Socio-demographic, medical and psychosocial factors associated with unfavourable risk factor control after coronary events

A cross-sectional study of a Norwegian coronary population with detailed analyses of elevated blood pressure and smoking

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bekymringsfullt at sekundærforebyggende behandling og oppfølging ikke er bedre i klinisk praksis i Norge. Vi har funnet at det er forskjellige faktorer som har betydning for blodtrykk og røyking. Sekundærforbyggende behandling og oppfølging bør derfor i fremtiden i større grad skreddersys til pasientenes sosiodemografiske, medisinske og psykososiale risikoprofil.
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Preface
This PhD-thesis is based on the three papers listed below. The work has been carried out at the Department of Medicine, Drammen Hospital, Vestre Viken Trust, Vestfold Hospital Trust, and Department of Behavioural Sciences in Medicine, the Faculty of Medicine, University of Oslo, during the years 2015-2017.

Paper I
Unfavourable risk factor control after coronary events in routine clinical practice.

Paper II
Medical and sociodemographic factors predict persistent smoking after coronary events.

Paper III
Optimal blood pressure control after coronary events: the challenge remains.
Abbreviations and definitions

ACEI  Angiotensin converting enzyme inhibitor
ARB  Angiotensin receptor blocker
BMI  Body Mass Index
BP  Blood pressure
CABG  Coronary artery bypass grafting operation
CHD  Coronary heart disease
CI  Confidence interval
CR  Cardiac rehabilitation
CVD  Cardiovascular disease
DALY  Disability-adjusted life-years
GP  General practitioner
HADS  Hospital anxiety and depression scale
HbA1c  Glycated haemoglobin A1c
LDL  Low density lipoprotein
MI  Myocardial infarction
NOAC  New oral anticoagulant
NSTEMI  Non-ST elevation myocardial infarction
OLS  Ordinary Least Squares
OR  Odds ratio
PCI  Percutaneous coronary intervention
PSWQ  Penn State Worry Questionnaire
RCT  Randomized clinical trial
SD  Standard deviation
STEMI  ST elevation myocardial infarction

Definitions

Risk factor control – outcome variables
Risk factor control is defined as achieving the treatment targets according to the European Guidelines on cardiovascular disease prevention\textsuperscript{1,2}. In the present thesis these include; not smoking, blood pressure <140/90 (140/80 in diabetic patients), low density lipoprotein cholesterol <1.8 mmol/L, BMI <25.0 kg/m\textsuperscript{2}, moderate to vigorous physical activity for 30 minutes \geq3 times weekly, and HbA1c <7.0% (diabetics). In the present thesis, risk factor control has been both an outcome variable and covariates (see also Method Section page 26-27)

Study factors - covariates
The NOR-COR study factors comprise the socio-demographic, psychosocial and medical factors defined below;

Socio-demographic factors
With the term socio-demographic factors, we understand demographic factors such as age, sex, ethnicity, and marital status and factors related to socio-economic status such as education level and
employment status. In the literature, socio-economic factors have been defined as both (socio-)demographic variables and psychosocial variables. We have chosen to categorize educational level and employment status in socio-demographic variables.

_Psychosocial factors_
There is no clear agreement upon how the term “psychosocial factors” should be defined. Factors related to the social environment, personality traits, and negative affect have been classified as psychosocial in a recent consensus document from the ESC work group. Rozanski et al. divided psychosocial factors into emotional factors such as depression, and anxiety and chronic stressors such as low socio-economic status, work stress and poor social support. In the present thesis we have categorized emotional factors (depression, anxiety, and worry) as psychosocial factors and low socio-economic status and marital status as socio-demographic factors. We have also classified type D personality, insomnia, motivation for lifestyle change, illness and risk perception as psychosocial factors.

_Medical factors_
The term medical factors include factors related to the coronary event, coronary risk factors, somatic comorbidity, medication and adherence. We have decided to categorize participation in cardiac rehabilitation as a medical factor.
Summary

Background
Coronary heart disease (CHD) is a leading cause of mortality and morbidity globally. The pivotal role of the major cardiovascular risk factors for development and progression of CHD and the beneficial effect of treatment of these risk factors on prognosis have been overwhelmingly documented. A major public health concern is that a significant majority of CHD patients fail to achieve the evidence-based recommendations for risk factor control across Europe. Data on risk factor control after CHD events in Norway are lacking. Varying participation rates and patient selection mainly from academic centres in European multi-centre studies may overestimate adherence to risk factor control in the general population of CHD patients. Estimates based on studies of everyday clinical practice are therefore needed. Understanding the factors that influence risk factor control is necessary in order to individualize treatment and modelling interventions that may improve risk factor control and prognosis. This PhD thesis aims to determine the control of the major coronary risk factors after a coronary event in a representative population, and to identify socio-demographic, medical and psychosocial factors associated with persistent smoking and unfavourable blood pressure.

Methods
NOR-COR is a cross-sectional explorative multi-centre (Drammen and Vestfold) study carried out at a routine clinical practice in Norway. The study included 1127 patients (83% participation rate) aged 31-80 years hospitalized with acute myocardial infarction and/or a coronary revascularization procedure (i.e. percutaneous coronary intervention, coronary by-pass graft operation) in 2011-14. Study data were collected from hospital medical records at the index event and a comprehensive self-report questionnaire with a clinical examination and blood samples after 2-36 (median 16) months follow-up.

Results
Mean age was 62 (SD 10) years and 21% were women. The index event was myocardial infarction in 80% of the patients and angina with angiographically verified coronary stenosis in 20% of the patients. Thirty percent had one or more previous coronary events. At follow-up, 21% of the patients were daily smokers. Obesity was found in 34%, while 60% had central obesity. Sixty percent reported less than 30 minutes moderate activity 2 to 3 times a week, while 18% reported physical activity less than once weekly. Although 93% were taking BP lowering drugs and statins, 46% had BP >140/90 (140/80 in diabetics) mmHg and 57% had LDL cholesterol >1.8mmol/L at follow-up. Fifty-nine percent of the diabetic patients had HbA1c >7.0%, while 35% had HbA1c >8.0%. The patients had on average three out of six risk factors not at target according to guidelines recommendations, and patients with >1 previous coronary event had the poorest overall risk factor control. In multi-adjusted analysis, the patients <50 years had higher prevalence of persistent smoking, obesity, and unfavourable HbA1c than patients ≥50 years, while unfavourable BP control was more prevalent in older patients. Persistent smoking, low physical activity and unfavourable LDL cholesterol were more prevalent in women than men.
At the index event thirty-six percent (n=390) of the patients were smoking, 57% of them reported persistent smoking at follow up, while 43% had managed to quit. In multi-adjusted analyses, unemployed or disability benefits (Odds ratio (OR) 4.1), low education (OR 3.5), longer smoking duration (OR 2.3) and not having ST-elevation myocardial infarction (STEMI) as index event (OR 2.3) were significantly associated with persistent smoking. Psychosocial factors were not associated with persistent smoking and these findings were consistent in sub-group analyses by age and gender. Almost all persistent smokers had a long (>20 years) history of smoking and 60% had been smoking for more than 40 years. Smokers rated use of tobacco as the most important cause of their coronary disease (6.8 on a 1–10 Likert scale). Persistent smokers reported high motivation (average 7.8 on a 1–10 Likert scale) for smoking cessation, and only 14% reported low (≤ 3) motivation. Two thirds of the smokers wanted help to quit smoking, while only 42% reported to have been offered nicotine replacement therapy or other cessation aids.

Mean BP at follow-up was 138 (SD 19.0) / 81 (SD 8.7) mmHg and 46% of the patients had BP >140/90 (80 in diabetics) mmHg. Diabetes (OR 2.4), higher body mass index (BMI) (OR 1.05 per 1.0 kg/m^2) and older age (OR 1.04 per year), were significantly associated with unfavourable BP control in adjusted analyses. Low socio-economic status, psychosocial factors, participation in cardiac rehabilitation, self-reported drug adherence or side-effects of BP lowering drugs were not associated with BP control. Age (standardized beta (β) 0.24), BMI (β 0.07) and lower sum score of Hospital Anxiety and Depression Scale depression (β -0.073, p<0.05) were the only factors associated with increasing systolic BP in adjusted linear analyses. Patients with unfavourable BP used on average 1.9 (SD 1.1) BP lowering drugs at hospital discharge and the proportion of patients treated with angiotensin converting enzyme inhibitor / angiotensin receptor and blocker beta-blockers decreased significantly (p<0.001) from discharge to follow-up.

**Conclusions**

The majority of patients from routine clinical practice in a representative Norwegian population had unfavourable risk factor control 2-36 months after a coronary event. The poorest overall control was found in patients with several coronary events, while the youngest patients had the least favourable lifestyle.

Low socio-economic status, longer duration of smoking, and not having STEMI as index event were associated with persisting smoking, while psychosocial factors and participation in cardiac rehabilitation were not. Persistent smokers in this study seemed to have an acceptable risk perception and were motivated to cease smoking, but did not receive assistance through cessation programmes including prescription of pharmacological aids.

Older age, obesity and diabetes were the major determinants of unfavourable BP control, while socio-economic status, self-reported drug adherence, and psychosocial factors did not predict failure to control BP in our study. Prescription of BP lowering drugs in hypertensive patients seems suboptimal. Overweight and intensified drug treatment thus emerges as the major factors to target in order to improve BP control.
It is concerning that secondary prevention fails in a country like Norway with a well-developed health care system, emphasizing both a great potential and urgent need for better management of the established risk factors. By analysing the comprehensive interdisciplinary data set as an integrated unity, we have demonstrated that different modifiable and non-modifiable factors influence each of the major cardiovascular risk factors. Secondary preventive management in clinical practice and future intervention studies should therefore be individually tailored to the patient’s underlying socio-demographic, medical and psychosocial risk profile.
1. Introduction

1.1 General introduction to coronary heart disease (CHD) prevention

Coronary heart disease (CHD) is the single leading cause of disability-adjusted life-years (DALY) and premature death globally and more than 13 000 patients are annually diagnosed with myocardial infarction (MI) in Norway. Costs related to CHD management represent a significant economic burden to the healthcare system in Europe. The major risk factors for the development of CHD are well established. Hypertension, unfavourable cholesterol, diabetes, smoking, physical inactivity, obesity, unhealthy diet, and psychosocial factors have been found to account for more than 90% of the population attributable risk of myocardial infarction in a large worldwide case-control study. Poor control of the major risk factors has also been associated with increased risk of subsequent cardiovascular events and mortality in patients with established CHD. Even though a complex array of genetic, inflammatory and non-inflammatory factors probably contributes to the development and progression of CHD, the current challenge in combating CHD is to efficiently target the established cardiovascular risk factors that account for most of the CHD events.

In the latest guidelines from the European Society of Cardiology, CHD prevention is defined as “a coordinated set of actions, at the population level or targeted at an individual, that are aimed at eliminating or minimizing the impact of CHDs and their related disabilities”. It is typically divided into primary prevention, i.e. reducing the cardiovascular risk in persons with elevated risk without established disease and secondary prevention, i.e. reducing disease progression and the risk of subsequent cardiovascular events in patients with established disease. Extensive scientific evidence has demonstrated that healthy lifestyle changes (non-smoking, healthy diet, regular exercise, weight reduction), appropriate medical treatment of unfavourably elevated blood pressure and blood cholesterol, as well as metabolic control in diabetes, improve prognosis in patients with and without CHD. It has been estimated that eliminating the established cardiovascular risk factors would prevent more than 80% of CVDs and even 40% of cancers. The present PhD thesis concerns itself with secondary coronary prevention of CHD, which will be the focus of the forthcoming chapters.

1.2 A brief review of the history of CHD prevention

Until the era of modern epidemiology from the middle of the 20th century, little was known about the causes of CHD. At that time CHD was believed to be mainly caused by age and heridity. Before the middle of the 20th century, epidemiological data were undervalued as scientific evidence and mainly applied in research of infectious diseases. This may partly explain the extensive time elapsed between an association between a risk factor and disease being detected and this finding being generally accepted, such as smoking and the risk of both lung cancer and CHD. The first reports about factors associated with increased risk of CHD were published before the 1950s, as described in a review by Paul. Examples are the studies reporting the association between hypertension (Allan 1934), obesity (Dublin 1931) and smoking (English et al 1940) and CHD. Even earlier, the observation that patients with CHD often had high levels of cholesterol (Mjassnikow 1925) had been reported. With the initiation of large-scale observational studies like the study on
smoking by Hammond et al\textsuperscript{39}, the Framingham heart study\textsuperscript{35}, and the British doctors study\textsuperscript{40}, the significance of these risk factors for CHD and other conditions was increasingly recognized. The era of CHD prevention emerged in the late fifties and the beginning of the sixties, when an increasing number of reports established hypertension\textsuperscript{41}, smoking\textsuperscript{39,40,42}, high cholesterol\textsuperscript{41}, unfavourable diet\textsuperscript{36} and obesity\textsuperscript{41} as risk factors for the development of CHD. In addition, the beneficial effect of physical activity on CHD events was documented\textsuperscript{43}. The effect of BP lowering drug therapy\textsuperscript{44}, healthy lifestyle changes including smoking cessation\textsuperscript{45}, improved diets\textsuperscript{36} and increased physical activity\textsuperscript{46} on CHD/CVD reduction was gradually documented during the 1960s and 1970s in observational studies and in randomized trials. The effect on cardiac prognosis of reducing LDL cholesterol with statins was first documented in the 4S Study in 1994\textsuperscript{47}. From then on, the number of scientific publications in coronary prevention has increased substantially and the first European guidelines on prevention of CVD were presented in the same year\textsuperscript{48}. In 2016, the sixth version of the European guidelines was published\textsuperscript{2}.

1.3 Burden and temporal trends of CHD and the association with cardiovascular risk factors

The age-adjusted CHD mortality rates have declined in the western world over the past three decades in both Europe\textsuperscript{49-51} and Norway\textsuperscript{52}. This mortality reduction is a result of successful primary preventive strategies and improved diagnostics and acute treatment of CHD events and their complications by coronary revascularisation procedures (i.e. the era of percutaneous coronary intervention), effective preventive drugs, in particular statins and antiplatelet therapy, and lifestyle measures\textsuperscript{51,53}. Together with an aging population\textsuperscript{54}, these factors have increased substantially the number of patients in need of optimal secondary prevention\textsuperscript{55}.

The risk of subsequent events in patients with established CHD remains high. In a representative Swedish registry study of nearly 100 000 patients with MI who were alive 1 week after discharge, subsequent cardiovascular events and death occurred in 18\% during the first year\textsuperscript{56}. For patients without an event during the first 365 days, 20\% had a new event in the following three years\textsuperscript{56}. Twenty-eight percent of the registered MIs in 2013 Norway were subsequent events\textsuperscript{10}. During the five-year follow-up period of CHD patients participating in the Global Registry of Acute Coronary Events\textsuperscript{57}, 13\% died due to CVD, 9\% suffered a new MI, 8\% suffered a stroke, whereas 17\% needed subsequent revascularization procedures. The rehospitalization rate was, on average, 1.6 per patient\textsuperscript{57}. The one-year risk of subsequent cardiovascular events was also inversely related to risk factor control in the large Reduction of Atherothrombosis for Continued Health (REACH) Registry\textsuperscript{58}. Trends in MI event rates and risk of recurrences after an incident coronary event in Norway have been described using data from the Cardiovascular Disease in Norway (CVD-NOR) Project\textsuperscript{59}, and even though the risk of recurrent MI has declined in patients older than 65 years between 2001 and 2008, it is concerning that no decline was observed in younger patients\textsuperscript{52}. The reduction in CHD mortality has also been more prominent in older (>65 years) patients, and the mortality among younger women has actually increased\textsuperscript{60}. Observations from another large population-based Norwegian cohort also revealed that the incidence of MI in patients <80 years increased in women\textsuperscript{61}. Whether the positive trend in cardiovascular epidemiology is changing
remains unknown, but these data underscore the importance of CHD management and monitoring today.\textsuperscript{1,17,62}

1.4 Coronary risk factor control in clinical practice

Even though preventive guidelines for more than 20 years have documented the extensive scientific evidence for the benefit of optimal secondary prevention on cardiac prognosis,\textsuperscript{48} lifestyle and risk factor management in clinical practice is far from optimal.\textsuperscript{1,63,64} Over a ten-year period from the mid-1990s, the proportion of European CHD patients with obesity increases by 13\% and with diabetes increased by 11\%, while the proportion of daily smokers and patients with elevated BP remained nearly unchanged.\textsuperscript{64} Only cholesterol management has improved due to the increased prescription of statins.\textsuperscript{64} The recently conducted EuroAspire IV study documented unhealthy lifestyle in European CHD patients as reflected in a high prevalence of daily smoking (16\%), physical inactivity (60\%), and obesity (38\%).\textsuperscript{63} Furthermore, diabetes (27\%) was prevalent and a large proportion did not meet the recommend treatment goals\textsuperscript{1} for BP (43\%) and LDL cholesterol (81\%) despite a high use of medication.\textsuperscript{63} Corresponding findings have been reported in other large-scale multi-centre studies like REACH\textsuperscript{13} and Clarify.\textsuperscript{65} Several multi-centre studies have documented large diversities in risk factors control between regions globally and in Europe.\textsuperscript{13,63,65-67} Norway did not participate in the aforementioned multi-centre studies, and national data on risk factor control in CHD patients are lacking. Furthermore, the patient selection to the aforementioned studies may potentially be a matter of concern. The patient inclusion in the EuroAspire studies was conducted mainly by academic centres, with potentially better preventive management than in general cardiac practice.\textsuperscript{68} Moreover, the average interview rate in the EuroAspire IV study was only 49\%, and the study non-participants had, most likely, even poorer risk factor control.\textsuperscript{63} In REACH\textsuperscript{69} and Clarify\textsuperscript{65}, patient identification and inclusion were conducted at outpatient clinics, often specialist centres, and patients attending them therefore may have been more concerned about their health. Thus, the prevalence estimates in these large-scale studies may overestimate risk factor control, and consequently the quality of secondary prevention compared to the reality in everyday clinical practice.

1.5 Cardiac rehabilitation in coronary prevention

Cardiac rehabilitation (CR) can be viewed as the clinical application of secondary preventive care and covers all professional activities, structured support and counselling that aim to assist the patients in adopting healthy lifestyle changes, adhere to medication, and restore or improve mental and functional capacity after a CHD event.\textsuperscript{55} CR plays a pivotal role in short and long-term risk reduction and care in CHD patients and is recommended with the highest level of evidence.\textsuperscript{1,55} CR comprises a large number of preventive activities ranging from hospital-based programmes to community and society-based programmes depending on the organization of the national and local healthcare system.\textsuperscript{70} In Norway and most Western European countries, different hospital based outpatient programmes are available during the first 6-12 months following hospitalisation for CHD events, whereas general practitioners (GPs) are the key actors coordinating and providing long-term management.\textsuperscript{1} Thus, GPs are essential, but often unrecognised members of the extended CR team.\textsuperscript{71}
In the 1940s, strict bed rest was recommended for 6 weeks following MI. This was gradually reduced to a few days during the 1970s as studies documented that resuming physical activity within one week after an uncomplicated MI was safe. Several exercise programmes for CHD patients were developed during the 1970s and 1980s, among them Wenger’s four phased rehabilitation programme. Physical activity has been the cornerstone of the CR programmes, but more comprehensive programmes have gradually been developed. Modern multidisciplinary CR programmes should involve several components, including physical exercise, smoking cessation, medical treatment of BP, LDL cholesterol and diabetes, along with education, patient information, and attention to psychosocial problems. A Cochrane review on exercise-based CR found favourable effects with reduction in CV and all-cause mortality and hospital admissions, but no effect on the risk of MI or revascularization. However, the randomized clinical trials (RCT) in this review included predominately middle-aged men with low risk, and, in addition, had several limitations. A review addressing lifestyle modification programmes in the era of modern CHD treatment found an effect on both mortality, subsequent events, and risk factor control. Even though CR following a CHD event has a class 1 recommendation, less than half of eligible CHD patients attend CR across Europe, and a substantial proportion of those attending do not complete the programmes. The attendance rate has not improved over the last decades and is lowest in women and high risk sub-groups such as the elderly, ethnic minorities and those with comorbidities. The recent introduction of alternative CR programmes, including tele-rehabilitation, home-based programmes, family based and nurse led programmes, may be promising initiatives.

1.6 Barriers of secondary prevention of CHD

The reasons for unfavourable lifestyle behaviour and the low achievement of treatment goals for BP, LDL-cholesterol, and diabetes are complex and multifactorial. The factors are often categorised as related to the patient, the treatment, the healthcare provider, and healthcare system. Examples of patient factors are demographic background, socio-economic status, social support, factors related to the CHD and other somatic comorbidities, psychological distress, motivation, illness and risk perception, complex treatment and side-effects, the frequency, duration and structure of CR, and other secondary preventive care measures, the patient-provider communication, lack of guideline knowledge and implementations are examples of healthcare and system factors of relevance. Identification of potentially modifiable factors of importance for risk profile and prognosis remains a public health priority and is important for individualizing secondary preventive treatment in clinical practice and for designing successful long-term preventive interventions that further reduce the burden of CHD.

1.6.1 Psychosocial factors, CHD and coronary risk factors

Comorbid psychosocial distress is prevalent after CHD events. The rate of clinically significant depressive symptoms has been estimated to be 40-65%, while 15-25% meet the criteria for major depression. The prevalence of anxiety symptoms has been estimated to be 25-40%, while type D (i.e. distressed) personality is estimated to be 18-28%. In observational studies, depression, anxiety, type D personality, insomnia, vital exhaustion, hostility and lack of social
support increase the risk of coronary events and deteriorate the prognosis and quality of life of patients with established CHD. The mechanisms by which psychosocial factors influence prognosis in CHD patients are not completely understood, but it is hypothesized to be mediated through both direct pathophysiological (i.e. autonomic nervous system dysfunction, dysregulation in the hypothalamic-pituitary-adrenal axis, inflammatory dysregulation) and bio-behavioural (i.e. unhealthy lifestyle, low adherence with medication, low participation in CR) pathways. Great variations in levels of psychosocial distress by socio-demographic and somatic background factors make the picture even more complex. To our knowledge, the relative importance of psychosocial factors to medical and socio-demographic factors in association with each established coronary risk factor not at target has not been studied. Further knowledge about these associations may be useful in the development of more effective tailored secondary prevention programmes.

1.7 Smoking and CHD
Smoking is the leading avoidable cause of death in the developed world. Smokers lose on average 10 years of life compared to never-smokers and smoking increases the risk of total mortality by up to 50%. About 25% of the mortality risk is due to CHD, while lung cancer, chronic obstructive lung disease, and other malignant, respiratory and cardiovascular diseases account for the rest. The mean age at first CHD event is approximately 10 years younger in smokers compared to non-smokers. Smoking has a negative influence on endothelial and platelet function, fibrinolysis, inflammation, lipids, and vasomotor function and increases the risk of coronary events by several pathological mechanisms that lead to the development of atherosclerosis and thrombus formation. Smoking cessation reduces the risk of mortality and subsequent CHD events by more than one third. The effect of smoking cessation is found to be more effective than controlling all other traditional cardiovascular risk factors and remains the single most important risk factor to modify in order to improve prognosis in CHD patients.

The percentage of daily smokers in the general population has declined significantly in Europe and the US the past decades, while it has increased in less-developed parts of the world. Even though the percentage of smokers has declined globally from 41% in 1980 to 31% in 2012, the total number of smokers has increased by 250 million due to population growth. Moreover, the decline in daily smoking has been slower in the youngest age-groups, raising concern as to whether the decline in smoking rates will stagnate. The reduction in daily smoking in US CHD patients was only 5% from 1980-2000, compared to 12% in the general population. Only a modest reduction in smoking from 20-16% over the past 20 years has been observed in the EuroAspire studies. The prevalence of daily smoking has actually increased among the youngest CHD patients. Whether this observation is partly explained by successful primary preventive strategies (i.e. those who persist smoking suffer a CHD events) remains unknown.

Motivational interview techniques, other cognitive behavioural therapies and pharmacological aids increase the likelihood of smoking cessation when tested in randomized studies. Behavioural interventions and drug therapy are therefore recommended with the highest level of evidence in guidelines and are more efficient when used together. Still, only about half of the CHD patients across Europe manage to quit smoking after a coronary event, and relapse rates are
high\textsuperscript{112}. A large number of factors influence smoking behaviour\textsuperscript{1,87,110,111,121-123}. Low levels of smoking cessation interventions and use of pharmacological aids in clinical practice have been described in CHD patients\textsuperscript{94,120,124}. Motivation is also essential for smoking cessation and hospitalization for a coronary event is seemingly a good opportunity to motivate patients\textsuperscript{1,122}. Long smoking duration\textsuperscript{125,126} and nicotine dependency\textsuperscript{111,121} are other examples of reported barriers for smoking cessation. In most countries smoking cessation has been less pronounced in groups with low socio-economic status\textsuperscript{110,127}, which has also been inversely related to smoking cessation in CHD patients\textsuperscript{85,126}. Smoking is thus an important factor in explaining the social inequalities in health\textsuperscript{1,110,128}. Psychosocial factors, in particular depression, but also anxiety and type D personality have been associated with the risk of daily smoking in CHD patients\textsuperscript{126,129-132}. In other studies, however, the association between psychosocial factors and smoking disappeared after adjusting for socio-demographic factors\textsuperscript{133}.

Even though several studies have explored some of the abovementioned factors associated with smoking behaviour, only a few studies from routine clinical practice have estimated the relative influence of socio-demographic, clinical, and psychosocial factors for persistent smoking after coronary events. Furthermore, most studies are limited to a three to six months follow-up period\textsuperscript{129}. Identifying modifiable factors associated with long-term smoking behaviour is crucial both for clinicians and for the development of novel cessation interventions for this specific population\textsuperscript{1,7,117}.

1.8 Blood pressure and CHD

Hypertension is the single risk factor that counts for the highest overall mortality and morbidity globally\textsuperscript{134}. Hypertension is not only an important risk factor the development\textsuperscript{1,135,136} and progression of CHD\textsuperscript{28,136}, but also a major risk factor for the development of heart failure\textsuperscript{28,136}, renal failure\textsuperscript{136,137}, stroke\textsuperscript{28,135,136}, and dementia\textsuperscript{137}. The guidelines for CHD prevention recommend a BP goal of <140/90 mmHg in patients with established CHD, and <140/80 mmHg in those with CHD and diabetes\textsuperscript{1}. Healthy lifestyle changes including weight reduction\textsuperscript{138,139}, diet modifications\textsuperscript{138,140}, physical exercise\textsuperscript{140,141} and salt restriction\textsuperscript{140,142} are found to improve BP control in general, but this has not been carefully studied in CHD patients. A meta-analysis of randomized trials of BP lowering drugs showed that lowering systolic BP by 10 mmHg led to a 25 % reduction of CHD events and a 38% reduction of stroke\textsuperscript{28}. The effect was similar in patients with or without pre-existing CVD\textsuperscript{28}. Despite documented benefits of BP reduction and a large number of effective BP lowering drug classes, the awareness of the BP targets and implementation of guideline recommendations among healthcare providers is limited\textsuperscript{96,99}, and more than 40% of the CHD patients in the latest EuroAspire study had unfavourable BP control\textsuperscript{65}. In the Clarify Registry, the frequency of unfavourably elevated BP ranged from 28-48% depending on the region\textsuperscript{65}. Older age, obesity, diabetes, and hypercholesterolemia are established interrelated risk factors associated with hypertension in patients with and without CHD\textsuperscript{143-145}. Suboptimal prescription of BP lowering drugs\textsuperscript{99,146,147} and, particularly, medication non-adherence (i.e. that the patients do not take their medications as prescribed by their healthcare providers) are more recently recognized factors that influence BP control\textsuperscript{148-150} and prognosis\textsuperscript{151}. The prevalence of hypertension has been shown to decline with higher level of education\textsuperscript{85}, while the association with psychosocial factors has been less clear\textsuperscript{152}. In a large
Norwegian population-based cohort both depression and anxiety were independently associated with lower BP during 11 years of follow-up. Further insight into the relative contribution of socio-demographic, medical and psychosocial determinants of BP is therefore needed.

