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Nutrient Intake and Retinopathy: The Tromsø Study

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Abstract

Introduction: Retinopathy is a retinal disorder characterized by microvascular complications due to ocular or systemic conditions. The non-invasive visualization of retinal blood vessels provides a way to study the early structural and pathological changes in the circulatory system. Hence retinopathies can be used to monitor risk factors for cardiovascular diseases. Several studies have explored the association between nutrients and retinopathies in specific groups of people, however, there is limited evidence regarding their association in the general population.

Objectives: This study was to investigate the association between the intake of various nutrients and retinopathy in a general Norwegian adult population.

Method: The cross-sectional study utilized data from the seventh Tromsø survey (2015-2016) to investigate the association between macronutrients and retinopathy. The final sample consisted of 4,724 participants. Descriptive statistics, including percentages, mean, and standard deviation, were calculated and presented based on retinopathy status. Chi-square tests and t-tests were conducted to assess differences between groups. Odds ratios (ORs) and confidence intervals (CIs) from binary logistic regression analysis were used to examine the association between macronutrients and retinopathy. All analyses were stratified by diabetes using the statistical software STATA.

Results: Findings from this study revealed an association only in polyunsaturated fat (PUFA) and monounsaturated fat (MUFA). In the fully adjusted model, intake of MUFA (OR 0.96 (95% CI 0.92-0.99)) and PUFA (OR 0.9 (95% CI 0.85-0.96)) were associated with decreased odds of retinopathy. However, this association was only significant in the group without diabetes. No significant association between the intake of any other macronutrient and retinopathy was found in the studied population.

Conclusion: Based on the current findings, no significant association was observed between macronutrients and retinopathy except in MUFA and PUFA. Further studies are required to investigate this association more comprehensively and prospectively.

Table of Contents

List of Tables

List of Figures

Supplementary materials

List of Appendix

Abbreviations

AGE	Advanced glycation end product
BMI	Body Mass Index
CVD	Cardiovascular disease
DAG	Directed Acyclic Graph
DM	Diabetes mellitus
DP	Dietary Patterns
DR	Diabetic retinopathy
FFQ	Food frequency questionnaire
GI	Glycaemic index
MUFA	Monounsaturated fat
NNR	Nordic Nutrition Recommendations
OR	Odds Ratio
PUFA	Polyunsaturated fat
RCT	Randomized Clinical Trials
ROP	Retinopathy of prematurity
SES	Socioeconomic status
Tromsø6	Sixth survey of the Tromsø study
Tromsø7	Seventh survey of the Tromsø study
WHO	World Health Organisation

1 Introduction

Retinopathy is a complex retinal disorder characterized by microvascular complications from ocular and systemic conditions, which can ultimately lead to vision loss (1). The human retina is supplied by a rich network of micro vessels. Thus, changes in the circulatory system and other systemic conditions such as Diabetes mellitus (DM) can be visualised non-invasively via the retina (2). Although there are cases of congenital retinopathies (3), retinopathy is predominant in the adults (4) and this study focuses on the adult population. The prevalence of retinopathy varies according to the type of retinopathy. According to World Health Organisation (WHO), diabetic retinopathy (DR), which is the most common type of retinopathy has an estimated global prevalence of about 93 million (5). In Norway, previous studies have reported the prevalence of DR to be between 11% and 28% (6, 7, 8, 9). Other forms of retinopathy, such as hypertensive retinopathy, has a prevalence below 10% (10, 11).

Studies have shown that the clinical signs of retinopathy can predict cardiovascular diseases (CVDs) and in some cases it is the earliest observed sign CVD (12, 13, 14, 15, 16) . In reviewing the evidence available on early life factors related with CVDs, such as diet (17, 18, 19), retinal vascular imaging is widely recognized as a valuable technique. In a previous study, retinal vascular diameter was found to be positively associated with diets high in fibre, seafood and low-fat dairy items (20). Nutrients which are found in diets can influence retinopathy (21) however, the association is dependent on the specific type and the magnitude of intake (21, 22, 23, 24). Majority of previous studies have investigated the association between macronutrients and DR with very few studies on other types of retinopathies. Given that retinopathies have been found in the absence of diabetes (6, 10, 25, 26, 27), it is crucial to study this association in the general population. So, this study investigates the association between macronutrients and retinopathy in the general population, encompassing individuals with and without diabetes.

1.1 Retinopathy

Retinopathy involves the pathological changes to the retinal blood vessels which are associated with transient and persistent microvascular damage from aging and systemic conditions such as diabetes (28). The retina, which is the innermost tissue of the human eye, includes various neurons that are responsible for vision production. These neurons (photoreceptors, ganglion cells, bipolar cells, amacrine cells and horizontal cells) are interconnected enabling the processing of visual information projected on the retina and then transmitted to the brain via the optic nerve (29). Given its complex cellular structure, the retina needs a specialized vascular

circulation to meet its metabolic needs without interfering with its neurotransmission, phototransduction, as well as metabolite interaction (30). The retina is supplied by the central retinal artery (the first branch of the ophthalmic artery) and the short posterior ciliary arteries. The central retinal artery runs in the centre of the optic nerve and emerges at the optic disc dividing into two main branches, which further divides into arterioles that supply the quadrants of the retina. The retina is drained by the vortex veins of the choroid and central retinal vein which leaves the eye through the optic nerve into the cavernous sinus (31).

Abnormalities in the retina includes cotton wool spots, exudates, micro aneurysms, haemorrhages, and changes in blood vessel structure. They may indicate retinal vascular dysfunction, systemic and ocular diseases, and disturbances of systemic circulation (30). In such cases, the retina may serve as a site for the manifestation of systemic diseases such as hypertension, diabetes, and sickle cell anaemia amongst others.

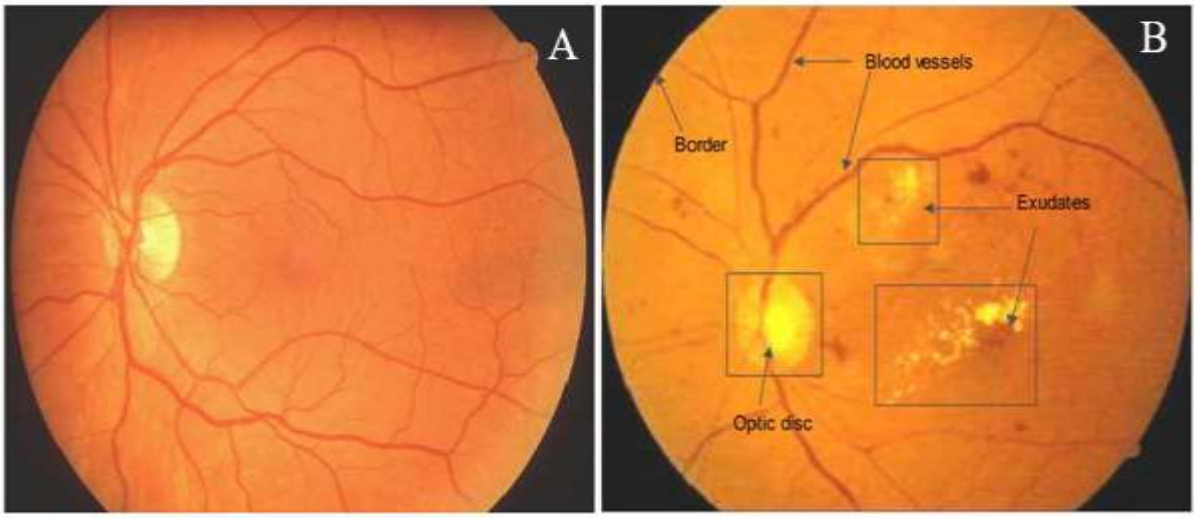


Figure 1: Image of a normal retina (A) and retina with retinopathy (B) (32)

1.2 Types of retinopathy

Several types of retinopathy arise from different causes, usually systemic diseases, that are connected to the vascular system of the body while other types of retinopathy are of ocular origins (33). Depending on the cause of the retinopathy, the clinical presentation may vary as each type of retinopathy has its own pathophysiology, course, and treatment. These types of retinopathies are discussed below, in three broad groups:

Diabetic retinopathy

This is the most renowned type of retinopathy that is caused by DM. The principal etiologic factor causing all the microvascular complications of diabetes, including DR, is chronic hyperglycaemia in people with diabetes. There are several possible biochemical mechanisms involved in the pathogenesis of DR such as aldose reductase, advanced glycation end product (AGE), protein kinase, photoreceptor metabolism, insulin receptor and glucose transporters (34). In addition to being a microvascular disease, neurodegeneration of the retina also takes place together with pericyte loss (35, 36). It is the most prevalent microvascular complication of DM and the leading cause of acquired vision loss in people aged 20 to 65 years worldwide (37, 38). Globally, DR is found in one third of those with diabetes and about 1 in 10 of these cases are visual-threatening levels of DR (39, 40, 41, 42).

Retinopathy without diabetes

Several retinopathies exist that are not caused by diabetes. The features of retinopathy and structural changes in the blood vessels are often seen in people without diabetes (25). The prevalence of retinopathy in people without diabetes ranges from 1% to 15% (10, 11, 43). Many of these retinopathies are related to the circulatory system of the body while some others are due to various conditions such as drug toxicity. Examples of these retinopathies are hypertensive retinopathy, drug toxicity induced retinopathy, HIV Associated retinopathy, Sickle cell retinopathy, cancer related retinopathy (44, 45).

Congenital retinopathies

These occur in the early stages of life. In some cases, infants are born with it as a result of poorly formed retinal blood vessels. A common aspect of this is retinopathy of prematurity (ROP); a proliferative vitreoretinopathy that affects premature infants and is one of the main causes of juvenile blindness globally (3). It causes irreversible vision loss in about 32,300 new-borns globally, of whom about 20,000 go blind or have severe visual impairment (46). Other congenital retinopathies are rubella retinopathy and coats disease.

However, for the purpose of this study, focus is on the retinopathies that occur later in life among working age adults. The aetiology of a retinopathy could be known and linked to one or more underlying causes (33). Notwithstanding, retinopathy could be a preclinical indication for

diabetes or hypertension, hence, it may be too early to determine its source, (47) or it could be idiopathic in which case the cause is unknown (48).

1.3 Risk factors of retinopathy

Age and sex

One of the well-established risk factors of retinopathy is age (4). Aging is linked to deterioration in various physiological processes that affect the circulatory system (49). Age is an independent risk factor for retinopathy (5, 50, 51), and as people age, they are exposed to additional risk factors for retinopathy over longer periods of time (52). Studies have found gender differences in the risk of retinopathy, where there is increased risk in men compared to women (53, 54, 55). However, there are reported studies of no gender difference in the risk of retinopathy (56, 57).

Lifestyle factors

Several lifestyle factors are associated with retinopathy. The lack of physical activity is linked to an increased risk of retinopathy (58). A diet rich in fruits, vegetables, and omega-3 fatty acids could lower risk the of retinopathy compared to a diet high in saturated fats, processed foods and sugar (59, 60). Retinopathy risk is higher in people who drink a lot of alcohol, according to research (61). Sleep disturbances, such as sleep apnoea (62, 63) and poor sleep quality (64, 65), may also increase the risk of retinopathy.

Socioeconomic status (SES)

Low SES increases the risk of the retinopathy (53). They are linked to some other retinopathy risk factors, including as lifestyle factors. Those who have low SES are less likely to have eye exams (66, 67), which are crucial for retinopathy care and screening. As a result, retinopathy may take longer to diagnose and properly manage. Other SES such as education and employment are also risk factors of retinopathy (68).

Cardiovascular risk factors

Cardiovascular risk factors are significant in the development of retinopathy especially in those without diabetes (10, 69, 70). Retinal ischemia, vascular leakage, and retinal oedema can all result from damaged blood vessels in the retina caused by increase in the blood pressure. (10, 27). Hypertension has a higher prevalent among those have retinopathy without diabetes compared to those who have diabetes (9, 71).

Diabetic risk factors

In the case of DR, there are specific risk factors such as metabolic control, duration of diabetes, cholesterol level, control of blood glucose level (72, 73, 74, 75). Early diagnosis and proper management reduce the risk of developing retinopathy in those with diabetes (76, 77). In other to increase early diagnosis and proper management, various national guidelines develop screening programmes for DR (78, 79, 80)**.** Other risk factors such as race/ethnicity medications/treatment plans (44, 81) are also crucial.

1.4 Symptoms and clinical features of retinopathy

During the initial phases of retinopathy, it frequently presents as asymptomatic, and individuals with retinopathy could continue their daily activities without perceiving any alterations in their vision (82). However, as the retinopathy progresses, the individual may experience significant changes to the vision. Such as poor vision, floaters (small specks, dots, or cobweb-like shapes in the field of vision), impaired colour vision and visual field disturbances to the peripheral and/or central vision (28, 82, 83). Severe cases of retinopathy and lack of proper management over an extended period can eventually lead to vision loss (84).

Retinopathy is characterized by several clinical features, such as microaneurysms or haemorrhages, cotton-wool spots, hard exudates, neovascularisation, and venous diameter abnormalities, including venous loops, venous tortuosity, and venous beading. Microaneurysms refer to sac-like protrusions of capillary walls that may cause fluid leakage, haemorrhages and intraretinal oedema (83, 85). The haemorrhages can take the form of either flame-shaped or dot-blot-like patterns depending on the retinal layer where they occur. Flame-shaped haemorrhages appear in the inner retina closer to the vitreous (nerve fibre layer), while dot-blot haemorrhages are deeper in the retina (86). Neovascularisation is an abnormal growth of new vessel within the retinal tissue or shunt vessels created through areas with insufficient vascular perfusion (87). Cotton-wool spots are microinfarcts in the nerve fibre layer of the retina. Regarding changes to the size, venous diameter abnormalities usually indicate severe retinal hypoxia, although in some cases, the retina may appear featureless due to extensive vascular loss (88).

Vision loss in retinopathy is commonly attributed to several factors. These include persistent vitreous haemorrhage, tractional retinal detachment, and diabetic macular oedema (89). When neovascularization occurs, the growth of fibrous tissue can distort the retina and lead to

tractional retinal detachment. Additionally, the newly formed blood vessels may bleed, resulting in either preretinal or vitreous haemorrhage. In the case of diabetic macular disease, vision loss can be attributed to macular oedema affecting the fovea or nonperfusion of capillaries in the central macula (90, 91).

1.5 Grading of retinopathy

The clinical features described above can be accessed by analysing images from diagnostic techniques to visualize the retina and its structures. An example is the retinal photography. It is a non-invasive diagnostic procedure that uses a specialized camera to capture detailed images of the retina (92, 93). The images captured through retinal photography provide valuable information about the structure and health of the retina and can be used to diagnose and monitor various ocular pathologies, including DR, age-related macular degeneration, and glaucoma (94) as well as monitor cardiovascular risk (95).

The grading of retinopathies is either based on their severity or on the location. A widely used grading system for DR is the International Clinical Diabetic Retinopathy and Diabetic Macular Oedema Severity Scales (ICDR) (96).

Table 1: International Clinical Diabetic Retinopathy and Diabetic Macular grading system for Diabetic Retinopathy (96)

Grade	Proposed disease severity level	Findings observable on dilated ophthalmoscopy
0	No apparent retinopathy	No abnormalities
1	Mild retinopathy	Microaneurysms only
2	Moderate retinopathy	More than just 20 microaneurysms, but less than severe retinopathy
3	Severe retinopathy	Any of the following -More than 20 intraretinal haemorrhages in each quadrant - Definite venous beading in 2+ quadrants -Prominent intraretinal microvascular abnormalities (IRMA) in 1+ quadrant
4	Proliferative retinopathy	Any of the following -Neovascularization -Vitreous haemorrhage -Preretinal haemorrhage

The ICDR scale uses the clinical features observed in the retina to classifies retinopathy into stages: No abnormalities (stage 0), Mild Non-Proliferative Retinopathy (Stage 1), Moderate

Non-Proliferative Retinopathy (Stage 2), Severe Non-Proliferative Retinopathy (Stage 3), and Proliferative Retinopathy (Stage 4).

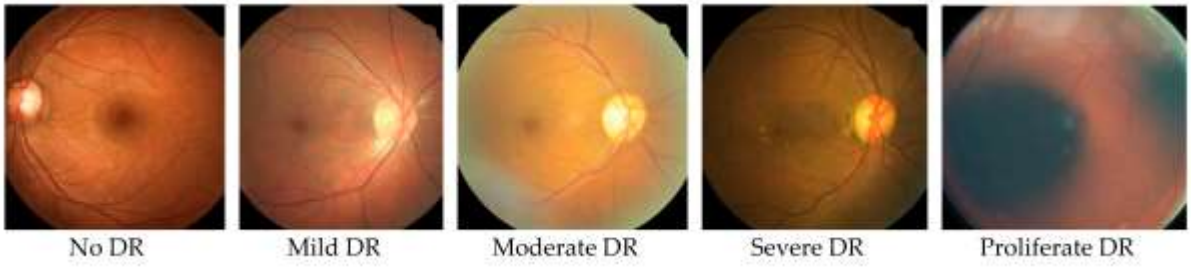


Figure 2: Stages of retinopathy based on the ICDR scale (97)

Other grading systems are the Early Treatment Diabetic Retinopathy Study (ETDRS) scale (98) for DR and Modified Scheie Classification of Hypertensive Retinopathy (99).

1.6 Retinal blood vessels and the central nervous system

The retinal blood vessels share similar anatomical and physiological traits with the cerebral and coronary circulations which is easily and noninvasively visible by retinal imaging (100). The retina, composed of interconnected specialized neurons, is considered part of the central nervous system (CNS). Light entering the eye is captured by photoreceptor cells, triggering neuronal signals that reach the retinal ganglion cells and form the optic nerve, which transmits visual information to the brain. The optic nerve, like other CNS fibre tracts, undergoes degeneration and creates a hostile environment upon injury, hindering axonal regeneration (101). The eye, including the retina, maintains regulated interactions with the immune system, resembling the immune-privileged nature of the CNS and possesses unique structures and barriers for immune defence.

1.7 Retinal blood vessels and cardiovascular system

Arteriolar damage from hypertension and other cardiovascular processes are reflected in retinal microvascular abnormalities such as generalised and localised arteriolar narrowing, arteriovenous nicking, and retinopathy (102). Structural changes in the retina blood vessels could act as predictor of CVD (12, 13, 14, 15, 16) as seen in previous studies. CVDs continue to be the world's leading cause of death as well as a major contributor to poor health and increased expenses for the healthcare system (103). Early studies of cardiovascular risk factors used retinal vascular imaging (104, 105) and some recent studies have also investigated their association with structural changes of the retinal blood vessels (106, 107). In a study of a Norwegian population, associations were found between retinal microvascular diameter and

cardiovascular risk factors such as blood pressure, cholesterol levels, body mass index (BMI) and smoking (108). Based on the evidence from a study on retinal imaging and early cardiovascular risk factor it was recommended that long term monitoring of cardiovascular risk factors and assessment of vascular changes should include retinal imaging (19).

