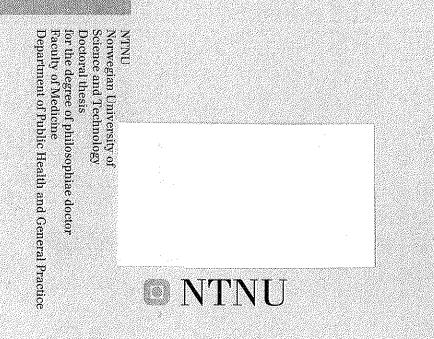
Doctoral Theses at NTNU 2005:40

Wenche Brenne Drøyvold

Epidemiological studies on weight change and health in a large population

The Nord-Trøndelag Health Study (HUNT)



Wenche Brenne Drøyvold

Epidemiological studies on weight change and health in a large population

Trondheim, February 2005

Doctoral thesis for the degree of philosophiae doctor

Norwegian University of Science and Technology Faculty of Medicine Department of Public Health and General Practice

O NTNU

en la filipio en la compañía de la c En la compañía de la c En la compañía de la c

.

Wenche Brenne Drøyvold

Epidemiological studies on weight change and health in a large population

The Nord-Trøndelag Health Study (HUNT)

Department of Public Health and General Practice Faculty of Medicine Norwegian University of Science and Technology Norway Wenche Brenne Drøyvold

ISBN 82-471-6947-9 Trondheim, Norway, 2005

an an an Arrainn an Arrainn an Arrainn Martainn an Arrainn an Arrainn an Arrainn

To my family!

3

.

Content

Abstract	6
Definitions and abbreviations	9
Acknowledgements	11
List of papers	
1. Introduction	
1.1 The obesity epidemic	15
2. Main objectives	35
3. Material	36
3.1 The Nord-Trøndelag County	
3.2 The Nord-Trøndelag Health Study 3.2.1 Study population	
3.3 Variables	
3.3.1 Study variables 3.3.2 Potentially confounding variables	45 47
4. Methods	49
4.1 Study design	49
4.2 Statistical analyses	49
4.3 Ethics	52
4.3 Funding	53
5. Reviews of papers	
5.1 Review of Paper I	54
5.2 Review of Paper II	56
5.3 Review of Paper III	58
5.4 Review of Paper IV	60
6. General discussion	62
6.1 Methodological considerations	62
6.1.1 Precision (Lack of random error) 6.1.2 Validity (Lack of systematic error)	62 63
6.2 Evaluation of the results	

6.2.1 Leisure time physical activity and change in BMI (Paper I, Paper II) 6.2.2 Change in BMI and its impact on blood pressure (Paper III) 6.2.3 Change in BMI and mortality (Paper IV)	75
6.3 The obesity epidemic in the future	
6.4 Future research	80
7. Conclusion	85
References	
Paper I-IV	•••••
Appendix	

Abstract

Background: The prevalence of overweight and obesity has increased in Norway and worldwide in recent decades, and is still increasing. Important factors are probably lifestyle, environmental changes, genetic susceptibility, and interactions between these factors.

Objective: The major objective of this thesis was to investigate, in an adult population and with a prospective epidemiological perspective, the association between body mass index and selected variables that probably are linked to the metabolic syndrome. More specific, the objectives were:

- To investigate the association between leisure time physical activity and change in body mass index
- To investigate the association between change in body mass index and its impact on blood pressure
- To investigate the association between change in body weight, and especially weight loss and mortality.

Material and methods: The thesis is based on data sets from the Nord-Trøndelag Health Study (HUNT), created by linkage of data from HUNT 1 in 1984-86 and HUNT 2 in 1995-97. Additional data sets were created by linkage of data from HUNT and from The Death Registry at Statistics Norway. About 46,000 inhabitants in Nord-Trøndelag County participated in both surveys in the Nord-Trøndelag Health Study. Multivariable regression analyses were applied.

Results: In apparently healthy women with normal weight (BMI 18.5-24.9 kg/m²) aged 20-49 years, we found a modest effect of leisure time physical activity on the

change in BMI. Those who were physically active at leisure on a high level increased less BMI compared to those who were physically active on a low level.

In apparently healthy men with normal weight aged 20-69 years we found that BMI increased less among those who were physically active at leisure compared to those who were not physically active. The study of the associations between low, moderate and high level of leisure time physical activity and change in BMI showed a U-shaped effect. In additional analyses men who were physically active with high intensity increased less in BMI compared to those who were physically active with a lower intensity. No strong linear relationship of the association between change in BMI and different levels of leisure time physical activity were found neither among women nor men.

Both in women and men the change in BMI had a substantial effect on diastolic and systolic blood pressure which was independent on the initial and attained BMI, where the odds ratio of having hypertension at the second survey was positively associated with increased BMI. Additionally, to change BMI category (World Health Organisation's categorisation) from the first to the second survey had a strong effect on systolic and diastolic blood pressure, which was independent of initial and attained BMI category.

Weight loss was positively associated with both total mortality, noncardiovascular mortality and cardiovascular mortality in women and men, and the estimated effects did not change substantially even if initial BMI, smoking status and leisure time physical activity was considered. Weight gain was not associated with any increased mortality.

Conclusion: This thesis has shown that to be physical active at leisure had a modest effect of change in BMI at population level both among women and men. Weight gain

in apparently healthy persons was associated with unfavourable consequences for blood pressure level, but had minimal effect on mortality. However, weight loss was associated with increased mortality, although the relationship has to be explored.

8

Definitions and abbreviations

HUNT	The Nord-Trøndelag Health Study
	(Helseundersøkelsen i Nord-Trøndelag)
BMI	Body mass index (an index of weight-for-height and is defined as
	weight in kilograms divided by the square of height in metres, kg/m^2)
WHO	World Health Organisation
LTPA	Leisure time physical activity
NIDDM	Non-insulin-dependent diabetes mellitus
CI	Confidence Interval
SD	Standard deviation
SPSS	Statistical Package for the Social Sciences
CVD	Cardiovascular disease
ICD	International Classification of Diseases
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure

Biobank:A systematic collection of cell, tissue or blood samples, which is storedto be retrieved for analysis (Norwegian Research Council 2001).

WHO's classification of adults according to BMI:

Underweight	BMI <18.5 kg/m ²
Normal weight	BMI 18.5-24.9 kg/m ²
Overweight	BMI 25.0-29.9 kg/m ²

Obesity $BMI \ge 30 \text{ kg/m}^2$

<u>Physical activity:</u> A global term referring to 'any bodily movement produced by skeletal muscle resulting in a substantial increase over the resting energy expenditure'¹.

and the second second second

<u>Leisure time physical activity:</u> Activity undertaken in the free time and is selected on the basis of personal needs and interests and includes exercise and sports¹.

Acknowledgements

The thesis is based on studies performed while I was receiving a research fellowship from the Norwegian Research Council. This financial support gave me the possibility to investigate interesting health perspectives and to get more knowledge about the obesity epidemic based on data from the Nord-Trøndelag Health Study.

I want to thank colleagues at the HUNT research centre in Verdal for being forthcoming and service minded to all my questions about both data access and administrative procedures. In addition I want to point out the humorous and friendly working environment at the HUNT research centre. Also very sincere thanks go to my colleagues at the Department of Public Health and General Practice in Trondheim for interesting discussions and working environment.

Jostein Holmen was my main mentor, and through the years of co-operation he gave me a unique opportunity to learn epidemiology. His interest and willingness to discuss my work, from methodological details to the practical implications of results, encouraged me to complete the thesis. Thank you!

In addition Tom Ivar Lund Nilsen has contributed both as a mentor, but also as a very good friend at the department. The work would have been much more difficult without his support and engagement. I especially want to thank him for interesting discussions about the way of thinking and about methodical considerations in epidemiology.

Kristian Midthjell is co-author of all papers in this work, and I am grateful for his engagement and willingness to take part in discussions about the manuscripts.

Special thanks to Peter Nilsson at the University Hospital in Malmö for interesting ideas and engagement in my work.

I want to give a very special thanks to my husband and good friend Terje who has supported and given me the opportunity to go through this process. In addition I want to mention our two children (Christian and Magnus) and my parents, who all are very valuable for me, for their good support through this process and by being themselves – thanks a lot.

Trondheim, 22.10.04

Wenche Brenne Drøyvold

List of papers

 $z_{i} \in \mathcal{T}$

The thesis is based on the following papers:

- Leisure time physical activity and change in body mass index: an 11-year follow-up study of 9357 normal weight healthy women 20-49 years old.
 J Womens Health (Larchmt). 2004 Jan-Feb; 13(1):55-62.
- II BMI change and leisure time physical activity (LTPA): an 11-y follow-up study in apparently healthy men aged 20-69 y with normal weight at baseline. Int J Obes Relat Metab Disord. 2004 Mar; 28(3):410-7.
- III Change in body mass index and its impact on blood pressure. A prospective population study. The Nord-Trøndelag Health Study (HUNT), Norway.
 (Submitted)
- IV Weight change and mortality. The Nord-Trøndelag Health Study. (Submitted)

1. Introduction

Humans of the 21th century are mainly a product of selection and adaptation, even if the play of chance also has been involved. Sufficient energy and the ability of being physically active have been main survival factors. In the ancient past, when man lived from hunting and gathering, the availability of food was not constant and great nutritional variation between days and seasons was an inevitable part of daily life. Under such circumstances, the deposition of fat in the body served as a buffer for harder periods, and was an important survival factor.

Today, the modern human has the same mechanisms for depositing fat, but the living conditions have changed dramatically during a very short time. In general, both in developed and developing countries of today, high-energy nutrition is easily available and the need for being physically active has decreased substantially. This may disturb both the energy balance and the metabolic profile.

As a consequence and a paradox, the fat storing mechanism has become a big threat against longevity, public health and the health care systems. With this perspective, prevention of overweight and obesity, and increased knowledge about environmental and biological mechanisms involved in the obesity epidemic, is necessary.

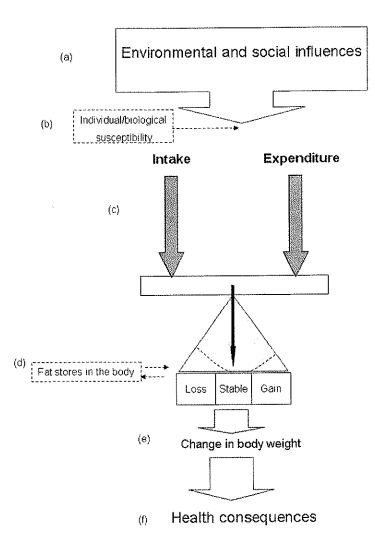
1.1 The obesity epidemic

In a historical perspective, body weight has been considered to be part of human health as far back as Graeco-Roman times, but only little scientific progress was made towards understanding the condition and its real impact on health until the 20th century¹. Additionally, epidemiology is a relative young science, and only since World War II a systemised body of principles on how to design and evaluate studies has emerged².

Body weight regulation

Total energy demands of the human body can be partitioned into basal metabolic rate (the requirement to sustain general cellular processes), resting metabolic rate and active energy expenditure in which physical activity is an important part^{1,3}. Diet has · the main function to balance the energy expenditure, and body weight is a function of energy and nutrient balance over an extended period of time. Energy expenditure is a continuous process, but energy intake is not. Therefore, because of the laws of thermodynamics, where energy can neither be created nor destroyed, the imbalance between intake and expenditure requires a possibility to temporarily store energy ⁴ (Figure 1). In energy deposition, both fat- and lean tissue are involved⁵. The main advantage by storing fat is the higher energy density. As a result of the involved processes, the body weight in adults is remarkably stable for long periods of time, and this fine balance is established, the body has a stronger defence against under nutrition and weight loss compared to over consumption and weight gain^{6,7}. The adipose tissue mass may mainly increase by two mechanisms; by fat cell hypertrophy

or by fat cell hyperplasia. In adults, the major change in adipose cellularity following weight loss is shrinkage of adipocytes with no change in cell number^{8,9}. The energy balance equation may be expressed as an equation between energy intake and energy expenditure, with energy storage as a buffer. This means that an imbalance between intake and expenditure requires mobilisation or storage of energy. Hence, an increase in body weight is a result of over nutrition or low energy expenditure, or both. Whatever mechanisms involved, the weight at which regulation occurs, differs from one person to another. This variation is partly caused by genetic and partly by developmental influences¹⁰, where both demographic, socio-cultural, biological and lifestyle factors are shown to be associated with overweight¹¹ (Figure 1).



.

. . «

Figure 1. The principles of energy balance and regulation in human body: (a) environmental and social factors as physical activity, marital status, education and smoking. (b) Genetic and other factors. (c) Energy balance. (d) Fat storage. (e) Energy imbalance results in weight change. (f) Change of body weight might have different health consequences.



Body mass index (BMI)

People with overweight and obesity carry an excessive amount of body fat, generally estimated by combining measures of height and weight. The most widely used weight-for height index is BMI, also referred to as the Quetelet's $Index^{12,13}$. BMI is calculated as body weight in kilograms divided by the squared value of body height in metres. Adult body height reflects both the genetic potential and the result of living conditions, e.g. nutrition, during childhood and adolescence^{14,15}. A good correlation between BMI and the percentage of body fat in large populations is documented¹⁶, and it is shown that BMI predicts amount body fat within ±5% in 7 out of every 10 people in the population¹⁷.

BMI categorisation

The World Health Organisation has developed categories of BMI that classifies persons into four weight groups: underweight (BMI<18.5 kg/m²), normal weight (BMI 18.5-24.9 kg/m²), overweight (BMI 25.0-29.9 kg/m²) or obesity (BMI≥30 kg/m²). This categorisation is based on the association between different BMI levels and morbidity and mortality rates¹, but the limits are not strict^{14,18}, and smoking may be a modifier of this association^{19,20}. The BMI cut points recommended from the WHO Consultation on Obesity were the first such cut-off points at international level, and they have been generally accepted even if the relevance to public health especially in Asia and the Pacific Regions is discussed²¹. Also, there is lack of consensus regarding whether the lowest risk of chronic diseases occurs among the leanest individuals, since there is some evidence that the relation between BMI and health outcomes is not linear, but rather J- or U-shaped^{14,22,23}. However, when

be at relatively low risk^{19,20,24}. There are several reasons for classifying people as overweight or obese: to provide meaningful comparisons of body weight status within and between populations; to identify individuals and groups at increased risk of morbidity and mortality; to identify priorities for intervention at individual and community levels and establish a firm basis for evaluating diverse intervention strategies¹.

The relative relationship between body weight and mortality is similar in men and women, which explaines why the WHO categorisation is not gender specific. However, the absolute mortality is much lower in women¹⁶. The same relative risk and the lower absolute risk associated with overweight and obesity among women compared to men implies that women probably tolerate body fat better than men¹⁴. One reason may be that excess body fat in women is usually distributed as subcutaneous fat and mainly peripherally (thighs, buttocks, breasts), and in men there is a relative excess of body fat stored in the abdominal cavity and as abdominal subcutaneous fat¹⁶. This is an example of the importance of not regarding women just as small men, though many medical studies were restricted to men, resulting in a medical practice defined as normal human physiology and pathophysiology²⁵. Within each WHO-category of BMI there can be substantial individual variation in total and visceral adiposity, and therefore also in several metabolic variables.

Fat and obesity

Obesity may develop when there has been an energy imbalance for a considerable period of time²⁶, and can be defined simply as the disease in which excess body fat has accumulated to such an extent that health may be adversely affected. But the amount of excess body fat, the distribution of fat within the body, and the associated

health consequences vary considerably between individuals^{1,27} (Figure 1 (f)). The adipose tissue, i.e. the subcutaneous and visceral fat, serves as storage of energy in the form of fat, where triglycerides are the main storage lipid. The amount of body fat differs between men and women in the normal weight category (BMI 18.5-24.9 kg/m²), where men has 15-20% body fat compared to 25-30% in women¹⁶. In extremely obese individuals the body fat might be up to 60-70% of body weight²⁸. The abdominal fat mass may vary dramatically within a narrow range of total body fat or BMI, and men have on average twice the amount of abdominal fat compared to what is generally found in premenopausal women²⁹. It seems that women have larger tolerance for overweight^{14,30}, and women accumulate relatively less abdominal adipose tissue than men³¹. Additionally, it is probably a positive correlation between a high BMI in adolescentce and adulthood³².

Increased amount of body fat does not only mean increased body weight. In 1994 the *OB* gene and the gene product leptin was discovered³³, which links the components in the energy balance equation together: Leptin is secreted by the adipocytes to the blood and receptores in the brain are responsible for the convertion of the signal from the adipose tissue to regulatory processes counteracting the tendency to accumulate fat in the fat tissue by decreasing food intake and increasing energy expenditure³⁴. These mechanisms create the possibility that development of obesity is caused by resistance in the regulatory system to the leptin signal or insufficient secretion of leptin from the adipocytes³⁵.

Body fat works as an endocrine organ, and central fat deposition, independent of fat storage in other anatomic areas, is associated with an altered metabolic profile. As an example, obese women have higher circulating testosterone level³⁶.

Studies have shown that the androgen pattern of fat accumulation is associated with a variety of metabolic derangements, including dyslipidemia, hypertension and glucose intolerance³⁷. Thus, even at the same weight level, individuals with a greater amount of visceral fat are more likely to develop serious health conditions associated with overweight and obesity. Abdominal adiposity is found to be an important component of the insulin resistance syndrome, and it has been recognised that regional fat distribution, as in centrally or abdominally obese persons, might be more strongly linked to CVD than overall fatness³⁸.

Prevalence of overweight and obesity

Until the 1960s, obesity was relatively rare worldwide. But since the late 1970s, the prevalence of obesity has increased rapidly^{30,39,40}. Today, the prevalence of overweight and obesity is increasing worldwide⁴¹⁻⁴³, and a significant weight gain has also been observed in the The Nord-Trøndelag Health Study⁴⁴. Figure 2 illustrates the increase in the proportion of obesity among women and men aged 40-44 years in Norway during recent decades. And the prevalence of obesity has increased in both genders during the last decades, but more rapidly in men than women. According to WHO, 1.1 billion people worldwide are overweight⁴⁵, which means that the number overweight people equals the number of people who are underfed and underweight (BMI<18.5 kg/m²). In the United States there are two million more obese women than obese men, and the prevalence of obesity, and the highest increase has been among women⁴⁶ (Table 1). In Brazil during the 14 years between 1975 and 1989, the obesity

prevalence among women increased from 3.1% to 5.9%, and from 8.2% to 13.3% among men⁴⁷.

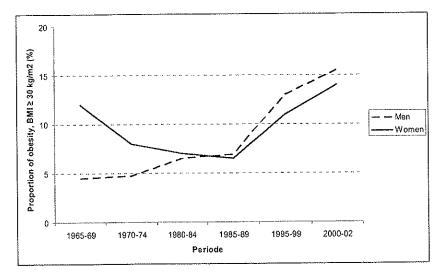


Figure 2. The prevalence of obesity in Norway among men and women aged 40-44 years (source: <u>www.fhi.no</u> (Engeland)).

The prevalence of obesity in European countries is estimated to be 10-12% in men and 10-25% in women¹, with the most dramaic increase in obesity prevalence in England where the proportions have more than doubled from 6 to15% in men and from 8% to 16.5% in women since 1989⁴⁸. This dramatic increase has occurred in both genders and across the major racial and ethnic groups⁴⁹.

Period of surveys (y)		Men		Women	
First	Last	First	Last	First	Last
1960-62	1988-94	10	20	15	25
1976	1993	1	2	3	3
1978	1991	39	58	59	77
1978-79	1991-93	10	14	10	11
1980	1983	9	12	8	13
1980	1995	6	15	8	17
1980-81	1988-89	5	5	9	9
1984-86	1995-97	7	16	11	21
1984-86	1995-97	8	14	13	18
1985	1992	14	21	22	27
1987	1995	6	8	8	8
1994-96	1997-99	10	14	9	12
	First 1960-62 1976 1978 1978-79 1980 1980 1980-81 1984-86 1984-86 1985 1987	First Last 1960-62 1988-94 1976 1993 1978 1991 1978 1991 1978 1991 1978 1991 1978 1991 1978 1991 1978 1991 1978 1991 1978 1991 1978 1991 1980 1983 1980 1995 1980-81 1988-89 1984-86 1995-97 1985 1992 1987 1995	First Last First 1960-62 1988-94 10 1976 1993 1 1978 1991 39 1978 1991-93 10 1980 1983 9 1980 1995 6 1980-81 1988-89 5 1984-86 1995-97 7 1985 1992 14 1987 1995 6	First Last First Last 1960-62 1988-94 10 20 1976 1993 1 2 1978 1991 39 58 1978-79 1991-93 10 14 1980 1983 9 12 1980 1995 6 15 1980-81 1988-89 5 5 1984-86 1995-97 7 16 1985 1992 14 21 1987 1995 6 8	First Last First Last First 1960-62 1988-94 10 20 15 1976 1993 1 2 3 1978 1991 39 58 59 1978-79 1991-93 10 14 10 1980 1983 9 12 8 1980 1995 6 15 8 1980 1995 5 5 9 1984-86 1995-97 7 16 11 1985 1992 14 21 22 1987 1995 6 8 8

Table 1. Prevalence of obesity (Body mass index≥30kg/m² in adult men and women at two different time points in selected North American and European countries and in countries in the Far East¹. Prevalence of obesity (%)

All non-diabetic women and men who participated in HUNT 1 and HUNT 2 aged 20 years or more at HUNT 1⁴⁴.
 All those who participated in both HUNT 1 and in HUNT 2 aged 20 years or more at HUNT 1.
 Prevalence of obesity among persons aged 40-42 years in two periods from eight counties³⁹.

The prevalence of obesity in the United States has been projected to be 39 % in 2008⁵⁰. Today, the prevalence of overweight is higher in men than in women, but the prevalence of obesity is higher in women compared with men^{51,52}. The increasing prevalence of obesity is documented simultaneously as the average caloric intake of American men and women may have decreased⁵³, but this is not consistent⁵⁴⁻⁵⁶. Also in Norway there is some evidence that the mean calorie intake has been stable⁵⁷.

The obesity epidemic is a growing threat to health and health care systems in both developed and developing countries¹, and next to cigarette smoking, obesity today is the second leading cause of preventable deaths in US⁸. In 1999, Allison et al ⁵⁸ estimated that between 280,000 and 325,000 deaths could be attributed to obesity in the United States. And by 2000 the obesity problem had already grown to such an extent that the World Health Organisation declared it was the greatest health threat facing the Western World¹.

The increase in obesity prevalence has been too rapid to be caused by major genetic changes. Therefore, the obesity epidemic is probably not due to a defect in the biological system. Biology clearly contributes to individual differences in weight and height, but the rapid weight gain during the past three decades is most probably a result of a changing environment. Overweight and obesity has become so common that it today has replaced the more traditional public health concerns, including under nutrition and infectious diseases. The medical-care cost burden of obesity has been calculated to be 5.5-7.0% of national health expenditures in the US and to be 2.0-3.5% in some other developed countries⁵⁹. We have no data on the economic burden of overweight and obesity in Norway, but the Norwegian Diabetic Association has estimated the direct costs of diabetes to NOK 4.4 billion in 1999⁶⁰.

Health consequences

Obesity has had disease status since 1985⁶¹. In addition a number of diseases can be linked to overweight and obesity, and each disease can further be classified into two pathophysiological categories⁶². The first arises from the increased mass of fat which includes the stigma of obesity and the behavioural responses it produces, musculoskeletal disorders⁶³, and sleep apnoea⁶⁴. The second category comprises metabolic changes associated with excess fat, and includes diabetes type 2⁶⁵, gallbladder disease⁶⁶, hypertension⁶⁷, cardiovascular disease⁶⁸, and some forms of cancer^{69,70}. It is suggested that weight change is associated with a more unfavourable relative change in fat-free mass in men than women, suggesting that the metabolic and health consequences of weight change may be dependent on gender⁷¹.

Obesity is related to several disturbances in the cardiac structure⁷², and weight loss has been found to be an important factor to reduce left ventricular hyperthrophy⁷³, which is one strong risk factor for morbidity and mortality⁷⁴. At present there is no universally accepted definition of the metabolic syndrome⁷⁵. According to WHO metabolic syndrome includes diabetes or impaired glucose tolerance or insulin resistance, and two of the following factors; elevated blood pressure, dyslipidemia, microalbuminuria and obesity⁷⁵. Upper abdominal obesity is associated with increased risk of post-menopausal breast cancer in women⁷⁶. Ovulatory infertility has been found to be attributable to overweight and sedentary lifestyle^{77,78}. Additionally, in both overweight and non-overweight women a positive association between weight loss and psychological well-being has been found⁷⁹. As a consequence of the close linkage between overweight/obesity and various diseases, it

is shown that use of health care resources increases proportionately with excess body fat⁸⁰.

Concern about the rising prevalence of obesity in various parts of the world has stimulated research into the health consequences of this phenomenon, and largescale epidemiological studies have established that obesity is an independent risk factor for mortality^{22,81,82}. Years of life lost due to obesity is age and gender specific, with the highest effects among young people and in men⁸³. In the Framingham Study, Peeters *et al*⁸⁴ estimated that overweight women lost 3.3 years of life and men lost 3.1 years compared to those with normal weight. In the obese, years of life lost were 7.1 in women and 5.8 in men compared to those with normal weight. In a study of extremely obese individuals (BMI>45 kg/m²), Fontaine *et al*⁸³ found 13 years life lost in men and 8 years life lost in women. Additionally, waist circumference is found to be a strong predictor of mortality¹⁸. Weight gain is associated with an increase in the health care costs, thus the observed obesity epidemic have dramatically economical consequences⁸⁵. As a consequence of the association between overweight/obesity and morbidity/mortality, a further increase in the prevalence of obesity will lead to an increase in unhealthy life-years and in direct and indirect health care costs⁸⁶.

Blood pressure and body weight

Measuring blood pressure has a long history, but recognition of diseases associated with high blood pressure is relatively new. Until the 1940s, high blood pressure was often referred to as benign essential hypertension, indicating a condition of unknown origin and with little consequence for health¹⁷. Since the 1960s it is recognised that elevated blood pressure is a strong risk factor for stroke^{87,88} and cardiovascular

disease⁸⁹⁻⁹¹, and the treatment of hypertension has increased gradually and considerably over the years.

The association between obesity and hypertension was first recognised in the early 1900s⁶⁷. Today 220,000-322,000 patients are on anti-hypertension drugs in Norway (Irene Hetlevik, personal communication). The cost of treating hypertension has increased substantially from 1990 and today nearly 1 billion NOK is spent on anti-hypertension medication in Norway⁹², which ilustrates the epidemic dimension.

Overweight and obesity is the most common cause of hypertension in most industrialised countries^{75,93}. The mechanisms leading to hypertension in obese persons are not completely known, but increased sympathetic nervous system activity, insulin resistance, structural changes in the kidney, altered vascular function, the reninangiotensin-aldosteron system, the sympathetic nervous system, and the hypothalamic pituitary and adrenal axis are probably involved^{67,94}. The fact that there is a clear association between BMI and blood pressure even in non-obese, lean populations, indicates that the effect of weight gain on blood pressure regulation may be more complex than can be explained simply by increasing adiposity⁹³. In cross-sectional and prospective studies there is a positive association between obesity and hypertension⁹⁵⁻⁹⁷. Risk estimates from the Framingham Heart Study suggest that approximately 78% of hypertension in men and 65% in women may be directly attributed to obesity⁹⁸.

Previously it is shown that a reduction of diastolic blood pressure of 5, 7.5, and 10 mm Hg were respectively associated with at least 34%, 46%, and 56% less stroke, and at least 21%, 29%, and 37% less cardiovascular disease⁹⁹. Results from the Framingham Study has illustrated that lowering the blood pressure 10 mm Hg was associated with a 30% reduction in the total attributable mortality¹⁰⁰. These examples

illustrate how important it is to get scientific knowledge about the separate association between change in blood pressure and change in weight, both in a health consequence and health prevention perspective.

The knowledge of the association between change in weight and change in blood pressure independent of initial and attained body weight is insufficient, even if short-term weight loss is associated with blood pressure reduction^{101,102}. As a consequence, we need to know more about how weight changes, especially weight gain in an obesity epidemic view, affect the blood pressure development at population level.

Obesity prevention

The world wide obesity epidemic calls for preventive actions. The World Health Organisation is strongly involved in marketing both primary and secondary strategies for improving health at a population and individual level¹. Influence on governments, the food industry, the media, and the consumers is important in the work against overweight and obesity. Effects from prevention actions in public health may not be reached in many years, because culture changes at both international and national levels must be reached^{103,104}. To prevent weight gain both at population and individual level, it is necessary to identify the reasons for the energy imbalance. In search for the causes of the obesity epidemic increased intake, decreased expenditure, and increased energy storage must be considered as possible pathogenic mechanisms through which the genes or the environmental factors may operate. Jeffery has described that the current obesity epidemic is caused largely by an environment promoting excessive food intake and discouraging physical activity¹⁰³. Possible factors in the environment that promote over-consumption of energy include the easy availability of a wide

variety of good-tasting, inexpensive, energy-dense foods, and serving of these foods in large portions⁵⁰. In addition studies have shown that the environment and travel patterns are important and strong predictors of obesity independent on gender and ethnicity, and reducing time in a car can be effective as health interventions¹⁰⁵. The urban planning and transportation systems should also be included in the prevention strategy for example by arranging for walking and bicycling^{106,107}.

A discrepancy has been documented between men and women in perceiving themselves as overweight. Gutiérrez-Fisac *et al* ¹⁰⁸ found that 42% of men and 25% of women with BMI between 25.0 and 28.9 did not perceive their weight as abnormally high. If people themselves, especially men, do not see their own overweight, it will probably be hard to accept implementation of prevention actions against overweight and obesity.

It has been tried to carry out intervention at a population level, but it is hard to measure and document the effects of the program^{109,110}.

The most common primary and especially secondary prevention at individual level of overweight and obesity is to engage in an energy restricted diet¹¹¹, and recent surveys suggest that about 40% of people in Western societies may be engaged in some form of energy restriction at any time¹¹². Almost twice as many women (61%) compared to men (32%) have tried to lose weight¹¹³ although men are at greater risk of developing abdominal obesity. Serdula *et al* ¹¹⁴ confirmed that weight loss and weight maintenance are common concerns for men and women, but the recommended combination of reducing calorie intake and increasing leisure time physical activity was not often followed by the majority. Today, an 'industry' with commercial and private actors offers strategies for losing weight, especially by dieting,¹¹⁵. They offer weight loss courses with diet-recommendations and slimming products, where weight

loss effects in few weeks are almost guaranteed. There are different recommendations for diets, but it seems that caloric balance (calories in vs. calories out), rather than macronutrient composition is the major determinant of weight loss^{116,117}. However, the effect of macronutrient content on long-term weight maintenance and adherence is not clear. Furthermore, it is not known whether maintenance of weight loss and dietary adherence are related to psychological issues (and brain neurochemistry), physiological parameters (e.g., hormones involved in body weight regulation such as insulin and leptin), physical activity, energy density, or some other factor(s)^{116,117}. Scientifically, the current most solid recommendation for people who want to lose weight and keep weight off is a permanent switch to a diet reduced in calories and fat in combination with physical activity¹¹⁸⁻¹²⁰. As a paradox to all the engagement and money spent for losing weight, weight stability after weight loss for an extended period of time is shown difficult, and most people regain weight after some years^{121,122}, The situations where a considerable proportion of women and men are not seeing their own overweight, parallel to the focus on slimming behaviour might seem as a paradox.

Waaler¹⁴ found a U-shaped association between BMI and mortality based on 18 million person-years. The results, however, do not tell anything about the reversibility, i.e. about the effect of actively reducing the BMI ratio by weight reduction. In animal models restricting energy intake has been shown to increase longevity¹²³. In several observational studies weight loss at population level has been associated with increased mortality¹²⁴⁻¹²⁶, but the results are not consistent^{127,128}. Whether weight loss in already overweight or obese adults reduces health risk to the level of individuals who never gained weight in the first place remains unclear^{129,130}.

Nevertheless diet and physical activity have been found to be important factors in a weight loss strategy¹³¹.

Although there is a general agreement that the environment of the modern societies is 'fueling' the obesity epidemic, the relative contributions of factors influencing food intake and physical activity are not clear.

Physical activity

The physical activity epidemiology has evolved from old ideas dating to the use of structured exercise for health promotion in China about 2500 B.C., the ancient Indian Ayurveda system of medicine of the ninth century B.C., and the use of vigorous exercise by ancient Greek physicians (Herodicus, Hippocrates, Asclepiades, and Galen)¹⁷. The modern scientific history of physical activity is short and began after World War II with focus on the epidemic of cardiovascular disease that was beginning to engulf the Western world¹³². In the early 1950s, Dr. Jeremy Morris observed that the active conductors on London's double-decker buses were at lower risk of coronary heart disease compared to the drivers¹³³. As a consequence, in 1996 the US Surgeon General's report recommended '30 min of moderate activity all days of the week', which was broadly adopted throughout much of the Western world mainly to reduce the risk of heart disease. Erlichman *et al*¹³² have investigated the magnitude and type of physical activity levels are needed for weight stability than for inducing substantial improvements in cardiovascular health.

Today the promotion of physical activity has emerged as an important initiative to improve public health¹³⁴. What levels of physical activity that are needed for prevention of overweight and obesity is still unclear, even if 45-60 minutes of

moderate physical activity has been stated as a daily dose needed for prevention of transition to overweight and obesity¹³⁵. Additionally, in individuals who are physically active the intensity may be important^{136,137}.

A measure of physical activity level is a surrogate/proxy of energy expenditure and depends on intensity, frequency, duration and type of the activity¹. The energy expenditure linked to leisure time physical activity is probably the most variable component of the total energy expenditure^{138,139}. Both energy used during the activity, in the restitution period after the activity, and change in resting metabolic rate are involved¹⁴⁰. Physical activity at leisure has an important influence on the physiological regulation of body weight by particular affecting total energy expenditure, fat balance and food intake. It is shown that there is a weak coupling between energy intake and expenditure by physical activity¹⁴¹. Small to moderate increase in physical activity will not be accompanied by compensatory increases in intake, and a decrease in physical activity is not necessarily followed with an equal amount decrease in energy intake¹⁴². The total amount of energy expended depends on the characteristics of the physical activity (mode, intensity, duration and frequency) and of the individual performing the exercise (body size, level of habituation and fitness). Nevertheless, in Norway it seems that the proportion of women and men being physical active at leisure has increased parallel to the obesity epidemic143.

Weight gain and a sedentary lifestyle are often associated with the development of the metabolic syndrome. In the light of the current obesity epidemic, the metabolic syndrome implies a serious and growing problem for public health authorities and decision makers¹⁴⁴. At population level the metabolic syndrome is closely linked to lifestyle factors. A moderate level of physical activity reduces the

risk of developing the metabolic syndrome¹⁴⁵. Physical activity is negatively associated with insulin consentrations independent of body weight and ethnicity¹⁴⁶. Further, physical activity and cardiorespiratory fitness is shown to be inversely related to the development of the metabolic syndrome¹⁴⁵.

Excess weight gain more often parallels reduced physical activity than increased caloric intake⁸. Inadequate physical activity has been estimated to cause nearly 30% of all deaths from heart disease, colon cancer, and diabetes¹⁴⁷. And probably the greatest health benefits at population level will derive if the most sedentary individuals start to be light-to-moderate physically active. In an editorial Tim Bayers wrote that without regular physical activity, weight control can usually not be achieved¹⁴⁸. But, regular physical activity can also improve longevity, even for those with body mass-indexes in the overweight range.

The importance of genetic factors and physical activity levels has been studied in twins by Maia *et al*¹⁴⁹. In men genetic factors accounted for 63% of the total participation in physical activity, whereas in females the estimate was about 40%. Men generally respond more favourably than women to the effects of exercise on weight loss, and one possible explanation involves the gender differences in fat distribution. Fat distributed in the upper body and abdominal regions shows active lipolysis to sympathetic nervous system stimulation and becomes preferentially mobilised for energy during exercise^{150,151}. Thus, upper-body fat distribution in the abdominal region has greater sensitivity to lose fat as a response to regular exercise. Large exercise-induced weight loss is associated with a preferential reduction in abdominal fat and a corresponding maintenance of fat free-mass¹⁵².

Hill *et al*⁵⁰ suggested that increasing the amount of regular physical activity and reducing energy intake by an amount equal to 100 kcal/day could prevent weight

gain in most of the population. There is some evidence that physical activity can minimize weight gain¹⁵³⁻¹⁵⁵. However, the level of physical activity needed is not well known.

•

and the second of the second second

The main objective of this thesis was to study selected aspects of the weight gaining epidemic in the HUNT population, as documented by Midthjell *et al*⁴⁴. Especially, we wanted to study the role of leisure time physical activity in the weight gaining epidemic. In addition, we wanted to study the association between change in body weight and the effect on blood pressure and mortality.

More specific, we aimed at these objectives:

- To study the association between different levels of leisure time physical activity and change in body mass index during 11-year follow-up in apparently healthy men and women with normal body weight at baseline.
- To study the association between change in body mass index and its impact on systolic and diastolic blood pressure at population level among healthy women and men aged 20 years or older. We wanted especially to separate the effect of initial and attained BMI, and the independent effect of change in BMI on blood pressure.
- To study weight change and mortality among apparently healthy men and women.

3. Material

3.1 The Nord-Trøndelag County

The Nord-Trøndelag County is located in the middle of Norway (Figure 3). The population of Norway was in 1984 about 4.1 million¹⁵⁶ and increased to 4.4 million in 1995¹⁵⁷. The Nord-Trøndelag population was about 127,000 individuals in 1984 and 127,500 in 1995. The population was, and is still today, relative stable and the geographical, demographic and occupational structure is fairly representative of Norway. The county has coast and inland areas, but lacks densely populated areas and larger cities with over 50,000 residents. The level of education and income is lower than the national average. In accordance with these limitations, extensive generalisation to Norway should be limited¹⁵⁸.

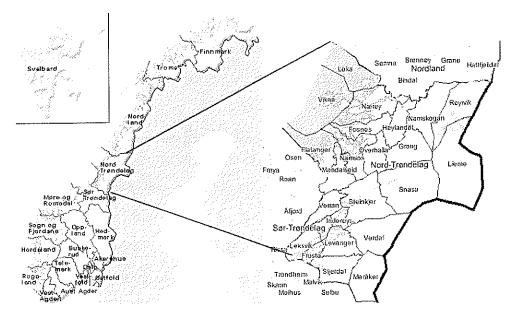


Figure 3. The Nord-Trøndelag County, Norway.

3.2 The Nord-Trøndelag Health Study

The first survey (HUNT 1)

The first survey in Nord-Trøndelag was conducted in 1984-86. All citizens in the county aged 20 years or older by Dec. 31st 1983 were invited (n=85,100 persons) and 88.1% participated¹⁵⁸. The survey consisted of four main studies including hypertension, diabetes, quality of life and lung diseases. The invitation letter was mailed to each participant and included questionnaire 1 which was delivered at attendance (Appendix 1.1). Questionnaire 2 (Appendix 1.2) was delivered at the survey station and returned by mail. Attached to questionnaire 2 was a specific questionnaire for those who self-reported diabetes and/or hypertension treatment (Appendix 1.3). The survey was carried out by employees of the National Health Screening Service, and they were organised in two survey teams. Each team consisted of five nurses and two technicians. All measurements were standardised. The participation rates were highest among middle aged people¹⁵⁹. Women younger than 65 years had a higher participation rate than men in the same age group (Figure 4). Among those who were 65 years or older, men had the highest participation rates.

The second survey (HUNT 2)

The second cross-sectional survey in Nord-Trøndelag was partly a follow-up study of HUNT 1 and was conducted in the two-year period from 1995-97. HUNT 2 comprised, however, a larger scientific program. All aged 13 years and older were invited (n=94,194), and in the age group 20 years or older 66,140 participated

(71.2%)¹⁶⁰. An information folder together with the invitation letter was sent by mail attached to questionnaire 1 (Appendix 2.1), which was to be completed prior to the screening and delivered at attendance to the screening site. A second questionnaire (Appendix 2.2) was handed out at the screening site and was completed and returned by mail. Another questionnaire was handed out for those who self-reported diabetes and/or hypertension treatment (Appendix 2.3). All analyses at the survey station were performed standardised by special trained nurses. In total, more women than men participated in the survey¹⁶⁰. Women younger than 70 years had a higher participation rate than men in the same age group (Figure 4). Among those who were 70 years or older, men had the highest participation rates. The Young-HUNT Study aimed at age group 13-19 years was organised separately. In this thesis we did not include data from the Young-HUNT Study.

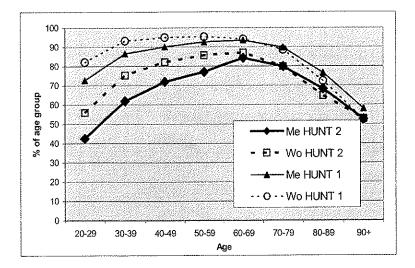


Figure 4. Participation at HUNT 1 and HUNT 2 by age in men and women¹⁶⁰.

Linkage of data

Every citizen of Norway is given a unique 11-digit personal identification number at birth, enabling linkage between data from both HUNT 1 and HUNT 2, and between different health- and register data sets in Norway. The use of identification number is strictly regulated through the Norwegian Data Inspectorate. A linkage was done between the first and the second survey for those who participated in both HUNT 1 and HUNT 2. In addition those who participated twice were linked to the Death Registry at Statistics Norway. Person-years and reasons of death were calculated from the date of the clinical examination until the date of death or until Dec. 31, 2001.

Statistics Norway is administratively placed under the Ministry of Finance and has its own government-appointed board. The most critical aspect in the Death Registry at Statistics Norway is the cause of death reported by the physicians. In 65

per cent of all deaths in 2002, the only sources of information were the death certificate. The main groups, where the underlying cause of death is based on this information are diseases in the genitourinary system, the respiratory system, diseases in the skin and the subcutaneous tissue and the circulatory system. The Death Registry is cooperating with The Cancer Registry and The Medical Birth Registry of Norway with extending information about diagnoses. The Death Registry is regulated by Norwegian law.

3.2.1 Study population

The study population in each paper was selected from individuals who participated in both surveys, but different selection criteria were introduced depending on the objective of the paper, and based on knowledge about possible mechanisms involved (Figure 5).

Paper I and paper II:

The objective of these papers was to investigate the association between leisure time physical activity and change in BMI. We wanted to analyse men and women separately because of gender differences both in aspects of physical activity and body weight (see also chapter 1). Physical fitness and heart rate is inversely correlated¹⁶¹, and it is shown that the measured heart rate was inversely linked to self-reported leisure time physical activity also in the Nord-Trøndelag Health Study¹⁶². Therefore, we wanted to use an inverse association of dose-response between leisure time physical activity and heart rate at HUNT 1 as inclusion criterion. Heart rate was therefore used to validate of the information of leisure time physical activity, and of our stratification of leisure time physical activity at the first survey in different strata

of age. This explains why women 20-49 years and men aged 20-69 years were included in paper I and paper II, respectively (Figure 6). We wanted to include a relative homogenous population with a potential of being physical active, i.e. with no obvious obstacles against physical activity, like disabling diseases and obesity/overweight. The selection criterion of weight range was based on knowledge from other studies, which had shown that overweight and obese persons often overreported their physical activity level¹⁶³. Other criteria for selection of the normal weight group were that overweight and obesity may be a barrier to a physical active lifestyle¹⁶⁴, and additionally we wanted to study the potential primary prevention effect of leisure time physical activity on changes in body weight. Both diabetes, dysfunctions in daily life and cardiovascular diseases may be associated with changes in body weight or leisure time physical activity level, or both. Therefore, based on these arguments we included apparently healthy men and women with normal body weight and without diabetes, CVD and dysfunctions (psychological and physical impairements) in daily life aged 20-49 years and 20-69 years, respectively, to study the association between leisure time physical activity and change in body weight.

Paper III:

In this paper we wanted to investigate the association between change in BMI and change in blood pressure in apparently healthy men and women. Because of the linkage between both change in blood pressure and change in BMI and different diagnoses, we excluded those reporting dysfunction in daily life, CVD or diabetes. In addition we wanted to use exclusion, not adjustment, for use of blood pressure medication to increase homogeneity of the study population.

Paper IV:

We wanted to investigate the association between weight change in apparently healthy men and women and mortality. Because both cardiovascular disease and diabetes may be linked to both change in body weight and mortality, we excluded those who reported cardiovascular disease and diabetes at the first survey.

٠

and the second second w

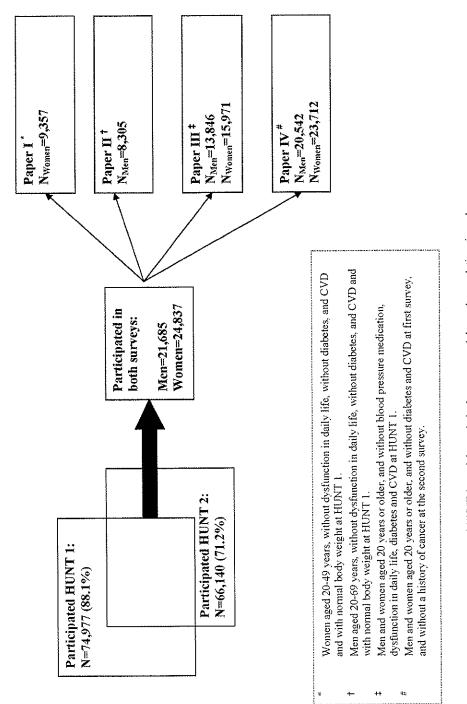


Figure 5. Participation in HUNT 1 and HUNT 2, participants in both surveys, and the study populations in each paper.

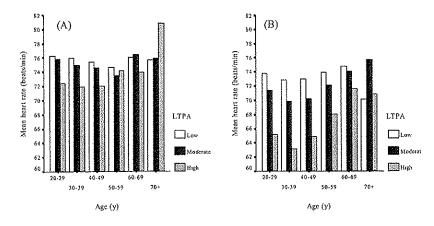


Figure 6. The association between leisure time physical activity (LTPA) and heart rate at HUNT 1 for different age-ranges for women (A) and men (B).

3.3 Variables

The first survey (HUNT 1) aimed at the study of diabetes, hypertension, tuberculosis and other lung diseases and quality of life. The main objectives of the second survey (HUNT 2) were the large public health issues like cardiovascular disease, diabetes, obstructive lung disease, osteoporosis and mental health. Exposure data in both surveys was collected from three sources; clinical data was obtained at survey stations, the self-administered questionnaires obtained information about demographic characteristics, self-reported diseases and lifestyle factors, and blood samples. To select factors to be included in our analyses, we used data from Seidell and Flegal as a basis¹⁶, in addition to consider potential physiological mechanisms involved. We used selected comparison groups by classification of the subjects into different exposure categories¹⁶⁵.

Age is a proxy measure for biological aging¹⁶⁶, and should always be considered. Therefore, we included age in all analyses, and it may also be important

to include age simply because some might not trust results that are not age adjusted². Age at HUNT I was defined as age at syntax data, i.e. the date of control of punched data, performed 7-21 days after the actual screening date. Age at HUNT 1 was thus defined as a mean of 14 days later than the actual date of screening. We used age either as a continuous or categorised variable.

3.3.1 Study variables

Body mass index

At both surveys weight (kg) and height (cm) were measured standardised without shoes, jacket or outdoor garments by special trained personnel¹⁶⁷. Body mass index (Quetlet's index) was calculated as body weight in kilogram (kg) divided by body length in meter (m) squared (kg/m²). We categorised change in BMI between the surveys into three categories: decreased, stable, and increased in paper III, and loss, stable, and gain in paper IV. A stable BMI was defined as +/-0.1 kg/m²/follow-up year according to Nilsson *et al*¹⁶⁸. In addition, we categorised BMI applying the World Health Organisation's (WHO) recommendation¹ (underweight: BMI

Leisure time physical activity (Appendix 1.2)

Men seem to be more physical active than women¹⁶⁹, and therefore we made gender specific categorisation of low, moderate and high levels of leisure time physical activity.

At HUNT 1 leisure time physical activity was self-reported by three questions about frequency, duration and intensity, each with five, four and three possible answers, respectively. Only those who had a frequency once a week or more answered the questions about intensity and duration. We categorised leisure time physical activity into low, moderate and high levels based on the questions about frequency, intensity and duration: A frequency of never or less than once a week was categorized as low. For those with a frequency of once a week or more, a summary was calculated by adding the values of frequency, intensity and duration. The sum value was then divided into moderate and high by dichotomizing at the median value. The median value was included in the moderate level to separate the most active part of the population in the high level group.

In paper II the population was additionally dichotomised into physically active or not physically active by defining low level of leisure time physical activity as not physical active. Participants with moderate and high levels were categorised as physically active. For the physically active part of the study population the intensity was dichotomised into low and high intensity with answer 1 as low and answers 2 and 3 as high by using self-reported breath status of the physical activity as cut-point.

