Ethnic inequalities in cardiovascular health

Kjersti Stormark Rabanal

Master in Public Health Science, 2011

30 Credits

Norwegian University of Life Sciences,

Department of Mathematical Sciences and Technology

Summary

This master thesis in Public Health Science concentrates on the topic *ethnic inequalities in cardiovascular health*, and consists of two parts. The first part contains a comprehensive presentation of relevant theory and a wide discussion of the topic. The second part is the article; *Ethnic differences in risk factors and total risk of cardiovascular disease in Norway: a cross sectional study*. The article gives detailed information about methods and results. The aim of this study is to examine the relationship between cardiovascular health and ethnic origin. The focus is based on previous studies demonstrating ethnic differences in risk of cardiovascular diseases.

The first part, which is the main thesis, links the topic to the field of public health by presenting detailed theory about relevant aspects. Such aspects include cardiovascular diseases and its risk factors, equality in health and the concept of ethnicity. Brief summaries of methods and results of the article are given in the corresponding sections of the main thesis. Some methodological considerations are further discussed before the results from the article are discussed in a wider theoretical context, emphasizing the relevance for public health.

The article describes a cross-sectional study based on data from a main sample of 62 145 individuals in the age range 40-65 years, who participated in the Cohort of Norway. The aim was to examine ethnic differences in risk factors and total risk of cardiovascular diseases, the latter calculated based on two different risk equations. Ethnic belonging was indicated by place of birth, and participants from 11 different geographical regions were included. Self-reported variables, blood samples and physical measurements were used to estimate age-adjusted mean levels of CVD risk factors and calculate total risk scores.

Significant differences were found in risk factors and total risk scores. In particular, participants from the Indian subcontinent and Former Yugoslavia showed higher levels of risk factors and total risk compared to the other ethnic groups. This was evident in both sexes. The results are discussed in the article.

The discussion in the main thesis presents possible explanations for ethnic differences in cardiovascular health. It is concluded that although ethnic differences in genetic susceptibility exist, it is likely that underlying causes can be found in the surrounding contexts of individuals. More research is needed, in order to identify and address root causes for ethnic inequalities in cardiovascular health.

Sammendrag

Denne masteroppgaven i Folkehelsevitenskap fokuserer på temaet *etniske ulikheter i kardiovaskulær helse*, og består av to deler. Den første delen er en kappe som inneholder en omfattende presentasjon av relevant teori og en bred diskusjon av emnet. Den andre delen er artikkelen; *Ethnic differences in risk factors and total risk of cardiovascular disease in Norway: a cross sectional study*. Artikkelen gir detaljert informasjon om metode og resultater. Studiens mål er å undersøke forholdet mellom kardiovaskulær helse og etnisk opprinnelse. Fokuset er basert på at tidligere studier har vist at det finnes etniske forskjeller i risiko for hjerte-karsykdommer.

I kappen knyttes temaet til folkehelsefeltet gjennom relevant teori om blant annet kardiovaskulære sykdommer, risikofaktorer, likhet i helse og etnisitetsbegrepet. Korte sammendrag av metode og resultater fra artikkelen gis i de tilsvarende inndelinger i kappen. Diskusjon rundt noen metodiske problemstillinger blir presentert, før resultatene fra artikkelen og teori blir diskutert i en større sammenheng som vektlegger temaets relevans for folkehelse.

Artikkelen beskriver en tverrsnittstudie som er basert på et utvalg bestående av 62 145 personer i alderen 40-65 år, som deltok i the Cohort of Norway. Målet var å undersøke etniske forskjeller i risikofaktorer og total risiko for hjerte-karsykdommer. Total risiko ble regnet ut ved hjelp av to forskjellige risiko-score systemer. Deltakernes fødested ble brukt som indikator på etnisk tilhørighet, og personer fra 11 ulike føde-regioner ble inkludert. Selvrapporterte variabler, blodprøver og fysiske målinger ble brukt for å estimere aldersjusterte gjennomsnittsverdier av risikofaktorer for hjerte-karsykdom, og kalkulere total risiko-scorer.

Signifikante forskjeller ble funnet både i risikofaktorer og i total risiko. Deltakerne fra det Indiske subkontinent og tidligere Jugoslavia skilte seg spesielt ut, med relativt høye nivå av både risikofaktorer og total risiko. Dette gjaldt både menn og kvinner. Resultatene diskuteres i artikkelen.

Diskusjonen i kappen presenterer mulige årsaksforklaringer for etniske forskjeller i kardiovaskulær helse. Det konkluderes med at selv om det er forskjeller i genetisk sårbarhet blant ulike etniske grupper, er det sannsynlig at underliggende årsaker har sitt utspring i omkringliggende faktorer. Det kreves mer forskning for å kunne identifisere og rette tiltak mot slike primære årsaker for etniske ulikheter i kardiovaskulær helse.

Acknowledgments

Contents

List of figures and tables

Main thesis:

Article:

Abbreviations

BMI   Body mass index

CHD   Coronary heart disease

CONOR   Cohort of Norway

CVD   Cardiovascular disease

HDL   High density lipoprotein

IHD   Ischaemic heart disease

LDL   Low density lipoprotein

MI   Myocardial infarction

mmHg   millimeter of Mercury

mmol/L   millimole per Liter

SES   Socioeconomic status

VLDL   Very low density lipoprotein

WHO   World Health Organization

WHR   Waist to hip ratio

1.0 Introduction

1.1 Clearing central concepts

Health is multidimensional and can be defined in several ways. The World Health Organization (WHO) constitution of 1948 defined health as "a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity" (WHO 1948).

Based on this holistic definition of health, public health work is comprehended as the integrative and organized efforts of a society to strengthen factors that promote and protect health, prevent diseases, and prolong people's lives (NOU 1998:18; WHO 1998). This comprehensive understanding emphasizes that public health is not the responsibility of the health sector alone, but should include all sectors involved in a society. It is further underlined that public health interventions generally aim at populations rather than the individual (NOU 1998:18). Prevention efforts aimed at hindering a first occurrence of disease is called primary prevention (WHO 1998).

Just like the health concept, ethnicity is a multidimensional concept, often defined differently by different actors. The concept derives from the Greek word "ethnos" which means nation. It contains social, cultural and genetic dimensions based on shared belonging, experiences and characteristics. Examples of such shared relations might be geographical or social origin, collective way of living, communication and beliefs. Consequently, the boundaries of ethnic belonging are often fluently and constantly changing (Anand 1999; Barth 1969; Bhopal 2007).

1.2 Cardiovascular diseases: a public health perspective

Cardiovascular diseases (CVDs) form a group of diseases highly relevant on the global public health agenda; CVD is the leading cause of death and a major cause of disability worldwide (WHO 2005). CVDs were previously known as "Western diseases" only affecting industrialized countries (WHO 2003) since these countries were first afflicted when industrialization gained speed in the 19th and 20th centuries. Changes in living conditions resulted in a transition of the most important causes of death and morbidity (Detels & Breslow 2002; Yusuf, S. et al. 2001). This shift is called *the epidemiologic transition,* first termed by Omran in 1971 *(Omran 2005),*

implying that nutritional deficiencies and infectious diseases became less common and were replaced by chronic diseases such as cancer, diabetes and CVD (Yassi et al. 2001; Yusuf, S. et al. 2001). The epidemiologic transition implied a corresponding transition in risk factors associated with diseases (WHO 2009). However, it may be worth to note that the epidemiologic transition is not something that belongs to the past. Countries throughout the world are constantly in different stages of the transition. In the first stage, representing the earliest stage of development, the predominating CVDs are related to infections and nutritional deficiencies. Examples of regions in the first stage are Sub-Saharan Africa and rural areas in South Asia (Yusuf, S. et al. 2001). In the second stage, the burden of infectious diseases is reduced and nutrition improves. CVDs related to hypertension such as stroke caused by bleeding in the brain increases. In the following third stage, life expectancy is continuously improving and the societies become more affluent. Diets high in fat, cigarette smoking and physical inactivity become more common. CVD consequently become predominating and add to the burden of communicable diseases. Urban areas in India are examples found in this stage. Norway, as a Western European country is in the fourth stage of the transition where efforts aimed to prevent, diagnose and treat CVDs have been able to delay the development of these diseases (Yusuf, S. et al. 2001). The number of deaths caused by CVD in Norway has decreased during the last decades, but CVD is still accounted as the leading cause of death on a national basis (Statistics Norway 2010c). A fifth stage of the epidemiologic transition has also been proposed for countries where war or social upheavals destroy the existing social and health structures of the society leading to reoccurrence of communicable diseases (Yusuf, S. et al. 2001).

CVDs are no longer only "Western diseases" although many may still have this notion. The trend has changed implying that the poorer and less developed countries are disproportionally affected carrying the greatest burden of CVD mortality; 82 per cent of the CVD deaths occur in low-and middle income countries (WHO 2011a). An additional concern is the fact that people in developing countries often die younger than in developed countries (WHO 2003). These countries are still struggling with communicable and deficiency-related diseases while being more exposed to CVD risk factors than developed countries (WHO 2011a). This is referred to as a "double burden" meaning that poorer countries are greatly exposed to both traditional risk factors and the risk factors associated with chronic diseases and their consequences (Mackay et al. 2004).

CVD may lead to disability and long lasting complications. In order to measure the burden of CVD not only through mortality numbers, it is possible to include the burden of disease using the concept of DALYs (Disability Adjusted Life Years). DALYs represent a statistical concept developed to accommodate the measuring of additional health aspects and to compare them across populations. DALYs combine the number of potential years lost due to premature death and years of productive life lost due to disability. This means that one DALY represents one lost year of healthy life (WHO 2003). It is estimated that 18 % of DALYS in high income countries are due to CVD, while the equivalent number is 10 % for low and middle income countries (Mackay et al. 2004). This reflects that people live longer with these diseases in higher income countries where the access to treatment and prevention efforts are improved.

1.3   Equality and equity in health

The definition of health stated by the WHO, in the preamble to its constitution, was further underlined as a basic human right. It stated that every human being has the right to enjoy "the highest attainable standard of health". The highest attainable standard sound as a diffuse measure of health, but can more specifically be understood as reflected by the standard of health enjoyed by the most socially advantaged groups in a society (Braveman & Gruskin 2003).

Equity in health has become an increasingly important focus in the field of public health and health promotion, both nationally and internationally (Elstad 2005; Klepp 2010; Marmot et al. 2008). In Norway this is now evident in the political processes, referring to a new public health law which, among other things, aims at reducing social inequalities in health (Prop. 90 L (2010-2011)). It is noted that the concept of equity is different from the concept of equality. Equity is both an ethical and value-laden word which includes aspects of justice and fairness in the sense that it addresses inequalities considered both unnecessary and avoidable (Braveman & Gruskin 2003). Efforts striving to achieve equity in health are moreover, based on an aspiration to fulfill the public health potentials (Braveman et al. 2011). Achieving equity in health demands identification and examination of health inequalities before interventions addressing root causes can be added (Whitehead et al. 2001).

A social model of health is based on a holistic understanding of the health concept, and points out how different determinants in the surrounding environment can impact on the health of an individual. This model of health is opposed to the medical model which solely emphasizes the presence or absence of disease determining a person's health (CDHN s.a.; Naidoo & Wills

2000). The rainbow showing modifiable layers of influence on health (Figure 1) explain how health is a result of complex interaction between personal, social, economic and environmental factors.
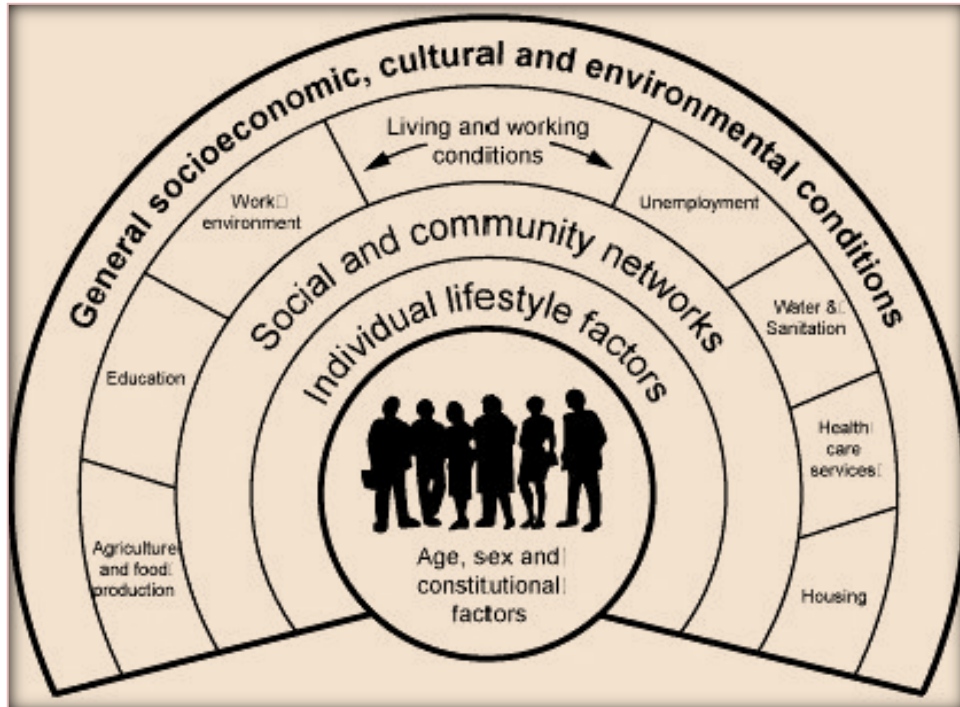
Figure 1: Dahlgren & Whiteheads social determinants of health rainbow (Whitehead et al. 2001).

Despite the increasing focus on equity; systematically inequitable distribution of social determinants can be found in many societies today (Braveman & Gruskin 2003; Marmot 2007).Differences do not only exist between countries (as explained in section 1.2.), but also within countries. Well documented is the so-called "social gradient" identified in several countries including Norway (Braveman et al. 2011; Claussen & Naess 2002; Phillimore et al. 1994; Strand & Tverdal 2004). This gradient implies that low socioeconomic status (SES), indicated by occupation, education and income, functions as a strong risk factor for mortality and morbidity in general, as well as for CVD mortality and morbidity in particular (Elstad 2005). With higher SES follows correspondingly a better health status and lower overall mortality found in each step of increased SES. This gradient may also play a role in health disparities according to ethnic belonging; identified as another important structure of the society impacting on general and cardiovascular health (Bhopal 2007). The relationship between ethnicity and health is still difficult to understand and it has been stressed that extensively more research is required in order to intervene on such health inequalities (Liburd & Jack 2005).

1.4   Ethnicity and CVDs

As is being explained more thoroughly in the article, several studies have previously shown that ethnicity affects the risk of developing CVD (Anand et al. 2000; Chaturvedi 2003; Grundy et al. 1999). Ethnicity may appear as an independent determinant or risk factor for disease without regard of the main risk factors (Grundy et al. 1999). It is disputed, however, whether ethnicity is a risk factor *per se* or if ethnic belonging put people at risk through other factors (Kain & Catto 2002; Nazroo 1998). What direction the risk is affected also varies based on the different ethnic groups and the type of disease in question (Chaturvedi 2003; Khan et al. 2004). Bhopal (2007, p.63) has stated that variation in disease and risk factors based on ethnicity may be even greater than variations based on more common epidemiological variables such as gender and socioeconomic status. It is therefore important not to ignore such differences, but to include them alongside with other epidemiological variables striving towards a better understanding of the underlying mechanisms for inequality in health.

Ethnicity versus race

When addressing differences in health, ethnicity is often the preferred exposure variable before race. Ethnicity is a complex concept including more aspects than that of race, although the concepts are overlapping and often used synonymously. Race is, more often than ethnicity, used to divide people according to their physical features and consequently ignores differences in environmental, social and economic circumstances. The concept of race is also encumbered with a history that associates it with misuse, in terms of ethnocentricity and racism (Bhopal 2007). Though the concept of ethnicity can be comprehended as both subjective and social, it is also related to birthplace and national origin which is one of the common ways of dividing people into ethnic groups (Bhopal 2007).

1.5   Immigrants in Norway

More than eleven per cent of the Norwegian population consists of people who have either immigrated to the country themselves (first-generation immigrants) or who have been born in Norway by two parents who immigrated (second-generation immigrants). There are immigrants in all counties, though the largest share lives in Oslo. The immigrants in Norway originate from 215 different countries; most of them from Poland, Sweden, Germany and Iraq. Among the largest groups are also Pakistan and Somalia if including second generation immigrants

5

(Statistics Norway 2010c). About 24 % of all immigrants who arrived in Norway during 1990-2008 came as refugees, another 24 % came as labour immigrants and 11 % to acquire an education. Additionally, 23 % came to Norway to reunite with their families and 17 % got residence permit because they had established a family (Statistics Norway 2010c). During the last twenty years, the share of immigrants (both first- and second generation immigrants) migrating from Asia, Africa, Latin-America and Turkey has increased (Statistics Norway 2010b). The total number of immigrants is suggested to increase further. Statistics Norway did a projection which predicts an increase from 460 000 first-generation immigrants in 2010 to 1-1,8 million first-generation immigrants in the year of 2060. Additionally, second generation immigrants will increase, implying that 22-28 per cent of the Norwegian population will be of a non-Norwegian ethnic origin in only fifty years to come (Statistics Norway 2010a). During 2010, the number of immigrants have already changed from 460 000 to 600 900 (Statistics Norway 2011). These numbers illustrate the importance of focusing on public health challenges relevant for a multiethnic society.

1.6 Cardiovascular diseases

CVD belongs to the group of non-communicable diseases and consists of different diseases and illnesses with different outcomes, all related to the heart and blood vessels. These diseases are (Mackay et al. 2004; WHO 2011a):

- Coronary heart disease (CHD) which is also termed ischemic heart disease (IHD) (Burns & Kumar 2003). This is a disease of the vessels supplying the heart, of which heart attack is a potential outcome.

- Cerebrovascular disease which is a disease of the vessels supplying the brain. Stroke is a potential outcome.

- Peripheral arterial disease which is a disease of the arteries supplying the legs and arms.

- Rheumatic heart disease involves damage to the heart muscle and valves following a streptococcal bacterial infection causing rheumatic fever.

- Congenital heart disease involves malformations of the heart structures already existing at birth.

- Deep vein thrombosis with possibly following pulmonary embolism (blood clots in the leg veins which can possibly dislodge and end up in the heart and lungs).

Cerebrovascular disease and ischaemic heart disease are the two diseases mainly responsible for CVD being the leading cause of death (WHO 2009).

1.6.1   *Pathogenesis and risk factors*

CVD is a multifactorial disease where different factors interact with each other in a multiplicative way creating atherosclerosis, which is the main underlying pathophysiologic mechanism (Kannel et al. 1976; Schoen & Cotran 2003). Congenital heart disease and rheumatic heart disease are of course exceptions with other specific causes, as described above. Atherosclerosis develops over years and is caused by deposits of plaque in the inner artery walls consisting of lipid, cholesterol, calcium and some other components. This plaque results in a hardening and thickening of the artery wall which can lead to a narrowing of the lumen that reduces the blood flow, or rupture of the plaque causing thrombosis (blood clot). This can eventually result in a blockage of the blood supply to the heart or brain, resulting in either a heart attack (myocardial infarction (MI)) or stroke (cerebrovascular infarction) depending on which of the two organs is affected (Remaley et al. 2005; Schoen & Cotran 2003). Stroke might also occur if a blood vessel ruptures creating bleeding into the brain (Mackay et al. 2004) . The complex medical mechanisms will not be discussed in more detail since that is too extensive for the intention of this paper.