1.9 Summary of background and basis for the thesis

CHD remains the leading global cause of mortality, morbidity and DALYs. In addition, treatment of CHD and its complications places a significant economic burden on the healthcare system. Serially conducted international prevalence studies have revealed substantial challenges in secondary prevention and poor risk factor control. At present, data on coronary risk factor control in CHD patients are not available for the Norwegian population. Patient selection and low participation rates in international multi-centre studies underscore the need for national prevalence estimates on adherence to secondary prevention.

A complex array of patient and healthcare factors influences control of established risk factors like smoking and elevated BP, after CHD events. Large scale registry studies have included a large number of CHD patients at the expense of collecting less data per patient. To gain greater insight into the complex interactions of mediators that influence risk factor control, comprehensive interdisciplinary data sets are needed.

The NORwegian CORonary (NOR-COR) Prevention project identifies socio-demographic, medical, and psychosocial factors (comprising the study factors) associated with unfavourable risk factor control and prognosis after a coronary event, in a cohort representing routine clinical practice (phase I) (Figure 1). The present PhD thesis will focus on the risk factors smoking and elevated BP. Defining the relative importance of the study factors for control of each of the major risk factors, and their predictive power with respect to subsequent cardiovascular events, will provide a better understanding of putative bio-behavioural mechanisms linking clinical and psychosocial factors to cardiovascular prognosis in CHD patients.
In analysis of factors associated with a given coronary risk factor, the other coronary risk factors will be included in the analysis as study factors.

2. Aims of the thesis

The main objective of the present PhD thesis, an integrated part of the NOR-COR project, is to determine the degree of control of the major coronary risk factors after a coronary event, and to identify the study factors associated with persistent smoking and unfavourable BP control.

More specifically we aim to:

1. Determine the control of the six major coronary risk factors smoking, physical inactivity, obesity, BP, LDL cholesterol, and blood glucose according to target recommendation in current guidelines 1–2-36 months after a coronary event, and to identify the influence of age, gender, number of coronary events, and time since the index event.

2. Explore the socio-demographic, medical, psychosocial factors associated with persistent smoking after a coronary event.

3. Explore the socio-demographic, medical, psychosocial factors associated with unfavourable BP control after a coronary event.

3. Hypotheses

The following hypotheses will be elucidated in the present PhD thesis:

1. The prevalence of unfavourable risk factor control in routine clinical practice in Norway is higher than reported in European studies with patient recruitment mainly from academic centres.

2. By using the comprehensive NOR-COR data set, it is possible to identify potentially modifiable medical and psychosocial factors associated with persistent smoking and BP control.
4. Methods

4.1 Design
This is a cross-sectional study with a retrospective component as illustrated in Figure 2.

Figure 2. The design of study phase I

4.2 Study population and material
The study was conducted at two Norwegian hospitals, Drammen and Vestfold, located in the eastern part of Norway. The total catchment area is 380,000 inhabitants from the southeast part of Buskerud County and Vestfold County corresponding to 7.4% of the Norwegian population. The catchment area has a blend of city and rural districts and is fairly representative of Norway in terms of geography, economy, age distribution, morbidity and mortality. Both participating hospitals provide a cardiac rehabilitation programme, but the local referral routines and the content and duration of these programmes differ significantly. Drammen hospital offers a multidisciplinary one day “heart school” at the outpatient clinic, and a six-week exercise program with two sessions per week according to the Ullevål model. The Hospital of Vestfold provides a comprehensive multidisciplinary cardiac rehabilitation programme lasting for up to six months with lifestyle intervention, patient education, and physical exercise. The program has been previously tested in a randomized trial as is described elsewhere.

The study inclusion criteria were: age 18-80 years and hospitalisation for a coronary event 2-36 months prior to study inclusion. The coronary index event was defined as a first or recurrent acute myocardial infarction and/or revascularisation treatment with coronary artery bypass grafting operation (CABG) or acute or elective percutaneous coronary intervention (PCI). The index event was defined as the latest event recorded prior to the time of study inclusion. Study exclusion criteria were: i) a diagnosis of type 2 myocardial infarction, ii) not being able to understand the Norwegian language, iii) cognitive impairment including living in nursing homes, iv) psychosis, v) alcohol and drug abuse, and/or vi) short life expectancy due to terminal heart (NYHA class 4), lung-, liver- or kidney disease (stage 5), or malignant disease.
4.3 Inclusion procedure

To identify eligible study patients, we screened the hospital discharge lists chronologically for the diagnosis of myocardial infarction (ICD-10 diagnosis I21 and I22), angina pectoris (ICD-10 diagnosis I20) or CHD (ICD-diagnosis I25.1) in last three years (2011-14) prior to study inclusion. We identified 1789 patients eligible for inclusion. After screening hospital records, we excluded 423 patients due to cognitive impairment (n=28), psychosis (n=18), active alcohol and/or drug abuse (n=10), short life expectancy (n=136), dead (n=160), not being able to understand Norwegian (n=44), or other reasons (n=27). The remaining 1366 patients who fulfilled the inclusion criteria were mailed a letter with study information, the comprehensive self-report questionnaire (see Appendix 2), and an appointment for the clinical examination and collection of the venous blood samples. All the blood samples were analysed at the laboratory at Drammen Hospital to avoid inter laboratory bias. In all, 1127 (n=580 patients from Drammen [82% participation rate] and n=547 patients [85% participation rate] from Vestfold,) patients (aged 31 to 80 [mean 62] years, 21% women, 3.4% with ethnic minority background) consented to participate in the study. All participants completed the questionnaire² and attended a clinical examination with blood sample collection 2-36 (median 16) months after the index event. In addition, hospital records data from index event were registered.

The 239 patients who refused study participation were asked to register hospital record data from the index event. Only ten patients (0.7%) actively refused data registration and the remaining 229 patients were included in the non-participant study. A study flow-chart is presented in Figure 3.

Figure 3. Study flow chart

Cohort Vestfold and Drammen (catchment of 380 000), n = 1789 CHD patients with acute myocardial infarction; percutaneous coronary intervention; coronary artery by-pass graft operation

Excluded, n = 423

Eligible for inclusion, n = 1366

Study non-participants, n=239

Included in the study (study participants), n = 1127 (83%)

Included in the non-participant study, allowed use of hospital record data, n = 229 (16%)

Refused non-participant study, n = 10 (0.7%)
4.4 Study assessment

4.4.1 Development of the study questionnaire
The NOR-COR questionnaire contains 249 questions derived from a number of socio-demographic, medical and psychosocial instruments that have previously, to some extent, been demonstrated to be associated with coronary risk factors, adherence to medication, and prognosis in population-based studies or studies in CVD patients. Moreover, the instruments have been selected and developed based on the expertise of the interdisciplinary research group. We used already validated questionnaires when available and created de novo questions when not available. The questionnaire was developed following an extensive process as recommended by Perk et al.\textsuperscript{94}. The first set of questions was prepared and revised by the interdisciplinary research group. After two revisions, two cardiac nurses and two CHD patients contributed further valuable comments, the latter in order to incorporate the patient perspective in all questions (“patient research partners”). A third version was tested in 20 randomly selected CHD patients admitted for an acute coronary event to estimate relevance, acceptance and feasibility. All patients understood and replied to all the items and accepted the questionnaire. A few minor changes were made before the questionnaire was approved by the research group. The development of the questionnaire is described in detail in the design and method paper\textsuperscript{7}. The reproducibility of the questionnaire has been tested in an independent study\textsuperscript{158}, where the test-retest reliability of the questionnaire was estimated after 4 weeks in 99 CHD patients. The completion rate was excellent and the reproducibility was highly acceptable for all key items and instruments. Reproducibility values for questions in the first part of the questionnaire did not differ from those in the last part. The Cronbach $\alpha$ values ranged (0.69-0.95) for most questionnaires\textsuperscript{158} and were comparable to previous studies\textsuperscript{159-162}.

4.4.2 Primary outcome variables: the major coronary risk factors

- **Smoking**: Smoking status at the index event was retrieved from the hospital medical records. Smoking status at follow-up was recorded from the self-report questionnaire. Smoking status was categorized as: never smoker (i.e. registered as non-smoker at the index event and reporting never smoking at follow-up), former smoker (registered as non-smoker or previous smoker at the index event and reporting previous smoking at follow-up), quitter (registered as daily smoker at the index event and previous smoker at follow-up), and persistent smoker (all patients who were smokers at the index event and reported daily smoking at follow-up). The primary outcome variable was smoking status at follow-up compared to smoking status at the index event. Data on cessation attempts and relapse between the index event and follow-up is not available.

- **Blood pressure**: BP was measured by trained cardiac nurses to the nearest 1.0 mmHg after the patient had been sitting for at least 4 minutes. A validated digital sphygmomanometer (Welch Allyn WA Connex ProBP 3400) was used\textsuperscript{163}. The lower cuff margin was placed 2 cm above the cubital fossa and two cuff sizes were available depending on the patient’s upper arm circumference. Three measurements with a 1-minute interval on a single occasion were performed\textsuperscript{2,137} and the mean of the last two measurements was calculated. Unfavourable BP control was defined as BP >140/90 mmHg (>140/80 mmHg in diabetics)\textsuperscript{1,2}. Systolic BP was also analysed continuously.
• **Low density lipoprotein (LDL) cholesterol**: LDL-cholesterol was analysed (Architect ci16200, Abbott Laboratories, Abbott Park, Illinois, USA) in non-fasting venous whole blood sampled in an EDTA-tube. Unfavourable LDL-cholesterol was defined as >1.8 mmol/l.

• **Overweight and obesity**: Height (nearest 0.5 cm) was measured using a wall-fixated mechanical measuring rod (SECA 264, DE). Body weight (nearest 0.5 kg) was measured in light clothes without shoes (SECA 813, DE). Overweight was defined as body mass index (BMI) >25 kg/m² and obesity as BMI >30kg/m². Waist circumference was measured with a non-stretchable tape (SECA 201, DE). A waist circumference >94 cm in men and >80 cm in woman was defined as central overweight and a circumference >102 cm in men and >88 cm in women was defined as central obesity.

• **Physical activity**: Physical activity was recorded from the self-report and validated instrument. Frequency (never, <1 time weekly, 1 time weekly, 2-3 times weekly and almost every day), intensity (light, medium and vigorous), and duration (<15minutes, 15-29 minutes, 30-60 minutes and >60 minutes) were reported. Low physical activity was defined as less than moderate activity level for 30 minutes 2-3 times a week. No physical activity was defined as physical activity less than once weekly.

• **Blood-sugar**: Blood sugar was assessed by haemoglobin A1c (HbA1c), analysed (Tosoh G8, Tosoh Medics Inc, San Francisco, CA, US) in venous whole blood sampled in an EDTA-tube with patients non-fasting. Unfavourable blood sugar control was defined as HbA1c ≥6.1% (non-diabetics) and >7.0% (diabetics).

4.4.3 Covariates (the NOR-COR study factors)
The covariates are listed below. When smoking status (paper II) or blood pressure (paper III) were the outcome variables, the remaining five risk factors listed above were included as covariates, as indicated according to our model (see also Figure 1).

4.4.3.1 Data collected from the hospital medical records at the time of the index event were categorized as:

• Socio-demographic factors:
  o Patient’s age in 1.0 years.
  o Gender: male/female.
  o Ethnicity: ethnic minorities defined as 1st and 2nd generation patients from Asia, Africa and South America.
  o Time since the index event was calculated from the time of the index event and time of inclusion (2-36 months).

• Medical factors:
  o Type of acute coronary event: Defined as ST-elevation myocardial infarction (STEMI), non-ST elevation infarction (NSTEMI) or stable or unstable angina pectoris.
  o Angiographic findings: Categorized as open vessels, atherosclerosis without significant stenosis, single and multi-vessel disease
  o Intervention: No intervention, percutaneous coronary intervention (PCI) or coronary artery bypass graft operation (CABG).
Somatic medication registered in the hospital discharge letter: Anti-platelets (aspirin, clopidogrel, ticagrelor, prasugrel), statins, BP lowering drugs, nitrates, oral anticoagulants, insulin, oral anti-diabetics.

Somatic comorbidity: Number of coronary events, heart failure, atrial fibrillation, stroke/transitory ischemic attack, peripheral artery disease, chronic obstructive pulmonary disorder, kidney failure, liver failure, stomach ulcers, inflammatory and rheumatic conditions were registered. Somatic comorbidity was also summarized according to the Charlson comorbidity index.165

Current or previous participation in the hospital based cardiac rehabilitation programmes was confirmed by the medical records and separate lists from the cardiac rehabilitation departments.

- **Psychosocial factors:**
  - Psychiatric variables: A diagnosis of anxiety, or depressive disorder, and/or treatment with anti-depressive medication recorded in the discharge letter.

4.4.3.2 Data collected from the self-report questionnaire at the follow-up visit:

- **Socio-demographic factors:**
  - Marital status: Defined as living with a partner or living alone.
  - Level of education: Low education was defined as completion of primary or secondary school only.
  - Employment status: Patients under the age of retirement were defined as either being employed or receiving social or disability benefits.

- **Medical factors:**
  - Provision of nicotine replacement therapy or other smoking cessation aids.
  - Perceived need for further help with smoking cessation (yes, no, maybe).
  - Diet: The frequency or amount of intake of different types of foods, beverages, and meals were recorded using a validated 10-item instrument.166
  - Medication at follow-up: Anti-platelets, statins, angiotensin converting enzyme inhibitors (ACEI) / angiotensin receptor blocker (ARB), beta-blockers.
  - Adherence to medication: The 8-item Morisky Medication Adherence Scale.148 Medium adherence was defined by a score >1 point and low adherence by a score of >2 point.
  - Side-effects of cardiovascular drugs were reported dichotomously (yes/no). Those who answered “yes” were then asked to describe their subjective side-effects qualitatively.
  - The number of follow-up visits in general practice the past 12 months.

- **Psychosocial factors:**
  - Anxiety and depression: The Hospital Anxiety and Depression Scale (HADS) contain 14 item covering symptoms of anxiety (HADS-A) and depression (HADS-D). It focuses on affective and cognitive symptoms and there are no somatic symptoms. Cut-off scores ≥8 on each subscale define doubtful cases and ≥11 define definite cases.167
o Worry: Penn State Worry questionnaire\textsuperscript{162}, contains 16 items measuring pathological worry.
o Insomnia: Bergen Insomnia Scale\textsuperscript{161}, contains 6 items about sleep onset, maintenance of sleep and early morning wakening.
o Type D personality: DS-14 questionnaire\textsuperscript{160}, contains 14 items, with 7 items each on subscales of negative affectivity and social inhibition. To be categorized with type D personality a score $\geq$10 points on both subscales is required.
o Illness perception: Brief illness perception questionnaire\textsuperscript{168}, contains nine items assessing the cognitive and emotional representations of illness (specified as CHD illness in the questionnaire) on a 1-10 Likert scale.
o Perceived risk perception\textsuperscript{95}, contains 3 items covering the patients perception of future cardiac risk assessed by a 1-10 Likert scale.
o Patient’s perception and understanding of perceived information: Four items assessing whether patients perceive themselves to be cured of their CHD and were in need of lifestyle change\textsuperscript{94}.
o Motivation for smoking cessation: Assessed by a 1-10 Likert scale.
o Beliefs regarding what caused the CHD: In all, 13 risk factors for CHD (age, genetic load, smoking, stress, blood pressure etc.) were rated to what extent they had caused the CHD on a 1 - 10 Likert scale.

The socio-demographic variables, somatic comorbidity including coronary history and treatment are descriptive factors, while the remaining medical and psychosocial factors are regarded as potentially modifiable\textsuperscript{7}.

4.5 Ethics and study approvals
The NOR-COR study was approved by the Regional Committee for Medical Research Ethics (REK Sør-Øst) February 12\textsuperscript{th}, 2014 (2013/1885). All participants gave informed consent before study participation. The Study is registered at www.ClinicalTrials.gov (IDNCT02309255). The study has been conducted in accordance with ethical principles that have their origin in the Declaration of Helsinki\textsuperscript{169}.

4.6 Statistical analyses
Epidemiology has been defined as the study of occurrence and distribution of health-related events in specific populations\textsuperscript{19}. Epidemiology includes the study of the determinants influencing such states and the use of this knowledge to control health problems\textsuperscript{19}. Regression models such as linear or logistic regression analyses are important and useful tools in epidemiological research to investigate the role of putative causal factors. The models can be used to explore the nature of the association between exposure (e.g. the NOR-COR factors) and outcome (i.e. the established risk factors) by controlling for confounders, mediators or effect-modifiers that can influence this association\textsuperscript{34}. All statistical analyses in this thesis have been performed using SPSS version 21 (SPSS Inc., Chicago State, USA).
4.6.1 Sample size calculations
Sample size calculations were made to have sufficient power to analyse composite CV endpoints (MI, CV mortality, need of revascularization) at follow-up after 5 years according to patient groups and study factors. We also differences within the participant group with respect to psychosocial risk factors such as HADS (assuming a 20% rate of positives) to be detected with 90% power for binary outcomes when there is a between-group difference of 10% and the overall level is 15% (even with continuity correction). Our calculations indicated that 400 cases of subsequent events would be sufficient. The incidence of the composite cardiovascular endpoint was 35% after 4 years follow-up in CHD patients participating in the REACH study. Therefore, 1100 patients were considered sufficient. The prevalence of the coronary risk factors ranged 16-80% in the EuroAspire IV Study. Given an equal distribution of risk factors in our study, a sample size of 1000 patients would allow for the inclusion of a considerable number of covariates (k~15) when the least prevalent risk factor (n=160) was used as a dichotomous outcome variable.

4.6.2 Descriptive statistics
Descriptive statistics have been used in all three papers to describe the study population. Continuous variables are presented as means with standard deviations, and dichotomous variables as frequencies and percentages. We applied the Student’s t-test to compare mean differences between groups for continuous variables and the $\chi^2$ test to compare proportions.

4.6.3 Logistic regression analyses
A binary logistic regression analysis can be used to estimate the extent to which the proportion of subjects with a given outcome for a dichotomous dependent variable Y (for example disease yes/no) increases or decreases with a unit change in continuous or categorical independents (X), commonly expressed as odds ratios (OR). To express the level of uncertainty in the estimate one usually reports 95% confidence intervals and/or p-values set to .05. The logistic regression model is robust as independents do not have to be normally distributed and equal variances within and between subgroups of the independents (homoscedastity) is not strictly assumed. Procedures for selecting the subset of the eligible independents for which all effects (ORs) are statistically significant include stepwise forward inclusion (successively including individual predictors that contribute significantly given the set that has already been selected) and backward stepwise elimination (successively removing the least significant predictor from a broadly defined set of eligible predictors).

Binary logistic regression analyses have been used in all three papers:

- Paper I: Binary logistic regression analyses were used to calculate ORs for unfavourable risk factor control adjusted for age, gender, number of coronary events, and time since the index event.
- Paper II: A forward, stepwise binary logistic regression analysis was used to calculate crude and multi-adjusted OR for smoking status at follow-up (quitter vs. persistent smoker). Interaction terms between independent variables were assessed and included when indicated. The level of significance was set to $p<0.05$. We also included covariates with p-values.
between 0.05 and 0.1 in crude analyses in the multivariate models since more traditional levels (i.e. 0.05) may fail to identify variables that might turn out to be significant when actually included in the adjusted models (due to so-called statistical suppression). A backward stepwise elimination procedure was also tested without significantly different result.

- Paper III: A binary logistic regression analysis was used to calculate crude OR and 95% confidence interval for BP ≥/< 140/90 (140/80 in diabetics) mmHg at follow-up. We used a backwards stepwise elimination procedure to fit a multivariable binary logistic model starting with the variables showing a p-value ≤0.1 in crude analyses. Age and sex were nevertheless forced into the final model regardless of their level of significance on the assumption that they were putative confounders.

4.6.4 Linear regression analyses
Linear regression assumes that the relationship between an outcome variable Y and the independent determinants X_i, X_{ii} etc. can be summarized as a straight line, where the (least squares) line is represented by two numbers, viz. the intercept with the y-axis and the slope of the line\(^\text{171}\). We applied a multiple linear regression analysis in paper III in order to explore the relative contribution of the various study factors on systolic BP. Residual Q-Q plots and histogram for systolic BP and covariates were visually checked and fitted the normal distribution excellently. A backwards stepwise elimination procedure was used to fit a linear regression model starting with the variables with p-values ≤0.1 in the crude analyses. Age and sex were forced into the model on a priori assumption that they were putative confounders.

4.6.5 General Linear Models (analysis of covariance)
Analysis of covariance (ANCOVA) is an example of a general linear model (based on Ordinary Least Squares (OLS)). The analysis is a mixture of regression analysis and analysis of variance (ANOVA). An ANCOVA evaluates if the mean for an outcome (dependent variable) is different between levels of independent categorical variables (“factors”) while controlling for the effects of continuous covariates that usually are not of primary interest. In paper I, we used an ANCOVA to estimate the marginal means of the total number of unfavourable risk factors (smoking, BMI, physical inactivity, BP, LDL cholesterol, and HbA1c) by the independent variables age, gender and number of coronary events entered simultaneously as dummies (factors), using time since event as a linear covariate.

4.6.6 Missing data
The frequency of missing values for the questionnaire based and clinical data was low, within the range of 0-10%.
5. Results

5.1 Paper I. Unfavourable risk factor control after coronary events in routine clinical practice

Mean age was 62 (SD 10) years at follow-up and 21% of patients were women. The index event was myocardial infarction in 80% of the patients and angina with angiographically verified coronary stenosis was present in 20% of the patients. All but one patient had an angiography performed, and 90% were revascularized. Thirty percent of the patients had >1 coronary event, and diabetes was twice as prevalent in this group compared to patients with 1 coronary event only (28% vs. 12%, p<0.001).

Risk factor control at follow-up 2-36 (median 16) months after the index event was far from optimal, as is shown in Figure 4. Twenty-one percent of patients were current smokers at follow-up and 56% of those smoking at the index event continued smoking. Obesity was found in 34% of patients, while 60% had central obesity. Sixty percent reported less than 30 minutes moderate activity 2 to 3 times a week, while 18% reported physical activity less than once weekly. Although 93% were taking BP lowering drugs and statins, 46% were still hypertensive and 57% had LDL cholesterol >1.8mmol/L at follow-up. Fifty-nine percent of the diabetic patients had HbA1c >7.0%, while 35% had HbA1c >8.0%. Almost half of the patients reported eating fish less than 2 times a week, and two thirds ate fruit and vegetables less than twice daily.

Figure 4. Proportion of coronary risk factors 2-36 months after the index coronary event

*Less than moderate activity level for 30 minutes 2 to 3 times a week, ** Never or less than once a week. †Low density lipoprotein.
Information about the diagnosis of diabetes was obtained from the medical records. The estimated marginal means for number of unfavourable risk factors (smoking, BMI >25, physical activity <30 min of moderate activity 2-3 times weekly, BP >140/90 mmHg (140/80 in diabetic patients), LDL cholesterol >1.8 mmol/l and HbA1c>7%) by gender, age and number of coronary events are shown in Figure 5. The patients had on average three out of six risk factors not at target according to guideline recommendations. 62% had ≥3 risk factors not at target and only 2% had all risk factors at target. Patient with at least one previous coronary event (β 0.43, p < 0.001) had the poorest overall risk factor control.

Figure 5. Estimated marginal means* of number of coronary risk factors†

*Estimated by General Linear Model (GLM, i.e. ANCOVA) with age, sex and number of events controlled as dummies simultaneously and with time since event entered as a linear covariate.
†Unfavourable risk factors defined as current smoking, body mass index >25 kg/m², physical activity <30 minutes of moderate activity 2-3 times a week, blood pressure >140/90 (140/80 in diabetic patients), low density lipoprotein cholesterol >1.8 mmol/l, HbA1c >7.0 % in diabetic patients or HbA1c ≥6.1 % in non-diabetic patients.

In multi-adjusted analysis we found current smoking (p<0.001), obesity (p<0.001), and unfavourable HbA1c (p<0.01) to be more prevalent in the younger patients, while unfavourable BP control (p<0.001) was more prevalent in older patients. Women were more frequently current smokers (p<0.05), had low physical activity (p=0.001), and unfavourable LDL cholesterol (p<0.001) compared to men. Low physical activity (p<0.001), obesity (p<0.05) and unfavourable LDL cholesterol (p=0.001) were more frequent in patients with >1 coronary event than in patients with only 1 coronary event. We observed a linear increase in smoking (p<0.01) and obesity (p<0.05) with increasing time since the index event.
5.2 Paper II. Medical and socio-demographic factors predict persistent smoking after coronary events

Data on smoking status were available in 1083 of 1127 patients (96%). A total of 390 (36%) patients were smoking at the time of the index event. Those who smoked at the index event were younger, were more likely to be female, had lower education levels, were more often unemployed or on disability benefits and had more often STEMI as index event compared to never and former smokers. At follow-up, 167 (43%) patients smoking at the index event had quit smoking, while 230 patients reported persistent smoking. Data on persistent smokers and quitters are presented in Table 1. Seventy-three percent of the persistent smokers reported to have reduced their cigarette consumption since the index event. Almost all persistent smokers had a long (>20 years) history of smoking and 60% had been smoking for more than 40 years.

Persistent smokers rated use of tobacco as the most important cause of their coronary disease (6.8 on a 1-10 Likert scale). A majority (72%) reported a high (≥7/10 on the 1-10 Likert scale) motivation for smoking cessation, and 83% of these patients wanted help to quit smoking. In contrast 41% of those with moderate to low motivation (<7/10 on the 1-10 Likert scale) wanted help to quit smoking (p<0.001). Only 42% of the persistent smokers had been offered nicotine replacement therapy or other cessation aids, and 35% reported having no current follow-up for their CHD in primary or specialist healthcare.

