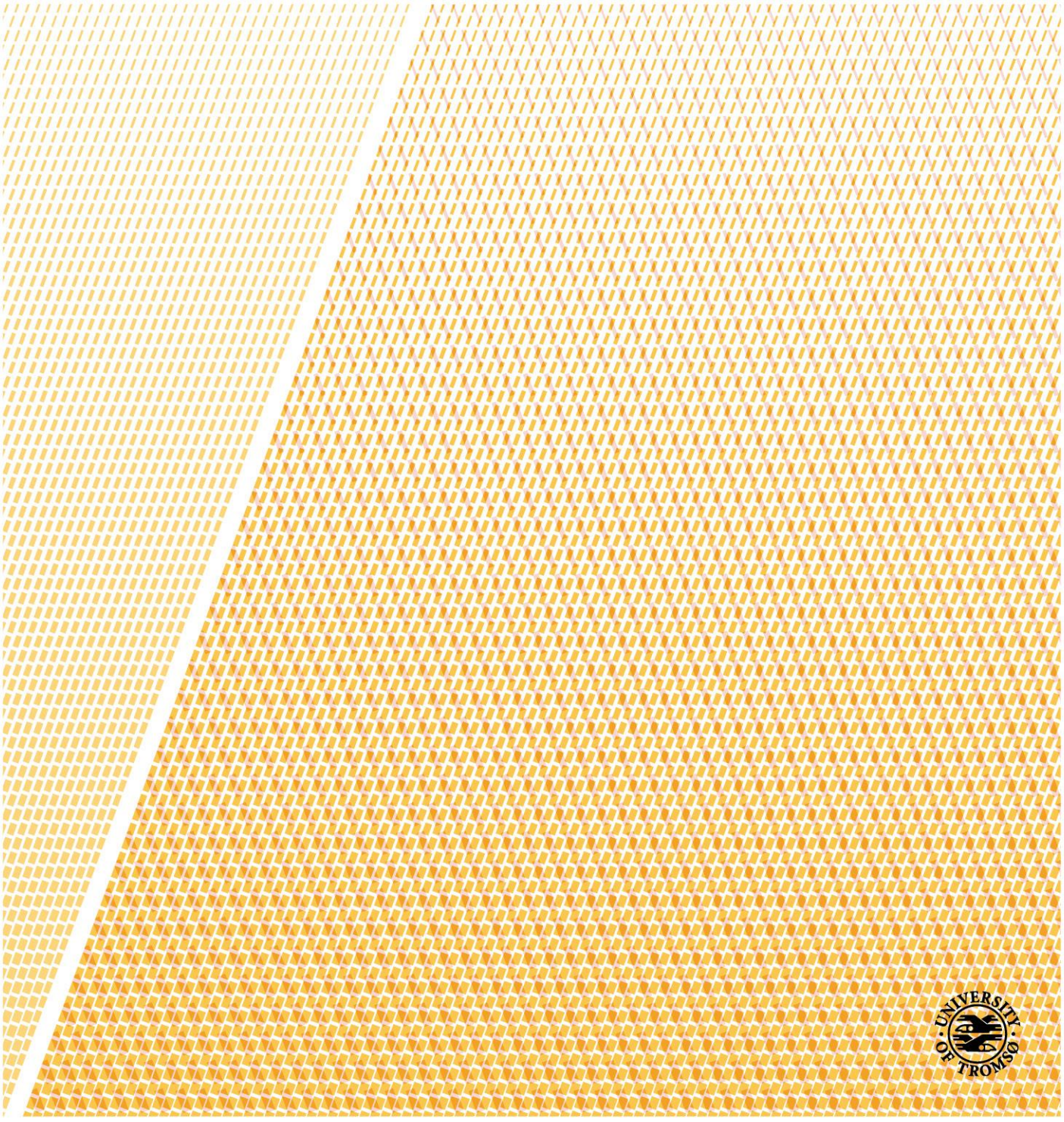

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Summary

Several studies have reported poorer health outcomes especially lifestyle related diseases (e.g. cardiovascular diseases and type 2 diabetes mellitus [T2DM]) among indigenous peoples throughout the world. Rapid industrialisation of the societies with a more sedentary lifestyle and increased calorie intake, which have taken place to varying degrees among both indigenous and benchmark populations have been implicated in this regard. As well as the lifestyle related changes, the indigenous Sami people in Norway, like many other indigenous peoples throughout the world, experienced centuries of stigmatisation and assimilation policies. Both the lifestyle changes and experienced assimilation policies might give rise to increased vulnerability to somatic and psychological disorders.

The present thesis aims to measure the prevalence and incidence of diabetes mellitus (DM) among Sami and non-Sami inhabitants of Northern Norway in order to explore ethnic difference and to elucidate any explanatory factor, which can account for the possible disparities.

Paper 1 was based on data from a cross-sectional population-based survey, the SAMINOR 1 Survey (2003–2004). A total of 27,151 individuals aged 36–79 years were invited and 15,208 were included in the analysis. Self-report (questionnaire) and/or non-fasting/random plasma glucose (RPG) ≥11.1 mmol/L were used to define DM and 7.8 mmol/L ≤ RPG <11.1 mmol/L was used to define pre-diabetes. Age-standardised prevalence of pre-diabetes and DM among Sami men was respectively 3.4% and 5.5%. Corresponding values for non-Sami men were 3.3% and 4.6%. Age-standardised prevalence of pre-diabetes and DM for Sami women was 2.7% and 4.8%, respectively, while corresponding values for non-Sami women were 2.3% and 4.5%. However, no statistical significant ethnic difference was observed in the overall age-adjusted prevalence of pre-diabetes and DM. Nevertheless, the prevalence of DM was

higher among Sami in southern regions and lower in northern regions compared with their non-Sami counterparts.

Paper 2 was based on data from another cross-sectional population-based survey, the SAMINOR 2 Clinical Survey (2012–2014). A total of 12,455 Sami and non-Sami inhabitants aged 40–79 years were invited to participate and 5878 were included in the analyses. Self-reported T2DM and/or HbA1c ≥ 6.5% were applied to define T2DM and 5.7% ≤ HbA1c <6.5% to define pre-diabetes. In men, the total age-standardised prevalence of pre-diabetes (37.9% vs 31.4%) and T2DM (10.8% vs 9.5%) were higher in Sami compared with non-Sami; the age-adjusted ethnic differences were statistically significant for both pre-diabetes (OR 1.42, 95% CI: 1.20–1.68) and T2DM (OR 1.31, 95% CI: 1.01–1.70). In women, pre-diabetes (36.4% vs 33.5%) and T2DM (8.6% vs 7.0%) were also more prevalent in Sami than non-Sami; the age-adjusted differences in both pre-diabetes (OR 1.20, 95% CI: 1.02–1.41) and T2DM (OR 1.38, 95% CI: 1.05–1.82) were also statistically significant. The observed ethnic difference in the waist-to-height ratio (WHtR) was a plausible explanation for the ethnic difference in the prevalence of pre-diabetes and T2DM.

The overall prevalence of pre-diabetes and DM was high among both Sami and non-Sami participants in both surveys. Although no ethnic difference was observed in the prevalence of pre-diabetes or DM in the SAMINOR 1 Survey (based on self-report and/or RPG ≥ 11.1 mmol/L), the prevalence values were higher among Sami participants relative to their non-Sami counterparts in the SAMINOR 2 Clinical Survey (based on self-report and/or HbA1c ≥6.5%). Higher obesity indices (BMI, WHtR) could be plausible explanatory factors for the observed differences.

Paper 3 was a longitudinal study, which followed participants in the SAMINOR 1 Survey to the SAMINOR 2 Clinical Survey. DM was defined based on self-report and/or HbA1c ≥6.5%.

The 8-year cumulative incidence of DM was calculated by dividing the number of incident DM cases by the number of DM-free participants in the SAMINOR 1 Survey. The 8-year cumulative incidence of DM was 6.1% (201 incident cases) with no statistically significant ethnic difference.

List of papers

1. **Naseribafrouei, Ali; Eliassen, Bent-Martin; Melhus, Marita; Broderstad, Ann Ragnhild.** *Ethnic difference in the prevalence of pre-diabetes and diabetes mellitus in regions with Sami and non-Sami populations in Norway – The SAMINOR1 study.* International Journal of Circumpolar Health 2016; Volume 75. ISSN 1239-9736.s DOI: 10.3402/ijch.v75.31697.
2. **Naseribafrouei, Ali; Eliassen, Bent-Martin; Melhus, Marita; Svartberg, Johan; Broderstad, Ann Ragnhild.** *Prevalence of pre-diabetes and type 2 diabetes mellitus among Sami and non-Sami men and women in Northern Norway - The SAMINOR 2 Clinical Survey.* International Journal of Circumpolar Health 2018; Volume 77 (1463786). ISSN 1239-9736.s DOI: 10.1080/22423982.2018.1463786.
3. **Naseribafrouei, Ali; Eliassen, Bent-Martin; Melhus, Marita; Svartberg, Johan; Broderstad, Ann Ragnhild.** The 8-year cumulative incidence of diabetes mellitus among Sami and non-Sami inhabitants of Northern Norway - The SAMINOR Study. (Submitted to BMC Endocrine Disorders)

Abbreviations

2hpp: 2 hour postprandial

ADA: American Diabetes Association

AI/AN: American Indian and Alaska Native

BMI: body mass index

CI: confidence interval

CM: centimeter

DM: diabetes mellitus

FPG: fasting plasma glucose

HbA1c: glycated haemoglobin

HDL cholesterol: high-density lipoprotein cholesterol

IFG: impaired fasting glucose

IGT: impaired glucose tolerance

LADA: Latent Autoimmune Diabetes of Adults

OGTT: oral glucose tolerance test

OR: Odds ratio

PPV: positive predictive value

RPG: random (non-fasting) plasma glucose

SCL-10: Hopkins symptom checklist, 10 items version

T1DM: type 1 diabetes mellitus

T2DM: type 2 diabetes mellitus

WC: waist circumference

WHO: World Health Organisation

WHtR: waist-to-height ratio

1 Introduction

Type 2 diabetes mellitus (T2DM) has evolved into an ever-increasing epidemic worldwide [1]. The disease is prevalent in both developed and developing countries, but the prevalence of the disease has been rising more rapidly in middle- and low-income countries [2]. T2DM is the major cause of blindness, renal failure, heart attacks, stroke and lower limb amputation in the world [2] and if no concerted efforts are made to address the risk factors, early diagnosis and treatment of the disease, the harmful microvascular and macrovascular complications of it will remain a major burden for decades to come [1]. Deficient action of insulin either due to inadequate insulin secretion or diminished tissue responses to insulin at one or more points in the complex pathway of hormone actions comprises the basis of T2DM [3]. Although genetic predisposition is a known risk factor for T2DM [4], many cases of T2DM can be prevented through lifestyle changes like increasing physical activity and restriction of calorie intake [2]. Adiposity is the most important risk factor for development of T2DM [5-7]. The protective effect of physical activity goes primarily through improved insulin sensitivity and glucose metabolism [8]. Although physical activity can play an important role in maintaining body weight and composition within normal ranges, a reduction in body weight is not necessary for the beneficial effect on glucose homeostasis [9].

A newly conducted Norwegian nationwide cohort study linked data from national registries with prospectively collected data on DM medication and diagnoses for all Norwegian residents aged 30 to 89 years (>3.2 million people) [10]. According to this study, while the prevalence of T2DM increased from 4.9% in 2009 to 6.1% in 2014, during the same period, the incidence of the disease decreased significantly from 609 cases per 100,000 person-years to 398 cases per 100,000 (an annual reduction of 10.1%). This decline was seen for both pharmacologically and non-pharmacologically treated T2DM and was present in all sex, age,

education level and place of birth subgroups [10]. The authors concluded that the observed rise in the prevalence of T2DM despite decreasing incidence of the disease might be due to diagnosis at a younger age and increased longevity. In 2004, the prevalence of known cases of any type of DM in the age group ≥30 years in all Norway was estimated to 3.4% [11]. Nystad reported the prevalence of known cases of any type of DM in selected municipalities of Northern and Mid-Norway to be 4.0% in men and 4.1% in women in 2003–2004 [12]. At the same time, the prevalence of metabolic syndrome was quite high yet not significantly different between the Sami and non-Sami inhabitants of Northern and Mid-Norway [13]. In the period 1959–1975, mortality due to ischemic heart disease in Norway was highest in Finnmark county [14]. This prompted several cardiovascular surveys in this county. As cardiovascular disease and DM are risk factors for each other and share many risk factors [15], it can be expected that if the prevalence of cardiovascular disease is high in a region, the prevalence of DM might be high as well.

The Sami people is an indigenous population who traditionally inhabited northern parts of Norway, Sweden, Finland and Kola Peninsula in Russia. They, in combination with other ethnic groups, comprise the heterogeneous population of Northern Norway with a large and longstanding interaction between the ethnic groups. The Sami people have experienced colonialism and have been victims of a state- and church-driven assimilation policy [16]. The pervasive assimilation policy brought about loss or extensive changes in traditional practices, languages, norms, and believes of the Sami people [17]. These changes in tandem with lifestyle changes due to rapid modernisation and industrialisation ensuing the Second World War, which affected all ethnic groups in the region, made the Sami people vulnerable and prone to lifestyle-related and chronic diseases like T2DM. Several studies have reported similar lifestyle trends with resultant higher incidence and prevalence of related diseases among other indigenous peoples throughout the world [18-26].

The scarcity of knowledge about health and living conditions of the Sami people in Norway prompted the Centre for Sami Health Research to conduct the SAMINOR Study (the SAMINOR 1 Survey in 2003–2004 and the SAMINOR 2 Survey in 2012–2014). The study provided invaluable insight into various social, psychological, and somatic aspects of health and living of the inhabitants in the included municipalities. The main aim of the present thesis was to promote more knowledge about the incidence and prevalence of DM among Sami and non-Sami inhabitants of the included municipalities, some risk factors for T2DM and any ethnic disparities in this regard.

1.1 Background

1.1.1 Diabetes Mellitus

Diabetes mellitus (DM) is a chronic progressive disease resulting from either insufficient insulin secretion or impairment in insulin action [2]. Incident cases of Type 1 diabetes mellitus (T1DM) are seen mainly in children and adolescents, but it can occur virtually at any age [3]. T2DM occurs predominantly in adults, but it affects increasingly adolescents and young adults [27].

1.1.2 Pathophysiology of diabetes mellitus

T1DM, which accounts for approximately 5–10% of DM cases [3], arises due to destruction of β-cells of the pancreas predominantly through an autoimmune process in over 95% of cases (type 1A) or idiopathic in less than 5% of cases (type 1B) [15]. If T1DM is left untreated it usually manifests itself as ketoacidosis [15]. The disease is a catabolic disorder with virtually absent circulating insulin, elevated plasma glucagon, and lack of pancreatic β-cells response to all insulinogenic stimuli, necessitating use of exogenous insulin [15]. In immune-mediated T1DM, approximately one-third of the disease susceptibility is gene-mediated and two-thirds is due to environmental factors [15]. In a mild form of autoimmune-mediated T1DM, patients initially retain enough β-cells function to avoid ketosis, but as the disease progresses later in life, they also become dependent on exogenous insulin. It is been reported that in Northern European countries, up to 15% of T2DM cases may actually have this mild form of T1DM (latent autoimmune diabetes of adults; LADA) [15]. The fact that the prevalence of T1DM is higher in Scandinavian countries and increases by migration to Northern Hemisphere supports the involvement of environmental factors in the development of T1DM [15].

T2DM represents a heterogeneous group of conditions, where circulating endogenous insulin is usually adequate to prevent ketoacidosis, but insufficient to prevent hyperglycaemia in the presence of increased needs due to tissue insensitivity (insulin resistance) [15]. Insulin resistance may occur in tissues like skeletal muscles, adipose tissue and liver [28]. This, in turn, leads to compensatory increased secretion of insulin to overcome insulin resistance [29]. At first, compensatory hyperinsulinemia maintains plasma glucose levels within the normal range, but eventually with the gradual decline in the insulin production by β-cells of the pancreas, the person enters overt diabetic phase [30, 31]. Nonetheless, most of the times, impairment of insulin secretion and insulin resistance coexist in the same patient and it is unclear which abnormality, if either alone, is the primary pathology [3]. Genetic and environmental factors interplay to develop both the insulin resistance and the β-cell loss (Figure 1) [15]. Several epidemiologic studies have indicated strong genetic associations, since in monozygotic twins over 40 years of age, there is a 70% one-year concordance in the development of T2DM [15]. Numerous genetic loci have so far been implicated in heightened risk of T2DM, most of them appear to encode proteins involved in β-cell development and function [15].

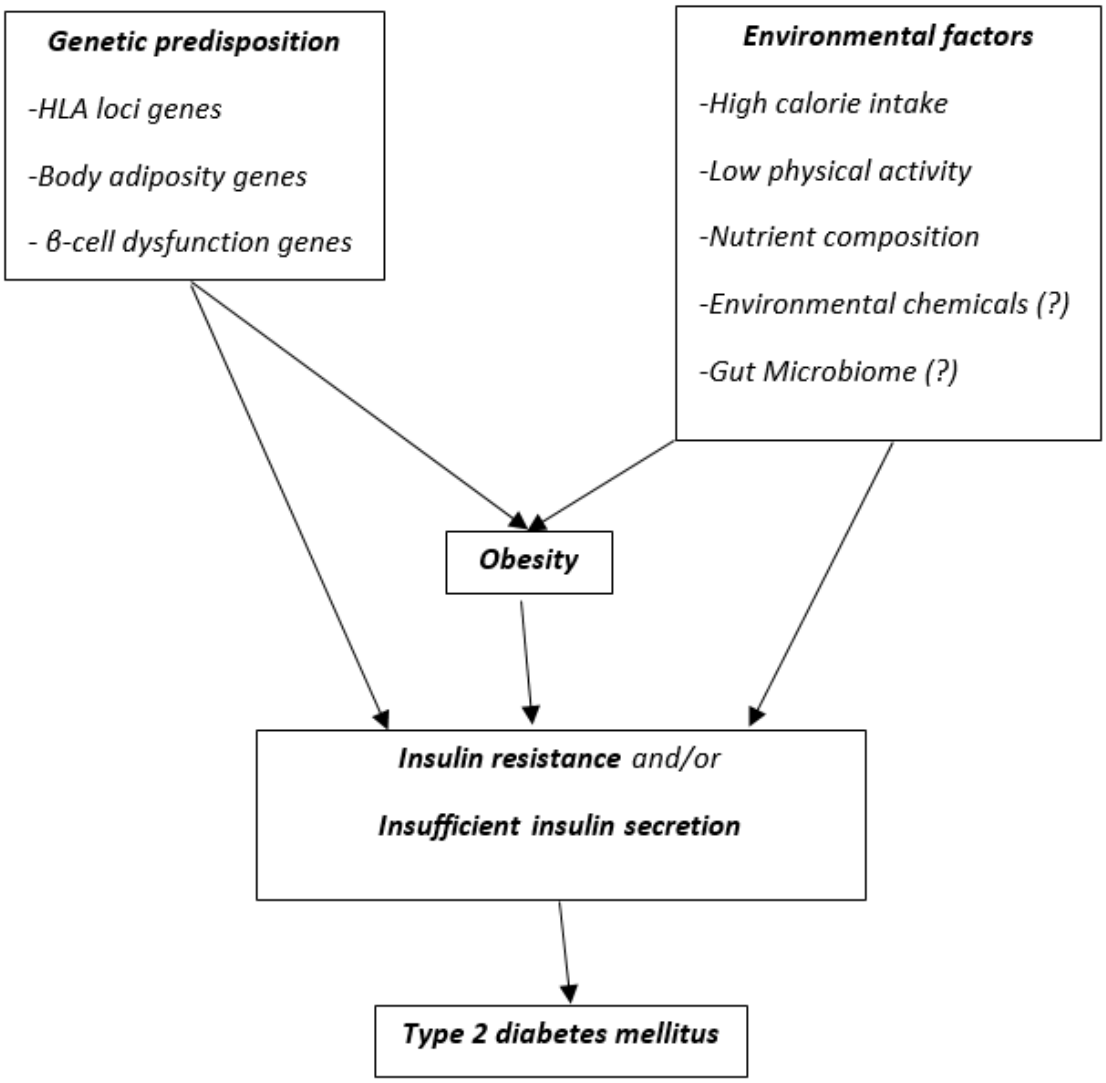


Figure 1. Pathophysiology of type 2 diabetes mellitus. Adapted from «Lancet 2014, 383(9922): p. 1068-83». [1].

1.1.3 Signs, symptoms, and late complications of type 2 diabetes mellitus

The majority of patients with T2DM (especially obese ones) have an insidious onset of hyperglycaemia and are asymptomatic initially [15]. Classic symptoms of T2DM include polyuria, polydipsia, unexplained weight loss, accompanied sometimes with polyphagia [3]. Complications of T2DM can be divided into microvascular and macrovascular complications. Microvascular complications include blurred vision due to retinopathy, numbness and tingling (paraesthesia) in the limbs (diabetic polyneuropathy), autonomic neuropathy and resultant gastrointestinal, genitourinary, and cardiovascular symptoms as well as sexual dysfunction [3, 4, 32]. Macrovascular complications of T2DM include coronary artery disease, stroke, arterial insufficiency (necrotic ulcers in the lower extremities leading sometimes to amputation), mesenteric ischemia, and diabetic nephropathy [4].

If the glycaemic state is poorly controlled, the patient may develop diabetic ketoacidosis or hyperglycaemic hyperosmolar coma [15].

By diagnosing patients in early phase of the disease, the development of the disease can, in most cases, either be prevented or delayed so that late complications are avoided to the greatest extent possible.

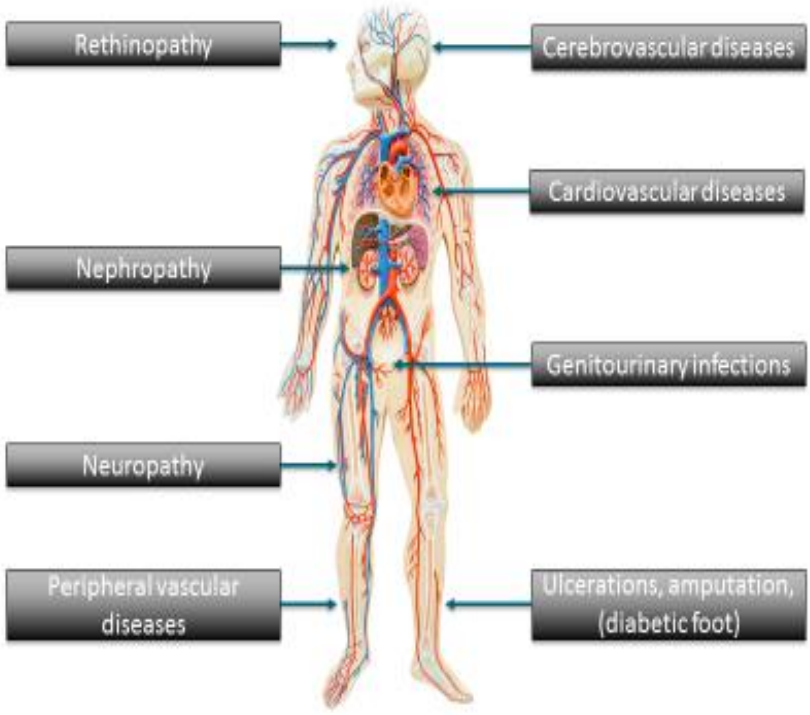


Figure 2. Late complications of diabetes mellitus. Source: Colourbox

1.1.4 Risk factors for type 2 diabetes mellitus

Several risk factors have been mentioned in the literature for development of T2DM. Although the exact mechanism of action of all these risk factors are not completely known, it is highly likely that factors like advanced age, overweight (BMI $\geq$ 25 kg/m^2) and obesity, low physical activity, and family history of T2DM have causal relationship to the development of T2DM. On the other hand, risk factors like hypertriglyceridemia, hypercholesterolemia, low HDL, hypertension, polycystic ovary syndrome, acanthosis nigricans, and history of cardiovascular disease might have only an association (non-causal relationship) with T2DM [15, 28, 33]. Male sex has been mentioned as a risk factor for undiagnosed diabetes, which might be a proxy for other unfortunate factors like abdominal obesity, smoking and lower willingness to seek medical care [34]. T2DM is more prevalent among African Americans, Latinos, Native Americans and some other ethnic groups, which might be due to a combined effect of genetic predisposition and environmental factors [4].

Discrimination has been reported to be associated with both obesity [35] and T2DM [36, 37]. There is a plethora of studies showing that early life events like child maltreatment, malnutrition, economic insecurity, low socioeconomic status, and even in-uterus exposure to gestational diabetes and maternal hyperglycaemia as contributors to the development of T2DM later in life [38-41]. On the other hand, it is been reported that breastfeeding was associated with reduced incidence of DM in mothers and offspring among indigenous people in Canada [42].

Of the mentioned risk factors, obesity is the most important factor causing insulin resistance [15]. While visceral obesity, owing to accumulation of fat in the omental and mesenteric regions, is highly correlated with insulin resistance; subcutaneous abdominal fat has less of an association with insulin insensitivity [15]. It is believed that in obese people, adipose tissue

releases higher amounts of non-esterified fatty acids, glycerol, hormones, pro-inflammatory cytokines, and other factors contributing to development of insulin resistance [28, 43]. The prevalence of general obesity (BMI $\geq$ 30 kg/m^2) was reported to be higher among participants who had Sami as their home language in three generations compared to Norwegian participants, both in men (26.9% vs 23.4%) and women (38.7% vs 24.3%) [44]. In the Finnmark study, BMI was reported to be the dominant risk factor for DM among men, while in women this association was less prominent. In both sexes a dose-response relationship between obesity and DM was observed [45].

High plasma triglycerides and low plasma HDL cholesterol have been shown to contribute to insulin resistance via increasing circulating levels of free fatty acids resulted from heightened insulin levels and enhanced chylomicron-assembly and secretion in the gut [46]. The strong observed association between hypertension and T2DM has been linked to insulin resistance [47], endothelial dysfunction [48], and inflammatory processes [49, 50] being present in both conditions. Smoking can increase the risk of T2DM through insulin resistance [51, 52] and inadequate compensatory insulin secretion response [53]. Physical activity has a well-known and strong protective effect against development of T2DM both directly by increasing insulin sensitivity [54] and indirectly by alteration in body mass and composition [55, 56]. Individuals of lower socioeconomic status (e.g. lower educated, unemployed) are at higher risk of developing T2DM and its complications [57-59]. Dietary habits have substantial impact on the development of T2DM [60]. Intake of foods with high glycaemic index was found to be an important risk factor for development of T2DM in numerous studies [61-63]. High consumption of sugar-sweetened beverages are strongly correlated with development of T2DM, particularly among children [64]. While regular consumption of white rice increased the risk of T2DM development, replacement of white rice with brown rice or other whole grains had a protective effect [65]. Higher intake of polyunsaturated fat and long-chain n.3

fatty acids was reported as being protective against development of T2DM, while higher intake of saturated fat and trans fat had adverse effect on glucose metabolism and insulin resistance [66]. Similarly, higher intake of butter, potato, and whole milk was reported to be associated with increased risk of development of T2DM, while higher consumption of fruits and vegetables was associated with lower risk of T2DM [67, 68]. The positive effect of plant-based regimens on insulin sensitivity and decreasing risk of T2DM has been attributed to their rich fibre content [69].

In Norway, Sami people live in both urban and rural areas in the inland or coastal regions and their diet, just like for other ethnic groups, differs from region to region. Furthermore, the dietary habits vary from generation to generation and from rural to urban settings [70]. As the T2DM is a chronic disease and its risk factors might have been present some years or decades before onset of the disease, it is challenging to attribute the development of T2DM to a specific kind of food eaten by a given ethnic group. In the inland regions, the consumption of reindeer is much higher among Sami people compared with non-Sami, while in the coastal regions this difference is less remarkable [71]. Interestingly, obesity is more prevalent in the inland regions where the reindeer consumption is highest [44, 72]. The higher intake of fat as spread on bread, total coffee, freshwater fish, reindeer meat, moose meat, and food made with animal blood and lower consumption of vegetables, potatoes, total fish, lean fish and chicken [71], might be an explanation for the higher prevalence of adiposity and consequently T2DM among Sami people [73].

1.2 Pre-diabetes

Pre-diabetes can be defined as impaired fasting glucose (IFG), impaired glucose tolerance test (IGT), or abnormal glycated haemoglobin (HbA1c) [74]. The term “pre-diabetes” implies a relatively high risk for future development of DM (although this is not always the case) [74]. The American Diabetes Association (ADA) stresses that IFG or IGT should not be regarded as clinical entities in their own right, but rather risk factors for T2DM as well as cardiovascular disease [3]. It has been reported that the incidence of T2DM among those with HbA1c levels at 6.0–6.4% is more than 10 times that of those with lower levels [75-78]. However, this HbA1c range fails to identify a substantial proportion of those who have IFG and/or IGT [3]. Prospective studies demonstrate that those with HbA1c range at 6.0–6.4% has a 5-year cumulative incidence of T2DM that ranges from 12 to 25% [75-78]. Data from the National Health and Nutrition Examination Survey (NHANES) indicate that among the nondiabetic adult population, a fasting plasma glucose (FPG) of 6.1 mmol/L corresponds to an HbA1c of 5.6% and an FPG of 5.6 mmol/L corresponds to an HbA1c of 5.4% [3].

Some trials have reported that among those with a pre-diabetes state, lifestyle interventions may prevent or delay onset of T2DM [79]. The ADA recommends that those with pre-diabetes state (especially those with HbA1c levels above 6.0%) being informed of their increased risk for T2DM and counselled about effective strategies such as weight reduction and physical activity to lower their risk [3].

1.3 Diagnosis of pre-diabetes and diabetes mellitus

T1DM is usually diagnosed based on its sufficiently characteristic clinical onset with relatively acute, extreme increases in glucose concentrations in the face of characteristic symptoms, such that specific blood glucose cut-offs are not required for diagnosis in most clinical settings [80]. Diagnosis of T2DM can be made through one of the criteria which follows [3]: (Criteria 1 through 3 should be confirmed by repeat testing.)

1) HbA1c ≥ 6.5% (48 mmol/L)

2) Fasting plasma glucose (FPG) ≥ 7 mmol/L (126 mg/dL)

3) 2-hour postprandial (2hpp) plasma glucose ≥ 11.1 mmol/L (200 mg/dL) after oral intake of 75g glucose

4) Random plasma glucose (RPG) ≥ 11.1 mmol/L (200 mg/dL) in the presence of classic symptoms of hyperglycaemia or hyperglycaemic crisis

In Norway, the HbA1c criterion is preferred for diagnosis of T2DM, and glucose measurements (fasting or oral glucose tolerance test [OGTT]) should be used if HbA1c is regarded inadequate [81, 82].

There is internationally an inconsistency as how to define pre-diabetes to the extent that "a transatlantic trip may cure or cause pre-diabetes simply as a result of small but important differences in diagnostic criteria" [83]. According to the ADA diagnostic criteria, pre-diabetes is IFG (fasting glucose= 5.6–6.9 mmol/L), IGT (two-hour glucose levels on the 75-gram oral glucose tolerance test=7.8–11.0 mmol/L), or HbA1c 5.7–6.4% [3]. In 2003, the International Expert Committee defined pre-diabetes as IFG 5.6–6.9 mmol/L or IGT 7.8–11.0 mmol/L [84]. In this report, the committee did not recommend HbA1c as a diagnostic test for DM due to lack of standardised methodology. In 2009, however, the committee approved HbA1c as a

diagnostic test for DM, recommending HbA1c 6.0–6.4% as pre-diabetic range [80]. The World Health Organisation (WHO) recommends IFG 6.0–6.9 mmol/L or IGT 7.8–11.0 mmol/L for categorising pre-diabetes [85]. It should be emphasised that as with the case with FPG and 2hpp, defining a lower cut-off for HbA1c to categorise pre-diabetes is somewhat arbitrary as the risk of T2DM with any measure or surrogate of glycaemia is a continuum, extending well into the normal ranges [3].

1.4 Non-fasting plasma glucose measurement

As mentioned above, random (non-fasting) plasma glucose (RPG) measurement can (especially in emergencies) be applied to check hyperglycaemia at the presence of classic signs and/or symptoms of hyperglycaemia. In some studies, especially in the past when other glycaemic indicators like HbA1c or FPG were not standardised or feasible, RPG measurement was used to ascertain DM. There are, however, some shortcomings of using RPG in both screening measures and epidemiological studies. Primarily, the RPG levels are strongly influenced by the postprandial time and times of the day [86]. Furthermore, sensitivity of RPG at ≥ 11.1 mmol/L for detecting DM is quite low [86]. According to the study conducted by Ziemer et al. the sensitivity, specificity and positive predictive value (PPV) of RPG for diagnosing DM at cut-off 140 mg/dL (7.8 mmol/L) was 20%, 97% and 26%, respectively (with the prevalence of DM around 5%) [86]. They did not present the sensitivity of the test at 11.1 mmol/l, but it is expected that higher cut-off of a test would yield even lower sensitivity. Based on these findings, Ziemer et al. suggested using RPG≥ 125 mg/dL (6.9 mmol/L) (sensitivity=40% and specificity=93%) as an opportunistic initial screening test for patients at risk of glucose intolerance. Johnson et al. reported 63% sensitivity and 87% specificity with an RPG cut-off of 130 mg/dL (7.2 mmol/L) [87]. Zhang et al. found that a (non-fasting) capillary glucose of 120 mg/dL (6.6 mmol/L) would provide a 89% specificity and 68% sensitivity [88]. The abovementioned studies used OGTT as the reference test. The common denominator for all these and numerous other studies is that the sensitivity of RPG measurement with cut-offs higher than 7.8 mmol/L for detecting undiagnosed DM is quite low. Perhaps the reason that only one RPG≥ 11.1 mmol/L (in the presence of classic signs and symptoms of DM) is sufficient for diagnosis of DM is the extremely high specificity and PPV of RPG measurement at this cut-off (albeit at the expense of extremely low sensitivity of the test).

1.5 Glycated haemoglobin

HbA1c demonstrates the ratio between glycated HbA1 and total HbA1 and represents the average plasma glucose concentration during the preceding 2–3 months [81]. The concentration of HbA1c is determined by concentration of glucose in blood and erythrocyte lifespan [89]. HbA1c has been used as an important biomarker for glycaemia control in patients with DM since 1980's [90]. In 2009, the International Expert Committee approved the diagnostic use of HbA1c in the wake of its standardisation [91]. In 2012, the Norwegian Directorate of Health approved and recommended HbA1c in the diagnosis of DM and stated that glucose measurements (fasting or OGTT) should be used when HbA1c is unreliable as a measure of the level of glycaemia such as in anaemia [82]. The ADA has recommended HbA1c $\geq$ 6.5% for diagnosing DM and 5.7 $\leq$ HbA1c < 6.5% for diagnosing pre-diabetes [3]. The diagnostic HbA1c cut-off levels were determined based on epidemiologic studies reflecting the strong association between HbA1c concentration and occurrence of retinopathy [3, 81, 92, 93]. HbA1c measurement has various advantages over glucose measurements like better sample stability, low intra-individual variation, independence of acute factors such as illness, recent food intake, stress, or exercise, and no need for prior fasting or glucose overload [94]. On the other hand, there are some conditions, which affect HbA1c values like iron deficiency anemia, chronic renal failure, pregnancy, and conditions causing shortened erythrocyte lifespan [95]. Beside these shortcomings of HbA1c, this test has been reported in several studies to be insensitive at the diagnostic cut-off of 6.5% [96-99]. The overlap between HbA1c and OGTT results was reported to be quite low [100, 101]. The ADA hope that greater practicality and convenience of the test would offset the low sensitivity of the test at the recommended diagnostic cut-off [3].

1.6 Prevalence of diabetes mellitus

1.6.1 Global burden of diabetes mellitus and its risk factors

T1DM comprises around 5–10% of DM cases [3]. T2DM accounts for approximately 90% of DM cases in all ages throughout the world [2]. In 2012, T2DM caused directly 1.5 million and indirectly 2.2 million deaths worldwide, being the eighth leading cause of death in both sexes [102]. The prevalence of T2DM is increasing worldwide and the number of affected people has risen from 108 million people in 1980 [103] to 422 million people in 2014 corresponding to 8.5% among adults >18 years [104]. The number of affected people worldwide is projected to increase to 592 million by the year 2035 [105]. Increasing global prevalence of T2DM is due to various factors like population growth, aging of societies, increasing risk factors for T2DM (e.g. obesity, sedentary lifestyle), more effective diagnostic instruments and case-finding schemes and increased longevity of the diseased [106]. Excess body fat, reflecting several aspects of diet and physical activity, is mentioned as the strongest risk factor for T2DM worldwide [106]. In 2014, it was estimated that globally one in three adults over 18 years were overweight and more than one in ten were obese [107]. At the same time, physical inactivity became a great concern throughout the world. Based on data from 2010, it was estimated that 27% of women and 20% of men from all countries were insufficiently physically active [107]. In 2017, health authorities in Norway reported that only around one third of Norwegian adult population applied the recommendations regarding physical activity [108].

1.6.2 Prevalence of type 2 diabetes mellitus in Europe

While in 1980 the estimated prevalence of T2DM among adults >18 years in Europe was 5.3%, this figure reached 7.3% in 2014 [106]. Data suggest that the incidence and prevalence of T2DM in European countries is frequently higher among people of lower socioeconomic status and these inequalities were mediated by BMI [109]. It should be mentioned that the

proportion of undiagnosed cases of T2DM varies widely from country to country and even in high-income countries this proportion might be as high as 30–50% [110]. A systematic review and meta-analysis study revealed that the prevalence of T2DM among ethnic minority groups resident in Europe was considerably higher than in ethnic Europeans [111].

1.6.3 Prevalence of diabetes mellitus in Norway

In 2004, the prevalence of known cases of any type DM in Norwegian adults over 30 years old was estimated 3.4% [11]. This estimate was based on data from nine regional surveys. The authors estimated also that the number of unknown cases might be nearly equal to the number of known cases in the age group ≥30 years old. According to the Norwegian Institute of Public Health, in 2017, approximately 245,000 (4.7%) Norwegians had known DM, of which 216,000 were estimated to have T2DM [112]. The annual number of new users of glucose-lowering agents in Norway is reported around 15,000–16,000 [113]. A recent nationwide cohort study based on national registries in Norway showed that the prevalence of known T2DM among inhabitants aged 30–89 years increased from 4.9% in 2009 to 6.1% in 2014 [10]. According to this study, at the same time, the incidence of known cases of the disease decreased significantly from 609 cases per 100,000 person-years in 2009 to 398 cases per 100,000 in 2014, an annual reduction of 10.1%. This decline was observed for both pharmacologically and non-pharmacologically treated T2DM and in all sex, age, education level and place of birth subgroups [10]. In 2006, the third Nord-Trøndelag Health Survey (HUNT 3) reported the prevalence of any type DM in adults aged 20 years and over living in the county of Nord-Trøndelag to be 4.3% [114]. In 2011, the direct costs of DM treatment in Norway reached €408 million; and indirect costs reached €108 million [115].

It is worth mentioning that, like in many other countries and societies, the prevalence of DM is not homogenous across Norway or even within its cities and districts. Results from three

population-based, cross-sectional studies conducted between 2000 and 2002 in Oslo revealed that the prevalence of self-reported adult DM was strikingly different between inhabitants of West and East Oslo [116]. While Western parts of Oslo have traditionally been inhabited by a wealthy, highly educated and ethnically homogeneous community, the historically disadvantaged and much more densely populated 'East' is popularly associated with immigration and social stigma [116]. According to the mentioned study, while the prevalence of self-reported DM in the Western parts of Oslo was 1.6%, this prevalence was 5.4% in the Eastern parts. The observed spatial disparity in the prevalence of DM remained highly significant even after adjustment for a range of covariates such as ethnicity, age or BMI. The results showed that ethnicity is a strong predictor for DM with being of non-Western origins increases the odds by a factor of almost 5 [116].

1.6.4 Diabetes among indigenous peoples

Higher prevalence and incidence of T2DM among indigenous peoples compared with benchmark populations worldwide seems to be a common phenomenon [117]. Indigenous peoples throughout the world are experiencing an unprecedented epidemic of T2DM [117]. While incidence rates of T2DM have been on the rise during the last decades, the disease disproportionately affects different racial and cultural groups [118].

The prevalence of self-reported DM among indigenous Australians aged ≥ 40 years was 37.3% (95% confidence interval (CI): 34.6–40.2%) in 2008, which was more than eight times higher than that in non-indigenous Australians [25]. This happens in spite of the fact that the prevalence of DM was extremely uncommon among Australian indigenous populations some 30 years ago (the rate was 10% of national rate) [117, 119]. A systematic review reported a great variation in the prevalence of DM between different segments of the Australian Indigenous population [120]. According to this study, the prevalence of (any type) DM was

greater among Indigenous Australian women compared to men and in remote compared to urban settings. A great deal of the disparities in the prevalence of DM can be attributed to disadvantageous socioeconomic status of indigenous people in Australia [121].

While the overall age-standardised prevalence of DM in Canada in 2008–2009 was 6.8%, the age-standardised prevalence of DM was 17.2% among First Nations people living on-reserve, 10.3% among those living off-reserve and 7.3% among Métis [122]. The corresponding prevalence among Inuit was similar to that of the general Canadian population [122]. Although the prevalence of DM in Canadian Inuit is now comparable to the general Canadian population, it was around 2% in 2001 [122]. The age-standardised prevalence of diagnosed DM increased 35% among adults aged > 20 years residing in rural Status Aboriginals in Alberta, Canada, from 10.9% (95 % CI: 10.4–11.5) in 1995 to 14.7% (95% CI: 14.2–15.2) in 2006. Corresponding prevalence in urban Status Aboriginals increased by 22% from 9.4% (95% CI: 8.5–10.3) in 1995 to 11.5% (95% CI: 10.9–12.1) in 2006 [123].

The Greenland population is a population isolate. While the prevalence of T2DM in Greenland was at a very low level in the 1960s, a study by Jørgensen et al. revealed that around 9% of adult (≥ 18 years) Inuit in Greenland suffered from DM in 2005–2010 with 79% of them being previously undiagnosed [124]. This prevalence is almost twice as high as the prevalence of T2DM in Denmark, a country that Greenland is culturally and politically linked to [125]. The study showed also an inverse correlation between the prevalence of DM and urbanisation with people of lower socioeconomic status living in small towns and villages being at higher risk. The high prevalence of T2DM in Greenland is despite the fact that Greenlanders mostly consume a traditional Inuit diet with a high content of marine mammals and fish [126]. Therefore, changes in traditional lifestyle risk factors cannot fully explain the high prevalence of T2DM in Greenland and some genetic risk factors might be involved as

well [127]. A newly performed association mapping of T2DM-related quantitative traits among 2575 Greenlandic DM-free individuals discovered a nonsense p.Arg684Ter variant in the gene TBC1D4 with an allele frequency of 17% [128]. According to authors of the study, homozygous carriers of this variant have significantly higher concentrations of plasma glucose and serum insulin 2 hours after an oral glucose load compared with individuals with other genotypes. Increasing number of p.Arg684Ter alleles leads to a severely decreased insulin-stimulated glucose uptake in skeletal muscles, leading to postprandial hyperglycaemia, impaired glucose tolerance and T2DM [128]. In recent years, the quality of DM health care in Greenland has improved and the prevalence of diagnosed DM has increased since 2008 due to heightened awareness, increased funding and case-finding schemes [129].

While T2DM was probably uncommon among American Indian and Alaska Native (AI/AN) populations before the 1940s [130] it was reported that in 2010 AI/AN had a higher rate (over 14%) of diagnosed T2DM than any other racial or ethnic group in the USA [131]. At the same time, the prevalence of overweight and obesity was also reported to be higher among AI/AN compared to White or Hispanic Americans [22, 132]. Diabetes-related mortality rates are three times higher among AI/AN compared with White Americans [133] and DM is the fifth leading cause of death among AI/AN [22]. A study performed by Fretts et al. revealed that around half of American Indians developed DM by age 55 years and a high proportion of those affected by DM remained undiagnosed [134]. Fretts et al. reported also in the same study that of 2001 adult (aged 19–74) AI/AN free of DM and cardiovascular diseases recruited in the study and followed for 8 years, 243 individuals (12.1%) developed DM with consuming processed meat being a significant risk factor for developing DM among AI/AN (OR: 1.63).

The incidence rates of T2DM among Pima Indians in Arizona aged 5 years or older was as high as 25 cases/1000 person-years between 1965 and 2003 [135]. They feature a classic and well-known example of high incidence and prevalence of a subtype of T2DM characterized by obesity, insulin resistance, and a relative insulin deficiency [136]. Just like many other indigenous peoples, they have experienced a transition from a traditional lifestyle with low-calorie diet and high physical activity to a sedentary lifestyle with high calorie intake [137]. Like Inuit in Greenland, Pima Indians are a population isolate, i.e. the population is derived from a small number of individuals with limited connection to other populations [137]. Generally, such populations exhibit a unique profile of rare diseases [138], and the prevalence of common diseases like T2DM might also be strikingly different from large, open populations [127]. Isolated populations are more vulnerable to rapid changes in the environment and lifestyle [127].

Unlike Inuit in Greenland and Pima Indians in the USA, Sami people in Norway have not been an isolated population, neither geographically nor genetically. Throughout the history, they have been in constant interaction with surrounding populations and now they live well-integrated lives as part of Norwegian society [139]. The prevalence of metabolic syndrome among both Sami and non-Sami inhabitants of Northern Norway was reported to be high in the SAMINOR 1 Survey [13]. While, according to this survey, the prevalence of self-reported DM was not different between the Sami and non-Sami groups, ethnicity appeared to affect DM treatment, which was more prevalent among Sami than non-Sami women.

In the Finnmark Study (1993) and the SAMINOR 1 and 2 Surveys, Sami women reported lower leisure-time physical activity than their non-Sami counterparts, while both Sami men and women were significantly more active during work [140-142].

1.7 Ethnicity

The concept of ethnicity is multifaceted. Self-defined ethnicity depends on the context the definition has been shaped and applied in and may change over time [143]. According to various sources, ethnicity can be defined as a group of individuals who identify themselves and have a sense of belonging to each other based on some similarities like assumed common ancestry, language, dialect, society, culture, religion, mythology, rituals, nation, history, homeland, dressing style, art, and physical appearance [144-147]. The complexity of individual identity makes writing with precision about ethnicity challenging [148]. The ethnicity is not a mutually exclusive concept and one may be assigned to or conceive his/herself as member of different ethnic groups according to country of origin, ancestry, birthplace, language and so on [149]. Bhopal in his book on race and ethnicity emphasises that in most cases the differences between individuals belonging to a certain ethnic group are larger than the differences between different ethnic groups [149]. This results in ethnic categories being broad with overlapping and obscure borders [148]. Due to these issues, it has been recommended that researchers should elaborate on how and on what basis they defined the ethnic groups [150].

Each definition of a given ethnic group relies on one or a few main feature(s) of that group and may in addition make use of other less important distinguishing features to further define the group. This may lead to having different definitions of an ethnic group, which might adversely affect results and comparisons. To ensure that any observed difference between ethnic groups is a result of real differences in the concerned endpoint of the study and not the applied definition of the ethnic group, sensitivity analyses can be helpful to avoid spurious conclusions [151].

1.8 The Sami people in Norway

The Sami are an indigenous people who have traditionally inhabited northern parts of Norway, Sweden and Finland, and in Russia's Kola Peninsula [152]. In Norway, their settlement area, Sápmi, encompasses Finnmark county in the north to Engerdal in Hedmark county in the south. According to Norwegian legislation, the Sami people are recognised as indigenous people [153]. Although there is no ethnic registry in Norway, it is estimated that the largest population of Sami people (proposed to be around 40,000) live in Norway [154]. The Sami people have traditionally pursued various livelihoods including reindeer husbandry, small-scale fishing, and agriculture [155, 156]. Today Sami people are active in almost all professions and only less than 10% of them are actively pursuing the traditional practices [157]. The Sami people in Norway consists of heterogeneous groups such as North, East/Skolt, Lule, Ume and South Sami, with various cultural, linguistic, and dietary features [158].

The history of Sami people has many similarities with the histories of other indigenous peoples throughout the world. The Sami people have for centuries been subject to discrimination and for more than 100 years victims of an official assimilation policy exerted by the Norwegian government. Motivation for this policy was "Social Darwinism" and national romantic ideologies [159]. Some areas like the areas bordering Russia and Finland as well as coastal Sami areas of Northern Troms and Finnmark were more exposed to this so-called Norwegianisation policy [155, 159, 160]. The education system was one of the most effective tools for Norwegian authorities to enforce this policy by banning the Sami language at schools and removing Sami children from their cultural and linguistic environments [161, 162]. Besides linguistic policies, Norwegian authorities encouraged thousands of people from other parts of Norway to immigrate and settle in Finnmark county, which turned Sami people into minority groups in their own traditional territories at the coastal areas [163]. During the

19th and 20th centuries, fishing industry, which traditionally was one of main livelihoods of Sami people in coastal areas, became industrialised. This had profound economic as well as lifestyle impact on Sami inhabitants in coastal areas [164]. The evacuation of coastal areas during and ensuing the second World War in tandem with rapid modernisation process occurring in almost all aspects of labour market did put extra pressure on Sami language and culture [155].

Nowadays, many Sami people are active in administrative and service sectors and almost the entire reindeer husbandry and agriculture are mechanised with less physical activity involved [165]. The pro-Sami movements and revitalisation policies implemented from 1960s have, to some extent, managed to reverse the adverse effects of the past Norwegianisation policies [139]. The changes in lifestyle towards so-called western and sedentary lifestyle with unhealthy diet, which have affected all ethnic groups in the region, have continued in the same direction until now [166]. Like a two-edged sword some of these changes have been unfavourable, others have had beneficial effects on the health situation.

The abovementioned colonisation, assimilation and marginalisation policies exerted on Sami people throughout the history might have made them more vulnerable to adverse health outcomes like cardiovascular diseases, obesity, metabolic syndrome and chronic muscle pain [167].

1.9 The Sami people and health studies

Research on the Sami people was started in the early 1800s by gathering skeletal material from archaeological excavations and autopsies at the Department of Anatomy, the University of Oslo, Norway [168]. These so-called research activities focused on racial attributes and used cranial indices and skull measurements with the intention of distinguishing ethnic groups. Rather than investigating Sami's lifestyle and culture, they tried to use physio-anthropological features to provide a scientific evidence for the superiority of the benchmark population. This discriminatory and racist approach to scientific methods left a deep impression on many Sami people causing them to distrust researchers [168].

Since the Second World War, a growing political awareness and generally higher levels of education among the Sami people, in combination with increasing interest and involvement of researchers with Sami affiliations have paved the way for new studies on Sami health issues with a totally different approach and ethical principles [168]. The first population-based study conducted in the Sami regions was the different surveys of the Finnmark Study (1974–2000), which included all ethnic groups living in the Finnmark county [169]. Before the Finnmark Study, only some isolated reports from practitioners working in North-Sami regions were published, reporting issues such as tuberculosis, echinococcosis and high rates of infant mortality [170-172].

The establishment of the Centre for Sami Health Research at UiT the Arctic University of Norway in 2001 was the turning point in meeting the increasing need for knowledge about Sami peoples' health and living conditions. Ever since, the centre has collaborated with several regional, national and international actors in the field of research among Sami or other indigenous peoples and facilitated a substantial increase in publications and reports in this regard [168, 173, 174].

1.10 Kvens

Kvens are descendants of Finnish ethnicity who immigrated from Sweden and Finland to Norway and settled in the northern parts of Norway in the 1700s and 1800s [175]. Since 1998, Kvens are recognised as a national minority in Norway [176]. The Kvens in Norway do not have indigenous status like the Sami. Similar to the Sami people, the Kvens have also experienced linguistic and cultural assimilation in the Norwegian society and enormous changes in lifestyle and way of living during the past centuries and decades. A large number of Kvens mentioned in the questionnaire affiliations to either Sami or Norwegian ethnic groups besides their main ethnicity. Due to relatively small number of Kvens in our surveys, they were not assessed separately in the present thesis.

1.11 The aims of the thesis

The inspiration and motivation for the present thesis was the paucity of publications regarding the incidence and prevalence of DM among the Sami people inhabiting rural districts in Norway, and several publications reporting higher incidence and/or prevalence of lifestyle related diseases, especially T2DM, among other indigenous peoples throughout the world. The overall aim of the thesis is to assess the burden of DM among Sami and non-Sami inhabitants of Northern Norway.

The specific aims of the thesis are:

1) To measure the prevalence of pre-diabetes and DM among inhabitants of the included municipalities of Northern Norway in two points of time; the SAMINOR 1 Survey (2003–2004) and the SAMINOR 2 Clinical Survey (2012–2014);
2) To explore any ethnic difference between Sami and non-Sami inhabitants of these municipalities in terms of dysglycaemia;
3) To determine the 8-year cumulative incidence of DM from the SAMINOR 1 Survey (2003–2004) to the SAMINOR 2 Clinical Survey (2012–2014);
4) To elucidate some possible explanatory factor(s) behind any ethnic difference in the prevalence or cumulative incidence of DM in the included municipalities.

