
# Modifiable determinants of newborn macrosomia and birth complications

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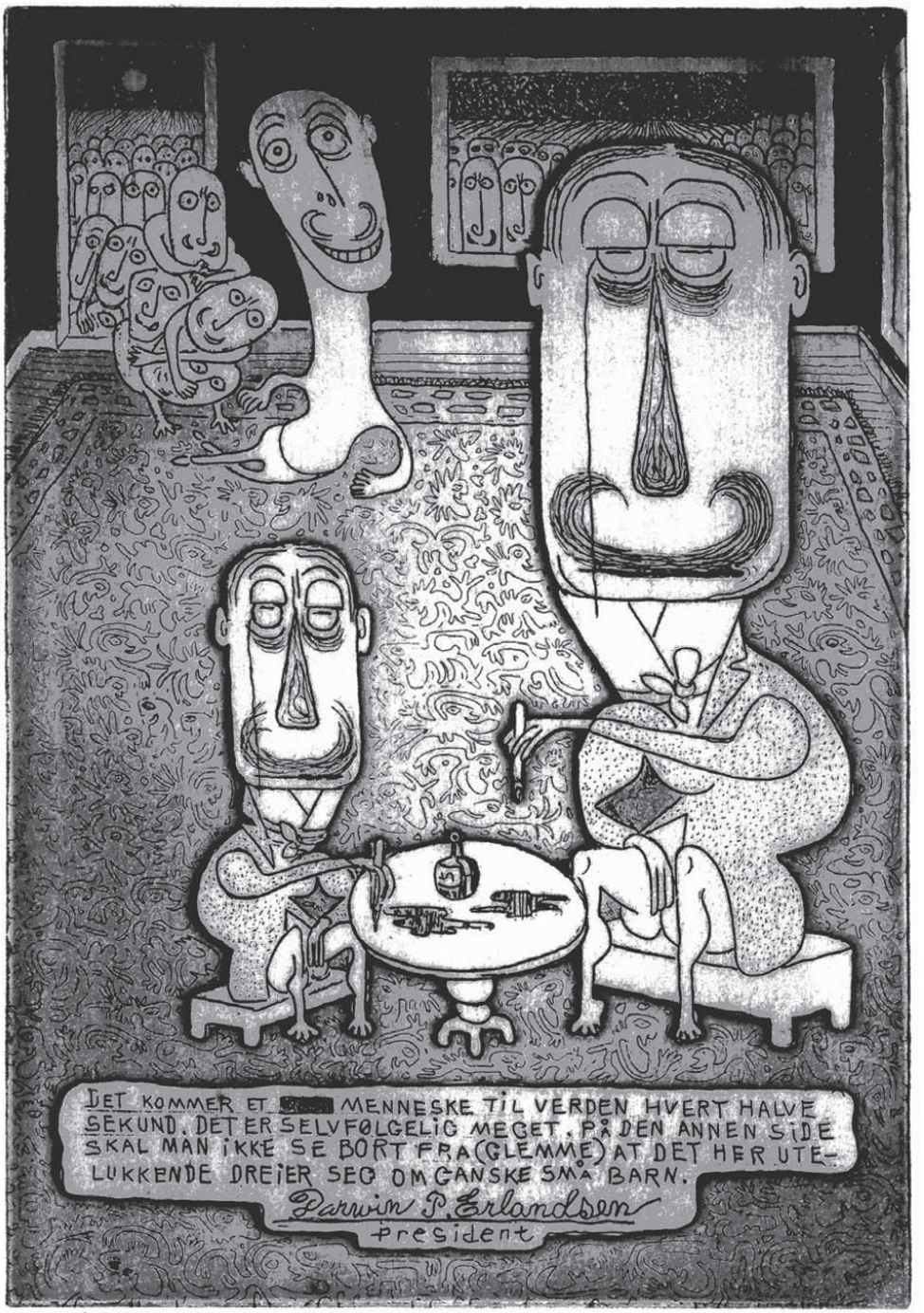

DET KOMMER ET [redacted] MENNESKE TIL VERDEN HVERT HALVE SEKUND. DET ER SELVFØLGELIG MEGET. PÅ DEN ANNEN SIDE SKAL MAN IKKE SE BORT FRA (GLEMME) AT DET HER UTE-LUKKENDE DREIER SEG OM GANSKE SMÅ BARN.
Darwin P. Erlandsen
president

40/50 Stabel

# Modifiable determinants of newborn macrosomia and birth complications

# 1 Preface

## *1.1 Acknowledgements*

The STORK-study was initiated by Professor Tore Henriksen at the Department of Obstetrics and Gynecology at Rikshospitalet, and Professor Jens Bollerslev at the Department of Medicine, Endocrinologic Section, both Oslo University Hospital, Rikshospitalet. The study has been carried out in collaboration with the Norwegian School of Sports Medicine and the Department of Nutrition, University of Oslo. To accomplish this clinical study, many people have been involved. I am very grateful for their enthusiasm and support.

Especially I wish to thank my main supervisor, Tore Henriksen, for his patience during all these years, and his ability to be present when needed. I also want to thank him for all the interesting discussions and valuable input on the thesis and related themes. I gratefully acknowledge the time he has spent guiding me into the fascinating field of developmental origins of health and disease (DOHaD).

I am also grateful to my co-supervisors Professor Kari Bø at the Norwegian School of Sports Medicine and Professor Jens Bollerslev for the support and all the positive comments throughout this work.

I am strongly indebted to Kathrine Frey Frøslie and the good teamwork that has developed. Without her good spirit and statistical exactness, the accomplishment of this work would have been more strenuous. I am impressed by her patience and enthusiasm. Elisabeth Qvigstad from the Section of Endocrinology came into the project after some years and her knowledge and contributions have been essential.

My co-authors Lene Haakstad from the Norwegian School of Sports Medicine and Kristin Godang from the Endocrinologic Section have contributed with important clinical skills from their fields. The nurses and the laboratory staff at the Section of Endocrinology have contributed with tremendous effort, completing the two-hour glucose tolerance test twice in all the participating women. The midwives and the nursemaids at the delivery and maternity units have been very friendly and helpful to collect important data. I am very indebted to all of you. Warm thanks also to Rakhee Sharma and Esther Baumann, who have kept track of the appointments for the women, filed the journals in neat order and helped out whenever needed.

I am also very grateful for the positive support from Thomas Åbyholm, the Head of Department of Obstetrics and Gynecology, who has showed great interest and been supportive of this project.

I am indebted to all the participants of childbearing women who have carefully come to all scheduled times, without you this study would never have come to an end.

While the project was ongoing, it was decided to continue the study to enlarge the cohort. STORK II was then established, and Camilla Friis and Marie Cecile Paasche Roland came into the study with good spirits and great enthusiasm. Thank you for all the good times. A special thanks to Pernille Frese for her friendly contribution these last weeks.

The study has been accomplished with grants from the Norwegian Health Association, Centre for Women's Health, Department of Obstetrics and Gynecology, University of Oslo, Rikshospitalet and from the Faculty of Medicine, Thematic Research Area, University of Oslo.

Last, but not least, I am very grateful to my large and noisy family, who has stayed with me all this time and never doubted my ability to accomplish this work. Thank you, Anders, Wanda, Helene, Nora, Paula and Bror.

### *1.2 Summary*

Globally the incidence of macrosomic newborns is increasing. In Norway the percentage of newborns weighing 4,000g or more has increased from 16 to 20% in less than three decades. Newborn macrosomia is associated with short-and long-term health risks for the infant, and increases the prevalence of birth complications. Parity, maternal age and gender of the child influence fetal growth. Maternal overweight is a risk factor for gestational diabetes (GDM) and newborn macrosomia. Some women with high body mass index (BMI) give birth to macrosomic newborns while others do not. Maternal lifestyle factors are potential predictors of pregnancy complications. Boys are heavier at birth than girls, but girls have higher fat mass at birth than boys.

During the period of 2002 and 2005 the STORK-study followed a total of 553 pregnant women through pregnancy and childbirth at the Department of Obstetrics and Gynecology, Rikshospitalet.

The aims of this thesis were to evaluate the contribution of modifiable factors that may influence the risk of fetal macrosomia. The first aim was to explore the roles of physical inactivity, BMI and fasting plasma glucose. A second aim was to study the impact of physical inactivity on delivery complications. The third aim was to examine whether maternal fasting plasma glucose levels influence birth weight in the two sexes differently.

Pregestational physical inactivity, glucose and high BMI were independent determinants of fetal macrosomia. Pregestational physical inactivity increased the risk of perineal laceration degree three to four. Overweight women with an increase in fasting plasma glucose from early to late pregnancy had a 4.5-fold increase in risk of newborn macrosomia compared to the remaining group with a high BMI. The effect of maternal fasting plasma glucose on birth weight in girls was twice as high as in boys. Paternal birth weight was significantly associated with birthweight of boys, but no such association was seen for girls

### *1.3 Abbreviations*

| | |
|---|---|
| NCD | Non-communicable diseases |
| PE | Preeclampsia |
| GDM | Gestational diabetes |
| NGT | Normal glucose test |
| IGT | Impaired glucose tolerance |
| BMI | Body mass index (weight (kg)/height ($cm^2$) |
| HDL | High density lipoprotein |
| FPG | Fasting plasma glucose |
| CVD | Cardiovascular disease |
| LGA | Large for gestational age |
| SGA | Small for gestational age |
| AGA | Average for gestational age |
| EDA | Epidural analgesia |
| DXA | Dual energy X-ray absorptiometri |
| FM | Total fat mass |
| FFM | Fat-free mass |
| CS | Cesarean Section |
| MET | metabolic equivalent task (1 MET=3.5 mL O2 uptake /kg body weight x 1 min or 1 kcal /kg body weight x h) |
| PAPQ | Physical Activity and Pregnancy Questionnaire |
| HOMA-IR | Insulin resistance ((FPG * FPI) / 22.5) |
| OGTT | Oral glucose tolerance test |
| T2DM | Type 2 diabetes mellitus |
| ACOG | The American College of Obstetricians and Gynecologists |

| | |
|---|---|
| IOM | Institute of Medicine |
| EGIR | European Group for the Study of Insulin Resistance |
| IDF | International Diabetes Federation |
| DOHaD | Developmental Origins of Health and Disease |
| FOAD | Fetal Origins of Adult Disease |

### *1.4 List of papers*

1. Voldner N, Froslie KF, Bo K, Haakstad L, Hoff C, Godang K, et al. Modifiable determinantsof fetal macrosomia: role of lifestyle-related factors. Acta Obstet Gynecol Scand 2008;87(4):423-9.

2. Voldner N, Froslie KF, Haakstad LA, Bo K, Henriksen T. Birth complications, overweight,and physical inactivity. Acta Obstet Gynecol Scand 2009;88(5):550-5.

3. Voldner N, Qvigstad E, Froslie KF, Godang K, Henriksen T, Bollerslev J. Increased risk ofmacrosomia among overweight women with high gestational rise in fasting glucose. J Matern Fetal Neonatal Med 2009 Sep 9:1-8.

4. Nanna Voldner, Kathrine Frey Frøslie, Kristin Godang, Jens Bollerslev, Tore Henriksen. Determinants of birth weight in boys and girls HUM ONTOGENET 3(1), 2009, 7–12.

## 2 Introduction

The prevalence of macrosomic newborns frequently defined as birth weight above 4000g or 4500g has increased both in Norway and world-wide the last 20 – 30 years. The Norwegian Medical Birth Registry has since 1967 registered all births in Norway and the prevalence of newborns with birth weight above 4000g has increased from about 16% in 1967 to 19% in 2008. Children weighing 4500g or more have increased from 3% to 4.5% in the same time interval (1). The prevalence of macrosomia is increasing also in other Scandinavian countries (2;3). By identifying some of the modifiable determinants it may be possible to reduce the numbers of infants born with high birth weight.

The increasing prevalence of overweight and obesity is becoming a world wide problem, in both developed and developing countries. The problem appears to increase most in the younger age groups (4). Between mid 1980 and mid 1990 the prevalence of obesity in Norway increased from 11 – 21% among women (5). The increase was highest in the youngest age groups. A consistency between low level of physical activity, overweight and high mean diastolic blood pressure has been shown (6).

Obesity may be seen as one of the key risk factors for non-communicable diseases (NCD), which accounts for about 60% of all deaths worldwide (7). In the USA smoking still remains the leading cause of mortality. However, poor diet and physical inactivity may soon overtake tobacco as the leading cause of death (8). In the UK, obesity is now the most common clinical risk factor encountered in obstetric practice (9). In a report from Confidential Enquiries into Maternal and Child Health obesity was identified as a risk factor for maternal death, finding that 35% of all mothers who died were obese, which represents a disproportionate number of deaths associated with obesity in childbearing women (10). Chronic diseases associated with obesity are cardiovascular diseases and hypertension, cancer, diabetes mellitus and gallbladder disease. What has recently been shown is that adipocytes are more than just fat

depots. They function as endocrine cells, producing hormones and as target cells for many hormones. Insulin resistance is often associated with obesity, and is especially pronounced with intra-abdominal fat accumulation (11).

# 3 Obesity

*3.1 Obesity & pregnancy*

As obesity alone represents increased disease risk, obesity in pregnancy presents added health problems (12). Several publications show significant associations between maternal obesity and complications during pregnancy and delivery (13;14). Pregnancy complications include at the most severe increased risk for intrauterine death (15), and nulliparous have increased risk for extreme premature delivery (16). It is also shown that risk of fetal growth restriction unrelated to preeclampsia was found in obese nulliparous women (12). This contrasts the finding that obesity protected against fetal growth restriction (17). Maternal obesity has been found to increase the risk for macrosomia (15), the risk for acute caesarean section, prolonged labor, and severe haemorrhage (18;19). Obese and overweight women have higher risks for developing complications such as preeclampsia (PE) and gestational diabetes (GDM) (19). It has been shown that fat-free mass in infants of the mothers with GDM was significantly less compared with the infants of the normal glucose test group (NGT) (13). Birth weight alone may not be a sensitive enough measure to recognize differences in fetal growth in certain populations. Insulin sensitivity in early pregnancy may therefore be related to maternal metabolic changes affecting fetoplacental growth and metabolism in the utero environment (13).

*3.2 Weight gain before pregnancy and weight retention after delivery*

Excessive weight gain in pregnancy and interpregnancy weight gain increase the risk for adverse outcome in the next pregnancy (20). Pregravid body mass index (BMI, weight (kg)/height $^2$ (m)) is often associated with macrosomic newborns and complications during pregnancy and delivery (16). It has been shown that weight gain between pregnancies increases the risk of adverse pregnancy outcome in the next pregnancy also in women who were not overweight, including increased risk for large for gestational age (LGA) (20). Increase in BMI from normal to obese between the first and second pregnancies showed enhanced risk for delivering a LGA infant, whereas a decrease in pregravid BMI from obese to normal in the second pregnancy attenuated the elevated risk for a LGA birth (21).

*3.3 Weight gain during pregnancy*

Both epidemiological studies and animal models have shown that there are strong associations between low birth weight and subsequent risk of developing metabolic syndrome, insulin resistance, obesity or coronary heart diseases in later life (22). In the Western societies the opposite problem may be more likely to occur, that is, newborn infants being borne too large. In the UK there has been a two-fold increase in women being recognized as obese at booking visits, and nearly one in five women booking for antenatal care in 2002/2004 at Glasgow Maternity Hospital was obese (15;23).

