Ability of a lean-seafood diet to modulate lipid and glucose metabolism in healthy humans

- a randomized controlled trial with a crossover design

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The PhD has been part of a collaborating project with Haukeland University Hospital (HUH), Hormone Laboratory, Norway; University of Aarhus, Department of Food Science, Denmark and Laval University (LU), School of Nutrition, Canada.

Supervisors were Dr. Ingvild Eide Graff and Dr. Bjørn Liaset at NIFES and co-supervisors were Dr. Hélène Jacques at LU, Dr. Asle Holthe at BUC and Prof. Gunnar Mellgren at UiB.

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Abstract

Background: A Westernised lifestyle, which involves a high-energy diet and reduced physical activity, is indisputably linked to the pandemics of obesity and type 2 diabetes. Prevention of cardiovascular disease and type 2 diabetes is a public health goal. Intake of fish has been associated with reduced risk of cardiovascular disease, but data from randomized controlled trials have been inconclusive. Lean fish contains relatively low amount of marine omega-3 fatty acids, and data from both animal and human studies indicate a beneficial effects on lipid metabolism, insulin sensitivity and glucose homeostasis. Studies investigating the potential protective effect of lean-seafood in healthy subjects are warranted.

Aim: The overall aim of this thesis was to elucidate how lean-seafood can modulate fasting and postprandial metabolism of lipids and glucose in healthy humans.

Subjects and Methods: Healthy Caucasian subjects were recruited from the great area of Bergen. The study included two 4-weeks experimental periods separated by a 5-weeks washout period in a crossover design. Prior to each experimental period, the subjects followed a diet in accordance with the Norwegian dietary recommendations for 3 weeks run-in periods. Half of the group (6 men and 8 women) was randomly assigned to begin with a lean-seafood diet and the other group (4 men and 9 women) to a nonseafood diet. The lean-seafood diet consisted of lunch- and dinner meals with cod, pollack, saithe and scallops and the nonseafood diet contained skinless chicken filet, lean beef, turkey, pork, egg, milk and milk products. The protein contribution from the experimental protein sources in both diets corresponded to 60 % of total protein intake, and the remaining dietary proteins came from vegetable and cereal sources.

Results: Healthy subjects had after 4 weeks lean-seafood intervention, a highly significant reduction in fasting and postprandial circulating TAG concentrations, relative to the 4 weeks nonseafood intervention. There is evidence that raised circulating TAG levels are associated with increased coronary heart disease risk. The TAG/ HDL-cholesterol ratio was decreased during the lean-seafood intervention and

increased during the nonseafood intervention. The intervention did not alter fasting and postprandial serum glucose or insulin concentration. However, lean-seafood intake reduced postprandial C-peptide and lactate concentrations. Lean-seafood intake improved mitochondrial oxidative capacity as indicated by human urinary metabolomics. All results are consistent, indicating an improved preservation of insulin-sensitivity after lean-seafood consumption.

Conclusion: Based on our data lean-seafood regulates fasting and postprandial lipids and glucose metabolism differently in healthy subjects after four weeks. Lean-seafood modulate fasting and postprandial lipids, and postprandial glucose metabolism in healthy individuals in a manner that may have an effect on the long-term development of cardiovascular disease, insulin-resistance and type 2 diabetes.

An increased intake of lean-seafood should be encouraged as a part of a healthy diet in the prevention of CVD and T2D, and therefore may have a part of combating the development of these health challenges.

List of publications

Paper I

Aadland, E. K., Lavigne, C., Graff, I. E., Eng, Ø., Paquette, M., Holthe, A., Mellgren, G., Jacques, H. and Liaset, B. (2015). ”Lean-seafood intake reduces cardiovascular risk factors in healthy subjects: results from a randomized controlled trial with a crossover design”. *Am J Clin Nutr*. 102(3):582-592

Paper II

Aadland, E. K., Graff, I. E., Lavigne, C., Eng, Ø., Paquette, M., Holthe, A., Mellgren, G., Madsen, L., Jacques, H. and Liaset, B. (2015). “Lean-seafood intake reduces postprandial C-peptide and lactate concentration in healthy subjects - results from a randomized controlled trial with a crossover design”. (Submitted *Sept 2015*).

Paper III

Schmedes, M., **Aadland, E. K**., Sundekilde, U. K., Jacques, H., Lavigne, C., Graff, I. E., Eng, Ø., Holthe, A., Mellgren, G., Young, J. F., Bertram, H. C., Liaset, B. and Clausen, M. R. (2015). “Lean-seafood intake improves mitochondrial oxidative capacity as indicated by human urinary metabolomics”. (Submitted *Sept 2015*).

The papers are from now on referred to by their roman numbers

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Abbreviations

ANOVA	Analysis of variance
ApoB	Apolipoprotein B
BMI	Body mass index
BUC	Bergen University College
CHD	Coronary heart disease
CM	Chylomicrons
CRP	C-reactive protein
CVD	Cardiovascular disease
DHA	Docosahexaenoic acid
DXA	Dual-energy x-ray absorptiometry
EGP	Endogenous glucose production
EI	Energy intake
EPA	Eicosapentaenoic acid
FFA	Free fatty acid
FFQ	Food frequency questionnaire
GLP-1	Glucagon-like peptide 1
HDL	High-density lipoprotein
HUH	Haukeland University Hospital
IDL	Intermediate-density lipoprotein
IS	Insulin sensitivity
LDL	Low-density lipoprotein
LC-MS	Liquid chromatography mass spectrometry
LPL	Lipoprotein lipase
LU	Laval University
MUFA	Monounsaturated fatty acids
NCD	Non-communicable diseases

NEFA	Non esterified fatty acids
NIFES	National Institute of Nutrition and Seafood Research
NMR	Nuclear magnetic resonance
PUFA	Polyunsaturated fatty acids
RCT	Randomized controlled trial
SEM	Standard error of the mean
SFA	Saturated fatty acids
TAG	Triacylglycerol
TMA	Trimethylamine
TMAO	Trimethylamine N-oxide
T2D	Type 2 diabetes
VLDL	Very low density lipoprotein
WHO	World Health Organization
2PY	N1-methyl-2/4-pyridone-5/3 carboxamide
3MH	3-methylsistidine

1. Introduction

1.1 The global strategy on diet and health

The global burden of non-communicable diseases (NCD) increases and is the biggest cause of deaths worldwide. More than 36 million people die annually from NCDs (63% of global deaths), including 14 million people before the age of 70. More than 90% of these deaths from NCDs occur in low- and middle-income countries (WHO 2013). The burden of mortality, morbidity and disability attributable to chronic disease problem is far from being limited to the developing countries. Chronic diseases are emerging both in poorer countries and in poorer population groups in developed countries (WHO 2004; Lozano *et al.* 2012).

For all countries, the underlying behavioural factors are largely the same. An elevated consumption of energy-dense, nutrient-poor foods that are high in fat, sugar and salt combined with reduced levels of physical activity are the main factors that increase the major chronic diseases (WHO 2004). The burden of diet-related chronic diseases is attributable to cardiovascular diseases mainly. Obesity and diabetes are also showing worrying trends, not only because they already affect a large proportion of the population, but also because they appear earlier in life. Chronic diseases are largely preventable diseases. The public health approach of primary prevention is considered to be the most cost-effective, affordable and sustainable course of action to cope with the chronic disease epidemic worldwide (WHO 2013; Ryden *et al.* 2007).

A Westernised lifestyle, which involves a high-energy diet and reduced physical activity, is indisputably linked to the pandemics of obesity and type 2 diabetes (Nolan *et al.* 2011). A meta-analysis of 102 prospective studies (almost 700 000 people) without previous vascular disease from the Emerging Risk Factors Collaboration provides information about the relation between glucose concentrations, diabetes status, and cardiovascular outcomes. The analysis showed that diabetes confers about a two-fold excess risk for coronary heart disease, major stroke subtypes, and deaths attributed to other vascular causes (Sarwar *et al.* 2010). Almost 4 of 5 cases of myocardial infarction in healthy men may be prevented with low-risk behaviour like;

healthy diet consumption, moderate alcohol consumption, no smoking, being physically active and having a healthy body weight. A healthy diet can prevent 1 of 5 myocardial infarction alone (Åkesson *et al.* 2014).

1.2 Healthy diets

The Mediterranean diet has been reported to be a model of healthy eating for its contribution to a favourable health status. A traditional Mediterranean diet is rich in bread, root- and green vegetables, fruit, oil (high in linoleic acid) and fish, and low in meat, butter and cream. A Mediterranean diet is associated with decreased cardiovascular risk (Sofi *et al*. 2008) and are shown to prevent secondary cardiovascular disease (CVD) (de Lorgeril *et al*. 1994; 1996; 1999). The protective effect of the Mediterranean dietary pattern was maintained for up to four years after the first myocardial infarction (de Lorgeril *et al*. 1999). In high cardiovascular risk subjects, intake of the Mediterranean diet, supplemented with extra-virgin olive oil or mixed nuts, resulted in a substantial reduction in the incidence of major cardiovascular events (Estruch *et al.* 2013). In this primary prevention trial Estruch et al. (2013) suggested a potentially greater benefit of the Mediterranean diet as compared with Western diets.

Differences in food cultures, limited accessibility to local resources and ecological aspect may hamper other populations, such as Scandinavians, from consuming a Mediterranean-like diet (Papadaki & Scott 2002). An alternative to the Mediterranean diet is the regional Nordic diet, using foods naturally grown in the Nordic countries, such as apples and berries, rye, rapeseed oil, salmon, roots, cabbages, peas, and dairy products; furthermore, the long coastlines provides rich sources of fish (Bere & Brug 2009). Intake of a healthy Nordic diet improved lipid profiles and insulin sensitivity, and decreased body weight and blood pressure in 88 Swedish hypercholesterolaemic subjects (Adamsson *et al*. 2011; Uusitupa *et al*. 2013). These results are in agreement with those of a controlled study conducted in 131 pre-diabetic Finnish participants

suggesting an improved glucose metabolism after consumption of a Nordic diet (Lankinen *et al*. 2011).

Behind all healthy diets, there is the concept of change of the usual diet towards a healthy dietary pattern using local and seasonal products. Development of country-specific guidelines is needed to provide practical educational instruments, which consider variation in dietary patterns, accessibility to foods, and agriculture in different regions globally (Ryden *et al.* 2007; Paulweber *et al*. 2010; Ley *et al.* 2014).

1.2.1 National dietary guidelines and food consumption in Norway

The Norwegian dietary recommendations (The Norwegian Directorate of Health 2014) are based on the Nordic Nutrition Recommendations 2012 published by The Nordic Council of Ministers (2014) and have a main focus to prevent chronic diet-related diseases in the population. The recommendations are directed primarily towards healthy adult subjects with normal levels of physical activity since the research that forms the knowledge base, is performed on this part of the population mainly (The Norwegian Directorate of Health 2011). The diet recommendations are based on foods and food cultures that are common in Norway. A healthy diet should be predominantly plant based and containing vegetables, fruits, berries, whole grains and fish. It is recommended to achieve energy balance and a healthy weight. Moreover, it is recommended to limit the intake of salt, added sugars, and energy intake from total fats, and to shift fat consumption from saturated fats (SFA) to unsaturated fats (The Norwegian Directorate of Health 2011; WHO 2013).

The daily average intake of salt in Norway is estimated to be around 10 grams per person. This is twice as high as recommended. Processed food and pre-prepared meals contributes to a large extent to the salt intake in the population. The consumption of sugar has decreased the last ten years. Today, added sugar contributes to 13 % of the daily energy intake, but is still higher than the recommended level of less than 10% of the daily energy intake (Totland *et al.* 2012). The average Norwegian population

spends three times as much money on sweets and soft drinks as on fish (The Norwegian Directorate of Health 2015).

The amount of fruits, vegetables and berries should be at least 500 gram per day, approximately half of the amount should be vegetables and the other half should consist of fruits and berries. Most individuals eat less than recommended (The Norwegian Directorate of Health 2011). The recommended intake of vegetables of at least 250 gram per day was achieved by about 15 % of men and women (Totland *et al.* 2012). It is desirable to have an increase in vegetable consumption (The Norwegian Directorate of Health 2015). The recommendation of four servings of whole grain products per day is equivalent to approximately 70-90 gram whole grains per day. The average intake of whole grains in the Norwegian population is estimated to be approximately 50 gram per day. Probably a large percentage of the population is therefore eating significantly less than the recommendations (The Norwegian Directorate of Health 2011). The recommended amount of two to three servings of fish for dinner and some servings of fish as spread per week, is equivalent to 300-450 grams per week. Both lean and fatty fish should be included, but at least 200 gram of fatty fish is recommended per week. In the national dietary survey Norkost 3, from 2010-11, the average intake of fish is 310 grams a week for women and 450 grams a week for men. From the average intake, lean fish contributed most with 60 percent of the total fish consumption, while fatty fish contributed with 40 percent. About half of the Norwegian population eats less fish than the national dietary recommendation. Among women it was 31 percent and among men it was 39 percent, who consumed more than 375 grams fish per week. In pregnant women the average total fish intake was 217 grams a week, and lower than the average women (Totland *et al.* 2012). The consumption of fish in Norway has been stable for the past ten years, but it is lower than desirable and substantially lower than the consumption of meat (560 gram per week) (The Norwegian Directorate of Health 2015).

In spite of several positive trends in food consumption in recent years, large parts of the Norwegian population have a diet with significant nutritional weaknesses that may contribute to the development of cardiovascular diseases, cancers, obesity, type 2

diabetes, constipation, tooth decay and iron deficiency. Adopting the Norwegian Directorate of Health's recommendations for a healthy diet and physical activity is likely to reduce the incidence of these diseases (The Norwegian Directorate of Health 2015).

1.3 Seafood consumption and prevention of cardiovascular diseases

An important component of a healthy dietary pattern is fish. During the last two decades, several epidemiological studies and clinical trials have indicated the beneficial effects of fish intake in the primary and secondary prevention of several diseases, including CVD. CVD is a collective term for conditions that affect the whole blood circulatory system, ie, the heart and blood vessels (The Norwegian Directorate of Health 2011). A large number of prospective studies have shown that regular fish consumption is related to a lower risk of CVD such as stroke (He *et al.* 2004a) and coronary heart disease (CHD) (He *et al.* 2004b). The health promoting effect of fish has primarily been ascribed to the long-chain n-3 polyunsaturated fatty acids (n-3 PUFA), eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) (Dyerberg *et al.* 1978). A meta-analysis of randomised intervention trials showed that n-3 PUFA significantly reduced deaths from cardiac causes in patients with CHD (Leon *et al.* 2008). Even though some of the cardioprotective effects of fish consumption could be ascribed to marine n-3 fatty acids (Mozaffarian & Wu 2011), one study showed that intake of as little as one or two dishes of lean fish a week, which contains relatively low amounts of marine n-3 fatty acids, was also inversely associated with CHD mortality (Kromhout *et al*. 1985).

The protein moiety in fish is also considered to have positive effects in relation to lifestyle-related diseases. The 26-year follow up Nurses` Health Study demonstrated a significant association between intake and outcome in risk of coronary heart disease from choosing fish, poultry, nuts and low fat dairy as major dietary protein source, compared to red meat and high-fat dairy (Bernstein *et al.* 2010).

Lean fish intake, whose major constituent is fish protein, has induced lower total cholesterol and triacylglycerol (TAG) concentrations in human subjects (Gunnarsdottir *et al.* 2008). In normolipidemic (Lacaille *et al.* 2000) and hypercholesterolemic (Beauchesne-Rondeau *et al.* 2003) men, lean fish intake provoked an increase of high-density lipoprotein (HDL), mainly as HDL_2, the cardioprotective lipoprotein fraction. The effects of lean fish consumption have been examined in premenopausal (Gascon *et al.* 1996) and postmenopausal (Jacques *et al.* 1992) women given well-controlled low-fat (30%), high PUFA:SFA (1:1) ratio diets. In these studies, intake of lean fish induced a lower plasma TAG concentration and higher concentrations of low-density lipoprotein (LDL) - apolipoprotein B (apo B) in plasma than other animal protein sources. In postmenopausal women, lean fish, compared with other animal protein sources, induced higher concentrations of plasma total and HDL cholesterol (Jacques *et al.* 1992). However, those studies have been conducted in human subjects in the fasting state only. In keeping with the fact that development of atherogenesis might be a postprandial phenomenon (Zilversmit 1979), postprandial studies is warranted. The three prospective studies, the Women`s Health Study (Bansal *et al*. 2007), the Norwegian Counties Study (Lindman *et al*. 2010) and the Copenhagen City Heart Study (Nordestgaard *et al*. 2007) have confirmed the association of postprandial TAG as a risk factor for CVD. Non-fasting lipid concentration might be a better indicator of average lipid concentrations in the blood rather than fasting concentrations (Nordestgaard & Varbo 2014). Furthermore, most people spend the majority of the day in the postprandial state.

1.3.1 Lipid metabolism

A healthy diet consist of all three major macronutrients: protein, carbohydrate and lipids. Over 95% of dietary lipids are TAGs, the rest are phospholipids, free fatty acids (FFAs), cholesterol (present in foods as free and esterified cholesterol), and fat-soluble vitamins. In the cells of the small intestine dietary TAG are packed with cholesterol and phospholipids into chylomicrons (CM), the largest of the lipoprotein-particles. The

CM particles also contain one apolipoprotein B48 (apoB48) as a structural protein. Ninety % of the chylomicron TAG is converted to fatty acids and glycerol, which are taken up by adipocytes and muscle cells for energy use or storage. After fat ingestion the concentration of CM in the blood increases transiently, as these particles have relatively short half-life in healthy subjects, approximately 5 min (Grundy & Mok 1976). Cholesterol-rich chylomicron remnants are taken up by the liver. Very low density lipoprotein (VLDL) particles are secreted continuously from the liver, for delivery of TAG in the postprandial state. In contrast to CM and chylomicron remnants, VLDL are characterized by their apoB100 content. The secretion of VLDL is under complex regulation, as secretion of the larger and more TAG-rich VLDL species are repressed by insulin signaling (Malmström *et al*. 1997; Adiels *et al*. 2007; Adiels *et al*. 2008). In circulation, the VLDL are converted to intermediate-density lipoprotein (IDL) and then further to LDL by lipoprotein lipase. LDL are depleted of TAGs, phospholipids and are enriched in cholesteryl esters. The LDL particles bind to LDL receptors on all cells, and the entire particle is taken up by the cells. Once inside the cell cholesterol can be used to produce steroid hormones or contribute as a structural element in the cell membranes. Raised concentrations of LDL cholesterol predisposes an individual to cardiovascular disease, and LDL lowering is a prime lipid target (Nordestgaard & Varbo 2014). HDL cholesterol is responsible for the removal of excess peripheral cholesterol and its return to the liver. HDL receptors in the liver are receiving cholesterol esters from HDL, enabling the HDL particle to continue the reverse cholesterol transport from peripheral tissues. In the liver cholesterol can be used to produce bile acids (Rashid *et al*. 2003; Tremblay *et al*. 2007; McQueen *et al*. 2008). Raised TAG concentrations are strongly associated with low concentrations of HDL cholesterol (Varbo *et al.* 2013). An overview of postprandial lipid metabolism are outlined in **Figure 1.1**.

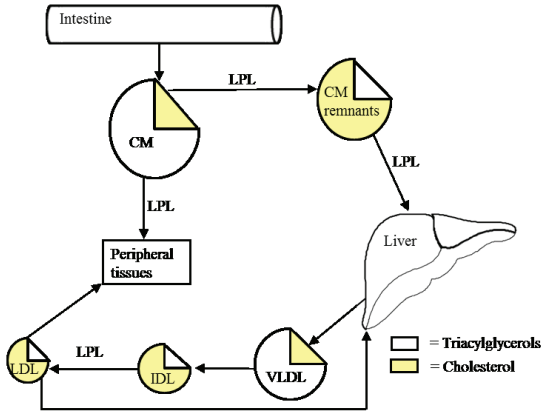

Figure 1.1 Simplified flowchart showing the essentials of lipoprotein metabolism. CM = chylomicron, LPL = Lipoprotein lipase, VLDL = very low density lipoprotein, IDL = intermediate-density lipoprotein, LDL = low density lipoprotein.

