Socioeconomic position and coronary heart disease in older age: associations and possible pathways

THESIS

presented for the degree of DOCTOR OF PHILOSOPHY

in the Faculty of Biomedical Sciences

Field of Study – Public Health Epidemiology

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April 2009

DECLARATION OF AUTHORSHIP

I, Sheena E. Ramsay, confirm that the work presented in this thesis is my own. I have used data from the British Regional Heart Study, which is an ongoing study on cardiovascular disease that was initiated in 1978.

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Abstract

Low socioeconomic position is known to be associated with greater coronary heart disease (CHD) risk in most developed countries. However, studies have largely focused on the association between socioeconomic position and CHD in middle-aged populations and little is known about the extent to which socioeconomic position affects CHD risk in later life. This thesis uses the British Regional Heart Study, a population-based cohort of British men to investigate the extent of socioeconomic inequalities in CHD in older age and the possible pathways to these inequalities. Issues addressed in detail include trends in socioeconomic inequalities in CHD with increasing age and over time, the extent of socioeconomic inequalities in CHD in older age (60-79 years), the contribution of established and novel coronary risk factors to these inequalities, and the influence of early life socioeconomic position on CHD risk in later life. Although CHD mortality declined over the last two decades in Britain, relative social class differences in CHD did not narrow between 1980 and 2005. With increasing age (from 40-59 years to 65-84 years), relative social class inequalities in CHD narrowed, although absolute differences widened with age. Marked socioeconomic differences in CHD were present in older age; CHD risk increased from the highest to the lowest social class group. Socioeconomic differences in behavioural coronary risk factors (particularly cigarette smoking) could explain at least a third of these inequalities; inflammatory markers made some additional contribution. Lower socioeconomic position in childhood was associated with increased CHD risk in older age; part of this association was due to the relationship of childhood socioeconomic position with adult behavioural factors. Appreciable socioeconomic inequalities were also present in disability among older men with CHD. The results suggest that important socioeconomic inequalities in CHD persist in older age; the implications for public health and further epidemiological research are discussed.

Acknowledgements

I would like to express my sincere gratitude to Dr Goya Wannamethee, Prof Peter Whincup and Dr Richard Morris, for their guidance, valuable teaching, support and encouragement in supervision over the last four years. I am grateful to Prof A. Gerry Shaper for his valuable comments on the thesis. Olia Papacosta has generously provided much support and guidance, for which I am very thankful. I am grateful to Andy Thomson, Lucy Lennon and Mary Thomas for their meticulous upkeep and provision of data, which has made this work possible. I am very grateful to Mary Thomas, Olia Papacosta, Richard Morris and Sarah Hardoon for proof-reading the thesis. Sincere thanks go to my parents, family and friends for their love and prayerful support, and above all to the Lord Jesus Christ for his sustaining grace and love.

The British Regional Heart Study is funded by the British Heart Foundation. I was supported for the duration of the study by a Department of Health grant and subsequently by a Medical Research Council Special Training Fellowship in Health of the Public.

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Chapter 1

Introduction

1.1 Summary

People from lower socioeconomic positions have poorer health than those in higher socioeconomic positions. Coronary heart disease (CHD), a leading cause of death, morbidity and disability, is an important contributor to socioeconomic inequalities in health, being more common in people from lower socioeconomic positions. Although CHD mortality rates have declined since the 1970s in Britain, changes in the extent of socioeconomic inequalities in CHD since that time are not well documented. That lower socioeconomic position is associated with greater CHD risk in middle-age is known, but the extent to which these inequalities persist into later life or older age has not been widely studied. The pathways underlying socioeconomic inequalities in CHD in later life are also not fully understood. Established coronary risk factors as well as more 'novel' coronary risk factors have been postulated to contribute to socioeconomic inequalities in CHD in middle-age; the effect of these coronary risk factors on socioeconomic inequalities in older age, however, is unclear. The influence of early life socioeconomic position on CHD risk in older age is also not well understood. This thesis investigates socioeconomic inequalities in CHD in older age using the British Regional Heart Study, a prospective population-based study of 7735 men followed-up from 1978-80 when aged 40-59 years. In particular, the thesis will investigate: 1) changes in the extent of socioeconomic inequalities in CHD with increasing age from middle-age and over time from the late 1970s; 2) the relationship of social class to coronary risk factors and to CHD in older men; 3) pathways to socioeconomic inequalities in CHD in older men; 4) influence of early life socioeconomic position on CHD risk in older men; and 5) the extent of socioeconomic inequalities in disability in those with CHD in later life. A crucial strength of the British Regional Heart Study to address these research questions is that it comprises a socioeconomically and geographically representative sample of middle-aged British men in 1978-80. Moreover, the study has high rates of follow-up and includes detailed information on socioeconomic conditions in early life, middle-age and later life, and on various coronary risk factors.

1.2 Coronary heart disease: pathophysiology and epidemiology

Coronary or ischaemic heart disease is defined by a joint International Society and Federation of Cardiology and World Health Organization task force as ‘myocardial impairment due to an imbalance between coronary blood flow and myocardial requirements caused by changes in the coronary circulation’.[1] The presentations of coronary heart disease (CHD) usually have their origin in longstanding underlying atherosclerosis of coronary arteries.[2;3] The gradual progression of arterial atherosclerosis starts with the deposition of lipid-laden macrophages (foam cells) to fatty streaks and fibrous plaques, which narrow the lumen and obstruct coronary blood flow causing ischaemia and chronic angina. Arterial plaques may also fissure or rupture inducing haemorrhage and occlusive thrombus formation.[3;4] Coronary thrombi may occlude the artery to block blood supply acutely, leading to infarction of myocardial tissues and resulting in an acute major coronary event such as myocardial infarction or sudden death. The major clinical manifestations of coronary (ischaemic) heart disease include angina pectoris, myocardial infarction (fatal and non-fatal) and sudden ischaemic death.[3;4] Myocardial infarction and angina are both characterised by symptoms of chest pain. Severe chest pain behind the sternum (breast bone), often radiating to the left arm, occurs in myocardial infarction. Angina is also associated with the symptom of chest pain similar to myocardial infarction, but is usually induced by exertion, and recedes with the ceasing of exertion. Angina is referred to as stable if the symptoms are predictable over a period of weeks or months, rather than worsening over time. Unstable angina is more difficult to define and has been described as the first attack of what later proves to be stable angina and angina at rest.[5] Unstable angina is associated with chest pain of a similar duration and intensity as in stable angina, although chest pain in unstable angina occurs at rest.

CHD rates in Britain increased from the beginning of the twentieth century until the 1980s. Although the incidence and mortality rates from CHD have declined in the UK since the 1970s, CHD remains the main cause of death in the UK where it accounts for around 101,000 deaths each year; one in five men and one in six women die from the disease.[6;7] CHD is one of the main forms of cardiovascular disease and CHD rates vary according to risk factors, age, gender and ethnicity at individual levels, and across countries, regions, social strata and time at population levels.[4] The incidence of myocardial infarction is higher in men than in women,[8;9] and CHD mortality rates are three to four times greater in men. The major risk factors for CHD include cigarette smoking, obesity, high blood pressure and raised serum total cholesterol.[10;11] Over the last two to three decades significant research interest has also been generated in ‘novel’ factors that increase the risk of CHD. These novel coronary risk factors include inflammatory and haemostatic markers such as C-reactive protein (CRP),[12;13] metabolic syndrome and insulin resistance.[14;15] The risk of CHD incidence and mortality also increases steeply with age and is the leading cause of death in over 65 year olds.[5;6;8]

1.3 Adult socioeconomic position and CHD

The health of populations is influenced by the society's social, economic and cultural setting, and the physical environment of workplaces and households.[16;17] Differences in the health of individuals according to their socioeconomic circumstances have long been observed.[18] Influences of socioeconomic factors are also clearly seen in the distribution of CHD.[19] In high-income countries including Britain, CHD risk varies by socioeconomic groups such that coronary disease is greater in lower compared with higher socioeconomic groups.[20-22] Socioeconomic position has been measured using indicators such as occupational social class, education and income.[23-25] Lower socioeconomic groups have been reported to have one-and-a-half to two times the risk of CHD of higher socioeconomic groups.[21;23-26] The public health significance of these disparities is reflected in the greater excess CHD risk experienced by lower socioeconomic groups; approximately a third or more of coronary deaths would be prevented if the CHD risk of the least socioeconomically disadvantaged group were experienced by all, and a fifth of CHD events can be attributed to the excess risk experienced by manual compared to non-manual groups.[27;28] Being a leading cause of morbidity and mortality, CHD is a major contributor to health inequalities. CHD is estimated to be the largest contributor to socioeconomic inequalities in mortality in Western countries.[29;30]

1.4 Adult socioeconomic inequalities in CHD in older age

Although widely studied in middle age, socioeconomic inequalities in CHD have been little studied in older age-groups. The dramatic population ageing of societies occurring since the early twentieth century has meant that CHD and associated inequalities are of increasing potential importance in older populations. In more developed countries, 15.3% of the population were aged over 65 years in 2005 compared with 7.9% in 1950, and this figure is projected to increase to 26% by 2050.[31] CHD risk also increases with age and continues into older age (>60 years).[6] Therefore, reducing socioeconomic inequalities in CHD, as well as reducing the overall burden of CHD in the elderly is likely to be important. However, the evidence so far on the extent of socioeconomic inequalities in CHD has largely focused on middle-aged populations. Studies have reported that socioeconomic inequalities in overall mortality persist in older age, with relative inequalities weakening with older age yet absolute differences increasing with age.[32;33] However, less is known about socioeconomic inequalities specifically in CHD in older age. Moreover, with the important social changes and the decline in overall CHD incidence/mortality rates since the 1970s in the United Kingdom,[7] it is also important to monitor the changes in socioeconomic inequalities in CHD over time. According to the Acheson Report, an increase in the relative socioeconomic inequalities in CHD mortality occurred between the early 1970s and the 1990s in Britain, reflecting a greater decline of CHD in higher social class groups.[34] But whether this trend has continued in the early part of the twenty-first century is not known. This raises the question of whether socioeconomic inequalities in CHD have changed over time and with increasing age from middle-age. It will also be important to assess the extent to which socioeconomic inequalities in CHD persist in older age.

1.4.1 Pathways between socioeconomic position and CHD in older age

Although the relation of socioeconomic position to CHD in middle-age is well-established, the pathways linking socioeconomic position to CHD in older age are not fully understood. In studies of middle-aged populations, the starting point for understanding socioeconomic inequalities in CHD has been to investigate the role of established coronary risk factors. However, the 'paradox' commonly observed is that established coronary risk factors do not fully account for socioeconomic inequalities in CHD.[35] While some studies have reported that cigarette smoking, obesity, lack of physical exercise and high blood pressure played a substantial part in contributing to socioeconomic inequalities in CHD,[35-37] others have reported a limited role of these risk factors.[25;38;39] More recently, levels of 'novel' coronary risk factors including inflammatory/haemostatic markers and metabolic syndrome, have also been found to be more prevalent in lower socioeconomic groups.[40-42] This has led to the hypothesis that these novel coronary risk factors could account for the socioeconomic inequalities in CHD which are not fully explained by established coronary factors. However, the role of these novel risk factors in socioeconomic inequalities is not fully established since these markers are also strongly related to established coronary risk factors including smoking and physical activity,[43-45] raising the possibility of confounding. Research on these possible underlying pathways for socioeconomic inequalities in CHD has been largely restricted to middle-aged populations. There is, therefore, a need to explore whether the same associations between socioeconomic position and both established and novel coronary risk factors persist in older age (>60 years), and to examine the extent to which these factors account for socioeconomic inequalities in CHD in older age.

1.4.2 Early life socioeconomic position and CHD risk in later life

A life course approach to chronic disease epidemiology seeks to define the importance of exposures operating early in life and their influence on health and disease risk experienced in adult life.[46] An early example of this approach was taken by Kermack and colleagues who demonstrated cohort differences in mortality between 1841-50 and 1921-30 in Britain, such that each successive generation had a lower mortality rate which lasted throughout adult life.[47] These findings along with Forsdahl's work are fundamental to understanding the importance of early life factors on adult disease. In the mid-1970s Anders Forsdahl demonstrated that in a Norwegian county, CHD mortality rates in people aged 40-69 years were correlated with infant mortality rates of the county during the early years of the same cohorts.[48] In the UK, the work of Professor David Barker and colleagues on the association of prenatal and postnatal growth with increased adult CHD risk provided much of the impetus for the hypothesis of the fetal/developmental origin of CHD.[49-51] Other early life exposures implicated in CHD risk in later life have included maternal nutrition, infant feeding, childhood infections and childhood socioeconomic position.[52] Several individual-level studies have sought to understand the role of childhood socioeconomic position in adult CHD risk.[53;54] Early life socioeconomic position has been assessed in terms of parental occupation, household amenities/conditions and overcrowding. Lower childhood socioeconomic position has been found to be associated with increased CHD risk in adult life.[53-55] This effect of childhood socioeconomic position has also been observed to continue, albeit weakened, when adult risk factors (cigarette smoking, body weight, blood pressure and serum total cholesterol) and adult socioeconomic position were taken into account.[56-58] CHD risk also appears to reflect the accumulation of early and later-life exposures.[46] However, it is not known if the influence of early life socioeconomic position on adult CHD persists into older age (>60 years). A greater understanding of this would provide important information on potential pathways to reducing CHD risk in the elderly. Therefore, it is important to address whether childhood socioeconomic position is associated with CHD in older age.

1.4.3 Socioeconomic inequalities in disability in those with CHD in older age

With the significant recent increase in the proportion of older people, it can be assumed that the burden of disability will increase in the population. Studies in Western populations show that the prevalence of disability (measured as problems in performing 'activities of daily living' including bathing, dressing and using the toilet) is approximately 20% in people aged 70 years and over and 50% in over 85 year olds.[59] Disability is also measured as problems with carrying out 'instrumental activities of daily living' such as cooking meals, carrying out household chores, and shopping.[60] CHD is not only a leading cause of morbidity and mortality in the elderly but is also a major contributor to disability in older people.[61] CHD has been observed to be associated with a 20-40% prevalence of disability and this proportion increases with age (50-70% in >75 year olds).[62-64] CHD has been estimated to cost the UK economy £9.0 billion, of which 21% is due to informal care of those with CHD.[8] As CHD risk increases with age, the risk of disability associated with CHD is also likely to increase in older age groups resulting in a greater burden on health and social care for individuals and for the healthcare system. It is known that a socioeconomic gradient exists in disability with lower social class groups having a greater prevalence of disability.[65;66] However, the extent of socioeconomic inequalities in disability amongst the elderly with CHD is not clear. When researching socioeconomic inequalities in CHD in older people, it will therefore also be important to explore the extent to which inequalities in disability exist in the elderly with CHD.

1.5 Key issues addressed in the thesis and the suitability of the BRHS

This thesis will focus on five important questions that remain to be answered in light of the above discussions including –

i) What has happened to the size of socioeconomic inequalities in total and CHD mortality over 25 years in Britain and do these inequalities increase with age?

ii) What is the relationship between social class and coronary risk factors in older age?

iii) What is the extent of socioeconomic inequalities in CHD in older age and how much do established and novel coronary risk factors contribute to these inequalities?

iv) Is there an association between early life socioeconomic position and CHD in older age?

v) What is the extent of socioeconomic inequalities in disability in the elderly with CHD?

The British Regional Heart Study (BRHS) is a prospective study of cardiovascular disease comprising 7735 middle-aged men drawn from one general practice in each of 24 towns representing all major British geographic regions. The men were initially recruited and examined between 1978 and 1980 when aged 40-59 years. Since recruitment, subjects have been followed-up through postal questionnaires, and through the National Health Service central register and general practice records for mortality and morbidity, with very high rates of follow-up. A re-examination of study participants involving physical examinations and blood measurements was carried out after 20 years of follow-up between 1998 and 2000. Further details of the BRHS have been described in Chapter 3. The original aims of the study were to explain the regional variation in cardiovascular mortality in Great Britain, and to identify risk factors for cardiovascular disease. This thesis will extend these aims by studying socioeconomic variations in CHD among older British men. There are several features of the BRHS that make it suitable for studying the objectives of this thesis. First, the BRHS has information on socioeconomic conditions of the study participants during their childhood, middle-age and older age together with detailed information on coronary risk factor status. Second, the BRHS is a population-based study comprising a socioeconomically and geographically representative sample. This is a crucial strength of the study when investigating socioeconomic differences in CHD making the results largely generalisable to the older British male population. However, the study sample, derived from medium-sized British towns with less mobile populations, mostly comprises white European men with little information on other ethnic groups. A third strength of the study is that regular accurate information on CHD (fatal and non-fatal myocardial infarction) has been obtained and this has enabled the investigation of socioeconomic inequalities in CHD, which is the main outcome of this thesis. Fourth, the BRHS is an observational study – no attempt was made either to influence clinical practice or to influence participants, thus representing 'natural' occurrence of CHD in the population.

1.6 Objectives and structure of the thesis

This thesis presents an epidemiological study with the aims of assessing socioeconomic inequalities in CHD in older British men and of investigating pathways to these inequalities. To address these aims, the five specific objectives of the thesis are:

1. To investigate trends in socioeconomic inequalities (relative and absolute) in CHD and overall mortality with increasing age (from 40-59 years to 65-84 years), and over 25 years from 1978-80 to 2005 in Britain.
2. To examine the relationship of coronary risk factors (established and novel) with social class in older age (60-79 years).
3. To examine the extent to which socioeconomic inequalities persist in older age, and to investigate the contribution (relative and absolute) of coronary risk factors to these inequalities.
4. To investigate the influence of childhood socioeconomic position on CHD risk in older age.
5. To assess the extent of socioeconomic inequalities in disability in those with CHD in older age.

The content of each Chapter is outlined below:

Chapter 1 gives an introduction to the relationship between socioeconomic position and CHD, outlines the importance of understanding socioeconomic inequalities in CHD in older age, and presents the objectives and structure of the thesis.

Chapter 2 presents the epidemiological background to the thesis including a review of socioeconomic inequalities in CHD, a review of measures of socioeconomic position in older age, a review of trends in socioeconomic inequalities over time in Britain, a review of evidence of socioeconomic inequalities in CHD in older age and evidence for the contribution of coronary risk factors to these inequalities, and a review of evidence on the influence of early life socioeconomic position on CHD in older age.

Chapter 3 describes the design and methodology of the British Regional Heart Study with a focus on the aspects of the study specifically related to the thesis.

Chapter 4 is the first of the five results Chapters (4 to 8). Changes in socioeconomic inequalities in CHD mortality and in overall mortality with increasing age from middle-age, and over time from 1978-80 to 2005 are presented.

Chapter 5 examines the relationship of adult social class to coronary risk factors in older age.

Chapter 6 examines the extent of adult socioeconomic inequalities in CHD in older age, and estimates the contribution of established and novel coronary risk factors to these inequalities.

Chapter 7 examines the association between childhood socioeconomic position and CHD risk in older age and investigates if this is independent of adult social class and behavioural coronary risk factors.

Chapter 8 examines the extent of socioeconomic inequalities in disability in those with CHD in older age.

Chapter 9 assimilates the thesis findings to consider their implications for public health and for future epidemiological studies.

The primary outcome studied in this thesis is the development of major CHD including non-fatal myocardial infarction and fatal CHD (angina was not included). As an exception, Chapter 4 presents results for CHD mortality as well as overall mortality over a 25-year follow-up from baseline (1978-80). Chapter 5 uses data from the 20-year re-examination of the subjects to investigate the relationship between coronary risk factors in older age (60-79 years) and social class. To investigate socioeconomic inequalities in older age, Chapter 6 presents results for CHD (non-fatal and fatal myocardial infarction) over a 6-year follow-up period from 1998-2000 when the subjects were aged 60-79 years. The longest-held occupation recorded in middle-age was used as the main measure of socioeconomic position in older age to examine if socioeconomic inequalities persisted later in life. Chapter 7 utilises 12-year follow-up data until 2004 on CHD (non-fatal and fatal myocardial infarction) starting from 1992 when information on childhood socioeconomic position was collected. Chapter 8 investigates the extent of socioeconomic inequalities in disability measured in 2003 (men aged 63-82 years) in elderly men with CHD.

The results Chapters (4 to 8) are presented in a similar format: summary of the chapter; a brief background to the objectives of the chapter (a detailed background to the thesis is reviewed in chapter 2); the objectives of the chapter; methods used in the chapter; results; and, discussion which includes interpretation of the findings and comparison with existing literature. Implications of the findings are not examined in the individual chapters, but are discussed together in Chapter 9. The thesis appendices include: i) a detailed social class distribution of subjects at the twenty-year follow-up by social class measured at baseline; ii) publications, to date, arising from the research presented in this thesis; and iii) BRHS questionnaires relevant to the thesis.

Chapter 2

Literature review

2.1 Summary

Inequalities in health exist according to socioeconomic position of groups within society. In developed countries these inequalities are present in coronary heart disease (CHD), such that people from disadvantaged socioeconomic backgrounds have a greater risk of CHD compared with those who are socioeconomically advantaged. Despite recent declines in CHD mortality and incidence rates in the UK, evidence suggests that socioeconomic inequalities in CHD mortality have probably widened from the 1970s to 1990s. Epidemiological studies exploring socioeconomic inequalities in CHD have mostly focused on middle-aged populations. Few studies have investigated the extent of socioeconomic inequalities in CHD specifically in older age. These studies show that socioeconomic inequalities in CHD, though smaller than in middle-age, are still present in older age. Few studies have investigated the pathways underlying socioeconomic inequalities in CHD in older age. One study showed that established coronary risk factors (smoking, obesity, hypertension) explained a substantial proportion of socioeconomic inequalities in CHD in older age, while another found little effect of established risk factors on socioeconomic inequalities. The results of studies in middle-aged populations suggest that novel coronary risk factors, such as inflammatory markers, may contribute to socioeconomic inequalities in CHD. However, no study has reported the role of novel coronary risk factors in socioeconomic inequalities in older age. In recent years, increasing interest in the importance of early life factors and their interplay with adult risk factors and disease patterns has led to a greater interest in life course epidemiology. While studies report that lower socioeconomic position in childhood is associated with increased CHD risk in middle-age, the influence of early life socioeconomic position on CHD risk in older age has not been established.

2.2 Structure of literature review

This Chapter presents the epidemiological background to the areas studied in this thesis. Section 2.3 starts with a historical perspective of the role of socioeconomic factors on health. Section 2.4 outlines the extent of social inequalities in health. In section 2.5, the approaches to measurement of socioeconomic position are described. Section 2.6 describes the socioeconomic inequalities in CHD in middle-age. Section 2.7 describes the relative and absolute measures for quantifying socioeconomic inequalities. Section 2.8 explores recent trends in socioeconomic inequalities in CHD in the United Kingdom (UK). Section 2.9 reviews the evidence for the presence of socioeconomic inequalities in CHD in older age. Section 2.10 describes the established and novel risk factors for CHD. Section 2.11 considers the possible pathways linking socioeconomic position to CHD in older age, in particular the role of coronary risk factors. In section 2.12 the influence of early life socioeconomic position on CHD risk in older age is reviewed. Section 2.13 examines socioeconomic inequalities in disability in the elderly with CHD. Section 2.14 draws conclusions from the issues arising in this review and outlines the purpose of the thesis.

2.3 Brief historical perspective of the importance of socioeconomic factors in ill-health

Public health is determined by the social, economic, cultural and physical environment of workplaces, households and society in general. Differences in the health of individuals according to their socioeconomic conditions have long been observed. Rudolf Virchow's work on the typhus epidemic in Upper Silesia in 1847-48 was important in highlighting the crucial role of social factors in the distribution and patterns of diseases and mortality.[17] He proposed interventions for better housing and adequate food supply since deprived living conditions predisposed certain social groups to the epidemic. He also argued that economic insecurity and political instability were linked to the social problems that influenced population health. Excess mortality in 1838 in manufacturing towns of England including Leeds and Manchester, reported by the physician, C.T. Thackrah, was partly attributed to environmental influences such as overcrowding, low standard of maternal care, malnutrition and poor housing, all of which were more common in the North of England.[67] In 1839, the average age at death in Bethnal Green, London, was 45 years for "Gentlemen, professional men and their families" compared with 26 years for "tradesmen and their families", and 16 years for "mechanics, servants, labourers, and their families".[18] Improvements in social and economic conditions resulting in better nutrition and sanitary conditions, contributed to the 'epidemiological transition' of disease patterns in Western societies, with a decline in infectious diseases in the nineteenth century and an increase in degenerative diseases (cancer and cardiovascular disease) since the 1940s.[68] This transition also brought with it a change in the social class-CHD association; lower social classes started to manifest greater CHD mortality than higher social classes. In the late twentieth century, the Inequalities in Health report (Black Report) and the Independent Inquiry into

Inequalities in Health (Acheson Report) were key in highlighting the problem of health inequalities in Britain.[34;69] The Black Report, published in 1980, demonstrated that although overall health had improved in Britain since the 1940s, marked inequalities in health were present.[69] People from poorer or lower social classes had much greater levels of mortality and morbidity from chronic diseases than higher social classes, and these inequalities had widened between the 1930s and 1970s. The Report highlighted the importance of social and economic factors such as housing, education, environment, income and work in contributing to the differences in health. The Acheson Report demonstrated a widening of social class differences in CHD mortality between the 1970s and the 1990s.[34]

2.4 Extent of social inequalities in health

Inequalities in health (systematic differences in the health of different groups in the population) have been observed in relation to socioeconomic factors, to gender, to ethnicity/race and to region.[39;70-73] The focus of this thesis is the relation of socioeconomic position to health, particularly to CHD, and this review Chapter will, therefore, be limited to inequalities according to socioeconomic position in CHD. The scope of inequalities according to socioeconomic position and its relation to health, affects many conditions and most stages of the life course. Poor socioeconomic conditions (for example, lower levels of education or income, lower occupational grades, lack of basic household amenities, not owning a car or house) are known to be related to higher infant mortality and overall mortality rates, as well as morbidity and mortality from cardiovascular disease (CHD and cerebrovascular disease), many cancers and respiratory diseases;[74-77] these socioeconomic inequalities in health are present in men and women. As described in the previous section, the pattern and extent of these socioeconomic differences in health can vary over time, giving an indication of the dynamic nature of socioeconomic factors and their impact on overall health as well as on specific diseases. A US study showed that socioeconomic differentials varied according to different causes of mortality; large differentials were observed for AIDS, diabetes, CHD and lung cancer, and weaker differences were present for leukaemia and other blood diseases.[71] Nevertheless, the influence of socioeconomic conditions on health is well-established in western populations.[29;74;78]

2.5 Measures of socioeconomic position

Socioeconomic position has been described as a term that refers to the social and economic factors that determine the position of individuals in society.[79;80] The concept of socioeconomic position has its origins in the works of Marx and Weber. Marx described class as structural positions within the social organisation of production, while Weber described class in terms of people sharing economic opportunities (resources, abilities, skills) as a result of market relations.[81;82] Class as a social relationship, therefore, provides insight into pathways to socioeconomic inequalities in health – employers or owners seek to reduce wages of workers and to have lower corporate taxes; employed workers seek to increase their wages, do more than one job and have more members of the household in the paid labour force. The concept of socioeconomic position comprises both resource-based measures reflecting actual resources such as income and education, as well as prestige-based measures such as status/rank, usually evaluated as access to goods and services, and knowledge.[80;83] The term ‘socioeconomic position’ has been used to describe the social and economic stratification of individuals in societies. Individuals within societies are stratified by levels of social, economic, cultural and political advantage; this determines the balance of health damaging or health promoting exposures and resources.[84] This is the essence of the nature of socioeconomic position and why it relates to health. A counter-argument, however, proposes that health determines socioeconomic position. This 'social selection' theory argues that healthier individuals come to occupy higher socioeconomic positions, while sick individuals drift down the social hierarchy, resulting in socioeconomic inequalities in health.[85;86] Although this phenomenon occurs, evidence shows that social mobility makes little contribution to the overall socioeconomic differentials in health and mortality.[87;88]

Socioeconomic position in adult life has been measured using different indicators including occupation, education and other markers of socioeconomic conditions such as income.[80;83] Ideally, to measure socioeconomic position in later life, the indicator should be characterised by ease of use, ability to demonstrate gradients in health, and stability over time and across the life course. The measurement, strength and limitations of different socioeconomic indicators and complexities in assessing socioeconomic position in older age will be discussed here.