In adjusted analyses (Table 2), unemployed or disability benefits (Odds ratio (OR) 4.1), low education (OR 3.5), longer smoking duration (OR 2.3), and not having STEMI as index event (OR 2.3) were significantly associated with persistent smoking. Psychosocial factors were not associated with persistent smoking at follow-up and these findings were consistent in sub-group analyses by age and gender.
Table 1. Socio-demographic, medical, and psychosocial factors in smokers and quitters at follow-up* after the coronary event

<table>
<thead>
<tr>
<th>Study factors</th>
<th>Quitted after the index event (n=167)</th>
<th>Persistent smokers (n=230)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Socio-demographic factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age at index event, mean (SD)</td>
<td>57.7 (9.4)</td>
<td>59.3 (9.3)</td>
<td>0.05</td>
</tr>
<tr>
<td>Number of months since the index event, mean (SD)</td>
<td>16.8 (10.9)</td>
<td>18.9 (10.8)</td>
<td>0.05</td>
</tr>
<tr>
<td>Female sex, n (%)</td>
<td>38 (22.8)</td>
<td>56 (24.3)</td>
<td>0.71</td>
</tr>
<tr>
<td>Ethnic minority background, n (%)</td>
<td>4 (2.4)</td>
<td>10 (4.3)</td>
<td>0.30</td>
</tr>
<tr>
<td>Living alone, n (%)</td>
<td>28 (16.8)</td>
<td>57 (24.8)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Low education, n (%)</td>
<td>116 (69.5)</td>
<td>189 (82.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Unemployed or on disability benefits, n (%)</td>
<td>33 (19.8)</td>
<td>85 (37.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Medical factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration of smoking, years, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20</td>
<td>11 (6.6)</td>
<td>8 (3.5)</td>
<td></td>
</tr>
<tr>
<td>20-39</td>
<td>99 (59.3)</td>
<td>64 (27.8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>&gt;40</td>
<td>53 (31.7)</td>
<td>137 (59.6)</td>
<td></td>
</tr>
<tr>
<td>ST-elevation infarction/non ST-elevation infarction and angina, n (%)</td>
<td>78 (46.7)</td>
<td>84 (36.5)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>More than 1 coronary event, n (%)</td>
<td>30 (18.0)</td>
<td>77 (33.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Participation in cardiac rehabilitation, n (%)</td>
<td>95 (56.9)</td>
<td>103 (44.8)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Charlson co-morbidity sum score, mean (SD)</td>
<td>3.8 (1.3)</td>
<td>4.0 (1.4)</td>
<td>0.14</td>
</tr>
<tr>
<td>Use of antiplatelets at follow-up, n (%)</td>
<td>164 (98.2)</td>
<td>221 (96.1)</td>
<td>0.22</td>
</tr>
<tr>
<td>Use of statins at follow-up, n (%)</td>
<td>160 (95.8)</td>
<td>207 (90.0)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Low physical activitya, n (%)</td>
<td>93 (55.7)</td>
<td>186 (80.9)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Body Mass Index &gt;30 kg/m², n (%)</td>
<td>61 (36.5)</td>
<td>58 (25.2)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Low density lipoprotein cholesterol &gt;1.8 mmol/l, n (%)</td>
<td>88 (52.7)</td>
<td>124 (53.9)</td>
<td>0.66</td>
</tr>
<tr>
<td>Blood pressure &gt;140/90 (140/80 diabetes) mmHg, n (%)</td>
<td>60 (35.9)</td>
<td>74 (32.2)</td>
<td>0.74</td>
</tr>
<tr>
<td>Diabetes, n (%)</td>
<td>16 (9.6)</td>
<td>36 (15.7)</td>
<td>0.08</td>
</tr>
<tr>
<td><strong>Psychosocial factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital Anxiety and Depression Score - depression ≥11, n (%)</td>
<td>12 (7.2)</td>
<td>13 (5.7)</td>
<td>0.61</td>
</tr>
<tr>
<td>Hospital Anxiety and Depression Score - anxiety ≥11, n (%)</td>
<td>22 (13.2)</td>
<td>27 (11.7)</td>
<td>0.81</td>
</tr>
<tr>
<td>Type D personality disorder, n (%)</td>
<td>41 (24.6)</td>
<td>56 (24.3)</td>
<td>0.89</td>
</tr>
<tr>
<td>Worry score (PSWQb), mean (SD)</td>
<td>40.1 (13.9)</td>
<td>40.3 (13.9)</td>
<td>0.87</td>
</tr>
<tr>
<td>Insomniac, n (%)</td>
<td>80 (47.9)</td>
<td>114 (49.6)</td>
<td>0.65</td>
</tr>
</tbody>
</table>

*2-36 months after the index coronary event

a Physical activity less than 30 minutes of moderate activity 2-3 times weekly

b Worry was assessed by the Penn State Worry Questionnaire (PSWQ), a 16 item measure of pathological worry

c Measured by Bergen insomnia Scale
<table>
<thead>
<tr>
<th>Study factors</th>
<th>Model 1 (OR, 95% CI)</th>
<th>p-value</th>
<th>Model 2 (OR, 95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Socio-demographic factors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean age at index event (OR per year)</td>
<td>1.02 (1.00-1.04)</td>
<td>p=0.05</td>
<td>0.97 (0.90-1.03)</td>
<td>p=0.27</td>
</tr>
<tr>
<td>Time since the index event (OR per year)</td>
<td>1.02 (1.00-1.04)</td>
<td>p=0.05</td>
<td>1.01 (0.98-1.05)</td>
<td>p=0.41</td>
</tr>
<tr>
<td>Female gender</td>
<td>1.09 (0.68-1.75)</td>
<td>p=0.71</td>
<td>2.17 (0.85-5.52)</td>
<td>p=0.10</td>
</tr>
<tr>
<td>Living alone</td>
<td>1.69 (1.01-2.82)</td>
<td>p&lt;0.05</td>
<td>1.23 (0.48-3.11)</td>
<td>p=0.67</td>
</tr>
<tr>
<td>Low education</td>
<td>2.20 (1.36-3.57)</td>
<td>p=0.001</td>
<td>3.35 (1.43-7.81)</td>
<td>p&lt;0.01</td>
</tr>
<tr>
<td>Unemployed or on disability benefits</td>
<td>3.01 (1.81-5.02)</td>
<td>p&lt;0.001</td>
<td>4.12 (1.80-9.41)</td>
<td>p=0.001</td>
</tr>
<tr>
<td><strong>Medical factors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not having ST-elevation infarction as index event</td>
<td>1.53 (1.02-2.29)</td>
<td>p&lt;0.05</td>
<td>2.30 (1.08-4.40)</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>More than 1 coronary event</td>
<td>2.30 (1.42-3.72)</td>
<td>p=0.001</td>
<td>1.53 (0.63-3.72)</td>
<td>p=0.35</td>
</tr>
<tr>
<td>Participation in cardiac rehabilitation</td>
<td>0.62 (0.41-0.92)</td>
<td>p&lt;0.05</td>
<td>0.78 (0.38-1.60)</td>
<td>p=0.50</td>
</tr>
<tr>
<td>Charlson comorbidity sum score</td>
<td>1.12 (0.96-1.32)</td>
<td>p=0.14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration of smoking (years)</td>
<td>2.93 (1.62-2.71)</td>
<td>p&lt;0.001</td>
<td>2.34 (1.41-3.88)</td>
<td>p=0.001</td>
</tr>
<tr>
<td><strong>Psychosocial factors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital Anxiety and Depression Score - total &gt;11</td>
<td>1.06 (0.70-1.62)</td>
<td>p=0.78</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type D personality</td>
<td>1.03 (0.65-1.65)</td>
<td>p=0.89</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Worry score (PSWQ&lt;sup&gt;c&lt;/sup&gt;)</td>
<td>1.00 (0.99-1.01)</td>
<td>p=0.87</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insomnia&lt;sup&gt;d&lt;/sup&gt;</td>
<td>1.10 (0.73-1.65)</td>
<td>p=0.65</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Perceived risk (1-10 Likert scale)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>What do you feel is the likelihood of having a new heart attack over the next 12 months?</td>
<td>1.15 (1.05-1.25)</td>
<td>p&lt;0.01</td>
<td>1.01 (0.86-1.18)</td>
<td>p=0.93</td>
</tr>
<tr>
<td>How much do you feel you can help reduce your risk of having another heart attack?</td>
<td>0.91 (0.84-0.99)</td>
<td>p&lt;0.05</td>
<td>0.88 (0.76-1.02)</td>
<td>p=0.09</td>
</tr>
<tr>
<td>How much do you think you will have to restrict your activities in the long-term due to your heart condition?</td>
<td>1.17 (1.08-1.27)</td>
<td>p&lt;0.001</td>
<td>1.00 (0.87-1.17)</td>
<td>p=0.90</td>
</tr>
<tr>
<td><strong>Brief Illness Perception Questionnaire (1-10 Likert scale)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>How much does your illness affect your life? (consequences)</td>
<td>0.99 (0.92-1.06)</td>
<td>p=0.76</td>
<td></td>
<td></td>
</tr>
<tr>
<td>How long do you think your illness will continue? (timeline)</td>
<td>0.98 (0.91-1.04)</td>
<td>p=0.45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>How much control do you feel you have over your illness? (personal control)</td>
<td>0.95 (0.88-1.02)</td>
<td>p=0.16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>How much do you think your treatment can help your illness? (treatment control)</td>
<td>0.80 (0.72-0.88)</td>
<td>p&lt;0.001</td>
<td>0.88 (0.75-1.02)</td>
<td>p=0.09</td>
</tr>
<tr>
<td>How much do you experience symptoms from your illness? (identity)</td>
<td>0.83 (0.92-1.07)</td>
<td>p=0.83</td>
<td></td>
<td></td>
</tr>
<tr>
<td>How concerned are you about your illness? (concern)</td>
<td>0.98 (0.93-1.06)</td>
<td>p=0.74</td>
<td></td>
<td></td>
</tr>
<tr>
<td>How well do you feel you understand your illness? (understanding)</td>
<td>0.97 (0.89-1.05)</td>
<td>p=0.41</td>
<td></td>
<td></td>
</tr>
<tr>
<td>How much does your illness affect you emotionally? (emotional response)</td>
<td>0.96 (0.90-1.02)</td>
<td>p=0.19</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Quitted smoking after the index event is the reference category

<sup>a</sup> Model 1, crude analyses,  
<sup>b</sup> Model 2, multi-adjusted with including all variables with p<0.1 in crude analysis (adjusted for all variables included in the model)  
<sup>c</sup> Worry was assessed by the Penn State Worry Questionnaire (PSWQ), a 16 item measure of pathological worry  
<sup>d</sup> Measured by Bergen insomnia Scale
5.3 Paper III. Optimal blood pressure control after coronary events: the challenge remains

Mean BP at follow-up was 138 (SD 19.0) / 81 (SD 8.7) mmHg, while 46% (N=457) patients had BP ≥140/90 (≥80 in diabetics) mmHg. Low socio-economic status, psychosocial factors, illness and risk perception did not predict unfavourable BP control. Neither did self-reported drug adherence or side-effect of BP lowering drugs. However, the sub-group of patients with low adherence reported more side-effects of BP lowering drugs (23% vs. 14%, p<0.05) and had a higher prevalence of depression (26.0% vs. 12.7%, p<0.001), insomnia (55.0% vs. 43.4%, p<0.05) and Type D personality (25.2% vs. 17.1%, p<0.05) than those with high adherence. Patients with unfavourable BP used on average 1.9 (SD 1.1) BP lowering drugs at hospital discharge and the proportion of patients treated with ACEI/ARBs and beta-blockers decreased significantly (p<0.001) from discharge to follow-up.

Diabetes (Odds ratio (OR) 2.4), higher BMI (OR 1.05 per 1.0 kg/m²) and older age (OR 1.04 per year) were significantly associated with unfavourable BP control in adjusted analyses (Table 3).

Table 3. Odds ratios for blood pressure target not reached at follow-up by study factors (calculated from logistic regression analysis).

<table>
<thead>
<tr>
<th>Study factors</th>
<th>Odds ratio (95% confidence interval)</th>
<th>Crude estimate</th>
<th>p-value</th>
<th>Multi-adjusted estimate</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at index event per year</td>
<td>1.04 (1.02-1.05)</td>
<td>p&lt;0.001</td>
<td></td>
<td>1.04 (1.03-1.06)</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>Female gender</td>
<td>0.80 (0.59-1.09)</td>
<td>n.s.</td>
<td></td>
<td>0.73 (0.53-1.00)</td>
<td>n.s.</td>
</tr>
<tr>
<td>More than 1 coronary event prior to the index event</td>
<td>1.42 (1.09-1.87)</td>
<td>p=0.01</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Participation in cardiac rehabilitation</td>
<td>0.75 (0.58-0.96)</td>
<td>p&lt;0.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Charlson comorbidity score</td>
<td>1.27 (1.16-1.40)</td>
<td>p&lt;0.001</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>2.56 (1.80-3.62)</td>
<td>p&lt;0.001</td>
<td></td>
<td>2.40 (1.67-3.46)</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>Body Mass Index per 1.0 kg/m²</td>
<td>1.04 (1.01-1.07)</td>
<td>p&lt;0.01</td>
<td></td>
<td>1.05 (1.02-1.08)</td>
<td>p&lt;0.01</td>
</tr>
<tr>
<td>HADSb - anxiety ≥8</td>
<td>0.69 (0.50-0.95)</td>
<td>p&lt;0.05</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a Adjusted for all variables with p≤ 0.1 retained in backward elimination binary logistic regression analysis and with age and sex forced into the final model. b Hospital Anxiety and Depression Scale

Age (standardized beta (β) 0.24), BMI (β 0.07) and lower sum score of HADS-depression (β -0.073, p<0.05) were associated with systolic BP in adjusted linear analyses (Table 4).
Table 4. Systolic blood pressure at follow-up regressed on study factors by linear regression analysis (OLS). Unstandardized (b) and standardized (β) regression coefficients.

<table>
<thead>
<tr>
<th>Study factors</th>
<th>Crude estimate</th>
<th>Multi-adjusted estimate(^a)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>b (standard error)</td>
<td>Standerdized β</td>
</tr>
<tr>
<td>Age at index event per year</td>
<td>0.476 (0.062)</td>
<td>0.234</td>
</tr>
<tr>
<td>Female gender</td>
<td>-0.909 (1.460)</td>
<td>-0.020</td>
</tr>
<tr>
<td>Ethnic minority</td>
<td>-8.397 (3.708)</td>
<td>-0.071</td>
</tr>
<tr>
<td>Charlson co-morbidity score</td>
<td>2.619 (0.451)</td>
<td>0.188</td>
</tr>
<tr>
<td>Body Mass Index per 1.0 kg/m(^2)</td>
<td>0.147 (0.133)</td>
<td>0.035</td>
</tr>
<tr>
<td>HADS(^b) - anxiety sum score</td>
<td>-0.549 (0.162)</td>
<td>-0.108</td>
</tr>
<tr>
<td>HADS(^b) - depression sum score</td>
<td>-0.534(0.188)</td>
<td>-0.091</td>
</tr>
<tr>
<td>Type D personality disorder</td>
<td>-3.496 (1.596)</td>
<td>-0.070</td>
</tr>
<tr>
<td>Insomnia(^c)</td>
<td>-2.924 (1.220)</td>
<td>-0.076</td>
</tr>
</tbody>
</table>

\(^a\) Adjusted for all variables with \(p \leq 0.05\) retained in backward elimination linear regression analysis and with age and sex forced into the final model.

\(^b\) Hospital Anxiety and Depression Scale

\(^c\) Insomnia (Bergen insomnia scale): a 7 -item self-report inventory designed to assess primary insomnia.
6 Methodological discussion

6.1 Study design
Epidemiologic research is a cornerstone discipline in patient-related evidenced based medicine\textsuperscript{171}. As described in section 1.2 in the introduction, results from epidemiological studies have been fundamental to the development of preventive medicine\textsuperscript{1,171}. The main objective for epidemiological studies is to reveal unbiased relationships between exposure and outcome. There are two main types of epidemiological studies; the observational and the experimental. The observational studies are by far the most common\textsuperscript{172}. The results derived from observational studies are mainly hypothesis generating and thus may be tested in clinical experiments in randomized trials to give a finite answer\textsuperscript{171}. Observational studies can be categorized in to case-control studies, prospective or retrospective cohort studies, and cross-sectional studies. The design of the present study is a cross-sectional study with a retrospective component.

Cross-sectional observational studies are useful in both determining the prevalence of a specific disease and in studying the relationship between exposure and a clinical outcome at a given time\textsuperscript{34}. As the outcome and the different exposures are collected at the same time, cross-sectional studies frequently cannot address causation since the time order of exposure relative to the outcome often is unknown. This is a major limitation of this study design\textsuperscript{34}. The major advantage with observational studies compared to intervention studies is that the exposure factors can be measured retrospectively within an extended time frame. Furthermore, exposure factors that cannot be manipulated in experiments, partly for ethical reasons (e.g. smoking), can be studied. The cross-sectional study is also time and resource efficient and allows the assessment of many variables in one study.

We wanted to determine the prevalence of risk factor control in a CHD population and to explore the associations between a broad range of socio-demographic, medical and psychosocial factors and risk factor control, for which a cross-sectional design is well suited. The comprehensive data set analysed as an interrelated whole enables us to control for a wide range of possible confounding factors. We have chosen a study design and inclusion and exclusion criteria similar to the EuroAspire studies\textsuperscript{63,64} in order to compare our data with other European data.

6.2 Random error
Random error in a study is a result of the natural, periodic fluctuation or variation in the precision of the true value and may occur during data sampling, data collection, data transfer, or data analysis\textsuperscript{173,174}. While systematic errors will distort results (point estimates or statistical associations) in one direction\textsuperscript{174}, random errors are equally likely to distort results in either positive or negative direction\textsuperscript{173}. Random errors tend to obscure real differences and weaken (attenuate) associations between the exposure and outcome\textsuperscript{175}. In turn, this increases the risk of committing type II errors\textsuperscript{158}. In clinical research, random error can be due to human variability and misunderstandings, but also to pure chance\textsuperscript{173}.

In this PhD thesis we have used a 95% confidence interval in all analyses. This means that the risk is less than 5% for rejection of the null hypothesis by chance given that there is in fact no relationship between the exposure and the outcome. The risk of falsely rejecting the null hypotheses decreases
with decreasing p-value\textsuperscript{176}. In the present work, the associations between a number of exposures (i.e. NOR-COR factors) and outcomes are studied. Therefore, the risk increases that some associations may turn out significant (i.e. p-value <0.05) as a result of chance. It is therefore important to interpret the study results in view of previous literature and discuss the plausibility and the clinical implications of the result carefully\textsuperscript{174}. In general, the estimates of the main study results underlying this PhD thesis were relatively unequivocal with narrow confidence intervals. However, in selected variables (i.e. ethnic minorities) and subgroups of patients (for example females who quitted smoking) relatively few participants necessarily yielded wider confidence intervals. It is therefore important to test hypotheses related to such subgroups in different populations and with other study designs.

Reduction in the random error can be achieved by increasing the sample size, reducing the variability in the measurements, or by increasing the number of measurements\textsuperscript{176}. To insure a satisfactory sample size when exploring the association between exposures and outcome, the number of cases with the outcome variable of interest should at least be ten times the number of covariates (i.e. exposures) that are included in the final model\textsuperscript{34}. Given a prevalence of the least frequent risk factor smoking in Euroaspire\textsuperscript{63} of 16\%, the estimated number of patients included (i.e. 1000) would result in at least 160 smokers, allowing us to include 16 covariates in the model. Due to the study sample size we did not have enough power to perform multi-adjusted analyses in subgroups of the persistent smokers (for example age and sex). However, no important differences in the major study factors were found in unadjusted sub-group analyses of age (<\textgtrless median age) or sex. Due to the cross-sectional design all measurements have only been performed once, with misclassification therefore being a possibility. However, the relatively large study population reduces the importance of this effect.

Questionnaires are frequently used in epidemiological research as they are easy to utilize, feasible and affordable\textsuperscript{174}. However, they may be affected by both random and systematic errors. Ambiguous questionnaires, poor understanding of the questions, distraction, confusion or the patients’ mood while answering a questionnaire could result in random errors, thereby reducing (attenuating) the observed statistical associations\textsuperscript{158,175}. The NOR-COR questionnaire was comprehensive. To evaluate the reproducibility and reliability of the questionnaire, a test–retest study was performed in 99 CHD patients who completed the questionnaire twice with four weeks in between\textsuperscript{158}. If there is no real change in the studied phenomena a reproducibility test will assess the magnitude of random measurement errors\textsuperscript{158}. The intra-individual reproducibility was found to be acceptable to excellent (Cronbach $\alpha$ values 0.69-1.0) for all key items and instruments\textsuperscript{158}. For example, the Cronbach $\alpha$ for current smoking was 1.0, while it was 0.94 for former smoking and 0.87 for never smoking. The Cronbach $\alpha$ values and the intraclass correlation coefficients for most instruments were comparable to previous studies\textsuperscript{159-162}. These findings are reassuring and indicate a relatively low risk of committing type II errors due to random measurement error\textsuperscript{158}.

### 6.3 Systematic error

Internal validity refers to the lack of bias (i.e. lack of systematic errors) in the study results. A high internal validity means that the observed association between the exposure and the outcome is likely
to be correct so that causal conclusions based on a study are warranted\textsuperscript{171,175}. Internal validity is a prerequisite for external validity\textsuperscript{34}. Systematic errors arise from flaws in the data sampling or measurement methods (i.e. a tendency to achieve consistently too low or high scores)\textsuperscript{173}. They are non-random variations in the study results and may lead to distortion of results in one direction\textsuperscript{174}. Unlike random errors, the adverse effects of systematic errors cannot be compensated for by increasing the sample size\textsuperscript{173}. Systematic errors are usually grouped into: selection bias, information bias, and confounding\textsuperscript{175}, which will be described below together with examples of errors that may influence the results of the present PhD project.

6.3.1 Selection bias
Selection bias is a distortion of the results caused by non-representative selection of participants in a study\textsuperscript{174}. Patients eligible for study inclusion were identified from discharge lists at the hospitals of Drammen and Vestfold in the period 2011-14. Thus, patients living in the catchment area suffering a coronary event during that time period without being admitted to these hospitals would not be identified for inclusion. An example would be patients undergoing elective coronary angiography at a University Hospital without being returned back to the local hospital afterwards. These patients are potentially healthier and could have better risk factor control, and this might cause us to overestimate comorbidity and unfavourable risk factor control. However, as the number of private cardiac specialists that can refer patients to coronary angiography is limited in Vestfold and Drammen, the number of such patients is most likely low.

Survival bias is a type of selection bias. Survivors may differ from those who died from the disease, which may lead to systematic loss of information\textsuperscript{174}. Patients who died at the index coronary event or during the period between the index event and study inclusion were not included in the study. These patients had, most likely, more comorbidity and poorer risk factor control than those who participated, thus possibly introducing a survival bias. However, this source of bias is most likely to be conservative, as risk factor control probably would have been even worse if these patients were eligible for inclusion. The patients were included 2-36 months after the index event to allow us to study changes in risk factor control with increasing time since the index event. This design may also introduce a possible bias as the number of excluded patients due to death or short life-expectancy might be higher with increasing time since the index event. However, the number of excluded patients was equally distributed according to the time span between the index event and study inclusion (Paper I). The risk of bias due to survival or short life expectancy is therefore most likely of minor importance.

Non-participation is a source of selection bias if the characteristics of the patients who do not participate differ from those who participate\textsuperscript{173}. Non-participation is a well-recognized challenge that frequently influences epidemiologic studies\textsuperscript{173}. The high participation rate of 83% is therefore a major strength of our study. We have also conducted a non-participation study\textsuperscript{177} that allowed us to explore the characteristics of almost all (i.e. 229 out of 239) non-participating patients. The comparisons between study participants and non-participants at the index hospitalization are rendered in Table 1 and 2 below\textsuperscript{177}. 
# Table 1. Comparison of socio-demographic factors, clinical characteristics and risk factors between participants and non-participants.

<table>
<thead>
<tr>
<th></th>
<th>Participants n = 1127</th>
<th>Non-participants n = 229</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time since the index coronary event, months</td>
<td>17.1</td>
<td>17.3</td>
<td>ns</td>
</tr>
<tr>
<td>Mean age at the coronary event (SD), years</td>
<td>61.6 (9.6)</td>
<td>61.8 (10.4)</td>
<td>ns</td>
</tr>
<tr>
<td>Female, %</td>
<td>21.0</td>
<td>27.9</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Socio-demographic factors, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethnic minorities, %</td>
<td>3.0</td>
<td>5.7</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Living alone, %</td>
<td>19.3</td>
<td>26.4</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Unemployed or on disability benefits, %</td>
<td>20.7</td>
<td>15.7</td>
<td>ns</td>
</tr>
<tr>
<td>Coronary diagnosis, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non ST elevation myocardial infarction</td>
<td>49.8</td>
<td>52.0</td>
<td>ns</td>
</tr>
<tr>
<td>ST elevation myocardial infarction</td>
<td>29.7</td>
<td>32.3</td>
<td>ns</td>
</tr>
<tr>
<td>Stable or unstable coronary heart disease</td>
<td>20.5</td>
<td>15.7</td>
<td>ns</td>
</tr>
<tr>
<td>Angiography findings, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Open vessels</td>
<td>1.3</td>
<td>0.9</td>
<td>ns</td>
</tr>
<tr>
<td>Atherosclerosis, no sig. stenosis</td>
<td>4.8</td>
<td>3.1</td>
<td>ns</td>
</tr>
<tr>
<td>Single-vessel disease</td>
<td>55.2</td>
<td>50.9</td>
<td>ns</td>
</tr>
<tr>
<td>Multi-vessel disease</td>
<td>38.6</td>
<td>45.1</td>
<td>ns</td>
</tr>
<tr>
<td>Coronary intervention, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percutaneous coronary intervention and stent</td>
<td>75.3</td>
<td>77.7</td>
<td>ns</td>
</tr>
<tr>
<td>Percutaneous coronary intervention without stent</td>
<td>2.0</td>
<td>0.9</td>
<td>ns</td>
</tr>
<tr>
<td>Coronary artery bypass graft operation</td>
<td>13.0</td>
<td>10.9</td>
<td>ns</td>
</tr>
<tr>
<td>No intervention</td>
<td>9.6</td>
<td>10.5</td>
<td>ns</td>
</tr>
<tr>
<td>More than one coronary event, %</td>
<td>29.9</td>
<td>32.7</td>
<td>ns</td>
</tr>
<tr>
<td>Coronary risk factors, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current smoking</td>
<td>34.8</td>
<td>37.0</td>
<td>ns</td>
</tr>
<tr>
<td>Hypertension&lt;sup&gt;a&lt;/sup&gt;</td>
<td>43.4</td>
<td>54.4</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Diabetes type 1 and 2</td>
<td>16.6</td>
<td>23.8</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Participation in cardiac rehabilitation, %</td>
<td>50.1</td>
<td>27.5</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

<sup>a</sup>Ns: non-significant; SD: standard deviation.
<sup>b</sup>Ethnic minorities means 1st and 2nd generation patients from Asia, Africa and South America.
<sup>c</sup>Hypertension noted in the hospital medical record.

# Table 2. Comparison of somatic and psychiatric comorbidity and medication between participants and non-participants.

<table>
<thead>
<tr>
<th></th>
<th>Participants n = 1127</th>
<th>Non-participants n = 229</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevalence of somatic comorbidity, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart failure</td>
<td>13.1</td>
<td>17.7</td>
<td>ns</td>
</tr>
<tr>
<td>Periphere vascular disease</td>
<td>89</td>
<td>11.6</td>
<td>ns</td>
</tr>
<tr>
<td>Apoplexy/transitory ischemic attacks</td>
<td>7.0</td>
<td>13.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>88</td>
<td>11.0</td>
<td>ns</td>
</tr>
<tr>
<td>Chronic kidney failure&lt;sup&gt;a&lt;/sup&gt;</td>
<td>13.4</td>
<td>18.2</td>
<td>ns</td>
</tr>
<tr>
<td>Atrial fibrillation or atrial flutter</td>
<td>9.4</td>
<td>14.9</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Liver disease</td>
<td>3.1</td>
<td>3.0</td>
<td>ns</td>
</tr>
<tr>
<td>Charlson comorbidity score (SD)</td>
<td>4.1 (1.4)</td>
<td>4.4 (1.7)</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Prevalence of psychiatric comorbidity, % Depression</td>
<td>6.3</td>
<td>19.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Anxiety</td>
<td>3.0</td>
<td>8.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Medication at discharge, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>985</td>
<td>99.4</td>
<td>ns</td>
</tr>
<tr>
<td>Warfarin or NOAK’s</td>
<td>73</td>
<td>8.8</td>
<td>ns</td>
</tr>
<tr>
<td>Statins</td>
<td>961.1</td>
<td>98.3</td>
<td>ns</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>848.6</td>
<td>90.6</td>
<td>ns</td>
</tr>
<tr>
<td>Antidiabetics</td>
<td>109.9</td>
<td>14.4</td>
<td>ns</td>
</tr>
<tr>
<td>Insulin</td>
<td>43.9</td>
<td>8.3</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>ACE-inhibitors</td>
<td>350.0</td>
<td>40.9</td>
<td>ns</td>
</tr>
<tr>
<td>Angiotensin-2-receptor blocker</td>
<td>203.2</td>
<td>21.0</td>
<td>ns</td>
</tr>
<tr>
<td>Aldosterone antagonists</td>
<td>28.0</td>
<td>3.9</td>
<td>ns</td>
</tr>
<tr>
<td>Nitrates</td>
<td>39.0</td>
<td>3.6</td>
<td>ns</td>
</tr>
<tr>
<td>Loop or thiazidi diuretics</td>
<td>215.2</td>
<td>24.4</td>
<td>ns</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>161.4</td>
<td>13.8</td>
<td>ns</td>
</tr>
<tr>
<td>Psychotropic drugs (antidepressant or anxiolytics)</td>
<td>50.1</td>
<td>12.2</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

<sup>a</sup>Ns: non-significant; SD: standard deviation; NOAC: novel anticoagulant drugs.
<sup>b</sup>Defined as estimated glomerular filtration rate < 60 ml/min/1.73 m².