2 Methods

2.1 The SAMINOR 1 Survey

In 2003–2004, the Centre for Sami Health Research at UiT The Arctic University of Norway, in collaboration with the Norwegian Institute of Public Health, conducted the SAMINOR 1 Survey, a cross-sectional population-based survey on health and living conditions in regions with both Sami and Norwegian populations [177]. The survey was first designed and planned as a cardiovascular screening in Northern Norway by the National Health Screening Service and then by joining the Centre for Sami Health Research took the form of the SAMINOR 1 Survey. This survey included municipalities and districts in Norway with a high proportion of people with Sami ethnicity, as determined by ethnicity and language information reported in the 1970 census and historical and local knowledge about traditional Sami settlements [178]. The included municipalities were: Karasjok, Kautokeino, Tana, Nesseby, Porsanger, Lebesby, Loppa, Kvalsund, Alta, Lyngen, Storfjord, Kåfjord, Kvænangen, Lavangen, Skånland, Narvik, Evenes,Tysfjord, Hattfjelldal, Røyrvik, Namsskogan, Grane, Snåsa and Røros (Figure 3). These municipalities are all except the Alta municipality located in rural areas. Only some districts of Hattfjelldal, Grane, Narvik, Namsskogan, Snåsa and Røros municipalities with considerable proportion of Sami inhabitants were included in the survey.

An invitation was mailed several weeks before the survey with information on the time and place of screening, relevant and required information about the survey, and questionnaires. The questionnaires were returned by the participants at the time of the clinical examination [177]. All residents aged 30 and 36–79 years registered in the National Registry in the selected regions were invited to participate in the SAMINOR1 Survey regardless of their ethnic background (n=27,987). Due to the small sample size and low participation rate among 30-year-old inhabitants – only 328 participants out of the 836 invited (39.2%) – , they were

excluded from analyses (DM prevalence estimates) in the first paper. Of the remaining individuals, 16,538 participated and gave consent to use their information in medical research (60.9%).

The survey included a short clinical examination, blood sampling and three self-administered questionnaires: an initial two-page questionnaire that contained a variety of questions, including questions about ethnicity; a three-page screening questionnaire that collected information about symptoms, lifestyle factors, and some diseases, including DM; and an additional four-page questionnaire that collected cultural, social, and nutritional information. The English version of the SAMINOR 1 Survey questionnaire is available at www.saminor.no.

In the first four municipalities, it was possible to participate with the initial questionnaire only, without taking part in the clinical examinations or fill in the main questionnaire. In addition, due to a design problem, some participants underwent clinical examinations without filling in the initial questionnaire. The questionnaires were prepared in Norwegian and translated into the three main Sami languages; however only the Northern Sami version was used in the six municipalities defined in the Sami Language Act at that time as the Sami Language Administrative District (Karasjok, Kautokeino, Tana, Nesseby, Porsanger, and Kåfjord). More than 98% of the participants completed the Norwegian version of the questionnaire. In 15 of the 24 municipalities, non-responders were offered a second chance to attend when the buses returned a couple of months later. Unlike inhabitants in Finnmark and Troms counties, inhabitants in Nordland and both Trøndelag counties did not receive a second invitation; thus, this design affected the participation rate in these areas.

A trained team of experienced fieldworkers undertook the practical work. The clinical examination was carried out in two buses that moved throughout the study area, spending 1–6

weeks in each of the municipalities included in this analysis [177]. Non-fasting venous blood samples were drawn with the participants in a seated position. Participants came to the examination buses throughout the day, from 8 o'clock in the morning to 19 o'clock in the afternoon. The time after the last meal ranged from immediate after meal to 9 hours with average postprandial time a little over 2 hours. The samples were left to coagulate for a minimum of 30 minutes and were centrifuged within 1.5 hours. Serum was sent by overnight mail to the Department of Clinical Chemistry, Ullevål University Hospital, Oslo, Norway, where glucose was measured directly by an enzymatic method (Hitachi 917 autoanalyzer, Roche Diagnostic, Switzerland). Autonorm Human Liquid was used as internal quality control material. The control material was analysed at the start and for every 30th sample. During physical examination, height (to the nearest 0.1 cm) and weight (to the nearest 100 grams) were measured with an electronic height and weight scale (DS-102, Dong Sahn Jenix, Seoul, Korea) with the participant wearing light clothing without shoes. Body mass index (BMI, kg/m^2) was calculated as weight (kg)/(height (m))^2 to the nearest 0.1 unit. Waist circumference (WC, cm) was measured at the umbilicus level to the nearest centimetre at the end of expiration with the individual standing and breathing normally [177].

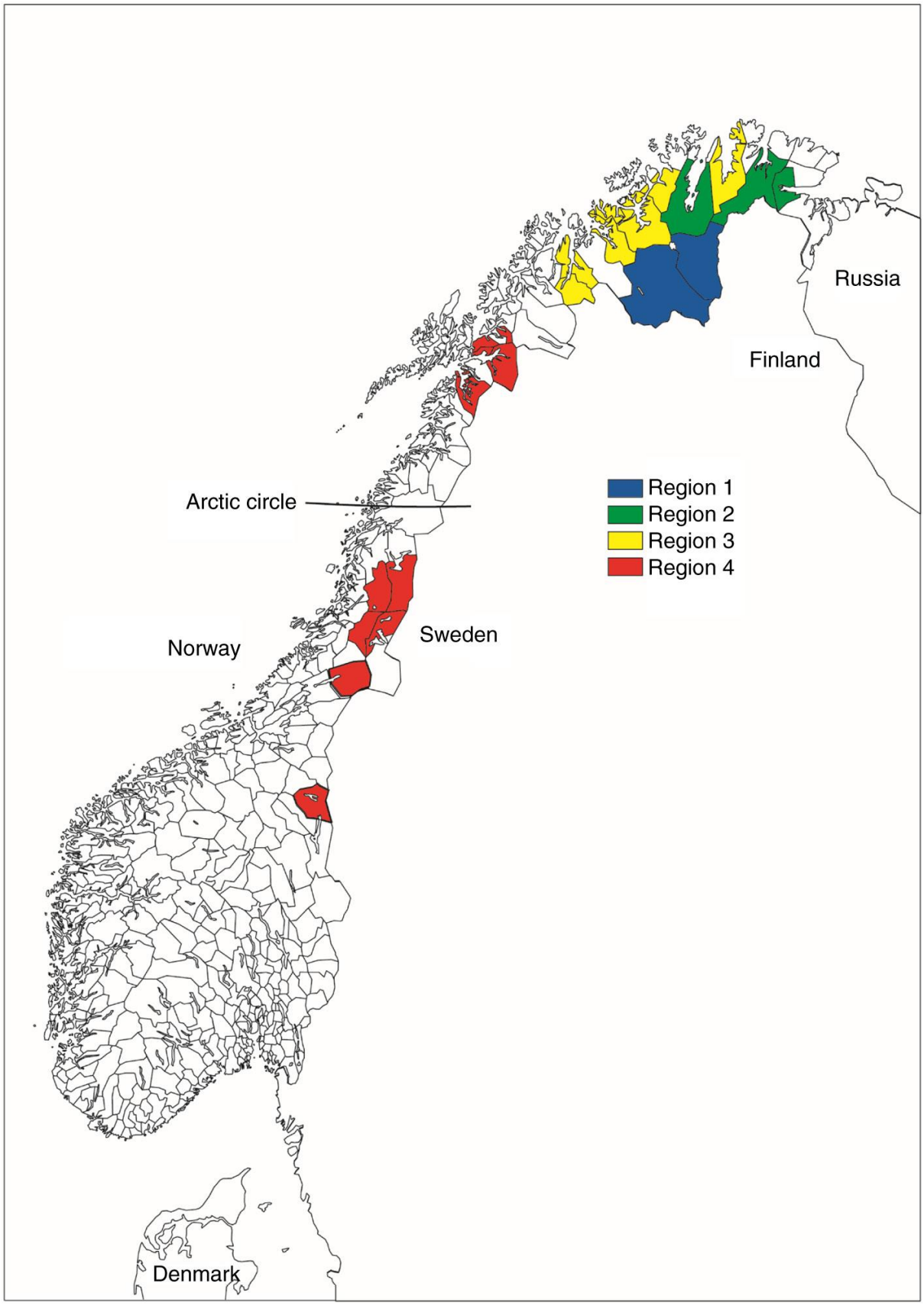


Figure 3. The map of the included municipalities and the 4 defined geographical regions. The SAMINOR 1 Survey. Published with permission from Centre for Sami Health Research.

2.2 The SAMINOR 2 Survey

The Centre for Sami Health Research conducted also the SAMINOR 2 Survey, which consisted of two parts. The first part of this survey, the SAMINOR 2 Questionnaire Survey, was purely questionnaire-based and was conducted in year 2012 among inhabitants aged 18–69 years from the same 24 municipalities and districts included in the SAMINOR 1 Survey in addition to the municipality of Sør-Varanger [179]. The second part of the survey, the SAMINOR 2 Clinical Survey was conducted in 2012–2014 and consisted of self-administered questionnaires, a clinical examination, and analysis of blood samples. The survey included individuals aged 40–79 years old from 10 municipalities of Finnmark, Troms, and Nordland counties: Kautokeino, Karasjok, Porsanger, Tana, Nesseby, Storfjord, Kåfjord, Lyngen, Skånland, and Evenes (Figure 4). Please note that these 10 municipalities were included also in the SAMINOR 1 Survey and the SAMINOR 2 Questionnaire Survey.

Like in the SAMINOR 1 Survey, an invitation was mailed several weeks in advance to eligible inhabitants of the designated municipalities along with pertaining information about the provided questionnaires and the time and place of the clinical examination. Participants were asked to present their completed questionnaires at the time of the clinical examination, which was performed at one of 10 research stations established in 9 municipalities (two research stations were set up in Kåfjord municipality in the communities of Manndalen and Birtavarre; participants living in Evenes visited the research station in neighbouring Skånland). In total, 12,455 were invited, and 6004 took part in the clinical examination. All the clinical examinations were performed within 2–7 weeks in each municipality.

During the clinical examination, trained personnel measured participants' height (to the nearest 0.1 cm) and weight (to the nearest 100 grams) using an electronic height and weight scale (DS-103, Dongsahn Jenix, Seoul, Korea) with participants wearing light clothing and no

shoes. These measures were then used to calculate body mass index (BMI, kg/m^2). Waist circumference (WC, cm) was measured at the umbilicus to the nearest cm with the participant standing and breathing normally. Waist-to-height ratio (WHtR) was calculated by dividing the waist by the height. Finally, blood samples were collected by venepuncture at normal venous pressure, with participants in a seated position. Blood samples were stored at -20°C in a freezer and after some weeks transported to the biobank at UiT The Arctic University of Norway where the serum samples were stored at -70°C in ultra-freezers. Random plasma glucose was analysed at the Laboratory of the Department of Clinical Chemistry, University Hospital of North Norway, Tromsø, Norway in the period of September 2014 – November 2014.

Glucose was measured on the Cobas 8000 system from Roche/Hitachi using an in-vitro test for the quantitative determination of glucose in human serum. The test principle is an ultraviolet test with enzymatic references method with hexokinase. Glucose values for human serum obtained on the Roche/Hitachi c 701 analyser were compared with those determined using the same reagent on the Roche/Hitachi cobas 501 analyzer. This method has been standardised against isotope dilution mass spectrometry reference measurement procedure. The analyser automatically calculates the analyte concentration of each sample by conversion factor mg/dl x 0.0555= mmol/L. All reagents were purchased from the same company.

Glycated haemoglobin (HbA1c) was analysed immediately on whole blood at the examination site, with The DCA Vantage™ (Siemens Medical Solutions Diagnostics, Tarrytown, NY), which is based on latex agglutination inhibition immunoassay methodology and provides results in 6 minutes.

Questionnaires differed by age group: participants aged 40–69 years received an 8-page questionnaire that covered a broad range of questions on lifestyle, diet, risk factors, and

diseases. In contrast, participants aged 70–79 years received a 4-page questionnaire with larger fonts. Only questions that were identical in the two questionnaires were included in the present analyses. Both questionnaires were originally prepared in Norwegian and then translated into the Northern Sami language owing to the fact that all 10 municipalities belong to the Northern Sami language area. In Kautokeino, Karasjok, Nesseby, and Tana municipalities, participants received both the Sami and Norwegian versions of the questionnaire. In Kåfjord, Storfjord, Porsanger, and Lyngen municipalities, the questionnaire in the Northern Sami language was available on request. Invitees in Skånland and Evenes municipalities received the Norwegian questionnaire only. Among all of our participants, less than 5% chose to use the Sami version of the questionnaire. The English version of the SAMINOR 2 Clinical Survey questionnaire for 40–69-year-old participants is available at www.saminor.no.

2.3 Ethics

The Norwegian Data Protection Authority approved the SAMINOR 1 Survey and the SAMINOR 2 surveys. The surveys were approved by Regional Committees for Medical and Health Research Ethics (REC North). All participants gave a written informed consent, which also included a consent to later linkages to national registers, previous censuses and cardiovascular screenings. Information letters and brochures were elaborated in co-operation with the Norwegian Data Protection Authority and REC North. This specific diabetes study was also accredited by REC North and the SAMINOR Project Board.

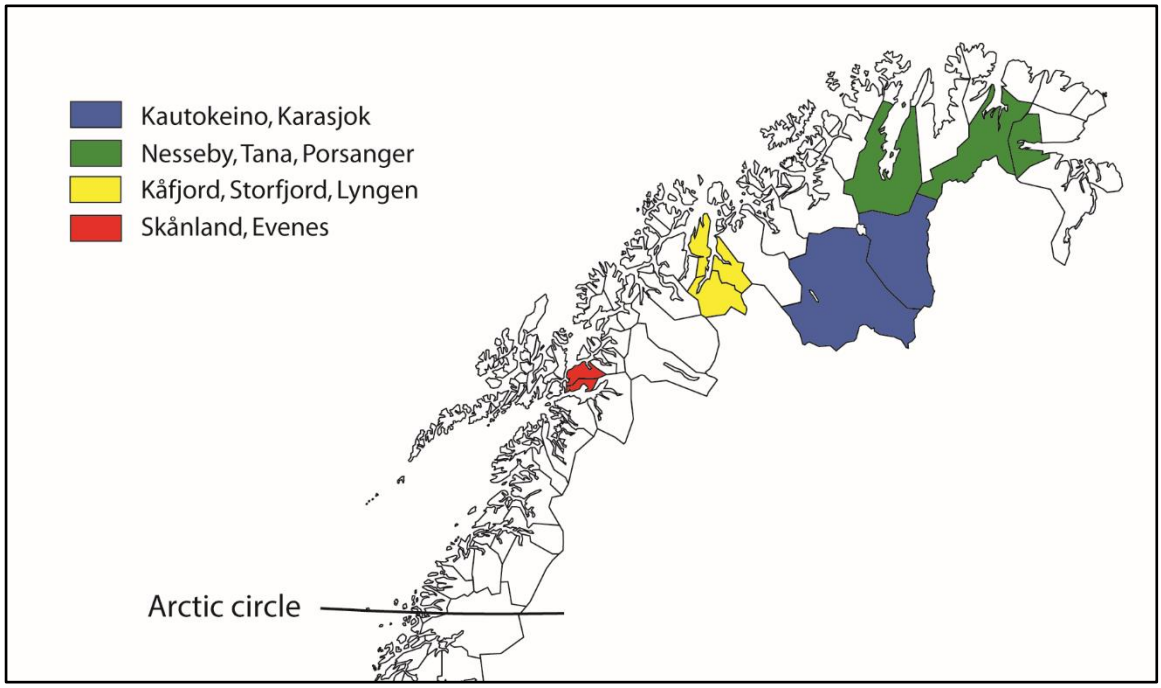
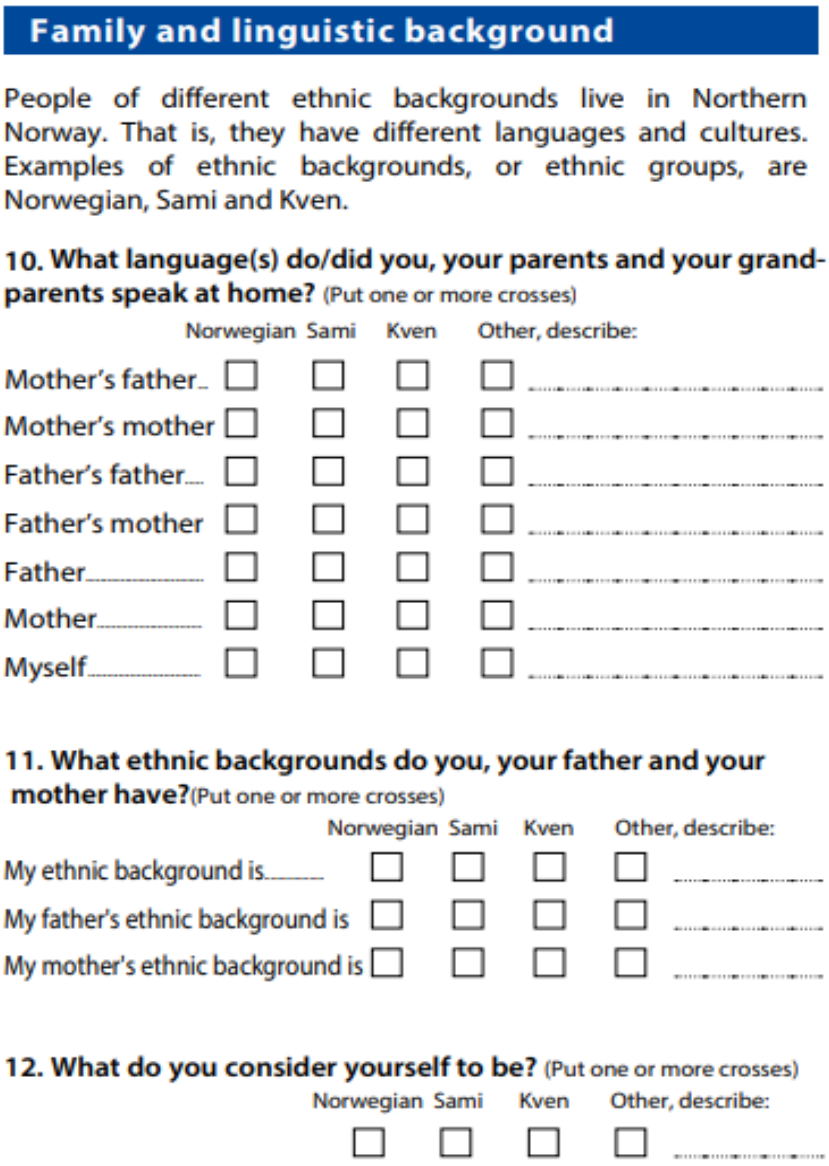


Figure 4. The map of the 10 municipalities included in the SAMINOR 2 Clinical Survey. Published with permission from Centre for Sami Health Research.

2.4 Definition of ethnicity

In both surveys, information on ethnicity was obtained from the questionnaires with identical questions regarding ethnicity (Figure 5). The questions were: “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 do you consider yourself to be?” On all items the response options were: “Norwegian”, “Sami”, “Kven”, and “Other”. The questions were to be answered separately for each relative and multiple answers were allowed. Sami ethnicity was defined based on two criteria: 1) self-identification as a Sami, and 2) a Sami language connection. Sami self-identification was regarded as fulfilled if the respondent considered him/herself to be Sami or reported having a Sami ethnic background. Sami language connection was defined if at least one grandparent, parent, or the participant him/herself spoke a Sami language at home. Participants who fulfilled both criteria were categorised as Sami. All other participants were categorised as non-Sami.

To assess the reproducibility of answers to ethnicity questions, results from the SAMINOR 1 and 2 Surveys were compared. Of a total of 3303 persons who participated in both the SAMINOR 1 Survey and the SAMINOR 2 Clinical Survey, respectively, and included in Paper 3, 1314 (39.8%) and 1317 (39.9%) reported having Sami ethnicity with a high agreement between answers given to ethnicity questions by each participant (Cohen’s Kappa=0.85, $p<0.01$).



Family and linguistic background

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.

10. 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	☐	☐	☐	☐ .....

11. What ethnic backgrounds do you, your father and your mother have? (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	☐	☐	☐	☐ .....

12. What do you consider yourself to be? (Put one or more crosses)

Norwegian	Sami	Kven	Other, describe:
☐	☐	☐	☐ .....

Figure 5. Questions on language and ethnicity from the questionnaire

2.5 Paper 1

2.5.1 Study participants

Of the 27,151 inhabitants (36–79 years) who were invited to the SAMINOR 1 Survey, 16,538 (60.9%) agreed to participate. After exclusion of those with either missing ethnicity variable or outcome variable (self-reported DM and/or non-fasting plasma glucose), 15,208 (56.0%) individuals were included in the study (Figure 6 and Table 1).

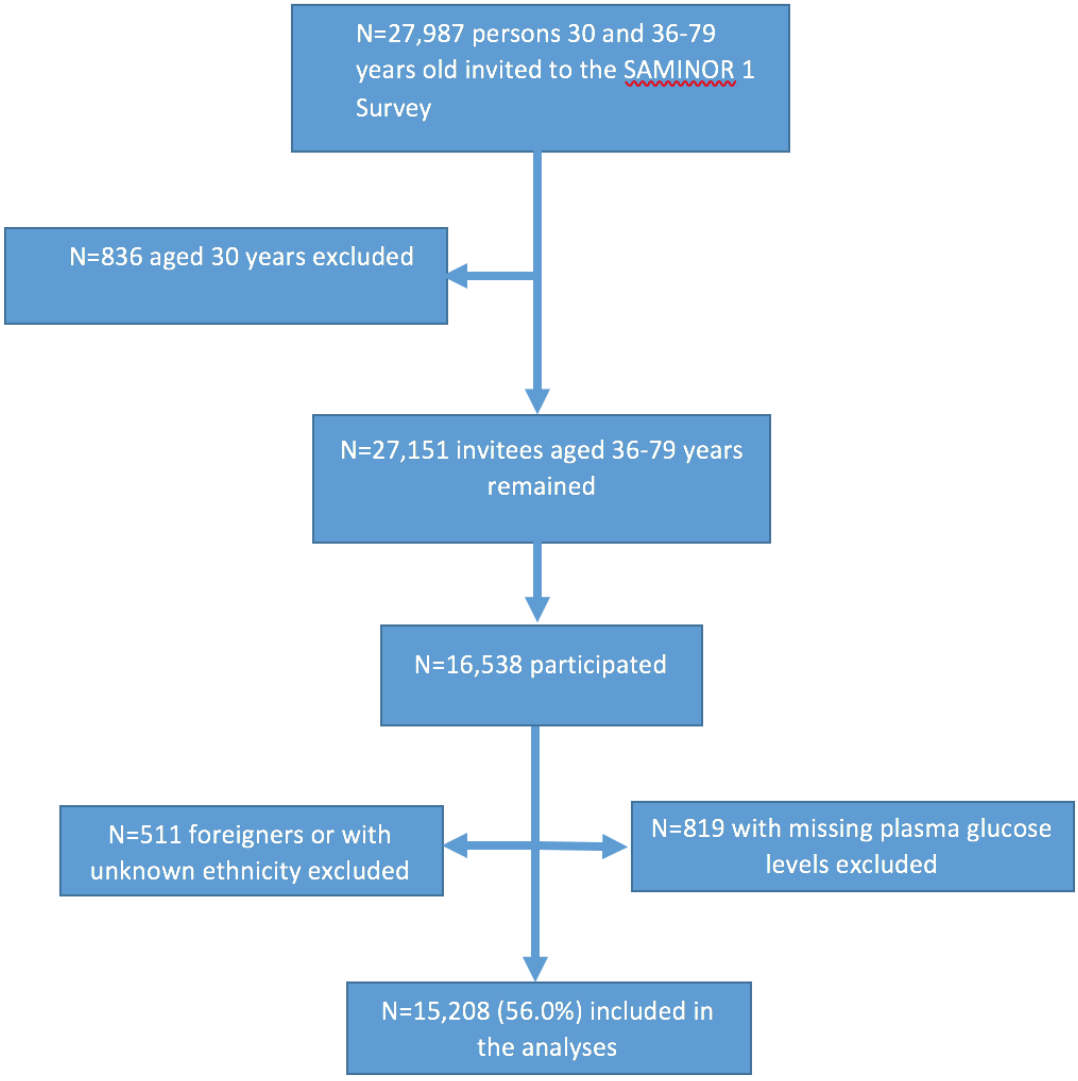


Figure 6. The invited in the SAMINOR 1 Survey, the participants, exclusions, and the actual study sample, paper 1

Table 1. Characteristics of the invited (36–79 years old), participants, sub-groups and the final working sample in paper 1. The SAMINOR 1 Survey

		Invited (%)	Total participation (%)	Participated in clinical examinations (%)	Clinical examinations and initial questionnaire (%)	Included in Paper 1 (%)
Number		27,151	16,538	15,718	15,515	15,208
Percent			60.9	57.9	57.1	56.0
Sex						
	Men	14114 (52)	7985 (48)	7529 (48)	7444 (48)	7315 (48)
	Women	13037 (48)	8553 (52)	8189 (52)	8071 (52)	7893 (52)
Age (years)						
	36–49	10748 (40)	6040 (37)	5722 (36)	5654 (36)	5492 (36)
	50–59	7739 (28)	5063 (31)	4833 (31)	4773 (31)	4681 (31)
	60–79	8664 (32)	5435 (33)	5163 (33)	5088 (33)	5035 (33)
Regions*						
	1	2704 (10)	1777 (11)	1366 (9)	1190 (8)	1169 (8)
	2	4174 (15)	2687 (16)	2301 (15)	2283 (15)	2221 (15)
	3	14078 (52)	8647 (52)	8631 (55)	8624 (56)	8465 (56)
	4	6195 (23)	3427 (21)	3420 (22)	3418 (22)	3353 (22)
Marital status						
	Single	6472 (24)	3202 (19)	2952 (19)	2903 (19)	2842 (19)
	Married	15175 (56)	10259 (62)	9848 (63)	9728 (63)	9535 (63)
	Widow(er)	1826 (7)	1066 (6)	1015 (6)	992 (6)	979 (6)
	Divorced	3054 (11)	1704 (10)	1614 (10)	1606 (10)	1574 (10)
	Separated	623 (2)	307 (2)	289 (2)	286 (2)	278 (2)
	Missing	1	0	0	0	0
Ethnicity						
	Sami		3932 (24)	3406 (22)	3406 (22)	3398 (22)
	Non-Sami		12095 (74)	11831 (77)	11831 (77)	11810 (78)
	Other		240 (1)	226 (1)	226 (1)	0
	Missing		271	255	52	0
Education						
	0–7 years		2551 (17)	2543 (17)	2474 (17)	2454 (17)
	8–12 years		7469 (51)	7452 (51)	7373 (51)	7265 (51)
	13+ years		4757 (32)	4749 (32)	4708 (32)	4562 (32)
	Missing		1761	974	960	927

*Region 1: Karasjok and Kautokeino municipalities;

Region 2: Porsanger, Tana and Nesseby municipalities;

Region 3: Lyngen, Storfjord, Kåfjord, Kvænangen, Alta, Loppa, Kvalsund and Lebesby municipalities;

Region 4: Lavangen, Narvik, Evenes, Skånland, Tysfjord, Hattfjelldal, Røyrvik, Namsskogan, Grane, Snåsa and Røros municipalities

2.5.2 Dysglycaemia

Both questionnaire information and non-fasting plasma glucose measurements were used to categorise participants as normoglycaemic, with pre-diabetes or DM. The question about DM was: “Do you have, or have you ever had diabetes?” Those who reported in the questionnaire that they currently have or previously had DM were classified as having DM. Missing answers were regarded as “no”. In addition, a random (non-fasting) plasma glucose measurement was used for ascertaining dysglycaemia. Participants with non-fasting plasma glucose levels of 11.1 mmol/L or higher were also classified as having DM, and those with a level of 7.8–11.0 mmol/L were classified as having pre-diabetes. The remaining participants were categorised as normoglycaemics.

2.5.3 Geographical regions

Four geographical regions were defined: “Region 1” consisted of areas in the inland of Finnmark county, including Karasjok and Kautokeino municipalities. “Region 2” consisted of both inland and coastal areas in Finnmark county, including Porsanger, Tana, and Nesseby municipalities. “Region 3” consisted of coastal areas in Finnmark and the northern part of Troms county, including Lyngen, Storfjord, Kåfjord, Kvænangen, Alta, Loppa, Kvalsund, and Lebesby municipalities. “Region 4” consisted of Marka, Lule, and South Sami areas in southern Troms, Nordland, Nord- and Sør-Trøndelag counties, including Lavangen, Narvik, Evenes, Skånland, Tysfjord, Hattfjelldal, Røyrvik, Namsskogan, Grane, Snåsa, and Røros municipalities (Figure 3).

2.5.4 Statistical analysis

Using the direct method, the European standard population of 2013 was used to age-standardise the prevalence values. Hence, these results may be compared with multiple studies as the European standard population is frequently used as reference. As the outcome variable (dysglycaemia) had three categories (diabetes, pre-diabetes, normoglycaemia), multinomial logistic regression analysis stratified by sex (and separately for four geographic regions) and adjusted for age was applied to determine the age-adjusted odds ratio (OR) for pre-diabetes and DM for Sami compared with non-Sami.

2.6 Paper 2

2.6.1 Study participants

Regardless of ethnic background, all inhabitants aged 40–79 years from the 10 municipalities included in the SAMINOR 2 Clinical Survey were invited to participate. Of the 12,455 invited, 6004 (48.2%) attended the clinical examination. After exclusion of those with uncompleted questionnaire (n=21), missing HbA1c results (n=22), missing ethnicity variable (n=72), and those with T1DM (n=11), 5878 individuals (47.2%) were included in the analyses (Figure 7, Table 2).

2.6.2 Type 2 diabetes mellitus

Information about DM was obtained from both questionnaires and HbA1c results. In the questionnaires, this information came from the question: "Have you ever been diagnosed with diabetes (elevated blood sugar levels)?" The available answers were "yes" or "no". Missing values were classified as "no". If the participant answered "yes", they were asked about the type (T1DM, T2DM, or gestational diabetes). In addition to participants who reported T2DM, those who reported DM without specifying the type (56 participants) were also categorised as having T2DM. Moreover, those who reported having T1DM and reported taking glucose-lowering medication for its treatment (26 participants) or never using insulin (6 participants), were recategorised as having T2DM.

In addition, those with HbA1c ≥ 6.5% were categorised as having T2DM. As virtually all individuals with T1DM are aware of their disease and are under treatment, all those who had high HbA1c (≥ 6.5%) without reporting DM in the questionnaires were regarded as having T2DM. Those who had 5.7% ≤ HbA1c < 6.5% were categorised as having pre-diabetes.

2.6.3 Geographical regions

The 10 municipalities were divided into three different regions: Region 1 consisted of areas in the inland of Finnmark county, including Karasjok and Kautokeino. Region 2 was comprised of both inland and coastal areas in Finnmark county, including Porsanger, Tana, and Nesseby. Region 3 consisted of the remaining municipalities (Evenes, Skånland, Lyngen, Storfjord, and Kåfjord) (Figure 4).

2.6.4 Statistical analysis

Differences in mean age, education, physical activity score, height, weight, WHtR, BMI, and WC by sex and ethnic groups were assessed using two-sample *t*-tests. Self-reported DM and categorised HbA1c were compared between groups using χ^2 tests. The direct method was used to age-standardise the prevalence of pre-diabetes and T2DM. To obtain estimates that better reflect the true prevalence values of T2DM in the selected municipalities and age groups, invitees in the SAMINOR 2 Clinical Survey were chosen as the standard population (age groups: 40–59, 60–69 and 70–79 years). Prevalence of pre-diabetes and T2DM by sex, age, and ethnic groups were presented as percentages with 95% confidence interval (based on normal approximation). Multinomial logistic regression analysis was used to calculate the odds ratio (OR) with 95 % CI of pre-diabetes and T2DM for Sami compared to non-Sami ethnicity stratified by sex and adjusted for age, physical activity, education, BMI, and WHtR.

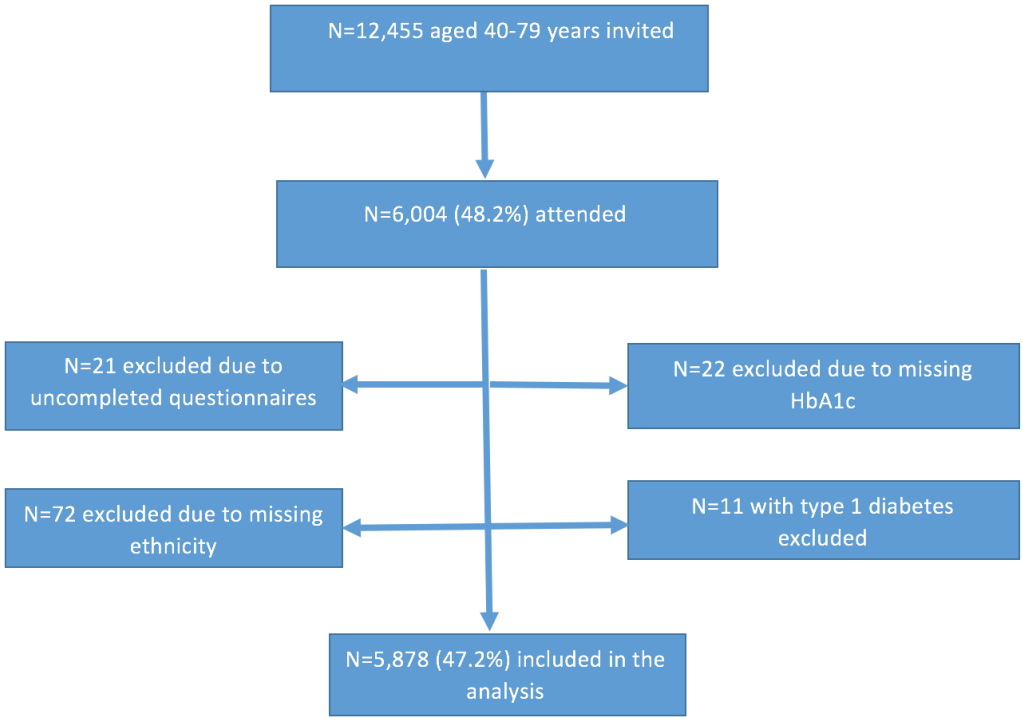


Figure 7. The invited in the SAMINOR 2 Clinical Survey, the participants, exclusions and actual study sample, paper 2

Table 2. Characteristics of the invited (40–79 years), participants, sub-groups, and working samples of paper 2, The SAMINOR 2 Clinical Survey

		Invited (%)	Participated in clinical examinations (%)	Clinical examinations and questionnaire (%)	Included in Paper 2 (%)
Number		12,455	6004	5983	5878
Percent			48.2	48.0	47.2
Sex					
	Men	6469 (52)	2747 (46)	2732 (46)	2688 (46)
	Women	5986 (48)	3257 (54)	3251 (54)	3190 (54)
Age (years)					
	40–59	6810 (54)	2868 (48)	2851 (48)	2800 (48)
	60–69	3589 (29)	2008 (33)	2004 (33)	1966 (33)
	70–79	2056 (17)	1128 (19)	1128 (19)	1112 (19)
Regions*					
	1	2616 (21)	1289 (21)	1288 (22)	1259 (21)
	2	4034 (32)	2011 (33)	2011 (34)	1976 (34)
	3	3605 (29)	1665 (28)	1651 (28)	1625 (28)
	4	2200 (18)	1039 (17)	1033 (17)	1018 (17)
Marital status					
	Married		3401 (57)	3401 (57)	3350 (57)
	Cohabitant		859 (15)	859 (15)	843 (14)
	Divorced		533 (9)	553 (9)	540 (9)
	Unmarried		722 (12)	722 (12)	712 (12)
	Widow(er)		389 (7)	389 (7)	380 (7)
	Missing		80	59	53
Ethnicity					
	Sami		2410 (41)	2410 (41)	2396 (41)
	Non-Sami		3380 (57)	3380 (57)	3365 (57)
	Other		118 (2)	118 (2)	117 (2)
	Missing		96	75	72
Education					
	0–7 years		672 (12)	672 (12)	669 (12)
	8–12 years		2738 (48)	2738 (48)	2697 (48)
	13+ years		2321 (40)	2321 (40)	2262 (41)
	Missing		273	252	250

*Region 1: Karasjok and Kautokeino municipalities;

Region 2: Porsanger, Tana and Nesseby municipalities;

Region 3: Lyngen, Storfjord, Kåfjord, Kvænangen, Alta, Loppa, Kvalsund and Lebesby municipalities;

Region 4: Lavangen, Narvik, Evenes, Skånland, Tysfjord, Hattfjelldal, Røyrvik, Namsskogan, Grane, Snåsa and Røros municipalities

2.7 Paper 3

2.7.1 Study participants

Individuals aged 30 and 36–71 years in SAMINOR 1 from the same 10 municipalities who participated in both the SAMINOR 1 Survey (2003–2004) and the SAMINOR 2 Clinical Survey (2012–2014) were included in the analysis. The two data files were merged by Statistics Norway using the unique 11-digit personal identification numbers assigned to all individuals who live in Norway. The merged file contains individuals born 1933–1968 and 1973 (i.e., aged 30 and 36–71 in the SAMINOR 1 Survey, and 40–41 and 44–79 in the SAMINOR 2 Clinical Survey). Due to collection of data over two calendar years in the SAMINOR 1 Survey and three calendar years in the SAMINOR 2 Clinical Survey, the time span between the two surveys varied from eight to eleven years, with a mean of 10.1 years.

In the SAMINOR 2 Clinical Survey, 12,455 people, aged 40–79 years were invited to take part, and 6004 participated (48.2%). We lack information about those invited to the SAMINOR 2 Clinical Survey who had also participated in the SAMINOR 1 Survey, as a linkage was only allowed for those who participated in both surveys. Therefore, loss to follow-up is described based on the SAMINOR 1 Survey participants who would have been invited to the SAMINOR 2 Clinical Survey, given that they had not died or moved from the 10 studied municipalities prior to invitation to the SAMINOR 2 Clinical Survey. There were 11,558 invitees to the SAMINOR 1 Survey, who, according to their birth year and municipality, would have been invited to the SAMINOR 2 Clinical Survey, given that none had moved or died. Of these, 6450 (55.8%) participated in the SAMINOR 1 Survey clinical examinations, of whom 6408 gave their consent to register linkages.

Among the 6408 individuals, 169 were excluded due to missing initial questionnaire, 2 with missing main questionnaire (containing diabetes information), and 27 with missing ethnicity

information in the SAMINOR 1 Survey. Based on self-report and random (non-fasting) plasma glucose (RPG) ≥ 11.1 mmol/L measurement in the SAMINOR 1 Survey, 260 prevalent cases of DM were excluded. To ensure exclusion of prevalent cases, additionally 75 participants were excluded, as they reported in the SAMINOR 2 Clinical Survey the time of DM diagnosis prior to (n=52), at the same time as (n=6) or during the first two years after participating in the SAMINOR 1 Survey (n=17, two years wash-out period). Of the remaining 5875 persons, 11 were not included in the final analysis due to missing main questionnaire (n=10) or HbA1c measurement (n=1) in the SAMINOR 2 Clinical Survey. A total of 2561 subjects from the SAMINOR 1 Survey did not participate in the SAMINOR 2 Clinical Survey as they died, moved out of the included municipalities during the follow-up period, or were not willing/able to participate. Hence, 3303 individuals were included in the analysis (Figure 8, Table 3).

Table 3. Characteristics of the invited, participants, sub-groups and the final working sample in paper 3. The SAMINOR 1 and 2 Clinical Surveys

		Invited (%)	Participated in clinical examinations and gave consent to linkage (%)	Participated in clinical examinations in both surveys (%)	Included in analysis, Paper 3 (%)
Number		11,558	6408	3624	3303
Percent			55.4	31.4	28.6
Sex					
	Men	6114 (53)	2998 (47)	1586 (44)	1447 (44)
	Women	5444 (47)	3410 (53)	2038 (56)	1856 (56)
Age (years)					
	30	336 (3)	111 (2)	51 (1)	45 (1)
	36–49	4978 (43)	2525 (39)	1329 (37)	1243 (38)
	50–59	3807 (33)	2285 (36)	1425 (39)	1302 (39)
	60–71	2437 (21)	1487 (23)	819 (23)	713 (22)
Marital status					
	Single	3378 (29)	1435 (22)	711 (20)	652 (20)
	Married	6218 (54)	3931 (61)	2364 (65)	2158 (65)
	Widow(er)	440 (4)	266 (4)	146 (4)	126 (4)
	Divorced	1253 (11)	650 (10)	332 (9)	300 (9)
	Separated	268 (2)	126 (2)	71 (2)	67 (2)
	Missing	1	0	0	0
Ethnicity					
	Sami		2464 (38)	1452 (40)	1314 (40)
	Non-Sami		3887 (61)	2145 (59)	1989 (60)
	Missing		57 (1)	27 (1)	0 (excluded)
Education					
	0–7 years		825 (13)	459 (13)	400 (12)
	8–12 years		3180 (50)	1810 (50)	1637 (50)
	13+ years		2058 (32)	1222 (34)	1132 (34)
	Missing		345 (5)	133 (3)	134 (4)

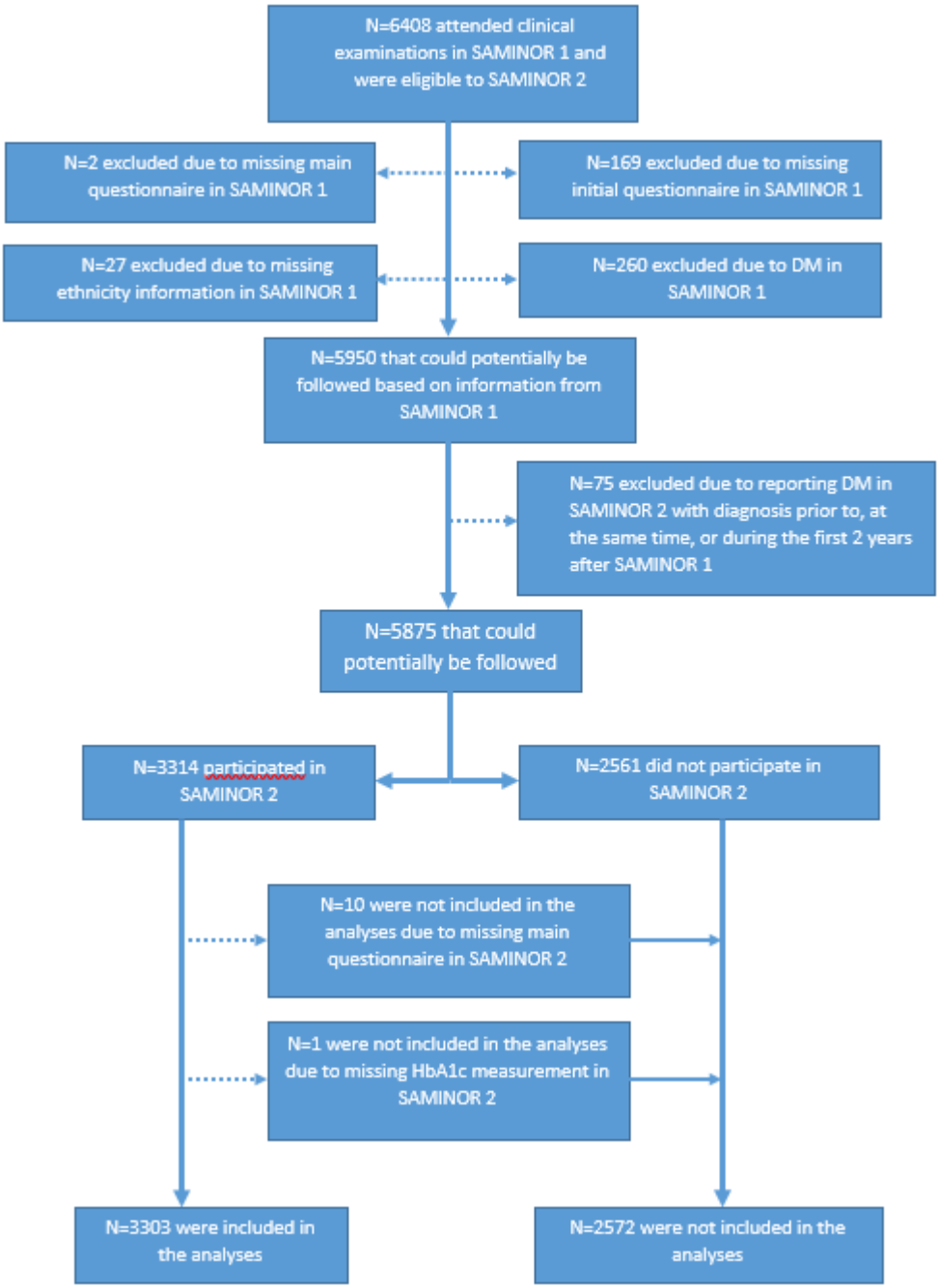


Figure 8. Participants in the SAMINOR 1 Survey, exclusions, those we would want to follow up, and, finally, those who were included in the final analysis and those not, paper 3

2.7.2 Diabetes mellitus

In the SAMINOR 1 Survey, both questionnaire information and random (non-fasting) plasma glucose (RPG) levels were used to categorise participants as having DM. In the SAMINOR 1 Survey, the question concerning DM was: "Do you have or have you had diabetes?" In the SAMINOR 2 Clinical Survey, the question was: "Have you ever been diagnosed with diabetes (elevated blood sugar levels)?" The available answers were "yes" or "no". Missing values were classified as "no".

All those who in the SAMINOR 1 Survey reported that they had DM as well as those with RPG levels ≥ 11.1 mmol/l were excluded from the analyses (They were prevalent cases). In addition, and as discussed above, those who in the SAMINOR 2 Clinical Survey reported the time of DM diagnosis as prior to, at the same time as or during the first two years after participating in the SAMINOR 1 Survey (wash-out period) were regarded as prevalent cases of DM in the SAMINOR Survey and were excluded from the final analysis. Thus, the follow-up time was around eight years.

In the SAMINOR 2 Clinical Survey, self-report and glycated haemoglobin (HbA1c ≥ 6.5%) were used to identify incident cases of DM. By dividing the number of incident cases of DM in the SAMINOR 2 Clinical Survey by the DM-free individuals in the SAMINOR 1 Survey, the 8-year cumulative incidence of DM was calculated.

2.7.3 Risk factors of type 2 diabetes mellitus

At the entrance of the SAMINOR 1 Survey, some potential risk factors for T2DM like age, BMI, WC, WHtR, family history of DM, marital status, education level, smoking, alcohol consumption, mental distress (Hopkins symptom checklist (SCL-10 score)), physical activity, and gross family income were measured or asked about. The levels of these risk factors were

compared in those who were finally included in the analysis with those who were not included. The same was done for Sami versus non-Sami participants in the final analysis.

2.7.4 Statistical analysis

Logistic regression analysis was used to assess the effect of ethnicity as well as available and relevant risk factor variables on the development of DM in men and women.

3 Summary of the results

3.1 Paper 1

Of the 15,208 participants in the SAMINOR 1 Survey included in the study sample, 696 (4.6%) were defined as having DM, and 426 (2.8 %) as having pre-diabetes. Among those defined as having DM, 636 (91.4%) reported DM in the questionnaire, whereas 60 (8.6%) were identified only by non-fasting plasma glucose.

Little or no ethnic difference was observed in the total age-standardised prevalence of pre-diabetes or DM in either sex. Total age-standardised prevalence of pre-diabetes and DM for Sami men was 3.4% and 5.5%, respectively. Corresponding values for non-Sami men were 3.3% and 4.6%. Total age-standardised prevalence of pre-diabetes and DM for Sami women was 2.7% and 4.8%, respectively, while corresponding values for non-Sami women were 2.3% and 4.5%.

In Region 1, the age-adjusted odds of having DM was significantly lower among Sami men than among non-Sami men (OR=0.29, 95% CI=0.10–0.82). The same was observed for Sami women in Region 2 (OR=0.46, 95% CI=0.23–0.91). In Region 4, the situation was opposite, with the age-adjusted odds for DM being significantly higher in both Sami men and women than in their non-Sami counterparts [OR=2.87 (95% CI=1.63–5.06) for men and OR=2.38 (95% CI=1.28–4.43) for women]. Odds for pre-diabetes was also significantly higher for Sami men compared to non-Sami men in this region (OR=2.05, 95% CI=1.06–3.96)

3.2 Paper 2

Based on self-report and/or HbA1c-measurements, a total of 2083 (35.4%) individuals were defined to have pre-diabetes and 565 (9.4%) to have T2DM in the SAMINOR 2 Clinical Survey. Of those who were categorised as having T2DM, 465 (82.3% of all cases) reported T2DM or elevated blood sugar levels in the questionnaire. The total age-standardised prevalence of pre-diabetes and T2DM were, respectively, 34.1% (95% CI: 33.1–35.1) and 8.7% (95% CI: 8.0–9.4).

In men, the total age-standardised prevalence of pre-diabetes (37.9% vs 31.4%) and T2DM (10.8% vs 9.5%) were higher in Sami compared with non-Sami. When adjusting for age as a continuous variable in a multinomial logistic regression analysis, the ethnic difference was statistically significant for both pre-diabetes (OR 1.42, 95 % CI: 1.20–1.68) and T2DM (OR 1.31, 95% CI: 1.01–1.70).

In women, pre-diabetes (36.4% vs 33.5%) and T2DM (8.6% vs 7.0%) were also more prevalent in Sami than non-Sami. The ethnic differences in both pre-diabetes (OR 1.20, 95% CI: 1.02–1.41) and T2DM (OR 1.38, 95% CI: 1.05–1.82) were also herein statistically significant.

Adjustment for WHtR had the largest impact on the OR for pre-diabetes and T2DM for Sami compared to non-Sami, especially in women; after adjusting for WHtR, the OR for pre-diabetes in women was 1.05 (p=0.59) and for T2DM 1.00 (p=1.00).

In men, the observed prevalence of pre-diabetes and T2DM was higher in Sami in all geographical regions; statistically significant ethnic difference was, however, only found for pre-diabetes in region 2 and for T2DM in region 3. In women, the observed prevalence of pre-diabetes and T2DM was higher in Sami in all geographical regions but region 2, wherein

fewer Sami had dysglycaemia. Statistically significant ethnic differences were, however, only observed for pre-diabetes in region 1 and for T2DM in regions 1 and 3.

3.3 Paper 3

A total of 201 incident cases of DM were identified in the SAMINOR 2 Clinical Survey, based on self-report (n=138) or HbA1c≥6.5% (without self-report) (n=63). All who reported DM had HbA1c ≥ 6.5%. This number (n=201) corresponds to a 6.1% (95% CI: 5.3–6.9) 8-year cumulative incidence of DM. No statistically significant difference in the 8-year cumulative incidence of DM was found between Sami and not-Sami of the same sex.

The age-adjusted logistic regression analysis showed no statistically significant difference between Sami and non-Sami in the odds for DM in men or women. Further adjustments for other risk factors of DM confirmed that there were no ethnic differences in the odds of contracting DM. BMI, waist circumference (WC) and waist-to-height ratio (WHtR) were, however, statistically significant risk factors for DM in both sexes (adjusted for age and ethnicity).

4 Discussion

4.1 Methodological considerations

The first two articles had cross-sectional design, and the last one had a longitudinal design. Cross-sectional studies, which are conducted in a population at a specific time and place, measure disease frequency and factors which may cause diseases, or both, simultaneously [180]. The primary output of cross-sectional studies is prevalence data, although associations between risk factors and diseases can be sought and tested [180]. Cross-sectional studies usually serve as hypothesis-generating studies and inferring any causal relationship between the exposure and the outcome may be challenging, as the proper temporal sequence of events needed to establish causality cannot be observed due to the design of the study [180]. If a cross-sectional survey is followed up for a defined period of time to measure a health outcome, this study can then be a cohort study (longitudinal study) [180]. Paper 3 was a longitudinal study and tried to measure the cumulative incidence (risk) of DM. Similar to cross-sectional studies, longitudinal studies provide associations between risk factors and disease outcomes and inferring causal relationship between exposure and outcome may be problematic. Contrary to cross-sectional studies, in longitudinal studies risk factors are measured prior to the occurrence of the outcome, hence the risk of temporal bias (reverse causation) is reduced. It is unlikely that ethnicity, place of living, or education being affected by DM. However, it is possible that after a person is diagnosed with either pre-diabetes or DM, they apply lifestyle advices and change their dietary and physical activity habits. Furthermore, DM can lead to weight loss and in many instances, the only reason for seeking medical evaluation is unexplained weight loss. Consequently, BMI, WC, WHtR and physical activity are all subject to temporal bias in cross-sectional studies.

The ethnicity as an exposure variable in itself, is rarely a cause of a disease, but it is rather an index of some other related variables such as cultural (perception of illness and wellbeing), socioeconomic (education, profession, income), or lifestyle-related (diet, physical activity, smoking, alcohol drinking) factors [149]. The definition of ethnicity concept has always been a matter of contention, as ethnicity is a culturally constructed concept rather than being based on objective differences [181]. When it comes to Sami ethnicity in literature, there have been various definitions [177, 182, 183]. While some definitions pay especial attention to linguistic features [177], the core in the Sami definition in the present thesis is self-perception. According to the applied definition of Sami in this thesis, Sami is a person who responded that they either considered themselves to be Sami or reported to have a Sami ethnic background, and, in addition, at least one of their grandparents, parents, or they themselves spoke a Sami language at home. All participants who did not meet this criterion were defined as non-Sami. As many Sami people lost their Sami language during decades of assimilation and Norwegianisation policies, the linguistic features are not central in this definition. This definition is not mutually exclusive, which means that a Sami person may have a sense of self-affiliation to other ethnic groups as well as Sami ethnicity or speak other languages at home as well. The Sami features are regarded dominant in the present study. While an ethnic Norwegian person hardly presents affiliation to Sami ethnicity, it is likely that a Sami has a sense of affiliation to both Sami and Norwegian identity. It is the sense of belonging to an ethnic group that brings about behavioural and cultural differences and can expose a person for or protect them against health/disease determinants. As mentioned before, self-perception ethnicity is subject to change with time and some participants who considered themselves as Sami in the SAMINOR 1 Survey did not do so in the SAMINOR 2 Clinical Survey and vice versa.

