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Blood pressure, obesity, serum iron and lipids as risk factors of ischaemic heart disease

The HUNT study, Norway

Thesis for the degree of Philosophiae Doctor

Trondheim, January 2012

Norwegian University of Science and Technology
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Science and Technology

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ISBN 978-82-471-3266-1 (printed ver.)
ISBN 978-82-471-3267-8 (electronic ver.)
ISSN 1503-8181

Doctoral theses at NTNU, 2012:6

Printed by NTNU-trykk

Blodtrykk, fedme, og jern- og feittinnhald i serum som risikofaktorar for iskjemisk hjartesjukdom

Det er gjort mange studiar på risikofaktorar for iskjemisk hjartesjukdom der blodtrykk, fedme, jern og lipidar har vist seg å spele ei rolle. Det er fleire som dør av hjartesjukdom relatert til høgt blodtrykk i eldre aldersgrupper, men den relative risikoen har vist seg å vere høgare hjå yngre. Det er uklart korleis fedme innverkar på samspelet mellom blodtrykk og død av hjartesjukdom; nokon meiner tynne har større risiko, medan andre meiner dei tjukke er mest utsette. Jern si rolle er også uklar; nokon meiner det er større risiko med for mykje, andre med for lite. Vidare er det mange ulike måtar å måle blodtrykk, fedme og lipidar på. Dei som har vist seg å gje mest informasjon i forhold til risiko for død av hjartesjukdom er gjennomsnittet av systolisk og diastolisk blodtrykk, forholdstalet mellom midje- og hoftemål justert for kroppsmasseindeks, og forholdstalet mellom total- og HDL-kolesterol.

Gjennom kopling av data frå Helseundersøkelsen i Nord-Trøndelag og Dødsårsaksregisteret har vi undersøkt samanhengen mellom blodtrykk og hjartesjukdom, og korleis alder og kroppsmasseindeks spelar inn på denne samanhengen. Vidare har vi undersøkt samanhengen mellom jernstatus i blod og risiko for død av hjartesjukdom, og korleis oppfølgingstida spelar inn på denne samanhengen. Vi har også undersøkt ulike indeksar av blodtrykk, fedme og lipidar for å finne ut kva indeks som gjev mest informasjon i forhold til risikoen for død av hjartesjukdom.

Vi fann at samanhengen mellom blodtrykk og død av hjartesjukdom var mykje sterkare hjå dei under 65 år enn dei over. Denne samanhengen var også sterkare blant dei tynne enn blant overvektige og tjukke. Vidare fann vi at lågt jerninnhald i blodet var relatert til auka risiko for død av hjartesjukdom, og at denne risikoen var sterkast tidleg i oppfølgingstida. Vi fann også at systolisk blodtrykk hjå menn og pulstrykk hjå kvinner, forholdstalet mellom midje- og hoftemål justert for kroppsmasseindeks hjå menn og kvinner, og forholdstalet mellom total- og HDL-kolesterol hjå menn og kvinner gav mest informasjon i forhold til risiko for død av hjartesjukdom.

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Acknowledgment

This work has been carried out at the Department of Public Health and General Practice, Faculty of Medicine, NTNU. I have received funding from the Research Council of Norway via the Medical Students Research Programme, NTNU. Many individuals have contributed to the thesis, and I wish to express my sincere gratitude to:

- The participants of the HUNT studies, who voluntarily have provided information about themselves and thereby enabled a multitude of studies related to health and disease.
- My supervisor, *Professor Lars Vatten*, who has been an excellent mentor in my training of epidemiology. He has mastered the art of challenging me to strive further and giving praise and encouragement to my accomplishments. I have particularly learnt the importance of high scientific standards and the joy of writing in medical research.
- My co-supervisor, *Professor Pål Romundstad*, who has been my mentor and teacher of methodology and statistical analyses. He has guided me away from potential methodological pitfalls and has always been generous towards my inquires and been dedicated to improve the quality of my work.
- My colleague, *Lars Erik Laugsand* who co-authored one of the papers in this thesis with great enthusiasm, and my latest teacher, *Imre Janszky*, from whom I have received many new ideas and learnt new methods in cardiovascular epidemiology.
- My friend, *Christian Dotterud*, with whom I started the Medical Students Research Programme at the same time and shared office with for many years. We have been interested in each other's work and have supported each other throughout the research process.
- The Medical Students Research Programme, that accepted me as a student and provided me with the opportunity to pursue the work presented here.
- My mother, father, brother and sister who always have supported and encouraged me.

Trondheim, August 2011
Bjørn Mørkedal

List of Papers

This thesis is based on the following three papers:

Paper I Mørkedal B, Romundstad PR, Vatten LJ. Mortality from ischaemic heart disease: age-specific effects of blood pressure stratified by body-mass index: the HUNT cohort study in Norway. Journal of Epidemiology and Community Health. 2011 Sep 12;65(9):814-9.

Paper II Mørkedal B, Laugsand LE, Romundstad PR, Vatten LJ. Mortality from ischaemic heart disease: sex-specific effects of transferrin saturation, serum iron, and total iron binding capacity. The HUNT study. European Journal of Cardiovascular Prevention and Rehabilitation. 2011 Feb 14;[Epub ahead of print].

Paper III Mørkedal B, Romundstad PR, Vatten LJ. Informativeness of indices of blood pressure, obesity and serum lipids in relation to ischaemic heart disease mortality: the HUNT-II study. European Journal of Epidemiology. 2011 Apr 3;(123):457-461.

1 Introduction

1.1 Blood pressure and ischaemic heart disease mortality

The first recorded attempt of measuring blood pressure in western science was done invasively in a horse by Stephen Hales in 1711 (1). However, the first non-invasive method of measuring blood pressure in humans is credited to Samuel Siegfried Karl Ritter von Basch who invented the sphygmomanometer in 1881 (2). Nikolai Sergeyevich Korotkov further improved the technique by measuring blood pressure in the brachial artery in 1905, using a stethoscope and noting tapping sounds as the cuff deflated (2). The first, large epidemiological study to evaluate the increased risk of mortality among those with high blood pressure was the Build and Blood Pressure Study in 1959 (3), with the emphasis on the importance of systolic blood pressure (4).

Over the last century, there has been much debate over which index of blood pressure is most useful. Whereas diastolic blood pressure was found to be best in most clinical trials and widely used as the best predictor of later disease, most observational studies found systolic blood pressure to be more informative (5). The emphasis shifted from diastolic blood pressure to systolic blood pressure around 1993 with the new classification described in the JNC-V report (6). Based on the largest meta-analysis thus far, the Prospective Studies Collaboration reported in 2002 that mid blood pressure (the average of systolic and diastolic blood pressure) is the most informative in relation to ischaemic heart disease mortality (7), followed by mean arterial pressure, systolic blood pressure, diastolic blood pressure and pulse pressure.

In addition to the different indices, the method of measuring blood pressure is also relevant. The office measurement should be standardized (8,9), and Ambulatory

Blood Pressure Monitoring or self-measurement should be considered if white coat hypertension is suspected (8,9). Furthermore, the apparatus itself, whether auscultatory or oscillometric, may also make a difference (10).

It has been suggested that the risk of ischaemic heart disease mortality starts to increase at around 140 mmHg (11). The Prospective Studies Collaboration have refuted this, however, stating that the risk of ischaemic heart disease mortality does not have a cut-off value, but shows a linear increase from about 115/75 mmHg (7). Still, the clinical treatment of hypertension does follow different cut-off values of blood pressure, with additional consideration of other risk factors or organ disease (9), which may often be prevalent in many individuals with high blood pressure (12).

Although the absolute rates of cardiovascular mortality are higher at old age, the proportional rates have been found to be stronger in middle age in both the western (7) and eastern (13) hemispheres. The reason for the association with age is not yet understood.

1.2 Obesity and ischaemic heart disease mortality

Obesity as a health problem has been recognized since the Hindu physician Sushruta discovered the sweet, diabetic urine in the obese around 600 BC (14). However, the medical awareness of obesity as a health problem was not fully realized until the Metropolitan Life Insurance Company published the results of a study from 1911-1935 (15), where obesity was objectively linked to excess mortality. Today, obesity is increasingly prevalent in both children and adults (16), with implications for cardiovascular health (17,18).

The concept of "ideal weight" was introduced during the turn of the 20th century (19), using a percentage of the average weight of persons of the same height. The Metropolitan Life Insurance Company introduced tables of height and weight in 1959 that became widely used (20). The body-mass index was described already in 1832 by Adolphe Quetelet (21), but was not widely used until 1972 (22). It has been the most used index of general adiposity since then, but the use of waist-to-hip ratio or waist circumference as a measure of abdominal adiposity has later been shown to be a better predictor of mortality when used together with body-mass index (23).

The association of body-mass index with all-cause mortality displays a "U-shaped" curve, with higher mortality at both ends of the distribution of body-mass index (24). Specifically, the curve for ischaemic heart disease mortality has a "J-shaped" form, with stronger associations at higher levels of body-mass index (24). However, the association of abdominal adiposity adjusted for body-mass index with mortality from ischaemic heart disease, appears to display a linear curve (23). The choice of whether to use waist-to-hip ratio or waist circumference as a measure of abdominal adiposity, adjusted for body-mass index, is still under debate. The waist-to-hip ratio benefits from having less potential of collinearity with body-mass index than waist circumference, while waist circumference is easier to measure (23).

There is an established positive association between obesity and blood pressure (25). It has also been shown that weight gain increases blood pressure (26) and that losing weight reduces blood pressure (27). However, the role of body-mass index as an effect modifier in the association of blood pressure with ischaemic heart disease mortality is unresolved. Some investigators have found that body mass index may be an effect modifier (28-34), but others have not confirmed this (35-39).

1.3 Iron status and ischaemic heart disease mortality

The health properties of iron have been known since the ancient Greeks administered iron against fatigue among soldiers, a treatment that was probably given due to blood loss and anaemia sustained at the battlefield (40). The haemoglobin protein was discovered in 1840 by Friedrich Ludwig Hünefeld (41), and its function in the blood was recognized by Sir George Gabriel Stokes in 1863 (42). In 1937, Vilém Laufberger discovered the ferritin protein (43). Transferrin was discovered later; when Arthur Schade and Leona Caroline described transferrin and its saturation with iron in human plasma in 1946 (44).

Thus, iron exists in many forms in the human body. Ferritin is the storage protein (45), and transferrin is the transport protein with two seats for free serum iron. The percentage of occupied transferrin seats to all seats is called transferrin saturation (45). Total iron-binding capacity is the number of seats for serum iron in total, or equivalent to two times transferrin. The transferrin receptor modulates iron release from transferrin (45). Haemoglobin consists of haem with iron and globin (46), and is therefore an important component of red blood cells.

In 1981, Jerome Sullivan hypothesized that the reason for increased risk of heart disease in men and post-menopausal women, compared to premenopausal women, was increased stores of iron in the blood (47). This suggestion has been supported by some studies on the association of transferrin saturation (48-53), serum iron (54), total iron binding capacity (52,55), and ferritin (56-60) with rates of cardiovascular disease. Opposite findings, showing that low levels of iron in the blood may be associated with

increased risk of cardiovascular disease, have also been reported for transferrin saturation (61), serum iron (50,53,62), and ferritin (52).

It is unclear why the associations of iron status with cardiovascular disease have differed so much between studies, but regression dilution and confounding by chronic or inflammatory diseases have been suggested to explain the discrepant findings (63).

1.4 Serum lipids and IHD mortality

The first discovery of a fat transport system in humans is credited to Robert Boyle in 1665 (64). Later, cholesterol was found in human blood by Felix Boudet in 1833 (65). During World War II, a research group from Harvard University studied human plasma intended for treating wounded soldiers and subsequently isolated low-density lipoprotein (LDL) and high-density lipoprotein (HDL) cholesterol, among other lipoproteins (66).

The positive association of triglycerides with cardiovascular disease was found in 1959 (67), and for total cholesterol in 1961 (68). From the Framingham study, the positive association of LDL cholesterol with cardiovascular disease (69), and the negative association of HDL cholesterol (70), were reported in 1966 and 1977, respectively. These results have remained consistent in later studies (71).

The most informative serum lipid index in relation to ischaemic heart disease mortality may be the ratio of total:HDL cholesterol, followed by non-HDL cholesterol, and total cholesterol (71). It appears that the use of apolipoproteins may be equivalent to using conventional lipid measurements in relation to coronary heart disease risk; thus, Apo B has a similar risk profile as non-HDL cholesterol and Apo A_1 is similar to that of

HDL cholesterol (72). However, based on the results of a case-control study, the ratio of Apo B/Apo A_1 may be more informative than the total:HDL cholesterol ratio in relation to myocardial infarction morbidity (73).

2 Objective

Our objective was to study associations of different risk factors with ischaemic heart disease mortality, and to assess potential effect modification by relevant factors. We also wanted to assess informativeness of different indices of known cardiovascular risk factors. More specifically, our aims were:

I To study the association of blood pressure with ischaemic heart disease mortality, and to assess potential effect modification by age and/or body-mass index.

II To study the association of iron status with ischaemic heart disease mortality, and to assess whether associations of iron status may vary according to follow-up time.