Recommended weight gain during pregnancy above guidelines is associated with high birth weight, macrosomia or LGA-infant (24), hypoglycemia or hyperbilirubinemia in the newborns (25). There is not yet a general agreement on the recommended weight gain for women in the different prepregnancy BMI classes (26). In 1990 the Institute of Medicine (IOM) reported in *Nutrition During Pregnancy* how much women should gain during pregnancy (27). The main focus was then on children born too small; now the focus has

shifted to those who gain too much. The recommendation was divided into BMI-groups (Table 1). Several studies confirm that a substantial proportion of normal-weight women and an even greater proportion of overweight women gain more than is recommended (28). Evaluating gestational weight gain in a normal weight group of pregnant women found that adherence to the current IOM guidelines results in lower risks for adverse pregnancy, labor and delivery outcomes when comparing all outcomes collectively. However, only 40% gained the recommended amount of weight during pregnancy, whereas 18% and 43% gained less or more than recommended (29). Gestational weight gain and pregnancy outcomes in obese women need to be divided into obesity classes; grade I (BMI 30 – 35), grade II (BMI 35 – 40), and grade III (BMI > 40). Minimal risk may correspond to a weight gain of 10 – 25 lb (4.5 - 11.3kg) for class I obese women, a weight gain of 0 – 9 lb (0 – 4.1kg) for class II obese women and a weigh loss of 0 – 9 lb (0 – 4.1kg) for class III obese women (26). The prevalence of pre eclampsia, LGA and caesarean delivery increase with increased BMI-classes, while the prevalence of SGA-children is reduced (30). Weight gain limits for BMI categories has decreased the risk for adverse obstetric and neonatal outcome has been associated with lower gestational weight gain than was earlier recommended, especially among obese women (26).

**Table 1** Recommended total weight gain ranges for pregnant women by pre-pregnancy BMI (9;26)

| **pre-pregnancy BMI** | **Recommended weight gain (kg) [Cedergren**(26)**]** | **Recommended weight gain (kg) [IOM** (27)**]** |
|---|---|---|
| **Underweight, < 19.8 [< 20]** | **4 - 10** | **12.5 – 18** |
| **Normal, 19.8 – 24.9 [20-24.9]** | **2 - 10** | **11.5 – 16** |
| **Overweight, 25 – 29.9 [25-29.9]** | **Less than 9** | **7 – 11.5** |
| **Obese, > 29.9 [≥ 30]** | **Less than 6** | **At least 6.8** |

## 4 The metabolic syndrome

The syndrome as we recognize it today was first described as “Syndrome X” by Reaven in 1988, and it was established as clinical important, although obesity was not included (31). This syndrome is now accepted as a cluster of metabolically related symptoms which predict a high risk of developing cardiovascular risk as well as diabetes (if not already present). There are several definitions to this syndrome worldwide made by the World Health Organization (WHO) (1999), the European Group for the Study of Insulin Resistance (EGIR) (1999), and National Cholesterol Education Program – Third Adult Treatment Panel (NCEP ATP III) (2001).The last attempt to make a world-wide definition has come from International Diabetes Federation (IDF) from a workshop in 2006 (32). Their definition is ethnic specific, and says that the syndrome contains of central obesity, raised triglycerides and reduced high density lipoprotein (HDL), cholesterol, raised blood pressure and raised fasting plasma glucose (FPG) (32). As pregnancy in itself is a metabolically state with features of metabolic syndrome these definitions cannot be relocated to pregnant women (33-35). An increasing number of women will have established a metabolic syndrome as they enter into pregnancy. The increased physiological demands that occur during pregnancy often unmask subclinical conditions that disappear after pregnancy but that may emerge later in women’s life. Pregnancy has therefore been considered as a “stress-test” for metabolic and cardiovascular conditions (36).

There are two differentiated metabolic stages during pregnancy. The first stage corresponds to the first two thirds of pregnancy, when fetal growth is limited. During this period the maternal metabolism is anabolic and directed towards storing accumulated nutrients. The last third period of pregnancy is a period of rapid fetal growth, and growth is supported by a maternal switch to catabolic metabolism and enhanced transfer of nutrients by the placenta. This metabolic balance is ideal to provide a continuous supply of nutrients of fetal and placental growth. In a population of women with impaired glucose tolerance (IGT) or GDM it was

found that obesity rather than glucose correlated best with fetal macrosomia (37). Only FPG levels at week 32-35 weeks of gestation correlated with fetal macrosomia. It is also speculated that obesity and metabolism are closer associated with macrosomia than hyperglycemia (38).

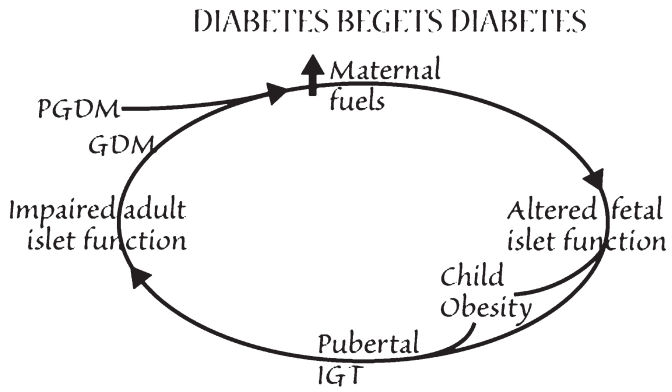
## 5 Gestational diabetes and glucose intolerance

GDM is described as carbohydrate intolerance of varying degrees of severity with onset or first recognition during pregnancy (39). The primarily reason to identify GDM was to detect women of risk to develop type 2 diabetes mellitus (T2DM) later in life. The focus has now shifted to identify women with GDM to detect pregnancies at risk for potential adverse outcome as a result of maternal hyperglycemia (40). In recent years it has been a global increase in the prevalence of obesity and T2DM, as well as an increasing prevalence of GDM (41). This may be due to increased maternal age, the epidemic of obesity and diabetes, decrease in physical activity and the adoption of modern lifestyles in developing countries (42). Considerable controversy exists regarding the thresholds and the numbers of the plasma glucose values considered for diagnosis of GDM during oral glucose tolerance testing (43). There still remains a need to develop diagnostic criteria for GDM that are based on the relationships between hyperglycemia and risk of adverse outcome (44). Pregnancy is characterized by an increased insulin secretion to compensate the increased requirements. In GDM the pancreatic ß-cell function is characterized by insufficiency to meet the body's insulin needs (45). Thus, robust plasticity of ß-cell function in the face of progressive insulin resistance prevail normal glucose regulation during pregnancy (46).

Women developing GDM is at high risk of developing type 2 diabetes mellitus (T2DM) later in life. As the registered prevalence of GDM in the United States varies from 3 – 12 %, it is inaccurate to predict who will develop T2DM (47). In Norway the registered prevalence of

GDM in 2006, was 10.6 per 1000. The lowest registered number was in the county of Telemark (3/1000), whereas the highest was in the neighbor county of Buskerud (19.4/1000) (1). In the same way as the metabolic syndrome, GDM can be seen as a stress test for the development of T2DM. There are indications that as many as 50% of women who develop GDM subsequently will develop T2DM (47). Women with previous GDM have a prevalence of cardiovascular disease (CVD) due to the cluster of establishing the metabolic syndrome (48). Many women have one or more additional pregnancies after the diagnosis of GDM, and a high proportion (> 50%) of these are reported to have GDM in the subsequent pregnancy (49).

**Figure 1**




Diabetes begets diabetes: the alterations of maternal fuel metabolism lead to altered fetal islet function (hyperinsulinism). This intrauterine event predisposes to, or identifies risk for childhood obesity and adolescent IGT, GDM, and later DM. Reproduced from Diabetes in Women. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2004 with permission (50)

In 1954 it was postulated that maternal hyperglycemia leads to high insulin and glucose in the fetus, the “Pedersen hypothesis” (51;52). Glucose is known to pass the placenta barrier, whereas insulin does not. Intrauterine exposure to maternal diabetes is associated with excess fetal growth in utero, possibly due to an increase in fetal fat mass and alterations in fetal hormones (53). Concentrations of insulin are raised, and it has also been shown that increase in fetal leptin is associated (54).

Historically, stillbirth was an important complication of diabetic pregnancies, whereas the most frequent types of morbidity now are associations with macrosomia and labor complications (55). Intrauterine exposure to an excess of glucose, may lead to permanent fetal changes, such as malformations, macrosomia, increased risk for obesity and developing T2DM in later life. Studies of the Pima Indians have shown that exposure to hyperglycemia in fetal life, are at increased risk of becoming obese and developing diabetes at young ages (54). The HAPO-study is a multicenter study with a total of 25 000 pregnant women at 15 different centers in nine countries. The results indicate strong and continuous associations between maternal blood glucose values, also below those diagnosed for GDM, with increased birth weight, primary cesarean delivery and cord-blood serum C-peptide levels. This study indicates the need to re-consider the criteria for diagnosing and treating hyperglycemia during pregnancy (56).

## 6 The role of intrauterine environment, Developmental Origins of Health and Disease (DOHaD)

David Barker proposed in the late nineteen eighties that newborns born small for gestational age performed higher risks of developing CVD, diabetes or obesity at an earlier age than newborns within a normal weight range (57). This work was, among others, based upon that of Forsdahl who in the late 1970s described that children with Finnish origin who grew up in Finmark (north of Norway) and were exposed to poor living conditions in childhood and adolescence experienced a higher mortality rate of coronary heart diseases when adult (58). As mentioned above, chronic NCD which include cardiovascular conditions, some cancers, and chronic respiratory conditions and T2DM are reaching epidemic proportions and account for 44% of premature deaths world wide. Commonly known risk factors include lack of

exercise and improper diet and smoking. Though these are diseases that are preventable, most countries have focused more on biomedical research and treatment rather than prevention (7). There is a growing body of evidence that intrauterine conditions may have a significant impact on future health and disease is becoming well acknowledged (57;59-65). “Intrauterine programming” is a process by which early insults at critical stages of development lead to permanent changes in tissue structure and function (66). This is now called the DOHaD concept, developing from the FOAD concept (Fetal Origins of Adult Disease). Studies suggest that early environmental factors may change development without affecting birth weight, and the most widely accepted phenomenon is that of programming. This is described as a process whereby a stimulus or insult has irreversibly long-term effects on development (67). There may be three different pathways of relevance. One is the so-called “mismatch” pathway, which reflects the studies by Barker and his group, describing newborns with impaired fetal growth and postnatal excessive weight gain. Another potential pathway are newborns with rapid weight gain, often due to formula feeding, whilst the third pathway reflects the macrosomic infants often born to diabetic or obese women, and who later become insulin resistant or obese (68).

## 7 Determinants of fetal growth

### *7.1 Pregnancy, nutrition and birth weight*

The placenta plays an important role as a new endocrine organ established only a few weeks of conception, secreting hormones that affect the nutrients metabolism. The adjustments are driven by hormonal changes, fetal demands and maternal nutrient supply, and each nutrient has more than one potential adjustment (69). There are thorough distinctions between

maternal nutrition and fetal nutrition. The fetus grows at the end of a long "supply line", starting with maternal diet at one end and fetal tissue uptake at the other (70).

**Figure 2**


The fetal supply line. Factors along the fetal 'supply line' which can mediate the differences between maternal nutrition and fetal nutrition. (71)

The placenta plays an important role in the fetal supply line and influences fetal nutrition via its own metabolic demand for nutrients. Lactogen and growth hormone that are produced by placenta contribute to maternal insulin resistance. The availability of glucose and other nutrients in the maternal circulation may increase the transfer to the fetus (71).

Most studies of the relationship between maternal nutritional status during pregnancy and glucose tolerance in offspring have looked at the effect of reduced maternal nutrition during pregnancy. Only a few studies have investigated the periconceptual nutritional status and long term health status. In sheep models it has been found that maternal periconceptual undernutrition impairs glucose tolerance in postpubertal offspring. Maternal nutrition around the time of conception may therefore have important implications for glycaemic regulation in the next generation (72).

The Dutch famine that occurred near the end of World War II is the most well known study on humans in relation to under nutrition during pregnancy. The association to adult health of infants born by pregnant women exposed to famine was related to the stage of pregnancy when exposed. Exposure of starvation during any stage of gestation was associated with impaired glucose tolerance (73), exposure to famine during early gestation was related to obesity in women (74) and a greater prevalence of coronary heart disease (75). Exposure of

famine during the last trimester of pregnancy reduced birth weigh by around 10% (75). Early, mid- or periconceptual exposure did not affect birth weight in one study (76), but mid-pregnancy exposure mildly reduced birth weight, and early gestation exposure slightly increased birth weight in another study (75). Several epidemiological studies are related to fetal nutritional deprivation as a strong programming stimulus. In many societies, however, maternal and postnatal nutrition are either sufficient or excessive. Obesity and excessive weight gain may therefore be the more common nutritional problems related to pregnancy complications in developed countries. Not many studies have investigated long-term consequences of maternal nutrient excess during pregnancy or lactation on development of obesity in offspring (77).

Growing evidence has pointed out that overweight or obesity and excessive weight gain during pregnancy is important maternal factors that increase the risk for macrosomia. The direct link between nutritional associations and birth weight is not obvious. It has been found that a maternal dietary pattern in pregnancy based on red and processed meat and high-fat diary, was associated with increased risk for small for gestational age (SGA) (78) whereas others have found that nutritional intake during pregnancy is not reflected with birth size variables (79). Other studies as well report negative associations between energy, carbohydrate and fat consumption in early pregnancy and size of newborn or placenta at birth (80;81). In Australia it was found that dietary composition of women in early, but not in late, pregnancy was associated with an increased size of infant at birth (82).

The influence of micronutrients on birth weight during pregnancy is less investigated. One human study investigating 20 different vitamins and micronutrients, found that only vitamin E was associated with birth weight (83). However, a study of Wistar rats during pregnancy found that high vitamin intake during pregnancy increases the phenotypic expression of obesity and components of the metabolic syndrome in both female and male rats fed an

obesogenic diet (84). Micronutrient metabolism and absorption can be influenced by dietary nutrient balance, and by factors such as infection, genetics and smoking (85).

Most information on pregnancy dietary composition is mainly based on studies in mid- to late pregnancy. However, the importance of periconceptual intake of nutrients, such as folate acid is established (85). Maternal weight at conception probably influences metabolic adaptation to pregnancy and may also indicate the importance of prepregnancy nutrition (69).

*7.2 Physical activity and exercise, pregnancy and birth weight*

Prior to the 1980s, the guidelines restricted pregnant women from strenuous exercise, believing that this would deprive the fetus of oxygenated blood flow and metabolic fuels causing fetal distress and growth restriction(86). Primary recommendations to promote and maintain health in healthy adults is moderate-intensity aerobic physical activity for a minimum on 30 min on five days each week or vigorous-intensity aerobic physical activity for a minimum of 20 min on three days each week (87). The American College of Obstetricians and Gynecologists (ACOG) now supports the guidelines that in the absence of either medical or obstetric complications, pregnant women also can adopt this recommendation (88). However, the optimal dose of recreational physical activity for a pregnant women remains to be determined, and the impact of prolonged and repeated aerobic exercise on outcome of clinical importance for mothers and infants is unknown (89;90). Some observational studies have associated physically demanding work with increased risk of preterm birth (91;92). It has also been reported increased risk of early miscarriage in pregnancies associated with high impact exercise (more than seven hours/week) (93).