As sensitive questions like those which may be construed as intrusive or invasive might produce lower participation rates, higher item non-response rates, or lower response accuracy [184], this was taken into account in the design and phrasing of the questions in the questionnaires. Cultural, social and historical attributes of the target society were also taken into consideration in the study design especially in the forming of questionnaires and during conduction of the surveys. To lessen the language barrier for Sami people, the questionnaires were provided in Sami languages, although only a few percent of Sami participants preferred to complete the Sami version of questionnaires.

4.2 Validity

Validity is the ultimate goal of any epidemiological study. Validity can be regarded as lack of bias and a study is called valid when it measures what it is supposed to measure correctly and without any distortion or deviation [185]. While *internal* validity indicates that the provided results are correct for the source population of the study sample, *external* validity ensures that the provided results are generalisable to other groups who were not included in the study [185]. There are two main types of errors, which can occur at virtually any step of the research process: systematic errors and random errors. While systematic errors, i.e. bias, affect comparison between groups, random errors affect the reliability (reproducibility) of the measurements and the precision of the estimates [186]. To control systematic errors, one should properly design the study, and to decrease random errors one could increase the sample size and apply good scientific techniques [186]. As error and bias cannot be fully controlled and avoided in epidemiological studies, the most important need is for systematic, cautious and critical interpretation of data and results [187].

Bias can create false patterns and misjudgements (either differences where none exist or failure to detect present differences) [187]. As mentioned above, to achieve high validity, one should avoid bias, which is categorised into three main types: selection bias, information bias, and confounding [188]. These issues are to be discussed here with respect to the conducted study.

4.2.1 Selection bias

Selection bias occurs when individuals have different probabilities of being included in the study groups according to relevant study characteristics, i.e. the exposure or outcome of interest [185]. This kind of bias creates erroneous prevalence estimates and distorted measure of association between exposure and outcome. As the included individuals are not representatives of the entire study population, the results are not generalisable to the study population or other groups [187].

4.2.1.1 Paper 1

Paper 1 is based on data from inhabitants of 24 municipalities included in the SAMINOR 1 Survey. Having a 56% participation rate in the SAMINOR 1 Survey might be a source of selection bias. It is not certain that non-participants had the same characteristics as the participants. Participation rates were different from county to county and from municipality to municipality, which to some extent is due to different designs in the recruitment phase. For example, inhabitants of some municipalities received a reminder and non-responders got the chance to attend when the buses returned a couple of months later.

Table 1 presents some characteristics of the invited, participants and working samples in paper 1 according to sex, age, regions, marital status, ethnicity, and education. The non-participants tended to be men, young and single. In addition, there are numerous other important features of non-responders, which are not known to us. Therefore, it is not possible to rule out the possibility of selection bias. Lack of ethnic registry in Norway made it impossible to determine whether the ethnic composition of the participants in the SAMINOR surveys reflected that of the actual population in our geographical regions. The fact that the SAMINOR Study might have been deemed as being primarily directed towards Sami people might have deterred the non-Sami inhabitants of the included municipalities to participate in

the survey. On the other hand, the history of study misconduct and abuse of participants in so-called scientific studies conducted throughout the periods when Sami people were regarded as an inferior race might be a source of concern and reluctance from Sami people to participate. As long as the participants in the survey are representatives of their respective ethnic groups, possible different participation rates in different ethnic groups would not affect the estimated prevalence of DM in each ethnic group and the odds ratio of DM. However, it might reduce the power of the study to show possible differences in the prevalence of DM. If participation rate, however, depends on both the exposure variable (ethnicity) and the outcome variable (DM), both the estimated prevalence of DM in each ethnic group and the odds ratio of DM would be distorted. In this case, the real prevalence of outcome variable (DM) in each ethnic group would also be a determinant of the total participation rate in that group.

To enhance the participation rate, Sami people themselves were involved in almost all aspects of the SAMINOR Study and several information sessions were held in the municipalities before, during and after study performance. The participants were ensured that the questionnaires were anonymised and personal and sensitive information of participants were to be safeguarded. Participants were also ensured right to withdraw from the study at any given point of time.

It is likely that those who are more conscious about their health (usually those with higher socioeconomic status) are more interested in participating in health studies like SAMINOR [189]. On the other hand, the severely ill and disabled may not be able to participate. This selection bias may lead to underestimation of the prevalence of DM.

In spite of relatively large number of included municipalities (n=24) and participants, generalisation of the results to Sami and non-Sami inhabitants in other regions is not advisable.

4.2.1.2 Paper 2

Paper 2 is based on data from the SAMINOR 2 Clinical Survey. As participation rate here is just under 50%, the chance of selection bias due to non-response is even higher than in the SAMINOR 1 Survey. Table 2 presents some characteristics of the invited and participants in paper 2. Like in paper 1, non-participants tended to be younger and male. Apart from that, there is little information at hand regarding non-participants in our surveys. Similar to our survey, in the Tromsø 2 Study, it was reported that non-participants were over-represented among young unmarried men [190] with increased mortality rates [191]. Of the 50,807 invited in the HUNT 3 Study (2006–2008), 54% participated [189]; it was revealed that the prevalence of diseases like cardiovascular diseases, DM, and psychiatric disorders was higher among non-participants. In addition, registry data revealed that the non-participants had lower socioeconomic status and higher mortality rate [189]. If this was the case in our surveys, it can be assumed that the prevalence of T2DM is underestimated. Nonetheless, it is not known if non-participation due to the mentioned factors affected Sami and non-Sami subjects equally or not. Selection bias due to non-participation is in most cases a greater threat to the validity of prevalence estimates than to the validity of the associations between exposure and outcome [192].

As the number of included municipalities in the SAMINOR 2 Clinical Survey was limited (n=10), the participants may be considered representative for the rural, Sami and non-Sami population in included municipalities in Finnmark and Troms counties. However, generalisations to the entire Sami or non-Sami populations in Norway are not advised.

4.2.1.3 Paper 3

This paper is a longitudinal study following participants in the SAMINOR 1 Survey up to the SAMINOR 2 Clinical Survey. From the 10 municipalities, which were included in the SAMINOR 1 Survey, 5875 participants could potentially be followed up to the SAMINOR 2 Clinical Survey. Of 5875 eligible individuals, 3303 (56.2%) were included in the analyses (Figure 8). Dropouts might be due to death, emigration, debilitating diseases, or conscious choice not to participate in the follow-up study. Loss-to-follow-up (attrition or censoring) is a source of selection bias if those who were eligible to participate in the SAMINOR 2 Clinical Survey but did not do so had different risk profile than those who participated in the SAMINOR 2 Clinical Survey. In fact, loss-to-follow-up should be independent of the outcome [185].

Table 4 presents some characteristics of individuals we were able to follow-up compared to those who were not followed up. Although some differences in the marital status, smoking, mental health score, yearly gross income of the household and leisure-time physical activity were found between the two groups, main risk factors for DM including age, obesity indices (BMI, WC, and WHtR) and family history of DM were not markedly different.

Linkage of data from the SAMINOR 1 Survey and the SAMINOR 2 Clinical Survey was done only for those who participated in both surveys and gave consent to linkage, so it was not feasible to keep track of those who were censored during the follow-up. Our dataset was not linked to the Cause of Death registry, so we do not have direct information about the number and death cause of those who died during the follow-up period. We do not expect that there were many participants who got DM during the follow-up period and died of the disease itself or its late complications.

Table 4. Characteristics of individuals we were able to follow-up compared to those who were not followed up among those who participated in SAMINOR 1 (2003–2004) and were eligible[1] for SAMINOR 2 (2012–2014), by sex (N=5875). Numbers are mean (standard deviation) for continuous variables (age, body mass index, waist circumference, and waist-to-height ratio) and percent (number of subjects) for categorical variables (family history of DM, married, education>12 years, SCL-10 score>1.85, alcohol, low-income, and inactive).

	Included in the follow-up analysis	Not followed up	p-value
Men	N=1447	N=1307	
Age (year)	52.4 (8.7)	51.2 (9.8)	<0.01
Body mass index (kg/m^2)	27.5 (3.5)	27.6 (4.2)	0.42
Waist circumference (cm)	92.3 (9.3)	93.0 (10.9)	0.07
Waist-to-height ratio	0.534 (0.054)	0.537 (0.064)	0.10
Sami ethnicity (%)	40.2 (581)	32.7 (866)	<0.01
Family history of DM[2] (%)	19.4 (280)	18.2 (238)	0.44
Married[3] (%)	64.5 (933)	52.8 (690)	<0.01
Education>12 years (%)	32.8 (458)	30.7 (381)	0.26
SCL-10 score[4] >1.85 (%)	5.3 (72)	9.5 (114)	<0.01
Current smoker[5] (%)	28.8 (416)	39.5 (516)	<0.01
Alcohol[6] (%)	30.7 (444)	31.1 (407)	0.80
Low-income[7] (%)	57.0 (825)	61.5 (804)	0.02
Inactive[8] (%)	18.8 (272)	23.1 (302)	0.01
Women	N=1856	N=1265	
Age (year)	51.6 (9.0)	50.7 (10.1)	<0.01
Body mass index (kg/m^2)	27.4 (4.6)	27.6 (4.9)	0.38
Waist circumference (cm)	84.0 (11.2)	84.2 (11.8)	0.08
Waist-to-height ratio	0.526 (0.074)	0.527 (0.076)	0.40
Sami ethnicity (%)	39.5 (733)	29.4 (372)	<0.01
Family history of DM[2] (%)	23.2 (430)	21.8 (276)	0.38
Married[3] (%)	66.0 (1225)	58.2 (736)	<0.01
Education>12 years (%)	38.0 (674)	36.3 (428)	0.34
SCL-10 score[4] >1.85 (%)	8.4 (141)	11.5 (130)	<0.01
Current smoker[5] (%)	30.6 (568)	40.9 (517)	<0.01
Alcohol[6] (%)	19.7 (365)	20.5 (259)	0.58
Low-income[7] (%)	58.7 (1090)	62.7 (793)	0.03
Inactive[8] (%)	19.1 (355)	22.9 (289)	0.01

1) Living in the 10 SAMINOR 2 municipalities at time of SAMINOR 1 with relevant year of birth
2) Those who had at least one with DM among father, mother, siblings or children
3) Married vs single, widow/widower, divorced, or separated
4) SCL-10 score: Hopkins symptom checklist score
5) Current smokers vs former smokers or never-smokers
6) Drinking alcohol at least once a week
7) Yearly gross income of the household less than 451,000 Norwegian Kroner
8) Leisure-time activities include reading, watching TV or other sedentary activities

If loss-to-follow happened due to diseases, which share risk factors with the outcome of interest (like cardiovascular diseases and DM), the risk of DM would be underestimated (competing risks). Cardiovascular diseases can be a complication of DM, but there is not a one-to-one correspondence between cardiovascular diseases and DM. In fact, most cases of cardiovascular diseases occur independently of DM and not all who get DM die of cardiovascular diseases. According to the Norwegian Institute of Public Health, cancers, not cardiovascular diseases, are the leading cause of death in people with similar age-span as our participants [193]. Based on numbers from Statistics Norway, one can expect around 330 deaths from year 2001 until 2010 (10 years) in a group of 5875 individuals with similar age-span and age-distribution as our participants (calculations not shown) [194]. Competing risks become more important with the increasing age of the population under study (increased risk of multimorbidity). As the mean baseline age of both those who were followed up and those who were not was around 52 years, and there were relatively few expected deaths (a total of 330 deaths), it is not expected that competing risks have substantially affected our estimate of the cumulative incidence of DM. Furthermore, studies have shown minimal or no difference between Sami and non-Sami individuals in the distribution of risk factors for cardiovascular diseases and/or the risk of acute myocardial infarction or cerebral stroke; hence, the relative risk of DM (between Sami and non-Sami) was also not considerably distorted [141, 195].

In Kautokeino and Karasjok, where a large share of the population is involved with reindeer husbandry, the SAMINOR Clinical Survey was conducted in winter-time, to avoid seasons when many Sami people would be out of their main living place due to reindeer husbandry. We do not have information on the participants in the SAMINOR 1 Survey, who due to moving to other regions, were not included in the final analysis, but they are expected to be few, and it is unlikely that they had any impact on the conclusions.

4.2.2 Information bias

Information bias (e.g., measurement error) occurs when information collected about and from study subjects is erroneous [185]. This type of bias leads to a person or population subgroup being put into the wrong category (misclassification) [187]. When the misclassification is random and independent of any other variable, it is called *non-differential* misclassification. When the mismeasurement (error) affects subgroups unequally, it is called *differential* misclassification [187]. Put in other words, when misclassification in the outcome variable depends on the exposure status or vice versa, the misclassification is differential [186].

Recall bias resulting from inaccurate recall of past events is a common source of misclassification in cross-sectional and case-control studies. This bias (differential misclassification) occurs as comparison populations or subgroups (e.g. diseased individuals) unequally recall and report outcome-related events and/or exposure to various risk factors [186].

Ethnicity was the exposure variable (or proxy of exposure) throughout the thesis and is more likely to be wrongly reported by Sami people than non-Sami. Reporting Sami ethnicity needs more conscious choice than reporting Norwegian ethnicity and the majority of people regard Norwegian as the default ethnicity. A Sami person might intentionally or unintentionally report his/her ethnicity as Norwegian. Intentional misreporting happens if the person has a sense of inferiority by being identified as Sami and it is not unimaginable taking the long history of stigmatisation and assimilation of Sami people into consideration. Unintentional misreporting of Sami ethnicity by a Sami person can occur if the person is not aware that they had a Sami-speaking grandparent or parent or they had a Sami ethnic background. Some Sami people might have misunderstood the question “What do you consider yourself to be?” as a question about their citizenship and in spite of answering positively to all other questions

regarding the Sami ethnicity, they reported that they considered themselves as Norwegians. Although according to our definition of Sami ethnicity, this person was regarded as Sami, if other answers were also erroneous, the person would not be categorised as Sami. As mentioned before, a high agreement was observed between Sami ethnicity classifications in the SAMINOR 1 and the SAMINOR 2 Clinical surveys.

We have to assume that misclassification in the exposure variable (ethnicity) does not depend on the outcome variable status (like e.g., dysglycaemia), therefore the misclassification is non-differential. As alternative definitions of Sami ethnicity produced quite similar results (results not shown) and considering that the participants were provided with comprehensive instructions regarding the meaning of questions and how to fill out the questionnaires, we do not think that this misclassification was of great importance in the present thesis.

DM in most situations is a chronic and life-long disease without a cure (except gestational diabetes). Contrary to T1DM where the patient would not survive without diagnosis and treatment, there are always a proportion of those with T2DM who are not aware of their disease and consequently do not report it in the questionnaires. As well as the disease itself, related details like the exact date of diagnosis, type of DM (especially gestational diabetes), medications (if prescribed), family history of DM, or risk factors for T2DM (e.g. unhealthy diet, low physical activity) might be reported imprecisely. A qualitative study argued that Sami people tend to underreport their diseases due to some cultural differences (different conceptualisation of diseases) [196]. If this was the case for our Sami participants, this might have led to underestimation of the prevalence of DM among them (differential misclassification).

As mentioned previously, the questionnaires were in some municipalities available in both Norwegian and Sami languages, and people in the age group 70–79 years were given

questionnaires with fewer questions and larger font size. Nonetheless, there are many elderly Sami who have difficulty reading and writing both Norwegian and Sami. In both the SAMINOR 1 and the SAMINOR 2 Clinical surveys, participants were offered help in filling out the questionnaire if they requested. Therefore, the probability of misclassification due to linguistic issues is negligible.

The validity of the questionnaires in the SAMINOR 1 and SAMINOR 2 Clinical surveys was not assessed, but according to literature, questionnaires are a valid and reliable source of acquiring information about prevalent, known cases of DM. According to the first HUNT Study performed in North Trøndelag, Norway, the sensitivity and PPV of applied questionnaire for self-reported DM were 99.4% and 96.4%, respectively [197]. A Dutch study reported no ethnic difference in the accuracy of self-reported DM when comparing Dutch patients with patients who were first-generation immigrants, mostly from Turkey and Surinam [198]. In a study conducted in Olmsted county, Minnesota, with 2037 participants aged ≥ 45 years, the sensitivity and PPV of self-reported DM were 66.0% and 94.3%, respectively [199]. The French national study of CADEUS reported a sensitivity and PPV of self-reported DM as 86.7% and 73.4%, respectively [32]. The Finnmark study, which applied a quite similar questionnaire to the questionnaire applied in our surveys, reported 66% agreement between positive answers to DM and medical records [200]. Of 33 participants who had reported DM at Finnmark 1 Study, 24 (73%) did so at Finnmark 2 Study conducted three years later. The test-retest reliability of self-reported T1DM and T2DM diagnoses was assessed between three self-administered questionnaires applied in Norwegian Women and Cancer Study (the NOWAC Study). According to the authors, the Cohen's kappa for T1DM was ≥ 0.73 in the 1991–2005 and the 1998–2005 test–retest studies, and 0.83 in the 1991–1998 test–retest study [201]. The kappa for T2DM was reported moderate (0.57) in the 1991–2005 test–retest study and high (≥0.66) in the 1991–1998 and 1998–2005 test–retest studies

[201]. All the above-mentioned studies applied medical records as reference standard. The validity of a questionnaire might vary from population to population and factors like age, education level, and health status of participants as well as phrasing of the question(s) might affect the performance of a given questionnaire. As mentioned before, the two main questions regarding DM were not identical in the SAMINOR 1 and the SAMINOR 2 Clinical surveys. Contrary to the SAMINOR 1 Survey, the question about DM was followed by questions about the type of DM in the SAMINOR 2 Clinical Survey. Furthermore, in the SAMINOR 2 Clinical Survey, "elevated blood sugar levels" was added in parentheses. This may have lead to more people answering "yes" to the question. This may render a different validity for both questionnaires.

Non-differential misclassification is more subtle and may have little or no effect on the final prevalence figure [187]. If non-differential misclassification is present when relating the (dichotomous) outcome of interest (DM in the present thesis) and the (dichotomous) exposure variable (ethnicity in the present thesis), the strength of association is always underestimated, so the problem is failing to find associations, which, in reality, are present [186, 187]. Misclassifications that affect confounding factors tend to have unpredictable effects [187].

The misclassification of ethnicity (the same definition in all papers) and self-reported DM is already discussed; thus in the rest of this section, misclassification of dysglycaemia (pre-diabetes and/or diabetes mellitus) will be discussed under the respective papers.

4.2.2.1 Paper 1

In paper 1, random plasma glucose (RPG) with cut-off 7.8 mmol/L and 11.1 mmol/L was used to categorise pre-diabetes and DM, respectively. Random plasma glucose in the presence of classic symptoms of hyperglycaemia or hyperglycaemic crisis is recommended by the ADA for diagnosis of DM [3]. In the SAMINOR Study, we did not perform medical

examination to find DM symptoms, so we relied on RPG values without knowing about classical DM symptoms to recognise DM. Neither did we carry out OGTT test, so we used RPG values (7.8–11.0 mmol/L) instead of 2hpp test results to categorise pre-diabetes.

The RPG levels are affected by the natural fluctuations of blood glucose throughout the day and the mentioned criterion can only detect DM that is poorly controlled [202]. Paper 1 used data from 24 municipalities included in the SAMINOR 1 Survey. In the SAMINOR 1 Survey neither other laboratory tests (e.g. fasting plasma glucose, 2hpp [glucose tolerance test] or HbA1c) nor medical examination was used to ascertain DM. The applied cut-offs have very low sensitivity to identify pre-diabetes and DM [86]. This is reflected in the low percentage of diagnosed cases using this cut-off (only 8.6% of all ascertained DM cases). Furthermore, the overall prevalence of pre-diabetes and DM was reported as 2.8% and 4.6%, respectively. We assume that the real prevalence of pre-diabetes and DM was underestimated in paper 1. In this paper, no overall difference in the prevalence of pre-diabetes and DM was found between Sami and non-Sami of the same sex. As mentioned before, non-differential misclassification in the outcome variable inevitably attenuates the strength of association between the exposure and the outcome [187]. Although same RPG cut-offs were applied to both ethnic groups, we cannot rule out any ethnic difference in the prevalence of pre-diabetes and/or DM in the entire survey region.

4.2.2.2 Paper 2

The choice of HbA1c cut-off of 6.5% was based on recommendation from the ADA [3] and Norwegian medical guidelines [81]. Numerous publications have reported different performance of HbA1c at different cut-offs [34, 96-98, 203-206]. According to the Tromsø OGTT Study, HbA1c cut-off at 6.5% gives a sensitivity and PPV of 34.7% and 41.2%, respectively (OGTT as reference test, prevalence=5.7%) [96]. It is worth mentioning that

there is no perfect test (gold standard) for diagnosing DM and each test has its strengths and limitations and can capture different groups of patients [101]. Although the mentioned sensitivity and PPV could yield a comparable number of false positive and false negative cases, which could compensate for each other to give a realistic prevalence of DM, the relatively low sensitivity and PPV lead to a large misclassification of T2DM. This misclassification is non-differential as it affects both ethnic groups equally. Nonetheless, as mentioned before, any non-differential misclassification in the outcome variable attenuates the strength of association between exposure (ethnicity) and outcome (T2DM). Despite the misclassification, a statistically significant ethnic difference in the prevalence of pre-diabetes and T2DM was observed in paper 2. It implies that the real ethnic difference in the prevalence of pre-diabetes and T2DM might be larger than what was observed. The same applies to other risk factors for T2DM like male sex, (general and abdominal) obesity, low education and low physical activity. The non-differential misclassification in the outcome variable might have diluted the association between each risk factor and the outcome so that the OR is underestimated and no longer statistically significant. Of the mentioned risk factor variables, misclassification was more likely to affect physical activity, as it was not measured objectively. It is not clear if the misclassification in physical activity was differential or non-differential. As mentioned before, misclassification in the confounding variable may have unpredictable effect on the measure of association between exposure and outcome [187]. Another source of (non-differential) misclassification in this paper was that all those who had HbA1c $\geq$ 6.5% (without mentioning T2DM in the questionnaire) were classified as having T2DM. New research have shown that a considerable proportion (approximately 10–15%) of those who get DM in adulthood and were previously diagnosed as T2DM have indeed Latent Autoimmune Diabetes of Adults (LADA) [207].

Unlike paper 1, Sami people had higher prevalence of pre-diabetes and T2DM in the majority of geographical regions (stratified by sex) in paper 2. This makes it less likely that the observed ethnic difference is a chance finding. Furthermore, regarding the non-differential misclassification in the outcome variable, it is more likely that the real ethnic differences were even larger than what was observed. As for paper 1, the relatively small number of participants in each geographical region should be taken into account before making any inference in this regard.

4.2.2.3 Paper 3

The same criterion for categorisation of incident cases of DM was used in this paper (self-report and/or HbA1c ≥ 6.5%) as in paper 2; thus the same issues with non-differential misclassification of DM cases due to low sensitivity and PPV of HbA1c test affect the strength of association between the exposure and outcome. Another source of misclassification is to classify prevalent cases of DM as the incident ones. We excluded those who reported the time of DM diagnosis during the first two years of follow-up after the SAMINOR 1 Survey (two years wash-out period). Nevertheless, regarding the low sensitivity of RPG ≥ 11.1 mmol/L to ascertain those with DM, there is still possibility that some prevalent cases of DM were classified as incident cases of DM.

Besides small sample size, the expected non-differential misclassification in the outcome (DM) variable (as well as in the exposure variable) might be possible explanations for lack of statistically significant ethnic difference in the 8-year cumulative incidence of DM.

4.2.3 Confounding, over-adjustment, and residual confounding

The term *confounding* refers to a situation in which a non-causal association between a given exposure and an outcome is observed due to the impact of a third variable (or group of variables), usually known as confounding variable(s) [208]. The confounder is defined as a variable, which is causally associated to the outcome and causally or non-causally associated with the exposure, but is not part of the causal pathway between the exposure and the outcome (intermediate variable) [208]. The potential for confounding is present whenever the cardinal rule "compare like-with-like" is broken [187]. Put in other words, when the comparison groups differ in characteristics other than the risk factor under study [187]. Confounding is of particular importance in differentiating between causal and non-causal (pure association) relations [187]. The confounding effect can be controlled for through various ways like randomisation (randomised controlled trials), matching (case-control studies), selecting comparable groups or restriction entry into study (e.g. same sex, age or socioeconomic status), stratification (e.g. by sex or age), adjustment (multiple regression analyses) and standardisation (directly or indirectly) [187].

In dealing with confounders, one should be aware of two pitfalls; *over-adjustment* and *residual confounding*. Over-adjustment occurs when adjustment is inadvertently carried out for a variable that either lies in the causal pathway between the exposure and the outcome or is so strongly related to either the exposure or the outcome that their true relation is distorted [209]. Over-adjustment may obscure a true effect or create an apparent effect which does not exist [210].

Residual confounding occurs when adjustment does not completely remove the confounding effect due to a given variable or set of variables [211]. The common sources of residual confounding are [211]:

1) Improper definition of the categories of the confounding variable
2) The variable used for adjustment is an imperfect marker of the condition or characteristic the investigator wishes to adjust for
3) Failure to adjust for other important confounders
4) Misclassification of confounding variable

4.2.3.1 Paper 1

Stratified by sex, we compared Sami and non-Sami aged 36–79 years inhabiting the same rural districts of Northern and Mid-Norway. The principle of comparing comparable people with each other is the cornerstone of the study design (the SAMINOR Study) and applies to all the papers. The total prevalence of pre-diabetes and DM among Sami and non-Sami men and women were age-standardised using European standard population of 2013. All analyses were stratified by sex and adjusted for age in the multinomial logistic regression analysis as sex and age are two known confounding factors. There were other important confounding variables, which were not adjusted for in the multinomial logistic regression, as they had not been measured precisely. Dietary habits and physical activity, for example, are very hard to objectively and precisely measure especially in the years prior to the DM diagnosis. Education is usually used in studies as a proxy for socioeconomic status, but education is not a perfect surrogate for socioeconomic status and was not considerably different between the two ethnic groups. Temporal bias was a great obstacle in this regard, as DM occurrence could negatively affect the income and physical activity of a person. Although weight and height (to measure BMI, WC and WHtR) had been objectively and reliably measured, they were not adjusted for in the multinomial logistic regression analysis as it was argued that DM and obesity were firmly related together and both were parts of metabolic syndrome. Occurrence of T2DM may affect obesity either through insulin resistance and resultant weight loss or through conscious changes in lifestyle. This may lead to temporal bias assessing the effect of

obesity on the development of T2DM. Furthermore, it was argued that obesity was on the causal pathway to DM (intermediate variable). Nonetheless, it may be argued that obesity should have been accounted for owing to the fact that it is a source of residual confounding. Obesity is neither a necessary nor a sufficient cause of DM and by adjustment for it, one can assess the effect of the exposure on the outcome which goes through obesity.

4.2.3.2 Paper 2

Participants were 40–79 years old inhabitants of 10 rural municipalities in Northern Norway. Age-standardisation of the prevalence values of pre-diabetes and T2DM was carried out based on the invited individuals in the SAMINOR 2 Clinical Survey. Prevalence values and analyses were all stratified by sex. In the multinomial logistic regression analysis age, education, BMI, WHtR and physical activity were adjusted for. Physical activity was self-scored from 1 to 10 and as this scoring was subjective, there is possibility of misclassification and residual confounding. As discussed before, education is not a perfect surrogate for socioeconomic status. The possibility of residual confounding cannot be ruled out as other potential confounding factors (like dietary habits) might have been overlooked. Although hypertension, hypertriglyceridemia, low HDL cholesterol are known risk factors for T2DM [33], they were not adjusted for in the regression analysis to avoid over-adjustment. These risk factors are firmly related to T2DM and are all part of metabolic syndrome [212]. As DM and family history for DM in an individual are firmly related to each other, adjustment was not performed for this variable to avoid over-adjustment.

4.2.3.3 Paper 3

Participants are 30 and 36–71 years old inhabitants of 10 rural municipalities in Northern Norway. All analyses were stratified by sex. The 8-year cumulative incidence is presented separately for men and women, and the older and younger age groups. In the logistic regression analysis, age, ethnicity, BMI, WC, WHtR, education, leisure-time physical activity, mental distress score, smoking, and alcohol drinking were adjusted for. These variables were measured in the SAMINOR 1 Survey (prior to development of diabetes mellitus) so the risk of temporal bias is reduced. The validity of the applied questions about leisure-time physical activity has been assessed in several studies and is shown to be good [213, 214], nevertheless, there is inevitable misclassification in the reported leisure-time physical activity. In the final analysis, this variable was dichotomised into low leisure-time physical activity (reading, watching TV or other sedentary activities) versus higher leisure-time physical activity. Smoking (current smoker vs ex-smoker or never-smoker), alcohol drinking (at least once a week versus lower or no alcohol drinking) and mental distress (Hopkins SCL-10 >1.85 versus others) were also used as dichotomous variables which opens the possibility for residual confounding. It is worth mentioning that the effects of the above-mentioned dichotomised variables were assessed separately and as original multi-categorical variables, which showed negligibly different results.

4.3 Interaction

Interaction (effect modification) is present when two or more risk factors modify the effect of each other with regard to the occurrence or level of a given outcome [215]. Put in other words, interaction occurs when the effect of a risk factor *A* on the risk of an outcome *Y* is not homogeneous in strata formed by a third variable *Z* [215]. To assess the interaction, one can either examine the interaction term in the regression analysis or stratify the population according to the potential effect modifier. Interaction is present if the interaction term in the logistic regression is statistically significant or the measure of risk is heterogeneous within the strata formed by the potential effect modifier [215]. In papers 1 and 2, there was observed a heterogeneity of risk between different geographical regions. According to the above definition, there is an interaction by geographical region when assessing the relation between ethnicity and the prevalence of pre-diabetes or DM.

4.4 External validity

Most of our study participants were inhabitants of rural municipalities and districts in Northern and Mid-Norway, all of which have substantial Sami settlements. We believe that the results from paper 1 can be generalised to the Sami and non-Sami people living in rural, Sami core areas in these regions. Generalisation to other Sami or non-Sami people living in urban areas or rural areas in other parts of Norway may be problematic. Results in paper 2 and 3 are based on data from only ten municipalities all located in Northern Norway. As there might be considerable differences in the living conditions of inhabitants of the ten municipalities and other municipalities and given the small number of included municipalities and participants, generalisation of the results from papers 2 and 3 to other regions might be even more problematic. The different geographic areas and population composition must be taken into account when comparing results from paper 1 and 2.

4.5 Statistical associations

The SAMINOR 1 Survey had a large sample size (n=16,538 after exclusion of 30-year-old participants). Large sample size increases the power of the study and the precision of the estimates. Paper 1, which was based on a large sample, had potential to detect small ethnic differences in the prevalence of pre-diabetes and DM.

The SAMINOR 2 Clinical Survey had considerably smaller sample size (n=6004); hence paper 2, which was based on the data obtained from this survey suffer from lower power and precision. Paper 3 had even smaller sample size (n=3303), and the study's power to catch any ethnic difference in the cumulative incidence of DM was not high. The large within-group variations worsened the issue.

The comparisons, which were made in rather small geographical regions or sex and age groups all suffer from having small sample size with low power and precision.

It is of particular importance that both Sami and non-Sami people represent a wide variety of life and are under constant effect of genetic and environmental factors. They comprise heterogeneous and dynamic populations and this heterogeneity was best echoed in the varying prevalence of pre-diabetes and DM in different sex and age groups residing in different geographical regions. Even municipalities, which were put into one geographical region (like Karasjok and Kautokeino) were not quite similar in terms of the prevalence of DM.

4.6 Brief discussion of main results and future research

In paper 1, we found no overall ethnic difference in the prevalence of pre-diabetes and DM. Nonetheless, there were some disparities in the prevalence of pre-diabetes and/or DM in some geographical regions. In spite of the large sample size of the SAMINOR 1 Survey, the use of RPG (≥11.1 mmol/L) lowered the ability of the study to catch undiagnosed cases of DM. While according to our definition of DM in paper 1 only about 8% of cases were previously undiagnosed, it is reported in a systematic review that globally, undiagnosed adult DM cases ranges from 24.1% to 75.1% across data regions [110]. The low sensitivity of our method to catch DM cases might have led to underestimated absolute prevalence of pre-diabetes and DM as well as failure to find any ethnic difference in the prevalence of pre-diabetes and/or DM.

In paper 2, a combination of self-report and HbA1c ≥ 6.5% was used to ascertain DM. It is known that HbA1c with this cut-off has generally low sensitivity and identifies one-third fewer cases of undiagnosed DM than a fasting glucose cut-off of ≥ 7.0 mmol/L [3]. In spite of this low sensitivity, the total age-standardised prevalence of pre-diabetes and T2DM was respectively 34.1% and 8.7%. According to these findings, more than one-third of the participants had pre-diabetes and run a substantial risk of developing T2DM later in life. Furthermore, the prevalence of both pre-diabetes and T2DM was statistically significantly higher among Sami compared with non-Sami. This pattern was present in almost all age and sex groups and geographical regions. Although the prevalence of pre-diabetes and T2DM was shown to be higher among Sami participants compared with their non-Sami counterparts, the 95% CIs around the odds ratios were quite wide. This reflects the uncertainty around the exact amount of the higher odds of having the disease and can be due to our relatively small sample size.

The higher estimated prevalence values of pre-diabetes and T2DM in the SAMINOR 2 Clinical Survey compared to corresponding values in the SAMINOR 1 Survey can partly be explained by the higher mean age of participants in the SAMINOR 2 Clinical Survey. The questions regarding DM was not identical in the two surveys and the question in the SAMINOR 2 Clinical Survey was followed by questions on the type of DM. Applying different methodology (HbA1c ≥ 6.5% vs RPG ≥ 11.1 mmol/L) is another important explanation for the observed difference in the estimated prevalence values. Nevertheless, it is quite likely that the prevalence of pre-diabetes and DM in the included municipalities increased during this period in harmony with the increase in the prevalence of DM in all Norway [10].

The most plausible explanation for the observed higher prevalence of pre-diabetes and T2DM in Sami subjects was higher WHtR (index of abdominal obesity). In the SAMINOR 2 Clinical Survey, WHtR was higher among both Sami men and women compared with their non-Sami counterparts. WHtR has in several studies been mentioned to be the best indicator of obesity and predictor of metabolic syndrome and cardiovascular diseases [216]. As Sami people are in average 5–6 cm shorter in stature than their non-Sami counterparts, WC does not seem to perform satisfactorily in comparison of abdominal obesity between them. Obesity was also more prevalent among Sami participants of both sexes in the SAMINOR 1 Survey [13, 44]. Obesity in combination with low physical activity (which was also reported by the Sami women) are well-established risk factors for T2DM. It has been reported that the prevalence of metabolic syndrome (hyperglycaemia, hypertension, obesity, hypertriglyceridemia, low HDL cholesterol) among participants of both ethnicities in the SAMINOR 1 Survey was quite high [13]. Hansen, in his study on the Sami populations ascribed a number of poor health outcomes like obesity, metabolic syndrome and DM to ethnic discrimination reported by Sami people in the SAMINOR 1 Survey [167]. In spite of revitalisation and integration of

Sami culture, language and identity, the Sami people still report ethnic discrimination more frequently than do ethnic Norwegians [217]. Experienced ethnic discrimination has been reported to be associated with adverse somatic and psychological health outcomes [218, 219], and this relationship is complex and multidimensional [220]. This association is usually stronger in areas where Sami populations live in minority compared to areas they live in majority [167]. As DM is known to be a chronic and multifactorial disease, it is more likely that various behavioral, environmental, biological and genetic factors to varying extent interact with stressful conditions like perceived discrimination and contribute to illness and early mortality [221].

Although no statistically significant difference in the 8-year cumulative incidence of DM was observed (paper 3), this lack of statistically significant difference can partly be explained by the small sample size. As for paper 2, indices of obesity (e.g. BMI and WHtR) were generally higher among Sami compared to their non-Sami counterparts.

Contrary to many indigenous peoples throughout the world, no huge difference in the prevalence or incidence of DM was observed between Sami people and ethnic Norwegians. This can be explained by the close interaction and similar standard of living between them. The rapid transition from traditional to so-called western and sedentary lifestyle has affected all inhabitants of the rural study areas regardless of their ethnicity.

5 Implications for public health policies

High prevalence of pre-diabetes and DM among both Sami and non-Sami people and the observed higher prevalence of pre-diabetes and T2DM among Sami compared to non-Sami (in the SAMINOR 2 Clinical Survey) needs attention from health authorities and policy-makers. A large proportion of both Sami and non-Sami people had high indices of obesity and obesity was the most plausible explanation for higher prevalence of pre-diabetes and T2DM among Sami people. These results were partly observed in previous studies as well [222]. The fact that more than one-third of inhabitants in the included municipalities suffer from pre-diabetes and run a higher risk of developing T2DM in the future underscores the need for promoting information campaigns to enhance inhabitants' insight into lifestyle-related diseases and potential consequences of obesity and T2DM. Primary health care personnel especially general practitioners have decisive roles in changing patients' health attitude and implementing preventive measures. Encouraging walking and daily physical activities, promotion of dietary balance in macronutrient intake, increasing fruit and vegetable consumption, reduction in high-sugar and fast-food intake, motivating people in consuming traditional food sources are examples of the preventive measures in this regard [223]. In tailoring any health promotion scheme, Sami language, culture, and perspectives should be taken into account. At-risk persons like those with family history of DM, personal history of gestational diabetes, IFG, IGT, high BMI or WC, hypertension or dyslipidemia should be encouraged to attend periodic medical encounters to diagnose any dysglycaemia at early stage and prevent T2DM development towards late complications. Vigorous efforts have so far been made by Centre for Sami Health Research to convey the findings of the SAMINOR Study to the inhabitants and health care providers of the included municipalities (at local level) and to draw health policy-makers' attention to the needs of local communities (at national level).

6 Further research

Follow-up studies in the future with especial emphasis on the risk factors of T2DM should be undertaken. The applied questionnaires should be validated in advance by comparison between the answers to the questions and medical records. The main concern regarding DM is its late complications (rather than hyperglycaemia itself) and these complications develop well below recommended medical cut-offs [3]. The main objective in most epidemiological studies is to measure the burden of diseases, their risk factors and consequences rather than diagnosing unknown cases. Therefore, instead of or besides using a certain cut-off for the applied test to dichotomise participants as having or not having DM, a risk score can be developed and calculated for each participant based on the continuum of risk at various test values. Sum of these scores can then be compared between the ethnic groups.

Norway has comprehensive registries and healthcare databases like the Norwegian Prescription Database, the Norwegian Patient Registry and primary care database. The future surveys of the SAMINOR Study can benefit from linkage of participants' data to these databases. To elucidate the role of genetic endowment in predisposition to and development of various diseases, gene analyses can be included in the future studies.

As obesity is highly prevalent amongst both Sami and non-Sami people and seems to be the most plausible cause of higher prevalence of T2DM among the Sami people, any future study should try to measure thoroughly the obesity indices. Beside traditional anthropometric measures (e.g. BMI, WC, WHtR), new techniques for measuring body composition (e.g. bioelectrical impedance, dual-energy X-ray absorptiometry, body density, and total body water estimates), physical activity and calorie expenditure should be applied. Potential risk factors for obesity like genetic predisposition, unhealthy dietary habits, low physical activity, and psychological stressors should be addressed and scrutinised.

Literature

1. Kahn, S.E., M.E. Cooper, and S. Del Prato, *Pathophysiology and treatment of type 2 diabetes: perspectives on the past, present, and future.* Lancet, 2014. **383**(9922): p. 1068-83.
2. *World Health Organization, Diabetes Fact Sheet No 312* [cited 2017 Accessed on: July 2017]; Available from: http://www.who.int/mediacentre/factsheets/fs312/en/
3. *American Diabetes Association. Diagnosis and classification of diabetes mellitus.* Diabetes Care, 2010. **33 Suppl 1**: p. S62-9.
4. Powers, A.C., *Diabetes Mellitus: Diagnosis, Classification, and Pathophysiology*, in *Harrisons's principles of internal medicine*. 2015: McGraw-Hill Education: New York. p. 2400.
5. Hu, F.B., et al., *Diet, lifestyle, and the risk of type 2 diabetes mellitus in women.* N Engl J Med, 2001. **345**(11): p. 790-7.
6. Chan, J.M., et al., *Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men.* Diabetes Care, 1994. **17**(9): p. 961-9.
7. Rana, J.S., et al., *Adiposity compared with physical inactivity and risk of type 2 diabetes in women.* Diabetes Care, 2007. **30**(1): p. 53-8.
8. O'Gorman, D.J., et al., *Exercise training increases insulin-stimulated glucose disposal and GLUT4 (SLC2A4) protein content in patients with type 2 diabetes.* Diabetologia, 2006. **49**(12): p. 2983-92.
9. Boule, N.G., et al., *Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials.* JAMA, 2001. **286**(10): p. 1218-27.
10. Ruiz, P.L.D., et al., *Decreasing incidence of pharmacologically and non-pharmacologically treated type 2 diabetes in Norway: a nationwide study.* Diabetologia, 2018. **61**(11): p. 2310–2318.
11. Stene, L.C., et al., *Prevalence of diabetes mellitus in Norway.* Tidsskrift for Den Norske Legeforening, 2004. **124**(11): p. 1511-1514.
12. Nystad, T.A., *A population-based study on cardiovascular risk factors and self-reported type 2 diabetes mellitus in the Sami population*, in *Centre for Sami Health Research, Institute of Community Medicine*. 2010, UiT The Arctic University of Norway: Tromsø. p. 33-34.
13. Broderstad, A.R. and M. Melhus, *Prevalence of metabolic syndrome and diabetes mellitus in Sami and Norwegian populations. The SAMINOR-a cross-sectional study.* BMJ Open, 2016. **6**(4): p. e009474.
14. Bjartveit, K., et al., *The cardiovascular disease study in Norwegian counties. Background and organization.* Acta Med Scand Suppl, 1979. **634**: p. 1-70.
15. Masharani, U., *Diabetes Mellitus & Hypoglycemia*, in *Current Medical Diagnosis & Treatment*, M.A. Papadakis and S.J. McPhee, Editors. 2016, McGraw-Hill Education New York.
16. Hinde, H., *Assimilation of the Sami: Implementation and Consequences*. 2005, Resource Centre for the Rights of Indigenous Peoples: Kautokeino.
17. Kvernmo, S., *Indigenous peoples*, in *The Cambridge Handbook of Acculturation Psychology*, D. Sam and J. Berry, Editors. 2006, Cambridge University Press: Cambridge. p. 233-250.
18. Tvermosegaard, M., et al., *Cardiovascular Disease Susceptibility and Resistance in Circumpolar Inuit Populations.* Canadian Journal of Cardiology, 2015. **31**(9): p. 1116-23.

19. Bruce, S.G., N.D. Riediger, and L.M. Lix, *Chronic disease and chronic disease risk factors among First Nations, Inuit and Metis populations of northern Canada.* Chronic Dis Inj Can, 2014. **34**(4): p. 210-7.
20. Sheikh, N., et al., *Changing dietary patterns and body mass index over time in Canadian Inuit communities.* International Journal of Circumpolar Health, 2011. **70**(5): p. 511-519.
21. Hutchinson, R.N. and S. Shin, *Systematic review of health disparities for cardiovascular diseases and associated factors among American Indian and Alaska Native populations.* PLoS One, 2014. **9**(1): p. e80973.
22. Cobb, N., D. Espey, and J. King, *Health behaviors and risk factors among American Indians and Alaska Natives, 2000-2010.* Am J Public Health, 2014. **104 Suppl 3**: p. S481-9.
23. Boyer, B.B., et al., *Metabolic syndrome in Yup'ik Eskimos: the Center for Alaska Native Health Research (CANHR) Study.* Obesity (Silver Spring), 2007. **15**(11): p. 2535-40.
24. Oster, R.T., et al., *Recent epidemiologic trends of diabetes mellitus among status Aboriginal adults.* CMAJ, 2011. **183**(12): p. E803-8.
25. Xie, J., et al., *Prevalence of self-reported diabetes and diabetic retinopathy in indigenous Australians: the National Indigenous Eye Health Survey.* Clin Exp Ophthalmol, 2011. **39**(6): p. 487-93.
26. McDermott, R.A., M. Li, and S.K. Campbell, *Incidence of type 2 diabetes in two Indigenous Australian populations: a 6-year follow-up study.* The Medical Journal of Australia, 2010. **192**(10): p. 562-5.
27. Lascar, N., et al., *Type 2 diabetes in adolescents and young adults.* Lancet Diabetes Endocrinol, 2017.
28. Powers, A.C., *Diabetes Mellitus: Diagnosis, Classification, and Pathophysiology*, in *Harrisons's principles of internal medicine*. 2015, McGraw-Hill Education: New York. p. 2400.
29. DeFronzo, R.A., R.C. Bonadonna, and E. Ferrannini, *Pathogenesis of NIDDM. A balanced overview.* Diabetes Care, 1992. **15**(3): p. 318-68.
30. Bloomgarden, Z.T., *Insulin resistance: current concepts.* Clin Ther, 1998. **20**(2): p. 216-31; discussion 215.
31. Stumvoll, M., B.J. Goldstein, and T.W. van Haeften, *Type 2 diabetes: principles of pathogenesis and therapy.* Lancet, 2005. **365**(9467): p. 1333-46.
32. *International Diabetes Federation, Diabetes Atlas*. 2017 [Cited November 2017]; 8th edition:[Available from: http://www.diabetesatlas.org/resources/2017-atlas.html.
33. Joseph, J., et al., *Incidence of and risk factors for type-2 diabetes in a general population: the Tromsø Study.* Scand J Public Health, 2010. **38**(7): p. 768-75.
34. Ginde, A.A., et al., *Value of risk stratification to increase the predictive validity of HbA1c in screening for undiagnosed diabetes in the US population.* Journal of General Internal Medicine, 2008. **23**(9): p. 1346-53.
35. McCubbin, L.D. and M. Antonio, *Discrimination and obesity among Native Hawaiians.* Hawaii J Med Public Health, 2012. **71**(12): p. 346-52.
36. Wagner, J.A., et al., *Self-reported discrimination, diabetes distress, and continuous blood glucose in women with type 2 diabetes.* J Immigr Minor Health, 2015. **17**(2): p. 566-73.
37. LeBron, A.M., et al., *Everyday discrimination, diabetes-related distress, and depressive symptoms among African Americans and Latinos with diabetes.* J Immigr Minor Health, 2014. **16**(6): p. 1208-16.

38. Basu, A., et al., *Childhood Maltreatment and Health Impact: The Examples of Cardiovascular Disease and Type 2 Diabetes Mellitus in Adults.* Clin Psychol (New York), 2017. **24**(2): p. 125-139.
39. Zhang, Y., et al., *Risk of Hyperglycemia and Diabetes after Early-Life Famine Exposure: A Cross-Sectional Survey in Northeastern China.* Int J Environ Res Public Health, 2018. **15**(6).
40. Reece, E.A., *The fetal and maternal consequences of gestational diabetes mellitus.* J Matern Fetal Neonatal Med, 2010. **23**(3): p. 199-203.
41. Pettitt, D.J. and W.C. Knowler, *Long-term effects of the intrauterine environment, birth weight, and breast-feeding in Pima Indians.* Diabetes Care, 1998. **21 Suppl 2**: p. B138-41.
42. Martens, P.J., et al., *Breastfeeding Initiation Associated With Reduced Incidence of Diabetes in Mothers and Offspring.* Obstet Gynecol, 2016. **128**(5): p. 1095-1104.
43. Pickup, J.C., *Inflammation and activated innate immunity in the pathogenesis of type 2 diabetes.* Diabetes Care, 2004. **27**(3): p. 813-23.
44. Nystad, T., et al., *Ethnic differences in the prevalence of general and central obesity among the Sami and Norwegian populations: the SAMINOR study.* Scand J Public Health, 2010. **38**(1): p. 17-24.
45. Njølstad, I., E. Arnesen, and P.G. Lund-Larsen, *Sex differences in risk factors for clinical diabetes mellitus in a general population: a 12-year follow-up of the Finnmark Study.* Am J Epidemiol, 1998. **147**(1): p. 49-58.
46. Taskinen, M.-R., *Diabetic dyslipidaemia: from basic research to clinical practice.* Diabetologia, 2003. **46**(6): p. 733-749.
47. Dawson, J. and A. Wyss, *Chicken or the Egg? Hyperuricemia, Insulin Resistance, and Hypertension.* Hypertension, 2017.
48. Meigs, J.B., et al., *Biomarkers of endothelial dysfunction and risk of type 2 diabetes mellitus.* JAMA, 2004. **291**(16): p. 1978-86.
49. Malenica, M., et al., *Importance of inflammatory markers and IL-6 for diagnosis and follow up of patients with type 2 diabetes mellitus.* Med Glas (Zenica), 2017. **14**(2): p. 169-175.
50. Huang, Z., et al., *Serum Markers of Endothelial Dysfunction and Inflammation Increase in Hypertension with Prediabetes Mellitus.* Genet Test Mol Biomarkers, 2016. **20**(6): p. 322-7.
51. Ohkuma, T., et al., *Dose- and time-dependent association of smoking and its cessation with glycemic control and insulin resistance in male patients with type 2 diabetes mellitus: the Fukuoka Diabetes Registry.* PLoS One, 2015. **10**(3): p. e0122023.
52. Bergman, B.C., et al., *Novel and reversible mechanisms of smoking-induced insulin resistance in humans.* Diabetes, 2012. **61**(12): p. 3156-66.
53. Morimoto, A., et al., *Impact of cigarette smoking on impaired insulin secretion and insulin resistance in Japanese men: The Saku Study.* J Diabetes Investig, 2013. **4**(3): p. 274-80.
54. Jelleyman, C., et al., *Associations of Physical Activity Intensities with Markers of Insulin Sensitivity.* Med Sci Sports Exerc, 2017.
55. Kay, S.J. and M.A. Fiatarone Singh, *The influence of physical activity on abdominal fat: a systematic review of the literature.* Obes Rev, 2006. **7**(2): p. 183-200.
56. Cloostermans, L., et al., *Independent and combined effects of physical activity and body mass index on the development of Type 2 Diabetes - a meta-analysis of 9 prospective cohort studies.* Int J Behav Nutr Phys Act, 2015. **12**: p. 147.

57. Kim, J.H., et al., *Association of Education and Smoking Status on Risk of Diabetes Mellitus: A Population-Based Nationwide Cross-Sectional Study.* Int J Environ Res Public Health, 2017. **14**(6).
58. Blomster, J.I., et al., *The impact of level of education on vascular events and mortality in patients with type 2 diabetes mellitus: Results from the ADVANCE study.* Diabetes Res Clin Pract, 2017. **127**: p. 212-217.
59. van Zon, S.K., et al., *The interaction of socioeconomic position and type 2 diabetes mellitus family history: a cross-sectional analysis of the Lifelines Cohort and Biobank Study.* BMJ Open, 2017. **7**(4): p. e015275.
60. Liese, A.D., et al., *Food intake patterns associated with incident type 2 diabetes: the Insulin Resistance Atherosclerosis Study.* Diabetes Care, 2009. **32**(2): p. 263-8.
61. Villegas, R., et al., *Prospective study of dietary carbohydrates, glycemic index, glycemic load, and incidence of type 2 diabetes mellitus in middle-aged Chinese women.* Arch Intern Med, 2007. **167**(21): p. 2310-6.
62. Cheng, G., et al., *Relevance of the dietary glycemic index, glycemic load and genetic predisposition for the glucose homeostasis of Chinese adults without diabetes.* Sci Rep, 2017. **7**(1): p. 400.
63. Schulze, M.B., et al., *Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women.* JAMA, 2004. **292**(8): p. 927-34.
64. Ley, S.H., et al., *Prevention and management of type 2 diabetes: dietary components and nutritional strategies.* Lancet, 2014. **383**(9933): p. 1999-2007.
65. Sun, Q., et al., *White rice, brown rice, and risk of type 2 diabetes in US men and women.* Arch Intern Med, 2010. **170**(11): p. 961-9.
66. Hu, F.B., R.M. van Dam, and S. Liu, *Diet and risk of Type II diabetes: the role of types of fat and carbohydrate.* Diabetologia, 2001. **44**(7): p. 805-17.
67. Montonen, J., et al., *Dietary patterns and the incidence of type 2 diabetes.* Am J Epidemiol, 2005. **161**(3): p. 219-27.
68. Satija, A., et al., *Plant-Based Dietary Patterns and Incidence of Type 2 Diabetes in US Men and Women: Results from Three Prospective Cohort Studies.* PLoS Med, 2016. **13**(6): p. e1002039.
69. Cho, S.S., et al., *Consumption of cereal fiber, mixtures of whole grains and bran, and whole grains and risk reduction in type 2 diabetes, obesity, and cardiovascular disease.* Am J Clin Nutr, 2013. **98**(2): p. 594-619.
70. Nilsson, L.M., et al., *A traditional Sami diet score as a determinant of mortality in a general northern Swedish population.* International Journal of Circumpolar Health, 2012. **71**(0): p. 1-12.
71. Petrenya, N., et al., *Food in rural northern Norway in relation to Sami ethnicity: the SAMINOR 2 Clinical Survey.* Public Health Nutr, 2018: p. 1-13.
72. Broderstad, A.R., et al., *Iron stores in relation to dietary patterns in a multiethnic population: the SAMINOR study.* Public Health Nutr, 2011. **14**(6): p. 1039-46.
73. Naseribafrouei, A., et al., *Prevalence of pre-diabetes and type 2 diabetes mellitus among Sami and non-Sami men and women in Northern Norway - The SAMINOR 2 Clinical Survey.* International Journal of Circumpolar Health, 2018. **77**(1): p. 1463786.
74. Barry, E., et al., *Efficacy and effectiveness of screen and treat policies in prevention of type 2 diabetes: systematic review and meta-analysis of screening tests and interventions.* BMJ Open, 2017. **356**: p. i6538.
75. Edelman, D., et al., *Utility of hemoglobin A1c in predicting diabetes risk.* J Gen Intern Med, 2004. **19**(12): p. 1175-80.