III To study the informativeness of different indices of blood pressure, obesity and serum lipids in relation to ischaemic heart disease mortality

3 Materials and methods

3.1 The Nord-Trøndelag Health Study (HUNT)

Norway is divided into 19 administrative regions, called counties, and Nord-Trøndelag is one of them. It is located in the central part of Norway, and is considered fairly representative for the Norwegian population. The first Nord-Trøndelag Health Study (HUNT 1) was conducted from January 1984 to February 1986. The second Nord-Trøndelag Health Study (HUNT 2) was conducted from August 1995 to June 1997. The population was approximately 127 000 in both 1984-86 and 1995-97 (74). The population of Nord-Trøndelag is stable, with a net migration out of the county of 0.1 % per year (1984-2010). It is also homogenous, with less than 3 % non-Caucasians at the time of HUNT 2 (75). Paper I in this thesis uses data from HUNT 1, whereas paper II and III use data from HUNT 2.

All inhabitants in Nord-Trøndelag aged 20 years or older were invited to participate in both HUNT 1 and HUNT 2. A total of 85 100 individuals were eligible to participate, and 74 977 (88.1 %) attended HUNT 1. In HUNT 2, a total of 92 936 individuals were eligible to participate, and 66 140 (71.2 %) attended.

In both HUNT 1 and HUNT 2, a questionnaire (questionnaire 1) was attached to the invitation letter (76). This questionnaire was to be completed prior to the clinical examination. A second questionnaire (questionnaire 2) was handed out at the clinical examination and should be completed and returned by mail in a pre-stamped envelope. Unless otherwise noted, all self-reported information that is used in this thesis comes from questionnaire 1.

Every study participant attended a clinical examination in both HUNT 1 and HUNT 2, and a non-fasting venous serum sample was drawn from each participant in HUNT 2. More comprehensive descriptions of HUNT1 and HUNT 2 are published elsewhere (75,77,78). The clinical measurements, laboratory tests, and self-reported information that were used in the studies forming this thesis are described in section 3.3.

The mortality follow-up of the HUNT cohorts was approved by the regional committee for ethics in medical research, and by the Norwegian Data Inspectorate. Participation in the HUNT study was voluntary, and each participant signed a written consent regarding the survey and subsequent follow-up, and to the use of data for research purposes.

The HUNT studies are collaboration between the HUNT Research Centre, The Faculty of Medicine, NTNU, The Norwegian Institute of Public Health, Nord-Trøndelag County Council, and the Central Norwegian Regional Health Authority.

3.2 The Cause of Death Registry in Norway

All deaths in Norway are reported by doctors who are required to complete a death certificate. Death certificates are collected by the Cause of Death Registry, for coding of information based on the International Classification of Diseases (ICD). Both the underlying and other causes of death are reported. The registry is owned by the Norwegian Institute of Public Health, but the data are collected and organised by Statistics Norway. In addition to using information from the local physicians and public health officers, Statistics Norway collects supplementary information from other sources, such as the Cancer Registry of Norway, the Medical Births Registry of

Norway, and autopsy reports. The unique 11-digit identification number of every Norwegian citizen enables linkage of data from the Cause of Death Registry with other data, such as data from the HUNT study. In paper I, the information on causes of death was complete through December 31, 2004. In paper II and paper III, it was complete through December 31, 2006.

3.3 Study variables

Information on date of death and causes of death comes from the Cause of Death Registry. All other information comes from the HUNT studies.

3.3.1 Blood pressure

In HUNT 1, blood pressure was measured after two minutes rest using calibrated mercury manometers with standard cuff size (12x24 cm) in the sitting position (77). The level of the first pulse sound (phase 1) was recorded as the systolic pressure, and the level at which the pulse disappeared (phase 5) as diastolic pressure. Measurements were repeated two minutes after the first recording, and both pressures were registered with an accuracy of 2 mmHg. We used the average of the two measurements.

In HUNT 2, systolic and diastolic blood pressure were measured using a Dinamap 845XT (Critikon) based on oscillometry, and the average of the second and third measurements was used.

In paper I, we divided systolic and diastolic blood pressure into seven categories (<119, 120-129, 130-139, 140-149, 150-159, 160-169, and ≥170 mmHg for systolic,

and <75, 75-79, 80-84, 85-89, 90-94, 95-99, and ≥100 mmHg for diastolic pressure) in the initial age-specific analyses. We also conducted analyses where effects were assessed by 20 mmHg higher systolic and by 10 mmHg higher diastolic pressure, using blood pressure as a continuous variable.

In the analyses of blood pressure stratified by body-mass index, systolic and diastolic blood pressure were divided into the four JNC-VII (8) categories (<120, 120-139, 140-159, and ≥160 mmHg for systolic, and <80, 80-89, 90-99, and ≥100 mmHg for diastolic blood pressure); these categories were chosen to avoid low numbers within combined categories of blood pressure and BMI.

In paper III, we calculated mean arterial pressure as the sum of systolic blood pressure and twice the diastolic blood pressure divided by three. Mid blood pressure was calculated as the average of systolic and diastolic blood pressure. Pulse pressure was calculated as the difference between systolic and diastolic blood pressure. We also calculated and used the product of pulse pressure and mean arterial pressure. In the analyses of informativeness, we tested the blood pressure indices as continuous variables.

3.3.2 Obesity indices

In paper I, body-mass index was calculated as body weight divided by the squared value of height (kg/m^2), and further subdivided into three categories: <25 (lean), 25–29.9 (overweight) and ≥30 (obese). In a separate analysis, we stratified body-mass index by four categories to investigate the possibility that low body-mass index (<18.5) could be attributed to prevalent but unknown disease at baseline.

In paper III, height was measured to the nearest centimetre, weight to the nearest half kilogram, and waist and hip circumferences to the nearest centimetre. From these indices we calculated waist-to-hip ratio, waist-to-height ratio and body-mass index.

3.3.3 Iron status

A non-fasting blood sample was drawn from each participant, and analysed for serum iron and serum transferrin concentrations, as described elsewhere (79). For the statistical analyses, serum iron (μmol/L), total iron binding capacity of transferrin (TIBC, twice the serum transferrin concentration, μmol/L), and transferrin saturation (100 times serum iron divided by TIBC, %) were categorised into sex-specific quartiles.

3.3.4 Serum lipids

Total serum cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides were measured on a Hitachi 911 Auto-analyzer, applying reagents from Boehringer, Mannheim. The day-to-day coefficients of variation were 1.3–1.9% for total cholesterol, 2.4% for HDL cholesterol and 0.7–1.3% for triglycerides.

In Paper III, we calculated non-HDL cholesterol as total cholesterol minus HDL cholesterol, and the total:HDL cholesterol ratio was calculated as total cholesterol divided by HDL cholesterol.

3.3.5 Age

In all the papers, we adjusted for attained age in the analyses. In paper I, we examined the joint effects of blood pressure and body-mass index with the risk of deaths from ischaemic heart disease within two groups according to attained age (<65 and ≥65 years). The cut-off at 65 years corresponded closely to the median attained age of the participants.

3.3.6 Other possible confounders

We stratified by sex in all analyses of the thesis, except the analysis of interaction with blood pressure, body-mass index and age in paper I.

Information on participants with previous myocardial infarction and stroke was self-reported and collected from questionnaire I, and these participants were excluded from the analyses in all three papers.

Information on prevalent diabetes mellitus was recorded as 'yes' or 'no' in questionnaire 1, and was adjusted for in paper I and II.

We had information on participants with current or former use of blood pressure medication, and this information was included in the online-only analyses in paper I.

In an attempt to adjust for seasonal variation in blood pressure (80), we adjusted for month of clinical examination in paper I.

From questionnaire 2 we had information on smoking habits, which were categorised as current, former, never or unknown, and adjusted for in paper I and II.

Time since last meal was recorded to the nearest hour, and was adjusted for in paper II.

3.3.7 End points

In this thesis, the primary end point was deaths caused by ischaemic heart disease (ICD 9: 410 – 414; ICD 10: I 20 – I 25) until the end of follow-up, that was 31 December 2004 in paper I, and 31 December 2006 in paper II and paper III.

3.4 Statistical analyses

All statistical analyses were conducted using Stata software, release 11.0 (paper I) or release 11.1 (paper II and III) for Windows (Stata Corp., College Station, Texas).

3.4.1 Cox regression

The main method of statistical analyses was the Cox Proportional hazards model (81), with adjustment for potential confounding factors.

In the analyses of blood pressure and ischaemic heart disease mortality (paper I), we calculated hazard ratios as the rate of death among participants within a given blood pressure category as compared with the rate of death in the reference category, i.e. 120-129 mmHg for systolic and 80-84 mmHg for diastolic blood pressure. In stratified analyses by body-mass index, the blood pressure reference categories (120-139 mmHg and 80-89 mmHg, respectively) combined with body-mass index < 25 kg/m^2 were used as the reference. These reference categories were chosen as the optimal values of blood pressure and body-mass index. We examined the joint effects of blood pressure and body-mass index in relation to deaths from ischaemic heart disease within two groups

according to attained age (<65 and ≥65 years). We used sub-analyses, according to the method of Fine and Gray (82), to test whether deaths from causes other than ischaemic heart disease could have competed with the observed associations with ischaemic heart disease mortality.

In the analyses of iron status and ischaemic heart disease mortality (paper II), we calculated hazard ratios as the rate of death among participants within a given iron status quartile as compared with the rate of death in the reference quartile, i.e. the reference value was the lowest quartile for serum iron and transferrin saturation and the highest quartile for total iron binding capacity. We first conducted analyses with adjustment for attained age, and with subsequent adjustment for other cardiovascular risk factors.

3.4.2 Proportional hazards assumption

We tested the proportional hazards assumption by comparing -ln-ln survival curves and by performing tests on Schoenfeld residuals from the Cox regression for each of the predictors in the analyses. If a variable did not satisfy the proportional hazards assumption this variable was defined as a time-dependent variable in the regression analysis whenever relevant.

3.4.3 Test for interaction

In paper I, we tested for interaction effects between blood pressure and body-mass index using likelihood ratio tests from the Cox regression analysis. To test whether the

interaction effect could differ by age we performed a three-way interaction test that included age, blood pressure, and body-mass index.

3.4.4 Time-varying analyses

In paper II, we explored whether the associations of iron status with ischaemic heart disease mortality varied during the follow-up period. This was done by calculating the scaled Schoenfeld residual from the Cox regression of a 1 standard deviation difference in transferrin saturation, serum iron and total iron binding capacity, and by constructing Lowess graphs of the hazard ratio by follow-up time (83). The purpose of this procedure was to assess whether prevalent, unknown disease related to iron status could have influenced the main results.

3.4.5 Informativeness

In the analyses of informativeness of different blood pressure, obesity and lipid indices in relation to ischaemic heart disease mortality (paper III), we used the method described by Peto et al. (84). Briefly, informativeness was calculated as the difference in twice the log-likelihood between a Cox proportional hazard model both adjusted for attained age and for blood pressure, obesity and serum lipid indices, and a model that only adjusted for attained age. The difference between these log-likelihoods follows a chi-square-distribution, and the greater the difference, the more "informative" that index is. In the analyses of waist-to-hip ratio adjusted for body-mass index, we included both variables simultaneously and compared with a model only adjusted for attained age.

In addition, we analysed the area under the receiver operating curves (AUC), pseudo R^2-values from logistic regression models, estimated hazard ratios associated with one standard deviation increase, net reclassification improvement using a 10 % cut-off and integrated discrimination improvement for all predictors adjusted for attained age. The purpose was to provide estimates that were comparable to those of other studies.

4 Results

4.1 Mortality from ischaemic heart disease: age-specific effects of blood pressure stratified by body-mass index: the HUNT cohort study in Norway

We examined whether the association of blood pressure with ischaemic heart disease (IHD) mortality was modified by age among 34 633 men and 36 749 women with no history of myocardial infarction or stroke at baseline. We also examined whether the age-specific effects of systolic and diastolic blood pressure with IHD mortality could be modified by body-mass index (BMI).

With increasing systolic blood pressure there was a monotonic increase in the mortality rate for both men and women, but there was a decline in the strength of the association with increasing age (p for interaction < 0.01). In men younger than 65 years, the relative risk of IHD mortality per 20 mmHg higher systolic pressure was 1.5 (95% CI: 1.4–1.7), and in men 65 years and older, the corresponding association was 1.2 (95% CI: 1.2–1.3). For women, the respective relative risks were also 1.5 (95% CI: 1.3–1.8) and 1.2 (95% CI: 1.2–1.3). Similar patterns were observed for diastolic blood pressure, and the strength of the association declined with increasing age (p for interaction < 0.01).

In people younger than 65 years there was a stronger risk increase with increasing systolic pressure among lean people (BMI < 25) compared to people with higher BMI (p for interaction < 0.01). Compared to the reference (systolic pressure 120–139 mmHg and BMI < 25), the relative risk associated with systolic pressure of

≥ 160 mmHg in the lean (BMI < 25) group was 5.8 (95% CI: 3.8–8.7), as compared to 2.4 (95% CI: 1.6–3.5) in overweight (BMI 25–29) and 1.6 (95% CI: 0.9–2.8) in obese (BMI ≥ 30) people.