Study non-participants had a higher proportion of females, ethnic minorities and patients living alone. Moreover, non-participants had a higher occurrence of hypertension, diabetes, somatic and psychiatric comorbidity recorded in the discharge letters, and a lower average participation rate in cardiac rehabilitation than participants. In adjusted analyses, somatic comorbidity (OR 3.4, 95% CI 2.8-4.3) and depression (OR 14.5, 95% CI 4.4-121.5) were the major factors associated with study non-participation.

Non-participation has larger implications for prevalence studies (i.e. point estimates) than for studies exploring the associations between exposure and outcome. Thus, the findings demonstrated in the non-participation study most likely have greatest impact upon the results reported in paper I. As important risk factors were more prevalent in non-participants at the time of the index event, it is reasonable to assume that the observed overall risk factor control would have been even poorer if we had been able to include all eligible patients. As the proportion of females and ethnic minorities was higher in the non-participants, the association between gender and minority and risk factor control might have been different if these patients had participated. Anxiety and depression reported in the hospital records were also more frequent in non-participants, and we might have found a different association between these factors and persistent smoking and BP control if these patients had participated.

6.3.2 Information bias
Information bias or measurement bias, refers to incorrect determination of exposure and/or outcome due to errors in a measurement. A measurement is valid (i.e. unbiased) if it has both high accuracy and a high precision. Accuracy refers to the degree of overlap between a measured value and the actual (“true”) value, while precision refers to the degree of which further measurements will show the same or similar results. Measurement bias is well recognized in self-reported data. For example, drug adherence as assessed by self-report instruments is frequently over-reported compared to more objective methods (i.e. data from pharmacy registries and/or direct concentration measurements in blood), which may influence the association between adherence and BP control in paper III.

Recall bias is a form of information bias and occurs when patients have a different recall of exposures. In NOR-COR, all patients underwent a coronary event prior to study inclusion. The time between the coronary event and inclusion ranges from 2-36 months, and recall of factors related to the event may be less accurate with the passing of time. However, most questionnaires items used in this PhD thesis are related to the patients’ current situation at the time of inclusion (i.e. risk factor control, current medication, current psychosocial status etc.). Therefore, the risk of recall bias introduced by increasing time since the event should be limited.

The study questionnaire has a high completion rate and missing data ranged from 0-10%. The level of accuracy of self-reported items is, nevertheless, difficult to assess. The questionnaire is comprehensive, and one might suspect increasingly attrition at the end of the questionnaire. However, the reproducibility values for questions in the first part of the questionnaire did not differ
from those in the last part. Data recorded from self-report questionnaire might be distorted by patients giving socially desirable answers. This may be even more pronounced in patients receiving secondary prevention after a subsequent CHD event. Potentially, this has led to an overestimation of self-report drug adherence and an underestimation of self-reported unhealthy lifestyle behaviours such as smoking, unhealthy diet and physical inactivity.

The study data from the time of the index event were retrospectively obtained from hospital medical records with the possibility of reporting bias. For example, the prevalence estimates of depression and anxiety from the hospital records are likely to be too low as psychiatric diagnosis reported in somatic hospital medical records would be less sensitive than standardized psychiatric diagnostic assessments.

6.3.3 Limitations, random and systematic errors in the study data
Random and/or systematic error can potentially influence all study variables (both independent and the dependent) in the present study. As the study has included a large number of variables, I have chosen to evaluate limitations and strengths of the most central variables.

- Smoking at follow-up was assessed by self-report. We do not have data on the use of smoking cessation aids among quitters, the date of smoking cessation or a specific questionnaire addressing nicotine dependence. Self-reported smoking has a sensitivity of 87.5% and a specificity of 89.2% against biological assessments with cotinine-testing. Non-reporting is a random error that can influence the prevalence estimate of smoking. Non-reporting may also be a systematic error if sub-groups of smokers (for example those with depression) systematically under-report smoking. Other examples of systematic errors are selection bias, recall bias and reporting bias. However, the equal prevalence of smoking among the study participants and non-participants and the high reproducibility of the study questionnaire indicate a relatively low the risk of systematic errors for this variable.

- BP at follow-up was assessed by standardized recommended procedures by a Welch Allyn WA Connex ProBP 34000 digital sphygmomanometer. Potential random and systematic errors include measurement errors, biological variation and cuff-sizes, while the number of measurements is an example of a potential systematic error. To minimize these errors, different cuff-sizes were applied in our study. Moreover, we used BP devices that have been previously validated as highly acceptable: mean ± standard deviation of the observer/device differences were 1.1 ± 7.5 mmHg for SBP and 2.1 ± 6.0 mmHg for DBP. We only used experienced study personnel in the study and a written detailed procedure for BP measurements was followed carefully. BP measurements at the index event were obtained from the hospital medical records and were not necessarily measured with standardized methods. Therefore, these data have not been included in the analysis in paper III. At follow-up, we do not have data on BP lowering drugs except for beta-blockers and ACEI/ARBs. White-coat hypertension is a well-known systematic error that influences BP measurements and may lead to an overestimation of unfavourable BP control. If this phenomenon is more prevalent in subgroups of patients, for example those without depression, this could make up for the association between depression and lower systolic BP.

- Physical activity was assessed by a self-report. The self-reported questionnaire has previously been validated against direct measurement of VO\text{\textsuperscript{2}}max. The correlation was fairly good and
Cohen’s kappa ranged 0.69-0.82 for test vs. retest. As the intervals in this questionnaire do not allow for cut-off values exactly according to guideline recommendations we have chosen a definition close to the minimum level of physical activity recommended. Non-reporting is a random error that can influence the prevalence estimate of physical activity. Examples of systematic errors are selection bias, recall bias and reporting bias. We do not have data on the level of physical activity among the study non-participants, but as these patients have higher occurrence of somatic comorbidity, we expect that these patients had a lower level of physical activity compared to study participants. Thus, the prevalence estimates of physical activity reported in paper I might be even lower if these patients had been included. In the reproducibility test of the questionnaire, a high intraclass correlation coefficient of 0.90 (0.55-0.95) for the physical activity sum score was found, which reduces the risk of random errors for this variable.

- Body height, body weight, and waist circumference were measured with objective methods. Measurement errors, body positioning during measurement and the weight of clothes are examples of potential random errors. In the literature, a high inter-observatory reliability (0.97) have been demonstrated for these methods. By using experienced study personnel and written detailed procedures for the measurements the risk of systematic errors is minimized.

- The diagnosis of diabetes was defined as either treatment with antidiabetic drugs or a diabetes diagnosis recovered from hospital records at the time of the index event. Underreporting by clinicians is therefore a potential random error. Study data from the hospital records were recovered by an experienced medical doctor which reduces the risk of other random errors. Diabetes was more prevalent among the study non-participants, which is a systematic error that influences the prevalence estimates.

- Analyses of LDL cholesterol and HbA1c are prone to measurement errors and biological variation (random errors), while the calibration of assays and analyses are potential systematic errors. All blood samples were analysed at Drammen hospital to avoid inter-laboratory bias. The laboratory has routines for calibrating the analysing equipment regularly, and variation coefficients between plasma LDL cholesterol are 4% (personal communication Trine-Lise Kristensen, Overbioingeniør Medisinsk biokjem, Drammen Hospital, Vestre Viken HF) and between HbA1c are 2% (personal communication Hanne Frydenlund Overbioingeniør, Medisinsk biokjem, Drammen Hospital, Vestre Viken Trust).

- Other study data from the hospital medical records (i.e. age, sex, ethnicity, coronary index/treatment, medication) have been researched by an experienced medical doctor. The risk of random and systematic errors is therefore most likely low.

- Participation in cardiac rehabilitation was extracted from the rehabilitation programmes in Vestfold and Drammen. Since we do not have a complete overview of participation in external rehabilitation programmes, underreporting is a potential random error that could possibly lead to our finding of no association between rehabilitation participation and smoking cessation or BP control.
Even though a large proportion of the other self-report instruments have been previously validated\textsuperscript{148,160-162,165-168,185}, these data are all prone to random and systematic errors as previously discussed.

### 6.3.4 Confounding and effect modifiers

A confounder is an extraneous variable that is associated both to the studied exposure and the outcome (common underlying cause), thus leading to misleading associations\textsuperscript{171,175}. Thus, to be a confounder, the variable has to be associated with both the outcome and the studied exposure\textsuperscript{172}. If a variable is fact an intermediate step in the causal pathway between a given exposure and the outcome, this variable is not a confounder but an effect mediator\textsuperscript{171}. An effect modifier may affect the strength or even direction of the association between an exposure and an outcome\textsuperscript{19,171}. Confounders need to be taken into account, otherwise the observed association between exposure and an outcome may be misleading (spurious)\textsuperscript{171}. Confounding can be controlled for by design (i.e. randomization), by restriction or matching, or during analysis of data by stratification, simultaneous statistical control or more complex multivariable analyses\textsuperscript{175}. In paper I-III, multivariable regression analyses have been conducted in an attempt to control for confounders. Although the dataset allows a comprehensive evaluation of determinants associated with risk factor control, additional confounders should be considered. For example, factors related to the patient–provider communication and different follow-up practice among GPs may be of importance.

### 6.4 External validity

Validity refers to our ability to measure what we intend to measure, and can be divided into internal and external validity\textsuperscript{34,175}. Internal validity is defined as the degree to which the study results are representative for the cohort being studied and is dependent on both accuracy and precision of the results. A proper evaluation of systematic errors and potentially confounders as alternative explanation of the results is therefore required\textsuperscript{175}. Extern validity refers to the degree to which results of a study can be generalized to other populations or groups that did not participate in the study\textsuperscript{19,175}. If the characteristic of the selected study population systematically differs from the other populations the study conclusions may not applicable to these other populations\textsuperscript{19}.

The population of Vestfold and Buskerud from which the study sample was drawn is fairly representative of Norway in terms of geography, economy, age distribution, morbidity and mortality\textsuperscript{59,155}. Furthermore, the level of education in our coronary population is in accordance with national data\textsuperscript{186}. Due to the study inclusion criteria only 3% of patients had an ethnic minority background, which is lower than the national average of 9\textsuperscript{187}. We also limited inclusion to patients below the age of 80 years, and, as such, generalizability to ethnic minorities and patients older than 80 years of age should therefore be made with caution. The routine clinical setting and the high study participation rate of 83\% are important strengths of the study that increase its external validity. Knowledge of the study non-participants is also important for external validity\textsuperscript{178}. When interpreting the study results it is therefore important to acknowledge the higher proportion of patients with somatic comorbidity and a diagnosis of anxiety and depression that was found among the study non-
participants. Despite this, the study results should be considered to be fairly representative of a general Norwegian CHD population.

The generalizability to international populations, however, must always be evaluated with caution as the distribution of coronary risk factors as well as socio-demographic, medical and psychosocial factors are known to differ considerably between different countries and regions.

6.5 Ethical considerations
The NORCOR Study was approved by the Regional Committee of Ethics in Medical research. Study participation was considered not to cause any significant disadvantage for the patients. Findings in the study that were considered potentially serious and required further medical attention were handled during patient inclusion. Patients with BP values >140/90 mmHg (>140/80 mmHg in Diabetes), LDL-cholesterol >2.5 mmol/L, HbA1c >8% in diabetics and HbA1c >6.1% in non-diabetics, were recommended to contact their GP’s for further follow-up. Furthermore, their GP’s were informed in a separate letter. A study cardiologist initiated and/or intensified BP lowering treatment in patients with BP>180/100 mmHg.

6.6 Summary of methodological considerations
Due to the cross-sectional design, causal relationships cannot be inferred. However, a cross-sectional design is well suited for the aims of this study. The coronary risk factors and study factors were measured at one point in time and are potentially prone to both measurement and recall bias. Coronary risk factor control may be even worse than reported due to a selection bias by survival effect and non-participation. Anxiety and depression reported in the hospital records were more frequent in non-participants compared to study participants. Thus, the observed association between these factors and persistent smoking and BP control might have been different if these patients had participated. We consider the data to be fairly representative for a Norwegian CHD population. However, caution should be made in generalizing the results to patients >80 years and to subgroups with ethnic minority background.

Although, comprehensive evaluations of determinants associated with persistent smoking and unfavourable BP control were performed, additional confounders should be considered. Adherence was assessed by self-report questionnaires and not by more objective methods, which may have influenced the role of adherence on BP control. We do not have enough power to perform multi-adjusted analyses in sub-groups of age and gender for persistent smoking. However, no important differences in the major study factors associated with persistent smoking were found in unadjusted sub-group analyses.

Important strengths of the study are patient inclusion from a routine clinical setting, the high participation rate (83%) and few missing data. The NOR-COR questionnaire have been evaluated in a reproducibility study which demonstrated highly acceptable test-retest values for all key items and instruments, indicating low random variation. The coronary risk factors are all evaluated with validated measures that are assessed to be fairly good. We have performed a non-participant study to give as complete picture as possible of the eligible study population.
7. Discussion of results from paper I-III

7.1 Risk factor control in Norway compared European and Nordic results

Prevalence data on CHD risk factors from different populations and regions are needed in order to monitor the quality of secondary prevention. The serially conducted EuroAspire studies have convincingly demonstrated that risk factor control in Europe is inadequate and with few improvements over the past decades. The patients are mainly included from academic specialist centres and the participation rate was only 49% in the most recent EuroAspire IV study. Compared to EuroAspire IV, we found quite similar levels of elevated BP (46% vs. 43%), obesity (34% vs. 38%) and low physical activity (both 60%) (paper I). Our patients had better LDL cholesterol control (81% vs. 57%), while the number of daily smokers (21% vs. 16%) was higher. Moreover, 59% of diabetics in our study had HbA1c >7% as opposed to 48% in EUROASPIRE IV. Since the Nordic countries have many similarities, it is also relevant to compare our data with the Swedish and Finnish subset of the EuroAspire IV study. Compared to these countries, only the frequency of elevated LDL cholesterol was lower in our population (57% vs. 81% in Sweden and 67% in Finland), while the respective frequencies of current smoking (21% vs. 14% and 8%), elevated BP (46% vs. 38% and 34%) and elevated HbA1c (59% vs. 43% and 28%) were significantly higher.

Norway has a high-performing health system, a good general health status, and a favourable socio-economic situation compared to the European average. The higher frequency of current smoking in our CHD population (21%) compared to EuroAspire and other international studies (ranging 12-18%), is noteworthy as Organisation for Economic Co-operation and Development (OECD) statistics indicate a lower prevalence of smoking in the general population in Norway (19%) than the European average (23% [Sweden 14%, Finland 19%]). Correspondingly, the rate of obesity in our study was also quite similar to the EuroAspire IV results, although the frequency of obesity in Norway is lower than most other European countries. Apparently, true differences between these CHD populations may exist, but these findings may also have methodological explanations (i.e. selection bias) since the patients were mainly recruited from academic centres with low participation rate. If so, risk factor control in clinical practice across Europe is even worse than previously reported.

Recent data from Norway revealed a 3% annual decline in CHD events from 1994 to 2010. This was mainly explained by declining incidence of STEMIIs and sudden cardiac death. Two thirds of the decline was explained by favourable changes in cholesterol and BP, in addition to declining rates of smoking. Despite these positive trends, our data reveal that smoking, elevated BP and poor control of other risk factors are prevalent after CHD events. The youngest CHD patients had the highest frequency of unfavourable lifestyle factors in our study (paper I), which may have contributed to an early onset of CHD. Unfavourable lifestyle may also partly explain why the positive decline in MI and cardiac mortality in Norway from 1994-2009 was less prominent in the youngest CHD patients. The relative risk of MI due to smoking is much higher in patients <50 years, than in elderly patients. Elevated BP in younger patients is also associated with higher risk of mortality and stroke. This underscores the particular importance of facilitating smoking cessation and...
achieving BP goals in the youngest patients. Patients with more than one previous coronary event had the poorest overall risk factor control (paper I), which might explain why they suffer repeated events. It will therefore also be crucial to improve secondary preventive management in this high-risk subgroup.

7.2 Socio-economic status and smoking and blood pressure control in CHD patients
Socio-economic status is usually measured by education, income and/or occupation\textsuperscript{3}. Education is the most commonly used socio-economic measure in epidemiological studies and the strongest socio-economic predictor of unfavourable risk factor control\textsuperscript{3}. Low socio-economic status has been associated with increased mortality\textsuperscript{200} and poorer self-assessment of health\textsuperscript{201}, and thereby negatively influences risk factor control and prognosis in CHD patients\textsuperscript{2,85}. We confirmed a strong and linear association between low education and unemployment or disability status and persistent smoking (paper II). Low education was also strongly associated with physical inactivity (paper I), confirming the well-known association between unfavourable lifestyle and low socio-economic status\textsuperscript{85,110,202}. Interestingly, however, low socio-economic status was not related to BP (paper II) or LDL-cholesterol\textsuperscript{203}. These findings were consistent in men and women, older and younger, and dichotomous and continuous analyses. Thus, socio-economic status seems to be of greater importance in the prevention of lifestyle factors than biological risk factors. Even though socio-economic status is not easily modifiable, our study results support current recommendations in guidelines\textsuperscript{2} that these patients should be systematically identified during hospitalisation for the CHD events and be given particular attention and follow-up care\textsuperscript{202}.

7.3 Psychosocial factors and smoking and blood pressure control in CHD patients
The role of psychosocial factors for smoking behaviour and BP control in CHD patients is complex and multifactorial\textsuperscript{2,5,204}. The NOR-COR dataset comprises a comprehensive battery of psychosocial factors where most had previously been demonstrated to be modifiable by psychological interventions\textsuperscript{7}. Knowledge of the relative importance of these factors for smoking and BP control by simultaneously controlling for socio-demographic and clinical factors may help designing tailored and potentially more effective psychosocial interventions for CHD patients.

Psychosocial factors, in particular depression, have been associated with higher frequency of smoking in populations with and without CHD\textsuperscript{5,129,132,188,205}. Thus, smoking has been suggested as one of the mechanisms linking psychosocial factors to poor prognosis in CHD patients\textsuperscript{2,5,110}. On the other hand, other studies could not verify any clear associations between smoking and either type D personality\textsuperscript{206}, anxiety or depression, when controlling for socio-demographic factors\textsuperscript{133,207}. Despite these previous contradictory findings, we were surprised to find no numerical differences in psychosocial factors between persistent smokers and quitters on average 1.7 years after the index event (paper II). The association between psychosocial factors and biological risk factors like hypertension and cholesterol has been more uncertain\textsuperscript{108,152}, even though an association between depression and low adherence with antihypertensive and lipid lowering drugs in CHD patients has been documented\textsuperscript{97,208,209}. There are a limited number of studies assessing the association between psychosocial factors and control of BP and LDL cholesterol in CHD patients. A recently published
paper from the EuroAspire IV found no association between psychosocial factors and control of either BP or LDL-cholesterol in CHD patients\textsuperscript{188}, whereas one other previous study found no association between psychosocial factors and LDL-cholesterol\textsuperscript{210}. Psychosocial factors were neither found to be associated with BP (paper III) nor LDL-cholesterol\textsuperscript{203} in our studies. These findings were consistent in men and women, older and younger, and in dichotomous and continuous analyses. In accordance with a large Norwegian population-based cohort study with 22 years of follow-up\textsuperscript{153}, we observed that depression was independently associated with lower systolic BP.

Several circumstances may explain why our study results are partially contradictory to some previous studies, but similar to others. First, the timing of psychosocial assessment and length of follow-up may be of importance. In contrast to many previous studies\textsuperscript{126,129,130}, psychosocial factors were assessed at follow-up and not at the time of the index event. Moreover, the length of follow-up is longer than in most previous studies\textsuperscript{129}. Some studies have reported that psychosocial distress may decrease with increasing time lapse following a coronary event\textsuperscript{211}. If so, the association between psychosocial factors and coronary risk factor control might have been different if these factors had been registered at the time of the index event. Second, self-report questionnaires used to measure psychosocial distress have been largely different\textsuperscript{129,212}, which may potentially explain the divergences observed. For example, in the studies by Dawood et al\textsuperscript{130} and Gerber\textsuperscript{126} et al reporting a significant association between higher depression scores and smoking in adjusted analyses, depression was measured by PHQ-5 and BDI, while the HADS scale was used in our study. In a recent publication based on the EuroAaspire IV study\textsuperscript{188}, current smoking was associated with depressive symptoms measured by HADS. Importantly however, in that study the reference group was all patients who did not smoke daily at follow-up, while the reference group in our study was quitters after the index event. Third, despite a high participation rate in NOR-COR, the non-participant study\textsuperscript{177} revealed a larger proportion with previous depression recorded in the hospital records from the index event. Patient selection thus may influence the association between psychosocial factors and smoking and BP control. Finally, anxiety and depression have been associated with low physical activity in CHD patients\textsuperscript{152,188,207,213}. Unpublished data from NOR-COR (Peersen et al. under preparation) revealed a significant association between low physical activity and depression, even in multi-adjusted analyses. As psychosocial factors appear to be differently associated with the major coronary risk factors, our data encourage future secondary preventive interventions to be individually tailored to specific cardiovascular risk factors and the associated psychosocial determinants\textsuperscript{7,204,214}.

### 7.4 Medical factors, motivation, illness and risk perception and persistent smoking in CHD patients

Smoking is prevalent in CHD patients, and the relapse rate among successful quitters remains high\textsuperscript{215,216}. A long smoking duration and not having STEMI as the index event were the major medical factors associated with persistent smoking in adjusted analyses (paper II). None of the factors are directly modifiable, but they are probably important for understanding why smoking cessation is difficult. A qualitative study found that patients with STEMI tended to perceive their illness as a life-threatening event, while patients with NSTEMI were more uncertain about both
symptoms and the diagnosis\textsuperscript{217}. The patients’ disease perception and to what extent they appreciate the seriousness of the condition might influence the motivation for smoking cessation\textsuperscript{217}. In a Swedish study, a significant number of PCI treated coronary patients perceived themselves to be cured from their CHD\textsuperscript{94,218} and did not see the need to change lifestyle\textsuperscript{94}. In our study, however, most persistent smokers rated smoking as the most important cause of their CHD, and perceived themselves to have a higher risk for subsequent coronary events compared to quitters. A majority also reported high motivation for smoking cessation, whereas 73\% had reduced their cigarette consumption after the index event (paper II). These findings, together with the long smoking history (60\% had been smoking for more than 40 years), indicate a substantial nicotine dependency\textsuperscript{111,121}. Both nicotine dependency and longer smoking duration are well recognized barriers to smoking cessation\textsuperscript{125,126}. In the EuroAspire III Study, 50\% of CHD patients smoking at follow-up had passed the pre-contemplation stage (i.e. no intention to quit smoking)\textsuperscript{125}. We have not assessed the different stages of change\textsuperscript{125}, but found that almost 7 out of 10 patients wanted help to quit smoking. Interestingly, among those who reported a high motivation for smoking cessation 83\% wanted help to quit smoking, while only 41\% of those with moderate to low motivation wanted help to quit. Together with a reported reduction in cigarette consumption, it is reasonable to assume that a large number of the persistent smokers have passed the pre-contemplation stage (“not ready to change state”), according to the Transtheoretical Model for behavior change\textsuperscript{219}. These patients who have past the pre-contemplation stage could benefit from motivational interview\textsuperscript{220} techniques to address ambivalence\textsuperscript{221} and facilitating smoking cessation. The chance of successful smoking cessation after a coronary event is highest after an immediate\textsuperscript{222} and abrupt\textsuperscript{216} quitting. Hospitalization for an acute CHD event often increases motivation to quit smoking\textsuperscript{122}, and European guidelines strongly recommend that smoking cessation should be raised with all smokers at the time of diagnosing a CHD event\textsuperscript{2}. The degree of addiction\textsuperscript{223} and motivation for quitting should be used to assess the need for pharmacological and non-pharmacological aids\textsuperscript{2}. With this in mind, it is concerning that only a minority (42\%) reported to have been offered nicotine replacement therapy or other smoking cessation aids (paper II), which is accordance with findings from other studies\textsuperscript{94,124}. Unpublished data from NOR-COR reveal limited information about smoking cessation and/or nicotine replacement therapy in the discharge letters (Munkhaugen et al. submitted to Tidsskriftet for den Norske Laegeforening in November 2017). High workload, limited time and system barriers to secondary prevention\textsuperscript{87,90,98} can potentially explain these observations. Alternatively, this insufficient information may indicate that smoking receives less attention from cardiologists than other risk factors\textsuperscript{111}.

More detailed discharge information, systematic prescription of nicotine replacement therapy and systematic referral to smoking cessation programs are therefore needed. The past Cochrane review from 2004 reported a small positive effect of exercise-based CR on smoking cessation\textsuperscript{25}, while such information was not included in the most recent report from 2014\textsuperscript{164}. Smoking has been associated with increased referral to CR, but also with increased likelihood of non-attendance and drop-out\textsuperscript{224}. Participation in the hospital-based CR programme was not associated with smoking cessation in the entire NOR-COR population (paper II), but in the Vestfold-cohort, participation in the comprehensive CR program at that hospital was associated with a 3 times lower likelihood of
smoking when compared with non-participating patients\textsuperscript{225}. Of note, the hospital-based CR content, duration and participation rate differed substantially between Drammen (18% participation) and Vestfold hospital (75% participation)\textsuperscript{225}. A recent Norwegian study found that average CR participation in PCI treated patients was only 28\%\textsuperscript{226}. It is well-documented in randomized trials that psychosocial interventions, nicotine replacement therapy and other pharmacological aids significantly increase quitting rates\textsuperscript{117,119,223,227}. It is therefore crucial to implement these evidence-based components in the existing hospital-based CR programmes and increase the participation rate by ensuring systematic referral to CR when patients are hospitalized for their CHD event. As the GPs are the key providers of long-term secondary prevention\textsuperscript{2}, it is also important to implement cessation interventions in primary care. We need to target factors at patient, healthcare, and system level to further reduce the prevalence of smoking in our CHD population\textsuperscript{17,111,223,228}.

7.5 Medical factors and blood pressure control in CHD patients

In line with previous studies in CHD patients\textsuperscript{229}, older age, obesity and diabetes were the major factors associated with unfavourable BP control (paper III). This was not surprising as they are all well-established interrelated risk factors for hypertension\textsuperscript{143,230,231}. The pathophysiological mechanisms linking these factors are complex and not yet completely understood, but involve arterial stiffening, reduced kidney function, inflammation, insulin resistance, neuro-hormonal dysfunction, and genetic factors\textsuperscript{143,230,232}. Arterial stiffness due to general arteriosclerosis, obesity and increasing age most likely plays an important role for BP control in many patients with established CHD\textsuperscript{233}. Even though BP reduction are found to reduce arterial stiffness\textsuperscript{234}, most available BP lowering drugs target peripheral resistance, and do not alter the arterial stiffening\textsuperscript{143}, which might explain why elderly and obese patient had poorer BP control. Lifestyle interventions through diet, exercise and behavioural changes often give initial weight-loss and improvements in BP control in obese patients, but are often unsuccessful in achieving long-term effects\textsuperscript{2,235}. Bariatric surgery in selected subgroups has documented positive effect of both long-term weight loss and hypertension, diabetes, and CVD risk\textsuperscript{235,236}, but is not without troublesome side-effects\textsuperscript{236}. CHD patients have been underrepresented in most studies assessing the effect of lifestyle intervention on BP control\textsuperscript{237}, however, the multi-disciplinary EuroAction Study combining lifestyle intervention with optimization of cardioprotective medication found an effect on BP control in CHD patients\textsuperscript{78}, and is a promising initiative\textsuperscript{237}.