76. Sato, K.K., et al., *Combined measurement of fasting plasma glucose and A1C is effective for the prediction of type 2 diabetes: the Kansai Healthcare Study.* Diabetes Care, 2009. **32**(4): p. 644-6.
77. Shimazaki, T., et al., *Hemoglobin A1c (HbA1c) predicts future drug treatment for diabetes mellitus: a follow-up study using routine clinical data in a Japanese university hospital.* Transl Res, 2007. **149**(4): p. 196-204.
78. Pradhan, A.D., et al., *Hemoglobin A1c predicts diabetes but not cardiovascular disease in nondiabetic women.* Am J Med, 2007. **120**(8): p. 720-7.
79. Goldberg, R.B., et al., *Effect of progression from impaired glucose tolerance to diabetes on cardiovascular risk factors and its amelioration by lifestyle and metformin intervention: the Diabetes Prevention Program randomized trial by the Diabetes Prevention Program Research Group.* Diabetes Care, 2009. **32**(4): p. 726-32.
80. *International Expert Committee report on the role of the A1C assay in the diagnosis of diabetes.* Diabetes Care, 2009. **32**(7): p. 1327-34.
81. Berg, J.P., *HbA1c as a diagnostic tool in diabetes mellitus.* Norsk Epidemiologi, 2013. **23**(1): p. 5-8.
82. Helsedirektoratet. *HbA1c som diagnostikum for diabetes (article in Norwegian)*. 2012 [cited 16 Feb 2018; Available from: http://legeforeningen.no/Fagmed/Norsk-forening-for-medisinsk-biokjemi/Nyheter/20121/HbA1c-som-diagnostikum-ved-diabetes/.
83. Borch-Johnsen, K. and S. Colagiuri, *Diagnosing diabetes--time for a change?* Diabetologia, 2009. **52**(11): p. 2247-2250.
84. Genuth, S., et al., *Follow-up report on the diagnosis of diabetes mellitus.* Diabetes Care, 2003. **26**(11): p. 3160-7.
85. *Definition and Diagnosis of Diabetes Mellitus and Intermediate Hyperglycemia: Report of WHO/IDF Consultation* 2006, World Health Organization Geneva.
86. Ziemer, D.C., et al., *Random plasma glucose in serendipitous screening for glucose intolerance: screening for impaired glucose tolerance study 2.* J Gen Intern Med, 2008. **23**(5): p. 528-35.
87. Johnson, S.L., B.P. Tabaei, and W.H. Herman, *The efficacy and cost of alternative strategies for systematic screening for type 2 diabetes in the U.S. population 45-74 years of age.* Diabetes Care, 2005. **28**(2): p. 307-11.
88. Zhang, P., et al., *Efficient cutoff points for three screening tests for detecting undiagnosed diabetes and pre-diabetes: an economic analysis.* Diabetes Care, 2005. **28**(6): p. 1321-5.
89. Little, R.R. and D.B. Sacks, *HbA1c: how do we measure it and what does it mean?* Curr Opin Endocrinol Diabetes Obes, 2009. **16**(2): p. 113-8.
90. *Definition and Diagnosis of Diabetes Mellitus and Intermediate Hyperglycemia: Report of WHO/IDF Consultation* 2006, World Health Organization Geneva.
91. Gillett, M.J., *International Expert Committee report on the role of the A1c assay in the diagnosis of diabetes: Diabetes Care 2009; 32(7): 1327-1334.* Clin Biochem Rev, 2009. **30**(4): p. 197-200.
92. Dahl-Jørgensen, K., et al., *Effect of near normoglycaemia for two years on progression of early diabetic retinopathy, nephropathy, and neuropathy: the Oslo study.* British Medical Journal (Clinical Research Edition), 1986. **293**(6556): p. 1195-9.
93. Ito, C., et al., *Correlation among fasting plasma glucose, two-hour plasma glucose levels in OGTT and HbA1c.* Diabetes Res Clin Pract, 2000. **50**(3): p. 225-30.
94. Sacks, D.B., *A1C versus glucose testing: a comparison.* Diabetes Care, 2011. **34**(2): p. 518-23.

95. Nitin, S., *HbA1c and factors other than diabetes mellitus affecting it.* Singapore Med J, 2010. **51**(8): p. 616-22.
96. Hutchinson, M.S., et al., *Glycated hemoglobin in diagnosis of diabetes mellitus and pre-diabetes; validation by oral glucose tolerance test. The Tromsø OGTT Study.* Journal of Endocrinological Investigation, 2012. **35**(9): p. 835-40.
97. van 't Riet, E., et al., *Relationship between A1C and glucose levels in the general Dutch population: the new Hoorn study.* Diabetes Care, 2010. **33**(1): p. 61-6.
98. Higgins, T.N., et al., *Is HbA1c a good screening test for diabetes mellitus?* Clin Biochem, 2011. **44**(17-18): p. 1469-72.
99. Lipscombe, L., *ACP Journal Club. HbA1c levels had low sensitivity but high specificity for screening for diabetes.* Ann Intern Med, 2011. **154**(8): p. JC4-9.
100. Picon, M.J., et al., *Hemoglobin A1c versus oral glucose tolerance test in postpartum diabetes screening.* Diabetes Care, 2012. **35**(8): p. 1648-53.
101. Hutchinson, M.S., et al., *Effects of Age and Sex on Estimated Diabetes Prevalence Using Different Diagnostic Criteria: The Tromsø OGTT Study.* Int J Endocrinol, 2013. **2013**: p. 613475.
102. *WHO mortality database, Health statistics and information systems*. 2016 [cited 2018; Available from: http://www.who.int/healthinfo/statistics/mort/en/.
103. Mathers, C.D. and D. Loncar, *Projections of global mortality and burden of disease from 2002 to 2030.* PLoS Med, 2006. **3**(11): p. e442.
104. *World Health Organisation. Diabetes Fact Sheet No 312.* 2017 [Cited Decembre 2017]; Available from: http://www.who.int/mediacentre/factsheets/fs312/en/.
105. Guariguata, L., et al., *Global estimates of diabetes prevalence for 2013 and projections for 2035.* Diabetes Res Clin Pract, 2014. **103**(2): p. 137-49.
106. *NCD Risk Factor Collaboration; Worldwide trends in diabetes since 1980: a pooled analysis of 751 population-based studies with 4.4 million participants.* Lancet, 2016. **387**(10027): p. 1513-30.
107. *Global status report on noncommunicable diseases 2015* Geneva: World Health Organization.
108. Owe, K., et al. *Physical Activity* Public Health Report 27.09.2017 [cited 30.06.2014; Available from: https://www.fhi.no/nettpub/hin/levevaner/fysisk-aktivitet/.
109. Mendis, S., S. Davis, and B. Norrving, *Organizational update: the world health organization global status report on noncommunicable diseases 2014; one more landmark step in the combat against stroke and vascular disease.* Stroke, 2015. **46**(5): p. e121-2.
110. Beagley, J., et al., *Global estimates of undiagnosed diabetes in adults.* Diabetes Res Clin Pract, 2014. **103**(2): p. 150-60.
111. Meeks, K.A., et al., *Disparities in type 2 diabetes prevalence among ethnic minority groups resident in Europe: a systematic review and meta-analysis.* Intern Emerg Med, 2016. **11**(3): p. 327-40.
112. *Folkehelseinstituttet: Folkehelserapporten, Diabetes*. 2017 [cited 2018; Available from: https://www.fhi.no/nettpub/hin/ikke-smittsomme/diabetes-i-norge---folkehelserappor/#vel-4-prosent-av-befolkningen-har-diagnosen-diabetes.
113. Strøm, H., et al., *No increase in new users of blood glucose-lowering drugs in Norway 2006-2011: a nationwide prescription database study.* BioMed Central Public Health, 2014. **14**: p. 520.
114. Krokstad, S. and M.S. Knudtsen, *Population health development. The HUNT Study : HUNT 1 (1984-86) - HUNT 2 (1995-97) - HUNT 3 (2006-08). 2011, Levanger, Norway: HUNT Research Centre*. 2011: Norway. p. 64-67.

115. Sørensen, M., et al., *Cost of diabetes in Norway 2011.* Diabetes Res Clin Pract, 2016. **122**: p. 124-132.
116. Nosrati, E., et al., *Ethnicity and place: the geography of diabetes inequalities under a strong welfare state.* Eur J Public Health, 2018. **28**(1): p. 30-34.
117. Naqshbandi, M., et al., *Global complication rates of type 2 diabetes in Indigenous peoples: A comprehensive review.* Diabetes Res Clin Pract, 2008. **82**(1): p. 1-17.
118. Young, T.K., et al., *Type 2 diabetes mellitus in Canada's first nations: status of an epidemic in progress.* CMAJ, 2000. **163**(5): p. 561-6.
119. *Royal Australian College of Ophthalmologists. The National Trachoma and Eye Health Program* 1980, Royal Australian College of Ophthalmologists: Sydney.
120. Minges, K.E., et al., *Diabetes prevalence and determinants in Indigenous Australian populations: A systematic review.* Diabetes Res Clin Pract, 2011. **93**(2): p. 139-49.
121. Cunningham, J., *Socio-economic gradients in self-reported diabetes for Indigenous and non-Indigenous Australians aged 18-64.* Aust N Z J Public Health, 2010. **34 Suppl 1**: p. S18-24.
122. Pelletier, C., et al., *Report summary. Diabetes in Canada: facts and figures from a public health perspective.* Chronic Dis Inj Can, 2012. **33**(1): p. 53-4.
123. Johnson, J.A., et al., *Increasing incidence and prevalence of diabetes among the Status Aboriginal population in urban and rural Alberta, 1995-2006.* Can J Public Health, 2009. **100**(3): p. 231-6.
124. Jørgensen, M.E., et al., *Diabetes in Greenland and its relationship with urbanization.* Diabet Med, 2012. **29**(6): p. 755-760.
125. *IDF Diabetes Atlas, Sixth Edition*. Available from: http://www.idf.org/diabetesatlas.
126. Jeppesen, C., P. Bjerregaard, and M.E. Jorgensen, *Dietary patterns in Greenland and their relationship with type 2 diabetes mellitus and glucose intolerance.* Public Health Nutr, 2014. **17**(2): p. 462-70.
127. Grarup, N., et al., *Diabetes in Population Isolates: Lessons from Greenland.* Rev Diabet Stud, 2015. **12**(3-4): p. 320-9.
128. Moltke, I., et al., *A common Greenlandic TBC1D4 variant confers muscle insulin resistance and type 2 diabetes.* Nature, 2014. **512**(7513): p. 190-3.
129. Pedersen, M.L. and J.L. Jacobsen, *Improvement of diabetes care in a small but geographically widely spread population in Greenland. Effects of a national diabetes care programme.* Diabet Med, 2011. **28**(11): p. 1425-32.
130. Satterfield, D., et al., *Health Promotion and Diabetes Prevention in American Indian and Alaska Native Communities-Traditional Foods Project, 2008-2014.* MMWR Suppl, 2016. **65**(1): p. 4-10.
131. *National Diabetes Fact Sheet: National Estimates and General Information on Diabetes and Prediabetes in the United States, ol 201*. 2011: Department of Health and Human Services, Centers for Disease Control and Prevention: Atlanta, GA, USA,.
132. Anderson, K.G., P. Spicer, and M.T. Peercy, *Obesity, Diabetes, and Birth Outcomes Among American Indians and Alaska Natives.* Matern Child Health J, 2016. **20**(12): p. 2548-2556.
133. Kelley, A., et al., *American-Indian diabetes mortality in the Great Plains Region 2002-2010.* BMJ Open Diabetes Res Care, 2015. **3**(1): p. e000070.
134. Fretts, A.M., et al., *Associations of processed meat and unprocessed red meat intake with incident diabetes: the Strong Heart Family Study.* Am J Clin Nutr, 2012. **95**(3): p. 752-8.
135. Pavkov, M.E., et al., *Changing patterns of type 2 diabetes incidence among Pima Indians.* Diabetes Care, 2007. **30**(7): p. 1758-63.

136. Schulz, L.O. and L.S. Chaudhari, *High-Risk Populations: The Pimas of Arizona and Mexico.* Curr Obes Rep, 2015. **4**(1): p. 92-8.
137. Baier, L.J. and R.L. Hanson, *Genetic studies of the etiology of type 2 diabetes in Pima Indians: hunting for pieces to a complicated puzzle.* Diabetes, 2004. **53**(5): p. 1181-6.
138. Arcos-Burgos, M. and M. Muenke, *Genetics of population isolates.* Clin Genet, 2002. **61**(4): p. 233-47.
139. Solbakk, J.T., *Samene - en håndbok*. 2004, Karasjok, Norway: Davvi Girji OS.
140. Hermansen, R., I. Njølstad, and V. Fønnebø, *Physical activity according to ethnic origin in Finnmark county, Norway. The Finnmark Study.* Int J Circumpolar Health, 2002. **61**(3): p. 189-200.
141. Siri, S.R.A., et al., *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): p. 638-646.
142. Hermansen, R., et al., *The impact of changes in leisure time physical activity on changes in cardiovascular risk factors: results from The Finnmark 3 Study and SAMINOR 1, 1987-2003.* Int J Circumpolar Health, 2018. **77**(1): p. 1459145.
143. Senior, P.A. and R. Bhopal, *Ethnicity as a variable in epidemiological research.* BMJ, 1994. **309**(6950): p. 327-30.
144. Peoples, J. and G. Bailey, *Humanitiy: An Introduction to Cultural Anthropology* 9th ed. 2010: Wadsworth Cengage learning.
145. Marmot, M.G., *General approaches to migrant studies: the relation between disease, social class and ethnic origin*, in *Ethnic factors in health and disease*, J.K. Cruickshank and D.G. Beevers, Editors. 1989, Wright: London. p. 12-17.
146. Last, J.M., *ethnicity*, in *A dictionary of epidemiology* 1988, Oxford University Press: New York.
147. Allen, R.E., *ethnicity*, in *Concise Oxford dictionary*. 1990, Oxford University Press: Oxford.
148. Kaplan, J.B. and T. Bennett, *Use of race and ethnicity in biomedical publication.* JAMA, 2003. **289**(20): p. 2709-16.
149. Bhopal, R., *Ethnicity, race, and health in multicultural societies : foundations for better epidemiology, public health and health care.* second ed. 2007, Oxford: Oxford University Press.
150. Egede, L.E., *Race, ethnicity, culture, and disparities in health care.* J Gen Intern Med, 2006. **21**(6): p. 667-9.
151. Connelly, R., V. Gayle, and P.S. Lambert, *Ethnicity and ethnic group measures in social survey research.* Methodological Innovations, 2016. **9**.
152. Berg-Nordlie, M. and H. Gaski. *Store Norske Leksikon*. 31 January 2017; Available from: https://snl.no/S%C3%A1pmi
153. *The Sámi Act, Act of 12 June 1987 No. 56 concerning the Sameting (the Sami parliament) and other Sami legal matters (the Sami Act).* [Cited Decembre 2017]; Available from: https://www.regjeringen.no/en/dokumenter/the-sami-act-/id449701/.
154. Pettersen, T., *Out of the backwater? Prospects for contemporary Sami demography in Norway*, in *Indigenous Peoples and Demography: The Complex Relation between Identity and Statistics*, P. Skøld, Editor. 2011, Berghahn Books.
155. Bjørklund, I., *Fjordfolket i Kvænangen: Fra samisk samfunn til norsk utkant 1550-1980*. . Tromsø: Universitetsforslaget; 1985.
156. Gjerde. A and J. Mosli, *Samiske næringers plass i samfunnsplanlegginga. Guovdageaidnu: Sami instituhtta; 1985*.

157. Jones-Bamman, R.W., *"As long as we continue to joik, we'll remember who we are": negotiating identity and the performance of culture: the Saami joik.* 1993, Seattle (WA): University of Washington.
158. Hilder, T., *Sámi musical performance and the politics of indigeneity in Northern Europe*. 2015, Anham(MD): Rowman & Littlefield.
159. Eriksen, K. and E. Niemi, *Den finske fare: sikkerhetsproblemer og minoritetspolitikk i nord 1860-1940*. Oslo: Universitetsforslaget; 1981.
160. Evjen, B., *Giftemål, næring og etnisk tilhørighet 1850-1930*, in *Nordlands kulturelle mangfold: Etniske relasjoner i historisk perspektiv.* , B. Evjen and L. Hansen, Editors. 2008, Pax Forlag: Oslo. p. 239-271.
161. Minde, H., *Assimilation of the Sami: Implementation and Consequesnces, .* Kautokeino: Resource Centre for the Rights of Indigenous Peoples; 2005.
162. Hansen, L. and B. Evjen, *Kjært barn - mange navn: Om forskjellige betegnelser på den samiske befolkningen i Nordland gjennom århundrene*, in *Nordlands kulturelle mangfold: Etniske relasjoner i historisk perspektiv*, L. Hansen and B. Evjen, Editors. 2008, Pax Forlag: Oslo. p. 17-47.
163. NOU, *2008: 5: Retten til fisken i havet utenfor Finnmark.*, in *Norges offentlige utredninger (NOU)*. 2008, Departementenes servicesenter, Informasjonsforvaltning; 2008 Oslo. p. 569.
164. Eyþórsson, E., *Ressurser, livsform og lokal kunnskap: Studie av en fjordbygd i Finnmark.* Tromsø: [E. Ethorsson]; 1991.
165. Riseth, J.Å., *Sustainable reindeer herding?*, in *Sami statistics speak, numbers and commentary*. 2018, Sami University College: Alta, Norway.
166. Brustad, M., *Sami Health - a Summary of published results of population studies in Norway*, in *Sami statistics speak*. 2018, Sami University College: Alta, Norway.
167. Hansen, K.L., *Ethnic discrimination and health: the relationship between experienced ethnic discrimination and multiple health domains in Norway's rural Sami population.* Int J Circumpolar Health, 2015. **74**: p. 25125.
168. *Proposal for Ethical Guidelines for Sámi Health Research and Research on Sámi Human Biological Material. 2018, The Sami Parliament. Available in English from https://www.sametinget.no/content/download/2341/35365*.
169. Forsdahl, A., et al., *Hjerte-karundersøkelsene i Finnmark 1974–2000: resultater fra undersøkelsene.* ISM Skriftserie, 2001. **58: ISBN : 8290262655**.
170. Berg. S, *Finnemisjonen eller Samemisjonen i Norge*. 1926: Trondhjem: Norges Finnemisjonsselskab.
171. Rein, K., *Spedbarnsdødeligheten i Kautokeino 1946–1955.* Tidsskrift for Den Norske Lægeforening, 1956. **76**(21): p. 815–816.
172. *Myrseth Echinokokksykdommen i Finnmark.* Tidsskrift for Den Norske Lægeforening, 1956. **76**(22): p. 867–871.
173. Anderson, I., et al., *Indigenous and tribal peoples' health (The Lancet-Lowitja Institute Global Collaboration): a population study.* Lancet, 2016. **388**(10040): p. 131-57.
174. Siri, S.R.A., *Bruk av samisk etnisitet i forskningsdatabaser og helseregistre*. 2015, Senter for samisk helseforskning (SSHF), Institutt for samfunnsmedisin, Det helsevitenskapelige fakultet, UiT Norges arktiske universitet: Tromsø
175. Niemi, E., *The Finns in Northern Scandinavia and minority policy*, in *Ethnicity and nation building in the Nordic world*, S. Tägil, Editor. 1995, Hurst: London. p. 145-178.

176. *National Minorities*. Indigenous peoples and minorities [cited 20 Feb 2017; Available from: https://www.regjeringen.no/en/topics/indigenous-peoples-and-minorities/national-minorities/id1404/.
177. Lund, E., et al., *Population based study of health and living conditions in areas with both Sami and Norwegian populations--the SAMINOR study.* Int J Circumpolar Health, 2007. **66**(2): p. 113-28.
178. Aubert, V., *Den samiske befolkningen i Nord-Norge.* Artikler fra Statistisk Sentralbyrå, 1978. **107**(27).
179. Brustad, M., et al., *A population-based study on health and living conditions in areas with mixed Sami and Norwegian settlements - the SAMINOR 2 questionnaire study.* Int J Circumpolar Health, 2014. **73**: p. 23147.
180. Bhopal, R., *Epidemiological study design and principles of data analysis*, in *Concepts of epidemiology*. 2008, Oxford University Press: Oxford.
181. Athena, S.L. and S. Grosby, *Nationalism and Ethnosymbolism: History, Culture and Ethnicity in the Formation of Nations*. 2006: Edinburgh University Press.
182. Eriksen, A.M., et al., *Emotional, physical and sexual violence among Sami and non-Sami populations in Norway: The SAMINOR 2 questionnaire study.* Scand J Public Health, 2015. **43**(6): p. 588-96.
183. Pettersen, T. and M. Brustad, *Which Sami? Sami inclusion criteria in population-based studies of Sami health and living conditions in Norway - an exploratory study exemplified with data from the SAMINOR study.* Int J Circumpolar Health, 2013. **72**: p. 21813.
184. Tourangeau, R. and T. Yan, *Sensitive questions in surveys.* Psychol Bull, 2007. **133**(5): p. 859-83.
185. Szklo, M. and F.J. Nieto, *Understanding lack of validity: Bias*, in *Epidemiology beyond the basics*. 2014, Jones & Bartlett Learning: Burlington.
186. Rothman, K.J., *Epidemiology: an introduction*. Second ed. 2012, Oxford: Oxford University Press.
187. Bhopal, R., *Error, bias, confounding and risk modification/interaction in epidemiology*, in *Concepts of epidemiology*. 2008, Oxford University Press: Oxford.
188. Grimes, D.A. and K.F. Schulz, *Bias and causal associations in observational research.* The Lancet, 2002. **359**(9302): p. 248-252.
189. Langhammer, A., et al., *The HUNT study: participation is associated with survival and depends on socioeconomic status, diseases and symptoms.* BMC Med Res Methodol, 2012. **12**: p. 143.
190. Jacobsen, B.K. and D.S. Thelle, *The Tromsø Heart Study: responders and non-responders to a health questionnaire, do they differ?* Scand J Soc Med, 1988. **16**(2): p. 101-4.
191. Jacobsen, B.K., et al., *Cohort profile: the Tromsø Study.* Int J Epidemiol, 2012. **41**(4): p. 961-7.
192. Knudsen, A.K., et al., *The health status of nonparticipants in a population-based health study: the Hordaland Health Study.* Am J Epidemiol, 2010. **172**(11): p. 1306-14.
193. *Folkehelseinstituttet, Dødsårsaksregistret, statistikkbank*. [cited 2018; Available from: http://statistikkbank.fhi.no/dar/.
194. *Statistics Norway, Døde, etter kjønn og aldersgruppe (per 100 000 av middelfolkemengden) 1866-1870 - 2011-2015*. [cited 2018; Available from: https://www.ssb.no/statbank/table/05848/.

195. Eliassen, B.M., et al., *Prevalence of self-reported myocardial infarction in Sami and non-Sami populations: the SAMINOR study.* International Journal of Circumpolar Health, 2015. **74**: p. 24424.
196. Bongo, B.A., *"Samer snakker ikke om helse og sykdom" : samisk forståelseshorisont og kommunikasjon om helse og sykdom : en kvalitativ undersøkelse i samisk kultur*. 2012, UiT The Arctic University of Norway, Det helsevitenskapelige fakultet, Institutt for helse- og omsorgsfag: Tromsø.
197. Midthjell, K., et al., *Is questionnaire information valid in the study of a chronic disease such as diabetes? The Nord-Trondelag diabetes study.* J Epidemiol Community Health, 1992. **46**(5): p. 537-42.
198. El Fakiri, F., M.A. Bruijnzeels, and A.W. Hoes, *No evidence for marked ethnic differences in accuracy of self-reported diabetes, hypertension, and hypercholesterolemia.* Journal of Clinical Epidemiology, 2007. **60**(12): p. 1271-1279.
199. Okura, Y., et al., *Agreement between self-report questionnaires and medical record data was substantial for diabetes, hypertension, myocardial infarction and stroke but not for heart failure.* Journal of Clinical Epidemiology, 2004. **57**(10): p. 1096-1103.
200. Tretli, S., P.G. Lund-Larsen, and O.P. Foss, *Reliability of questionnaire information on cardiovascular disease and diabetes: cardiovascular disease study in Finnmark county.* J Epidemiol Community Health, 1982. **36**(4): p. 269-73.
201. Sheikh, M.A., E. Lund, and T. Braaten, *Test-retest reliability of self-reported diabetes diagnosis in the Norwegian Women and Cancer Study: A population-based longitudinal study (n =33,919).* SAGE Open Med, 2016. **4**: p. 2050312115622857.
202. *Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus*, in *Diabetes Care*. 1997. p. 1183-97.
203. Hellgren, M., K. Hjorleifsdottir Steiner, and L. Bennet, *Haemoglobin A1c as a screening tool for type 2 diabetes and prediabetes in populations of Swedish and Middle-East ancestry.* Prim Care Diabetes, 2017. **11**(4): p. 337-343.
204. Yu, E.Y., et al., *Can HbA1c replace OGTT for the diagnosis of diabetes mellitus among Chinese patients with impaired fasting glucose?* Fam Pract, 2015. **32**(6): p. 631-8.
205. Homko, C.J., et al., *A Single A1C >= 6.5% Accurately Identifies Type 2 Diabetes/Impaired Glucose Tolerance in African Americans.* J Prim Care Community Health, 2012. **3**(4): p. 235-8.
206. Huang, H., et al., *The diagnostic threshold of HbA1c and impact of its use on diabetes prevalence-a population-based survey of 6898 Han participants from southern China.* Prev Med, 2013. **57**(4): p. 345-50.
207. Grill, V. and B.O. Asvold, *A form of autoimmune diabetes in adults named LADA - an update on essential features and controversies.* Curr Diabetes Rev, 2018.
208. Szklo, M. and F.J. Nieto, *Identifying noncausal association: confounding*, in *Epidemiology beyond the basics*. 2014, Jones & Bartlett Learning: Burlington.
209. Last, J., *A dictionary of epidemiology*. 2000, NY: Oxford University Press: New York.
210. Breslow, N., *Design and analysis of case-control studies.* Annu Rev Public Health, 1982. **3**: p. 29-54.
211. Szklo, M. and F.J. Nieto, *Stratification and adjustment: multivariate analysis in epidemiology*, in *Epidemiology beyond the basics*. 2014, Jones & Bartlett Learning: Burlington.
212. Engin, A., *The Definition and Prevalence of Obesity and Metabolic Syndrome.* Adv Exp Med Biol, 2017. **960**: p. 1-17.

213. Grimby, G., et al., *The "Saltin-Grimby Physical Activity Level Scale" and its application to health research.* Scandinavian Journal of Medicine & Science in Sports, 2015. **25 Suppl 4**: p. 119-25.
214. Emaus, A., et al., *Does a variation in self-reported physical activity reflect variation in objectively measured physical activity, resting heart rate, and physical fitness? Results from the Tromso study.* Scand J Public Health, 2010. **38**(5 Suppl): p. 105-18.
215. Szklo, M. and F.J. Nieto, *Defining and assessing heterogeneity of effects: interaction,* in *Epidemiology beyond the basics*. 2014, Jones & Bartlett Learning: Burlington.
216. Ashwell, M., P. Gunn, and S. Gibson, *Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis*. Obes Rev, 2012. **13**(3): p. 275-86.
217. Hansen, K.L., et al., *Ethnic discrimination and bullying in the Sami and non-Sami populations in Norway: the SAMINOR study.* Int J Circumpolar Health, 2008. **67**(1): p. 97-113.
218. Hansen, K.L., M. Melhus, and E. Lund, *Ethnicity, self-reported health, discrimination and socio-economic status: a study of Sami and non-Sami Norwegian populations.* Int J Circumpolar Health, 2010. **69**(2): p. 111-28.
219. Hansen, K.L. and T. Sorlie, *Ethnic discrimination and psychological distress: a study of Sami and non-Sami populations in Norway.* Transcult Psychiatry, 2012. **49**(1): p. 26-50.
220. Myers, H.F., *Ethnicity- and socio-economic status-related stresses in context: an integrative review and conceptual model.* J Behav Med, 2009. **32**(1): p. 9-19.
221. Ahrens, W. and I. Pigeot, *Handbook of Epidemiology*. 2014, New York: NY: Springer.
222. Nystad, T., *A population-based study on cardiovascular risk factors and self-reported type 2 diabetes mellitus in the Sami population*. 2010, UiT The Arctic University of Norway, Faculty of Health Sciences, Department of Community Medicince, Centre for Sami Health Research: Tromsø.
223. Zienczuk, N., et al., *Dietary correlates of an at-risk BMI among Inuit adults in the Canadian high arctic: cross-sectional international polar year Inuit health survey, 2007-2008.* Nutr J, 2012. **11**: p. 73.

Paper I

INTERNATIONAL JOURNAL OF CIRCUMPOLAR HEALTH

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ORIGINAL RESEARCH ARTICLE

Ethnic difference in the prevalence of pre-diabetes and diabetes mellitus in regions with Sami and non-Sami populations in Norway – the SAMINOR1 study

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Objective. The aim of this study was to measure the prevalence of pre-diabetes and diabetes mellitus in rural populations of Norway, as well as to explore potential ethnic disparities with respect to dysglycaemia in Sami and non-Sami populations.
Design. Cross-sectional population-based study.
Methods. The SAMINOR1 study was performed in 2003–2004. The study took place in regions with both Sami and non-Sami populations and had a response rate of 60.9%. Information in the SAMINOR1 study was collected using two self-administered questionnaires, clinical examination and laboratory tests. The present analysis included 15,208 men and women aged 36–79 years from the SAMINOR1 study.
Results. Age-standardised prevalence of pre-diabetes and diabetes mellitus among Sami men was 3.4 and 5.5%, respectively. Corresponding values for non-Sami men were 3.3 and 4.6%. Age-standardised prevalence of pre-diabetes and diabetes mellitus for Sami women was 2.7 and 4.8%, respectively, while corresponding values for non-Sami women were 2.3 and 4.5%. Relative risk ratios for dysglycaemia among Sami participants compared with non-Sami participants were significantly different in different geographical regions, with the southern region having the highest prevalence of pre-diabetes and diabetes mellitus among Sami participants.
Conclusion. We observed a heterogeneity in the prevalence of pre-diabetes and diabetes mellitus in different geographical regions both within and between different ethnic groups.

Keywords: *prevalence*; *dysglycaemia*; *indigenous people*; *SAMINOR*

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Norway is home to many ethnic groups, including Norwegians, Kvens and Sami. Kvens are descendants of Finnish ethnicity who immigrated to and settled in the northern parts of Norway in the 1700s and 1800s (1). The Sami people are an indigenous population inhabiting the northern parts of Norway, Sweden, Finland and the Kola Peninsula in Russia. The traditional Sami settlements in Norway span from Finnmark in the north to Engerdal in the Hedmark county in the south. The Sami population harbours a rich variety of languages, cultures and other social circumstances. However, the process of industrialisation has introduced changes in their lifestyle and living conditions. Today, many of the Sami have a sedentary lifestyle, which predisposes them to obesity and type 2 diabetes mellitus (2,3).

Diabetes mellitus is a chronic disease with long-term complications. These complications have become a major cause of morbidity and mortality worldwide and are predicted to increase further in the coming decades (4). The prevalence of diabetes in rural areas has increased to an alarming level in both low- to middle-income countries and high-income countries during the past few decades (5). The estimated prevalence of self-reported diabetes mellitus in people aged ≥ 30 years in Norway was 3.4% in 2004 (6). The Nord-Trøndelag Health Survey (HUNT) reported a prevalence of diagnosed cases of

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adult diabetes of 4.3% in 2006 (7). The prevalence of diabetes mellitus and impaired glucose tolerance among the indigenous people of Greenland, the Inuit, has also increased (8).

We lack up-to-date knowledge about the prevalence of pre-diabetes and diabetes mellitus among the inhabitants of northern and mid-Norway, especially regarding eventual ethnic differences. The aim of this study was to measure the prevalence of pre-diabetes and diabetes mellitus in rural populations of Norway, as well as to explore potential ethnic disparities with respect to dysglycaemia in Sami and non-Sami populations.

Methods

The SAMINOR1 study

In 2003–2004, the Centre for Sami Health Research at the University of Tromso (UiT) The Arctic University of Norway, in collaboration with the Norwegian Institute of Public Health, conducted the SAMINOR1 study, the first population-based study on health and living conditions in regions with both Sami and Norwegian populations (9). This survey included municipalities and districts in Norway with a high proportion of people with Sami ethnicity, as determined by ethnicity and language information reported in the 1970 census and historical and local knowledge about traditional Sami settlements. These municipalities and districts were almost all located in rural areas. All residents aged 30 and 36–79 years registered in the National Registry in the selected regions were invited to participate in the SAMINOR1 study, regardless of their ethnic background (n = 27,987). Each study participant completed two self-administered questionnaires, which were provided in Norwegian and the three main Sami languages. The clinical investigation was done in two buses moving from place to place throughout the study area. Non-fasting blood samples were taken to determine plasma glucose levels. The Regional Committee for Medical Research Ethics approved the SAMINOR1 study, and all participants gave informed written consent.

Data collection

The SAMINOR1 study collected information through questionnaires, physical examinations, including anthropometric measures and blood pressure, and blood sampling. The questionnaires covered topics such as language and ethnicity; use of health services and the satisfaction with these services; socio-economic factors; accidents; discrimination; self-reported diseases and illnesses; diseases in the family; mental health symptoms; medication; some questions on diet, smoking, alcohol, physical activity and social networks; and for women only, questions on menstruation, fertility and use of exogenous hormones. Ethnicity was determined through questions such as: "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?". The respondents were also asked whether they considered themselves to be Norwegian, Sami, Kven or other. The respondents could answer "Sami", "Norwegian", "Kven" or "other". Participants could tick more than one answer for all questions mentioned above. Participants were categorised as Sami if they responded that they either considered themselves to be Sami or reported to have a Sami ethnic background, and if at least one of their grandparents, parents or they themselves spoke a Sami language at home. All other participants were categorised as non-Sami.

Both questionnaire information and non-fasting plasma glucose measurements were used to ascertain the presence of pre-diabetes and diabetes mellitus. Those who reported in the questionnaire that they currently have or previously had diabetes mellitus were classified as having diabetes. In addition, we used a random, non-fasting plasma glucose measurement as an objective method for diagnosing dysglycaemia. Participants with non-fasting plasma glucose levels of $\geq$11.1 mmol/L were also classified as having diabetes, and those with a level of 7.8–11.0 mmol/L were classified as having pre-diabetes. The remaining participants were categorised as normoglycaemic.

Geographical regions

We defined four geographical regions: "Region 1" consisted of areas in the inland of Finnmark county, including Karasjok and Kautokeino municipalities; "Region 2" consisted of both inland and coastal areas in Finnmark county, including Porsanger, Tana and Nesseby municipalities; "Region 3" consisted of coastal areas in Finnmark and the northern part of Troms county, including Lyngen, Storfjord, Kåfjord, Kvænangen, Alta, Loppa, Kvalsund and Lebesby municipalities; "Region 4" consisted of Marka, Lule and south Sami areas in southern Troms, Nordland, Nord- and Sør-Trøndelag counties, including Lavangen, Narvik, Evenes, Skånland, Tysfjord, Hattfjelldal, Røyrvik, Namsskogan, Grane, Snåsa and Røros municipalities (Fig. 1).

Statistical analysis

The data management and statistical analysis were performed using STATA version 14.1 (StataCorp, College Station, TX, USA). Age difference between ethnic groups across regions and genders was assessed using two sample t-tests (Table II). Education level was not included in the final model as it was not a significant confounding factor and had many missing values. Variables which were strongly correlated to diabetes and/or were parts of metabolic syndrome such as hypertension, dyslipidaemia, obesity and family history of diabetes mellitus were not included in the final regression analysis to avoid

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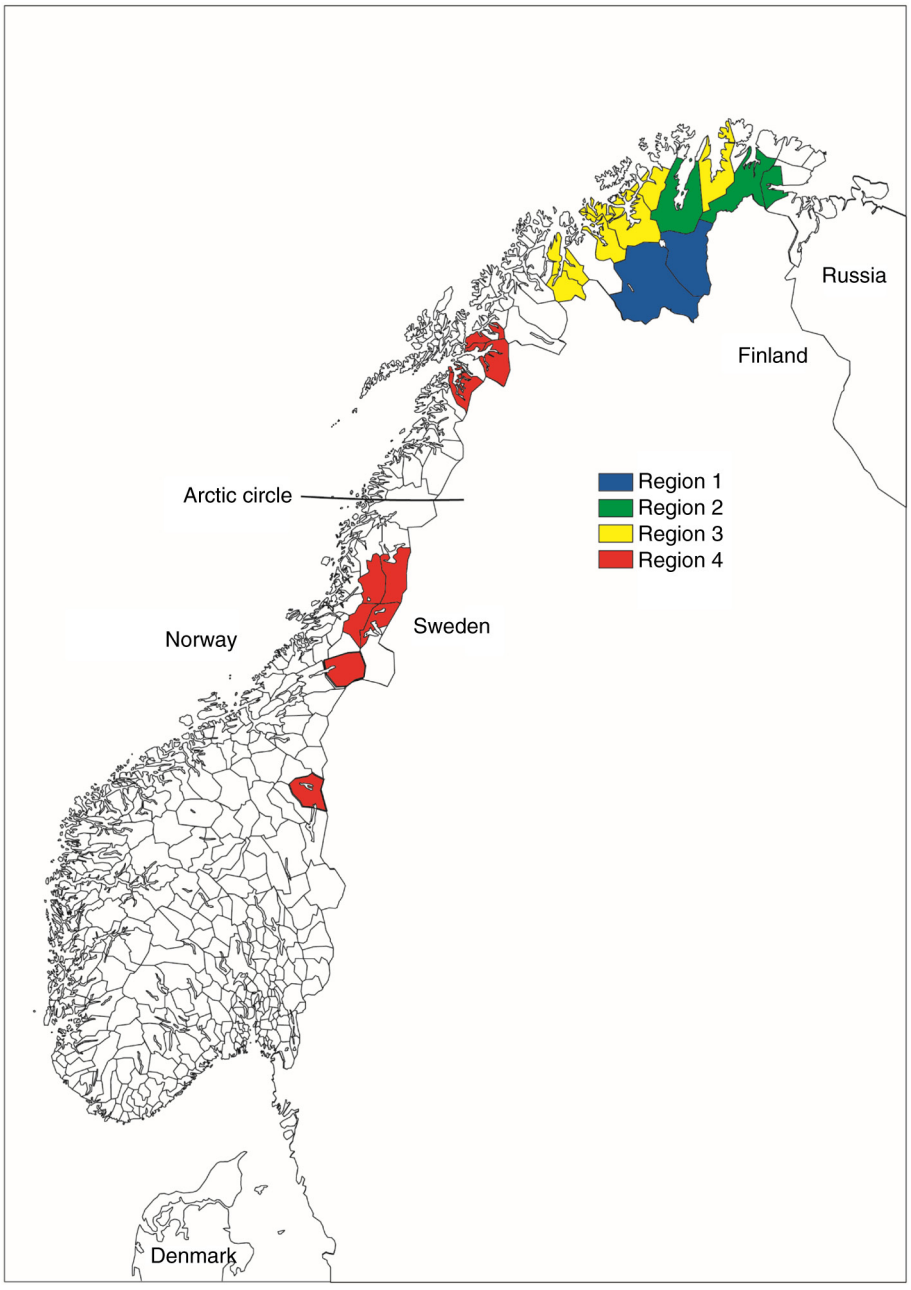

Fig. 1. The four geographical regions and the municipalities in each region.

overadjustment. Although the questionnaire contained several questions related to lifestyle and socio-economic status, we decided not to include them in the final analysis. The answers to these questions were neither precise nor objective. Furthermore, these factors may have altered since the onset of the disease. There were also many missing values in these variables which could have reduced the statistical strength. The direct method was applied to age-standardise the prevalence of pre-diabetes and diabetes mellitus using the European standard population of 2013 (10). Total prevalence of pre-diabetes and diabetes mellitus for each sex and ethnic group was adjusted according to regional differences in working sample rates. To achieve these adjusted values, the regional prevalence was weighted inversely proportional to the corresponding final working sample percentages (Table I). Multinomial logistic regression stratified by gender and the four geographical regions was used to evaluate age-adjusted relationship between ethnicity (main predictor) and dysglycaemia (outcome). The measure of association is presented as relative risk ratio ($\text{rrr} = exp(\beta)$), where β is the beta coefficient of the ethnicity variable in the multinomial logistic regression model.

Table I. Number of the invitees, participation rates and final working sample in each geographic region by sex (The SAMINOR1 study 2003–2004)

	Invited	Total participation (%)	Working sample (%)
Men	14,114	7,985 (56.6)	7,315 (51.8)
Region 1	1,419	840 (59.2)	528 (37.2)
Region 2	2,202	1,307 (59.4)	1,063 (48.3)
Region 3	7,293	4,186 (57.4)	4,108 (56.3)
Region 4	3,200	1,652 (51.6)	1,616 (50.5)
Women	13,037	8,553 (65.6)	7,893 (60.5)
Region 1	1,285	937 (72.9)	641 (49.9)
Region 2	1,972	1,380 (70.0)	1,158 (58.7)
Region 3	6,785	4,461 (65.7)	4,357 (64.2)
Region 4	2,995	1,775 (59.3)	1,737 (58.0)

Note: We excluded those with unknown ethnicity or ethnicity other than Sami, Norwegian or Kven and those with unknown plasma glucose values from the working sample.

Results

Study sample

Due to a low participation rate among 30-year-olds, they were excluded from the study, leaving 27,151 invitees aged 36–79 years. Of these, 16,538 (60.9%) agreed to participate and gave consent to medical research. Participants who reported their ethnic and linguistic background to be other than Sami, Norwegian or Kven or who had missing answers to these questions were excluded (n = 511), as were those with missing plasma glucose levels (n = 819). Thus, 15,208 participants were finally included in the present analysis (Table I).

Of the 15,208 participants included in the study sample, 696 (4.6%) were defined as having diabetes mellitus and 426 (2.8%) as having pre-diabetes. Among those defined as having diabetes mellitus, 636 (91.4%) reported diabetes in the questionnaire, whereas 60 (8.6%) were diagnosed only by non-fasting plasma glucose (data not shown). Table II shows age distribution of participants of both ethnic groups.

Little or no ethnic difference was seen in the total age-standardised prevalence of pre-diabetes and diabetes mellitus in either sex. Total age-standardised prevalence of pre-diabetes and diabetes mellitus for Sami men was 3.4 and 5.5%, respectively. Corresponding values for non-Sami men was 3.3 and 4.6%. Total age-standardised prevalence of pre-diabetes and diabetes mellitus for Sami women was 2.7% and 4.8%, respectively, while corresponding values for non-Sami women were 2.3 and 4.5% (Table III). In both ethnic groups, the prevalence of pre-diabetes and diabetes mellitus increased considerably with age.

Both Sami men and women had their highest prevalence of pre-diabetes and diabetes in Region 4. While non-Sami men had their highest prevalence of pre-diabetes and diabetes in Region 1, non-Sami women had their lowest prevalence of diabetes in this region (Table IV).

In Region 1, the relative risk of having diabetes was significantly lower among Sami men than among non-Sami men (rrr = 0.29) after adjustment for age. The same was observed for Sami women in Region 2 (rrr = 0.46). In Region 4, the situation was reversed, with a relative risk for diabetes mellitus that was significantly higher (rrr = 2.87 for men and rrr = 2.38 for women) in both Sami men and women than in their non-Sami counterparts. Relative risk for pre-diabetes was also significantly higher for Sami men compared with non-Sami men in this region (rrr = 2.05) (Table V).

Table II. Age distribution of the participants by sex, ethnicity and geographical region (the SAMINOR1 study 2003–2004)

Age (years)[a]	n	Sami	n	Non-Sami	p
Men					
Region 1	458	53.3 (52.4–54.3)	70	53.2 (50.7–55.8)	0.94
Region 2	478	55.6 (54.6–56.6)	585	53.8 (52.0–54.6)	0.005
Region 3	541	55.6 (54.7–56.6)	3,567	54.4 (54.1–54.8)	0.017
Region 4	193	54.8 (53.3–56.4)	1,423	56.4 (55.9–57.0)	0.063
Total	1,670	54.9 (54.4–55.4)	5,645	54.8 (54.6–55.1)	0.82
Women					
Region 1	554	53.1 (52.2–54.1)	87	52.4 (50.1–54.7)	0.58
Region 2	504	54.1 (53.2–55.1)	654	53.6 (52.7–54.4)	0.39
Region 3	489	54.7 (53.7–55.8)	3,868	54.2 (53.8–54.5)	0.30
Region 4	181	54.8 (53.1–56.5)	1,556	55.8 (55.3–56.4)	0.26
Total	1,728	54.0 (53.5–54.6)	6,165	54.5 (54.2–54.8)	0.14

Values are mean in years with 95% confidence interval (in parenthesis).
[a]Tested by two sample *t*-tests with equal variances.

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Table III. Prevalence of pre-diabetes and diabetes mellitus by sex, age and ethnic group (the SAMINOR1 study, 2003–2004)

Men							
	Sami			Non-Sami			
Age (years)	n	Pre-diabetes	Diabetes	n	Pre-diabetes	Diabetes	p[a]
36–49	576	10 (1.7%)	10 (1.7%)	1,957	41 (2.1%)	29 (1.5%)	0.79
50–59	552	20 (3.6%)	29 (5.2%)	1,776	60 (3.4%)	73 (4.1%)	0.49
60–79	542	26 (4.8%)	51 (9.4%)	1,912	87 (4.5%)	154 (8.0%)	0.57
Total crude	1,670	56 (3.3%)	90 (5.4%)	5,645	188 (3.3%)	256 (4.5%)	0.35
Total age-standardised[b] (95% CI)		3.4% (2.5–4.2%)	5.5% (4.4–6.6%)		3.3% (2.9–3.8%)	4.6% (4.1–5.2%)	
Women							
	Sami			Non-Sami			
Age (years)	n	Pre-diabetes	Diabetes	n	Pre-diabetes	Diabetes	
36–49	687	11 (1.6%)	10 (1.7%)	2,272	24 (1.1%)	38 (1.7%)	0.41
50–59	521	16 (3.1%)	15 (2.9%)	1,832	44 (2.4%)	78 (4.3%)	0.26
60–79	520	18 (3.5%)	49 (9.4%)	2,061	69 (3.3%)	156 (7.6%)	0.37
Total crude	1,728	45 (2.6%)	78 (4.3%)	6,165	137 (2.2%)	272 (4.4%)	0.63
Total age-standardised[a] (95% CI)		2.7% (1.9–3.4%)	4.8% (3.7–5.9%)		2.3% (1.9–2.6%)	4.5% (4.0–5.1%)	

[a]p-values show the significance level in Pearson's chi-square test
[b]Direct standardisation using European standard population of 2013 as reference.
CI, confidence interval.

Table IV. Crude regional and total prevalence of pre-diabetes and diabetes mellitus, together with total prevalence adjusted for regional working sample by sex, geographical region and ethnic group (the SAMINOR1 study, 2003–2004)

Men						
	Sami			Non-Sami		
	n	Pre-diabetes	Diabetes	n	Pre-diabetes	Diabetes
Region 1	458	12 (2.6%)	13 (2.8%)	70	3 (4.3%)	6 (8.6%)
Region 2	478	13 (2.7%)	24 (5.0%)	585	14 (2.4%)	22 (3.8%)
Region 3	541	19 (3.5%)	35 (6.5%)	3,567	191 (3.3%)	171 (4.8%)
Region 4	193	12 (6.2%)	18 (9.3%)	1,423	52 (3.6%)	57 (4.0%)
Crude total	1,670	56 (3.3%)	90 (5.4%)	5,645	188 (3.3%)	256 (4.5%)
Region-adjusted prevalence[a]		3.6%	5.7%		4.4%	4.4%
Women						
	Sami			Non-Sami		
	n	Pre-diabetes	Diabetes	n	Pre-diabetes	Diabetes
Region 1	554	15 (2.7%)	29 (5.2%)	87	2 (2.3%)	2 (2.3%)
Region 2	504	10 (2.0%)	12 (2.4%)	654	18 (2.7%)	31 (4.7%)
Region 3	489	12 (2.4%)	23 (4.7%)	3,868	71 (1.8%)	181 (4.7%)
Region 4	181	8 (4.4%)	14 (7.7%)	1,556	46 (3.0%)	58 (3.7%)
Crude total	1,728	45 (2.6%)	78 (4.5%)	6,165	137 (2.2%)	272 (4.4%)
Region-adjusted prevalence[a]		2.8%	4.8%		2.2%	4.3%

[a]Weighted according to regional working sample rates (see Table I).

Table V. Age-adjusted relative risk ratios (rrr[a]) for pre-diabetes and diabetes mellitus for Sami compared to non-Sami participants in different regions (the SAMINOR1 study 2003–2004)

	Pre-diabetes			Diabetes		
n	rrr	p	95% CI	rrr	p	95% CI
Men						
Region 1						
526	0.56	0.38	0.15–2.04	0.29	0.02	0.10–0.82
Region 2						
1,059	1.12	0.76	0.52–2.42	1.24	0.48	0.68–2.26
Region 3						
4,104	1.03	0.90	0.63–1.69	1.29	0.20	0.87–1.88
Region 4						
1,610	2.05	0.03	1.06–3.96	2.87	0.00	1.63–5.06
Women						
Region 1						
638	1.18	0.82	0.26–5.30	2.23	0.28	0.52–9.64
Region 2						
1,155	0.68	0.33	0.31–1.49	0.46	0.03	0.23–0.91
Region 3						
4,337	1.31	0.39	0.70–2.43	0.97	0.91	0.62–1.53
Region 4						
1,733	1.63	0.21	0.75–3.53	2.38	0.01	1.28–4.43

[a]The measure of association is presented as relative risk ratio (rrr) = exp(β), where β is the beta coefficient of the ethnicity variable in the multinomial logistic regression model.
95% CI, 95% confidence interval.

Discussion

In this study, we found statistically significant differences in the relative risk of diabetes mellitus between the Sami and non-Sami populations in some geographical regions. While the odds of having diabetes were lower for Sami men in Region 1 and Sami women in Region 2, the opposite was seen in the southern region, where the Sami were more prone to diabetes mellitus. Except for men in Region 4, prevalence of pre-diabetes was not significantly different between the Sami and non-Sami populations.

Two other studies based on data from the SAMINOR1 study have focused on diabetes prevalence. Nystad in her PhD showed no difference in the prevalence of type 2 diabetes mellitus between Sami and non-Sami populations (11). However, the definition of Sami ethnicity in Nystad's study focused more on linguistic features. Moreover, that study considered only self-reported diabetes and did not take into account regional differences. It is worth mentioning that if we merged participants of the same ethnicity from all the geographical regions we considered, there would be no statistically significant difference between the two ethnic groups. Broderstad and Melhus showed that although there was no ethnic difference in the prevalence of diabetes, ethnicity appeared to affect the type of diabetes treatment (12).

The HUNT3 study was conducted in 2006 in North Trøndelag county in the middle part of Norway and reported a prevalence of known (i.e. previously diagnosed) diabetes mellitus of 4.9 and 3.9%, respectively, in men and women aged ≥20 years. However, the prevalence of undiagnosed type 2 diabetes mellitus was estimated to be as high as that of known type 2 diabetes (7). However, considering the higher age of our participants (≥36), our use of non-fasting plasma glucose to diagnose diabetes mellitus and the heterogeneity of the prevalence of diabetes mellitus in the different geographical regions, it would be challenging to compare the results. In a follow-up study of the first Finnmark study (1974–1975), it was established that Sami women were more obese but did not have a higher incidence of diabetes mellitus than other women (13). Our findings were similar to that of the Finnmark study, which indicated that Sami women had higher truncal obesity (results not shown) but not a significantly higher rate of pre-diabetes or diabetes mellitus. In another study recently conducted in Greenland, the prevalence of type 2 diabetes among the Inuit was estimated around 9%, of which 79% were previously unknown cases (14). In a cross-sectional study, the prevalence of diabetes mellitus varied among the three Alaskan Inuit populations, with the Siberian Yupik (9.6%) having the highest rates, followed by the Central Yupik (2.8%) and Inupiat participants (3.7%). In the Alaskan study, diabetes was more prevalent in women than in men (8.8% vs. 4.2%), and of the people identified with diabetes in the study, 47% had not been previously diagnosed (15).

In contrast to these studies, the prevalence of undiagnosed diabetes mellitus was not so high in our study (8.6%). This may be the result of an effective and affordable health system in Norway, with sufficient coverage in rural areas with indigenous inhabitants. Another explanation for this may be the low sensitivity of non-fasting blood glucose to diagnose diabetes mellitus.

In 2004, the estimated sex- and age-standardised prevalence of known diabetes mellitus among those aged ≥30 years in Norway was 3.4% (6). Although this prevalence was lower than ours, the age composition of participants and the methods applied to diagnose diabetes mellitus were rather different from ours, making it difficult to compare the results. In 2002, the prevalence of diabetes among people aged 45–64 years in Iceland was reported to be 4.9% in men and 2.9% in women, reflecting an increase of around 50% over a period of 30 years (16). Previous estimates of age- and sex-specific prevalence of known diabetes mellitus in Denmark, Finland and Sweden are also comparable to our results (17–19).