Li & Wong (19), found that retinal vascular imaging can be used as a tool in the study of the early life factors associated with CVD. This review identified diet as a large contributor to the risk of CVD (19). Many studies including clinical trials have shown the association between dietary patterns and CVD (109, 110, 111).

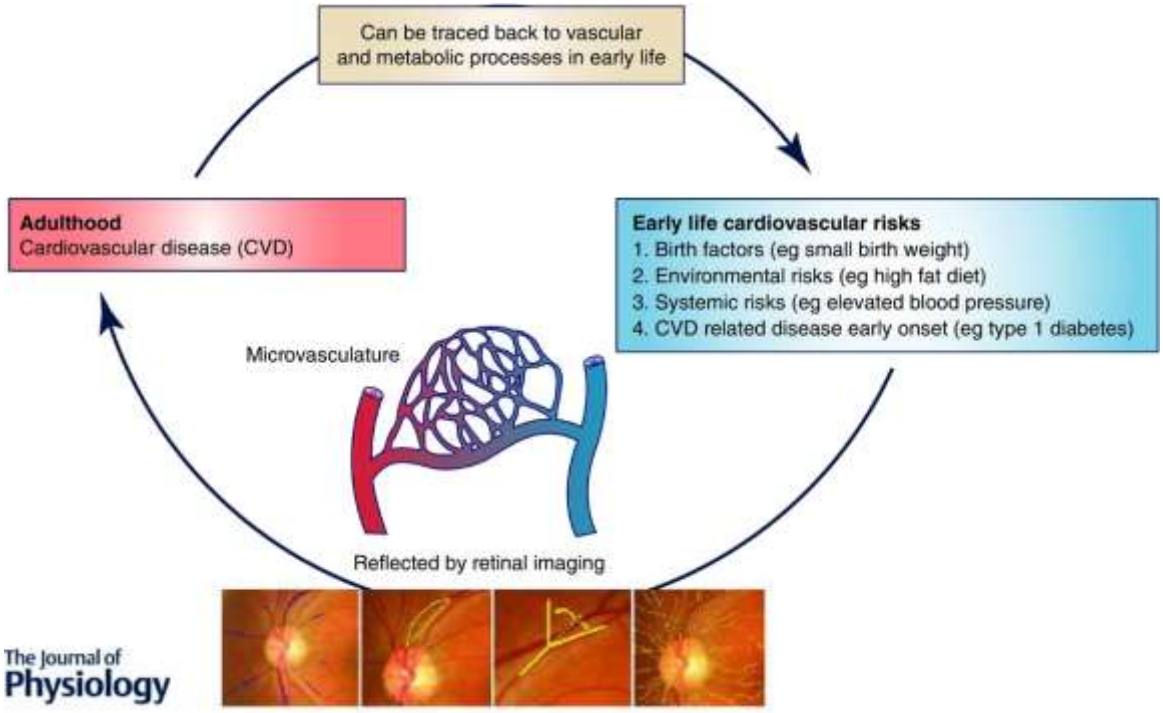


Figure 3: Early cardiovascular risk factors reflected by retinal imaging (112).

1.8 Dietary intake and retinopathy

There is growing evidence that the relationship between nutrition and clinical cardiovascular outcomes may, in part, be mediated by the microcirculation (113, 114, 115). It is crucial to investigate whether dietary intake influences retinal vascular changes. The association between diet and retinal vascular changes have been investigated in previous studies (116, 117, 118). In the Irish Nun Eye Study, a cross sectional study on dietary intake (grouped into healthy and unhealthy pattern) and retinal vessels abnormalities, unhealthy dietary pattern was associated with unfavourable retina profile (wider central retinal venular and narrower central retinal arteriolar) (116). Healthy pattern was characterised by high intake of fruit, wholegrains, vegetables, and oily fish while unhealthy pattern was sugar, chips, French fries, and high fat

dairy products. In a randomized clinical trial (RCT) comparing the risk of retinopathy in type II diabetes among an intervention group on Mediterranean diet (with olive oil or nuts) and a control group only on low diet, there was reduced risk in the intervention group (117). In a prospective cohort study, association between diet (based on the Australian Healthy eating Index) and retinal microvasculature, showed no association. Although, the authors suggests that overall diets should be examined in further studies instead of specific nutrients as they are not consumed independently (118).

Diet is a complex mixture of various nutrients, foods, and compounds; hence it is usually difficult to separate the effects of one dietary factor from another (119). Also, the variability of dietary intake over time makes it challenging to investigate diet, especially in cross-sectional studies. In comparison, nutrients such as fatty acids do not vary so much between individuals and can represent overall diet (120). Thus, this present study investigated specific associations between nutrients and retinopathy which could indicate if there are beneficial or harmful nutrients associated with retinopathy.

1.9 Nordic nutritional recommendations

In Norway, national nutritional recommendations are based on the Nordic Nutrition Recommendations (NNR) (121) and are designed for the general population to guide the composition of a healthy diet. The NNR covers the scientific basis for national dietary reference values and food-based dietary guidelines in Nordic countries (Norway, Sweden, Iceland, Finland, and Denmark) (122). There are five editions of the NNR, the last was published in 2012. NNR's sixth edition is expected to be published in June 2023 (122). This periodically updating is done to reflect the latest scientific evidence and advancements in nutrition research.

The NNR forms the basis for food and nutrition policies, providing a framework for policymakers to develop guidelines and regulations that promote healthy eating habits at a population level. It serves various purposes, including providing guidelines for dietary planning, helping individuals and healthcare professionals create balanced and nutritious meal plans. They also serve as a tool to assess dietary intake, allowing for comparisons between actual food consumption and recommended guidelines (123). The NNR 2012 established recommended intake ranges for macronutrients in levels that vary according to the available scientific evidence used to set the ranges.

1.10 Nutrients and retinopathy

Nutrients are chemical components needed by the body to provide energy, control body processes, promote growth, build, and repair body tissues to stay healthy and function effectively (124). The pathogenic processes mentioned above can be influenced by nutrients, which can impede or facilitate the development and progression of retinopathies (21).

A group of nutrients known as macronutrient play a significant role in maintaining optimal health. Consuming the appropriate balance of macronutrients is vital to ensure that the body receives the necessary energy and nutrients it requires to function correctly (125). Macronutrients are carbohydrate, dietary fibre, protein, alcohol, and fat, of which includes saturated fat, and unsaturated fat such as monounsaturated fat (MUFA), polyunsaturated fat (PUFA) and trans-fat (126). This thesis centres on the intake of macronutrients.

Carbohydrate and retinopathy

Carbohydrates, which include sugars, starches, and fibres, are a significant source of energy in the diet (127, 128). Carbohydrate intake, particularly high glycaemic index (GI) carbohydrates are associated with an increased risk of retinopathy (20). When blood glucose levels rise quickly due to high GI, oxidative stress, and inflammation occurs (129). In addition, GI have is connected to cardiovascular health (20). According to some studies (59, 130), the intake of carbohydrates is associated with DR. The body converts carbohydrates, especially those taken in the form of sucrose, into glucose and fructose. As a result of the fructose metabolism in the diabetic retina, lactate is created, which has a pathogenic role in the progression of retinopathy (21, 130). Furthermore, it has been demonstrated that consuming a lot of fructose increases the production of AGEs (131). In a cohort study of Japanese patients (132), carbohydrate intake was not associated with the incidence of DR. The study concluded that proportions of carbohydrate intake were not associated with the development of diabetes complications. Another study (133) found no significant association; however, this was a cross sectional study.

Dietary fibre and retinopathy

Dietary fibre is a form of carbohydrate that the body does not break down into glucose and is essential for the functioning of the digestive system (126). A diet rich in dietary fibre, particularly from whole grains, fruits, and vegetables, is beneficial (134). A high dietary fibre intake was associated with a decreased risk of retinopathy (60). The Diabetic Control and Complications Trial (DCCT) showed a negative correlation between dietary fibre intake and

DR (60). A population-based based cross-sectional study that examined the impact of dietary fibre intake on diabetes and diabetic microangiopathies found that a lower dietary fibre intake was associated with type II diabetes and the presence of retinopathy, sight-threatening DR, and microalbuminuria (135). Another study found significantly higher daily intakes of total carbohydrates, water-soluble dietary fibres, insoluble dietary fibres, and glucose among those without retinopathy compared to with retinopathy (59). This study was done only in a group of individuals with diabetes.

Protein and retinopathy

Protein is usually found in meals, such as meat, fish, eggs, beans, and nuts and is necessary for the development and repair of tissues (136). A crucial part of several physiological processes, including vision, is played by proteins (137). The retina, among other eye structures, depends on protein for its upkeep and development (138). According to research, the retina's homeostasis is maintained in large part by the way that ketones are metabolised. The ketone BHB, which is produced when lipids in the retinal pigment epithelium (RPE) break down, activates the GPR109A receptor. This causes the activation of signalling pathways downstream that improve mitochondrial function, lower oxidative stress, suppress proapoptotic factors, and stimulate the production of anti-inflammatory proteins, suggesting potential neuroprotective properties. (139, 140). It has been reported to improve glycaemic control and hyperglycaemia in people with diabetes and pre-diabetes (141). In a study by Roy et al., examining the impact of nutritional factors in those with diabetes with and without retinopathy, patients without retinopathy consumed a significantly lower proportion of their total daily calories from protein (59). Other studies (24, 60), did not find any association.

Fat and retinopathy

Lower total fatty acid intake is associated with DR, according to a previous study (60). In another study by Sasaki. et al (23), no association was found between fatty acid and retinopathy although this was a cross-sectional study. However, to better understand the association between fat and retinopathy the various types of fats (PUFA, MUFA, saturated fat and trans-fat) are discussed below. This is because studies have shown that these types of fat vary in their associations with retinopathy.

PUFA and retinopathy

PUFAs are also known as “good fats” (142) and can be found in fish with high levels of omega-3 and omega-6 fatty acids, as well as nuts and seeds (flaxseed oil, sunflower, safflower, and soybean oil) (143). Given that they have repeatedly been linked to a lower risk of developing CVDs and dyslipidaemia, PUFAs are one of the nutritional components that have positive impacts on health (144, 145). Research has shown that a high consumption of PUFA, particularly omega-3 fatty acids, may protect against retinopathy (60). They are also essential components of the retina and have been implicated in protecting against retinopathy and other retina vascular changes (146, 147). In a study by Houtsmuller et al., the effects of unsaturated fats on the progression of DR were observed over a 6-year period. One group followed a saturated-fat diet while the other group followed an unsaturated-fat diet rich in linoleic acid. The results suggest that a linoleic acid-rich diet, when administered over an extended period of time, may have an inhibitory effect on the development of microangiopathy and the deterioration of DR (148).

Another study has shown that among patients with well-controlled diabetes, increasing PUFA intake was associated with a reduced likelihood of the presence and severity of DR (23). These findings provide support for previous suggestions that PUFA may have a protective effect against DR (149). Not all PUFAs are the same, and varieties of PUFA might have various effects on retinopathy (150). Omega-3 fatty acids, for instance, have been demonstrated to have anti-inflammatory characteristics and may be advantageous for those with diabetes. Omega-3 fatty acids are often prescribed as a nutritional supplement to address visual field abnormalities with vascular origin (151). On the other hand, if ingested in excess, omega-6 fatty acids, might have pro-inflammatory qualities and raise the risk of vascular changes in the retina (152).

MUFA and retinopathy

MUFAs are regarded as beneficial and has been linked to a number of advantages, such as lowering the risk of heart disease and enhancing glycaemic management (153). Plant-based oils including olive oil, canola oil, and avocado oil are common sources of MUFAs (143). In a case-control study (133), comparing patients without DR and patients with DR, higher intake of MUFA and oleic acid was associated with a reduced frequency of DR, particularly in patients with a longer duration of diabetes. These findings suggest that incorporating MUFA-rich foods and sources of oleic acid in the diet may have a protective effect against the development of

DR in individuals with type II DM. In contrast a retrospective cohort study(60) found that MUFA is positively correlated to progression of retinopathy although the magnitude was small (13%).

Saturated fat and retinopathy

In addition to some plant-based sources like coconut oil, saturated fats are frequently found in animal products including meat, butter, and cheese (154). They are also referred to as "non-essential fats," because the body has the capacity to synthesise them when required (155). The association between retinopathy and unsaturated fat is still unclear although earlier studies by Cundiff and Nigg (60) have observed such associations. Nonetheless, a different study (23), showed that higher consumption of saturated fatty acids was associated with an increased risk of both the existence and severity of DR especially in people with well-controlled diabetes.

Trans-fat and retinopathy

Trans-fats can be found in foods derived from ruminant animals like cows and sheep, as well as in foods that contain partially hydrogenated vegetable oils (156). It has been shown (157, 158) to have adverse health effects. Very few studies have investigated the association between trans-fat and retinopathy. In a case control study on the intake of various macronutrients and DR among patients with type 2 diabetes, trans-fat was not statistically significant (133).

Alcohol and retinopathy

There are conflicting results from the several studies that have investigated the relationship between alcohol use and the prevalence of DR. In a prospective study (159), the risk for DR was two times greater in those with heavy alcohol intake, compared to those with no or moderate alcohol intake. This finding suggests a link between alcohol use and DR risk, but only for those who consume heavy amounts of alcohol. This is different from the protective effect that has been reported by several previous studies (22, 160, 161). The protective association was between light to moderate alcohol consumption and the presence of DR. However, these studies were cross-sectional. Other studies did not find any significant association between alcohol and retinopathy (60, 162).

1.11 Rationale for the thesis

The rationale for conducting this research stems from the paucity of studies that have examined the association between nutrient intake and retinopathy in the general population. Existing

literature focuses on specific types of retinopathies, such as diabetic and hypertensive retinopathy (60, 130, 135, 149). Regardless of diabetes status, there is a knowledge gap on the link between dietary intake and retinopathy in the general population. Retinopathy was found to be prevalent in those without diabetes in many previous studies (25, 27, 163). This finding underlines the necessity of critically investigating the association between nutrient intake and retinopathy in the general population as opposed to simply people with diabetes.

1.12 Research question

What is the association between intake of various nutrients and retinopathy in a general Norwegian adult population?

2 Method

2.1 Study design methodology

This is an analytical cross-sectional study using data from the seventh survey of the Tromsø Study (Tromsø7). The data used includes a sub study of the Tromsø Study known as the Tromsø Eye Study.

2.2 Study population

The Tromsø Study is an ongoing population-based survey conducted in the Tromsø municipality of Northern Norway, consisting of 7 surveys to date (Tromsø1- Tromsø7) (164, 165). The Tromsø Study began in 1974 to investigate the risk factors associated with CVD which had high prevalence in Northern Norway during that time. In later surveys, it has been expanded to include other diseases, behavioural factors as well as lifestyle factors (164). The most recent study, Tromsø7 (166) was carried out in 2015-2016. The survey included two consecutive visits 2-4 weeks apart. Invitations to visit 1 were sent out to all inhabitants aged 40 years and older living in Tromsø (n=32,591). In total 21,083 (65%) of the invitees attended the first visit. A pre-marked random sample of 20% aged 40-59 years and 50% aged 60-84 years (n=9,925) as well as previous participants attending dual-energy X-ray absorptiometry (DXA), echocardiography and/or eye examinations in Tromsø6 (n=3,103) were invited to attend the second visit. In total, 13,028 of the originally invited sample were eligible for the second visit. Of these, 9253 attended visit 1. Invitations to visit 2 were given at visit 1. In total 8,356 attended visit 2, thus the attendance was 64% of the originally pre-marked sample (and 90% of those attending visit 1).

2.2.1 Sampling Criteria

The sampling criteria for this study comprised of participants from the second visit of Tromsø7 with valid images that were gradable from retinal photographic examinations and valid data from completion of a food frequency questionnaire (FFQ) from the first visit. The retinal images were classified valid if the quality of the photograph was sufficient for the detection of tiny microaneurysms in at least an area equal to four out of the five 45-degree photographic fields (9). Valid FFQ data as defined by the production described in Lundblad et al (167) i.e., inclusion of participants with ≥90% completion of the FFQ and exclusion of participants with unrealistic energy intake (too low or too high). Also, participants with missing data for covariates were excluded from this study. Below is the flowchart based on the sampling criteria. A total of 4,724 participants were included in the present study (fig 4).

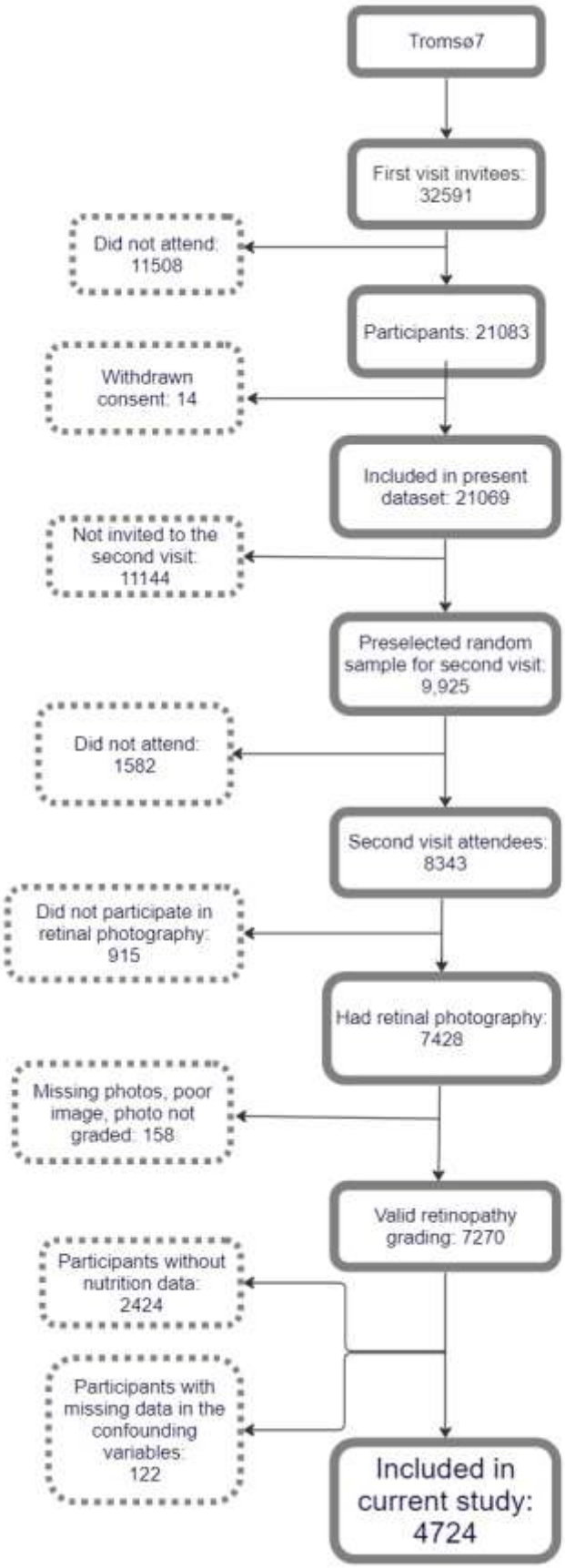


Figure 4: Flowchart for participants in dataset

See supplementary material 1 for a detailed flowchart that outlines the exclusion of participants based on the different criteria of having complete nutrient data.