Systolic and diastolic blood pressure (SBP and DBP)

At both surveys, blood pressure was measured by specially trained nurses or technicians with the participant sitting with the arm resting on a table at heart-level. SBP and DBP were read off to the nearest 2 mm Hg. At the first survey, the blood pressure was measured twice on a sphygmomanometer, and the second reading was used in our analyses¹⁷⁰. At the second survey, blood pressure was measured three times using a Dinamap 845XT (Criticon, Tampa, FL) based on oscillometry, and we

used the mean value of the second and third measurement in our analyses¹⁷⁰. At the first survey the cuff-size was equal for all participants (15 cm x 55 cm), but at the second survey the cuff-size used was based on the arm circumference (arm \leq 24 cm: cuff 12 cm x 37 cm, arm 25-35 cm: cuff 15 cm x 50 cm, arm \geq 36 cm: cuff 17 cm x 60 cm). In paper III hypertension was defined as both systolic blood pressure \geq 140 mm Hg and diastolic blood pressure \geq 90 mm Hg. We calculated change in SBP and DBP between the surveys by subtracting SBP and DBP at the second survey from SBP and DBP at the first survey for each individual.

Mortality

ICD-9 and ICD-10 codes (ICD=International Classification of Diseases) were used for identification of cause specific mortality in paper IV.

3.3.2 Potentially confounding variables

Lifestyle variables (Appendix 1.2)

In paper I-III we classified information about smoking into daily or not daily smoking. In paper IV we categorised the information of smoking into never, current and former. Alcohol use was categorised into three categories (abstained, not drinking last 14 days, drinking last 14 days) or four categories (none, 1-4 times, \geq 5 times, teetotaller) based on data on frequency of drinking the past two weeks. To investigate confounding effects of leisure time physical activity we used either active or not (yes/no), or low, moderate, and high levels of leisure time physical activity.

Demographic variables (Appendix 1.2)

Marital status was divided into married, unmarried, widow/widower (widowed in paper I-III), divorced/separated.

Socioeconomic variables (Appendix 1.2)

Education was categorised as ≤ 12 years at school, >12 years at school in paper I and paper II, or ≤ 9 years at school, 10-12 years at school and > 12 years at school in paper III. In paper IV education was categorised in four categories: middle school, high school, <4 years of college/university, ≥ 4 years of college/university). Difference in categorisation did not change the results.

Anthropometric variables

When BMI was considered as a potential confounder, we used BMI as a continuous or categorised variable.

Medical and other physiological variables

Heart rate was used as a continuous variable or in quartiles. In paper IV SBP was used in quintiles. Status of blood pressure medication use was dichotomised (y/n) both in the data from HUNT 1 and HUNT 2 (Appendix 1.2 and 2.1).

4. Methods

4.1 Study design

In this thesis we used a prospective cohort design. Generally, in a prospective cohort study a group of individuals is selected at random from a defined population. Then, after the cohort is selected, baseline information is collected, and the individuals are followed over a time to identify the incidence or risk of disease between those exposed and not exposed to the factors at interest. The Nord-Trøndelag Health Study (HUNT 1 and HUNT 2) gave the possibility to investigate the association between change in body weight and health with a prospective design by using HUNT 1 as baseline.

4.2 Statistical analyses

We used multivariable regression methods (linear, logistic, and Cox regression) to investigate the role of chance. The type of regression model depends on the distribution of the independent variable (Y).

In general, regression is a robust procedure and may be used in many situations where the assumptions are not met, as long as the measurements are fairly reliable and the appropriate regression model is used¹⁷¹.

In epidemiological studies multivariable regression modelling gives the opportunity to include and control for variables with potential confounding effects. A confounder is a factor that is both associated with the exposure and, independent of that, is a risk factor for disease¹⁷². In paper I and II we used the magnitude of change

in the estimates and p-values as criteria for the variables to be included or excluded in the regression models. In paper III the inclusion of variables in the regression model was solely based on the magnitude of change in the estimated associations. We considered a change of 10-15% in the estimates as important. The evaluation of whether a variable qualified as a confounder was done by using the magnitude of discrepancy between the multivariable adjusted estimates and age-adjusted estimates. In paper IV we conducted multivariable analyses to assess potential confounding. All analyses were performed using SPSS for Windows (SPSS Inc. Illinois, US, version 11.0).

All analyses were done stratified by gender independent of statistical significance of interactions, because it is biologically plausible that gender differences are present in all the main associations under study here. Additionally, the main knowledge of health from epidemiological studies has often been based on studies of men, and an extrapolation of these results may not apply to women.

Linear regression

Linear regression attempts to model the relationship between two variables by fitting a linear equation to observed data. One variable is considered to be an explanatory variable or independent variable (X), and the other is considered to be a dependent variable (Y).

In paper I and paper II we used linear regression to investigate the association between change in BMI and baseline leisure time physical activity, and we used BMI at the second survey as the dependent variable (Y) and leisure time physical activity as the independent variable (X), adjusted for BMI at the first survey. In paper III we used linear regression to investigate the association between change in systolic and

diastolic blood pressure (Y) and change in BMI (X). Additionally, we used linear regression to investigate the association between systolic and diastolic blood pressure (Y) and BMI at HUNT 2 categorised (X) in strata of BMI at HUNT 1. Some methodological considerations should be done when linear regression is selected:

- > Linearity (average value of Y for a given value of X is a linear function of X)
- > Homoscedasticity (the variance of Y for a given X is the same for all Xs)
- Normality (for each value of X, the Y variable is assumed to have a normal distribution).

The linearity was investigated by bivariate scatter plots for each independent variable. The model assumptions were tested by using scatter plots of residuals against independent variables (X's), further by using histogram with frequencies plotted against residuals, and in addition by consideration of probability-probability plots (P-P plots). We checked visually for the fit of the theoretical distribution to the observed data by examining the P-P plot where the observed cumulative distribution function is plotted against the theoretical cumulative distribution function. If the theoretical cumulative distribution approximates the observed distribution well, then all points in this plot should fall onto the diagonal line.

Logistic regression

Logistic regression is commonly used when the independent variables include both numerical and nominal measures and the dependent variable is binary (dichotomised). The logistic model gives the probability that the outcome (dependent variable) occurs as an exponential function of the independent variables. A chi-square test is used to determine whether a variable adds significantly to the prediction of the outcome. We were not interested in prediction, and therefore we focused our model building on the

changes in the estimated associations. Logistic regression requires no assumptions about the distribution of the independent variables. The regression coefficients in logistic regression denote the magnitude of change in the log odds produced by one unit change in the value of the independent variable. The logistic regression coefficients are odds ratios which are estimates of the relative risks (RR) under some conditions. We used logistic regression in the paper III, where elevated blood pressure or not, according to our definition, was the binary dependent variable (Y) and change in BMI categorised was the independent variable (X).

Survival analysis

Survival analysis is concerned with studying the time between entry into a study and a subsequent event. Cox proportional hazards regression is a statistical technique for exploring the relationship between survival and explanatory variables. The main purpose of the Cox model is to simultaneously explore the effects of several variables on survival, and this is a robust mathematical model. The Cox method assumes that the values of all covariates are determined at the point when follow-up began on each subject, and that these values do not change over the period of observation¹⁷³. We used a graphical procedure to evaluate if the different variables were constant during the follow-up period. Parallel ln(-ln) curves indicated a constant proportional hazard during the follow-up.

4.3 Ethics

Both surveys were approved by the Norwegian Data Inspectorate. In addition the Regional Ethical Committee for Medical Research (which was not established when HUNT 1 was performed) approved HUNT 2. The participants were informed about

the nature of the study in the invitation, and signed a personal consent. Participants may withdraw from the study and refuse any of their data at any time. In the data files available for scientific actions, name of each individual and the 11-digit person number have been removed to provide anonymity.

4.3 Funding

This thesis has been completed while I have been receiving a research fellowship from the Norwegian Research Council.

The planning, data collection and primary analysis of the first survey (HUNT 1) were funded by the following institutions: The Norwegian Research Council for Science and the Humanities, The Department for Health and Social Affairs, The Nord-Trøndelag County Council, The National Institute of Public Health.

The data collection of the second survey (HUNT 2) was a financial collaboration between HUNT Research Centre (previously a part of the Norwegian Institute of Public Health), Department of Public Health and General Practice, Faculty of Medicine, Norwegian University of Science and Technology (NTNU), Verdal, Norwegian Institute of Public health, Oslo, Nord-Trøndelag County Council, and Innherred Hospital, Levanger.

5.1 Review of Paper I

Leisure time physical activity and change in body mass index. An 11-year followup study of 9,357 normal weight healthy women 20-49 years old. The Nord-Trøndelag Health Study (HUNT), Norway, 1984-86 and 1995-97.

Wenche B. Drøyvold, Jostein Holmen, Øystein Krüger, Kristian Midthjell

Objective: To study the association between self-reported leisure time physical activity at baseline (1984-86) and change in body mass index (BMI) during an 11 year follow up period. The study population was 9,357 healthy women, aged 20-49 years who had a normal body weight (BMI 18.5-24.9 kg/m²) at baseline. *Methods:* A general population based health survey was performed both in 1984-86 (HUNT 1) and 1995-97 (HUNT 2). Leisure time physical activity at HUNT 1 was categorized into high, moderate and low levels based on self-reported intensity, duration and frequency. Women, who at baseline reported diabetes, stroke, angina, and myocardial infarction and/or long-term illnesses impairing their activities of living, were excluded.

Results: Physical activity was a significant predictor of BMI at HUNT 2 adjusted for BMI, age and education at HUNT 1. Low level of leisure time physical activity compared to high level at baseline was significantly associated with a higher BMI 11

years later. Those with high level of activity gained 0.18 kg/m^2 (95 % CI: 0.05, 0.32) less than low level of physical activity over 11 years.

Conclusion: This study has demonstrated that leisure time physical activity had a moderate effect on BMI. However, not even high level of leisure time physical activity was sufficient to prevent weight gain and BMI increased in all subgroups of the study population.

5.2 Review of Paper II

BMI change and leisure time physical activity (LTPA). An 11-y follow-up study in apparently healthy men aged 20-69 with normal weight at baseline. The Nord-Trøndelag Health Study (HUNT), Norway.

Wenche B. Drøyvold, Jostein Holmen, Kristian Midthjell, Stian Lydersen

Objective: To study the association between self-reported leisure time physical activity (LTPA) at baseline and change in body mass index (BMI). Design: Prospective observational study with 11-year follow-up period. Setting: A total population based health survey in one county was performed in 1984-86 (HUNT 1) and repeated in 1995-97 (HUNT 2). *Participants:* 21,685 men participated in both surveys. In the present study we included only apparently healthy 20-69 years old men participating in both surveys and who had a normal body weight (BMI 18.5-24.9 kg/m²) at baseline – leaving 8,305 men for the analyses. 6,945 men answered all questions about leisure time physical activity and 6,749 men had complete data in the multivariate analyses. *Measurements and main results:* At HUNT 1, the participants answered questions

(self-reported) about intensity, frequency and duration of LTPA. The association between change in BMI and LTPA was investigated in multivariate linear regression analyses. Adjusted for smoking, education, age and BMI at baseline the physical active cohort gained less weight than the inactive cohort. Low, moderate and high levels of LTPA showed a U-shaped effect adjusted for smoking, education, age and

BMI at baseline. Adjusted for BMI and age at baseline the high intensity part of the physical active cohort gained less weight than the low intensity group. *Conclusion:* This study has demonstrated a moderate BMI effect of LTPA at population level; however, even high level of LTPA did not prevent weight gain during the 11 year follow-up period.

an an an an an an an an Allanda a Allanda an Al

5.3 Review of Paper III

Change in body mass index and its impact on blood pressure.

A prospective population study.

The Nord-Trøndelag Health Study (HUNT), Norway

Wenche B. Drøyvold, Kristian Midthjell, Tom Ivar Lund Nilsen, Jostein Holmen

Background: Overweight and obesity increase the risk of elevated blood pressure, but the knowledge of the effect of weight change on blood pressure is sparse. *Objective:* To investigate the association between change in body mass index (BMI) and change in diastolic blood pressure (DBP), systolic blood pressure (SBP), and hypertension status.

Design: Two population based cross-sectional studies, one in 1984-86 and the other in 1995-97.

Setting: The Nord-Trøndelag Health Study (HUNT).

Participants: We included 15,971 women and 13,846 men who were 20 years or older at the first survey, without blood pressure medication at both surveys and without diabetes, cardiovascular disease or dysfunction in daily life at baseline. *Measurements:* Weight, height and blood pressure were measured standardised. Change in BMI was categorised as stable (initial BMI +/- 0.1 kg/m² each follow up year), increased or decreased, and BMI was categorised by using World Health Organisation's categorisation (underweight BMI: <18.5 kg/m², normal weight BMI: 18.5-24.9 kg/m², overweight BMI: 25.0-29.9 kg/m², obesity BMI≥30 kg/m²).

Results: An increase in BMI and a decrease in BMI were significantly associated with increased and decreased SBP and DBP, respectively, compared to a stable BMI in both genders and all age groups, although the strongest effect was found among those who were 50 years and older. The adjusted odds ratio (OR) for having hypertension at HUNT 2 was 1.8 (95% Cl: 1.5, 2.2) among women and 1.6 (95% Cl: 1.4,1.8) among men aged 20-49 years who increased their BMI compared to those who had stable BMI. A similar, but weaker association was found among women and men aged 50 years or more. Mean change in both SBP and DBP was higher for those who changed BMI category from first to the second survey than for those who were in the same BMI class at both surveys.

Conclusions: Our result supports an independent effect of change in BMI on change in SBP and DBP in both women and men, and that people who increase their BMI are at increased risk for hypertension.

5.4 Review of Paper IV

Weight change and mortality.

The Nord-Trøndelag Health Study (HUNT), Norway

Wenche B. Drøyvold, Kristian Midthjell, Stian Lydersen, Peter Nilsson, Jan-Åke Nilsson, Tom Ivar Lund Nilsen, Jostein Holmen

Background: The prevalence of obesity is increasing worldwide, and overweight and obese people have increased mortality compared to normal weight people. We have prospectively investigated the effect of weight change on mortality. *Methods:* We utilized data from two large population-based health studies in Nord-Trøndelag, Norway, the first conducted in 1984-86 and the second in 1995-97. A total of 20,542 men and 23,712 women aged 20 years or more were followed-up on allcause mortality for five years after the second survey. Cox proportional hazards models were used to calculate mortality rate ratios (RRs) with 95% confidence intervals (CIs) between people with a stable weight and people who lost or gained weight.

Results: We found no association between weight gain and mortality, but people who lost weight had a higher total mortality rate compared to those who were weight stable (RR was 1.6 (95% CI: 1.4, 1.8) in men and 1.7 (95% CI: 1.5, 2.0) in women). Similar associations were also found for cardiovascular and non-cardiovascular mortality. Additional analysis showed a linear increase in mortality rates across categories of weight loss for both men and women ($P_{trend} < 0.001$). Moreover, there was a

statistically significant interaction between weight change and initial BMI, but only among men ($P_{\text{interaction}} = 0.001$).

and a second of the

Conclusions: Weight loss, but not weight gain, was associated with increased mortality both among men and women. Although underlying undiagnosed disease is the most plausible explanation for this finding, the similar associations found for total mortality, cardiovascular mortality, and non-cardiovascular mortality makes the causal pathway somewhat enigmatic.

6. General discussion

6.1 Methodological considerations

Epidemiology is the study of the distribution and determinants of disease frequency in human populations. Additionally, through epidemiological methods we can not only measure the occurrence of disease, but even seek to identify the causes of disease by interpreting observed patterns of variation in disease occurrence. However, we are only able to indicate whether associations are present or not. We have to evaluate both the role of chance in the observed association and the potential effect of bias and confounding.

6.1.1 Precision (Lack of random error)

In epidemiological studies sampling error will always be present, because even if all individuals in a population were included, the study subjects could be viewed as a sample of the potential biologic experience of an even broader population². To evaluate and quantify the degree to which chance variability may account for the observations an appropriate test of statistical significance should be performed¹⁷². The p-value is defined as the probability that an effect at least as extreme as that observed could have occurred by chance alone, given that there is truly no relationship between the exposure and disease. The role of chance in the precision, also called sampling error, can primarily be improved by increasing the study size or increasing the study efficiency². A more informative measure of the role of chance is the confidence

interval (CI), where the width of the confidence interval provides information about the variability in the estimates and/or the study size. The wider the confidence interval, the smaller sample size and greater variability.

Because the p-value does not give any information about whether the exposure under study is responsible for the effect or include any information about biologically importance, we have not used the p-values as strict borders. We have also assessed the confidence interval for evaluating the role of chance.

One strength of the HUNT Study is the high number of participants. In this thesis the primary study population was 46,534 individuals participating in both surveys (Figure 5). However, due to selection criteria, the study population in each paper was smaller.

6.1.2 Validity (Lack of systematic error)

The validity of a study is often divided into internal and external validity. The internal validity is defined as the degree to which the results are representative for the particular cohort being studied². External validity is about whether the results are applicable to other populations. HUNT 1 and HUNT 2 were performed in a rural area without large cities and the average education and income was somewhat lower than the average of Norway as a whole. Despite high participation rates and large number of participants, a generalisation should be done with caution. Further, all epidemiological investigations require evaluation of the potential systematic error by considering bias and confounding as alternative explanations of the results¹⁶⁵. Bias may be defined as the error related to the ways the targeted and sampled populations differ which threatens the validity of a study¹⁷⁴. Confounding is generated by factors that are independently associated with both the exposure and the outcome under

investigation². The major advantage of the prospective design is that the risk profile was established before assessment of the outcome. Therefore, any information obtained at baseline is unlikely to be biased by knowledge of the outcome status.

Survival bias

Individuals in a cohort with undiagnosed disease at baseline may be more likely to die during the follow-up period than healthy people, resulting in a cohort of healthy survivors as the length of the follow-up increases. The 11 years between HUNT 1 and HUNT 2 may have introduced losses to follow-up. This may have introduced a reduction of a potential bias linked to undiagnosed disease at baseline, because persons with an undiagnosed and underlying disease at HUNT 1 may have died during the period between the surveys, and thus not being in our study population. In addition a criterion of participation twice with 11 years between the surveys may have caused a "survival of the fittest" effect, which may have resulted in underestimated effects because of the strong association between overweight/obesity and mortality. This may have occurred because those with high body weight at the first survey and with low level of physical activity, and additionally weight gain after HUNT 1 may have died before HUNT 2.

Selection bias

The common element in selection bias is that the relation between exposure and disease differ between those who participated compared to the potentially total population for the study (participants and non-participants). If a selection bias is present the results will be a product of the association investigated and the selection. Some degree of non-participation is common in large population studies. In the

HUNT Study the participation rate in HUNT 1 was 88.1%. Among those aged 30 years or older at HUNT 2 the participation rate was 83.3%. Both in HUNT 1 and HUNT 2 a non-responder study was performed^{159,160}. In paper I and paper II we used selective age-ranges (20-49 years in women, and 20-69 years in men). The total participation rate among women aged 20-49 years in HUNT 1 and HUNT 2 was 90.0% and 80.8%, respectively. The total participation rate in HUNT 1 and HUNT 2 among men aged 20-69 years was 81.0% and 73.4%. In HUNT 1 no association between health status and participation rate was found neither in men nor in women aged under 55 years¹⁵⁹. Among men and women aged 55 years or more there was, however, a weak positive association between bad health and non-participation. In HUNT 2 there was no strong linkage between participation or non-participation and health status in young individuals¹⁶⁰, but old non-participants had significantly more health problems than old participants. In paper I and paper II 16.1% and 15.3% missing data of the variable leisure time physical activity, respectively. However, because of the prospective design it is unlikely that the results found in this thesis were a consequence of a selection bias. In paper III the medication use was based on self-report, but even here it is unlikely that the results are affected by selection effects. In paper IV the codes of deaths are critical. In Norway about 90% of the classification in the ICD-10 range 100-199 is based on the death report from the physicians, and 7.5% of the encoding is a result of autopsy (source: http://www.ssb.no/dodsarsak). This illustrates the importance of high competence of the physicians, but it is unlikely that misclassifications present are selective in relation to weight changing categorisation.

In conclusion, it is unlikely that the results presented in this thesis are a result of selection bias, but estimated effects may have been underestimated due to selection bias.

Information bias

Information bias results from incorrect determination of exposure or outcome, or both¹⁷⁵, and can be separated into differential and non-differential. The usual consequence of non-differential misclassification in the principal exposure variable is attenuation of the measure of effect estimate; that is, bias toward the null. Non-differential misclassification of an individual by exposure status is generally difficult to avoid in observational studies based on self-reported measures of exposure like in the HUNT Study. Non-differential misclassification in a confounding variable limits the ability to control for this confounding in the analysis¹⁷⁶. When misclassification is limited in only one stratum, residual confounding may be concentrated in this stratum and give the erroneous impression of effect modification.

To obtain valid and objective estimates of physical activity in large populations is difficult, because no other methods are useful in practice than selfreporting questionnaires. Even though the possible answers from the leisure time physical activity questions were quantitative, the relative effects between the participants will differ and introduce a variation in the self-reported answer that will be non-differential.

BMI is calculated as body weight divided by body height squared (kg/m²). Even if BMI is rather well correlated with fatness in a general population, BMI does not consider the body fat distribution because only body weight and body height is

included in the formula. A description of body fatness based on BMI will therefore introduce misclassification, which will result in an underestimation of the effects. At the first survey waist circumference was not measured, and we therefore lack data for including change in body proportions in our analyses. Even other factors as bone, muscle mass and even increased plasma volume induced by exercise training may affect the numerator of the BMI equation⁸, and probably give an underestimation of effects. As a consequence of the lack of inclusion of body proportion, our results may be underestimated, because of the importance of the body fat distribution, and not only the amount of body fat mass.

The HUNT Study used special trained nurses and standardised procedures to prevent systematic errors. Therefore it is unlikely that the results in this thesis are a product of systematic errors in the measurements performed by the different nurses.

In paper IV we used data from the HUNT Study and from The Death Registry at Statistics Norway. Standardised procedures can prevent selective misclassifications, but it is hard to administer registries without random misclassifications. Also the Death Registry at Statistics Norway may have some degree of misclassification, but this misclassification is probably not linked to the degree of change in BMI between the surveys, and is therefore of a non-differential nature. We used a prospective design in our studies, which means that information was collected before the 'event' of interest. Therefore it is unlikely that differential misclassification was present in our analysis.

Additionally, in paper I and paper II the association between leisure time physical activity and BMI was probably underestimated, because intra individual changes in physical activity level during follow-up were not taken into account¹⁷⁷. Some individuals may have changed physical activity level during the follow-up from

low to higher level or from high to lower level, which will result in an underestimation of the body weight effects found.

The role of confounding

Confounding occurs in epidemiological research when measured association between exposure and disease occurrence is distorted by an imbalance between exposed and non-exposed persons with regard to one or more other risk factors for the disease. When confounding is present this bias can be corrected provided that the confounding was anticipated and the requisite information gathered¹⁷⁵. Confounding can be controlled for, and the purpose is to achieve homogeneity between study groups. Often used methods to adjust for confounding are multivariable analyses, restriction or stratification. Many confounders may be insufficiently known or are unquantifiable; therefore in some situations a better strategy might be to use restrictions¹⁷⁸. The goal of restriction is to obtain a population segment that is homogeneous in respect of a particular risk factor. Variables that have not been measured accurately, or has not been categorised or modelled in such a way as to fully capture the nature of its relationship to disease and/or exposure, will leave unquantifiable residual confounding¹⁷⁹. A definition of the residual is the difference between the true estimate of effect predicted by the model¹⁷³.

To reduce residual confounding in large population based studies, as the HUNT Study, restriction to people who do not have any of such risk factors might be chosen. One disadvantage of restriction is the need of large sample sizes, but this is often not a problem in large population studies.

In all papers in this thesis we used restriction in the selection of study populations, adjustment for potential confounders and stratified analyses to adjust for confounding.

en al l'autoritation de la composition Composition de la comp

6.2 Evaluation of the results

"It makes little sense to expect individuals to behave differently from their peers; it is more appropriate to seek a general change in behavioural norms and in the circumstances which facilitate their adoption." (Rose, 1982)

A high incidence rate of a disease may reflect a shift of the underlying risk distribution in a population as whole; and a reversal of the shift may be the key to effective reduction of the incidence rate. Small shift of the distribution in either direction might imply a surprisingly large impact on the total burden of disease, because of the involvement of many individuals. As a consequence, a population wide change may be one of the most important ways to help high-risk individuals^{180,181}. Geoffery Rose has discussed prevention as separated into 'high-risk' strategy and 'population' strategy, where 'high-risk' strategy has the focus on individuals at risk. The 'population' strategy is about shifting the whole distribution of exposure in a favourable direction. His conclusion is that both have to be considered, but the main priority should be to discover and to control the causes of disease at population level¹⁰⁰. Other studies have shown the possibility of shifting distribution of cardiovascular risk factors for a whole population over time and thereby to influence the prevalence of corresponding disease states¹⁸¹. Figure 7 illustrates the effect on the distribution of BMI categories by mean BMI increase of 2.5 kg/m² (SD 2.1) (women, paper I) and 1.8 kg/m² (SD 1.7) (men, paper II). All had normal weight at baseline, while only 60 % and 55 % remained in the normal weight group, and about 40 % and 45 % were now categorised as overweight or obese at HUNT 2 in women and men,

respectively. This illustrates how relative small shift in mean BMI at population level may influence on the prevalence of overweight and obesity.

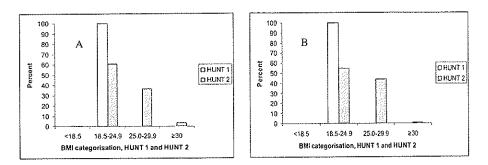


Figure 7. The effect of the distribution of BMI categories by mean BMI increase of 2.5 kg/m2 (SD 2.1) (A: women, paper I) and 1.8 kg/m2 (SD 1.7) (B: men, paper II). All had normal BMI (BMI 18.5-24.9 kg/m2) at baseline (white column). At HUNT 2, 60% of women and 55% of men remained in the normal weight category, while almost 40% women and 45% men were categorised as overweight or obese.

6.2.1 Leisure time physical activity and change in BMI (Paper I, Paper II)

In paper I on leisure time physical activity and change in BMI, including apparently healthy, normal weight women aged 20-49 years, we found that those with high level of leisure time physically activity at baseline gained less weight than those with low level.

In paper II we studied a population of apparently healthy men aged 20-69 years of normal weight in different groups based on information about frequency, intensity and duration of the leisure time physical activity. Those being physically active gained less weight compared to those being inactive. Additionally, in those being physically active, the intensity had a protective effect on BMI increase.

In an overview article Fogelholm & Kukkonen-Harjula¹⁸² concluded that baseline physical activity, i.e. at the beginning of the observation period in population studies, was not particularly linked with the subsequent rate of weight gain. In

general, data from observational studies on the association between leisure time physical activity and change in weight is inconsistent¹³². It is possible that the discrepancy in the observations may be ascribed to the measurement of physical activity, because there is no gold-standard for measuring physical activity. In population studies, as the Nord-Trøndelag Health Study, with high participation numbers, only questionnaires can be used in collecting data on physical activity at leisure. Self-reported leisure time physical activity is a proxy measure of a part of the total energy expenditure. Also in the HUNT Study, measurement of physical activity at leisure might be inaccurate. However, the inverse association between heart rate and leisure time physical activity suggests that the reported leisure time physical activity is valid both among women and men in the selected age-groups. Previous studies have observed a significant discrepancy between objective measures and selfreported measures of body weight and height, where those being overweight or obese often under-report their body weight¹⁸³. Therefore it is important to have standardised measurements of body height and body weight, as in the HUNT Study. The U-shaped association in men between leisure time physical activity and change in BMI may be a result of a discrepancy in the abdominal fat mass between those being low, moderate or highly physical active^{152,184}, because BMI does not differ between muscle and fat mass and body proportions. If those being leisure time physical active at high level had a higher increase in muscle mass compared to those being physical active at lower levels, the health effects of leisure time physical activity in our analysis might be underestimated. The insufficiency of BMI to differ between muscle and fat mass was probably of more importance among men than women, because of the gender differences in muscle mass, and additionally the wish to use physical activity to built muscles was probably higher among men.

One might argue that this moderate reduced weight gain in 11 years (0.3 kg in women and 0.9 kg in men) is hardly a strong motivating factor for physical activity, if the aim is weight control. In modern people weight control is probably a strong motive for being physical active by attending training centres or other forms of sports. There is firm evidence that physical activity is associated with improved physical¹⁸⁵ and mental health^{186,187} and general well-being¹⁸⁸. According to social-cognitive theory, an individual's motivation to engage in physical activity is based on three postulates: self-efficacy, outcome expectations, and self-evaluated satisfaction or dissatisfaction¹⁸⁹. Today one might have the impression that both women and men have unrealistic expectations about the effect of physical activity on the body weight, and short-time improvements are wanted. The importance of combining physical activity with a reduced calorie diet might be undervalued. In our study we had no reliable data on energy intake, but the fact that even those with high level of leisure time physical activity gained weight, demonstrates that the general energy intake in this population was too high to prevent weight gain. On the other hand; even if the weight gain reduction was moderate on the individual level, the potential effect in an apparently healthy and normal weight population should not be underestimated. In women (based on data in paper I) an increase of mean body weigh of 0.3 kg or 0.2 kg/m² is corresponding to a 3% increase in the prevalence of overweight and 0.2% increase in the obesity prevalence [Calculations are based on the following: ((% increase in overweight or obesity/mean weight gain (kg/m²)) multiplied with the mean weight change (kg/m²)]. Based on data in paper II (men) an increase of mean body weigh of 0.3 kg/m² corresponds to 7.3% increase in prevalence of overweight and to 0.1% increase of obesity.

Due to the general high proportion of people trying to lose weight at any time, and because diet seems to be the favourite method, the isolated effect of leisure time physical activity on change in body weight is more difficult to study. In clinical studies¹²⁰ dieting was associated with weight loss followed by regain after intervention, whereas exercise alone produced smaller weight loss but better maintenance. Questions about slimming behaviour were not included in the Nord-Trøndelag Health Study, which may have influenced on our results in view of the factors mentioned above. If those who were physical active at low level tried to lose weight by dieting more frequent than those who were physical active at higher levels, our effects found were probably underestimated due to this circumstances.

Both in paper I and paper II the effects of leisure time physical activity on change in BMI were based on conservative estimates. Taking into account the weight gain reduction effect and the fitness effect, our results may be important in a public health perspective. But as illustrated in paper I, BMI at the first survey explained 41% of the variation in BMI at the second survey. Other variables such as age, education and leisure time physical activity explained only 0.5% of the variance at the second survey. The fact that physical activity at leisure explained only a small part of the variance in BMI at the second survey in a healthy and normal weight population of men and women might be a result of non-differential misclassification, or that the general physical activity is positively related to energy expenditure, but to document effects of physical activity on changes in weight in populations seems to be hard probably because of methodological insufficiency and the complexity of the phenomenon.

en de la terretaria de la companya d

6.2.2 Change in BMI and its impact on blood pressure (Paper III)

Our results confirm findings by others where change in BMI and change in blood pressure was positively associated^{190,191}. In the HUNT Study both blood pressure and body weight and height were measured standardised at both surveys. Different methods were used for blood pressure measurement at HUNT 1 and HUNT 2, with sphygmomanometer and Dinamap, respectively. The change in systolic blood pressure was not significantly affected by this difference in methods, but diastolic blood pressure was measured somewhat lower at the second survey. Therefore, reduction in diastolic blood pressure might be overestimated and increase in diastolic blood pressure might be underestimated¹⁷⁰. Nevertheless, the differences were so small that our results should not be seriously affected by these changes in methods.

Hypertension is also linked to waist circumference¹⁹². Therefore, our results may be underestimated because of potential confounding or effect modification effects on change in waist circumference parallel to changes in weight. This could not be ruled out. We have illustrated that not only high BMI, but also an increase in BMI independent of initial BMI, was associated with blood pressure elevation. Weight gaining women aged 20-49 years and 50+ had 80% and 50% increased risk of having blood pressure \geq 140/90 mm Hg after 11 years, respectively, compared to those being weight stable. Weight gaining men aged 20-49 years and 50+ had 60% and 50% increased risk of having blood pressure \geq 140/90 mm Hg after 11 years, respectively, compared to those being weight stable.

Today about 220,000-322,000 patients are taking blood pressure medication, the government is spending 1 billion NOK each year on blood pressure treatment, and the treatment volume is increasing each year (Irene Hetlevik, personal communication). The prevalence of blood pressure medication use has been rapidly

increasing during the past decades both in women and men¹⁹³. If the obesity epidemic is still increasing in the years to come, an additional increase in the number of blood pressure patients and increase in health care consumption and increased morbidity and mortality due to the obesity is easily predicted (Figure 8).

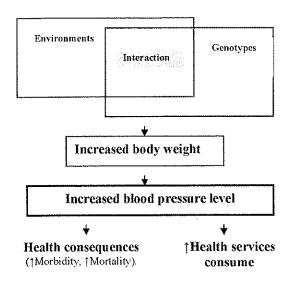


Figure 8. Increased body weight, the association between increased body weight and blood pressure level, and consequences for health and for health care services: Increased body weight gives increased blood pressure level, which in turn is resulting in increased morbidity and mortality, and increased health services consumption. Today I billion NOK is spent on antihypertensive drug treatment alone.

6.2.3 Change in BMI and mortality (Paper IV)

The traditional view is that weight gain is unhealthy and weight loss is healthy, especially in overweight and obese individuals. Our data showed, however, that weight loss was associated with increased mortality. The rate ratios for both total mortality, cardiovascular mortality and non-cardiovascular mortality were

consistently increased for those losing weight. In additional analyses we found no increased rate ratios of sudden death associated with weight loss (data not shown). Neither BMI nor physical activity and smoking explained or modified the results significantly. One might suspect that the findings might be explained by underlying disease. Even if we cannot exclude this possibility, the results were robust and consistent through all analyses and despite all effort to control for underlying disease. Similar results are found in other studies^{125,126,168}. Therefore we conclude that our data support that losing weight is associated with increased mortality. The biological explanation behind the increased mortality at weight loss are not yet established. Results from the SOS Study demonstrated that the initial positive effects on blood pressure associated with weight loss in obese individuals had vanished after eight years¹⁹⁴. In the future when even more events can be included, HUNT data should be reanalysed to study the long-term effects of these associations.

Another striking result was that the data did not show increased mortality among those gaining weight, as one might expect. Given the high number of cases and the confidence intervals, lack of statistical power can hardly explain the results. However, in our study mean follow-up period after the second survey was only five years, i.e. shorter follow-up compared to other studies¹⁶⁸. Some diseases associated with weight gain might be delayed, i.e. it might take many years to erupt, like type 2 diabetes. Cardiovascular diseases might also take many years to develop, though in epidemiological studies effect on mortality is often obvious after only few years. The lack of association between weight gain and mortality in our data is therefore unexplained.

If the results from the present mortality analyses should be confirmed in other populations, one might speculate if weight gain in the 1990-ies probably was more

benign than some decades ago. May be that the total cardiovascular risk profile was lower than previously, and therefore not resulted in increased mortality. Only future populations studies with longer follow-up will answer this question. 1. j. e

6.3 The obesity epidemic in the future

"May be we have to handle the obesity epidemic like driving a car: With full speed ahead there is no good idea to reverse the direction before the car has stopped. (Droyvold, 2004)

Obesity has been recognised as a chronic disease only since 1985, but since then huge resources are spent to achieve better scientific knowledge. So far it seems reasonable to involve more the intellect than the instinct to maintain a healthy weight¹⁹⁵. It is probably unlikely that we will ever return to an environment where body weight regulation can be handed over to the instinct. In future, each individual must probably know which diet balance the energy expenditure, or vice versa, as a prerequisite to achieve a stable body weight. A perspective of this is 'what happens to the health when we have to ignore our instincts throughout life'?

To substantially reduce the number of overweight or obese persons in the world may be a goal totally out of reach in a short-term. The public health response to overweight and obesity is largely based on the individual need to change behaviour, though this approach has generally been ineffective¹⁰⁶. A more feasible public health goal might be to implement weight stability population strategies to stop the weight gaining epidemic. The experience so far is that we do not have much success in getting overweight people maintaining weight loss for a period of time. May be weight loss is not even healthy. Therefore, it is important to put emphasis on

developing methods and policies to prevent weight gain, and probably with children and adolescents as prioritized target groups.

In a public health perspective it is important to consider that during the last 50 years the prevalence has increased within a genetically stable population, suggesting the importance of obesity promoting and inhibitory characteristics of the macroenvironment. As I have shown in this thesis, leisure time physical activity is probably one factor in the puzzle to prevent weight gain, but the effects were not strong, illustrating the complexity of the obesity epidemic. The 'society forces' involved in the obesity epidemic today is so strong, that even high levels of leisure time physical activity in group do not prevent weight gain. It is documented that social factors as family, friends etc. are more important in promoting physical activity in women compared to men. To increase the physical activity level, different strategies in women and men are probably needed.

On a individually level leisure time physical activity is an important factor in energy expenditure. However, in order to identify effects of leisure time physical activity on body weight at population level in epidemiological studies may be more difficult. Of major concern is the risk of developing a vicious circle in which physical activity level decreases as a consequence of higher prevalence of overweight and obesity. There is overwhelming scientific evidence that a physically active lifestyle is one important factor for optimal health¹⁹⁶. Regular physical activity¹⁹⁷ and reduced dietary fat intake¹⁹⁸ reduce weight gain in normal weight subjects and weight regain after weight loss in obese individuals. One challenge is to find what level of physical activity can balance the established environments to prevent weight gain, and to maintain a relative good health at population level. In addition, it is important to understand more about the often found increased mortality associated with weight

loss. One question is: 'Is the increased mortality associated with weight loss only a measure of the unknown and undiagnosed disease at population level present at any time, or are there biological destructive mechanisms involved when the body lose weight?'. It is important to decide if weight loss should be recommended as secondary prevention, as is common practice today, or if weight stability should be focused both in primary and secondary prevention.

6.4 Future research

Generally

Most prospective population studies up to day have not been especially designed to investigate overweight and obesity aspects. In future surveys data should be recorded according to a standard protocol including standardised measurements systems as WHO classification of body weight, and especially anthropometric values made on standardised measurements rather than self-report. Several studies have demonstrated that waist circumference at population level has increased independently of BMI in recent years^{112,199}. In addition, the properties and distribution of fat and the changes in body fat amount, are important arguments for especially including measurements of body proportions, at least waist circumference, to reduce the inadequacies of BMI. Waist circumference has been found to be an independent risk factor for elevated blood pressure^{97,192,200}, and a measure of body proportion should probably be included in most health investigations because of both the mechanical and endocrinological properties of the body fat. The normal weight category defined by WHO is defined to be the most healthy weight category based on previous available mortality-data, but due to the global obesity epidemic the normal weight category is now less common³⁰.

As a consequence, a probably more precise name of the normal weight category is now the healthy weight category. In a pessimistic view, the normal weight category might only be useful as a historical documentation. As the shift of the population is skewed to higher BMI values, new categorisations should probably be constructed.

In general, the physical activity level is decreasing worldwide and the effects of being physical active or not, the interactions, should be investigated in detail in all plausible associations. The trend with decreasing physical activity level and increasing proportion of overweight and obesity, may introduce a vicious circle. As a consequence, an interesting aspect will be what happens to the variance of leisure time physical activity by time. Epidemiological science needs variation within a population to identify relative risk effects. Valid and reliable measurements of physical activity at leisure are necessary, and there is a need to describe more clearly the involvement of intensity, frequency and duration of leisure physical activity associated with outcomes as change in body weight.

The health consequences of weight loss should be further investigated in well designed studies to get closer to the enigmatic association between weight loss and mortality. The degree of intentionality of the weight loss should probably be included, even if valid measure of intentionality is difficult to achieve in epidemiologically population studies based on questionnaires. May be, to study the association between weight loss and mortality in randomised and controlled trials would theoretically be the best design to study this issue. However, because of the high prevalence of people trying to lose weight, and because of ethical considerations, it will be exceedingly difficult to perform such a study. The most practical approach to study weight loss and mortality is probably careful designed observational studies. Even if a considerable part of obesity is due to environmental factors and lifestyle, between 40

and 70% of the variation of BMI is estimated to be heritable²⁰¹. The weight gaining epidemic is multifactorial, and is linked to several genes and environmental factors. Because of the high prevalence of obesity, but also the great variations between individuals and populations, the role of the genetics in human body weight regulation has to be focused. Population based biobanks will probably introduce new ways to study the obesity epidemic. George Bray stated in 1996 that 'genetics load the gun, but the environment pulls the trigger'²⁰². The scientist will be able to include biological material and genetic data in their analyses. One main question why some do not gain weight, while others do, living in almost equal environments.

Genes involved in weight gain increase the susceptibility of an individual to the development of obesity when exposed to environmental conditions that favour a positive energy balance. It is likely that mutations of genes favouring energy storage and metabolic efficiency have conferred a survival advantage to individuals when food supply was scarce and during periods of famine. The combination of an easy access to energy-dense food and a decrease in physical activity has made these genes maladaptive. Thus obesity is most likely a polygenic disease characterised by interactions between genetic and environmental factors. One hypothesis is the 'thrifty genotype hypothesis' which was put forward by James Neel in 1962 ²⁰³ where he postulated that an imbalance in energy could be traced by genotype. The 825T allele associated with the metabolic syndrome has a high prevalence in 'old' ethnicities, e.g. bushmen and Australian aborigines as well as in black populations (80-90%)²⁰⁴, and is one of several genotypes which should be studied in association with phenotypes.

The technological development will likely introduce better methods and more precision in making the disease diagnosis, which may reduce the degree of non-

differential misclassification in epidemiological studies. Consequently, stronger estimates than were found in this thesis might be the result. The technological skills and the medical expertise, and therefore the quality level of a diagnosis, is not equal for all diseases. This might be one good argument for performing 'reproduction studies' of associations considered as 'well known'. High quality end-point registers for a number of diagnoses are a prerequisite for future epidemiological studies. The quality of end-point registries is of vital importance, even in national registries like The Death Registry. There are concerns about the misclassification of the cause of death today, and in the future the problem might even increase due to the reduced capacity for post-mortem autopsies²⁰⁵.

Including foetal life, childhood, and adolescents as well as adulthood is important for performing life-course analyses. Additionally, future data might answer if overweight and obesity in the future has equal impact on public health as today.

More effort should be invested in clinical and controlled intervention studies using comprehensive interventions, as physical activity and diet. One way of introducing changes in diet at population level is to influence and making regulations for the food industry. Prevention actions should be directed at population level as well as at high-risk groups.

In HUNT

In near future the most exiting research area of the HUNT Study will probably be the genetic epidemiology. By including data from the HUNT biobank, analyses of the associations between various genotypes and phenotypes will probably improve our understanding of the obesity epidemic.

The association between weight change and physical activity at leisure and different diseases (such as cancer, osteoporosis and mental health) can also be generated from the HUNT Study.

A third health survey (HUNT 3) is planned in Nord-Trøndelag, and will probably be performed during the period 2006-2008, including all inhabitants in the county aged 10-13 or older, i.e. ca. 105,000 invited individuals. Measurements of body proportion as waist and hip in the whole population and fat-mass measurements in selected groups are needed. In addition, a valid measure of physical activity is important, which can be achieved through validated questionnaires, which often is the only feasible method in population studies. Questionnaires could be combined with objective measurements in sub-samples. (Nanna Kurtze is performing a validation study of the leisure time physical activity questions used in the HUNT Study). Excellent statistics can not align a bad designed study. May be also some smaller substudies with streamlined design could be included in HUNT 3. Additionally, by including children, life-course studies could be conducted.

7. Conclusion

- In apparently healthy women aged 20-49 and men aged 20-69 with normal body weight, leisure time physical activity was associated with change in BMI during an 11-year period. Among women, we found that being leisure time physical active at a high level was associated with less increase in BMI compared to those being leisure time physical active at low level. Among men we found that both being physical active at leisure, and the intensity of the leisure time physical activity, were associated with less increase in BMI compared to those not being physical active (leisure time physical active at low level) and to those being physical active with low intensity, respectively.
- Change in BMI was strongly associated, independent of initial BMI, with change in both systolic and diastolic blood pressure in apparently healthy women and men. The odds ratio of having hypertension at the second survey was positively associated with increased BMI. Additinally, changing BMI category (World Health Organisation's categorisation) from the first to the second survey had a strong effect on systolic and diastolic blood pressure, independently of initial and attained BMI category.
- Weight loss was associated with increased mortality both in apparently healthy women and men. The estimated effects did not change substantially even if initial BMI, smoking status and leisure time physical activity was considered. The effect was consistent for total mortality, cardiovascular mortality and non-

.

cardiovascular mortality. Weight gain was not associated with increased

.

mortality in this study.

References

and the set of the

1. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organ Tech Rep Ser* 2000;**894**:i-XII 1-253.

2. Rothman K, Greenland S. Modern epidemiology. Second edition. Winters R. Philadelphia: Lippincott-Raven 1998.

3. Speakman JR. Obesity: the integrated roles of environment and genetics. *J Nutr* 2004;**134**(8):2090-105.

4. Jequier E, Tappy L. Regulation of body weight in humans. *Physiol Rev* 1999;**79**(2):451-80.

5. Payne PR, Dugdale AE. Mechanisms for the control of body-weight. *Lancet* 1977;1(8011):583-6.

6. Blundell JE, King NA. Overconsumption as a cause of weight gain: behaviouralphysiological interactions in the control of food intake (appetite). *Ciba Found Symp* 1996;**201**:138-54.

7. DiGirolamo M. Cellular, metabolic, and clinical consequences of adipose mass enlargement in obesity. *Nutrition* 1991;7(4):287-9.

8. McArdle WD, Katch FI, Katch, Victor L. Exercise physiology. Energy, Nutrition, and Human Performance. Fifth edition. 2001.

9. Dizar Õ, Alymac E. Obesity: an endocrine tumor? *Med Hypotheses* 2004;63(5):790-2.

10. Loos RJ, Bouchard C. Obesity-is it a genetic disorder? J Intern Med 2003;254(5):401-25.

11. Isomaa B. A major health hazard: the metabolic syndrome. *Life Sci* 2003;73(19):2395-411.

12. Quetelet LAJ. Anthropométrie on mesure des différentes facultés de l'homme. Brussels: C.Muquardt. 1871, pp:479.

13. Keys A, Fidanza F, Karvonen MJ, Kimura N, Taylor HL. Indices of relative weight and obesity. *J Chronic Dis* 1972;**25**(6):329-43.

14. Waaler HT. Height, weight and mortality. The Norwegian experience. Acta Med Scand Suppl 1984;679:1-56.

15. Willet WC. Nutritional epidemiology. New York: Oxford University; 1998.

16. Seidell JC, Flegal KM. Assessing obesity: classification and epidemiology. *Br Med Bull* 1997;**53**(2):238-52.

17. Dishman RK, Washburn RA, Heath GW. Physical activity epidemiology. Bahrke M. Champaign: Human Kinetics. 2004.

18. Baik I, Ascherio A, Rimm EB, Giovannucci E, Spiegelman D, Stampfer MJ et al. Adiposity and mortality in men. *Am J Epidemiol* 2000;**152**(3):264-71.

19. Manson JE, Willett WC, Stampfer MJ, Colditz GA, Hunter DJ, Hankinson SE et al. Body weight and mortality among women. *N Engl J Med* 1995;**333**(11):677-85.

20. Sidney S, Friedman GD, Siegelaub AB. Thinness and mortality. *Am J Public Health* 1987;77(3):317-22.

21. Weisell RC. Body mass index as an indicator of obesity. *Asia Pac J Clin Nutr* 2002;**11 Suppl 8**:681-4.

22. Meyer HE, Søgaard AJ, Tverdal A, Selmer RM. Body mass index and mortality: the influence of physical activity and smoking. *Med Sci Sports Exerc* 2002;**34**(7):1065-70.

23. Jarrett RJ, Shipley MJ, Rose G. Weight and mortality in the Whitehall Study. Br Med J (Clin Res Ed) 1982;285(6341):535-7.

24. Lee IM, Manson JE, Hennekens CH, Paffenbarger Jr RS. Body weight and mortality. A 27-year follow-up of middle-aged men. JAMA 1993;270(23):2823-8.

25. Legato MJ. Gender-specific physiology: how real is it? How important is it? Int J Fertil Womens Med 1997;42(1):19-29.

26. Girardier L. L'auto-regulation du poids et de la composition corporelle chez l'homme. Une approche systemique par modelisation et simulation. *Arch Int Physiol Biochim Biophys* 1994;**102**(4):23-35.

27. Garrow JS. Obeisty and related diseases. London, Churchill Livingstone, 1988:1-16.

28. Hausman DB, DiGirolamo M, Bartness TJ, Hausman GJ, Martin RJ. The biology of white adipocyte proliferation. *Obes Rev* 2001;2(4):239-54.