1.4 Seafood consumption and prevention of diabetes type 2

Lifestyle changes that include moderate weight loss and regular physical activity (150 min/week), with dietary strategies including reduced energy intake and reduced intake of dietary fat, may reduce the risk for developing diabetes and are therefore recommended as shown in the Diabetes Prevention Program (Knowler *et al.* 2002; Bantle *et al.* 2008) and in the European evidence-based guideline (Paulweber *et al.* 2010). The WHO has estimated that 90% of type 2 diabetes (T2D) can be prevented by changes in diet, physical activity and smoking habits (WHO 2013). Several studies among people with overweight and impaired glucose tolerance have shown that changing dietary and exercise habits in line with current recommendations can prevent or reduce the risk of T2D by 40-60% over a period of approximately a decade (Tuomilehto *et al.* 2001; Knowler *et al.* 2002; Lindstrom *et al.* 2006). Intake of diets rich in whole grains, fruits, vegetables, legumes, nuts and low in refined grains, red or processed meats, and sugar-sweetened beverages have been shown to reduce the risk of diabetes and to improve glycaemic control and blood lipids in subjects with diabetes (Ley *et al.* 2014). The potential impact of seafood consumption on the development of insulin-resistance is yet not fully clarified. A number of prospective studies have

explored the association between fish consumption and risk of T2D, with inconclusive results. The protective effect from total fish consumption was observed in Japanese men, but not in women (Nanri *et al.* 2011). Lower incidence of T2D was reported in Chinese women after consumption of fish (Villegas *et al.* 2011). Other prospective cohort studies reported that a higher fish intake did not prevent T2D (van Woudenbergh *et al.* 2009; Kaushik *et al.* 2009; Djousse *et al.* 2012). One difference in the conflicting findings between fish consumption and risk of diabetes was reported between geographical regions, as meta-analyses of prospective studies conducted in North America and Europe indicated an increased risk, while studies performed in Asia showed a protective effect of T2D with fish consumption (Xun & He 2012; Wallin *et al.* 2012; Wylie-Rosett *et al.* 2012). All the above mentioned meta-analyses concluded that further investigation is warranted. Some of the discrepancy in the varying outcomes from the different prospective cohort studies might be the lack of distinction between fatty and lean fish. A recent prospective population based cohort study of Norwegian women (NOWAC) showed inverse association between lean fish consumption and T2D development. The authors marked that it was unclear whether lean fish itself had a protective effect on T2D, or if lean fish consumers have a protective life style that was not possible to take into account in the study (Rylander *et al.* 2014). However, it is also likely that some of the discrepancy in the different prospective cohort studies is caused by the use of validated semi-quantitative food-frequency questionnaires that may cause erroneous food intake reporting. Another contributing factor to the discrepant results may be the differences in amount of fish intake, the different preparation or the cooking methods used in the different locations (Mozaffarian *et al.* 2003; Patel *et al.* 2012).

To detect and understand the association between total and type of fish intake and insulin-sensitivity, an increasing number of intervention studies have been conducted recently. In a test meal-study, healthy women received three test meals with 45 g protein either as cod fillet, cottage cheese (milk protein), or soy protein isolate. Ingestion of the cod protein meal resulted in lower serum insulin/glucose and insulin/C-peptide ratios, as compared to the cottage cheese meal, suggesting that different protein sources affect glucose and insulin metabolism differently (von Post-Skagegard *et al*.

2006). In a 4 week intervention study with cross-over design, dietary cod protein, as compared to a similar diet containing lean beef, pork, veal, eggs, milk, and milk products, improved the insulin sensitivity in insulin-resistant individuals and thus could contribute to prevention of type 2 diabetes by reducing the metabolic complications related to insulin resistance (Ouellet *et al*. 2007). From the same study, a reduced plasma concentration of the systemic inflammation marker C-reactive protein (CRP) was reported (Ouellet *et al*. 2008). The underlying mechanism to the improved insulin-sensitivity by lean fish intake remains to be completely elucidated. However, from studies with rats fed a high-sucrose diet, both cod and soy proteins reduced fasting and postprandial glucose and insulin responses and increased peripheral insulin sensitivity compared with casein (Lavigne *et al*. 2000). In follow-up studies cod protein feeding, as compared to soy protein and casein, prevented rats from developing skeletal muscle insulin-resistance (Lavigne *et al*. 2001). In support of these studies, a free-living randomized study with overweight adults receiving capsules with cod protein or placebo for 8 weeks improved glucose homeostasis and favorably altered body composition in the participants (Vikoren *et al.* 2013). Human studies exploring the association between seafood consumption and risk of T2D have previously only been conducted among overweight or insulin-resistant individuals given single nutrient or a single meal.

1.4.1 Fasting and postprandial glucose metabolism in healthy subjects

In the fasting state the blood glucose concentration is maintained by endogenous glucose production, mainly from hepatic glycogenolysis and gluconeogenesis under the direction of glucagon among others. The brain cannot synthesize glucose or store glycogen, and are therefore dependent on a continuous supply of glucose from plasma (Nolan *et al*. 2011).

After a meal, blood glucose concentration is transiently elevated, which stimulates insulin secretion by islet ß-cells and suppresses glucagon secretion after activation of glucagon-like peptide 1 (GLP-1) (Nolan *et al*. 2011). C-peptide is secreted into the

bloodstream in equal quantities to insulin. Since C-peptide has a longer half-life than insulin (20 – 30 versus 3-5 minutes), and is commonly used in preference to insulin measurement when assessing ß-cell function (Jones & Hattersley 2013). GLP-1 is an incretin hormone, which increases glucose-stimulated insulin secretion and glucose-suppression of glucagon secretion. At the same time endogenous glucose production is suppressed, which helps to curtail total glucose input into blood. Glucose uptake into insulin-sensitive peripheral tissues, such as skeletal muscle and adipose tissue is activated (Nolan *et al*. 2011). The splanchnic bed (liver and gut), the skeletal muscles, and the non-insulin responsive tissues (in particular the brain) each dispose of ~ 1/3 of the ingested glucose (Kelley *et al.* 1988; Woerle *et al.* 2003; Moore *et al.* 2003; Nolan *et al*. 2011). Majority of the postprandial glucose taken up by the liver is stored as glycogen (Kelley *et al.* 1988). In healthy subjects, direct glucose storage accounted for 33% and glycolysis for 67% of the total disposal during the postprandial period. Most of the glucose is oxidized (43.5 %) and about 23.5 % undergoes non-oxidative glycolysis (Woerle *et al.* 2003). Hence, the ability of the liver to store glucose as glycogen after a mix meal, with subsequent release of hepatic glucose from glycogen in the post absorptive phase is important for normal glucose homeostasis in healthy subjects (Nolan *et al*. 2011). An overview of fasting and postprandial glucose metabolism in healthy subjects are outlined in **Figure 1.2**.

Lactate metabolism is profoundly related to glucose metabolism, as lactate formation is believed to arise from pyruvate as part of glycolysis (Garcia-Alvarez *et al.* 2014). Glucose is one of the most important sources of lactate while lactate is a major substrate to synthesize endogenous glucose. In the postabsorptive state, it has been estimated that approximately 65 % of the lactate is derived from glucose while 16 – 20 % of the lactate stems from alanine (Perriello *et al.* 1995). When oxidative capacity decreases, plasma lactate concentration increases as a consequence of greater flux through glycolytic pathways (Del Prato *et al.* 1993).

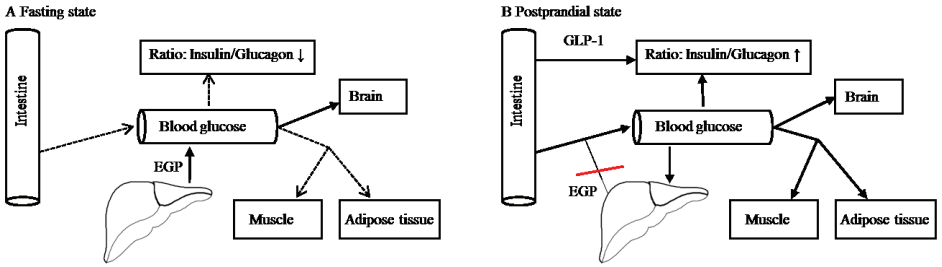

Figure 1.2 A simplified flowchart showing normal fasting and postprandial glucose metabolism. In the fasting state the blood glucose level are decreasing, leading to a low insulin-to-glucagon ratio in plasma. Glucagon markedly increase the release of glucose by the liver. The brain in dependent on glucose, while the entry of glucose into muscle and adipose tissue decreases in response to a low insulin level (A). In the postprandial state the incretin hormone, glucagon-like peptide 1 (GLP-1), increases glucose-stimulated insulin secretion and glucose-suppression of glucagon secretion. Insulin stimulates blood glucose removal by reducing endogenous glucose production (EGP), stimulating peripheral glucose uptake and stimulating glycogen production (B). Modified from Nolan *et al*. 2011.

1.4.2 From a healthy to a diabetic state

Type 2 diabetes is characterized primarily by abnormally high levels of glucose in the blood as a consequence of insulin resistance and relatively impaired ß-cells function. As a compensatory mechanism to the reduced insulin sensitivity, more insulin is released from pancreatic ß-cells. Reduced insulin sensitivity in insulin-responsive tissues may develop as a consequence of obesity, physical inactivity, and genetic predisposition due to an increase in islet ß-cells function (Weir & Bonner-Weir 2004). In a pre-diabetes state robust islet ß-cells are able to successfully compensate insulin secretion as required, and limit increase in liver fat. In this way, blood nutrient level are maintained within the normal range and other tissues, such as the liver, skeletal muscle, heart, and ovaries, are not damaged (Nolan *et al.* 2011). This first stage of evolving ß-cells dysfunction during progression to diabetes can last for years (Weir & Bonner-Weir 2004). The duration of this pre-diabetes state may vary between different ethnic groups. It seems that Caucasian and their descendants differ from other ethnic groups because they can withstand more obesity, particularly increased waist

circumference, before they develop T2D. The WHO therefore recommends to set the body mass index (BMI) limit for overweight to 23 kg/m^2 and obesity to 25 kg/m^2 in Asians. Most likely many non-Western populations develop T2D at a lower BMI, because of genetic factors in combination with rapid lifestyle changes (Barba *et al.* 2004).

The following steps towards developing T2D are crucial. The islet ß-cells are unable to compensate the necessary amount of insulin to maintain a normal glucose level. The high blood glucose levels are caused by increased glucagon secretion and reduced incretin response, increased endogenous glucose production, increased release of free fatty acids from the adipose tissue and development of peripheral insulin resistance (Weir & Bonner-Weir 2004; Nolan *et al.* 2011). In patients with T2D non-oxidative glycolysis is enhanced, and lactate production is consequently increased (Del Prato *et al.* 1993).

Development of T2D is often slow with no clear symptoms early in the disease-phase. The diagnosis is therefore often set too late, and it may be complications already at time of diagnosis (The Norwegian Directorate of Health 2011). Studies suggest that up to 25% of Norwegian individuals with acute myocardial infarction have an undiagnosed diabetes (The Norwegian Directorate of Health 2009). Most people who get T2D will for several years undergo a stage with impaired glucose tolerance before they develop diabetes (Weir & Bonner-Weir 2004). Detection of diabetes and early intervention to reverse hyperglycaemia and other cardiovascular risk factors therefore is important. Both animal and human studies indicate a cardiovascular and T2D preventive effect of lean-seafood consumption.

1.5 The human metabolome

Metabolomics is the study of the complete collection of metabolites present in cells, tissues or biofluids under a particular set of conditions, generating a biochemical profile (Nicholson & Lindon 2008). To characterize and quantify molecules in a biological

sample, methods such as nuclear magnetic resonance (NMR) spectroscopy and mass spectrometry (MS) are often used. MS studies usually require the metabolites to be separated before detection, typically by using liquid chromatography (LC-MS). NMR is generally used to detect hydrogen atoms in metabolites. All hydrogen-containing molecules in a sample will give an NMR spectrum. A typical biological-fluid sample, such as human urine, will contain signals from hundreds of metabolites as long as they are present in concentrations above the detection limit (**Figure 1.3 A**). Each metabolite will be identified by combining spectra analysis and database queries and by comparing, when available, at least two different parameters of the metabolite with those of a reference compound (Sumner *et al*. 2007). Different diets, diseases and environment might give different spectra which are possible to separate by this method. The intensities of peaks in a spectrum are used as coordinates in multidimensional plots of metabolic activity. Each metabolite can be reduced to two- or three-dimensional graphs (**Figure 1.3 B**). Clustering of points can help to visualize and characterize the data (Nicholson & Lindon 2008).

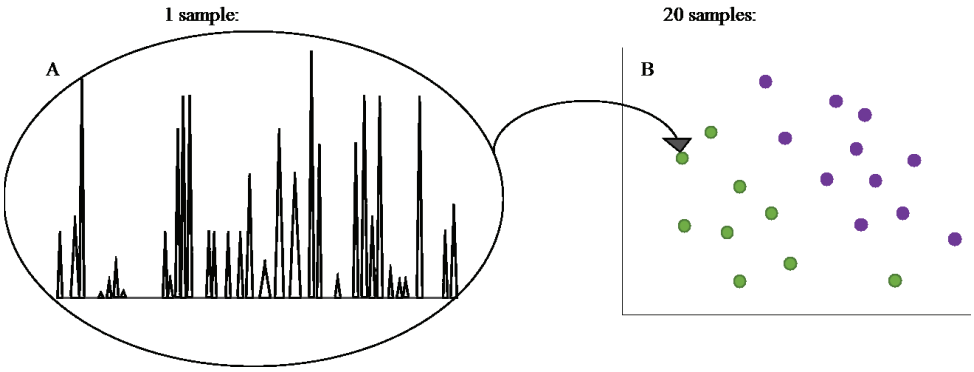

Figure 1.3 Raw individual data (1 sample) from an NMR spectrum of a biological fluid with signal of metabolites (A) will be identified before pattern recognition techniques reduce multivariate data to a two-dimensional plot. This example shows a spectra of biological fluids from 20 samples; nine control subjects (green points) and data from eleven disease subjects (purple points) (B). Adapted from Nicholson & Lindon, 2008.

Targeted approaches focus on a specific subset of the metabolome and provide data only on a predefined set of molecules, while untargeted approaches allow for the discovery of new molecules and generate new hypothesis (Suhre 2014). Untargeted metabolite profiling is used in nutritional studies for a comprehensive analysis of exogenous and endogenous low molecular weight metabolites in a biological fluid after a dietary intervention (Bertram *et al.* 2007; Pellis *et al*. 2012).

1.5.1 Metabolomics as a tool for discovery of metabolic health

Metabolomics have previously been used to identify early markers of cardiovascular diseases and insulin resistance and type 2 diabetes (Roberts *et al.* 2014; Suhre 2014; Soininen *et al*. 2015)

The chemical composition of urine is of particular interest, because it reveals key information not only about a person's health, but also about what they have eaten. Food intake may be reflected by the composition of the urine through two different routes. Firstly, food components that are absorbed in the intestine, but not metabolized, catabolized or modified in the body will be detected directly in the urine. The detection of such urinary metabolites may often directly reflect the composition of the diet. Secondly, specific food components may affect specific biochemical processes and modify the metabolic state of an organism. Such diet-induced cellular metabolic alterations are also reflected in the urine and provide information about how diet impacts the metabolic status of the subject.

1.6 Aims

The overall aim of this thesis was to elucidate how lean-seafood can modulate fasting and postprandial metabolism of lipid and glucose in healthy humans.

1.6.1 Specific aims in the papers

The primary outcome of the study was to elucidate the potential of lean-seafood to regulate fasting and postprandial plasma lipids and lipoproteins, in order to promote cardiovascular health (paper I).

A secondary outcome of the study was to elucidate the potential of lean-seafood to regulate plasma glucose metabolism, in order to prevent development of type 2 diabetes (paper II).

Another predefined outcome was to profile the urinary metabolic response by NMR spectroscopy and LC-MS analyses in order to improve understanding of the diet-induced changes in healthy subjects (paper III).

2. Subjects and methods

2.1 Experimental design

The study was performed in accordance with the ethical standards of the regional committee on human experimentation. The Regional Committee for Medical and Health Research Ethics of Western Norway approved the protocol, informed consent and advertisements (Reference # 2012/1084).

The study design was a randomized crossover design with two experimental periods. Cross-over design allocate each participant to a sequence of interventions. Each participant received either intervention A or B in the first period and the opposite in the succeeding period. The order in which A and B were given to each participant was randomized. Approximately half of the participant received the intervention in the sequence AB and the other half in the sequence BA. This is so that residual effect from first period to second period can be eliminated in the estimate of group differences in response. Cross-over designs have a number of possible advantages over parallel group trials. Every participant receives every intervention, which in this study means that the participant received both a lean-seafood and a nonseafood diet. Therefore, the design allows each participant to serve as his/her own control, eliminating among-participant variation (Senn 2002). At the end of the first experimental period the participants return to their usual dietary habits. A period between interventions is known as a washout period as a means of reducing carryover. We had a five-week washout period because of Easter time in-between the two periods. Prior to each experimental period, the subjects followed a diet in accordance with the Norwegian dietary recommendations for 3 weeks (run-in period), with additional specifications to include a maximum of one fatty fish (salmon, trout, mackerel or herring) meal per week. The last week of the run-in periods and throughout the experimental periods the subjects were instructed to avoid alcohol, chocolate or candy, industrial baked cakes or cookies, fast food,

probiotics, and fish or fish oil supplements. The subjects were instructed to maintain their normal physical activity level during the run-in-, experimental- and washout-periods. Body weight (kg) was monitored every day for the first week, and every second day for the three last weeks in each experimental period. We aimed to maintain a stable body weight (± 2 kg) in each experimental period.

At the first and last day of each experimental period, the subjects ingested a defined test meal with fasting and postprandial blood sampling. Morning spot urine was collected. At the test day, the subjects were resting and were allowed to drink water only, during the 6 hours after ingestion of the test meal. The cross-over study design for this study is shown in **Figure 2.1**.

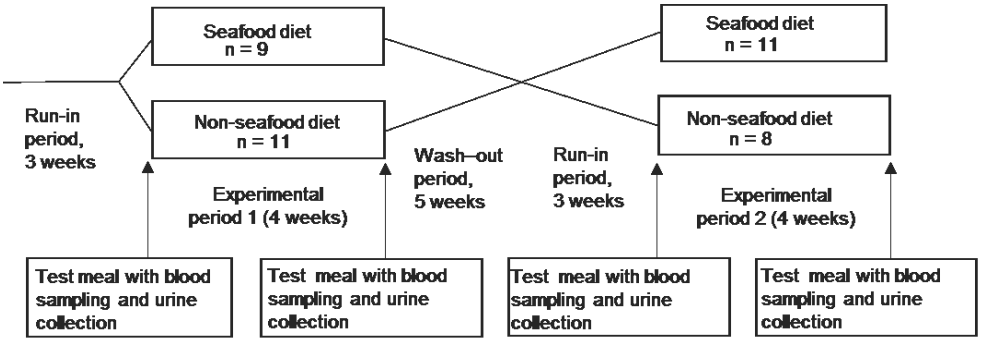

Figure 2.1 The cross-over study design.

2.2 Study participants

Healthy Caucasian study participants were recruited from the great area of Bergen through web page and newspaper advertisements (**Appendix I**) during October and November 2012. The PhD candidate conducted telephone-interviews with those who were interested (n = 148) by using a detailed screening questionnaire (**Appendix II**). The exclusion criteria were: not available in both study periods, use of tobacco; diabetes; use of medication known to affect glucose and lipid metabolisms, including hormone-based contraceptives; significant alternation in body mass (± 10 %) within the last six months; chronic, metabolic or acute disease or major surgery within the last

three months; hysterectomy, cholecystectomy, abnormal bleeding during the last 6 months, and dietary incompatibility with calcium supplementation and/or seafood consumption (allergy, intolerance, dislike). The inclusion criteria were healthy Caucasian aged between 18 and 65 years old. Those who met the inclusion criteria were invited to a pre-study visit (n = 41), were a consultation with physician were conducted and fasting blood samples were collected.

Also, each subject completed a medical and food history questionnaire (**Appendix III**) and a validated food frequency questionnaire (FFQ) (Andersen *et al*. 2003) (**Appendix IV**). Written informed consent was obtained from all the subjects after they had received oral and written information about the study. Based on consultations with a physician, 6 subjects were not meeting the inclusion criteria, and additional 5 subjects declined to participate. Thirty healthy subjects were invited to participate in the study, 27 subjects accepted to start. Half of the group (6 men and 8 women) was randomly assigned to begin with the lean-seafood diet and the other group (4 men and 9 women) to the nonseafood diet. During the first experimental period 6 subjects withdrew; 5 for personal reasons and 1 because of an accident. One subject withdrew after period one for personal reason. Twenty subjects completed period one (7 men and 13 women) and 19 subjects (7 men and 12 women) completed the total study (**Figure 2.2**). Average age was for the men 49.7 ± 7.0 (n = 7), for the women 51.0 ± 3.9 (n = 13) and for all 50.6 ± 3.4 (n = 20). The numbers of subjects that received the lean-seafood and the nonseafood diets in period 1 and 2 are outlined in **Figure 2.1**.

The participant`s physical and clinical characteristics from the pre-study visit are shown in **Table 2.1**.

Table 2.1 The subjects' physical and clinical characteristics[1].