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In our study, we compared the prevalence of pre-diabetes and diabetes mellitus between the Sami and non-Sami and found a heterogeneity across sexes and geographical regions. The four geographical regions that we considered in our study all have their own characteristic features such as location, climate, majority or minority status of the Sami population, implementation of preservation measures for Sami language, dialect, diet and religion.

In Region 1, Inland in Finnmark County, the Sami comprise 80–90% of the population (9), and some of the most important Sami-related institutions, such as the Sami Parliament and Sami University College (Sámi allaskuvla), are located there. Reindeer husbandry is more prevalent here than in other regions; hence, it is quite natural that reindeer is a large part of the diet of the inhabitants (2). In this region, Sami men had significantly lower prevalence of diabetes mellitus. Although Sami women were more obese than their non-Sami counterparts, no significant differences were observed in their prevalence of pre-diabetes or diabetes mellitus.

In Region 2, the Sami account for about half of the population (9). The municipalities in this region have both coastal and inland regions, with many farmers, fishermen and reindeer herders. The prevalence of diabetes in this region was significantly lower in Sami women than their non-Sami counterparts.

Region 3 represents a traditional coastal Sami population. Assimilation policies (Norwegianisation process) had a huge effect in these coastal regions (20), and in most of these municipalities, the Sami are now a minority. We found no ethnic difference in pre-diabetes or diabetes mellitus prevalence in this region.

Region 4 has a more heterogeneous population than the other regions. Three distinct Sami groups inhabit this region: the Marka Sami, Lule Sami and South Sami. Each has their own Sami language. By the second half of the 19th century, the Sami languages were already in retreat in this region (21). The proportion of the population with Sami ethnicity is lower in this region than in any of the other geographical regions we investigated (9). The prevalence of diabetes mellitus among the Sami in this region was more than twice as high as that among the non-Sami population. It is not clear which factor is responsible for this high prevalence. However, one interesting common feature observed in the groups with the highest prevalence of diabetes mellitus (the Sami in Region 4 and the non-Sami in Regions 1 and 2) was that they lived in a minority setting. Further studies need to be performed to clarify this phenomenon.

Strength and limitations

A relatively high participation rate (60.9%) and large sample size (15,208) in 24 municipalities made it possible for us to perform an in-depth analysis of diabetes status and related explanatory variables. As opposed to former studies on the prevalence of dysglycaemia, we were able to take into account the difference between geographical regions from which participants were recruited and heterogeneity across ethnic groups.

In our analysis, definition of the Sami was based on whether participants self-identified as Sami or had a Sami ethnic background, and if they, their parents or grandparents spoke Sami. This definition is rather different from the definitions of Sami used in the Finnmark study, "Ung i Nord" (The North Norwegian Youth Study) or former publications from the SAMINOR1 study, which used language as a basis. We chose to emphasise self-identification, as the Sami language has been subject to discrimination and stigmatisation and much of it might have been lost (22). The difference in how Sami ethnicity was defined might make comparison between our results and those from other studies difficult (23).

In this study, we used both self-reported diabetes and non-fasting plasma glucose to ascertain diabetes mellitus status. A non-fasting plasma glucose value of 11.1 mmol/L (200 mg/dl) or greater, together with symptoms, is an established diagnostic criterion for diabetes, but this method is not very reliable. The reliability of this diagnostic criterion is affected by the natural fluctuations of blood glucose throughout the day and can usually only detect diabetes that is poorly controlled (24). By the time this study was performed, HbA1c had not been standardised and approved to be applied for diagnosing diabetes mellitus. The SAMINOR1 study had a large number of participants attending per day, thus it was not feasible to conduct a 2-hour plasma glucose tolerance test. It was furthermore inadvisable to have participants arrive at the medical station after overnight fasting, as the time schedule was distributed during the day. In the present study, we did not perform any medical examination to find signs and symptoms of hyperglycaemia nor did we use other tests such as the glucose tolerance test or fasting plasma glucose to confirm the results of non-fasting plasma glucose tests. Furthermore, the use of self-reported information on diabetes may lead to some uncertainty and misclassification. Indeed, although some studies have proven that questionnaires are a convenient, yet valid, tool for studying chronic diseases such as diabetes and have satisfactory concordance with medical records (25), the validity of the self-administered questionnaire used in the SAMINOR1 study has not yet been determined.

In the present study, we did not distinguish between type 1, type 2 and gestational diabetes due to a lack of information and the need for exhaustive tests. Considering that around 80% of diabetes cases are type 2 diabetes mellitus (26), and given the age of the participants (36–79 years), we assumed that almost all of the cases in our study were of type 2 diabetes mellitus.

The present study had a cross-sectional design, making it difficult to assess potential causal relationships due to temporal bias. We decided not to include physical activity due to the possibility of temporal bias, which might have obscured the relationship between exposure and outcome. Moreover, diabetes or its comorbidities and/or complications might have altered the health-related behaviour and attitudes of those affected. Education was also excluded from the regression analyses as no confounding effect was observed for it. In addition, those risk factors which were part of metabolic syndrome like hypertension, dyslipidaemia and obesity were not included in the regression analysis to avoid overadjustment.

As we stratified the data by sex and region, we reduced the number of participants in each regression analysis and consequently reduced the statistical strength. An uneven distribution of participants from different ethnic groups in different geographical regions exacerbated this problem.

Non-responders tended to be younger, single and male (27), but other than this, there was very limited information, making it difficult to assess potential selection bias. As it was not possible to determine the response rate by ethnicity, it is not possible to attribute the pure burden and differences in the prevalence of pre-diabetes or diabetes mellitus to differences in participation rates. Another limitation of the study is that it was conducted in 2003–2004. Considering the relatively long time since then, caution should be exercised before applying the results to present-day populations.

Conclusion

The most striking finding in our study was the heterogeneity in the prevalence of pre-diabetes and diabetes mellitus in different geographical regions. While the prevalence of diabetes mellitus was lower in the Sami population of some northern regions, it was much higher in the southern region compared with their non-Sami counterparts. In future, further studies should be performed to address the potential explaining factors behind the observed heterogeneous discrepancies between the prevalence of pre-diabetes and diabetes mellitus in the two ethnic groups. Preventive measures should be implemented at the population level to reduce the levels of established risk factors for developing diabetes, with a special focus on those with pre-diabetes and people living in regions where a higher prevalence of diabetes mellitus has been reported.

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References

1. Niemi E. The Finns in Northern Scandinavia and minority policy. In: Tägil S, editor. Ethnicity and nation building in the Nordic world. London: Hurst; 1995. p. 145–78.
2. Brustad M, Parr CL, Melhus M, Lund E. Dietary patterns in the population living in the Sami core areas of Norway – the SAMINOR study. Int J Circumpolar Health. 2008;67:82–96.
3. Ross AB, Johansson A, Ingman M, Gyllensten U. Lifestyle, genetics, and disease in Sami. Croat Med J. 2006;47:553–65.
4. Danaei G, Finucane MM, Lu Y, Singh GM, Cowan MJ, Paciorek CJ, et al. National, regional, and global trends in fasting plasma glucose and diabetes prevalence since 1980: systematic analysis of health examination surveys and epidemiological studies with 370 country-years and 2.7 million participants. Lancet. 2011;378:31–40.
5. Zabetian A, Sanchez IM, Narayan KM, Hwang CK, Ali MK. Global rural diabetes prevalence: a systematic review and meta-analysis covering 1990–2012. Diabetes Res Clin Pract. 2014;104:206–13.
6. Stene LC, Midthjell K, Jenum AK, Skeie S, Birkeland KI, Lund E, et al. [Prevalence of diabetes mellitus in Norway]. Tidsskr Nor Laegeforen. 2004;124:1511–4.
7. Krokstad S, Knudtsen MS. Folkehelse i endring: Helseundersøkelsen Nord-Trøndelag: HUNT 1 (1984–86) – HUNT 2 (1995–97) – HUNT 3 (2006–08). Levanger, Norway: HUNT forskningssenter; 2011.
8. Jorgensen ME, Bjeregaard P, Borch-Johnsen K, Backer V, Becker U, Jorgensen T, et al. Diabetes and impaired glucose tolerance among the Inuit population of Greenland. Diabetes Care. 2002;25:1766–71.
9. Lund E, Melhus M, Hansen KL, Nystad T, Broderstad AR, Selmer R, et al. Population based study of health and living conditions in areas with both Sami and Norwegian populations – the SAMINOR study. Int J Circumpolar Health. 2007;66:113–28.
10. Eurostat. Revision of the European standard population – report of Eurostat's task force. Luxembourg: Eurostat; 2013.
11. Nystad TA. A population-based study on cardiovascular risk factors and self-reported type 2 diabetes mellitus in the Sami population. Tromsø: The Arctic University of Norway; 2010.
12. Broderstad AR, Melhus M. Prevalence of metabolic syndrome and diabetes mellitus in Sami and Norwegian populations. The SAMINOR – a cross-sectional study. BMJ Open. 2016;6: e009474.
13. Njolstad I, Arnesen E, Lund-Larsen PG. Cardiovascular diseases and diabetes mellitus in different ethnic groups: the Finnmark study. Epidemiology. 1998;9:550–6.
14. Jorgensen ME, Borch-Johnsen K, Witte DR, Bjerregaard P. Diabetes in Greenland and its relationship with urbanisation. Diabet Med. 2012;29:755–60.
15. Ebbesson SO, Schraer CD, Risica PM, Adler AI, Ebbesson L, Mayer AM, et al. Diabetes and impaired glucose tolerance in three Alaskan Eskimo populations. The Alaska-Siberia Project. Diabetes Care. 1998;21:563–9.
16. Bergsveinsson J, Aspelund T, Gudnason V, Benediktsson R. Prevalence of type 2 diabetes mellitus in Iceland 1967–2002. Laeknabladid. 2007;93:397–402.
17. Glumer C, Jorgensen T, Borch-Johnsen K Inter99 study. Prevalences of diabetes and impaired glucose regulation in a Danish population: the Inter99 study. Diabetes Care. 2003;26: 2335–40.
18. Rajala U, Keinanen-Kiukaanniemi S, Uusimaki A, Reijula K, Kivela SL. Prevalence of diabetes mellitus and impaired glucose tolerance in a middle-aged Finnish population. Scand J Prim Health Care. 1995;13:222–8.

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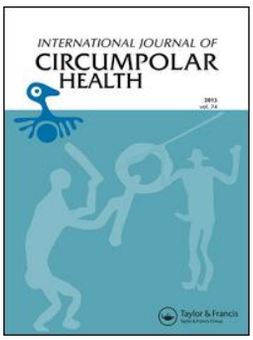
19. Jansson SP, Fall K, Brus O, Magnuson A, Wandell P, Ostgren CJ, et al. Prevalence and incidence of diabetes mellitus: a nationwide population-based pharmaco-epidemiological study in Sweden. Diabet Med. 2015;32:1319–28.
20. Minde H. Assimilation of the Sami: implementation and consequences. Kautokeino: Galdu Resource Centre for the Rights of Indigenous Peoples; 2005.
21. Hansen L, Evjen B. Kjært barn – mange navn: Om forskjellige betegnelser på den samiske befolkningen i Nordland gjennom århundrene. In: Hansen L, Evjen B, editors. Nordlands kulturelle mangfold: Etniske relasjoner i historisk perspektiv. Oslo: Pax Forlag; 2008. p. 17–47.
22. Stordal V. Samene: Fra "lavtstaaende race" til "urbefolkning. In: Eriksen TH, editor. Flerkuturell forståelse. Oslo: Tano Aschehaug; 1997. p. 139–52.
23. Pettersen T, Brustad M. Which Sami? Sami inclusion criteria in population-based studies of Sami health and living conditions in Norway – an exploratory study exemplified with data from the SAMINOR study. Int J Circumpolar Health. 2013; 72:21813, doi: http://dx.doi.org/10.3402/ijch.v72i0.21813
24. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Report of the Expert Committee on the diagnosis and classification of diabetes mellitus. Diabetes Care. 1997;20:1183–97.
25. Skinner KM, Miller DR, Lincoln E, Lee A, Kazis LE. Concordance between respondent self-reports and medical records for chronic conditions: experience from the Veterans Health Study. J Ambul Care Manage. 2005;28:102–10.
26. Velho G, Froguel P, Mann J, Toeller M. Type 2 diabetes. In: Ekoe J, Zimmet P, Williams R, editors. The epidemiology of diabetes mellitus. Chichester, England: Wiley; 2001. p. 133–53.
27. Nystad T, Melhus M, Brustad M, Lund E. Ethnic differences in the prevalence of general and central obesity among the Sami and Norwegian populations: the SAMINOR study. Scand J Public Health. 2010;38:17–24.

Paper II



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Prevalence of pre-diabetes and type 2 diabetes mellitus among Sami and non-Sami men and women in Northern Norway – The SAMINOR 2 Clinical Survey

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ABSTRACT

The aim of this study was to determine and compare the prevalence of pre-diabetes and type 2 diabetes mellitus (T2DM) among Sami and non-Sami men and women of rural districts in Northern Norway. The SAMINOR 2 Clinical Survey is a cross-sectional population-based study performed in 2012–2014 in 10 municipalities of Northern Norway. A total of 12,455 Sami and non-Sami inhabitants aged 40–79 years were invited to participate and 5878 were included in the analyses. Participants with self-reported T2DM and/or a glycated haemoglobin (HbA1c) result ≥6.5% were categorised as having T2DM. Those with 5.7%≤HbA1c<6.5% were categorised as pre-diabetics. In men, the total age-standardised prevalence of pre-diabetes (37.9% vs 31.4%) and T2DM (10.8% vs 9.5%) were higher in Sami compared with non-Sami; the ethnic difference was statistically significant for both pre-diabetes (OR 1.42, $p < 0.001$) and T2DM (OR 1.31, $p = 0.042$). In women, pre-diabetes (36.4% vs 33.5%) and T2DM (8.6% vs 7.0%) were also more common in Sami than non-Sami; the differences in both pre-diabetes (OR 1.20, $p = 0.025$) and T2DM (OR 1.38, $p = 0.021$) were also statistically significant. The observed ethnic difference in the waist-to-height ratio (WHtR) was a plausible explanation for the ethnic difference in the prevalence of pre-diabetes and T2DM.

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HbA1c; Norwegian; indigenous; native; aboriginal; ethnicity; ethnic minority; abdominal obesity; waist-to-height ratio

Introduction

The prevalence of type 2 diabetes mellitus (T2DM) is increasing globally. In 2014 it was estimated that 422 million people worldwide were affected by the disease, and the prevalence of diabetes mellitus (DM) among adults over 18 years of age reached to 8.5% [1]. In 2011, the direct costs of DM treatment in Norway amounted to €408 million; indirect costs amounted to €108 million [2]. There has been no nation-wide survey from Norway on the prevalence of diabetes, but in 2013, it was reported that 2.7% of the country's population was being treated with glucose-lowering medications [3], and the annual number of new users of glucose-lowering medications in Norway levelled off in recent years [4]. However, there are many individuals who remain undiagnosed of T2DM, or who received a diagnosis but manage their T2DM solely by changes in diet and/or physical activity [5].

The Sami are an indigenous people whose traditional settlement area (Sápmi) covers the northern parts of Norway, Sweden and Finland, and the Kola Peninsula of Russia [6]. However, many Sami are today settled outside Sápmi, especially in larger cities [7]. No valid or updated demographic record of the Sami exists. However, rough estimates of the total number of Sami tend to vary between 50,000 and 100,000, of whom 40,000–50,000 are settled in Norway [8]. The Sami harbour a rich variety of cultures, traditions and languages, but for many decades they were subjected to discrimination and assimilation policies; consequently, many Sami abandoned their native culture and language [9].

The Population-based Study on Health and Living Conditions in Regions with Sami and Norwegian Populations (the SAMINOR Study) aims to investigate the health and living conditions of the Sami and non-Sami people in northern parts of Norway. While the prevalence of lifestyle-related diseases and metabolic syndrome is generally higher among indigenous people as compared to general populations [10,11], studies

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based on data from the SAMINOR 1 Survey (2003–2004) and the SAMINOR 2 Clinical Survey (2012–2014, hereinafter referred to as SAMINOR 2), found overall high, yet rather similar prevalence of DM in the Sami and non-Sami populations [12–13]. In these studies, DM was recognised by self-report and/or non-fasting plasma glucose measurements. Nevertheless, further studies on the prevalence of DM in these populations, using a more reliable and valid disease indicator, are warranted.

Therefore, the aim of this study was to use HbA1c measurements together with self-reported T2DM collected in SAMINOR 2 to determine and compare the prevalence of pre-diabetes and T2DM among Sami and non-Sami men and women of rural districts in Northern Norway.

Methods

The present analyses are based on cross-sectional data from SAMINOR 2 which was conducted by the Centre for Sami Health Research at UiT The Arctic University of Norway in 2012–2014. The survey included inhabitants from ten of the municipalities of Finnmark, Troms, and Nordland counties: Kautokeino, Karasjok, Porsanger, Tana, Nesseby, Lyngen, Storfjord, Kåfjord, Skånland and Evenes (Figure 1). All inhabitants in the selected region (i.e. registered in the National Registry of Norway as resident in one of the mentioned municipalities) aged 40–79 years (n = 12,455) were invited to participate, regardless of ethnic background. The survey included a self-administered questionnaire and a clinical examination, including collection of a blood sample. Of the 12,455 inhabitants, 6004 (48.2%) attended. Of these 6004, 21 were excluded due to uncompleted questionnaires, 22 were excluded due to missing glycated haemoglobin (HbA1c) results, 72 participants were excluded due to missing ethnicity variable, and 11 with type 1 DM were excluded. Hence, 5878 individuals (47.2%) were included in the analyses. The selected municipalities were divided into three different regions: "Region 1" comprised of areas in the inland of Finnmark County, including Kautokeino and Karasjok municipalities. "Region 2" consisted of both inland and coastal areas in Finnmark County, including Porsanger, Tana and Nesseby municipalities. "Region 3" was made up of the remaining municipalities, all located in Troms and Nordland counties (Lyngen, Storfjord, Kåfjord, Skånland and Evenes) (Figure 1).

The SAMINOR Study was approved by the Norwegian Data Inspectorate and by the Regional Committees for Medical and Health of Research Ethics North (REC North). The committee also approved the present study. All participants gave written informed consent for medical research.

Data collection

Invitations were mailed several weeks before the clinical examinations started in each municipality. The invitation contained relevant information about the survey, including the time and place of the clinical examination, and the study questionnaire. Participants were to hand in their completed questionnaires at the time of the clinical examination, which was performed at one of ten research stations established in nine municipalities (two research stations were set up in Kåfjord, while

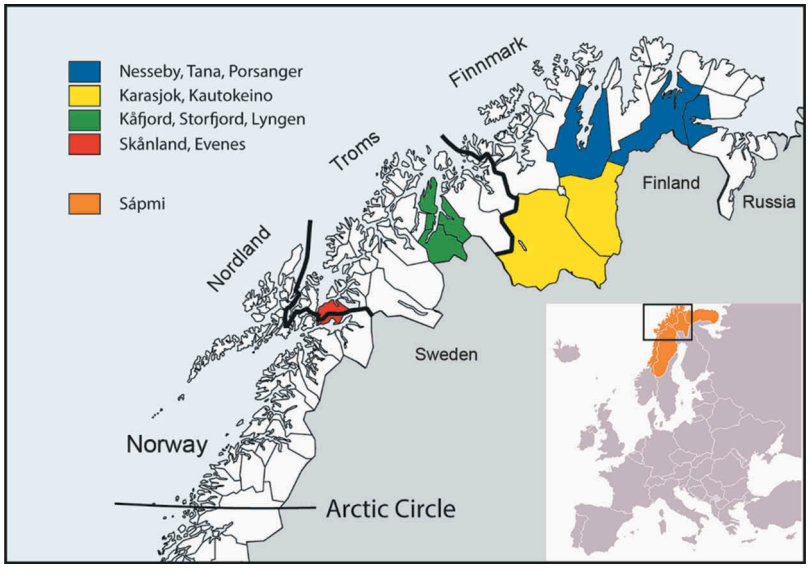

Figure 1. Map of Northern Norway, Sápmi and the included municipalities in the SAMINOR 2.

participants living in Evenes visited the research station in neighbouring Skånland). All clinical examinations were performed within 2–7 weeks in each municipality.

During the clinical examination, trained personnel measured participants' height (to the nearest 0.1 cm) and weight (to the nearest 100 g) using an electronic height and weight scale (DS-103, Dongsahn Jenix, Seoul, Korea) with participants wearing light clothing and no shoes. These measures were then used to calculate body mass index (BMI, kg/m^2). Waist circumference (WC) was measured at the umbilicus to the nearest cm with the participant standing and breathing normally. Waist-to-height ratio (WHtR) was calculated by dividing waist circumference by height. Whole blood samples collected by venipuncture were used for HbA1c testing using DCA Vantage™ (Siemens Medical Solutions Diagnostics, Tarrytown, NY, USA).

Questionnaires differed by age group: participants aged 40–69 years received an 8-page questionnaire that covered a broad range of questions on lifestyle, diet, risk factors and diseases. In contrast, participants aged 70–79 years received a 4-page questionnaire with fewer questions and larger fonts. The present study only included questions that were identical in the two questionnaires. Both questionnaires were originally prepared in Norwegian and then translated into the Northern Sami language. The questionnaires, also a translated English version of the 40–69 year questionnaire, may be reviewed at www.saminor.no. In Kautokeino, Karasjok, Nesseby, and Tana, invitees received both the Sami and Norwegian versions of the questionnaire. In Kåfjord, Storfjord, Porsanger and Lyngen, the Sami version was available on request. Invitees in Skånland and Evenes received the Norwegian questionnaire only. Among all of our participants, less than 5% chose to use the Sami version of the questionnaire.

Information on ethnicity was recorded based on participants' answers to the following 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 do you consider yourself to be?" The response options were: "Norwegian", "Sami", "Kven" (another national ethnic minority group) [14] and "Others". Participants were to apply the information for each of the mentioned relatives separately, and multiple languages/ethnicities were allowed. Participants were defined as Sami if they responded that they either considered themselves to be Sami or reported to have a Sami ethnic background, and if in addition at least one of their grandparents, parents, or they themselves spoke a Sami language at home. All participants who did not meet this criterion were defined as non-Sami.

Information on DM was taken from both questionnaires and HbA1c results. First, self-reported type 2 diabetics were ascertained. In the questionnaire this information came from the question: "Have you ever been diagnosed with diabetes (elevated blood sugar levels)?" The available answers were "yes" or "no". Missing values were classified as "no". If the participant answered "yes", they were asked about the type (type 1 DM, T2DM, or gestational diabetes). In addition to participants who reported T2DM, those who reported DM without specifying the type (56 participants) were also categorised as having T2DM. Moreover, those who reported having type 1 DM (T1DM), but reported taking glucose-lowering medication for its treatment (26 participants) or never using insulin (6 participants), were recategorised as having T2DM.

In addition to self-report, those with HbA1c ≥6.5% were also categorised as having T2DM. As virtually all individuals with T1DM are aware of their disease and are under treatment, all those who had high HbA1c (≥6.5%) without reporting diabetes in the questionnaires were regarded as having T2DM. Those who had 5.7%≤HbA1c<6.5% were categorised as pre-diabetics. The pre-diabetes category was defined based on HbA1c only.

Participants gave information on their level of physical activity on a scale of one (very low) to ten (very high). The participants were informed that household chores and professional activities as well as regular exercise and other physical activity, such as walking/hiking, should be taken into account when answering. This scale was validated in middle-aged women in Tromsø, Norway [15]. Educational attainment was reported in the questionnaire in years, and all completed school years were counted.

Statistical analysis

The data management and statistical analysis were done using STATA version 14.1 (StataCorp, College Station, TX, USA). Differences in mean age, education, physical activity score, height, weight, WHtR, BMI, and WC by sex and ethnic groups were assessed using two-sample *t*-tests. The prevalence of self-reported T2DM and categorised HbA1c was compared between groups using χ^2 tests (Table 1). Prevalence of pre-diabetes and T2DM is presented as percentages with 95% confidence intervals (CIs) by sex, age and for Sami versus non-Sami participants (Table 2). Due to large samples, CIs were calculated based on normal approximation. The direct method was used to age-standardise the prevalence of pre-diabetes and T2DM. To obtain estimates that better reflect the true prevalences in the selected

Table 1. Crude characteristics of participants in the SAMINOR 2 Clinical Survey (2012–2014), $n = 5878$[a].

	Total	Sami	Non-Sami	
	$n = 2688$	$n = 1114$	$n = 1574$	p-Value[b]
Men				
Age (years)	60.1	59.9	60.3	0.25
Education (years)	11.7	11.4	11.8	0.01
Physical activity (self-rated score)	5.2	5.1	5.2	0.24
Height (cm)	173.1	170.1	175.2	<0.01
WC (cm)	99.6	98.7	100.3	<0.01
WHtR	0.576	0.580	0.572	<0.01
Weight (kg)	84.7	82.0	86.6	<0.01
BMI (kg/m^2)	28.2	28.3	28.2	0.36
HbA1c<5.7%, *n* (%)	1456 (54.2)	556 (49.9)	900 (57.2)	0.01
5.7%≤HbA1c<6.5%, *n* (%)	1001 (37.2)	456 (40.9)	545 (34.6)	
6.5%≤HbA1c, *n* (%)	231 (8.6)	102 (9.2)	129 (8.2)	
Self-reported T2DM, *n* (%)	254 (9.4)	107 (9.6)	147 (9.3)	0.79
Women	$n = 3190$	$n = 1282$	$n = 1908$	
Age (years)	58.9	58.5	59.1	0.16
Education (years)	12.3	12.5	12.3	0.13
Physical activity (self-rated score)	5.4	5.2	5.6	<0.01
Height (cm)	160.0	156.8	162.2	<0.01
WC (cm)	93.2	93.6	92.9	0.13
WHtR	0.583	0.597	0.573	<0.01
Weight (kg)	71.6	70.0	72.7	<0.01
BMI (kg/m^2)	28.0	28.5	27.6	<0.01
HbA1c<5.7%, *n* (%)	1776 (55.7)	685 (53.4)	1091 (57.2)	0.11
5.7%≤HbA1c<6.5%, *n* (%)	1232 (38.6)	521 (40.6)	711 (37.3)	
6.5%≤HbA1c, *n* (%)	182 (5.7)	76 (6.0)	106 (5.5)	
Self-reported T2DM, *n* (%)	211 (6.6)	88 (6.9)	123 (6.4)	0.57

Numbers are mean unless stated otherwise.
[a]The number of participants for each variable may differ as some of the measures have missing values. The highest number of missing was for physical activity ($n = 278$).
[b]*p*-values are from two independent samples *t*-test or Pearson chi-square test.
WC: waist circumference; WHtR: waist-to-height ratio; BMI: body mass index; HbA1c: glycated haemoglobin; T2DM: type 2 diabetes mellitus.

municipalities and age groups, invitees in SAMINOR 2 were chosen as the standard population (age groups: 40–59, 60–69 and 70–79 years). Multinomial logistic regression analysis was used to calculate the odds ratio (OR) of pre-diabetes and T2DM for Sami compared to non-Sami, stratified by sex. For each sex, five models were run with dysglycaemia as dependent variable and ethnicity (Sami vs. non-Sami) as independent variable: In addition to ethnicity, the first model adjusted for age as continuous variable. In addition to age, the next four models also adjusted for each of the variables physical activity, education, BMI and WHtR, one at a time (Table 3). All these variables were treated as continuous. Comparison between men and women was also performed using multinomial logistic regression adjusted for age. All tests were two-sided with a 5% significance level.

Results

Some characteristics of the participants are shown in Table 1. Mean WHtR was higher in Sami men compared to their non-Sami counterparts. In women, both mean BMI and WHtR was significantly higher among Sami compared to non-Sami. On average, Sami women reported significantly lower physical activity than did their non-Sami counterparts (Table 1).

The overall age-standardised prevalence of self-reported T2DM was 7.4% (95% CI: 6.8–8.0) (results not shown). While more men than women reported T2DM, there was observed no ethnic difference in the prevalence of self-reported T2DM (Table 1). In total, 2083 (35.4%) individuals were ascertained as pre-diabetics (5.7%≤HbA1 < 6.5%) and 565 (9.4%) as type 2 diabetics (self-reported T2DM and/or 6.5%≤HbA1c). Of those who were categorised as having T2DM, 465 (82.3% of all cases) reported T2DM themselves (results not shown). The total age-standardised prevalence of pre-diabetes and T2DM were, respectively, 34.1% (95% CI: 33.1–35.1) and 8.7% (95% CI: 8.0–9.4) (results not shown).

In Sami men, the total age-standardised prevalences of pre-diabetes and T2DM were 37.9% and 10.8%, respectively. Corresponding numbers for non-Sami men were 31.4% and 9.5% (Table 2). The 95% confidence intervals of T2DM prevalence overlapped, but as this does not rule out statistical significance, multinomial logistic regression was performed. When adjusting for age as a continuous variable in a multinomial logistic regression, the ethnic difference was statistically significant for both pre-diabetes (OR 1.42, $p < 0.001$) and T2DM (OR 1.31, $p = 0.042$) (Table 3). In women, the age-standardised prevalences of pre-diabetes were 36.4% in Sami vs 33.5% in non-Sami and of T2DM 8.6% in Sami vs 7.0% in non-Sami (Table 2). The ethnic

Table 2. Prevalence of pre-diabetes and type 2 diabetes mellitus (T2DM) by sex, age and for Sami versus non-Sami participants. Pre-diabetes is based on 5.7%≤HbA1c<6.5% and T2DM is based on self-report and/or HbA1c≥6.5%.

	Men									
	Sami (n = 1114)					Non-Sami (n = 1574)				
Age (years)	n	Pre-D	% (95% CI)	T2DM	% (95% CI)	n	Pre-D	% (95% CI)	T2DM	% (95% CI)
40–59 years	511	168	32.9 (28.8–37.1)	36	7.0 (5.0–9.6)	686	185	27.0 (23.7–30.4)	31	4.5 (3.1–6.3)
60–69 years	388	160	41.2 (36.3–46.3)	61	15.7 (12.2–19.7)	562	195	34.7 (20.8–38.8)	81	14.4 (11.6–17.6)
70–79 years	215	105	48.8 (42.0–55.7)	32	14.9 (10.4–20.3)	326	132	40.5 (35.1–46.0)	57	17.5 (13.5–22.0)
Total crude	1114	433	38.9 (36.0–41.8)	129	11.6 (9.8–13.6)	1574	512	32.5 (30.2–34.9)	169	10.7 (9.2–12.4)
Total age-standardised* (95%CI)			37.9 (35.0–40.8)		10.8 (9.1–12.6)			31.4 (29.1–33.7)		9.5 (8.1–10.9)
	Women									
	Sami(n = 1282)					Non-Sami (n = 1909)				
Age (years)	n	Pre-D	% (95% CI)	T2DM	% (95% CI)	n	Pre-D	% (95% CI)	T2DM	% (95% CI)
40–59 years	670	181	27.0 (23.7–30.5)	23	3.4 (2.2–5.1)	933	220	23.5 (20.9–26.4)	31	3.3 (2.3–4.7)
60–69 years	403	182	45.2 (40.2–50.2)	55	13.6 (10.4–17.4)	613	272	44.4 (40.4–48.4)	51	8.3 (6.2–10.8)
70–79 years	209	110	52.6 (45.6–59.6)	35	16.7 (11.9–22.5)	362	173	47.8 (42.5–53.1)	62	17.1 (13.4–21.4)
Total crude	1282	473	36.9 (34.2–39.6)	113	8.8 (7.3–10.5)	1908	665	34.8 (32.7–37.0)	144	7.5 (6.4–8.8)
Total age-standardised[a] (95%CI)			36.4 (33.9–39.0)		8.6 (7.1–10.0)			33.5 (31.5–35.6)		7.0 (5.9–8.1)

The SAMINOR 2 Clinical Survey (2012–2014), n = 5878.
[a]The direct method using the invited sample in the SAMINOR 2 Clinical Survey as the reference population.
Pre-D: pre-diabetes; T2DM: type 2 diabetes mellitus; HbA1c: glycated haemoglobin; CI: Confidence interval.

Table 3. Odds ratios for pre-diabetes and type 2 diabetes mellitus (T2DM) for Sami compared to non-Sami stratified by sex. The SAMINOR 2 Clinical Survey (2012–2014), n = 5878.

	Pre-diabetes			Type 2 diabetes mellitus		
	OR Sami vs. non-Sami	95% CI	p-Value[a]	OR Sami vs. non-Sami	95% CI	p-Value[a]
Men						
Adjusted for[b]:						
Age	1.42	1.20–1.68	<0.001	1.31	1.01–1.70	0.042
Age + education	1.39	1.16–1.64	<0.001	1.23	0.94–1.61	0.123
Age + physical activity	1.38	1.17–1.65	<0.001	1.26	0.96–1.65	0.093
Age + BMI	1.41	1.18–1.67	<0.001	1.31	1.00–1.71	0.050
Age + WHtR	1.36	1.14–1.62	<0.001	1.19	0.91–1.56	0.197
Women						
Adjusted for[b]:	OR Sami vs. non-Sami	95% CI	p-Value[a]	OR Sami vs. non-Sami	95% CI	p-Value[a]
Age	1.20	1.02–1.41	0.025	1.38	1.05–1.82	0.021
Age + education	1.21	1.02–1.43	0.023	1.41	1.06–1.88	0.017
Age + physical activity	1.19	1.01–1.40	0.040	1.29	0.96–1.73	0.094
Age + BMI	1.12	0.95–1.32	0.166	1.22	0.92–1.63	0.166
Age + WHtR	1.05	0.89–1.23	0.589	1.00	0.74–1.34	1.00

[a]p-values present the statistical significance of the corresponding ORs for pre-diabetes or T2DM vs. normoglycaemics.
bNumber of individuals in each regression analysis may vary due to some missing values in each adjusted variable.

OR: odds ratio; CI: confidence interval; BMI: body mass index; WHtR: waist-to-height ratio.

differences in both pre-diabetes (OR 1.20, p = 0.025) and T2DM (OR 1.38, p = 0.021) were also herein statistically significant (Table 3).

Adjustment for WHtR had the largest impact on the OR for pre-diabetes and T2DM for Sami compared to non-Sami, especially in women (Table 3); after adjusting for WHtR, the OR for pre-diabetes in Sami versus non-Sami women was 1.05 (p = 0.589) and for T2DM 1.00 (p = 1.00).

In men, the observed prevalence of pre-diabetes and T2DM was higher in Sami in all geographical regions; statistically significant ethnic difference was, however, only found for pre-diabetes in region 2 and for T2DM in region 3 (results not shown).In women, the observed prevalence of pre-diabetes and T2DM was higher in Sami in all geographical regions but region 2, wherein fewer Sami had dysglycaemia. Statistically significant ethnic difference was, however, only observed for pre-diabetes in region 1 and for T2DM in regions 1 and 3 (results not shown).

Discussion

The overall age-standardised prevalence of pre-diabetes and T2DM in the 10 municipalities were, respectively, 34.1% and 8.7%. In spite of overlapping confidence intervals of age-standardised prevalence of pre-diabetes (in women) and T2DM (in both sexes) of Sami versus non-Sami participants, the age-adjusted ORs of pre-diabetes and T2DM for Sami versus non-Sami were statistically significant in both sexes. Furthermore, the prevalence of T2DM was statistically significantly higher in men. Ethnic differences in WHtR seems to be a plausible explanation for ethnic

difference in pre-diabetes and T2DM, especially in women as it explained the entire ethnic difference in pre-diabetes and T2DM.

Self-report of T2DM in combination with HbA1c results were used to categorise participants as having T2DM. HbA1c results reflect average plasma glucose concentration during the preceding 2–3 months [16]. The firm association between HbA1c results and late complications of DM was first documented in a Norwegian study [17]. Due to its high pre-analytical stability, high reproducibility, less day-to-day perturbations during periods of stress and illness, and convenience (no need for prior fasting or glucose overload), HbA1c is being increasingly utilised in medical settings for both diagnosis and follow-up of patients with DM [5]. In 2009, the International Expert Committee recommended the use of HbA1c to diagnose DM. However, they stressed that there was a continuum of risk for DM across HbA1c results [18], admitting that, although the risk of retinopathy escalates drastically at HbA1c ≥6.5%, the risk of developing DM and its other complications may clearly begin well under this cut-off [18].

In our study, more than one third of participants were diagnosed as having pre-diabetes. The American Diabetes Association recommend HbA1c≥5.7 for pre-diabetes [5]. The sensitivity of this cut-off is also quite low [19,20]. The American Diabetes Association recommends that individuals with HbA1c levels of 5.7–6.4% be informed of their increased risk for DM and cardiovascular diseases and counselled about effective preventive strategies such as weight reduction and increased physical activity [5]. It should be kept in mind that the risk of developing DM follows a continuum of risk rather than a certain cut-off [5]. However, different guidelines recommend that clinicians have two HbA1c results ≥6.5% to establish a diagnosis of DM [5,18,21,22]. In the Tromsø OGTT Study, the sensitivity, specificity, positive and negative predictive values for HbA1c ≥ 6.5% were, respectively, 34.7%, 97.1%, 41.2% and 96.1% using OGTT (oral glucose tolerance test) as gold standard [23]. As both the sensitivity and specificity of the HbA1c test are <100%, a misclassification in the outcome variable (T2DM) can be expected. This misclassification is most likely non-differential with regard to ethnic groups.

In this study, questionnaires were applied to acquire information on T2DM. As the performance of questionnaires may be affected by issues like recall bias, unawareness of the disease, or misinterpretation of the questions, self-reported data may be inadequate to reflect the true prevalence of a disease. In a study performed in Olmsted County, Minnesota, with 2037 participants aged ≥45 years, the sensitivity and positive predictive value of self-reported DM were 66.0% and 94.3%, respectively [24]. However, the CADEUS study in France reported a sensitivity and positive predictive value of self-reported DM of 86.7% and 73.4%, respectively [25]. All the mentioned studies used medical records as reference standard. It should be noted, however, that the phrasing of questions and types of criterion standard affect the sensitivity and positive predictive value of questionnaires [26]. Furthermore, some publications have reported that the Sami people may be more inclined than non-Sami to underreporting diseases due to some cultural differences and/or language barriers (differential misclassification) [27].

The ethnicity (exposure variable) of the participants was ascertained based on the obtained data from the questionnaires. Contrary to reporting non-Sami ethnicity, reporting Sami ethnicity demands a conscious choice. Due to decades of stigmatisations and histories of study misconduct exerted on the Sami people, there are still some Sami people who are hesitant to either participate in such studies or report their ethnicity as Sami. As a result, some Sami people may have been misclassified as non-Sami, while the opposite is extremely unlikely. This leads to a non-differential misclassification in the exposure variable. The joint effect of the mentioned misclassifications in the exposure and outcome variables might have diluted the measure of association in our study [28]. It is possible that the real difference between the Sami and non-Sami with regard to the prevalence of T2DM was higher than what was observed.

In our study, the estimated age-standardised prevalence of self-reported T2DM was 7.4%. Data from the Norwegian Prescription Database show that in 2014, 6.8% of inhabitants aged 40–79 years in the 10 municipalities included in our study were using oral glucose-lowering medications for T2DM. This may serve as a validation of our estimate of known cases of T2DM in the study population.

The observed difference in the prevalence of pre-diabetes and T2DM between Sami and non-Sami of the same sex in the present study is in discordance with results from previous studies [12,13,29]. This might be due to our use of HbA1c as the diagnostic test in contrast to previous studies which were based on self-report and/or non-fasting (random) plasma glucose. This is supported by the fact that our study showed no ethnic difference in the prevalence of self-reported T2DM. However, the observed ethnic discrepancy may also be attributed to various genetic, biological, environmental, and lifestyle-related risk factors. It should also be mentioned that some of the previous

publications are based on data from a larger geographic area than our study.

Adjustment for WHtR in the multinomial logistic regression analysis diminished or eliminated the ethnic difference in the prevalence of pre-diabetes and T2DM, and this impact was most striking for T2DM among women (Table 3). It should be mentioned that as Sami people are generally shorter in stature than their non-Sami counterparts (Table 1), it is more appropriate to use WHtR than WC. According to Table 1, both BMI and WHtR in women, and WHtR in men, were higher among Sami individuals. It is believed that adipose tissue in obese people releases higher amounts of non-esterified fatty acids, glycerol, hormones, pro-inflammatory cytokines and other factors which play an important role in the development of dysglycaemia and eventually T2DM [30]. Higher prevalence of obesity (especially abdominal) and its implication in the higher prevalence of T2DM among indigenous peoples have been reported in a number of publications [31–33].

In the present study, there was observed higher prevalence of pre-diabetes and T2DM among Sami compared to their non-Sami counterparts in almost all geographical regions. However, due to small numbers in each region, only very large differences would have been statistically significant.

Traditionally, most of the population in Northern Norway has relied on primary industries, such as small-scale farming and fishing, and for parts of the Sami population: reindeer herding. A combination of these industries were common. Today, fewer people work in primary industries; instead the number of people employed in service industries has grown. Fewer people have physically active jobs, and even farming and reindeer herding are largely reliant on motor-vehicle transport. This transition from a physically-demanding to a more sedentary lifestyle, which has taken place in both Sami and non-Sami populations, may have increased the risk of developing T2DM [34,35].

Whereas publications on the prevalence of T2DM among other Arctic indigenous populations in the age span 40–79 years are rather sparse, there is compelling evidence that indigenous peoples still suffer from poorer health and social outcomes than do benchmark populations in most countries [36]. There are numerous studies reporting that the prevalence of T2DM and some other lifestyle-related chronic diseases in indigenous peoples are generally either higher than benchmark populations or on the rise. For example, the prevalence of T2DM among Greenland Inuit (age≥18 years) in 2005–2010 was reported to be 9%, of which 79% were previously unknown cases [37]. The overall prevalence of T2DM among Canadian Inuit was in 2006 comparable to the general Canadian population (6.8%), while it was around 2% in 2001 [38]. First Nations Aboriginals in Canada were reported to have a much higher prevalence of DM (15.3%) than the Métis (5.8%) and Inuit (4.3%) [39]. However, in 2007–2008 the prevalence of T2DM in Canadian Inuit aged ≥50 years was 12.2% [40]. In 2010, the prevalence of diagnosed DM among American Indians and Alaska Native individuals was over 14% which is higher than any other racial or ethnic group in the USA [41]. Contrary to indigenous people in most other countries, the Sami people in Norway have living conditions and a socio-economic status that are comparable to those of other Norwegians. This could explain the lack of a huge difference in the prevalence of T2DM between Sami and other Norwegians.

The overall prevalence of T2DM was lower in women compared to men. Although some references do not mention sex as an independent risk factor for T2DM [42], the prevalence of T2DM was reported in several studies to be lower among women [43–45] especially in developed countries [46]. The male excess in the incidence and prevalence of T2DM, which is found in some populations, has been attributed to sex-related differences in insulin sensitivity, consequences of obesity and regional body fat deposition and other contributing factors such as hypertension, smoking and alcohol intake [47,48].

Strengths and limitations

The strengths of this study include its large total sample size (n = 5878) and acceptable participation rate, as well as the use of HbA1c as a diagnostic test, which provided us with valuable estimates of the prevalence of pre-diabetes and T2DM in the inhabitants of the included municipalities. By targeting municipalities with a substantial proportion of Sami inhabitants, we ensured a large proportion of Sami in our sample.

Limited knowledge is at hand regarding non-responders, except that there were more non-responders among men and in the younger age groups. It is also likely that the severely sick had restricted ability to participate in the study and those who were more conscious about their health status had higher tendency to participate (selection bias). Furthermore, it is not certain whether the distribution of ethnic groups in our study reflects the actual ethnic composition of the included municipalities, as there is no ethnic registry in Norway. However, the response was particularly high in some of the municipalities where the Sami are in majority, which may indicate a higher overall response among Sami compared to non-Sami. Only 10 municipalities were included in SAMINOR 2, hence

generalisations to the entire Sami or non-Sami populations in Norway is not advised.

Glucose-based tests (fasting plasma glucose and glucose tolerance test) as well as a physical examination to detect signs and symptoms of DM were not performed due to practical issues.

Our definition of ethnicity is not a mutually exclusive one, as individuals might have expressed a sense of belonging to more than one ethnic group. For example if a participant ticked other ethnicity-related options in addition to Sami in the questionnaire, he/she was categorised as Sami. As a consequence of the assimilation policy, many Sami have abandoned their Sami culture and identity, or choose to conceal their background. Therefore, there are participants of Sami descent, who are categorised as non-Sami in our study. Contrary to some other definitions, our definition gave more emphasis to self-identification than linguistic features. As there have been other definitions of Sami ethnicity in the literature, comparison between our results and results from studies with different definitions should be made with caution. The fact that sensitivity analyses performed with different ethnicity definitions produced overall similar results strengthen our findings.

Conclusion

The overall age-standardised prevalence of pre-diabetes and T2DM were high in the study population. Overall, the prevalence of pre-diabetes and T2DM was higher among Sami compared to their non-Sami counterparts with a higher WHtR in Sami being a plausible explanation. Women in general had lower prevalence of T2DM. Longitudinal studies aiming at assessing the risk of T2DM in Sami and non-Sami, and with a special focus on risk factors such as diet, BMI and WHtR should be undertaken. However, it is at present critical to implement drastic measures in order to reduce the levels of key risk factors and the overall prevalence of T2DM in this population.

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References

[1] Diabetes Fact Sheet No 312 [Internet]. Geneva: World Health Organisation; 2017 Nov [cited 2017 Dec 15]. Available from: http://www.who.int/mediacentre/factsheets/fs312/en/
[2] Sorensen M, Arneberg F, Line TM, et al. Cost of diabetes in Norway 2011. Diabetes Res Clin Pract. 2016;122:124–132.
[3] www.fhi.no [Internet]. Oslo: Norwegian Institute of Public Health; [cited 2017 Dec]. Available from: https://www.fhi.no/nettpub/hin/helse-og-sykdom/diabetes-i-norge—folkehelserappor/#vel-4-prosent-av-befolkningen-har-diagnosen-diabetes
[4] Strøm H, Selmer R, Birkeland KI, et al. No increase in new users of blood glucose-lowering drugs in Norway 2006–2011: a nationwide prescription database study. BMC Public Health. 2014;14:520.
[5] American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2010;33(Suppl. 1):S62–9.
[6] Berg-Nordlie M, Gaski H. Store Norske leksikon; [cited 2017 Nov]. Available from: https://snl.no/S%C3%A1pmi
[7] Broderstad AR, Broderstad EG. Flytting og urbanisering: Sápmi i forandring, i Sametinget 25 år; 1989–2014 [Emmigration and urbanisation: Sápmi in the change. In the Sami Parlament 25 years; 1989–2014]. Karasjok: Sametinget; 2014. p. 153–170. Norwegian.
[8] Pettersen T. Indigenous peoples and demography: the complex relation between identity and statistics. Oxford (UK): Berghahn Books; 2011. Chapter 11, Out of the backwater? Prospects for contemporary sami demography in Norway.
[9] Stordal V. Flerkuturell forståelse. Oslo: Tano Aschehaug; 1997. p. 139–152. Samene: Fra "lavtstaaende race" til "urbefolkning".
[10] Tvermosegaard M, Dahl-Petersen IK, Nielsen NO, et al. Cardiovascular disease susceptibility and resistance in

circumpolar inuit populations. Can J Cardiol. 2015;31(9):1116–1123.
[11] Hutchinson RN, Shin S. Systematic review of health disparities for cardiovascular diseases and associated factors among American Indian and Alaska Native populations. PLoS One. 2014;9(1):e80973.
[12] Naseribafrouei A, Eliassen B-M, Melhus M, et al. Ethnic difference in the prevalence of pre-diabetes and diabetes mellitus in regions with Sami and non-Sami populations in Norway - the SAMINOR1 study. Int J Circumpolar Health. 2016;75:31697.
[13] Broderstad AR, Melhus M. Prevalence of metabolic syndrome and diabetes mellitus in Sami and Norwegian populations. The SAMINOR-a cross-sectional study. BMJ Open. 2016;6(4):e009474.
[14] National Minorities [Internet]. Indigenous peoples and minorities. Oslo: The Norwegian Government; 2017 [cited 2017 Dec]. Available from: https://www.regjeringen.no/en/topics/indigenous-peoples-and-minorities/national-minorities/id1404/
[15] Borch KB, Ekelund U, Brage S, et al. Criterion validity of a 10-category scale for ranking physical activity in Norwegian women. Int J Behav Nutrition Physic Act. 2012;9:2.
[16] Nathan DM, Kuenen J, Borg R, et al. Translating the A1C assay into estimated average glucose values. Diabetes Care. 2008;31(8):1473–1478.
[17] Dahl-Jørgensen K, Brinchmann-Hansen O, Hanssen KF, et al. Effect of near normoglycaemia for two years on progression of early diabetic retinopathy, nephropathy, and neuropathy: the Oslo study. British Med J. 1986;293(6556):1195–1199.
[18] International Expert Committee report on the role of the A1C assay in the diagnosis of diabetes. Diabetes Care. 2009;32(7):1327–1334.
[19] Selvin E, Steffes MW, Gregg E, et al. Performance of A1C for the classification and prediction of diabetes. Diabetes Care. 2011;34(1):84–89.
[20] Barry E, Roberts S, Oke J, et al. Efficacy and effectiveness of screen and treat policies in prevention of type 2 diabetes: systematic review and meta-analysis of screening tests and interventions. BMJ Open. 2017;356:i6538.
[21] World Health Organization. Use of glycated hemoglobin (HbA1c) in the diagnosis of diabetes mellitus. Abbreviated report of a WHO consultation. Geneva: WHO; 2011.
[22] Berg JP. HbA1c as a diagnostic tool in diabetes mellitus. Norsk Epidemiologi. 2013;23(1):5–8.
[23] Hutchinson MS, Joakimsen RM, Njolstad I, et al. Glycated hemoglobin in diagnosis of diabetes mellitus and prediabetes; validation by oral glucose tolerance test. The Tromsø OGTT study. J Endocrinol Invest. 2012;35(9):835–840.
[24] Okura Y, Urban LH, Mahoney DW, et al. Agreement between self-report questionnaires and medical record data was substantial for diabetes, hypertension, myocardial infarction and stroke but not for heart failure. J Clin Epidemiol. 2004;57(10):1096–1103.
[25] Fourrier-Reglat A, Cuong HM, Lassalle R, et al. Concordance between prescriber- and patient-reported previous medical history and NSAID indication in the CADEUS cohort. Pharmacoepidemiol Drug Saf. 2010;19(5):474–481.
[26] Schneider AL, Pankow JS, Heiss G, et al. Validity and reliability of self-reported diabetes in the Atherosclerosis Risk in Communities Study. Am J Epidemiol. 2012;176(8):738–743.
[27] Johannessen LB Samer snakker ikke om helse og sykdom [The Sami people do not speak about the health and disease]. Tidsskrift Den Norske Legeforening. Norwegian; 2015 [cited 2017 Nov 6]. Available from: http://tidsskriftet.no/2015/02/aktuelt-i-foreningen/samer-snakker-ikke-om-helse-og-sykdom
[28] Bhopal R. Concepts of epidemiology. 2nd ed. Oxford: Oxford University Press; 2008. Chapter 4, Error, bias, confounding and risk modification/interaction in epidemiology; p. 103–109.
[29] Nystad TA. A population-based study on cardiovascular risk factors and self-reported type 2 diabetes mellitus in the Sami population [dissertation]. Tromsø (Norway): Centre for Sami Health Research, Institute of Community Medicine. UiT The Arctic University of Norway; 2010.
[30] Pickup JC. Inflammation and activated innate immunity in the pathogenesis of type 2 diabetes. Diabetes Care. 2004;27(3):813–823.
[31] Anderson KG, Spicer P, Peercy MT. Obesity, diabetes, and birth outcomes among American Indians and Alaska Natives. Matern Child Health J. 2016;20(12):2548–2556.
[32] Chateau-Degat ML, Dewailly E, Charbonneau G, et al. Obesity risks: towards an emerging Inuit pattern. Int J Circumpolar Health. 2011;70(2):166–177.
[33] Young TK, Bjerregaard P, Dewailly E, et al. Prevalence of obesity and its metabolic correlates among the circumpolar Inuit in 3 countries. Am J Public Health. 2007;97(4):691–695.
[34] Sjölander P. What is known about the health and living conditions of the indigenous people of northern Scandinavia, the Sami? Glob Health Action. 2011;4(1):8457.
[35] Lund E, Brustad M, Høgmo A. The Sami—living conditions and health. Int J Circumpolar Health. 2008;67(1):6–8.
[36] Anderson I, Robson B, Connolly M, et al. Indigenous and tribal peoples' health (The Lancet-Lowitja Institute Global Collaboration): a population study. Lancet. 2016;388:131–157.
[37] Jørgensen ME, Borch-Johnsen K, Witte DR, et al. Diabetes in Greenland and its relationship with urbanization. Diabet Med. 2012;29(6):755–760.
[38] Aboriginal Peoples Survey, 2006: Inuit Health and Social Conditions [Internet]. Canada: Statistics Canada; 2008 [cited 2017 Dec]. Available from: http://www.statcan.gc.ca/pub/89-637-x/89-637-x2008001-eng.pdf
[39] Diabetes in Canada: Facts and Figures from a Public Health Perspective [Internet]. Canada: Public Health Agency of Canada; 2011 [cited 2017 Jul 17]. Available from: http://www.phac-aspc.gc.ca/cd-mc/publications/diabetesdiabete/facts-figures-faits-chiffres-2011/highlightssaillants-eng.php#chp6
[40] Egeland GM, Cao Z, Young TK. Hypertriglyceridemic-waist phenotype and glucose intolerance among Canadian Inuit: the International Polar Year Inuit Health

Survey For Adults 2007–2008. Can Med A J. 2011;183(9): E553–558.