29. Hodge AM, Zimmet PZ. The epidemiology of obesity. *Baillieres Clin Endocrinol Metab* 1994;8(3):577-99.

30. Schoenborn CA, Adams PF, Barnes PM. Body weight status of adults: United States, 1997-98. *Adv Data* 2002;**330**:1-15.

31. Lemieux S, Prud'homme D, Bouchard C, Tremblay A, Despres JP. Sex differences in the relation of visceral adipose tissue accumulation to total body fatness. *Am J Clin Nutr* 1993;**58**(4):463-7.

32. Engeland A, Bjørge T, Tverdal A, Søgaard AJ. Obesity in adolescence and adulthood and the risk of adult mortality. *Epidemiology* 2004;**15**(1):79-85.

33. Zhang Y, Proenca R, Maffei M, Barone M, Leopold L, Friedman JM. Positional cloning of the mouse obese gene and its human homologue. *Lancet* 1994;**372**:425-32.

34. Friedman JM. The alphabet of weight control. Nature 1997;385(6612):119-20.

35. Sørensen TI, Echwald S, Holm JC. Leptin in obesity. BMJ 1996;313(7063):953-4.

36. Sowers MF, Beebe JL, McConnell D, Randolph J, Jannausch M. Testosterone concentrations in women aged 25-50 years: associations with lifestyle, body composition, and ovarian status. *Am J Epidemiol* 2001;**153**(3):256-64.

37. Overweight, obesity, and health risk. National Task Force on the Prevention and Treatment of Obesity. *Arch Intern Med* 2000;**160**(7):898-904.

38. Stevens J. Obesity, fat patterning and cardiovascular risk. Adv Exp Med Biol 1995;**369**:21-7.

39. Tverdal A. Forekomsten av fedme blant 40-42-åringer i to perioder. *Tidsskr Nor Laegeforen* 2001;**121**(6):667-72.

40. Chou S, Grossman M, Saffer H. An economic analysis of adult obesity: results from the behavioral factor surveillance system. National Bureau of Economic Reaserch Working Paper 9247. National Bureau of Economic Research, Cambridge, 2002.

41. Seidell JC. Time trends in obesity: an epidemiological perspective. *Horm Metab Res* 1997;**29**(4):155-8.

42. Martinez JA, Moreno B, Martinez-Gonzalez MA. Prevalence of obesity in Spain. Obes Rev 2004;5(3):171-2.

43. Jacobsen BK, Njølstad I, Thune I, Wilsgaard T, Lochen ML, Schirmer H. Increase in weight in all birth cohorts in a general population: The Tromsø Study, 1974-1994. *Arch Intern Med* 2001;**161**(3):466-72.

44. Midthjell K, Krüger O, Holmen J, Tverdal A, Claudi T, Bjørndal A et al. Rapid changes in the prevalence of obesity and known diabetes in an adult Norwegian population. The Nord-Trøndelag Health Surveys: 1984-1986 and 1995-1997. *Diabetes Care* 1999;22(11):1813-20.

45. Gardner G, Halweil B. Hunger, escaping excess. World Watch 2000;13(4):25-35.

46. Mokdad AH, Ford ES, Bowman BA, Dietz WH, Vinicor F, Bales VS et al. Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *JAMA* 2003;289(1):76-9.

47. Monteiro CA, Mondini L, de Souza AL, Popkin BM. The nutrition transition in Brazil. *Eur J Clin Nutr* 1995;49(2):105-13.

48. Gill TP, Antipatis VJ, James WPT. The global epidemic of obesity. *Asia Pacific J Clin Nutr* 1999;**8**:75-81.

49. Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960-1994. *Int J Obes* 1998;**22**(1):39-47.

50. Hill JO, Wyatt HR, Reed GW, Peters JC. Obesity and the environment: where do we go from here? *Science* 2003;299(5608):853-5.

51. Keil U, Kuulasmaa K. WHO MONICA Project: risk factors. Int J Epidemiol 1989;18(3 SUPPL 1):46-55.

52. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999-2002. *JAMA* 2004;**291**(23):2847-50.

53. Heini AF, Weinsier RL. Divergent trends in obesity and fat intake patterns: the American paradox. *Am J Med* 1997;102(3):259-64.

54. Nielsen SJ, Siega-Riz AM, Popkin BM. Trends in energy intake in U.S. between 1977 and 1996: similar shifts seen across age groups. *Obes Res* 2002;**10**(5):370-8.

55. Popkin BM. The nutrition transition and obesity in the developing world. *J Nutr* 2001;**131**(3):871-3.

56. Popkin BM, Haines PS, Reidy KC. Food consumption trends of US women: patterns and determinants between 1977 and 1985. *Am J Clin Nutr* 1989;49(6):1307-19.

57. NORKOST 1993-94. NORKOST 1997. Oslo: Statens ernæringsråd.

58. Allison DB, Fontaine KR, Manson JE, Stevens J, VanItallie TB. Annual deaths attributable to obesity in the United States. *JAMA* 1999;**282**(16):1530-8.

59. Thompson D, Wolf AM. The medical-care cost burden of obesity. *Obes Rev* 2001;2(3):189-97.

60. Norges diabetes forbund. Diabetes for 10 milliarder ! Diabetikeren nr. 7 1999.

61. Health implications of obesity. National Institutes of Health Consensus Development Conference Statement. Ann Intern Med 1985;103(6 (PT 2)):1073-7.

62. Bray GA. Medical consequences of obesity. J Clin Endocrinol Metab 2004;89(6):2583-9.

63. Larsson UE. Influence of weight loss on pain, perceived disability and observed functional limitations in obese women. *Int J Obes* 2004;**28**(2):269-77.

64. Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. *N Engl J Med* 1993;**329**(14):1008-12.

65. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. *JAMA* 1999;**282**(16):1523-9.

66. Torgerson JS, Lindroos AK, Naslund I, Peltonen M. Gallstones, gallbladder disease, and pancreatitis: cross-sectional and 2-year data from the Swedish Obese Subjects (SOS) and SOS reference studies. *Am J Gastroenterol* 2003;**98**(5):1032-41.

67. Rocchini AP. Obesity hypertension. Am J Hypertens 2002;15(2 PT 2):50-2.

68. Rosengren A, Wedel H, Wilhelmsen L. Body weight and weight gain during adult life in men in relation to coronary heart disease and mortality. A prospective population study. *Eur Heart J* 1999;**20**(4):269-77.

69. Calle EE, Kaaks R. Overweight, obesity and cancer: epidemiological evidence and proposed mechanisms. *Nat Rev Cancer* 2004;4(8):579-91.

70. Engeland A, Tretli S, Bjørge T. Height, body mass index, and ovarian cancer: a follow-up of 1.1 million Norwegian women. J Natl Cancer Inst 2003;95(16):1244-8.

71. Heitmann BL, Garby L. Composition (lean and fat tissue) of weight changes in adult Danes. Am J Clin Nutr 2002;75(5):840-7.

72. Lauer MS, Anderson KM, Kannel WB, Levy D. The impact of obesity on left ventricular mass and geometry. The Framingham Heart Study. *JAMA* 1991;**266**(2):231-6.

73. Karason K, Wallentin I, Larsson B, Sjostrom L. Effects of obesity and weight loss on left ventricular mass and relative wall thickness: survey and intervention study. *BMJ* 1997;**315**(7113):912-6.

74. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. *N Engl J Med* 1990;**322**(22):1561-6.

75. Henriksson KM, Lindblad U, Gullberg B, Agren B, Nilsson-Ehle P, Rastam L. Development of hypertension over 6 years in a birth cohort of young middle-aged men: the Cardiovascular Risk Factor Study in southern Sweden (CRISS). *J Intern Med* 2002;**252**(1):21-6.

76. Stoll BA. Upper abdominal obesity, insulin resistance and breast cancer risk. Int J Obes 2002;26(6):747-53.

77. Rich-Edwards JW, Spiegelman D, Garland M, Hertzmark E, Hunter DJ, Colditz GA et al. Physical activity, body mass index, and ovulatory disorder infertility. *Epidemiology* 2002;13(2):184-90.

78. Diamanti-Kandarakis E, Bergiele A. The influence of obesity on hyperandrogenism and infertility in the female. *Obes Rev* 2001;2(4):231-8.

79. Rumpel C, Ingram DD, Harris TB, Madans J. The association between weight change and psychological well-being in women. *Int J Obes* 1994;**18**(3):179-83.

80. Quesenberry Jr CP, Caan B, Jacobson A. Obesity, health services use, and health care costs among members of a health maintenance organization. *Arch Intern Med* 1998;**158**(5):466-72.

81. Pi-Sunyer FX. Medical hazards of obesity. Ann Intern Med 1993;119(7 PT 2):655-60.

82. Engeland A, Bjorge T, Selmer RM, Tverdal A. Height and body mass index in relation to total mortality. *Epidemiology* 2003;14(3):293-9.

83. Fontaine KR, Redden DT, Wang C, Westfall AO, Allison DB. Years of life lost due to obesity. *JAMA* 2003;**289**(2):187-93.

84. Peeters A, Barendregt JJ, Willekens F, Mackenbach JP, Al Mamun A, Bonneux L. Obesity in adulthood and its consequences for life expectancy: a life-table analysis. *Ann Intern Med* 2003;**138**(1):24-32.

85. Elmer PJ, Brown JB, Nichols GA, Oster G. Effects of weight gain on medical care costs. *Int J Obes* 2004;**28**(11):1365-73.

86. Visscher TL, Rissanen A, Seidell JC, Heliovaara M, Knekt P, Reunanen A et al. Obesity and unhealthy life-years in adult Finns: an empirical approach. *Arch Intern Med* 2004;164(13):1413-20.

87. Leppala JM, Virtamo J, Fogelholm R, Albanes D, Heinonen OP. Different risk factors for different stroke subtypes: association of blood pressure, cholesterol, and antioxidants. *Stroke* 1999;**30**(12):2535-40.

88. Johansson BB. Hypertension mechanisms causing stroke. *Clin Exp Pharmacol Physiol* 1999;**26**(7):563-5.

89. Lloyd-Jones DM, Larson MG, Leip EP, Beiser A, D'Agostino RB, Kannel WB et al. Lifetime risk for developing congestive heart failure: the Framingham Heart Study. *Circulation* 2002;**106**(24):3068-72.

90. Vasan RS, Levy D. The role of hypertension in the pathogenesis of heart failure. A clinical mechanistic overview. *Arch Intern Med* 1996;**156**(16):1789-96.

91. Levy D, Larson MG, Vasan RS, Kannel WB, Ho KK. The progression from hypertension to congestive heart failure. *JAMA* 1996;**275**(20):1557-62.

92. http://www/legemiddelforbruk.no/.

93. Hall JE, Brands MW, Hildebrandt DA, Kuo J, Fitzgerald S. Role of sympathetic nervous system and neuropeptides in obesity hypertension. *Braz J Med Biol Res* 2000;**33**(6):605-18.

94. Hsueh WA, Buchanan TA. Obesity and hypertension. *Endocrinol Metab Clin* North Am 1994;23(2):405-27.

95. Stamler R, Stamler J, Riedlinger WF, Algera G, Roberts RH. Weight and blood pressure. Findings in hypertension screening of 1 million Americans. *JAMA* 1978;240(15):1607-10.

96. Cassano PA, Segal MR, Vokonas PS, Weiss ST. Body fat distribution, blood pressure, and hypertension. A prospective cohort study of men in the normative aging study. *Ann Epidemiol* 1990;1(1):33-48.

97. Troisi RJ, Weiss ST, Segal MR, Cassano PA, Vokonas PS, Landsberg L. The relationship of body fat distribution to blood pressure in normotensive men: the normative aging study. *Int J Obes* 1990;14(6):515-25.

98. Garrison RJ, Kannel WB, Stokes J 3, Castelli WP. Incidence and precursors of hypertension in young adults: the Framingham Offspring Study. *Prev Med* 1987;16(2):235-51.

99. MacMahon S, Peto R, Cutler J, Collins R, Sorlie P, Neaton J et al. Blood pressure, stroke, and coronary heart disease. Part 1, Prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. *Lancet* 1990;**335**(8692):765-74.

100. Rose G. Sick individuals and sick populations. *Int J Epidemiol* 2001;**30**(3):427-32.

101. Stevens VJ, Corrigan SA, Obarzanek E, Bernauer E, Cook NR, Hebert P et al. Weight loss intervention in phase 1 of the Trials of Hypertension Prevention. The TOHP Collaborative Research Group. *Arch Intern Med* 1993;**153**(7):849-58.

102. Jones DW. Body weight and blood pressure. Effects of weight reduction on hypertension. *Am J Hypertens* 1996;9(8):50-4.

103. Jeffery RW. Public health strategies for obesity treatment and prevention. Am J Health Behav 2001;25(3):252-9.

104. French SA, Story M, Jeffery RW. Environmental influences on eating and physical activity. *Annu Rev Public Health* 2001;22:309-35.

105. Frank LD, Andresen MA, Schmid TL. Obesity relationships with community design, physical activity, and time spent in cars. *Am J Prev Med* 2004;**27**(2):87-96.

106. Hardeman W, Griffin S, Johnston M, Kinmonth AL, Wareham NJ. Interventions to prevent weight gain: a systematic review of psychological models and behaviour change methods. *Int J Obes* 2000;**24**(2):131-43.

107. Crane R. The influence of urban form on travel: an interpretive review. J Planning Lit 2000;15:3-23.

108. Gutierrez-Fisac JL, Lopez Garcia E, Rodriguez-Artalejo F, Banegas Banegas JR, Guallar-Castillon P. Self-perception of being overweight in Spanish adults. *Eur J Clin Nutr* 2002;**56**(9):866-72.

109. Taylor CB, Fortmann SP, Flora J, Kayman S, Barrett DC, Jatulis D et al. Effect of long-term community health education on body mass index. The Stanford Five-City Project. *Am J Epidemiol* 1991;**134**(3):235-49.

110. Jeffery RW, Gray CW, French SA, Hellerstedt WL, Murray D, Luepker RV et al. Evaluation of weight reduction in a community intervention for cardiovascular disease risk: changes in body mass index in the Minnesota Heart Health Program. *Int J Obes* 1995;**19**(1):30-9.

111. Kruger J, Galuska DA, Serdula MK, Jones DA. Attempting to lose weight: specific practices among U.S. adults. *Am J Prev Med* 2004;**26**(5):402-6.

112. Williamson DF, Serdula MK, Anda RF, Levy A, Byers T. Weight loss attempts in adults: goals, duration, and rate of weight loss. *Am J Public Health* 1992;82(9):1251-7.

113. Bendixen H, Madsen J, Bay-Hansen D, Boesen U, Ovesen LF, Bartels EM et al. An observational study of slimming behavior in Denmark in 1992 and 1998. *Obes Res* 2002;10(9):911-22.

114. Serdula MK, Mokdad AH, Williamson DF, Galuska DA, Mendlein JM, Heath GW. Prevalence of attempting weight loss and strategies for controlling weight. *JAMA* 1999;**282**(14):1353-8.

115. Astrup A, Meinert Larsen T, Harper A. Atkins and other low-carbohydrate diets: hoax or an effective tool for weight loss? *Lancet* 2004;**364**(9437):897-9. 116. Freedman MR, King J, Kennedy E. Popular diets: a scientific review. *Obes Res* 2001;**9 Suppl 1**:1-40.

117. Kennedy ET, Bowman SA, Spence JT, Freedman M, King J. Popular diets: correlation to health, nutrition, and obesity. *J Am Diet Assoc* 2001;**101**(4):411-20.

118. Singh RB, Dubnov G, Niaz MA, Ghosh S, Singh R, Rastogi SS et al. Effect of an Indo-Mediterranean diet on progression of coronary artery disease in high risk patients (Indo-Mediterranean Diet Heart Study): a randomised single-blind trial. *Lancet* 2002;**360**(9344):1455-61.

119. The Diabetes Prevention Program. Design and methods for a clinical trial in the prevention of type 2 diabetes. *Diabetes Care* 1999;**22**(4):623-34.

120. Skender ML, Goodrick GK, Del Junco DJ, Reeves RS, Darnell L, Gotto AM et al. Comparison of 2-year weight loss trends in behavioral treatments of obesity: diet, exercise, and combination interventions. *J Am Diet Assoc* 1996;**96**(4):342-6.

121. Methods for voluntary weight loss and control. NIH Technology Assessment Conference Panel. Consensus Development Conference, 30 March to 1 April 1992. Ann Intern Med 1993;**119**(7 PT 2):764-70.

122. Crawford D, Jeffery RW, French SA. Can anyone successfully control their weight? Findings of a three year community-based study of men and women. Int J Obes 2000; 24(9):1107-10.

123. Lemonnier D, de Gasquet P, Mackay S, Planche E, Alexiu A, Rosselin G et al. Different levels of food restriction have opposite effects on adipocyte cellularity and lipoprotein-lipase activity in obese rats. *Diabete Metab* 1989;**15**(6):394-402.

124. Yaari S, Goldbourt U. Voluntary and involuntary weight loss: associations with long term mortality in 9,228 middle-aged and elderly men. *Am J Epidemiol* 1998;148(6):546-55.

125. Newman AB, Yanez D, Harris T, Duxbury A, Enright PL, Fried LP. Weight change in old age and its association with mortality. *J Am Geriatr Soc* 2001;**49**(10):1309-18.

126. Wedick NM, Barrett-Connor E, Knoke JD, Wingard DL. The relationship between weight loss and all-cause mortality in older men and women with and without diabetes mellitus: the Rancho Bernardo study. *J Am Geriatr Soc* 2002;**50**(11):1810-5.

127. French SA, Folsom AR, Jeffery RW, Williamson DF. Prospective study of intentionality of weight loss and mortality in older women: the Iowa Women's Health Study. *Am J Epidemiol* 1999;**149**(6):504-14.

128. Yang D, Fontaine KR, Wang C, Allison DB. Weight loss causes increased mortality: cons. Obes Rev 2003;4(1):9-16.

129. Kassirer JP, Angell M. Losing weight--an ill-fated New Year's resolution. N Engl J Med 1998;338(1):52-4.

130. Yanovski SZ, Bain RP, Williamson DF. Report of a National Institutes of Health-Centers for Disease Control and Prevention workshop on the feasibility of conducting a randomized clinical trial to estimate the long-term health effects of intentional weight loss in obese persons. *Am J Clin Nutr* 1999;**69**(3):366-72.

131. Sarlio-Lahteenkorva S, Rissanen A. Weight loss maintenance: determinants of long-term success. *Eat Weight Disord* 1998;**3**(3):131-5.



132. Erlichman J, Kerbey AL, James WP. Physical activity and its impact on health outcomes. Paper 2: Prevention of unhealthy weight gain and obesity by physical activity: an analysis of the evidence. *Obes Rev* 2002;**3**(4):273-87.

133. Morris J, Heady J, Parks J. Coronary heart-disease and physical activity of work. *Lancet* 1953;**265**(6795):1053-7.

134. Blair SN, Brodney S. Effects of physical inactivity and obesity on morbidity and mortality: current evidence and research issues. *Med Sci Sports Exerc* 1999;**31**(11 SUPPL):646-62.

135. Saris WH, Blair SN, van Baak MA, Eaton SB, Davies PS, Di Pietro L et al. How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. *Obes Rev* 2003;4(2):101-14.

136. Rafamantanantsoa HH, Ebine N, Yoshioka M, Yoshitake Y, Tanaka H, Saitoh S et al. The role of exercise physical activity in varying the total energy expenditure in healthy Japanese men 30 to 69 years of age. *J Nutr Sci Vitaminol (Tokyo)* 2003;**49**(2):120-4.

137. Bernstein MS, Costanza MC, Morabia A. Association of physical activity intensity levels with overweight and obesity in a population-based sample of adults. *Prev Med* 2004;**38**(1):94-104.

138. Hulens M, Vansant G, Lysens R, Claessens AL, Muls E. Exercise capacity in lean versus obese women. *Scand J Med Sci Sports* 2001;11(5):305-9.

139. Matthews CE, Ainsworth BE, Thompson RW, Bassett Jr DR. Sources of variance in daily physical activity levels as measured by an accelerometer. *Med Sci Sports Exerc* 2002;**34**(8):1376-81.

140. Westerterp KR, Meijer GA, Schoffelen P, Janssen EM. Body mass, body composition and sleeping metabolic rate before, during and after endurance training. *Eur J Appl Physiol Occup Physiol* 1994;**69**(3):203-8.

141. Blundell JE, King NA. Physical activity and regulation of food intake: current evidence. *Med Sci Sports Exerc* 1999;**31**(11 SUPPL):573-83.

142. Blundell JE, King NA. Exercise, appetite control, and energy balance. *Nutrition* 2000;**16**(7-8):519-22.

143. Statens råd for ernæring og fysisk aktivitet. Vekt og helse. Rapport nr. 1/2000. Norway. År 2000.

144. Zimmet P, Alberti KG, Shaw J. Global and societal implications of the diabetes epidemic. *Nature* 2001;24(7):782-7.

145. Laaksonen DE, Lakka HM, Salonen JT, Niskanen LK, Rauramaa R, Lakka TA. Low levels of leisure-time physical activity and cardiorespiratory fitness predict development of the metabolic syndrome. *Diabetes Care* 2002;**25**(9):1612-8.

146. Kriska AM, Pereira MA, Hanson RL, de Courten MP, Zimmet PZ, Alberti KG et al. Association of physical activity and serum insulin concentrations in two populations at high risk for type 2 diabetes but differing by BMI. *Diabetes Care* 2001;24(7):1175-80.

147. Martinez ME, Heddens D, Earnest DL, Bogert CL, Roe D, Einspahr J et al. Physical activity, body mass index, and prostaglandin E2 levels in rectal mucosa. *J Natl Cancer Inst* 1999;**91**(11):950-3.

148. Byers T. Body weight and mortality. N Engl J Med 1995;333(11):723-4.

149. Maia JA, Thomis M, Beunen G. Genetic factors in physical activity levels: a twin study. *Am J Prev Med* 2002;23:87-91.

150. Staten MA. The effect of exercise on food intake in men and women. *Am J Clin Nutr* 1991;53(1):27-31.

151. Lofgren P, Hoffstedt J, Ryden M, Thorne A, Holm C, Wahrenberg H et al. Major gender differences in the lipolytic capacity of abdominal subcutaneous fat cells in obesity observed before and after long-term weight reduction. *J Clin Endocrinol Metab* 2002;**87**(2):764-71.

152. Mayo MJ, Grantham JR, Balasekaran G. Exercise-induced weight loss preferentially reduces abdominal fat. *Med Sci Sports Exerc* 2003;35(2):207-13.

153. Haapanen N, Miilunpalo S, Pasanen M, Oja P, Vuori I. Association between leisure time physical activity and 10-year body mass change among working-aged men and women. *Int J Obes* 1997;**21**(4):288-96.

154. Rissanen AM, Heliovaara M, Knekt P, Reunanen A, Aromaa A. Determinants of weight gain and overweight in adult Finns. *Eur J Clin Nutr* 1991;45(9):419-30.

155. Williamson DF, Madans J, Anda RF, Kleinman JC, Kahn HS, Byers T. Recreational physical activity and ten-year weight change in a US national cohort. *Int J Obes* 1993;17(5):279-86.

156. Population by age and marital status 31 December 1984. Oslo, Kongsvinger: Statistics Norway. Official Statistics of Norway, 1985.

157. Population Statistics 1995, Volume 1. Population changes in municippalities 1993-1995. Oslo, Kongsvinger: Statistics Norway, 1995.

158. Holmen J, Midthjell K, Forsen L, Skjerve K, Gorseth M, Oseland A. Helseundersøkelsen i Nord-Trøndelag 1984-86. Fremmøtet og sammenlikning av dem som møtte og dem som ikke møtte. *Tidsskr Nor Lægeforen* 1990;**110**(15):1973-7.

159. Holmen J, Forsen L, Skjerve K, Gorseth M, Midthjell K, Oseland A. Participation-non-participation. The Nord-Trøndelag Health Survey 1984-86. Report no. 5. 1989.

160. Holmen J, Midthjell K, Krüger Ø, Langhammer A, Holmen TL, Bratberg GH et al. The Nord-Trøndelag Health Study 1995-97 (HUNT 2): Objectives, contents, methods and participation. *Nor J Epidemiol* 2003;**13**(1):19-32.

161. Vitartaite A, Vainoras A, Sedekerskiene V, Poderys J. The influence of aerobics exercise to cardiovascular functional parameters of 30-40 year old women. *Medicina (Kaunas)* 2004;**40**(5):451-8.

162. Rangul V. Validitet og reliabilitet på selvrapportert fysisk aktivitet ved bruk av spørreskjema brukt i Helseundersøkelsen i Nord-Trøndelag (HUNT 2-1995-97). En pilotstudie med referanse til spørsmål om fysisk aktivitet videreutviklet fra HUNT 1 (1984-86). Hovedfagsoppgave, Høgskolen i Nord-Trøndelag, Levanger. 2003.

163. Forbes GB. Diet and exercise in obese subjects: self-report versus controlled measurements. *Nutr Rev* 1993;**51**(10):296-300.

164. Ball K, Crawford D, Owen N. Too fat to exercise? Obesity as a barrier to physical activity. *Aust N Z J Public Health* 2000;**24**(3):331-3.

165. Charles H H, Julie E B. Epidemiology in medicine. 1987.

166. Holliday R. The multiple and irreversible causes of aging. J Gerontol A Biol Sci Med Sci [A] Biol Sc 2004;59(6):B568-72.

167. Holme J, Midthjell K, Bjartveit K, Hjort PF, Lund-Larsen PG, Moum T et al. The Nord-Trøndelag Health Survey 1984-86. Puspose, background and methods.Report no. 4 - 1990.

168. Nilsson PM, Nilsson JA, Hedblad B, Berglund G, Lindgarde F. The enigma of increased non-cancer mortality after weight loss in healthy men who are overweight or obese. *J Intern Med* 2002;**252**(1):70-8.

169. Jones DA, Ainsworth BE, Croft JB, Macera CA, Lloyd EE, Yusuf HR. Moderate leisure-time physical activity: who is meeting the public health recommendations? A national cross-sectional study. *Arch Fam Med* 1998;7(3):285-9.

170. Lund-Larsen PG. Blood pressure measured with a sphygmamanometer and with Dinamap under field conditions-a comparison. *Nor J Epidemiol* 1997;7:235-41.

171. Dawson B, Trapp RG. Basic and clinical biostatistics. Fourth edition. 2004.

172. Hennekens CH, Buring JE. Epidemiology in medicine. *Philadelphia: Lippinicott Williams & Wilkins*. 1987.

173. Hosmer DW, Lemeshow S. Applied survival analysis. Regression modeling of time to event data. 1999.

174. James PT, Leach R Kalamara E, Shayeghi M. The worldwide obesity epidemic. Obes Res 2001;9 Suppl 4:228-33.

175. Grimes DA, Schulz KF. Bias and causal associations in observational research. *Lancet* 2002;**359**(9302):248-52.

176. Greenland S. The effect of misclassification in the presence of covariates. *Am J Epidemiol* 1980;**112**:564-9.

177. Andersen LB. Relative risk of mortality in the physically inactive is underestimated because of real changes in exposure level during follow-up. *Am J Epidemiol* 2004;**160**(2):189-95.

178. Vandenbroucke JP. When are observational studies as credible as randomised trials? *Lancet* 2004;**363**(9422):1728-31.

179. Koepsell T, Weiss N. Epidemiologic methods. Studying the occurrence of illness. 2003.

180. Helmchen LA, Henderson RM. Changes in the distribution of body mass index of white US men, 1890-2000. Ann Hum Biol 2004;**31**(2):174-81.

181. Laaser U, Breckenkamp J, Ullrich A, Hoffmann B. Can a decline in the population means of cardiovascular risk factors reduce the number of people at risk? *J Epidemiol Community Health* 2001;55(3):179-84.

182. Fogelholm M, Kukkonen-Harjula K. Does physical activity prevent weight gaina systematic review. *Obes Rev* 2000;1(2):95-111.

183. Flood V, Webb K, Lazarus R, Pang G. Use of self-report to monitor overweight and obesity in populations: some issues for consideration. *Aust N Z J Public Health* 2000;**24**(1):96-9.

184. Ball K, Owen N, Salmon J, Bauman A, Gore CJ. Associations of physical activity with body weight and fat in men and women. *Int J Obes* 2001;25(6):914-9.

185. Manson JE, Rimm EB, Stampfer MJ, Colditz GA, Willett WC, Krolewski AS et al. Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. *Lancet* 1991;**338**(8770):774-8.

186. Craft LL, Perna FM. The Benefits of Exercise for the Clinically Depressed. Prim Care Companion J Clin Psychiatry 2004;6(3):104-11.

187. Lautenschlager NT, Almeida OP, Flicker L, Janca A. Can physical activity improve the mental health of older adults? *Ann Gen Hosp Psychiatry* 2004;3(1):12.

188. Janisse HC, Nedd D, Escamilla S, Nies MA. Physical activity, social support, and family structure as determinants of mood among European-American and African-American women. *Women Health* 2004;**39**(1):101-16.

189. Netz Y, Raviv S. Age differences in motivational orientation toward physical activity: an application of social-cognitive theory. *J Psychol* 2004;**138**(1):35-48.

190. Sonne-Holm S, Sørensen TI, Jensen G, Schnohr P. Independent effects of weight change and attained body weight on prevalence of arterial hypertension in obese and non-obese men. *BMJ* 1989;**299**(6702):767-70.

191. Wilsgaard T, Schirmer H, Arnesen E. Impact of body weight on blood pressure with a focus on sex differences: the Tromso Study, 1986-1995. *Arch Intern Med* 2000;**160**(18):2847-53.

192. Guagnano MT, Ballone E, Colagrande V, Della Vecchia R, Manigrasso MR, Merlitti D et al. Large waist circumference and risk of hypertension. *Int J Obes* 2001;25(9):1360-4.

193. Mosterd A, D'Agostino RB, Silbershatz H, Sytkowski PA, Kannel WB, Grobbee DE et al. Trends in the prevalence of hypertension, antihypertensive therapy, and left ventricular hypertrophy from 1950 to 1989. *N Engl J Med* 1999;**340**(16):1221-7.

194. Torgerson JS, Sjøstrøm L. The Swedish Obese Subjects (SOS) study--rationale and results. *Int J Obes* 2001;25 Suppl 1:2-4.

195. Peters JC, Wyatt HR, Donahoo WT, Hill JO. From instinct to intellect: the challenge of maintaining healthy weight in the modern world. *Obes Rev* 2002;3(2):69-74.

196. Bauman AE. Updating the evidence that physical activity is good for health: an epidemiological review 2000-2003. *J Sci Med Sport* 2004;7(1 SUPPL):6-19.

197. Jakicic JM. The role of physical activity in prevention and treatment of body weight gain in adults. *J Nutr* 2002;**132**(12):3826-9.

198. Astrup A, Astrup A, Buemann B, Flint A, Raben A. Low-fat diets and energy balance: how does the evidence stand in 2002? *Proc Nutr Soc* 2002;**61**(2):299-309.

199. Lahti-Koski M, Pietinen P, Mannisto S, Vartiainen E. Trends in waist-to-hip ratio and its determinants in adults in Finland from 1987 to 1997. *Am J Clin Nutr* 2000;**72**(6):1436-44.

200. Kannel WB, Brand N, Skinner Jr JJ, Dawber TR, McNamara PM. The relation of adiposity to blood pressure and development of hypertension. The Framingham study. *Ann Intern Med* 1967;67(1):48-59.

201. Comuzzie AG, Allison DB. The search for human obesity genes. *Science* 1998;**280**(5368):1374-7.

202. Bray GA. Leptin and leptinomania. Lancet 1996;348(9021):140-1.

203. Neel J. Diabetes mellitus: a "thrifty" genotype rendered detrimental by "progress"? Am J Hum Genet 1962;14:353-62.

204. Siffert W, Forster P, Jockel KH, Mvere DA, Brinkmann B, Naber C et al. Worldwide ethnic distribution of the G protein beta3 subunit 825T allele and its association with obesity in Caucasian, Chinese, and Black African individuals. *J Am Soc Nephrol* 1999;**10**(9):1921-30.

205. Sandvig H, Holthe G. Wrong cause of death in four of ten cases (In Norwegian). http:// www.nrk.no/programmer/tv/puls/3632775.html. 2004.

Paper I-IV

a an tarabén di karang kar Karang karang

.

Paper I i not included due to copyright.

Paper II i not included due to copyright.

Paper III

Change in body mass index and its impact on blood pressure.

A prospective population study.

The Nord-Trøndelag Health Study (HUNT), Norway.

Wenche B. Drøyvold M.Sc.¹⁾, Kristian Midthjell M.D., Ph.D.¹⁾, Tom Ivar Lund Nilsen Ph.D.¹⁾, Jostein Holmen M.D., Ph.D.¹⁾

¹⁾Department of Public Health and General Practice, Faculty of Medicine, Norwegian University of Science and Technology, Norway

Address correspondence to:

Wenche B. Drøyvold

Department of Public Health and General Practice

Norwegian University of Science and Technology

University Medical Centre

N-7489 Trondheim, Norway

 Office phone:
 +47 73 59 88 76

 Office fax:
 +47 73 59 75 77

 E-mail:
 wenche.b.droyvold@medisin.ntnu.no

ABSTRACT

Background: Overweight and obesity increase the risk of elevated blood pressure, but the knowledge of the effect of weight change on blood pressure is sparse.

Objective: To investigate the association between change in body mass index (BMI) and change in diastolic blood pressure (DBP), systolic blood pressure (SBP), and hypertension status.

Design: Two population based cross-sectional studies, one in 1984-86 and the other in 1995-97.

Setting: The Nord-Trøndelag Health Study (HUNT).

Participants: We included 15971 women and 13846 men who were 20 years or older at the first survey, without blood pressure medication at both surveys and without diabetes, cardiovascular disease or dysfunction in daily life at baseline. *Measurements:* Weight, height and blood pressure were measured standardised. Change in BMI was categorised as stable (initial BMI +/- 0.1 kg/m² each follow up year), increased or decreased, and BMI was categorised by using World Health Organisation's categorisation (underweight BMI: <18.5 kg/m², normal weight BMI: 18.5-24.9 kg/m², overweight BMI: 25.0-29.9 kg/m², obesity BMI≥30 kg/m²).

Results: An increase in BMI and a decrease in BMI were significantly associated with increased and decreased SBP and DBP, respectively, compared to a stable BMI in both genders and all age groups, although the strongest effect was found among those who were 50 years and older. The adjusted odds ratio (OR) for having hypertension at HUNT 2 was 1.8 (95% CI: 1.5, 2.2) among women and 1.6 (95% CI: 1.4,1.8) among men aged 20-49 years who increased their BMI

이야지 않는 것 같은 것 같은 것 같은 물건을 많은 것 같이 있는 것 같이 많이 있다.

compared to those who had stable BMI. A similar, but weaker association was found among women and men aged 50 years or more.

The mean change in both SBP and DBP was higher for those who changed BMI category from first to the second survey than for those who were in the same BMI class at both surveys.

Conclusions: Our result supports an independent effect of change in BMI on change in SBP and DBP in both women and men, and that people who increase their BMI are at increased risk for hypertension.

INTRODUCTION

The prevalence of overweight and obesity is increasing worldwide¹, and overweight and obesity are associated with elevated blood pressure^{2,3}. Further, studies have shown a progressive increase in blood pressure with ageing in developed countries. This effect begins in childhood and continues into adulthood⁴ and may be caused by weight gaining by age³. The exact mechanism whereby change in body weight causes elevated blood pressure is still unknown. Hypertension and the risk of having cardiovascular disease is strongly linked^{5,6} and to investigate the effect on blood pressure when the body weight change is important in a public health perspective.

In Nord-Trøndelag County, Norway, two large health surveys were conducted in 1984-86 (HUNT 1) and 1995-97 (HUNT 2), respectively, with information on weight, height and blood pressure. This gave us the opportunity to prospectively examine the association between change in body mass index (BMI) and its impact on blood pressure during 11-year follow-up in a large population of both men and women aged 20 years or more.

METHODS

Study population

In the first health survey (HUNT 1, 1984-86) all citizens residing in the county aged 20 years and older (n=85100) were invited and 74994 participated (88.1%)⁷. In the second health survey (HUNT 2, 1995-97) 66140 adults aged 20 years or older participated (71.2%)⁸. All together 21685 men and 24837 woman participated in both surveys. Information was collected from self-reported questionnaires and a standardised clinical examination. In total, 15971 women and 13862 men without self-reported diabetes, cardiovascular disease (angina pectoris, stroke and myocardial infarction) and dysfunction in daily life in the first survey were included. In addition the participants reported no use of blood pressure medication at the first or at the second survey. Each participant's record was linked to the 11-digit personal identification number, which is unique to every citizen in Norway, enabling a linkage of data from the first and the second survey for each individual.

Standardised measurements (height, weight and blood pressure)

In both surveys, height was measured without shoes to the nearest centimetre, and weight was measured wearing light clothes without shoes to the nearest half kilogram. Similar methods were applied in both surveys and are described elsewhere^{8,9}. Body mass index (BMI) was calculated as body weight in kilogram divided by the squared value of body height in meter (kg/m²). Change in BMI between the surveys was categorised into stable, increased and decreased with stable defined as BMI at first survey +/- 0.1 kg/m² each follow-up year according

to Nilsson et al¹⁰. At both surveys, blood pressure was measured by specially trained nurses or technicians with the participant sitting with the arm resting on a table at heart-level. SBP and DBP were read off to the nearest 2 mm Hg. At the first survey, the blood pressure was measured twice on a sphygmomanometer after the participants had been seated for at least four minutes with the cuff placed on the right upper arm, and we used the second measurement in our analysis. At the second survey, blood pressure was measured using a Dinamap 845XT (Criticon, Tampa, FL) based on oscillometry. The Dinamap was started when the participant had been seated for two minutes with the cuff on the arm. The blood pressure was measured three times, and the mean of the second and third measurement has been used in our analyses. At the first survey the cuffsize was equal for all participants (15 cm x 55 cm), but at the second survey the cuff-size was based on the arm circumference (arm ≤24 cm: cuff 12 cm x 37 cm, arm 25-35 cm: cuff 15 cm x 50 cm, arm \ge 36 cm: cuff 17 cm x 60 cm). Hypertension was defined as having both a systolic blood pressure ≥ 140 mm Hg and a diastolic blood pressure ≥ 90 mm Hg. We calculated change in SBP and DBP between the surveys by subtracting SBP and DBP at the second survey from SBP and DBP at the first survey for each individual.

Statistical analysis

The association between change in blood pressure and change in BMI was investigated using multivariable linear regression with change in SBP and change in DBP as the dependent variable. Additionally, the association between BMI categorisation at the first and the second survey and change in SBP and DBP was investigated by linear regression analysis. Multivariable logistic

regression was used to investigate the association between change in BMI and hypertension (BP \geq 140/90) at the second survey. All analyses included age in one-year categories, and confounding by other factors was evaluated by the magnitude of change from the age-adjusted results. These potentially confounding variables included baseline BMI (continuous), pulse (quartiles), smoking status (daily smoker, not daily smoker), education (\leq 9 years at school, 10-12 years at school, > 12 years at school), leisure time physical active (yes/no), alcohol consumption (abstainer, not drinking last 14 days, drinking last 14 days) and marital status (unmarried, married, separated/divorced and widowed).

The analyses were done separately by gender and stratified by age (50 years as cut-point). All analyses were performed using SPSS, version 11.0 (SPSS, Chicago, Ill, USA).

Ethics

The participation was completely voluntary and each participant signed a written consent. Both studies were recommended by the Norwegian Data Inspectorate. The second study was approved by the Regional Ethical Committee for Medical Research. (At the first survey the Regional Ethical Committee was not yet established).

RESULTS

Men and women who decreased BMI between the surveys had the highest ageadjusted mean BMI, systolic- and diastolic blood pressure at baseline compared to those who were stable in BMI or increased BMI, but at the second survey the situation was reversed (Table 1). In both genders the age-adjusted mean BMI was within the overweight category (BMI: 25.0-29.9 kg/m²) in both surveys.

Women and men who increased BMI had a significantly higher increase in SBP compared to those with a stable BMI (Table 2). Participants with reduced BMI between the surveys had a significant lower increase in SBP compared to those who were stable. The greatest effect on change in SBP was observed among those who lost BMI and were 50 years or older both among women [-5.0 (95% CI: -7.0, -3.0) mmHg] and men [-6.6 (95% CI: -8.6, -4.4) mmHg] with stable BMI used as reference. Women and men who reduced BMI had a lower change in DBP compared to participants who were stable in BMI, but for women aged 20-49 years the association was marginally significant. In both genders and both age-groups, an increase in BMI was associated with significantly higher DBP compared to those who had stable BMI. The greatest effects of weight change on DBP were observed among men aged 50 years or more.

The odds ratio (OR) of having hypertension at the second survey adjusted for age at the first survey was significantly higher in participants who increased their BMI compared to those with a stable BMI (Table 3). These results were not changed after adjustment for BMI at baseline, and further adjustment for hypertension status at the first survey did not change the estimates. The OR for hypertension at the second survey was lower in men who

decreased their BMI compared to those with a stable BMI, both in the age group 20-49 years [multivariable adjusted OR=0.7 (95% CI: 0.5, 1.0)] and in the age group 50 years or older [multivariable adjusted OR=0.6 (95% CI: 0.5, 0.8)]. In women who decreased their BMI between the surveys we found non-significantly reduced OR for hypertension among the oldest [0.8 (95% CI: 0.6, 1.0), and no effect among the youngest [1.0 (95% CI: 0.7, 1.5)] after adjustment for baseline BMI and blood pressure level.

In additional analysis we found that people who advanced from one BMI category to a higher level category between the surveys had a greater change in both SBP and DBP than those who were classified within the same category at both surveys (Table 4). Compared to men who were classified as normal weight at both surveys, men who changed from normal weight to obese had a 4.9 mmHg (95% CI: 1.2, 8.6) higher increase in SBP. Women who changed BMI classification from normal weight to obese had a 7.6 mmHg (95% CI: 5.7, 9.5) higher increase in SBP than those who were normal weight at both surveys. We also found that people who reduced their BMI between the surveys had a significantly lower blood pressure than those who were normal weight at both surveys.

DISCUSSION

In this 11-year prospective population study we found a strong association between change in BMI and change in SBP and DBP both among women and men, but the effect was strongest among the oldest. Further we found that the risk for hypertension at the second survey was linked to change in BMI, but with the strongest effect in the youngest. Additinally, to change BMI category (WHO's categorisation) from the first to the second survey had a strong effect on systolic and diastolic blood pressure, which was independent of initial and attained BMI category.

The exact underlying pathophysiological mechanisms between change in BMI and blood pressure are still not clear. What is known is that weight gain stimulates sympathetic activation, and also that probably insulin and leptin are involved¹¹. Also activation of the renin-angiotensin system as well as physical compression of the kidney may be important factors in linking body weight and elevated blood pressure¹².

Our data confirmed results from previous studies reporting a positive association between change in BMI and change in blood pressure^{13,14}. Additionally, the relation between change in BMI and blood pressure stratified for initial and attained BMI has been previously studied in men by other investigators¹⁵, and our results supported these results, even if different limits for categorisation of BMI was used.

In a large study, Huang *et al*¹⁶ included 82473 US nurses and investigated the association between change in body weight and incident cases of hypertension based on self-report and recall of body weight, and found that weight gain substantially increased the risk for hypertension. In contrast to our

results, a reduced risk of hypertension associated with body weight reduction was found among women.

Additionally, the association between change in body weight and blood pressure has also been investigated in randomized intervention studies. In The Hypertension Optimal Treatment Study, a randomized intervention study among overweight individs, it was reported that weight loss, even after only three months follow-up, was associated with improved blood pressure¹⁷.

One strength of the present study was the large number of both women and men participating in two health surveys, permitting a prospective design with a mean follow-up period of 11 years. Another strength was the standardised and similar methods for measuring body weight and body height at both surveys, because others have found a significant discrepancy between selfreported and objective values for weight and height^{18,19}. However, as described in the method part in this article blood pressure was measured standardised, but not similar, at both surveys. Different methods, cuff-size and cuff-time may have introduced some bias to our results²⁰. However, this bias is likely to be nondifferential, and may thus have underestimated our results.

Another bias to our results could have been that our study population consisted of those who had survived until the second survey and accepted to participate twice. In addition, medication was an exclusion criterion, with the strongest effect in the oldest age groups²¹. Because of the prospective design, it is unlikely that these aspects have influenced considerably on the results.

We had the opportunity to adjust for the potentially confounding effect of different variables, but only baseline blood pressure and BMI had impact on the results. In contrast to the findings among men, women who

decreased in BMI did not reduce the risk of hypertension compared with women with a stable BMI. One possible explanation is the gender differences in fat distribution and adipose tissue metabolism^{22,23}. Additionally, the gender differences in body proportion is also a plausible explanation for why hypertension-estimates were considerably changed by adjustment for initial BMI ecpecially for women aged 20-49 years.

Unintentional weight loss can be considered as marker for underlying disease. In our study participants who decreased their BMI improved their blood pressure, but we were not able to distinguish subjects who intended to lose weight from those who not intended.

Previous studies have shown that waist circumference displays a stronger association with cardiovascular outcomes and risk factors than does BMI²⁴. However, waist circumference was not measured in the first survey, and hence, we were not able to evaluate this association.

Residual confounding due to other unmeasured factors can not be ruled out.

Elevated blood pressure is a risk factor for morbidity and mortality, and odds ratios of 1.9 and 3.7 for coronary heart disease related to elevated blood pressure have been found in non-diabetic and diabetics men, respectively²⁵. Additionally, previous studies have found a reduced incidence of stroke (42%) and myocardial infarction (14%) by lowering diastolic blood pressure with 5 mm Hg²⁶. In a study by Levy *et al*²⁷ hypertension was found to carry the greatest attributable risk at population level for developing congestive heart failure (39% in men and 59% in women). This illustrates the potential for

prevention of morbidity by stabiliting BMI, but also the consequence of not intervening against the obesity epidemic.

In conclusion, our results support a causal association between change in blood pressure and change in BMI in both genders independent of initial and attained BMI. By this, stabilisation or reduction of BMI may serve to prevent or reduce future increase in blood pressure and to reduce the prevalence of hypertension.

ACKNOWLEDGEMENT

The Nord-Trøndelag Health Study is collaboration between HUNT Research Centre, Department of Public Health and General Practice, Norwegian University of Science and Technology, the Norwegian Institute of Public Health and Nord-Trøndelag County. and the second

REFERENCES

 Seidell JC. Obesity: a growing problem. Acta Paediatr Suppl 1999;88(428):46-50.

 Kannel WB. The relation of adiposity to blood pressure and development of hypertension. The Framingham study. *Ann Intern Med* 1967;67(1):48-59.
 Obesity: preventing and managing the global epidemic. Report of a WHO

consultation. World Health Organ Tech Rep Ser 2000;894:i-XII 1-253.

4. Kotchen JM, McKean HE, Kotchen TA. Blood pressure trends with aging. *Hypertension* 1982;4(5 PT 2):III128-34.

5. Kannel WB. Blood pressure as a cardiovascular risk factor: prevention and treatment. *JAMA* 1996;275(20):1571-6.

6. Rosengren A, Welin L, Tsipogianni A, Wilhelmsen L. Impact of cardiovascular risk factors on coronary heart disease and mortality among middle aged diabetic men: a general population study. *BMJ* 1989;299(6708):1127-31.

 Holmen J, Midthjell K, Bjartveit K, Hjort PF, Lund-Larsen PG, Moum T et al. The Nord-Trøndelag Health Survey 1984-86. Purpose, background and methods.
 Participation, non-participation and frequency distributions. Report no 4. 1990;.
 Holmen J, Midthjell K, Krüger Ø, Langhammer A, Holmen TL, Bratberg GH et al. The Nord-Trøndelag Health Study 1995-97 (HUNT 2): Objectives, contents, methods and participation. *Norwegian Journal of Epidemiology* 2003;13(1):19-32.

9. Midthjell K, Kruger O, Holmen J, Tverdal A, Claudi T, Bjorndal A et al. Rapid changes in the prevalence of obesity and known diabetes in an adult Norwegian population. The Nord-Trondelag Health Surveys: 1984-1986 and 1995-1997. *Diabetes Care* 1999;**22**(11):1813-20.

10. Nilsson PM, Nilsson JA, Hedblad B, Berglund G, Lindgarde F. The enigma of increased non-cancer mortality after weight loss in healthy men who are overweight or obese. *J Intern Med* 2002;**252**(1):70-8.

11. Masuo K, Mikami H, Ogihara T, Tuck ML. Weight gain-induced blood pressure elevation. *Hypertension* 2000;**35**(5):1135-40.