2.5.1 Occupation-based measures

Occupation-based social class is a widely used measure of socioeconomic position. Following from Marxist and Weberian theories, social class refers to the location of people within the economy and is defined by economic relationships, particularly those between the employer and employee.[80] Therefore, social class describes how and why the social and economic well-being of people is stratified in the society. The Registrar General's Classification was first introduced by the Registrar General, Dr. T.H.C. Stevenson in 1911, defining occupations on the basis of degree of skill, in order to analyse infant mortality rates in England and Wales.[89] This classification comprises six social class groups – I (professionals), II (managerial), IIInon-manual (semi-skilled non-manual), III manual (semi-skilled manual), IV (partly skilled) and V (unskilled). A limitation of using an occupation-based measure is the exclusion of populations outside of the labour force such as the unemployed, housewives, students and children. Some of the critiques of the Registrar General's classification are that it was devised to bring about mortality differences, and that it lacks an explicit theoretical basis.[90] It was argued that the classification was carried out by combining socioeconomic groups with high mortality rates into lower social classes and vice versa.[90] However, reclassification of occupations into social classes do not appear to contribute to social class differentials in mortality; similar social class gradients for 1981-85 were observed, regardless of whether occupations were coded according to the 1971 or 1981 classification.[85] Occupation, along with skill and professional qualifications, has remained the primary basis of the Registrar General's classification and it has been refined over time so as to incorporate employment status. This has enabled distinction between people within the same occupation but with different levels of responsibility, such as foreman or employer, in addition to the occupational group.[91] This classification has been widely used to assess health inequalities across different socioeconomic groups and to assess changes in inequalities over time. Although lack of an explicit theoretical basis for the classification may limit its explanatory power, social classes based on different occupations and skills can be argued to encapsulate dimensions of socioeconomic position including education, assets, income, status and social circumstances. Other occupation-based measures of socioeconomic position are the Cambridge Scale and the Erikson-Goldthorpe scheme, which have been used to stratify societies. The Cambridge Scale differentiates people according to 'social distances' between occupations defined by similarities in lifestyle and resources.[92] The Erikson-Goldthorpe (E-G) scheme is based on conditions of employment, degree of occupational security and promotion aspects.[93] While there is an explicit theoretical basis for the E-G scheme, working relations are likely to change and the classification would require constant revision. The more recent occupational classification, the National Statistics Socioeconomic Classification, is similar to the theoretical basis for the E-G scheme and is based on employment relations, labour market situation and work situations.[83]

The main indicator of socioeconomic position in older age used in this thesis is occupational social class. Although occupation-based measures can be difficult to ascertain in post-retirement age, the social class measure used in the thesis is based on the longest-held occupation ascertained in middle-age. A major strength of this measure is that it would reflect social position across most of the adult life, and is likely to also be related to socioeconomic conditions in older age after retirement. In the present study, the Registrar General's classification was used to categorise social class groups. The classification offers advantages in being easy to use and has the ability to demonstrate socioeconomic gradients in health.

2.5.2 Education

Education has been commonly used as an indicator of socioeconomic position as it influences potential earnings, material resources, cognitive abilities, informational resources, and behavioural patterns related to health and lifestyle.[79;94;95] Education is measured as the length of education (number of years or age of completion of education) or as the level of education attained (primary, high school, or higher education). While education has been used as a generic measure of socioeconomic position, it is strongly determined by parental socioeconomic circumstances and, therefore, can be argued to be more of a marker of early life socioeconomic position.[79]

Since education is influenced by resources in early life, and affects adult occupation and income (own socioeconomic position), it captures both the effects of early life socioeconomic position and the influence of adult resources (influenced by education) on health.[79] The meaning of education may, therefore, be complex to interpret. Proposed strengths of education are that it is easy to measure through questionnaires and is a relatively fixed measure, which remains generally stable after early adulthood. Education is not likely to be affected by health status in adulthood, thus limiting the potential of 'reverse causation'.[94] Although education as a marker of socioeconomic position is applicable to people in most circumstances - working or retired, young and old, men and women - the meaning of educational attainment can vary markedly across time, gender and place.[79;80;94] The value of different levels of educational attainment changes over time with changes in educational opportunities. For example, older birth cohorts are likely to be classified as less educated when compared with younger cohorts and relative earnings may also vary for a graduate between the 1950s and 1990s.[80] Another limitation of education as a marker of socioeconomic position is encountered when using years of education as a measure of educational attainment since it places equal value on any single year of education, regardless of whether the year difference is experienced at secondary school or higher education level.[96]

2.5.3 Other measures of socioeconomic position

Housing-based indicators including housing tenure and housing conditions have been used to measure the material aspects of socioeconomic circumstances.[79;84] Housing tenure is the most commonly used indicator, often grouped as - owner-occupier, renting privately or renting from local authority.[79] Housing conditions include presence of dampness, quality of housing and overcrowding. Housing conditions are used as markers of material circumstances as well as possible exposures for specific diseases.

Car ownership is another marker of material resources and circumstances, and is related to health outcomes.[97] Wealth measured as household assets has also been used as an indicator of socioeconomic position. These indicators (house and car ownership and wealth) are easy to measure but their relative importance can change over time.[79] A disadvantage of these measures in older age is that housing status and car ownership are likely to change in older age and can be influenced by health status. Income is another measure of socioeconomic position, which directly captures material circumstances. However, being a sensitive question it is not always easy to elicit a meaningful response from participants.[79]

2.6 Socioeconomic inequalities in CHD in middle-age

Studies in the UK and other Western countries, over the last 20-30 years show marked socioeconomic differences in CHD in middle-aged populations; about a two-fold greater risk of CHD in lower compared with higher socioeconomic groups has been frequently reported in middle-aged adults from studies using different measures of socioeconomic position including social class, income and education.[23;24;34;39;98] This section presents a brief review of some of the UK-based studies showing the relationship between socioeconomic position and CHD in middle-age. The regular publication of the Decennial Supplements using census data for England & Wales demonstrated social class differences in all-cause mortality as well as CHD mortality. According to the 1997 Decennial Supplement, CHD mortality rates were higher in lower social class groups.[99]

The Whitehall study comprising London-based male civil servants aged 40-69 years was initiated between 1967 and 1969 to examine socioeconomic differences in CHD and to explore reasons for these inequalities.[100] 17,530 civil servants in the study were classified into different employment grades – administrative, executive, professional, clerical and 'other'. 'Other' grades were lowest in status, which included messengers and other unskilled manual workers. After seven and a half years of follow-up, a social class gradient in CHD mortality was observed such that the lower the employment grade, the higher was the CHD mortality risk. Men in the lowest grade ('other') had 3.6 times the CHD mortality rate compared to men of the highest grade (administrative).[100] Coronary risk factors including cigarette smoking, cholesterol, blood pressure, body mass index and physical activity accounted for some of the social class variations in CHD mortality, although about 60% of the inequalities remained unaccounted for in this analysis. The Whitehall II study included men and women in a cohort of 10,308 London-based civil servants aged 35-55 years examined between 1985 and 1988.[25] After an average follow-up of 5.3 years, the risk of doctor-diagnosed CHD in low compared to higher employment grades was two-fold in men, and about 30% greater in women.[25]

The Caerphilly and Speedwell studies recruited 4,860 men from Caerphilly (South Wales) and Speedwell (a district of Bristol) who were aged 45-63 years when examined during 1979-1982.[101] Information was collected on occupation and coronary risk factors. Using the Registrar General's classification, CHD prevalence was found to increase from social class I to social class V.[102] Smoking levels were greater in manual social classes, while total cholesterol levels were slightly higher in non-manual groups. Haemostatic markers including fibrinogen and white cell count were higher in lower social classes, although cigarette smoking accounted for these associations.

The Scottish Heart Health Study comprised a representative sample of Scottish population with just over 10,000 men and women aged, 40-59 years recruited in 1984-1987.[103] The prevalence of CHD was greater in manual compared to non-manual social classes, and in those who were not home owners compared to home owners.[103] After a follow-up of over 7 years, risk of CHD incidence and mortality was found to be about 50% higher in those who did not own homes compared to owner-occupiers.[104] About 40% of this socioeconomic difference was accounted for by cigarette smoking.

Similar social class differences in CHD in middle-age were also observed in the British Regional Heart Study. In men aged 40-59 years followed-up for an average of 6 years, manual social classes had a 44% greater CHD incidence rate than non-manual groups.[37] This increased risk in manual groups was reduced to 24% after taking CHD risk factors, particularly smoking, into account. Studies comprising women have also shown socioeconomic differences in CHD risk. The mothers of the 1958 British birth cohort of lower social classes had a greater risk of CHD mortality compared with those of higher social classes.[105] Differences in CHD risk were also observed in the British Women's Heart and Health Study according to social class.[106] Thus, UK-based studies have consistently shown that the current socioeconomic variation in CHD is such that lower compared to higher socioeconomic position is associated with greater CHD risk.

2.7 Quantifying socioeconomic inequalities

Socioeconomic inequalities can be expressed in relative as well as absolute terms, and both these measures have important, yet distinct implications.[34;107] An absolute measure of inequality represents the (absolute) difference in the rate of the disease between one group and the specified reference point; this indicates the extra burden in the lower socioeconomic groups and, thus, shows the absolute magnitude of the inequalities.[34;107] A relative measure expresses the inequality in terms of a chosen reference point, for example as a rate ratio, and is useful for assessing the strength of the association between socioeconomic position and the outcome. Thus, relative inequalities indicate the risk in a group compared to a reference group or the extent of disparities, while absolute inequalities express the public health impact of relative inequalities taking account of the absolute risk levels in the population. Both relative and absolute measures are, therefore, useful ways of expressing inequalities. However, it is possible that relative and absolute measures may yield different results particularly when comparing inequalities over time – if the underlying rates of the disease decrease, a decrease in absolute inequalities may still co-exist with an increase in relative inequalities.[107]

2.8 Time trends in socioeconomic inequalities in CHD

Influences of socioeconomic factors are clearly seen in the distribution of chronic diseases including CHD. CHD risk varies by socioeconomic groups; greater in lower than higher socioeconomic groups,[39;74] although this has not always been the case.[39] The 1931 census of England & Wales showed a greater prevalence of coronary disease in social classes I and II compared to lower social classes.[39;108] Later by the 1960s, lower socioeconomic groups had greater CHD mortality rates than higher socioeconomic groups.[109] Cigarette smoking, which in the early twentieth century was more common in higher social classes due to its high price, and the dietary patterns of these social classes resulted in higher socioeconomic groups having greater CHD rates as observed in the 1931 census.[109-111] However, while higher social classes started to reduce their exposure to these factors in response to new knowledge, these risk behaviours started to increase in lower social classes.[111] This resulted in the reversal of the social class-CHD relation by the 1960s. This again reflects the importance of socioeconomic factors in the distribution of CHD over time. This pattern of lower compared with higher socioeconomic groups having greater CHD risk currently remains in Britain, and is also seen in other developed countries.[19;20;29]

The Acheson report in 1998 showed that relative social class differences in CHD mortality had continued to widen from the early 1970s to early 1990s (see Figure 2.1).[34] Although the overall and CHD mortality rates had declined in the population as a whole, mortality rates had fallen more in higher social classes than lower social classes leading to the increasing differential.[34;112] Such widening socioeconomic inequalities in mortality have continued till the end of the 1990s.[76] However, the pattern of more recent trends in socioeconomic inequalities in CHD is not known. Some evidence is available from the Department of Health status report (2005) on the Programme for Action.[113] According to this report, between 1995-97 and 2001-2003 the absolute gap in circulatory disease mortality between the most deprived areas and average circulatory death rate for England had narrowed by 22%, reflecting overall decline in rates, but the relative gap in circulatory death rates had not narrowed (see Figure 2.2).

2.9 Socioeconomic inequalities in CHD in later life

Studies have shown that for health outcomes including mortality, morbidity and self-rated health, relative health inequalities tend to be smaller in older than middle ages;[33;94;114-117] this pattern has been observed for different indicators of socioeconomic position measured both in middle-age and older age, including occupation, education and income. Even though relative socioeconomic inequalities in total mortality have been observed to narrow with increasing age, studies show that these inequalities in overall mortality are still present in old age. However, little is known as to whether socioeconomic inequalities in CHD persist into older age. CHD is a major contributor to mortality and also has a strong socioeconomic gradient.[30] This section reviews the evidence on the extent of socioeconomic inequalities in CHD in older age. Long-term prospective as well as cross-sectional studies with relevant data provide some evidence of socioeconomic inequalities in CHD in older populations. Findings from these studies are summarised in Table 2.1 and Table 2.2.

<u>Whitehall Study</u>: The Whitehall Study recruited about 18,000 British civil servants aged 40-69 years between 1967 and 1970.[100] The subjects underwent a physical examination at baseline and were followed-up for mortality. After 25 years of follow-up, differences in mortality from various causes were investigated according to socioeconomic position.[118] Socioeconomic position was measured as employment grade assessed at study entry, which comprised administrative (the highest grade), professional and executive, clerical, and 'other' grades. There were differences in mortality rates from almost all chronic diseases. CHD related deaths were greater in lower compared with higher employment grades. 'Other' grades had nearly twice the risk of CHD deaths compared with administrative grades; the CHD mortality rate per 1000 person years was 6.41 in the administrative grade and 10.07 in 'other' grades, resulting in an absolute difference of almost 4 per 1000 person years between these grades. Age-specific CHD mortality rates were also reported. Significant socioeconomic gradients in CHD mortality were observed in older subjects, albeit, less steep than younger subjects; relative risks for 'other' vs. administrative grades were 2.57 in 40-64 year olds, 1.71 in 65-69 year olds, and 1.44 in >70 year olds. However, the study comprised an unusual

combination of socioeconomic groups. The employment grades were virtually all non-manual and were in general more privileged than the general population.[97;118] Therefore, although the direction of the socioeconomic differences is similar to what might be expected in the general population, the extent of the gradient may not be representative. Finer stratification by employment grades might have resulted in a steeper social gradient.[97]

<u>Studies in other European populations</u>: Some studies have reported socioeconomic inequalities in western European populations. One study used cross-sectional data from eight European countries including Britain.[74] Education, as a marker of socioeconomic position, was categorised into lower (no education or primary education) and higher (secondary education and above) education levels. Differences in self-report of various chronic diseases was analysed according to educational levels. The authors found strong variations in the prevalence of all chronic diseases according to education. The risk for CHD was greater in lower compared with higher socioeconomic groups; this was observed in individuals of all age groups. Relative inequalities tended to be smaller in older ages (60-79 years, odds ratio 1.18; 95%CI 1.04, 1.33) compared with the younger age group (25-59 year, odds ratio 1.29; 95%CI 1.09, 1.53). Another study in 11 western European countries and the United States compared mortality rates between non-manual and manual social classes.[78] The data for some countries were cross-sectional from population censuses of 1981, while longitudinal data from 1980-89 were available for other countries including England and Wales. Except for Portugal, CHD-related mortality was greater in manual compared to non-manual classes in all other countries. While the focus of this study was to examine between-country variations in CHD-related socioeconomic inequalities, age-specific results were also presented. The

relative difference in CHD mortality between manual and non-manual groups was lower in older compared with younger age groups; for example in England and Wales the rate ratio for CHD mortality was 1.68 in 30-44 year olds and 1.26 in 60-64 year olds. For the period of the 1990s, another study in 10 western European countries showed that relative socioeconomic inequalities in CHD were present in older age.[22] The relative risk for CHD in low compared with middle/high educational levels was 1.55 in 30-59 year old men and was 1.22 in men aged 60 years and over. A similar study in eight western European countries reported that relative inequalities by educational levels in CHD mortality rates appeared to be lower in those aged ≥75 years compared to younger age groups (40-59 years).[29] Absolute difference in CHD mortality, however, was greatest in old age (≥75 years). The study also showed that CHD was the single largest contributor (about 18%) to socioeconomic differences in total mortality. While these studies indicate that a general pattern of socioeconomic inequalities in CHD persists in older ages in different countries, most reports were based on cross-sectional data.

<u>English Longitudinal Study of Aging</u>: Approximately 12,000 participants aged over 50 years from three surveys of the Health Survey for England (1998, 1999 and 2001) were included in the English Longitudinal Study of Aging (ELSA).[119] As part of Wave 1 of ELSA, a cross-sectional study was carried out in 2002 in this nationally representative sample. Information on CHD was based on self-report of doctor-diagnosed heart disease. A social class gradient in the prevalence of coronary disease was present in older age, but showed evidence of narrowing with increasing age. Amongst men aged 50-59 years, the prevalence of heart disease in routine/manual groups was 14.3% compared with 8.9% in professional/managerial groups, implying a relative risk of 1.61 in routine/manual groups. In men aged 60-74 years, the prevalence of heart disease was

23% in routine/manual groups and 17.4% in professional/managerial groups (a relative risk of 1.32 for routine/manual vs. professional/managerial groups). Absolute difference in prevalence of heart disease between the highest and lowest social classes, however, did not appear to be substantially different in the older compared with younger age groups. Another study on the first wave of ELSA reported the prevalence of self-reported chronic diseases according to education and income levels in participants aged 55-64 years.[120] Relative inequalities by education and income levels were present for myocardial infarction. Those of low compared with high educational levels had a 1.32 times greater relative risk of myocardial infarction (prevalence of heart disease 4.5% vs. 3.4%). The risk of myocardial infarction was greater in low income levels (6.5%) compared with high income groups (2.4%), nearly a three-fold greater relative risk and a four-fold absolute risk difference. However, since these results were based on cross-sectional data, causality or a temporal relationship between socioeconomic position and CHD in older age cannot be entirely established; although educational level is not likely to be affected, it is possible for income levels to be influenced by health status. Also, self-report of disease can be argued to be a less accurate measure of disease status than medical records due to reporting bias; awareness of disease or reporting may differ according to socioeconomic groups. However, socioeconomic differences in self-reported heart disease were consistent with socioeconomic differences in behavioural (cigarette smoking, physical activity) and biological risk factors (HDL-cholesterol, CRP and fibrinogen) in this study.

Swedish Annual Level-of-Living Survey: This study comprised a random sample of Swedish men and women who participated in an annual national survey conducted in 1988 and 1989.[121] The analyses were based specifically on individuals aged ≥65 years followed-up for CHD events (fatal and non-fatal) for a mean period of 8.2 years. Occupation was used to measure socioeconomic position; categories included manual workers, lower level employees, middle level employees and professionals, and self-employed and farmers. Compared with middle level employees and professionals, manual workers and lower level employees had a relative risk of about 1.50 for CHD. This study indicates the relationship of socioeconomic position with CHD in a representative older population. However, the study was restricted to hospitalised cases of CHD, thereby excluding any non-hospitalised live events which were possibly less severe, and the socioeconomic gradient in this group was not known.

Prospective study in South Korea: Health insurance data on South Korean government officials and teachers were used to analyse age-specific socioeconomic differences in all-cause and CHD mortality.[36] Nearly 600,000 subjects aged 30-64 years were followed-up for 9 years for this analysis. This study reports both the relative and absolute extent of socioeconomic inequalities. Income was used as a measure of socioeconomic position. The absolute difference in CHD mortality rate per 100,000 between low and high income groups was greater at 55-64 years (38) than at 30-44 years (25). A weak increased relative risk for CHD mortality in low income groups compared with high income groups was observed in older age (age-adjusted hazard ratio for 55-64 year olds was 1.22; 95%CI 0.97, 1.53). While this age group (55-64 years) represents the younger end of the older age spectrum, the results give an indication of socioeconomic inequalities in CHD in different age groups. Relative inequalities in CHD mortality were weaker in 55-64 year old subjects compared with subjects aged 30-44 years (hazard ratio 1.40). It is possible that since this cohort comprised a homogenous group of public sector employees, a more socioeconomically representative sample would perhaps have shown greater inequalities in CHD mortality.

Copenhagen Male Study: A prospective Danish study of older men with a mean age of 63 years (53-75 years) reported that lower social class groups had a 44% relative risk increase of CHD incidence compared with higher social classes.[26;122] Age-specific results were not presented, so comparison of inequalities in CHD between older and younger age groups could not be made. Absolute inequalities in CHD were also not shown. The subjects were selected from certain private and public companies such as railways, telephone, post, road construction, custom, and medical industry. It is likely that the subjects were healthier than the general population or those not working in these industries and with possibly a different risk factor profile.

In summary, there are a few studies, some cross-sectional, in older populations which demonstrate that socioeconomic inequalities in CHD are present in later life (findings are summarised in Table 2.1 and Table 2.2). These socioeconomic inequalities in older age were observed regardless of whether socioeconomic position was measured in middle-age[26;36;118] or later in life.[119;121] The socioeconomic inequalities in CHD in older age appear to be weaker compared to that in middle-age. Weakening of inequalities in older age could be due to a healthy survivor effect as a result of individuals from lower socioeconomic position dying earlier and healthier subjects surviving into old age. However, one of the above studies excluded subjects with prior disease to limit the possibility of a healthy survivor effect.[36] A prospective study in a representative sample of adults in the USA investigated the impact of survival bias on socioeconomic inequalities in health in older age.[114] The study found that results based on the surviving sample were robust even when subjects who were lost to follow-up or who had died were included. Therefore, although selective survivorship remains likely when studying older populations, it does not appear to explain the age-related convergence in relative health inequalities.

2.10 Coronary risk factors

2.10.1 Established coronary risk factors

The major established risk factors for CHD include cigarette smoking, blood pressure, dietary fat-blood lipids, physical inactivity and obesity.[10;11;123;124] These have been designated as major risk factors because of their high prevalence in populations (particularly in Western countries), their impact on coronary risk, and their preventability and reversibility.[125]

The British Doctors Study first reported the increased risk of CHD in smokers compared to non-smokers in 1954. Since then, observational studies have established that cigarette smoking increases the risk of CHD by about 1.5 to 3 times or more.[126-129] The increased risk of CHD associated with cigarette smoking persists in older age.[130] Smoking cessation has been found to reduce the risk of subsequent mortality and CHD risk even among those with CHD compared to current smokers.[131-133] Passive smoking has also been shown to be associated with increased risk of CHD. Non-smokers living with smokers may have up to 30% greater coronary risk,[134;135] although this risk can be greater when exposure to passive smoking outside of homes is also considered.[136;137]

Evidence from prospective studies demonstrates the increased risk of CHD due to raised blood pressure. Results from the Prospective Studies Collaboration demonstrated that a difference of 20 mm Hg in systolic blood pressure or 10 mm Hg in diastolic pressure was associated with a two-fold difference in risk of CHD mortality.[123] There was no evidence of a ‘threshold’, at least down to 115 mm Hg for systolic and 75 mm Hg for diastolic blood pressure, below which blood pressure was not associated with CHD mortality.[123] Although the effect of blood pressure on CHD attenuates with increasing age, a 20 mm Hg lower systolic blood pressure was shown to be associated with about one-third less CHD mortality even at the age of 80-89 years.[123] Several trials and meta-analyses based on trials have demonstrated a decrease in CHD risk associated with a reduction in blood pressure through medications such as beta-blockers, angiotensin converting enzyme (ACE) inhibitors and diuretics and their combinations.[138-140] A meta-analysis of trials of these drugs in older people (>60 years) showed that a decrease of 10 mm Hg in systolic blood pressure and 4 mmHg in diastolic blood pressure is associated with 23% reduction in risk of CHD (non-fatal and fatal).[141] Dietary salt intake has been identified as one of the major determinants of blood pressure in populations.[142-144] Other factors that influence blood pressure are physical inactivity, increased body mass index (BMI),[145] low dietary intakes of potassium and fruit and vegetables,[146;147] and low birth weight.[148]

Several studies in different populations have consistently shown increasing cholesterol levels to be associated with raised CHD risk. The nature of this association is such that there is no ‘threshold’ below which cholesterol levels are not associated with increased CHD risk.[124;149] Total cholesterol is also associated with increased CHD risk in the elderly, although more weakly than in younger ages.[124] The Prospective Studies

Collaboration showed that a prolonged difference of 1 mmol/L lower total cholesterol was associated with lower CHD mortality risk of about a half in early middle-age (40-49 years) and about a sixth risk in old age (70-89 years).[124] The low density lipoprotein (LDL) and high density lipoprotein (HDL) components of total cholesterol have opposite associations with CHD risk – higher HDL-cholesterol and lower non-HDL cholesterol (largely LDL-cholesterol) decrease the risk of CHD mortality,[124] The Prospective Studies Collaboration also showed that on average, 0.33 mmol/L higher HDL cholesterol and 1 mmol/L lower non-HDL cholesterol were each associated with about a third lower CHD mortality.[124] Randomised controlled trials on statins have shown the protective effect of reducing LDL-cholesterol levels on CHD risk, even in older populations. A meta-analysis of such trials demonstrated a 23% proportional reduction in 5-year CHD risk (non-fatal myocardial infarction and CHD deaths) per mmol/L reduction in LDL-cholesterol.[150] Dietary intake of fat is a major determinant of blood cholesterol levels. Studies have shown that dietary cholesterol increases blood cholesterol and that saturated fatty acids are the main determinant of blood cholesterol, while polyunsaturated fatty acids lower blood cholesterol levels.[151-153]

Physical inactivity as a risk factor for CHD was highlighted by the work of Professor Jeremy Morris and colleagues. The incidence of CHD (fatal and non-fatal) in male British civil servants who engaged in vigorous exercise or physical activity was found to be less than half that of those who were inactive.[154;155] Several other studies have confirmed the protective effect of physical activity on CHD risk in both men and women in different population groups.[156-162] Physical activity also lowers CHD risk in older age.[161;163;164] Furthermore, taking up physical activity in sedentary individuals has been reported to reduce CHD risk even in older age.[165;166] While earlier studies indicating a strong protective effect of vigorous activity on CHD risk suggested a threshold effect of physical activity as a coronary risk factor, later studies demonstrated that moderate physical activity also is associated with lower CHD risk emphasising a more continuous relationship of physical activity with CHD.[162;163;167]

The risk of CHD is also positively associated with increased body mass index [(BMI), a measure of generalised obesity/adiposity] in both men and women.[168-171] A meta-analysis of prospective studies demonstrated that overweight (BMI 25.0-29.9) and obesity (BMI≥30) were associated with increased CHD risk.[172] Although this increased CHD risk associated with being overweight was to a large extent (45%) accounted for by blood pressure and blood cholesterol, an increased CHD risk of nearly 50% for obesity remained after adjustment for blood pressure and cholesterol levels.[172] This meta-analysis also revealed that a 5-unit BMI increment was associated with a 16% increased risk of CHD after adjustment for blood pressure and cholesterol levels. A previous systematic review yielded similar results; a 14% increase in CHD risk was associated with a 2-unit increase in BMI.[173] Greater CHD risk associated with increased BMI or with obesity has also been reported in older age.[174;175] Studies also suggest that measures of obesity or adiposity other than BMI such as weight circumference or waist to hip ratio, which better reflect central adiposity, may be better indicators of CHD risk.[175-177] BMI has been referred to in the literature both as a 'behavioural' and as a 'biological' risk factor for CHD[178-180] – behavioural since it is a product of lifestyle factors including dietary patterns and physical activity, and biological since it is essentially an anthropometric measure, which is also influenced by genetic and physiological mechanisms.[181] In this thesis, BMI is referred to as a behavioural risk factor for CHD.