[41] Centers for Disease Control and Prevention. National Diabetes Fact Sheet: National Estimates and General Information on Diabetes and Prediabetes in the United States, 2011; U.S. Department of Health and Human Services. Vol. 201. Atlanta (GA): Centers for Disease Control and Prevention; 2011.

[42] Powers AC. Diabetes mellitus: diagnosis, classification, and pathophysiology. In: Casper DL, Fauci AS, Hauser SL, Dan L. Longo, J. Larry Jameson, Joseph Loscalzo, editors. Harrisons's principles of internal medicine. 19th ed. New York (NY): McGraw Hill Education; 2015. p. 2400.

[43] IDF Diabetes Atlas, 8th edition [Internet]. Brussels: linternational Diabetes Federation; 2017 [cited 2017 Nov]. Available from: http://www.diabetesatlas.org/resources/2017-atlas.html

[44] Ginde AA, Cagliero E, Nathan DM, et al. Value of risk stratification to increase the predictive validity of HbA1c in screening for undiagnosed diabetes in the US population. J Gen Intern Med. 2008;23(9):1346–1353.

[45] Joseph J, Svartberg J, Njølstad I, et al. Incidence of and risk factors for type-2 diabetes in a general population: the Tromsø study. Scand J Public Health. 2010;38(7):768–775.

[46] Xu Z, Yu D, Yin X, et al. Socioeconomic status is associated with global diabetes prevalence. Oncotarget. 2017;8(27):44434–44439.

[47] Onat A, Hergenc G, Keles I, et al. Sex difference in development of diabetes and cardiovascular disease on the way from obesity and metabolic syndrome. Metabolism. 2005;54(6):800–808.

[48] Meisinger C, Thorand B, Schneider A, et al. Sex differences in risk factors for incident type 2 diabetes mellitus: the MONICA Augsburg cohort study. Arch Intern Med. 2002;162(1):82–89.

Paper III

Title: The 8-year cumulative incidence of diabetes mellitus among Sami and non-Sami inhabitants of Northern Norway - The SAMINOR Study

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Abstract

Objectives: The aim of the study was to estimate and compare the 8-year cumulative incidence of diabetes mellitus (DM) among Sami and non-Sami inhabitants of rural districts in Northern Norway.

Design: Longitudinal study based on linkage of two cross-sectional surveys.

Methods: Ten municipalities in rural Northern Norway were included in the study. DM-free participants aged 30 and 36–71 years were followed from two years after the SAMINOR 1 Survey (2003–2004) to the SAMINOR 2 Clinical Survey (2012–2014). The average follow-up time was 8.1 years. Of 5875 subjects who had participated in SAMINOR 1 and could potentially be followed to SAMINOR 2, 3303 were included in the final analysis. Self-report and/or HbA1c $\geq$ 6.5% were used to identify incident cases of DM.

Results: At baseline, body mass index (BMI) and waist-to-height ratio (WHtR) were higher among Sami than among their non-Sami counterparts. After 8 years of follow-up, 201 (6.1%) incident cases of DM were identified. No statistically significant difference was observed in the sex-specific cumulative incidence of DM between the Sami and non-Sami.

Conclusions: No statistically significant difference in the 8-year cumulative incidence of DM among Sami and non-Sami was observed, although Sami men and women had higher baseline BMI and WHtR.

Key words: cumulative incidence, diabetes mellitus, indigenous, native, Norwegian, SAMINOR, HbA1c, Sami

INTRODUCTION

Type 2 diabetes mellitus (DM) is one of the most prevalent and disabling chronic diseases affecting millions of people worldwide [1]. Indigenous peoples throughout the world are facing an unprecedented epidemic of type 2 DM [2], but publications concerning the incidence of the disease among these groups are rather sparse. This could in part be due to the need for costly and cumbersome cohort studies or the lack of available robust data from national registries.

The Sami are an indigenous people, who have traditionally inhabited northern parts of Norway, Sweden, and Finland, and the Kola Peninsula of Russia. While no statistically significant difference was observed in the prevalence of DM between Sami and non-Sami in the SAMINOR 1 Survey (2003–2004), using self-report and/or non-fasting plasma glucose [3, 4], the prevalence of both pre-diabetes and type 2 DM was higher among Sami people in the SAMINOR 2 Clinical Survey (2012–2014), using self-report and/or HbA1c [5]. There is a lack of longitudinal studies estimating the incidence of DM among Sami and non-Sami inhabitants of rural municipalities in Northern Norway.

The aim of this study is to measure and compare the 8-year cumulative incidence of DM among Sami and non-Sami inhabitants of rural districts in Northern Norway.

METHODS

In 2003–2004, the Centre for Sami Health Research at UiT The Arctic University of Norway, in collaboration with the Norwegian Institute of Public Health, conducted the SAMINOR 1 Survey (hereafter referred to as SAMINOR 1) [6]. This survey included 24 mostly rural municipalities and districts in Northern and Central Norway with a considerable proportion of Sami inhabitants.

In 2012–2014, the Centre for Sami Health Research undertook a two-part second survey, the SAMINOR 2 Questionnaire Survey [7] and the SAMINOR 2 Clinical Survey. The present analyses are based on data from the SAMINOR 2 Clinical Survey (hereafter referred to as SAMINOR 2), which, similarly to SAMINOR 1, consisted of self-administered questionnaires, a clinical examination, and analysis of blood samples. The survey was conducted in 10 municipalities in Finnmark, Troms, and Nordland counties, all previously included in SAMINOR 1: Kautokeino, Karasjok, Tana, Nesseby, Porsanger, Lyngen, Storfjord, Kåfjord, Skånland, and Evenes (Figure 1).

Study sample

The present analyses are based on longitudinal data of those participating in both SAMINOR 1 and SAMINOR 2 from the above-mentioned ten municipalities. In SAMINOR 2, 12,455 subjects, aged 40–79 years, were invited to take part, and 6004 participated (48.2%). We lack information about those invited to SAMINOR 2, who had also participated in SAMINOR 1 but who failed to participate in SAMINOR 2, as a linkage is only allowed for those who participated in both surveys. Therefore, loss to follow-up is described based on SAMINOR 1 participants who would have been invited to SAMINOR 2, given that they had not died or moved from the 10 studied municipalities prior to invitation to SAMINOR 2. There were 11,558 invitees to SAMINOR 1, who, according to their birth year and municipality, would

have been invited to SAMINOR 2, given that they had not moved or died. Of these, 6450 (55.8%) participated in the SAMINOR 1 clinical examinations, of whom 6408 gave their consent to register linkages. The two data files were merged by Statistics Norway, using the unique 11-digit personal identification number assigned to all subjects residing in Norway.

Figure 2 displays the population and exclusions applied. Among the 6408 individuals, the following were excluded: 169 due to missing initial questionnaire; 2 due to missing main questionnaire (containing diabetes information); and 27 due to missing ethnicity information in SAMINOR 1. Based on self-report and random (non-fasting) plasma glucose (RPG) ≥11.1 mmol/L measurement in SAMINOR 1, 260 prevalent cases of DM were excluded. To ensure exclusion of prevalent cases, in total 75 participants were excluded, as, in SAMINOR 2, they reported the date at the time of DM diagnosis as prior to (n=52), at the same time as (n=6) or during the first two years after participating in SAMINOR 1 (n=17, two years wash-out period. Of the remaining 5875 persons, 11 were not included in the final analysis due to missing main questionnaire (n=10) or HbA1c measurement (n=1) in SAMINOR 2. A total of 2561 did not participate in SAMINOR 2 as they had died, moved out of the included municipalities during the follow-up period, or were not willing or able to participate in SAMINOR 2. Hence, 3303 individuals (participation rate: 56.2%) were included in the analysis (Figure 2).

The data collection for SAMINOR 1 took place over two calendar years and over three calendar years for SAMINOR 2, and the municipalities were not visited in the same order in the two surveys. Thus, the time span between the two examinations varied from eight to eleven years, with a mean of 10.1 years. The merged file contains individuals born in the period 1933–1968 and in 1973, who were aged 30 and 36–71 years in SAMINOR 1 and 40–41 and 44–79 years in SAMINOR 2.

Blood sampling

In both SAMINOR 1 and 2, blood samples were taken by venipuncture at normal venous pressure with the participant in a seated position. In SAMINOR 1, blood samples were mailed directly to the laboratory for analysis. Among the included analyses was RPG. The applied methods and procedures in SAMINOR 1 are described in detail elsewhere [6]. In SAMINOR 2, glycated haemoglobin (HbA1c) was measured immediately on site from whole blood, using DCA Vantage™ (Siemens Medical Solutions Diagnostics, Tarrytown, NY, USA). In SAMINOR 1, HbA1c was not measured.

Ethnicity

Ethnic information was collected through self-report in SAMINOR 1. The questions were: "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 do you consider yourself to be?" For all items, the response options were: "Norwegian", "Sami", "Kven", and "Other". The questions were to be answered separately for each relative, and multiple answers were allowed. Sami ethnicity was defined based on two criteria: 1) self-identification as a Sami, and 2) a Sami language connection. Sami self-identification was regarded as fulfilled if the respondent considered him/herself to be Sami or reported having a Sami ethnic background. Sami language connection was defined if at least one grandparent, parent, or the participant him/herself spoke a Sami language at home. Participants who fulfilled both criteria were categorised as Sami. All other participants were categorised as non-Sami.

Diabetes mellitus

In SAMINOR 1, both questionnaire information and RPG levels were used to categorise participants as having DM. The question concerning diabetes was: "Do you have, or have you had, diabetes? (yes/no)". Those who answered "yes", or who had RPG levels of 11.1 mmol/L or higher, were considered prevalent cases of DM.

In SAMINOR 2, the question was: “Have you ever been diagnosed with diabetes (elevated blood sugar levels)? (yes/no)”. Missing self-report of DM was classified as “no”. Participants who answered “yes” or had HbA1c ≥ 6.5% were categorised as incident cases.

Risk factors for type 2 DM

All potential risk factors for DM included in the present study were measured at the start of the study, i.e., in SAMINOR 1.

Height (cm) and weight (kg) were measured using an electronic height and weight scale, with participants wearing light clothing without shoes. Body mass index (BMI) was calculated as weight in kilogrammes, divided by the square of the height in metres (kg/m^2). Waist circumference (WC) was measured in centimetres at the umbilicus, with the participant standing and breathing normally. Waist-to-height ratio (WHtR) was calculated as waist circumference divided by height.

Those who reported in the questionnaire that at least one of their parents, siblings or offspring had DM were regarded as having a positive family history of DM. Marital status (married vs single, widowed/widower, divorced or separated), education (highly educated with more than 12 years of education vs lower education), cigarette smoking (current smoker vs ex-smoker or never-smoker), alcohol drinking (drinking at least once a week vs drinking less often), annual family gross income (lower than 451,000 Norwegian Kroner vs higher income) were also assessed.

Hopkins Symptom Checklist (SCL-10) was used for measuring mental distress [8]. Ten items relevant for mental health are included in the SCL-10: experiencing fear, frightened/ anxiousness, faintness/dizziness, tenseness/upset, insomnia/sleeplessness, easily blaming yourself, being dejected/melancholia, being useless or of little value, experiencing everything as a struggle, being hopeless regarding the future. Each question was answered on a four-

point scale ranging from 1 = “Not affected” to 4 = “Extremely affected”. In total, 418 participants had at least one missing answer to one of the mentioned ten questions. Imputation was performed for those with one (n=130) or two (n=31) missing answers, by assigning the mean values of the respective questions to them, as described by Strand et al. [9]. For records with three or more missing responses, the SCL-10 score was not calculated. The mean of the ten scores was then calculated for each participant, by dividing the sum of the scores by ten. A SCL-10 score over 1.85 is considered indicative of mental distress [8, 9].

Participants scored their leisure-time physical activity during the past year on a four-point scale: 1) “reading, watching TV, or other sedentary activities”; 2) “walking, cycling, or similar forms of exercise at least four hours a week”; 3) “at least four hours a week of recreational sports, heavy gardening, etc.”; and 4) “hard training or sports competitions regularly and several times a week” [10]. Those who reported reading, watching TV, or other sedentary activities were regarded as inactive.

Statistical analysis

Data management and statistical analysis were performed using Stata version 15.0 (Stata Corp., College Station, TX, USA). All tests were two-sided with a 5% significance level and were performed separately for men and women.

Those who were included in the analysis were compared with those we would wish to follow up but were not able to include (due to death, emigration, or lack of participation or insufficient information in SAMINOR 2) with regard to the available baseline characteristics and risk factors for DM (Table 1). Differences in mean age, BMI, WC, and WHtR were tested by two-sample *t*-tests. For the categorical variables, Sami ethnicity, having positive family history of DM, marital status, being highly educated, SCL-10 score > 1.85 (mental distress), smoking, drinking alcohol, having low income, and being inactive in leisure-time, the groups

were compared using Pearson's χ^2 tests. The same variables were compared for Sami vs non-Sami subjects included in the analyses (Table 2).

Those who were categorised as having DM in SAMINOR 2, but not in SAMINOR 1 or the first two years after it, were regarded as incident cases of DM, and, by dividing the number of incident cases by the number of DM-free participants in SAMINOR 1 (at-risk individuals), the approximately 8-year cumulative incidence of DM was estimated. Participants were divided into two age groups: 30 or 36–52-year-old participants, and 53–71-year-old participants in SAMINOR 1. The 8-year cumulative incidence of DM was estimated for and compared between Sami and non-Sami participants from the same sex and age group, using Pearson's χ^2 tests (Table 3).

Multiple logistic regression analysis was used to assess the effect of ethnicity (Sami vs non-Sami), as well as various risk factors, on the development of DM in men and women (Table 4). The first model included ethnicity and age. Then, in addition to age and ethnicity, each of the potential risk factors was included in separate models. Finally, the effect of age, ethnicity, WHtR and education on the cumulative incidence of DM was assessed.

Ethics

The SAMINOR Study was approved by the Norwegian Data Inspectorate and by the Regional Committees for Medical and Health Research Ethics North (REC North). The committee also approved the present study, with approval number 2016/173. All participants gave written informed consent for medical research and to have their data linked to other registers or surveys. The study was also approved by the SAMINOR Project Board.

Table 1. Characteristics of individuals we were able to follow-up, compared to those who were not followed up, among those who participated in SAMINOR 1 (2003–2004) and were eligible[1] for SAMINOR 2 (2012–2014), by sex (N=5875). Numbers are mean (standard deviation) for continuous variables and percent (number of subjects) for categorical variables.

	Included in the follow-up analysis	Not followed up	p-value
Men	N=1447	N=1307	
Age (year)	52.4 (8.7)	51.2 (9.8)	<0.01
Body mass index (kg/m^2)	27.5 (3.5)	27.6 (4.2)	0.42
Waist circumference (cm)	92.3 (9.3)	93.0 (10.9)	0.07
Waist-to-height ratio	0.534 (0.054)	0.537 (0.064)	0.10
Sami ethnicity (%)	40.2 (581)	32.7 (866)	<0.01
Family history of DM[2] (%)	19.4 (280)	18.2 (238)	0.44
Married[3] (%)	64.5 (933)	52.8 (690)	<0.01
Education>12 years (%)	32.8 (458)	30.7 (381)	0.26
SCL-10 score >1.85 (%)	5.3 (72)	9.5 (114)	<0.01
Current smoker[4] (%)	28.8 (416)	39.5 (516)	<0.01
Alcohol[5] (%)	30.7 (444)	31.1 (407)	0.80
Low-income[6] (%)	57.0 (825)	61.5 (804)	0.02
Inactive[7] (%)	18.8 (272)	23.1 (302)	0.01
Women	N=1856	N=1265	
Age (year)	51.6 (9.0)	50.7 (10.1)	<0.01
Body mass index (kg/m^2)	27.4 (4.6)	27.6 (4.9)	0.38
Waist circumference (cm)	84.0 (11.2)	84.2 (11.8)	0.08
Waist-to-height ratio	0.526 (0.074)	0.527 (0.076)	0.40
Sami ethnicity (%)	39.5 (733)	29.4 (372)	<0.01
Family history of DM[2] (%)	23.2 (430)	21.8 (276)	0.38
Married[3] (%)	66.0 (1225)	58.2 (736)	<0.01
Education>12 years (%)	38.0 (674)	36.3 (428)	0.34
SCL-10 score >1.85 (%)	8.4 (141)	11.5 (130)	<0.01
Current smoker[4] (%)	30.6 (568)	40.9 (517)	<0.01
Alcohol[5] (%)	19.7 (365)	20.5 (259)	0.58
Low-income[6] (%)	58.7 (1090)	62.7 (793)	0.03
Inactive[7] (%)	19.1 (355)	22.9 (289)	0.01

1) Living in the 10 SAMINOR 2 municipalities at time of SAMINOR 1 with relevant year of birth
2) Those who had at least one with DM among father, mother, siblings or children
3) Married vs single, widow/widower, divorced, or separated
4) Current smokers vs former smokers or never-smokers
5) Drinking alcohol at least once a week
6) Yearly gross income of the household less than 451,000 Norwegian Kroner
7) Leisure-time activities include reading, watching TV or other sedentary activities

Table 2. Baseline characteristics of diabetes-free participants in SAMINOR 1 (2003–2004) followed-up to SAMINOR 2 (2012–2014), N=3303. Numbers are mean (standard deviation) for continuous variables (age, body mass index, waist circumference, and waist-to-height ratio) and percent (number of subjects) for categorical variables (family history of DM, married, education>12 years, SCL-10 score>1.85, alcohol, low-income, and inactive).

	Sami	Non-Sami	p-value
Men	N=581	N=866	
Age (year)	51.8 (8.8)	52.8 (8.7)	0.04
Body mass index (kg/m^2)	27.8 (3.8)	27.3 (3.3)	0.02
Waist circumference (cm)	91.7 (9.8)	92.8 (9.0)	0.03
Waist-to-height ratio	0.540 (0.060)	0.529 (0.050)	<0.01
Family history of DM[1] (%)	20.5 (119)	18.6 (161)	0.37
Married[2] (%)	59.2 (344)	68.0 (589)	<0.01
Education>12 years (%)	32.6 (184)	32.9 (274)	0.89
SCL-10 score >1.85 (%)	6.3 (34)	4.6 (38)	0.17
Current smoker[3] (%)	29.6 (172)	28.2 (244)	0.55
Alcohol[4] (%)	27.4 (159)	32.9 (285)	0.02
Low-income[5] (%)	60.2 (350)	54.8 (475)	0.04
Inactive[6] (%)	20.3 (118)	17.8 (154)	0.23
Women	N=733	N=1123	
Age (year)	50.7 (8.9)	52.1 (8.9)	<0.01
Body mass index (kg/m^2)	28.0 (4.8)	27.0 (4.5)	<0.01
Waist circumference (cm)	84.5 (11.3)	83.6 (11.2)	0.11
Waist-to-height ratio	0.539 (0.075)	0.516 (0.072)	<0.01
Family history of DM[1] (%)	24.6 (180)	22.3 (250)	0.25
Married[2] (%)	60.3 (442)	69.7 (783)	<0.01
Education>12 years (%)	42.7 (298)	35.0 (376)	<0.01
SCL-10 score >1.85 (%)	9.0 (60)	8.0 (81)	0.47
Current smoker[3] (%)	31.6 (232)	29.9 (336)	0.43
Alcohol[4] (%)	14.3 (105)	23.1 (260)	<0.01
Low-income[5] (%)	61.0 (447)	57.3 (643)	0.11
Inactive[6] (%)	25.0 (183)	15.3 (172)	<0.01

1) Those who had at least one with DM among father, mother, siblings or children
2) Married vs single, widow/widower, divorced, or separated
3) Current smokers vs former smokers or never-smokers
4) Drinking alcohol at least once a week
5) Yearly gross income of the household less than 451,000 Norwegian Kroner
6) Leisure-time activities include reading, watching TV or other sedentary activities

RESULTS

Compared to subjects who took part in SAMINOR 1, but were not followed up, subjects who participated in both surveys were on average older, and more likely to be married and report Sami ethnicity. Furthermore, those included in the follow-up analyses were more physically active and less likely to be current smokers, reporting mental disorders, and having low income (Table 1).

Table 2 shows some baseline characteristics of DM-free individuals in SAMINOR 1 who were followed up until SAMINOR 2. In both sexes, Sami had higher mean WHtR and BMI compared to non-Sami. Mean WC was higher among non-Sami men, while no statistically significant difference was observed in the mean WC between Sami and non-Sami women. Among women, more Sami than non-Sami were considered inactive (Table 2).

A total of 201 incident cases of DM were identified in SAMINOR 2, based on self-report (n=138) or HbA1c ≥ 6.5% (without self-report) (n=63). We noted that all the self-reported cases had HbA1c ≥ 6.5% (results not shown). This number corresponds to a 6.1% (95% confidence interval: 5.3–6.9) 8-year cumulative incidence of DM. No statistically significant difference in the 8-year cumulative incidence of DM was found between Sami and non-Sami of the same sex and age group (Table 3).

Table 3. Estimated 8-year cumulative incidence of diabetes mellitus in % (number of cases) among Sami and non-Sami subjects, according to self-report and/or HbA1c≥6.5%, by sex and age at baseline. SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014), N=3303.

	Age groups	Total % (n)	Non-Sami % (n)	Sami % (n)	p-value*
Men	30, 36–52 years	5.5 (38)	5.3 (21)	5.8 (17)	0.79
	53–71 years	7.8 (59)	7.5 (35)	8.4 (24)	0.65
	Total	6.7 (97)	6.5 (56)	7.1 (41)	0.66
Women	30, 36–52 years	3.8 (37)	3.2 (18)	4.5 (19)	0.32
	53–71 years	7.7 (67)	8.3 (47)	6.5 (20)	0.35
	Total	5.6 (104)	5.8 (65)	5.3 (39)	0.67

* p-values are from Pearson's χ^2- test

We found a positive relationship between age and the odds of DM during follow-up. This relationship was statistically significant in women ($p<0.01$) but not in men ($p=0.29$). The age-adjusted logistic regression analysis showed no statistically significant difference between Sami and non-Sami in the odds for DM in men or women. Further adjustments for other risk factors of DM confirmed that there were no ethnic differences in the odds of contracting DM (Table 4). BMI, WC and WHtR were statistically significant risk factors for DM in both sexes (adjusted for age and ethnicity).

Table 4. Adjusted odds ratios (OR) with 95% confidence interval (95% CI) for incident cases of diabetes mellitus (DM) for Sami compared to non-Sami subjects, and various risk factors for DM, stratified by sex. SAMINOR 1 (2003–2004) and SAMINOR 2 (2012–2014), N=3303.

Models	Adjusted OR (95% CI) for Sami vs non-Sami	p-value	Adjusted OR (95% CI) for respective risk factors (apart from age and ethnicity)	p-value
Men (n=1447)				
Age+ethnicity	1.11 (0.73–1.69)	0.62	-	-
Age+ethnicity+BMI[1]	0.91 (0.59–1.41)	0.68	1.27 (1.20–1.34)	<0.01
Age+ethnicity+WC[2]	1.15 (0.75–1.78)	0.51	1.08 (1.06–1.10)	<0.01
Age+ethnicity+WHtR[3]	0.84 (0.54–1.32)	0.46	1.16 (1.12–1.20)	<0.01
Age+ethnicity+education	1.09 (0.71–1.66)	0.70	0.95 (0.89–1.01)	0.11
Age+ethnicity+inactivity[4]	1.10 (0.72–1.67)	0.65	1.49 (0.92–2.42)	0.10
Age+ethnicity+alcohol[5]	1.09 (0.72–1.65)	0.68	0.67 (0.42–1.11)	0.12
Age+ethnicity+smoking[6]	1.10 (0.72–1.68)	0.64	0.97 (0.61–1.54)	0.90
Age+ethnicity+mental distress[7]	1.15 (0.74–1.78)	0.53	0.40 (0.10–1.64)	0.20
Age+ethnicity+WHtR+education	0.85 (0.54–1.33)	0.47	WHtR: 1.17 (1.12–1.21) education: 0.98 (0.92–1.04)	<0.01 0.55
Women (n=1856)				
Age+ethnicity	0.97 (0.64–1.47)	0.89	-	-
Age+ethnicity+BMI[1]	0.82 (0.53–1.25)	0.35	1.14 (1.10–1.18)	<0.01
Age+ethnicity+WC[2]	0.91 (0.59–1.39)	0.66	1.07 (1.05–1.09)	<0.01
Age+ethnicity+WHtR[3]	0.72 (0.47–1.11)	0.14	1.11 (1.08–1.13)	<0.01
Age+ethnicity+education	1.02 (0.66–1.57)	0.92	0.93 (0.87–0.99)	0.02
Age+ethnicity+inactivity[4]	0.95 (0.63–1.43)	0.80	1.28 (0.79–2.08)	0.31
Age+ethnicity+alcohol[5]	0.95 (0.63–1.43)	0.80	0.72 (0.41–1.24)	0.23
Age+ethnicity+smoking[6]	0.97 (0.64–1.46)	0.90	1.08 (0.70–1.66)	0.73
Age+ethnicity+mental distress[7]	1.10 (0.71–1.71)	0.66	0.94 (0.43–2.07)	0.88
Age+ethnicity+WHtR+education	0.78 (0.50–1.22)	0.28	WHtR: 1.10 (1.08–1.13) education: 0.96 (0.90–1.02)	<0.01 0.20

1) BMI: body mass index (kg/m^2)
2) WC: waist circumference (cm)
3) WHtR: waist-to-height ratio. For the OR to be more understandable, this variable is multiplied by 100
4) Leisure-time physical activity includes reading, watching TV or other sedentary activities
5) Drinking alcohol at least once a week vs drinking alcohol less often
6) Current smokers vs ex-smokers and never-smokers
7) SCL-10 score >1.85

DISCUSSION

The present study is the first to estimate the cumulative incidence of DM among Sami and non-Sami inhabitants of Northern Norway. After eight years of follow-up, 201 (6.1%) incident cases of DM were identified, based on self-report and/or HbA1c $\geq$ 6.5%. The 8-year cumulative incidence of DM was not statistically significantly different between Sami and non-Sami counterparts of the same sex.

Of 5875 SAMINOR 1 participants who were eligible to participate in SAMINOR 2, 3303 were included in the follow-up analysis. To assess the risk of selection bias, we compared some relevant and available risk factors for DM between those who were included in the analysis and those who were not. Although those who were not included in the final analysis were on average younger, the age discrepancy was only around one year, which may not have affected the estimated cumulative incidence of DM. Not being married, being a smoker, having a higher SCL-10 score (mental distress indicator), having lower income and having lower level of leisure-time physical activity, were some attributes of those who were not included in the analysis. In the second survey of the Tromsø Study, it was found that non-participants were over-represented among young and unmarried men [11]. Results from the Tromsø Study indicate lower mortality in subjects who attended several surveys rather than only one [12]. Results from similar studies in Norway indicate that non-participants have higher levels of chronic diseases, higher mortality rates, higher prevalence of disability pension and belong to lower socioeconomic groups [13, 14]. On the other hand, BMI, WC, WHtR (indicators of obesity) and having a positive family history of DM (an indicator of genetic predisposition to DM) were not statistically significantly different between those included in our analysis and those not, making it less likely that the two groups were systematically different with regard to the risk of DM.

If loss to follow-up is due to the outcome (DM), its complications or diseases with shared risk factors (e.g. cardiovascular diseases), the cumulative risk is underestimated (competing risk effect) [15]. Our dataset was not linked to the Cause of Death Registry, so we do not have direct information about the number and causes of death of those who died during the follow-up period. It is unlikely that a participant contracted DM during the follow-up period and died of the disease itself or its late complications. On the other hand, deaths due to competing risks (like cardiovascular diseases) inevitably lead to underestimation of the cumulative incidence of DM. Based on numbers from Statistics Norway, one can expect there to have been around 330 deaths from 2001 to 2011 (10 years) in a group of 5875 persons with similar age span and age distribution to those of our participants (calculations not shown) [16].

According to the Norwegian Institute of Public Health, cancers are the leading cause of death in people with a similar age span to those of our participants, followed by cardiovascular diseases (mutual risk factors for DM) [17]. Competing risks become more important with the increasing age of the population under study (increased risk of multimorbidity). As the mean baseline age of both groups, those that were followed up and those that were not, was around 52 years, and there were relatively few expected deaths (330 deaths totally), it is not thought that competing risks have substantially affected our estimate of the cumulative incidence of DM. Furthermore, studies have shown minimal or no difference between Sami and non-Sami individuals in the distribution of risk factors for cardiovascular diseases and/or the risk of acute myocardial infarction or cerebral stroke [18, 19]. We do not have information on the participants in SAMINOR 1, who, due to emigration, were not included in the final analysis, but they were few, and it is unlikely that they had any impact on the conclusions.

At the end of the follow-up period (SAMINOR 2), self-reported DM and/or HbA1c $\geq$ 6.5% was used to identify incident cases of DM. This HbA1c cut-off is suggested by the American Diabetes Association, as well as the Norwegian Directorate of Health [20, 21], and is being

largely applied in practice. According to the Tromsø OGTT study, an HbA1c cut-off ≥ 6.5% provides sensitivity and specificity of around 35% and 97%, respectively [22]. The low performance of the test leads to substantial misclassification of DM, but it must be assumed to be unrelated to categorisation as a Sami or not.

The HbA1c reflects average plasma glucose concentration during the preceding two to three months [23]. The test has high levels of pre-analytical stability and reproducibility, fewer day-to-day perturbations during periods of stress and illness, and convenience (no need for fasting state or glucose overload) [20]. These attributes might, to some extent, offset the low performance of the test [23].

The questionnaire applied in the present study was not validated. However, the sensitivity and positive predictive value of self-reported DM were reported as 86.7% and 73.4%, respectively, in the CADEUS study in France, using medical records as standard [24]. The validity of self-reported DM in the HUNT 1 Survey was reported to be excellent by comparison with the general practitioners' records, with positive and negative predictive values of 96% and 99.7%, respectively [25].

Categorisation of the participants into Sami and non-Sami was based on the information provided from the SAMINOR 1 questionnaires. It is extremely unlikely that a non-Sami individual would report their ethnicity as Sami, while, due to decades of the governmental assimilation policy (Norwegianisation) and the stigmatisation of Sami people, it is quite likely that some Sami people might report their ethnicity as non-Sami.

These misclassifications must be expected to be unrelated to the DM diagnosis, and have most likely substantially attenuated the measure of association (the possible ethnic difference in DM risk) [26, 27]. The lack of statistically significant difference in the 8-year cumulative incidence of DM between Sami and non-Sami might be explained by the misclassifications or

the relatively small study sample size. Similar standards of living, high awareness about lifestyle diseases like type 2 DM and fair access to healthcare services for both ethnic groups in the study municipalities are other possible explanations.

According to a newly published cohort study, the estimated prevalence of diagnosed type 2 DM for all residents in Norway aged 30 to 89 years increased from 4.9% in year 2009 to 6.1% in 2014 [28]. Nevertheless, the incidence rate of type 2 DM decreased significantly from 609 cases per 100,000 person-years in 2009 to 398 cases per 100,000 in 2014, an annual reduction of 10.1%. Our estimated cumulative incidence of DM (6.1% in 8 years or around 762 cases in 100,000 participants in a year) is comparable to the reported 609 cases per 100,000 person-years in year 2009. It should be kept in mind that our estimate included all types of DM, while the mentioned study reported known cases of type 2 DM. However, due to the age of the new cases, they must be expected to be mainly type 2 DM. In the HUNT Study (from 1995–1997 to 2006–2008), the 11-year cumulative incidence of any diabetes was around 4.5% among adults ($20 \leq age < 70$) using self-report, RPG $\geq$ 11.1 mmol/L, fasting plasma glucose $\geq$ 7 mmol/L, HbAc1 $\geq$ 6.5% or 2-hour 75g OGTT $\geq$ 11.1 mmol/L [25]. The different age span of participants in the HUNT Study is the most likely explanation for the difference between our results and those from the HUNT Study.

While incidence rates of type 2 DM have been reported to be on the rise worldwide in the last 30 years, the disease disproportionally affects indigenous populations [29, 30]. Higher incidence and prevalence of type 2 DM among indigenous peoples, in comparison to the benchmark populations, seems to be a shared phenomenon worldwide [2]. For example, the age-standardised incidence of type 2 DM of 1814 Australian Aboriginal and Torres Strait Islander adults from 1999 to 2007 was reported to be 30.5 in 1000 person-years. The estimated incidence rate is nearly four times higher than that for the non-Indigenous

population and 50% higher than the incidence reported 10 years ago in Australian Aboriginals [31].

Results from the present study, as well as results from our previous studies, which found either no or not a marked ethnic difference in the incidence or prevalence of DM between Sami and non-Sami people in Norway [3-5], imply substantial better conditions for Sami people in Norway, compared with those of other indigenous peoples throughout the world. This is probably due to the Sami enjoying quite similar living and healthcare standards to those of other Norwegian citizens.

Strengths and limitations

Some of the strengths of the present study lie in the application of a comprehensive questionnaire and the use of trained personnel, enabling us to obtain copious amounts of information on several aspects of living and health-related conditions, as well as the use of HbA1c, in addition to self-report, to ascertain DM. The present study is the first longitudinal study to measure the cumulative incidence of DM in Sami-inhabited regions in Norway.

A conventional participation rate, relatively small sample size, limited number of included municipalities, lack of sufficient dietary information, no differentiation between types of DM, lack of linkage to national health registers such as prescription databases, the Cause of Death Register, or discharge register, are some of the limitations of the present study. It is also a limitation that we lack information about which of the SAMINOR 1 participants were actually invited to SAMINOR 2.

We did not have reliable data on the exact time of diagnosis/occurrence of the disease, which made calculation of the incidence rate of DM impossible.

CONCLUSIONS

We observed no ethnic difference in the 8-year cumulative incidence of DM, although mean WHtR and BMI were higher among Sami than non-Sami participants of both sexes. There may be a need for larger studies in the future, to track and elucidate any ethnic difference in the cumulative incidence or incidence rate of DM.

List of abbreviations:

BMI: body mass index

DM: diabetes mellitus

HbA1c: glycated hemoglobin

RPG: random plasma glucose

TV: television

WC: waist circumference

WHtR: waist-to-height ratio

DECLARARTION

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Competing interests*:* The authors declare that they have no competing interests.

Ethics approval and consent to participate: The SAMINOR Study was approved by the Norwegian Data Inspectorate and by the Regional Committees for Medical and Health Research Ethics North (REC North). The committee also approved the present study with the approval number 2016/173. All participants gave written informed consent for medical research and to have their data linked to other registers or surveys.

Consent for publication: Not applicable

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Contributors' contributions: ARB conceptualised and initiated the study. AN as the corresponding author analyzed the data and wrote the article. MM assisted in statistical analyses as well as material and methods' descriptions. BME, MM, JS and ARB contributed to the interpretation of the results and drafting of the manuscript. All authors read and approved the final manuscript.

Availability of data and materials: The data that support the findings of this study were used under licence for the current study and are therefore not publicly available. Data are available from the SAMINOR Study upon reasonable request (www.saminor.no), but restrictions apply to the availability of these data, due to Norwegian privacy regulations.

REFERENCES

1. Vos, T., et al., *Global, regional, and national incidence, prevalence, and years lived with disability for 310 diseases and injuries, 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015.* The Lancet, 2016. **388**(10053): p. 1545-1602.
2. Naqshbandi, M., et al., *Global complication rates of type 2 diabetes in Indigenous peoples: A comprehensive review.* Diabetes Res Clin Pract, 2008. **82**(1): p. 1-17.
3. Naseribafrouei, A., et al., *Ethnic difference in the prevalence of pre-diabetes and diabetes mellitus in regions with Sami and non-Sami populations in Norway - the SAMINOR1 study.* International Journal of Circumpolar Health, 2016. **75**: p. 31697.
4. Broderstad, A.R. and M. Melhus, *Prevalence of metabolic syndrome and diabetes mellitus in Sami and Norwegian populations. The SAMINOR-a cross-sectional study.* BMJ Open, 2016. **6**(4): p. e009474.
5. Naseribafrouei, A., et al., *Prevalence of pre-diabetes and type 2 diabetes mellitus among Sami and non-Sami men and women in Northern Norway - The SAMINOR 2 Clinical Survey.* International Journal of Circumpolar Health, 2018. **77**(1): p. 1463786.
6. Lund, E., et al., *Population based study of health and living conditions in areas with both Sami and Norwegian populations--the SAMINOR study.* International Journal of Circumpolar Health, 2007. **66**(2): p. 113-28.
7. Brustad, M., et al., *A population-based study on health and living conditions in areas with mixed Sami and Norwegian settlements - the SAMINOR 2 questionnaire study.* Int J Circumpolar Health, 2014. **73**: p. 23147.
8. Sørlie, T., K.L. Hansen, and O. Friborg, *Do Norwegian Sami and non-indigenous individuals understand questions about mental health similarly? A SAMINOR 2 study.* International Journal of Circumpolar Health, 2018. **77**(1): p. 1481325.
9. Strand, B.H., et al., *Measuring the mental health status of the Norwegian population: a comparison of the instruments SCL-25, SCL-10, SCL-5 and MHI-5 (SF-36).* Nordic Journal of Psychiatry, 2003. **57**(2): p. 113-8.
10. Grimby, G., et al., *The "Saltin-Grimby Physical Activity Level Scale" and its application to health research.* Scandinavian Journal of Medicine & Science in Sports, 2015. **25 Suppl 4**: p. 119-25.
11. Jacobsen, B.K. and D.S. Thelle, *The Tromso Heart Study: responders and non-responders to a health questionnaire, do they differ?* Scandinavian journal of social medicine, 1988. **16**(2): p. 101-4.
12. Jacobsen, B.K., et al., *Cohort profile: the Tromso Study.* International Journal of Epidemiology, 2012. **41**(4): p. 961-7.
13. Knudsen, A.K., et al., *The health status of nonparticipants in a population-based health study: the Hordaland Health Study.* American Journal of Epidemiology, 2010. **172**(11): p. 1306-14.
14. Langhammer, A., et al., *The HUNT study: participation is associated with survival and depends on socioeconomic status, diseases and symptoms.* BMC Medical Research Methodology, 2012. **12**: p. 143.
15. Szklo, M. and F.J. Nieto, *Measuring Disease Occurrence*, in *Epidemiology Beyond the basics*. 2014, Jones & Bartlett Learning: Burlington.
16. *Statistics Norway, Døde, etter kjønn og aldersgruppe (per 100 000 av middelfolkemengden) 1866-1870 - 2011-2015*. [cited 2018; Available from: https://www.ssb.no/statbank/table/05848/.
17. *Folkehelseinstituttet, Dødsårsaksregistret, statistikkbank*. [cited 2018; Available from: http://statistikkbank.fhi.no/dar/.

18. Siri, S.R.A., et al., *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): p. 638-646.
19. Eliassen, B.M., et al., *Prevalence of self-reported myocardial infarction in Sami and non-Sami populations: the SAMINOR study.* International Journal of Circumpolar Health, 2015. **74**: p. 24424.
20. *American Diabetes Association. Diagnosis and classification of diabetes mellitus.* Diabetes Care, 2010. **33 Suppl 1**: p. S62-9.
21. Helsedirektoratet. *Diagnistiske kriterier for diabetes*. Nasjonal faglig retningslinje for diabetes; Diagnostikk av diabetes, risikovurdering og oppfølging av personer med høy risiko for å utvikle diabetes [cited 2018; Available from: https://helsedirektoratet.no/retningslinjer/diabetes/seksjon?Tittel=diagnostikk-av-diabetes-risikovurdering-2679#diagnostiske-kriterier-for-diabetessterk-anbefaling.
22. Hutchinson, M.S., et al., *Glycated hemoglobin in diagnosis of diabetes mellitus and pre-diabetes; validation by oral glucose tolerance test. The Tromsø OGTT Study.* Journal of Endocrinological Investigation, 2012. **35**(9): p. 835-40.
23. *International Expert Committee report on the role of the A1C assay in the diagnosis of diabetes.* Diabetes Care, 2009. **32**(7): p. 1327-34.
24. Fourrier-Reglat, A., et al., *Concordance between prescriber- and patient-reported previous medical history and NSAID indication in the CADEUS cohort.* Pharmacoepidemiology and Drug Safety, 2010. **19**(5): p. 474-81.
25. Åsvold, B.O., et al., *Prolonged sitting may increase diabetes risk in physically inactive individuals: an 11 year follow-up of the HUNT Study, Norway.* Diabetologia, 2017. **60**(5): p. 830-835.
26. Bhopal, R., *Error, bias, confounding and risk modification/interaction in epidemiology*, in *Concepts of epidemiology*. 2008, Oxford University Press: Oxford.
27. Szklo, M. and N.O. Nielsen, *Understanding lack of validity: Bias*, in *Epidemiology Beyond the Basics*. 2014, Jones & Bartlett Learning: Burlington.
28. Ruiz, P.L.D., et al., *Decreasing incidence of pharmacologically and non-pharmacologically treated type 2 diabetes in Norway: a nationwide study.* Diabetologia, 2018. **61**(11): p. 2310–2318.
29. Harris, S.B., J.W. Tompkins, and B. TeHiwi, *Call to action: A new path for improving diabetes care for Indigenous peoples, a global review.* Diabetes Research and Clinical Practice, 2017. **123**: p. 120-133.
30. Horn, O.K., et al., *Incidence and prevalence of type 2 diabetes in the First Nation community of Kahnawa:ke, Quebec, Canada, 1986-2003.* Canadian Journal of Public Health, 2007. **98**(6): p. 438-43.
31. McDermott, R.A., M. Li, and S.K. Campbell, *Incidence of type 2 diabetes in two Indigenous Australian populations: a 6-year follow-up study.* The Medical Journal of Australia, 2010. **192**(10): p. 562-5.

Figure 1. Map of Northern Norway, Sápmi, and the included municipalities in the SAMINOR 2 Clinical Survey (2012–2014).

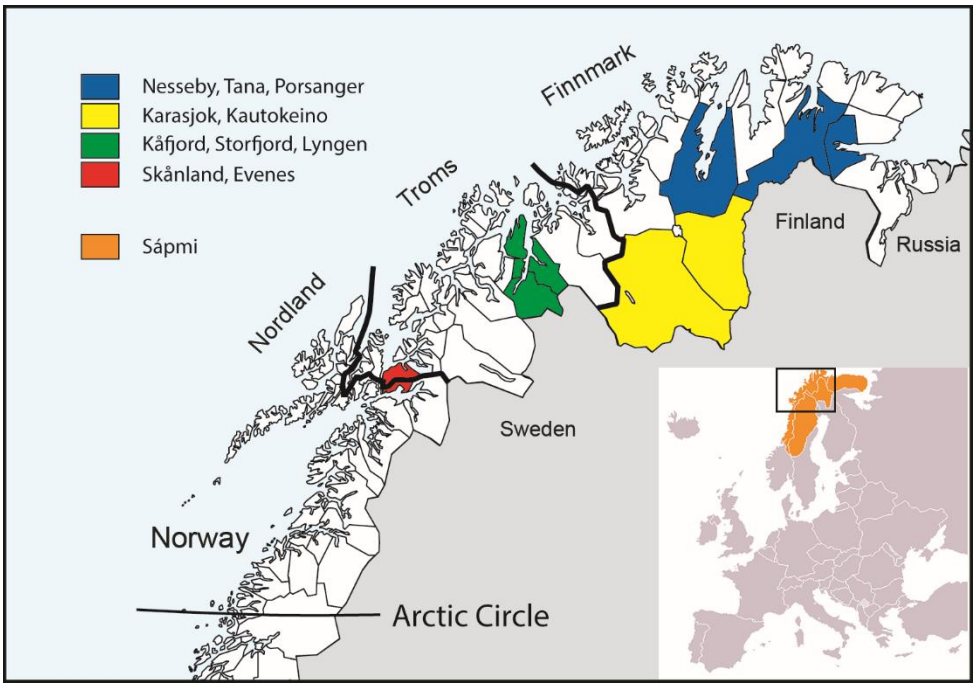
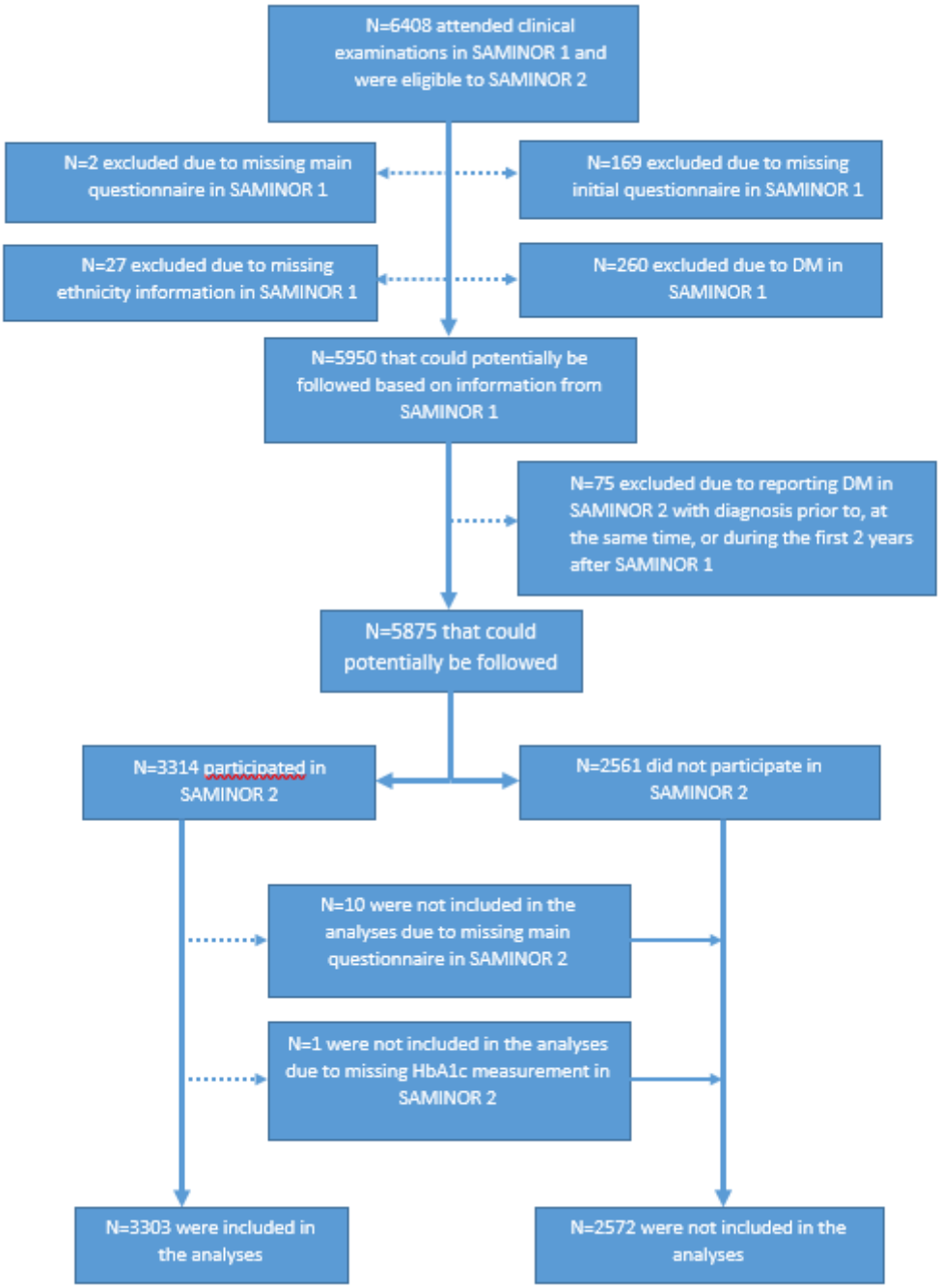


Figure 2. Flow chart demonstrating persons included for final analysis.



Appendix

Appendix 1

The SAMINOR 1 Survey

Original Norwegian versions of:

- **Information brochure**
- **Invitation letter**
- **Informed written consent form**
- **Main+ Initial questionnaire**
- **Additional questionnaire**

https://en.uit.no/forskning/forskningsgrupper/sub?p_document_id=591555&sub_id=591661

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.

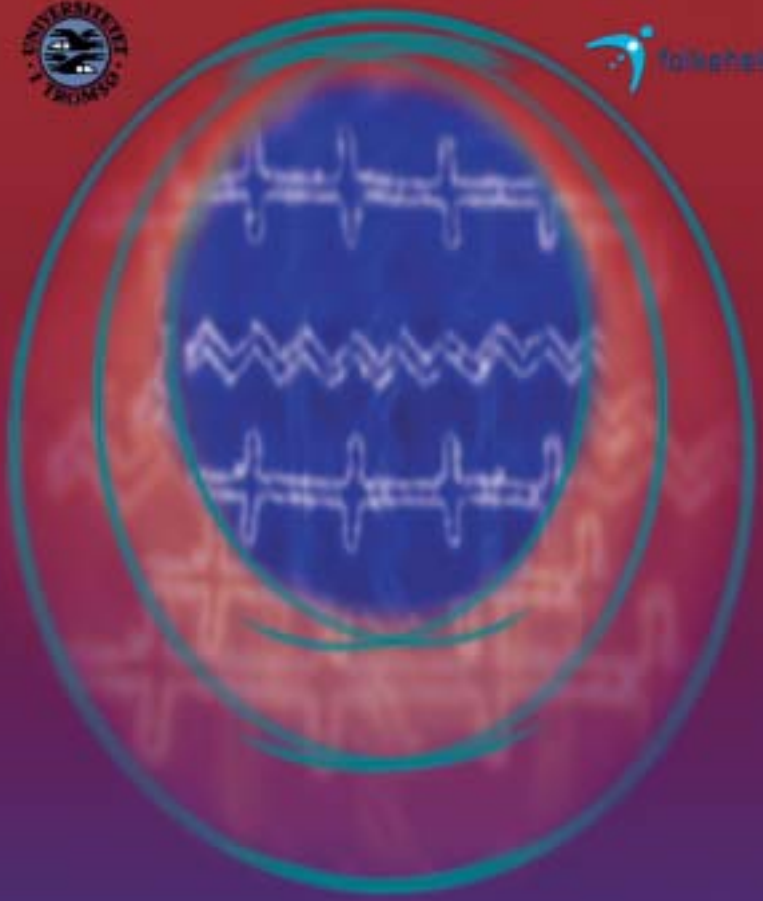
Vaikke dása dál mieđat, de sáhtát maŋŋil molsut oaivila ja bivdit sihkkot iskkadeamis dieđitkeahttá makkárge ákka dasa. Dán dagat čálalaččat Institutt for samfunnsmedisinii; **Institutt for samfunnsmedisin, UiTø, 9037 Tromsø**. Du varraiskkus dalle bálkestuvvo.

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, ožžu 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,*

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

Anne Kirsten Anti
Senter for samisk helseforskning
Karasjok

Eiliv Lund
Institutt for samfunnsmedisin
Tromsø

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

Helse- og levekårs-undersøkelsen

Personlig innbydelse

1. EGEN HELSE

Hvordan er helsen din nå? *(Sett bare ett kryss)*

☐ Dårlig (1) ☐ Ikke helt god (2) ☐ God (3) ☐ Svært god (4)

Har du, eller har du hatt?	JA	NEI	Alder første gang
Astma	☐	☐	
Kronisk bronkitt/emfysem/KOLS	☐	☐	
Diabetes (sukkersyke)	☐	☐	
Fibromyalgi/kronisk smertesyndrom	☐	☐	
Psykiske plager som du har søkt hjelp for	☐	☐	
Hjerteinfarkt (sår på hjertet)	☐	☐	
Angina pectoris (hjertekrampe)	☐	☐	
Hjerneslag/hjerneblødning	☐	☐	
Multippel sklerose (MS)	☐	☐	
Ulcerøs kolitt	☐	☐	

	JA	NEI
Får du smerter eller ubehag i brystet når du: Går i bakker, trapper eller fort på flatmark?	☐	☐
Kan slike smerter opptre selv om du er i ro?	☐	☐

2. MUSKEL OG SKJELETTPLAGER

	JA	NEI
Har du i løpet av *det siste året* vært plaget med smerter og/eller stivhet i muskler og ledd som har vart *i minst 3 måneder* sammenhengende?	☐	☐

Har du noen gang hatt:	JA	NEI	Alder siste gang
Brudd i håndledd/underarm?	☐	☐	
Lårhalsbrudd?	☐	☐	

3. MAGE OG TARM SYMPTOMER

	JA	NEI
Har du hatt sure oppstøt, halsbrann eller brystbrann nesten daglig i minst en uke?	☐	☐
Har du noen gang hatt smerter eller verk i magen som har vart i minst 2 uker?	☐	☐

Hvis JA, hvor i magen sitter smertene? *(Sett ett kryss)*

☐ Øvre del ☐ Nedre del ☐ Hele magen

Er smertene eller «verken» jevnt over tilstede? *(Sett ett kryss)*

I perioder av ukers varighet ☐

I perioder av måneders varighet ☐

Bestandig ☐

	JA	NEI
Er du ofte plaget av oppblåsthet, rumling i magen eller rikelig luftavgang?	☐	☐

3. MAGE OG TARM SYMPTOMER (fortsettelse)

Er avføringen din vanligvis: *(Sett ett eller flere kryss)*

☐ Normal ☐ Løs ☐ Hard og perlete

☐ Vekslende hard og løs ☐ Illeluktende

	JA	NEI
Har du i perioder tre eller flere avføringer daglig?	☐	☐
Har du hatt plager i mage/tarm etter inntak av melk?	☐	☐

Er det andre i familien som har de samme magesymptomene?

