Dietary patterns in Norwegian women aged 50-69 years

Marianne Skov Markussen

Dissertation for the degree of Philosophiae Doctor (Ph.D.)

Department of Nutrition
Institute of Basic Medical Sciences
Faculty of Medicine

UNIVERSITY OF OSLO

Oslo 2016

Series of dissertations submitted to the
Faculty of Medicine, University of Oslo

ISBN 978-82-8333-237-7

Cover: Hanne Baadsgaard Utigard
Printed in Norway: 07 Media AS – www.07.no

Acknowledgement

The work presented in this thesis was performed at the Department of Nutrition, Institute of Basic Medical Sciences, Faculty of Medicine, University of Oslo and was financed by grants from the Norwegian Research Council. The project “Diet and breast cancer in Norway” was initiated by Giske Ursin and the data used in the present thesis was collected in 2007-08. First of all I would like to thank every participant taking part in this project; this work could not have been done without you.

My sincere gratitude goes out to my principal supervisor Lene Frost Andersen, for being encouraging and supportive throughout the PhD period, and especially during the intensive and long finalisation phase. She has with her superior knowledge given me excellent scientific guidance through this work. Also, her wonderful laugh that can be heard through several walls have many a time made me smile when I have felt great hardship. I would also like to thank my co-supervisors Giske Ursin and Marit Bragelien Veierød. Giske, thank you for giving me the opportunity to join the “Diet and breast cancer in Norway” project as a PhD student. Also, I would like to thank you for valuable guidance and constructive feedback in the writing process and for sharing your enormous knowledge in the field of epidemiology. Marit, thank you for your help in solving statistical challenges and interpreting data, for your constructive feedback on the manuscripts and for your constant positive support.

I would like to thank my co-authors Rune Blomhoff, Amrit Kaur Sakhi, Merete Ellingjord-Dale and Anne Lene Kristiansen for their valuable contribution to the project and constructive feedback on the manuscripts. A special thanks goes to Anne Lene Kristiansen for introducing me to dietary pattern analysis. I would also like to thank Anette Hjartåker for valuable discussions on epidemiological methods.

To all my former and present colleagues at the Department of Nutrition, in particular those with whom I shared an office; Merete, Mekdes, Sonja, Jannicke, Anne Marte, Nicolai and Mari, and fellow Phd students; Anine and Anne, thank you for all scientific and non-scientific discussions and for all hilarious episodes and more serious issues that we shared.

To my dear friend and colleague Hege Berg Henriksen, thank you for introducing me to the Department of Nutrition, for your constant support and for all the good times we have had.

Thanks to all my friends for all your support and inspiration and for being a part of my life. Special thanks go to those who have contributed with inspiring and fun social disruptions as lunches, dinners and week-end trips – you know who you are.

To my parents, brothers, aunts and mother in law – thank you for your support and help.

To my children, Mathilde, Mathias and Henrik, thank you for joyful distraction from work and who make me remember what is most important in life. Finally, thanks to Håvard, my husband, for all your patience. You have believed in me and supported me no matter what and helped me balance my perspectives, and for that I am deeply grateful.

Oslo, February 2016

Marianne Skov Markussen

Table of contents

Summary

Background Dietary exposure is complex since foods are eaten in combinations and the foods contain a combination of several nutrients. It is likely that there is an interaction and synergy between these foods and nutrients. The cumulative effect of various foods may be detectable, while the effect of a single food might be undetectable. The complexity of the individual dietary intake can be explored by dietary pattern analyses and this approach might be especially useful if many dietary components are relevant for a disease. Such insight can provide information for setting priorities for changing dietary patterns in a population by public health initiatives. Dietary patterns are meant to reflect dietary behaviour in a population and are based on information gathered using various respondent-based dietary assessment instruments that attempt to measure habitual dietary intake. These instruments are associated with measurement errors that may influence the validity of the dietary patterns. Different methods such as nutritional biomarkers and identification of misreporters of dietary intake may be applied to evaluate the dietary patterns.

Aims The current thesis aimed to (a) identify dietary patterns among Norwegian women aged 50-69 years, (b) evaluate the dietary patterns by using plasma carotenoids as biomarkers of fruits and vegetables, (c) perform sensitivity analyses by excluding the low energy reporters from the study sample to investigate whether our dietary patterns were affected by under-reporting of energy intake, (d) investigate how the under-reporting of energy intake affected the associations between dietary patterns and self-reported chronic diseases and (e) examine the associations between dietary patterns and nutrient intake, sociodemographic factors and key risk factors for NCDs.

Methods The study population was 6974 women aged 50-69 years participating in the Norwegian Breast Cancer Screening Program administered by the Cancer Registry of Norway. Dietary intake was assessed by a 253-item food frequency questionnaire. A total of 3263 women provided blood samples and plasma carotenoids were analysed in a subset of these women (n=361). We identified low energy reporters by using the revised Goldberg cut-off method. The 253 food items were categorised into 46 (Paper I) and 49 (Paper II and III) food groups and dietary patterns were identified using principal component analysis. The association between $\log_e$-transformed plasma carotenoids and dietary pattern scores were estimated by partial correlation coefficients and multiple regression analyses (Paper I). A logistic regression model was used to estimate the association between dietary pattern scores

and the prevalence of self-reported noncommunicable chronic diseases among the total study population and the study population were low energy reporters were excluded (Paper II). We estimated the correlation between nutrients and dietary pattern scores by Pearson's correlation coefficients and multiple regression analyses were used to examine the associations between risk factors for noncommunicable chronic diseases and dietary pattern scores (Paper III).

Results We identified three dietary patterns in the total study population; the healthy "Prudent" pattern and the less healthy "Western" and "Continental" patterns. In the smaller subset of 361 women, four dietary patterns were identified; the "Vegetarian", "Western", "Continental" and "High-protein" pattern. An increasing score for the "Vegetarian" pattern was associated with an increasing concentration of plasma carotenoids whereas an increasing score for the "Western" and "Continental" patterns were associated with a decreasing concentration. A total of 18% of the women were identified as low energy reporters, and their presence in the study sample did not appreciably affect the composition of food groups that loaded highly ($\geq$0.30) on the dietary patterns. However, we observed an attenuation of the associations between dietary pattern scores and several of the self-reported chronic diseases when low energy reporters were included in the study sample, especially among the overweight/obese women. Furthermore, women with high adherence to the "Prudent" pattern were older, more highly educated and had a generally healthy lifestyle. Women with high adherence to the "Western" pattern were older, had lower education and, except for having a low alcohol intake, had a generally unhealthy lifestyle. Finally, women with high adherence to the "Continental" pattern were younger, more highly educated, and had a generally unhealthy lifestyle.

Conclusions We found important dietary patterns among Norwegian women aged 50-69 years. The evaluation of the dietary patterns by plasma carotenoids showed that the "Vegetarian", "Western" and "Continental" patterns were meaningful patterns. The under-reporting of energy intake did not affect the composition of high loaded food groups in the patterns, but the under-reporting attenuated the associations between dietary patterns and self-reported chronic diseases, especially among overweight/obese women. Women with a high adherence to the "Prudent" pattern had a generally healthier lifestyle than women with high adherence to the "Western" and "Continental" dietary pattern.

List of papers

Paper I

Marianne Skov Markussen, Marit Bragelien Veierød, Amrit Kaur Sakhi, Merete Ellingjord-Dale, Rune Blomhoff, Giske Ursin, Lene Frost Andersen. Evaluation of dietary patterns among Norwegian postmenopausal women using plasma carotenoids as biomarkers. *British Journal of Nutrition* 2015; 113(4):672-82.

Paper II

Marianne Skov Markussen, Marit Bragelien Veierød, Giske Ursin, Lene Frost Andersen. The effect of under-reporting of energy intake on dietary patterns and on the associations between dietary patterns and self-reported chronic disease in women aged 50-69. *British Journal of Nutrition*. Submitted.

Paper III

Marianne Skov Markussen, Marit Bragelien Veierød , Anne Lene Kristiansen, Giske Ursin, Lene Frost Andersen. Dietary patterns of women aged 50-69 and associations with nutrient intake, sociodemographic factors and key risk factors for noncommunicable diseases. *Public Health Nutrition* 2016; 19: 1-9.

Abbreviations

24-h recall	24 hours recall
BMI	Body mass index
BMR	Basal metabolic rate
BMRest	Estimated basal metabolic rate
CI	Confidence interval
DBS	Dried blood spots
DLW	Doubly labelled water
EE	Energy expenditure
EI	Energy intake
EIrep	Reported energy intake
FFQ	Food frequency questionnaire
GBAQ	Global Physical Activity Questionnaire
H	Height
HPLC-UV	High pressure liquid chromatografy-ultra violet
IPAQ	International Physical Activity Questionnaire
MET	Metabolic equivalent task
NBCSP	Norwegian Breast Cancer Screening Program
NCD	Noncommunicable disease
NOWAC	Norwegian Women and Cancer Study
OR	Odds ratio
PAL	Physical activity level
PCA	Principal component analysis
QC	Quality control
r	Pearson's correlation coefficient
radj	Partial correlation coefficient
SD	Standard deviation
W	Body weight
β	Regression coefficient

1 Introduction

Analysis of dietary data has evolved over time from focusing on foods consumed, to assessing nutrients, till focusing on diet as a whole [1-3]. Consumption patterns are shaped by several factors, such as income, prices, education, individual preferences and beliefs and cultural traditions [2]. When we eat a meal, we consume a variety of foods with complex combinations of nutrients that are likely to interact with each other or have a synergistic effect [4]. Food consumption often occurs in patterns of meals and in-between meal consumption. Therefore, analysing diet as a multidimensional exposure is a complementary approach to the study of single foods or single nutrients in order to understand the relationship between diet and disease [5]. Dietary pattern analysis is a popular tool in the study of these associations. Dietary patterns are based on information gathered using various respondent-based dietary assessment instruments that attempt to measure habitual dietary intake, such as 24-h dietary recalls, dietary records and food frequency questionnaires (FFQs). What kind of dietary assessment instrument one should use depends on the study objectives, available resources, the population under study and the study design. The FFQ is the most common dietary assessment instrument used to obtain dietary patterns in large epidemiological studies, as it is cheaper to administer and less burdensome for the participants than the dietary records [6, 7]. The FFQ is designed to measure the average long-term dietary intake, and the participant is asked to describe his/her usual frequency of consumption of different foods. It is important that the FFQ is validated for the target population [8]. To be able to determine absolute validity, a gold standard is needed [8]. However, there is no perfect measure or gold standard of dietary intake and the degree of measurement error in the estimation of usual dietary intake cannot be accurately determined. All dietary assessment instruments are therefore associated with different extents of random and systematic measurement errors, and the true intake is not possible to assess. This is a big challenge in nutritional epidemiology. Different methods such as nutritional biomarkers and identification of energy misreporters have been applied to account for some of the apparent measurement errors in order to provide better estimates of the relationship between diet and disease or risk factors for disease [9-11]. In the following sections methods to derive dietary patterns will be introduced, and the validity and reproducibility of dietary patterns will be considered. Finally, I will provide a brief overview of what has previously been reported on the relationship between dietary patterns and modifiable risk factors for noncommunicable diseases (NCDs).

1.1 Dietary patterns

Dietary patterns are measures of the total intake of food combinations in individuals and groups [12], and help to distinguish individuals according to the combination of foods they eat. The usual dietary intake may be captured using a FFQ or multiple non-consecutive 24-h recalls. The dietary pattern methods will then summarize the diet using a smaller number of food items or food groups in a particular combination, which is called a dietary pattern.

1.1.1 Methods to derive dietary patterns

1.1.1.1 A priori approach

Dietary patterns can be defined in an *a priori* approach, or theoretically. By using this approach, scores or indices of dietary quality will express the overall healthiness of the diet [2]. They can be summary measures of the degree to which an individual's diet conforms to specific dietary recommendations. An example is the healthy eating index (HEI) which is a measure of diet quality in terms of conformance to the Dietary Guidelines for Americans [13]. Higher HEI-scores indicate closer conformance with dietary guidance. Diet scores have also been developed based on a specific dietary pattern that has been found to promote health. An example is the Mediterranean diet score [14] which has been found to be associated with a reduction in total mortality in a Greek population.

Dietary quality scores can be useful tools to monitor the overall adherence to dietary guidelines, and the dietary quality of a population [2]. The strengths of the *a priori* approach is that the dietary quality scores rely on the scientific evidence from studies on health and disease prevention, and they are easy to compute and thereby easily reproducible and comparable [2]. However, they are based on current knowledge. As some dietary quality scores are dependent of dietary guidelines, the quality of the dietary guidelines will influence them [2]. Another limitation is that dietary quality scores do not necessarily describe the overall dietary pattern because the focus is on selected parts of the diet.

1.1.1.2 A posteriori approach

Dietary patterns can also be defined in an *a posteriori* approach, or empirically, where data-driven statistical methods such as cluster analysis, principal component analysis (PCA)

or exploratory factor analysis are used to explore the structure of existing dietary patterns in the population, without a preassessment of their importance or quality [12].

Cluster analysis is an approach that aims to build mutually exclusive non-over-lapping clusters of individuals with similar diets [15]. More specifically, the dietary data are categorised into food groups before the analysis. A standardisation of the food groups is necessary before the analysis since the method is sensitive to outliers. Thus, food groups are commonly divided by total energy intake and the percentage of energy contributed by each food group is calculated and used in the cluster analysis [15]. The analysis are based on distance measures between individuals with respect to their dietary intake, and minimises the variation within clusters and maximises the differences between clusters [8]. Each cluster represents a dietary pattern with a specific food and nutrient composition that are specific to a group of participants in the study population. The advantage with cluster analyses is that a specific dietary pattern is assigned to each participant, and a participant can belong to one cluster only. Limitations are the subjective decisions taken by the investigator that can influence the results. That is, which foods to be grouped together and the prespecification of the number of clusters [2, 5]. Another limitation is that the standardisation of the food group variables could give minor food groups a greater influence than they actually have which might dilute differences in the dietary patterns [2].

In both PCA and exploratory factor analysis the dietary variables (food items or food groups) are reduced into a smaller set of dimensions, called either principal components or factors, based on their interrelationships. The correlations between the dietary variables are arranged in a table, also called a correlation matrix. There are some theoretical differences between PCA and exploratory factor analysis. The PCA analyses the total variance in a correlation matrix and reduces the number of dietary variables to a smaller number of principal components (dietary patterns). The principal components are generated sequentially and are uncorrelated to each other, i.e., the first principal component identified accounts for most of the variance in the data, the second principal component identified accounts for the second largest amount of variance and is uncorrelated to the first principal component, and so on [16]. Exploratory factor analysis analyses the common variance in a correlation matrix and estimates underlying factors (dietary patterns) which represent groups of variables that correlate highly with each other, but not with variables outside the group [16]. Several investigators have concluded that the dietary patterns generated from PCA differ little from those derived from exploratory factor analysis [15-17], and in dietary pattern analysis PCA is the method most commonly used [2]. In PCA and exploratory factor analysis, the participant will

get a factor score on each dietary pattern. As for the cluster analysis, a limitation with PCA and exploratory factor analysis is the investigators' subjective decisions regarding the pregrouping of foods and how many dietary patterns to retain [5].

In studies where PCA and cluster analysis were used simultaneously to derive dietary patterns, results have shown good evidence of comparability [18-20].

In the present work, dietary patterns were derived by PCA and this method is described in more detail in section 1.1.2.

1.1.1.3 Hybrid approach

Hybrid approaches to derive dietary patterns combines theoretically and empirically approaches, with the reduced rank regression method [21] being the most common [2]. This method creates linear combinations of dietary intake variables that best explain the variance in a set of response variables, usually biomarkers of disease. For example, the identification of linear functions of food groups that explain as much variation as possible in a set of risk markers for cardiovascular disease (HDL-cholesterol, LDL-cholesterol, lipoprotein (a), C-peptide and C-reactive protein) [15]. The reduced rank regression method has the advantage of building on *a priori* knowledge of biological relations or disease aetiology, in combination with exploratory statistical analyses to extract dietary patterns that are likely to be related to a specific disease [15, 21]. This method could be useful in generating hypotheses about foods that may contribute to disease risk through specified causal pathways [12]. The application of the reduced rank regression method is limited to those health outcomes for which sufficient knowledge about intermediate risk factors are available [2, 15].

1.1.2 Dietary patterns derived by PCA

In deriving dietary patterns by PCA, some preparations of the dietary data are usually done before the analysis. The dietary data are often reduced by grouping individual foods into nutritionally similar food group variables [5]. The food group variables can be adjusted for total energy intake or transformed to more normal distributions before entering them into the PCA. However, several studies have reported insignificant differences in effect estimates between dietary patterns and outcome whether the food group variables were adjusted for energy intake or not before entering them into the PCA [7, 22, 23], and that it is sufficient to make energy adjustments when analysing the effects of the dietary patterns on the outcome of interest [7].

The PCA will aggregate the food groups in linear combinations called principal components (dietary patterns), according to the degree to which the food groups are correlated to each other [5]. To improve interpretability, the components are usually rotated by an orthogonal transformation to achieve a simpler structure [5, 22-33]. The analysis will produce as many principal components as there are variables entered into the PCA. The components are generated sequentially and are uncorrelated. To determine how many components or dietary patterns to retain, there are several criteria to consider [5, 15, 16]. The Kaiser criterion (eigenvalues >1) in conjunction with the scree test, the magnitude of factor loadings and observing the general interpretability of the dietary patterns are commonly used criteria for this purpose.

The dietary patterns derived by PCA usually account for only a modest proportion of the variance in dietary intake in a study population. The proportion of variance explained varies with the number of food group variables entered into the analysis. That is, a smaller number of input variables explains a greater percentage of the variance in dietary intake compared to a larger number of input variables [5].

The output of the PCA is the linear combinations of food groups, with each food group having a factor loading which gives the importance of a food group in a dietary pattern and can be interpreted as correlation coefficients [34]. An absolute factor loading ≥0.3 are often used as cut-off to decide which food groups make up which dietary patterns [16]. Individual factor scores for each dietary pattern can be calculated by summing the standardised food groups weighted by their factor loadings. The factor scores rank the individuals according to the extent to which they consume foods from groups that are highly weighted in a dietary pattern [12]. An individual will have one factor score on each dietary pattern which can be used simultaneously in a regression analysis since they are supposed to be uncorrelated. It is possible for an individual to have a high factor score on more than one dietary pattern [12]. The overall dietary pattern of an individual is represented by his/her factor scores on all the identified dietary patterns and reflect one aspect of an individual's diet, but do not provide the total picture of what exactly is consumed [1].

1.1.3 Evaluation of dietary patterns

1.1.3.1 Measurement errors in dietary assessment

Measurement error is the difference between the observed or measured value and the true value [35]. There are generally two types of measurement errors, random or systematic [8,

[36]. Random measurement error is “the portion of variation in a measurement that has no apparent connection to any other measurement or variable, generally regarded as due to chance” [37]. For example, the day-to-day variations in an individual’s dietary intake, the unintentional omission or addition of foods in recall methods or ticking off a wrong box in the FFQ. Systematic measurement error is “an error that is consistently wrong in a particular direction” [37], or the measurement differ in a systematic manner from the true values. For example, that some individuals systematically under-report unhealthy food and over-report healthy food. Measurement errors, both random and systematic, can be non-differential or differential. Random measurement error is often thought of as non-differential, i.e., errors that are randomly distributed around a true value and unrelated to the outcome. Non-differential measurement error or random measurement error in an exposure and/or an outcome typically cause the categories under comparison (participants with a specific health outcome versus participants without) to become more similar and might lead to an attenuation of an effect [36].On the other hand, if the measurement error in the exposure (e.g. dietary intake) occurs to a different extent in those who have a specific health outcome compared to those without, the measurement error is differential. The effect of differential measurement error on an association between the exposure and the outcome are generally harder to predict than those of non-differential measurement error, and can either exaggerate or underestimate an effect [36].

The effect of random measurement errors can be reduced by increasing sample size or number of measurements of each subject, and the average value will then approach the true value [8]. An estimate with little random error may be described as precise [36]. The effect of systematic measurement errors will not be reduced by increasing sample size or number of measurements, and would be present even with an infinitely large study sample. In order to measure the amount of systematic error in the exposure variables, a validity or calibration study is required [8]. An estimate with little systematic error may be described as valid [36].

The measurement errors in the dietary data will transfer to the obtained dietary patterns and might distort the composition of food groups in the patterns. Different methods can be used to evaluate the dietary patterns and will be described in more detail in the next section.

1.1.3.2 Evaluation strategies for dietary patterns

The obtained dietary patterns can be evaluated by using different methods, such as examining their correlation to objective nutritional biomarkers, or by investigating how under-reporting of energy intake (EI) may affect the dietary patterns. The reproducibility of dietary patterns is also important to evaluate. These issues will be described in more detail in three separate sections below.

Evaluation by biomarkers

A nutritional biomarker is a biological specimen that can have various uses. It can be used as a surrogate for actual dietary intake, as a measure for nutrient status, as a marker for compliance in intervention studies or finally, as a marker to validate dietary assessments [8]. The concentration in blood or tissue of the nutrient used as a nutritional biomarker is influenced by variation in the absorption, transport and distribution, metabolism and excretion of the nutrient [8]. These processes may be influenced further by genetic characteristics of individuals, other dietary intakes, lifestyle factors and pathophysiological factors. In general, dietary biomarkers can be divided into three categories; recovery-, predictive-, and concentration biomarkers, depending on the relationship between intake and biomarker.

A recovery biomarker is directly related to dietary intake and not subject to homeostasis or substantial inter-individual differences in metabolism and provides an estimate of absolute intake level [8, 9]. Examples are the doubly labelled water (DLW) method used to measure the metabolic rate and total energy expenditure [11], and the urinary total nitrogen/potassium used to estimate total daily protein consumption [38] and potassium intake [39], respectively. Unfortunately, these methods are technically challenging and extremely expensive and therefore not possible to implement in most epidemiological studies.

The predictive biomarkers are also sensitive, time-dependent and show a dose-response relationship with intake levels. However, their overall recovery is lower than for the recovery biomarkers but correlations with intake are high. Therefore, values of predictive biomarkers might be used to estimate absolute intakes [40]. Examples of predictive biomarkers are urinary sucrose and fructose as markers of sugar intake [40].

The concentration biomarkers are subject to complex metabolic pathways in their regulation and cannot be translated into absolute levels of intake. Concentration biomarkers correlate with intakes of corresponding foods or nutrients, although the strength of correlation is lower than for the recovery biomarkers. The differences in metabolic and genetic factors

between individuals will also affect the correlation of a biomarker with the relevant dietary exposure [9]. Examples of concentration biomarkers are carotenoids, vitamins, blood lipids and urinary electrolytes.

