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# It Breaks a Man's Heart - Socioeconomic Differences in the Onset of Cardiovascular Disease in Contemporary Sweden

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# It Breaks a Man's Heart

## Socioeconomic Differences in the Onset of Cardiovascular Disease in Contemporary Sweden

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# List of Abbreviations

| | |
|---|---|
| BMI | Body mass Index |
| CHD | Coronary heart disease |
| CVD | Cardiovascular disease |
| DCM | Demand and control model |
| ERI | Effort reward imbalance |
| ICD | International classification of disease |
| IHD | Ischemic heart disease |
| MDCS | Malmö Diet and Cancer Study |
| MI | Myocardial infarct |
| SES | Socioeconomic status |
| SLI | Swedish Longitudinal Immigrant database |
| WHO | World Health Organization |

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Chapter 1

# Introduction

## 1.1. Motivation of the Thesis

The development of human populations is determined by the demographic dynamics of fertility, mortality and migration. Health and disease patterns are a major component of all three demographic events. Epidemiology, the study of the distribution of disease and mortality as well as their causes and consequences, is therefore a substantial component of public policies and public interest.

Researchers have identified global patterns of development in disease and mortality. One of the most fundamental global patterns is the theory of the *Epidemiologic Transition* by Omran (1971). The theory describes three stages of disease patterns in the transition from a population mainly challenged by pestilence and famines to a population facing primarily degenerative and "*man-made"* diseases. During the first stage, population growth is limited by Malthusian *positive checks*, which refer to population stagnation or reduction caused by famines, pandemics and violent deaths. The transition theory goes on to describe how changes in socioeconomic, cultural and political circumstances shifted the demographic pattern (decline and stabilization of mortality and fertility rates, rising life expectancy) to arrive in an era determined by degenerative and *"man-made"* disease, such as cancer or diseases of the cardiovascular system.

The effects of the epidemiological transition are not only noticeable in the size and composition of the population but also in the economic output produced by the population. Decreased mortality and morbidity due to lower prevalence of infectious disease increases economic productivity and labor efficiency (Omran 1971). Through the prolonged survival of adults, the attainment and transmission of knowledge and skills is intensified and supports the development of national economic growth.

While leaving behind some obstacles for population growth and economic development, a society whose health status is largely determined by degenerative diseases faces new challenges. With continuously increasing life expectancy in most developed countries, a large share of the population lives not only until the end of their economically active period, but as also well beyond that. Entering the last stage of Omran's Epidemiologic Transition during the middle of the 20$^{th}$

century, the developed world experienced a strong increase in cardiovascular disease (CVD) mortality until the 1980s. From that time, mortality due to cardiovascular diseases began to fall again, while the morbidity rate for CVD was still increasing. These diverging trends in mortality and morbidity are caused by improvements in the treatment of acute cardiovascular events, leading to a higher share of patients surviving an initial event.

Alongside high and increasing survival rates among CVD patients, the prevalence of CVD and CVD-related medical problems is also very high. Currently, CVD is the leading cause of death in developed countries, accounting for about 40 percent of all deaths.

This thesis examines the case of Sweden, where over 40 percent of all-cause mortality is caused by CVD (Socialstyrelsen 2009). Treatment for diseases of the cardiovascular system is rather intense and therefore costly, because it not only includes medical intervention for acute events but also treatment for risk factors such as diabetes and hypertension, as well as medical management of chronic heart disease.

Sweden has a universal healthcare system that is almost entirely tax-financed, implying that the costs associated with CVD account for a substantial share of the overall public healthcare costs in Sweden. In 2010, the direct and indirect costs of CVD amounted to roughly SEK 61.5 billion (~$9.3 billion). Direct costs contain expenditures for physicians, hospitalization, medication and home healthcare. Indirect costs account for costs of lost future productivity caused by premature mortality. The expenditures for healthcare of CVD patients represent around eight percent of the total healthcare expenditures in Sweden in 2010 (Steen Carlsson and Persson 2012).

Given the high public costs for treatment of CVD and the economic loss due to premature morbidity and mortality among CVD patients, research on the causes and consequences of CVD has high priority within the field of public health. The prevention of the onset of the disease as well as the prolongation of general good health has become the focus of recent research on CVD. Those efforts are reinforced by the findings of previous research demonstrating that a substantial share of CVD is not inevitable and the onset and course of CVD can be altered by actions of the healthcare system and, more importantly, by the individual itself, through the maintenance of a healthy lifestyle.

One important impact factor for CVD was found in socioeconomic status (SES) (Adler et al 1994). SES could be linked to many CVD risk factors and appears to be the origin of major direct and indirect impact pathways to the onset and progress of the disease. A large share of studies in the field of social epidemiology, sociology and public health focus on the relationship between SES and various health outcomes, including CVD. Some large-scale studies have

had the specific aim of evaluating the link between SES and CVD such as the Framingham Heart Study (Dawber and Kannel 1958) the Whitehall Study II and the co called "Black report" (Department of Health and Social Security 1980). During recent decades many studies have confirmed the existence of a social gradient, finding better health among individuals in the higher social classes (Cabrera et al 2001; Mackenbach et al 1997; Pocock et al 1987).

The link between SES and health is not a straightforward one, however. One reason for the complexity of the relationship between SES and CVD lies in the variety of SES measurements. SES can be operationalized in a number of manners, such as occupation, economic performance, education, labor market attachment or other characteristics, and most of these measurements are highly correlated with each other. Formal education can be expected to lead to occupational success, while both characteristics are the basis for income attainment and labor market participation. While all these forms of operationalization will influence the risk for CVD in a similar way, regarding the direction of the effect, the magnitude of the effect can vary substantially. Furthermore, the effects of SES will vary depending on the demographic characteristics of individual. The individual ethnic background and marital status will shape SES impact on CVD as will the sex and age of a person.

This thesis investigates SES differences in the onset of CVD among samples of the population in contemporary Sweden. The overall aim is to achieve a broad picture of SES impact factors and their direct as well as indirect effects on CVD, taking other risk factors and individual characteristics into account. Throughout the papers, included in this thesis, SES is operationalized in different forms. Therefore, each paper investigates a different aspect of the relationship between SES and CVD, emphasizing the complexity of the relationship. The findings from this study will be useful for identifying opportunities for future CVD prevention programs aiming at reducing SES differences and the resulting health impact among the population.

For many of the CVD risk factors there is the risk of reverse causality. On the one hand, lower SES could be the reason for unhealthy lifestyles and therefore increase the risk for CVD. On the other hand, the incidence of CVD could cause changes in labor market attachment and income level. This thesis is taking part of the causality problem into account by limiting the analysis to the onset of CVD (only the first CVD event for every person). Furthermore, the empirical part of the thesis focuses on coronary heart disease as the main subgroup of CVD, thereby excluding more rare forms of CVD, mainly incidences of stroke, for which the empirical results of SES impact have been less consistent.Building on established theories and models, this thesis identifies new aspects and impact

pathways of SES in relation to the onset of CVD, taking into account a set of additional risk factors and their potential effects on CVD.

## 1.2. Cardiovascular Disease and Their Impact Factors

With the secular decline in infectious diseases, the era of cardiovascular diseases (CVD) started in the middle of the 20th century in most developed countries. Although the incidence rates for CVD peaked and started to decrease again in the 1980s and 1990s, CVDs are still the leading cause of death in the developed world (WHO 2011a, 2011b). Data from a recent Swedish national health report (Folkshälsorapport 2009) shows that mortality due to heart disease and stroke declined constantly in recent decades. For women, the heart disease-related mortality rates have been declining since the beginning of the 1960s, while for men the rates continued to climb until the 1980s before they started decreasing as well. In 1952 roughly 500 women and 600 men died due to heart disease per 100,000 individuals. By 2006 these numbers had fallen to 220 women and 360 men per 100,000 individuals.

While the absolute number of CVD incidences has been steadily decreasing together with the case fatality rate (share of fatal cases among all incidences), CVD morbidity has increased. These opposing trends of decreasing mortality and increasing morbidity have several reasons. For one, the survival rate of individuals afflicted with acute CVD, such as angina pectoris and myocardial infarction (MI) has improved. Additionally, better treatment for individuals who are diagnosed with CVD (tertiary prevention) is more effective and available.

The need for understanding and preventing CVDs as well as their risk factors has become a priority of many public health interventions. In order to establish useful preventive programs, one has to understand the disease itself and its influencing factors (Marmot and Elliot 2005). The following section will provide an overview of CVD, their mechanisms and risk factors.

The term cardiovascular disease includes all diseases affecting the heart and the blood vessels in all parts of the body, including the heart and the brain. The majority of CVDs can be categorized in either cerebrovascular disease or coronary heart diseases, i.e. CVD of the brain or CVD of the heart. The term stroke is used for a number of specific cerebrovascular diseases and identifies problems of the blood supply to the brain which can have three reasons. The first reason is a blood clot (ischemic stroke), blocking the blood supply to regions of the brain. Non-detection or delayed treatment of a stroke can provoke major loss of brain

function or death. These blood clots can have two origins. They form in the left heart chamber due to a reduced or arrhythmic heartbeat and can move with the blood stream up into the arteries of the brain. Blood clots, containing plaque tissue and blood, can also originate from atherosclerotic plaque in the arteries of the throat.

Other forms of stroke are hemorrhagic stroke and subarachnoid hemorrhage, both defined as bleeding in different parts of the brain. The result of all forms of stroke is a shortage of blood, oxygen and nutrients due to decreased blood supply beyond the place of the clot or bleeding in the brain. Acute symptoms of cerebrovascular disease are for example loss of control over limbs, lack of ability to understand others (impressive aphasia) or to express themselves comprehensively (expressive aphasia). The success of treatment is strongly correlated to the time passed since the incidence and start of treatment (Khan 2005).

The underlying causes of cerebrovascular disease vary considerably, and the impact mechanism from various risk factors is less clear in comparison with coronary heart disease (CHD). Furthermore, socioeconomic differences and lifestyle characteristics are more strongly correlated with CHD than with the incidence of stroke. Thus, this study exclusively investigates incidences of CHD, with a focus on their socioeconomic causes.

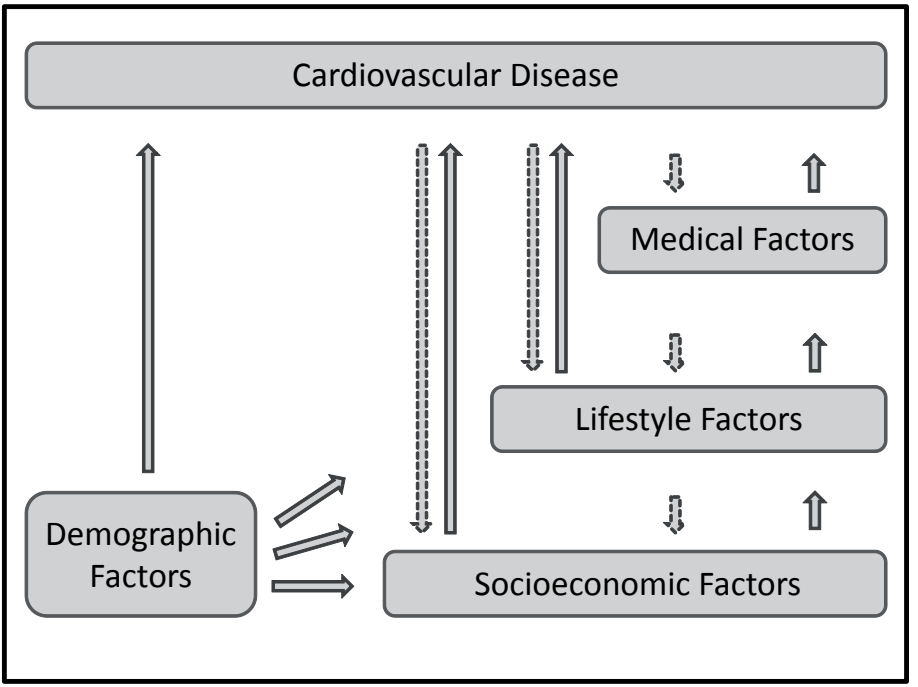
CVD of the heart – CHD – includes dysfunctions of the arteries which provide the heart with blood, oxygen and nutrients, as well as the dysfunction of heart muscle itself. One of the most common diagnoses is ischemic heart disease (IHD), defined as insufficient blood supply to the heart, with atherosclerosis as underlying cause. Although the loss of function of the heart is not necessarily fatal in its initial stage, early diagnosis and treatment are important to prevent future heart disease incidence and increasing morbidity (Kannel and Belanger 1991). A heart attack, or myocardial infarction (MI), appears if the blood supply to the heart is completely stopped. The main cause for such a blockage (thrombus) in the coronary arteries is a rupture of atherosclerotic plaque. Another cause of MI is, for example, a spasm of the arteries, which is independent from atherosclerosis but occurs with much lower frequency than an atherosclerotic thrombus. The resulting lack of oxygen can cause damage to the heart muscle and can lead to a complete cessation of heart function in the worst case. A non-fatal MI incidence decreases the blood stream and causes an insufficient supply of blood to the heart muscle (ischemia). Ischemia of the heart due to MI can lead to the death of heart muscle cells, which can cause cardiac arrest if left unattended or if treatment is delayed. An often-described symptom of IHD is sudden chest pain (angina pectoris) and it signals the urgent need for treatment in a patient.

The risk factors causing CVD, CHD and IHD are multifaceted, and therefore the following sections will provide an overview of the main causes of CVD

and their impact mechanisms. The overview will to some extent follow the recommendation of the American Heart Association to distinguish the impact factors by the criteria of being preventable (smoking, hypertension, overweight) and impact factors which are not preventable (age and sex of the individual). To illustrate the structures of the various risk factors and their effects on CVD, figure 1 presents the interactions between risk factors and disease.

The main risk factors for CHD and IHD come from a variety of fields – socioeconomic, psychosocial, behavioral, medical and genetic. Socioeconomic factors are often seen as positioned early in the chain of causality and therefore influencing other risk factors and consequently influencing the risk for CHD and IHD using an indirect impact pathway. This overview will start with a description of atherosclerosis as a main underlying factor for CVD. Following this, the medical conditions of diabetes and hypertension and their impact on IHD and CHD will be discussed.

**Figure 1**: Interrelation of risk factors and their impact on CVD




Following the structure of figure 1, several important lifestyle factors and their interaction with each other and with medical conditions as well as their direct effects on CVD are described. Subsequently, an introduction of socioeconomic

factors is provided. The differences in socioeconomic conditions and their effect on risk for CVD is the main focus of this thesis. An understanding of the underlying mechanisms can only be achieved if the interrelations between socioeconomic conditions, lifestyle factors and medical output are taken into account. Therefore the section tries to capture the indirect and direct effects of socioeconomic conditions. As demonstrated in figure 1, demographic factors are not part of the causal chain from socioeconomic factors to disease. Characteristics such as sex and age are inevitable and cannot be influenced by other risk factors. Simultaneously, demographic characteristics have strong impact on both heart disease and the above-mentioned risk factors for heart disease.

Figure 1 shows that there are potential feedback effects (dashed arrows) following the opposite impact direction. Regarding the feedback effect of CVD on any of the risk factor groups, this thesis minimizes the risk of bias due to the focus on the onset of CVD. This methodological strategy included the censorship of individuals from the analysis once they experienced a CVD event.

### 1.2.1. Medical Factors

The human body is complex, and the occurrence of malfunction due to one disease may also have an impact on other bodily functions and diseases. CVDs are influenced by several medical conditions which can have both behavioral causes as well as genetic origins. In the following section, some medical conditions and their working mechanisms which are specifically important for the onset and progress of CVD are described. Although the three medical conditions of hypertension, diabetes and atherosclerosis are described in separate paragraphs, this thesis makes an effort to emphasize the interrelationship between CVDs, their causes and consequences.

CVDs are influenced by much more than the medical factors presented here (e.g. blood cholesterol, triglycerides and alpha 1 antitrypsin (Marmot and Elliot 2005)). Studies have shown that the level of cholesterol and genetic preconditions for CVD or CVD risk factors have a strong influence on the development of CVDs in affected individuals. Nevertheless, this study focuses on the three medical conditions of atherosclerosis, diabetes and hypertension for two important reasons. First, hypertension, diabetes and atherosclerosis are important underlying medical causes for CHD and IHD, the analyzed health outcome variables. Second, information about prevalence and incidence of those three medical conditions are provided by both of the databases used in this thesis. Another reason this study is limited to the analysis of those three medical

conditions is the consistently strong correlation with various CVDs which has been found in numerous studies, as the following sections will show.

*Atherosclerosis*

The medical condition of atherosclerosis is not a risk factor for CVD, but rather part of the disease process. The onset and progress of atherosclerosis are long term processes, and the diagnosis of different stages of atherosclerosis can indicate the risk for future CVD incidences. Given the importance of this medical condition for all CVDs, the progress and impact mechanism is described in the following.

The term atherosclerosis means, according to its Greek origin, hardening of the arteries. It describes the accumulation of fatty tissue in the artery walls as well as the calcification of the artery walls. This process has two dangers. The first is caused by the thickening of the artery wall, the loss of blood vessel's elasticity, with an increasing risk for higher blood pressure (hypertension). The heart muscle will need more effort to transport a sufficient amount of blood through the hardened arteries, which can cause premature ischemia of the heart. The second dangerous consequence of atherosclerosis is thrombotic plaque. The calcification of the artery walls produces a rough surface on the inside wall of the arteries which can serve as a source of plaque. Furthermore, plaque inside the artery walls build up with the accumulation of lipid cells, including inflammatory cells. The membrane which keeps the plaque inside the wall is called the fibrous cap and with decreasing thickness of the fibrous cap the risk for a rupture increases. In cases where the plaque capsule breaks, the lipid and inflammatory cells are released into the blood vessel and enter into the circulatory system (Hansson 2005). While inflammatory cells represent a danger to the inflammatory system, lipid cells can clot and act as thrombus in the arteries, provoking a blockage of blood which can cause MI (heart) or stroke (brain). The relation between elevated inflammatory reaction and the onset of atherosclerosis has been given special attention in research (Libby 2002; Ross 1999).

Atherosclerosis is a natural byproduct of aging and has a long accumulation period prior to becoming a health hazard. The onset of atherosclerosis has been occasionally found in very young patients, but the disease does not usually become a health hazard before adult ages (Charakida, Tousoulis and Stefanadis 2006). Despite the inevitable onset of the disease, there are many factors which influence the progress of atherosclerosis. It shares many impact factors with the two CVD risk factors of hypertension and diabetes, and co-morbidity with any of them increases the progress of atherosclerosis. While physical inactivity increases the progress of the disease, healthy nutrition and a stress free environment can decelerate the process. Smoking is a strong health risk for atherosclerosis as well.

Other impact factors are ethnicity and sex of the individual (Frohlich and Lear 2002) as well as high blood cholesterol (McNamara 2000).

*Hypertension*

Hypertension is the chronic medical condition of elevated arterial blood pressure. The heart muscle of individuals with hypertension has to exert more effort to maintain a constant blood flow and, as with any muscle, it will tear faster with overuse. The normal level for blood pressure is around 100-140mmHg for systolic blood pressure (contracting heart muscle before heart beats) and 60-90mmHg for diastolic blood pressure (relaxed heart muscle between heart beats)(Appel et al 2006). When blood pressure is only occasionally in the upper range, it is called pre-hypertensive state and when individuals suffer from continuous blood pressure above 140 /90mmHg (systolic/diastolic) it is classified as hypertension. Studies showed that patients with moderately elevated blood pressure (pre-hypertensive) should be monitored, because they are under a higher risk of developing hypertension, compared with individuals who have a normal blood pressure (Henriksson et al 2002). While hypertension cannot be cured and only treated, pre-hypertensive states are still reversible with appropriate treatment, which can include medical treatment as well as lifestyle changes regarding diet and intensity of physical exercise.

Blood pressure increases commonly with age, so elderly people are more prone to develop hypertension. The increased pressure in the arteries leads to higher demands on the heart muscle and makes an atony of the heart muscle (losing strength in the muscle) more probable. The elevated demands of the heart muscle will accelerate the ageing of cells and increase the risk for premature ischemia of the heart. Therefore, hypertension is a major risk factor for CVDs in general (Hansen et al 2007) and IHD in particular. According to the World Health Organization (WHO 2011a) hypertension causes up to 7.5 million deaths per year, or 12.5% of all annual deaths. The origin of hypertension is partially found in genetic pre-conditioning. However, important impact factors for the development of hypertension are found in individual lifestyle and health behaviors. Besides the independent effect on CVD, smoking is, after age, one of the major causes of hypertension. Furthermore, physical inactivity and unhealthy nutritional habits, especially an overuse of salt in food preparation are positively correlated with the onset and course of hypertension (Appel et al 2006; Folkow 1982).

Hypertension is also correlated with other cardiovascular risk factors like diabetes and atherosclerosis (Sipahi et al 2006), making co-morbidity with those diseases a very dangerous combination as they imply direct and indirect pathways leading to the onset of CVD.

*Diabetes*

Diabetes is the inability of the body to metabolize sugar due to either insufficient insulin production (Type I diabetes) or to a missing response to insulin at the cellular level (Type II diabetes) (Rhodes 2005). Type II diabetes accounts for the majority of diabetes cases within the general population. Because the symptoms progress very slowly, individuals can live with undetected Type II diabetes for years. Type I diabetes is age-independent, and is diagnosed shortly after rapidly-appearing symptoms. Because this type of diabetes can occur in children and adolescents, it is often referred to as juvenile diabetes. There are several forms of Type I diabetes, depending on which dysfunction of the immunological system causes the impairment of insulin production. A special form of Type II diabetes is gestational diabetes, occurring in pregnant women who were not diagnosed with diabetes prior to the pregnancy. This form of diabetes can continue into the development of permanent Type II diabetes, but in most cases resolves after the end of the pregnancy.

As with hypertension, diabetes is a chronic disease which can be treated, for example with insulin medication (Type I) or lifestyle changes (Type II), but not cured. It was estimated that about 1.3 million deaths were attributed to diabetes globally in 2008 (WHO 2011b). A predisposition for diabetes can be inherited for Type I and Type II diabetes (O'Rahilly, Barroso and Wareham 2005), but the incidence of diabetes is also strongly correlated with lifestyle factors such as smoking and diet, with obesity being considered a main risk factor for the onset of Type II diabetes (Lazar 2005). The alarming increase of overweight and obesity among children and young adults prompted scientists to lower the mean age of onset for Type II diabetes, which was traditionally considered to be in middle adulthood (age 35-45). Early life overweight and obesity is a strong predictor for overweight and obesity in adult life and, correspondingly, diabetes, and thus a risk factor for cardiovascular disease (Friedemann et al 2012).

Due to the strong correlation between diabetes and overweight and obesity, patients with diabetes are recommended to sustain a normal weight or reduce their weight if it exceeds a body mass index of 25 by changing dietary habits in combination with increased levels of physical exercise (Aucott 2008). Studies found a health promoting effect of regular exercise for diabetic overweight patients (Sluik et al 2012) leading to the assumption that the primary underlying health effect of weight loss is found in the increased level of physical activity rather than the change in body weight itself.

Among other effects, diabetes can potentially damage kidneys and the nervous system, and cause problems in the eyes (diabetic retinopathy). In terms of CVD, diabetes causes damage to small and large blood vessels, making it a strong risk

factor of CVD. Especially in combination with other risk factors as hypertension or high cholesterol (Fuller et al 2001), diabetes increases the risk of the onset and progress of CVD.

From the sections above, a picture appears of highly correlated medical risk factors for CVD. The development of one of the diseases increases the risk for the other risk factors. This is partly due to the symptoms of the different diseases, but also to their common causes. Smoking, for instance, has been reported to increase the risk for all three medical conditions. However, smoking is not the only behavioral lifestyle impact factor for CVD. The following section investigates several of the main lifestyle factors known to alter the risk for CHD and IHD.

### 1.2.2. Lifestyle Factors

As mentioned above, several of the cardiovascular diseases and related medical conditions have a strong connection to health behaviors and lifestyle. In the following section the most important lifestyle factors related with CVD are presented, including research results on their impact on CVD risk. While smoking, dietary preferences, and the degree of physical activity all have direct bio-medical effects on the cardiovascular performance of the individual, social capital and stress will be presented as sources of indirect impact on CVD.

*Smoking*

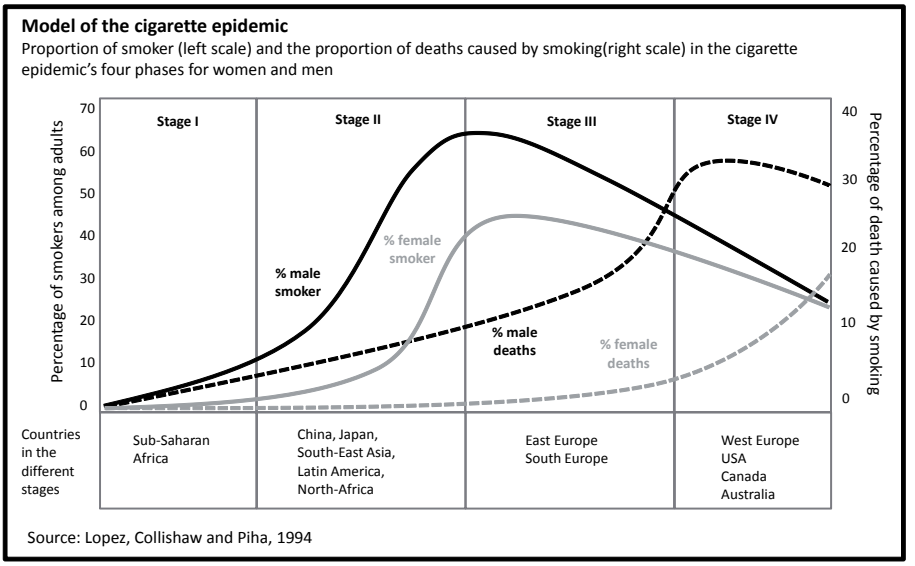
Statistics suggest that smoking is the most important cause of premature death in the developed world (Socialstyrelsen 2009; WHO 2011b). The prevalence and incidence of smoking, as well as the resulting rates of mortality and morbidity, follow global patterns. Lopez, Collishaw and Piha (1994) suggest four different phases of a general smoking epidemic model. Figure 2 illustrates the different stages of the smoking epidemic and categorized global regions according to which stage they are currently experiencing.

In stage I, only a small share of the population smokes and mortality caused by tobacco consume is relatively low. In stage II, both tobacco consumption and cigarette-related mortality increase drastically. While smoking prevalence reaches the peak in stage III, tobacco-related mortality is still climbing, caused by the time delay between long-term hazard accumulation and the onset of health problems. During the second half of stage III and all of stage IV prevalence of smoking is decreasing. In all four stages the share of smokers and deaths due to smoking is lower for women than for men, caused by a later uptake of smoking among women, and a traditionally lower share of female smokers than male smokers.

The cigarette epidemic model suggests that the rates for male and female smokers will converge with time, until the share of women might even exceed the share of men. As a consequence of the different prevalence of smoking between men and women, their tobacco-related mortality shows a different development over time as well. As illustrated in stage IV, mortality for men decreases slowly, adjusting to the lower prevalence of male smokers, while female mortality related to tobacco consumption still rises.

According to the time of epidemic onset, the global regions are currently in different stages of the cigarette epidemic model (Ezzati and Lopez 2003). Sweden and the rest of the Nordic countries have already reached stage IV with a declining prevalence of cigarette smoking and a convergence between the sexes.

**Figure 2**: Model of cigarette epidemic by Lopez Collishaw and Piha (1994)




The detrimental effects of smoking on non-communicable diseases are manifold. On the one hand, carcinogenic substances in tobacco are released into the body. These substances bind with the DNA and can cause mutations or death of cells. Therefore, cancer is a disease commonly correlated with smoking, especially lung cancer and cancers of the mouth and pancreas (Feng et al 2006). On the other hand, smoking affects the cardiovascular system in several ways. First, it has an immediate effect of increasing the heart rate. Second, the carbon monoxide in the smoke reduces the amount of oxygen transported in the arteries and therefore requires the heart to work harder to supply sufficient oxygen. Third, the substances present in smoke, mainly nicotine, also cause a narrowing of the

blood vessels, which as explained earlier has a negative effect on the function of the heart and the cardiovascular system as a whole (Haldane 1895). Accordingly, these effects accelerate the development of atherosclerosis and CVD risk factors such as hypertension.

The effects of smoking depend on the age at take-up, the length of the smoking career and the amount of cigarettes smoked per day (or the amount of tar contained by the cigarettes, cigars or other forms of tobacco (Peto 1986)). Thus, some of the effects work in the long-term, with the difference in mortality and morbidity risk between smokers and non-smokers becoming more visible in older ages (LaCroix et al 1993). Long-term studies have shown that half of all smokers will die due to their smoking habit, and that about 20% of all CVDs in Europe are attributable to smoking (Allender et al 2008).

The WHO considers smoking to be a fully avoidable risk factor for CVD, because smoking is a voluntary task (first-hand smoke). They claim that six million people die of tobacco smoke, second-hand smoke, and their consequences each year, accounting for 6% of all female and 12% of all male deaths (WHO 2011b). It is assumed that the share of women dying due to smoking-induced diseases will rise because nearly as many women as men currently smoke in developed countries, which was not the case in previous decades. There is a social gradient in smoking, with a higher prevalence of cigarette smoking among the lower social classes in developed countries (Jarvis and Wardle 2011). Countries which are still in earlier stages of the cigarette epidemic might show different social distribution of smoking prevalence.

This thesis analyzes the case of Sweden, where an estimated 14% of all men and 18% of all women over the age of 15 were smokers in 2005 (Allender et al 2008). Furthermore, use of another form of tobacco known as *snus* (oral, moist, smokeless tobacco) is widespread in Sweden. The effect of this alternative to cigarette smoking on CVD mortality and morbidity has not been consistently found (Hansson et al 2009), however, and its effects are therefore not under consideration in this thesis.

Information about individual smoking habits is available for the first paper in this thesis and is used as a categorical impact factor for the onset of CHD. Individuals are distinguished as being non-smokers, regular smokers, occasional smokers or those who have stopped smoking. For the other three empirical papers no information about smoking was available, and therefore smoking habits could not be taken into consideration in those papers.

*Diet, Physical Inactivity, Overweight and Obesity*

The importance of nutrition for health and well-being is no longer a debatable issue. The consequences of poor food choices and unhealthy dietary habits often take the form of cardiovascular mortality and morbidity. Modern processed food makes it easy for an individual to consume the amount of energy the body needs for the daily activities. Unfortunately, in the developed world abundant food supplies also increase the chance to exceed these basic needs.

An unhealthy diet contains high amounts of sugar, salt, cholesterol and saturated fat, and often a combination of those (Méjean et al 2011). Common ingredients in processed food increase the prevalence of other CVD risk factors like hypertension (high salt intake), diabetes (high sugar intake) or the development of atherosclerosis (high saturated fat intake). When referring to an unhealthy diet as a preventable CVD risk factor (WHO 2011a), one must keep in mind that food choices are made not only by taste preferences. They also depend on cost, availability, convenience, perceived health-promoting features and social, cultural, religious and psychological considerations (Shepherd 1990). Convenience and cost factors are especially significant in the choice of unhealthy over healthy food, and SES is also highly correlated with dietary choices.

A special case of dietary habits is the consumption of alcohol. The direction and magnitude of health effects of alcohol depend on the amounts and type of alcohol consumed, as well on the physical characteristics of the individual (Bobak and Marmot 2011). A less clear social gradient is observed for alcohol compared with other lifestyle factors. Certain amounts of specific alcohol types are assumed to be beneficial, but there is a great amount of uncertainty about the benchmarks. Excessive consumption of alcohol, especially of high-strength drinks, has been found to be a health risk regarding CVD (Renaud and Delorgeril 1992).

As mentioned above, a healthy diet can be achieved with balanced level of dietary habits and physical activity. Low physical activity at work, caused by predominantly sedentary tasks, or mainly inactive behavior during leisure time was detected early in cardiovascular research as a risk factor for MI and stroke as well as other health problems (Eriksen and Bruusgaard 2004; Paffenbarger, Wing and Hyde 1978; Salonen, Puska and Tuomilehto 1982). The underlying effects of an increase in physical exercise include lowering of blood pressure, plasma fibrinogen, plasma viscosity and improvements in glucose metabolism as well as blood lipid levels (Lindström, Hanson and Östergren 2001). Maintaining a regular level of physical activity is an important part of a healthy lifestyle. Even modest levels of activity have preventive effects for CVD disease, in particular for older individuals (Buchner et al 1992; Wagner et al 1992). Moderate activity of 150 minutes weekly is estimated to reduce the risk for IHD by 30% and the

risk for diabetes by 27% (WHO 2007). Estimations suggest that more than 30% of the global population did not meet this minimum level of physical activity in 2009 (Hallal et al 2012). Given these numbers, researcher have started to call the high prevalence of physical inactivity a "Global pandemic" (Kohl et al 2012). The effects of physical inactivity make it an important impact factor for cardiovascular mortality and morbidity (Lee et al 2012).

On the other hand, physical exertion has also been found to increase the risk for heart disease (Hallqvist et al 2000). These findings lead to the conclusion that the cardiovascular system is a sensible system which requires regular maintenance and suffers from both under-use and over-use.

Consumed energy units that are not used will be stored in the body as a reserve, in case of need at a later point in time. When energy is stored in such a way over a long period, individuals risk becoming overweight or even obese. The classification of overweight and obesity is geared to the measurement of the Body Mass Index (BMI) calculated in the metric system as the ratio of weight in kilograms to the square of the height in meters. A measured BMI of over 25 is regarded as overweight, and obesity begins at a BMI value of over 30. In 2008, the estimated total number of overweight adults above the age of 20 in the world was 34% (WHO 2011a). In the UK every year, approximately 30,000 people die due to the effects of obesity. In the US the number is about ten times higher, having already overtaken smoking as the most prevalent avoidable risk factor by 2005 (Haslam and James 2005).

This thesis analyses the case of Sweden, where national studies estimated that, for the period 2004-2005, 26 percent of women and 41 percent of men in the age range of 16-84 were overweight. In comparison with data from 1980-1981 (women: 22 percent and men: 30 percent), this implies a relatively rapid increase, at least for men. The share of obese individuals in Sweden doubled in the same time from five percent in 1980-1981 to ten percent in 2004-2005 (Socialstyrelsen 2009).

In fact, the recent development of obesity in the developed world has led researchers to speak of an "Obesity epidemic" (Marinou et al 2010). Obesity as result of improper dietary choices and physical inactivity is an independent risk factor for CVD. While overweight and obesity seem to have at least partial genetic origins (Carlsson et al 2011), a strong focus in research is still on the lifestyle factors regulating surplus energy intake with too few opportunities to expend this energy.

All three factors – dietary preferences, physical activity and elevated body weight – are strongly interrelated with each other. While higher physical activity can reduce an elevated body weight, overweight and obesity have an impact on physical activity in return. It is much more exhausting to do regular exercise for a

person who is overweight or obese, and the extra weight puts increased pressure on joints and can complicate movements. Therefore, an elevated BMI is often correlated with a decreasing level of physical activity. The overall recommendation of maintaining a healthy lifestyle therefore combines a balanced diet, regular exercise and a stable BMI under the value of 25. There has been critique about the measurement of BMI, because the BMI value is sensitive to changes in body composition (i.e. the ratio of fat to muscle tissue). For individual risk evaluation, other measures such as Hip-Waist-Ratio or percentage of body fat might be more accurate, but for population-level analysis, BMI is an appropriate measure of CVD risk groups.

BMI, as a comprehensive measurement and indirect indicator of healthy dietary habits and physical exercise, is included in paper I, because the data is available. The database used for the other three empirical papers is register-based and therefore does not include any information about individual anthropometric measurements or health behaviors such as dietary preferences and physical exercise. For that reason, it was impossible to include the indirect and direct health impact pathways leading from socioeconomic differences to variations in CVD risk for the empirical papers II-IV. Nonetheless, the correlation between lifestyle and risk of CVD is part of the explanations in all papers.

### *Social Capital*

While the origins of the concept of social capital are based in sociology (Bourdieu 1986; Coleman 1988), its use spread during the last decades to many different scientific areas, connecting a whole spectrum of human behavior and social life in general. In particular Putnam and colleagues introduced social capital into social epidemiology as macro-level characteristics (Putnam 2000; Putnam, Leonardi and Nanetti 1993). Given today's acceptance and variety of usage in so many disciplines, it is less than surprising that social capital has been referred to as "*one of the most successful exports from sociology"* (Portes 2000).

Within studies of infectious diseases, individual contacts and social networks have long played a role. More recently, investigations have discovered the potential health impact from social capital for other kinds of diseases, including CVD (André-Petersson et al 2007; Harpham, Grant and Thomas 2002; Kawachi 1997).

Glaeser, Laibson and Sacerdote (2002) proposed in their economic approach to social capital that social capital could be seen as *the social component of human capital*, which would greatly decrease the importance of social capital as an independent factor. The authors showed that social capital accumulation follows patterns similar to the standard economic investment model, arguing that people

tend to gather assets of social capital if the private motivation is high. A different economic approach is provided by Grossman (1972). His study interpreted the connection between social capital and health as a general demand-for-good-health model, with health working as both an investment commodity and a consumption commodity, with social capital as a health promoting-asset.

As with many health risk factors, the concept of social capital faces causation problems. On the one hand, a strong social network could provide the individual with additional access to health-promoting information and services, thereby increasing personal health. On the other hand, fewer and less intense social bonds could be the result of health problems and subsequent limited access to social life.

While both causation directions are possible, the majority of current research investigates the potential health impact caused by differences in social capital assets. One of the first large-scale studies directly connecting social networks and health outcomes was the Alameda County Study, carried out from 1965 to 1974. Results showed that a lack of contacts with friends and relatives and low group membership were directly associated with higher overall mortality. People with fewer social contacts were 1.9 to 3.1 times more likely to die in the nine year follow-up study. Furthermore, this connection of isolation and higher mortality rates was found to be independent of behavioral aspects such as smoking, alcohol consumption or general physical activity (Berkman and Syme 1979).

Additionally, studies found a strong connection between recovery rates of patients with severe diseases and their social integration and social support resources (Cassel 1976; Cobb 1976; Seeman 1996). Study results indicated that the degeneration processes due to aging could be reduced through intervention in physical and social activity (Buchner et al 1992; Wolinsky, Stump and Clark 1995).

To explain the repeated finding of health benefits from high levels of social capital, Islam (2007) listed four possible channels of how social capital could determine individual health, which were first mentioned by Kawachi, Kennedy and Glass (1999). First, social capital could have an effect on psychological stress reduction and health-related behaviors through norms and values. Second, there could be potential direct changes of the personal health behavior through social capital relationships such as religious affiliation or club membership. Third, the individual health could be affected by better access to health care and communal amenities provided by the social network (Richardson and Norris 2010). Fourth, individual health could be influenced by increased social order, including lower crime rates and general safety (Putnam et al 1993).

The impact of social capital at the macro level on the health status of individuals can take several forms. According to Berkman and Glass (2000) common norms regarding health behaviors, such as diet, smoking and physical

activity, are a possible impact factor on the behavior of individuals in the same social environment. Good and healthy lifestyles can serve as a role model for others to follow.

The impact from social groups can potentially also promote detrimental health behaviors. Well known examples are smoking and alcohol consumption, especially among young adults (Cleary et al 1988). Regardless of the direction of the impact, all health behaviors will have a possible effect on the individual's risk for the onset of CVD, implying that the social environment can play an important part in disease prevention.

Another health impact pathway from social capital is alluded to within the field of bio-medicine, with studies connecting social isolation with lower functioning of the immune system. Glaser et al (1985) found a lower level of natural killer cell activity among a population of students who reported feeling lonely. This effect could cause an indirect impact from social conditions on latent infections due to a suppressed immune system.

Psychosocial stress originating from imbalanced social networks may also increase the propensity for psychosomatic health problems. An unstable family or employment situation has been shown to increase stress and subsequently influence health outcomes (Seeman 1996), and a strong relationship was found between the symptoms of depression and the incidence of CVD (Goble and Le Grande 2008).

Another operationalization of social capital is the degree of integration of an individual in the social environment. Social support and resources, as well as indicators for SES, were repeatedly found to influence CVD risks (Cassel 1976; House, Landis and Umberson 1988; Sundquist et al 2004; Valente 2010) Social support is assumed to play an especially important role in the survival after the onset of CVD (Berkman, Leo-Summers and Horwitz 1992). The positive impact of social support on other CVD risk factors was demonstrated as well (Hanson and Isacsson 1992; Sirven and Debrand 2008), and the social effects have been extended to include the impact of the immediate social environment (neighborhood) on the health of the individual and vice versa (Chaix, Rosvall and Merlo 2007).

Despite the widespread use of social capital, there are certain drawbacks to using the concept. On the one hand, the popularity of social capital in research brought advantageous attention to the term. On the other hand, however, the wide spectrum and non-uniform use of the concept makes generalization difficult, and increases uncertainty related to the topic. Parallel to the increasing number of studies dealing with the concept of social capital and its potential health impacts, there is an increasing confusion about its definition and interpretation.

The term social capital has a variety of concepts and approaches. This discordance among the different schools of social capital is leading to problems regarding the comparability of studies and results. "*One of the most confusing and frustrating aspects of social capital [...] has been the lack of consensus concerning its definition. [...] There is no single definition of social capital that everyone would agree upon; nor is there a standardized approach to measuring it – at least not so far"* (Kawachi, Subramanian and Kim 2008).

Given this substantial problem of definition and measurement, the lack of consensus as to which aspects of social capital are indeed important, there is the risk of potential distortion of results through inappropriate classification. While not disregarding the potential importance of the concept, it was decided not to include social capital as impact factor for CVD in this thesis.

*Stress*

Along with the classical CVD risk factors described in the section above, psychological stress has long been suspected to influence CVD risk, and health in general. Various situations have been identified as a potential source for stress, while the magnitude of influence varies between stressors at the individual level and stressors on the macro-level. A stressful work environment could alter the health status of its employees, complicated family problems can affect all family members, and economic and social crisis can influence whole populations and societies.

Stress and the human adaptation process to external stressors have been the topic of many studies. Much research is based on the work of Selye (1936, 1955). In his articles he explained possible pathways through which stress impacts the endocrine and nervous systems. While he insisted on the incompleteness of existing theories, more modern studies have become much more confident about their understanding of stress and its trigger mechanism for disease.

Researchers now agree on the mechanism of hormonal stress reaction, well-known as the *fight or flight* reaction. In times of stress in the form of acute external stimulus, the human body prepares for either a defense response or escape attempt (Brunner 2002). Both actions require energy amounts which exceed the average consumption by far. The process of adaptation is also called allostasis, which describes a process of adapting bodily functions to the new requirements during the time the body is exposed to the stimulus (Sterling and Eyer 1988) and thus achieving a new balance (homeostasis). The endocrinological changes, which include the adaptation process, work as protection for against the new stimulus. The release of stress hormones is induced from the adrenal medulla (catecholamines) or adrenal cortex (glucocorticoids) (McEwen 2002). They

prepare the body to work properly under the new conditions and to achieve homeostasis again. The adaptive process enhances the immune system by promoting the transportation of immune cells to the affected tissue parts.

Homeostasis during time of stimulus exposure represents a system running under special conditions, and if the stress continues for a longer time-span the beneficial effect of adaptation can reverse to a disease-promoting dysfunction of the body. The enhanced immune activity in the long run decelerates the response to acute stress. Some studies have found that long-term stress is rather immune-suppressive than immune-enhancing, which is in line with the health-damaging aspect of persistent stress (Dhabhar 2000). The allostatic systems are activated rapidly in acute situations and terminated quickly when their purpose is over. A dysfunction of the allostasis process appears when the long-term exposure to stress prohibits the system from stopping its adaptation process and returning to normal homeostasis. (McEwen and Gianaros 2010).

While the response to stress takes place in the endocrinological system, located all over the human body, the brain is the origin of this reaction (McEwen and Seeman 1999). The initial stimulus is transmitted to the brain where it is evaluated. Given the interpretation of degree and urgency of the stressor, the brain regulates the proper body response (Foley and Kirschbaum 2010). The evaluation of stress depends on developmental history, but it is also influenced by the personal experiences of the individual. The aforementioned stress hormones support the memorization of stressful events in short-term stress situations. Accordingly, a reoccurring stressor is judged differently than a new stressor, and the reaction process therefore varies. Under chronic stress, this mechanism damages the neurons and leads to atrophy in the hippocampus (McEwen 2002). Other regions of the brain play major roles in the stress adaptation process as well, like the amygdala and the prefrontal cortex (McEwen and Gianaros 2010), but their exploration would go beyond the scope of this thesis.

The two main aspects deciding if an external stressor is acting as stimulant or health hazard are duration of the stressful period and intensity. While short-term or low intensity stress (eustress) enhances the immune system and other bodily functions and does not have to result in long-term health consequences, persistent or high peak stress (distress) slows down protective functions, disturbs the endocrinological and metabolic systems of the body and increases susceptibility for several diseases (Dhabhar 2000). McEwen (1998) presented an overview of adapting systems (Table 1), their function and mal-function, under normal and abnormal stress situations.

Table 1 illustrates the complexity of reactions and the interactivity of all systems in order to prepare the body for a new challenge, triggered by a stressor. It also underlines the protective mechanism of all systems under short-term stress,

which can potentially turn into health risks during persisting stressful situations. Among all the systems affected by stress, the cardiovascular system appears to be especially susceptible for stress-induced reactions (McEwen and Gianaros 2010), which makes intensive stress an important CVD risk factor.

**Table 1**: Stress-triggered interactive adaptive systems in the human body and their short-term (acute) and long-term (chronic) response

| Interacting Adaptive Systems of the Body | | |
|---|---|---|
| System | Actute Response to Challenge | Problems Associated with Chronic Activity or Inactivity |
| Cardiovascular | Maintaining erect posture (avoiding "black out") | Hypertension, potential for stroke, MI |
| | Physical exertion | |
| Metabolic | Activating and maintaining energy reserves, including energy supply to the brain | Obesity, diabetes, atherosclerosis |
| Immune | Response to pathogens | Inflammatory, autoimmune disorders |
| | Surveillance for tumors | Immunosupression |
| Brain, Central Nervous System | Learning, memory | Neuronal atrophy, death of nerve cells |
| | Neuroendocrine and autonomic regulation | |

Table from McEwen, 1998, Stress, Adaptation, and Disease – Allostasis and Allostatic Load, p.38

In order for this reaction to be harmful, the stress situation does not have to be life-threatening. In early periods of human history, the *fight or flight* mechanism was literally meant to save individuals who came under sudden attacks by an external force. In modern society, many sources of stress originate from lifestyle and social structures (Karasek 1979), with many, if not the majority, of those stressors being of psychosocial nature.

Studies have identified two possible pathways – direct and indirect – from stress to disease (László et al 2010; Stansfeld and Marmot 2002). A direct impact is characterized by changes in the metabolic, inflammatory and homeostatic systems through the dysfunction of the autonomic-nervous-system and of the hypothalamus-pituitary-adrenal-axis, provoked by the stressor (László et al 2010). Higher inflammatory markers were observed in individuals with high economic stress, measured as low annual income (Gémes, Ahnve and Janszky

2008). Elevated inflammation and thrombotic functions support the progress of coronary atherosclerosis (Hansson 2005; Libby 2002).

Indirect impact provokes disturbances in health-related behavior, such as changes in nutritional habits, level of physical activity, or use and abuse of substances such as tobacco, alcohol and other drugs. Dallman et al (2003) present an example for indirect effects of stress with their analysis of so called *comfort food*. Food falling into this category is often rich in carbohydrates, fat, salt and sugar and has a stress releasing effect on the short run. If the consumption of comfort food becomes a regular reaction to stress, persisting stress can lead to medical problems as diabetes or obesity.

However, there is still some debate regarding the actual relationship between stress and dietary choices. On the one hand, some studies have suggested that acute stress can increase the demand for energy intake as well (Vanstrien et al 1986; Weidner et al 1996). An explanation for higher food intake could be the higher levels of energy needed to cope with stressful situations. On the other hand, studies have also found discouraged energy intake under circumstances of acute stress. One explanation for this result is that acute stress can suppress upper gastrointestinal mobility (O'Brien et al 1987). Another supporting theory is that stress-induced activation of the sympathetic nervous system, which relocates blood from the digestive system to muscles and releases endorphins (adrenaline) into the endocrine system, can suppress the digestion process (Gue and Bueno 1996). The alert status of the body under stress takes parts of the necessary energy from fat resources of the body instead of relying on energy production from the digestion process, which additionally discourages new food intake (Friedman 1995).

Wardle and Gibson (2002) point out that, whatever the direction of change in dietary behavior, the effect of stress is strongly dependent on the nature of the stressor. Furthermore, the authors emphasize the importance of susceptibility for different stressors in every individual (human and non-human). They summarized multiple pathways for stressors to induce a higher energy intake (Figure 3).

**Figure 3**: Proposed pathways through which stress may lead to a final common effect of eating energy-dense snack foods

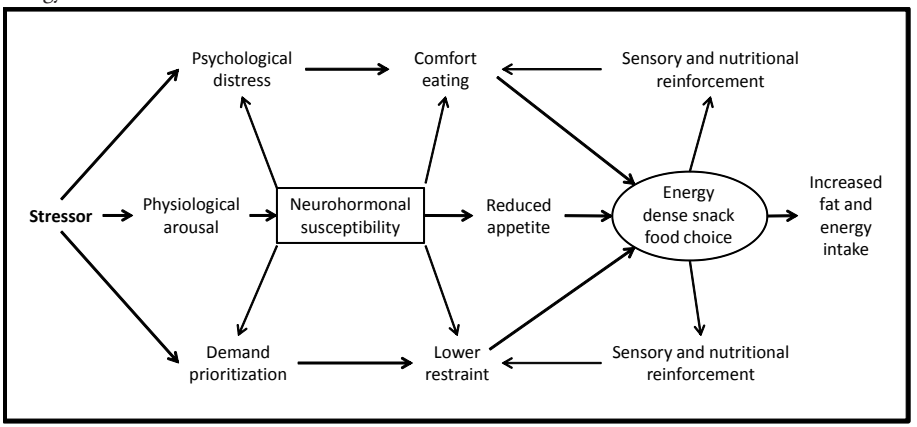



Figure based on Wardle & Gibson: "Impact of stress on diet: processes and implications" In: Stress and the Heart. Eds. Stansfeld, S. & Marmot, M. (2002), page 141

While the changes in weight caused by acute stress or critical life events are temporary, at least for women (Deurenberg and Hautvast 1989), long-term stress is suspected to influence the physiology of the individual permanently. As mentioned above, higher body weight, measured as BMI, Hip-Waist-Ratio or share of body fat, increases the risk for CVD.

The sources and impact mechanisms of stress are manifold as described above. Stress in the form of job strain is the focus of paper I of this thesis. Economic hardship and potential psychosocial stress are investigated in paper II. Papers III and IV include the concept of stress in the form of labor market integration of immigrants and intermarriage.

### 1.2.3. Socioeconomic factors

Socioeconomic status (SES) is an umbrella term for several characteristics of individuals which indicate their place inside the hierarchy of society and define their connection to other individuals within that society. This position in the hierarchy can be measured by several indicators such as level of education, occupational position, religious membership, ethnic minority, material resources and social influence or political power. As a matter of prestige and economic well-being, it is associated with important health impacts. Several large-scale investigations with the specific aim to analyze social determinants and their health have been conducted, such as the Framingham Heart Study (Dawber and Kannel

1958) the Whitehall Study II and the co called "Black report" (Department of Health and Social Security 1980).

Studies using data from the above-mentioned or other sources have found a universal social gradient, which implies that members of higher SES are usually in better health than individuals affiliated with lower social classes (Cabrera et al 2001; Pocock et al 1987; Rosvall et al 2006). The effect is universal in the sense that it has been found across countries (Mackenbach et al 1997) and among different health outcomes. Socioeconomic differences in mortality were found to continue beyond the retirement age in several European countries (Huisman et al 2004).

There is an ongoing discussion about the validity of the two main explanatory theories for this social gradient. Researchers disagree as to whether the correlation between SES and health is caused by selection or if having higher SES results in better health through material and non-material resources (Marmot 1989).

If observed health differences between the social classes are due to selection, then health problems early in life could prevent individuals from achieving higher aims in education, professional life and consequently higher SES (Falkstedt and Hemmingsson 2011). Consequently, the association would be the result of reverse causality, with early life health affecting both SES and later life health (Bengtsson and Mineau 2009; Palloni et al 2009). However, the association between SES and health could still be in the expected direction if the different health outcomes in early life are the consequence of lower SES affiliation of the family the individual was born into or raised with (Lynch, Kaplan and Salonen 1997a).

The second pathway of explanation is that a healthy lifestyle is more affordable and more important due to achievements in life – education, occupation and material security. While individuals with lower SES may spend more time, effort and resources on basic needs such as food, housing and clothing, persons with greater access to material resources can afford to spend more money on health-promoting activities as sports, counseling and healthy food. The individual's SES can therefore indirectly influence the incidence of CVD via environmental conditions, lifestyle and medical risk factors (Lynch et al 2006).