Many risk factors for CVD have been identified, in fact more than 300 different risk factors are said to be associated with CVD. It is also stated that about eighty per cent of the coronary heart and cerebrovascular disease cases is mainly a result of behavioral risk factors (WHO 2011a). The most important behavioural risk factors are unhealthy diet, physical inactivity and smoking. The effects of behavioural risk factors can further be seen in individuals as increased blood pressure, increased blood sugar levels, increased levels of lipids in the blood, overweight and obesity. These effects are called intermediate risk factors because they are results of primary risk factors while still functioning as independent risk factors for CVD outcomes (WHO 2011a). The intermediate risk factors have a more direct effect on the atherosclerotic process (WHO 2009). The relationship between primary and intermediate risk factors is illustrated in Figure 2. Other risk factors not preventable are increasing age, male gender, family history and genetic disposition (Schoen & Cotran 2003).
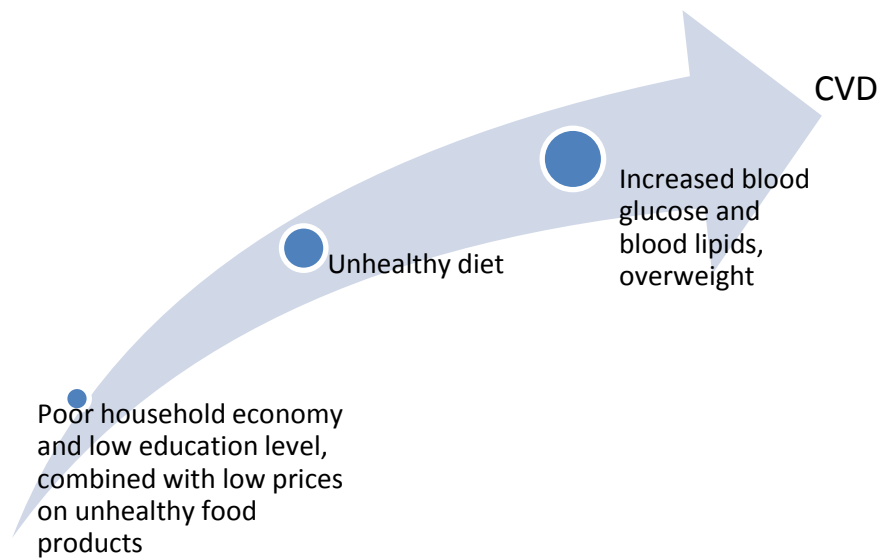
Figure 2: A simplified illustration of the causal chain in developing CVD
(Kjersti Stormark Rabanal)

Based on the fact that current knowledge on most important risk factors is derived from developed countries, Yusuf et.al. (2004) carried out a standardised case-control study of acute myocardial infarction (MI) in 52 countries represented by all inhabited continents of the world. This study concluded that nine risk factors accounted for most of the risk of MI in both sexes, in all ages and in all regions. The risk factors (abnormal lipids, smoking, hypertension, diabetes, abdominal fat, diet patterns, activity-level, alcohol and psychosocial factors) can consequently be held as risk factors in different ethnic groups (Yusuf et al. 2004).

1.6.2 Short description of some risk factors

Smoking

Smoking is a well-documented risk factor for CVD and no safe level has been identified (Schoen & Cotran 2003; Yusuf et al. 2004). Cessation is therefore recommended for all smokers. Smoking harms the cardiovascular system in several ways. In example, smoking leads to raised blood pressure, reduces the oxygen supply to the tissue and reduces the tolerance of physical exercise as well as contributing to atherosclerosis by increasing the clotting of the blood (AHA 2011).

Blood pressure

High blood pressure (hypertension) changes the structure of the arteries (WHO 2009) and is an important risk factor for developing several cardiovascular diseases such as cerebrovascular disease, ischaemic heart disease, and heart failure. Diet, especially one with high contents of salt and saturated fat, alcohol, lack of exercise, overweight and stress are factors that raise the blood pressure (Whitworth 2003; WHO 2009). However, more than 90 % of the cases of hypertension do not have a clear cause (Oparil et al. 2003). Definitions of hypertension differ according to where and when (daytime/nighttime) it is measured. Blood pressure measured at the doctor's office is expected to be slightly raised due to a so-called "white coat effect". Office hypertension is therefore defined as ≥140/90 mmHg as opposed to ≥135/80 if it had been measured at home (Mancia et al. 2007).

Diabetes and blood glucose

Diabetes mellitus is a chronic disorder of the metabolism of carbohydrate, fat and proteins. It is characterized by a relative or absolute deficiency in secretion or action of insulin; an anabolic hormone essential for controlling the glucose (sugar) levels in the blood by permitting cells to use glucose for energy (Clare-Salzler et al. 2003). Consequently without insulin, the blood glucose persists in the blood, leading to raised levels, also referred to as hyperglycemia (Freeman 2005). Diabetes is classified into two major variants; type 1 and 2 (although other variants exist). Type 1 is characterized by autoimmune destruction of the insulin-producing cells in pancreas. Type 2 generally has its outset in higher age, and is characterized by insulin resistance and defective insulin secretion. The latter constitutes the majority of the cases (Freeman 2005). Although the two types are different both in pathogenesis and metabolic characteristics, they both have increasing effect on the risk of CVD. Genetic factors play a central role in the development of both types (Clare-Salzler et al. 2003). In those genetically susceptible, changes in diet, increasing age, obesity and reduction in activity levels increases insulin resistance and thereby the risk of diabetes type 2 (Freeman 2005; WHO 2009). It is noted that hyperglycemia is an independent risk factor for CVD whether the individual has diabetes or not (WHO 2009), the mechanism for how it induces atherosclerosis are complex (Aronson & Rayfield 2002).

Blood lipids and lipoproteins

The lipids (fats) in the blood are transported as lipoproteins, attached to specific apolipoproteins (Schoen & Cotran 2003). Both lipids and lipoproteins are related to CVD with important effects on the atherosclerotic process (Schoen & Cotran 2003). High levels of lipids, such as

triglycerides and cholesterol, have been held as independent risk factors for CVD (Castelli 1996). However, the independend role of triglycerides is somewhat debated; it is unclear whether the relation with CVD is primarily direct or indirect (Cullen 2000). Moreover, triglycerides have traditionally been measured fasting, but there are increasing indications about imposed risk from non-fasting triglyceride levels as well (Bansal et al. 2007). Special emphasis, regarding high levels of lipids, has been put on cholesterol which is a well-documented independent risk factor for CVD (Bansal et al. 2007; Schoen & Cotran 2003). Yet, focusing on lipids is not enough when assessing risk, since the lipoproteins play an important role. Important lipoproteins include; high density lipoprotein (HDL), very low density lipoprotein (VLDL) (rich in triglycerides) and low density lipoprotein (LDL) (rich in cholesterol and a result of metabolised VLDL) (Despopoulos & Silbernagl 2003). HDL is often considered "the good" cholesterol and LDL the "bad" based on their effect on atherosclerosis, although this is an oversimplified statement since lipoproteins are transporters of lipids and consist of more than cholesterol (Biggerstaff & Wooten 2004). HDL cholesterol cleans up excess cholesterol and transports it from the tissue to the liver (Remaley et al. 2005). The ratio between total cholesterol and HDL cholesterol has been found to be a good predictor of the risk (Castelli 1996). Lipids are both made in the body and obtained from food (Mackay et al. 2004). Examples of factors that increases the levels of lipids in the blood are diets high in saturated fat and cholesterol, lack of physical activity as well as genetics (WHO 2009).

Overweight/obesity

Overweight and obesity is defined as "abnormal or excessive fat accumulation that may impair health" (WHO 2011b). The causes of obesity are not completely understood, and genetic factors as well as environmental and psychological factors play a role. Obesity leads to hypertension, diabetes, hypertriglyceridemia and it decreases HDL (Schoen & Cotran 2003). The body mass index focuses on the relation between weight and height, and is used to define overweight and obesity. Calculation of BMI is described in the article. A BMI of ≥25 is defined as overweight and ≥30 as obesity. The definition of overweight has been questioned in relation to Asian populations. This was addressed by an expert consultation of the WHO, who found that the observed health risk varied from a BMI of 22-25 in different Asian populations. The consultation consequently concluded that the international cut-off should be maintained for all populations (WHO 2004). Other than BMI, the waist to hip ratio (WHR) can help accommodate identification of excessive abdominal fat distribution, which is, in particular, a strong risk factor for CVD (WHO 2000). Which measure of overweight is best in relation to risk of CVD, has been

disputed, but Dalton et al. (2003) found that the WHR was somewhat preferable to BMI and waist circumference alone, although all measures showed predictive values in relation to type 2 diabetes, hypertension and dyslipidemia (Dalton et al. 2003).

1.7 Total risk versus high levels of single risk factors

Based on the interaction between risk factors, total risk scores have been derived to account for the total risk, not able to assess through focus on single risk factors (Kannel et al. 1976). The interaction between the risk factors means that when several risk factors appear together they increase the effect of each other (Norheim et al. 2009).

1.7.1 Systems established for assessing total risk

The Framingham Heart Study is a cohort-study which started in 1945 (Framingham Heart Study 2011b), and has contributed with important knowledge about risk factors for CVD. The study has its' name after the American city where participants were recruited. Based on that specific population, the findings of the study are mostly representative for white, American populations. Yet, many findings are appraised as fundamental and internationally valid (Framingham Heart Study 2011b). For example, the Framingham Heart Study is the one that identified the main risk factors for CVD (Framingham Heart Study 2011a) and several risk score equations have been established based on the Framingham Heart Study. Such equations calculate a person's risk of CVD events, based on an individuals' values of different risk factors (Anderson et al. 1991a; Anderson et al. 1991b; D'Agostino et al. 1994; D'Agostino et al. 2008; Kannel et al. 1976; Kannel et al. 1999; Wilson et al. 1998). The Framingham equation chosen for this study calculates 10-year risk of general CVD (including both fatal and non-fatal events) based on the risk factors age, gender, total cholesterol, HDL cholesterol, treated and untreated systolic blood pressure, smoking and diabetes (D'Agostino et al. 2008).

Framingham risk equations were previously used in European context, but in 2003 the European Society of Cardiology (ESC) made a new recommendation (De Backer et al. 2003). This new recommendation implied shifting to another system called SCORE (the European Systematic Coronary Risk Evaluation), because Framingham equations had a tendency to overestimate the risk of CVD in populations with generally lower risk, especially in the more southern parts of Europe (Conroy et al. 2003). The SCORE-project started in 1994 and resulted in a total risk

score system considered more valid for European populations (De Backer et al. 2003; Sleight 2002).

However, a general problem using SCORE is that it is based on the cardiovascular mortality 15-20 years ago. The model has therefore been found to overestimate the risk of CVD mortality in Norway (Lindman et al. 2006; Lindman et al. 2007). Based on this fact, Selmer and co-workers developed a new model for estimating risk, called NORRISK (also referred to as SCORE Norway). This model is calibrated according to the level of mortality in Norway during the time period of 1999-2003. It is based on numbers on mortality from Statistics Norway, mortality follow-up from Norwegian cardiovascular surveys and levels of risk factors from regional health examinations. NORRISK estimates 10-year risk of death from CVD based on the risk factors age, gender, systolic blood pressure, total cholesterol in serum, and smoking. This model is more applicable for the Norwegian population than the original SCORE model (Selmer et al. 2008). Because NORRISK focuses on mortality instead of morbidity, age becomes a more significant risk factor in NORRISK than it does in Framingham (Norheim et al. 2009).

The Norwegian Directorate of Health recommends application of NORRISK to asymptomatic persons who present with high levels of risk factors, have family members with early established CVD or present other reasons for calculating their total risk score. It is further noted that other risk factors which are not included in the equation must be considered additionally when assessing risk (Norheim et al. 2009).

1.8 Aim of the study

The aim of this study is to examine the relationship between cardiovascular health and ethnic origin. This will be done by investigating differences in cardiovascular risk factors and total risk scores among people originating from different geographical regions, living in Norway.

2.0 Material and methods

The material and methods are described in the article (starting page 3), and only the most essential parts will therefore be summarized here. Additional considerations not described in the article will also be accounted for. The relevant part of the questionnaire used in CONOR can be found in appendix I.

2.1 Summary of method

This study used data from the Cohort of Norway (CONOR) which is a large database constituted of ten regional health surveys. These surveys were not explicitly mentioned in the article, and will therefore be summed up here. CONOR includes the following (Næss et al. 2008);

- The Tromsø Health Study (Tromsø IV and V)
- The Troms and Finnmark Health Study (TROFINN)
- The Nord-Trøndelag Health Study (HUNT II and III)
- The Oslo Health Study (HUBRO)
- The Second Oslo Study (Oslo II)
- The Oslo Immigrant Health Study (I-HUBRO)
- The second part of the Romsås in Motion Study (MORO II)
- The Oppland and Hedmark Health Study (OPPHED)
- The Hordaland Health Study (HUSK)

We restricted the sample to the first time subjects participated in a CONOR-survey, and thereby excluded participants who were registered for the second or the third time (Næss et al. 2008). The CONOR-sample totally consists of 174 430 individuals.

The design of this study is cross-sectional aiming to examine ethnic differences in cardiovascular risk factors and total cardiovascular risk according to the NORRISK and Framingham equations described in the article and mentioned in section 1.7. Eleven different ethnic groups living in different parts of Norway were compared holding birth place as an indicator of ethnic belonging. This included Norwegian-born residents and people who had immigrated from the regions; Eastern Europe, Former Yugoslavia, North Africa, Sub-Saharan Africa, the Middle East, the Indian subcontinent, East Asia, North America, South America and South-East Asia.

The sample was restricted according to exclusion criteria related to selected regions and plausible values of risk factors included in the total risk score systems. This resulted in a main sample of 62 145 (53 % women) participants in the age range 40-65 years old. Further exclusions were subsequently performed according to exclusion criteria for the two risk score equations, which resulted in two smaller samples; one for each equation.

Differences between the groups were examined stratified by sex and adjusted for age using one-way analysis of variance by applying a generalized linear model. Glucose and triglyceride estimates were additionally adjusted for time since last meal. The PASW Statistics version 18 was used for all analyses. Post-hoc tests were done for the risk scores when significant overall differences between the ethnic groups were found. Such post-hoc tests involved comparing the immigrant-groups to Norway, which was held as reference. P-values less than 0,05 were considered statistically significant.

It is added that the definition of high total cholesterol/HDL ratio used in the article was based on studies referring to the predictive value of this ratio in relation to coronary heart disease (Kinosian et al. 1994) as well as the threshold found in the Norwegian electronic medical handbook (NEL- Nevrologiske Prosedyrer 2001).

2.2   Additional information about the sample

As mentioned in section 1.5, most of the immigrants in Norway live in Oslo. Correspondingly, immigrants in this study were best represented through the Oslo-surveys (containing 73 % of all immigrants in the sample). Naturally I-HUBRO was the one with most participants born outside of Norway since I-HUBRO only included people born in Turkey, Iran, Pakistan, Sri Lanka and Vietnam (NIPH 2005).

Although we did not know the single birth country that each participant originated from, it was possible to see who had participated in the Oslo Immigrant Health Study and get an idea of how some of the countries were represented. In our main sample of 62 145 participants, 385 (55 %) of the 669 people coming from the Middle East participated in the Oslo Immigrant Health Study and was therefore either Turkish or Iranian (the only two Middle East countries represented in I-HUBRO). Correspondingly, 507 people (47 %) of the 1071 from the Indian subcontinent in our

sample were immigrants from either Sri Lanka or Pakistan, and 259 (42 %) of the South East Asian group of 611 individuals were Vietnamese.

2.3   Additional considerations: choosing birth regions

The total population in CONOR originated from 16 different birth regions, and in addition to the regions that were included and mentioned above; Western Europe, The Pacific, Central America, Oceania/Pacific and Central Asia were among those.

Western Europe was left out because it was considered as similar to Norway and we wanted to limit the number of groups. The other four regions were excluded because they constituted small samples.

2.3.1 Checking the possibility of merging some birth regions

When choosing the birth regions, the possibility of merging some of them into larger regions was examined. The intention of merging was to preserve a large sample size (groups with few representatives could be included in the study if merged into larger regions) and give a better overview having fewer groups. Examining this possibility meant examining differences in risk factors between the two groups in question. If no significant difference in important risk factors were found, then merging would be conceivable. Otherwise, the groups with very few representatives would be excluded. Groups that were considered as possible to unify were those considered close in a geographical sense; Eastern Europe (n=405) with Former Yugoslavia (n=341), Central America (n=55) with South America (n=243), and East Asia (n=225) with South-East Asia (1272). When examining the possibilities, independent samples t-tests were performed to check for significant differences in the mean values of important risk factors. Regression analysis to adjust for age and gender were then performed to control for possible confounding.

Eastern Europe and former Yugoslavia

Independent sample t-tests showed that Eastern Europe was significantly different from former Yugoslavia in several of the risk factors. Differences were found in triglycerides (mean difference = -0,259 mmol/L, p=0,004), HDL (mean difference = 0,277, p<0,001), BMI (mean difference = -1,28, p<0,001), WHR (mean difference = -0,024, p<0,001) and age (mean difference = 7,05 p<0,001). The differences found in triglycerides, HDL and age were

considered most important since these were to be included in risk score calculations later. HDL has different recommendations based on gender (Mackay et al. 2004) and it was therefore decided to control for gender when evaluating the differences found in the t-test. Age was also included as a possible confounder. A linear regression analysis showed that these birth regions had a significant effect of 0,334 mmol/L on triglycerides (p<0,001) when controlling for age and gender. Combining these two regions was therefore out of the question since that would have increased the heterogeneity in such a hypothesized merged region. A regression analysis was not performed to control the difference in HDL and age since the difference in triglycerides was already found significant when controlling for age and gender.

Central America and South America

The significant differences in risk factors between Central America and South America revealed in an independent sample t-test, was a mean difference of 0,136 mmol/L in HDL (p=0,015) and a mean difference of 4,64 mmHg in systolic blood pressure (p=0,05). As mentioned above, HDL levels vary based on gender, and a following regression analysis showed that these birth regions had an effect of -0,106 mmol/L when controlling for age and gender which was statistically significant (p=0,046). Gender also had a significant (p < 0,001) effect of 0,250 mmol/L on HDL-levels in the same regression analysis. Different gender distribution in the two countries might therefore be responsible for some of the difference found as significant in the t-test, knowing that the effect of gender was larger and significant on a higher level than that of birth regions. Women constituted 66 % in the group originating from Central America and 55 % in the group from South America. It was decided to exclude Central America to avoid increased heterogeneity. In a possibly merged region, South America would in any case have been the predominating group with more than four times the number of representatives than of Central America.

East Asia and South-East Asia

T-tests checking for differences in mean values of risk factor between East Asia and South-East Asia showed statistically significant differences in triglyceride and HDL levels. The mean difference in triglycerides was -0,212 mmol/L (p=0,006) while the mean difference in HDL was 0,089 (p=0,001). A following regression analysis controlling the difference in triglycerides for age and gender, showed that these birth regions had a statistically significant effect of 0,032 (p=0,009). This finding excluded the possibility of merging the two regions.

3.0 Ethics

This study is in accordance with the World Medical Association Declaration of Helsinki; a set of ethical principles regarding medical research that involves human subjects (WMA 2008). All the participants in CONOR signed a written informed consent form (Næss et al. 2008). The data was made unrecognizable by the CONOR Steering Committee before access to the data was acquired, and the project protocol has been evaluated by legal employers at the Norwegian Institute of Public Health (NIPH). All the studies in CONOR have been approved by the Norwegian Data Inspectorate and evaluated by the Regional Committees for Medical Research Ethics (Søgaard 2007).

Focusing on ethnicity in research also requires some ethical considerations (Ingierd & Fossheim 2009). In this study, the concept of ethnicity has been chosen before the concept of race; in part because it is apprehended as less potentially offensive. As mentioned earlier, research on race and ethnicity has historically been unethically carried out. It is therefore required to show caution in the interpretation and presentation of findings - which has been attempted here (Ingierd & Fossheim 2009). It is further noticed that when dividing people into groups as was done here, there are possibilities of creating or supporting stigmatization, although that is not the intention. Research on ethnicity and health is important, and not taking ethnic differences into consideration would also be unethical (Bhopal 1997). The intention of this study is that ethnic groups might benefit in the future.

4.0 Results

The results were explained in the article and will therefore briefly be summarized here.

4.1 Summary of results

The analyses of variance showed significant differences in risk factors, and in total 10-year risk of general CVD based on the Framingham equation, between ethnic groups. This was observed in both men and women. Differences in total 10-year risk of CVD mortality using NORRISK was, however, only observed in men. Most of the groups showed high levels of inactivity, highest in immigrant groups from less developed regions.

Immigrants from countries of the Indian subcontinent showed the highest prevalence of diabetes, the highest levels of blood glucose, WHR, triglycerides and the lowest HDL cholesterol levels. This group also had the third highest score in total risk of general CVD.

Immigrants from the Former Yugoslavia showed high levels of several risk factors and had the highest total risk of general CVD according to the Framingham equation. This applied to both men and women. Regarding risk factors, Former Yugoslavians had the highest total cholesterol/HDL ratio and high levels of blood pressure, overweight indicators and smoking.

