



The lifestyle of patients with severe mental illness, receiving treatment in mental care facilities in Asker, Norway

Master thesis by Madeleine E. Angelsen

Department of Nutrition Institute of basal medical sciences Faculty of Medicine

UNIVERSITY OF OSLO May 2023

# The lifestyle of patients with severe mental illness, receiving treatment in mental care facilities in Asker, Norway

Madeleine E. Angelsen



Supervisors: Kjetil Retterstøl Dawn E. Peleikis

Department of Nutrition, Institute of Medicine

# © Madeleine E. Angelsen 2023

The lifestyle of patients with severe mental illness, receiving treatment in mental care facilities in Asker, Norway

Madeleine E. Angelsen

# Acknowledgements

The completion of this master thesis depended on several contributors. The patients from Asker DPS that have participated from 2021 until 2023 deserve most gratitude for their efforts. Their willingness to share personal details have been indispensable on the path to make scientific advancements on the topic at hand. A big salute and thank you to each participant.

Thanks also to Emma N. Johannesen, for being a true role model and pioneer within dietary assessment in the psychiatric ward. It is an understatement to say that she has served as an inspiration during the conduction of the current project; her work laid the very foundation that this thesis is constructed upon. She also provided her services as a dietary counsellor for patients that participated in this edition, voluntarily. For that, both the patients and I are deeply grateful (and quite impressed).

A generous thanks is directed to both supervisors of this master project, professor and senior physician Kjetil Retterstøl & senior psychiatrist Dawn Peleikis. Dawns insights and knowledge of the psychiatric field supplied this thesis with solid scientific background. Her long experience and useful advice made barriers in the mental care ward manageable to overcome. Kjetils' expertise and long experience made me confident that the project would arrive safely at the finish line from the start of. Being a part of Kjetils' research team at Domus Medica has also inspired me to prosper for an academic career in the future. This thesis' quality is truly due to both of my counselors outstanding expertise.

Hege B. Henriksen deserves thankful regards for tutoring with the DIGIKOST-FFQ and for quick replies and supportive comments throughout the conduction of the thesis. The FACT- and Sikta teams in Asker are also entitled to a special notice for their readiness to welcome a new and strange interactor in the middle of an already demanding work situation. A special thanks to psychiatrist Marek Kieliszczyk, who readily supplied blood parameters and helped with participant recruitment.

I also want to thank the student counsellor at Domus Medica who told me that I would not manage to complete this education if I prospered to be a high-performance boxer at the same time. Her words have motivated me through so many obstacles since 2017. My deepest gratitude, as I prepare for the upcoming European Games while submitting this thesis.

Finally, I want to thank you Elias, for inspiring me to gain more knowledge about the dietary challenges of psychiatric patients. I would not have all the insights that I have as a dietitian today if it was not for the story that you told me three years ago. This thesis is dedicated to you and your mother. - *Madeleine E. Angelsen, Oslo 2023* -

# Abstract

Introduction: The life expectancy of patients with severe mental illness (SMI) is shortened by 15-20 years compared to the average population. This is mainly due to cardiovascular disease (CVD). Many modifiable risk factors for CVD such as diabetes type 2 (DMT2) and the metabolic syndrome (MetS) are prevalent in SMI patients. Anti-psychotic medication is also known to increase appetite and promote weight gain. SMI patients have previously been found to lead unhealthy lifestyles with little exercise, unhealthy dietary habits, frequent smoking, and high alcohol consumption. Food based dietary guidelines (FBDG) in Norway have been developed to promote a healthy diet and avoid premature death and morbidity. A previous assessment in Asker experienced barriers regarding somatic screening and dietary assessment of SMI patients, and knowledge of Norwegian SMI patient's lifestyle is still limited. In this study we hypothesized low compliance to FBDG and recommendations for exercise, high prevalence of metabolic syndrome (MetS) and deviant biomarkers for liver enzymes and micronutrients. Methods: Adult SMI-patients (schizophrenia spectrum-/bipolar disorder) receiving treatment in Asker, Norway was included in this study. Anthropometric measures and blood samples relevant to assess risk for CVD were collected from all participants. Compliance to FBDG were assessed with an online FFQ. Lifestyle related statements were documented for qualitative nuancing. New data was merged with the assessment from 2022 and analyzed together. *Results:* Forty-two SMI-patients (male=23, female=18) were assessed between 2021 and 2023. Mean age was 41.5 years (±SD 12.1), and obesity (BMI  $\ge$  30 kg/m<sup>2</sup>) were highly prevalent with the median BMI being 29.9 kg/m<sup>2</sup> ( $\pm$ SD 5,4). In 42 participants, 67% were deemed abdominally obese, and the mean waist circumference (WC) was 106.2 cm (±12.8). MetS was present in 50% of the participants, and serum total cholesterol, LDL- and non-HDL cholesterol exceeded the treatment goals for high-risk individuals. Vitamin D deficiency was identified in 17.3% of the subjects, but the median (nmol/L) of 79.7 (±SD 38.3) in men and 75.8 (±SD 17.1) in women indicated that serum concentration of vitamin D was too low. The median Diet Score was 7.5, corresponding to low/moderate compliance to FBDG. Reported exercise was below the recommended amount but might be even lower due to recall- and social desirability bias. Smoking was more prevalent than in average Norwegians, while reported alcohol intake was lower than expected. Conclusion: Obesity and MetS was highly prevalent, posing the SMIpatients at high risk for CVD. Compliance to FBDG was low, and physical activity level was below recommended. SMI-patients in Norway are thought to benefit from lifestyle-based interventions led by dietary professionals.

# **Table of contents:**

ABSTRACT	III
LIST OF FIGURES	VI
LIST OF TABLES	VI
LIST OF APPENDICES	VII
ABBREVIATIONS	VIII
1. BACKGROUND	1
1.1 Severe Mental Illness	1
<b>1.2 ANTI-PSYCHOTIC MEDICATION</b>	5
<b>1.3 SOMATIC HEALTH IN SMI PATIENTS</b>	6
1.4 METABOLIC RISK FACTORS FOR ACVD	7
1.5 UNHEALTHY LIFESTYLE IN SMI PATIENTS	9
<b>1.6 Prevention of lifestyle-induced morbidity</b>	13
1.7 KNOWLEDGE OF NORWEGIAN SMI-PATIENTS' LIFESTYLE	15
2. AIM	17
2.1 Objective	17
<b>2.2 Hypothesis in the present thesis</b>	17
3. MATERIALS AND METHODS	18
3.1 Study Design	18
3.2 RECRUITMENT	19
3.3 DATA COLLECTION	19
3.4 DIETARY COUNSELLING	21
<b>3.5 DATA HANDLING AND STORAGE</b>	21
<b>3.6 STATISTICAL ANALYSIS</b>	23
4. RESULTS	25
4.1 CHARACTERISTICS AND ANTHROPOMETRY	25
4.2 BIOCHEMISTRY	28
4.3 PREVALENCE OF METS	28
4.4 VITAMIN D	29
4.5 INTAKE OF 10 FOOD GROUPS	30
4.6 LIFESTYLE INDEX	32
4.7 QUALITATIVE ASSESSMENT	34
5. DISCUSSION	39
5.1 METHODOLOGICAL CONSIDERATIONS	39
5.2 DISCUSSION OF RESULTS	42
5.3 ETHICAL CONSIDERATIONS	48
5.4 Further research	48
6. CONCLUSION	49
REFERENCES	50

# **List of Figures**

Figure 1: Positive symptoms (illustration)
Figure 2: Negative symptoms (illustration)
Figure 3: Manic phase (illustration)
Figure 4: Risk factors for ACVD in SMI patients (illustration)
Figure 5: Diagnosis criteria for Mets (WHO 2009)
Figure 6: Treatment goals for high ACVD-risk individuals
Figure 7: Vitamin D metabolism (illustration)
Figure 8: Norwegian guidelines for a healthy diet
Figure 9: Flow chart of the study design
Figure 10: Flow chart of recruitment 2021/22 + 2022/23
Figure 11: Distribution of participants across BMI-categories
Figure 13: Distribution of Diet Scores: Low, Intermediate and High
Figure 14: Distribution of Alcohol Consumption: Low, Intermediate and High

Figure 15: Patient Case: Controversy of somatic screening in the mental care ward

# List of Tables

- Table 1: Requisitioned blood parameters.
- Table 2: Calculation of DIGIKOST-score
- Table 3: Calculation of Lifestyle Index

Table 4: Participant Characteristics

Table 5: Prescribed psychotropic agents

Table 6a: Anthropometry in groups of gender

Table 6b: Anthropometry in groups of diagnosis

Table 7: Biochemistry values

Table 8a: Prevalence of MetS in groups of gender

Table 8b: Prevalence of MetS in groups of diagnosis

Table 9: Vitamin D Status

Table 10: Intake of 10 food groups

Table 11: Compliance to Norwegian FBDG

Table 12a: Health Index of each domain
Table 12b: Distribution across the Health Index
Table 13a: Qualitative statements: Side effects of AP-medication
Table 13b: Qualitative statements: Barriers during hospitalization
Table 13c: Qualitative statements: Nutrition Literacy
Table 13d: Qualitative statements: Signs of eating disordered behavior.
Table 13e: Qualitative statements: Perception of lifestyle and health

# **List of Appendices**

Appendix 1: Patient case (Figure 15: Illustration)

Appendix 2: Study description (mental care facilities in Asker DPS)

Appendix 3: Consent form and information about the study (participants)

Appendix 4: List of blood parameters (Clinicians requisitioning blood samples)

Appendix 5: DIGIKOST FFQ

Appendix 6: Regional Ethical Committee (REK) for Medical Research approval of the study

# Abbreviations

SMI; Severe Mental Illness CVD; Cardiovascular Disease FBDG; Food Based Dietary Guidelines SCZ; Schizophrenia SSD; Schizophrenia Spectrum Disorder(s) **BPD**; Bipolar Disease DPS: District Psychiatric Center FACT; Flexible Assertive Community Treatment AP; Anti-Psychotic FGA; First Generation Antipsychotic SGA; Second Generation Antipsychotic ACVD; Atherosclerotic Cardiovascular Disease MetS; Metabolic Syndrome TG; Triglycerides DMT2; Diabetes Mellitus Type 2 LDL; Low Density Lipoprotein **BP**; Blood Pressure BED; Binge Eating Disorder FFQ; Food Frequency Questionnaire SD; Standard Deviation

# 1. Background

### **1.1 Severe Mental Illness**

SMI is a term used for mental disorders which vigorously reduces the level of functioning, ability to self-care, and quality of life of those affected. SMI's vary in both neurocognitive pathophysiology and presentation of symptoms (1). Most SMI diagnoses are thought to result from both genetic susceptibility and environmental exposure (2-4). Those suffering from a SMI are often not capable of maintaining employment, depending on the severity of their symptoms (5). Taken together, SMI constitutes a detrimental economic, social, and personal cost when measured as years of life lost to premature mortality, and years lived with morbidity (6-8).

### 1.1.1 Schizophrenia and Psychosis Disorders

Schizophrenia (SCZ) is considered the most severe psychotic disorder (9). This SMI is a complex neuropsychiatric *syndrome*; demonstrating that it is diagnosed by symptomatic presentation. Mental afflictions with SCZ-characteristics are often collectively referred to as schizophrenia spectrum disorders (SSD) (10). In the ICD-10 registry for psychiatric- and behavioral disorders, SSDs include diagnoses between F20 and F29 (1). Diagnoses that are encompassed under SSD are recognized by derangement of emotions, belief, perception and cognitive function (11). SSDs present with both *negative* and *positive* symptoms. These symptoms are often continuously present, in varying degree and proportion to each other. In



Figure 1:Positive symptoms entails hallucinations, delusions and paranoid conceptions that poses detrimental stress on the person afflicted.

periods where the illness intensifies, positive and negative symptoms can overwhelm the patient's normal cognitive function (12).

#### Positive symptoms: Psychosis

SCZ/SSD diagnosis require that *positive* symptoms are present. 'Positive symptoms' are intuitively misleading, implying that they have a beneficial impact on those affected. But, 'positive' solely refers to mental perceptions being present *in addition* to normal sensatory experiences in the patient. Positive symptoms include hallucinations, delusion, and paranoid conceptions, which are known symptoms of psychosis. Hallucinations and delusions might disturb visual impressions and entail hearing voices. This often results in a state of grave confusion and anxiety in the affected person (10). Qualitative assessments of experiences in SCZ patients confirms the detrimental consequences that psychosis entails (12)

Negative symptoms: Apathy and loss of affection Besides the positive symptoms, patients suffering from SSDs are likely to experience *loss* of mental attributes; referred to as *negative* symptoms. These symptoms include apathy, indifference, emotional dissociation, depression, and derangement of concentration (9). Such symptoms often lead to self-isolation, impairment of social functioning and limits work ability. Ability to organize, think in a structured manner and working memory are often affected too (9).



**Figure 2:** Negative symptoms deprives the patient of usual abilities, and include depression, loss of affection, feeling of dissociation and inability to function properly in work- or education related tasks.

### Prevalence and prognosis

The lifetime risk of developing an SSD has persistently been estimated to 1% (10). In Norway, that accounts for 5-600 diagnosed cases each year. People who undergo psychosis typically experience symptoms for the first time during early adulthood (16-35 years of age). The disease can vary in symptom manifestation and severity during the life course (9). The recovery rate from SSDs have been estimated to 14%, implying that 1 out of 7 patients who develop a SSD will recover completely (16).

#### 1.1.2 Bipolar disorder

Bipolar Disease (BPD) is encompassed in the ICD-10 chapter of affective disorders, F30-39 (1). BPD, including all subtypes, are neurobiologically or clinically related to the formerly used diagnosis manic-depressive psychosis, but does not have to entail positive symptoms (17). BPD is generally characterized by profound alterations in mood and perception. The mood of BPD-patients manifests as opposite poles, hence the name *bipolar* disorder. The "poles" present as manic periods with excitation and increased levels of energy, followed by periods of depression (18). Severe BPD may also present with psychotic functioning and delusions.



Figure 3: Persons experiencing a manic episode might undertake an uncritical and over-energetic character. The manic episode may be experienced as overwhelmingly positive while it lasts but might also provoke behavior with adverse repercussions.

#### Manic symptoms

Manic periods in BPD are recognized by increased energy, flood of ideas and speech, hyperactivity, and decreased desire to sleep. Such symptoms are present in hypomanic episodes, known to be less severe than actual mania. During full-blown mania, delusions and behavioral disturbance increase progressively into psychosis, disrupting the patients perception of reality severely (19). Mania and hypomania include elevated mood with both euphoria and agitation. A manic period, with its euphoric character, might be perceived as intensely positive for the affected person while it lasts. However it can lead to negative consequences following the uncritical character affected patients often undertake (20).

#### **Depressive symptoms**

Untreated depressive phases of BPD can last 6-9 months, and are characterized by apathy, lack of initiative and interest. Inability to react emotionally can be the most outspoken clinical symptom of depression in BPD (18). Hormones regulating circadian rhythm are influenced during depressive phases, leading to alterations in sleep pattern and hypersomnia (21). Symptoms of anxiety can be present during depressive phases in BPD, leading to social isolation (18).

#### BPD type 1 and -2

Many subtypes of BPD exist, separated by the severity of illness and nature of symptoms. The main division is based upon presentation of the elevated mood; BPD type 1 is characterized by manic and depressive mood, while BPD type 2 is characterized by depressive and hypomanic episodes, which is a milder form for mania (22). Generally, BPD 1 is considered the more detrimental subtype of the two, as psychosis is the symptom leading to most functional impairment. BPD type 1 patients have been found to suffer from poorer quality of life and lower work ability than healthy controls (23, 24).

#### Prevalence and prognosis

BPD type 1 occurs in 0.5-1% of the population, while BPD type 2 in as much as 2.5% (25). There is a strong genetic component associated with BPD, proposing children of BPD-

patients to 10 times higher risk of developing BPD (26). Traumatic events might also provoke establishment of BPD. Disease debut is common in teenage, while it may present as behavioral disturbance, social anxiety and introversion already in childhood (18). Many BPD-patients need lifelong treatment, including medicines that stabilizes mood and improve life performance and -quality (25).

#### 1.1.3 Treatment of SMI

SMI-patients require lifelong treatment. In Norway, patients receive therapy in different sections of psychiatric health wards, based on their needs and level of functioning. This study is focused on patients receiving treatment in Asker, Norway. Asker District Psychiatric Center (DPS) is a psychiatric outpatient clinic in Asker, which investigates and treats more than 1800 people over the age of 18 for mental disorders and drug problems.

#### 1.1.4 Out-patient treatment

Many patients with an SMI are directed to out-patient treatment in the psychiatric ward. These patients are more likely to manage selfcare and ordinary life tasks and can meet for scheduled appointments with mental care providers. In this study, mental care providers in Asker DPS contacted eligible SMI-patients that received out-patient treatment to propose study participation.

#### 1.1.5 Special-care facilities (ambulant services)

Other SMI- patients require more support and are less likely to meet for scheduled appointments in an outpatient setting. Such patients might need close contact with mental care providers, including home visits and ambulant service. The Flexible Assertive Community Treatment (FACT) -model is a model for psychiatric treatment that aims to provide as much or little support as each patient needs, in order to promote patients' self-management and feeling of proficiency (27). FACT is an underlying section of the psychiatric ward in Asker, providing mental care and help with different life tasks for various psychiatric patients. The distribution between high- and low priority patients is dynamic, and patients can be moved between these groups according to their current phase of illness (27).

#### 1.1.6 In-patient treatment

Hospitalization of SMI-patients can be due to acute circumstances in their mental illness, where they might constitute a danger to themselves or their surroundings. Such events can be

mania, psychosis, or severe depressive phases. In cases where these conditions pose suicidal risk on the patient, long term hospitalization in special psychiatric facilities can be necessary. If the mentioned phases are thought to be short term and does not constitute suicidal hazard, patients can be voluntarily hospitalized in psychiatric care facilities. In Asker DPS, 24-hour treatment is offered to patients who need stabilization and continuous treatment up to a few weeks. The study at hand invited patients from the 24-hour section in Asker (Sikta). These patients have ability to consent and are hospitalized with an average duration of 14 days.

### **1.2 Anti-Psychotic medication**

During psychosis, there is a hyperdopaminergic state in the brain and central nervous system. This is the main target of alteration with medical treatment (28). Anti-Psychotic (AP) drugs function by modulating distorted receptor activity, leading to a relief of psychotic symptoms. Currently, more than 20 different AP-drugs are prescribed for psychosis in patients with SSD and BPD. Each AP drug have slightly different working mechanism, efficacy, and presentation of side effects (29, 30).

### 1.2.1 First Generation AP-medication

The first AP agents were invented around 1950 and are known today as First-Generation AP (FGA) medication. FGA medication acts by blocking dopamine receptors, which downscales psychosis symptoms. Side effects of FGA-medication include indifference, sedation, Parkinson-like motoric symptoms and hyperprolactinemia (31). Introduction of FGA medication like chlorpromazine was a crucial first step in evolving sufficient and humane AP treatment of SMI patients with psychosis symptoms (32).

#### 1.2.2 Second-Generation AP-medication

Second-generation AP (SGA) agents were developed later in the 2000<sup>th</sup> century. The effect of SGA medication is more specific than FGA, and less afflicted with parkinsonism, agitation, and hyperprolactinemia. However, there are numerous other side effects following SGA-medication, with varying severity of repercussions with different drug types (33). Well known side effects of SGA agents are those afflicting metabolism and increasing appetite, leading to weight gain, and comprised metabolic health (34). However, SGA medication has consistently proved to be more potent, and cause less extra-pyramidal side-effects than FGA agents (35).

#### 1.2.3 Challenged compliance to AP-medication

AP-medication is a crucial component in the treatment of psychosis in SMI. However, patients might be discouraged to sustain AP-medication when experiencing unpleasant side-effects. Limiting side effects is therefore important to increase patients' compliance to AP-medication (33). An important goal in SMI-treatment is to model a tolerable medication plan, both regarding type of medication, dosage, and administration form. Oftentimes, an emerging dilemma is to balance the burden of AP-induced side effects with the treatment effect (33).

### **1.3 Somatic health in SMI patients**

Patients with an SMI have 15-20 years shorter life expectancy relative to the general population (36). This is also the case in well-developed countries such as our neighboring ones in Scandinavia (37, 38). In a register-based cohort study including more than 100.000 SMI patients living in Denmark, Finland and Sweden, researchers found the patients to have a shortage of 11-20 years compared to the life expectancy of mentally healthy inhabitants. Standardized mortality rates were 2-3 times higher in SMI afflicted persons, for both BPD and SCZ. The increased mortality was not explained by unnatural causes like suicide or accidents, but arising from regular somatic diseases, especially those of the cardiovascular system (37).

# 1.3.1 Cardiovascular disease

CVD includes several types of pathologic conditions, including coronary heart -, cerebrovascular-, peripheral arterial-, rheumatic heart- and congenital heart disease, as well as deep vein thrombosis and pulmonary embolism (39). Worldwide, CVD accounted for 32% of all deaths in 2019. Noncommunicable diseases caused 17 million premature deaths in 2019, and 38% of these were due to CVD (39). The main reason for premature death in SMI patients has proved to be CVD (40, 41).

#### 1.3.2 Pathophysiology of atherosclerotic cardiovascular disease (ACVD)

Atherosclerosis is a CVD affecting the blood vessels, but with repercussions affecting the entire circulatory system. Atherogenesis, development of atherosclerotic lesions, is a complex process provoked by multiple physiological factors.

In short, atherosclerosis is present when the inside of the arteries develops lesions and get thickened by a lipid containing substance called plaque. This causes calcification and

narrowing of the blood vessels, with subsequent restrained blood flow. Destabilization and secession of plaque generating blood clots is a feared consequence of atherosclerosis, that may cause myocardial infarction or ischemic stroke.

Atherosclerotic lesions is caused by retention of lipid cells in the intima of the artery is facilitated by inflammation of the endothelial cells as well as elevated levels of blood cholesterol (42). Chronic inflammation and high blood glucose drive atherosclerosis. Inflammation increases endothelial permeability and accumulation of debris, leading to the release of proinflammatory cytokines. Elevated blood glucose modifies lipid particles, increasing oxidative stress and activating the protein kinase C-pathway (42), leading to the initiation of atherosclerosis.

# 1.3.3 Disease burden of ACVD

Atherosclerotic CVD (ACVD) is on the course of becoming the leading reason for morbidity and mortality worldwide (43). Globally, myocardial infarction and ischemic stroke are the two leading causes of death, and responsible for most years of life lost as well as loss of disability-adjusted life years (44).

# 1.3.4 Genetic and modifiable disposition

There are many conditions predisposing for atherosclerosis and CVD, among them genetic

**Figure 4:** Risk factors for atherosclerotic cardiovascular disease, associated with severe mental illness include impairment of metabolism, obesity, hypertension, unhealthy diet, sedentary behavior, and anti-psychotic medication.

variation (45). Somatic health conditions are usually multi-faceted, but in regard of ACVD it is fundamental to address lifestyle and behavior coupled to increased ACVD risk. This entails smoking, inactivity, unhealthy diet, adverse use of medication among adverse metabolic conditions that increases the risk for ACVD development (46).

# **1.4 Metabolic risk factors for ACVD**

Hyperglycemia, hyperlipidemia, hypertension, and obesity are underlying factors that drive the development of ACVD (47). These metabolic conditions are comprised in the umbrella diagnosis known as the Metabolic Syndrome (MetS). Risk factors for MetS are preventable, and if already prevalent, reversible at least to some degree (48, 49).

Central Obesity F: > 88 cm M: > 102 cm	Y ↓HDL Cholesterol F: <1,3 mmol/L M: <1,0 mmol/L			
1	<b>↑ Fasting Triglycerides</b> ≥ 1,7 mmol/L			
<b>◆Fasting Gluco</b> ≥ 5,6 mmol/L	Hypertension ≥ 130 mmHg (D) ≥ 85 mmHg (S)			

**Figure 5**: Diagnosis Criteria for The Metabolic Syndrome (MetS). Co-existence of at least 3 conditions qualifies for MetS diagnosis. WHO 2009

#### 1.4.1 The Metabolic Syndrome

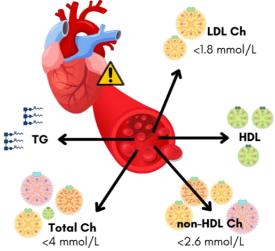
MetS is defined as co-occurrence of 3 or more of 5 metabolic hallmarks for increased ACVDrisk: dyslipidemia, elevated fasting glucose and -triglycerides (TG), hypertension, obesity and/or central obesity (50). Cut off-values for each condition is presented in figure 5.

Acknowledging that the mortality from CVD is increased in patients with SMI, it is important to

address that also MetS, obesity and diabetes mellitus type 2 (DMT2) are more prevalent in this group, compared to mentally healthy controls (51-53). Taken together, SMI patients are more than 58% more likely to be afflicted with MetS than healthy controls (54).

#### 1.4.2. Dyslipidemia and treatment goals

Diagnosis criterions for MetS include limits for HDL cholesterol and TG regarding harmful levels of blood lipids. However, other blood lipid-parameters are also important to appraise the risk for ACVD. Generally, Total serum Cholesterol (TC) is considered slightly elevated when exceeding 6 mmol/L, elevated if >7 mmol/L and severely so when >8 mmol/L (55). LDLcholesterol is recommended to be kept <3mmol/L (55). However, these limits might not be optimal treatment goals for an "at-risk" population like SMI patients. Since the risk of ACVD is elevated in SMI individuals,



**Figure 6:** Individuals at high risk for atherosclerotic cardiovascular disease are recommended to aim for lower levels of lipids associated with increased atherogenesis: LDL-Ch levels <1.8 mmol/L, non-HDL-Ch <2.6 mmol/L and total-Ch <4 mmol/L. Ch; Cholesterol.

ESC/EAS guidelines, suggesting LDL kept <1.8 mmol/L and non-HDL cholesterol <2.6 mmol/L could be appropriate targets for SMI-patients. ESC/EAS recommends total cholesterol to be <4mmol/L (56).

# 1.5 Unhealthy lifestyle in SMI patients

Unhealthy diet and sedentary behavior increase the risk for obesity, DMT2 and MetS, and are modifiable risk factors for development of ACVD. Prevention of the mentioned conditions is possible by intervening with diet and physical activity (57).

SMI patients are known to eat unhealthier and engage less in exercise. Smoking, alcohol, and use of prohibited substances also alters the risk for CVD, and these habits are more inherent in persons with SMI (58). AP drugs potently increases appetite and preference for energy dense food, which often leads to substantial weight gain (59). These risk factors represent independent points of why SMI patients are excessively liable to develop MetS, obesity and DMT2.

### 1.5.1 Diet of SMI patients

Comprehensive reports have stated that patients with SMI have unfortunate dietary patterns with high amounts of saturated fat, refined carbs and highly processed foods, and low intake of fiber, fruit, vegetables, and fish (60-62). Socioeconomic status is intertwined with dietary choices, explaining why persons with low income and education, such as SMI patients, might have a less healthy diet (63, 64). Depressive symptoms of SCZ and BPD are of importance when considering underlying reasons for why this patient group finds little interest in planning, preparing, and consuming a wholesome diet. There is even suggestions that underlying genetic mechanisms that predisposes SMI, both potentiates the urge to eat unhealthy food and causes impairment in glucose metabolism (65).