Regardless of disagreements concerning the origins of the social gradient, most studies agree on the existence of health variations caused by socioeconomic differences. This social gradient is not only observable in the risk for CHD, but also in its risk factors, which supports the indirect effect of SES on CVD via lifestyle and medical risk factors. The National Health Report of Sweden (Socialstyrelsen 2009) showed that the distribution of BMI varies depending on educational degree. Those individuals with the highest education showed a higher concentration in the normal BMI range, while lower educated persons were more frequently in the BMI ranges which are defined as overweight and

obese. A similar finding was presented by Deboosere, Gadeyne and Van Oyen (2009), demonstrating significantly lower life expectancy among individuals with lower education compared with highly educated persons.

Regarding education, the assumed correlation with health outcomes operates through cognitive skills, acquired during education, which support the individual in making health decisions concerning lifestyle and also to achieve better occupational and economic positions (Lager 2011). The different factors related to socioeconomic performance seem to be interrelated strongly with each other, producing a broad and complex picture of causes and consequences (Anderson and Armstead 1995; Lahelma et al 2004). For this reason, the following section is focusing on a summary of the empirical results regarding correlations between SES, CVD, and CVD risk factors.

An extensive overview of SES and its effects on CHD is provided by Marmot and Bartley (2002). A direct inverse effect of SES on the risk of onset and the course of CVD has been found by many other researchers (Baigi et al 2002; Hallqvist et al 1998; Smith, Shipley and Rose 1990b). Furthermore, the social gradient was found to influence atherosclerosis (Diez-Roux et al 1995) and CVD risk factors such as obesity (Brunner et al 1997; Kaplan and Keil 1993) and smoking (Bucher and Ragland 1995). Education and occupation are two very important components of SES, and they also act as mediators for later CVD risk differences (Droomers et al 1998; Smith et al 1998; Wing et al 1992). For the change of the correlation between SES and CVD over time the Black report (Department of Health and Social Security 1980) suggested three possible sources. First the effect of SES on health and second the effect of health on SES could have changed over time. Third, changes in the SES composition (selective social mobility, changes in the share of female labor participation and immigrant work force) are a potential origin of changes of the relationship between SES and CVD risk.

SES is operationalized in various forms in the papers in this thesis; educational degree, occupational class affiliation and income are all used as covariates to explain differences in CVD risk. Their specific impact mechanisms and usage are explained in the sections below.

### *Occupation*

Together with education and income, occupation is one of the most frequent forms of operationalization of SES. These three SES characteristics are strongly inter-correlated with each other (Geyer and Peter 2000; Winkleby et al 1992). The formal education acquired as a child and young adult will be the basis for the choice of occupation and success in the labor market, including occupational

class affiliation and income. Occupational class affiliation and its impact on health is the focus of the paper III in this thesis.

Besides affiliation to a specific occupation, labor market attachment and working conditions can have health effects as well. The field of occupational health suggests, along with other findings, that occupational stress can take many forms and therefore is a potential risk factor for various diseases, including CVD.

Furthermore, unemployment has been shown to be correlated with poor health outcomes (Carson et al 2009). These detrimental health outcomes can result from a general insecurity of employment, insomuch as the threat of losing employment places psychosocial pressure on individuals and their dependents. Unstable employment situations may also have detrimental consequences for life outside the work place, because income, future plans and material resources are affected. However, even under stable conditions, the working environment and job characteristics can produce stress of psychological and physiological nature (Bortkiewicz et al 2010).

As discussed above, the cardiovascular system is very susceptible to stress induced changes. Therefore, many studies have analyzed the effects of occupational stress and job strain on CVD and CVD risk factors. Johnson et al (1989) found a much slower cardiovascular aging structure among persons with low strain in their work environment. Furthermore, they concluded that people with higher occupational stress have higher probabilities of dying of heart disease. In the review of occupational stress and cardiovascular diseases by Byrne and Espnes (2007), the authors found a strong relationship in the literature between blood pressure, the development of hypertension, and work-related stress.

Over the years, several models were developed to measure job strain. Two of the most influential ones are the Effort Reward Imbalance model (ERI) and the Demand Control Model (DCM). The first was introduced by Johannes Siegrist (1992, 1996) and focuses on the imbalance of efforts spent on tasks and the psychological or material reward received for them. The key aspect here is reciprocity, which brings the efforts and the expected and received compensation in balance. Studies have shown that non-reciprocal imbalances in working environments are only tolerated by persons without alternatives or on a short-term basis, linked to either a highly competitive market or with a suggested postponed reward (Kuper et al 2002; Siegrist 2005). If the expected compensation is not realized, a higher risk for various CVDs, CVD risk factors, and other negative health outcomes were observed (Head et al 2007; Hintsanen et al 2007; Nakata, Takahashi and Irie 2011; Niedhammer et al 2004; Peter et al 1998; Siegrist 2011).

The second job strain measurement – DCM – chooses another approach to measure the effect of job characteristics on health issues. Because the DCM

model is used in the paper I of this thesis, it will be explained in a bit more detail in the following section. However, both models have shown their validation and common results in several comparisons (Bosma et al 1998; Siegrist and Rödel 2006).

*Demand-Control Model*

In 1979, Robert Karasek published the first of many contributions regarding the job demand and job control model and its relationship to psychological stress. His idea was to combine two dimensions which have produced contradictory results when analyzed separately. The two-dimensional job characteristics were supposed to overcome the problem that, for certain persons or professions, higher demands have motivating and challenging effects, while for others an increase in job demands produces psychological stress, which in turn can trigger CVD. When defining the dimensions of job demand and job control as being either *low* or *high*, both dimensions together will result in four possible combinations. The overview of those combinations is given in figure 4.

**Figure 4**: Karasek's job strain model

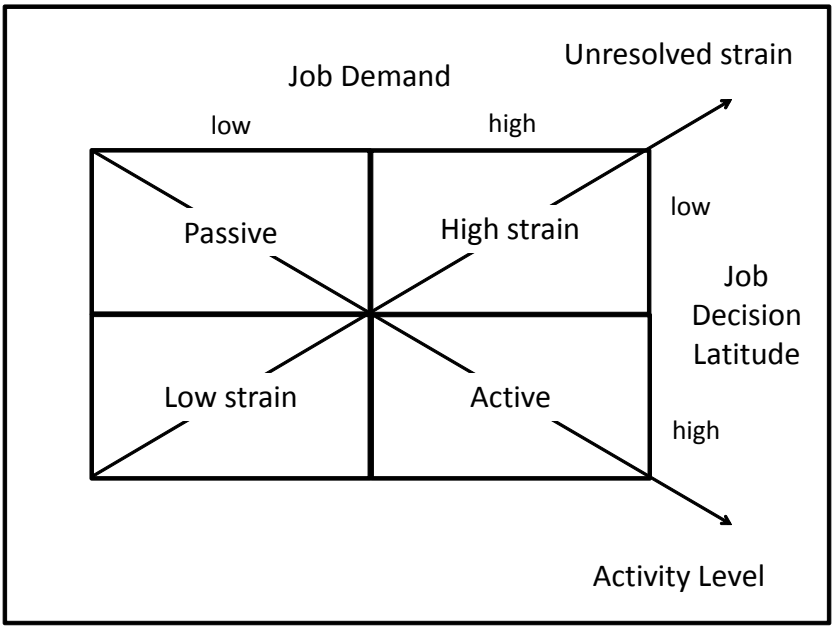



Figure after Karasek, R.A. 1979. "Job demands, job Decision Latitude, and Mental strain. Implications for Job Redesign."

If job demand and job control (originally termed by Karasek as *job decision latitude*) have the same value, the combination is called balanced. In case demands and control are both low, the job is characterized as *passive*, neither challenging, nor producing strain. The opposite case, high demand combined with high control is called *active* and identifies a challenging job with enough freedom to master the high load of demands. An increase of both dimensions without leaving the balanced state would appear along the *activity level*.

In cases where the dimensions are unbalanced, job demand and job control take different values. The combination of high job control with low job demands was called by Karasek *low strain* because the combination is supposed to be the most advantageous of all job strain categories. These relaxed working conditions are expected to produce the least amount of stress for the individual. The opposite case, having high job demands in combination with low job control, is called *high strain* and was observed to produce the most stress of all possible job strain categories.

The separation of the single dimensions involved in this classification does not have to be bipolar. Karasek and other authors have experimented with a threefold division (low, medium, high) or quartiles of the job dimensions (Collins, Karasek and Costas 2005; Kuper and Marmot 2003). However, the two-by-two dimension combination has remained the most widespread version of the job demand and control model (Belkic et al 2004; László et al 2010; Schnall, Landsbergis and Baker 1994).

The dimensions of demand and control needed for this type of analysis are generated from a battery of questions regarding job characteristics and occupational opportunities provided by the workplace. Individuals are asked, on the one hand, to evaluate whether their job requires high skills, if they have the opportunity to learn new things during their work, if their work is creative, if the job allows freedom, if the individual is free to make independent decisions, if the individual participates in group decisions and if he/she has a say regarding job characteristics. On the other hand, individuals must also state if their job involves repetitive tasks, if they have to work very quickly or hard, if they have a large workload to master, if they lack time to finish specific tasks, if their job requires excessive work, if they have enough time for their job and if they are confronted with conflicting situations. Since the introduction of this battery of questions, the psychosocial job strain questionnaire has been used in many surveys and has occasionally undergone changes according to time and place of the research study such as the introduction of a question about shift-work (Alfredsson, Karasek and Theorell 1982b) or social support at work (André-Petersson et al 2007). However, the main results for the job strain group are impressively consistent.

With help of factor analysis, the answers are first separated into the demand or control dimensions and then they are given the appropriate weight inside the dimensions, according to the sample characteristics. Using the factor loadings of each single item, every individual is given a score for their specific demand and control dimensions. In the two-by-two dimensions version of the model the complete range of each dimension is divided into *low* and *high* at the median. Each person is then placed in one of the four job strain categories according to their values of the job demand and job control dimensions (see figure 4).

The four groups are used to investigate whether there are different health outcomes for the different combinations of job demand and job control. The original study by Karasek (1979) analyzed the effect of job strain on psychological phenomena such as exhaustion, depression, satisfaction with job and life in general.

The job strain model established itself very quickly in the field of stress- related health research, and it has been used regularly to predict and explain differences in CVD (Karasek et al 1988; Reed et al 1989; Kuper and Marmot 2003) and CVD risk factors (Lallukka et al 2009; Siegrist and Rödel 2006; Theorell and Karasek 1996; Tsutsumi et al 1999).

In 1994 (Schnall et al) and 2004 (Belkic et al) there were attempts at summarizing and comparing studies using the job strain model to explain CVD differences. Both meta-analyses confirmed three things. First, the overall consistency of the results, regardless of the differences in measurement and study design. Second, both research reviews summarized the universal finding of an increased CVD risk under higher job strain conditions. Third, connecting to findings which already appeared in the original article by Karasek, there is evidence of the dominance of the job control dimension (job decision latitude) over the job demand dimension, regarding the impact magnitude on CVD. As mentioned before, higher work demands could serve as an additional challenge and motivation or they could produce higher stress loads. A higher magnitude of control over the work tasks has a positive effect on psychological and physiological health outcomes in general.

Job strain, measured as categories of the demand and control model, and its health impact, is the focus of paper I of this thesis.

### *Income and Financial Resources*

Another important aspect of SES is income and other financial resources available to the individual. Income and other assets, or more precisely the lack of these, were observed to be associated with different health outcomes, including diseases of the cardiovascular system. Because income and its potential impact on IHD

is the focus of paper II, a separate section is dedicated to the connection of individual economic resources and CVD.

Financial hardship, due to poor labor market performance, unemployment or other individual factors can provoke rather strong stress reactions, which in turn can increase the risk for CVD (László et al 2010; Lynch, Kaplan and Shema 1997b; Rios and Zautra 2011). The negative effects of economic insecurity appear to have especially harmful effects on the human body, causing health problems in the long-term (Catalano 1991; Dooley and Catalano 1980). Lack of planning and organization under economic hardship is the driving force which, if prolonged, can cause serious psychological stress and, consequently, physical problems. Unemployment in itself is not a health hazard, but the financial consequences and the frustration of long-term failure to find an employment places pressure on the individual (Kozieł et al 2010). Therefore, many studies find worse health outcomes, especially for CVD, for unemployed individuals (Jin, Shah and Svoboda 1997; Stronks et al 1997; Weber and Lehnert 1997). The effects of unemployment, however, may differ depending on the larger macro environment, as shown by Henriksson et al (2003). Their study showed decreased CVD differences between employed and unemployed during times of high unemployed rates in Sweden. Being unemployed, while others have work seems to produce more psychological pressure than being unemployed in a period of general recession (Ruhm 2000).

Income and financial resources not only affect the individual, but also other members of the same household, especially those who are economically dependent upon the unemployed individual. Thus, household income, as well as individual income has been shown to affect an individual's risk for CVD (Andersen et al 2003; Herrin et al 2000; Kinnunen and Feldt 2004).

Considering income as an indicator of SES, many studies have observed a health gradient within the income distribution. Given that the absolute amount of available fortune could be the root of all evil, many explanations of health differences can be found in the field of neo-materialistic theory (Blane, Bartley and Smith 1997; Macintyre et al 1998). As mentioned in the section on lifestyle factors, maintaining good health can be costly, considering the price of healthy food, physical activity (cost of sports club membership or equipment and time) and other health-promoting items and activities. Additionally, income is assumed to be related to the quality of housing, which is, on the one hand, related to health hazards of the housing itself (damp or moist walls, insufficient protection against forces of the weather, contamination with chemical or biological substances) and, on the other hand, to external stressors (neighborhood safety, noise and air pollution). Individuals at the lower end of the income distribution are assumed to

struggle more to satisfy all basic needs required for maintaining a healthy lifestyle (Lynch et al 1997a).

Another aspect related to income is the access to health services and knowledge. Strong effects from the level of absolute income on health outcomes are often found in countries like the US, where economic status is more directly linked to access to health insurance and medical services (Yngwe et al 2001). Due to Sweden's universal health care system, access to health treatment should be comparatively weakly linked to the individual's income.

Furthermore, a branch of research focuses on inequality of income distribution and the resulting health differences for people at opposite ends of the distribution (Wilkinson 1996). Smaller health differences were found globally in societies with a more equal income distribution (Babones 2008; Osler et al 2003), with Sweden falling into this group (Zandvakili and Gustafsson 1998). The explanation for these small differences connects the feeling of fairness and equality within a society with psychological well-being, while still acknowledging the material importance of a more equal distribution of resources among the population.

The focus on absolute income fails to take into account the implications of widely varying income distributions across different occupations. Whether an achieved income is to be interpreted as high or low depends strongly on the characteristics of the individual such as age, sex and characteristics of SES. According to the relative deprivation hypothesis, another key dimension to consider is the comparison of an individual's performance to the performance of others with similar backgrounds. Underlining the importance of an individual's relative rather than absolute position, Eibner et al (2005) suggest that a substantial part of the decline in mortality that is associated with an increase in income is due to a *relative deprivation effect*. Individuals who earn less although having comparable characteristics, can feel relatively deprived, which can lead to psychological stress. On the other side, individuals who economically outperform those with similar demographic and human capital assets could benefit from relative satisfaction, providing the reassurance that their investment in human capital and career was successful. Indications of the existence of a physiological response to relative status are provided by studies on nonhuman primates, essentially confirming similar mechanisms as the response to absolute status. Sapolsky (2005) outlines a number of responses to prolonged stress in form of change of position in the social hierarchy, which increase the risk for CVD risk factors, such as hypertension, elevated heart rate and increased circulating levels of lipids and cholesterol among the exposed primates.

Achieved levels of SES, measured as income and financial resources, are highly correlated with demographic characteristics such as sex and age as well as levels of human capital (Geyer and Peter 2000; Huijts, Eikemo and Skalicka

2010). Higher educational investments are in general rewarded with higher wage levels, to compensate for the lost time and financial resources which were spent outside the labor market to gain those extra skills. Because higher education and occupational status are also directly related to healthy lifestyles, an indirect effect on CVD risk can be found using income as mediator (Winkleby et al 1992).

The number of ways to define and measure income have led to the argument that the result of income on health will vary depending on the measurement used (Fritzell, Nermo and Lundberg 2004). On the one hand, income can be seen as a long-term measurement of material and financial security. On the other hand, an instant effect can be assumed to be the result of major changes in the income situation (e.g. loss of employment). Despite the possibilities of both effects, the effect of stress resulting from detrimental economic situations is assumed to be stronger the longer the period of economic hardship last (Benzeval and Judge 2001).

This thesis uses income, measured as inflation adjusted pre-tax individual income, as an SES indicator in paper II, III and IV. In paper II the impact of income on the onset of IHD is the focus of the study. The database used for paper I does not contain any information on income, and thus income could not be taken into consideration for the analysis of job strain effects on the onset of CHD in this paper.

### 1.2.4. Demographic Factors

As seen in figure 1, demographic factors take a special place in the set of covariates for the risk of CVD. Demographic factors such as age, sex and ethnic background (country of birth) cannot be altered by lifestyle, SES factors or the incidence of CVD. This does not, however, mean that the effect of age or sex does not change depending on time, place or policies. The demographic factors have a direct impact on the risk of CVD and influence all of the CVD risk factors which have been discussed so far in this thesis. Besides age, sex and ethnic background, demographic factors include marital status and intermarriage in this thesis.

*Age and Sex*

CVDs are, if not pre-determined by genetic heritage, a phenomenon mainly experienced in older ages. This is due to the biological aging process of organs, systems and cells in the human body. As described earlier, insufficient blood supply to the heart and brain can be caused by weakening of the heart muscle itself, which can be a natural byproduct of aging. Furthermore, many of the risk

factors of CVD are also dependent on the age of the individual. The development of hypertension, Type II diabetes and atherosclerosis are all long-term processes. Due to an unhealthy lifestyle, these processes can be accelerated, but these problems do not generally appear in young ages.

Unfortunately, the spread of unhealthy dietary habits and physical inactivity with overweight and obesity as results among children and adolescents is pushing the age limit for the onset of these CVD risk factors further down (Friedemann et al 2012). As described in the previous section, SES and, in particular, attained income are age-dependent variables. On average, a person will begin their economic career with a low wage which will increase over time, dependent on human capital accumulation and experience. Because SES is expressed in terms of occupation, education and economic performance, the individual SES should be dynamic over the life course.

**Figure 5**: Deaths with Acute Myocardial Infarct (AMI) as underlying cause of death among women by age group and year in Sweden between the years 1987-2010 in absolute numbers.

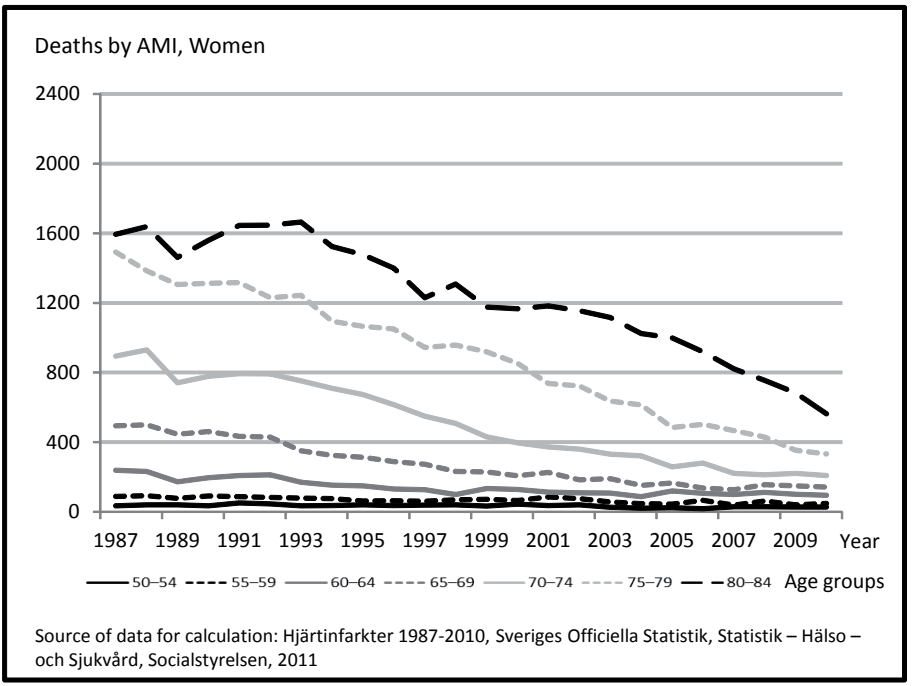
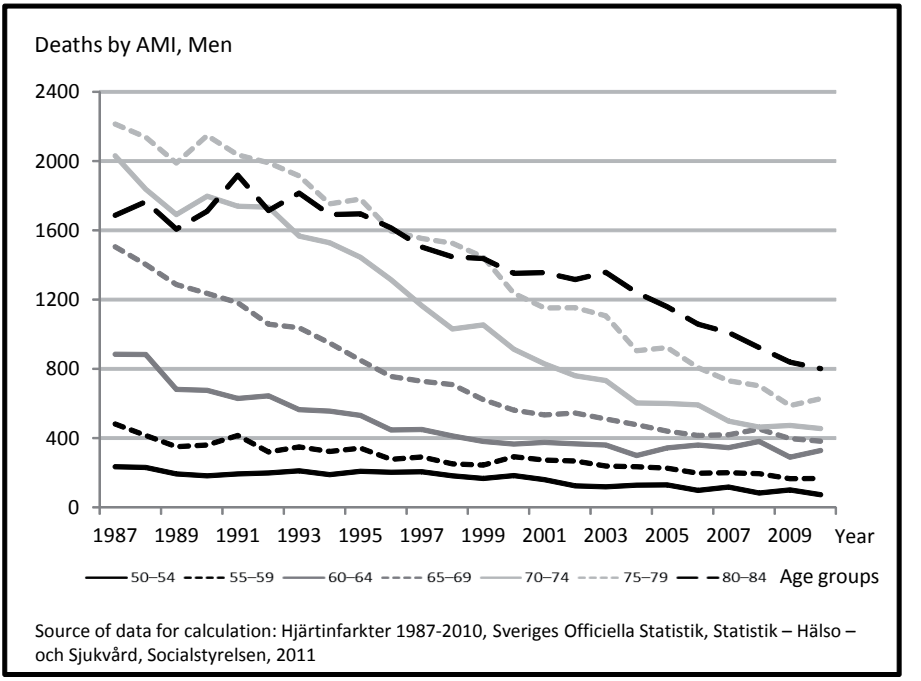



Figure 5 and figure 6 show mortality caused by acute myocardial infarction (AMI) for women and men in different age groups in Sweden during recent decades. The most apparent characteristic is the increasing risk with higher age. Furthermore, the decreasing trend in mortality due to AMI in the last two

decades is very explicit. This is partly due to better prevention, but mainly to improved medical opportunities for intervention in case of an incidence. The trend in morbidity is still rising, since an increasing share of individuals survives CVD incidences, although often with resultant major or minor health problems. Given the importance of age for the risk of onset and progress of CVD, all papers in this thesis take the age of the individual into account. In the first paper, age is used as the duration variable of the survival analysis. The other three papers use age as continuous covariate.

**Figure 6**: Deaths with Acute Myocardial Infarct (AMI) as underlying cause of death among men by age group and year in Sweden between the years 1987-2010 in absolute numbers.




Source of data for calculation: Hjärtinfarkter 1987-2010, Sveriges Officiella Statistik, Statistik – Hälso – och Sjukvård, Socialstyrelsen, 2011

A large difference in the prevalence and incidence of CVD is also observed between the sexes. In developed countries, women tend to experience only around one-third of the CVD incidence of men in a given age group, but roughly as many women die of heart conditions as men, and women show poorer prognosis for recovery once the events has occurred in the short- and long-term perspective (Coppieters, Collart and Levêque 2012). Since women experience CVD events on average later in life than men, they lose fewer years to the disease (WHO 2011a), however studies have also shown that medical treatment and cardiac rehabilitation are assigned differently to men and women. While this

might be the outcome of the different incidence rates between men and women, it is assumed that sex-biased treatment could influence the health condition and recovery of women, compared with men (De Feo et al 2012).

While part of the sex difference is due to different lifestyle characteristics of men and women, a portion is also assumed to be determined by genetic preconditions and gender differences in the endocrine system (Kajantie and Phillips 2006). The exposure to sexual hormones is assumed to play an important role in the different CVD risks between men and women. One of the leading explanations is derived from an anthropological-biological viewpoint. The exposure of a fetus to high doses of stress hormones during pregnancy could alter the metabolic programming of the child and therefore provoke later life disadvantage, including higher cardiovascular susceptibility. To avoid harming the fetus, the endocrine system of the women limits the stress hormones to a minimum. Those stress hormones, which are useful for acute stress response, are assumed to have long-term effects on the cardiovascular system and because men are exposed in higher doses over the life-time, they would experience a higher propensity for cardiovascular incidents (Kajantie 2008). In return, women were found to react more sensitively to similar stressors than men, which could be a result of the lack of these stress hormones (Hallman et al 2001).

Regarding the important role of the endocrine system for stress adaptation and women's advantage for later CVD development, it can be further assumed that men and women display differences in stress-coping strategies (Torkelson and Muhonen 2004). For the majority of stressful events, men prefer the rational and detached coping strategies, while women were found to prefer emotional and avoidance coping strategies (Eaton and Bradley 2008). Women are assumed to take over a larger share of family and household tasks and responsibilities, which increases the risk for fatigue (Nelson and Burke 2002). Due to the extra burden of these tasks, which include time-constraints, women possibly struggle more to achieve higher SES, measured as occupation or income, due to their expected additional role as care-giver. Thus, regardless of the woman's work situation, they appear more likely to experience a feeling of work-overload due to their domestic duties, potentially translating into experiencing psychological stress which can trigger CVD or several of the medical CVD risk factors.

The main source of daily stress varies between men and women. Women more often report stress due to problems in the family and health-related issues, while men report that their profession and financial concerns are more often the reason for daily stress (Matud 2004).

As mentioned above, there are sex differences in the prevalence and influence of CVD risk factors. The different distribution of education and occupation among men and women leads to variations in income attainment and differences in SES

(Nikiforov and Mamaev 1998). The existence of gender-biased recruitment and promotion processes where women are disadvantaged are suggested as explanation for sex differences in the labor market as well (Baumgartner and Schneider 2010). Although most developed countries aim for gender-neutral treatment in the labor market and social life, the traditional view of male and female roles persists and leads to sex-differences in the labor market of men and women (Arber and Ginn 1995; Moss-Racusin et al 2012).

A large difference between men and women can also be found in the prevalence of CVD risk factors (Ferrari et al 2012). The different distribution of health-related behaviors such as diet, physical exercise and smoking among men and women contributes to the sex differences observed in CVD mortality and morbidity (Brunner et al 1993).

Given the sex differences in the endocrine system, lifestyle factors and SES distribution, a separation of men and woman is an essential part of any research on CVD. Regarding the lower incidence of CVD among women, especially in the younger ages, many studies fail to provide statistically significant conclusions about the effect on CVD among women, due to the lack of statistical power offered by the few CVD cases experienced by women in the samples. Therefore, many studies on cardiovascular outcomes were conducted on purely male samples, especially when focusing on the economically active population (Belkic et al 2004).

All analyses in this thesis are performed using separate models for men and women to account for the medical and social aspects of the sex differences.

### *Marital status and Intermarriage*

Research has shown that marriage has a rather universal and beneficial effect on health (Burman and Margolin 1992; Molloy et al 2009). While widowed and divorced people are assumed to have higher CVD risk due to the changes in lifestyle following divorce or the death of a partner (Koskenvuo et al 1980; Nef et al 2010), single people also face higher risks due to the potential lack of support and shared resources. The beneficial effect of marriage was found to be stronger for men than for women, which is partly due to the differential distribution of employment and domestic tasks between partners (Johnson et al 2000). The weakening of the importance of marital status as a general pattern in society has an effect on the strength of the findings for the impact of marriage on health. In societies where cohabitation is fully established as an alternative union form, many people in the group of *unmarried* will enjoy the benefits of shared resources and responsibilities as well, without being officially classified as

married. This blending of the terms *unmarried* and *single* could cause problems in the interpretation of results.

Furthermore, studies have found that the effects for individuals not only depend on whether they are married or have a partner, but also on the characteristics of the partner. Intermarriage (here in the sense of partnership with a person of a different country of origin) is a potential health impact factor, especially in societies with a large share of foreign-born individuals.

It is important to keep in mind that intermarriage is strongly dependent on several aspects. First, there is the aspect of opportunity in the marriage market. For immigrants, the partner choice will be limited if they consider only a person from the same origin, since their co-ethnics account for only a small share of the entire marriage market in the host country. Only a sufficient number of people in an appropriate age range, sex and SES, available in a certain geographical range, can ensure a partner from the same origin (Cretser 1999). This situation explains why the rates of intermarriage are higher among small groups of immigrants. Second, the aspect of cultural background is important (Coleman 1994). The greater the cultural distance between home and host country, the higher the chances of resistance to a partnership with a native person, due to important values such as religion, traditions and beliefs, which are more difficult to combine in a mixed couple (Lucassen and Laarman 2009). In fact, the family and co-ethnic social network of the individual presents an important benchmark for the decisions of union formation (Huschek 2011).

Primary socialization through the family will teach the individual the values and beliefs they stand for. When the ethnic community is very traditional, the social education of the person will be as well. When the family is rather integrated in the native society, the individual will have more experience with, and access to, the native culture. Immigrant children, who grow up with parents that interact with a more ethnically diverse group of people have higher chances of entering an inter-ethnical relationship (Clark-Ibanez and Felmlee 2004).

Intermarriage is widely seen as a positive sign of successful integration among immigrants (Dribe and Lundh 2008), with economic benefits for immigrants who are married to a native partner being a consistent finding in the literature (Behtoui 2010; Meng and Gregory 2005). There are two types of explanations for this phenomenon. First, the effect could result from assortative partner choice, making the better integrated and adapted immigrants more attractive in the native marriage market (Chiswick and Houseworth 2011; Furtado 2012). Secondly, immigrants who decided to marry a native could benefit from the country-specific human and social capital of their native spouses to achieve better integration in the labor market and society as a whole.

There is no wider discussion of potential health effects of intermarriage, neither for natives nor for immigrants, in the literature. Given the strong effects of intermarriage on aspects of SES, health differences between immigrants with partners from their own origin and immigrants with partners from the host country can be assumed. Paper IV of this thesis analyses this possible health impact originating from intermarriage.

*Ethnic background*

The topic of ethnic background and migration was introduced with the description of effects due to intermarriage. While explaining the possible pathways of health effects it is important to understand the migration history of the population under investigation. The following section will first provide an overview of approaches connecting ethnic background, migration and health, and subsequently provide a brief description of the Swedish migration history.

In line with the research on SES, a consistent difference in CVD between foreigners and natives has been found worldwide (Dassanayake et al 2011; Williams et al 2009), with similar effects also being found in Sweden (Sundquist and Johansson 1997; Gadd et al 2003; Gadd et al 2005). This health difference is not only observable in CVD morbidity and mortality, but also in its risk factors as many studies have demonstrated (Dotevall et al 2000; Koochek et al 2008; Tomson and Åberg 1994; Vandenheede et al 2012; Wändell et al 2004). Further, country of birth has been shown to have an effect on survival following the onset of CVD (Hedlund et al 2008).

The origin of these health differences can be explained by several approaches. First, it is important to distinguish between the various purposes of migration. Refugees leave their home country under very different circumstances than labor migrants or persons migrating for family reunification reasons. A health disadvantage could emerge from the immediate psychological and physiological stress which characterized the situation from which the refugee escaped. Labor migrants, on the other hand, are assumed to have a health advantage, compared with their countrymen who stayed in the country of origin (Hedlund et al 2007), since the initial health conditions of this group have to match the labor demands of the receiving country. Therefore, labor migrants comprise a selected group of healthy people in the prime working ages, since this gives them an advantage in the host country labor market. This type of selection among labor migrants, coupled with the fact that unhealthy individuals do not tend to migrate, regardless of their reasons, leads to what is known as the *healthy migrant effect* (Wingate and Alexander 2006).

Secondly, the migration process itself can be a source of stress for the migrating person. Along with the burden of preparation, a new social, cultural, and climatic environment awaits the immigrant in the host country. Since integration and acculturation are processes which take a certain amount of time, the length of stay in the host country can influence the health status of the immigrant (Albin et al 2005; Alfredsson, Ahlbom and Theorell 1982a). The longer a migrant is exposed to the host country environment and conditions, the higher the probability that their health and health behavior converge to the native pattern. For immigrants from countries with worse health, that would mean an enhancement of health. For other persons, converging to native health behavior may alter their health for the worse, if the host society offers inferior conditions (Nakanishi et al 2004; Sundquist and Winkleby 2000).

Several characteristics of immigrants can either accelerate or slow down their integration process. Immigrants from a cultural background which is similar to the host country generally find a faster way to adapt to the host country's conditions than individuals whose traditional and cultural systems are very different. Furthermore, the age of the immigrant at arrival (Saraiva Leão et al 2009) plays an important role for the integration process (Pudaric 2002; Pudaric, Sundquist and Johansson 2000; Torres 2006). While young immigrants adapt faster to the new environment, older migrants who have lived with the traditions and culture of their country of origin for a longer time might experience greater problems of acculturation.

When the individual experiences integration problems (e.g. distance to the host culture, exclusion from the labor market) the subjective health perception of immigrants could decrease with time (Lindström 2001; Wiking, Johansson and Sundquist 2004). These problems can result in psychological stress which can have effects on the risk for CVD (Pudaric, Sundquist and Johansson 2003) or other diseases (Johansson et al 1997). Economic integration is strongly dependent on the immigrant being able to find employment in the host country. Being economically independent and having regular contact with the host society will increase integration intentions and opportunities. Different occupational opportunities vary greatly among migrant groups (Helgertz 2010; Rooth and Ekberg 2006), mainly due to cultural distance and differences in human capital as well as problems of transferability of some occupational skills and knowledge.

The reason for differences in mortality and morbidity between immigrants and the native population appears to be multifactorial. Selection among countries of departure as well as selection with regard to the host countries are important aspects. Furthermore, differences in lifestyle factors, including dietary preferences, smoking habits, physical exercise and social integration, are strong covariates

for the observed differences between morbidity and mortality (Deboosere and Gadeyne 2005).

All the evidence of ethnic differences in stress levels and CVD risk make several issues essential for a native-foreign health comparison. On the one hand, individual characteristics such as education, employment, occupation, age and other aspects of SES have to be taken into account to avoid biased interpretation of the results. These characteristics are assumed to vary between the natives and different immigrant groups, and therefore differences in health arising from these disparities could be incorrectly interpreted as ethnic differences.

On the other hand, in the case of Sweden the group of immigrants contains persons from very different countries and cultural backgrounds. Because differences in SES are also existent between various groups of immigrants, the specific migration background can help to identify health problems. A simple comparison of natives to the total share of all immigrants might be biased by the composition of the immigrant population. A separation of origins, at least at the country level, is suggested to avoid that bias.

In a country like Sweden, where one-quarter of the population is foreign-born or has at least one parent who was born outside the Sweden, an impact from ethnic diversity is undoubtedly present. A careful analysis of ethnic differences is therefore an important part of health studies conducted in Sweden.

Ethnic background and distribution of immigrants is a relevant point of interest throughout the thesis, and is the focus of paper III and paper IV. In paper II, ethnic background has been included in the models as one covariate among many. The share of foreign-born individuals in the database used in the first paper has proven to be selective and not representative, necessitating a restriction of the sample to only Swedish born persons.

In the papers where country of birth is used, the distribution of nationalities made it necessary to group some of the ethnic backgrounds while others could be analyzed separately. The choice of groups for country of origin is strongly correlated with the migration history of Sweden. To better understand why specific countries of origin are chosen to be analyzed and others not, the following section provides an overview of the important migration waves in Sweden during the second half of the 20$^{th}$ century. Given the facts that CVD is a disease of older ages and Sweden is a relatively new immigration country, beginning only in the second half of the 20$^{th}$ century, only first generation immigrants are considered for investigation in this thesis. While there are information available on the children and grandchildren of immigrants, these individuals have not reached the age range at risk for CVD in sufficient numbers.

*Migration History of Sweden*

Until the Second World War, Sweden's population movements were marked by net outmigration to the New World. With its neutral position during the Second World War, Sweden became, for the first time in centuries, a net immigration country, as refugees from neighboring countries and later from the rest of Europe found their way to the country. While some of these refugees stayed in Sweden, many of these war-time immigrants left Sweden to either return to their home countries or move on to a third country (Scott 1999).

Not being involved in active combat left the Swedish economy and industry largely intact in the years following the war, while large parts of Europe struggled for their economic survival, having faced massive destruction. This economic head-start allowed the Swedish economy to bloom, and made it attractive to workers in Europe. In the early 1950s, the need for manual labor exceeded the supply of Swedish workers and, due to pressure from industry and the manufacturing lobbies, the Swedish government changed the country's rather restrictive immigration policies. To satisfy these labor needs, an immigration stock of 10,000 individuals was recommended by the government (Bengtsson, Lundh and Scott 2005).

To meet this aim two main changes were introduced in that time. First the Common Nordic Labor Market Agreement was established, which removed the demand for labor and residence permits for citizens of Denmark, Norway, Iceland and Finland, in 1954.

The second change, introduced in the 1950s, consisted of active labor recruitment outside the Nordic Countries. The new liberal immigration policy encouraged workers from all over Europe to come to Sweden and search for work, without having to apply for a work visa prior their arrival. Consequently, a large number of immigrants arrived in Sweden using tourist visas, applying for work visas only after finding employment. Swedish recruitment offices were also established in important sending countries to attract workers still living in their home country and send them, with employment contracts in hand, to Sweden. These actions opened Sweden to a large flow of migrants, first mainly from Germany, Austria and Italy, and later from the regions of former Yugoslavia, Greece and Turkey as well (Bengtsson et al 2005). Over the entire recruitment period, which ended in the late 1960s, over half a million immigrant workers and their family members arrived to Sweden. This is a massive inflow over a short period, especially given that Sweden had only eight million inhabitants at that time (Lundh and Ohlsson 1994).

The fact that the immigration was of such a large magnitude and occurred in a rather short time span attracted the attention of the strong centralized trade

union lobby. They accused industry of using foreign labor to hold back wage development, and the political pressure reached such levels that the government was forced to change the immigration policy again. The earlier liberal policy was rescinded, and prospective labor migrants now had to report evidence of an employment offer, housing arrangements, and a valid work permit (granted in coordination with the trade unions) before they were granted permission to migrate (Helgertz 2010). Although this change occurred in the late 1960s, the immediate effect was buffered by unexpected high streams of refugees from South and East Europe coming at the same time, as well as continued high demand for labor which could only be met through continuation of the active recruitment (Scott 1999). The true effects of this change were only felt later, in 1972, when nearly all work permits from non-Nordic residents were rejected on the recommendation for the Swedish Trade Union Confederation. This move by the unions effectively halted all non-Nordic labor migration overnight.

After this period of labor recruitment, refugees and asylum seekers became the leading group of non-Nordic immigrants. While accounting for only a small share of immigrants from the late 1950s until the end of the 1960s, their amount increased dramatically from the early 1970s. Initially, immigrants from Eastern and Southern Europe provided the main share of refugees; later on, global crises expanded the geographical range of immigrants in Sweden. Political struggles in Latin America brought the first large-scale waves of non-European immigrants to Sweden in the 1970s (Chile and El Salvador). These were followed by refugees from Vietnam and Ethiopia, Iran and Iraq in the 1980s, and refugees from the former Yugoslavian countries in the 1990s.

Sweden entered the European Union in 1995, again opening immigration policies to EU citizens. Despite new possibilities for labor migration, the main stream of immigrants was still characterized by non-European refugees and family reunification migrants.

While the early labor migrants found relatively easy entrance into the Swedish labor force, later immigrations streams have faced greater difficulties achieving successful integration. In recent decades, the Swedish government has been confronted with a very diverse composition of immigrants, with many of them arriving from geographically and culturally distant countries. Due to a general lack of country-specific human capital, these immigrants face problems integrating in the Swedish labor market (Helgertz 2010).

By the end of 2009, 14.3% of all inhabitants in Sweden were born outside Sweden, a number which rises to 18.9% when also accounting for individuals who were been born in Sweden but had both parents born abroad (Statistics Sweden, 2011). This large foreign influence in the Swedish population must have a substantial impact not only for integration policies and labor market dynamics,

but also for the healthcare system and public health intervention programs. This makes it essential to account for differences in ethnic background in any analyses of CHD or IHD.

## 1.3. Data

The empirical part of this thesis is based on two separate databases. For the first paper, data from the Malmö Diet and Cancer Study (MDCS) is used, because it provides the necessary information on individual job strain. The second database is the Swedish Longitudinal Immigrant database (SLI), which is used for the final three studies. Sample selection, according to the specific aims of every paper, is explained in detail in the data and method section of each paper. Provided below is a general overview about purpose and construction of the two databases to facilitate an understanding of the choices concerning variable construction, method and statistical strategy.

### 1.3.1. Malmö Diet and Cancer Study (MDCS)

The data resource for the first paper in this thesis is the Malmö Diet and Cancer Study (MDCS). Malmö is the third largest city in Sweden, with about a quarter of a million inhabitants at the time of data collection. The initial target population of the study were all men and women born between 1926 and 1945 and living in Malmö in 1991-1992 (n= 53,491). However, the study population was extended by widening the birth year range to 1923-1950 three years later, in 1995. Accordingly, data collection took place throughout the whole period between 1991 and 1996. As a result, about 17,000 women and 11,000 men took part in this prospective study, achieving an overall participation rate of 40.6% (n= 28,098). All potential participants were contacted via post and gave their written consent before the actual interviews were conducted (Lindström, 2000). Some participants showed up spontaneously at the Study Centre to receive information or make an appointment for an interview. Participants had to have sufficient Swedish writing and reading skills in order to participate.

The database was designed to study dietary habits and the onset and course of cancer diseases by the International Agency for Research on Cancer (IARC), Lyon, France The Swedish Cancer Society and representatives of the Faculty of Medicine, Lund University, Sweden (Berglund et al 1993). Extensive

questionnaires containing issues like dietary preferences and food consumption were conducted in personal interviews. During the interviews, anthropometric variables (blood pressure, height, weight, lean body mass and body fat mass) were recorded and blood samples drawn by clinical personnel. The samples were collected and stored in a bio-bank for analysis and later comparison (Pero et al 1998). This information has been linked to a general cross-sectional questionnaire containing information about (i) heredity; (ii) demographic and socio-economic variables; (iii) social network and support; (iv) previous and current occupation; (v) physical activity; (vi) smoking; (vii) alcohol consumption and (viii) previous and current diseases, symptoms and medication.

After a period of approximately two weeks, participants were re-invited to meet a nutritionist and discuss their data concerning nutrition and the first results of their brief medical examination. In cases of unadjusted health problems, such as hypertension, the participants were referred to their local practitioner.

After the phase of initial data collection, the individuals were linked to official registers such as the registry at the Department of Pathology at the Malmö General Hospital and the Regional and National Cancer Registries. Medical incidences were coded according to the ICD9/ICD10 catalog.

Subsamples of the MDCS have been invited to participate in further studies, with a focus on medical issues other than cancer (Hanson et al 1997). One of those studies aimed at the investigation of carotid artery disease and the progress of atherosclerosis, which made the inclusion of cardiovascular follow-up information necessary (Hlebowicz et al 2011).

For the MDCS, the medical data was updated every two years to keep track of health developments of the participating individuals. The version used for this thesis contains the medical information of every individual until 31 December 2006. The construction of the study lends itself to analysis in the form of survival analysis with cross-sectional baseline data. The data contains information concerning demographic background, socioeconomic situation, and detailed information about the job situation and the balance of demands and control at the work place, which made the use of the demand-control model possible. The data construction of the MDCS study perfectly matched the requirements of the research aim of paper I.

Information about the history of heart disease for the participants is obtained from register data using the ICD coding system. For this study aggregated information are available defined as CVD in general and divided in the subsections – CHDs and cerebrovascular diseases. Therefore, the first empirical paper in this thesis investigates the onset of CHD.

However, one drawback of the database is the selectivity among the share of the foreign-born in Malmö's population. Many of the foreign-born potential

participants had to be excluded from data collection due to insufficient language skills. The remaining immigrants who took part in the questionnaire showed significantly different characteristics than the overall foreign population in Malmö. Since the immigrant share is highly selective and does not represent the share of foreign-born in the overall population, it was deemed prudent to exclude all foreign-born from the analysis of the first paper in this thesis.

### 1.3.2. Swedish Longitudinal Immigrant Database (SLI)

The three remaining papers in this thesis are based on the Swedish Longitudinal Immigrant database (SLI), administered at the Centre for Economic Demography, Lund University, Sweden. The SLI contains information from the Swedish Tax Register (inkomst och förmögenhetsregister), the censuses of 1970 until 1990, the Total Population Register (RTB), and information from the Hospital Discharge Register (patientregistret). The Swedish personal ID number was used to combine information from the different sources for the same individual. Initially, a small sample of immigrants under the age of 65 representing the 16 largest immigrant-sending countries was randomly selected from the census in 1970. Immigrants were then added to the sample from the immigration registers, to account for the flow of migrants between 1971 and 2001. To allow comparison with natives, the study included stratified random samples of natives drawn from the censuses in 1970 and 1980. A further extension of the data was achieved by linking the sampled individuals (Swedish and foreign) with their parents, children, grandchildren and spouses in so far as these individuals were residing in Sweden at any point in time between the years 1968 and 2001. The end result of this procedure is a dataset which contains roughly 550,000 unique individuals. (Helgertz 2010).

The various registers contribute with different regularity and precision to this database. While tax register data is available on a yearly basis continuously from 1968 until 2001, census data only provides information every five years starting in 1970 until 1990. The source of critical importance for this thesis is the Hospital Discharge Register (patientregistret). The medical information is only available with the onset of the Hospital Discharge Register in 1987 and the absence of earlier coverage is very important because the onset of CVD is the focus of analysis. Without information concerning potential events which occurred before the beginning of data collection, it is difficult to ascertain whether the observed events are the first of their kind or simply a continuation of an unobserved, but ongoing, disease. In order to investigate the onset of IHD

rather than the progress of the disease, all three papers use a proxy for the first experienced IHD event.

Pre-analysis using the SLI revealed that over 95% of all repeated IHD events occurred less than five years from the previous event. In order to maximize the chances that only the initial IHD cases are taken into account for the analysis, individuals have to be observed for five years without experiencing any IHD event before they are considered for analysis. Strict selection on individuals who have been observed for five consecutive years without experiencing an IHD event increases the certainty of analyzing only first IHD events.

The papers II – IV are based on the SLI and complete ICD codes are available for this thesis, making it easy to identify specific diagnoses, in this case IHD. For the analysis ICD 9 codes (410-414) and ICD 10 codes (I20-I24) were used to identify cases of IHD. In the same way atherosclerosis is defined as ICD 9 codes (440) and ICD 10 codes (I70); hypertension as ICD 9 codes (401-405) and ICD 10 codes (I10-I15); diabetes as ICD 9 codes (250) and ICD 10 codes (E10-E14).

## 1.4. Summary of Chapters

### 1.4.1. Paper I: The impact of job strain and occupational class on the incidence of coronary heart disease

For several decades, the analysis of causes and consequences of cardiovascular disease (CVD) have caught the attention of epidemiological studies, because the extensive care and medical attention required by patients affected by CVD account for a substantial share of overall healthcare expenditure. Among many medical conditions, behavioral characteristics play an important role for the individual risk of onset and progression of CVD. Work-related conditions and psychosocial stress were found repeatedly to influence individual CHD risk.

This study analyzes job strain in combination with occupational position and its influence on coronary heart disease (CHD), a major group of CVD. Using the MDCS database with a baseline sample of 17,000 women and 11,000 men, Cox proportional hazard models display the effect of the different job strain groups in combination with occupational class. Individuals with a history of cardiovascular disease are excluded from the sample to avoid feedback effects from the consequences of former cardio events. All models are sex-stratified to account for the sex differences in incidence of IHD and sex difference among occupational groups and work tasks.

Job strain is generated following the demand and control model suggested by Karasek. The model combines subjective perception of various work-related conditions and divides them into the two groups of job demand and job control. The combination of either low or high levels of demand and control results into four categories of job strain with different levels of work-related stress (high strain, low strain, passive and active). To account for differences between the sexes in the labor market, the job strain categories are generated in sex-separated samples, producing four job strain categories of equal size for both, men and women.

Results show increased risk for men and women in high strain positions, confirming findings of earlier studies of job strain. The integration of typical CHD risk factors such as smoking, BMI and genetic precondition for cardiovascular diseases diminish the effects from the job strain categories substantially. In the full model including all potential CHD risk factors, no significant difference among the job strain categories can be observed for women. Size and direction of the estimates suggests that job strain has an impact on the onset of CHD for men and women.

In general, results for men and women vary quite considerably, which supports the sex-separated sample choice. The occupation-stratified models show that the effect of job strain takes very different pathways for the various occupational positions. The size and direction of the effects of the job strain categories vary among occupational groups and among the sexes. However, most coefficient estimates do not achieve statistical significance, and therefore the some of the results remain inconclusive. Additional models investigating the correlation between job strain and the prevalence of several of the CHD risk factors do, however, demonstrate that job strain also affects the onset of CHD through indirect pathways.

In conclusion this study confirms previous findings establishing high job strain as a risk factor for the incidence of CHD. These effects decrease and lose significance for women when the model is extended with classical CHD risk factors. Furthermore, the study emphasizes the importance of sex-separated analysis on work-related job strain models and their effect on CVD in general and CHD in particular. The main finding is that job strain has very different impact depending on the individual's occupational position and gender, following direct as well as indirect pathways.

### 1.4.2. Paper II: Economic Stress in the Short and Long Term and the Onset of Ischemic Heart Disease

The main motivation for this study is the link repeatedly found in empirical research between income and the main subgroup of cardiovascular disease, IHD. The correlation between higher income and better health has been explained with greater purchasing power for health-promoting goods and services.

This study exploits a sample from the longitudinal SLI database consisting of about 50,000 Swedish men and women who are observed during the years 1992-2001. The study examines the short, medium, and long-term effects of income on the risk of experiencing the first IHD event using logistic random effect regressions, taking several IHD impact factors into account.

The emphasis is on the analysis of the onset of IHD. The focus on the onset rather than IHD events of any order cancels out the feedback effect of IHD on economic performance for later IHD cases. The degenerative nature of the disease increases the risk for another IHD event as well as reducing the economic output for individuals after experiencing an initial event. Retrospective information on the CVD history of the individuals was not available for this study. In order to only analyze the onset of IHD, all individuals have to survive a five year period without an IHD event before they were accepted in the final sample, to increase the probability that the observed IHD event is the first of this kind.

More specifically, the study examines whether absolute income or relative income deprivation is more important to an individual's health. Absolute income is measured as logged and lagged pre-tax income from wage on a yearly basis. A positive correlation between absolute income and lower IHD risks would support the neo-materialistic theory, assuming that higher income increases the possibility to purchase more health-related goods and services, which promote the maintenance of a healthy lifestyle. In this paper, relative deprivation is measured as the share of the individual's earnings compared to the average earnings of people who share the same age, gender and educational level and type. Therefore, the relative income measurement evaluates economic performance given the demographic characteristics of the individual and their previous investment in human capital. The hypothesis is that individuals who performed worse compared to others with whom they share the same characteristics experience a feeling of deprivation and therefore are exposed to higher levels of psychosocial stress, which, in turn, can increase the risk for IHD. On the other hand, individuals who perform better than people with similar characteristics might experience relative satisfaction and a feeling of reassurance about earlier investments for human capital. This could have a health-promoting psychosocial effect.

Unlike previous studies, the results show no consistent health benefit from higher income – either relative or absolute. The general conclusion of the article questions the existence of a causal link between the individual's absolute and relative income attainment and the risk for IHD onset.

However, disregarding the lack of statistical significance, a few patterns emerge which potentially could imply the existence of a weak link. Among men, higher absolute or relative income appears to be associated with a lowered risk of experiencing an IHD event. Furthermore, this effect seems to increase slightly with the duration of the observed high income. The opposite situation applies to women, where higher income in the medium or long-term is associated with an increased risk for IHD on average. This controversial result could be explained by women facing greater challenges in achieving and maintaining an elevated position in the labor market.

### 1.4.3. Paper III: Labor Migration and Ischemic Heart Disease in Sweden

Given the increasing share of international migrants in Sweden, public health studies have focused more on migration and its long-term consequences for health. The health differences between natives and the foreign-born, that have been shown to be a global phenomenon, are found as well in Sweden. The source of these differences is an ongoing discussion.

Today, the Swedish population has a substantial share of foreign-born individuals, showing great variety regarding factors like country of origin, level of integration, purpose of migration, age and sex distribution as well as human capital and labor force participation.

This large heterogeneity among the foreign-born increases the difficulties for identification and analysis of the origin of health differences between foreign-born and natives, as well as between the foreign-born from different origins. Some of these characteristics are not observed, and therefore add bias to the results. The unique history of migration policies in Sweden provides an opportunity to analyze a rather homogeneous group of immigrants, at least in terms of purpose of immigration and length of stay. This study tests the hypothesis that a large portion of the observed health differences between immigrants and natives originates from a failure to adequately account for heterogeneity within the migrant population.