☐ Mor ☐ Far ☐ Søsken ☐ Barn ☐ Ingen

4. ANDRE PLAGER

Under finner du en liste over ulike problemer. Har du opplevd noe av dette *den siste uken* (til og med i dag)?

(Sett ett kryss for hver plage)

	Ikke plaget (1)	Litt plaget (2)	Ganske mye (3)	Veldig mye (4)
Plutselig frykt uten grunn	☐	☐	☐	☐
Føler deg redd eller engstelig	☐	☐	☐	☐
Matthet eller svimmelhet	☐	☐	☐	☐
Føler deg anspent eller oppjaget	☐	☐	☐	☐
Lett for å klandre deg selv	☐	☐	☐	☐
Søvnproblemer	☐	☐	☐	☐
Nedtrykt, tungsindig	☐	☐	☐	☐
Følelse av å være unyttig, lite verd	☐	☐	☐	☐
Følelse av at alt er et slit	☐	☐	☐	☐
Følelse av håpløshet mht. framtida	☐	☐	☐	☐
Tenkt på å gjøre slutt på livet ditt	☐	☐	☐	☐

5. SYKDOM I FAMILIEN

	JA	NEI	VET IKKE
Har en eller flere av dine foreldre eller søsken hatt hjerteinfarkt eller angina pectoris?	☐	☐	☐

Kryss av for de slektningene som har eller har hatt noen av sykdommene og angi deres alder for når de fikk sykdommene. *(Hvis flere søsken, før opp den som fikk det tidligst i livet)*

	Mor	Far	Søster	Bror	Barn	Ingen	Alder første gang
Hjerteinfarkt før 60-års alder	☐	☐	☐	☐	☐	☐	
Hjerteinfarkt etter 60 års-alder	☐	☐	☐	☐	■	☐	
Diabetes	☐	☐	☐	☐	☐	☐	
Hjerneslag	☐	☐	☐	☐	☐	☐	
Astma	☐	☐	☐	☐	☐	☐	
Tykktarmskreft	☐	☐	☐	☐	☐	☐	
Brystkreft	☐	☐	☐	☐	☐	☐	
Eggstokkreft	☐	■	☐	■	☐	☐	

Hvor mange søsken har du? ☐☐ Brødre ☐☐ Søstre

6. BRUK AV MEDISINER

Med medisiner mener vi her medisiner kjøpt på apotek. Kosttilskudd og vitaminer regnes ikke med her.

Bruker du?	Nå	Før, men ikke nå	Aldri brukt
Medisin mot høyt blodtrykk	☐	☐	☐
Kolesterolsenkende medisin	☐	☐	☐
Insulin	☐	☐	☐
Tabletter mot sukkersyke	☐	☐	☐

Hvor ofte har du i løpet av *de siste 4 ukene* brukt følgende medisiner? *(Sett ett kryss pr. linje)*

	Ikke brukt siste 4 uker	Sjeldnere enn hver uke	Hver uke, men ikke daglig	Daglig
Smertestillende uten resept	☐	☐	☐	☐
Smertestillende på resept	☐	☐	☐	☐
Sovemedisin	☐	☐	☐	☐
Beroligende medikamenter	☐	☐	☐	☐
Medisiner mot depresjon	☐	☐	☐	☐
Annen medisin på resept	☐	☐	☐	☐
	1	2	3	4

For de medisinene du har krysset av for i de to punktene ovenfor og som du har brukt i løpet av *de siste 4 ukene*:

Angi navnet og hvilken grunn det er til at du tar/har tatt disse (sykdom eller symptom): *(Kryss av for hvor lenge du har brukt medisinen)*

		Hvor lenge?	
Navn på medisinen: *(sett ett navn pr. linje)*	Grunn til bruk av medisinen:	Inntil 1 år	1 år eller mer
		☐	☐
		☐	☐
		☐	☐
		☐	☐
		☐	☐

Dersom det ikke er nok plass her, kan du fortsette på eget ark som du legger ved.

7. MAT OG DRIKKE

Hvor ofte spiser du vanligvis disse matvarene?
(Sett ett kryss pr. linje)

	Sjelden/ aldri	1-3 g. pr.mnd	1-3 g. pr. uke	4-6 g. pr. uke	1-2 g. pr. dag	3 g. el. mer pr. dag
Frukt	☐	☐	☐	☐	☐	☐
Bær	☐	☐	☐	☐	☐	☐
Ost *(alle typer)*	☐	☐	☐	☐	☐	☐
Poteter	☐	☐	☐	☐	☐	☐
Kokte grønnsaker	☐	☐	☐	☐	☐	☐
Rå grønnsaker/salat	☐	☐	☐	☐	☐	☐
	1	2	3	4	5	6

7. MAT OG DRIKKE (fortsettelse)

Hva slags fett bruker du oftest? *(Sett ett kryss pr. linje)*

	Bruker ikke	Meieri-smør	Hard margarin	Myk/lett margarin	Oljer	Annet
På brødet	☐	☐	☐	☐	☐	☐
I matlagingen	☐	☐	☐	☐	☐	☐
	1	2	3	4	5	6

Bruker du følgende kosttilskudd:

	Ja, daglig	Iblant	Nei
Tran, trankapsler?	☐	☐	☐
Fiskeoljekapsler *(omega 3)?*	☐	☐	☐
Vitamin- og/eller mineraltilskudd?	☐	☐	☐

Hvor mye drikker du vanligvis av følgende? *(Sett ett kryss pr. linje)*

	Sjelden/ aldri	1-6 glass pr. uke	1 glass pr. dag	2-3 glass pr. dag	4 glass el. mer pr. dag
Helmelk, kefir, yoghurt	☐	☐	☐	☐	☐
Lettmelk, cultura, lett yoghurt	☐	☐	☐	☐	☐
Skummet melk (sur, søt)	☐	☐	☐	☐	☐
Ekstra lettmelk	☐	☐	☐	☐	☐
Fruktjuice	☐	☐	☐	☐	☐
Vann	☐	☐	☐	☐	☐
Brus/Cola med sukker	☐	☐	☐	☐	☐
Brus/Cola uten sukker	☐	☐	☐	☐	☐
	1	2	3	4	5

Hvor mange kopper kaffe og te drikker du *daglig?*
(Sett 0 for de typene du ikke drikker daglig)

	Antall kopper
Filterkaffe	☐☐
Kokekaffe/trykkanne	☐☐
Annen kaffe	☐☐
Te	☐☐

Omtrent hvor ofte har du i løpet av det siste året drukket alkohol? *(Lettøl og alkoholfritt øl regnes ikke med)*

Har aldri drukket alkohol ☐1	Har ikke drukket siste år ☐2	Noen få ganger siste år ☐3	Omtrent 1 gang i måneden ☐4
2-3 ganger pr. måned ☐5	Ca. 1 gang i uka ☐6	2-3 ganger i uka ☐7	4-7 ganger i uka ☐8

Til dem som har drukket siste år:

Når du har drukket, hvor mange glass eller drinker har du vanligvis drukket? Antall ☐☐

Omtrent hvor mange ganger det siste året har du drukket så mye som minst 5 glass eller drinker i løpet av ett døgn? Antall ganger ☐☐

Når du drikker, drikker du da vanligvis: *(Sett ett eller flere kryss)*

☐ Øl ☐ Vin ☐ Brennevin

8. RØYKING OG BRUK AV SNUS

Hvor lenge er du vanligvis daglig i et røykfylt rom? Antall hele timer ☐☐

Røykte noen av de voksne hjemme da du vokste opp? JA ☐ NEI ☐

Bor du, eller har du bodd, sammen med noen dagligrøykere etter at du fylte 20 år? JA ☐ NEI ☐

Har du røykt/røyker du daglig? Ja, nå ☐ Ja, før ☐ Aldri ☐

Hvis du røyker daglig nå, røyker du:

	JA	NEI
Sigaretter?	☐	☐
Sigarer/sigarillos/pipe?	☐	☐
Rulletobakk/rullings?	☐	☐

Hvis du har røykt daglig *tidligere*, hvor lenge er det siden du sluttet? Antall år ☐☐

Hvis du røyker daglig nå, eller har røykt tidligere:
Hvor mange sigaretter røyker/røykte du vanligvis daglig? Antall sigaretter ☐☐

Hvor gammel var du da du begynte å røyke daglig? Alder i år ☐☐

Hvor mange år til sammen har du røykt daglig? Antall år ☐☐

Har du brukt/bruker du snus daglig? Ja, nå ☐ Ja, før ☐ Aldri ☐

Hvis du bruker/har brukt snus, hvor mange år til sammen har du brukt snus? Antall år ☐☐

9. MOSJON OG FYSISK AKTIVITET

Hvordan har din fysiske aktivitet i <u>fritiden</u> vært <u>det siste året</u>? *(Tenk deg et ukentlig gjennomsnitt for året. Arbeidsvei regnes som fritid. Besvar begge spørsmålene)*

	Timer pr. uke: Ingen	Under 1	1-2	3 og mer
Lett aktivitet *(Ikke svett/andpusten)*	☐	☐	☐	☐
Hard fysisk aktivitet *(Svett/andpusten)*	☐ 1	☐ 2	☐ 3	☐ 4

Angi bevegelse og kroppslig anstrengelse i *<u>din fritid</u>*. Hvis aktiviteten varierer meget f. eks. mellom sommer og vinter, så ta et gjennomsnitt. Spørsmålet gjelder *bare det siste året*.
(Sett kryss i den ruta som passer best)

Leser, ser på fjernsyn eller annen stillesittende beskjeftigelse? ☐ 1

Spaserer, sykler eller beveger deg på annen måte *<u>minst 4 timer i uka</u>*? ☐ 2
(Regn også med gang eller sykling til arbeidsstedet, søndagsturer m.m.)

Driver mosjonsidrett, tyngre hagearbeid e.l.? ☐ 3
(Merk at aktiviteten skal vare minst 4 timer i uka)

Trener hardt eller driver konkurranseidrett regelmessig og *<u>flere ganger i uka?</u>* ☐ 4

10. UTDANNING OG ARBEID

Hvor mange års skolegang har du gjennomført?
(Ta med alle år du har gått på skole eller studert) Antall år ☐☐

Hvordan trives du i din jobb?
1 ☐ Svært godt 2 ☐ Godt 3 ☐ Dårlig 4 ☐ Veldig dårlig

Mener du at du står i fare for å miste ditt nåværende arbeid eller inntekt de nærmeste 2 årene? JA ☐ NEI ☐

Mottar du noen av følgende ytelser?

	JA	NEI
Sykepenger	☐	☐
Attføring	☐	☐
Sosialhjelp/-stønad	☐	☐
Overgangsstønad for enslige forsørgere	☐	☐

11. RESTEN AV SKJEMAET SKAL BARE BESVARES AV KVINNER

Hvor gammel var du da du fikk menstruasjon aller første gang? Alder i år ☐☐

Hvis du ikke lenger får menstruasjon, hvor gammel var du da den sluttet? Alder i år ☐☐

Er du gravid nå? Ja ☐ 1 Nei ☐ 2 Usikker ☐ 3 Over fruktbar alder ☐ 4

Hvor mange barn har du født? Antall barn ☐☐

Hvis du har født barn, fyll ut hvert barns fødselsår, og hvor mange måneder du ammet etter fødselen.
(Hvis du ikke ammet, skriv 0)

Barn:	Fødselsår:	Ammet antall mnd.:
1. barn	☐☐☐☐	☐☐
2. barn	☐☐☐☐	☐☐
3. barn	☐☐☐☐	☐☐
4. barn	☐☐☐☐	☐☐
5. barn	☐☐☐☐	☐☐

(Hvis flere barn, bruk ekstra ark)

Bruker du, eller har du brukt? *(Sett ett kryss for hver linje)*

	Nå	Før, men ikke nå	Aldri
P-pille/minipille/p-sprøyte	☐	☐	☐
Hormonspiral (ikke vanlig spiral)	☐	☐	☐
Østrogen (tabletter eller plaster)	☐	☐	☐
Østrogen (krem eller stikkpiller)	☐	☐	☐

Hvis du bruker/har brukt *reseptpliktig* østrogen:
Hvor lenge har du brukt dette? Antall år ☐☐

Hvis du bruker p-pille, minipille, p-sprøyte, hormonspiral eller østrogen; hvilket merke bruker du?

Spesifiser:

Ikke skriv her ☐☐ ☐☐

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BRUK AV HELSETJENESTER

Hvor mange ganger de *siste 12 måneder* har du selv brukt:
(sett ett kryss for hver linje)

	Ingen	1-3 ganger	4 eller flere
Kommunelege/fastlege	☐	☐	☐
Spesialist	☐	☐	☐
Legevakt	☐	☐	☐
Sykehus innleggelse	☐	☐	☐
Hjemmesykepleie	☐	☐	☐
Kommunal hjemmehjelp	☐	☐	☐
Fysioterapeut	☐	☐	☐
Kiropraktor	☐	☐	☐
Tannlege	☐	☐	☐
Alternativ behandler	☐	☐	☐

Hvor *mange leger* har du selv vært hos de siste 12 måneder?

☐☐ *(angi antall)*

Har du fått tildelt navngitt fastlege? ☐ Ja ☐ Nei

Når du er til undersøkelse, hvilket språk kommuniserer du og legen på? *(sett ett eller flere kryss)*

☐ Norsk ☐ Samisk ☐ Bruker tolk ☐ Annet språk

Tror du det skjer noen gang at du og legen misforstår hverandre p.g.a. språklige problemer?

☐ Aldri ☐ Sjelden ☐ Av og til ☐ Ofte ☐ Usikker

Dersom det er behov for tolk, synes du at legen er flink nok til å be om det?

☐ Ja, alltid ☐ Ja, som regel ☐ Nei, ikke alltid
☐ Nei, aldri ☐ Jeg liker ikke å bruke tolk

Hvor fornøyd eller misfornøyd er du med følgende sider ved den kommunale *legetjenesten* i din bostedskommune?
(sett ett kryss per linje)

	Meget fornøyd	Fornøyd	Misfornøyd	Meget misfornøyd	Vet ikke
Avstand til legen	☐	☐	☐	☐	☐
Legens tilgjengelighet på telefon	☐	☐	☐	☐	☐
Ventetid på legetime	☐	☐	☐	☐	☐
Tid inne hos legen	☐	☐	☐	☐	☐
Mulighetene for å få fortalt om dine plager	☐	☐	☐	☐	☐
Legens forståelse av din kulturelle bakgrunn	☐	☐	☐	☐	☐
Legens informasjon om dine helseplager, undersøkelse og behandlingsopplegg	☐	☐	☐	☐	☐

BRUK AV HELSETJENESTER (fortsettelse)

	Meget fornøyd	Fornøyd	Misfornøyd	Meget misfornøyd	Vet ikke
Legens språkbeherskelse (samisk eller norsk)	☐	☐	☐	☐	☐
Totalt sett, hvor fornøyd eller misfornøyd er du med den kommunale *legetjenesten*?	☐	☐	☐	☐	☐

Hvor lenge er det siden du var hos lege sist? *(angi i hele tall)*

☐☐ *(år)* ☐☐ *(måneder)*

Dersom du noen gang har benyttet alternative behandlere, hvilke har du brukt? *(sett ett eller flere kryss)*

☐ Helbreder *(guvllár, leser, blåser, håndspålegger)*
☐ Healer
☐ Akupunktør
☐ Soneterapeut, homeopat, kinesiolog osv.

Dersom du har benyttet en alternativ behandler, hvor lenge er det siden sist? *(angi i hele tall)*

☐☐ *(år)* ☐☐ *(måneder)*

Tenk deg at du i dag skulle få behov for hjelp/bistand fra den kommunale helse- og sosialtjenesten *(hjemmesykepleie, hjemmehjelp, sosiale tjenester, fysioterapi o.s.v.)*

Vet du hvor du skal henvende deg?

☐ Ja ☐ Nei ☐ Usikker

Er du trygg på at du får hjelp hvis du trenger det?

☐ Ja ☐ Nei ☐ Usikker

Dersom du i dag får hjelp fra den kommunale helse- og sosial tjenesten, er du fornøyd med tilbudet?

☐ Ja ☐ Nei ☐ Usikker

SKADER/ULYKKER

Har du vært utsatt for noen ulykker som medførte behandling hos lege og/eller sykehusinnleggelse?

Lege ☐ Ja ☐ Nei ☐☐ antall ganger

Sykehus innleggelse ☐ Ja ☐ Nei ☐☐ antall ganger

SKADER/ULYKKER (fortsettelse)

Hvis ja, hva slags ulykke(r) er du blitt behandlet for?
(sett ett eller flere kryss pr. linje)

	Arbeid	Hjem	Fritid	Ingen
Bil	☐	☐	☐	☐
Motorsykkel	☐	☐	☐	☐
Snøscooter	☐	☐	☐	☐
Firehjulssykkel	☐	☐	☐	☐
Traktor	☐	☐	☐	☐
Fallulykke	☐	☐	☐	☐
Kuttskade	☐	☐	☐	☐
Annet	☐	☐	☐	☐

Har ulykken(e) ført til nedsatt arbeidsevne?

☐ Helt ☐ Delvis ☐ Ikke i det hele tatt

FAMILIE OG SPRÅKBAKGRUNN

I Nord-Norge bor det folk med ulik etnisk bakgrunn. Det vil si at de snakker ulike språk og har forskjellige kulturer. Eksempler på etnisk bakgrunn, eller etnisk gruppe er norsk, samisk og kvensk.

Hvilket hjemmespråk har/hadde du, dine foreldre og besteforeldre? *(sett ett eller flere kryss)*

	Norsk	Samisk	Kvensk	Annet, beskriv
Morfar:	☐	☐	☐	☐ .....
Mormor:	☐	☐	☐	☐ .....
Farfar:	☐	☐	☐	☐ .....
Farmor:	☐	☐	☐	☐ .....
Far:	☐	☐	☐	☐ .....
Mor:	☐	☐	☐	☐ .....
Jeg selv:	☐	☐	☐	☐ .....

Hva er din, din fars og din mors etniske bakgrunn?
(sett ett eller flere kryss)

	Norsk	Samisk	Kvensk	Annet, beskriv
Min etniske bakgrunn er:	☐	☐	☐	☐ .....
Fars etniske bakgrunn er:	☐	☐	☐	☐ .....
Mors etniske bakgrunn er:	☐	☐	☐	☐ .....

Hva regner du deg selv som? *(sett ett eller flere kryss)*

Norsk	Samisk	Kvensk	Annet, beskriv
☐	☐	☐	☐ .....

ARBEIDSLIV/ØKONOMI

Hvilken type arbeid/livsopphold har du? *(sett ett eller flere kryss)*

☐ Fastlønnet, heltid ☐ Fastlønnet, deltid
☐ Sesongarbeid ☐ Selvstendig næringsdrivende
☐ Arbeidsledig ☐ Hjemmeværende
☐ Alderstrygd ☐ Uføretrygd
☐ Annet *(beskriv)* .....

ARBEIDSLIV/ØKONOMI (fortsettelse)

Kunne du tenke deg å flytte fra din bostedskommune dersom du fikk tilbud om arbeid et annet sted?

☐ Ja ☐ Nei ☐ Deler av året ☐ Usikker

Dersom du er *arbeidsledig*, angi hvor lenge du har vært arbeidssøker: *(angi i hele tall)*

☐☐ *(år)* ☐☐ *(måneder)*

Dersom du er *selvstendig næringsdrivende*, hvilken type næring jobber du i? *(sett ett eller flere kryss)*

☐ Reindrift ☐ Fiske ☐ Jordbruk ☐ Skogbruk
☐ Forretningsvirksomhet ☐ Annet *(spesifiser)* .....

Hvor mange personer bor det i din husstand?

☐☐ *(antall personer)*

Hvor stor er familiens/husstandens bruttoinntekt per år?

☐ Under kr. 150 000 ☐ Kr. 150 000–300 000
☐ Kr. 301 000–450 000 ☐ Kr. 451 000–600 000
☐ Kr. 601 000–750 000 ☐ Over kr. 750 000

Hvor ofte spiller du på ulike pengespill slik som lotto, tipping, spilleautomater og lignende?

☐ Aldri/sjelden ☐ 1-3 ganger i mnd.
☐ 1 gang i uka ☐ 2-6 ganger i uka ☐ Hver dag

Hvor mye spiller du for ukentlig i gjennomsnitt?

☐ Under kr. 100 i uka ☐ Kr. 100-500 i uka
☐ Kr. 501–1000 i uka ☐ Over kr. 1000 i uka

MOBBING

Med mobbing mener vi når en eller flere personer *gjentatte ganger* sier eller gjør vonde ting mot deg, og du har vanskeligheter med å forsvare deg.

Har du vært utsatt for mobbing?

☐ Ja, de siste 12 mnd. ☐ Ja, før ☐ Nei

Dersom du har vært utsatt for mobbing, hvilken type mobbing er du blitt utsatt for? *(sett ett eller flere kryss)*

☐ Baksnakking ☐ Ignorering
☐ Diskriminerende bemerkninger ☐ Annet

Kan du angi hvor dette foregår/foregikk?
(sett ett eller flere kryss)

☐ På skolen ☐ På skoleinternat ☐ I yrkeslivet
☐ I lokalsamfunnet ☐ Annet

P.I.J.

Dato for utfylling:

Dag ☐☐ Måned ☐☐ År ☐☐☐☐

TILLEGGSSPØRSMÅL TIL HELSE- OG LEVEKÅRSUNDERSØKELSEN

Takk for fremmøte til helseundersøkelsen. På denne måten har du bidratt til å skaffe ny viten om helse og levekår i områder med samisk og norsk bosetting. Hovedformålet med undersøkelsen har vært å skaffe ny viten om hjerte- kar sykdommer for å kunne forebygge dem. I tillegg skal undersøkelsen gi oss kunnskap om andre sykdommer og plager slik at vi kan lage en oversikt over folks helse i fylket. Vi ber deg derfor svare på noen spørsmål om forhold som kan ha betydning for disse og andre sykdommer.

Det utfylte skjemaet sendes i vedlagte svarkonvolutt. Portoen er betalt. På forhånd takk for hjelpen!

Med vennlig hilsen:
Senter for samisk helseforskning og
Nasjonalt folkehelseinstitutt

1. SYMPTOMER

	JA	NEI
Hoster du omtrent daglig i perioder av året?	☐	☐
Har du hatt slik hoste så lenge som i en 3 måneders periode i begge de to siste år?	☐	☐
Hender det at du er plaget av søvnløshet?	☐	☐

Hvis ja, når er du mest plaget av søvnløshet?
(Sett ett eller flere kryss)
☐ Hele året ☐ Vår ☐ Sommer ☐ Høst ☐ Vinter

	JA	NEI
Har du det siste året vært plaget av søvnløshet slik at det har gått ut over arbeidsevnen?	☐	☐

Er du stort sett fornøyd med tilværelsen?
☐ Meget fornøyd
☐ Ganske fornøyd
☐ Litt misfornøyd
☐ Meget misfornøyd

	JA	NEI
Hender det at du i lengre perioder – i minst 14 dager- er trist og nedfor?	☐	☐

Har du i de siste 14 dager følt deg ute av stand til å takle dine vanskeligheter?
☐ Nei ☐ Av og til ☐ Ofte ☐ Nesten hele tiden

Hender det at du føler deg ensom?
☐ Nei ☐ Av og til ☐ Ofte

2. KOSTHOLD NÅ

Vi vil gjerne spørre deg om hvor ofte du pleier å spise enkelte matvarer. Tenk på gjennomsnittet det siste året. Sett *ett kryss* pr. linje for antall ganger. Hvis du ikke husker nøyaktig, fyll ut så godt du kan.

Hvor mange ganger i uken pleier du å spise middag? Antall ganger ☐☐

Hvor ofte pleier middagen å inneholde:

	Aldri/ sjelden	1 pr. mnd.	2-3 pr. mnd.	1 pr. uke	2 pr. uke	3 pr. uke	4 pr. uke	5+ pr. uke
Fisk	☐	☐	☐	☐	☐	☐	☐	☐
Kjøtt (helt, oppmalt)	☐	☐	☐	☐	☐	☐	☐	☐
Verken fisk el. kjøtt	☐	☐	☐	☐	☐	☐	☐	☐

Hvor ofte spiser du kokt torsk og sei til middag?

	Aldri	1-11 pr. år	1 pr. mnd.	2-3 pr. mnd.	1 pr. uke	2 pr. uke	3+ pr. uke
Torsk *(f.eks. fersk, lettsaltet, røkt, bokna)*	☐	☐	☐	☐	☐	☐	☐
Sei *(f.eks. fersk, bokna)*	☐	☐	☐	☐	☐	☐	☐

Hvor ofte spiser du annen kokt fisk til middag?

	Aldri	1-5 pr. år	6-11 pr. år	1 pr. mnd.	2-3 pr. mnd.	1 pr. uke	2+ pr. uke
Fete fiskeslag *(f.eks. kokt laks, kveite, uer, røye, ørret, sik)*	☐	☐	☐	☐	☐	☐	☐
Magre fiskeslag *(f.eks. kokt hyse/ kolje, abbor, gjedde, harr)*	☐	☐	☐	☐	☐	☐	☐

Hvor ofte spiser du stekt fisk til middag?

	Aldri	1-5 pr. år	6-11 pr. år	1 pr. mnd.	2-3 pr. mnd.	1 pr. uke	2+ pr. uke
Fete fiskeslag *(f.eks. stekt laks, sild, røye, ørret, sik)*	☐	☐	☐	☐	☐	☐	☐
Magre fiskeslag *(f.eks. stekt sei, torsk, abbor, gjedde, harr)*	☐	☐	☐	☐	☐	☐	☐

Hvor ofte spiser du fiskemat til middag?

	Aldri	1-5 pr. år	6-11 pr. år	1 pr. mnd.	2-3 pr. mnd.	1 pr. uke	2+ pr. uke
Fiskekaker/boller/ pudding	☐	☐	☐	☐	☐	☐	☐
Fiskegrateng, plukkfisk	☐	☐	☐	☐	☐	☐	☐
Fiskepinner, panert fisk	☐	☐	☐	☐	☐	☐	☐

Hvor ofte spiser du fiskepålegg?

	Aldri	1-11 pr. år	1 pr. mnd.	2-3 pr. mnd.	1-2 pr. uke	3-4 pr. uke	5+ pr. uke
Speket/saltet fisk	☐	☐	☐	☐	☐	☐	☐
Røkt fisk	☐	☐	☐	☐	☐	☐	☐
Makrell i tomat	☐	☐	☐	☐	☐	☐	☐
Nedlagt sild	☐	☐	☐	☐	☐	☐	☐
Kaviar	☐	☐	☐	☐	☐	☐	☐
Annet fiskepålegg	☐	☐	☐	☐	☐	☐	☐

Hvor mange *ganger pr. år* spiser du fiskeinnmat?

	0	1-3	4-6	7-9	10+
Fiskelever	☐	☐	☐	☐	☐
Rogn	☐	☐	☐	☐	☐

Hvor ofte spiser du følgende retter?

	Aldri	1-5 pr. år	6-11 pr. år	1 pr. mnd.	2-3 pr. mnd.	1 pr. uke	2+ pr. uke
Pizza	☐	☐	☐	☐	☐	☐	☐
Spagetti, pastaretter	☐	☐	☐	☐	☐	☐	☐
Hamburger i brød	☐	☐	☐	☐	☐	☐	☐
Kjøttkaker/ karbonader	☐	☐	☐	☐	☐	☐	☐
Pølser	☐	☐	☐	☐	☐	☐	☐
Gryterett	☐	☐	☐	☐	☐	☐	☐

Hvor ofte spiser du rent kjøtt til middag *(f.eks. koteletter, steik, grytekjøtt, biff, filet)*?

	Aldri	1-5 pr. år	6-11 pr. år	1 pr. mnd.	2-3 pr. mnd.	1 pr. uke	2+ pr. uke
Kylling	☐	☐	☐	☐	☐	☐	☐
Svin	☐	☐	☐	☐	☐	☐	☐
Okse/storfe	☐	☐	☐	☐	☐	☐	☐
Sau/lam	☐	☐	☐	☐	☐	☐	☐
Elg	☐	☐	☐	☐	☐	☐	☐
Hval	☐	☐	☐	☐	☐	☐	☐

Hvor mange egg fra sjøfugl spiser du pr. år?

	0	1-3	4-6	7-9	10+
Antall egg	☐	☐	☐	☐	☐

Hvor ofte spiser du kjøtt av rein?

	Aldri	1-11 pr. år	1 pr. mnd.	2-3 pr. mnd.	1 pr. uke	2 pr. uke	3+ pr. uke
Kokt reinkjøtt *(buljong)*	☐	☐	☐	☐	☐	☐	☐
Stekt reinkjøtt *(helt, skavet, oppmalt)*	☐	☐	☐	☐	☐	☐	☐
Røkt reinkjøtt	☐	☐	☐	☐	☐	☐	☐
Tørket reinkjøtt	☐	☐	☐	☐	☐	☐	☐

Hvor ofte spiser du andre matvarer av rein?

	Aldri	1-5 pr. år	6-11 pr. år	1 pr. mnd.	2-3 pr. mnd.	1+ pr. uke
Blodmat av rein	☐	☐	☐	☐	☐	☐
Margbein	☐	☐	☐	☐	☐	☐
Reintunge	☐	☐	☐	☐	☐	☐
Reinlever	☐	☐	☐	☐	☐	☐

Hvor ofte spiser du bær?

Én gang tilsvarer 1 brødskive med syltetøy, tyttebær til 1 porsjon middag, 1 porsjon dessert, 1 glass saft, eller en tur hvor du spiste friske bær.

	Aldri	1-5 pr. år	6-11 pr. år	1 pr. mnd.	2-3 pr. mnd	1-2 pr. uke	3+ pr. uke
Molter:							
Friske, frosne, rørte	☐	☐	☐	☐	☐	☐	☐
Kokt/kjøpt syltetøy	☐	☐	☐	☐	☐	☐	☐
Tyttebær:							
Friske, frosne, rørte	☐	☐	☐	☐	☐	☐	☐
Kokt/kjøpt syltetøy	☐	☐	☐	☐	☐	☐	☐
Blåbær:							
Friske, frosne, rørte	☐	☐	☐	☐	☐	☐	☐
Kokt/kjøpt syltetøy	☐	☐	☐	☐	☐	☐	☐
Saft	☐	☐	☐	☐	☐	☐	☐
Krøkebær:							
Friske, frosne	☐	☐	☐	☐	☐	☐	☐
Saft	☐	☐	☐	☐	☐	☐	☐

Hvordan pleier du/ditt hushold å skaffe følgende råvarer til eget bruk? *(Sett ett eller flere kryss)*

	Spiser aldri/ sjelden	Helt selv-forsynt	Delvis selv-forsynt	Kjøper i butikk	Kjøper privat	Bytter eller får
Kjøtt:						
Rein	☐	☐	☐	☐	☐	☐
Sau	☐	☐	☐	☐	☐	☐
Elg	☐	☐	☐	☐	☐	☐
Fisk:						
Ferskvann	☐	☐	☐	☐	☐	☐
Saltvann	☐	☐	☐	☐	☐	☐
Bær:						
Molter	☐	☐	☐	☐	☐	☐
Tyttebær	☐	☐	☐	☐	☐	☐

Hvor ofte pleier du å jakte, fiske og plukke bær?

	Aldri	Sjelden	Av og til	Mye av fritiden
Jakte rype/småvilt	☐	☐	☐	☐
Jakte storvilt	☐	☐	☐	☐
Fiske	☐	☐	☐	☐
Plukke bær	☐	☐	☐	☐

Hvor ofte har du spist et hovedmåltid fra ditt husholds jakt/fiske siste år?

	Aldri	1-5 pr. år	6-11 pr. år	1 pr. mnd.	2-3 pr. mnd.	1 pr. uke	2+ pr. uke
Hovedmåltid jakt	☐	☐	☐	☐	☐	☐	☐
Hovedmåltid fiske	☐	☐	☐	☐	☐	☐	☐

3. KOSTHOLD I OPPVEKSTEN

Tenk på maten du fikk hjemme før du flyttet for deg selv. Hvis du bodde mesteparten av året på skoleinternat, tenk på maten du fikk der.

Bodde du på internat (statsinternat eller privat) da du gikk på barne- og ungdomsskolen?

- ☐ Ja, ungdomsskolen
- ☐ Ja, barneskolen
- ☐ Ja, både barne- og ungdomsskolen
- ☐ Nei, ingen av delene

Hvis ja, hvor mange klassetrinn? ☐☐

Hvor lenge var du på internat i snitt for hvert klassetrinn?
☐ 1-3 mnd. ☐ 4-6 mnd. ☐ 7-9 mnd.

Hvor ofte spiste du fisk og reinkjøtt i oppveksten?

	Aldri	1-11 pr. år	1 pr. mnd.	2-3 pr. mnd.	1-2 pr. uke	3-4 pr. uke	5+ pr. uke
Kokt/stekt fisk	☐	☐	☐	☐	☐	☐	☐
Reinkjøtt	☐	☐	☐	☐	☐	☐	☐

Hvor ofte spiste du andre matvarer i oppveksten?

	Aldri	1-11 pr. år	1 pr. mnd.	2-3 pr. mnd.	1 pr. uke	2 pr. uke	3+ pr. uke
Blodmat	☐	☐	☐	☐	☐	☐	☐
Sauekjøtt	☐	☐	☐	☐	☐	☐	☐
Kjøttkaker, pølser	☐	☐	☐	☐	☐	☐	☐
Fiskemat	☐	☐	☐	☐	☐	☐	☐
Fiskelever og rogn	☐	☐	☐	☐	☐	☐	☐
Grøt, pannekaker	☐	☐	☐	☐	☐	☐	☐

	JA	NEI
Fikk du medisinsk tran i oppveksten?	☐	☐
Fikk du servert tran til for eksempel fisk (i stedet for annet fett)?	☐	☐

Hvor ofte spiste du ville bær og planter i oppveksten?

	Aldri	1-5 pr. år	6-11 pr. år	1 pr. mnd.	2-3 pr. mnd	1-2 pr. uke	3+ pr. uke
Ville bær	☐	☐	☐	☐	☐	☐	☐
Syregress	☐	☐	☐	☐	☐	☐	☐
Kvann	☐	☐	☐	☐	☐	☐	☐

Er maten du spiser nå, forskjellig fra det du fikk i oppveksten?

- ☐ Nei
- ☐ Litt forskjellig
- ☐ Ganske forskjellig
- ☐ Veldig forskjellig

4. NATTSPISING

	JA	NEI
Våkner du ofte opp for å spise etter at du har lagt deg om kvelden?	☐	☐

Hvis «ja», besvar de neste 4 spørsmålene:

Når har du oftest plagene? *(Sett ett eller flere kryss)*
☐ Hele året ☐ Vår ☐ Sommer ☐ Høst ☐ Vinter

Hva spiser du om natten? *(Sett ett eller flere kryss)*
☐ Kjøtt ☐ Brødmat ☐ Godteri ☐ Annet

	JA	NEI
Spiser du mer enn halvparten av døgnets matmengde etter kl. 20 om kvelden?	☐	☐

Er andre i familien plaget med nattspising?

JA	NEI	VET IKKE
☐	☐	☐

	JA	NEI
Har du skiftarbeid, nattarbeid eller går vakter?	☐	☐

5. OPPVEKST, FAMILIE OG VENNER

I hvilken kommune har du bodd lengre enn *ett* år?
Kommune:

1. Fødested: ..... fra 0 år til ☐☐ år
2. ..... fra ☐☐ år til ☐☐ år
3. ..... fra ☐☐ år til ☐☐ år
4. ..... fra ☐☐ år til ☐☐ år
5. ..... fra ☐☐ år til ☐☐ år

(Hvis du har bodd i flere kommuner, bruk eget ark.)

	JA	NEI
Bor du sammen med ektefelle/samboer?	☐	☐

Har du delt eller daglig omsorg for	JA	NEI
Barn?	☐	☐
Foreldre/andre?	☐	☐

Hvor mange gode venner har du?
(De som du kan snakke fortrolig med og som kan gi deg hjelp dersom du trenger det. Tell ikke med de du bor sammen med.) Antall venner ☐☐

Er du tilknyttet noen av de følgende menigheter/trossamfunn: *(Sett ett eller flere kryss)*

- ☐ Medlem i statskirka
- ☐ Den Læstadianske menighet
- ☐ Annen menighet
- ☐ Ikke medlem av noen menighet

Føler du at du kan påvirke det som skjer i lokalsamfunnet der du bor? *(Sett bare ett kryss)*

- ☐ Ja, i stor grad
- ☐ Ja, en del
- ☐ Ja, i liten grad
- ☐ Nei
- ☐ Har ikke forsøkt

6. VERDITILKNYTNING

TIL ALLE:

Er det viktig for deg å ha kontakt med naturen?

Meget viktig ☐ Viktig ☐ Lite viktig ☐ Helt uviktig ☐

Er utnytting av naturen gjennom fiske, jakt og bærplukking viktig for deg?

Meget viktig ☐ Viktig ☐ Lite viktig ☐ Helt uviktig ☐

Er bevaring av slekts- og familietradisjoner viktig for deg?

Meget viktig ☐ Viktig ☐ Lite viktig ☐ Helt uviktig ☐

Har du opplevd at du er blitt mobbet eller diskriminert på grunn av din etniske (*samisk, kvensk, russisk, tamilsk, norsk, etc.)* **bakgrunn?**

Svært mange ganger ☐ Noen ganger ☐ En sjelden gang ☐ Aldri ☐

Tror du at diskriminering av etniske minoriteter kan ha negative helsemessige konsekvenser?

I stor grad ☐ I noen grad ☐ I liten grad ☐ Absolutt ikke ☐

Føler du deg presset ut av næringen din?

I stor grad ☐ I noen grad ☐ I liten grad ☐ Absolutt ikke ☐

7. TIL DEM MED SAMISK BAKGRUNN:

Er samiske klestradisjoner viktige for deg?

Meget viktig ☐ Viktig ☐ Lite viktig ☐ Helt uviktig ☐

Hvilken betydning har duodji for deg?

Meget stor betydning ☐ Stor betydning ☐ Liten betydning ☐ Ingen betydning ☐

Hva betyr bevaring og utvikling av det samiske språket for deg?

Meget stor betydning ☐ Stor betydning ☐ Liten betydning ☐ Ingen betydning ☐

Er det viktig for deg å bo i et lokalsamfunn der du daglig kan møte andre samer?

Meget viktig ☐ Viktig ☐ Lite viktig ☐ Helt uviktig ☐

Synes du at bevaring av typiske samiske næringer er viktig?

Meget viktig ☐ Viktig ☐ Lite viktig ☐ Helt uviktig ☐

Er utviklingen av det moderne samiske skoleverket viktig for deg?

Meget viktig ☐ Viktig ☐ Lite viktig ☐ Helt uviktig ☐

Er det viktig for deg at samiske lokalsamfunn bør få et større innslag av moderne arbeidsplasser?

Meget viktig ☐ Viktig ☐ Lite viktig ☐ Helt uviktig ☐

Hva betyr samiske media (radio, TV, aviser, bøker) for deg?

Meget stor betydning ☐ Stor betydning ☐ Liten betydning ☐ Ingen betydning ☐

Hva betyr moderne samisk kunst (billedkunst, musikk, film og teater) for deg?

Meget stor betydning ☐ Stor betydning ☐ Liten betydning ☐ Ingen betydning ☐

Hvordan ser du på at samisk samfunn og kultur med årene har fått en sterkere internasjonal kontakt?

Meget viktig ☐ Viktig ☐ Lite viktig ☐ Helt uviktig ☐

Hva betyr Sametinget for deg?

Meget stor betydning ☐ Stor betydning ☐ Liten betydning ☐ Ingen betydning ☐

Opplever du forurensning av eller inngrep i naturen som en trussel mot din samiske tilværelse?

I stor grad ☐ I noen grad ☐ I liten grad ☐ Absolutt ikke ☐

Føler du at den moderne utviklingen fortrenger den samiske kulturen?

I stor grad ☐ I noen grad ☐ I liten grad ☐ Absolutt ikke ☐

TAKK FOR HJELPEN!
HUSK Å POSTLEGGE SKJEMAET I DAG!

Appendix 2

The SAMINOR 2 Clinical Survey

Original Norwegian versions of:

- **Pamphlet**
- **Information brochure**
- **Invitation letter** **(Illustrated by information letter to Karasjok municipality)**
- **Informed written consent form**
- **Questionnaire (for 40–69 years participants)**
- **Questionnaire (for 70–79 years participants)**

https://en.uit.no/forskning/forskningsgrupper/sub?p_document_id=591555&sub_id=591882

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-ÅRE IVERSEN, FORMIDLINGSTJENESTEN, UIT.NO/HELSEFAK

UNIVERSITETET I TROMSØ UIT
DET HELSEVITENSKAPELIGE FAKULTET

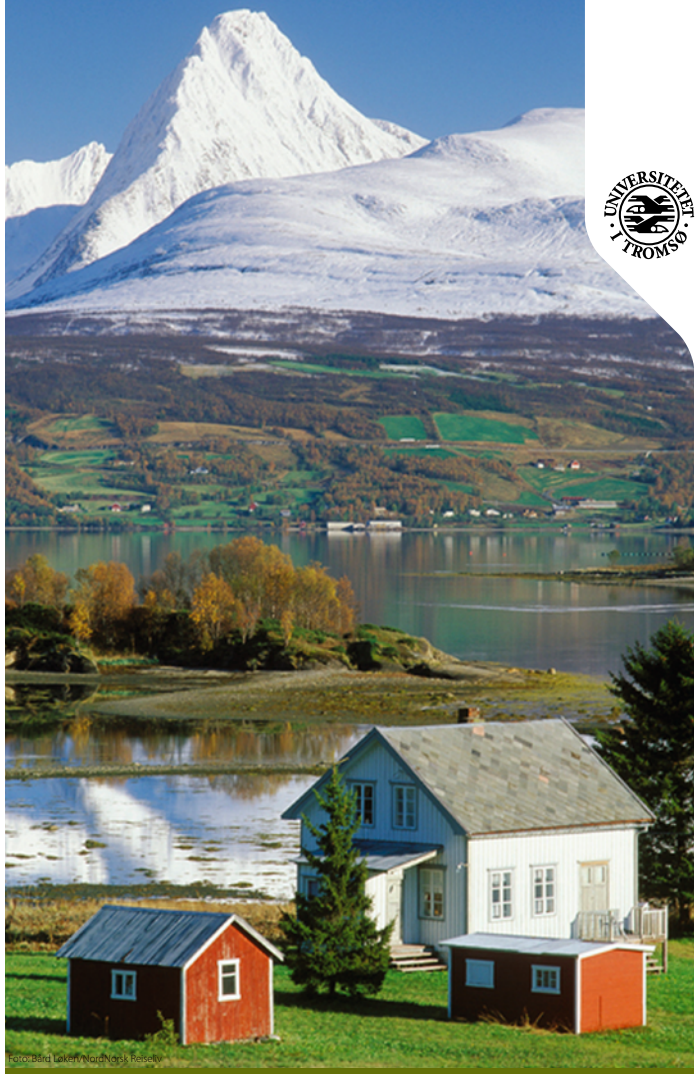


Foto: Bård Løkeli/NordNorsk Reiseliv

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.

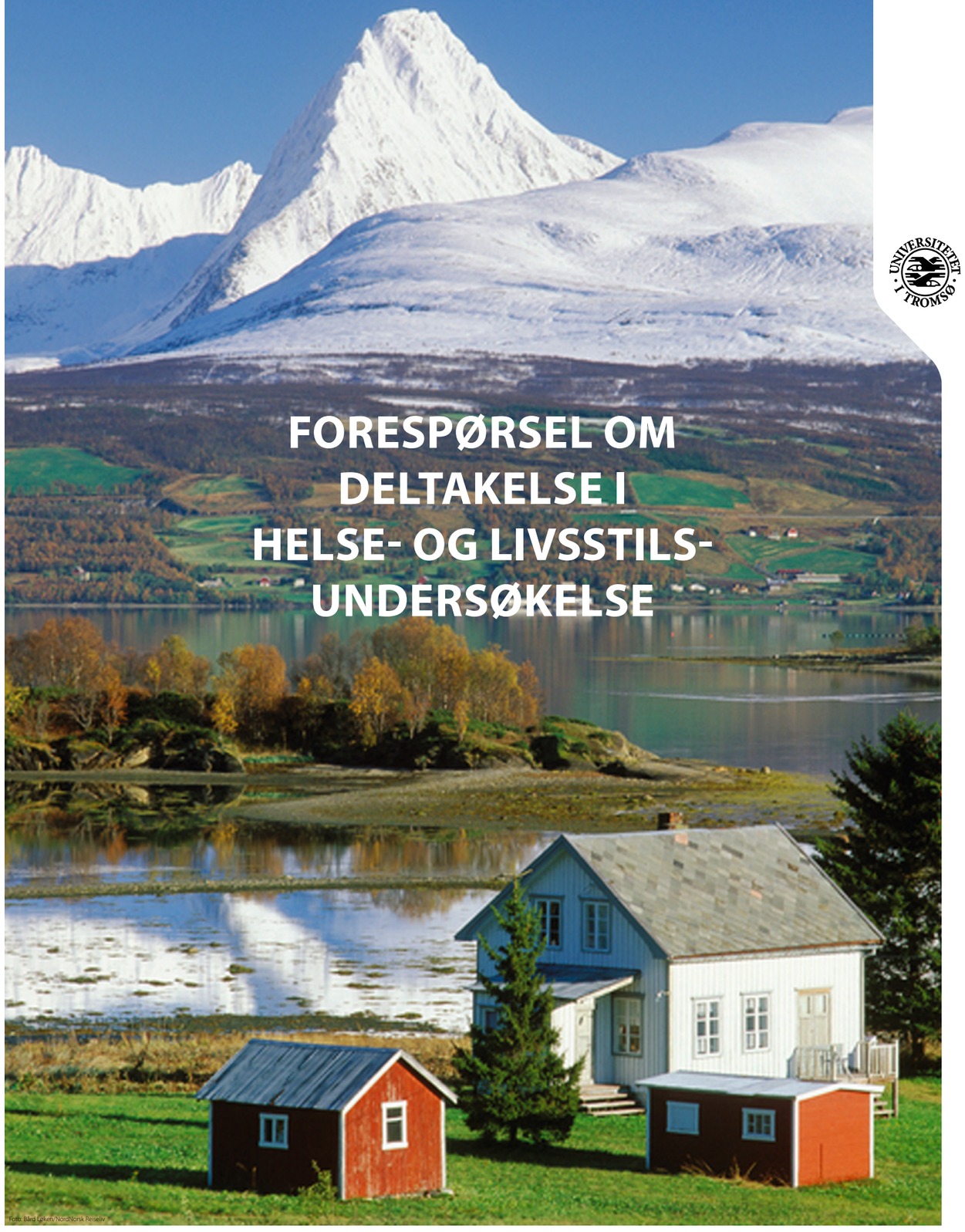
SAMINOR 2

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

GRAFISK FORM: JØRN-KÅRE IVERSEN, FORMDUNGSTJENESTEN, UIT, NO/HELSEFAK

UNIVERSITETET I TROMSØ UIT

DET HELSEVITENSKAPELIGE FAKULTET



FORESPØRSEL OM DELTAKELSE I HELSE- OG LIVSSTILS-UNDERSØKELSE

Foto: Bård Løken/Nordnorsk Reiseliv

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. Karasjok kommune er først ut i Finnmark.

Vi inviterer deg til å møte opp på denne undersøkelsen som vil finne sted i tidsrommet **28. januar til 21. februar 2013** ved:

Sentrumsbygget, Fidnodatgeaidnu 39 i Karasjok.

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 **11:00**, og vi har lunsj i tidsrommet **12:00 -12:30**.

	Mandag	Tirsdag	Onsdag	Torsdag	Fredag	Lørdag
Uke 5	10:00 - 16:30	10:00 - 18:00	10:00 - 16:30	10:00 - 16:30	10:00 - 16:30	STENGT
Uke 6	10:00 - 16:30	10:00 - 18:00	STENGT	10:00 - 16:30	10:00 - 16:30	10:00 - 14:00
Uke 7	10:00 - 16:30	10:00 - 18:00	10:00 - 16:30	10:00 - 16:30	10:00 - 16:30	10:00 - 14:00
Uke 8	10:00 - 16:30	10:00 - 18:00	10:00 - 16:30	10:00 - 16:30	STENGT	STENGT

Senter for samisk helseforskning / Sámi dearvvašvuođadutkama guovddáš
Det helsevitenskapelige fakultet, Institutt for samfunnsmedisin,
Universitetet i Tromsø, NO-9037 Tromsø
http://site.uit.no/helseoglivsstil/ • 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.

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 (samisk talende).

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,
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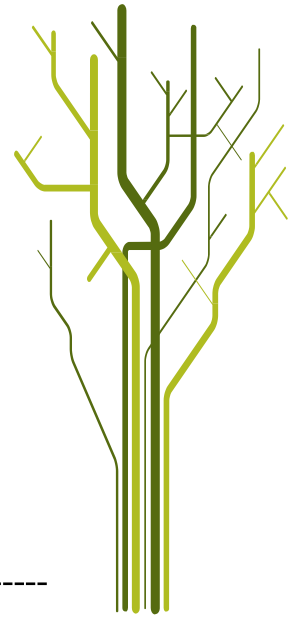
Sentralbord: 77 64 40 00 • Faks: 77 64 48 31• Mobil: 404 90 467

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

Helse- og livsstils-undersøkelse

Vi ber deg fylle ut spørreskjema så nøye som mulig og levere det ved oppmøte til den innkalte helseundersøkelsen. Skjema skal leses optisk. Vennligst bruk blå eller sort penn. Bruk blokkbokstaver. Du kan ikke bruke komma, forhøy for eksempel 0,5 til 1.

1. **I hvilket år er du født?** ..... Årstall

2. **Er du?** ..... Kvinne ☐ Mann ☐

3. **Hva er din sivilstatus?**

☐ Gift ☐ Samboer ☐ Skilt
☐ Ugift ☐ Enke/enkemann

4. **Hvor mange personer bor det i din husstand?** ..... Antall personer

5. **Hvor mange års skolegang har du gjennomført?**
(Ta med alle år du har gått på skole eller studert) ..... Antall år

6. **Hvor stor er familiens/husstandens bruttoinntekt pr. år?**

☐ Under kr 150 000 ☐ Kr 150 000–300 000
☐ Kr 301 000–450 000 ☐ Kr 451 000–600 000
☐ Kr 601 000–750 000 ☐ Kr 751 000–900 000
☐ Over kr 900 000

Hjerte-karsykdommer

7. **Bruker du medisin mot høyt blodtrykk?** ..... Ja, nå ☐ Før, men ikke nå ☐ Aldri brukt ☐

8. **Hvis du bruker eller tidligere har brukt blodtrykksmedisin, omtrent hvor gammel var du første gang du begynte med slik medisin?** Alder

9. **Har du hatt hjerteinfarkt?**

☐ Nei, aldri ☐ 1 gang ☐ 2 ganger ☐ 3 eller flere ganger

10. **Hvis ja, hva var din alder første gang du fikk hjerteinfarkt?** ..... Alder

11. **Har du angina pectoris (hjertekrampe)?** ..... ☐ Ja ☐ Nei

12. **Hvis ja, hva var din alder første gang du fikk angina pectoris?** ..... Alder

13. **Hvis ja, hvor ofte har du merket slike smerter i løpet av den siste måneden?**

Sjelden	1 gang pr. uke	2-3 ganger pr. uke	4-6 ganger pr. uke	7 eller flere ganger pr. uke
☐	☐	☐	☐	☐

14. **Har du blitt hjerteoperert (bypass)?** ..... ☐ Ja ☐ Nei

15. **Har du blitt blokket/fått innsatt stent?** ..... ☐ Ja ☐ Nei

16. **Har legen sagt at du har hjerteflimmer?** ..... ☐ Ja ☐ Nei

Fysisk aktivitet

17. **Vi ber deg angi din fysiske aktivitet etter en skala fra svært lite til svært mye da du var 14 år, 30 år og i dag.** Skalaen nedenfor går fra 1-10. Med fysisk aktivitet mener vi både arbeid i hjemmet og i yrkeslivet, samt trening og annen fysisk aktivitet som turgåing o.l. Sett kryss under det tallet som best angir ditt nivå av fysisk aktivitet.