However, a growing body of evidence supports the idea that physical activity is beneficial in preventing or treating maternal-fetal diseases. The health risk associated with continuation or adoption of a sedentary lifestyle during pregnancy may contribute to conditions such as

childhood obesity, GDM, preeclampsia, deep vein thrombosis and poor psychological adjustment to the physical changes of pregnancy (86). Many studies are related to glucose values and the risk of GDM, and evidence shows that regular physical activity before pregnancy is associated with lower GDM risk (94;95). As GDM is a risk factor for macrosomia, reducing GDM will reduce the risk of delivering macrosomic infants as well. Physical activity among GDM's reduces the prevalence of obesity, which is known to be strongly associated with macrosomia (96-98). The long-term effects of continuing exercise during pregnancy has recently been evaluated. By following women 18–20 years after pregnancy it was shown that women who continue vigorous recreational exercise after their index pregnancy maintain their long-term fitness and have a low cardiovascular risk profile in the perimenopausal period (99). Most studies on physical exercise and birth outcomes are small and they measure different outcomes. It is not always specified whether they measure physical activity (defined as any bodily movement produced by skeletal muscles resulting in energy expenditure), exercise and/or training (repetitive bouts of exercise with the intension of developing physical fitness) or moderate exercise (corresponding to brisk walking) (100;101). Also, there are methodological aspects as how to best assess information of physical activity among women during pregnancy.

One of the main concerns about physical exercise during pregnancy has been the fear of growth restriction due to competition with maternal environment for excess to main energy resource or that physical activity reduces insulin secretion and may cause reduction of fetal growth (86).

The reasons for discrepancy in studies concerning prenatal physical activity and birth weight are complex. The estimates of total activity load may be inaccurate. The effects vary with differences in type, intensity, duration and frequency and the exercise effects can be modified by maternal diet (102). It has also been shown that reduced birth weight delivered by

exercising mothers, was due to reduction of fetal fat mass, or contrary, that that reduced fat mass in the newborn was associated with brisk walking during pregnancy by the mother (103;104).

Validated questionnaires in prospective studies measured at different time points during pregnancy that measures the type, frequency and duration of physical activity or accelerometers worn by the pregnant women is the most reliable tool to assess physical activity during pregnancy (90). Also, asking women to recall pregnancy recreational physical activity after delivery has major limitations because delivery experience and pregnancy outcome are likely to bias maternal recall (90).

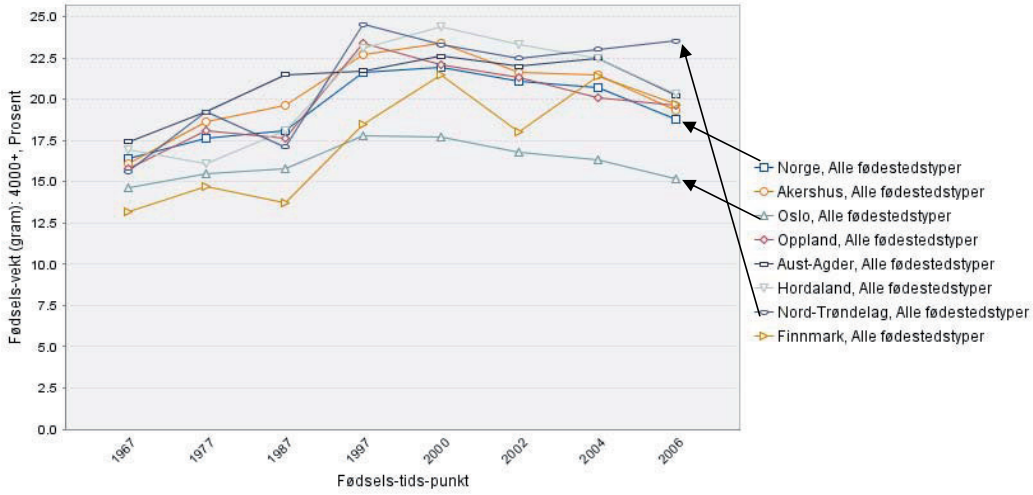
## 8 Prevalence of macrosomia

Newborn macrosomia is an increasing obstetric challenge worldwide. It seems closely related to the current epidemics of obesity, found both in the westernized parts of the world and the developing countries. In Norway the prevalence of macrosomia is high, and the prevalence of newborns weighing 4000g or more has increased from 16% in 1968 to about 20% in 2006 (1). The percentage of newborns with high birth weight has been reduced the last ten years in Norway but the variations across the country are high. In Oslo, 15% of the newborns weighed 4000g or above in 2006, whilst in Nord-Trøndelag 23% of the newborns weighed more than 4000g. At the same time period there has been a significant decrease in infants born after gestational week 41 or 42 (1). This is illustrated in Figure 2. Some possible explanations have been suggested, but systematic investigations are lacking. There might have been a shift in obstetric management, reducing the macrosomic infants by identifying the rapid growing fetuses by ultrasound. The number of immigrants is higher in Oslo than in other parts of the country, especially from south Asia. In average, their infants weigh approximately 300g less

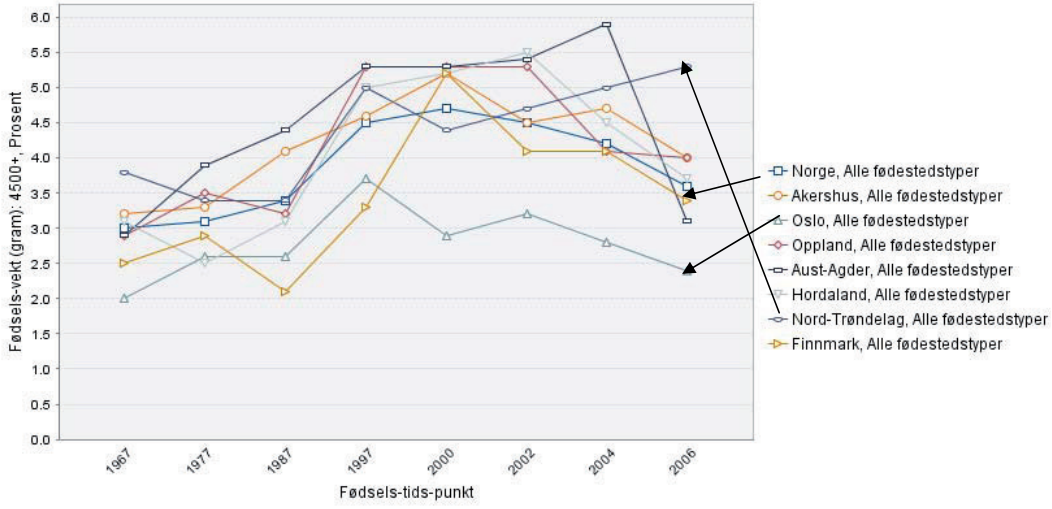
than Norwegian newborn (105). In addition, the urban population may have life style factors that differ from women in rural Norway.

**Figure 2 a**

Birth weight above 4000g and 4500g in different counties in Norway (from Medical Birth Registry (1))



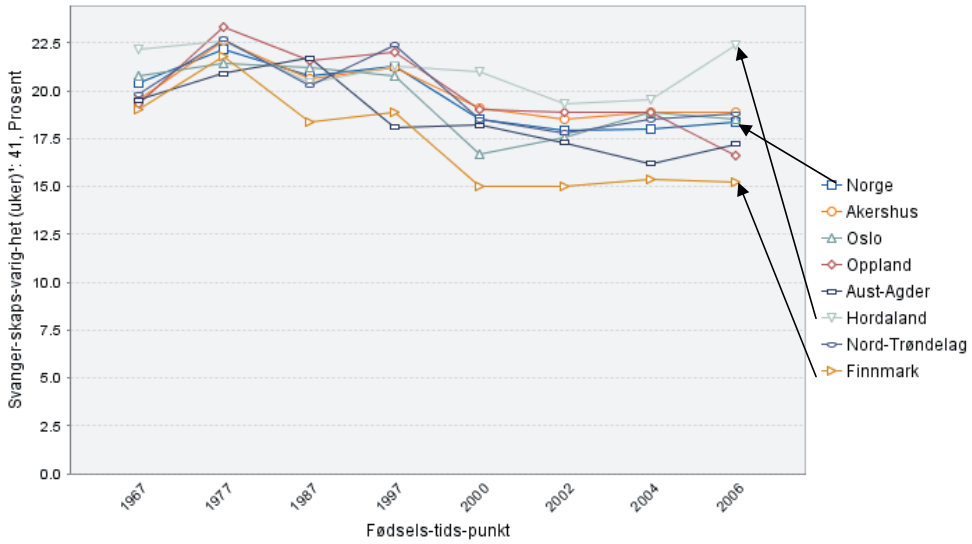

Birth weight > 4000g



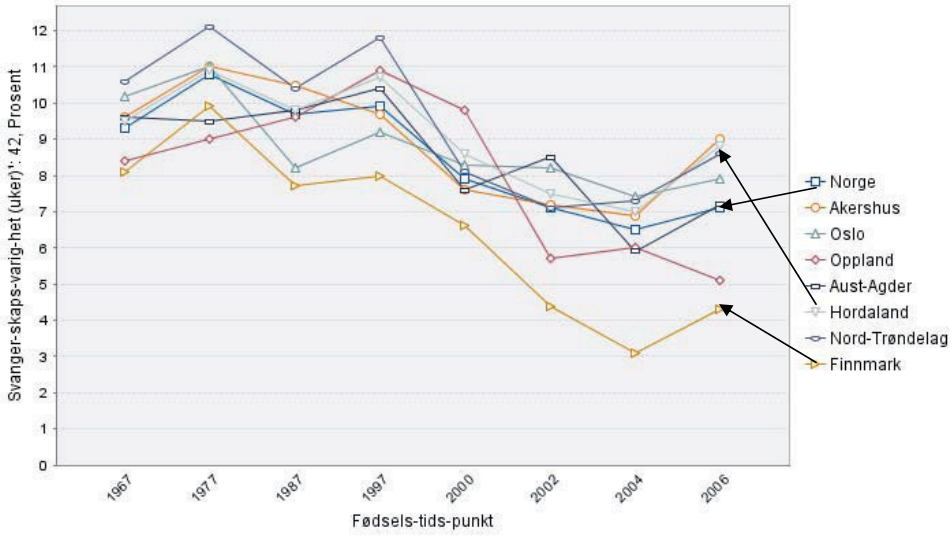

Birth weight > 4500g

**Figure 2 b**

Gestational age above 41 weeks and 42 weeks in different counties in Norway (From Medical Birth Registry (1))




Gestational age > 41 weeks




Gestational age > 42 weeks

*8.1 Definitions of macrosomia*

There are no existing international or national definitions of a macrosomic newborn. Commonly used definitions are based on birth weight in grams. Suggested cut-points for the macrosomic definition are newborn weight above 4000g, 4500g or 5000g (106). These cut-points seem random, referring to a "round number", as a multiply of five or 10. Similarly, cut-points based on observed percentiles have been suggested. Birth weight above the $90^{th}$ or $95^{th}$ percentile is used to define LGA. Correspondingly, SGA is defined as birth weight below the $10^{th}$ percentile, whereas those between $10^{th}$ and $90^{th}$ percentile are average for gestational age (AGA). Relative birth weight (ponderal index; weight/length $^{3}$) is another method to evaluate newborn body composition. One consequence is that various definitions of macrosomia and various cut-points have been used in different studies, which make comparison difficult (107). However, there is an increasing focus of the infant's body composition rather than birth weight (13). Birth weight alone may be a too crude measure of anthropometric and metabolic features of relevance for short and long term health of the newborn.

## 9 Consequences of macrosomia

Giving birth to a macrosomic newborn include short- and long-term consequences for both the mother and the newborn infant.

*9.1 Maternal health consequences, short term risks*

Unexplained intrauterine death is often associated with fetal macrosomia (108). Women delivering macrosomic infants more often experiences a number of adverse birth outcome (109-112) and prolonged labor (110). Prolonged second stage of labor may increase the risk of postpartum hemorrhage (113), the incidence of acute cesarean section (114) or vaginal

operative delivery (111). Delivering of a macrosomic fetus also increases the risk of third or fourth degree lacerations (110). It has recently been shown that the use of epidural analgesia (EDA) as pain relief during labor is associated with high birth weight (115). As both the use of EDA and macrosomic infants are associated with prolonged labor this needs to be investigated further.

*9.2 Maternal health consequences, long term risks*

Long term maternal consequences related to the delivery of a macrosomic fetus must be seen in relation to the actual delivery. A number of women develop post traumatic stress symptoms post partum, and it seems that obstetric interventions increase the prevalence (116). Serious complications in nulliparous may have consequences in regard to reproductive health. Some women suffer persistent perineal defects and urinary and anal dysfunction after perineal lacerations (111).

*9.3 Neonatal health consequences, short term risks*

Newborn macrosomic infants are associated with traumas such as shoulder dystocia, clavicular fracture, hypoglycemia and hyperbilirubinemia, and are therefore more often transferred to neonatal intensive care unit after birth (111;117). Immediate skin-to-skin contact between the mother and infant promotes breastfeeding initiation and duration and strengthens the maternal attachment behavior (118).

*9.4 Neonatal health consequences, long term risks*

High birth weight is related to obesity and the metabolic syndrome in adult life (63;119). Being born macrosomic by a mother with a high BMI often in a combination with GDM or

T2DM increases the risk of developing T2DM in early life of the offspring (120). Especially for young females this may influence later reproduction (96).

## 10 Causes of macrosomia

Birth weight is a measure of fetal growth, and is a composite of many components including bone, internal organs, muscle, fat and fluids. These may be determined both by genetically and environmental regulatory mechanisms (121). The genetic part probably plays a small role in the recent change in birth weight distribution, because genetic changes will take generations to affect birth weight. Prediction and explanation of the increasing prevalence of macrosomia have been subjected to a number of studies (13;53;117;122-128). The causes are multifactorial, and the cause may somewhat schematically be divided into non-modifiable and modifiable explanations.

### *10.1 Non-modifiable explanations*

Among the known non-modifiable variables influencing birth weight is maternal age, parental birth weight, parity, gestational age and gender of child (117), although it has been shown that maternal age has less impact when controlled for parity (13). Genetics and transgenerational inheritance has also shown to have impact on birth weight, but to which extent has not been clearly clarified (129-131).

### *10.2 Modifiable explanations*

Of the modifiable variables we find maternal anthropometry such as pre-gestational weight, BMI, body composition and gestational weight gain (53;98;125;126;132-135). Smoking is well documented as reducing birth weight (136;137). The level of physical activity may also

be associated with birth weigh (138), intake of nutrients (both macro-and micronutrients) and metabolic parameters (139-141). Pre-gravid BMI predicts fetal weight and macrosomia (13;20;142), as well as weight gain during pregnancy (30;143).

*10.3 Prevention or treatment of macrosomia?*

The body composition of the newborn probably also plays an important role. This is related to the maternal body composition, such as maternal birth weight, height and weight, weight gain and subcutaneous fat. The endocrine intrauterine environment also affects body composition, though much less is known of the impact of the glucose an insulin metabolism, as well as inflammatory factors.

*10.4 Identifying macrosomia*

In the discourse of the delivery of macrosomic infants the focus is often on the detection of macrosomia. The methods used may be either clinical or ultrasound estimations, and in most cases the clinical estimation is about as accurate as those derived from ultrasonography. The accuracy by two-dimensional ultrasound seems to predict birth weight up to 3500g pretty well (144). The problem arises, however, when the fetus is getting larger. Increased maternal BMI has shown to be associated with reduced accuracy in estimating fetal weight by ultrasound (145). It is thought that by identifying the macrosomic fetuses, birth outcome would improve. However, induction of labor for suspected macrosomia in non-diabetic women appears to increase the Cesarean Section (CS) rate, without improving perinatal outcome (146). Prevention of macrosomia would therefore be the best way to avoid the birth complications associated with the delivery of a large infant.