For this analysis the SLI database is used. This study analyzes foreign-born individuals who arrived as labor migrants to Sweden between the years 1955 and 1970. Given this restriction, the sample shows less variation in terms of length of

stay in Sweden, purpose of migration and labor force participation. Furthermore, the active recruitment of foreign labor during this time provides large numbers of individuals from a small range of countries, so that country-specific analysis is possible.

The reasonably homogeneous sample of foreigners is then compared to natives regarding the onset of IHD during an observation period between 1992 and 2001. The hypothesis is that this group will show fewer health differences compared to natives than the immigrant population as a whole, due to their common purpose of entry and length of stay. A second hypothesis is that any remaining health differences may be the result of the different distribution of foreign-born and natives in the labor market. To test this hypothesis, occupation-stratified models are estimated, which compare only foreign-born and natives who have worked in the same occupation, and therefore shared occupation-specific health effects.

Results show that most of the groups of foreign-born do not display significant differences to natives after the sample is restricted to labor migrants. The exception is found in immigrants from Finland, who show consistently increased risk for IHD for both, men and women. This corresponds with earlier research on the health condition of Finnish immigrants in Sweden. In the occupationally stratified models, no differences could be identified. However, the health disadvantage of Finnish immigrants remained as well in the occupationally-stratified models, especially in the blue-collar group, where most labor migrants in 1970 were concentrated.

In conclusion, the results provide an indication that the heterogeneity among immigrants, especially regarding purpose of migration and labor force participation, is a primary source of observed health differences. It also found that immigrants from Finland are a specific group among all foreign-born, with a health disadvantage regarding IHD which is not caused by a disadvantaged distribution in the Swedish labor market.

### 1.4.4. Paper IV: Intermarriage and its Impact Paths to Ischemic Heart Disease

By 2009, 18.6% of the Swedish population was born abroad or had both parents born outside Sweden. This large share of foreigners has an impact on the labor market, community life, and public health. Given the exposure to different health regimes, the foreign-born and natives are assumed to show different health patterns.

These health differences should be more apparent in the prevalence of diseases which are strongly connected to behavior and lifestyle, such as CVD. The direct

cost of treatment, medication and hospitalization for CVD patients accounts for about 8% of the total healthcare expenditure in Sweden. Furthermore, CVD is highly correlated with the demographic background of the individual and health behaviors. A remarkably consistent finding is a reduced risk for the onset and course of IHD, the main subgroup of CVD, among married people. Due to shared assets and responsibilities, a stable relationship can act as a buffer for stress and as source for material, financial and emotional support.

Given a population with a large share of immigrants, marriage as a CVD impact factor can be studied in a new dimension – intermarriage. Marriage between foreign-born and natives has been the focus of many studies concerned with the integration process of immigrants in the host society, with intermarriage being found to be an indicator for integration and economic success among foreigners.

This is the first study, to the best of the author's knowledge, which investigates the potential health impact of intermarriage for the foreign-born as well as for the natives. More precisely, this study analyzes if there are different IHD patterns depending on the country of birth of the individual as well as the origin of the corresponding partner. The hypothesis is that foreign-born individuals will show lower risk for IHD if they are married to a native. On the one hand, this beneficial health effect could result from assortative matching among immigrants with attributes seen as favorable in the Swedish marriage market. On the other hand, a native partner could improve the integration process of the foreign individual, providing knowledge and access to country-specific health-related assets.

For the analysis, a sample of the SLI database is used containing all native and foreign individuals who could be connected to a partner in the same database (married people and cohabitating people with common children). About 44,000 men and 39,000 women are analyzed in sex-stratified models, including classical IHD risk factors to isolate the effect of intermarriage on the onset of IHD. Intermarriage is defined as partnership with a person from a different country of birth. In the case of non-Swedes that could take two different outcomes – Swedish partner and foreign partner of a different origin.

Models measuring the direct effect of intermarriage on the onset of IHD as well as the effect of intermarriage on some major IHD risk factors (high education, income, hypertension and diabetes) are performed. Additionally, a path-analysis was added to demonstrate the direct and indirect impact pathways of intermarriage on the onset of IHD. Finally the country-specific effect of intermarriage for six groups of foreigners is analyzed separately to take the country of origin into account.

The results show that intermarriage has a positive impact for foreign men married to a Swedish partner. They showed significantly lower IHD rates compared with foreign men married to partners of the same origin. The effects

of intermarriage on IHD impact factors showed significant results for both male and female foreigners and natives. The finding that intermarriage is correlated with higher income for foreign men and women, while it is correlated with lower income for Swedish men and women, indicates the validity for the social exchange theory. The theory assumed that economic success of the foreigner can be exchanged for country-specific social and human capital of less economically successful natives.

The results could not verify whether the effect of intermarriage on health for foreigners is due to more rapid integration and more intensive contact to the host society or if there was a health selection among those engaging in intermarriage. The country-specific models did not verify significant differences between the groups of country of birth. However, the variation between the coefficients of the country group gives some indication that intermarriage has different effects for individuals of various origins.

## 1.5. Conclusion

This thesis analyzes the onset of CVD and potential socioeconomic impact factors in contemporary Sweden. The study was motivated by the importance of CVD as the main cause of death in developed countries, and the resulting economic burden for the individual and public healthcare, given the large expenditures spend on the treatment and prevention of the disease. The overall aim of public health interventions for CVD focuses on the prevention of the disease and conditions which promote the disease. In order to achieve this aim, the mechanisms of CVD have to be analyzed and more influential factors must be identified.

The analyses in this thesis contribute to this aim by investigating a variety of origins of health differences in order to detect the beneficial or detrimental effects of socioeconomic aspects at the individual level. Acquiring knowledge concerning potential risk factors is the foundation for reducing health inequalities and health differences at the national level.

The theoretical portion of this thesis provided the necessary background information to understand CVDs, their medical mechanisms, and potential socioeconomic risk factors. It was demonstrated that the various risk factors are strongly interrelated with each other, forming a complex structure of beneficial and detrimental conditions for every individual. On the one hand, the complexity of risk factors provides obstacles for the appropriate analyses of direct and indirect

impact pathways. On the other hand, a variety of important risk factors allows for many opportunities for public health interventions to approach the prevention of CVD.

The attempt was made to present major contributions of earlier scientific analysis in this field of research. The summary of the explored fields of cardiovascular research leads to the identification of missing information and research needed on the topic which forms the basis for the empirical analyses in this thesis. Much research has been done in the last century on the causes and consequences of heart disease. Socioeconomic impact factors are the focus of many studies, and several of the universal findings are presented in the theoretical chapter of this thesis. Throughout the single papers, forming the empirical part of this thesis, some of those established theories and models in social epidemiology are used and challenged to achieve new understanding of the impact of socioeconomic characteristics on the onset of CVD.

The first conclusion drawn from the results is that SES is a very multifaceted concept. Throughout the different papers, SES was operationalized as occupational class, job strain categories (paper I), education, income attainment (paper II) or ethnic background (paper III –IV). On the one hand, individual SES cannot be adequately delineated as a single value or category. Often a variety of characteristics have impacts on the individual's SES. On the other hand, many of the forms of operationalization of SES are intercorrelated with each other, so that additional covariates could add bias to a model, instead of greater precision. A careful choice of SES characteristics and covariates is therefore essential to identifying the potential impact on health in general, and CVD in particular.

Another important finding is the importance of interdisciplinarity for research into SES differences and its impact on CVD. Being a complex medical condition, CVD needs to be analyzed taking certain medical mechanisms into account. The risk factors of CVD can be interpreted from many perspectives – sociology, anthropology, demography, health policies, medicine and economics. The reason for rising or decreasing incidence rates of various heart diseases and risk factors can be found in individual preferences, group affiliations, societal conditions, or environmental changes. The combination of disciplines seems rather essential in order to draw a complete picture of SES structures and their impact pathways to the onset of CVD.

Rather than simply testing established theories on empirical data, this thesis attempts to contribute to the existing body of literature with modifications of models or new theories. The demand and control model is well known and widely used in the field of occupational health. This study proposed a sex-differentiated generation of the job strain categories to be able to account for differences between men and women with respect to labor market participation,

sex discrimination, stress coping strategies and other SES-related differences (paper I). The social gradient among income categories could not be confirmed using a set of thorough measurements of individual income (paper II). The initially observed health differences between many immigrant groups and natives in Sweden could be reduced to applying to only one specific immigrant group through the use of a very homogenous group of immigrants and natives (paper III). The established health advantage of married individuals and the health impact from ethnic background have been combined to test the original hypothesis that intermarriage is correlated with CVD risk (paper IV).

The development of the methodological and theoretical novelties in this thesis was only possible thanks to the major contributions already existing in the field of social epidemiology. However, the results of the various papers demonstrated that there is reason to continuously question and challenge existing knowledge and theories.

The overall achievement of the thesis can be described as a broadening of our knowledge of socioeconomic impact factors and impact mechanisms on the risk for the onset of CVD. The use and elaboration of established theories and models in social epidemiology emphasizes the ongoing necessity for a critical view of former research. The use of the existing body of information and the enhancement of this knowledge are the aim of social epidemiology and a key feature of successful public health interventions to increase overall health and diminish health inequality and health differences.

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Chapter 2

# The impact of job strain and occupational class on the incidence of coronary heart disease

## 2.1. Abstract

Diseases of the heart and the blood circulation system are the leading cause of death in developed countries. Sweden is no exception, with 40% of all deaths due to cardiovascular disease (CVD). For several decades, epidemiologic studies have analyzed the causes and consequences of CVD. Among the many contributing factors, work-related psychosocial stress and occupational affiliation play important roles in an individual's risk for the onset and progression of CVD. This study analyzes job strain in combination with occupational position and their influence on Coronary Heart Disease (CHD), a major subgroup of CVD. Using the Malmö Diet and Cancer Study (MDCS) with a baseline sample of 8,092 women and 4,656 men, Cox-proportional hazard models demonstrate the effects of different job strain groups in combination with occupational class. Job strain is analyzed based on sex-separated samples using the job strain model developed by Karasek.

The results show an increased CHD risk for high strain positions in men and women, which is in line with previous research. The integration of CHD risk factors such as BMI, smoking and socioeconomic position diminishes the effects of the job strain categories. The results for men and women differ substantially, confirming the sex-separated approach. The models, which are stratified by occupational class, display that job strain works differently in various occupational positions. Although many of the analyzed health differences are not statistically significant, the size and direction of the effects of the job strain categories indicate that job strain has an effect on the onset of CHD, varying among occupational groups and between sexes. Additional models investigating the correlation between job strain and the prevalence of CHD risk factors show that job strain also affects CHD through indirect pathways.

In conclusion, this study confirms the detrimental effects of high job strain on CHD risk. Furthermore, it emphasizes the importance of using a sex-separated analysis for work-related job strain models and cardiovascular studies. The study concludes that job strain affiliation is correlated with differences in CHD onset following direct and indirect pathways.

## 2.2. Introduction

Despite modern medical achievements and health education, CVD is still the main cause of death in developed countries, and it is on the rise in many developing countries (WHO 2011a, 2011b). The causes and consequences of CVD have been the focus of public health studies for many years. One important subgroup of CVD is coronary heart diseases (CHD). Among other contributors, work-related conditions have been identified as a potential CHD risk factor. This study analyzes psychosocial work stress in combination with occupational class and the impact of this combination on the risk for onset of CHD in a section of the Swedish population.

In recent decades, increased emphasis has been placed on the negative effects of working conditions and psychosocial stress on physiological well-being. The shift in modern economies from primarily manufacturing to service sector jobs has exposed a large section of the working population to psychosocial rather than physiological demands. While the health effects of physically demanding work are usually manifested in problems of the locomotive system or in injuries due to accidents, the health effects of psychosocial threats are less obvious. However, they are no less harmful for the individual and the economy.

Several models have contributed to the scientific discussion of work-related stress (Stavroula and Houdmont 2010). Two of the most successful models are Siegrist's (1996) effort and reward imbalance model (ERI) and Karasek's job strain model (1979). Both models have been independently validated, and their predictive power for different groups of CVDs has been demonstrated (Bosma et al 1998). The current study uses the job strain model because the results for the job strain categories are extremely consistent.

The combination of high demands and low control (including the components of freedom and opportunity to use skills) has repeatedly been found to be a risk factor for cardiovascular diseases and other illnesses (Belkic et al 2004; Schnall et al 1994; Theorell and Karasek 1996). This consistency is impressive, considering that labor market structures and working conditions have varied in recent decades.

Analyzing work-related stress provokes the idea that different combinations of work-related conditions might be connected with individual occupational status. Individuals in similar occupations might share many work-related health conditions. Some occupations might put more strain on the employees than others. The question remains: how strongly are occupational class and job strain correlated with each other? This study investigates the hypothesis that the effects of the different job strain categories vary among the various occupational classes. It is hypothesized that the different combinations of demand and control have different effects depending on socioeconomic status (SES), which is measured by occupational class.

The study analyzes the effect of sex-difference on CHD risk, taking the job strain categories into account. To reduce the sex-difference bias, which is common in the labor market and in occupational distribution, the job strain categories are generated in sex-separated samples.

Controlling for typical cardio risk factors is essential to the analysis of CHD risk. The effect of job strain is weaker once the other CHD risk factors are included in the models. To address this effect, this current study additionally tests the hypothesis that job strain not only influences CHD directly but potentially as well indirectly via correlations with various CHD risk factors.

## 2.3. Theoretical Background

Modern economies in developed countries share many principles. One of them is undoubtedly the concentration of the workforce in jobs of paid labor. According to Marmot et al (2002), there are four reasons for the centrality of work in the modern society, which illustrate the close connection of work and health. First, most individuals depend on wages from their job as their main income source. The amount and distribution of income is assumed to influence individual health because healthy living styles and medical services can been seen as investments. Secondly, the education of children and young adults is aimed at achieving the skills and knowledge needed for an occupation. Educational achievements and occupational status are a major part of the identity of an individual outside of its family. Further, higher education has been correlated with better health outcomes and a lower CHD risk.

Third, work partially defines an individual's SES inside the society and influences non-work-related aspects of SES, such as leisure time activities and health behavior. Many studies have found a social gradient in health outcomes,

including CHD (Kaplan and Keil 1993; Marmot et al 1991). Fourth, due to the importance of work in the society, the working population spends a considerable amount of time at their work places, exposed to the work-related health conditions of each occupation. Therefore, the work environment can be a source of health impacts, both beneficial and detrimental in nature.

In addition to the obvious occupational hazards of specific occupations, such as mine workers, policemen and construction workers, various occupations show health differences originating from different sources. Specific work conditions are identified as risk factors for cardiovascular disease such as shift work (Alfredsson et al 1982b; Vyas et al 2012), work-related life events (Möller et al 2005) and social isolation or support at the workplace (André-Petersson et al 2007; Johnson et al 1989). Several studies that analyzed the data collected from the Whitehall studies, one of the first and largest surveys for social determinants of health, found a health gradient among the different hierarchical levels of British civil servants (Marmot et al 1991; Smith et al 1990b). A similar gradient has also been found in other populations (Dalstra et al 2002; Vahtera et al 1999; Wilkinson 1997). Part of the explanation for this health gradient is the varying levels of psychosocial strain at the different hierarchical levels.

Work can be the source of wealth, status and self-esteem, in addition to a potential source of health threats. A much slower cardiovascular aging structure was found among people with low strain in the work environment (Johnson et al 1989). A review of occupational stress and CHD verified a strong relationship between blood pressure and hypertension as result of work-related stress (Byrne and Espnes 2007).

Over the last decades, more precise methods have been developed to measure job strain. The one used for this study is the job strain model developed by Karasek (1979). The author noticed that both the level of demand and the level of control at work influenced individual health outcomes. However, the reported effects of these work dimensions varied in the literature. As a consequence, Karasek included both dimensions and analyzed the effect of one dimension, dependent on the value of the other one. This analysis demonstrated that, of the two work dimensions, the level of control showed stronger effects on health (Karasek et al 1988; Tsutsumi et al 1999). While an increase of control is generally beneficial for health outcomes, rising demands can have beneficial as well as detrimental effects on an individual's health. Higher demands can also be perceived as challenging and motivational, and they can have a beneficial effect on health, especially in combination with high levels of control.

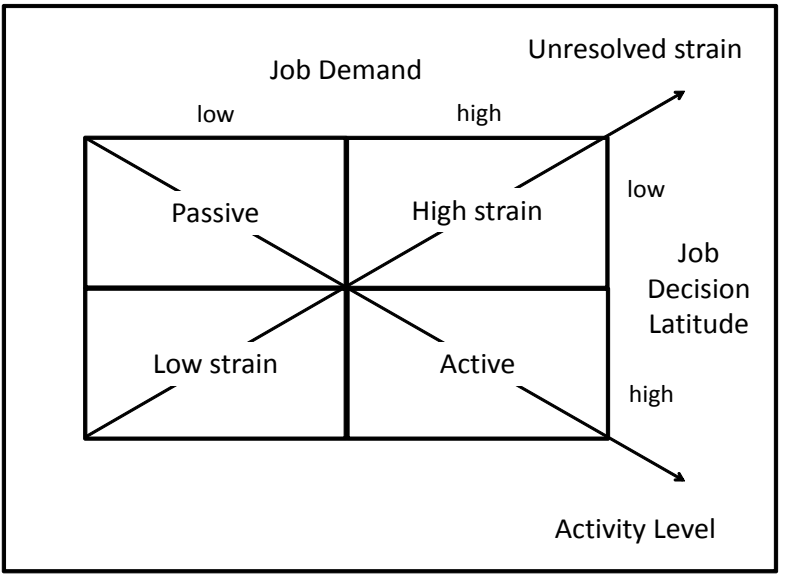
For research purposes, both variables, job demand and job control, are generated from a battery of questions concerning the individual's work conditions. Karasek (1979) used a psychosocial questionnaire of 14 statements regarding working

conditions, including opportunities and freedoms in the work procedures of the current job. Since its first use, this battery of questions has been used in many surveys and undergone occasional changes according to the time and place of the research study, e.g., the inclusion of shift-work (Alfredsson et al 1982b); the Swedish job strain questionnaire (László et al 2010); and the inclusion of social support at work (André-Petersson et al 2007).

With the help of factor analysis, the underlying patterns of the 14 single statements are reduced to two factors. The first factor is based on responses to the following statements: *high skill level*, *learn more things*, *creative*, *allows freedom*, *make one's own decision*, *participate in decisions* and *have say in the job*. Therefore, the factor was called job decision latitude; in later studies, it was also called job control. The second factor is mainly based on responses to the following items: *work fast*, *work very hard*, *not enough time*, *excessive work* and *no time to finish*. As a result, the factor was called job demands because it mainly characterizes additional sources of stress for the individual. Given the factor loadings for each item and the individual's answers to the questions, every individual is assigned a predicted value for their level of control and level of demand at work.

Individuals are placed in the group of *low control* if their individual control value is in the range of the lower 50% of all control values in the sample. If the individual value is in the higher 50% range, the individual is defined as experiencing *high control.* A similar process using the 50% benchmark is performed for the demand variable. The combination of the two possible values from each dimension results in four different groups of job strain. Figure 1 illustrates the values for control and demand and the resulting four job strain groups. To achieve more precise groups, separation into three or four categories in each dimension is possible and has been used in several studies (Collins et al 2005; Kuper and Marmot 2003). However, the majority of research studies are conducted with the four groups of job strain as shown below.

**Figure 1**: Job strain categories based on the value of job demand and job control after Karasek, 1979. The axis from low strain to high strain measured the level of unresolved strain. The axis from a passive state to an active state indicates the activity level.




The combination of low demands and high control is called *low strain*, and it represents the most advantageous combination, according to the literature (Karasek, 1979). The opposite case is classified as *high strain* and marks people in the unfortunate situation of high job demands and a low level of control. Both combinations are unbalanced because the demand and control level have different values.

The balanced combinations are called *passive* (both low) and *active* (both high). While the latter is regarded as a potentially motivating and positively challenging combination, the passive group is neither challenging nor stress-inducing, and therefore, it is regarded as less harmful. Karasek (1979) discovered in his work that individuals in the low strain group are more satisfied with their job and life in general and are less vulnerable to depression. Individuals in the active and passive groups show slightly higher values than those in the low strain groups. The high strain group was noteworthy in all models as the most disadvantaged group.

To take into account the possible impact pathways from socioeconomic and behavioral characteristics, the models of CHD risk are completed step-wise with a set of covariates. Occupational class has been shown to have a strong impact on CHD (Hallqvist et al 1998; Volkers, Westert and Schellevis 2007). Occupational class and other operationalizations of SES are used as additional

covariates in studies analyzing job strain. It is hypothesized that occupational class and prevalence of job strain are correlated. Occupationally stratified models are applied here to test whether job strain affiliation has different effects within the different occupational groups.

The job strain model primarily measures the level of psychosocial stress in an individual's workplace. While short-term stress can activate resources and improve productivity, long-term stress is known to have damaging effects on the human organism (McEwen 2002; Selye 1955). Changes due to stress in the metabolism (Chandola, Brunner and Marmot 2006) and endocrine system (Kajantie and Phillips 2006) can cause elevated blood circulation or increase the risk of inflammation due to a suppressed immune system (McEwen and Gianaros 2010).

This study uses the job strain model to identify harmful work-related conditions in relation to the risk for CHD in a sample of Swedish men and women. Men and women have different biological mechanisms leading to cardiovascular diseases such as CHD. In a given age group, women experience much fewer numbers of CHD incidences than men. In total, including all ages, women experience the same level of CHD mortality over the course of life, but they experience CHD later in life than men on average (WHO 2011a). The difference is, at least partially, due to genetic and endocrinological differences in the bodies of men and women (Kajantie and Phillips 2006).

Furthermore, previous studies have found many differences between the sexes regarding education, occupational class and health behavior (Hemingway 2007). There is evidence that women and men are differently distributed in the labor market and follow different behavioral schemata in the work place (Nikiforov and Mamaev 1998). Additionally, a concentration in specific occupations and labor market sectors for men and women, as well as a hierarchical segregation, can be observed in the labor market (Arber and Ginn 1995). Studies have shown that men and women use different strategies to cope with work-related stress (Kajantie 2008; Matud 2004) and that men and women make different use of social support structures in stressful situations (Day and Livingstone 2003). Analyzing men and women in separate models to capture all biological and socio-behavioral differences is an established method (Belkic et al 2004).

While the sex difference in CHD risk is widely accepted, there is no clear sex separation in the job strain models. Many studies analyzing job strain have done so only for a male population (Belkic et al 2004). The studies that have considered populations containing men and women have usually found different results for the job strain groups (Hellerstedt and Jeffrey 1997). However, accounts of sex separation are methodologically inconsistent because the demand and control variables are typically generated using the whole sample, including men and

women. Consequently, the job strain categories (*low strain, high strain, passive* and *active*) are unevenly distributed once the population is divided into the male and female samples. As explained above, sex differences in the labor market and stress coping strategies can lead to differing distributions of men and women among the job strain groups. Therefore, this study uses job strain categories that are generated from sex-specific samples. As a result, the distribution of individuals among the job strain categories is much more even. The aim of this strict sex separation is to investigate the effects of job strain on the risk of CHD, reducing the bias of gender roles and sex-specific behavior on the labor market.

Other studies have shown that, in addition to the direct pathway, there is a potential indirect impact pathway that mediates the stress effect through changes in the health behavior of the individual (Chandola et al 2008; Gémes et al 2008; László et al 2010). Among the behavioral changes that have a strong impact on cardiovascular disease are dietary preferences, physical exercise and substance use, such as smoking, alcohol or drug consumption, which are all risk factors of CHD. Previous models have primarily measured the direct effect of job strain group affiliation on the onset of CHD. The close relationship between CHD and some of its risk factors causes to hypothesize that there may be potential correlations between the various job strain groups and the CHD risk factors. In the case that this assumption is valid, the indirect effects of job strain via the risk factors should be taken into account when quantifying the effect of job strain on the risk for CHD. To test if there are indirect effects, additional models are performed to evaluate the correlation between job strain classification and the prevalence of several CHD risk factors.

## 2.4. Data and Methodology

This study uses data from the Malmö Diet and Cancer Study (MDCS), a prospective cohort study conducted in Malmö, the third largest city in Sweden, which had approximately a quarter of a million inhabitants at the time of the study. The target population of the study included all men and women born between 1926 and 1945 and living in Malmö (n= 53,491) when the initial data collection took place in 1991-1992. In 1995, the study population was extended, widening the birth year range to 1923-1950. The overall participation rate was 40.6%. Between the years 1991 and 1996, a total of 17,325 women and 11,252 men were enrolled in this prospective study.

All potential participants were contacted via post and gave their consent before the actual interview. Participants had to have sufficient Swedish writing and reading skills to participate, which excluded a share of the targeted migrant population living in Malmö. Consequently, the migrant share of the study is underrepresented and not representative of the total migrant population in Malmö (Lindström 2000). To avoid selection bias, this paper only analyzes individuals born in Sweden (10,236 men and 16,128 women). However, no difference between the participants of the MDCS and the general population in a comparable age group was found in terms of the distribution of gender, age and marital status (Rosvall et al 2000).

Participants were followed after the initial questionnaires, monitoring medical outcomes. The sample analyzed in this study uses medical information updated until the 31. December 2006. Individuals may be followed up to 15 years after their baseline interview as long as they stay within the national borders of Sweden. While the information on potential CHD events is available with daily precision, all control variables are taken from the baseline interview and must therefore be considered as constant. This produces potential methodological problems because not all individual characteristics and behaviors are static. The analysis of changes in socioeconomic conditions and health behaviors is not included in this study.

Among many other variables, the baseline questionnaire includes the Swedish version of the job strain questionnaire. To avoid any bias from retrospective answers, this investigation considers only individuals who stated they were employed at the time of the baseline questionnaire and were thus exposed to the stated job strain conditions at the time. This restriction excludes 4,357 men and 6,434 women who identified themselves as unemployed or otherwise not economical active at the time of the baseline interview. Consistent with the focus on the working population, it is also necessary to censor individuals once they enter retirement. Because the transition into retirement is not recorded in this study, individuals are censored when they reach the official retirement age of 65. There are 112 men and 102 women excluded from the start because they had already reached the age of 65 at the time of the baseline interview.

This study focuses on the onset of CHD rather than the progression of the disease. Therefore, individuals with a known cardiovascular history are excluded from this analysis (245 men and 187 women). Due to missing information for other covariates listed below, the sample is further reduced by 866 men and 1,313 women. The final sample includes 4,656 men and 8,092 women with complete information on job strain, socio-demographic background and CHD risk factors. Given the study format of a longitudinal dependent variable with cross-sectional background information collected at the time of the baseline questionnaire,

survival analysis is performed to estimate the CHD risk. Cox-proportional-hazard regressions are conducted to obtain the parameter estimates, using the statistical software package STATA 12.

Age is used as the time variable for the survival analysis, with the baseline interview marking the date when the individual entered the observation period and therefore became at risk for experiencing a CHD event. Throughout the survival analysis, participants are followed until they *i)* experience a CHD event, *ii)* reach age 65, *iii)* reach the end of the study (31. December 2006) or *iv)* drop out due to other reasons. Due to the restrictions of the assumed retirement age, the range of ages at baseline is 44.5-65 years, with a mean age of 55.2 for men and 53.0 years for women. The mean observation time from baseline until censorship is 8.7 years for men and 9.6 years for women.

*Dependent variable (Coronary heart disease)*

The risk of CHD is measured from the time of the baseline interview onwards. The mean observation time from baseline until a CHD event is 5.4 years for men and 6.8 years for women. Due to the previously described focus on disease onset, this study does not include repeated events. An individual is censored from further observation after experiencing an initial CHD event. This study does not distinguish between fatal and non-fatal CHD events.

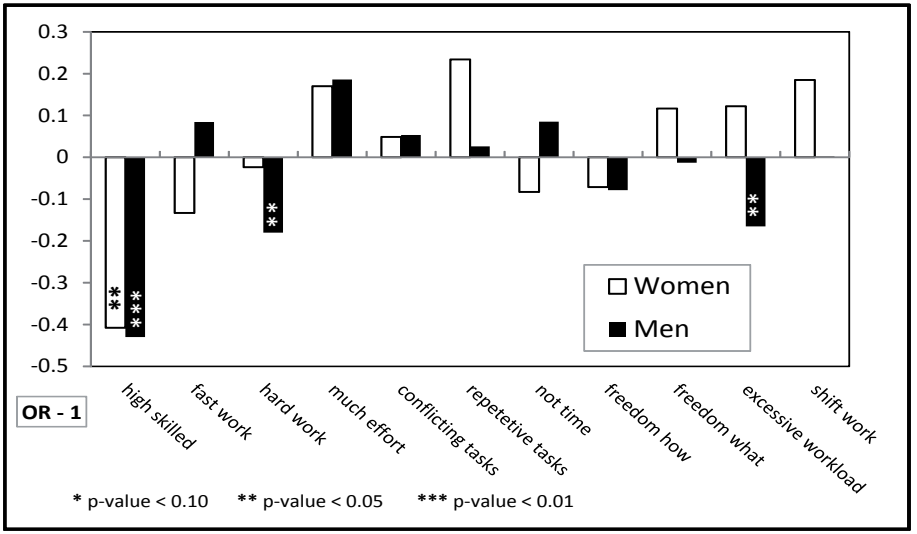
Individuals who experience a CHD event usually suffer from damage to the heart muscle and the blood vessel system; therefore, the risk of a repeated event is greatly increased after an initial CHD event. Based on their medical history, individuals who had already experienced a cardiovascular event are excluded from the sample. Restricting the analysis to people free of former CHD events enables a focus on the onset rather than the progression of coronary heart disease.

In addition to the potential CHD event that might be experienced during the observation time, the data contain information about hereditary predispositions for CHD. This is an uncommonly precise variable for genetic predisposition that was generated from detailed information about close blood relatives and their medical history. The necessary information for this heredity indicator was collected from the individual during the baseline interview. The prevalence of CHD cases in close blood-related family members indicates the individual's risk of CHD originating from genetic predisposition. The variable is operationalized as a categorical variable that can take the values of *low heredity score*, *medium heredity score* and *high heredity score* with the score approximating the risk level for CHD.

*Demand and Control Model*

To generate the necessary variables of demand and control for use in the job strain model, this study uses the Swedish psychosocial questionnaire on work stress that was included in the MDCS baseline questionnaire. The information from 11 single questions is combined to create the two variables of work demand and work control. The questions include information on the following: whether the work is hard, has to be performed very quickly, is repetitive, needs high skills or much effort; if the individual has enough time to perform the task, has freedom to decide what and how the tasks are performed, if the work load is excessive and if the work includes conflicting situations or shift work (the complete questionnaire is displayed in the appendix, Table A1). To illustrate the effect of the individual items on the risk of the onset on CHD, logistic regressions are performed including only the variables from the psychosocial questionnaire. Figure 2 displays the results for these regressions, stratified by sex.

**Figure 2**: Effect of individual work conditions on the incidence of CHD estimated by logistic regression, stratified by sex. Significance level of the single estimations is marked with a scale of asterisks. Data used for the calculation was obtained from the MDCS database.




Two important characteristics become apparent in figure 2. First, the effects of the individual items vary considerably in size and direction. Second, the effect of some items varies between the sexes. While the effect of an excessive workload increases the risk for CHD among women, it seems to have a significant beneficial effect for men. Only a few of the items show significant results. To consolidate

those effects and make the effect of work-related conditions more visible, the Karasek job strain model is used in this study.

Using factor analysis, the answers from the psychosocial questionnaire are defined into values of job demand and job control, according to the main factor loadings in the sex-separated samples. All of the items that appeared in the original psychosocial work stress questionnaire developed by Karasek (all but *shift work*) have similar factor loadings and therefore drive the dimensions of demand and control in a similar amount relative to Karasek's original study (the table of factor loadings is found in the appendix, Table A2). This is a good indicator of the consistency of the job strain model. According to the individual values for job demand and job control, the four categories of job strain (*low strain*, *high strain*, *passive* and *active*) are produced as previously described in the theoretical background.

*Occupational class* is generated using the Swedish socioeconomic classification (SEI). Three groups of occupational class based on aggregated information on employment in different sectors and professional position are used for this analysis. These occupational classes are defined as blue collar, white collar and upper white collar. The detailed generation of this variable using the original information from the MDCS is displayed in the appendix (Table A3). Although a more detailed separation of occupational class would be desirable, the number of CHD cases does not support more than three occupational groups.

*Education* is included initially in the models as a categorical variable taking the values of primary education (up to 8 years of schooling), secondary education (9-12 years of schooling) and university education (post-high-school education). According to the literature, it is common to find a higher risk for CHD with lower education levels (Falkstedt and Hemmingsson 2011; Smith et al 1998).

In addition to work-related factors, CHD has other potential covariates that need to be addressed. Being overweight or obese increases the risk for CHD. Furthermore, excess weight and obesity are also risk factors for other CHD influencing conditions, such as hypertension and diabetes, and could thus have an indirect impact on the risk for CHD. Therefore, the body mass index (BMI) of the individual at baseline is included. BMI is defined as the ratio of weight in kilograms to the square of the height in meters. Here it is used as a categorical variable: normal weight ($BMI \leq 24.9$), overweight ($25.0 < BMI \leq 30$) and obese ($BMI > 30$).

Another major risk factor for CHD is *smoking*, which increases the risk for hypertension due to a narrowing of the blood vessels. The CHD risk from smoking is controlled for using a categorical variable (non-smoker, regular smoker and occasional smoker at time of baseline).

There are also two variables indicating if the individual was diagnosed with *hypertension* or *diabetes* at the time of the baseline interview. Both medical conditions are treatable but not curable and known as major independent risk factors for cardiovascular disease. Because hypertension and diabetes are reported at the baseline and therefore treated as constant over the follow-up period, this study is unable to incorporate newly diagnosed cases during the observation period. Due to the database format, this study assumes that the variables of BMI and smoking are constant over time. The assumption of constant lifestyle covariates is not realistic and may add bias to the estimates, but this is an unavoidable limitation of the study design.

## 2.5. Results

This paper emphasizes the importance of sex differences in health outcomes and occupational structures. The novelty of this study is in the sex-separated generation of workplace control and demand variables. To display the difference between the original and the sex-separated generation of job strain categories, control and demand have been generated using both methods. Table 1 displays the difference in the variables characteristics and distribution of job strain categories generated with the mixed sex sample and with sex-specific samples.

**Table 1**: Comparison of job demand and job control dimension for men and women between a combined and sex-separated model, MDCS database

| | Combined sample | | Sex-separated sample | |
|---|---|---|---|---|
| **Job strain dimension** | Men | Women | Men | Women |
| **Demand** | | | | |
| Range | (-1.83 - 2.84) | (-1.83 - 2.84) | (-1.85 - 2.78) | (-1.81 - 2.87) |
| Mean | 0.01 | 0.00 | 0.00 | 0.00 |
| SD | 0.98 | 1.01 | 1.00 | 1.00 |
| **Control** | | | | |
| Range | (-2.49 - 2.30) | (-2.54 - 2.27) | (-2.94 - 2.09) | (-2.39 - 2.41) |
| Mean | 0.25 | -0.15 | 0.00 | 0.00 |
| SD | 0.93 | 1.01 | 1.00 | 1.00 |
| **Cases** | 4656 | 8092 | 4656 | 8092 |
| **Job strain categories in %** | | | | |
| active | 30.09 | 20.64 | 25.00 | 23.23 |
| passive | 20.96 | 30.28 | 25.41 | 25.43 |
| low strain | 28.93 | 20.84 | 24.59 | 25.74 |
| high strain | 20.02 | 28.25 | 25.00 | 25.59 |

The characteristics of the variables of job demand and job control vary given the method of generation. The original reason to distinguish the job strain categories at the 50% quartile benchmark of the demand and control dimensions was to achieve similar distributions among the four resulting job strain categories. If the job strain categories are generated in the sample with men and women combined, the resulting job strain categories distribution is uneven, as demonstrated in table 1. The sex-specific distribution of the labor market and sex-specific stress coping strategies produce strong sex differences regarding perceived demand and control levels. Therefore, this study generates demand and control variables for sex-separated samples, and as a result, each job strain category contains approximately one quarter of the individuals for both men and women. Disregarding this occupational sex-difference could cause bias in the interpretation of the effects of job strain on CHD. As a sensitivity test, both versions of the job demand and job control variable are analyzed according to their effect on CHD risk (Table 2). In both cases, the control variable shows a much stronger and significant effect on CHD than the demand variable. Given the consistent effects of demand

and control using both the sex separated and combined methods, this study continues with the sex-separated generation of demand and control and thus the sex-separated generation of job strain categories.

**Table 2**: Bivariate logistic regression on CHD for two different methods to generate job strain dimensions – demand and control for men (N=4,656) and women (N=8,092), MDCS database

| | Combined sample | | Sex-separated sample | |
|---|---|---|---|---|
| **Job strain dimension** | **Men** | **Women** | **Men** | **Women** |
| | Odds ratio | | Odds ratio | |
| **Demand** | 1.003 | 0.969 | 1.021 | 0.959 |
| **Control** | 0.801*** | 0.866* | 0.822*** | 0.863* |

*** p<0.01, ** p<0.05, * p<0.1

To illustrate the distribution of covariates among the job strain categories, table 3 and table 4 provide overviews of all of the covariates used for each job strain category for men and women, respectively. From the raw distribution, the high strain group shows the highest incidence of CHD, and the low strain group shows the lowest. The CHD incidence rates for the passive and active groups are in between. While this distribution is similar for men and women, the overall CHD incidence rate is much lower for women. The sex differences between socioeconomic covariates, such as occupational group and educational level, support the sex-separated generation of job strain categories.

**Table 3**: Distribution of CHD cases and covariates by job strain category for men, MDCS database

| **Men** | | Job strain category | | | | |
|---|---|---|---|---|---|---|
| | | active | passive | low strain | high strain | total |
| | | % | % | % | % | % |
| CHD cases | | 3.69 | 3.97 | 2.88 | 5.67 | 4.06 |
| Occupational class | blue collar | 21.48 | 54.78 | 31.35 | 54.90 | 40.72 |
| | white collar | 38.57 | 35.84 | 40.96 | 31.01 | 36.58 |
| | upper white collar | 39.95 | 9.38 | 27.69 | 14.09 | 22.70 |
| Education | primary | 18.04 | 61.28 | 23.49 | 57.22 | 40.16 |
| | secondary | 36.00 | 30.09 | 38.60 | 31.87 | 34.11 |
| | university | 45.96 | 8.62 | 37.90 | 10.91 | 25.73 |
| BMI | normal | 37.89 | 39.39 | 40.00 | 38.06 | 38.83 |
| | overweight | 51.20 | 49.62 | 50.39 | 48.97 | 50.04 |
| | obese | 10.91 | 10.99 | 9.61 | 12.97 | 11.13 |
| Heredity score | low | 65.64 | 65.68 | 66.81 | 61.94 | 65.01 |
| | medium | 29.30 | 29.42 | 28.47 | 30.93 | 29.53 |
| | high | 5.07 | 4.90 | 4.72 | 7.13 | 5.46 |
| Smoking | non-smoker | 75.09 | 69.06 | 73.54 | 64.95 | 70.64 |
| | occasional smoker | 5.33 | 5.83 | 5.15 | 6.19 | 5.63 |
| | smoker | 19.59 | 25.11 | 21.31 | 28.87 | 23.73 |
| Hypertension | | 12.63 | 14.62 | 13.54 | 16.49 | 14.33 |
| Diabtes | | 2.23 | 3.38 | 2.36 | 2.75 | 2.68 |
| Total number | | 1164 | 1183 | 1145 | 1164 | 4656 |

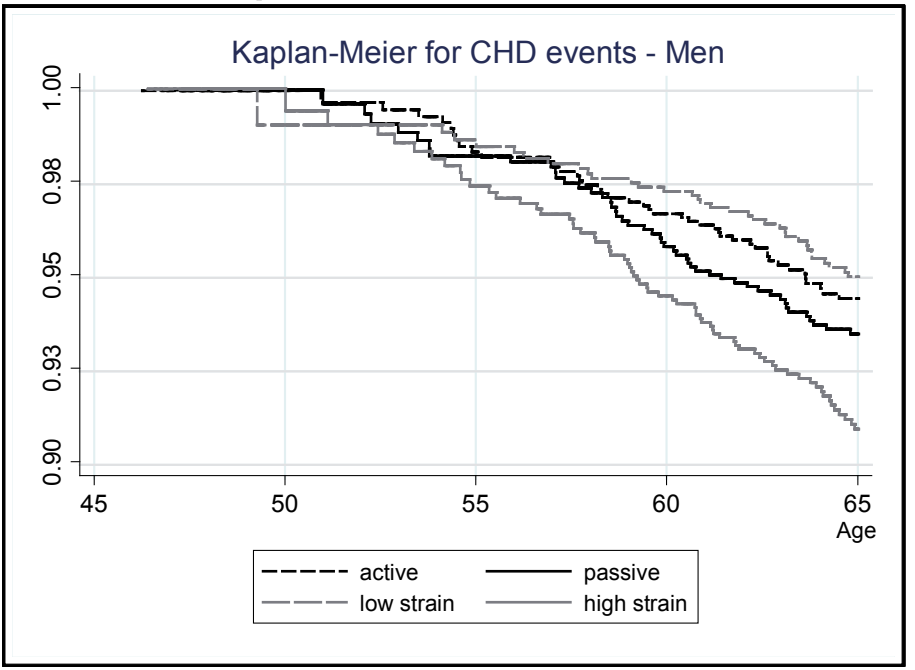
By comparing table 3 and table 4, it is apparent that men and women differ from each other not only in socioeconomic covariates but also in medical and lifestyle factors. Men have a higher prevalence of being overweight and obese but slightly lower prevalence of being a regular smoker than women. Hypertension and diabetes are also more common among men than among women.

**Table 4**: Distribution of CHD cases and covariates by job strain category for women, MDCS database

| **Women** | | Job strain category | | | | |
|---|---|---|---|---|---|---|
| | | active | passive | low strain | high strain | total |
| | | % | % | % | % | % |
| CHD cases | | 0.80 | 0.83 | 0.67 | 1.30 | 0.90 |
| Occupational class | blue collar | 19.73 | 43.15 | 31.45 | 46.60 | 35.58 |
| | white collar | 56.38 | 54.57 | 53.67 | 49.59 | 53.48 |
| | upper white collar | 23.88 | 2.28 | 14.88 | 3.81 | 10.94 |
| Education | primary | 9.95 | 41.01 | 24.48 | 35.06 | 28.02 |
| | secondary | 26.81 | 50.29 | 37.59 | 47.03 | 40.73 |
| | university | 63.24 | 8.70 | 37.93 | 17.91 | 31.25 |
| BMI | normal | 60.59 | 53.94 | 60.78 | 56.35 | 57.86 |
| | overweight | 29.36 | 34.84 | 29.72 | 31.72 | 31.45 |
| | obese | 10.05 | 11.22 | 9.51 | 11.93 | 10.69 |
| Heredity score | low | 62.18 | 61.42 | 63.27 | 62.77 | 62.42 |
| | medium | 31.54 | 31.58 | 31.01 | 29.84 | 30.98 |
| | high | 6.28 | 7.00 | 5.71 | 7.39 | 6.60 |
| Smoking | non-smoker | 71.17 | 68.17 | 72.11 | 64.99 | 69.07 |
| | occasional smoker | 4.95 | 5.05 | 6.10 | 5.26 | 5.35 |
| | smoker | 23.88 | 26.77 | 21.80 | 29.74 | 25.58 |
| Hypertension | | 9.79 | 11.47 | 10.66 | 12.70 | 11.18 |
| Diabtes | | 1.01 | 1.75 | 1.20 | 1.55 | 1.38 |
| Total number | | 1880 | 2058 | 2083 | 2071 | 8092 |

Before performing the Cox-proportional hazard models on the onset of CHD for the different job strain categories, Kaplan-Meier Survival curves are calculated and presented to achieve a first overview of the survival of the included individuals over the observation period. To illustrate the different survival curves for the four job strain categories more clearly, the graphs for men and women use different scales for the incidence of CHD events.

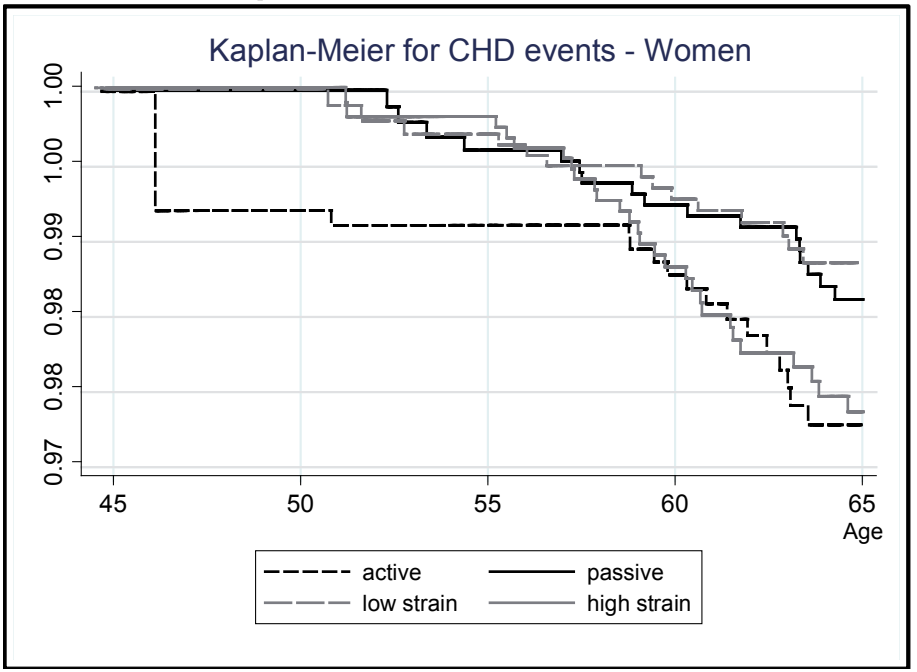
**Figure 3**: Kaplan-Meier survival curve for men (N = 4,656) stratified by job strain categories, calculations based on a sample from the MDCS database




For men (Figure 3), the group of high strain workers has a much lower survival rate than the other job strain categories. The survival curves for the other three job strain categories overlap until an age of approximately 58, after which a clear gradient is observed, showing that low strain is the most beneficial group, followed first by active and then passive workers.

For women (Figure 4), the pattern is different, and the gradient is less clear. However, the survival curves of the job strain categories fall into two groups. The groups of low strain and passive show similar patterns with higher survival than the both groups high strain and active. This is the first indication that job strain is having a different effect on men than on women.

**Figure 4**: Kaplan-Meier survival curve for women (N = 8,092) stratified by job strain categories calculations based on a sample from the MDCS database




Further analysis aims to quantify the effect of job strain affiliation on the onset of CHD and the roles of the various covariates. Table 5 displays the results of the survival analysis using Cox-proportional hazard models to measure the risk of CHD with a step-wise integration of sets of covariates for men. Basic Model 1 contains only the job strain categories from the sex-separated samples as impact factors for the risk of CHD. As observed in the Kaplan-Meier survival curves, the high strain group has a significantly increased risk for CHD, using the low strain group as the reference level. The estimates for the passive and active groups show elevated risks as well, but their estimates are not significantly different from the reference category.

Model 2 and Model 3 display the results when, in addition to job strain, the model controls for SES, measured as occupational group and educational level. As expected, the risk estimates show a gradient for education, showing those individuals with higher levels of education to be at an advantage. For occupational group, the trend is non-linear. The group with the least risk is the white-collar workers. As seen in the distribution of covariates, only 20 percent of all workers have an upper white-collar position. It might be assumed that those positions contain a higher level of responsibilities and work-related stress factors, which

could explain the higher CHD risk relative to white-collar workers. However, statistically, both white-collar groups show a health benefit over the blue-collar workers, although they cannot be significantly distinguished from each other. The correlation between education and occupation is quite high (correlation coefficient of 0.4), and the inclusion of both SES measurements in the same model (Model 4) interferes with the estimates of both covariates. Because both variables show similar, expected effects, the study continues using only the occupational group as a socioeconomic indicator for the later models.

In Model 5, the variables that indicate lifestyle factors are included. All three variables show the expected effect on CHD risk, with an elevated risk for overweight and obese men, smokers and those with a higher hereditary precondition, although the results for the categories of the latter variable remain non-significantly different from each other. Controlling for these CHD risk factors, as well as for hypertension and diabetes, (Model 6) does have an effect on the risk estimates for the job strain categories. With the rising number of control variables, the direct effect of the job strain categories decreases. The loss of significance could be a result of weaker direct effects or of a lower number of events per each covariate ratio when more controls are added to the model.

However, the complete model (Model 6) does include all covariates, and the high strain categories still show a significantly elevated risk for CHD (hazard ratio = 1.5) compared to the low strain group, although the estimated difference is lower than in the initial model.

**Table 5:** Results from the step-wise models of Cox-proportional-hazard models for men, MDCS database

| Men | | Model 1 HR (95% CI) | Model 2 HR (95% CI) | Model 3 HR (95% CI) | Model 4 HR (95% CI) | Model 5 HR (95% CI) | Model 6 HR (95% CI) |
|---|---|---|---|---|---|---|---|
| Job strain | active | 1.219 (0.776 - 1.916) | 1.267 (0.808 - 1.985) | 1.268 (0.805 - 1.998) | 1.296 (0.825 - 2.037) | 1.262 (0.804 - 1.982) | 1.273 (0.811 - 1.997) |
| | passive | 1.424 (0.912 - 2.225) | 1.290 (0.812 - 2.050) | 1.216 (0.758 - 1.951) | 1.164 (0.720 - 1.883) | 1.240 (0.779 - 1.974) | 1.227 (0.772 - 1.950) |
| | low strain | 1 | 1 | 1 | 1 | 1 | 1 |
| | highstrain | 1.965*** (1.294 - 2.983) | 1.771*** (1.153 - 2.721) | 1.705** (1.111 - 2.615) | 1.611** (1.043 - 2.487) | 1.573** (1.022 - 2.420) | 1.543** (1.002 - 2.375) |
| Occupation | blue collar | | 1 | | 1 | 1 | 1 |
| | white collar | | 0.584*** (0.414 - 0.824) | | 0.610*** (0.427 - 0.873) | 0.639** (0.451 - 0.903) | 0.621*** (0.439 - 0.879) |
| | upper white collar | | 0.702* (0.467 - 1.056) | | 0.773 (0.501 - 1.191) | 0.767 (0.510 - 1.154) | 0.778 (0.518 - 1.169) |
| Education | primary | | | 1 | 1 | | |
| | secundary | | | 0.760 (0.539 - 1.073) | 0.874 (0.610 - 1.253) | | |
| | university | | | 0.609** (0.395 - 0.938) | 0.668* (0.420 - 1.062) | | |
| BMI | normal weight | | | | | | |
| | overweight | | | | | 1.521** (1.092 - 2.117) | 1.427** (1.022 - 1.994) |
| | obese | | | | | 2.469*** (1.600 - 3.811) | 2.029*** (1.278 - 3.220) |
| Heredity score | low | | | | | 1 | 1 |
| | medium | | | | | 1.236 (0.907 - 1.684) | 1.242 (0.912 - 1.693) |
| | high | | | | | 1.449 (0.813 - 2.582) | 1.380 (0.767 - 2.482) |
| Smoking | non-smoker | | | | | 1 | 1 |
| | occasional smoker | | | | | 2.207*** (1.290 - 3.778) | 2.175*** (1.266 - 3.738) |
| | smoker | | | | | 2.710*** (1.999 - 3.672) | 2.755*** (2.030 - 3.738) |
| Hypertension | | | | | | | 1.852*** (1.286 - 2.666) |
| Diabetes | | | | | | | 2.210** (1.156 - 4.224) |
| Individuals | | 4656 | 4656 | 4656 | 4656 | 4656 | 4656 |
| CHD cases | | 189 | 189 | 189 | 189 | 189 | 189 |
| Time at risk | | 40552 | 40552 | 40552 | 40552 | 40552 | 40552 |
| Degrees of freedom | | 3 | 5 | 5 | 7 | 11 | 13 |
| Wald chi2 | | 11.95 | 21.28 | 16.61 | 23.49 | 80.23 | 107 |
| Log Likelihood | | -1504 | -1499 | -1501 | -1497 | -1471 | -1463 |

*** p<0.01, ** p<0.05, * p<0.1

For women (Table 6), the effects of the job strain categories are similar to those for men. However, the active group shows higher risk than the passive group, although they are not significantly different. The same strategy of step-wise addition of covariates is performed, with results similar to those for the male sample.