Besides these established risk factors, alcohol consumption has been reported to be associated with CHD risk. Several studies have demonstrated a U-shaped or J-shaped relationship between alcohol consumption and CHD risk, with a greater CHD risk in non-drinkers and those who drink excessively compared to those who are light/moderate drinkers.[182-184] However, this relationship of alcohol consumption with CHD has been questioned because of the characteristics of the subjects in different categories of alcohol consumption – regular drinkers tend to have characteristics advantageous to health; non-drinkers comprise teetotallers as well as ex-drinkers who have a higher proportion of co-morbidities; and change in alcohol intake over time such as movement from heavy or moderate drinking to non-drinking is not always taken into account.[184] Despite these issues, the association of heavy drinking with increased CHD risk has been reported by several studies.[182;183;185;186]

2.10.2 Novel coronary risk factors

Inflammatory and haemostatic markers: Increasing amounts of research has been carried out in investigating novel factors responsible for CHD. Inflammatory and haemostatic markers, through their roles in arterial plaque formation, plaque rupture and thrombosis, have been implicated as novel factors which can increase risk of CHD events.[12] Markers particularly implicated include C-reactive protein (CRP), fibrinogen, von Willebrand factor (vWF), white blood cell count, and tissue plasminogen activator antigen. Trauma, infections or coronary events such as myocardial infarction result in a cascade of inflammatory response including elevations in proinflammatory cytokines [e.g. interleukin 6 (IL-6)], acute phase proteins including CRP, and haemostatic markers including fibrinogen, factor VIII, vWF and tissue plasminogen activator (t-PA) antigen.[12] These haemostatic changes alongside platelet activation result in greater prothrombotic activity, and are also accompanied by increased circulation of fibrinolytic markers (e.g. fibrin D-dimer). Higher levels of fibrinogen and white blood cells also increase blood and plasma viscosity, which in turn can reduce blood flow to the myocardium causing ischaemia and infarction. Prospective studies and meta-analyses have shown that these inflammatory and haemostatic markers are associated with an increased risk of CHD in both middle-age and older age.[13;187-194] It has, however, been argued that these inflammatory markers could be consequences of ischaemic events rather than causes of CHD.[12] Additionally, inflammatory markers and CHD are both related to established coronary risk factors (smoking, physical inactivity, alcohol consumption, obesity and blood lipids) which could, therefore, confound the inflammatory markers-CHD associations.[43;44;195-197] Studies have also shown no strong evidence of an association between genetic variants of CRP and fibrinogen and CHD risk.[198;199] Nevertheless, given the increasing interest in these markers as 'emerging' or 'novel' coronary risk factors, the relation of these markers to social class and their potential contribution to the socioeconomic position-CHD relationship has been investigated.[40;102]

<u>Metabolic syndrome</u>: Reaven in 1988 first described the concept of syndrome X, in which resistance of peripheral tissues, mainly skeletal muscle, to insulin-mediated glucose disposal, leads to hyperinsulinemia.[200] Other risk factors associated with insulin resistance include impaired glucose tolerance, elevated triglycerides, decreased HDL-cholesterol, increased blood pressure and obesity. This concurrence of metabolic abnormalities has come to be known more commonly as the 'insulin resistance syndrome' or 'metabolic syndrome'. Different definitions of the syndrome have evolved, such as those put forward by the American Diabetes Association (ADA), the

World Health Organisation (WHO), and the U.S. National Cholesterol Education Programme (NCEP).[201] Although the major components, insulin resistance, dyslipidemia, hypertension, and obesity or central adiposity are common to these definitions, the specific diagnostic criteria differ. Circulating insulin (a marker of insulin resistance) has been found to have a modest association with increased CHD risk.[202] Since its conception, the metabolic syndrome has been described as a major public health problem,[203] and this clustering of cardiovascular risk factors has generated interest in investigating its association with increased coronary risk.[15] Despite studies which report metabolic syndrome to be related to CHD risk, the evidence is not consistent,[204;205] and the use of the concept of metabolic syndrome has been critically examined.[201] The definitions of metabolic syndrome lack universal consensus, although the NCEP criteria are often used because they are less complex than the WHO definition. Moreover, the pathophysiology underlying the metabolic syndrome is unclear, and whether the associated CHD risk is greater above and beyond the individual coronary risk factors that constitute the syndrome is not fully known.[201]

<u>Dietary factors</u>: The Seven Countries Study was important in investigating the role of diet in CHD and in influencing the diet-heart hypothesis, whereby dietary fat intake (higher saturated fat and lower polyunsaturated fat) is implicated in increasing CHD risk.[206] Blood cholesterol has been proposed to act as a key intermediary between dietary fat and CHD.[206] Higher intakes of dietary saturated fat and lower intakes of polyunsaturated fat have been shown to increase blood cholesterol levels, which in turn increases CHD risk.[206] The strong associations of total blood cholesterol and LDL-cholesterol (or non-HDL-cholesterol) with increased CHD risk has been highlighted above. More recently vitamin C, due to its antioxidant properties, has also been

implicated as offering protection from CHD.[207] However, no evidence for this was found in large randomised controlled trials.[208;209]

2.11 Possible pathways linking socioeconomic position to CHD in older age

Attempts to understand the basis of socioeconomic inequalities in CHD have traditionally started with exploring the role of established coronary risk factors such as cigarette smoking, blood lipids, blood pressure and excess body weight.[36;38;39;97;210;211] Since most studies in middle-age populations show that these established risk factors do not explain a substantial proportion of the relationship between socioeconomic position and CHD, novel coronary risk factors such as inflammatory and haemostatic markers have been postulated to influence this relationship.[40;212;213] Few studies have explored the pathways to socioeconomic inequalities in CHD in older populations beyond 60 years of age. This section reviews the evidence on pathways to socioeconomic inequalities in CHD in older age.

2.11.1 Established coronary risk factors

The association of established coronary risk factors with socioeconomic position has been widely reported in middle-aged populations.[100;102;180;214;215] Cigarette smoking, high blood pressure, lower physical activity and obesity are more common in those from lower compared to higher socioeconomic positions, while consistent socioeconomic variations in blood cholesterol levels have not been observed. There is, however, relatively less evidence on whether socioeconomic position is related to these established coronary risk factors in older age. Some studies have shown that higher levels of smoking, physical inactivity, obesity and heavy alcohol consumption are present in older people of lower socioeconomic groups.[121;216] As in middle-age, lower compared with higher socioeconomic groups are associated with higher blood pressure in old age.[121;216;217] The evidence for the relation of blood lipids with socioeconomic position, however, has not been strong and consistent. In both middle-aged and older subjects, total cholesterol has been reported to be, if anything, lower in lower socioeconomic groups.[37;39;180;218;219] However, a study in older women observed low levels of HDL-cholesterol and higher triglycerides in lower socioeconomic groups.[220]

Studies in middle-aged populations show that established coronary risk factors can have a limited influence (15%-45%) and do not contribute substantially to the relationship between socioeconomic position and CHD.[24;38;98;211;221] In the Whitehall Study comprising 40-64 year old subjects (aged mostly under 60 years), about 40% of the relative social class inequalities in CHD mortality was accounted by established coronary risk factors particularly smoking and blood pressure, while the rest remained unexplained inequalities.[39] After a follow-up for 15-year mortality risk in the Whitehall study, reducing coronary risk factors (cigarette smoking, blood pressure, total cholesterol and blood glucose) was reported to account for 69% of the absolute social class difference in CHD mortality.[222] Much less evidence is available in older populations (>60 years). Studies in European countries, including data on England and Wales, which report socioeconomic inequalities in CHD in older age have not investigated the pathways to these inequalities.[22;29;30] One of the few studies to investigate the role of established coronary risk factors in socioeconomic inequalities in CHD in older age was a nationally representative study comprising ≥65 year old Swedish participants.[121] Cigarette smoking, physical inactivity, BMI and hypertension were found to be largely responsible for the relative social class differences in CHD in this older population. A South Korean study reporting age-specific results found cigarette smoking to be the largest contributor (26%) to the relative socioeconomic inequalities in CHD in 55-64 year olds, while blood pressure and cholesterol made little contributions.[36] In a Danish study comprising older men (mean age 63 years), established coronary risk factors including blood pressure, lipids, smoking and physical activity made little contributions to the increased relative risk of CHD in lower social classes.[26] Further research is required to assess the contribution of established coronary risk factors to socioeconomic inequalities in CHD in older age.

2.11.2 Inflammatory and haemostatic markers

Earlier studies have suggested that inflammatory and haemostatic markers are associated with socioeconomic position; lower socioeconomic groups have been observed to have higher levels of inflammatory and haemostatic markers in middle-age.[40;213;223] This has led to the hypothesis that these novel risk factors could be important contributors to the relationship between socioeconomic position and coronary risk, possibly through psychosocial processes.[223;224] It has been postulated that psychological stresses, which can be greater in lower socioeconomic groups, could induce procoagulant pathways and haemostatic activities resulting in greater coronary risk.[224] Studies in middle-aged and some older (mean age >60 years) populations have shown that CRP levels are greater in lower socioeconomic groups.[40;41;213;225;226] Similar results, mostly in middle-aged populations, have been observed for fibrinogen and IL-6.[40;41;213;223;226-228] The relationship between socioeconomic position and von Willebrand factor (vWF) is less consistent. Although the Whitehall II Study with subjects aged 39-63 years found a social class gradient in vWF,[229] the Caerphilly study (men aged 45-59 years) found no relationship between social class and vWF and other haemostatic markers.[230] The relationships observed between these novel markers and socioeconomic position could, however, be confounded by behavioural factors since inflammatory and haemostatic markers are strongly related to behavioural risk factors.[43;44;195;196] In some studies the relationship between inflammatory markers and social position was attenuated by behavioural factors including smoking, BMI and alcohol consumption,[41;227;231] although in other studies the relationships were independent of behavioural factors.[213;225;226;229] The evidence of an independent association between inflammatory markers and socioeconomic position is, therefore, inconsistent. Moreover, most previous studies were largely restricted to middle-aged populations aged <60 years. A study in 70-79 year old men and women, reported higher levels of CRP and IL-6 in lower socioeconomic groups.[41] These associations were, however, attenuated when behavioural factors (smoking, alcohol consumption, physical activity and BMI) were taken into account.

The contribution of these novel risk factors related to inflammation and haemostasis in explaining the socioeconomic position-CHD relationship has been examined in a small number of studies. A study in professional women found that CRP and fibrinogen made a small contribution to socioeconomic inequalities in cardiovascular disease in middle-age (mean age 54 years),[212] while a Scottish study in 40-59 year old men and women reported that fibrinogen had an important influence on socioeconomic inequalities in CHD in middle-age.[104] However, previous studies have not investigated the role of novel coronary risk factors in socioeconomic inequalities in CHD in older age. Inflammatory and haemostatic markers are known to increase with age as co-morbidities increase.[232] More research is needed to understand the nature of the relationship of inflammatory and haemostatic markers with socioeconomic position in older age, and whether these risk factors influence socioeconomic inequalities in CHD in the elderly.

2.11.3 Metabolic syndrome

Studies have shown that lower socioeconomic position is associated with increased risk of metabolic syndrome.[14;233] It has, therefore, been postulated that the metabolic syndrome could be responsible for the relationship between socioeconomic position and CHD risk.[14;233] The metabolic syndrome has been shown to be linked to neuroendocrine and autonomic functioning, through psychosocial processes, resulting in insulin resistance, dyslipdemia and hypertension.[234;235] However, although studies have reported a greater risk of having metabolic syndrome among lower socioeconomic groups than in higher ones, some studies have found the association to be weak in men.[236;237] Moreover, most studies so far have been cross-sectional in nature, not providing causal evidence for a prospective relation between socioeconomic position and metabolic syndrome. Behavioural risk factors including smoking, physical inactivity and BMI are associated with increased risk of metabolic syndrome,[45;238;239] and are also closely related to socioeconomic position.[19;214] Therefore, these factors could be possible confounders of the relationship between socioeconomic position and metabolic syndrome. A population-based study in France comprising middle-aged men and women found lower educational and household income levels to be associated with the metabolic syndrome.[236] When adjusted for smoking, physical activity and BMI, household income was associated with metabolic syndrome only in women, while the association of low educational levels with increased risk of metabolic syndrome in men and women remained after the adjustments. A South Korean National Health and Nutrition Examination survey on a randomly selected population aged >20 years, showed a lower risk of metabolic syndrome in people of higher socioeconomic groups compared to those in lower ones.[240] This relationship, however, differed according to behavioural factors – high income groups were less likely to have metabolic syndrome compared to low income groups in subjects who did not smoke or among those who did not drink heavily, whereas among subjects who were smokers or heavy drinkers, high income groups had a higher risk of metabolic syndrome. A study based on a sample of residents of Newcastle (UK) aged 49-51 years found a weak association between social class and metabolic syndrome.[241] Most of the variance in the metabolic syndrome was explained by lifestyle factors (smoking, physical activity, alcohol consumption and dietary intake of fat). The Copenhagen City Heart Study with participants aged >20 years found a three-fold increased risk of metabolic syndrome in the least compared with the most educated group; this effect was not attenuated when adjusted for behavioural factors.[242] In the participants of the British 1946 birth cohort aged 53 years, lower educational level was associated with a two-fold increased risk of metabolic syndrome, while occupational social class was not strongly related with metabolic syndrome.[243] The Whitehall II study, comprising London-based civil servants aged 45-69 years, also observed an approximately two-fold increased risk of metabolic syndrome in lower compared to higher employment grades;[14] behavioural risk factors appeared to account for little of this increased risk. Household wealth was also reported to be inversely associated with metabolic syndrome in the Whitehall II study participants, independent of behavioural risk factors.[244] A study in Finland in a middle-aged population also reported a greater prevalence of metabolic syndrome in people of lower educational levels, independent of behavioural factors.[233] Prospective analysis, however, showed that metabolic syndrome made only a slight contribution to the socioeconomic (educational levels) inequalities in CHD; greater contributions to socioeconomic inequalities in CHD were made by the individual metabolic and coronary risk factors.[233] The evidence for the relationship between metabolic syndrome and socioeconomic position comes largely from studies in middle-aged populations. Therefore, the nature of this association has yet to be established in older age.

2.11.4 Dietary factors

Dietary intake has also been widely observed to be associated with socioeconomic position.[245-247] People of poorer or lower socioeconomic groups have more unhealthy eating patterns than people of higher socioeconomic groups. A British national dietary survey showed that manual social classes had a lower consumption of fresh fruit and vegetables.[248] The Scottish Heart Health Study found that dietary patterns in middle-age varied markedly by social class;[245] the intake of fibre, vitamin C, beta carotene and vitamin E were lowest in manual groups compared with non-manual groups, while total fat, saturated fat, cholesterol and sugar intake were higher in manual social classes. The National Food Survey in Britain, however, showed that dietary fat levels fell in all social class groups between the mid-1970s and early 1980s, thus reflecting a limited contribution of saturated or polyunsaturated fat to the corresponding increase in the social class gradient in CHD that occurred in the early 1980s.[249] Smoking levels, however, had fallen more in higher social classes, contributing much more than diet to the widening socioeconomic inequalities.[249] Other dietary studies comprising representative samples from Britain including the Diet and Nutrition Survey in 1986-87 and the National Survey of Health and Development (1946 birth cohort), which collected dietary data in 1982 when participants were aged 36 years, did not show socioeconomic differences in dietary fat and saturated fat, although there were some social class differences in polyunsaturated fat intake.[250;251] A study reporting results from the Quebec Nutrition Study (QNS) and the National Health and Nutrition Examination Study (NHANES) III in the USA also did not report consistent relations between dietary fat and socioeconomic position.[252] Dietary cholesterol levels were lower in higher income groups in the NHANES study but not in the QNS subjects. Saturated fatty acids showed a positive association with income in the QNS study but an inverse association in the NHANES study population. Polyunsaturated fatty acid levels were associated with income but not education or occupation and also did not show consistent relationships with socioeconomic position in both studies. Additionally, stronger socioeconomic differences in micronutrients such as fibre and vitamin C might reflect differences in overall food consumption patterns, while less clear socioeconomic differences in dietary lipids might indicate a limited role of diet in explaining socioeconomic differences in CHD. Nevertheless, the relationship of dietary factors with socioeconomic position needs to be further investigated in older age, so as to explore the potential for dietary factors to influence socioeconomic inequalities in CHD in older age.

2.12 Early life socioeconomic position and CHD risk in later life

The role of unfavourable early life socioeconomic exposures on adult health has been long recognised.[47] When researching influences on CHD risk in adult life, a strong case has been made for the influence of early life factors.

2.12.1 Conceptual framework for the influence of early life factors on CHD

Conceptual models have been described to account for the possible pathways linking socioeconomic exposures across the life course and health in adult life.[253] The 'critical period model' postulates that an exposure acts in a specific period of life and produces a long-term effect on the body such that it cannot be modified by later life experience. This model is the basis of the 'biological programming' or 'fetal origins of adult disease' hypothesis, whereby fetal undernutrition or birth weight is associated with adult blood pressure and coronary risk.[50] A 'critical period model with later effect modifiers' extends the previous model by allowing exposures in later life to enhance or diminish the effect of early life exposures; for example the relationship of low birth weight with CHD or diabetes is stronger in those who are obese in adulthood.[50] An alternative model is the 'accumulation of risk model' in which risk factors accumulate over the life course and increase their cumulative impact on disease risk. These risk factors across the life course could be independent, or clustered as in an 'accumulation model with risk clustering'. Exposures related to socioeconomic position tend to cluster together; low childhood socioeconomic position is associated with low birth weight, unhealthy diet and poor educational attainment. A 'chain of risk model' refers to a series of exposures where one exposure leads to another, subsequently resulting in increased disease risk; for example poor childhood social environment leads to unhealthy diet in childhood, resulting in childhood and later adult obesity, which increases CHD risk. These models provide a useful framework for understanding and investigating life course influences on adult disease. The models, are, however, not exclusive of each other and may act together.[253] Despite such explicit frameworks, employing a life course approach to understanding adult disease is not free from methodological challenges.[253] Longitudinal data with repeated measures at different stages of life are required, and loss to follow-up and selection bias have to be minimised. Analytical problems also arise when adjusting for closely correlated variables, or in the case of misclassification of exposures (particularly when early life factors are retrospectively collected).[254]

A range of exposures in early life including low birth weight, accelerated postnatal growth, bottle feeding in infancy, maternal undernutrition and childhood infections have been reported to be associated with increased CHD risk in adult life.[52] The fetal origins hypothesis suggests that fetal undernutrition leads to low birth weight and also results in 'programmed' changes in lipid and carbohydrate metabolism, which can increase CHD risk in adult life.[50;52] Breastfeeding in infancy has been reported to be associated with favourable coronary risk factors such as low blood pressure, cholesterol and obesity (though associations are generally modest in strength), and with lower cardiovascular risk compared to infant bottle feeding.[52] *Helicobacter pylori*, a bacterial infection likely to be acquired in childhood as a result of overcrowding or poor housing conditions, has been implicated as a risk factor associated with CHD in adult life.[52] Though the evidence has been conflicting, it has been suggested that these early life exposures act independently of childhood or adult socioeconomic position.

2.12.2 Influence of early life socioeconomic position on CHD risk

While the association of socioeconomic position in adult life with chronic diseases such as CHD has been widely investigated, there is relatively less evidence on whether socioeconomic position in early life has an independent impact on CHD in adult life.[255] Forsdahl showed that infant mortality rates (a marker of socioeconomic conditions) were correlated with CHD mortality rates 70 years later in a Norwegian county, which implied that poor early life conditions were related to CHD in adult life.[48] A study in England & Wales found that the infant mortality rates in 1921-25 were related to CHD mortality rates in 1968-78.[256] Another such ecological study in England & Wales reported that infant mortality of 1895-1908 was correlated with CHD mortality in 1969-73 in 65-74 year olds.[257] This association was, however, attenuated when present adult social class and adult deprivation were adjusted for. The importance, therefore, of both current as well as early life socioeconomic position was highlighted in these results. Several studies, mostly prospective, have shown that lower childhood socioeconomic position is associated with increased CHD risk in adult life.[53-55;58;258] Different measures of childhood socioeconomic position have been used in these studies including father's occupation (most common), father's education, mother's occupation or education, housing conditions, overcrowding in house, and amenities such as hot water supply. These measures of early life socioeconomic position may affect CHD in adulthood because of their influence on coronary risk factors, or even due to their association with adult socioeconomic position; studies have shown that lower socioeconomic position in early life is associated with adverse behavioural factors including smoking, obesity and greater physical inactivity in adolescence and adulthood.[180;259-263] Lower socioeconomic position in childhood has also been found to be associated with higher levels of adult biological coronary risk factors including blood pressure and cholesterol.[180;264;265] Lower socioeconomic position in childhood also increases the chances of having greater socioeconomic disadvantage in adult life.[58;262] Some, but not all studies, have investigated these links between early life socioeconomic position and CHD; seven of ten studies in a systematic review did not take adult behavioural risk factors into account, while most studies had taken adult socioeconomic position into account.[54] A prospective study in the west of Scotland found that men of manual social class in childhood (based on father's occupation) had a greater risk for CHD mortality (odds ratio 1.52) compared with men of non-manual childhood social class.[58] This increased risk was weakened (odds ratio 1.25) when adjusted for adult socioeconomic position (occupational social class and car ownership), behavioural risk factors (smoking, BMI), and other coronary risk factors (blood pressure and cholesterol). Similar results were observed when the effect of greater number of siblings (a measure of overcrowding or an indicator of material resources in childhood home) with increased CHD risk was attenuated by adult social class and adult risk factors.[56] A population-based study of British women reported an attenuation of the effect of childhood social class when adjusted for adult socioeconomic position.[105] Some studies have, however, reported an independent association between childhood socioeconomic position and CHD risk.[57;105;266;267] In these prospective studies lower childhood socioeconomic position was observed to be associated with a 12% to two-fold greater CHD risk. One of the drawbacks of many studies investigating the role of childhood socioeconomic position in subsequent CHD risk is that of inadequate statistical power, which limits the ability to reveal robust estimates for the potentially modest associations. Some recent Norwegian and Swedish studies with large sample sizes have, however, been able to overcome this problem by using record linkage and census data.[258;268;269] These studies have also shown that lower childhood socioeconomic position is independently associated with a greater CHD risk, although the effect was weakened by adjustment for adult social class raising the possibility of residual confounding. Caution has to be exercised in interpreting these results. When the effect of childhood socioeconomic position is attenuated or weakened by adult socioeconomic position or adult risk factors, it does not necessarily nullify the importance of early life factors;[54;257] the influence of childhood socioeconomic position can still be crucial because of their influence on adult factors thereby setting a social trajectory.[54;253] It is difficult to fully disentangle these issues in statistical models by mutual adjustment of childhood and adult factors. Using information on socioeconomic position at different stages of the life course provides insight into the importance of exposures in life stages in contributing to disease risk.[253;262] Several studies have also shown a cumulative effect of childhood and adult socioeconomic position on CHD risk, which supports the accumulation of risk model.[58;105;106;263;270] In these studies, the contribution of lower socioeconomic positions in childhood and adulthood combined were observed to increase CHD risk to a greater extent than either lower childhood or adult socioeconomic position on their own.

Another potential limitation arises in some of these previous studies when childhood socioeconomic position is assessed retrospectively in adulthood.[58;105;106;180;259;261] Although recall of childhood socioeconomic conditions is unlikely to differ by CHD status, inaccurate recall or reporting of socioeconomic position is probable. A Scottish study reported moderate agreement between recall in middle-age of childhood socioeconomic position and childhood social class measured prospectively.[271] However, there was a tendency in the study for adults to report a higher or more favourable childhood social class than that actually recorded in early life.[271] Therefore, it is possible that retrospectively assessed early life socioeconomic position may underestimate the effect of childhood socioeconomic position on CHD risk. It has been argued that this underestimation of childhood socioeconomic position is also possible when assessing its impact relative to adult socioeconomic position on CHD, since retrospectively collected childhood position is more likely to be poorly measured compared with present adult socioeconomic position.[54] A Finnish study comprising males found a strong relationship between historically-measured childhood socioeconomic position and CHD risk, and a weaker association of recall-based childhood socioeconomic position with CHD.[260]

Despite these limitations of previous studies and challenges in interpretation of results, there is evidence that childhood socioeconomic position has an association with CHD risk in adult life. The evidence from previous studies, however, is limited to middle-aged men. In the British Regional Heart Study, an association between lower childhood socioeconomic position (father of manual social class) and greater risk of CHD, was observed in middle-aged men.[272] It is not, however, known whether the effect of childhood socioeconomic position persists in older age. Therefore, in trying to understand pathways to health in later life, there is a need for more evidence on the association between early life socioeconomic position and CHD risk in older age.

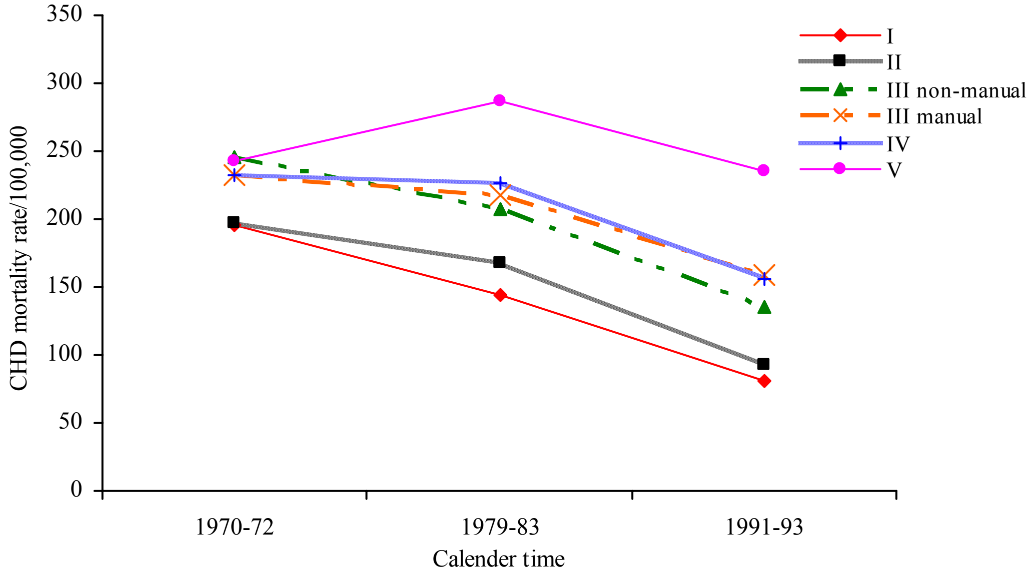
2.13 Socioeconomic inequalities in disability in the elderly with CHD

CHD, as well as making a large contribution to socioeconomic inequalities in total mortality, is also an important cause of disability in the elderly.[61;64] Since CHD shows strong socioeconomic gradients, it can be expected that people from poorer socioeconomic backgrounds will have greater disability related to CHD. Socioeconomic position has been observed to be associated with disability; people from lower compared to higher socioeconomic positions have greater disability.[66;273] Studies have demonstrated a difference of about one and a half to two-fold in disability (incidence and prevalence) in older populations according to markers of socioeconomic position including education, occupation, income and housing tenure.[65;273-278] The studies have, however, tended to focus on functional limitations (especially mobility problems). However, a small number of studies have used measures of disability such as problems with activities of daily living (ADL).[273;279;280] ADLs and instrumental activities of daily living (IADL) include routine tasks such as eating, dressing, light cooking, managing money and light shopping, and enable disability to be measured as limitations in performing social roles and independent living. Moreover, the extent of socioeconomic disparities in disability in older populations with CHD has not been well-documented. Given the greater burden of disability in older subjects with CHD, studying socioeconomic inequalities in disability in this population would reflect inequalities in overall health/quality of life associated with CHD. Therefore, there is a need to assess the extent of socioeconomic inequalities in disability in the elderly with CHD.

2.14 Conclusions and purpose of the thesis

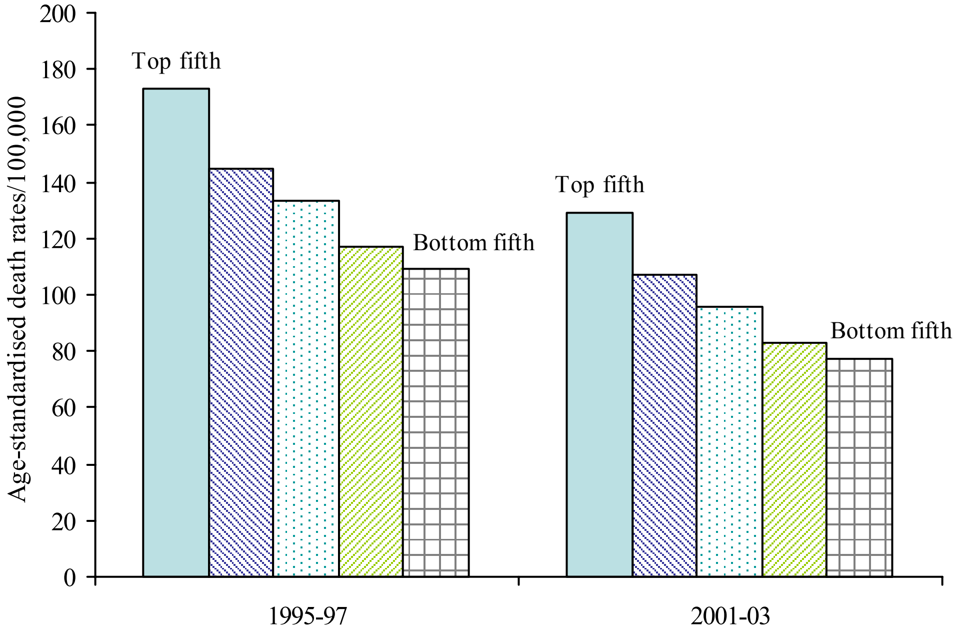
Although CHD mortality rates have declined in the U.K., the extent of recent changes in socioeconomic inequalities in CHD are not fully known. Socioeconomic inequalities in CHD are present in older age regardless of whether social class, income or education is used, and are generally weaker than those in younger ages. The review of this Chapter highlights that studies in older populations are limited in number compared with the evidence for socioeconomic inequalities in CHD in middle-age. Furthermore, studies so far have not fully addressed the possible links or pathways underlying these inequalities in older age. While socioeconomic position is associated with established coronary risk in older subjects, much less is known about the role of these risk factors in influencing socioeconomic inequalities in older age. The association of socioeconomic position with novel coronary risk factors including inflammatory markers and the metabolic syndrome in older age is less clear. Also, the potential contribution of inflammatory markers to the relationship between socioeconomic position and coronary risk in older age is not known. Evidence from middle-aged populations shows that early life socioeconomic position could play a role in adult CHD risk. However, the role of childhood socioeconomic position in CHD risk in older age is not known. Socioeconomic inequalities in disability in the elderly with CHD can be a useful way of describing the extent of health inequalities in this population. In particular, the issues that need to be addressed are: 1) along side the decline in CHD mortality rates over the last three decades in Britain, have socioeconomic inequalities in CHD narrowed?; 2) do socioeconomic inequalities in CHD change with increasing age, both in relative and absolute terms?; 3) are coronary risk factors (established and novel) in older age related to socioeconomic position?; 4) what is the extent of socioeconomic inequalities in CHD in older age, and what is the contribution of coronary risk factors (established and novel) to these inequalities?; 5) does early life socioeconomic position have an influence on CHD risk in older age?; and 6) what is the extent of socioeconomic inequalities in disability in the elderly with CHD? These questions will be addressed in this thesis using a cohort of older British men.