	Men	Women	All
N	n=7	n=13	n=20
Age (years)	49.7 ± 7.0	51.0 ± 3.9	50.6 ± 3.4
Anthropometric measurements			
Body mass (kg)	86.2 ± 3.2	70.0 ± 2.2	75.7 ± 2.5
BMI (kg/m^2)	26.4 ± 1.1	25.2 ± 0.9	25.6 ± 0.7
Waist circumference (cm)	95.2 ± 4.2	83.3 ± 2.3	87.5 ± 2.4
Hip circumference (cm)	98.9 ± 2.0	101.7 ± 1.2	100.8 ± 1.1
Blood pressure and heart rates			
Systolic blood pressure (mmHg)	130.7 ± 3.5	125.5 ± 2.6	127.3 ± 2.1
Diastolic blood pressure (mmHg)	78.6 ± 3.2	75.6 ± 2.7	76.6 ± 2.1
Heart rate (number/min)	63 ± 3	65 ± 1	64 ± 1
Lipid parameters			
Total cholesterol (mmol/L)	5.0 ± 0.4	5.5 ± 0.3	5.3 ± 0.2
LDL -cholesterol (mmol/L)	3.5 ± 0.3	3.6 ± 0.3	3.6 ± 0.2
HDL –cholesterol (mmol/L)	1.3 ± 0.1	1.9 ± 0.1	1.7 ± 0.1
Total triacylglycerol (mmol/L)	1.1 ± 0.2	0.9 ± 0.1	1.0 ± 0.1
Glucose metabolism			
Glucose (mmol/l)	5.3 ± 0.2	5.1 ± 0.1	5.1 ± 0.1
Insulin (pmol/L)	49 ± 11	35 ± 3	41 ± 4
HbA1C (%)	5.5 ± 0.1	5.4 ± 0.1	5.4 ± 0.1
Kidney function			
Creatinine (umol/L)	79 ± 3	67 ± 2	71 ± 2
Liver function			
Alanine aminotransferase (U/L)	36.0 ± 6.2	22.6 ± 1.2	27.3± 2.6
Albumin (g/L)	47.6 ± 1.2	46.9 ± 0.7	47.1 ± 0.6
Gamma-glutamyltransferase (U/L)	36 ± 9	18 ± 4	25 ± 4
Total bilirubin (umol/L)	8.4 ± 0.6	7.9 ± 0.8	8.1 ± 0.6
Alkaline phosphatase (U/L)	63 ± 2	65 ± 4	65 ± 3
C-reactive protein (mg/L)	2.8 ± 0.4	1.5 ± 0.2	2.3 ± 0.2
Hematology			
Iron (umol/L)	16.3 ± 1.5	16.4 ± 1.0	16.3 ± 0.8
Ferritin (ug/L)	155 ± 38	110 ± 23	126 ± 20
Hemoglobin (g/dl)	15.0 ± 0.3	14.3 ± 0.3	14.6 ± 0.2
Erytrocytes (10^{12}/L)	4.9 ± 0.1	4.6 ± 0.1	4.7 ± 0.1
Hematocrit (%)	44.6 ± 1.2	42.5 ± 0.7	43.2 ± 0.7
Leukocytes (10^9/L)	5.8 ± 0.6	5.2 ± 0.4	5.4 ± 0.3
Thrombocytes (10^9/L)	218 ± 15	270 ± 13	252 ± 11
Thyroid function			
TSH (mlU/L)	3.0 ± 0.7	2.2 ± 0.3	2.5 ± 0.3

[1] All values are means ± SEM

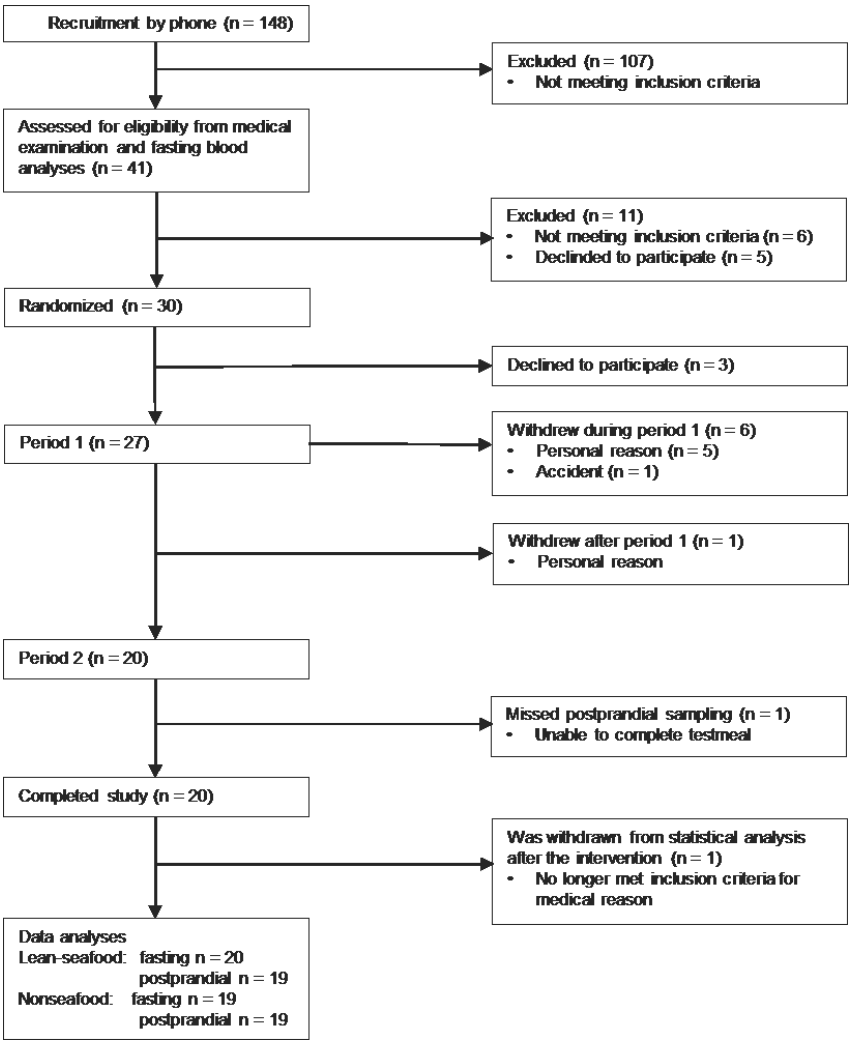

Figure 2.2 Consolidated flow chart for recruitment, randomization and data sampling from the participants of the study.

2.3 Dietary assessment

At the pre-visit day all study participants answered a FFQ and a medical and food history questionnaire. The medical and food history questionnaire (**Appendix III**) was designed to capture the excluding criteria like change in body mass the last 6 month, allergies, food intolerances, use of tobacco, drugs, alcohol, prescriptive medications, dietary supplements and/or hormone-based contraceptives and also questions about their usual average frequency of fish consumption. Questions regarding seafood intake included two summary questions concerning consumption of seafood as dinner and as spread. Some of the questions in the medical and food history questionnaire were repeated from the oral screening questionnaire by phone (**Appendix II**). The optical mark readable FFQ (**Appendix IV**) have been developed and validated at the Institute of Basic Medical Sciences, Department of Nutrition, at the University of Oslo (UoO) and are designed to capture the habitual food intake among adults (Andersen *et al*. 2003). The questionnaire contained approximately 180 food items, the options on the frequency of consumption of particular food types varied from several times a day to once a month, with portion-size choices based on typical household units: slices, glasses, cups, pieces, spoons and ladle. Questions about the use of dietary supplements, such as cod liver oil, fish oil capsules and some vitamins/minerals were included in the FFQ. Intake of nutrients and energy was calculated using a food database in the software systems (KBS - `kostberegningssystem`) at the UoO. Since under- and over reporting is a prevalent error in dietary self-reports (Kroke *et al.* 1999; Subar *et al.* 2003; Scagliusi *et al.* 2008), the energy intakes estimated from the FFQ was compared with calculated (Harris and Benedict equations) and recommended (Nordic reference) energy intakes before the energy level for each subject was chosen (**Table 2.2**).

The Harris and Benedict equations provide a method of calculating the energy a person expends at rest based on inputs such as their height, age and weight. This value can then be multiplied by a correction factor based on the person's activity level creating an estimate for actual energy expenditure (Harris & Benedict 1918). Also the Nordic energy requirement references are based on body weight, height, age and either a low, average or high physical activity level (The Nordic Council of Ministers 2004). The

subjects began the study at the energy level closest to their habitual intake, as chosen from comparing the results from International Harris-Benedict equation, the Nordic energy requirement references and the self-reportet FFQ (**Tabel 2.2**). Based on the subjects habitual intake six energy levels were established for this study: 7500, 8300, 9600, 10900, 12200 and 13500 kJ/day.

Table 2.2 Weight, height, BMI, physical activity level, the international Harris-Benedict equation, the Nordic energy requirement references, the self-reported FFQ and the level of chosen kJ in the study for each subject.

ID	Weight kg	Height cm	BMI kg/m^2	Exercise level	Harris-Benedict kJ calculated	Nordic reference kJ requirement	FFQ kJ self-reported	Level of kJ chosen
1	72.6	164.0	27.0	1.7	11086.41	10700	14129	10900
2	52.0	169.0	18.2	1.5	8460.54	8300	7443	8300
3	65.1	169.5	22.7	1.5	8659.83	8100	9164	8300
4	69.8	170.5	23.2	1.6	9461.88	9200	13390	8300
5	64.5	173.0	21.6	1.5	8584.36	8100	9712	8300
6	76.9	165.0	28.2	1.6	9613.40	9200	9813	9600
7	71.4	166.5	25.8	1.5	8668.19	8100	10038	8300
8	75.8	172.0	25.6	1.6	9603.52	9200	4120	8300
9	74.1	169.0	25.9	1.5	8846.32	8100	16260	8300
10	65.6	163.0	24.7	1.5	8127.67	7400	7930	7500
11	72.4	162.0	27.6	1.5	8502.86	7400	9566	7500
12	65.5	165.0	23.3	1.6	8588.85	8500	9193	7500
13	84.0	162.0	29.4	1.5	9067.35	7400	9505	9600
14	77.0	185.0	22.5	1.6	12604.56	12300	10648	12200
15	92.7	188.0	26.2	1.5	13084.67	10700	13469	13500
16	83.4	182.0	25.2	1.7	13079.88	13300	25915	13500
17	99.3	180.0	30.6	1.5	12116.01	9300	12226	12200
18	85.3	182.0	25.8	1.6	10999.13	10600	11807	10900
19	90.2	174.5	29.6	1.5	11113.97	9300	7949	10900
20	74.0	175.0	24.2	1.6	12022.18	10600	21059	13500

2.4 Developing diets

2.4.1 The experimental diets

The experimental diets were given as 7-day rotating menus and were formulated to meet the Norwegian nutrition recommendations, rich in dietary fiber, vegetables, unsaturated fatty acids and limited in added sugar and salt (The Norwegian Directorate of Health 2011). We designed two balanced diets that varied in the main protein sources. The lean-seafood diet consisted of lunch- and dinner meals with cod, pollack, saithe and scallops and the nonseafood diet contained skinless chicken filets, lean beef, skinless turkey filets, pork, egg and small amounts of dairy products.

To calculate the diets we started with the middle energy level, 10900 kJ. The energy distribution from the macronutrients were 19 % protein, 29 % fat and 52 % carbohydrates of the total energy. The energy content from protein, fat and carbohydrates in mixed diet were calculated using respectively conversion factor 16.7, 37.4 and 16.7 kJ per gram (The Nordic Council of Ministers 2004). Nineteen % protein of the total energy level 10900 kJ per day are 2071 kJ protein per day, divided with conversion factor 16.7 kJ per gram, gave 124.0 gram protein per day. The protein contribution from the experimental protein sources in both diets corresponded to 60 % of total protein intake (74.4 gram), and the remaining dietary proteins came from vegetable and cereal sources (49.6 gram). The composition of the experimental diets were calculated using the Norwegian Nutrition File database (Norwegian Food Safety Authority, Mat på data, version 5.1, 2009 linked to Norwegian Food Database 2006). A detailed example of a one-day menu from the lean-seafood intervention at the energy level of 10900 kJ/day is shown in **Table 2.3** and all the other menus from both lean-seafood and nonseafood are given in **Appendix V**. All food were precisely measured and weighed to the nearest 0.1 g (**Figure 2.3**). The subjects consumed their breakfasts, evening meal and snacks at home, dinners were prepared and served at the University College of Bergen, and prepared lunches were provided for the day after. Weekend lunches and dinners were distributed on Fridays. The subjects were instructed not to consume any food besides the experimental diets.

The experimental diets were balanced with equivalent amounts of dietary fiber, carbohydrates, protein, lipids, monounsaturated (MUFA), polyunsaturated (PUFA), saturated fatty acids (SFA) and content of marine n-3 fatty acids. To balance for the marine n-3 fatty acids, 7-day lean-seafood and nonseafood menus including breakfasts, lunches, dinners, evening meal and snacks were homogenized, freeze dried, powdered and the fatty acid composition were analysed. To balance for endogenous marine n-3 fatty acids present in the lean-seafood diets, cod liver oil (Möller`s Cod Liver Oil) was added (blinded to the participants) to all dinners of the nonseafood diets prior to serving. On average, 3.3 gram cod liver oil was added in all dinners at the 10900 kJ nonseafood diet. After balancing, the mean EPA+ DHA was 0.82 g per day in both diets. The vitamin D level of the lean-seafood diet was lower than the Nordic recommendations (The Nordic Council of Ministers 2004), and daily vitamin D_3 (10 µg or 400 IU) supplement was therefore given to the subjects during the lean-seafood intervention. Participants did not drink milk during any of the intervention, and only small amounts of dairy products were included in the nonseafood diet. Therefore, the subjects were given daily calcium supplement; 750 mg during the lean-seafood intervention and 500 mg during the nonseafood intervention to meet the Nordic recommendations for calcium intake. The nutrient composition of the 7-day menus for the 10900 kJ/day lean-seafood and nonseafood diets are outlined in **Table 2.4**.

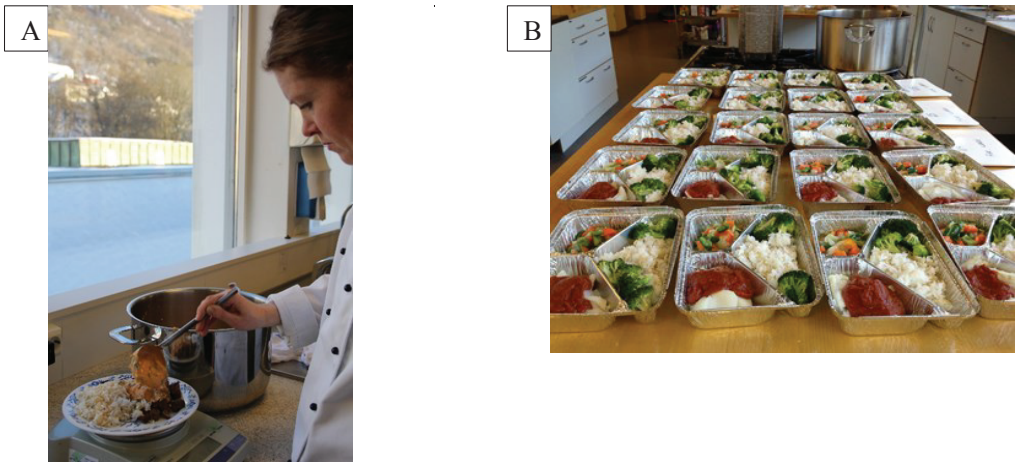

Figure 2.3 All food were precisely measured and weighed on scale Metos MII-600 (A). Prepared Saturday lunches containing cod in sweet & soursauce with broccoli and rice (B). Photos: NIFES

Table 2.3 Food ingredients and nutrition composition of Monday menu of the 10 900 kJ/d lean-seafood diet

		Energy kJ	Protein g	Fat g	SFA g	MUFA g	PUFA g	Chol* mg	Carbo* g	Total fiber g	Ca mg	Vit D µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	12 g Peanut butter	317	2.7	6.4	1.4	2.6	2.2	0	1.5	0.8	4	0
	40 g Jam	305	0.3	0.2	0	0	0.1	0	16.6	1.2	10	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Lunch	100 g Stockfish	680	37.8	1	0	0	0.4	152	0	0	80	2.4
	100 g Spinach	89	2.6	0.6	0.1	0	0.3	0	0.4	1.9	130	0
	80 g Lettuce	35	1	0.1	0	0	0.1	0	0.6	0.8	38	0
	50 g Onion	67	0.6	0.1	0	0	0	0	2.9	1	11	0
	20 g Tomato puree	66	0.9	0	0	0	0	0	2.6	0.7	5	0
	20 g Olive oil	736	0	19.9	2.8	14.7	1.5	0	0	0	0	0
	34 g Parsley root	63	0.6	0.1	0	0	0.1	0	2.3	1.3	20	0
	120 g Potato	368	2.3	0.1	0	0	0.1	0	18.2	1.9	7	0
Dinner	180 g Saithe	670	37.1	0.7	0.2	0.2	0.2	110	0.7	0	18	1.6
	50 g Onion	67	0.6	0.1	0	0	0	0	2.9	1	11	0
	4 g Garlic	18	0.3	0	0	0	0	0	0.7	0.1	1	0
	10 g Sesame oil	370	0	10	1.5	3.8	4.3	0	0	0	1	0
	120 g Coconut milk	1033	2.4	25.6	22.7	1.1	0.2	0	1.8	1.8	22	0
	6 g Lime	2	0	0	0	0	0	0	0	0	1	0
	70 g Carrot	106	0.5	0.1	0	0	0.1	0	4.7	1.9	20	0
	80 g Celery root	91	1.3	0.2	0.1	0	0.2	0	2.2	2.8	38	0
	90 g Whole-wheat bread	934	6.6	3.8	0.4	1.8	1.3	0	38	4.4	15	0
Evening meal	100 g Cereals	1560	10	9.5	2.2	4.1	3.1	0	56.9	8.9	43	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Snacks	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	32 g Oat biscuits	614	2.5	6.1	2.2	2.8	0.8	1	19.5	1.8	11	0
	Total	10868	125	87.1	33.9	31.5	16	263	303	46.1	574	4
	% energy		19.2	30.2					46.6	3.4		
	MUFA:SFA					0.93						
	PUFA:SFA						0.47					

* Cholesterol
* Carbohydrate

Table 2.4 Nutritional composition of 7 day menu without supplements of the 10900 kJ/day lean-seafood and nonseafood[1] diet[2]

Lean-seafood	Energy	Prot	Carbohydrate[3]	Fat	SFA	MUFA	PUFA	M:S	P:S	Cholesterol	Fiber	Ca	Vit D
		% E	% E	% E	g	g	g			mg	g	mg	µg
Monday	10868	19.2	46.6	30.2	33.9	31.5	16.0	0.93	0.47	263	46.1	574	4.0
Tuesday	10885	18.6	49.6	27.7	36.5	26.4	10.5	0.72	0.29	271	46.8	329	6.5
Wednesday	11097	19.7	50.0	26.4	24.5	31.5	16.6	1.29	0.68	395	42.8	413	9.7
Thursday	10091	19.5	47.3	29.0	21.4	36.9	13.6	1.72	0.63	312	43.6	421	5.8
Friday	11863	18.6	49.1	28.2	26.0	40.9	14.8	1.57	0.57	264	49.0	433	7.1
Saturday	10453	19.0	47.8	29.7	15.8	36.3	25.2	2.30	1.60	245	37.5	310	8.0
Sunday	10914	18.9	48.2	29.4	19.1	42.3	18.5	2.32	1.61	268	37.0	336	7.3
Mean	10881.6	19.1	48.4	28.7	25.3	35.1	16.5	1.5	0.8	288	43.3	402	6.9
SEM	208.1	0.2	0.5	0.5	2.9	2.1	1.7	0.2	0.2	19,4	1.7	34.2	0.7

Nonseafood	Energy	Prot	Carbohydrate[4]	Fat	SFA	MUFA	PUFA	M:S	P:S	Cholesterol	Fiber	Ca	Vit D
		% E	% E	% E	g	g	g			mg	g	mg	µg
Monday	11108	18.9	47.3	30.4	23.8	38.5	18.9	1.62	0.79	1097[2]	38.3	705	15.9
Tuesday	10971	19.1	48.4	28.4	24.2	29.2	21.8	1.21	0.90	221	49.3	603	9.6
Wednesday	10908	21.4	49.3	25.0	25.8	22.8	18.8	0.88	0.73	288	46.7	875	9.6
Thursday	9996	19.2	47.4	29.6	25.7	35.4	10.7	1.38	0.42	255	37.9	623	6.9
Friday	11100	18.5	49.2	28.4	21.6	41.6	13.3	1.92	0.62	225	46.4	686	7.7
Saturday	10762	19.5	47.6	29.3	26.3	37.0	14.9	1.41	0.57	297	38.4	537	8.7
Sunday	11451	18.9	45.6	31.8	29.3	42.1	19.1	1.44	0.65	374	42.6	863	9.1
Mean	10899.9	19.4	47.8	29.0	25.2	35.2	16.8	1.4	0.7	394	42.8	699	9.6
SEM	171.0	0.4	0.5	0.8	0.9	2.6	1.5	0.1	0.1	118.8	1.8	48.6	1.1

[1] Including cod liver oil
[2] Without vitamin D and calcium supplements
[3] Carbohydrates (% of energy) are without fiber
[4] Due to one omelette (egg) serving. Mean cholesterol value in the 7 d nonseafood menu without omelette, 273 ± 20 mg

2.4.2 The test meals

At day 0 and day 28 identical test meals were served to the subjects. The test meal consisted of cod fillet or lean beef, served with pasta, sauce, vegetables and a cinnamon bun, and differed only in the source of experimental protein (**Table 2.5**). Men were given a test meal of 3000kJ and women a test meal of 2250kJ, both consisting of 20% energy from proteins, 28% energy from fat and 52% energy from carbohydrates.