**Table 6**: Results from the step-wise models of Cox-proportional-hazard models for women, MDCS database

| Women | | Model 1<br>HR (95% CI) | Model 2<br>HR (95% CI) | Model 3<br>HR (95% CI) | Model 4<br>HR (95% CI) | Model 5<br>HR (95% CI) | Model 6<br>HR (95% CI) |
|---|---|---|---|---|---|---|---|
| Job strain | active | 1.226<br>(0.587 - 2.562) | 1.315<br>(0.626 - 2.762) | 1.415<br>(0.663 - 3.020) | 1.434<br>(0.671 - 3.065) | 1.231<br>(0.586 - 2.585) | 1.242<br>(0.589 - 2.619) |
| | passive | 1.199<br>(0.592 - 2.429) | 1.124<br>(0.545 - 2.318) | 1.037<br>(0.504 - 2.133) | 1.033<br>(0.498 - 2.141) | 0.999<br>(0.485 - 2.057) | 0.97<br>(0.471 - 1.997) |
| | low strain | 1 | 1 | 1 | 1 | 1 | 1 |
| | highstrain | 1.955**<br>(1.024 - 3.733) | 1.794*<br>(0.922 - 3.489) | 1.749*<br>(0.906 - 3.376) | 1.696<br>(0.871 - 3.301) | 1.579<br>(0.805 - 3.094) | 1.512<br>(0.772 - 2.961) |
| Occupation | blue collar | | 1 | | 1 | 1 | 1 |
| | white collar | | 0.563**<br>(0.348 - 0.911) | | 0.634*<br>(0.372 - 1.080) | 0.655*<br>(0.403 - 1.064) | 0.663*<br>(0.407 - 1.079) |
| | upper white collar | | 0.58<br>(0.223 - 1.505) | | 0.71<br>(0.244 - 2.069) | 0.67<br>(0.258 - 1.737) | 0.68<br>(0.264 - 1.755) |
| Education | primary | | | 1 | 1 | | |
| | secundary | | | 0.697<br>(0.416 - 1.167) | 0.813<br>(0.464 - 1.426) | | |
| | university | | | 0.484**<br>(0.237 - 0.988) | 0.593<br>(0.259 - 1.362) | | |
| BMI | normal weight | | | | | 1 | 1 |
| | overweight | | | | | 1.714**<br>(1.038 - 2.829) | 1.526<br>(0.907 - 2.567) |
| | obese | | | | | 1.922*<br>(0.970 - 3.808) | 1.589<br>(0.767 - 3.289) |
| Heredity score | low | | | | | 1 | 1 |
| | medium | | | | | 1.327<br>(0.796 - 2.213) | 1.274<br>(0.766 - 2.119) |
| | high | | | | | 2.746***<br>(1.410 - 5.350) | 2.428**<br>(1.185 - 4.973) |
| Smoking | non-smoker | | | | | 1 | 1 |
| | occasional smoker | | | | | 2.397*<br>(0.915 - 6.280) | 2.446*<br>(0.934 - 6.408) |
| | smoker | | | | | 4.040***<br>(2.463 - 6.628) | 4.077***<br>(2.493 - 6.669) |
| Hypertension | | | | | | | 2.086**<br>(1.156 - 3.764) |
| Diabetes | | | | | | | 4.919***<br>(1.854 - 13.05) |
| Individuals | | 8092 | 8092 | 8092 | 8092 | 8092 | 8092 |
| CHD cases | | 73 | 73 | 73 | 73 | 73 | 73 |
| Time at risk | | 77476 | 77476 | 77476 | 77476 | 77476 | 77476 |
| Degrees of freedom | | 3 | 5 | 5 | 7 | 11 | 13 |
| Wald chi2 | | 5.254 | 11.35 | 10.32 | 14.15 | 50.81 | 88.34 |
| Log Likelihood | | -613.1 | -610.3 | -610.8 | -609.3 | -588.9 | -581.7 |

*** p<0.01, ** p<0.05, * p<0.1

Once the lifestyle factors are included in Model 5, the high job strain group shows results similar to the male high strain group, but the estimate can no longer be significantly distinguished from the reference category. The highly elevated risk for CHD for the high strain group (hazard ratio = 1.5) indicates that the lack of significance is, at least partially, a result of missing statistical power due to the low number of CHD cases observed for women before retirement age (73 CHD incidences).

For both samples, men and women, the effects of the various job strain categories change once the model controls for occupational group. That working conditions are correlated with the occupational affiliation of the individual seems reasonable. Greater control and responsibility might be an advantage for the higher occupational hierarchical level but of no health effect at lower levels. Additional demands might be manageable in some occupations but exceed the individual's capabilities in others. The question emerges whether the dimensions of job demand and job control and thus, the job strain categories have different effects on CHD risk depending on the occupational level of the individual. Therefore, this study performs the Cox-proportional hazard model for the risk of CHD on occupationally stratified samples (Table 7).

Among the three occupational classes, job strain and CHD covariates are again added step-wise to observe the correlations between the variables. The first set of models includes only the job strain categories (Model a); the second set additionally controls for information on BMI, heredity score and smoking (Model b); and the final set represents the full model (Model c), including hypertension and diabetes. The advantage of this method is that the effect of the job strain model can be observed for the separated occupational groups. The disadvantage of the stratification is the reduced numbers of individuals and CHD cases in each model. The model specifications are displayed in table 7 for each model. The problem of a low number of cases is especially prominent among the higher occupational classes and among women in general.

For men, the occupationally stratified models show that there are different job strain patterns for the various occupational strata. The groups of blue-collar and upper white-collar male workers show a similar pattern as seen before, with high strain as the most detrimental condition and active and passive having higher CHD risk than the low strain reference group but less than the high strain group. For the upper white-collar group, the active and the high strain groups also show significant difference, despite the small number of CHD cases in those strata. There is a different pattern among white-collar male workers. The high strain and active groups show lower risk than the reference category, although not significantly.

Again the results for women differ quite substantially from the results for men. The effects of the job strain categories show a different pattern for each occupational group. Among blue-collar female workers, only the high strain group shows an elevated CHD risk. For white-collar workers, the high strain and active groups show elevated risks. However, none of the results for blue or white-collar job strain categories are significantly different from the reference categories, which is most likely because of the missing statistical power due to the low number of CHD events (37 CHD cases for the blue-collar and 30

**Table 7**: Cox-proportional hazard models on CHD incidence and the effect of job strain stratified by occupation and sex, MDCS database

| **Men** | | **Blue collar worker** HR (95% CI) | | | **White collar worker** HR (95% CI) | | | **Upper white collar worker** HR (95% CI) | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Model a | Model b | Model c | Model a | Model b | Model c | Model a | Model b | Model c |
| Job strain | active | 1.207 | 1.115 | 1.131 | 0.827 | 0.798 | 0.858 | 3.008** | 3.178** | 3.063** |
| | | (0.576 - 2.527) | (0.528 - 2.355) | (0.535 - 2.390) | (0.383 - 1.786) | (0.366 - 1.739) | (0.393 - 1.873) | (1.021 - 8.861) | (1.080 - 9.347) | (1.046 - 8.972) |
| | passive | 1.053 | 0.990 | 0.986 | 1.225 | 1.128 | 1.194 | 2.971 | 2.788 | 2.446 |
| | | (0.563 - 1.972) | (0.526 - 1.861) | (0.522 - 1.864) | (0.590 - 2.541) | (0.543 - 2.340) | (0.580 - 2.459) | (0.746 - 11.84) | (0.689 - 11.28) | (0.605 - 9.886) |
| | low strain | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| | highstrain | 1.676* | 1.444 | 1.449 | 0.915 | 0.799 | 0.823 | 5.455*** | 5.469*** | 4.689*** |
| | | (0.937 - 2.998) | (0.806 - 2.588) | (0.803 - 2.616) | (0.407 - 2.058) | (0.349 - 1.829) | (0.360 - 1.880) | (1.740 - 17.10) | (1.731 - 17.28) | (1.477 - 14.88) |
| Individuals | | 1896 | 1896 | 1896 | 1703 | 1703 | 1703 | 1057 | 1057 | 1057 |
| CHD cases | | 101 | 101 | 101 | 51 | 51 | 51 | 37 | 37 | 37 |
| Wald chi2 | | 5.224 | 39.410 | 55.670 | 1.102 | 28.200 | 37.540 | 8.651 | 31.380 | 46.690 |
| Log Likelihood | | -715.9 | -698.8 | -694.8 | -354.2 | -344.7 | -342.0 | -234.5 | -226.6 | -221.6 |

| **Women** | | **Blue collar worker** HR (95% CI) | | | **White collar worker** HR (95% CI) | | | **Upper white collar worker** HR (95% CI) | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Model a | Model b | Model c | Model a | Model b | Model c | Model a | Model b | Model c |
| Job strain | active | 1.030 | 0.983 | 1.086 | 1.610 | 1.537 | 1.512 | 1.408 | 1.301 | 1.243 |
| | | (0.301 - 3.520) | (0.288 - 3.362) | (0.301 - 3.922) | (0.556 - 4.662) | (0.526 - 4.493) | (0.514 - 4.449) | (0.125 - 15.89) | (0.111 - 15.31) | (0.104 - 14.91) |
| | passive | 1.024 | 0.907 | 0.921 | 0.974 | 0.898 | 0.888 | 6.774 | 4.361 | 4.603 |
| | | (0.390 - 2.690) | (0.350 - 2.346) | (0.346 - 2.449) | (0.315 - 3.010) | (0.296 - 2.723) | (0.289 - 2.724) | (0.446 - 103.0) | (0.237 - 80.10) | (0.236 - 89.88) |
| | low strain | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| | highstrain | 1.581 | 1.361 | 1.330 | 1.620 | 1.532 | 1.513 | 7.666* | 6.586 | 5.724 |
| | | (0.653 - 3.826) | (0.572 - 3.233) | (0.533 - 3.318) | (0.572 - 4.592) | (0.524 - 4.483) | (0.521 - 4.391) | (0.731 - 80.42) | (0.463 - 93.67) | (0.422 - 77.67) |
| Individuals | | 2879 | 2879 | 2879 | 4328 | 4328 | 4328 | 885 | 885 | 885 |
| CHD cases | | 37 | 37 | 37 | 30 | 30 | 30 | 6 | 6 | 6 |
| Wald chi2 | | 1.794 | 22.380 | 61.160 | 1.612 | 25.090 | 22247.0 | 5.147 | 5358.0 | 123.0 |
| Log Likelihood | | -274.2 | -263.4 | -255.2 | -232.4 | -221.8 | -221.1 | -35.2 | -32.4 | -30.9 |

*** p<0.01, ** p<0.05, * p<0.1; Model a) includes only job strain categories; Model b) Model a + BMI, heredity score and smoking; Model c) Model b + hypertension, diabetes

CHD cases for the white-collar group). The female upper white-collar group is the smallest, with only 6 cases of CHD. Nevertheless, the results show strong health disadvantages for the high strain and passive groups of women in the upper white-collar group. The effects are not significant in the full model, which contains a large set of covariates, but the high estimates support the argument that the lack of significance is mainly due to the low incidence of CHD cases among women in the observed age range.

Examining the results from the step-wise model performed so far makes it apparent that the categories of job strain and some of the other CHD risk factors interact with each other because the estimates for the effects of job strain vary considerably depending on the additional covariates used in the models. The models with job strain as the only impact factor show a strong and direct effect on CHD risk. The question emerges of whether job strain has similar effects on some of the other CHD risk factors. This would explain part of the interference in the models when job strain and the risk factors are controlled for. To test if job strain has not only a direct effect on CHD but also potentially an indirect effect via the CHD risk factors, logistic regressions are performed measuring the effect of the job strain category affiliation on the single CHD risk factors.

Table 8 displays the results of those models for men and women. Some of the risk factors were modified for this analysis. The category of overweight is now defined as all individuals with a BMI over 25, which include the former obese category. As a result, when overweight is analyzed as a dependent variable, the reference group contains only people with a BMI lower than 25. Similar adjustments were made for the categories of medium heredity score and occasional smoker.

As the results for men show in table 8, affiliation with the high job strain group has a significant effect on obesity, heredity score, smoking and hypertension. There is also a significant effect of the passive group on hypertension for men. The active group consistently shows no effect on any of the CHD risk factors for men.

For women, the results again show a different set of patterns. Affiliation with the high job strain group shows detrimental effects regarding obesity, heredity score, smoking and hypertension. Additionally, women affiliated with the passive job strain group show an elevated risk for many of the CHD risk factors compared to the reference group of low job strain. The estimates for increased risk among the passive group are significant for overweight, obesity, high heredity score and regular smoking. This analysis strongly indicates that job strain not only has a direct effect on CHD but also has an important influence on several of the risk factors for CHD.

**Table 8**: Univariate logistic regressions on CHD risk factors and the impact of job strain, stratified by sex, MDCS database

| **Men** | **Dependen variable** | **Overweight** | **Obese** | **Medium heredity score** | **High heredity score** | **Occasional smoker** | **Smoker** | **Hypertension** | **Diabetes** |
|---|---|---|---|---|---|---|---|---|---|
| | | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** |
| Job strain | active | 1.093 | 1.152 | 1.054 | 1.079 | 1.036 | 0.899 | 0.923 | 0.946 |
| | | (0.925 - 1.292) | (0.880 - 1.509) | (0.887 - 1.252) | (0.739 - 1.575) | (0.718 - 1.494) | (0.735 - 1.101) | (0.725 - 1.176) | (0.549 - 1.631) |
| | passive | 1.026 | 1.162 | 1.052 | 1.042 | 1.140 | 1.238** | 1.094 | 1.449 |
| | | (0.869 - 1.211) | (0.888 - 1.519) | (0.886 - 1.249) | (0.712 - 1.523) | (0.797 - 1.630) | (1.020 - 1.501) | (0.866 - 1.382) | (0.883 - 2.378) |
| | low strain | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| | highstrain | 1.085 | 1.403** | 1.237** | 1.551** | 1.214 | 1.498*** | 1.262** | 1.171 |
| | | (0.918 - 1.283) | (1.081 - 1.820) | (1.043 - 1.467) | (1.090 - 2.208) | (0.852 - 1.730) | (1.239 - 1.812) | (1.003 - 1.587) | (0.697 - 1.967) |
| Constant | | 1.500*** | 0.106*** | 0.497*** | 0.050*** | 0.054*** | 0.271*** | 0.157*** | 0.024*** |
| | | (1.333 - 1.688) | (0.087 - 0.129) | (0.439 - 0.562) | (0.038 - 0.065) | (0.042 - 0.071) | (0.235 - 0.312) | (0.132 - 0.185) | (0.017 - 0.035) |
| Individuals | | 4656 | 4656 | 4656 | 4656 | 4656 | 4656 | 4656 | 4656 |
| CHD incidences | | 189 | 189 | 189 | 189 | 189 | 189 | 189 | 189 |
| Wald chi2 | | 1.544 | 6.731 | 6.877 | 8.483 | 1.458 | 32.690 | 7.829 | 3.552 |
| Prob (chi2) | | 0.672 | 0.081 | 0.076 | 0.037 | 0.692 | 0.000 | 0.050 | 0.314 |
| Log Likelihood | | -3109 | -1622 | -3011 | -982 | -1008 | -2535 | -1909 | -574 |
| Pseudo R2 | | 0.000 | 0.002 | 0.001 | 0.004 | 0.001 | 0.006 | 0.002 | 0.003 |

| **Women** | **Dependen variable** | **Overweight** | **Obese** | **Medium heredity score** | **High heredity score** | **Occasional smoker** | **Smoker** | **Hypertension** | **Diabetes** |
|---|---|---|---|---|---|---|---|---|---|
| | | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** |
| Job strain | active | 1.008 | 1.064 | 1.048 | 1.105 | 0.802 | 1.126 | 0.909 | 0.840 |
| | | (0.887 - 1.145) | (0.863 - 1.313) | (0.921 - 1.192) | (0.850 - 1.437) | (0.609 - 1.055) | (0.970 - 1.306) | (0.740 - 1.118) | (0.461 - 1.531) |
| | passive | 1.323*** | 1.204* | 1.082 | 1.242* | 0.820 | 1.312*** | 1.086 | 1.466 |
| | | (1.170 - 1.497) | (0.985 - 1.471) | (0.954 - 1.227) | (0.966 - 1.596) | (0.628 - 1.070) | (1.138 - 1.513) | (0.894 - 1.319) | (0.877 - 2.450) |
| | low strain | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| | highstrain | 1.200*** | 1.289** | 1.022 | 1.317** | 0.856 | 1.519*** | 1.219** | 1.292 |
| | | (1.061 - 1.358) | (1.058 - 1.571) | (0.901 - 1.159) | (1.028 - 1.687) | (0.657 - 1.114) | (1.320 - 1.748) | (1.008 - 1.475) | (0.763 - 2.188) |
| Constant | | 0.645*** | 0.105*** | 0.580*** | 0.061*** | 0.065*** | 0.279*** | 0.119*** | 0.012*** |
| | | (0.591 - 0.705) | (0.091 - 0.122) | (0.531 - 0.635) | (0.050 - 0.073) | (0.054 - 0.078) | (0.251 - 0.309) | (0.104 - 0.137) | (0.008 - 0.018) |
| Individuals | | 8092 | 8092 | 8092 | 8092 | 8092 | 8092 | 8092 | 8092 |
| CHD incidences | | 73 | 73 | 73 | 73 | 73 | 73 | 73 | 73 |
| Wald chi2 | | 27.890 | 7.771 | 1.682 | 5.569 | 3.277 | 38.730 | 9.194 | 4.763 |
| Prob (chi2) | | 0.000 | 0.051 | 0.641 | 0.135 | 0.351 | 0.000 | 0.027 | 0.190 |
| Log Likelihood | | -5495 | -2747 | -5356 | -1965 | -1687 | -4582 | -2830 | -588 |
| Pseudo R2 | | 0.003 | 0.001 | 0.000 | 0.001 | 0.001 | 0.004 | 0.002 | 0.004 |

*** p<0.01, ** p<0.05, * p<0.1

## 2.6. Discussion

This study investigates the effect of Karasek's job strain categories on the onset of CHD among a sample of Swedish men and women. The categories of job strain are generated with sex-specific samples to account for the differences in the distribution of men and women in the labor market and occupational classes. In general, this study confirms the findings of previous studies using the job strain model, showing a significantly elevated risk as a direct effect of high job strain on the onset of CHD for men. For women, the effect was similarly strong (Hazard ratio = 2), which could be the result of the sex-separated generation of job strain categories. However, the effect decreases when other CHD risk factors are taken into account to HR = 1.5, which is no longer significant for women. The general lack of significance in the female-only models is partially due to the low numbers of individuals and CHD cases for women.

The occupationally stratified models illustrate clearly that job strain has varying effects on the different occupation levels. The strongest detrimental effects of high job strain are found in the upper white-collar group, whereas for men, the active group shows significantly elevated CHD risks. In the female models, the passive group shows indications of detrimental effects among upper white-collar workers. Due to an extremely small number of women in that occupational group and only 6 cases of CHD, the difference from the reference category of low strain could not be statistically confirmed in the full model, which includes several other CHD risk factors. Despite the non-significance of results in the small samples of the occupationally stratified models, the size of effect of job strain is a strong indicator that job strain does indeed follow different patterns among the various occupational groups.

Throughout the step-wise integration of CHD risk factors, the estimates of health differences between the job strain groups changed. Therefore, the assumption was made that job strain not only has a direct effect on the onset of CHD but is also correlated with the other risk factors for CHD. The results of the analysis that evaluated the effect of job strain on several of the CHD risk factors demonstrate that job strain not only influences CHD risk in a direct way but also in an indirect way via CHD risk factors.

The risk estimates of the different models demonstrate that the effects of job strain can be different for men and women. This study generates job strain categories in sex-specific samples to avoid an uneven distribution of men and women among the job strain categories. Both the direct and indirect effects of different job strain combinations showed sex-specific patterns. A separation of

the male and female samples is therefore strongly recommended for future studies on job strain and cardiovascular disease.

The main limitation of this study is the cross-sectional format of the job strain categories, occupational affiliation and other covariates. Changes in health behavior and SES during the observation period are not measured and cannot be included in the analysis. Additionally, this limitation makes it impossible to investigate any causal relationship in the strong correlation between job strain affiliation and the other CHD risk factors because the sequence of events is not recorded. Although only the onset of CHD is analyzed, a causal relationship between job strain category affiliation and the incidence of CHD is not proven. Unobserved health problems, which can lead to a higher propensity for the onset of CHD, could also influence the choice of occupation or determine the individual's sensitivity to work-related stress. The causal relationship between health differences and the different job strain categories should be the topic of future research.

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# 2.8. Appendix

**Table A1**: Psycho-social job strain questions from the MDCS used for this study

| Variable name in MDCS dataset | Question |
|---|---|
| uy36 | Does your work demand that you work very fast? |
| uy37 | Does your work demand that you work very hard? |
| uy38 | Does your work demand too great an effort? |
| uy39 | Do you have enough time for your working tasks? |
| uy40 | Does your work often involve conflicting demands? |
| uy44 | Does your work mean that you have to do the same thing over and over again? |
| uy45 | Do you have the freedom to determine how your work will be performed? |
| uy46 | Do you have the freedom to determine what will be performed at your work? |
| uy47 | How often do you have to work overtime? |
| uy48 | Does your work mean inconvenient working hours or shift work? |
| uy1 | Highest level of education (higher education operationalized as "high skilled") |

**Table A2**: Factor loadings for the demand and control dimension of the single items generated separately for men and women, MDCS database

| | Rotated factor loadings | | | | | |
|---|---|---|---|---|---|---|
| | Men | | | Women | | |
| Variable | Demand | Control | Uniqueness | Demand | Control | Uniqueness |
| high skill | 0.089 | **0.483** | 0.759 | 0.253 | **0.499** | 0.687 |
| fast work | **0.704** | -0.175 | 0.474 | **0.628** | -0.305 | 0.513 |
| hard work | **0.713** | -0.037 | 0.490 | **0.704** | -0.044 | 0.502 |
| high effort | **0.769** | -0.034 | 0.408 | **0.778** | -0.031 | 0.393 |
| conflicting situation | **0.494** | 0.203 | 0.715 | **0.594** | 0.138 | 0.629 |
| repetative work | 0.007 | **-0.613** | 0.624 | -0.098 | **-0.594** | 0.638 |
| no time to finish | **0.659** | 0.122 | 0.552 | **0.709** | 0.039 | 0.495 |
| freedom on work process | -0.075 | **0.742** | 0.444 | -0.097 | **0.771** | 0.396 |
| freedom of work tasks | -0.010 | **0.738** | 0.455 | -0.070 | **0.733** | 0.459 |
| excessive workload | **0.390** | 0.194 | 0.810 | **0.474** | 0.268 | 0.704 |
| shift work | **0.269** | -0.163 | 0.901 | 0.183 | **-0.191** | 0.930 |

**Table A3**: generation of occupational class using the Swedish socioeconomic classification in the MDCS

| Swedish ses classification (SEI) | Description | Occupational class |
|---|---|---|
| 11 | Unskilled employee in goods production | Blue collar |
| 12 | Unskilled employee in service production | Blue collar |
| 21 | Skilled employees in goods production | Blue collar |
| 22 | Skilled employees in service production | Blue collar |
| 33 | Assistant non-manual employees, lower level | White collar |
| 36 | Assistant non-manual employees, higher level | White collar |
| 46 | Intermediate non-manual employees | White collar |
| 56 | Professionals and other higher non-manual employees | Upper white collar |
| 57 | Upper-level executives | Upper white collar |
| 60 | Employers, self-employed (no farmers) | Blue collar |
| 70 | Employers with 1-9 employees, (no farmers) | Upper white collar |
| 79 | Employers with more than 10 employees (no farmers) | Upper white collar |
| 89 | Employers, Farmers | Excluded from analysis |

Chapter 3

# Economic Stress in the Short and Long-Term and the Onset of Ischemic Heart Disease

**Authors**: Tina Hannemann, Jonas Helgertz

## 3.1. Abstract

Cardiovascular diseases (CVD) are today the main causes of death in most developed countries, accounting for more than 40 percent of all deaths. This study revisits the link between income attainment and the onset of one of the most important subgroup of CVD, namely Ischemic Heart Disease (IHD). Examining a longitudinal database consisting of around 90,000 men and women in Sweden, the analysis spans the years 1992-2001. In analyzing the influence of the individual's income attainment, the article distinguishes between the individual's absolute and relative position in the short, medium and long-term income distribution. Using random effect logistic regression, the article fails to find consistent support for the hypothesis that stress, operationalized as poor income attainment – either in the short, medium or long run – is associated with a substantial risk for the onset of IHD. This result holds both regarding absolute and relative income. Despite a predominance of statistically non-significant results, parameter estimates do, however, indicate that low-performing males are worse off than those at elevated positions in the income distribution. Among women, the estimates generally suggest that the highest performing women are experiencing the highest risk for IHD onset. This is argued to potentially be linked to gender-roles in the labor market and domestically, causing a considerably higher effort among women in attaining and maintaining an elevated labor market position.

## 3.2. Introduction

Empirical research into the determinants of Ischemic Heart Disease (IHD) has resulted in numerous articles in academic journals. Given that IHD and other CVDs are the main cause of death in developed countries (WHO 2011a), the importance of obtaining a better understanding regarding its determinants is difficult to overestimate. This is, however, not only of concern to the individual, who – in avoiding experiencing IHD – may avoid dying prematurely. Taking into account the high prevalence of CVD and the considerable medical interventions that are frequently necessary, the societal costs are substantial. In the UK, £29.1 billion were spent during 2004 on healthcare expenditure related to CVD, accounting for 18 percent of all healthcare costs in that year (Luengo-Fernández et al 2006). The corresponding figures for the US amount to US$ 297.7 billion, corresponding to 16 percent of all healthcare costs (Roger et al 2012). Due to the prevalence of CVD, costs associated with this diagnostic group generally represent the single largest share of total healthcare costs.

This article examines the case of Sweden, where over 40 percent of all-cause mortality is due to CVD, of which over half is attributable to IHD (Socialstyrelsen 2009). Unlike the USA, Sweden has a universal healthcare system that is almost entirely financed by taxes. Unsurprisingly, the healthcare costs associated with CVD represent a substantial share of the overall healthcare costs also in Sweden. Figures from 2010 suggest that the yearly cost for patients being treated for any CVD amounted to roughly SEK 61,5 billion, including both direct costs (e.g. hospital care, pharmaceuticals) and indirect costs (e.g. sickness benefits). Only considering the direct costs, the expenditures for the care of the CVD patients represent around eight percent of the total healthcare expenditures in Sweden (Steen Carlsson and Persson 2012).

A frequent finding from previous research on contemporary developed countries suggests a negative association between an individual's economic attainment and the incidence of IHD (see for example Cabrera et al 2001; Henriksson et al 2003; Toivanen 2007). Explanations to the existence of such a relationship point towards differences in access to healthcare and the ability to purchase a range of health promoting commodities that is linked to the individual's position in the income distribution. As a consequence, low income earning individuals are believed to suffer from an increased risk for IHD.

This article re-examines the link between income attainment and the risk for IHD, using an unprecedentedly large longitudinal sample of around 90,000 men and women in Sweden between 1992 and 2001. The importance of such re-examination is based on several reasons. First, whereas previous studies have

occasionally focused on the incidence of IHD indiscriminately, this article focuses solely on the onset of IHD. Distinguishing between any IHD-event and its onset is of potentially paramount importance, as the survival and recurrence rate from experiencing IHD is high. An initial IHD event will weaken the heart muscle and the blood vessels, needed to provide the heart with sufficient oxygen and nutrients for functioning. As a consequence, an individual will face a substantially elevated risk for IHD in the case of already previously experienced IHD events, compared with a person without CVD history (Kannel and Belanger 1991).

Second, having previously experienced the disease may have permanently diminished the individual's economic productivity, thereby causing a lowered income attainment. Consequently, a previous history of IHD may both diminish the individual's income attainment as well as act as a predictor of future IHD events. Therefore, the first hypothesis of this study is that the correlation between income differences and IHD is less prominent if only first IHD cases – the onset of the disease – are taken into account.

Being a country with universal health care, the existence of a link between income attainment and IHD risk should not primarily be due to differences in access to treatment for risk factors such as diabetes and hypertension. However, enjoying a high income could facilitate access to various other health promoting goods, such as the ability to purchase membership in sports clubs and affording healthy dietary habits. A higher income could also be associated with a higher socioeconomic status (SES), which can – in return – be correlated with better health behaviors.

While absolute income measures the financial ability to obtain health-promoting commodities and services, the article also investigates the relevance of the relative deprivation hypothesis. More specifically, it is hypothesized that a low relative income – relative deprivation – could be an alternate source of psychosocial stress, as it represents a situation where an individual performs poorly compared with otherwise similar individuals. Therefore, the second hypothesis of this study is that the expected health benefit for individuals in the upper range of the income distribution is more an effect of relative higher income, instead of more income in absolute terms.

In considering the role of absolute and relative income attainment as determinants of the onset of IHD, theory suggests the need to distinguish between short, medium and long-term exposure to a given condition. Therefore, in investigating the influence of income attainment, this article considers time periods extending as far back as ten years prior to the year at risk for IHD. The resulting third research question is how much the income effect varies, considering the various time spans of income attainment.

## 3.3. Theoretical framework

Income is considered as indicator for the SES of an individual. Numerous studies found a relationship between SES and various measures of health in contemporary contexts. Of particular interest for this article is the link between SES, measured as an individual's absolute and relative income attainment, and the risk of experiencing the onset of IHD. While income attainment by itself is unlikely to directly affect the incidence of IHD, it is believed to affect the individual's risk for IHD through two possible pathways. The neo-materialistic explanation focuses on the role of income in determining the individual's consumption capabilities (Blane et al 1997; Macintyre et al 1998). Individuals with higher earnings are capable of maintaining a high standard of living, including higher quality of housing (Pollack, von dem Knesebeck and Siegrist 2004), living in safer environments, participating in various health promoting activities (Droomers et al 1998; Pill, Peters and Robling 1995) and affording a healthy diet. Consequently, a higher prevalence of physical inactivity, smoking (Pomerleau and Pomerleau 1991) and unhealthy food choices (Dallman, Pecoraro and la Fleur 2005) was shown to be increasingly common within lower income groups (Hemingway 2007). The above listed factors have a direct correlation with the onset and progress of the underlying cause of IHD – atherosclerosis and IHD risk factors such as diabetes and hypertension. In addition to the lack of the monetary resources to afford various costly health commodities, other sources of stress and inconvenience can be linked to working unconventional hours, for instance shift-work, that is more prevalent in lower income groups.

Apart from the neo-materialistic explanation of why individuals with lower earnings are likely to experience poorer health, income is also related to other socioeconomic characteristics such as education and occupational status (Winkleby et al 1992). More specifically, individuals with lower educational and occupational attainment are typically more concentrated at the lower end of the income distribution. Since lower education and lower occupational status affiliation are correlated with worse health, observed health differences between income groups can, at least partly, originate from the distribution of educational and occupational level (Stronks et al 1997). The individual's educational attainment is correlated with their knowledge regarding how to maintain a healthy lifestyle through physical exercise, healthy diet and consumption of addictive substances as smoking, alcohol and other drugs. As a result, education acts independently as a health promoting asset. Similarly, numerous aspects of the individual's working conditions are directly linked to their position in the occupational hierarchy. More advanced positions are typically associated with a

higher degree of independence as well as the feeling of being able to affect one's working conditions.

The aforementioned discussion postulates that the individual's position in the income distribution contains information regarding the individual's knowledge and use of a range of health-promoting behaviors, through their purchasing power. Hence, a given income – in absolute terms – is associated with common consumption constraints. Thus, while individuals naturally have different consumption preferences, a certain attained income is believed to subject any individual to a given consumption constraint, regardless of their sex or age.

The second proposed mechanism through which an individual's income attainment may affect their risk of experiencing IHD views the income concept from different perspective. The relative deprivation hypothesis (Eibner and Evans 2005) focuses on a different source as the main stressor, underlying the onset of disease. Human capital investments are made based on – at least partly – expectations about an individual's future returns in terms of monetary remuneration. In the case where these expectations are met, the individual could be expected to feel psychologically satisfied about the investment made in education and training. The relative deprivation hypothesis proposes that individuals who perform poorly in the labor market *compared with a relevant reference group* are likely to suffer from elevated psychosocial stress (Wilkinson 1997), with a subsequent psychosomatic health impact. Psychosomatic diseases are assumed to originate from psychological stress which emerges when the individual is confronted with physical and psychological demands that the individual deems to be excessive. The human body is prepared to cope with a certain level of stress, but under conditions of very high levels of stress or unusually prolonged periods of stress, problems of the immunological, endocrine and other system can emerge (Fava and Sonino 2000). This applies in particular to the cardiovascular system, with an elevated heart rate and ischemia, both being potential consequences of exposure to prolonged stress.

Eibner and Evans (2005) suggest that a substantial part of the decline in mortality that is associated with an increase in absolute income is due to the *relative deprivation effect*. To the extent that this is correct, health benefits from an increased income is more likely to originate from psychological well-being, caused by a comparison with one's peers, than due to the increase in the individual's consumption capabilities. Evidence of the existence of a physiological response to relative status was provided by studies on nonhuman primates. Sapolsky (2005) examined the physical response of primates who were exposed to shifts in the hierarchy of their social group. The study found a number of responses, including hypertension, elevated heart rate and increased circulating levels of lipids and cholesterol, which are all risk factors for CVD. The study found that all risk factors

increased among those exposed to stressful situations. In human populations, studies have repeatedly found a positive health effect that is associated with the individual's relative position, measured in terms of the position in the income hierarchy. Increased health among relatively better earners was found in studies in the US (Subramanyam et al 2009), the UK (Jones and Wildman 2008) and Japan (Kondo et al 2008). In German (Blanco-Perez 2011) and Swedish populations (Yngwe et al 2003) relative income was found to have stronger effects on well-being than absolute income. In Norway, individuals with lower relative income were observed to face higher mortality rates (Elstad, Dahl and Hofoss 2006).

Psychosocial stress originating from relative deprivation could arguably be operationalized in several ways. In this article, the focus is directed towards the individual's relative position in the income distribution, investigating the link between income attainment and the onset of IHD. Here, the individual's attainment is compared with a reference group characterized by the same age, educational attainment and gender – all relevant labor market characteristics. According to the hypothesis, individuals who find themselves in the lower end of the relative income distribution are likely to suffer from psychosocial stress, resulting from a feeling of performing inadequately. Thus, the hypothesis hinges upon the assumption that an individual by and large compares their own performance to peers who share their most relevant characteristics, rather than to the population as a whole.

The greater the negative divergence between the actual and the expected performance, the greater would be the feeling of relative deprivation. To the extent that relative deprivation is related to psychosocial stress, this could, consequently, act as a trigger for IHD. On the other hand, individuals who perform better than a comparable reference group could benefit from a feeling of relative satisfaction (Ball and Chernova 2008; Blanco-Perez 2011), resulting in corresponding health benefits.

A key distinction between the absolute and relative deprivation hypotheses is, that it is only the latter that explicitly accounts for the assumption that individuals are aware of how they should be performing, and that they can and do compare their achievement with others. Whereas two individuals with identical incomes according to the absolute deprivation hypothesis are assumed to have equal access to health promoting goods, the relative deprivation hypothesis distinguishes between a janitor with appropriate training and a janitor trained as a medical doctor. Whereas the former could be expected to be content with their outcome according to the relative deprivation hypothesis, the latter should be less likely to do so.

Regardless if an individual's income attainment is measured in absolute or relative terms, properly modeling its effect on the risk for IHD emerges as

potentially complex. An individual's income attainment is dynamic from year to year, and measuring the economic status of an individual at one point in time only captures a snapshot of their performance. Furthermore, a long-term perspective of an individual's economic performance arguably provides a more accurate picture of the individual's experienced circumstances, net of cyclical variations in the income attainment.

Several of the outlined mechanisms which link the individual's income attainment to the risk for IHD, furthermore suggest the relevance of taking the long-term rather than the short-term exposure into account. A sudden negative income shock may cause a considerable, albeit temporary, physical reaction, including an elevated heart rate, arrhythmia or psychological distress. However, the initiated reaction triggered by stress is a protection mechanism and aims at adapting the physical and psychological functions to work properly even under the influence of acute stress (McEwen 2002).

However, the physiological adaptation process may result in a permanent and hazardous malfunction if the stressor remains over a longer period of time, since the body only is designed to function under a high stress load for a limited time. This was also indicated in previous research, suggesting that stress that negatively influences the individual's health is more likely to result from a long-term, as opposed to short-term, exposure to economic hardship (Hansson 2005). The relevance of taking a long-term perspective when considering the influence of income attainment would also appear to apply regarding their influence on various risk factors for CVD as well. More specifically, the consequences of poor dietary habits, physical inactivity, smoking and alcohol consumption are known to influence the risk for CVD risk factors, such as overweight and diabetes (Holmes, Ekkekakis and Eisenmann 2010) as well as hypertension (Hansen et al 2007) and ischemia of the heart (Nauman et al 2010), particularly in the medium to long run.

Besides the link between income attainment in the short, medium and long run and the risk for IHD, the incidence of CVD in general differs substantially between men and women, given a certain age group. Women suffer from IHD later in life than men, assumedly partly due to a hormonal advantage caused by the reproductive functions (Kajantie and Phillips 2006). Therefore, when considering individuals in working ages, women typically experience a lower IHD incidence rate than men.

Thus, in the study population of this article, the baseline risk for IHD for women is markedly lower than among men. While this appears to be strongly linked to biological factors, between-gender differences in the experience of and coping with work-related stress potentially offers interesting explanations. The individual's income attainment is operationalized as income from work,

implying that the individual's position in the relative and absolute income distribution essentially is linked to their performance in the workplace. Ample previous research has shown differences between sexes in how work-related stress is perceived as well as coped with (Day and Livingstone 2003; Eaton and Bradley 2008; Matud 2004; Torkelson and Muhonen 2004). Among women in particular, the main sources of stress appear to be strongly connected to structures in the labor market and difficulties in dealing with traditional gender roles concerning the woman's role as a caregiver. Women typically carry a greater domestic workload, consisting of tasks which are to a great extent time-inflexible. More specifically, the typical female-tasks often need to be carried out despite the existence of other interferences, potentially making it difficult to relax after work and therefore causing fatigue and, potentially, ill health (Nelson and Burke 2002). Thus, regardless of the woman's work situation, they appear more likely to experience a feeling of work-overload due to their domestic duties, potentially translating in to psychological stress.

In the labor market, countless studies have suggested the existence of a glass-ceiling, implying that women face obstacles from attaining the most elevated positions in the labor market. The explanations to the phenomenon are numerous, including the existence of recruitment and promotion processes where women are disadvantaged (Baumgartner and Schneider 2010). As a consequence, it could be expected that women who indeed manage to attain such elevated positions are required to put in a considerable workload to overcome those obstacles, potentially even more than men in a similar position. Furthermore, the effort required to maintain the attained position may also be considerable.

This line of thought would suggest a greater prevalence of work-related stressors experienced by women. While women report more psycho-physiological symptoms, including insomnia, nervousness and sleeping disorders, the symptoms resulting from work stress displayed by men tend to be more severe. One explanation to this phenomenon is the fact that women to a greater extent seek medical help for their problems at a comparatively early stage, while men frequently internalize stress and fail to get medical treatment in time (Nelson and Burke 2002).

## 3.4. Data and Methods

In the article, a sample of approximately 90,000 men and women is analyzed during the time period 1992-2001. The study population of individuals between

the ages 40-65 was selected from the Swedish Longitudinal Immigrant database (SLI), administered at the Centre for Economic Demography, Lund University, Sweden. The SLI is a register-based database, containing a representative sample of Swedes as well as immigrants from over fifty unique countries of origin. As a result of the design of the database, individual level demographic, economic and health-related information is available with a high level of detail and chronological precision, resulting in a truly longitudinal database.

The information, required to operationalize the onset of IHD, is provided by the hospital discharge register. The register not only allows for the recording of up to nine simultaneous diagnoses, coded according to ICD9/10, but also the date of hospital admission and discharge. A potential caveat associated with the hospital discharge register is, however, that any visit to the hospital that was not followed by the individual being admitted is not observed in the data. Due to the nature of the disease of interest, typically requiring extensive hospital care, this feature is not believed to cause any noteworthy under-reporting of IHD events.

A key feature of this article is the explicit focus on the determinants of the onset of IHD, rather than indiscriminately, focusing on any IHD event. This is arguably an essential distinction, due to the nature of the disease. More specifically, the survival as well as recurrence rate of IHD is high in developed countries. According to the SLI, the median time between subsequent IHD events in the same individual amounts to less than a year. Indeed, only five percent of reoccurring IHD events occurs more than five years subsequent to the previous event. While a substantial share of individuals suffering from IHD survives, the IHD event may nevertheless have important short as well as long run consequences. Of particular relevance for the approach of this article is the risk that the individual's work capacity may be temporarily or permanently reduced as a result of the prior experience of IHD, thereby causing a corresponding income decline.

The principal interest of the article concerns the potential link between the individual's short, medium, and long-term income attainment and the onset of IHD. Based on the discussion above, it is imperative to consider the potential reverse causality issues that may arise in an indiscriminant analysis of IHD events, in attempting to approach a causal estimation of such a link. To the extent that having previously experienced IHD acts as a predictor for future IHD events at the same time as it causes diminished income attainment. Finding a negative relationship between income attainment and IHD risk should emerge as a result of the endogenous process. In attempting to circumvent this potentially substantial empirical problem, the strategy of this article consists of examining a sample of individuals who have no prior history of IHD. In doing so, reverse causality issues should largely be avoided, thereby increasing the likelihood of obtaining an

unbiased empirical estimation of the relationship between the individual's short, medium and long-term income attainment and the onset of IHD.

The hospital discharge register provides information on the individual's disease history from 1987 and onwards, meaning that IHD events occurring in or before 1986 are unobserved. In order to minimize the risk that an individual who have experienced an IHD event before 1987 are included in the sample, individuals are only included in the sample if they are IHD-free for no less than five years prior to being considered at risk of experiencing the disease. As a consequence, the follow-up period of the paper stretches from 1992 and until 2001, where individuals considered at risk from 1992 and onwards consequently did not experience IHD during the time period 1987-1991. While it is possible that this procedure fails to identify all left censored cases, the fact that 95 percent of the individuals in the SLI experience a subsequent IHD event within five years should serve to minimize this risk. Having maximized the likelihood that individuals are IHD free when they become at risk of experiencing the onset of IHD, in the empirical analysis, individuals are followed from the age of 40 or later and until what comes first out of i) onset of IHD, ii) the year 2001, iii) turning 65 years of age, iv) transitioning into retirement, or v) censoring due to no further observations.

The study population consists of individuals in the ages between 40 and 65. While IHD is a recurring disease among the survivors, its onset predominantly occurs in the ages past 65. This also becomes evident in the sample analyzed in this article, where only 1,583 of the approximately 90,000 included individuals experience their first IHD during the time of observation. While this figure may appear small, especially given the importance of IHD as a cause of death in the population as a whole, it corresponds well to macro estimates provided by the Swedish Board of Health and Welfare (Socialstyrelsen 2011a). However, focusing on the economically active share of the population, makes the limitation of individuals under the age of 65 an essential part of the data preparation.

The theoretical section hypothesizes that exposure to poor labor market performance or economic stress, either in absolute or relative terms, may cause physiological responses which trigger the onset of IHD. Primarily based on à priori expectations regarding the individual's ability to cope with various forms of stress, it is also discussed whether the focus should be directed towards the importance of short-term or prolonged insults. The SLI contains records from the tax register, providing detailed information regarding the individual's income obtained from various sources on a yearly basis. In this article, individual's income from work will be used, including mainly income from wage, but as well from work-related sources such as benefits from unemployment and sickness absence. This study is

operationalizing economic stress as lower labor market performance, measured as low income.

In order to distinguish short, medium and long-term stress this study uses income obtained during the year prior to the year at risk for IHD as well as average income over five and ten consecutive years prior to the year at risk. While the first measurement is sensitive to short-term fluctuations in the individual's income attainment, it remains temporally separated from the outcome of interest, since the income by definition is obtained prior to the year when the individual is at risk of experiencing IHD. The medium and long-term income attainment of the individual are designed to minimize the influence of short-term fluctuations. As a consequence, individuals must be observed with non-missing income observations during all of the ten-years prior to the year at risk in order to be included in the sample. While this restriction aims at including only individuals with complete income record and therefore continuous economic performance, it will exclude cases where at least one year of income information is missing, regardless the reason. All income information is inflation-adjusted to make records of different periods more comparable.

Based on the income variables, the individual's position in the short, medium and long run, income attainment distribution was calculated. The use of absolute income is primarily motivated by the hypothesis that a given income is associated with corresponding consumption possibilities, where the duration of exposure to a given position in the absolute income distribution may be associated with stress and, consequently, IHD risk. The absolute income variables are inflation adjusted and expressed in 1995 SEK, based on which thresholds denoting the $20^{th}$, $40^{th}$, $60^{th}$ and $80^{th}$ percentile were calculated. Following this, each observation in the sample was allocated to its respective group in the income distribution, in the short, medium or long-term. Similar strategies, albeit with different thresholds, were applied in previous research (see for example Andersen et al 2003; Chaix, Rosvall and Merlo 2007 or Osler et al 2003).

This study also investigates the relevance of the individual's income attainment, viewed in relative terms. More specifically, the relative deprivation hypothesis claims that the individual's position compared with their peers rather than to the population as a whole may be the source of stress and, consequently, IHD risk. In this article, relative income is arguably measured with unprecedented precision, taking a range of relevant individual-level characteristics into account. Based on the total Swedish population, for each year, the mean income was calculated for each unique combination of educational level, educational type, age and gender. Hence, the individual's income is compared with the mean income of those with identical characteristics, and the resulting relative income indicates how well the individual performed compared with otherwise similar individuals.

**Table 1**: Absolute and relative income means for income attainment quintiles

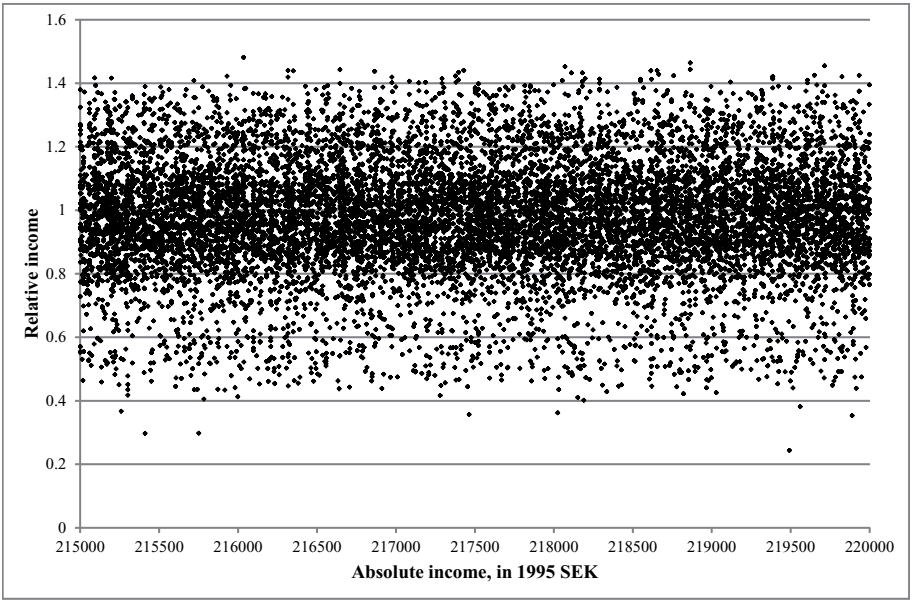
| Percentile: | 0-20 | 20-40 | 40-60 | 60-80 | 80-100 |
|---|---|---|---|---|---|
| Absolute income, time t-1 | 59,485 | 145,640 | 187,214 | 229,133 | 373,837 |
| Absolute income, 5-year-lag-average | 62,240 | 137,359 | 178,604 | 218,729 | 346,447 |
| Absolute income, 10-year-lag-average | 66,340 | 129,554 | 169,346 | 208,002 | 321,855 |
| | | | | | |
| Relative income, time t-1 | 0.27 | 0.68 | 0.87 | 1.03 | 1.45 |
| Relative income, 5-year-lag-average | 0.29 | 0.66 | 0.85 | 1.01 | 1.38 |
| Relative income, 10-year-lag-average | 0.33 | 0.64 | 0.83 | 0.98 | 1.32 |

Source: SLI, own calculations

Note: Absolute income means reported as 1995 SEK

A main prerequisite for the relevance of this paper is naturally that the two income measurements – absolute and relative income – are essentially measuring different things. More specifically, differences in the income distribution between individuals with different levels and types of education should imply that a given absolute income is associated with a range of different relative incomes. Figure 1 shows the distribution of relative incomes around the sample mean value for the absolute income variable (SEK 218,000 in time t-1). The figure clearly suggests a considerable relative income variation at any given value of absolute income. For example; an absolute income of SEK 218,000 in 1995 values is associated with relative incomes ranging between 40 percent and 140 percent.

**Figure 1**: Scatterplot displaying absolute income observations in the interval SEK 215,000-220,000 (1995 values) and the corresponding relative income value.




In the models, several medical and socio-demographic characteristics are included. A binary variable indicates if the individual was diagnosed with hypertension or diabetes in conjunction with a hospital admission. Note that this is likely to cause a considerable under-reporting of these diseases, since they are conditions which typically not require hospital admission and are usually detected while treating another medical problem. As a consequence, individuals observed with these diseases are likely to represent more severe cases and the parameter estimates of both should be overestimating the true effect.

Another potential risk factor, due to being an indicator of morbidity, is represented by previous spells of sickness absence. In deriving spells of sickness absence, yearly information on the receipt of sickness benefits is used. In the case that the benefits received for sickness absence exceed ten percent of the individual's income, the individual is considered to have poor health during the actual year. The variable is operationalized as a set of binary variables, and the models control for sickness absence spells independently in each of the five preceding years. Information on civil status and education is also included, indicating the marital status and highest educational level obtained by the individual in the given year. A dummy variable for living in a metropolitan area as well as the regional unemployment rate is included. The region of origin of the individual is also included in all models measured, distinguishing between individuals place

of birth being either Sweden, the Nordic countries, Europe and other Western countries, or lastly, all remaining countries.

**Table 2**: Distribution of the dependent health variable – first IHD – and the socioeconomic covariates in the sample obtained from the SLI database, presented as mean and standard deviation, stratified by sex

| | **Men** | | **Women** | |
|---|---|---|---|---|
| | Mean | Std.dev | Mean | Std.dev |
| IHD | 0.004 | 0.065 | 0.001 | 0.038 |
| Hypertension | 0.011 | 0.106 | 0.008 | 0.090 |
| Diabetes | 0.010 | 0.100 | 0.005 | 0.073 |
| Sickness absence history: | | | | |
| One year before | 0.088 | 0.284 | 0.120 | 0.324 |
| Two years before | 0.079 | 0.269 | 0.104 | 0.306 |
| Three years before | 0.078 | 0.268 | 0.102 | 0.302 |
| Four years before | 0.084 | 0.277 | 0.109 | 0.311 |
| Five years before | 0.091 | 0.287 | 0.117 | 0.321 |
| Civil status (married=1) | 0.641 | 0.480 | 0.589 | 0.492 |
| Age | 48.8 | 6.096 | 48.2 | 5.925 |
| Highest attained education: | | | | |
| Primary schooling | 0.283 | 0.451 | 0.230 | 0.421 |
| Secondary schooling | 0.426 | 0.495 | 0.425 | 0.494 |
| Tertiary schooling | 0.291 | 0.454 | 0.345 | 0.475 |
| Region of origin: | | | | |
| Sweden | 0.540 | 0.498 | 0.547 | 0.498 |
| Nordic countries | 0.108 | 0.311 | 0.146 | 0.353 |
| Europe and other Western countries | 0.232 | 0.422 | 0.215 | 0.411 |
| Rest of the World | 0.120 | 0.325 | 0.091 | 0.288 |
| Year | 1996.8 | 2.890 | 1996.9 | 2.877 |
| Metropolitan residence (Stockholm/Gothenburg/Malmö) | 0.485 | 0.500 | 0.499 | 0.500 |
| Regional unemployment rate | 8.833 | 2.153 | 8.788 | 2.164 |
| N individuals | 48,676 | | 43,650 | |
| N observations | 285,015 | | 253,175 | |

The structure of the data implies that each individual is observed for a part of its adult life, conditional on the restrictions for sample selection previously reported. Since income is recorded in yearly intervals, the time at risk for the onset of IHD for each individual can be represented between one and ten observations (years). In the latter case, this is represented by an individual who is at risk during all years between 1992 and 2001.

Due to the clustered nature of the data, with repeated observations for each individual, the analysis attempts to cancel out the potentially biasing influence of unobserved and time constant individual characteristics. Due to the infrequent

nature of the outcome variable, fixed effect regression was deemed inappropriate, as the estimates will be based on a subsample displaying within-cluster variation in the dependent variable. Consequently, such an estimation would reduce the sample from approximately 90,000 individuals to less than 2,000. Furthermore, it would also result in an estimation of the link between income attainment and IHD among individuals who at some point experience the disease. Instead, this study chooses to estimate random effect regression models. While this estimation method is associated with other, also cumbersome, assumptions, its ability to take into account the influence of unobserved heterogeneity, combined with utilizing the entire database were considered to be highly desirable.

Due to the dichotomous nature of the dependent variable, the analysis consistently relies on logistic regression models. Furthermore, several studies demonstrated that logistic regressions are an efficient estimator for rare events data, provided that the number of events per independent variable (EPV) in the model exceeds a given threshold. Simulation studies have suggested different thresholds regarding the required events-to-variable-ratio, typically indicating an EPV of 10 as a minimum (Peduzzi et al 1996; Vittinghoff and McCulloch 2007). In this study, the EPV in the models estimated for the men is never below 30 and among women the EPV value never drops below the minimum of 10.