Figure 2.1 CHD mortality according to social class from 1970-72 to 1991-93 in England and Wales[34]



Data source: Sir Donald Acheson. Independent Inquiry into Inequalities in Health. London: The Stationary Office; 1998

Figure 2.2 Age-standardised death rates from circulatory diseases, for ages <75 years, by fifths of area deprivation in 1995-97 and 2001-03 in England[113]



Data source: Department of Health. Tackling Health Inequalities: Status Report on the Programme for Action. London: Department of Health Publications; 2005

Table 2.1 Summary of studies examining socioeconomic inequalities in CHD in older age

Study	Design	Setting	Age & gender	Outcome	Measure of SEP	Relative risks (95%CI) unadjusted	Adjusted for risk factors
Whitehall Study[118]	Prospective	London, UK	40-69 years Men	CHD mortality	Employment grade 'Other' grade vs. administrative	HR 65-69 years = 1.71 (1.22, 2.41) >70 years = 1.44 (1.18, 1.76)	Not reported for older age groups
Dalstra et al[74]	Cross-sectional	8 European countries including Great Britain	25-79 years Men & women	Self-reported heart disease	Education Lowest vs. highest	OR 60-79 years = 1.18 (1.04, 1.33)	Not reported
Kunst et al[78]	Cross-sectional and prospective	11 European countries including England & Wales (1980s)	30-64 years Men	CHD mortality	Occupation Manual vs. non-manual social class	RR (England & Wales) 60-64 years = 1.26 (1.10, 1.45)	Not reported
Avendano et al[22]	Prospective	10 European countries including England & Wales (early 1990s)	≥30 years Men & women	CHD mortality	Education Low vs. middle/high	RR Men ≥60 years = 1.22 (1.21, 1.24) Women ≥ 60 years = 1.36 (1.33, 1.38)	Not reported
Huisman et al[29]	Prospective	8 European countries including England & Wales (1990s)	≥45 years Men & women	CHD mortality	Education Low vs. high	Men 60-74 years RR = 1·32 (1.28, 1.36) Rate difference = 193 Men ≥75 years RR = 1.14 (1.10, 1.18) Rate difference = 312 Women 60-74 years RR = 1.66 (1.56, 1.76) Rate difference = 128 Men ≥75 years RR = 1.26 (1.21, 1.31) Rate difference = 322	Not reported

SEP=socioeconomic position; CI=confidence intervals; HR=hazard ratio; OR=odds ratios; RR=rate ratio; RD=risk difference

[Contd. in Table 2.2 overleaf]

Table 2.2 (Contd.) Summary of studies examining socioeconomic inequalities in CHD in older age

Study	Design	Setting	Age & gender	Outcome	Measure of SEP	Relative risks (95%CI) unadjusted	Adjusted for risk factors
ELSA[119]	Cross-sectional	England	>50 years Men & women	Self-reported heart disease	Occupation Routine/manual vs. professional/managerial	Relative risk 60-74 years = 1.32	Not reported
Sundquist et al[121]	Prospective	Sweden	≥65 years Men & women	CHD incidence	Occupation	HR Middle-level/professional employees = 1.00 Manual workers = 1.49 (1.09, 2.03) Lower-level = 1.50 (1.00, 2.23)	HR adjusted for sex, smoking, BMI, physical activity, diabetes and hypertension Manual workers = 1.34 (0.97, 1.83) Lower-level = 1.42 (0.94, 2.13)
Khang et al[36]	Prospective	South Korea	30-64 years Men & women	CHD mortality	Income Low vs. high	55-64 years HR = 1.22 (0.97, 1.53) RD = 38	HR adjusted for smoking, BP = 1.14 (0.91, 1.43) Smoking = 26% relative risk reduction
Copenhagen Male Study[26]	Prospective	Copenhagen, Denmark	53-75 years Men	CHD incidence	Occupation Lower vs. higher social classes	HR = 1.44 (1.10, 1.90)	HR adjusted for age, BP, blood lipids, smoking and physical activity = 1.38 (1.00, 1.90)

SEP=socioeconomic position; ELSA=English Longitudinal Study of Ageing; CI=confidence intervals; HR=hazard ratio; OR=odds ratios; RR=rate ratio; RD=risk difference; BP=blood pressure

Chapter 3

Methods

3.1 Summary

The British Regional Heart Study is a prospective study of cardiovascular disease initiated in 1978-80 in middle-aged men (40-59 years) from 24 towns across Britain. The cohort continues to be followed-up for morbidity through general practice records, and for mortality through the Office of National Statistics General Register Office. Questionnaires at regular intervals during the follow-up have been used to collect information on self-report of health and disease, lifestyle, disability, and personal and socioeconomic conditions. Physical examination including a range of physiological measurements and blood sampling for biochemical measurements was carried out at the start of the study and at a subsequent follow-up 20 years later. This Chapter consists of a description of the British Regional Heart Study with a focus on the methods and data used in the thesis. Further methods for the analyses for each of the results chapters are described in more detail in the relevant chapters.

3.2 Introduction

This Chapter starts with an overview of the British Regional Heart Study (BRHS) with its objectives (section 3.3), study design and methodology (sections 3.4 to 3.7). Section 3.8 provides details of the re-examination of the BRHS men 20 years from baseline. Sections 3.9 and 3.10 describe the measures of socioeconomic position used in the thesis and the representativeness of the BRHS. The Chapter will end with a description of data and statistical methods used in the thesis (section 3.11).

3.3 The British Regional Heart Study

The British Regional Heart Study is a prospective study of a representative sample of 7735 British men, aged 40-59 years at baseline, randomly selected from general practices in 24 towns across Britain in 1978-80. The men have been followed-up since baseline for mortality and morbidity. The British Regional Heart Study was primarily undertaken to explain the substantial regional variation in coronary heart disease and stroke mortality observed in Great Britain.[37;281]

3.3.1 Aims

The aims of the British Regional Heart Study were:

a) to explain the regional variation in cardiovascular mortality in Great Britain;

b) to define the risk factors responsible for cardiovascular disease in individuals;

c) to examine the effect of changes in their levels over time.

While the primary focus was the aetiology and prevention of cardiovascular disease, there has subsequently been an interest in the social patterns of cardiovascular-related disabilities and co-morbidities, clinical prevention and care in this cohort of men as they reached greater ages.

3.4 Selection procedures

3.4.1 Selection of towns

The towns represent all major geographic regions. Seven criteria were used in selecting the towns[281]:

1. All standard regions should be represented.
2. Towns should be discrete entities and with populations of 50,000-100,000 at the 1971 Census. One larger town in England, Ipswich was included. In Scotland, towns with populations less than 50,000 were considered.
3. The choice of towns within region was to reflect the variations in mortality from cardiovascular disease and water hardness.
4. The towns were to be representative of the region in socioeconomic terms.
5. Towns with noticeable movement or with an unusual population structure were avoided.
6. The study included some towns that were apparent "outliers" when cardiovascular disease mortality and water hardness were plotted against each other; for example Hartlepool, Exeter and Harrogate.
7. If similar towns met the above criteria, random selection was made between such towns.

Figure 3.1 shows the 24 towns included in the British Regional Heart Study. The standardised mortality ratios for cardiovascular disease in men aged 35-64 years in

1969-73, number of men examined, and the percentage response rates in the 24 towns are given in Table 3.1.

3.4.2 Selection of practices

It was decided that subjects would be selected from one general practice in each town so as to have a good initial response and a good subsequent follow-up.[281] Criteria for selecting each practice included its size (practice population over 7500 and two or more GP principals), and its representativeness in terms of socioeconomic characteristics and composition of the town. A list of practices for each town was obtained from the District Medical Officer and potential practices were sent information about the BRHS. Based on their interest and willingness to participate, the practices were visited to further assess their representativeness. One general practice was selected from each town and invited to participate.

3.4.3 Selection of participants

In order to have enough men to study risk factors patterns, about 300 men were needed in each town.[281] 450 men aged 40-59 years, stratified into equal sized five-year age groups, were selected at random from the age and sex register in the general practice in each town. An age and sex register had to be constructed in 18 of the 24 practices. The doctors were asked to exclude from this list men who they considered would be unable to participate because of severe physical and mental disability (<10 in each town). An emphasis was laid on not excluding subjects with cardiovascular problems. The remaining subjects were sent invitations to participate in the study through a letter signed by the practice doctors encouraging them to attend the cardiovascular health check at a local venue, usually the practice premises.

3.5 Baseline examination

From almost 10,000 subjects who were invited to participate, 7735 men aged 40-59 years attended the examination and were recruited into the study, a response rate of 78%.[282] At the start of the study in 1978, detailed examination of the men was carried out in each of the towns by a team of three nurses and a questionnaire was also administered. Ethical approval was provided by all relevant local research ethics committees. All men provided written informed consent to the investigations carried out in accordance with the Declaration of Helsinki. The examination of all men in the 24 towns was completed by 1980. The nurses were provided with training before and during the study to ensure standardisation of procedures including administration of questionnaires. The response rate averaged 78% and ranged from 70-85% (Table 3.1). A questionnaire was administered by a nurse to each participant as part of the baseline examination. Information was collected on date and place of birth, medical and family history, present and past occupations, medication use, and lifestyle. The examination included physical measurements, a resting ECG and collection of a non-fasting blood sample.

3.6 Follow-up from baseline

The cohort has been followed-up since study entry in 1978-80 for mortality and morbidity outcomes and through regular postal questionnaires, as shown in Figure 3.2 (page 97).

3.6.1 Mortality

Details of the study participants were sent to the National Health Service Central Register (NHSCR) in Southport for England and Wales and in Edinburgh for Scotland, to identify and tag the subjects. Information on mortality was collected through the established procedure of 'flagging' research participants. At three-monthly intervals, the Central Register sent death certificates containing identification details, date and place of death, and cause of death coded to the International Classification of Disease 9th Revision (ICD-9) and subsequently to the 10th Revision (ICD-10). Deaths from ischaemic heart disease with ICD-9 codes 410-414 (equivalent to ICD 10th revision codes I20-I25) were defined as a fatal coronary heart disease event. Information on deaths was also sent by the general practice as part of a periodic review (see below), which ensured that all events were verified.

3.6.2 Morbidity

In each general practice of the 24 towns a practice coordinator usually a receptionist, was identified to liaise with the study centre. Every two years during the follow-up of the cohort, the practice coordinator was sent a list of the study participants to carry out a review of the medical records. The practice coordinator updated the list with known deaths and emigrations, with the date of events and information on movement from the practice. For the remaining men, a systematic check of each set of medical records (including hospital and clinic correspondence) was carried out by the coordinator for newly diagnosed cardiovascular events (myocardial infarction, angina, stroke and transient ischaemic attack) in the preceding two years. Confirmation of each subject's current address was also provided. Since 1986, information has also been collected on diabetes and on treatments not available at the onset of the study e.g. coronary artery bypass graft, percutaneous transluminal coronary angioplasty). All newly reported non-fatal myocardial infarction events are followed-up with an enquiry form to the general practitioner or consultant to obtain evidence of prolonged chest pain, positive electrocardiogram findings and raised cardiac enzyme levels. In accordance with World

Health Organisation criteria,[283;284] any two of these three conditions were to be met for an event to be accepted as a case of definite myocardial infarction.

3.6.3 Change of practice and tracing procedures

Through the two-yearly review, subjects who had moved from a town or beyond the boundary of the original practice were identified. Information on the new registration area or general practitioner was obtained from the local health authority. Contact was then established with the new general practice, which was requested to complete an enquiry form with the same information as that supplied by the original practices on the review sheets. If no information was available from the local health authority, the NHSCR was contacted. Information on emigrations and re-instatements was notified by the NHSCR. However, if any subject had emigrated or died abroad no death certificate was readily available.

3.7 Follow-up questionnaires

Questionnaires have been sent to the study participants at regular intervals during follow-up (see Figure 3.2 on page 97). The first postal self-administered questionnaire was sent out five years after the start of the study in 1983-85, followed by questionnaires in 1992 and 1996. In 1998-2000 the study participants were invited for a re-examination, when a nurse-administered questionnaire was completed. This was followed by postal questionnaires in 2003, 2005 and most recently in 2007. For the purpose of this thesis, questionnaire data from baseline and thereafter from 1992 till 2003 have been included and details of these questionnaires are described below. The response rates to questionnaires in 1992, 1996, 1998-2000 and 2003 were 91%, 88%, 78% and 80% respectively. The questionnaires collected information on medical history and treatment use, health status, lifestyle, and socioeconomic factors as described below.

3.7.1 Medical history

On each occasion, the subjects were asked if they had been told by a doctor that they have had heart attack (or coronary thrombosis or myocardial infarction), angina, ‘other heart trouble’, high blood pressure, stroke and diabetes. The questionnaires in 1992, 1996 and 2003 included peptic ulcer, gout, gall bladder disease, thyroid disease, arthritis, bronchitis and asthma. Heart failure, aortic aneurysm, narrowing/hardening of leg arteries, deep vein thrombosis and pulmonary embolism were included in the 1998-2000 and 2003 questionnaires. Subjects were also asked if they were taking regular medical treatment including antihypertensive drugs, anti-coagulants, lipid lowering drugs, insulin and oral drugs for diabetes. Medications were coded according to the British National Formulary schedule. Subjects were asked if they had undergone cardiological investigations and invasive treatments including exercise ECG, angiogram, angioplasty (‘balloon treatment’) and coronary artery bypass graft (CABG) operation.

3.7.2 Health status and disability

Starting from 1985, all subsequent questionnaires were used to ask subjects to describe their health as **excellent**, **good**, **fair** or **poor**. In the questionnaires sent in 1998-2000 and in 2003, mobility limitation was determined by asking subjects whether they currently had difficulty carrying out any of the following activities on their own as a result of a long term health problem: difficulty going up or down stairs, difficulty bending/straightening up, difficulty maintaining balance, difficulty walking for a quarter of a mile on the level. Additional questions on ascertaining problems with activities of daily living and instrumental activities of daily living were asked in 2003 to capture the extent of disability.

3.7.3 Smoking

Detailed questions were asked in each questionnaire about number of cigarettes smoked and changes in smoking habits. In 1992, subjects were grouped into four categories: never smokers, long-term ex-smokers (current non-smokers at 1992 and ex-smokers at baseline), recent ex-smokers (current non-smokers at 1992 and given up since baseline), and current smokers at 1992. In 1998-2000, subjects were categorised into six groups: never smoker, long-term ex-smoker (since baseline, more than 20 years), gave up smoking 15-20 years back, gave up smoking 10-15 years ago, gave up smoking 5-10 years ago, recent ex-smoker (gave up within last 5 years), and current smoker.

3.7.4 Alcohol consumption

In each questionnaire, alcohol consumption was recorded using questions on the frequency and quantity of alcohol intake, similar to those used in the 1978 General Household Survey.[285;286] The men were initially classified as: non-drinkers, men drinking on special occasions or 1-2 drinks per month), weekend drinkers (1-2, 3-6 or >6 drinks per day) and men drinking daily or on most days (1-2, 3-6 or >6 drinks per day).[285] Based on this information using the estimated weekly alcohol intake, alcohol consumption was categorised into five groups of: none, occasional (<1 unit/week), light (1-15 units/week, which included weekend 1-2, 3-6 and daily 1-2), moderate (16-42 units/week, which included daily 3-6 and weekend >6) and heavy (>42 units/week, which included >6 daily).[285] One drink was defined as half a pint of beer, a glass of wine, or a tot of spirit (8-10gms).

3.7.5 Physical activity

Information on physical activity was collected in each questionnaire. The men were asked to indicate their usual patterns of physical activity under the headings of regular walking or cycling, recreational activity and sporting (vigorous) activity. Regular walking and cycling related to weekday journeys, which included travel to and from work. Recreational activity included gardening, pleasure walking, and do-it-yourself jobs. Sporting activity included running, golf, swimming, tennis, sailing, and digging. A physical activity score was derived for each man according to the frequency and type (intensity) of physical activity.[162] Scores were assigned for each type of activity and duration on the basis of the intensity and energy demands of the activities reported. The total score for each man is not a measure of total time spent in physical activity but rather is a relative measure of how much physical activity has been carried out. Based on this score the men were grouped into six categories of a physical activity index: inactive (score 0-2), occasional (score 3-5; regular walking or recreational activity only), light (score 6-8; more frequent recreational activities or vigorous exercise less than once a week), moderate (score 9-12; cycling or very frequent recreational activities or sporting activity once a week), moderately-vigorous (score 13-20; sporting activity at least once a week or frequent cycling, plus frequent recreational activities or walking, or frequent sporting activity only), and vigorous (score ≥21; very frequent sporting exercise or frequent sporting exercise plus other recreational activities). The physical activity score was validated at both the baseline examination and the re-screening in 1998-2000 by using heart rate and forced expiratory volume in 1 second (FEV_1) in men free of pre-existing CHD.[43;162] Mean heart rate decreased and FEV_1 increased significantly with increasing levels of physical activity even after adjustment for potential confounders.

3.7.6 Dietary intake

In 1998-2000, a separate self-administered detailed standardised 7 day recall food-frequency questionnaire developed for use in the World Health Organization's Monitoring Trends and Determinants in Cardiovascular Disease Survey[287] and later for the Scottish Heart Health Study,[288] was used to collect information on dietary intake and patterns. Study participants were asked to recall their usual intake during the past 7 days by reporting amounts and frequency of food consumed, which included 86 different foods and drinks. Nutrient intakes were calculated by using a validated program that multiplied the food frequency by the standard portion sizes for each food and by the nutrient composition of the foods obtained from the UK food composition tables.[289] Carbohydrate and dietary fat intakes were expressed as percentages of total energy intake.

3.8 Twenty-year re-examination

After 20 years of follow-up, the study participants (now aged 60-79 years) were invited to attend a re-screening, which took place between 1998-2000 and comprised physical examination and completion of a questionnaire at a local health centre.[290] The men were requested to fast for a minimum of 6 hours, during which time they were instructed to drink only water, and to then attend a measurement session at a specified time between 0800 and 2000 hours. Men with diabetes who were taking insulin or oral hypoglycaemic treatment were instructed to eat and drink normally. All men were asked to provide a blood sample, which was collected by using the Sarstedt Monovette system (Sarstedt, Numbrecht, Germany).[290] Of the 5565 surviving men 4252 (77%) attended the re-examination. Details of the re-examination are described below.

3.8.1 Physical examination

Physical examination at the re-screening in 1998-2000 included anthropometric and physiological measurements. Anthropometric variables measured at the re-examination included height, weight, waist and hip circumferences, percentage body fat, and fat mass.[290] Subjects were measured in light clothing without shoes. Height and weight were both measured while the subjects were standing. Height was measured with a Harpenden stadiometer (Critikon Service Center, Berkshire, United Kingdom) to the last complete 0.1 cm and weight with a Soehnle digital electronic scale (Critikon Service Center) to the last complete 0.1 kg. Body mass index (BMI) was calculated for each man as weight/(height)^2 in kg/m^2. BMI was not available for 10 men. Waist and hip circumferences were measured in duplicate with an insertion tape (CMS Ltd, London, United Kingdom). Hip circumference was measured at the point of maximum circumference over the buttocks. Waist measurement was taken from the midpoint between the iliac crest and the lower ribs measured at the sides. Within-subject variation for waist circumference and BMI was examined in a repeatability study of 110 subjects measured by the same team of observers on both occasions. The correlations between measurements taken 1 week apart were 0.995 for BMI and 0.992 for waist circumference.[290]

Blood pressure was measured twice in the right arm with a Dinmap 1846 oscillometric blood pressure recorder with the subject seated and the arm supported. Systolic blood pressure (Dinamap reading) was adjusted by subtracting 8 mmHg from the reading to accord with the Hawksley random zero sphygmanometer readings at baseline.[291] Blood pressure was adjusted for observer variation within each town.[292]

3.8.2 Blood measurements

From the blood samples collected at the re-screening in 1998-2000, plasma and serum samples were centrifuged, separated and frozen at –20 degrees C within 6 hours on the day of collection and transferred to central laboratories for analysis. Blood lipids and glucose were analysed at the Department of Chemical Pathology, Royal Free Hospital (Prof. A. Winder, Dr M. Thomas), and insulin was measured at the Department of Diabetes and Metabolism, University of Newcastle (Prof. KGMM Alberti, Ms P. Shearing). Serum total and high-density lipoprotein (HDL) cholesterol, and triglycerides were measured using a Hitachi 747 automated analyser (Hitachi, Tokyo, Japan).[293] Total and HDL cholesterol were analysed using the Siedel et al[294] and Sugiuchi et al[295] methods respectively. Low-density lipoprotein (LDL) cholesterol was calculated using the Fredrickson-Friedwald equation.[296] Plasma glucose was measured by a glucose oxidase method with a Falcor 600 (A Menarini Diagnostics, Wokingham, United Kingdom) automated analyser.[297] Serum insulin was measured using a Drew Hb Gold HPLC analyser (Drew Scientific Group Plc, Barrow in Furness, UK). LDL-cholesterol, triglycerides, glucose, and insulin concentrations were adjusted for the effects of fasting duration and time of day.[293]

Haemostatic and inflammatory markers were measured in citrated blood plasma at the Department of Medicine, University of Glasgow (Prof. GDO Lowe, Dr A Rumley). Blood was anticoagulated with K_2 EDTA (1.5 mg/ml) for measurement of haematocrit, white blood cell (WBC) count, and platelet count in an automated cell counter; and plasma viscosity at 37°C in a semi-automated capillary viscometer (Coulter Electronics, Luton, UK). Blood was also anticoagulated with 0.109 M trisodium citrate (9:1 v/v) for measurement of clottable fibrinogen (Clauss method) as well as coagulation factor VIII, activated partial thromboplastin time and activated protein C (APC) ratio in an MDA-180 coagulometer (Organon Teknika, Cambridge, UK). Plasma levels of tissue plasminogen activator antigen (t-PA) and fibrin D-dimer were measured with enzyme-linked immunosorbent assays (Biopool AB, Umea, Sweden) as was von Willebrand factor antigen (DAKO, High Wycombe, UK). C-reactive protein was assayed by ultra-sensitive nephelometry (Dade Behring, Milton Keynes, UK). Interleukin-6 was assayed by a high-sensitivity ELISA (R and D Systems, Oxford, UK).

Plasma vitamin C was measured with HPLC that included ultraviolet and fluorescent detection[298;299] by using extracts of plasma treated with metaphosphoric acid at the point of collection and then snap-frozen with dry ice. Laboratory-blinded split samples were used to ensure quality control throughout the study.

3.9 Socioeconomic position

Several measures reflecting the socioeconomic circumstances of the subjects were collected in the study including occupational social class (adult and childhood), education, house and car ownership in adult life, pension arrangements, and childhood household amenities. The choice and use of these measures is described below.

3.9.1 Adult social class

The longest-held occupation of subjects was asked at baseline when they were middle-aged (40-59 years). The Registrar General's Classification of Occupations[300] was used to classify the subjects into six social class categories (I, II, III non-manual, III manual, IV, and V; see Table 3.2). The social class distribution of the subjects aged 40-59 years is presented in Table 3.3. Nearly all subjects (99.81%) except 15 men were allocated a social class. Of these subjects, 231 (3%) subjects in the Armed Forces were grouped into a separate category and were excluded from all the analyses. The occupation of the men was recorded again in the twenty-year follow-up questionnaire (1998-2000) when subjects were aged 60-79 years. The subjects were again classified into six social class categories based on the Registrar General's Classification of Occupations. Table 3.4 shows that some movement between social class groups had occurred from baseline to the twenty-year follow-up.

To enable an overall comparison of change in social class groups, Table 3.4 shows the number of men in non-manual and manual groups at baseline and at the 20-year follow-up [social classes I, II, III non-manual were grouped as 'non-manual' and social classes III manual, IV, V as 'manual'. A detailed social class distribution of subjects at the twenty-year follow-up according to social class measured at baseline is presented in Appendix I (page 255)]. At the twenty-year follow-up, most of the non-manual and manual social class groups remained within the same group, 86% and 83% respectively (Table 3.4). Only 9% of the subjects had changed their social class status over the twenty-year period, which confirms the stability of social class measured at baseline (estimated from $\tau = 0.5 - 0.5\sqrt{(N-2n)/N}$; where N = individuals with social class measure at baseline and 20-year follow-up and n = disagreements in social class measurements at the two time points).[28;301]

Thus, the longest-held occupation was confirmed to be a stable marker of social class, and is also likely to fulfil the criterion of having a single measure of adult social class, which would act as a reference point over the entire study period. Moreover, since social class at baseline was based on the longest-held occupation of the subjects when aged 40-59 years, it was considered to most adequately reflect the socioeconomic position of the subjects for most of their adult life. Therefore, longest-held occupation was used as the main measure of adult socioeconomic position in the thesis to investigate how socioeconomic inequalities in CHD changed with increasing age and to examine if these inequalities persisted in older age. The current or last occupation held just before retirement was not used in the thesis because it may not be representative of socioeconomic position over most of adult life.

In most of the analyses, the full Registrar General's classification of social classes from I to V was used in this thesis. However, to enable an overall comparison of social class groups, the dichotomous categories of non-manual (social classes I, II, III non-manual) and manual (social classes III manual, IV, V) were also adopted. This was used in Chapter 4 to describe the overall trend in socioeconomic inequalities in CHD mortality over 25 years. In Chapter 8, where the analyses was restricted to men with CHD, social classes I+II, and IV+V were combined due to a small number of men in social classes I and V.

3.9.2 Other socioeconomic measures

Besides adult social class, information was collected on other measures of socioeconomic position. Questions on house and car ownership in adult life were asked in the 1992, 1996, 2000 and 2003 questionnaires. Age at leaving full time education was asked in 1992. Information on pension arrangements was collected in the 1996 questionnaire.

3.9.3 Childhood socioeconomic position

In the questionnaire in 1992, information on childhood socioeconomic position was collected. The subjects were asked about the kind of job their father had done for the longest period of his (father's) life. Registrar General's Classification of Occupations 1931 (close to the mid-year of birth of study participants) was used to classify subjects into different childhood social class groups (I, II, III, IV, and V; see Table 3.5).[108;302] The questionnaire in 1992 also collected information on childhood household amenities (bathroom in the house, hot water supply and family car ownership). This information allowed a more detailed assessment of early life socioeconomic position.

3.10 Representativeness of the study participants

The social class distribution of the study participants compared with that of men of a comparable age (45-64 years) from the 1981 census in Great Britain was found to be similar (see Table 3.6).[303] This confirms the representativeness of the study participants from the 24 towns in Britain. In the study participants, there was some under-representation of social classes III non-manual, IV and V, and an over-representation of I and III manual social classes. However, the proportion of men in non-manual and manual social class groups in the study participants was similar to that from the census data (approximately 40:60 in both groups).[303] The geographically and socially representative nature of the study population allows the results from the British Regional Heart Study to be generalised to middle-aged British men. However, since the study sample is derived from medium-sized British towns with less mobile populations, the cohort almost entirely comprises white European men and has little information on other ethnic groups. Although the study also lacks populations from major conurbations or from rural areas, all major regions of Britain (England, Scotland and Wales) are represented in the study.

Characteristics of participants at the start of the study have been compared to those of the non-responders.[303] Men who did not participate in the study were younger and more likely to have less skilled occupations compared to study participants. Non-participants also had a higher risk of death but this was observed only in the first three years of follow-up and declined subsequently.[303] Death rates by different causes of death were similar in magnitude particularly for cardiovascular disease and neoplasms, which suggests that analyses based on these causes of death are not likely to be biased due to factors related to non-participants.

3.11 Data and methods used in this thesis

Data for this thesis includes mortality data obtained from NHSCR, morbidity data from record reviews, data on physical re-examination in 1998-2000 and regular questionnaires at baseline (1978-80), 1992, 1996, 1998-2000 and 2003.

3.11.1 Outcomes

The particular focus of this thesis is on morbidity and mortality related to coronary heart disease (CHD). The main outcome of the thesis is the development of myocardial infarction (fatal and non-fatal events) and CHD mortality has been used as a key outcome throughout. In Chapter 4, trends in socioeconomic inequalities over 25 years from baseline (1978-80) to 2005 were investigated using CHD mortality and total mortality. The relationship of adult socioeconomic position and childhood socioeconomic position to CHD was examined in Chapters 6 and 7 using myocardial infarction (non-fatal and fatal) and CHD deaths as outcomes. Chapters 5 and 8 do not use CHD outcomes. Chapter 5 describes the associations between adult social class and coronary risk factors in older age measured at the re-examination in 1998-2000 when the men were aged 60-79 years. As an exception, the last results Chapter (8) presents socioeconomic inequalities in disability in older men with CHD as an indicator of the overall extent of health inequalities in those with CHD in old age.

3.11.2 Statistical methods

While the methods used for statistical analyses are specified in subsequent Chapters, some of the main statistical methods used are described below.