#### 1.5.2 Physical Activity level of SMI patients

In recent studies, SMI patients have proved to be more inactive than healthy controls (60, 66). Less engagement in exercise can be partly explained by some of the negative symptoms associated with SMI; lack of initiative and interest, poorer selfcare, social anxiety and isolation. There is also a plausible connection between socioeconomic status and engagement in exercise (67, 68). In addition, many psychotropic agents have proved to increase sedation (69, 70). Reviews of the current knowledge have concluded that physical training offer both somatic and psychiatric relief in patients with mental illness (71). The biggest effect on anthropometric and biochemical outcomes was achieved when exercise was applied in addition to a dietary intervention, according to reviews from 2015 and 2021 (72, 73).

Programs with moderate to vigorous aerobic exercise seems to be especially useful in improving somatic co-morbidity in SMI patients (74, 75).

#### 1.5.3 Disordered eating behavior in SMI

Eating disorders include behavior that leads to excessive energy intake and weight gain, such as binge eating, emotional- and comfort eating, continuous snacking and eating at night (76). A recent meta-analysis concluded that while the lifetime prevalence of anorexia- and bulimia nervosa was 0.16% and 0.63% respectively, as much as 1.53% of the general population would suffer from a binge eating disorder (BED) during their life scope (77). The link between disordered eating behavior and SMI have previously been established (78, 79). The prevalence of bulimia and BED was assessed in 156 patients with an SSD. While no cases of bulimia were identified, 23% of the patients suffered from a BED (76). This aligns with evidence from the psychiatric ward of SMI patients, with both overeating and refusal of food being present (80, 81). Disordered eating behavior has proved to be closely related to the manifestation of positive symptoms in SSD-patients (81).

#### 1.5.4 Smoking, alcohol, and substance abuse in SMI

Known to be a causal risk factor for CVD, smoking is documented to be highly prevalent in persons with an SMI. SSD patients have been reported to be frequent smokers of tobacco, even more than other SMI patients (82). Also patients with BPD were found to smoke heavily compared to the general population (83). Furthermore, research of this matter has found that smoking is associated with increased suicidal risk, proving that there is a consistent relation between tobacco smoking and mental unhealthiness (84, 85).

Besides smoking (regular) tobacco, a Danish cohort study reported an alarmingly high rate of alcohol- and illegal substance abuse among patients with a BPD or SSD, compared to the average population (86). Several reports have also been made of increased risk for drug-overdose in BPD-patients compared to controls, and of increased rate of overdose-cases in persons experiencing psychosis (87). Suggestions have been made that the genetic determinants predisposing for SCZ also puts the individual at high risk for developing a substance use disorder (88).

A study from 2011 found that homeless drug abusers in Norway have a poor diet with high sugar consumption. This was reflected in biomarkers for high blood glucose (fasting glucose,

HbA1c) and elevated TG (89). Based on the body of literature, mental morbidity seems to be intertwined with abuse of tobacco, alcohol and illegal substances and inability to maintain an adequate diet, with no clear direction of causality.

#### 1.5.5 Metabolic side effects of AP-medication

Almost all types of SGA medication cause metabolic side effects and significant weight gain (34, 59). This is recognized by both receivers and providers of psychiatric treatment. SGA agents were reported to be associated with substantial weight gain, by Allison et al. already in 1999 (90). Their findings have been confirmed repeatedly in subsequent years (91). Reports of weight gain ranging from 0.5 to 15 kg have been made, and drastic increases in body weight are especially profound in drug-naïve patients (92). In addition to weight gain, AP-agents have proved to increase fasting blood glucose and cholesterol (93). SGA-agents like olanzapine, clozapine, risperidone, and quetiapine are often highlighted as AP-agents with profound weight gaining properties (33, 93). However, even SGAs with lower risk of weight gain have been found to increase body weight compared to placebo (91).

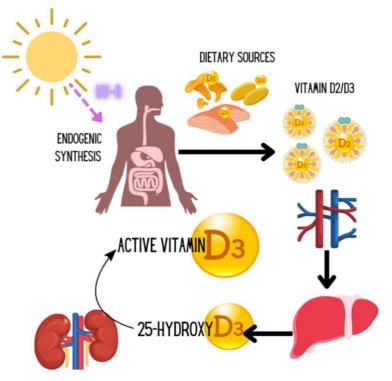
#### 1.5.6 Metabolic side effects of lithium

Mood stabilizing drugs are commonly prescribed for BPD, among them lithium (17). The window of treatment with lithium is, however, extremely narrow. Both acute and chronic intoxication are feared outcomes related to lithium medication. Adverse effects of lithium are hypercalcemia, hypothyroidism, weight gain, altered kidney function and nephrogenic diabetes insipidus (94). Weight gain affects 11-65% of patients prescribed with lithium, and approximately 20% will gain as much as 20 kg (95). Mechanisms for the weight increasing potential of lithium is not well understood, but theories regarding hypothalamic appetite regulation have been proposed (94).

#### 1.5.7 Vitamin D and SMI

Vitamin D is a pro hormone *and* vitamin that have dual function in the human body. As a hormone, it acts by binding to specific receptors regulating gene expression. As a vitamin, it is essential for absorption of calcium and maintain bone health (96).

Deficiency of vitamin D is common, especially in northern regions (97). This can be explained both by insufficient sunlight exposure to asset endogenic synthesis and limited dietary sources to vitamin D. Those that spend little time exposed to sunlight, have strong pigmentation or are obese are at increased risk for vitamin D deficiency (98).



**Figure 7:** Vitamin D can be synthesized in human skins cells upon exposure to ultraviolet radiation from the sun. There are few dietary sources to vitamin D, limited to fatty fish, fish oil and certain mushrooms. These are the main reasons for why a northern population is at higher risk for vitamin-D deficiency. Both liver and kidney are organs involved in enzymatic activation of vitamin D precursors to the active prohormone 1,25-hydroxy-vitamin D.

SMI patients have more obesity and a poorer diet compared to the general population (53, 60, 99), and are therefore at higher risk for insufficient vitamin D levels. A cross-sectional study found three-fold higher risk for vitamin-D deficiency among SCZ-patients compared to matched controls (100). A prospective cohort study from 2019 even found an association between high serum vitamin-D levels at baseline and less SCZ symptoms in first episode psychosis patients (101).

Clinical deficiency of vitamin D is prevalent when serum concentrations are <50 nmol/L (98). There is currently little consensus considering the optimal level of serum vitamin D. Absorption of calcium peaks when serum concentration reaches 80 nmol/ but current findings suggest that even higher levels of vitamin D are beneficial to prevent osteoporosis (102). Supplementation with vitamin D might be a safe and effective way to ensure adequate serum levels (103).

#### 1.6 Prevention of lifestyle-induced morbidity

A recent meta-analysis summarized the effect of lifestyle on the risk of ACVD (46). Diet, exercise, smoking and body weight has been identified as the four corner stones to intervene with to achieve better arterial health (97). The mentioned meta-analysis concluded that a moderate to high level of exercise was superior in risk reduction for ACVD. Interestingly, exercise and dietary quality was protective of ACVD risk through every category of BMI. In line with other findings, this suggests that beneficial lifestyle choices are preventive of ACVD regardless of initial body weight (46).

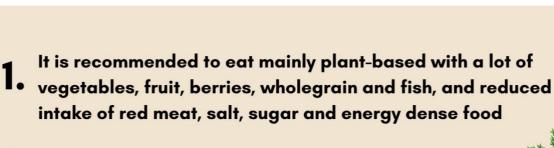
#### 1.6.1 Healthy dietary pattern

The preventive effect of healthy diet on development of CVD is well documented (104, 105). A typical healthy dietary pattern is recognized by high consumption of vegetables, fruit, grain, legumes, fish, and olive oil, a moderate intake of dairy and wine, and a low intake of red and processed meat, and other highly processed salty and sweet foodstuffs (104).

A health promoting diet may prevent ACVD by reducing plasma-TG and LDL-cholesterol, body weight, abdominal obesity, and blood pressure (BP) (106). Although any energy restricted diet can be a successful in reducing body weight, studies find that improvement of dietary quality can lead to beneficial health effects, even when weight loss is not primarily targeted (104).

#### 1.6.2 Norwegian Food Based Dietary Guidelines

The Norwegian Directorate of Health published updated dietary guidelines in 2011. These FBDG emerged from a thorough synthetization of current knowledge of the relationship between diet and health, conducted by leading scientists of the nutrition field in Norway (107). The Norwegian FBDG are listed in figure 8. Norwegian FBDG are mostly in accordance with other countries' current dietary recommendations. Most official FBDG in 2021 recommended a high intake of fruits, vegetables, wholegrain cereals, legumes, nuts and fish, a moderate intake of milk and dairy products, and a reduced intake of red and processed meat and sugary and salty food products (108). Water is the recommended beverage, and plant oil is proposed superior to dairy butter and hard margarine (109).



- 2. Keep a balance between energy intake and consumption
- 3. Eat 5 portions of fruit, vegetables and berries every day
- **4**. Eat at least 4 portions of whole grain every day
- 5. Eat fish equivalent to 2-3 dinner portions per week
- 6. Consume low-fat dairy products every day
- **7.** Eat lean meat, limit red and processed meat
- 8. Choose plant oil and soft margarine instead of butter and lard
- **9.** Water is recommended for drinking
- **10.** Limit the intake of added sugar
  - 1. Limit the intake of salt
- **12.** Dietary supplements might be useful to avoid deficiency in certain groups of the population
- **13.** 30 minutes of physical activity per day is recommended

**Figure** 8: Guidelines for improved health and reduced morbidity in Norway. A heavy body of sound data from epidemiological studies, randomized controlled trials and literature reviews constitutes the background of these guidelines.

#### 1.6.3 DIGIKOST-FFQ

The DIGIKOST food frequency questionnaire (FFQ) was developed to assess dietary quality, exercise level and tobacco use, and score this according to Norwegian recommendations for the respective health behaviors. The questionnaire aims to appraise the participants habitual food intake, with instructions stating that the participants should have the last month in mind when submitting the FFQ. A qualitative assessment of the usability of DIGIKOST found it to be easily interpreted, with an average time of 19 minutes spent from start to completion (110). Validation of the FFQ is currently submitted and will be published shortly. This validation found that DIGIKOST provided valid data on dietary intake, and was able to differentiate individuals' compliance to recommendations for diet and exercise (111).

# 1.7 Knowledge of Norwegian SMI-patients' lifestyle

Until recently, the association between diet, lifestyle and SMI had not been assessed in a Norwegian environment. Emma N. Johannessen was the first to report that SMI patients in Norway had lower compliance to FBDG compared to mentally healthy Norwegians, in her master thesis from 2022 (99). This master thesis was a unique Norwegian contribution to the science-based knowledge of the dietary habits of patients with an SMI.

# 1.7.1 Assessment barriers

The master student identified several challenges regarding nutritional assessment of SMI patients during her project conduction in 2021/22. Measurements of somatic health parameters were scarce, and weight, waist circumference, BP and blood parameters measuring glucose and lipids were rarely recorded in the patients' journals. This made identification of MetS unprecise, but a lot of the participants would probably sort into this diagnosis considering the high rate of obesity (99).

# 1.7.2 Findings from dietary assessment

The dietary assessment in 2021/22 revealed low to moderate compliance to Norwegian FBDG. SMI-patients in Asker had low adherence to FBDG regarding intake of fruit and berries, processed meat, sugary drinks and foods rich in sugar and salt. A large proportion of the participants also reported low adherence to recommendations for exercise (99).

### 1.7.3 Qualitative nuancing

During the conduction of the project, the master student reported several unintended qualitative observations. These entailed patient reports of severe weight gain following APmedication, and emotional difficulties related to eating. Secondly, Johannessen reported difficulties during recruitment and conduction, not because of unwillingness in participating patients, but due to the unaccustomedness of working with dietary health professionals in the mental health ward (99).

# **2.** Aim

# 2.1 Objective

The aim of this study was to further assess the diet and lifestyle of patients with an SMI diagnosis receiving treatment in Norway. The main goal was to determine compliance to Norwegian FBDG using the DIGIKOST-FFQ. Secondly, we wanted to determine the rate of MetS accurately, using new anthropometric measures and relevant blood samples. Thirdly, the SMI-patients' serum concentration of vitamins, micronutrients and liver enzymes were assessed to identify deviations and deficiencies. We also wanted to record nutrition- and lifestyle related beliefs and statements in a more structured way.

# 2.2 Hypothesis in the present thesis

- In the study population of patients with SMI, Adherence to Norwegian FBDG is thought to be lower than the general Norwegian population.
- In the study population of patients with SMI, Sedentary behavior is hypothesized to be more prevalent compared to the general Norwegian population.
- In the study population of patients with SMI, a high prevalence of MetS is thought to be found using relevant anthropometric measures and biomarkers.
- Procurement of new blood samples are hypothesized to identify deviant biomarkers for liver enzymes and micronutrients.

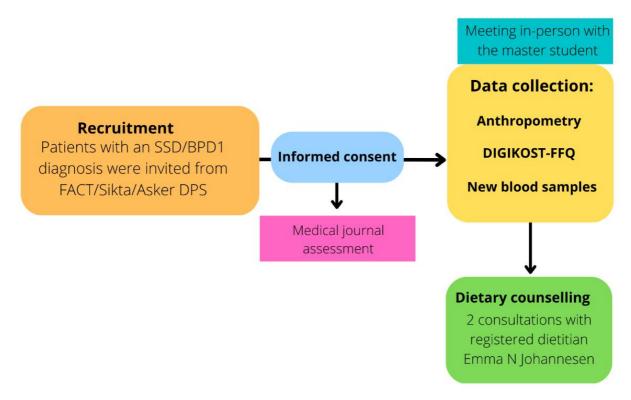


# 3. Materials and methods

The following section describe the data collection that took place in the mental care ward of Asker from 2022 to 2023. Details of materials and methods in the master project from 2021/2022 is thoroughly described in Emma N. Johannesen's thesis (99). This was a continuation of Johannesen's assessment, with improvement of methodology. ChatGTP was not utilized at any point of data collection or - handling in the study at hand, neither in the process of writing the manuscript of the thesis.

# 3.1 Study Design

We studied the dietary habits in patients with an SMI, receiving treatment in the Norwegian municipality of Asker. Alongside the nutritional assessment, updated anthropometric data and blood samples were obtained to get valid measuring points to determine the prevalence of MetS. Participants' statements and beliefs regarding nutrition and lifestyle were documented by the master student throughout the meetings. After assessment the patients were offered two dietary consultations with Emma N. Johannesen, who is now a registered dietitian. Figure 9 is a brief illustration of the study design.



**Figure 9:** Flow chart of the study design. After participants consented to perform, they were eligible for medical journal assessment and collection of anthropometric data, dietary habits measured with DIGIKOST, and requisition of blood samples.

# **3.2 Recruitment**

Recruitment was done in the mental care ward in Asker (figure 9). Patients from the regular outpatient clinic (Allmennpsykiatrisk poliklinikk), the FACT-section and the 24-hour inpatient section (Sikta) were invited. The latter two have locations in the same building in the municipality. The master student kept track on patients being hospitalized at Sikta and conferred the mental care providers whether the person at question was eligible for participation. If eligible, the master student invited the patient to participate in the study. A written or oral consent, collected either by mental care providers or the master student, enabled the master student to contact the patient if they were discharged prior to data collection.

# 3.2.1 Inclusion Criteria

Patients aged 18 or more with an SMI receiving therapy in the mental care ward in Asker were qualified to participate in the trial. Patients with BPD 1 or an SSD were mainly targeted, but any SMI with AP or mood stabilizing medication prescribed, deemed the patient eligible for participation. Health professionals working in the mental care ward would asset with consideration of patient's ability to participate, depending on their mental state and phase of the illness. Patient enrolled to involuntary treatment were not eligible to participate.

# **3.3 Data collection**

Patient data emerged from medical journals and measurements done at the patients' meeting with the master student. At this meeting, participants were asked to give written consent to participate, if this was not attained priorly (appendix 3). A short oral introduction was given to the study design, implications of participating and the purpose of the study.

# 3.3.1 Journal assessment

Patients that consented to participate had their journals assessed by the master student. This was done in the mental care facilities in Asker, using the digital journal system DIPS. Information about psychotropic medication and diagnosis was attained from medical journals before the patient meeting.

#### 3.3.2 Anthropometric measures

Height was self-reported if the participant knew how tall they were. Body weight was measured using a *Health Select* personal electronic scale. The participants were asked to wear light clothing and no shoes. Body mass index (BMI) was calculated by formula:

$$BMI = \frac{kg}{m^2}$$

Waist circumference (WC) was measured using a measuring tape. WC was recorded by the master student twice, and the mean value was calculated. BP measurement was obtained using a *Microlife* 'BP2 Basic' BP monitor.

The sample from 2021/22 had anthropometric measurements collected by different methods. None of the participants had WC measurements, but abdominal fatness was assumed in cases where BMI exceeded  $30 \text{kg/m}^2$ . If recorded, data on BP derived from patient journals (99).

#### 3.3.3 DIGIKOST-FFQ

Participants used personal ID keys to access the digital FFQ and were briefed on its use by the master student. The student supervised the submission to prevent any misunderstandings, remaining neutral if participants did not seek clarification. The entire DIGIKOST-FFQ is found in appendix 5.

#### 3.3.4 Biochemistry and blood samples

All participants provided new blood samples after anthropometric measures and DIGIKOST submission (table 1). Doctors at FACT and Sikta requisitioned the samples from Bærum or Blakstad hospital. Out-clinic patients submitted samples before breakfast. Patients with frequent blood sampling for AP-medication measured the parameters at their next scheduled sampling. Missing parameters were added in the next requisitioned panel with consultation from the responsible doctor.

#### 3.3.5 Observations and statements from meetings

Important qualitative statements and observations were expected to emerge during patient meetings, based on the reports from the previous assessment (99). Before patient meetings

#### **Table 1: Blood parameters**

Blood parameter	Unit
Hemoglobin	g/dl
Ferritin	μg/L
ASAT	U/L
ALAT	U/L
Fasting Glucose	mmol/L
HbA1c	mmol/mol
Fasting TG	mmol/L
Total Cholesterol	mmol/L
LDL	mmol/L
HDL	mmol/L
Folate	nmol/L
Vitamin-D	nmol/L

Table 1: 12 blood parameters were<br/>requisitioned for biochemistry<br/>assessment. TG; Triglycerides, LDL;<br/>Low Density Lipoprotein, HDL; High<br/>Density Lipoprotein.

began, the master student created a journal to record significant events. This journal contained anonymous statements about experiences with diet, exercise, and body weight, as well as reflections and emotions related to the topic. The purpose was to add qualitative detail to the master thesis, so the observations were divided and quantified into domains based on emerging themes.

#### 3.4 Dietary counselling

The participants were offered dietary counselling as a recognition for participating in the project. The offer entailed two meetings with registered dietitian Emma N. Johannesen, who has experience of working with SMI patients. Participants were able to see the dietitian in the evening, at two optional locations connected to the mental care facilities in Asker (Sikta/FACT and Asker DPS main location "Skysstasjonen"). The consultation could also be carried out by phone if the patient did not wish to meet in person. Dietary counselling was entirely voluntary, and the participants were informed that they were free to decline or cancel the consultation meetings if it felt overwhelming or invading.

#### 3.4.1 Journal from dietary consultations

The dietitian also kept journal notes from the patients' consultations. Each journal note entailed information about who was present for the consultation, where it took place, and the occasion and purpose for the meeting. They also comprised information about the patient's nutrition literacy, beliefs, and motivation for lifestyle change. Journal notes were apprised by the master student and included in the exhibition of qualitative statements.

#### 3.5 Data handling and storage

Patient data was stored in Services for Sensitive Data (TSD), operated by the University of Oslo. The master student was granted access to TSD at the study initiation in august 2022. Anthropometric, biochemistry and dietary data from Emma N. Johannessen's assessment in 2021/22 was stored in in the same TSD-server (99). When data collection was finalized in January 2023, the data from and the current sample was merged with the previous assessments' data and analyzed together.

### 3.5.1 Calculation of DIGIKOST score

Data from DIGIKOST submission were used to determine the participants compliance to Norwegian FBDG. Table 2 is constituted of the determinants and cut-off values used to categorize a participant in groups of 'Low', 'Moderate', or 'High' compliance to FBDG.

FBDG	Low Moderate		Hi	igh		Points			
Fruit and berries $\tau$	<	125	125-250		>250		0	1.5	3
Vegetables	<	125	125-	250	>2	250	0	1.5	3
Whole grains (F M) $\omega$	<35	<45	35-70	45-90	>75	>90	0	1.5	3
Unsalted nuts (OW NW) $\boldsymbol{\xi}$	<10	30>, <10	10-20	10-20	>20	20-30	0	0.5	1
Fish	<	21.5	21.5	5-43	>4	43	0	0.5	1
Low fat dairy	<	<50	50-	100	>1	.00	0	0.5	1
Cooking fat $\psi$	Mainly	saturated	A comb	vination	Mainly u	nsaturated	0	0.5	1
Red meat	>	-71	35.5	5-71	<3	5.5	0	0.5	1
Processed meat	>	>20	10-	20	<	10	0	0.5	1
Sugar and fat	>	20	10-	20	<	10	0	0.5	1
Sugary drinks	>	20	10-	20	<	10	0	0.5	1
Supplements	>0	units	N/	Ά	0 u	nits	0	0.5	1
Total FBDG score	0 t	o <7	7-3	13	13	-18	<7	7-13	>13

# **Table 2: DIGIKOST Diet Score Calculation**

Table 2: Calculation of DIGIKOST-score, divided into low, intermediate, or high adherence to Norwegian FBDG according to intake (g/day) of 12 food groups. τ: Including 200ml of juice per day. ω: Distinction between intake recommendation for women and men, respectively. F: Female, M: Male. ξ: Distinction between intake recommendation for individuals <25kg/m<sup>2</sup> or <. OW: Overweight, NW: Normal Weight. ψ: Healthy choices deemed as soft margarine or plant oil, butter, and hard margarine as unhealthy.</p>

Based on the total adherence to Norwegian FBDG, and guidelines for BMI, exercise, alcohol intake and tobacco smoking, a 'Lifestyle Index'-score was generated (table 3). A score of 5 indicates almost perfect adherence to national guidelines for a healthy lifestyle.

Lifestyle determinant	Low	Intermediate	High	Lifestyle-index score		score
Diet	<7	7-13	>13	0	0.5	1
Body weight	$<18.5 \text{ kg/m}^2>30$	25-29.9 kg/m <sup>2</sup>	18.5-24.9 kg/m <sup>2</sup>	0	0.5	1
Physical Activity	<75min/week	75-149.5min/week	>150min/week	0	0.5	1
Tobacco	Smoking	-	Non-smoking	0	-	1
Alcohol	>4.29g/d	0-4.29g/d	0g/d	0	0.5	1

 Table 3: Calculation of Lifestyle Index based upon five domains; diet (DIGIKOST-score), body weight within a healthy BMI, physical activity level within the recommended range, and tobacco- and alcohol consumption.

 Total Lifestyle-index range from 0-5.

# 3.6 Statistical analysis

All statistical analysis was conducted using IBM SPSS Statistics Version 28.0.0.0 (SPSS Inc. Chicago). Presentation of data and choice of statistical tests was according to data distribution.

# 3.6.1 Test of normality

Data distribution was assessed by appraisal of histograms, Q-Q-plots, and boxplots in SPSS. Shapiro-Wilks test was performed to detect non-normality, as the study sample size was <50 (112). Skewed data was reported as median with 25- and 75-percentiles, and normal data was reported as mean value with standard deviation (SD).

# 3.6.2 Descriptive statistics

Characteristics of the study population were analyzed and presented in groups of diagnosis and/or gender. If the data was normally distributed, Student's t-test was performed to determine difference between groups. If data was skewed, Mann-Whitney U-test was conducted instead. Significance level was set to p<0.005 for both tests.

# 3.6.3 Determination of MetS and nutritional deviations

Dummy-variables were created in SPSS for each state of metabolic deviation; 'elevated TG', 'elevated glucose', 'low HDL', 'Abdominal obesity', and 'hypertension.' This was also done for 'MetS' and 'Vitamin-D deficiency'. Difference between groups of gender and diagnosis was appraised with Fishers' Exact test.

# 3.6.4 Analysis of dietary intake and exercise

Dietary intake was analyzed as absolute intake, defined as grams per day of 10 food groups and as compliance to Norwegian FBDG for the given food group (table 2). Intake of fat sources and taking dietary supplements was defined as compliant with the recommendations or not. Differences in dietary intake were analyzed with Mann Whitney U-test, and Fishers exact test to identify distinction between groups of gender and diagnosis in low, moderate, and high compliance to FBDG. Adherence to FBDG about intake of whole grain was analyzed separately for men and women, and intake of unsalted nuts separate for individuals with BMI< or >25 kg/m<sup>2</sup>, because of distinction in the FBDG for these respective groups. Total time spent sedentary and active (moderate/high intensity) was analyzed separately, and groups of gender and diagnosis were tested for differences within categories of low, intermediate, or high compliance to recommendations for exercise, using Fisher's Exact test.

#### 3.6.5 Analysis of tobacco and alcohol consumption

Tobacco consumption was analyzed as categories of 'never used, 'previously used' and 'currently using' for both snus-tobacco and cigarette smoking. The latter category included both regular and occasional consumption of tobacco.

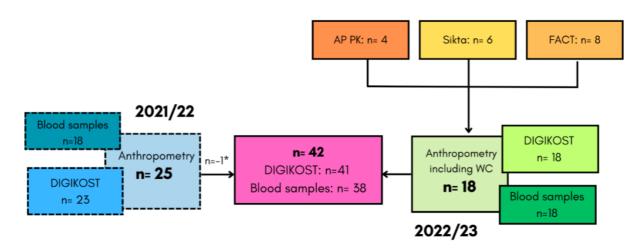
Variables of alcohol consumption is generated in DIGIKOST, calculated from intake of alcoholic beverages (beer, wine, and liquor) into total ethanol per day. To distinguish between high, moderate, and non-consumers of alcohol, dummy variables were created for alcohol consumption >4.26 g/day (high), 0-4.29 g/day (moderate) and 0 g/day (low). These cut-offs yielded 0, 0.5 or 1 point in the lifestyle index, respectively.

Differences in alcohol- and tobacco consumption was tested for in groups of diagnosis and gender using Fisher's exact test.

# 4. Results

42 participants were recruited from Asker DPS between 2021 and 2023. Figure 10 provides a summary of which department in Asker DPS n participants were recruited from, and how many participants that had blood samples, anthropometric measures, and DIGIKOST-submissions available for analysis.

**Figure 10:** Flow Chart of recruitment in 2021/22 and 2022/23. Description of obtained data from n participants. \*One participant that already was enrolled in 2021, was later identified again in the 2022/23. The latter participation was deemed the most valid one in terms of completeness of biochemistry, anthropometric measures and DIGIKOST submission. This participant was thus ruled out from the 2021/22 sample. AP PK; Allmennpsykiatrisk Poliklinikk, FACT; Flexible Assertive Community Treatment.