North Americans were the highest educated group in this study sample and showed relatively low levels of several risk factors. East Asia showed lower total risk score according to Framingham, and low levels of overweight measures and lipids.

5.0 Discussion

The results were discussed in the article and will therefore not be discussed in detail here. In addition to a discussion of methodological considerations, I will discuss the findings in a larger context underlining their relevance to the field of public health.

5.1   Methodological considerations

This is a cross-sectional study where all the information about the participants refers to one point in time (Rothman 2002). In this case; the first time the subjects participated in a CONOR survey. Associations may be assessed in cross-sectional studies, but no causal conclusions can be drawn (Rothman 2002).

5.1.1   Errors affecting reliability and validity

Two types of error are of general concern in epidemiologic studies; random error and systematic error (Rothman 2002). Random error represents stochastic variations in the data that cannot readily be explained by the researcher (Bjørndal & Hofoss 2008). Systematic error is also referred to as bias (Rothman 2002) and may be a problem if the research methods systematically afflict the results. These two types of error can influence the reliability and validity of the study. Reliability require accurate measurements, and is often defined as consistency of repeated measures (Aalen 1994; Winter 2000). The validity of a study demands reliable data, and further that what is actually measured corresponds with what was intended to be measured (Aalen 1994).

Uncertainty in the estimates caused by random error is, in this study, demonstrated by the application of confidence intervals and standard deviations in the tables of the article. CONOR contains a large number of participant data (Næss et al. 2008). This accommodates relatively large sample sizes which reduces the problem with systematic error. However, after exclusions, some of the groups ended up having relatively small sample sizes, which was further demonstrated by large confidence intervals. This was particularly evident in immigrant groups compared to Norway which had the largest sample size and greater certainty in its estimates.

Different types of systematic bias can be categorized into selection bias, information bias and confounding (Rothman 2002). The latter is of less importance here, since the multidimensional concept of ethnicity allows many aspects to explain causes for the differences found in the study.

Selection bias is a possible problem that affects the external validity concerning transferability (Bjørndal & Hofoss 2008). The participation rate was lowest in Oslo and the urban areas and participation rate among immigrants was low in the Oslo Health Study (Næss et al. 2008; Søgaard et al. 2004). However, the CONOR-variables of age, sex, birth country, education and smoking have been found to correspond with the Norwegian population (Aamodt et al. 2010). In relation to the different ethnic groups, a phenomenon referred to as "the healthy migrant effect" imply that the immigrants might represent particularly healthy parts of the population in their country of origin (Fennelly 2007).

An example of information bias includes possible under-reporting of self-reported variables, a possible cause for error of measurement in this study (Aamodt et al. 2010). A particular example is self-reported data on smoking, which may be encumbered with uncertainty as a consequence of underreporting (Patrick et al. 1994). Still, questionnaire information on chronic diseases have been found to be valid among patients with diabetes in Norway (Midthjell et al. 1992).

Also regarding information bias and error of measurement is that some measurements are in general not always reliable. For example, blood pressure may vary based on who is performing it, the device and when or where it is measured (Aalen 1994). This was, to some extent, accounted for in CONOR by measuring the blood pressure after each subject had rested two minutes and then using three different measurements to calculate a more reliable blood pressure mean. The reliability of the measurements in CONOR has been somewhat secured by letting trained and experienced personnel conduct the procedures following a standard procedure in all the surveys (Næss et al. 2008). Moreover, although blood samples were analyzed at different laboratories, calibration procedures between the laboratories have been performed and the consistency of the analyses have been evaluated and considered acceptable.

The application of large birth regions as indicators of ethnicity is an important question regarding the validity of this study. Do the birth regions actually reflect the participants' ethnic belonging? A problem is that the regions are in all likelihood heterogeneous ethnic groups that include several ethnic subgroups varying in risk factors (Bhopal et al. 1999; Bhopal 2007; Nazroo 1998). However, this way of defining ethnicity may be an advantage in the sense that it

is relatively common, and therefore accommodates the possibilities of comparing with other studies. There is no current consensus on the most appropriate way to use the concept of ethnicity when linking it to differences in health. Yet, the currently preferred way of defining ethnic belonging seems to be that of self-assessment, but self-assessment may change over time and be even more fluent than other definitions (Bhopal 2007; Nazroo 1998). The birth regions applied here are therefore considered as relatively valid, though not ideal, indicators of ethnic belonging.

5.1.2 The risk score equations; valid indicators of total risk for all ethnic groups?

NORRISK was derived using mean mortality data in Norway and is meant for Norwegian populations (Selmer et al. 2008). It has not been validated for other ethnic groups. It is, however, the one recommended for national use (Norheim et al. 2009) and thereby relevant for all ethnic groups living in Norway.

Some Framingham risk score versions (Anderson et al. 1991a; Wilson et al. 1998) are the ones that have been validated most for different ethnic groups compared to other available equations (Berger et al. 2010; Bhopal et al. 2005). Framingham derived equations have been validated in Caucasian Americans, African Americans, Europeans, and Mediterranean and Asian populations, but are less valid among some European and Asian populations. Some of the validations for different ethnic groups (living in the UK and the United States) have been done through recalibration using mean risk factor levels and ratio of survival estimates from minority cohort studies or prevalence data on different minority groups (Brindle et al. 2006; D'Agostino et al. 2001).

The updated Framingham CVD equation applied here has not been validated in the same extent as older versions, but has been validated in Iranian (Bozorgmanesh et al. 2011) and Australian populations (Zomer et al. 2011) and was also found to be moderately effective in a population of the United Kingdom (Simmons et al. 2009) - although it overestimated the true risk in all the populations. The intention of calculating total risk in this study was not to recommend treatment, but to investigate differences based on ethnicity. It is therefore considered an advantage to apply this equation in addition to NORRISK, since it is more valid for different ethnic groups.

5.2 Ethnic inequalities in cardiovascular health - in light of the rainbow

This study revealed ethnic inequalities in cardiovascular health indicated by important risk factors and calculated total risk scores. A fundamental question regarding social inequalities in health, is whether the causes lie in inherent or imposed characteristics, and if they are to be found in the individual or in social contexts (Krieger 2001). Are people from Former Yugoslavian countries genetically programmed to have poor cardiovascular health compared to other ethnic groups? Can the explanations be related to how the immigrants are welcomed and treated in the host country? Or perhaps psychological and behavioural effects of possible discrimination, combined with experiences before time of migration are more important? Moreover, it may be related to their cultural way of life, the type of food they eat and the way they prepare it. Possible answers are many, and uncovering all of them is not an object here. However, in the light of presented theory, I will suggest some circumstances that may contribute to unequal distribution of cardiovascular health determinants. This will be discussed in a wider sense than in the article, aiming to demonstrate the complexity of health inequalities and the need for a better understanding to finally address its root causes. The results from the cross-sectional study presented in the article will, however, form the basis for the discussion.

The presented rainbow of the main determinants of health (figure 1 in section 1.3), shows how health is determined by a complex chain of causes.

5.2.1 Age, sex & hereditary factors

The inner layer of the rainbow of determinants is often considered as constituted by unalterable and predetermined factors. Increasing age leads to increased risk of CVD, and is the way of nature. Additionally, men are at greater risk of developing CVD than women, although women's risk increases after menopause (Schoen & Cotran 2003). Whether individuals are born as a boy or a girl is not something they get to have a say in, and neither is the process of ageing. One simply has to accept genetics. Sex and age are both characteristics that unavoidably lead to inequalities in health, and following the definition of equity, these inequalities cannot be held as inequitable. They are unavoidable differences. The following question is then; are ethnic inequalities predetermined as functions of *genetic* characteristics and thereby also predetermined and unavoidable? Previous research focusing on racial (biologic) differences has tried to divide humans into sub-species, but have not succeeded doing so (Bhopal 1997). Research on ethnicity

has, as mentioned, historically been carried out with unethical intentions and researchers focused on racial differences in order to justify racial discrimination. Such researchers stated that biology determined social position, and thereby explained the differences through the viewpoint of biological determinism (Bhopal 1997). Although some genetic differences do exist, we are more alike than we are different, and it is not possible to say that different ethnic groups have completely different compositions of genetic characteristic (Bhopal 2007). A conclusion in the literature is also that modifiable factors are important for actual risk of CVD in all human beings despite possible differences in genetic susceptibility (Yusuf, Salim et al. 2001; Yusuf et al. 2004).

Although individuals cannot decide what genetics to be born with, genes are not unaffected by environmental conditions. This is reflected in theories of phenotype plasticity stated by evolutionary scientists (Via et al. 1995). "The thrifty phenotype hypothesis", first proposed by Neel in the 1960's (Neel 1962), is a relevant example in this context. The theory suggests that poor nutritional conditions experienced in early life, even as early as during gestation, may lead to a phenotypic adaption to poor nutritional conditions (Barker 2007; Bateson et al. 2004). Such adaption implies that the individual becomes better adapted to environmental conditions similar to what was experienced and "forecasted" in early life. The adaption consequently results in the individual being born with a smaller body and a modified metabolism that copes better when there is a shortage of food (Bateson et al. 2004).However, when the individual is brought up in affluent environments with excess of food, as is often the case in developed countries, this thrifty phenotype may lead to increased risk of coronary heart disease, type 2 diabetes and hypertension (Bateson et al. 2004). Many researchers have focused on such associations, and the Norwegian doctor Anders Forsdahl was one of the first to suggest that poor conditions in early life could lead to increased risk of CVD in later life. This was based on his findings of associations between infant mortality rates and later mortality of CVD among men in Finnmark (Forsdahl 1977; Vangen et al. 2005). Later researchers such as David J.P. Barker, although focusing on *fetal* conditions and the risk of CVD (Barker & Martyn 1992), found support for the findings of Forsdahl. The thrifty phenotype hypothesis, sometimes referred to as the "Barker hypothesis" has gained support in many different populations across the world (Hales & Barker 2001). It is one of the possible hypotheses that may explain some of the excess risk of diabetes and other metabolic disturbances for immigrants from the Indian subcontinent (Bavdekar et al. 1999; Yajnik 2001) found in this study.

Similar adaptive mechanisms have also been proposed for explaining some of the excess risk of hypertension in immigrants from the African continent due to a salt-sensitivity originally favourable for their forefathers living in hot and dry climates (Adair & Prentice 2004; Weder 2007). However, in this study, only the female part of the group from Sub-Saharan Africa seemed to be in comparative high risk of hypertension. The thrifty phenotype hypothesis does further not only function as a possible explanation of increased risk in individuals migrating from less developed to more developed countries. It may also explain some of the increasing risk experienced in developing countries as a consequence of urbanizations and rapid epidemiologic transitions leading to more affluent societies (Yusuf, S. et al. 2001).

5.2.2 Lifestyle

More than eighty per cent of all the CHD and cerebrovascular diseases are said to be consequences of behavioural risk factors which implies great possibilities for prevention. It also suggests that focusing on individual behavior is important. Behavioural risk factors are likely to play an important role in all ethnic groups (Yusuf, Salim et al. 2001). The most important behavioural risk factors are as mentioned; diet, smoking and physical inactivity. According to the social model of health, however, individuals can only be held responsible for their own health as long as the access to a healthy life is equal and they are equally supported in making healthy choices (WHO 2005). It is therefore essential to examine whether ethnic groups in Norway have equal premises for adopting healthy lifestyles.

Reasons for peoples lifestyles are complex; physical, mental, social and economic factors all play a part (Kerr et al. 2005). Immigrants in our study showed greater tendencies to physical inactivity than the Norwegian-born participants. This finding might correspondingly be due to different reasons. Factors that have been associated with behavioural cardiovascular determinants in other immigrant populations demonstrate the complexity. Some of the factors mentioned are; culture and belief, psychological fatalism, lack of knowledge about modifiable risk factors, mental reactions to both experienced and expected discrimination as well as less advantaged neighbourhoods (Evenson et al. 2004; Kandula et al. 2010; Williams 1998). Hence, some of the obstacles for healthy lifestyles seem to be linked to the fact that ethnic minority groups represent vulnerable groups in societies (Yassi et al. 2001).

Social structures in relation with behavior is understood not only in terms of putting constraints on the individual, but also by functioning as enabling, influencing and motivating (Siegrist & Marmot 2004). The experiences of a positive self in individuals, in particular self-efficacy and

self-esteem, have been identified as important factors for a person's motivation and disposition for healthy behaviour (Bandura 1977; Siegrist & Marmot 2004). A social structure that excludes individuals from belonging and taking part in the society as well as getting positive feedback, undermines the experiences of a positive self (Siegrist & Marmot 2004). Thus, integration of immigrants is essential when enabling people to adopt healthy lifestyles. Integration is a two-way process (IMDi 2008) and can be defined as "harmonic co-existence with mutual exchange of culture and values" (Kogstad 2002, p. 136, own translation). Although it is stated that the integration of immigrants in education and working life in Norway has had a positive trend the latest years, immigrants are still less employed, receive lower income and live in worse conditions than the Norwegian population as a whole (IMDi 2008). This is especially true in the immigrant groups where most have migrated as refugees. Many immigrants report that they have been discriminated when trying to get employed (IMDi 2008). Such experiences may be contributing to an absent experience of positive self and thereby obstructing motivations for healthy lifestyles.

Lifestyle changes are the first choice in primary prevention and should be advised to all people with unhealthy lifestyle regardless of their risk of CVD. Unlike medical treatment, adopting a healthy lifestyle has no known adverse side effects, it is generally less expensive and rather add positive effects to a person's physical and mental wellbeing (Whitworth 2003). In Romsås, Norway; a low-cost, population-based intervention program have shown positive effects on the physical activity level and other risk factors for CVD (Jenum et al. 2006). In the follow-up study, 18 % of the participants were non-Western immigrants and the intervention was a comprehensive package of many different strategies taking a holistic approach, based on theories that included aspects of the participants experience of self, along with other important aspects of behaviour (Jenum et al. 2006).

5.2.3 Social and community networks

As mentioned in the previous subsection, belonging and participating in a society are important factors for positive experiences of self, and thereby for motivations for healthy lifestyles. Immigrants move from their countries for different reasons that involve different health challenges. Either way, migrating involves breaking up from social networks and starting over. Involuntary migration will, in particular, make readjustments even more challenging (Kogstad 2002). The possible consequences of cultures that meet, in relation with immigration, are several (Berry 1997). Integration was defined in the previous section, and may further be understood as

participating in employment, education and the society in general, something that requires efforts from both the majority population as well as from the immigrants (IMDi 2008). How the immigrants adapt to the culture in the host society, highly determines their well-being and health through different ways of social interaction (Berry 1997; Kogstad 2002).

After breaking out from social networks in the country of origin, lack of social networks is a possible consequence for immigrants. This can contribute making them more susceptible for risk of CVD since social networks accommodate the availability of social support, shown to have an important positive effect of several health aspects (Cohen & Wills 1985; Hånes 2008; Kessler & McLeod 1985). Since CVD is multifactorial, positive effects from social support on health in general may also apply for cardiovascular health in particular. Findings from the Oslo Health Study showed that the immigrants from low- and middle-income countries had less social support, less income and lessvpaid work than Norwegian born and immigrants from high-income countries. Lack of social support and social participation have been linked to CVD through possible pathways such as being stress-inducing and leading to unhealthy lifestyles (Dalgard & Lund Håheim 1998). In addition, circumstances like living alone, lacking a confidant, social isolation, low emotional support, low perceived support and lack of available support have in particular been shown to have a negative effect on the prognosis for people who have already experienced a CVD event (Rozanski et al. 2005).

Immigrants in Norway may be exposed to discrimination and racism although the extent is unknown and difficult to measure (NOU 2002:12 2002). Discrimination and racism affect health aspects in a negative way both directly and indirectly (Williams 1999). In the United States, experienced racism have been associated with hypertension, increased psychological distress, depression, stress, poor self-rated health, higher number of smoked cigarettes, low birth weight in the children of the ones who experienced racism, and more reported days spent unwell in bed (Karlsen & Nazroo 2002). Social support was also suggested to mediate the relationship between racism and health (Karlsen & Nazroo 2002).

5.2.4 Living and working conditions

Living and working conditions influence health in various ways. In general, people need to work to earn money so that they can afford what they need to survive and stay healthy. Work may also influence the cardiovascular health of a person through psychosocial factors at the workplace (Karasek et al. 1981). As mentioned, immigrants in general are worse off when it comes to conditions in living and working, than the population as a whole. Immigrants in Norway

experience more lasting poverty than non-immigrants (Bhuller & Aaberge 2010) which might entail higher exposure of stressors that increases the risk of CVD (Elstad 2005; Rosengren et al. 2004). The pathways for increased risk from stress may be both physiological as well as behavioural. In example stress, which occur when challenges are apprehended as too overwhelming for the individual to cope with (Folkman et al. 1986), can lead to increased blood pressure, increased blood glucose and unhealthy lifestyles (Elstad 2005; Surwit et al. 1992).

The social gradient can be found in many ethnic groups, although not always similar - displaying a complexity in the association between SES and cardiovascular health. In some ethnic groups, the social gradient seems to be reversed. As an example, immigrants from Pakistan and Sri Lanka in the Oslo Immigrant Health Profile with the highest education reported about more psychological problems than people with low education – the opposite of other ethnic groups (Kumar et al. 2008). Similar reversed gradients were seen in Turkish and Iranians in relation with smoking (Kumar et al. 2008). This might be related to the epidemiologic transition, and which stage of the transition that the birth countries are in (Yusuf, S. et al. 2001).

Smoking is suggested to be a behavioural risk factor that goes through four stages like an epidemic in populations; the association with education changing from the first to the last stage (Cavelaars et al. 2000). In the first stage, smoking is associated with higher SES, but in the last stage it is associated with lower. Norway and other North European countries, are now in the fourth stage where smoking is most prevalent in the lower SES (Elstad 2005; Strand & Tverdal 2004). Immigrants may come from countries in earlier stages which might be the case with the Turkish and Iranians mentioned above (Cavelaars et al. 2000). Also pointed out is the fact that even if education is considered the most important indicator of SES in relation with CVD in the Nordic region (Norheim et al. 2009), it might be a poor indicator for immigrants knowing that highly educated immigrants in Norway are more likely to live in poverty than the highly educated non-immigrants (Bhuller & Aaberge 2010).

Health care access is also an important determinant of cardiovascular health in populations (Bambra et al. 2010). In America, improvements in CVD have shown to be inequitable distributed among different racial and ethnic minorities partly due to poorer healthcare in the minorities than in Caucasian patients when controlled for income and insurance status (Ferdinand & Armani 2007). Inferior access or utilization of health care, following language and communications barriers may also be a challenge for immigrants in Norway (Larsen 2009).

5.2.4 General socio-economic, cultural and environmental conditions

The epidemiologic transition serves as an illustration of the importance of the wider social, economic and environmental factors in public health. Changes in the environment and the level of development in a society, have shown to determine what type of risk factors populations are exposed to (Yusuf, S. et al. 2001). Following the globalization of today, epidemiologic transition in a country is not an isolated process, since migrants crossing borders might originate from countries in different phases of the transition. This outer layer of the rainbow impact on all the inner layers, and even our genetics seem to be affected -previously exemplified by the "thrifty phenotype hypothesis".

Social and economic contexts may influence on both the maintenance and creation of ethnic differences in health(Gushulak & MacPherson 2006). Addressing ethnic inequalities is therefore also a political concern. An example of this, and the fact that public health is a cross-sector responsibility, is the importance of public policies and taxations in influencing health behaviour. Smoking taxation, pricing, banning of both advertising and smoking in public places, are interventions that have shown to have an effect on reducing smoking symptoms or habits (Eagan et al. 2006; Fichtenberg & Glantz 2002; Hamilton et al. 1997; Saffer & Chaloupka 2000; Warner 1986). In Britain, pricing policies were found to be especially effective on minimizing smoking prevalence among people in the lower socioeconomic groups, possibly due to their purchasing power (Townsend et al. 1994). This might imply that high prices on tobacco can help reduce the overall prevalence of smoking. It may also further contribute to reduce ethnic inequalities in CVD knowing that people with lower SES are most inclined to smoke, and immigrants more likely to be poor in Norway, both mentioned previously.