 Hall JE, Brands MW, Hildebrandt DA, Kuo J, Fitzgerald S. Role of sympathetic nervous system and neuropeptides in obesity hypertension. *Braz J Med Biol Res* 2000;**33**(6):605-18.

Czernichow S, Mennen L, Bertrais S, Preziosi P, Hercberg S, Oppert JM.
 Relationships between changes in weight and changes in cardiovascular risk
 factors in middle-aged French subjects: effect of dieting. *Int J Obes* 2002;26(8):1138-43.

 Wilsgaard T, Schirmer H, Arnesen E. Impact of body weight on blood pressure with a focus on sex differences: the Tromso Study, 1986-1995. *Arch Intern Med* 2000;**160**(18):2847-53.

15. Sonne-Holm S, Sorensen TI, Jensen G, Schnohr P. Independent effects of weight change and attained body weight on prevalence of arterial hypertension in obese and non-obese men. *BMJ* 1989;**299**(6702):767-70.

16. Huang Z, Willett WC, Manson JE, Rosner B, Stampfer MJ, Speizer FE et al.
Body weight, weight change, and risk for hypertension in women. *Ann Intern Med* 1998;128(2):81-8.

17. Jones DW. Body weight and blood pressure. Effects of weight reduction on hypertension. *Am J Hypertens* 1996;9(8):50s-4S.

16

1.1 1

Villanueva EV. The validity of self-reported weight in US adults: a population based cross-sectional study. *BMC Public Health* 2001;1(1):11.
 Spencer EA. Validity of self-reported height and weight in 4808 EPIC-Oxford participants. *Public Health Nutr* 2002;5(4):561-5.

20. Lund-Larsen PG. Blood pressure measured with a sphygmomanometer and with Dinamap under field conditions - a comparison. *Norwegian Journal of Epidemiology* 1997;7(2):235-41.

21. Holmen J. Detecting hypertension: screening versus case finding in Norway.*BMJ* 1991;302(6770):219-22.

22. Montague CT, Prins JB, Sanders L, Digby JE, O'Rahilly S. Depot- and sexspecific differences in human leptin mRNA expression: implications for the control of regional fat distribution. *Diabetes* 1997;**46**(3):342-7.

23. Bjorntorp P. The regulation of adipose tissue distribution in humans. Int J Obes 1996;20(4):291-302.

24. Janssen I, Heymsfield SB, Allison DB, Kotler DP, Ross R. Body mass index and waist circumference independently contribute to the prediction of nonabdominal, abdominal subcutaneous, and visceral fat. *Am J Clin Nutr* 2002;75(4):683-8.

25. Kannel WB, Cupples LA, Ramaswami R, Stokes J 3, Kreger BE, Higgins M. Regional obesity and risk of cardiovascular disease; the Framingham Study. *J Clin Epidemiol* 1991;44(2):183-90.

26. Collins R. Blood pressure, stroke, and coronary heart disease. Part 2, Shortterm reductions in blood pressure: overview of randomised drug trials in their epidemiological context. *Lancet* 1990;**335**(8693):827-38. 27. Levy D, Larson MG, Vasan RS, Kannel WB, Ho KK. The progression from hypertension to congestive heart failure. *JAMA* 1996;**275**(20):1557-62.

18

na na series de la s La series de la serie

in parentheses.					
Gender	Variables		BMI change categorisation		Total
		Decreased (2SE)	Stable (2SE)*	Increased (2SE)	
Women	N	985	3928	11058	15971
	Mean age (y)	50.2 (1.1)	44.2 (0.38)	38.4 (0.26)	40.9 (0.22)
	BMI first survey (kg/m ²)	26.6 (0.28)	24.2 (0.22)	24.3 (0.22)	25.0 (0.22)
	BMI second survey (kg/m ²)	24.0 (0.34)	24.3 (0.26)	27.4 (0.24)	25.2 (0.24)
	SBP first survey (mmHg)	134.3 (1.2)	131.7 (1.0)	131.5 (1.0)	132.5 (0.9)
	SBP second survey (mmHg)	142.0 (1.6)	142.5 (1.2)	146.1 (1.2)	143.5 (1.2)
	DBP first survey (mmHg)	81.3 (0.8)	80.0 (0.6)	80.3 (0.6)	80.5 (0.6)
	DBP second survey (mmHg)	79.6 (1.0)	79.8 (0.8)	82.0 (0.8)	80.5 (0.8)
Men	Z	680	4693	8473	13846
	Mean age (y)	47.7 (1.06)	43.4 (0.46)	39.4 (0.22)	40.9 (0.20)
	BMI first survey (kg/m ²)	26.4 (0.26)	24.8 (0.18)	24.9 (0.18)	25.4 (0.18)

¢

stratified for categories of change in BMI (decreased, stable, increased) during the follow-up with 95% confidence interval (2Standard Errors (SE)) Table 1. Age-adjusted mean body mass index (BMI), systolic blood pressure (SBP) and diastolic blood pressure (DBP) at first and second survey

Miner her i statiet er er er

BMI second survey (kg/m ²)	24.4 (0.28)	27.5 (0.2)	27.5 (0.2)	25.7 (0.2)
SBP first survey (mmHg)	139.3 (1.4)	136.0 (1.0)	135.3 (0.9)	136.8 (0.9)
SBP second survey (mmHg)	142.1 (1.6)	143.4 (1.2)	146.5 (1.1)	144.0 (1.2)
DBP first survey (mmHg)	85.1 (0.9)	82.9 (0.6)	82.8 (0.6)	83.6 (0.6)
DBP second survey (mmHg)	81.0 (1.0)	82.1 (0.7)	84.9 (0.6)	82.7 (0.6)

*Stable defined as initial BMI \pm 0.1 kg/m² each follow-up time.

•

.

	ווח ארירטות אוו		j un ugo au						
95% confic	tence interval	95% confidence interval (CI) in parentheses.							
Gender	Age	BMI change *	u	Change ir	Change in systolic blood pressure	sure	Change in	Change in diastolic blood pressure	sure
	HUNT I	HUNT 2 – HUNT I		Н	HUNT 2 – HUNT 1		Н	HUNT 2 – HUNT I	
			:	Age-adj	Multi adj [†]	p-value	Age-adj	Multi adj ⁱ	p-value
Women	20-49	Stable	2596	Reference	Reference		Reference	Reference	I
		Decreased	506	-1.4	-2.2 (-3.6, -0.8)	0.001	-1.2	-1.0 (-1.9,0.0)	0.058
		Increased	8870	3.8	3.8 (3.2, 4.4)	<0.001	1.9	1.9 (1.4, 2.3)	<0.001
	50+	Stable	1332	Reference	Reference	ı	Reference	Reference	1
		Decreased	479	-5.0	-5.0 (-7.0,-3.0)	<00.0>	-1.9	-1.7 (-3.0, -0.4)	0.012
		Increased	2188	3.9	3.9 (2.6,5.3)	<0.001	2.2	2.2 (1.4, 3.1)	<0.001
Men	20-49	Stable	3084	Reference	Reference	1	Reference	Reference	ţ
		Decreased	319	-2.5	-2.6 (-4.2,-1.0)	0.002	-3.0	-2.6(-3.7,-1.4)	<0.001
		Increased	7004	4.2	4.2 (3.6, 4.7)	<0.001	2.9	2.9 (2.5, 3.3)	<0.001
	50+	Stable	1609	Reference	Reference	1	Reference	Reference	ı

Table 2. The association between change in systolic and diastolic blood pressure and change in body mass index (BMI) between the first (HUNT 1, 1984-86) and second survey (HUNT 2, 1995-97) in age and multivariable linear regression. Totally, 15971 women and 13846 men were included.

1.1.1.2.2	• •				$\left\ \left\ \left$
<0.00	<0.001				
v					
-3.5 (-4.8,-2.1) <0.001	2.9 (2.1, 3.7)				
(-4.8	(2.1,				
-3.5	2.9				
-3.8	2.8				
`ı' '	(1				
01	10				
<0.001	<0.001				
-6.6 (-8.6,-4.4)	3.0 (1.7, 4.4)				
5 (-8.	0(1.7				
-9.	τ, -				
-6.6	3.0	year. '.			
		* Stable defined as initial BMI \pm 0.1 kg/m ² each follow-up year. [†] Adjusted for age at first survey and BMI at second survey.			
_	66	follor ond s			
361	1469	each at sec			
		g/m ² 3MI a			
		0.1 k and]			
Decreased	ased	MI ± urvey			
Decre	Increased	ial B irst su			
		is init e at f			
		ined i for ag			
		e def isted			
		Stabl Adju			
		* ***			

.

(1) Statistical and the statistical statisti statistical statisticae statisticae statisticae statisticae statisticae statisticae statis

> Table 3. Adjusted odds ratio (OR) for having hypertension (defined as blood pressure ≥140/90 mm Hg) at the second survey (HUNT 2) and 95% confidence interval (CI) stratified by gender, age (years) at first survey and change in body mass index (BMI). The Nord-Trøndelag Health Study (HUNT).

	Age		Hypertensio	on (HUNT 2)
		Change BMI *	Age adj. OR	Multi adj. OR [†]
			(95% CI)	(95% CI)
Women	20-49	Decreased	1.6 (1.2, 2.2)	1.0 (0.7, 1.5)
		Stable	1.0	1.0
		Increased	1.7 (1.4, 2.0)	1.8 (1.5, 2.2)
	50+	Decreased	0.9 (0.7, 1.2)	0.8 (0.6, 1.0)
		Stable	1.0	1.0
		Increased	1.4 (1.2, 1.7)	1.5 (1.2, 1.7)
Men	20-49	Decreased	1.1 (0.8, 1.5)	0.7 (0.5, 1.0)
		Stable	1.0	1.0
		Increased	1.5 (1.4, 1.7)	1.6 (1.4, 1.8)
	50+	Decreased	0.8 (0.6, 1.0)	0.6 (0.5, 0.8)
		Stable	1.0	1.0
		Increased	1.4 (1.2, 1.6)	1.5 (1.3, 1.7)

* Stable defined as $\pm 0.1 \text{ kg/m}^2$ each follow-up year.

[†] OR adjusted for: Age and BMI at first survey (HUNT 1) and blood pressure status HUNT 1.

		Change in SBP (95% CI), mmHg	95% CI), mmHg			Change in DBP	Change in DBP (95% CI), mmHg	
BMI at first		BMI at second survey	nd survey			BMI at sec	BMI at second survey	
survey	Underweight	Normal weight	Overweight	Obese	Underweight	Normal weight	Overweight	Obese
Women								
Underweight	1.7 (-2.0, 5.3)	2.0 (0.0, 4.0)	11.9 (4.2, 19.6)	NC	0.7 (-1.7, 3.2)	1.0 (-0.3, 2.4)	8.2 (3.0, 13.5)	NC
Normal weight	nt -5.0 (-9.7, -0.3)	Reference	3.7 83.0, 4.39	7.6 (5.7, 9.5)	-2.4 (-5.5, 0.8)	Reference	1.8 (1.4, 2.2)	5.3 (4.0, 6.7)
Overweight	NC	-2.3 (-4.3, -0.3)	2.0 (1.2, 2.8)	6.8 (5.9, 7.8)	NC	-1.3 (-2.6, 0.04)	0.3 (-0.2, 0.9)	2.7 (2.0, 3.3)
Obese	NC	-12.0 (-21.3, -2.7)	-4.8 (-8.0, -1.6)	2.5 (1.4, 3.6)	NC	-7.8 (-14.1, -1.4)	-7.8 (-14.1, -1.4) -5.1 (-7.2, -2.9) -1.3 (-2.0, -0.5)	-1.3 (-
Men								
Underweight	0.7 (-7.3, 8.6)	0.3 (-4.9, 5.6)	NC	NC,	-4.1 (-9.6, 1.5)	1.4 (-2.2, 5.0)	NC [*]	NC
Normal weight	nt -9.4 (~16.3, -2.5)	Reference	2.7 (2.0, 3.4)	4.9 (1.2, 8.6)	-3.5 (-8.3, 1.2)	Reference	2.1 (1.6, 2.6)	5.7 (3.1, 8.2)
Overweight	NC*	-3.5 (-5.5, -1.5)	1.6 (0.9, 2.2)	3.9 (2.9, 4.9)	NC [*]	-2.5 (-3.9, -1.2)	0.4 (-0.03, 0.9)	1.9 (1.3, 2.6)
Obese	NC	NC	-3.7 (-7.1, -0.2)	1.5 (0.0, 3.0)	NC	NC	-4.9 (-7.2, -2.5) -2.4 (-3.3, -1.4)	-2.4 (-:

Table 4. Age-adjusted change in mean systolic and diastolic blood pressure (SBP and DBP) with normal weight at both surveys as reference group and 95% confidence

*NC = not calculated due to N \leq 10

Paper IV

1

Weight change and mortality

The Nord-Trøndelag Health Study

Wenche B. Drøyvold¹⁾, Tom I. L. Nilsen¹⁾, Stian Lydersen¹⁾, Kristian Midthjell¹⁾, Peter M. Nilsson²⁾, Jan-Åke Nilsson²⁾, Jostein Holmen¹⁾

¹⁾ Department of Public Health and General Practice, Faculty of Medicine,

Norwegian University of Science and Technology, Norway.

²⁾ Department of Medicine, University Hospital, Malmö, Sweden.

Address correspondence to:

Wenche B. Drøyvold Department of Public Health and General Practice Norwegian University of Science and Technology University Medical Centre N-7489 Trondheim, Norway Office phone: +47 73 59 75 37 Office fax: +47 73 59 87 89 E-mail: wenche.b.droyvold@medisin.ntnu.no

Abstract

Background: The prevalence of obesity is increasing worldwide, and overweight and obese people have increased mortality compared to normal weight people. We have prospectively investigated the effect of weight change on mortality.

Methods: We utilized data from two large population-based health studies in Nord-Trøndelag, Norway, the first conducted in 1984-86 and the second in 1995-97. A total of 20 542 men and 23 712 women aged 20 years or more were followed-up on all-cause mortality for five years after the second survey. Cox proportional hazards models were used to calculate mortality rate ratios (RRs) with 95% confidence intervals (CIs) between people with a stable weight and people who lost or gained weight.

Results: We found no association between weight gain and mortality, but people who lost weight had a higher total mortality rate compared to those who were weight stable (RR was 1.6 (95% CI: 1.4, 1.8) in men and 1.7 (95% CI: 1.5, 2.0) in women). Similar associations were also found for cardiovascular and non-cardiovascular mortality. Additional analysis showed a linear increase in mortality rates across categories of weight loss for both men and women ($P_{trend} < 0.001$). Moreover, there was a statistically significant interaction between weight change and initial BMI, but only among men ($P_{interaction} = 0.001$). *Conclusions:* Weight loss, but not weight gain, was associated with increased mortality both among men and women. Although underlying undiagnosed disease is the most plausible explanation for this finding, the similar associations found for total mortality, cardiovascular mortality, and non-cardiovascular mortality makes the causal pathway somewhat enigmatic.

Keywords: weight change, physical activity, smoking, body mass index

1. 11. 11

Introduction

A body mass index (BMI) outside the normal weight range (18.5-24.9 kg/m²) is associated with increased mortality^{1,2}, and the nadir of the mortality curve has been found at a BMI of 22-24 kg/m^{23,4}. Obesity has had disease status since 1985². Additionally, a number of diseases can be linked to overweight and obesity, and each disease can in main be classified into two pathophysiological categories⁵. The first arises from the increased mass of fat which may include the stigma of obesity and the behavioural responses it produces, and musculoskeletal disorders^{6,7}. The second category comprises metabolic changes associated with excess fat, and examples of these includes diabetes type 2⁸, gallbladder disease⁹, hypertension¹⁰, cardiovascular disease¹¹, and some forms of cancer¹².

The prevalence of overweight and obesity is rapidly increasing^{2,13}, and consequently a large proportion of people are trying to lose weight. Weight loss is associated with short-time improvements in risk factors such as blood pressure¹⁴, cholesterol¹⁵ and diabetes^{16,17}. Controversially, weight loss has also been associated with increased mortality in observational studies¹⁸⁻²⁴, but the results are not consistent^{1,25,26}. Additionally, the knowledge about the association between change in BMI and subsequent mortality is mainly based on studies on men^{19,27,28}.

We have utilized information on height, weight and cause specific mortality data in a large population of Norwegian men and women who participated in the Nord-Trøndelag Health Study both in 1984-86 and in 1995-97, to investigate the association between weight change and mortality.

We wanted especially to study the potential effect modification of initial BMI, leisure time physical activity and smoking status on the association between weight change and mortality.

Materials and methods

Study population

In 1984-86 and 1995-97, two general health surveys were conducted in Nord-Trøndelag County (127 000 inhabitants), the Nord-Trøndelag Health Study, Norway. The participation rates were 88.1% and 71.2%, respectively. Data collection was based on self-reported questionnaires and standardised measurements of physiological variables such as height and weight. In total 24 837 women and 21 685 men participated in both surveys. We excluded participants who reported preexisting diabetes or cardiovascular disease at baseline or who had a history of cancer at the second survey. A total of 23712 women and 20542 men aged 20 years or more at the first survey and with information on body weight and body height at both surveys were available for analyses.

Follow-up

The unique 11-digit identification number of every Norwegian citizen enabled linkage between the collected information and the Death Registry at Statistics Norway to determine vital status (alive, emigrated, dead) and cause-specific deaths. Each participant contributed person-years from the date of the second survey until the date of death, emigration, or end of follow-up (December 31th, 2001). Mean time between the surveys was 11 years (range 9-13 years), and mean follow-up after the second survey was 5 years (range 0-6 years). Cardiovascular mortality was classified using the 9th revision of the International Classification of Diseases (cardiovascular

diagnosis codes 390-459) before 1997, and the 10th revision (codes I00-I99) thereafter.

Body mass index

Body mass index was calculated as body weight in kilograms divided by the squared value of body height in meters (kg/m⁻). In both surveys height was measured without shoes to the nearest centimetre and weight was measured wearing light clothes without shoes to the nearest half-kilogram at the survey site. Change in BMI between the surveys was categorised into loss, stable, gain. A stable BMI was defined as a change in BMI equal to or less than 0.1 kg/m² per follow-up year²⁹. We categorised BMI at the first survey applying the World Health Organisation's (WHO) recommendation (underweight: <18.5 kg/m², normal weight 18.5-24.9 kg/m², overweight 25.0-29.9 kg/m², and obesity \geq 30 kg/m²).

Leisure time physical activity

At the first survey, leisure time physical activity was self-reported by three questions about frequency, duration and intensity, each with five, four and three possible answers, respectively. Only those who reported a frequency of once a week or more answered the questions about intensity and duration. We categorised leisure time physical activity into low, moderate and high levels based on the questions about frequency, intensity and duration: A frequency of never or less than once a week was categorized as low. For those with a frequency of once a week or more, a summary was calculated by adding the values of frequency, intensity and duration. The sum value was then divided into moderate and high by dichotomizing at the median value.

Smoking status

We classified smoking status at the first survey in three categories, where never were individuals who had never smoked daily, and those who reported previous or present daily smoking were classified as former or current smokers, respectively.

Statistical Analyses

Cox regression analysis was used to calculate age-adjusted and multivariable adjusted mortality rate ratios (RRs) with 95% confidence intervals (CIs) associated with change in BMI (loss, stable, gain), using the weight stable group as reference. The analyses were performed separately for males and females, and in strata of initial BMI (WHO categorisation), leisure time physical activity levels and smoking status (never, former, current). Due to few cases, adjusted RR estimates were not calculated for men and women in the underweight group. We conducted multivariable analyses to assess potential confounding by the following variables measured at the first survey: age (<40, 40-44, ..., ≥80 years), body mass index (<18.5, 18.5-24.9, 25.0-29.9, ≥30 kg/m²), systolic blood pressure (quintiles), blood pressure medication (no, yes), smoking (never, former, current), alcohol drinking past two weeks (none, 1-4 times, ≥5 times, teetotaller), leisure time physical activity (low, moderate, high), marital status (married, unmarried, widow/widower, divorced/separated), education (middle school, high school, <4 years of college/university, ≥4 years of college/university). All analyses were performed using the statistical software SPSS for Windows, version 11.0 (SPSS, Chicago, ILL, USA).

Ethics

The participation was completely voluntary and each participant signed a written consent. The Norwegian Data Inspectorate recommended both surveys, and the second survey was also approved by the Regional Ethical Committee for Medical Research. At the time of the first survey, the Regional Ethical Committee was not yet established.

Results

During five years of follow-up (mean = 5.4 years) we observed 2672 deaths altogether, 1551 among men and 1119 among women (Table 1). Out of these, 709 men and 475 women died from cardiovascular causes. Among men who lost weight we found that 24.2% died during follow-up, while 8.8% of men who were weight stable and 5.0% who gained weight died. Similar figures for women were 16.6%, 5.9% and 2.8%, respectively. However, only 6.5% of men and 8.3% of women had lost weight between the surveys, while as much as 58.9% of men and 66.0% of women had gained weight. Persons who lost weight had on average a higher BMI at the first survey than those who were weight stable or gained weight. The mean weight loss was 2.2 kg/m² in men and 2.7 kg/m² in women, while mean weight gain was 2.7 kg/m² and 3.4 kg/m² for men and women, respectively. (Table 1 here).

In analyses of weight change and mortality (Table 2) we found that people who lost weight had a higher total mortality rate compared to those who were weight stable (multivariable RR was 1.6, 95% CI: 1.4, 1.8 in men and 1.7, 95% CI: 1.5, 2.0 in women). Similar associations were also found in analyses of cardiovascular and non-cardiovascular mortality. People who gained weight between the studies had the same mortality rate as those who were weight stable (total mortality RR = 1.0, 95% CI: 0.9, 1.1 in men and 0.9, 95% CI: 0.8, 1.0 in women). (Table 2 here). Additional analysis showed a statistically significant linear increase in mortality rates across categories of weight loss for both men and women ($P_{trend} < 0.001$), but no linear relation across categories of weight gain ($P_{trend} = 0.26$ among men and 0.11 among women) (Figure 1). (Figure 1 here).

In subsequent analyses on total mortality we found a statistically significant interaction between weight change and initial BMI among men ($P_{\text{interaction}} = 0.001$),

but not among women ($P_{\text{interaction}} = 0.31$) (Table 3). Among men who had a normal weight (BMI: 18.5-24-9 kg/m²) at the first survey the total mortality RR where 2.0 (95% CI: 1.6, 2.4) for those who lost weight compared to those who were weight stable. Among overweight or obese men, the RRs comparing weight loss and weight stable were 1.4 (95% CI: 1.2, 1.8) and 1.5 (95% CI: 1.0, 2.3), respectively. Although no statistically significant interaction was found for women, we observed that overweight women who lost weight had a higher RR than normal weight women who lost weight (2.0, 95% CI: 1.6, 2.6 vs. 1.5, 95% CI: 1.1, 1.9). We found no statistically significant interaction with physical activity or smoking status, although the results may indicate some effect modification by smoking status. Current smoking men who lost weight had an RR of 2.1 compared to weight stable men, while the same association among former smoking men was 1.2. However, among women the findings were somewhat opposite those for men, since the strongest association was found among former smokers (RR = 2.5 comparing loss and stable) and the weakest among never and current smokers (RR = 1.6 in both groups). (Table 3 here).

In supplementary analyses we explored potential confounding by disease status reported at the second survey for events that could have occurred between the two surveys, such as myocardial infarction, stroke, diabetes, angina, chronic obstructive lung syndrome, and asthma, but none of these factors influenced the estimated association between weight change and mortality (data not shown). In an attempt to evaluate the role of pre-diagnosed disease as a plausible cause for our findings we excluded the first three years of follow-up, but the results remained similar (total mortality RR comparing weight loss and weight stable was 1.5 (95% CI: 1.3, 1.8) among men and 1.7 (95% CI: 1.4, 2.1) among women).

Discussion

In this prospective study we found a statistically significant higher mortality rate among people who had lost weight compared to people with a stable weight, both in analysis of total mortality, CV mortality and non-CV mortality. Also previous studies have reported that people who have lost weight have higher rates of both total mortality and cause specific mortality than those who are weight stable^{20-24,30}, although some studies have not found this association^{1,25,26}. Additionally, we observed a linearly increasing mortality rate ratio with increasing weight loss, which is somewhat contradictory to the findings by Williamson et al³¹, who reported a Jshaped association with increasing weight loss. However, they studied the effect of self- reported intentional weight loss, while we did not have the ability to distinguish between intentional and unintentional loss. It has been argued that this distinction is necessary^{32,33}, since those who intend to lose weight are doing so for health promotion and disease prevention rather than treatment of weight-related health conditions. The validity about the knowledge of intentional vs. non-intentional weight loss in observational studies has been questionned³⁴, and in a general population a large proportion will state that they try to lose weight at any time^{35,36}, but the effort put into it will probably be extremely variable. In addition, others have suggested that both intentional and unintentional weight loss may follow the development of disease37.

In agreement with previous studies¹¹, we found that weight loss was associated with increased mortality in all categories of initial body mass index. However, the highest mortality rates associated with weight loss was seen among normal weight men and overweight women, indicating some effect modification by initial body mass index.

The higher mortality rate among normal weight men may indicate that weight loss is more hazardous if the initial body mass is low, although a similar reasoning is not as obvious for women. Previous data has suggested that weight change is associated with a more unfavourable relative change in fat-free mass in men than women, suggesting that the metabolic and health consequences of weight change may be dependent on gender³⁸. Due to the low number of people who initially were underweight and then lost weight, we have not presented results for this strata.

Smoking status is likely to be an important factor when studying weight loss and mortality, since it is associated both with lower body weight and increased morbidity³⁹ and mortality⁴⁰. However, only few prospective studies have investigated the potential effect modification of smoking on the association between weight loss and mortality^{11,41}. In our study weight loss was associated with increased mortality both in never, former and current smoking men and women, although the strongest association was found among current smoking men and former smoking women. Current smoking men may both lose more weight and have a higher mortality rate than never and former smoking men, and our results confirm findings by others¹¹. The increased mortality among former smoking women who lost weight could be a result of 'confounding by indication'; i.e. some women may have received information on high blood pressure and/or high cholesterol levels and thus ceased smoking, but still remained at a higher risk for dying.

Weight loss was also associated with increased mortality within all levels of leisure time physical activity, and none of the associations were markedly different between the activity strata. Hence, effect modification by innitial physical activity level are not likely to be present. To our knowledge, this has not been explored in any other studies.

Previous studies have found that weight gain is associated with increased mortality^{11,42}, but contradictory to these studies we found that people who gained weight between the studies had similar mortality rates as those who were weight stable. One might speculate that weight gain among generally healthy people is not linked to increased mortality. Moreover a relatively short follow-up time in this study may have contributed to this finding, since the effect of weight gain on mortality is likely a relatively slow process? It is likely that future analyses based on HUNT-data with longer follow-up time included may answer this question.

Previous studies have shown that pre-existing disease is associated with both weight loss and mortality^{24,43,44}. In our study we have excluded participants who reported diabetes or cardiovascular disease at the first study, and also those who reported a history of cancer at the second survey. Additionally, adjustment for diabetes, cardiovascular disease, Chronic Obstructive Pulmonary Disease (COBD) and asthma at the second survey did not reduce the increased mortality rate associated with weight loss. In an attempt to assess the potential importance of undiagnosed disease at baseline, we excluded the first three years of follow-up, but the results still remained similar as in the overall analysis. This method has also been applied in other studies^{45,46}, but the validity of this method is not entirely agreed upon. In a meta-analysis, Allison *et al*⁴⁷ supported not to exclude early deaths in BMI-mortality studies.

The strengths of our study includes the high numbers of participants both in women and men, the wide age-range, information on a large number of potential confounders, the standardised measurements of height and weight, and the linkage of the data to the national Registry of Death at Statistics Norway ensuring complete follow-up on vital status. The potential bias due to misclassification in the death

certificates is unlikely to be related to different levels of change in body weight and thus to explain the results. Additionally, the prospective design of the study makes it unlikely that the results may be biased due to selection of participants or differential misclassification of information.

Although undiagnosed disease is probably the most plausible explanation for the observed increase in mortality among people who lost weight, one may speculate in other explanations for the finding. One possible mechanism may be that a reduction in weight initiates a stress response in the body, and it has been shown that more biological mechanisms are involved in prevention of weight loss than of weight gain⁴⁸.

In conclusion, this study has shown that weight loss, but not weight gain, is associated with increased mortality rates in apparently healthy men and women, compared to people with a stable weight. This finding was similar in analysis of both total mortality, CV mortality, and non-CV mortality. Stratified analysis indicates that this effect may be modified by initial body mass index and smoking status. The most plausible explanation for this finding is the existence of undiagnosed disease, although the consistent results between different causes of death may suggest a role for other potential mechanisms.

References

1. Maru S, van der Schouw YT, Gimbrere CH, Grobbee DE, Peeters PH. Body mass index and short-term weight change in relation to mortality in Dutch women after age 50 y. *Am J Clin Nutr* 2004;**80**(1):231-6.

Obesity: preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organ Tech Rep Ser* 2000;894:i-XII 1-253.
 Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath Jr CW. Body-mass index and mortality in a prospective cohort of U.S. adults. *N Engl J Med* 1999;341(15):1097-105.

4. Baik I, Ascherio A, Rimm EB, Giovannucci E, Spiegelman D, Stampfer MJ et al.
Adiposity and mortality in men. Am J Epidemiol 2000;152(3):264-71.

 Bray GA. Medical consequences of obesity. J Clin Endocrinol Metab 2004;89(6):2583-9.

6. Larsson UE. Influence of weight loss on pain, perceived disability and observed functional limitations in obese women. *Int J Obes* 2004;**28**(2):269-77.

 Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. *N Engl J Med* 1993;**329**(14):1008-12.

 Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. *JAMA* 1999;282(16):1523-9.
 Torgerson JS, Lindroos AK, Naslund I, Peltonen M. Gallstones, gallbladder disease, and pancreatitis: cross-sectional and 2-year data from the Swedish Obese Subjects (SOS) and SOS reference studies. *Am J Gastroenterol* 2003;98(5):1032-41.
 Rocchini AP. Obesity hypertension. *Am J Hypertens* 2002;15(2 PT 2):50S-2S.

 Rosengren A, Wedel H, Wilhelmsen L. Body weight and weight gain during adult life in men in relation to coronary heart disease and mortality. A prospective population study. *Eur Heart J* 1999;20(4):269-77.

12. Calle EE, Thun MJ. Obesity and cancer. Oncogene 2004;23(38):6365-78.

 Seidell JC. Obesity, insulin resistance and diabetes--a worldwide epidemic. Br J Nutr 2000;83 Suppl 1:S5-8.

14. Bacon SL, Sherwood A, Hinderliter A, Blumenthal JA. Effects of exercise, diet and weight loss on high blood pressure. *Sports Med* 2004;34(5):307-16.

15. Poobalan A, Aucott L, Smith WC, Avenell A, Jung R, Broom J et al. Effects of weight loss in overweight/obese individuals and long-term lipid outcomes--a systematic review. *Obes Rev* 2004;6(2):43-50.

16. Aucott L, Poobalan A, Smith WC, Avenell A, Jung R, Broom J et al. Weight loss in obese diabetic and non-diabetic individuals and long-term diabetes outcomes--a systematic review. *Diabetes Obes Metab* 2004;6(2):85-94.

 Sjostrom CD, Peltonen M, Wedel H, Sjostrom L. Differentiated long-term effects of intentional weight loss on diabetes and hypertension. *Hypertension* 1999;7(5):20 5.

 Higgins M, D'Agostino R, Kannel W, Cobb J, Pinsky J. Benefits and adverse effects of weight loss. Observations from the Framingham Study. *Ann Intern Med* 1993;119(7 PT 2):758-63.

 Peters ET, Seidell JC, Menotti A, Arayanis C, Dontas A, Fidanza F et al.
 Changes in body weight in relation to mortality in 6441 European middle-aged men: the Seven Countries Study. *Int J Obes* 1995;19(12):862-8.

20. Nilsson PM. The enigma of increased non-cancer mortality after weight loss in healthy men who are overweight or obese. *J Intern Med* 2002;**252**(1):70-8.

 21. Mikkelsen KL, Heitmann BL, Keiding N, Sorensen TI. Independent effects of stable and changing body weight on total mortality. *Epidemiology* 1999;10(6):671-8.
 22. Lee IM, Paffenbarger Jr RS. Change in body weight and longevity. *JAMA* 1992;268(15):2045-9.

Sorensen TI. Weight loss causes increased mortality: pros. Obes Rev 2003;4(1):3-7.

24. Pamuk ER. Weight loss and subsequent death in a cohort of U.S. adults. Ann Intern Med 1993;119(7 PT 2):744-8.

25. Williamson DF. The association between weight loss and increased longevity. A review of the evidence. *Ann Intern Med* 1993;**119**(7 PT 2):731-6.

26. Yang D. Weight loss causes increased mortality: cons. *Obes Rev* 2003;4(1):9-16.
27. Blair SN, Shaten J, Brownell K, Collins G, Lissner L. Body weight change, all-cause mortality, and cause-specific mortality in the Multiple Risk Factor Intervention Trial. *Ann Intern Med* 1993;119(7 PT 2):749-57.

28. Wannamethee G, Shaper AG. Weight change, perceived health status and mortality in middle-aged British men. *Postgrad Med J* 1990;44(2):910-3.

29. Nilsson PM, Nilsson JA, Hedblad B, Berglund G, Lindgarde F. The enigma of increased non-cancer mortality after weight loss in healthy men who are overweight or obese. *J Intern Med* 2002;**252**(1):70-8.

 Yaari S, Goldbourt U. Voluntary and involuntary weight loss: associations with long term mortality in 9,228 middle-aged and elderly men. *Am J Epidemiol* 1998;148(6):546-55.

31. Williamson DF, Thompson TJ, Thun M, Flanders D, Pamuk E, Byers T.
Intentional weight loss and mortality among overweight individuals with diabetes. *Diabetes Care* 2000;23(10):1499-504.

32. Williamson DF. Intentional weight loss: patterns in the general population and its association with morbidity and mortality. *Int J Obes* 1997;**21 Suppl 1**:S14-9.

33. Gregg EW. Intentional weight loss and death in overweight and obese u.s. Adults35 years of age and older. *Ann Intern Med* 2003;138(5):383-9.

34. Fontaine KR, Allison DB. Does intentional weight loss affect mortality rate? *Eat Behav* 2001;**2**(2):87-95.

35. Hjartaker A, Laake P, Lund E. Body mass index and weight change attempts among adult women. The Norwegian Women and Cancer Study. *Eur J Public Health* 2001;11(2):141-6.

36. French SA, Jeffery RW, Folsom AR, Williamson DF, Byers T. Relation of weight variability and intentionality of weight loss to disease history and health-related variables in a population-based sample of women aged 55-69 years. *Am J Epidemiol* 1995;**142**(12):1306-14.

 Wannamethee SG, Shaper AG, Whincup PH, Walker M. Characteristics of older men who lose weight intentionally or unintentionally. *Am J Epidemiol* 2000;151(7):667-75.

38. Heitmann BL, Garby L. Composition (lean and fat tissue) of weight changes in adult Danes. *Am J Clin Nutr* 2002;**75**(5):840-7.

39. Grace SL, Fry R, Cheung A, Stewart DE. Cardiovascular Disease. *BMC Womens Health* 2004;4 Suppl 1:S15.

40. Schnohr C, HOjbjerre L, Riegels M, Ledet L, Larsen T, Schultz-Larsen K et al. Does educational level influence the effects of smoking, alcohol, physical activity, and obesity on mortality? A prospective population study. *Scand J Public Health* 2004;**32**(4):250-6.

 Wannamethee SG, Shaper AG, Walker M. Weight change, body weight and mortality: the impact of smoking and ill health. *Int J Epidemiol* 2001;**30**(4):777-86.
 Fulton JE, Shekelle RB. Cigarette smoking, weight gain, and coronary mortality: results from the Chicago Western Electric Study. *Circulation* 1997;**96**(5):1438-44.
 Newman AB, Yanez D, Harris T, Duxbury A, Enright PL, Fried LP. Weight change in old age and its association with mortality. *J Am Geriatr Soc* 2001;**49**(10):1309-18.

44. Wannamethee SG, Shaper AG, Walker M. Weight change, weight fluctuation, and mortality. *Arch Intern Med* 2002;162(22):2575-80.

45. Williamson DF, Pamuk E, Thun M, Flanders D, Byers T, Heath C. Prospective study of intentional weight loss and mortality in never-smoking overweight US white women aged 40-64 years. *Am J Epidemiol* 1999;7(5):1128-41.

46. Stevens J, Juhaeri, Cai J. Changes in body mass index prior to baseline among participants who are ill or who die during the early years of follow-up. Am JEpidemiol 2001;153(10):946-53.

47. Allison DB, Faith MS, Heo M, Townsend-Butterworth D, Williamson DF. Metaanalysis of the effect of excluding early deaths on the estimated relationship between body mass index and mortality. *Obes Res* 1999;7(4):342-54.

48. Blundell JE, King NA. Overconsumption as a cause of weight gain: behaviouralphysiological interactions in the control of food intake (appetite). *Ciba Found Symp* 1996;**201**:138-54 DISCUSSION 15.

Men		Men			Women	
Variables	Loss	Stable	Gain	Loss	Stable	Gain
No. of participants (% within gender)	1319 (6.4)	7121 (34.7)	12 102 (58.9)	1971 (8.3)	6092 (25.7)	15 649 (66.0)
No. of all deaths (% within weight category)	319 (24.2)	627 (8.8)	607 (5.0)	327 (16.6)	357 (5.9)	435 (2.8)
Mean follow-up time, yrs (SD)	4.9 (1.4)	5.3 (1.0)	5.4 (0.8)	5.2 (1.2)	5.4 (0.8)	5.4 (0.7)
Mean age at death from all causes, yrs (SD)	68.2 (9.1)	65.4 (10.5)	62.8 (11.1)	70.5 (8.9)	67.7 (10.1)	62.5 (12.1)
Mean age at first survey, yrs (SD)	54.3 (13.8)	47.2 (13.5)	41.0 (12.8)	54.0 (15.8)	47.8 (14.8)	42.4 (12.9)
Mean BMI at first survey, kg/m ² (SD)	26.9 (3.6)	25.1 (2.9)	25.0 (2.9)	27.7 (5.2)	24.6 (4.1)	24.2 (3.8)
Mean BMI at second survey, kg/m ² (SD)	24.8 (3.4)	25.4 (2.9)	27.7 (3.3)	25.0 (4.6)	24.9 (4.0)	27.6 (4.4)
Mean change in BMI, kg/m ² (SD)	-2.2 (1.1)	0.2 (0.6)	2.7 (1.4)	-2.7 (1.9)	0.2 (0.6)	3.4 (1.9)

IS Table 1. Characteristics of 20 542 men and 23 712 women aged 20 years or more who participated in the Nord-Trøndelag Health Study, Norway in 1984-86 and in 1995-97, ne fredd 11 on o dae New yn de gwleite

Person- No. of Age-adj. Multivariable* No. of Age-adj. in BMI* years deaths RR R (95% CI) deaths RR 6521 319 1.7 1.6 (1.4-1.8) 150 1.7 1.5 (1.2-1.9) 169 1.8 6521 319 1.7 1.6 (1.4-1.8) 150 1.7 1.5 (1.2-1.9) 169 1.8 65124 607 1.0 1.0 285 1.0 1.0 342 1.0 65124 607 1.0 1.0 (0.9-1.1) 274 1.0 1.0 (0.9-1.2) 333 0.9 6 3263 337 1.8 1.7 (1.5-2.0) 150 1.8 1.7 (1.3-2.1) 177 1.7 84 931 435 0.8 0.9 (0.8-1.0) 170 1.0 1.0 1.0 1.0 84 931 435 0.8 0.9 (0.8-1.0) 1.78 1.0 1.0 1.0 1.0				Total mortality	Total mortality		CV mortality	ality		Non-CV mortality	ortality
years deaths R.R R.R (95% CI) deaths R.R (95% CI) deaths R.R (95% CI) deaths R.R 6521 319 1.7 1.6(1.4-1.8) 150 1.7 1.5(1.2-1.9) 169 1.8 37 617 627 1.0 1.0 285 1.0 1.0 342 1.0 65 124 607 1.0 1.0 274 1.0 1.0 342 1.0 65 124 607 1.0 1.0 0.9 333 0.9 10 214 327 1.8 1.7(1.5-2.0) 150 1.8 1.7(1.3-2.1) 177 1.7 32 683 357 1.0 1.0 1.0 1.0 1.0 1.0 1.0 84 931 435 0.8 0.9 (0.8-1.0) 178 1.0 1.0 1.0 1.0		Person-	No. of	Age-adj.	Multivariable ^e	No. of	Age-adj.	Multivariable ^c	No. of	Age-adj.	Multivariable
6521 319 1.7 1.6(1.4-1.8) 150 1.7 1.5(1.2-1.9) 169 1.8 37617 627 1.0 1.0 285 1.0 1.0 342 1.0 65124 607 1.0 1.0 274 1.0 1.0 333 0.9 10214 327 1.8 1.7(1.5-2.0) 150 1.8 1.7(1.3-2.1) 1.7 1.7 10214 327 1.8 1.7(1.5-2.0) 150 1.8 1.7(1.3-2.1) 1.7 1.7 32683 357 1.0 1.0 1.0 1.0 1.0 1.0 1.0 1.0 1.0 84931 435 0.8 0.9(0.8-1.0) 178 1.0 1.0(0.8-1.3) 257 0.8	Change in BMI ^a	years	deaths	RR	RR (95% CI)	deaths	RR	RR (95% CI)	deaths	RR	RR (95% CI)
6521 319 1.7 1.6 (1.4-1.8) 150 1.7 1.5 (1.2-1.9) 169 1.8 37 617 627 1.0 1.0 285 1.0 1.0 342 1.0 65 124 607 1.0 1.0 (0.9-1.1) 274 1.0 1.0 (0.9-1.2) 333 0.9 10 214 327 1.8 1.7 (1.5-2.0) 150 1.8 1.7 (1.3-2.1) 177 1.7 23 683 357 1.0 1.0 1.0 1.0 1.0 1.0 1.0 84 931 435 0.8 0.9 (0.8-1.0) 178 1.0 1.0 1.0 1.0 1.0 1.0	Males										
37617 627 1.0 1.0 285 1.0 1.0 342 1.0 65124 607 1.0 1.0(0.9-1.1) 274 1.0 1.0(0.9-1.2) 333 0.9 10214 327 1.8 1.7(1.5-2.0) 150 1.8 1.7(1.3-2.1) 177 1.7 232683 357 1.0 1.0 1.47 1.0 1.0 1.0 1.0 84931 435 0.8 0.9(0.8-1.0) 178 1.0 1.0(0.8-1.3) 257 0.8	Loss	6521	319	1.7	1.6 (1.4-1.8)	150	1.7	1.5 (1.2-1.9)	169	1.8	1.6 (1.4-1.9)
65 124 607 1.0 1.0 (0.9-1.1) 274 1.0 1.0 (0.9-1.2) 333 0.9 10 214 327 1.8 1.7 (1.5-2.0) 150 1.8 1.7 (1.3-2.1) 1.7 1.7 1.7 10 214 327 1.8 1.7 (1.5-2.0) 150 1.8 1.7 (1.3-2.1) 1.7 1.7 10 214 327 1.0 1.0 1.0 1.0 1.0 1.0 1.0 12 4931 435 0.8 0.9 (0.8-1.0) 178 1.0 1.0 (0.8-1.3) 257 0.8	Stable	37 617	627	1.0	1.0	285	1.0	1.0	342	1.0	1.0
10 214 327 1.8 1.7 (1.5-2.0) 150 1.8 1.7 (1.3-2.1) 177 1.7 32 683 357 1.0 1.0 1.0 147 1.0 1.0 210 1.0 84 931 435 0.8 0.9 (0.8-1.0) 178 1.0 1.0 (0.8-1.3) 257 0.8	Gain	65 124	607	1.0	1.0 (0.9-1.1)	274	1.0	1.0 (0.9-1.2)	333	0.9	1.0 (0.8-1.1)
10 214 327 1.8 1.7 (1.5-2.0) 150 1.8 1.7 (1.3-2.1) 177 1.7 a 32 683 357 1.0 1.0 1.0 1.0 1.0 1.0 a 32 683 357 1.0 1.0 1.0 1.0 1.0 1.0 a 4931 435 0.8 0.9 (0.8-1.0) 178 1.0 1.0 (0.8-1.3) 257 0.8	Females										
32 683 357 1.0 1.0 1.0 1.0 1.0 1.0 1.0 1.0 84 931 435 0.8 0.9 (0.8-1.0) 1.78 1.0 1.0 (0.8-1.3) 257 0.8	Loss	10 214	327	1.8	1.7 (1.5-2.0)	150	1.8	1.7 (1.3-2.1)	177	1.7	1.7 (1.4-2.0)
84 911 435 0.8 0.9 (0.8-1.0) 178 1.0 1.0 (0.8-1.3) 257 0.8	Stable	32 683	357	1.0	1.0	147	1.0	1.0	210	1.0	1.0
	Gain	84 931	435	0.8	0.9 (0.8-1.0)	178	1.0	1.0(0.8-1.3)	257	0.8	0.8 (0.7-1.0)

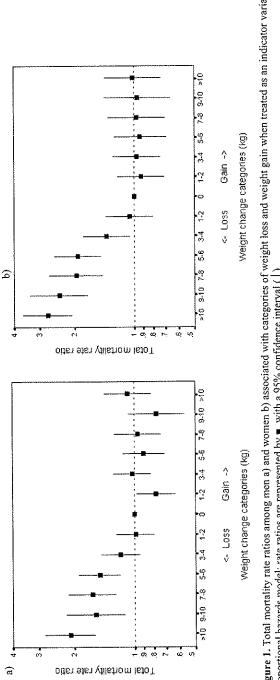
.