	Svært lite									Svært mye
Alder	1	2	3	4	5	6	7	8	9	10
14 år	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
30 år	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
I dag	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Diabetes (sukkersyke)

18. **Har du noen gang fått påvist diabetes (for høyt blodsukker)?** ..... ☐ Ja ☐ Nei
Dersom nei, gå videre til spørsmål 28 om spisevaner

19. **Dersom ja, hvilken type diabetes har du fått påvist?**
(Sett ett eller flere kryss)

Svangerskapsdiabetes ..... ☐
Diabetes type I ..... ☐
Diabetes type II ..... ☐

20. **Hvordan ble din diabetes oppdaget?**

Jeg søkte lege pga. symptomer ..... ☐ Ja ☐ Nei

Ble oppdaget uten at jeg hadde symptomer
(legeattest, bedriftskontroll, svangerskapskontroll, undersøkelse for annen sykdom e.l.) ..... ☐ Ja ☐ Nei

21. **Hvor gammel var du da din diabetes ble oppdaget?** ..... Alder

INSULIN

22. **Bruker du insulin mot din diabetes?** ..... Ja, nå ☐ Før, men ikke nå ☐ Aldri brukt ☐

Dersom du bruker eller har brukt insulin:

23. **Hvor gammel var du da du begynte med insulin?** ..... Alder ☐☐

24. **Hvor mange ganger pr. dag tar du/tok du vanligvis insulin?** ..... ☐☐ ganger

25. **Hvor mange enheter insulin tar du/tok du vanligvis til sammen pr. dag?** ..... ☐☐ enheter(E)

TABLETTER

26. **Bruker du tabletter mot din diabetes?**	Ja, nå	Før, men ikke nå	Aldri brukt
	☐	☐	☐

Dersom du bruker eller har brukt tabletter:

27. **Hvor gammel var du da du begynte med tabletter mot diabetes?** ..... Alder ☐☐

Spisevaner

Sett et kryss i ruten under det tallet som beskriver spisevanene dine slik du synes de har vært **de siste 4 ukene:**

28. **Hvor fornøyd har du vært med spisevanene dine?** (Sett ett kryss)

	1	2	3	4	5	6	7	
Svært misfornøyd	☐	☐	☐	☐	☐	☐	☐	Svært fornøyd

29. **Har du trøstespist eller spist ekstra på grunn av at du har vært nedstemt eller følt deg utilfreds?** (Sett ett kryss)

	1	2	3	4	5	6	7	
Aldri	☐	☐	☐	☐	☐	☐	☐	Hver dag

30. **Har du hatt skyldfølelse i forbindelse med spising?** (Sett ett kryss)

	1	2	3	4	5	6	7	
Aldri	☐	☐	☐	☐	☐	☐	☐	Hver dag

31. **Har du følt at det er nødvendig for deg å følge strenge dietter eller andre matritualer for å holde kontroll med hvor mye du spiser?** (Sett ett kryss)

	1	2	3	4	5	6	7	
Aldri	☐	☐	☐	☐	☐	☐	☐	Hver dag

32. **Har du følt at du er for tykk?** (Sett ett kryss)

	1	2	3	4	5	6	7	
Aldri	☐	☐	☐	☐	☐	☐	☐	Hver dag

Røykevaner

33. **Har du noen gang røykt daglig?** ..... ☐ Ja ☐ Nei

Dersom du **aldri har røykt daglig**, kan du gå videre til spørsmål 38.

34. **Røyker du daglig nå?** ..... ☐ Ja ☐ Nei

35. **Hvis du har sluttet å røyke daglig, hvor gammel var du da du sluttet?** ..... Alder ☐☐

36. **Hvor mange år til sammen har du røykt daglig?** År ☐☐

37. **Hvor mange sigaretter/rulletobakk har du i gjennomsnitt røykt daglig i de årene du har røykt daglig?** ..... ☐☐☐

38. **Bor du sammen med noen som røyker?** ..... ☐ Ja ☐ Nei

Smerter

39. **Har du smerter nå som har vart i tre måneder eller lengre?** ..... ☐ Ja ☐ Nei

40. **Hvis ja, vennligst angi hvor sterke smerter du har hatt den siste uken:** (Sett ett kryss)

Ingen smerte										Verste tenkelige smerter
0	1	2	3	4	5	6	7	8	9	10
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

41. **Angi hvor smertene er mest plagsomme:** (Sett ett kryss)

☐ Nakke ☐ Korsrygg ☐ Annet

Kosthold

Vi er interessert i å få kjennskap til hvordan kostholdet ditt er **vanligvis**. Kryss av for hvert spørsmål om **hvor ofte (antall ganger)** du i **gjennomsnitt siste året** har brukt den aktuelle matvaren, og **hvor mye** du pleier å spise/drikke hver gang.

DRIKKE

42. **Hvor mange glass melk drikker du vanligvis av hver type?** (Sett ett kryss pr. linje)

	aldri/ sjelden	1–4 pr. uke	5–6 pr. uke	1 pr. dag	2–3 pr. dag	4+ pr. dag
Helmelk *(søt, sur)*	☐	☐	☐	☐	☐	☐
Lettmelk *(søt, sur)*	☐	☐	☐	☐	☐	☐
Ekstra lettmelk	☐	☐	☐	☐	☐	☐
Skummet *(søt, sur)*	☐	☐	☐	☐	☐	☐

43. **Hvor mange kopper kaffe/te drikker du vanligvis av hver sort?** (Sett ett kryss pr. linje)

	aldri/ sjelden	1–6 pr. uke	1 pr. dag	2–3 pr. dag	4–5 pr. dag	6–7 pr. dag	8+ pr. dag
Kokekaffe, presskanne	☐	☐	☐	☐	☐	☐	☐
Traktekaffe	☐	☐	☐	☐	☐	☐	☐
Espresso	☐	☐	☐	☐	☐	☐	☐
Latte	☐	☐	☐	☐	☐	☐	☐
Pulverkaffe	☐	☐	☐	☐	☐	☐	☐
Svart te	☐	☐	☐	☐	☐	☐	☐
Grønn te	☐	☐	☐	☐	☐	☐	☐

44. **Bruker du følgende i kaffe?**

Sukker *(ikke kunstig søtstoff)* ..... ☐ Ja ☐ Nei

Melk eller fløte ..... ☐ Ja ☐ Nei

45. **Bruker du følgende i te?**

Sukker *(ikke kunstig søtstoff)* ..... ☐ Ja ☐ Nei

Melk eller fløte ..... ☐ Ja ☐ Nei

46. Hvor mange glass vann drikker du vanligvis?
(Sett ett kryss pr. linje)

	aldri/ sjelden	1–6 pr. uke	1 pr. dag	2–3 pr. dag	4–5 pr. dag	6–7 pr. dag	8+ pr. dag
Springvann	☐	☐	☐	☐	☐	☐	☐
Flaskevann	☐	☐	☐	☐	☐	☐	☐

47. Hvor mange glass juice, saft og brus drikker du vanligvis? (Sett ett kryss pr. linje)

	aldri/ sjelden	1–3 pr. uke	4–6 pr. uke	1 pr. dag	2–3 pr. dag	4+ pr. dag
Appelsinjuice	☐	☐	☐	☐	☐	☐
Annen juice	☐	☐	☐	☐	☐	☐
Saft/brus med sukker	☐	☐	☐	☐	☐	☐
Saft/brus, sukkerfri	☐	☐	☐	☐	☐	☐

YOGHURT/KORNBLANDING

48. Hvor ofte spiser du yoghurt (1 beger)? (Sett ett kryss)

☐ Aldri/sjelden ☐ 1–3 pr. uke
☐ 4–6 pr. uke ☐ 1 + pr. dag

49. Hvor ofte spiser du kornblanding, havregryn eller müsli?
(Sett ett kryss)

☐ Aldri/sjelden ☐ 1–3 pr. uke
☐ 4–6 pr. uke ☐ 1 + pr. dag

BRØDMAT

50. Hvor mange skiver brød/rundstykker og knekkebrød/ skonrokker spiser du vanligvis? (½ rundstykke = 1 brødskive)
(Sett ett kryss pr. linje)

	aldri/ sjelden	1–4 pr. uke	5–7 pr. uke	2–3 pr. dag	4–5 pr. dag	6+ pr. dag
Grovt brød	☐	☐	☐	☐	☐	☐
Kneipp/halvfint	☐	☐	☐	☐	☐	☐
Fint brød/baguett	☐	☐	☐	☐	☐	☐
Knekkebrød o.l.	☐	☐	☐	☐	☐	☐

Nedenfor er det spørsmål om bruk av ulike påleggstyper. Vi spør om hvor mange brødskiver/knekkebrød med det aktuelle pålegget du pleier å spise. Dersom du også bruker matvarene i andre sammenhenger enn til brød (f.eks. til vafler, frokostblandinger, grøt), ber vi om at du tar med dette når du besvarer spørsmålene.

51. På hvor mange brødskiver/knekkebrød bruker du?
(Sett ett kryss pr. linje)

	aldri/ sjelden	1–3 pr. uke	4–6 pr. uke	1 pr. dag	2–3 pr. dag	4+ pr. dag
Syltetøy	☐	☐	☐	☐	☐	☐
Brunost *(helfet)*	☐	☐	☐	☐	☐	☐
Brunost *(halvfet, mager)*	☐	☐	☐	☐	☐	☐
Hvitost *(helfet)*	☐	☐	☐	☐	☐	☐
Hvitost *(halvfet, mager)*	☐	☐	☐	☐	☐	☐
Rekesalat, italiensk o.l.	☐	☐	☐	☐	☐	☐
Leverpostei	☐	☐	☐	☐	☐	☐
Magert kjøttpålegg *(kokt skinke o.l.)*	☐	☐	☐	☐	☐	☐
Fett kjøttpålegg *(salami, fenalår o.l.)*	☐	☐	☐	☐	☐	☐

52. På hvor mange brødskiver/knekkebrød pr. uke har du i gjennomsnitt siste året spist: (Sett ett kryss pr. linje)

	aldri/ sjelden	1 pr. uke	2–3 pr. uke	4–6 pr. uke	7–9 pr. uke	10+ pr. uke
Makrell i tomat, røkt makrell	☐	☐	☐	☐	☐	☐
Kaviar	☐	☐	☐	☐	☐	☐
Sild/ansjos	☐	☐	☐	☐	☐	☐
Laks *(gravet/røkt)*	☐	☐	☐	☐	☐	☐
Annet fiskepålegg	☐	☐	☐	☐	☐	☐

53. Dersom du bruker fett på brødet, hvor tykt lag pleier du å smøre på? (En kuvertpakke med margarin veier 12 gram) (Sett ett kryss)

☐ Skrapet *(3 g)* ☐ Tynt lag *(5 g)*
☐ Godt dekket *(8 g)* ☐ Tykt lag *(12 g)*

54. Hva slags fett bruker du vanligvis på brødet?
(Sett gjerne flere kryss)

☐ Bruker ikke fett på brødet
☐ Smør
☐ Hard margarin *(f.eks. Melange)*
☐ Myk margarin *(f.eks. Soft, Vita)*
☐ Smørblandet margarin *(f.eks. Bremyk)*
☐ Brelett
☐ Lettmargarin *(f.eks. Soft light, Vita Lett)*
☐ Margarin med olivenolje *(f.eks. Brelett oliven, Soft oliven)*

FRUKT OG GRØNNSAKER

55. Hvor ofte spiser du frukt? (Sett ett kryss pr. linje)

	aldri/ sjelden	1–3 pr. mnd.	1 pr. uke	2–4 pr. uke	5–6 pr. uke	1 pr. dag	2+ pr. dag
Epler/pærer	☐	☐	☐	☐	☐	☐	☐
Appelsiner o.l.	☐	☐	☐	☐	☐	☐	☐
Bananer	☐	☐	☐	☐	☐	☐	☐
Annen frukt	☐	☐	☐	☐	☐	☐	☐

56. Hvor ofte spiser du potet? (Sett ett kryss pr. linje)

	1-4 ganger pr. mnd	5-6 ganger pr. uke	1 gang pr. dag	2–4 pr. uke	2 ganger pr. dag
Kokt	☐	☐	☐	☐	☐
Most	☐	☐	☐	☐	☐
Stekt/fritert	☐	☐	☐	☐	☐

57. Hvor ofte spiser du ulike typer grønnsaker?
(Sett ett kryss pr. linje)

	aldri/ sjelden	1–3 pr. mnd.	1 pr. uke	2 pr. uke	3 pr. uke	4–5 pr. uke	6–7 pr. uke
Gulrøtter	☐	☐	☐	☐	☐	☐	☐
Kål	☐	☐	☐	☐	☐	☐	☐
Kålrot	☐	☐	☐	☐	☐	☐	☐
Brokkoli/blomkål	☐	☐	☐	☐	☐	☐	☐

	aldri/ sjelden	1–3 pr. mnd.	1 pr. uke	2 pr. uke	3 pr. uke	4–5 pr. uke	6–7 pr. uke
Blandet salat	☐	☐	☐	☐	☐	☐	☐
Tomat	☐	☐	☐	☐	☐	☐	☐
Grønnsakblanding	☐	☐	☐	☐	☐	☐	☐
Løk	☐	☐	☐	☐	☐	☐	☐
Bønner	☐	☐	☐	☐	☐	☐	☐
Erter	☐	☐	☐	☐	☐	☐	☐
Andre grønnsaker	☐	☐	☐	☐	☐	☐	☐

58. **For de grønnsakene du spiser, kryss av for hvor mye du spiser hver gang:** (Sett ett kryss for hver sort)

Gulrøtter	☐ 1/2 stk.	☐ 1 stk.	☐ 1 1/2 stk.	☐ 2+ stk.
Potet	☐ 1-2 stk.	☐ 3-4 stk.	☐ 5-6 stk.	☐ 7+ stk.
Kål	☐ 1/2 dl	☐ 1 dl	☐ 1 1/2 dl	☐ 2+ dl
Kålrot	☐ 1/2 dl	☐ 1 dl	☐ 1 1/2 dl	☐ 2+ dl
Brokkoli/blomkål	☐ 1–2 buketter	☐ 3–4 buketter	☐ 5+ buketter	
Blandet salat	☐ 1 dl	☐ 2 dl	☐ 3 dl	☐ 4+ dl
Tomat	☐ 1/4 stk.	☐ 1/2 stk.	☐ 1 stk.	☐ 2+ stk.
Grønnsakblanding	☐ 1/2 dl	☐ 1 dl	☐ 2 dl	☐ 3+ dl
Bønner	☐ 1–2 ss	☐ 3–4 ss	☐ 5–6 ss	☐ 7+ ss
Erter	☐ 1–2 ss	☐ 3–4 ss	☐ 5–6 ss	☐ 7+ ss

RIS, SPAGHETTI, GRØT, SUPPE

59. **Hvor ofte bruker du ris og spaghetti/makaroni?**
(Sett ett kryss pr. linje)

	aldri/ sjelden	1–3 pr. mnd.	1 pr. uke	2 pr. uke	3+ pr. uke
Ris	☐	☐	☐	☐	☐
Spaghetti, makaroni, nudler	☐	☐	☐	☐	☐

60. **Hvor ofte spiser du grøt?** (Sett ett kryss pr. linje)

	aldri/ sjelden	1 pr. mnd.	2–3 pr. mnd.	1 pr. uke	2–6 pr. uke	1+ pr. dag
Risengrynsgrøt	☐	☐	☐	☐	☐	☐
Annen grøt *(havre o.l.)*	☐	☐	☐	☐	☐	☐

61. **Hvor ofte spiser du suppe?** (Sett ett kryss pr. linje)

	aldri/ sjelden	1–3 pr. mnd.	1 pr. uke	2 pr. uke	3+ pr. uke
Som hovedrett	☐	☐	☐	☐	☐
Som forrett, lunsj eller kveldsmat	☐	☐	☐	☐	☐

FISK

62. **Vi vil gjerne vite hvor ofte du pleier å spise fisk, og ber deg fylle ut spørsmålene om fiskeforbruk så godt du kan.** Tilgangen på fisk kan variere gjennom året. Vær vennlig å markere i hvilke årstider du spiser de ulike fiskeslagene.

	aldri/ sjelden	like mye hele året	vinter	vår	sommer	høst
Torsk, sei, hyse, lyr	☐	☐	☐	☐	☐	☐
Steinbit, flyndre, uer	☐	☐	☐	☐	☐	☐
Laks, sjøørret	☐	☐	☐	☐	☐	☐
Kveite	☐	☐	☐	☐	☐	☐
Makrell	☐	☐	☐	☐	☐	☐
Sild	☐	☐	☐	☐	☐	☐
Ferskvannsfisk *(abbor, gjedde, harr, røye, sik, ørret)*	☐	☐	☐	☐	☐	☐
Annen fisk	☐	☐	☐	☐	☐	☐

63. **Med tanke på de periodene av året der du spiser fisk, hvor ofte pleier du å spise følgende til <u>middag</u>?** (Sett ett kryss pr. linje)

	aldri/ sjelden	1 pr. mnd.	2–3 pr. mnd.	1 pr. uke	2+ pr. uke
<u>Kokt</u> torsk, sei, hyse, lyr	☐	☐	☐	☐	☐
<u>Stekt</u> torsk, sei, hyse, lyr	☐	☐	☐	☐	☐
Steinbit, flyndre, uer	☐	☐	☐	☐	☐
Laks, sjøørret	☐	☐	☐	☐	☐
Kveite	☐	☐	☐	☐	☐
Makrell	☐	☐	☐	☐	☐
Sild	☐	☐	☐	☐	☐
Ferskvannsfisk *(abbor, gjedde, harr, røye, sik, ørret)*	☐	☐	☐	☐	☐
Annen fisk	☐	☐	☐	☐	☐

64. **Dersom du spiser fisk, hvor mye spiser du vanligvis hver gang?** (1 skive/stykke = 150 gram)

Kokt fisk *(skive)*	☐ 1	☐ 1 ½	☐ 2	☐ 3+
Stekt fisk *(stykke)*	☐ 1	☐ 1 ½	☐ 2	☐ 3+

65. **Hvor mange ganger pr. år spiser du fiskeinnmat?**
(Sett ett kryss pr. linje)

	0	1–3	4–6	7–9	10+
Rogn	☐	☐	☐	☐	☐
Fiskelever	☐	☐	☐	☐	☐

66. **Dersom du spiser fiskelever, hvor mange spiseskjeer pleier du å spise hver gang?** (Sett ett kryss)

☐ 1 ☐ 2 ☐ 3–4 ☐ 5–6 ☐ 7+

67. **Hvor ofte bruker du følgende typer fiskemat?** (Sett ett kryss pr. linje)

	aldri/ sjelden	1 pr. mnd.	2–3 pr. mnd.	1 pr. uke	2+ pr. uke
Fiskekaker/-pudding/-boller	☐	☐	☐	☐	☐
Plukkfisk/fiskegrateng	☐	☐	☐	☐	☐
Frityrfisk/fiskepinner	☐	☐	☐	☐	☐
Andre fiskeretter	☐	☐	☐	☐	☐

68. **Hvor stor mengde pleier du vanligvis å spise av de ulike rettene?** (Sett ett kryss pr. linje)

Fiskekaker/pudding/boller *(stk.) (2 fiskeboller=1 fiskekake)*	☐ 1	☐ 2	☐ 3	☐ 4+
Plukkfisk, fiskegrateng *(dl)*	☐ 1–2	☐ 3–4	☐ 5+	
Frityrfisk, fiskepinner *(stk.)*	☐ 1–2	☐ 3–4	☐ 5–6	☐ 7+

I tillegg til informasjon om fiskeforbruk er det viktig å få kartlagt hvilket tilbehør som blir servert til fisk.

69. Hvor ofte bruker du følgende til fisk?

	aldri/ sjelden	1 pr. mnd.	2–3 pr. mnd.	1 pr. uke	2+pr. uke
Smeltet/fast smør	☐	☐	☐	☐	☐
Smeltet/fast margarin	☐	☐	☐	☐	☐
Seterrømme *(35%)*	☐	☐	☐	☐	☐
Lettrømme *(20%)*	☐	☐	☐	☐	☐
Saus med fett *(hvit/brun)*	☐	☐	☐	☐	☐
Saus uten fett *(hvit/brun)*	☐	☐	☐	☐	☐

70. For de ulike typene tilbehør du bruker til fisk, vær vennlig å kryss av for hvor mye du vanligvis pleier å spise:

Smeltet/fast smør *(ss)*	☐ ½	☐ 1	☐ 2	☐ 3	☐ 4+
Smeltet/fast margarin *(ss)*	☐ ½	☐ 1	☐ 2	☐ 3	☐ 4+
Seterrømme *(ss)*	☐ ½	☐ 1	☐ 2	☐ 3	☐ 4+
Lettrømme *(ss)*	☐ ½	☐ 1	☐ 2	☐ 3	☐ 4+
Saus med fett *(dl)*	☐ ¼	☐ ½	☐ ¾	☐ 1	☐ 2+
Saus uten fett *(dl)*	☐ ¼	☐ ½	☐ ¾	☐ 1	☐ 2+

71. Hvor ofte spiser du skalldyr? (f.eks. reker, krabbe og skjell)
(Sett ett kryss)

☐ Aldri/sjelden ☐ 1 pr. mnd.
☐ 2–3 pr. mnd. ☐ 1+ pr. uke

72. Hvor mange måseegg eller egg fra annen sjøfugl spiser du i året? (Sett ett kryss)

Aldri	1-3	4-6	7-9	10-15	16+
☐	☐	☐	☐	☐	☐

73. Hvor ofte har du spist ferskvannsfisk? (abbor, gjedde, harr, røye, sik, ørret) (Sett ett kryss pr. linje)

	aldri/ sjelden	1 pr. mnd.	2-3 pr. mnd.	1 pr. uke	2-3 pr. uke	4+ pr. uke
Barndom	☐	☐	☐	☐	☐	☐
Ungdom 13-19 år	☐	☐	☐	☐	☐	☐
Voksen (før siste året)	☐	☐	☐	☐	☐	☐

KJØTT

74. Hvor ofte spiser du følgende kjøttretter?
(Sett ett kryss for hver rett)

	aldri/ sjelden	1-2 pr. mnd.	3-4 pr. mnd.	2-3 pr. uke	4-6 pr. uke	7+ pr. uke
Reinkjøtt	☐	☐	☐	☐	☐	☐
Elgkjøtt	☐	☐	☐	☐	☐	☐

75. Hvor ofte spiser du følgende kjøtt- og fjærkreretter?
(Sett ett kryss for hver rett)

	aldri/ sjelden	1 pr. mnd.	2–3 pr. mnd.	1 pr. uke	2+ pr. uke
Steik *(okse, svin, får)*	☐	☐	☐	☐	☐
Koteletter *(okse, svin, får)*	☐	☐	☐	☐	☐
Biff *(okse, svin, får)*	☐	☐	☐	☐	☐
Kjøttkaker, karbonader	☐	☐	☐	☐	☐
Pølser	☐	☐	☐	☐	☐
Rype, annen viltfugl	☐	☐	☐	☐	☐
Gryterett, lapskaus	☐	☐	☐	☐	☐
Pizza med kjøtt	☐	☐	☐	☐	☐
Kylling med skinn	☐	☐	☐	☐	☐
Kylling uten skinn	☐	☐	☐	☐	☐
Bacon, flesk	☐	☐	☐	☐	☐
Andre kjøttretter	☐	☐	☐	☐	☐
Blodmat *(får, storfe, rein, elg)*	☐	☐	☐	☐	☐

76. Dersom du spiser følgende retter, oppgi mengden du vanligvis spiser: (Sett ett kryss pr. linje)

Steik *(skiver)*	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5+
Koteletter *(stk.)*	☐ ½	☐ 1	☐ 1½	☐ 2+	
Kjøttkaker, karbonader *(stk.)*	☐ 1	☐ 2	☐ 3	☐ 4+	
Pølser *(stk. à 150g)*	☐ ½	☐ 1	☐ 1½	☐ 2+	
Gryterett, lapskaus *(dl)*	☐ 1–2	☐ 3	☐ 4	☐ 5+	
Pizza m/kjøtt *(stykke à 100 g)*	☐ 1	☐ 2	☐ 3	☐ 4+	

77. Hvilke sauser bruker du til kjøttretter og pastaretter?
(Sett ett kryss pr. linje)

	aldri/ sjelden	1 pr. mnd.	2–3 pr. mnd.	1 pr. uke	2+ pr. uke
Brun saus	☐	☐	☐	☐	☐
Sjysaus	☐	☐	☐	☐	☐
Tomatsaus	☐	☐	☐	☐	☐
Saus med fløte/rømme	☐	☐	☐	☐	☐

78. Hvor mye bruker du vanligvis av disse sausene?

Brun saus *(dl)*	☐ ¼	☐ ½	☐ ¾	☐ 1	☐ 2+
Sjysaus *(dl)*	☐ ¼	☐ ½	☐ ¾	☐ 1	☐ 2+
Tomatsaus *(dl)*	☐ ¼	☐ ½	☐ ¾	☐ 1	☐ 2+
Saus med fløte/rømme *(dl)*	☐ ¼	☐ ½	☐ ¾	☐ 1	☐ 2+

ANDRE MATVARER

79. Hvor mange egg spiser du vanligvis i løpet av en uke?
(stekte, kokte, eggerøre, omelett) (Sett ett kryss)

☐ 0 ☐ 1 ☐ 2 ☐ 3–4 ☐ 5–7 ☐ 8-14 ☐ 15+

80. Hvor ofte spiser du iskrem? (til dessert, Krone-is osv.)
(Sett ett kryss for hvor ofte du spiser iskrem om sommeren, og ett kryss for resten av året)

	aldri/ sjelden	1 pr. mnd.	2–3 pr. mnd.	1 pr. uke	2+ pr. uke
Om sommeren	☐	☐	☐	☐	☐
Resten av året	☐	☐	☐	☐	☐

81. Hvor mye is spiser du vanligvis hver gang?
(Sett ett kryss)

☐ 1 dl ☐ 2 dl ☐ 3 dl ☐ 4+ dl

82. Hvor ofte spiser du bakevarer som boller, kaker, wienerbrød eller småkaker? (Sett ett kryss pr. linje)

	aldri/ sjelden	1–3 pr. mnd.	1 pr. uke	2–3 pr. uke	4–6 pr. uke	1+ pr. dag
Gjærbakst *(boller o.l.)*	☐	☐	☐	☐	☐	☐
Wienerbrød, kringle	☐	☐	☐	☐	☐	☐
Kaker	☐	☐	☐	☐	☐	☐

	aldri/ sjelden	1–3 pr. mnd.	1 pr. uke	2–3 pr. uke	4–6 pr. uke	1+ pr. dag
Pannekaker	☐	☐	☐	☐	☐	☐
Vafler	☐	☐	☐	☐	☐	☐
Småkaker, kjeks	☐	☐	☐	☐	☐	☐
Lefser, lomper	☐	☐	☐	☐	☐	☐

83. **Hvor ofte spiser du dessert?** (Sett ett kryss pr. linje)

	aldri/ sjelden	1 pr. mnd.	2–3 pr. mnd.	1 pr. uke	2–3 pr. uke	4+ pr. uke
Pudding *(f.eks. sjokolade/ karamell)*	☐	☐	☐	☐	☐	☐
Riskrem, fromasj	☐	☐	☐	☐	☐	☐
Kompott, fruktgrøt, hermetisk frukt	☐	☐	☐	☐	☐	☐
Jordbær *(friske, frosne)*	☐	☐	☐	☐	☐	☐
Andre bær *(friske, frosne)*	☐	☐	☐	☐	☐	☐

84. **Hvor ofte spiser du sjokolade?** (Sett ett kryss pr. linje)

	aldri/ sjelden	1–3 pr. mnd.	1 pr. uke	2–3 pr. uke	4–6 pr. uke	1+ pr. dag
Mørk sjokolade	☐	☐	☐	☐	☐	☐
Lys sjokolade	☐	☐	☐	☐	☐	☐

85. **Dersom du spiser sjokolade, hvor mye pleier du vanligvis å spise hver gang?** Tenk deg størrelsen på en Kvikk-Lunsj sjokolade, og oppgi hvor mye du spiser i forhold til den.

☐ ¼ ☐ ½ ☐ ¾ ☐ 1 ☐ 1½ ☐ 2+

86. **Hvor ofte spiser du annet søtt godteri?** (Sett ett kryss)

aldri/ sjelden	1–3 pr. mnd.	1 pr. uke	2–3 pr. uke	4–6 pr. uke	1+ pr. dag
☐	☐	☐	☐	☐	☐

87. **Hvor ofte spiser du salt snacks?** (Sett ett kryss pr. linje)

	aldri/ sjelden	1–3 pr. mnd.	1 pr. uke	2–3 pr. uke	4–6 pr. uke	1+ pr. dag
Potetchips	☐	☐	☐	☐	☐	☐
Peanøtter	☐	☐	☐	☐	☐	☐
Andre nøtter	☐	☐	☐	☐	☐	☐
Annen snacks	☐	☐	☐	☐	☐	☐

TRAN OG FISKEOLJEKAPSLER

88. **Bruker du tran (flytende)?** ☐ Ja ☐ Nei

89. **Hvis ja, hvor ofte tar du tran?** (Sett ett kryss pr. linje.)

	aldri/ sjelden	1–3 pr. mnd.	1 pr. uke	2–6 pr. uke	daglig
Om vinteren	☐	☐	☐	☐	☐
Resten av året	☐	☐	☐	☐	☐

90. **Hvor mye tran pleier du å ta hver gang?**

☐ 1 ts ☐ ½ ss ☐ 1+ss

91. **Bruker du tranpiller/fiskeoljekapsler?** ☐ Ja ☐ Nei

92. **Hvis ja, hvor ofte tar du tranpiller/fiskeoljekapsler?** (Sett ett kryss pr. linje.)

	aldri/ sjelden	1–3 pr. mnd.	1 pr. uke	2–6 pr. uke	daglig
Om vinteren	☐	☐	☐	☐	☐
Resten av året	☐	☐	☐	☐	☐

93. **Hvilken type tranpiller/fiskeoljekapsler bruker du vanligvis, og hvor mange pleier du å ta hver gang?**

Navn på produkt: _____

Antall: ☐ 1 ☐ 2 ☐ 3+

Kosttilskudd

94. **Bruker du kosttilskudd?** (vitaminer/mineraler) ☐ Ja ☐ Nei

Alkohol

95. **Er du totalavholdskvinne/mann?** ☐ Ja ☐ Nei

96. **Hvis nei, hvor ofte og hvor mye drakk du i gjennomsnitt siste året?** (Sett ett kryss pr. linje)

	aldri/ sjelden	1 pr. mnd.	2–3 pr. mnd.	1 pr. uke	2–4 pr. uke	5–6 pr. uke	1 pr. dag	2+ pr. dag
Øl/rusbrus *(½ l.)*	☐	☐	☐	☐	☐	☐	☐	☐
Vin *(glass)*	☐	☐	☐	☐	☐	☐	☐	☐
Brennevin *(drink/shot)*	☐	☐	☐	☐	☐	☐	☐	☐
Likør/hetvin *(glass)*	☐	☐	☐	☐	☐	☐	☐	☐

Tannhelse

97. **Sist du gikk til tannlege, gikk du til en tannlege/tannpleier i privat praksis eller til en tannlege/tannpleier ansatt i den offentlige tannhelsetjenesten?** (Sett kryss)

☐ Tannlege i privat praksis
☐ Tannlegespesialist i privat praksis
☐ Tannpleier i privat praksis
☐ Tannlege ansatt på offentlig tannklinikk
☐ Tannlegespesialist ansatt på offentlig tannklinikk
☐ Tannpleier ansatt på offentlig tannklinikk
☐ Tannlege i utlandet

98. **Når var du sist hos tannlege/tannpleier?** (Sett ett kryss)

☐ Mindre enn ett år siden ☐ 1-2 år siden
☐ 3-5 år siden ☐ Mer enn 5 år siden

99. **Hvis det er mer enn 2 år siden, hva er da grunnen?** (Sett ett kryss)

☐ Jeg har ikke blitt innkalt ☐ Det er lang ventetid hos tannlegen
☐ Jeg har ikke hatt tid ☐ Økonomiske årsaker
☐ Jeg har ikke hatt behov for tannbehandling ☐ Jeg er redd eller engstelig for å gå til tannlege
☐ Andre årsaker: _____

100. **Hvor mye har du betalt i alt for din egen tannbehandling (tannlege, spesialist og tannpleier) de siste 12 månedene?** (Sett ett kryss)

- ☐ Ingenting (har ikke vært hos tannlegen)
- ☐ Ingenting (har fått kostnadene dekket)
- ☐ Mindre enn 1000 kroner
- ☐ 1000-5000 kroner
- ☐ 5001-10.000 kroner
- ☐ 10.001-20.000 kroner
- ☐ Over 20.000 kroner

101. **Sett kryss for de to viktigste forhold med tennene for deg personlig?**

- At tennene er pene når jeg snakker og smiler ☐
- At tennene er smertefrie ☐
- At jeg kan tygge uten problemer ☐
- At min pust er god ☐
- At jeg har mine tenner resten av livet ☐

102. **Hvordan vurderer du tannhelsen din?** (Sett ett kryss)

☐ Dårlig ☐ Ikke helt god ☐ God ☐ Svært god

103. **Har du tannprotese/gebiss/tannbro?** ☐ Ja ☐ Nei

Soling

104. **Har du vært i syden eller på annen solferie i løpet av den siste måneden?** ☐ Ja ☐ Nei

105. **Hvor mye har du vært ute i dagslys i løpet av de siste 7 dagene?** ☐☐ timer

106. **Har du vært i solarium i løpet av den siste måneden?**

☐ Nei ☐ 1 - 2 ganger ☐ 3+ ganger

Hudpleiemidler

107. **Hvor ofte (antall ganger) bruker du følgende hudpleiemidler?** (Sett ett kryss pr. linje)

	aldri/ sjelden	1–3 pr. mnd.	1 pr. uke	2–4 pr. uke	5–6 pr. uke	1 pr. dag	2+ pr. dag
Ansiktskrem	☐	☐	☐	☐	☐	☐	☐
Håndkrem	☐	☐	☐	☐	☐	☐	☐
Bodylotion	☐	☐	☐	☐	☐	☐	☐
Parfyme/aftershave	☐	☐	☐	☐	☐	☐	☐
Deodorant	☐	☐	☐	☐	☐	☐	☐
Hårprodukt *(utenom shampo/ balsam)*	☐	☐	☐	☐	☐	☐	☐

Egne barn og amming

108. **Hvis du er kvinne og har født barn, kan du angi fødselsår på barna og ca. hvor mange måneder du ammet hvert av disse barna?**

	Fødselsår	Antall mnd. barnet ble ammet	Ikke ammet
Barn nr. 1	☐☐☐☐	☐☐	☐
Barn nr. 2	☐☐☐☐	☐☐	☐
Barn nr. 3	☐☐☐☐	☐☐	☐
Barn nr. 4	☐☐☐☐	☐☐	☐
Barn nr. 5	☐☐☐☐	☐☐	☐

Dersom flere barn, skriv på eget ark.

Familie og språkbakgrunn

109. **Hvordan var de økonomiske forhold i familien under din oppvekst?** (Sett ett kryss)

☐ Meget gode ☐ Gode ☐ Vanskelige ☐ Meget vanskelige

I Nord-Norge bor det folk med ulik etnisk bakgrunn. Det vil si at de snakker ulike språk og har forskjellige kulturer. Eksempler på etnisk bakgrunn, eller etnisk gruppe er norsk, samisk og kvensk.

110. **Hvilket hjemmespråk har/hadde du, dine foreldre og besteforeldre?** (Sett ett eller flere kryss pr. linje)

	Norsk	Samisk	Kvensk	Annet, beskriv:
Morfar	☐	☐	☐	☐
Mormor	☐	☐	☐	☐
Farfar	☐	☐	☐	☐
Farmor	☐	☐	☐	☐
Far	☐	☐	☐	☐
Mor	☐	☐	☐	☐
Jeg selv	☐	☐	☐	☐

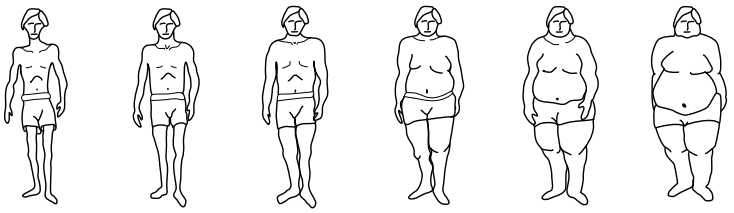
111. **Hva er din, din fars og din mors etniske bakgrunn?** (Sett ett eller flere kryss pr. linje)

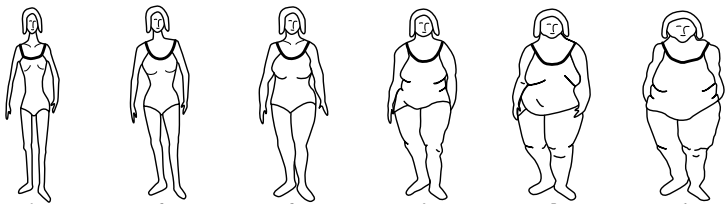
	Norsk	Samisk	Kvensk	Annet, beskriv:
Min etniske bakgrunn er	☐	☐	☐	☐
Min fars etniske bakgrunn er	☐	☐	☐	☐
Min mors etniske bakgrunn er	☐	☐	☐	☐

112. **Hva regner du deg selv som?** (Sett ett eller flere kryss)

Norsk ☐ Samisk ☐ Kvensk ☐ Annet, beskriv: ☐

Kroppsfigur





Figur nr.

113. **Hvilken figur ligner mest på deg?** .....

	Figur nr. mann	Figur nr. kvinne
114. **Hvilken figur tilsvarer en kropp som du synes ser mest sunn ut?**		
115. **Hvilken figur er den første i stigende rekkefølge som du oppfatter som tykk?**		
116. **Hvilken figur er den første i synkende rekkefølge som du oppfatter som tynn?**		

117. **Hva oppfatter du deg selv som?** (Sett ett kryss)

Alt for tykk	For tykk	Passe	For tynn	Alt for tynn
☐	☐	☐	☐	☐

118. **Har du forsøkt å gå ned i vekt/slanket deg de siste 6 måneder?** ..... ☐ Ja ☐ Nei

119. **Hvis ja, hvor mange kilo har du gått ned de siste 6 måneder?** ..... ☐☐ Kg

120. **Hvilke metoder brukte du for å gå ned?**
(Sett ett eller flere kryss)

☐ Spiste mindre ☐ Spiste sunnere ☐ Andre kostendringer
☐ Mosjon ☐ Slankemidler ordinert fra lege ☐ Slankepulver
☐ Annet, beskriv: .....

Andre ubehag

121. **Under finner du en oppstilling av plager som man av og til har. Les nøye gjennom dem, en for en, og angi deretter hvor mye hvert enkelt problem har plaget deg eller vært til besvær i løpet av de siste 4 ukene?** (Sett ett kryss for hver plage)

	Ikke plaget	Litt plaget	Ganske mye	Veldig mye
Nervøsitet, indre uro	☐	☐	☐	☐
Stadig redd eller engstelig	☐	☐	☐	☐
Følelse av håpløshet med tanke på fremtiden	☐	☐	☐	☐
Mye bekymring eller urolig	☐	☐	☐	☐
Nedtrykt, tungsindig	☐	☐	☐	☐

Søvn

Vi vil gjerne stille deg noen spørsmål om dine søvnvaner. Vær oppmerksom på at klokkeslettene må angis i 24 t., det vil si at 11:00, er elleve på formiddagen, og 23:00, er elleve om kvelden.

122. **Har du hatt skiftarbeid (natt- og eller kveldsarbeid) de siste tre månedene?** ..... ☐ Ja ☐ Nei

123. **Hvor mange dager i uken har du ikke anledning til å velge fritt når du vil sove og når du vil stå opp?** (Kan f.eks. gjelde for dager hvor du skal på arbeid, skole etc.) (Sett ett kryss)

0	1	2	3	4	5	6	7
☐	☐	☐	☐	☐	☐	☐	☐

124. **På dager jeg ikke har anledning til å velge fritt når jeg vil sove/stå opp:**

	Time	Minutt
Da går jeg til sengs klokken	☐☐	☐☐
Jeg gjør meg klar til å sovne klokken	☐☐	☐☐
Antall minutter det vanligvis tar før jeg sovner helt ..		☐☐
Jeg våkner klokken	☐☐	☐☐

Jeg våkner ved hjelp av:
☐ Vekkerklokke ☐ Ytre påvirkning (f.eks. støy fra familie eller andre) ☐ Av meg selv

Antall minutter det tar før jeg vanligvis står opp ..... ☐☐

Sover du i tillegg på slike dager også på andre tider av døgnet? (f.eks. middagshvil) ..... ☐ Ja ☐ Nei

	Time	Minutt
Når (kl.) skjer det vanligvis	☐☐	☐☐
Antall minutter du da sover		☐☐

125. **Når jeg fritt kan sove/stå opp:**

	Time	Minutt
Da går jeg til sengs klokken	☐☐	☐☐
Jeg gjør meg klar til å sovne klokken	☐☐	☐☐
Antall minutter det vanligvis tar før jeg sovner helt		☐☐
Jeg våkner klokken	☐☐	☐☐

Jeg våkner ved hjelp av:
☐ Vekkerklokke ☐ Ytre påvirkning (f.eks. støy fra familie eller andre) ☐ Av meg selv

Antall minutter det tar før jeg vanligvis står opp ..... ☐☐

Sover du i tillegg på slike dager også på andre tider av døgnet? (f.eks. middagshvil) ..... ☐ Ja ☐ Nei

Takk for at du deltok i undersøkelsen!

LUNDBLAD MEDIA AS – SVANEGODKJENT TRYKKSAK – 241 762 | 0-110901 – NORSK

Helse- og livsstils-undersøkelse

Vi ber deg fylle ut spørreskjema så nøye som mulig og levere det ved oppmøte til den innkalte helseundersøkelsen.
Skjema skal leses optisk. Vennligst bruk blå eller sort penn.
Bruk blokkbokstaver. Du kan ikke bruke komma, forhøy for eksempel 0,5 til 1.

1. **I hvilket år er du født?** ..... Årstall

2. **Er du?** ..... Kvinne ☐ Mann ☐

3. **Hva er din sivilstatus?**
☐ Gift ☐ Samboer ☐ Skilt
☐ Ugift ☐ Enke/enkemann

4. **Hvor mange års skolegang har du gjennomført?** (Ta med alle år du har gått på skole eller studert) ..... Antall år

5. **Hvis du er kvinne: Hvor mange barn har du født?** ..... Antall

6. **Hvis du er kvinne: Hvor mange barn har du ammet?** ..... Antall

Egen helse

7. **Hvordan er helsen din?** (Sett ett kryss)
☐ Dårlig ☐ God
☐ Ikke helt god ☐ Svært god

8. **Hvordan vurderer du tannhelsen din?** (Sett ett kryss)
☐ Dårlig ☐ God
☐ Ikke helt god ☐ Svært god

9. **Har du tannprotese/gebiss/tannbro?** ..... ☐ Ja ☐ Nei

10. **Når var du sist hos tannlege eller tannpleier?**
☐ Mindre enn ett år siden ☐ 1–2 år siden
☐ 3–5 år siden ☐ Mer enn 5 år siden

11. **Hvor fornøyd er du med tannhelsetjenesten i din kommune?** (Sett ett kryss)
Svært misfornøyd ☐ ☐ ☐ ☐ ☐ ☐ Svært fornøyd ☐ Vet ikke

Hjerte-karsykdommer

12. **Har du eller har du hatt høyt blodtrykk?** ..... ☐ Ja ☐ Nei

13. **Hvis ja, hvor gammel var du da du fikk høyt blodtrykk?** ..... Alder

14. **Bruker du medisin mot høyt blodtrykk?** ..... Ja, nå ☐ Før, men ikke nå ☐ Aldri ☐

15. **Hvis du bruker eller tidligere har brukt blodtrykksmedisin, omtrent hvor gammel var du første gang du begynte med slik medisin?** ..... Alder

16. **Har du hatt hjerteinfarkt?**
☐ Nei, aldri ☐ 1 gang ☐ 2 ganger ☐ 3 eller flere

17. **Hvis ja, hvor gammel var du første gang du fikk hjerteinfarkt?** ..... Alder

18. **Har du angina pectoris (hjertekrampe)?** ..... ☐ Ja ☐ Nei

19. **Hvis ja, hvor ofte har du merket slike smerter i løpet av den siste måneden?** .....
Sjelden ☐ 1 gang pr. uke ☐ 2-3 ganger pr. uke ☐ 4-6 ganger pr. uke ☐ 7 eller flere ganger pr. uke ☐

20. **Hvor gammel var du første gang du fikk angina pectoris?** ..... Alder

21. **Har du blitt hjerteoperert (bypass)?** ..... ☐ Ja ☐ Nei

22. **Har du blitt blokket/fått innsatt stent?** ..... ☐ Ja ☐ Nei

23. **Har legen sagt at du har hjerteflimmer?** ..... ☐ Ja ☐ Nei

24. **Hvor gammel var du første gang du fikk hjerteflimmer?** ..... Alder

Diabetes (sukkersyke)

25. **Har du noen gang fått påvist diabetes (for høyt blodsukker)?** ☐ Ja ☐ Nei

Dersom nei, gå videre til spørsmål 35.

26. **Dersom ja, hvilken type diabetes har du fått påvist?** (Sett ett eller flere kryss)

Svangerskapsdiabetes ☐

Diabetes type I ☐

Diabetes type II ☐

27. **Hvordan ble din diabetes oppdaget?**

Jeg søkte lege pga. symptomer ☐ Ja ☐ Nei

Ble oppdaget uten at jeg hadde symptomer (legeattest, bedriftskontroll, svangerskapskontroll, undersøkelse for annen sykdom e.l.) ☐ Ja ☐ Nei

28. **Hvor gammel var du da din diabetes ble oppdaget?** Alder ☐☐

INSULIN

29. **Bruker du insulin mot din diabetes?**	Ja, nå	Før, men ikke nå	Aldri brukt
	☐	☐	☐

Dersom du bruker eller har brukt insulin:

30. **Hvor gammel var du da du begynte med insulin?** Alder ☐☐

31. **Hvor mange ganger pr. dag tar du/tok du vanligvis insulin?** ☐☐ ganger

32. **Hvor mange enheter insulin tar du/tok du vanligvis til sammen pr. dag?** ☐☐ enheter(E)

TABLETTER

33. **Bruker du tabletter mot din diabetes?**	Ja, nå	Før, men ikke nå	Aldri brukt
	☐	☐	☐

Dersom du bruker eller har brukt tabletter:

34. **Hvor gammel var du da du begynte med tabletter mot diabetes?** Alder ☐☐

Andre sykdommer

35. **Har du eller har du noen gang hatt?**

	Ja	Nei	Alder første gangen
Astma	☐	☐	☐☐
Eksem	☐	☐	☐☐
Kronisk bronkitt, emfysem, KOLS	☐	☐	☐☐
Multippel sklerose (MS)	☐	☐	☐☐
Psoriasis	☐	☐	☐☐
Bechterews sykdom	☐	☐	☐☐

Smerter

36. **Har du smerter nå som har vart i tre måneder eller lengre?** ☐ Ja ☐ Nei

37. **Hvis ja, vennligst angi hvor sterke smerter du har hatt den siste uken:** (Sett ett kryss)

Ingen smerte										Verste tenkelige smerter
0	1	2	3	4	5	6	7	8	9	10
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

38. **Angi hvor smertene er mest plagsomme:**
(Sett ett kryss)

☐ Nakke ☐ Korsrygg ☐ Annet

Fysisk aktivitet

39. **Vi ber deg angi din fysiske aktivitet etter en skala fra svært lite til svært mye da du var 14 år, 30 år og i dag.** Skalaen nedenfor går fra 1-10. Med fysisk aktivitet mener vi både arbeid i hjemmet og i yrkeslivet, samt trening og annen fysisk aktivitet som turgåing o.l. Sett kryss under det tallet som best angir ditt nivå av fysisk aktivitet.

	Svært lite									Svært mye
Alder	1	2	3	4	5	6	7	8	9	10
14 år	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
30 år	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
I dag	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Alkohol

40. **Er du totalavholdskvinne/mann?** ☐ Ja ☐ Nei

41. **Hvis nei, hvor ofte og hvor mye drakk du i gjennomsnitt siste året?** (Sett ett kryss for hver linje)

	aldri/ sjelden	1 pr. mnd.	2–3 pr. mnd.	1 pr. uke	2–4 pr. uke	5–6 pr. uke	1 pr. dag	2+ pr. dag
Øl/rusbrus *(½ l.)*	☐	☐	☐	☐	☐	☐	☐	☐
Vin *(glass)*	☐	☐	☐	☐	☐	☐	☐	☐
Brennevin *(drink/shot)*	☐	☐	☐	☐	☐	☐	☐	☐
Likør/hetvin *(glass)*	☐	☐	☐	☐	☐	☐	☐	☐

Røykevaner

42. **Har du noen gang røykt daglig?** ☐ Ja ☐ Nei

Dersom du **aldri har røykt daglig**, kan du gå videre til spørsmål 47.

43. **Røyker du daglig nå?** ☐ Ja ☐ Nei

44. **Hvis du har sluttet å røyke daglig, hvor gammel var du da du sluttet?** Alder ☐☐

45. **Hvor mange år til sammen har du røykt daglig?** År ☐☐

46. **Hvor mange sigaretter/rulletobakk har du i gjennomsnitt røykt daglig i de årene du har røkt daglig?** Antall ☐☐

47. **Bor du sammen med noen som røyker?** ☐ Ja ☐ Nei

Språk og bruk av tolk

48. **Hvilket språk ønsker du først og fremst å snakke med helsepersonell på?** (Sett ett eller flere kryss)

Norsk ☐ Samisk ☐ Annet, beskriv: ☐ .....

49. **Hvis du har svart «samisk», men ikke fikk tilbud om samisktalende lege ved siste legebesøk, ble det da tilbudt tolk?**

Hos fastlegen

☐ Ja ☐ Nei
☐ Ønsker ikke bruke tolk ☐ Ikke aktuelt

På sykehus/ hos spesialist

☐ Ja ☐ Nei
☐ Ønsker ikke bruke tolk ☐ Ikke aktuelt

Familie og språkbakgrunn

50. **Hvordan var de økonomiske forhold i familien under din oppvekst?** (Sett ett kryss)

☐ Meget gode ☐ Gode ☐ Vanskelige ☐ Meget vanskelige

I Nord-Norge bor det folk med ulik etnisk bakgrunn. Det vil si at de snakker ulike språk og har forskjellige kulturer. Eksempler på etnisk bakgrunn, eller etnisk gruppe er norsk, samisk og kvensk.

51. **Hvilket hjemmespråk har/hadde du, dine foreldre og besteforeldre?** (Sett ett eller flere kryss)

	Norsk	Samisk	Kvensk	Annet, beskriv:
Morfar.....	☐	☐	☐	☐ .....
Mormor.....	☐	☐	☐	☐ .....
Farfar.....	☐	☐	☐	☐ .....
Farmor.....	☐	☐	☐	☐ .....
Far.....	☐	☐	☐	☐ .....
Mor.....	☐	☐	☐	☐ .....
Jeg selv.....	☐	☐	☐	☐ .....

52. **Hva er din, din fars og din mors etniske bakgrunn?** (Sett ett eller flere kryss)

	Norsk	Samisk	Kvensk	Annet,beskriv:
Min bakgrunn:.....	☐	☐	☐	☐ .....
Min fars bakgrunn:.....	☐	☐	☐	☐ .....
Min mors bakgrunn:.....	☐	☐	☐	☐ .....

53. **Hva regner du deg selv som?** (Sett ett eller flere kryss)

Norsk ☐ Samisk ☐ Kvensk ☐ Annet, beskriv: ☐ .....

Erfaringer og bruk av helsetjenester

54. Den legen du vanligvis bruker er:

☐ Din fastlege ☐ Annen lege

55. Hvor lenge har du hatt din nåværende fastlege?

☐ Mindre enn 6 mnd ☐ 6 til 11 måneder

☐ 12 til 24 mnd ☐ Mer enn 2 år

56. Har du i løpet av de siste 12 mnd kontaktet fastlegen din for hjelp eller råd til deg selv? ☐ Ja ☐ Nei

Hvis ja, opplevde du at du fikk den hjelpen du ba om?

☐ Aldri ☐ Av og til ☐ Vanligvis ☐ Alltid

57. Hvor fornøyd eller misfornøyd er du med følgende sider ved fastlegetjenesten? (Sett ett kryss)

	Meget fornøyd	For-nøyd	Misfor-nøyd	Meget misfor-nøyd	Vet ikke
Fastlegens tilgjengelighet på telefon	☐	☐	☐	☐	☐
Ventetid for å få time hos fastlege	☐	☐	☐	☐	☐
Tid hos fastlegen	☐	☐	☐	☐	☐
Fastlegens forståelse for dine problem	☐	☐	☐	☐	☐
Fastlegens informasjon om dine helseplager, undersøkelse og behandlingsopplegg.	☐	☐	☐	☐	☐
Totalt sett, hvor fornøyd eller misfornøyd er du med den kommunale helsetjenesten?	☐	☐	☐	☐	☐

De neste spørsmålene omhandler spesialisthelsetjenesten.

Med spesialisthelsetjenesten menes det sykehus, distriktspsykiatrisk senter (DPS), spesialistlegesenter eller enkeltspesialist.

58. Har du i løpet av de siste 12 måneder vært til undersøkelse eller behandling for *fysiske plager* hos:

☐ Sykehus ☐ Spesialistlegesenter

☐ Privatpraktiserende spesialist ☐ Ingen av delene

59. Har du i løpet av de siste 12 måneder vært til undersøkelse eller behandling for *psykiske plager* hos:

☐ Psykiatrisk sykehus ☐ Distriktspsykiatrisk senter

☐ Privatpraktiserende spesialist ☐ Ingen av delene

60. Dersom du har vært til behandling hos spesialist for fysiske eller psykiske plager, svar på følgende spørsmål: (Sett ett kryss)

Svar på en skala fra 0 til 10 (0 = i liten grad 10 = i stor grad)

Fikk du anledning til å fortelle det du følte var viktig om din tilstand?

	0	1	2	3	4	5	6	7	8	9	10	Ikke aktuelt
For fysiske plager	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
For psykiske plager	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Snakket legene/behandlerne til deg slik at du forstod dem?

	0	1	2	3	4	5	6	7	8	9	10	Ikke aktuelt
For fysiske plager	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
For psykiske plager	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Alt i alt, har du tillit til sykehuset eller spesialisten du var hos?

	0	1	2	3	4	5	6	7	8	9	10	Ikke aktuelt
For fysiske plager	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
For psykiske plager	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Alt i alt, hvor tilfreds er du med pleien og behandlingen du eventuelt fikk?

	0	1	2	3	4	5	6	7	8	9	10	Ikke aktuelt
For fysiske plager	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
For psykiske plager	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Takk for at du deltok i undersøkelsen!