In the older group (≥ 65 years), the results displayed a similar, but substantially weaker pattern (p for interaction = 0.06). Thus, compared to the reference, the relative risk associated with systolic blood pressure ≥ 160 mmHg was 2.1 (95% CI: 1.8–2.4) in the lean, 1.7 (95% CI: 1.5–1.9) in the overweight, and 1.8 (95% CI: 1.4–2.3) in the obese group.

The stratified analyses for diastolic pressure and BMI displayed patterns that were weaker, but similar to the results for systolic pressure. However, in the combined analyses of diastolic pressure and BMI, the interaction tests indicated different patterns than for systolic pressure. Although the interaction test was statistically significant (p for interaction, 0.042) for diastolic pressure and BMI in people younger than 65 years, there was stronger evidence for an interaction between diastolic blood pressure and BMI in relation to deaths from ischaemic heart disease (p for interaction < 0.01) in older people (≥ 65 years).

4.2 Mortality from ischaemic heart disease: sex-specific effects of transferrin saturation, serum iron and total iron binding capacity. The HUNT Study.

We assessed sex-specific associations of iron status with ischaemic heart disease (IHD) mortality, and explored whether the strength of the associations changed during follow-

up in 28 154 men and 32 644 women without known myocardial infarction or stroke at baseline.

There was a gradual increase in risk of death from IHD with decreasing level of transferrin saturation, with similar associations in men and women. For women in the lowest quartile of transferrin saturation, the hazard ratio of death from IHD was 1.4 (95% CI: 1.0–1.9) compared to women in the highest quartile (reference), and in men, the corresponding hazard ratio was 1.3 (95% CI: 1.0–1.6). The difference between men and women was not statistically significant (p for interaction = 0.87). The association of low iron status with ischaemic heart disease mortality was much stronger in the early than later stage of the follow-up period. Thus, in both genders there was a monotonic reduction in the hazard ratios with increasing follow-up time associated with one standard deviation lower transferrin saturation. In men, the hazard ratio associated with one standard deviation decrease in transferrin saturation was about 2.5 during the early stage, but decreased to about 1.5 towards the end of follow-up. There was a similar corresponding reduction among women, from about 2.0 to about 1.0. For both genders, the test of proportional hazards assumption during follow-up was $p < 0.01$ in men and $p = 0.29$ in women.

Among men, but not in women, the risk of death from IHD increased with decreasing level of serum iron. The hazard ratio among men in the lowest compared to the highest quartile was 1.5 (95% CI: 1.1–1.9), and in women, the corresponding association was 1.1 (95% CI: 0.8–1.4). The difference between men and women was not statistically significant (p for interaction = 0.40). There was a reduction in the strength of the hazard ratios related to serum iron during follow-up. In men, the hazard ratio associated with one standard deviation decrease in serum iron was about 3.0 during the

early stage of follow-up, with a decrease to about 1.8 towards the end, and a similar reduction was observed for women (from 2.0 to 1.3). The proportional hazards assumption during follow-up was $p < 0.01$ in men and $p = 0.84$ in women.

For total iron binding capacity (TIBC), risk of death from IHD increased with increasing TIBC in women, but not for men. For women in the highest compared to the lowest quartile (reference) of TIBC, the hazard ratio was 1.5 (95% CI: 1.1–2.0), and the corresponding association was 0.9 (95% CI: 0.8–1.2) for men. The gender difference was statistically significant (p for interaction < 0.01). For TIBC, there was a less consistent reduction in the hazard ratios over the follow-up period in both men and women. In men, the hazard ratio associated with one standard deviation increase in TIBC was about 2.3 in the beginning of follow-up, which decreased to about 1.5 at the end of the follow-up period, with a corresponding reduction from 2.2 to 1.4 among women. In the analysis of TIBC, the test of proportional hazards assumption during the follow-up period was $p = 0.08$ in men and $p < 0.01$ in women.

It is noteworthy that the crude analyses, only adjusting for attained age, yielded results that were essentially identical to the results obtained after multivariable adjustments.

4.3 Informativeness of indices of blood pressure, obesity and serum lipids in relation to ischaemic heart disease mortality: the HUNT-II study.

We assessed the informativeness of various indices of blood pressure, obesity and serum lipids in relation to ischaemic heart disease (IHD) mortality in 28 158 men and 32 573 women without known myocardial infarction or stroke at baseline.

The informativeness of blood pressure indices related to IHD mortality differed between men and women. In men, systolic blood pressure was the most informative index (χ^2_1=16.8), followed by mid blood pressure (χ^2_1=16.3), whereas in women, pulse pressure was the most informative index (χ^2_1=8.8). Mid blood pressure, diastolic blood pressure and mean arterial pressure were the least informative indices in women.

The informativeness of expressions of obesity and serum lipids displayed similar results for men and women. The waist-to-hip ratio (WHR) adjusted for body-mass index was the most informative index (χ^2_2=13.6 and χ^2_2=18.2 for men and women, respectively) whereas WHR was slightly less informative (χ^2_1=9.7 and χ^2_1=17.2 for men and women, respectively). However, both these indices were more informative than the waist-to-height ratio. Although the informativeness of waist circumference alone was lower than using combinations of waist and hip, or waist and height measurements, the informativeness of waist circumference alone was nonetheless higher than that of body-mass index (χ^2_1=0.1 and χ^2_1=0.2 for BMI in men and women, respectively) in these data.

In relation to serum lipids, the ratio of total cholesterol to high-density lipoprotein (HDL) cholesterol was more informative (χ^2_1=35.2 and χ^2_1=28.3 for men

and women, respectively) than any of the other lipids in relation to IHD mortality. However, serum triglycerides showed a clear difference by sex (χ^2_1=6.8 and χ^2_1=24.7 for men and women, respectively), with substantially higher informativeness among women than men.

5 Discussion

In our studies, we have used epidemiological methods to investigate mortality from ischaemic heart disease. Mainly, we have used survival analysis (Cox regression) in prospective cohorts from the HUNT studies and examined associations of non-invasive measurements (paper I and III), anthropometric measures (paper I and III), and biomarkers (paper II and III). Our principal findings were:

- The positive association of blood pressure with risk of death from ischaemic heart disease was modified by body-mass index in middle age, and was much stronger in lean than in overweight and obese people.
- Low iron status was associated with increased risk of death from ischaemic heart disease, and the associations of iron status were much stronger in the early stage of follow-up than in later stages of follow-up.
- Among the indices of blood pressure, obesity and serum lipids that we assessed, the most informative indices related to risk of death from ischaemic heart disease were systolic blood pressure in men and pulse pressure in women, waist-to-hip ratio adjusted for body-mass index both in men and women, and the ratio of total cholesterol to high-density lipoprotein (HDL) cholesterol for both men and women. Serum triglycerides also yielded high informativeness in women, but not in men.

5.1 Precision

Could chance be a plausible explanation for our results? Chance is often equated to random error, and precision is the opposite of random error. A major determinant of precision in cohort studies is the study size (85); thus, a large study will typically yield more precise estimates than a smaller study. We assessed precision using statistical tests, and indicated the precision of the estimates by p-values or confidence intervals. The p-value represents the probability that the outcome of the test statistic is at least as extreme as the one that was actually observed, given that the null hypothesis is true. On the other hand, a 95 % confidence interval represents the interval in which we are 95 % certain that the true point estimate is, given the absence of bias and confounding. Usually, it is preferable to use the confidence interval, since it expresses information on both precision and the strength of the association.

In our studies we had approximately 30 000 participants of each sex. This gives us precise results with narrow confidence intervals in the main analyses. In sub-group analyses, however, the precision will be reduced. For example, in the analyses of blood pressure and body-mass index in paper I, the category “obese” had about 7 000 people, which yielded wider confidence intervals. In paper II, on the other hand, the participants were divided evenly into sex-specific quartiles of transferrin saturation, serum iron and total iron binding capacity. This resulted in more cases for each stratum, making the confidence intervals narrower.

Although the study population is large, it is a sample of an even larger population, both geographically and taking into account different time periods. Also, the proportion of participants who end up as cases is relevant in this regard. The high

attendance to the HUNT studies reduces the sampling variation, therefore reducing the likelihood that chance plays an important role in explaining our results. The large number of deaths from ischaemic heart disease adds to this, making chance an unlikely explanation for our findings.

5.2 Validity

Is the observed association between exposure and disease true? The objective of an epidemiologic study is to obtain estimates that are both valid and generalizable to relevant target populations (86). Validity is the opposite of systematic error, or bias. However, that a study provides valid results does not necessarily mean that the results represent causal associations between a given factor and a given outcome. Causality is a matter of inference, but high validity is necessary for making causal inferences.

Validity is also classified as internal or external. In the assessment of internal validity of a certain study, one needs to evaluate the potential for confounding, selection bias and information bias. External validity, however, refers to whether the results of the study can be generalized to other populations or groups of people.

5.2.1 Confounding

The association of an exposure with an outcome may be confounded by other factors (86). Thus, the exposure may be associated with a third factor, which also has an effect on the outcome. However, a factor that is on the causal pathway from exposure to outcome should not be treated as a confounder. Therefore, the assessment of potential

confounding should be done with care, and preferably on the basis of previous knowledge and using careful reasoning, rather than basing the assessment of confounding on p-values linked to the co-variates entered into the analyses.

There are usually two ways of dealing with confounders; one is to use stratification and the other is to adjust for potential confounding in regression analyses. In our papers, we have used both strategies. We have mostly done separate analyses in men and women, and thus stratified by sex. We have also used regression analysis to adjust for sex in the analyses of blood pressure, body-mass index and age in paper I. In relation to age, we adjusted for attained age in all analyses and stratified by the median attained age in paper I.

Furthermore, we have used regression analysis to adjust for diabetes mellitus (paper I and II), smoking status (paper I and II), body-mass index (paper I), systolic blood pressure (paper II), total:HDL cholesterol ratio (paper II), waist-to-hip ratio (paper II), and time since last meal (paper II).

Factors we had information on, but were not used or considered as confounders in the main analyses, included the use of blood pressure medication (confounding by indication), alcohol use (too many missing), and exercise frequency (too many missing).

5.2.2 Selection bias

Whenever the relation between exposure and disease in a population study differs between participants and non-participants in the population, a selection bias may explain the results (86). Unlike confounders, it is not possible to adjust for the effects of

a bias; bias needs to be dealt with in the planning of a study. However, it is possible to investigate whether those who participate differ from those who do not.

In our studies, the population of an entire county older than 20 years of age was eligible to participate. If attendance is high, any effect of selection bias will be reduced. However, if the attendance is not that high, the possibility for selective non-participation could disturb the estimated results. In HUNT 1, 11.9 % of those who were invited, did not participate, and in HUNT 2, the corresponding proportion was 28.8 % (75). A non-responder study was conducted after HUNT 2, and the major reason for not participating was lack of time in younger people and immobilising disease in the elderly (87). The non-responders differed in that they reported a higher level of smoking, which is an important factor that could have influenced our results.

We made certain restrictions of participants in the analyses, and possibly, some of these restrictions could have introduced a selection bias. In order to investigate a healthy population, we excluded participants with a history of heart disease or stroke at baseline. We performed sensitivity analyses where these participants were included in the analyses, after which the associations became weaker (data not shown). However, the directions and patterns of the point estimates were essentially the same. Thus, it appears that the sub-group with prevalent cardiovascular disease has random characteristics that would attenuate the estimates of effect towards the null value.

5.2.3 Information bias

Information bias is a systematic inaccuracy or error in the collected information. It is called misclassification bias if the information is on a categorical scale (88). This bias

can be subdivided into differential and non-differential misclassification. A differential misclassification is when the errors of one variable depend on the actual values of other variables. As a result, the point estimates could go in either direction. A non-differential misclassification is unrelated to the actual values of other variables, and will usually attenuate the estimated associations towards the null value.

In our studies, many variables may be prone to information bias. The use of standard blood pressure cuffs in paper I could lead to overestimation of blood pressure in overweight and obese people, or among people with large upper arms. This source of differential misclassification could cause people with large upper arms to be incorrectly classified with hypertension. Thus, the estimated association of blood pressure with IHD mortality may be weaker than the true association in these people.

There may be information bias in self-reported data, such as time since last meal, smoking status and prevalence of diabetes mellitus. There may also be misclassification in causes of death. Furthermore, the measured values of weight, height, waist and hip circumference, serum iron, total iron-binding capacity, total and HDL cholesterol and triglycerides may suffer from random measurement error which may attenuate the estimates towards the null value.

To avoid differential misclassification, we excluded participants with previous heart disease or stroke from all analyses. In these individuals, the medical attention or treatment may have altered both self-reported and measured data.

Prospective cohort studies are less prone to information bias than other studies (e.g. case-control studies). However, during long follow-up periods, measured values at baseline may change. For instance, people with detected hypertension at HUNT 1 were referred to their primary physician (78). These may have started anti-hypertensive

medication that could have resulted in underestimation of the risk of high blood pressure.