# 4.1 Characteristics and anthropometry

19 women and 23 men were recruited. Half of the participants were diagnosed with SCZ or an

SSD, and the other half with BPD type 1. Statistics is presented in table 4.

Table 4:	Charac	teristics	of	participants
----------	--------	-----------	----	--------------

Total participants n=42					
Gender n (%)Diagnosis n (%)Medication n (%)					ation n (%)
Female	Male	SCZ/SSD	BPD	WGA	Not WGA
19 (45.2)	23 (54.8)	21 (50)	21 (50)	27 (64.3)	15 (35.7)

**Table 4:** Characteristics of 42 participants recruited in Asker from 2021-23. SCZ; Schizophrenia, SSD;Schizophrenia Spectrum Disorder, BPD; Bipolar Disease.

# 4.1.1 Prescribed medication

At the time of data collection, 27 (64.3%) participants were prescribed with AP- or mood stabilizing medication known to have medium to high risk of weight gain. Some patients had prescriptions of several psychotropic agents in combination. Table 5 provides insight in how many patients that were prescribed with AP-agents with known high risk of weight gain; Olanzapine, Clozapine, Paliperidone and Quetiapine (113). Eight (19%) participants were

prescribed with lithium, and 7 (16.7%) with AP medication like aripiprazole, brexpiprazole or other psychotropic agents with low risk of weight gain (59). One participant did not take any medication at the time of participation.

Psychotropic agent	n (%)
Olanzapine $\Omega$	11 (26.2)
Clozapine $\Omega$	5 (11.9)
Paliperidone $\Omega$	2 (4.8)
Quetiapine $\Omega$	14 (33.3)
Lithium $\pi$	8 (19.0)
Lamotrigine $\lambda$	10 (23.8)
Other $\alpha\Omega\lambda$	10 (23.8)
None	1 (2.4)

Table 5: Psychotropic agents prescribed in study population.

**Table 5:** The most frequently prescribed weight gain-associated agents listed with n (%) participants prescribed with respective drug.  $\alpha$ : First-Generation antipsychotic agent,  $\Omega$ : Second-Generation antipsychotic agent,  $\pi$ : Mood stabilizing agent,  $\lambda$ : Other psychotropic agents; anti-epileptics, anti-depressive- and anxiolytic agents.

### 4.1.2 Age and anthropometry

All participants had available data for age, weight, height, and BMI. 34 (80.9%) participants were weighed on site, while 8 (19%) of the participants' weight was self-reported or obtained from the patients' journal. 18 (42.8%) participants had WC measured on site and 34 (80.9%) patients had BP measured on site or previously. Women had significantly lower height, body weight and BPD than men (p<0.001, p=0.05, p=0.015). There was no significant difference in anthropometry between diagnostic groups. Table 6a and 6b provides information of age and anthropometry as mean with SD in groups of gender and diagnosis.

		Mean ± SD		
	Total (n=42)	Male (n=23)	Female (n=19)	p***
Age (years)	41.5±12.1	41.0±12.0	42.1±12.6	0.79
Height (cm)	174±11.0	181±0.9	165±0.7	< 0.001
Weight (kg)	90.6±17.6	95.3±16.4	84.8±17.6	0.05
BMI (kg/m <sup>2</sup> )	29.9±5.4	29.0±3.9	30.9±6.8	0.25
WC (cm)*	106.2±12.8	108.6±13.6	103.2±11.9	0.39
BPS (mmHg)**	132.0±17.3	135.6±18.0	128.4±16.4	0.23
BPD (mmHg)**	83.7±9.8	87.7±9.6	79.6±8.6	0.015

#### Table 6a: Age and anthropometry

**Table 6a:** Age and anthropometry of men and women. reported as means with std. deviation. \*Participants with WC measured: n=18 (42.8%). SD; Standard Deviation \*\*Participants with BP measured: n=34 (80.9%). \*\*\*P-value for statistical difference between groups of gender. Independent samples T-test. Significance-level p<0.05

	Mean	: SD	
	SSD (n=21)	BPD (n=21)	p**
Age (years)	40.1±11.7	43.0±12.7	0.44
Height (cm)	173±1.0	175±1.1	0.44
Weight (kg)	90.5±16.0	90.7±19.4	0.97
BMI (kg/m <sup>2</sup> )	30.4±5.7	29.3±5.2	0.53
WC (cm)*	105.4±12.9	107.2±13.5	0.78
BPS (mmHg)**	128.1±15.7	137.0±18.5	0.14
BPD (mmHg)**	82.5±9.1	85.2±10.8	0.43

Table 6b: Age and anthropometry in diagnostic groups

**Table 6b**: Age and anthropometry of diagnostic groups, reported as means with std. deviation. \*Participants with WC measured: n=18 (42.8%), SD; Standard Deviation \*\*Participants with BP measured: n=34 (80.9%), \*\*\*P-value for statistical difference between groups of gender. Independent samples T-test. Significance-level p<0.05

#### 4.1.3 BMI and central obesity

The mean (SD) BMI was 29.0 (3.9) kg/m<sup>2</sup> for men and 30.9 (6.8) kg/m<sup>2</sup> for women (p=0.25). One (2.4%) participant was classified as underweight, while 4 (9.52%) participants were within the normal range of BMI. Eighteen (42.9%) were overweight, and 19 (45.2%) were classified as obese (Figure 11). Within the obese participants, 12 (28.5%) had obesity degree I, 5 (11.9%) degree II and 2 (4.7%) degree III (Figure 11). 24 participants (57%) were deemed abdominally obese. Out of the participants with abdominal obesity, 10 (41.6%) were in this category based on having BMI > 30 kg/m<sup>2</sup>, while 14 (58.3%) exceeded the criteria for abdominal obesity measured by WC (57).



**Figure 11**: Distribution of participants (n=42) across BMI categories: underweight (<18.5 kg/m<sup>2</sup>), normal (18.5-24.9 kg/m<sup>2</sup>), overweight (25-29. 9 kg/m<sup>2</sup>), obese degree I (30-34.9 kg/m<sup>2</sup>), obese degree II (35-39.9 kg/m<sup>2</sup>), and obese degree III (≥40 kg/m<sup>2</sup>). Mean (Standard Deviation) BMI was 29.9 kg/m<sup>2</sup> (5.4).

#### **4.2 Biochemistry**

Seventeen (40.4%) of the participants had new blood samples drawn and analyzed after meeting the master student. The remaining participants were assessed for blood sample values deriving from previous testing. These values were, if ever analyzed, found in the medical journal. The number of participants missing values of each parameter, and the average values with SD are reported in table 7.

	Mean (± SD	)) or Median (25%, 75%	<b>)</b>	
Parameter	MV (%)*	Men	Women	p**
Hemoglobin (g/dl)	5 (11.9)	15.3±1.8	13.7±1.1	< 0.001
Ferritin (µg/L)	12 (28.5)	194.5±117.9	68.0±40.2	< 0.001
ASAT (U/L)	15 (35.7)	25.6±10.12	22.6±7.0	0.405
ALAT (U/L)	2 (4.7)	32.0 (20.0, 63.0) <sup>1</sup>	22.4±6.7	0.022
HbA1c (mmol/mol)	9 (21.4)	35±3.2	36.0 (34.0, 38.5) <sup>1</sup>	0.309
f-Glucose (mmol/L)	5 (11.9)	5.4±0.7	5.2 (4.8, 5.6) <sup>1</sup>	0.729
f-TG (mmol/L)	7 (16.6)	1.9±0.9	1.9±1.3	0.919
Tot-Ch (mmol/L)	4 (9.5)	5.0±0.9	5.0±0.9	0.892
LDL-Ch (mmol/L)	6 (14.3)	3.6±0.7	3.5±0.8	0.556
HDL-Ch (mmol/L)	4 (9.5)	1.2±0.4	1.2±0.3	0.433
Non-HDL-Ch (mmol/L)	4 (9.5)	3.8±0.9	3.8±1.0	0.834
Folate (nmol/L)	17 (40.5)	$11.5 (10.2, 26.0)^1$	17.8 (11.3, 21.3) <sup>1</sup>	0.347
Vitamin D (nmol/ml)	13 (30.9)	79.7±38.3	75.8±17.1	0.731

#### Table 7: Biochemistry values

Table 7: Biochemistry from blood samples, presented as mean value with std. deviation when normallydistributed, and <sup>1</sup>median with 25- and 75-percentiles when skewed. \*Participants (%) missing values (MV) forthe given blood parameter. \*\*Difference between groups were analyzed using Student's t-test if data werenormally distributed, and Mann-Whitney U-test if data were skewed in one or both groups. Significance levelwere set to p<0.05. SD; Standard Deviation, MV; Missing Values, TG; Triglycerides, Ch; Cholesterol, LDL;</td>Low Density Lipoprotein, HDL; High Density Lipoprotein.

#### **4.3 Prevalence of MetS**

Twenty-one (50%) participants, 52.2% of the men and 47.4% of the women met the diagnostic criteria's for MetS. Conversely, 57.1% of SSD- and 42.8% of the BPD patients were deemed afflicted with MetS. The most fulfilled criteria for MetS were abdominal obesity, whereas 52.2% of the men and 63.2% of the women were abdominally obese (p=0.542). In both groups of SSD and BPD, 57.1% of the patients had abdominal obesity (p=1.00). Fulfillment of diagnostic criteria and proportion of participants with MetS are listed in table 8a and 8b, for groups of gender and diagnosis, respectively.

	Prevalence of	of MetS (%) by gender		
	Men n=23 (%)	Women n=19 (%)	p*	<b>Total</b> n=42(%)
Abdominal obesity	12 (52.2)	12 (63.2)	0.542	24 (57.1)
Low HDL	6 (26.1)	11 (57.9)	0.048	17 (40.5)
Hypertriglyceridemia	11 (47.8)	6 (31.6)	0.315	17 (40.5)
Hyperglycemia	6 (26.0)	5 (26.3)	1.00	11 (26.2)
Hypertension	11 (47.8)	9 (47.4)	0.728	20 (47.6)
MetS	12 (52.2)	9 (47.4)	1.00	21 (50.0)

#### Table 8a: Metabolic Syndrome in groups of gender

 Table 8a: Prevalence of MetS in groups of gender. Availability of data is presented in table 7 \*Statistic significant difference between groups identified with Fishers' Exact test, p<0.005. HDL; High Density Lipoprotein, MetS; Metabolic Syndrome</th>

#### Table 8b: Metabolic Syndrome in groups of diagnosis

	Prevalence of M	letS (%) by diagnost	ic group	
	SSD n=21(%)	BPD n=21 (%)	p*	Total n=42(%)
Abdominal obesity	12 (57.1)	12 (57.1)	1.00	24 (57.1)
Low HDL	9 (42.8)	8 (38.0)	1.00	17 (40.5)
Hypertriglyceridemia	9 (42.8)	8 (38.0)	1.00	17 (40.5)
Hyperglycemia	7 (33.3)	4 (19.0)	0.495	11 (26.2)
Hypertension	9 (42.8)	11 (52.4)	0.171	20 (47.6)
MetS	12 (57.1)	9 (42.8)	0.538	21 (50.0)

**Table 8b:** Prevalence of MetS in groups of diagnosis. Availability of data is presented in table 7 \*Statisticsignificant difference between groups identified with Fisher' Exact test, p<0.005. SSD; Schizophrenia Spectrum</td>Disorder, BPD; Bipolar Disorder, HDL; High Density Lipoprotein, MetS; Metabolic Syndrome

#### 4.4 Vitamin D

Twenty-nine (69.0%) of the SMI-patients had available analysis of serum Vitamin-D concentration (table 8). Five (17.3%) of these participants proved to have low serum levels of vitamin D (<50 nmol/L). Nineteen (65.5%) of the participants had serum concentrations < 80 nmol/L. There was no statistically significant difference between vitamin D insufficiency in groups of gender, diagnosis, or abdominal obesity. No adverse findings were made on other micro-nutrients that were measured. Table 9 presents the number of participants within each category of vitamin D-status.

Vitamin D status	Concentration	n (%)
Deficiency	<50 nmol/L	5 (17.3)
Suboptimal level	50-79 nmol/L	14 (48.2)
Optimal level	>80 nmol/L	10 (34.5)

Table 9:	Vitamin-D	status
----------	-----------	--------

**Table 9:** Participants within categories of vitamin D status

#### 4.4.1 Vitamin-D supplementation

Fifteen (36.6%) of the participants reported supplementation with vitamin D in DIGIKOST, while 2 (4.9%) supplemented multi-vitamin containing vitamin D. Twenty-four (58.5%) of the participants took no vitamin D supplement.

## 4.5 Intake of 10 food groups

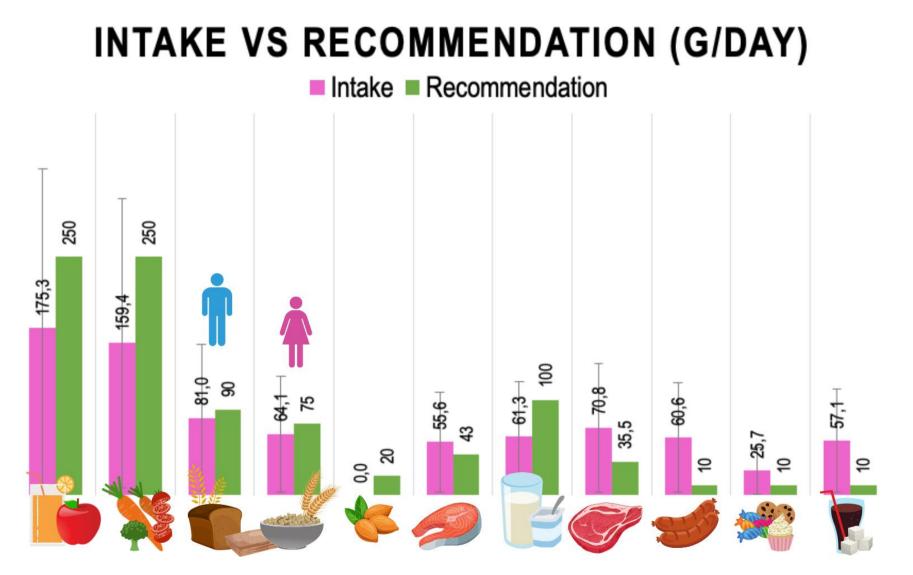
Intake of every food group was statistically equal in both groups of gender and diagnosis. Conclusively, the study populations median intake did not meet intake recommendations for fruit and berries (70.1% of recommended intake), vegetables (63.6%), whole grain (85.1 and 90% for women and men, respectively), unsalted nuts (0%) and low-fat dairy (61.3%). Intake of fish was sufficient (129.3%), while intake of red meat (199.4%), processed meat (606%), foods rich in sugar and fat (257%) and sweet beverage (571%) exceeded the recommendation. Table 10 and figure 12 presents and visualizes the median intake relative to the Norwegian recommendations for 10 food groups.

Food Group	Median intake	e (25, 75)-percentile	% of recommendation
Fruit and berries $\tau$	175.3 (	81.0, 274.1)	70.1
Vegetables	159.4 (	87.3, 285.5)	63.6
Whole grain (F M) $\omega$	64.1 (42.6, 83.7)	81.0 (38.8, 140.5)	85.1   90.0
Unsalted nuts $\xi$	0.0	(0.0, 4.2)	0.0
Fish	55.6 (	20.6, 91.5)	129.3
Low fat dairy	61.3 (	5.0, 298.0)	61.3
Red meat	70.8 (2	28.8, 110.8)	199.4
Processed meat	60.6 (	27.5, 87.4)	606.0
Sugar and fat	25.7 (	13.6, 68.2)	257.0
Sugary drinks	57.1 (	0.0, 142.8)	571.0

#### Table 10: Intake of 10 food groups

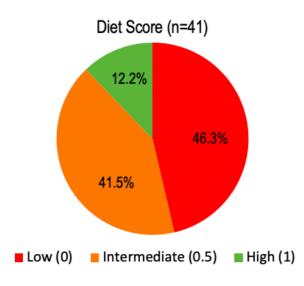
**Table 10:** Median (25, 75)-intake of 10 food groups in grams/day.  $\tau$ : Including 200ml of juice per day.  $\omega$ :Distinction between intake recommendation for women vs men.  $\xi$ : Intake of unsalted nuts was similar in both<br/>groups > and <25 kg/m<sup>2</sup>. F; Female, M; Male.

Figure 12: Median intake (95% CI-error bars) of 10 food groups compared to Norwegian food based dietary guidelines for respective food groups.



#### 4.6 Lifestyle Index

The following section will conclude the domains that the Lifestyle Index score was based upon. The conclusive Lifestyle Index for this population is presented in table 12a and 12b.





<u>4.6.1 Adherence to FBDG (Diet-score)</u> Nineteen (46.3%) of the patients had low compliance to Norwegian FBDG, 17 (41.5%) were deemed moderately compliant and 5 (12.2%) had high compliance (figure 13). A comprehensive presentation of scoring within each FBDG is in table 11.

**Figure 13:** Distribution of 41 participants within low, moderate, or high total compliance to FBDG. Low diet equals score <7, Moderate equals 7-13, and High score >13.

The median (25, 75%) Diet Score in the population was 7.5 (5.5, 12.5) and median (25, 75%) Lifestyle Index score for diet was 0.5 (0.0, 0.5). No difference was detected in terms of diet score or compliance to FBDG in groups of gender (p=0.275, p=0.561) or diagnosis (p=0.781, p=0.791).

#### **Dietary supplements**

Taking dietary supplements gave a score of 0, while restraining from supplements provided 1 point. Twenty-seven (65.9%) of the patients scored 0 points, taking 1 or more supplements daily, while 14 (34.1%) reported no supplementation and got 1 point in the DIGIKOST Diet-score. No difference between groups of gender or diagnosis were detected.

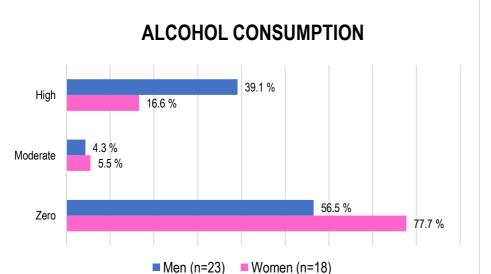
FBDG	Low (%)	Moderate (%)	High (%)	Points
Fruit and berries	41.5	24.4	34.1	1.5
Vegetables	36.6	36.6	26.8	1.5
Whole grains	24.4	26.8	48.8	1.5
Unsalted nuts	82.9	7.3	4.9	0
Fish	29.3	9.8	61.0	1
Low fat dairy	39.0	17.1	43.9	0.5
Cooking fat	19.5	26.8	53.7	1
Red meat	48.8	22.0	29.3	0.5
Processed meat	82.9	2.4	14.6	0
Sugar and fat	58.5	17.1	24.4	0
Sugary drinks	56.1	-	43.9	0

**Table 11: Compliance to Norwegian FBDG** 

**Table 11:** Distribution of the participants' compliance to FBDG within categories of low, moderate, and high compliance. Table 2 provides the scoring system for each FBDG. 11 FBDG were included. <sup>1</sup>Median points yielded for this FBDG, FBDG; Food Based Dietary Guidelines.

#### 4.6.2 Tobacco and alcohol

Eight (19.5%) participants reported to be daily smokers, while 3 (7.3%) smoked occasionally. 11 (26.8%) were previous smokers, while 19 (46.3%) reported to never have smoked daily or occasionally. Seventeen (41.5%) of the participants were current snus consumers, either daily or occasionally. Twenty-four (58.5%) had never used snus, while no participants reported to be previous snusers.



#### Figure 14: Distribution of alcohol consumption

**Figure 14:** The charts visualize distribution of 23 men and 18 women within categories of zero (no alcohol), moderate (0-4.29 g ethanol/d) and high (>4.29 g ethanol/d) alcohol intake, based on their reported alcohol habits in DIGIKOST.

More than half of the participants (65.8%) reported to not drink any alcohol. One woman and 1 man (4.8%) had a moderate alcohol intake between 0-4.29 g ethanol /d, while 3 women and 9 men (29.3%) reported an intake of alcohol that corresponded to high consumption, with more than 4.29 g ethanol/day. No statistical difference was identified between groups of gender nor diagnosis. Figure 14 visualizes the distribution of participants within groups of alcohol consumption.

#### 4.6.3 Exercise and sedentary time

The median (25-, 75%) time of physical activity per week was 119.9 (36.5, 226.2) minutes. Median (25-, 75%) time with moderate intensity exercise was 70.6 (36.5, 189.6) minutes/week, and 0.0 (0.0, 21.1) minutes/week with high intensity. Median time spent sedentary per day was 8 (5, 10) hours.

Of all participants, 44% were categorized as highly compliant with Norwegian guidelines for physical activity (>150 min/week). Intermediate compliance yielded 15% (15-149.5 min/week) of the participants, and 41% of the participants scored low (<75min/week).

#### 4.6.4 Health Index Score

The median health index in the population was 2.5 (2, 3). Median score within each domain and conclusive health index is found in table 12a. Distribution of participants across the health index scoring scale (0-5 points) is shown in table 12b: 19.7% scored 1.5 point or lower, 53.6% scored 2-3 points and the remaining 29% scored between 3.5 and 4 points. None of the participants scored above 4.

Table 12a:	Health	index	scoring
------------	--------	-------	---------

Lifestyle domain	Points (25, 75%)
Diet	0.5 (0, 0.5)
Body weight	0.5 (0, 0.5)
Exercise	0.5 (0, 1)
Tobacco	0 (0, 1)
Alcohol	1 (0, 1)
Health Index	2.5 (2, 3)

**Table 12a:** Median conclusive health index within each lifestyle domain with 25- and 75-percentiles

#### 4.7 Qualitative assessment

Health Index	n (%)
0	3 (7.3)
0.5	2 (4.9)
1.0	0 (0)
1.5	5 (12.2)
2.0	6 (14.6)
2.5	10 (24.4)
3.0	6 (14.6)
3.5	7 (17.1)
4.0	2 (4.9)

#### Table 12b: Distribution of Health Index score

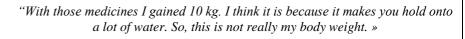
**Table 12b:** Distribution of 41 participants across thehealth index scoring scale. 0-1.5 points deemed lowscoring, 2-3 points as intermediate and 3.5 points or moredeemed high.

0(0)

4.5-5.0

Twenty-seven statements and observations

emerged during participant meetings. The statements are retold in table 13a-13d, categorized in five separate domains (n, %): Side effects of AP medication (9, 33.3%), Barriers during hospitalization (2, 7.4%) Nutrition Literacy (7, 25.9%), Disordered eating behavior (3, 11.1%), Perception of lifestyle and health (6, 22.2%).



"It was almost like the Zyprexa melted on my tounge, and right after I could find myself devouring an entire loaf of bread.»

"They make us take all kinds of stuff here. I think it is scary because they (mental care providers) know about the side effects, but we, who are the ones to take those things (AP-medicine), don't.

".. I lost 14 kg the past months but regained 2 kg the past two weeks. When I'm depressed, I have the worst appetite and lose weight rapidly. But now! The last week my appetite has gone through the roof, I can hardly believe how much I find myself eating. 2 sandwiches and 4 crispbreads for breakfast! » (This patient has recently been prescribed with Olanzapine)

"Zyprexa (Olanzapine), oh that is the witchcraft-medicine."

"I gained 20 kg on that medicine (Quetiapine)"

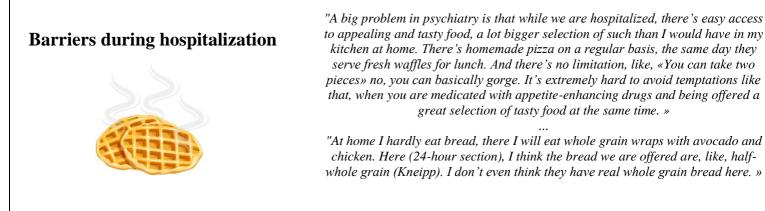
"The worst AP medicine (Olanzapine) makes you incredibly hungry. You can eat two enormous portions of dinner and be just as hungry shortly after."

Patient NN talks briefly about gaining a lot of weight after initiation of APagents.

"I used to be a very active person and were in the gym training several times per week. On medicines (Quetiapine) I have turned way more drowzy and indifferent. I don't wake up in the morning like 'ding!' anymore, awake. That's the hardest side effect of the medicines to deal with. I turn indifferent.»

 Table 13a:
 9 statements emerged regarding experiences patients had with AP medication and nutrition related side effects and/or weight gain.

#### Table 13b: Barriers during hospitalization

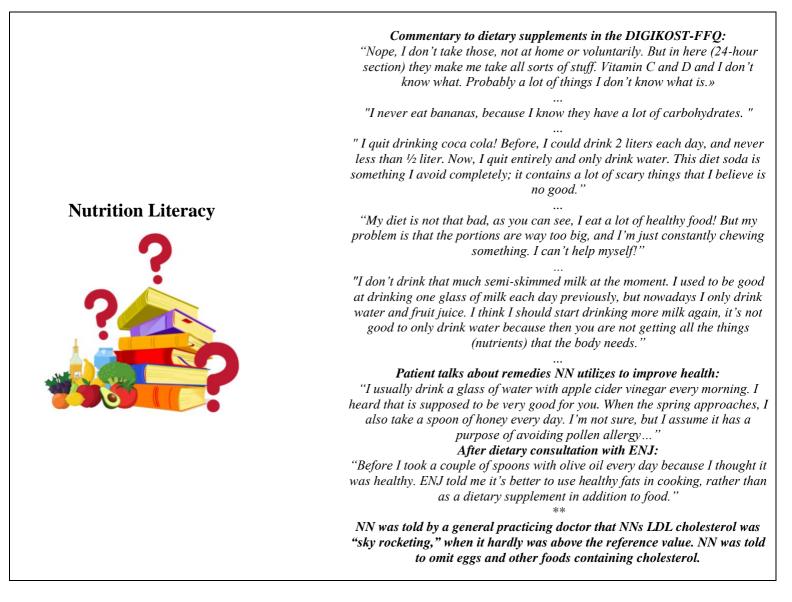


**Table 13b:** 2 statements were made on barriers patients experienced during hospitalization in the mental ward.

#### Side effects of AP medication



#### **Table 13c: Nutrition Literacy**



**Table 13c**: 7 statements were categorized as a manifestation of patients' nutrition literacy as well as the knowledge of nutrition among professionals in the health ward. ENJ; Emma N. Johannessen, registered dietitian.