Table 2.5 Ingredients in the test meal with cod and beef as protein source for men, 3000 kJ.

Test meal with cod	Test meal with beef
87 g Cod	75 g beef
30 g Cauliflower	30 g Cauliflower
124 g Potatoes	124 g Potatoes
14 g Butter	12 g Butter
4 g Rapeseed oil	2 g Rapeseed oil
95 g Canned tomatoes	95 g Canned tomatoes
22 g Onion	22 g Onion
60 g Carrot	60 g Carrot
40 g Broccoli	40 g Broccoli
100 g Pasta, whole grain	100 g Pasta, whole grain
50 g Cinnamon bun	50 g Cinnamon bun
	0.9 g Cod liver oil

2.5 Sample collection and analyses

A detailed description of the sample collection and analyses are given in the papers.

2.5.1 Blood sampling and analyses

In brief, at the first and last day of each experimental period blood samples were drawn from the antecubital vein after an overnight fast. After the subjects had ingested the test meal (15 minutes) postprandial blood samples were taken after 0, 30, 60, 120, 240 and 360 minutes. Serum (after 30 min clotting) and EDTA plasma were separated by centrifugation at 2500 g for 5 min at 4 °C and aliquoted to pre-marked tubes. All blood samples were thereafter frozen at – 80 °C, except for the plasma used for chylomicrons separation that were ultra-centrifuged before stored at – 80 degree freezer (**Figure 2.4**).

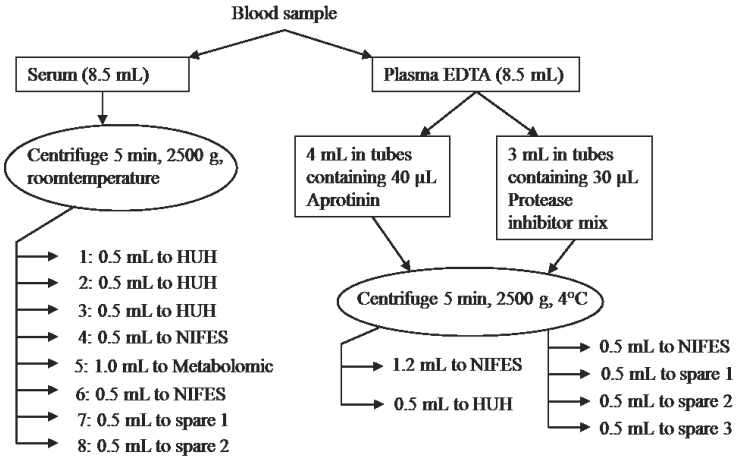

Figure 2.4 Overview of blood sampling. Each blood sample was divided into two vacutainers in prioritized order, serum and EDTA plasma. Serum was allowed to cloth in room-temperature for 30 minute before being centrifuged, while plasma was immediately split into two containers and thereafter centrifuged separately. Serum and plasma was aliquoted into dedicated tubes in prioritized order, and stored at -80 degree freezer until transported to different laboratory. HUH = Haukeland University Hospital, Department of Laboratory Medicine and Pathology.

Serum and plasma samples were analysed for standard clinical chemistry parameters and hormones involved in the regulation of lipid- and glucose metabolism. Overview of analyses on serum and plasma, and at which laboratory they were performed are outlined in **Table 2.6**.

Table 2.6 Analyses performed on fasting and postprandial blood samples.

Analyses	Matrix	Laboratory
Lipid metabolism		
TAG	s	HUH
Cholesterol, total	s	HUH
HDL-cholesterol	s	HUH
LDL-cholesterol	s	HUH
Apolipoprotein B	s	HUH
Apolipoprotein A1	s	HUH
TAG in CM-fraction	ep	HUH
TAG in non-CM fraction	ep	HUH
Lipoproteins by NMR	ep	LipoScience[1]
Glucose metabolism		
Glucose	s	HUH
Total bile acids	s	HUH
NEFA	s	NIFES
OH-butyrate	s	NIFES
Lactate	s	NIFES
Glycerol	s	NIFES
Urea	s	NIFES
Insulin	s	HUH
C-peptide	s	HUH
Glucagon	ep	HUH
Adiponectine	s	NIFES
Ferritin	s	HUH
Transferrin reseptor	s	HUH
C-reactive protein	s	HUH
Metabolomics		
Metabolomics by NMR	s	University of Aarhus

[1] = LipoScience (Raleigh, NC, USA)
s = serum
ep = EDTA plasma
HUH= Haukeland University Hospital, Department of Laboratory Medicine and Pathology

EDTA plasma was ultra-centrifuged at 41 000 g for 35 min at 4 °C at NIFES on the blood sampling day. The top and lower fraction were collected and stored at -80 °C freezer before analysed for respectively TAG in chylomicrons and TAG in non-chylomicrons (containing very low-density lipoproteins, chylomicron remnants, low-density lipoproteins and high-density lipoproteins). A comprehensive lipoprotein profile was determined by NMR and particle number of total VLDL (VLDL-P, nmol/L), LDL (LDL-P, nmol/L) and HDL (HDL-P, µmol/L), along with large, medium and small subclasses and a weighted average particle diameter for each. These analyses were bought from LipoScience (Raleigly NC, USA).

2.5.2 Urine sampling

Morning spot urine was collected on day 1 and 28 of each experimental period. The urine was aliquoted, frozen at – 80 °C before analysed, using NMR spectroscopy and LC-MS platforms. These analyses were performed by a PhD student at University of Aarhus.

2.5.3 Dual-energy x-ray absorptiometry (DXA)

Dual-energy x-ray absorptiometry (DXA) was used to measure body composition; total fat mass, fat % and total lean mass. All scanning procedures were conducted by trained medical staff following the same protocol.

2.6 Statistical analyses

The number of research participants are based on a Canadian study, in which similar interventions previously were performed (Ouellet *et al.* 2007; Ouellet *et al.* 2008). Sample size has been calculated based on the efficacy of lean fish to reduce plasma VLDL-TAG (Gascon *et al.* 1996) and on the procedure described by Wellek and Blettner (2012) for crossover design in clinical trials. A minimum of 16 subjects was needed to detect a treatment difference of ~25-30% in fasting plasma TAG at a probability level inferior to 0.05 and a power level corresponding to 80%. A similar calculation has been performed based on postprandial TAG response to dietary protein (Mortensen *et al.* 2009). A minimum of 12 subjects was needed to detect a treatment protein effect of ~30% at a probability level inferior to 0.05 and a power level corresponding to 80%. Therefore, from those calculations and in order to be conservative, we chose the power calculation based on fasting VLDL-TAG.

We increased our number of subjects to 30 per experiment by taking into account an eventual 25% loss of subjects. We therefore invited 30 subjects to participate in the study, 27 subjects started, 20 completed period 1 and we obtained postprandial samples from 19 (**Figure 2.2**).

Statistical analyses were performed using the Statistical Analysis System (version 9.3; SAS Institute, Cary, NC). The PROC MIXED procedure for an ANOVA for crossover design with two periods as described by Hills and Armitage (1979) was used to compare the effects of the two dietary treatments on anthropometric measurements, lipid and glucose parameters. As no effect of experimental period or diet sequence and no residual effect of the first experimental period over the second period was observed for any of the measured variables, the data for experimental period, diet sequence, and dietary treatment were pooled. Standard Bonferroni correction has been performed to reduce the chances of obtaining false-positive results. Furthermore, repeated-measures analysis of variance were applied for variables with repeated measures over time during the test meal. The Least Squares Means Test was performed to compare the changes (post to pre) values for each diet. For all measures, $P < 0.05$ was considered as statistically significant. Data are expressed as means ± SEM.

3. Summary of results

3.1 Paper I

Lean-seafood intake reduces cardiovascular lipid risk factors in healthy subjects

Objective: The primary aim of this study was to elucidate the potential of two main dietary protein sources, lean-seafood or nonseafood, to modulate fasting and postprandial lipids in healthy subjects. We hypothesized that lean-seafood intake would reduce cardiovascular lipid risk factors in healthy subjects, as compared to intake of nonseafood protein sources.

Results:

- Lean-seafood, as compare to nonseafood intake, reduced fasting ($P = 0.03$) and postprandial ($P = 0.01$) serum TAG concentrations.
- Lean-seafood intake reduced postprandial ($P = 0.02$) medium-sized VLDL particle concentrations.
- Lean-seafood intake prevented elevation in fasted ($P = 0.03$) and postprandial ($P = 0.01$) total- to HDL cholesterol ratio.

Conclusions: Dietary protein source determines fasting and postprandial lipids in healthy subjects in a manner that may have beneficial effect on long-term development of cardiovascular disease.

3.3 Paper II

Lean-seafood intake reduces postprandial C-peptide and lactate concentrations in healthy subjects

Objective: Our secondary aim was to elucidate the potential of two main dietary protein sources, lean-seafood or nonseafood, to modulate fasting and postprandial glucose metabolism in healthy subjects. We hypothesized that lean-seafood intake would affect postprandial glucose metabolism differently from nonseafood in healthy subjects.

Results:

- The dietary intervention did not cause significant changes in fasting and postprandial serum insulin and glucose concentrations.
- Lean-seafood intake reduced postprandial C-peptide ($P = 0.04$) concentrations.
- Lean-seafood intake reduced postprandial lactate ($P = 0.04$) concentrations.
- Lean-seafood intake reduced fasting ($P = 0.002$) and postprandial ($P = 0.002$) TAG/ HDL-cholesterol ratio.

Conclusion: Dietary protein source determines postprandial glucose metabolism in healthy subjects in a manner that may have impact on long-term development of insulin-resistance, type 2 diabetes and cardiovascular disease.

3.4 Paper III

Lean-seafood intake improves mitochondrial oxidative capacity as indicated by human urinary metabolomics

Objective: We hypothesized that intake of different protein sources would alter key metabolic pathways, and that these modified effects were reflected in the urine metabolome. The profiling was conducted using NMR spectroscopy and LC-MS analysis, in order to improve understanding of the effects of dietary changes on metabolic status.

Results:

- Nonseafood intake increased the urinary level of 3-methylhistidine (3MH) ($P = 0.002$).
- Lean-seafood intake reduced the urinary level of N1-methyl-2/4-pyridone-5/3 carboxamide (2PY) ($P < 0.01$).
- Lean-seafood intake reduced the urinary levels of L-carnitine ($P < 0.01$) and an acylcarnitine ($P < 0.03$).
- Lean-seafood intake increased the urinary level of trimethylamine N-oxide (TMAO) ($P < 0.01$).
- The content of TMAO was higher in the lean-seafood diet ($P \leq 0.001$).

Conclusion:

Based on the urinary metabolomics analyses, our data suggest that in healthy subjects four weeks of lean-seafood intervention improved mitochondrial oxidative capacity that may have facilitated lipid catabolism. In contrast, four weeks on nonseafood intervention may have increased protein catabolism.

4. Discussion

4.1 Methodological considerations

4.1.1 The study design

In a randomized controlled trial (RCT) the subjects are randomly assigned to treatment groups. Both groups will be treated identically in all respects except for the intervention being tested. RCTs are the most powerful tools in clinical research and are considered to be the "gold standard" for generating reliable evidence (Moher *et al*. 2012). The randomization avoids systematic errors, and thus confounding effects are statistically less likely to occur (Higgins & Green 2011). The subjects in the present study were randomly assigned to start with one of the two dietary interventions. No biochemical or anthropometric variables were used for randomization purpose. Randomization was done by putting 30 pieces of papers, each piece with one of the participants ID number, and in an alternating order assigning the subjects to start with the seafood or nonseafood diet by picking the paper pieces sequentially from the box. Randomization does not necessarily result in equal groups with respect to age, gender or other characteristics, but in a cross-over design each subject is receiving both interventions. Since each subject is receiving all treatments, also variability is reduced, because the measured effects of the interventions are the difference in an individual subject's response to the intervention, in the present study being lean-seafood and nonseafood interventions. This reduction in variability makes it possible to use smaller sample sizes, while retaining the ability to detect specific differences in response (Friedman *et al.* 2010). Since dropout is common in clinical trials, this was taken into account in the power calculation. To account for an expected dropout in such an intervention, we recruited more subjects than necessary to maintain the statistical power at the end of the study. A reduction in variability was also achieved by including only Caucasian subjects, leading to a more homogenous group and probably resulting in lower variations of the data, and more accurate results (Moher *et al*. 2012).

Ideally, neither the subjects nor the investigator should know who is in which group to avoid any influence of pre-conceived ideas among the participants or the investigators. In the present study the sequence of dietary intervention was concealed until one week before study start. Thereafter, blinding was not feasible due to the nature of the intervention. Blinding was re-established during sample analysis and statistical data analysis, since un-blinded laboratory and data analysis may introduce bias through the choice of analytical approach (Gluud, 2006). In this study the technicians performing the analyses were blinded by giving the samples unique number codes.

There are several challenges related to conduction of crossover studies, e.g. the risk of carryover effect and avoiding drop-outs. To avoid that the effect of the intervention during the first period must not carry over into the second period a washout period of 5 weeks was included in the design. Also a 3 week run-in period was completed in advance of each intervention period to minimize order effects (Higgins & Green 2011). During this period, participants were instructed to avoid certain foods e.g. chocolate or candy, industrial baked cakes and fast foods. Another challenge with crossover studies is the risk of drop-outs due to their longer duration. Since the study was demanding to the subjects and the scientists, it is of importance to minimize the experimental duration. We also choose the intervention periods to be 4 weeks, based on previous experience shown to be sufficient to obtain diet-induced changes in fasting lipid concentrations (Jacques *et al*. 1992; Gascon *et al.* 1996; Ouellet *et al.* 2007). To avoid drop-out all lunches and dinners were free and a lot of effort was put into the flavor development of the experimental diets. In addition to that the meals should look and taste good, the atmosphere in the serving room should be welcoming to the participants every day. The participants were given an economic compensation after every test day. This compensation was meant to cover travel expenses during the study. Throughout the study period we had daily talks with the study participants and were available if they had any questions. After completion of the study we arranged a gathering were the participants got an individual feedback on some of their results from the study (fasting concentrations of TAG, total cholesterol, LDL-C, HDL-C, glucose and insulin) and body composition (body weight, lean mass, fat mass, fat %).

Half of the study group started with lean-seafood diet, and the other half with the nonseafood diet. Use of crossover design has an advantage that fewer participants are required, to avoid between-participant variation when estimating the intervention effect. The ability to justify the use of crossover as a design still depends on a test for carryover that includes between-participant variability (Friedman *et al*. 2010). The present study had no effect of experimental period or diet sequence and no residual effect of the first experimental period over the second period for any of the measured variables. One exception was the body mass, as the participants lost more body weight in the second period compared to the first period. This period effect was not influenced by diet, and the weight reduction was equally in both interventions. A possible explanation for increased weight loss in the second period may be because the participants were more physically active due to the season in the second period (April to May). Hills & Armitage (1979) pointed out that also the scientific community should be convinced by the substantial evidence that the design has no carryover effect.

4.1.2 The Food Frequency Questionnaire

The Food Frequency Questionnaire is the most common dietary assessment method used in large epidemiologic studies of diet and health. The self-administered FFQ asked the subjects to report the frequency of consumption and portion size of food items over a defined period of time (e.g. the last month; the last six months). To answer an FFQ relies heavily on the subjects` ability to recall the foods he/she usually eats and to conceptualize portion sizes. The FFQ is therefore susceptible to measurement errors, as with the use of any dietary assessment instrument (Kipnis *et al.* 2002).

The validated FFQ we used had 180 food items included (Andersen *et al*. 2003) and may for some of the participants have felt burdensome and possibly led to careless completion and lower data quality. For that reason, we could have used a shorter FFQ that was easier for the participants to complete, if the purpose was only to assess the total energy intake (EI). However, important information might be missed and only the most commonly consumed foods would have been captured. Whether the participants

are male or female could impact the FFQ results. Young women are possibly more weight conscious than young men, and this could lead to higher underreporting of FFQ. In studies from USA (Subar *et al.* 2003), Brazil (Scagliusi *et al*. 2008) and Germany (Kroke *et al.* 1999) women showed to underestimate the EI when using an FFQ, as compared to diet recalls, diet histories and food records methods. In a Norwegian study comparing EI assessed with an FFQ, the underestimation among women was not statistically significant (Andersen *et al*. 2003). Underreporting varied largely from one study and country to another, but taken together the evidence points to no general difference according to gender (Subar *et al.* 2003; Scagliusi *et al*. 2006; Freedman *et al.* 2014). This is in agreement with our finding were 54 % of the women and 57 % of the men were either under- or over reporting their EI (**Table 2.1**). Regardless of gender, 40 % of the participants were over estimating, and only 15 % of the participants were under estimating their EI in the present study. The over and under estimation of intakes can result from a time consuming process of answering the FFQ, or that the participants answer the questionnaire in the fasting state. Individual factors might, however, make the participants to misreport to the same extent regardless of dietary assessment method. The FFQ method is anyway inaccurate for assessing the individuals intake, therefore we compared the participants self- reported intake with calculated (Harris & Benedict 1918) and recommended (The Nordic Council of Ministers 2004) energy intake. Thus, the energy intake level of each participant was validated using three different dietary assessment methods.

4.1.3 The experimental diets

The experimental diets were balanced with equivalent amounts of dietary fiber, carbohydrates, proteins, lipids, MUFA, PUFA, SFA and content of marine n-3 fatty acids. The participants were not eating nutrients but foods, and a challenge in developing experimental diets was to prepare appetizing dishes. Food that looks unappetizing are often not eaten. Foods that are discolored, in odd shapes, or otherwise atypical are usually regarded with suspicion. But less obvious is the fact that visual

cues can alter not just the acceptability of foods, but also modify the perception of taste, odor and flavor (Delwiche 2004). The literature indicate that increase in color level in a dish, also increases taste and/or flavor intensity, while other studies means that the effect is resulting from learned associations rather than from inherent psychophysical characteristics (Clydesdale 1993).

The color, smell, taste, temperature, texture and sound interact with each other, and they all have an impact on flavor ratings and have a tremendous impact on whether foods and drinks will be accepted or rejected, and liked or disliked. Of additional importance in developing healthy experimental diets were to use herbs, fresh seafood and meat, vegetable with different colors and vegetable with lots of flavor such as garlic, onion, parsley root and celeriac. The subjects` acceptance of the diets can be attributable to that they really liked it, or that their perception of the diets performance benefits, because the diets are "therapeutic" or "healthy" in itself. A "healthy diet" in a study will have a different set of expectations by the participants from an "ordinary diet" (Civille & Oftedal 2012).

Development of enriched food products is commonly used in industry, and often are the ingredient being enriched imparting a distinct flavor or texture. To balance for the endogenous marine n-3 fatty acids present in the lean-seafood diet, we added cod liver oil to all of the nonseafood dinners. During dietary intervention study like this, high compliance is crucial, and so reduction of off-flavors or bitter tastes becomes increasingly important. Masking the cod-liver oil is challenging, as the ingredient has distinctive visual characteristics and a particularly strong flavor and aftertaste. Through pilot testing among the kitchen personel, we found that dinner sauces in the nonseafood diet was the best delivery method to mask the cod-liver oil flavor. Since some of the subjects in the pilot testing, felt the cod-liver oil flavor because they knew it was added, we decided to blind the cod-liver oil for the participants in the study.

The nutritional composition of the experimental diets were equal, they only varied in the main protein sources. In addition we aimed to make the two diets look as similar as possible, similar in physical characteristics (gross morphology, appearance, volume

and texture) and sensory qualities (mouthfeel, taste, palatability and breakdown characteristics in the mouth). We managed this to a large extent by using the same side dishes, vegetable or the same ingredients in the sauce. Since different preparation and cooking method may have an impact on the results (Patel *et al.* 2012) we prepared the meals in the same convection oven, except for the red meat that was fried in a dry pan without frying media.