Different concentration biomarkers have been used for evaluating dietary patterns. Dietary patterns consistent with current notions of a healthy diet or unhealthy diet have been reported to be associated with serum vitamin C, folate, most carotenoids and vitamin E in the expected direction [41-44]. Plasma carotenoids are the biomarkers focused on in the present work (Paper I).

Carotenoids are pigments predominantly found in fruits and vegetables [45], and as they cannot be synthesised by humans, they are considered to be good candidates for biomarkers of fruits and vegetables [46]. Of the approximately 600 carotenoids found in plant species [47], only α-carotene, β-carotene, β-cryptoxanthin, lutein, lycopene and zeaxanthin are found in appreciable concentrations in human plasma (or serum) [48]. As mentioned above, carotenoids are concentration biomarkers and several factors affect their concentrations in plasma, such as food preparation, the individual's body size, gender, smoking status, alcohol use, cholesterol level and inter-individual variability in absorption [49-52]. Furthermore, different populations tend to have different carotenoid profiles depending on their fruit and vegetable intake. The carotenoids' validity as biomarkers can be affected among subjects with low vitamin A status, because α-carotene, β-carotene and β-cryptoxanthin will then be partially metabolised to retinol [53, 54]. Moreover, smoking and high consumption of alcohol leads to oxidative stress, and since carotenoids has a role as antioxidants, there have been observed decreased plasma concentrations of some carotenoids in subjects having these habits [9]. Although there are a lot of factors that will affect the response of plasma carotenoids to fruit and vegetable consumption, they do appear to be useful biomarkers for these food groups [46]. Their correlation with fruit and vegetables are moderate, with Pearson's correlation coefficient ranging from approximately 0.2 to over 0.5 [55]. Plasma carotenoids are relatively stable when stored frozen, and are therefore suitable for use as biomarkers in prospective cohort studies [56].

A healthy dietary pattern characterised by high factor loadings for fruits and vegetables are expected to be positively associated with plasma carotenoids and vice versa for a dietary pattern not characterised by these food groups. In some studies, carotenoids have been used as reference method in the evaluation of dietary patterns [30, 43, 44].

Exclusion of under-reporters

All nutrients must be provided within the quantity of food needed to fulfil the energy requirement, therefore, the reported EI is a substitute measure of the total quantity of food intake [11]. Energy misreporting, either under- or over-reporting, is a common source of measurement error in dietary surveys [10, 57-59]. The two most common methods to evaluate the EI are based on the assumption that EI must equal the energy expenditure (EE) when maintaining a stable weight [11]. The DLW method [60]is regarded as the gold standard for measuring total EE. In practice, the subjects are given a dose of water enriched with the stable isotopes deuterium and oxygen 18. The urine samples are collected before and after administration of the dose, and then analysed by isotope ratio mass spectrometry to determine the rate of disappearance of each isotope from the body. From these estimates the total EE can be calculated. In validity studies of dietary assessment methods, the DLW method has uncovered a frequent under-estimation of dietary intake [11].

A more simple and less expensive method, the Goldberg cut-off method [57, 58], has been proposed as an alternative to identify potentially misreporters of EI, and was the method used in the present work (Paper II). The Goldberg cut-off method is based on the principle that EI equals EE when weight is stable (equation (1): EI=EE) [10, 57]. EE can also be expressed as multiples of basal metabolic rate (BMR) and physical activity level (PAL), and replacing EE in equation (1) with BMR x PAL gives equation (2): EI/BMR=PAL. The idea by Goldberg and colleagues were that the ratio EI/BMR could be estimated and evaluated against an expected PAL for a population. Based on this idea Goldberg et al. derived an equation [57], which has later been revised by Black [10], that calculates the 95% confidence limits (cut-offs) for the plausible EI. The value of these cut-offs varies according to PAL, number of days of food recording and whether the evaluation of EI/BMR is at the individual or group level (the equations are described in more detail in section 3.4). Individuals are identified as plausible reporters, low energy reporters or high energy reporters according to whether their EI/BMR is within, below or above the 95% confidence limits, respectively.

Many studies have observed that under-reporting is not random. Women, elderly, obese, individuals in lower socio-economic classes and individuals with lower education are more likely to under-report their EI [11, 61-66].

Some studies have investigated the effect of under-reporting on dietary patterns derived by cluster analysis [67-74]. Four studies reported that dietary patterns derived plausible reporters (low energy reporters excluded) were relatively similar to the patterns derived among all reporters [67, 69-71]. One study found that the number of dietary patterns obtained

differed between plausible reporters and all reporters [68]. Several studies have observed that low energy reporters tend to report higher intakes of fruits and/or vegetables, and lower intakes of the more unhealthy foods [75-79] than plausible reporters. Deriving dietary patterns by cluster analysis allows the researchers to examine the distribution of low energy reporters across patterns. Studies have reported that the low energy reporters were not uniformly distributed across patterns, and whether the highest proportion of low energy reporters were found in the healthy or unhealthy clusters differed between the studies [67, 68, 70-74]. Recently, in a study among Swedish adults the researchers investigated the effect of excluding low energy reporters on dietary patterns derived by PCA [80] and found that the patterns were largely consistent. To the best of our knowledge that study is the only study that has investigated the effect of under-reporting of EI on dietary patterns derived by PCA.

Reproducibility of dietary patterns

Reproducibility, or reliability, refers to the extent to which results of a measurement can be replicated [81]. Reproducibility of dietary patterns derived by PCA has been assessed by obtaining dietary patterns from repeated dietary assessments on the same subjects. For example, Hu et al. [30] investigated reproducibility of a "Prudent" and a "Western" pattern among participants who completed the same validated FFQ twice, one year apart. The correlations were 0.70 for the "Prudent" pattern and 0.67 for the "Western" pattern, indicating a good reproducibility.

It has been found that the reproducibility of dietary patterns may differ considerably between different dietary pattern solutions (i.e., if 2, 3, 4, 5 or 6 dietary patterns were extracted from the PCA) [17]. The researchers in that study found that different pattern solutions contained patterns with different compositions of significantly contributing food groups. They also found that the choice of the final dietary pattern solution affected the association between dietary patterns and disease risk. The researchers reported that the quantitative criteria for how many dietary patterns to obtain, i.e., the Kaiser criterion, recommended extracting considerably more patterns than the researchers found interpretable. Their conclusion was that the best way to decide how many dietary patterns to extract was by using half-split techniques (the study population are randomly half-split and dietary pattern solutions from PCA in one half is confirmed using confirmatory analysis in the other half), and by visually inspecting the scree plot.

1.1.4 Often observed dietary patterns

Two dietary patterns have frequently been found in numerous studies [5]. One of the patterns is defined by several healthy food groups with high factor loadings, including fruits, vegetables, legumes and fish, and is often called the "Prudent" pattern. The other pattern is defined by several less healthy food groups with high factor loadings, including red and processed meat, potatoes, refined grains and sugar and is often called the "Western" pattern. Also patterns high in desserts or sweets, or high in alcohol have been identified repeatedly [5]. Balder et al. [23] derived dietary patterns from four different cohort studies in Finland, the Netherlands, Sweden and Italy. They reported that some dietary patterns were shared by the four populations, whereas other patterns were population specific.

1.1.5 Dietary patterns and associations with sociodemographic factors and modifiable behavioural key risk factors for noncommunicable chronic diseases

There is a growing burden of NCDs worldwide that represents major health challenges to global economic and social development [82]. A NCD, is a chronic medical condition or disease that can be defined as non-infectious and non-transmissible among people [82]. They are of long duration and generally slow progression. NCDs are reaching epidemic proportions worldwide and currently cause more deaths than all other causes combined. In 2012, 38 million deaths were due to NCDs and are projected to increase to 52 million by 2030 [82]. Cardiovascular diseases, cancer, chronic respiratory diseases and diabetes are four major NCDs that are responsible for 82% of deaths from NCDs. These diseases are linked to modifiable behavioural key risk factors: unhealthy diet, physical inactivity, obesity, smoking and harmful use of alcohol. An important way to reduce NCDs is to change people's habits with respect to these risk factors [82].

Identification and characterisation of major dietary patterns and their association with sociodemographic factors and risk factors for NCDs in a population represent important knowledge for health authorities when forming strategies to promote a healthier diet and lifestyle.

In Norway, there have been some studies investigating the associations between dietary patterns and sociodemographic and modifiable risk factors for NCDs. In a study among Norwegian women (the Norwegian Women and Cancer Study, NOWAC) Engeset et al. [83] used dietary data collected by a 50-item FFQ in 1998 that focused on traditional

Norwegian food habits. They derived six dietary patterns and investigated the associations between the patterns and lifestyle factors. They found an inverse association between a "Traditional fish eater" pattern and income and education. Furthermore, positive associations were found between both the "Healthy eater" and "Alcohol users" patterns and income, and persons in the "Alcohol users" pattern were more likely to be current smokers. In another Norwegian study among adult working Oslo citizens, four dietary patterns were derived based on an 82-item FFQ [31]. The "Modern" and the "Sweet" dietary patterns were inversely associated with physical activity, even if the "Modern" pattern was the healthiest of the four patterns because of the high factor loading for vegetables. The unhealthy "Sweet" pattern was inversely associated with body mass index (BMI), which was unexpected, while the unhealthy "Western" pattern was positively associated with BMI and waist/hip ratio. In another study from the same research group [32] a positive association was found between a "Prudent" pattern and education and occupational group (ranging from unskilled manual workers till higher-grade professionals /managers).

Numerous studies have reported that different dietary patterns are differently related to age, sociodemographic factors and behavioural risk factors for NCDs, such as smoking, BMI, alcohol intake and physical activity [73, 80, 84-92] . In these studies, dietary patterns with high loadings of fruits and vegetables was often positively associated with education and physical activity but inversely associated with smoking. Patterns associated with alcohol intake have previously been reported to be linked to cigarette smoking [84, 93].

1.2 Rationale and significance of the thesis

Investigating the relation between dietary patterns and disease or risk factors for disease can lead to new insight which is important for the development of dietary guidelines. Dietary pattern analysis using PCA has become a popular method for studying the total dietary intake in a population. However, this analysis is based on data from respondent-based dietary assessment instruments which include considerable measurement errors [18]. This may influence the composition of food groups that contributes significantly to the dietary patterns and thereby lead to erroneous conclusions when investigating associations between dietary patterns and disease or risk factors for disease. Therefore, we believed that an evaluation of the dietary patterns was important. Moreover, the effect of under-reporting of EI on associations between dietary patterns derived by PCA and disease has not been much studied.

Such information is valuable for setting focus on the impact measurement errors in dietary data may have on the associations between dietary patterns and disease.

Although there have been some dietary pattern studies in Norway [31, 32, 83, 94-102], there are no studies with recently collected detailed dietary data that have covered the whole country on associations between dietary patterns and nutrient intakes, sociodemographic factors and risk factors for NCDs.

2 Aims

The overall aim of this study was to identify and evaluate dietary patterns among Norwegian women aged 50-69 years, and study the association between the dietary patterns and sociodemographic factors and risk factors for noncommunicable chronic diseases. Moreover, to study the impact of under-reporting of EI on associations between dietary patterns and self-reported chronic disease.

Specific aims:

- To identify dietary patterns using PCA in a subset of the study sample of Norwegian women aged 50-69 years, and evaluate the patterns by examine their associations with plasma carotenoid concentrations (**Paper I**).

- Investigate the effect of under-reporting of EI among women aged 50-69 years on (a) the composition of food groups with high factor loadings in the dietary patterns derived by PCA and (b) the associations between the dietary patterns and self-reported chronic diseases. (**Paper II**).

- Identify major dietary patterns using PCA among women aged 50-69, and study associations between the dietary patterns and nutrient intake, sociodemographic factors and modifiable risk factors for NCDs; physical activity, BMI, alcohol intake and smoking (**Paper III**).

3 Subjects and methods

3.1 Study population

The present work is based on data from the Norwegian Breast Cancer Screening Program, a governmentally funded national screening program administered by the Cancer Registry of Norway [(103)]. All Norwegian women aged 50-69 years are invited to undergo a mammographic examination every second year, and the participation rate is 76% [(104)]. In 2006/2007, the Norwegian Breast Cancer Screening Program's invitation letter for mammographic screening included a question on willingness to complete a dietary questionnaire. A total of 67,527 women agreed to participate. In 2008, a consent form and a FFQ were sent to a random sample of 10,000 of these women living all over Norway. A total of 6974 returned the FFQ and more than 90% agreed to provide saliva and blood samples. A self-collection kit containing equipment both for saliva and fingertip blood samples on filter paper were sent to 4597 women. We received 3258 saliva and 3263 blood samples. Funding was available for a subset of laboratory analyses. The main aim in 2006/2007 was to study dietary intake in relation to breast cancer and mammographic density[(105)], therefore analyses of blood samples were restricted to women who also had an analogue mammogram (n=632) and of these, 387 had fulfilled the inclusion criteria for that study. Papers I-III had a common set of exclusion criteria: The FFQ was not filled in (n=46); missing data on height and/or weight (n=158), age (n=5), smoking status (n=41); height <125 cm (n=7) weight <30 kg or >170 kg (n=13); age not within the range 50-69 years (n=15); or energy intake <2100 kJ/day or >15,000 kJ/day (n=204). In total 489 women were excluded, and 6485 women were available for analyses. Further exclusions were done differently for the three papers as described below.

In paper I women were excluded if BMI <15 kg/m^2 or >50 kg/m^2 (n=4), thus the total study sample consisted of 6481 women. From the subsample of 387 women that provided blood samples, we restricted analyses to those who followed the instructions for the storage of blood samples (n=26 were excluded). We were then left with blood samples from 361 women in whom the carotenoid analyses were conducted.

In paper II and III women were excluded if they had missing data on education (n=79) or physical activity (n=104), which left us with a study sample of 6302 women.

In paper II women were further excluded if BMI<18.5 kg/m^2 or ≥40 kg/m^2 (n=98). In this paper we focused on the associations between dietary patterns and self-reported diseases.

Women with BMI >40 kg/m^2 are characterised as “very severely obese” [106] and the risks of comorbidity and mortality associated with this BMI category is described as “very severe” [107].Women with BMI <18.5 kg/m^2 are characterized as underweight [106], which might be secondary to or symptomatic of an underlying disease. Therefore, women with BMI <18.5 kg/m^2 or ≥40 kg/m^2 were kept out of this study, and left us with a total study sample of 6204 women.

In paper III women were excluded if BMI<15 kg/m^2 or >50 kg/m^2 (n=4). This left us with a total study sample of 6298 women.

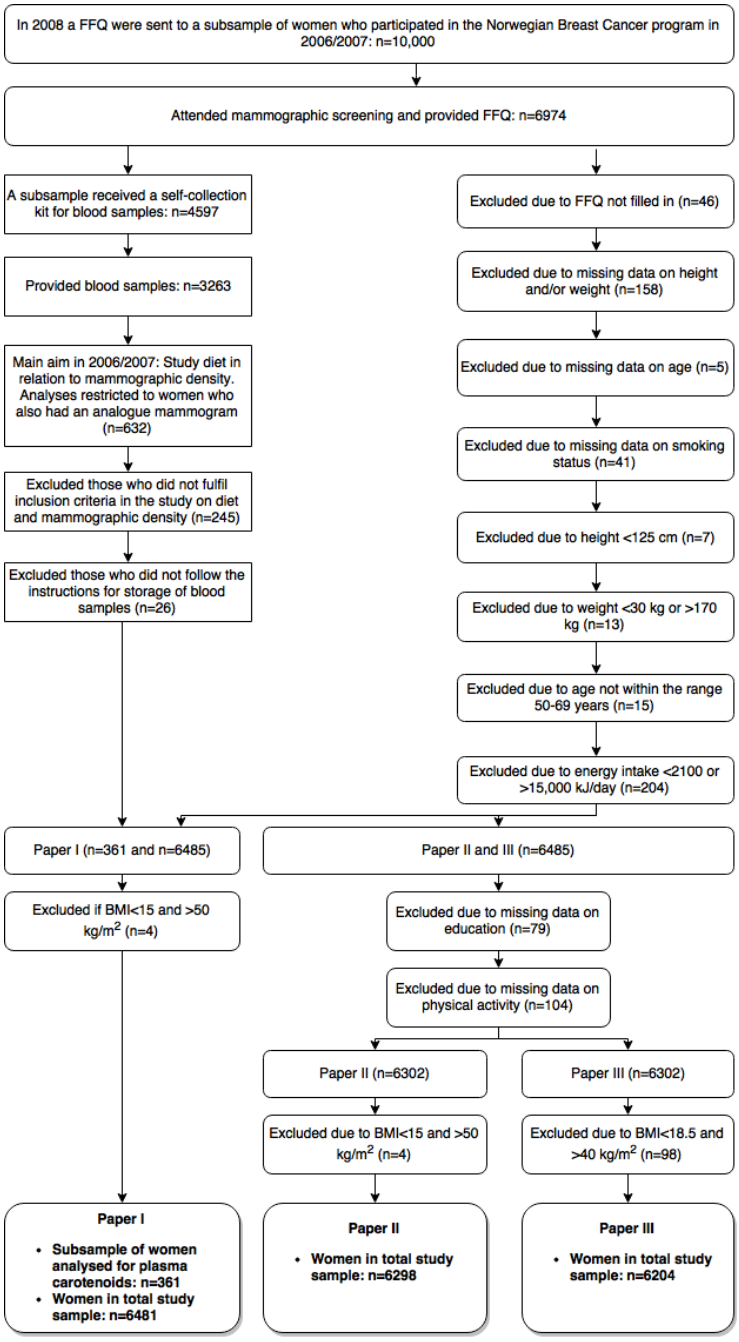

Figure 1. Flow chart for the study samples in Paper I-III

3.1.1 Approvals

The study protocol was approved by the Regional Committee for Medical Research Ethics and the Norwegian Data Protection Authority. A written informed consent was obtained from all women.

3.2 Questionnaire

3.2.1 Dietary assessment

Dietary data were collected by using a self-administered and optically readable FFQ (Appendix I). The 16-page, 253-item FFQ was designed to capture the habitual food intake among Norwegian adults the preceding year, with an extra focus on fruit, vegetables, antioxidant-rich foods and beverages. It was based on a previously validated 180-item FFQ designed to measure the total energy intake in the Norwegian population [108], which later was expanded to a 270-item FFQ to cover the most antioxidant-rich foods and beverages in Norway [109]. The energy and food intake estimated from the 270-item FFQ has been validated [109, 110]. The energy intake was evaluated against independent measures of energy expenditure using the ActiReg® system (motion detection), whereas 7-days weighed food records were used to study the relative validity of food and nutrient intake [109, 110]. The correlation coefficient between energy intake and energy expenditure was 0.54. Correlations between FFQ and the weighed food records were 0.41 for berries, 0.61 for fruit and 0.38 for vegetables [109]. This FFQ has also been validated for ranking individuals according to their usual intake of fruit, juice and vegetables by using the method of triads with two independent and specific biomarkers of fruit and vegetables, the FFQ and 7-d weighed food records. The validity coefficients ranged from 0.60 to 0.94[110]. We revised the 270-item FFQ by removing 17 items (curly kale, red cabbage, mushroom, globe artichoke, sundried tomatoes, tofu, cumin, turmeric, ginger powder, caraway, cloves, piri piri, sage, rosehip tea, organic blueberry juice, organic blackcurrant juice and crowberry juice) that was seldom or never eaten.

For each food item in the 253-item FFQ used in the present work, participants indicated their frequency of consumption ranging from never/seldom to several times per day. The portion size per consumption was asked in household units such as slices, glasses, cups, pieces, spoons and teaspoons. When a respondent only reported the frequency, but not the portion size, the food item was given the smallest portion size. If only the amount of the food

item was given, the food item was considered as not used and treated as null intake. The questionnaire also collected information about dietary supplements. The computation of daily dietary intake was performed using the food database AE-07 and KBS software system (KBS, version 4.9 2008) developed at the Department of Nutrition, University of Oslo, Norway. The food database AE-07 is based on the 2006 edition of the Norwegian food composition table (www.norwegianfoodcomp.no). Intakes from dietary supplements were included in the calculations.

The 253 food items were categorized into 46 food groups in Paper I (Table 1 in Paper I) and 49 food groups in Paper II and III (Supplemental Table 1 in Paper II and III), and the food groups was based on similarity in ingredients, nutrient profile or culinary usage.

3.2.2 Non-dietary variables

The questionnaire included questions about height, weight, physical activity, smoking habits, diseases and medication.

3.2.2.1 Physical activity assessment

In Papers II and III, we described the level of physical activity among the participants. Recreational physical activity was assessed using a modified version [111] of the physical activity questionnaire used in the California Teachers Study [112]. Subjects were asked to assess habitual weekly physical activity, and report all physical activity lasting at least 10 minutes per session. Physical activity included three variables referring to light physical activity (e.g. walking or cross-country skiing at a slow pace), moderate physical activity (defined as activities where some effort is required and which cause increased breathing, e.g. bicycling, swimming or cross-country skiing at a moderate pace, jogging at a slow pace, dancing) and strenuous physical activity (defined as activities that require hard effort and causes substantial increased breathing, e.g. aerobics, running, cross-country skiing or bicycling at a brisk pace). Each physical activity variable comprised seven categories: (1) none, (2) <0.5 h/week, (3) 0.5-1 h/week, (4) 1.5-2 h/week, (5) 2.5-3.5 h/week, (6) 4-6 h/week, (7) ≥7 h/week. We created separate light, moderate and strenuous physical activity variables in minutes per week by summing up hours per week for each level of activity multiplied with 60.

We also calculated a variable indicating energy expenditure by multiplying the number of hours of each physical activity by its estimated metabolic cost [113] and expressed this variable in metabolic equivalent task (MET–h/week).

3.2.2.2 Disease assessment

In Paper II, we described the prevalence of self-reported chronic disease among the participants and we investigated the associations between the dietary patterns and self-reported chronic diseases. The questionnaire included questions about selected current or previously diagnosed chronic diseases: asthma, joint inflammation, muscle or skeletal disorder, chronic gastrointestinal disease, chronic respiratory disease, depression or psychiatric disorder, stroke, heart attack or angina, hypertension and diabetes (type 1 or type 2). We defined six disease groups: Total chronic disease (composed of all of the following disease groups), cardiovascular disease (stroke, heart attack, angina and hypertension), diabetes (type 1 and 2), chronic respiratory disease (asthma and chronic respiratory disease), cancer and joint/muscle/skeletal disorders (joint inflammation, muscle and skeletal disorders). A participant was identified to belong to a disease group if she had at least one of the diseases in the group.