While the models control for a range of determinants of IHD, the SLI database does not include information on several health behaviors that are relevant for IHD risk, such as smoking and alcohol consumption. While these are evidently non-negligible omitted variables, characteristics of the econometric method as well as how they correlate with the key independent variable, however, leads to the assumption that this is less of a problem. First, to the extent that such characteristics can remain constant within a given individual over the time span in which they are observed, the logistic random effect specification would control for this individual set of unobserved characteristics. Should this assumption be violated, which appears to be likely since behavior is of a dynamic nature, it is believed that the omitted variables are negatively correlated with income attainment. As a consequence, the income effect would be overestimated in the models. Therefore, the interpretation of the income effect should be careful to take this overestimation into account.

All models include year dummies and age, making them essentially analogous to duration models. Since the output variable IHD and the variables of interest – absolute and relative income – are strongly influenced by the sex of the individual the analysis is stratified by sex. While the following tables only display the estimates for the income groups, full parameter estimates for all discussed models are presented in their entirety in the Appendix.

## 3.5. Results

Based on the assumption that stress originates from the individual's absolute income attainment due to resulting consumption constraints, the absolute income thresholds applied are identical for the entire sample. Hence, a certain absolute income is here explicitly assumed to cause a similar sense of well-being or stress, regardless of the individual's gender or age. Unsurprisingly, given the typically lower female labor supply, combined with a greater predominance of employment in lower-wage positions, women are over-represented in the lowest absolute income quintiles as displayed in Table 3.

**Table 3**: Sample distribution across absolute income quintiles, by gender in percent

| Absolute income | Time t-1 | | 5-year-lag average | | 10-year-lag average | |
|---|---|---|---|---|---|---|
| quintiles | Men | Women | Men | Women | Men | Women |
| 00-20 | 17.8 | 22.6 | 17.0 | 23.3 | 15.1 | 25.5 |
| 20-40 | 12.5 | 28.4 | 12.3 | 28.6 | 12.8 | 28.1 |
| 40-60 | 17.6 | 22.7 | 17.0 | 23.3 | 16.9 | 23.5 |
| 60-80 | 23.2 | 16.4 | 23.8 | 15.7 | 24.7 | 14.7 |
| 80-100 | 28.9 | 10.0 | 29.8 | 9.0 | 30.5 | 8.2 |
| Total | 100 | 100 | 100 | 100 | 100 | 100 |

Table 4 presents odds ratios for models estimating the effect of absolute income on the probability of experiencing the onset of IHD. In all models, the reference category is represented by individuals in the third income quintile, with absolute incomes between the 40$^{th}$ and 60$^{th}$ percentile.

With a sample which, to a great extent, only consists of previously IHD-free individuals, a rather consistent and gender-specific pattern emerges at all income lags. Among men, a negative relationship between income attainment and the odds of experiencing the onset of IHD appears to be emerging. Applying 95 percent confidence intervals, it does, however, remain an impossibility to distinguish any of the odds ratios from one. Given the infrequency of the outcome of interest, the lack of statistical significance emerges as a partially expected consequence. Therefore, attention should still be directed towards the direction and size of the parameter estimates. From the odds ratios, it can be observed that men belonging to the highest income quintile on average enjoy odds of experiencing IHD onset that hover about 30 percentage points below that observed among the lowest income quintile, regardless of whether it is a condition that has lasted in the short, medium or long-term.

**Table 4**: Random effect logistic regression on first IHD events, impact from income quintiles in 1987 inflation adjusted SEK, 48,676 men and 43,650 women

| **MEN** | Model 1 Time t-1 | | Model 2 5-year-lag average | | Model 3 10-year-lag average | |
|---|---|---|---|---|---|---|
| Absolute income quintile | OR | 95 % confidence interval | OR | 95 % confidence interval | OR | 95 % confidence interval |
| 00-20 | 1.307 * | (0.998 - 1.712) | 1.297 * | (0.965 - 1.742) | 1.137 | (0.830 - 1.558) |
| 20-40 | 1.126 | (0.856 - 1.482) | 1.267 | (0.950 - 1.688) | 1.232 | (0.916 - 1.657) |
| 40-60 | ref | | ref | | ref | |
| 60-80 | 1.148 | (0.896 - 1.469) | 1.087 | (0.837 - 1.411) | 0.864 | (0.661 - 1.130) |
| 80-100 | 0.956 | (0.717 - 1.275) | 0.884 | (0.654 - 1.195) | 0.762 * | (0.562 - 1.031) |

| **WOMEN** | Model 4 Time t-1 | | Model 5 5-year-lag average | | Model 6 10-year-lag average | |
|---|---|---|---|---|---|---|
| Absolute income quintile | OR | 95 % confidence interval | OR | 95 % confidence interval | OR | 95 % confidence interval |
| 00-20 | 1.263 | (0.752 - 2.121) | 1.033 | (0.593 - 1.801) | 1.224 | (0.680 - 2.203) |
| 20-40 | 0.892 | (0.560 - 1.421) | 0.654 * | (0.395 - 1.084) | 1.190 | (0.715 - 1.980) |
| 40-60 | ref | | ref | | ref | |
| 60-80 | 1.426 | (0.858 - 2.371) | 0.900 | (0.510 - 1.590) | 1.334 | (0.744 - 2.394) |
| 80-100 | 1.444 | (0.715 - 2.914) | 1.425 | (0.675 - 3.005) | 1.742 | (0.806 - 3.764) |

Notes: Full model estimates are reported in Table A1 and A2, Appendix
*** 1%, ** 5%, * 10% statistical significance

Among women, the results confirm an on average increasing IHD risk among the lowest quintile of absolute income, particularly accentuated at time t-1 and for the 10-year-lag average. However, the effects fail to be significant at the five percent level, why it remains difficult to draw any substantial conclusions regarding to what extent low income earning women de facto are associated with an elevated risk for IHD onset.

At the other end of the absolute income distribution, an interesting development can be observed among the highest earning women who, similar to the lowest performers, experience an elevated IHD risk. This is particularly accentuated in the long-term perspective, suggesting a 74 percent increase in the odds of the onset of IHD, compared with the reference category. This could possibly be partially attributed to different gender roles facing men and women, both domestically and in the labor market. Despite enjoying higher average levels of education, women, to a considerable extent, fail in obtaining incomes in the highest quintile, as indicated in Table 3. While women on average take a greater responsibility for childcare and other domestic duties, which could impede the individual's career prospects due to the reduced labor supply. As one progresses up an occupational hierarchy, one is likely to be facing an environment that is becoming increasingly male dominated. A phenomenon which was even more valid at the time of the observation period (Arber and Ginn 1995) than it is today. In competing in such an environment, it is likely that women to a much

greater extent have to prove themselves worthy of an elevated position. Hence, the work effort associated with enjoying a position in the highest part of the income distribution over the past ten years is likely to be associated with a considerable workload. Therefore, the estimates could be suggestive of a situation where women who attain and maintain a very high income do so at the expense of exposing themselves to a particularly stressful and persisting situation. While the parameter estimates remain statistically non-significant, this is likely to – at least partially – be due to the low baseline risk for women in the analyzed age range.

Turning to the analysis of the relationship between relative income and the risk for IHD onset, this essentially hinges upon the expectation that income in relative terms measures something fundamentally different than income in absolute terms. While men and women displayed differing patterns according to the absolute income distribution, those differences by and large disappear when considering the concept of relative income, as displayed in Table 5. The most evident change emerges in the lowest and highest quintiles, where men and women now appear to be represented by about equally large proportions. Again, the within-gender pattern remains largely consistent regardless of the timing of the relative income measurement.

**Table 5**: Sample distribution across relative income quintiles, by gender in percent

| Relative income | Time t-1 | | 5-year-lag average | | 10-year-lag average | |
|---|---|---|---|---|---|---|
| quintiles | Men | Women | Men | Women | Men | Women |
| 00-20 | 21.8 | 18.0 | 21.3 | 18.6 | 20.2 | 19.8 |
| 20-40 | 20.8 | 19.2 | 20.1 | 19.9 | 19.3 | 20.8 |
| 40-60 | 19.5 | 20.6 | 19.8 | 20.3 | 19.7 | 20.4 |
| 60-80 | 17.7 | 22.6 | 18.3 | 21.9 | 19.7 | 20.4 |
| 80-100 | 20.3 | 19.7 | 20.5 | 19.4 | 21.2 | 18.7 |
| Total | 100 | 100 | 100 | 100 | 100 | 100 |

Table 6 displays odds ratios for the relationship between the individual's position in the relative income distribution, in the short, medium and long-term, and the risk for IHD onset for men and women. Among men, an expected pattern emerges, suggesting an increasing risk for IHD onset that is associated with a lower relative income. More interestingly, the estimates appear to suggest a pattern that is accentuating with the duration of the actual condition. Compared with the reference category, a relative income belonging to the lowest quintile is associated with an increased risk of about seven percent when only the relative income during the previous year is taken into account. Extending the duration of this condition to five and ten years causes the risk to increase to 15 and 23 percent, respectively. Again, however, the size of the confidence intervals makes

it impossible to separate the effects of the reference category using a 95 percent confidence interval, thus implying a considerable degree of uncertainty regarding the relevance of the estimate.

**Table 6**: Random effect logistic regression on first IHD events, impact from relative income quintiles, adjusted for age, sex, education and year, 48,676 men and 43,650 women

| **MEN** | Model 7 Time t-1 | | Model 8 5-year-lag average | | Model 9 10-year-lag average | |
|---|---|---|---|---|---|---|
| Relative income centiles | OR | 95 % confidence interval | OR | 95 % confidence interval | OR | 95 % confidence interval |
| 00-20 | 1.072 | (0.828 - 1.387) | 1.146 | (0.873 - 1.504) | 1.227 | (0.922 - 1.632) |
| 20-40 | 1.015 | (0.794 - 1.297) | 0.992 | (0.767 - 1.282) | 0.985 | (0.752 - 1.291) |
| 40-60 | ref | | ref | | ref | |
| 60-80 | 0.909 | (0.703 - 1.175) | 0.765 * | (0.581 - 1.007) | 0.762 * | (0.576 - 1.009) |
| 80-100 | 0.842 | (0.632 - 1.120) | 0.834 | (0.621 - 1.121) | 0.769 * | (0.566 - 1.044) |

| **WOMEN** | Model 10 Time t-1 | | Model 11 5-year-lag average | | Model 12 10-year-lag average | |
|---|---|---|---|---|---|---|
| Relative income centiles | OR | 95 % confidence interval | OR | 95 % confidence interval | OR | 95 % confidence interval |
| 00-20 | 0.941 | (0.540 - 1.640) | 1.521 | (0.820 - 2.821) | 0.936 | (0.502 - 1.747) |
| 20-40 | 0.623 * | (0.364 - 1.064) | 0.988 | (0.559 - 1.746) | 0.864 | (0.498 - 1.501) |
| 40-60 | | | ref | | ref | |
| 60-80 | 0.853 | (0.522 - 1.394) | 1.419 | (0.830 - 2.426) | 0.852 | (0.499 - 1.456) |
| 80-100 | 0.976 | (0.563 - 1.694) | 1.392 | (0.755 - 2.566) | 1.271 | (0.714 - 2.262) |

Notes: Full model estimates are reported in Table A3 and A4, Appendix
*** 1%, ** 5%, * 10% statistical significance

At the other end of the relative income distribution, a similar accentuation of the advantage experienced by the highest earners, as indicated by the parameter estimates, occurs. Compared with the reference category, the risk for IHD onset, measuring relative income during the previous year and as the 10-year-lag average is 16 and 23 percent lower, respectively. While not statistically significant using 95 percent confidence intervals, the advantage from being in the top two relative income quintiles in the long-term is significant at the ten percent level.

Among women, the pattern emerges as considerably more erratic, partly attributable to the on average lower degree of precision with which the parameters are estimated. Regardless of whether the stressor is considered during the previous year or as a ten-year-lagged average, the estimated pattern shows no clear tendency as regards the importance of one's position in the relative income distribution.

The possible exception to an otherwise erratic pattern is represented by women belonging to the highest relative income quintile during the previous five or ten years, who are characterized by odds ratios exceeding one, indicating an elevated risk for IHD onset. Given the results from the analysis of absolute income, this does not, however, emerge as unexpected. Here, it needs to be underlined that

the women in this category are those enjoying incomes being 112 percent or higher than expected, given their sex, age, educational type and educational level. As a consequence, while a high relative income is synonymous with the attainment of a high income, it is not necessarily equivalent with a high absolute income. Considering women's averaged lower labor force participation and assumed greater domestic responsibilities, the attainment of a relative income in the uppermost quintile is likely to be associated with a considerable extra effort, potentially combined with a fair amount of psychosocial stress.

Among remaining parameters, in the short as well as in the long run, the individual's position in the relative income distribution appears to be unrelated to the risk for IHD onset. More specifically, there is no indication that would suggest that being in the lowest relative income quintile, either in the short or the long run, is associated with an elevated risk for IHD onset. The possible exception is observed using the five-year-lag average relative income, suggesting a U-shaped pattern concerning the IHD risk.

A possible explanation could be the transitional process of adaptation. While a low income in the short-term might be buffered by other resources, exposure to such a situation over a time period of five years might be beyond the individual's coping capabilities. Further short-term stress might not be perceived as detrimental by the individual if enough psychosocial support is available. As a consequence, stress from economic hardship would be more prominent in the medium run than in the short run, when economical and psychosocial resources are exhausted. However, in case the situation persist in the long run, over at least ten years, the individual's consumption and living style possibly has adjusted to the new level of income since the situation is accepted as being permanent.

## 3.6. Conclusions

Contrary to the results of several other studies, those obtained in this article do not suggest a strong and consistent impact from the individual's income attainment on the onset of Ischemic Heart Disease (IHD). Examining a large longitudinal population in Sweden between the years 1992-2001, the study analyzes the relationship between the individual's position in the absolute and relative income hierarchy on the onset of IHD. The contributions of the article are not only attributable to its empirical results, but also in highlighting the importance of a sound study design in order to approach estimates that may be interpreted in causal terms.

This article examines a sample of individuals without an IHD history prior to the observation period. Their short, medium and long-term income attainment is tracked in order to estimate their role in the onset of IHD. In the age range of the study population, IHD remains a rare event, afflicting only about two percent of the study population. Despite the infrequency of events, certain parameters are observed to consistently be important determinants of IHD onset, such as being diagnosed with hypertension or diabetes. As regards the role of income attainment, the article focuses on two separate concepts; absolute and relative income. Whereas the former concept refers to the individual's position in the absolute income hierarchy, indicating the individual's objectively defined consumption capabilities, the latter measurement is intended to gauge the relative deprivation hypothesis. Furthermore, following theory, links are examined over longer time periods, as exposure to long-term stress could have different implications than stress experienced only in the short-term.

The general conclusion of the article can be none other than to question the existence of a causal link between the individual's income attainment and the risk for IHD onset – either for absolute or relative income. Applying standard 95 percent confidence intervals, the models consistently fail to find statistically significant links between the individual's position in the income hierarchy and the risk for IHD. Despite the lack of statistical significance, a few patterns emerge which could imply the existence of a link, albeit a weak one. Among men, enjoying absolute or relative incomes belonging to the uppermost quintile appears to be associated with a lowered risk of experiencing an IHD event. Furthermore, this advantage appears to be increasing slightly with the duration of this condition.

Interestingly, the opposite situation seems to apply to women, where belonging to the top quintile in the medium or long-term on average is associated with an increased risk for IHD. While seemingly counterintuitive due to the presumed benefits associated with enjoying favorable outcomes in the labor market, this tendency could be explained by women facing larger challenges in achieving, as well as maintaining, an elevated position in the labor market. In this study women are only compared with other women, what especially for the relative income makes a difference. A future project could make the assumption that women potentially suffer from relative deprivation if compared with men who have comparable characteristic, entering the discussion of gender equality in the labor market.

Furthermore, the worst performing males are typically observed with parameter estimates that would suggest an elevated IHD risk. Although this presumably linear income effect for men fulfills the aforementioned expectations, almost none of the estimates taken prior the year of the event are significantly different from the reference group. Therefore, the results must remain inconclusive, what stands

in opposition to the findings of other studies. Therefore, this study reopens the discussion of the direct effects of income differences on health in general and the onset of IHD in particular.

## 3.7. References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# 3.8. Appendix

**Table A1**: Complete estimates, Models 1-3. Beta-coefficients.

| Men | Model 1 Time t-1 | | Model 2 5-year-lag average | | Model 3 10-year-lag average | |
|---|---|---|---|---|---|---|
| | Coef. | 95% CI | Coef. | 95% CI | Coef. | 95% CI |
| Absolute income centile | | | | | | |
| 00-20 | 0.268* | (-0.002 - 0.538) | 0.260* | (-0.035 - 0.555) | 0.128 | (-0.186 - 0.443) |
| 20-40 | 0.119 | (-0.155 - 0.393) | 0.236 | (-0.051 - 0.524) | 0.209 | (-0.088 - 0.505) |
| 40-60 | ref | | ref | | ref | |
| 60-80 | 0.138 | (-0.110 - 0.385) | 0.083 | (-0.178 - 0.344) | -0.146 | (-0.414 - 0.122) |
| 80-100 | -0.045 | (-0.333 - 0.243) | -0.123 | (-0.424 - 0.178) | -0.272* | (-0.575 - 0.031) |
| Hypertension | 4.769*** | (4.349 - 5.188) | 4.832*** | (4.412 - 5.253) | 4.783*** | (4.362 - 5.205) |
| Diabetes | 3.295*** | (2.819 - 3.771) | 3.333*** | (2.852 - 3.813) | 3.301*** | (2.823 - 3.778) |
| Sickness absence history | | | | | | |
| One year before | 0.517*** | (0.292 - 0.743) | 0.461*** | (0.186 - 0.735) | 0.452*** | (0.179 - 0.725) |
| Two years before | | | 0.206 | (-0.227 - 0.640) | 0.198 | (-0.234 - 0.630) |
| Three years before | | | -0.002 | (-0.637 - 0.633) | -0.001 | (-0.633 - 0.632) |
| Four years before | | | -0.219 | (-1.154 - 0.715) | -0.231 | (-1.161 - 0.700) |
| Five years before | | | -0.146 | (-1.163 - 0.871) | -0.143 | (-1.156 - 0.869) |
| Civil status (married ==1) | 0.038 | (-0.165 - 0.241) | 0.049 | (-0.157 - 0.255) | 0.053 | (-0.151 - 0.258) |
| Age | 0.875*** | (0.637 - 1.112) | 0.894*** | (0.654 - 1.134) | 0.888*** | (0.651 - 1.126) |
| Age squared | -0.007*** | (-0.009 - -0.004) | -0.007*** | (-0.009 - -0.005) | -0.007*** | (-0.009 - -0.005) |
| Highest attained education | | | | | | |
| Primart schooling | 0.780*** | (0.467 - 1.093) | 0.749*** | (0.432 - 1.066) | 0.728*** | (0.413 - 1.043) |
| Secondary schooling | 0.659*** | (0.366 - 0.952) | 0.641*** | (0.344 - 0.937) | 0.627*** | (0.332 - 0.921) |
| Tertiary schooling | ref | | ref | | ref | |
| Metropolitan residence | -0.133 | (-0.348 - 0.081) | -0.133 | (-0.350 - 0.085) | -0.126 | (-0.342 - 0.090) |
| Regional unemployemnet rate | 0.037** | (0.004 - 0.069) | 0.0363** | (0.004 - 0.069) | 0.036** | (0.003 - 0.068) |
| Region of origin | | | | | | |
| Sweden | ref | | ref | | ref | |
| Nordic countries | 0.278* | (-0.0392 - 0.595) | 0.272* | (-0.0485 - 0.593) | 0.266 | (-0.0522 - 0.585) |
| Europe and Western countries | 0.345*** | (0.0881 - 0.603) | 0.321** | (0.0595 - 0.582) | 0.301** | (0.0401 - 0.562) |
| Rest of the World | 0.022 | (-0.330 - 0.374) | -0.0305 | (-0.389 - 0.329) | -0.078 | (-0.439 - 0.283) |
| Years | | | | | | |
| 1992 | -0.659*** | (-1.055 - -0.263) | -0.686*** | (-1.085 - -0.287) | -0.685*** | (-1.083 - -0.288) |
| 1993 | -0.286* | (-0.623 - 0.051) | -0.289* | (-0.628 - 0.050) | -0.300* | (-0.638 - 0.038) |
| 1994 | 0.005 | (-0.301 - 0.312) | 0.006 | (-0.302 - 0.314) | 0.000 | (-0.307 - 0.307) |
| 1995 | ref | | ref | | ref | |
| 1996 | 0.271* | (-0.031 - 0.573) | 0.263* | (-0.040 - 0.566) | 0.267* | (-0.035 - 0.570) |
| 1997 | 0.257 | (-0.062 - 0.577) | 0.245 | (-0.076 - 0.566) | 0.250 | (-0.070 - 0.569) |
| 1998 | 0.367** | (0.024 - 0.710) | 0.360** | (0.015 - 0.705) | 0.362** | (0.019 - 0.706) |
| 1999 | 0.336* | (-0.023 - 0.694) | 0.331* | (-0.030 - 0.691) | 0.330* | (-0.029 - 0.689) |
| 2000 | 0.402** | (0.024 - 0.780) | 0.397** | (0.017 - 0.777) | 0.388** | (0.010 - 0.767) |
| 2001 | 0.289 | (-0.111 - 0.689) | 0.281 | (-0.122 - 0.683) | 0.263 | (-0.139 - 0.664) |
| Constant | -38.81*** | (-45.41 - -32.21) | -39.49*** | (-46.14 - -32.85) | -39.05*** | (-45.62 - -32.47) |
| N of Individuals | 48,676 | | 48,676 | | 48,676 | |
| N of Observations | 285,015 | | 285,015 | | 285,015 | |

*** p<0.01, ** p<0.05, * p<0.1

**Table A2**: Complete estimates, Models 4-6. Beta-coefficients.

| Women | Model 4 Time t-1 | | Model 5 5-year-lag average | | Model 6 10-year-lag average | |
|---|---|---|---|---|---|---|
| | Coef. | 95% CI | Coef. | 95% CI | Coef. | 95% CI |
| Absolute income centile | | | | | | |
| 00-20 | 0.234 | (-0.285 - 0.752) | 0.033 | (-0.522 - 0.588) | 0.202 | (-0.385 - 0.790) |
| 20-40 | -0.114 | (-0.580 - 0.351) | -0.424* | (-0.930 - 0.081) | 0.174 | (-0.336 - 0.683) |
| 40-60 | ref | | ref | | ref | |
| 60-80 | 0.355 | (-0.153 - 0.863) | -0.105 | (-0.674 - 0.464) | 0.288 | (-0.296 - 0.873) |
| 80-100 | 0.367 | (-0.335 - 1.069) | 0.354 | (-0.393 - 1.100) | 0.555 | (-0.216 - 1.325) |
| Hypertension | 7.983*** | (7.228 - 8.739) | 8.682*** | (7.897 - 9.467) | 8.446*** | (7.669 - 9.223) |
| Diabetes | 4.160*** | (2.875 - 5.444) | 4.332*** | (2.941 - 5.723) | 4.255*** | (2.895 - 5.616) |
| Sickness absence history | | | | | | |
| One year before | 0.857*** | (0.461 - 1.252) | 0.723*** | (0.237 - 1.209) | 0.704*** | (0.222 - 1.186) |
| Two years before | | | 0.697* | (-0.001 - 1.396) | 0.674* | (-0.019 - 1.368) |
| Three years before | | | -0.900 | (-2.144 - 0.344) | -0.915 | (-2.152 - 0.321) |
| Four years before | | | -0.410 | (-2.323 - 1.504) | -0.331 | (-2.219 - 1.557) |
| Five years before | | | 0.901 | (-1.064 - 2.866) | 0.820 | (-1.108 - 2.748) |
| Civil status (married ==1) | 0.091 | (-0.309 - 0.491) | 0.099 | (-0.319 - 0.518) | 0.087 | (-0.326 - 0.499) |
| Age | 1.634*** | (1.089 - 2.180) | 1.799*** | (1.206 - 2.392) | 1.758*** | (1.182 - 2.335) |
| Age squared | -0.013*** | (-0.018 - -0.008) | -0.015*** | (-0.020 - -0.009) | -0.014*** | (-0.020 - -0.009) |
| Highest attained education | | | | | | |
| Primart schooling | 0.917*** | (0.293 - 1.540) | 0.932*** | (0.268 - 1.596) | 0.915*** | (0.261 - 1.569) |
| Secondary schooling | 1.042*** | (0.475 - 1.609) | 1.055*** | (0.448 - 1.661) | 1.044*** | (0.447 - 1.641) |
| Tertiary schooling | ref | | ref | | ref | |
| Metropolitan residence | -0.119 | (-0.551 - 0.313) | -0.123 | (-0.577 - 0.330) | -0.115 | (-0.562 - 0.333) |
| Regional unemployemnet rate | 0.067** | (0.002 - 0.131) | 0.071** | (0.003 - 0.138) | 0.069** | (0.002 - 0.135) |
| Region of origin | | | | | | |
| Sweden | ref | | ref | | ref | |
| Nordic countries | 0.704*** | (0.174 - 1.234) | 0.710** | (0.155 - 1.265) | 0.712** | (0.165 - 1.259) |
| Europe and Western countries | 0.089 | (-0.470 - 0.649) | 0.08 | (-0.509 - 0.669) | 0.091 | (-0.491 - 0.672) |
| Rest of the World | 0.329 | (-0.441 - 1.099) | 0.307 | (-0.508 - 1.121) | 0.293 | (-0.513 - 1.098) |
| Years | | | | | | |
| 1992 | -1.032** | (-1.945 - -0.119) | -1.198** | (-2.169 - -0.228) | -1.142** | (-2.093 - -0.190) |
| 1993 | -0.295 | (-1.040 - 0.450) | -0.381 | (-1.158 - 0.396) | -0.357 | (-1.123 - 0.410) |
| 1994 | 0.144 | (-0.514 - 0.803) | 0.138 | (-0.537 - 0.813) | 0.135 | (-0.534 - 0.805) |
| 1995 | ref | | ref | | ref | |
| 1996 | 0.722** | (0.092 - 1.351) | 0.726** | (0.078 - 1.374) | 0.727** | (0.085 - 1.368) |
| 1997 | 0.950*** | (0.296 - 1.605) | 1.072*** | (0.398 - 1.745) | 1.038*** | (0.371 - 1.704) |
| 1998 | 0.600 | (-0.151 - 1.352) | 0.723* | (-0.051 - 1.496) | 0.699* | (-0.065 - 1.463) |
| 1999 | 1.125*** | (0.385 - 1.864) | 1.267*** | (0.504 - 2.031) | 1.249*** | (0.495 - 2.002) |
| 2000 | 1.110*** | (0.321 - 1.899) | 1.277*** | (0.462 - 2.091) | 1.252*** | (0.448 - 2.056) |
| 2001 | 1.063** | (0.229 - 1.897) | 1.224*** | (0.362 - 2.085) | 1.203*** | (0.353 - 2.052) |
| Constant | -68.67*** | (-83.55 - -53.78) | -74.69*** | (-91.01 - -58.37) | -73.26*** | (-89.10 - -57.43) |
| N of Individuals | 43,650 | | 43,650 | | 43,650 | |
| N of Observations | 253,175 | | 253,175 | | 253,175 | |

*** p<0.01, ** p<0.05, * p<0.1

**Table A3**: Complete estimates, Models 7-9. Beta-coefficients.

| Men | Model 7 Time t-1 | | Model 8 5-year-lag average | | Model 9 10-year-lag average | |
|---|---|---|---|---|---|---|
| | Coef. | 95% CI | Coef. | 95% CI | Coef. | 95% CI |
| Relative income centile | | | | | | |
| 00-20 | 0.069 | (-0.189 - 0.327) | 0.136 | (-0.136 - 0.408) | 0.204 | (-0.081 - 0.490) |
| 20-40 | 0.015 | (-0.231 - 0.260) | -0.008 | (-0.265 - 0.248) | -0.015 | (-0.285 - 0.255) |
| 40-60 | ref | | ref | | ref | |
| 60-80 | -0.095 | (-0.352 - 0.162) | -0.268* | (-0.543 - 0.007) | -0.272* | (-0.552 - 0.008) |
| 80-100 | -0.172 | (-0.458 - 0.113) | -0.181 | (-0.476 - 0.114) | -0.263* | (-0.569 - 0.043) |
| Hypertension | 4.704*** | (4.280 - 5.128) | 4.663*** | (4.236 - 5.090) | 4.752*** | (4.328 - 5.175) |
| Diabetes | 3.251*** | (2.777 - 3.725) | 3.223*** | (2.750 - 3.696) | 3.284*** | (2.807 - 3.760) |
| Sickness absence history | | | | | | |
| One year before | 0.505*** | (0.282 - 0.729) | 0.456*** | (0.186 - 0.727) | 0.452*** | (0.180 - 0.725) |
| Two years before | | | 0.196 | (-0.233 - 0.625) | 0.202 | (-0.229 - 0.633) |
| Three years before | | | -0.003 | (-0.630 - 0.624) | -0.006 | (-0.637 - 0.625) |
| Four years before | | | -0.219 | (-1.140 - 0.703) | -0.217 | (-1.145 - 0.711) |
| Five years before | | | -0.138 | (-1.139 - 0.862) | -0.143 | (-1.152 - 0.865) |
| Civil status (married ==1) | 0.035 | (-0.167 - 0.236) | 0.045 | (-0.156 - 0.246) | 0.058 | (-0.146 - 0.261) |
| Age | 0.846*** | (0.612 - 1.079) | 0.834*** | (0.602 - 1.066) | 0.856*** | (0.620 - 1.091) |
| Age squared | -0.006*** | (-0.009 - -0.004) | -0.006*** | (-0.008 - -0.004) | -0.006*** | (-0.009 - -0.004) |
| Highest attained education | | | | | | |
| Primart schooling | 0.842*** | (0.540 - 1.144) | 0.850*** | (0.548 - 1.151) | 0.862*** | (0.558 - 1.166) |
| Secondary schooling | 0.701*** | (0.414 - 0.987) | 0.699*** | (0.413 - 0.984) | 0.709*** | (0.421 - 0.997) |
| Tertiary schooling | ref | | ref | | ref | |
| Metropolitan residence | -0.132 | (-0.345 - 0.081) | -0.131 | (-0.343 - 0.082) | -0.128 | (-0.343 - 0.088) |
| Regional unemployemnet rate | 0.036** | (0.004 - 0.068) | 0.036** | (0.004 - 0.068) | 0.036** | (0.004 - 0.068) |
| Region of origin | | | | | | |
| Sweden | ref | | ref | | ref | |
| Nordic countries | 0.282* | (-0.032 - 0.597) | 0.279* | (-0.034 - 0.591) | 0.267* | (-0.050 - 0.584) |
| Europe and Western countries | 0.346*** | (0.090 - 0.601) | 0.319** | (0.064 - 0.574) | 0.288** | (0.028 - 0.548) |
| Rest of the World | 0.025 | (-0.323 - 0.373) | -0.014 | (-0.362 - 0.334) | -0.083 | (-0.442 - 0.275) |
| Years | | | | | | |
| 1992 | -0.649*** | (-1.044 - -0.254) | -0.624*** | (-1.019 - -0.229) | -0.668*** | (-1.065 - -0.271) |
| 1993 | -0.282* | (-0.618 - 0.054) | -0.272 | (-0.607 - 0.064) | -0.292* | (-0.629 - 0.045) |
| 1994 | 0.007 | (-0.299 - 0.312) | 0.015 | (-0.290 - 0.320) | 0.003 | (-0.304 - 0.309) |
| 1995 | ref | | ref | | ref | |
| 1996 | 0.265* | (-0.036 - 0.566) | 0.259* | (-0.042 - 0.559) | 0.264* | (-0.038 - 0.566) |
| 1997 | 0.242 | (-0.076 - 0.561) | 0.230 | (-0.088 - 0.548) | 0.240 | (-0.079 - 0.559) |
| 1998 | 0.352** | (0.011 - 0.694) | 0.336* | (-0.006 - 0.677) | 0.351** | (0.009 - 0.694) |
| 1999 | 0.320* | (-0.036 - 0.677) | 0.301* | (-0.055 - 0.657) | 0.315* | (-0.043 - 0.673) |
| 2000 | 0.381** | (0.005 - 0.756) | 0.356* | (-0.019 - 0.731) | 0.366* | (-0.011 - 0.744) |
| 2001 | 0.264 | (-0.134 - 0.661) | 0.232 | (-0.165 - 0.630) | 0.238 | (-0.162 - 0.638) |
| Constant | -37.79*** | (-44.29 - -31.28) | -37.34*** | (-43.79 - -30.88) | -38.15*** | (-44.69 - -31.62) |
| N of Individuals | 48,676 | | 48,676 | | 48,676 | |
| N of Observations | 285,015 | | 285,015 | | 285,015 | |

*** p<0.01, ** p<0.05, * p<0.1

**Table A4**: Complete estimates, Models 10-12. Beta-coefficients.

| Women | Model 10 Time t-1 | | Model 11 5-year-lag average | | Model 12 10-year-lag average | |
|---|---|---|---|---|---|---|
| | Coef. | 95% CI | Coef. | 95% CI | Coef. | 95% CI |
| Relative income centile | | | | | | |
| 00-20 | -0.0608 | (-0.616 - 0.495) | 0.419 | (-0.198 - 1.037) | -0.0659 | (-0.689 - 0.558) |
| 20-40 | -0.474* | (-1.009 - 0.062) | -0.0124 | (-0.582 - 0.557) | -0.146 | (-0.698 - 0.406) |
| 40-60 | ref | | ref | | ref | |
| 60-80 | -0.159 | (-0.650 - 0.332) | 0.35 | (-0.186 - 0.886) | -0.16 | (-0.696 - 0.376) |
| 80-100 | -0.0241 | (-0.575 - 0.527) | 0.331 | (-0.281 - 0.942) | 0.24 | (-0.337 - 0.816) |
| Hypertension | 8.625*** | (7.851 - 9.399) | 8.619*** | (7.838 - 9.401) | 8.303*** | (7.548 - 9.058) |
| Diabetes | 4.297*** | (2.935 - 5.660) | 4.325*** | (2.943 - 5.706) | 4.223*** | (2.935 - 5.511) |
| Sickness absence history | | | | | | |
| One year before | 0.883*** | (0.478 - 1.288) | 0.709*** | (0.225 - 1.193) | 0.701*** | (0.222 - 1.180) |
| Two years before | | | 0.693* | (-0.003 - 1.388) | 0.679* | (-0.010 - 1.368) |
| Three years before | | | -0.909 | (-2.151 - 0.334) | -0.907 | (-2.135 - 0.320) |
| Four years before | | | -0.314 | (-2.216 - 1.587) | -0.32 | (-2.201 - 1.560) |
| Five years before | | | 0.86 | (-1.086 - 2.806) | 0.812 | (-1.103 - 2.727) |
| Civil status (married ==1) | 0.112 | (-0.304 - 0.528) | 0.111 | (-0.306 - 0.528) | 0.104 | (-0.303 - 0.512) |
| Age | 1.809*** | (1.224 - 2.393) | 1.813*** | (1.226 - 2.399) | 1.702*** | (1.139 - 2.265) |
| Age squared | -0.015*** | (-0.020 - -0.009) | -0.015*** | (-0.020 - -0.009) | -0.014*** | (-0.019 - -0.009) |
| Highest attained education | | | | | | |
| Primart schooling | 0.806** | (0.185 - 1.427) | 0.792** | (0.168 - 1.416) | 0.815*** | (0.208 - 1.423) |
| Secondary schooling | 0.930*** | (0.365 - 1.496) | 0.925*** | (0.357 - 1.493) | 0.947*** | (0.396 - 1.498) |
| Tertiary schooling | ref | | ref | | ref | |
| Metropolitan residence | -0.100 | (-0.548 - 0.349) | -0.109 | (-0.560 - 0.341) | -0.126 | (-0.567 - 0.314) |
| Regional unemployemnet rate | 0.068** | (0.001 - 0.135) | 0.068** | (0.001 - 0.135) | 0.068** | (0.002 - 0.134) |
| Region of origin | | | | | | |
| Sweden | ref | | ref | | ref | |
| Nordic countries | 0.698** | (0.148 - 1.248) | 0.697** | (0.144 - 1.249) | 0.706** | (0.167 - 1.245) |
| Europe and Western countries | 0.083 | (-0.500 - 0.665) | 0.069 | (-0.518 - 0.657) | 0.096 | (-0.478 - 0.670) |
| Rest of the World | 0.281 | (-0.523 - 1.085) | 0.267 | (-0.543 - 1.077) | 0.312 | (-0.480 - 1.103) |
| Years | | | | | | |
| 1992 | -1.244** | (-2.206 - -0.281) | -1.201** | (-2.167 - -0.235) | -1.103** | (-2.043 - -0.164) |
| 1993 | -0.384 | (-1.153 - 0.385) | -0.381 | (-1.155 - 0.394) | -0.337 | (-1.098 - 0.424) |
| 1994 | 0.121 | (-0.551 - 0.793) | 0.139 | (-0.535 - 0.813) | 0.149 | (-0.517 - 0.815) |
| 1995 | ref | | ref | | ref | |
| 1996 | 0.750** | (0.107 - 1.394) | 0.722** | (0.076 - 1.368) | 0.721** | (0.082 - 1.359) |
| 1997 | 1.044*** | (0.374 - 1.714) | 1.059*** | (0.388 - 1.730) | 1.035*** | (0.373 - 1.697) |
| 1998 | 0.716* | (-0.0531 - 1.485) | 0.705* | (-0.066 - 1.477) | 0.686* | (-0.073 - 1.446) |
| 1999 | 1.264*** | (0.505 - 2.023) | 1.265*** | (0.504 - 2.026) | 1.235*** | (0.487 - 1.982) |
| 2000 | 1.277*** | (0.467 - 2.087) | 1.275*** | (0.463 - 2.087) | 1.232*** | (0.434 - 2.029) |
| 2001 | 1.247*** | (0.392 - 2.101) | 1.225*** | (0.368 - 2.083) | 1.183*** | (0.340 - 2.026) |
| Constant | -74.59*** | (-90.67 - -58.52) | -75.12*** | (-91.25 - -58.98) | -71.11*** | (-86.51 - -55.71) |
| N of Individuals | 43,650 | | 43,650 | | 43,650 | |
| N of Observations | 253,175 | | 253,175 | | 253,175 | |

*** p<0.01, ** p<0.05, * p<0.1

Chapter 4

# Labor migration and Ischemic Heart Disease in Sweden

**Authors**: Tina Hannemann, Kirk Scott

## 4.1. Abstract

Health differences between natives and individuals of foreign origin are an important aspect in the fields of epidemiology and international migration, and previous research has identified a number of potential explanations for these differences. This study investigates the impact of two of those explanations in a sample of natives and immigrants in Sweden. Health difference is measured as risk for the onset of IHD during the years of 1992 until 2001 using a sample of about 40,000 individuals who were economically active in 1970. The sample selection focuses on a group of immigrants who arrived in Sweden between 1955 and 1970 which increases the level of homogeneity among the immigrants for various characteristics. In a second step immigrants and natives are compared, stratified by their occupational group, to account for the different distribution in the labor market for natives and immigrants. The results show that no health differences could be found for this homogeneous sample of immigrants except in the case of Finnish immigrants. On the other hand, the health disadvantage of Finnish immigrants remained even in the occupational stratified models. Concluding, this study has shown that large parts of the health differences between natives and migrants in Sweden is due to characteristics of migration, other than the country of origin. As outstanding exception, the group of Finnish immigrants showed very robust health disadvantages compared with Swedes, even after accounting for the different labor market distribution.

## 4.2. Introduction

Given the increasing trend of international migration all around the world, public health studies are placing increasing focus on differences in health outcomes between immigrants and natives, with many epidemiological studies showing that immigrants experience different health outcomes compared with the native population (see for example Dassanayake et al 2011; Gadd et al 2003; Singh and Siahpush 2002). These studies have examined a wide range of possible impact factors which could explain these differences, with one prime explanation being a change in health regime which immigrants experience while moving to a new destination. Considering that the health systems in both locations can vary substantially, different patterns for mortality and morbidity for newly-arrived immigrants and natives are a reasonable assumption. However, health is not a static condition and its development, at the national level as well as the individual level, depends on many factors.

Often health differences tend to be larger when cultural and behavioral characteristics of the home country and host country show considerable differences. The distance between the home country and country of destination can be of cultural, climatic or geographic nature. Immigrants from neighboring countries might show similar health patterns and behavior as natives, due to long-term economic and cultural exchange between the countries. On the other hand, immigrants from more remote origins, whose home country has only weak connection with the country of destination, can show substantial health differences after arrival (Wiking et al 2004).

Observed health disadvantages between immigrants and natives can also originate from structural problems, such as lack of information or access to health services, which do not apply for the majority of the native population. Inability to communicate in the local language is an obvious source of problems faced by immigrants, which could lead to a lower use of health care services (Fassaert, Hesselink and Verhoeff 2009).

Another source of health differences may be found in the underlying purpose of migration. The health status of an immigrant escaping from war and persecution can vary significantly from the health status of a labor migrant or family reunification immigrant. While some migration processes are planned and prepared in advance, others have to be carried out under substantial risks for health and safety and on short notice. Further, demographic structures such as age distribution and sex-ratio will vary between waves of refuges, labor migrants and family reunion migrants. Compared with a general population of natives, these structural differences can lead to health differences observed between immigrants

and natives, since age and sex are important impact factors of health (Ferrari et al 2012).

The differences between migrant types do not end with arrival in the host country. Employment and economic independence are keys conditions for successful integration, and the different groups of immigrants may follow different patterns in the labor market. Even if only economically active immigrants are investigated, one can observe a different distribution in the labor market and within the occupational hierarchy both within the immigrant population itself and compared with natives. Position in the labor market and economic success has been shown to influence health outcomes (Cabrera et al 2001; Rosvall et al 2006). Assuming that immigrants are disproportionately represented in lower occupational groups, the health disadvantage originating from this socioeconomic status could inflate the observed health problems among migrants and lead to an overestimation of health problems attributable to immigrants.

The sum of these factors makes the group of immigrants a very heterogeneous mix. To address the country of origin as an overall health impact factor without taking other characteristics into account might lead to a biased interpretation. Thus, any study of immigrant health must take both individual and group level characteristics into account. Given that immigrants vary in so many aspects, it stands to reason that some of the observed health differences between immigrants and natives might be overestimated, due to the distribution of characteristics such as length of stay and purpose of migration.

This study investigates the health differences between natives and immigrants in Sweden, measured as risk for Ischemic Heart Disease (IHD). IHD belongs to the group of cardiovascular diseases, which is the main cause of death in most developed countries and is increasing rapidly in developing countries all over the world. The overall hypothesis is that a portion of the observed health difference between immigrants and natives is due to heterogeneity among immigrants in terms of the factors discussed above, rather than the specific country of origin itself. In order to test this hypothesis, this study analyzes a selected group of migrants who had similar migration motivation and had already spent a substantial amount of time in Sweden at the beginning of the study period.

The secondary hypothesis is that an additional portion of remaining health differences might be driven by labor market inequalities between natives and immigrants. Therefore, this study investigates a sample of immigrants and natives while considering the different labor market distribution through comparing immigrants and natives from the same occupational groups.

## 4.3. Theoretical framework

People migrate from one country to another for a wide variety of reasons. Some of the most common motivations are work, refuge, education, and family reunification. Immigrants from different groups vary in more than one respect. While labor migrant and family reunion migrants make the decision to migrate on a rather voluntary basis, many refugees are forced to leave their country, often without time to adequately plan the move. While this latter group often does not know when or if they can ever return to their home country, a return to the home country is generally possible for other groups of immigrants.

Another source of differentiation is found in the degree of destination-specific human capital which is achieved in preparation for the process of migration. Labor migrants and, to a substantial degree family reunion immigrants, have the possibility to plan and prepare their stay in the new destination prior to the migration itself. This can include achieving skills and knowledge concerning language, customs and administrative steps required both for the migration process and subsequent integration. The majority of refugees and asylum seekers do not have the time or opportunity to make extensive preparations before arriving in the country of destination. The resulting lack of destination-specific knowledge can lead, for example, to lower usage of health services which may be available to them (Blochliger et al 1998), and therefore influence health indirectly.

Individuals from the same origin tend to share a set of country-specific characteristics, whose usefulness in the destination varies from country to country. An individual's health-promoting and health-damaging behavior is correlated to the general behavior of its origin society and therefore linked to the specific country of origin. The distribution and prevalence of behaviors such as smoking, physical exercise, use of medical services and dietary preferences may all be determined by experiences in the country of origin (Dotevall et al 2000; Tomson and Åberg 1994). Due to tradition, habit, or continued connection to the home country, these health behaviors may be retained long after the migration process (Pudaric et al 2000). National identity and political and religious affiliations are further factors to take into account, when analyzing different countries of origin (Hjelm et al 2003). Therefore, analyzing immigrants from many different origins as a common group has the potential to introduce bias through the conflating of country-specific factors.

Due to the circumstances of arrival in the host country, demographic structures such as age distribution and sex-ratio will vary between the groups of immigrants. Immigrants who arrive in the country of destination for the sake of labor show

a different demographic structure due to a self-selection process occurring in the sending country.

While above factors are discussed that may lead to worse health among immigrants, there are a number of studies which find that immigrants may actually have a health premium, rather than a health disadvantage. This phenomenon of better health outcomes among migrants is often referred to as *Healthy Migrant Effect* (Singh and Siahpush 2001; Wingate and Alexander 2006). Mainly the term refers to the selection of advantageous age distribution and health conditions among immigrants at time before arrival which then result in better health status in the host country. In order to actually migrate, a minimum of physical and mental health is required as well as some economic assets to cover the cost of migration. Therefore, the share of individuals migrating in good health is likely to be larger than the share in good health in the home country.

The literature also describes that the health benefit from the healthy migrant effect fades with time (Singh and Siahpush 2001). The long-term exposure to host country health conditions can therefore have a detrimental effect for immigrant groups who initially showed better health. For immigrants who arrive in worse health condition, this long-term adaption could also serve to decrease their overall health situation. The direction of change of health behavior and health patterns depends on the general health in the country of origin, the host country and degree of adaptation (Nakanishi et al 2004).

Working to mitigate this negative health trend, country-specific skills such as language and knowledge about the healthcare system and services can, with time, increase integration and reduce health differences. Therefore, immigrants who have been in the host country for a significant amount of time are assumed to show health patterns more similar to natives, compared with recently arrived immigrants (Albin et al 2005; Lebrun 2012; Wiking et al 2004).

The different demographic structure among immigrants of the various types is a potential bias in any health analysis, either comparing immigrants with each other or with the native population. The gender balance among immigrants differs dramatically depending on the reason for migration, with labor migrants being predominantly male in most contexts. Given the high share of males among labor migrants, family reunion migration is driven mainly by women, children and to a lesser extent parents and other close relatives.

While health outcomes depend to a certain degree on the health situation of immigrants before migration, integration also plays an important role, as mentioned above. The probability for successful integration depends strongly on the migration and integration policies of the host country, as well as the willingness of immigrants and natives to co-exist. A cornerstone of successful integration is the economic independence of the individual, but the opportunities

for obtaining employment vary among the different types of immigrants and are strongly connected to the economic situation in the host country. Even during periods of active labor recruitment, labor migrants do not distribute evenly across the labor market and occupational groups.

Occupational group affiliation, and therefore the socioeconomic status of the individual, has an impact on health. Literature shows many examples of how health disadvantages are more prominent in lower social classes (Diez-Roux et al 1995; Marmot 1989; Pocock et al 1987; Smith, Bartley and Blane 1990a). Disadvantageous working conditions such as shift work, irregular working hours and overtime are stressful for body and mind (Alfredsson et al 1982b) and they are also more common among the lower occupational groups. The affiliation with different occupational groups also depends on individual characteristics such as age, sex and educational level. Given the aforementioned varying demographic patterns among the types of immigrants, an additional factor of different labor market distributions between natives and immigrants becomes apparent.

Lower occupational group affiliation and possible educational and occupational mismatch can produce psychosocially stressful situations. Physical health outcomes originating from psychosocial stress have been the focus of more recent studies in epidemiology (Wang et al 2007; Williams et al 2009). Premature aging and signs of wear are potential consequences. Often, psychological stress has an indirect effect on health, via the endocrinological system (Brunner et al 1997) and metabolic syndrome (Björntorp 1991; Chandola et al 2006). Long-term stress from adverse working conditions has been found to increase the risk for IHD (Bosma et al 1998; Chandola et al 2008; Kuper and Marmot 2003). The effect of work-related stress can be direct when increased demands exhaust the individual reserves and the body suffers from a reaction to constant stress (Gémes et al 2008). Psychosocial stress can be correlated indirectly with IHD risk via changes in health behavior such as lack of physical activity due to mainly sedentary work tasks. Possible time constraints due to shift work and over time have detrimental effects on health and are more common among the lower occupational groups. The prevalence of worse health behaviors among the lower occupational groups reflects higher vulnerability to the onset of IHD for the affected individuals (Chandola et al 2006).

Taking into account that natives and immigrants exhibit different distributions across occupational groups and that affiliation with specific occupational groups can influence health, the hypothesis arises that the health differences between immigrants and natives might be partially due to the occupational distribution. Inasmuch as immigrants are overrepresented in the lower occupational classes, observed health differences could be erroneously attributed to country of origin, and rather be at least partially due to the socioeconomic distribution.

## Sweden's Migration History

Following a century of being a large-scale sender of migrants, Sweden became a net immigration country during the Second World War. The Swedish post-war migration history can be divided into two very different periods in terms of both migration policies and the characteristics of the arriving migrants. During the two decades following the Second World War, the Swedish economy was expanding at a rate which could not be satisfied by the domestic labor supply. During this period, Swedish companies actively recruited migrants from abroad to allow the economy to grow unfettered by labor shortages (Gadd et al 2003). The first streams of work-seeking immigrants came in the 1950s from Western and Southern Europe, followed in the 1960s by large-scale migration from Greece, former Yugoslavia and Turkey (Bengtsson et al 2005).

The immigration streams increased rapidly following the Second World War, and the shortage of labor was kept at bay. During the late 1960s, housing shortages, coupled with worries that migration reduced wage growth, led to discussions of the sustainability of the labor migration regime. Under pressure of the strong labor unions, a change in migration policy was introduced in 1968 and came to action gradually during the late 1960s and early 1970s. Stricter policies essentially ended the "tourist migration" of the 1960s, requiring potential migrants to be in possession of both housing and employment prior to entry into the country (Bengtsson et al 2005). Consequently, non-Nordic migration came to a rather abrupt stop, while Nordic migration continued under the auspices of the free Nordic labor market. From the 1970s onwards, new streams of immigrants came primarily as family reunification migrants and refugees. In addition to new motivations for immigration, the individuals moving to Sweden after 1970 also came from more distant origins such as Latin and South America, the Middle East and Africa.

The aim of the study is to quantify the effect of heterogeneity among immigrants and of labor market distribution on differences in health outcome. To this end this study exploits the fact that migrants arriving before 1970 are more homogenous in terms of migration reasons than migrants after 1970, allowing us to remove some uncertainty caused by heterogeneity. Onset of IHD has been chosen as the health measurement of interest in this study, and the correlation between SES, here measured as occupational group, and mortality and IHD risk is well documented (Mackenbach et al 1997; Marmot 1989; Marmot and Bartley 2002). Using IHD as health outcome makes it essential to control for several medical and demographic characteristics to avoid bias. Age, marital status, prevalence of IHD risk factors such as hypertension, diabetes and atherosclerosis

as well as education and income are included step-wise in the model to take their direct effect on IHD into account.

## 4.4. Data and method

To test the hypothesis, this study uses data from the Swedish Longitudinal Immigrant database (SLI). The SLI contains information from the Swedish Tax Register (inkomst och förmögenhetsregister), the censuses of 1970 and 1985, the Total Population Register (RTB), and information from the Hospital Discharge Register (patientregistret). The Swedish personal identification number was used to link the different sources, and the final database contains a random sample of both native-born Swedes and a representative share of immigrants from various origins. The detailed data from the various sources is aggregated to one observation per year and per person.

The dependent variable, IHD, is measured as a dichotomous variable indicating if a person had an IHD event in a given year. This information is obtained from the Hospital Discharge Register and is classified according to ICD9 and ICD10 codes. Hospital Discharge Register entries capture every medical incidence which results in the admission of the patient to the hospital for inpatient care. Given the severity and the extensive need for medical intervention associated with IHD, this study assumes that there is no significant under-presentation of cases. Even in the event that a person experienced a fatal IHD case outside the hospital, the event and cause of death would be registered in the nearest hospital. Therefore, the data collection, regarding IHD cases can be assumed to be rather complete.

However, the precise measurement of the onset of IHD bears some obstacles using the information from the Hospital Discharge Register, which was introduced nationwide in 1987. No information about medical events before that date is available for this study. As a consequence, observed IHD cases do not necessarily have to represent the first of its kind for the individual in question. In order to account for this problem, this study utilizes a property of IHD to minimize the probability for not observing the first event.