Blood pressure values may vary over time, and compared to baseline levels, blood pressure during follow-up is not likely to be constant. Therefore, the lack of longitudinal measurements during the follow-up period is a weakness of our study. Most likely, this weakness has caused a "dilution" effect, meaning that the estimated associations are underestimates of the true associations related to blood pressure. It has been suggested that this regression dilution bias (89) could be corrected by using repeated measurements in a reasonably representative sample of participants in the study (7).

5.2.4 Generalizability

Where and to whom do our results apply? Some believe that a representative study should sample the study population to closely resemble the population at large, while others believe a homogenous study population is necessary to identify more valid causal relations (86). In our studies we have both a representative sample by age and sex, and a homogenous sample by ethnicity and geographical location. In other words, our sample is representative for the Nord-Trøndelag adult population, and may therefore allow the assessment of risk factors in relation to ischaemic heart disease mortality. The external validity of our results may be limited if these associations differ between Nord-Trøndelag and other populations.

5.3 Appraisal of the principal findings

5.3.1 The age-specific associations of blood pressure and body-mass index with ischaemic heart disease (IHD) mortality

We found a relatively stronger, positive association of blood pressure with IHD mortality among younger, lean individuals, thus giving evidence for effect modification by age and by body-mass index (BMI).

The effect modification by age, where the association of blood pressure with IHD mortality is stronger at younger ages, is consistent with previous findings (7). The effect modification by BMI, where the association of blood pressure with IHD mortality is stronger in lean than overweight people, is consistent with some studies (28-34), but not with others (35-39). To our knowledge, the combined effect modification of age and BMI has only been assessed and reported in one previous study (34), and in that study, there was no significant difference between age groups. However, it was reported that low BMI was more strongly associated with increased risk of coronary heart disease in men both at young and older ages, but in women, there were no similar findings.

Between studies, it has not been consistently shown that lean hypertensive people are at greater risk of cardiovascular disease than obese hypertensive people. Lean hypertensive people tend to be at greater risk in studies where the main outcome is mortality (28-34), whereas obese hypertensive people appear to be at greater risk in studies where the main outcome is morbidity (35-39). We only studied mortality as an outcome, and our findings correspond to those of previous studies using mortality. Other possibilities for the different results between studies include heterogeneity between populations and different analytical strategies (33,36). It is also possible that

the relation of blood pressure and BMI with cardiovascular disease may truly differ between populations.

Hypertension in lean and young people may be a sign of a relatively more life threatening type of hypertension that may cause premature death. A relatively strong association of hypertension with death from IHD in lean people may reflect genetic susceptibility (90,91), increased peripheral resistance (92) or underlying biological mechanisms that may differ from other mechanisms of hypertension (93,94). In young people, the relatively stronger association of hypertension may further reflect a more severe type of hypertension than is typically observed among elderly people. As a consequence, the relatively weaker association of hypertension with deaths from IHD at older ages may reflect selective survival of individuals who are less vulnerable to high blood pressure.

Smokers usually have lower BMI than non-smokers (95), and this could have influenced our results. However, after adjustment for smoking, we found that the results remained essentially the same. This is also in accordance with the findings of others (28,30,33).

As mentioned in section 5.2.3, the use of standard cuff size in people with wide arm circumference could lead to incorrectly elevated blood pressures (96). However, previous studies that were able to account for this possibility, did not provide evidence that the results could be attributed to inappropriate cuff size (29,30,33).

The age-related differences in relative risks of death from IHD could be attributed to cohort effects. Thus, different birth cohorts could have experienced different exposures, especially during childhood and adolescence. It is possible that such potential differences could be relevant for long term effects of blood pressure on

cardiovascular health (97,98). It also seems possible that secular trends in nutrition from a relatively sparse diet at a young age, to a richer diet later in life, could be of importance for the interaction between blood pressure and body-mass index (99).

5.3.2 The sex-specific association of iron status with ischaemic heart disease mortality (IHD) mortality

We found a linear, negative association of iron status with IHD mortality, but the associations were stronger during the early phase of follow-up than in the later phase. This may suggest that low iron status could be a late sign of IHD pathology or that unknown prevalent disease at baseline could influence the associations.

The association of low transferrin saturation with higher cardiovascular mortality has previously been shown in one study (61), but not specifically for IHD mortality as reported in paper II. The corresponding association of low serum iron, as we found among men, has been reported by others (62,100). In two other studies, low serum iron was associated with higher risk in women, but not in men (50,53). It has also been suggested that high levels of serum iron, in both men and women, may be associated with the risk of dying from myocardial infarction (54). Our finding that high levels of total iron binding capacity (TIBC) may be associated with increased risk of death from IHD differs from the results of others; previously, either opposite (52,55) or no associations (50-52,58) have been reported.

Low iron status could be a late sign in the pathogenesis of IHD, and an inflammatory response has, for example, been suggested as one possible explanation (101), and reverse causality may be another possibility. Thus, unknown but underlying

disease could influence iron status and the association with IHD mortality. For example, unknown cardiovascular disease, cancer (102), chronic infections (102), rheumatic diseases (103), or chronic kidney disease (104) could cause low iron status and thereby influence the association with IHD mortality. Since we found stronger associations during the early phase of follow-up, both possibilities may be supported by our findings.

Our results may also reflect the association of iron with haem (105), since anaemia could increase the risk of cardiovascular morbidity and mortality (106). Specifically, it has been shown that anaemia is associated with increased risk of cardiovascular events in patients with hypertension (107), atrial fibrillation (108), coronary heart disease (109-112), and chronic heart failure (113,114).

5.3.3 The sex-specific informativeness of blood pressure, obesity and serum lipid indices with ischaemic heart disease (IHD) mortality

Our study of informativeness suggests that different indices of known risk factors may differ in their usefulness in relation to estimates of IHD mortality. In relation to blood pressure, we found that systolic blood pressure in men and pulse pressure in women may be more informative than other indices. In relation to obesity, waist-to-hip ratio adjusted for body-mass index (BMI) was more informative than other indices of body mass in both sexes. For serum lipids, we found that the ratio of total cholesterol to high-density lipoprotein (HDL) cholesterol was more informative than other indices for both men and women.

Our results related to obesity and serum lipids are consistent with findings of previous studies (23,71). It has been suggested that waist-to-height ratio may be the

most informative obesity index (115), but we found it to be less informative than waist-to-hip ratio adjusted for BMI. Our results related to blood pressure, suggesting that systolic blood pressure is the most informative in men and pulse pressure in women, deviate from the findings of others. Thus, mid blood pressure has been suggested to be the most informative index (7), and we also found that index to be relatively informative in men, but not in women. Also, others have found that pulse pressure may be informative both in middle and old age, with no clear sex difference, which corresponds to our findings (116).

The reason for a sex difference in blood pressure indices is not clear, but others have suggested that pulse pressure may be particularly informative in old age (116-118). Since there is a substantial age difference by sex in being diagnosed with ischaemic heart disease (119), the age difference may be important for the difference in informativeness related to pulse pressure. It has also been suggested that men and women have different patterns of arterial aging (120), and this could be of important for our findings.

We did not find mid blood pressure to be as informative as reported in other studies (7). This could be due to the low informativeness of diastolic blood pressure in our study, since diastolic blood pressure is a component of mid blood pressure.

The waist-to-hip ratio adjusted for BMI combines information on abdominal obesity and general obesity, which may explain the high informativeness related to this obesity index. This combined index has also been suggested to improve informativeness in relation to all-cause mortality, especially in people with low BMI (23).

The ratio of total cholesterol and high-density lipoprotein (HDL) cholesterol was the most informative expression of serum lipids related to IHD mortality in both men

and women. The reason for the high informativeness may be the combination of two relatively informative indices into one.

5.4 Conclusions and implications

- We found that the association of blood pressure with ischaemic heart disease mortality was modified by age and body-mass index. Thus, lean and relatively young individuals with hypertension were at increased risk of death from ischaemic heart disease. These findings are in accordance with similar studies of cardiovascular disease mortality, but not with studies of morbidity. This discrepancy is worthy of further investigations, and hopefully, other large, prospective studies that can use both morbidity and mortality end-points could elucidate this further. Also, the relative differences in the association of blood pressure between lean and overweight/obese people could imply that the pathophysiology between these groups may differ both in relation to hypertension and ischaemic heart disease. To investigate this difference further, it might be useful to apply the approaches of some relatively small cross-sectional studies in larger prospective studies, in particularly, by including metabolic, neuroendocrine and cardiovascular characteristics in the analyses (121), and by evaluating sympathetic nervous system activity (93,94). Furthermore, our results may support that young and lean hypertensive people could benefit from closer monitoring by their physicians.
- Our results indicate that low iron status is associated with increased risk of death from ischaemic heart disease. Specifically, we found this for transferrin

saturation, serum iron and total iron binding capacity. This is in accordance with some studies, but not with others. It is interesting to note that this association was much stronger during the early phase of the follow-up period. This could imply that low iron status is a late marker in the pathogenesis of ischaemic heart disease or alternatively, that unknown but underlying disease could have influenced iron status. These findings warrant confirmation by other studies, and it could be useful to investigate this issue in studies with information on other iron markers and related biomarkers (e.g. ferritin, transferrin receptor, haemoglobin, and C-reactive protein). Furthermore, our results may support that low iron status should stimulate physicians to search for underlying mechanisms.

- There are many indices of blood pressure, obesity and serum lipids, and our results suggest that systolic blood pressure in men, pulse pressure in women, waist to hip ratio adjusted for body-mass index in men and women, and total:HDL cholesterol ratio in men and women are the most informative in relation to assessing ischaemic heart disease mortality. Other studies support our findings for obesity and serum lipids, but for blood pressure, the mid blood pressure was the most informative in a large meta-analysis (7). The most informative index should be considered by researchers when choosing which index to study or to adjust for. Also, the approach of determining informativeness could be investigated for other indices and other diseases. Furthermore, our results suggest that it may be useful for physicians to use more informative measurements in the clinical management of patients.

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Paper I

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Paper II

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Paper III

Eur J Epidemiol (2011) 26:457–461
DOI 10.1007/s10654-011-9572-7

CARDIOVASCULAR DISEASE

Informativeness of indices of blood pressure, obesity and serum lipids in relation to ischaemic heart disease mortality: the HUNT-II study

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Received: 23 January 2011 / Accepted: 23 March 2011 / Published online: 3 April 2011

Abstract The informativeness of blood pressure, obesity and serum lipids associated with cardiovascular events may depend on how the indices are expressed, and mid blood pressure, waist-to-hip ratio adjusted for body-mass index (BMI) and the ratio of total to HDL cholesterol may be more informative than other expressions. Our aim was to study the informativeness of indices of blood pressure, obesity and serum lipids associated with ischaemic heart disease mortality in a large, homogeneous population. Blood pressure, weight, height, waist and hip circumference, total and HDL cholesterol, and triglycerides were measured at baseline (1995–1997) in 28,158 men and 32,573 women. Information on deaths from ischaemic heart disease (IHD) was obtained from the Causes of Death Registry in Norway from baseline until the end of 2007. Informativeness was analysed using the difference in twice the log-likelihood of a Cox model with and without each index. During 11 years of follow-up, 597 men and 418 women had died from IHD. Systolic blood pressure in men and pulse pressure in women were the most informative predictors of blood pressure, and waist-to-hip ratio adjusted for BMI was the most informative expression of obesity in both men and women. Among serum lipids, the most informative predictor was the ratio of total cholesterol to HDL cholesterol. Using more informative expressions of conventional risk factors for ischemic heart disease may improve both the validity and precision of estimates of risk, and may be useful both clinically and for preventive purposes.

Keywords Ischaemic heart disease · Blood pressure · Obesity · Lipids · Risk · Informativeness

Introduction

Blood pressure, obesity and serum lipids are associated with the risk of ischaemic heart disease, but the preferred expression of these indices appears to change over time, maybe reflecting their evolving conceived importance [1–3]. The informativeness of an index is meant to capture how well it predicts the outcome. In relation to blood pressure, for example, mid blood pressure has been suggested to be the most informative index in relation to ischaemic heart disease mortality [4]. Similarly, among obesity indices, the ratio of waist-to-hip circumference, adjusted for body-mass index, may be the most informative predictor [5], but the ratio of waist-to-height has also been suggested [6]. Among serum lipids, the ratio of total cholesterol to HDL cholesterol may be the most informative predictor for ischaemic heart disease mortality [7].

The ranking of indicators is based on the results of meta-analyses of studies consisting of heterogeneous populations [4, 5, 7]. However, we have assessed informativeness of various indices of blood pressure, obesity and serum lipids in relation to ischaemic heart disease mortality using data from a large, homogeneous cohort, with separate analyses of men and women.

Electronic supplementary material The online version of this article (doi:10.1007/s10654-011-9572-7) contains supplementary material, which is available to authorized users.

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Methods

Study population

The adult population 20 years of age and older in Nord Trøndelag County in Norway was invited to participate in a health survey (the HUNT II Study) from August 1995 to June 1997. Briefly, 92,936 individuals were eligible to participate in the study, and 64,939 (69.9%) accepted the invitation, filled in a questionnaire that was included with the invitation letter, and attended a clinical examination conducted by trained nurses. Among other factors the examination included standardised measurements of blood pressure, body weight, body height, waist and hip circumference, and a non-fasting blood sample with subsequent measurements of total serum cholesterol, HDL cholesterol and triglycerides. The study has been described in more detail elsewhere [8].