3.3 Blood collection and carotenoid analysis

A self-collection kit containing necessary equipment and a detailed instruction on how to collect finger-tip blood samples on filter paper was sent to the participants by mail. The first two spots on the filter paper were impregnated with a proprietary stabilizing solution (Vitas AS, Oslo, Norway). The participants were instructed to collect blood samples after fasting overnight, collect capillary blood from a fingertip directly on the filter paper (Whatman 903 paper, GE Healthcare, USA) and subsequently dry it for 8 hours. The filter paper with the dried blood spots (DBS cards) should then be stored in an air tight alumina bag together with a silica drying medium (Whatman, Sanford, USA) and mailed by regular mail to the study centre. The DBS cards were stored at -80˚C at the study centre.

Quantification of carotenoids in DBS was performed by the contract laboratory Vitas AS, Oslo, Norway. In short, five punches of 3.2 mm from each DBS were punched into vials, added distilled water, mixed, proteins precipitated and carotenoids extracted with isopropanol added internal standard (β-Apo-8-carotenal, Sigma Aldrich, St-Louis, MO, USA). HPLC-UV

analysis was performed on an 1100-series HPLC with a 1260 diode array detector (453nm) (Agilent Technologies, Palo Alto, CA). Separations was performed on a 3 µm, YMC C30 (150 mm × 4.6 mm i.d.) column (YMC, Kyoto, Japan). Calibration was performed by analysing DBS calibrators spotted with full blood with known concentration of the carotenoids: lutein, zeaxanthin, β-cryptoxanthin, α-carotene, β-carotene and lycopene. The known concentrations are obtained by analysis of serum from the same full blood. The calibrator for these values was NIST SRM-1950 (National Institute of Standards and Technology, Gaithersburg, MD, USA). A fixed haematocrit value of two was used to convert from DBS to plasma values. DBS quality control (QC) samples were run alongside the study samples. The coefficient of variation (%) (n=50) for these QC samples were 7.8-9.0% for lutein, β-carotene, lycopene, β-cryptoxanthin, 21.0% for the low abundant zeaxanthin and 20.1% for α-carotene. The total carotenoid concentration in plasma was calculated as the sum of the individual carotenoid concentrations quantified in this analysis.

3.4 Definition of low-energy reporters of energy intake

Low-energy reporters were determined using the Goldberg cut-off method [57] revised by Black [10]. The method evaluates the energy intake by comparing the ratio of reported energy intake (EI_{rep}) to the estimated basal metabolic rate (BMR_{est}) with the individuals' expected absolute energy requirement. The expected absolute energy requirement is the ratio of energy expenditure (EE) to the BMR, or also known as physical activity level (PAL) [114]. The BMR_{est} was calculated as given by Henry [115], where W is body weight in kilograms and H is height in metres:

BMR_{est} women 31-60 years: 0.0433 W + 2.57 H – 1.180 (1)

BMR_{est} women 61-70 years: 0.0342 W + 2.10 H – 0.0486 (2)

The Goldberg and Black's cut-off values were established as follows:

$$\frac{EI_{rep}}{BMRest} > PAL \times \exp\left(SD_{min} \times \frac{\frac{S}{100}}{\sqrt{n}}\right) \quad (3)$$

$$\frac{EI_{rep}}{BMRest} < PAL \times \exp\left(SD_{max} \times \frac{\frac{S}{100}}{\sqrt{n}}\right) \quad (4)$$

where n is equal to 1 (for data at the individual level), the standard deviation (SD) is -2 for the 95% lower confidence limit (SD_{min}) and +2 for the 95% upper confidence limit (SD_{max}). S is the factor that takes account of the variation in intake, BMR and energy requirements, and is given by:

$$S = \sqrt{\left(\frac{CV^2_{wEI}}{d} + CV^2_{wBMR} + CV^2_{tP}\right)} \quad (5)$$

where d is the number of recording days, CV_{wEI} is the within-subject variation in EI (23%), CV_{wBMR} is the precision of the BMR_{est} relative to the measured BMR (8.5%) and CV_{tP} is the between-subject variation in PAL (15%).

From equations (3) – (5) we can see that the values of the cut-offs varies according to physical activity level (PAL), number of days of food recording and whether the evaluation of EI_{rep}/BMR_{est} is at the individual or group level. Subjects are defined as plausible-, low energy- or high energy reporters from their ratio of EI_{rep}/BMR_{est} according to whether this ratio are within, below or above the 95% confidence limits calculated, respectively.

Black calculated a lower cut-off value of 1.10 and an upper cut-off value of 2.19 assuming a PAL of 1.55, number of days of dietary recording set to infinity (habitual dietary intake measured by an FFQ) at the individual level [(10)]. Therefore, all women with $EI_{rep}/BMR_{est}<1.10$ were classified as low-energy reporters in this study. Thus, we hypothesized a moderately inactive lifestyle for the entire sample to avoid exaggerating the extent of under-reporting [(57)]. The total study sample was defined as all reporters.

3.5 Statistical methods

Characteristics of the study sample were presented using means and SDs for continuous variables, and frequencies (%) for categorical variables. Independent-samples *t* test and Pearson's chi-square test were used to examine group differences for continuous and categorical variables, respectively. Physical activity and alcohol intake were $\log_e$-transformed in these analyses in Paper III.

In all three papers we used PCA to derive dietary patterns. Prior to the PCA the 253 food items were categorised into 46 (Paper I) and 49 (Papers II and III) food groups according to similarity in ingredients, nutrient profile or culinary usage. The food items was grouped before applying PCA in order to reduce the number of variables, since the proportion of

explained variance per principal component decreases with the number of variables entered [15]. Prior to extracting components, the suitability for using PCA was assessed by the Kaiser-Meyer-Olkin measure of sampling adequacy and the Bartlett's test of sphericity which tests whether our correlation matrix is significantly different from an identity matrix [116]. The Kaiser-Meyer-Olkin value was 0.63 in Paper I and 0.76 in Papers II and III, which is above the suggested minimum of 0.50 [117], and Bartlett's test of sphericity was significant (P<0.001), supporting the suitability of the data for PCA. The input variables were standardised by using the correlation matrix of the food group variables in the PCA. To determine the number of meaningful components to retain, we considered the eigenvalue > 1 criterion, the scree test, the proportion of variance accounted for and the interpretability of the patterns [15]. The eigenvalue > 1 criterion is based on the rationale that each factor retained should explain more variance than a single original variable in the data set [84]. However, by using eigenvalue > 1 criterion sixteen dietary patterns should have been retained, which is a number too large for further analysis. The scree plot of the eigenvalues gave us the opportunity to distinguish between the components with relatively high eigenvalues and those with relatively low eigenvalues, since the components before a "break" in the scree plot are assumed to be meaningful. In order to facilitate the interpretation of the components that are considered relevant, a rotation method is usually followed. Rotation can be explained as a variety of methods used to further analyse initial components, aiming to make the factor loadings clearer, more well-defined and, thus reveal a simple structure of the initial information [118]. There are two types of rotation, the orthogonal and the non-orthogonal (oblique) rotation. By using the orthogonal rotation, the rotated components will be orthogonal to each other and the data are believed to be uncorrelated. By using the non-orthogonal rotation, the components are not required to be orthogonal to each other and the data are allowed to be correlated [118]. In dietary pattern analysis, the orthogonal rotation method has been the most commonly used rotation method [5, 22-33, 84, 119, 120] . In this work we applied the orthogonal rotation method to the components, using the varimax type of rotation. We considered food groups with absolute factor loadings ≥0.3 as significantly contributing to a dietary pattern, which is the most applied factor loading cut-off [16]. Factor loadings can be interpreted as correlation coefficients between food groups and dietary patterns [84]. Finally, each woman's factor score was calculated for each of the retained components, by summing the standardized food groups weighted by their factor loadings. The factor scores represent standardised variables with mean = 0 and standard deviation = 1. The factor scores were used to study associations between dietary patterns and plasma carotenoids, self-reported chronic diseases, nutrients, age,

education and risk factors for NCDs. We interpreted the retained components as dietary patterns and labelled them according to the more or less healthy combinations of food groups.

Due to skewed distributions of plasma carotenoids, these were $\log_e$-transformed in the analyses described below.

The correlation between $\log_e$-transformed plasma carotenoids and dietary patterns were estimated by partial correlation coefficients (r_{adj}), adjusting for age (continuous), total energy intake (continuous), BMI (continuous), smoking status (yes/no), intake of vitamin supplements (yes/no) and alcohol intake (continuous), and presented with 95% CIs based on 1000 bootstrap samples (Paper I). The associations between dietary patterns and selected micro-/macro nutrients were estimated by Pearson's correlation coefficient (r), and presented with 95% CIs based on 1000 bootstrap samples (Paper III).

Multiple linear regression analyses were used to examine the associations between $\log_e$-transformed plasma carotenoids (exposure variable) and dietary pattern scores categorized into quartiles (outcome variable) (Paper I), in order to see increasing or decreasing trends. The regression models included the same covariates as in the calculation of partial correlation coefficients. We reported adjusted back-transformed marginal means of plasma carotenoids (μmol/l) with 95% CIs. The associations between dietary pattern scores (exposure variables) and categories of age (50-55, 56-60, 61-65, 66-69 years), smoking (never, former, current), BMI (<18.5, 18.5-24.9, 25.0-29.9, ≥30.0 kg/m^2), education (primary school, secondary school, upper secondary school, academy/college/university ≤ and > four years), and physical activity (quartiles, MET-h/wk) (outcome variables) were also studied by multiple linear regression analyses (Paper III). All variables were adjusted for each other and for energy intake. Regression coefficients (βs) with 95% confidence intervals (CIs) were presented.

A logistic regression model was used to estimate the association between dietary pattern scores (exposure variable) and the prevalence of chronic diseases (outcome variable) among all and plausible reporters (Paper II). The dietary pattern scores were categorized into tertiles and we estimated the adjusted odds ratios (ORs) and 95% CIs for each tertile compared with the lowest tertile of each dietary pattern. We made adjustment for age (continuous), education (categorical), smoking (categorical), physical activity (continuous) and energy intake (continuous). Potential interaction effects were tested.

We analysed trends across categories of a variable by assigning equally spaced values (e.g. 1, 2, 3 or 4) to the categories and treating the variable as a continuous variable in the regression analysis.

All tests were two sided and $P<0.05$ was considered statistically significant. The analyses were conducted using SPSS version 20.0 (IBM Corp., Somers, New York, USA).

4 Summary of results

4.1 Paper I

The aim of this paper was to identify important dietary patterns in Norwegian women aged 50-69 years (n=361), and evaluate these patterns against the plasma carotenoids α-carotene, β-carotene, β-cryptoxanthin, lutein, lycopene and zeaxanthin as biomarkers for fruit and vegetable intake.

After analysing 46 food groups by PCA, the scree plot showed a clear point of inflexion justifying retaining four dietary patterns, all with eigenvalues ≥2.0. The four dietary patterns accounted for 23% of the total variance, and were labelled "Western", "Vegetarian", "Continental" and "High-protein" (Table 3, Paper I).

The "Western" pattern scores were significantly inversely correlated with plasma lutein, zeaxanthin, lycopene and total carotenoids ($-0.25 \leq r_{adj} \leq -0.13$). We found a significant decreasing trend across quartiles of the "Western" pattern scores only for plasma lycopene ($P_{trend} < 0.001$). The "Vegetarian" pattern scores were significantly positively correlated with all the plasma carotenoids ($0.15 \leq r_{adj} \leq 0.24$), and with significant increasing trends across quartiles ($P_{trend} \leq 0.01$). The "Continental" pattern scores were significantly inversely correlated with plasma lutein and α-carotene ($r_{adj} = -0.13$), and we found significant decreasing trends for these carotenoids across quartiles of the "Continental" pattern ($P_{trend} \leq 0.05$). Moreover, this pattern had a non-significant positive correlation with plasma lycopene ($r_{adj} = 0.10$, $P_{trend} = 0.07$).We found no significant association between the "High-protein" pattern scores and the plasma carotenoids.

4.2 Paper II

In this paper we investigated the effect of under-reporting of EI among Norwegian women aged 50-69 years (n=6204) on (a) the dietary patterns derived by PCA and (b) the association between the dietary patterns and self-reported chronic disease.

A total of 1133 (18%) women were identified as low energy reporters. They reported significantly higher BMI, lower physical activity, lower alcohol intake and lower education compared to plausible reporters ($0.001 \leq P \leq 0.02$). They also reported significantly higher prevalence of total chronic disease, cardiovascular disease, diabetes and joint/muscle/skeletal disorders ($0.001 \leq P \leq 0.04$) than the plausible reporters (n=5071).

Forty-nine food groups were analysed by PCA, and three dietary patterns were identified among both all and plausible reporters: "Prudent", "Western" and "Continental" (Table 3, Paper II). The patterns accounted for 17.5% and 16.7% of the total variance among all and plausible reporters, respectively. Among all reporters the "Prudent" pattern was the pattern explaining most of the variance in the dietary data. After excluding the low energy reporters from the study sample the "Western" pattern explained the highest and the "Prudent" pattern the lowest amount of variance in the dietary data. The composition of food groups characterising the three dietary patterns derived from all reporters were mainly the same as those characterising the three dietary patterns derived from plausible reporters.

Due to significant statistical interaction between dietary pattern score and BMI (two categories) with respect to self-reported diseases ($P \leq 0.005$), the results are presented stratified by BMI:

Associations between dietary patterns and self-reported chronic diseases among normal weight women (Table 4, Paper II):

Among plausible reporters the "Prudent" pattern was significantly positively associated with self-reported total chronic disease [odds ratio (OR) for highest compared to lowest tertile: 1.43; 95% CI: 1.14, 1.80; $P_{trend}=0.002$]. Among all reporters the effect estimates were attenuated and no longer significant, however the trend was still significant [OR for highest compared to lowest tertile: 1.24; 95% CI: 1.00, 1.55; $P_{trend}=0.05$]. The "Prudent" pattern was significantly positively associated with joint/muscle/skeletal disorder among both plausible and all reporters, but also here the effect estimates were attenuated among all reporters compared to plausible reporters [OR for highest compared to lowest tertile for; plausible reporters: 1.69; 95% CI: 1.31, 2.18; $P_{trend}<0.001$; all reporters: 1.44; 95% CI: 1.13, 1.85; $P_{trend}= 0.003$]. The "Continental" pattern was inversely associated with

joint/muscle/skeletal disorder among both plausible and all reporters [OR for highest compared to lowest tertile for; plausible reporters: 0.77; 95% CI: 0.62, 0.97; P_{trend}=0.04; all reporters: 0.77; 95% CI: 0.62, 0.95; P_{trend}=0.02], but the trend was weaker among plausible reporters.

Associations between dietary patterns and self-reported chronic diseases among overweight/obese women (Table 5, Paper II):

The "Prudent" pattern was significantly positively associated with total chronic disease, and the OR was only slightly higher in highest related to lowest tertile for plausible reporters compared to all reporters [OR for highest compared to lowest tertile for; plausible reporters: 1.46; 95% CI: 1.14, 1.87; P_{trend}=0.003; all reporters: 1.45; 95% CI: 1.14, 1.84; P_{trend}=0.003]. When we looked at each disease separately, we found that there was a significant positive association between the "Prudent" pattern and cardiovascular disease among both plausible reporters and all reporters [OR for highest compared to lowest tertile for plausible reporters: 1.63; 95% CI: 1.19, 2.23; P_{trend}=0.002; all reporters: 1.69; 95% CI: 1.23, 2.27; P_{trend}=0.001]. The "Prudent" pattern was significantly positively associated with diabetes among both plausible and all reporters [OR for highest compared to lowest tertile for; plausible reporters: 3.82; 95% CI: 1.95, 7.51; P_{trend}<0.001; all reporters: 2.80; 95% CI: 1.62, 4.87; P_{trend}<0.001], and with chronic respiratory disease among plausible reporters [OR for highest compared to lowest tertile: 1.62; 95% CI: 1.09, 2.40; P_{trend}=0.02]. Also, the "Western" pattern was significantly positively associated with cancer among plausible reporters [OR for highest compared to lowest tertile: 1.68; 95% CI: 1.02, 2.77; P_{trend}=0.03]. The effect estimates observed between the "Prudent" as well as the "Western" pattern and diabetes, chronic respiratory disease and cancer among plausible reporters were all attenuated among all reporters. Finally, the "Prudent" pattern was significantly positively associated to joint/muscle/skeletal disorder among both plausible and all reporters, however the effect estimate and P_{trend} was weaker among plausible reporters [OR for highest compared to lowest tertile for; plausible reporters: 1.33; 95% CI: 1.01, 1.75; P_{trend}=0.04; all reporters: 1.44; 95% CI: 1.11, 1.87; P_{trend}=0.007].

The highest effects of under-reporting on the associations between dietary patterns and self-reported chronic diseases were observed among the overweight/obese women.

4.3 Paper III

The aim of this study was to examine dietary patterns among women aged 50-69 years (n=6298) and how the dietary patterns were associated with nutrient intake, sociodemographic factors and key risk factors for NCDs; physical activity, BMI, alcohol intake and smoking.

Forty-nine food groups were analysed by PCA, and the interpretation of the components and the point of inflexion on the curve in the scree plot justified retaining three components, all with eigenvalues ≥2.0. The dietary patterns accounted for 17.5% of the total variance, and were labelled "Prudent", "Western" and "Continental" (Table 1, Paper III).

The "Prudent" pattern was positively associated with protein, fibre, vitamin D, vitamin B_{12}, calcium, iron and magnesium ($0.12 \leq r \leq 0.74$), and inversely associated with saturated fat, carbohydrate and sugar ($-0.24 \leq r \leq -0.13$). An increasing score for the "Western" pattern was associated with increasing intakes of total fat, saturated fat, carbohydrate, sugar, vitamin B12, calcium and magnesium ($0.13 \leq r \leq 0.36$), and decreasing intakes of alcohol, protein and fibre ($-0.37 \leq r \leq -0.17$). An increasing score for the "Continental" pattern was associated with increasing intakes of alcohol, total fat and saturated fat ($0.22 \leq r \leq 0.31$), and decreasing intakes of carbohydrate, fibre and calcium ($-0.36 \leq r \leq -0.14$).

Adherence to the "Prudent" pattern were related to older age, higher education, higher BMI, more physical activity ($P_{trend} < 0.001$), and being a non-smoker ($P < 0.001$). Adherence to the "Western" pattern were related to older age, lower education, higher BMI, less physical activity ($0.001 \leq P_{trend} \leq 0.006$) and lower alcohol intake ($r = -0.28$). Adherence to the "Continental" pattern were related to younger age, higher education, higher BMI, less physical activity, ($P_{trend} < 0.001$) , being a smoker ($P < 0.001$) and a higher alcohol intake ($r = 0.36$).

5 General discussion

The aims of this thesis were to identify and evaluate dietary patterns among Norwegian women aged 50-69 years, and study the association between the dietary patterns and nutrients, sociodemographic factors and risk factors for chronic disease. We also aimed to study the effect of under-reporting of EI on the derived dietary patterns and on the association between dietary patterns and self-reported chronic diseases.

Firstly, the methods used in this thesis will be evaluated by discussing their strengths and limitations (section 5.1). Secondly, the main findings will be discussed and compared with those of other published results (section 5.2).

5.1 Methodological considerations

Several methodological issues may have affected the results in Paper I-III and are important to consider before proceeding to the discussion of results.

Validity in dietary assessment methods describes the degree to which a dietary method measure what it is intended to measure [8]. Dietary assessment methods designed to characterise usual dietary intake over a defined period of time are difficult to validate because the truth is never known with absolute certainty. Therefore, investigators assess relative validity by comparing the new dietary assessment method with a method that are believed to have a greater degree of validity (a gold standard) [8] or it can be validated against an independent, external criterion reference instrument such as a biomarker of intake. Still, measurement errors will be present, and the possible random and systematic measurement errors in the dietary data in this study will be considered in section 5.1.2.

The validity of studies can be separated into internal and external validity. The internal validity refers to the degree to which the study is free from bias in the way data is collected, analysed and interpreted [36, 121]. The three main forms of bias; selection bias, information bias and confounding [36], and how they might have affected the results in the present work will be discussed below. Moreover, random measurement errors in the obtained information about the participants in a study can introduce bias in an effect estimate and will also be considered. Finally, the external validity will be discussed. That is, the degree to which the results can be generalised to populations that did not participate in the study [37].

5.1.1 Study design

A cross-sectional study design was used in the present work. This study design is useful when the aim is to describe a population or a subgroup within the population with respect to a specific exposure, disease or any other health-related event at a particular point in time [36]. Despite the obvious advantages of resource efficiency (time and cost) and the ability to study many variables, the cross-sectional study design does not allow us to draw any conclusions about causality of the direction of effects [36, 121]. However, an observed association may be used for development and specification of hypothesis [121]. In Papers II-III we aimed to investigate associations between dietary patterns and self-reported diseases, sociodemographic factors and risk factors for NCDs, but could not draw any conclusions about causality between exposure and outcome.

5.1.2 Considerations concerning the dietary intake assessment

Measuring dietary intake is a difficult task and different factors can affect the accuracy of the information collected. It is therefore crucial to consider the degree to which dietary assessment methods can measure the true intake. The FFQ has become the most used dietary assessment method in large-scale epidemiological studies, because it measures the habitual food intake, is relatively inexpensive, easy to administer and easy for participants to complete [8]. The validity of a FFQ is often evaluated by comparing the method with a more accurate method, usually diet records because these are likely to have least correlated errors [8]. Both these methods will introduce measurement errors, therefore, to avoid incorrectly high estimates of validity it is important that the errors are as independent as possible.

The 253-item FFQ used in this study has not been validated, but it was based on a previously validated 270-item FFQ designed to measure the total energy intake and cover the most antioxidant-rich foods and beverages in Norway [109]. The 253-item FFQ was created by removing 17 food items from the 270-item FFQ that were seldom or never eaten (see section 3.2.1), and we assumed that this would have minimal effect on the validity.

Measurement errors might have occurred during the assessment of dietary intake in this study. For example, the FFQ had an extra focus on fruit, vegetables, antioxidant-rich foods and beverages, therefore an overestimation of these foods might have occurred. Also, the FFQ might have covered less of the total diet of some individuals than others due to the closed food list. Furthermore, participants may unintentionally have omitted or added foods because of memory lapses. Incorrect estimation of portion size is another measurement error

that can have occurred which arise from participants failing to quantify accurately the amount of food consumed, or from misconceptions of an "average" portion size. Some participants might also have omitted supplement usage from the FFQ, causing measurement error in the calculated nutrient intakes. Measurement errors could arise when daily intake of energy, nutrients and foods were computed using the food database AE-07 and KBS software system. In a food database the values in the composition table is an average value of a small sample of each food. Only a small fraction of the table values is based on analytical values and some values are calculated from recipes [122]. Also, database values may differ from nutrients in the food actually consumed due to seasonal variations.

Random error in the dietary assessment might have led to biased effect estimates between for example dietary patterns and risk factors for NCDs and self-reported chronic diseases. Such bias would most likely have been towards the null value.