2.3 Data collection

In Tromsø7, the invitees were sent an invitation letter and an information brochure by mail that contained comprehensive information on the Tromsø Study alongside a unique username and password to complete questionnaires online before study attendance (165). Questionnaires included queries on sociodemographic factors, lifestyle factors and health.

At the first visit, the participants went through clinical examinations (including blood pressure and anthropometric measurements). The participants also received a paper version of a comprehensive food frequency questionnaire (FFQ) developed at the University of Oslo (UiO) (168). This FFQ was used to assess the dietary intake of each participant and has previously been validated for estimating the intake of foods and nutrients (169, 170). It included questions about 261 foods (including beverages) and dietary supplements. The participants were asked to indicate the habitual frequency and amount of the consumption of each item during the past year. The participants could complete the questionnaire at the examination site or return the questionnaire by mail using a pre-paid envelope. The FFQ data was scanned and sent to UiO for the computation of food and nutrient intakes using the food database KBS AE14 and KBS software system (167). This food database is based on the Norwegian food composition tables from 2014 to 2015 for calculating the food and nutrient intakes.

At the second visit, the participants went through several comprehensive examinations. The eye examinations included a visual acuity test and refraction measured with an autorefractor (Nidek ARK 560, Gamagori, Japan), intra-ocular pressure with an I-care tonometer (model TA01i; Helsinki, Finland) on both eyes, retinal photography with a Visucam 500 (Carl Zeiss Meditec, Jena, Germany) and optical coherence tomography of both eyes with a Cirrus HD-OCT 4000 (Carl Zeiss Meditec, USA) (166).

2.4 Variables

2.4.1 Outcome variable: retinopathy

The outcome variable “retinopathy” was obtained from the retinal images of the participants that was taken during the second visit. Data was obtained from the retinal photographs such as the severity of the retinopathy, the presence and count of microaneurysms and retinal haemorrhages, as well as the presence of hard or soft exudates, macular oedema, intraretinal

microvascular abnormalities, or venous beading. The study outcome, which was retinopathy, was determined by the presence of any of these specific lesions, regardless of whether the individual had diabetes or not. The lesions were then graded using the International Clinical Diabetic Retinopathy and Diabetic Macular Oedema Severity Scales (96). The images were graded as zero (0) for absence of retinopathy and graded 1- 4 for presence of retinopathy, based on the characteristics and severity of retinopathy. These grading was done separately for both eyes, during data cleaning a single variable was created taking the retinopathy grade in the worst eye, since cases of asymmetric retinopathy has been reported in previous studies (171, 172). For the purpose of analysis, all participants without retinopathy (graded as 0), were coded as 0 as retinopathy-no, while all participants graded 1- 4 were recoded into 1 as retinopathy-yes. This was because there were few participants in some groups hence there will be no power to carry out an ordinal logistic regression.

2.4.2 Exposure variable: macronutrients

The exposure variables are macronutrients including carbohydrates, proteins, fats (saturated, trans, MUFA and PUFA), added sugar, dietary fibre, and alcohol. These were categorized based on the NNR (173).

Table 2: The Nordic Nutrition recommendations of intake for the selected macronutrients

Macronutrients	Recommendation
Carbohydrates	45-60 % of total energy intake
Dietary fibre	>25 grams for women & >35 grams for men per day
Added sugar	<10% of total energy intake
Total fat	25-40 % of total energy intake
Saturated fatty acids	< 10 % of total energy intake
Monounsaturated fatty acids	10-20 of total energy intake
Polyunsaturated fatty acids	5-10 of total energy intake
Trans fat	As low as possible
Protein	10-20 % of total energy intake
Alcohol	≤5 % of total energy intake

The nutrients were reported as percentage of the total energy intake, which was used to create categories of compliance to the Nordic nutrition guidelines except for dietary fibre which had to be converted into grams (NNR recommendation only in grams). Dietary fibre intake (g/day) was calculated as: $\frac{\text{Total energy intake in kJ/day}}{4.184} \times \frac{\text{Dietary fiber intake (as \% of energy)}}{2\ \text{kcal/g}}$.

2.4.3 Covariates

Age was used as a descriptive variable for the study population and included in the statistical model, as it is an established confounder (16, 52). The age at enrolment was categorized into a 10-year groups: 40-49 years, 50-59 years, 60-69 years, 70-79 years and 80 plus years.

BMI was calculated using the height and weight measurements from the first visit. This was then categorized into four groups according to the World Health Organization (WHO) BMI criteria: "*underweight*" for a BMI less than 18.5 kg/m^2, "*normal weight*" for a BMI of 18.5 to 24.9 kg/m^2, "*overweight*" for a BMI of 25 to 29.9 kg/m^2, and "*obese*" for a BMI greater than 30 kg/m^2.

Blood pressure was measured three times with two minutes interval and a mean value from the last two measurements was obtained. Both the systolic and diastolic blood pressure was recorded. In addition, self-reported use of antihypertensive medications was also obtained; Participants were asked "*Do you use, or have you used blood pressure lowering drugs?*" and they could choose from any of the response alternatives provided: *"Never used", "Currently", and "Previously, not now"*. Hypertension was defined as a systolic blood pressure >130 mmHg and/or diastolic blood pressure >80 mmHg in accordance with the definition by the American Heart Association (174), and/or current use of antihypertensive medication.

Information on the diabetes status of the participants was obtained from self-reported diabetes status, self-reported use of antidiabetics, and the level of glycated haemoglobin (HbA1c). In the health and disease section of the questionnaire the participants were asked "*Do you have, or have you had diabetes?*" the response alternatives were "*No*", "*Yes now*" and *"Yes, previously"*. They were also asked *"Do you use, or have you used tablets for diabetes?"* and *"Do you use, or have you used insulin?"*. The response alternatives provided to both questions were *"Never used", Currently", and "Previously, not now"*. HbA1c ≥6.5% (175) and/or self-reported current diabetes and/or current use of antidiabetics (insulin and other diabetic treatments) was categorized as "*yes*" in the diabetes variable.

Level of education was obtained from the questionnaire. Participants were asked "*What is the highest levels of education you have completed*?" education was categorized into four groups: primary/partly secondary education, (up to 10 years of schooling), upper secondary education (a minimum of 3 years), tertiary education, short (college/university less than 4 years) and

tertiary education, long (college/university 4 years or more). Level of education included as a proxy for SES in this study.

Physical activity was obtained from the questionnaire, by asking the participants "*How often do you exercise (i.e walking, skiing, swimming, or training/sports*)? This was grouped into: never, less than once a week, once a week, 2-3 times a week and approximately every day. Physical activity was used as a proxy for lifestyle factors.

2.4.4 Selection of potential confounders

Some of the possible covariates from the literature and available data were age, BMI, gender, smoking, physical activity, level of education, hypertension, diabetes, stroke, use of antihypertensive and duration of diabetes (133, 176, 177). Directed Acyclic Graph (DAG) was used to systematically differentiate between confounders, colliders, and mediators. The DAG, which was created using DAGitty's online software, helps as a causal diagram to reduce bias in epidemiological studies(178, 179).

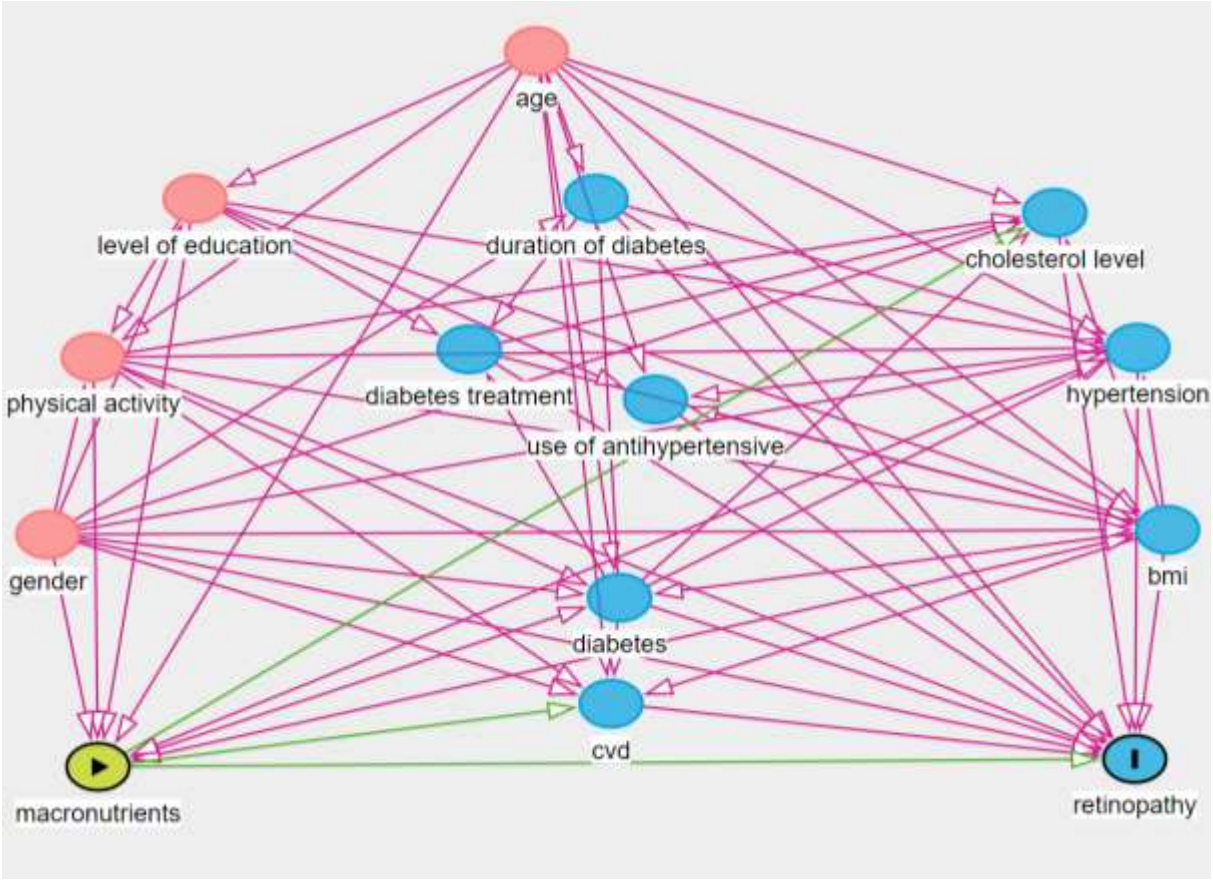


Figure 5: DAG representing the association between nutrients and retinopathy and possible confounders (180).

The confounders identified were age, physical activity, education, BMI, hypertension, diabetes treatment and duration of diabetes. Since this is a cross sectional study, which is characterized by lack of temporality, the DAG was therefore used only for illustrative purpose.

2.5 Statistical analysis

Data was cleaned and all missing variables were excluded. New variables such as diabetes, hypertension and retinopathy were created from existing variables while some were recoded into new variables. All new variables were then double-checked to ensure that they were correctly coded and accurately represented the original data. In addition, all variables were inspected by their means, range, identifying possible outliers and histograms to display the distribution of each of the variables. After which a complete case analysis was done.

The descriptive statistics was carried out on all necessary characteristics of the participants were which were stratified by retinopathy. The proportion in each group of categorical variables were calculated while mean values and standard deviations was used for continuous variables. In comparison to retinopathy, the categorical variables were accessed using Chi square while independent t-test was used to compare the mean values of the continuous variables. The exposure, outcome and covariates were categorized as described above in section 3.4.

Binary logistic regression was performed to investigate the association between nutrients and retinopathy since the dependent variable has been collapsed into a binary variable. The dependent variable-retinopathy was included as a binary outcome (yes/no) while the exposure variable was macronutrients (table 2). Stratification was done based on the diabetes status in all logistic regression. In all regression analysis, an age and gender adjusted model was done first, before adjusting for physical activity, hypertension, BMI, and level of education. In addition, use of diabetic treatment and duration of diabetes were adjusted for in the group who had diabetes. There were several analyses conducted; the first set of analyses compared odds of retinopathy to the compliance of recommended level of each of the nutrients, each nutrient was included in a separate logistic regression model with the outcome and adjusted as described above. This association was then further investigated by regression for nutrients that had range in the NNR (carbohydrate, protein, total fat, MUFA and PUFA). This was done to investigate if there was difference in the association between the individuals whose dietary intake was below the recommended level and those who had intakes above the recommended level. In

another set of analysis, the nutrients were included as continuous variables into the models to investigate the linear association with retinopathy.

To assess the overall impact of healthy nutrient intake on retinopathy, the odds of retinopathy was compared based on the compliance of each macronutrient to the recommended guidelines. For each participant, a new variable was created which represented the cumulative compliance of all the nutrients. Compliance was determined based on whether each nutrient intake met the recommended levels (compliant = 1, non-compliant = 0) and was included in the statistical model as a continuous variable. Odds ratios (OR) with confidence intervals (CI) were used to estimate these associations. P values below 0.05 were considered statistically significant. Data science statistical software (STATA 17) was used for all analysis and was set at 5% significance level.

2.6 Ethical consideration

The data collection of the Tromsø7 was approved by the Regional Committee of Medical and Health Research Ethics (REK) North (reference 2014/90) and the Norwegian Data Protection Authority (reference 14/01463-4/CGN). The study complies with the declaration of Helsinki, International Ethical Guidelines for Biomedical Research Involving Human Subjects, and the International Guidelines for Ethical Review of Epidemiological Studies. Each participant gave a written consent. The dataset used for the current analysis was anonymized by Tromsø Study before the data was handed out. The anonymization process performed by the Tromsø Study was ensured by grouping potentially identifiable variables such as age, BMI, and comorbidities. Thus, approval from REK is not needed.

2.7 Access to data and data storage.

Data from the Tromsø Study is stored in the EUTRO database (Forskningsdata i system), which is an IT solution designed to protect and manage data. EUTRO implements the Data Protection Impact Assessment (Personvernkonsekvensvurdering - DPIA) and is evaluated and approved by the Norwegian Data Protection Authority (165). Access to data was applied for to the Tromsø Study Data and Publication Committee (DPU). The application was presented together with the list of necessary variables required to answer the research question. After permission was granted, and data received, it was stored in a password protected laptop and will be deleted upon completion and submission.

3 Result

3.1 Descriptive statistics

The study included a total of 4,724 participants, 4,145 (87.7%) without retinopathy and 579 (12.3%) with retinopathy. Most participants were in the age group of 60-69 years (42.2%) followed by those aged 70-79 years (20.8%). Higher percentage of the participants who had retinopathy were found among the age group of 60-69 years (43.7%). Gender distribution revealed that more than half of the participants were female (53.3%). The prevalence of retinopathy was slightly higher in males (52%) than in females (48%). The mean BMI was (27.82 ± 4.67) among those with retinopathy and (27.12 ± 4.26) among those without retinopathy. Regarding the distribution of BMI categories, majority of the participants fell into the overweight category (45.1% of those without retinopathy, 44% of those with retinopathy), and then normal weight category (32.3% of those without retinopathy, 28.2% of those with retinopathy).

The proportion of participants with hypertension was higher among those with retinopathy (72.5%) compared to those without (59.7%), likewise, the proportion of participants with diabetes was also higher among those with retinopathy (15.7%) compared to those without (4.3%). The prevalence of retinopathy was slightly higher in individuals who reported being physically active less than once a week (12.8%) and once a week (14.3%) and never (5.4%). However, the proportion of participants with primary or partly secondary education was higher among those with retinopathy (31.6%) compared to those without (24.1%). Among those with long tertiary education, those without retinopathy (27.4%) were more than those with retinopathy (19.2%). Regarding dietary factors, the mean energy intake was similar between the two groups, with a total mean intake of 9,534 ± 2,924 kJ/day. There were no significant differences in carbohydrate, dietary fibre, protein, total fat, saturated fat, trans-fat, cis monounsaturated fat, cis polyunsaturated fat, and alcohol intake between individuals with and without retinopathy (table 3).

Table 3: Baseline characteristics according by retinopathy status. The Tromsø Study 2015-2016.

Characteristics	No retinopathy	Retinopathy	Total sample	ρ
Participants, n (%)	4145(87.74)	579(12.26)	4724(100)	
Age group, n (%)				
40-49 years	681(16.43)	68(11.74)	749(15.86)	
50-59 years	801(19.32)	81(13.99)	882(18.67)	
60-69 years	1738(41.93)	253(43.70)	1991(42.15)	
70-79 years	822(19.83)	162(27.98)	984(20.83)	
80+ years	103(2.48)	15(2.59)	118(2.5)	<.001
Gender, n (%)				
Female	2242(54.09)	278(48.01)	2520(53.34)	
Male	1903(45.91)	301(51.99)	2204(46.66)	.006
BMI (kg/m^2), m (SD)	27.12(4.27)	27.82(4.67)	27.21(4.32)	.0002
BMI, n (%)				
Underweight	22(0.53)	3(0.52)	25(0.53)	
Normal weight	1341(32.35)	163(28.15)	1504(31.84)	
Overweight	1871(45.14)	255(44.04)	2126(45.00)	
Obese	911(21.98)	158(27.29)	1069(22.63)	.03
Hypertension, n (%)				
No	1671(40.31)	159(27.46)	1830(38.74)	
Yes	2474(59.69)	420(72.54)	2894(61.26)	<.001
Diabetes, n (%)				
No	3965(95.66)	488(84.28)	4453(94.26)	
Yes	180(4.34)	91(15.72)	271(5.74)	<.001
Physical activity, n (%)				
Never	135(3.26)	31(5.35)	166(3.52)	
Less than once a week	448(10.81)	74(12.78)	522(11.07)	
Once a week	549(13.24)	83(14.34)	632(13.4)	
2-3 times a week	1799(43.40)	240(41.45)	2,039(43.16)	
Approximately every day	1214(29.29)	151(26.08)	1365(28.90)	0.028
Level of education, n (%)				
Primary/partly secondary education.	999(24.10)	183(31.61)	1182(25.02)	
Upper secondary education	1176(28.37)	162(27.98)	1338(28.32)	
Tertiary education, short	834(20.12)	123(21.24)	957(20.26)	
Tertiary education, long	1136(27.41)	111(19.17)	1247(26.40)	<.001
Energy (kJ/day), m (SD)	9527.95(2929.19)	9575.46(2888.86)	9533.77(2924.01)	.71
Carbohydrate (E%), m (SD)	42.18(6.15)	42.27(5.92)	42.19(6.12)	.75
Dietary fibre (g), m (SD)	26.99 (9.14)	26.65 (8.42)	26.95(9.06)	.40
Sugar (E%), m (SD)	5.55(3.20)	5.32(3.25)	5.52(3.21)	.11
Protein (E%), m (SD)	17.68(2.51)	17.88(2.49)	17.71(2.51)	.08
Total fat, (E%), m (SD)	34.24(5.70)	34.11(5.77)	34.22(5.71)	.61
Saturated fat (E%), m (SD)	12.47(2.70)	12.63(2.70)	12.49(2.70)	.16
Trans-fat (E%), m (SD)	0.31(0.13)	0.31(0.13)	0.31(0.13)	.99
MUFA (E%), m (SD)	12.62(2.64)	12.46(2.83)	12.60(2.66)	.17
PUFA (E%), m (SD)	6.03(1.55)	5.92(1.57)	6.02(1.55)	.13
Alcohol (E%), m (SD)	3.47(3.92)	3.35(3.91)	3.46(3.92)	.48

BMI: Body mass index, PUFA: Polyunsaturated fat, MUFA: Monounsaturated.