3.11.2.1 Survival analysis and Cox proportional hazards regression analysis

Survival analysis was used to investigate the probability of having an event, when the time taken to develop that event, called survival time, is known.[304] The probability of survival can be calculated by using the Kaplan Meier method when the exact time of an event occurring is known. The probability of survival is calculated each time an event occurs and is plotted to obtain a Kaplan Meier survival curve. Observations or subjects in whom the event has not occurred, are lost to follow-up, die of a cause other than the event of interest, or if the time of event occurring is not known, are taken to be 'censored' observations. The survival time for censored observations is the time that they are no longer in the study.

Cox proportional hazards regression analysis is used to investigate the effects of several variables on survival. It is a semi-parametric method, which assumes that the effect of different variables on survival is constant over time. The model is based on the hazard function $h(t)$, representing the risk of dying at time t, assuming survival till time t. With different independent variables from X_1 to X_p and their regression coefficients b_1 to b_p, $h(t)$ is,

$$h(t) = h_0(t) \times \exp(b_1 X_1 + b_2 X_2 + ... + b_p X_p)$$

$h_0(t)$ can be estimated from the data and is the baseline hazard function when all the variables are zero.

$h(t)$ is the hazard or risk of dying at time t, so adding up all the hazards up to time t is the cumulative hazard, H(t).

$$H(t) = H_0(t) \times \exp(b_1 X_1 + b_2 X_2 + ... + b_p X_p)$$

where $H_0(t)$ is the cumulative baseline hazard function.

The probability of survival to time, t, is $S(t) = \exp[-H(t)]$.

A hazard ratio comparing the hazards for two different values (x_1 and x_2) of a covariate can be calculated as,

$h_1(t)/ h_2(t) = h_0(t) \times \exp(bx_1)/ h_0(t) \times \exp(bx_2) = \exp(bx_1 - bx_2) = \exp[(b(x_1-x_2)]$.

95% confidence interval for log hazard ratio = $b \pm (1.96 * \text{standard error of } b)$

3.11.2.2 Generalised linear models

These methods are applied to analyse the relationship between a dependent variable and one or more independent variables.[304] Linear regression is used when the dependent variable (Y) is continuous and is related to independent variables (X_1, X_2, … X_n) as,

$Y = a + b_1X_1 + b_2X_2 + … + b_nX_n$

b_1 *to* b_n is the regression coefficient, a is the intercept which is the value of Y for which $X = 0$.

Logistic regression analysis is used to relate a dichotomous dependent variable to a set of explanatory variables. A transformation of the probability of the outcome is carried out, called logit transformation, logit(p) to restrict the probability of outcome from 0 to 1.

logit(p) = log (p/1-p) = $a + b_1x_1 + b_2x_2 + \ldots b_nx_n$

where p = probability of the outcome; 1-p = probability of not having the outcome; $b_1 \ldots b_n$ are regression coefficients for explanatory variables ($x_1 \ldots x_n$).

p/1-p is called the odds and logit(p) is the log odds. The odds (p_1 and p_2) in two groups of an independent variable (say x_i, with values of 1 and 0) can be compared to obtain the log odds ratio as,

Log odds ratio = logit(p_1) – logit(p_2) = $(a + b_1x_1 + b_2x_2 + \ldots + b_ix_i) - (a + b_1x_1 + b_2x_2)$

$$= b_i$$

Odds ratio = exp(b_i); 95% confidence interval for log odds ratio = $b \pm$ (1.96 * standard error of b)

3.11.2.3 Analysis of variance

Analysis of variance (ANOVA) is a statistical method used to compare the difference in mean levels of a continuous dependent variable according to groups of a categorical independent variable.[304] ANOVA involves calculation of the overall variation (total sum of squares), variation between groups (between-group sum of squares is the sum of squares of the difference between mean of each group and overall mean), and variation within groups (within-group sum of squares is the sum of squares of the difference between mean of each observation and the mean of its relevant group). According to the null hypothesis, all groups have the same mean and there is no difference in between-groups and within-group variance. F distribution is used to compare the variances between and within groups. ANOVA can be extended to include more than one independent variable, to control or adjusted for other variables. This method is called Analysis of Covariance (ANCOVA).

3.11.2.4 Bootstrap resampling

The bootstrap is a data-based simulation method for assessing statistical precision.[305] It is used when the sampling distribution of an estimator is not known and therefore, classical methods of statistical analysis cannot be used.[305;306] Samples of the same size as the original are drawn, by sampling with replacement from the observed data. The number of samples required depends on the measure of interest; 1000 are recommended to obtain a bootstrap confidence interval. The statistic of interest (for example difference in survival rates) is calculated for each resample. The distribution of these values is used to estimate the underlying distribution. The approximate confidence intervals is given by the 100 (α/2) and 100(1 - α/2) percentiles of the distribution, so that a 95% confidence interval is given by the range between the 2.5th and 97.5th percentiles. However, the bootstrap distribution of the statistic may not be accurate; the estimate of the statistic from the original data may differ from the median of the estimated values from the bootstrap sample. The difference should then be added to the percentiles to give *bias-corrected percentiles*.

Figure 3.1 Map of Great Britain showing the 24 towns of the British Regional Heart Study

Figure 3.2 Follow-up in the British Regional Heart Study

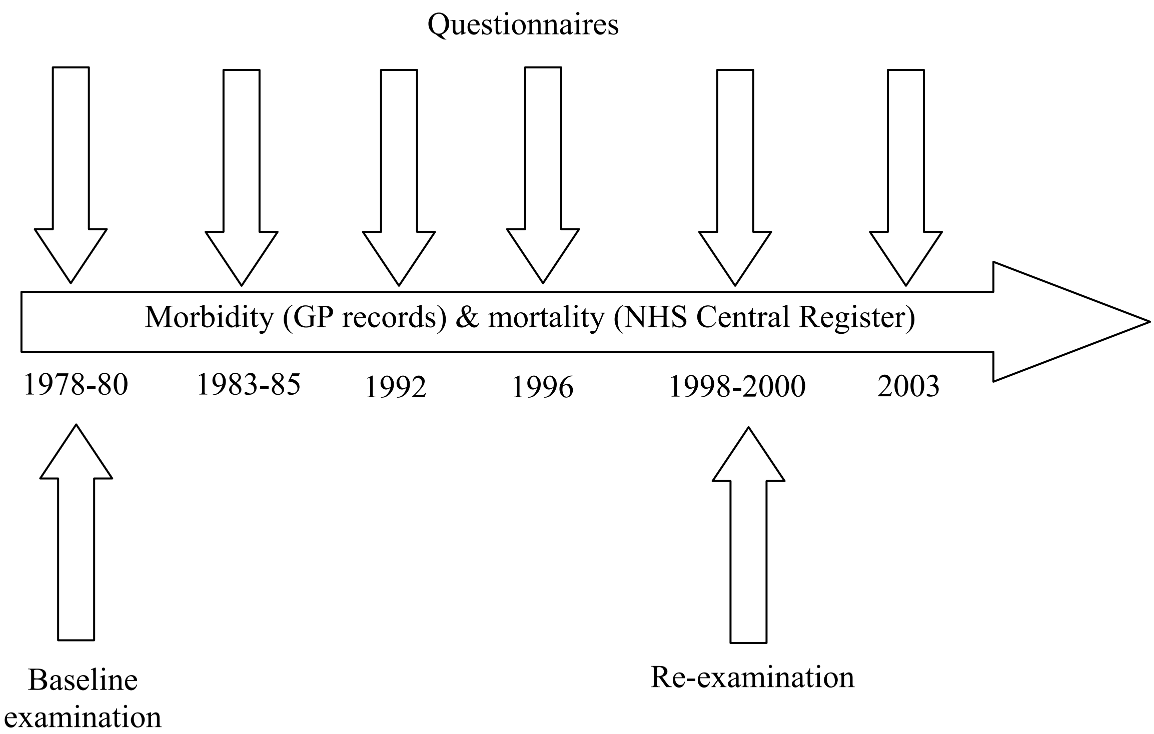


Table 3.1 Towns in the British Regional Heart Study

Town	SMR for cardiovascular disease in men aged 35-64 years in 1969-73	Number of men examined	Response rate (%)
Ayr	140	301	70
Bedford	80	303	73
Burnley	114	286	80
Carlisle	121	389	85
Darlington	109	382	82
Dewsbury	142	326	79
Dunfermline	118	350	80
Exeter	90	332	84
Falkirk	98	308	75
Gloucester	84	309	73
Grimbsy	96	318	71
Guildford	78	335	82
Harrogate	82	280	77
Hartlepool	101	334	70
Ipswich	92	362	85
Lowestoft	85	324	83
Maidstone	99	319	72
Mansfield	95	321	80
Merthyr Tydfil	135	282	76
Newcastle-upon-Lyme	115	293	77
Scunthorpe	109	313	76
Shrewsbury	95	310	83
Southport	114	322	80
Wigan	134	337	77

SMR= standardised mortality ratio

Data source: Shaper AG et al. British Regional Heart Study: cardiovascular risk factors in middle-aged men in 24 towns. BMJ 1981; 283:179-186

Table 3.2 Registrar General's Classification of Occupations 1980

Social class	Description	Examples of occupations
I	Professional occupations	Barristers, physicians, engineers
II	Intermediate occupations	Teachers, sales managers
III non-manual	Skilled non-manual occupations	Clerks, shop assistants
III manual	Skilled manual occupations	Bricklayers, coalminers
IV	Partly skilled occupations	Bus conductors, postmen
V	Unskilled occupations	Porters, general labourers

Table 3.3 Social class distribution of men in the British Regional Heart Study aged 40-59 years in 1978-80

Social class based on longest-held occupation recorded at 40-59 years	**n (%)**
I	606 (8)
II	1735 (23)
III non-manual	720 (10)
III manual	3326 (44)
IV	784 (10)
V	318 (4)

Table 3.4 Comparing number of subjects in non-manual and manual social class groups at baseline (1978-80) and at twenty-year follow-up (1998-2000)

Social class at baseline	**Social class at follow-up in 1998-2000**		
	Non-manual	Manual	Total
Non-manual	1624 (86%)	269 (14%)	1893 (100%)
Manual	346 (17%)	1684 (83%)	2030 (100%)
Total	1970	1953	3923

Table 3.5 Registrar General's Classification of Occupations 1931

Social class	Description	Examples of occupation
I	Professional occupations	Engineers, physicians, clergymen, bankers
II	Intermediate occupations	Farmers, coal mine owners/managers,
III	Skilled occupations	Gardeners, farm or factory foremen,
IV	Partly skilled occupations	Shepherds, fishermen, miners, quarriers
V	Unskilled occupations	Masons or builders' labourers, porters, messengers

Table 3.6 Social class distribution (%) of participants in the British Regional Heart Study compared with the 1981 Census data

Social class	National Census 1981		24 towns Census 1981		BRHS participants 1978-80	
I	5.2		5.1		7.8	
II	23.8	39.5	23.2	41.5	22.5	39.6
III non-manual	10.5		13.2		9.3	
III manual	33.9		33.7		43.0	
IV	17.8	58.1	16.6	56.1	10.3	57.4
V	6.4		5.8		4.1	
Armed Forces	2.4		2.4		3.0	

Data source: Walker M et al. Non-participation and mortality in a prospective study of cardiovascular disease. J Epidemiol Community Health 1987; 41:295-299

Chapter 4

Trends in socioeconomic inequalities in coronary heart disease mortality in Britain from 1978 to 2005

4.1 Summary

Earlier studies have suggested that socioeconomic inequalities in life expectancy may have widened in Britain in 1980s and 1990s. In this Chapter, socioeconomic inequalities in coronary heart disease (CHD) mortality and all-cause mortality in British men were examined between 1978 and 2005, to investigate whether these inequalities had changed over time and with increasing age. All subjects in the British Regional Heart Study were followed-up from baseline (1978-80) until 2005 for mortality from CHD and all-causes. Relative hazards and absolute risk differences for CHD and all-cause deaths comparing manual with non-manual social classes were calculated, with subjects divided into four 5-year age groups and five 5-year calendar periods. Mortality rates from CHD and from all causes declined over the 25-year period. Risks of mortality from CHD and all-causes were persistently higher in manual social class groups compared to non-manual throughout the 25-year follow-up. With increasing age, the relative difference in mortality between manual and non-manual groups narrowed. However, the relative difference between these social class groups tended to increase over time. The overall relative increase in hazard ratio comparing manual to non-manual groups over a 20-year calendar period was 1.75 (95%CI 0.89, 3.45, p=0.11) for CHD mortality and 1.22 (95%CI 0.83, 1.80, p=0.31) for all-cause mortality. However, the absolute difference in probability of survival to age 65 between non-manual and manual groups fell from

17% to 7% for CHD mortality and from 29% in 1981 to 9% in 2001 for all-cause mortality. Relative differences in CHD and all-cause mortality between manual and non-manual social class groups persisted and may have increased during this period. However, absolute differences in mortality between these social class groups decreased because of falling overall mortality rates. Greater efforts are needed if socioeconomic inequalities in CHD mortality are to be reduced in the new millennium.

4.2 Introduction

Marked socioeconomic inequalities in health and mortality in the UK have been present for many years.[69] The Independent Inquiry into Inequalities in Health[34] summarised evidence that socioeconomic inequalities were persisting during the 1990s. There has been concern that socioeconomic inequalities in mortality and life expectancy have been increasing rather than declining during recent years.[72;76;113;307;308] Though studies in Britain have reported on inequalities in mortality or in life expectancy,[72;76;307;308] there is limited evidence on recent trends in socioeconomic inequalities in coronary heart disease (CHD) mortality. More evidence on the secular changes in the direction and extent of socioeconomic inequalities in CHD is needed so as to enable policies to address these appropriately. Socioeconomic inequalities in health have also been extensively described in middle age, in relation to occupation.[19] However, there is uncertainty about how inequalities related to occupational social class change with increasing age.

4.3 Objectives

The aim of this Chapter is to examine the extent of socioeconomic inequalities, based on occupation, in CHD mortality (the single most important cause of death) and all-cause mortality among British men followed up from 1978-80 for a 25-year period. The objectives were two-fold:

i) To examine the size of social class differences (relative and absolute) in CHD mortality and all-cause mortality with increasing age.

ii) To investigate whether relative social class differences in CHD and all-cause mortality have changed over time (1978-80 to 2005), independent of age. The absolute differences in mortality between social classes and changes over time were also examined, since mortality rates (from CHD and all causes) had declined during the study period, both in the whole population[7] and in this cohort.[309]

4.4 Methods

Data on mortality on BRHS men were obtained by the established procedure of 'flagging' participants with the NHS Central Register. The period of follow-up used for this Chapter was from 1978-80, when the participants were enrolled in the study, up to 31st October 2005. The longest-held occupation of each man was recorded at the study entry and categorised using the Registrar General's Social Class Classification (I, II, III non-manual, III manual, IV and V).[300] Cause of death was coded from death certificates using the *International classification of diseases*, 9th revision (ICD-9). CHD deaths were those with ICD-9 code 410–414.

4.4.1 Rationale for analyses

Trends in socioeconomic inequalities in CHD and all-cause mortality were examined from 1978-80 to 2005 (164,120 person years). These inequalities were examined both with increasing age and over the follow-up period, independent of age. The extent of the changes in socioeconomic inequalities in CHD and all-cause mortality was assessed in relative as well as absolute terms. Trends in relative socioeconomic inequalities would indicate the strength of the relationship between socioeconomic position and mortality (all-cause and CHD) over the study period. Absolute socioeconomic differences in inequalities would reflect the change in the magnitude of these inequalities over time and with increasing age. Adult social class based on the longest-held occupation was used as the measure of socioeconomic position. Social classes I, II and III non-manual were grouped as 'non-manual' while social classes III manual, IV and V were grouped as 'manual' to provide a single overall summary of socioeconomic inequalities and their trends.

4.4.2 Statistical methods

4.4.2.1 Relative social class differences in mortality

All analyses were carried out using SAS version 8, with the exception of analyses examining social class*age and social class*period interactions, carried out with STATA version 9. Survival analysis was carried out and Kaplan Meier survival curves were plotted to investigate whether the probability of survival from CHD and all-cause mortality differed according to social class (I, II, III non-manual, III manual, IV and V). Men in the Armed Forces (n=231) were not included in the analyses. Cox's proportional hazard models were used to assess the relation between social class and CHD and all-cause mortality. The models were adjusted for age, which was fitted as a continuous variable.

The follow-up period was truncated at 25 years and divided into five equal calendar periods starting from the baseline period of 1978-80: 0-5 years (1978-80 to 1983-85), 5-10 years (1983-85 to 1988-90), 10-15 years (1988-90 to 1993-95), 15-20 years (1993-95 to 1998-2000) and 20-25 years (1998-2000 to 2003-05). Age at baseline was divided into four groups of 40-44, 45-49, 50-54 and 55-59. Overall hazard ratios with 95%CI for all-cause and CHD mortality comparing manual with non-manual groups were calculated for the four age groups and for the five time periods. Age-adjusted hazard ratios were also calculated for each age group within each time period. Cox models included effects of age, period effect, social class, social class*age interaction (to ascertain whether social class effects changed as subjects aged), and social class*period interaction (to ascertain whether the social class effect changed over calendar time). The social class*age estimate from the Cox model was used to calculate the change in hazard ratio over a 20-year increase in age. The social class*period estimate was used to calculate the change in hazard ratio over a 20-year calendar period. Because of the sampling structure of the study where men were chosen from within towns, robust standard errors were calculated which adjusted for the clustering of responses by men within towns.[310]

4.4.2.2 Absolute social class differences in mortality

Rates of death from CHD and all-cause mortality were estimated in all men, and separately in manual and non-manual groups, to ascertain the absolute difference in survival between these groups. This was done using the same age groups and calendar periods defined for looking at relative differences described above. In particular, the probability of survival to age 65 years in non-manual and manual groups for conditions prevailing in 1981 and 2001 was calculated. Different values for social class (non-manual and manual) and period (1981 and 2001) were chosen to calculate cumulative hazard functions, and thus survival probability, for these particular social class/period combinations.[311] Survival probability was calculated as:

$S(t, x) = \exp[-H(t, x)]$,

where H was the cumulative hazard function; x represented social class, period, and social class*period interaction effects estimated from the appropriate Cox proportional hazards model; and t represented age. Crude survival rates were estimated for every year of age from 40 years (age of youngest cohort members at the beginning of follow-up) to age 65 year. For each year of age, data used included every subject who passed through that year of age. These crude estimates were then added together to give a cumulative hazard function from age 40 years.

Uncertainty associated with these modelled estimates of survival probability was addressed by taking 1000 bootstrap samples, using the bias-corrected method for obtaining 95% confidence intervals,[306] as described in section 3.11.2.4 on page 95.

4.5 Results

Analyses are based on 7489 men aged 40-59 years at entry to the study, followed for 25 years (158,993 person years at risk) to age 65-84 years. During this period of follow-up, 2910 deaths occurred from all causes, of which 969 were attributed to CHD. Figure 4.1 and Figure 4.2 present Kaplan Meier survival curves according to social class groups for all-cause and CHD mortality respectively. The survival curves show the social class variations in all-cause and CHD mortality - the probability of survival after 25 years from all-causes decreased from 0.72 in social class I to 0.51 in social class V; survival probability for CHD mortality after 25 years was 0.89 in social class I and 0.82 in social class V. The relationship of social class and mortality is also demonstrated in Table 4.1, which shows age-adjusted hazard ratios for all-cause and CHD mortality according to different social classes. The hazard ratios for both all-cause mortality and CHD mortality increased from social class I and was highest in social class V. The hazard ratios comparing men of manual with non-manual social class were very similar for CHD mortality (1.50; 95%CI 1.31, 1.72) and all-cause mortality (1.52; 95%CI 1.41, 1.64).

Table 4.2, Table 4.3 and Table 4.4 present estimates for CHD mortality and overall mortality for each five year time-period between 1978-80 and 2005, and for each of the five 5-year age groups. The estimates in these tables can be compared as follows:

i. Down the columns, estimates are observed with *increasing age* in different 5-year age groups within each period of follow-up;
ii. Diagonally downwards to the right, estimates are observed with *increasing age, independent of time period*, by following the same cohort of individuals as they get older through different periods of follow-up; and
iii. Along the rows, estimates are observed *over time* in each 5-year calendar period, within each 5 year age-group.

Table 4.2 presents overall and CHD mortality rates from 1978-80 to 2005 in 5-year age groups and according to five 5-year calendar periods. Following the estimates down each column shows an increase in mortality rates with increasing age, within each time period. For example, in the period between 1983-85 and 1988-90, the CHD mortality rate per 1000 person years was 1.52 in 45-49 year old men and 10.78 in 60-64 years old men. Following the estimates diagonally downwards to the right shows that the increase in mortality rates with increasing age was independent of time period (same cohort of individuals getting older in different time periods). For example, the CHD mortality rate for men aged 45-49 was 1.92 in 1978-80 to 1983-85, and this increased to 7.35 when the same men were aged 65-69 in 1998-2000 to 2003-2005. In Table 4.2, following the estimates horizontally along the rows shows that mortality rates had declined over the 25 year period, independent of age. For example, CHD mortality rate per 1000 person years in 55-59 years olds had halved from 6.46 in the first 5-year period (1978-80 to 1983-85) to 3.00 in men aged 55-59 years in the 15-20 year period (1993-95 to 1998-2000).

4.5.1 Relative social class differences: the influence of age

Table 4.3 shows age-adjusted hazard ratios comparing manual with non-manual social class groups for CHD and all-cause mortality for different 5-year age groups in the five 5-year calendar periods during the 25-year follow-up. There was evidence that the effect of social class on mortality lessened as the men grew older. This is displayed in Table 4.3, by following the estimates with increasing age both down the columns of the table and along the table diagonally downwards to the right. Down the columns (examining age effects within each time period) the hazard ratios (manual vs. non-manual) for all-cause and CHD mortality appear to decrease with increasing age groups. For example, within the 5-year period of 1983-85 to 1988-90, the hazard ratio for CHD mortality decreased from 1.68 in men aged 45-49 years to 1.10 in men aged 60-64 years. A similar pattern was observed in the subsequent periods of follow-up, as well in the overall hazard ratios (last column on the right for the entire follow-up period). Another way of examining change in relative inequalities with increasing age is by observing the estimates in Table 4.3 diagonally downwards to the right. These estimates for CHD and all-cause mortality, which are for the same cohort of men as they get older in different time periods, also appear to decrease; for example, the hazard ratio (manual vs. non-manual) for CHD mortality in 55-59 year old men at baseline decreased from 1.99 in the first 5-year period to 1.03 in the last 5-year period of follow-up, when aged 75-79. Although this pattern was not consistently seen in all age groups, an analysis across all time periods showed that the ratio of hazard ratios for social class differences for a 20-year increase in age for CHD mortality was 0.73 (95%CI 0.55, 0.98); representing a 27% decrease in the relative social class difference in CHD mortality risk for a 20-year increase in age. The estimated hazard ratio for a manual social class subject would be 1.84 at age 55, but only 1.34 at age 75 years. The ratio of hazard ratios for social class differences in all-cause mortality was 0.77 (95%CI 0.65, 0.91), representing a 23% decrease in the relative social class difference in risk for a 20-year increase in age. Fitting a term 'social class*age' in the model to test for the possibility that the relationship between social class and mortality changed significantly with a 20-year increase in age provided evidence suggesting that the effect of social class was modified by age (p for test for interaction was 0.03 for CHD mortality and 0.003 for all-cause mortality).

4.5.2 Relative social class differences: the influence of period

The extent to which relative differences in risks of death comparing manual with non-manual groups had changed over time, independent of age, can be observed in Table 4.3 by following the estimates horizontally along the rows. The relative hazard for CHD and all-cause mortality for men of manual social class appeared to increase over time. For example, in men aged 55-59 years, the hazard ratio for CHD mortality in the first 5-

year period of follow-up (early 1980s) was 1.99, while for men aged 55-59 in the 15-20 year period of follow-up (late 1990s) it was 2.68; the corresponding hazard ratios for all-cause mortality increased from 1.70 to 2.25. Although this pattern was not seen in all the five individual time periods, an analysis extending trends across all age groups showed that over a 20-year calendar period the hazard ratio for the change in manual:non-manual social class was 1.22 (95%CI 0.83, 1.80) for total mortality and 1.75 (95%CI 0.89, 3.45) for CHD, representing estimated relative increases in the size of social class differences of 22% and 75% respectively. Fitting a term 'social class*period' in the model to test for the possibility that the relationship between social class and mortality changed significantly with a 20-year increase in period did not provide strong evidence that the effect of social class was modified by period (p for test for interaction was 0.11 for CHD mortality and 0.31 for total mortality). Thus, although there was no conclusive evidence of any change in relative inequalities, the result observed suggested an increase, rather than a decrease, in relative inequalities in mortality over time.

4.5.3 Absolute social class differences: the influence of age and period

Absolute rate differences in all-cause and CHD mortality between manual and non-manual social class groups for different calendar periods and age groups are presented in Table 4.4. Changes in the absolute difference in mortality rate with increasing age can be observed in Table 4.4 by following the estimates both down the columns (increasing age within each period) and diagonally downwards to the right (increasing age within the same cohort of men). The estimates displayed in each column show that within each time period, the absolute difference in mortality rate (manual versus non-manual) was greater in older age groups. For example, in 1988-90 to 1993-95, the absolute difference in CHD mortality rate per 1000 person years increased from 1.70 in

50-54 year old men to 4.10 in 65-69 year old men. A similar pattern was observed over the entire period of follow-up as seen in the overall absolute rate differences in mortality increasing with baseline age, presented in the last column on the right (the absolute difference in CHD mortality rate per 1000 person years was 1.27 for men aged 40-44 years at baseline compared to 2.47 for those aged 55-59 years at baseline). Observing the estimates in Table 4.4 diagonally downwards to the right, shows the absolute difference in CHD mortality between manual and non-manual groups with increasing age in the same cohort of individuals. The absolute social class differences in mortality rates appeared to increase, independent of time period, as the men grew older; for example the absolute difference per 1000 person years increased from 2.19 when the men were aged 45-49 years (in 1978-80 to 1983-85) to 5.10 when they were aged 65-69 years (in 1998-2000 to 2003-05).

The extent to which absolute social class differences in mortality change over time can be examined by comparing estimates within each age-group horizontally across the rows of Table 4.4. There appears to be some decrease in absolute risk difference between manual and non-manual groups over time, though this is not very consistently observed, particularly in the age-groups above 65 years. The absolute difference per 1000 person years in CHD mortality was 3.96 in men aged 55-59 years in 1978-80 to 1983-85 compared to 2.44 in those aged 55-59 in 1993-95 to 1998-2000. Results from modelling showed that the absolute difference in probability of survival from death of any cause from age 40 to age 65 between non-manual and manual subjects was 29% (95% bootstrap CI: 7% to 60%) in 1981 (the mid-point of first 5-year period of our follow-up) and 19% (95% CI: 4% to 47%) in 2001 (mid-point of last 5-year period of follow-up). Similarly, the estimated absolute difference in probability of survival to age

65 from CHD decreased from 17% (95% CI: 0 to 64%) in 1981 to 7% (95% CI: 0 to 35%) in 2001.

4.6 Discussion

In this study of middle-aged and older British men, total and CHD mortality rates declined over the period from 1978 to 2005. However, socioeconomic inequalities in all-cause and CHD mortality (both relative and absolute) appeared to persist over this period. Men in manual social classes had a greater risk of total mortality and CHD mortality compared to men in non-manual social classes. Relative socioeconomic inequalities in mortality from all-causes and CHD for manual compared with non-manual groups narrowed from middle-age as the men got older, although the absolute differences increased with age. The relative difference in all-cause and CHD mortality between manual and non-manual social class groups appeared to increase rather than to decline in the period from 1978 to 2005, though the differences were not statistically significant. However, the absolute magnitude of the social class differences appeared to decline because of the fall in overall mortality rates over this period.

4.6.1 Strengths and limitations of findings

The findings are based on data from a socioeconomically representative sample of middle-aged British men. More than 98% of the cohort has been followed-up for over 25 years through the NHS Central Register and general practice records.[282] The main strength of these results is that they quantify the extent of socioeconomic inequalities in overall mortality and CHD mortality, a leading cause of death, in a defined population over an extended period with high rates of follow-up, using a stable indicator of social class status, in a way which few earlier studies have been able to do. The social class measure used was based on the longest-held occupation, which was recorded at baseline in 1978-80 when subjects were 40-59 years. The longest-held occupation (classified as non-manual or manual) is an extremely stable and well-established marker of social class, which was defined for almost all study participants. Only 9% of subjects changed their social class status under this definition over a 20 year period, confirming the stability of the measure (reported in Chapter 3; section 3.9.1 page 87).[28] There are limitations attributed to the use of an occupation-based social class measure such as the inability to capture ethnic/racial disparities within the social classes, and the exclusion of those outside the labour force.[80] However, this cohort consisted almost entirely of White European men, and information on the longest-held occupation was available for most of the subjects. Despite possible limitations of using social class based on occupation, it was essential for this analysis to have a single measure of socioeconomic position which would act as a reference point over the entire study period; longest-held occupation is likely to have fulfilled this criterion better than many other measures. Dichotomising social class into manual and non-manual groups in the analyses provides a stable indicator of changes in the two main social class groups than would be possible with six groups. Using these stable and well-defined groups provides a useful summary of the extent of inequalities over time to obtain an overall direction of change in socioeconomic inequalities for total and CHD mortality.