IHD has a degenerative character, producing a loss of function of the heart itself as well as the supporting blood vessel system. Consequently, individuals who have experienced an initial IHD event are at much greater risk of experiencing another IHD event, compared with individuals without IHD history. In the database over 50 percent of the individuals who experience an IHD event suffer from an additional IHD event during the observation period. Thus, not taking

the former IHD history of the individual into account could greatly overestimate the risk for IHD for individuals who already experienced an event.

Pre-analysis of the SLI database has shown that the mean time-span between two IHD events, experienced by the same person, is 1.03 years. In total, 95 percent of all repeated IHD events appear less than five years after the first observed case. Given this information, the sample is restricted to include only individuals who have been observed for five consecutive years without experiencing an IHD event, and thereby assume with a reasonably high degree of certainty that the observed IHD is the first. While there are undoubtedly some cases which are in actuality subsequent IHD events, this study suggest that the separation of five years minimizes the relation to an earlier event, and thereby minimizes any potential bias. The focus on the onset of IHD in this study implies that individuals are censored from the sample after experiencing an IHD event during the observation period.

This study compares the risk for the onset of IHD among the native population and immigrants groups in Sweden. As laid out before, in order to test the hypotheses, it appears essential to distinguish between several characteristics among the group of immigrant to take the heterogeneity appropriately into account. This study therefore, focuses exclusively on labor migrants who migrated to Sweden between 1955 until 1970, or in other words during the active labor recruitment period. This migration wave provided previously unseen numbers of immigrants arriving in Sweden in a rather short period from a few specific countries.

The observation window of 1992-2001 means that all included immigrants must have spent at least 22 years in Sweden, which eliminates the risk that health issues directly related to the migration process or short-term stay in the host country are included. Given the high labor demand in Sweden, the majority of this selected group of immigrants was successfully integrated into the labor market before, or shortly after, arrival (Scott 1999). Economic independence facilitates social integration and information regarding access to health services, so this characteristic is of great importance.

While the SLI contains information on just over 500,000 individuals, the restrictions this study imposes on the sample greatly reduce this number. Given the observation period of 1992 to 2001, natives and immigrants have to be registered during that time in Sweden in order to appear in the data, the sample is reduced to 282,130 individuals. Further, immigrants and natives had to be recorded as economically active in the 1970 census to be included in the models, since the occupation in 1970 is used as a proxy for socioeconomic status. The focus on immigrants who arrived between 1955 and 1970, who have been in registered employment in 1970 reduced the sample to 42,487 individuals, of

which 25,618 are men and 16,869 are women. During the observation period, 2,281 men and 797 women suffer of a first IHD event.

The focus on labor migrants is only one step to isolate the effect of country of origin on health. As explained above, the uneven distribution of immigrants and natives in the labor market makes both groups difficult to compare with each other. A higher share of immigrants in low occupational groups might overestimate the health disadvantage of this group due to the negative effects of the occupational group affiliation. In order to take the detrimental health effects of lower occupational class into account, occupational class is introduced in the models. As consequence, immigrants in specific occupation groups are compared with natives in exactly the same occupations. If labor market distribution is the driving force behind health differences, the estimates would show strongly reduced differences in IHD risk between natives and immigrants once occupational group is controlled for. This study also performs occupation-stratified models to verify if health differences between natives and immigrants are varying among the occupational groups.

Foreign-born individuals in this sample come from a very small number of countries, due to the nature of active labor recruitment. This study analyzes the health differences between immigrants from six countries (Norway, Denmark, Finland, Italy, former Yugoslavia and Germany). The group of German immigrants contains individuals from both sections of the formerly divided country, and former Yugoslavia includes all former states of Yugoslavia. Further, two collective groups of countries (East and West European) have been included. They include countries which present too small numbers of individuals for separate analysis, but share at least region of origin.

Individuals who do not belong to one of the aforementioned countries are not considered for this study, since their numbers are too small for an appropriate analysis. This focus on a few selected countries enables separate analyses to take shared health baseline and health behaviors into account. Throughout all analysis steps, the group of Swedish individuals is used as the reference category for the risk for the onset of IHD. The number of individuals, their share among the sample and the incidence of IHD cases during the observation period for each country are displayed in Table 1.

**Table 1**: Sample stratified by country of origin and sex, including number of unique individuals and number of first IHD cases in each group

| | Men | | | Women | | |
|---|---|---|---|---|---|---|
| | number of individuals | % of total | IHD incidence | number of individuals | % of total | IHD incidence |
| Sweden | 17615 | 68.76 | 8.3 | 11658 | 69.11 | 4.0 |
| Norway | 674 | 2.63 | 10.2 | 437 | 2.59 | 8.0 |
| Danmark | 874 | 3.41 | 13.0 | 442 | 2.62 | 5.4 |
| Finland | 1978 | 7.72 | 10.0 | 1699 | 10.07 | 6.9 |
| Italy | 527 | 2.06 | 10.4 | 192 | 1.14 | 7.8 |
| F. Yugoslavia | 1592 | 6.21 | 9.4 | 1028 | 6.09 | 5.3 |
| Germany | 809 | 3.16 | 7.5 | 427 | 2.53 | 5.2 |
| West | 297 | 1.16 | 7.7 | 139 | 0.82 | 2.2 |
| East | 1252 | 4.89 | 11.7 | 847 | 5.02 | 6.7 |
| Total | 25618 | 100% | | 16869 | 100% | |

Data were taken from SLI database, calculations are the authors

The second aim of the study is to quantify the effect of uneven distribution of immigrants within the Swedish labor market. Therefore occupational class is taken into consideration. The groups of occupation were generated from the Swedish socioeconomic index (SEI) reported in the census of 1970. The study uses the early socioeconomic position as proxy for later life socioeconomic status. The occupation was aggregated into three dummy variables, distinguishing the affiliation of every individual to either the upper white-collar, white-collar or blue-collar occupational group. In the first models these three groups are included, with blue-collar workers as reference. Later analysis is performed on occupation-stratified samples. In the stratified models, an additional covariate is introduced, indicating if the individual changed occupational group at some point from the 1970 census until the census of 1985. The variable of occupational mobility is used as categorical variable taking the values of either upward, downward mobility, unchanged occupational group or no registered occupation in 1985.

In additional to occupational group, the model controls for the branch of employment. Branch is coded categorically as manufacturing, retail and transport, private sector and public sector, taken from the information of the census of 1970. The distribution of occupations varies among the branches and therefore a different health impact from various branches is possible.

The share of immigrants working in the same branch as the individual is also included. This information was derived from a more detailed four digit code of branches in the 1970 census and used to indicate branches dominated by immigrants. A branch which is dominated by foreign labor, likely because it is

avoided by the majority of the native population, may indicate poor working conditions which may also lead to higher IHD risk.

To verify the impact of potential IHD risk factors, several covariates are included step-wise into the models. As a degenerative disease, it is necessary to control for age, since higher age is a direct risk factor for IHD. The square of age is included as well to control for possible non-linear effects of age on the risk for IHD. Living in a stable partnership can have stress-buffering effects and provide a source of economic, social and emotional support and can therefore act as preventive influence for heart disease (Johnson et al 2000; Molloy et al 2009). Marital status, used as categorical variables (never-married, married, divorced and widowed), is included as time varying variable. A dichotomous variable is added indicating if the individual has one or more underage children in the household. This variable is derived from the tax information on child subsidy and indicates an additional source of stress in the individual's environment, which could in turn influence the risk for IHD.

The register-based data does not contain information on health behavior. Consequently, typical IHD risk factors such as smoking, low physical activity and dietary habits cannot be taken into account. Often these health behaviors have other consequences such as diabetes, atherosclerosis and hypertension. To the extent that they are diagnosed, the prevalence of these diseases is captured in the Hospital Discharge Register. All three diseases are included in some models as independent risk factors as well as a proxy for the non-observed health behaviors.

The models also contain information on education, taking the values of primary, secondary or university degree. Education is strongly connected to occupational group, which is an established risk factor for IHD. Higher education is generally correlated with better overall health and lower risk for IHD (Falkstedt and Hemmingsson 2011; Smith et al 1998). Further, another variable was introduced to distinguish socioeconomic conditions. The full model contains the individual income before taxes, measured as an inflation-adjusted logged average over the 5 years prior to the observed year. This provides a proper indication of the financial security from a medium-term perspective (Hannemann and Helgertz work in progress). Potential bias, due to income fluctuations unobserved in the point measurements of income, is therefore reduced.

In addition to the abovementioned determinants, IHD shows a different pattern for men and women. Women experience all cardiovascular disease, including IHD, later in life than men, due to differences in their endocrine system (Kajantie and Phillips 2006) and health behavior (Nikiforov and Mamaev 1998). Consequently, in certain age classes the risk for IHD for women is much lower than the risk for men. Further, there are sex differences in the labor market for men and women (Arber and Ginn 1995). While the contemporary Swedish

labor market is assumed to be one of the most equal in terms of sex-differences, this was not the case 40 years ago. Typical female and male occupations were separate then, with men being dominant in the manual and leading positions, and women were disproportionally present in lower white-collar occupations and in the service sector. In order to account for the various sex-specific effects, separate models for men and women are performed.

The observation period from 1992 until 2001 has been chosen due to the specifics of the dependent variable IHD, as explained earlier. The period is of historic and economic interest since the economic crisis of the early 1990s in Sweden falls into this time period. To capture the exogenous year-specific characteristics, all models contain year dummies. All covariates and their descriptive statistics stratified by sex and origin of the individual are displayed in Table 2 below.

This study analyzes IHD risk with help of random effects logistic regression models to capture effects specific to the individuals which are unobserved in the database. The results of the Hausman-test show that the estimates from fixed and random effects models are not systematically different from each other, and therefore the application of random effect models is seen as appropriate for this study.

**Table 2**: Covariates used for the analysis, grouped for step-wise introduction in the models

| Covariates | | Men | | Women | | |
|---|---|---|---|---|---|---|
| | | Native | Foreign | Native | Foreign | |
| IHD cases in % | | 8.3 | 10.2 | 4.0 | 6.3 | Basic covariates |
| Age (mean) | | 56.4 | 58.8 | 55.4 | 57.9 | |
| Age, squared (mean) | | 3290.5 | 3548.2 | 3181.1 | 3455.7 | |
| Marital status (%) | unmarried | 19.4 | 12.3 | 18.0 | 10.4 | Socio-medical |
| | married | 51.3 | 60.6 | 43.3 | 49.8 | |
| | divorced | 26.2 | 23.9 | 30.1 | 28.1 | |
| | widowed | 3.2 | 3.3 | 8.6 | 11.7 | |
| Child subsidy (%) | | 20.9 | 13.4 | 21.3 | 12.0 | |
| Hypertension (%) | | 3.0 | 3.6 | 2.3 | 3.5 | |
| Diabetes (%) | | 2.4 | 2.6 | 1.5 | 1.9 | |
| Atherosclerosis (%) | | 0.5 | 0.7 | 0.3 | 0.4 | |
| Education (%) | | | | | | Education and Income |
| | primary | 42.5 | 38.9 | 33.8 | 45.6 | |
| | secundary | 27.3 | 27.1 | 36.7 | 30.1 | |
| | university | 30.4 | 34.0 | 29.5 | 24.3 | |
| Income (mean over five prior years) | | 11.6 | 11.3 | 11.3 | 11.2 | |
| Immigrant share in occupation (%) | | 8.7 | 13.7 | 8.4 | 13.1 | Occupation |
| Occupation (%) | Blue collar | | | | | |
| | White collar | 34.1 | 19.0 | 56.1 | 24.2 | |
| | Upper white collar | 7.2 | 6.3 | 2.8 | 2.9 | |
| Branch (%) | Manufacturing | 55.2 | 71.5 | 21.0 | 48.3 | |
| | Retail / Transport | 24.6 | 14.1 | 25.9 | 17.9 | |
| | Private | 10.4 | 7.2 | 15.2 | 8.6 | |
| | Public | 9.8 | 7.2 | 37.9 | 25.1 | |
| Occupational mobility 1970-1985 (%) | | | | | | Stratified models |
| | upwards | 19.4 | 12.2 | 27.0 | 22.2 | |
| | stable | 58.3 | 59.7 | 46.5 | 47.0 | |
| | downwards | 8.8 | 4.7 | 7.4 | 4.1 | |
| | no information | 13.5 | 23.4 | 19.1 | 26.7 | |
| Period (%) | 1992 | 10.7 | 11.0 | 10.4 | 10.6 | Basic covariates |
| | 1993 | 10.6 | 10.8 | 10.3 | 10.5 | |
| | 1994 | 10.4 | 10.6 | 10.2 | 10.4 | |
| | 1995 | 10.2 | 10.4 | 10.2 | 10.2 | |
| | 1996 | 10.1 | 10.1 | 10.1 | 10.1 | |
| | 1997 | 9.9 | 9.9 | 10.0 | 9.9 | |
| | 1998 | 9.8 | 9.6 | 9.9 | 9.8 | |
| | 1999 | 9.6 | 9.4 | 9.8 | 9.7 | |
| | 2000 | 9.4 | 9.1 | 9.7 | 9.6 | |
| | 2001 | 9.3 | 8.9 | 9.6 | 9.4 | |

Data were taken from SLI database, calculations are the authors

# 4.5. Results

The hypothesis of the study is that a large share of the health differences between natives and immigrants is primarily due to factors other than country of origin. On the one hand, the heterogeneity of length of stay and purpose of immigration adds noise to the estimates. On the other hand, the uneven distribution of immigrants in the labor market could result in biased effects of country of origin. In order to test this hypothesis, the first analysis is done on a non-restricted sample of immigrants in comparison to Swedes.

**Table 3**: Logistic regression of the risk for the onset of IHD using an unrestricted sample of immigrants in Sweden from the SLI database, stratified by sex

| | Men | | Women | |
|---|---|---|---|---|
| **Country of origin** | **Model A1 OR (95% CI)** | **Model A2 OR (95% CI)** | **Model A1 OR (95% CI)** | **Model A2 OR (95% CI)** |
| Norway | 1.225*** | 1.225*** | 1.258** | 1.217** |
| | (1.068 - 1.406) | (1.065 - 1.408) | (1.054 - 1.501) | (1.017 - 1.457) |
| Danmark | 1.033 | 0.984 | 0.956 | 0.946 |
| | (0.900 - 1.185) | (0.855 - 1.131) | (0.778 - 1.175) | (0.768 - 1.165) |
| Finland | 1.516*** | 1.373*** | 1.672*** | 1.442*** |
| | (1.365 - 1.684) | (1.233 - 1.529) | (1.475 - 1.896) | (1.267 - 1.640) |
| Italy | 1.241** | 1.198* | 1.077 | 0.908 |
| | (1.024 - 1.505) | (0.986 - 1.455) | (0.752 - 1.544) | (0.629 - 1.310) |
| Former Yugoslavia | 1.097 | 1.060 | 1.250*** | 1.080 |
| | (0.972 - 1.239) | (0.936 - 1.201) | (1.071 - 1.459) | (0.918 - 1.270) |
| Germany | 0.907 | 0.971 | 0.899 | 0.967 |
| | (0.762 - 1.078) | (0.815 - 1.157) | (0.712 - 1.136) | (0.763 - 1.224) |
| West | 0.547*** | 0.592*** | 0.663** | 0.751 |
| | (0.412 - 0.727) | (0.444 - 0.789) | (0.448 - 0.980) | (0.506 - 1.114) |
| East | 1.136** | 1.152** | 1.323*** | 1.366*** |
| | (1.019 - 1.268) | (1.030 - 1.289) | (1.167 - 1.498) | (1.200 - 1.554) |
| Other | 1.139*** | 1.076* | 1.140** | 0.986 |
| | (1.055 - 1.231) | (0.988 - 1.171) | (1.022 - 1.272) | (0.870 - 1.117) |
| Age age2 and year dummies | yes | yes | yes | yes |
| Social-medical covariates | no | yes | no | yes |
| Educational and economical | no | yes | no | yes |
| Occupational covariates | no | no | no | no |
| Observations | 1,486,761 | 1,486,761 | 1,516,962 | 1,516,962 |
| Number of indiv | 182,064 | 182,064 | 183,183 | 183,183 |
| IHD cases | 4,785 | 4,785 | 2,702 | 2,702 |
| Log Likelihood | -26,908 | -25,824 | -16,095 | -15,307 |
| Degrees of freedom | 20 | 30 | 20 | 30 |
| Wald chi2 | 4,790 | 8,470 | 3,281 | 6,074 |

*** p<0.01; ** p<0.05; * p<0.1

All the national groups which have been defined before are used for this analysis with an additional group including all other nationalities appearing in the SLI database. No restriction on immigration period or purpose of migration has been done for these models.

Model A1 only includes the country of origin, age variables and year dummies, while Model A2 includes more sets of covariates. Both models, stratified by sex, are displayed in Table 3 and represent the raw country of origin difference in IHD risk, compared with the native population in Sweden.

As the estimates from the logistic regression demonstrate in Table 3, there are quite strong health differences between the various immigrant countries and the Swedish comparison group. In the empty Model A1, men from all countries except Denmark, former Yugoslavia and Germany show elevated risk for IHD. Men from Western countries show significantly lower IHD risk. The results for women are of a similar magnitude. Different than for men, Italian women do not show significant differences, but women from former Yugoslavia show an elevated IHD risk. The effects are somewhat weaker in Model A2 where the various covariates are included. The results of Table 3 are taken as a starting point for all further analysis which will be performed on the restricted immigrant sample.

Model 1 (Table 4 for men and Table 5 for women) is the empty model containing only the dummies for country of origin. Men and women of several immigrant countries show significantly higher IHD risk than natives. That large parts of those health differences are due to variations in age distribution and observation period is shown in Model 2, when the age variables and year dummies are added. For men only Finnish and Danish immigrants retain significant health disadvantages.

Model 3 includes the variables of socio-medical interest and Model 4 also controls for educational level and income. Model 5 displays the estimates for the full model, including the variables for occupational group and branch. For men, only the Finnish immigrants show a significantly different IHD risk than the native group. The estimates of most of the other countries show estimates close to the value of 1. The odds ratios for Western and German male immigrants show a lower risk for IHD, but these estimates are not significant. The two groups are the smallest among the immigrant share and therefore it is possible that the shown effect is real, but the small numbers of individuals and IHD cases do not allow statistical confirmation.

Unlike other studies, the strict sample selection in this study illuminates a general lack of health differences for most of the male immigrant groups. No significant risk for the onset of IHD can be observed except for the case of male immigrants from Finland. The Finnish immigrants have a special role in

Sweden's migration history. Due to the close geographic, economic, and historical relationship, Finnish people represent the highest share of immigrants in Sweden. As their counterparts in Norway and Denmark, they could move freely inside the Nordic Labor Market. Due to the geographical proximity, Sweden is one of the most a favorite destination choice among Finnish emigrants. In country comparisons, individuals living in Finland show worse health patterns than their Nordic neighbors (Hedlund et al 2007). Immigrants from Finland also exhibit worse health than Swedes after immigration to Sweden. In comparison with the Finns who stayed in Finland, the Finnish immigrants in Sweden show better health pattern, however. Once the Finnish migrants arrive in Sweden, they show one of the highest risks for cardiovascular diseases of all immigrants in Sweden (Gadd et al 2003; Hedlund et al 2008), which is in line with the findings of this study. With length of stay this gap is converging but not closing completely (Alfredsson et al 1982a).

This phenomenon can be confirmed by the findings of this study. Given the sample selection for this study individuals, who are observed between 1992 and 2001 and had entered Sweden by 1970, all individuals have stayed in Sweden for at least 22 years. Nevertheless, Finnish immigrants still show significantly elevated IHD risk compared with the native population. The consistency of the IHD risk difference between Swedish and Finnish men using such a homogeneous sample, which eliminated health differences for all other countries of origin, is remarkable.

**Table 4**: Risk for first IHD in different immigrant groups, compared with native Swedes, men

| Men | Model 1 OR (95% CI) | Model 2 OR (95% CI) | Model 3 OR (95% CI) | Model 4 OR (95% CI) | Model 5 OR (95% CI) |
|---|---|---|---|---|---|
| Norway | 1.319** | 1.001 | 1.012 | 1.055 | 1.071 |
| | (1.034 - 1.681) | (0.784 - 1.278) | (0.792 - 1.294) | (0.824 - 1.350) | (0.836 - 1.372) |
| Danmark | 1.698*** | 1.232** | 1.154 | 1.141 | 1.119 |
| | (1.402 - 2.058) | (1.015 - 1.494) | (0.948 - 1.404) | (0.937 - 1.389) | (0.917 - 1.364) |
| Finland | 1.291*** | 1.371*** | 1.292*** | 1.229*** | 1.227** |
| | (1.112 - 1.499) | (1.179 - 1.593) | (1.110 - 1.504) | (1.055 - 1.433) | (1.048 - 1.438) |
| Italy | 1.313** | 1.184 | 1.203 | 1.179 | 1.183 |
| | (1.002 - 1.722) | (0.902 - 1.554) | (0.915 - 1.580) | (0.897 - 1.550) | (0.897 - 1.561) |
| F. Yugoslavia | 1.164* | 1.093 | 1.089 | 1.054 | 1.049 |
| | (0.983 - 1.378) | (0.922 - 1.296) | (0.916 - 1.294) | (0.886 - 1.253) | (0.873 - 1.261) |
| Germany | 0.949 | 0.833 | 0.810 | 0.855 | 0.854 |
| | (0.734 - 1.227) | (0.644 - 1.078) | (0.625 - 1.049) | (0.658 - 1.111) | (0.657 - 1.111) |
| West | 0.962 | 0.730 | 0.734 | 0.788 | 0.793 |
| | (0.636 - 1.455) | (0.482 - 1.105) | (0.484 - 1.113) | (0.519 - 1.197) | (0.521 - 1.205) |
| East | 1.474*** | 1.123 | 1.072 | 1.131 | 1.125 |
| | (1.243 - 1.748) | (0.945 - 1.333) | (0.900 - 1.276) | (0.948 - 1.349) | (0.939 - 1.348) |
| Age, age2 and year dummies | no | yes | yes | yes | yes |
| Social-medical covariates | no | no | yes | yes | yes |
| Educational and economical | no | no | no | yes | yes |
| Occupational covariates | no | no | no | no | yes |
| Observations | 234,176 | 234,176 | 234,176 | 234,176 | 234,176 |
| Number of indiv | 25,618 | 25,618 | 25,618 | 25,618 | 25,618 |
| IHD cases | 2,281 | 2,281 | 2,281 | 2,281 | 2,281 |
| Log Likelihood | -12,807 | -12,267 | -11,846 | -11,832 | -11,824 |
| Degrees of freedom | 8 | 19 | 26 | 29 | 35 |
| Wald chi2 | 58 | 1,062 | 2,361 | 2,381 | 2,393 |

*** p<0.01; ** p<0.05; * p<0.1

The results for the women (Table 5) show a similar pattern. The largest health differences are observed for Model 1, and these results gradually disappear with the inclusion of the different sets of covariates. In the full model, most immigrant groups do not show significantly different risks for experiencing the onset of IHD compared with Swedish women. The exceptions are women from Finland and Norway. Both groups show elevated risks, with the estimates for Norwegian women being significant only at the 90 percent level.

The results for women from Western countries are also interesting. They show strongly reduced risks consistently in all models, but in none of the models can the estimates be significantly distinguished from the reference group of Swedish women. One explanation for this result is the small sample size. There are only

139 women from Western countries in the sample who came between the years of 1955 and 1970 to Sweden as labor migrants and only three of those women experienced an IHD event during the observation period. The health difference of the Norwegian women is significant until the variables of occupation and branch are included. This indicates that the initially observed health differences are possibly due to differences in socioeconomic characteristics between women from Sweden and Norway.

In both cases men and women coming to Sweden as labor migrants between 1955 and 1970 show much lower health differences than in the unrestricted sample (Table 3) compared with natives in Sweden. Therefore, some of the initially observed country effects are actually due to differences in observable characteristics, and not in unobserved country-specific factors. The lack of significant differences in most of the immigrant groups in the full models suggests that the group of labor migrants arriving in Sweden in the mid-50s and 60s has adapted to the Swedish society and possibly adopted Swedish health patterns over the time of stay. The exception is the immigrant group from Finland, which shows elevated risk for the onset of IHD both in the restricted sample and after the inclusion of other covariates.

**Table 5**: Risk for first IHD in different immigrant groups, compared with native Swedes, women

| Women | Model 1 OR (95% CI) | Model 2 OR (95% CI) | Model 3 OR (95% CI) | Model 4 OR (95% CI) | Model 5 OR (95% CI) |
|---|---|---|---|---|---|
| Norway | 2.110*** (1.495 - 2.979) | 1.543** (1.091 - 2.183) | 1.464** (1.030 - 2.081) | 1.443** (1.014 - 2.052) | 1.358* (0.952 - 1.937) |
| Danmark | 1.427* (0.946 - 2.153) | 1.099 (0.727 - 1.661) | 1.123 (0.741 - 1.701) | 1.122 (0.740 - 1.700) | 1.080 (0.711 - 1.639) |
| Finland | 1.806*** (1.475 - 2.212) | 1.821*** (1.483 - 2.234) | 1.675*** (1.360 - 2.063) | 1.622*** (1.315 - 2.001) | 1.486*** (1.190 - 1.857) |
| Italy | 2.017*** (1.204 - 3.380) | 1.436 (0.855 - 2.411) | 1.282 (0.757 - 2.169) | 1.226 (0.724 - 2.077) | 1.097 (0.642 - 1.875) |
| F. Yugoslavia | 1.322* (0.997 - 1.754) | 1.339** (1.007 - 1.781) | 1.231 (0.921 - 1.644) | 1.176 (0.878 - 1.576) | 1.041 (0.763 - 1.419) |
| Germany | 1.317 (0.858 - 2.022) | 0.980 (0.637 - 1.507) | 1.058 (0.687 - 1.631) | 1.100 (0.713 - 1.698) | 1.053 (0.681 - 1.627) |
| West | 0.547 (0.176 - 1.705) | 0.409 (0.131 - 1.276) | 0.452 (0.145 - 1.414) | 0.504 (0.161 - 1.579) | 0.489 (0.156 - 1.534) |
| East | 1.735*** (1.317 - 2.286) | 1.176 (0.891 - 1.552) | 1.189 (0.897 - 1.576) | 1.281* (0.963 - 1.703) | 1.190 (0.889 - 1.592) |
| Age, age2 and year dummies | no | yes | yes | yes | yes |
| Social-medical covariates | no | no | yes | yes | yes |
| Educational and economical | no | no | no | yes | yes |
| Occupational covariates | no | no | no | no | yes |
| Observations | 159,955 | 159,955 | 159,955 | 159,955 | 159,955 |
| Number of indiv | 16,869 | 16,869 | 16,869 | 16,869 | 16,869 |
| IHD cases | 797 | 797 | 797 | 797 | 797 |
| Log Likelihood | -4,992 | -4,626 | -4,443 | -4,438 | -4,433 |
| Degrees of freedom | 8 | 19 | 26 | 29 | 35 |
| Wald chi2 | 61 | 674 | 1,303 | 1,306 | 1,315 |

*** p<0.01; ** p<0.05; * p<0.1

For men and women the addition of the covariates for occupation and branch of work reduces the observed health differences for many of the countries. Part of the hypothesis was that health differences between immigrants and natives may have their origin in an uneven distribution in the labor market. The results shown before indicate that occupation is influencing these health differences.

The effects of the covariates, which are displayed in the appendix, are according to expectations and the findings of established literature. Higher education is correlated with health benefits, especially for people in higher occupational positions. There is no significant effect for having underage children in the household. Although the three medical IHD risk factors (diabetes, hypertension

and atherosclerosis) are more common among men, the effect on IHD, once they are diagnosed in the individual, are stronger for women. The effect of income on the onset of IHD varies between the sex and among the models. The five-year-average income is assumed to give a more robust and long-term indication of the financial situation of the individual. Income from single time points could be biased by temporary fluctuations. Higher income was found to reduce the IHD risk for men significantly in the non-stratified models. In the occupationally stratified models, the results show very similar income effects for men, even though they are not significant. For women, a higher income appears to have no or even a harming effect. However, in no model is the effect of income is statistically significant for women.

In a further step this study will investigate if the health differences among the single country of origin groups vary by occupational group. In order to do so, occupational group stratified models are estimated. Stratifying the sample by occupational group will decrease the sample sizes of the separated strata. To demonstrate the size differences of the occupational groups, Table 6 shows the distribution of individuals by men and women stratified in the three different occupational groups of 1970.

**Table 6**: Sample stratified by sex and country of origin and occupational group in 1970

| **Men** | **% of all individuals per country of origin** | | |
|---|---|---|---|
| | **Upper white collar** | **White collar** | **Blue collar** |
| Sweden | 7.45 | 33.97 | 58.58 |
| Norway | 10.39 | 37.39 | 52.23 |
| Danmark | 9.84 | 14.76 | 75.40 |
| Finland | 2.68 | 10.97 | 86.35 |
| Italy | 4.36 | 18.60 | 77.04 |
| Former Yugoslavia | 1.44 | 4.52 | 94.03 |
| Germany | 10.51 | 32.88 | 56.61 |
| West | 12.79 | 45.12 | 42.09 |
| East | 11.34 | 26.68 | 61.98 |
| **Women** | **% of all individuals per country of origin** | | |
| | **Upper white collar** | **White collar** | **Blue collar** |
| Sweden | 2.87 | 55.54 | 41.59 |
| Norway | 3.43 | 30.21 | 66.36 |
| Danmark | 4.30 | 28.51 | 67.19 |
| Finland | 0.94 | 20.31 | 78.75 |
| Italy | 0.52 | 20.83 | 78.65 |
| Former Yugoslavia | 0.49 | 5.54 | 93.97 |
| Germany | 3.98 | 44.26 | 51.76 |
| West | 7.91 | 59.71 | 32.37 |
| East | 8.62 | 32.47 | 58.91 |

Data were taken from SLI database, calculations are the authors

Taking the native population as reference, some differences in the occupational distribution can be observed. Danish and Italian men, and especially men from Finland and former Yugoslavia, are much more concentrated in the blue-collar occupations than Swedish men. Men from Norway and the Western countries show a higher concentration in the white-collar occupation than Swedish men.

In comparison with the occupational distribution of the women, it important to notice that the majority of Swedish women worked in white-collar occupations, and only a very small share of women worked in upper white-collar positions. All other groups have the majority of individuals working in blue-collar occupations. The only exception is the group of women from Western countries, which exceed the native numbers of employees in the white and upper white-collar group.

Table 7 displays the results for the random effects logistic regression of IHD risk among the separated occupational groups, stratified by sex. As seen from the occupational distribution in Table 6, there are only very few women in 1970 who work in upper white-collar occupations. In the case of former Yugoslavia,

Germany and Western countries, there was no variation among the dependent variable for women in the upper white-collar group, consequently no odds ratios could be estimated for those groups. Taking account of the small numbers of women from other countries in this occupational group, the estimates from this group will not be interpreted further. The highly significant increased risk for Italian women in the upper white-collar group is based on only one IHD case among 23 individuals.

**Table** 7: Impact of country of origin on the risk of experiencing a first IHD case for men and women in models stratified by occupational group

| | Men | | | Women | | |
|---|---|---|---|---|---|---|
| **Country** | Blue collar | White collar | Upper white collar | Blue collar | White collar | Upper white collar |
| Norway | 0.971 | 1.105 | 1.508 | 1.562** | 0.677 | 2.514 |
| | (0.681 - 1.385) | (0.738 - 1.655) | (0.746 - 3.049) | (1.055 - 2.313) | (0.245 - 1.867) | (0.284 - 22.28) |
| Danmark | 1.12 | 0.958 | 1.469 | 1.117 | 0.947 | 0.848 |
| | (0.889 - 1.412) | (0.571 - 1.608) | (0.788 - 2.738) | (0.687 - 1.817) | (0.383 - 2.340) | (0.082 - 8.786) |
| Finland | 1.248** | 1.023 | 0.929 | 1.437*** | 1.590* | 3.039 |
| | (1.049 - 1.485) | (0.616 - 1.699) | (0.373 - 2.311) | (1.109 - 1.862) | (0.982 - 2.574) | (0.571 - 16.16) |
| Italy | 1.178 | 0.973 | 1.69 | 1.06 | 0.969 | 38.55*** |
| | (0.864 - 1.607) | (0.456 - 2.077) | (0.518 - 5.516) | (0.580 - 1.938) | (0.231 - 4.060) | (3.029 - 490.6) |
| F. Yugoslavia | 1.05 | 0.499 | 0.487 | 0.986 | 2.377 | - |
| | (0.863 - 1.277) | (0.159 - 1.566) | (0.067 - 3.534) | (0.707 - 1.375) | (0.734 - 7.698) | |
| Germany | 0.785 | 0.888 | 1.165 | 1.186 | 0.971 | - |
| | (0.549 - 1.124) | (0.556 - 1.420) | (0.574 - 2.363) | (0.685 - 2.053) | (0.469 - 2.007) | |
| West | 0.439** | 1.185 | 0.807 | 0.409 | 0.633 | - |
| | (0.195 - 0.990) | (0.687 - 2.042) | (0.249 - 2.616) | (0.057 - 2.947) | (0.155 - 2.590) | |
| East | 0.987 | 1.141 | 1.998*** | 1.148 | 1.276 | 1.596 |
| | (0.775 - 1.257) | (0.810 - 1.607) | (1.209 - 3.303) | (0.785 - 1.677) | (0.763 - 2.134) | (0.438 - 5.821) |
| Observations | 148,891 | 69,141 | 16,144 | 81,110 | 74,320 | 4,525 |
| Number of indiv | 16,300 | 7,486 | 1,832 | 8,655 | 7,722 | 492 |
| IHD cases | 1,452 | 619 | 210 | 495 | 270 | 32 |
| Log Likelihood | -7,506 | -3,257 | -1,025 | -2,701 | -1,547 | -147 |
| Degrees of freedom | 34 | 35 | 34 | 34 | 35 | 34 |
| Wald chi2 | 1,569 | 646 | 222 | 700 | 547 | 77 |

*** p<0.01; ** p<0.05; * p<0.1

Disregarding the results from the upper white-collar group, the main health differences between immigrants and natives can be observed for men within the blue-collar group. There are some variations in the white-collar group (e.g. former Yugoslavian men with OR = 0.5) but they are based on very few cases again. In the blue-collar group most foreign men show similar IHD risk than natives. Significant differences to the reference group can be observed for men from Finland and Western countries. While Finns show elevated IHD risk, men from Western countries show strongly reduced risk for IHD.

For women, the patterns are similar in the blue-collar group, with significantly higher IHD risk for Finnish women and reduced risk for women from Western countries. The latter estimate, although similar in magnitude as for men, is not significant different. Additionally, the group of Norwegian women shows a significantly elevated risk. For women there are also health variations among the group of white-collar workers, potentially triggered by the high concentration of women in those occupations.

Women from Finland show an even stronger health difference, compared with Sweden, in the white-collar group than in the blue-collar group, although only significant at the 90 percent level. There are more estimates which differ substantially from the reference group, but none of them is significant. In total the main health differences for IHD risk seems to concentrate in the blue-collar occupation for men and women.

Overall, the health disadvantages of men and women from Finland in the blue-collar groups as well of women in the white-collar group are the most pronounced. The Finnish immigrants are historically the largest immigrant group in Sweden, and, as seen in Table 6, the majority of Finns were working in the blue-collar sector in 1970, which is correlated with the strongest health disadvantage. Even when restricting the sample to this disadvantaged group, the Finnish health deficit remains strong.

Since the results so far showed that the occupational distribution appears to influence the health differences between immigrants and natives, additional analyses are done dividing the blue-collar group into blue-collar employment in the service sector and manual labor jobs (results not displayed). The hypothesis is that a concentration of Finns in the manual jobs, where the health disadvantage is assumed to be strongest, could trigger the strong health differences, observed in the earlier models. The manual jobs are the majority among the blue-collar occupations, and Finns correspondingly show a higher concentration among the manual jobs, even when compared with other immigrant groups. Logistic regression of the health differences among Finns stratified by the two blue-collar occupational groups showed that the health disadvantage for Finns was much lower in the sample of blue-collar service jobs compared with the sample of manual blue-collar jobs. For women, the health differences are present in both blue-collar groups, although somewhat stronger in the sample of service blue-collar occupation.

Occupational group affiliation was used as an indicator of socioeconomic position. As a sensitivity analysis, similarly stratified models are estimated using branch groups instead of occupation (results not displayed). The results are comparable to the analysis using occupation as an SES indicator. The strongest health differences are found in the manufacturing sector, were most of the immigrants worked by

1970. Again the immigrants from Finland showed significantly increased risks for IHD, compared with the native population.

## 4.6. Conclusions

This study investigates the risk for the onset of IHD for foreign-born individuals compared with natives in Sweden including the observation period 1992-2001. The hypothesis of this study is that differences in IHD risk between immigrants and natives, as they have been found in earlier studies, could be at least partially due to heterogeneity among the immigrant group, rather than the country of origin. In order to test this hypothesis, the sample was restricted to migrants who arrived between 1955 and 1970 in Sweden under the liberal migration policies and a period of active labor recruitment.

The results show that after cancelling out the possible bias from variations in length of stay, purpose of migration and socioeconomic integration via employment, hardly any difference in the onset of IHD could be identified among the immigrant countries. From the health differences between many of the immigrant groups and native Swedes, which were shown in an unrestricted immigrant sample, only the group of immigrants from Finland continued showing significantly higher IHD risk. Men and women from Finland show elevated risk for the onset of IHD compared with Swedish worker, even after controlling for several sets of potential IHD impact factors.

The lack of observed differences between most immigrant groups and Swedes after addition of socioeconomic controls supports the hypothesis that heterogeneity among the group of immigrants is driving many of the observed health differences found in earlier studies. Literature focusing on the case of Finnish individuals immigrating to Sweden has made a strong point of the health disadvantage of this specific group before and after the point of migration. The outstanding role of Finnish immigrants among other countries of origin was visible in the results of this study as well. The robust health differences among this group could arise from detrimental health behavior, such as high consumption of tobacco or alcohol, as well as dietary habits and other factors.

A second part of the hypothesis was that the uneven distribution of immigrants in the host country labor market could bias the health differences compared with natives. Because occupational group affiliation, which is used as indicator for socioeconomic status in this study, has an independent effect on the risk for IHD and immigrants, especially from the labor recruitment period, are concentrated in

the lower occupational groups, it stands to reason that observed health differences are, at least partially, a consequence of labor market distribution. Controlling for occupational class reduced the health difference to the Swedish comparison group. Occupational stratified models demonstrated that much of the health difference between immigrants and natives is concentrated among the lower occupational groups.

Given the special case of Swedish migration policy history and the strict sample selection resulting in a homogenous group of labor migrants, who have spent a substantial time in the host country, this study provides a possibility to more clearly focus on the effects of country of origin. The nearly complete lack of significant health differences shown in the results underlines the importance of accounting for heterogeneity of the immigrant groups in further studies.

The first of the two main findings of this study is that reducing the study population to a reasonably homogenous selection of labor migrants arriving in Sweden during the labor recruitment period reduced overall health differences among many of the groups of immigrants and the native population. On the one hand, these results could reflect successful integration of the labor migrants from the 50s and 60s in Sweden. Taking the immediate employment in the host country into account, which should have accelerated integration, the labor migrants from that period had a great advantage over the family reunification migrants and refugees arriving in Sweden in later periods. On the other hand, the study only analyzed immigrants who remained in Sweden until the beginning of the observation period. This could potentially bias the results if return migration was disproportionately a selection of ill-health individuals who preferred to return to their home countries.

The second main finding is the dramatically different position of immigrants from Finland, compared with both other immigrants and natives. Throughout all steps of analysis, the Finnish immigrant group showed a robust health disadvantage compared with Swedes. The occupationally stratified models demonstrated that most of the health difference between Finns and Swedes is concentrated among the lower occupational classes. Because the health difference persisted even with additional occupational covariate in the model, this study assumes unobserved health behavior as the origin of the health disadvantage. This leads to an important topic of study – if other immigrant groups show no significant difference to natives after controlling for heterogeneity, why do the Finns differ? It may be possible that the sheer size of the Finnish migrant group facilitated the retention of poor health behavior through a lack of integration, but this is a topic for future study.

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# 4.8. Appendix

**Table 3A**: Full estimates for Table 3, Logistic regression on IHD using the unrestricted sample of immigrants in Sweden from the SLI database, stratified by sex

| | Men | | Women | |
|---|---|---|---|---|
| | **Model A1** | **Model A2** | **Model A1** | **Model A2** |
| Variables | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** |
| Age | 1.337*** | 1.351*** | 1.330*** | 1.344*** |
| | (1.313 - 1.361) | (1.317 - 1.385) | (1.305 - 1.356) | (1.306 - 1.383) |
| Age, squared | 0.998*** | 0.998*** | 0.998*** | 0.998*** |
| | (0.998 - 0.998) | (0.998 - 0.999) | (0.998 - 0.998) | (0.998 - 0.998) |
| Norway | 1.225*** | 1.258** | 1.225*** | 1.217** |
| | (1.068 - 1.406) | (1.054 - 1.501) | (1.065 - 1.408) | (1.017 - 1.457) |
| Denmark | 1.033 | 0.956 | 0.984 | 0.946 |
| | (0.900 - 1.185) | (0.778 - 1.175) | (0.855 - 1.131) | (0.768 - 1.165) |
| Finland | 1.516*** | 1.672*** | 1.373*** | 1.442*** |
| | (1.365 - 1.684) | (1.475 - 1.896) | (1.233 - 1.529) | (1.267 - 1.640) |
| Italy | 1.241** | 1.077 | 1.198* | 0.908 |
| | (1.024 - 1.505) | (0.752 - 1.544) | (0.986 - 1.455) | (0.629 - 1.310) |
| F. Yugoslavia | 1.097 | 1.250*** | 1.06 | 1.08 |
| | (0.972 - 1.239) | (1.071 - 1.459) | (0.936 - 1.201) | (0.918 - 1.270) |
| Germany | 0.907 | 0.899 | 0.971 | 0.967 |
| | (0.762 - 1.078) | (0.712 - 1.136) | (0.815 - 1.157) | (0.763 - 1.224) |
| Western countries | 0.547*** | 0.663** | 0.592*** | 0.751 |
| | (0.412 - 0.727) | (0.448 - 0.980) | (0.444 - 0.789) | (0.506 - 1.114) |
| Eastern countries | 1.136** | 1.323*** | 1.152** | 1.366*** |
| | (1.019 - 1.268) | (1.167 - 1.498) | (1.030 - 1.289) | (1.200 - 1.554) |
| Other countries | 1.139*** | 1.140** | 1.076* | 0.986 |
| | (1.055 - 1.231) | (1.022 - 1.272) | (0.988 - 1.171) | (0.870 - 1.117) |
| Year 1993 | 0.922 | 1.148 | 0.903 | 1.125 |
| | (0.807 - 1.053) | (0.946 - 1.393) | (0.790 - 1.032) | (0.926 - 1.367) |
| Year 1994 | 0.968 | 1.121 | 0.936 | 1.079 |
| | (0.850 - 1.102) | (0.925 - 1.358) | (0.821 - 1.066) | (0.890 - 1.309) |
| Year 1995 | 0.949 | 1.279*** | 0.905 | 1.222** |
| | (0.834 - 1.080) | (1.064 - 1.538) | (0.795 - 1.030) | (1.015 - 1.471) |
| Year 1996 | 0.899 | 1.097 | 0.842*** | 1.033 |
| | (0.790 - 1.022) | (0.909 - 1.323) | (0.740 - 0.959) | (0.855 - 1.248) |
| Year 1997 | 0.735*** | 1.098 | 0.681*** | 1.019 |
| | (0.643 - 0.840) | (0.912 - 1.322) | (0.595 - 0.779) | (0.846 - 1.229) |
| Year 1998 | 0.779*** | 0.985 | 0.715*** | 0.899 |
| | (0.684 - 0.887) | (0.816 - 1.188) | (0.627 - 0.816) | (0.744 - 1.086) |
| Year 1999 | 0.786*** | 1.082 | 0.714*** | 0.984 |
| | (0.691 - 0.894) | (0.902 - 1.298) | (0.627 - 0.813) | (0.819 - 1.183) |
| Year 2000 | 0.743*** | 0.949 | 0.666*** | 0.849* |
| | (0.653 - 0.845) | (0.789 - 1.142) | (0.584 - 0.758) | (0.704 - 1.024) |
| Year 2001 | 0.722*** | 1.002 | 0.636*** | 0.885 |
| | (0.635 - 0.821) | (0.836 - 1.201) | (0.559 - 0.724) | (0.737 - 1.064) |

Continuation of **Table 3A**

| | Men | | Women | |
|---|---|---|---|---|
| | **Model A1** | **Model A2** | **Model A1** | **Model A2** |
| Variables | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** | **OR (95% CI)** |
| Married | | | 1.148*** | 1.07 |
| | | | (1.047 - 1.258) | (0.935 - 1.225) |
| Divorced | | | 1.269*** | 1.159** |
| | | | (1.147 - 1.404) | (1.003 - 1.339) |
| Widowed | | | 1.141* | 1.109 |
| | | | (0.976 - 1.334) | (0.958 - 1.283) |
| Child subsidy | | | 1.126** | 0.992 |
| | | | (1.029 - 1.233) | (0.833 - 1.181) |
| Hypertension | | | 4.177*** | 4.919*** |
| | | | (3.829 - 4.556) | (4.430 - 5.461) |
| Diabetes | | | 3.619*** | 3.451*** |
| | | | (3.301 - 3.967) | (3.073 - 3.876) |
| Atherosclerosis | | | 2.040*** | 2.082*** |
| | | | (1.702 - 2.446) | (1.604 - 2.704) |
| Secondary edu. | | | 0.983 | 0.899** |
| | | | (0.912 - 1.060) | (0.809 - 1.000) |
| University edu. | | | 0.758*** | 0.650*** |
| | | | (0.703 - 0.818) | (0.572 - 0.739) |
| Income | | | 0.992 | 0.994 |
| | | | (0.981 - 1.004) | (0.979 - 1.009) |
| Constant | 0.000 | 0.000 | 0.000 | 0.000 |
| | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) |
| Observations | 1,486,761 | 1,516,962 | 1,486,761 | 1,516,962 |
| Number of indiv | 182,064 | 183,183 | 182,064 | 183,183 |
| Log Likelihood | -26,908 | -16,095 | -25,824 | -15,307 |
| Degrees of freedom | 20 | 20 | 30 | 30 |
| Wald chi2 | 4,790 | 3,281 | 8,470 | 6,074 |

*** p<0.01 ** p<0.05 * p<0.1

**Table 4A**: Full estimates from Table 4, logistic regression on the onset of IHD for different immigrant groups compared with Swedes, including sets of covariates step-wise, men

| Variables | Model 1<br>OR (95% CI) | Model 2<br>OR (95% CI) | Model 3<br>OR (95% CI) | Model 4<br>OR (95% CI) | Model 5<br>OR (95% CI) |
|---|---|---|---|---|---|
| Age | | 1.213*** | 1.195*** | 1.212*** | 1.215*** |
| | | (1.164 - 1.265) | (1.145 - 1.248) | (1.160 - 1.266) | (1.163 - 1.269) |
| Age, squared | | 0.999*** | 0.999*** | 0.999*** | 0.999*** |
| | | (0.999 - 0.999) | (0.999 - 0.999) | (0.999 - 0.999) | (0.999 - 0.999) |
| Norway | 1.319** | 1.001 | 1.012 | 1.055 | 1.071 |
| | (1.034 - 1.681) | (0.784 - 1.278) | (0.792 - 1.294) | (0.824 - 1.350) | (0.836 - 1.372) |
| Denmark | 1.698*** | 1.232** | 1.154 | 1.141 | 1.119 |
| | (1.402 - 2.058) | (1.015 - 1.494) | (0.948 - 1.404) | (0.937 - 1.389) | (0.917 - 1.364) |
| Finland | 1.291*** | 1.371*** | 1.292*** | 1.229*** | 1.227** |
| | (1.112 - 1.499) | (1.179 - 1.593) | (1.110 - 1.504) | (1.055 - 1.433) | (1.048 - 1.438) |
| Italy | 1.313** | 1.184 | 1.203 | 1.179 | 1.183 |
| | (1.002 - 1.722) | (0.902 - 1.554) | (0.915 - 1.580) | (0.897 - 1.550) | (0.897 - 1.561) |
| F. Yugoslavia | 1.164* | 1.093 | 1.089 | 1.054 | 1.049 |
| | (0.983 - 1.378) | (0.922 - 1.296) | (0.916 - 1.294) | (0.886 - 1.253) | (0.873 - 1.261) |
| Germany | 0.949 | 0.833 | 0.81 | 0.855 | 0.854 |
| | (0.734 - 1.227) | (0.644 - 1.078) | (0.625 - 1.049) | (0.658 - 1.111) | (0.657 - 1.111) |
| Western countries | 0.962 | 0.73 | 0.734 | 0.788 | 0.793 |
| | (0.636 - 1.455) | (0.482 - 1.105) | (0.484 - 1.113) | (0.519 - 1.197) | (0.521 - 1.205) |
| Eastern countries | 1.474*** | 1.123 | 1.072 | 1.131 | 1.125 |
| | (1.243 - 1.748) | (0.945 - 1.333) | (0.900 - 1.276) | (0.948 - 1.349) | (0.939 - 1.348) |
| Year 1993 | | 0.814** | 0.799** | 0.800** | 0.799** |
| | | (0.676 - 0.982) | (0.662 - 0.964) | (0.663 - 0.965) | (0.663 - 0.965) |
| Year 1994 | | 1.019 | 0.984 | 0.985 | 0.985 |
| | | (0.855 - 1.214) | (0.824 - 1.173) | (0.826 - 1.176) | (0.826 - 1.175) |
| Year 1995 | | 0.91 | 0.862 | 0.865 | 0.864 |
| | | (0.761 - 1.089) | (0.720 - 1.032) | (0.723 - 1.036) | (0.722 - 1.035) |
| Year 1996 | | 0.865 | 0.804** | 0.807** | 0.806** |
| | | (0.723 - 1.036) | (0.670 - 0.963) | (0.673 - 0.967) | (0.672 - 0.966) |
| Year 1997 | | 0.728*** | 0.668*** | 0.671*** | 0.670*** |
| | | (0.605 - 0.878) | (0.553 - 0.806) | (0.556 - 0.810) | (0.556 - 0.809) |
| Year 1998 | | 0.734*** | 0.667*** | 0.671*** | 0.670*** |
| | | (0.610 - 0.883) | (0.553 - 0.804) | (0.557 - 0.809) | (0.556 - 0.808) |
| Year 1999 | | 0.809** | 0.724*** | 0.730*** | 0.728*** |
| | | (0.676 - 0.968) | (0.604 - 0.868) | (0.609 - 0.875) | (0.607 - 0.873) |
| Year 2000 | | 0.654*** | 0.576*** | 0.583*** | 0.581*** |
| | | (0.542 - 0.790) | (0.476 - 0.697) | (0.482 - 0.705) | (0.480 - 0.703) |
| Year 2001 | | 0.657*** | 0.566*** | 0.574*** | 0.572*** |
| | | (0.545 - 0.792) | (0.469 - 0.684) | (0.475 - 0.694) | (0.473 - 0.691) |

Continuation of **Table 4A**

| Variables | Model 1 OR (95% CI) | Model 2 OR (95% CI) | Model 3 OR (95% CI) | Model 4 OR (95% CI) | Model 5 OR (95% CI) |
|---|---|---|---|---|---|
| Married | | | 1.142** | 1.173*** | 1.178*** |
| | | | (1.015 - 1.284) | (1.042 - 1.321) | (1.046 - 1.326) |
| Divorced | | | 1.174** | 1.180** | 1.175** |
| | | | (1.027 - 1.341) | (1.032 - 1.349) | (1.028 - 1.344) |
| Widowed | | | 1.132 | 1.151 | 1.156 |
| | | | (0.929 - 1.378) | (0.945 - 1.402) | (0.949 - 1.408) |
| Child subsidy | | | 0.947 | 0.959 | 0.957 |
| | | | (0.807 - 1.110) | (0.817 - 1.125) | (0.816 - 1.123) |
| Hypertension | | | 3.791*** | 3.819*** | 3.840*** |
| | | | (3.355 - 4.284) | (3.380 - 4.315) | (3.398 - 4.339) |
| Diabetes | | | 3.146*** | 3.107*** | 3.119*** |
| | | | (2.745 - 3.605) | (2.711 - 3.560) | (2.721 - 3.574) |
| Atherosclerosis | | | 1.940*** | 1.918*** | 1.923*** |
| | | | (1.489 - 2.528) | (1.472 - 2.499) | (1.476 - 2.505) |
| Secondary edu. | | | | 0.981 | 1.007 |
| | | | | (0.886 - 1.087) | (0.908 - 1.117) |
| University edu. | | | | 0.776*** | 0.815*** |
| | | | | (0.695 - 0.866) | (0.718 - 0.926) |
| Income | | | | 0.967* | 0.970* |
| | | | | (0.934 - 1.000) | (0.936 - 1.004) |
| Immigrant share | | | | | 0.994* |
| | | | | | (0.987 - 1.001) |
| Upper white collar | | | | | 0.964 |
| | | | | | (0.813 - 1.141) |
| White collar | | | | | 0.829*** |
| | | | | | (0.742 - 0.926) |
| Manufacturing | | | | | 0.925 |
| | | | | | (0.786 - 1.089) |
| Retail and transport | | | | | 0.908 |
| | | | | | (0.766 - 1.078) |
| Private sector | | | | | 0.991 |
| | | | | | (0.816 - 1.204) |
| Constant | 0.009*** | 0.000 | 0.000 | 0.000 | 0.000 |
| | (0.009 - 0.010) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) |
| Observations | 234,176 | 234,176 | 234,176 | 234,176 | 234,176 |
| Number of indiv | 25,618 | 25,618 | 25,618 | 25,618 | 25,618 |
| Log Likelihood | -12807 | -12267 | -11846 | -11832 | -11824 |
| Degrees of freedom | 8 | 19 | 26 | 29 | 35 |
| Wald test (chi2) | 57.94 | 1062 | 2361 | 2381 | 2393 |