Table 2. Multivariable-adjusted rate ratios and 95% confidence intervals (CIs) of total mortality, cardiovascular (CV) mortality, and non-CV mortality associated with 10-year Pa Sh

Multivariable ⁶ RR (95% CI)
Stable Gain
1.0 0.8 (0.6-1.0)
1.0 1.0 (0.8-1.2)
1.0 1.1 (0.8-1.6)
$P_{\text{interaction}} = 0.08$
1.0 0.9 (0.7-1.1)
1.0 0.9 (0.7-1.1)
0.8 (0.5-1.2)
$P_{\text{interaction}} = 0.95$
1.0 0,9 (0.7-1.1)
1.0 1.1 (0.7-1.8)
1.0 1.0 (0.7-1.4)
$P_{\text{interaction}} = 0.46$
· · · · · · · · · · · · · · · · · · ·

Table 3. Multivariable adjusted rate ratios (RRs) with 95% confidence intervals (CIs) of total mortality associated with 11-year change in body mass index (BMI) in three

and the second second





and a second second second

Appendix

Appendix 1

Questionnaires used in HUNT 1

A 1.1 Questionnaire 1

A 1.2Questionnaire 2 (English translation found in¹⁶⁷)

A 1.3 Questionnaire 3

Appendix 2

Questionnaires used in HUNT 2

A 2.1 Questionnaire 1

A 2.2 Questionnaire 2

A 2.3 Questionnaire 3

Appendix 1

Questionnaires used in HUNT 1

A 1.1 Questionnaire 1

A 1.2Questionnaire 2 (English translation found in^{167})

A 1.3 Questionnaire 3

MELDING OM SKJERMBILDEFOTOGRAFERING UNDERSØKELSE AV BLODTRYKK OG BLODSU	OG IKKER	Skjermbildefotograferingen kommer nå til ditt distrikt. Denne gangen inngår fotograferingen i en større helse- undersøkelse, og vi viser til orienteringen som er gltt i den vedlagte brosjyre.
		Tid og sted for frammøte vil du finne nedenfor.
		Vennligst fyll ut spørreskjemaet på baksiden og ta det med til undersøkelsen. Ta også med skjermbildebevis, tuberkulinkort eller helsebok om du har.
		Det er viktig at du møter fram selv om du nylig har fåti kontrollert blodtrykk eller blodsukker, og selv om du er under behandling for høyt blodtrykk eller for sukkersyke.
		Med vennlig hilsen
1	I	Statens skjermbildefotografering Postboks 8155 Dep, Oslo 1
		Fylkeslegen Helserådet Statens Institutt For Folkehelse
		Kratsor

Født dato	Personr.	Kommune		Kreusiv.	
Møtested			Første bokstav øtlemavn Dag og dato	Klokkesiett	

·

 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L
 L</t

				na an a
A.	Hvordan er helsa di for tida?	Î		SEGRUETAVIBLODTRYKKSMALINGEN I DEN VEDLAGTE BROSJYREN
	(Sett kryss i bare en rute.)			
	Dêvlia	. —		JA NEJ VET
	Dårligs Ikke helt god		1 2	
	God		2	i. Er blodtrykket ditt målt noen gang før?
	Svært god		4	
B	Har du i løpet av de siste 12 måneder vært hos			J. Hvilket år ble blodtrykket målt siste gang?
.	The day oper av de alste 12 manader vært nust	JA	NEI	19 vet ikke
{	Almenpraktiserende lege (distriktslege, privat-			
	praktiserende lege,turnuskandidat)s			Skriv ârstallet her (ca.)
}	Bedriftslege 5 Militærlege 5	3		K. Hvor ble blodtrykket målt siste gang?
ļ	Lege ved sykehus (uten at du var innlagt) s			(Sett kryss i bare en rute.)
1	Annen lege			
i i			, ,	Hos almenpraktiserende lege (distriktslege, privat- praktiserende lege, turnuskandidat
		JA	NEI	Hos bedriftslege 2
c.	Har du vært innlagt i sykehus de siste 5 åra? s	6		Hos militærlege 3
		Ì		På sykehus 4
D.				Hos annen lege 5 Vet ikke
1	biodtrykk? 5	7 L		
			······	L. Hva ble resultatet av målingen? (Sett kryss i bare en rute.)
E.	Har du eller har du hatt noen av disse sykdommene?	JA	NEI	Jeg skulle begynne med eller fortsette med
	-			medisin for høyt blodtrykk
	Sukkersyke		$\left\{ - \right\}$	Jeg skulle komme til kontroll, men skulle ikko
	Hjerteinfarkt51 Angina pectoris (hjertekrampe)68		+	ta medisin 2
	Hjerneslag eller hjerneblødning		\square	Jeg skulle ikke ta medisin og ikke komme til
				M. Dersom denne helseundersøkelsen viser at du bør undersøkes nærmere: Hvilken almenprak-
ĺ				tiserende lege ønsker du da å bli henvist til?
F.	Har du noen langvarig sykdom, skade eller li- delse av fysisk eller psykisk art som nedsetter	JA	NEI	Skriv navnet på legen her
	ding funksioner i ditt daglige liv? (Med langvarig			
ļ	menes at det har vart, eller vil vare i minst ett år.) 62	ــــا ۱		
				Ingen spesiell lege 78
	Hvis «JA», vil du si at dine funksjoner er litt, middels eller mye nedsatt?		ILS MYE	OMARTELET AND A CONTRACTOR
	Er hovogoloophommot		1	
	Er bevegelseshemmet		-	N. Er du i arbeid for tida? (Sett kryss i bare <i>en</i> rute.)
	Har nedsatt hørsel			Ja, heltidsarbeid (utenom husarbeid)
	Hemmet pga. kroppslig sykdom	┊┝╾┽╍		Ja, dellidsarbeid (utenom husarbeid) 2
	Hemmet pga. psykiske plager 67	· L		Ja, heltids husarbeid 3
				Nei, ikke i arbeid 4
		AL	NEI	
-			·	O. Hvis du ikke er i heltids arbeld, er det på grunn av: (Sett kryss i bare en rute.)
G,	Har du noen søsken? (Nålevende eller døde) 68			Arbeidsløshet, permittering 82
	Hvis «JA», har en eller flere av dem hatt noen av disse sykdommene?	JA N	EI IKKE	Pensjon eller trygd
	Sukkersyke 69			Utdanning eller militærtjeneste 3
	Hjerteinfarkt/hjertekrampe	1		Annet 4
	Forhøyet blodtrykk 71			WAS DUENI AND TO AVAILUDE TRANSPORT
		1		MAS DUELH ALEED, VENNUGEREVARIA DE NESHE ID SRUEHATENE
H.	Non du toution at knowlow du kay dat toutta			P. Er det mye stress og mas på arbeidet ditt?
• •	Når du tenker på hvordan du har det for tida, er du stort sett fornøyd med tilværelsen, eller			(Sett kryss i bare en rute.)
	er du stort sett misfornøyd? (Sett kryss i bare en rute.)		ł	Nei, ikke i det hele tatt 83 1
•	-			
	Svært fornøyd		,	Ja, en god del 3 Ja, nesten hele tida 4
	Meget fornøyd		1	Q. Kan du sjøl bestemme hvordan arbeidet ditt
	Ganske fornøyd			skal legges opp? (Sett kryss i bare en rute)
	Bâde/og	·	4	Nei, ikke i det hele tatt 84 1
	Noksâ misfornøyd			1 liten grad 2
	Meget misfornøyd Svært misfornøyd			Ja, stort sett
		' ï		Ja, det bestemmer jeg sjøl 4

		RØYKEVANER	incerci (Mi	·····
Vi takker for frammøtet til undersøkelsen.	inment			
VI vit også be deg være vennlig å fylle ut dette spørresk Opplysninger vil bli brukt i et større forskningsarbeid om forhi har betydning for helsen.	jemaet. old som	Røyker du daglig for tiden?		JA NI
Svar etter beste skjønn. Kryss av for bare en av svar-mulig (dersom det ikke star nevnt noe annet) Det utfylte skjørn neres i vedladte svarkonvolutt. Porto er betall.	shetene a retur-	Hvis du svarte «JA», røyker du DAGLIG for tiden:		JA N
Alle opplysningene er underlagt streng taushetsplikt.		Sigaretter?		
		Pipe?	- 11	\vdash
Med hilsen Statens skjermbildefotografering		Sigarer (eller serutter/sigarillos)?	50 1	بطبيه
Fylkeslogen Helseradet Statens Institutt For Folkehelse Institutt for anvendt sosialvitenskapelig forskning/ Institutt for samtunnsforskning		Hvis du IKKE røyker SIGARETTER daglig for tiden: Har du røykt SIGARETTER daglig	ļ	N AL
Navn:		tidligere?	21	ـــبــا
Adr. :		Hvis du svarte «JA», hvor lenge er det siden du sluttet å røyke sigaretter daglig?		
		Mindre enn 3 måneder	,, -	η,
Declar		3 måneder- 1 år	. [2
Postnr. Postkontor		1~5 år		- 2
F.nr. :		Mer enn 5 år		4
MOSJON		Hvis du røyker SIGARETTER daglig nå, eller har gjort det tidligere:		۲
í mosjon mener vi at du f.eks. gär tur, går på ski,		Hvor mange sigaretter røyker eller røykte du pr. dag? (Oppgi antall pr. dag medregnet håndrullede)	23	
mmer eller driver trening/idrett.		Besvares av dem som røyker daglig nå		Anta
ofte driver du mosjan? gjennomsnilt)		eller har røykt daglig tidligere: (Gjelder både sigarett-, pipe- og sigar-røykere)		2
ri,		Hvor gammel var du da du begynte å røyke daglig?	25	
nere enn en gang i uka	2	Hvor mange år tilsammen har du røykt daglig?	27	
in gang i uka	3	The mange of manniner for we write we gog.	-	
2–3 ganger i uka Omtrent hver dag		·	ŀ	
Online in the day				
· · · · · · · · · · · · · · · · · · ·	· ·	ALKOHOLBRUK		n ny ny di Ta
som du driver slik mosjon så ofte som en r flere ganger i uka: r hardt mosjonerer du?	43.1	4 Hvor otte har du drukket alkonol (Ø), Vin		363
gjennomsnift)	ند رند ا	eller brennevin) de SISTE 14 DAGENE?	[. ⁻	
Tar det rolig uten å bli andpusten eller svett	13 13 11 2		`	er Ser Ser Ser
Tar det så hardt at jeg blir andpusten og svett		Jeg har ikke drukket alkonol, men	-	
Tar meg nesten helt ut	3	er ikke totalavholdende	29	1
or lenge holder du på hver gang? et gjennomsnitt)	. · ·	Jeg har drukket 1~4 ganger Jeg har drukket 5-10 ganger		·····
Mindre enn 15 minutter	, h	Jeg har drukket mer enn 10 ganger Jeg er totalavholdende, drikker aldri alkohol	Ľ	
16-30 minutter	14		E.	
30 minutter-1 time			l r	
Mer enn 1 time		and the man det inter ar de noen gang nar inter		
SALT	ny ent tañ sa		30	لىسىيە مەربى
r ofte bruker du salt kjøtt eller salt		drukket for mye, eller i hvert fall i meste laget?		
sild til middag?		Nei	۳F	
Aldri, eller sjeldnere enn en gang i måneden	15 1		Ľ	
1-2 ganger i måneden	2		F	
Opptil en gang i uka	3			
Opptil to ganger i uka				
Mer enn to ganger i uka or ofte pleier du å strø ekstra salt på ddagsmaten?	- سبب ا			
•	" - ₁ .			,
Sjelden eller aldrr	16 1			,
Av og til				
00+				
Ofte Alltid eller nesten alitid				

,

 A set of the set of the first set of the s					1999 - 1999 1997 - 1997 1997 - 1997	م میں پر معامل کر میں اور معامل اور ایک میں میں اور
BOSITUASJONEN		Hvis du er i arbeid (gjelder også heltids husarbeid),				
Bor du alene eller sammen med andre? Kryss av for de du bor sammen med. (Her kan du sette flere kryss.)	n de la composition de la comp	ber vi deg fylle ut de neste spørsmålene: Er arbeidet ditt så fysisk anstrengende at du ofte er sliten i kroppen etter en arbeidsdag?				
Bor alene		Ja, nesten alltid	45		1 2	
Ektefelle sller samboer 33 Foreldre eller svigerforeldre 34 Andre voksne personer 35		Ganske sjelden Aldri, eller nesten aldri	ļ		3	
Barn under 5 är						
Barn over 15 är	JA NEt	Krever arbeidet ditt så mye konsentrasjon og oppmerksomhet at du ofte føler deg utslitt etter en arbeidsdag?				
Bor du fast i institusjon? (sykehjern, aldershjern eller liknende)		Ja, nesten alltid Ganske ofte Ganske sjelden	46		1 2 3	
UTDANNINGEN		Aldri, eller nesten aldri			4	
Hvilken utdanning har du fullført? Oppgi bare høyest fullførte utdanning.		Hvordan trives du alt i alt med arbeidet ditt?		<u>.</u>	, , , , , , , , , , , , , , , , , , ,	
7-årig folkeskole eller kortere	1	Veldig godt Ganske godt Godt	47		1 2 3	
9-årig grunnskole Real- eller middelskole, grunnskolens 10. år		lkke særlig godt Därlig			4 5,	
Ett- eller to-ärig videregående skole Artium, økonomisk gymnas eller almenfaglig retning i videregående skoler		Hvis du er gårdbruker eller annen selvstendig				
Høyskole eller universitet, mindre enn 4 år	- 7.5 p	næringsdrivende, har du noen ansatte som arbeider fast for deg? Ingen fast ansatte	48	line'n'	1. 1.	
Har du fullført annen heldags utdanning, og i tilfelle i hvor mange år?		1-2 fast ansatte 3-10 fast ansatte			, 2 3 4	
Skriv antali år her 41	âr Ale a	•Mer enn 10 fast ansatte .				
ARBEID		HVORDAN HAR DU DET?		- 	f	
Hvis du er eller har vært i inntektsgivende arbeid, kan du angi hvilken av disse yrkesgruppene ditt yrke faller innenfor? (Hvis du ikke er i arbeid nå, svarer du ut fra det yrket du hadde sist.)		Når du tenker på hvordan du har det for tida, er du stort sett fornøyd med tilværelsen, eller er du stort sett misfornøyd?				
Hvis du har en ektefelle (eller samboer) som er i inntektsgivende arbeid nå, eller har vært det tid- ligere, angi tilsvarende hvilken yrkesgruppe han/ hun tilhører. (Evt. angi om han/hun ikke har hatt inn-	Deg selv Ekteleilen	Svært fornøyd Meget fornøyd Nokså fornøyd			1 2 3	
tektsgivende arbeid.) Spesialarbeider, ufaglært arbeider		Noksa fornøyd Både - og Nokså misfornøyd			4	
Fagarbeider, håndverker, formann		2 Meget mistornøyd Svært mistornøyd			6 7	
offentlige tjenester) Fagfunksjonær (f.eks. sykepleier, tekniker, lærer) Overordnet stilling i offentlig eller privat virksomhet		s Føler du deg stort sett sterk og opplagt, eller trett og sliten?				
Gårdbruker eller skogeier Fisker		6 Meget sterk og opplagl	50		1 "	
Selvstendig i akademisk erverv (f.eks. tannlege, advokat)		8 Sterk og opplagt 8 Ganske sterk og opplagt			2	
Selvstendig næringsdrivende (Industi, transport, handel) Har <i>ikke</i> hatt inntektsgivende arbeid		9 Ganske trett og sliten Trett og sliten			4 5 6	
(f.eks. pga. heltids husarbeid, studier, trygd)		0 Svært trett og sliten			7	
					÷	
L <u></u>						

MEDISIN/PLAGER				HVORDAN ER DU?
Har du vanligvis:		<u>_</u>	NEI	
Hoste om morgenen?	51			Har du tendens til å ta dine oppgaver mer alvorli enn folk flest?
Oppspytt fra brystet om morgenen?	52			Ja, nettopp slik er jeg
-Fr-F)	<i>~~</i>			Ja, stort sett
Hvor ofte har du brukt smertestillende medisin den siste måneden?				Både - og Nei, stort sett ikke
Daglig				Nei, tvert imot
Hver uke, men ikke hver dag	53		1	
Sjeldnere enn hver uke			3	
Aldri			4	Har du i løpet av det siste året ofte følt at du har presset deg, eller stadig drevet deg selv framover?
Hvor ofte har du brukt avslappende/beroligende medisin eller sovemedisin den siste måneden?				
Daglig	54		۱.	Føler du deg alltid under tidspress, også når det gjelder daglige gjøremål?
Hver uke, men ikke hver dag			`2	også när det gjelder dagnge gjøremar:
Sjeldnere enn hver uke			3	Alltid, eller nesten alitid
Aldri	ł		4	Noen ganger Aidri
Har du i løpet av siste måned vært plaget av				multi
nervøsitet (irritabel, urolig, anspent eller rastløs)?				Er du vanligvis glad eller nedstemt?
Nesten hele tida	55		1	
Ofte	ļ		2 :	Svært nedstemt
Av og til	ŀ		3	Noksā nedstemt
Aldri	ł	J	4	Både ~ og
Har du i løpet av siste måned hatt innsoving-				Nokså glad
eller søvnproblemer?				Glad Svært glad
Nesten hver natt	56		1	Svært giða
Ofte Αν οg til	ŕ		2	
Av og til	Ī		3	
Har du i det store og hele en rolig og god				HVA ER VIKTIG?
følelse inne i deg?				Synes du det er viktig at man prøver å være
Nesten hele tida	57		1	fornøyd med det man har?
Ofte	ŀ		2	Dette er særlig viktig
Av og til	ţ		3 4	Dette er viktig
	Ì			Bâde - og
	1			Dette er mindre viktig Dette er overhodet ikke viktig
VENNER/HJELP				
Dersom du ble syk og måtte holde senga i lengre	-			Synes du det er viktig at man kan slå av på kravene?
tid, hvor sannsynlig tror du det er at du kunne fâ nødvendig hjelp og støtte av familie.	l			Dette er særlig viktig
venner eller naboer?	{			Dette er viktig
Svært sannsynlig	58		1	Både - og Dette er mindre viktig
Nokså sannsynlig	-		2	Dette er overhodet ikke viktig
Usikkert	ŀ		3	
Usannsynlig		-	4	Synes du det er viktig at man alltid er i godt humør?
Helt usennsynlig	ľ	لسبسا	5	Dette er særlig viktig
				Dette er viktig
Hender det ofte at du føler deg ensom?				Bâde - og
Meget ofte	59		1	Dette er mindre viktig
Ofte	ł		2	Dette er overhodet ikke viktig
Av og til	ł		3	
Meget sjelden Aldri	ŀ		4 5	[
	ľ	ليبسب	~	1
				Tusen takk for den hjelp du har gitt oss

TILLEGGS-SKJEMA OM BLOD	TRYK	(K	Hvis du har brukt medisin for blodtrykket før,		
På skjemaet du leverte ved helseundersøkelsen, svar			men ikke nå: Når slutta du med medisiner? (Skriv årstallet i ruta)		
eller har brukt, medisin for høyt blodtrykk.			-	19	
I Nord-Trøndelag har det siden 1980 pågått en und blodtrykksbehandling. Formålet ved undersøkelsen handlingen bedre. En viktig del av undersøkelsen lysninger om hvordan du og alle andre med høyt blod og hvilke erfaringer dere har gjort.	erågjør eråfå	re be- i opp-	Vet ikke …		
Det er derfor meget viktig at du fyller ut dette skje som mulig.	maet så	пøуе	Hvorfor slutta du med medisinene? (Sett ett eller flere kryss)		
Enkelte spørsmål kan være vanskelig å svare på. Prøv etter beste skjønn, og legg vekt på det som er vanlig snittlig for deg.	likevel å eller gjer	svare mom-	Legen bestemte det Jeg fikk plager av medisinene		
Alle opplysninger blir behandlet av oss med streng ta	aushetso	likt	Jeg mente det ikke var nødvendig med medisiner		
På forhånd takk!			Jeg var redd medisinene var skadelige Annen årsak (skriv hvilken nedenfor)		
Når ble det påvist at du hadde høyt blodtrykk			Skriv hvilken årsak det evt. var	89	fkke skriv her.
ørste gang? (Skriv årstallet i ruta)	19	2011 - 1211-1211	Univ Hvirkut disak Uel evi, var		
Vet ikke .			Har legen gitt deg andre råd i forbindelse med at du har for høyt blodtrykk? (Sett kryss i bare <i>en</i> av rutene)		
H vor ble det pävist? (Sett kryss i bare <i>en</i> av rutene)		1983년 1983 1984 - 1984	Net		
Hos almenpraktiserende lege (distriktsiege,			Nei Ja		t
privatpraktiserende lege, turnuskandidat) Hos militærlege På sykehus		2	Husker ikke		3
Vet ikke		4	Hvis «JA»; Hvilke råd?	-	
Bruker du medisin for blodtrykk nå?	70		· · · · · · · · · · · · · · · · · · ·	92 94	ikke skriv her
lvis «NEI»: Gå til de to siste spm. nederst til venstre.			Hvordan opplever du behandlingen for		
ivis «JA»: Når begynte du med medisiner for bodtrykket? (Skriv årstallet i ruta)	19		blodtrykket? Gir det deg: (Sett ett eller flere kryss)		
Vet ikke .	71		Letielse, ro, trygghet	96	
		JA NEI	Anspenthet, engstelse, redsel, uro	97	
ruker du doserings-eske for tabletter?	220		Dårlig humør, depresjon,		
lar du medisinkort som viser va slags medisin du skal ta?			Ingen spesielle følelser	99	
Hender det at du glemmer å ta medisinene? Sett kryss i bare en av rutene)			Synes du at det er noen ulemper ved det at du må ha behandling for høyt blodtrykk?		
Aldri			Nei, ingen ulemper Ja	100	
Sjelden (ca. en gang i mnd.) Oftere		2	Hvis «JA»: Hva synes du er mest plagsomt?		
lvor viktig mener du at det er for deg at du ta lodtrykksmedisinen(e) akkurat som foreskrev Sett kryss i bare <i>en</i> av rutene)			(Sett ett eller flere kryss)		
Ikke så viktig	. 74		At du må bruke medisiner hver dag At du må gå til legekontroll		
Viktig			At du mâ følge de råd som legen har gitt		
Meget viktig		3	At du har ubehag av medisinene	104	
et du hva blodtrykket ditt var ved siste kontro sett kryss i bare en rute)	oll?		At du er engstelig for at det er noe alvorlig som feiler deg	105	
Nei			At du synes det er leit å bli betraktet som «pasient»	106	
Ja Usikker		2	Annet	107	
lvis «JA» eller «USIKKER», kriv hvor mye du tror det var:	76				
	79	Ikke skriv her			
	79				10 St. 18 St. 19

TILLEGGS-SKJEMA FOR SUKKERS	SVI	E	Om du bruker sprøyter, hva heter den insulinen du bruker?		44 1 - 23 - 1
Du har opplyst at du har sukkersyke. Et viktig mål for he søkelsen er å finne ut hvordan sukkersyke best kan beh å gi minst mulig plager.	eiseu	nder-	(Skriv navnet som står på glasset, begge dersom du bruker to sorter).		20
Alle som har eller har hatt sukkersyke, bes derfor om å sva som mulig på disse spørsmålene om sukkersyke.	re si	gođt		128	ikke skr
Noen har svart på et lignende skjema høsten 1982. Det er	like	elav		130	ليبا آ هل
stor betydning at disse fyller ut dette skjemaet. Alle opplysninger blir behandlet av oss med streng taushe	etsol	d.	Bruker du tabletter mot sukkersyken?	132	· []
På forhånd takk!					
		\neg	Om du bruker tabletter mot sukkersyken, skriv neden- for hva de heter, antall mg. som står på glasset/ pakningen og hvor mange slike tabletter du tar hver dag (Skriv om begge sorter dersom du bruker mer enn en	;:	
år ble sukkersyken din oppdaget? 19	108		type tabletter mot sukkersyke)		
kriv årstallet i ruta)		, e , , ; , ;	Skriv navn på tablotlen her mg. pr. tabl. antall pr. dag	139	2
vordan ble sukkersyken din oppdaget?			Skhv navn pa tabletten ner ing, pr. ceor. anter pr. veg		<u>ب</u> چېږېچ کړ
Jeg søkte lege på grunn av symptomer	110			146	ikke
Ble oppdaget uten at jeg hadde symptomer (ved legeattest, bedriftskontroll, undersøkelse for annen sykdom i eller utenfor sykehus)			Skriv navn på tabletten her mg. pr. tabl. antali pr. dag		** . .
,		은 VET NAS 이 가격	Hvor mange måltider spiser du hver dag?	147	۵۹
va slags plager hadde du i tilfelle da ukkersyken ble oppdaget? (kryss evt. i flere ruter).			Føler du at du vet nok om hva slags mat du kan spise?	148	JA
Ingen plager			Hvis du skal svare på hva du virkelig spiser. og		
Unormal tørste		-	ikke hva legen din har sagt du bør spise. Vli		
Stor vannlating		-13.33	du da si at du: (Kryss av bare i den ruta som kommer nærmest det du virkelig gjør)		
Slapphet		- 1833			
Vekttap			Spiser stort sett det samme som de som ikke har sukkersyke	149	
Underlivskløe					
Andre plager	117		Spiser hva jeg vil unntatt sukker og søtsaker		- 72
Hvis «ANDRE PLAGER», skriv hvilke:			Bruker på øyemål bestemt mengde brød, potet, melk og frukt		
artic particular and a state of the particular and the particular and the particular and the particular and the	118	ikke skriv her	Veier/måler bestemt mengde brød, potet, melk og evt. frukt en eller flere dager i uka		
	120				
		JA NEI	Kontrollerer du hjemme hvor mye sukker du har i urinen?(Kryss av også om noen hjelper		JA
lar noen av dine foreldre, søsken eller arn hatt sukkersyke?	122		deg eller gjør det for deg)	150	
lvis «JA», bruker eller brukte noen av			Hva heter den metoden du i tilfelle bruker til â mâle sukker i urinen?		
isse insulinsprøyter?	123			151	
			Skriv navnet som står på pakningen her		JA
BEHANDLING			Kontrollerer du noen gang hjemme hvor mye sukker du har i blod (blodsukker)?		1 7-11
		JA NEI	(Kryss av også om noen hjelper deg eller gjør det for deg)	152	
ruker du insulinsprøyter mot sukkersyken?	124		Hva heter den metoden du i tilfelle bruker til å måle blodsukker?		
lvis «JA», bruker du sprøyter daglig?				153	
Sprøyte en gang daglig	125		Skriv navnet på pakningen og navn på evt. apparat du mårer med.		
Sprøyte en gang daglig Sprøyte to eller flere ganger daglig	14	2	Hvis du selv kontrollerer sukker i urin eller blod, hvor ofte gjør du det?		
			(Kryss av også om noen hjelper deg eller gjør det for deg)		
Om du bruker sprøyter, hvor mye insulin ar du tilsammen hver dag?			Hver dag	154	
ar du tilsammen nver dag r Skriv antall mili ruta – 1 «strek» svarer til 0,1 ml)	120		2-3 dager i uka		
· · ·		ml	En dag i uka		$\left - \right $
			En dag hver 14. dag		\vdash
			En dag i måneden		$\left - \right $
			Sjeldnere enn en dag i måneden		<u> </u>

 A state of the second second second second second second second second second second second second second second second second second second second seco second second sec					2 - 2 	
		JA NEI	Har du selv hatt noen vedvarende (kroniske) plager etter at du fikk sukkersyke?	191	lkko sitriv	, h¢
Hvis du selv kontrollerer sukker i urin eiler blod: måler du flere ganger om dagen de dagene du gjør det?	155		(Skriv hva slags sykdom/plager på linjene under).	193 193 195		
		· .		197 199		
Dersom du tar urin- eller blodprøve selv, tar du resultatene med til legen ved kontroll? (kryss av i den ruta som passer best)				201		
Aldri	156		UNDERVISNING - STØTTE			-
Av og til Oftest		2	Fu du modiere es Novere l'andata dunad		JA N	EI C
Alhid		4	Er du medlem av Norges Landsforbund for Sukkersyke?	203]. ?::
		JA NEI	Har du noen gang deltatt på kurs eller møte om sukkersyke?	204		
Går du til regelmessig kontroll hos lege for sukkersyken din?	157		Får du grunnstønad gjennom trygdekontoret for sukkersyken?	205		
Hvis «JA», hvor lenge var det mellom de to siste gangene du var hos legen din til kontroll for sukkersyken?			Har du søkt om og fått særfradrag i skattelikninga fordi du har sukkersyke?	206		
Antall måneder (skriv i ruta)	158	mindr.	HVORDAN HAR DU DET?			
Hva slags lege går du til kontroll hos for sukkersyken? (Sett kryss i bare <i>en</i> rute)			Synes du det er vanskelig å ha sukkersyke? (kryss av i den ruta som passer best).			
Vanlig lege (distriktslege, aimenpraktiserende lege, bedriftslege osv.)	160		Ja, jeg føler det er som en plage hver dag Ja, jeg tenker ofte på det	207	- 2	
Sykehuslege (poliklinikk på sykehus) Er innlagt i sykehjern eller annen institusjon			Ja, av og til Nei, sjelden		3)	
og får kontroli der Andre		3	Nei, jeg tenker nesten aldri på det Føler meg akkurat som alle som ikke har sukkersyke			
		licke skriv her	Dersom du synes det er vanskelig å ha sukker- syke, hva synes du er verst? (Skriv det du mener på linja nedenfor).			
Hvis «andre», skriv hva slags lege på linja over	161				ikke skriv	he
ANNEN SYKDOM			Skriv her		Stead of the second	
Bruker du regelmessig medisin for annet enn sukkersyken?	. 162		Forteller du til andre at du har sukkersyke? (kryss av i den ruta som passer best).			
Dersom «JA», skriv hva disse medisinene heter			Ja, alltid nâr jeg mener de bør vite det Ja, men bare om de spør	210	1	
(Skriv det navnet som står på glasset eller pakningen. Ta med alle sortene du bruker regelmessig. Skriv x bak navnet om du brukte dette også før du fikk sukkersyke).	163	likke skriv her	Nei, helst ikke Jeg er redd for at andre skal få greie på det		30	
•	166 169					年間の後
	172				JA N	EI
	175 178		Har du noen gang hatt for lavt blodsukker? («føling», «insulinsjokk»)	211		׀ ֛
Tror du man er mer utsatt for å få enkelte andre sykdommer dersom man har dåvis kentrellert auktoruter?	. 181		Hvis «JA», hvor mange ganger har du hatt det den siste uka? (Skriv antall ganger i ruta)	212		
dårlig kontrollert sukkersyke?		ANY SUR	Hvor mange ganger har du vært innlagt i syke- hus de siste 5 årene? (Skriv antall ganger i ruta)	213		
Hvis «JA», nevn navnet på 3 slike sykdommer: (Du behøver ikke å ha hatt disse sykdommene selv).			Dersom du har ligget i sykehus de siste 5 årene, hva har du ligget der for? (Skriv på linjene nedenfor)			
	185	lkké skriv her		214	lkke skriv	. 10
	187 189			216 218		

n na 199 An Angeland (1997) An

Appendix 2

Questionnaires used in HUNT 2

A 2.1 Questionnaire 1

A 2.2 Questionnaire 2

A 2.3 Questionnaire 3

HELSEUNDERSØKELSEN I NORD-TRØNDELAG

ſ



Personlig innbydelse



Spørreskjemaet er en viktig del av Helseundersøkelsen. Her finner du spørsmål om tidligere sykdom og om andre forhold som har betydning for helsa. Vennligst fyll ut skjemaet på forhånd og ta det med til Helseundersøkelsen. Dersom enkelte spørsmål er uklare, lar du dem bare stå ubesvarte til du møter fram, og drøfter dem med personalet som gjennomfører undersøkelsen. Alle svar vil bli behandlet strengt fortrolig.

Flere steder i skjemaet ber vi deg oppgi din alder da eventuell sykdom inntrådte. Hvis du ikke husker nøyaktig hvor gammel du var, skriver du et tall som er nærmest det du antar er korrekt.

Når resultatene fra undersøkelsen foreligger, vil det være enkelte som trenger ny undersøkelse hos egen lege. Dette vil du få beskjed om i det brevet som vi sender deg om dine resultater. Samtidig sender vi melding om resultatene dine til legen din. Det er derfor

om å gjøre at du i rubrikken helt til slutt i skjemaet oppgir navnet på den allmennpraktiserende lege, kommunelege eller det helsesenter som du ønsker skal ta hånd om eventuell etterundersøkelse, og som vi skal sende resultatene til.

		Med vennlig hilsen		
Helsetjenesten i Nord-Trøndelag	٠	Statens helseundersøkelser	٠	Statens Institutt for Folkehels

DET HANDLER OM HELSA DI	STOFFSKIFTE
Hvordan er helsa di nå?	JA NEI Alder Jarste gang
	nar ou noen gang ian pavisi.
Bare ett kryss Dårlig	IUI IIØYI SIDIISKINE
During minimum	
Ikke helt god 2 God 3	struma 42 år
Svært god	annen sykdom i skjoldbruskkjertelen
0.00.1.get	Bruker du eller har du brukt
LUFTVEGSPLAGER	noen av disse medisinene:
JA NEI	
Hoster du daglig i perioder av året?	Neo-Mercazole 51 ar
Hvis JA:	Er du operert i skjoldbruskkjertelen <u>år</u>
Er hosten vanligvis ledsaget av oppspytt? 14	Har du fått radiojodbehandling 57
Har du hatt hoste med oppspytt i minst 3 mnd.	MUSKEL/SKJELETT-PLAGER
sammenhengende i hvert av de to siste åra?	Har du i løpet av det siste året vært plaget
	med smerter og/eller stivhet i muskler
Har du hatt noe anfali med pipende eller	og ledd som har vart i minst 3 måneder JA NEI
tung pust de siste 12 måneder? 16	sammenhengende? 60
Alder Alder	-
JA NEI forste gang	Hvis NEI, gå videre til neste side øverst. Hvis JA, svar på følgende:
Har du eller har du hatt astma? 17	Illuse has du hatt disso plegene?
Har du brukt eller bruker du JA NEI	Nakke 61
astmamedisiner? 20	Skuldre (aksier)
	Albuer
HJERTE-KARSYKDOMMER, DIABETES	Håndledd, hender
JA NEI Alder første gang	Bryst/mage 65
Har du, eller har du natt:	Øvre del av ryggen
	Korsryggen
Angina pecions (njenekranipe) 4	Hofter
Hjerneslag/hjerneblødning 27	Knær
Diabetes (sukkersyke) 30	Ankier, føtter
	Hvis du har hatt plager i flere områder i minst 3 mnd. det siste året,
Hva ble resultatet siste gang du målte blodtrykket ditt?	setter du ring rundt det ja-krysset hvor plagene har vart lengst
Bare ett kryss	Hvor lenge har plagene vart sammenhengende?
Begynne med/fortsette med blodtrykksmedisin 33 🛄 1	Svar for det området hvor plagene har vart lengst Antali mnd.
Komme til kontroll, men ikke ta blodtrykksmedisin	Hvis under 1 år, oppgi antali mnd 71
Ingen kontroll og ingen medisin nødvendig 3	Antail år
Har aldri fått målt blodtrykket 🔲 4	Hvis 1 år eller mer, oppgi antall år 73
m for the second state of the s	
Bruker du medisin mot høyt blodtrykk?	Har plagene redusert din arbeidsevne det siste året?
Bare ett kryss Nå	Gjelder også hjemmearbeidende. Bare ett kryss
Na	Nel/ubetydelig I noen grad I betydelig grad Vet ikke
Aldri brukt	
	JA NEI KKET
How an other flows on foundary other products	Har du vært sykmeldt pga. disse JA NEL ARBEID plagene det siste året?
Har en eller flere av foreldre eller søsken hatt hjerteinfarkt (sår på hjertet) eller JA NEI VET	
hatt hjerteinfarkt (sår på hjertet) eller JA NEI IKKE angina pectoris (hjertekrampe)?	Har plagene ført til redusert aktivitet i fritida?
anyma perions (njenemanipe):	

Har lege noen gang sagt at du har/har hatt noen av disse sykdommene:	Røykte noen av de voksne hjem da du vokste opp?
Beinskjørhet (osteoporose)	Bor du, eller har du bodd, samm dagligrøykere etter at du fylte 20
Slitasjegikt (artrose) Bechterews sykdom	Hvor lenge er du vanligvis dagli til stede i røykfylt rom? Sett 0 hvis du ikke oppholder deg i røykfy
Andre langvarige skjelett- eller muskelsykdommer	Røyker du selv? Sigaretter daglig? Sigarer/sigarillos daglig? Pipe daglig? Aldri røykt daglig
ANDRE PLAGER	lenge er det siden du sluttet?
i hvilken grad har du hatt disse plagene i de siste 12 månedene? Ikke plaget Litt plaget Mye plaget Kvalme	Hvis du røyker daglig nå eller ha tidligere: Hvor mange sigaretter røyker ell røykte du vanligvis daglig? Hvor gammel var du da du begy røyke daglig? Hvor mange år tilsammen har du daglig?
ANDRE SYKDOMMER	KAFFE/TE/ALKOHOL
Har du eller har du noen gang hatt: OK Vict første gang Epilepsi 102 år Psykiske plager hvor du har søkt hjelp år Kreftsykdom 108 Annen langvarig sykdom 111	Hvor mange kopper kaffe/te drik Sett 0 hvis du ikke drikker kaffe/te daglig Kokekaffe Annen kaffe Te
DAGLIGE FUNKSJONER	Alkohol:
Har du noen langvarig sykdom, skade eller lidelse av fysisk eller psykisk art som ned- setter dine funksjoner i ditt daglige liv? 112	Er du total avholdsmann/-kvinn Hvor mange ganger i måneden vanligvis alkohol? Regn ikke med lettøl. Sett 0 hvis mindre
lidelse av fysisk eller psykisk art som ned- setter dine funksjoner i ditt daglige liv? 112 JA NEI Langvarig: minst elt år Hvis JA: Hvor mye vil du si at dine funksjoner er nedsatt? Litt nedsatt Middels nedsatt Mye nedsatt Er bevegelseshemmet 113 III III III Har nedsatt syn III III III III	Hvor mange ganger i måneden vanligvis alkohol? Regn ikke med lettel. Sett 0 hvis mindre Hvor mange glass øl, vin eller b du vanligvis i løpet av to uker? Regn ikke med lettel. Sett 0 hvis du ikke drikker alkohol 153
lidelse av fysisk eller psykisk art som ned- setter dine funksjoner i ditt daglige liv? 112 JA NEI Langvarig: minst elt år Hvis JA: Hvor mye vil du si at dine funksjoner er nedsatt? Litt nedsatt Middels nedsatt Mye nedsatt Er bevegelseshemmet 113 Image: Comparing the second se	Hvor mange ganger i måneden vanligvis alkohol? Regn ikke med lettøl. Sett 0 hvis mindre Hvor mange glass øl, vin eller b du vanligvis i løpet av to uker? Regn ikke med lettøl. Sett 0 hvis du ikke drikker alkohol 153 FYSISK AKTIVITET I FRITIDA
lidelse av fysisk eller psykisk art som ned- setter dine funksjoner i ditt daglige liv? JA NEI setter dine funksjoner i ditt daglige liv? 112 III Langvarig: minst elt år Hvis JA: Hvor mye vil du si at dine funksjoner er nedsatt? Middels Mye nedsatt Er bevegelseshemmet 113 III III Har nedsatt syn III IIII IIII Har nedsatt hørsel IIII IIIIII IIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIII	Hvor mange ganger i måneden vanligvis alkohol? Regn ikke med lettøl. Sett 0 hvis mindre Hvor mange glass øl, vin eller b du vanligvis i løpet av to uker? Regn ikke med lettøl. Sett 0 hvis du ikke drikker alkohol 153 FYSISK AKTIVITET
lidelse av fysisk eller psykisk art som ned- setter dine funksjoner i ditt daglige liv? 112 JA NEI Langvarig: minst elt år 112 Hvis JA: Hvor mye vil du si at dine funksjoner er nedsatt? Middels Mye nedsatt Fue source er nedsatt? It Middels Mye nedsatt Har nedsatt syn It It It Har nedsatt syn It It It Har nedsatt hørsel It It It Hemmet pga. kroppslig sykdom. It It It Hemmet pga. psykiske plager It It It MENN fortsetter øverst neste spalle It Antall barn Hvor mange barn har du født? It Antall barn	Hvor mange ganger i måneden vanligvis alkohol? Regn ikke med lettøl. Sett 0 hvis mindre Hvor mange glass øl, vin eller b du vanligvis i løpet av to uker? Regn ikke med lettøl. Sett 0 hvis du ikke drikker alkohol 153 FYSISK AKTIVITET I FRITIDA Hvordan har din fysiske aktivite
lidelse av fysisk eller psykisk art som ned- setter dine funksjoner i ditt daglige liv? 112 JA NEI Langvarig: minst elt år Hvis JA: Hvor mye vil du si at dine funksjoner er nedsatt? Middels Mye Hvor mye vil du si at dine funksjoner er nedsatt? Litt Middels Mye Har nedsatt syn 113 Imedsatt Imedsatt Imedsatt Har nedsatt syn Imedsatt Imedsatt Imedsatt Imedsatt Imedsatt Har nedsatt hørsel Imedsatt	Hvor mange ganger i måneden vanligvis alkohol? Regn ikke med lettal. Sett 0 hvis mindre Hvor mange glass øl, vin eller b du vanligvis i løpet av to uker? Regn ikke med lettal. Sett 0 hvis du ikke drikker alkohol IS3 FYSISK AKTIVITET I FRITIDA Hvordan har din fysiske aktivitet året? Tenk deg et ukentlig gjennomsnik Arbeidsveg regnes som fritid Lett aktivitet (ikke Ingen svett/andpusten) 159 Hard fysisk aktivitet (svett/andpusten) 160 1 UNDER ARBEID 1
lidelse av fysisk eller psykisk art som ned- setter dine funksjoner i ditt daglige liv? 112 JA NEI Langvarig: minst elt år 112 112 Hvor mye vil du si at dine funksjoner er nedsatt? Middels Mye nedsatt Fue species shemmet 113 112 Har nedsatt syn 113 113 112 Har nedsatt syn 113 113 113 Har nedsatt syn 113 113 113 Har nedsatt syn 113 113 113 Har nedsatt hørsel 113 113 113 Har nedsatt syn 117 113 113 Har nedsatt hørsel 117 114 114 Hemmet pga, psykiske plager 117 118 118 118 MENN fortsætter øverst neste spalle 118 118 118 118 Sett 0 hvis du ikke har født barn 118 118 118 120 118 Hvor gammel var du da du fødte ditt første barn? 120 120 År	Hvor mange ganger i måneden vanligvis alkohol? Regn ikke med lettal. Regn ikke med lettal. Sett 0 hvis mindre Hvor mange glass øl, vin eller b du vanligvis i løpet av to uker? Regn ikke med lettal. Sett 0 hvis du ikke drikker alkohol ISA FYSISK AKTIVITET I FRITIDA Hvordan har din fysiske aktivitet året? Tenk deg et ukentlig gjennomsnik Arbeidsveg regnes som fritid Lett aktivitet (ikke Ingen svett/andpusten) 150 Hard fysisk aktivitet 1 (svett/andpusten) 1 UNDER ARBEID Hvis du er i lønnet eller ulennet arbeid: Hvorledes vil du beskrive arbeid Bare ett kryss For det meste stillesittende arbeid (teks. skrivebordsarbeid, montering)
lidelse av fysisk eller psykisk art som ned- setter dine funksjoner i ditt daglige liv? 112 JA NEI Langvarig: minst elt år Hvis JA: Hvor mye vil du si at dine funksjoner er nedsatt? Middels Mye Hvor mye vil du si at dine funksjoner er nedsatt? Litt Middels Mye Har nedsatt syn 113 Imagesett Imagesett nedsatt nedsatt Har nedsatt syn Imagesett Imagesett nedsatt nedsatt Imagesett nedsatt nedsatt Imagesett nedsatt Imagesett Imagesett nedsatt Imagesett Net Imagesett Imagesett<	Hvor mange ganger i måneden vanligvis alkohol? Regn ikke med lettal. Sett 0 hvis mindre Hvor mange glass øl, vin eller b du vanligvis i løpet av to uker? Regn ikke med lettal. Sett 0 hvis du ikke drikker alkohol 153 FYSISK AKTIVITET I FRITIDA Hvordan har din fysiske aktivite året? Tenk deg et ukentlig gjennomsnik Arbeidsveg regnes som frild Lett aktivitet (ikke svett/andpusten) Hard fysisk aktivitet (svett/andpusten) Hvis du er i lønnet eller ulønnet arbeid: Hvorledes vil du beskrive arbeid: Bare ett kryss For det meste stillesittende arbeid
lidelse av fysisk eller psykisk art som ned- setter dine funksjoner i ditt daglige liv? 112 JA NEI Langvarig: minst elt år Hvis JA: Middels Mye Hvor mye vil du si at dine funksjoner er nedsatt? Litt Middels Mye redsatt nedsatt nedsatt nedsatt IIII IIII IIII IIIII IIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIII	Hvor mange ganger i måneden vanligvis alkohol? Regn ikke med lettal. Sett 0 hvis mindre Hvor mange glass øl, vin eller b du vanligvis i løpet av to uker? Regn ikke med lettal. Sett 0 hvis du ikke drikker alkohol IS3 FYSISK AKTIVITET I FRITIDA Hvordan har din fysiske aktivitet året? Tenk deg et ukentlig gjennomsnik Arbeidsveg regnes som fritid Lett aktivitet (ikke ungen svett/andpusten) Hard fysisk aktivitet (svett/andpusten) Horiedes vil du beskrive arbeid Bare ett kryss For det meste stillesittende arbeid (teks. ekspeditararb, lett industriarb., Arbeid som krever at du går myd
lidelse av fysisk eller psykisk art som ned- setter dine funksjoner i ditt daglige liv? 112 JA NEI Langvarig: minst elt år Hvis JA: Middels Mye Hvor mye vil du si at dine funksjoner er nedsatt? Litt Middels Mye redsatt nedsatt? nedsatt nedsatt IIII IIIII IIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIII	Hvor mange ganger i måneden vanligvis alkohol? Regn ikke med lettal. Sett 0 hvis mindre Hvor mange glass øl, vin eller b du vanligvis i løpet av to uker? Regn ikke med lettal. Sett 0 hvis du ikke drikker alkohol 153 FYSISK AKTIVITET I FRITIDA Hvordan har din fysiske aktivitet året? Tenk deg et ukentlig gjennomsnik Arbeidsveg regnes som frilid Lett aktivitet (ikke svett/andpusten) Hard fysisk aktivitet (svett/andpusten) Hvis du er i lønnet eller ulønnet arbeid: Hvorledes vil du beskrive arbeid Bare ett kryss For det meste stillesittende arbeid (leks. skrivebordsarbeid, montering) Arbeid som krever at du går myd (leks. ekspedilørarb., lett industriarb., Arbeid hvor du går og løfter myde

and a start of the
RØYKING
Røykte noen av de voksne hjemme JA NEI da du vokste opp? 126
Bor du, eller har du bodd, sammen med noen JA NEI dagligrøykere etter at du fylte 20 år? 127
Hvor lenge er du vanligvis daglig Antall timer til stede i røykfylt rom? 128 Sett 0 hvis du ikke oppholder deg i røykfylt rom 128
Røyker du selv? JA NEJ Sigaretter daglig? 130 Sigarer/sigarillos daglig? 132 Pipe daglig? 132 Aldri røykt daglig (Sett kryss)
Hvis du har røykt daglig tidligere, hvor Antall år lenge er det siden du sluttet?
Hvis du røyker daglig nå eller har røykt tidligere: Hvor mange sigaretter røyker eller røykte du vanligvis daglig? hvor mange sigaretter røyker eller røykte du vanligvis daglig? hvor mange sigaretter røyker eller røykte du vanligvis daglig? hvor mange sigaretter røyker eller
røyke daglig? 140
Hvor mange år tilsammen har du røykt Antall år daglig? 142
KAFFE/TE/ALKOHOL
Hvor mange kopper kaffe/te drikker du daglig? Sett 0 hvis du ikke drikker kaffe/te daglig Antail kopper
Kokekaffe 144 Annen kaffe 148 Te 146
Alkohol:
Hvor mange ganger i måneden drikker du vanligvis alkohol? Antall ganger Begn ikke med lettøl. Sett 0 hvis mindre enn 1 gang i mnd.
Hvor mange glass øl, vin eller brennevin drikker du vanligvis i løpet av to uker? <u>øl Vin Brennevin</u>
Regn ikke med lettel. Sett 0 hvis du ikke drikker alkohol 153
FYSISK AKTIVITET
I FRITIDA Hvordan har din fysiske aktivitet i fritida vært det siste året? Tenk deg et ukentlig gjennomsnilt for året.
Arbeidsveg regnes som frilid Timer pr. uke
Lett aktivitet <i>(ikke</i> Ingen Under 1 1-2 3 og mer svett/andpusten) 159 🗌 🔲 🔲 🗍 Hard fysisk aktivitet
(svett/andpusten) 160 \Box_1 \Box_2 \Box_3 \Box_4 UNDER ARBEID
Hvis du er i lønnet eller ulønnet arbeid:
Hvorledes vII du beskrive arbeidet ditt? Bare ett kryss For det meste stillesittende arbeid
(f.eks. skrivebordsarbeid, montering)
(f.eks. ekspeditørarb., lett industriarb., undervisning) 2 Arbeid hvor du går og løfter mye (f.eks. postbud, pleier, bygningsarbeid)
Tungt kroppsarbeid (I.eks. skogsarbeid, tungt jordbruksarb.,tungt bygningsarb.)
Bla oul

HVORLEDES FØLER DU DEG?	UTDANNING
Har du de siste to ukene følt deg:	Hvilken utdanning er den høyeste du har fullført?
Nei Litt del mye	Grunnskole 7-10 år, framhaldsskole,
Trygg og rolig? 162	folkehøgskole 182 🗍 1
Glad og optimistisk?	Realskole, middelskole, yrkesskole, 1-2 årig
	videregående skole
Plaget av angst? 165	Artium, øk.gymnas, allmennfaglig retning i videregående skole
	Høgskole/universitet, mindre enn 4 år
	Høgskole/universitet, 4 år eller mer
Ensom? 166 [] [] [] [] [] [] [] []	ARBEID
the second state of the se	
Her kommer noen flere spørsmål om hvorledes du føler deg. For hvert spørsmål setter du kryss for ett av de fire svarene som best beskriver dine følelser den siste uka. Ikke tenk for lenge på svaret - de spontane	Hva stags arbeidssituasjon har du nå? Ett eller flere kryss
svarene er best	Lønnet arbeid 183
Jeg gleder meg fortsatt over ting slik jeg pleide før 169 Avgjort like mye	Selvstendig næringsdrivende
kke fullt så mye	Heltids husarbeid
Jeg har en uroføielse	Arbeidsledig, permittert
som om noe forferdelig vil skje 170	Pensjonist/trygdet 188
Ja, og noe svært ille 1 Litt, bekymrer meg lite . 3	Uner manage times format ashaid har du Anlail timer
Ja, ikke så veldig ille 2 Ikke i det hele tatt 4	Hvor mange timer lønnet arbeid har du
Jeg kan le og se det morsomme i situasjoner 171 Like mye ná som før □ 1 Avgjort ikke som før □ a	
Ikke like mye nå som før 2 Ikke i det hele tatt 4	Har du skiftarbeid, nattarbeid eller går vakt?
Jeg har hodet fullt av bekymringer 172 Veldig ofte	ALT I ALT
Ganske ofte 2 2 En gang i blant	Når du tenker på hvorden du har det for tida,
Jeg er i godt humør 173	er du stort sett fornøyd med tilværelsen
Aldri	eller er du stort sett misfornøyd?
Noen ganger 2 For det meste 4	Bare ett kryss
Jeg kan sitte i fred og ro og	Svært fornøyd
kjenne meg avslappet 174 Ja, helt klart	Meget fornøyd 2 Ganske fornøyd
Vanligvis	Både/og
Jeg føler meg som om alt går langsommere 175	Nokså misfornøyd
Nesten hele tiden 1 : Fra tid til annen 3	Meget misfornøyd
Svært ofte 2 1kke i det hele tatt	Svært misfornøyd
Jeg føler meg urolig som om jeg har sommerfugler i magen 176	TO DATE OF SALES AND A DATE OF SALES
Ikke i det hele tatt	Hvis denne helseundersøkelsen viser at du bør
Fra tid til annen	undersøkes nærmere, hvilken allmennpraktiserende
Jeg bryr meg ikke lenger om hvordan jeg ser ut 177	lege/kommunelege ønsker du skal foreta under-
Ja, har sluttet å bry meg 1 Kan hende ikke nok 3	søkelsen?
Ikke som jeg burde 2 Bryr meg som før	Skriv navnet på legen her:
Jeg er rastløs som om jeg stadig må være aktiv 178	
Uten tvil svært mye 1 1 kke så veldig mye 13 Ganske mye	
98.	Sand Sand Sand Sand Sand Sand Sand Sand
Jeg ser med glede frem til hendelser og ting 179	7akk for utfyllingen!
Like mye som før	Nok en gang:
Jeg kan plutselig få en følelse av panikk 180	Velkommen til NORD- TRØNDELAG
Uten tvil svært ofte 1 1 Ikke så veldig ofte 3 Ganske ofte	undersøkelsen. TRøndelag
Jeg kan glede meg over gode bøker, radio og TV 181 Ofte	
Offe I i ikke sa offe	
a dans	
What allow in the second second second	