The present study, however, was based on older men, excluding subjects from inner cities, and towns with high mobility, thus excluding ethnic minorities and highly mobile people.[282] The results may not, therefore, be completely generalisable to younger subjects, women and ethnic minority groups and may not be directly generalisable to other Western countries. Within the study population, it is possible that the extent of socioeconomic inequalities in early life will have differed appreciably between the 1920s and 1930s, when unemployment was particularly high. This is difficult to examine in the current analyses, in which the effect of age and calendar period was considered. Therefore, it was not possible to define the influence of the year of birth of the cohort, as all these three effects (age, period and cohort) cannot be taken into account in the same statistical model. However, in this cohort of men the influence of childhood socioeconomic position on CHD risk was modest, both among subjects born in the 1920s and 1930s (presented in Chapter 7; section 7.5.2 on page 199). Moreover, if socioeconomic inequalities were stronger in the 1930s, the effect on the results would be to *under*-estimate either or both the decline in socioeconomic inequalities with age and the increase in socioeconomic inequalities with calendar time.

4.6.2 Time trends in social class differences in all-cause and CHD mortality: comparison with previous studies

In the present study, the extent to which socioeconomic inequalities in mortality from all causes and from CHD, the leading cause of death in the UK,[7] have changed over a period of time in Britain was investigated. Though there are previous studies reporting on trends in health inequalities in Britain,[34;72;76;307;308;312] little is known about recent trends in socioeconomic class inequalities in CHD mortality in the UK since the 1990s. The findings from this Chapter are similar to those of some recent studies, which have shown that relative inequalities have not narrowed, and may have increased over time in Britain.[76;307;312] The Acheson report demonstrated a clear widening in relative socioeconomic inequalities in all-cause and CHD mortality between the early 1970s and 1990s.[34] A recent Department of Health report demonstrated that absolute differences in socioeconomic inequalities, measured by area deprivation, in circulatory disease mortality were falling until the early 2000s, with signs of widening relative inequalities.[113] This decline in absolute difference, which is important in public health terms, probably reflects the declines both in total mortality rates and in CHD mortality rates that were observed in this study, and are known to have occurred over the last 3 decades in the UK.[313;314]

With increasing age from middle-age, the relative difference in mortality rates between social classes declined, but persisted at older ages, while absolute differences increased, as a result of the higher death rates among older subjects. The decrease in relative socioeconomic differences with increasing age alongside increasing absolute difference was also observed in a study on total mortality in 11 European populations.[32] The persistence of socioeconomic differences in mortality at older ages is consistent with the results of other British and European studies.[32;315] The relative decline in the importance of social class at older ages is in keeping with the widely observed attenuation of other risk factor-chronic disease relations at older ages.[123;124]

In this Chapter, the extent of inequalities over time has been estimated, but the possible causal pathways or mechanisms have not been investigated. It has been previously reported that an increase in socioeconomic inequalities could be attributed to a decrease in rate of disease in higher social class with little or no improvement in lower social classes.[34;307] This implies greater beneficial changes in social classes I and II compared to lower social classes. A more rapid pace of favourable changes in health-related behaviours such as smoking and physical activity amongst higher compared to lower socioeconomic groups could play a role in contributing to widening inequalities, especially for a leading cause of death like coronary disease.[307] The cumulative effect of these behavioural and other factors over the life course has also been implicated as a pathway of inequalities.[316;317]

4.6.3 Interpretation of findings

The results of the present study suggest that in recent years in Britain, relative socioeconomic inequalities in CHD mortality and total mortality have not appeared to reduce. This implies that the association between socioeconomic position and mortality has continued over the last 25 years in Britain – manual social classes remain at an increased risk of mortality compared to non-manual social classes. However, alongside overall declines in total mortality and CHD mortality in Britain over the last three decades, absolute inequalities were observed to have narrowed. Nevertheless, absolute socioeconomic differences in CHD mortality still persist in Britain. With increasing age, relative socioeconomic inequalities in mortality narrowed. Absolute differences, however, increased with age, reflecting the higher mortality rates in older ages. This increased absolute socioeconomic difference in older ages reflects the public health burden of these inequalities in CHD in later life.

4.6.4 Conclusions

Despite a decrease in absolute social class difference in CHD mortality over 25 years until 2005 there is still considerable scope for reducing existing inequalities in mortality. The absence of any appreciable reduction in relative socioeconomic inequalities over this period implies that manual social class groups continue to be at a disadvantage compared to non-manual groups. The results of this Chapter affirm that greater efforts are needed or alternative strategies need to be explored if the gap between the health of those at the higher and lower end of the socioeconomic hierarchy is to be narrowed.

Figure 4.1 Kaplan Meier survival curves comparing all-cause mortality according to social class in men aged 40-59 years followed-up from 1978-80 to 2005

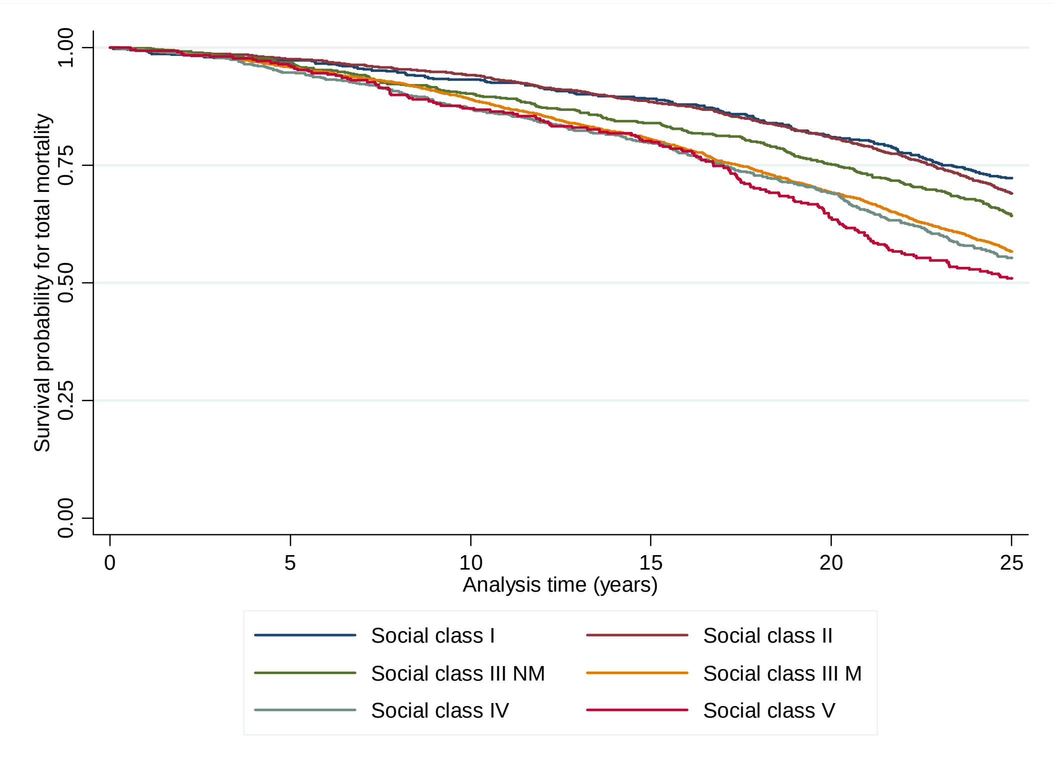


Figure 4.2 Kaplan Meier survival curves comparing CHD mortality according to social class in men aged 40-59 years followed-up from 1978-80 to 2005

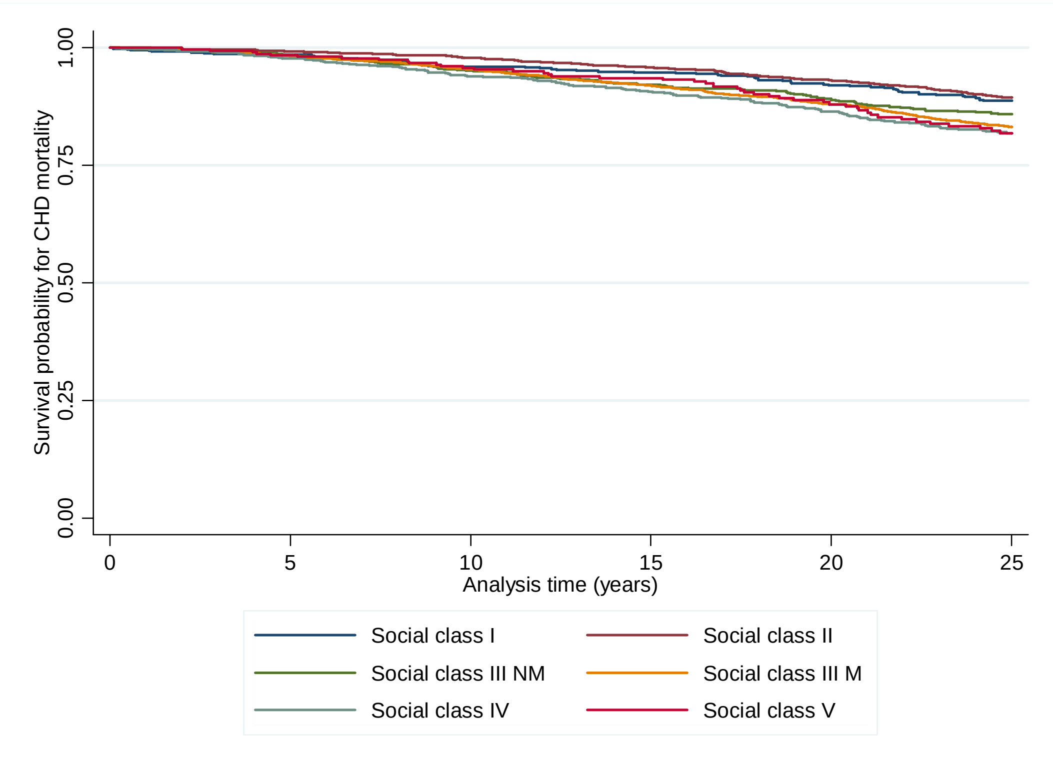


Table 4.1 All-cause and CHD mortality according to social class in men aged 40-59 years followed-up from 1978-80 to 2005

	All-cause mortality HR (95%CI)	CHD mortality HR (95%CI)
Social class I	1.00	1.00
Social class II	1.05 (0.88, 1.25)	0.86 (0.64, 1.15)
Social class III non-manual	1.26 (1.03, 1.53)	1.19 (0.86, 1.64)
Social class III manual	1.62 (1.38, 1.90)	1.41 (1.09, 1.84)
Social class IV	1.67 (1.39, 2.01)	1.54 (1.14, 2.09)
Social class V	2.04 (1.64, 2.53)	1.56 (1.07, 2.28)

HR=hazard ratio; CI=confidence intervals

Table 4.2 Incidence rates per 1000 person years for all-cause and CHD mortality (number of deaths) by age and in 5-year time periods from 1978-80 to 2005

Age (years)	0-5 years 1978-80 to 1983-85	5-10 years 1983-85 to 1988-1990	10-15 years 1988-90 to 1993-95	15-20 years 1993-95 to 1998-2000	20-25 years 1998-2000 to 2003-05
40-44					
N	1677				
All-cause (n)	1.97 (16)				
CHD (n)	0.46 (4)				
45-49					
N	1838	1661			
All-cause (n)	4.58 (41)	3.75 (31)			
CHD (n)	1.92 (16)	1.52 (13)			
50-54					
N	1911	1797	1630		
All-cause (n)	7.52 (69)	7.34 (62)	5.89 (47)		
CHD (n)	3.40 (30)	3.51 (29)	2.29 (17)		
55-59					
N	1853	1842	1735	1583	
All-cause (n)	14.22 (129)	13.45 (119)	11.28 (94)	10.39 (78)	
CHD (n)	6.46 (58)	5.75 (52)	4.95 (41)	3.00 (22)	
60-64					
N		1724	1723	1641	1505
All-cause (n)		27.07 (220)	19.37 (160)	15.07 (119)	13.52 (101)
CHD (n)		10.78 (88)	7.44 (63)	5.14 (41)	2.78 (21)
65-69					
N			1504	1563	1522
All-cause (n)			30.99 (218)	32.94 (237)	27.65 (198)
CHD (n)			9.45 (68)	9.91 (71)	7.35 (54)
70-74					
N				1286	1326
All-cause (n)				52.78 (295)	45.99 (277)
CHD (n)				16.12 (89)	12.31 (76)
75-79					
N					991
All-cause (n)					74.96 (313)
CHD (n)					22.65 (92)

Table 4.3 Age-adjusted hazard ratios (manual compared with non-manual social classes) for all-cause and CHD mortality by age and in 5-year time periods from 1978-80 to 2005

Age (years)	0-5 years 1978-80 to 1983-85	5-10 years 1983-85 to 1988-1990	10-15 years 1988-90 to 1993-95	15-20 years 1993-95 to 1998-2000	20-25 years 1998-2000 to 2003-05	Overall for each baseline age group (95%CI)
40-44						
All-cause	1.64					1.75 (1.35, 2.26)
CHD	2.25					2.05 (1.25, 3.42)
45-49						
All-cause	1.82	2.55				1.67 (1.39, 2.01)
CHD	5.26	1.68				2.11 (1.53, 2.93)
50-54						
All-cause	1.09	1.84	1.62			1.60 (1.39, 1.85)
CHD	0.87	2.90	2.48			1.47 (1.15, 1.88)
55-59						
All-cause	1.70	1.49	1.66	2.25		1.37 (1.21, 1.54)
CHD	1.99	1.52	1.49	2.68		1.28 (1.04, 1.58)
60-64						
All-cause		1.65	1.99	1.74	1.39	
CHD		1.10	1.52	1.99	1.61	
65-69						
All-cause			1.25	1.69	1.57	
CHD			1.56	1.33	2.03	
70-74						
All-cause				1.42	1.55	
CHD				1.27	1.91	
75-79						
All-cause					1.15	
CHD					1.03	
Overall for each calendar period (95%CI)						
All-cause	1.58 (1.21, 2.05)	1.68 (1.36, 2.07)	1.55 (1.29, 1.86)	1.63 (1.39, 1.90)	1.32 (1.15, 1.52)	
CHD	1.71 (1.13, 2.62)	1.43 (1.05, 1.95)	1.59 (1.17, 2.17)	1.49 (1.13 1.97)	1.49 (1.15, 1.95)	

Table 4.4 Absolute difference in incidence rates per thousand person years between manual and non-manual social classes for all-cause and CHD mortality by age and in 5-year time periods from 1978-80 to 2005

Age (years)	0-5 years 1978-80 to 1983-85	5-10 years 1983-85 to 1988-1990	10-15 years 1988-90 to 1993-95	15-20 years 1993-95 to 1998-2000	20-25 years 1998-2000 to 2003-05	Overall for each baseline age group
40-44						
All-cause	0.88					3.53
CHD	0.35					1.27
45-49						
All-cause	2.54	3.10				5.81
CHD	2.19	0.78				2.94
50-54						
All-cause	0.68	4.09	2.70			8.63
CHD	-0.46	3.33	1.70			2.46
55-59						
All-cause	6.99	5.06	5.45	7.49		8.97
CHD	3.96	2.29	1.93	2.44		2.47
60-64						
All-cause		12.66	12.23	7.92	4.12	
CHD		1.03	3.08	3.28	1.18	
65-69						
All-cause			6.80	15.96	12.26	
CHD			4.10	2.63	5.10	
70-74						
All-cause				17.22	19.00	
CHD				3.60	7.65	
75-79						
All-cause					10.49	
CHD					0.76	
Overall for each calendar period						
All-cause	3.06	6.59	7.19	12.10	11.48	
CHD	1.65	2.01	2.76	3.08	3.92	

[Permission to reproduce Tables 4.3 and 4.4 has been obtained from the Journal of Epidemiology & Community Health]

Chapter 5

Relationship of adult socioeconomic position with established and novel coronary risk factors in older age

5.1 Summary

The mechanisms by which adult socioeconomic position influences coronary heart disease (CHD) are not fully understood. Socioeconomic differences in coronary risk factors could play an important role in linking socioeconomic position with CHD. This Chapter aims to investigate the relationship of social class with both established and novel coronary risk factors in older age. Established coronary risk factors include behavioural (smoking, physical activity, body weight, alcohol consumption) and biological factors (blood pressure and blood lipids). In addition to established risk factors, novel coronary risk factors including inflammatory and haemostatic markers, metabolic syndrome, insulin resistance, and dietary factors have been hypothesised to influence the relationship between social class and CHD. This Chapter investigates the relationship of social class with both established and novel factors in older age using the British Regional Heart Study, when the men were aged 60-79 years in 1998-2000. Lower social class groups had higher levels of cigarette smoking, physical inactivity and obesity. Men in lower social classes were more likely to have higher levels of triglycerides and lower mean HDL-cholesterol. Lower social class was associated with higher levels of inflammatory and haemostatic markers. However, these associations were largely explained by behavioural risk factors (smoking, physical activity and BMI), though some associations, particularly those of social class with von Willebrand

factor, interleukin-6 and factor VIII remained statistically significant after the adjustments. Metabolic syndrome was also inversely associated with social class; adjustment for behavioural factors attenuated this association. Dietary intake of fibre, carbohydrates, vitamin C, fresh fruit and vegetables were lower in lower social classes. CHD-related dietary nutrients (fat and cholesterol) did not demonstrate social class variations.

5.2 Introduction

As observed in Chapter 4, socioeconomic gradients in coronary heart disease (CHD) are present in middle-aged men, with those from lower socioeconomic positions having a higher risk of CHD. These inequalities in CHD mortality tended to weaken in relative terms with increasing age although the absolute difference increased (Chapter 4). Adverse health behavioural risk factors for CHD including cigarette smoking, obesity and alcohol consumption,[10;11;318] are known to be more frequent in people of lower socioeconomic position in middle-age.[102;214;319] Similar relations have been observed for biological coronary risk factors such as blood pressure, but less so for blood lipids.[24;25;180] Lower socioeconomic groups have been observed to have lower total cholesterol levels than people of higher socioeconomic position.[37;100;180] However, whether socioeconomic position is related to these established coronary risk factors in older age is not fully known. Moreover, in observational studies, mostly in middle-aged populations, the socioeconomic gradient in CHD is often not substantially explained by these established coronary risk factors.[39;85] In more recent years, 'novel' coronary risk factors including inflammatory and haemostatic markers such as C-reactive protein (CRP), fibrinogen and interleukin-6 (IL-6),[189;320] have also been reported to be higher in lower socioeconomic groups.[40;41;213;225;228] This has led to the hypothesis that inflammatory and haemostatic markers could influence the socioeconomic variation in CHD.[40;102;227;321] Most studies so far have reported on the relationships between socioeconomic position and inflammatory markers such as fibrinogen, CRP and IL-6,[40;41;225;228] and less is known about the relationships of haemostatic markers implicated in CHD risk (e.g. von Willebrand factor, factor VIII, tissue plasminogen activator antigen) with socioeconomic position. More recently also, the clustering of some cardiovascular risk factors (obesity, dyslipidemia, hyperglycaemia, hypertension and insulin resistance) in the form of the metabolic syndrome has also been reported to be associated with an increased risk of CHD,[322;323] although not all studies provide evidence for this.[204] Even though the role of the metabolic syndrome as a cardiovascular risk marker is contentious,[201] there has been an increasing interest in investigating the relationship of socioeconomic position with the clustering of metabolic risk factors.[14;236;324] It has also been postulated that metabolic syndrome might explain the link between socioeconomic position and CHD.[233;242] However, the association between socioeconomic position and metabolic syndrome has not been completely consistent between studies,[14;240;241] and it is possible that the relationship is confounded by behavioural coronary risk factors, which are strongly related both to metabolic syndrome and to socioeconomic position.[45;236;237;240;241;261] An association between socioeconomic position and the metabolic syndrome independent of behavioural factors has been proposed to form a direct pathway linking socioeconomic position and CHD,[14;233;242] possibly working through neuroendocrine mechanisms responsible for dyslipidemia, insulin resistance, high blood pressure and obesity.[234] Dietary intake, another health behavioural factor, particularly the consumption of fat, fruits and vegetables has also been reported to be related to CHD[247;325-327] as well as to socioeconomic position, with lower socioeconomic groups consuming less healthy diets compared with higher socioeconomic groups.[246;252] This Chapter aims to assess the relation between socioeconomic position and coronary risk factors in later life, at ages 60-79 years with a particular focus on novel risk factors. While this Chapter examines the extent of social class differences in coronary risk factors, the following Chapter will address the contribution of these risk factors to explaining socioeconomic differences in CHD.

5.3 Objectives

The objectives of this Chapter are:

i) To examine the association of social class with behavioural coronary risk factors (cigarette smoking, physical activity, alcohol consumption and body mass index);

ii) To assess the association of social class with biological coronary risk factors (blood pressure, blood lipids);

iii) To examine the association between social class and inflammatory/haemostatic markers known to be related to CHD, in men free of chronic diseases and taking account of the potential confounding effects of behavioural risk factors (cigarette smoking, body mass index, physical activity and alcohol consumption);

iv) To evaluate the association between social class and metabolic syndrome and to assess whether this is independent of behavioural factors (smoking, physical activity and alcohol consumption);

v) To assess the association between social class and dietary intake implicated in CHD risk.

5.4 Methods

Data for this Chapter comes from the 20 year re-examination of the British Regional Heart Study in 1998-2000, when subjects were aged 60-79 years. All men completed a mailed questionnaire providing information on their lifestyle and medical history, had a physical examination and provided a fasting blood sample. The men were requested to fast for a minimum of 6 hours and to attend a measurement session at a specified time between 0800 and 1800. 4252 men (77% of those still alive) attended the examination and 4094 men (74%) had at least one measurement of the biological factors. The indicator of socioeconomic position used was occupational social class, based on the longest-held occupation measured at baseline when men were aged 40-59 years, and classified using the Registrar General's Social Class Classification into social class I, II, III non-manual (III NM), III manual (III M), IV and V.[300]

5.4.1 Behavioural risk factors

Detailed information on cigarette smoking habits, physical activity and alcohol consumption was collected at age 60-79 years, as described in the methods Chapter (section 3.7.3 to 3.7.5 page 82). Based on frequency and number of alcoholic drinks consumed on average in a week, occasional drinking was defined as <1 unit of alcohol/week, light/moderate drinking as 1-42 units/week and heavy drinking >42 units/week (1 UK unit = 10g). Physical activity scores were assigned on the basis of the frequency and type of activity and the men were divided into 6 groups: none, occasional, light, moderate, moderately-vigorous and vigorous. Subjects in the categories of 'none' or 'occasional' activity were grouped together as 'inactive'. Body mass index (BMI) was calculated (weight/height^2 in kg/m^2) for each man at the physical examination. BMI greater than or equal to 30 kg/m^2 was defined as obese.

5.4.2 Biological risk factors

As part of the physical examination, blood pressure was measured and fasting blood samples were collected as described in Chapter 3 (sections 3.8.1 and 3.8.2 page 85 and 86). Biological risk factors investigated in this Chapter were systolic and diastolic blood pressure, triglycerides, total cholesterol, high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C). Total and HDL cholesterol were analysed using the methods of Siedel et al[294] and Sugiuchi et al[295] respectively. Low-density lipoprotein (LDL) cholesterol was calculated using the Fredrickson-Friedwald equation.[296]

5.4.3 Inflammatory and haemostatic markers

A range of inflammatory and haemostatic markers, many of which were associated with CHD in previous reports,[13;189;193;320;328;329] were measured in the blood samples – C-reactive protein (CRP), fibrinogen, interleukin-6 (IL-6), factor VIII, von Willebrand factor (vWF), tissue plasminogen activator (t-PA) antigen, plasma viscosity, fibrin D-dimer, white blood cell (WBC) count, platelet count, activated protein C (APC) ratio, activated partial thromboplastin time (aPTT), and haematocrit. Details of the laboratory measurements of these markers are described in Chapter 3 (section 3.8.2 page 86).

5.4.4 Metabolic syndrome and insulin resistance

Metabolic syndrome was defined according to the National Cholesterol Education Programme criteria which included three or more of the following: (1) fasting plasma glucose of at least 110 mg/dL (6.1 mmol/L), (2) serum triglycerides of at least 150 mg/dL (1.7 mmol/L), (3) serum HDL-C less than 40 mg/dL (1.04 mmol/L), (4) blood pressure of at least 130/85 mmHg or on anti-hypertensive treatment or (5) waist circumference of more than 102 cm.[330] Insulin resistance was estimated using the homeostasis model assessment (HOMA) as the product of fasting glucose (mmol/l) and insulin (μU/ml) divided by the constant 22.5.[331]

5.4.5 Dietary factors

Dietary intake was recorded from a detailed 7-day food frequency questionnaire as described in Chapter 3 (section 3.7.6 page 84).[288] Nutrient intakes were calculated from a validated program using the food frequency of standard portion sizes for each food and the nutrient composition of the foods obtained from the UK food composition tables.[289] Dietary factors implicated in CHD risk were included – total fat, polyunsaturated fat, saturated fat, cholesterol, total fibre, and vitamin C.[206] To assess intake of fresh fruit and vegetables, men were asked how often (number of days each week) they ate fresh fruit and vegetables in summer and winter. Information on plasma vitamin C was also available from blood samples.

5.4.6 Rationale for analyses

The relationship of socioeconomic position with coronary risk factors in older age was examined by using occupational social class as the measure of socioeconomic position. The levels of different coronary risk factors were examined according to social class groups (I to V). The relationship of social class with metabolic syndrome was adjusted for behavioural risk factors (smoking, physical activity and alcohol consumption) which are potential confounders related both to metabolic syndrome and social class.[14;45] Similarly the relationship between social class and insulin resistance (being in the top fourth of the HOMA distribution) was adjusted for behavioural risk factors (smoking, physical activity and alcohol consumption). After adjustment for these behavioural factors, the effect of insulin resistance was also adjusted for BMI, since BMI is strongly associated with insulin resistance and social class.[290;332;333] The analysis on metabolic syndrome and insulin resistance excluded men with prevalent diabetes, identified as those with a doctor diagnosis of diabetes or fasting glucose of ≥7 mmol per litre (n=385), because of the greater prevalence of metabolic syndrome and insulin resistance in diabetics.

Analyses of the association between social class and inflammatory/haemostatic markers excluded 1570 men with chronic diseases or conditions that have been shown to be associated with changes in levels of haemostatic and inflammatory factors.[12;197;334] Subjects with chronic diseases were those who reported a doctor-diagnosis of myocardial infarction, angina, stroke, or diabetes, as well as those who were currently taking anti-inflammatory drugs for musculoskeletal disorders or taking warfarin. To take into account potential confounders, factors which have been shown to be associated with haemostatic and inflammatory markers in the present study and in other reports, such as smoking, BMI, alcohol intake and physical activity, were also adjusted for.[43;44;196;335] Further adjustment for systolic blood pressure, HDL-C, triglycerides and insulin and glucose levels was also carried out.

5.4.7 Statistical methods

Logistic regression was used to calculate age-adjusted odds ratios with 95% confidence intervals (CI) for categorical variables including cigarette smoking, alcohol consumption, obesity, and metabolic syndrome for the six social class groups (social I was the reference group). Similarly, odds ratios (95%CI) according to social class were calculated for the individual components of the metabolic syndrome and for being in the top fourth of the distribution of HOMA (insulin resistance). Analysis of covariance was used to obtain age-adjusted mean levels of biological risk factors, inflammatory/haemostatic markers and dietary factors according to social class. The distributions of triglycerides, CRP, IL-6, fibrin D-dimer, WBC count, aPTT, dietary total fat, saturated fat, polyunsaturated fat, dietary cholesterol, dietary cereal fibre, vitamin C, plasma vitamin C, were positively skewed and required log transformation. Mean levels of dietary nutrients were adjusted for total energy intake. For the adjustments, age, BMI, systolic blood pressure, HDL-C, triglycerides and insulin and glucose levels were fitted as continuous variables. Tests for trend were carried out by fitting social class as a continuous variable in the models.

5.5 Results

5.5.1 Social class and behavioural factors

The relationship of social class with cigarette smoking is presented in Table 5.1. Men in lower social class groups were more likely to be current smokers. Men in manual social classes (social classes IIImanual, IV, V) compared to non-manual social classes (I, II, IIInon-manual), were twice as likely to be current smokers (age-adjusted odds ratio was 2.04; 95%CI 1.68, 2.48). On the other hand, the likelihood of being 'never smokers' was lower in manual social classes and decreased from social class I to V (age-adjusted odds ratio for social class V compared to social class I was 0.26; 95%CI 0.16, 0.42). The prevalence of ex-smokers was greater in lower compared to higher social class groups.