## 11 The current study

*11.1 Study design*

The study followed a prospective cohort design. However, a retrospective assessment of physical activity was chosen. The inclusion period was from 2002 to 2005, and the cohort consists of 553 pregnant women and their newborns.

Definitions: Macrosomia was defined as birth weight ≥ 4200g. Gestational age was based on ultrasound at weeks 17 – 19.

*11.2 Characteristics of the cohort*

Healthy pregnant women of Scandinavian heritage booked for birth place at Rikshospitalet, Oslo University Hospital were invited to participate in the study. Women with multiple and pre-gestational diabetes were excluded.

About 2000 invitations were sent out; five hundred and eighty-eight women attended the study. Of these, 16 were excluded due to fetal malformations, duplex or spontaneous abortion and 19 were lost to follow up delivering at another hospital or moving out of the area. The participants were subjected to a scheduled prospective follow up, with four visits during pregnancy (Table 2). The first visit was conducted between gestational week 14-16 (visit one). Maternal anthropometric measures considered to be potential predictors of macrosomia were collected at each visit. Weight (kg) without shoes and heavy clothes was measured on a digital scale, followed by calculation of BMI. Subcutaneous fat was estimated using a Holtain calliper (Holtain, Crymych, UK).

A standard oral glucose tolerance-test with 75 g glucose after 10-12 h fasting was performed twice (visit one and three). At visit one and three the participants filled out a self-administered quantitative food frequency questionnaire. Intake of macronutrients during pregnancy was calculated as the mean of the values obtained at visit one and three.

Physical exercise was assessed by a questionnaire. It was handed out at gestational weeks 32 and collected at weeks 36, describing physical activity before pregnancy at each trimester.

**Table 2:** Design and follow up through pregnancy

| | Weeks of gestation | | | |
|---|---|---|---|---|
| **Investigation** | **Visit 1** | **Visit 2** | **Visit 3** | **Visit 4** |
| | **14 -16** | **22 – 24** | **30– 32** | **36 - 38** |
| General follow up * | + | + | + | + |
| Glucose tolerance test | + | | + | |
| Fasting blood samples † | + | + | + | + |
| Fetal ultrasound ‡ | | + | + | + |
| Food intake § | + | | + | |
| Physical activity ‖ | * | * | * | + |

* Including blood pressure, weight, sub-scapular skin folds
† Serum EDTA, citrate plasma and buffy coat for immediate freezing at -70° C
‡ Fetal growth rate assessed by ultrasound and Doppler flow profiles in the uterine and umbilical arteries
§ Food frequency questionnaire
‖ Level of physical exercise pre-gestational and at each visit was obtained by a questionnaire answered at visit 4.

## 12 The overall aim

The overall aim of the present study was to extend insights into modifiable determinants of fetal overgrowth as reflected in newborn macrosomia and related delivery complications

**Paper 1:**

In this study we particularly wanted to investigate the effect of maternal body mass, fasting glucose values and physical inactivity on the risk of newborn macrosomia. In previous studies it has been questioned whether BMI and plasma glucose are independent determinants of newborn macrosomia. The role of physical activity was by large unknown.

**Main hypothesis:** Physical inactivity, body mass index and plasma glucose play an independent role in determining newborn macrosomia

**Paper 2:**

As delivery complications are associated with newborn macrosomia, we also set out to investigate the role of determinants of macrosomia on delivery complications.

We therefore investigated if physical inactivity has an independent effect on birth outcome besides contributions by other modifiable factors, including nutritional intake, plasma glucose levels, and maternal weight gain.

**Main hypothesis:** Physical inactivity before and or during pregnancy is associated with delivery complications.

**Paper 3:**

In the general non-diabetic population it is not well understood why some women with high BMI give birth to macrosomic newborns while others do not. Given the central role of glucose in mediating fetal overgrowth in GDM and the metabolic **continuum** between individuals

with and without GDM, we wanted to explore the effects of change in fasting plasma glucose, insulin and insulin resistance in different body mass index groups on the risk of newborn macrosomia in a population of pregnant women who did not develop insulin-dependent GDM.

**Main hypothesis:** The effect of gestational rise in fasting glucose on the risk of newborn macrosomia is dependent on maternal body mass index.

**Paper 4:**

There is some evidence that determinants of birth weight have different effects on the sexes. We therefore wanted to examine whether parental anthropometrics, parental birth weight and fasting maternal plasma glucose levels influence birth weight differently in the two sexes.

**Main hypothesis:** Maternal and paternal determinants of birth weight have different effects in the two sexes.

# 13 General discussions

## 13.1 Main endpoints

### *13.1.1 High birth weight*

The main outcome in this study has been high birth weight (macrosomia). The choice of using absolute birth weight and not relative birth weight was based on two arguments. First, birth weight gives newborn size which is more closely related to delivery complications than measures of relative birth weight. Secondly, current evidence indicates that among newborn anthropometric parameters birth weight is a better predictor of adolescent fat distribution than is relative birth weight (147). Furthermore, reduced relative birth weight show a poorer association with fat mass in newborn than does birth weight (148).

Dual energy X-ray absorptiometri (DXA), first developed for assessment of bone mass, provides information on total fat mass (FM) and fat-free mass (FFM) or "lean" soft tissue, and their distribution in the trunk and upper limbs(149-151). Over the past decade, DXA has been increasingly used to assess body composition in research and clinical practice, including applications to direct treatment (152;153). DXA is generally accepted as a precise noninvasive technique to assess body composition in vivo, and there is a large quantity of data about the method in adults (154-158). Body composition would have been a potentially important supplement to birth weight in the current study. However, DXA-measurement of newborns was not available at the start of the project. Besides birth weight, fetal body composition near term, especially percent body fat, may be an independent determinant of delivery complications. This notion is supported by the fact that at any given birth weights, infants born by diabetic women have higher risk of shoulder dystocia. The rise in birth weight the last generation may also have been accomplished by an increased percentage of body fat. Interestingly, it has been registered that in a general obstetric population the need for operative delivery at a given birth weight was markedly increased from the 1990's as compared to the 1940's (3). Further studies should therefore take into consideration the role of body composition for birth complications.

*13.1.2 Delivery complications*

Normal birth may be described as spontaneous onset of labor with a normal progression without drugs and spontaneous delivery. There seems be no generally accepted definitions of birth complications. In our study we have chosen CS (emergency and elective), operative vaginal delivery (forceps and/or vacuum extraction), severe haemorrhage (≥ 1000 ml) or perineal laceration of third or fourth degree as definitions of birth complications. The choices have been based upon clinical practice. We have only included term deliveries in the analyses,

and the inclusion criteria excluded mothers with severe diseases or diabetes and multiple pregnancies. Apart from preterm deliveries, we have included all the women in the study group in the analyses, including those with pregnancy complications. Some of these may have been chosen for induction of labor or elective cesarean section. It is well known that obstetric interventions in labor tend to lead from one to another (159).

## 13.2 Methodological considerations

### *13.2.1 Is the cohort representative?*

The population in this study is extracted from the women offered delivery service at Department of Obstetrics and Gynecology, Rikshospitalet University Hospital. Around two thousand women give birth at the hospital every year. We selected women with a Scandinavian background, and excluded women with known pregestational diabetes, serious heart, gastrointestinal, pulmonary or renal disease, as well as multiple pregnancies. The women asked to participate in the study were chosen "randomly" from the booking list without any selection beyond the exclusion criteria. The number of inclusions per month had to be adjusted according to logistic frames of the study. Thus, approximately five women could be included each week. Due to these limitations, we could not invite all the eligible women to participate in the study. Around one third of the women approached accepted the invitation. These circumstances might have caused a selection bias.

We therefore compared the women delivering at Rikshospitalet, Oslo University Hospital who declined participation with the women accepting. We found that age, parity, work load and education were equal in the two groups. The women in the study group were slightly heavier around conception time, but they gained less weight throughout pregnancy than the non-participants. We also compared the women in the study with a randomly sampled group of Scandinavian women at another University clinic in Oslo (Ullevål, Oslo University Hospital).

The study group and the Ullevål group were comparable except that the prevalence of single mothers and women smoking during pregnancy were slightly higher at Ullevål, Oslo University Hospital.

This information makes us to assume that the cohort is fairly representative for an urban Norwegian population. However, the higher maternal age and education level presumably makes the current cohort to deviate from a rural population in Norway.

### 13.3 Data collection

*13.3.1 Assessment of physical activity, the questionnaire*

A self-reporting questionnaire (Physical Activity and Pregnancy Questionnaire (PAPQ)) designed by The Norwegian School of Sport Sciences was used for this study. When the study started, no validated questionnaires for pregnant women existed to our knowledge. There are few reports and limited knowledge about level of physical activity or exercise during pregnancy. One aim was therefore to assess level of total physical activity (at work, in transportation, household and recreational exercise), both pre-gestational and trimester-specific (100). The questionnaire was distributed at gestational weeks 32, and returned at 36 weeks of pregnancy. It may be argued that the physical exercise data were obtained retrospectively in a study with a prospective design. The pre-gestational data had, however, to be obtained retrospectively, anyway, which made us choose a retrospective method for all the physical activity data.

To our knowledge, there has not been performed many studies where exercise data was assessed prospectively before pregnancy. When planning the study, the general assumption was that physical activity during pregnancy would have greater impact than pre-gestational exercise had. We therefore chose to ask the women while pregnant to secure the data during pregnancy, rather than after delivery.

*13.3.2 Maternal body mass index and fat mass*

In this study we have obtained maternal anthropometric parameters to describe body composition. At each visit every women was weighed on the same scale, in addition to sampling anthropometric measures with a caliper from three sites of the body (sub scapular, triceps and iliac crest). Height was self reported. In a subgroup of 26 women we measured their heights after reporting, and the correlation was $r_p = 0.96$. In the analyses we have chosen to use BMI. This is a common and well known method, and the results were almost identical when we used the parameters from the calipers in the analyses. BMI is a crude method of describing body composition, as muscle mass and fat mass are not discerned. Studies of adults in the normal to mildly obese BMI ranges have shown that the relationship between BMI and percent body fat differs by age and gender. One longitudinal study, however, has determined the relationship between BMI and percent body fat in overweight/obese women (BMI > 25) before and during pregnancy. The correlation between BMI and percent body fat remained significant during pregnancy. However, the correlation weakened as the pregnancy advanced (160). In the current study BMI was assessed at 14-16 weeks as pregestational BMI could not be obtained with sufficient validity.

*13.3.3 Food frequency questionnaire*

The questionnaire was evaluated for a Norwegian population in 1997 and found suitable (161). However, it has not been tested on a pregnant population, but at the time when the study started there was to our knowledge not any such questionnaire available. Food habits have changed the recent years, and some of the responders commented that it did not quite cover their diet. However, energy intake and intake of macronutrients may no have changed to the extent that is the case with food items.

*13.3.4 Newborn birth weight*

All the newborn children were weighed within two hours in the delivery room by the attendant midwife or the assistant. The scales are identical and regularly calibrated. This is a well established routine at the ward. Thus the assessment of the main endpoint, birth weight is considered valid.

*13.3.5 Fasting plasma glucose and insulin*

The women came for four antenatal visits during pregnancy in the early morning starting with drawing blood samples after an overnight fast. At gestational weeks 14-16 and 30-32 FPG were measured. A standard oral glucose tolerance-test with 75g glucose after 10-12 h fasting was performed twice (visit one and three). Plasma glucose was measured immediately in EDTA blood, venous, by Accu-Check glucose test strips and glucometer (Roche Diagnostics, Basel, Switzerland). Fasting plasma insulin values (FPI) were measured at weeks 14-16, 22-24, 30-32 and 36-38. Samples were collected in 7 ml Vaccutainer tubes, centrifuged without delay at room temperature at 3000 G for 10 min. Serum was aliquoted immediately and stored at -80 ºC until analyzed. Insulin samples were assayed in duplicate (RIA, DPC, Los Angeles, CA, USA) and the intra- and inter-assay CV were 4.9% and 5.4%, respectively. Insulin resistance (HOMA-IR) was calculated by the equation presented by Matthews's et.al. (162) and validated by Kirwan et.al (163) in a pregnant population ((FPG * FPI) / 22.5).

Due to the large scale of this study, we chose to measure glucose with a glucometer validated in tests in our clinic against standard laboratory methods. We are aware that glucometers formerly demonstrated low reproducibility of results, however in recent years the test quality has improved considerably (164;165).

## 14 Discussion of the results:

*14.1 Statistical considerations*

In this study, we have followed the tradition of dichotomizing continuous variables, presenting the results in multiple regression analyses. As cut-off points we have used clinical experience and traditional cut-off points.

1. **Birth weight**: There are no clear definitions of macrosomia, the most common cut-offs are 4000g or 4500g, or above the 90$^{th}$ or 95$^{th}$ percentile of the population. Mean birth weight and the prevalence of small and large for gestational age vary strongly about different populations. SGA is often defined as birth weight below the 10$^{th}$ percentile. The 90$^{th}$ percentile in Norway has the past five years been approximately 4200g (106). The risk of delivery complications increases when birth weight reaches the range of 4200-4500g (111).

   On this background birth weight ≥ 4200g was chosen as definition of macrosomia in the current study.
2. **Body mass index**: Median BMI in this study was approximately 25 women with BMI > 25 are classified as overweight (WHO), and this was a natural cut-off point.
3. **Plasma glucose**: The glucose values are measured to identify possible gestational diabetes and high birth weight. Accumulating data indicate that there is a continuous (none-brake point) relation between plasma glucose and obstetrical complications (HAPO)(56). We also had an interest in evaluating the pregnant women with high glucose values, but below the definition of diabetes. We therefore chose to use quartiles, comparing the upper quartile group with the rest.
4. **Pathological post partum haemorrhage** in Norway is defined as bleeding above 500 ml. It is usually estimated visually, an inherently inaccurate method. By choosing

1000 ml as a cut-off, we considered it less likely to include women with normal post partum blood loss.

5. **Physical activity:** Often physical activity is measured in METS (metabolic equivalent task). Our questionnaire did not allow conversion into METS. But by combining the answers of the questions 1) How often do you exercise (times per week), and 2) For how long do you usually exercise (minutes)?, information of the mean weekly time spent on physical exercise was obtained. Women exercising less than 1 hour/week were defined as physically inactive.

Clinical research has a tradition of converting continuous variables into categorical variables by grouping the values into two or more categories, while in epidemiological studies it is customary to create several categories or continuous variables (107).

Data which are subjected to clinical decisions during a project constitute methodological challenges. Especially in paper 2 selection mechanisms caused by decisions of elective and acute CSs, makes analysis of confounding factors a complex task. It can not be ruled out that possible confounders might have contributed to the results if they were to be considered at a later stage in the process. This is illustrated by the notification about effect of macrosomia on operative vaginal deliveries. However, this counterfactual way of thinking was considered beyond the scope of this work.

*14.2 Physical activity*

Paper 1 and paper 2

The relation between size at birth and level of maternal physical activity is not straightforward (166). Most studies of relations between physical exercise and birth weight have actual weight as endpoint, and have mainly studied effect of physical exercise during pregnancy. The effect

of a given exercise regimen on actual birth weight depends upon several factors including type, frequency, intensity and time point in pregnancy (103;167).