Categorical variables are presented as proportions.

Continuous variables are presented as means with standard deviation (SD) in parenthesis.

Nutrients are presented as mean % of energy intake (E%) except dietary fibre which is presented as mean of the intake in grams(g).

These descriptive factors were also stratified by both gender and diabetes to investigate potential differences in retinopathy. See Supplementary Materials 2 and 3 for the tables. The participants included a relatively even distribution of gender and age groups, with a higher proportion of participants with hypertension and diabetes in the group with retinopathy. Individuals with retinopathy were more likely to be older, male, have a higher BMI, have hypertension, diabetes, and have a lower level of education compared to those without retinopathy. Physical activity levels and dietary intake did not significantly differ between the two groups.

3.2 Descriptive statistics based on NNR.

Table 4 shows the descriptive statistics comparing the compliance levels to NNR for macronutrients in by retinopathy status. Most participants did not consume the recommended levels of carbohydrate, dietary fibre, and saturated fat. On the other hand, most participants consumed sugar, protein, alcohol, total fat, trans-fat, PUFA and MUFA at the recommended level. Almost all participants consumed trans-fat above the recommended level. Additionally, there was no significant difference in compliance rates between the non-retinopathy and retinopathy groups.

Table 4: Compliant levels of participants to the NNR according to the retinopathy status. The Tromsø Study 2015-2016.

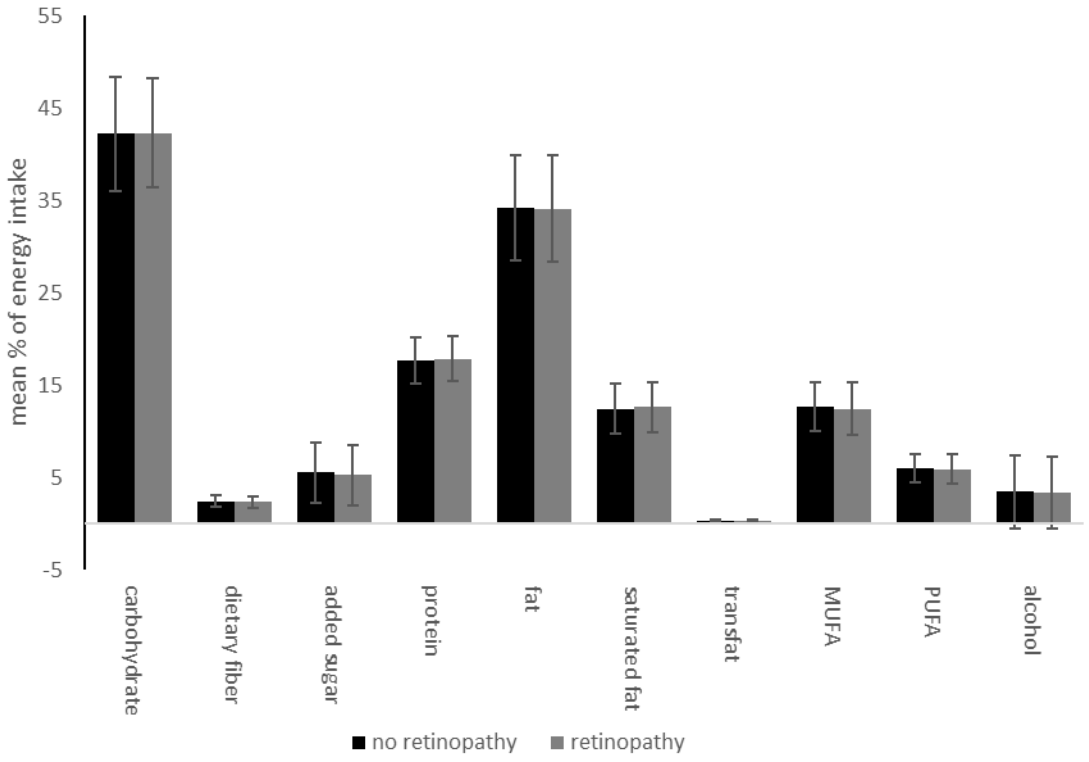
Characteristics n (%)	No retinopathy	Retinopathy	Total sample	*ρ*
Carbohydrate				
Compliant: No	2811(67.82)	383(66.15)	3194(67.61)	
Yes	1334(32.18)	196(33.85)	1530(32.39)	.42
Below	2803(67.62)	381(65.8)	3184(67.4)	
Normal	1334(32.18)	196(33.85)	1530(32.39)	
Above	8(0.19)	2(0.35)	10(0.21)	.54
Sugar				
Compliant: No	347(8.37)	48(8.29)	395(8.36)	
Yes	3798(91.63)	531(91.71)	4329(91.64)	.95
Dietary fibre				
Compliant: No	2612(63.02)	387(66.84)	2999(63.48)	
Yes	1533(36.98)	192(33.16)	1725(36.52)	.07
Protein				
Compliant: No	671(16.19)	108(18.65)	779(16.49)	
Yes	3474(83.81)	471(81.35)	3945(83.51)	.13
Below	6(0.14)	1(0.17)	7(0.15)	
Normal	3474(83.81)	471(81.35)	3945(83.51)	
Above	665(16.04)	107(18.48)	772(16.34)	.33
Alcohol				
Compliant: No	1028(24.8)	139(24.01)	1167(24.7)	
Yes	3117(75.2)	440(75.99)	3557(75.3)	.68
Total fat				
Compliant: No	739(17.83)	92(15.89)	831(17.59)	
Yes	3406(82.17)	487(84.11)	3893(82.41)	.25
Below	165(3.98)	22(3.8)	187(3.96)	
Normal	3406(82.17)	487(84.11)	3893(82.41)	
Above	574(13.85)	70(12.09)	643(13.63)	.49
Saturated fat				
Compliant: No	3,511(84.7)	494(85.32)	4005(84.78)	
Yes	634(15.3)	85(14.68)	719(15.22)	.7
Trans fat				
Compliant: No	8(0.19)	0(0)	8(0.17)	
Yes	4137(99.81)	579(100)	4724(99.83)	.29
MUFA				
Compliant: No	625(15.08)	105(18.13)	730(15.45)	
Yes	3520(84.92)	474(81.87)	3994(84.55)	.06
Below	576(13.9)	94(16.23)	670(14.81)	
Normal	3520(84.92)	474(81.87)	3994(84.55)	
Above	49(1.18)	11(1.90)	60(1.27)	.1
PUFA				
Compliant: No	1154(27.84)	164(28.32)	1318(27.9)	
Yes	2991(72.16)	415(71.68)	3406(72.1)	.81
Below	1084(26.15)	153(26.42)	1237(26.19)	
Normal	2991(72.16)	415(71.68)	3406(72.1)	
Above	70(1.69)	11(1.9)	81(1.71)	.92

PUFA: Polyunsaturated fat, MUFA: Monounsaturated.

Compliance to the NNR as yes for those whose E% met the recommended levels.

The NNR recommends a range of amounts for MUFA, PUFA, total fat, protein, and carbohydrates. As a result, three categories for these nutrients were developed: Normal (within the recommended level), above the recommended level, and below the recommended level.

The bar chart (figure 6) of the mean dietary intake of macronutrients showed the similarity between the group with retinopathy and the group without. Both groups had similar mean and standard deviation values for the macronutrient intake.



PUFA: Polyunsaturated fat, MUFA: Monounsaturated.

Figure 6: Bar chart of mean % of energy intake and standard deviations of various macronutrient by retinopathy groups

3.3 Logistic regression

Results from the binary logistic regression analysis between the compliance to the NNR and retinopathy in people with and without diabetes are shown in table 5. For participants with or without diabetes, none of the macronutrients based on compliance to NNR was associated with retinopathy both in the age and gender adjusted and the multivariable models.

Table 5: Logistic regression analysis between macronutrients compliance to NNR and retinopathy. The Tromsø Study 2015-2016.

	No diabetes		**Diabetes**	
	Age & gender adjusted OR (95% CI)	Multivariable* OR (95% CI)	Age & gender adjusted OR (95% CI)	Multivariable** OR (95% CI)
Carbohydrate	0.91(0.75-1.12)	0.92(0.76-1.13)	1.02(0.56-1.86)	0.71(0.31-1.63)
Sugar	1.03(0.74-1.44)	1(0.71-1.4)	0.78(0.25-2.41)	0.87(0.11-7.08)
Dietary fibre	1.08(0.87-1.34)	1.01(0.81-1.26)	0.82(0.45-1.51)	0.76(0.34-1.71)
Protein	1.1(0.85-1.41)	1.06(0.83-1.37)	1.14(0.63-2.03)	1.18(0.54-2.57)
Alcohol	0.99(0.79-1.22)	1.02(0.82-1.28)	0.74(0.37-1.47)	0.82(0.34-1.96)
Total fat	0.84(0.65-1.1)	0.85(0.65-1.11)	0.87(0.47-1.59)	0.65(0.28-1.53)
Saturated fat	1.03(0.79-1.34)	1.04(0.8-1.35)	1.58(0.76-3.31)	1.5(0.53-4.27)
Trans-fat	-	-	-	-
MUFA	1.22(0.96-1.56)	1.21(0.95-1.55)	0.93(0.46-1.91)	1.64(0.19-2.21)
PUFA	1.09(0.89-1.34)	1.09(0.88-1.34)	0.67(0.35-1.27)	0.73(0.28-1.9)

PUFA: Polyunsaturated fat, MUFA: Monounsaturated, OR.: odds ratio, CI: confidence interval

Reference group is the those who met the recommended levels.

*Multivariable model adjusted for age, gender, education, physical activity, hypertension, BMI (Body mass index).

**Multivariable model adjusted for age, gender education, physical activity, hypertension, BMI, duration of diabetes and use of diabetes treatment.

Trans-fat was omitted in the analysis because of no observation in some of its group.

Some of the recommended levels of nutrients are given in ranges, so there were groups of people who were below the compliant level and above the compliant level. The reference group used was individuals with normal intake of macronutrients. Compliance with NNR for macronutrients was not significantly associated with retinopathy both in those with and without diabetes (table 6).

Table 6: Logistic regression analysis between macronutrients compliance to NNR (for only nutrients recommended as range) and retinopathy. The Tromsø Study 2015-2016.

	No diabetes		**Diabetes**	
	Age & gender adjusted OR (95% CI)	Multivariable* OR (95% CI)	Age & gender adjusted OR (95% CI)	Multivariable** OR (95% CI)
Carbohydrate				
Normal	Ref (1.0)	Ref (1.0)	Ref (1.0)	Ref (1.0)
Below	0.91(0.75-1.11)	0.92(0.76-1.13)	0.9(0.51-1.59)	0.71(0.31-1.61)
Above	2.26(0.47-10.86)	2.18(0.45-10.55)	-	-
Protein				
Normal	Ref (1.0)	Ref (1.0)	Ref (1.0)	Ref (1.0)
Below	2.09(0.23-18.97)	2.07(0.22-18.99)	-	-
Above	1.09(0.85-1.4)	1.06(0.82-1.37)	1.19(0.66-2.14)	1.24(0.56-2.72)
Total fat				
Normal	Ref (1.0)	Ref (1.0)	Ref (1.0)	Ref (1.0)
Below	0.97(0.6-1.54)	0.97(0.61-1.55)	0.32(0.04-2.69)	-
Above	0.8(0.59-1.09)	0.81(0.6-1.10)	0.95(0.51-1.78)	0.94(0.49-1.8)
MUFA				
Normal	Ref (1.0)	Ref (1.0)	Ref (1.0)	Ref (1.0)
Below	1.23(0.95-1.58)	1.21(0.94-1.56)	0.64(0.26-1.57)	0.5(0.11-2.32)
Above	1.15(0.45-2.92)	1.23(0.48-3.15)	2.01(0.63-6.47)	0.99(0.15-6.62)
PUFA				
Normal	Ref (1.0)	Ref (1.0)	Ref (1.0)	Ref (1.0)
Below	1.11(0.9-1.37)	1.12(0.9-1.378)	0.5(0.24-1.07)	0.62(0.2-1.95)
Above	0.67(0.27-1.68)	0.65(0.26-1.63)	1.52(0.49-4.71)	1.05(0.21-5.24)

PUFA: Polyunsaturated fat, MUFA: Monounsaturated, OR.: odds ratio, CI: confidence interval

*Multivariable model adjusted for age, gender, education, physical activity, hypertension, BMI (Body mass index).

**Multivariable model adjusted for age, gender, education, physical activity, hypertension, BMI, duration of diabetes and use of diabetes treatment.

In the group who had diabetes, above the recommended level of carbohydrate, below the recommended level of protein and below recommended total fat was omitted in the analysis because of lack of power.

This association was further investigated by including the nutrient variables in its continuous form. For individuals who had diabetes, there were no significant finding. For individuals with diabetes, after adjusting for age and gender, higher MUFA intake was associated with a lower odd of retinopathy, with an OR of 0.96 (95% CI: 0.92-0.99, p = .02) in the age and gender adjusted model and an OR of 0.96 (95% CI: 0.92-0.99, p = .03) in the multivariable model. Also, higher PUFA intake was associated with reduced odds of retinopathy, with an OR of 0.9 (95% CI: 0.85-0.96, p = .002) in the age and gender adjusted model and an OR of 0.9 (95% CI: 0.85-0.96, p = .002) in the multivariable model (table 7).

Table 7: Logistic regression analysis between macronutrients and retinopathy. The Tromsø Study 2015-2016.

	No diabetes		**Diabetes**	
	Age & gender adjusted OR (95% CI)	Multivariable* OR (95% CI)	Age & gender adjusted OR (95% CI)	Multivariable** OR (95% CI)
Carbohydrate	1(0.99-1.02)	1.01(.99-1.02)	.99(.95-1.03)	1.01(.95-1.07)
Sugar	0.99(0.96-1.02)	0.98(0.95-1.01)	0.99(0.9-1.08)	0.96(0.83-1.1)
Dietary fibre	0.99(0.98-1)	1(0.98-1.01)	1.01(0.98-1.04)	1.02(0.99-1.06)
Protein	1.02(0.98-1.06)	1.02(0.98-1.06)	1.01(0.92-1.11)	1.04(0.91-1.19)
Alcohol	0.99(0.97-1.02)	1(0.97-1.02)	0.98(0.91-1.06)	1.02(0.92-1.12)
Total fat	0.99(0.97-1.01)	0.99(0.97-1.01)	1.02(0.98-1.05)	0.98(0.93-1.04)
Saturated	1.03(1-1.07)	1.03(0.99-1.07)	0.98(0.91-1.06)	0.94(0.84-1.06)
Trans-fat	1.42(0.68-2.98)	1.54(0.74-3.22)	0.4(0.07-2.41)	0.25(0.02-3.58)
MUFA	**0.96(0.92-0.99)**	**0.96 (0.92-0.99)**	1.06(0.99-1.15)	0.98(0.87-1.11)
PUFA	**0.9(0.85-0.96)**	**0.9(0.85-0.96)**	1.12(0.97-1.29)	1(0.81-1.24)

PUFA: Polyunsaturated fat, MUFA: Monounsaturated, OR.: odds ratio, CI: confidence interval

*Multivariable model adjusted for age, gender, education, physical activity, hypertension, BMI (Body mass index).

**Multivariable model adjusted for age, gender, education, physical activity, hypertension, BMI, duration of diabetes and use of diabetes treatment.

To compare the overall healthy intake of these nutrients the compliant level across all the macronutrients is presented in Table 8. The association did not reach statistical significance in the age and gender adjusted model and also after adjusting for potential confounders in the multivariable model.

Table 8: Logistic regression analysis between overall compliance to NNR for all the macronutrients and retinopathy. The Tromsø Study 2015-2016.

	No diabetes		**Diabetes**	
	Age & gender adjusted OR (95% CI)	Multivariable* OR (95% CI)	Age & gender adjusted OR (95% CI)	Multivariable** OR (95% CI)
Overall compliant nutrient intake	0.98(0.91-1.06)	0.98(0.91-1.06)	1.09(0.88-1.35)	1.17(0.87-1.57)

OR.: odds ratio, CI: confidence interval

*Multivariable model adjusted for age, gender, education, physical activity, hypertension, BMI (Body mass index).

**Multivariable model adjusted for age, gender, education, physical activity, hypertension, BMI, duration of diabetes and use of diabetes treatment.

3.4 Characteristics of those included vs those excluded in study sample

The sensitivity analysis result comparing characteristics of the study participants that were included and excluded from the study. The included participants consisted of 4,724 individuals, representing 22.4% of the total sample size of 21,069 participants, while the excluded participants were 16,345 (77.6%). The included and excluded groups had similar gender distributions, BMI, and diabetes status, with no significant differences observed. However, age distribution of the included and excluded participants differed, with a higher percentage of participants aged 60-69 years (42.2%) in the included group compared to the excluded group (19.5%). The included group had a higher percentage of participants with hypertension (61.3%) than the excluded group (51.2%), the included group also reported higher physical activity levels, with a larger proportion reporting engaging in physical activity 2-3 times per week (43.2%) compared to the excluded group (41%) and level of education was also significantly different between the included and excluded groups ($p<0.001$). The analysis shows that included participants were more likely to be between the ages of 40-69 years, have hypertension, be more physically active, and have higher levels of education compared to those who were excluded (table 9).

Table 9: Characteristics of those included vs those excluded in study sample. The Tromsø Study 2015-2016.