For the present study, we excluded 769 participants with missing information on waist or hip circumference and 132 participants with missing information on total cholesterol, HDL cholesterol or triglycerides, as well as 63 participants with missing information on blood pressure and 194 participants with missing information on weight and height. We also excluded 3,050 participants with a history of myocardial infarction or stroke at baseline. Thus, 60,731 participants (28,158 men and 32,573 women) were included in the main sex-specific analyses of informativeness of predictors of ischaemic heart disease mortality.

The mortality follow-up of the HUNT cohort was approved by the regional committee for ethics in medical research, by the national Directorate of Health, and by the Norwegian Data Inspectorate.

Blood pressure, obesity and lipid indices

Systolic and diastolic blood pressure were measured using a Dinamap 845XT (Critikon) based on oscillometry, and the average of the second and third measurements was used. From these indices we calculated mean arterial pressure as the sum of systolic blood pressure and twice the diastolic blood pressure divided by three. Mid blood pressure was calculated as the average of systolic and diastolic blood pressure. Pulse pressure was calculated as the difference between systolic and diastolic blood pressure. Pulse pressure $\times$ Mean arterial pressure is the product of these indices.

At the clinical examination, height was measured to the nearest centimetre; weight to the nearest half kilogram; and waist and hip circumferences to the nearest centimetre. From these indices we further calculated waist-to-hip ratio, waist-to-height ratio and body-mass index (kg/m^2).

A blood sample (non-fasting) was drawn from all the participants, centrifuged at the study site, and sent in a cooler to the laboratory. Total serum cholesterol, HDL cholesterol, and triglycerides were measured on a Hitachi 911 Auto-analyzer, applying reagents from Boehringer Mannheim. The day-to-day coefficients of variation were 1.3–1.9% for total cholesterol, 2.4% for HDL cholesterol and 0.7–1.3% for triglycerides.

We calculated non-HDL cholesterol as total cholesterol minus HDL cholesterol, and the total:HDL cholesterol ratio was calculated as total cholesterol divided by HDL cholesterol.

End points

The mandatory reporting of deaths by physicians and public health officers to the national Cause of Death Registry in Norway constitutes the basis for the coding of underlying causes of death. Mortality follow-up to the Cause of Death Registry is virtually complete. In this study, the primary end point was deaths caused by ischaemic heart disease (ICD 9: 410–414; ICD 10: I 20–I 25).

Statistical analyses

Follow-up time (person-time) was calculated from the baseline date of participating in the HUNT Study until date of death, or until the end of follow-up, 31 December 2006, whichever occurred first.

We analysed informativeness of different blood pressure, obesity and lipid indices in relation to ischaemic heart disease mortality, according to the method described by Peto et al. [9]. Briefly, informativeness was calculated as the difference in twice the log-likelihood between a Cox proportional hazard model both adjusted for attained age and each respective blood pressure, obesity or serum lipid index, and a model that only adjusted for attained age. The difference between these log-likelihoods follows a chi-square-distribution, and the greater the difference, the more "informative" that index is. In the analyses of waist-to-hip ratio adjusted for BMI, we included both variables simultaneously and compared with a model only adjusted for attained age.

We tested the proportional hazards assumption by comparing -ln-ln survival curves and by performing tests on Schoenfeld residuals for each of the predictors of the study. If a predictor did not satisfy the proportional hazards assumption this was defined as time-dependent whenever relevant.

In addition, we analysed the area under the receiver operating curves (AUC), pseudo R^2 values from logistic regression models, estimated hazard ratios associated with one standard deviation increase, net reclassification

improvement using a 10% cut-off and integrated discrimination improvement for all predictors adjusted for attained age, and present the results as a web-only appendix for the purpose of comparison with other studies.

All statistical analyses were conducted using Stata software, release 11.1 for Windows (Stata Corp., College Station, Texas).

Results

During 11.4 years of follow-up (over 600,000 person-years) 1,015 men and women had died from ischaemic heart disease (Table 1), yielding a mortality rate of 166.8 per 100,000 person-years.

The informativeness of blood pressure indices related to ischaemic heart disease (IHD) mortality differed between men and women (Table 2). In men systolic blood pressure was the most informative index ($\chi^2_1 = 16.8$), followed by mid blood pressure ($\chi^2_1 = 16.3$), whereas in women, pulse pressure was the most informative index ($\chi^2_1 = 8.8$). Mid blood pressure, diastolic blood pressure and mean arterial pressure were the least informative indices in women.

The informativeness of expressions of obesity and serum lipids displayed similar results for men and women (Tables 3 and 4). The waist-to-hip ratio (WHR) adjusted for body-mass index was the most informative index ($\chi^2_2 = 13.6$ and $\chi^2_2 = 18.2$ for men and women, respectively) whereas WHR was slightly less informative ($\chi^2_1 = 9.7$ and $\chi^2_1 = 17.2$ for men and women, respectively) (Table 3). However, both these indices were more informative than the waist-to-height ratio. Although the informativeness of waist circumference alone was lower than using combinations of waist and hip, or waist and height measurements, the informativeness of waist circumference alone was nonetheless higher than that of body-mass index ($\chi^2_1 = 0.1$ and $\chi^2_1 = 0.2$ for BMI in men and women, respectively) in these data.

In relation to serum lipids (Table 4), the ratio of total cholesterol to HDL cholesterol was more informative ($\chi^2_1 = 35.2$ and $\chi^2_1 = 28.3$ for men and women, respectively) than any of the other lipids in relation to IHD mortality. However, serum triglycerides showed a clear difference by sex ($\chi^2_1 = 6.8$ and $\chi^2_1 = 24.7$ for men and women, respectively), with substantially higher informativeness among women than men.

Table 1 Characteristics of the study population ($n = 60{,}731$)

	Men	Women
Participants, *n* (%)	28,158 (46)	32,573 (54)
Age at baseline, years	49 (16)	50 (17)
Systolic blood pressure, mmHg	140 (19)	135 (23)
Diastolic blood pressure, mmHg	82 (12)	79 (12)
Mean arterial pressure, mmHg	101 (13)	98 (15)
Mid blood pressure, mmHg	111 (14)	107 (17)
Pulse pressure	58 (13)	57 (16)
Waist-to-hip ratio	0.90 (0.06)	0.80 (0.06)
Body-mass index, kg/m^2	26 (3)	26 (5)
Waist circumference, cm	92 (9)	81 (11)
Hip circumference, cm	102 (6)	102 (9)
Weight, kg	83 (12)	70 (12)
Total:HDL-cholesterol ratio, mmol/l	5.0 (1.7)	4.2 (1.5)
Non-HDL-cholesterol, mmol/l	4.6 (1.2)	4.4 (1.4)
Total cholesterol, mmol/l	5.8 (1.2)	5.9 (1.3)
HDL-cholesterol, mmol/l	1.2 (0.3)	1.5 (0.4)
Triglycerides, mmol/l	2.0 (1.2)	1.5 (1.0)
Deaths from ischaemic heart disease, *n*	597	418

Continuous variables are expressed as mean (SD)

Discussion

In this prospective study of 60,731 men and women who were free from known cardiovascular disease at baseline, we assessed the informativeness of different indices of blood pressure, obesity and serum lipids in relation to IHD mortality. The most informative indices were systolic blood pressure in men and pulse pressure in women, waist-to-hip ratio adjusted for body-mass index both in men and women, and the ratio of total cholesterol to HDL cholesterol for both men and women. However, serum triglycerides also yielded high informativeness in women, but not in men.

There is evidence in the literature to support our results related to indices of obesity and serum lipids [5, 7, 10], but we were unable to find results related to blood pressure that corresponded to ours. Although it has been suggested that the waist-to-height ratio may be an informative obesity index [6], we found it to be less informative than the waist-to-hip ratio adjusted for BMI. Previously, it has been suggested that mid blood pressure may be the most informative blood pressure index [4], but in our data, this index showed relatively high informativeness in men, but not in women. We found that systolic pressure may be most informative in men, and pulse pressure in women. Previously, it has been suggested that pulse pressure may be particularly informative both in middle and old age, but with no clear difference between men and women [11].

This cohort consists of the majority of adults in a stable, homogeneous population in Norway. The population is well suited for follow-up studies, partly because of excellent national end-point registries, and partly because of the unique identification number allocated to each citizen. The prospective study design reduces the possibility of biased estimates of effect, and the large study size makes

Table 2 Informativeness of different blood pressure indices related to ischaemic heart disease mortality, by sex

Men	χ^2	*P*	Women	χ^2	*P*
Systolic blood pressure	16.8	<0.001	Pulse pressure	8.8	0.003
Mid blood pressure	16.3	<0.001	Pulse pressure × mean arterial pressure	4.1	0.043
Pulse pressure × mean arterial pressure	16.2	<0.001	Systolic blood pressure	3.0	0.086
Mean arterial pressure	15.0	<0.001	Mid blood pressure	0.7	0.393
Pulse pressure	10.3	0.001	Diastolic blood pressure	0.6	0.448
Diastolic blood pressure	9.6	0.002	Mean arterial pressure	0.2	0.687

Adjusted for attained age. Sex-specific sorted by most informative as defined by high value of χ^2

Table 3 Informativeness of different obesity indices related to ischaemic heart disease mortality, by sex

Men	χ^2	*P*	Women	χ^2	*P*
Waist-to-hip ratio adjusted for BMI	15.4	<0.001	Waist-to-hip ratio adjusted for BMI	20.3	<0.001
Waist-to-hip ratio	12.0	<0.001	Waist-to-hip ratio	19.3	<0.001
Waist-to-height ratio	11.3	<0.001	Waist-to-height ratio	8.0	0.005
Weight	2.3	0.128	Waist circumference	5.7	0.017
Waist circumference	4.0	0.044	BMI	0.2	0.671
Hip circumference	0.3	0.610	Weight	0.1	0.763
BMI	<0.1	0.808	Hip circumference	<0.1	0.873

Adjusted for attained age. Sex-specific sorted by most informative as defined by high value of χ^2

Table 4 Informativeness of different serum lipid indices related to ischaemic heart disease mortality, by sex

Men	χ^2	*P*	Women	χ^2	*P*
Total:HDL-cholesterol ratio	35.2	<0.001	Total:HDL-cholesterol ratio	28.3	<0.001
Non-HDL cholesterol	29.8	<0.001	Triglycerides	24.7	<0.001
Total cholesterol	19.1	<0.001	HDL cholesterol	17.6	<0.001
HDL cholesterol	14.5	<0.001	Non-HDL cholesterol	14.8	<0.001
Triglycerides	6.8	0.009	Total cholesterol	6.6	0.010

Adjusted for attained age. Sex-specific sorted by most informative as defined by high value of χ^2

chance an unlikely explanation of the main findings. Separate analysis of men and women is another strength of this study. In the analyses, we used the difference in twice the log-likelihood from a Cox regression model, instead of using AUC and pseudo R^2 from a logistic regression model. Our approach has the benefit of considering person-time and censoring and is likely to yield more valid estimates than the other methods [9].

Some limitations should, however, be considered. It has been suggested that the informativeness of blood pressure indices changes with age [12], and despite the large sample size, we did not have sufficient statistical power to study age-specific informativeness of these indices in detail. Furthermore, we did not have information available on non-fatal myocardial infarction or stroke and therefore, censoring for these events could not be done. Another limitation is the single measurements at one time point and no repeated measurements. Thus, we did not have data available that allowed adjustment for possible regression dilution effects [4].

The reason for the difference in informativeness of blood pressure between men and women is uncertain, but others have suggested that pulse pressure may be particularly informative in old age [11–13] and women are usually diagnosed with ischaemic heart disease at an older age than men [14]. It has also been suggested that men and women have different patterns of arterial aging [15], and this could be of important for our findings.

The relatively low informativeness of mid blood pressure, that others have suggested to be highly informative in relation to IHD mortality [4], could be due to the lack of informativeness of diastolic blood pressure in our study, since diastolic blood pressure is a component of mid blood pressure.

The waist-to-hip ratio adjusted for BMI combines information on abdominal obesity and general obesity,

which may explain the high informativeness related to this obesity index. This combined index has also been suggested to improve informativeness in relation to all-cause mortality, especially in people with low BMI [5].

Similarly, by combining total cholesterol and HDL cholesterol to a ratio, this index was the most informative expression of serum lipids related to IHD mortality in both men and women. The superiority of combining the two measurements into a single index may simply be due to the individual strength of both total and HDL cholesterol. Serum triglycerides were especially informative in women, but not in men. The reason for the sex difference related to triglycerides is uncertain, but an imbalance between calorie intake, content and composition of diet, and a sedentary life style has been suggested [16].

Conclusions

The use of the most informative indices may improve the prediction of ischaemic heart disease mortality, and may be useful in choosing a preferred index in cardiovascular research. Also, our findings suggest that different indices of blood pressure in men and women may be useful, and that using the waist-to-hip ratio adjusted or stratified by BMI could also improve estimates of risk.