Culture as one aspect of ethnicity, includes shared beliefs, values, customs and behaviours (Kuhrana 2007). Culture related to lifestyle is an important cardiovascular health determinant and may be responsible for some of the ethnic differences that were found in risk factors. An example of how culture can impact on health behaviour, is the religion Sikhism which prohibits consumption of smoking (Bhopal 2007). Culture may also be one of several explanations to why there are gender differences in smoking, as was evident in the results of our study (Grunberg et al. 1991). Diet, as another behavioural risk factor, is also related to cultural dimensions (Jackson 1994). The culture may determine what kind of food is eaten, how it is prepared and so on. Larger social and economic factors related to the epidemiologic transition may also influence the culture and trends in nutrition in populations (Drewnowski & Popkin 1997).

The layers of the rainbow influencing health are interlinked and influence cardiovascular health in complex manners. It is underlined that this discussion of possible contributing factors to ethnic differences in cardiovascular health, is by no means thought of as exhaustive. Only a few of the many possible interacting factors have been mentioned here as examples of the existing complexity.

Focusing on social and structural factors that creates health inequalities, as has been discussed, imply a redirecting of focus from individual factors towards "the causes of the causes" (Marmot 2007). More research is needed in order to really understand the causal chain of existing inequalities in cardiovascular health, and it is important that research revealing differences among ethnic groups is not only put in the black box of epidemiology - which means that causal mechanisms behind associations remain hidden and not further investigated (Skrabanek 1994).

6.0 Conclusion and implications

CVD is a complex group of diseases highly relevant on the public health agenda. Many risk factors of CVD have been identified and total risk scores developed, which makes it possible to assess risk in individuals free of CVD. Ethnic inequalities in risk of CVD have been revealed and shown to exist in the Norwegian society. Both intermediate and primary risk factors, including those of behaviour, were unequally distributed among the eleven ethnic groups in this study. Factors contributing to the development of atherosclerosis and CVD are many, and they may be found in the surrounding environment as well as in the individual. Even though this study cannot actually explain the causes of ethnic differences in cardiovascular health, suggestions have been discussed and laid out. Following a holistic and social understand of health and its determinants, possible causes may be found in all the layers of the rainbow.

Although there are differences in genetic susceptibility, it seems most likely that the causes for ethnic differences in cardiovascular health are imposed and maintained by structural and social contexts; thereby being both avoidable and inequitable. Knowing that the number of immigrants in Norway will increase; attention towards ethnicity and health from public health policy makers is required. In accordance with the rainbow of determinants, it is necessary to act on several levels, in order to succeed in preventing CVDs in all the ethnic groups. The example from an intervention study in Romsås suggests that low-cost preventions efforts can be successful when taking a holistic approach. There is a call for more research in order to understand the ethnic differences in cardiovascular health, and prevent it from becoming black box epidemiology.

References

Aalen, O. O. (1994). *Innføring i statistikk, med medisinske eksempler*. 2 ed.: Ad Notam Gyldendal AS.

Aamodt, G., Søgaard, A., Næss, Ø., Beckstrøm, A. & Samuelsen, S. (2010). CONOR-databasen–et lite stykke Norge. *Tidsskrift for Den norske legeforening*, 3 (130): 264-265.

Adair, L. S. & Prentice, A. M. (2004). A Critical Evaluation of the Fetal Origins Hypothesis and Its Implications for Developing Countries. *The Journal of Nutrition*, 134 (1): 191-193.

AHA. (2011). *Cigarette smoking and Cardiovascular Diseases*: American Heart Association. Available at: http://www.americanheart.org/presenter.jhtml?identifier=4545 (accessed: 01.05.2011).

Anand, S., Yusuf, S., Vuksan, V., Devanesen, S., Teo, K., Montague, P., Kelemen, L., Yi, C., Lonn, E. & Gerstein, H. (2000). Differences in risk factors, atherosclerosis, and cardiovascular disease between ethnic groups in Canada: the Study of Health Assessment and Risk in Ethnic groups (SHARE). *The Lancet*, 356 (9226): 279-284.

Anand, S. S. (1999). Using ethnicity as a classification variable in health research: perpetuating the myth of biological determinism, serving socio-political agendas, or making valuable contributions to medical sciences? *Ethnicity & health*, 4 (4): 241-244.

Anderson, K. M., Odell, P. M., Wilson, P. W. F. & Kannel, W. B. (1991a). Cardiovascular disease risk profiles. *American heart journal*, 121 (1): 293-298.

Anderson, K. M., Wilson, P., Odell, P. M. & Kannel, W. B. (1991b). An updated coronary risk profile. A statement for health professionals. *Circulation*, 83 (1): 356-362.

Aronson, D. & Rayfield, E. (2002). How hyperglycemia promotes atherosclerosis: molecular mechanisms. *Cardiovascular Diabetology*, 1 (1): 1-10.

Bambra, C., Gibson, M., Sowden, A., Wright, K., Whitehead, M. & Petticrew, M. (2010). Tackling the wider social determinants of health and health inequalities: evidence from systematic reviews. *Journal of Epidemiology and Community Health*, 64 (4): 284-291.

Bandura, A. (1977). Self-efficacy: toward a unifying theory of behavioral change. *Psychological review*, 84 (2): 191-215.

Bansal, S., Buring, J. E., Rifai, N., Mora, S., Sacks, F. M. & Ridker, P. M. (2007). Fasting Compared With Nonfasting Triglycerides and Risk of Cardiovascular Events in Women. *The Journal of the American Medical Association*, 298 (3): 309-316.

Barker, D. & Martyn, C. (1992). The maternal and fetal origins of cardiovascular disease. *British Medical Journal*, 46 (1): 8-11.

Barker, D. J. P. (2007). The origins of the developmental origins theory. *Journal of Internal Medicine*, 261 (5): 412-417.

Barth, F. (1969). *Ethnic groups and boundaries*: Little, Brown.

Bateson, P., Barker, D., Clutton-Brock, T., Deb, D., D'Udine, B., Foley, R. A., Gluckman, P., Godfrey, K., Kirkwood, T., Lahr, M. M., et al. (2004). Developmental plasticity and human health. *Nature*, 430 (6998): 419-421.

Bavdekar, A., Yajnik, C. S., Fall, C., Bapat, S., Pandit, A. N., Deshpande, V., Bhave, S., Kellingray, S. D. & Joglekar, C. (1999). Insulin resistance syndrome in 8-year-old Indian children: small at birth, big at 8 years, or both? *Diabetes*, 48 (12): 2422-2429.

Berger, J. S., Jordan, C. O., Lloyd-Jones, D. & Blumenthal, R. S. (2010). Screening for cardiovascular risk in asymptomatic patients. *Journal of the American College of Cardiology*, 55 (12): 1169-1177.

Berry, J. W. (1997). Immigration, acculturation, and adaptation. *Applied Psychology*, 46 (1): 5-34.

Bhopal, R. (1997). Is research into ethnicity and health racist, unsound, or important science? *British Medical Journal*, 314 (7096): 1751-1756.

Bhopal, R., Unwin, N., White, M., Yallop, J., Walker, L., Alberti, K., Harland, J., Patel, S., Ahmad, N. & Turner, C. (1999). Heterogeneity of coronary heart disease risk factors in Indian, Pakistani, Bangladeshi, and European origin populations: cross sectional study. *British Medical Journal*, 319 (7204): 215-220.

Bhopal, R., Fischbacher, C., Vartiainen, E., Unwin, N., White, M. & Alberti, G. (2005). Predicted and observed cardiovascular disease in South Asians: application of FINRISK, Framingham and SCORE models to Newcastle Heart Project data. *Journal of Public Health*, 27 (1): 93-100.

Bhopal, R. S. (2007). *Ethnicity, Race, and Health in Multicultural Societies. Foundations for better epidemiology, public health and health care*. Oxford: Oxford University Press.

Bhuller, M. & Aaberge, R. (2010). *Vedvarende økonomisk fattigdom blant innvandrere, en empirisk analyse for perioden 1993-2007*. Rapporter 32/2010. . Oslo-Kongsvinger: Statistics Norway. Available at: http://www.ssb.no/emner/05/01/rapp_201032/rapp_201032.pdf (accessed: 01.05.2011).

Biggerstaff, K. D. & Wooten, J. S. (2004). Understanding lipoproteins as transporters of cholesterol and other lipids. *Advances in Physiology Education*, 28 (3): 105-106.

Bjørndal, A. & Hofoss, D. (2008). *Statistikk for helse- og sosialfagene.* 2 ed. Oslo: Gyldendal Akademisk.

Bozorgmanesh, M., Hadaegh, F. & Azizi, F. (2011). Predictive accuracy of the 'Framingham's general CVD algorithm' in a Middle Eastern population: Tehran Lipid and Glucose Study. *International Journal of Clinical Practice*, 65 (3): 264-273.

Braveman, P. & Gruskin, S. (2003). Defining equity in health. *Journal of Epidemiology and Community Health*, 57 (4): 254-258.

Braveman, P. A., Egerter, S. A. & Mockenhaupt, R. E. (2011). Broadening the Focus: The Need to Address the Social Determinants of Health. *American Journal of Preventive Medicine*, 40 (1, Supplement 1): S4-S18.

Brindle, P., May, M., Gill, P. S., Cappuccio, F., D'Agostino, R., Fischbacher, C. & Ebrahim, S. B. J. (2006). Primary prevention of cardiovascular disease: a web-based risk score for seven British black and minority ethnic groups. *Heart*, 92 (11): 1595-1602.

Burns, D. K. & Kumar, V. (2003). The Heart. In Kumar, V., Cotran, R. S. & Robbins, S. L. (eds) *Robbins basic pathology*: Saunders Philadelphia, PA.

Castelli, W. P. (1996). Lipids, risk factors and ischaemic heart disease. *Atherosclerosis*, 124 (Supplement 1): S1-S9.

Cavelaars, A. E. J. M., Kunst, A. E., Geurts, J. J. M., Crialesi, R., Grötvedt, L., Helmert, U., Lahelma, E., Lundberg, O., Matheson, J. & Mielck, A. (2000). Educational differences in smoking: international comparison. *British Medical Journal*, 320 (7242): 1102-1107.

CDHN. (s.a.). *The Social Model of Health*: Community Development and Health Network. Available at: http://www.cdhn.org/documentbank/uploads/Inscape%20Briefing%20Paper%201%20-%20Social%20Model.pdf (accessed: 22.04.2011).

Chaturvedi, N. (2003). Ethnic Differences in Cardiovascular Disease. *Heart*, 89 (6): 681-686.

Clare-Salzler, M. J., Crawford, J. M. & Kumar, V. (2003). The Pancreas. In Kumar, V., Cotran, R. S. & Robbins, S. L. (eds) *Robbins basic pathology*: Saunders Philadelphia, PA.

Claussen, B. & Naess, O. (2002). Dødelighet i Oslo etter ulikheter i yrkesklasse. *Tidsskrift for Den norske lægeforening*, 122 (19): 1867-1869.

Cohen, S. & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological bulletin*, 98 (2): 310-357.

Conroy, R., Pyörälä, K., Fitzgerald, A., Sans, S., Menotti, A., De Backer, G., De Bacquer, D., Ducimetiere, P., Jousilahti, P. & Keil, U. (2003). Estimation of ten-year risk of fatal cardiovascular disease in Europe: the SCORE project. *European Heart Journal*, 24 (11): 987-1003.

Cullen, P. (2000). Evidence that triglycerides are an independent coronary heart disease risk factor. *The American journal of cardiology*, 86 (9): 943-949.

D'Agostino, R. B., Wolf, P. A., Belanger, A. J. & Kannel, W. B. (1994). Stroke risk profile: adjustment for antihypertensive medication. The Framingham Study. *Stroke*, 25 (1): 40-43.

D'Agostino, R. B., Grundy, S., Sullivan, L. M. & Wilson, P. (2001). Validation of the Framingham coronary heart disease prediction scores: results of a multiple ethnic groups investigation. *The Journal of the American Medical Association*, 286 (2): 180-187.

D'Agostino, R. B., Sr., Vasan, R. S., Pencina, M. J., Wolf, P. A., Cobain, M., Massaro, J. M. & Kannel, W. B. (2008). General cardiovascular risk profile for use in primary care: the Framingham Heart Study. *Circulation*, 117 (6): 743-53.

Dalgard, O. S. & Lund Håheim, L. (1998). Psychosocial risk factors and mortality: a prospective study with special focus on social support, social participation, and locus of control in Norway. *Journal of Epidemiology and Community Health*, 52 (8): 476-481.

Dalton, M., Cameron, A. J., Zimmet, P. Z., Shaw, J. E., Jolley, D., Dunstan, D. W., Welborn, T. A. & On behalf of the AusDiab steering, c. (2003). Waist circumference, waist–hip ratio and body mass index and their correlation with cardiovascular disease risk factors in Australian adults. *Journal of Internal Medicine*, 254 (6): 555-563.

De Backer, G., Ambrosioni, E., Borch-Johnsen, K., Brotons, C., Cifkova, R., Dallongeville, J., Ebrahim, S., Faergeman, O., Graham, I. & Mancia, G. (2003). European guidelines on cardiovascular disease prevention in clinical practice. *European Heart Journal*, 24 (17): 1601-1610.

Despopoulos, A. & Silbernagl, S. (2003). *Color Atlas of Physiology*. Berlin: Georg Thieme Verlag.

Detels, R. & Breslow, L. (2002). Current scope and concerns in public health In Detels, J., McEwen, J., Beaglehole, R. & Tanaka, H. (eds) *Oxford Textbook of Public Health* pp. 149-170. New York: Oxford University Press.

Drewnowski, A. & Popkin, B. M. (1997). The Nutrition Transition: New Trends in the Global Diet. *Nutrition Reviews*, 55 (2): 31-43.

Eagan, T. M. L., Hetland, J. & Aarø, L. E. (2006). Decline in respiratory symptoms in service workers five months after a public smoking ban. *Tobacco Control*, 15 (3): 242-246.

Elstad, J. I. (2005). *Sosioøkonomiske ulikheter i helse - teorier og forklaringer* Oslo: Norwegian Directorate for Health and Social Affairs.

Evenson, K. R., Sarmiento, O. L. & Ayala, G. X. (2004). Acculturation and physical activity among North Carolina Latina immigrants. *Social Science & Medicine*, 59 (12): 2509-2522.

Fennelly, K. (2007). The" healthy migrant" effect. *Minnesota medicine*, 90 (3): 51-53.

Ferdinand, K. & Armani, A. (2007). Combating racial and ethnic disparities in cardiovascular disease: What is being done? *Current Cardiovascular Risk Reports*, 1 (2): 124-132.

Fichtenberg, C. M. & Glantz, S. A. (2002). Effect of smoke-free workplaces on smoking behaviour: systematic review. *British Medical Journal*, 325 (7357): 1-7.

Folkman, S., Lazarus, R. S., Gruen, R. J. & DeLongis, A. (1986). Appraisal, coping, health status, and psychological symptoms. *Journal of personality and social psychology*, 50 (3): 571-579.

Forsdahl, A. (1977). Are poor living conditions in childhood and adolescence an important risk factor for arteriosclerotic heart disease? *British journal of preventive & social medicine*, 31 (2): 91-95.

Framingham Heart Study. (2011a). *About the Framingham Heart Study participants*. Available at: http://www.framinghamheartstudy.org/participants/index.html (accessed: 23.04.2011).

Framingham Heart Study. (2011b). *History of the Framingham Heart Study*. Available at: http://www.framinghamheartstudy.org/about/history.html (accessed: 23.04.2011).

Freeman, V. S. (2005). Carbohydrates. In Bishop, M. L., Fody, E. P. & Schoeff, L. E. (eds) *Clinical Chemistry: principles, procedures, correlations*: Lippincott Williams & Wilkins.

Grunberg, N. E., Winders, S. E. & Wewers, M. E. (1991). Gender differences in tobacco use. *Health Psychology*, 10 (2): 143-153.

Grundy, S., Pasternak, R., Greenland, P., Smith Jr, S. & Fuster, V. (1999). Assessment of cardiovascular risk by use of multiple-risk-factor assessment equations: a statement for healthcare professionals from the American Heart Association and the American College of Cardiology. *Circulation*, 100 (13): 1481-1492.

Gushulak, B. D. & MacPherson, D. W. (2006). The basic principles of migration health: population mobility and gaps in disease prevalence. *Emerging Themes in Epidemiology*, 3 (3).

Hales, C. N. & Barker, D. J. P. (2001). The thrifty phenotype hypothesis. *British Medical Bulletin*, 60 (1): 5-20.

Hamilton, V. H., Levinton, C., St-Pierre, Y. & Grimard, F. (1997). The effect of tobacco tax cuts on cigarette smoking in Canada. *Canadian Medical Association Journal*, 156 (2): 187-191.

Hånes, H. (2008). *Sosial støtte og ensomhet - Faktaark*: Norwegian Institute of Public Health. Available at:
http://www.fhi.no/eway/default.aspx?pid=233&trg=MainLeft_5648&MainArea_5661=5648:0:1 5,2917:1:0:0:::0:0&MainLeft_5648=5544:69765::1:5647:58:::0:0 (accessed: 08.05.2011).

IMDi. (2008). *Hvordan går det med integreringen? Årsrapport 2008*. IMDis årsapport: The Directorate of Integration and Diversity in Norway,. Available at:
http://www.imdi.no/no/Arsrapporter/Aarsrapport2008/Arsrapport-2008-i-pdf/ (accessed: 08.05.2011).

Ingierd, H. & Fossheim, H. J. (2009). *Etniske grupper*: FBIB, Forskningsetisk Bibliotek. Forskningsetiske Komiteer. Available at: http://www.etikkom.no/no/FBIB/Temaer/Forskning-pa-bestemte-grupper/Etniske-grupper/#_Toc229229605 (accessed: 06.05.2011).

Jackson, M. Y. (1994). Diet, culture, and diabetes. In Joe, J. R. & Young, R. (eds) *Diabetes as a Disease of Civilization: The Impact of Culture Change on Indigenous Peoples*, pp. 381-406. Berlin: Walter de Gruyter & Co.

Jenum, A. K., Lorentzen, C., Anderssen, S. A., Birkeland, K. I., Holme, I., Lund-Larsen, P. G., Ommundsen, Y., Raastad, T., Thelle, D. S. & Bahr, R. (2006). Promoting physical activity in a multi-ethnic district-methods and baseline results of a pseudo-experimental intervention study. *Diabetes Care*, 29 (7): 1605-1612.

Kain, K. & Catto, A. J. (2002). Insulin resistance syndrome in South Asians. *The British Journal of Diabetes & Vascular Disease*, 2 (5): 398-402.

Kandula, N. R., Tirodkar, M. A., Lauderdale, D. S., Khurana, N. R., Makoul, G. & Baker, D. W. (2010). Knowledge Gaps and Misconceptions About Coronary Heart Disease Among US South Asians. *American Journal of Preventive Medicine*, 38 (4): 439-442.

Kannel, W. B., McGee, D. & Gordon, T. (1976). A general cardiovascular risk profile: the Framingham Study. *The American journal of cardiology*, 38 (1): 46-51.

Kannel, W. B., D'Agostino, R. B., Silbershatz, H., Belanger, A. J., Wilson, P. W. F. & Levy, D. (1999). Profile for estimating risk of heart failure. *Archives of internal medicine*, 159 (11): 1197-1204.

Karasek, R., Baker, D., Marxer, F., Ahlbom, A. & Theorell, T. (1981). Job decision latitude, job demands, and cardiovascular disease: a prospective study of Swedish men. *American Journal of Public Health*, 71 (7): 694-705.

Karlsen, S. & Nazroo, J. Y. (2002). Relation Between Racial Discrimination, Social Class, and Health Among Ethnic Minority Groups. *American Journal of Public Health*, 92 (4): 624-631.

Kerr, J., Weitkunat, R. & Moretti, M. (eds). (2005). *ABC of Behavior Change. A guide to successful disease prevention and health promotion.* Oxford, UK: Elsevier Churchill Livingstone.

Kessler, R. C. & McLeod, J. D. (1985). Social support and mental health in community samples. In Cohen, S. & Syme, S. L. (eds) *Social support and health*, pp. 219-240. San Diego, CA: Academic Press.

Khan, F. A., Zia, E., Janzon, L. & Engstrom, G. (2004). Incidence of stroke and stroke subtypes in Malmo, Sweden, 1990-2000: marked differences between groups defined by birth country. *Stroke*, 35 (9): 2054-2058.