In the current study the participants were asked if they performed a certain level of physical exercise or not before and/or during pregnancy. By this definition about 1 in 10 women reported low level of exercise. The cut-off we used in this paper defined women exercising less than one hour per week as physically inactive.

However, we also found an effect on macrosomia when a cut-off level of physical exercise at 1.88 hours per week was used (unpublished data). Our findings indicate that being physically inactive before pregnancy is associated with increased risk of fetal macrosomia.

Interestingly, we found no relationships between physical exercise before pregnancy and BMI at week 14 to 16 ($p = 0.28$). We did not find any effect of physical exercise during pregnancy on the risk of macrosomia. Clapp et al (166) have shown that both the timing and the type of exercise during pregnancy are important variables that influence the fetoplacental growth, and may accordingly influence birth weight**.** The current study is another indication that timing of exercise relative to the gestational period may be a determinant of fetal size. The finding that high BMI and physical inactivity before pregnancy were independent determinants of macrosomia indicate that the metabolic status of the mother at the start of pregnancy may have significant influence on growth and fat deposition of the fetus (21).

Although macrosomia was the endpoint of the current study we did also find a higher *mean* birth weight of the infants delivered by the women being physically inactive before pregnancy (3833g versus 3660g, $p = 0.06$) (unpublished data). Thus, also the mean birth weight of a population may be affected by the level of pre-gestational physical exercise.

Evaluating physical activity with a questionnaire during week 32-36 has limitations.

It is, however, unlikely that there has been a recall bias among women who delivered macrosomic newborns or later suffered perineal lacerations, i.e. that these women reported

lower level of physical activity than they actually performed compared to the women who delivered macrosomic infants or with less risk of perineal lacerations.

The mechanisms that may underlie the effect of physical inactivity can only be subject of speculations. Poor physical condition of the mother may reduce the capacity to push resulting in prolonged second stage which is associated higher risk of perineal lacerations (168;169). Physical inactivity may also be associated with poor function of the pelvic levator muscles resulting in insufficient rotational forces and prolonged second stage of delivery.

*14.3 Food frequency questionnaire*

The apparent absence of effect of energy and macronutrients should be interpreted cautiously as previous studies have shown effects of maternal diet during pregnancy on average birth weight (80-82;170). Consumption of carbohydrate with high glycaemic index seems to be associated with higher average birth weight. Its role in determining risk of fetal macrosomia is, however, unresolved. It may be argued that the study group consisted of a group of well nourished women, which will support the fetus of sufficient nutrients throughout pregnancy. This means that these fetuses never have been undernourished. Glucose passes freely across the placenta; the transport of amino acids and other nutrients is not that simple. We chose to analyze the macronutrients (fat, carbohydrates, protein and per cent of total energy), eliminating the micronutrients, knowing that this also may influence birth weight. We also selected to calculate mean values of macro nutrients, questionnaire answered at two different time points during pregnancy (visit one and visit three). Energy intake is closely related to energy expenditure. These are calculations we did not analyze.

*14.4 Glucose values*

We found that fasting glucose plasma values at week 30-32 predicted high birth weight better than the two hour oral glucose tolerance test (OGTT). This being the best predictor we therefore chose to use this parameter in the analyses. During the study period we realized that this was in accordance with other recent studies, which suggests that the fasting values are better predictors for high birth weight while two-hour OGTT better predicts the risk of developing diabetes later in life (40;56).

## 15 Limitations of the study; internal and external validity

Women have been asked to participate in a study that is quite time consuming. We do not have information about the reasons why some women declined to participate. This study can only give information about those who wanted to go through all the visits. It was confirmed by several women that being offered three extra ultrasound examinations motivated for participation. This is a study undertaken in one of the maternity hospitals in Oslo, where approximately 2000 newborn infants are born each year. As data from The Medical Birth Registry shows there are variations throughout the country regarding both mothers and the newborn infants anthropometric measures and birth outcomes(1). This study encompasses an urban population. As mentioned above, the differences in maternal characteristics of the population in the study group and Ullevål University Hospital delivery unit, was similar. One may therefore assume that the results in this study are representative not only for the population studied, but also for women living within the same urban conditions.

*15.1 Food frequency questionnaire*

The food frequency questionnaire was detailed and time consuming to fill in. Even though it was complicated and did not always cover their food habit, most women did. Only four participants did not hand it in at the first visit, and 41 failed to deliver it at the third visit. As the questionnaires were pre-numbered and to be read electronically they were not substituted if lost.

*15.2 Physical activity questionnaire*

The purpose of the physical activity questionnaire was to assess the total physical activity level of pregnant women, and to investigate the association between weight gain, physical activity and exercise during pregnancy, and to evaluate the influence of physical activity on newborn weight and pregnancy outcome. The questionnaire has now been validated with a portable activity monitor (ActiReg®, PreMed AS, Oslo, Norway), but not yet published (171). Seventy-seven pregnant women wore the ActiReg sensors during waking hours for seven consecutive days and answered the PAPQ. The results indicated only small differences between the PAPQ and the ActiReg in cross-tabulation of total physical activity level and proportion of participants meeting the current exercise guidelines. The study therefore concludes that there may be several advantages in combining these two types of instruments for registrations of physical activity level during pregnancy. Further studies may benefit from using both questionnaire and motion sensors.

## 16 Summary of results

1. Body Mass Index / fat mass

We found the same results when using BMI or calculated fat mass assessed with caliper measures.

- Women with BMI higher than 25 kg/$m^2$ in early pregnancy have increased risk of delivering an infant weighing ≥ 4200g

2. Weight and weight gain throughout pregnancy

- Women gaining more than 10 kg from weeks 14-16 to 36-38 have increased risk of delivering an infant weighing ≥ 4200g

3. Nutrition

- There was no significant association between intake of fat, carbohydrates or protein and risk of delivering an infant weighing ≥ 4200g

4. Plasma glucose

- Fasting plasma glucose measured at weeks 30-32 gave the strongest association with the risk of delivering an infant weighing ≥ 4200g
- Increasing fasting plasma glucose between weeks 14-16 and 30-32 in women with high BMI (BMI > 27 (upper quartile)) is associated with the risk of delivering an infant weighing ≥ 4200g

5. Fasting plasma insulin

- There were no significant association between fasting plasma insulin and the risk of delivering an infant weighing ≥ 4200g

6. Physical inactivity before pregnancy (less than one hour/week)

- Women who defined themselves as physically inactive before pregnancy had increased risk of delivering an infant weighing ≥ 4200g

- Women who were physically inactive before pregnancy increased the risk for a perineal laceration degree three or four.

7. Birth complications

- Significant predictors for induction of labor were parity, maternal age, gestational age and BMI ≥30.
- High birthweight and high BMI were overrepresented among Cesarean Sections.
- Emergency Cesarean Sections was associated with birthweight, parity, maternal age and induction of labor.
- After excluding Cesarean Sections, operative vaginal delivery was associated with parity and gender.
- Perineal laceration was associated with operative vaginal delivery and pregestational physical inactivity
- Haemorrhage was associated with high birthweight and BMI ≥30.

8. Sex differences

- Maternal BMI in early pregnancy and weight gain in pregnancy is associated with both girls and boys birth weight.
- Birth weight of girls is associated with maternal fasting plasma glucose at week30-32 and weight gain in pregnancy. No such association was significant in boys.
- Birth weight of boys is associated with paternal birth weight.

## 17 Conclusive remarks

The present work gives support to the notion that pre gestational metabolic conditions of the woman are important determinants of pregnancy and delivery complications. High body mass index and physical inactivity are according to the present work two such conditions both of which are clearly associated with energy metabolism. In particular, pre gestational physical activity has, to our knowledge, not been associated with risk of fetal macrosomia and perineal lacerations. The findings of the effect of pre gestational physical inactivity may have major impact in preventing obstetrical complications if confirmed in larger studies.

The present study also points to the upcoming issue of diverting effects of determinants of birth weight and body composition between the sexes.

It is well known that boys are heavier at birth than girls. After controlling for high birth weight and head circumference, we still found that boys are more often exposed to vacuum extraction than girls. We also found that girls and boys have different responses to maternal and parental parameters in relation to birth weight.

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# Appendix

Rikshospitalet

Kvinneklinikken

Postadresse:
0027 OSLO

Besøksadresse
Sognsvannsv.

## Til deg som er gravid og har fødeplass på Rikshospitalet

## Forespørsel om å delta i STORK-prosjektet

**(Maternelt metabolsk syndrom, store barn og svangerskapskomplikasjoner)**

Stork-prosjektet søker å finne svar på hvorfor forekomsten av store barn er økende. Fra 1990 til 1999 har andelen av barn med fødselsvekt over 4kg økt fra 16 % til vel 20 %. Dette er en uheldig utvikling. Det er kjent at store barn kan medføre økt risiko for mor og barn under fødselen.

Bakgrunnen for undersøkelsen er at vi ønsker å vite mer om hvorfor stadig flere kvinner føder store barn. Det vil gi mulighet til å identifisere kvinner som har økt risiko for å føde stort barn tidlig i svangerskapet, og utvikle støttetiltak for å motvirke denne utviklingen. Det finnes i dag ikke noe entydig svar på hvorfor det nå fødes flere store barn. Hos noen er det genetisk bestemt. Andre faktorer som også trolig påvirker fosterets vekst, er kvinnens fysiske aktivitet, type og mengde mat hun spiser og omsetningen av sukker i blodet.
Ved å delta i undersøkelsen vil du bli fulgt opp av en fast jordmor som er prosjektleder her på Kvinneklinikken.
Alle undersøkelsene er gratis, og du er forsikret på vanlig måte gjennom pasientskadeordningen.
Undersøkelsen er kun ute etter å samle informasjon og vil *ikke ha noen påvirkning på svangerskapet eller barnet ditt*Det er helt frivillig å bli med i undersøkelsen. Selv om du har begynt, kan du når som helst og uten grunn trekke deg. Dine data vil da bli slettet og ikke brukt i undersøkelsen.

## Undersøkelsene:

Hvis du velger å delta, vil det bety fire kontroller på Rikshospitalet i løpet av svangerskapet. Noen av disse vil kunne være i stedet for kontroller hos egen lege / jordmor.

Hver gang møter du fastende, det betyr at du ikke skal spise eller drikke etter klokka 24.00. Du får litt vann når du kommer.

1. gang ved ca. 14-16 ukers svangerskapsvarighet
   - Kostholdsskjema som du har fått i posten skal leveres
   - Fastende blodsukkerbelastning. Du får drikke 75 g. druesukker. Deretter tas det blodprøve fire ganger (hver ½-time) for å måle hvordan kroppen reagerer på en viss mengde sukker. Vi setter inn en veneflon (et tynt plastrør) i en blodåre på armen, slik at det bare blir ett stikk. Prøven tar litt over to timer, så det er lurt å ta med lesestoff og evt. en matpakke til å spise like etterpå. Svaret vil foreligge med en gang, og du vil få vite resultatet.

2. gang ved 22-24 uker.
   - Det blir tatt blodprøve til nedfrysing. Det tar ca. 15 minutter.
   - Ultralyd av barnet for å måle vekst og trivsel. Ultralydundersøkelsen vil også måle blodgjennomstrømningen i navlesnor og til livmoren. Dette gir oss kunnskap om fosterets trivsel.

3. gang ved 30-32 uker-
   - Sukkerbelastning som første gang, pluss
   - ultralyd som andre gang
   - nytt kostholdsskjema

4. gang ved 36-38 uker
   - Blodprøver som andre gang, pluss
   - ultralyd som tidligere
   - fysisk aktivitetsskjema

Det vil hver gang tas blodprøver som skal fryses ned. Disse prøvene vil bli analysert først etter at hele prosjektet er gjennomført.

Resultatene av undersøkelsen vil bli offentliggjort i godkjente tidsskrifter.

Studien er vurdert av datatilsynet og regional etisk komite.

Med vennlig hilsen

……..
Nanna Voldner
jordmor / stipendiat

*Hvis du ønsker å være med i prosjektet er det fint hvis du så raskt som mulig gir en tilbakemelding til Kvinneklinikken.*

*Du kan ringe til sekretær Tone Hassel 23 07 29 27 eller mobil 93 02 22 54 og si at du skal være med i STORK-prosjektet og du kan få time over telefon.*

*Du kan også sende en e-post til* tone.hassel@rikshospitalet.no
*og få time tilbake på mail.*

*Spørsmål kan rettes til Nanna Voldner tlf: 23 07 29 26, mobil 99 73 82 90*
*eller på mail.* nanna.voldner@rikshospitalet.no

Jeg har mottatt skriftlig informasjon om denne undersøkelsen og samtykker i å delta.

_____
_____
Dato Underskrift

Jordmors erklæring:

Jeg bekrefter at kvinnen har fått skriftlig og muntlig informasjon om hva det innebærer å delta i prosjektet.

_____
_____
Dato Underskrift (jordmor Nanna Voldner)

Rikshospitalet

Kvinneklinikken

Postadresse:
0027 OSLO

Besøksadresse:
Sognsvannsv. 20

Sentralbord: 23 07 00 00
Dir. linje: 23 07 26 30/40
Telefaks: 23 07 26 50

Org.nr. NO 970 897 771

Deres ref:
Vår ref:
Dato:

Takk for at du vil delta i STORK-prosjektet

Du har fått time til undersøkelse ved KK føde poliklinikk

Til deg som kommer 1. og 3. gang:
Du skal ta fastende blodukker på endokrinologisk lab. først. Der åpner de litt før halv åtte, så du kan komme så tidlig hvis du vil, men helst ikke senere enn 8:15. Prøven tar ca. to timer, og du må passe på å spise før du kommer på føde poliklinikk. Du kan godt ta med matpakke.

Til deg som kommer 2. og 4. gang:
Du skal ta en fastende blodprøve som tar kort tid, slik at du må komme på endokrinologisk lab. en liten stund før du har time. Du bør også passe på å spise før du kommer på føde poliklinikken.

Endokrinologisk lab. ligger i 1.etasje i glassgaten. Gå inn hovedinngangen, og mot venstre et lite stykke, så vil du se skilt på høyre side.

Hvis tiden ikke skulle passe, ber vi deg vennligst gi beskjed på tlf: 23 07 46 47

*Vennligst henvend deg i poliklinikkens ekspedisjon i 3. etasje på Kvinneklinikken når du kommer dit, (avsnitt E2) både før og etter konsultasjonen.*

*Velkommen*
*Vennlig hilsen for Kvinneklinikken*

Kode _____

# SPØRRESKJEMA OM GRAVIDITET OG FYSISK AKTIVITET

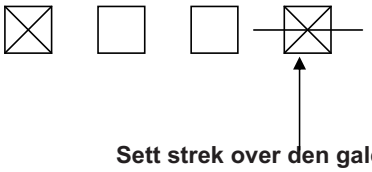
## Les dette nøye før du starter:

På de neste sidene følger noen spørsmål om fysisk aktivitet og helse. Velg den svarkategorien som passer best for deg og sett kryss. Dersom du markerer feil sett da en strek over ~~X~~, og sett et nytt kryss. Hvis det er noe du lurer på kan du spørre jordmor Nanna Voldner om hjelp.