Medical treatment with BP lowering drugs is necessary to achieve BP control in almost all CHD patients with hypertension\textsuperscript{136}. A recent meta-analysis found that the proportional risk reduction of BP lowering was broadly similar among patient with or without CVD, and that patients with the highest risk of new CV probably would have the largest benefit from BP reduction\textsuperscript{238}, emphasizing the importance of efficient BP lowering treatment in CHD patients. Combination therapy with BP lowering drugs from different classes is recommended and found to be far more effective than increasing the dosage of a single drug\textsuperscript{2,239}. Combination therapy is particularly important in obese patients as resistant hypertension is very common and these patients often require the additive effect of several BP lowering drugs to achieve BP control\textsuperscript{235}. The patients with unfavourable BP control in our study were on average only prescribed 1.9 BP lowering drugs at discharge after the index event.
The number of patients receiving beta-blockers (72% vs. 83%) and ACEI/ARB (50% vs. 75%) was lower than reported in the EuroAspire IV study and declined significantly from the index event to follow-up (paper III). Suboptimal drug prescription has been demonstrated in other studies of CHD patients with hypertension. In a recent Swedish study, the average number of BP lowering drugs was 2.1, despite only 27% of had appropriate BP control. Even though information on other BP lowering drugs at follow-up in our study unfortunately remains unknown, it seems to be mandatory to intensify the drug treatment in CHD patients with unfavourable BP control.

Several studies have documented that low adherence with BP lowering drugs is associated with unfavourable BP control, CVD events, and mortality in hypertensive patients. Suboptimal drug prescription has been demonstrated in other studies of CHD patients with hypertension. In a recent Swedish study, the average number of BP lowering drugs was 2.1, despite only 27% of had appropriate BP control. Even though information on other BP lowering drugs at follow-up in our study unfortunately remains unknown, it seems to be mandatory to intensify the drug treatment in CHD patients with unfavourable BP control.

Several studies have documented that low adherence with BP lowering drugs is associated with unfavourable BP control, CVD events, and mortality in hypertensive patients. Non-adherence is also identified as an important cause of poorly controlled hypertension. We were therefore surprised not finding any association between unfavourable BP control and drug adherence (paper III). The total number of patients who reported low (9%) and medium (29%) adherence was also lower than reported in previous studies of CHD patients. A recent meta-analysis estimated that about 50% of the hypertensive patients had stopped taking their BP lowering drugs one year after treatment initiation, but the prevalence of non-adherence in the studies included varied significantly (12-88%). Differences in and limitations of measurement methods to assess drug adherence are probably of great importance for the observed results. Drug adherence has traditionally been monitored by patient self-report questionnaires, as applied in our study (paper III). As previously discussed, a major concern with this indirect method is overestimation of drug intake due to recall bias or patient manipulation.

Potentially, self-report bias leads to falsely high adherence in our study and thus masks a true association between adherence and BP control. However, as we found a strong association between low drug adherence and unfavourable LDL-cholesterol in another NOR-COR paper, the results may potentially also be explained by confounding variables. For example, psychosocial factors are associated with both low drug adherence and lower BP control (Paper III). Assessment of adherence by pill counts, prescription fill rates from pharmacy registries or electronic pillboxes are considered more accurate than self-reported methods. Still, these are also indirect methods without accurate documentation of tablet intake. Direct methods such as directly observed therapy (DOT) or measurement of the actual drug or its metabolite(s) in body fluids, are regarded as the most objective and accurate alternatives for the assessment of adherence today. In a recent study, 35% of patients with apparent therapy resistant hypertension had no detectable drug concentrations in blood by direct chromatography measurement method. Whether this direct method will identify CHD patients with unfavourable BP control that are non-adherent with antihypertensive drugs remains to be tested.

Objective markers for side-effects of BP lowering drugs are lacking and studies identifying such markers would be highly welcome. Therefore, self-reported side-effects were used in the present study. This is a challenging method, as the side-effects of BP lowering drug may constitute a large number of unspecific symptoms (i.e. dizziness or reduced physical capacity) that can also be related to the CHD disease itself, comorbidities or to other drugs. We found that the side-effects of BP
lowering medication were not associated with BP control (paper III), but significantly associated with non-adherence in unadjusted analyses. This is in line with a review, reporting side-effects to be a common barrier to patients’ adherence to BP lowering medication\textsuperscript{98}. Patients experiencing many side-effects might therefore have stopped their medication themselves resulting in poor BP control and fewer reported side-effects. The level of BP lowering drugs in blood is individual and determined by dosage, absorption, distribution, metabolism, excretion, drug-interactions and the liver and kidney function. Altered drug metabolism in the individual patient may be due to CYP polymorphisms, which can lead to reduced or increased effect of consumed medication leading to side effects or poor treatment response (i.e. lack of blood pressure reduction)\textsuperscript{189}. Better insight into these mechanisms is needed to improve the clinical management of side effects and to better understand the role of side-effects for BP control.
8. Conclusions

The majority of CHD patients from routine clinical practice in a representative Norwegian population did not achieve control of the major coronary risk factors. The poorest overall control was found in patients with more than one coronary event, while the youngest patients had the poorest lifestyle control. It is concerning that secondary prevention of CHD fails in a country with a well-developed health care system. The great potential of and urgent need for better management of the established risk factors in clinical practice is thus emphasized.

Three out of five patients who smoked at the time of the index coronary event persisted smoking at follow-up after on average 16 months. Low socio-economic status, duration of smoking, and not having STEMI as index event were associated with persisting smoking, while psychosocial factors were not. Persistent smokers seemed to be aware of the risk of smoking and reported high motivation for smoking cessation. Still, 57% of the smokers reported to not have been offered any assistance with smoking cessation including prescription of pharmacological aids. Thus, there is a considerable potential for better implementation of smoking cessation programmes with prescription of pharmacological aids. CHD patients who smoke should probably be identified and referred systematically to cessation programmes at the time of the index event.

BP control was insufficient as almost half of the CHD patients did not achieve the recommended BP target at follow-up. Older age, obesity and diabetes were the major factors associated with unfavourable BP control, while low socio-economic status, psychosocial factors, and, surprisingly, low self-reported drug adherence did not predict failure to control BP. Prescription of BP lowering drugs in hypertensive patients seems suboptimal. Overweight and intensified drug treatment thus emerges as the major factors to target in order to improve BP control.

The quality of secondary prevention of CHD patients in clinical practice, beyond academic centres, seems to be suboptimal. Clinicians and researchers should therefore recognize this major public health challenge and strive to further improve long-term CR, as even modest improvement in the established cardiovascular risk factors will reduce morbidity, mortality and healthcare costs significantly. By analysing the comprehensive and interdisciplinary data set as an integrated unity, we have demonstrated that different modifiable and non-modifiable factors influence each of the major cardiovascular risk factors smoking and BP. We therefore suggest that secondary preventive management in clinical practice and future intervention studies should address the respective factors most clearly associated with the particular risk factor, and be more individually tailored to the patient’s underlying socio-demographic, medical and psychosocial risk profile.
9. Proposal for future research

In accordance with most other comparable studies, the coronary risk factors and the predictors are analysed in a cross-sectional design with one measurement point in time. Serially conducted cross-sectional studies like EuroAspire in Europe and the National Health and Nutrition Examination Survey in the US provide information on how risk factor control develops over time at the population level. The changes of risk factor control at the CHD patient level, however, are not addressed in these models. Therefore, long-term follow-up data of lifestyle behaviour and risk factor management is urgently needed in order to improve the quality and outcome of care in CHD patients. The natural course of coronary risk factors and the predictors of risk factor control over time thus remain largely unexplored. The relative importance of coronary risk factor control and other patient and healthcare factors on cardiac prognosis also needs to be defined.

The unfavourable risk-factor control demonstrated in both European studies and in the present study clearly emphasises the need for the development and implementation of more effective hospital-based CR programmes as well as interventions in order to strengthen the secondary prevention in the primary care setting. National data on participation, content and duration of CR programmes are needed. Discharge routines including patient information and transition of information between cardiac wards and general practitioners are potentially important factors that should be addressed in future studies.

The present findings encourage further randomized studies to model interventions that facilitate smoking cessation by targeting smoking addiction and ensuring systematic referral to CR and other behavioural interventions that use motivational interview techniques. Future studies should also model interventions to facilitate cessation of smoking, e.g. including prescription of pharmacological aids, and to provide sufficiently potent cardiovascular drugs. Adherence and side-effects needs to be appropriately addressed.

A main problem in treatment of hypertension is thought to be non-adherence. Non-adherence is often measured by self-report, which is hampered by recall bias and the desire to give socially acceptable answers. Only 9% of patients in the NOR-COR study reported low drug adherence. In a small study of patients with apparent treatment resistant hypertension using a directly chromatography measurement method, 35% had no detectable drug concentrations in blood. Whether a chromatography measurement method will identify more CHD patients non-adherent to BP lowering drugs remains to be explored. Further knowledge of potentially modifiable factors associated with non-adherence with cardiovascular drugs may prove to be useful for the development of long-term interventions that target non-adherence and thus risk factor control in CHD patients.
10. References


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Appendix 1

Paper I-III
Unfavourable risk factor control after coronary events in routine clinical practice

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Abstract

Background: Risk factor control after a coronary event in a recent European multi-centre study was inadequate. Patient selection from academic centres and low participation rate, however, may underscore failing risk factor control in routine clinical practice. Improved understanding of the patient factors that influence risk factor control is needed to improve secondary preventive strategies. The objective of the present paper was to determine control of the major risk factors in a coronary population from routine clinical practice, and how risk factor control was influenced by the study factors age, gender, number of coronary events, and time since the index event.

Methods: A cross-sectional study determined risk factor control and its association with study factors in 1127 patients (83% participated) aged 18-80 years with acute myocardial infarction and/or revascularization identified from medical records. Study data were collected from a self-report questionnaire, clinical examination, and blood samples after 2-36 months (median 16) follow-up.

Results: Twenty-one percent were current smokers at follow-up. Of those smoking at the index event 56% continued smoking. Obesity was found in 34%, and 60% were physically inactive. Although 93% were taking blood-pressure lowering agents and statins, 46% were still hypertensive and 57% had LDL cholesterol >1.8 mmol/L at follow-up. Suboptimal control of diabetes was found in 59%. The patients failed on average to control three of the six major risk factors, and patients with >1 coronary events (p < 0.001) showed the poorest overall control. A linear increase in smoking (p < 0.01) and obesity (p < 0.05) with increasing time since the event was observed.

Conclusions: The majority of coronary patients in a representative Norwegian population did not achieve risk factor control, and the poorest overall control was found in patients with several coronary events. New strategies for secondary prevention are clearly needed to improve risk factor control. Even modest advances will provide major health benefits.

Keywords: Secondary prevention, Coronary heart disease, Risk factors, Guidelines

Background

Over the recent years, there has been a decline in mortality rates worldwide [1] leaving a large number of coronary heart disease (CHD) patients in need of optimal secondary prevention. A positive trend in acute myocardial event rates and recurrences from 1994-2009 were also found in Norway [2]. The association between modifiable risk factors and CHD is overwhelmingly documented [3], likewise the benefit of achieving risk factor control to reduce the risk of subsequent events [3, 4]. Despite evidence-based guidelines [5] and cardiac rehabilitation programs for more than 20 years, the EuroAspire studies revealed that the implementation of secondary prevention is far from optimal, with increasing prevalence of smoking in patients <50 years, physical inactivity, obesity and diabetes [6, 7]. In the European cohort of the REACH Study (2003-2004), 40% of symptomatic cardiovascular disease patients had poor control of at least three of the five risk factors assessed [8]. In the Clarify study conducted a decade later, some
improvements were found, but even in Europe, the best region, 50% did not achieve risk factor control [9].

Even though the abovementioned studies provide valuable data on the quality of secondary prevention, patient selection could potentially be a matter of concern. In EuroAspire IV [6] patient inclusion was conducted mainly from academic centres, with potentially better secondary prevention than general cardiac practice. Furthermore, the average interview rate was 49%, and the remaining non-participants were probably more likely to have an even poorer risk factor control. In other multinational studies [9–11], patient identification and inclusion has been conducted at outpatient clinics, often specialist centres, and patients attending them may be more concerned about their health. Previous prevalence estimates thus most likely overestimate adherence to guidelines in the general population of CHD patients. Estimates based on studies of everyday clinical practice are clearly needed.

The reasons for unhealthy lifestyle and low risk factor control are complex and poorly understood and the identification of patient and healthcare factors of importance for coronary risk profile remains a public health priority [5]. The overall aim of the The NOrwegian CORonary (NOR-COR) Prevention Study is to identify medical, and psychosocial factors associated with unfavourable risk factor control after a cardiovascular event. The present paper determines control of the six major coronary risk factors based in routine clinical practice, and identifies the influence of age, gender, number of coronary events, and time since the index event.

Methods
Design and population
The design, methods, and baseline characteristics of the NOR-COR Study have been described elsewhere [12]. Briefly, 1789 consecutive patients aged 18-80 years with a first or recurrent coronary event defined as acute myocardial infarction, coronary artery by-pass graft operation, or percutaneous coronary intervention (PCI) were identified from hospital discharge lists from 2011-14. In patients with recurrent coronary events, the index event was defined as the last event recorded prior to the time of study inclusion. Of these patients, 423 were excluded due to cognitive impairment (n = 28), psychosis (n = 18), drug abuse (n = 10), short life expectancy (n = 136), deaf (n = 160), not able to understand Norwegian (n = 44), and other (n = 27). Of the remaining 1366 invited patients, 1127 (83%) participated in attending a clinical visit and completing a comprehensive questionnaire [12] after 2-36 months (median 16) follow-up. The frequency of missing values for the questionnaire based data was low, within the range from 0 - 10%.

The study was conducted at two Norwegian hospitals (Drammen and Vestfold) with a total catchment of 380,000 inhabitants corresponding to 7.4% of the Norwegian population. The catchment area has a representative blend of city and rural districts and is representative of Norwegian geography, economy, age distribution, morbidity, and mortality [13]. The cardiac rehabilitation program at Drammen Hospital includes a multi-disciplinary one day “heart school”, and exercise training twice per week for 6 weeks. The Hospital of Vestfold provides comprehensive lifestyle intervention described elsewhere [14].

Ethics, consent and permission
The study was approved by the Regional Committee of Ethics in Medical Research. All patients signed a written informed consent prior to study participation.

Study assessments
Medication and co-morbidity at the index event were registered from the hospital medical records. Cardiovascular medication, risk factors and study factors at follow-up were obtained from the self-report questionnaire, the clinical examination and blood-samples. All blood samples were analysed at Drammen hospital. Diet was assessed by a brief diet questionnaire including seven selected quantitative questions (the frequency of intake of different types of foods and beverages). These questions have been validated against intake of matching food groups [15]. Time since the index coronary event was calculated from index event to the date of study inclusion. Low education was defined by completion of primary- and secondary school only.

Major coronary risk factors

- **Smoking**: categorized as current, former or never.
- **Overweight and obesity**: Body weight was measured in light clothes without shoes (SECA 813, DE). Height was measured using a wall fixed mechanical measuring rod (SECA 264, DE). Overweight and obesity was defined as body mass index (BMI) >25 kg/m² and >30 kg/m², respectively. Waist circumference was measured with a non-stretchable tape (SECA 201, DE). A waist circumference above 94 cm and 102 cm in men and above 80 cm and 88 cm in women was defined as central overweight and obesity, respectively.
- **Physical activity**: assessed by frequency (never, <1 time weekly, 1 time weekly, 2-3 times weekly and almost every day), intensity (light, medium and vigorous), and duration (<15 min, 15-29 min, 30-60 min and >60 min). Low physical activity was defined
as less than moderate activity level for 30 min of 2-3 times a week.

- **Blood Pressure (BP) control**: BP was measured after standard procedures using a Welch Allyn digital sphygmomanometer. Unfavourable BP control was defined as BP > 140/90 mmHg (>140/80 mmHg in diabetics).

- **Blood-sugar control**: assessed by HbA1c analysed - Tosoh G8, Ca, US. Unfavourable blood sugar control was defined as HbA1c ≥6.1% (non-diabetics) and >7.0% (diabetics) [5].

- **Low density lipoprotein (LDL) cholesterol**: analysed - Architect ci16200, Ca, US. Elevated LDL cholesterol was defined > 1.8 mmol/l [5].

**Statistics**

Statistical analyses have been performed using SPSS version 21. Parametric descriptive statistics were applied. Binary logistic regression analysis was used to calculate odds ratios (ORs) for unfavourable risk factor control and adjusted for age, gender, number of coronary events, and time since the index event.

General Linear Model (ANCOVA) was used to estimate marginal means for number of unfavourable risk factors (smoking, BMI, physical inactivity, BP, LDL cholesterol, and HbA1c) by age, gender and number of coronary events with all independents controlled as dummies simultaneously, and with time since event entered as a linear covariate.

**Results**

Baseline characteristics are presented in Table 1. Myocardial infarction and stable CHD was the index event in 80% and 20% of the patients, respectively. Angiography was performed in all patients but one, and 90% were revascularized. Patients with >1 coronary event amounted to 30% with a median number of events of 2 (range 2-11). In this group, the proportion of patients with diabetes was more than twice that seen among those with one event only (28% vs. 12%, p < 0.001).

The prescription rate of recommended preventive drugs [5] was high at discharge. All the patients treated with PCI were prescribed dual anti-platelet treatment. At follow-up, there was a small reduction in the use of beta-blockers (from 85 to 72%) and angiotensin converting enzyme inhibitor (ACEI)/angiotensin receptor blocker (ARB) (from 56 to 50%), while the proportions that used at least one statin (93%) and anti-platelet agent (97%) were almost identical. At the time of follow-up, 50% of the patients had attended cardiac rehabilitation.

The proportion of unfavourable risk factors at follow-up was high (Fig. 1). Of those who smoked at baseline, 56% continued to do so. The majority of patients (84%) had an increased waist circumference, and 60% had central obesity. Ninety-three per cent of the patients used at least one BP lowering drug at discharge after the index event (Table 1), and the same percentage reported use of statin at follow-up. However, the frequency of elevated BP and LDL cholesterol at follow-up were still high. Of the diabetic patients 59% had HbA1c >7% although 79%

| Table 1 Characteristics of the patients (n = 1127) at the time of the index coronary event |
|-----------------------------------------------|-------------------|
| Mean age at index event (Standard Deviation) | 61.6 (9.6)        |
| Women (%)                                      | 21                |
| Smoking (%)                                    | 35                |
| Diagnoses                                      |                   |
| ST-elevation infarction (%)                    | 30                |
| Non ST-elevation infarction (%)                | 50                |
| Stable or unstable angina (%)                  | 20                |
| More than 1 coronary event (%)                 | 30                |
| Angiographic findings                          |                   |
| No significant stenoses (%)                    | 6                 |
| Singel vessel disease (%)                      | 55                |
| Multi-vessels disease (%)                      | 39                |
| Intervention                                   |                   |
| PCIa with stent (%)                            | 75                |
| PCIa without stent (%)                         | 2                 |
| Coronary artery bypass graft operation (%)     | 13                |
| No intervention (%)                            | 10                |
| Previous or ongoing participation in cardiac rehabilitation (%) | 50 |
| Co-morbidity                                   |                   |
| Hypertension (%)                               | 43                |
| Diabetes type I or II (%)                      | 17                |
| Heart failure (%)                              | 13                |
| Atrial fibrillation (%)                        | 9                 |
| Stroke or transitory ischemic attack (%)       | 7                 |
| Peripheral artery disease (%)                  | 9                 |
| Medication at discharge after the index event  |                   |
| Aspirin (%)                                    | 99                |
| Other antiplateles (%)                         | 88                |
| Statins (%)                                    | 96                |
| Beta blockers (%)                              | 85                |
| ACE inhibitors or ARBb (%)                     | 56                |
| Calcium channel blockers (%)                   | 16                |
| Diuretics (%)                                  | 22                |
| Antidiabetic (%)                               | 11                |
| Insulin (%)                                    | 4                 |
| Wafarin or NOACc (%)                           | 7                 |

All information was obtained from the hospital medical records

aPercutaneous coronary intervention, bACE, angiotensin converting enzyme; ARB, angiotensin receptor blocker. cNOAC, new oral anticoagulants
used blood sugar lowering medication. In patients without known diabetes, 21% had an HbA1c value ≥ 6.1% and of these patients 8% had HbA1c ≥ 6.5% indicating diabetes [16]. The proportion that reported to eat fish less than 3 times a week was 46%, while 62% ate fruits or vegetables less than two times daily, and 40% less than once daily. Current smoking (25% vs. 12%, p < 0.001) and physical inactivity (64% vs 34%, p < 0.001) were significantly more frequent in patients with low vs. high education, while overweight, unfavorably blood pressure, blood glucose and LDL cholesterol control were not. The estimated marginal means for number of unfavourable risk factors [5] by gender, age and number of coronary events are shown in Fig. 2. On average, the patients had three of the six measured risk factors not at target according to guideline recommendations [5]. Less than 2% achieved control for all risk factors, while 62% had three or more unfavourable risk factors. Patients with more than one coronary event (β 0.43, p < 0.001) had the poorest overall risk factor control.

Multi-adjusted odds ratios (OR) for unfavourable coronary risk factors at follow-up by age, gender, number of coronary events, and time since the index event are shown in Additional file 1. Current smoking (p < 0.001), obesity (p < 0.001) and elevated HbA1c (p < 0.01) were significantly more frequent in the younger patients, while inadequate BP control (p < 0.001) was more frequent with increasing age. ORs for current smoking, low physical activity, and LDL >1.8 mmol/l were significantly higher in women compared to men. ORs for low physical activity, obesity, and elevated LDL cholesterol were significantly higher in patients with several coronary events. There were no significant differences in ORs between the four time groups since the index event, but for smoking (p < 0.01) and obesity (p < 0.05) the test for linear trend was statistically significant with reduction in risk factor control with increasing time since the event.

Discussion

Of the CHD patients included from a high income country with a well-developed health care system [17], the majority had a poor risk factor control and thus did not achieve adequate secondary prevention. There were high proportions of current smoking, obesity and physical inactivity. Blood pressure, cholesterol and blood sugar control were inadequate despite the high reported use of medications. Only a minority of patients (<2%) fulfilled the guidelines recommendations [5] for all coronary risk factors, and more than half of them had inadequate control of three or more risk factors. The measured study factors influenced risk factor control with the poorest overall lifestyle control in the youngest patients. Patients with several previous CHD events had the poorest overall coronary risk factor control. There was a higher prevalence of smoking and obesity with increasing time since the coronary event.

There are certain limitations of the study. First, the coronary risk factors and study factors were measured at one point in time and thus are prone to measurement and recall bias. Moreover, diet is calculated by a semi-quantitative measure, only. Our questions about physical activity have
been validated [18], and we have chosen cut off values as close as possible to guidelines recommendations. Information about the number and the different types of antiplatelet agents at follow-up is not available. The routine clinical setting and the high participation rate (83%) are important strengths of the study. The time span from the index event to follow-up was 2-36 months allowing us to assess how time influences risk factor control. This might impose a selection bias by survival effect. The contribution of excluded patients due to death and short life expectancy is, however, quite similar among the groups with an index event within one year (33%), two years (34%), and three years (33%), respectively, prior to inclusion. Thus, the risk for bias by survival should be minor.

The latest EuroAspire Study [6] had similar inclusion criteria and age distribution as the NOR-COR Study, and in comparison they found a higher proportion of LDL cholesterol >1.8 mmol/l (81% vs. 57%) and diabetes (27% vs. 17%), but fewer diabetic patients had HbA1c >7% (48% vs. 59%). Low physical activity was defined differently, but both studies showed that low physical activity was predominant (60% vs. 60%). The frequencies of hypertension (43% vs. 46%), obesity (38% vs. 34%), and central obesity (58% vs. 60%) were quite similar. The proportion of current smoking was significantly higher (21%) in our CHD population compared to both EuroAspire IV [6] (16%), and other international studies [9–11, 19] (12-18%). Statistics from OECD indicate a lower prevalence in Norway versus average EU regarding smoking (19% vs. 23% [average EU]) [20] and obesity (10% vs. 18% [OECD average]) [21]. It is therefore a paradox that a higher rate of smoking was found among CHD patients in Norway compared to Europe, while the rate of obesity was quite similar. This paradox can be explained by the aforementioned risk of selection bias [6, 7] and by the contribution of non-responders. In the present study with high participation rate from routine clinical practice, these factors are to a higher degree accounted for. There is an ample risk that previous studies [6, 7] have underestimated the prevalence of smoking and obesity in CHD patients.

We found a higher use of anti-platelets (97% vs. 94%), and statins (93% vs. 86%), but lower use of beta-blockers (72% vs. 83%) and ACEI/ARBs (50% vs. 75%) compared with EuroAspire IV [6]. However, there were significant differences in the use of these drugs in various European countries [6].

Large studies from different regions worldwide have also demonstrated that 30-80% of CHD patients had diabetes, were obese, and had LDL cholesterol and BP above the recommended targets [9–11]. In the REACH Registry, one-year risk of subsequent cardiovascular events was inversely related to risk factor control [22], emphasizing the importance of reaching these treatment goals.

The reasons for the low adherence to secondary prevention are complex and multi-factorial [5, 23]. Low socioeconomic status is known to affect both risk factor control and the course of CHD negatively [24, 25], and we confirmed the well-known association between low
education and unfavourable lifestyle. Psychosocial factors such as anxiety, depression, type-d personality and lack of social support may affect both etiological factors, lifestyle and adherence, and are associated with adverse outcomes in CHD patients [26]. Furthermore many revascularized patients have no symptoms. In a recent post PCI study, many patients perceived that they were cured from their CHD [27]. Few reported lifestyle-style post PCI study, many patients perceived that they were revascularized patients have no symptoms. In a recent outcomes in CHD patients [26]. Furthermore many of social support may affect both etiological factors, life-style such as anxiety, depression, type-d personality and lack education and unfavourable lifestyle. Psychosocial factors may act as barriers to lifestyle changes, treatment adherence and may moderate the effects of cardiac rehabilitation [26]. The predictors of good adherence to risk factor control are likely to differ by patient characteristics and risk factors, indicating a need for more tailored interventions [34]. Accordingly, we found different impact of age, gender, education, time since the event, and the number of events on the major risk factors. In the further studies, we aim to explore the relative importance of a number of potentially modifiable factors on risk factor control [12].

**Conclusion**

The majority of CHD patients from a routine clinical practice in a representative Norwegian population did not achieve control of the major coronary risk factors. The measured non-modifiable study factors had different impact on the risk factors, and the poorest overall control was found in patients with several coronary events. It is concerning that secondary prevention of CHD fails in a country with a well-developed health care system. Further knowledge about factors associated with poor risk factor control and strategies for implementation of these factors are strongly needed to improve secondary prevention. Even modest advances will provide major health benefits.

**Additional file**

Additional file 1: Multi-adjusted odds ratio for unfavourable coronary risk factors 2-36 months after the index coronary event. (DOCX 16 kb)

**Abbreviations**

ACE: Angiotensin converting enzyme; ARB: Angiotensin receptor blocker; BMI: Body mass index; BP: Blood pressure; CHD: Coronary heart disease; CI: Confidence interval; LDL: Low density lipoprotein; NOAC: New oral anticoagulant; OR: Odds ratio; PCI: Percutaneous coronary intervention

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**Availability of data and materials**

According to Norwegian legislation, the Norwegian Data Protection Authority, and the Committee of Ethics, we are not allowed to share original study data publicly. However, except for anthropometric data, the other
essential data in which the conclusions in the article are based on will be provided upon request.

**Authors’ contributions**

ES performed the statistical analyses and was responsible for interpretation of data. Furthermore, she drafted the manuscript. KP helped with data interpretation and helped to draft the manuscript. EP helped in the design of the study, helped with interpretation of data, and helped to draft the manuscript. EG participated in the design of the study, helped with interpretation of data, and helped to draft the manuscript. LC participated in the design of the study, helped with interpretation of data, and helped to draft the manuscript. TM helped to perform the statistical analyses, helped with interpretation of data, and helped to draft the manuscript. JD participated in the design of the study, helped with interpretation of data, and helped to draft the manuscript. TD participated in the design of the study, helped with interpretation of data, and helped to draft the manuscript. JM participated in the design of the study, helped to perform the statistical analyses, helped with interpretation of data, and helped to draft the manuscript. All authors read and approved the final manuscript.