Table 5.2 shows the association between social class and alcohol consumption. The proportion of non-drinkers and occasional drinkers increased from social class I to social class V (p for trend <0.0001 for both). There were fewer men who were light/moderate drinkers in lower social classes. The age-adjusted odds ratio for being a light/moderate drinker in social class V compared to social class I was 0.35 (95%CI 0.23, 0.63). The proportion of heavy drinkers was greater in social class V compared to social class I, although this association was not statistically significant possibly due to the small number of heavy drinkers (the age-adjusted odds ratio for heavy drinking in social class V vs. social class I was 2.09; 95%CI 0.67, 6.51; p for trend 0.44).

Table 5.3 shows the association of social class with physical activity, obesity and mean BMI. Men in lower social class groups were more likely to be physically inactive and were less likely to be engaged in moderate-vigorous physical activity (the age-adjusted odds ratio for physical inactivity in social class V vs. I was 2.55, 95%CI 1.66, 3.91; p for trend <0.0001). The proportion of men who were obese increased from social class I to V. The age-adjusted odds ratio for obesity in social class V compared with I was 2.35 (95%CI 1.41, 3.91). The mean BMI levels tended to be greater in manual social classes.

5.5.2 Social class and biological coronary factors

Table 5.4 shows the relationships of social class with blood pressure and blood lipids. Mean systolic and diastolic blood pressure levels did not vary by social class. Higher mean levels of triglycerides were present in lower social class groups (age-adjusted p for trend 0.0007); this association was markedly attenuated when adjusted for BMI (p for trend 0.13). Age-adjusted mean levels of HDL-C decreased from social class I to social class V (p for trend <0.0001). This trend did not substantially change when adjusted either for BMI (p for trend 0.008) or for triglycerides (p for trend 0.003). Total cholesterol and LDL-C did not demonstrate a significant variation by social class in age-adjusted analyses.

5.5.3 Social class and inflammatory and haemostatic markers

Table 5.5, Table 5.6 and Table 5.7 show the relationships of social class with mean levels of inflammatory and haemostatic markers in men with no previous history of doctor-diagnosed diabetes or cardiovascular disease and who were not taking warfarin or medications for musculoskeletal disorders. In age-adjusted analyses, mean levels of inflammatory/haemostatic markers (CRP, fibrinogen, fibrin D-dimer, WBC, IL-6, vWF, factor VIII, plasma viscosity, and platelet count) increased from social classes I to V (all p for trend <0.05. See Table 5.5 and Table 5.6), showing inverse social class gradients. When further adjusted for behavioural risk factors (cigarette smoking, physical activity, BMI and alcohol consumption), social class gradients in levels of fibrinogen, fibrin D-dimer and WBC count were considerably weakened (Table 5.5). The social class gradient in CRP level was also weakened, although the trend remained significant (Table 5.5). In separate stepwise analyses examining the effect of adjustment for individual behavioural risk factors, it was apparent that most of these attenuations were caused by cigarette smoking. The relations of mean levels of IL-6, vWF, factor VIII, plasma viscosity and platelet count according to social class were only slightly altered after adjustment for behavioural factors (Table 5.6). Further adjustment for biological coronary risk factors (systolic blood pressure, HDL-C, triglycerides, insulin and glucose) did not appreciably alter the above relationships. No association was seen between social class and t-PA antigen, haematocrit, APC ratio or aPTT in age-adjusted analyses (Table 5.7).

5.5.4 Social class and metabolic syndrome

Among men without prevalent diabetes defined in 1998-2000, 817 men (28%) had metabolic syndrome. Table 5.8 shows the prevalence of and odds ratios for metabolic syndrome according to social class. Age-adjusted analysis showed an inverse social gradient in metabolic syndrome, with increasing odds of the metabolic syndrome from social class I to social class V (age-adjusted odds ratio for having metabolic syndrome in social class V compared to I was 1.64; 95%CI 0.98, 2.76; p for trend 0.0005). This association was markedly attenuated when adjusted for behavioural risk factors including cigarette smoking, physical activity and alcohol consumption; the trend in the social class gradient was no longer significant after these adjustments (odds ratio for social class V vs. social class I attenuated to 1.22; 95%CI 0.71, 2.08; p for trend 0.06).

Table 5.9 shows the relationship of social class with individual components of the metabolic syndrome. Social class was associated with higher levels of 4 of the 5 metabolic syndrome components; the risk of having high blood pressure, high triglycerides, high waist circumference and low HDL-cholesterol was greatest in social class V and lowest in social class I. Adjustment for behavioural risk factors attenuated these associations between social class and the individual components of metabolic syndrome, except for those for waist circumference and high blood pressure, which were little affected by adjustment. Compared with social class I, social class V had a two-fold greater risk of having low HDL-cholesterol levels (<1.04 mmol/L) and a similar increased risk of having high waist circumference (>102 cm). After adjustment for behavioural factors (smoking, physical activity and alcohol consumption), the odds ratio for social class V compared with social class I for low-HDL cholesterol attenuated to 1.55 (95%CI 0.86, 2.79), while an increased risk for high waist circumference in social class V remained (odds ratio 1.71; 95%CI 1.02, 2.88). Table 5.10 shows the association between social class and HOMA (insulin resistance). The risk of being in the top fourth of HOMA distribution increased from social class I to social class V (p for trend 0.02). This gradient was attenuated when adjusted for behavioural factors. The age-adjusted odds ratio for high HOMA was 2.47 (95%CI 1.44, 4.23) for social class V compared with social class I, which reduced to 1.98 (95%CI 1.13, 3.46; p for trend 0.17) after adjustment for behavioural factors (smoking, physical activity and alcohol consumption). This increased risk of high HOMA in social class V was attenuated to 1.46 (95%CI 0.79, 2.68) when further adjusted for BMI.

5.5.5 Social class and dietary factors

Age-adjusted mean levels of different dietary factors for each social class group are presented in Table 5.11. Dietary intake of total fat, saturated fat, polyunsaturated fat and cholesterol did not show social class differences. Fibre intake (total, cereal and vegetable fibre) decreased from social class I to social class V. Intakes of vitamin C and fresh fruit and vegetables were also significantly lower in lower social class groups. These relationships were not substantially altered when adjusted for cigarette smoking or BMI (results not shown). Plasma vitamin C also showed inverse social class gradients, with decreasing plasma levels from social class I to social class V.

5.6 Discussion

Several established and novel coronary risk factors in this study of older British men were related to social class. Lower social classes had higher levels of unfavourable risk factors, including higher prevalences of cigarette smoking, physical inactivity, obesity, higher triglycerides levels and low HDL-cholesterol. The levels of inflammatory and haemostatic markers, and metabolic syndrome increased with decreasing social class. These social class relationships with inflammatory/haemostatic markers and metabolic syndrome were substantially explained by behavioural risk factors, but some with vWF and factor VIII persisted. Dietary fat intake (total, saturated and polyunsaturated) and dietary cholesterol did not demonstrate associations with social class. Lower social class was associated with lower consumption of fresh fruit and vegetables and vitamin C, and also with plasma vitamin C; levels of these factors decreased from the highest to lowest social class.

5.6.1 Strengths and limitations of findings

A particular strength of these results is that they are based on a socioeconomically and geographically representative sample of older British men. Such a socially representative sample is a particular strength when studying socioeconomic variations in coronary risk factors. Using this sample, the relation of a range of different haemostatic/inflammatory markers with social class has been explored, some of which have not been studied before in this context. Another strength of these results is the validity of the measure of socioeconomic position used, which can be difficult to characterise in older populations. Socioeconomic position was based on the longest-held occupation assessed at middle-age when the men were aged 40-59 years. Therefore, this measure is likely to be a stable measure of socioeconomic position over most of adult life, and is likely to reflect socioeconomic conditions even in older age. A limitation of the results is that the study sample of older British men excluded subjects from inner cities and towns with high mobility, thus with little information on ethnic minority groups and highly mobile people. The results may not, therefore, be generalisable to younger subjects, women, ethnic minority groups and other Western populations. Also, the results are based on cross-sectional analyses, which limits the extent to which causal inferences can be drawn between social class and particularly, inflammatory markers such as vWF and factor VIII.

A potential limitation of the results arises due to the possibility of bias in reporting of health behavioural risk factors including cigarette smoking, alcohol consumption and physical activity. If random misreporting of these risk factors occurred in all social class groups, the strength of the associations presented would be underestimated. Systematic misreporting of behavioural factors in different social class groups could result in altering the results in either direction depending on which social class groups over/under report. Although it was not possible to validate whether misreporting of these risk factors differed systematically by social class, a close agreement between reported smoking and cotinine levels had been previously observed in this population sample, and in other studies;[136;336] the agreement between cotinine levels and reported smoking was similar in non-manual and manual social class groups. It is also possible that smokers and heavy drinkers with higher mortality rates had died at an earlier age, resulting in a healthier sample at 60-79 years with fewer smokers and heavy drinkers. Nevertheless, this issue of selection or survival bias is inherent when studying older populations. While this issue may weaken the associations between social class and behavioural risk factors in an older population, the results presented in this Chapter are consistent with the relationship of smoking, physical activity and alcohol consumption with social class observed when the study participants were middle-aged.[37;285]

Imprecise measurement of physiological and biological markers due to measurement error or short-term deviation from average levels is also likely to have occurred. Previous work using a one-week repeatability study indicated that measurement errors existed to some extent for nearly all biological and inflammatory markers except for HDL-C and BMI.[337;338] This implies that the relationships of these biological and novel risk factors with social class presented in this Chapter are possibly weaker than the true associations. Single measurements and inaccurate reporting of dietary intake could also result in measurement errors for dietary intake. An indication of the validity of reported dietary intake, however, is that social class gradient in dietary vitamins C was consistent with that of plasma vitamin C.

5.6.2 Comparison with previous studies

5.6.2.1 Social class and behavioural risk factors

A number of studies in middle-aged populations have explored the relationship of socioeconomic position with behavioural risk factors.[24;37;102;180;214;261;319] The social class differences in cigarette smoking, physical inactivity, and obesity observed in the older men of the British Regional Heart Study were similar to the patterns of association previously described in middle-age, with greater levels of adverse behavioural factors in lower socioeconomic groups.[24;37;102;180;214;261;319] However, few such studies have been carried out in older people and only one of these were based in the UK, although some evidence is available from studies in other countries. In the English Longitudinal Study of Ageing (ELSA), men and women aged >60 years in routine/manual social class were more likely to be current smokers and less physically active compared to those aged >60 years in professional/managerial social classes.[119] Manual workers in a Swedish population-based study of men and women ≥65 years showed greater rates of cigarette smoking, physical inactivity and obesity than professional employees.[121] Similar differences in cigarette smoking were observed according to education and income levels in a US study of men and women aged ≥65 years, in which lower educational and income levels were associated with greater prevalences of current smokers.[218] Obesity, physical activity and heavy alcohol consumption were also greater in people of lower educational levels, and lower occupational social classes in a Spanish noninstitutionalised population aged over 60 years.[216] Heavy drinking did not vary significantly by social class in the present study, possibly due to the small proportion (3%) of heavy drinkers in this group of older men. In middle-age, however, when heavy drinking was more prevalent, the men of manual social classes in the British Regional Heart Study were observed to be far more likely to be heavy drinkers than non-manual social classes.[285] In the present study, older men of lower social classes were more likely to be non-drinkers or occasional drinkers, which maybe due to a greater prevalence of co-morbidities in these social classes resulting in reducing/giving up alcohol consumption. Regular light/moderate drinking was greater in higher social classes, which was also previously observed when the cohort was middle-aged.[285] This social class pattern of alcohol consumption (higher prevalence of light drinking in higher social classes) is consistent with previous studies in middle-aged populations.[214;261]

5.6.2.2 Social class and biological coronary risk factors

Among biological coronary risk factors, social class appeared to be related only to triglycerides and HDL-cholesterol, while there was no evidence of social class variation in total cholesterol, LDL-cholesterol or blood pressure. The association of social class with triglycerides could be due to greater levels of BMI in lower social class groups since the association was reduced on adjustment for BMI. However, the cross-sectional nature of the analyses and the fact that BMI is more precisely measured than triglyceride limits the assumption that BMI completely explains the association between social class and triglyceride. The associations of socioeconomic position with blood lipids are known to be inconsistent from studies in middle-aged subjects; some studies found no association between triglycerides or HDL-cholesterol and socioeconomic position,[102;339] while others did.[227;259;333] Evidence for a relationship between socioeconomic position and total cholesterol in the literature has also been weak,[24;102;340] and if anything lower socioeconomic groups have been observed to have lower levels of total cholesterol in middle-age.[37;39;180] This was also observed in some studies in older populations.[218;219] The social class variations in blood pressure in the above results appeared to be weaker than the associations seen when the study participants were middle-aged.[37] While most studies in middle-aged men,[24;180;340] have demonstrated an inverse relation between socioeconomic position and blood pressure, there are fewer studies in older subjects. In ELSA, there did not appear to be a social class variation in hypertension in older men (>60 years), although among women (>60 years) the prevalence of hypertension was greater in those of routine/manual social classes compared to those in managerial groups.[119] Some studies report lower socioeconomic position to be associated with higher blood pressure in older age,[121;216;217] while another study reported a weak association in subjects ≥65 years.[218]

5.6.2.3 Social class and inflammatory/haemostatic markers

Various inflammatory and haemostatic markers are now known to be related to CHD risk not only in middle-age but also in the elderly.[12;194;232] Inflammatory and haemostatic markers are increasingly being seen as important potential influences on the relation between social class and coronary heart disease.[40] The results of this Chapter showed an inverse social class gradient in inflammatory and haemostatic markers independent of chronic diseases in older British men free from cardiovascular disease, diabetes or musculoskeletal disease. The gradients were, however, substantially explained by behavioural risk factors, particularly cigarette smoking, but also BMI and physical activity; the socioeconomic gradients were substantially attenuated for fibrinogen, fibrin-D dimer and WBC count, and also weakened for CRP, after adjustment for behavioural factors. These findings are consistent with some studies,[41;227;231] although other studies have reported a social class relationship of markers like CRP and fibrinogen independent of these risk factors.[40;213;223;225] It is well established that there are higher levels of inflammatory/haemostatic markers in cigarette smokers, those who are more inactive and those who are obese.[43;44;196;335] Because these behavioural factors are also graded by socioeconomic position,[85;97;180] they could have a strong confounding effect on the association of social class with inflammatory or haemostatic markers. While most of these previous studies are in middle-aged subjects, one study in an elderly population showed that behavioural risk factors were largely responsible for the relationship between inflammatory markers (CRP and IL-6) and socioeconomic position.[41] In the results of this Chapter, the social class gradient in the levels of some markers were only slightly diminished after taking behavioural risk factors into account; IL-6, plasma viscosity and platelet count had a modest independent relationship with social class, while the vWF–factor VIII complex had a stronger, consistent relation. Though a similar gradient existed for vWF in the Whitehall II study,[229] no such social class relationship was found in vWF and other haemostatic markers in the Caerphilly study.[230] In the Whitehall II study, a greater and more prolonged response of vWF and factor VIII to mental stress was also associated with low social class.[224] Although a graded association between social class and vWF independent of lifestyle/behavioural factors was observed in the present study, the cross-sectional nature of the data cannot establish a causal association between social class and vWF, nor can it provide direct evidence that vWF mediates the relationship between social class and CHD. To do this, prospective study information would be needed. There is also a possibility of residual confounding underlying the apparent relationship of haemostatic markers such as vWF with social class. Other confounding factors such as social area deprivation,[341] and genetic factors,[342] which have been reported to be related to higher levels of inflammatory or haemostatic markers, may explain the socioeconomic variation in these markers. Second, imprecision in the measurement/ascertainment of covariates such as smoking and physical activity, leading to less precise adjustments, may also result in residual confounding.

5.6.2.4 Social class and metabolic syndrome

The clustering of cardiovascular risk factors (hypertension, insulin resistance, hyperglycaemia and dyslipidemia) in the form of the metabolic syndrome,[200] has in recent years been found to be associated with increased CHD risk.[15] The metabolic syndrome has also been reported to be related to socioeconomic position.[14;42;237] Given that established coronary risk factors do not always substantially explain the socioeconomic gradient in CHD in middle-aged populations,[38;39] the metabolic syndrome has been postulated to be a possible biological mediator for socioeconomic inequalities in CHD.[14] Behavioural factors including physical inactivity and cigarette smoking, which are important risk factors for the metabolic syndrome,[45;241] are also related to socioeconomic position[261] and can, therefore, be expected to confound the association between socioeconomic position and metabolic syndrome. In the Whitehall II Study (subjects aged 39-63 years), lower compared to higher employment grades had about a two-fold greater risk of metabolic syndrome, although behavioural factors (smoking, physical exercise and alcohol consumption) made little contribution to this relationship.[14] Lower household wealth was also found to be associated with a greater risk of metabolic syndrome, independent of behavioural factors, in the Whitehall II Study.[244] Studies in Finnish and Danish populations, also reported socioeconomic inequalities in metabolic syndrome by educational levels, which remained after adjustment for behavioural factors.[233;242] However, evidence for the association between socioeconomic position and metabolic syndrome has not been entirely consistent.[14;42;233;236;237;244] In the National Survey of Health and Development, when the participants were aged 53 years, lower education was associated with an increased risk of metabolic syndrome, while social class was significantly associated in women but not in men; the influence of behavioural factors on these relationships was not reported.[243] A population-based study in South Korea, also found no significant variation in metabolic syndrome by educational or income levels in men, whereas stronger independent associations were observed in women.[237] Similarly, a weaker association between socioeconomic position and metabolic syndrome in men compared to women was observed in two other studies.[42;236] The influence of adult behavioural risk factors, particularly physical activity, on metabolic syndrome was found to be much stronger than that of social class, in a study in Newcastle, UK, comprising subjects aged 49-51 years.[241] Another study reported that behavioural factors modified the relationship between socioeconomic position and metabolic syndrome.[240] Most of the studies so far have been on middle-aged subjects. However, in the older men of the British Regional Heart Study an independent association between social class and insulin resistance or the metabolic syndrome was not observed. The results of this Chapter showed that behavioural risk factors (physical activity, smoking and alcohol consumption) were responsible for the relationship between social class and metabolic syndrome, as seen in other studies.[240;241] In the present study, the risk of lower social class groups having greater insulin resistance (high HOMA) was also diminished when behavioural risk factors were taken into account and was further attenuated by BMI. This lack of an independent relationship of social class with metabolic syndrome and insulin resistance is consistent with the previously reported finding in this cohort of the lack of an association between social class and incident type 2 diabetes,[343] which is strongly related to insulin resistance and metabolic syndrome.[204;344;345]

Of the individual components of the metabolic syndrome, social class had a particularly strong relationship with high waist circumference (central adiposity) in the above results, an effect that was independent of behavioural risk factors. This association of social class with central adiposity was stronger than with metabolic syndrome itself. Obesity or central adiposity has also been reported to be strongly associated both with social class,[14] and metabolic functioning.[45;346;347] While the metabolic syndrome has been widely reported to increase the risk of CHD, it has also been found that the syndrome itself may not predict CHD risk any more than the individual components.[348] It is, therefore, likely that the role of metabolic syndrome in socioeconomic inequalities in CHD is largely due to behavioural factors and central adiposity/obesity, which are important coronary risk factors in their own right.[349]

5.6.2.5 Social class and dietary factors

In the results of this Chapter, there was no evidence of social class differences in the dietary intakes of most of the CHD-related nutrients – dietary intake of total fat, cholesterol, saturated fat and polyunsaturated fat were not associated with social class, while the intakes of dietary fibre and of fresh fruit and vegetables increased from lower to higher social class. The lack of an association between social class and dietary fat is consistent with the lack of an association observed between social class and blood cholesterol. Dietary as well as plasma levels of vitamin C were lower in lower social class groups. These patterns of socioeconomic differences in dietary consumption (social class differences absent in fat intakes but present for fibre and fruit and vegetable consumption) observed in the present study are consistent with results from other British studies.[248;251;350] Socioeconomic differences in nutrients like vitamin C and fibre intake could reflect variations in food consumption patterns, such as the social class variations in fruit and vegetable intake. Previous reports have alluded to determinants of dietary behaviours such as nutritional knowledge, access to and availability of healthy food products, and aspects of local food environment.[351-353] Although vitamin C has been suggested to have a protective effect on coronary disease,[207] there is no evidence to substantiate this from large randomised trials.[208;209] Dietary fibre also has been found to have only a weak protective effect on CHD independent of other coronary risk factors.[354-358] Vitamin C and dietary fibre are, therefore, unlikely to play an important role in influencing the relationship between socioeconomic position and CHD. The weak association between dietary fat intake and socioeconomic position suggests that dietary factors have a limited role in contributing to socioeconomic differentials in CHD.[249;359] A decline in CHD mortality in social classes I and II between 1974 and 1981 in Britain was observed to correspond more closely with a decline in smoking rates in these social classes, while dietary fat intake patterns did not vary by social class.[249] Although adjustment for cigarette smoking did not appreciably alter the relation between social class and dietary factors in the results of this Chapter, strong interrelationships of smoking, and dietary patterns have been reported, with smokers having lower intake of fibre compared with non-smokers.[246] This highlights the greater potential contribution of smoking to socioeconomic variations in CHD than dietary factors.[246;359]

5.6.3 Interpretation of findings

Several established and novel coronary risk factors in older age were found to be related with socioeconomic position. In older men of the British Regional Heart Study, established behavioural coronary risk factors, particularly cigarette smoking, physical inactivity and obesity were more prevalent in lower social classes. Biological coronary risk factors, such as HDL-cholesterol in older age were also observed to show social class variations. Of the novel risk factors, haemostatic markers including vWF appeared to be related to social class independent of behavioural factors. The relationships of these coronary risk factors with socioeconomic position imply their potential for contributing to socioeconomic inequalities in older age.

5.6.4 Conclusions

Established as well as novel coronary risk factors are related to social class. Higher levels of adverse behavioural risk factors such as cigarette smoking, physical inactivity and obesity were observed in older British men who were of lower social class groups. Social class gradients in biological coronary risk factors were weak except for HDL-cholesterol, which was lower in lower social classes. Dietary fat intake did not show strong social class variations, although intakes of dietary fibre, vitamin C and of fresh fruit and vegetables decreased from higher to lower social classes. Metabolic syndrome was not related to social class independent of behavioural risk factors. The social class gradient in circulating inflammatory and haemostatic markers was to a large extent explained by the higher levels of adverse behavioural risk factors (particularly smoking) in lower social class groups. The contribution of established and novel coronary risk factors, including inflammatory and haemostatic markers, to the socioeconomic gradient in CHD in older age needs to be further evaluated in prospective analyses, as presented in Chapter 6.

Table 5.1 Relationship of social class with cigarette smoking in men aged 60-79 years in 1998-2000

	Social class						
	I	**II**	**III non-manual**	**III manual**	**IV**	**V**	**p for trend**
N	402	1128	432	1672	370	121	
Current smokers							
n (%)*	24 (6)	102 (9)	44 (10)	258 (15)	77 (21)	15 (12)	
OR (95%CI)†	1.00	1.58 (0.99, 2.50)	1.80 (1.07, 3.02)	2.90 (1.88, 4.47)	4.21 (2.6, 6.83)	2.21 (1.12, 4.36)	<0.0001
Never smokers							
n (%)*	198 (49)	380 (34)	146 (34)	370 (22)	93 (25)	25 (21)	
OR (95%CI)†	1.00	0.53 (0.42, 0.66)	0.53 (0.40, 0.7)	0.29 (0.23, 0.37)	0.35 (0.26, 0.48)	0.26 (0.16, 0.42)	<0.0001
Ex-smokers							
n (%)*	180 (45)	646 (57)	242 (56)	1044 (62)	200 (54)	81 (67)	
OR (95%CI)†	1.00	1.65 (1.31, 2.07)	1.56 (1.18, 2.05)	2.04 (1.64, 2.55)	1.42 (1.06, 1.88)	2.59 (1.68, 3.98)	<0.0001

*Cells show number of subjects reporting health behaviour (% of all those in that social class)
†OR=age-adjusted odds ratios; CI=confidence intervals

Table 5.2 Relationship of social class with alcohol consumption in men aged 60-79 years in 1998-2000

	Social class						
	I	**II**	**III non-manual**	**III manual**	**IV**	**V**	**p for trend**
Non-drinkers							
n (%)*	17 (4)	86 (8)	33 (8)	197 (12)	63 (17)	20 (17)	
OR (95%CI)†	1.00	1.84 (1.08, 3.13)	1.81 (0.99, 3.30)	2.98 (1.79, 4.95)	4.55 (2.60, 7.94)	4.67 (2.35, 9.27)	<0.0001
Occasional drinkers (<1 unit/week)							
n (%)*	76 (19)	263 (24)	132 (31)	474 (29)	110 (30)	34 (29)	
OR (95%CI)†	1.00	1.29 (0.97, 1.71)	1.83 (1.33, 2.53)	1.68 (1.28, 2.21)	1.80 (1.28, 2.51)	1.72 (1.07, 2.75)	<0.0001
Light/moderate drinkers (1-42 units/week)							
n (%)*	291 (74)	721 (65)	256 (59)	920 (56)	172 (47)	59 (50)	
OR (95%CI)†	1.00	0.65 (0.51, 0.84)	0.52 (0.39, 0.70)	0.45 (0.35, 0.58)	0.32 (0.24, 0.44)	0.35 (0.23, 0.53)	<0.0001
Heavy drinkers (>42 units/week)							
n (%)*	8 (2)	37 (3)	8 (2)	46 (3)	13 (4)	5 (4)	
OR (95%CI)†	1.00	1.67 (0.77, 3.62)	0.93 (0.34, 2.49)	1.40 (0.66, 2.99)	1.84 (0.75, 4.49)	2.09 (0.67, 6.51)	0.44

*Cells show number of subjects reporting health behaviour (% of all those in that social class)
†OR = age-adjusted odds ratios; CI=confidence intervals

Table 5.3 Relationship of social class with physical activity, obesity and body mass index (BMI) in men aged 60-79 years in 1998-2000

	Social class						
	I	**II**	**III non-manual**	**III manual**	**IV**	**V**	**p for trend**
Physically inactive							
n (%)*	103 (26)	331 (30)	163 (39)	588 (37)	144 (40)	55 (47)	
OR (95%CI)†	1.00	1.22 (0.94, 1.58)	1.77 (1.31, 2.39)	1.65 (1.29, 2.11)	1.87 (1.37, 2.55)	2.55 (1.66, 3.91)	<0.0001
Moderate-vigorous physical activity							
n (%)*	222 (56)	577 (53)	197 (47)	675 (42)	138 (39)	39 (33)	
OR (95%CI)†	1.00	0.87 (0.69, 1.1)	0.69 (0.53, 0.92)	0.58 (0.46, 0.72)	0.50 (0.37, 0.67)	0.37 (0.24, 0.58)	<0.0001
Obese (BMI≥30 kg/m^2)							
n (%)*	49 (12)	58 (14)	69 (16)	338 (20)	73 (20)	30 (25)	
OR (95%CI)†	1.00	1.18 (0.84, 1.67)	1.38 (0.93, 2.05)	1.85 (1.34, 2.55)	1.80 (1.22, 2.67)	2.35 (1.41, 3.91)	<0.0001
BMI (kg/m^2)							
Mean (standard error)	26.2 (0.18)	26.7 (0.11)	26.8 (0.18)	27.2 (0.09)	27.1 (0.19)	27.6 (0.34)	<0.0001

*Cells show number of subjects (% of all those in that social class)
†OR = age-adjusted odds ratios; CI=confidence intervals

Table 5.4 Age-adjusted levels of blood pressure and blood lipids according to social class in men aged 60-79 years in 1998-2000

	Social class						
	I	**II**	**III non-manual**	**III manual**	**IV**	**V**	**p for trend**
	Mean (SE)	Mean (SE)	Mean (SE)	Mean (SE)	Mean (SE)	Mean (SE)	
Systolic blood pressure	148 (1.19)	149 (0.71)	150 (1.15)	149 (0.59)	150 (1.24)	149 (2.19)	0.95
Diastolic blood pressure	85 (0.56)	85 (0.33)	85 (0.54)	85 (0.27)	86 (0.58)	83 (1.02)	0.55
Triglycerides*	1.54 (1.47, 1.61)	1.57 (1.53, 1.62)	1.65 (1.57, 1.73)	1.70 (1.66, 1.74)	1.60 (1.52, 1.68)	1.60 (1.47, 1.75)	0.0007
Total cholesterol	6.05 (0.05)	5.99 (0.03)	6.04 (0.05)	5.99 (0.03)	5.95 (0.06)	5.99 (0.10)	0.40
HDL-cholesterol	1.40 (0.02)	1.34 (0.01)	1.31 (0.02)	1.30 (0.01)	1.33 (0.02)	1.31 (0.03)	<0.0001
LDL-cholesterol	3.93 (0.05)	3.91 (0.03)	3.93 (0.05)	3.88 (0.02)	3.86 (0.05)	3.88 (0.09)	0.21