Another aspect to consider is site or location where the meals are served, the atmosphere, the temperature in the room, table, chairs, use of napkins, lighting and so on do all have an impact of the appealing to the servings (Nyberg & Grindland 2008). In our study we aimed to standardized these conditions.

4.2 General discussion

The main results of this thesis is that intake of lean-seafood positively influenced the risk factors for CVD and T2D. Prevention of CVD and T2D is a public health goal and comprises several avenues of action. Achieving a healthy lifestyle true particular increased physical activity, dietary patterns and nutrient intake can play a role in CVD and T2D prevention.

Healthy subjects had after 4 weeks lean-seafood intervention, a significant reduction in fasting and postprandial circulating TAG concentrations adjusted according to Bonferroni correction, relative to the 4 weeks nonseafood intervention (Paper I). There is evidence that raised circulating TAG levels are associated with increased coronary heart disease risk, adjusted for established coronary risk factors (Nordestgaard *et al.* 2007). In the same meta-analyses, using data from more than 300 000 participants in prospective studies, the hazard ratio for coronary heart disease, adjusted for age and sex only, was 1.10 per 16% higher TAG concentration (Sarwar *et al.* 2010). In our study, we observed that lean-seafood intervention reduced the fasting concentration of TAG with 16%, whereas intervention with nonseafood increased the fasting concentration of TAG with 13%. The postprandial TAG concentration was also lower

after the lean-seafood intervention, and the average difference in postprandial TAG concentration over the 6h time-course was about 0.3 mmol/L after ingestion of cod and beef test meals. A meta-regression analysis of pharmacological lowering of TAG with fibrates indicated that a 0.1 mmol/L decrease in triglycerides caused a 5 % reduction in coronary events (Jun *et al.* 2010). Based on the estimates from the Emerging Risk Factors Collaboration (Sarwar *et al.* 2010), the lean-seafood intervention in our study reduced the coronary heart disease hazard ratio by close to 20% relative to the nonseafood intervention. Our finding with dietary protein from lean-seafood, compared to protein from nonseafood, reduced circulating TAG concentration in healthy subjects at a magnitude that might be preventive against future CVD.

The present study showed a lipid-lowering effect after consumption of lean-seafood. Seafood is a rich source of taurine (2-aminoethanesulfonic acid) whereas terrestrial animal proteins generally are lower in taurine, and milk proteins and vegetable proteins are virtually deficient in taurine (Spitze *et al.* 2003). This corresponds to the higher level of taurine in the experimental diet with lean-seafood (0.3 weight% of total amino acids), compared to the experimental diet with nonseafood (0.1 weight% of total amino acids) (Paper I). Taurine supplementation has been reported to reduce blood TAG in overweight and obese non-diabetic humans (Zhang *et al.* 2004). Fish consumption significantly results in inverse relationship between urinary taurine excretion and coronary heart disease mortalities (WHO-CARDIAC Study) (Yamori *et al.* 2004). Taurine may be an important amino acid in fish to explain the beneficial effects of fish on the prevention of CVD. Other amino acids could also play a role. Conclusion based on the high level of taurine in the lean-seafood diet alone should not be drawn, but taurine is a conditionally essential nutrient.

The amount of total cholesterol concentration decreased significantly after both lean-seafood and nonseafood diets intervention. Also the LDL cholesterol concentration decreased by both interventions (Paper I). The reduced concentration of total cholesterol and LDL cholesterol may be explained by the healthy diet high in dietary fiber and the complete absence of alcohol, chocolate or other sweets. The healthy diets, in combination with the reduced body weight during the interventions, may have

contributed to the reduced total cholesterol and LDL cholesterol concentrations. Both diets also reduced concentration of the independent CVD risk factor HDL-cholesterol (Rader & Hovingh 2014) but the lean-seafood intervention attenuated this decrement. Thus the change in the strong cardiovascular risk predictor circulating total- to HDL-cholesterol ratio, was significant lower after the lean-seafood intervention (Paper I). The subjects had no change in apolipoprotein A1 or total HDL particle concentrations, suggesting that after the lean-seafood intervention the HDL particles contained more cholesterol per HDL-particle than after the nonseafood intervention. Our results after 4-week with nonseafood diet are consistent with previous studies, showing that increases in circulating TAG concentration are associated with greater reductions in HDL cholesterol than in ApoA1 (Tremblay *et al.* 2007; McQueen *et al.* 2008). A mechanism has been suggested in which core lipids are exchanged between HDL and VLDL particles, in combination with elevated hepatic lipase activity, producing cholesterol-poor HDL particles or reduced size (Rashid *et al.* 2003). In our study, the nonseafood intervention led to a lower ratio of HDL- to total cholesterol, and higher concentration of medium-sized VLDL particles, resembling the altered lipoprotein interaction previously suggested to take place during development of dyslipidemia (Rashid *et al.* 2003; Tremblay *et al.* 2007; McQueen *et al.* 2008). Our results suggest that 4 weeks of lean-seafood intervention protected healthy subjects from developing a metabolic dyslipidemia pattern with elevated postprandial concentrations of TAG and VLDL particles, and an elevated total-to-HDL- cholesterol ratio.

The lipoprotein profile obtained after lean-seafood intervention is contradictory to the diabetic dyslipidemia frequently preceding development of type 2 diabetes (Adiels *et al.* 2008), suggesting that insulin signaling could be involved in the diet-induced alterations of lipoprotein profiles observed in the present study.

Since insulin, but not C-peptide, is extracted by the liver, serum C-peptide level reflects endogenous insulin secretion more directly than does serum insulin level (Weir & Bonner-Weir 2004). In our study postprandial C-peptide concentration decreased after the lean-seafood intervention, resulting in a significant ($P = 0.04$) difference between the two diet interventions (Paper II). This indicated different insulin release from

pancreatic ß-cells. The increased insulin secretion after consumption of nonseafood, may be an indicator for early insulin resistance (Weir & Bonner-Weir 2004; Nolan *et al.* 2011). Despite the reduced postprandial C-peptide concentration after the lean-seafood intervention, the glucose concentration was maintained. Our results indicated improved insulin sensitivity after lean-seafood diet, which was also observed in insulin resistant subjects after 4 weeks on a cod-based diet compared with subjects ingesting a meat-based diet (Ouellet *et al.* 2007). Another test meal-study in healthy humans showed lowered insulin levels and reduced insulin-to-C-peptide and insulin-to-glucose ratios when given a cod protein meal, compared with a milk protein meal (von Post-Skagegard *et al.* 2006). These results indicates that insulin works better, or that other components amplifies the insulin signal after eating cod or lean-seafood. Our study was conducted in healthy subjects, with healthy balanced diets, and still resulted in a significant different postprandial C-peptide concentration. This shows that adjustment in diet, may have preventive effect on T2D development.

Lactate is a marker of glucose oxidation (**Figure 4.1**). In our study postprandial lactate concentration decreased after lean-seafood, and increased after the nonseafood intervention, despite equal postprandial glucose concentrations in the subjects (Paper II). An increased circulating lactate concentration has traditionally been used as an indicator of energy imbalance related to vigorous exercise and hypoxia (Sabatine *et al.* 2005). However, in our study lactate was not increased as a result of exercise since all the participant were resting during the test day. A possible explanation for the increased lactate levels could be an inadequate oxidative capacity at the cellular level. Indeed a study in subjects with impaired glucose tolerance or insulin sensitivity demonstrated increased plasma lactate production during a 75 g oral glucose tolerance test (Berhane *et al.* 2015). In our study we served our subjects a test meal, with carbohydrate levels of 64 grams for women and 85 grams men. Juraschek *et al.* (2013) have also observed a strong, graded relationship between plasma lactate and subsequent risk of incident type 2 diabetes over a 9-year follow-up period.

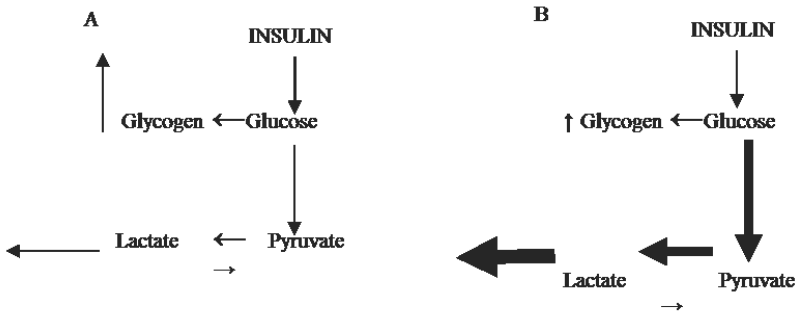

Figure 4.1 Lactate in glycolysis in healthy and type 2 diabetic subjects. Insulin stimulate glucose uptake into the insulin-responsive tissues. In healthy subjects ~1/3 of glucose is stored as glycogen, and rest goes true glycolysis where most is oxidized (A). The ability to synthesize and store glycogen after meal is impaired in subjects with impaired insulin-sensitivity and type 2 diabetes, and more of the glucose enters glycolysis. The oxidative capacity decreases, and plasma lactate concentration increases as a consequence of greater flux through glycolytic pathways (B).

Lactate metabolism is profoundly related to glucose metabolism. After meal ingestion, the blood glucose and insulin transiently increase. The increased level of circulating insulin stimulate uptake of glucose and storage to glycogen. Glucose is producing pyruvate true glycolysis. Pyruvate is either being converted to acetyl CoA that can enter the Krebs cycle or being converted to lactate leading to higher circulating lactate concentration (**Figure 4.1**). The relative increase in postprandial lactate concentration after the nonseafood intervention in the present study might indicate impaired insulin sensitivity (Juraschek *et al.* 2013; Berhane *et al.* 2015). Whether decreased oxidative capacity itself is a cause or consequence of insulin resistance and diabetes is unknown. Another potential explanation for the relationship between lactate and incident diabetes is expression and activity of GLUT4, the glucose transporter in muscle and fat cells. In response to insulin, GLUT4 sequesters glucose into muscle and fat cells where it might undergo glycolysis. There is evidence that elevation of plasma lactate suppressed glycolysis before its effect on insulin-stimulated glucose uptake, consistent with the hypothesis that suppression of glucose metabolism could precede and cause insulin resistance. Lactate-induced insulin resistance was associated with impaired insulin signaling and decreased insulin-stimulated glucose transport in skeletal muscle (Choi *et al.* 2002). From a study with rats fed a high-fat diet, the cod protein feeding,

compared to soy protein and casein feeding, prevented rats from developing skeletal muscle insulin-resistance (Lavigne *et al.* 2001). Our finding that lean-seafood, relative to nonseafood intervention reduced postprandial lactate, is another indication that lean-seafood intake may preserve normal glucose metabolism in healthy subjects.

Intake of lean-seafood in 4 weeks as the main protein source resulted in significant higher level of TMAO in urine. This finding can be explained by the high level of endogenously TMAO from marine fish in the lean-seafood diet (Paper III). The content of TMAO in marine fish is between 40 – 120 mg/kg, compared to fresh-water fish which contain only 0 – 5 mg/kg (Belitz *et al.* 2009). This corresponds to the significant higher level of TMAO in the experimental diet with lean-seafood, compared to the experimental diet with nonseafood (Paper III). TMAO may also be derived from hepatic oxidation of trimethylamine (TMA), generated by gut flora from dietary phosphatidylcholine, choline and carnitine (Tang *et al.* 2013). Hence, TMAO may end in the human urine either from dietary sources, but may also be produced through the gut flora. TMAO produced through the gut flora pathway has been associated with the development of atherosclerosis (Koeth *et al.* 2013) and cardiovascular disease (Tang & Hazen, 2014). Since especially marine fish is a major source of TMAO, a higher rate of CVD among fish-consuming people would be expected. In contrast, a low rate of CVD have been reported with regular fish consumption in a large number of prospective studies (Dyerberg *et al.* 1978; Kromhout *et al.* 1985; Leon *et al.* 2008; Mozaffarian & Wu 2011). The present study showed a relatively clear anti-atherogenic lipoprotein profile after consumption of lean-seafood. Conclusion based on the total TMAO alone should not be drawn, but TMAO could merely be a biomarker and not a causal compound.

In the present study we observed a significantly increased level of 3MH in urine samples collected after nonseafood intervention (Paper III). This is in line with studies showing that 3MH is excreted in the urine after meat consumption (Huszar *et al.* 1983; Neuhauser *et al*. 1984). However, frequent meat-eaters (Spain) had higher urinary 3-Methylhistidine (3MH) as compared to frequent fish-eaters (Japan) (Horie *et al*. 1990). Our data with increased 3MH from the `meat-eaters` support that urinary level from

3MH in fact comes from muscle protein degradation or protein turnover. As insulin attenuates protein-catabolism in healthy subjects, protein-catabolism, and thus urinary 3MH excretion, is increased in insulin resistance subjects (Lattuada *et al*. 2004). This suggests impaired insulin sensitivity after the nonseafood intervention in the present study.

Our metabolomics data showed a significant increased level of an acylcarnitine in the urine samples after nonseafood intervention. An increase in plasma acylcarnitines may be a biomarker of dysfunctional mitochondrial fatty acid oxidation (Adams *et al.* 2009), and is associated with impaired insulin sensitivity (Mihalik *et al.* 2010). We observed a significantly decreased level of 2PY (a NAD catabolite) in the urine samples collected after lean-seafood intervention compared to the nonseafood intervention, possibly suggesting a better maintenance of mitochondrial respiration (Paper III). This reduction-oxidation complex aiding the electron transport in the mitochondria is required to produce energy, which could indicate a regulation or a metabolic switch in energy production. Decreased NAD catabolism may protect against mitochondrial saturation, suggesting why lean-seafood reveal decreased TAG and VLDL and not an increase in urinary acyl carnitines. Our results may suggest improved preservation on insulin sensitivity after lean-seafood, while nonseafood intervention may lead to impaired insulin sensitivity.

As discussed, a diet high in lean-seafood is linked to beneficial outcomes. It is evident that the prevalence of lifestyle related diseases is increasing. CVD and stroke were the top cause of death in the world in 1990, and remains the top in 2010. Noteworthy, CVD and stroke increased 26-35% over the interval. The cause-specific death rate seem to drive a broad shift from communicable, maternal, neonatal, and nutritional causes towards non-communicable diseases (Lozano *et al.* 2012). According to the "Reseptregisteret" more than 170.000 Norwegians were on diabetes medication in 2014 (Norwegian Institute of Public Health 2015). The increased use oral hypoglycaemic agents, of insulin in type 2 diabetes, and the frequent prescription of statins and antihypertensive agents may have resulted in further reliance on pharmacological rather than nutritional treatment. This thesis has shown that lean-

seafood intake may reduce risk factors for development of CVD and T2D, ranked number 1 and 9, respectively, on global death causes in 2010 (Lozano *et al.* 2012). An increased intake of lean-seafood should be encouraged as a part of a healthy diet in the prevention of CVD and T2D, and therefore have a part of combating these health challenges.

4.3 Conclusions

Based on our data lean-seafood regulates fasting and postprandial lipids and glucose metabolism differently in healthy subjects after four weeks.

- Lean-seafood intake modulates fasting and postprandial lipids in healthy individuals in a manner that may have an effect on the long-term development of cardiovascular disease.
- Lean-seafood intakes determines postprandial glucose metabolism in healthy subjects in a manner that may have impact on long-term development of insulin-resistance, type 2 diabetes and cardiovascular disease.
- Lean-seafood intake, based on the urinary metabolomics in healthy subjects, suggests improved mitochondrial oxidative capacity that may have facilitated lipid catabolism.

4.4 Future perspectives

This intervention with two hot dishes per day gave a higher intake of lean-seafood per week, compared with the recommended amount. In addition, the intervention was different from the common dietary pattern and food culture in Norway, which consists of only one hot meal per day. Further studies could be carried out with only one hot dinner serving per day, resulting in a lower weekly intake closer to the average intake of lean-seafood in Norway. To test if also lower intake for a longer period of time would give similar results.

In addition to performing the study in a strict-controlled intervention, another trial could be conducted in a more realistic setting. A free-living intervention would test the real-world setting. The general population might consider free-living studies more achievable, due to less demanding condition for the subjects. To retain sufficient statistical power, the study sample should be increased to be able to detect differences between the dietary intervention groups.

In this intervention a diet high in lean-seafood resulted in beneficial outcomes in healthy subjects. It could also be of interest to evaluate the therapeutic potential of lean-seafood. Further studies could be carried out with insulin resistant or diabetic type 2 subjects.

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Appendices

Appendix I: Newspaper advertisement

Appendix II: Screening questionnaire

Appendix III: Medical and food history questionnaire

Appendix IV: Food frequency questionnaire

Appendix V: All menus

I

Appendix I

NIFES

NASJONALT INSTITUTT FOR ERNÆRINGS- OG SJØMATFORSKNING

Telefon: 95 79 43 56
E-post: magerfisk@nifes.no
www.nifes.no

Studien er godkjent av Regional komité for medisinsk og helsefaglig forskningsetikk, (REK Vest).

Invitasjon til deg som er mellom 18 og 65 år

Kan du tenke deg å delta i en studie om hvordan mager fisk påvirker omsetningen av fett og sukker i kroppen?

Dersom du føler deg frisk, ønsker vi å komme i kontakt med deg. Du må ikke røyke, være allergisk mot fisk, eller gå på medikamenter som påvirker sukker- og fettomsetningen i kroppen.

Vi ønsker å undersøke om inntak av mager fisk påvirker fordelingen av fett og muskler i kroppen, og om omsetningen av fett og sukker endres hos friske personer.

Selve studien har oppstart i januar 2013, og alle deltakerne vil i uke 4-8 og uke 16-20 få servert lunsj og middag, som enten består av ulike typer kjøtt eller ulike typer sjømat. Du vil komme til kontroll fire ganger, ca en hel dag hver gang. Ved hver kontroll vil vi gi deg et måltid og ta blodprøver av deg både før måltidet og ved faste tidspunkt etterpå. Når studien er over, vil du få vite kroppssammensetningen din.

Undersøkelsen, maten og oppfølgingen er gratis. Etter hver fullførte kontroll med blodprøver, vil du få dekket 1000 kroner for transportkostnader og tapt arbeidsfortjeneste. Dersom du tror dette er interessant for deg, ta kontakt med oss for nærmere informasjon. Interesserte deltakere vil deretter bli kontaktet per telefon med spørsmål om medisinsk historie og kostvaner.

Appendix II

II

NIFES
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ID: _____

Dato: _____

Rekruttering per telefon

Etternavn : _____ **Alder :** _____

Fornavn : _____ **Kjønn :** **K** **M**

Telefon : _____ (hjemme) _____ (arbeid) _____ (mobil)

Postadresse: _____

E-post: _____

Er du av kaukasisk avstamming (hvit hudfarge)? ☐ Ja ☐ Nei ☐ Vet ikke

Kroppsvekt (kg) _____
Høyde (m) _____
BMI (kg/m^2) _____

Vil du være tilgjengelig uke 4-8 og uke 16-20, 2013? ☐ Ja ☐ Nei ☐ Vet ikke

Vil det være mulig for deg å bruke en hel dag i uke 4, 8, 16 og 20? ☐ Ja ☐ Nei ☐ Vet ikke

Kvinner:

Menopause? ☐ Ja ☐ Nei

Hormonbehandling? ☐ Ja ☐ Nei Dersom ja, hvilke hormoner? _____

Hormonprevensjon? ☐ Ja ☐ Nei Dersom ja, hvilke hormoner? _____

(Evt. kan du sjekke?)

Har du:

Diabetes ☐ Ja ☐ Nei ☐ Vet ikke Dersom ja, tar du medisiner for det? ____

Høyt blodtrykk ☐ Ja ☐ Nei ☐ Vet ikke Dersom ja, tar du medisiner for det? ____

Høyt kolesterol ☐ Ja ☐ Nei ☐ Vet ikke Dersom ja, tar du medisiner for det? ____

Høye triglyserider ☐ Ja ☐ Nei ☐ Vet ikke Dersom ja, tar du medisiner for det? ____

Andre sykdommer ☐ Ja ☐ Nei ☐ Vet ikke Dersom ja, tar du medisiner for det? ____

(Evt. kan du sjekke?)

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ID:_____

Har du hatt:

Operasjon de siste 3 mnd? ☐ Ja ☐ Nei

Vektøkning/- reduksjon de siste 6 mnd? ☐ Ja ☐ Nei

Dersom ja, hvor mye? _____

Tar du vitamin tilskudd? ☐ Ja ☐ Nei ☐ Vet ikke Dersom ja, hvilke? _____

Tar du tran eller omega 3? ☐ Ja ☐ Nei ☐ Vet ikke Dersom ja, hvilke? _____

Vil det være OK for deg og ikke ta vitaminer, tran el omega 3 tilskudd i 5 mnd?