**Competing interests**

The authors declare that they have no competing interests.

**Consent for publication**

Not applicable.

**Ethics approval and consent to participate**

All participants gave informed consent before study participation. The NORCOR study was approved by the Regional Committee of Ethics (REK Sør-Ost) on 12 February, 2014 (2013/1885).

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Medical and sociodemographic factors predict persistent smoking after coronary events

Elise Sverre1,2*, Jan Erik Otterstad3, Erik Gjertsen1, Lars Gullestad4, Einar Husebye1, Toril Dammel2, Torbjørn Moum2 and John Munkhaugen1

Abstract

Background: Understanding the determinants of persistent smoking after a coronary event constitutes the basis of modelling interventions of smoking cessation in secondary prevention programs. We aim to identify the potentially modifiable medical, sociodemographic and psychosocial factors, comprising the study factors, associated with unfavourable risk factor control after CHD events.

Methods: A cross-sectional explorative study used logistic regression analysis to investigate the association between study factors and smoking status in 1083 patients hospitalized with myocardial infarction and/or coronary revascularization. Hospital record data, a self-report questionnaire, clinical examination and blood samples were applied.

Results: At the index hospitalization, 390 patients were smoking and at follow-up after 2–36 months 167 (43%) of these had quit, while 230 reported persistent smoking. In adjusted analyses, unemployed or disability benefits (Odds ratio (OR) 4.1), low education (OR 3.5), longer smoking duration (OR 2.3) and not having ST-elevation myocardial infarction (STEMI) as index event (OR 2.3) were significantly associated with persistent smoking. Psychosocial factors at follow-up were not associated with persistent smoking. Smokers reported high motivation for cessation, with 68% wanting help to quit. Only 42% had been offered nicotine replacement therapy or other cessation aids. Smokers rated use of tobacco as the most important cause of their coronary disease (6.8 on a 1–10 Likert scale).

Conclusions: Low socioeconomic status, prior duration of smoking, and not having STEMI as index event were associated with persisting smoking. Persistent smokers in this study seem to have an acceptable risk perception and were motivated to cease smoking, but needed assistance through cessation programs including prescription of pharmacological aids.

Trial registration: Registered at ClinicalTrials.gov: NCT02309255, registered retrospectively.

Keywords: Smoking, Smoking cessation, Coronary heart disease (CHD), Secondary prevention, Sociodemographic factors, Medical risk factors, Psychosocial risk factors
Background

The causal role of cigarette smoking in the development and progression of coronary heart disease (CHD) is overwhelmingly documented [1, 2]. Smoking is the leading avoidable cause of death in the developed world [3] and increases the risk of coronary events [4] and total mortality by up to 50% [1, 4]. Smoking thus remains the single most important cardiovascular risk factor to modify in order to improve prognosis in CHD patients [2–4].

Different smoking cessation programs and pharmacological treatment [5–7] increase the likelihood of cessation in clinical studies. Still, clinical practice across Europe suggests that about half of the daily smokers surviving a coronary event continue smoking [8]. While the prevalence of daily smoking in the general European population decreased substantially over the past decade [9], only a modest reduction (20% to 16%) was seen in CHD patients over the past 20 years in the EuroAspire studies [8, 10]. In the US, the reduction in the number of daily smokers from 1980 to 2000 was 12% in the general population compared to 5% in CHD patients [11]. It is concerning that the prevalence of daily smoking actually has increased among the youngest CHD patients [10].

A complex array of patient and healthcare factors influence smoking behaviour in coronary patients [2, 3, 12–17]. Identification of potentially modifiable medical and psychosocial factors associated with persistent smoking after coronary events could be important for the development of individually tailored interventions of smoking cessation with sustained effect [2, 5, 7, 18].

The NORwegian-CORonary (NOR-COR) Prevention Study identifies sociodemographic, medical, and psychosocial factors, comprising the study factors, associated with unfavourable risk factor control after CHD events (phase I). Moreover, the project aims to target the study factors of importance for risk factor control in tailored interventions (phase II) [18]. The present exploratory analysis aims to identify the study factors associated with persistent smoking in a cross-sectional survey.

Methods

Design and population

The design, methods, and baseline characteristics of the NOR-COR Study have been described elsewhere [18]. A cross-sectional study was conducted at two general Norwegian hospitals (Drammen and Vestfold) with a total catchment area of 380,000 inhabitants, corresponding to 7.4% of the Norwegian population. In total, 1789 consecutive patients aged 18–80 years with a first or recurrent coronary event/treatment (i.e. acute myocardial infarction, coronary artery by-pass graft operation, and/or percutaneous coronary intervention) were identified from hospital discharge lists over the three years (2011–14) prior to study inclusion. The index event was defined as the last coronary event prior to inclusion. Of the identified patients, 423 were excluded due to cognitive impairment (n = 28), psychosis (n = 18), drug abuse (n = 10), short life expectancy (n = 136), death (n = 160), not being able to understand Norwegian language (n = 44), and other (n = 26). Of the remaining 1366 eligible patients, 1127 (83%) consented to participate in attending a clinical visit and completing a comprehensive questionnaire [18] at 2–36 months follow-up after the index event. Smoking status at follow-up was missing in 44 patients, leaving 1083 in the final analyses. As smoking status at the index event was missing in seven patients who reported daily smoking at follow-up, there is thus a minor discrepancy between the proportion of smokers reported at index event and at follow-up.

All participants gave informed consent before study participation. The NOR-COR study was approved by the Regional Committee of Ethics (REK Sør-Øst) 12. February, 2014 (2013/1885).

Outcome assessment

The primary outcome variable was smoking status at follow-up compared to smoking status at the index event, categorized as persistent smoker vs. quitter. Smoking status at index event was recovered from hospital medical records. Patients who smoked at the time of the index event were categorized as current smokers. Smoking at follow-up was recorded from the self-report questionnaire. All patients who reported daily smoking [cigarettes (n = 225), pipes (n = 0) or cigars (n = 5)] were categorized as persistent smokers.

Covariates (study factors)

Study data registered at the time of the index event

Demographic variables, risk factors, somatic comorbidity summarized according to the Charlson comorbidity index [19] and information about the index coronary event and treatment were registered from hospital medical records [18].

Study factors registered at follow-up 2–36 months after the index event

Sociodemographic factors included marital status, education, and employment status. Medical factors included coronary risk factors and cardiovascular medication. Psychosocial factors included anxiety and depression (Hospitality Anxiety and Depression Scale) [20], Type D personality [21], worry (Penn State Worry Questionnaire) [22], insomnia (Bergen Insomnia Scale) [23], illness perception (Brief illness perception questionnaire) [24] and perceived risk [25]. Moreover, information about smoking behaviour, motivation and treatment needs were obtained by the self-report questionnaire [18]. Participation in cardiac rehabilitation programs was based on hospital
medical records, including lists from the cardiac rehabilitation departments.

Statistics
Using SPSS version 21, data were analysed in a cross-sectional design with the study factors as the main exposure variables and smoking status (persistent smokers vs. quitters) at follow-up as the main outcome variable. The distribution of study factors according to smoking status (never smoker, former smoker, current smoker) at the index event and at follow-up (persistent smokers, quitters) were reported as frequencies and percentages. A forward, stepwise binary logistic regression analysis was used to calculate crude and multi-adjusted odds ratio (OR) and 95% confidence intervals (CI) for study outcomes with interaction terms between independent variables as indicated. The level of significance was set to \(p < 0.05\). Covariates with \(p\)-values between 0.05 and 0.1 in crude analyses were also selected as candidates for the multivariate analyses since more traditional levels (i.e. 0.05) can fail to identify variables that might turn out to be significant when actually included in the adjusted models.

Results
Characteristics of the study population at the time of the index event are presented in Table 1. Mean age was 62 (SD 10) years and 21% were women. A total of 390 (36%) patients were smoking at the time of the index event. Compared to former and never smokers, the group of current smokers consisted of more females than males, and were characterized as a group by their younger age, lower education levels, more often having ST-elevation myocardial infarction (STEMI) as index event as well as having fewer previous coronary events.

Sociodemographic, medical, and psychosocial factors in persistent smokers and quitters at follow-up 2–36 (mean 16) months after the index event are shown in

### Table 1 Patient characteristics according to smoking status at the index coronary event

<table>
<thead>
<tr>
<th>Study factors</th>
<th>Never smoker (n = 250)</th>
<th>Former smoker (n = 436)</th>
<th>Current smoker (n = 390)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sociodemographic factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age at index event, mean (SD)</td>
<td>63.3 (10.3)</td>
<td>63.2 (8.7)</td>
<td>58.5 (9.5)</td>
</tr>
<tr>
<td>Number of months since the index event, mean (SD)</td>
<td>16.5 (10.4)</td>
<td>16.5 (10.2)</td>
<td>18.2 (10.9)</td>
</tr>
<tr>
<td>Female sex, n (%)</td>
<td>51 (20.4)</td>
<td>79 (18.1)</td>
<td>97 (24.9)</td>
</tr>
<tr>
<td>Ethnic minority background, n (%)</td>
<td>12 (4.8)</td>
<td>8 (1.8)</td>
<td>11 (2.8)</td>
</tr>
<tr>
<td>Low education, n (%)</td>
<td>156 (62.4)</td>
<td>291 (66.7)</td>
<td>296 (75.9)</td>
</tr>
<tr>
<td><strong>Medical factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary index event and treatment:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ST-elevation infarction, n (%)</td>
<td>54 (21.6)</td>
<td>102 (23.4)</td>
<td>167 (42.8)</td>
</tr>
<tr>
<td>Non-ST-elevation infarction, n (%)</td>
<td>137 (54.8)</td>
<td>218 (50.0)</td>
<td>182 (46.7)</td>
</tr>
<tr>
<td>Stable or unstable angina, n (%)</td>
<td>59 (23.6)</td>
<td>116 (26.6)</td>
<td>44 (11.3)</td>
</tr>
<tr>
<td>More than 1 coronary event, n (%)</td>
<td>71 (28.4)</td>
<td>151 (34.6)</td>
<td>97 (24.9)</td>
</tr>
<tr>
<td>Comorbidity:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Charlson co-morbidity sum score, mean (SD)</td>
<td>4.0 (1.3)</td>
<td>4.3 (1.5)</td>
<td>3.9 (1.4)</td>
</tr>
<tr>
<td>Heart failure, n (%)</td>
<td>21 (8.4)</td>
<td>65 (14.9)</td>
<td>54 (13.8)</td>
</tr>
<tr>
<td>Stroke or transient ischemic attack, n (%)</td>
<td>14 (5.6)</td>
<td>39 (8.9)</td>
<td>24 (6.2)</td>
</tr>
<tr>
<td>Peripheral artery disease, n (%)</td>
<td>12 (4.8)</td>
<td>39 (8.9)</td>
<td>42 (10.8)</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease, n (%)</td>
<td>2 (0.8)</td>
<td>45 (10.3)</td>
<td>47 (12.1)</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>129 (51.6)</td>
<td>231 (53.0)</td>
<td>249 (63.8)</td>
</tr>
<tr>
<td>Diabetes, n (%)</td>
<td>35 (14.0)</td>
<td>92 (21.1)</td>
<td>49 (12.6)</td>
</tr>
<tr>
<td><strong>Treatment at hospital discharge</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin, n (%)</td>
<td>247 (98.8)</td>
<td>425 (97.5)</td>
<td>387 (99.2)</td>
</tr>
<tr>
<td>Additional antiplatelet therapy, n (%)</td>
<td>212 (84.8)</td>
<td>373 (85.6)</td>
<td>360 (92.3)</td>
</tr>
<tr>
<td>Statins, n (%)</td>
<td>239 (95.6)</td>
<td>414 (95.0)</td>
<td>379 (97.2)</td>
</tr>
<tr>
<td>ACEI(^a) or ARBs(^b), n (%)</td>
<td>141 (56.4)</td>
<td>249 (57.1)</td>
<td>207 (53.1)</td>
</tr>
<tr>
<td>Beta-blockers, n (%)</td>
<td>212 (84.8)</td>
<td>373 (85.6)</td>
<td>329 (84.4)</td>
</tr>
</tbody>
</table>

\(^a\)ACEI, angiotensin converting enzyme inhibitor
\(^b\)ARB angiotensin receptor blocker
At follow-up, 167 (43%) of the registered smokers at the index event had quit smoking, while 230 patients reported persistent smoking. Almost all persistent smokers had been smoking for 20 years or more, and 60% had been smoking for more than 40 years. Seventy-three percent of the persistent smokers reported having reduced their cigarette use since the index event.

In bivariate analyses, low education, living alone, unemployed or disability benefit, longer smoking duration, more than one coronary event prior to the index event, non-participation in cardiac rehabilitation, not having STEMI as index event, low physical activity, and no prescription of statins were significantly more prevalent in persistent smokers than in quitters. No significant differences in psychosocial factors were found between persistent smokers and quitters. These findings were consistent in sub-group analyses by age and gender.

The persistent smokers reported a high motivation (average 7.8 on a 1–10 Likert scale) for smoking cessation and only 14% reported low (≤3) motivation. Sixty-eight percent wanted help to quit smoking. However, only 42% reported having been offered nicotine replacement therapy or any other form of cessation aid. In total, 35% of the persistent smokers and 27% of the quitters ($p = 0.14$).

<table>
<thead>
<tr>
<th>Study factors</th>
<th>Quitted after the index event ($n = 167$)</th>
<th>Persistent smokers ($n = 230$)</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sociodemographic factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age at index event, mean (SD)</td>
<td>57.7 (9.4)</td>
<td>59.3 (9.3)</td>
<td>$p = 0.05$</td>
</tr>
<tr>
<td>Number of months since the index event, mean (SD)</td>
<td>16.8 (10.9)</td>
<td>18.9 (10.8)</td>
<td>$p = 0.05$</td>
</tr>
<tr>
<td>Female sex, n (%)</td>
<td>38 (22.8)</td>
<td>56 (24.3)</td>
<td>$p = 0.71$</td>
</tr>
<tr>
<td>Ethnic minority background, n (%)</td>
<td>4 (2.4)</td>
<td>10 (4.3)</td>
<td>$p = 0.30$</td>
</tr>
<tr>
<td>Living alone, n (%)</td>
<td>28 (16.8)</td>
<td>57 (24.8)</td>
<td>$p &lt; 0.05$</td>
</tr>
<tr>
<td>Low education, n (%)</td>
<td>116 (69.5)</td>
<td>189 (82.2)</td>
<td>$p &lt; 0.001$</td>
</tr>
<tr>
<td>Unemployed or on disability benefits, n (%)</td>
<td>33 (19.8)</td>
<td>85 (37.0)</td>
<td>$p &lt; 0.001$</td>
</tr>
<tr>
<td><strong>Medical factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration of smoking, years, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 20</td>
<td>11 (6.6)</td>
<td>8 (3.5)</td>
<td>$p &lt; 0.001$</td>
</tr>
<tr>
<td>20–39</td>
<td>99 (59.3)</td>
<td>64 (27.8)</td>
<td></td>
</tr>
<tr>
<td>&gt; 40</td>
<td>53 (31.7)</td>
<td>137 (59.6)</td>
<td></td>
</tr>
<tr>
<td>ST-elevation infarction/non ST-elevation infarction and angina, n (%)</td>
<td>78 (46.7)/ 89 (53.3)</td>
<td>84 (36.5)/ 146 (63.5)</td>
<td>$p &lt; 0.05$</td>
</tr>
<tr>
<td>More than 1 coronary event, n (%)</td>
<td>30 (18.0)</td>
<td>77 (33.5)</td>
<td>$p &lt; 0.001$</td>
</tr>
<tr>
<td>Participation in cardiac rehabilitation, n (%)</td>
<td>95 (56.9)</td>
<td>103 (44.8)</td>
<td>$p &lt; 0.05$</td>
</tr>
<tr>
<td>Charlson co-morbidity sum score, mean (SD)</td>
<td>3.8 (1.3)</td>
<td>4.0 (1.4)</td>
<td>$p = 0.14$</td>
</tr>
<tr>
<td>Use of antplatelets at follow-up, n (%)</td>
<td>164 (98.2)</td>
<td>221 (96.1)</td>
<td>$p = 0.22$</td>
</tr>
<tr>
<td>Use of statins at follow-up, n (%)</td>
<td>160 (95.8)</td>
<td>207 (90.0)</td>
<td>$p &lt; 0.05$</td>
</tr>
<tr>
<td>Low physical activity$^b$, n (%)</td>
<td>93 (55.7)</td>
<td>186 (80.9)</td>
<td>$p &lt; 0.001$</td>
</tr>
<tr>
<td>Body Mass Index &gt;30 kg/m², n (%)</td>
<td>61 (36.5)</td>
<td>58 (25.2)</td>
<td>$p &lt; 0.05$</td>
</tr>
<tr>
<td>Low density lipoprotein cholesterol &gt;1.8 mmol/l, n (%)</td>
<td>88 (52.7)</td>
<td>124 (53.9)</td>
<td>$p = 0.66$</td>
</tr>
<tr>
<td>Blood pressure &gt; 140/90 (140/80 diabetes) mmHg, n (%)</td>
<td>60 (35.9)</td>
<td>74 (32.2)</td>
<td>$p = 0.74$</td>
</tr>
<tr>
<td>Diabetes, n (%)</td>
<td>16 (9.6)</td>
<td>36 (15.7)</td>
<td>$p = 0.08$</td>
</tr>
<tr>
<td><strong>Psychosocial factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital Anxiety and Depression Score - depression ≥11, n (%)</td>
<td>12 (7.2)</td>
<td>13 (5.7)</td>
<td>$p = 0.61$</td>
</tr>
<tr>
<td>Hospital Anxiety and Depression Score - anxiety ≥11, n (%)</td>
<td>22 (13.2)</td>
<td>27 (11.7)</td>
<td>$p = 0.81$</td>
</tr>
<tr>
<td>Type D personality disorder, n (%)</td>
<td>41 (24.6)</td>
<td>56 (24.3)</td>
<td>$p = 0.89$</td>
</tr>
<tr>
<td>Worry score (PSWQ$^c$), mean (SD)</td>
<td>40.1 (13.9)</td>
<td>40.3 (13.9)</td>
<td>$p = 0.87$</td>
</tr>
<tr>
<td>Insomnia$^d$, n (%)</td>
<td>80 (47.9)</td>
<td>114 (49.6)</td>
<td>$p = 0.65$</td>
</tr>
</tbody>
</table>

$^a$2–36 months after the index coronary event
$^b$Physical activity less than 30 min of moderate activity 2–3 times weekly
$^c$Worry was assessed by the Penn State Worry Questionnaire (PSWQ), a 16 item measure of pathological worry
$^d$Measured by Bergen Insomnia Scale
reported having no current follow-up for their CHD in primary or specialist healthcare. Smoking was rated as the most important cause of CHD by both persistent smokers and quitters, but quitters rated the importance of smoking as risk factor higher (7.5 vs. 6.8 on a 1–10 Likert scale, \( p < 0.05 \)). Persistent smokers felt they had a higher likelihood of having a heart attack within the next 12 months than quitters (3.3 vs. 2.5 on a 1–10 Likert scale, \( p < 0.01 \)). They also felt they could do less to help reduce that risk (6.4 vs. 7.0 on a 1–10 Likert scale, \( p < 0.05 \)). Compared to quitters, persistent smokers thought they would have to restrict their daily activities more in the long-term (3.0 vs. 4.2 on a 1–10 Likert scale, \( p < 0.001 \)). Persistent smokers scored significantly lower on treatment control compared to quitters (7.8 vs. 6.6 on a 1–10 Likert scale, \( p < 0.001 \)), while no other differences in illness perception were found. A considerable subgroup of both persistent smokers (21%) and quitters (13%) perceived “no need to change lifestyle”.

The odds ratios for persistent smoking compared to quitting smoking after the index event by study factors are shown in Table 3. In crude analyses, unemployment or disability benefits were the strongest predictors of persistent smoking, followed by longer duration of smoking, low education, >1 coronary event, living alone, no participation in cardiac rehabilitation and not having STEMI as index event. In multi-adjusted analyses the study factors significantly associated with persistent smoking were: unemployment or disability benefits, low education, longer duration of smoking, and not having STEMI as index event.

Discussion

Almost 60% of patients in routine clinical practice persisted smoking 2–36 months after a coronary event. In particular, low socioeconomic status, longer duration of smoking and not having STEMI as index event were associated with persistent smoking in multi-adjusted analyses, while psychosocial factors and participation in cardiac rehabilitation were not. Almost all persistent smokers had been smoking for a long time, with 60% having been smokers for more than 40 years. Most persistent smokers seemed to be aware of the risk associated with smoking and reported a high motivation for cessation. More than 70% of persistent smokers had reduced the number of cigarettes smoked after the index event. However, only 42% reported having been offered any smoking cessation aids, and 35% had no current follow-up in primary or specialist healthcare for their CHD.

Low education, unemployment and the claiming of disability benefits were the factors most strongly associated with persistent smoking, concurring to the well-established inverse association between smoking and socioeconomic status [2, 3, 26]. Even though socioeconomic position is not easily modified, our findings suggest that these patients should be systematically identified during hospitalisation for coronary events and offered smoking cessation programs.

In the present study, STEMI as the coronary index event was significantly associated with quitting smoking. A qualitative study finding that the urgency of managing STEMI bolsters the impression of suffering a life-threatening event could be relevant in this context since such a perception may increase the patient’s motivation and willingness to change lifestyle [27]. In contrast, the initial uncertainty of the diagnosis in non ST-elevation myocardial infarction (NSTEMI) and unstable angina pectoris may lead to reduced appreciation of the seriousness of the condition [27]. The majority of persistent smokers in the present study, however, appear to be aware of the risk associated with smoking, with smoking rated as the most important cause of CHD.

Most persistent smokers reported a high motivation for smoking cessation and 73% reported having reduced their smoking since the coronary event, while almost 70% reported that they wanted immediate assistance with cessation. Even though we have not measured the different stages of change [28], this suggests that these patients have passed the pre-contemplation stage (i.e. no intention to quit). By comparison, 50% of CHD patients smoking at follow-up had passed the pre-contemplation stage in the EuroAspire III study. As recommended in clinical guidelines [2], motivation and readiness for smoking cessation should be assessed in all patients admitted to hospital for coronary events. A long smoking history, the reported reduction in consumption of cigarettes and the high level of motivation for cessation without success suggest substantial nicotine dependency. Nicotine dependency is a well-known barrier to smoking cessation [12, 14], and the longer the duration of smoking, the less likely is a change in smoking behaviour [28]. It is of concern, therefore, that only 42% of the persistent smokers in the present study had been offered cessation aid, which is in accordance with other studies of CHD patients [29, 30]. Smoking receives less attention from cardiologists than other risk factors [12], possibly because physicians believe themselves to possess limited intervention skills in behavioural counselling [16]. It may also reflect a reluctance among health professionals to believe that their patients have the ability to quit smoking. This may partly explain why smoking cessation therapy is not offered as indicated [16].

Surprisingly, none of the measured psychosocial factors (anxiety, depression, worry, insomnia, type D personality) differed between quitters and persistent smokers on average 1.7 years after the index coronary event. Our findings are contrary to studies reporting that individuals with depression are more likely to smoke and less likely to quit.
smoking successfully [31, 32]. Other studies have found no association between persistent smoking and either depression or anxiety [33]. The frequency of smoking in patients with type D personality is higher than in patients without type D in some studies [34], while others have found no differences [35]. The level of psychosocial distress decreases with increasing time since the coronary event [36] and the timing of assessment of psychosocial factors may explain the conflicting association between smoking and psychosocial factors observed in previous studies. Variation in measurement methods used for evaluation of psychological symptoms [32, 37] is another possible explanation. Psychosocial factors were not measured at the index event.

### Table 3

<table>
<thead>
<tr>
<th>Study factors</th>
<th>Model 1 (OR, 95% CI)</th>
<th>p-value</th>
<th>Model 2 (OR, 95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sociodemographic factors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean age at index event (OR per year)</td>
<td>1.02 (1.00–1.04)</td>
<td>p = 0.05</td>
<td>0.97 (0.90–1.03)</td>
<td>p = 0.27</td>
</tr>
<tr>
<td>Time since the index event (OR per year)</td>
<td>1.02 (1.00–1.04)</td>
<td>p = 0.05</td>
<td>1.01 (0.98–1.05)</td>
<td>p = 0.41</td>
</tr>
<tr>
<td>Female gender</td>
<td>1.09 (0.68–1.75)</td>
<td>p = 0.71</td>
<td>2.17 (0.85–5.52)</td>
<td>p = 0.10</td>
</tr>
<tr>
<td>Living alone</td>
<td>1.69 (1.01–2.82)</td>
<td>p = 0.05</td>
<td>1.23 (0.48–3.11)</td>
<td>p = 0.67</td>
</tr>
<tr>
<td>Low education</td>
<td>2.20 (1.36–3.57)</td>
<td>p &lt; 0.001</td>
<td>3.35 (1.43–7.81)</td>
<td>p &lt; 0.01</td>
</tr>
<tr>
<td>Unemployed or on disability benefits</td>
<td>3.01 (1.81–5.02)</td>
<td>p &lt; 0.001</td>
<td>4.12 (1.80–9.41)</td>
<td>p = 0.001</td>
</tr>
<tr>
<td><strong>Medical factors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not having ST-elevation infarction as index event</td>
<td>1.53 (1.02–2.29)</td>
<td>p = 0.05</td>
<td>2.30 (1.08–4.40)</td>
<td>p = 0.05</td>
</tr>
<tr>
<td>More than 1 coronary event</td>
<td>2.30 (1.42–3.72)</td>
<td>p = 0.001</td>
<td>1.53 (0.63–3.72)</td>
<td>p = 0.35</td>
</tr>
<tr>
<td>Participation in cardiac rehabilitation</td>
<td>0.62 (0.41–0.92)</td>
<td>p &lt; 0.05</td>
<td>0.78 (0.38–1.60)</td>
<td>p = 0.50</td>
</tr>
<tr>
<td>Charlson co-morbidity sum score</td>
<td>1.12 (0.96–1.32)</td>
<td>p = 0.14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration of smoking (years)</td>
<td>2.93 (1.62–2.71)</td>
<td>p &lt; 0.001</td>
<td>2.34 (1.41–3.88)</td>
<td>p = 0.001</td>
</tr>
<tr>
<td><strong>Psychosocial factors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital Anxiety and Depression Score - total &gt; 11</td>
<td>1.06 (0.70–1.62)</td>
<td>p = 0.78</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type D personality</td>
<td>1.03 (0.65–1.65)</td>
<td>p = 0.89</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Worry score (PSWQ)</td>
<td>1.00 (0.99–1.01)</td>
<td>p = 0.87</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insomnia</td>
<td>1.10 (0.73–1.65)</td>
<td>p = 0.65</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Perceived risk (1–10 Likert scale)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>What do you feel is the likelihood of having a new heart attack over the next 12 months?</td>
<td>1.15 (1.05–1.25)</td>
<td>p &lt; 0.01</td>
<td>1.01 (0.86–1.18)</td>
<td>p = 0.93</td>
</tr>
<tr>
<td>How much do you feel you can help reduce your risk of having another heart attack?</td>
<td>0.91 (0.84–0.99)</td>
<td>p &lt; 0.05</td>
<td>0.88 (0.76–1.02)</td>
<td>p = 0.09</td>
</tr>
<tr>
<td>How much do you think you will have to restrict your activities in the long-term due to your heart condition?</td>
<td>1.17 (1.08–1.27)</td>
<td>p &lt; 0.001</td>
<td>1.00 (0.87–1.17)</td>
<td>p = 0.90</td>
</tr>
<tr>
<td><strong>Brief Illness Perception Questionnaire (1–10 Likert scale)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>How much does your illness affect your life? (consequences)</td>
<td>0.99 (0.92–1.06)</td>
<td>p = 0.76</td>
<td></td>
<td></td>
</tr>
<tr>
<td>How long do you think your illness will continue? (timeline)</td>
<td>0.98 (0.91–1.04)</td>
<td>p = 0.45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>How much control do you feel you have over your illness? (personal control)</td>
<td>0.95 (0.88–1.02)</td>
<td>p = 0.16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>How much do you think your treatment can help you? (treatment control)</td>
<td>0.80 (0.72–0.88)</td>
<td>p &lt; 0.001</td>
<td>0.88 (0.75–1.02)</td>
<td>p = 0.09</td>
</tr>
<tr>
<td>How much do you experience symptoms from your illness? (identity)</td>
<td>0.83 (0.92–1.07)</td>
<td>p = 0.83</td>
<td></td>
<td></td>
</tr>
<tr>
<td>How concerned are you about your illness? (concern)</td>
<td>0.98 (0.93–1.06)</td>
<td>p = 0.74</td>
<td></td>
<td></td>
</tr>
<tr>
<td>How well do you feel you understand your illness? (understanding)</td>
<td>0.97 (0.89–1.05)</td>
<td>p = 0.41</td>
<td></td>
<td></td>
</tr>
<tr>
<td>How much does your illness affect you emotionally? (emotional response)</td>
<td>0.96 (0.90–1.02)</td>
<td>p = 0.19</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Quitted smoking after the index event is the reference category

- Model 1: crude analyses
- Model 2, multi-adjusted with including all variables with p < 0.1 in crude analysis (adjusted for all variables included in the model)
- Worry was assessed by the Penn State Worry Questionnaire (PSWQ), a 16 item measure of pathological worry
- Measured by Bergen insomnia Scale
and we therefore do not know if these factors predict persistent smoking at follow up.