Waster

ie 332 5201 - 50.000 - 09.96

SKJEMA FOR KVINNER 20-69 ÅR

hunt

Helseundersøkelsen i Nord-Trøndelag

Takk for frammøtet til undersøkelsen! Vi vil også be deg fylle ut dette spørreskjemaet. Opplysningene vil bli brukt i større forskningsarbeider om fore-byggende helsearbeid. Noen av spørsmålene likner på spørsmål du har svart på i det skjemaet du fylle ut heime og leverte ved frammøte til helseundersøkelsen. Det er likevel viktig at du svarer på alle spørsmålene også i dette skjemaet. Det utfylte skjemaet returneres i vedlagte svarkonvolutt. Porto er betalt. Alle opplysningene er underlagt streng taushetsplikt.

oppiysningene er undenegt streng tausnerspint. Vennlig hilsen Helsetjenesten i Nord-Trøndelag	Hvis du ikke ønsker å besvare spørre- skjemaet, sett kryss her og returner skjemaet. Da slipper du purring.
Statens Institutt for Golkehelse Statens helseund	ersøkelser Jegønsker ikke å besvare skjemaet ()
UTFYLLING	BOLIG
Dato for utfylling av skjema: / 19 19	Hvem bor du sammen med? Ett kryss for hver linje og angi antall Ja Nel
ODDVEKST	Ektefelie/samboer St. C. Andre personer over 18 år
OPPVEKST	Personer under 18 år
I hvilken kommune bodde du da du fyite 1 år? Hvis du ikke bodde i Norge, oppgi land i stedet for kommune.	Anias
	Hvor mange av barna har plass i barnehage?
24	Hvilken type bolig bor du i? Bare ett kryss
ARBEID	Enebolig/villa 🕫 🖵 1
Nåværende eller tidligere arbeid: Hva slags inntektsgivende arbeid har du og event. din ektefelle/samboer? Hvis du/dere ikke har inntektsgivende arbeid nå: Oppgi det siste yrket. Deg Ektefelle/ selv samboer Spesialarbeider eller ufaglært arbeider	Gårdsbruk 2 Biokk/terrasselellighet 3 Rekkehus/2-4 mannsbolig 4 Annen bolig 5 Hvor stor er din boenhet? 64 Ja Nel 5 Er det heldekkende tepper i stua? 67 Er det heldekkende tepper på ditt soverom? 1 Er det heldekkende tepper på ditt soverom? 1 Er det hund i boligen? 63 Er det andre nelskiedde dvr eller fugler i boligen? 1
Overordnet stilling i off. eller privat virksomhet	Er det andre pelskledde dyr eller fugler i boligen? 🛛 🗌
	OKONOMI
Selvstendig i akademisk erverv (f.eks. tannlege, advokat)	ØKONOMI Mottar du noen av følgende offentlige ytelser? Ja Nei Sykepenger/sykelønn/rehabiliteringspenger 72 Ytelser under yrkesrettet attføring 0 Uførepensjon 74 Alderspensjon 0
Hvis du NÅ ikke har inntektsgivende arbeid eller du ikke har heltids husarbeid: Gå til BOLIG.	Sosialstøtte
Har du i løpet av de siste 12 månedene hatt sykefravær: Ja Nei med egenmelding	Overgangsstønad
Hvis «Ja»: Hvor lenge <i>tilsammen?</i> Bare ett kryss 2 uker eller mindre	Har det i løpet av det siste året hendt at husholdningen har hatt vansker med å klare de løpende utgifter til mat, transport, bolig og liknende? <i>Bare ett kryss</i> si Ja, ofte
Har du i løpet av de siste 12 månedene Ja Nei	
vurdert å skifte yrke eller arbeidsplass? 👳 🗌 🗌	
	VENNER
Er arbeidet ditt så fysisk anstrengende at du ofte er sliten i kroppen etter en arbeidsdag? Bare ett kryss si Ja, nesten alltid	Hvor mange gode venner har du? Regn med de du kan snakke fortrolig med og som kan gi deg god hjelp når du trenger det
Krever arbeidet ditt så mye konsentrasjon og oppmerk- somhet at du ofte føler deg utslitt etter en arbeidsdag? ⁵² Ja, nesten alltid	<i>Ja Nei</i> Føler du at du har mange nok gode venner? 84 Hvor ofte tar du vanligvis del i foreningsvirksomhet som
Hvordan trives du alt i alt med arbeidet ditt? Soldig godt 1 lkke særlig godt 2 Dårlig	f.eks. syklubb, idrettslag, politiske lag, religiøse eller andre foreninger? 85 Aldri, eller noen få ganger i året 🔤 i Omtrent en gang i uka 🔲 1 1-2 ganger i måneden 2 Mer enn en gang i uka 🗌 2
	and the second

DER DU BOR

	DOIN			
Svar ut fra nærmiljøet, dvs. nabolaget/grenda: Ett kryss for hvert spørsmål				
Jeg føler Helt enig		esskap med de Usikker 📋 3	e som bor her Delvis 🗖 4 uenig	' ∺ Helt ⊡ ₅ uenig ⊡ ₅
		atlv, er det ing	en som blir n	ied på
det som s Helt enig	ettes i gang Deivis 🗌 enig	her 67 Usikker	Delvis 🗀 uenlg	Helt 🔲 uenig
		, vil jeg lengte	tilbake as	
Helt 🖂 enig	Delvis enig	Usikker 🗌	Delvis 🗆 uenig	Helt uenig
Man kan l	ikke stole på	i hverandre he	1 1 89	
Helt enig	Delvis 🗔 enig	Usikker 🖂	Delvis 🗆 uenig	Helt uenig 🗆
Når noe s	kal gjøres h	er, er det lett å	få folk med	
Helt 🗂 enig	Delvis enig	Usikker	Delvis 🖂 uenig	Heit uenig 🗆
Det er va		kontekt med fo	olk her Bi	
Helt 🗆 enig	Delvis enig	Usikker 🗌	Delvis 🗆 uenig	Helt uenig 🗀
	dt samhold i	ner 92		
Helt enig	Delvis 🗆 enig	Usikker 🖂	Delvis 🗆 uenig	Helt uenig
		tiv til noe leng	erher es	
Helt 🗆 enig	Delvis 🗆 enig	Usikker 🗌	Delvis uenig	Helt uenig
Folk trive	s godt her s	4		
Helt 🗆 enig	Deivis 🖂 enig	Usikker 🖂	Delvis 🗆 uenig	Helt 🖂
Folk her i		problemer ute		
Helt 🗆 enig	Delvis 📺 enig	Usikker 🗌	Delvis 🗆 uenig	Heit 🗆 uenig
		n tar initiativ ti	l å løse nødve	endige
oppgave	nher ⊪ Detvis ┌┐	Usikker 📇		Helt —
Helt	enig		uenig	
11.11	ker lite med	I hverandre he	T 97 Debde —	Holt _
Helt 🗆 1 enig	Delvis 🗆 2 enig	Usikker 📋 3	Delvis 🛛 1 uenig	Helt □ ⁵ uenig □

SYKDOM I FAMILIEN

Kryss av for de slektningene som har eller har hatt noen av sykdommene. Kryss av for "ingen" hvis ingen av slektningene har hatt denne sykdommen: Evt. flere kryss på hver linje Mor Far Bror Søster Bam Ingen					
Hjerneslag eller hjerneblødning 88					
Hjerteinfarkt før 60 års alder 104 Astma 110					
Allergi 116 Kreftsykdom 122	g				
Høyt blodtrykk 128 Psykiske plager 134 Osteoporose					
(benskjørhet) 140 Diabetes					
(sukkersyke) 146 Alder da de fikk					
diabetes Ja Nei Har du selv høysnue eller neseallergi?					

BRUK AV HELSETJENESTER
Har du l løpet av de siste 12 månedene vært hos; Ett kryss på hver linje Ja Nel allmennpraktiserende lege (kommunelege, privatpraktiserende lege, tumuskandidat) Image: Communelege, bedriftslege Image: Communelege, bedriftslege Image: Communelege, lege ved sykehus (uten at du var innlagt) Image: Communelege, lege ved sykehus (uten at du var innlagt) Image: Communelege, interpreter Image: Communelege, fysloterapeut. Image: Communelege, kiropraktor Image: Communelege, homøopat. Image: Communelege, annen behandler (naturmedisiner, fotsoneterapeut, Image: Communelege, håndspålegger, "healer", "synsk", e.l.) Image: Communelege, Ja Nel Image: Communelege, Har du vært innlagt i sykehus de siste 5 åra? Image: Communelege,
ALKOHOL
Hvis du er totalavholdskvinne: Gå til KOSTHOLD. Ett kryss for hver spørsmål Har du noen gang følt at du burde Ja Nel redusere alkoholforbruket ditt?
Har andre noen gang kritisert Ja Nei aikoholbruken din?
Har du noen gang følt ubehag eller Ja Nei skyldfølelse pga. alkoholbruken din?
Har det å ta en drink noen gang vært det første du har gjort om morgenen for å roe nervene, <i>Ja Nei</i> kurere bakrus eller som en oppkvikker?
KOSTHOLD
Hvor mange måltider spiser du vanligvis Amsil daglig (middag og brødmåltid)?
Hvor mange dager i uka spiser du varm middag?
Hva slags type brød (kjøpt eller hjemmebakt) spiser du vaniigvis? Inntil to kryss Fint Kneipp- Grov- Knekke- Brødtypen ligher Loff brød brød mest på 176 Image: Spiser state Image: Spiser state
Hva slags fett blir vanligvis brukt i din husholdning? Ett kryss for matlaging og ett kryss for brød Bruker ikke smør eller margarin 11 Meierismør 2 Hard margarin 3 Bløt (soft) margarin blanding 5 Lettmargarin 6 Oljer 7
MEDISINBRUK
Har du i deler av de siste 12 måneder brukt Ja Nei noen medisiner daglig eller nesten daglig?
Hvis «Ja»: Angi hvor mange måneder du brukte følgende medisiner: Sett 0 hvis du ikke har brukt medisinene Antaŭ modr.
smertestillende 186 hjertemedisin (ikke
sovemedisin 189 blodtrykksmedisin)
beroligende medisin annen medisin medisin mot depresjon Kosttilskudd:
allergimedisin 194 jemtabletter 202
astmamedisin
Hvor ofte har du brukt avslappende/beroligende medisin eller sovemedisin den siste måneden? 200 Daglig 1 Sjeldnere enn hver uke 3 Hver uke, men ikke hver dag . 2 Aldri 4

tina di seconda d

HODERING Har du vært plaget av hodepine Antali anfall	Ja Nei
I løpet av de siste 12 måneder? 209 siste 12 mndr. 210	Har du smerter i belna når du er I ro?
Ja, anfailsvis (migrene) 1 Ja, annen slags hodepine 2	Er smertene verst når du ligger i senga?
Nej	Blir søvnen forstyrret av smertene?
Hvis «Nei»: Gå til MUSKEL-/SKJELETTPLAGER	Får du mindre vondt når belnet ligger høyt?
Omtrent hvor mange dager i pr. måned har du hodepine? Mindre enn 7 dager 📑 7 til 14 dager 🗌 2 Mer enn 14 d. 🗔 3	Får du mindre vondt når belnet ligger lavt,
Hvor lenge varer hodepinen vanligvis hver gang? 213	f.eks. om beinet henger utfor sengekanten?
Mindre enn 4 timer 🗋 4 timer-3 døgn 🗋 ² Mer enn 3 døgn 🗍 ³	Bedres smertene når du står opp og går litt?271 🗆 🗍
Hvor ofte er hodepinen preget av eller ledsaget av:	MENSTRUASJON
Ett kryss på hver linje Sjelden Av og til Ofte eller aldri	
bankende/dunkende smerte214	Ja Nei Har du menstruasjon fremdeles? 272 🔲 🗌
pressende smerte halvsidighet, alltid samme side	
halvsidighet, vekselvis h. og v. side 🔲 🔲	
smerter i «hele hodet»	Hvis «Nei»: Hvor gammel var du da den sluttet? 273 السيسية
tys- og/eller tydskyhet	Ja Nei Vet ikke
forverring ved fysisk aktivitet	Er du gravid nå? 275
Hvor mange tabletter/stikkpiller har du eventuelt brukt av	Ja Nei
disse medisinene alt i alt i løpet av den siste måneden?	Har du innsatt spiral nå?
Skriv 0 hvis du ikke har brukt medisinen. Cafergot Anervan Imigran	Dag Måned År
223 225 227 227	Når hadde du siste menstruasjon? 277
MUSKEL-/SKJELETTPLAGER	Husker du ikke dag, bare angi måned og år, husker du bare år, angi år.
Har du hatt plager (smorter, verk, ubehag) i muskier og/eller iedd i <i>den siste måneden?</i> 201 Ja Nei	
	Menstruasjonen din de siste 12 måneder:
Hvis «Ja»: Hvor har du hatt disse plagene (ett eller flere kryss) og omtrent hvor mange dager tilsammen var du	Menor dagoner an do dide 12 manodel.
plager (Sett kryss)	Har du det siste året hatt regelmessige menstruasjoner? Ja Nei Usikker
Nakke	At menstruasjonen har vart omtrent like lenge hver gang Ja Nei USINKØF med omtrent like lange mellomrom
Skuldre/aksier233	
Øvre del av ryggen	Hvor mange dager hadde du blødning siste Antali dager
Korsryggen242	gang du hadde menstruasjon? 284
Handledd/hender245	Antall dager
Hofter	Hvor mange dager var du uten blødning mellom nest siste og siste menstruasjon? 28
Ankier/figitier	
Dersom flere kryss: Sett ring rundt	Har menstruasjonen din det siste året uteblitt Ja Nei
krysset der plagen var verst	i mer enn 3 måneder uten at du var gravid? 200
Har plagene hindret deg i å utføre daglige aktiviteter den	Antall mode
siste måneden? Ja Nei Larbeidet	Hvis «Ja»: Hvor mange måneder i trekk har du vært uten menstruasjonsbiødninger?
I fritida	Ja Nei
SMERTER I BEINA	Hvis «Ja»: Oppsøkte du lege? 282 🗆 🗆
Har du sâr pâ tâ, fot eller ankel ja Nei	
som ikke vil gro?	
Har du smerter i det ene eller i begge beina når du går?	Menstruasjonen tidligere (dvs. før de siste 12 månedene):
Har du oppsøkt lege p.g.a. smerter i beina?261	
Hvis «NEI» på disse spørsmålene: Gå til MENSTRUASJON	Har menstruasjonen din tidligere uteblitt Ja Nei uten at du var gravid?
Jan Nei	
Kan du gå lenger enn 50 meter?	Hvis «Ja»: Hvor lenge og hvor ofte var den borte sammen-
Må du sette deg for at smerten skal gå over? 264 🗔 🗔	hengende? Sett kryss eventuelt flere steder 1 gang 2 ganger Oftere
Hvor gjør det mest vondt? Ett kryss 205	3-6 måneder
Fot 🗌 Legg 🛄 Lår 🔲 Hofte 🗔	6-12 måneder
	Over ett år 296

de Not Ver Her du noen gang blitt operet i	OPERASJONER I UNDERLIVET	GRAVIDITETER, FØDSLER OG AWIWING
Hvis -Ja:: Kryas av for hver operasjon: Ja Net 'da' Fjernet bagge eggstokkene in vor Fjernet bagge eggstokkene, hvor gammel var du da? Image barn in de forste 7: gespioninger om fiedentik og arming minister du anning barn in de forste 7: gespioninger om fiedentik og arming minister du anning barn in de forste 7: gespioninger om fiedentik og arming minister du anning barn in de forste 7: gespioninger om fiedentik og arming minister du anning barn in de forste 7: gespioninger om fiedentik og arming minister du anning minister du anning minister arming minister du anning minister du anning minister du anning minister arming minister du anning minister du anning minister arming minister du anning minister du anning minister du anning minister arming minister du anning minister du annin	Har du noen gang blitt operert i ikke	Regn med alle svangerskap, spontane eller selv-
Hvis -Jack Kryss av for hver operaelon: Jack Nev Ver Fjernet bage agslokkne (bles vers of eggslokkne, hvor		Hvor mange harn bar du født?
Hvis du har fjernet bege eggetokkene, hvor gammel var du da?	/kke Fjernet deler av eller bare én eggstokk238 🔲 🔲 🗌	Fyll ut for hvert barn (de første 7) opplysninger om fødselsår og omtrent antall måneder du ammet hvert barn og antall måneder menstruasjonen din var borte etter fødselen (fylles ut også for
Jan Nev Ver 1 30 1 <	Hvis du har fjernet begge eggstokkene, hvor	måneder med blødningsfrie
Har du noen gang brukt p-piller, ja Mei Har du noen gang brukt p-piller, ja Mei Hvis «Ja»: Hvor gammel var du første gang so so Hvis «Ja»: Hvor gammel var du første gang so so Hvis «Ja»: Hvor gammel var du første gang so so Hvor lenge har du brukt p-piller i alt? ja mei Hvis under ett år, antall måneder so ga Nei Bruker du p-piller nå? ga Nei Hvikk merke bruker du? sa ga Nei Honom p-piller Har du lakkagie? so ga Hvike merke bruker du? sa ga Mei hosting, nysing, lattør, tunge loft ga Kilogest, Overstin, Progrovne, Trisekversa, Ma Før Ald Hor Hvis du bruker søtrogenmedisin, og omtent hvor mange å brukte du bit gravid? so da Mei Hvis «Jae: Hvor gammel var du første gang du fikk ga Mei Hvor ange gass meik (alle sorter, også drikkeyoghurt) Hvis «Jae: Hvor gammel var du første gang du fikk so mei du plage svært stort problem Hvis «Jae: Hvor gammel var du første gang du fikk ga Mei <th>Ja Nei Vet Ikke Operert for endometriose</th> <th>2 342 19</th>	Ja Nei Vet Ikke Operert for endometriose	2 342 19
Har du noen gang brukt p-piller, Ja. Nel Hvis «Ja»: Hvor gammel var du første gang * Hvis «Ja»: Hvor gammel var du første gang * Hvor lenge har du brukt p-piller i alt? * Hvis under ett år, antall måneder ** * Hvike under ett år, antall måneder ** * Hvike under ett år, antall måneder ** * Hvike trenke bruker du? ** * Hviket merke bruker du? ** * Hviket merke bruker du? ** * Hord noppiller * Hvor lenge har du brukt p-piller i alt? * Hviket merke bruker du? ** * Hviket merke bruker du? ** * Hord noppiller * Har du lekkasje av urin i forbindelse med * Hord noppiller * Har du lekkasje av urin i forbindelse med * Har du lekkasje av urin i forbindelse med * Har du lekkasje av urin i forbindelse med * Har du lekkasje av urin i forbindelse med * Har du lekkasje av urin i forbindelse med * Har du lekkasje av urin i forbindelse med * Har du søkt lege på grunn av urinlekkasje? * Har du søkt lege på grunn av urinlekkasje? * Hvor lenge har du hatt urinlekkasje? * Har du søkt lege på grunn av urinlekkasje? * Har du søkt lege på grunn av urinlekkasje? * Har du søkt lege på grunn av urinlekkasje? * Har du noen gang prøvd i mer enn ett år * his du noen gang oppsek	P-PILLER	URINLEKKASJE
du brukte p-piller? so Hvor lenge har du brukt p-piller i alt? so Hvis under ett år, antall måneder so ga Nei Hvis under ett år, antall måneder so ga Nei Bruker du p-piller nå? ga Nei Hviket merke bruker du? so ga Nei Hviket merke bruker du? so ga Nei Hotsmann p-piller ga Nei Har du noen gang brukt medisiner som inneholder østro- gen? Vanlige navn på sike medisiner som inneholder østro- gen? Vanlige navn på	Har du noen gang brukt p-piller, Ja Nei minipiller inkludert?	Har du ufrivillig uriniekkasje?
Hvis under ett år, antall måneder avs	du brukte p-pilier?	Hvor ofte har du urinlekkasje? 378 sjeldnere enn en gang pr. måned I en eller flere ganger pr. måned I en eller flere ganger pr. uke I
Bruker du p-piller nå? image: starre mengder Hviiket merke bruker du? 310 image: starre mengder Hviiket merke bruker du? 310 image: starre mengder Har du lekkasje av urin i forbindelse med hosting, nysing, latter, tunge left image: starre mengder Har du neen gang brukt medisiner som inneholder østro- gen? Vanlige navn på sike medisiner er: Cyclabil, Estraderm, Kilogest, Ovesterin, Progynova, Trisekvens. Ma Tabletter eller plaster image: starre mengder Ma Hvis «Ja»: Hvor gammel var du første gang du fikk østrogenmedisin, og omtrent hvor mange år brukte du silk medisin? ma Hvis «Ja»: Hvor gammel var du første gang du fikk østrogenmedisin nå, hvilket merke bruker du? 328 ma Hvis «Ja»: Hvor gammel var du første gang du hadde problemer med å bli gravid? ja Har du noen gang prøvd i mer enn ett år å bli gravid? ja Har du noen gang oppsøkt lege fordi du hadde problemer med å bli gravid? ja Har du noen gang oppsøkt lege fordi du hadde problemer med à bli gravid? ja Har du noen gang oppsøkt lege fordi du hadde problemer med à bli gravid? ja Har du noen gang oppsøkt lege fordi du hadde problemer med à bli gravid? ja Har du noen gang oppsøkt lege fordi du hadde problemer med à bli gravid? ja Har du noen gang oppsøkt lege fordi du hadde problemer med à bli g	Hvor lenge har du brukt p-piller i alt?	
Hosting, nysing, latter, tunge løft	Ja Nei Bruker du p-piller nå?	dråper eller lite 🗆 små skvetter 🗆 større mengder 🗆
Ulenom p-piller Har du noen gang brukt medisiner som inneholder østro- gen? Vanlige navn på slike medisiner er: Cyclabil, Estraderm, Kliogest, Ovesterin, Progynova, Trisekvens. Ma Før Aldri Tabletter eller plaster		
gen? Vanlige navn på slike medisiner er: Cyclabil, Estraderm, Kilogest, Ovesterin, Progynova, Trisekvens. Nå Tabletter eller plaster Krem eller stikkpiller Hvis «Ja»: Hvor gammel var du første gang du fikk østrogenmedisin, og omtrent hvor mange år brukte du Silk medisin? Din Antali alder Are bilteter eller plaster Tabletter eller plaster Bilteter eller bilteter eller plaster Bilteter eller bilteter eller bilteter eller bilteter eller bilteter eller bilteter Bilteter eller bilteteter Bilteter eller b	Utenom p-piller	plutselig og sterk vannlatingstrang? 382 🛛 🖓
Tabletter eller plaster Krem eller stikkpiller Hvis «Ja»: Hvor gammel var du første gang du fikk østrogenmedisin, og omtrent hvor mange år brukte du sik medisin? Din Antali alder dater	gen? Vanlige navn på slike medisiner er: Cyclabil, Estraderm, Kilogest, Ovesterin, Progynova, Trisekvens.	0-5 år 🗋 5-10 år 🗌 Over 10 år 🗌 Ja <u>Ne</u> i
Hvis «Ja»: Hvor gammel var du første gang du fikk østrogenmedisin, og omtrent hvor mange år brukte du silk medisin? Tabletter eller plaster Tabletter eller plaster Krem eller stikkpiller Mindre østrogenmedisin nå, hvilket merke bruker du? 328 PROEILEIMER MED A BLI CRAVID Har du noen gang prøvd i mer enn ett år Åbi gravid? Hvis «Ja»: Hvor gammel var du første gang du hadde problemer med å bli gravid? Arei Har du noen gang oppsøkt lege fordi du hadde Ja Nei Har du noen gang oppsøkt lege fordi du hadde Ja Nei Kalktabletter eller benmel Witamin D-tilskudd Witamin D-tilskudd Witamin D-tilskudd	Tabletter eller plaster	Hvordan opplever du lekkasjeplagene dine?
Krem eller stikkpiller	østrogenmedisin, og omtrent hvor mange år brukte du slik medisin? alder år	en liten plage 🔲 svært stort problem 🗍
merke bruker du? 328 drikker du vanligvis daglig? Bare ett kryss 386 PROBLEMER MED A BLI GRAVID 1-2 glass	•	KALK I KOSTEN OG KOSTTILSKUDD
PROBLEMER MED A BLI GRAVID Har du noen gang prøvd i mer enn ett år Ja Nei à bli gravid? 329 Hvis «Ja»: Hvor gammel var du første gang 1 du hadde problemer med à bli gravid? 330 Har du noen gang oppsøkt lege fordi du hadde Ja Nei problemer med à bli gravid? 332 Har du noen gang oppsøkt lege fordi du hadde Ja Nei Ja Nei Ja Nei Har du noen gang oppsøkt lege fordi du hadde Ja Nei Ja Nei Ja Nei Bruker du vanligvis noen av disse kosttilskuddene? Vitamin D-tilskudd 332		drikker du vanligvis daglig? Bare ett kryss 386
Har du noen gang prøvd i mer enn ett år Ja Nei å bli gravid? 329 1 Hvis «Ja»: Hvor gammel var du første gang 1 du hadde problemer med å bli gravid? 330 Har du noen gang oppsøkt lege fordi du hadde Ja problemer med å bli gravid? 332 Mar du noen gang oppsøkt lege fordi du hadde Ja Nei 332	PROBLEMER MED A BLIGRAVID	Mindre enn ett니 º 3 eller mer 니 4
Hvis «Ja»: Hvor gammel var du første gang du hadde problemer med å bli gravid? and forste gang br Har du noen gang oppsøkt lege fordi du hadde problemer med å bli gravid? Ja Nei Ja kalktabletter eller benmel Ja	Har du noen gang prøvd i mer enn ett år Ja Nei å bli gravid?	daglig? Bare ett kryss
Har du noen gang oppsøkt lege fordi du hadde Ja Nei problemer med å bli gravid? 332 Image: Strategy oppsøkt lege fordi du hadde Ja Nei kalktabletter eller benmel Image: Strategy oppsøkt lege fordi du hadde		Bruker du vanligvis noen av disse kosttilskuddene?
	Har du noen gang oppsøkt lege fordi du hadde Ja Nei problemer med å bli gravid?	vitamin D-tilskudd 388 🔲 🛄

n an an an Argenting California an an an Argenting An an an Argenting an Argenting California an Argenting California an Argenting California and Argenting California

HUMØR OG TRIVSEL	HVORDAN DU HAR HALL DEL
Ett kryss på hver linje Angi hvordan du har følt deg den siste måneden: i godt humør	Har det noen gang i løpet av ditt liv vært sammen- hengende perioder på 2 uker eller mer da du: Ja Nel følte deg deprimert, trist og nedfor
Er du rask til å oppfatte treg treg rask rask et humoristisk poeng? 392	hadde problemer med å konsentrere deg eller vanskelig for å ta beslutninger
	hadde minst tre av de problemene som er nevnt
Er du enig i at det er noe ansvarsløst over folk som	ovenfor samtidig
stadig prøver å være morsomme? 393 Nei, slett ikke	
Nei, slett ikke \Box^1 Ganske enig \Box^3 I noen grad \Box^2 Ja, absolutt \Box^4	HVORDAN DU SER PÅ DEG SELV
	Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn
Er du en munter person?394	hvor enig eller uenig du er. Ett kryss på hver linje
Nei, slett ikke	Svært Svært enig Enig Uenig uenig
	Jeg har en positiv holdning til meg selv
SINNE	Jeg føler meg virkelig ubrukelig til tider
Sett kryss på det svaret som best beskriver deg i forhold til de	
to påstandene nedenfor:	Jeg føler at jeg ikke har mye å være stolt av
Jeg gir uttrykk for mitt sinne, og andre mennesker vet at	Jeg føler at jeg er en verdifuli
Jeg er sint ₂ss Nesten aldri	person, i atlefali på lik linje
Nesten aldri	med andre
	Synes du at du har funnet et virkelig Ja Nei betydningsfullt innhold i livet ditt?
Jeg koker av sinne, men jeg viser det ikke til andre 386 Nesten aldri	
Noen ganger	Føler du at du iever fullt ut?
	HVORDAN DU FØLER DEG NA
HVILE OG AVSLAPPING	
	Sett kryss i den ruta utenfor det svaret som best beskriver dine følelser den siste uka. Bare ett kryss
Hvor mange timer tilbringer du vanligvis i liggende stilling i løpet av et døgn?	Er du vanligvis glad eller nedstemt?
(nattesøvn, middagshvil)	Svært nedstemt
Hvor mange timer tilbringer du vanligvis i	Nedstemt
sittende stilling i løpet av et døgn?	Både og
(arbeid, måltider, TV, bil etc.)	Nokså glad
Hvor ofte er du plaget av søvnløshet? 🗤	Glad
Aldri, eller noen få ganger i året	
1–2 gender i måneden ²	Har du i det store og hele en rolig og god følelse
Omfrent 1 gang i uka	Inne i deg? 419 Nesten hele tide
Mer enn en gang i uka	
Har du siste år vært plaget av søvnløshet Ja Nei	Av og til
slik at det har gått ut over arbeidsevnen?	Aldri
Har du i løpet av siste måned hatt innsovnings-	Føler du deg stort sett sterk og opplagt, eller trøtt og
problemer? Bare ett kryss 403	silten? 420
Nesten hver natt	Meget sterk og opplagt
	Ganske sterk og opplagt
Har du I løpet av siste måned våknet for tidlig og ikke	Både – og
fått sove igjen? Bare ett kryss, 404	Ganske trøtt og sliten
Nesten hver natt	Svært trøtt og sliten
Har du liøpet av siste måned vært plaget av	Legg det utfylte spørreskjemaet i den ved-
nervøsitet (irritabel, urolig, anspent eller rastiøs)? 405 Nesten hele tida	lagte svarkonvolutten og postlegg den så
Ofte	snart som mulig!
Av og til	Porto er betalt.
Aldri	Hjertelig takk for hjelpa!
	Steinvier Trykkeri AS – 74 16 30 00

ŗ.

- 22

hunt Helseundersøkelsen i Nord-Trøndelag	20–69 ÅR
kk for frammøtet til undersøkelsen! /il også be deg fylle ut dette spørreskjemaet. Opplysningene vil bli brukt i stø gende hetsearbeid. Noen av spørsmålene likner på spørsmål du har sva me og leverte ved frammøte til helseundersøkelsen. Det er likevel viktig at så i dette skjemaet. Det utfylte skjemaet returneres i vedlagte svarkonvolutt. I e opplysningene er underlagt streng taushetspilkt.	nt på i det skjemaet du fylte ut du svarer på alle spørsmålene
e opplysningene er undenagt streng tausnetsplakt. Vennlig hilsen	Hvis du ikke ønsker å besvare spørre-
Helsetjenesten i Nord-Trøndelag Statens Institutt for Golkehelse Statens helseun	skjemaet, sett kryss her og returner skjemaet. Da slipper du purring.
•	
UTFYLLING	BOLIG
Dato for utfylling av skjema: / 19 19	Hvem bor du sammen med? Ett kryss for hver linje og angi antall Ja Nei Ektefelle/samboer 54
OPPVEKST	Andre personer over 18 år 55
I hvilken kommune bodde du da du fylte 1 år? Hvis du ikke bodde i Norge, oppgi land i stedet for kommune.	Personer under 18 år 59 🗆 🗖
	Hvor mange av barna har plass i barnehage?
24	Hvilken type bolig bor du i? Bare ett kryss
ARBEID	Enebolig/villas3
Nåværende eller tidligere arbeid: Hva slags inntektsgivende arbeid har du og event. din	Gårdsbruk Blokk/terrasseleilighet
ektefelle/samboer? Hvis du/dere ikke har inntektsgivende arbeid	Rekkehus/2-4 mannsbolig Annen holin
nå: Oppgi det siste yrket. Deg Ektefelle/ selv samboer	
Spesialarbeider eller ufaglært arbeider ₂s □ □ ∞ Fagarbeider, handverker, formann □ □ □	Hvor stor er din boenhet?
Underordnet funksjonær (f.eks. butikk,	Er det heldekkende tepper i stua?
kontor, off. tjenester)	Er det heldekkende tepper på ditt soverom?
Fagfunksjonær (f.eks. sykepleier, tekniker,	Er det katt i boligen?
Overordnet stilling i off. eller privat virksomhet	Er det andre pelskiedde dyr eller fugler i boligen?
Sjåfør	······································
Gårdbruker eller skogeier	
Fisker	ØKONOMI
tannlege, advokat)	Mottar du noen av følgende offentlige ytelser? Ja
Annen selvstendig næringsvirksomhet	Sykepenger/sykelønn/rehabiliteringspenger
Har ikke vært i inntektsgivende arbeid 35 📋 🔲 46	Uførepensjon
Hvis du NÅ ikke har inntektsgivende arbeid eller du ikke	Alderspensjon
har heltids husarbeid: Gå til BOLIG.	Sosialstøtte
Har du I løpet av de siste 12 månedene	Arbeidsløshetstrygd
hatt sykefravær: Ja Nei	Etterlattepensjon
med egenmelding 47 🛄	Andre ytelser
med sykmelding fra lege 48 🔲 🗍	
Hvis «Ja»: Hvor lenge tilsammen? Bare ett kryss	Har det i løpet av det siste året hendt at husholdningen har hatt vansker med å klare de løpende utgifter til mat,
2 uker eller mindre 49 🔲 1 2-8 uker	transport, bolig og liknende? Bare ett kryss si
2-8 uker 2 Mer enn 8 uker	Ja, ofte In Ja, en sjelden gang
	Ja, av og til
Har du i løpet av de siste 12 månedene Ja Nei vurdert å skifte yrke eller arbeidsplass? ∞ □ □	
	VENNER
	Hvor mange gode venner har du?
Er arbeidet ditt så fysisk anstrengende at du ofte er sliten I kroppen etter en arbeidsdag? Bare ett kryss 51 Ja, nesten alltid	Regn med de du kan snakke fortrolig med og som kan gi deg god hjelp når du trenger det
Krever arbeidet ditt så mye konsentrasjon og oppmerk-	Ja
somhet at du ofte føler deg utsiltt etter en arbeidsdag? ⁵² Ja, nesten alltid	Føler du at du har mange nok gode venner? 😝 🗌
Ganske ofte 🗍 2 Aldri, eller nesten aldri 🏾 4	Hvor ofte tar du vanligvis del i foreningsvirksomhet son
Hvordan trives du ait i alt med arbeidet ditt? 🖘	f.eks. syklubb, idrettslag, politiske lag, religiøse eller andre foreninger? 65
Veldig godt I 1 Ikke særlig godt 3	Aldri, eller noen få ganger i året 1 Omtrent en gang i uka
Godt C 2 Dârlig	1-2 ganger i måneden

72

DER DU BOR Svar ut fra nærmiljøet, dvs. nabolaget/grenda. Ett kryss for hvert spørsmål Jeg føler et sterkt fellesskap med de som bor her 🕫 uenig 6 Helt 1 Delvis 2 Usikker 3 Delvis 4 enia enig Selv om noen tar initiativ, er det ingen som blir med på det som settes i gang her av Delvis 🖂 Helt Helt uenig Delvis Usikker enig enia uenig Hvis jeg flytter herfra, vil jeg lengte tilbake 🕫 Delvis Helt uenig Helt Delvis Usikker uenia enia enig Man kan ikke stole på hverandre her 89 Delvis uenig 🗆 Delvis Usikker enig eniq uenia Når noe skal gjøres her, er det lett å få folk med Delvis 📋 Helt enig uenig 🗆 Delvis Usikker enig uenia Det er vanskelig å få kontakt med folk her 91 Helt uenig 🗆 Delvis uenig Helt 🗆 enig Delvis Usikker enia Det er godt samhold her 22 Delvis Heit uenig Helt anig Delvis Usikker enig uenig Ingen orker å ta initiativ til noe lenger her 🕫 Delvis Delvis uenig uenig Usikker Helt 🖂 enig Folk trives godt her 94 Delvis 🖂 uenig Delvis Usikker 🗌 Helt C enig uenia Folk her kan ha store problemer uten at naboen vet noe 95 Delvis Helt uenig Heit Delvis Usikker uenia enia enia Det er alltid noen som tar initiativ til å løse nødvendige oppgaver her 96 Delvis Helt uenig Heit Usikker Delvis uenig enia enia Folk snakker lite med hverandre her 97 uenig 🗆 🕫 enia enia uenia

SYKDOM | FAMILIEN

Kryss av for de slektningene som har eller har hatt noen av sykdommene. Kryss av for «ingen» hvis ingen av slektningene har hatt denne sykdommen. Evt. flere kryss på hver linje Mor Far Bror Søster Ban Ingen

		•				
Hjerneslag eller hjerneblødning	98					
Hjerteinfarkt før 60 års alder Astma Allergi Kreftsykdom Høyt blodtrykk						
Psykiske plager			Ō		Ē	
Osteoporose (benskjørhet) Diabetes	140					
(sukkersyke)	146					
Alder da de fikk diabetes	152 6/	Âr	Ar	<u>s</u> ,	ór	
Ja Nei Har du selv høysnue eller neseallergi? 152 🗌 🗌						

BRUK AV HELSETJENESTER Har du i løpet av de siste 12 månedene vært hos : Ja Nei Ett kryss på hver linje allmennpraktiserende lege (kommunelege, privatpraktiserende lege, turnuskandidat) 163 bedriftslege..... lege ved sykehus (uten at du var innlagt) annen lege fysioterapeut..... kiropraktor homeopat...... annen behandler (naturmedisiner, fotsoneterapeut, håndspålegger, "healer", "synsk", e.i.) Nei Ja Har du vært innlagt i sykehus de siste 5 åra?...... 171 ALKOHOL Hvis du er totalavholdsmann: Gå til KOSTHOLD. Ett kryss for hver spørsmål Har du noen gang følt at du burde Ja Nei Har andre noen gang kritisert Nel alkoholbruken din? Har du noen gang følt ubehag eller Nei Har det å ta en drink noen gang vært det første du har gjort om morgenen for å roe nervene, Ja Nei kurere bakrus eller som en oppkvikker? 175 🗔 🗍 KOSTHOLD Hvor mange måltider spiser du vanligvis Anial daglig (middag og brødmåltid)?......176 Hvor mange dager i uka spiser du varm middag? Hva slags type brød (kjøpt eller hjemmebakt) spiser du vanligvis? Inntil to kryss. Grov- Knekke-Fint Kneipp-Brødtypen ligner Loff brød brød brød brød mest på 178 🗔 Π m Hva slags fett blir vanligvis brukt i din husholdning? Ett kryss for matlaging og ett kryss for brød Til matlaging På brød Bruker ikke smør eller margarin 183 1 184 1 Meierismør..... 2 [] 3 Hard margarin..... []4 Bløt (soft) margarin 5 Smør/margarin blanding 6 Lettmargarin 7 Oljer MEDISINBRUK Ja Nei Har du i deler av de siste 12 måneder brukt noen medisiner daglig eller nesten daglig? 185 🔲 🗍 Hvis «la»' Angi hvor mange måneder du brukte følgende medisiner: Sett 0 hvis du ikke har brukt medisinene Antal modr. ntali mnd smertestillende 186 hjertemedisin (ikke blodtrykksmedisin) sovemedisin..... 188 annen medisin beroligende medisin Kosttilskudd: medisin mot depresion allergimedisin..... 194 jerntabletter 2 aetmamodiein vitamintilskudd

	tran/fiskeoljer 200
ivor ofte har du brukt avslappe nedisin eller sovemedisin den	siste måneden? 208
aglig	1 Sjeldnere enn hver uke 🛄 3
lver uke men ikke hver dag.	2 Aldri