#### Table 13d: Eating disordered behavior

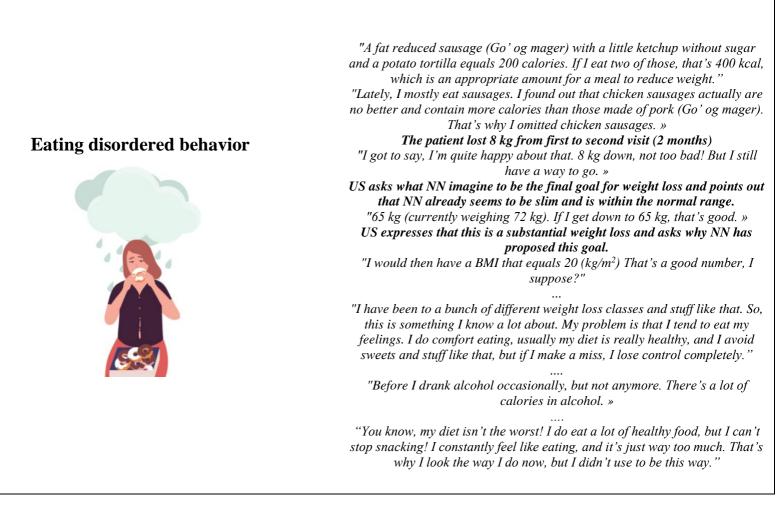


 Table 13d: 4 single statements were recognized as signs of a troubled relationship with food and/or body image.

 US; Undersigned.

#### Table 13e: Perception of lifestyle and health

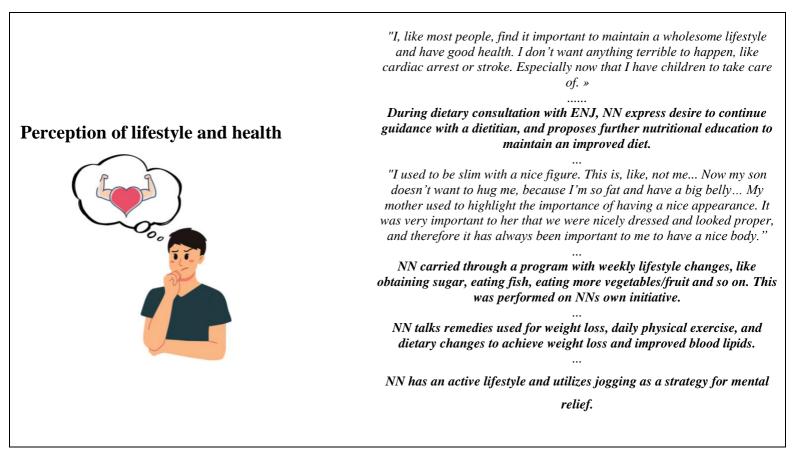


 Table 13e: Six observations and statements were interpreted as signs of the participants subjective valuation of lifestyle and diet, and the importance of maintaining good somatic health. ENJ; Emma N. Johannessen, registered dietitian.

# 5. Discussion

**Summary**: Forty-two patients with an SSD or BPD receiving treatment in mental care facilities in Asker were examined for anthropometry, biochemistry, and dietary habits between 2021 and 2023. A large prevalence of obesity and MetS was identified, and the SMI-patients had overall poor dietary quality, low physical activity level and high prevalence of tobacco usage.

## 5.1 Methodological considerations

The following section will discuss strengths and limitations of the current study, and compare it to the methodology of the assessment that took place in 2021/22 (99).

## 5.1.1 Limitations

#### Subjects and study flow

The master student in 2021/22 reported difficulties with recruitment due to the psychiatric ward in Asker being unfamiliar with dietary health professionals (99). Despite the assumed accustoming from that assessment, the master student faced similar obstacles during recruitment in the psychiatric ward in Asker also in 2022/23. The recruited participants in 2022/23 were more severely affected by SMI and had lower functioning than those in 2021/22, resulting in a smaller sample than desired.

#### Selection bias during recruitment

Recruitment relied heavily on mental care providers at the Sikta and FACT-section to identify eligible participants, potentially leading to selection bias. Mental care providers were hesitant to recommend patients for the study, citing concerns about the psychological strain of a dietary assessment. This may have been justified given the severity of the patient groups. As a result, patients with an interest in lifestyle change may have been unintentionally favored, leading to possible recruitment bias in the study sample.

#### **DIGIKOST**-assessment

A considerable limitation regarding the dietary data obtained in this assessment is that it was self-reported. Self-reported dietary intake is known to be prone to bias and inaccuracy. This can be due to social desirability and -approval bias, which might have been the case in this study, as participants were supervised by the master student while submitting DIGIKOST

(114). People with BMI  $\geq$  30 kg/m<sup>2</sup> have proved to be especially liable to underreport their dietary intake (115). Considering the high rate of obesity, there might have been cases of underreporting dietary intake in the current sample. Memory is also a limiting factor, which might be especially relevant for SMI patients (10).

DIGIKOST is an FFQ, which only measures habitual intake of certain foods. Daily energy intake is not measured, thus subjects with excessive energy intake were not identified in this assessment. Binge eating disorder and night snacking is known to be frequent in SMI patients (116), but FFQs neither investigates *when* eating occasions take place, or how *much* food that is consumed at once. Clues of such eating disordered behavior was therefore not detected with the DIGIKOST-FFQ.

#### Comparison of dietary data

Currently, the general populations dietary habits have not been assessed using the DIGIKOST-FFQ. Norkost 3 is the most recent national dietary survey, providing data of dietary intake in Norwegian adults aged 18-70 years (117). Norkost 3 is thought to be the most valid way to compare the study populations dietary habits to the general populations. Validity of comparison between these studies is limited, due to different methods for dietary assessment.

#### **Biochemistry and clinical measures**

The 2021/22 sample had inconsistent measurements of blood samples and BP, with uncertain validity. Some measurements were likely not conducted in fasting subjects, and most parameters were collected from medical journals. Additionally, variation may occur when analysis is performed in different laboratories or with different BP monitors. Ideally, all participants should have been screened under equal conditions for all proposed parameters.

#### 5.1.2 Strengths

This assessment is an important addition to a scarce scientific body of evidence regarding the lifestyle of SMI patients in Norway.

#### Subjects and study flow

This assessment succeeded to recruit 18 patients from the outpatient-, FACT and 24-hour section in the mental care ward of Asker. 25 patients were recruited from the out-patient

section in 2021/22 (99). Together this constituted a sample of 42 participants, with equal proportions of patients within groups of both gender and diagnosis. Conclusively, the assessed sample is now representative for a broad variety of psychiatric patients receiving treatment in Asker.

#### **DIGIKOST**-assessment

Compared to a comprehensive dietary assessment like a 24-hour recall interview, DIGIKOST determines dietary habits instead of a single days' intake. Some participants might experience psychological discomfort when sharing personal details of their dietary intake. With that in mind, DIGIKOST is a less invading dietary assessment where the participant can describe their habits in general terms. DIGIKOST and similar validated FFQs might therefore be the most appropriate tool for measuring dietary habits of the population at hand. Validation of DIGIKOST found it to provide appropriate data of individuals' habitual dietary intake (111). DIGIKOST was also found to be easily interpreted and time efficient in a qualitative study (110). The master student could motivate patients to participate in the assessment, by arguing that the dietary assessment only consisted of a simple online questionnaire. This is thought to have benefited recruitment in 2022/23.

#### **Biochemistry and clinical measures**

Assessments were conducted in a standardized manner for all patients recruited in 2022/23, providing sound data of anthropometry and biochemistry that allowed accurate determination of MetS. In the most recent sample, all participants except of one had new blood samples drawn according to the protocol of this thesis. It is thought that the methodology in the current study truthfully was improved by requisitioning new blood samples and performing anthropometric measures under standardized preconditions for all participants.

#### Advantages of study participation

Participating in this study offered SMI-patients a break from their ordinary psychiatric treatment. Based on the scientific background for this thesis, introducing lifestyle counselling in an SMI population is much needed, and carries great potential for improving the patients' somatic health (72). Receiving two consultations with a registered dietitian was indeed a generous offer, considering the low availability of dietitians in the health ward. Participants expressed gratitude for the dietary consultations, and even proposed more long-lasting dietary coaching (table 13e).

#### **5.2 Discussion of results**

This section will consider the implications of the results and discuss how they should be interpreted.

#### 5.2.1 Characteristics of the SMI population

Based on an average BMI of 29 kg/m<sup>2</sup> in men and 30.9 kg/m<sup>2</sup> in women, the average participant in this sample was (borderline) obese. Abdominal obesity was prevalent in 57% of the patients. Contrary to what one would expect, there was no difference in WC between men and women. This indicates that female participants were more severely obese than the men. AP-medication might be a driver for obesity and weight gain in the current population (59). Especially olanzapine is highlighted as a drug that causes massive weight gain shortly after medication has started (118). In the current study, 64.5% of the participants were prescribed to AP-medication with high risk of weight gain, and 26.3% of these with olanzapine.

#### 5.2.2 Metabolic syndrome

In Norway, the prevalence of MetS has been estimated to be 25.9% (119). Comparably, participants in this study were twice as likely to be afflicted with MetS. This poses the assessed SMI patients at a much higher risk for developing ACVD relative to mentally healthy Norwegians (47). In this study, 50% of the SMI patients fulfilled at least 3 diagnosis criterions for MetS. The most frequently fulfilled criteria was abdominal obesity and hypertension, which both are modifiable with dietary intervention (104).

Assumption of abdominal obesity might however be underestimated in the sample from 2021/22, as several participants in the most recent sample had a BMI  $< 30 \text{ kg/m}^2$  but were deemed abdominally obese when measuring WC. Monitoring WC might be a useful tool to assess abdominal obesity and risk of ACVD in psychiatric patients, rather than solely relying on BMI. This is supported in previous trials that found WC to be a better predicator of metabolic morbidity than BMI alone (120).

#### 5.2.3 Serum lipids compared to treatment goals

The treatment goals for serum levels of blood lipids determined by ESC/EAS for persons at high risk for ACVD are lower than the cut off limits recommended for the average population (55, 56). SMI patients in Asker were rarely screened for serum lipids in the psychiatric ward, and physicians were reluctant to do so (appendix 1). The treatment goals for high-risk

individuals might be more appropriate cut off limits for the patients at topic, considering the prevalence of metabolic risk factors we identified. Relative to these treatment goals, total cholesterol in the current sample was too high (5.0 mmol/L  $\pm$ SD 0.9, recommended <4 mmol/L). The same was true for LDL- (3.6 $\pm$ SD 0.7 mmol/L for men and 3.7 $\pm$ SD 0.8 for women, recommended <1.8 mmol/L) and non-HDL Cholesterol (3.8 $\pm$ SD 0.9/ $\pm$ 1.0 mmol/L for men/women, recommended <2.6 mmol/L) (56). The number of participants prescribed with statins have not been assessed thoroughly in this thesis, but Emma N. Johannessen reported a low percentage of patients prescribed with lipid lowering drugs (99). To the best of knowledge, the same was true for patients recruited in 2022/23 (appendix 1).

#### 5.2.3 Vitamin D insufficiency and -supplementation

Low levels of vitamin D can be due to insufficient dietary intake or low exposure to sunlight (98). Observations made by mental care providers indicate that the SMI patients in Asker engage little in outdoor activities, and the current assessment found the dietary quality to be low. Obesity, which was highly prevalent among SMI-patients in this study, is also coupled to low levels of vitamin D (121).

In Norway, a cross-sectional study found the prevalence of vitamin D deficiency to be 40% in a sample of 2.460 adults (122). In the study at hand, 17.3% had a clinical deficiency of vitamin D (serum level < 50 nmol/L), while as much as 65.5% had suboptimal serum levels (< 80 nmol/L). However, the true prevalence of vitamin D deficiency might be higher than 17.3%, since screening of vitamin D only were sporadic in the sample from 2021/22. The median serum value was < 80 nmol/L in both men and women, which indicates that low serum levels of vitamin D was highly prevalent in the assessed subjects.

Knowing that vitamin D deficiency is common in the general Norwegian population, the DIGIKOST scoring of '0' for taking any dietary supplements might be worth reconsideration. This is especially true regarding the patient group at hand; individuals that engage in little outdoor activities and are likely to be obese. Scientific consensus agrees that vitamin D fortification and -supplementation is a safe strategy to ensure adequate serum levels of vitamin D (123). Screening for serum vitamin D with eventual supplementation can be an appropriate strategy to prevent deficiency in SMI patients.

#### 5.2.4 Lifestyle index

Currently, no comparison to the general Norwegian population can be made with scoring of lifestyle index generated with the DIGIKOST questionnaire. The median lifestyle index of 2.5 in the study at hand indicates moderate to low compliance to recommendations for a healthy lifestyle. A small proportion of the participants scored within the health index area deemed 'high' in DIGIKOST. Diet, healthy body weight and exercise level had a similar score of 0.5 or 'moderate'. Tobacco usage was highly prevalent, corresponding to a overall score of 0. Reported alcohol consumption in the sample was low enough to yield 1 point, contrary to what previous studies in SMI populations have found (124).

#### 5.2.5 Diet Score

Almost half of the participants (46.5%) had an overall Diet score corresponding to low compliance to Norwegian FBDG. The median Diet Score was 7.5, which indicates moderate to low compliance. The Lifestyle Index regarding diet was 0.5, with the lower percentile being 0 and the higher 0.5. This distribution indicates that most participants had a score in the lower range of the median "moderate" dietary quality.

#### 5.2.6 Adherence to single Norwegian FBDG

Current dietary recommendations concludes that 1 out of 5 daily portions of fruit, berries and vegetables can be constituted of juice (107). An important notice regarding this FBDG, is that a greater proportion of juice is included into the intake variable in DIGIKOST, compared to in Norkost 3 (117). Thus, the compliance to the FBDG about fruit, vegetables, and berries might be overestimated when using DIGIKOST. The population at hand can be thought to have a health benefit of substituting juice with whole produce fruit and vegetables, as this can help with increased satiety and prevent overeating (125). Taken together, the compliance to FBDG regarding fruit, berries and vegetables might be overestimated in this study, due to daily intake of fruit juice in certain participants. Intake of fruit, vegetables and berries was nonetheless lower than recommended in the examined population, which is similar to what was found in Norkost 3.

Norkost 3 found that Norwegians eat to little whole grains and fish (117). Contrary to this, several participants in the current study reported intakes compliant with the Norwegian FBDG for fish and whole grain products. However, the reported intake of red and processed meat was much higher than recommended, and exceeded the general populations intake in Norkost

3, which also was too high (117). Norkost 3 found Norwegians to eat too much added sugar and saturated fat (117). The intake of foods rich in sugar and fat, as well as beverages with added sugar was much higher than recommended in the current study. Taken together, the dietary pattern of SMI patients is quite similar to that of the regular Norwegian population. A high intake of energy dense food might partly explain the high prevalence of obesity as well as reported weight gain coupled to initiation of AP medication in the current subjects.

#### 5.2.7 Exercise and sedentary time

Norwegian health authorities recommend adults to be physically active *at least* 150 min/week (107). Throughout the recommendations for exercise, it is underscored that *more* physical activity is coupled to better health, and that any exercise is better than none. It is also communicated that high intensity exercise has great health benefits (126). A controlled trial using accelerometers found only 36% of Norwegian women and 30% of Norwegian men to be compliant with national recommendations for physical activity (127).

Contrary, and quite surprisingly, 44% of the participants in this study reported physical activity corresponding to high compliance to national recommendations, 40% had low compliance, and only 16% were categorized as an intermediate of the two. The high proportion of highly compliant SMI-patients was due to frequent reports of sufficient time spent moderately active, as almost none reported habitual vigorous activity or exercise. It is also not possible to determine each participants' interpretation of "moderately intense" exercise. Some might have understood this as any physical activity like walking or doing basic chores like shopping. Self-reported exercise is also prone to social desirability and - approval bias, resulting in overreporting (128, 129).

Median time (25-, 75%) spent moderately active was 119.9 min/week (36.5, 226.2), which is quite far from the recommended *minimum* of moderately intense activity (126). The distribution of the median indicates that there was a distinct discrepancy between the participants' reported exercise. Furthermore, the median lifestyle index regarding exercise was 0.5, corresponding to intermediate compliance. The apparent high compliance to exercise recommendations in the current sample should therefore be interpreted critically.

#### Sedentary time

Using objective measures, the average Norwegian was found to spend 9 hours daily sedentary (127). In the current study, participants reported less inactive time, with a median of 8 hours. This is contrary to what was hypothesized, but there are some important notions that must be made. Studies have shown that individuals with low education, which was true for most participants in the population at hand, are more likely to overreport exercise (130). Recall bias is another possible explanation, leading to inability to accurately estimate regular sedentary time. Time spent inactive is not something a regular person usually keeps track of and might be hard to estimate accurately. Conclusively, sedentary time might be underestimated, and time spent active overestimated in the current study. To make precise estimates of the physical activity level in SMI patients living in Norway, objective measures must be used.

#### 5.2.8 Tobacco and Alcohol

Only 5% of Norwegians aged 25-49 reported to be daily smokers in 2022 (131). Meanwhile, 19% of Norwegians aged 16-74 used snus-tobacco either daily or occasionally (132). Taken together, the trend of tobacco usage in Norway seems to be coinciding with what we discovered in the current study. We found that 26.8% of the participants smoke cigarettes daily or occasionally, and an equal proportion were previous smokers. Interestingly, none of the participants reported to have stopped using snus, while 41.5% used snus either occasionally or daily. The absolute rate of tobacco usage is higher in SMI patients compared to the overall population, but similar to mentally healthy Norwegians, it seems that tobacco smoking is on a decline while snus tobacco is gaining more popularity (131, 132).

Alcohol intake in the current study was estimated based on a question in DIGIKOST: "Do you drink alcohol habitually?" with following specifications about how often, and how many units of diverse alcoholic beverages the participant drank. No assumption can be made of whether this was understood as total abundance, or something they rarely indulge in. Participants reporting no habitual intake could also be previous drinkers, which was not assessed in the DIGIKOST-FFQ. Binge drinking, which is a typical Norwegian phenomenon (133), might also be interpreted as not having a habitual intake of alcohol.

In Norkost 3, the mean alcohol intake in both men and women were below the recommended 5 E% per day (117). This limit equals approximately 16 grams of ethanol per day for an individual consuming 2000 kilocalories per day. There is a substantial discrepancy between

the determination in DIGIKOST of a high alcohol consumption being anything above 4.29 g/d, whereas the general recommendation is <10 g/d for women and <20g/d for men (134). In this study, 65.8% of the participants reported to not have a habitual alcohol intake, while 29.3% reported an intake that was deemed high (>4.29 g/day) according to DIGIKOST. Compared to this, the average Norwegian would be deemed a high-consumer of alcohol when submitting DIGIKOST, considering what was reported in Norkost 3 (117)

SMI patients should be modest in their alcohol consumption, as drinking is coupled to more distress when mentally ill (135, 136). This has likely been communicated by mental care providers, thus it makes sense that many SMI-patients truthfully abstained from alcohol at the time of DIGIKOST-submission.

#### 5.2.9 Qualitative findings

The qualitative statements that emerged provided valuable nuancing to the data from this assessment. Many patients told personal stories about weight gain after AP-medication was initiated (table 13a). They also shared experiences on how these metabolic repercussions affected their quality of life.

One participant expressed concern related to the fact that mental care providers were aware of the side effects that comes with AP-medication, while the patients were not. This matter is worth reflecting on. Medication with AP-agents is not something SMI patients choose by themselves, and it can be discussed whether it is ethically defendable to prescribe patients with obesogenic medication, without informing thoroughly about these side effects and provide preventive lifestyle counselling. This can be interpreted as contrary to a central principle in health care that is "to not cause harm"(137).

Interestingly, some participants shared stories about how hospitalization contributed to a higher, and more calorie dense, food intake than usual (table 13b). This too can be questioned ethically; patients are enrolled to psychiatric treatment to improve their health and quality of life. However, they are placed in a food environment that offers energy dense treats and high-palatable meals. Simultaneously, most patients are medicated with appetite-increasing medication. This exposure to obesogenic factors probably promotes the experienced weight gain and subsequent high rate of obesity.

Patient statements also gave useful insight in the level of knowledge about diet and lifestyle that they presented with (table 13c). The participant's lack of knowledge about diet and health highlights the value that dietary education could offer this patient group. Some of the statements also indicated that patients already made independent efforts to gain knowledge about diet and lifestyle. The health ward should ensure that nutrition information delivered to psychiatric patients is in line with scientific consensus. This can be achieved with more frequent employment of dietary health professionals in the mental care ward.

Additionally, statements interpreted as signs of eating disordered behavior underscores the importance of having caution when recommending weight loss or dietary changes in this population (table 13d). It has been shown previously that lifestyle-based interventions in SMI-patients are more successful when they are supervised by a registered dietitian (138). Dietary counselling with a health professional like a registered dietitian is also a safer delivery method since weight loss and diet can be sensitive topics.

Lastly, statements that emerged from patient meetings carried a voice of their own valuation of somatic health (table 13e). Many patients expressed desire for weight loss and wanted to improve their lifestyle. This is an important argument for why lifestyle-based interventions are both necessary and expedient in an SMI patient group. An additional patient story from the assessment is presented in appendix 1.

#### **5.3 Ethical considerations**

The fact that SMI patients lead unwholesome lifestyles have been confirmed many times. Considering the shortage of lifetime this represents, measures should be implemented to improve lifestyle and lower the risk of ACVD in this population. It is questionable that screening of somatic health and measures to prevent adverse weight gain is not an established part of the standard procedure when AP-medication is initiated. The time is well due to make a change in the psychiatric ward to ensure wholesome care of SMI patients' health.

#### **5.4 Further research**

More detailed appraisal of SMI patients' dietary intake can provide valuable insights in macro- and micronutrient intake as well as total energy intake. This can be managed if validated and feasible assessment methods are applied. Promising results are emerging from new web-based food record tools, which could be a low-invasive option of dietary assessment (139). Alternatively, gold standard methods like weighed dietary registration and double labeled water could be utilized to yield high-quality data (140). Self-report of exercise is probably not sufficient to determine valid data of SMI-patients physical activity level. Future assessments should prosper to objectively measure the amount and intensity of habitual exercise in SMI patients.

Metabolic morbidity is reversible with lifestyle intervention (104), thus prevention of somatic morbidity in SMI-patients is possible. Lifestyle-based interventions have been successfully implemented in SMI populations, with beneficial outcome on somatic *and* mental health (72, 141). It seems that applying an intervention including exercise is the most effective method, and that registered dietitians should lead the dietary counselling (138). Lifestyle interventions for improved diet and increased exercise may be successful in lowering the risk for ACVD, even when weight loss is not achieved (46).

Qualitative statements that emerged in meetings with SMI patients receiving treatment in Asker implied that they were interested in their current somatic health, and motivated to achieve a healthier lifestyle with dietary counselling (table 13d). Lifestyle-based intervention trials led by dietary health professionals can be an important next step to determine whether dietary counselling is effective, and to battle AP-medication related weight gain and metabolic abnormalities in this patient group.

## 6. Conclusion

Most SMI-patients were obese, and the prevalence of MetS was twice that of the general Norwegian population, with abdominal obesity being the most profound metabolic risk factor for ACVD. Serum lipids were high relative to the treatment goals for high risk-individuals. Adherence to Norwegian FBDG were low/moderate and compliance to exercise recommendations was low. Smoking was more common than in the regular Norwegian population, but most SMI-patients reported to abstain from alcohol. Low serum levels of vitamin D were highly prevalent, while no other micronutrient deviations were identified. Vitamin D supplementation can be necessary to obtain adequate serum levels in SMI patients in Norway. Interventions to improve diet and increase exercise is thought to benefit the patient's receiving treatment for SMI in Norway. Alongside smoking cessation, these are important steps to prevent morbidity and promote longevity in this patient group.

# References

- 1. World Health O. ICD-10 : psykiske lidelser og atferdsforstyrrelser : kliniske beskrivelser og diagnostiske retningslinjer. Oslo: Universitetsforl.; 1999.
- 2. Lichtenstein P, Yip BH, Björk C, Pawitan Y, Cannon TD, Sullivan PF, et al. Common genetic determinants of schizophrenia and bipolar disorder in Swedish families: a population-based study. Lancet. 2009;373(9659):234-9.
- Misiak B, Stramecki F, Gawęda Ł, Prochwicz K, Sąsiadek MM, Moustafa AA, et al. Interactions Between Variation in Candidate Genes and Environmental Factors in the Etiology of Schizophrenia and Bipolar Disorder: a Systematic Review. Mol Neurobiol. 2018;55(6):5075-100.
- 4. Wells R, Jacomb I, Swaminathan V, Sundram S, Weinberg D, Bruggemann J, et al. The Impact of Childhood Adversity on Cognitive Development in Schizophrenia. Schizophr Bull. 2020;46(1):140-53.
- 5. Bell MD, Lysaker PH. Psychiatric symptoms and work performance among persons with severe mental illness. Psychiatric Services. 1995;46:508-10.
- 6. Whiteford HA, Degenhardt L, Rehm J, Baxter AJ, Ferrari AJ, Erskine HE, et al. Global burden of disease attributable to mental and substance use disorders: findings from the Global Burden of Disease Study 2010. Lancet. 2013;382(9904):1575-86.
- Charlson FJ, Ferrari AJ, Santomauro DF, Diminic S, Stockings E, Scott JG, et al. Global Epidemiology and Burden of Schizophrenia: Findings From the Global Burden of Disease Study 2016. Schizophr Bull. 2018;44(6):1195-203.
- 8. Ferrari AJ, Stockings E, Khoo JP, Erskine HE, Degenhardt L, Vos T, et al. The prevalence and burden of bipolar disorder: findings from the Global Burden of Disease Study 2013. Bipolar Disord. 2016;18(5):440-50.
- 9. Helsebiblioteket. Schizofreni. Helsebiblioteketno: BMJ, Best Practice. 2019.
- 10. Malt UR, Jan Ivar. Schizofreni. Store medisinske leksikon. 2022.
- 11. Anticevic A, Cole MW, Repovs G, Murray JD, Brumbaugh MS, Winkler AM, et al. Characterizing Thalamo-Cortical Disturbances in Schizophrenia and Bipolar Illness. Cerebral Cortex. 2013;24(12):3116-30.
- 12. Peleikis DE, Felldal SC. Schizofreni : til å leve med. Oslo: Gyldendal akademisk; 2017.
- 13. Wang B, Zartaloudi E, Linden JF, Bramon E. Neurophysiology in psychosis: The quest for disease biomarkers. Translational Psychiatry. 2022;12(1):100.
- 14. Pantelis C, Velakoulis D, McGorry PD, Wood SJ, Suckling J, Phillips LJ, et al. Neuroanatomical abnormalities before and after onset of psychosis: a cross-sectional and longitudinal MRI comparison. The Lancet. 2003;361(9354):281-8.
- 15. Choi S, Kim M, Park H, Kim T, Moon S-Y, Lho SK, et al. Volume deficits in hippocampal subfields in unaffected relatives of schizophrenia patients with high genetic loading but without any psychiatric symptoms. Schizophrenia Research. 2022;240:125-31.
- 16. Jääskeläinen E, Juola P, Hirvonen N, McGrath JJ, Saha S, Isohanni M, et al. A systematic review and meta-analysis of recovery in schizophrenia. Schizophr Bull. 2013;39(6):1296-306.
- 17. Haver B, Ødegaard KJ, Andreassen OA, Fasmer OB. Bipolare lidelser. Bergen: Fagbokforl.; 2012.
- 18. Malt UR, Jan Ivar. Bipolar Lidelse. Store medisinske leksikon. 2021.
- 19. Wikipedia. Mania. 2022.
- 20. Malt UR, Jan Ivar. Mani. Store medisinske leksikon.
- 21. Melo MCA, Abreu RLC, Linhares Neto VB, de Bruin PFC, de Bruin VMS. Chronotype and circadian rhythm in bipolar disorder: A systematic review. Sleep Medicine Reviews. 2017;34:46-58.
- 22. Aminoff SR, Onyeka IN, Ødegaard M, Simonsen C, Lagerberg TV, Andreassen OA, et al. Lifetime and point prevalence of psychotic symptoms in adults with bipolar disorders: a systematic review and meta-analysis. Psychological Medicine. 2022:1-13.
- 23. Bonnín CM, Jiménez E, Solé B, Torrent C, Radua J, Reinares M, et al. Lifetime Psychotic Symptoms, Subthreshold Depression and Cognitive Impairment as Barriers to Functional Recovery in Patients with Bipolar Disorder. J Clin Med. 2019;8(7).
- 24. Namjoshi MA, Buesching DP. A review of the health-related quality of life literature in bipolar disorder. Quality of Life Research. 2001;10(2):105-15.
- 25. legemiddelhåndbok FfuaN.
- T5.6 Bipolar lidelse. Terapikapitler. 2021.
- 26. Smoller JW, Finn CT. Family, twin, and adoption studies of bipolar disorder. Am J Med Genet C Semin Med Genet. 2003;123c(1):48-58.