Characteristics	Included	Excluded	Total sample	ρ
Participants, n (%)	4724(22.42)	16345(77.58)	21,069(100)	
Age group, n (%)				
40-49 years	749(15.86)	5677(34.73)	6426(30.5)	
50-59 years	882(18.67)	5150(31.51)	6032(28.63)	
60-69 years	1991(42.15)	3185(19.49)	5176(24.57)	
70-79 years	984(20.83)	1691(10.35)	2675(12.7)	
80+ years	118(2.5)	642(3.93)	760(3.61)	<.001
Gender, n (%)				
Female	2520(53.34)	8543(52.27)	11,063(52.51)	
Male	2204(46.66)	7802(47.73)	10,006(47.49)	.19
BMI (kg/m^2), m (SD)	27.2(4.32)	27.38(4.61)	27.34(4.54)	.02
BMI, n (%)				
Underweight	25(0.53)	89(0.54)	114(0.54)	
Normal weight	1504(31.84)	5130(31.39)	6634(31.49)	
Overweight	2126(45)	7070(43.25)	9196(43.65)	
Obese	1069(22.63)	4056(24.81)	5125(24.32)	.02
Hypertension, n (%)				
No	1830 (38.74)	7973(48.78)	9803(46.53)	
Yes	2894(61.26)	8372(51.22)	11266(53.47)	<.001
Diabetes, n (%)				
No	4453(94.26)	15412(94.29)	19865(94.29)	
Yes	271(5.74)	933(5.71)	1204(5.71)	.94
Physical activity, n (%)				
Never	166(3.51)	663(4.15)	829(4.01)	
Less than once a week	522(11.05)	1938(12.14)	2460(11.89)	
Once a week	632(13.38)	2462(15.42)	3094(14.95)	
2-3 times a week	2039(43.16)	6543(40.97)	8582(41.47)	
Approximately every day	1365(28.9)	4365(27.32)	5728(27.68)	<.001
Level of education, n (%)				
Primary/partly secondary education.	1182(25.02)	3613(22.63)	4795(23.17)	
Upper secondary education	1338(28.32)	4410(27.62)	5748(27.78)	
Tertiary education, short	957(20.26)	3048(19.09)	4005(19.36)	
Tertiary education, long	1247(26.4)	4896(30.66)	6143(26.69)	<.001

BMI: Body mass index.

Categorical variables are presented as proportions.

Continuous variables are presented as means with standard deviation (SD) in parenthesis.

4 Discussion

4.1 Results

The result of this study gives insight into the association between retinopathy and various macronutrients among adults from a general population in Norway. In the main analysis, a significant association was not found with the macronutrients except MUFA and PUFA. However, among those with diabetes, the association was diminished and was no longer statistically significant.

The current study found an association between MUFA and retinopathy among those without diabetes: MUFA was associated with a decreased odds of retinopathy among those without diabetes. It, (to a great extent) has not found any study that has investigated this association in those without diabetes. There is, however, studies investigating the association between MUFA and retinopathy among those with diabetes. A previous study (133) comparing the intake of MUFA among those with DR and those without DR, found a decreased risk that was stronger among those with longer duration of diabetes. Although the study found a similar association as the current study, the differences in the characteristics of the study population makes it difficult to compare the results.

A study (23) from Australia in the diabetes management project found that increasing the intake of PUFA reduces the odds of retinopathy among those with diabetes. The current study also found decreased odds of retinopathy in those without diabetes, but an unsignificant association in those with diabetes. One possible reason could be the large participants in the diabetic group for the other study, since it was the focus population. Another study (149) also found a protective effect of PUFA particularly diet rich in linoleic acids. The participants of the study were randomized to either a low carbohydrate or a fat diet rich in linoleic acid with a follow up time of 7 years. Like other studies, this protective effect was found in those with diabetes, in contrast, the current study was significantly associated only in those without diabetes.

In all other macronutrients, the current study did not find any significant association with retinopathy. There are some studies that found some association but only in those with diabetes. Studies (132, 133) investigating the association between carbohydrate and retinopathy, did not find any association. However, higher intake of dietary fibre has been previously reported (59) in the group without retinopathy in comparison to those with retinopathy. The study also observed that patients without retinopathy had a lower proportion of their total daily calories derived from protein compared to those with retinopathy (59). Other studies (22, 160) have

however reported a protective association between light to moderate alcohol consumption and the presence of retinopathy, while heavy alcohol intake has been associated with an increased risk of retinopathy (22). These significant association found in these studies were among those with diabetes. The current study did not find any association in these nutrients both in those with diabetes are those without diabetes.

Furthermore, in the present study, the intake of each nutrient was categorised based on the NNR to assess its association with retinopathy based on the level of compliance with these recommendations and its potential association on retinopathy. The findings revealed no significant association between nutrient intake based on compliance to the NNR and retinopathy. This coheres with a previous study (118) that investigated dietary intake based on a different guideline, the Australian Healthy Eating Index. It did not also find any significant association between nutrient intake and retinopathy. It is important to highlight that the study examined the overall diet rather than individual nutrients making it difficult to compare it with the current study. Despite the different guidelines used and the overall dietary approach, both studies consistently demonstrated a lack of association between dietary intake and retinopathy.

4.2 Possible explanations

There are a number of possible explanations as to why this study, in contrast to earlier investigations, reported more generally non-significant associations.

Dietary patterns are influenced by cultural and environmental factors (126), and these changes may explain why there are discrepancies amongst research regarding the relationship between nutrient intake and retinopathy. Similarities in nutrient intake can be attributed to factors including regional food accessibility, cultural dietary preferences, and traditional foods (181). The study participants may have had a high intake of traditional Norwegian foods regardless of their retinopathy status. Also, the participants in this study included middle-aged and older population who may have consciously changed their diets and included dietary supplements as part of their attempts to support their general health and well-being (126). This proactive attitude is frequently seen in older persons who are more likely to embrace healthy living habits. Increased consumption of nutrient-rich foods, such as fruits, vegetables, and whole grains, as well as the use of supplements that target particular nutrients are examples of these possible dietary changes.

People who have specific pathologies, such as diabetes and hypertension, are frequently recommended to change their diets in order to better control their health. These dietary adjustments could entail cutting back on some nutrients, consuming more of others, or following special dietary regimens suggested for those with diabetes or high blood pressure (182). It is likely that the participants in this study who had diabetes or hypertension made deliberate efforts to change their nutrient consumption to conform to the dietary recommendations given to them. This might lead to nutritional profiles that are different to that of people who do not have these pathologies. For instance, those who had diabetes may have increased their consumption of dietary fibre and beneficial fats while decreasing their intake of added sugar and unhealthy fats.

Furthermore, a previous study from the Tromsø Study by Lundblad et al., 2019 (167), showed that the participants in Tromsø7 had a high level of NNR compliance. Since the participants are drawn from the same population, it is plausible that they would have similar dietary behaviours and adhere to the same dietary guidelines. The observed adherence to the NNR may indicate that a significant portion of the population currently adheres to a diet that meets the advised nutritional intakes. Thus, it is possible that this population under study has a homogeneous dietary pattern in terms of nutrient intake. Given that most of the population already meets or closely adheres to the recommended nutrient intakes, it may be difficult to detect substantial changes in the intake of nutrients when comparing groups.

Most early life risk factors of the cardiovascular system are due to cumulative effect, diet is not an exception (124). It is likely that the participants' present dietary consumption is insufficient to explain the possible association with retinal vascular changes. This study's cross-sectional methodology limits it to a particular point in time and only offers a snapshot of nutritional intake. A cross-sectional investigation may miss the potential relationships between dietary consumption and retinopathy since they may take more time to manifest. Some of the previous studies were conducted as prospective cohort studies (133). A more precise representation of the exposure to nutrients and, thus, the association to retinopathy, could be obtained by observing the nutrient intake cumulatively over a longer time period. The use of a single nutrient intake measurement in the current study may have made it impossible to accurately represent the individuals' long-term nutrient intake.

4.3 Methodological discussion

The association between macronutrient intake and retinopathy was examined in this study using a cross-sectional study design. Cross-sectional studies have innate limitations in demonstrating causality, although they are useful in investigating relationships at a particular point in time. Therefore, associations rather than causal links should be used to interpret the results of this study. DAG was used to order the choice of covariates for the statistical models and assisted in the identification of potential confounders. This was done to reduce confounding and improve the internal validity of the findings by incorporating variables found in the DAG (178, 179). This study's methodology has both strengths and limitations in this study. The results of this study must be viewed in context with the fact that the majority of prior investigations on the association between dietary intake and retinopathy mostly focused on individuals who had diabetes (183). To gain a more comprehensive understanding of the association between dietary intake and retinopathy which is not limited to people with diabetes, were included the general population in our study. However, there were only 5.7 % cases of diabetes in our study sample, which was a low prevalence. This low prevalence could have affected the subgroup analyses and may have contributed to the non-significant outcomes.

4.4 Strengths and limitations

One of the main strengths of this study is the use of a large population-based sample. Tromsø7 has a high attendance (65%), hence the sample is likely a representative sample of the Tromsø population and further a general Norwegian or Nordic population. As presented in Lundblad et al (167), Tromsø municipality has similar demography (age, sex and education) as to the general Norwegian population. Another strength in this study is the measure of the exposure which is a validated FFQ for estimating energy and nutrient intake (167). Also, the outcome retinopathy was obtained from retinal photographs using a standard retinal camera and images were graded using the International Clinical Diabetic Retinopathy and Diabetic Macular Oedema Scales (96). This makes it possible to detect all grades of retinopathy. The grading is a subjective, however the grader received training similar to that of Tromsø6 in which the grade was able to achieve kappa 0.75 compared with another retina specialist (9). Using these standard measures, increases the strength of this study and the obtained result. The use of the standard measurement tools and validated FFQ increases the internal validity of this study and reduction of information bias.

This study was done using a cross sectional study design and investigation into the association between nutrient and retinopathy was accessed at one point in time (184). Hence, the study

design did not take into account temporality and the previous nature of the exposure. However, cross sectional study design is useful in estimating this association and to provide more knowledge on the association between nutrients and retinopathy. Another limitation of this study is that it cannot be used to determine incidence of retinopathy in the study sample.

Confounding bias: Residual confounding is a major challenge when working with any kind of observational data. The confounder being associated with both the exposure and outcome, if not accounted for, can introduces bias into the exposure-disease relation (185). Although this study lists the confounders identified from the existing literature and included them in the model using a DAG, there are likely to be residual confounders. In an attempt to adjust for all potential confounding factors, there is a possibility that the statistical model may have become overfitted. The model could have become too complex and specific to the data at hand, resulting in decreased generalizability to new data (186).

Information bias: A possible drawback of using a FFQ is the potential of information bias. The FFQ is susceptible to recall bias and measurement error, just like any self-reported dietary assessment instrument, which can cause nutrient intake to be misclassified and limit the capacity to identify associations between nutrients and health outcomes (187). This could have led specifically to differential misclassification since the exposure was taken at the same time as the outcome. The self-reporting of the questionnaire on food consumed within a year is prone to misclassification bias even though the questionnaire is validated (185, 187). Covariates especially lifestyle factors amongst others are often subject to reporting biases. Participants will over report good qualities such as physical activity and under report bad qualities such as smoking and alcohol intake. This is also known as the social desirability bias (188). The possible misclassification of participants with retinopathy who have preclinical diabetes, but not diagnosed with diabetes into the population without diabetes (189). Another limitation is the potential loss of information due to collapsing variables into categories during the analysis process or due to anonymized data received. Although categorical variables can simplify the data and make it easier to interpret, they also entail a loss of detailed information that may be relevant to understanding the underlying associations.

Selection bias: The Tromsø7 has 65% attendance among the invitees, however, the 35% non-attendance could introduce selection bias especially if the participants who did not respond are systematically different from the participants who responded. Usually, non-responders of a survey are different in sociodemographic factors and lifestyle factors and these factors can

influence dietary intake which is the exposure on the measured association (190). The exclusion of participants with missing data decreased the power size and could have led to a sample which might not be representative of the general population because the excluded individuals were older, had a higher prevalence of diabetes, and exercised less. Also, there is possibility of healthy participant bias, where more healthy, physically active, and middle-aged individuals participate in a study compared to older individuals who have poorer health. As presented in Lundblad et al (167), the final Tromsø7 FFQ sample recommended to be used in analyses (with exclusions as described in the method section) is considered similar to the total Tromsø7 sample, although with a higher proportion of women, non-smokers, and participants with higher education. Although the invitation to Tromsø7 was sent to all inhabitants of Tromsø 40 years and older, some invitees choose not to participate, sort of a self-selection. In addition, the selection was only inhabitants of Tromsø who were aged 40 years and older, this might not affect the internal validity of the study but could affect the external validity of the study in terms of generalizability to a younger population.

4.5 Further recommendation

The findings of this study highlight several important considerations for future research on the association between nutrient intake and retinopathy. Building upon the results obtained and the limitations identified, the following recommendations are proposed. Prospective cohort studies with long-term follow-up should be conducted to monitor the progression of retinopathy in relation to the consumption of specific nutrients. Retinopathy is a chronic condition that develops over time, and studying its progression in association with nutrient intake requires a comprehensive understanding of the temporal relationship. Prospective cohort studies can provide valuable insights into the long-term effects of nutrient consumption on retinopathy development and progression. Future research efforts should prioritize the establishment of robust relationships between specific nutrients and retinopathy by conducting well-designed cohort studies or RCT. By following a large group of individuals over an extended period, cohort studies can capture detailed dietary information and assess its impact on retinopathy risk. These studies can provide more conclusive evidence on the association between nutrient intake and the incidence or severity of retinopathy. The inclusion of repeated measures of dietary intake in prospective cohort studies is crucial to account for changes in dietary patterns over time. By collecting dietary information at multiple time points, researchers can better understand the influence of varying nutrient intakes on retinopathy outcomes. This longitudinal approach allows for the evaluation of any modifications in nutrient consumption and their

potential effects on the development and progression of retinopathy. To overcome the limitations observed in the current study of minimal variations in the mean % of energy, future research should consider conducting multi-country studies that encompass a broader spectrum of food intake. By examining diverse populations with different dietary patterns, researchers can better understand the influence of cultural and regional variations in nutrient composition on retinopathy risk. This approach can provide valuable insights into the impact of macronutrients on retinopathy, considering the potential differences in dietary habits and nutrient availability across different regions.

4.6 Relevance to the field of public health

A major public health issue of concern is DR because, if left untreated, it can lead to visual impairment. It is a major cause of blindness among people who are working age in developed nations, and this leads to an increase in the disability adjusted life years (DALYs) due to blindness and reduction in the quality of life. Majority of the cases of vision loss can be avoided with adequate management of people with retinopathy, underscoring the urgent need to recognise and understand the risk factors linked it. DR also places a significant economic strain on healthcare systems. In order to lessen the financial burden on healthcare systems, preventive techniques can be created and put into action by understanding the role of nutrient intake in retinopathy. Utilising funds on initiatives that encourage nutritious eating and address certain nutrient-related risk factors can reduce costs, enhance patient outcomes, and better use of healthcare resources.

Retinopathies in general, have implications that go beyond vision loss. It is a sign of vascular health and an indicator to CVDs, which are collectively the leading cause of death globally (103). Morbidity, mortality, and the total burden of disease on society are all strongly affected by CVD. More understanding of the risk factors is needed to create better interventions targeted at reducing the burden of disease from CVDs.

4.7 Conclusion

The aim of this study was to investigate the association between the intake of nutrients and retinopathy in a general adult population. This study found that the intake of PUFA and MUFA was negatively associated with retinopathy among those without diabetes. No association was found between retinopathy and the consumption of any other macronutrients. Although a significant amount of evidence from earlier studies suggests that these nutrients may either be positively or negatively associated with retinopathy, the available epidemiological data did not

provide conclusive evidence of these associations. The result obtained was influenced by numerous factors, including the lack of temporality, therefore, more research investigating this association prospectively is recommended.

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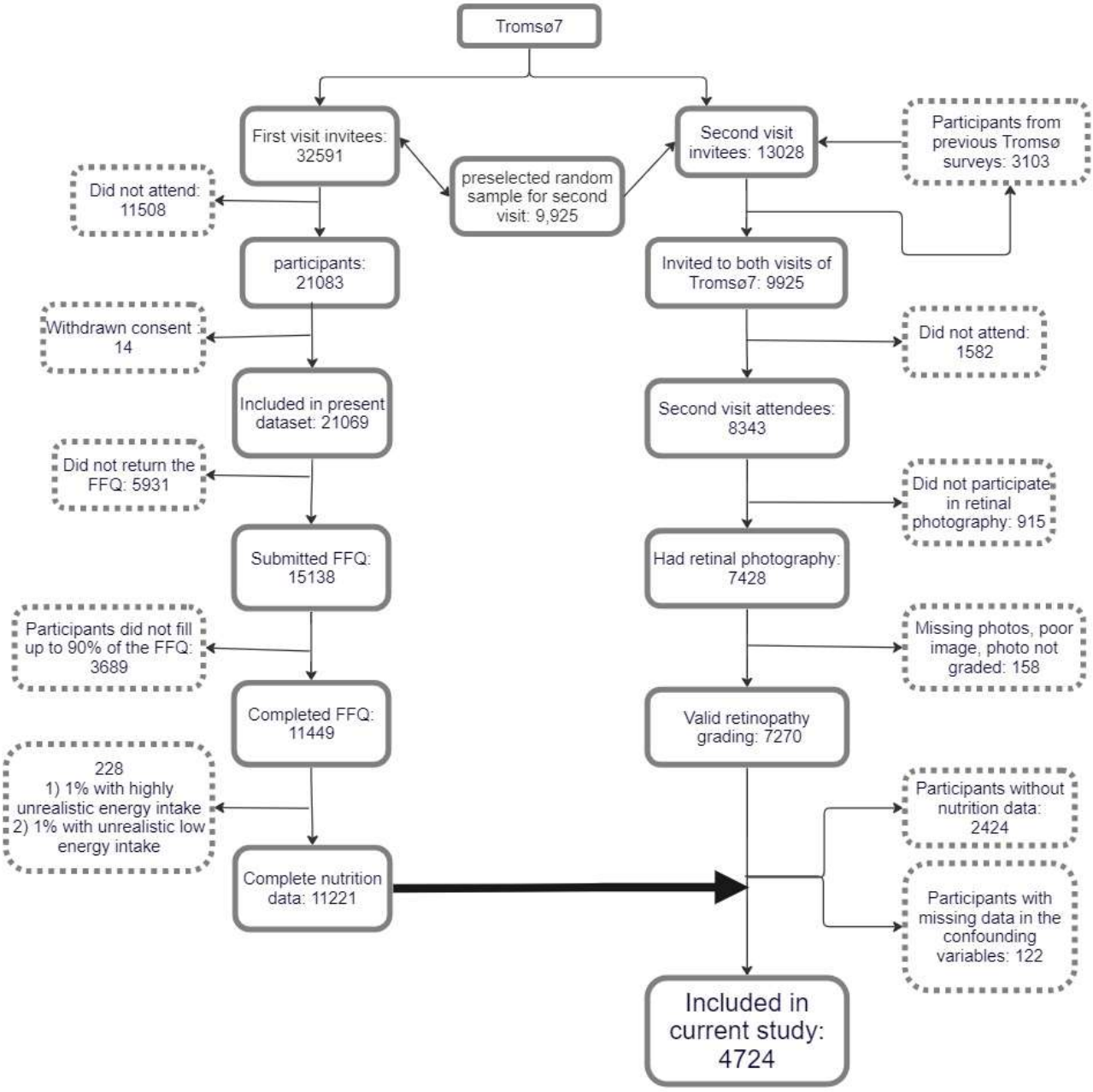
Supplementary materials

Supplementary material 1: Detailed flowchart of participants



Supplementary material 2: Baseline characteristics according to retinopathy status by sex. The Tromsø Study 2015-2016.