n D

HODEPINE	
Har du vært plaget av hodepine Antall anfall	Ja Nei
I løpet av de siste 12 måneder? 209 siste 12 mndr. 210	Har du smerter i beina når du er I ro?265
Ja, anfallsvis (migrene)	Er smertene verst når du ligger i senga?
Nei	Blir søvnen forstyrret av smertene?
Hvis «Nei»: Gå til MUSKEL-/SKJELETTPLAGER	Får du mindre vondt når beinet ligger høyt? 269 🔲 🗔
Omtrent hvor mange dager i pr. måned har du hodepine? Mindre enn 7 dager 1 7 til 14 dager 2 Mer enn 14 d. 3	Får du mindre vondt når belnet ligger lavt, f.eks. om beinet henger utfor sengekanten?270
Hvor lenge varer hodepinen vanligvis hver gang? 213	Bedres smertene når du står opp og går litt? 271 🔲 🗔
Mindre enn 4 timer 🗌 14 timer –3 døgn 🗋 2 Mer enn 3 døgn 🗔 3	URINVEGS- OG PROSTATAPLAGER
Hvor ofte er hodepinen preget av eller ledsaget av:	
Ett kryss på hver linje Sjelden Av og til Ofte eller aldri	Ett kryss på hver linje Har du noen gang blitt fortalt av lege at du har: Ja Nei
bankende/dunkende smerte	forstørret prostata
pressende smerte	prostatakreft
hatvsidighet, alttid samme side 🔲 🔲 🗌	
haivsidighet, vekselvis h. og v. side 🔲 📙 📙	Har du gjennomgått noe av følgende: Ja Nei
smerter i «hele hodet»	sterilisering
kvalme	kirurgisk tjerning av prostata (helt eller delvis)276
forverring ved fysisk aktivitet	anal gior i porting av prostata (non oner desvio)
synsforstyrrelser før hodepine	De neste spørsmålene gjelder siste måned
Hvor mange tabletter/stikkplier har du eventuelt brukt av	Bare ett kryss for hvert hver spørsmål
disse medisinene alt i alt i løpet av den siste måneden?	
Skriv 0 hvis du ikke har brukt medisinen.	Hvor ofte har du hatt følelsen av at blæren ikke er blitt
Calergot Anervan imigran	fulistendig tømt etter avsluttet vannlating? 277 Aldri
	Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger
MUSKEL-/SKJELETTPLAGER	Omtrent 1 av 3 ganger I ³ Nesten alltid
Har du hatt plager (smerter, verk, ubehag) i	
muskler og/eller ledd i den siste måneden? 229 Ja Nei	Hvor ofte har du måttet late vannet på nytt mindre
	enn 2 timer etter forrige vannlating? 276
Hvis «Ja»: Hvor har du hatt disse plagene (ett eller flere kryss) og omtrent hvor mange dager tilsammen var du	Aldri
Planale Planale	Omtrent 1 av 5 ganger□² Omtrent 2 av 3 ganger□⁵ Omtrent 1 av 3 ganger□³ Nesten alltid
Plager (Sett kryss)	Unauesc (av 3 danger Li ^v Neslen alla)
Nakke	
Nakke	Hvor ofte har du måttet stoppe og starte flere ganger under vannlatingingen? 279
Nakke	Hvor ofte har du måttet stoppe og starte flere ganger under vannlatingingen? 279 Aldri
Nakke	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri
Nakke	Hvor ofte har du måttet stoppe og starte flere ganger under vannlatingingen? 279 Aldri
Nakke	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri
Nakke 230 Skuldre/aksler233 34 Øvre del av ryggen 239 Albuer 239 Korsryggen 242 Handledd/hender 245 48	Hvor ofte har du måttet stoppe og starte flere ganger under vannlatingingen? 279 Aldri
Nakke 230 Skuldre/aksler23 230 Øvre del av ryggen 239 Albuer 239 Korsryggen 242 Handledd/hender 245 48 Knær	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 1 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 0 3 Nesten alltid 0 3 Nesten alltid 1 av 3 ganger 5 0 3 Nesten alltid 1 av 3 ganger 5 1 av 3 ganger 5 1 av 3 ganger 5 Hvor ofte syns du det har vært vanskelig å holde igjen når 6 Hvor ofte syns du det har vært vanskelig å holde igjen når 6 Aldri 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1
Nakke 230 Skuldre/aksler233 Øvre del av ryggen Albuer 239 Korsryggen 242 Handledd/hender 245 Hofter	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 280 Aldri 1 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 1 0 1 1 Hvor ofte syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 280 1 1 1 Midri 1 1 2 1 1 1 4 Omtrent 1 av 5 ganger 2 2 0 3 3 3 3
Nakke 230 Skuldre/akslør	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 1 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 0 3 Nesten alltid 0 3 Nesten alltid 1 av 3 ganger 5 0 3 Nesten alltid 1 av 3 ganger 5 1 av 3 ganger 5 1 av 3 ganger 5 Hvor ofte syns du det har vært vanskelig å holde igjen når 6 Hvor ofte syns du det har vært vanskelig å holde igjen når 6 Aldri 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1
Nakke 220 Skuldre/aksler 230 Øvre del av ryggen 239 Aibuer 239 Korsryggen 242 Handledd/hender 245 Hofter Nakke figer 251 Ankler/føtter 251 Dersom flere kryss: Sett ring rundt krysset der plagen var verst	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 280 Aldri 1 0 mtrent annenhver gang 4 Omtrent 1 av 5 ganger 1 0 mtrent annenhver gang 4 Omtrent 1 av 5 ganger 1 0 mtrent annenhver gang 4 Omtrent 1 av 5 ganger 1 0 mtrent annenhver gang 4 Omtrent 1 av 3 ganger 2 0 mtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6
Nakke 230 Skuldre/aksler 230 Øvre del av ryggen 239 Aibuer 239 Korsryggen 242 Handledd/hender 245 Hofter Ankler/føtter 251 Ørse der plagen var verst Dersom flere kryss: Sett ring rundt krysset der plagen var verst Har plagene hindret deg i å utføre daglige aktiviteter den	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 280 Aldri 1 Omtrent annenhver gang 5 Omtrent 1 av 5 ganger 2 Omtrent annenhver gang 5 Omtrent 1 av 5 ganger 3 Nesten alltid 5 Hvor ofte har du hatt svak urinstråle? 20 20 20
Nakke 230 Skuldre/aksler233 Skuldre/aksler233 Øvre del av ryggen 239 Albuer 239 Korsryggen 242 Handledd/hender 245 Hofter	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 280 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent annenhver gang 4 Omtrent 1 av 3 ganger 1 Omtrent annenhver gang 6 Hvor ofte har du hatt svak urinstråle? 280 6 6 Hvor ofte har du hatt svak urinstråle? 280 7 7
Nakke 230 Skuldre/aksler233 Skuldre/aksler233 Øvre del av ryggen 239 Albuer 239 Korsryggen 242 Handledd/hender 245 Hofter	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 280 Aldri 1 Omtrent annenhver gang 5 Omtrent 1 av 5 ganger 2 Omtrent annenhver gang 5 Omtrent 1 av 5 ganger 1 Omtrent annenhver gang 5 Omtrent 1 av 5 ganger 2 Nesten alltid 5 Omtrent 1 av 3 ganger 3 Nesten alltid 5 Hvor ofte har du hatt svak urinstråle? 281
Nakke 230 Skuldre/aksler233 Skuldre/aksler233 Øvre del av ryggen 239 Albuer 239 Korsryggen 242 Handledd/hender 245 Hofter	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 1 Omtrent 1 av 5 ganger 2 2 Omtrent 1 av 3 ganger 3 Nesten alltid Omtrent 1 av 3 ganger 3 Nesten alltid Image: Straight of the syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 280 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 3 Nesten alltid 5 Omtrent 1 av 5 ganger 3 Nesten alltid 5 Omtrent 1 av 3 ganger 3 Nesten alltid 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte har du hatt svak urinstråle? 281 4 6 Hvor ofte har du hatt svak urinstråle? 281 4 6 Hvor ofte har du hatt svak urinstråle? 281 4 6 Omtrent 1 av 5 ganger 2 0 6 6 Hvor ofte har du hatt svak urinstråle? 281 5 6 6
Nakke 230 Skuldre/aksler233 Øvre del av ryggen Albuer 239 Korsryggen 242 Handledd/hender 245 Hofter Ankler/føtter 244 Korsom flere kryss: Sett ring rundt krysset der plagen var verst Har plagene hindret deg i å utføre daglige aktiviteter den siste måneden? Ja Nei I arbeidet 257 I I fritida 258 I	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 1 Omtrent 1 av 5 ganger 2 2 Omtrent 1 av 3 ganger 3 Nesten alltid Omtrent 1 av 3 ganger 3 Nesten alltid Image: Strate fiere ganger 3 Nesten alltid Omtrent 1 av 3 ganger 3 Nesten alltid Image: Strate fiere ganger 1 Nesten alltid Image: Strate fiere ganger 1 Nesten alltid Image: Strate fiere ganger 1 Omtrent annenhver gang Image: Strate fiere ganger 1 1
Nakke 220 Skuldre/aksler 230 Øvre del av ryggen 239 Albuer 239 Korsryggen 242 Handledd/hender 245 44 Hofter 240 Ankler/føtter 253 Ørsom flere kryss: Sett ring rundt krysset der plagen var verst Har plagene hindret deg i å utføre dagilge aktiviteter den siste måneden? Ja Nei I arbeidet 257 1 I fittida 259 1	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 1 Omtrent 1 av 5 ganger 2 2 Omtrent 1 av 3 ganger 3 Nesten alltid Omtrent 1 av 3 ganger 3 Nesten alltid Image: Straight of the syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 280 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 3 Nesten alltid 5 Omtrent 1 av 5 ganger 3 Nesten alltid 5 Omtrent 1 av 3 ganger 3 Nesten alltid 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte har du hatt svak urinstråle? 281 4 6 Hvor ofte har du hatt svak urinstråle? 281 4 6 Hvor ofte har du hatt svak urinstråle? 281 4 6 Omtrent 1 av 5 ganger 2 0 6 6 Hvor ofte har du hatt svak urinstråle? 281 5 6 6
Nakke 230 Skuldre/aksler233 Øvre del av ryggen Albuer 239 Korsryggen 242 Handledd/hender 245 Hofter Nakke 242 Handledd/hender 245 Hofter Ankler/føtter 251 Dersom flere kryss: Sett ring rundt krysset der plagen var verst Har plagene hindret deg i å utføre daglige aktiviteter den siste måneden? Ja Nei I arbeidet 257 I I fritida 258 I	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 280 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte har du hatt svak urinstråle? 281 4 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 3 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte har du måttet trykke eller presse for å begynne vannlatingen? 8 Hvor ofte har du måttet trykke eller presse for å begynne vannlatingen? 6 Hvor ofte har du måttet trykke aller presse for å b
Nakke 220 Skuldre/aksler 230 Øvre del av ryggen 230 Albuer 230 Korsryggen 242 Handledd/hender 245 44 Hofter 240 Hofter 240 Handledd/hender 245 44 Hofter 254 Dersom flere kryss: Sett ring rundt Krysset der plagen var verst Har plagene hindret deg i å utføre daglige aktiviteter den siste måneden? Ja Nei I arbeidet 257 1 I fritida 258 1 SMERTIER LEIEINA Ja Nei 259 Har du sår på tå, fot eller ankel Ja Nei Har du snerter i det ene eller i begge 1	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 1 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 280 Aldri 1 Omtrent annenhver gang 5 Omtrent 1 av 5 ganger 2 Omtrent annenhver gang 5 Omtrent 1 av 5 ganger 1 Omtrent annenhver gang 5 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte har du hatt svak urinstråle? 281 4 6 Hvor ofte har du nåttet trykke eller presse for å begynne vannlatingen? 6 6 Hvor ofte har du måttet trykke eller presse for å begynne vannlatingen? 6
Nakke 220 Skuldre/aksler 230 Øvre del av ryggen 230 Albuer 230 Albuer 230 Handledd/hender 245 40 Hofter 242 Handledd/hender 245 40 Hofter 250 Ankler/føtter 251 Ørsom flere kryss: Sett ring rundt krysset der plagen var verst Har plagene hindret deg i å utføre daglige aktiviteter den siste måneden? Ja Nei I arbeidet 257 I fittida 258 SMHELTER I BEINA Ja Nei Har du sår på tå, fot eller ankel Ja Nei som ikke vil gro? 250 Har du sår på tå, fot eller ankel 250	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 280 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte har du hatt svak urinstråle? 281 4 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 3 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte har du måttet trykke eller presse for å begynne vannlatingen? 2 1 0mtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 1 0 1 6 <td< th=""></td<>
Nakke 220 Skuldre/aksler 230 Øvre del av ryggen 230 Albuer 230 Korsryggen 242 Handledd/hender 245 44 Hofter 240 Hofter 240 Handledd/hender 245 44 Hofter 254 Dersom flere kryss: Sett ring rundt Krysset der plagen var verst Har plagene hindret deg i å utføre daglige aktiviteter den siste måneden? Ja Nei I arbeidet 257 1 I fritida 258 1 SMERTIER LEIEINA Ja Nei 259 Har du sår på tå, fot eller ankel Ja Nei Har du snerter i det ene eller i begge 1	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri
Nakke 220 Skuldre/aksler 230 Øvre del av ryggen 230 Albuer 230 Albuer 230 Handledd/hender 245 40 Hofter 242 Handledd/hender 245 40 Hofter 250 Ankler/føtter 251 Ørsom flere kryss: Sett ring rundt krysset der plagen var verst Har plagene hindret deg i å utføre daglige aktiviteter den siste måneden? Ja Nei I arbeidet 257 I fittida 258 SMHELTER I BEINA Ja Nei Har du sår på tå, fot eller ankel Ja Nei som ikke vil gro? 250 Har du sår på tå, fot eller ankel 250	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 1 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 280 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte har du hatt svak urinstråle? 281 6 Hvor ofte har du måttet trykke eller presse for å begynne vanlatingen? 2 0mtrent 2 av 3 ganger 6 Hvor ofte har du måttet trykke eller presse for å begynne vanlatingen? 4 0 6 Hvor ofte har du måttet trykke eller presse for å begynne vanlatingen? 5 6 Hvor ofte har du måttet trykke aller presse for å begynne vanlatingen? 5 6 Hvor menge ganger mar du vanligvis måttet stå opp 5 6 </th
Nakke 220 Skuldre/aksler233 Skuldre/aksler233 Øvre del av ryggen 239 Albuer 239 Korsryggen 242 Handledd/hender 245 Hofter Hofter 248 Krær 254 Ankler/føtter 254 Ankler/føtter 254 Ankler/føtter 254 Ørsom flere kryss: Sett ring rundt Krysset der plagen var verst Har plagene hindret deg i å utføre daglige aktiviteter den siste måneden? Ja Nei I arbeidet 257 I I fritida 258 I SMIELINER I BEINA Ja Nei 258 Har du sår på tå, fot eller ankel Ja Nei 258 Som ikke vil gro? 259 I Har du sår på tå, fot eller ankel Ja Nei 259 Har du sår på tå, fot eller ankel Ja Nei 250 Har du sår på tå, fot eller ankel 250 I Har du oppsøkt lege p.g.a. smerter I beina? 250 I Har du oppsøkt lege p.g.a. smerter I beina? 251 I Hvis «NEI» på disse spørsmå	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 278 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nøsten alltid 6 Hvor ofte syns du det har vært vanskelig å holde ligjen når du har følt trang til å late vannet? 280 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nøsten alltid 6 Hvor ofte har du hatt svak urinstråle? 281 4 6 Hvor ofte har du måttet trykke eller presse for å begynne vanlatingen? 6 6 Hvor ofte har du måttet trykke eller presse for å begynne vanlatingen? 6 6 Hvor ofte har du måttet trykke eller presse for å begynne vanlatingen? 6 6 Hvor ofte har du måttet trykke aller presse for å begynne vanlatingen? 6 6 Hvor ofte har du måttet trykke aller presse for å begynne vanlatingen? <
Nakke 220 Skuldre/aksler233 Skuldre/aksler233 Øvre del av ryggen 239 Aibuer 239 Korsryggen 242 Handledd/hender 245 Hofter Hofter 251 Ankler/føtter 253 Ørse del av ryggen 244 Handledd/hender 245 Hofter Har plagene hindret deg i å utføre dagilge aktiviteter den siste måneden? Ja Nei I arbeidet 257 I fritida 258 SMERUER EEINA Ja Nei Har du sår på tå, fot eller ankel Ja Nei som ikke vil gro? 252 Har du smerter i det ene eller i begge 250 belan når du går? 250 Har du oppsøkt lege p.g.a. smerter i beina? 251 Hvis «NEI» på disse spørsmålene: Gå til UFINVEGS Ja Nei	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 1 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nøsten alltid 6 Hvor ofte syns du det har vært vanskelig å holde ligjen når du har følt trang til å late vannet? 280 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 3 Nøsten alltid 6 Hvor ofte har du hatt svak urinstråle? 281 4 Aldri 1 Omtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nøsten alltid 6 Hvor ofte har du måttet trykke eller presse for å begynne vanlatingen? 2 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 5 <td< th=""></td<>
Nakke 220 Skuldre/aksler 230 Øvre del av ryggen 230 Albuer 230 Korsryggen 242 Handledd/hender 245 44 Hofter 248 Krær 254 Ankler/føtter 254 Ørsom flere kryss: Sett ring rundt krysset der plagen var verst Har plagene hindret deg i å utføre daglige aktiviteter den siste måneden? Ja Nei I arbeidet 257 I fritida 258 SMIELTER EIEINA Ja Nei Har du sår på tå, fot eller ankel Ja Nei som ikke vil gro? 259 Har du sår på tå, fot eller ankel Ja Nei som ikke vil gro? 250 Har du sår på tå, fot eller ankel 250 Har du oppsøkt lege p.g.a. smerter I beina? 250 Har du oppsøkt lege p.g.a. smerter I beina? 250 Hvis «NEI» på disse spørsmålene: Gå til URINVEGS 250	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 280 4 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte har du hatt svak urinstråle? 281 4 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte har du hatt svak urinstråle? 281 4 6 Hvor ofte har du måttet trykke eller presse for å begynne vanlatingen? 6 6 Hvor ofte har du måttet trykke eller presse for å begynne vanlatingen? 6 Hvor ofte har du måttet trykke eller presse for å begynne vanlatingen? 6 Hvor mange ganger har du vanligvis måttet
Nakke 220 Skuldre/aksler 230 Øvre del av ryggen 230 Albuer 230 Korsryggen 242 Handledd/hender 245 Hofter Hofter 240 Handledd/hender 245 Hofter Hofter 240 Har plagene hindret deg i å utføre daglige aktiviteter den siste måneden? Ja Nei I arbeidet 257 I frida 258 SMIECUERT ESEINA Ja Nei Har du sår på tå, fot eller ankel Ja Nei som ikke vil gro? 252 Har du smerter i det ene eller i begge 1 beina når du går? 250 Har du oppsøkt lege p.g.a. smerter i beina? 261 Hvis «NEI» på disse spørsmålene: Gå til URINVEGS. Ja Nei Kan du gå lenger enn 50 meter? 252 1	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 0mtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte syns du det har vært vanskelig å holde løjen når du har følt trang til å late vannet? 280 Aldri 1 0mtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte har du hatt svak urinstråle? 281 4 Aldri 1 0mtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 1 0mtrent 2 av 3 ganger 6 Hvor ofte har du måttet trykke eller presse for å begynne vanlatingen? 6 Hvor ofte har du måttet trykke eller presse for å begynne vanlatingen? 6 Hvor mange ganger har du vanligvis måttet stå op
Nakke 220 Skuldre/aksler233 Øvre del av ryggen Albuer 239 Korsryggen 242 Handledd/hender 245 Hofter 249 Hofter 249 Handledd/hender 245 Hofter 249 Handledd/hender 245 Hofter 249 Hardledd/hender 245 Har plagene hindret deg i å utføre daglige aktiviteter den siste måneden? Ja Nei I arbeidet 257 I fritida 257 I fritida 259 SMUERTER ESEINA Ja Nei Har du sår på tå, fot eller ankel Ja Nei I arbeidet 257 I fritida 259 SMERTER ESEINA Ja Nei Har du sår på tå, fot eller ankel Ja Nei som ikke vil gro? 259 Har du sår på tå, fot eller ankel Ja Nei som ikke vil gro? 250 Har du sørerter i det ene eller i begge 1 beina når du går? 250 Har du oppsøkt lege p.g.a. smerter i beina? 251 Hvis «	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri
Nakke 220 Skuldre/aksler233 Skuldre/aksler233 Øvre del av ryggen 239 Albuer 239 Korsryggen 242 Handledd/hender 245 Hofter Hofter 249 Handledd/hender 245 Hofter Nakker/føtter 251 Ankler/føtter 251 Ørsom flere kryss: Sett ring rundt Krysset der plagen var verst Har plagene hindret deg i å utføre daglige aktiviteter den siste måneden? Ja Nei I arbeidet 257 Imagen I frida 257 Imagen Kan du sår på tå, fot eller ankel Ja Nei som ikke vil gro? 250 Imagen Har du sår på tå, fot eller ankel Ja Nei som ikke vil gro? 250 Imagen Har du oppsøkt lege p.g.a. smerter I beina? 250 Imagen I hvis «NEI» på disse spørsmålene: Gå til URINVEGS. Ja Nei I som kar på disse spørsmålene: Gå til URINVEGS. Ja Nei Må du sette deg for at smerten skal gå over? Imagen Imagen Må du sette deg for at smerten skal gå over? 261 Imagen	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 0mtrent 1 av 5 ganger 2 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte syns du det har vært vanskelig å holde løjen når du har følt trang til å late vannet? 280 Aldri 1 0mtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 6 Hvor ofte har du hatt svak urinstråle? 281 4 6 Hvor ofte har du måttet trykke eller presse for å begynne vannlatingen? 5 6 Mdri 1 0 6 6 Hvor ofte har du måttet trykke eller presse for å begynne vannlatingen? 6 6 Hvor ofte har du måttet trykke aller presse for å begynne vannlatingen? 6 6
Nakke 220 Skuldre/aksler233 Skuldre/aksler233 Øvre del av ryggen 239 Albuer 239 Handledd/hender 245 Hofter Hofter 249 Hofter 249 Handledd/hender 245 Hofter Hofter 249 Har plagene hindret deg i å utføre daglige aktiviteter den siste måneden? Ja Nei I arbeidet 257 I fritida 257 SMIETTER ESEINA Ja Nei Har du sår på tå, fot eller ankel Ja Nei I arbeidet 257 I fritida 259 Øren ikke vil gro? 259 Har du sår på tå, fot eller ankel Ja Nei som ikke vil gro? 259 Har du smerter i det ene eller i begge 1 beina når du går? 250 Har du oppsøkt lege p.g.a. smerter i beina? 261 Hvis «NEI» på disse spørsmålene: Gå til UFINVEGS Ja Nei Kan du gå lenger enn 50 meter? 262 Ør 1 1 Må du sette deg for at smerten skal gå over? 24	Hvor ofte har du måttet stoppe og starte fiere ganger under vannlatingingen? 279 Aldri 1 0mtrent 1 av 5 ganger 2 Omtrent 1 av 5 ganger 2 0mtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte syns du det har vært vanskelig å holde løjen når du har følt trang til å late vannet? 280 Aldri 1 Omtrent annenhver gang 4 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 5 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte har du hatt svak urinstråle? 281 4 Aldri 1 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 2 Omtrent 2 av 3 ganger 5 Omtrent 1 av 3 ganger 3 Nesten alltid 6 Hvor ofte har du måttet trykke eller presse for å begynne vannlatingen? 282 Aldri 1 3 Sanger 5 Omtrent 1 av 3 ganger 1 0

an an tha an

HUMØR OG TRIVSEL	HVORDAN DU HAR HALT DET
Ett kryss på hver linje Angi hvordan du har følt Noen Ganske For det deg den siste måneden: Aldri ganger ofte meste i godt humør	Har det noen gang i løpet av ditt liv vært sammen- hengende perioder på 2 uker eller mer da du: Ja Nei følte deg deprimert, trist og nedfor
Svært Ganske Ganske Svært Er du rask til å oppfatte treg treg rask rask et humoristisk poeng? 201	virkelig bebreidet deg selv og følte deg verdiløs hadde problemer med å konsentrere deg eller vanskelig for å ta beslutninger
Er du enig i at det er noe ansvarsløst over folk som stadig prøver å være morsomme? 2000	ovenfor samtidig
Nei, slett ikke	HVORDAN DU SER PÅ DEG SELV
	Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn
Er du en munter person?₂₅₅ Nei, slett ikke□¹ Ganske munter□³ I noen grad□² Ja, absoiutt□4	hvor enig eller uenig du er. Ett kryss på hver linje Svært Svært enig Enig Uenig uenig
	Jeg har en positiv holdning til meg selv
SINNE	Jeg føler meg virkelig ubrukelig til tider
Sett kryss på det svaret som best beskriver deg i forhold til de to påstandene nedenfor:	Jeg føler at jeg ikke har mye å være stolt av
Jeg gir uttrykk for mitt sinne, og andre mennesker vet at jeg er sint. 290 Nesten aldri	Jeg føler at jeg er en verdifull person, i allefall på lik linje med andre
Noen ganger \Box^2 Nesten alltid \Box^4	Synes du at du har funnet et virkelig Ja Nei
Jeg koker av sinne, men jeg viser det ikke til andre. 291 Nesten aldri□1 Ganske ofte□3 Noen ganger□2 Nesten alltid□4	betydningsfullt innhold i livet ditt?
	HVORDAN DU FØLER DEG NA
HVILE OG AVSLAPPING	Sett kryss i den ruta utenfor det svaret som best beskriver
Hvor mange timer tilbringer du vanifgvis i liggende stilling i løpet av et døgn? (nattesøvn, middagshvil)	dine følelser den siste uka. Bare ett kryss Er du vanligvis glad eller nedstemt? 313 Svært nedstemt
	Nedstemt
Hvor mange timer tilbringer du vanilgvis i Anstitiener sittende stilling i løpet av et døgn?	Både og
(arbeid, måltider, TV, bil etc.)	
Hvor ofte er du plaget av søvnløshet? 296	Glad
Aldri, eller noen få ganger i året	
1-2 ganger i måneden	Har du i det store og hele en rolig og god følelse inne i deg? 314
Omtrént 1 gang i uka	Nesten hele tida
	Ofte
Har du siste år vært plaget av søvnløshet Ja Nei slik at det har gått ut over arbeidsevnen?	AV og til
Har du l løpet av siste måned hatt Innsovnings-	Føler du deg stort sett sterk og opplagt, eller trøtt og sliten? 315
Problemer? Bare ett kryss 238 Nesten hver natt	Meget sterk og opplagt
Ofte	Sterk og opplagt
Har du i løpet av siste måned våknet for tidlig og ikke	Både – og
fått sove igjen? Bare ett kryss 299	Gapske trøtt og sliten
Nesten hver natt	Trøtt og sliten
Ofte	Legg det utfylte spørreskjemaet i den ved- lagte svarkonvolutten og postlegg den så snart som mulig! Porto er betalt. Hjertelig takk for hjelpa!
	Steinker 74 18 30 00

2

hunt

Helseundersøkelsen i Nord-Trøndelag

SKJEMA FOR KVINNER 70 ÅR OG ELDRE

Takk for frammøtet til undersøkelsen! Vi vil også be deg fylle ut dette spørreskjemaet. Opplysningene vil bli brukt i større forskningsarbeider om fore-byggende helsearbeid. Noen av spørsmålene likner på spørsmål du har svart på i det skjemaet du fylte ut heime og leverte ved frammøte til helseundersøkelsen. Det er likevel viktig at du svarer på alle spørsmålene også i dette skjemaet. Det utfylte skjemaet returneres i vedlagte svarkonvolutt. Porto er betalt. Alle opplysningene er underlagt streng taushetspilkt.

oppiysingene er underlagt snorg tausierspinn. Vennlig kilsen Helsotionesten i Nord-Trandelag.	Hvis du ikke ansker å besvare spørre- skjemaet. Sett kryss her og returner skjemaet. Da slipper du purring
Helsetjenesten i Nord-Trøndelag Statens Institutt for Øolkehelse – Statens helseund	lersøkelser Jegønsker ikke å besvare skjemaet O
UTFYLLING	SIVILSTAND
Dato for utfylling av skjema: / 19 19	Hva er din sivilstand? 105 Gift
OPPVEKST	Skill/separert 2 Har aldri vært gift 4
I hvilken kommune bodde du da du fylte 1 år? Hvis du ikke bodde i Norge, oppgi land i stedet for kommune	BRUK AV HELSETJENESTER
24	BRUN AV HELSETJENESTEN
BOLIG Hvilken type bolig bor du i? Bare ett kryss Enebolig/villa	Har du i løpet av de siste 12 månedene vært hos: Ett kryss på hver linje Ja Nei allmennpraktiserende lege (kommunelege,
Gårdsbruk	privatpraktiserende lege, turnuskandidat)
Trygdebolig/aldersbolig/servicebolig 5 Sykeheim/aldersheim 6 Annen bolig	annen lege
Hvor stor er din boenhet? %	annen behandier (naturmedisiner, fotsoneterapeut, håndspålegger, "healer", "synsk", e.l.)
Er det heldekkende tepper i stua?	SYKEHUS Ja Nei
Er det andre pelskiedde dyr eller fugler i boligen? 🗌 🗌	Har du vært innlagt i sykehus de siste 5 åra? 113 🔲 🗌
Hvem bor du sammen med? Ett eller flere kryss Ektefelle/samboer	Hvis «Ja»: Svar ut fra siste gang du var innlagt Synes du at du ble utskrevet for tidlig, i passe tid eller for seint? 114 For tidlig I passe tid For seint
SYKDOM I FAMILIEN	Hvor ble du utskrevet til? 115 Heim Kuropphold
Kryss av for de slektningene som har eller har hatt noen av sykdommene. Kryss av for "ingen" hvis ingen av slektningene har hatt denne sykdommen. <i>Evt. flere kryss på hver linje</i>	Sykeheim
Mor Far Bror Søster Barn Ingen Hjerneslag eller hjerneblødning	Fikk du tilstrekkelig hjelp og oppfølging Ja Nei etter utskrivingen?
60 års alder 46	HEIMEHJELP
Kreftsykdom 64 1 1 1 Høyt blodtrykk 70 1 1 1 Psykiske plager 76 1 1 1	Har du heimehjelp? Ja Nei Privat
Osteoporose (benskjørhet)	Dersom du har KOMMUNAL heimehjelp: Har du nok kommunal heimehjelp, eller trenger du mer? 119 Ja, jeg har nok
Aider da de fikk diabetes	Nei, jeg trenger mer I I tilfelle du IKKE har kommunal heimehjelp: Ja
Har du <i>seiv</i> høysnue eller neseallergi? 104 🗌 🗌	Trenger du kommunal heimehjelp? 120
Same and a start of the second sec	

	and the second secon
HEIMESYKEPLEIE Ja Nei	Hvor mange glass melk (alle sorter, også drikkeyoghurt)
Har du heimesykepleie? 121	drikker du vanligvis daglig? Bare ett kryss 136
Hvis «Ja»:	Ingen □ 1 1-2 glass □ 3 Mindre enn ett □ 2 3 eller mer □ 4
Har du nok heimesykepiele, eller trenger du mer?	
Ja, jeg har nok	Hvor mange brødskiver med kvitost spiser du vanligvis
Nei, jeg trenger mer	daglig? Bare ett kryss 137 Ingen □ 1–2 skiver
SYKEHEIM	
Har du vært innlagt på sykeheim i løpet av de siste	HVILE OG AVSLAPPING
12 månedene? 123	Hvor mange timer tilbringer du vanligvis i
Ja, jeg har vært der en periode	liggende stilling i løpet av et døgn?
Ja, jag bor der fast	(nattesøvn, middagshvil)
Hvis «Nei», kan du hoppe over de neste to spørsmålene	Hvor mange timer tilbringer du vanligvis i
	sittende stilling i løpet av et døgn?
Hvis «Ja»: Hvor var du FØR du ble innlagt på sykeheimen siste	(arbeid, måltider, TV, bil etc.)
gang? 124	Har du i løpet av siste måned hatt innsovnings-
Bodde i egen heim	problemer? Bare ett kryss 142
Var innlagt i sykehus	Nesten hver natt 1 Av og til 3 Ofte 2 Aldri 4
Hvis du har vært på sykeheimen EN PERIODE i løpet av de siste 12 mndr.:	Har du i løpet av siste måned våknet for tidlig og ikke fått sove igjen? Bare ett kryss 143
Bodde du på sykeheimen passe lenge? 125	Nesten hver natt
Det var for kort tid	Ofte
Passe tid	
	MEDISINBRUK
KOMMUNAL HJELP ALT I ALT	Har du i deler av de siste 12 måneder brukt Ja Nai noen medisiner daglig eller nesten daglig? 14
Hvordan er du alt i alt fornøyd med hjelpa du får fra	
kommunen? 126 Meget fornøvd 1 jeg får ingen hjelp,	Hvis "Ja":
Meget fornøyd 📑 jeg får ingen hjelp, Nokså fornøyd 🗋 2 men burde ha hatt det 🗋 5	Angi hvor mange måneder du brukte følgende medisiner: Sett 0 hvis du ikke har brukt medisinene
Nokså misfornøyd 🛛 🔹 🛛 Jeg får ingen hjelp, 🔄 🔄	Antal mndr
Meget misfornøyd og trenger det ikke	smertestillende
	sovemedisin
	beroligende medisin annen medisin
KOSTHOLD	medisin mot depresion Kosttiiskudd:
Anta3	allergimedisin
Hvor mange måltider spiser du vanligvis	andre vitamintilskudd
daglig (mlddag og brødmåltid)? ¹²⁷	tran/fiskeoljer167
Hvor mange dager i uka spiser du varm middag?	
the stand have build (high all a blan mabala)	Hvor ofte har du brukt avslappende/beroligende medisin eller sovemedisin den siste måneden? 169
Hva slags type brød (kjøpt eller hjemmebakt) spiser du vanligvis? Inntil to kryss	Dealig 🔅 🖓 Sieldhere enn hver uke 🖓
Fint Knelpp- Grov- Knekke-	Hver uke, men ikke hver dag 🗋 2 Aldri
Brødtypen ligner Loff brød brød brød brød mest på	
Hva slags fett blir vanligvis brukt i din husholdning? Ett kryss for matlaging og ett kryss for brød Til matlaging På brød	VENNER
Ett kryss for matlaging og ett kryss for brød Til matlaging På brød Bruker ikke smør eller margarin 134 [] ¹ 135 [] ¹	Hvor mange gode venner har du?
Meierismør	Regn med de du kan snakke fortrolig med og som kan gi deg god hjelp når du trenger det 170
Hard margarin	Contract & and Social and an inclusion and and
	Tell ikke med de du bor sammen med, men regn med andre
Bløt (soft) margarin	Tell ikke med de du bor sammen med, men regn med andre slektninger
Biøt (soft) margarin 4 4 Smøt/margarin blanding 5 5 Lettmargarin 6 6	slektninger Ja Nei
Biøt (soft) margarin	sløktninger

Hvor ofte tar du vanligvis del i foreningsvirksomhet som f.eks. syklubb, eldresenter, pensjonistforening, politiske lag, religiøse eller andre foreninger? Bare ett kryss 173 Aldri, eller noen få ganger i året	Hvor ofte er hodepinen preget av eller ledsaget av: Ett kryss på hver linje Sjelden Av og til Ofte eller aldri bankende/dunkende smerte
HUMØR OG TRIVSEL Ett kryss på hver linje Angi hvordan du har følt Noen Ganske For det deg den siste måneden: Aldri ganger ofte meste i godt humør 174 I I I I i dårlig humør 175 I I I I	halvsidighet, alllid samme side
Svært Ganske Ganske Svært Er du rask til å oppfatte treg treg rask rask et humoristisk poeng? 176	Cafergot Anervan Imigran Imigr
Er du enig i at det er noe ansvarsløst over folk som stadig prøver å være morsomme? 177 Nei, stett ikke	Har du lekkasje av urin (uansett mengde) minst Ja Nei to ganger per måned? Hvis «Nei»: Gå til MENSTRUASJON OG OVERGANG Hvor ofte har du urinlekkasje? noen få ganger per måned en eller flere ganger per uke hver dag og/eller natt
MUSKEL-/SKJELETTPLAGER Her du hatt plager (smerter, verk, ubehag) I Ja Nei	Hvor mye urin lekker du vanligvis hver gang? 200 dråper eller lite små skvetter eller mer Har du lekkasje av urin i forbindelse med
muskier og/eiler ledd i <i>den siste måneden?</i> 179 🗆 🗖 Hvis «Nei»: Gå til HODEPINE Hvis «Ja»: Hvor har du hatt disse plagene (ett eller flere	hosting, nysing eller latter
kryss) og omtrent hvor mange dager tilsammen var du plaget? Nakke	Hvordan opplever du lekkasjeplagene dine? Bare ett kryss ikke noe problem en liten plage mye plaget svært stort problem
Albuer	Ja Nei Har du søkt lege pga. urinlekkasje? 235 🔲 🗌
	Hvor gammel var du da menstruasjonen sluttet?
Har plagene hindret deg i å utføre daglige Ja Nei aktiviteter den siste måneden? 207	HORMONBIEHANDLING Utenom p-piller Har du noen gang brukt medisiner som inneholder østro- gen? Vanlige navn på slike medisiner er: Cyclabil, Estraderm, Kilogest, Ovesterin, Progynova, Trisekvens.
HODEPINE Antall antall Har du vært plaget av hodepine Antall antall i løpet av de siste 12 måneder? 2008 Siste 12 mndr. 2009	Nå Før Aldri Tabletter eller plaster 238 1 1 Krem eller stikkpiller 239 1 1
Ja, anfalisvis (migrene) 1 Ja, annen slags hodepine 2 Nei	Hvis «Ja»: Hvor gammel var du første gang du fikk østrogenmedisin, og omtrent hvor mange år brukte du slik medisin? Din Antall alder år
Omtrent hvor mange dager pr. måned har du hodepine? Mindre enn 7 dager []1 7 til 14 dager []2 Mer enn 14 d. []3	Tabletter eller plaster 240 Krem eller stikkpiller 244
Hvor lenge varer hodepinen vanligvis hver gang? 212 Mindre enn 4 timer [] 14 timer-3 døgn [] 2 Mer enn 3 døgn [] 3	Hvls du bruker østrogenmedisin nå, hvilket merke bruker du? 249

OPERASJONER I UNDERLIVET	
Ja Nei Vet Har du fått fjernet begge ikke eggstokkene (totait)?	Er du vanligvis glad eller nedstemt? Svært nedstemt 1 Nokså glad 5 Nedstemt 2 Glad 6 Nokså nedstemt 3 Svært glad 7
Hvis du har fjernet begge eggstokkene, hvor gammel var du da? 250 47	Både – og
Ja Nei Vet	LEGEMLIGE FUNKSJONER
Ikke Har du fått fjernet hele livmoren? 252	Klarer du selv, uten hjelp av andre, i det daglige å: Ett kryss på hver linje Med noe
Hvis du har fjernet hele livmoren, hvor gammel var du da? 253 41	Ja hjelp Nei Gå innendørs i samme etasje 303 333 333 Gå på toalettet 333 333 333
GRAVIDITETER, FØDSLER OG AMMING	Vaske deg på kroppen
Hvor mange ganger har du vært gravid totalt? Regn med alle svangerskap, spontane eller selv- bestemte aborter, så vel som fødsler (også dødlødsler). 255	Kle på og av deg 307 307 Legge deg og stå opp 307 307 Spise selv 309 307
Hvor mange barn har du født? 257 barn	Hvis du har hatt hjelp til noe av dette, omtrent hvor lenge
Fyll ut for hvert barn (de første 6) opplysninger om fødselsår og omtrent antall måneder du ammet hvert barn og antall måneder menstruasjonen din var borte etter fødselen (fylles ut også for dødlødle eller for barn som er døde senere i livet). Barn Fødselsår Antall Antall	har du hatt hjelp? Bare ett kryss 310 Under 3 måneder 1 5 år 3 – 6 måneder 2 Mer enn 5 år 1/2 – 1 år 3
måneder med blødningsfrie amming måneder	Hvis du trenger hjelp til ett eller flere av disse
	gjøremålene, hvem er det som for det meste hjelper deg?
1 ²⁵⁸ 15	Bare ett kryss Ektefelle/samboer
3 270 19	Barn/svigerbarn
4 276 19	Søster/bror Ll 3
5 282 19	DAGLIGE OPPGAVER
6 200 19	
HVORDAN DU SER PÅ DEG SELV	A second s
HVONDAN DO SEN PA DEG SELV	Klarer du selv disse gjøremålene i det daglige uten hjelp fra andre2 Ett knos og bygring
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei
	fra andre? Ett kryss på hver ilnje Med noe Ja hjelp Nei Lage varm mat
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. <i>Ett kryss på hver linje</i> Svært Svært enig Enig Uenig uenig	fra andre? Ett kryss på hver ilnje Med noe Ja hjelp Nei Lage varm mat 312 Image Gjøre lett husarbeid Image Image (f.eks. oppvask) Image Image
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig eller uenig du er. <i>Ett kryss på hver linje</i> Svært Svært enig Enig Uenig uenig Jeg har en positiv holdning	fra andre? Ett kryss på hver ilnje Med noe Ja hjelp Nei Lage varm mat 312 Image:
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig eller uenig du er. <i>Ett kryss på hver linje</i> <i>Svært</i> <i>enig Enig Uenig uenig</i> Jeg har en positiv holdning til meg selv	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 Image: State Sta
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. <i>Ett kryss på hver linje</i> Svært Svært enig Enig Uenig uenig Jeg har en positiv holdning	fra andre? Ett kryss på hver linje Med noe Ja hjelp Nei Lage varm mat 312 Image:
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. Ett kryss på hver linje Svært Svært Svært Svært enig Enig Uenig uenig Jeg har en positiv holdning Image selv Image selv Jeg føler meg virkelig ubrukelig Image selv Image selv	fra andre? Ett kryss på hver linje Med noe Ja hjelp Nei Lage varm mat 312 Image: State in the state in
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig eller uenig du er. <i>Ett kryss på hver linje</i> Svært Svært enig Enig Uenig uenig Jeg har en positiv holdning til meg selv	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 Image: Image
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig eller uenig du er. Ett kryss på hver linje Svært Svært svært Svært enig Enig Jeg har en positiv holdning III til meg selv 24 Jeg føler meg virkelig ubrukelig III til tider 255 Jeg føler at jeg ikke har mye a være stolt av Jeg føler at jeg er en verdifull person, i allefall på lik linje	fra andre? Ett kryss på hver linje Med noe Ja hjelp Nei Lage varm mat 312 Image: State in the state in
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig eller uenig du er. Ett kryss på hver linje Svært Svært svært Svært enig Enig Uenig usnig Jeg har en positiv holdning til meg selv 294 Image:	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 Image: State
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. Ett kryss på hver linje Svært Svært svært Svært enig Enig Uenig uenig Jeg har en positiv holdning til meg selv 294 Image: Strand	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 Image: State
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig eller uenig du er. Ett kryss på hver linje Svært Svært Svært Svært svært svært svært svært enig Enig Uenig uenig Jeg har en positiv holdning til meg selv 234 Image: Ima	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 Image: State
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. Ett kryss på hver linje Svært Svært Svært Svært Svært svært enig Enig Uenig uenig Jeg har en positiv holdning til meg selv 294 Image: Image	fra andre?Ett kryss på hver linjeMed noeJahjeipNeiLage varm mat 312 \Box Gjøre lett husarbeid $(f.eks. oppvask)$ \Box (f.eks. oppvask) \Box \Box Gjøre tyngre husarbeid \Box \Box (f.eks. gutvvask) \Box \Box Vaske klær \Box \Box Betale regninger \Box \Box Ta medisinene \Box \Box Gjøre innkjøp \Box \Box Ta bussen \Box \Box Hvis du trenger hjelp til ett eller flere av disse \Box gjøremålene, omtrent hvor lenge har du hatt hjelp?Bare ett kryssBare ett kryss Ξ \Box Under 3 måneder \Box \Box $1 - 5$ år \Box \Box $1/2 - 1$ år \Box
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. Ett kryss på hver linje Svært Svært Svært Svært svært svært svært svært enig Enig Uenig uenig Jeg har en positiv holdning til meg selv 234 Image: Ima	fra andre?Ett kryss på hver linjeMed noeJahjeipNeiLage varm mat 312 \Box Gjøre lett husarbeid $(f.eks. oppvask)$ \Box (f.eks. oppvask) \Box \Box Gjøre tyngre husarbeid \Box \Box (f.eks. gutvvask) \Box \Box Vaske klær \Box \Box Betale regninger \Box \Box Ta medisinene \Box \Box Gjøre innkjøp \Box \Box Ta bussen \Box \Box Hvis du trenger hjelp til ett eller flere av disse \Box gjøremålene, omtrent hvor lenge har du hatt hjelp? $Bare ett kryss zet$ Under 3 måneder \Box \Box 1 $1 - 5$ år \Box $1/2 - 1$ år \Box Hvis du trenger hjelp til ett eller flere av disseHvis du trenger hjelp til ett eller flere av disseHvis du trenger hjelp til ett eller flere av disse
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig eller uenig du er. Ett kryss på hver linje Svært Svært Svært grade ser en sostiv holdning Enig Uenig uenig Jeg har en positiv holdning til meg selv 234 Image Image Image Jeg føler meg virkelig ubrukelig Image Image Image Image Jeg føler at jeg ikke har mye 3 Image	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 Image: State
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. Ett kryss på hver linje Svært Svært enig Enig Uenig uenig Jeg har en positiv holdning III meg selv IIII meg selv Jeg føler meg virkelig ubrukelig IIII tider IIII tider Jeg føler at jeg ikke har mye IIIII meg selv IIIIII Jeg føler at jeg ikke har mye IIIIIII IIIIIII Jeg føler at jeg er en verdifull IIIIIIII IIIIIIIIII person, i allefall på lik linje IIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIII	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 Image: State
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. Ett kryss på hver linje Svært Svært Svært enig Enig Uenig uenig Jeg har en positiv holdning til meg selv	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 Image: State
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. Ett kryss på hver linje Svært Svært Svært Svært Svært svært enig Enig Uenig uenig Jeg har en positiv holdning 1 1 1 jeg føler meg virkelig ubrukelig 1 1 1 jeg føler at jeg ikke har mye 3 1 1 jeg føler at jeg er en verdifull 1 1 1 jeg føler at jeg er en verdifull 1 1 1 jeg føler at jeg er en verdifull 1 1 1 jeg føler at jeg er en verdifull 1 1 1 person, i allefall på lik linje 2 1 1 med andre 2 2 1 1 Synes du at du har funnet et virkelig Ja Nøi 1 1 betydningsfullt innhold i livet ditt? 2 2 1 Føler du at du lever fullt ut? 2 2 1 HVORDAN DU FØLER DEC NA 1 3 2 1 Hvors i den ruta utenfor det svaret som best beskriver 1 1	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 1 Gjøre lett husarbeid 1 1 (f.eks. oppvask) 1 1 Gjøre tyngre husarbeid 1 1 (f.eks. gutvvask) 314 1 1 Vaske klær 1 1 1 Betale regninger 316 1 1 Ta medisinene 1 1 1 1 Komme deg ut 316 1 1 1 Gjøre innkjøp 1 1 1 5 Ta bussen 2 1 1 5 1 Bare ett kryss 21 1 1 5 1 Ja 6 måneder 2 1 1 5 Ja 6 måneder 1 1 1 5 Bare ett kryss 22 1 1 5 5 Jz 1 1 1 5 3 5
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. Ett kryss på hver linje Svært Svært Svært Svært Svært svært enig Enig Uenig uenig Jeg har en positiv holdning til meg selv 294 Image Image Jeg føler meg virkelig ubrukelig Image Image Image Image Jeg føler at jeg ikke har mye 3være stolt av 295 Image Image Image Jeg føler at jeg er en verdifull person, i allefall på lik linje Image	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 1 Gjøre lett husarbeid 1 1 (f.eks. oppvask) 1 1 Gjøre tyngre husarbeid 1 1 (f.eks. gutvvask) 314 1 1 Vaske klær 1 1 1 Betale regninger 316 1 1 Ta medisinene 1 1 1 1 Komme deg ut 316 1 1 1 Gjøre innkjøp 1 1 1 5 Ta bussen 2 1 1 5 1 Bare ett kryss 21 1 1 5 1 Ja 6 måneder 2 1 1 5 Ja 6 måneder 1 1 1 5 Bare ett kryss 22 1 1 5 5 Jz 1 1 1 5 3 5
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. Ett kryss på hver linje Svært Svært Svært Svært Svært svært enig Enig Uenig uenig Jeg har en positiv holdning til meg selv 24 Image:	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 1 Gjøre lett husarbeid 1 1 (f.eks. oppvask) 314 1 Gjøre tyngre husarbeid 1 1 (f.eks. gutvvask) 314 1 Vaske klær 1 1 Betale regninger 316 1 Ta medisinene 1 1 Komme deg ut 316 1 Gjøre innkjøp 1 1 Ta bussen 320 1 Hvis du trenger hjelp til ett eller flere av disse 1 gjøremålene, omtrent hvor lenge har du hatt hjelp? 1 Bare ett kryss 321 1 1 Under 3 måneder 2 Mer enn 5 år 5 1/2 - 1 år 3 3 1 5 Hvis du trenger hjelp til ett eller flere av disse 1 1 5 Bare ett kryss 322 Ektefelle/samboer 1 1 6 Barn/svigerbarn 2 Andre 5 5 <tr< th=""></tr<>
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. Ett kryss på hver linje Svært Svært Svært Svært Svært svært enig Enig Uenig uenig Jeg har en positiv holdning til meg selv 294 Image Image Jeg føler at positiv holdning Image Image Image Image Jeg føler meg virkelig ubrukelig Image Image Image Image Image Jeg føler at jeg ikke har mye avære stolt av 296 Image	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 Image: State in the state in t
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. Ett kryss på hver linje Svært Svært Svært Svært Svært svært enig Enig Uenig uenig Jeg har en positiv holdning til meg selv 34 Image Image Jeg føler meg virkelig ubrukelig Image Image Image Image Jeg føler at jeg ikke har mye 4 Image Image Image Image Jeg føler at jeg er en verdifull person, i allefall på lik linje Image	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 1 Gjøre lett husarbeid 1 1 (f.eks. oppvask) 314 1 Gjøre tyngre husarbeid 1 1 (f.eks. gutvvask) 314 1 Vaske klær 1 1 Betale regninger 316 1 Ta medisinene 1 1 Komme deg ut 316 1 Gjøre innkjøp 1 1 Ta bussen 320 1 Hvis du trenger hjelp til ett eller flere av disse 1 gjøremålene, omtrent hvor lenge har du hatt hjelp? 1 Bare ett kryss 321 1 1 Under 3 måneder 2 Mer enn 5 år 5 1/2 - 1 år 3 3 1 5 Hvis du trenger hjelp til ett eller flere av disse 1 1 5 Bare ett kryss 322 Ektefelle/samboer 1 1 6 Barn/svigerbarn 2 Andre 5 5 <tr< th=""></tr<>
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig elier uenig du er. Ett kryss på hver linje Svært Svært Svært Svært Svært svært enig Enig Uenig uenig Jeg har en positiv holdning til meg selv 294 Image Image Jeg føler at positiv holdning Image Image Image Image Jeg føler meg virkelig ubrukelig Image Image Image Image Image Jeg føler at jeg ikke har mye avære stolt av 296 Image	fra andre? Ett kryss på hver linje Med noe Ja hjeip Nei Lage varm mat 312 Image: State in the state in t

hunt

SKJEMA FOR MENN 70 ÅR OG ELDRE

Takk for frammøtet til undersøkelsen!