- 27. Veldhuizen Rv, Nasjonal kompetansetjeneste samtidig rus- og psykisk l. FACT : flexible assertive community treatment : visjon, modell og organisering av FACT-modellen. 2. utg. ed. Brumunddal: Nasjonal kompetansetjeneste ROP; 2013.
- 28. Helsedirektoratet og Tips RJoSD. e-Læring Psykose.5.
- 29. de Bartolomeis A, Barone A, Begni V, Riva MA. Present and future antipsychotic drugs: A systematic review of the putative mechanisms of action for efficacy and a critical appraisal under a translational perspective. Pharmacological Research. 2022;176:106078.
- 30. Leucht S, Cipriani A, Spineli L, Mavridis D, Orey D, Richter F, et al. Comparative efficacy and tolerability of 15 antipsychotic drugs in schizophrenia: a multiple-treatments meta-analysis. Lancet. 2013;382(9896):951-62.
- 31. Hovd MHØ, Ivar. Antipsykotika. Store medisinske leksikon.
- 32. Jørgensen HA. Medikamentell behandling av schizofreni. Tidsskr Nor Lægeforen. 2002;22.
- 33. Leucht S, Cipriani A, Spineli L, Mavridis D, Örey D, Richter F, et al. Comparative efficacy and tolerability of 15 antipsychotic drugs in schizophrenia: a multiple-treatments meta-analysis. The Lancet. 2013;382(9896):951-62.
- Hirsch L, Yang J, Bresee L, Jette N, Patten S, Pringsheim T. Second-Generation Antipsychotics and Metabolic Side Effects: A Systematic Review of Population-Based Studies. Drug Safety. 2017;40(9):771-81.
- 35. Divac N, Prostran M, Jakovcevski I, Cerovac N. Second-Generation Antipsychotics and Extrapyramidal Adverse Effects. BioMed Research International. 2014;2014:656370.
- 36. de Mooij LD, Kikkert M, Theunissen J, Beekman ATF, de Haan L, Duurkoop P, et al. Dying Too Soon: Excess Mortality in Severe Mental Illness. Front Psychiatry. 2019;10:855.
- 37. Laursen TM, Wahlbeck K, Hällgren J, Westman J, Ösby U, Alinaghizadeh H, et al. Life Expectancy and Death by Diseases of the Circulatory System in Patients with Bipolar Disorder or Schizophrenia in the Nordic Countries. PLOS ONE. 2013;8(6):e67133.
- 38. Nordentoft M, Wahlbeck K, Hällgren J, Westman J, Osby U, Alinaghizadeh H, et al. Excess mortality, causes of death and life expectancy in 270,770 patients with recent onset of mental disorders in Denmark, Finland and Sweden. PLoS One. 2013;8(1):e55176.
- 39. WHO. Cardiovascular diseases (CVDs). Newsroom, World Health Organization. 2021.
- 40. Newcomer JW, Hennekens CH. Severe Mental Illness and Risk of Cardiovascular Disease. JAMA. 2007;298(15):1794-6.
- 41. Ringen PA, Engh JA, Birkenaes AB, Dieset I, Andreassen OA. Increased mortality in schizophrenia due to cardiovascular disease a non-systematic review of epidemiology, possible causes, and interventions. Front Psychiatry. 2014;5:137.
- 42. Beverly JK, Budoff MJ. Atherosclerosis: Pathophysiology of insulin resistance, hyperglycemia, hyperlipidemia, and inflammation. Journal of diabetes. 2020;12(2):102-4.
- 43. Levenson JW, Skerrett PJ, Gaziano JM. Reducing the global burden of cardiovascular disease: the role of risk factors. Preventive cardiology. 2002;5(4):188-99.
- 44. Lallukka T, Millear A, Pain A, Cortinovis M, Giussani G. GBD 2015 Mortality and Causes of Death Collaborators. Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2015 (vol 388, pg 1459, 2016). The Lancet (British edition). 2017;389(10064):E1-E.
- 45. Wong B, Kruse G, Kutikova L, Ray KK, Mata P, Bruckert E. Cardiovascular Disease Risk Associated With Familial Hypercholesterolemia: A Systematic Review of the Literature. Clin Ther. 2016;38(7):1696-709.
- 46. Acosta S, Johansson A, Drake I. Diet and Lifestyle Factors and Risk of Atherosclerotic Cardiovascular Disease—A Prospective Cohort Study. Nutrients. 2021;13(11):3822.
- 47. Guembe MJ, Fernandez-Lazaro CI, Sayon-Orea C, Toledo E, Moreno-Iribas C. Risk for cardiovascular disease associated with metabolic syndrome and its components: a 13-year prospective study in the RIVANA cohort. Cardiovasc Diabetol. 2020;19(1):195.
- 48. Gurusamy J, Gandhi S, Damodharan D, Ganesan V, Palaniappan M. Exercise, diet and educational interventions for metabolic syndrome in persons with schizophrenia: A systematic review. Asian J Psychiatr. 2018;36:73-85.
- 49. Chang SH, Chien NH, Yu CY. Long-Term Lifestyle Intervention in Elderly With Metabolic Syndrome. Clin Nurs Res. 2019;28(6):658-75.
- 50. Rochlani Y, Pothineni NV, Kovelamudi S, Mehta JL. Metabolic syndrome: pathophysiology, management, and modulation by natural compounds. Ther Adv Cardiovasc Dis. 2017;11(8):215-25.
- 51. Mitchell AJ, Vancampfort D, Sweers K, van Winkel R, Yu W, De Hert M. Prevalence of Metabolic Syndrome and Metabolic Abnormalities in Schizophrenia and Related Disorders—A Systematic Review and Meta-Analysis. Schizophrenia Bulletin. 2011;39(2):306-18.

- 52. McEvoy JP, Meyer JM, Goff DC, Nasrallah HA, Davis SM, Sullivan L, et al. Prevalence of the metabolic syndrome in patients with schizophrenia: Baseline results from the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) schizophrenia trial and comparison with national estimates from NHANES III. Schizophr Res. 2005;80(1):19-32.
- 53. Dickerson FB, Brown CH, Kreyenbuhl JA, Fang L, Goldberg RW, Wohlheiter K, et al. Obesity among individuals with serious mental illness. Acta Psychiatr Scand. 2006;113(4):306-13.
- 54. Vancampfort D, Stubbs B, Mitchell AJ, De Hert M, Wampers M, Ward PB, et al. Risk of metabolic syndrome and its components in people with schizophrenia and related psychotic disorders, bipolar disorder and major depressive disorder: a systematic review and meta-analysis. World Psychiatry. 2015;14(3):339-47.
- 55. (NEL) NEL. Kolesterol. Norsk Helseinformatikk. 2019.
- 56. Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk The Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS). European Heart Journal. 2020;41(1):111-88.
- 57. Saklayen MG. The Global Epidemic of the Metabolic Syndrome. Curr Hypertens Rep. 2018;20(2):12.
- 58. Correll CU, Robinson DG, Schooler NR, Brunette MF, Mueser KT, Rosenheck RA, et al. Cardiometabolic Risk in Patients With First-Episode Schizophrenia Spectrum Disorders: Baseline Results From the RAISE-ETP Study. JAMA Psychiatry. 2014;71(12):1350-63.
- 59. Barton BB, Segger F, Fischer K, Obermeier M, Musil R. Update on weight-gain caused by antipsychotics: a systematic review and meta-analysis. Expert Opinion on Drug Safety. 2020;19(3):295-314.
- 60. van Zonneveld SM, Haarman BCM, van den Oever EJ, Nuninga JO, Sommer IEC. Unhealthy diet in schizophrenia spectrum disorders. Curr Opin Psychiatry. 2022;35(3):177-85.
- 61. Nunes D, Eskinazi B, Rockett FC, Delgado VB, Perry IDS. Nutritional status, food intake and cardiovascular disease risk in individuals with schizophrenia in southern Brazil: A case–control study. Revista de Psiquiatría y Salud Mental (English Edition). 2014;7(2):72-9.
- 62. Lopresti AL, Jacka FN. Diet and Bipolar Disorder: A Review of Its Relationship and Potential Therapeutic Mechanisms of Action. J Altern Complement Med. 2015;21(12):733-9.
- 63. Shahar D, Shai I, Vardi H, Shahar A, Fraser D. Diet and eating habits in high and low socioeconomic groups. Nutrition. 2005;21(5):559-66.
- 64. Khosravi M. Biopsychosocial factors associated with disordered eating behaviors in schizophrenia. Annals of General Psychiatry. 2020;19(1):67.
- 65. Fernandez-Egea E, Bernardo M, Parellada E, Justicia A, Garcia-Rizo C, Esmatjes E, et al. Glucose abnormalities in the siblings of people with schizophrenia. Schizophr Res. 2008;103(1-3):110-3.
- 66. Onaolapo OJ, Onaolapo AY. Nutrition, nutritional deficiencies, and schizophrenia: An association worthy of constant reassessment. World J Clin Cases. 2021;9(28):8295-311.
- 67. Bauman AE, Reis RS, Sallis JF, Wells JC, Loos RJF, Martin BW. Correlates of physical activity: why are some people physically active and others not? The Lancet. 2012;380(9838):258-71.
- 68. Sterdt E, Liersch S, Walter U. Correlates of physical activity of children and adolescents: A systematic review of reviews. Health Education Journal. 2014;73(1):72-89.
- 69. Muench J, Hamer AM. Adverse effects of antipsychotic medications. Am Fam Physician. 2010;81(5):617-22.
- 70. Anagha K, Shihabudheen P, Uvais NA. Side Effect Profiles of Selective Serotonin Reuptake Inhibitors: A Cross-Sectional Study in a Naturalistic Setting. Prim Care Companion CNS Disord. 2021;23(4).
- 71. Jacka FN, O'Neil A, Opie R, Itsiopoulos C, Cotton S, Mohebbi M, et al. A randomised controlled trial of dietary improvement for adults with major depression (the 'SMILES' trial). BMC Med. 2017;15(1):23.
- 72. Fernández-Abascal B, Suárez-Pinilla P, Cobo-Corrales C, Crespo-Facorro B, Suárez-Pinilla M. In- and outpatient lifestyle interventions on diet and exercise and their effect on physical and psychological health: a systematic review and meta-analysis of randomised controlled trials in patients with schizophrenia spectrum disorders and first episode of psychosis. Neuroscience & Biobehavioral Reviews. 2021;125:535-68.
- 73. Manu P, Dima L, Shulman M, Vancampfort D, De Hert M, Correll CU. Weight gain and obesity in schizophrenia: epidemiology, pathobiology, and management. Acta Psychiatrica Scandinavica. 2015;132(2):97-108.
- 74. Firth J, Cotter J, Elliott R, French P, Yung AR. A systematic review and meta-analysis of exercise interventions in schizophrenia patients. Psychol Med. 2015;45(7):1343-61.

- 75. Rosenbaum S, Tiedemann A, Sherrington C, Curtis J, Ward PB. Physical activity interventions for people with mental illness: a systematic review and meta-analysis. J Clin Psychiatry. 2014;75(9):964-74.
- 76. de Beaurepaire R. Binge Eating Disorders in Antipsychotic-Treated Patients With Schizophrenia: Prevalence, Antipsychotic Specificities, and Changes Over Time. J Clin Psychopharmacol. 2021;41(2):114-20.
- 77. Qian J, Wu Y, Liu F, Zhu Y, Jin H, Zhang H, et al. An update on the prevalence of eating disorders in the general population: a systematic review and meta-analysis. Eating and Weight Disorders Studies on Anorexia, Bulimia and Obesity. 2022;27(2):415-28.
- 78. McElroy SL, Crow S, Biernacka JM, Winham S, Geske J, Cuellar Barboza AB, et al. Clinical phenotype of bipolar disorder with comorbid binge eating disorder. Journal of Affective Disorders. 2013;150(3):981-6.
- 79. Malaspina D, Walsh-Messinger J, Brunner A, Rahman N, Corcoran C, Kimhy D, et al. Features of schizophrenia following premorbid eating disorders. Psychiatry Research. 2019;278:275-80.
- 80. Yum SY, Hwang MY, Halmi KA. Eating disorders in schizophrenia. Psychiatric times. 2006;23(7):10-.
- Osuji PN, Onu JU. Feeding behaviors among incident cases of schizophrenia in a psychiatric hospital: Association with dimensions of psychopathology and social support. Clinical Nutrition ESPEN. 2019;34:125-9.
- 82. de Leon J, Diaz FJ. A meta-analysis of worldwide studies demonstrates an association between schizophrenia and tobacco smoking behaviors. Schizophrenia Research. 2005;76(2):135-57.
- Diaz FJ, James D, Botts S, Maw L, Susce MT, De Leon J. Tobacco smoking behaviors in bipolar disorder: a comparison of the general population, schizophrenia, and major depression. Bipolar disorders. 2009;11(2):154-65.
- 84. Li D, Yang X, Ge Z, Hao Y, Wang Q, Liu F, et al. Cigarette smoking and risk of completed suicide: A meta-analysis of prospective cohort studies. Journal of Psychiatric Research. 2012;46(10):1257-66.
- 85. Tanskanen A, Viinamäki H, Hintikka J, Koivumaa-Honkanen H-T, Lehtonen J. Smoking and suicidality among psychiatric patients. American Journal of Psychiatry. 1998;155(1):129-30.
- Toftdahl NG, Nordentoft M, Hjorthøj C. Prevalence of substance use disorders in psychiatric patients: a nationwide Danish population-based study. Social Psychiatry and Psychiatric Epidemiology. 2016;51(1):129-40.
- 87. van Draanen J, Tsang C, Mitra S, Phuong V, Murakami A, Karamouzian M, et al. Mental disorder and opioid overdose: a systematic review. Social Psychiatry and Psychiatric Epidemiology. 2022;57(4):647-71.
- 88. Khokhar JY, Dwiel LL, Henricks AM, Doucette WT, Green AI. The link between schizophrenia and substance use disorder: A unifying hypothesis. Schizophr Res. 2018;194:78-85.
- 89. Sæland M, Haugen M, Eriksen FL, Wandel M, Smehaugen A, Böhmer T, et al. High sugar consumption and poor nutrient intake among drug addicts in Oslo, Norway. British Journal of Nutrition. 2011;105(4):618-24.
- 90. David B. Allison, Ph.D. ,, Janet L. Mentore, M.S.Ed. ,, Moonseong Heo, Ph.D. ,, Linda P. Chandler, Ph.D. ,, Joseph C. Cappelleri, Ph.D., M.P.H. ,, Ming C. Infante, M.S. , and, et al. Antipsychotic-Induced Weight Gain: A Comprehensive Research Synthesis. American Journal of Psychiatry. 1999;156(11):1686-96.
- 91. Bak M, Fransen A, Janssen J, van Os J, Drukker M. Almost All Antipsychotics Result in Weight Gain: A Meta-Analysis. PLOS ONE. 2014;9(4):e94112.
- 92. Álvarez-Jiménez M, González-Blanch C, Crespo-Facorro B, Hetrick S, Rodriguez-Sánchez JM, Pérez-Iglesias R, et al. Antipsychotic-Induced Weight Gain in Chronic and First-Episode Psychotic Disorders. CNS Drugs. 2008;22(7):547-62.
- 93. Rognoni C, Bertolani A, Jommi C. Second-Generation Antipsychotic Drugs for Patients with Schizophrenia: Systematic Literature Review and Meta-analysis of Metabolic and Cardiovascular Side Effects. Clin Drug Investig. 2021;41(4):303-19.
- 94. Solberg DK. LEGEMIDLER I PRAKSIS Oppfølging av pasienter som bruker litium. Tidsskr Nor Lægeforen. 2008(12).
- 95. Pijl H, Meinders AE. Bodyweight change as an adverse effect of drug treatment. Mechanisms and management. Drug Saf. 1996;14(5):329-42.
- 96. Lips P, van Schoor NM. The effect of vitamin D on bone and osteoporosis. Best Pract Res Clin Endocrinol Metab. 2011;25(4):585-91.
- 97. Khera AV, Emdin CA, Drake I, Natarajan P, Bick AG, Cook NR, et al. Genetic risk, adherence to a healthy lifestyle, and coronary disease. New England Journal of Medicine. 2016;375(24):2349-58.
- 98. Holick MF. Vitamin D deficiency. N Engl J Med. 2007;357(3):266-81.

- 99. Johannesen EN. Dietary intake in adults with severe mental illness, receiving outpatient treatment in a Scandinavian clinic. 2022.
- 100. Crews M, Lally J, Gardner-Sood P, Howes O, Bonaccorso S, Smith S, et al. Vitamin D deficiency in first episode psychosis: a case-control study. Schizophr Res. 2013;150(2-3):533-7.
- Lally J, Ajnakina O, Singh N, Gardner-Sood P, Stubbs B, Stringer D, et al. Vitamin D and clinical symptoms in First Episode Psychosis (FEP): A prospective cohort study. Schizophr Res. 2019;204:381-8.
- 102. Heaney RP, Dowell MS, Hale CA, Bendich A. Calcium absorption varies within the reference range for serum 25-hydroxyvitamin D. J Am Coll Nutr. 2003;22(2):142-6.
- 103. Itkonen ST, Andersen R, Björk AK, Brugård Konde Å, Eneroth H, Erkkola M, et al. Vitamin D status and current policies to achieve adequate vitamin D intake in the Nordic countries. Scand J Public Health. 2021;49(6):616-27.
- 104. Estruch R, Ros E, Salas-Salvadó J, Covas M-I, Corella D, Arós F, et al. Primary Prevention of Cardiovascular Disease with a Mediterranean Diet Supplemented with Extra-Virgin Olive Oil or Nuts. New England Journal of Medicine. 2018;378(25):e34.
- 105. Lorgeril Md, Salen P, Martin J-L, Monjaud I, Delaye J, Mamelle N. Mediterranean Diet, Traditional Risk Factors, and the Rate of Cardiovascular Complications After Myocardial Infarction. Circulation. 1999;99(6):779-85.
- 106. Martínez-González MA, Gea A, Ruiz-Canela M. The Mediterranean Diet and Cardiovascular Health. Circ Res. 2019;124(5):779-98.
- 107. ernæring Nrf. Kostråd for å fremme folkehelsen og forebygge kroniske sykdommer: metodologi og vitenskapelig kunnskapsgrunnlag. 2011.
- 108. ernæring Nrf. Kostråd om fett- en oppdatering og vurdering av kunnskapsgrunnlaget 2017.
- 109. Cámara M, Giner RM, González-Fandos E, López-García E, Mañes J, Portillo MP, et al. Food-Based Dietary Guidelines around the World: A Comparative Analysis to Update AESAN Scientific Committee Dietary Recommendations. Nutrients. 2021;13(9):3131.
- 110. Henriksen HB, Knudsen MD, Carlsen MH, Hjartåker A, Blomhoff R. A Short Digital Food Frequency Questionnaire (DIGIKOST-FFQ) Assessing Dietary Intake and Other Lifestyle Factors Among Norwegians: Qualitative Evaluation With Focus Group Interviews and Usability Testing. JMIR Formative Research. 2022;6(11):e35933.
- 111. Hege Berg Henriksen MK, Anette Hjartåker, Rune Blomhoff and Monica H. Carlsen. . Relative validity of the new digital food frequency questionnaire (DIGIKOST-FFQ) assessing adherence to the Norwegian food based dietary guidelines and other national lifestyle recommendations. Manus in work. 2023.
- 112. Mishra P, Pandey CM, Singh U, Gupta A, Sahu C, Keshri A. Descriptive statistics and normality tests for statistical data. Ann Card Anaesth. 2019;22(1):67-72.
- 113. Dayabandara M, Hanwella R, Ratnatunga S, Seneviratne S, Suraweera C, de Silva VA. Antipsychoticassociated weight gain: management strategies and impact on treatment adherence. Neuropsychiatr Dis Treat. 2017;13:2231-41.
- 114. Hebert JR, Clemow L, Pbert L, Ockene IS, Ockene JK. Social Desirability Bias in Dietary Self-Report May Compromise the Validity of Dietary Intake Measures. Int J Epidemiol. 1995;24(2):389-98.
- 115. Wehling H, Lusher J. People with a body mass index ≥30 under-report their dietary intake: A systematic review. J Health Psychol. 2019;24(14):2042-59.
- 116. Sankaranarayanan A, Johnson K, Mammen SJ, Wilding HE, Vasani D, Murali V, et al. Disordered Eating among People with Schizophrenia Spectrum Disorders: A Systematic Review. Nutrients. 2021;13(11).
- 117. Totland M, Lundberg-Hallén, Helland-Kigen, Lund-Blix, Myhre, Johansen, Løken, Andersen. Norkost
   3- En landsomfattende kostholdsundersøkelse blant menn og kvinner i Norge i alderen 18-70 år, 2010 11. Helsedirektoratet. 2012.
- 118. Smith GC, Vickers MH, Shepherd PR. Olanzapine effects on body composition, food preference, glucose metabolism and insulin sensitivity in the rat. Archives of Physiology and Biochemistry. 2011;117(4):241-9.
- 119. Hildrum B, Mykletun A, Hole T, Midthjell K, Dahl AA. Age-specific prevalence of the metabolic syndrome defined by the International Diabetes Federation and the National Cholesterol Education Program: the Norwegian HUNT 2 study. BMC Public Health. 2007;7(1):220.
- 120. Janssen I, Katzmarzyk PT, Ross R. Waist circumference and not body mass index explains obesityrelated health risk. The American journal of clinical nutrition. 2004;79(3):379-84.
- 121. Parikh SJ, Edelman M, Uwaifo GI, Freedman RJ, Semega-Janneh M, Reynolds J, et al. The Relationship between Obesity and Serum 1,25-Dihydroxy Vitamin D Concentrations in Healthy Adults. The Journal of Clinical Endocrinology & Metabolism. 2004;89(3):1196-9.

- 122. Mai X-M, Chen Y, Camargo CA, Jr, Langhammer A. Cross-Sectional and Prospective Cohort Study of Serum 25-Hydroxyvitamin D Level and Obesity in Adults: The HUNT Study. American Journal of Epidemiology. 2012;175(10):1029-36.
- 123. Lamberg-Allardt C. Vitamin D in foods and as supplements. Progress in Biophysics and Molecular Biology. 2006;92(1):33-8.
- 124. Regier DA, Farmer ME, Rae DS, Locke BZ, Keith SJ, Judd LL, et al. Comorbidity of mental disorders with alcohol and other drug abuse: results from the Epidemiologic Catchment Area (ECA) study. Jama. 1990;264(19):2511-8.
- 125. Flood-Obbagy JE, Rolls BJ. The effect of fruit in different forms on energy intake and satiety at a meal. Appetite. 2009;52(2):416-22.
- 126. Helsedirektoratet og Tips RJoSD. Fysisk aktivitet i forebygging og behandling [nettdokument]. 2019.
- 127. Hansen BH, Kolle E, Steene-Johannessen J, Dalene KE, Ekelund U, Anderssen SA. Monitoring population levels of physical activity and sedentary time in Norway across the lifespan. Scandinavian Journal of Medicine & Science in Sports. 2019;29(1):105-12.
- 128. Curtis RG, Olds T, Plotnikoff R, Vandelanotte C, Edney S, Ryan J, et al. Validity and bias on the online active Australia survey: Activity level and participant factors associated with self-report bias. BMC Med Res Methodol. 2020;20(1):6-.
- 129. Adams SA, Matthews CE, Ebbeling CB, Moore CG, Cunningham JE, Fulton J, et al. The Effect of Social Desirability and Social Approval on Self-Reports of Physical Activity. American Journal of Epidemiology. 2005;161(4):389-98.
- Winckers AN, Mackenbach JD, Compernolle S, Nicolaou M, van der Ploeg HP, de Bourdeaudhuij I, et al. Educational differences in the validity of self-reported physical activity. BMC Public Health. 2015;15(1):1299-.
- 131. Statistisk Sentralbyrå F. Tobakk i Norge. 2018.
- 132. Norway S. Tobakk i Norge. 2016.
- 133. Pedersen W, von Soest T. Socialization to binge drinking: A population-based, longitudinal study with emphasis on parental influences. Drug and Alcohol Dependence. 2013;133(2):587-92.
- 134. Helsedirektoratet og Tips RJoSD. Anbefaling angående inntak av alkohol ved forebygging av hjerte- og karsykdom. 2019.
- 135. Oquendo MA, Currier D, Liu S-M, Hasin DS, Grant BF, Blanco C. Increased Risk for Suicidal Behavior in Comorbid Bipolar Disorder and Alcohol Use Disorders: Results From the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). J Clin Psychiatry. 2010;71(7):902-9.
  - 136. Boden JM, Fergusson DM. Alcohol and depression. Addiction. 2011;106(5):906-14.
- 137. Ursin L. De fire prinsipper. Store medisinske leksikon. 2021.
- 138. Kwan CL, Gelberg HAL, Rosen JA, Chamberlin V, Shah C, Nguyen C, et al. Nutritional Counseling for Adults with Severe Mental Illness: Key Lessons Learned. Journal of the Academy of Nutrition and Dietetics. 2014;114(3):373-4.
- 139. Lucassen DA, Brouwer-Brolsma EM, Slotegraaf AI, Kok E, Feskens EJM. DIetary ASSessment (DIASS) Study : Design of an Evaluation Study to Assess Validity, Usability and Perceived Burden of an Innovative Dietary Assessment Methodology. Nutrients. 2022;14(6):1156.
- 140. Carlsen MH, Lillegaard IT, Karlsen A, Blomhoff R, Drevon CA, Andersen LF. Evaluation of energy and dietary intake estimates from a food frequency questionnaire using independent energy expenditure measurement and weighed food records. 2010.
- 141. Teasdale SB, Ward PB, Rosenbaum S, Samaras K, Stubbs B. Solving a weighty problem: Systematic review and meta-analysis of nutrition interventions in severe mental illness. British Journal of Psychiatry. 2017;210(2):110-8.