Acknowledgments Nord-Trøndelag Health Study (The HUNT Study) is a collaboration between the Faculty of Medicine, Norwegian University of Science and Technology, Trondheim, the Nord-Trøndelag County Council and The Norwegian Institute of Public Health. This work was supported by the Norwegian University of Science and Technology; and by the Norwegian Research Council.

Conflict of interest None.

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Appendix A Crude analyses, adjusting only for attained age

Table I. Systolic Blood Pressure and Relative Risk of Ischaemic Heart Disease Mortality, by Sex and Attained age, Nord-Trøndelag, Norway, 1984-2005.

		< 65 years (n = 35 605)			**≥ 65 years** (n = 35 777)		
SBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI
Men							
<119	4 298	32	0.9	0.6, 1.4	90	1.0	0.8, 1.3
120-129	7 670	61	1.0	Reference	182	1.0	Reference
130-139	8 074	65	1.0	0.7, 1.4	296	1.1	0.9, 1.4
140-149	5 947	70	1.6	1.1, 2.2	358	1.2	1.0, 1.5
150-159	3 518	66	2.7	1.9, 3.9	322	1.4	1.1, 1.6
160-169	2 145	26	2.1	1.3, 3.4	293	1.5	1.3, 1.9
≥170	2 981	54	4.7	3.3, 6.9	614	2.1	1.8, 2.5
Per 20 mmHg		374	1.6	1.5, 1.8	2155	1.2	1.2, 1.3
Women							
<119	10 487	12	0.8	0.4, 1.8	30	0.8	0.5, 1.2
120-129	7 350	12	1.0	Reference	75	1.0	Reference
130-139	5 347	17	1.9	0.9, 3.9	147	1.3	1.0, 1.7
140-149	3 976	19	3.1	1.5, 6.5	218	1.6	1.2, 2.1
150-159	2 966	12	3.1	1.4, 6.9	213	1.6	1.2, 2.1
160-169	2 279	6	2.7	1.0, 7.2	256	2.1	1.6, 2.7
≥170	4 344	20	7.4	3.6, 15.6	682	2.6	2.1, 3.3
Per 20 mmHg		98	1.6	1.4, 1.9	1621	1.3	1.2, 1.3

CI, confidence interval; RR, relative risk adjusted for attained age (continuous); SBP, systolic blood pressure in mmHg.

Table II. Diastolic Blood Pressure and Relative Risk of Ischaemic Heart Disease Mortality, by Sex and Attained age, Nord-Trøndelag, Norway, 1984-2005.

		< 65 years (n = 35 605)			**≥ 65 years** (n = 35 777)		
DBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI
Men							
<75	4 854	22	0.7	0.4, 1.2	163	1.0	0.8, 1.2
75-79	4 691	30	0.9	0.6, 1.4	193	1.0	0.8, 1.2
80-84	6 846	56	1.0	Reference	318	1.0	Reference
85-89	5 962	65	1.3	0.9, 1.8	337	1.0	0.9, 1.2
90-94	5 136	64	1.4	1.0, 2.0	365	1.1	0.9, 1.2
95-99	3 260	48	1.6	1.1, 2.3	308	1.3	1.1, 1.5
≥100	3 884	89	2.8	2.0, 3.9	471	1.5	1.3, 1.7
Per 10 mmHg		374	1.4	1.3, 1.6	2155	1.1	1.1, 1.2
Women							
<75	8 964	11	0.6	0.3, 1.3	151	1.0	0.8, 1.3
75-79	5 579	7	0.5	0.2, 1.2	133	1.0	0.8, 1.2
80-84	6 563	17	1.0	Reference	239	1.0	Reference
85-89	5 294	16	1.2	0.6, 2.3	268	1.1	0.9, 1.3
90-94	4 273	16	1.5	0.8, 3.0	281	1.2	1.0, 1.4
95-99	2 725	11	1.7	0.8, 3.6	213	1.3	1.0, 1.5
≥100	3 351	20	2.8	1.4, 5.3	336	1.4	1.2, 1.7
Per 10 mmHg		98	1.5	1.3, 1.8	1621	1.1	1.1, 1.2

CI, confidence interval; DBP, diastolic blood pressure in mmHg; RR, relative risk adjusted for attained age (continuous).

Table III. Relative Risk of Ischaemic Heart Disease Mortality in Relation to the Distribution of Systolic Blood Pressure and Body-mass index, by Attained age, Nord-Trøndelag, Norway, 1984-2005.

		BMI <25 (n = 38 827)			**BMI 25-29.9** (n = 25 040)			**BMI ≥30** (n = 7 515)		
SBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI	Deaths	RR	95% CI
< 65 years										
<120	11 599	22	0.6	0.4, 1.0	20	0.9	0.5, 1.4	2	0.4	0.1, 1.8
120-139	17 419	54	1.0	Reference	75	2.0	1.4, 2.9	26	3.4	2.1, 5.4
140-159	5 516	64	3.2	2.2, 4.6	70	1.6	1.1, 2.1	33	1.4	0.8, 2.3
≥160	1 071	39	6.1	4.0, 9.3	42	2.3	1.6, 3.3	25	1.7	1.0, 3.0
p for interaction < 0.001										
≥ 65 years										
<120	3 186	62	0.8	0.6, 1.0	48	0.8	0.6, 1.1	10	1.3	0.7, 2.6
120-139	11 022	289	1.0	Reference	331	1.2	1.0, 1.4	80	1.1	0.9, 1.4
140-159	10 891	382	1.3	1.1, 1.5	534	1.2	1.1, 1.3	195	1.2	0.9, 1.6
≥160	10 678	623	2.0	1.7, 2.3	818	1.5	1.3, 1.7	404	1.7	1.3, 2.2
p for interaction = 0.046										

BMI, body-mass index; CI, confidence interval; RR, relative risk adjusted for attained age (continuous); SBP, systolic blood pressure in mmHg.

Table IV. Relative Risk of Ischaemic Heart Disease Mortality in Relation to the Distribution of Diastolic Blood Pressure and Body-mass index, by Attained age, Nord-Trøndelag, Norway, 1984-2005.

		BMI <25 (n = 38 827)			**BMI 25-29.9** (n = 25 040)			**BMI ≥30** (n = 7 515)		
DBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI	Deaths	RR	95% CI
< 65 years										
<80	16 599	41	0.6	0.4, 0.9	28	0.7	0.5, 1.1	1	0.1	0.0, 1.1
80-89	12 433	59	1.0	Reference	73	1.7	1.2, 2.4	22	2.6	1.6, 4.3
90-99	5 083	54	2.1	1.4, 3.0	62	1.2	0.8, 1.6	23	0.8	0.5, 1.5
≥100	1 490	25	3.4	2.1, 5.4	44	1.9	1.3, 2.7	40	2.0	1.2, 3.4
p for interaction = 0.023										
≥ 65 years										
<80	7 489	304	0.9	0.8, 1.1	261	0.9	0.8, 1.1	75	1.3	1.0, 1.8
80-89	12 232	421	1.0	Reference	565	1.2	1.1, 1.4	176	1.3	1.0, 1.5
90-99	10 311	397	1.3	1.1, 1.5	539	1.0	0.9, 1.1	231	1.1	0.9, 1.4
≥100	5 745	234	1.6	1.3, 1.9	366	1.2	1.1, 1.4	207	1.3	1.0, 1.6
p for interaction = 0.002										

BMI, body-mass index; CI, confidence interval; DBP, diastolic blood pressure in mmHg; RR, relative risk adjusted for attained age (continuous).

Appendix B Adjusting for competing risks (non-IHD deaths)

Table I. Systolic Blood Pressure and Relative Risk of Ischaemic Heart Disease Mortality, by Sex and Attained age, Nord-Trøndelag, Norway, 1984-2005.

		< 65 years (n = 35 605)			**≥ 65 years** (n = 35 777)		
SBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI
Men							
<119	4 298	32	1.0	0.6, 1.5	90	1.0	0.8, 1.3
120-129	7 670	61	1.0	Reference	182	1.0	Reference
130-139	8 074	65	1.0	0.7, 1.4	296	1.1	0.9, 1.4
140-149	5 947	70	1.5	1.1, 2.2	358	1.2	1.0, 1.5
150-159	3 518	66	2.5	1.8, 3.6	322	1.4	1.2, 1.7
160-169	2 145	26	1.9	1.2, 3.1	293	1.6	1.3, 2.0
≥170	2 981	54	4.0	2.7, 6.0	614	2.2	1.8, 2.6
Per 20 mmHg		374	1.5	1.4, 1.7	2155	1.2	1.2, 1.3
Women							
<119	10 487	12	0.8	0.4, 1.9	30	0.7	0.5, 1.1
120-129	7 350	12	1.0	Reference	75	1.0	Reference
130-139	5 347	17	1.8	0.9, 3.7	147	1.3	1.0, 1.8
140-149	3 976	19	2.9	1.4, 6.1	218	1.7	1.3, 2.2
150-159	2 966	12	2.7	1.2, 6.1	213	1.7	1.3, 2.2
160-169	2 279	6	2.3	0.8, 6.4	256	2.2	1.7, 2.9
≥170	4 344	20	5.9	2.7, 13.0	682	2.8	2.2, 3.5
Per 20 mmHg		98	1.5	1.3, 1.8	1621	1.3	1.2, 1.3

CI, confidence interval; RR, relative risk adjusted for attained age (continuous), body mass index (continuous), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no), adjusted for competing risks; SBP, systolic blood pressure in mmHg.

Table II. Diastolic Blood Pressure and Relative Risk of Ischaemic Heart Disease Mortality, by Sex and Attained age, Nord-Trøndelag, Norway, 1984-2005.

		< 65 years (n = 35 605)			**≥ 65 years** (n = 35 777)		
DBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI
Men							
<75	4 854	22	0.6	0.3, 1.4	163	1.0	0.8, 1.2
75-79	4 691	30	0.5	0.2, 1.3	193	1.0	0.9, 1.2
80-84	6 846	56	1.0	Reference	318	1.0	Reference
85-89	5 962	65	1.1	0.5, 2.1	337	1.1	0.9, 1.2
90-94	5 136	64	1.4	0.7, 2.8	365	1.1	0.9, 1.2
95-99	3 260	48	1.4	0.6, 3.1	308	1.2	1.1, 1.5
≥100	3 884	89	2.1	1.0, 4.3	471	1.3	1.2, 1.6
Per 10 mmHg		374	1.4	1.2, 1.7	2155	1.1	1.0, 1.1
Women							
<75	8 964	11	0.7	0.4, 1.1	151	1.0	0.8, 1.3
75-79	5 579	7	0.9	0.6, 1.4	133	0.9	0.8, 1.2
80-84	6 563	17	1.0	Reference	239	1.0	Reference
85-89	5 294	16	1.3	0.9, 1.8	268	1.1	0.9, 1.3
90-94	4 273	16	1.4	1.0, 2.0	281	1.1	0.9, 1.3
95-99	2 725	11	1.6	1.1, 2.4	213	1.2	1.0, 1.4
≥100	3 351	20	2.5	1.7, 3.6	336	1.3	1.1, 1.5
Per 10 mmHg		98	1.4	1.3, 1.5	1621	1.1	1.0, 1.1

CI, confidence interval; DBP, diastolic blood pressure in mmHg; RR, relative risk adjusted for attained age (continuous), body mass index (continuous), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no), adjusted for competing risks.

Table III. Relative Risk of Ischaemic Heart Disease Mortality in Relation to the Distribution of Systolic Blood Pressure and Body-mass index, by Attained age, Nord-Trøndelag, Norway, 1984-2005.

		BMI <25 (n = 38 827)			**BMI 25-29.9** (n = 25 040)			**BMI ≥30** (n = 7 515)		
SBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI	Deaths	RR	95% CI
< 65 years										
<120	11 599	22	0.8	0.5, 1.4	20	1.1	0.7, 1.8	2	0.5	0.1, 2.0
120-139	17 419	54	1.0	Reference	75	1.9	1.3, 2.7	26	4.0	2.5, 6.4
140-159	5 516	64	3.0	2.1, 4.3	70	1.5	1.1, 2.1	33	1.4	0.8, 2.3
≥160	1 071	39	5.8	3.8, 8.8	42	2.4	1.6, 3.5	25	1.6	0.9, 2.8
p for interaction < 0.001										
≥ 65 years										
<120	3 186	62	0.8	0.6, 1.0	48	0.8	0.6, 1.1	10	1.3	0.7, 2.5
120-139	11 022	289	1.0	Reference	331	1.3	1.1, 1.5	80	1.2	1.1, 1.6
140-159	10 891	382	1.3	1.1, 1.5	534	1.2	1.1, 1.4	195	1.3	1.1, 1.7
≥160	10 678	623	2.2	1.9, 2.6	818	1.7	1.5, 2.0	404	1.9	1.5, 2.4
p for interaction = 0.180										

BMI, body-mass index; CI, confidence interval; RR, relative risk adjusted for attained age (continuous), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no) and sex, adjusted for competing risks; SBP, systolic blood pressure in mmHg.

Table IV. Relative Risk of Ischaemic Heart Disease Mortality in Relation to the Distribution of Diastolic Blood Pressure and Body-mass index, by Attained age, Nord-Trøndelag, Norway, 1984-2005.