Helseundersøkelsen i Nord-Trøndelag Vi vil også be deg fylle ut dette spørreskjemaet. Opplysningene vil bli brukt i større forskningsarbeider om fore-byggende helsearbeid. Noen av spørrsmålene likner på spørsmål du har svart på i det skjemaet du fylte ut heime og leverte ved frammøte til helseundersøkelsen. Det er likevel viktig at du svarer på alle spørsmålene også i dette skjemaet. Det utfylte skjemaet returneres i vedlagte svarkonvolutt. Porto er betalt. Alle opplysningene er underlagt streng taushetspilkt. Hvis du ikke ønsker å besvare spørre-skjernaet, sett kryss her og returner skjernaet. Da slipper du purring. Vennlig hilsen Helsetjenesten i[']Nord-Trøndelag Statens Institutt for Golkehelse Statens helseundersøkelser Jeg ønsker ikke å besvare skjemaet 🔘 UTFYLLING SIVILSTAND Dato for utfylling av skjema: 19 1 12 OPPVEKST I hvilken kommune bodde du da du fyite 1 år? Hvis du ikke bodde i Norge, oppgi land i stedet for kommune BRUK AV HELSETJENESTER 24 BOLIG Hvilken type bolig bor du i? Bare ett kryss Har du i løpet av de siste 12 månedene vært hos: Enebolig/villa..... 25 🔲 1 Ett kryss på hver linje Ja Nei 2 allmennpraktiserende lege (kommunelege, Gårdsbruk 3 4 privatpraktiserende lege, turnuskandidat) 108 🔲 Blokk/terrasseleilighet..... ΠĒ Rekkehus/2-4 mannsbolig lege ved sykehus (uten at du var innlagt) annen lege Trygdebolig/aldersbolig/servicebolig 5 Sykeheim/aldersheim 6 fysioterapeut..... kiropraktor **□** ⁷ Annen bolig..... homøopat......111 🗍 🗍 annen behandler (naturmedisiner, fotsoneterapeut, Hvor stor er din boenhet?..... 26 kv/a håndspålegger, "healer", "synsk", e.l.) Ja Nei Er det heldekkende tepper I stua? 28 🔲 🔲 Er det heldekkende tepper på ditt soverom?...... SYKEHUS Er det hund i boligen?..... Ja Ne. Har du vært innlagt i sykehus de siste 5 åra? 113 🔲 🗌 Er det andre pelskledde dyr eller fugler i boligen? Hvem bor du sammen med? Ett eller flere kryss Hvis «Ja»: Svar ut fra siste gang du var innlagt Synes du at du ble utskrevet for tidlig, i passe tid eller for seint? 114 Bor alene 36 Andre 38 D For tidlig \square I passe tid For seint Hvor ble du utskrevet til? 115 SYKDOM I FAMILIEN Heim Kryss av for de slektningene som har eller har hatt noen av Kuropphold sykdommene. Kryss av for "ingen" hvis ingen av slektningene Sykeheim har hatt denne sykdommen. Evt. flere kryss på hver linje Far Bror Søster Barn Ingen Fikk du tilstrekkelig hjelp og oppfølging Mor Ja Nei etter utskrivingen? 116 Hjerneslag eller hjerneblødning 40 🗌 Hierteinfarkt før 60 års alder 46 🔲 Astma..... 52 🗋 HEIMEHJELP Allergi 58 🗋 \Box Kreftsykdom..... 64 🔲 \Box Har du heimehjelp? Ja Nei Høyt blodtrykk...... 70 🔲 Privat 117 Psykiske plager...... 76 D Kommunal 118 🔲 🗌 Osteoporose (benskjørhet)..... B2 Dersom du har KOMMUNAL heimehjelp: Har du nok kommunal heimehjelp, eller trenger du mer? 119 Diabetes (sukkersyke) 🕫 🛛 Ja, jeg har nok Alder da de fikk Nei, jeg trenger mer ár år diabetes 94 | Nei I tilfelle du IKKE har kommunal heimehjelp: Ja Har du selv høysnue eller neseallergi?..... 101 Trenger du kommunal heimehjelp? 120 🔲 🗍

n na sense se se version d'avail à la diggi d'and and and an sense a company

Ja Nei Har du heimesykepleie? Hvis «Ja»: Har du nok heimesykepleie, eller trenger du mer? Ja, jeg har nok Nei, jeg trenger mer	Hvor mange glass melk (alle sorter, også drikkeyoghurt) drikker du vanligvis daglig? Bare ett kryss 136 Ingen 1 1-2 glass 3 Mindre enn ett 2 3 eller mer 4 Hvor mange brødskiver med kvitost spiser du vanligvis daglig? Bare ett kryss 137 1 1-2 skiver 3 Mindre enn en 2 3 eller mer 4
SYKEHEIM	
Har du vært innlagt på sykehelm i løpet av de siste 12 månedene? 123 Nei Ja, jeg har vært der en periode Ja, jeg bor der fast Hvis «Ja»: Hvor var du FØR du ble innlagt på sykeheimen siste gang? 124 Bodde i egen heim Var innlagt i sykehus Var annet sted Hvis du har vært på sykeheimen EN PERIODE i løpet av de siste 12 mndr.: Bodde du på sykeheimen passe lenge? 125 Det var for kort tid	Hville OG AVSLAPPING Hvor mange timer tilbringer du vanligvis i liggende stilling i løpet av et døgn? (nattesøvn, middagshvil) Immet immet Hvor mange timer tilbringer du vanligvis i sittende stilling i løpet av et døgn? (arbeid, måltider, TV, bil etc.) Immet immet Har du i løpet av siste måned hatt innsovnings- problemer? Bare ett kryss 140 Har du i løpet av siste måned våknet for tidlig og ikke fått sove igjen? Bare ett kryss 143 Har du i løpet av siste måned våknet for tidlig og ikke fått sove igjen? Bare ett kryss 143 Av og til 3 Ofte 1 Av og til 3 Ofte 2 Aldri 3 Ofte 2 Aldri 4
	MEDISINBRUK
KOMMUNAL HJELP ALT I ALT Hvordan er du alt i alt fornøyd med hjelpa du får fra kommunen? 126 Meget fornøyd 1 Jeg får ingen hjelp, men burde ha hatt det Nokså fornøyd 2 men burde ha hatt det Nokså misfornøyd 3 Jeg får ingen hjelp, og trenger det ikke	Har du i deler av de siste 12 måneder brukt noen medisiner daglig eller nesten daglig? Ja Nei Hvis "Ja":
KOSTHOLD	allergimedisin
Hvor mange måltider spiser du vanligvis Antai daglig (middag og brødmåltid)?	Astmamedisin
Hva slags fett blir vanligvis brukt i din husholdning? Ett kryss for matlaging og ett kryss for brød Til matlaging På brød Bruker ikke smør eller margarin 134 1 135 1 Meierismør 2 2 2 Hard margarin 3 3 3 Blet (soft) margarin blanding 5 5 5 Lettmargarin 6 6 6	VENNER Hvor mange gode venner har du? Regn med de du kan snakke fortrolig med og som kan gi deg god hjelp når du trenger det

HEIMESYKEPLEIE

		n na standard V
Hvor ofte tar du vanligvis del i foreningsvirksomhet som f.eks. syklubb, eldresenter, pensjonistforening, politiske lag, religiøse eller andre foreninger? Bare ett kryss 173 Aldri, eller noen få ganger i året	Hvor ofte er hodepinen preget av eller ledsaget av: Ett kryss på hver linje Sjelden Av og til Ofte eller aldri bankende/dunkende smerte 213 pressende smerte IIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIII	
HUMØR OG TRIVSEL	halvsidighet, vekselvis h. og v. side	
Ett kryss på hver linje Angi hvordan du har følt Noen Ganske For det deg den siste måneden: Aldri ganger ofte meste i godt humør	forverring ved fysisk aktivitet	
i dårlig humør175 🔲 🗍 🗍	Hvor mange tabletter/stikkpiller har du eventuelt brukt av disse medisinene alt i alt i løpet av den siste måneden?	
Svært Ganske Ganske Svært Er du rask til å oppfatte treg treg rask rask et humoristisk poeng? 176	Skriv 0 hvis du ikke har brukt medisinen. Cafergot Anervan Imigran	
Er du enig i at det er noe ansvarsløst over folk som stadig prøver å være morsomme? 177 Nei, slett ikke	222 224 226	
I noen grad	URINVEGS- OG PROSTATAPLAGER	
Er du en munter person? ¹⁷⁸ Nei, slett ikke	Ett kryss på hver linje	
MUSKEL-/SKJELETTPLAGER	Har du gjennomgått noe av følgende: Ja Nei	
Har du hatt plager (smerter, verk, ubehag) i Ja Nei muskler og/eller ledd i <i>den siste måneden</i> ? 178 🔲 🗌	sterilisering	
Hvis «Nei»: Gå til HODEPINE	De neste spørsmålene gjelder siste måned	
Hvis «Ja»: Hvor har du hatt disse plagene (ett eller flere kryss) og omtrent hvor mange dager tilsammen var du	Bare ett kryss for hvert hver spørsmål	
Plager (Sett kryss)	Hvor ofte har du hatt følelsen av at blæren ikke er blitt fullstendig tømt etter avsluttet vannlating? 233 Aldri	
Korsryggen	Hvor ofte har du måttet late vannet på nytt mindre enn 2 timer etter forrige vannlating? 234 Aldri	
₩ ₩ Har plagene hindret deg I å utføre dagilge Ja Nei aktiviteter den siste måneden?	Hvor ofte har du måttet stoppe og starte flere ganger under vannlatingingen? ²³⁵ Aldri	
HODEPINE		
Har du vært plaget av hodepine i løpet av de siste 12 måneder? 200 Ja, anfallsvis (migrene) 1 Antali anfall siste 12 mndr. 209 Ja, annen slags hodepine 2 Nei	Hvor ofte syns du det har vært vanskelig å holde igjen når du har følt trang til å late vannet? 236 Aldri	
Omtrent hvor mange dager pr. måned har du hodepine? Mindre enn 7 dager 1 7 til 14 dager 2 Mer enn 14 d. 3 Hvor lenge varer hodepinen vanligvis hver gang? 212 Mindre enn 4 timer 1 4 timer 3 døgn 2 Mer enn 3 døgn 3	Hvor ofte har du hatt svak urinstråle? 237 Aldri	

Hvor ofte har du måttet trykke eller presse for å begynne vamlatingen? 208 Aldri	Er du vanligvis glad eller nedstemt? 249 Svært nedstemt 1 Nokså glad
Hvor mange ganger har du vanligvis måttet stå opp i løpet av natta for å late vannet? 239` Ingen []1 2 ganger []3 4 ganger []5 1 gang []2 3 ganger []4 5 ganger eller mer []5	LEGEMLIGE FUNKSJONER Klarer du selv, uten hjelp av andre, i det daglige å: Ett kryss på hver linje Med noe Ja hjelp Gå innendørs i samme etasje 250
Hvis du resten av livet måtte leve med de vannlatings- problemene du har nå, hvordan ville du føle det? 240 Være meget godt fornøyd	Gå på toalettet
HVORDAN DU SER PÅ DEG SELV	Hvis du har hatt hjelp til noe av dette, omtrent hvor lenge har du hatt hjelp? Bare ett kryss 257 Under 3 måneder
Folk ser på seg selv på ulike måter. Kryss av for hvert utsagn hvor enig eller uenig du er. Ett kryss på hver linje Svært Svært	Hvis du trenger hjelp til ett eller flere av disse gjøremålene, hvem er det som for det meste hjelper deg?
enig Enig Uenig uenig Jeg har en positiv holdning til meg selv	Bare ett kryss Ektefelle/samboer
Jeg føler meg virkelig ubrukelig til tider	DAGLIGE OPPGAVER
Jeg føler at jeg ikke har mye å være stolt av243 🗌 🔲 🔲 🔲	Klarer du selv disse gjøremålene i det daglige uten hjelp fra andre? Ett kryss på hver linje Med noe Ja hjelp Nei Lage varm mat
Jeg føler at jeg er en verdifull person, i allefall på lik linje med andre24	Gjøre lett husarbeid (f.eks. oppvask) Gjøre tyngre husarbeid (f.eks. gulvvask)
Synes du at du har funnet et virkelig Ja Nei betydningsfulit innhold i livet ditt?245	Betale regninger Ta medisinene Komme deg ut Gjøre innkjøp Ta bussen
Føler du at du lever fullt ut?245 🗍 🗌	Hvis du trenger hjelp til ett eller flere av disse gjøremålene, omtrent hvor lenge har du hatt hjelp?
HVORDAN DU FØLER DEG NA Sett kryss i den ruta utenfor det svaret som best beskriver dine følelser den siste uka. Bare ett kryss	Bare ett kryss ₂ee Under 3 måneder
Føler du deg stort sett sterk og opplagt, eller trøtt og sliten? sliten? 247 Meget sterk og opplagt 1 Sterk og opplagt 2 Trøtt og sliten 6 Ganske sterk og opplagt 3 Både – og 4	Hvis du trenger hjelp til ett eller flere av disse gjøremålene, hvem er det som for det meste hjelper deg? Bare ett kryss 289 Ektefelle/samboer Barn/svigerbarn 2 Andre Søster/bror 3 Legg det utfylte spørreskjemaet i den ved- lagte svarkonvolutten og postlegg den så
Har du i det store og hele en rolig og god følelse Inne i deg? 248 Nesten hele tida 1 Av og til 3 Ofte 2 Aldri	snart som mulią! Porto er betalt. Hjertelig takk for hjelpa! survijer Tryweri AS-74 1630 00
an an tha dealachte ann tha start an an tha start an an tha an an tha an an tha an a	an an tha an tha

an an Arlandia an Araba an Araba. An an Araba an Araba an Araba an Araba an Araba. An Araba an Araba an Araba an Araba an Araba.

.

.

Dissertations at the Faculty of Medicine, NTNU

1977

- 1. Knut Joachim Berg: EFFECT OF ACETYLSALICYLIC ACID ON RENAL FUNCTION
- 2. Karl Erik Viken and Arne Ødegaard: STUDIES ON HUMAN MONOCYTES CULTURED IN VITRO

1.19

- 1978
- 3. Karel Bjørn Cyvin: CONGENITAL DISLOCATION OF THE HIP JOINT.
- 4. Alf O. Bridakk: METHODS FOR STUDYING FLOW DYNAMICS IN THE LEFT VENTRICLE AND THE AORTA IN MAN.
- 1979

5. Geirmund Unsgaard: CYTOSTATIC AND IMMUNOREGULATORY ABILITIES OF HUMAN BLOOD MONOCYTES CULTURED IN VITRO

- 1980
- 6. Størker Jørstad: URAEMIC TOXINS
- 7. Arne Olav Jenssen: SOME RHEOLOGICAL, CHEMICAL AND STRUCTURAL PROPERTIES
- OF MUCOID SPUTUM FROM PATIENTS WITH CHRONIC OBSTRUCTIVE BRONCHITIS
- 1981
 - 8. Jens Hammerstrøm: CYTOSTATIC AND CYTOLYTIC ACTIVITY OF HUMAN
 - MONOCYTES AND EFFUSION MACROPHAGES AGAINST TUMOR CELLS IN VITRO 1983
 - 9. Tore Syversen: EFFECTS OF METHYLMERCURY ON RAT BRAIN PROTEIN.
 - 10. Torbjørn Iversen: SQUAMOUS CELL CARCINOMA OF THE VULVA.
- 1984
- 11. Tor-Erik Widerøe: ASPECTS OF CONTINUOUS AMBULATORY PERITONEAL DIALYSIS.
- 12. Anton Hole: ALTERATIONS OF MONOCYTE AND LYMPHOCYTE FUNCTIONS IN REALTION TO SURGERY UNDER EPIDURAL OR GENERAL ANAESTHESIA.
- REALTION TO SURGERY UNDER EPIDORAL OR GENERAL ANALSTIESTA.
 13. Terje Terjesen: FRACTURE HEALING AN STRESS-PROTECTION AFTER METAL PLATE FIXATION AND EXTERNAL FIXATION.
- 14. Carsten Saunte: CLUSTER HEADACHE SYNDROME.
- 15. Inggard Lereim: TRAFFIC ACCIDENTS AND THEIR CONSEQUENCES.
- 16. Bjørn Magne Eggen: STUDIES IN CYTOTOXICITY IN HUMAN ADHERENT MONONUCLEAR BLOOD CELLS.
- 17. Trond Haug: FACTORS REGULATING BEHAVIORAL EFFECTS OG DRUGS.
- 1985
- 18. Sven Erik Gisvold: RESUSCITATION AFTER COMPLETE GLOBAL BRAIN ISCHEMIA.
- 19. Terje Espevik: THE CYTOSKELETON OF HUMAN MONOCYTES.
- 20. Lats Bevanger: STUDIES OF THE Ibc (c) PROTEIN ANTIGENS OF GROUP B STREPTOCOCCI.
- 21. Ole-Jan Iversen: RETROVIRUS-LIKE PARTICLES IN THE PATHOGENESIS OF PSORIASIS.
- 22. Lasse Eriksen: EVALUATION AND TREATMENT OF ALCOHOL DEPENDENT BEHAVIOUR.
- 23. Per I. Lundmo: ANDROGEN METABOLISM IN THE PROSTATE.
- 1986
- 24. Dagfinn Berntzen: ANALYSIS AND MANAGEMENT OF EXPERIMENTAL AND CLINICAL PAIN.
- 25. Odd Arnold Kildahl-Andersen: PRODUCTION AND CHARACTERIZATION OF MONOCYTE-DERIVED CYTOTOXIN AND ITS ROLE IN MONOCYTE-MEDIATED CYTOTOXICITY.
- 26. Ola Dale: VOLATILE ANAESTHETICS.
- 1987
- 27. Per Martin Kleveland: STUDIES ON GASTRIN.
- 28. Audun N. Øksendal: THE CALCIUM PARADOX AND THE HEART.
- 29. Vilhjalmur R. Finsen: HIP FRACTURES
- 1988
- Rigmor Austgulen: TUMOR NECROSIS FACTOR: A MONOCYTE-DERIVED REGULATOR OF CELLULAR GROWTH.
- 31. Tom-Harald Edna: HEAD INJURIES ADMITTED TO HOSPITAL.
- 32. Joseph D. Borsi: NEW ASPECTS OF THE CLINICAL PHARMACOKINETICS OF METHOTREXATE.

- 33. Olav F. M. Sellevold: GLUCOCORTICOIDS IN MYOCARDIAL PROTECTION.
- 34. Terje Skjærpe: NONINVASIVE QUANTITATION OF GLOBAL PARAMETERS ON LEFT VENTRICULAR FUNCTION: THE SYSTOLIC PULMONARY ARTERY PRESSURE AND CARDIAC OUTPUT.
- 35. Eyvind Rødahl: STUDIES OF IMMUNE COMPLEXES AND RETROVIRUS-LIKE ANTIGENS IN PATIENTS WITH ANKYLOSING SPONDYLITIS.

- 36. Ketil Thorstensen: STUDIES ON THE MECHANISMS OF CELLULAR UPTAKE OF IRON FROM TRANSFERRIN.
- 37. Anna Midelfart: STUDIES OF THE MECHANISMS OF ION AND FLUID TRANSPORT IN THE BOVINE CORNEA.
- 38. Eirik Helseth: GROWTH AND PLASMINOGEN ACTIVATOR ACTIVITY OF HUMAN GLIOMAS AND BRAIN METASTASES - WITH SPECIAL REFERENCE TO TRANSFORMING GROWTH FACTOR BETA AND THE EPIDERMAL GROWTH FACTOR RECEPTOR.
- 39. Petter C. Borchgrevink: MAGNESIUM AND THE ISCHEMIC HEART.
- 40. Kjell-Ame Rein: THE EFFECT OF EXTRACORPOREAL CIRCULATION ON SUBCUTANEOUS TRANSCAPILLARY FLUID BALANCE.
- 41. Ame Kristian Sandvik: RAT GASTRIC HISTAMINE.
- 42. Carl Bredo Dahl: ANIMAL MODELS IN PSYCHIATRY.
- 1989
- 43. Torbjørn A. Fredriksen: CERVICOGENIC HEADACHE.
- 44. Rolf A. Walstad: CEFTAZIDIME.
- 45. Rolf Salvesen: THE PUPIL IN CLUSTER HEADACHE.
- 46. Nils Petter Jørgensen: DRUG EXPOSURE IN EARLY PREGNANCY.
- 47. Johan C. Ræder: PREMEDICATION AND GENERAL ANAESTHESIA IN OUTPATIENT GYNECOLOGICAL SURGERY.
- 48. M. R. Shalaby: IMMUNOREGULATORY PROPERTIES OF TNF-α AND THE RELATED CYTOKINES.
- 49. Anders Waage: THE COMPLEX PATTERN OF CYTOKINES IN SEPTIC SHOCK.
- 50. Bjarne Christian Eriksen: ELECTROSTIMULATION OF THE PELVIC FLOOR IN FEMALE URINARY INCONTINENCE.
- 51. Tore B. Halvorsen: PROGNOSTIC FACTORS IN COLORECTAL CANCER.
- 1990
- 52. Asbjørn Nordby: CELLULAR TOXICITY OF ROENTGEN CONTRAST MEDIA.
- 53. Kåre E. Tvedt: X-RAY MICROANALYSIS OF BIOLOGICAL MATERIAL.
- 54. Tore C. Stiles: COGNITIVE VULNERABILITY FACTORS IN THE DEVELOPMENT AND MAINTENANCE OF DEPRESSION.
- 55. Eva Hofsli: TUMOR NECROSIS FACTOR AND MULTIDRUG RESISTANCE.
- 56. Helge S. Haarstad: TROPHIC EFFECTS OF CHOLECYSTOKININ AND SECRETIN ON THE RAT PANCREAS.
- 57. Lars Engebretsen: TREATMENT OF ACUTE ANTERIOR CRUCIATE LIGAMENT INJURIES.
- 58. Tarjei Rygnestad; DELIBERATE SELF-POISONING IN TRONDHEIM.
- Ame Z. Henriksen: STUDIES ON CONSERVED ANTIGENIC DOMAINS ON MAJOR OUTER MEMBRANE PROTEINS FROM ENTEROBACTERIA.
- Steinar Westin: UNEMPLOYMENT AND HEALTH: Medical and social consequences of a factory closure in a ten-year controlled follow-up study.
- 61. YIva Sahlin: INJURY REGISTRATION, a tool for accident preventive work.
- 62. Helge Bjørnstad Pettersen: BIOSYNTHESIS OF COMPLEMENT BY HUMAN ALVEOLAR MACROPHAGES WITH SPECIAL REFERENCE TO SARCOIDOSIS.
- 63. Berit Schei: TRAPPED IN PAINFUL LOVE.
- 64. Lars J. Vatten: PROSPECTIVE STUDIES OF THE RISK OF BREAST CANCER IN A COHORT OF NORWEGIAN WOMAN.
- 1991
- 65. Kåre Bergh: APPLICATIONS OF ANTI-C5a SPECIFIC MONOCLONAL ANTIBODIES FOR THE ASSESSMENT OF COMPLEMENT ACTIVATION.
- 66. Svein Svenningsen: THE CLINICAL SIGNIFICANCE OF INCREASED FEMORAL ANTEVERSION.
- 67. Olbjørn Klepp: NONSEMINOMATOUS GERM CELL TESTIS CANCER: THERAPEUTIC OUTCOME AND PROGNOSTIC FACTORS.

- 68. Trond Sand: THE EFFECTS OF CLICK POLARITY ON BRAINSTEM AUDITORY EVOKED POTENTIALS AMPLITUDE, DISPERSION, AND LATENCY VARIABLES.
- Kjetil B. Åsbakk: STUDIES OF A PROTEIN FROM PSORIATIC SCALE, PSO P27, WITH RESPECT TO ITS POTENTIAL ROLE IN IMMUNE REACTIONS IN PSORIASIS.
 Arnulf Hestnes: STUDIES ON DOWN'S SYNDROME.
- Arhun Hesines: STUDIES ON DOWN 3 STUDIOME.
 Randi Nygaard: LONG-TERM SURVIVAL IN CHILDHOOD LEUKEMIA.
- 72. Bjørn Hagen: THIO-TEPA.
- 73. Svein Anda: EVALUATION OF THE HIP JOINT BY COMPUTED TOMOGRAMPHY AND ULTRASONOGRAPHY.
- 1992
- 74. Martin Svartberg: AN INVESTIGATION OF PROCESS AND OUTCOME OF SHORT-TERM PSYCHODYNAMIC PSYCHOTHERAPY.
- 75. Stig Arild Slørdahl: AORTIC REGURGITATION.
- 76. Harold C Sexton: STUDIES RELATING TO THE TREATMENT OF SYMPTOMATIC NON-PSYCHOTIC PATIENTS.
- 77. Maurice B. Vincent: VASOACTIVE PEPTIDES IN THE OCULAR/FOREHEAD AREA.
- 78. Terje Johannessen: CONTROLLED TRIALS IN SINGLE SUBJECTS.
- 79. Turid Nilsen: PYROPHOSPHATE IN HEPATOCYTE IRON METABOLISM.
- 80. Olav Haraldseth: NMR SPECTROSCOPY OF CEREBRAL ISCHEMIA AND REPERFUSION IN RAT.

81. Eiliv Brenna: REGULATION OF FUNCTION AND GROWTH OF THE OXYNTIC MUCOSA. 1993

- 82. Gunnar Bovim: CERVICOGENIC HEADACHE.
- 83. Jarl Arne Kahn: ASSISTED PROCREATION.
- 84. Bjørn Naume: IMMUNOREGULATORY EFFECTS OF CYTOKINES ON NK CELLS.
- 85. Rune Wiseth: AORTIC VALVE REPLACEMENT.
- 86. Jie Ming Shen: BLOOD FLOW VELOCITY AND RESPIRATORY STUDIES.
- 87. Piotr Kruszewski: SUNCT SYNDROME WITH SPECIAL REFERENCE TO THE AUTONOMIC NERVOUS SYSTEM.
- 88. Mette Haase Moen: ENDOMETRIOSIS.
- 89. Anne Vik: VASCULAR GAS EMBOLISM DURING AIR INFUSION AND AFTER DECOMPRESSION IN PIGS.
- 90. Lars Jacob Stovner: THE CHIARI TYPE I MALFORMATION.
- 91. Kjell Å. Salvesen: ROUTINE ULTRASONOGRAPHY IN UTERO AND DEVELOPMENT IN CHILDHOOD.
- 1994
- 92. Nina-Beate Liabakk: DEVELOPMENT OF IMMUNOASSAYS FOR TNF AND ITS SOLUBLE RECEPTORS.
- 93. Sverre Helge Torp: erbB ONCOGENES IN HUMAN GLIOMAS AND MENINGIOMAS.
- 94. Olav M. Linaker: MENTAL RETARDATION AND PSYCHIATRY. Past and present.
- 95. Per Oscar Feet: INCREASED ANTIDEPRESSANT AND ANTIPANIC EFFECT IN COMBINED TREATMENT WITH DIXYRAZINE AND TRICYCLIC ANTIDEPRESSANTS.
- Stein Olav Samstad: CROSS SECTIONAL FLOW VELOCITY PROFILES FROM TWO-DIMENSIONAL DOPPLER ULTRASOUND: Studies on early mitral blood flow.
- 97. Biørn Backe: STUDIES IN ANTENATAL CARE.
- 98. Gerd Inger Ringdal: QUALITY OF LIFE IN CANCER PATIENTS.
- 99. Torvid Kiserud: THE DUCTUS VENOSUS IN THE HUMAN FETUS.
- 100. Hans E. Fjøsne: HORMONAL REGULATION OF PROSTATIC METABOLISM.
- 101.Eylert Brodtkorb: CLINICAL ASPECTS OF EPILEPSY IN THE MENTALLY RETARDED.
- 102.Roar Juul: PEPTIDERGIC MECHANISMS IN HUMAN SUBARACHNOID HEMORRHAGE.
- 103. Unni Syversen: CHROMOGRANIN A. Physiological and Clinical Role.
- 1995

104.0dd Gunnar Brakstad: THERMOSTABLE NUCLEASE AND THE nuc GENE IN THE DIAGNOSIS OF Staphylococcus aureus INFECTIONS.

- 105. Terje Engan: NUCLEAR MAGNETIC RESONANCE (NMR) SPECTROSCOPY OF PLASMA IN MALIGNANT DISEASE.
- 106.Kirsten Rasmussen: VIOLENCE IN THE MENTALLY DISORDERED.
- 107. Finn Egil Skjeldestad: INDUCED ABORTION: Timetrends and Determinants.

108.Roar Stenseth: THORACIC EPIDURAL ANALGESIA IN AORTOCORONARY BYPASS SURGERY.

- 110.Svend Aakhus: NONINVASIVE COMPUTERIZED ASSESSMENT OF LEFT VENTRICULAR FUNCTION AND SYSTEMIC ARTERIAL PROPERTIES. Methodology and some clinical applications.
- 111.Klaus-Dieter Bolz: INTRAVASCULAR ULTRASONOGRAPHY.
- 112.Petter Aadahl: CARDIOVASCULAR EFFECTS OF THORACIC AORTIC CROSS-CLAMPING.
- 113. Sigurd Steinshamn: CYTOKINE MEDIATORS DURING GRANULOCYTOPENIC INFECTIONS.
- 114. Hans Stifoss-Hanssen: SEEKING MEANING OR HAPPINESS?
- 115. Anne Kvikstad: LIFE CHANGE EVENTS AND MARITAL STATUS IN RELATION TO RISK AND PROGNOSIS OF CANSER.
- 116. Torbjørn Grøntvedt: TREATMENT OF ACUTE AND CHRONIC ANTERIOR CRUCIATE LIGAMENT INJURIES. A clinical and biomechanical study.
- 117. Sigrid Hørven Wigers: CLINICAL STUDIES OF FIBROMYALGIA WITH FOCUS ON ETIOLOGY, TREATMENT AND OUTCOME.
- 118.Jan Schjøtt: MYOCARDIAL PROTECTION: Functional and Metabolic Characteristics of Two Endogenous Protective Principles.
- 119.Marit Martinussen: STUDIES OF INTESTINAL BLOOD FLOW AND ITS RELATION TO TRANSITIONAL CIRCULATORY ADAPATION IN NEWBORN INFANTS.
- 120. Tomm B. Müller: MAGNETIC RESONANCE IMAGING IN FOCAL CEREBRAL ISCHEMIA. 121. Rune Haaverstad: OEDEMA FORMATION OF THE LOWER EXTREMITIES.
- 122. Magne Børset: THE ROLE OF CYTOKINES IN MULTIPLE MYELOMA, WITH SPECIAL REFERENCE TO HEPATOCYTE GROWTH FACTOR.
- 123. Geir Smedslund: A THEORETICAL AND EMPIRICAL INVESTIGATION OF SMOKING, STRESS AND DISEASE: RESULTS FROM A POPULATION SURVEY.

1997

- 124. Torstein Vik: GROWTH, MORBIDITY, AND PSYCHOMOTOR DEVELOPMENT IN INFANTS WHO WERE GROWTH RETARDED IN UTERO.
- 125. Siri Forsmo: ASPECTS AND CONSEQUENCES OF OPPORTUNISTIC SCREENING FOR CERVICAL CANCER. Results based on data from three Norwegian counties.
- 126. Jon S. Skranes: CEREBRAL MRI AND NEURODEVELOPMENTAL OUTCOME IN VERY LOW BIRTH WEIGHT (VLBW) CHILDREN. A follow-up study of a geographically based year cohort of VLBW children at ages one and six years.
- 127.Knut Bjørnstad: COMPUTERĬZED ECHOCĂRDIOGRAPHY FOR EVALUTION OF CORONARY ARTERY DISEASE.
- 128.Grethe Elisabeth Borchgrevink: DIAGNOSIS AND TREATMENT OF WHIPLASH/NECK SPRAIN INJURIES CAUSED BY CAR ACCIDENTS.
- 129. Tor Elsås: NEUROPEPTIDES AND NITRIC OXIDE SYNTHASE IN OCULAR AUTONOMIC AND SENSORY NERVES.
- 130. Rolf W. Gråwe: EPIDEMIOLOGICAL AND NEUROPSYCHOLOGICAL PERSPECTIVES ON SCHIZOPHRENIA.
- 131. Tonje Strømholm: CEREBRAL HAEMODYNAMICS DURING THORACIC AORTIC CROSSCLAMPING. An experimental study in pigs.

- 132.Martinus Bråten: STUDIES ON SOME PROBLEMS REALTED TO INTRAMEDULLARY NAILING OF FEMORAL FRACTURES.
- 133. Ståle Nordgård: PROLIFERATIVE ACTIVITY AND DNA CONTENT AS PROGNOSTIC INDICATORS IN ADENOID CYSTIC CARCINOMA OF THE HEAD AND NECK.
- 134. Egil Lien: SOLUBLE RECEPTORS FOR TNF AND LPS: RELEASE PATTERN AND POSSIBLE SIGNIFICANCE IN DISEASE.
- 135.Marit Bjørgaas: HYPOGLYCAEMIA IN CHILDREN WITH DIABETES MELLITUS
- 136.Frank Skorpen: GENETIC AND FUNCTIONAL ANALYSES OF DNA REPAIR IN HUMAN CELLS.
- 137.Juan A. Pareja: SUNCT SYNDROME. ON THE CLINICAL PICTURE. ITS DISTINCTION FROM OTHER, SIMILAR HEADACHES.
- 138. Anders Angelsen: NEUROENDOCRINE CELLS IN HUMAN PROSTATIC CARCINOMAS AND THE PROSTATIC COMPLEX OF RAT, GUINEA PIG, CAT AND DOG.

- 139. Fabio Antonaci: CHRONIC PAROXYSMAL HEMICRANIA AND HEMICRANIA CONTINUA: TWO DIFFERENT ENTITIES?
- 140. Sven M. Carlsen: ENDOCRINE AND METABOLIC EFFECTS OF METFORMIN WITH SPECIAL EMPHASIS ON CARDIOVASCULAR RISK FACTORES.

- 141. Terje A. Murberg: DEPRESSIVE SYMPTOMS AND COPING AMONG PATIENTS WITH CONGESTIVE HEART FAILURE.
- 142.Harm-Gerd Karl Blaas: THE EMBRYONIC EXAMINATION. Ultrasound studies on the development of the human embryo.
- 143. Noèmi Becser Andersen: THE CEPHALIC SENSORY NERVES IN UNILATERAL HEADACHES. Anatomical background and neurophysiological evaluation.
- 144. Eli-Janne Fiskerstrand: LASER TREATMENT OF PORT WINE STAINS. A study of the efficacy and limitations of the pulsed dye laser. Clinical and morfological analyses aimed at improving the therapeutic outcome.
- 145.Bård Kulseng: A STUDY OF ALGINATE CAPSULE PROPERTIES AND CYTOKINES IN RELATION TO INSULIN DEPENDENT DIABETES MELLITUS.
- 146. Terje Haug: STRUCTURE AND REGULATION OF THE HUMAN UNG GENE ENCODING URACIL-DNA GLYCOSYLASE.
- 147. Heidi Brurok: MANGANESE AND THE HEART. A Magic Metal with Diagnostic and Therapeutic Possibilites.
- 148. Agnes Kathrine Lie: DIAGNOSIS AND PREVALENCE OF HUMAN PAPILLOMAVIRUS INFECTION IN CERVICAL INTRAEPITELIAL NEOPLASIA. Relationship to Cell Cycle Regulatory Proteins and HLA DQBI Genes.
- 149. Ronald Márvik: PHARMACOLOGICAL, PHYSIOLOGICAL AND PATHOPHYSIOLOGICAL STUDIES ON ISOLATED STOMACS.
- 150.Ketil Jarl Holen: THE ROLE OF ULTRASONOGRAPHY IN THE DIAGNOSIS AND TREATMENT OF HIP DYSPLASIA IN NEWBORNS.
- 151. Irene Hetlevik: THE ROLE OF CLINICAL GUIDELINES IN CARDIOVASCULAR RISK INTERVENTION IN GENERAL PRACTICE.
- 152.Katarina Tunòn: ULTRASOUND AND PREDICTION OF GESTATIONAL AGE.
- 153. Johannes Soma: INTERACTION BETWEEN THE LEFT VENTRICLE AND THE SYSTEMIC ARTERIES.
- 154. Arild Aamodt: DEVELOPMENT AND PRE-CLINICAL EVALUATION OF A CUSTOM-MADE FEMORAL STEM.
- 155. Agnar Tegnander: DIAGNOSIS AND FOLLOW-UP OF CHILDREN WITH SUSPECTED OR KNOWN HIP DYSPLASIA.
- 156.Bent Indredavik: STROKE UNIT TREATMENT: SHORT AND LONG-TERM EFFECTS
- 157. Jolanta Vanagaite Vingen: PHOTOPHOBIA AND PHONOPHOBIA IN PRIMARY HEADACHES
- 2000
- 158.Ola Dalsegg Sæther: PATHOPHYSIOLOGY DURING PROXIMAL AORTIC CROSS-CLAMPING
- 159. CLINICAL AND EXPERIMENTAL STUDIES
- 160. Christina Vogt Isaksen: PRENATAL ULTRASOUND AND POSTMORTEM FINDINGS A TEN YEAR CORRELATIVE STUDY OF FETUSES AND INFANTS WITH DEVELOPMENTAL ANOMALIES.
- 161.Holger Seidel: HIGH-DOSE METHOTREXATE THERAPY IN CHILDREN WITH ACUTE LYMPHOCYTIC LEUKEMIA: DOSE, CONCENTRATION, AND EFFECT CONSIDERATIONS.
- 162. Stein Hallan: IMPLEMENTATION OF MODERN MEDICAL DECISION ANALYSIS INTO CLINICAL DIAGNOSIS AND TREATMENT.
- 163.Malcolm Suc-Chu: INVASIVE AND NON-INVASIVE STUDIES IN CROSS-COUNTRY SKIERS WITH ASTHMA-LIKE SYMPTOMS.
- 164.Ole-Lars Brekke: EFFECTS OF ANTIOXIDANTS AND FATTY ACIDS ON TUMOR NECROSIS FACTOR-INDUCED CYTOTOXICITY.
- 165. Jan Lundbom: AORTOCORONARY BYPASS SURGERY: CLINICAL ASPECTS, COST CONSIDERATIONS AND WORKING ABILITY.
- 166. John-Anker Zwart: LUMBAR NERVE ROOT COMPRESSION, BIOCHEMICAL AND NEUROPHYSIOLOGICAL ASPECTS.
- 167.Geir Falck: HYPEROSMOLALITY AND THE HEART.

- 168.Eirik Skogvoll: CARDIAC ARREST Incidence, Intervention and Outcome.
- 169. Dalius Bansevicius: SHOULDER-NECK REGION IN CERTAIN HEADACHES AND CHRONIC PAIN SYNDROMES.
- 170.Bettina Kinge: REFRACTIVE ERRORS AND BIOMETRIC CHANGES AMONG UNIVERSITY STUDENTS IN NORWAY.
- 171. Gunnar Qvigstad: CONSEQUENCES OF HYPERGASTRINEMIA IN MAN
- 172. Hanne Ellekjær: EPIDEMIOLOGICAL STUDIES OF STROKE IN A NORWEGIAN
- POPULATION. INCIDENCE, RISK FACTORS AND PROGNOSIS 173. Hilde Grimstad: VIOLENCE AGAINST WOMEN AND PREGNANCY OUTCOME.
- 173. Astrid Hjelde: SURFACE TENSION AND COMPLEMENT ACTIVATION: Factors influencing bubble formation and bubble effects after decompression.
- 175.Kjell A. Kvistad: MR IN BREAST CANCER A CLINICAL STUDY.
- 176. Ivar Rossvoll: ELECTIVE ORTHOPAEDIC SURGERY IN A DEFINED POPULATION. Studies on demand, waiting time for treatment and incapacity for work.
- 177. Carina Seidel: PROGNOSTIC VALUE AND BIOLOGICAL EFFECTS OF HEPATOCYTE
- GROWTH FACTOR AND SYNDECAN-1 IN MULTIPLE MYELOMA. 2001
- 178. Alexander Wahba: THE INFLUENCE OF CARDIOPULMONARY BYPASS ON PLATELET FUNCTION AND BLOOD COAGULATION – DETERMINANTS AND CLINICAL CONSEQUENSES
- 179.Marcus Schmitt-Egenolf: THE RELEVANCE OF THE MAJOR hISTOCOMPATIBILITY COMPLEX FOR THE GENETICS OF PSORIASIS
- 180. Odrun Arna Gederaas: BIOLOGICAL MECHANISMS INVOLVED IN 5-AMINOLEVULINIC ACID BASED PHOTODYNAMIC THERAPY
- 181. Pål Richard Romundstad: CANCER INCIDENCE AMONG NORWEGIAN ALUMINIUM WORKERS
- 182.Henrik Hjorth-Hansen: NOVEL CYTOKINES IN GROWTH CONTROL AND BONE DISEASE OF MULTIPLE MYELOMA
- 183. Gunnar Morken: SEASONAL VARIATION OF HUMAN MOOD AND BEHAVIOUR
- 184.Bjørn Olav Haugen: MEASUREMENT OF CARDIAC OUTPUT AND STUDIES OF VELOCITY PROFILES IN AORTIC AND MITRAL FLOW USING TWO- AND THREE-
- DIMENSIONAL COLOUR FLOW IMAGING 185.Geir Bråthen: THE CLASSIFICATION AND CLINICAL DIAGNOSIS OF ALCOHOL-RELATED SEIZURES
- 186. Knut Ivar Aasarød: RENAL INVOLVEMENT IN INFLAMMATORY RHEUMATIC DISEASE. A Study of Renal Disease in Wegener's Granulomatosis and in Primary Sjögren's Syndrome
- 187. Trude Helen Flo: RESEPTORS INVOLVED IN CELL ACTIVATION BY DEFINED URONIC ACID POLYMERS AND BACTERIAL COMPONENTS
- 188.Bodil Kavli: HUMAN URACIL-DNA GLYCOSYLASES FROM THE UNG GENE: STRUCTRUAL BASIS FOR SUBSTRATE SPECIFICITY AND REPAIR
- 189, Liv Thommesen: MOLECULAR MECHANISMS INVOLVED IN TNF- AND GASTRIN-MEDIATED GENE REGULATION
- 190. Turid Lingaas Holmen: SMOKING AND HEALTH IN ADOLESCENCE; THE NORD-TRØNDELAG HEALTH STUDY, 1995-97
- 191.Øyvind Hjertner: MULTIPLE MYELOMA: INTERACTIONS BETWEEN MALIGNANT PLASMA CELLS AND THE BONE MICROENVIRONMENT
- 192. Asbjørn Støylen: STRAIN RATE IMAGING OF THE LEFT VENTRICLE BY ULTRASOUND. FEASIBILITY, CLINICAL VALIDATION AND PHYSIOLOGICAL ASPECTS
- 193.Kristian Midthjell: DIABETES IN ADULTS IN NORD-TRØNDELAG. PUBLIC HEALTH ASPECTS OF DIABETES MELLITUS IN A LARGE, NON-SELECTED NORWEGIAN POPULATION.
- 194. Guanglin Cui: FUNCTIONAL ASPECTS OF THE ECL CELL IN RODENTS
- 195.Ulrik Wisløff: CARDIAC EFFECTS OF AEROBIC ENDURANCE TRAINING: HYPERTROPHY, CONTRACTILITY AND CALCUIM HANDLING IN NORMAL AND FAILING HEART
- 196.Øyvind Halaas: MECHANISMS OF IMMUNOMODULATION AND CELL-MEDIATED CYTOTOXICITY INDUCED BY BACTERIAL PRODUCTS
- 197. Tore Amundsen: PERFUSION MR IMAGING IN THE DIAGNOSIS OF PULMONARY EMBOLISM

- 198. Nanna Kurtze: THE SIGNIFICANCE OF ANXIETY AND DEPRESSION IN FATIQUE AND PATTERNS OF PAIN AMONG INDIVIDUALS DIAGNOSED WITH FIBROMYALGIA: RELATIONS WITH QUALITY OF LIFE, FUNCTIONAL DISABILITY, LIFESTYLE, EMPLOYMENT STATUS, CO-MORBIDITY AND GENDER
- 199. Tom Ivar Lund Nilsen: PROSPECTIVE STUDIES OF CANCER RISK IN NORD-TRØNDELAG: THE HUNT STUDY. Associations with anthropometric, socioeconomic, and lifestyle risk factors
- 200. Asta Kristine Håberg: A NEW APPROACH TO THE STUDY OF MIDDLE CEREBRAL ARTERY OCCLUSION IN THE RAT USING MAGNETIC RESONANCE TECHNIQUES 2002
- 201 Knut Jørgen Arntzen: PREGNANCY AND CYTOKINES
- 202. Henrik Døllner: INFLAMMATORY MEDIATORS IN PERINATAL INFECTIONS
- 203. Asta Bye: LOW FAT, LOW LACTOSE DIET USED AS PROPHYLACTIC TREATMENT OF ACUTE INTESTINAL REACTIONS DURING PELVIC RADIOTHERAPY. A PROSPECTIVE RANDOMISED STUDY.
- 204. Sylvester Moyo: STUDIES ON STREPTOCOCCUS AGALACTIAE (GROUP B STREPTOCOCCUS) SURFACE-ANCHORED MARKERS WITH EMPHASIS ON STRAINS AND HUMAN SERA FROM ZIMBABWE.

205.Knut Hagen: HEAD-HUNT: THE EPIDEMIOLOGY OF HEADACHE IN NORD-TRØNDELAG 206.Li Lixin: ON THE REGULATION AND ROLE OF UNCOUPLING PROTEIN-2 IN INSULIN PRODUCING &-CELLS

207. Anne Hildur Henriksen: SYMPTOMS OF ALLERGY AND ASTHMA VERSUS MARKERS OF LOWER AIRWAY INFLAMMATION AMONG ADOLESCENTS

- 208. Egil Andreas Fors: NON-MALIGNANT PAIN IN RELATION TO PSYCHOLOGICAL AND ENVIRONTENTAL FACTORS. EXPERIENTAL AND CLINICAL STUDES OF PAIN WITH FOCUS ON FIBROMYALGIA
- 209. Pål Klepstad: MORPHINE FOR CANCER PAIN
- 210. Ingunn Bakke: MECHANISMS AND CONSEQUENCES OF PEROXISOME PROLIFERATOR-INDUCED HYPERFUNCTION OF THE RAT GASTRIN PRODUCING CELL
- 211. Ingrid Susann Gribbestad: MAGNETIC RESONANCE IMAGING AND SPECTROSCOPY OF BREAST CANCER
- 212. Rønnaug Astri Ødegård: PREECLAMPSIA MATERNAL RISK FACTORS AND FETAL GROWTH
- 213. Johan Haux: STUDIES ON CYTOTOXICITY INDUCED BY HUMAN NATURAL KILLER CELLS AND DIGITOXIN
- 214. Turid Suzanne Berg-Nielsen: PARENTING PRACTICES AND MENTALLY DISORDERED ADOLESCENTS
- 215.Astrid Rydning: BLOOD FLOW AS A PROTECTIVE FACTOR FOR THE STOMACH MUCOSA. AN EXPERIMENTAL STUDY ON THE ROLE OF MAST CELLS AND SENSORY AFFERENT NEURONS

- 216.Jan Pål Loennechen: HEART FAILURE AFTER MYOCARDIAL INFARCTION. Regional Differences, Myocyte Function, Gene Expression, and Response to Cariporide, Losartan, and Exercise Training.
- 217. Elisabeth Qvigstad: EFFECTS OF FATTY ACIDS AND OVER-STIMULATION ON INSULIN SECRETION IN MAN
- 218. Arne Åsberg: EPIDEMIOLOGICAL STUDIES IN HEREDITARY HEMOCHROMATOSIS: PREVALENCE, MORBIDITY AND BENEFIT OF SCREENING.
- 219. Johan Fredrik Skomsvoll: REPRODUCTIVE OUTCOME IN WOMEN WITH RHEUMATIC DISEASE. A population registry based study of the effects of inflammatory rheumatic disease and connective tissue disease on reproductive outcome in Norwegian women in 1967-1995.
- 220. Siv Mørkved: URINARY INCONTINENCE DURING PREGNANCY AND AFTER DELIVERY: EFFECT OF PELVIC FLOOR MUSCLE TRAINING IN PREVENTION AND TREATMENT
- 221. Marit S. Jordhøy: THE IMPACT OF COMPREHENSIVE PALLIATIVE CARE
- 222. Tom Christian Martinsen: HYPERGASTRINEMIA AND HYPOACIDITY IN RODENTS -CAUSES AND CONSEQUENCES
- 223. Solveig Tingulstad: CENTRALIZATION OF PRIMARY SURGERY FOR OVARAIN CANCER. FEASIBILITY AND IMPACT ON SURVIVAL

- 224.Haytham Eloqayli: METABOLIC CHANGES IN THE BRAIN CAUSED BY EPILEPTIC SEIZURES
- 225. Torunn Bruland: STUDIES OF EARLY RETROVIRUS-HOST INTERACTIONS VIRAL DETERMINANTS FOR PATHOGENESIS AND THE INFLUENCE OF SEX ON THE SUSCEPTIBILITY TO FRIEND MURINE LEUKAEMIA VIRUS INFECTION
- 226. Torstein Hole: DOPPLER ECHOCARDIOGRAPHIC EVALUATION OF LEFT VENTRICULAR FUNCTION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION 227. Vibeke Nossum: THE EFFECT OF VASCULAR BUBBLES ON ENDOTHELIAL FUNCTION
- 228. Sigurd Fasting: ROUTINE BASED RECORDING OF ADVERSE EVENTS DURING
- AÑAESTHEŠIA APPLICATION IN QUALITY IMPROVEMENT AND SAFETY 229. Solfrid Romundstad: EPIDEMIOLOGICAL STUDIES OF MICROALBUMINURIA. THE NORD-TRØNDELAG HEALTH STUDY 1995-97 (HUNT 2)
- 230.Geir Torheim: PROCESSING OF DYNAMIC DATA SETS IN MAGNETIC RESONANCE IMAGING
- 231. Catrine Ahlén: SKIN INFECTIONS IN OCCUPATIONAL SATURATION DIVERS IN THE NORTH SEA AND THE IMPACT OF THE ENVIRONMENT
- 232. Amulf Langhammer: RESPIRATORY SYMPTOMS, LUNG FUNCTION AND BONE MINERAL DENSITY IN A COMPREHENSIVE POPULATION SURVEY. THE NORD-TRØNDELAG HEALTH STUDY 1995-97. THE BRONCHIAL OBSTRUCTION IN NORD-TRØNDELAG STUDY
- 233.Einar Kjelsås: EATING DISORDERS AND PHYSICAL ACTIVITY IN NON-CLINICAL SAMPLES
- 234. Ame Wibe: RECTAL CANCER TREATMENT IN NORWAY STANDARDISATION OF SURGERY AND QUALITY ASSURANCE
- 2004
- 235. Eivind Witsø: BONE GRAFT AS AN ANTIBIOTIC CARRIER
- 236.Anne Mari Sund: DEVELOPMENT OF DEPRESSIVE SYMPTOMS IN EARLY ADOLESCENCE
- 237.Hallvard Lærum: EVALUATION OF ELECTRONIC MEDICAL RECORDS A CLINICAL TASK PERSPECTIVE
- 238. Gustav Mikkelsen: ACCESSIBILITY OF INFORMATION IN ELECTRONIC PATIENT RECORDS; AN EVALUATION OF THE ROLE OF DATA QUALITY
- 239. Steinar Krokstad: SOCIOECONOMIC INEQUALITIES IN HEALTH AND DISABILITY. SOCIAL EPIDEMIOLOGY IN THE NORD-TRØNDELAG HEALTH STUDY (HUNT), NORWAY
- 240. Ame Kristian Myhre: NORMAL VARIATION IN ANOGENITAL ANATOMY AND MICROBIOLOGY IN NON-ABUSED PRESCHOOL CHILDREN
- 241. Ingunn Dybedal: NEGATIVE REGULATORS OF HEMATOPOIETEC STEM AND PROGENITOR CELLS
- 242.Beate Sitter: TISSUE CHARACTERIZATION BY HIGH RESOLUTION MAGIC ANGLE SPINNING MR SPECTROSCOPY
- 243.Per Ame Aas: MACROMOLECULAR MAINTENANCE IN HUMAN CELLS REPAIR OF URACIL IN DNA AND METHYLATIONS IN DNA AND RNA
- 244. Anna Bofin: FINE NEEDLE ASPIRATION CYTOLOGY IN THE PRIMARY INVESTIGATION OF BREAST TUMOURS AND IN THE DETERMINATION OF TREATMENT STRATEGIES
- 245. Jim Aage Nøttestad: DEINSTITUTIONALIZATION AND MENTAL HEALTH CHANGES AMONG PEOPLE WITH MENTAL RETARDATION
- 246.Reidar Fossmark: GASTRIC CANCER IN JAPANESE COTTON RATS
- 247. Wibeke Nordhøy: MANGANESE AND THE HEART, INTRACELLULAR MR RELAXATION AND WATER EXCHANGE ACROSS THE CARDIAC CELL MEMBRANE
- 2005
- 248.Sturla Molden: QUANTITATIVE ANALYSES OF SINGLE UNITS RECORDED FROM THE HIPPOCAMPUS AND ENTORHINAL CORTEX OF BEHAVING RATS
- 249. Wenche Brenne Drøyvold: EPIDEMIOLOGICAL STUDIES ON WEIGHT CHANGE AND HEALTH IN A LARGE POPULATION. THE NORD-TRØNDELAG HEALTH STUDY (HUNT)