**Marker slik:** 

**IKKE slik :**  **eller** 

**Dersom du markerer feil:**



**Sett strek over den gale markeringen**

**Før du går videre er det fint om du fyller ut dato nedenfor**

**Dato: _____/_____ 02**
*(Dag) (Måned)*

## BAKGRUNNSOPPLYSNINGER

**1.** Alder: ☐ år

**2.** Svangerskapsuke: ☐

**3.** Hvilken sivilstand har du nå?

- ☐ Gift/samboer
- ☐ Skilt/separert
- ☐ Enslig
- ☐ Enke

**4.** Hva er din høyeste fullførte utdannelse?

- ☐ Grunnskole
- ☐ Videregående yrkesfaglig
- ☐ Videregående allmennfaglig
- ☐ Høgskole/universitet inntil 4 år
- ☐ Høgskole/universitet mer enn 4 år
- ☐ Annen utdannelse

**5.** Yrke/stilling: .....

**6.** Hvor stor stillingsprosent har du?

| | **Før graviditet** | **Uke 1-12 (1. trimester)** | **Uke 13- 27 (2. trimester)** | **Uke 28-40 (3.trimester)** |
|---|---|---|---|---|
| 100% | | | | |
| Mer enn 50% | | | | |
| Mindre enn 50% | | | | |
| Arbeidsledig | | | | |
| Sykemeldt | | | | |
| Jobber ikke (f.eks. student) | | | | |

**7.** Hvilken arbeidstid har du på nåværende tidspunkt?

- ☐ Fast dagtid
- ☐ Fast ettermiddag/kveld
- ☐ Fast nattarbeid
- ☐ Skiftarbeid eller turnusordning
- ☐ Ingen fast ordning *( ekstrahjelp, vikar o.l.)*
- ☐ Jobber ikke *(arbeidsledig, sykemeldt, student o.l)*

## HELSE OG LIVSSTIL

**8.** Høyde: ☐ m

**9.** Vekt før graviditet: ☐ kg

**10.** Er du tilfreds med vektøkningen du har hatt så langt?

☐ JA ☐ NEI ☐ Vet ikke

**11.** Hvor mange kg har du lagt på deg?

- ☐ -5-0 kg
- ☐ 0-5 kg
- ☐ 6-10
- ☐ 11-15 kg
- ☐ 16-20 kg
- ☐ 20-25 kg
- ☐ Mer enn 25 kg

**12.** Hvordan vil du karakterisere kostvanene dine?

| | **Svært bra** | **Bra** | **Middels** | **Dårlig** | **Svært dårlig** |
|---|---|---|---|---|---|
| Før graviditet | | | | | |
| I dag | | | | | |

**13 a)** Røyker du daglig?

☐ JA ☐ NEI

**b)** Hvis JA bes du svare så nøyaktig som mulig på antall sigaretter

..... pr. dag

**c)** Hvis NEI, har du røykt tidligere?

☐ JA ☐ NEI

**d)** Er du utsatt for passiv røyking hjemme eller på arbeid?

☐ JA ☐ NEI

**14.** Hvor ofte drikker du alkohol?

| | **Før graviditet** | **Uke 1-12 (1. trimester)** | **Uke 13- 27 (2. trimester)** | **Uke 28-40 (3. trimester)** |
|---|---|---|---|---|
| Sjelden eller aldri | | | | |
| Mindre enn 1 gang per måned | | | | |
| 1-3 ganger per måned | | | | |
| 1 gang i uka | | | | |
| Flere dager i uken | | | | |
| Hver dag | | | | |

## HELSEPLAGER

**15 a)** Har du problemer urin-lekkasje?

☐ JA ☐ NEI

**b)** Hvis JA, når skjer dette?

| | Før graviditet | Uke 1-12 (1. trimester) | Uke 13- 27 (2. trimester) | Uke 28-40 (3. trimester) |
|---|---|---|---|---|
| Når jeg er fysisk aktiv | | | | |
| Når jeg hoster og/eller nyser | | | | |
| Når jeg ler | | | | |
| Ved sterk vannlatingstrang | | | | |

**16 a)** Har du problemer med å holde på luft eller avføring?

☐ JA ☐ NEI

**b)** Hvis JA, når skjer dette?

| | Før graviditet | Uke 1-12 (1. trimester) | Uke 13- 27 (2. trimester) | Uke 28-40 (3. trimester) |
|---|---|---|---|---|
| Når jeg er fysisk aktiv | | | | |
| Når jeg hoster og/eller nyser | | | | |
| Når jeg ler | | | | |
| Når jeg må veldig på do | | | | |

**17 a)** Hvor ofte har du avføring?

| | Før graviditet | Uke 1-12 (1. trimester) | Uke 13- 27 (2. trimester) | Uke 28-40 (3. trimester) |
|---|---|---|---|---|
| Mindre enn 2 ganger per uke | | | | |
| Annenhver dag | | | | |
| Hver dag<br>Flere ganger per dag | | | | |

**b)** Må du ”trykke” for å få ut avføring?

☐ Sjelden eller aldri ☐ Ofte

☐ Av og til ☐ Alltid

**18 a)** Har du i løpet av dette svangerskapet vært plaget med smerter i bekkenområdet (bekkenløsning)?

☐ JA ☐ NEI

**b)** Hvis JA, har du hatt så store vansker med å gå at du må bruke stokk eller krykker?

| | Før graviditet | Uke 1-12 (1. trimester) | Uke 13- 27 (2. trimester) | Uke 28-40 (3. trimester) |
|---|---|---|---|---|
| Ikke i det hele tatt | | | | |
| Ikke så ofte | | | | |
| I perioder | | | | |
| Mesteparten av tiden | | | | |

**19 a)** Har du i tidligere svangerskap vært plaget med smerter i bekkenområdet (bekkenløsning)?

☐ JA ☐ NEI

**b)** Hvis JA, når sluttet plagene?

☐ Mindre 6 uker etter fødselen ☐ 5-10 måneder etter fødsel
☐ 6-20 uker etter fødselen ☐ Har fortsatt vedvarende plager

## JOBBAKTIVITETER

**Dersom du i dag ikke har jobb eller betalt arbeid utenfor hjemmet, vennligst gå videre til spørsmål nr. 26 a)**

**20.** Arbeider du stående og/eller gående?

☐ Sjelden eller aldri ☐ JA, mindre enn 50% av tiden
☐ Av og til, men ikke daglig ☐ JA, mer enn 50% av tiden

**21.** Arbeider du med armene løftet i skulderhøyde eller høyere?

☐ Av og til, men ikke daglig ☐ JA, mer enn 50% av tiden

**22.** Må du vri eller bøye deg mange ganger i løpet av en arbeidsdag?

☐ Sjelden eller aldri ☐ JA, mindre enn 50% av tiden
☐ Av og til, men ikke daglig ☐ JA, mer enn 50% av tiden

**23.** Hvor ofte opplever du belastende løft på arbeidsplassen?

☐ Sjelden eller aldri ☐ 10-20 ganger daglig
☐ Mindre enn 20 ganger ukentlig ☐ Mer enn 20 ganger daglig
☐ Mer enn 20 ganger ukentlig

**24**. Vil du karakterisere jobben din som fysisk krevende?

☐ JA, spesifiser .....

☐ Av og til, spesifiser .....

☐ NEI, spesifiser .....

## TRANSPORTAKTIVITETER

**25 a)** Hvordan kommer du deg vanligvis til jobb nå som du er gravid?

(*Sett gjerne flere kryss dersom mer enn et av alternativene passer)*

☐ Kjører bil ☐ Går

☐ Offentlig kommunikasjon ☐ Annet, spesifiser .....

☐ Sykler

**b)** Hvor lang tid bruker du til og fra hjem og arbeidssted *(en vei)*?

☐ Mindre enn 5 min ☐ 30-60 min

☐ 5-15 min ☐ Mer enn 60 min

☐ 15-30 min ☐ Annet, spesifiser .....

**26 a)** Har du barn du skal bringe/hente?

☐ JA, daglig ☐ JA, av og til

☐ JA, annenhver dag ☐ NEI

**b)** Hvis JA, hvordan bringer/henter du vanligvis barna nå som du er gravid?
(*Sett gjerne flere kryss dersom mer enn et av alternativene passer)*

☐ Kjører bil ☐ Går

☐ Offentlig kommunikasjon ☐ Annet, spesifiser .....

☐ Sykler

**27 a)** Kan du angi hvor mye du totalt **går** (bruker bena) i løpet av en dag (utenom arbeidstid)?

*(F.eks. til og fra arbeid, hente/bringe barn, til og fra butikken, osv.)*

☐ Mindre enn 5 min ☐ 30-60 min

☐ 5-15 min ☐ Mer enn 60 min

☐ 15-30 min ☐ Går sjelden eller aldri

**b)** Er dette mindre tid enn du normalt ville brukt bena (gått) dersom du ikke var gravid?

☐ JA ☐ NEI

**28 a)** Kan du angi hvor mye du totalt **sykler** i løpet av en dag?

*(F.eks. til og fra arbeid, hente/bringe barn, til og fra butikken, osv.)*

☐ Mindre enn 5 min ☐ 30-60 min

☐ 5-15 min ☐ Mer enn 60 min

☐ 15-30 min ☐ Sykler sjelden eller aldri

**b)** Er dette mindre tid enn du normalt ville brukt dersom du ikke var gravid?

☐ JA ☐ NEI

**29 a)** Bruker du trapper fremfor heis/rulletrapp?

☐ JA ☐ Av og til ☐ NEI

**b)** Ville du brukt mer trapper dersom du ikke var gravid?

☐ JA ☐ NEI

## AKTIVITET I HJEM OG NÆRMILJØ

**30 a)** Har du barn fra før?

☐ JA ☐ NEI

**b)** Hvis JA, hvor mange barn under 18 år har du omsorg for?

☐ 1 ☐ 2 ☐ 3 ☐ 4 eller flere

**31 a)** Har du hage/gårdsplass?

☐ JA ☐ NEI

**b)** Hvis JA, hvor ofte i en vanlig uke gjør du **tungt fysisk** hagearbeid eller tilsvarende?

*(F.eks. snømåking, klippe plenen, løfte tunge steiner, hugge ved, gravearbeid, oppussingsarbeid)*

| | **Før graviditet** | **Uke 1-12 (1. trimester)** | **Uke 13- 27 (2. trimester)** | **Uke 28-40 (3.trimester)** |
|---|---|---|---|---|
| Aldri | | | | |
| Mindre enn 1 gang i uka | | | | |
| 1-3 ganger i uka | | | | |
| 3-5 ganger i uka | | | | |
| Hver dag | | | | |
| Mer enn 1 gang per dag | | | | |

**c)** Hvis JA, hvor ofte i en vanlig uke gjør du **lett til middels anstrengende** hagearbeid eller tilsvarende?
*(F.eks. bære lette ting, rydde, vedlikeholdsarbeid, luke i blomsterbed, koste og rake)*

| | Før graviditet | Uke 1-12 (1. trimester) | Uke 13- 27 (2. trimester) | Uke 28-40 (3.trimester) |
|---|---|---|---|---|
| Aldri | | | | |
| Mindre enn 1 gang i uka | | | | |
| 1-3 ganger i uka | | | | |
| 3-5 ganger i uka | | | | |
| Hver dag | | | | |
| Mer enn 1 gang per dag | | | | |

**32.** Hvor ofte i en vanlig uke gjør du med **lett til middels anstrengende** arbeid i hjemmet?
*(F.eks. støvsuge, vaske gulv, trappevask, innkjøp av mat, pleie og omsorgsoppgaver)*

| | Før graviditet | Uke 1-12 (1. trimester) | Uke 13- 27 (2. trimester) | Uke 28-40 (3.trimester) |
|---|---|---|---|---|
| Aldri | | | | |
| Mindre enn 1 gang i uka | | | | |
| 1-3 ganger i uka | | | | |
| 3-5 ganger i uka | | | | |
| Hver dag | | | | |
| Mer enn 1 gang per dag | | | | |

**33.** Hvor fysisk anstrengende er dine daglige omsorgsoppgaver og gjøremål i og rundt hjemmet?

☐ Veldig lett
☐ Lett
☐ Litt anstrengende
☐ Anstrengende
☐ Svært anstrengende

## FRITIDSAKTIVITETER; SPORT OG REKREASJON

**MERK:** *Fysisk aktivitet* defineres som 1 eller flere treningsaktiviteter per uke med minst 20 minutters varighet per gang

**34.** Var du regelmessig fysisk aktiv før graviditet?
*(1 eller flere mosjonsaktiviteter per uke med minst 20 minutters varighet per gang)*

☐ JA ☐ NEI

**35.** Er du som gravid regelmessig fysisk aktiv?

*(1 eller flere mosjonsaktiviteter per uke med minst 20 minutters varighet per gang)*

| | JA | NEI |
|---|---|---|
| 1-12 svangerskapsuke (1. trimester) | | |
| 13-27 svangerskapsuke (2. trimester) | | |
| 28-40 svangerskapsuke (3. trimester) | | |

**Dersom du har svart NEI på både spørsmål 34 og 35, vennligst gå videre til spørsmål nr. 43**

**36.** Hvor lenge har du drevet med regelmessig fysisk aktivitet før nåværende svangerskap?

*(1 eller flere mosjonsaktiviteter per uke med minst 20 minutters varighet per gang)*

☐ Mindre enn 6 måneder
☐ 6 mnd -1 år
☐ 1-4 år
☐ 5-10 år
☐ Mer enn 10 år

**37.** Har du opprettholdt samme fysisk aktivitetsnivå som før graviditet?

| | Mer aktiv før graviditet | Like aktiv som før | Mindre aktiv nå |
|---|---|---|---|
| 1-12 svangerskapsuke (1. trimester) | | | |
| 13-27 svangerskapsuke (2. trimester) | | | |
| 28-40 svangerskapsuke (3. trimester) | | | |

**38.** Hva slags type fysisk aktivitet driver du vanligvis? (*Sett maks tre kryss*)

| | Før graviditet | Uke 1-12 (1. trimester) | Uke 13- 27 (2. trimester) | Uke 28-40 (3. trimester) |
|---|---|---|---|---|
| Går tur | | | | |
| Jogger / løper | | | | |
| Svømmer | | | | |
| Sykler | | | | |
| Styrke / vekttrening | | | | |
| Ballsport | | | | |
| Langrenn / rulleski | | | | |
| Skøyter / rollerblades | | | | |
| Kampsport | | | | |
| Aerobic | | | | |
| Aerobic for gravide | | | | |
| Bevegelighetstrening / avspenning | | | | |
| Dans | | | | |
| Annet | | | | |

**39.** Hvor ofte driver du med fysisk aktivitet?

| | Før graviditet | Uke 1-12 (1. trimester) | Uke 13- 27 (2. trimester) | Uke 28-40 (3. trimester) |
|---|---|---|---|---|
| 1 gang i uka | | | | |
| 2-3 ganger i uka | | | | |
| 4-5 ganger i uka | | | | |
| 5-6 ganger i uka | | | | |
| Hver dag | | | | |
| Mer enn 1.gang per dag | | | | |

**40.** Hvor lang tid bruker du i gjennomsnitt når du trener?

*(Ikke medregnet tid til skift, dusj, reisevei osv.)*

| | Før graviditet | Uke 1-12 (1. trimester) | Uke 13- 27 (2. trimester) | Uke 28-40 (3. trimester) |
|---|---|---|---|---|
| Mindre enn 30 min | | | | |
| 30-60 min | | | | |
| 1-2 timer | | | | |
| Over 2 timer | | | | |

**41.** På hvilken intensitet trener du vanligvis?

| | Før graviditet | Uke 1-12 (1. trimester) | Uke 13- 27 (2. trimester) | Uke 28-40 (3. trimester) |
|---|---|---|---|---|
| Uten å bli svett eller andpusten *(oppleves lite anstrengende)* | | | | |
| Blir svett og lett andpusten *(oppleves anstrengende )* | | | | |
| Blir veldig svett og puster tungt *(oppleves svært anstrengende)* | | | | |

**42 a)** Gjør du 1 gang i uken eller mer styrkeøvelser på egenhånd hjemme?

☐ JA ☐ NEI

**b)** Hvis JA, gjør du øvelser for disse musklene?

| | Magemusklene | Ryggmusklene | Bekkenbunns-musklene |
|---|---|---|---|
| Før graviditet | | | |
| 1-12 svangerskapsuke (1. trimester) | | | |
| 13-27 svangerskapsuke (2. trimester) | | | |
| 28-40 svangerskapsuke (3. trimester) | | | |

## STØTTE, BARRIERER OG MOTIVASJON

**43.** Var det noen i din nære familie (mor, far eller søsken) som drev regelmessig fysisk aktivitet under din oppvekst (før du fylte 18 år)?