*** p<0.01, ** p<0.05, * p<0.1

**Table 5A**: Full estimates from Table 5, logistic regression on the onset of IHD for different immigrant groups compared with Swedes, including sets of covariates step-wise, women

| Variables | Model 1<br>OR (95% CI) | Model 2<br>OR (95% CI) | Model 3<br>OR (95% CI) | Model 4<br>OR (95% CI) | Model 5<br>OR (95% CI) |
|---|---|---|---|---|---|
| Age | | 1.284*** | 1.242*** | 1.238*** | 1.235*** |
| | | (1.199 - 1.374) | (1.153 - 1.338) | (1.150 - 1.333) | (1.147 - 1.330) |
| Age, squared | | 0.999*** | 0.999*** | 0.999*** | 0.999*** |
| | | (0.998 - 0.999) | (0.998 - 0.999) | (0.998 - 0.999) | (0.998 - 0.999) |
| Norway | 2.110*** | 1.543** | 1.464** | 1.443** | 1.358* |
| | (1.495 - 2.979) | (1.091 - 2.183) | (1.030 - 2.081) | (1.014 - 2.052) | (0.952 - 1.937) |
| Denmark | 1.427* | 1.099 | 1.123 | 1.122 | 1.08 |
| | (0.946 - 2.153) | (0.727 - 1.661) | (0.741 - 1.701) | (0.740 - 1.700) | (0.711 - 1.639) |
| Finland | 1.806*** | 1.821*** | 1.675*** | 1.622*** | 1.486*** |
| | (1.475 - 2.212) | (1.483 - 2.234) | (1.360 - 2.063) | (1.315 - 2.001) | (1.190 - 1.857) |
| Italy | 2.017*** | 1.436 | 1.282 | 1.226 | 1.097 |
| | (1.204 - 3.380) | (0.855 - 2.411) | (0.757 - 2.169) | (0.724 - 2.077) | (0.642 - 1.875) |
| F. Yugoslavia | 1.322* | 1.339** | 1.231 | 1.176 | 1.041 |
| | (0.997 - 1.754) | (1.007 - 1.781) | (0.921 - 1.644) | (0.878 - 1.576) | (0.763 - 1.419) |
| Germany | 1.317 | 0.98 | 1.058 | 1.1 | 1.053 |
| | (0.858 - 2.022) | (0.637 - 1.507) | (0.687 - 1.631) | (0.713 - 1.698) | (0.681 - 1.627) |
| Western countries | 0.547 | 0.409 | 0.452 | 0.504 | 0.489 |
| | (0.176 - 1.705) | (0.131 - 1.276) | (0.145 - 1.414) | (0.161 - 1.579) | (0.156 - 1.534) |
| Eastern countries | 1.735*** | 1.176 | 1.189 | 1.281* | 1.19 |
| | (1.317 - 2.286) | (0.891 - 1.552) | (0.897 - 1.576) | (0.963 - 1.703) | (0.889 - 1.592) |
| Year 1993 | | 1.047 | 1.025 | 1.028 | 1.027 |
| | | (0.744 - 1.474) | (0.727 - 1.446) | (0.729 - 1.449) | (0.728 - 1.449) |
| Year 1994 | | 1.163 | 1.124 | 1.129 | 1.127 |
| | | (0.836 - 1.618) | (0.806 - 1.566) | (0.810 - 1.573) | (0.808 - 1.571) |
| Year 1995 | | 1.149 | 1.096 | 1.104 | 1.103 |
| | | (0.829 - 1.594) | (0.789 - 1.523) | (0.794 - 1.534) | (0.793 - 1.532) |
| Year 1996 | | 0.989 | 0.93 | 0.94 | 0.939 |
| | | (0.708 - 1.382) | (0.664 - 1.301) | (0.671 - 1.315) | (0.671 - 1.315) |
| Year 1997 | | 0.951 | 0.885 | 0.896 | 0.896 |
| | | (0.682 - 1.328) | (0.633 - 1.237) | (0.641 - 1.254) | (0.640 - 1.253) |
| Year 1998 | | 0.731* | 0.665** | 0.676** | 0.675** |
| | | (0.515 - 1.037) | (0.467 - 0.946) | (0.475 - 0.962) | (0.474 - 0.961) |
| Year 1999 | | 1.067 | 0.963 | 0.982 | 0.98 |
| | | (0.776 - 1.468) | (0.698 - 1.329) | (0.711 - 1.355) | (0.709 - 1.353) |
| Year 2000 | | 0.921 | 0.817 | 0.834 | 0.832 |
| | | (0.665 - 1.275) | (0.588 - 1.134) | (0.600 - 1.159) | (0.598 - 1.156) |
| Year 2001 | | 0.87 | 0.757* | 0.775 | 0.773 |
| | | (0.628 - 1.206) | (0.545 - 1.053) | (0.557 - 1.079) | (0.555 - 1.076) |

Continuation of **Table 5A**

| Variables | Model 1<br>OR (95% CI) | Model 2<br>OR (95% CI) | Model 3<br>OR (95% CI) | Model 4<br>OR (95% CI) | Model 5<br>OR (95% CI) |
|---|---|---|---|---|---|
| Married | | | 1.007 | 1.024 | 1.02 |
| | | | (0.812 - 1.248) | (0.826 - 1.270) | (0.822 - 1.265) |
| Divorced | | | 1.013 | 1.026 | 1.018 |
| | | | (0.808 - 1.272) | (0.818 - 1.289) | (0.811 - 1.279) |
| Widowed | | | 0.985 | 0.98 | 0.974 |
| | | | (0.783 - 1.241) | (0.777 - 1.235) | (0.773 - 1.228) |
| Child subsidy | | | 0.82 | 0.832 | 0.834 |
| | | | (0.553 - 1.216) | (0.561 - 1.235) | (0.562 - 1.237) |
| Hypertension | | | 3.994*** | 3.961*** | 3.958*** |
| | | | (3.278 - 4.866) | (3.251 - 4.827) | (3.247 - 4.824) |
| Diabetes | | | 3.653*** | 3.623*** | 3.593*** |
| | | | (2.916 - 4.576) | (2.891 - 4.541) | (2.865 - 4.506) |
| Atherosclerosis | | | 1.569 | 1.582* | 1.617* |
| | | | (0.911 - 2.701) | (0.919 - 2.725) | (0.938 - 2.787) |
| Secondary edu. | | | | 0.912 | 0.943 |
| | | | | (0.770 - 1.081) | (0.792 - 1.122) |
| University edu. | | | | 0.702*** | 0.750** |
| | | | | (0.561 - 0.879) | (0.585 - 0.961) |
| Income | | | | 1.047 | 1.051 |
| | | | | (0.954 - 1.150) | (0.955 - 1.157) |
| Immigrant share | | | | | 1.018*** |
| | | | | | (1.005 - 1.031) |
| Upper white collar | | | | | 1.084 |
| | | | | | (0.740 - 1.588) |
| White collar | | | | | 0.932 |
| | | | | | (0.778 - 1.115) |
| Manufacturing | | | | | 0.959 |
| | | | | | (0.785 - 1.171) |
| Retail and transport | | | | | 1.089 |
| | | | | | (0.891 - 1.332) |
| Private sector | | | | | 0.96 |
| | | | | | (0.741 - 1.244) |
| Constant | 0.004*** | 0.000 | 0.000 | 0.000 | 0.000 |
| | (0.004 - 0.005) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) |
| Observations | 159,955 | 159,955 | 159,955 | 159,955 | 159,955 |
| Number of indiv | 16,869 | 16,869 | 16,869 | 16,869 | 16,869 |
| Log Likelihood | -4992 | -4626 | -4443 | -4438 | -4433 |
| Degrees of freedom | 8 | 19 | 26 | 29 | 35 |
| Wald test (chi2) | 60.61 | 673.5 | 1303 | 1306 | 1315 |

*** p<0.01, ** p<0.05, * p<0.1

**Table 7A**: Full estimates of Table 7: Logistic regression on IHD risk by immigrants and natives experienced in the various occupational groups, stratified by sex

| | Men | | | Women | | |
|---|---|---|---|---|---|---|
| Variables | Blue collar | White collar | Upper white collar | Blue collar | White collar | Upper white collar |
| Age | 1.222*** | 1.209*** | 1.209** | 1.226*** | 1.304*** | 0.884 |
| | (1.157 - 1.290) | (1.107 - 1.320) | (1.012 - 1.444) | (1.115 - 1.347) | (1.145 - 1.486) | (0.569 - 1.374) |
| Age (suqared) | 0.999*** | 0.999*** | 0.999 | 0.999*** | 0.999*** | 1.002 |
| | (0.998 - 0.999) | (0.998 - 1.000) | (0.998 - 1.000) | (0.998 - 1.000) | (0.998 - 1.000) | (0.999 - 1.005) |
| Norway | 0.971 | 1.105 | 1.508 | 1.562** | 0.677 | 2.514 |
| | (0.681 - 1.385) | (0.738 - 1.655) | (0.746 - 3.049) | (1.055 - 2.313) | (0.245 - 1.867) | (0.284 - 22.28) |
| Danmark | 1.12 | 0.958 | 1.469 | 1.117 | 0.947 | 0.848 |
| | (0.889 - 1.412) | (0.571 - 1.608) | (0.788 - 2.738) | (0.687 - 1.817) | (0.383 - 2.340) | (0.0819 - 8.786) |
| Finland | 1.248** | 1.023 | 0.929 | 1.437*** | 1.590* | 3.039 |
| | (1.049 - 1.485) | (0.616 - 1.699) | (0.373 - 2.311) | (1.109 - 1.862) | (0.982 - 2.574) | (0.571 - 16.16) |
| Italy | 1.178 | 0.973 | 1.69 | 1.06 | 0.969 | 38.55*** |
| | (0.864 - 1.607) | (0.456 - 2.077) | (0.518 - 5.516) | (0.580 - 1.938) | (0.231 - 4.060) | (3.029 - 490.6) |
| F. Yugoslavia | 1.05 | 0.499 | 0.487 | 0.986 | 2.377 | |
| | (0.863 - 1.277) | (0.159 - 1.566) | (0.0670 - 3.534) | (0.707 - 1.375) | (0.734 - 7.698) | |
| Germany | 0.785 | 0.888 | 1.165 | 1.186 | 0.971 | |
| | (0.549 - 1.124) | (0.556 - 1.420) | (0.574 - 2.363) | (0.685 - 2.053) | (0.469 - 2.007) | |
| Western countries | 0.439** | 1.185 | 0.807 | 0.409 | 0.633 | |
| | (0.195 - 0.990) | (0.687 - 2.042) | (0.249 - 2.616) | (0.0567 - 2.947) | (0.155 - 2.590) | |
| Eastern countries | 0.987 | 1.141 | 1.998*** | 1.148 | 1.276 | 1.596 |
| | (0.775 - 1.257) | (0.810 - 1.607) | (1.209 - 3.303) | (0.785 - 1.677) | (0.763 - 2.134) | (0.438 - 5.821) |
| Year 1993 | 0.826 | 0.813 | 0.647 | 1.07 | 0.889 | 1.479 |
| | (0.656 - 1.041) | (0.546 - 1.212) | (0.372 - 1.125) | (0.704 - 1.627) | (0.467 - 1.692) | (0.238 - 9.186) |
| Year 1994 | 1.003 | 1.236 | 0.477** | 1.122 | 1.183 | 0.835 |
| | (0.806 - 1.248) | (0.864 - 1.768) | (0.262 - 0.869) | (0.746 - 1.687) | (0.650 - 2.152) | (0.113 - 6.161) |
| Year 1995 | 0.872 | 1.048 | 0.528** | 0.875 | 1.474 | 2.167 |
| | (0.697 - 1.090) | (0.727 - 1.512) | (0.297 - 0.936) | (0.572 - 1.339) | (0.837 - 2.596) | (0.414 - 11.33) |
| Year 1996 | 0.745** | 1.056 | 0.702 | 0.861 | 1.103 | 0.844 |
| | (0.592 - 0.938) | (0.735 - 1.517) | (0.412 - 1.197) | (0.563 - 1.315) | (0.614 - 1.984) | (0.130 - 5.464) |
| Year 1997 | 0.639*** | 0.886 | 0.478** | 0.841 | 0.967 | 1.096 |
| | (0.505 - 0.810) | (0.611 - 1.286) | (0.264 - 0.864) | (0.551 - 1.283) | (0.535 - 1.747) | (0.185 - 6.484) |
| Year 1998 | 0.645*** | 0.936 | 0.355*** | 0.706 | 0.633 | 0.519 |
| | (0.510 - 0.815) | (0.649 - 1.349) | (0.187 - 0.676) | (0.457 - 1.090) | (0.335 - 1.197) | (0.0680 - 3.961) |
| Year 1999 | 0.735*** | 0.756 | 0.693 | 0.955 | 1.049 | 0.699 |
| | (0.587 - 0.921) | (0.517 - 1.105) | (0.408 - 1.179) | (0.638 - 1.430) | (0.595 - 1.851) | (0.107 - 4.564) |
| Year 2000 | 0.571*** | 0.772 | 0.312*** | 0.744 | 0.97 | 0.761 |
| | (0.450 - 0.725) | (0.530 - 1.123) | (0.161 - 0.606) | (0.488 - 1.133) | (0.549 - 1.712) | (0.123 - 4.710) |
| Year 2001 | 0.537*** | 0.737 | 0.475** | 0.915 | 0.542* | 0.51 |
| | (0.423 - 0.682) | (0.506 - 1.075) | (0.267 - 0.845) | (0.613 - 1.367) | (0.290 - 1.015) | (0.0746 - 3.480) |
| Married | 1.123 | 1.278** | 1.27 | 0.994 | 1.143 | 0.92 |
| | (0.969 - 1.301) | (1.019 - 1.603) | (0.805 - 2.005) | (0.751 - 1.314) | (0.800 - 1.633) | (0.238 - 3.559) |
| Divorced | 1.097 | 1.278* | 1.441 | 1.086 | 0.951 | 1.204 |
| | (0.931 - 1.291) | (0.978 - 1.669) | (0.864 - 2.403) | (0.812 - 1.452) | (0.645 - 1.403) | (0.276 - 5.255) |
| Widowed | 1.032 | 1.308 | 1.56 | 1.042 | 0.914 | 1.034 |
| | (0.798 - 1.335) | (0.920 - 1.861) | (0.799 - 3.048) | (0.774 - 1.402) | (0.613 - 1.364) | (0.310 - 3.451) |
| Child subsidy | 0.939 | 0.915 | 1.376 | 0.847 | 0.808 | 1.965 |
| | (0.771 - 1.145) | (0.665 - 1.259) | (0.819 - 2.312) | (0.508 - 1.411) | (0.423 - 1.546) | (0.185 - 20.84) |
| Hypertension | 3.811*** | 3.839*** | 4.183*** | 3.865*** | 3.872*** | 13.53*** |
| | (3.256 - 4.461) | (3.059 - 4.819) | (2.854 - 6.131) | (3.019 - 4.948) | (2.685 - 5.583) | (4.929 - 37.14) |
| Diabetes | 3.322*** | 2.590*** | 3.637*** | 3.218*** | 5.213*** | 2.766 |
| | (2.810 - 3.928) | (1.964 - 3.414) | (2.302 - 5.746) | (2.431 - 4.260) | (3.481 - 7.808) | (0.668 - 11.45) |
| Atherosclerosis | 2.243*** | 1.216 | 1.918 | 1.391 | 3.878*** | 0 |
| | (1.652 - 3.046) | (0.658 - 2.247) | (0.646 - 5.692) | (0.692 - 2.796) | (1.600 - 9.399) | (0 - ) |
| Secondary edu. | 1.027 | 0.98 | 0.998 | 0.896 | 0.92 | 0.702 |
| | (0.906 - 1.164) | (0.785 - 1.223) | (0.666 - 1.493) | (0.715 - 1.122) | (0.679 - 1.246) | (0.221 - 2.235) |
| University edu. | 0.864 | 0.848 | 0.560*** | 0.693* | 0.721* | 1.222 |
| | (0.720 - 1.037) | (0.675 - 1.065) | (0.366 - 0.858) | (0.461 - 1.042) | (0.499 - 1.041) | (0.345 - 4.327) |
| Upward mobility | 0.839* | 0.895 | | 1.088 | 1.121 | |
| | (0.703 - 1.002) | (0.709 - 1.130) | | (0.850 - 1.391) | (0.788 - 1.594) | |
| Downward mobility | | 1.141 | 1.081 | | 0.767 | 0.697 |
| | | (0.906 - 1.438) | (0.787 - 1.484) | | (0.467 - 1.261) | (0.247 - 1.962) |
| Income | 0.963* | 0.981 | 0.995 | 1.005 | 1.076 | 1.411 |
| | (0.922 - 1.005) | (0.910 - 1.059) | (0.900 - 1.099) | (0.899 - 1.125) | (0.908 - 1.274) | (0.724 - 2.749) |
| Immigrant share in branch | 0.995 | 0.997 | 0.971 | 1.015* | 1.012 | 1.147*** |
| | (0.987 - 1.003) | (0.982 - 1.012) | (0.937 - 1.007) | (1.000 - 1.030) | (0.979 - 1.046) | (1.059 - 1.243) |
| Manufacturing sector | 1.103 | 0.883 | 1.106 | 1.023 | 0.941 | 2.323 |
| | (0.807 - 1.508) | (0.690 - 1.131) | (0.662 - 1.850) | (0.805 - 1.299) | (0.610 - 1.453) | (0.619 - 8.715) |
| Retail and Transport | 1.168 | 0.852 | 0.673* | 1.277* | 0.913 | 2.4 |
| | (0.840 - 1.624) | (0.652 - 1.114) | (0.432 - 1.049) | (0.974 - 1.676) | (0.656 - 1.270) | (0.564 - 10.21) |
| Private sector | 1.253 | 0.934 | 0.81 | 1.022 | 0.868 | 2.03 |
| | (0.858 - 1.831) | (0.695 - 1.255) | (0.513 - 1.279) | (0.710 - 1.469) | (0.575 - 1.310) | (0.525 - 7.841) |
| Constant | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) |
| Observations | 148,891 | 69,141 | 16,144 | 81,110 | 74,320 | 4,525 |
| Number of indiv | 16,300 | 7,486 | 1,832 | 8,655 | 7,722 | 492 |
| Log Likelihood | -7,506 | -3,257 | -1,025 | -2,701 | -1,547 | -147 |
| Degrees of freedom | 34 | 35 | 34 | 34 | 35 | 34 |
| Wald chi2 | 1,569 | 646 | 222 | 700 | 547 | 77 |

*** p<0.01, ** p<0.05, * p<0.1

Chapter 5

# Intermarriage and its Impact Paths to Ischemic Heart Disease

## 5.1. Abstract

This paper combines the repeatedly found results of health differences between immigrants and natives as well as the health advantage of married people over non-married people. It is the first paper investigating the potential health effects of intermarriage. Exploring a database on 83,000 natives and immigrants in Sweden, the study analyzes the onset of Ischemic Heart Disease (IHD) among people in endogamous and exogamous relationships, defined as relationship between individuals of the same country of origin or individuals born in different countries. Logistic regression analysis with step-wise added covariates show a significant health advantage for foreign-born men if they are married to a native. Further, the study investigates possible indirect effects of intermarriage on the established IHD risk factors income, education, hypertension and diabetes. Individuals in exogamous relationships show different associations, depending on the risk factor and origin and sex of the individual. Overall, there are strong indicators that intermarriage is correlated directly and indirectly with the risk for the onset of IHD in the analyzed sample of natives and immigrants in Sweden.

## 5.2. Introduction

During the second half of the 20th century, Sweden became an immigrant country. As a result of several waves of immigrant inflow, a substantial part of the Swedish population is now foreign-born or has foreign-born parents (Bengtsson et al 2005). In 1970, after the first wave of migrants who primarily sought labor jobs, 6.7 percent of the population in Sweden was born outside of the national borders; by 2009, this proportion had risen to 14.3 percent. According to official statistics, foreign-born individuals and individuals born in Sweden to

two foreign-born parents account for 18.6 percent of the population (Statistics Sweden 2011).

Foreign-born individuals in Sweden come from a variety of different countries and migrated to Sweden for different purposes and at different times. This significant foreign cultural influence affects more than Sweden's population size. Several international studies have shown that foreigners and natives often do not follow the same health patterns (Dassanayake et al 2011; Gadd et al 2003; Singh and Siahpush 2002). The differences in the health patterns and health behaviors of natives and foreigners depend on many factors at the macro level (e.g., integration policies, discrimination, and the economic condition of the host country) and at the individual level (e.g., an individual's demographic background, level of human capital, and health behaviors). Studies have shown that immigrants are more likely to adapt to local health behaviors the longer the stay in their host country; therefore, compared with new immigrants, long-term immigrants are assumed to have health patterns that are more similar to those of natives (Albin et al 2005; Lebrun 2012; Wiking et al 2004).

In developed countries, such as Sweden, diseases of the cardiovascular system (CVD) are the main cause of death (WHO 2011a). Given the extensive medical interventions required for its prevention and care, CVD is a cost-intensive epidemic. Medication, treatment, and hospitalization expenses for CVD patients accounted for eight percent of the total healthcare cost in Sweden in 2010 (Steen Carlsson and Persson 2012). Given that individuals from different countries have different CVD patterns (Pudaric et al 2000; Sundquist and Johansson 1997), it can be assumed that the large proportion of foreign-born individuals in Sweden has substantially affected the national pattern of CVD development in recent decades (Dotevall et al 2000) and will continue to do so in future decades.

Ischemic heart disease (IHD) is the largest subgroup of CVD. The occurrence of IHD depends strongly on individual demographic and socioeconomic factors. One remarkably consistent finding is that married people have a reduced risk for the onset and course of IHD. Many studies have confirmed that married people and people in stable partnerships have a health advantage compared with single people (Gémes et al 2008; Pudaric et al 2000). For married people, the partner provides support and resources that act as a psychosocial stress buffer (Lett et al 2005). The differences between never-married and people in partnerships are small but consistent. Newly divorced and widowed people are at a considerable health disadvantage as well (Molloy et al 2009). Especially for widowers, the abrupt loss of the partner can lead to immediate health problems, including shock, arrhythmia, or ischemia of the heart.

When investigating a population such as Sweden's, with a high proportion of foreign-born individuals, marriage cannot be viewed as a dichotomous

characteristic (married vs. unmarried). The literature has shown that intermarriage (in this case, the marriage between a native and a foreign-born) affects the couple's socioeconomic status (SES) (Meng and Gregory 2005). Intermarriage can be seen as indicator of a high level of integration (Dribe and Lundh 2012; Song 2009), regardless of whether the partnership resulted from a high level of integration (Chiswick and Houseworth 2011; Kalmijn and van Tubergen 2006) or was the cause for efforts to achieve better integration (Dribe and Lundh 2008; Kalmijn 1998). In return, a high level of integration among the foreign members of the population could explain reductions in the health differences between foreign-born and natives. For foreign-born individuals, having a native partner could facilitate access to health-related information and services. However, intermarriage could also present potential health risks. Studies have found a higher risk for union dissolution among partnerships in which the partners have different ethnicities (Dribe and Lundh 2012; Eeckhaut et al 2011).

While the literature on intermarriage mainly focuses on the factors leading to intermarriage and the socioeconomic consequences of intermarriage for the couple and society as a whole, this study focuses on the potential health consequences of intermarriage for the individual.

This study investigates the effects of intermarriage among foreigners and natives in Sweden and their potential effect on the onset of IHD. More precisely, this study analyzes whether IHD patterns differ according to an individual's country of birth or his/her partner's country of origin. The hypothesis is that foreign-born individuals who are married to a native will have a lower risk for IHD. On the one hand, this beneficial health effect could result from assortative matching among immigrants who have attributes that are considered favorable in the Swedish marriage market. On the other hand, a native partner could improve the foreign individual's integration process and provide him/her with knowledge of, and access to, country-specific health-related assets.

## 5.3. Theoretical background

### Migration history in Sweden

The analysis of the health effects of intermarriage, which is defined as a partnership between two people born in different countries, requires a certain proportion of immigrants in a given country. As mentioned in the introduction, Sweden has a rather large proportion of foreign-born individuals, most of who arrived during

the second half of the 20th century. To take into account the cultural and ethnic differences of this heterogeneous immigrant group, it is important to understand Sweden's unique migration history. The following paragraphs provide a brief overview of Sweden's migration waves and the origins of the current share of immigrants.

Since the Second World War, Sweden has been an immigrant country, with several strong waves of immigrants arriving for different reasons and from different countries. After the war-related immigration and return-migration waves, Sweden experienced a period of labor immigration in the 1950s and 1960s to satisfy a labor shortage in the manufacturing and industrial sectors. Changes in the immigration law, such as active labor recruitment in the workers' home countries and the establishment of the Common Nordic Labor Market, allowed large numbers of primarily manual laborers to enter Sweden. The first waves of immigrants came from the other Nordic countries and from Southern and Southeastern Europe (Bengtsson et al 2005).

Decreasing needs for foreign labor and resentment about the high numbers of foreign workers by the country's strong labor unions forced the government to stop foreign labor recruitment rather abruptly at the end of the 1960s, resulting in a sudden stop of the migration inflow. In the following decades, family reunification and the intake of refugees were the main reasons for immigration to Sweden. Worldwide political struggles also changed the face of migration in Sweden. More recently, the greatest sources of migrants to Sweden have been former Yugoslavia, Chile, Iran, Iraq and African countries. By 2009, 14 percent of the individuals living in Sweden were born abroad, and one-quarter had at least one parent who was born outside Sweden (Statistics Sweden 2011).

Given the heterogeneity of Sweden's foreign-born population, intermarriage varies substantially among different immigrant groups. This variation has a potential influence on the health effects of intermarriage. Therefore, the following section discusses several known factors that influence immigrant groups' intermarriage rates and their potential health influences.

## Intermarriage and its impact factors

The majority of studies examining intermarriage concentrate on the factors that support or hamper intermarriage. The fact that intermarriage's potential effects on health outcomes could be driven by group and individual behavior, which influenced the decision to intermarry in the first place, makes it necessary for this study to consider several of the socioeconomic factors related to intermarriage.

The intermarriage impact factors can be categorized roughly into group factors and individual factors. The group factors illustrate the importance of considering cultural and ethnic influences regarding marriage decisions. *Assimilation theory* states that an individual of a certain immigrant background will be more likely to marry a native if his/her immigrant group is more integrated into the native society (Dribe and Lundh 2008; Kalmijn and van Tubergen 2006). Integration can mean both social and economic participation and shared beliefs and values. Well-integrated immigrants could therefore exhibit dietary preferences or health behaviors, such as smoking habits or the degree of physical exercise, which are similar to those of the native population. More importantly, well-integrated immigrants are better able to take advantage of health services and preventive methods because of their closeness to the native culture, including the possession of country-specific skills, such as language skills.

The degree of integration, and therefore the prevalence of intermarriage, depends on several aspects of the cultural/ethnic background and can vary substantially among groups of immigrants. Immigrants from culturally and geographically distant places often have lower levels of integration and therefore lower rates of intermarriage (Coleman 1994). The tendency to engage in partnerships outside the immigrant's own group is determined not only by the individual's cultural background but also by his/her family and ethnic community (Huschek 2011). Further, studies have found health differences between natives and various groups of immigrants (Dassanayake et al 2011; Gadd et al 2003). The health differences between intermarried immigrants and natives may be less notable than the differences between immigrants and natives in general.

A strong predictor of intermarriage is size of the ethnic group within the host country (Chiswick and Houseworth 2011). While individuals from large immigrant groups have statistically more opportunities to find a matching partner of their own ethnic background (Cretser 1999), people from smaller immigrant groups might struggle to find a partner among their countrymen. Furthermore, individuals from small ethnic groups have more exposure to the host society because they lack access to a large ethnic community. When ethnic similarity is not the most important aspect of partner choice, individuals from smaller ethnic groups can include natives among their marriage choices. The importance of immigrant group size to immigrants' partner choice increases when the sex ratio of immigrants within a group is unbalanced (Kalmijn and van Tubergen 2006) because of differences in the immigration patterns of men and women.

Because the preference for a partner from the same origin who shares similar values and traditions can be the primary factor in partner choice, certain immigrant groups prefer to import a spouse from their country of origin (González-Ferrer 2006). Members of the native population may also import spouses. The high

level of international travel and communication in modern developed countries has dramatically increased the opportunity to meet individuals from other countries. In certain cases, natives might meet a suitable partner abroad and choose to bring him/her to the home country. In other cases, a suitable partner may be found through matchmaking agencies (for example, mail-order brides see as well Niedomysl, Osth and van Ham 2010). The import of spouses can have health consequences for both the imported spouse and the importing partner. In both cases, it is highly probable that the imported partner is using the partner in the host country – foreign or native – as the main source of information about country-specific health issues. Therefore, marriage-immigrants are, to a certain extent, dependent on their partners' skills and knowledge in the host country, which could make a difference when a medical intervention is necessary.

An immigrant group that is well established and sizable in the host country might not depend on contact with the native society and is therefore *self-sustainable* (Blau 1977). Furthermore, the more pronounced in-group focus of such immigrant groups could raise the native population's suspicion of and aversion toward individuals of this immigrant group, thereby increasing the barriers to intermarriage on both sides and consequently reinforcing health differences that emerge from cultural differences in health behaviors.

In cases in which the ethnic community's social control is notably strong, an individual's decision to choose a partner against the will of his/her family or ethnic community could have social consequences for the individual (Lucassen and Laarman 2009). While an immigrant in a partnership with a native could theoretically benefit from his/her partner's country-specific skills and knowledge, the stress resulting from the break with the ethnic community or family can increase the risk for IHD and other negative health outcomes.

The most important country-specific skill and knowledge is the acquisition of native language skills because they enable the immigrants to communicate with the host population and facilitate administrative affairs. Learning a new language is a challenge, and it is more difficult for immigrants whose native language does not use the same alphabet, phonetics or grammatical structure as the language in the host country (Dribe and Lundh 2011). Having a native partner could facilitate communication between physicians and patients and mediate discussions about health problems and their treatment or prevention when the individual has not acquired skills in the host country's language. A native partner could also motivate and support his/her immigrant partner's efforts to learn the native language to independently communicate health issues.

However, language is not the only kind of human capital correlated with intermarriage and health. There seems to be a correlation between higher education and the propensity for intermarriage (Furtado 2012). Two factors could explain

this effect. On the one hand, the more international work environments of highly educated people and individuals in higher occupational positions increases the probability of meeting suitable partners with origins different from their own (Niedomysl et al 2010). On the other hand, highly educated immigrants have better opportunities on the marriage market in general. People tend to build partnerships with people who resemble them in cultural, economic and ethnic terms (Behtoui 2010; Kalmijn 1998). Education is also correlated with several health outcomes (Smith et al 1998), including heart disease (Winkleby et al 1992). The effect of higher education could have a direct effect on the risk for IHD and an indirect effect caused by the selection of highly educated immigrants into intermarriage.

Connected to the selection of highly educated immigrants into intermarriage is the *social exchange theory*. According to the theory, it is partners' differences, rather than their similarities, that promote intermarriage. An economically successful immigrant who marries a native trades his/her economic power for the native partner's country-specific social and human capital (Qian and Lichter 2007). A health effect for both partners can be assumed because an increase in the native partner's SES and better access to the host country society for the immigrant partner can be correlated with better health outcomes.

## The effect of intermarriage on health

In summary, all of the theories and assumptions mentioned above explain why intermarriage occurs more often in some groups of immigrants and is less common among others. This study focuses on the consequences of intermarriage, rather than its origins. Other studies have already found a beneficial effect on the economic performance of immigrants married to a native (Meng and Gregory 2005; Meng and Meurs 2009). However, to the best of the author's knowledge, no studies have addressed the consequences of intermarriage on individual health outcomes. Consequently, the aim of this study is to investigate the potential effects of intermarriage on the individual risk for IHD for both the foreign and the native partners in intermarriages.

The potential health effect of intermarriage could follow two different mechanisms. On the one hand, intermarriage could accelerate integration and decrease the initial health differences between the foreign and the native partner. While marriage in general has a health-promoting effect resulting from shared financial, social and psychological resources, intermarriage can also provide better access to host-country specific assets. In a stable relationship, the partner is a fundamental source of support when health problems occur. Given that IHD

and its prevention are correlated with medical services and physician counseling, a native partner might be able to provide an immigrant partner with knowledge and access to medical services that a partner with the same foreign background could not provide. Following this line of thought, immigrants who enter a marriage with a native would exhibit health benefits compared with immigrants who are married to a nonnative. Additionally, it may be assumed that immigrants married to a native adapt more quickly to native health behaviors than do those married to other immigrants. Given their daily exposure to native food, behavior and health conditions, immigrants who marry natives could be assumed to adapt faster to native health behavior and health patterns as their time in the relationship increases. Intermarriage could therefore reduce the differences in the prevalence of detrimental health behaviors between immigrants and natives that have been found in other studies (Gadd et al 2005). For example, in Sweden, there are strict nonsmoking policies for such public environments as work places, restaurants and public transportation. A smoker from a country with less strict smoking prohibitions might be discouraged from continuing to smoke given the public pressure.

The assumed health benefit of intermarriage for immigrants could also be the result of a selection process that occurs before the onset of the relationship with a native. As mentioned above, people tend to build partnerships with persons who resemble them in cultural, economic and ethnic terms (Behtoui 2010; Kalmijn 1998). Therefore, immigrants who already have behaviors and human capital levels similar to those of the native population might be more attractive to potential native partners. Along with the fact that well-integrated foreigners have more opportunities to meet natives on a regular basis, their level of adaptation to the host society can be seen as an attractive characteristic in the native marriage market (Chiswick and Houseworth 2011). Following this line of argument, the health effect that is assumed for immigrants who are married to natives is not a result of intermarriage itself. Rather, the health-influencing characteristics would have been present in the immigrant before the partnership began; in fact, they would have influenced the partner choice.

Regardless which of the mechanisms is valid, the argument leads to the assumption that immigrants who intermarry experience a health benefit compared with immigrants who have non-native partners. This study focuses on quantifying the potential health effects of intermarriage for both the foreign and the native partner, as measured in terms of risk for the onset of IHD.

IHD has several important impact factors that are well-studied. Previous studies have confirmed the detrimental effect of overweight and obesity (Beauchamp et al 2010; Brunner et al 1997), hypertension (Hansen et al 2007; Sipahi et al 2006) and diabetes (Fuller et al 2001). These medical conditions are often the

result of behavioral habits, especially dietary preferences and increasing physical inactivity. Over the most recent decades, several studies have confirmed a social gradient among health behavior and, consequently, health outcomes (Baigi et al 2002; Chandola et al 2004; Lynch et al 1996; Marmot 1989). Intermarriage can be assumed to be correlated with many of these IHD impact factors. Because intermarriage is more common among economically successful immigrants, intermarriage itself could be used as an indicator of higher SES among migrants. As previously mentioned, several studies have found differences between foreign-born and native individuals in the prevalence of IHD risk factors. The question emerges concerning what influence intermarriage has, not only directly on the risk for IHD onset, but also on the impact factors triggering IHD. Therefore, a second aim of this study is to investigate the impact of intermarriage on several IHD risk factors.

## 5.4. Data and Method

To achieve the established aims of this study, the Swedish Longitudinal Immigrant (SLI) database was exploited. This database is register-based, combining information from different national sources. Yearly information about financial affairs is provided by the Swedish Tax Register. Information about demographic and socioeconomic characteristics is obtained from censuses from 1970 to 1990. Health-related information is obtained through the Hospital Discharge Register. The Swedish person number, a unique identification code given to all individuals is used to link information from the different sources. The database contains information on more than 500,000 individuals of Swedish and foreign origin. The samples were chosen in a way that identified family relationships between individuals via a coding key. In that way, people connected by close blood relationship (i.e., children or parents) or a registered partnership can be identified.

In Sweden, intermarriage is defined as the registered partnership between two individuals of different origin, measured as the country of birth. Registered partnerships consist mainly of married couples. However, since 1990, the official registers have also included cohabiting couples with common children, even if the couple is not married. This study uses the term "partner" to refer to individuals in both forms of partnerships (married and cohabitating with common children). Therefore, the first step of sampling was to identify all of the individuals who have a partner registered in the SLI database. Both individuals' countries of birth

had to be provided for the couple to be included in the study's sample. This approach left the sample with exactly 213,110 unique individuals (105,548 men and 107,480 women) with multiple observations. The number of men and women is not identical because individuals represented in the data may have exited one relationship and entered another during the observation time.

Given the low incidence rate of IHD among younger people, observations of individuals below the age of 40 were not considered for the sample; 87,284 individuals who did not reach age 40 during the observation period were excluded. Because of missing information on the various covariates, another 43,257 individuals were removed from the analysis. The final sample analyzed in this study contains 43,818 men and 38,751 women.

The dependent variable IHD was measured as dichotomous variable, indicating whether a person had had an IHD event in a given year. This information was obtained from the Hospital Discharge Register, which classified it according to ICD 9 and ICD 10 codes. The Hospital Discharge Register entries capture every medical incidence that results in admission to the hospital for at least one day. Given the disease's severity and its extensive need for medical intervention, no significant underrepresentation of cases is assumed. Even in the event that a person experienced a fatal IHD episode outside the hospital, the event and cause of death would be registered at the nearest hospital. Therefore, the data regarding IHD cases among the population in Sweden can be assumed to be close to complete.

However, the precise measurement of IHD onset encounters some obstacles using the SLI database. Information about the occurrence of IHD events is not available from before 1987 when the Hospital Discharge Register was launched. As a consequence, the observed IHD cases are not necessarily the first for the individual in question. This presents a major problem for the analysis of the impact of intermarriage on the onset of IHD.

IHD has a degenerative nature; consequently, individuals who have experienced and survived an initial IHD event are exposed to a much higher risk of experiencing another IHD event. Over 50 percent of the individuals represented in the SLI database who experience one IHD event sufferer from another IHD event during the observation period of the database. Given the high standard of medical intervention for cardiovascular diseases (Hedlund et al 2008), this degenerative effect could add bias to the analysis. Not taking an individual's former IHD history into account could overestimate the risk for IHD for individuals who have already experienced an event.

Instead of using IHD events of any order, this study focused on the onset of IHD and therefore only considers the first case of IHD for every individual. This approach excludes the feedback effects of previous IHD history. Because medical

information was only available from 1987 onwards, this study uses a proxy for experiencing the first IHD case. Pre-analysis of the SLI database has shown that the mean time span between two IHD events experienced by the same person is 1.03 years. In total, 95 percent of all repeated IHD events appear less than five years after the first observed event. By only including individuals who have gone five consecutive years without experiencing an IHD event, this study increases the degree of certainty that the observed IHD events were the first for the specific individual.

Starting in 1987, the first year for which medical data were recorded, individuals had to pass the next five years without experiencing an IHD event to be included in this analysis. The individuals who experienced an IHD event during those five years were censored from the sample because there was no way to verify that the observed IHD event was the first for that individual. Consequently, the observation period of this study starts in 1992 and lasts until 2001 when the data available for this study ends. Individuals are censored from the sample after they experienced an IHD case. During the observation time, 5.6 percent of the observed men and 2.5 percent of the women experienced a first IHD event.

To measure intermarriage, this study distinguishes between endogamy and two forms of exogamy. Endogamy exists in a marriage between individuals with the same characteristics; in the case of this study, the characteristic in question is country of origin, that is, Swedes married to another Swede and immigrants married to another immigrant of the same origin. The first type of exogamy is the partnership between a foreigner and a native. The third option is marriage between two immigrants from two different countries of origin. Throughout the analyses, the endogamous relationship group is used as the reference group because endogamy appears to be the most frequent form of partnership in this sample (Table 3).

As the history of migration in Sweden has shown, immigrants living in modern Sweden have notably different backgrounds. Because the pattern of IHD risk varies for immigrants of various origins (Gadd et al 2003), this study also analyzes the country-specific effects of intermarriage. The size of different ethnic minority populations suggested the need to aggregate some countries of origin to collect enough first IHD cases in every group to allow statistical analysis.

Being culturally and historically close to Sweden, Denmark and Norway have been combined into one group (Huijts et al 2010). Historically, the greatest percentage of immigrants in Sweden comes from Finland. Although it is a member state of the Common Nordic Labor Market and it historically shares many characteristics with the other Nordic countries, the Finnish population shows notably different health behaviors compared with Swedes (Tomson and Åberg 1994). Immigrants from Finland who migrate to Sweden have better health

outcomes in many aspects than do their countrymen who stay in Finland, but Finnish immigrants still have worse health outcomes than Swedes do (Hedlund et al 2007). Therefore, Finland was represented in its own group in this analysis.

The majority of immigrants from Italy and Germany came as labor migrants between 1955 and 1970. Consequently, individuals from both countries share many characteristics, and they were merged into a common group for this analysis. Polish immigrants form a separate group. A large number of people arrived after 1992 from the states of the territory of former Yugoslavia. Very few of these immigrants have been re-coded as immigrants from specific countries, and for the majority only *Yugoslavia* is provided as their country of birth. Therefore, because it is impossible to analyze the affected states separately, all individuals from former Yugoslavia were included in a single group in this analysis, even though health patterns might vary among them.

The last group of immigrants includes people from Chile, Turkey, Iran and Iraq. Although immigrants from those countries are very different in their socioeconomic and cultural backgrounds, they all belong to the group of non-European immigrants. These immigrants are expected to have the highest cultural distance from Swedish society.

Individuals who did not belong to one of the aforementioned countries were not considered for this study. The number of individuals and IHD events in this group did not permit appropriate statistical analysis, and their cultural and historical backgrounds did not support useful aggregation. Table 1 provides an overview of the distribution of countries of origin used in this analysis, their grouping and the percentage of overall IHD events experienced in each group.

**Table 1:** Distribution of observations and individuals by country of origin and the corresponding number of IHD cases, stratified by the sex of the individual

| Origin of individual | Men individuals number | | in % | IHD incidence | Women individuals number | | in % | IHD incidence |
|---|---|---|---|---|---|---|---|---|
| Sweden | 19,437 | | 44.36 | 4.84 | 16,848 | | 43.48 | 2.23 |
| All foreign-born | 24,381 | | 55.64 | 6.16 | 21,903 | | 56.52 | 2.72 |
| **Foreign-born by country** | | | | | | | | |
| Finland | 2,785 | | 6.39 | 8.21 | 3,298 | | 8.53 | 3.93 |
| Poland | 2,136 | | 4.88 | 6.45 | 2,979 | | 7.69 | 2.45 |
| Former Yugoslavia | 3,862 | | 8.82 | 4.79 | 3,225 | | 8.33 | 1.86 |
| Norway | 1,494 | > | 7.60 | 8.13 | 1,603 | > | 8.33 | 3.38 |
| Denmark | 1,827 | | | | 1,616 | | | |
| Italy | 940 | > | 5.66 | 6.50 | 471 | > | 4.60 | 2.64 |
| Germany | 1,531 | | | | 1,307 | | | |
| Turkey | 3,511 | > | 22.29 | 5.29 | 2,726 | > | 19.05 | 2.38 |
| Chile | 1,873 | | | | 1,631 | | | |
| Iraq | 1,967 | | | | 1,245 | | | |
| Iran | 2,407 | | | | 1,774 | | | |
| Total | 43,818 | | | 5.57 | 38,751 | | | 2.51 |

Data were taken from SLI database, calculations are the authors

Because the sample was selected from the SLI database, it includes more foreign-born individuals than Swedes for both sexes. The size of the different immigrant groups varies considerably, as does the number of IHD cases observed in each group. In addition to the six groups for country of origin, this study includes another characteristic of the migration background. Based on Swedish migration history, all of the immigrants were categorized into three groups indicating the time of arrival in Sweden. These groups mark the three major migration waves (before 1955; between 1955 and 1970; after 1970) and therefore are used as a proxy for the unobserved reason for migration.

The full model includes a set of covariates that independently affect the risk for IHD onset. Because IHD is a degenerative disease, it is necessary to control for age because higher age will increase the risk for IHD independent of other individual characteristics. The squared age is included to control for possible nonlinear effects of age.

Education, as an indicator of SES, has been shown to predict cardiovascular disease events. In general, a direct positive effect of higher education on health outcomes is assumed (Volkers et al 2007; Winkleby et al 1992). In this study, education is used as categorical variable with the values primary, secondary

and university level, using primary level as reference. Another SES indicator, individual income, is included in the model. The income measurement contains pretax inflation-adjusted logged income. To achieve a more stable and long-term measure of financial resources, this study uses the average income of the five years prior the year at risk. Following earlier research, the assumption is that higher income as an expression of economic and social success is correlated with a lower risk for the onset of IHD.

The SLI database does not contain information about health behavior. Consequently, typical IHD risk factors, such as smoking, low physical activity and overweight, could not be taken into account. These health behaviors often have other consequences, such as diabetes and hypertension, which are also risk factors for IHD. To the extent that these diseases are diagnosed, their prevalence is registered in the Hospital Discharge Register. The prevalence of both diseases are included in the model to control for any possible direct effect on IHD and indirect effects of unobserved health behaviors, such as smoking and alcohol over-consumption.

A variable measuring the time since the onset of the partnership was included because the beneficial effect of a partnership is assumed to increase as the relationship continues. Additionally, all of the models contain year dummies to take possible health trends during the observation period into account. The year dummies also take into account exogenous factors specific to each year.

Studies have shown different IHD patterns for men and women. In total, the same proportion of men and women die of heart disease. However, because women experience diseases of the cardiovascular system at later ages on average than men do, age-specific incidence rates can vary substantially between men and women, putting men at much higher risk in a given age range (WHO 2011a). The different age-specific incidence rates for men and women may be triggered by differences in the endocrine system (Kajantie and Phillips 2006).

Further, it is also assumed that men and women experience different rates of intermarriage for religious reasons (e.g., continuation of their religion for future generations via the husband's religiosity) and because of the different distribution of educational and economic characteristics between men and women (Nikiforov and Mamaev 1998), which independently influence the probability of intermarriage. The need to take all of these sex-specific effects into account makes it essential to use sex-stratified models when analyzing the effect of intermarriage on the risk for IHD onset.

All covariates and their distribution stratified by sex and origin are displayed in Table 2 below. The variables age, length of relationship and the distribution of cases over the observation period were notably similar among all groups.

**Table 2**: Descriptive statistics for all included covariates, stratified by sex and country of origin of the individual

| Covariates | Foreign Men | Foreign Women | Swedish Men | Swedish Women |
|---|---|---|---|---|
| Income (mean over 5 prior years) | 10.36 | 9.95 | 11.74 | 11.18 |
| Education (%) | | | | |
| primary | 42.19 | 48.84 | 33.37 | 30.09 |
| secondary | 22.09 | 22.82 | 25.44 | 34.70 |
| university | 35.72 | 28.34 | 41.19 | 35.21 |
| Hypertension (%) | 2.44 | 2.04 | 2.30 | 1.67 |
| Diabetes (%) | 2.18 | 1.78 | 1.79 | 1.21 |
| Age (mean) | 53.87 | 52.44 | 53.09 | 52.04 |
| Immigration phase (%) | | | | |
| before1955 | 3.38 | 2.87 | | |
| 1955-1970 | 28.47 | 25.06 | | |
| after1970 | 68.14 | 72.06 | | |
| Years of relationship (mean) | 15.26 | 16.02 | 14.51 | 15.95 |
| Years of relationship, squared (mean) | 326.93 | 342.41 | 274.52 | 316.92 |
| Period | | | | |
| 1992 | 8.39 | 8.09 | 8.94 | 8.53 |
| 1993 | 8.82 | 8.56 | 9.16 | 8.90 |
| 1994 | 9.23 | 9.09 | 9.34 | 9.16 |
| 1995 | 9.56 | 9.52 | 9.58 | 9.47 |
| 1996 | 9.87 | 9.89 | 9.81 | 9.80 |
| 1997 | 10.19 | 10.31 | 10.11 | 10.15 |
| 1998 | 10.48 | 10.60 | 10.37 | 10.48 |
| 1999 | 10.89 | 11.00 | 10.70 | 10.89 |
| 2000 | 11.16 | 11.33 | 10.97 | 11.22 |
| 2001 | 11.40 | 11.61 | 11.01 | 11.38 |

Data were taken from SLI database, calculations are the authors

A closer look at the income and education variables reveals differences between the sexes and between natives and foreign-born individuals. Both native and foreign-born women on average earn less than their male counterparts. Furthermore, there is a wage gap between natives and the foreign-born that is even stronger than the difference between the sexes. These income differences may have several explanations, one of which is the diverse distribution of education. Foreign men and women are more concentrated in the primary education group (42 percent of

the men and 49 percent of the women) than natives. While only approximately 20 percent of foreign men and women reported having a secondary degree, 36 percent (men) and 28 percent (women) achieved a university degree. For the native group, the education distribution was more balanced; however, the highest percentage of native men and women had a university degree.

Given the structure of the SLI database, this study analyzes health differences originating from intermarriage patterns using logistic regression models. Including the duration variables of age and the squared age, as well as period dummies, the logistic regression estimates are notably close to estimates of an equivalent survival analysis. In a further step, this study analyzes the possible indirect effects of intermarriage. A standard OLS regression verifies whether intermarriage is correlated with several IHD risk factors, namely income, education, hypertension and diabetes. To illustrate the direct and indirect effect mechanisms of intermarriage, a path analysis was performed. The path analysis uses structural equations to separate the direct and indirect pathways of influence in the defined model. Standard OLS regressions measure the direct and indirect effects, which are reported as standardized regression coefficients. The statistical software package STATA 12 was used to perform the various steps of the analyses.

## 5.5. Results

Below, Table 3 illustrates the distribution of endogamy and the two types of exogamy in the sample, presented as the percentage among the different countries. Because the sample construction allowed the recording of several relationships during the observation period for the same individual, the sum of all relationships for each country of origin exceeds 100 percent by small amounts.

**Table 3**: Distribution in percentage of exogamy and endogamy by origin of the partnerships included in the sample, stratified by sex

| | Men | | | Women | | |
|---|---|---|---|---|---|---|
| | Origin of spouse in % | | | Origin of spouse in % | | |
| Origin of individual | Same | Swedish | Different | Same | Swedish | Different |
| Sweden | 73.17 | | 26.83 | 76.84 | | 23.16 |
| All foreign-born | 74.78 | 18.81 | 6.41 | 70.11 | 23.89 | 6.00 |
| **Country groups** | | | | | | |
| Norway / Denmark | 45.55 | 44.53 | 9.92 | 45.70 | 47.98 | 6.31 |
| Finland | 73.72 | 20.73 | 5.55 | 59.94 | 31.30 | 8.77 |
| Italy / Germany | 31.68 | 56.29 | 12.02 | 40.00 | 50.36 | 9.64 |
| Poland | 87.36 | 10.07 | 2.57 | 52.30 | 38.56 | 9.14 |
| Former Yugoslavia | 83.48 | 7.25 | 9.27 | 84.74 | 7.52 | 7.74 |
| Chile / Turkey / Iran / Iraq | 89.85 | 6.41 | 3.75 | 93.51 | 4.79 | 1.70 |
| Total | 55.87 | 32.44 | 11.69 | 54.74 | 35.17 | 10.09 |

Data were taken from SLI database, calculations are the authors

One apparent finding from Table 3 is that endogamy was the main type of partnership in this sample. Approximately three-quarters of the Swedish males were in a partnership with another Swede. In the case of Swedish women, the proportion was even higher. For the combined group of all foreign-born participants, the same picture emerges. The share of foreign men had partners from their own country of origin in 75 percent of the cases and the foreign women in 70 percent of the cases. The rest of the foreign-born participants were mainly in partnerships with Swedish people. Only a small share was married or cohabiting with a foreigner from a country of origin different from their own.

While endogamy is widespread among the overall group of non-Swedes, Table 3 also shows that the distribution of endogamy and exogamy varies substantially between the groups of immigrants. In the group of Norwegian and Danish people, approximately 46 percent were in endogamous partnerships, and a similar proportion had exogamous partnerships with natives. In the group of Italian and German immigrants, the number of exogamous relationships exceeded the number of endogamous partnerships for both men and women.