☐ Ja ☐ Nei ☐ Vet ikke

Allergi eller intoleranse? ☐ Ja ☐ Nei _____

Røyker? ☐ Ja ☐ Nei _____

Snus? ☐ Ja ☐ Nei Dersom ja, hvor ofte?_____

Alkohol? ☐ Ja ☐ Nei _____

Gjennomsnittlig inntak og hvor ofte? _____

Vil det være OK for deg og ikke drikke vin, øl eller annen alkohol i 2 perioder på 5 uker?

☐ Ja ☐ Nei

Vil det være OK for deg å unngå følgende matvarer i 4 uker?

Melk ☐ Ja ☐ Nei _____

Ost ☐ Ja ☐ Nei _____

Yoghurt ☐ Ja ☐ Nei _____

Is/ yoghurtis ☐ Ja ☐ Nei _____

Andre melkeprodukter (pudding, saus, rømme) ☐ Ja ☐ Nei _____

N I F E S
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ID:_____

Egg ☐ Ja ☐ Nei _____

Fiskeprodukter ☐ Ja ☐ Nei _____

Kjøttprodukter (kylling, biff, svin) ☐ Ja ☐ Nei _____

Har du deltatt i forskningsprosjekter de siste 3 måneder? ☐ Ja ☐ Nei

Dersom ja, hvilken? _____

Kan du tenke deg å ha navnet ditt i en "navne bank" for fremtidige forskningsprosjekter ved NIFES?

☐ Ja ☐ Nei

Hvordan vil du bli kontaktet? _____

Kommentarer _____

_____

_____

_____

_____

Skjema er utarbeidet etter mal fra og i samarbeid med Laval Universitet, Canada

UNIVERSITÉ LAVAL

Institut des nutraceutiques et des aliments fonctionnels
La science au service de l'alimentation

CHUQ
CENTRE HOSPITALIER UNIVERSITAIRE DE QUÉBEC
Pavillon CHUL

Appendix III

III

N I F E S
NASJONALT INSTITUTT FOR ERNÆRINGS- OG SJØMATFORSKNING

ID: _____

Dato: _____

BAKGRUNN OG MATHISTORISK SPØRRESKJEMA

I dette spørreskjemaet ber vi deg ha fokus på ulike aspekter ved din helse og dine matvaner.

Sett kryss ved ditt svar alternativ.

All besvarelser er viktig for forskningen, men du kan selv velge å besvare undersøkelsen fullstendig eller bare deler av den. Dine svar vil være konfidensielle.

1. Hvilket kjønn er du? ☐ Kvinne ☐ Mann

2. Når er du født? ___/___/_____

3. Hvor mye veier du nå?

_____ kg

4. Hva var din vekt da du var 25 år?

ca. _____ kg

Dersom du ennå ikke har fylt 25 år, ikke besvar dette spørsmålet.

5. Har du gått ned eller opp i vekt de siste 6 månedene?

☐ Ned

☐ Opp

☐ Nei, min kroppsvekt er stabil (gå til spørsmål 7)

☐ Vet ikke (gå til spørsmål 7)

6. Dersom du har gått ned/opp i vekt de siste 6 månedene, spesifiser hvor mye:

☐ 0 - 2 kg

☐ 2.1 - 5 kg

☐ 5.1 - 7 kg

☐ 7.1 - 10 kg

☐ Mer enn 10 kg

☐ Vet ikke

7. Planlegger du å gå ned i vekt de kommende månedene?

☐ Ja

☐ Nei

☐ Vet ikke

8. Lider du av en av de følgende sykdommer, eller har du fått utført større operasjoner?

Kryss av for det som gjelder deg:

	Ja	Nei	Vet ikke	Hvis Ja, spesifiser	Alder ved diagnose
Diabetes type 1 (insulinavhengig)	☐	☐	☐	_____	_____
Diabetes type 2 (livsstilsdiabetes)	☐	☐	☐	_____	_____
Hjerte-karsykdom (f.eks. hjerteinfarkt, angina, bypass, slag)	☐	☐	☐	_____	_____
Høyt blodtrykk	☐	☐	☐	_____	_____
Høyt kolesterol	☐	☐	☐	_____	_____
Høyt triglyseridnivå	☐	☐	☐	_____	_____
Stoffskiftesykdom	☐	☐	☐	_____	_____
Mage- tarmsykdom	☐	☐	☐	_____	_____
Leversykdom	☐	☐	☐	_____	_____
Nyresykdom	☐	☐	☐	_____	_____
Kreft	☐	☐	☐	_____	_____
Kirurgi (tidligere eller planlagt)	☐	☐	☐	_____	_____
Annet	☐	Spesifiser _____			

9. Reseptbelagte medisiner

Tar du noen medisiner nå? ☐ Ja ☐ Nei

Medisin navn	Dose (oppgi str.)	Hyppighet	Symtomer	Start dato	Slutt dato
_____	_____	_____	_____	___/___/___	___/___/___
_____	_____	_____	_____	___/___/___	___/___/___
_____	_____	_____	_____	___/___/___	___/___/___
_____	_____	_____	_____	___/___/___	___/___/___
_____	_____	_____	_____	___/___/___	___/___/___
_____	_____	_____	_____	___/___/___	___/___/___
_____	_____	_____	_____	___/___/___	___/___/___

Dersom du tar medisiner for å kontrollere ditt kolesterol, vil du være i stand til å avslutte behandlingen minst 6 uker før start av studien, med tillatelse fra din lege?

☐ Ja ☐ Nei ☐ Ikke aktuelt

Har du noen former for allergi mot medisiner?

☐ Ja Hvis Ja, spesifiser: _____

☐ Nei

10. Helsekostprodukter

Har du de siste 3 månedene brukt noen helsekostprodukter (kosttilskudd, vitaminer/mineraler, homøopatisk produkter, probiotisk, urtemedisiner, omega-3 kapsler, naturlegemidler)?

☐ Ja ☐ Nei

Produkt navn	Dose (oppgi stk.)	Hyppighet	Start mnd/år	Slutt mnd/år
_____	_____	_____	____/____	____/____
_____	_____	_____	____/____	____/____
_____	_____	_____	____/____	____/____
_____	_____	_____	____/____	____/____
_____	_____	_____	____/____	____/____
_____	_____	_____	____/____	____/____

Dersom du benytter noen helsekostprodukter, er det ok for deg å slutte med disse minst 6 uker før start av studien?

☐ Ja ☐ Nei ☐ Ikke aktuelt

11. Bruker du eller har du jevnlig brukt tobakk (sigaretter, snus)?

☐ Ja

☐ Nei

Hvis Ja, hvor mange ganger daglig? _____

Dersom du har sluttet å bruke tobakk, oppgi mnd/ år for sluttdato: _____

12. Bruker du eller har du jevnlig brukt alkohol?

☐ Ja

☐ Nei

Hvis Ja, type alkohol? _____

_____

Hvor mange ganger i uken? _____

Enheter konsumert hver gang? _____

(1 enhet = 1 øl, 1 glass vin, 1 liten drink)

Dersom du har sluttet å bruke alkohol, oppgi mnd/ år for sluttdato: _____

13. Bruker du eller har du jevnlig brukt narkotiske stoffer?

☐ Ja

☐ Nei

Hvis Ja, type narkotiske stoffer? _____

_____

Hvor mange ganger i uken? _____

Mengde konsumert hver gang? _____

Dersom du har sluttet å bruke narkotiske stoffer, oppgi mnd/ år for sluttdato: _____

14. Er du i fysisk aktivitet (inkludert gå/sykle til jobb, søndagstur, lagtrening, helsestudio, osv.)?

☐ Ja ☐ Nei

Type aktivitet	Hyppighet	Varighet
_____	_____/uke	____ (timer): _____ (minutter)
_____	_____/uke	____ (timer): _____ (minutter)
_____	_____/uke	____ (timer): _____ (minutter)
_____	_____/uke	____ (timer): _____ (minutter)

15. Solvaner

A. Hvor ofte bruker du solarium?

☐ 1-2 ganger i uken

☐ 2-3 ganger i mnd

☐ 1 gang i mnd

☐ Sjeldnere enn 1gang i mnd

☐ Aldri

B. Hvor mange uker de tre siste månedene har du vært på badeferie (Norge eller Syden)?

☐ 7 uker eller mer

☐ 4-6 uker

☐ 2-3 uker

☐ 1 uke

☐ Har ikke vært på badeferie

C. Hvor mye utendørsaktivitet har du om sommeren (turer, hagearbeid, jobb)?

☐ Ute nesten hele tiden

☐ Ganske mye

☐ Middels

☐ Lite

Sjømatinntak

16. Hvor ofte bruker du fisk, fiskeprodukter eller annen sjømat som middagsmat?

☐ Mer enn 5 ganger /uke

☐ 3 ganger eller mer / uke

☐ 1-2 ganger / uke

☐ 1-3 ganger / måned

☐ Sjeldnere enn 1 gang / måned

☐ Aldri

ID: _____

17. Hvis du spiser fisk, fiskeprodukter eller annen sjømat til middag, hvor mye spiser du vanligvis?
(1 porsjon = 150 gram, tilsvarer for eksempel 1 laksekotelett eller 3 fiskekaker eller 2 dl reker u/skall)

☐ 1/2 porsjon eller mindre

☐ 1 porsjon

☐ 1 ½ porsjon

☐ 2 porsjoner

☐ 3 porsjoner

18. Hvor ofte bruker du sjømat som pålegg, i salat, mellommåltid, snacks eller lignende?

☐ Mer enn 5 ganger /uke

☐ 3 - 5 ganger eller mer / uke

☐ 1-2 ganger / uke

☐ 1-3 ganger / måned

☐ Sjeldnere enn 1 gang / måned

☐ Aldri

19. Hvis du bruker sjømat som pålegg, i salat, mellommåltid, snacks eller lignende, beskriv hvor mye du vanligvis spiser?
(for eksempel boks makrell i tomat, antall fiskekaker, dl reker til antall brødskiver/knekkebrød)

_____
_____
_____
_____

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ID: _____

20. Hvor ofte spiser du vanligvis følgende sjømat som middag?

	3 ganger eller mer/uke	1-2 ganger/uke	1-3 ganger /mnd	Sjeldnere enn 1 gang/mnd	Aldri
Laks, ørret	☐	☐	☐	☐	☐
Makrell	☐	☐	☐	☐	☐
Sild	☐	☐	☐	☐	☐
Kveite	☐	☐	☐	☐	☐
Uer	☐	☐	☐	☐	☐
Steinbit	☐	☐	☐	☐	☐
Flyndre, rødspette	☐	☐	☐	☐	☐
Torsk	☐	☐	☐	☐	☐
Sei	☐	☐	☐	☐	☐
Hyse	☐	☐	☐	☐	☐
Abbor, gjedde (ferskvann)	☐	☐	☐	☐	☐
Røye, sik (ferskvann)	☐	☐	☐	☐	☐
Reker	☐	☐	☐	☐	☐
Krabbe	☐	☐	☐	☐	☐
Hummer	☐	☐	☐	☐	☐
Blåskjell	☐	☐	☐	☐	☐
Kamskjell	☐	☐	☐	☐	☐
Fiskekaker	☐	☐	☐	☐	☐
Fiskeboller	☐	☐	☐	☐	☐
Fiskepudding	☐	☐	☐	☐	☐
Fiskegrateng	☐	☐	☐	☐	☐
Fiskepinner	☐	☐	☐	☐	☐
Fiskesuppe	☐	☐	☐	☐	☐
Klippfisk	☐	☐	☐	☐	☐

21. Hvor ofte spiser du vanligvis følgende sjømat <u>som pålegg</u>?

	3 ganger eller mer/uke	1-2 ganger/uke	1-3 ganger /mnd	Sjeldnere enn 1 gang/mnd	Aldri
Makrell i tomat	☐	☐	☐	☐	☐
Sardin på boks	☐	☐	☐	☐	☐
Brisling	☐	☐	☐	☐	☐
Ansjos	☐	☐	☐	☐	☐
Røkt laks, ørret	☐	☐	☐	☐	☐
Gravet laks, ørret	☐	☐	☐	☐	☐
Tunfisk på boks	☐	☐	☐	☐	☐
Sild (sursild, rømmesild, kryddersild el.lign.)	☐	☐	☐	☐	☐
Kaviar	☐	☐	☐	☐	☐
Crabsticks	☐	☐	☐	☐	☐
Svolværpostei	☐	☐	☐	☐	☐
Lofotpostei	☐	☐	☐	☐	☐
Annet sjømat (spesifiser):	☐	☐	☐	☐	☐

_____

_____

_____

_____

22. Spiser du innmat av fisk?

☐ Ja ☐ Nei

Dersom ja, hvor mange ganger per år spiser du fiskeinnmat?

	1-3 ganger/år	4-6 ganger/år	7-9 ganger/år	≥ 10 ganger/år
Rogn	☐	☐	☐	☐
Fiskelever	☐	☐	☐	☐

23. Har du noen former for matallergi?

☐ Ja Hvis Ja, spesifiser: _____

☐ Nei

☐ Vet ikke

24. Har du noen former for matintoleranse?

☐ Ja Hvis Ja, spesifiser: _____

☐ Nei

☐ Vet ikke

25. Har du bestemte spisevaner (f.eks vegetar, lav-karbo, religiøse tilpasninger, osv.)?

☐ Ja Hvis Ja, spesifiser: _____

☐ Nei

☐ Vet ikke

26. Bruker du vektreduserende produkter (f.eks. Nutrilett, Allévo)

☐ Ja Hvis Ja, spesifiser: _____

☐ Nei

☐ Vet ikke

27. Hvor mange måltider spiser du daglig? _____ stk

28. Hvor mange mellommåltider spiser du daglig? _____ stk

29. Generelt, hvem lager måltidene hjemme hos deg? (Sett kun *ett kryss*)

- ☐ Jeg
- ☐ Min ektefelle/samboer
- ☐ Begge
- ☐ Annet familiemedlem
- ☐ Jeg spiser som oftes ferdigmat (f.eks. Fjordland eller tilsvarende)
- ☐ Jeg ønsker ikke å svare

30. Spiser du på restaurant (inkludert gatekjøkken, kafé, hamburgerrestaurant, osv)?

- ☐ Ja Hvis Ja, hvor ofte? _____
- ☐ Nei

31. Spiser du vanligvis måltidene sammen med andre personer (venner, ektefelle, familie, osv)?

- ☐ Ja
- ☐ Nei

Utenom deg selv, har andre i din familie hatt eller har en av følgende sykdommer:

32. Familiehistorie
Dersom Ja, spesifiser: F = far, M = mor, B = bror, S = søster, G = besteforeldre, U = ukjent (f.eks.adoptert)

	Ja	Nei	Vet ikke	Hvis Ja, spesifiser hvem:
Diabetes type 1 (insulinavhengig)	☐	☐	☐	_____
Diabetes type 2 (livsstilsdiabetes)	☐	☐	☐	_____
Hjerte-karsykdom (f.eks. hjerteinfarkt, angina, bypass, slag)	☐	☐	☐	_____
Høyt blodtrykk	☐	☐	☐	_____
Høyt kolesterol	☐	☐	☐	_____
Høyt triglyseridnivå	☐	☐	☐	_____
Stoffskiftesykdom	☐	☐	☐	_____
Kreft	☐	☐	☐	_____
Annet	☐	☐	☐	_____

De neste spørsmålene (spm. 33-45) er ment kun for kvinner:

33. Ved hvilken alder hadde du din første menstruasjon?

_____ år ☐ Vet ikke

34. Planlegger du å bli gravid:

☐ Ja

☐ Nei

☐ Vet ikke

35. Bruker du hormonprevensjon?

☐ Ja ☐ Nei

Hvis ja, hvilke?

☐ Tabletter (p-pille) Spesifiser produktnavn:_____

☐ Hormonspiral Spesifiser produktnavn:_____

☐ P-plaster Spesifiser produktnavn:_____

☐ P-implantat Spesifiser produktnavn:_____

☐ Annet, spesifiser _____

36. Er du i overgangsalderen (12 mnd siden siste menstruasjon)?

☐ Ja (hvis Ja, besvar spørsmålene 37 til 40)

☐ Nei (gå til spørsmål 41)

☐ Vet ikke (gå til spørsmål 41)

37. Alder for overgangsalder:

_____ år gammel ☐ Vet ikke

38. Type overgangsalder:

☐ Naturlig

☐ Fjerning av livmor

☐ Delvis fjerning av livmor og én eggstokk

☐ Delvis fjerning av livmor og begge eggstokker

☐ Kvinnelig sterilisering

☐ Fremprovosert av kreftbehandling eller annen medisinsk behandling

☐ Vet ikke

39. Tar du hormonerstatninger nå for tiden?

☐ Ja

☐ Nei

Hvis Ja, startdato: ___ / ___ / ___
Hvis Ja, type hormoner: _____

40. Har du tidligere vært behandlet med hormonerstatning?

☐ Ja

☐ Nei

Hvis Ja, slutt dato: ___ / ___ / ___
Hvis Ja, type hormoner: _____

41. Antall graviditeter (inkludert spontanaborter og aborter):

_____ ☐ Ikke aktuelt

42. Har du noen ganger hatt <u>diagnosen</u> diabetes under graviditet (svangerskapsdiabetes)?

☐ Ja (hvis Ja, besvar spørsmål 43-45)

☐ Nei

☐ Ikke aktuelt

43. Har du vært under medisinsk oppfølging grunnet din diabetes under graviditet?

☐ Ja

☐ Nei

☐ Ikke aktuelt

Hvis Ja, spesifiser hvilken graviditet? _____

44. Har du fått kostveiledning grunnet din diabetes under graviditet?

☐ Ja

☐ Nei

☐ Ikke aktuelt

Hvis Ja, spesifiser hvilken graviditet? _____

45. Har du tatt medisiner grunnet din diabetes under graviditet?

☐ Ja

☐ Nei

☐ Ikke aktuelt

Hvis Ja, spesifiser hvilken graviditet? _____

Hvis Ja, type medisin (insulin eller annet) _____

Takk for innsatsen med besvarelsen!

Spørreskjema er utarbeidet etter mal fra og i samarbeid med Laval Universitet, Canada

UNIVERSITÉ LAVAL

Institut des nutraceutiques et des aliments fonctionnels
La science au service de l'alimentation

CHUQ
CENTRE HOSPITALIER UNIVERSITAIRE DE QUÉBEC
Pavillon CHUL

Appendix IV

IV

SPØRRESKJEMA OM KOSTHOLD

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

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

Riktig markering er slik: [X]

Ved feil markering, fyll hele ruten slik: ■

Av hensyn til den maskinelle lesingen – pass på at arkene ikke brettes.

Alle svar vil behandles fortrolig.

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

UNIVERSITETET I OSLO

Eksempel på utfylling av spørsmålene.

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

1. Hvor mye brød pleier du å spise?

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

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

Sum skiver pr. dag = 6

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

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

1. Hvor mye brød pleier du å spise?

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

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

Sum skiver pr. dag = _____

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

2. Hva pleier du å smøre på brødet?

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

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

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

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

4. Hvilke typer pålegg spiser du?

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

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

5. Frokostgryn

Svar enten per måned eller per uke.

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

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

(1 glass = 2 dl)

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

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

Svar enten per måned eller per uke.

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

8. Kalde drikker

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

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

9. Alkoholholdige drikker

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

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

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

10. Varme drikker

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

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

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

11. Middagsretter

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

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

Middagsretter fortsetter neste side.....

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

Middagsretter forts...