Previous research has revealed extensive misreporting, especially under-reporting, of self-reported dietary intake [123-126]. The misreporting can be general under-reporting of food intake, or under- or over-reporting of certain food groups related to social desirability [127-129] and will introduce systematic measurement errors in our dietary data. Under-reporting can involve both under-recording and undereating. Under-recording is defined as a discrepancy between reported energy intake and measured energy expenditure without any change in body mass [130]. Undereating occurs when participants eat less than usual or less than required to maintain body weight, and is accompanied by a decline in body mass [130]. Previous investigations of the characteristics of under-reporters have shown that women [63-66, 130], the elderly [62, 64-66, 130] and overweight individuals [62, 63, 65, 130-132] are more likely to under-report their EI. A systematic over-reporting of healthy foods and/or under-reporting of unhealthy foods by a selection of women in our study might have introduced bias into the effect estimates in paper II and III.

In Paper II we investigated the magnitude of measurement errors due to under-reporting of EI by using the revised Goldberg cut-off method [10]. The sensitivity of this method increases if it is possible to assign participants to low, medium and high physical activity categories based on the total amount of physical activity. Unfortunately, only recreational light, moderate and vigorous physical activity was assessed, and not occupational or household physical activities, which are important contributors to total energy expenditure. If it had been possible to calculate three different cut-offs based on three different PALs, we might have found a higher prevalence of low energy reporters in Paper II [10].The questionnaire used in the present work was designed for a study on diet and breast cancer.

Recreational physical activity has been found to have the strongest association with risk of breast cancer, hence the questions about physical activity in the questionnaire was designed to capture the women's total amount of recreational physical activity[133]. In the present work, we chose to use the theoretical value of PAL proposed by FAO/WHO/UNU for a sedentary lifestyle population (1.55) [134] in order to not over-estimate the extent of under-reporting. Therefore, we assume that a misclassification of more active participants could exist.

To summarise this section, it is important to be aware that dietary data collected by FFQs contains measurement errors, both random and systematic, and it is difficult to predict how this will influence the characterisation of the dietary patterns. These measurement errors in the dietary intake might have led to bias in the effect estimates calculated in Paper II and III.

5.1.3 Considerations concerning dietary patterns derived by PCA

When defining *a posteriori* dietary patterns, one of the major methodological concern is the subjective decisions made by the researcher at various points in the analysis [17]. For example, the decision of whether or not grouping the foods, how to group foods, and whether to energy adjust the food/food groups or not before the analysis. Also, in PCA, there are decisions to make for type of rotation of the components, and for which cut-off to use for food group factor loadings [3, 34]. Furthermore, the Kaiser criterion, scree test and interpretability, i.e., criteria that either tend to over-extract and/or are subjective , are commonly used to determine the number of dietary patterns to retain [17, 34]. Finally, naming the patterns also involves subjectivity, and they have previously been named either using quantitative criteria, i.e., being named after the food groups with the highest factor loadings, or qualitative criteria, i.e., being named after the nutritional quality of the food groups with highest factor loadings [5]. Given these subjective decisions by the investigators, the reproducibility of the dietary patterns and thereby comparison across studies are a concern.

In this work, the unrotated pattern solution showed a “Western” and a “Prudent” pattern that shared many of the same high loaded food groups (absolute factor loading ≥ 0.3) with the “Western” and “Prudent” patterns in the rotated pattern solution. However, the third pattern derived in the unrotated compared to the rotated pattern solution had a mix of highly loaded food groups from the “Continental” and the “Prudent” pattern found in the rotated pattern solution. We chose to use the orthogonal varimax rotation method in order to improve the interpretability of the dietary patterns and thus, found one healthy and two unhealthy patterns. The effects of rotation methods on composition and interpretability of dietary

patterns have been shown to differ according to the factor loading cut-off used, and that less remarkable differences has been found by applying higher cut-offs, i.e., ≥ 0.25 [120].

We categorised the 253 food items into 46 (Paper I) and 49 (Papers II and III) food groups based on similarity in ingredients, nutrient profile or culinary usage. We found that the first three patterns derived in Paper I ; the "Western", "Vegetarian" and "Continental" patterns, was very similar to the "Western", "Prudent" and "Continental" patterns derived in Paper II and III, respectively. We chose to use a high number of food groups in order to increase the level of detail in the dietary patterns. As a result, the total variance explained was quite low (23% in Paper I, 17.5% and 16.7% in Paper II and 17.5% in Paper III) , since it generally decreases with increasing number of food group variables being entered into the PCA [135]. Aggregation of food items in more broadly classified food groups have been reported to increase the variance explained by the dietary patterns [5, 135]. Schwerin et al. [136] reported that their dietary patterns explained 55.3% of the variance in the dietary data, which is relatively high. However, the analysis was based on only 15 food groups, from which seven dietary patterns were retained. The dietary pattern solutions (number of patterns retained) in our study represent a much higher variable reduction, i.e., from 46/49 food groups to 4/3 dietary patterns. The explained variance depends largely on the degree to which the variable are reduced, and one cannot conclude that a study with a higher percentage explained variance but with a lower variable reduction is more precise than a study with a lower percentage explained variance but with a much higher variable reduction [84]. In studies investigating associations between dietary patterns and disease it might be more important with a greater detail in food intake information rather than variance explained by the dietary patterns. McCann et al. [135] suggested that by using broadly classified food groups, foods weakly associated with a pattern may be classified in the same broad category as foods more strongly associated, thus increasing the amount of information that a specific pattern might capture. These researchers investigated associations between endometrial cancer and dietary patterns generated from 168 food items, 56 food groups and 36 food groups. They found that when the level of aggregation of food items increased, the association between dietary patterns and endometrial cancer was attenuated and the confidence interval became wider [135]. An explanation was that, in a multidimensional exposure such as diet, greater detail in the food group variables entered into the PCA may be necessary to adequately capture differences in dietary exposure between diseased and non-diseased subjects, at least when there is a true diet-disease relationship.

We considered different dietary pattern solutions, and found that a dietary pattern accounting for less than 5% of the variance in the dietary data was not interpretable, i.e., did not make sense. This interpretation together with the visual inspection of the scree plot resulted in a 4-pattern solution in Paper I and a 3-pattern solution in Paper II and III. Previous studies have retained patterns accounting for as little as 2% or 4% of the variance in dietary intake [137, 138], and it could be discussed if these patterns are of truly substantive importance, especially if they have just a few food groups with high factor loadings. It might be a better solution to retain fewer patterns with a higher amount of variance explained [17]. Most studies that have derived patterns with PCA, have found the patterns to describe between 13% and 30% of the variance in the dietary data [3]. Thus, the dietary patterns derived will represent the optimal model with respect to the explained variance, but leave sufficient room for other patterns to prevail in the study population [15].

We assume that the higher explained total variance in dietary data by the four dietary patterns in Paper I compared to the variance explained by the three patterns in Paper II and III is due fewer food group variables into the PCA in Paper I and that a four pattern solution was chosen.

In Papers II and III we decided to split the following food groups from Paper I: “fish” into “fish, dinner” and “fish, bread spread”; “fruit and berries” into “fruit” and “berries”; “herbs and spices” into “herbs and spices” and “barbecue and taco seasoning”, resulting in 49 food groups in contrast to 46 in Paper I. This may have influenced the derived dietary patterns in Paper II and III, and is important to have in mind when comparing the food groups in these patterns to those in Paper I. Furthermore, we decided not to energy adjust the food group variables before entering them into the PCA, since several studies have reported that this will not have any major impact on the association between the derived dietary patterns and the outcome [7, 22], and that it is sufficient to make energy adjustment when analysing the effects of the dietary patterns on the outcome of interest.

We used carotenoids as dietary biomarkers in Paper I as a mean of evaluating the dietary patterns, and the associations between plasma carotenoids and dietary patterns were in the expected directions. In Papers II and III we tested the robustness of our dietary patterns by randomly splitting the dataset in two halves and conducted the PCA in each of these samples. In both Paper II and III the same components were extracted from the split samples with food groups having only minor differences in factor loadings. This was not possible in Paper I, since the sample sizes in each half would be too small to derive meaningful dietary patterns by PCA [16]. We found only minor differences in the food group composition characterising

the dietary patterns after excluding the low energy reporters from the study sample (Paper II). Interestingly, the dietary pattern explaining the highest extent of variance in the dietary intake differed between all and plausible reporters, with the "Prudent" pattern explaining the highest extent of variance among all reporters and the "Western" pattern explaining the highest extent of variance among plausible reporters. This may be related to the fact that low energy reporters tend to over-report foods perceived as healthy or/and under-report foods perceived as unhealthy [61, 64, 129, 139] as described in section 5.1.2. From Paper I we concluded that the "Western", "Prudent" and "Continental" dietary patterns were meaningful according to the associations between the patterns and plasma carotenoids. From Paper II and III we concluded that the derived dietary patterns were robust on the basis of (a) the rerun of PCA on the two split samples and (b) that we found only minor differences in the food groups characterising the dietary patterns among all reporters and plausible reporters. In Paper I and III, the low energy reporters were not identified and the effect of under-reporting on the dietary patterns derived in these studies was not investigated.

5.1.4 Considerations concerning disease assessment

The participants were asked to report diseases they had currently or previously been diagnosed for. The purpose of asking subjects to report these diseases were to be able to exclude individuals with prevalent disease from future follow-up studies of diet and incident cases of those diseases. We did not ask for independent verification of the disease status, and therefore do not know anything regarding the accuracy of the self-report. If there was measurement error in the disease assessment, it may have been nondifferential with respect to dietary patterns. In which case the result would most likely have been an underestimation of the effect estimate between the dietary pattern and the self-reported chronic disease in Paper II. On the other hand, we cannot exclude the possibility of differential measurement errors in this cross sectional design. For example, if women reporting a high adherence to the prudent pattern were more likely to report (or not report) a disease than women having a low adherence to the prudent pattern. Then the effect estimate between the dietary patterns and self-reported diseases in Paper II might have been biased, but it is difficult to predict the direction of the bias.

5.1.5 Participants – selection bias

Selection bias is a systematic error in a study described as distortions that result from procedures used to select subjects and from factors that influence participation in the study [37]. As a result, the association between exposure and outcome differs between participants and non-participants (those who should be theoretically eligible for the study) in the study [36].

The participation rate of women aged 50-69 years in every screening round in the Norwegian Breast Cancer Screening Program is 76% [104]. Of these 76% a total of 10,000 were invited to participate in the present study and the participation rate was 70%. A common source of selection bias is self-selection bias. This can occur when people volunteer to participate in a study, like in the present study. A moderate response rate can make the study vulnerable to self-selection bias, and especially interpretation of estimated prevalence data must be done with care. We know that the proportion of smokers was slightly lower in our study compared to all eligible Norwegian women aged 50-69 years (Table 1). Also, it seems as if there is a somewhat higher proportion of women with higher education (>4 years) in our study sample than among the female population in Norway aged >16 years (Table 1). The associations between the exposure variables and various outcomes in Paper II and III will only be biased if the non-responders differ from the responders not only on the exposure variable (e.g., smoking) but also on the outcome status (e.g., high adherence to the "Continental" pattern). That is, smokers respond less to the study than non-smokers, and those of the smokers who do respond have a different adherence to dietary patterns than smoking non-responders. If that was the case, the effect estimate between smoking and dietary patterns may be biased.

The response rate in our study was high and, even if there was small differences in the proportion of smokers and those with higher education among responders and non-responders, there was no strong indications that the responders in this study was systematically different from the non-responders. However, the prevalence data should be interpreted with the differences in smoking and education in mind.

5.1.6 Information bias

Bias in estimating an effect can be caused by measurement errors in the assessment of exposure or outcome, and is referred to as information bias [36].

In our cross-sectional study, all participants were provided with identical questionnaires and information on exposures and outcomes was collected at the same time.

There was no personal contact between researcher and participant. All exposure and outcome variables in this study are self-reported (except from the plasma carotenoids) and therefore susceptible to information bias.

Questions about dietary intake and disease was presented in the same questionnaire, therefore, the women having a disease might have recalled their diet differently than those who did not have a disease. This could have introduced differential measurement errors in the dietary data and it is difficult to predict in which direction this would have biased the effect estimate between dietary patterns and self-reported diseases (Paper II).

Random measurement errors in the exposures in Paper II (dietary patterns) and Paper III (age, education, BMI, physical activity, smoking) might have led to nondifferential misclassification of participants in the different categories within each exposure. In general such random measurement error would be expected to lead to a bias towards the null value [36]. However, for exposure variables with more than two categories, such random measurement error can under certain conditions also result in bias away from the null value [36].

If the overweight/obese women in this study systematically over-reported healthy food and under-reported unhealthy food or systematically under-reported EI, this misreporting would most likely have biased the effect estimate between dietary patterns and self-reported chronic diseases towards the null value in Paper II.

5.1.7 Confounding and statistical interaction

A confounder is a variable that is 1) associated with the outcome (either as a cause or a proxy for a cause, but not as an effect of the outcome), 2) associated with the exposure, and 3) not an effect of the exposure [36, 121]. The presence of confounding will lead to bias in the effect estimate between the exposure and the outcome. It is essential to adjust for confounding in the statistical analysis of observational studies if data on confounders are measured [121].

Several factors such as age, BMI, smoking, alcohol intake and use of dietary supplements have been reported to affect the concentrations of carotenoids in plasma [49-52]. In Paper I we found that these factors were also associated with dietary patterns (exposures), but not an effect of the dietary patterns, and therefore potential confounders of the association between dietary patterns and plasma carotenoids (outcomes). Hence, age, BMI, smoking, alcohol intake and use of dietary supplements were adjusted for in the correlation and regression analyses of dietary patterns and plasma carotenoids in Paper I.

In Paper II we investigated the association between dietary patterns and self-reported chronic diseases. In the multivariable method dietary patterns were the exposures and self-reported chronic diseases the outcomes. Age, education, physical activity and smoking are associated with dietary patterns and the self-reported chronic diseases but not an effect of either of them, and considered to be potential confounders and we adjusted for them in the regression analysis.

In Paper III we investigated the association between sociodemographic factors, risk factors for NCDs and dietary patterns. In the multivariable method age, education, BMI, physical activity and smoking were the exposures and dietary patterns the outcomes. All the exposures were factors that can be associated with dietary patterns, and with each other, therefore, all of these factors are potential confounders and they were all adjusted for in the regression analysis.

Associations between dietary patterns and plasma carotenoids, sociodemographic factors, risk factors for NCDs or chronic disease might be confounded by differences among the participants in total energy intake resulting from differences in body size, physical activity and metabolic efficiency. Total energy intake was therefore also included as a potential confounder in the regression (Paper I-III) and correlation analyses (Paper I).

In this study (Papers I-III) we attempted to adjust for potential confounding in the statistical models, however, since there is a possibility of measurement errors in the confounders we cannot rule out the possibility of residual confounding, i.e., confounding that persists after adjustment for the putative confounders [37].

Statistical interaction (effect modification) occurs when the magnitude of the effect of the primary exposure on an outcome differs depending on the level of a third variable, and can be handled in the statistical analysis by using stratification or a multivariable method [36]. In Paper II, we tested for statistical interaction between dietary patterns and BMI (two categories) with respect to self-reported chronic diseases, and found a statistical significant interaction. As a consequence the analysis of the associations between dietary patterns and self-reported disease was performed on two separate strata of BMI (normal weight and overweight/obese).

5.1.8 External validity

External validity is the degree to which results of a study may be generalised to populations that did not participate in the study [36]. Internal validity is a prerequisite for external validity.

The results of this thesis were based on studies of a large sample size of women aged 50-69 years, recruited from women who attended mammographic screening. We know attenders have better health than non-attenders, and it is also possible that those who responded to the questionnaires for our studies represented a selected group. Our study sample included somewhat fewer smokers and more highly educated women compared to the general Norwegian female population (Table 1). More specifically, we found that the proportion of smokers among all eligible Norwegian women in the age groups 45-54, 55-64 and 65-74 was 27%, 22% and 18%, respectively (Table 1, Source: Statistics Norway). Since the majority in our study population was aged 50 to 60 years, it seems as if there were fewer smokers in our study population than in the general female Norwegian population in the same age group. Data for education among Norwegian women aged 16 years and above in 2008 showed a lower proportion of highly educated women (>4 years) than in our study population. However, no data were available for the specific age group 50-69 years. One could argue that our study population was not a representative sample of the general female population aged 50-69 in Norway. To what extent this affected the dietary patterns, is unknown.

Table 1. Proportion of smoking and level of education among Norwegian women in 2008

	Norwegian women[1]	**Study population**
Smoking (%)		
Age 50-69 years	-	20
Age 45-54 years	27	-
Age 55-64 years	22	-
Age 65-74 years	18	-
Education (%)[2]		
Primary and secondary school	30	20
Upper secondary school	40	40
Academy/college/university (≤4y)	24	24
Academy/college/university (>4y)	5	16

[1] Source; Statistics Norway (www.ssb.no)
[2] Data from Statistics Norway are women aged 16 years and above

5.2 Discussion of main findings

5.2.1 Major dietary patterns derived by PCA among women aged 50-69

In Paper II and Paper III PCA was conducted on the total study population and three dietary patterns were derived: the "Prudent", "Western" and "Continental". In Paper I, PCA was conducted on a smaller subsample of the study population, and four dietary patterns were derived: the "Western", "Vegetarian", "Continental" and "High-protein". The "Prudent" and the "Vegetarian" were the patterns perceived as healthy, while the "Western" and the "Continental" were the patterns perceived as less healthy. In this section the dietary patterns found in Paper I-III will be discussed in light of dietary patterns derived by PCA among women in the western world.

In most studies investigating dietary patterns, a healthy and a less healthy dietary pattern have been identified and are frequently named the "Prudent" and the "Western" pattern, respectively [2, 5, 8]. The "Prudent" pattern in Paper II and III was characterised by typical healthy food groups like fruits, vegetables, legumes and fish [5]. While the "Western" pattern was characterized by more unhealthy foods like red meat, processed meat, refined grains, potatoes and sugar. This corresponds to the food groups other researchers have found to characterise their "Prudent" and "Western" patterns [5].

Several Nordic studies have presented dietary patterns in women derived by PCA [80, 83, 88, 95, 140-143]. Two Swedish [80, 141] and two Danish [88, 140] studies identified a healthy and an unhealthy pattern as the first two patterns derived from the PCA, just as in our study. In a Norwegian study [83], the first pattern derived was dominated by fish and fish products, but it was not characterized as a healthy pattern because of the high loadings of "melted fat on fish", "fatty sauce on fish" and "fat on bread". The second pattern derived in that study was a healthy pattern corresponding to the "Prudent" patterns in our studies. In a Scandinavian study [95], the first pattern derived from the Norwegian cohort was the "Cereal" pattern, which was a pattern characterised by fruit, yoghurt and cereals, but not vegetables and fish which are considered to be typical food groups in a "Prudent" pattern. The "Cereal" pattern was perceived as the healthy pattern and described as a "common Scandinavian whole grain pattern" [95]. The second pattern in the Norwegian cohort in that study resembled a typical "Western" dietary pattern except for the high loadings on whole grains. The "Western" pattern in Paper I in the present thesis was also characterised by whole grains. In Norway, even high fat eaters consume a high amount of whole grains [144], which can explain the high

factor loading for this food group in the unhealthy patterns in our study and in the Norwegian cohort in the Scandinavian study [95].

Also in other European studies, dietary patterns corresponding to the typical "Prudent" and "Western" patterns have frequently been found to be the first two patterns derived from the PCA [17, 84, 145-150]. However, in a French study [138] the investigators derived six dietary patterns, where the first three patterns were the healthy patterns and the last three patterns were the unhealthy ones. Had the researchers in that study chosen to extract fewer dietary patterns from the PCA, it might be that a healthy and an unhealthy pattern had been the first two patterns to be derived. In a Dutch study [17] the researchers found that going from a 2-pattern solution with a "Western" and a "Prudent" pattern to a 4-pattern solution resulted in a subdivision of each of these patterns giving two "Western" patterns and two "Prudent" patterns.

As in European studies, US and Australian studies [151-156] have reported "Prudent" and "Western" patterns to be the patterns explaining the highest variance in the dietary intake among women.

In summary, it seems as if the healthy "Prudent" and the unhealthy "Western" patterns, characterized by some typical food groups, are two dietary patterns that are universal among women. However, it is important to be aware that these patterns may also contain food groups differing between populations due to differences in dietary habits, dietary assessment methods, and the subjective decisions taken by the investigator when deriving dietary patterns by PCA as described in section 5.1.3. This is important to have in mind when comparing patterns across populations.

In addition to healthy "Prudent" and unhealthy "Western" patterns, other patterns have frequently been observed like patterns high in desserts or sweets and patterns high in alcoholic beverages [5]. Dietary patterns that are less distinct and country, culture and population specific may also appear. The third pattern identified in the present work was the "Continental" pattern which was characterised by food groups like tomato sauce, pasta, fat-rich potatoes, salty snacks, pizza, processed meat, red meat, sweets and wine. Comparable patterns have been reported in other female populations in Norway, Denmark, Finland and the US [83, 88, 142, 154]. The "Continental" pattern reflects the change in eating and drinking habits in Norway during the last decades towards more continental habits, which might be related to a general increase in the standard of living and more travelling abroad [157].

5.2.2 Dietary patterns and associations with dietary biomarkers

Evaluating dietary patterns by examining the relationship between the patterns and dietary biomarkers in serum/plasma provides us with useful information as to whether these dietary patterns are meaningful. Patterns consistent with a healthy diet have been reported to be positively associated with serum vitamin C, folate, most carotenoids, and vitamin E [30, 41-44, 158]. Several index-based dietary patterns have also been reported to be consistently positively associated with serum biomarkers of fruit and vegetable intake [41, 159-161]. In Paper I, we investigated the associations between the derived dietary patterns and plasma carotenoids in order to evaluate the patterns, and found a positive association for the healthy "Prudent" pattern and an inverse association for the less healthy "Western" and "Continental" patterns. These results correspond to what have been found in other studies [30, 44, 162]. The studies that used cluster analysis to derive dietary patterns reported a lower concentration of plasma carotenoids in participants belonging to the less healthy cluster compared to the healthy cluster [44, 162]. Based on our findings we concluded that our "Prudent", "Western" and "Continental" patterns were meaningful patterns. However, we did not find any association between the "High-protein" pattern and plasma carotenoids, even if vegetables had high loading in this pattern and it could therefore be discussed if the "High-protein" pattern was a meaningful pattern.

5.2.3 The effect of under-reporting of energy intake on the associations between dietary patterns and self-reported chronic disease

Participants conscious or unconscious urge to leave out food items from a dietary assessment could be the reason for under-reporting of EI, or it could be due to under-eating or dieting in the study period. Therefore, in the present work the term low energy reporters, and not under-reporters, have been used for this group of subjects. Also over-reporting or over-eating has been shown [59], and the term high energy reporters have been used for this group.