	No retinopathy			Retinopathy			
Characteristics	Female	male	*ρ*	female	male	*ρ*	Total
Participants, n (%)	2242(54.09)	1903(45.91)		278(48.01)	301(51.99)		4724(100)
Age group, n (%)							
40-49 years	381(16.99)	300(15.69)		35(12.59)	33(10.96)		749(15.86)
50-59 years	473(21.10)	328(17.27)		43(15.47)	38(12.62)		882(18.67)
60-69 years	925(41.26)	813(42.81)		113(40.65)	140(46.51)		1991(42.15)
70-79 years	416(18.55)	406(21.33)		77(27.70)	85(28.24)		984(20.83)
80+ years	47(2.10)	56(2.94)	.003	10(3.60)	5(1.66)	.35	118(2.5)
BMI (kg/m^2), m (SD)	26.71(4.57)	27.59(3.83)		27.69(5.19)	27.95(4.14)		27.21(4.32)
BMI, n (%)							
Underweight	20(0.89)	2(0.11)		2(0.72)	1(0.33)		25(0.53)
Normal weight	856(38.18)	485(25.49)		94(33.81)	69(22.92)		1504(31.84)
Overweight	900(40.14)	971(51.02)		97(34.89)	158(52.49)		2126(45.00)
Obese	466(20.79)	445(23.38)	<.001	85(30.58)	73(24.25)	<.001	1069(22.63)
Hypertension, n (%)							
No	1036(52.64)	635(33.37)		86(39.93)	73(24.25)		1830(38.74)
Yes	1206(47.36)	1268(66.63)	<.001	192(60.07)	228(75.75)	.07	2894(61.26)
Diabetes, n (%)							
No	2,166(96.61)	1799(94.53)		243(87.41)	245(81.40)		4453(94.26)
Yes	76(3.39)	104(5.47)	.001	35(12.59)	56(18.60)	.05	271(5.74)
Physical activity, n (%)							
Never	65(2.9)	70(3.68)		15(5.40)	16(5.32)		166(3.52)
Less than once a week	182(8.13)	266(13.98)		29(10.43)	45(14.95)		522(11.07)
Once a week	262(11.71)	287(15.08)		39(14.03)	44(14.62)		632(13.4)
2-3 times a week	1017(45.44)	779(40.94)		114(41.01)	126(41.86)		2039(43.16)
Approximately every day	713(31.81)	501(26.33)	<.001	81(29.14)	70(23.26)	.37	1365(28.90)
Level of education, n (%)							
Primary/partly secondary education.	581(25.91)	418(21.97)		98(35.25)	85(28.24)		1182(25.02)
Upper secondary education	607(27.07)	569(29.90)		68(24.46)	94(31.23)		1338(28.32)
Tertiary education, short	397(17.71)	437(22.96)		48(17.27)	75(24.92)		957(20.26)
Tertiary education, long	657(29.30)	479(25.17)	<.001	64(23.02)	47(15.61)	.005	1247(26.40)
Energy (kJ/day), m (SD)	8839(2706)	10336(2976)	<.001	8678(2626)	10404(2877)	<.001	9532.57(2924.11)
Carbohydrate (E%), m (SD)	42.00(6.21)	42.40(6.08)	.04	42.28(5.78)	42.25(6.05)	.96	42.19(6.12)
Dietary fibre (g), m (SD)	27.2(9.12)	26.75(9.17)	.12	26.56(8.32)	26.74(8.53)	.79	26.95(9.06)
Sugar (E%), m (SD)	5.37(3.08)	5.75(3.32)	<.001	5.18(3.05)	5.45(3.43)	.32	5.52(3.21)
Protein (E%), m (SD)	17.86(2.49)	17.48(2.52)	<.001	18.19(2.48)	17.59(2.46)	.00	17.71(2.51)

Total fat, (E%), m (SD)	34.54(5.65)	33.88(5.75)	<.001	34.30(5.5)	33.93(6.01)	.44	34.22(5.71)
Saturated fat (E%), m (SD)	12.57(2.69)	12.34(2.71)	.005	12.82(2.86)	12.46(2.54)	.11	12.49(2.7)
Trans-fat (E%), m (SD)	.32(.13)	.29(.12)	<.001	.33(.14)	.29(.12)	.00	.31(.13)
MUFA (E%), m (SD)	12.79(2.68)	12.42(2.57)	<.001	12.49(2.63)	12.42(3)	.78	12.6(2.66)
PUFA (E%), m (SD)	5.98(1.51)	6.09(1.59)	.04	5.82(1.45)	6.02(1.67)	.13	6.02(1.55)
Alcohol (E%), m (SD)	2.98(3.42)	4.05(4.38)	<.001	2.62(3.34)	4.03(4.27)	.00	3.46(3.92)

BMI: Body mass index, PUFA: Polyunsaturated fat, MUFA: Monounsaturated.

Categorical variables are presented as proportions.

Continuous variables are presented as means with standard deviation (SD) in parenthesis.

Nutrients are presented as mean % of energy intake (E%) except dietary fibre which is presented as mean of the intake in grams(g).

Supplementary material 3: Baseline characteristics according to retinopathy status by diabetes status. The Tromsø Study 2015-2016.

	No retinopathy			**Retinopathy**			
Characteristics	No diabetes	Diabetes	***ρ***	No diabetes	Diabetes	***ρ***	Total
Participants, n (%)	3965(95.66)	180(4.34)		488(84.22)	91(15.72)		4724(100)
Age group, n (%)							
40-49 years	675(17.02)	6(3.33)		58(11.89)	10(10.99)		749(15.86)
50-59 years	778(19.62)	23(12.78)		74(15.16)	7(7.69)		882(18.67)
60-69 years	1650(41.61)	88(48.89)		215(44.06)	38(41.76)		1991(42.15)
70-79 years	768(19.37)	54(30.00)		127(26.02)	35(38.46)		984(20.83)
80+ years	94(2.37)	9(5.00)	<.001	14(2.87)	1(1.10)	.08	118(2.5)
Sex, n (%)							
Female	2166(54.63)	76(42.22)		243(49.80)	35(38.46)		2520(53.34)
Male	1799(45.37)	104(57.78)	.001	245(50.20)	56(61.54)	.05	2204(46.66)
BMI (kg/m^2), m (SD)	26.99(4.19)	26.79(4.99)		27.36(4.38)	30.32(5.35)		27.21(4.32)
BMI, n (%)							
Underweight	21(0.53)	1(0.56)		3(0.61)	0(0.00)		25(0.53)
Normal weight	1314(38.18)	27(15.00)		152(31.15)	11(12.09)		1504(31.84)
Overweight	1801(40.14)	70(38.89)		218(44.67)	37(40.66)		2126(45.00)
Obese	829(20.79)	82(45.56)	<.001	115(23.57)	43(47.25)	<.001	1069(22.63)
Hypertension, n (%)							
No	1629(41.08)	42(23.33)		146(29.92)	13(14.29)		1830(38.74)
Yes	2336(58.92)	138(76.67)	<.001	342(70.08)	78(85.71)	.002	2894(61.26)
Physical activity, n (%)							
Never	118(2.98)	17(9.44)		21(4.30)	10(10.99)		166(3.52)
Less than once a week	411(10.37)	37(20.56)		61(12.50)	13(14.29)		522(11.07)
Once a week	528(13.32)	21(11.67)		67(13.73)	16(17.58)		632(13.4)
2-3 times a week	1732(43.68)	67(37.22)		203(41.60)	37(40.66)		2039(43.16)
Approximately every day	1176(29.66)	38(21.11)	<.001	136(27.87)	15(16.48)	.02	1365(28.90)
Level of education, n (%)							
Primary/partly secondary education.	933(25.53)	66(36.67)		150(30.74)	33(36.26)		1182(25.02)
Upper secondary education	1118(28.20)	58(32.22)		135(27.66)	27(29.67)		1338(28.32)
Tertiary education, short	802(20.23)	32(17.78)		106(21.72)	17(18.68)		957(20.26)
Tertiary education, long	1112(28.05)	24(13.33)	<.001	97(19.88)	14(15.38)	.57	1247(26.40)
Energy (kJ/day), m (SD)	9543(2923)	9195(3042)	.12	9527(2894)	9836(2861)	.35	9532.57(2924.11)
Carbohydrate (E%), m (SD)	42.24(6.13)	40.78(6.28)	.12	42.66(5.68)	40.18(6.70)	.96	42.19(6.12)
Dietary fibre (g), m (SD)	27.01(9.14)	26.57(9.20)	.53	26.50(8.32)	27.44(8.93)	.79	26.95(9.06)
Sugar (E%), m (SD)	5.59(3.21)	4.57(2.84)	<.001	5.49(3.29)	4.43(2.91)	.004	5.52(3.21)
Protein (E%), m (SD)	17.65(2.50)	18.32(2.66)	<.001	17.79(2.46)	18.34(2.61)	.05	17.71(2.51)
Total fat, (E%), m (SD)	34.17(5.62)	35.64(7.24)	.001	33.67(5.50)	36.46(6.62)	<.001	34.22(5.71)

Saturated fat (E%), m (SD)	12.45(2.65)	12.90(3.61)	.03	12.63(2.72)	12.67(2.57)	.88	12.49(2.7)
Trans-fat (E%), m (SD)	0.31(0.13)	0.30(0.16)	.59	.31(.13)	.28(.12)	.03	.31(.13)
MUFA (E%), m (SD)	12.60(2.68)	13.11(3.06)	.001	12.20(2.52)	13.85(3.84)	<.001	12.6(2.66)
PUFA (E%), m (SD)	6.01(1.54)	6.39(1.74)	.002	5.77(1.48)	5.75(1.79)	<.001	6.02(1.55)
Alcohol (E%), m (SD)	3.50(3.93)	2.77(3.70)	.01	3.48(4.01)	2.65(3.30)	.06	3.46(3.92)

BMI: Body mass index, PUFA: Polyunsaturated fat, MUFA: Monounsaturated.

Categorical variables are presented as proportions.

Continuous variables are presented as means with standard deviation (SD) in parenthesis.

Nutrients are presented as mean % of energy intake (E%) except dietary fibre which is presented as mean of the intake in grams(g).

Appendix

Appendix 1: Invitation letter from The Tromsø Study 2015-2016


Forespørsel
om deltakelse i
Tromsøundersøkelsen
Hva er Tromsøundersøkelsen?
Tromsøundersøkelsen er en folkehelseundersøkelse. Formålet er å samle inn opplysninger til forskning som gir økt kunnskap om helse og sykdom, og hvordan folke-helsen kan forbedres gjennom forebygging og behandling.
Tromsøundersøkelsen startet i 1974 med bakgrunn i den høye forekomsten av hjerte -og karsykdom i Nord-Norge. Siden den gang er undersøkelsen gjennomført med 6-7 års mellomrom og dette er den sjuende runden.
Ved å delta bidrar du til viktig forskning om forekomst, forebygging og behandling av sykdom, hva som fremmer god helse, og hva som er årsak til helseproblemer.
Ditt bidrag teller!

Hvorfor spør vi deg?

Alle innbyggere i Tromsø kommune fra 40 år og oppover spørres om å delta. I tillegg inviterer vi ca.1000 personer i alderen 21-25 år. Hver deltaker er like viktig, enten du er ung eller gammel, frisk eller syk.

Sammen med denne informasjonsbrosjyren finner du en invitasjon med praktiske opplysninger om undersøkelsen.

Det er gratis å delta i Tromsøundersøkelsen. Trenger du videre undersøkelse eller oppfølging av fastlegen eller spesialisthelsetjenesten, betaler du vanlig egenandel.

Slik foregår undersøkelsen

Alle deltakere inviteres til en hovedundersøkelse som omfatter spørreskjema, intervju, blodprøver og undersøkelser. Et helt tilfeldig utvalg av deltakere inviteres tilbake til en spesialundersøkelse som omfatter flere prøver og mer omfattende undersøkelser. Alle undersøkelsene gjennomføres av helsepersonell.

Tilbakemelding

Noen uker etter undersøkelsen får du et brev med noen resultater, det vil si høyde, vekt, BMI, hemoglobin, blodtrykk, kolesterolnivå og om du har diabetes. Det gis ikke rutinemessig tilbakemelding om resultater av andre blodprøver eller målinger. Dersom prøveresultatet viser at det er nødvendig med oppfølging av lege eller henvisning til spesialist, vil du få råd om det. Ved behov for henvisning til spesialist, sørger vi for å sende henvisning.

Du kan reservere deg mot å få vite resultatene av prøvene dine. Men hvis et prøveresultat krever rask legebehandling, vil du likevel bli kontaktet.

Du vil også få informasjon om undersøkelsen underveis gjennom aviser, sosiale medier (Facebook, Twitter m.m) samt på arrangementer som "Lørdagsuniversitetet" og "Forskningsdagene".

Frivillig deltakelse

Det er frivillig å delta i Tromsøundersøkelsen.
Om du sier ja til å delta, kan du når som helst trekke tilbake samtykket.

Hva omfatter den sjuende Tromsøundersøkelsen?

Hva skal vi forske på?

I denne runden av Tromsøundersøkelsen er det mer enn 50 prosjekter som skal forske på forekomst, forebygging og behandling av folkehelseproblemer.

Det skal blant annet forskes på hjerte- og karsykdommer, kreft, lungesykdommer, aldring og demens, fedme, diabetes, legemiddelbruk, psykisk helse, kronisk smerte, tannhelse, muskel- og skjelettplager, risikofaktorer som alkohol, fysisk aktivitet og kosthold, nyrer og urinveier, hudproblemer, miljøgifter, infeksjoner og antibiotikaresistens, nervesystemet, sosial ulikhet, samspill mellom arv og miljø, søvn og bruk av helsetjenester.

Du finner mer informasjon om forskningen på vår internettside, www.tromsoundersokelsen.no

Spørreskjema

Deltakernes informasjon om egen helse er en svært viktig del av Tromsøundersøkelsen. Vi ber deg derfor fylle ut to spørreskjema. Alle spørsmål kan besvares på nett. Det ene skjemaet er vedlagt i papirform, hvis du foretrekker det. Fyll det gjerne ut før du møter opp så sparer du tid under undersøkelsen. Hvis du trenger assistanse vil personalet hjelpe deg på undersøkelsen hvor det også er satt opp egne datamaskiner til dette.

Utfylte svar i spørreskjema er like viktig for forskningen som resultater fra blodprøver og kliniske undersøkelser.

Du kan delta på Tromsøundersøkelsen selv om du ikke ønsker å være med på alle deler av undersøkelsen.

Hovedundersøkelsen

Helsepersonell veileder deg gjennom undersøkelsen som varer ca. en time hvis du har fylt ut spørreskjemaene på forhånd. Du får også time til spesialundersøkelsen hvis du er valgt ut til denne.

Vi starter med noen enkle spørsmål knyttet til undersøkelsene du skal gjennomføre. Videre måler vi høyde, vekt, hofte- og livvidde, blodtrykk og puls.

Det tas deretter prøver og gjøres noen kliniske undersøkelser:

Blodprøve. Det tas blodprøver til bruk for forskning som samlet er mye mindre enn det en blodgiver gir. Det fryses ned prøver til bruk for senere analyser og forskning. Arvestoff (DNA/RNA) vil bli lagret til bruk for forskning.

Bakterieprøve fra nese og hals for å se etter gule stafylokokker, en bakterie som normalt finnes på hud og slimhinner hos mennesker, men som i enkelte tilfeller kan forårsake alvorlige infeksjoner. Prøvene tas med en fuktet vattpensel.

Spyttprøver til bruk for forskning knyttet til tannhelse, virusinfeksjon og kreft.

Smertefølsomhet måles med to metoder. Først holder du hånden i kaldt vann i opptil 90 sekunder,deretter får du en blodtrykksmansjett plassert rundt leggen som blåses opp. Underveis angir du hvor mye smerte du opplever, og kan avbryte testene når som helst hvis det blir for ubehagelig.

Tannsjekk som omfatter et røntgenbilde av kjeven, registrering av hull i tennene og betennelsessykdom i tannkjøttet.

Fysisk aktivitet og kosthold. Utvalgte deltakere blir bedt om å registrere fysisk aktivitet ved bruk av aktivitetsmåler og registrering av kosthold i en periode.

Du får også utdelt utstyr for innlevering av urin- og avføringsprøve hvis du er valgt ut til spesialundersøkelsen.

Spesialundersøkelsen

Et tilfeldig utvalg av deltakere inviteres til spesialundersøkelsen som gjennomføres noen uker etter hovedundersøkelsen. Denne varer totalt ca. 2 timer, avhengig av hvor mange deler du blir spurt om å være med på.

Ved oppmøte vil urinprøvene samles inn, og det tas noen nye blodprøver. Deler av blodprøvene fryses ned for senere forskning beskrevet i denne brosjyren.

Videre inviteres du til én eller flere av disse undersøkelsene:

EKG er en registrering av hjerterytmen som også kan gi informasjon om hjertesykdom. Ved registrering festes ledninger til kroppen.

Kognitiv funksjon testes ved hjelp av enkle spørsmål knyttet til gjenkjenning av ord, kopling av symboler og tall samt grad av fingerbevegelighet.

Fysisk funksjon undersøkes ved å teste balanse, gange og gripestyrke.

Ultralyd av halspulsåre gjøres for å se etter forkalkninger og innsnevringer av årene. Undersøkelsen kartlegger også blodforsyningen til hjernen.

Fotografering av øyebunnen gir bilder som både sier noe om synet og om tilstanden til blodkarene i kroppen. Det gis en øyendråpe i hvert øye en tid før fotografering for at pupillene skal utvide seg. Dette kan svi noe og synet kan forbigående bli noe uklart. Effekten går gradvis over, og er borte etter en time. I tillegg gjøres det en enkel synstest som du får svar på umiddelbart.

Lungefunksjonen testes ved at du puster så hardt du klarer gjennom et munnstykke. Hvor mye luft som blåses ut pr. sekund, er et mål på lungefunksjonen din. I tillegg vil det gjøres lydopptak av lungelyder og hjertelyder.

Måling av beintetthet. Ved hjelp av ultralyd foretas det beintetthetsmåling som brukes til å undersøke risiko for beinskjørhet og brudd.

Ultralyd av hjertet gjøres for å undersøke hjertets form og funksjon.

Videre bruk av opplysninger og prøver i forskning

Personvern

All informasjon du gir til Tromsøundersøkelsen behandles med respekt for personvern og privatliv, og i samsvar med lover og forskrifter.
Alle medarbeidere som jobber med undersøkelsen har taushetsplikt. Opplysningene som samles inn skal bare brukes til godkjente forskningsformål. Det vil ikke være mulig å identifisere deg når resultatene av forskningen publiseres.