## Appendix 1: Patient case: Controversy of somatic screening in the mental care ward

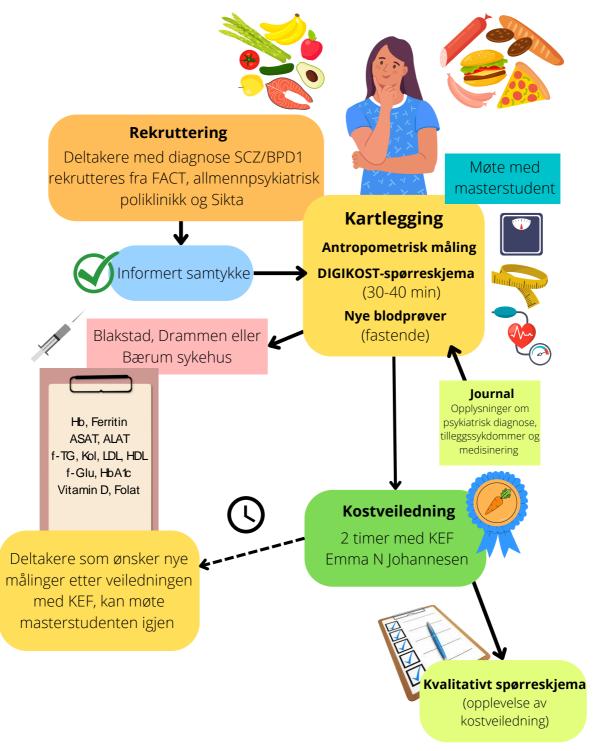
At study initiation, the master student experienced some reluctance among the physicians working in the mental care ward. This was specifically coupled to requisitioning of blood samples in accordance with the study protocol. Some physicians thought it was unnecessary and stressful for the patients to include somatic screening without having a direct medical indication to do so. There were also some physicians who thought that the SMI-patients would not be able to maintain the appointments for blood sampling, especially since they had to be drawn in a fasting state. However, already in the first subject who enthusiastically consented to participate with DIGIKOST-submission and somatic screening, severely elevated blood cholesterol was discovered. The subject at hand was subsequently prescribed with lipid-lowering medicines and enrolled to monitoring of blood cholesterol routinely.



**Figure 15:** Physicians in the psychiatric ward expected patients to be unwilling of somatic screening. The master student experienced however the participants to be interested in gaining more knowledge about their current health status. All participants invited in 2022, except one, were willing to draw new blood samples in accordance with the study protocol.

Appendix 2: Study description (mental care facilities in Asker DPS)

# Hvordan er kostholdet til pasienter med en alvorlig psykisk lidelse?



# VIL DU DELTA I FORSKNINGSPROSJEKTET «HVORDAN ER KOSTHOLDET HOS PASIENTER MED DIAGNOSE SCHIZOFRENI /PSYKOSE- ELLER BIPOLAR AFFEKTIV LIDELSE SOM BEHANDLES PÅ EN ORDINÆR POLIKLINIKK (ASKER DPS)»?

#### FORMÅLET MED PROSJEKTET OG HVORFOR DU BLIR SPURT

Dette er et spørsmål til deg om å delta i et forskningsprosjekt for å studere kostholdet til personer med en alvorlig psykisk lidelse (schizofreni/-psykose eller bipolar affektiv lidelse.)

Din deltakelse vil kunne være med på å bedre nåværende behandlingstilbud, og redusere forekomsten av hjerte- og karsykdom hos personer i denne gruppen, gjennom belysning av behov for ernæringsbehandling.

Vi spør deg om å delta i studien fordi din behandler har definert deg som egnet deltager for prosjektet

#### HVA INNEBÆRER PROSJEKTET FOR DEG?

Deltagelsen i prosjektet er todelt:

- En samtale med masterstudent i ernæring for samtale om kostholdet ditt. Samtalen foregår i form av et intervju, varighet på 45min-1time. Pause kan tas når du får behov for det. Intervjuet gjennomføres på Asker DPS, men kan også gjennomføres som videosamtale hvis du heller ønsker det. Halvparten av deltakerne får kostveiledninger av en klinisk ernæringsfysiolog ved studiens start og den andre halvparten får kostveiledning ved studiens slutt etter 2 måneder
- 2) En måned etter intervjuet vil du bli bedt om å fylle ut et digitalt spørreskjema. Her er det også spørsmål om ditt kosthold, og noen spørsmål om din helse, (f.eks.: vaner knyttet til fysisk aktivitet.) Hvordan utfylling av digitalt spørreskjema gjøres, viser jeg deg i det første intervjuet. Samtidig får du et skriv med informasjon om gjennomføring av det digitale spørreskjemaet. Jeg kan også bistå deg over telefon/video dersom du ønsker veiledning når du fyller ut spørreskjemaet.

I prosjektet vil vi innhente og registrere opplysninger om deg fra din pasientjournal. Kun informasjon relevant til prosjektet hentes ut, eksempelvis din vekt, høyde og andre prøver som er tatt relatert til kosthold/helse (f.eks. vitamin-status, kolesterol, blodsukker og blodtrykk).

#### MULIGE FORDELER OG ULEMPER

Deltagelse i dette prosjektet gir deg en mulighet til å påvirke og optimalisere fremtidig behandling av pasienter. Du vil medvirke til økt fokus på riktig kosthold og på den måten også bidra til at pasienter med alvorlig psykisk lidelse får lavere forekomst av hjerte- og karsykdommer.

Når du deltar i prosjektet, vil du samtidig få en samtale med en masterstudent med kunnskap om ernæring og hvordan kostholdet påvirker kroppen. Det vil være mulig å stille spørsmål om kosthold og ernæring, og om du ønsker det kan du få tilbakemelding og råd knyttet til ditt kosthold. Deltagelse i dette prosjektet vil kreve at du møter opp til intervjuet, enten ved Asker DPS eller på video, i tillegg til utfylling av det digitale spørreskjemaet en måned etter intervjuet. Å snakke om eget kosthold kan oppleves som ubehagelig for enkelte, og dette kan derfor for noen være en ulempe ved å delta. Intervjuformen som skal benyttes, brukes derimot ofte i ernæringsforskning fordi det er en enkel og lite belastende metode for deltageren. Samtalen vil også være et unikt tilbud for deg som pasient ved Asker DPS, som normalt ikke inngår i forløpet, hvor vi kun vil snakke om ernæring og helse.

#### FRIVILLIG DELTAKELSE OG MULIGHET FOR Å TREKKE DITT SAMTYKKE

Det er frivillig å delta i prosjektet. Dersom du ønsker å delta, undertegner du samtykkeerklæringen på siste side. Du kan når som helst, og uten å oppgi noen grunn, trekke ditt samtykke. Det vil ikke ha noen negative konsekvenser for deg eller din behandling hvis du ikke vil delta eller hvis du senere velger å trekke deg.

Dersom du trekker tilbake samtykket, vil det ikke forskes videre på dine helseopplysninger. Du også kan kreve innsyn i opplysningene som er lagret om deg, og opplysningene vil da utleveres innen 30 dager. Du kan også kreve at dine helseopplysninger i prosjektet slettes.

Adgangen til å kreve destruksjon, sletting eller utlevering gjelder ikke dersom materialet eller opplysningene er anonymisert eller publisert. Denne adgangen kan også begrenses dersom opplysningene er inngått i utførte intervjuer.

Dersom du senere ønsker å trekke deg eller har spørsmål til prosjektet, kan du kontakte prosjektleder (se kontaktinformasjon på neste side).

#### HVA SKJER MED OPPLYSNINGENE OM DEG?

Alle involverte i informasjonsinnsamlingen er underlagt taushetsplikt.

Opplysningene som registreres om deg skal kun brukes slik som beskrevet under formålet med prosjektet, og planlegges brukt til 2023. Eventuelle utvidelser i bruk og oppbevaringstid kan kun skje etter godkjenning fra REK og andre relevante myndigheter.

Du har rett til innsyn i hvilke opplysninger som er registrert om deg og rett til å få korrigert eventuelle feil i de opplysningene som er registrert. Du har også rett til å få innsyn i sikkerhetstiltakene ved behandling av opplysningene. Du kan klage på behandlingen av dine opplysninger til Datatilsynet og institusjonen sitt personvernombud.

Alle opplysningene vil bli behandlet uten navn og fødselsnummer eller andre direkte gjenkjennende opplysninger (=kodede opplysninger). En kode knytter deg til dine opplysninger gjennom en navneliste. Det er kun prosjektleder Dawn E. Peleikis så vel som prosjektmedarbeider/mastergradsstudent Madeleine Angelsen som har tilgang til disse. Prosjektleder og mastergradsstudenten er kjent med at de har et selvstendig ansvar vedrørende alle forhold som omtales i Prosedyre for lagring av forskningsdata. Da mastergradsstudenten ikke har et ansettelsesforhold til Vestre Viken, sørger prosjektleder for at nødvendige taushetserklæringer blir underskrevet, og at avtaler som sikrer at Vestre Vikens prosedyrer blir fulgt.

Publisering av resultater er en nødvendig del av forskningsprosessen. All publisering skal gjøres slik at enkeltdeltakere ikke skal kunne gjenkjennes, men vi plikter å informere deg om at vi ikke kan utelukke at det kan skje.

Opplysningene om deg vil bli oppbevart i fem år etter prosjektslutt av kontrollhensyn. Disse lagres i Tjenester for sensitive data (TSD) ved Universitetet i Oslo.

#### KONTAKTOPPLYSNINGER

Dersom du har spørsmål til prosjektet eller ønsker å trekke deg fra deltakelse, kan du kontakte mastergradsstudenten, Madeleine Angelsen: e-post: <u>m.e.angelsen@studmed.uio.no</u> tlf: 47234028

Dersom du har spørsmål om personvernet i prosjektet, kan du kontakte personvernombudet ved institusjonen: <u>personvern@vestreviken.no</u>.

JEG SAMTYKKER TIL Å DELTA I PROSJEKTET OG TIL AT MINE PERSONOPPLYSNINGER BRUKES SLIK DET ER BESKREVET

Sted og dato

Deltakers signatur

Deltakers navn med trykte bokstaver

Appendix 4: List of blood parameters to be tested (physicians in the mental

care ward)

# Blodprøver til deltakelse i kostholdsprosjekt

Rekvireres etter første og andre møte. Må tas fastende

- □ Hemoglobin (Hb)
- □ Ferritin
- $\Box$  ASAT
- □ Fastende Glukose (f-Glu)
- □ Langtidsblodsukker (HbA1c)
- □ Fastende triglyserider (f-TG)
- □ Totalt kolesterol
- □ LDL-kolesterol
- □ HDL-kolesterol
- □ Vitamin D
- □ Folat

## Appendix 5: DIGIKOST FFQ



#### Ditt fødselsnummer \*

I denne undersøkelsen spør vi om dine livsstilsvaner, slik som kosthold, fysisk aktivitet og tobakksvaner.

Ha den siste 1 måneden i tankene når du fyller ut spørreskjemaet:

- Vi er klar over at livsstil varierer fra dag til dag, prøv derfor så godt du kan å gi et gjennomsnitt av dine livsstilsvaner.
- Vi er ute etter ditt vanlige inntak av mat og drikke og din aktivitet per uke i løpet av den siste 1 måneden.

Til å hjelpe deg med å bestemme mengder og porsjoner har vi noen steder lagt inn bilder av porsjoner av ulike typer mat.

Bildene er ikke alltid samme matvare som vi spør om, men en som ligner i størrelse og type.

Det vil ta ca. 15 minutter å fylle ut skjemaet.

Samtykke om deltakelse til å fylle ut DIGIKOST spørreskjema \*

Jeg har lest gjennom informasjonen om spørreskjemaet, og er villig til å delta

## 1. Frukt og bær

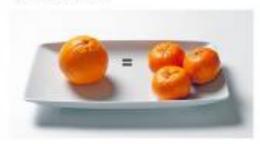
## 1.1 Epler, pærer eller tilsvarende

Hvor mange ganger pr. uke spiser du epler, pærer eller tilsvarende? \*

Her kan du oppgi det du spiser rå, i matlaging og/eller som pålegg.

O Aldri/Sjelde	n 01	(	2	O 3
O 4	0 5	(	) 6-7	85 O
Hvor mange e	pler, pære <mark>r e</mark> ll	ler tilsvarende s	spiser du hver	gang? *
	valgt i spørsmål	ersom alternativet et «Hvor mange ga		
Et vanlig eple eller	pære veler ca.	135 gram		
O 1/2 stit	O 1 stk	O 2 stk	O 3 stk	O ≥ 4 sik

# 1.2 Appelsin

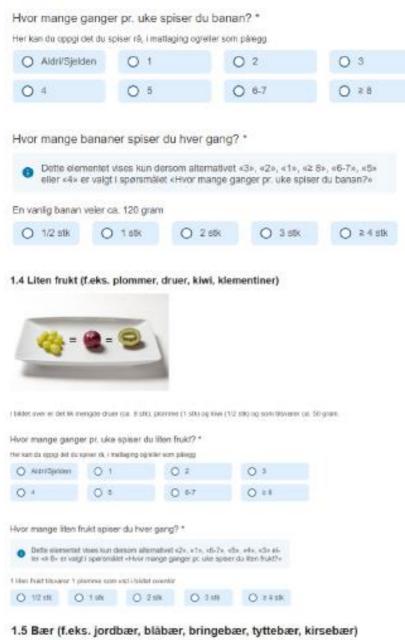


Bildet over viser en vanlig appelsin (195 gram) som tilsvarer ca. 3 små klementiner

Hvor mange ganger pr. uke spiser du appelsiner? \*

O Aktri/Sjelden	01	C	2	O 8
O 4	O 5	C	6-7	O ≥8
Vor mange van	un com veses	rsom alternativet +	2 8×, «6-7», «6×,	
	Last 1 minuteroran Miles	<ul> <li>A star in particular star</li> </ul>	CONTRACTOR DISTORT	
eller «t» er va appelsiner?»	lgt i spørsmåle	( «Hvor mange ga	iger pr. uke spise	r du

## 1.3 Banan





Smoothie inngår også i dette spørsmålet.

Her kan du rapportere froane og ferske bær, og bær som er rørt ut i litt sukker.

Hvor mange ganger pr. uke spiser du bær? \*

O Alcri/Sjeiten	01	0 2	O 3	
0 4	O 5	O 6-7	0 88	

Hvor mye bær spiser du hver gang? \*

 Dette elementet vises kun dersom alternativet «1», «5», «4», «3», «2», «2 8» elter «6-7» er valgt i spæramålet «Hvor mange ganger pr. uke spiser du bær?»

Bærene i bildene nedenfor er servert i en suppetallerken (17cm)

Det ligger en spiseskje i hver talierken.





O A = ca. 50 gram

O B = ca. 100 gram





O C = ca.150 gram

O D = ca. 260 gram

#### 2. Notter

livingår nøtter som en del av ditt ukentlige kosthold? \*

```
O AR
```

 Dette elementet visee kan gesont abervativet «ale» er seigt i spersmillet sinngår nøfter som en del av dit ukentlige kosholich»



 Dette stenentint vises kun dessen alternativet «Ja» er saigt i spenendist «Inngår netter som en dat av dit skantlige kostiskist?»

Bilder til vervetre viter ca. 20 gran notfer (=1 neve) Bilder til køyne viter ca. 142 gran notfer (=7 neve) Etet ligger en toskjo på Iver tafterion

Vi vil fenit spære deg ne usaitede nieter og deretter salteda nøtter.

 Dette elementet vises kan cessors alternativet «Ja» er valgt i spertvrålet «inngår natter som en del av ditt ukentige kosthold?»

## 2.1 Usaltede nøtter (f.eks. mandler, valnøtter, cashewnøtter, ferdige nøtteblandinger, peanøtter)

Hvor mange ganger pr. uke spiser du usaltede nøtter? \*

0	<ul> <li>Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår nøtter som en del av ditt ukentlige kosthold?»</li> </ul>							
0	AldrifSjelden	01	O 2	0 3				
0	4	0 5	0 67	() ≥8				
Hvor	mye usaltede	nøtter spiser du hver	r gang? *					
0		vises kun dersom alternati ti spørsmålet: «Hvor mange						
0	1-2 never	O 3-4 never O	5-6 never () ×7	' never				
0	<ul> <li>Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår nøtter som en del av ditt ukentlige kosthold?»</li> </ul>							
0	Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår nøtter som en del av dtt ukentlige kosthold?»							
	2.2 Saltede nøtter (f.eks. peanøtter, chilinøtter, ferdige nøtteblandinger, pekannøtter, cashewnøtter)							

#### Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår nøtter som en del av dit ukentlige kosthold?» O a Aldri/Sjelden O 2 $O_{-1}$ O 4 Ó 5 O 6-7 () ≥ 8 Hvor mye saltede nøtter spiser du hver gang? \* Dette elementet vises kun dersom alternativet «z 8», «6-7», «6», «4», «3», «2» 0 eller «1= er valgt i spørsmålet «Hvor mange ganger pr. uke spiser du saltede noter?+ O 3-4 never O 5-6 never ○ ≥7 never. 1-2 never

Hvor mange ganger pr. uke spiser du saltede nøtter? \*

#### 3. Grønnsaker

#### Vi vil nå spørre deg om de grønnsakene du vanligvis spiser.

Det kan være at du spiser en blanding av det vi spar deg om. Et tips er da å velge litt av hver type grønnsak, så det tilsammen stemmer med det du spiser.

#### 3.1 Gulrot

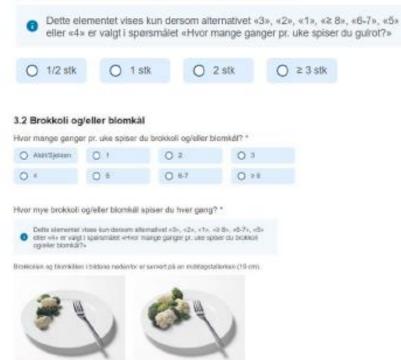


Bildet viser en værlig gulrut og revet gulrut som tilsværer 80 grunn i mengde Gulrates er servert på en middagstallerken (19 cm) Det ligger en spissesivje på tatlanken.

Hvor mange ganger pr. uke spiser du gulrot? \*

O Aldnitsjelden	01	0 2	O 3
0.4	0 0	O 6-7	O ≥ð

Hvor mange vanlige gulrøtter spiser du hver gang? \*



And the second second

O Arrea 50 gram

C Brick 100 gram



#### 3.3 Tomat



Bildet over viser friske tomater der en vanlig tomat (95 gram) tilsvarer 6-7 små cherrytomater

vor mange gange	er pr. uke spiser du	tomater? *	
Aldri/Sjelden	01	O 2	0 3
04	0 6	O 6-7	0 28
vor mange vanlig	te tomater spiser du	uhver gang? *	
<ul> <li>Dette elementet eller «4» er valg</li> </ul>	vises kun dersom altern t i sparsmålet «Hvor ma	tativet <3a, +2a, +1a, ei	er du tomater?»
	a separation of the time	uße Beißer hit eine abra	of the company of

#### 3.4 Tomatprodukter (f.eks. tomatsaus, hermetiske tomater, ketchup)

Hvor mange ganger pr.uke spiser du tomatprodukter \*

O 1/2 slk O 1 slk O 2 slk O ≥ 3 slk

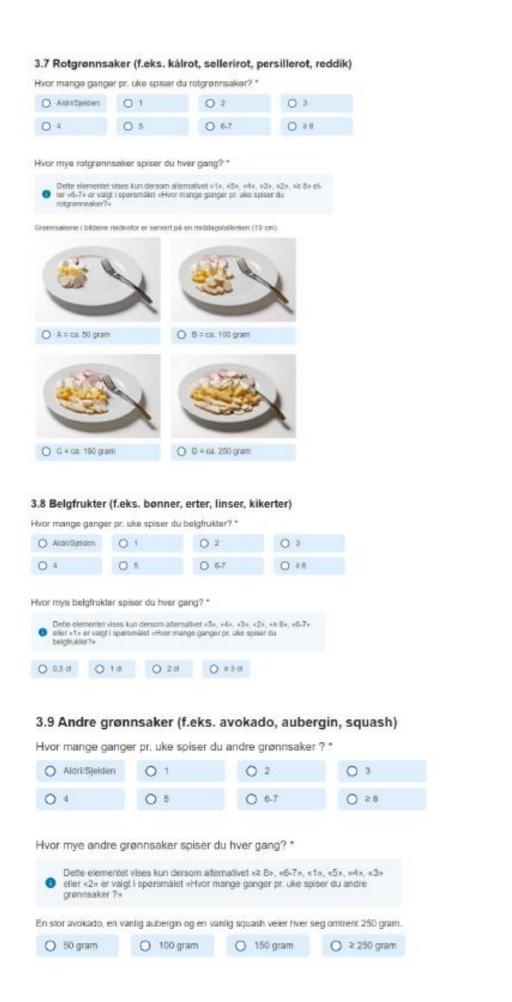


## 3.5 Løk, vårløk og purreløk

Inngår løk, purreløk ogreller vårlek som en del av ditt ukentlige kosthold? \*

O fiel				
Hvor mange ganger	pr. uke spiser (	du lek, värlek ogjelle	er purnelæk? *	
Dotta elementet e tak, purrelek ogni	tees kun dennen of Ner viktak som en s	ternstävet «Jas er salgt i s det av citt skentlige koede	operumblot + megde old?+	
O Astronom	01	0 2	O 5	
O 4	O 8	D 67	C) 2.8	
	up-provident +Hsice			
<ul> <li>alter &lt;2x ar o og/eller pum</li> </ul>	valgt i aperande ekek?x ig lek og revert le i en middagstale	et «Hvor mange gang k som tilsværer 150 g skan (10 cm).	8a, a6.7a, e1a, e5a, e4a ar pr. uko apikar du lek, y ara i mangda	
contribute and strate	odia dana bia maina			
Hvor mye hver	nama enicor o			
	Recif stysel r	du lek, värlek og/	eller purreløk? *	
Dette eleme	olat visas kun di valgt i sporanda	ersom alternativet ⊲≥	eller purreløk? " da, så 7a, ots, såa, ola jerpt uke spiserdu løk, v	
Dete eleme eler «2» er	olat visas kun di valgt i sporsmåk	ersom alternativet ⊲≥	da, ado7a, eta, ada, eda	
Dete eleme     eler <2+ er     og/eller pum	olat visas kun di valgt i sporsmåk	ersom alternativet ⊲≥	da, ado7a, eta, ada, eda	
Derte elerno     eler «2» er     og/eler pun t ss lek = 10 gean     O 1 as	ntet visees kum di valgt i sportsmåk elak Tis	ersom alternativet «2 et «Hvor mange gang	dia, x6-7a, eta, x5a, e4a erpt. uke spiser du lek, v	
Dente elerne eler x2x er og/eler pun t ss lok = 10 gram O 1 ss 3.6 Blandet sa	nlet vises kun di valgt i sporsmåk elak?> Q 2 ss ilat	ersom atemativet «2 et «Hvor mange gang O 3 ss	Ba, eði,7a, cta, eða, eða er pr. uke spiser du lek, v ⊖ ±4 ss	
Dente eleme     elem 42e er     og/elet pun      tiss lek = 10 gran      1 as      3.6 Blandet sa  Hvor mange gang	ntet viceos kun di valgti apotsmåk () 2 ss klat per pr. uke spis	ersom alternativet «2 et «Hvor mange gang O 3 ss ser du blandet sela	Ba, edi-7a, eta, eta, eda, eta erpt: uke spiser du lek, v ○ 2:4 ss st7 *	
Derte elerne eler x2x er og/elet pun t ss lak = 10 gran O 1 ss     135     3.6 Blandet sa Hvor menge gang     O Akaridjeden	ntet viewe kun d valgt i spersmåk elak?> Q 2 ss klat per pr. uke spit	ersom alternativet «2 et «Hvor mange gang O 3 as ser du blandet sala	Ba, eði 7a, cha, eða, eða er pr. uke apiser du lek, v O 2.4 ss st7 *	
Dente eleme     elem 42e er     og/elet pun      tiss lek = 10 gran      1 as      3.6 Blandet sa  Hvor mange gang	ntet viceos kun di valgti apotsmåk () 2 ss klat per pr. uke spis	ersom alternativet «2 et «Hvor mange gang O 3 ss ser du blandet sela	Ba, edi-7a, eta, eta, eda, eta erpt: uke spiser du lek, v ○ 2:4 ss st7 *	
Device element     element     opticlet puin     tiss left = 10 grann     1 as     1 as     16 Blandet sa     Hvor mange gang     Austrägeten     0 4	olet views kun d valgt i spotsmåk clasify 2 ss liat per pr. uke spit 0 1 0 3	erson atemativet «2 et «Hvormange gang O 3 ss ser du blandet sela O 2 O 8-7	Ba, eði 7a, cha, eða, eða er pr. uke apiser du lek, v O 2.4 ss st7 *	
Defe eleme eler x2x er ogletet pun t ss lok = 10 gam 1 ss 3.6 Blandet sa Hvor menge gang Acardiston 4 Hvor mye blandet	ntet viese kun d valgt i sportsmåk elak?> 0 2 ss lat per pr. uke spid 0 1 0 3 salat spiser 0	ersom alternativet -2 et «Hvor mange gang O 3 ss ser du blandet sale O 2 O 6-7 Au hver gang? *	Ba, edi.7a, c1a, e5a, ota, er pt. uke apiser du lek, v O 2.4 ss at7 * O 3 O 4.8	
Dette eleme     dete x2x et a     optietet puin     tiss lek = 10 grant     1 as     1 as     16 Blandet sa Hvor mange gang     Ausitägeten     0 4 Hvor mye blandet     Dete elemente	ntet visees kun di valgt i spotsmåk () 2 ss klat () 1 () 3 () 3 () 3 () 3 () 3	erson atemativel 42 erson atemativel 42 et «Hvormange gang 0 3 ss ser du blandet sela 0 8-7 tu hver gang? * m atemativet «14, «5	Ba, eði 7a, cha, eða, eða er pr. uke apiser du lek, v O 2.4 ss st7 *	
Dete elema     der x2 er     opteter pun      tiss lak = 10 gran      1 as      1 as      16 Blandet sa      Hvor mange gang      Austisjetten      4      Dete elemente     lier s5 To er vol     selatTo	ntet views kun di valgt i spotsmåk () 2 ss lat per pr. uke spit () 1 () 3 () 3 () 3 () 3 () 3 () 3 () 3 () 3	erson atemativel 42 erson atemativel 42 et «Hvormange gang 0 3 ss ser du blandet sela 0 8-7 tu hver gang? * m atemativet «14, «5	Ba, edi-7a, efa, edia, edia erpt: uke spiker du lok, v ○ 2435 alf7* ○ 3 ○ 3 ○ 3 ○ 3 ○ 3 ○ 4 3 ○ 4 3 ○ 4 0 3 ○ 3	
Dete elema     der x2 er     opteter pun      tiss lak = 10 gran      1 as      1 as      16 Blandet sa      Hvor mange gang      Austisjetten      4      Dete elemente     lier s5 To er vol     selatTo	ntet views kun di valgt i spotsmåk () 2 ss lat per pr. uke spit () 1 () 3 () 3 () 3 () 3 () 3 () 3 () 3 () 3	ersom alternativet «2 ersom alternativet «2 et «Hvormange gang 0 3 ss eer du blandet sala 0 2 0 8-7 hu liver gang? * maternativet «1% «5	Ba, edi-7a, efa, edia, edia erpt: uke spiker du lok, v ○ 2435 alf7* ○ 3 ○ 3 ○ 3 ○ 3 ○ 3 ○ 4 3 ○ 4 3 ○ 4 0 3 ○ 3	
Dete elema     der x2 er     opteter pun      tiss lak = 10 gran      1 as      1 as      16 Blandet sa      Hvor mange gang      Austisjetten      4      Dete elemente     lier s5 To er vol     selatTo	ntet views kun di valgt i spotsmåk () 2 ss lat per pr. uke spit () 1 () 3 () 3 () 3 () 3 () 3 () 3 () 3 () 3	ersom alternativet «2 ersom alternativet «2 et «Hvormange gang 0 3 ss eer du blandet sala 0 2 0 8-7 hu liver gang? * maternativet «1% «5	Ba, edi-7a, efa, edia, edia erpt: uke spiker du lok, v ○ 2435 alf7* ○ 3 ○ 3 ○ 3 ○ 3 ○ 3 ○ 4 3 ○ 4 3 ○ 4 0 3 ○ 3	
Dete elema     der x2 er     opteter pun      tiss lak = 10 gran      1 as      1 as      16 Blandet sa      Hvor mange gang      Austisjetten      4      Dete elemente     lier s5 To er vol     selatTo	Inter views kun di valgt i spotsmåde clais 75. Q 2 35 latt per pr. uke spit Q 1 Q 3 salat spiser 0 et vises kun derso gi i sponsmådel al snedsvilk er serv	ersom alternativet «2 ersom alternativet «2 et «Hvormange gang 0 3 ss eer du blandet sala 0 2 0 8-7 hu liver gang? * maternativet «1% «5	As, still 7s, c1s, c5s, c4s erpt uke spiker du lok, v 0 2435 at7 * 0 3 0 48 s, s4s, s2s, at 8s et- uke spiker du biordel ries (19 cm).	