Studies performed in industrialised countries tend to identify a high prevalence of low energy reporters and a low prevalence of high energy reporters [70, 76, 131, 139, 163, 164]. This is also what we found in our study (Paper II), where the proportion of low energy reporters and high energy reporters were 18% and 4%, respectively. Comparable prevalence has been reported in other European populations when a similar methodology was used [63, 131, 165, 166]. Due to the marginal over-reporting in our study, we focused on the effects of under-reporting.

The low energy reporters in this study reported higher BMI than the plausible reporters and as much as 63% of the low energy reporters were categorised as overweight/obese. They also reported lower physical activity, lower alcohol intake, lower education and higher prevalence of chronic diseases than the plausible reporters. Our results corresponds to those reported in previous studies investigating characteristics of low energy reporters [11, 61, 64, 69, 129, 139, 167]. Several studies have found that over-reporting of foods perceived as healthy or/and under-reporting of foods perceived as unhealthy are typical characteristics among low energy reporters [61, 64, 129, 139]. This was probably present among the low energy reporters in our study as well.

Results showed that the associations between dietary patterns and self-reported chronic diseases generally became stronger when analyses were restricted to plausible reporters, and especially among the overweight/obese women. Particularly the associations between the "Prudent" pattern and self-reported chronic diseases strengthened. The positive relationship between the "Prudent" pattern and several of the chronic diseases indicated that the participants tried to eat healthy in order to reduce either the symptoms of their condition, or reduce the likelihood of possible detrimental consequences. Positive relationship between a healthy dietary pattern and disease has also been reported in a Swedish study, where the highest prevalence of previously known health problems was observed in the healthy "Fruit & vegetables" cluster among women [168].

To the best of our knowledge no studies have investigated the effect of under-reporting on the association between dietary patterns derived by PCA and health outcomes. In a study investigating the association between dietary patterns derived by cluster analysis and risk of major coronary events, diabetes and mortality among participants in the Whitehall II study, adjustments were made for energy misreporting (both over- and under-reporting) and several other potential confounders [169]. The researchers in that study did not find an association between energy misreporting and healthiness of a dietary pattern, and they reported that only small changes in hazard ratios were found when energy misreporting were adjusted for. In two other studies among participants in the Whitehall II study, adjustment for energy misreporting were made in their analyses of associations between dietary patterns derived by reduced rank regression analysis and (1) insulin resistance and incidence of type II diabetes [170] and (2) blood lipids [171]. In neither of those studies the effect of misreporting on the associations between dietary patterns and (1) and (2) were reported.

A Swedish study investigated the effect of under-reporting on the association between risk of breast cancer and alcohol intake [172]. The researchers reported an increased risk of

breast cancer with high alcohol intakes, and the risk estimates were strengthened among the plausible reporters compared to all reporters. A study in the US [173] investigated the use of calibrated energy consumption to account for under-reporting. They produced the calibrated consumption estimates based on calibration equations developed in a substudy among 544 women where DLW was used to estimate total energy expenditure and urinary nitrogen was used as recovery biomarker for protein [174]. The researchers investigated the association between risk of breast, colon, endometrial and kidney cancer and calibrated and uncalibrated energy consumption.They found calibrated energy consumption to be positively associated with the risk of breast, colon, endometrial, and kidney cancer, whereas uncalibrated energy consumption was not.

Our results and those of other studies show that it is important to consider the under-reporting in dietary studies and the effect this might have on associations between dietary patterns and health outcomes.

5.2.4 Dietary patterns and nutrient intake

The dietary patterns were related to the estimated intake of selected macro- and micronutrients which indicated the dietary quality of the patterns (Paper III). Results showed that the "Prudent" pattern was positively correlated with protein, fibre, vitamin D, vitamin B_{12}, calcium, iron and magnesium and inversely correlated with saturated fat, carbohydrate and added sugar. The "Western" and "Continental" patterns had positive correlations with total fat and saturated fat, and inverse correlations with protein and fibre. The "Western" pattern was also positively correlated with carbohydrate and sugar. The micronutrient profiles of the "Western" and "Continental" patterns differed. An adherence to the "Western" pattern was significantly positively correlated with vitamin B_{12}, calcium and magnesium. An increasing score for the "Continental" pattern was significantly inversely correlated with calcium, but no significant associations were found between the other micronutrients and this pattern. Other studies have also reported that an adherence to a healthy or unhealthy pattern was associated with a favourable or less favourable nutrient intake, respectively [3, 80, 88, 175].

According to the Nordic Nutrition Recommendations [176] the following set of food selection changes have been identified to promote health and wellbeing: Decrease energy density, increase micronutrient density and improve carbohydrate quality; improve dietary fat quality; limit processed and red meat; limit the use of salt. Women with a high adherence to

the “Prudent” pattern and low adherence to the “Western” and “Continental” patterns in our study seem to comply with these recommendations.

In summary, the observed associations between the nutrients and dietary patterns in this work indicate the dietary qualities one would expect of healthy and unhealthy dietary patterns, suggesting that they may represent a relevant dietary exposure.

5.2.5 Dietary patterns and sociodemographic factors and key risk factors for NCDs

In Paper III, we found that a high adherence to a “Prudent” and a “Western” pattern was associated with older age, while a high adherence to a “Continental” pattern was associated with younger age. Our findings that different patterns are differently related to age are consistent with previous studies. For example a Swedish study among adult women and men [80] identified a “Healthy” and a “Swedish traditional” pattern that corresponded to our “Prudent” and “Western” patterns, which were also associated with older age. In another study using data from the Swedish mammography screening cohort [177], the investigators found that age was inversely associated with their “drinker” dietary pattern, which had some food groups in common with our “Continental” pattern. In contrast to our study, no associations were found between the “Healthy” and “Western” patterns and age in that study. In a study using data from the Danish national survey of diet and physical activity [88], the investigators found no significant associations between the more unhealthy “Traditional” and the “Health conscious” patterns and age. However, they reported an inverse association between age and their “fast food” pattern, which was comparable to our “Continental” pattern. Furthermore, it is quite striking that many previous studies have found a positive association between a healthy pattern and age [150, 154, 178-180].

In this study we found that women with high adherence to the “Prudent” pattern were more highly educated, less likely to smoke and more physical active than those with low adherence to this pattern. Women with high adherence to the “Western” pattern had lower education, lower alcohol intake and lower physical activity compared to those who had a low adherence to this pattern. Finally, women with high adherence to the “Continental” pattern were more highly educated, more likely to smoke, had a higher alcohol intake and lower physical activity than those with a low adherence to this pattern. All three patterns were associated with a higher BMI. The unexpected increasing BMI with an increasing score for the “Prudent” pattern may indicate that overweight women adopt a healthy diet to lose weight.

Another explanation could be an over-reporting of foods considered “healthy” and under-reporting of foods considered “unhealthy” related to social desirability among those with a higher BMI as discussed in section 5.1.2.

Healthy patterns has been found to be inversely associated with smoking and positively associated with physical activity and vice versa for the unhealthy patterns [84, 150, 154, 179-182]. Also, numerous studies have reported that more healthful patterns have been associated with more highly educated participants and less healthful patterns have been associated with lower education [3, 88, 137, 183, 184]. Interestingly, we found that the less healthy “Continental” pattern in our study were positively associated with education and alcohol intake, with wine as the main source. This could indicate that women with high adherence to this pattern had a higher socioeconomic position. Higher education is associated with a higher income, i.e. a higher socioeconomic position, and previous studies have found a positive association between a higher socioeconomic position and alcohol consumption [185-187]. Dietary patterns with high loadings for alcoholic beverages has also been found to be positively associated with smoking [84, 93, 178], and are consistent with our findings for the “Continental” pattern.

The “clustering” of unhealthy and healthy behaviours indicates that dietary patterns might interact with other lifestyle behaviours and these interrelationships increases the confidence that the dietary patterns are meaningful.

6 Conclusions

I. Four dietary patterns, the “Vegetarian, “Western”, “Continental” and “High-protein”, was identified in a smaller subset of the study sample of Norwegian women aged 50-69 years. We found that the healthy “Vegetarian” pattern was positively associated with all the plasma carotenoids, while inverse associations were observed between the less healthy “Western” and “Continental” patterns and most of the plasma carotenoids. Therefore, we concluded that these patterns were meaningful. No associations were found between the plasma carotenoids and the “High-protein” pattern, despite the high loading for vegetables in this pattern. Therefore, it can be discussed if this was a meaningful pattern.

II. In the large study sample of women aged 50-69 years, we identified three dietary patterns among both all and plausible reporters: the “Prudent”, “Western” and “Continental” pattern. Under-reporting of EI did not alter the food group composition characterising the “Prudent”, “Western” and “Continental” patterns, but it altered somewhat the food group loadings, and thereby also the women’s pattern scores. The under-reporting attenuated the associations between dietary patterns and self-reported chronic diseases, especially among overweight/obese women. This suggests that it is important to consider the effect of measurement errors resulting from under-reporting on the estimated association between dietary patterns and disease.

III. Three dietary patterns were identified in the large study sample of women aged 50-69 years: the “Prudent”, “Western” and “Continental” patterns. While high adherence to the healthy “Prudent” pattern was associated with a favourable nutrient profile, high adherence to the less healthy “Western” and “Continental” patterns were associated with mostly unfavourable nutrient profiles. Women with high adherence to the “Prudent” pattern were older, more highly educated and had a generally healthy lifestyle. Women with high adherence to the “Western” pattern were older, had lower education and, except for having a low alcohol intake, had a generally unhealthy lifestyle. Finally, women with

high adherence to the “Continental” pattern were younger, more highly educated, and had a generally unhealthy lifestyle. These results indicate that dietary patterns interact with other lifestyle behaviours.

7 Future perspectives

A large body of evidence now shows that changing the modifiable behavioural risk factors for noncommunicable chronic diseases can help people achieve and maintain good health and reduce the risk of chronic disease throughout all stages of the lifespan [188]. An important part of a complex solution to promote health is the national dietary guidelines. These guidelines serve as the evidence-based foundation for nutrition education which can help people to choose foods that provide a healthy diet. The new Dietary guidelines for Americans 2015-2020 [188] lay particular stress on the importance of focusing, not on individual nutrients or foods in isolation, but on healthy dietary patterns as a whole to bring about lasting improvements in individual and population health.

The various approaches (*a priori* and *a posteriori*) for studying the whole diet are complimentary. The *a priori* approach such as dietary quality scores can be useful tools to monitor the overall adherence to dietary guidelines, and the dietary quality of a population. Further insight into the protective role of the dietary recommendations against diseases can also be gained with this method [2]. The *a posteriori* methods such as principal component analysis and cluster analysis, are independent of definitions of what is a healthy dietary pattern and have the advantages related to studying existing dietary behaviour in a population or identification of new dietary patterns that may affect disease risk [189]. The *a posteriori* approach might be especially useful if many dietary components are relevant for a disease. Such insight can provide information for setting priorities for changing dietary patterns in a population by public health initiatives [2].

Application of dietary pattern analyses might not be appropriate in situations where the effect is caused by one specific nutrient or food, for example fruit and vegetables, since their effect will most probably be diluted.

Future studies of a single nutrient, food or food group could use dietary patterns as covariates [84]. That is, the confounding by the overall diet can be addressed by adjusting for dietary pattern scores, in order to establish whether the nutrient/food/food component-related effect is independent of overall dietary patterns.

Among the methods used to determine dietary patterns *a posteriori*, PCA is the most frequent method used in epidemiological studies. It is a fact that the dietary patterns derived by PCA explain a limited extent of the variance in the dietary data and that there still remain important dietary habits that account for a considerable proportion of between-individual

variation in dietary intake. Still, the patterns derived represent the optimal model with respect to the explained variance, which could be important dietary patterns existing in the population.

Because of the subjective decisions made by investigators when deriving dietary patterns, it is challenging to compare patterns across studies. Therefore, in future studies it is of high importance that investigators report as much detail as possible in how all decisions were made when deriving the dietary patterns. That is, from describing the FFQ till grouping of the dietary data and ending with how patterns are defined and named.

The problems of misreporting of energy intake associated with self-reported dietary data transfers to the obtained dietary patterns, and could be one possible explanation for conflicting and/or inconsistent results when studying associations between dietary patterns and health outcomes. Therefore, in future studies it is important to assess the extent of misreporting so measurement error adjustment can be made. One approach could be to use the Goldberg cut-off method revised by Black [(10)] that categorise individuals as plausible, low- or high energy reporters. For this method to be sensitive the collection of information about the total amount of physical activity (occupational-, home-, and leisure-time) is of high importance so that individuals can be assigned to the proper physical activity level groups, so the correct cut-offs can be applied. Future studies investigating causal relationship between dietary patterns and disease should investigate the effect both in the total study population and in a subpopulation where the low energy reporters are excluded. This research would indicate if under-reporting of energy intake really impairs inferences concerning dietary patterns and health outcome. It would also be valuable to use biomarkers of fruits and vegetables such as plasma carotenoids to evaluate the dietary patterns, since over-reporting of healthy foods such as fruit and vegetables is common in subgroups of individuals sharing some specific characteristics.

In cohorts recruiting middle-aged individuals, a causal relationship between diet and disease could be difficult to find because women and men in this age group may already suffer from a chronic disease. As a consequence, they probably have changed their diet in order to reduce either the symptoms of their condition or reduce the likelihood of possible detrimental consequences. Chronic diseases might originate in childhood, and nutrition through the early period of life may have long-lasting consequences. There might also be a cumulative effect of diet through the years and the long-term impact may lead to chronic disease decades later. Future studies of chronic diseases should therefore examine dietary intake at various times in life and with long follow-up.

8 References

1. Wirfalt E, Drake I, Wallstrom P (2013) What do review papers conclude about food and dietary patterns? *Food Nutr Res* **57,** 1-14.
2. Ocke MC (2013) Evaluation of methodologies for assessing the overall diet: dietary quality scores and dietary pattern analysis. *Proc Nutr Soc* **72**, 2, 191-199.
3. Kant AK (2004) Dietary patterns and health outcomes. *J Am Diet Assoc* **104**, 4, 615-635.
4. Hu FB (2002) Dietary pattern analysis: a new direction in nutritional epidemiology. *Curr Opin Lipidol* **13**, 1, 3-9.
5. Newby PK, Tucker KL (2004) Empirically derived eating patterns using factor or cluster analysis: a review. *Nutr Rev* **62**, 5, 177-203.
6. Crozier SR, Inskip HM, Godfrey KM*, et al.* (2008) Dietary patterns in pregnant women: a comparison of food-frequency questionnaires and 4 d prospective diaries. *Br J Nutr* **99**, 4, 869-875.
7. Northstone K, Ness AR, Emmett PM*, et al.* (2008) Adjusting for energy intake in dietary pattern investigations using principal components analysis. *Eur J Clin Nutr* **62**, 7, 931-938.
8. Willett WC (2013) Nutritional Epidemiology. 3rd ed. p. New York: Oxford University Press.
9. Jenab M, Slimani N, Bictash M*, et al.* (2009) Biomarkers in nutritional epidemiology: applications, needs and new horizons. *Hum Genet* **125**, 5-6, 507-525.
10. Black AE (2000) Critical evaluation of energy intake using the Goldberg cut-off for energy intake:basal metabolic rate. A practical guide to its calculation, use and limitations. *Int J Obes Relat Metab Disord* **24**, 9, 1119-1130.
11. Livingstone MB, Black AE (2003) Markers of the validity of reported energy intake. *J Nutr* **133 Suppl 3**, 895s-920s.
12. Tucker KL (2010) Dietary patterns, approaches, and multicultural perspective. *Appl Physiol Nutr Metab* **35**, 2, 211-218.
13. Guenther PM, Casavale KO, Reedy J*, et al.* (2013) Update of the Healthy Eating Index: HEI-2010. *J Acad Nutr Diet* **113**, 4, 569-580.
14. Trichopoulou A, Kouris-Blazos A, Wahlqvist ML*, et al.* (1995) Diet and overall survival in elderly people. *Bmj* **311**, 7018, 1457-1460.
15. Michels KB, Schulze MB (2005) Can dietary patterns help us detect diet-disease associations? *Nutr Res Rev* **18**, 2, 241-248.
16. Field A (2013) Discovering statistics using IBM SPSS Statistics. 4th edition ed. p. London, UK: SAGE Publications Ltd.
17. Fransen HP, May AM, Stricker MD*, et al.* (2014) A posteriori dietary patterns: how many patterns to retain? *J Nutr* **144**, 8, 1274-1282.
18. Devlin UM, McNulty BA, Nugent AP*, et al.* (2012) The use of cluster analysis to derive dietary patterns: methodological considerations, reproducibility, validity and the effect of energy mis-reporting. *Proc Nutr Soc* **71**, 4, 599-609.
19. Crozier SR, Robinson SM, Borland SE*, et al.* (2006) Dietary patterns in the Southampton Women's Survey. *Eur J Clin Nutr* **60**, 12, 1391-1399.
20. Cunha DB, Almeida RM, Pereira RA (2010) A comparison of three statistical methods applied in the identification of eating patterns. *Cad Saude Publica* **26**, 11, 2138-2148.
21. Hoffmann K, Schulze MB, Schienkiewitz A*, et al.* (2004) Application of a new statistical method to derive dietary patterns in nutritional epidemiology. *Am J Epidemiol* **159**, 10, 935-944.
22. Smith AD, Emmett PM, Newby PK*, et al.* (2013) Dietary patterns obtained through principal components analysis: the effect of input variable quantification. *Br J Nutr* **109**, 10, 1881-1891.
23. Balder HF, Virtanen M, Brants HA*, et al.* (2003) Common and country-specific dietary patterns in four European cohort studies. *J Nutr* **133**, 12, 4246-4251.

24. Kristiansen AL, Lande B, Sexton JA*, et al.* (2013) Dietary patterns among Norwegian 2-year-olds in 1999 and in 2007 and associations with child and parent characteristics. *Br J Nutr* **110**, 1, 135-144.
25. Reedy J, Wirfalt E, Flood A*, et al.* (2010) Comparing 3 dietary pattern methods--cluster analysis, factor analysis, and index analysis--With colorectal cancer risk: The NIH-AARP Diet and Health Study. *Am J Epidemiol* **171**, 4, 479-487.
26. Northstone K, Emmett PM, Rogers I (2008) Dietary patterns in pregnancy and associations with nutrient intakes. *Br J Nutr* **99**, 2, 406-415.
27. Charreire H, Kesse-Guyot E, Bertrais S*, et al.* (2011) Associations between dietary patterns, physical activity (leisure-time and occupational) and television viewing in middle-aged French adults. *Br J Nutr* **105**, 6, 902-910.
28. Kiefte-de Jong JC, de Vries JH, Escher JC*, et al.* (2013) Role of dietary patterns, sedentary behaviour and overweight on the longitudinal development of childhood constipation: the Generation R study. *Matern Child Nutr* **9**, 4, 511-523.
29. Bessaoud F, Tretarre B, Daures JP*, et al.* (2012) Identification of dietary patterns using two statistical approaches and their association with breast cancer risk: a case-control study in Southern France. *Ann Epidemiol* **22**, 7, 499-510.
30. Hu FB, Rimm E, Smith-Warner SA*, et al.* (1999) Reproducibility and validity of dietary patterns assessed with a food-frequency questionnaire. *Am J Clin Nutr* **69**, 2, 243-249.
31. Kjollesdal MR, Holmboe-Ottesen G, Mosdol A*, et al.* (2010) The relative importance of socioeconomic indicators in explaining differences in BMI and waist:hip ratio, and the mediating effect of work control, dietary patterns and physical activity. *Br J Nutr* **104**, 8, 1230-1240.
32. Raberg Kjollesdal MK, Holmboe-Ottesen G, Wandel M (2010) Associations between food patterns, socioeconomic position and working situation among adult, working women and men in Oslo. *Eur J Clin Nutr* **64**, 10, 1150-1157.
33. Hearty AP, Gibney MJ (2009) Comparison of cluster and principal component analysis techniques to derive dietary patterns in Irish adults. *Br J Nutr* **101**, 4, 598-608.
34. Moeller SM, Reedy J, Millen AE*, et al.* (2007) Dietary patterns: challenges and opportunities in dietary patterns research an Experimental Biology workshop, April 1, 2006. *J Am Diet Assoc* **107**, 7, 1233-1239.
35. Subar AF, Freedman LS, Tooze JA*, et al.* (2015) Addressing Current Criticism Regarding the Value of Self-Report Dietary Data. *J Nutr* **145,** 12, 2639-45.
36. Rothman KJ, Greenland S, Lash TL (2008) Modern Epidemiology. 3 ed. p. Philadelphia, USA: Lippincott Williams & Wilkins.
37. Porta M (2014) A dictionary of epidemiology. 6 ed. p. New York: Oxford University Press.
38. Bingham SA (2003) Urine nitrogen as a biomarker for the validation of dietary protein intake. *J Nutr* **133 Suppl 3**, 921s-924s.
39. Day N, McKeown N, Wong M*, et al.* (2001) Epidemiological assessment of diet: a comparison of a 7-day diary with a food frequency questionnaire using urinary markers of nitrogen, potassium and sodium. *Int J Epidemiol* **30**, 2, 309-317.
40. Tasevska N, Runswick SA, McTaggart A*, et al.* (2005) Urinary sucrose and fructose as biomarkers for sugar consumption. *Cancer Epidemiol Biomarkers Prev* **14**, 5, 1287-1294.
41. Neuhouser ML, Patterson RE, King IB*, et al.* (2003) Selected nutritional biomarkers predict diet quality. *Public Health Nutr* **6**, 7, 703-709.
42. Weinstein SJ, Vogt TM, Gerrior SA (2004) Healthy Eating Index scores are associated with blood nutrient concentrations in the third National Health And Nutrition Examination Survey. *J Am Diet Assoc* **104**, 4, 576-584.
43. Kant AK, Graubard BI (2005) A comparison of three dietary pattern indexes for predicting biomarkers of diet and disease. *J Am Coll Nutr* **24**, 4, 294-303.