☐ JA ☐ NEI

**44.** Hvor vanlig er det å drive fysisk aktivitet i din nærmeste omgangskrets?

☐ Ikke vanlig ☐ Forekommer ☐ Svært vanlig

**45.** Hvilket av disse alternativene passer best for deg?

☐ Jeg trener ikke, og jeg har ikke tenkt til å begynne
☐ Jeg trener ikke, men det er mulig jeg begynner
☐ Jeg trener noen ganger, men ikke regelmessig
☐ Jeg trener regelmessig, men har akkurat startet
☐ Jeg har trent regelmessig mer enn 6 måneder

**46 a)** Trener du sammen med noen?

☐ Aldri ☐ Av og til ☐ Alltid ☐ Trener ikke

**b)** Hvis du har svart alltid eller av og til, hvem trener du vanligvis med?
(*Sett gjerne flere kryss dersom mer enn et av alternativene passer)*

☐ Familie/ektefelle/partner ☐ Idrettsklubb
☐ Venner ☐ Helsestudio/aerobic (mennesker jeg møter der)
☐ Arbeidskollegaer ☐ Hund

**47.** Dersom du i dag **ikke er** regelmessig fysisk aktiv, hva er de to viktigste grunnene til dette?
(*Sett maks to kryss*)

☐ Har ikke tid
☐ Er ikke interessert
☐ Får nok mosjon gjennom min jobb og/eller i hjemmet
☐ Det krever for mye å komme i gang
☐ Passer ikke med barn/omsorg
☐ Har ingen å trene sammen med
☐ Vanskelig å kombinere med arbeid/utdanning
☐ Dårlige treningsmuligheter
☐ Negative opplevelser i forbindelse med fysisk aktivitet
☐ Svangerskapskomplikasjoner
☐ Har aldri trent, ingen erfaring
☐ Sykdom/handikap
☐ Frykt/redsel for mitt ufødte barn
☐ Helsepersonell råder meg til ikke å være fysisk aktiv

**48**. Dersom du i dag **er** regelmessig fysisk aktiv, hva er de to viktigste grunnene til dette?
(*Sett maks to kryss*)

- ☐ Det er gøy/opplevelse
- ☐ Gir bedre utseende/kropp
- ☐ Avreagere/avkobling
- ☐ Trener til større eller mindre konkurranser
- ☐ Gir bedre fysisk form/forebygger helseplager
- ☐ Gir psykisk overskudd/velvære/glede
- ☐ Holde vekta nede (slik at jeg ikke legger for mye på meg under graviditeten)
- ☐ Øker selvtilliten/selvfølelsen
- ☐ Reduserer svangerskapsplager
- ☐ Motvirker angst og depresjon
- ☐ Fordi jeg føler at jeg bør
- ☐ Det er sosialt

**49.** Bekymrer du deg for barnet inne i magen når du driver med fysisk aktivitet?

☐ JA ☐ Av og til ☐ NEI ☐ Trener ikke

**50 a)** Har lege/jordmor gitt deg råd om hvordan drive fysisk aktivitet i svangerskapet?

☐ JA ☐ NEI

**b)** Hvis JA, hvilke råd fikk du, vennligst spesifiser nærmere?

……

.....

.....

.....

## ROLIGE AKTIVITETER

**51.** Hvor mange timer ser du på TV?

| | **Hverdag** | **Helg/fridag** |
|---|---|---|
| Mindre enn 1 time | | |
| 1-2 timer | | |
| 2-3 timer | | |
| 3-4 timer | | |
| 4-5 timer | | |
| Mer enn 5 timer | | |

**52.** Hvor lang tid bruker du på å lese bøker/aviser/blader, løse kryssord eller lignende?

| | **Hverdag** | **Helg/fridag** |
|---|---|---|
| Mindre enn 1 time | | |
| 1-2 timer | | |
| 2-3 timer | | |
| 3-4 timer | | |
| 4-5 timer | | |
| Mer enn 5 timer | | |

**53 a)** Hvor mange timer sover du vanligvis i løpet av et døgn?

| | **Hverdag** | **Helg/fridag** |
|---|---|---|
| Mindre enn 4 timer | | |
| 4-6 timer | | |
| 6-8 timer | | |
| 8-10 timer | | |
| 10-12 timer | | |
| Mer enn 12 timer | | |

**b)** Er dette mer tid enn du normalt ville sovet dersom du ikke var gravid?

☐ JA ☐ NEI

**TUSEN TAKK FOR HJELPEN**

# HVA SPISER DU?

I dette skjemaet spør vi om dine spisevaner slik de er nå.
Vi er klar over at kostholdet varierer fra dag til dag. Prøv derfor så godt du kan å gi et "gjennomsnitt" av dine spisevaner. Der du er usikker, anslå svaret.

Skjemaet skal leses av en maskin, og det er derfor viktig at du setter et tydelig kryss i avmerket rute.

Riktig markering er slik: ☒

Bruk helst bløt blyant. Feil kan da rettes med viskelær.
Kulepenn og svart tusjpenn kan også brukes.

Av hensyn til den maskinelle lesingen pass på at arkene ikke blir brettet.

*Alle svar vil bli behandlet strengt fortrolig.*

Rikshospitalet
Universitetsklinikk

EKSEMPEL PÅ UTFYLLING AV SPØRSMÅL 1.
Kari Nordmann spiser daglig 5 skiver brød og ett knekkebrød. Hun spiser vanligvis kneippbrød, men i helgene blir det en del loff. I tillegg spiser hun et knekkebrød hver dag. Hun fyller ut første spørsmål slik:

## 1. HVOR MYE BRØD PLEIER DU Å SPISE?

Legg sammen det du bruker til alle måltider i løpet av en dag.
(1/2 rundstykke = 1 skive, 1 baguett = 5 skiver)

| | Antall skiver pr. dag | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 1/2 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12+ |
| **Fint brød**<br>(loff, baguetter, fine rundstykker o.l.) | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **Mellomgrovt brød**<br>(lys helkorn, lys kneipp, lys hj.bakt o.l.) | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **Grovt brød**<br>(fiberkneipp, mørk kneipp, mørkt hi. bakt o.l.) | ☐ | ☐ | ☐ | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **Knekkebrød**<br>(kavring, grov skonrok o.l.) | ☒ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

Sum skiver pr. dag = 5
Antall skiver pr. uke: 5 x 7 = 35. Tallet brukes i spørsmål 5.

# 1. HVOR MYE BRØD PLEIER DU Å SPISE?

Legg sammen det du bruker til alle måltider i løpet av en dag.
(1/2 rundstykke = 1 skive, 1 baguett = 5 skiver, 1 ciabatta = 4 skiver)

| | Antall skiver pr. dag | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 1/2 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12+ |
| Fint brød (loff, baguetter, fine rundstykker o.l.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Mellomgrovt brød (lys helkorn, lys kneipp, lys hj.bakt o.l.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Grovt brød (fiberkneipp, mørk kneipp, mørkt hj. bakt o.l.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Knekkebrød (kavring, grov skonrok o.l.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

Sum skiver pr. dag = ___
Antall skiver pr. uke: ___ x 7 = ___. Tallet brukes i spørsmål 5.

# 2. HVA PLEIER DU Å SMØRE PÅ BRØDET?

Merk av både for hverdag og helg, selv om du bruker det samme.

| Hverdager | | Lørdager, søndager |
|---|---|---|
| ☐ | Bruker ikke | ☐ |
| ☐ | Smør (meierismør) | ☐ |
| ☐ | Bremykt | ☐ |
| ☐ | Brelett | ☐ |
| ☐ | Soft-, soyamargarin (pakke, beger) | ☐ |
| ☐ | Solsikke | ☐ |
| ☐ | Oliven | ☐ |
| ☐ | Vita | ☐ |
| ☐ | Olivero | ☐ |
| ☐ | Omega | ☐ |
| ☐ | Soft light | ☐ |
| ☐ | Vita lett | ☐ |
| ☐ | Annen margarin | ☐ |

# 3. OM DU BRUKER FETT PÅ BRØD, HVOR MYE BRUKER DU?

En porsjonspakning på 12 g rekker til antall skiver

1 ☐
2 ☐
3 ☐
4 ☐
5 ☐

# 4. MELK SOM DRIKK

(1 glass = 1,5 dl)

| | Drikker sjelden/ ikke | Antall glass pr. dag: 1/2 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8+ |
|---|---|---|---|---|---|---|---|---|---|---|
| Helmelk, søt, sur | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Lettmelk, søt, sur | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Lettmelk, ekstra lett | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Skummet melk, søt, sur | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

## 5.PÅLEGGSSORTER

Bruk sum skiver pr. uke fra spørsmål 1.

Til antall skiver pr. uke

| | 0 | 1/2 | 1 | 2-3 | 4-5 | 6-7 | 8-14 | 15-21 | 22-28 | 29-35 | 36+ |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Brun ost, prim | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Hvit ost, helfet, 27% fett (Jarlsberg, Norvegia o.l., smøreost; eske, tube) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Hvit ost, halvfet, 16% fett (Jarlsberg, Norvegia o.l. smøreost; eske, tube) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Ost med mer enn 27% fett (kremoster, Normanna, Ridderost) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

| | 0 | 1/2 | 1 | 2-3 | 4-5 | 6-7 | 8-14 | 15-21 | 22-28 | 29-35 | 36+ |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Leverpostei, vanlig | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Leverpostei, mager | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Servelat, vanlig | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Lett servelat, kalverull, kokt skinke, okserull o.l. | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Salt pølse, spekepølse (fårepølse, salami o.l.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

| | 0 | 1/2 | 1 | 2-3 | 4-5 | 6-7 | 8-14 | 15-21 | 22-28 | 29-35 | 36+ |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Kaviar | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Makrell i tomat, røkt makrell | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Sardiner, sursild, ansjos o.l. | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Laks, ørret | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Reker, krabbe | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

| | 0 | 1/2 | 1 | 2-3 | 4-5 | 6-7 | 8-14 | 15-21 | 22-28 | 29-35 | 36+ |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Syltetøy, marmelade, frysetøy | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Honning, sirup, sjokolade-, nøttepålegg | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

| | 0 | 1/2 | 1 | 2-3 | 4-5 | 6-7 | 8-14 | 15-21 | 22-28 | 29-35 | 36+ |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Grønnsaker som pålegg (agurk, tomat o.l.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Frukt som pålegg (banan, eple o.l.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Salater med majones | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Majones på smørbrød | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

## 6. EGG

Antall pr. uke

| | 0 | Mindre enn 1 | 1 | 2 | 3-4 | 5-6 | 7 | 8+ |
|---|---|---|---|---|---|---|---|---|
| (kokt, stekt, eggerøre, omelett) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

## 7. FROKOSTGRYN, GRØT OG YOGHURT

Svar enten pr. måned eller pr. uke. <1 betyr sjeldnere enn 1 gang.

| | Gang pr. måned | | | | | Gang pr. uke | | | | | | Mengde pr. gang | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | <1 | 1 | 2 | 3 | 1 | 2-3 | 4-5 | 6-7 | 8+ | | 1 | 11/2 | 2 | 3+ |
| Havregryn,kornblandinger (4-korn, usøtet müsli o.l.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | ☐ | ☐ | ☐ | ☐ |
| Cornflakes, puffet ris, havrenøtter o.l. | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1 ☐ | 11/2 ☐ | 2 ☐ | 3+ ☐ |
| Havregrøt | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7 ☐ |
| Sukker til frokostgryn, grøt | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (ts) | 1 ☐ | 2 ☐ | 3-4 ☐ | 5+ ☐ |
| Yoghurt, naturell, frukt | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (beger) | 1/2 ☐ | 1 ☐ | 11/2 ☐ | 2+ ☐ |
| Lettyoghurt | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (beger) | 1/2 ☐ | 1 ☐ | 11/2 ☐ | 2+ ☐ |
| Go'morgen yoghurt, inkl. müsli | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (beger) | 1/2 ☐ | 1 ☐ | 11/2 ☐ | 2+ ☐ |
| Melk søt, sur på gryn, grøt og dessert | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 3/4 ☐ | 1 ☐ | 2 ☐ | 3+ ☐ |

## 8. KAFFE OG TE

(1 kopp kaffe = 1,2 dl  1 kopp te = 2 dl)

| | Drikker ikke/ikke daglig | Antall kopper pr. dag 1/2 | 1 | 2 | 3-4 | 5-6 | 7-8 | 9-10 | 11+ |
|---|---|---|---|---|---|---|---|---|---|
| Kaffe, kokt | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Kaffe, traktet, filter | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Kaffe, pulver (instant) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Kaffe, koffeinfri | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Te | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Nypete, urtete | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

| | Antall teskjeer eller biter pr. kopp 0 | 1/2 | 1 | 2 | 3 | 4+ |
|---|---|---|---|---|---|---|
| Sukker til kaffe | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Sukker til te | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Kunstig søtstoff til kaffe eller te | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Fløte til kaffe | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

## 9. ANDRE DRIKKER

Svar enten pr. måned eller pr. uke. < 1 betyr sjeldnere enn 1 gang. Merk at porsjonsenhetene er forskjellige. 1/3 liter tilsvarer en halvflaske øl og 2/3 liter tilsvarer en helflaske.

| | Gang pr. måned | | | | | Gang pr. uke | | | | | Mengde pr. gang | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | <1 | 1 | 2 | 3 | 1 | 2-3 | 4-5 | 6-7 | 8+ | | 1/2 | 1 | 2 | 3 | 4 | 5+ |
| Vann | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (glass) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Appelsinjuice | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (glass) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Annen juice, most, nektar | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (glass) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Saft, solbærsirup m. sukker | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (glass) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Saft, kunstig søtet | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (glass) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Brus, Cola, Solo o.l. med sukker | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (liter) | 1/4 ☐ | 1/3 ☐ | 1/2 ☐ | 2/3 ☐ | 1 ☐ | 11/2+ ☐ |
| Brus, Cola, Solo o.l. kunstig søtet | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (liter) | 1/4 ☐ | 1/3 ☐ | 1/2 ☐ | 2/3 ☐ | 1 ☐ | 11/2+ ☐ |
| Farris, Selters, Soda o.l. | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (liter) | 1/4 ☐ | 1/3 ☐ | 1/2 ☐ | 2/3 ☐ | 1 ☐ | 11/2+ ☐ |
| Alkoholfritt øl, vørterøl, lettøl | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (liter) | 1/4 ☐ | 1/3 ☐ | 1/2 ☐ | 2/3 ☐ | 1 ☐ | 11/2+ ☐ |
| Pilsnerøl | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (liter) | 1/4 ☐ | 1/3 ☐ | 1/2 ☐ | 2/3 ☐ | 1 ☐ | 11/2+ ☐ |
| Vin | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (glass) | 1 ☐ | 2 ☐ | 3 ☐ | 4 ☐ | 5 ☐ | 6+ ☐ |
| Brennevin, likør | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (1 dram =4cl) | 1 ☐ | 2 ☐ | 3 ☐ | 4 ☐ | 5 ☐ | 6+ ☐ |