UiT Norges arktiske universitet ved universitetsdirektøren er ansvarlig for behandlingen av personopplysninger. Tromsøundersøkelsen har konsesjon fra Datatilsynet. Regional komité for medisinsk og helsefaglig forskningsetikk i Nord-Norge (REK nord) har gjort en etisk og helsefaglig vurdering av undersøkelsene som gjennomføres, samt godkjent innsamlingen av prøver.

Hvilke data lagres i Tromsøundersøkelsen?

I Tromsøundersøkelsen lagres opplysninger gitt av deltakere i de forskjellige rundene av Tromsøundersøkelsen. Det lagres også opplysninger om kreftdiagnoser og dødsårsaker fra Kreftregisteret og Dødsårsaksregisteret.
For deltakere som har eller får diagnoser innen hjerte- og karsykdom, diabetes og beinbrudd, innhentes opplysninger fra sykejournalen i spesialist- og primærhelsetjenesten som er nødvendig for å kvalitetssikre aktuelle diagnoser. Dette for å sikre forskning av høy kvalitet. Tilsvarende vil også kunne bli aktuelt for andre sykdommer det forskes på i Tromsøundersøkelsen.

Hvordan lagres dine opplysninger og prøver?

Alle opplysningene og prøvene lagres uten navn og fødselsnummer.

En kode knytter deg til dine opplysninger og prøver. Det er kun noen få autoriserte personer som kan finne tilbake til deg gjennom en egen kodenøkkel.

De biologiske prøvene lagres i godkjent forskningsbiobank ved Institutt for samfunnsmedisin, UiT. Leder av Tromsøundersøkelsen er ansvarlig for biobanken. Den er registrert i Folkehelseinstituttets Biobankregister (nr 2397). Det biologiske materialet kan bare brukes etter godkjenning fra REK.

Utlevering av opplysninger og prøver til forskere

Hvis du sier ja til å delta i studien, samtykker du til at dine opplysninger og prøver kan brukes videre i forskning på ubestemt tid. Medisinsk forskning forandrer seg hele tiden, og i fremtiden kan data bli brukt i forskningsprosjekter forutsatt at det er i samsvar med gjeldende lover og forskrifter.

Alle forskningsprosjekter som får data fra Tromsøundersøkelsen må være i samsvar med lover og forskrifter. Prosjektleder må tilhøre en kompetent forskningsinstitusjon. Den enkelte forsker vil kun få tilgang til personidentifiserende opplysninger etter å ha innhentet nødvendige godkjenninger fra REK, og/eller Datatilsynet.

I noen forskningsprosjekter kan prøver og avidentifiserte opplysninger bli utlevert til andre land. Det vil skje i en slik form at våre utenlandske samarbeidspartnere ikke kan knytte prøvene opp mot deg som person.

I noen prosjekter kan det bli aktuelt å kontakte deg igjen for å samle inn flere data, f.eks. ved spørreskjema, intervju eller kliniske undersøkelser. Du vil da få ny informasjon og bes om nytt samtykke til det konkrete prosjektet.

Ved å delta i Tromsøundersøkelsen bidrar du til viktig forskning på sykdom og helse, oppbygging av fagmiljøer og bedre pasientbehandling.

Sammenstilling med andre registre

I noen forskningsprosjekter vil opplysninger om deg kunne bli sammenstilt med:

Opplysninger du har gitt i tidligere runder av Tromsøundersøkelsen hvis du har deltatt i Tromsøundersøkelsen før.

Opplysninger fra barn, søsken, foreldre og besteforeldre som har deltatt i Tromsøundersøkelsen.

Opplysninger om deg i nasjonale helseregistre som Reseptregisteret, Medisinsk fødselsregister, Kreftregisteret, Norsk pasientregister, Hjerte- og karregisteret, Dødsårsaksregisteret, infeksjonsregistre og andre nasjonale sykdoms- og kvalitetsregistre.

Helseopplysninger om deg fra primær- og spesialisthelsetjenesten.

Opplysninger om sosiale forhold som arbeid, utdanning, inntekt, boforhold osv. fra registre hos bl.a. Statistisk sentralbyrå og NAV.

Slike sammenstillinger krever som regel forhåndsgodkjenning av offentlige instanser, som REK og/eller Datatilsynet.

Rett til innsyn og sletting av dine 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 også rett til å få korrigert eventuelle feil i 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 er brukt i vitenskapelige artikler.

Finansiering

Tromsøundersøkelsen er finansiert av UiT Norges arktiske universitet, Helse Nord RHF, Universitetssykehuset Nord-Norge (UNN) samt ulike forskningsfond.

Forsikring

Deltakere i Tromsøundersøkelsen er forsikret gjennom Norsk Pasientskadeerstatning.

Samtykke til deltakelse i studien

Hvis du vil delta i den sjuende Tromsøundersøkelsen, må du gi skriftlig samtykke ved oppmøte. Personalet vil gi mer informasjon og svare deg dersom du har spørsmål i forbindelse med samtykket.

Du kan når som helst trekke tilbake samtykket ditt.


Dine svar bidrar til bedre folkehelse for våre kommende generasjoner
Her finner du oss:
Heiloveien 6 (tidligere Langnes legesenter)
9015 Tromsø
Telefon 77 62 07 00
Epost tromso7@uit.no
Nettside www.tromsoundersokelsen.no
Tromsø-undersøkelsen
UNIVERSITETET I TROMSØ

Appendix 2: Food frequency questionnaire from The Tromsø Study 2015-2016

UiT
NORGES
ARKTISKE
UNIVERSITET

Tromsø-
undersøkelsen

Spørreskjema om kosthold

Utfylte svar i spørreskjema er like viktig for Tromsøundersøkelsen som resultater fra blodprøver og kliniske undersøkelser.

Forskere har funnet ut at kosthold er den risikofaktoren som fører til flest tapte leveår og dager med sykdom eller skade i Norge. Det finnes lite informasjon om kostholdet i Nord-Norge. Vi ber deg derfor fylle ut dette spørreskjemaet som vil gi viktige data for forskningen.

Vi spør her om matvanene dine og hvor ofte du vanligvis spiser og drikker ulike typer mat og drikke. Vi er klar over at kostholdet varierer fra dag til dag, men prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine. Ha det siste året i tankene når du fyller ut skjemaet. Der du er usikker anslår du svaret ditt.

Alle svar lagres og behandles uten navn og fødselsnummer, i samsvar med lover og forskrifter.

Dersom du fyller ut skjemaet hjemme, bruk vedlagte svarkonvolutt.

Takk for at du tar deg tid til å fylle ut skjemaet.

UiO : Det medisinske fakultet

UNIVERSITETET I TROMSØ

Skjemaet skal lese av en maskin og det er derfor viktig at du setter tydelige kryss i rutene. Bruk blå eller sort kulepenn.

☒ Riktig markering i rutene er slik
■ Ved feil markering, fyll hele ruten slik

Har du spørsmål om utfyllingen av skjemaet kan du ta kontakt med personalet på undersøkelsen eller sende e-post til: tromso7@ism.uit.no

Eksempel

Kari Normann spiser daglig 5 skiver brød og ett grovt knekkebrød. Hun spiser vanligvis kneippbrød, men i helgene spiser hun som oftest loff. Spørsmål 1 fyller hun ut slik:

1. Hvor mye brød pleier du å spise?
Legg sammen det du bruker til alle måltider i løpet av en dag.
(1/2 rundstykke = 1 skive, 1 baguett = 4 skiver, 1 ciabatta = 2 skiver)

	Aldri/ sjelden	Antall skiver pr. dag ½	1	2	3	4	5	6	7	8	9	10	11	12+
Fint brød (loff, baguetter, fine rundstykker, ciabatta)	☐	☐	☒	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Mellomgrovt brød (helkornbrød, kneipp, grove rundstykker)	☐	☐	☐	☐	☐	☒	☐	☐	☐	☐	☐	☐	☐	☐
Grovt brød (mer enn 50 % sammalt, mørkt rugbrød)	☒	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Fint knekkebrød (kavring)	☒	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Grovt knekkebrød (grov skonrok)	☐	☐	☒	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Sum skiver pr. dag = 6

Antall skiver pr. uke: 6 x 7 = 42. Tallet brukes i spørsmål 4.

Prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine.
Ha det siste året i tankene når du fyller ut.

1. Hvor mye brød pleier du å spise?

Legg sammen det du bruker til alle måltider i løpet av en dag.
(1/2 rundstykke = 1 skive, 1 baguett = 4 skiver, 1 ciabatta = 2 skiver)

	Aldri/ sjelden	Antall skiver pr. dag ½	1	2	3	4	5	6	7	8	9	10	11	12+
Fint brød (loff, baguetter, fine rundstykker, ciabatta)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Mellomgrovt brød (helkornbrød, kneipp, grove rundstykker)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Grovt brød (mer enn 50 % sammalt, mørkt rugbrød)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Fint knekkebrød (kavring)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Grovt knekkebrød (grov skonrok)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Sum skiver pr. dag = _____

Antall skiver pr. uke: _____ x 7 = _____. Tallet brukes i spørsmål 4.
(sum skriver pr. dag)

2. Hva pleier du å smøre på brødet?

Legg sammen det du bruker på skivene i løpet av en uke.
(1/2 rundstykke = 1 skive, 1 baguett = 4 skiver, 1 ciabatta = 2 skiver)

	Aldri/ sjelden	Antall skiver pr. uke 1-5	6-14	15-21	22-28	29-35	36-42	43-49	50-56	57+
Smør (meierismør)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Bremykt	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Brelett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Myk margarin (Soft Flora, Soft Ekstra)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Vita	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Soft Light, Vita Lett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Melange	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Annen margarin	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Olivenolje, annen olje på brød	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Majones, remulade på brød	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

3. Hvis du bruker smør/margarin på brødet, hvor mye bruker du?

	Antall skiver ½	1	2	3	4	5 eller flere
En porsjonspakke smør/margarin på 12 g rekker til antall skiver:	☐	☐	☐	☐	☐	☐

Prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine.
Ha det siste året i tankene når du fyller ut.

4. Hvilke typer pålegg spiser du?

	Aldri/ sjelden	Antall skiver pr. uke								
		1	2-3	4-5	6-7	8-12	13-18	19-24	25-30	31+
Brunost/prim	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lett/mager brunost/prim	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hvitost (eks. Norvegia, Gulost)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lett/mager hvitost	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Dessertost (eks. Brie, Gräddost, blåmuggoster)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Smøreost (eks. kremost, Philadelfia)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lett/mager smøreost	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Leverpostei	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Mager leverpostei	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Servelat	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kokt skinke, lettservelat, kalkunpålegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Salami, fårepølse, spekepølse	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kaviar	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Svolværpostei, Lofotpostei	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Makrell i tomat	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Røkt, gravet laks/ørret	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sardiner, sursild, ansjos	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Tunfisk	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Reker, krabbe	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Egg (kokt, stekt, eggerøre)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Syltetøy, marmelade	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lett syltetøy, frysetøy	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Peanøttsmør	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sjokolade-, nøttepålegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Annet søtt pålegg (eks. honning, Sunda, sirup)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Cottage cheese	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Majonessalat (eks. italiensk salat)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Majonessalat lett (eks. lett italiensk salat)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Frukt som pålegg (eks. banan, eple)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Grønnsaker som pålegg (eks. agurk, tomat)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine.
Ha det siste året i tankene når du fyller ut.

5. Frokostgryn

Svar enten per måned eller per uke.

	Aldri/ sjelden	Gang pr. måned 1	2	3	eller 1	Gang pr. uke 2-3	4-5	6-7	8+	Mengde pr. gang 1	1½	2	3+
Havregrøt	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl) ☐	☐	☐	☐
Havregryn, 4-korn	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl) ☐	☐	☐	☐
Mysli, søtet (eks. Solfrokost)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl) ☐	☐	☐	☐
Mysli, usøtet (eks. Go'Dag)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl) ☐	☐	☐	☐
Cornflakes	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl) ☐	☐	☐	☐
Honnikorn/Frosties/Chocofrokost	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl) ☐	☐	☐	☐
All Bran, Weetabix, Havrefras o.l.	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl) ☐	☐	☐	☐
Puffet ris, havrenøtter	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl) ☐	☐	☐	☐
	Aldri/ sjelden	Gang pr. måned 1	2	3	eller 1	Gang pr. uke 2-3	4-5	6-7	8+	Mengde pr. gang 1	1½	2	3+
Syltetøy til frokostgryn, grøt	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss) ☐	☐	☐	☐
Sukker til frokostgryn, grøt	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts) ☐	☐	☐	☐

6. Melk (Husk også å ta med melk du bruker på frokostgryn, grøt og dessert)

(1 glass = 2 dl)

	Aldri/ sjelden	Antall glass pr. dag ½	1	2	3	4	5	6	7+
Helmelk, kefir, kultur	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lettmelk	☐	☐	☐	☐	☐	☐	☐	☐	☐
Ekstra lettmelk	☐	☐	☐	☐	☐	☐	☐	☐	☐
Skummet melk, skummet kultur	☐	☐	☐	☐	☐	☐	☐	☐	☐
Biola/Cultura naturell	☐	☐	☐	☐	☐	☐	☐	☐	☐
Biola/Cultura med bær/frukt	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sjokolademelk, jordbærmelk	☐	☐	☐	☐	☐	☐	☐	☐	☐
Drikkeyoghurt	☐	☐	☐	☐	☐	☐	☐	☐	☐

7. Yoghurt (Husk å ta med yoghurt du bruker til frokostgryn)

Svar enten per måned eller per uke.

	Aldri/ sjelden	Gang pr. måned 1	2	3	eller 1	Gang pr. uke 2-3	4-5	6-7	8+	Beger pr. gang ½	1	2	3+
Yoghurt naturell (125 g)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Yoghurt med frukt (125 g)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Go'morgen yoghurt m/mysli	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lettyoghurt med frukt (125 g)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lettyoghurt m/mysli	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine.
Ha det siste året i tankene når du fyller ut.

8. Kalde drikker

Svar enten per uke eller per dag, <1 betyr sjeldnere enn 1 gang. Merk at porsjonsenhetene er forskjellige, 1/5 liter tilsvarer ett glass (2 dl), mens 1/3 liter tilsvarer 0,33 liter glassflaske/boks.

	Aldri/ sjelden	Gang pr. uke <1	1-2	3-4	5-6	eller Gang pr. dag 1	2	3	4+	Mengde pr. gang				
Vann (springvann)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(glass)	1 ☐	2 ☐	3 ☐	4+ ☐
Flaskevann med/uten kullsyre (eks. Farris, Imsdal)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(liter)	1/5 ☐	1/3 ☐	½ ☐	1+ ☐
Appelsinjuice	☐	☐	☐	☐	☐	☐	☐	☐	☐	(glass)	1 ☐	2 ☐	3 ☐	4+ ☐
Eplejuice, annen juice	☐	☐	☐	☐	☐	☐	☐	☐	☐	(glass)	1 ☐	2 ☐	3 ☐	4+ ☐
Eplenektar, annen nektar	☐	☐	☐	☐	☐	☐	☐	☐	☐	(glass)	1 ☐	2 ☐	3 ☐	4+ ☐
Saft med sukker	☐	☐	☐	☐	☐	☐	☐	☐	☐	(glass)	1 ☐	2 ☐	3 ☐	4+ ☐
Saft, kunstig søtet	☐	☐	☐	☐	☐	☐	☐	☐	☐	(glass)	1 ☐	2 ☐	3 ☐	4+ ☐
Brus med sukker	☐	☐	☐	☐	☐	☐	☐	☐	☐	(liter)	1/5 ☐	1/3 ☐	½ ☐	1+ ☐
Brus, kunstig søtet	☐	☐	☐	☐	☐	☐	☐	☐	☐	(liter)	1/5 ☐	1/3 ☐	½ ☐	1+ ☐
Iste med sukker	☐	☐	☐	☐	☐	☐	☐	☐	☐	(liter)	1/5 ☐	1/3 ☐	½ ☐	1+ ☐
Iste, kunstig søtet	☐	☐	☐	☐	☐	☐	☐	☐	☐	(liter)	1/5 ☐	1/3 ☐	½ ☐	1+ ☐
Alkoholfritt øl (eks. Vørterøl, Munkholm)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(liter)	1/5 ☐	1/3 ☐	½ ☐	1+ ☐

9. Alkoholholdige drikker

Svar enten pr. måned eller pr. uke. Merk at porsjonsenhetene er forskjellige, 1/5 liter tilsvarer ett glass (2 dl), mens 1/3 liter tilsvarer 0,33 liter glassflaske/boks.

	Aldri/ sjelden	Gang pr. måned 1	2	3	eller Gang pr. uke 1	2-3	4-5	6-7	Mengde pr. gang						
Øl, sterk øl, pils	☐	☐	☐	☐	☐	☐	☐	☐	(liter)	1/3 ☐	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Lettøl	☐	☐	☐	☐	☐	☐	☐	☐	(liter)	1/3 ☐	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Rusbrus, Cider m/alkohol	☐	☐	☐	☐	☐	☐	☐	☐	(liter)	1/5 ☐	1/3 ☐	½ ☐	1 ☐	1½ ☐	2+ ☐
Rødvin	☐	☐	☐	☐	☐	☐	☐	☐	(vinglass)	1 ☐	2 ☐	3 ☐	4 ☐	5 ☐	6+ ☐
Hvitvin	☐	☐	☐	☐	☐	☐	☐	☐	(vinglass)	1 ☐	2 ☐	3 ☐	4 ☐	5 ☐	6+ ☐
Hetvin (portvin, sherry o.l.)	☐	☐	☐	☐	☐	☐	☐	☐	(1 glass = 4cl)	1 ☐	2 ☐	3 ☐	4 ☐	5 ☐	6+ ☐
Brennevin, likør	☐	☐	☐	☐	☐	☐	☐	☐	(1 dram = 4cl)	1 ☐	2 ☐	3 ☐	4 ☐	5 ☐	6+ ☐
Blandede drinker, cocktail	☐	☐	☐	☐	☐	☐	☐	☐	(drink)	1 ☐	2 ☐	3 ☐	4 ☐	5 ☐	6+ ☐

Prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine.
Ha det siste året i tankene når du fyller ut.

10. Varme drikker

Svar enten per uke eller per dag, < 1 betyr sjeldnere enn 1 gang.

	Aldri/ sjelden	Gang pr. uke <1	1-2	3-4	5-6	eller 1	Gang pr. dag 2	3	4+	Mengde pr. gang (kopp) 1	2	3-4	5-6	7-8	9+
Kaffe - kokt og presskanne ***1 kopp = 2 dl***	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kaffe - traktet, filter ***1 kopp = 2 dl***	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kaffe - pulver (instant) ***1 kopp = 2 dl***	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Espresso ***1 kopp = 0,3 dl***	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Caffe latte ***1 kopp = 3 dl***	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Cappucino ***1 kopp = 3 dl***	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kakao/varm sjokolade ***1 kopp = 2 dl***	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sort te (eks. Earl Grey, solbær) ***1 kopp = 2 dl***	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Grønn te ***1 kopp = 2 dl***	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Urtete (eks. nype, kamille, Rooibos) ***1 kopp = 2 dl***	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

	Bruker ikke	Antall pr. kopp ½	1	2	3	4+
Sukker til te (ts/sukkerbit)	☐	☐	☐	☐	☐	☐
Sukker til kaffe (ts/sukkerbit)	☐	☐	☐	☐	☐	☐
Sukketter til te (stk)	☐	☐	☐	☐	☐	☐
Sukketter til kaffe (stk)	☐	☐	☐	☐	☐	☐
Melk/fløte til te (ss)	☐	☐	☐	☐	☐	☐
Melk/fløte til kaffe (ss)	☐	☐	☐	☐	☐	☐

Prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine.
Ha det siste året i tankene når du fyller ut.