44. Talegawkar SA, Johnson EJ, Carithers TC*, et al.* (2008) Serum carotenoid and tocopherol concentrations vary by dietary pattern among African Americans. *J Am Diet Assoc* **108**, 12, 2013-2020.
45. Goodwin TW (1986) Metabolism, nutrition, and function of carotenoids. *Annu Rev Nutr* **6**, 273-297.
46. Baldrick FR, Woodside JV, Elborn JS*, et al.* (2011) Biomarkers of fruit and vegetable intake in human intervention studies: a systematic review. *Crit Rev Food Sci Nutr* **51**, 9, 795-815.
47. Hammond BR, Jr., Renzi LM (2013) Carotenoids. *Adv Nutr* **4**, 4, 474-476.
48. Maiani G, Caston MJ, Catasta G*, et al.* (2009) Carotenoids: actual knowledge on food sources, intakes, stability and bioavailability and their protective role in humans. *Mol Nutr Food Res* **53 Suppl 2**, S194-218.
49. Alberg A (2002) The influence of cigarette smoking on circulating concentrations of antioxidant micronutrients. *Toxicology* **180**, 2, 121-137.
50. Vioque J, Weinbrenner T, Asensio L*, et al.* (2007) Plasma concentrations of carotenoids and vitamin C are better correlated with dietary intake in normal weight than overweight and obese elderly subjects. *Br J Nutr* **97**, 5, 977-986.
51. Al-Delaimy WK, Ferrari P, Slimani N*, et al.* (2005) Plasma carotenoids as biomarkers of intake of fruits and vegetables: individual-level correlations in the European Prospective Investigation into Cancer and Nutrition (EPIC). *Eur J Clin Nutr* **59**, 12, 1387-1396.
52. Yong LC, Forman MR, Beecher GR*, et al.* (1994) Relationship between dietary intake and plasma concentrations of carotenoids in premenopausal women: application of the USDA-NCI carotenoid food-composition database. *Am J Clin Nutr* **60**, 2, 223-230.
53. Fraser PD, Bramley PM (2004) The biosynthesis and nutritional uses of carotenoids. *Prog Lipid Res* **43**, 3, 228-265.
54. Nagao A (2004) Oxidative conversion of carotenoids to retinoids and other products. *J Nutr* **134**, 1, 237s-240s.
55. Kaaks R, Riboli E, Sinha R (1997) Biochemical markers of dietary intake. *IARC Sci Publ*, 142, 103-126.
56. Al-Delaimy WK, Natarajan L, Sun X*, et al.* (2008) Reliability of plasma carotenoid biomarkers and its relation to study power. *Epidemiology* **19**, 2, 338-344.
57. Goldberg GR, Black AE, Jebb SA*, et al.* (1991) Critical evaluation of energy intake data using fundamental principles of energy physiology: 1. Derivation of cut-off limits to identify under-recording. *Eur J Clin Nutr* **45**, 12, 569-581.
58. Black AE, Goldberg GR, Jebb SA*, et al.* (1991) Critical evaluation of energy intake data using fundamental principles of energy physiology: 2. Evaluating the results of published surveys. *Eur J Clin Nutr* **45**, 12, 583-599.
59. Black AE (2000) The sensitivity and specificity of the Goldberg cut-off for EI:BMR for identifying diet reports of poor validity. *Eur J Clin Nutr* **54**, 5, 395-404.
60. Speakman JR (1998) The history and theory of the doubly labeled water technique. *Am J Clin Nutr* **68**, 4, 932s-938s.
61. Lafay L, Mennen L, Basdevant A*, et al.* (2000) Does energy intake underreporting involve all kinds of food or only specific food items? Results from the Fleurbaix Laventie Ville Sante (FLVS) study. *Int J Obes Relat Metab Disord* **24**, 11, 1500-1506.
62. Johansson G, Wikman A, Ahren AM*, et al.* (2001) Underreporting of energy intake in repeated 24-hour recalls related to gender, age, weight status, day of interview, educational level, reported food intake, smoking habits and area of living. *Public Health Nutr* **4**, 4, 919-927.
63. Yannakoulia M, Panagiotakos DB, Pitsavos C*, et al.* (2007) Low energy reporting related to lifestyle, clinical, and psychosocial factors in a randomly selected population sample of Greek adults: the ATTICA Study. *J Am Coll Nutr* **26**, 4, 327-333.
64. Rasmussen LB, Matthiessen J, Biltoft-Jensen A*, et al.* (2007) Characteristics of misreporters of dietary intake and physical activity. *Public Health Nutr* **10**, 3, 230-237.

65. Poslusna K, Ruprich J, de Vries JH*, et al.* (2009) Misreporting of energy and micronutrient intake estimated by food records and 24 hour recalls, control and adjustment methods in practice. *Br J Nutr* **101 Suppl 2**, S73-85.
66. Lutomski JE, van den Broeck J, Harrington J*, et al.* (2011) Sociodemographic, lifestyle, mental health and dietary factors associated with direction of misreporting of energy intake. *Public Health Nutr* **14**, 3, 532-541.
67. Funtikova AN, Gomez SF, Fito M*, et al.* (2015) Effect of Energy Under-Reporting on Secular Trends of Dietary Patterns in a Mediterranean Population. *PLoS One* **10**, 5, e0127647.
68. Shaneshin M, Jessri M, Rashidkhani B (2014) Validity of energy intake reports in relation to dietary patterns. *J Health Popul Nutr* **32**, 1, 36-45.
69. Bailey RL, Mitchell DC, Miller C*, et al.* (2007) Assessing the effect of underreporting energy intake on dietary patterns and weight status. *J Am Diet Assoc* **107**, 1, 64-71.
70. Winkvist A, Hornell A, Hallmans G*, et al.* (2009) More distinct food intake patterns among women than men in northern Sweden: a population-based survey. *Nutr J* **8**, 12, 1-9.
71. Martikainen P, Brunner E, Marmot M (2003) Socioeconomic differences in dietary patterns among middle-aged men and women. *Soc Sci Med* **56**, 7, 1397-1410.
72. Scagliusi FB, Ferriolli E, Pfrimer K*, et al.* (2008) Under-reporting of energy intake is more prevalent in a healthy dietary pattern cluster. *Br J Nutr* **100**, 5, 1060-1068.
73. Holmback I, Ericson U, Gullberg B*, et al.* (2009) Five meal patterns are differently associated with nutrient intakes, lifestyle factors and energy misreporting in a sub-sample of the Malmo Diet and Cancer cohort. *Food Nutr Res* **53,** 1-16.
74. Pryer JA, Nichols R, Elliott P*, et al.* (2001) Dietary patterns among a national random sample of British adults. *J Epidemiol Community Health* **55**, 1, 29-37.
75. Pryer JA, Vrijheid M, Nichols R*, et al.* (1997) Who are the 'low energy reporters' in the dietary and nutritional survey of British adults? *Int J Epidemiol* **26**, 1, 146-154.
76. Johansson L, Solvoll K, Bjorneboe GE*, et al.* (1998) Under- and overreporting of energy intake related to weight status and lifestyle in a nationwide sample. *Am J Clin Nutr* **68**, 2, 266-274.
77. Pomerleau J, Ostbye T, Bright-See E (1999) Potential underreporting of energy intake in the Ontario Health Survey and its relationship with nutrient and food intakes. *Eur J Epidemiol* **15**, 6, 553-557.
78. Hirvonen T, Mannisto S, Roos E*, et al.* (1997) Increasing prevalence of underreporting does not necessarily distort dietary surveys. *Eur J Clin Nutr* **51**, 5, 297-301.
79. Tonstad S, Gorbitz C, Sivertsen M*, et al.* (1999) Under-reporting of dietary intake by smoking and non-smoking subjects counselled for hypercholesterolaemia. *J Intern Med* **245**, 4, 337-344.
80. Ax E, Warensjo Lemming E, Becker W*, et al.* (2015) Dietary patterns in Swedish adults; results from a national dietary survey. *Br J Nutr*, **115**, 1, 95-104.
81. Kelsey JL, Whittemore AS, Evans AS*, et al.* (1996) Methods in observational epidemiology. 2nd ed. p. New York, US: Oxford University Press.
82. World Health Organization (2014). Global Status Report on Noncommunicable Diseases. Geneva, Switzerland.
83. Engeset D, Alsaker E, Ciampi A*, et al.* (2005) Dietary patterns and lifestyle factors in the Norwegian EPIC cohort: the Norwegian Women and Cancer (NOWAC) study. *Eur J Clin Nutr* **59**, 5, 675-684.
84. Schulze MB, Hoffmann K, Kroke A*, et al.* (2001) Dietary patterns and their association with food and nutrient intake in the European Prospective Investigation into Cancer and Nutrition (EPIC)-Potsdam study. *Br J Nutr* **85**, 3, 363-373.
85. Tseng M, DeVellis RF (2001) Fundamental dietary patterns and their correlates among US whites. *J Am Diet Assoc* **101**, 8, 929-932.
86. Al Suwaidi J (2015) Dietary patterns and their association with acute coronary heart disease: Lessons from the REGARDS Study. *Glob Cardiol Sci Pract* **2015**, 4, 56.

87. Denova-Gutierrez E, Tucker KL, Flores M*, et al.* (2016) Dietary Patterns Are Associated with Predicted Cardiovascular Disease Risk in an Urban Mexican Adult Population. *J Nutr* **146**, 1, 90-97.
88. Knudsen VK, Matthiessen J, Biltoft-Jensen A*, et al.* (2014) Identifying dietary patterns and associated health-related lifestyle factors in the adult Danish population. *Eur J Clin Nutr* **68***,* 736-740.
89. Denoth F, Scalese M, Siciliano V*, et al.* (2015) Clustering eating habits: frequent consumption of different dietary patterns among the Italian general population in the association with obesity, physical activity, sociocultural characteristics and psychological factors. *Eat Weight Disord*.
90. Noori MA, Ghiasvand R, Maghsoudi Z*, et al.* (2016) Evaluation of dietary pattern stability and physical activity in three consecutive generations of women. *Int J Public Health* **61**, 1, 29-38.
91. Sanchez-Villegas A, Delgado-Rodriguez M, Martinez-Gonzalez MA*, et al.* (2003) Gender, age, socio-demographic and lifestyle factors associated with major dietary patterns in the Spanish Project SUN (Seguimiento Universidad de Navarra). *Eur J Clin Nutr* **57**, 2, 285-292.
92. James WP, Nelson M, Ralph A*, et al.* (1997) Socioeconomic determinants of health. The contribution of nutrition to inequalities in health. *Bmj* **314**, 7093, 1545-1549.
93. Slattery ML, Boucher KM, Caan BJ*, et al.* (1998) Eating patterns and risk of colon cancer. *Am J Epidemiol* **148**, 1, 4-16.
94. Engeset D, Dyachenko A, Ciampi A*, et al.* (2009) Dietary patterns and risk of cancer of various sites in the Norwegian European Prospective Investigation into Cancer and Nutrition cohort: the Norwegian Women and Cancer study. *Eur J Cancer Prev* **18**, 1, 69-75.
95. Engeset D, Hofoss D, Nilsson LM*, et al.* (2014) Dietary patterns and whole grain cereals in the Scandinavian countries - differences and similarities. The HELGA project. *Public Health Nutr* **18**, 5, 905-15.
96. Kjollesdal MR, Holmboe-Ottesen G, Wandel M (2011) Frequent use of staff canteens is associated with unhealthy dietary habits and obesity in a Norwegian adult population. *Public Health Nutr* **14**, 1, 133-141.
97. Lockheart MS, Steffen LM, Rebnord HM*, et al.* (2007) Dietary patterns, food groups and myocardial infarction: a case-control study. *Br J Nutr* **98**, 2, 380-387.
98. Jacka FN, Mykletun A, Berk M*, et al.* (2011) The association between habitual diet quality and the common mental disorders in community-dwelling adults: the Hordaland Health study. *Psychosom Med* **73**, 6, 483-490.
99. Konstantinova SV, Tell GS, Vollset SE*, et al.* (2008) Dietary patterns, food groups, and nutrients as predictors of plasma choline and betaine in middle-aged and elderly men and women. *Am J Clin Nutr* **88**, 6, 1663-1669.
100. Brantsaeter AL, Haugen M, Samuelsen SO*, et al.* (2009) A dietary pattern characterized by high intake of vegetables, fruits, and vegetable oils is associated with reduced risk of preeclampsia in nulliparous pregnant Norwegian women. *J Nutr* **139**, 6, 1162-1168.
101. Torjusen H, Lieblein G, Naes T*, et al.* (2012) Food patterns and dietary quality associated with organic food consumption during pregnancy; data from a large cohort of pregnant women in Norway. *BMC Public Health* **12**, 612.
102. Sommer C, Sletner L, Jenum AK*, et al.* (2013) Ethnic differences in maternal dietary patterns are largely explained by socio-economic score and integration score: a population-based study. *Food Nutr Res* **57**.
103. Hofvind SS, Wang H, Thoresen S (2003) The Norwegian Breast Cancer Screening Program: re-attendance related to the women's experiences, intentions and previous screening result. *Cancer Causes Control* **14**, 4, 391-398.
104. Hofvind S, Geller B, Vacek PM*, et al.* (2007) Using the European guidelines to evaluate the Norwegian Breast Cancer Screening Program. *Eur J Epidemiol* **22**, 7, 447-455.
105. Ellingjord-Dale M, dos-Santos-Silva I, Grotmol T*, et al.* (2014) Vitamin D intake, month at mammography and mammographic density in Norwegian women aged 50-69. *PLoS One* **10,** 5.

106. World Health Organization (1998). Obesity: Preventing and managing the global epidemic. Geneva.
107. Kral JG, Kava RA, Catalano PM*, et al.* (2012) Severe obesity: the neglected epidemic. *Obes Facts* **5**, 2, 254-269.
108. Andersen LF, Solvoll K, Johansson LR*, et al.* (1999) Evaluation of a food frequency questionnaire with weighed records, fatty acids, and alpha-tocopherol in adipose tissue and serum. *Am J Epidemiol* **150**, 1, 75-87.
109. Carlsen MH, Lillegaard IT, Karlsen A*, et al.* (2010) Evaluation of energy and dietary intake estimates from a food frequency questionnaire using independent energy expenditure measurement and weighed food records. *Nutr J* **9**, 37.
110. Carlsen MH, Karlsen A, Lillegaard IT*, et al.* (2011) Relative validity of fruit and vegetable intake estimated from an FFQ, using carotenoid and flavonoid biomarkers and the method of triads. *Br J Nutr* **105**, 10, 1530-1538.
111. Qureshi SA, Ellingjord-Dale M, Hofvind S*, et al.* (2012) Physical activity and mammographic density in a cohort of postmenopausal Norwegian women; a cross-sectional study. *Springerplus* **1**, 1, 75.
112. Dallal CM, Sullivan-Halley J, Ross RK*, et al.* (2007) Long-term recreational physical activity and risk of invasive and in situ breast cancer: the California teachers study. *Arch Intern Med* **167**, 4, 408-415.
113. Ainsworth BE, Haskell WL, Whitt MC*, et al.* (2000) Compendium of physical activities: an update of activity codes and MET intensities. *Med Sci Sports Exerc* **32**, 9 Suppl, S498-504.
114. Report of a joint FAO/WHO/UNU Expert Consultation (1985) Energy and protein requirements. *World Health Organ Tech Rep Ser* **724**, 1-206.
115. Henry CJ (2005) Basal metabolic rate studies in humans: measurement and development of new equations. *Public Health Nutr* **8**, 7a, 1133-1152.
116. Tabachnick BG FL (2007) Using Multivariate Statistics. 5th ed. p: Pearson Education Publications.
117. Hutcheson G, Sofroniou N (1999) The multivariate social scientist. p. London: Sage.
118. Bountziouka V, Panagiotakos D (2012) The role of rotation type used to extract dietary patterns through principal component analysis, on their short-term repeatability. *J Data Sci* **10**, 19-36.
119. Varraso R, Garcia-Aymerich J, Monier F*, et al.* (2012) Assessment of dietary patterns in nutritional epidemiology: principal component analysis compared with confirmatory factor analysis. *Am J Clin Nutr* **96**, 5, 1079-1092.
120. Castro MA, Baltar VT, Selem SS*, et al.* (2015) Empirically derived dietary patterns: interpretability and construct validity according to different factor rotation methods. *Cad Saude Publica* **31**, 2, 298-310.
121. Veierod MB, Lydersen S, P. L (2012) Medical Statistics in Clinical and Epidemiological Research. 1 ed. p. Oslo, Norway: Gyldendal Norsk Forlag.
122. Rimestad AH, Løken EB, Nordbotten A (2000) The Norwegian food composition table and calculation system used at the Intitute for Nutrition Research. *Norwegian Journal of Epidemiology* **10**, 107-116.
123. Schoeller DA (1995) Limitations in the assessment of dietary energy intake by self-report. *Metabolism* **44**, 2 Suppl 2, 18-22.
124. Schoeller DA (1990) How accurate is self-reported dietary energy intake? *Nutr Rev* **48**, 10, 373-379.
125. Subar AF, Kipnis V, Troiano RP*, et al.* (2003) Using intake biomarkers to evaluate the extent of dietary misreporting in a large sample of adults: the OPEN study. *Am J Epidemiol* **158**, 1, 1-13.
126. Jonnalagadda SS, Mitchell DC, Smiciklas-Wright H*, et al.* (2000) Accuracy of energy intake data estimated by a multiple-pass, 24-hour dietary recall technique. *J Am Diet Assoc* **100**, 3, 303-308; quiz 309-311.

127. Connor Gorber S, Tremblay MS (2010) The bias in self-reported obesity from 1976 to 2005: a Canada-US comparison. *Obesity (Silver Spring)* **18**, 2, 354-361.
128. Hebert JR, Hurley TG, Peterson KE*, et al.* (2008) Social desirability trait influences on self-reported dietary measures among diverse participants in a multicenter multiple risk factor trial. *J Nutr* **138**, 1, 226s-234s.
129. Scagliusi FB, Polacow VO, Artioli GG*, et al.* (2003) Selective underreporting of energy intake in women: magnitude, determinants, and effect of training. *J Am Diet Assoc* **103**, 10, 1306-1313.
130. Gibson RS (2005) Principles of Nutritional Assessment. 2 ed. p. Australia: Oxford University Press Australia.
131. Mendez MA, Popkin BM, Buckland G*, et al.* (2011) Alternative methods of accounting for underreporting and overreporting when measuring dietary intake-obesity relations. *Am J Epidemiol* **173**, 4, 448-458.
132. Mendez MA, Wynter S, Wilks R*, et al.* (2004) Under- and overreporting of energy is related to obesity, lifestyle factors and food group intakes in Jamaican adults. *Public Health Nutr* **7**, 1, 9-19.
133. Lynch BM, Neilson HK, Friedenreich CM (2011) Physical activity and breast cancer prevention. *Recent Results Cancer Res* **186**, 13-42.
134. FAO/WHO/UNU Consultation (2001). Human energy requirements.
135. McCann SE, Marshall JR, Brasure JR*, et al.* (2001) Analysis of patterns of food intake in nutritional epidemiology: food classification in principal components analysis and the subsequent impact on estimates for endometrial cancer. *Public Health Nutr* **4**, 5, 989-997.
136. Schwerin HS, Stanton JL, Riley AM, Jr.*, et al.* (1981) Food eating patterns and health: a reexamination of the Ten-State and HANES I surveys. *Am J Clin Nutr* **34**, 4, 568-580.
137. Mishra GD, McNaughton SA, Ball K*, et al.* (2010) Major dietary patterns of young and middle aged women: results from a prospective Australian cohort study. *Eur J Clin Nutr* **64**, 10, 1125-1133.
138. Le Port A, Gueguen A, Kesse-Guyot E*, et al.* (2012) Association between dietary patterns and depressive symptoms over time: a 10-year follow-up study of the GAZEL cohort. *PLoS One* **7**, 12, e51593.
139. Krebs-Smith SM, Graubard BI, Kahle LL*, et al.* (2000) Low energy reporters vs others: a comparison of reported food intakes. *Eur J Clin Nutr* **54**, 4, 281-287.
140. Lau C, Glumer C, Toft U*, et al.* (2008) Identification and reproducibility of dietary patterns in a Danish cohort: the Inter99 study. *Br J Nutr* **99**, 5, 1089-1098.
141. Terry P, Suzuki R, Hu FB*, et al.* (2001) A prospective study of major dietary patterns and the risk of breast cancer. *Cancer Epidemiol Biomarkers Prev* **10**, 12, 1281-1285.
142. Ovaskainen ML, Tapanainen H, Laatikainen T*, et al.* (2015) Perceived health-related self-efficacy associated with BMI in adults in a population-based survey. *Scand J Public Health*.
143. Osler M, Heitmann BL, Gerdes LU*, et al.* (2001) Dietary patterns and mortality in Danish men and women: a prospective observational study. *Br J Nutr* **85**, 2, 219-225.
144. Jacobs DR, Jr., Meyer HE, Solvoll K (2001) Reduced mortality among whole grain bread eaters in men and women in the Norwegian County Study. *Eur J Clin Nutr* **55**, 2, 137-143.
145. Buck K, Vrieling A, Flesch-Janys D*, et al.* (2011) Dietary patterns and the risk of postmenopausal breast cancer in a German case-control study. *Cancer Causes Control* **22**, 2, 273-282.
146. Hardcastle AC, Aucott L, Fraser WD*, et al.* (2011) Dietary patterns, bone resorption and bone mineral density in early post-menopausal Scottish women. *Eur J Clin Nutr* **65**, 3, 378-385.
147. Wood AD, Strachan AA, Thies F*, et al.* (2014) Patterns of dietary intake and serum carotenoid and tocopherol status are associated with biomarkers of chronic low-grade systemic inflammation and cardiovascular risk. *Br J Nutr* **112**, 8, 1341-1352.
148. Waijers PM, Ocke MC, van Rossum CT*, et al.* (2006) Dietary patterns and survival in older Dutch women. *Am J Clin Nutr* **83**, 5, 1170-1176.