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

12. Poteter, ris, spagetti, grønnsaker

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

	Aldri/ sjelden	Gang pr. måned 1	2	3	eller 1	Gang pr. uke 2-3	4-5	6-7	8+		Mengde pr. gang				
Poteter, kokte og bakte	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Potetmos	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Potetsalat m/majones	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	1 ☐	2-3 ☐	4-5 ☐	6-7 ☐	8+ ☐
Fløtegratinerte poteter	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Stekte poteter	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Pommes frites (gatekjøkken, frityrstekt)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Pommes frites, varmet i ovn	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Bønner/linser	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Ris	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Spagetti, makaroni, pasta	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Pølsebrød, lomper	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Gulrot	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Hodekål	☐	☐	☐	☐	☐	☐	☐	☐	☐	(skalk)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Kålrot	☐	☐	☐	☐	☐	☐	☐	☐	☐	(skive)	1/2 ☐	1 ☐	2 ☐	3 ☐	4+ ☐
Blomkål	☐	☐	☐	☐	☐	☐	☐	☐	☐	(hode)	1/8 ☐	1/6 ☐	1/4 ☐	1/3 ☐	1/2+ ☐
Brokkoli	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/8 ☐	1/4 ☐	1/2 ☐	3/4 ☐	1+ ☐
Rosenkål	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Løk, rå og stekt	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Salat (eks. issalat, ruccola)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1/2 ☐	1 ☐	11/2 ☐	2 ☐	21/2+ ☐
Paprika	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ring)	1-2 ☐	3-4 ☐	5-6 ☐	7-8 ☐	9+ ☐
Avokado	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/4 ☐	1/2 ☐	3/4 ☐	1 ☐	11/2+ ☐
Tomat	☐	☐	☐	☐	☐	☐	☐	☐	☐	(stk)	1/2 ☐	1 ☐	11/2 ☐	2 ☐	21/2+ ☐
Mais	☐	☐	☐	☐	☐	☐	☐	☐	☐	(ss)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Frosne grønnsakblandinger	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐
Blandet salat (eks. salat, tomat, agurk, mais)	☐	☐	☐	☐	☐	☐	☐	☐	☐	(dl)	1 ☐	2 ☐	3 ☐	4 ☐	5+ ☐

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

13. Saus og dressing

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

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

(Velg en eller to typer)

Smør/margarin

- ☐ Smør (meierismør)
- ☐ Bremykt
- ☐ Melange
- ☐ Soft Flora, Soft Ekstra
- ☐ Vita
- ☐ Soft Oliven
- ☐ Flytende margarin på flaske (Vita, Melange, Bremykt o.l.)
- ☐ Annen margarin

Oljer

- ☐ Olivenolje
- ☐ Soyaolje
- ☐ Maisolje
- ☐ Solsikkeolje
- ☐ Valnøttolje
- ☐ Rapsolje
- ☐ Vita hjertego
- ☐ Andre oljer

15. Frukt

Svar enten per måned eller per uke.

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

16. Grønnsaker og frukt

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

Mindre enn 1 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5+ ☐

Hvor mange frukt spiser du vanligvis pr. dag?

Mindre enn 1 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5+ ☐

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

17. Desserter, kaker, godteri

Svar enten per måned eller per uke.

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

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

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

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

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

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

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

19. Måltider

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

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

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

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

20. Kjønn

Mann ☐

Kvinne ☐

21. Alder

Alder: ☐☐ år

22. Vekt og høyde

Høyde: ☐☐☐ cm

Vekt: ☐☐☐ kg

23. Eventuelle andre matvarer

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

BRUK BLOKKBOKSTAVER

Tusen takk for innsatsen!

UNIVERSITETET I OSLO

Appendix V

Appendix Food ingredients and nutrition composition of Tuesday - Sunday menu of the 10 900 kJ/d lean-seafood diet

Tuesday menu of the 10 900 kJ/d Lean-seafood diet		Energy kJ	Protein g	Fat g	SFA g	MUFA g	PUFA g	Chol* mg	Carbo* g	Total fiber g	Ca mg	Vit D µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	12 g Peanut butter	317	2.7	6.4	1.4	2.6	2.2	0	1.5	0.8	4	0
	40 g Jam	305	0.3	0.2	0	0	0.1	0	16.6	1.2	10	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Lunch	140 g Saithe	521	28.8	0.6	0.1	0.1	0.1	85	0.6	0	14	1.3
	1.5 g Salt	0	0	0	0	0	0	0	0	0	1	0
	150 g Carrot	228	1	0.2	0	0	0.2	0	10.1	4.1	42	0
	9 g Wheat flour	124	1.1	0.2	0	0	0.1	0	5.8	0.3	2	0
	1.6 g Parsley	2	0	0	0	0	0	0	0	0.1	3	0
	18 g Butter	552	0.2	14.8	9.7	4	0.3	39	0.2	0	1	1.4
	90 g Whole-wheat bread	934	6.6	3.8	0.4	1.8	1.3	0	38	4.4	15	0
Dinner	200 g Cod	798	45.2	0.8	0.2	0	0.4	146	0	0	20	3.8
	100 g Carrot	152	0.7	0.1	0	0	0.1	0	6.7	2.7	28	0
	100 g Apple	207	0.3	0.2	0	0	0	0	10.6	2.1	6	0
	40 g Onion	53	0.5	0	0	0	0	0	2.3	0.8	8	0
	20 g Parsley root	23	0.3	0.1	0	0	0	0	0.6	0.7	10	0
	100 g Coconut milk	861	2	21.3	18.9	0.9	0.2	0	1.5	1.5	18	0
	1.5 g Salt	0	0	0	0	0	0	0	0	0	0	0
	8 g Lemon	6	0.1	0	0	0	0	0	0.2	0.2	2	0
	15 g Olive oil	552	0	14.9	2.1	11.1	1.1	0	0	0	0	0
	100 g Potatoes	339	1.9	0.1	0	0	0.1	0	17.1	1.6	9	0
Evening meal	100 g Oatmeal	1638	11.4	7.8	1.1	2.7	2.5	0	63.1	10.6	40	0
	60 g Banana	211	0.7	0.2	0.1	0	0.1	0	10.9	1	4	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Snacks	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	32 g Oat biscuits	614	2.5	6.1	2.2	2.8	0.8	1	19.5	1.8	11	0
	Total	10885	121	80.1	36.5	26.4	10.5	271	323.3	46.8	329	6.5
	% energy		18.6	27.7					49.6	3.4		
	MUFA:SFA					0.72						
	PUFA:SFA						0.29					

* Cholesterol
* Carbohydrate

Wednesday menu of the 10 900 kJ/d Lean-seafood diet		Energy kJ	Protein g	Fat g	SFA g	MUFA g	PUFA g	Chol* mg	Carbo* g	Total fiber g	Ca mg	Vit D µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	12 g Peanut butter	317	2.7	6.4	1.4	2.6	2.2	0	1.5	0.8	4	0
	40 g Jam	305	0.3	0.2	0	0	0.1	0	16.6	1.2	10	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Lunch	100 g Scallops	433	22.4	1.4	0.3	0.1	0.6	158	0	0	9	5.2
	20 g Butter	614	0.2	16.4	10.8	4.4	0.3	44	0.2	0	1	1.6
	50 g Barley	671	4.3	0.6	0.1	0.1	0.3	0	31.4	5.3	12	0
	80 g Tomato	59	0.6	0.2	0	0	0.1	0	2	1.2	5	0
	40 Onion	53	0.5	0	0	0	0	0	2.3	0.8	8	0
	30 g Leeks	35	0.5	0.1	0	0	0	0	1.1	0.8	16	0
	10 g Rapeseed oil	365	0	9.9	0.7	5.9	2.8	0	0	0	0	0
	3 g Chives	3	0.1	0	0	0	0	0	0	0.1	4	0
	7 g Lemon	5	0.1	0	0	0	0	0	0.2	0.1	2	0
Dinner	255 g Saithe	949	52.5	1	0.3	0.3	0.3	156	1	0	26	2.3
	120 g Canned tomatoes	100	1.2	0.2	0	0	0.1	0	3.6	1.1	14	0
	8 g Olive oil	294	0	8	1.1	5.9	0.6	0	0	0	0	0
	15 g Olives	80	0.2	1.9	0.3	1.3	0.2	0	0	0.6	9	0
	2 g Parsley	3	0.1	0	0	0	0	0	0	0.1	3	0
	80 g Tofu (soyabean cheese)	291	6.2	3.7	0.5	1.1	1.9	0	1.9	2	102	0
	6 g Butter	184	0.1	4.9	3.2	1.3	0.1	13	0.1	0	0	0.5
	50 g Pasta	274	2.4	2	0.4	0.6	0.9	23	9.1	0.5	6	0.1
	40 g Lettuce	21	0.3	0	0	0	0	0	0.6	0.4	7	0
	60 g Tomato	32	0.5	0.1	0	0.1	0.1	0	0.7	0.7	8	0
	30 g Squash	21	0.4	0	0	0	0	0	0.7	0.3	5	0
	10 g Leeks	12	0.2	0	0	0	0	0	0.4	0.3	5	0
	101 g Whole bread roll	1125	7.7	2.7	0.7	0.5	1.1	0	51.6	2	13	0
Evening meal	100 g Cereals	1560	10	9.5	2.2	4.1	3.1	0	56.9	8.9	43	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Snacks	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	32 g Oat biscuits	614	2.5	6.1	2.2	2.8	0.8	1	19.5	1.8	11	0
	Total	11097	130.9	77.8	24.5	31.5	16.6	395	331.9	42,8	413	9.7
	% energy		19.7	26.4					50.0	3.1		
	MUFA:SFA					1.29						
	PUFA:SFA						0.68					

* Cholesterol, * Carbohydrate

Thursday menu of the 10 900 kJ/d		Energy	Protein	Fat	SFA	MUFA	PUFA	Chol*	Carbo*	Total fiber	Ca	Vit D
Lean-seafood diet		kJ	g	g	g	g	g	mg	g	g	mg	µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	12 g Peanut butter	317	2.7	6.4	1.4	2.6	2.2	0	1.5	0.8	4	0
	40 g Jam	305	0.3	0,2	0	0	0.1	0	16.6	1.2	10	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Lunch	150 g Pollack fillets	558	30.9	0.6	0.2	0.2	0.2	92	0.6	0	15	1.4
	80 g Noodles	580	3.8	3.4	1.6	1.3	0.3	0	22.2	1.5	6	0
	100 g Celeriac	114	1.6	0.3	0.1	0	0.2	0	2.8	3.5	48	0
	150 g Carrot	228	1	0.2	0	0	0.2	0	10.1	4.1	42	0
	20 g Peppers	16	0.2	0	0	0	0	0	0.5	0.4	1	0
	50 g Broccoli	63	1.6	0.1	0	0	0.1	0	1.1	1.5	23	0
	20 g Butter	614	0.2	16.4	10.8	4.4	0.3	44	0.2	0	1	1.6
	1 g Salt	0	0	0	0	0	0	0	0	0	1	0
Dinner	115 g Stockfish	782	43.5	1.2	0	0	0.5	175	0	0	92	2.8
	160 g Potato	491	3	0.2	0	0	0.2	0	24.3	2.6	10	0
	60 g Onions	80	0.7	0.1	0	0	0	0	3.4	1.2	13	0
	3 g Garlic	13	0.2	0	0	0	0	0	0.5	0	1	0
	60 g Peppers	73	0.7	0.1	0.1	0	0.1	0	2.8	1.1	4	0
	170 g Tomato	135	1.5	0.3	0	0	0.2	0	4.8	2.0	16	0
	20 g Tomato puree	66	0.9	0	0	0	0	0	2.6	0.7	5	0
	20 g Olive oil	736	0	19.9	2.8	14.7	1.5	0	0	0	0	0
	15 g Rapeseed oil	548	0	14.8	1.1	8.9	4.2	0	0	0	0	0
	100 g Honeydew	116	0.6	0.1	0	0	0	0	5.6	0.9	9	0
Evening meal	60 g Oatmeal	983	6.8	4.7	0.7	1.6	1.5	0	37.9	6.4	24	0
	60 g Banana	211	0.7	0.2	0.1	0	0.1	0	10.9	1	4	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Snacks	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	32 g Oat biscuits	614	2.5	6.1	2.2	2.8	0.8	1	19.5	1.8	11	0
	Total	10091	118.1	77.6	21.4	36.9	13.6	312	285.9	43.6	421	5.8
	% energy		19.5	29.0					47.3	3.5		
	MUFA:SFA					1.72						
	PUFA:SFA						0.63					

* Cholesterol
* Carbohydrate

Friday menu of the 10 900 kJ/d		Energy	Protein	Fat	SFA	MUFA	PUFA	Chol*	Carbo*	Total fiber	Ca	Vit D
Lean-seafood diet		kJ	g	g	g	g	g	mg	g	g	mg	µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	12 g Peanut butter	317	2.7	6.4	1.4	2.6	2.2	0	1.5	0.8	4	0
	40 g Jam	305	0.3	0.2	0	0	0.1	0	16.6	1.2	10	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Lunch	140 g Cod	559	31.6	0.6	0.1	0	0.3	102	0	0	14	2.7
	120 g Potato	407	2.3	0.1	0	0	0.1	0	20.5	1.9	11	0
	3 g Garlic	13	0.2	0	0	0	0	0	0.5	0	1	0
	10 g Olive oil	368	0	9.9	1.4	7.4	0.7	0	0	0	0	0
	30 g Leeks	35	0.5	0.1	0	0	0	0	1.1	0.8	16	0
	10 g Butter	307	0.1	8.2	5.4	2.2	0.2	22	0.1	0	0	0.8
	30 g Tomato	21	0.2	0.1	0	0	0	0	0.8	0.4	3	0
	40 g Lettuce	21	0.3	0	0	0	0	0	0.6	0.4	7	0
	80 g Tortilla	998	6.7	5.7	1.5	2.9	0.7	0	38.6	2	34	0
Dinner	190 g Cod	758	42.9	0.8	0.2	0	0.4	139	0	0	19	3.6
	70 g Tortilla chips	1424	4.5	17.6	8.3	5.9	2.3	0	39.2	3.6	49	0
	8 g Olive oil	294	0	8	1.1	5.9	0.6	0	0	0	0	0
	70 g Canned tomatoes	58	0.7	0.1	0	0	0.1	0	2.1	0.6	8	0
	100 g Beans	400	4.8	0.6	0.1	0.1	0.3	0	14.3	6.6	48	0
	60 g Avocado	482	1.1	11.7	2.5	7.3	1.3	0	0.3	3.1	7	0
	8 g Lime	3	0.1	0	0	0	0	0	0.1	0	2	0
	50 g Tofo (soy-bean cheese)	182	3.9	2.3	0.3	0.7	1.2	0	1.2	1.3	64	0
Evening meal	100 g Oatmeal	1638	11.4	7.8	1.1	2.7	2.5	0	63.1	10.6	40	0
	60 g Banana	211	0.7	0.2	0.1	0	0.1	0	10.9	1	4	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Snacks	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	32 g Oat biscuits	614	2.5	6.1	2.2	2.8	0.8	1	19.5	1.8	11	0
	Total	11863	132.2	88,8	26.0	40.9	14.8	264	349	49.0	433	7.1
	% energy		18.6	28.2					49.1	3.3		
	MUFA:SFA					1.57						
	PUFA:SFA						0.57					

* Cholesterol
* Carbohydrate

Saturday menu of the 10 900 kJ/d Lean-seafood diet		Energy kJ	Protein g	Fat g	SFA g	MUFA g	PUFA g	Chol* mg	Carbo* g	Total fiber g	Ca mg	Vit D µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	12 g Peanut butter	317	2.7	6.4	1.4	2.6	2.2	0	1.5	0.8	4	0
	40 g Jam	305	0.3	0.2	0	0	0.1	0	16.6	1.2	10	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Lunch	140 g Cod	559	31.6	0.6	0.1	0	0.3	102	0	0	14	2.7
	20 g Rapeseed oil	730	0	19.7	1.5	11.9	5.5	0	0	0	0	0
	30 g Onion	40	0.4	0	0	0	0	0	1.7	0.6	6	0
	40 g Peppers	33	0.3	0.1	0	0	0.1	0	1.1	0.8	2	0
	40 g Carrot	61	0.3	0	0	0	0	0	2.7	1.1	11	0
	40 g Tomato puree	157	1.3	0.1	0	0	0	0	7.3	0.9	6	0
	4 g Cornstarch	59	0	0	0	0	0	0	3.5	0.1	0	0
	2 g Bouillon cube	12	0.3	0.1	*	*	*	0	0.2	0	1	0
	40 g Pineapple	118	0.2	0	0	0	0	0	6.6	0.3	2	0
	2 g Sugar	34	0	0	0	0	0	0	2	0	0	0
	1 g Parsley	1	0	0	0	0	0	0	0	0.1	2	0
	190g Rice	817	4.6	0.6	0.2	0.2	0.2	0	41	2.7	55	0
	50 g Broccoli	63	1.6	0.1	0	0	0.1	0	1.1	1.5	23	0
Dinner	195 g Cod	778	44.1	0.8	0.2	0	0.4	142	0	0	20	3.7
	90 g Peppers	110	1.0	0.2	0	0	0	0	4.1	2.3	6	0
	37 g Onion	56	0.6	0	0	0	0	0	2.3	0.7	7	0
	60g Canned tomatoes	50	0,6	0,1	0	0	0.1	0	1.8	0,5	7	0
	20 g Rapeseed oil	730	0	19.7	1.5	11.9	5.5	0	0	0	0	0
	80 g Whole-wheat bread	831	5,9	3.4	0.3	1.6	1.1	0	33.8	3.9	14	0
	20 g Margarine	594	0	16	6.8	2.4	6	0	0.1	0	0	1.6
Evening meal	60 g Cereals	936	6	5.7	1.3	2.5	1.9	0	34.1	5.3	26	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Snacks	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	32 g Oat biscuits	614	2.5	6.1	2.2	2.8	0.8	1	19.5	1.8	11	0
	Total	10453	119	82.2	15.8	36.3	25.2	245	299	37.5	310	8
	% energy		19.0	29.7					47.8	2.9		
	MUFA:SFA					2.30						
	PUFA:SFA						1.60					

* Cholesterol
* Carbohydrate

Sunday menu of the 10 900 kJ/d Lean-seafood diet		Energy kJ	Protein g	Fat g	SFA g	MUFA g	PUFA g	Chol* mg	Carbo* g	Total fiber g	Ca mg	Vit D µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	12 g Peanut butter	317	2.7	6.4	1.4	2.6	2.2	0	1.5	0.8	4	0
	40 g Jam	305	0.3	0.2	0	0	0.1	0	16.6	1.2	10	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Lunch	140 g Cod	559	31.6	0.6	0.1	0	0.3	102	0	0	14	2.7
	2 g Garlic	9	0.2	0	0	0	0	0	0.3	0	0	0
	30 g Leeks	36	0.5	0	0	0	0	0	1.1	0.9	15	0
	10 g Sesame oil	370	0	10	1.5	3.8	4.3	0	0	0	1	0
	8 g Cornstarch	119	0	0	0	0	0	0	6.9	0.1	1	0
	4 g Sugar	64	0	0	0	0	0	0	3.8	0	7	0
	12 g Rapeseed oil	438	0	11.8	0.9	7.1	3.3	0	0	0	0	0
	17 g Lime	7	0.1	0	0	0	0	0	0.1	0.1	4	0
	10 g Sweet chilie	118	0	0	*	*	*	*	6.8	*	*	*
	190 g Rice	817	4.6	0.6	0.2	0.2	0.2	0	41	2.7	55	0
Dinner	190 g Cod	758	42.9	0.8	0.2	0	0.4	139	0	0	19	3.6
	100 g Canned tomatoes	83	1	0.2	0	0	0.1	0	3	0.9	12	0
	40g Garlic	18	0.4	0	0	0	0	0	0.6	0	0	0
	15 g Olive oil	552	0	14.9	2.1	11.1	1.1	0	0	0	0	0
	60 g Onion	80	0.7	0.1	0	0	0	0	3.4	1.2	13	0
	30 g Olives	430	0.7	10.8	1.5	7.5	1.2	0	0.5	1.2	18	0
	30 g Peppers	36	0.3	0.1	0	0	0	0	1.4	0.6	2	0
	2 g Parsley	3	0.1	0	0	0	0	0	0	0.1	3	0
	170 g Potato	576	3.2	0.2	0	0	0.2	0	29.1	2.7	15	0
	12 g Butter	368	0.1	9.8	6.5	2.7	0.2	26	0.1	0	0	1
Evening meal	100 g Cereals	1560	10	9.5	2.2	4.1	3.1	0	56.9	8.9	43	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Snacks	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	32 g Oat biscuits	614	2.5	6.1	2.2	2.8	0.8	1	19.5	1.8	11	0
	Total	10914	116.8	84.6	19.1	42.3	18.5	268	323.1	37.0	336	7.3
	% energy		18.9	29.4					48.2	2.9		
	MUFA:SFA					2.32						
	PUFA:SFA						1.61					

* Cholesterol
* Carbohydrate

Food ingredients and nutrition composition of Monday - Sunday menu of the 10 900 kJ/d non-seafood diet