		BMI <25			**BMI 25-29.9**			**BMI ≥30**		
		(n = 38 827)			(n = 25 040)			(n = 7 515)		
DBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI	Deaths	RR	95% CI
< 65 years										
<80	16 599	41	0.7	0.5, 1.1	28	0.7	0.5, 1.2	1	0.1	0.0, 1.0
80-89	12 433	59	1.0	Reference	73	1.6	1.2, 2.3	22	3.4	2.1, 5.6
90-99	5 083	54	1.9	1.3, 2.8	62	1.1	0.8, 1.6	23	0.8	0.4, 1.4
≥100	1 490	25	3.2	2.0, 5.1	44	1.8	1.2, 2.6	40	1.6	0.9, 2.7
p for interaction = 0.035										
≥ 65 years										
<80	7 489	304	0.9	0.8, 1.1	261	0.9	0.8, 1.1	75	1.3	1.0, 1.7
80-89	12 232	421	1.0	Reference	565	1.3	1.1, 1.5	176	1.3	1.1, 1.6
90-99	10 311	397	1.3	1.1, 1.5	539	1.0	0.9, 1.1	231	1.0	0.8, 1.2
≥100	5 745	234	1.5	1.3, 1.8	366	1.2	1.0, 1.3	207	1.2	1.0, 1.4
p for interaction < 0.001										

BMI, body-mass index; CI, confidence interval; DBP, diastolic blood pressure in mmHg; RR, relative risk adjusted for attained age (continuous), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no) and sex, adjusted for competing risks.

Appendix C Analyses with BMI in four strata

Table I. Relative Risk of Ischaemic Heart Disease Mortality in Relation to the Distribution of Systolic Blood Pressure and Body-mass index, by Attained age, Nord-Trøndelag, Norway, 1984-2005.

		BMI <18.5 (n = 993)			**BMI 18.5-24.9** (n = 37 834)			**BMI 25-29.9** (n = 25 040)			**BMI ≥30** (n = 7 515)		
SBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI	Deaths	RR	95% CI	Deaths	RR	95% CI
< 65 years													
<120	11 599	1	21.5	21.0, 22.0	21	0.8	0.5, 1.4	20	1.1	0.7, 1.8	2	0.5	0.1, 2.0
120-139	17 419	0	NC	NC	54	1.0	Reference	75	1.9	1.3, 2.7	26	4.0	2.5, 6.3
140-159	5 516	0	NC	NC	64	2.9	2.0, 4.2	70	1.5	1.1, 2.1	33	1.4	0.8, 2.3
≥160	1 071	2	NC	NC	37	5.4	3.6, 8.3	42	2.4	1.6, 3.5	25	1.6	0.9, 2.8
p for interaction < 0.001													
≥ 65 years													
<120	3 186	3	NC	NC	59	0.8	0.6, 1.1	48	0.9	0.6, 1.2	10	1.3	0.7, 2.5
120-139	11 022	14	2.6	1.5, 4.5	275	1.0	Reference	331	1.3	1.1, 1.5	80	1.4	1.1, 1.8
140-159	10 891	9	0.6	0.3, 1.5	373	1.3	1.1, 1.5	534	1.2	1.0, 1.4	195	1.2	0.9, 1.6
≥160	10 678	15	0.9	0.5, 1.9	608	2.2	1.9, 2.5	818	1.7	1.5, 1.9	404	1.8	1.4, 2.3
p for interaction = 0.058													

BMI, body-mass index; CI, confidence interval; NC, not computable; RR, relative risk adjusted for attained age (continuous), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no) and sex; SBP, systolic blood pressure in mmHg.

Table II. Relative Risk of Ischaemic Heart Disease Mortality in Relation to the Distribution of Diastolic Blood Pressure and Body-mass index, by Attained age, Nord-Trøndelag, Norway, 1984-2005.

		BMI <18.5 (n = 993)			**BMI 18.5-24.9** (n = 37 834)			**BMI 25-29.9** (n = 25 040)			**BMI ≥30** (n = 7 515)		
DBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI	Deaths	RR	95% CI	Deaths	RR	95% CI
< 65 years													
<80	16 599	0	NC	NC	41	0.8	0.5, 1.1	28	0.7	0.5, 1.2	1	0.1	0.0, 1.0
80-89	12 433	2	3.5	0.9, 14.4	57	1.0	Reference	73	1.7	1.2, 2.4	22	3.5	2.1, 5.7
90-99	5 083	1	1.0	0.1, 10.6	53	2.0	1.4, 2.9	62	1.1	0.8, 1.6	23	0.8	0.4, 1.4
≥100	1 490	0	NC	NC	25	3.3	2.1, 5.3	44	1.8	1.3, 2.7	40	1.6	1.0, 2.7
p for interaction = 0.014													
≥ 65 years													
<80	7 489	16	NC	NC	288	0.9	0.8, 1.0	261	0.9	0.8, 1.1	75	1.3	1.0, 1.7
80-89	12 232	11	1.6	0.9, 2.9	410	1.0	Reference	565	1.3	1.1, 1.4	176	1.4	1.2, 1.7
90-99	10 311	9	1.1	0.5, 2.7	388	1.4	1.2, 1.6	539	1.0	0.9, 1.2	231	1.1	0.9, 1.3
≥100	5 745	5	1.2	0.4, 3.5	229	1.7	1.5, 2.0	366	1.3	1.1, 1.5	207	1.3	1.1, 1.6
p for interaction < 0.001													

BMI, body-mass index; CI, confidence interval; DBP, diastolic blood pressure in mmHg; NC, not computable; RR, relative risk adjusted for attained age (continuous), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no) and sex.

Appendix D Analyses adjusting also for blood pressure medication at baseline

Table I. Systolic Blood Pressure and Relative Risk of Ischaemic Heart Disease Mortality, by Sex and Attained age, Nord-Trøndelag, Norway, 1984-2005.

		< 65 years (n = 35 605)			**≥ 65 years** (n = 35 777)		
SBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI
Men							
<119	4 298	32	1.0	0.6, 1.5	90	1.0	0.8, 1.3
120-129	7 670	61	1.0	Reference	182	1.0	Reference
130-139	8 074	65	1.0	0.7, 1.4	296	1.1	0.9, 1.4
140-149	5 947	70	1.5	1.1, 2.1	358	1.2	1.0, 1.4
150-159	3 518	66	2.4	1.7, 3.4	322	1.3	1.1, 1.6
160-169	2 145	26	1.7	1.1, 2.8	293	1.5	1.2, 1.8
≥170	2 981	54	3.5	2.3, 5.1	614	1.9	1.6, 2.3
Per 20 mmHg		374	1.5	1.3, 1.6	2155	1.2	1.2, 1.3
Women							
<119	10 487	12	0.9	0.4, 1.9	30	0.8	0.5, 1.2
120-129	7 350	12	1.0	Reference	75	1.0	Reference
130-139	5 347	17	1.7	0.8, 3.6	147	1.3	1.0, 1.7
140-149	3 976	19	2.8	1.3, 5.8	218	1.5	1.2, 2.0
150-159	2 966	12	2.5	1.1, 5.7	213	1.5	1.1, 2.0
160-169	2 279	6	2.1	0.8, 5.8	256	1.9	1.5, 2.5
≥170	4 344	20	5.3	2.4, 11.5	682	2.3	1.8, 3.0
Per 20 mmHg		98	1.5	1.3, 1.8	1621	1.3	1.2, 1.3

CI, confidence interval; RR, relative risk adjusted for attained age (continuous), blood pressure medication (yes, no), body mass index (continuous), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no); SBP, systolic blood pressure in mmHg.

Table II. Diastolic Blood Pressure and Relative Risk of Ischaemic Heart Disease Mortality, by Sex and Attained age, Nord-Trøndelag, Norway, 1984-2005.

		< 65 years (n = 35 605)			**≥ 65 years** (n = 35 777)		
DBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI
Men							
<75	4 854	22	0.7	0.3, 1.4	163	1.0	0.9, 1.3
75-79	4 691	30	0.5	0.2, 1.3	193	1.0	0.8, 1.2
80-84	6 846	56	1.0	Reference	318	1.0	Reference
85-89	5 962	65	1.0	0.5, 2.1	337	1.1	0.9, 1.3
90-94	5 136	64	1.3	0.7, 2.6	365	1.1	0.9, 1.3
95-99	3 260	48	1.2	0.6, 2.7	308	1.2	1.0, 1.4
≥100	3 884	89	1.9	0.9, 3.7	471	1.3	1.0, 1.5
Per 10 mmHg		374	1.3	1.1, 1.6	2155	1.1	1.0, 1.1
Women							
<75	8 964	11	0.7	0.4, 1.2	151	1.0	0.8, 1.2
75-79	5 579	7	0.9	0.6, 1.4	133	1.0	0.8, 1.2
80-84	6 563	17	1.0	Reference	239	1.0	Reference
85-89	5 294	16	1.3	0.9, 1.8	268	1.0	0.9, 1.2
90-94	4 273	16	1.4	0.9, 1.9	281	1.0	0.9, 1.3
95-99	2 725	11	1.5	1.0, 2.2	213	1.2	1.1, 1.5
≥100	3 351	20	2.2	1.5, 3.1	336	1.3	1.2, 1.6
Per 10 mmHg		98	1.3	1.2, 1.5	1621	1.1	1.1, 1.2

CI, confidence interval; DBP, diastolic blood pressure in mmHg; RR, relative risk adjusted for attained age (continuous), blood pressure medication (yes, no), body mass index (continuous), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no).

Table III. Relative Risk of Ischaemic Heart Disease Mortality in Relation to the Distribution of Systolic Blood Pressure and Body-mass index, by Attained age, Nord-Trøndelag, Norway, 1984-2005.

		BMI <25 (n = 38 827)			**BMI 25-29.9** (n = 25 040)			**BMI ≥30** (n = 7 515)		
SBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI	Deaths	RR	95% CI
< 65 years										
<120	11 599	22	0.9	0.5, 1.5	20	1.1	0.7, 1.9	2	0.5	0.1, 2.0
120-139	17 419	54	1.0	Reference	75	1.8	1.3, 2.6	26	3.7	2.3, 5.9
140-159	5 516	64	2.8	2.0, 4.1	70	1.4	1.0, 2.0	33	1.3	0.8, 2.2
≥160	1 071	39	5.1	3.4, 7.8	42	2.1	1.4, 3.1	25	1.4	0.8, 2.4
p for interaction < 0.001										
≥ 65 years										
<120	3 186	62	0.8	0.6, 1.1	48	0.9	0.7, 1.2	10	1.3	0.7, 2.6
120-139	11 022	289	1.0	Reference	331	1.2	1.0, 1.4	80	1.2	1.0, 1.6
140-159	10 891	382	1.2	1.1, 1.4	534	1.1	1.0, 1.3	195	1.1	0.9, 1.5
≥160	10 678	623	2.0	1.7, 2.3	818	1.5	1.3, 1.7	404	1.6	1.3, 2.1
p for interaction = 0.046										

BMI, body-mass index; CI, confidence interval; RR, relative risk adjusted for attained age (continuous), blood pressure medication (yes, no), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no) and sex; SBP, systolic blood pressure in mmHg.

Table IV. Relative Risk of Ischaemic Heart Disease Mortality in Relation to the Distribution of Diastolic Blood Pressure and Body-mass index, by Attained age, Nord-Trøndelag, Norway, 1984-2005.

		BMI <25			**BMI 25-29.9**			**BMI ≥30**		
		(n = 38 827)			(n = 25 040)			(n = 7 515)		
DBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI	Deaths	RR	95% CI
< 65 years										
<80	16 599	41	0.7	0.5, 1.1	28	0.7	0.5, 1.2	1	0.1	0.0, 1.0
80-89	12 433	59	1.0	Reference	73	1.6	1.2, 2.3	22	3.4	2.1, 5.6
90-99	5 083	54	1.9	1.3, 2.8	62	1.1	0.8, 1.6	23	0.8	0.4, 1.4
≥100	1 490	25	3.2	2.0, 5.1	44	1.8	1.2, 2.6	40	1.6	0.9, 2.7
p for interaction = 0.035										
≥ 65 years										
<80	7 489	304	0.9	0.8, 1.1	261	0.9	0.8, 1.1	75	1.3	1.0, 1.7
80-89	12 232	421	1.0	Reference	565	1.3	1.1, 1.5	176	1.3	1.1, 1.6
90-99	10 311	397	1.3	1.1, 1.5	539	1.0	0.9, 1.1	231	1.0	0.8, 1.2
≥100	5 745	234	1.5	1.3, 1.8	366	1.2	1.0, 1.3	207	1.2	1.0, 1.4
p for interaction < 0.001										

BMI, body-mass index; CI, confidence interval; DBP, diastolic blood pressure in mmHg; RR, relative risk adjusted for attained age (continuous), blood pressure medication (yes, no), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no) and sex.

Appendix E Adjusting for month of baseline measurments

Table I. Systolic Blood Pressure and Relative Risk of Ischaemic Heart Disease Mortality, by Sex and Attained age, Nord-Trøndelag, Norway, 1984-2005.