149. Touvier M, Niravong M, Volatier JL*, et al.* (2009) Dietary patterns associated with vitamin/mineral supplement use and smoking among women of the E3N-EPIC cohort. *Eur J Clin Nutr* **63**, 1, 39-47.
150. Bedard A, Garcia-Aymerich J, Sanchez M*, et al.* (2015) Confirmatory Factor Analysis Compared with Principal Component Analysis to Derive Dietary Patterns: A Longitudinal Study in Adult Women. *J Nutr* **145**, 7, 1559-1568.
151. Chan JM, Gong Z, Holly EA*, et al.* (2013) Dietary patterns and risk of pancreatic cancer in a large population-based case-control study in the San Francisco Bay Area. *Nutr Cancer* **65**, 1, 157-164.
152. Dalvi TB, Canchola AJ, Horn-Ross PL (2007) Dietary patterns, Mediterranean diet, and endometrial cancer risk. *Cancer Causes Control* **18**, 9, 957-966.
153. Chang ET, Lee VS, Canchola AJ*, et al.* (2008) Dietary patterns and risk of ovarian cancer in the California Teachers Study cohort. *Nutr Cancer* **60**, 3, 285-291.
154. Link LB, Canchola AJ, Bernstein L*, et al.* (2013) Dietary patterns and breast cancer risk in the California Teachers Study cohort. *Am J Clin Nutr* **98**, 6, 1524-1532.
155. Flood A, Rastogi T, Wirfalt E*, et al.* (2008) Dietary patterns as identified by factor analysis and colorectal cancer among middle-aged Americans. *Am J Clin Nutr* **88**, 1, 176-184.
156. Kolahdooz F, Ibiebele TI, van der Pols JC*, et al.* (2009) Dietary patterns and ovarian cancer risk. *Am J Clin Nutr* **89**, 1, 297-304.
157. Bentzen J, Smith V (2004) Are the fluctuations in the consumption of alcohol pro-or counter-cyclical? Empirical evidence from the Scandinavian countries using long-run time series data. *Oenometrie*.
158. Kant AK (2010) Dietary patterns: biomarkers and chronic disease risk. *Appl Physiol Nutr Metab* **35**, 2, 199-206.
159. Hann CS, Rock CL, King I*, et al.* (2001) Validation of the Healthy Eating Index with use of plasma biomarkers in a clinical sample of women. *Am J Clin Nutr* **74**, 4, 479-486.
160. Gerber MJ, Scali JD, Michaud A*, et al.* (2000) Profiles of a healthful diet and its relationship to biomarkers in a population sample from Mediterranean southern France. *J Am Diet Assoc* **100**, 10, 1164-1171.
161. Newby PK, Hu FB, Rimm EB*, et al.* (2003) Reproducibility and validity of the Diet Quality Index Revised as assessed by use of a food-frequency questionnaire. *Am J Clin Nutr* **78**, 5, 941-949.
162. Pryer JA, Cook A, Shetty P (2001) Identification of groups who report similar patterns of diet among a representative national sample of British adults aged 65 years of age or more. *Public Health Nutr* **4**, 3, 787-795.
163. Murakami K, Livingstone MB (2015) Prevalence and characteristics of misreporting of energy intake in US adults: NHANES 2003-2012. *Br J Nutr* **115**, 2, 294-304.
164. Goris AH, Westerterp-Plantenga MS, Westerterp KR (2000) Undereating and underrecording of habitual food intake in obese men: selective underreporting of fat intake. *Am J Clin Nutr* **71**, 1, 130-134.
165. Berta Vanrullen I, Volatier JL, Bertaut A*, et al.* (2014) Characteristics of energy intake under-reporting in French adults. *Br J Nutr* **111**, 7, 1292-1302.
166. Ferrari P, Slimani N, Ciampi A*, et al.* (2002) Evaluation of under- and overreporting of energy intake in the 24-hour diet recalls in the European Prospective Investigation into Cancer and Nutrition (EPIC). *Public Health Nutr* **5**, 6B, 1329-1345.
167. Park HA, Lee JS, Kuller LH (2007) Underreporting of dietary intake by body mass index in premenopausal women participating in the Healthy Women Study. *Nutr Res Pract* **1**, 3, 231-236.
168. Wirfalt E, Mattisson I, Gullberg B*, et al.* (2000) Food patterns defined by cluster analysis and their utility as dietary exposure variables: a report from the Malmo Diet and Cancer Study. *Public Health Nutr* **3**, 2, 159-173.
169. Brunner EJ, Mosdol A, Witte DR*, et al.* (2008) Dietary patterns and 15-y risks of major coronary events, diabetes, and mortality. *Am J Clin Nutr* **87**, 5, 1414-1421.

170. McNaughton SA, Mishra GD, Brunner EJ (2008) Dietary patterns, insulin resistance, and incidence of type 2 diabetes in the Whitehall II Study. *Diabetes Care* **31**, 7, 1343-1348.
171. McNaughton SA, Mishra GD, Brunner EJ (2009) Food patterns associated with blood lipids are predictive of coronary heart disease: the Whitehall II study. *Br J Nutr* **102**, 4, 619-624.
172. Mattisson I, Wirfalt E, Aronsson CA*, et al.* (2005) Misreporting of energy: prevalence, characteristics of misreporters and influence on observed risk estimates in the Malmo Diet and Cancer cohort. *Br J Nutr* **94**, 5, 832-842.
173. Prentice RL, Shaw PA, Bingham SA*, et al.* (2009) Biomarker-calibrated energy and protein consumption and increased cancer risk among postmenopausal women. *Am J Epidemiol* **169**, 8, 977-989.
174. Neuhouser ML, Tinker L, Shaw PA*, et al.* (2008) Use of recovery biomarkers to calibrate nutrient consumption self-reports in the Women's Health Initiative. *Am J Epidemiol* **167**, 10, 1247-1259.
175. Northstone K, Smith AD, Cribb VL*, et al.* (2014) Dietary patterns in UK adolescents obtained from a dual-source FFQ and their associations with socio-economic position, nutrient intake and modes of eating. *Public Health Nutr* **17**, 7, 1476-1485.
176. Nordic Council of Ministers (2014) Nordic Nutrition Recommendations 2012 : Integrating nutrition and physical activity. 5 ed. p. Copenhagen.
177. Terry P, Hu FB, Hansen H*, et al.* (2001) Prospective study of major dietary patterns and colorectal cancer risk in women. *Am J Epidemiol* **154**, 12, 1143-1149.
178. Zazpe I, Sanchez-Tainta A, Toledo E*, et al.* (2014) Dietary patterns and total mortality in a Mediterranean cohort: the SUN project. *J Acad Nutr Diet* **114**, 1, 37-47.
179. Chocano-Bedoya PO, O'Reilly EJ, Lucas M*, et al.* (2013) Prospective study on long-term dietary patterns and incident depression in middle-aged and older women. *Am J Clin Nutr* **98**, 3, 813-820.
180. Fung TT, Feskanich D (2015) Dietary patterns and risk of hip fractures in postmenopausal women and men over 50 years. *Osteoporos Int* **26**, 6, 1825-1830.
181. Martinez-Gonzalez MA, Zazpe I, Razquin C*, et al.* (2015) Empirically-derived food patterns and the risk of total mortality and cardiovascular events in the PREDIMED study. *Clin Nutr* **34**, 5, 859-867.
182. Bertin M, Touvier M, Dubuisson C*, et al.* (2015) Dietary patterns of French adults: associations with demographic, socio-economic and behavioural factors. *J Hum Nutr Diet*.
183. Kesse-Guyot E, Bertrais S, Peneau S*, et al.* (2009) Dietary patterns and their sociodemographic and behavioural correlates in French middle-aged adults from the SU.VI.MAX cohort. *Eur J Clin Nutr* **63**, 4, 521-528.
184. Harrington JM, Dahly DL, Fitzgerald AP*, et al.* (2014) Capturing changes in dietary patterns among older adults: a latent class analysis of an ageing Irish cohort. *Public Health Nutr* **17**, 12, 2674-2686.
185. Mishra GD, Prynne CJ, Paul AA*, et al.* (2004) The impact of inter-generational social and regional circumstances on dietary intake patterns of British adults: results from the 1946 British Birth Cohort. *Public Health Nutr* **7**, 6, 737-744.
186. Hart CL, Davey Smith G, Upton MN*, et al.* (2009) Alcohol consumption behaviours and social mobility in men and women of the Midspan Family study. *Alcohol Alcohol* **44**, 3, 332-336.
187. Mashta O (2009) Middle classes in Britain drink most alcohol, but smoking is at lowest recorded level. *Bmj* **338**, b312.
188. U.S Department of Health and Human Services and the U.S. Department of Agriculture Dietary Guidelines for Americans 2015-2020: http://health.gov/dietaryguidelines/2015/guidelines/.
189. van Dam RM (2005) New approaches to the study of dietary patterns. *Br J Nutr* **93**, 5, 573-574.

Papers I-III

Appendix I

UNIVERSITETET I OSLO
DET MEDISINSKE FAKULTET

Undersøkelse om kost og brystkreft i Norge

Kjære deltager. Januar 2008

Universitetet i Oslo (UiO) gjennomfører denne undersøkelsen om kosthold og brystkreft. Du har tidligere svart "ja" til å delta i en kostholdsundersøkelse i forbindelse med din mammografiundersøkelse. Dette er viktig for å forstå hvordan brystkreft og andre kroniske sykdommer kan forebygges hos kvinner. Målet vårt er å inkludere 40 000 kvinner i denne studien. Statistisk sentralbyrå (SSB) bistår oss med den praktiske gjennomføring av datainnsamlingen.

Du har mottatt et spørreskjema om dine spisevaner og en frankert returkonvolutt. På side 3 i dette skjemaet finner du en samtykkeerklæring. Vi ber om at du leser igjennom informasjonen på baksiden av dette brevet før du skriver under på samtykkeerklæringen. Deretter kan du fylle ut spørreskjemaet. Dette tar ca en time å fylle ut. Det er meget viktig at samtykkeerklæringen er underskrevet før du returnerer det ferdig utfylte spørreskjemaet. Vi setter stor pris på at du returnerer skjemaet (inkludert underskrevet samtykkeerklæring) innen fristen som står øverst på skjemaet. Det er frivillig å delta, og du kan trekke deg fra denne undersøkelsen så lenge du sier i fra før dataene er anonymiserte (se baksiden).

Informasjonen vil bare bli brukt til dette forskningsformålet, og resultatene vil behandles konfidensielt. Dersom du har spørsmål til undersøkelsen, kan du ringe 22 85 13 80 og legge igjen en beskjed slik at vi ringer deg tilbake. Du kan også sende en e-post til marit.hilsen@medisin.uio.no.

Har du spørsmål om den praktiske gjennomføringen av undersøkelsen, kan du kontakte SSB på telefon 62 88 50 00, eller sende en e-post til dagfinn.sve@ssb.no.
Oppgi hvilken undersøkelse det dreier seg om.

Tusen takk for hjelpen!

Med vennlig hilsen

Giske Ursin
Lege og Professor
Prosjektleder

Marit Hilsen
Prosjektmedarbeider

Christian A. Drevon
Lege og Professor

KOST OG BRYSTKREFT I NORGE

INFORMASJON

Ansvarlig: Professor Giske Ursin, Avdeling for ernæringsvitenskap, Universitetet i Oslo (UiO)

Vi ber om tillatelse til å stille deg noen spørsmål om kosthold som ledd i vår undersøkelse av kosthold og kreft hos kvinner.

HENSIKTEN MED STUDIEN

Som ledd i vårt arbeid med å forebygge brystkreft og andre kroniske sykdommer hos kvinner, ønsker vi å undersøke hvordan kosthold og hormoner virker inn på normalt brystvev slik vi ser det på mammografi.

METODE

Vi ber om at du svarer på det vedlagte spørreskjemaet, og at du returnerer det i den frankerte svarkonvolutten. Vi ber deg også om å gi oss et telefonnummer nederst på samtykkeerklæringen i tilfelle vi i løpet av de nærmeste ukene har noen spørsmål om utfyllingen av spørreskjemaet ditt. Vi vil undersøke følgende hos alle deltagerne samlet: tetthet i brystvevet, informasjon om eventuell hormonbruk ved tidligere screeninger, opplysninger fra kostholdsspørreskjemaet og opplysninger fra skjemaet du fikk sammen med din invitasjon til screeningen. Vi vil senere søke tillatelse om å koble dataene fra alle deltagerne mot kreftregisteret/mammografiscreeningen for å forstå hvordan kreft kan forebygges.

PERSONVERN OG TILGANG TIL OPPLYSNINGER

UiO er databehandlingsansvarlig for undersøkelsen. Statistisk sentralbyrå (SSB) er databehandler og bistår oss med datainnsamlingen. Det er derfor SSB som skal ha de utfylte skjemaene (inkludert underskrevet samtykkeerklæring) og vil stå for skanning av disse før de oversendes til UiO. Ved UiO vil skjemaene bli avidentifisert. Resultatene vil bli behandlet fortrolig og alle involverte har taushetsplikt. Alle opplysninger vil kun bli brukt til dette forskningsformålet. Prosjektet forventes avsluttet ved utgangen av 2012, men opplysningene kan bli lagret etter prosjektslutt for fremtidige data analyser og koblinger med mammografiscreeningen/kreftregisteret i inntil 15 år etter prosjektslutt, og kan bli lagret i anonym form etter dette. Resultatene fra studiene vil bli publisert i anonym form i internasjonale medisinske tidsskrifter. Prosjektet har konsesjon fra Datatilsynet og tilrådning fra Regional komité for medisinsk og helsefaglig forskningsetikk.

DELTAGELSE I STUDIEN

Det er frivillig å delta. Hvorvidt du deltar eller ikke spiller ingen rolle for din mammografi-undersøkelse eller eventuell annen behandling du måtte trenge. Du kan trekke deg uten at du trenger å gi en begrunnelse. Det får ingen konsekvenser for deg, og vi vil i så fall slette dine opplysninger fra forskningsprosjektet. Dette forutsetter at du trekker deg før vi har gjort dataene anonyme. Du vil ikke ha personlig nytte av å delta i denne studien. Du vil ikke få noen resultater fra denne undersøkelsen, ettersom det er usikkert i hvilken grad tettheten i brystvevet har noen klinisk betydning.

KOST OG BRYSTKREFT I NORGE

Hva spiser du?

Samtykkeerklæring

Jeg har lest informasjonsbrevet (på forrige side) og samtykker i å delta i denne undersøkelsen, og i at opplysningene kan lagres og brukes i forskning etter prosjektslutt som angitt i informasjonsbrevet.

Navn (BRUK STORE BOKSTAVER):

Adresse:

Dato

Underskrift

Dersom vi har spørsmål om ditt kostholdsskjema, ber vi om ditt telefonnummer:

Det er i orden at vi ringer mellom kl. og kl.

Er du villig til også å avgi en spyttprøve og en liten fingerprikk blodprøve? JA: ☐ NEI: ☐

Du kan ta prøvene selv med utstyr og instruksjoner vi sender. Prøvene brukes til å måle ulike næringsstoffer som fettsyrer, antioksidanter og proteiner, samt til å se på genetiske varianter som har betydning for omsetningen av disse stoffene. Laboratorieanalysene gjøres på avidentifiserte prøver.

Om skjemautfyllingen

Vi spør om dine spisevaner slik de vanligvis er. Vi er klar over at kostholdet varierer fra dag til dag. Prøv derfor å angi et gjennomsnitt av dine spisevaner. Ha det siste året i tankene når du fyller ut skjemaet. Der du er usikker, anslå svaret.

Riktig markering er: ☒ Bruk gjerne blå eller svart kulepenn eller tusj.

Husk å krysse av "aldri" for de matvarene du ikke spiser.

Forkortelser brukt i skjemaet:
< 1 = sjeldnere enn 1 gang
cl = centiliter
dl = desiliter
g = gram
hg = hekto
stk = stykke
ss = spiseskje
ts = teskje
bs = barneskje
o.l. = og lignende

Ved spørsmål kan du ringe Avdeling for ernæringsvitenskap, Universitetet i Oslo, tlf. 22 85 13 80

Eksempel på utfylling av spørsmål 1.
Kari Nordmann spiser daglig 5 skiver brød og ett mørkt knekkebrød. Hun spiser vanligvis lys kneipp men i helgene blir det en del loff. Hun fyller ut første spørsmål 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 chiabatta=2 skiver)

		Antall skiver pr. dag												
	Sjelden /aldri	1/2	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 42 brukes i spørsmål 4.

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)

	Sjelden /aldri	Antall skiver pr. dag											
		1/2	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:
(sum skiver pr. dag): ____ x 7 = _____. Tallet brukes i spørsmål 4.

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)

	Sjelden /aldri	Antall skiver pr. uke								
		1	2-3	4-5	6-7	8-12	13-18	19-24	25-30	31+
Smør (eks. meierismør)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Bremykt	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Brelett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Melange	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Myk margarin (Soft Flora, Soft Ekstra o.l.)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Soft Oliven	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Vita	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Soft Light, Vita Lett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Annen margarin	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Bruker ikke noe	☐									

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

	Antall skiver					
	1	2	3	4	5	6 +
En porsjonspakke smør/margarin på 12 g rekker til antall skiver:	☐	☐	☐	☐	☐	☐

4. Hvilke typer pålegg spiser du? Bruk sum skiver per uke fra spørsmål 1.

	Sjelden /aldri	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)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Smøreost	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lett/mager smøreost	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Leverpostei	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Mager leverpostei	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Servelat	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kokt skinke, lettservelat, kalkunpålegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Salami, fårepølse	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kaviar	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Svolværpostei, Lofotpostei	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Makrell i tomat	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Røkt laks/ørret	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sardiner, sursild, ansjos	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Reker, krabbe	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Syltetøy, marmelade	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lett syltetøy, frysetøy	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Peanøttsmør	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sjokolade-, nøttepålegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Honning	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Annet søtt pålegg (eks. Sunda, sirup)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Majonessalat (eks. italiensk salat)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Majonessalat lett (eks. lett italiensk salat)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Frukt som pålegg (eks. banan)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Grønnsaker som pålegg (eks. agurk, tomat)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

5. Egg

		Antall egg pr. uke						
	Sjelden /aldri	<1	1	2	3-4	5-6	7	8+
Kokt, stekt, eggerøre, omelett	☐	☐	☐	☐	☐	☐	☐	☐

6. Frokostgryn Svar enten per måned eller per uke.

	Sjelden /aldri	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 (Solfrokost o.l.)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Mysli, usøtet (Go'Dag o.l.)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Cornflakes	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Honnikorn/Frosties/Chocofrokost	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	☐	☐	☐	☐
All Bran, Weetabix o.l.	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Puffet ris/havrenøtter/hvetenøtter	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	☐	☐	☐	☐
	Sjelden /aldri	**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	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐

7. Melk (Husk å ta med melk du bruker på frokostgryn, grøt og dessert) (1 glass = 2 dl)

	Sjelden /aldri	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	☐	☐	☐	☐	☐	☐	☐	☐	☐

8. Yoghurt (Husk å ta med yoghurt du bruker på frokostgryn) Svar enten per måned eller per uke.

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

9. Kald drikke

Svar enten per uke eller per dag. Merk at porsjonsenhetene er forskjellige.
1/5 liter tilsvarer ett glass, mens 1/3 liter tilsvarer 0,33 l glassflaske/boks.

	Sjelden /aldri	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+ ☐
Mineralvann (Farris o.l.)	☐	☐	☐	☐	☐		☐	☐	☐	☐	(liter)	1/5 ☐	1/3 ☐	½ ☐	1+ ☐
Juice med blåbær og aronia	☐	☐	☐	☐	☐		☐	☐	☐	☐	(glass)	1 ☐	2 ☐	3 ☐	4+ ☐
Juice med nype og appelsin	☐	☐	☐	☐	☐		☐	☐	☐	☐	(glass)	1 ☐	2 ☐	3 ☐	4+ ☐
Juice med tranebær og bringebær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(glass)	1 ☐	2 ☐	3 ☐	4+ ☐
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 (Vørterøl, Munkholm o.l.)	☐	☐	☐	☐	☐		☐	☐	☐	☐	(liter)	1/5 ☐	1/3 ☐	½ ☐	1+ ☐

10. Alkoholholdig drikke

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

	Sjelden /aldri	Gang pr. måned 1	2	3	eller	Gang pr. uke 1	2-3	4-5	6-7	Mengde pr. gang						
Øl (pils)	☐	☐	☐	☐		☐	☐	☐	☐	(liter)	1/3 ☐	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Lettøl	☐	☐	☐	☐		☐	☐	☐	☐	(liter)	1/3 ☐	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Rusbrus, Cider	☐	☐	☐	☐		☐	☐	☐	☐	(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+ ☐
Brennevin, likør	☐	☐	☐	☐		☐	☐	☐	☐	(1 dram = 4cl)	1 ☐	2 ☐	3 ☐	4 ☐	5 ☐	6+ ☐
Blandede drinker, cocktail	☐	☐	☐	☐		☐	☐	☐	☐	(drink)	1 ☐	2 ☐	3 ☐	4 ☐	5 ☐	6+ ☐

11. Kaffe og te

(1 kopp kaffe =1,2 dl, 1 kopp te =2,5 dl, 1 kopp caffe latte =3dl, 1 kopp cappucino =1,5 dl, 1 kopp espresso =0,3 dl)

	Drikker ikke daglig	Antall kopper pr. dag ½	1	2	3-4	5-6	7-8	9-10	11+
Kaffe, kokt (eks. presskanne)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kaffe, traktet, filter	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kaffe, pulver (instant)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Espresso	☐	☐	☐	☐	☐	☐	☐	☐	☐
Caffe latte	☐	☐	☐	☐	☐	☐	☐	☐	☐
Cappucino	☐	☐	☐	☐	☐	☐	☐	☐	☐
Te, sort (eks. Earl Grey, Solbær)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Grønn te	☐	☐	☐	☐	☐	☐	☐	☐	☐
Urtete (eks. nype, kamille, Rooibush)	☐	☐	☐	☐	☐	☐	☐	☐	☐

	Bruker ikke	Antall teskjeer eller sukkerbiter pr. kopp ½	1	2	3	4+
Sukker til kaffe	☐	☐	☐	☐	☐	☐
Sukker til te	☐	☐	☐	☐	☐	☐
Sukketter til te/kaffe	☐	☐	☐	☐	☐	☐

12. Middagsretter

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

	Sjelden /aldri	Gang pr. måned 1	2	3	4	5-6	7-8	9+	Mengde pr. gang	½	1	1½	2	3+
Kjøttpølse av storfe- og svinekjøtt	☐	☐	☐	☐	☐	☐	☐	☐	(pølse)	☐	☐	☐	☐	☐
Kjøttpølse av kylling eller kalkun	☐	☐	☐	☐	☐	☐	☐	☐	(pølse)	☐	☐	☐	☐	☐
Grillpølse/wienerpølse av storfe- og svinekjøtt	☐	☐	☐	☐	☐	☐	☐	☐	(pølse)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Grillpølse/wienerpølse av kylling eller kalkun	☐	☐	☐	☐	☐	☐	☐	☐	(pølse)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Hamburger, karbonader	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Hamburger-, pølsebrød, lomper	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Kjøttkaker, medisterkaker, kjøttpudding	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Kjøttsaus	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Taco (med kjøtt og salat)	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Kebab	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Lasagne, moussaka	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Pastaretter (Pasta di Napoli o.l.)	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐

Middagsretter fortsetter neste side ...

Middagsretter forts...