Kinosian, B., Glick, H. & Garland, G. (1994). Cholesterol and Coronary Heart Disease: Predicting Risks by Levels and Ratios. *Annals of Internal Medicine*, 121 (9): 641-647.

Klepp, K.-I. (2010). Health Promotion - achieving good health for all. IS-1846E.

Kogstad, R. E. (2002). *Miljørettet helsevern. Folkehelse, forebygging og samfunn. Kap.8: Nettverksarbeid.* Bergen: Fagbokforlaget.

Krieger, N. (2001). Theories for social epidemiology in the 21st century: an ecosocial perspective. *International Journal of Epidemiology*, 30 (4): 668-677.

Kuhrana, A. (2007). Understanding Cultural Formulation. *Resident's Journal*, 2 (10): 3-4. Available at: http://ajp.psychiatryonline.org/cgi/data/164/10/A34/DC2/1 (accessed: 09.05.2011).

Kumar, B. N., Grøtvedt, L., Meyer, H. E., Søgaard, A. J. & Strand, B. H. (2008). The Oslo Immigrant Health Profile. *Diabetes*, 7. Oslo,: Norwegian Institute of Public Health.

Larsen, B. I. (2009). Migrasjon og helse - utfordringer og utviklingstrekk. *Norwegian Directorate of Health*, IS-1663.

Liburd, L. C. & Jack, L. (2005). Intervening on the social determinants of cardiovascular disease and diabetes. *American Journal of Preventive Medicine*, 29 (5): 18-24.

Lindman, A. S., Selmer, R., Tverdal, A., Pedersen, J. I., Eggen, A. E. & Veierød, M. B. (2006). The SCORE risk model applied to recent population surveys in Norway compared to observed mortality in the general population. *European Journal of Cardiovascular Prevention & Rehabilitation*, 13 (5): 731-737.

Lindman, A. S., Veierød, M. B., Pedersen, J. I., Tverdal, A., Njølstad, I. & Selmer, R. (2007). The ability of the SCORE high-risk model to predict 10-year cardiovascular disease mortality in Norway. *European Journal of Cardiovascular Prevention & Rehabilitation*, 14 (4): 501-507.

Mackay, J., Mensah, G. A., Mendis, S. & Greenlund, K. (2004). *The atlas of heart disease and stroke*. Geneva: World Health Organization.

Mancia, G., De Backer, G., Dominiczak, A., Cifkova, R., Fagard, R., Germano, G., Grassi, G., Heagerty, A. M., Kjeldsen, S. E., Laurent, S., et al. (2007). 2007 Guidelines for the management of arterial hypertension. *European Heart Journal*, 28 (12): 1462-1536.

Marmot, M. (2007). Achieving health equity: from root causes to fair outcomes. *The Lancet*, 370 (9593): 1153-1163.

Marmot, M., Friel, S., Bell, R., Houweling, T. A. J. & Taylor, S. (2008). Closing the gap in a generation: health equity through action on the social determinants of health. *The Lancet*, 372 (9650): 1661-1669.

Midthjell, K., Holmen, J., Bjørndal, A. & Lund-Larsen, G. (1992). Is questionnaire information valid in the study of a chronic disease such as diabetes? The Nord-Trøndelag diabetes study. *Journal of Epidemiology and Community Health*, 46 (5): 537-542.

Naidoo, J. & Wills, J. (2000). *Health promotion: foundations for practice*. 2. ed. London: Elsevier Limited. Bailliere-Tindale & Royal College of Nursing.

Nazroo, J. Y. (1998). Genetic, Cultural or Socio-economic Vulnerability? Explaining Ethnic Inequalities in Health. *Sociology of Health & Illness*, 20 (5): 710-730.

Neel, J. V. (1962). Diabetes mellitus: a "thrifty" genotype rendered detrimental by "progress"? *American journal of human genetics*, 14 (4): 353-362.

NEL- Nevrologiske Prosedyrer. (2001). *HDL-kolesterol*. In Ljøstad, U. & Mygland, Å. (eds). Trondheim: Norsk Helseinformatikk AS (NHI.no). Available at: http://nevro.legehandboka.no/prover-og-svar/klinisk-kjemi/blodprover/hdl-kolesterol-2980.html (accessed: 13.0.2011).

NIPH. (2005). *The Oslo Immigrant Health Study*: Norwegian Institutet of Public Health. Available at: http://www.fhi.no/eway/default.aspx?pid=238&trg=MainLeft_5895&MainArea_5811=5895:0:1 5,4562:1:0:0:::0:0&MainLeft_5895=5825:53584::1:5896:3:::0:0 (accessed: 05.04.2011).

Norheim, O. F., Gjelsvik, B., Kjeldsen, S. E., Klemsdal, T. O., Madsen, S., Meland, E., Narvesen, S., Negård, A., Njølstad, I., Tonstad, S., et al. (2009). Nasjonale retningslinjer for individuell primærforebygging av hjerte- og karsykdommer. *Norwegian Directorate of Health*, IS-1550.

NOU 1998:18. *Det er bruk for alle. Styrking av folkehelsearbeidet i kommunene*. Oslo: Norwegian Ministry of Health and Social Affairs.

NOU 2002:12. *Rettslig vern mot etnisk diskriminering*. Oslo: Norwegian Ministry of Labour.

Næss, Ø., Søgaard, A. J., Arnesen, E., Beckstrøm, A. C., Bjertness, E., Engeland, A., Hjort, P. F., Holmen, J., Magnus, P., Njølstad, I., et al. (2008). Cohort Profile: Cohort of Norway (CONOR). *International Journal of Epidemiology*, 37 (3): 481-485.

Omran, A. R. (2005). The epidemiologic transition: a theory of the epidemiology of population change. *Milbank Quarterly*, 83 (4): 731-757.

Oparil, S., Zaman, M. A. & Calhoun, D. A. (2003). Pathogenesis of Hypertension. *Annals of Internal Medicine*, 139 (9): 761-776.

Patrick, D. L., Cheadle, A., Thompson, D. C., Diehr, P., Koepsell, T. & Kinne, S. (1994). The validity of self-reported smoking: a review and meta-analysis. *American Journal of Public Health*, 84 (7): 1086-1093.

Phillimore, P., Beattie, A. & Townsend, P. (1994). Widening inequality of health in northern England, 1981-91. *British Medical Journal*, 308 (6937): 1125-1128.

Prop. 90 L (2010-2011). *Proposisjon til Stortinget (forslag til lovvedtak). Lov om folkehelsearbeid (folkehelseloven)*. Oslo: Ministry of Health and Care Services.

Remaley, A. T., Judith, R. M. & Warnick, G. R. (2005). Lipids and Lipoproteins. In Bishop, M. L., Fody, E. P. & Schoeff, L. E. (eds) *Clinical chemistry: principles, procedures, correlations*: Lippincott Williams & Wilkins.

Rosengren, A., Hawken, S., Ôunpuu, S., Sliwa, K., Zubaid, M., Almahmeed, W. A., Blackett, K. N., Sitthi-amorn, C., Sato, H. & Yusuf, S. (2004). Association of psychosocial risk factors with risk of acute myocardial infarction in 11 119 cases and 13 648 controls from 52 countries (the INTERHEART study): case-control study. *The Lancet*, 364 (9438): 953-962.

Rothman, K. J. (2002). *Epidemiology. An Introduction.* New York: Oxford University Press.

Rozanski, A., Blumenthal, J. A., Davidson, K. W., Saab, P. G. & Kubzansky, L. (2005). The epidemiology, pathophysiology, and management of psychosocial risk factors in cardiac practice:: The emerging field of behavioral cardiology. *Journal of the American College of Cardiology*, 45 (5): 637-651.

Saffer, H. & Chaloupka, F. (2000). The effect of tobacco advertising bans on tobacco consumption. *Journal of health economics*, 19 (6): 1117-1137.

Schoen, F. J. & Cotran, R. S. (2003). The Blood Vessels. In Kumar, V., Cotran, R. S. & Robbins, S. L. (eds) *Robbins basic pathology*: Saunders Philadelphia, PA.

Selmer, R., Lindman, A., Tverdal, A., Pedersen, J., Njølstad, I. & Veierød, M. (2008). Modell for estimering av kardiovaskulær risiko i Norge. *Tidsskrift for Den norske legeforening*, 128 (3): 286-290.

Siegrist, J. & Marmot, M. (2004). Health inequalities and the psychosocial environment-two scientific challenges. *Social Science & Medicine*, 58 (8): 1463-1473.

Simmons, R. K., Coleman, R. L., Price, H. C., Holman, R. R., Khaw, K.-T., Wareham, N. J. & Griffin, S. J. (2009). Performance of the UK Prospective Diabetes Study Risk Engine and the Framingham Risk Equations in Estimating Cardiovascular Disease in the EPIC- Norfolk Cohort. *Diabetes Care*, 32 (4): 708-713.

Skrabanek, P. (1994). The emptiness of the black box. *Epidemiology*, 5 (5): 553-555.

Sleight, P. (2002). *The Systematic Coronary Risk Evaluation (SCORE)*. Medscape Public Health & Prevention. Available at: http://www.medscape.com/viewarticle/438575 (accessed: 11.05.2011).

Statistics Norway. (2010a). *Continued high growth of the number of immigrants* Available at: http://www.ssb.no/innvfram_en/ (accessed: 23.04.2011).

Statistics Norway. (2010b). *Immigrants and Norwegian-born to immigrant parents, by country background. 1970-2010.* Available at: http://www.ssb.no/english/subjects/02/01/10/innvbef_en/fig-2010-04-29-01-en.html (accessed: 23.04.2011).

Statistics Norway. (2010c). *Immigration and immigrants*. Available at: http://www.ssb.no/english/subjects/00/00/10/innvandring_en/.

Statistics Norway. (2011). *Many new European immigrants.* Available at: http://www.ssb.no/innvbef_en/ (accessed: 05.05.2011).

Strand, B. H. & Tverdal, A. (2004). Can cardiovascular risk factors and lifestyle explain the educational inequalities in mortality from ischaemic heart disease and from other heart diseases? 26 year follow up of 50 000 Norwegian men and women. *Journal of Epidemiology and Community Health*, 58 (8): 705-709.

Surwit, R. S., Schneider, M. S. & Feinglos, M. N. (1992). Stress and diabetes mellitus. *Diabetes Care*, 15 (10): 1413-1422.

Søgaard, A. J., Selmer, R., Bjertness, E. & Thelle, D. (2004). The Oslo Health Study: The impact of self-selection in a large, population-based survey. *International Journal for Equity in Health*, 3 (1): 1-12.

Søgaard, A. J. (2007). *Cohort Norway (CONOR): Materials and methods* Norwegian Institute of Public Health. Available at: http://www.fhi.no/dokumenter/fc136f94b6.pdf (accessed: 04.04.2011).

Townsend, J., Roderick, P. & Cooper, J. (1994). Cigarette smoking by socioeconomic group, sex, and age: effects of price, income, and health publicity. *British Medical Journal*, 309 (6959): 923-927.

Vangen, S., Nordhagen, R. & Lie, K. (2005). Gjensyn med Forsdahl-Barker-hypotesen. *Tidsskrift for Den norske lægeforening*, 125: 451-453.

Via, S., Gomulkiewicz, R., De Jong, G., Scheiner, S. M., Schlichting, C. D. & Van Tienderen, P. H. (1995). Adaptive phenotypic plasticity: consensus and controversy. *Trends in Ecology & Evolution*, 10 (5): 212-217.

Warner, K. E. (1986). Smoking and Health Implications of a Change in the Federal Cigarette Excise Tax. *The Journal of the American Medical Association*, 255 (8): 1028-1032.

Weder, A. B. (2007). Evolution and Hypertension. *Hypertension*, 49 (2): 260-265.

Whitehead, M., Dahlgren, G. & Gilson, L. (2001). *Developing the policy response to inequities in health: a global perspective*. Challenging inequities in health: From ethics to action. Available at: http://ot.creighton.edu/community/Readings/Literature%20Bank%20I%20OTD522/LB%20Advocacy/Developing_the_Policy_Response.pdf (accessed: 11.05.2011).

Whitworth, J. (2003). 2003 World Health Organization (WHO)/International Society of Hypertension (ISH) statement on management of hypertension. *Journal of hypertension*, 21 (11): 1983-1992.

WHO. (1948). *Preamble to the Constitution of the World Health Organization as adopted by the International Health Conference, New York, 19-22 June, 1946; signed on 22 July 1946 by the representatives of 61 States (Official Records of the World Health Organization, no. 2, p. 100) and entered into force on 7 April 1948*: World Health Organization. Available at: http://whqlibdoc.who.int/hist/official_records/2e.pdf (accessed: 20.04.2001).

WHO. (1998). *Health Promotion Glossary*. Geneva: World Health Organization.

WHO. (2003). *The world health report 2003: shaping the future*. Geneva: World Health Organization.

WHO. (2004). Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. World Health Organization Expert Consultation,. *The Lancet*, 363 (9403): 157-163.

WHO. (2005). *Preventing chronic disease: a vital investment*. Geneva: World Health Organization.

WHO. (2009). *Global health risks: mortality and burden of disease attributable to selected major risks*. Geneva: World Health Organization. .

WHO. (2011a). *Cardiovascular diseases (CVDs)*. Fact Sheet °317: World Health Organization. Available at: http://www.who.int/mediacentre/factsheets/fs317/en/index.html (accessed: 05.03.2011).

WHO. (2011b). *Obesity and overweight*. Fact sheet N°311: World Health Organization. Available at: http://www.who.int/mediacentre/factsheets/fs311/en/ (accessed: 01.05.2011).

WHO. (2000). *Obesity: preventing and managing the global epidemic. Report of a WHO Consultation*. WHO Technical Report Series; 894. Geneva: World Health Organization.

Williams, D. R. (1998). African-American health: the role of the social environment. *Journal of Urban Health*, 75 (2): 300-321.

Williams, D. R. (1999). Race, Socioeconomic Status, and Health The Added Effects of Racism and Discrimination. *Annals of the New York Academy of Sciences*, 896 (1): 173-188.

Wilson, P., D'Agostino, R., Levy, D., Belanger, A., Silbershatz, H. & Kannel, W. (1998). Prediction of coronary heart disease using risk factor categories. *Circulation*, 97 (18): 1837-1847.

Winter, G. (2000). *A comparative discussion of the notion of validity in qualitative and quantitative research*. The qualitative report. Available at: http://www.nova.edu/ssss/QR/QR4-3/winter.html (accessed: 11.05.2011).

WMA. (2008). *World Medical Association Declaration of Helsinki. Ethical Principles for Medical Research Involving Human Subjects. Adopted June 1964, last amended october 2008.*: The World Medical Association. Available at: http://www.wma.net/en/30publications/10policies/b3/17c.pdf (accessed: 01.05.2011).

Yajnik, C. S. (2001). The Insulin Resistance Epidemic in India: Fetal Origins, Later Lifestyle, or Both? *Nutrition Reviews*, 59 (1): 1-9.

Yassi, A., Kjellstrøm, T., de Kok, T. & Guidotti, T. L. (2001). *Basic environmental health*. New York: Oxford University Press.

Yusuf, S., Reddy, S., Ounpuu, S. & Anand, S. (2001). Global burden of cardiovascular diseases: part I: general considerations, the epidemiologic transition, risk factors, and impact of urbanization. *Circulation*, 104 (22): 2746-2753.

Yusuf, S., Reddy, S., Ounpuu, S. & Anand, S. (2001). Global Burden of Cardiovascular Diseases: Part II: Variations in Cardiovascular Disease by Specific Ethnic Groups and Geographic Regions and Prevention Strategies. *Circulation*, 104 (23): 2855-2864.

Yusuf, S., Hawken, S., Ôunpuu, S., Dans, T., Avezum, A., Lanas, F., McQueen, M., Budaj, A., Pais, P. & Varigos, J. (2004). Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *The Lancet*, 364 (9438): 937-952.

Zomer, E., Owen, A., Magliano, D. J., Liew, D. & Reid, C. (2011). Validation of two Framingham cardiovascular risk prediction algorithms in an Australian population: the 'old' versus the 'new' Framingham equation. *European Journal of Cardiovascular Prevention & Rehabilitation*, 18 (1): 115-120.

Article:

Ethnic differences in risk factors and total risk of cardiovascular

disease in Norway: a cross sectional study.

ETHNIC DIFFERENCES IN RISK FACTORS AND TOTAL RISK OF CARDIOVASCULAR DISEASE IN NORWAY: A CROSS SECTIONAL STUDY

Kjersti S. Rabanal [a], Anja S. Lindman [b], Geir Aamodt [a, c]
[a] Norwegian University of Life Sciences, [b] Norwegian Knowledge Center for the Health Services, [c] Norwegian Institute of Public Health, Oslo, Norway.

Abstract

Background Risk of cardiovascular disease (CVD) varies between ethnic groups. The aim of this study was to investigate differences in cardiovascular risk factors and total cardiovascular risk using NORRISK and Framingham equations, between ethnic groups in Norway.

Design A cross-sectional study using data from the Cohort of Norway (CONOR).

Methods A sample of 62 145 participants, 40-65 years of age, originating from 11 different geographical regions including Norway, were examined. Self-reported variables, blood samples and physical measurements were used to estimate age-adjusted mean levels of CVD risk factors. 10-year risk of CVD events were calculated for 60 015 participants using an updated version of the Framingham risk equation. NORRISK was used to calculate 10-year risk of cardiovascular mortality in 54 027 of the participants.

Results Differences in risk factors and in Framingham risk scores were observed in both men and women. NORRISK showed differences in men only. Immigrants from the Indian subcontinent had the highest levels of blood glucose, waist hip ratio (WHR), triglycerides, the lowest high density lipoprotein (HDL) levels, and the highest prevalence of diabetes. Immigrants from Former Yugoslavia had the highest Framingham risk scores and the highest total cholesterol/HDL ratio, high levels of blood pressure, overweight measures and smoking. Lower risk levels were particularly observed in immigrants from East Asia. Most of the groups reported high levels of inactivity and smoking.

Conclusion Previously reported excess risk of diabetes and other risk factors of CVD in immigrants from the Indian subcontinent were supported in this study. We also showed that immigrants from Former Yugoslavian countries have a higher total 10-year risk of CVD events than other ethnic groups. CVDs are preventable diseases, and this study adds new information about ethnic groups in Norway which need to be addressed with further research and primary prevention efforts.

Keywords cardiovascular diseases, risk factors, ethnic groups, lipids, NORRISK, Framingham

Introduction

Cardiovascular disease (CVD) is the leading cause of death worldwide, a major cause of disability and at the same time highly preventable (WHO 2007). Affecting both rich and poor countries, CVD is increasing in developing countries carrying the heaviest load of more than 80 per cent of global deaths (Yusuf, S. et al. 2001). Improvements in prevention, better diagnostics and treatment in many developed countries have resulted in an opposite trend (Yusuf, S. et al. 2001). Norway is an example having experienced a significant decline in cardiovascular mortality the past twenty years (Statistics Norway 2010). Still CVD persists as the national leading cause of death, emphasizing the importance of focusing on prevention. Asymptomatic patients with high risk need to be identified in order to offer primary prevention to those most likely to benefit from it (Norheim et al. 2009).

A growing amount of evidence indicates unequal distribution of CVD events and CVD risk factors based on ethnicity (Chaturvedi 2003; Forouhi & Sattar 2006; Kurian & Cardarelli 2007). In particular, immigrants from South Asia (the Indian Subcontinent) have repeatedly been found to have increased risk of ischaemic heart disease (IHD) (Lee et al. 2001; McKeigue et al. 1989; Wild & Mckeigue 1997), higher prevalence of insulin resistance and several other metabolic disturbances (Chambers et al. 2001; Mather & Keen 1985; Tillin et al. 2005). People of African descents have been reported to have increased risk of hypertension and stroke when compared to Europeans (Balarajan 1991; Chaturvedi et al. 1993). Despite several studies revealing such disparities, a lack of data on this topic implies a demand for more research (Ranganathan & Bhopal 2006). This need is especially pressing in other European countries than the UK, where most existing studies have been carried out (Bhopal 2000).

Two previous studies in Norway documented differences in CVD risk among immigrants living in Oslo. Individuals originating from five countries in Asia and the Middle East, in addition to people born in Norway were compared in both studies (Glenday et al. 2006; Kumar et al. 2009). No study has explored ethnic differences in risk of CVD among other immigrant groups in Norway, nor studied immigrants living outside of Oslo in this respect. Using data from a large Norwegian cohort, this study's main objective was to investigate variations in risk factors for and total risk of CVD among eleven ethnic groups. Total risk of CVD was calculated using NORRISK and Framingham risk score models.