11. Middagsretter

Vi spør både om middagsmåltidene og det du spiser til andre måltider. Legg til slutt sammen hvor mange retter per måned du har merket av for å se om summen virker sannsynlig.

	Aldri/ sjelden	Gang pr. måned 1	2	3	4	5-6	7-8	9+	Mengde pr. gang					
Kjøtt/kjøttretter														
Kjøttpølse av storfe/svin	☐	☐	☐	☐	☐	☐	☐	☐	(pølse)	½ ☐	1 ☐	1½ ☐	2 ☐	3+ ☐
Kjøttpølse av storfe/svin, lett/mager	☐	☐	☐	☐	☐	☐	☐	☐	(pølse)	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Kjøttpølse av kylling/kalkun	☐	☐	☐	☐	☐	☐	☐	☐	(pølse)	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Grillpølse/wienerpølse av storfe/svin	☐	☐	☐	☐	☐	☐	☐	☐	(pølse)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Grillpølse/wienerpølse av kylling/kalkun	☐	☐	☐	☐	☐	☐	☐	☐	(pølse)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Hamburger (m/brød)	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Karbonade	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Kjøttkaker, medisterkaker, kjøttpudding	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Kjøttsaus, gryterett med kjøttdeig	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Taco (tacoskjell med kjøtt og salat)	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Tortilla lefse (med kjøtt og salat)/ wrap	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Kebab	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	½ ☐	1 ☐	1½ ☐	2 ☐	3+ ☐
Lasagne, moussaka	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Pizza (en Grandiosa = ca 550 g)	☐	☐	☐	☐	☐	☐	☐	☐	(pizza)	1/8 ☐	1/4 ☐	½ ☐	3/4 ☐	1+ ☐
Calzone (1 stk = 250-300 g)	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	½ ☐	1 ☐	1½ ☐	2 ☐	2½+ ☐
Pai/quiche	☐	☐	☐	☐	☐	☐	☐	☐	(bit)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Vårruller	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Biff (svin, okse, lam)	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	½ ☐	1 ☐	1½ ☐	2 ☐	2½+ ☐
Koteletter (svin, okse, lam)	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	½ ☐	1 ☐	1½ ☐	2 ☐	2½+ ☐
Stek (svin, okse, lam)	☐	☐	☐	☐	☐	☐	☐	☐	(skive)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Stek (elg, hjort, reinsdyr, rådyr)	☐	☐	☐	☐	☐	☐	☐	☐	(skive)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Gryterett med helt kjøtt, frikassé, fårikål	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Lapskaus, suppelapskaus, betasuppe	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐

Middagsretter fortsetter neste side.....

Prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine.
Ha det siste året i tankene når du fyller ut.

Middagsretter forts...

	Aldri/ sjelden	Gang pr. måned 1	2	3	4	5-6	7-8	9+	Mengde pr. gang					
Kjøtt/kjøttretter forts...										1-2	3-4	5-6	7-8	9+
Bacon, stekt flesk	☐	☐	☐	☐	☐	☐	☐	☐	(skive)	☐	☐	☐	☐	☐
Grillet kylling	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/4 ☐	1/3 ☐	1/2 ☐	3/4 ☐	1+ ☐
Kyllingfilet	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	½ ☐	1 ☐	1½ ☐	2 ☐	3+ ☐
Wok med kjøtt/kylling og grønnsaker	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Kyllinggryte	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Fisk/fiskeretter														
Fiskekaker, fiskepudding	☐	☐	☐	☐	☐	☐	☐	☐	(kake)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Fiskeboller	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-2 ☐	3-4 ☐	5-6 ☐	7-9 ☐	10+ ☐
Torsk, sei, hyse, steinbit, uer (kokt)	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Torsk, sei, hyse, steinbit, uer (stekt, panert)	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Fiskepinner	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-2 ☐	3-4 ☐	5-6 ☐	7-9 ☐	10+ ☐
Sild (fersk, speket, røkt)	☐	☐	☐	☐	☐	☐	☐	☐	(filet)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Makrell (fersk, røkt)	☐	☐	☐	☐	☐	☐	☐	☐	(filet)	½ ☐	1 ☐	1½ ☐	2 ☐	3+ ☐
Laks, ørret (kokt, stekt)	☐	☐	☐	☐	☐	☐	☐	☐	(skive)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Fiskegryte, fiskesuppe	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Fiskegrateng	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Reker, krabbe	☐	☐	☐	☐	☐	☐	☐	☐	(dl, renset)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Wok med sjømat og grønnsaker	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Annet														
Rømmegrøt	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Risengrynsgrøt, annen melkegrøt	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Pannekaker	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Suppe (tomat, blomkål, ertesuppe)	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Vegetarrett, vegetarpizza, grønnsaksgrateng	☐	☐	☐	☐	☐	☐	☐	☐	(bit/dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Hurtignudler (eks. Mr Lee)	☐	☐	☐	☐	☐	☐	☐	☐	(pakke)	½ ☐	1 ☐	1½ ☐	2 ☐	3+ ☐
Omelett	☐	☐	☐	☐	☐	☐	☐	☐	(av antall egg)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐

Prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine.
Ha det siste året i tankene når du fyller ut.

12. Poteter, ris, spagetti, grønnsaker

Svar enten per måned eller per uke.
Disse spørsmålene dreier seg først og fremst om tilbehør til middagsretter, men spiser du for eksempel en rå gulrot eller salat til lunsj, skal det tas med her.

	Aldri/ sjelden	Gang pr. måned			eller	Gang pr. uke					Mengde pr. gang					
		1	2	3		1	2-3	4-5	6-7	8+						
Poteter, kokte og bakte	☐	☐	☐	☐		☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Potetmos	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Potetsalat m/majones	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ss)	1 ☐	2-3 ☐	4-5 ☐	6-7 ☐	8+ ☐
Fløtegratinerte poteter	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Stekte poteter	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Pommes frites (gatekjøkken, frityrstekt)	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Pommes frites, varmet i ovn	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Bønner/linser	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Ris	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Spagetti, makaroni, pasta	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Pølsebrød, lomper	☐	☐	☐	☐		☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Gulrot	☐	☐	☐	☐		☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Hodekål	☐	☐	☐	☐		☐	☐	☐	☐	☐	(skalk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Kålrot	☐	☐	☐	☐		☐	☐	☐	☐	☐	(skive)	1/2 ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Blomkål	☐	☐	☐	☐		☐	☐	☐	☐	☐	(hode)	1/8 ☐	1/6 ☐	1/4 ☐	1/3 ☐	1/2+ ☐
Brokkoli	☐	☐	☐	☐		☐	☐	☐	☐	☐	(stk)	1/8 ☐	1/4 ☐	1/2 ☐	3/4 ☐	1+ ☐
Rosenkål	☐	☐	☐	☐		☐	☐	☐	☐	☐	(stk)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Løk, rå og stekt	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ss)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Salat (eks. issalat, ruccola)	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	1 1/2 ☐	2 ☐	2 1/2+ ☐
Paprika	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ring)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Avokado	☐	☐	☐	☐		☐	☐	☐	☐	☐	(stk)	1/4 ☐	1/2 ☐	3/4 ☐	1 ☐	1 1/2+ ☐
Tomat	☐	☐	☐	☐		☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	1 1/2 ☐	2 ☐	2 1/2+ ☐
Mais	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ss)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Frosne grønnsakblandinger	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Blandet salat (eks. salat, tomat, agurk, mais)	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐

Prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine.
Ha det siste året i tankene når du fyller ut.

13. Saus og dressing

	Aldri/ sjelden	Gang pr. måned: 1	2	3	4	5-6	7-8	9+		Mengde pr. gang
Brun/hvit saus	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	½ ☐ 1 ☐ 1½ ☐ 2 ☐ 3+ ☐
Bearnéssaus, hollandés	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	½ ☐ 1 ☐ 1½ ☐ 2 ☐ 3+ ☐
Smeltet margarin/smør	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐ 1 ☐ 1½ ☐ 2 ☐ 3+ ☐
Kryddersmør	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	½ ☐ 1 ☐ 1½ ☐ 2 ☐ 3+ ☐
Majones/remulade vanlig	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐ 1 ☐ 2 ☐ 3 ☐ 4+ ☐
Majones/remulade lett	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐ 1 ☐ 2 ☐ 3 ☐ 4+ ☐
Seterrømme (35 % fett)	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐ 1 ☐ 2 ☐ 3 ☐ 4+ ☐
Lettrømme (20 % fett)	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐ 1 ☐ 2 ☐ 3 ☐ 4+ ☐
Ekstra lett rømme (10 % fett)	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐ 1 ☐ 2 ☐ 3 ☐ 4+ ☐
Dressing (eks. Thousand Island)	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐ 1 ☐ 2 ☐ 3 ☐ 4+ ☐
Lett dressing (eks. lett Thousand Island)	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐ 1 ☐ 2 ☐ 3 ☐ 4+ ☐
Oljedressing, vinagrette	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐ 1 ☐ 2 ☐ 3 ☐ 4+ ☐
Soyasaus	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐ 1 ☐ 2 ☐ 3 ☐ 4+ ☐
Pesto	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐ 1 ☐ 2 ☐ 3 ☐ 4+ ☐
Tomatsaus, salsa	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	1-2 ☐ 3-4 ☐ 5-6 ☐ 7-8 ☐ 9+ ☐
Ketchup	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐ 1 ☐ 2 ☐ 3 ☐ 4+ ☐
Sennep	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐ 1 ☐ 2 ☐ 3 ☐ 4+ ☐

14. Hvilken type smør/margarin/olje bruker du mest til matlaging?

(Velg en eller to typer)

Smør/margarin	Oljer
☐ Smør (meierismør)	☐ Olivenolje
☐ Bremykt	☐ Soyaolje
☐ Melange	☐ Maisolje
☐ Soft Flora, Soft Ekstra	☐ Solsikkeolje
☐ Vita	☐ Valnøttolje
☐ Flytende margarin på flaske (Vita, Melange, Bremykt o.l.)	☐ Rapsolje
☐ Annen margarin	☐ Vita hjertego
	☐ Andre oljer

Prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine.
Ha det siste året i tankene når du fyller ut.

15. Frukt

Svar enten per måned eller per uke.

	Aldri/ sjelden	Gang pr. måned 1	2	3	eller 1	Gang pr. uke 2-3	4-5	6-7	8+		Mengde pr. gang			
Eple	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Pære	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Banan	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Appelsin	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Klementiner	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4+ ☐
Grapefrukt	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Fersken, nektarin	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4+ ☐
Kiwi	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4+ ☐
Druer	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-10 ☐	11-20 ☐	21-40 ☐	41+ ☐
Melon	☐	☐	☐	☐	☐	☐	☐	☐	☐	(skive)	1 ☐	2 ☐	3 ☐	4+ ☐
Jordbær (friske, frosne)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Bringebær (friske, frosne)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Blåbær	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Multer	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Rosiner	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Tørket frukt (eks. aprikos, fiken)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-5 ☐	6-10 ☐	11-15 ☐	16+ ☐
Frukt- og nøtteblanding	☐	☐	☐	☐	☐	☐	☐	☐	☐	(neve)	1 ☐	2 ☐	3 ☐	4+ ☐

16. Grønnsaker og frukt

Hvor mange porsjoner grønnsaker (utenom potet) spiser du vanligvis pr. dag? (En porsjon er f. eks. 1 gulrot, 1 bolle salat)

Mindre enn 1 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5+ ☐

Hvor mange frukt spiser du vanligvis pr. dag?

Mindre enn 1 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5+ ☐

Prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine.
Ha det siste året i tankene når du fyller ut.

17. Desserter, kaker, godteri

Svar enten per måned eller per uke.

	Aldri/ sjelden	Gang pr. måned 1	2	3	eller Gang pr. uke 1	2-3	4-5	6-7	8+		Mengde pr. gang			
Iskrem (1 dl=1 pinne=1 kremmerhus)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Saftis/sorbet (1 dl=1 pinne)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Hermetisk frukt, fruktgrøt	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4+ ☐
Frisk fruktsalat	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4+ ☐
Pudding (eks. sjokolade, karamell)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4+ ☐
Vaniljesaus	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Pisket krem	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	1 ☐	2 ☐	3 ☐	4+ ☐
Boller, julekake, kringle	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Skolebrød, skillingsbolle	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Wienerbrød, -kringle	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Muffins, formkake	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Vafler	☐	☐	☐	☐	☐	☐	☐	☐	☐	(plate)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Lefse, påsmurt	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Sjokoladekake, brownie	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Marsipankake, bløtkake	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Søt kjeks, kakekjeks (eks. Cookies, Bixit, Hob Nobs)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-2 ☐	3-4 ☐	5-6 ☐	7+ ☐
Kokosbolle	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4+ ☐
Sjokolade (60 g) (eks. melkesjokolade, snickers)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Mørk sjokolade (70% kakao)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(biter)	1-3 ☐	4-6 ☐	7-9 ☐	10+ ☐
Sjokoladebiter/konfekt	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-3 ☐	4-6 ☐	7-9 ☐	10+ ☐
Pastiller uten sukker	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-3 ☐	4-6 ☐	7-9 ☐	10+ ☐
Drops, pastiller, lakris, seigmenn	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-3 ☐	4-6 ☐	7-9 ☐	10+ ☐
Smågodt (1 hg = 100g)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(hg)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Potetgull	☐	☐	☐	☐	☐	☐	☐	☐	☐	(neve)	1-2 ☐	3-5 ☐	6-10 ☐	11+ ☐
Annen snacks (skruer, crisp, saltstenger, lettsnacks o.l.)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(neve)	1-2 ☐	3-5 ☐	6-10 ☐	11+ ☐
Peanøtter, cashewnøtter (1 neve = 25 gram)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(neve)	1-2 ☐	3-4 ☐	5-6 ☐	7+ ☐
Mandler, hasselnøtter, valnøtter (1 neve = 25 gram)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(neve)	1-2 ☐	3-4 ☐	5-6 ☐	7+ ☐

Prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine.
Ha det siste året i tankene når du fyller ut.

18. Kosttilskudd (ts = teskje, bs = barneskje)

	Aldri/ sjelden	Gang pr. uke 1	2-3	4-5	6-7	Mengde pr. gang			
Tran	☐	☐	☐	☐	☐		1 ts ☐	1 bs ☐	1 ss ☐
Trankapsler	☐	☐	☐	☐	☐	(kapsler)	1 ☐	2 ☐	3 ☐	4+ ☐
Fiskeoljekapsler, omega-3 tilskudd	☐	☐	☐	☐	☐	(kapsler)	1 ☐	2 ☐	3 ☐	4+ ☐
Seloljekapsler	☐	☐	☐	☐	☐	(kapsler)	1 ☐	2 ☐	3 ☐	4+ ☐

Multipreparater	Aldri/ sjelden	Gang pr. uke 1	2-3	4-5	6-7	Mengde pr. gang	1	2	3	4+
Sana-sol	☐	☐	☐	☐	☐	(bs)	☐	☐	☐	☐
Biovit	☐	☐	☐	☐	☐	(bs)	☐	☐	☐	☐
Mulitvitamin og mineral (eks. Vitamineral)	☐	☐	☐	☐	☐	(tablett)	☐	☐	☐	☐
Multivitaminer (uten mineraler)	☐	☐	☐	☐	☐	(tablett)	☐	☐	☐	☐

Jernpreparater	Aldri/ sjelden	Gang pr. uke 1	2-3	4-5	6-7	Mengde pr. gang	1	2	3	4+
Duroferon Duretter, Ferromax	☐	☐	☐	☐	☐	(tablett)	☐	☐	☐	☐
Hemofer, hemjern	☐	☐	☐	☐	☐	(tablett)	☐	☐	☐	☐
Amino Jern	☐	☐	☐	☐	☐	(tablett)	☐	☐	☐	☐
Jernmikstur (eks. Floradix)	☐	☐	☐	☐	☐	(bs)	☐	☐	☐	☐

Annet	Aldri/ sjelden	Gang pr. uke 1	2-3	4-5	6-7	Mengde pr. gang	1	2	3	4+
B-vitaminer (flere b-vitaminer i samme tablett)	☐	☐	☐	☐	☐	(tablett)	☐	☐	☐	☐
C-vitamin (60 mg/tablett)	☐	☐	☐	☐	☐	(tablett)	☐	☐	☐	☐
D-vitamin (10 µg/tablett)	☐	☐	☐	☐	☐	(tablett)	☐	☐	☐	☐
E-vitamin (30 mg/tablett)	☐	☐	☐	☐	☐	(tablett)	☐	☐	☐	☐
Folat (folsyre) (200 µg/tablett)	☐	☐	☐	☐	☐	(tablett)	☐	☐	☐	☐

Annet (inkludert helsekostpreparater). Noter navn på preparatet, hvor ofte og hvor mye du tar pr. gang.

Prøv så godt du kan å gi et «gjennomsnitt» av matvanene dine.
Ha det siste året i tankene når du fyller ut.

19. Måltider

Hvor ofte pleier du å spise følgende måltider i løpet av en uke? (Sett ett kryss for hvert måltid)

	Aldri/ sjelden	1 gang i uken	2 ganger i uken	3 ganger i uken	4 ganger i uken	5 ganger i uken	6 ganger i uken	Hver dag
Frokost	☐	☐	☐	☐	☐	☐	☐	☐
Formiddagsmat/lunsj	☐	☐	☐	☐	☐	☐	☐	☐
Middag	☐	☐	☐	☐	☐	☐	☐	☐
Kveldsmat	☐	☐	☐	☐	☐	☐	☐	☐

Hvor mange ganger i løpet av dagen pleier du å spise et eller annet utenom hovedmåltidene? (eks. godteri, frukt, brødskive)

Sjelden	1 gang om dagen	2 ganger om dagen	3 ganger om dagen	4 ganger om dagen	Mer enn 4 ganger om dagen
☐	☐	☐	☐	☐	☐

20. Eventuelle andre matvarer

Bruker du regelmessig matvarer, drikker eller andre produkter som ikke er nevnt i spørreskjemaet? Skriv ned dette så detaljert som mulig. Skriv også hvor ofte du spiser/drikker dette (ganger per måned eller uke) og hvor mye du spiser av dette per gang.

BRUK BLOKKBOKSTAVER

Tromsø-
undersøkelsen

Ditt bidrag teller!

Takk for at du stiller opp og bidrar til viktig forskning.

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