Monday menu of the 10 900 kJ/d nonseafood diet		Energy kJ	Protein g	Fat g	SFA g	MUFA g	PUFA g	Chol* mg	Carbo* g	Total fiber g	Ca mg	Vit D µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	16 g Norwegian brown cheese	308	1.8	4.6	3.1	1.2	0.1	17	6.2	0	56	0
	20 g Cow-milk cheese	292	5.4	5.4	3.5	1.5	0.1	16	0	0	146	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
	5 g Margarine	148.5	0	4	1.2	0.75	1.8	0	0	0	0	0.4
Lunch	200 g egg	1186	24.9	20.3	5.8	8.4	2.6	844	0.6	0	115	7.6
	40 g Low fat milk	64	1.3	0.3	0.2	0.1	0	1	1.8	0	40	0.2
	9 g Rapeseed oil	329	0	8.9	0.7	5.3	2.5	0	0	0	0	0
	30 g Onion	40	0.4	0	0	0	0	0	1.7	0.6	6	0
	40 g Peppers	48	0.4	0.1	0	0	0	0	1.8	0.8	2	0
	30 g Celery	19	0.3	0	0	0	0	0	0.4	0.8	26	0
	60 g Potato	203	1.1	0.1	0	0	0.1	0	10.3	1	5	0
	60 g Tomato	44	0.4	0.1	0	0	0.1	0	1.5	0.9	4	0
	20 g Ham	87	3.6	0.7	0.3	0.3	0.1	10	0	0	1	0
	100 g Whole-wheat bread	1038	7.4	4.2	0.4	2	1.4	0	42.2	4.9	17	0
Dinner	155 g Beef	905	41.1	5.6	2.2	2	0.6	110	0	0	6	0
	12 g Olive oil	441	0	11.9	1.7	8.8	0.9	0	0	0	0	0
	50 g Peppers	61	0.6	0.1	0.1	0	0.1	0	2.4	0.9	3	0
	80 g Carrots	122	0.6	0.1	0	0	0.1	0	5.4	2.2	22	0
	35 g Spinach	31	0.9	0.2	0	0	0.1	0	0.1	0.7	46	0
	180 g Pasta	985	8.6	7.2	1.4	2	3.2	81	32.8	1.8	20	0.4
	50 g Low fat milk	80	1.6	0.3	0.2	0.1	0	2	2.3	0	50	0.2
	6 g Cornstarch	89	0	0	0	0	0	0	5.2	0.1	1	0
	3.3 g Cod liver oil	122	0	3.3	0.5	1.5	0.9	16	0	0	0	7.1
Evening meal	100 g Cereals	1560	10	9.5	2.2	4.1	3.1	0	56.9	8.9	43	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Snacks	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	Total	11108	125.5	89.6	23.8	38.5	18.9	1097	314.6	38.3	705	15.9
	% energy		18.9	30.4					47.3	2.8		
	MUFA:SFA					1.62						
	PUFA:SFA						0.79					

* Cholesterol, Carbohydrate

Tuesday menu of the 10 900 kJ/d nonseafood diet		Energy kJ	Protein g	Fat g	SFA g	MUFA g	PUFA g	Chol* mg	Carbo* g	Total fiber g	Ca mg	Vit D µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	16 g Norwegian brown cheese	308	1.8	4.6	3.1	1.2	0.1	17	6.2	0	56	0
	20 g Cow-milk cheese	292	5.4	5.4	3.5	1.5	0.1	16	0	0	146	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
	5 g Margarine	148.5	0	4	1.2	0.75	1.8	0	0	0	0	0.4
Lunch	100 g Pork	562	25.9	3.3	1.1	1.5	0.4	71	0	0	5	0
	15 g Rapeseed oil	548	0	14.8	1.1	8.9	4.2	0	0	0	0	0
	260 g Bouillon water	31	1	0.3	*	*	*	0	0.3	0	0	0
	130 g Carrot	198	0.9	0.1	0	0	0.1	0	8.7	3.5	36	0
	110 g Cauliflower	109	2.1	0.2	0.1	0	0.1	0	2.5	2.5	23	0
	35 g Leeks	41	0.6	0.1	0	0	0	0	1.3	1	18	0
	20 g Celery root	23	0.3	0.1	0	0	0	0	0.6	0.7	10	0
	60 g Asparagus	55	1.9	0.1	0.1	0	0.1	0	0.7	0.9	13	0
	135 g Whole-wheat bread	1402	10	5.7	0.6	2.7	1.9	0	57	6.7	23	0
	15 g Margarine	445	0	12	5.1	1.8	4.5	0	0	0	0	1.2
Dinner	120 g Chicken	626	33.5	1.6	0.5	0.7	0.2	97	0	0	7	0
	120 g Potato	407	2.3	0.1	0	0	0.1	0	20.5	1.9	11	0
	50 g Onion	67	0.6	0.1	0	0	0	0	2.9	1	11	0
	35 g Leeks	41	0.6	0.1	0	0	0	0	1.3	1	18	0
	20 g Coconut milk	172	0.4	4.3	3.8	0.2	0	0	0.3	0.3	4	0
	80 g Skimmed milk	113	2.6	0.1	0.1	0	0	1	3.8	0	80	0
	100 g Canned tomatoes	83	1	0.2	0	0	0.1	0	3	0.9	12	0
	3 g Sugar	51	0	0	0	0	0	0	3	0	0	0
	20 g Peanuts	483	5.2	9.2	1.6	4.2	2.9	0	2.5	1.5	12	0
	45 g Whole-wheat bread	467	3.3	1.9	0.2	0.9	0.6	0	19	2.2	8	0
	3.7 g Cod liver oil	137	0	3.7	0.6	1.7	1	19	0	0	0	8
Evening meal	100 g Oatmeal	1638	11.4	7.8	1.1	2.7	2.5	0	63.1	10.6	40	0
	60 g Banana	211	0.7	0.2	0.1	0	0.1	0	10.9	1	4	0
Snacks	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	Total	10971	125.2	82.5	24.2	29.2	21.8	221	318.1	49.3	603	9.6
	% energy		19.1	28.4					48.4	3.6		
	MUFA:SFA					1.21						
	PUFA:SFA						0.90					

* Cholesterol
* Carbohydrate

Wednesday menu of the 10 900 kJ/d nonseafood diet		Energy kJ	Protein g	Fat g	SFA g	MUFA g	PUFA g	Chol* mg	Carbo* g	Total fiber g	Ca mg	Vit D µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	16 g Norwegian brown cheese	308	1.8	4.6	3.1	1.2	0.1	17	6.2	0	56	0
	20 g Cow-milk cheese	292	5.4	5.4	3.5	1.5	0.1	16	0	0	146	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
	5 g Margarine	148.5	0	4	1.2	0.75	1.8	0	0	0	0	0.4
Lunch	110 g Turkey	655	36.9	0.8	0.2	0.2	0.2	84	0	0	6	0
	10 g Margarine	297	0	8	3.4	1.2	3	0	0	0	0	0.8
	60 g Barley	805	5.2	0.7	0.1	0.1	0.3	0	37.7	6.4	14	0
	43 g Onion	66	0.7	0	0	0	0	0	2.8	0.8	9	0
	1 g Sugar	16	0	0	0	0	0	0	0.9	0	2	0
	12 g Lemon	10	0.1	0	0	0	0	0	0.3	0.3	3	0
	60 g Pomegranate	145	0.8	0.1	0	0.1	0	0	4.7	6	7	0
	8 g Rapeseed oil	292	0	7.9	0.6	4.7	2.2	0	0	0	0	0
Dinner	130 g Chicken	679	36.3	1.7	0.5	0.8	0.3	105	0	0	8	0
	45 g Onion	75	0.9	0	0	0	0	0	3.1	0.9	9	0
	130 g Canned tomatoes	133	1.7	0,2	0	0	0,1	0	4.9	1.5	17	0
	10 g Margarine	297	0	8	3,4	1,2	3	0	0	0	0	0,8
	10 g Wheat flour	138	1,2	0,2	0	0	0,1	0	6,4	0,4	2	0
	110 g Low fat milk	176	3,6	0,8	0,4	0,2	0	3	5,1	0	110	0,4
	10 g Parmesan cheese	188	4,2	3	2,1	0,8	0,1	8	0,4	0	138	0
	20 g Cow-milk cheese	292	5,4	5,4	3,5	1,5	0,1	16	0	0	160	0
	50 g Pasta	274	2,4	2	0,4	0,6	0,9	23	9,1	0,5	6	0,1
	91 g Whole-wheat bread	945	6,7	3,8	0,4	1,8	1,3	0	38,4	4,5	15	0
	140 g Mixed salad	86	1.4	0.1	0	0.1	0.1	0	2.4	1.7	25	0
	3.3 g Cod liver oil	122	0	3.3	0.5	1.5	0.9	16	0	0	0	7.1
Evening meal	100 g Cereals	1560	10	9.5	2.2	4.1	3.1	0	56.9	8.9	43	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Snacks	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	Total	10908	139.9	72.2	25.8	22.75	18.8	288	322.3	46.7	875	9.6
	% energy		21.4	25.0					49.3	3.4		
	MUFA:SFA					0.88						
	PUFA:SFA						0.73					

* Cholesterol
* Carbohydrate

Thursday menu of the 10 900 kJ/d nonseafood diet		Energy kJ	Protein g	Fat g	SFA g	MUFA g	PUFA g	Chol* mg	Carbo* g	Total fiber g	Ca mg	Vit D µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	16 g Norwegian brown cheese	308	1.8	4.6	3.1	1.2	0.1	17	6.2	0	56	0
	20 g Cow-milk cheese	292	5.4	5.4	3.5	1.5	0.1	16	0	0	146	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
	5 g Margarine	148.5	0	4	1.2	0.75	1.8	0	0	0	0	0.4
Lunch	100 g Chicken	616	27.1	4.2	1.3	1.4	1.2	80	0	0	6	0
	10 g Olive oil	368	0	9.9	1.4	7.4	0.7	0	0	0	0	0
	90 g Noodles	653	4.2	3.9	1.8	1.4	0.4	0	24.9	1.7	7	0
	150 g Carrot	228	1	0.2	0	0	0.2	0	10.1	4.1	42	0
	50 g Broccoli	63	1.6	0.1	0	0	0.1	0	1.1	1.5	23	0
	20 g Celery root	23	0.3	0.1	0	0	0	0	0.6	0.7	10	0
	10 g Asparges	9	0.3	0	0	0	0	0	0.1	0.2	2	0
	2 g Garlic	9	0.2	0	0	0	0	0	0.3	0	0	0
Dinner	130 g Beef	1153	37.6	13.9	6.9	5.1	0.4	113	0	0	21	0
	15 g Olive oil	552	0	14.9	2.1	11.1	1.1	0	0	0	0	0
	8 g Cornstarch	119	0	0	0	0	0	0	6.9	0.1	1	0
	4 g Sugar	64	0	0	0	0	0	0	3.8	0	7	0
	90 g Yogurt	270	3.5	3.4	2.3	0.9	0.1	14	5	0	115	0
	6 g Tomato puree	20	0.3	0	0	0	0	0	0.8	0.2	2	0
	40 g Sugar peas	70	1.4	0	0	0	0	0	2.1	1.2	22	0
	145 g Rice	624	3.5	0.4	0.1	0.1	0.1	0	31.3	2	42	0
	100 g Melon	116	0.6	0.1	0	0	0	0	5.6	0.9	9	0
	3 g Cod liver oil	111	0	3	0,5	1.4	0.8	15	0	0	0	6.5
Evening meal	100 g Oatmeal	1638	11.4	7.8	1.1	2.7	2.5	0	63.1	10.6	40	0
	60 g Banana	211	0.7	0.2	0.1	0	0.1	0	10.9	1	4	0
Snacks	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	Total	9996	114.9	78.6	25.7	35.4	10.7	255	284	37.9	623	6.9
	% energy		19.2	29.6					47.4	3.0		
	MUFA:SFA					1.38						
	PUFA:SFA						0.42					

* Cholesterol
* Carbohydrate

Friday menu of the 10 900 kJ/d nonseafood diet		Energy kJ	Protein g	Fat g	SFA g	MUFA g	PUFA g	Chol* mg	Carbo* g	Total fiber g	Ca mg	Vit D µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	16 g Norwegian brown cheese	308	1.8	4.6	3.1	1.2	0.1	17	6.2	0	56	0
	20 g Cow-milk cheese	292	5.4	5.4	3.5	1.5	0.1	16	0	0	146	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
	5 g Margarine	148.5	0	4	1.2	0.75	1.8	0	0	0	0	0.4
Lunch	105 g Chicken	548	29.3	1.4	0.4	0.6	0.2	85	0	0	6	0
	15 g Olive oil	552	0	14.9	2.1	11.1	1.1	0	0	0	0	0
	80 g Tortilla	998	6,7	5.7	1.5	2.9	0.7	0	38.6	2	34	0
	200 g Mixed salad	169	2.2	0.4	0	0.1	0.3	0	5.3	3.7	52	0
	1 g Parmesan cheese	19	0.4	0.3	0.2	0.1	0	1	0	0	14	0
	5 g Olive oil	184	0	5	0.7	3.7	0.4	0	0	0	0	0
Dinner	130 g Chicken	572	30.9	1.3	0.4	0.4	0.3	73	0	0	5	0
	10 g Olive oil	368	0	9.9	1.4	7.4	0.7	0	0	0	0	0
	34 g Onion	59	0.6	0	0	0	0	0	2.4	0.7	7	0
	30 g Leeks	35	0.5	0.1	0	0	0	0	1.1	0.8	16	0
	40 g Carrot	61	0.3	0	0	0	0	0	2.7	1.1	11	0
	100 g Tomato	132	1.4	0.3	0	0	0.2	0	4.6	2.1	14	0
	50 g Corn	189	1.3	0.5	0.1	0.1	0.3	0	8.3	1	3	0
	10 g Lime	4	0.1	0	0	0	0	0	0.1	0.1	2	0
	50 g Lettuce	26	0.4	0.1	0	0	0.1	0	0.8	0.6	9	0
	40 g Squash	28	0.5	0	0	0	0	0	0.9	0.4	6	0
	25 g Avocado	201	0.5	4.9	1	3	0.6	0	0.1	1.3	3	0
	20 g Cow-milk cheese	292	5.4	5.4	3.5	1.5	0.1	16	0	0	160	0
	120 g Whole-wheat bread	1246	8.9	5.1	0.5	2.4	1.7	0	50.7	5.9	20	0
	3.4 g Cod liver oil	126	0	3.4	0.5	1.6	0.9	17	0	0	0	7.3
Evening meal	100 g Oatmeal	1638	11.4	7.8	1.1	2.7	2.5	0	63.1	10.6	40	0
	50 g Jam	381	0.3	0.3	0	0.1	0.1	0	20.8	1.5	12	0
	60 g Banana	211	0.7	0.2	0.1	0	0.1	0	10.9	1	4	0
Snacks	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	Total	11100	122.7	83.5	21.6	41.6	13.3	225	327.1	46.4	686	7.7
	% energy		18.5	28.4					49.2	3.3		
	MUFA:SFA					1.92						
	PUFA:SFA						0.62					

* Cholesterol
* Carbohydrate

Saturday menu of the 10 900 kJ/d nonseafood diet		Energy kJ	Protein g	Fat g	SFA g	MUFA g	PUFA g	Chol* mg	Carbo* g	Total fiber g	Ca mg	Vit D µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	16 g Norwegian brown cheese	308	1.8	4.6	3.1	1.2	0.1	17	6.2	0	56	0
	20 g Cow-milk cheese	292	5.4	5.4	3.5	1.5	0.1	16	0	0	146	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
	5 g Margarine	148.5	0	4	1.2	0.75	1.8	0	0	0	0	0.4
Lunch	130g Pork	1014	34.5	11.6	4.3	5.1	1.4	100	0	0	22	0
	12 g Butter	368	0.1	9.8	6.5	2.7	0.2	26	0.1	0	0	1
	15 g Olive oil	552	0	14.9	2.1	11.1	1.1	0	0	0	0	0
	30 g Onion	40	0.4	0	0	0	0	0	1.7	0.6	6	0
	40 g Peppers	33	0.3	0.1	0	0	0.1	0	1.1	0.8	2	0
	40 g Carrot	61	0.3	0	0	0	0	0	2.7	1.1	11	0
	40 g Tomato puree	157	1.3	0.1	0	0	0	0	7.3	0.9	6	0
	4 g Cornstarch	59	0	0	0	0	0	0	3.5	0.1	0	0
	40 g Pineapple	118	0.2	0	0	0	0	0	6.6	0.3	2	0
	225 g Rice	968	5.4	0.7	0.2	0.2	0.2	0	48.6	3.1	65	0
	50 g Broccoli	63	1.6	0.1	0	0	0.1	0	1.1	1.5	23	0
Dinner	150 g Chicken	783	41.8	2	0.6	0.9	0.3	122	0	0	9	0
	12 g Rapeseed oil	438	0	11.8	0.9	7.1	3.3	0	0	0	0	0
	90 g Tomato	57	0.8	0.2	0	0.1	0.1	0	1.6	1.0	12	0
	60 g Peppers	63	0.5	0.2	0	0	0.1	0	2.1	1.5	4	0
	60 g Squash	42	0.7	0.1	0	0	0	0	1,3	0.6	9	0
	32 g Onion	49	0.6	0	0	0	0	0	2.0	0.6	6	0
	25 g Tomato puree	83	1.1	0.1	0	0	0	0	3.2	0.9	7	0
	45 g Whole-wheat roll	464	3.6	2.4	0.9	0.4	0.9	0	17.6	1.7	6	0.2
	3.3 g Cod liver oil	122	0	3.3	0.5	1.5	0.9	16	0	0	0	7.1
Evening meal	100 g Cereals	1560	10	9.5	2.2	4.1	3.1	0	56.9	8.9	43	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Snacks	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	Total	10762	125.8	83.7	26.3	37.0	14.9	297	306.8	38.4	537	8.7
	% energy		19.5	29.3					47.6	2.9		
	MUFA:SFA					1.41						
	PUFA:SFA						0.57					

* Cholesterol, Carbohydrate

Sunday menu of the 10 900 kJ/d nonseafood diet		Energy kJ	Protein g	Fat g	SFA g	MUFA g	PUFA g	Chol* mg	Carbo* g	Total fiber g	Ca mg	Vit D µg
Breakfast	120 g Whole-wheat bread	1120	10.8	1.8	0.2	0.4	0.8	0	47.2	8.4	24	0
	16 g Norwegian brown cheese	308	1.8	4.6	3.1	1.2	0.1	17	6.2	0	56	0
	20 g Cow-milk cheese	292	5.4	5.4	3.5	1.5	0.1	16	0	0	146	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
	5 g Margarine	148.5	0	4	1.2	0.75	1.8	0	0	0	0	0.4
Lunch	125 g Chicken	653	34.9	1.6	0.5	0.7	0.3	101	0	0	8	0
	10 g Olive oil	736	0	19.8	2.8	14.8	1.4	0	0	0	0	0
	42 g Onion	62	0.7	0	0	0	0	0	2.6	0.8	8	0
	15 g Whole-wheat bread	141	1.4	0.2	0	0	0.1	0	6	1	3	0
	22 g Egg	130	2.7	2.2	0.6	0.9	0.3	92	0.1	0	13	0.8
	20 g Avocado	161	0.4	3.9	0.8	2.4	0.4	0	0.1	1	2	0
	165 g Yoghurt	495	6.4	6.3	4.1	1.7	0.2	26	9.1	0	211	0
	200 g Rice	860	4.8	0.6	0.2	0.2	0.2	0	43.2	2.8	58	0
Dinner	130 g Beef	759	34.5	4.7	1.8	1.7	0.5	92	0	0	5	0
	10 g Sesame	244	1.8	5	0.7	1.9	2.2	0	1.1	1.2	98	0
	10 g Rapeseed oil	365	0	9.9	0.7	5.9	2.8	0	0	0	0	0
	40 g Celeriac	46	0.6	0.1	0	0	0.1	0	1.1	1.4	19	0
	100 g Tomato	53	0.9	0.2	0	0.1	0.1	0	1.2	1.2	13	0
	70 g Peas	199	3.6	0.3	0.1	0	0.1	0	5.6	3.9	17	0
	10 g Margarine	296	0	8	3.4	1.2	3.0	0	0	0	0	0.8
	4 g Sugar	68	0	0	0	0	0	0	4	0	0	0
	15 g Cream cheese	171	0.9	4.2	2.8	1.1	0.1	14	0	0	13	0
	120 g Carrot	182	0.8	0.1	0	0	0.1	0	8	3.2	34	0
	1 g Garlic	4	0.1	0	0	0	0	0	0.2	0	0	0
	140 g Potato	475	2.7	0.1	0	0	0.1	0	23.9	2.2	13	0
	3.3 g Cod liver oil	122	0	3.3	0.5	1.5	0.9	16	0	0	0	7.1
Evening meal	100 g Cereals	1560	10	9.5	2.2	4.1	3.1	0	56.9	8.9	43	0
	30 g Jam	229	0.2	0.2	0	0	0.1	0	12.5	0.9	7	0
	200 g Orange juice	364	1.2	0	0	0	0	0	20	0.2	22	0
Snacks	120 g Apple	248	0.4	0.2	0	0	0	0	12.7	2.5	7	0
	100 g Banana	352	1.1	0.3	0.1	0	0.1	0	18.1	1.6	6	0
	Total	11451	129.7	96.7	29.3	42.1	19.1	374	312.7	42.6	863	9.1
	% energy		18.9	31.8					45.6	3.0		
	MUFA:SFA					1.44						
	PUFA:SFA						0.65					

* Cholesterol, Carbohydrate