Materials and Methods

Cohort of Norway (CONOR)

This is a cross sectional study, using data from the Cohort of Norway (CONOR); a database made up of ten different Norwegian health surveys. The surveys were carried out during the time period 1994-2003. A total number of 174 430 individuals participated in at least one CONOR-survey; some of them participated in one or two more. Overall participation rate is 58 %. Details on each of the surveys have been reported elsewhere (Næss et al. 2008). CONOR was established through collaboration between the Norwegian Institute of Public Health (NIPH) and the epidemiological centers at the University of Tromsø, The Norwegian University of Science and Technology in Trondheim, the University of Bergen and the University of Oslo (Aamodt et al. 2010; Næss et al. 2008).

In all the surveys, invited persons were asked to fill in a questionnaire covering variables such as smoking habits, drug use, and health condition. Included variables from the CONOR questionnaire are self-reported diseases such as diabetes, myocardial infarction (MI) and angina pectoris. Other questions revealing smoking habits, physical activity, time since last meal, use of blood pressure and lipid lowering medication have also been included. Physical inactivity was defined as reporting less than 1 hour of both light (no sweating or out of breath) and hard (sweating or out of breath) physical activity when the respondents were asked to estimate an average of activity in a typical week during the previous year. Places of birth act as indicators of ethnicity which is understood as a concept including biologic, social and cultural aspects (Bhopal 2004). In the available dataset for our study, countries of birth were merged into 16 larger birth regions; Table 1 shows which countries that belong to each region. Information about the number of participants from each country represented in each region, are thus not obtainable in the current study.

Moreover, a simple clinical examination was performed. Body weight (kilograms) and height (centimeters) were measured manually until the year of 2000, after that an electronic height and weight scale was applied. All participants wore light clothing without shoes for these measurements. Body mass index (BMI) was calculated as body weight (in kilograms) divided by the square of height (in metres). Waist and hip circumference were measured with a tape of steel placed horizontally while the participants were standing and breathing normally. The waist to hip ratio (WHR) was calculated based on these measurements. Blood pressure was measured in mmHg using an automatic instrument (DINAMAP, Criticon, Tampa, FL, USA). The participants

rested for two minutes followed by three blood pressure measurements with one-minute intervals. The last two measurements were used to calculate mean systolic blood pressure which were applied in our analyses.

At the clinical examination, a non-fasting venous blood sample was drawn. Total cholesterol (mmol/L), high density lipoprotein (HDL) cholesterol (mmol/L), glucose (mmol/L) and triglycerides (mmol/L) were measured directly using an enzymatic method (Boehringer 148393, Boehringer-Mannheim, Federal Republic of Germany –from 2000 Hitachi 917 auto analyzer, Roche Diagnostics, Switzerland). The analyses were performed by the Department of Clinical Chemistry, Ullevål University Hospital, in Oslo; the Department of Clinical Chemistry, Levanger Hospital; and the Department of Clinical Chemistry, University Hospital North-Norway, Tromsø. In connection to these surveys, calibration procedures between the laboratories were carried out (Næss et al. 2008).

Study sample

The sample was restricted to the first time the individuals participated in a CONOR-survey. A number of 38 066 individuals missed information on birth place and were consequently excluded. Regions with few representatives (the Pacific Ocean (n=8), Central America (n=55), Oceania/Pacific (n=31), Central Asia (n=13) and one unspecified group (n=1) were excluded. Western Europe (n=3329) was left out because the region was considered as relatively similar to Norway and it was a preference to maintain the perspective having fewer regions in the analyses. After exclusions, the total number of participants was 132 927, originating from eleven different geographical regions.

Further exclusion was based on risk factors included in the risk score calculations. The sample was limited to participants in the age range 40-65 years old (n= 63 186 excluded) since this is the age group relevant for the NORRISK algorithm (Selmer et al. 2008) and within the age range of the Framingham sample(D'Agostino et al. 2008). Participants with missing triglyceride levels (n=94), triglyceride levels < 0, 2 mmol/L or triglyceride levels > 20 mmol/L (n=11) were excluded. One extreme total cholesterol value (48 mmol/L), missing blood pressure values (n=58) and missing HDL cholesterol values (n=17) were all further excluded giving the sample plausible values of the risk factors. Participants missing information on self-reported diabetes (n=948), use of blood pressure medication (n=482) and smoking status (5984) were also

excluded. One person who answered both yes and no on the question about daily smoking was excluded, giving a total sample of 62 145 participants.

From the main sample further exclusions resulted in two slightly different sample sizes according to the risk equations. Both Framingham and NORRISK are restricted to asymptomatic subjects consequently excluding people with history of MI, angina pectoris or stroke giving a sample of 60 015 individuals for the calculation of Framingham 10 year risk of CVD. For NORRISK people with diabetes, people who reported either current or previous use of antihypertensive and/or lipid-lowering treatment were also excluded. According to the European Guidelines on CVD prevention they were already declared to be in high risk (Graham et al. 2007). Thus, the total number of people in the NORRISK sample was 54 027.

Definition of risk factors

Hypertension was defined in individuals currently using blood pressure treatment and those having a systolic blood pressure $\geq$ 140 mmHg in accordance with the definitions of the World Health Organization (WHO) and International Society of Hypertension (Whitworth 2003). High total cholesterol was defined according to the European Society of Cardiology (ESC) Guidelines (Graham et al. 2007) recommending a total cholesterol level of less than 5 mmol/L although this is a relatively low threshold. Low HDL cholesterol levels were defined following the same guidelines suggesting low levels to be < 1 mmol/L in men and < 1.2 mmol/L in women. A high cholesterol/HDL ratio was defined as > 4. High WHR was defined as 1.0 or greater in men, and 0.85 or greater in women (WHO 2000) and high BMI as $\geq$25 using the WHO criteria (WHO 1995).

Statistical analyses and risk score calculation

Data were analysed using the PASW version 18. The different variables were reported as fractions for categorical variables and mean values including either standard deviation or 95% confidence intervals for continuous variables. Age-adjustment was performed by linear regression, and done separately for each single risk factor, calculated risk scores (calculation described below) and the prevalence estimates. Triglycerides and glucose were additionally adjusted for time since last meal. Differences in risk factors and calculated total risk between the ethnic groups were assessed using one-way analyses of variance. Post hoc tests with Norway as reference group were performed to study differences between total risk scores when overall

variance was found significant. All analyses were stratified by sex. P-values less than 0, 05 were considered statistically significant.

We calculated 10-year risk of general CVD events for each individual using a recent version of the Framingham risk equation based on age, gender, total cholesterol, HDL cholesterol, treated and untreated systolic blood pressure, smoking (yes/no) and diabetes (yes/no) (D'Agostino et al. 2008). The Framingham equation was derived based on a Caucasian American population free of CVD and in the age range 30-74 years old(Anderson et al. 1991). General CVD includes both fatal and non-fatal events such as coronary death, MI, coronary insufficiency, angina, ischaemic stroke, hemorrhagic stroke and transient ischaemic attack, peripheral artery disease and heart failure. Chosen threshold for dividing participants into the categories high or low risk was 10 %, although such thresholds can be defined differently depending on the acceptance of risk (Fretheim et al. 2002; Greenland et al. 2010)

10-year risk of CVD mortality was calculated using NORRISK, the Norwegian version of the European SCORE (Systematic COronary Risk Evaluation) algorithm (Selmer et al. 2008). NORRISK is recently calibrated to national mortality figures as recommended in the European guidelines (Graham et al. 2007) since SCORE was found to overestimate risk in the Norwegian population (Lindman et al. 2006; Lindman et al. 2007). NORRISK calculates total risk based on age, gender, systolic blood pressure, total cholesterol and smoking (yes/no) (Selmer et al. 2008). Defined thresholds for high risk using NORRISK differ according to age. People aged 40-49 years are recommended to have less than 1 % risk, people aged 50-59 are recommended less than 5 %, while people aged 60-69 should have less than 10 % total risk (Norheim et al. 2009). We chose, however, to have a cut-off of 5 % when dividing people into high/low risk in order to make comparing of ethnic groups more manageable. Also, the prevalence of people with high risk was estimated adjusted to the age of 47,7 years in men and 47,6 years in women - neither far from the age of 50 making the chosen cut-off more justifiable. 5 % is also the limit used for SCORE in the ESC Guidelines (Graham et al. 2007).

Ethics

All the participants gave their written informed consent (Næss et al. 2008) and the project is approved by the CONOR Steering Committee.

Results

The study included 32 684 women and 29 461 men aged 40-65 years from eleven different birth regions, representing 4 continents. In Table 2-4 we show summary statistics for main variables for the different ethnic groups.

We found significant differences in systolic blood pressure, serum glucose, WHR and BMI between ethnic groups (Table 2) (p <0,001 for overall equality). Men born in Norway and Former Yugoslavia had the highest mean values of systolic blood pressure, while among women the highest blood pressure was found in Sub-Saharan Africans. People from the Indian subcontinent had the highest levels of glucose and WHR, whereas North American immigrant women had the lowest levels of glucose and WHR. Both Former Yugoslavians and people from the Indian subcontinent showed relatively high levels of BMI, though not the highest.

Furthermore, significant differences in total cholesterol, HDL cholesterol, total cholesterol/HDL cholesterol ratio and triglycerides were observed (Table 3) (p < 0,001 of overall equality). Men and women from East Asia had the lowest mean of total cholesterol. Participants from the Indian subcontinent had the lowest adverse levels of HDL cholesterol along with men from Former Yugoslavia and North Africa. HDL cholesterol was naturally lower in men than in women. East Asian women had the lowest total cholesterol/HDL ratio while the highest was seen in Former Yugoslavians, both men and women. East Asia and North America was among the regions with lower levels of triglycerides and people from the Indian subcontinent had the highest triglyceride levels.

Years of education, prevalence of diabetes, smoking, inactivity, previous MI and angina, use of blood pressure and lipid lowering medication varied significantly between ethnic groups (Table 4) (p <0,001 for overall equality). Reported history of stroke, however, did not differ significantly in men or women (p=0,212 and p=0,074 respectively). Highest prevalence of diabetes was found among the group from the Indian subcontinent. There were large differences in smoking prevalence according to gender; generally more men than women smoked. In some groups this trend was especially evident such as among North Africans. European women seemed most inclined to smoking. Inactivity was relatively common, especially for participants from the Asian and African regions as well as Former Yugoslavia. In each ethnic group the prevalence of previous CVD events (MI, angina or stroke) was less than ten percent. Men from

the Indian subcontinent and North Africa both had the highest prevalence of MI in the male part of the population.

In Figure 1 we show the prevalence of people with high values of some of the risk factors. These include total cholesterol/HDL ratio, total cholesterol, blood pressure, WHR and BMI. All differed significantly between ethnic groups (p < 0,001 for overall equality and p=0,031 for hypertension in women,). The highest prevalence of hypertension corresponded with the highest values of mean systolic blood pressure, but additionally revealed high prevalence of hypertension among people from the Indian subcontinent. Prevalence of high WHR was also highest in men and women from this region corresponding with the highest mean values (Fig.1.). Lower prevalence of overweight measures corresponded with low mean values of WHR and BMI in East Asians.

Framingham

In Table 5 we show calculated 10-year risk of general CVD based on the Framingham equation for the different ethnic groups. Statistical differences were observed in both sexes, and men scored generally higher than women (p<0, 01 for overall equality). Immigrants from Former Yugoslavian countries had the highest mean scores (men and women) although not statistically different. No ethnic group had significantly higher scores than Norwegians. Immigrants from the Indian subcontinent had the third highest risk scores in both sexes. When examining the proportions of people defined in high risk of general CVD (Fig.2.a.), some regions showed slightly higher percentages than men from Former Yugoslavia. Several birth regions had significantly lower risk scores compared to people born in Norway. East Asians had the lowest predicted risk scores (men and women) along with women from South East Asia, with the lowest score among women (p<0, 05, compared to Norway).

NORRISK

Based on calculated risk of CVD mortality using NORRISK, the mean risk values between the ethnic groups only differed significantly in the male part of the population (Table 6) (p<0,001 for overall equality). Here, three regions; the Middle East, South East Asia and North Africa, differed significantly from Norway having lower scores (p<0,001, p=0,001 and p=0,043,

respectively). North American men had the highest score and North African men the lowest, but these two did not differ significantly from each other (confidence intervals not overlapping). We did not observe any differences among women, but the confidence intervals for Norwegian and Middle East women did not overlap.

Discussion

The results in this cross sectional study revealed significant variations in risk of CVD among ethnic groups living in Norway. We observed significant differences in almost all the variables. Differences in total risk were identified between Norway and other birth regions. Ethnic groups with markedly higher levels of risk factors and total risk were in particular the Indian subcontinent and Former Yugoslavia showing tendencies evident in both sexes.

In general, risk scores were higher using the Framingham equation than NORRISK. This was expected since Framingham has a wider range of predicted outcomes than NORRISK. The equation also includes additional risk factors compared to NORRISK, which also had a different sample than Framingham, excluding people in risk with diabetes and those being treated for hypertension and hyperlipidemia. This had an attenuating effect on the scores making it more difficult to get a clear picture of ethnic differences when using NORRISK.

Some countries of the East Asian region (Japan and China) have previously been found to have lower risk of CVD (Menotti et al. 1993; Yusuf, Salim et al. 2001). This concur with our results showing comparative lower levels of overweight and lipids, further reflected in lower calculated risk according to the Framingham equation.

North America, despite its known increasing epidemic of obesity, especially in the United States (U.S.) (Mokdad et al. 2001), was comparably low in total cholesterol/HDL ratio, triglycerides, diabetes and WHR and BMI. Possible explanations for their favorable levels might be the inclusion of Canada since Canadians in general have not yet reached the same levels in overweight as their U.S. neighbors (Katzmarzyk & Mason 2006). An additional explanation is "the healthy migrant effect", suggesting that these immigrants constitute a selectively healthy group from their countries of origin (Fennelly 2007; Lu 2008). North Americans in our study represent a highly educated part of their countries. North American immigrants had the highest mean value in years of education, which is an important social determinant of health in North

American countries (Braveman et al. 2011; Choiniere et al. 2000). However, men from North America had the highest mean score in 10-year risk of CVD mortality.

People of African descent have been reported to have higher values of blood pressure than other ethnic groups (Chaturvedi et al. 1993), which was confirmed in the female part of this study sample.

Previous findings from both national (Glenday et al. 2006; Kumar et al. 2009) and international studies (Chambers et al. 2001; Hayes et al. 2002; Kain & Catto 2002; Mather & Keen 1985; McKeigue et al. 1988; Tillin et al. 2005) are in accordance with our findings suggesting a higher risk of insulin resistance and other metabolic risk factors in immigrants from the Indian subcontinent. This group had high scores in total risk of general CVD, high prevalence of diabetes and inactivity, elevated mean levels of blood glucose, general overweight (BMI) (especially in women), abdominal fat (WHR), triglycerides and a high total cholesterol/HDL ratio due to low HDL cholesterol levels. People from the Indian subcontinent have also been reported to have increased risk of IHD based on mortality numbers in the UK (Balarajan 1991; Wild & Mckeigue 1997). Such statements cannot be made here, but our findings indicate general accordance. Men from the Indian Subcontinent had among the highest prevalence of MI, although the prevalence was generally low in all groups. Previous studies (McKeigue 1992; McKeigue et al. 1988) have not been able to explain the excess risk of CHD in people from the Indian subcontinent as caused by smoking or hypertension. Relatively low smoking prevalence and intermediate mean values of systolic blood pressure in this study support this. However, an examination of proportions with hypertension shows high prevalence in the Indian subcontinent relative to other groups. In search for explanations of the excess risk in this group, researchers have also directed attention to people who have not migrated, but still live in the Indian subcontinent. Studies comparing immigrants in the UK with their counterparts in India suggest that migration to higher income countries might result in an increased risk of CVD (Patel et al. 1995; Patel et al. 2006). The prevalence of diabetes is also 2-3 fold higher in the Indian subcontinent than reported in Western countries (Jafar 2006). A hypothesis called the "thrifty phenotype hypothesis" has been proposed based on observations where small birth weight and maternal malnutrition is associated with increased risk of chronic diseases in later life (Hales & Barker 2001). This might serve as an explanation for high levels of CVD risk factors among immigrants from the Indian subcontinent, emphasizing environmental influences in early life as part of the pathogenesis (Stein et al. 1996; Yajnik 2001). In countries of the Indian subcontinent

as in other developing countries undergoing epidemiologic transition, the prevalence of diabetes is also increasing as a consequence of urbanization, correspondingly finding higher prevalence of diabetes in urban areas than in rural areas (Goyal & Yusuf 2006; Mohan 2004; Singh et al. 1997).

To our knowledge, no previous study has examined the risk of CVD in Former Yugoslavian immigrants in Norway. However, a study from Switzerland (Grossmann et al. 2010) found Former Yugoslavian immigrants more likely to be overweight than the Swiss native born. This is supported in our study where both men and women from Former Yugoslavia had significantly higher levels of overweight measures than several other groups (according to the 95 % CI) and ranked high in prevalence of both general overweight and abdominal fat. This could be due to nutrition since people from Former Yugoslavia are known to have a rich diet with high use of cooking oil, and prevalence of overweight was also reported as high even before the Balkan war (Kruseman et al. 2003). Participants from former Yugoslavia had the highest calculated 10-year risks of CVD according to the Framingham equation particularly reflected in their elevated total cholesterol/HDL ratio. The fact that Former Yugoslavians ranked highest in this score exemplifies the importance of estimating total risk when assessing ethnic differences, since this group also showed intermediate levels in some risk factors. This group did not score higher than Norwegians significantly, probably a consequence of small groups sizes in this region compared to Norway and that Norwegians also had high scores. Reason for migration of the immigrants in this study is not known, nevertheless, immigrants from Former Yugoslavian countries might represent post war-refugees struggling with post-war effects. This has been reported among Former Yugoslavian immigrants in other countries (Markovic et al. 2002; van den Heuvel 1998). Post-traumatic stress disorder is one of the possible consequences associated with CVD (Boscarino & Chang 1999; Kibler et al. 2009)

This study has several strengths such as the possibility of comparing different ethnic groups living in both rural and urban parts of Norway. Secondly, we have a large sample size resulting in comparable subgroups, although the statistical strength was still somewhat limited due to group sizes after exclusion.

Limitations include heterogeneity due to the application of large birth regions as indicators of ethnic belonging. South Asians is known to be a heterogeneous group regarding CVD risk factors (Bhopal et al. 1999) which might as well apply for the other regions. This demands cautious interpretation of the findings. It is also noted that the national studies mentioned cannot

be referred to as entirely different studies since present study might include duplicate participant data. About 1151 participants from our CONOR sample participated in the Oslo Immigrant Health study which was used in the Norwegian studies mentioned earlier. Another limitation is the relatively low participation rate which increases the risk of selection bias.

Conclusion

This study adds information on CVD risk factors among immigrant groups living in Norway that has not been documented previously. Immigrants from Former Yugoslavia and the Indian subcontinent show higher risk in both sexes, reflected both in single risk factors and total risk scores. Culturally adapted lifestyle interventions would be recommended for most of the groups since many reported unhealthy lifestyles. Although this study cannot explain the differences between ethnic groups to the fully or the etiology of such, it might function as a first step towards a better understanding. More research is needed in order to improve the adaption of primary prevention efforts according to the different ethnic groups in Norway. Examining risk in smaller ethnic groups within our large regions would be of particular interest, before possible explanations for the inequalities can be examined, identified and further addressed.

References

Aamodt, G., Søgaard, A., Næss, Ø., Beckstrøm, A. & Samuelsen, S. (2010). CONOR-databasen–et lite stykke Norge. *Tidsskrift for Den norske legeforening*, 3 (130): 264-265.

Anderson, K. M., Odell, P. M., Wilson, P. W. F. & Kannel, W. B. (1991). Cardiovascular disease risk profiles. *American heart journal*, 121 (1): 293-298.

Balarajan, R. (1991). Ethnic differences in mortality from ischaemic heart disease and cerebrovascular disease in England and Wales. *British Medical Journal*, 302 (6776): 560-564.