In accordance with two randomized clinical trials [38, 39], we found no effect of participation in cardiac rehabilitation on smoking cessation in adjusted analyses. The most recent Cochrane review on cardiac rehabilitation programs [40] did not address the effect of cardiac rehabilitation on smoking cessation, while the 2004 review [41] found a small positive effect of cardiac rehabilitation on cessation. Better smoking cessation interventions are urgently needed in clinically implemented cardiac rehabilitation programs [2].

A Cochrane review of tobacco treatment trials found that intensive counselling, initiated during hospitalization, significantly increased quit rates at 12 months follow-up [7]. Adding nicotine replacement therapy further increased quitting compared with counselling alone [7, 17]. The small reduction in daily smoking in CHD patients across Europe over the past few decades [8, 10], however, substantiates the need of novel and better strategies to ensure the implementation of evidenced based cessation programs in clinical practice. Despite a long and heavy smoking history, most patients in this cohort seem to be aware of the risk associated with smoking and were motivated to quit smoking. Hospitalization for a coronary event provides an important opportunity for quitting smoking and the chance of successful smoking cessation has recently been shown to be higher with immediate [42] and abrupt [43] quitting. Effective, proactive counselling tailored to the readiness to quit smoking should therefore be the standard of care for managing all smokers during hospitalization for the index coronary event [17]. Furthermore, systematic referral to outpatient smoking cessation programs adapted to each patient's profile and needs [18] and routinely prescribing pharmacological aids may further facilitate cessation. High risk sub-groups with low socioeconomic status, a long history of smoking and those having less dramatic coronary events such as NSTEMI or angina, are at increased risk of persistent smoking and should receive particular attention and be considered for extended follow-up.

Study limitations and strengths
Smoking and other important study factors were measured by self-reporting, and are thus prone to measurement errors and recall bias. Additional confounders should be considered. Data for the date of smoking cessation, the use of cessation aids among quitters and specific questionnaires addressing nicotine dependence were not available. We do not have enough power to perform multi-adjusted analyses in sub-groups of age and gender, but no important differences in the major study factors were found in unadjusted sub-group analyses. High participation rate (83%), the routine clinical setting and few missing data are important strengths of the study. A reproducibility study of the NOR-COR questionnaire demonstrated highly acceptable test-retest values for all key items and instruments [44].

Conclusion
Low socioeconomic status, prior duration of smoking, and not having STEMI as index event were associated with persistent smoking in coronary patients, while psychosocial factors and participation in cardiac rehabilitation were not. A majority of persistent smokers appeared to be aware of the risk associated with smoking and were motivated for smoking cessation. Given the well-documented benefit of smoking cessation, there is considerable potential for better interventions to facilitate cessation in CHD patients, including systematic referral to cessation programs including prescription of pharmacological aids.

Abbreviations
ACEI: Angiotensin converting enzyme inhibitor; ARB: Angiotensin receptor blocker; CHD: Coronary heart disease; CI: Confidence interval; NSTEMI: Non-ST elevation infarction; OR: Odds ratio; PSWQ: Penn State Worry Questionnaire; SD: Standard deviation; STEMI: ST-elevation myocardial infarction

Acknowledgments
The NOR-COR project originates from the Department of Medicine Drammen Hospital and the study was carried out at Drammen and Vestfold Hospitals. The concept was developed by the project in collaboration with communities at the University of Oslo. The authors thank study patients for participating and study personnel for their invaluable contribution. The authors would also like to thank Matthew McGee, Morbid Obesity Centre, Vestfold Hospital Trust, for proofreading the manuscript.

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Availability of data and materials
According to Norwegian legislation, the Norwegian Data Protection Authority and the Committee of Ethics, we are not allowed to share original study data publicly. However, except for anthropometric data, the other essential data by which the conclusions in the article are based will be provided upon reasonable request from the corresponding author.

Authors’ contributions
TD, LG, EG, EH and JM contributed to the design of the work. ES, TM and JM contributed to the analysis and all authors contributed to the interpretation of data. ES drafted the manuscript. JM, JEO, EG, EH, TD, TM, and LG critically revised the manuscript. All contributors gave final approval and agreed to be accountable for all aspects of the work, thus ensuring integrity and accuracy.

Ethics approval and consent to participate
All participants gave informed consent before study participation. The NOR-COR study was approved by the Regional Committee of Ethics (REK Sør-Ost) 12 February 2014 (2013/1885).

Consent for Publication
Not applicable.

Competing interests
The authors declare that they have no competing interests.
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Appendix 2

The NOR-COR study questionnaire

1. BOSITUASJONEN OG SIVILSTATUS

Bor du alene eller sammen med andre?
Sett ett eller flere kryss.

Bor alene……………………………………………………………………………
Ektefelle eller samboer……………………………………………………..
Separert/skilt……………………………………………………………
Bor for tiden på sykehjem, aldershjem eller liknende…………………..

2. UTDANNING

Hvilken utdanning er den høyeste du har fullført?
Sett ett kryss

Grunnskole 7-10 år, framhaldsskole, folkehøgskole………………………..
Realskole, middelskole, yrkesskole, 1-2 årig videregående skole………
Artium, øk. gymnas, allmennfaglig retning i videregående skole………
Høgskole/universitet, mindre enn 4 år……………………………………
Høgskole/universitet, mer enn 4 år…………………………………………

3. ARBEID/TRYGD

Hva slags arbeidssituasjon har du nå?
Ett eller flere kryss

Lønnet arbeid i 100 % stilling………………………………………………
Lønnet arbeid i redusert stilling………………………………………………
Pensjonist ………………………………………………………………………
Under utdanning………………………………………………………………
Arbeidsledig, permittert………………………………………………………..
Sykemeldt eller arbeidsavklaringspenger……………………………………
Midlertidig eller varig uføretrygdet…………………………………………
4. INTERNETT OG MOBILBRUK

4.1 Har du internett hjemme?................................. Ja ☐ Nei ☐

4.2 Bruker du internett til vanlig?............................. Ja ☐ Nei ☐

4.3 Har du en mobiltelefon med internetttilgang?......... Ja ☐ Nei ☐

4.4 Søker du regelmessig helseinformasjon på nett?... Ja ☐ Nei ☐

4.5 Søker noen av dine nærmeste regelmessig
helseinformasjon på nett?......................................... Ja ☐ Nei ☐

5. MEDISINER

5.1 Kryss av dersom du bruker en eller flere av de følgende hjertemedisiner fast:
Sett ett eller flere kryss

Blodfortynnende (f.eks. Albyl E, Marevan, Plavix, Brilique, Efient, Xarelto, Eliquis, eller Pradaxa) .......................................................... ☐

Kolesterol softenende (f.eks. Simvastatin, Lipitor, Lescol, Pravachol, Zocor, Pravastatin Lovastatin, Atorvastatin, Crestor) .................................. ☐

Betablokker (f.eks. Selo-zok, Emconcor, Metoprolol, Carvedilol, Atenolol, Tenormin, Bisprolol, Unilock, Sotalol, Inderal eller Pranolol) ......................... ☐

ACE-hemmer/ARB (f.eks Triatec, Ramipril, Enalapril, Zestril, Renitec, Captopril, Zanicoress, Zestoretic, Losartan, Diovan, Valsartan, Aprovel, CoAprovel, Micardis, Atacand, Cozaar eller Exforge) .................. ☐

5.2 Vennligst oppgi navn og styrke på din(-e) kolesterol softenende medisin (-er):

Navn: .......................  Styrke: .................. mg.

Navn: .......................  Styrke: .................. mg.

Sett ett kryss her dersom du ikke tar noen kolesterol softenende medisiner: ☐

5.3 Har du opplevd bivirkninger når du tar dine hjertemedisiner?

Nei ☐  Kanskje ☐  Ja ☐  I så fall hvilke? .........................

5.4 Har du opplevd seksuelle problemer (impotens etc.) når du tar hjertemedisiner?

Nei ☐  Ja ☐  Jeg husker ikke ☐
5.5 Har du noen gang fått informasjon av lege om at seksuelle problemer kan forekomme ved behandling med hjertemedisiner?
Nei ☐    Jeg husker ikke ☐    Ja ☐    I så fall hvilke? ..........................

5.6 Hvor ofte tok du dine kolesterolønkende medisiner som forskrevet sist uke?
Sett ett kryss
☐ Hver dag    ☐ 6 av 7 dager    ☐ 5 av 7 dager    ☐ 4 av 7 dager    ☐ < 4 av 7 dager    ☐ Jeg tar ikke

5.7 Hvor ofte tok du dine blodfortynnende medisiner som forskrevet sist uke?
Sett ett kryss
☐ Hver dag    ☐ 6 av 7 dager    ☐ 5 av 7 dager    ☐ 4 av 7 dager    ☐ < 4 av 7 dager    ☐ Jeg tar ikke

5.8 Hvor ofte tok du dine medisiner som forskrevet av lege den siste måneden?
Sett ett kryss
Hele tiden (100 %)......................................................................................................................... ☐
Nesten hele tiden (ca. 90 %).......................................................................................................... ☐
Det meste av tiden (ca. 75 %)........................................................................................................ ☐
Omtrent halvparten av tiden (ca. 50 %).......................................................................................... ☐
Mindre enn 50 % av tiden.................................................................................................................. ☐

5.9 Hvor ofte glemte du å ta 1 eller fler av dine reseptbelagte medisiner den siste måneden?
Sett ett kryss
 semester
Aldri.................................................................................................................................................. ☐
En gang i løpet av siste måned......................................................................................................... ☐
2-3 ganger i løpet av siste måned...................................................................................................... ☐
En gang per uke........................................................................................................................................ ☐
Flere ganger per uke............................................................................................................................ ☐
Omtrent hver dag.................................................................................................................................. ☐

5.10 Hvor ofte bestemte du deg for å la være å ta 1 eller fler av dine reseptbelagte medisiner i løpet av den siste måneden?
Sett ett kryss
Aldri.................................................................................................................................................. ☐
En gang i løpet av siste måned......................................................................................................... ☐
2-3 ganger i løpet av siste måned.................................................................
En gang per uke..............................................................................................
Flere ganger per uke....................................................................................... 
Omtrent hver dag.............................................................................................

6 Ett kort spørreskjema om medisinbruk
Sett ett kryss

Hender det at du av og til glemmer å ta dine medisiner?........... Ja ☐ Nei ☐

Enkelte glemmer å ta sine medisiner av andre grunner enn forglemmelse. Hvis du tenker på de siste 2 ukene, var det noen dager du ikke tok dine medisiner?............................... Ja ☐ Nei ☐

Har du noen gang kuttet ned eller stoppet å ta dine medisiner uten å informere din lege fordi du følte deg verre når du tok de?... Ja ☐ Nei ☐

Når du er på reise eller drar hjemme fra, hender det at du av og til glemmer å ta med medisinene dine?....................................................... Ja ☐ Nei ☐

Tok du alle dine medisiner i går?................................................................. Ja ☐ Nei ☐

Når du føler at symptomene dine er under kontroll, hender det at du slutter å ta dine medisiner?................................................................. Ja ☐ Nei ☐

Det å ta medisiner hver dag oppleves som ubeleilig for enkelte. Har du noen ganger følt at det er vanskelig å ta dine medisiner som forskrevet av lege?................................................................. Ja ☐ Nei ☐

Hvor ofte har du problemer med å huske å ta alle dine medisiner?

Aldri/sjelden.................................................................................................
En gang i blant............................................................................................... 
Noen ganger.................................................................................................
Vanligvis........................................................................................................
Hele tiden......................................................................................................

7. FYSISK AKTIVITET

7.1 Hvor ofte driver du med fysisk aktivitet?
(Ta et gjennomsnitt)

Aldri...............................................................................................................
Sjeldnere enn 1 gang i uka
En gang i uka
2-3 ganger i uka
Omtrent hver dag

7.2 Dersom du driver fysisk aktivitet så ofte som en eller flere ganger i uka:
Hvor hardt tar du i?
(Ta et gjennomsnitt)
Tar det rolig uten å bli andpusten eller svett
Tar det så hardt at jeg blir andpusten og svett
Tar meg nesten helt ut

7.3 Hvor lenge holder du på hver gang?
(Ta et gjennomsnitt)
Mindre enn 15 minutter
16-30 minutter
30 minutter – 1 time
Mer enn 1 time

8. RØYKING
Sett ett eller flere kryss

8.1 Røyker du selv?
Sigarett daglig
Sigarer/sigarillos daglig
Pipe daglig

8.2 Jeg har aldri røkt daglig (sett kryss)

8.3 Har du noen gang røkt daglig?

8.4 Hvor mange sigaretter røyker eller røykte du vanligvis daglig?
(angi antall sigaretter)

8.5 Hvor mange år har du til sammen røkt daglig? (angi antall år)

8.6 Bruker du daglig snus?

8.7 Omtrent hvor mange bokser snus bruker du per uke?
### 9. Kosthold

#### 9.1 Hvor mange ganger per uke inntar du følgende matvarer?

Sett ett kryss på hver rad

<table>
<thead>
<tr>
<th>Matvarer</th>
<th>Mer enn 2 ganger/dag</th>
<th>Ca. 1 gang/dag</th>
<th>4-6 ganger/uke</th>
<th>Mindre enn 3 ganger/uke</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fisk som pålegg/middag</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grønnsaker</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frukt/bær</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pølser/hamburger og tilsvarende de…</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brus/saft med sukker</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pasta/ris</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poteter</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sjokolade/smågodt</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### 10. HØYDE/VEKT

10.1 Hvor mye veier du uten klær?………………………………………………………………………………..  □ □ kg.

10.2 Hvor høy er du? ……………………………………………………………………………………………..  □ □ cm.

10.3 Hva stemmer om din vekt?

Sett ett kryss

Ikke overvektig □  Litt overvektig □  Svært overvektig □

De neste spørsmålene omfatter hjertesykdommen din. Med hjertesykdom mener vi at du enten har hatt et hjerteinfarkt, hatt behov for ny utblokking av hjertets kransårer eller har blitt hjerteoperert.

### 11. DIN OPPFATNING AV HJERTESYKDOMMEN OG RISIKOFAKTORER

#### 11.1 Hvilke av de følgende utsagn føler du stemmer for deg?

Sett ett kryss

Jeg er nå frisk igjen av hjertesykdommen, men må endre min livsstil (dvs. omlegging av kosthold, økt fysisk aktivitet og/eller røykestopp) ………………………………………… □

Jeg er nå frisk igjen av hjertesykdommen og behøver ikke å endre livsstil… □

Jeg har fortsatt hjertesykdom og må endre min livsstil…………………………………… □

Jeg har fortsatt hjertesykdom, men behøver ikke endre min livsstil………………… □
Jeg fikk ingen informasjon om sykdommen/jeg husker ikke

11.2 Hva tror du er sannsynligheten for at du får et nytt anfall med hjertesykdom (dvs. enten et nytt hjerteinfarkt, behov for ny utblokking eller ny hjerteoperasjon) i løpet av de neste 12 månedene?  
Sett ring rundt ett tall  
Helt usannsynlig 0 1 2 3 4 5 6 7 8 9 10 Helt sikkert

11.3 Hvor mye føler du at du kan selv kan gjøre for å redusere din risiko for å få et nytt et nytt hjerteinfarkt, behov for ny utblokking eller ny hjerteoperasjon?  
Sett ring rundt ett tall  
Ingen ting 0 1 2 3 4 5 6 7 8 9 10 Svært mye

11.4 I hvilken grad tror du din hjertesykdom vil begrense dine daglige aktiviteter i fremtiden?  
Sett ring rundt ett tall  
Ingen ting 0 1 2 3 4 5 6 7 8 9 10 Svært mye

12. LIVSSTILSENDRINGER  
Spørsmål 12.1-12.4 besvares kun av røykere eller tidligere røykere

12.1 Dine røykevaner etter at du fikk påvist hjertesykdommen  
Jeg har sluttet å røyke…………………………………………………… Ja  Nei
Jeg røyker mindre nå enn før jeg fikk påvist hjertesykdommen……  Ja  Nei
Jeg røyker mer nå enn før jeg fikk påvist hjertesykdommen………  Ja  Nei
Jeg har forsøkt å redusere eller slutte å røyke…………………………  Ja  Nei

12.2 Har du blitt tilbudt hjelp til å slutte å røyke av helsevesenet (f.eks. fastlege)?  
Ja  Nei  Jeg husker ikke  

12.3 Har du blitt tilbudt nikotinerstatning som f.eks. røykeplaster, tyggegummi?  
Ja  Nei  Jeg husker ikke  

12.4 Har du lyst til å slutte å røyke eller redusere røykingen?  
Sett ring rundt ett tall  
Har ikke lyst 0 1 2 3 4 5 6 7 8 9 10 Har veldig lyst
12.5 Hvor mye har du økt ditt fysiske aktivitetsnivå etter at du fikk påvist hjertesykdommen?
Sett ring rundt ett tall

Ingen ting  0  1  2  3  4  5  6  7  8  9  10  Svært mye

12.6 Har du lyst til å øke ditt fysiske aktivitetsnivå ytterligere?
Sett ring rundt ett tall

Har ikke lyst  0  1  2  3  4  5  6  7  8  9  10  Har veldig lyst

12.7 Hvor mye mer sunt (hjertevennlig) spiser du etter at du fikk påvist hjertesykdommen?
Sett ring rundt ett tall

Ingen ting  0  1  2  3  4  5  6  7  8  9  10  Svært mye

12.8 Har du lyst til å gjøre kostholdet enda sunnere/hjertevennlig?
Sett ring rundt ett tall

Har ikke lyst  0  1  2  3  4  5  6  7  8  9  10  Har veldig lyst

13. INFORMASJON OM HJERTESYKDOMMEN OG RISIKOFAKTORER

13.1 Var det noen til stede på sykehuset den dagen du fikk informasjon om hjertesykdommen?
Sett ett eller flere kryss

Ektefelle/samboer eller nære familie…………………………………………………………………………………
Venner eller bekjente……………………………………………………………………………………………………
Jeg var alene………………………………………………………………………………………………………………
Jeg husker ikke fordi det er lenge siden jeg fikk diagnose……………………………………………………

13.2 Fikk du råd og veiledning om hvordan du skal forebygge nye hjertehendelser (dvs. ett nytt hjerteinfarkt, behov for ny utblokking eller ny hjerteoperasjon) før du ble skrevet ut fra sykehuset?
Sett ett kryss

Ja  [ ]  Nei  [ ]  Husker ikke/vet ikke  [ ]
13.3 Har du fått informasjon fra sykehuset om at dine barn eller andre i din nære familie bør undersøkes for hjertesykdom?

Ja ☐ Nei ☐ Husker ikke/vet ikke ☐

13.4 Hvor har du fått informasjon om hjertesykdommen og/eller risikofaktorer etter at du ble utskrevet fra sykehus?
Sett ett eller flere kryss

Gjennom familie og venner………………………………………………………………………….. ☐

Internett…………………………………………………………………………………………………….. ☐

Fra brosjyrer, aviser, bøker eller blader………………………………………………………………… ☐

Fra fastlegen ………………………………………………………………………………………………….. ☐

Fra interesseorganisasjoner som f.eks. Landsforeningen for hjertesyke (LHL)….. ☐

Informasjon fra apoteket……………………………………………………………………………………… ☐

Fra annet helsepersonell (fysioterapeut, sykepleier el.lign.)……………………………………… ☐

Fra Hjerterehabiliteringen på sykehuset……………………………………………………………………. ☐

Angi dersom du har fått informasjon fra ett annet sted …………………………………………..

Jeg har ikke fått informasjon om hjertesykdommen og/eller risikofaktorer etter at jeg ble utskrevet fra sykehus………………………………………………………………………………… ☐

13.5 I hvilken grad opplever du at du har fått tilstrekkelig informasjon om hjertesykdommen og hvordan du kan forebygge nye tilfeller (dvs. ett nytt hjerteinfarkt, behov for ny utblokking eller ny hjerteoperasjon)?

Ingen grad 0 1 2 3 4 5 6 7 8 9 10 I stor grad

13.6 Skulle du ønske du hadde fått mer informasjon om hjertesykdommen?
Sett ett kryss

Ja, absolutt ☐ Ja ☐ Kanskje ☐ Nei ☐

13.7 I hvilken grad opplever du at du har fått tilstrekkelig informasjon om hjertevennlig kosthold, fysisk aktivitet, blodtrykk, kolesterol og tobakk?
Sett ring rundt ett tall

Ingen grad 0 1 2 3 4 5 6 7 8 9 10 I stor grad
13.8 Skulle du ønske du hadde fått mer informasjon om risikofaktorer for hjertesykdom?
Ja, absolutt ☐  Ja ☐  Kanskje ☐  Nei ☐

14. OPPFØLGING FRA HELSEVESENET ETTER AT DU SIST VAR INNLAGT PÅ SYKEHUS MED HJERTESYKDOMMEN

14.1 Hvilken oppfølging ble du anbefalt og/eller tilbudt de første ukene/månedene etter at du var innlagt på sykehus med hjertereinfarkt, utblokking eller hjerteoperasjon?
Sett ett eller flere kryss

Jeg har aldri blitt anbefalt/tilbudt noen spesiell oppfølging……………………………………………………… ☐
Jeg ble tilbudt hjerteskole i regi av sykehuset med bl.a. sykepleier…………. ☐
Jeg ble anbefalt/tilbudt videre oppfølging hos min fastlege……………………………. ☐
Jeg ble anbefalt/tilbudt fysisk aktivitet i regi av fysioterapeut på sykehuset. ☐
Jeg ble anbefalt/tilbudt fysisk aktivitet i regi av fysioterapeut i kommunen…. ☐
Jeg ble anbefalt/tilbudt hjerterehabilitering på et rehabiliteringssenter…….. ☐

14.2 Fulgte du opp den denne anbefalingen  Ja ☐  Nei ☐  Jeg husker ikke ☐

14.3 Dersom du svarte nei på spørsmål 14.2, hva var de viktigste årsakene til det?
Sett ett eller flere kryss

Jeg visste ikke om disse tilbudene………………………………………………………………………………………. ☐
Tilbud (-ene) lå for langt unna der jeg bor……………………………………………………………………………………… ☐
Tidspunktene passet ikke……………………………………………………………………………………………………… ☐
Jeg hadde ikke tid……………………………………………………………………………………………………………………… ☐
Jeg så ikke behovet for disse tilbudene……………………………………………………………………………………… ☐
Jeg deltok på en annen oppfølging av hjertesykdommen enn de nevnt i 14.1.. ☐
Andre årsaker…………………………………. ☐

14.4 Ble noen av dine nærmeste pårørende tilbudt å delta på deler av sykehusets hjerteskole?
Ja ☐  Nei ☐  Jeg husker ikke ☐
15. OPPFØLGING FRA HELSEVESENET I DAG
Sett ett eller flere kryss

15.1 Hvordan følges din hjertesykdom opp av helsevesenet i dag?

Jeg har ingen spesiell oppfølging i dag…………………………………………………………… ☐

Jeg følges opp hos min fastlege 3 ganger i året eller oftere…………………………………… ☐

Jeg følges opp hos min fastlege 1-2 ganger i året .......................................................... ☐

Jeg følges opp av min fastlege mindre enn 1 gang i året ................................. ☐

Jeg deltar på jevnlig fysisk aktivitet hos fysioterapeut eller i annen kommunal regi………………………………………………………………………………… ☐

Jeg deltar på jevnlig oppfølging hos ernæringsfysiolog eller tilsvarende…….. ☐

Jeg deltar på jevnlig oppfølging hos psykolog/psykiater for lære å mestre hjertesykdommen og/eller få hjelp til å endre livsstil ............................ ☐

Jeg deltar jevnlig på et privat tilbud (som betales helt av egne midler) der fysisk aktivitet og/eller hjelp til livsstilsendringer er viktig................................. ☐

15.2 Dersom du har annen oppfølging av hjertesykdommen fra helsevesenet enn nevnt ovenfor, vennligst oppgi denne her

………………………………

15.3 Skulle du ønske du hadde hatt mer oppfølging av din hjertesykdom enn du får i dag?
Ja, absolutt ☐ Ja ☐ Kanskje ☐ Nei ☐

16. DINE TANKER OM HJERTESYKDOMMEN

16.1 I hvor stor grad tror du noe av det følgende har bidratt til at du har utviklet hjerteinfarkt, behov for utblokking eller hjerteoperasjon?
Sett ring rundt ett tall

Går i familien/arv

<table>
<thead>
<tr>
<th>Ingen grad</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>I svært stor grad</th>
</tr>
</thead>
</table>

Kostholdet mitt

<table>
<thead>
<tr>
<th>Ingen grad</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>I svært stor grad</th>
</tr>
</thead>
</table>
For lite fysisk aktivitet
Ingen grad 0 1 2 3 4 5 6 7 8 9 10 I svært stor grad

Røyking
Ingen grad 0 1 2 3 4 5 6 7 8 9 10 I svært stor grad

Overvekt
Ingen grad 0 1 2 3 4 5 6 7 8 9 10 I svært stor grad

Stress i hverdagen
Ingen grad 0 1 2 3 4 5 6 7 8 9 10 I svært stor grad

Tretthet eller dårlig søvn
Ingen grad 0 1 2 3 4 5 6 7 8 9 10 I svært stor grad

Depresjon/tristhet
Ingen grad 0 1 2 3 4 5 6 7 8 9 10 I svært stor grad

Høyt blodtrykk
Ingen grad 0 1 2 3 4 5 6 7 8 9 10 I svært stor grad

Sukkersyke (diabetes)
Ingen grad 0 1 2 3 4 5 6 7 8 9 10 I svært stor grad

Høyt nivå av kolesterol i blodet (lipider)?
Ingen grad 0 1 2 3 4 5 6 7 8 9 10 I svært stor grad

Alderen?
Ingen grad 0 1 2 3 4 5 6 7 8 9 10 I svært stor grad
**Tilfeldigheter/uflaks**

![Gradskala med gradene fra 0 til 10]

**Annen årsak**

16.2 Siden du fikk en påvist hjertesykdom har du kanske gjort enkelte endringer av din livsstil. Kan du i dag tenke deg å gjøre ytterligere endringer av din livsstil?