		< 65 years (n = 35 605)			**≥ 65 years** (n = 35 777)		
SBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI
Men							
<119	4 298	32	1.0	0.6, 1.5	90	1.0	0.8, 1.3
120-129	7 670	61	1.0	Reference	182	1.0	Reference
130-139	8 074	65	1.0	0.7, 1.4	296	1.2	1.0, 1.4
140-149	5 947	70	1.5	1.1, 2.1	358	1.2	1.0, 1.5
150-159	3 518	66	2.5	1.8, 3.6	322	1.4	1.1, 1.7
160-169	2 145	26	1.9	1.2, 3.0	293	1.6	1.3, 1.9
≥170	2 981	54	4.0	2.7, 5.9	614	2.1	1.8, 2.5
Per 20 mmHg		374	1.5	1.4, 1.7	2155	1.2	1.2, 1.3
Women							
<119	10 487	12	0.9	0.4, 1.9	30	0.7	0.5, 1.1
120-129	7 350	12	1.0	Reference	75	1.0	Reference
130-139	5 347	17	1.8	0.8, 3.7	147	1.3	1.0, 1.7
140-149	3 976	19	2.9	1.4, 6.0	218	1.6	1.2, 2.1
150-159	2 966	12	2.7	1.2, 6.1	213	1.6	1.2, 2.1
160-169	2 279	6	2.3	0.9, 6.3	256	2.1	1.6, 2.7
≥170	4 344	20	5.9	2.8, 12.7	682	2.6	2.0, 3.3
Per 20 mmHg		98	1.5	1.3, 1.8	1621	1.3	1.2, 1.3

CI, confidence interval; RR, relative risk adjusted for attained age (continuous), body mass index (continuous), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no), month of baseline measurements; SBP, systolic blood pressure in mmHg.

Table II. Diastolic Blood Pressure and Relative Risk of Ischaemic Heart Disease Mortality, by Sex and Attained age, Nord-Trøndelag, Norway, 1984-2005.

		< 65 years (n = 35 605)			**≥ 65 years** (n = 35 777)		
DBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI
Men							
<75	4 854	22	0.7	0.3, 1.4	163	1.0	0.8, 1.3
75-79	4 691	30	0.5	0.2, 1.3	193	1.0	0.8, 1.2
80-84	6 846	56	1.0	Reference	318	1.0	Reference
85-89	5 962	65	1.1	0.5, 2.1	337	1.1	1.0, 1.4
90-94	5 136	64	1.4	0.7, 2.8	365	1.2	1.0, 1.4
95-99	3 260	48	1.4	0.6, 3.0	308	1.3	1.0, 1.5
≥100	3 884	89	2.2	1.1, 4.3	471	1.4	1.2, 1.7
Per 10 mmHg		374	1.4	1.2, 1.7	2155	1.1	1.0, 1.1
Women							
<75	8 964	11	0.7	0.4, 1.2	151	1.0	0.8, 1.2
75-79	5 579	7	0.9	0.6, 1.4	133	1.0	0.8, 1.2
80-84	6 563	17	1.0	Reference	239	1.0	Reference
85-89	5 294	16	1.3	0.9, 1.8	268	1.1	0.9, 1.2
90-94	4 273	16	1.4	1.0, 2.0	281	1.1	0.9, 1.3
95-99	2 725	11	1.6	1.1, 2.4	213	1.3	1.1, 1.6
≥100	3 351	20	2.6	1.8, 3.7	336	1.5	1.3, 1.7
Per 10 mmHg		98	1.4	1.3, 1.5	1621	1.1	1.0, 1.1

CI, confidence interval; DBP, diastolic blood pressure in mmHg; RR, relative risk adjusted for attained age (continuous), body mass index (continuous), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no), month of baseline measurements.

Table III. Relative Risk of Ischaemic Heart Disease Mortality in Relation to the Distribution of Systolic Blood Pressure and Body-mass index, by Attained age, Nord-Trøndelag, Norway, 1984-2005.

		BMI <25 (n = 38 827)			**BMI 25-29.9** (n = 25 040)			**BMI ≥30** (n = 7 515)		
SBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI	Deaths	RR	95% CI
< 65 years										
<120	11 599	22	0.9	0.5, 1.4	20	1.1	0.7, 1.8	2	0.5	0.1, 2.0
120-139	17 419	54	1.0	Reference	75	1.9	1.3, 2.7	26	4.0	2.5, 6.4
140-159	5 516	64	3.0	2.1, 4.3	70	1.5	1.1, 2.1	33	1.4	0.8, 2.3
≥160	1 071	39	5.8	3.8, 8.8	42	2.4	1.6, 3.5	25	1.6	0.9, 2.8
p for interaction < 0.001										
≥ 65 years										
<120	3 186	62	0.8	0.6, 1.0	48	0.9	0.6, 1.1	10	1.3	0.7, 2.5
120-139	11 022	289	1.0	Reference	331	1.3	1.1, 1.5	80	1.3	1.1, 1.7
140-159	10 891	382	1.3	1.1, 1.5	534	1.2	1.1, 1.4	195	1.2	0.9, 1.6
≥160	10 678	623	2.1	1.9, 2.5	818	1.6	1.4, 1.9	404	1.7	1.4, 2.2
p for interaction = 0.048										

BMI, body-mass index; CI, confidence interval; RR, relative risk adjusted for attained age (continuous), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no), month of baseline measurements and sex; SBP, systolic blood pressure in mmHg.

Table IV. Relative Risk of Ischaemic Heart Disease Mortality in Relation to the Distribution of Diastolic Blood Pressure and Body-mass index, by Attained age, Nord-Trøndelag, Norway, 1984-2005.

		BMI <25			**BMI 25-29.9**			**BMI ≥30**		
		(n = 38 827)			(n = 25 040)			(n = 7 515)		
DBP	Total, n	Deaths	RR	95% CI	Deaths	RR	95% CI	Deaths	RR	95% CI
< 65 years										
<80	16 599	41	0.7	0.5, 1.1	28	0.8	0.5, 1.2	1	0.1	0.0, 1.0
80-89	12 433	59	1.0	Reference	73	1.6	1.2, 2.3	22	3.4	2.1, 5.5
90-99	5 083	54	2.0	1.3, 2.8	62	1.2	0.8, 1.6	23	0.8	0.4, 1.4
≥100	1 490	25	3.3	2.0, 5.2	44	1.9	1.3, 2.7	40	1.6	1.0, 2.7
p for interaction = 0.043										
≥ 65 years										
<80	7 489	304	0.9	0.8, 1.1	261	0.9	0.8, 1.1	75	1.3	1.0, 1.7
80-89	12 232	421	1.0	Reference	565	1.3	1.1, 1.4	176	1.3	1.1, 1.6
90-99	10 311	397	1.4	1.2, 1.6	539	1.0	0.9, 1.1	231	1.1	0.9, 1.3
≥100	5 745	234	1.7	1.4, 2.0	366	1.3	1.1, 1.5	207	1.3	1.1, 1.6
p for interaction < 0.001										

BMI, body-mass index; CI, confidence interval; DBP, diastolic blood pressure in mmHg; RR, relative risk adjusted for attained age (continuous), categories of smoking (never, former, current, unknown), diabetes mellitus (yes, no), month of baseline measurements and sex.

Online only material Paper III

ONLINE ONLY MATERIAL

Webtable 1 Other methods of calculating informativeness of different blood pressure, obesity and serum lipid indices related to ischaemic heart disease mortality, for men

Men	**AUC**	**R^2**	**HR**	**NRI**	**IDI**
Blood pressure					
Systolic blood pressure	0.841	0.162	1.153	0.020	0.004
Mid blood pressure	0.839	0.160	1.157	0.012	0.003
Pulse pressure × Mean arterial pressure	0.841	0.162	1.148	0.020	0.004
Mean arterial pressure	0.838	0.159	1.154	0.013	0.002
Pulse pressure	0.840	0.160	1.114	0.024	0.004
Diastolic blood pressure	0.835	0.156	1.128	0.011	<0.001
Obesity					
Waist-to-hip ratio adjusted for BMI	0.838	0.158	1.202	0.015	0.002
Waist-to-hip ratio	0.838	0.157	1.149	0.006	<0.001
Waist-to-height ratio	0.838	0.158	1.149	0.017	0.002
Weight	0.834	0.154	0.935	-0.005	<0.001
Waist circumference	0.835	0.155	1.086	-0.003	-0.001
Hip circumference	0.833	0.153	0.979	0.011	0.001
Body-mass index	0.833	0.153	1.010	0.003	<0.001
Serum lipids					
Total:HDL-cholesterol ratio	0.838	0.160	1.256	0.023	0.003
Non-HDL cholesterol	0.837	0.160	1.259	0.026	0.003
Total cholesterol	0.835	0.158	1.203	0.022	0.002
HDL cholesterol	0.836	0.156	0.857	0.012	0.001
Triglycerides	0.834	0.154	1.114	-0.001	<0.001

Sorted as the corresponding tables in the article. Adjusted for attained age. AUC: area under the curve; R^2: pseudo R^2 from logistic regression; HR: hazard ratio associated with 1 standard deviation increase; NRI: net reclassification improvement using a 10% cut-off; IDI: integrated discrimination improvement.

Webtable 2 Other methods of calculating informativeness of different blood pressure, obesity and serum lipid indices related to ischaemic heart disease mortality, for women

Women	**AUC**	**R^2**	**HR**	**NRI**	**IDI**
Blood pressure					
Pulse pressure	0.885	0.207	1.145	0.020	0.003
Pulse pressure × Mean arterial pressure	0.883	0.205	1.103	0.006	0.002
Systolic blood pressure	0.882	0.204	1.086	0.008	0.001
Mid blood pressure	0.880	0.201	1.041	0.002	<0.001
Diastolic blood pressure	0.878	0.199	0.968	<0.001	<0.001
Mean arterial pressure	0.879	0.200	1.018	<0.001	<0.001
Obesity					
Waist-to-hip ratio adjusted for BMI	0.883	0.205	1.264	0.015	0.002
Waist-to-hip ratio	0.883	0.205	1.242	0.010	0.002
Waist-to-height ratio	0.882	0.203	1.158	-0.006	<0.001
Waist circumference	0.880	0.201	1.132	-0.008	<0.001
Body-mass index	0.878	0.199	1.021	-0.007	-0.001
Weight	0.878	0.199	0.985	0.002	<0.001
Hip circumference	0.878	0.199	0.992	-0.007	<0.001
Serum lipids					
Total:HDL-cholesterol ratio	0.882	0.207	1.231	0.035	0.004
Triglycerides	0.882	0.206	1.222	0.020	0.003
HDL cholesterol	0.881	0.204	0.814	0.015	0.003
Non-HDL cholesterol	0.881	0.204	1.220	0.009	0.001
Total cholesterol	0.880	0.202	1.144	-0.014	<0.001

Sorted as the corresponding tables in the article. Adjusted for attained age. AUC: area under the curve; R^2: pseudo R^2 from logistic regression; HR: hazard ratio associated with 1 standard deviation increase; NRI: net reclassification improvement using a 10% cut-off; IDI: integrated discrimination improvement.

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27. Per Martin Kleveland: STUDIES ON GASTRIN.
28. Audun N. Øksendal: THE CALCIUM PARADOX AND THE HEART.
29. Vilhjalmur R. Finsen: HIP FRACTURES

1988

30. 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-Arne Rein: THE EFFECT OF EXTRACORPOREAL CIRCULATION ON SUBCUTANEOUS TRANSCAPILLARY FLUID BALANCE.
41. Arne 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.
59. Arne Z. Henriksen: STUDIES ON CONSERVED ANTIGENIC DOMAINS ON MAJOR OUTER MEMBRANE PROTEINS FROM ENTEROBACTERIA.
60. Steinar Westin: UNEMPLOYMENT AND HEALTH: Medical and social consequences of a factory closure in a ten-year controlled follow-up study.
61. Ylva 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.
69. Kjetil B. Åsbakk: STUDIES OF A PROTEIN FROM PSORIATIC SCALE, PSO P27, WITH RESPECT TO ITS POTENTIAL ROLE IN IMMUNE REACTIONS IN PSORIASIS.
70. Arnulf Hestnes: STUDIES ON DOWN´S SYNDROME.
71. 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: *erb*B 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.
96. Stein Olav Samstad: CROSS SECTIONAL FLOW VELOCITY PROFILES FROM TWO-DIMENSIONAL DOPPLER ULTRASOUND: Studies on early mitral blood flow.
97. Bjørn 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. Phsysiological and Clinical Role.

1995

104. Odd 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.
109. Arild Faxvaag: STUDIES OF IMMUNE CELL FUNCTION *in mice infected with* MURINE RETROVIRUS.

1996

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 CANCER.
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.

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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: COMPUTERIZED ECHOCARDIOGRAPHY 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

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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.

1999

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 Mårvik: 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

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158. Ola Dalsegg Sæther: PATHOPHYSIOLOGY DURING PROXIMAL AORTIC CROSS-CLAMPING CLINICAL AND EXPERIMENTAL STUDIES
159. xxxxxxxxx (blind number)
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 Sue-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.
174. 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.

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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

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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 ß-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

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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 ANAESTHESIA – 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
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