Below, Table 4 displays the results of an empty model containing only the variable of interest (intermarriage) and age and year dummies. The estimates for the latter two covariates are displayed in the appendix (Table 4A). The model is stratified by the sex and origin of the individual. The endogamous partnerships are used as the reference category. For the group of natives, there was no statistical difference in the onset of IHD, regardless of whether they were married to other natives or to a foreigner. For the foreign-born men and women, there were

observable differences. Foreign men in a partnership with a native had an 18 percent lower risk for IHD compared with foreign men in partnerships with individuals from the same country of origin. The estimate for foreign women was notably similar; however, it was only significantly different from the reference category at the 90 percent confidence level, while the estimates for foreign men were highly significant (99 percent CI). Foreign men and women in partnerships with foreigners from a different origin did not show any significantly different risk for the onset of IHD. The lack of significance for women may have resulted from the small number of IHD cases among the groups, especially for Swedish women.

**Table 4**: Logistic regression of IHD onset by intermarriage status, stratified by sex and country of origin of the individual

| **Model 1** | **Foreign Men OR (95% CI)** | **Foreign Women OR (95% CI)** | **Swedish Men OR (95% CI)** | **Swedish Women OR (95% CI)** |
|---|---|---|---|---|
| Spouse origin | | | | |
| Same | ref. | ref. | ref. | ref. |
| Swedish | 0.822*** | 0.838* | | |
| | (0.716 - 0.945) | (0.684 - 1.025) | | |
| Different | 0.942 | 1.056 | 0.933 | 0.896 |
| | (0.763 - 1.162) | (0.754 - 1.480) | (0.806 - 1.081) | (0.701 - 1.146) |
| Number of individuals | 24,381 | 21,903 | 19,437 | 16,848 |
| IHD cases | 1,502 | 595 | 940 | 376 |
| Log Likelihood | -8,132 | -3,605 | -5,144 | -2,302 |
| Degrees of freedom | 13 | 13 | 12 | 12 |
| Wald chi2 | 665 | 435 | 824 | 435 |

*** p<0.01, ** p<0.05, * p<0.1

models include age, age squared and year dummies

Table 5 presents the results of the logistic regression measuring the association between intermarriage and the risk for IHD onset, including the aforementioned covariates. The significant health benefit for foreign-born men persists notwithstanding the inclusion of typical IHD risk factors. The estimate is rather robust compared to Model 1, which did not include the covariates (Table 4). Foreign-born women still showed lower IHD risks compared with Model 1, but the difference from the reference category was smaller and became insignificant once the covariates were introduced.

In Model 1, the estimates for natives in partnerships with foreigners indicated IHD risks that were lower, though not significantly, than those in endogamous partnerships. Considering the IHD risk factors, Model 2 shows that natives in

partnerships with foreigners have elevated risks for IHD, which are still not significantly different from those of the subjects in endogamous partnerships.

**Table 5**: Logistic regression with random effects measuring the impact of intermarriage on the onset of IHD; full model separated by sex and country of origin of the individual

| **Model 2** | **Foreign Men OR (95% CI)** | **Foreign Women OR (95% CI)** | **Swedish Men OR (95% CI)** | **Swedish Women OR (95% CI)** |
|---|---|---|---|---|
| Spouse origin | | | | |
| Same | ref. | ref. | ref. | ref. |
| Swedish | 0.821** | 0.908 | | |
| | (0.707 - 0.955) | (0.732 - 1.127) | | |
| Different | 0.964 | 1.079 | 1.017 | 1.15 |
| | (0.777 - 1.196) | (0.765 - 1.522) | (0.875 - 1.180) | (0.894 - 1.478) |
| Income | 0.983* | 1.013 | 0.932** | 1.017 |
| | (0.964 - 1.001) | (0.982 - 1.044) | (0.876 - 0.991) | (0.920 - 1.123) |
| Education | | | | |
| primary | ref. | ref. | ref. | ref. |
| secondary | 0.931 | 1.057 | 0.860* | 0.841 |
| | (0.810 - 1.071) | (0.852 - 1.311) | (0.728 - 1.015) | (0.653 - 1.083) |
| university | 0.769*** | 0.827 | 0.653*** | 0.486*** |
| | (0.676 - 0.875) | (0.646 - 1.058) | (0.550 - 0.774) | (0.342 - 0.690) |
| Hypertension | 4.721*** | 7.036*** | 3.850*** | 6.049*** |
| | (4.057 - 5.493) | (5.660 - 8.747) | (3.175 - 4.667) | (4.392 - 8.332) |
| Diabetes | 3.812*** | 3.115*** | 3.209*** | 3.905*** |
| | (3.246 - 4.478) | (2.411 - 4.026) | (2.592 - 3.975) | (2.684 - 5.681) |
| Age | 1.175*** | 1.309*** | 1.339*** | 1.231*** |
| | (1.118 - 1.235) | (1.198 - 1.430) | (1.265 - 1.418) | (1.126 - 1.345) |
| Age (squared) | 0.999*** | 0.998*** | 0.998*** | 0.999*** |
| | (0.999 - 0.999) | (0.998 - 0.999) | (0.998 - 0.999) | (0.998 - 1.000) |
| Immigration phase | | | | |
| before1955 | 0.975 | 1.159 | | |
| | (0.715 - 1.329) | (0.687 - 1.955) | | |
| 1955-1970 | ref. | ref. | | |
| after1970 | 0.865** | 1.016 | | |
| | (0.763 - 0.982) | (0.830 - 1.243) | | |
| Years of relationship | 0.988 | 0.991 | 0.940*** | 0.957* |
| | (0.973 - 1.004) | (0.964 - 1.020) | (0.919 - 0.961) | (0.916 - 1.000) |
| Years of relationship ^2 | 1.000 | 1.000 | 1.002*** | 1.001 |
| | (1.000 - 1.001) | (0.999 - 1.001) | (1.001 - 1.002) | (1.000 - 1.002) |
| Number of individuals | 24,381 | 21,903 | 19,437 | 16,848 |
| IHD cases | 1,502 | 595 | 940 | 376 |
| Log Likelihood | -7,752 | -3,409 | -4,943 | -2,181 |
| Degrees of freedom | 22 | 22 | 19 | 19 |
| Wald chi2 | 1,875 | 1,115 | 1,426 | 558 |

*** p<0.01, ** p<0.05, * p<0.1

models include as well year dummies

As expected, higher ages and the prevalence of hypertension and diabetes exerted a strong influence on the IHD risk, regardless of the individual's sex and country of origin. The exceptionally high odds ratios might be the result of underreporting of the diseases. The SLI database can only register cases of hypertension and diabetes if they have been diagnosed. Because neither disease occurs suddenly, they could remain undetected within a given time period. As consequence, the cases of hypertension and diabetes that are diagnosed and included in the database have a high probability of being more severe. Therefore, their effect on the onset of IHD can be overestimated.

Two other variables showed a strong impact on IHD risk: income and university education. A higher five-year average income was correlated with lower IHD risk but only for men, both Swedish and foreign-born. The results for women did not show such a tendency. Regarding education, all individuals except the foreign women showed significantly lower IHD risk when they hold a university degree; however, the effect was stronger for Swedes than for foreigners.

## The Effect of Intermarriage on IHD Risk Factors

The theoretical background section made a strong argument for several factors that could be affected by intermarriage in addition to the risk for IHD. Intermarriage has a potential impact on typical IHD risk factors and therefore, the full model could cover the direct effects of intermarriage on the risk for IHD.

Consequently, in a second step, this study performs an OLS regression (Table 6) to verify the effects of intermarriage on the 4 outstanding IHD impact factors from the earlier model – income, high education, hypertension and diabetes. The model includes all of the other covariates (age, age squared, immigration period, duration of partnership and year dummies), only the coefficients of the effect of intermarriage are presented below. The full results of all covariates are displayed in the appendix (Table 6a and 6B).

The effect of intermarriage on income (Model 3) was rather strong and statistically significant for all strata. For immigrants, both men and women, income was significantly higher when they were in a partnership with a Swede. The effect was stronger for women than for men. This result is in line with other studies that found a wage benefit for immigrants married to a native partner (Meng and Gregory 2005). The opposite effect was observed for Swedes. Male and female Swedish natives showed significantly lower incomes when they were married to immigrants. These results support the social exchange theory, favoring partnerships between economically successful immigrants and less economically successful natives for the sake of exchanging economic resources and host country-specific social and human capital.

Among those with a high education level, the effect of intermarriage differs. Immigrants have a significantly higher likelihood of having a high education level if they are not married to a person of the same origin, regardless of whether the partner is native or from a different foreign country. The effect is similar for foreign men and women. Unlike for income, the effect is the same for Swedes. Although the effects are smaller, Swedish men and women show higher probabilities of having a university degree if they are married to a foreigner. Those results could be an indication of the validity of the globalization theory of the marriage market (Niedomysl et al 2010). Given the assumption that highly educated people more frequently work in positions that demand international contacts, the opportunity for meeting a suitable partner outside the local marriage market is higher for them compared with persons with lower education.

**Table 6**: OLS model estimates presenting the effect of intermarriage on IHD risk factors (income, higher education, hypertension and diabetes), stratified by sex and country of origin

| | **Foreign Men** Coef. (95% CI) | **Foreign Women** Coef. (95% CI) | **Swedish Men** Coef. (95% CI) | **Swedish Women** Coef. (95% CI) |
|---|---|---|---|---|
| **Model 3: Income** | | | | |
| Same | ref. | ref. | ref. | ref. |
| Swedish | 0.671*** | 1.154*** | | |
| | (0.642 - 0.700) | (1.124 - 1.184) | | |
| Different | 0.532*** | 0.664*** | -0.091*** | -0.191*** |
| | (0.482 - 0.583) | (0.605 - 0.724) | (-0.104 - -0.077) | (-0.212 - -0.170) |
| **Model 4: High Education** | | | | |
| Same | ref. | ref. | ref. | ref. |
| Swedish | 0.133*** | 0.131*** | | |
| | (0.126 - 0.139) | (0.126 - 0.137) | | |
| Different | 0.083*** | 0.122*** | 0.023*** | 0.037*** |
| | (0.073 - 0.093) | (0.112 - 0.132) | (0.017 - 0.029) | (0.030 - 0.043) |
| **Model 5: Hypertension** | | | | |
| Same | ref. | ref. | ref. | ref. |
| Swedish | 0.003** | -0.005*** | | |
| | (0.001 - 0.005) | (-0.006 - -0.003) | | |
| Different | -0.005*** | 0.006*** | 0.003*** | -0.002*** |
| | (-0.008 - -0.002) | (0.003 - 0.010) | (0.001 - 0.005) | (-0.004 - -0.001) |
| **Model 6: Diabetes** | | | | |
| Same | ref. | ref. | ref. | ref. |
| Swedish | -0.005*** | -0.011*** | | |
| | (-0.007 - -0.004) | (-0.012 - -0.009) | | |
| Different | -0.001 | -0.008*** | 0.003*** | -0.002** |
| | (-0.004 - 0.002) | (-0.010 - -0.005) | (0.002 - 0.005) | (-0.003 - -0.000) |
| Number of observations | 158,798 | 143,736 | 137,465 | 117,891 |
| IHD cases | 1,502 | 595 | 940 | 376 |

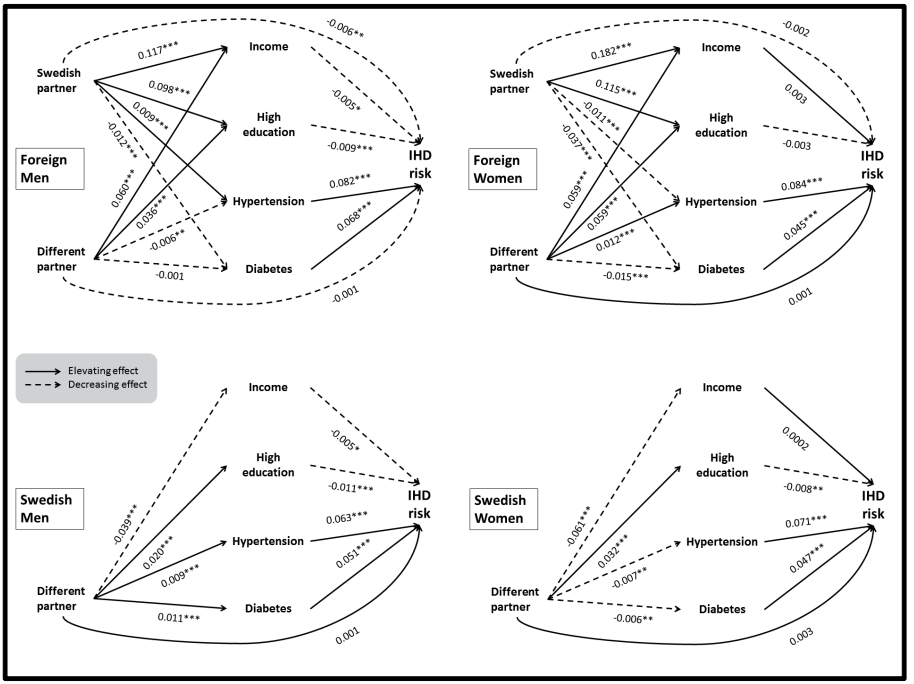
*** p<0.01, ** p<0.05, * p<0.1
All models include age, age squared, immigration period, relationship duration, year dummies

The results for the effect of intermarriage on the prevalence of hypertension and diabetes are less straightforward. Except among foreign men with a higher risk for hypertension, foreigners with Swedish partners had a reduced risk for hypertension and diabetes. Swedish men married to foreigners showed significantly elevated risks of hypertension and diabetes, while the risk for both diseases was reduced among Swedish women with foreign partners. Because both diseases are strongly related to individual health behaviors, a different pattern of merged health behavior between spouses can be assumed depending on the sex and origin of both partners. The low coefficients for hypertension and diabetes can be explained by the potential underreporting of both diseases and the selection of the more severe cases among the afflicted individuals.

## Path Analysis

The results show that intermarriage has a direct influence on several IHD risk factors and on the onset of IHD. The question remains how the different impact pathways work together. To clarify the impact mechanisms, a path analysis was performed. The models included all covariates and the paths for intermarriage, and the four risk factors and their combined effects on IHD are shown in figure 1. Positive correlations are marked with solid arrows, while negative associations are marked with dashed arrows.

**Figure 1**: Path analysis for the effect of intermarriage on IHD directly and indirectly on IHD risk factors, full model, stratified by sex and country of origin. The effects are represented as standardized regression beta coefficients, * p-value <0.1; ** p-value <0.5; *** p-value <0.01




The first glance at the four groups of men and women of foreign and native origin shows that the impact pathways from intermarriage via the four risk factors for IHD differ for the groups. Representing earlier results, the only significant direct effect of intermarriage was found for foreign men married to a native. Taking the mediating effect of the IHD risk factors into account illustrates that some of the indirect effects of intermarriage are amplified, while others are reduced, and their independent effect on IHD originates from the risk factors. For example, being married to a native is correlated with higher income for foreign men, which, in return, decreases the risk for IHD; together, these factors create an amplified effect of intermarriage. However, having a Swedish partner also increases the probability that foreign men will be diagnosed with hypertension, which increases the risk for IHD.

A rather consistent finding is that the effect of intermarriage on the IHD risk factors is significant for men, women, foreigners and Swedes. This result indicates that the effect of intermarriage on IHD is indirect. When IHD risk factors and intermarriage are combined in one model (Model 2), it appears possible that the

indirect effect of intermarriage is covered by the direct effects of IHD risk factors on IHD.

### Country-specific Effects

To date, this study has only analyzed intermarriage effects for the aggregated group of foreign-born individuals; however, country-specific models were also developed (appendix Table 7A and 7B). The first outstanding finding was that none of the results show significant differences between the study groups and the reference group of individuals with endogamous partnerships. The only exception was women from Poland who had partners from a different country of origin. Those women show a significantly elevated risk (OR = 2.01) compared with Polish women who were in partnerships with other Polish individuals. Notwithstanding the lack of significance of the results, which can be assumed to be driven by the small number of individuals and IHD cases in the country-stratified samples, the results varied greatly among the various countries. For example, women from Norway or Denmark with partners from another non-Swedish country exhibited an elevated IHD risk, while women from Finland with partners from a different foreign country show substantially reduced IHD risks. The results also varied between the sexes. Men from former Yugoslavia showed elevated risks when they had a partner from a different foreign country, while women from former Yugoslavia showed the highest risk when they had partners who were also from former Yugoslavia. The small numbers and lack of general significance do not allow conclusive findings for the country-specific model of intermarriage's impact on the risk for the onset of IHD. However, the large variation among the estimations permits the assumption that the country effect does matter. Future studies should investigate this correlation further.

## 5.6. Conclusions

This study analyzes the possible effects of intermarriage on health. The aim was to verify the hypothesis that having a Swedish partner is beneficial for foreign-born immigrants in terms of reducing the risk for IHD onset. A second aim of this study was to investigate potential direct and indirect effects of intermarriage on IHD. Intermarriage could follow direct impact pathways and influence the risk for IHD directly as a result of selective partner choice or by accelerating the

integration process. Indirect effects of intermarriage could include its influence on strong IHD impact factors, such as education or hypertension.

The various models support the hypothesis that intermarriage is correlated with the onset of IHD. The size of this effect depends on an individual's sex and country of origin. The results support the hypothesis that having a native partner is beneficial for reducing the risk for IHD in foreign men. For foreign-born women, the correlation was weaker and not significant.

The analysis of the effect of intermarriage, first on the onset of IHD directly and second mediated by typical IHD risk factors has shown that intermarriage indeed uses direct and indirect pathways. Visualized with help of the path-analysis, intermarriage shows significant effect on IHD risk factors, which, in return, have significant direct effects on the onset of IHD.

In the theoretical background, two possible explanations were presented for the potential effect of intermarriage on health in general and on IHD specifically. On the one hand, a native partner is a source of host country-specific social and human capital for the foreign individual. This additional support could accelerate the integration process and lead to a convergence of the health behaviors of the foreign and native partners. On the other hand, individual characteristics and behaviors could be the reason for the decision to engage in exogamy and could also influence IHD risk. This explanation has two sides. Well-integrated foreigners, who already exhibit health behavior similar to those of natives, might have an advantage on the native marriage market. Foreigners with socioeconomic resources could also trade those assets for natives' host country-specific social and human capital; in return, the native partner would receive economic resources that they lack themselves. The results presented in this study support this social exchange theory. Both foreign men and women exhibited a correlation between intermarriage and higher incomes, while Swedish men and women had significant lower incomes if they were married to a foreigner.

Although the results support the existence of a correlation between intermarriage and the risk for IHD, this study was unable to determine which of the explanations of this correlation was valid. To verify whether selection into intermarriage or the health effects of intermarriage is the underlying mechanism, the exact timeline of events must be taken into account. Although the models in this study included a length of relationship variable, the included information was not adequate for such an analysis. The available data only measured the time since the official recording of the marriage or partnership. A potential health effect of intermarriage can be assumed to emerge long before the marriage date; specifically, it would likely begin at the start of the relationship. Because the length of the relationship before marriage is not reported in the SLI database, the health effects of intermarriage on IHD cannot be verified with certainty.

A similar problem emerges in the analysis of intermarriage and its impact on income. Because the length of a partnership before it is officially recorded is unreported in this database, this study cannot verify whether the positive correlation between higher income among foreigners who have a native partner is a cause or a consequence of intermarriage.

Furthermore, the introduction presents a strong argument for country-specific health patterns. Therefore, the correlation between intermarriage and the onset of IHD was investigated for country groups separately. The small sample sizes and resulting low numbers of IHD cases in the separated country groups did not allow conclusive results. However, the variation in results among the country groups is an indicator that country of origin is an important factor influencing the correlation between intermarriage and health.

This study investigated the effect of intermarriage on the onset of IHD. This health outcome was selected because of the importance of IHD and other cardiovascular disease as a primary cause of death in Sweden and other developed countries. IHD has been shown to be influenced by individual behaviors and life choices, including intermarriage. This study verified a positive correlation between intermarriage among foreign men and natives and a reduced risk for the onset of IHD, as well as effects of intermarriage on various IHD impact factors. This study is the first to analyze the possible health effects of intermarriage. Future studies should investigate the potential health impact of intermarriage on other non-communicable diseases that are behavior-driven.

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# 5.8. Appendix

**Table 4A**: Full estimates for Table 4, effect of intermarriage on IHD onset with only taken age, age squared and year dummies as covariates

| **Model 1**<br>Variables | **Foreign Men**<br>**OR (95% CI)** | **Foreign Women**<br>**OR (95% CI)** | **Swedish Men**<br>**OR (95% CI)** | **Swedish Women**<br>**OR (95% CI)** |
|---|---|---|---|---|
| Swedish partner | 0.822*** | 0.838* | | |
| | (0.716 - 0.945) | (0.684 - 1.025) | | |
| Different partner | 0.942 | 1.056 | 0.933 | 0.896 |
| | (0.763 - 1.162) | (0.754 - 1.480) | (0.806 - 1.081) | (0.701 - 1.146) |
| Age | 1.193*** | 1.299*** | 1.328*** | 1.257*** |
| | (1.137 - 1.251) | (1.192 - 1.415) | (1.255 - 1.404) | (1.153 - 1.370) |
| Age (squared) | 0.999*** | 0.999*** | 0.998*** | 0.999*** |
| | (0.999 - 0.999) | (0.998 - 0.999) | (0.998 - 0.999) | (0.998 - 1.000) |
| Year 1993 | 1.067 | 1.407* | 0.687** | 0.827 |
| | (0.846 - 1.345) | (0.947 - 2.090) | (0.511 - 0.924) | (0.511 - 1.340) |
| Year 1994 | 0.921 | 0.89 | 0.999 | 0.993 |
| | (0.727 - 1.167) | (0.578 - 1.370) | (0.764 - 1.306) | (0.628 - 1.571) |
| Year 1995 | 0.819 | 1.257 | 0.963 | 1.148 |
| | (0.644 - 1.041) | (0.847 - 1.865) | (0.737 - 1.258) | (0.741 - 1.781) |
| Year 1996 | 0.911 | 1.36 | 0.788* | 1.076 |
| | (0.722 - 1.148) | (0.925 - 1.999) | (0.596 - 1.041) | (0.693 - 1.673) |
| Year 1997 | 0.704*** | 1.121 | 0.675*** | 0.661* |
| | (0.552 - 0.899) | (0.755 - 1.664) | (0.507 - 0.900) | (0.405 - 1.080) |
| Year 1998 | 0.883 | 0.97 | 0.665*** | 0.674 |
| | (0.702 - 1.110) | (0.648 - 1.451) | (0.500 - 0.885) | (0.416 - 1.092) |
| Year 1999 | 0.793** | 0.987 | 0.656*** | 1.001 |
| | (0.628 - 1.000) | (0.663 - 1.469) | (0.495 - 0.871) | (0.648 - 1.546) |
| Year 2000 | 0.859 | 0.992 | 0.602*** | 0.616** |
| | (0.686 - 1.077) | (0.669 - 1.471) | (0.452 - 0.801) | (0.380 - 0.998) |
| Year 2001 | 0.778** | 1.036 | 0.611*** | 0.788 |
| | (0.619 - 0.978) | (0.703 - 1.527) | (0.461 - 0.811) | (0.503 - 1.236) |
| Constant | 0.000*** | 0.000*** | 0.000*** | 0.000*** |
| | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) |
| Number of individuals | 24,381 | 21,903 | 19,437 | 16,848 |
| IHD cases | 1,502 | 595 | 940 | 376 |
| Log Likelihood | -8,132 | -3,605 | -5,144 | -2,302 |
| degrees of freedom | 13 | 13 | 12 | 12 |
| chi2 | 665 | 435 | 824 | 435 |

z-statistics in parentheses

*** p<0.01, ** p<0.05, * p<0.1

**Table 6A**: Full estimates for Table 6, effect of intermarriage on IHD risk factors income and high education for foreigner and natives, men and women

| | Model 3: Income | | | | Model 4: High education | | | |
|---|---|---|---|---|---|---|---|---|
| Variables | Foreign men Coef. (95% CI) | Foreign women Coef. (95% CI) | Swedish men Coef. (95% CI) | Swedish women Coef. (95% CI) | Foreign men Coef. (95% CI) | Foreign women Coef. (95% CI) | Swedish men Coef. (95% CI) | Swedish women Coef. (95% CI) |
| Swedish partner | 0.671*** | 1.154*** | | | 0.133*** | 0.131*** | | |
| | (0.642 - 0.700) | (1.124 - 1.184) | | | (0.126 - 0.139) | (0.126 - 0.137) | | |
| Different partner | 0.532*** | 0.664*** | -0.091*** | -0.191*** | 0.083*** | 0.122*** | 0.023*** | 0.037*** |
| | (0.482 - 0.583) | (0.605 - 0.724) | (-0.104 - -0.077) | (-0.212 - -0.170) | (0.073 - 0.093) | (0.112 - 0.132) | (0.017 - 0.029) | (0.030 - 0.043) |
| Age | -0.085*** | -0.132*** | 0.048*** | 0.092*** | 0.041*** | 0.006*** | 0.026*** | 0.005*** |
| | (-0.098 - -0.072) | (-0.147 - -0.116) | (0.044 - 0.053) | (0.086 - 0.099) | (0.039 - 0.043) | (0.004 - 0.008) | (0.024 - 0.028) | (0.003 - 0.007) |
| Age (squared) | 0.000*** | 0.001*** | -0.000*** | -0.001*** | -0.000*** | -0.000*** | -0.000*** | -0.000*** |
| | (0.000 - 0.00) | (0.001 - 0.001) | (-0.001 - -0.000) | (-0.001 - -0.001) | (-0.000 - -0.000) | (-0.000 - -0.000) | (-0.000 - -0.000) | (-0.000 - -0.000) |
| Migrated before 1955 | 0.264*** | -0.133*** | | | -0.069*** | -0.048*** | | |
| | (0.213 - 0.315) | (-0.207 - -0.059) | | | (-0.086 - -0.052) | (-0.063 - -0.033) | | |
| Migrated after 1970 | -1.050*** | -0.940*** | | | 0.075*** | 0.066*** | | |
| | (-1.076 - -1.025) | (-0.968 - -0.912) | | | (0.069 - 0.080) | (0.061 - 0.072) | | |
| Years of relationship | 0.189*** | 0.200*** | 0.019*** | -0.007*** | -0.007*** | -0.006*** | -0.001* | 0 |
| | (0.185 - 0.193) | (0.195 - 0.205) | (0.016 - 0.021) | (-0.011 - -0.004) | (-0.008 - -0.001) | (-0.007 - -0.005) | (-0.002 - 0.000) | (-0.002 - 0.001) |
| Years of relationship (squared) | -0.003*** | -0.003*** | -0.000*** | 0.000*** | 0.000*** | 0.000*** | 0.000*** | 0.000*** |
| | (-0.003 - -0.002) | (-0.003 - -0.002) | (-0.000 - -0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) |
| Year 1993 | -0.035 | 0.007 | 0 | 0.038** | 0.006 | 0.010* | 0.006 | 0.01 |
| | (-0.087 - 0.018) | (-0.058 - 0.072) | (-0.022 - 0.023) | (0.004 - 0.072) | (-0.005 - 0.016) | (-0.001 - 0.020) | (-0.006 - 0.018) | (-0.002 - 0.022) |
| Year 1994 | -0.164*** | -0.073** | -0.002 | 0.056*** | 0.021*** | 0.027*** | 0.014** | 0.020*** |
| | (-0.218 - -0.109) | (-0.138 - -0.008) | (-0.024 - 0.020) | (0.022 - 0.090) | (0.010 - 0.031) | (0.016 - 0.037) | (0.002 - 0.026) | (0.008 - 0.033) |
| Year 1995 | -0.244*** | -0.123*** | -0.0211* | 0.057*** | 0.024*** | 0.035*** | 0.017*** | 0.028*** |
| | (-0.299 - -0.189) | (-0.189 - -0.057) | (-0.044 - 0.001) | (0.023 - 0.091) | (0.014 - 0.035) | (0.025 - 0.046) | (0.005 - 0.028) | (0.016 - 0.040) |
| Year 1996 | -0.336*** | -0.205*** | -0.040*** | 0.055*** | 0.033*** | 0.046*** | 0.023*** | 0.036*** |
| | (-0.392 - -0.280) | (-0.272 - -0.139) | (-0.063 - -0.017) | (0.021 - 0.089) | (0.022 - 0.043) | (0.036 - 0.056) | (0.011 - 0.035) | (0.024 - 0.048) |
| Year 1997 | -0.414*** | -0.292*** | -0.045*** | 0.056*** | 0.041*** | 0.058*** | 0.026*** | 0.047*** |
| | (-0.471 - -0.358) | (-0.359 - -0.226) | (-0.068 - -0.022) | (0.022 - 0.090) | (0.030 - 0.052) | (0.048 - 0.068) | (0.014 - 0.038) | (0.034 - 0.059) |
| Year 1998 | -0.478*** | -0.325*** | -0.035*** | 0.075*** | 0.048*** | 0.068*** | 0.026*** | 0.056*** |
| | (-0.535 - -0.421) | (-0.392 - -0.258) | (-0.059 - -0.012) | (0.041 - 0.109) | (0.037 - 0.058) | (0.058 - 0.078) | (0.015 - 0.038) | (0.044 - 0.068) |
| Year 1999 | -0.480*** | -0.331*** | -0.019 | 0.104*** | 0.052*** | 0.078*** | 0.030*** | 0.062*** |
| | (-0.537 - -0.423) | (-0.398 - -0.264) | (-0.043 - 0.005) | (0.070 - 0.138) | (0.042 - 0.063) | (0.068 - 0.089) | (0.019 - 0.042) | (0.050 - 0.074) |
| Year 2000 | -0.468*** | -0.311*** | 0.008 | 0.139*** | 0.056*** | 0.089*** | 0.033*** | 0.067*** |
| | (-0.525 - -0.411) | (-0.378 - -0.244) | (-0.016 - 0.032) | (0.105 - 0.173) | (0.046 - 0.067) | (0.078 - 0.099) | (0.021 - 0.044) | (0.055 - 0.079) |
| Year 2001 | -0.481*** | -0.306*** | 0.040*** | 0.177*** | 0.059*** | 0.096*** | 0.036*** | 0.074*** |
| | (-0.539 - -0.424) | (-0.373 - -0.240) | (0.016 - 0.064) | (0.143 - 0.212) | (0.049 - 0.070) | (0.086 - 0.106) | (0.025 - 0.048) | (0.062 - 0.086) |
| Constant | 12.94*** | 12.98*** | 10.44*** | 8.867*** | -0.684*** | 0.247*** | -0.162*** | 0.419*** |
| | (12.57 - 13.31) | (12.55 - 13.41) | (10.31 - 10.57) | (8.683 - 9.050) | (-0.744 - -0.623) | (0.184 - 0.310) | (-0.222 - -0.102) | (0.355 - 0.483) |
| Observations | 158,798 | 143,736 | 137,465 | 117,891 | 158,798 | 143,736 | 137,465 | 117,891 |
| R-squared | 0.157 | 0.135 | 0.016 | 0.017 | 0.046 | 0.067 | 0.037 | 0.054 |
| Degrees of freedom | 17 | 17 | 14 | 14 | 17 | 17 | 14 | 14 |
| F-test | 1,401 | 1,094 | 207 | 394 | 608 | 797 | 711 | 1,062 |
| R-squared, adjusted | 0.157 | 0.135 | 0.016 | 0.017 | 0.046 | 0.067 | 0.037 | 0.054 |

Robust t-statistics in parentheses; *** p<0.01, ** p<0.05, * p<0.1

**Continuation of Table 6A**: Full estimates for Table 6, effect of intermarriage on IHD risk factors hypertension and diabetes for foreigner and natives, men and women

| | Model 5: Hypertension | | | | Model 6: Diabetes | | | |
|---|---|---|---|---|---|---|---|---|
| Variables | Foreign men Coef. (95% CI) | Foreign women Coef. (95% CI) | Swedish men Coef. (95% CI) | Swedish women Coef. (95% CI) | Foreign men Coef. (95% CI) | Foreign women Coef. (95% CI) | Swedish men Coef. (95% CI) | Swedish women Coef. (95% CI) |
| Swedish partner | 0.003** | -0.005*** | | | -0.005*** | -0.011*** | | |
| | (0.001 - 0.004) | (-0.006 - -0.003) | | | (-0.007 - -0.004) | (-0.012 - -0.009) | | |
| Different partner | -0.005*** | 0.006*** | 0.003*** | -0.002*** | -0.001 | -0.008*** | 0.003*** | -0.002** |
| | (-0.008 - -0.002) | (0.003 - 0.010) | (0.001 - 0.005) | (-0.004 - -0.001) | (-0.004 - 0.002) | (-0.011 - -0.005) | (0.002 - 0.005) | (-0.003 - -0.000) |
| Age | 0.001** | -0.003*** | 0.001* | -0.001 | -0.002*** | -0.003*** | -0.001** | -0.002*** |
| | (0.000 - 0.002) | (-0.004 - -0.002) | (-0.000 - 0.002) | (-0.002 - 0.000) | (-0.003 - -0.001) | (-0.004 - -0.002) | (-0.002 - -0.000) | (-0.003 - -0.002) |
| Age (squared) | 0.000 | 0.000 | 0.000 | 0 | 0.000 | 0.000 | 0.000 | 0.000 |
| | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) |
| Migrated before 1955 | 0.004 | -0.008** | | | 0.00423 | -0.006** | | |
| | (-0.005 - 0.012) | (-0.015 - -0.001) | | | (-0.002 - 0.011) | (-0.010 - -0.001) | | |
| Migrated after 1970 | -0.005*** | -0.007*** | | | -0.004*** | 0.004*** | | |
| | (-0.007 - -0.003) | (-0.009 - -0.005) | | | (-0.005 - -0.002) | (0.002 - 0.006) | | |
| Years of relationship | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | -0.002*** |
| | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (-0.002 - -0.001) |
| Years of relationship (squared) | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) | (0.000 - 0.000) |
| Year 1993 | 0.003** | 0.002 | 0.002 | 0.001 | 0.001 | 0.001 | 0.001 | 0.001 |
| | (0.000 - 0.006) | (-0.001 - 0.004) | (-0.001 - 0.005) | (-0.002 - 0.004) | (-0.001 - 0.004) | (-0.002 - 0.004) | (-0.002 - 0.004) | (-0.002 - 0.004) |
| Year 1994 | 0.005*** | 0.004*** | 0.004** | 0.002 | 0.004** | 0.002* | 0.002 | 0.002 |
| | (0.002 - 0.008) | (0.001 - 0.008) | (0.001 - 0.007) | (-0.001 - 0.005) | (0.001 - 0.006) | (-0.000 - 0.005) | (-0.001 - 0.005) | (-0.001 - 0.004) |
| Year 1995 | 0.006*** | 0.005*** | 0.007*** | 0.003** | 0.005*** | 0.004*** | 0.003* | 0.002 |
| | (0.003 - 0.009) | (0.002 - 0.008) | (0.003 - 0.010) | (0.001 - 0.006) | (0.002 - 0.008) | (0.001 - 0.007) | (-0.000 - 0.006) | (-0.001 - 0.005) |
| Year 1996 | 0.009*** | 0.007*** | 0.007*** | 0.006*** | 0.007*** | 0.005*** | 0.003** | 0.002* |
| | (0.006 - 0.012) | (0.004 - 0.010) | (0.004 - 0.010) | (0.002 - 0.009) | (0.004 - 0.010) | (0.002 - 0.008) | (0.000 - 0.007) | (-0.000 - 0.005) |
| Year 1997 | 0.010*** | 0.008*** | 0.008*** | 0.007*** | 0.009*** | 0.005*** | 0.004** | 0.002 |
| | (0.007 - 0.014) | (0.005 - 0.011) | (0.005 - 0.012) | (0.004 - 0.010) | (0.006 - 0.012) | (0.003 - 0.008) | (0.001 - 0.007) | (-0.001 - 0.004) |
| Year 1998 | 0.013*** | 0.009*** | 0.010*** | 0.007*** | 0.010*** | 0.006*** | 0.004*** | 0.002 |
| | (0.010 - 0.016) | (0.006 - 0.012) | (0.010 - 0.013) | (0.004 - 0.010) | (0.007 - 0.013) | (0.003 - 0.009) | (0.001 - 0.008) | (-0.001 - 0.005) |
| Year 1999 | 0.015*** | 0.011*** | 0.011*** | 0.008*** | 0.011*** | 0.007*** | 0.005*** | 0.003* |
| | (0.011 - 0.018) | (0.008 - 0.014) | (0.007 - 0.014) | (0.005 - 0.011) | (0.007 - 0.014) | (0.004 - 0.010) | (0.002 - 0.008) | (0.000 - 0.005) |
| Year 2000 | 0.016*** | 0.013*** | 0.013*** | 0.009*** | 0.012*** | 0.007*** | 0.005*** | 0.003** |
| | (0.013 - 0.019) | (0.009 - 0.016) | (0.009 - 0.016) | (0.006 - 0.013) | (0.008 - 0.015) | (0.004 - 0.010) | (0.002 - 0.009) | (0.000 - 0.006) |
| Year 2001 | 0.019*** | 0.015*** | 0.015*** | 0.011*** | 0.013*** | 0.007*** | 0.005*** | 0.003** |
| | (0.015 - 0.022) | (0.012 - 0.018) | (0.012 - 0.019) | (0.008 - 0.014) | (0.010 - 0.016) | (0.004 - 0.010) | (0.002 - 0.008) | (0.000 - 0.006) |
| Constant | -0.057*** | 0.052*** | -0.051*** | -0.005 | 0.037*** | 0.054*** | 0.007 | 0.065*** |
| | (-0.081 - -0.033) | (0.022 - 0.082) | (-0.074 - -0.027) | (-0.031 - 0.022) | (0.010 - 0.063) | (0.026 - 0.082) | (-0.015 - 0.029) | (0.042 - 0.088) |
| Observations | 158,798 | 143,736 | 137,465 | 117,891 | 158,798 | 143,736 | 137,465 | 117,891 |
| R-squared | 0.019 | 0.017 | 0.018 | 0.018 | 0.014 | 0.017 | 0.011 | 0.010 |
| Degrees of freedom | 17 | 17 | 14 | 14 | 17 | 17 | 14 | 14 |
| F-test | 131 | 88 | 116 | 81 | 90 | 79 | 63 | 34 |
| R-squared, adjusted | 0.019 | 0.017 | 0.018 | 0.018 | 0.014 | 0.017 | 0.011 | 0.010 |

Robust t-statistics in parentheses; *** p<0.01, ** p<0.05, * p<0.1

**Table 7A**: Logistic regression on the effect of intermarriage on the onset of IHD, including covariates, stratified by country of origin – men

| **Men** | | | | | | |
|---|---|---|---|---|---|---|
| Variables | Norway/Denmark | Finland | Italy/Germany | Poland | Former Yugslavia | Non-European |
| Swedish partner | 0.888 | 1.01 | 0.901 | 1.284 | 1.343 | 0.763 |
| | (0.660 - 1.195) | (0.633 - 1.612) | (0.610 - 1.331) | (0.642 - 2.568) | (0.650 - 2.772) | (0.467 - 1.245) |
| Different partner | 1.113 | 1.466** | 1.301 | 1.976*** | 2.277*** | 2.059*** |
| | (0.812 - 1.525) | (1.052 - 2.043) | (0.872 - 1.940) | (1.372 - 2.848) | (1.656 - 3.131) | (1.674 - 2.534) |
| Income | 1.083** | 1.086** | 1.151*** | 1.062* | 0.992 | 0.966*** |
| | (1.018 - 1.153) | (1.009 - 1.168) | (1.061 - 1.250) | (0.997 - 1.132) | (0.941 - 1.045) | (0.943 - 0.990) |
| Secundary education | 0.873 | 1.317 | 1.128 | 1.006 | 1.632** | 1.017 |
| | (0.635 - 1.201) | (0.913 - 1.899) | (0.746 - 1.706) | (0.623 - 1.625) | (1.102 - 2.416) | (0.766 - 1.351) |
| University education | 0.735* | 0.625** | 0.999 | 1.15 | 1.854*** | 0.929 |
| | (0.534 - 1.013) | (0.392 - 0.997) | (0.671 - 1.487) | (0.765 - 1.730) | (1.242 - 2.767) | (0.739 - 1.167) |
| Hypertension | 6.228*** | 13.43*** | 8.610*** | 13.06*** | 8.873*** | 9.595*** |
| | (4.425 - 8.768) | (9.173 - 19.66) | (5.311 - 13.96) | (8.119 - 20.99) | (5.624 - 14.00) | (7.171 - 12.84) |
| Diabetes | 10.06*** | 8.311*** | 7.783*** | 6.080*** | 12.01*** | 11.17*** |
| | (6.711 - 15.09) | (5.552 - 12.44) | (4.664 - 12.99) | (3.384 - 10.92) | (7.578 - 19.04) | (8.470 - 14.74) |
| Age | 1.303*** | 1.405*** | 1.332*** | 1.288*** | 1.545*** | 1.520*** |
| | (1.197 - 1.418) | (1.287 - 1.534) | (1.194 - 1.486) | (1.168 - 1.421) | (1.377 - 1.734) | (1.432 - 1.613) |
| Age (squared) | 0.999*** | 0.998*** | 0.999*** | 0.999** | 0.997*** | 0.997*** |
| | (0.998 - 0.999) | (0.997 - 0.999) | (0.998 - 1.000) | (0.998 - 1.000) | (0.996 - 0.998) | (0.997 - 0.998) |
| Migrated before 1955 | 0.341*** | 0.380*** | 0.559** | 0.487 | 2.25E-09 | 0.403 |
| | (0.200 - 0.579) | (0.185 - 0.778) | (0.312 - 1.000) | (0.148 - 1.601) | (0 - ) | (0.114 - 1.419) |
| Mirgrated after 1970 | 1.590*** | 1.748*** | 1.861*** | 2.623*** | 1.396* | 1.576*** |
| | (1.175 - 2.153) | (1.259 - 2.428) | (1.239 - 2.794) | (1.677 - 4.102) | (0.976 - 1.997) | (1.135 - 2.188) |
| Years of relationship | 1.022* | 1.077*** | 1.069*** | 1.015 | 1.027 | 1.033** |
| | (0.996 - 1.049) | (1.042 - 1.114) | (1.031 - 1.108) | (0.971 - 1.060) | (0.980 - 1.077) | (1.002 - 1.066) |
| Years of relationship (squared) | 1 | 0.999* | 0.999* | 1.001 | 1 | 1.001 |
| | (1.000 - 1.001) | (0.998 - 1.000) | (0.999 - 1.000) | (1.000 - 1.002) | (0.999 - 1.001) | (1.000 - 1.002) |
| Year 1993 | 1.463** | 1.549*** | 1.528** | 1.384 | 1.191 | 1.14 |
| | (1.049 - 2.040) | (1.115 - 2.152) | (1.019 - 2.290) | (0.906 - 2.116) | (0.744 - 1.905) | (0.878 - 1.480) |
| Year 1994 | 1.604*** | 0.943 | 1.214 | 1.254 | 1.662** | 0.989 |
| | (1.167 - 2.206) | (0.656 - 1.354) | (0.798 - 1.848) | (0.820 - 1.919) | (1.099 - 2.511) | (0.765 - 1.278) |
| Year 1995 | 1.188 | 0.928 | 0.986 | 1.066 | 1.638** | 0.769* |
| | (0.845 - 1.669) | (0.649 - 1.329) | (0.639 - 1.522) | (0.692 - 1.645) | (1.093 - 2.457) | (0.591 - 1.000) |
| Year 1996 | 1.573*** | 1.076 | 0.956 | 0.791 | 1.456* | 0.904 |
| | (1.153 - 2.147) | (0.767 - 1.509) | (0.624 - 1.463) | (0.501 - 1.247) | (0.972 - 2.180) | (0.709 - 1.152) |
| Year 1997 | 0.872 | 0.75 | 0.985 | 0.483*** | 0.735 | 0.831 |
| | (0.611 - 1.243) | (0.521 - 1.079) | (0.650 - 1.494) | (0.289 - 0.806) | (0.459 - 1.179) | (0.654 - 1.057) |
| Year 1998 | 1.048 | 0.519*** | 0.603** | 0.633* | 1.002 | 0.692*** |
| | (0.745 - 1.476) | (0.351 - 0.769) | (0.378 - 0.962) | (0.399 - 1.005) | (0.663 - 1.514) | (0.542 - 0.884) |
| Year 1999 | 0.718* | 0.584*** | 0.515*** | 0.514*** | 0.74 | 0.621*** |
| | (0.496 - 1.040) | (0.399 - 0.854) | (0.320 - 0.829) | (0.319 - 0.828) | (0.483 - 1.134) | (0.486 - 0.793) |
| Year 2000 | 0.689** | 0.533*** | 0.506*** | 0.593** | 0.524*** | 0.689*** |
| | (0.476 - 0.998) | (0.362 - 0.786) | (0.316 - 0.809) | (0.376 - 0.936) | (0.334 - 0.824) | (0.544 - 0.872) |
| Year 2001 | 0.606*** | 0.345*** | 0.388*** | 0.513*** | 0.498*** | 0.595*** |
| | (0.415 - 0.886) | (0.227 - 0.526) | (0.236 - 0.638) | (0.323 - 0.814) | (0.318 - 0.780) | (0.468 - 0.756) |
| Constant | 0.000*** | 0.000*** | 0.000*** | 0.000*** | 0.000*** | 0.000*** |
| | (0.000 -0.000) | (0.000 -0.000) | (0.000 -0.000) | (0.000 -0.000) | (0.000 -0.000) | (0.000 -0.000) |
| Observations | 80616 | 75397 | 61737 | 46531 | 87415 | 184200 |
| Number of individuals | 5966 | 5478 | 4186 | 3601 | 6400 | 17778 |
| Log Likelihood | -3490 | -3234 | -2142 | -2041 | -2328 | -6599 |
| degrees of freedom | 22 | 22 | 22 | 22 | 22 | 22 |
| chi2 | 850.7 | 864.2 | 540.8 | 530.2 | 588.1 | 1609 |

z-statistics in parentheses

*** p<0.01, ** p<0.05, * p<0.1

**Continuation of Table 7A**: Logistic regression on the effect of intermarriage on the onset of IHD, including covariates, stratified by country of origin – women

| **Women** | | | | | | |
|---|---|---|---|---|---|---|
| Variables | Norway/Denmark | Finland | Italy/Germany | Poland | Former Yugslavia | Non-European |
| Swedish partner | 1.38 | 0.967 | 0.904 | 0.866 | 0.426 | 0.246* |
| | (0.862 - 2.210) | (0.591 - 1.585) | (0.371 - 2.203) | (0.513 - 1.461) | (0.0934 - 1.938) | (0.0569 - 1.061) |
| Different partner | 1.542 | 1.570** | 2.886** | 2.107*** | 3.259*** | 1.774*** |
| | (0.881 - 2.697) | (1.009 - 2.445) | (1.244 - 6.696) | (1.276 - 3.479) | (1.915 - 5.544) | (1.206 - 2.611) |
| Income | 1.145*** | 1.246*** | 1.631*** | 1.041 | 1.018 | 1.004 |
| | (1.033 - 1.268) | (1.114 - 1.394) | (1.194 - 2.229) | (0.959 - 1.131) | (0.942 - 1.100) | (0.965 - 1.045) |
| Secundary education | 1.890** | 1.437 | 1.605 | 0.76 | 0.836 | 0.785 |
| | (1.162 - 3.073) | (0.913 - 2.263) | (0.682 - 3.779) | (0.423 - 1.363) | (0.372 - 1.880) | (0.425 - 1.449) |
| University education | 0.819 | 0.676 | 0.721 | 0.87 | 2.399** | 1.129 |
| | (0.409 - 1.637) | (0.360 - 1.268) | (0.259 - 2.006) | (0.518 - 1.463) | (1.171 - 4.916) | (0.699 - 1.824) |
| Hypertension | 12.41*** | 11.27*** | 7.743*** | 11.37*** | 24.30*** | 12.86*** |
| | (6.755 - 22.81) | (7.059 - 18.01) | (2.414 - 24.84) | (6.097 - 21.19) | (12.66 - 46.65) | (8.429 - 19.63) |
| Diabetes | 10.36*** | 7.982*** | 11.86*** | 6.149*** | 21.75*** | 9.389*** |
| | (4.955 - 21.65) | (4.028 - 15.81) | (4.011 - 35.08) | (2.647 - 14.28) | (10.71 - 44.16) | (6.221 - 14.17) |
| Age | 1.381*** | 1.407*** | 1.159 | 1.288*** | 1.516*** | 1.489*** |
| | (1.172 - 1.626) | (1.246 - 1.590) | (0.917 - 1.465) | (1.112 - 1.493) | (1.242 - 1.850) | (1.332 - 1.666) |
| Age (squared) | 0.999** | 0.998*** | 1 | 0.999* | 0.997*** | 0.998*** |
| | (0.997 - 1.000) | (0.997 - 0.999) | (0.998 - 1.002) | (0.998 - 1.000) | (0.996 - 0.999) | (0.997 - 0.999) |
| Migrated before 1955 | 0.416* | 0.436* | 1.283 | 4.18E-09 | 0 | 3.982 |
| | (0.167 - 1.038) | (0.174 - 1.090) | (0.355 - 4.637) | (0 - ) | (0 - ) | (0.120 - 132.6) |
| Mirgrated after 1970 | 1.421 | 2.002*** | 1.1 | 1.703* | 0.895 | 3.709*** |
| | (0.865 - 2.336) | (1.321 - 3.033) | (0.443 - 2.733) | (0.935 - 3.101) | (0.516 - 1.553) | (1.722 - 7.988) |
| Years of relationship | 1.053** | 1.002 | 1.01 | 1.014 | 1 | 1.065* |
| | (1.000 - 1.109) | (0.956 - 1.049) | (0.927 - 1.100) | (0.937 - 1.098) | (0.916 - 1.092) | (0.994 - 1.141) |
| Years of relationship (squared) | 0.999 | 1.001 | 1 | 1 | 1 | 1 |
| | (0.998 - 1.000) | (1.000 - 1.002) | (0.998 - 1.002) | (0.998 - 1.003) | (0.998 - 1.002) | (0.998 - 1.002) |
| Year 1993 | 2.235*** | 0.96 | 3.085*** | 1.356 | 1.917* | 0.82 |
| | (1.353 - 3.694) | (0.559 - 1.647) | (1.373 - 6.932) | (0.677 - 2.715) | (0.932 - 3.942) | (0.486 - 1.386) |
| Year 1994 | 0.869 | 0.935 | 1.562 | 1.271 | 2.126** | 0.728 |
| | (0.457 - 1.651) | (0.553 - 1.580) | (0.608 - 4.012) | (0.649 - 2.491) | (1.071 - 4.217) | (0.440 - 1.203) |
| Year 1995 | 1.209 | 0.826 | 2.431** | 1.488 | 1.424 | 0.851 |
| | (0.688 - 2.125) | (0.487 - 1.402) | (1.052 - 5.619) | (0.796 - 2.779) | (0.685 - 2.961) | (0.540 - 1.339) |
| Year 1996 | 0.946 | 1.145 | 2.374** | 2.191*** | 1.4 | 0.788 |
| | (0.523 - 1.712) | (0.715 - 1.836) | (1.048 - 5.377) | (1.268 - 3.785) | (0.680 - 2.882) | (0.507 - 1.224) |
| Year 1997 | 0.791 | 0.918 | 1.311 | 0.931 | 1.069 | 0.739 |
| | (0.427 - 1.465) | (0.561 - 1.503) | (0.516 - 3.329) | (0.472 - 1.836) | (0.497 - 2.300) | (0.480 - 1.136) |
| Year 1998 | 0.864 | 0.673 | 1.282 | 1.108 | 0.713 | 0.662* |
| | (0.473 - 1.579) | (0.400 - 1.130) | (0.498 - 3.304) | (0.593 - 2.071) | (0.309 - 1.644) | (0.430 - 1.017) |
| Year 1999 | 0.477** | 0.684 | 0.618 | 0.832 | 0.612 | 0.455*** |
| | (0.238 - 0.953) | (0.412 - 1.137) | (0.202 - 1.893) | (0.430 - 1.612) | (0.262 - 1.432) | (0.288 - 0.719) |
| Year 2000 | 0.652 | 0.628* | 0.805 | 0.681 | 0.692 | 0.595** |
| | (0.350 - 1.214) | (0.376 - 1.048) | (0.289 - 2.243) | (0.343 - 1.351) | (0.316 - 1.515) | (0.388 - 0.914) |
| Year 2001 | 0.64 | 0.361*** | 1.223 | 0.511* | 0.735 | 0.617** |
| | (0.343 - 1.193) | (0.203 - 0.641) | (0.474 - 3.156) | (0.247 - 1.058) | (0.347 - 1.557) | (0.405 - 0.940) |
| Constant | 0.000*** | 0.000*** | 0.000*** | 0.000*** | 0.000*** | 0.000*** |
| | (0.000 -0.000) | (0.000 -0.000) | (0.000 -0.000) | (0.000 -0.000) | (0.000 -0.000) | (0.000 -0.000) |
| Observations | 79263 | 92003 | 47545 | 64165 | 80524 | 162136 |
| Number of individuals | 6102 | 6557 | 3368 | 5434 | 6065 | 16353 |
| Log Likelihood | -1293 | -1673 | -557.4 | -998.1 | -797.8 | -2094 |
| degrees of freedom | 22 | 22 | 22 | 22 | 22 | 22 |
| chi2 | 336.6 | 454.1 | 153.8 | 307.7 | 299.3 | 782.9 |

z-statistics in parentheses

*** p<0.01, ** p<0.05, * p<0.1

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