O C = ca. 100 gram



#### 4. Brød, knekkebrød og pålegg

Brødskalaens fire kategorier har vi satt inn for å hjelpe deg å vurdere grovheten på brødproduktene du spiser.









25-50% sammalt mel/hele kom



50-75% sammalt mel/hele kom

75-100% sammalt mel/hele kom

## 4.1 Brød og knekkebrød

# Hvor mye brød og knekkebrød spiser du?

## I dette spørsmålet skal du oppgi antall skiver pr. dag

Mengden oppgis i antall skiver:

1/2 rundstykke = 1 skive; 1 baguett = 4 skiver; 1 ciabatta = 2 skiver; 1 knekkebrød= 1 skive

	0	1	2	з	4	5	6	7	8	9
Fint bred, 0-25% sammalt mel (t.eks. loff, fine rundstyk- ker, clabatta)	0	0	0	0	0	0	0	0	0	0
Halvgrovt bræd, 25-50% sam- malt mel (f.eks. helikombrød, kneip, grove rundstykker)	0	0	0	0	0	0	0	0	0	0
Grovi bred, 50-75% sammat mei (T.eks.havrebrad)	0	0	0	0	0	0	0	0	0	0
Ekstra grovbred, 75-100% sæmmalt mel (Leks. merkt rugbred)	0	0	0	0	0	0	0	0	0	0
Pint knakkebrad (f.aks. Kav- ring, fickost kneikebrad)	0	0	0	0	0	0	0	0	0	0
Grovi knekkebrad (f.eks. Huaman, Sport, Solrufa)	0	0	0	0	0	0	0	0	0	0
4										÷.

VI har regnet ut at du bruker 0 brødskiver og knekkebrød per uke

# 4.2 Pålegg

Man kan bruke flere pålegg pr. brødskive eller knekkebrød

Hvor mye pålegg har du vanligvis på de 0 brødskivene og eller knekkebrødene (viser til antall brødskiver og knekkebrød per uke beregnet i spørsmålet ovenfor)?

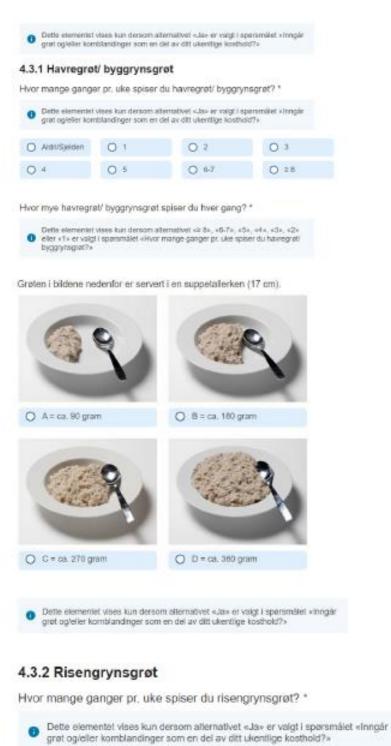
	0	1-3 skiver	4-7 skiver	8-12 skiver	13-18 skiver	19-25 skiver	$\ge 26$ skiver
l'ete oster som pålegg (Leks. helfet Norvagia, halfet Jarisberg, brunost, prim, brie) *	0	0	0	0	0	0	0
Magre oster som pålegg (f.eks. lett Norvegla, lett Jartsberg, cottage choose) *	0	0	0	0	0	0	0
Fiskepålegg (f.eks. makrell i tornat, roket/gravet laks, sikt) *	0	0	0	0	0	0	0
Rødt kjøtt (f.eks. salami, skinke, servelat, leverpostel) *	0	0	0	0	0	0	0
Hvitt kjätt (Leks. kytlingpålegg, kalk- unpålegg, kytlingleverpostel) *	0	0	0	0	0	0	0
Pålegg med suiker (Leks. honning, syttetøy, nottepälegg) *	0	0	0	0	0	0	0
Egg (kokt, steldt, eggerøre) *	0	0	0	0	0	0	0

# 4.3 Grøt og kornblandinger

Inngår grøt og/eller kornblandinger som en del av ditt ukentlige kosthold? \*

0	Ja
0	Nei

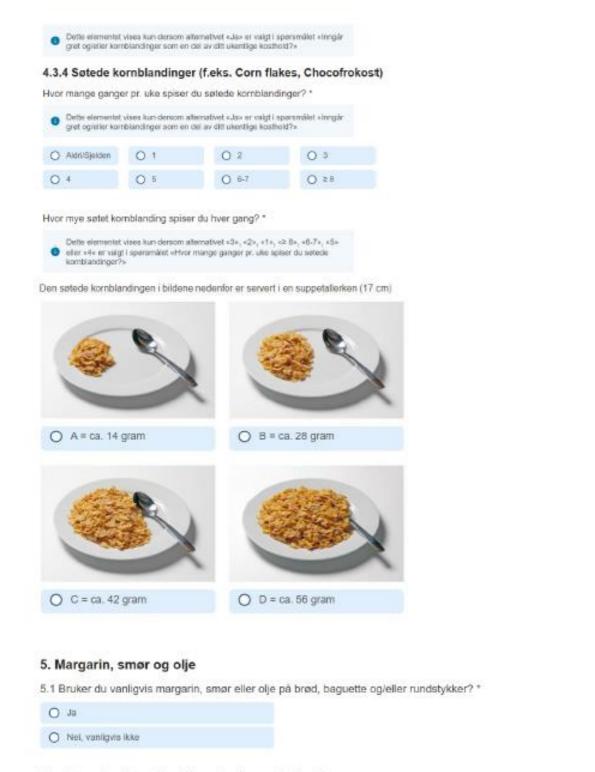
Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår grøt og/eller komblandinger som en del av ditt ukentlige kosthold?»



O Aldri/Sjelden	01	O 2	O 3
O 4	O 5	O 6.7	O 28

Hvor mye risengrynsgrøt spiser du hver gang? \*





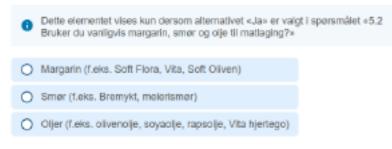
Hva bruker du oftest på brød, baguette eller rundstykker? \*



5.2 Bruker du vanligvis margarin, smør og olje til matlaging? \*



Hva bruker du oftest til matlaging? \*



# 6. Fisk

Inngår fisk som en del av ditt ukentlige kosthold? \*

O Ja		
O Nei		

 Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår fisk som en del av ditt ukentlige kosthold?»

Vi vil først spørre om fet fisk, deretter mager fisk og bearbeidet fisk.

 Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår fisk som en del av ditt ukentlige kosthold?»

# 6.1 Fet fisk (f.eks. laks, ørret, sild, kveite)

Hvor mange ganger pr. uke spiser du fet fisk? \*





 Dette elementet vises kun dersom alternativet «Ja» er valgt i sparsmålet «Inngår fak som en det av dtt ukentige kosthokt?»



Obligatoriske felter er merket med stjerne \*

# Kjøtt

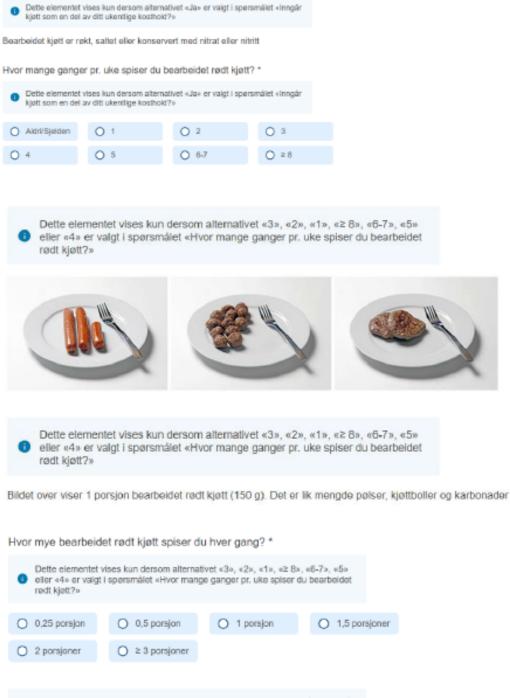
Inngår kjøtt som en del av ditt ukentlige kosthold? \*

0	Ja
0	Nei

#### 7.1 Rødt kjøtt (f.eks. storfe, svin eller sau/lam)



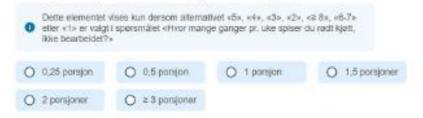
#### 7.1.1 Bearbeidet rødt kjøtt (f.eks. pølser, hamburger, kjøttboller, sommerkotelett, kjøttdeig)



 Dette eiementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «inngår kjøtt som en del av ditt ukentlige kosthold?»

Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår 0 kjøtt som en del av ditt ukentlige kosthold?» 7.1.2 Rødt kjøtt, ikke bearbeidet (f.eks. biff, stek, grytekjøtt) Hvor mange ganger pr. uke spiser du rødt kjøtt, ikke bearbeidet? \* Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår 0 kjøtt som en del av ditt ukentlige kosthold?» 0 3 Aldri/Sjelden 0 2 01 0 28 0 6.7 04 0 5 Dette elementet vises kun dersom alternativet «5», «4», «3», «2», «2 8», «6-7» eller «1» er valgt i spørsmålet «Hvor mange ganger pr. uke spiser du rødt kjøtt, 0 likke bearbeidet?» Dette elementet vises kun dersom alternativet «5», «4», «3», «2», «2 8», «6-7» 0 eller «1» er valgt i sporsmålet «Hvor mange ganger pr. uke spiser du rødt kjøtt, likke bearbeidet?> Dette bilde viser 1 porsjon rødt kjøtt, ikke bearbeidet, tilsvarende 150 gram og som er servert på en middagstallerken (19 cm).

Hvor mye rødt kjøtt, ikke bearbeidet, spiser du hver gang? \*





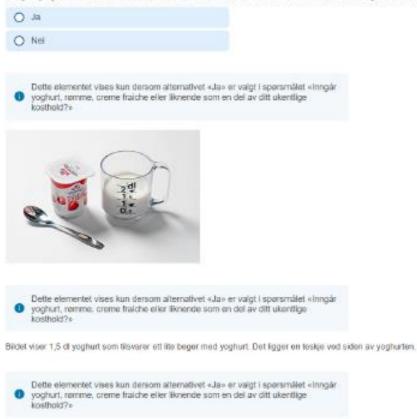
#### 7.2.2 Hvitt kjøtt, ikke bearbeidet (f.eks. kyllingfilet, kalkunbrystfilet)



Obligatoriske felter er merket med sterne \*

#### 8.0 Yoghurt, rømme, creme fraiche eller liknende

Inngår yoghurt, rømme, creme fraiche eller liknende som en del av ditt ukentlige kosthold? \*



8.1	Lett yoghurt (f.eks. all youghurt me	d "lett",	"0%", "0,1%"	" i navnet el-
ler	yoghurt naturell)			

Hvor mange ganger pr. uke spiser du lett yoghurt? \*

•		es kun dersom alternat erne fraiche eller liknen				
0	Aldri/Sjelden	0 1	O 2	0 3		
0	4	0.5	O 4-7	0 =0		
Hvor		t spiser du hver ga	-			
0		es kun detsom alternat iperamblet «Hvor mang				
0	0,5 di O 1	d () 1,5 di	0 20	O -2,5 dI (	0.65	
0		es kun dersom afternat erne fraiche eller Tiknen				
0		vises kun dersom alt creme fraiche eller li			nngår	
8.2	Yoghurt (f.e	eks. God more	gen yoghurt,	fruktyoghu	rt)	
Hvor	r mange gange	er pr. uke spiser d	lu yoghurt? *			
		vises kun dersom alt			nngår	
•	yoghurt, rømme, kosthold?»	oreme fraiche eller li	knende som en del	av ditt ukentlige		
0	Aldri/Sjelden	0.1	O 2	0	3	
0	4	0 5	0 6-7	0	≥ 8	
Hvor	Dette elementet	spiser du hver ga vises kun dersom alt	emativet «3», «2»,			
Ť	eller +4> er valgt	l i spørsmålet «Hvor i	nange ganger pr. u	ke spiser du yoghi	at?×	
0	0,5 dl O	1 d () 1,	5 di 🔿 2 di	O 2,5 c	1 () ×3	dI
0		vises kun dersom alt creme traiche eller l			Inngår	
9. R	ømme, crei	me fraiche og	liknende			
	Dette elementet	vises kun dersom alt creme fraiche eller l	ternativet «Ja» er vi		imgår	
		eme fraiche o er inneholder			f.eks. lettra	əmme, ma-
-		ar pr. uke spiser (			ned lavt fettin	nhold? *
0		vises kun dersom alt creme fraiche eller i			Inngär	
	Provide rule of a			_		
0	Aldri/Sjelden	01	O 2	0	3	
0	4	0 5	0 6-7	0	≥8	
0	ler x6-7> er valg	vises kun dersom alt t i spørsmålet «Hvor I. med lavt fettinnhold	mange ganger pr. u			

	9	>		
		om alternativet «1».		
creme traid	he où, med lavt fetti		r pr. uke spiser du	namme,
Seichet open visien en	s spineskje (i s) med	rateria		
Hvor mye rømr	me, creme fraict	he o.l. med lavt f	ettinnhold spis	er du hver gang
0 lat +6-7 + at		on alternativel «1», drtsor mænge gange rehold?»		
Oppgi mengde i	spisæskjæer (ss)			
O 0.5 ss	Q 198	O 11/2 ss	O 288	O 3 88
O 24m				
		nalvet «Jae or valgt i s		
kathold?v	, creme lisk be whet like	nende kom en det av de	(Lines)gr	
Dette elementat	charge by in elastic care other	and the second se	A second s	
		nande sotroan del av di		
<ul> <li>yoghun, tarrensi konfhotti?»</li> </ul>	, creme hukde, eller lik		sukenlige	old (f.eks. set
<ul> <li>yoptan, tarress kontrott?s</li> <li>2 Remme, cr</li> </ul>	eme fraiche og	nande sotrikih del av di	høyt fettinnho	· · · · · · · · · · · · · · · · · · ·
2 Remme, creme	eme fraiche og fraiche, eller i	nande som an del av di I likmende med	høyt fettinnho enn 20% fett)	28
yoghut, terrete kestbedfe      Remme, cre mme, creme vor mange gange      Dete terretet      yoghut, seme	erne fraiche og fraiche, eller is er pr. uke spioer di vises kun denom alter	i liknende med nneholder mer	høyt fettinnho enn 20% fett) liche o.l. med høy ponndet skrigte	28
yogtut, termin kothot/le     Romme, cr mme, creme vor mange gange     Date emerant	erne fraiche og fraiche, eller is er pr. uke spioer di vises kun denom alter	) liknende med nneholder mer stamme, creme ha matvet «Ja- er satzt i s	høyt fettinnho enn 20% fett) liche o.l. med høy ponndet skrigte	28
yoghut, terrete kestbedfe      Remme, cre mme, creme vor mange gange      Dete terretet      yoghut, seme	erne fraiche og fraiche, eller is er pr. uke spioer di vises kun denom alter	) liknende med nneholder mer stamme, creme ha matvet «Ja- er satzt i s	høyt fettinnho enn 20% fett) liche o.l. med høy ponndet skrigte	28
yoduk, same kasheddis      Remme, cr emme, creme vor mange ganp vor mange ganp voduk, same koshednis      Austigeden	erne fraiche og fraiche, eller is er pr. uke spioer di vises kun denom alter	I likmende worden del av di I likmende med nneholder mer I terrame, creme fra I terrame, creme fra I terrame, creme fra I terrame, creme del av di	hoyt fettinnho enn 20% fett) Note o.l. med høy permiske stregte nukestige	28
yodur, umme kathetifs 2 Remme, cr emme, creme wor mange ganp Oate eareveat yodur, some keekeams Austiteden Austiteden	erne hidde eller in eme fraiche og fraiche, eller i er pr. uke spiser du vien kun derton alle orere flakte eller ik O 1	Iliknende med nneholder mer stamme, oreme ha retivet «Ja» er valar in retide son en de av de	hoyt fettinnho enn 20% fetti kishe o.l. med hay pannake «tinge ti westige O 0 0 2	t fetlinnhold? *
yogdua, samme katheaths 2 Remine, creme mine, creme wor mange ganp      Dete Herrestat     yogdua, samme kerbewiths      Assistenden      +	eren hicke ekrik eme fraiche og fraiche, eller i er pr. uke spiser du vise kun denon alte dere hicke ekrik O 1 O 1 O 5	Iliunende med nineholder mer a tarame, oreme ha retivet «Ja» er vagt i a retivet «Ja» er vagt i a	høyt fettinnh enn 20% fetti kiste oll med høy pomiler «tropir t verstige 0 s 0 s 0 s 0 s 0 s 0 s 0 s 0 s 0 s	t fetlinnhold? *
yogduar, samme kastheaths     2 Remme, crismine, creme wor mange gamp Dete Hernester yogduar, samme keebearths     Association     4     Association     4     Association     Associatio     Association     Associatio     Association     Association	eren hicke eller is eme fraiche og fraiche, eller is er pr. uke spiser du vies kun denom alte derre fraiche eller is O 1 O 5 come fraiche o.i. vies hun denom alte	Iliunende med nineholder mer a tamme, oreme ha retivet «da» er vagt i a retivet «da» er vagt i a	høyt fettinnh enn 20% fetti kiste oll med høy pomiler «tropir t verstige 0 s 0 s 0 s 0 s 0 s 0 s 0 s 0 s 0 s	t fetlinnhold? *
yogduar, samme kastheaths     2 Remme, crismine, creme wor mange gamp Dete Hernester yogduar, samme keebearths     Association     4     Association     4     Association     Associatio     Association     Associatio     Association     Association	erren halde eller in eme fraiche og fraiche, eller in er pr. uke spiser du vien kun denon alle overe fraiche eller in O 1 O 2 come fraiche ol, vien kun denon alle	Iliunende med nineholder mer a tamme, oreme ha retivet «da» er vagt i a retivet «da» er vagt i a	hayt fettimhin enn 20% fetti kithe oll med hay parmäke «tropie ti veetige 0 s 0 sa 0 sa 1 spisser du hver scale, stale, sta	t fetlinnhold? *
yogdaa, samme kastbastins 2 Remme, crieme minne, creme vor mange gange vor mange gange vor ange gange vor ange gange vor ange gange vor ange gange vor ange gange vor mye samme. or mye samme. or mye samme. or mye samme.	eren hicke eller in eme fraiche og fraiche, eller i er pr. uke opiser du vise kun denon alte oere fraiche eller is O 1 O 5 cheme fraiche ol. vise hun denom viser i gemmane viser	Iliunende med nineholder mer a tamme, oreme ha retivet «da» er vagt i a retivet «da» er vagt i a	hayt fettimhin enn 20% fetti kithe oll med hay parmäke «tropie ti veetige 0 s 0 sa 0 sa 1 spisser du hver scale, stale, sta	t fetlinnhold? *
yoghur, samme kestheutins      Remme, creme mmme, creme mmme, creme vor mange ganp      Dete elementat     yoghur, samme kesteurins      Austitieden      er mye samme.      Dete elementat      vor mye samme.      Dete elementat      of elementation      of elementation      O. Ris og pa	eren hicke eller in eme fraiche og fraiche, eller in er pr. uke spiser du vies kun denom alle dere fraiche eller in O 1 O 5 come fraiche ol. vies kun denom alle come fraiche ol.	Iliunende med nneholder mer a tarame, oreme ha nativet «da» er vagt in rende som en de av de o av o av o av o av med høyt kellinnho nativet «da, säx, «da, nativet «da, säx, «da, nativet «da, säx, «da,	hoyt fettinnh enn 20% fetti kite ol. med hay parmäler stropp t verstige 0 = 0 0 = 0 0 = 0 kite ol. solar ki spiser du hver solar, solar, so	t fetlinnhold? *
yogdua, samme katheaths 2 Remme, creme samme, creme wor mange ganp      Deto Herrestat     yogdua, samme kesheaths      AustBecken      +      Deto Herrestat     yogdua, samme kesheaths      AustBecken      +      Deto Herrestat     conservation      AustBecken      -      AustBecken      -      C. Ris og pa angder ris eller p	eren hicke eller in eme fraiche og fraiche, eller in er pr. uke spiser du vies kun denom alle dere fraiche eller in O 1 O 5 come fraiche ol. vies kun denom alle come fraiche ol.	Iliunende med nineholder mer a tamme, oreme ha retivet «da» er vagt i a retivet «da» er vagt i a	hoyt fettinnh enn 20% fetti kite ol. med hay parmäler stropp t verstige 0 = 0 0 = 0 0 = 0 kite ol. solar ki spiser du hver solar, solar, so	t fetlinnhold? *
yogdua, samme kasteatins 2 Remine, creme mine, creme wor mange ganp Dete elemental yoghus, same keeven Austigeden Austigeden  Austigeden	eren hicke eller in eme fraiche og fraiche, eller in er pr. uke spiser du vies kun denom alle dere fraiche eller in O 1 O 5 come fraiche ol. vies kun denom alle come fraiche ol.	Iliunende med nneholder mer a tarame, oreme ha nativet «da» er vagt in rende som en de av de o av o av o av o av med høyt kellinnho nativet «da, säx, «da, nativet «da, säx, «da, nativet «da, säx, «da,	hoyt fettinnh enn 20% fetti kite ol. med hay parmäler stropp t verstige 0 = 0 0 = 0 0 = 0 kite ol. solar ki spiser du hver solar, solar, so	t fetlinnhold? *
yogdua, samme katheaths 2 Remme, creme samme, creme wor mange ganp      Deto Herrestat     yogdua, samme kesheaths      AustBecken      +      Deto Herrestat     yogdua, samme kesheaths      AustBecken      +      Deto Herrestat     conservation      AustBecken      -      AustBecken      -      C. Ris og pa angder ris eller p	eren hicke eller in eme fraiche og fraiche, eller in er pr. uke spiser du vies kun denom alle dere fraiche eller in O 1 O 5 come fraiche ol. vies kun denom alle come fraiche ol.	Iliunende med nneholder mer a tarame, oreme ha nativet «da» er vagt in rende som en de av de o av o av o av o av med høyt kellinnho nativet «da, säx, «da, nativet «da, säx, «da, nativet «da, säx, «da,	hoyt fettinnh enn 20% fetti kite ol. med hay parmäler stropp t verstige 0 = 0 0 = 0 0 = 0 kite ol. solar ki spiser du hver solar, solar, so	t fetlinnhold? *
yogduc, samme katheaths 2 Remme, creme semme, creme vor mange ganp      Dete emresel     vor mange ganp      AustBecker      AustBecker      enters      AustBecker      enters      AustBecker      come fastbeck      O. Ris og pa  mgår ris eller p      Ja	eren hicke eller in eme fraiche og fraiche, eller in er pr. uke spiser du vies kun denom alle dere fraiche eller in O 1 O 1 O 5 comme fraiche ol. vies kun demon alle comme fraiche ol.	Iliunende med nneholder mer a tarame, oreme ha nativet «da» er vagt in rende som en de av de o av o av o av o av med høyt kellinnho nativet «da, säx, «da, nativet «da, säx, «da, nativet «da, säx, «da,	hoyt fettinnh enn 20% fetti kite ol. med hay parmäler stropp t verstige 0 = 0 0 = 0 0 = 0 kite ol. solar ki spiser du hver solar, solar, so	t fetlinnhold? *
yogdua, samme katheaths 2 Remme, creme wor mange ganp     Dete elementa     yogius, seme kedework      Assidenter      elementation     elementation      Assidenter      or mye ramme,     dete elementation      Assidenter      or mye ramme,     dete elementation      as      or Ris og pa      ingår ris eller p      Ja      Net      Dete element	erene halde eller lie eme fraiche og fraiche, eller is er pr. uke spiser du vise kun denom alte overe fraiche eller lie O 1 O 1 O 5 coerne fraiche out, vises kun denom alte i seed hagt televeraud	Ilionende med nineholder mer a tarame, oreme ha retivet «Ja» er vagt i n retivet «Ja» er vagt i	hoyt fettinnh enn 20% fetti kite ol. med hay parmäke «tropie tuketige 0 0 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1	t lettimhold? * gang? *
yogdua, samme katheaths 2 Remme, creme samme, creme wor mange ganp      Deto Herrestat     yogdua, samme ketheaths      AustBecken      AustBecken      AustBecken      AustBecken      AustBecken      Cette Herrestat      cense tractes a      cense tractes	erene halde eller lie eme fraiche og fraiche, eller is er pr. uke spiser du vise kun denom alle overe fraiche eller lie D 1 D 5 Cherre fraiche ell. vises kun denom alle i seat hald heller elle stata esta som en del net vises kun denom	Ilionende med nneholder mer storame, oreme ha nativet site er sagt i a serde son en de av d	hoyt fettinnh enn 20% fetti kite ol. med hay parmäke «tropie tuketige 0 0 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1	t lettimhold? * gang? *
yogdua, samme katheaths 2 Remme, creme samme, creme wor mange ganp      Deto Herrestat     yogdua, samme ketheaths      AustBecken      AustBecken      AustBecken      AustBecken      AustBecken      Cette Herrestat      cense tractes a      cense tractes	errere haldhe eller lik eme fraiche og fraiche, eller is er pr. uke opioer du view kun denom alte derre fraiche eller is D 1 D 1 D 5 coerre fraiche eller is coerre fraiche elle use haut bestroaut sta esta som en del	Ilionende med nneholder mer storame, oreme ha nativet site er sagt i a serde son en de av d	hoyt fettinnh enn 20% fetti kite ol. med hay parmäke «tropie tuketige 0 0 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1	t lettimhold? * gang? *
yoduc, samme katheaths     2 Remine, creme     smme, creme     vor mange ganp     Debe elements     vor mye rememe     vor mye remove     elements     vor mye remove     elements     come tracte o      Ris og pa mgår ris eller p     Ja     Nes     Debe elements     o.1 Brun ris	erene halde eller lie eme fraiche og fraiche, eller is er pr. uke spiser du vise kun denom alle overe fraiche eller lie D 1 D 5 Cherre fraiche ell. vises kun denom alle i seat hald heller elle stata esta som en del net vises kun denom	Ilionende med nneholder mer stamme, oreme ha nature son en del av di o a nature son en del av di o a o ao nature son en del av di o a o ao ned heyt tettinsho naturet sis, sis, etc. av citt ukentlig ko naturet vet sos er nertig kostholt?» korn)	hoyt fettinnh enn 20% fetti kite ol. med hay parmäke «tropie tuketige 0 0 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1	t lettimhold? * gang? *
yoghac, samme katheaths     2 Remme, creme     somme, creme     yoghac, samme, creme	erene haldhe eller lik eme fraiche og fraiche, eller is er pr. uke spiser du vise kun denom alte overe fraiche eller is overe fraiche eller i de fraiche ol. vise hun denom alte overe fraiche eller i sed hugt helerend sta esta som en del ent viseu kun denom som er de av dit u (upolert, full)	Ilionende med nineholder mer stamme, oreme ha matuet sola er saga i andere son en det av di andere son en det av di alionen en det av di alionen er saga i av dit ukentig ko natuet sola, sola, et s arge ganger pr. die sp re- rege ganger pr. die sp re- sola ganger p	In usentige howy for the formation where cill med have parameter at most in design of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of t	t lettimhold? * gang? *
yoduct, sames kathestin     2 Remine, creme     smme, creme     vor mange ganp     vor mange ganp     vor mye remeater     vor mye remeater     vor mye remover     eveneme     eveneme     creme reader     vor mye remover     eveneme     creme reader     vor mye remover     of Ris og pa mgår ris eller p     of Ja     Ses     Dete elemente     of Nes     Dete elemente     vor mange gan     Dete elemente	erene haldhe eller lik eme fraiche og fraiche, eller is er pr. uke spiser du vise kun denom alle overe haldhe eler lik 0 1 0 5 cherne fraiche ol. vises kun denom alle 1 med hauf helerend 1 med hauf helerend stat asta som en del net vises kun darsom som en del av dit u (upolert, full) nger pr. uke spise	Ilionende med nineholder mer stamme, oreme ha matuet sola er saga i andere son en det av di andere son en det av di alionen en det av di alionen er saga i av dit ukentig ko natuet sola, sola, et s arge ganger pr. die sp re- rege ganger pr. die sp re- sola ganger p	In usentige howy for the formation where cill med have parameter at most in design of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of the formation of t	t feffinnhold? * gang? *
yedua, arrene kenteatris 2 Remme, creme samme, creme vor mange ganp      Dete enrectat     yoytur, enree kenteatris      Austitectes	erene haldhe eller lik eme fraiche og fraiche, eller is er pr. uke spiser du vise kun denom alle overe haldhe eler lik 0 1 0 5 cherne fraiche ol. vises kun denom alle 1 med hauf helerend 1 med hauf helerend stat asta som en del net vises kun darsom som en del av dit u (upolert, full) nger pr. uke spise	Ilionende med nineholder mer stamme, oreme ha nativet site er satgt i a erde son er de av d o	In unentige	t feffinnhold? * gang? *