## 10. MIDDAGSRETTER

Vi spør både om middagsmåltidene og det du spiser til andre måltider. Tell til slutt sammen antall retter du har merket av for å se om summen virker sannsynlig. En "dl" tilsvarer omtrent mengden i en suppeøse. Med "ss" menes en spiseskje.

| | Gang pr. måned | | | | | | | | | | Mengde pr. gang | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | <1 | 1 | 2 | 3 | 4 | 5-6 | 7-8 | 9+ | | 1/2 | 2/3 | 1 | 11/2 | 2+ |
| Kjøttpølse, medisterpølse | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (kjøttpølse) | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ |
| Hamburger, karbonader o.l. | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ |
| Grill- og wienerpølse | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (pølse) | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ |
| Hamburger-, pølsebrød, lomper | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ |
| Kjøttkaker, medisterkaker, kjøttpudding | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ |
| Kjøttdeigretter (saus eller gryte med kjøttdeig, lasagne o.l.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ |
| Taco (med kjøtt og salat) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ |
| Pastaretter | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | ☐ | ☐ | ☐ | ☐ | ☐ |

| | Gang pr. måned | | | | | | | | | | Mengde pr. gang | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | <1 | 1 | 2 | 3 | 4 | 5-6 | 7-8 | 9+ | | 1/8 | 1/4 | 1/2 | 3/4 | 1+ | |
| Pizza (500-600 g) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (pizza) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1/2 | 1 | 11/2 | 2 | 21/2+ | |
| Biff (alle typer kjøtt) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1/2 | 1 | 11/2 | 2 | 21/2+ | |
| Koteletter (lam, okse, svin) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1-2 | 3-4 | 5-6 | 7-8 | 9+ | |
| Stek (lam, okse, svin) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (skive) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1-2 | 3-4 | 5-6 | 7-8 | 9+ | |
| Stek (elg, hjort, reinsdyr o.l.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (skive) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| Gryterett med helt kjøtt, frikassè, fårikål o.l. | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ | |
| Lapskaus, suppelapskaus, betasuppe | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ | |
| | | | | | | | | | | | 1-2 | 3-4 | 5-6 | 7-8 | 9+ | |
| Bacon, stekt flesk | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (skive) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1/4 | 1/3 | 1/2 | 3/4 | 1+ | |
| Kylling, høne | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1-2 | 3-4 | 5-6 | 7-8 | 9+ | |
| Leverretter | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (skive) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | 0 | <1 | 1 | 2 | 3 | 4 | 5-6 | 7-8 | 9+ | | 1 | 2 | 3 | 4 | 5+ | |
| Fiskekaker, fiskepudding, fiskeboller | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (kake) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1-2 | 3-4 | 5-6 | 7-9 | 10+ | |
| Fiskepinner | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ | |
| Torsk, sei, hyse (kokt) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ | |
| Torsk, sei, hyse (stekt, panert) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ | |
| Sild (fersk, speket, røkt) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (filet) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1/2 | 1 | 11/2 | 2 | 3+ | |
| Makrell (fersk, røkt) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (filet) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ | |
| Laks, ørret (sjø, oppdrett) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (skive) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| Fiskegryte, -grateng, suppe med fisk | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ | |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ | |
| Reker, krabbe | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl, renset) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | 0 | <1 | 1 | 2 | 3 | 4 | 5-6 | 7-8 | 9+ | | | | | | | |
| Risgrøt, annen melkegrøt | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ | |
| | | | | | | | | | | | 1-2 | 3-4 | 5-6 | 7-8 | 9+ | |
| Pannekaker | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| Suppe (tomat, blomkål, ertesuppe o.l.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ | |
| Vegetarrett, vegetarpizza, grønnsaksgrateng, -pai | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (bit/dl) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ | |
| | 0 | <1 | 1 | 2 | 3 | 4 | 5-6 | 7-8 | 9+ | | 1/2 | 1 | 11/2 | 2 | 21/2+ | |
| Brun/hvit saus | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| Smeltet margarin, smør til fisk | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (ss) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ | |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ | |
| Bearnaisesaus o.l. | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (ss) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ | |
| Majones, remulade | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (ss) | ☐ | ☐ | ☐ | ☐ | ☐ | |
| | | | | | | | | | | | 1 | 2 | 3 | 4 | 5+ | |
| Ketchup | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (ss) | ☐ | ☐ | ☐ | ☐ | ☐ | |

## 11. POTETER, RIS, SPAGHETTI, GRØNNSAKER

Svar enten pr. måned eller pr. uke. < 1 betyr sjeldnere enn 1 gang.
Disse spørsmålene dreier seg først og fremst om tilbehør til middagsretter, men spiser du for eksempel en rå gulrot eller salat til lunsj, skal det tas med her.

| | Gang pr. måned | | | | | Gang pr. uke | | | | | | Mengde pr. gang | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | <1 | 1 | 2 | 3 | 1 | 2-3 | 4-5 | 6-7 | 8+ | | 1 | 2 | 3 | 4 | 5+ |
| Poteter, kokte | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ | ☐ |
| Pommes frites, stekte poteter | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1 ☐ | 2 ☐ | 3 ☐ | 4 ☐ | 5+ ☐ |
| Potetmos, -stuing, gratinerte poteter | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1 ☐ | 2 ☐ | 3 ☐ | 4 ☐ | 5+ ☐ |
| Ris | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ |
| Spaghetti, makaroni, pasta | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ |
| Gulrot | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | 1/2 ☐ | 1 ☐ | 11/2 ☐ | 2 ☐ | 3+ ☐ |
| Hodekål | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (skalk) | 1 ☐ | 2 ☐ | 3 ☐ | 4 ☐ | 5+ ☐ |
| Kålrot | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (skive) | 1 ☐ | 2 ☐ | 3 ☐ | 4 ☐ | 5+ ☐ |
| Blomkål | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (bukett) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ |
| Brokkoli | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (bukett) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ |
| Rosenkål | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ |
| Grønnkål | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1 ☐ | 2 ☐ | 3 ☐ | 4 ☐ | 5+ ☐ |
| Løk | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (ss) | 1 ☐ | 2 ☐ | 3 ☐ | 4 ☐ | 5+ ☐ |
| Spinat, andre bladgrønns. | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1 ☐ | 2 ☐ | 3 ☐ | 4 ☐ | 5+ ☐ |
| Sopp | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ |
| Avocado | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | 1/4 ☐ | 1/2 ☐ | 3/4 ☐ | 1 ☐ | 11/4+ ☐ |
| Paprika | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (strimmel) | 1 ☐ | 2 ☐ | 3 ☐ | 4 ☐ | 5+ ☐ |
| Tomat | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | 1/2 ☐ | 1 ☐ | 11/2 ☐ | 2 ☐ | 3+ ☐ |
| Tomatbønner, bønner/linser | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1 ☐ | 2 ☐ | 3 ☐ | 4 ☐ | 5+ ☐ |
| Mais | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (ss) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7-8 ☐ | 9+ ☐ |
| Erter, frosne grønnsak-blandinger | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1 ☐ | 2 ☐ | 3 ☐ | 4 ☐ | 5+ ☐ |
| Salatblandinger | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1 ☐ | 2 ☐ | 3 ☐ | 4 ☐ | 5+ ☐ |
| Dressing | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (ss) | 1/2 ☐ | 1 ☐ | 2 ☐ | 3 ☐ | 4+ ☐ |
| Rømme | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (ss) | 1/2 ☐ | 1 ☐ | 2 ☐ | 3 ☐ | 4+ ☐ |

Hvor mange ganger om dagen spiser du vanligvis grønnsaker utenom grønnsakene du spiser til middag?

0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5+ ☐

## 12. TYPE FETT TIL MATLAGING

| Smør/margarin | Oljer |
|---|---|
| ☐ Smør (meierismør) | ☐ Olivenolje |
| ☐ Bremykt | ☐ Soyaolje |
| ☐ Melange, Per | ☐ Maisolje |
| ☐ Soft-, soyamargarin (pakke, beger) | ☐ Solsikkeolje |
| ☐ Solsikke | ☐ Valnøttolje |
| ☐ Oliven | ☐ Andre oljer |
| ☐ Annen margarin | |

## 13. FRUKT

Svar enten pr. måned eller pr. uke. < 1 betyr sjeldnere enn 1 gang.

| | Gang pr. måned | | | | | Gang pr. uke | | | | | | Mengde pr. gang | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | <1 | 1 | 2 | 3 | 1 | 2-3 | 4-5 | 6-7 | 8+ | | 1/2 | 1 | 2 | 3+ |
| Eple | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ |
| Appelsin, mandarin, grapefrukt | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ |
| Banan | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ |
| Druer | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (klase) | ☐ | ☐ | ☐ | ☐ |
| Eksotisk frukt (kiwi, mango) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ |
| Annen frukt (fersken, pære m.v.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | ☐ | ☐ | ☐ | ☐ |
| Jordbær, bringebær (friske, frosne) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | ☐ | ☐ | ☐ | ☐ |
| Blåbær | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | ☐ | ☐ | ☐ | ☐ |
| Multer | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | ☐ | ☐ | ☐ | ☐ |

Hvor mange frukter spiser du vanligvis pr. dag? 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9+ ☐

## 14. DESSERT, KAKER, GODTERI

Svar enten pr. måned eller pr. uke. < 1 betyr sjeldnere enn 1 gang.

| | Gang pr. måned | | | | | Gang pr. uke | | | | | | Mengde pr. gang | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | <1 | 1 | 2 | 3 | 1 | 2-3 | 4-5 | 6-7 | 8+ | | 1/2 | 1 | 2 | 3+ |
| Hermetisk frukt, fruktgrøt | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | ☐ | ☐ | ☐ | ☐ |
| Puddinger (sjokolade, karamell o.l.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1 ☐ | 2 ☐ | 3 ☐ | 4+ ☐ |
| Is (1 dl=1 pinne=1 kremmerhus) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1 ☐ | 2 ☐ | 3 ☐ | 4+ ☐ |
| Boller, julekake, kringle | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | 1 ☐ | 2 ☐ | 3 ☐ | 4+ ☐ |
| Skolebrød, skillingsbolle | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | 1 ☐ | 2 ☐ | 3 ☐ | 4+ ☐ |
| Wienerbrød, -kringle o.l. | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | 1 ☐ | 2 ☐ | 3 ☐ | 4+ ☐ |
| Smultring, formkake | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | 1 ☐ | 2 ☐ | 3 ☐ | 4+ ☐ |
| Vafler | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (plate) | 1/2 ☐ | 1 ☐ | 2 ☐ | 3+ ☐ |
| Sjokoladekake, bløtkake, annen fylt kake | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | 1/2 ☐ | 1 ☐ | 2 ☐ | 3+ ☐ |
| Søt kjeks, kakekjeks (Cookies, Bixit, Hob Nobs) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7+ ☐ |
| Sjokolade (60 g) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (plate) | 1/2 ☐ | 1 ☐ | 2 ☐ | 3+ ☐ |
| Drops, lakris, seigmenn o.l. | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (stk) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7+ ☐ |
| Smågodt (1 hg = 100g) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (hg) | 1/2 ☐ | 3/4 ☐ | 1 ☐ | 11/2+ ☐ |
| Potetgull (1 pose 100g=7 dl) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7+ ☐ |
| Annen snacks (skruer, crisp, saltstenger, lettsnacks o.l.) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (dl) | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7+ ☐ |
| Peanøtter, andre nøtter (1 pose 100g = 4 never) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (neve) | 1 ☐ | 2 ☐ | 3 ☐ | 4+ ☐ |

## 15. KOSTTILSKUDD (bs = barneskje, ts = teskje)

| | Hele året | Bare vinter-halvåret | Gang pr. uke: 0 | <1 | 1 | 2-3 | 4-5 | 6-7 | Mengde pr. gang | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | 1 ts | 1 bs | 1 ss | |
| Tran | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | | ☐ | ☐ | ☐ | |
| Trankapsler | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | kapsler | 1 ☐ | 2+ ☐ | | |
| Fiskeoljekapsler | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | kapsler | 1-2 ☐ | 3-4 ☐ | 5-6 ☐ | 7+ ☐ |
| Multipreparater | | | | | | | | | | | | | |
| | | | 0 | <1 | 1 | 2-3 | 4-5 | 6-7 | | 1 | 2 | 3 | 4+ |
| Sanasol | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | bs | ☐ | ☐ | ☐ | ☐ |
| Biovit | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | bs | ☐ | ☐ | ☐ | ☐ |
| Vitaplex | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| Kostpluss | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| Vitamineral | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| Annet | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| | | Hvis annet, hvilket?..... | | | | | | | | | | | |
| Jernpreparater | | | | | | | | | | | | | |
| | | | 0 | <1 | 1 | 2-3 | 4-5 | 6-7 | | 1 | 2 | 3 | 4+ |
| Ferro C | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| Hemofer | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| Duroferon Duretter | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| Annet | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| | | Hvis annet, hvilket?..... | | | | | | | | | | | |
| | | | 0 | <1 | 1 | 2-3 | 4-5 | 6-7 | | 1 | 2 | 3 | 4+ |
| B-vitaminer | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| C-vitamin | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| D-vitamin | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| E-vitamin | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| Folat (folsyre) | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| | | | 0 | <1 | 1 | 2-3 | 4-5 | 6-7 | | 1 | 2 | 3 | 4+ |
| Kalktabletter | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| Fluortabletter | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| Annet | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | tablett | ☐ | ☐ | ☐ | ☐ |
| | | Hvis annet, hvilket?..... | | | | | | | | | | | |

## 16. NÅR SPISER DU PÅ HVERDAGER?

HOVEDMÅLTIDER som frokost, formiddagsmat, middag, kvelds.

Omtrent klokken

| 6 | 8 | 10 | 12 | 14 | 16 | 18 | 20 | 22 | 24 | 2 | 4 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ |

MELLOMMÅLTIDER som kaffe, frukt, godteri, snacks m.v.

Omtrent klokken

| 6 | 8 | 10 | 12 | 14 | 16 | 18 | 20 | 22 | 24 | 2 | 4 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ | ☐ ☐ |

## 17. MENER DU SVARENE I SPØRRESKJEMAET GIR ET BRUKBART BILDE AV KOSTHOLDET

Ja ☐ Nei ☐

Er det matvarer/produkter du regelmessig bruker, og som ikke er nevnt i skjemaet?

.....

.....

## 18. ER DU FORNØYD MED KROPPSVEKTEN DIN SLIK DEN ER NÅ?

- ☐ Ja
- ☐ Nei, jeg ønsker å slanke meg
- ☐ Nei, jeg ønsker å legge på meg

## 19. KJØNN

Mann ☐ Kvinne ☐

Vennligst se etter at du har svart på alle spørsmål.

**Takk for innsatsen!**



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# I

This article is removed.

# II

This article is removed.

III

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IV

This article is removed.