	Sjelden /aldri	Gang pr. måned 1	2	3	4	5-6	7-8	9+	Mengde pr. gang					
Pizza (1 stk =500-600 g)	☐	☐	☐	☐	☐	☐	☐	☐	(pizza)	1/8 ☐	1/4 ☐	½ ☐	3/4 ☐	1+ ☐
Biff (alle typer kjøtt)	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	½ ☐	1 ☐	1½ ☐	2 ☐	2½+ ☐
Koteletter (lam, okse, svin o.l.)	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	½ ☐	1 ☐	1½ ☐	2 ☐	2½+ ☐
Stek (lam, okse, svin o.l.)	☐	☐	☐	☐	☐	☐	☐	☐	(skive)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Stek (elg, hjort, reinsdyr o.l.)	☐	☐	☐	☐	☐	☐	☐	☐	(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+ ☐
Bacon, stekt flesk	☐	☐	☐	☐	☐	☐	☐	☐	(skive)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Grillet kylling	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/4 ☐	1/3 ☐	1/2 ☐	3/4 ☐	1+ ☐
Kyllingfilet	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	½ ☐	1 ☐	1½ ☐	2 ☐	3+ ☐
Wok med kjøtt og grønnsaker	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Fiskekaker, fiskepudding	☐	☐	☐	☐	☐	☐	☐	☐	(kake)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Fiskeboller	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-2 ☐	3-4 ☐	5-6 ☐	7-9 ☐	10+ ☐
Fiskepinner	☐	☐	☐	☐	☐	☐	☐	☐	(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+ ☐
Sild (fersk, speket, røkt)	☐	☐	☐	☐	☐	☐	☐	☐	(filet)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Makrell (fersk)	☐	☐	☐	☐	☐	☐	☐	☐	(filet)	½ ☐	1 ☐	1½ ☐	2 ☐	3+ ☐
Makrell (røkt)	☐	☐	☐	☐	☐	☐	☐	☐	(filet)	½ ☐	1 ☐	1½ ☐	2 ☐	3+ ☐
Laks, ørret (kokt, stekt)	☐	☐	☐	☐	☐	☐	☐	☐	(skive)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Fiskegryte, fiskegrateng, fiskesuppe	☐	☐	☐	☐	☐	☐	☐	☐	(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+ ☐
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 o.l.)	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Vegetarrett, vegetarpizza, grønnsaksgrateng, -pai	☐	☐	☐	☐	☐	☐	☐	☐	(bit/dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Hurtignudler (eks. Mr Lee)	☐	☐	☐	☐	☐	☐	☐	☐	(pakke)	½ ☐	1 ☐	1½ ☐	2 ☐	3+ ☐

13. Saus og dressing

	Sjelden /aldri	Gang pr. måned 1	2	3	4	5-6	7-8	9+	Mengde pr. gang					
Brun/hvit saus	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	½ ☐	1 ☐	1½ ☐	2 ☐	3+ ☐
Smeltet margarin/smør	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Bearnéssaus, hollandés	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	½ ☐	1 ☐	1½ ☐	2 ☐	3+ ☐
Majones/remulade vanlig	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Majones/remulade lett	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Lettrømme (20 % fett)	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Seterrømme (35 % fett)	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Dressing (Thousand Island o.l.)	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Oljedressing, vinagrette	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Pesto	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Ketchup	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Sennep	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Tomatsaus	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Tacosaus	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	½ ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Soyasaus	☐	☐	☐	☐	☐	☐	☐	☐	(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
☐ Soft Oliven	☐ Rapsolje
☐ Flytende margarin på flaske (Vita, Melange, Bremyk o.l.)	☐ Andre oljer
☐ Annen margarin	

15. 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.

	Sjelden /aldri	Gang pr. måned 1	2	3	eller 1	Gang pr. uke 2-3	4-5	6-7	8+	Mengde pr. gang					
Poteter, kokte	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Potetmos	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Potetsalat m/majones	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Gratinerte poteter	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Stekte poteter	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Pommes frites, gatekjøkken	☐	☐	☐	☐	☐	☐	☐	☐	☐	(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+ ☐
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+ ☐
Spinat	☐	☐	☐	☐	☐	☐	☐	☐	☐	(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+ ☐

Hvor mange ganger om dagen spiser du vanligvis grønnsaker (utenom potet)?

Sjelden /aldri	1	2	3	4	5+
☐	☐	☐	☐	☐	☐

16. Krydder

Oppgi den mengden krydder som er tilsatt de porsjonene mat du spiser og ta **ikke** med det som kan være i ferdigprodukter. Svar enten pr. måned eller pr. uke. Husk å krysse av for mengde.

	Gang pr. måned				eller	Gang pr. uke				Mengde pr. gang					
	Aldri	< 1	1	2	3	1	2-4	5-7	8+	Enhet	1/4	1/2	1	2	3+
Basilikum, tørket	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	☐	☐	☐	☐	☐
Chili, pulver	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	☐	☐	☐	☐	☐
Kanel	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	☐	☐	☐	☐	☐
Tacokrydder	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	☐	☐	☐	☐	☐
Grillkrydder	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	☐	☐	☐	☐	☐
Kardemomme	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	☐	☐	☐	☐	☐
Karri	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	☐	☐	☐	☐	☐
Timian, tørket	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	☐	☐	☐	☐	☐
Sort pepper	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	☐	☐	☐	☐	☐
Oregano, tørket	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	☐	☐	☐	☐	☐
Paprika, pulver	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	☐	☐	☐	☐	☐
Rosmarin, tørket	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	☐	☐	☐	☐	☐
Hvitløk	☐	☐	☐	☐	☐	☐	☐	☐	☐	(fedd)	☐	☐	☐	☐	☐

	Aldri	< 1	1	2	3	1	2-4	5-7	8+		1/2	1-2	3-4	5-6	7+
Frisk basilikum	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐	☐
Frisk chili	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ts)	☐	☐	☐	☐	☐
Frisk dill	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐	☐
Frisk ingefær	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐	☐
Frisk oregano	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐	☐
Frisk peppermynte	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐	☐
Frisk persille	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐	☐
Frisk timian	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐	☐

	< 1	1	2	3	1	2-4	5-7	8+		1/2	1-2	3-4	5-6	7+
Annet krydder, tørket eller friskt, spesifiser:														
.....	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐	☐
.....	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐	☐

17. Frukt Svar enten per måned eller per uke.

	Sjelden /aldri	Gang pr. måned 1	2	3	eller	Gang pr. uke 1	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, mandariner	☐	☐	☐	☐		☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4+ ☐
Fersken, nektarin	☐	☐	☐	☐		☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4+ ☐
Kiwi	☐	☐	☐	☐		☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4+ ☐
Druer	☐	☐	☐	☐		☐	☐	☐	☐	☐	(stk)	1-5 ☐	6-10 ☐	11-20 ☐	21+ ☐
Melon	☐	☐	☐	☐		☐	☐	☐	☐	☐	(skive)	1 ☐	2 ☐	3 ☐	4+ ☐
Granateple	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Svisker	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Tørket frukt (aprikos o.l)	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Rosiner	☐	☐	☐	☐		☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐

Hvor mange frukter spiser du vanligvis pr. dag? Mindre enn 1 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5+ ☐

18. Nøtter og frø Ta med nøtter og frø i løsvekt og i bakverk. Husk å krysse av for mengde

	Aldri	Gang pr. måned <1	1	2	3	eller	Gang pr. uke 1	2-3	4-5	6-7	8+	Mengde pr. gang	1	2-3	4-5	6+
Cashewnøtter	☐	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Valnøtter	☐	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Hasselnøtter	☐	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Mandler (brune, uskållede)	☐	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Peanøtter	☐	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Pecannøtter	☐	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Pinjekjerner	☐	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Pistasjenøtter	☐	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Sesamfrø	☐	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Solsikkekjerner	☐	☐	☐	☐	☐		☐	☐	☐	☐	☐	(ss)	☐	☐	☐	☐

19. Bær

Oppgi inntaket av bær for sesongen og resten av året hver for seg. Med "sesong" mener vi de 1-2 månedene bærene kan spises friske. "Resten av året" er de resterende 10-11 månedene. Inkluder både friske bær, frosne bær og frysetøy. Frysetøy= bær som du har rørt selv og frosset ned (f.eks. hjemmelaget syltetøy).

Bær - i sesongen Svar enten pr. måned eller pr. uke.

		Gang pr. måned				eller	Gang pr. uke				Mengde pr. gang				
	Aldri	< 1	1	2	3		1-2	3-4	5-6	7+	Enhet	1/2	1	2-3	4+
Bjørnebær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Blåbær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Bringebær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Jordbær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Kirsebær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Moreller	☐	☐	☐	☐	☐		☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Multer	☐	☐	☐	☐	☐		☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Nyper	☐	☐	☐	☐	☐		☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Rips	☐	☐	☐	☐	☐		☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Solbær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Tyttebær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(ss)	☐	☐	☐	☐

Bær - resten av året Svar enten pr. måned eller pr. uke.

		Gang pr. måned				eller	Gang pr. uke				Mengde pr. gang				
	Aldri	< 1	1	2	3		1-2	3-4	5-6	7+	Enhet	1/2	1	2-3	4+
Bjørnebær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Blåbær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Bringebær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Jordbær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Kirsebær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Moreller	☐	☐	☐	☐	☐		☐	☐	☐	☐	(dl)	☐	☐	☐	☐
Multer	☐	☐	☐	☐	☐		☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Nyper	☐	☐	☐	☐	☐		☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Rips	☐	☐	☐	☐	☐		☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Solbær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(ss)	☐	☐	☐	☐
Tyttebær	☐	☐	☐	☐	☐		☐	☐	☐	☐	(ss)	☐	☐	☐	☐

20. Desserter, kaker, godteri

Svar enten per måned eller per uke.

	Sjelden /aldri	Gang pr. måned 1	2	3	eller 1	Gang pr. uke 2-3	4-5	6-7	8+	Mengde pr. gang				
Hermetisk frukt, fruktgrøt	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Frisk fruktsalat	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Pudding (eks. sjokolade, karamell)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4+ ☐
Is (1 dl=1 pinne=1 kremmerhus)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4+ ☐
Saftis (1 dl=1 pinne=1 kremmerhus)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4+ ☐
Pisket krem	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	1 ☐	2 ☐	3 ☐	4+ ☐
Vaniljesaus	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	2 ☐	3+ ☐
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+ ☐
Sjokoladekake	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Marsipankake, bløtkake	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Søt kjeks, kakekjeks (Cookies, Bixit, Hob Nobs o.l.)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-2 ☐	3-4 ☐	5-6 ☐	7+ ☐
Pastiller uten sukker	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-2 ☐	3-4 ☐	5-6 ☐	7+ ☐
Drops, pastiller, lakris, seigmenn	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-2 ☐	3-4 ☐	5-6 ☐	7+ ☐
Smågodt (1 hg = 100g)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(hg)	1/2 ☐	1 ☐	2 ☐	3+ ☐
Potetgull	☐	☐	☐	☐	☐	☐	☐	☐	☐	(neve)	1-2 ☐	3-5 ☐	6-10 ☐	10+ ☐
Annen snacks (skruer, crisp, saltstenger, lettsnacks o.l.)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(neve)	1-2 ☐	3-5 ☐	6-10 ☐	10+ ☐
Ekstra mørk sjokolade (minst 70% kakao)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(100g)	1/4 ☐	1/2 ☐	1 ☐	2+ ☐
Vanlig mørk sjokolade, mørk kokesjokolade, Dronningsjokolade o.l	☐	☐	☐	☐	☐	☐	☐	☐	☐	(100g)	1/4 ☐	1/2 ☐	1 ☐	2+ ☐
Annen sjokolade, Melkesjokolade, Firkløver, Kvikklunch, Mars o.l	☐	☐	☐	☐	☐	☐	☐	☐	☐	(100g)	1/4 ☐	1/2 ☐	1 ☐	2+ ☐

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

	Sjelden /aldri	Gang pr. uke 1	2-3	4-5	6-7	Mengde pr. gang			
						1 ts	1 bs	1 ss	
Tran	☐	☐	☐	☐	☐	☐	☐	☐	
						1	2	3	4+
Trankapsler	☐	☐	☐	☐	☐	(kapsler) ☐	☐	☐	☐
Fiskeoljekapsler	☐	☐	☐	☐	☐	(kapsler) ☐	☐	☐	☐
Omega-3 kapsler	☐	☐	☐	☐	☐	(kapsler) ☐	☐	☐	☐
Seloljekapsler	☐	☐	☐	☐	☐	(kapsler) ☐	☐	☐	☐
Mulitvitamin (tablett)	☐	☐	☐	☐	☐	(tablett) ☐	☐	☐	☐
Multimineral (tablett)	☐	☐	☐	☐	☐	(tablett) ☐	☐	☐	☐
B-vitaminer (evt flere b-vitaminer i samme tablett)	☐	☐	☐	☐	☐	(tablett) ☐	☐	☐	☐
C-vitamin (tablett)	☐	☐	☐	☐	☐	(tablett) ☐	☐	☐	☐
D-vitamin (dråper)	☐	☐	☐	☐	☐	(dråper) ☐	☐	☐	☐
E-vitamin (tablett)	☐	☐	☐	☐	☐	(tablett) ☐	☐	☐	☐
Folat (folsyre) (tablett)	☐	☐	☐	☐	☐	(tablett) ☐	☐	☐	☐
Kalsium (tabletter)	☐	☐	☐	☐	☐	(tablett) ☐	☐	☐	☐
Jerntilskudd (tabletter)	☐	☐	☐	☐	☐	(tablett) ☐	☐	☐	☐
						1 ts	1 bs	1 ss	
Jerntilskudd (flytende)	☐	☐	☐	☐	☐	☐	☐	☐	

Hvis du har krysset av for noen av alternativene ovenfor, vennligst oppgi fullstendig produktnavn
(BRUK BLOKKBOKSTAVER - en bokstav i hver rute)

Hvis du bruker andre kostttilskudd enn alternativene oppgitt ovenfor, vennligst oppgi fullstendig produktnavn
(BRUK BLOKKBOKSTAVER - en bokstav i hver rute)

22. Helse

Vennligst oppgi:

Høyde: ____ cm Vekt: ____ kg Alder: ____ år

Har du, eller har du hatt noen av de følgende diagnosene?

☐ astma	☐ kronisk betennelsessykdom i mage / tarm	☐ hjerteinfarkt, angina
☐ leddbetennelse (leddgikt, urinsyregikt el. andre leddplager)	☐ kronisk betennelsessykdom i luftveiene	☐ diabetes ☐ type 1 ☐ type 2
☐ kreft	☐ slag	☐ høyt blodtrykk
☐ muskel/skjelettlidelser	☐ depresjoner/psykiske lidelser	

Annen sykdom: ____

23. Medisiner

Har du brukt noen medisiner de siste 3 månedene? (Ta med medisiner du har brukt sammenhengende (daglig) i mer enn 1 uke. Husk også medisiner kjøpt uten resept, men ikke ta med helsekostpreparater)

☐ Ja ☐ Nei

HVIS JA, fyll ut:

NAVN på medisinene du bruker/har brukt de siste 3 mnd (en bokstav i hver rute, de første 14 bokstavene holder)	Kryss av hvis du bruker dette nå	Antall måneder eller år du har brukt medisinene

Dersom du ikke husker navnet, skriv for eksempel: Østrogener, Betennelsesdempende, Smertestillende

____	☐	____ mnd	**eller**	____ år	
____	☐	____ mnd	**eller**	____ år	
____	☐	____ mnd	**eller**	____ år	
____	☐	____ mnd	**eller**	____ år	
____	☐	____ mnd	**eller**	____ år	

24. Fysisk aktivitet

Har du noen kroniske sykdommer eller tilstander som gjør at du ikke kan utføre fysisk aktivitet?

☐ Nei ☐ Ja, angi grunn { ☐ leddgikt ☐ hofte/kneplager ☐ ryggplager ☐ annet: _____

Tenk gjennom hvor lang tid du i løpet av en vanlig uke tilbringer med fysisk aktivitet. Ta bare med episoder som varer i alle fall 10 minutter. Hvor lang tid tilbringer du hver uke på:

Turgåing (og rolig skigåing)	Middels anstrengende aktiviteter som krever moderat innsats og får deg til å puste litt mer enn vanlig (som å sykle i moderat tempo, svømme i moderat tempo, jogge rolig, gå relativt raskt på ski, dans):	Meget anstrengende aktiviteter som krever hard innsats og får deg til å puste mye mer enn vanlig (som aerobics, løpe eller sykle fort, svømme fort, gå raskt på ski):
timer per uke	timer per uke	timer per uke
☐ ingenting	☐ ingenting	☐ ingenting
☐ mindre enn 1/2 time	☐ mindre enn 1/2 time	☐ mindre enn 1/2 time
☐ 1/2 til 1 time	☐ 1/2 til 1 time	☐ 1/2 til 1 time
☐ 1 1/2 - 2 timer	☐ 1 1/2 - 2 timer	☐ 1 1/2 - 2 timer
☐ 2 1/2 -3 1/2 timer	☐ 2 1/2 -3 1/2 timer	☐ 2 1/2 -3 1/2 timer
☐ 4-6 timer	☐ 4-6 timer	☐ 4-6 timer
☐ 7 eller flere timer	☐ 7 eller flere timer	☐ 7 eller flere timer

25. Røyking

Ta med både fabrikklagde og hjemmerullede sigaretter.

Røyker du? ☐ Nei ☐ Ja

Hvis ja, hvor mye? ☐☐☐ Sigaretter pr. uke **eller** ☐☐☐ Sigaretter pr. dag

Har du tidligere røykt og sluttet? ☐ Nei ☐ Ja

Hvis ja, hvor mye pleide du å røyke? ☐☐☐ Sigaretter pr. uke **eller** ☐☐☐ Sigaretter pr. dag

Hvor mange år eller måneder røykte du? ☐☐ år **eller** ☐☐ mnd

Hvor mange år eller måneder er det siden du sluttet å røyke? ☐☐ år **eller** ☐☐ mnd

26. Eventuelle andre matvarer

Bruker du regelmessig matvarer, drikker eller andre produkter (feks. kosttilskudd) som ikke er nevnt i spørreskjemaet? Skriv ned dette så detaljert som mulig. Ta med produktnavn og produsent hvis 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**.**

27. Har du noen kommentarer til skjemaet kan du skrive det her.

Har du husket å skrive under på samtykkeerklæringen på side 3?

(vi kan ikke bruke skjemaet ditt hvis du ikke har skrevet under på denne)

Tusen takk for innsatsen!

Appendix II

UNIVERSITETET I OSLO
DET MEDISINSKE FAKULTET

Kjære deltager. April 2009

Takk for at du var villig til å delta i vår kostholdsundersøkelse som er viktig for å forstå hvordan brystkreft og andre kroniske sykdommer kan forebygges hos kvinner. Målet vårt er å inkludere 40,000 kvinner i denne undersøkelsen.

På kostholdsskjemaet ditt svarte du "ja" til å avgi en spytt/fingerprikk blodprøve.

Vedlagt finner du en samtykkeerklæring med mer informasjon om undersøkelsen. Vi ber om at du leser igjennom og skriver under på denne. Du har også mottatt instruksjoner og prøvetakingsutstyr for spytt- (se grønt ark) og fingerprikkprøven (gult ark) samt en frankert returkonvolutt. Les nøye igjennom instruksene før du tar spytt og fingerprikk prøvene. Vi setter stor pris på at du returnerer prøvene sammen med underskrevet samtykkeerklæring i løpet av en uke eller to.

Informasjonen vil bare bli brukt til dette forskningsformålet, og resultatene vil behandles konfidensielt. Dersom du har noen spørsmål kan du ringe 22 85 13 80 og legge igjen en beskjed slik at vi ringer deg opp igjen.

Tusen takk for hjelpen!

Med vennlig hilsen

Giske Ursin
Lege og Professor
Prosjektleder

Amrit Kaur Sakhi
Postdoktor
Prosjektmedarbeider

Christian A. Drevon
Lege og Professor

KOST OG BRYSTKREFT I NORGE

SPYTT OG FINGERPRIKK BLODPRØVE

INFORMASJON OG SAMTYKKE ERKLÆRING

Ansvarlig: Professor Giske Ursin, Avdeling for ernæringsvitenskap, Universitetet i Oslo (UiO)

Vi ber om tillatelse til å ta en spytt- og fingerprikkprøve som ledd i vår undersøkelse av kosthold, mammografi og kreft hos kvinner.

HENSIKTEN MED STUDIEN
Som ledd i vårt arbeid for å forebygge brystkreft og andre kroniske sykdommer hos kvinner ønsker vi å lære mer om kosthold hos kvinner, hvordan kostholdet innvirker på faktorer i blodet, og hvordan kosthold og hormoner virker inn på normalt brystvev slik vi ser det på mammografi.

METODE
Vi vil undersøke om kostholdsprodukter og hormoner i blodet samt gener som kan påvirke nedbrytningen av disse kan forutsi tettheten i brystvevet slik vi ser det på mammogrammer. Vi vil også undersøke følgende hos alle deltakerne samlet: tetthet i brystvevet, informasjon om eventuell hormonbruk ved tidligere screeninger, opplysninger fra kostholdsspørreskjemaet, opplysninger fra skjemaet som du leverte på mammografi screeningen og informasjon fra spytt- og fingerprikkprøvene. Vi ønsker også å koble dataene fra alle deltagerne mot blant annet kreftregisteret/mammografiscreeningen, men også reseptregisteret og pasientregisteret, for å forstå hvordan kreft og annen kronisk sykdom kan forebygges. Spytt-/fingerprikkprøvene vil inngå i en biobank. Ansvarlig for denne biobanken er Dr. Giske Ursin. Prøvene skal kun brukes til forskningsformål og planlegges analysert i Norge, men kan også bli analysert i utlandet.

PERSONVERN OG TILGANG TIL OPPLYSNINGER
Resultatene vil bli behandlet fortrolig og alle involverte har taushetsplikt. Alle opplysninger vil kun bli brukt til forskningsformålet som beskrevet over. Prosjektet forventes avsluttet ved utgangen av 2012, men opplysningene kan bli lagret i anonym form etter prosjektslutt for fremtidige data analyser og koblinger med helseregistere i inntil 15 år. Resultatene fra studiene vil bli publisert i anonym form i internasjonale medisinske tidsskrifter. Prosjektet har konsesjon fra Datatilsynet.

DELTAGELSE I STUDIEN
Det er frivillig å delta. Hvorvidt du deltar eller ikke spiller ingen rolle for din mammografi-undersøkelse eller eventuell annen behandling du måtte trenge. Du kan trekke deg uten at du trenger å gi en begrunnelse. Det får ingen konsekvenser for deg, og vi vil i så fall slette dine opplysninger fra forskningsprosjektet, og destruere din spytt- og fingerprikkprøve. Dette forutsetter at du trekker deg før vi har gjort dataene anonyme. Du vil ikke ha personlig nytte av å delta i denne studien. Du vil ikke få noen resultater fra denne undersøkelsen, ettersom det er usikkert i hvilken grad tettheten i brystvevet eller noen av testene vi utfører på spytt- eller fingerprikkprøvene har noen klinisk betydning.

SAMTYKKEERKLÆRING
Jeg samtykker i å delta i denne undersøkelsen, og i at opplysningene kan lagres og brukes i forskning etter prosjektslutt som angitt ovenfor.

Navn: _____

Underskrift:_____Dato: _____

TLF: _____(dersom vi har spørsmål angående prøvene dine)

OK å ringe mellom kl. _____ og kl._____

Kan vi kontakte deg igjen senere i løpet av prosjektperioden og spørre om du vil være med i en annen undersøkelse om kosthold og mammogrammer? _____Nei _____Ja