 Detre elementet vises iun demain alternativet <3x, <2x, <1x, <2 to, <dx-7x, <dxalter <1x er vagt i spetemålet «Hvor wange ganger pr. uke spisor du brun Hv?» Rosen i bildene nedenfor er servert på en middagstallerken (19 cm). Oppgi mengde som kokt ns.



## 10.2 Hvit ris (polert)

Hvor mange ganger pr. uke spiser du hvit ris \*



Hvor mye hvit ris spiser du hver gang? \*

Dette elementet vises kun dersom alternativet «3», «2», «1», «≥ 8», «6»7», «5» eller «4» er valgt i spørsmålet «Hvor mange ganger pr. uke spiser du hvit ris»

Risen i bildene nedenfor er servert på en middagstallerken (19 cm). Oppgi mengde som kokt ris.



O A= ca. 40 gram





() C ≈ ca. 160 gram

O D = ca. 320 gram

 Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «inngår ris eller pasta som en del av ditt ukentlig kosthold?»

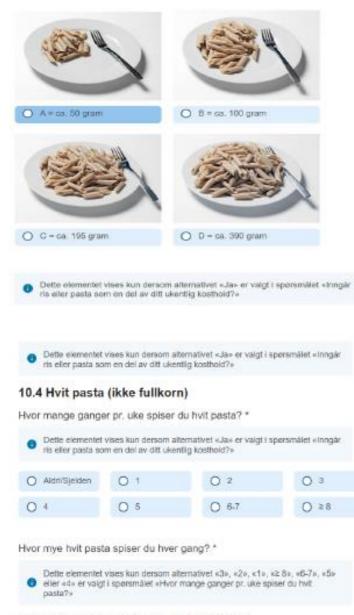




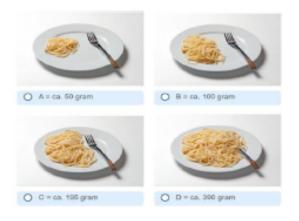
Hvor mye fullkomspasta spiser du hver gang? \*

Dette elementet vises kun dersom alternativet viz 8+, v6-7+, v5+, v4+, v3+, v2+ eller x1+ er valigt i sporsmålet «Phor mange ganger pr. uke spiser du fullkomspasta?»

Pastaen i blidene nederfor er servert på en middagstatierken (19 cm). Oppgi mengde som kokt pasta.



Pastaen i bildene nedentor er servert på en middagstallerken (10 cm). Oppgi mengde som kokt pasta.



Obligatoriska faiter er market med stjerne \*

 Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «inngär fis eller pasta som en del av dit ukentlig kosthold?»

#### 11. Kalde og varme drikker

VI vil først spørre deg om kalde drikker, og deretter varme drikker.

#### 11.1 Kalde drikker i glass

Hvor ofte drikker du vanligvis de ulike typene drikker i listen under?



Bildet over viser et vanlig kjøkkenglass tilsvarende 2 dl.

"Glass/uke" betyr antali glass i uken, "glass/dag" betyr antali glass pr. dag. "=7 glass/dag" betyr 7 eller fiere glass pr. dag.

Husk å ta med melk du bruker på frokosigryn, grøt og dessert.

	Aldricijekten	5-3 giana laka	4-6 glassi luka	1-2 glassidag	3-4 gianaidag	5-6 giaso/dag	i: T glass./dag
Vann (springvann) *	0	0	0	0	0	0	0
Flasicevann med og uten kullsyre (t.aka. Paris, Insdar) *	0	0	0	0	0	0	0
Heimelic, kelic, kulturmelic*	0	0	0	0	0	0	0
Lettmelk (1% eller 0,5%), skummet melk, skummet katumetk *	0	0	0	0	0	0	0
Juice (f.eks. epiejuice, appelisinjuice uten tisalt sukker) *	0	0	0	0	0	0	0
Saft og isle med tilsatt sokker *	0	0	0	0	0	0	0
Saft og løle uten tilsatt sukker, kans tig satet *	0	0	0	0	0	0	0
Annen drikke uten bibait sukker (Ceka istitione) *	0	0	0	0	0	0	0
Amen drikke med libeit suiker (f.eks. brus, neida; energidrikke) *	0	0	0	0	0	0	0

## 11.2 Kaffe og te

Inngår kaffe eller te som en del av ditt ukentlige kosthold? \*



Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår kafte eller te som en del av ditt ukentlige kosthold?»

#### Hvor ofte drikker du vanligvis de ulike typene drikker i listen under?

Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår kaffe eller te som en del av ditt ukentlige kosthold?»



Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår kafte eller te som en del av ditt ukentlige kosthold?»

1 kopp te tilsvarer ca. 2,5 dl

1 kopp vanlig kaffe tilsvarer ca. 2,0 dl

1 kopp espresso filsvarer ca. 0,3 dl

 Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår kaffe eller te som en del av ditt ukentlige kosthold?»

	Altal/Sjelden	1-3 koppertuke	4-5 kopper/uke	1-2 koppet/dag	0-4 kopperidag	5-6 koppenidag	i⊨7 koppes/dag
Kaffe (irantet, filler) *	0	0	0	0	0	0	0
Presskanne kaffe, kokekaffe, kaffokapser "	0	0	0	0	0	0	0
Espresso *	0	0	0	0	0	0	0
Annen kaffe (Faka, cappucho, caffe late, macchiato, andre espresso relatert) *	0	0	0	0	0	0	0
Te (f.eks. svart. grann) *	0	0	0	0	0	0	0

#### 12. Alkoholholdige drikker

Drikker du vanligvis alkoholholdige drikker? \*



 Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Drikker du vanligvis alkoholholdige drikker?»

#### Hvor mange ganger pr. uke drikker du alkoholholdige drikker?



"gangluke" betyr antall ganger i uken.

 Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Drikker du vanligvis alkoholholdige drikker?»

		gangluk	gangerlu ka	gangechi	ganges/u	gangen/a	gangeriu	≥8 gangiak e
68, steek al, pile *	0	0	0	0	0	0	0	0
100.1	0	0	0	0	0	0	0	0
Essenaryis *	0	0	0	0	0	0	0	0

#### Hvor mye drikker du hver gang?

 Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Drikker du vanligvis alkoholhoidige drikker?»



 Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Drikker du vanligvis alkoholholdige drikker?»

Bildet over viser vanlig vinglass tilsvarende 1,2 dl, ølglass tilsvarende 4,0 dl og brennevinsglass tilsvarande 0,4 dl.

Oppgi mengde i antall glass.

 Dette elementet vises kun dersom alternativet «Ja» er valgt i spøramälet «Drikker du vanligvis alkoholholdige drikker?»

	0	1/2 glass	1 glass	2 glass	3 giass	4 glass	5 glass	≿ 6 glass
ØI, sterk øl, pils (glæss, 4 dl) *	0	0	0	0	0	0	0	0
Vin (glass, 1,2 dl) *	0	0	0	0	0	0	0	0
Drennevin (glass, 0,4 dl)	0	0	0	0	0	0	0	0

Obligatoriske felter er merket med stjørne \*

#### 13. Kaker, dessert, godteri

Inngår kaker, dessert og godteri i ditt ukentlige kosthold? \*

O Nei	0	Ja
	0	Nei

 Dette elementet vises kun dersom alternativet «Ja» er valgt i spersmälet «Imgår kaker, dessert og gottert i dit ukentlige kosthold?»

#### Hvor ofte spiser du vanligvis de ulike matvarene i listen under?

Dette elementet vises kun denom alternativet «Jas» er valgt i spensmälet «Inngår kaker, dessen og gotten i ditt ukentlige kostholoft»

	Autor/System	1 emailable	3-5 enhetanska	45 enhetenuke	6-7 enheteriuke	1-3 onthefos/sike	ic 50 emiliar taka
Hater, Evenielanist, valter, sait lipina (1-entret=cai dd galaitt, 1 kaliumlykke=1 bolle=1 saiflebate= 8 sinå-lipina)*	0	0	0	0	0	0	0
Desset (f.eks. is, hermetisk tuid, pudding) (1 enhet= 1,5 dl) *	0	0	0	0	0	0	0
Spholade, godieri (1 posjon- tecişiani) *	0	0	0	0	0	0	0
Potetuli, chips (1 entetri 1 neveri 15 gram) *	0	0	0	0	0	0	0

## 14. Kosttilskudd

Inngår kosttilskudd i ditt ukentlige kosthold? \*

0	Ja
0	Nel

 Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår keattliskudd i ditt ukentlige keathold?»

#### Hvor ofte spiser du kosttilskuddene i listen under?

 Dette elementet vises kun dersom alternativet «Ja» er valgt i spørsmålet «Inngår kosttilskudd i ditt ukentlige kosthold?»

	Aldri/Sjeiden	1-2 enheter/uke	3-4 enheter/uke	1 enhetidag	2 enheteritlag	3 enhetenitlag	≥ 4 enheteridag
Tran (1 enhel= 1 barneeskje) *	0	0	0	0	0	0	0
Trankapsler, fiskeoljekapsler, omega-3 tiskudd (1 ennet=1 kapsel) *	0	0	0	0	0	0	0
Vitamin D (1 enhet= 1 pille) *	0	0	0	0	0	0	0
Multivitamin tilskund (1 enhet= 1 pille) *	0	0	0	0	0	0	0
Jem (1 enhet= 1 pille) *	0	0	0	0	0	0	0
Kalsium (1 enhet= 1 pille) *	0	0	0	0	0	0	0
Andre kostiliskudd *	0	0	0	0	0	0	0

Obligatoriske felter er merket med stjerne \*

#### 15. Fysisk aktivitet, tid i ro og søvn

Vi vil først spørre deg om din fysiske aktivitet der du blir lett og veldig andpusten, og deretter hvor lenge du er i ro og sover i løpet av ett vanlig døgn.

15.1 Hvor mange ganger pr. uke er du fysisk aktiv der du blir lett andpusten (moderat intensitet)? \*

F.eks. hurtig gange, hardt husarbeid, fysisk aktiv i arbeid

O Aldri/Sjeiden	O 1	O 2	O 3
0 4	O 5	O 6-7	O 28

15.1.1 Hvor lenge var du fysisk aktiv hver gang (minutter) i moderat intensitet? \*

<ul> <li>Dette elementet vises kun dersom alternativet «de, «z.e., «1», «z.e», «er», «t»</li> <li>eller «fix er valgt i spersmålet «15.1 Hvor mange ganger pr. uke er du fysisk aktiv der du blir lett andpusten (moderat intensitet)?&gt;</li> </ul>								
O 1-4 minutter	O 5-9 minuter	O 10-15 minuter	O 16-20 minuter					
O 21-30 minutler	O 31-45 minutler	O 46-80 minutter	O ≥ 61 minutter					
0 1.00	0	0	0					
15.2 Hvor mange ganger pr. uke er du fysisk aktiv der du blir veldig andpusten (høy intensitet)? *								
F.eks. jogging, skigåing, hard fysisk aktivitet, driver idrett								

<ul> <li>Aldri/Sjelden</li> </ul>	O 1	O 2	O 3
O 4	0 6	0 6-7	() ≥8

15.2.2 Hvor lenge var du fysisk aktiv hver gang (minutter) i høy intensitet? \*

Dette elementet vises kun dersom alternativet «3», «1», «2 8», «6-7», «5» eller «1» er valgt i spørsmålet «15.2 Hvor mange ganger pr. uke er du fysisk aktiv der du blir veidig andpusten (hey intensitet)?»			
O 1-4 minutter	O 5-8 minutter	O 10-15 minuter	O 16-20 minutter
O 21-30 minutter	O 31-45 minutler	O 46-60 minutter	○ ≥ 61 minutter

# Vi vil nå spørre deg om hvor lenge du vanligvis er i ro og sover i løpet av ett døgn (24 timer).

Summen av de tre neste spørsmålene kan ikke bli mer enn 24 timer som tilsvarer ett døgn.

15.3 Hvor mange timer sitter du i ro i løpet av en vanlig arbeidsdag? \*

Med ro menes stillesittende aktivitet, f.eks. transport til og fra arbeid (bil, tog, buss, trikk etc.), å lese dokumenter, tid brukt til mållider, sitter i møter, sitter foran PC, sitter med en mobiltelefon eller en annen skjerm.

Hvis du ikke er i arbeid kan du svare 0 (null) her og gå til neste spørsmål.

15.4 Hvor mange	timer sitter di	u i ro i løpet a	v din fritid eller	i løpet av en van	ilia daa? '

Med ro menes stillesittende aktivitet, f.eks. å lese bok, tid brukt til måltider, sitter eller ligger og ser på TV, sitter med en PC, mobiltelefon eller annen skjerm.

15.5 Hvor mange timer sover du vanligvis pr. døgn? \*

#### 16. Røykevaner

Hva passer best for å beskrive dine røykevaner nå?\*

- Røyker daglig
   Røyker av og til
- Har sluttet helt å røyke
- Har aldri røykt verken daglig eller av og til

Hvor mange år er det siden du sluttet å røyke siste gang? \*

 Dette elementet vises kun dersom alternativet «Har sluttet helt å røyke» er valgt i spersmålet «Hva passer best for å beskrive dine røykevaner nå?»

Hvor mange år har du røykt sammenhengende? Trekk fra de periodene du ikke har røykt, hvis du har sluttet å røyke i lengre perioder \*



#### Antall sigaretter \*

 Dette elementet vises kun dersom alternativet «Har sluttet helt å røyke» er valgt i spørsmålet «Hva passer best for å beskrive dine røykevaner nå?»

Hvor mange sigeretter pleide du å røyke pr. uke før du sluttet?

Obligatoriske felter er merket med stjerne \*

## 17. Snus

Hva passer best for å beskrive dine snusvaner: \*



Hvor mange är er det siden du sluttet ä snuse siste gang? \*

Dette elementet vises kun dersom alternativet «Har sluttet å bruke snus» er valgt i spørsmålet «Hva passer best for å beskrive dine snusvaner:»

Hvor mange år har du snuset sammenhengende, trekk fra de periodene du ikke har snust, hvis du har sluttet å snuse i lengre perioder. \*

0	Dette elementet vises kun dersom alternativet «Bruk snus daglig» eller «Har sluttet å bruke snus» er valg best for å beskrive dine snusvaner.»	
0	1-5 ár	
0	6-10 ár	
0	0-10 al	
0	11-15 år	
0	11-10 @	
0	16-20 ár	
~	10-20 81	
0	21-25 år	
$\sim$	6.7% of 10	
0	Mer enn 25 år	
0		

Hvor mange bokser med snus bruker du pr. dag? \*

 Dette elementet vises kun dersom alternativet «Bruker snus daglig» er valgt i sporsmålet «Hva passer best for å beskrive dine snusvaner.»

Hvor mange bokser med snus brukte du pr. uke før du sluttet? \*

 Dette elementet vises kun dersom alternativet «Har sluttet å bruke snus» er valgt i spørsmålet «Hva passer best for å beskrive dine snusvaner:»

## 18. Generelle opplysninger

Alder \*

Vennligst oppgi alder i år.

Kjønn \*

0	Mann
0	Kvinne

Vekt \*

Vennligst oppgi vekt i kg.

Høyde \*

Vennligst oppgi høyde i cm.

Bosituasjon \*

- O Bor sammen med en eller flere
- Bor alene

Bosituasjon \*

- Bor sammen med en eller flere
- O Bor alene

Hvilken utdanning er den høyeste du har fullført? \*

- O Grunnskole 7-10 år, framhaldsskole, folkehøgskole
- Realskole, middelskole, yrkesskole, 1-2 årig videregåen de
- Artium, økonomisk gymnas, allmennfaglig retning
- Høgskole/Universitet, mindre enn 4 år
- O Høgskole/Universitet, 4 år eller mer
- Fagbrev

#### Arbeidsførhet \*

O Student

I arbeid (helt eller delvis)
 Hjemmeværende (sehvalgt)
 Pensjonist
 Arbeidsledig
 Sykmeldt
 Under attføring/rehabilitering
 Midlertidig uferetrygdet
 Varig uføretrygdet

Flere svar er mulig, hvis annet spesifiser i neste spørsmål

0	Europa
0	Afrika
0	Asia
0	Annet

Hvis annen etnisk bakgrunn, spesifiser her:

<ul> <li>Dette elementet vises kun dersom alternativet «Annet» er valgt i spørsmål «Hva slags øtnisk bakgrunn har din far?»</li> </ul>	et
Hva slags etnisk bakgrunn har din mor? *	
Flere svar er mulig, hvis annet spesifiser i neste spersmål	
O Europa	

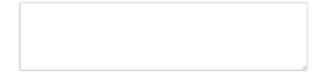
0	Afrika
0	Asia
0	Annet

Hvis annen etnisk bakgrunn, spesifiser her:

•	Dette elementet vises kun dersom alternativet «Annet» er valgt i «Hva slags etnisk bakgrunn har din mor?»	spørsmålet
•	«Hva slags etnisk bakgrunn har din mor?»	

Hvor lenge (år) har du bodd i Norge? \*

Her har du mulighet til å skrive ned andre matvarer du spiser som vi ikke har spurt deg om. Dette er valgfritt.



Tusen takk for at du tok deg tid til å svare på dette spørreskjemaet!

# Appendix 6: Regional Ethic Committee for Medical Research's approval of the study



Claus Henning Thorsen

Telefon: 22845515

Vár dato: 06.05.2021 Vår referanse: 251226

Dawn Elizabeth Peleikis

REK annual C

Prosjektsøknad: Kostholdet til pasienter med en alvorlig psykisk lidelse(schizofreni eller bipolar affektiv lidelse) i en skandinavisk psykiatrisk poliklinikk Søknadsnummer: 251225 Forskningsansvarlig institusjon: Vestre Viken HF

# Prosjektsøknad godkjennes av REK

#### Søkers beskrivelse

Personer med en alvorlig psykisk lidelse som schizofreni og bipolar affektiv lidelse har redusert gjennomsnittlig forventet levetid, særlig grunnet økt forekomst av hjerte- og karsykdommer (HKS). I prosjektet skal kostholdet til denne gruppen kartlegges for å avdekke eventuelle kostholdsfaktorer som bidrar til økt risiko for HKS. Både 241 recall intervjuer og digitalt spørreskjema (DIGIKOST) skal benyttes som metoder. Informasjon fra pasientjournal skal også innhentes. Herunder vil BMI, vekthistorie, blodtrykk samt biokjemiske mål (fastende blodglukose, HbA1c, fastende triglyserider, Hb, Ferritin, ASALT/ALAT og total kolesterol, LDL- og HDL-kolesterol) være relevante for å vurdere ernæringsstatus samt risiko for hjerte og karsykdommer. Grunnet bivirkninger som fører til økt apetitt og følgelig endret inntak og vektoppgang, er også eventuelle medikamenter relevante. Gjennom prosjektet vil vi kunne belyse behovet for ernæringsintervensjon- og behandling i denne pasientgruppen, og slik optimalisere behandlingstilbudet for å redusere forekomst av hjerte- og karsykdom hos personer med diagnosene schizofreni og bipolar affektiv lidelse.

Vi viser til søknad om forhåndsgodkjenning mottatt 17.03.2021.

Søknaden ble behandlet av Regional komité for medisinsk og helsefaglig forskningsetikk (REK sør-øst C) i møtet 15.04.2021. Vurderingen er gjort med hjemmel i helseforskningsloven (hfl.) § 10.

#### **REKs vurdering**

Personer med alvorlig psykisk lidelse, så som schizofreni eller bipolar affektiv lidelse, har forkortet levetid grunnet kardiovaskulære incidenter. I dette prosjektet, som inngår i en master i klinisk ernæringsfysiologi, vil man kartlegge eventuelle kostholdsfaktorer som kan være bakomliggende årsaker til kardiovaskulær sykdom. Man tar sikte på å inkludere 30 pasienter ved Asker DPS med diagnosene schizofreni og bipolar affektiv lidelse. Potensielle deltakere identifiseres ved diagnosekoder i DIPS, og pasientens behandler vurderer samtykkekompetanse og om de er i en stabil fase.

Samtykke til deltakelse i studien innhentes av samme prosjektmedarbeider som tidligere har informert om studien, alternativt pasientens ansvarlige behandler.

Studiedeltakelse innebærer besvarelse av spørreskjemaet DIGIKOST, som er basert på et validert papirskjema, og planlegges validert i løpet av 2021. Gjennom spørreskjemaet registreres kostinntak, røykevaner og fysisk aktivitet basert på de norske helsemyndigheters anbefalinger om kosthold og andre livsstilsfaktorer.

Det gjennomføres også intervju (24t recall intervjuer for kartlegging av matinntak de siste 24 timene). Dagen det registreres inntak fra, vil være tilfeldig og en vil derfor ikke fange opp variasjon.

Det skal også innhentes diverse variabler fra pasientjournal (DIPS),

Komiteen mener deltakelse i prosjektet kan ha en nytteverdi for pasientene, og da det i tillegg fremstår som lite invasivt, har komiteen ingen forskningsetiske innvendinger til gjennomføringen.

Komiteen anbefaler at samtykke til studiedeltakelse innhentes av prosjektmedarbeider, og ikke av pasientens ansvarlige behandler.

Komiteen har ingen merknader til innholdet i informasjonsskrivet, men forutsetter at det fremgår hvem som har informert om studien.

## Vedtak

Komiteen har gjort en helhetlig forskningsetisk vurdering av alle prosjektets sider. Prosjektet godkjennes med hjemmel i helseforskningsloven § 10.

Komiteen gjør samtidig oppmerksom på at etter ny personopplysningslov må det også foreligge et behandlingsgrunnlag etter personvernforordningen. Det må forankres i egen institusjon.

Tillatelsen er gitt under forutsetning av at prosjektet gjennomføres slik det er beskrevet i søknaden og protokollen, og de bestemmelser som følger av helseforskningsloven med forskrifter.

Tillatelsen gjelder til 15.05.2026. Av dokumentasjons- og oppfølgingshensyn skal opplysningene likevel bevares inntil 15.05.2031. Opplysningene skal lagres avidentifisert, dvs. atskilt i en nøkkel- og en opplysningsfil. Opplysningene skal deretter slettes eller anonymiseres, senest innen et halvt år fra denne dato.

Komiteens avgjørelse var enstemmig.

## Sluttmelding

Prosjektleder skal sende sluttmelding til REK på eget skjema via REK-portalen senest 15.05.2026 + 6 måneder, jf. helseforskningsloven § 12. Dersom prosjektet ikke starter opp eller gjennomføres meldes dette også via skjemaet for sluttmelding.

## Søknad om endring

Dersom man ønsker å foreta vesentlige endringer i formål, metode, tidsløp eller organisering må prosjektleder sende søknad om endring via portalen på eget skjema til REK, jf. helseforskningsloven § 11.

# Klageadgang

Du kan klage på REKs vedtak, jf. forvaltningsloven § 28 flg. Klagen sendes på eget skjema via REK portalen. Klagefristen er tre uker fra du mottar av dette brevet. Dersom REK opprettholder vedtaket, sender REK klagen videre til Den nasjonale forskningsetiske komité for medisin og helsefag (NEM) for endelig vurdering, jf. forskningsetikkloven § 10 og helseforskningsloven § 10»

Med vennlig hilsen

Britt Ingjerd Nesheim Prof. dr. med. Leder REK sør-øst C

Claus H. Thorsen Seniorrådgiver

Dokumentet er elektronisk signert

Kopi til:

Vestre Viken HF

Emma Johannessen