Bhopal, R., Unwin, N., White, M., Yallop, J., Walker, L., Alberti, K., Harland, J., Patel, S., Ahmad, N. & Turner, C. (1999). Heterogeneity of coronary heart disease risk factors in Indian, Pakistani, Bangladeshi, and European origin populations: cross sectional study. *British Medical Journal*, 319 (7204): 215-220.

Bhopal, R. (2000). What is the risk of coronary heart disease in South Asians? A review of UK research. *Journal of Public Health*, 22 (3): 375-385.

Bhopal, R. (2004). Glossary of terms relating to ethnicity and race: for reflection and debate. *Journal of Epidemiology and Community Health*, 58 (6): 441-445.

Boscarino, J. A. & Chang, J. (1999). Electrocardiogram abnormalities among men with stress-related psychiatric disorders: implications for coronary heart disease and clinical research. *Annals of Behavioral Medicine*, 21 (3): 227-234.

Braveman, P. A., Egerter, S. A. & Mockenhaupt, R. E. (2011). Broadening the Focus: The Need to Address the Social Determinants of Health. *American Journal of Preventive Medicine*, 40 (1, Supplement 1): S4-S18.

Chambers, J. C., Eda, S., Bassett, P., Karim, Y., Thompson, S. G., Gallimore, J. R., Pepys, M. B. & Kooner, J. S. (2001). C-reactive protein, insulin resistance, central obesity, and coronary heart disease risk in Indian Asians from the United Kingdom compared with European whites. *Circulation*, 104 (2): 145-150.

Chaturvedi, N., McKeigue, P. & Marmot, M. (1993). Resting and ambulatory blood pressure differences in Afro-Caribbeans and Europeans. *Hypertension*, 22 (1): 90-96.

Chaturvedi, N. (2003). Ethnic Differences in Cardiovascular Disease. *Heart*, 89 (6): 681-686.

Choiniere, R., Lafontaine, P. & Edwards, A. C. (2000). Distribution of cardiovascular disease risk factors by socioeconomic status among Canadian adults. *Canadian Medical Association Journal*, 162 (9 Suppl): S13-24.

D'Agostino, R. B., Sr., Vasan, R. S., Pencina, M. J., Wolf, P. A., Cobain, M., Massaro, J. M. & Kannel, W. B. (2008). General cardiovascular risk profile for use in primary care: the Framingham Heart Study. *Circulation*, 117 (6): 743-53.

Fennelly, K. (2007). The" healthy migrant" effect. *Minnesota medicine*, 90 (3): 51.

Forouhi, N. G. & Sattar, N. (2006). CVD risk factors and ethnicity - A homogeneous relationship? *Atherosclerosis Supplements*, 7 (1): 11-19.

Fretheim, A., Bjørndal, A., Oxman, A. D., Dyrdal, A., Golding, M., Ose, L., Reikvam, Å. & Teisberg, P. (2002). Diagnostikk og behandling. Retningslinjer for medikamentell primærforebygging av hjerte-og karsykdommer–hvem bør behandles? *Tidsskrift for Den norske lægeforening*, 122: 2277-81.

Glenday, K., Kumar, B., Tverdal, A. & Meyer, H. (2006). Cardiovascular disease risk factors among five major ethnic groups in Oslo, Norway: the Oslo Immigrant Health Study. *European Journal of Cardiovascular Prevention & Rehabilitation*, 13 (3): 348-355.

Goyal, A. & Yusuf, S. (2006). The burden of cardiovascular disease in the Indian subcontinent. *Indian Journal of Medical Research*, 124 (3): 235-244.

Graham, I., Atar, D., Borch-Johnsen, K., Boysen, G., Burell, G., Cifkova, R., Dallongeville, J., De Backer, G., Ebrahim, S. & Gjelsvik, B. (2007). European guidelines on cardiovascular disease prevention in clinical practice: executive summary. *European Heart Journal*, 28 (19): 2375–2414.

Greenland, P., Alpert, J. S., Beller, G. A., Benjamin, E. J., Budoff, M. J., Fayad, Z. A., Foster, E., Hlatky, M. A., Hodgson, J. M., Kushner, F. G., et al. (2010). 2010 ACCF/AHA Guideline for Assessment of Cardiovascular Risk in Asymptomatic Adults: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation*, 122 (25): e584-636.

Grossmann, F. F., Leventhal, M. E., Auer-Böer, B., Wanner, P. & Bischoff, A. (2010). Self-Reported Cardiovascular Risk Factors in Immigrants and Swiss Nationals. *Public Health Nursing*: 1-11.

Hales, C. N. & Barker, D. J. P. (2001). The thrifty phenotype hypothesis. *British Medical Bulletin*, 60 (1): 5-20.

Hayes, L., White, M., Unwin, N., Bhopal, R., Fischbacher, C., Harland, J. & Alberti, K. (2002). Patterns of physical activity and relationship with risk markers for cardiovascular disease and diabetes in Indian, Pakistani, Bangladeshi and European adults in a UK population. *Journal of Public Health*, 24 (3): 170-178.

Jafar, T. H. (2006). The growing burden of chronic kidney disease in Pakistan. *New England Journal of Medicine*, 354 (10): 995-997.

Kain, K. & Catto, A. J. (2002). Insulin resistance syndrome in South Asians. *The British Journal of Diabetes & Vascular Disease*, 2 (5): 398-402.

Katzmarzyk, P. T. & Mason, C. (2006). Prevalence of class I, II and III obesity in Canada. *Canadian Medical Association Journal*, 174 (2): 156-157.

Kibler, J. L., Joshi, K. & Ma, M. (2009). Hypertension in Relation to Posttraumatic Stress Disorder and Depression in the US National Comorbidity Survey. *Behavioral Medicine*, 34 (4): 125-131.

Kruseman, M., Stoll, B. E. & Stalder, H. (2003). Interactive group education for refugees from the Former Yugoslavia to reduce their oil consumption. *Patient Education and Counseling*, 49 (2): 171-176.

Kumar, B. N., Selmer, R., Lindman, A. S., Tverdal, A., Falster, K. & Meyer, H. E. (2009). Ethnic differences in SCORE cardiovascular risk in Oslo, Norway. *European Journal of Cardiovascular Prevention and Rehabilitation*, 16 (2): 229-34.

Kurian, A. K. & Cardarelli, K. M. (2007). Racial and ethnic differences in cardiovascular disease risk factors: a systematic review. *Ethnicity and Disease*, 17 (1): 143.

Lee, J., Heng, D., Chia, K. S., Chew, S. K., Tan, B. Y. & Hughes, K. (2001). Risk factors and incident coronary heart disease in Chinese, Malay and Asian Indian males: the Singapore Cardiovascular Cohort Study. *International Journal of Epidemiology*, 30 (5): 983-988.

Lindman, A. S., Selmer, R., Tverdal, A., Pedersen, J. I., Eggen, A. E. & Veierød, M. B. (2006). The SCORE risk model applied to recent population surveys in Norway compared to observed mortality in the general population. *European Journal of Cardiovascular Prevention & Rehabilitation*, 13 (5): 731-737.

Lindman, A. S., Veierød, M. B., Pedersen, J. I., Tverdal, A., Njølstad, I. & Selmer, R. (2007). The ability of the SCORE high-risk model to predict 10-year cardiovascular disease mortality in Norway. *European Journal of Cardiovascular Prevention & Rehabilitation*, 14 (4): 501-507.

Lu, Y. (2008). Test of the 'healthy migrant hypothesis': A longitudinal analysis of health selectivity of internal migration in Indonesia. *Social Science & Medicine*, 67 (8): 1331-1339.

Markovic, M., Manderson, L. & Kelaher, M. (2002). The health of immigrant women: Queensland women from the former Yugoslavia. *Journal of Immigrant Health*, 4 (1): 5-15.

Mather, H. M. & Keen, H. (1985). The Southall Diabetes Survey: prevalence of known diabetes in Asians and Europeans. *British Medical Journal (Clinical research ed.)*, 291 (6502): 1081-1084.

McKeigue, P., Miller, G. & Marmot, M. (1989). Coronary heart disease in south Asians overseas: a review. *Journal of Clinical Epidemiology*, 42 (7): 597-609.

McKeigue, P. (1992). Coronary heart disease in Indians, Pakistanis, and Bangladeshis: aetiology and possibilities for prevention. *British heart journal*, 67 (5): 341-342.

McKeigue, P. M., Marmot, M. G., Syndercombe Court, Y. D., Cottier, D. E., Rahman, S. & Riemersma, R. A. (1988). Diabetes, hyperinsulinaemia, and coronary risk factors in Bangladeshis in east London. *British heart journal*, 60 (5): 390-396.

Menotti, A., Keys, A., Kromhout, D., Blackburn, H., Aravanis, C., Bloemberg, B., Buzina, R., Dontas, A., Fidanza, F. & Giampaoli, S. (1993). Inter-cohort differences in coronary heart disease mortality in the 25-year follow-up of the seven countries study. *European Journal of Epidemiology*, 9 (5): 527-536.

Mohan, V. (2004). Why are Indians more prone to diabetes? *The Journal of the Association of Physicians of India*, 52: 468-74.

Mokdad, A. H., Bowman, B. A., Ford, E. S., Vinicor, F., Marks, J. S. & Koplan, J. P. (2001). The Continuing Epidemics of Obesity and Diabetes in the United States. *The Journal of the American Medical Association*, 286 (10): 1195-1200.

Norheim, O. F., Gjelsvik, B., Kjeldsen, S. E., Klemsdal, T. O., Madsen, S., Meland, E., Narvesen, S., Negård, A., Njølstad, I., Tonstad, S., et al. (2009). Nasjonale retningslinjer for individuell primærforebygging av hjerte- og karsykdommer. *Norwegian Directorate of Health*, IS-1550.

Næss, Ø., Søgaard, A. J., Arnesen, E., Beckstrøm, A. C., Bjertness, E., Engeland, A., Hjort, P. F., Holmen, J., Magnus, P., Njølstad, I., et al. (2008). Cohort Profile: Cohort of Norway (CONOR). *International Journal of Epidemiology*, 37 (3): 481-485.

Patel, D., Winterbotham, M., Britt, R., Sutton, G., Bhatnagar, D., Mackness, M. I., Creed, F., Tomenson, B., Durrington, P. & Anand, I. (1995). Coronary risk factors in people from the Indian subcontinent living in West London and their siblings in India. *The Lancet*, 345 (8947): 405-409.

Patel, J. V., Vyas, A., Cruickshank, J. K., Prabhakaran, D., Hughes, E., Reddy, K. S., Mackness, M. I., Bhatnagar, D. & Durrington, P. N. (2006). Impact of migration on coronary heart disease risk factors: Comparison of Gujaratis in Britain and their contemporaries in villages of origin in India. *Atherosclerosis*, 185 (2): 297-306.

Ranganathan, M. & Bhopal, R. (2006). Exclusion and inclusion of nonwhite ethnic minority groups in 72 North American and European cardiovascular cohort studies. *The Public Library of Science Medicine*, 3 (3): 0329-0336.

Selmer, R., Lindman, A., Tverdal, A., Pedersen, J., Njølstad, I. & Veierød, M. (2008). Modell for estimering av kardiovaskulær risiko i Norge. *Tidsskrift for Den norske legeforening*, 128 (3): 286-290.

Singh, R. B., Sharma, J. P., Rastogi, V., Raghuvanshi, R. S., Moshiri, M., Verma, S. P. & Janus, E. D. (1997). Prevalence of coronary artery disease and coronary risk factors in rural and urban populations of north India. *European Heart Journal*, 18 (11): 1728-1735.

Statistics Norway. (2010). *Cardiovascular diseases and cancer main causes of death. Causes of deaths, 2008*. Available at: http://www.ssb.no/english/subjects/03/01/10/dodsarsak_en/arkiv/ (accessed: 10.03.2011).

Stein, C., Fall, C., Kumaran, K., Osmond, C., Cox, V. & Barker, D. (1996). Fetal growth and coronary heart disease in South India. *The Lancet*, 348 (9037): 1269-1273.

Tillin, T., Forouhi, N., Johnston, D., McKeigue, P., Chaturvedi, N. & Godsland, I. (2005). Metabolic syndrome and coronary heart disease in South Asians, African-Caribbeans and white Europeans: a UK population-based cross-sectional study. *Diabetologia*, 48 (4): 649-656.

van den Heuvel, W. J. A. (1998). Health status of refugees from former Yugoslavia: descriptive study of the refugees in the Netherlands. *Health*, 39 (3).

Whitworth, J. (2003). 2003 World Health Organization (WHO)/International Society of Hypertension (ISH) statement on management of hypertension. *Journal of hypertension*, 21 (11): 1983-1992.

WHO. (1995). *Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee*. WHO Technical Report Series: 854. Geneva: World Health Organization.

WHO. (2007). *Prevention of Cardiovascular Disease: Guidelines for assessment and management of cardiovascular risk*. Geneva: World Health Organization.

WHO. (2000). *Obesity: preventing and managing the global epidemic. Report of a WHO Consultation*. WHO Technical Report Series; 894. Geneva: World Health Organization.

Wild, S. & Mckeigue, P. (1997). Cross sectional analysis of mortality by country of birth in England and Wales, 1970-92. *British Medical Journal*, 314 (7082): 705.

Yajnik, C. S. (2001). The Insulin Resistance Epidemic in India: Fetal Origins, Later Lifestyle, or Both? *Nutrition Reviews*, 59 (1): 1-9.

Yusuf, S., Reddy, S., Ounpuu, S. & Anand, S. (2001). Global burden of cardiovascular diseases: part I: general considerations, the epidemiologic transition, risk factors, and impact of urbanization. *Circulation*, 104 (22): 2746.

Yusuf, S., Reddy, S., Ounpuu, S. & Anand, S. (2001). Global Burden of Cardiovascular Diseases: Part II: Variations in Cardiovascular Disease by Specific Ethnic Groups and Geographic Regions and Prevention Strategies. *Circulation*, 104 (23): 2855-2864.

Tables and figures

Table 1: An overview of the countries included in each birth region.

Birth region:	Countries:
Eastern Europe, n=220 (72 % women)	Bulgaria, Estonia, Belarus, Latvia, Poland, Romania, the Soviet-Union, Lithuania, Moldova, Russia, Czechoslovakia, the Ukraine, DDR (code no longer in use), Hungary, Slovakia, the Czech Republic
Former Yugoslavia, n=179 (47 % women)	Albania, Croatia, Yugoslavia, Slovenia, Bosnia-Herzegovina, Macedonia
North Africa, n=71 (40,8 % women)	Algeria, Egypt, Libya, Morocco, Sudan, Tunisia
Sub-Saharan-Africa, n=183 (39 % women)	Angola, Botswana, Equatorial-Guinea, the Ivory Coast/Côte d'Ivoire, Eritrea, Ethiopia, Dijbouti, Gambia, Ghana, Guinea, Guinea-Bissau, Cameroon, Cape Verde, Kenya, Congo, Zaire, Liberia, Madagascar, Mauretania, Mauritius, Namibia, Nigeria, Mozambique, Zimbabwe, Rwanda, Senegal, Sierra Leone, Somalia, South Africa, Tanzania, Togo, Uganda, Zambia
Middle East, n=669 (40 % women)	Turkey, Armenia, Azerbaijan, Iraq, Iran, Jordan, Kuwait, Lebanon, Saudi Arabia, Syria, Yemen
Indian Subcontinent, n=1071 (41 % women)	Bangladesh, Myanmar (Burma), Sri Lanka, India, Nepal, Pakistan
East Asia, n=104 (58 % women)	Taiwan, Hong Kong, Japan, China, North Korea, South Korea
North America, n=180 (60 % women)	Canada, USA
South America, n=159 (55 % women)	Argentina, Bolivia, Brazil, Guyana, Chile, Colombia, Ecuador, Paraguay, Peru, Suriname, Uruguay, Venezuela
South-East Asia, n=611 (63 % women)	The Philippines, Indonesia, Kampuchea, Laos, Malaysia, Singapore, Thailand, Vietnam

n, number of participants in the main sample of a total of 62 145 participants.

Table 2: Characteristics of 62 145 participants, mean values (95 % CI), adjusted for age. Data from Cohort of Norway, 1994-2003.

	N	Age, years (SD)	Systolic blood pressure, mmHg		Serum glucose[a], mmol/L		WHR		BMI	
MEN										
Norway	27 699	48,6 *(6,9)*	135,0	*(134,8-135,1)*	5,46	*(5,44-5,48)*	0,908	*(0,907-0,909)*	26,5	*(26,5-26,6)*
Eastern Europe	62	51,1 *(8,2)*	131,7	*(127,8-135,5)*	5,34	*(4,97-5,72)*	0,912	*(0,897-0,927)*	26,7	*(25,8-27,6)*
Former Yugoslavia	94	45,5 *(6,2)*	132,8	*(129,7-136,0)*	5,72	*(5,42-6,02)*	0,920	*(0,908-0,933)*	27,8	*(27,1-28,5)*
North Africa	42	47,1 *(7,6)*	121,7	*(117,0-126,4)*	6,11	*(5,66-6,57)*	0,912	*(0,894-0,930)*	26,8	*(25,8-27,9)*
Sub-Saharan Africa	112	45,2 *(5,7)*	131,5	*(128,7-134,4)*	5,42	*(5,14-5,70)*	0,887	*(0,875-0,898)*	25,6	*(24,9-26,2)*
Middle East	403	46,1 *(5,9)*	126,8	*(125,2-128,3)*	5,81	*(5,67-5,96)*	0,912	*(0,906-0,918)*	27,1	*(26,8-27,5)*
Indian subcontinent	635	46,7 *(6,3)*	129,7	*(128,5-130,9)*	6,42	*(6,31-6,54)*	0,947	*(0,942-0,951)*	26,7	*(26,5-27,0)*
East Asia	44	47,8 *(7,1)*	126,0	*(121,4-130,6)*	5,84	*(5,40-6,28)*	0,889	*(0,871-0,907)*	23,8	*(22,8-24,8)*
North America	72	47,4 *(7,0)*	131,3	*(127,7-134,9)*	5,51	*(5,16-5,86)*	0,909	*(0,894-0,923)*	26,4	*(25,6-27,2)*
South America	72	45,2 *(4,6)*	127,3	*(123,7-130,9)*	5,58	*(5,50-6,20)*	0,928	*(0,914-0,942)*	27,9	*(27,1-28,7)*
South-East Asia	226	47,1 *(6,0)*	127,0	*(124,9-129,0)*	5,9	*(5,71-6,10)*	0,884	*(0,876-0,891)*	24,4	*(23,9-24,9)*
WOMEN										
Norway	30 999	48,4 *(6,9)*	128,5	*(128,3-128,7)*	5,24	*(5,23-5,26)*	0,797	*(0,797-0,798)*	25,6	*(25,5-25,6)*
Eastern Europe	158	46,8 *(6,6)*	124,9	*(122,2-127,5)*	5,23	*(5,03-5,43)*	0,780	*(0,769-0,790)*	25,6	*(24,9-26,2)*
Former Yugoslavia	85	46,1 *(6,3)*	128,4	*(124,8-132,0)*	5,79	*(5,53-6,05)*	0,806	*(0,793-0,820)*	28,5	*(27,6-29,5)*
North Africa	29	44,9 *(5,1)*	123,8	*(117,6-130,0)*	5,72	*(5,28-6,16)*	0,850	*(0,826-0,873)*	30,7	*(29,1-32,3)*
Sub-Saharan Africa	71	44,5 *(4,6)*	131,0	*(127,0-134,9)*	5,82	*(5,53-6,10)*	0,814	*(0,798-0,829)*	28,4	*(27,4-29,4)*
Middle East	266	46,6 *(5,9)*	122,6	*(120,6-124,7)*	5,84	*(5,69-5,99)*	0,825	*(0,817-0,833)*	29,2	*(28,6-29,7)*
Indian subcontinent	436	46,7 *(5,9)*	126,3	*(124,7-127,9)*	6,11	*(6,00-6,23)*	0,864	*(0,858-0,870)*	28,8	*(28,4-29,2)*
East Asia	60	46,9 *(7,4)*	122,2	*(117,9-126,5)*	5,97	*(5,66-6,29)*	0,786	*(0,769-0,802)*	23,5	*(22,3-24,6)*
North America	108	45,8 *(5,6)*	125,5	*(122,3-128,7)*	5,08	*(4,84-5,31)*	0,773	*(0,761-0,786)*	25,6	*(24,8-26,4)*
South America	87	45,0 *(4,9)*	120,9	*(117,3-124,4)*	5,30	*(5,04-5,56)*	0,819	*(0,805-0,833)*	26,6	*(25,7-27,5)*
South-East Asia	385	46,6 *(5,6)*	126,0	*(124,3-127,7)*	5,61	*(5,48-5,73)*	0,811	*(0,804-0,817)*	24,3	*(23,9-24,7)*

SD, standard deviation; CI, confidence interval; WHR, waist hip ratio; BMI, body mass index.   [a]Adjusted for time since last meal. All variables had P-values < 0,001 for overall equality.