Ja [ ] Nei [ ] Kanskje hvis jeg fikk hjelp og støtte fra helsepersonell [ ]

16.3 Hvilke av de følgende tiltakene kunne du tenke deg å gjøre dersom du fikk hjelp og støtte fra f.eks. en lege eller sykepleier?

Sett ett eller flere kryss

- Få et mer «hjertevennlig» kosthold (mindre mettet fett, sukker, salt)?
- Øke mitt fysiske aktivitetsnivå?
- Slutte å røyke?
- Ta hjertemedisinene mine som forskrevet?
- Leve mindre hektisk (mindre stressende)?
- Gå ned i vekt?
- Jeg ønsker ikke å gjøre noen tiltak?
- Jeg har allerede gjort de tiltakene jeg mener er nødvendige?

17. DINE BEHOV FOR OPPFØLGING AV DIN HJERTESYKDOM

17.1 Hvilke tilbud kan helsevesenet bidra med for at du skal få best mulig oppfølging av din hjertesykdom?

Skriv ned inntil 4 tilbud i prioritert rekkefølge

1. ..........................................
2. ..........................................
3. ..........................................
4. ..........................................
17.2 Hvilke av tilbudene nedenfor kunne du tenke deg dersom de var tilgjengelig i dag?
Sett ett kryss

A. Oppfølging via telefon, e-post, SMS
Mulighet for og bli kontaktet eller selv kontakte helsepersonell (f.eks. en erfaren hjertesykepleier) på telefon, e-post eller SMS når jeg trenger det.
Ja, absolutt ☐ Ja ☐ Kanskje ☐ Nei ☐

Dersom du svarer ja, hvor mange ganger per år kan du tenke deg dette til budet? .............

B. Oppfølging av en hjertesykepleier
Mulighet for oppfølging av en hjerte-sykepleier på poliklinikken på sykehuset. Et slikt tilbud vil omfatte både en samtale, en klinisk undersøkelse og justering av mine hjertemedisiner ved behov
Ja, absolutt ☐ Ja ☐ Kanskje ☐ Nei ☐

Dersom du svarer ja, hvor mange ganger per år kan du tenke deg dette til budet? .............

Mulighet for besøk av en erfaren hjertesykepleier i mitt eget hjem. Et slikt tilbud vil omfatte både samtale, en klinisk undersøkelse og justering av mine hjertemedisiner ved behov
Ja, absolutt ☐ Ja ☐ Kanskje ☐ Nei ☐

Dersom du svarer ja, hvor mange ganger per år kan du tenke deg dette til budet? .............

C. Gruppesamling med medpasienter
Mulighet for samlinger på sykehuset der jeg kan møte medpasienter og diskutere felles utfordringer ved hjertesykdommen. Ulike tema som motivasjon, psykiske utfordringer, kosthold, trening, medisiner osv. kan diskuteres. Timene ledes av en hjertesykepleier.
Ja, absolutt ☐ Ja ☐ Kanskje ☐ Nei ☐

Dersom du svarer ja, hvor mange ganger per år kan du tenke deg dette tilbudet? .............

D. Oppfølging av fysioterapeut
Mulighet for oppfølging av en fysioterapeut for veiledning og hjelp til å øke mitt fysiske aktivitetsnivå.
Ja, absolutt ☐ Ja ☐ Kanskje ☐ Nei ☐
Dersom du svarer ja, hvor mange ganger per år kan du tenke deg dette tilbuedet? ............

E. Oppfølgning av ernæringsfysiolog

Mulighet for oppfølgning av en ernæringsfysiolog for veiledning og råd for å bedre mitt kunnskapsnivå om et sunt og «hjertevennlig» kosthold.

Ja, absolutt [ ]   Ja [ ]   Kanskje [ ]   Nei [ ]

Dersom du svarer ja, hvor mange ganger per år kan du tenke deg dette tilbuedet? .............

F. Oppfølgning av psykolog eller psykiater

Mulighet for oppfølgning av en psykolog eller psykiater for å lære strategier som kan hjelpe meg å oppnå ønskede livsstilsendringer, samt hjelpe meg å mestre psykiske utfordringer relatert til sykdommen/stressmestring.

Ja, absolutt [ ]   Ja [ ]   Kanskje [ ]   Nei [ ]

Dersom du svarer ja, hvor mange ganger per år kan du tenke deg dette tilbuedet? .............

G. Tverrfaglig oppfølging

Mulighet for tverrfaglig oppfølging av både hjertesykepleier, psykolog/psykiater, fysioterapeut, ernæringsfysiolog og lege. Innholdet i tilbuedet vil være en blanding av det som er beskrevet ovenfor.

Ja, absolutt [ ]   Ja [ ]   Kanskje [ ]   Nei [ ]

Dersom du svarer ja, hvor mange ganger per år kan du tenke deg dette tilbuedet? .............

H. Oppfølging via internett

Jeg kunne tenke meg å få tilgang til en passord-beskyttet internetside med følgende innhold:

1. Kvalitetssikret kunnskap og gode råd for meg og mine pårørende om livsstilsendringer og oppfølgingen av sykdommen.
2. Mulighet for registrering av oppdatert medisinliste og helseatferd (kosthold, fysisk aktivitet etc.).
3. Ett forum der man kommuniserer med helsepersonell og eventuelt medpasienter dersom hvis ønskelig.

Ja, absolutt [ ]   Ja [ ]   Kanskje [ ]   Nei [ ]
I. Oppfølgning ved bruk av en mobil applikasjon (APP)

Jeg kunne tenke meg at helsevesenet utviklet en mobil applikasjon (APP) med bl.a. følgende innhold:
1. Hjelp til å holde oversikt over mine medisiner og minne meg på og ta de til rett tid.
2. Hjelp til og enten endre kosthold, økt mitt fysiske aktivitetsnivå, gå ned i vekt eller slutte å røyke (dersom dette er ett behov).
3. Eventuelt mulighet for å kommunisere med min behandler eller medpasienter.

Ja, absolutt ☐  Ja ☐  Kanskje ☐  Nei ☐

Jeg ser ikke behovet for noe ytterligere oppfølgningstilbud fra helsevesenet for min hjertesykdom

Enig ☐  Uenig ☐

Jeg kunne tenke meg et tilbud fra helsevesenet over internett

Ja, absolutt ☐  Ja ☐  Kanskje ☐  Nei ☐

17.3 Hvilke av tilbudene angitt i spørsmål 17.2 kunne du tenke deg å delta på i dag på dersom de var tilgjengelige?
Skriv ned 4 tilbud i prioritert rekkefølge
1. ………………………………
2. ………………………………
3. ………………………………
4. ………………………………

18 ALKOHOLBRUK
18.1 Omtrent hvor ofte har du i løpet av de siste 12 måneder drukket alkohol? *(Regn ikke med lettøl)*
Sett ett kryss

4-7 ganger pr. uke ☐  Ca. 1 gang pr. måned ☐

2-3 ganger pr. uke ☐  Noen få ganger pr. år ☐

Ca. 1 gang pr. uke ☐  Ingen ganger siste år ☐

2-3 ganger pr. måned ☐  Aldri drukket alkohol ☐
18.2 Har du drukket alkohol i løpet av de siste 4 ukker?  
Ja ☐  Nei ☐

_Hvis ja:_  
Har du drukket så mye at du har kjent deg sterkt beruset (full)?

Nei ☐  Ja, 1-2 ganger ☐  Ja, 3 ganger eller mer ☐

18.3 Hvor mange glass øl, vin eller brennevin drikker du vanligvis i løpet av 2 uker?  
_(Regn ikke med lettøl)_
_Sett 0 hvis du ikke drikker alkohol_

<table>
<thead>
<tr>
<th>Øl</th>
<th>Vin</th>
<th>Brennevin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Antall glass

18.4 Hvor ofte drikker du 5 glass eller mer av øl, vin eller brennevin ved samme anledning?  
_Sett ett kryss_

Aldri............................... ☐  Ukentlig ......................... ☐
Månedlig ............................ ☐  Daglig............................... ☐

19.1 Har noen av dine foreldre eller søsken fått påvist koronar hjertesykdom (hjerteinfarkt, behov for utblokking eller hjerteoperasjon) før 65 (kvinner) og 55 (menn) års alder?  
Ja ☐  Nei ☐  Vet ikke/husker ikke ☐

19.2 Har du fått informasjon fra sykehuset om at dine barn eller andre i din nære familie bør undersøkes for hvorvidt de også er disponert for hjertesykdom?  
Ja ☐  Nei ☐  Vet ikke/husker ikke ☐

20. LIVSKVALITET, HUMØR, MESTRING OG SØVN

Nedenfor kommer en del spørsmål om livskvalitet, humør, psykiske helseplager og søvn. Dette er svært relevante problemstillinger for mange hjertepasienter. Enkelte av spørsmålene kan ligne på hverandre, men dette er meningen.

1. Stort sett, vil du si at din helse er  
_Sett ett kryss_

Utmerket ☐  Meget god ☐  God ☐  Nokså god ☐  Dårlig ☐
De neste spørsmålene handler om aktiviteter som du kanskje utfører i løpet av en vanlig uke. 

**Er din helse slik at den begrenser deg i utførelsen av disse aktivitetene nå? Hvis ja, hvor mye?**

2. Moderate aktiviteter som å flytte ett bord, støvsuge, gå en tur eller drive med hagearbeid
   - Ja, begrenser meg mye
   - Ja, begrenser meg litt
   - Nei, begrenser meg ikke i det hele tatt

3. Gå opp trappen flere etasjer
   - Ja, begrenser meg mye
   - Ja, begrenser meg litt
   - Nei, begrenser meg ikke i det hele tatt

I løpet av den siste uken, har du hatt noen av de følgende problemer i ditt arbeid eller i andre av dine daglige gjøremål på grunn av din fysiske helse?

4. Du har utrettet mindre enn du hadde ønsket
   - Ja
   - Nei

5. Du har vært hindret i å utføre visse typer arbeid eller gjøremål
   - Ja
   - Nei

I løpet av de siste 4 ukene, har du hatt noen av de følgende problemer i ditt arbeid eller i andre av dine daglige gjøremål på grunn av følelsesmessige problemer (som for eksempel å være deprimert eller engstelig)?

6. Du har utrettet mindre enn du hadde ønsket
   - Ja
   - Nei

7. Du har utført arbeidet eller andre gjøremål mindre grundig enn vanlig
   - Ja
   - Nei

8. I løpet av de siste 4 ukene, hvor mye har smerter påvirket ditt vanlige arbeid (gjelder både arbeid utenfor hjemmet og husarbeid)?
   - Ikke i det hele tatt
   - Litt
   - En del
   - Mye
   - Svært mye

De neste spørsmålene handler om hvordan du har følt deg og hvordan du har hatt det de siste 4 ukene. For hvert spørsmål, vennligst velg det svaralternativet som best beskriver hvordan du har hatt det. Hvor ofte i løpet av siste 4 ukene har du:

9. Følt deg rolig og harmonisk?
   - Hele tiden
   - Nesten hele tiden
   - Mye av tiden
   - En del av tiden
   - Litt av tiden
   - Ikke i det hele tatt
10. Hatt mye overskudd?

Hele tiden ❑ Nesten hele tiden ❑ Mye av tiden ❑ En del av tiden ❑ Litt av tiden ❑ Ikke i det hele tatt ❑

11. Følt deg nedenfor og trist?

Hele tiden ❑ Nesten hele tiden ❑ Mye av tiden ❑ En del av tiden ❑ Litt av tiden ❑ Ikke i det hele tatt ❑

12. I løpet av de siste 4 ukene, hvor mye av tiden har din fysiske helse eller følelsesmessige problemer påvirket din sosiale omgang (som det å besøke venner, slektninger osv.)?

Hele tiden ❑ Nesten hele tiden ❑ En del av tiden ❑ Litt av tiden ❑ Ikke i det hele tatt ❑

Her er en liste med ting folk noen ganger gjør eller tenker når de føler seg nedtrykt, trist eller deprimert. Les hver av dem og kryss av for hvor ofte du gjør eller tenker det som beskrives når du føler deg slik. NB: det vi er interessert i er hva du faktisk gjør/tenker, ikke hva du synes du bør gjøre/tenke

<table>
<thead>
<tr>
<th>Nr.</th>
<th>Lemmel</th>
<th>Jenta</th>
<th>Parking</th>
<th>Ofte</th>
<th>Alltid</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Tenker på hvor ensom du føler deg</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
</tr>
<tr>
<td>2</td>
<td>Tenker ”Hvis jeg ikke klarer å komme meg ut av dette, får jeg ikke gjort jobben min”</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
</tr>
<tr>
<td>3</td>
<td>Tenker på dine følelser av utmattethet og smerte</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
</tr>
<tr>
<td>4</td>
<td>Tenker på hvor vanskelig det er å konsentrere seg.</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
</tr>
<tr>
<td>5</td>
<td>Tenker ”Hva er det jeg gjør for å fortjene dette”?</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
</tr>
<tr>
<td>6</td>
<td>Tenker på hvor passiv og umotivert du føler deg.</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
</tr>
<tr>
<td>7</td>
<td>Analyserer nylige hendelser for å prøve å forstå hvorfor du er deprimert.</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
</tr>
<tr>
<td>8</td>
<td>Tenker på hvorfor det virker som om du ikke føler noe lenger.</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
</tr>
<tr>
<td>9</td>
<td>Tenker ”Hvorfor kommer jeg meg ikke i gang?”.</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
</tr>
<tr>
<td>10</td>
<td>Tenker ”Hvorfor reagerer jeg alltid på denne måten?”.</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
<td>❑</td>
</tr>
</tbody>
</table>
11. Er for deg selv og tenker på hvorfor du føler som du gjør. 


14. Tenker "Hvis jeg fortsetter å føle meg på denne måten, kommer jeg ikke til å kunne konsentrere meg". 

15. Tenker "Hvorfor har jeg problemer som andre mennesker ikke har?". 

16. Tenker "Hvorfor takler jeg ikke ting bedre?". 

17. Tenker på hvor trist du føler deg. 

18. Tenker på alle dine mangler, svakheter og feil. 

19. Tenker på hvorfor du ikke føler deg i stand til å gjøre noen ting. 

20. Analyserer personligheten din for å prøve å forstå hvorfor du er deprimert. 

21. Drar et sted alene for å tenke over dine følelser. 

22. Tenker på hvor sint du er på deg selv. 


<table>
<thead>
<tr>
<th>Uɔrktsg</th>
<th>Ganske uriktig</th>
<th>Verken riktig eller uriktig</th>
<th>Ganske riktig</th>
<th>Riktig</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

1. Jeg oppnår lett kontakt når jeg møter mennesker.
2. Jeg lager ofte oppstyr rundt uviktige ting. ☐ ☐ ☐ ☐ ☐
3. Jeg snakker ofte med fremmede. ☐ ☐ ☐ ☐ ☐
4. Jeg føler meg ofte ulykkelig. ☐ ☐ ☐ ☐ ☐
5. Jeg er ofte irritert. ☐ ☐ ☐ ☐ ☐
6. Jeg føler meg ofte hemmet i sosialt samvær. ☐ ☐ ☐ ☐ ☐
7. Jeg har et negativt/pessimistisk syn på ting. ☐ ☐ ☐ ☐ ☐
8. Jeg finner det vanskelig å starte en samtale. ☐ ☐ ☐ ☐ ☐
9. Jeg er ofte i dårlig humør. ☐ ☐ ☐ ☐ ☐
10. Jeg er en lukket person. ☐ ☐ ☐ ☐ ☐
11. Jeg foretrekker å holde andre mennesker på avstand. ☐ ☐ ☐ ☐ ☐
13. Jeg er ofte "nede i grøfta". ☐ ☐ ☐ ☐ ☐
14. Når jeg snakker med andre, finner jeg ikke de rette tingene å snakke om. ☐ ☐ ☐ ☐ ☐

Her kommer noen spørsmål om hvorledes du føler deg. For hvert spørsmål setter de kryss for ett av de fire svarene som best beskriver dine følelser den siste uken. Ikke tenk for lenge på svaret – de spontane svarene er best.

<table>
<thead>
<tr>
<th>1. Jeg er nervøs eller anspent</th>
<th>8. Jeg føler meg som om alt går langsommere</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ For det meste</td>
<td>☐ Nesten hele tiden</td>
</tr>
<tr>
<td>☐ Ofte</td>
<td>☐ Svært ofte</td>
</tr>
<tr>
<td>☐ Noen ganger</td>
<td>☐ Fra tid til annen</td>
</tr>
<tr>
<td>☐ Ikke i det hele tatt</td>
<td>☐ Ikke i det hele tatt</td>
</tr>
<tr>
<td>2. Jeg gleder meg fortsatt over ting jeg pleide å glede meg over</td>
<td>9. Jeg føler meg urolig liksom jeg har sommerfugler i magen</td>
</tr>
<tr>
<td>---------------------------------------------------------------</td>
<td>---------------------------------------------------------------</td>
</tr>
<tr>
<td>☐ Avgjort like mye</td>
<td>☐ Ikke i det hele tatt</td>
</tr>
<tr>
<td>☐ Ikke fullt så mye</td>
<td>☐ Fra tid til annen</td>
</tr>
<tr>
<td>☐ Bare lite grann</td>
<td>☐ Ganske ofte</td>
</tr>
<tr>
<td>☐ Ikke i det hele tatt</td>
<td>☐ Svært ofte</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Helt sikkert og svært ille</td>
<td>☐ Ja, helt klart</td>
</tr>
<tr>
<td>☐ Ja, men ikke så veldig ille</td>
<td>☐ Jeg bryr meg ikke så mye som jeg burde</td>
</tr>
<tr>
<td>☐ Litt ille, men det bekymrer meg ikke så mye</td>
<td>☐ Det kan nok hende jeg ikke bryr meg nok</td>
</tr>
<tr>
<td>☐ Ikke i det hele tatt</td>
<td>☐ Jeg bryr meg utseendet like mye som jeg alltid har gjort</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>4. Jeg kan le og se det morsomme i situasjoner</th>
<th>11. Jeg føler meg rastløs som om jeg stadig må være i aktivitet</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Like mye som jeg alltid har gjort</td>
<td>☐ Uten tvil svært mye</td>
</tr>
<tr>
<td>☐ Ikke like mye nå som før</td>
<td>☐ Ganske mye</td>
</tr>
<tr>
<td>☐ Avgjort ikke så mye nå som før</td>
<td>☐ Ikke så veldig mye</td>
</tr>
<tr>
<td>☐ Ikke i det hele tatt</td>
<td>☐ Ikke i det hele tatt</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>5. Jeg har hodet fullt av bekymringer</th>
<th>12. Jeg ser med glede frem til hendelser og ting</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Veldig ofte</td>
<td>☐ Like mye som jeg alltid har gjort</td>
</tr>
<tr>
<td>☐ Ganske ofte</td>
<td>☐ Heller mindre enn jeg pleier</td>
</tr>
<tr>
<td>☐ Av og til</td>
<td>☐ Avgjort mindre enn jeg pleier</td>
</tr>
<tr>
<td>☐ En gang i blant</td>
<td>☐ Nesten ikke i det hele tatt</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6. Jeg er i godt humør</th>
<th>13. Jeg kan plutselig få en følelse av panikk</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Aldri</td>
<td>☐ Uten tvil svært ofte</td>
</tr>
<tr>
<td>☐ Noen ganger</td>
<td>☐ Svært ofte</td>
</tr>
<tr>
<td>☐ Ganske ofte</td>
<td>☐ Ikke så veldig ofte</td>
</tr>
<tr>
<td>☐ For det meste</td>
<td>☐ Ikke i det hele tatt</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>7. Jeg kan sitte i fred og ro og kjenne meg avslappet</th>
<th>14. Jeg kan glede meg over en god bok eller et radio- eller et TV program</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Ja, helt klart</td>
<td>☐ Ofte</td>
</tr>
<tr>
<td>☐ Vanligvis</td>
<td>☐ Fra tid til annen</td>
</tr>
<tr>
<td>☐ Ikke så ofte</td>
<td>☐ Ikke så ofte</td>
</tr>
<tr>
<td>☐ Ikke i det hele tatt</td>
<td>☐ Svært sjelden</td>
</tr>
</tbody>
</table>
For hvert av utsagnene nedenfor krysser du av for det svaralternativet som beskriver deg best, eller som er mest typisk for deg.

<table>
<thead>
<tr>
<th>Tekst</th>
<th>Ikke beskrivende</th>
<th>Noe beskrivende</th>
<th>Veldig beskrivende</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Jeg blir ikke bekymret selv om jeg ikke har tid å gjøre alt.</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2. Jeg blir overveldet av mine bekymringer.</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>3. Jeg pleier ikke å bekymre meg.</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Jeg blir bekymret i mange situasjoner.</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5. Jeg vet jeg ikke burde bekymre meg, men jeg klarer ikke la være.</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>6. Jeg bekymrer meg mye når jeg blir stresset.</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7. Jeg bekymrer meg alltid for noe.</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>8. Jeg synes det er lett å se bort fra bekymringer.</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9. Straks jeg er ferdig med en oppgave begynner jeg å bekymre meg for alt annet jeg må gjøre.</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>10. Jeg bekymrer meg aldri for noe som helst.</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>11. Når det ikke er noe jeg kan gjøre med et problem, slutter jeg å bekymre meg.</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>12. Jeg har vært en som bekymrer seg hele mitt liv.</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>13. Jeg har merket meg at jeg har bekymringer.</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>14. Har jeg først begynt å bekymre meg, kan jeg ikke slutte.</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>15. Jeg bekymrer meg hele tiden.</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>16. Jeg bekymrer meg for oppgaver inntil de alle er gjennomførte.</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

23
**Instruksjon.** På spørreskjemaet under er det 6 spørsmål knyttet til søvn og tretthet. Vær vennlig og sett ring rundt det alternativet (antall dager pr uke) som passer best for deg. 0 er ingen dager i løpet av en uke, 7 er alle dager i løpet av en uke.

**Eksempel**
Hvis du 3 dager i løpet av en uke har brukt mer enn 30 minutter på å sovne etter at du har slukket lyset, setter du ring rundt alternativ 3.

I løpet av den siste måneden, hvor mange dager pr. uke har du brukt mer enn 30 minutter for å sovne inn etter at lysene ble slukket?

<table>
<thead>
<tr>
<th>Antall dager pr. uke</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

1. I løpet av den siste måneden, hvor mange dager pr. uke har du brukt mer enn 30 minutter for å sovne inn etter at lysene ble slukket?

<table>
<thead>
<tr>
<th>Antall dager pr. uke</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

2. I løpet av den siste måneden, hvor mange dager pr. uke har du vært våken mer enn 30 minutter innimellom søvnen?

<table>
<thead>
<tr>
<th>Antall dager pr. uke</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

3. I løpet av den siste måneden, hvor mange dager pr. uke har du våknet mer enn 30 minutter tidligere enn du har ønsket uten å få sove igjen?

<table>
<thead>
<tr>
<th>Antall dager pr. uke</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

4. I løpet av den siste måneden hvor mange dager pr. uke har du følt deg for lite uthvilt etter å ha sovet?

<table>
<thead>
<tr>
<th>Antall dager pr. uke</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

5. I løpet av den siste måneden, hvor mange dager pr. uke har du vært så søvnig/trett at det har gått ut over skole/jobb eller privatlivet?

<table>
<thead>
<tr>
<th>Antall dager pr. uke</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

6. I løpet av den siste måneden, hvor mange dager pr. uke har du vært misfornøyd med søvnen din?

<table>
<thead>
<tr>
<th>Antall dager pr. uke</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
</tbody>
</table>

Har du brukt sovemedisiner siste uke? (sett kryss)  
Ja ___Nei____

Dersom du har svart JA:

Navn _____________________Styrke (mg)__________  Antall dager siste uke:_______
Nedenfor følger noen spørsmål om dine søvnvaner

1. Snorker du?
   - a. Ja
   - b. Nei
   - c. Vet ikke.

   Hvis du snorker:
   2. Er din snorking:
      - a. Litt høyere enn lyden av pusten din
      - b. Like høy som tale/snakking
      - c. Høyere enn tale/snakking
      - d. Veldig høy – kan høres i tilstøtende rom

3. Hvor ofte snorker du
   - a. Nesten hver dag
   - b. 3-4 netter i uken
   - c. 1-2 netter i uken
   - d. 1-2 netter i måneden
   - e. Aldri eller nesten aldri

4. Har snorkingen din plaget andre?
   - a. Ja
   - b. Nei
   - c. Vet ikke

5. Har noen lagt merke til at du stopper å puste i søvne?
   - a. Nesten hver dag
   - b. 3-4 ganger i uken
   - c. 1-2 ganger i uken
   - d. 1-2 ganger i måneden
   - e. Aldri eller nesten aldri
6. Hvor ofte føler du deg trett eller utslitt etter at du har sovet?

☐ a. Nesten hver dag
☐ b. 3-4 ganger i uken
☐ c. 1-2 ganger i uken
☐ d. 1-2 ganger i måneden
☐ e. Aldri eller nesten aldri

7. I løpet av tiden du er våken, føler du deg trett, utslitt eller ikke helt på topp

☐ a. Nesten hver dag
☐ b. 3-4 dager i uken
☐ c. 1-2 dager i uken
☐ d. 1-2 dager i måneden
☐ e. Aldri eller nesten aldri

8. Har du noen gang duppet av eller sovnet mens du har vært fører av et kjøretøy?

☐ a. Ja
☐ b. Nei

Hvis ja:

9. Hvor ofte skjer dette?

☐ a. Nesten hver dag
☐ b. 3-4 ganger i uken
☐ c. 1-2 ganger i uken
☐ d. 1-2 ganger i måneden
☐ e. Aldri eller nesten aldri

Her følger noen spørsmål om hvordan hjertesykdommen påvirker deg. Vennligst sett en ring rundt det tallet som best samsvarer med din mening.

1. Hvor mye påvirker sykdommen livet ditt?

Ingen påvirkning 0 1 2 3 4 5 6 7 8 9 10 Voldsom påvirkning
2. Hvor lenge tror du at sykdommen din vil vare?

Svært kort tid 0 1 2 3 4 5 6 7 8 9 10 For alltid

3. Hvor mye kontroll føler du at du har over sykdommen din?

Absolutt ingen kontroll 0 1 2 3 4 5 6 7 8 9 10 Svært stor kontroll

4. Hvor mye mener du at behandlingen din kan hjelpe mot sykdommen din?

Ikke i det hele tatt 0 1 2 3 4 5 6 7 8 9 10 Svært hjelpsom

5. Hvor mye opplever du symptomer fra sykdommen din?

Ingen symptomer i det hele tatt 0 1 2 3 4 5 6 7 8 9 10 Mange alvorlige symptomer

6. Hvor bekymret er du angående sykdommen din?

Ikke bekymret i det hele tatt 0 1 2 3 4 5 6 7 8 9 10 Svært bekymret

7. Hvor godt føler du at du forstår sykdommen din?

Forstår ikke i det hele tatt 0 1 2 3 4 5 6 7 8 9 10 Forstår svært godt

8. Hvor mye påvirker sykdommen din deg følelsesmessig? (dvs. gjør den deg sint, redd, urolig eller deprimert?)

Ikke påvirket følelsesmessig i det hele tatt 0 1 2 3 4 5 6 7 8 9 10 Svært følelsesmessig påvirket

9. Vennligst skriv ned i rekkefølge de tre viktigste faktorene som du tror forårsaket sykdommen din.

De aller viktigste årsaker for meg:"

1. ____________________________________________________________________________

2. ____________________________________________________________________________

3. ____________________________________________________________________________

21. HVILKE RÅD OG TIPS KUNNE DU TENKE DEG Å GI OSS SOM JOBBER I HELSEVESENET MED HJERTEPASIENTER?

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Vi håper du nå kan legge alle arkene i den vedlagte konvolutten og sende de til oss så snart som mulig. Tusen takk for at du tok deg tid til å svare på disse spørsmålene!