Table 3: Blood lipids, mean values mmol/L for 62 145 participants (95 % CI), adjusted for age. Data from Cohort of Norway, 1994-2003.

	N	Total cholesterol		HDL cholesterol		Total cholesterol/HDL ratio		Triglycerides[a]	
MEN									
Norway	27 699	5,95	(5,94-5,95)	1,24	(1,24-1,25)	5,11	(5,09-5,13)	2,03	(2,01-2,04)
Eastern Europe	62	5,65	(5,38-5,92)	1,23	(1,15-1,32)	4,94	(4,54-5,34)	2,27	(1,94-2,60)
Former Yugoslavia	94	5,59	(5,37-5,80)	1,08	(1,02-1,15)	5,46	(5,14-5,79)	2,32	(2,06-2,59)
North Africa	42	5,34	(5,01-5,66)	1,09	(0,99-1,19)	5,14	(4,65-5,62)	2,08	(1,68-2,48)
Sub-Saharan Africa	112	5,42	(5,22-5,62)	1,37	(1,30-1,43)	4,19	(3,89-4,49)	1,65	(1,40-1,90)
Middle-East	403	5,51	(5,41-5,62)	1,13	(1,10-1,17)	5,16	(5,00-5,32)	2,38	(2,25-2,50)
Indian subcontinent	635	5,60	(5,52-5,68)	1,08	(1,05-1,11)	5,45	(5,33-5,58)	2,69	(2,59-2,80)
East Asia	44	5,34	(5,02-5,66)	1,33	(1,23-1,43)	4,25	(3,78-4,73)	1,84	(1,45-2,23)
North America	72	5,83	(5,59-6,08)	1,32	(1,24-1,40)	4,85	(4,48-5,22)	1,78	(1,47-2,08)
South America	72	5,81	(5,56-6,05)	1,15	(1,07-1,22)	5,28	(4,91-5,65)	2,31	(2,00-2,61)
South-East Asia	226	5,63	(5,49-5,77)	1,23	(1,18-1,27)	4,82	(4,61-5,03)	2,62	(2,45-2,79)
WOMEN									
Norway	30 999	5,82	(5,81-5,83)	1,52	(1,51-1,52)	4,08	(4,07-4,10)	1,43	(1,42-1,44)
Eastern Europe	158	5,56	(5,39-5,72)	1,62	(1,56-1,68)	3,66	(3,45-3,88)	1,36	(1,22-1,50)
Former Yugoslavia	85	5,86	(5,64-6,09)	1,38	(1,29-1,46)	4,55	(4,26-4,85)	1,87	(1,68-2,06)
North Africa	29	5,49	(5,10-5,87)	1,32	(1,18-1,47)	4,27	(3,77-4,77)	1,61	(1,28-1,94)
Sub-Saharan Africa	71	5,50	(5,25-5,74)	1,57	(1,48-1,67)	3,62	(3,30-3,94)	1,28	(1,07-1,49)
Middle East	266	5,36	(5,24-5,49)	1,35	(1,31-1,40)	4,14	(3,97-4,30)	1,75	(1,64-1,86)
Indian subcontinent	436	5,41	(5,31-5,51)	1,26	(1,23-1,30)	4,49	(4,36-4,62)	2,11	(2,03-2,20)
East Asia	60	5,18	(4,91-5,44)	1,59	(1,49-1,69)	3,44	(3,09-3,79)	1,33	(1,10-1,56)
North America	108	5,44	(5,24-5,64)	1,57	(1,49-1,64)	3,68	(3,42-3,94)	1,34	(1,17-1,51)
South America	87	5,68	(5,46-5,90)	1,42	(1,34-1,51)	4,18	(3,89-4,47)	1,59	(1,40-1,78)
South-East Asia	385	5,52	(5,42-5,63)	1,49	(1,45-1,53)	3,92	(3,78-4,06)	1,67	(1,58-1,76)

CI, confidence interval; HDL, high density lipoprotein.   [a]Adjusted for time since last meal. All variables had P-values < 0,001 for overall equality.

Table 4: Prevalence (%) and estimated mean of self-reported variables in 62 145 participants, adjusted for age. Data from Cohort of Norway, 1994-2003.

	N	Diabetes	Daily smoking	Being inactive	History of MI	History of angina pectoris	History of stroke	Using BP medicine	Using lipid medicine	Years of education (SD)
MEN										
Norway	27 699	2	35	19	2	3	1	8	7	12,1 *(3,33)*
Eastern Europe	62	3	44	17	5	9	0	6	8	14,9 *(3,39)*
Former Yugoslavia	94	3	45	36	2	4	0	13	10	12,5 *(3,39)*
North Africa	42	10	55	38	6	8	5	13	10	11,6 *(3,37)*
Sub-Saharan Africa	112	4	32	30	3	8	0	9	7	14,6 *(3,39)*
Middle East	403	6	56	35	5	8	1	10	11	12,7 *(3,41)*
Indian subcontinent	635	14	28	44	6	8	2	15	13	12,3 *(3,53)*
East Asia	44	5	43	50	3	7	2	14	6	12,3 *(3,65)*
North America	72	3	29	20	0	3	0	6	4	16,1 *(3,31)*
South America	72	5	48	33	1	1	2	7	6	13,2 *(3,31)*
South-East Asia	226	7	56	44	4	7	1	12	8	11,3 *(3,46)*
WOMEN										
Norway	30 999	1	39	15	1	1	1	7	5	11,8 *(3,52)*
Eastern-Europe	158	3	35	21	1	4	1	8	2	14,6 *(3,27)*
Former Yugoslavia	85	4	38	39	4	4	4	10	8	10,6 *(3,32)*
North Africa	29	7	2	38	0	1	0	20	13	7,5 *(3,55)*
Sub-Saharan Africa	71	5	5	42	0	1	2	10	3	11,9 *(3,62)*
Middle East	266	8	25	41	0	4	1	14	9	10,2 *(3,26)*
Indian subcontinent	436	14	2	42	1	3	2	15	11	10,1 *(3,34)*
East Asia	60	4	21	39	0	4	0	11	6	12,7 *(3,41)*
North America	108	2	27	16	1	1	1	5	6	15,3 *(3,22)*
South America	87	3	28	24	0	3	1	8	8	12,9 *(3,26)*
South-East Asia	385	5	5	39	3	5	1	10	3	10,6 *(3,34)*

SD, standard deviation; MI, myocardial infarction; BP, blood pressure. All variables had P-values < 0,001 for overall equality except for stroke in men (p= 0,212) and in women (p=0,074).

Table 5: Framingham 10-year risk of CVD event. Data from Cohort of Norway, 1994-2003.

	Men				Women			
Calculated 10-year risk % in 60 015 participants [a] (95% CI), adjusted for age.							
	N	Mean	95 % CI	P (equality of immigrants)	N	Mean	95 % CI	P (equality of immigrants)
Norway	26 400	14,16	14,06-14,25	Reference	30 384	7,05	7,00-7,11	Reference
Eastern Europe	54	13,81	11,71-15,91	0,743	152	5,99	5,17-6,81	0,012*
Former Yugoslavia	90	15,14	13,52-16,77	0,237	78	8,00	6,86-9,15	0,105
North Africa	37	14,76	12,22-17,29	0,644	29	6,29	4,41-8,17	0,429
Sub-Saharan Africa	104	11,95	10,44-13,46	0,004**	70	6,26	5,05-7,47	0,201
Middle East	366	14,72	13,91-15,53	0,175	256	6,09	5,46-6,73	0,003**
Indian Subcontinent	574	14,74	14,10-15,38	0,080	420	6,65	6,16-7,14	0,113
East Asia	41	11,42	9,02-13,83	0,026*	58	5,62	4,29-6,95	0,035*
North America	70	12,69	10,85-14,53	0,118	106	5,65	4,67-6,64	0,005**
South America	71	14,65	12,82-16,48	0,597	84	5,89	4,78-6,99	0,039*
South-East Asia	207	13,72	12,65-14,79	0,419	364	5,60	5,07-6,13	<0,001***
Total	28 014				32 001			
P (overall equality)				0,010**				<0,001***

[a]Participants free of CVD. *P<0.05, **P<0.01, ***P<0.001

Table 6: NORRISK 10-year risk of CVD death. Data from Cohort of Norway, 1994-2003.

|	Men				Women		
Calculated 10-year risk % in 54 027 participants [a] (95% CI), adjusted to age.							
	N	Mean	95 % CI	P (equality of immigrants)	N	Mean	95 % CI
Norway	23 880	4,99	4,94-5,05	Reference	27 510	1,73	1,70-1,76
Eastern Europe	51	5,26	4,08-6,43	0,660	132	1,65	1,25-2,05
Former Yugoslavia	75	4,67	3,70-5,64	0,512	68	2,00	1,44-2,55
North Africa	31	3,44	1,93-4,95	0,043*	21	1,91	0,91-2,91
Sub-Saharan Africa	94	4,80	3,93-5,67	0,662	59	2,04	1,44-2,64
Middle East	297	4,11	3,62-4,59	<0,001***	193	1,36	1,03-1,69
Indian Subcontinent	438	4,67	4,27-5,07	0,119	304	1,67	1,41-1,94
East Asia	34	3,60	2,16-5,04	0,059	50	1,62	0,97-2,27
North America	63	5,52	4,46-6,58	0,333	98	1,71	1,25-2,18
South America	66	4,23	3,19-5,26	0,148	78	1,74	1,22-2,26
South-East Asia	175	3,91	3,27-4,55	0,001***	310	1,70	1,44-1,96
Total	25 204				28 823		
P (overall equality)				<0,001***			0,676

[a]Participants free of CVD, diabetes and who have never used antihypertensive- and/or lipid-lowering treatment. Post hoc test was not performed for women since overall variance was not statistically significant. *P<0.05, **P<0.01, ***P<0.001.
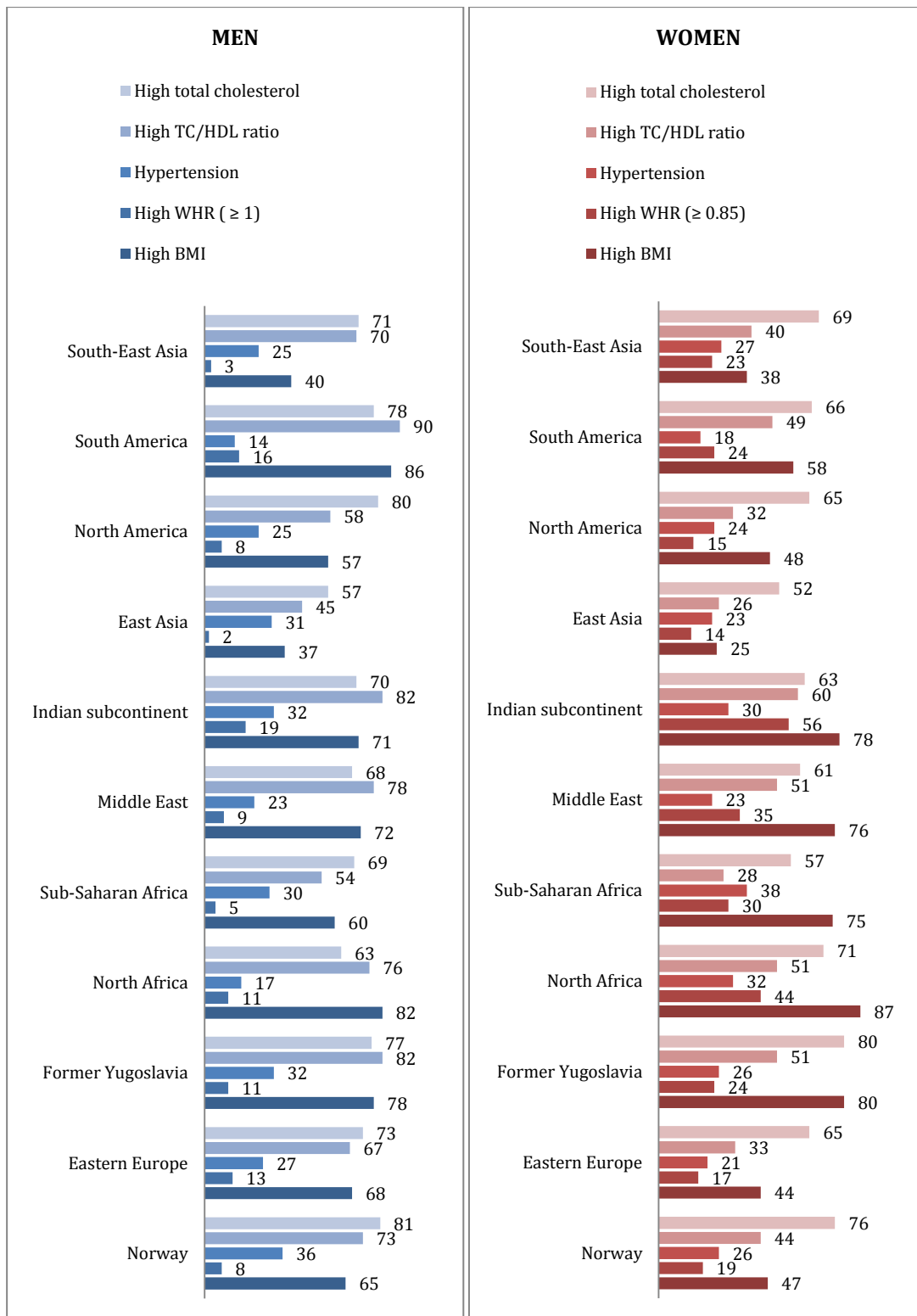
Figure 1: Prevalence (%) of risk factors in 62 145 participants, adjusted to age. TC, total cholesterol; HDL, high density lipoprotein; WHR, waist hip ratio; BMI, body mass index. All prevalence variables had P-values < 0,001 for overall equality except for hypertension in women (p=0,031). Data from Cohort of Norway, 1994-2003.
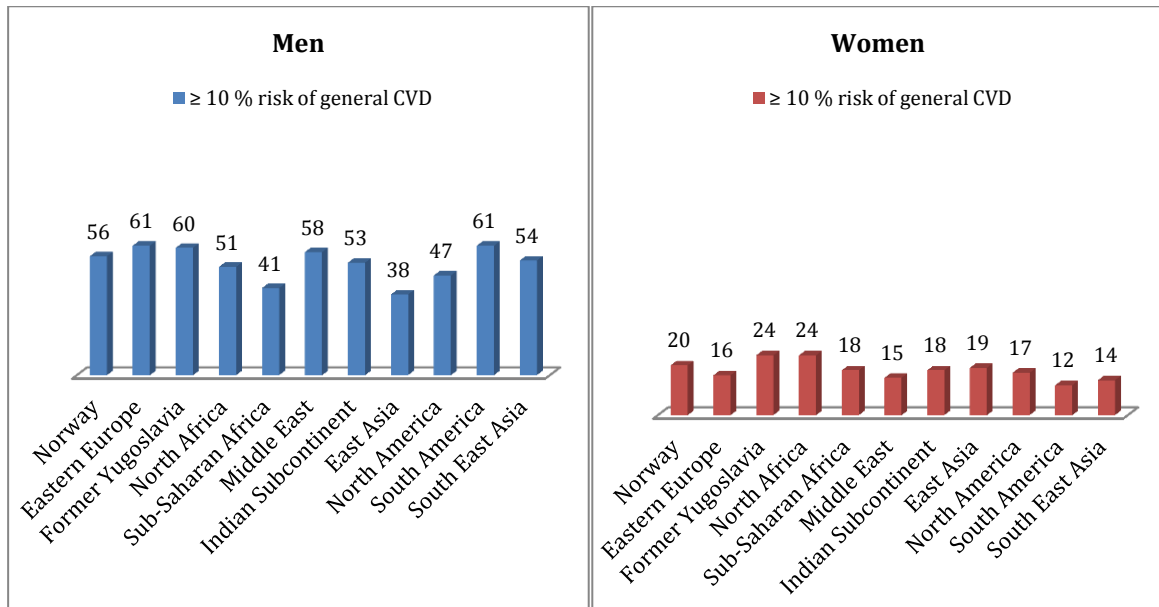
Figure 2a: Prevalence (%) of people defined as in high risk of developing CVD within 10 years in 60 015 participants according to the Framingham equation. Estimates are adjusted to age. Overall variance was significant in both men (p=0,001) and women (p=0,002). Data from Cohort of Norway, 1994-2003.
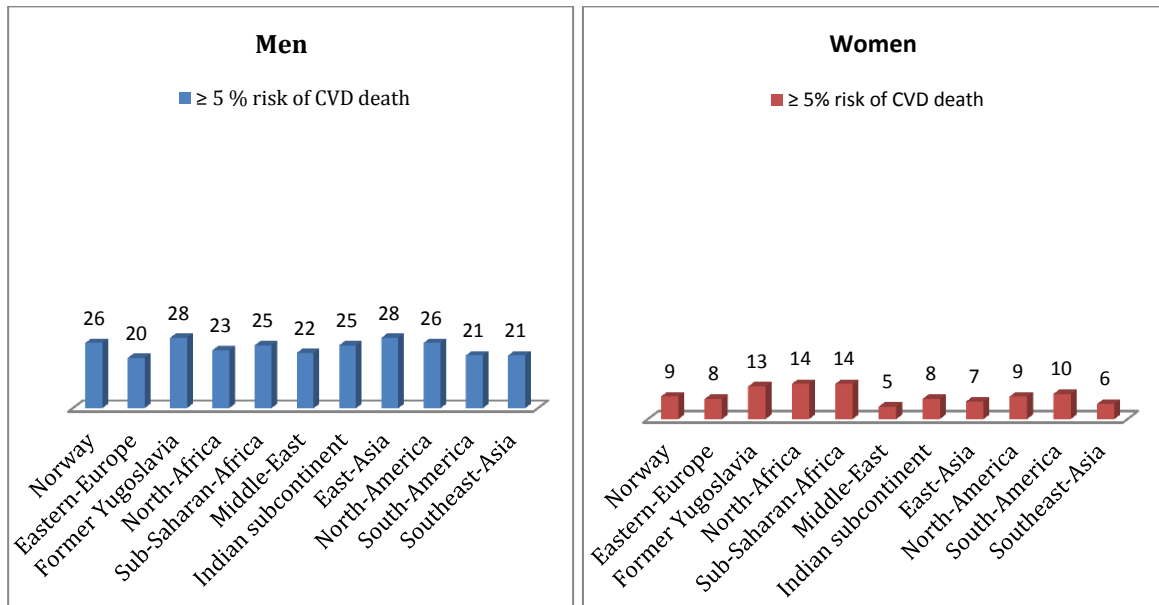
Figure 2b: Prevalence (%) of people defined as in high risk of dying from CVD within 10 years in 54 027 participants according to the NORRISK equation. Estimates are adjusted to age. Overall variance was significant in men (p=0,033) but not in women (p=0,103). Data from Cohort of Norway, 1994-2003.

Appendix:

Extract from the CONOR-questionnaire:

2. Do you have, or have you had?

	Yes	No	Age first time

Heart attack
Angina pectoris (heart cramp)
Cerebral stroke/Brain haemorrhage
Asthma
Diabetes

5a. How has your physical activity during leisure time been over the last year?

Think of your weekly average for the year. Time spent going to or from work counts as leisure time
Hours per week

	None	Less than 1	1-2	3 or more

Light activity
 (*not sweating or out of breath*)

Hard physical activity
(*sweating/out of breath*)

9. Do you smoke?

	Yes	No

Cigarettes daily
Cigars/cigarillos daily
Pipe daily

18 b. How many years education have you completed all together?
(Count every year you went to school)
Number of years………….

45. Do you take?

	Currently	Previously	Never

Lipid lowering drugs

Medications for high blood pressure

The complete CONOR-questionnaire can be downloaded through this webpage:

http://www.fhi.no/dokumenter/4fe7c0f0e2.xls