*Geometric mean (95% confidence interval)
SE=standard error

Table 5.5 Inflammatory and haemostatic markers according to social class in men aged 60-79 years in 1998-2000

	Social class						
	I	**II**	**III non-manual**	**III manual**	**IV**	**V**	**p for trend**
CRP - geometric mean*							
Age-adjusted	1.17 (1.02, 1.33)	1.39 (1.28, 1.51)	1.78 (1.56, 2.03)	1.74 (1.62, 1.86)	1.80 (1.55, 2.08)	1.64 (1.27, 2.12)	0.009
Age and behavioural factors†	1.36 (1.2, 1.55)	1.47 (1.36, 1.59)	1.83 (1.62, 2.08)	1.64 (1.54, 1.76)	1.59 (1.38, 1.83)	1.47 (1.15, 1.88)	0.02
Fully adjusted‡	1.37 (1.21, 1.55)	1.47 (1.36, 1.58)	1.82 (1.6, 2.06)	1.64 (1.54, 1.75)	1.62 (1.41, 1.87)	1.50 (1.17, 1.91)	0.02
Fibrinogen - mean (SE)							
Age-adjusted	3.13 (0.04)	3.14 (0.03)	3.27 (0.04)	3.29 (0.02)	3.31 (0.05)	3.27 (0.09)	<0.001
Age and behavioural factors†	3.20 (0.04)	3.17 (0.03)	3.29 (0.04)	3.26 (0.02)	3.24 (0.05)	3.22 (0.08)	0.07
Fully adjusted‡	3.20 (0.04)	3.17 (0.03)	3.29 (0.04)	3.26 (0.02)	3.24 (0.05)	3.23 (0.08)	0.05
Fibrin d-dimer - geometric mean*							
Age-adjusted	71.8 (65.6, 78.5)	73 (69, 77.2)	86 (78.5, 94.3)	81.5 (77.8, 85.5)	87.1 (78.7, 96.4)	80.2 (67.2, 95.8)	0.0002
Age and behavioural factors†	76.3 (69.6, 83.5)	76.3 (72.1, 80.7)	86.8 (79.3, 95)	80.1 (76.4, 84)	81.9 (74, 90.6)	76.5 (64.2, 91.3)	0.10
Fully adjusted‡	76.5 (69.9, 83.7)	74.1 (70.1, 78.4)	86 (78.6, 94.2)	80.4 (76.7, 84.3)	82.2 (74.3, 90.9)	77.9 (65.3, 92.8)	0.06
WBC - geometric mean*							
Age-adjusted	6.37 (6.17, 6.58)	6.64 (6.51, 6.78)	6.70 (6.49, 6.93)	6.80 (6.69, 6.92)	6.98 (6.73, 7.23)	6.56 (6.15, 6.99)	0.0006
Age and behavioural factors†	6.56 (6.36, 6.78)	6.73 (6.6, 6.87)	6.77 (6.55, 6.99)	6.72 (6.61, 6.84)	6.76 (6.52, 7)	6.45 (6.06, 6.86)	0.73
Fully adjusted‡	6.57 (6.36, 6.78)	6.73 (6.6, 6.87)	6.75 (6.54, 6.97)	6.72 (6.61, 6.83)	6.78 (6.54, 7.02)	6.42 (6.04, 6.83)	0.77

*Geometric mean (95% confidence interval)
†Behavioural factors include BMI, smoking, alcohol consumption and physical activity
‡Fully adjusted - adjusted for age, behavioural factors and blood pressure, HDL, triglycerides, insulin and glucose; SE= standard error

[Contd. in Table 5.6 overleaf]

Table 5.6 (Contd.) Inflammatory and haemostatic markers according to social class in men aged 60-79 years in 1998-2000

	Social class						
	I	II	III non-manual	III manual	IV	V	p for trend
IL6 - geometric mean*							
Age-adjusted	1.92 (1.78, 2.08)	2.12 (2.02, 2.22)	2.42 (2.24, 2.62)	2.51 (2.41, 2.61)	2.50 (2.3, 2.73)	2.65 (2.28, 3.08)	<0.0001
Age and behavioural factors†	2.09 (1.94, 2.25)	2.18 (2.08, 2.28)	2.46 (2.29, 2.65)	2.44 (2.34, 2.54)	2.33 (2.14, 2.53)	2.48 (2.15, 2.87)	<0.0001
Fully adjusted‡	2.09 (1.94, 2.25)	2.18 (2.08, 2.28)	2.45 (2.28, 2.64)	2.44 (2.34, 2.53)	2.35 (2.16, 2.55)	2.51 (2.17, 2.9)	<0.0001
von Willebrand factor- mean (SE)							
Age-adjusted	129 (3)	132 (2)	137 (3)	136 (1)	145 (3)	147 (5)	<0.0001
Age and behavioural factors†	132 (3)	133 (2)	137 (3)	135 (1)	143 (3)	145 (5)	0.002
Fully adjusted‡	131 (3)	132 (2)	137 (3)	136 (1)	144 (3)	145 (5)	0.0004
Factor VIII - mean (SE)							
Age-adjusted	126 (2)	127 (1)	130 (2)	132 (1)	137 (2)	137 (4)	<0.0001
Age and behavioural factors†	127 (2)	127 (1)	130 (2)	131 (1)	136 (2)	135 (4)	<0.0001
Fully adjusted‡	127 (2)	126 (1)	130 (2)	131 (1)	137 (2)	135 (4)	<0.0001
Plasma viscosity - mean (SE)							
Age-adjusted	1.27 (0.005)	1.27 (0.003)	1.28 (0.005)	1.29 (0.002)	1.29 (0.005)	1.29 (0.009)	<0.0001
Age and behavioural factors†	1.28 (0.005)	1.27 (0.003)	1.28 (0.005)	1.29 (0.002)	1.29 (0.005)	1.28 (0.009)	0.003
Fully adjusted‡	1.28 (0.005)	1.27 (0.003)	1.28 (0.005)	1.29 (0.002)	1.29 (0.005)	1.28 (0.009)	0.003
Platelet count - mean (SE)							
Age-adjusted	234 (4)	231 (2)	234 (4)	242 (2)	243 (4)	233 (7)	0.002
Age and behavioural factors†	235 (4)	232 (2)	234 (4)	242 (2)	240 (4)	229 (7)	0.03
Fully adjusted‡	235 (4	232 (2)	235 (4)	242 (2)	240 (4)	229 (7)	0.04

*Geometric mean (95% confidence interval)
†Behavioural factors include BMI, smoking, alcohol consumption and physical activity
‡Fully adjusted - adjusted for age, behavioural factors and blood pressure, HDL, triglycerides, insulin and glucose; SE= standard error

[Contd. in Table 5.7 overleaf]

Table 5.7 (Contd.) Inflammatory and haemostatic markers according to social class in men aged 60-79 years in 1998-2000

	Social class						
	I	**II**	**III non-manual**	**III manual**	**IV**	**V**	**p for trend**
tPA							
Mean (SE) - age-adjusted	10.64 (0.26)	10.47 (0.16)	10.63 (0.27)	10.84 (0.14)	10.32 (0.29)	11.39 (0.52)	0.25
Haematocrit							
Mean (SE) - age-adjusted	0.45 (0.002)	0.45 (0.001)	0.45 (0.002)	0.45 (0.001)	0.45 (0.002)	0.45 (0.004)	0.25
APC ratio							
Mean (SE) - age-adjusted	3.29 (0.03)	3.27 (0.02)	3.23 (0.03)	3.25 (0.02)	3.25 (0.03)	3.20 (0.06)	0.09
aPTT							
Geometric mean* - age-adjusted	30.8 (30.5, 31.2)	30.4 (30.2, 30.7)	30.3 (29.9, 30.7)	30.7 (30.5, 30.9)	30.6 (30.2, 31.0)	30.9 (30.2, 31.7)	0.38

*Geometric mean (95% confidence interval)
SE= standard error

Table 5.8 Metabolic syndrome according to social class in men aged 60-79 years in 1998-2000

	Social class						
	I	**II**	**III non-manual**	**III manual**	**IV**	**V**	**p for trend**
Metabolic syndrome - n (%)*	70 (23)	209 (24)	84 (27)	348 (31)	77 (29)	29 (33)	
OR (95%CI)							
Age-adjusted	1.00	1.08 (0.80, 1.48)	1.24 (0.86, 1.79)	1.47 (1.10, 1.98)	1.37 (0.94, 1.99)	1.64 (0.98, 2.76)	0.0005
Adjusted for age and behavioural factors†	1.00	1.02 (0.74, 1.40)	1.11 (0.76, 1.61)	1.27 (0.94, 1.73)	1.15 (0.78, 1.70)	1.22 (0.71, 2.08)	0.06

*Cells show number of subjects with metabolic syndrome (% of all those in that social class)
†Behavioural factors included smoking, physical activity and alcohol consumption
OR=odds ratio; CI=confidence intervals

Table 5.9 Individual components of the metabolic syndrome according to social class in men aged 60-79 years in 1998-2000

	Social class						
	I	**II**	**III non-manual**	**III manual**	**IV**	**V**	**p for trend**
High blood pressure n (%)*	245 (76)	695 (77)	293 (86)	963 (80)	226 (81)	73 (79)	
OR (95%CI) - age-adjusted	1.00	1.10 (0.82, 1.48)	2.05 (1.37, 3.07)	1.32 (0.99, 1.77)	1.32 (0.90, 1.95)	1.35 (0.76, 2.38)	0.03
Adjusted for age and behavioural factors†	1.00	1.11 (0.82, 1.50)	2.10 (1.40, 3.16)	1.34 (0.99, 1.82)	1.33 (0.89, 1.99)	1.34 (0.75, 2.40)	0.04
High glucose n (%)*	53 (17)	132 (15)	48 (15)	209 (18)	55 (21)	14 (16)	
OR (95%CI) - age-adjusted	1.00	0.86 (0.61, 1.22)	0.85 (0.55, 1.30)	1.07 (0.77, 1.49)	1.23 (0.81, 1.87)	0.93 (0.49, 1.77)	0.10
Adjusted for age and behavioural factors†	1.00	0.90 (0.63, 1.28)	0.87 (0.56, 1.34)	1.14 (0.80, 1.60)	1.27 (0.83, 1.96)	0.94 (0.49, 1.81)	0.08
High triglycerides n (%)*	117 (38)	352 (41)	129 (41)	529 (46)	111 (42)	37 (42)	
OR (95%CI) - age-adjusted	1.00	1.13 (0.86, 1.47)	1.15 (0.83, 1.59)	1.40 (1.08, 1.81)	1.18 (0.84, 1.65)	1.13 (0.70, 1.83)	0.03
Adjusted for age and behavioural factors†	1.00	1.11 (0.86, 1.45)	1.16 (0.85, 1.59)	1.31 (1.02, 1.69)	1.08 (0.78, 1.50)	0.98 (0.61, 1.56)	0.23
Low HDL-cholesterol n (%)*	46 (15)	159 (19)	56 (18)	248 (22)	39 (15)	24 (27)	
OR (95%CI) - age-adjusted	1.00	1.29 (0.90, 1.84)	1.24 (0.81, 1.90)	1.57 (1.11, 2.21)	0.98 (0.62, 1.56)	2.07 (1.18, 3.64)	0.04
Adjusted for age and behavioural factors†	1.00	1.17 (0.81, 1.69)	1.04 (0.68, 1.62)	1.28 (0.89, 1.83)	0.73 (0.45, 1.17)	1.55 (0.86, 2.79)	0.82
High waist circumference n (%)*	68 (21)	198 (22)	94 (28)	364 (31)	80 (29)	35 (38)	
OR (95%CI) - age-adjusted	1.00	1.06 (0.77, 1.44)	1.45 (1.02, 2.08)	1.65 (1.23, 2.21)	1.51 (1.04, 2.19)	2.28 (1.38, 3.76)	<0.0001
Adjusted for age and behavioural factors†	1.00	1.00 (0.73 1.38)	1.31 (0.90 1.89)	1.48 (1.09, 2.01)	1.33 (0.90, 1.96)	1.71 (1.02, 2.88)	0.0002

*Cells show number of subjects with the individual component of the metabolic syndrome (% of all those in that social class)
†Behavioural factors included smoking, physical activity and alcohol consumption. OR=odds ratio; CI=confidence intervals

Table 5.10 Top fourth of HOMA (insulin resistance) distribution according to social class in men aged 60-79 years in 1998-2000

	Social class						
	I	**II**	**III non-manual**	**III manual**	**IV**	**V**	**p for trend**
Top fourth of HOMA - n (%)*	50 (17)	150 (18)	53 (17)	208 (18)	51 (19)	29 (33)	
OR (95%CI)							
Age-adjusted	1.00	1.08 (0.76, 1.53)	1.05 (0.69, 1.60)	1.14 (0.81, 1.60)	1.23 (0.80, 1.89)	2.47 (1.44, 4.23)	0.02
Adjusted for age and behavioural factors†	1.00	1.04 (0.73, 1.49)	0.95 (0.62, 1.46)	1.04 (0.73, 1.48)	1.10 (0.71, 1.72)	1.98 (1.13, 3.46)	0.17

*Cells show number of subjects in the top fourth of HOMA distribution (% of all those in that social class)
†Behavioural factors included smoking, physical activity and alcohol consumption. OR=odds ratio; CI=confidence intervals

Table 5.11 Age-adjusted nutrient composition of dietary intake, and plasma vitamin C according to social class in men aged 60-79 years in 1998-2000 (dietary nutrients were adjusted for total calorie intake)

	Social class						
	I	II	III non-manual	III manual	IV	V	p for trend
Total fat (gm/day)*	66.5 (65.2, 67.9)	67.4 (66.6, 68.2)	67.8 (66.5, 69.2)	67.2 (66.5, 67.8)	67.5 (66.1, 68.9)	66.3 (63.9, 68.9)	0.93
Saturated fat (gm/day)*	26.5 (25.8, 27.3)	27.1 (26.7, 27.6)	27.2 (26.4, 27.9)	26.8 (26.4, 27.1)	27.1 (26.3, 27.9)	26.4 (25.1, 27.8)	0.76
Polyunsaturated fat (gm/day)*	9.7 (9.4, 9.9)	9.7 (9.5, 9.8)	10.1 (9.8, 10.4)	9.9 (9.7, 10.0)	9.9 (9.6, 10.2)	9.4 (8.9, 9.9)	0.20
Cholesterol (mg/day)*	250 (242, 258)	258 (253, 263)	253 (245, 260)	252 (248, 256)	251 (242, 259)	257 (242, 273)	0.48
Cereal fibre (gm/day)*	10.5 (9.9, 11.1)	9.8 (9.5, 10.1)	10.0 (9.5, 10.5)	9.2 (8.9, 9.4)	8.9 (8.5, 9.4)	8.4 (7.7, 9.2)	<0.0001
Vegetable fibre (gm/day) Mean (SE)	15.6 (0.2)	15.2 (0.1)	14.8 (0.2)	14.5 (0.1)	14.5 (0.2)	13.3 (0.4)	<0.0001
Total fibre (gm/day) Mean (SE)	27.5 (0.4)	26.5 (0.2)	26.4 (0.4)	25.3 (0.2)	25.0 (0.4)	23.5 (0.7)	<0.0001
Carbohydrates (gm/day) Mean (SE)	274 (2)	274 (1)	277 (2)	279 (1)	281 (2)	282 (3)	<0.0001
Energy (kcal/day) Mean (SE)	2099 (26)	2086 (15)	2097 (25)	2144 (13)	2108 (27)	2087 (48)	0.07
Vegetable consumption (average days/week)	5.39 (0.09)	5.17 (0.05)	4.89 (0.09)	4.76 (0.04)	4.82 (0.10)	4.45 (0.17)	<0.0001
Fruit consumption (average days/week)	5.56 (0.11)	5.32 (0.06)	5.16 (0.10)	5.09 (0.05)	5.12 (0.11)	4.98 (0.20)	<0.0001
Vitamin C (gm/day)*	85.3 (81.7, 89.0)	81.0 (78.9, 83.0)	77.0 (73.9, 80.3)	71.0 (69.5, 72.5)	68.8 (65.8, 72.0)	66.1 (61.1, 71.5)	<0.0001
Plasma vitamin C (µmol/L)*	27.1 (24.8, 29.7)	24.6 (23.3, 25.9)	24.1 (22.0, 26.3)	19.9 (19.0, 20.8)	17.8 (16.2, 19.6)	21.2 (17.9, 25.1)	<0.0001

*Geometric means (95% confidence interval); SE=standard error

Chapter 6

Socioeconomic position and CHD risk in older British men: contribution of established and novel coronary risk factors

6.1 Summary

The extent of socioeconomic inequalities in coronary heart disease (CHD) in older age and the pathways leading to these inequalities are not fully understood. In this Chapter, data from the British Regional Heart Study were used to assess the extent of socioeconomic inequalities in CHD in older age, and the contribution of established and novel coronary risk factors to these inequalities. The men, aged 60-79 years in 1998-2000, were followed-up for at least 6 years for CHD mortality and CHD incidence (fatal and non-fatal myocardial infarction). Several measures of socioeconomic position were examined including occupational social class, education, house and car ownership, and pension arrangements. Age at leaving full-time education, pension arrangements (state versus private), and house and car ownership were associated with CHD risk. However, these associations were largely attenuated after adjustment for occupational social class. Amongst the different measures of socioeconomic position, occupational social class showed the strongest associations with CHD risk. There was a graded relationship between social class (based on longest-held occupation recorded at 40-59 years) and both CHD incidence and mortality (both higher in manual social classes). Compared with social class I, the age-adjusted hazard ratio for CHD incidence for social class V was 2.70 (95%CI 1.37, 5.35; p for trend 0.008). Detailed analyses of the contributions

of established and novel risk factors were carried out for occupational social class. The hazard ratio for social class V compared with social class I was reduced to 2.14 (95%CI 1.06, 4.33; p for trend 0.11) after adjustment for behavioural risk factors (particularly cigarette smoking, and physical activity, BMI, alcohol consumption), which explained 38% of the social class gradient in relative risk (41% of the absolute risk gradient) for CHD incidence. After additional adjustment for novel coronary risk factors (C-reactive protein, interleukin-6 and von Willebrand factor), 55% of the relative risk and 59% of the absolute risk gradient in CHD incidence was explained (hazard ratio for CHD incidence in social class V compared to I was 1.88 95%CI 0.93, 3.81). Other established coronary risk factors (systolic blood pressure and lipids) made little difference to these estimates; similar results were observed for CHD mortality. Socioeconomic inequalities in CHD persisted in older age. Relative and absolute social class differences in CHD incidence and mortality were substantially explained by behavioural risk factors, and also by novel inflammatory markers.

6.2 Introduction

Differences in rates of coronary heart disease (CHD) according to socioeconomic position have been widely reported in several countries. When compared with those in higher socioeconomic position, people in lower socioeconomic positions have a greater CHD risk.[19;78] CHD is the single most important cause of morbidity and mortality in middle-aged and older men and shows a strong socioeconomic gradient in middle age.[6;37] Although both incidence and prevalence of CHD rise steeply with increasing age,[6] the extent to which socioeconomic inequalities in CHD persist in later life is not fully known.

The pathways through which socioeconomic inequalities in coronary heart disease operate also remain uncertain, particularly in later life. From studies on middle-aged populations, behavioural and biological cardiovascular risk factors, adverse socioeconomic circumstances across the life course, medication and treatment use, and psychosocial stress have been implicated as mediators of the relationship between socioeconomic position and CHD.[19;25;37-39] However, in observational studies, the contribution of established risk factors including smoking, physical inactivity, obesity and hypertension to socioeconomic inequalities in heart disease has generally been reported to be limited.[23;24;26;35;38;39;210;211;360;361] Most of these studies report relative inequalities between socioeconomic groups. More recently, studies have also attempted to understand the extent to which established coronary risk factors contribute to absolute socioeconomic differences in CHD risk as well as a relative ones.[35;36] These studies suggest that a greater proportion of absolute risk difference could be explained by established coronary factors than relative socioeconomic inequalities in CHD. Novel coronary risk factors including inflammatory and haemostatic markers such as C-reactive protein (CRP),[12] have in recent years been increasingly implicated as possible contributors to socioeconomic inequalities in heart disease.[41;212] However, most studies examining the contribution of coronary risk factors to socioeconomic inequalities in CHD were conducted in middle-aged subjects and little is known about whether the same factors contribute importantly to socioeconomic differences in older age. Measures of socioeconomic position in older age used in previous studies include education, income and occupational social class.[22;118-120] This Chapter investigates the extent of socioeconomic inequalities in CHD in older age using different indicators of socioeconomic position (education, house ownership, car ownership, pension arrangement and occupational social class). A better understanding of the extent of socioeconomic inequalities in CHD risk in later life (assessed in relative and absolute risks) and the role of underlying coronary risk factors (established and novel) would enable appropriate initiatives and policy action to be taken to reduce health inequalities in older age.

6.3 Objectives

The objectives of this Chapter are:

i) To examine the extent of socioeconomic inequalities in CHD incidence and CHD mortality, in older British men (aged 60-79 years), using different indicators of socioeconomic position.

ii) To investigate the extent to which established behavioural (cigarette smoking, alcohol consumption, body mass index and physical activity),[10;11] biological coronary risk factors (blood pressure, triglycerides, low density lipoprotein cholesterol, high density lipoprotein cholesterol)[10;11] and novel coronary risk factors (CRP, interleukin-6 and von Willebrand factor)[12;13;188] contribute to socioeconomic differences in CHD in older men in both relative and absolute terms.

6.4 Methods

The British Regional Heart Study was used to investigate the above objectives. In 1998-2000 all surviving men, now aged 60-79 years, were invited to a 20th year re-assessment, which included completion of a questionnaire on medical history and behavioural factors, a physical examination and collection of blood sample after a minimum 6 hour fast. 4252 men (77%) attended the examination and 4094 men (74%) had at least one measurement of the biological factors. The main outcomes for this Chapter were CHD incidence and CHD mortality. CHD incidence included non-fatal and fatal myocardial infarction cases. In accordance with the World Health Organisation criteria, non-fatal myocardial infarction was defined by the presence of at least two of – severe prolonged chest pain, ECG evidence of myocardial infarction and cardiac enzyme changes consistent with myocardial infarction.[283;284] This was ascertained by regular two-yearly reviews of general practitioner records including hospital and clinic correspondence. Information from death certificates using the *International Classification of Diseases*, 9th revision (ICD-9) was used to identify fatal myocardial infarction cases as deaths with code 410–414 (equivalent to ICD 10th revision codes I20–I25). For this Chapter outcome data from 1998-2000 until June 2006 was used.

6.4.1 Measures of socioeconomic position

The longest-held occupation of subjects at study entry when aged 40-59 years was used to define social class using the Registrar Generals' Social Class Classification (I, II, IIInon-manual, IIImanual, IV and V). Men in the Armed Forces [112 (2.6%)] were excluded from this analysis; information on social class was not available for 8 men. Information on car and house ownership was collected through questionnaires in 1998-2000; subjects were asked if they had a car available for their own use, and were asked to describe their accommodation as owned, rented from local authority, rented privately and other. Information on age at leaving full-time education and pension arrangements was collected through questionnaires in 1996. Subjects were asked whether their retirement support was or would be state pension only, occupational pension, or private pension.

6.4.2 Coronary risk factors

Behavioural (cigarette smoking, physical activity, alcohol consumption, body mass index), biological [systolic blood pressure, triglycerides, high-density lipoprotein-cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C)], and novel risk factors were measured at the re-examination in 1998-2000 as described in Chapter 3 (section 3.7.3 to 3.7.5 on page 82, and sections 3.8.1 to 3.8.2 on pages 85-86). Information on cigarette smoking collected in 1998-2000 and previous questionnaires, was used to classify subjects as never smokers, long-term ex-smokers (>20 years), ex-smokers who quit smoking 15-20 years ago, ex-smokers who quit smoking 10-15 years ago, ex-smokers who quit smoking 5-10 years ago, ex-smokers who quit smoking within 5 years, and current smokers. Based on their alcohol intake, subjects were classified into – none, occasional (<1 unit/week), light (1-15 units/week), moderate (16-42 units/week) and heavy (>42 units/week). Physical activity scores based on frequency and type of activity were derived and included 6 categories (none, occasional, light, moderate, moderately-vigorous and vigorous). Subjects in the categories of 'none' or 'occasional' activity were grouped together as 'inactive'. Novel coronary risk factors included CRP, interleukin-6 (IL-6) and von Willebrand factor (vWF), which are markers of inflammation and endothelial dysfunction related to coronary risk[12;13;188] and social class.[41] von Willebrand factor antigen levels were measured with enzyme-linked immunosorbent assays (DAKO, High Wycombe, UK). C-reactive protein was assayed by ultra-sensitive nephelometry (Dade Behring, Milton Keynes, UK). Interleukin-6 was assayed by a high-sensitivity ELISA (R and D Systems, Oxford, UK).

6.4.3 Rationale for analyses

The relationship between socioeconomic position and CHD risk (incidence and mortality) was examined using different measures of socioeconomic position including occupational social class, education, car and house ownership, and pension arrangement. Social class differences for CHD events were similar whether men with previous myocardial infarction were included in the analysis or not; men with previous myocardial infarction were retained in the analysis. Age at leaving full-time education was grouped into ≤14, 15-18 and >18 years. Information on accommodation was used to classify men into those who owned their house and those who did not. Pension arrangement was categorised into state pension only, and other pension arrangements including occupational or private pension. Analyses examining relative and absolute contributions of different risk factors to socioeconomic differences were carried out using occupational social class. Regression models included age and behavioural risk factors and were further adjusted for biological risk factors. Novel risk factors (CRP, IL-6, vWF) were individually adjusted for in addition to behavioural factors. Fully adjusted models included all of these risk factors.

There is evidence from studies in middle-aged populations, that a composite score combining different socioeconomic indicators can demonstrate greater mortality risks than individual measures of socioeconomic position.[97;362] Additional analyses was carried out by combining different socioeconomic indicators, to explore their association with CHD risk. A score [from 0 (highest socioeconomic position) to 5 (lowest socioeconomic position)] was combined by giving one point each to manual social class, not owning a house, not owning a car, having state pension only and full-time education till ≤14 years.

6.4.4 Statistical methods

Cox proportional hazards models were used to calculate hazard ratios with 95% confidence intervals (CI) for CHD incidence and CHD mortality according to different measures of socioeconomic position (occupational social class, education, car and house ownership, and pension arrangement). The proportionality assumption for Cox models was assessed by testing the Schoenfeld residuals,[363] and was found to be valid. Social class I was the reference category. Fitted as a continuous variable, regression coefficients and hazard ratios (95%CI) per unit increase for social class were also obtained. For the adjustments age, BMI, systolic blood pressure, triglycerides, LDL-cholesterol, HDL-cholesterol, CRP, IL-6 and vWF were fitted as continuous variables; social class (six levels), smoking (seven levels), physical activity (five levels) and alcohol intake (five levels) were fitted as ordinal variables. CRP and IL-6 distributions were positively skewed and required log transformation. The contribution of risk factors to the relative social class difference was calculated by $[(\beta 0-\beta 1)/\beta 0]*100$; where $\beta 0$ was age-adjusted log hazard ratio per unit increase in social class, and $\beta 1$ was the log hazard ratio adjusted for different risk factors.[28]

Survival probability at 6.5 years, the mean survival time, was calculated for each social class by applying average levels of age and risk factors to all social classes. Event probability for CHD incidence and CHD mortality was calculated as 1–survival probability, expressed as a percentage. Absolute social class difference explained by risk factors was calculated by [(AD0-AD1)/AD0]*100; where AD0 is the age-adjusted absolute difference in event probability between social classes I and V; AD1 is difference in event probability between social classes I and V adjusted for different risk factors. Approximate 95%CI for the estimates of relative and absolute risk explained in each model were calculated using bias-corrected bootstrap re-sampling of size 1000 to estimate the upper and lower limits.[306]

Population attributable risk fraction (PARF) comparing manual with non-manual social class was calculated for CHD incidence and CHD mortality using the formula $p(RR-1)/(1+p[RR-1])$, where p is the proportion of manual social class in the study population, and RR is the relative risk for CHD for manual compared with non-manual social classes (the relative risk was approximated by the relative hazard from Cox proportional hazards model[28;364]). PARF adjusted for coronary risk factors was obtained using hazard ratios adjusted for the different risk factors.

6.5 Results

Among 4132 men aged 60-79 years who attended the 20 year re-examination, complete information on all coronary risk factors was available for 3761 men (mean age 69 years). The age and social class distribution of this group did not differ from that of the original sample of 4132 men; both groups had a mean age of 69 years and included 48% subjects from non-manual social classes. The proportion of smokers was slightly greater (15%) in the group with missing data than in the group without missing data (12%); mean BMI and blood pressure levels were similar in the two groups. Missing information was largely due to unavailability of blood measurements in men who declined to provide blood samples. In the group of 3761 men, 274 incident (non-fatal and fatal) CHD cases and 191 CHD deaths occurred during a mean 6.5 years of follow-up.

6.5.1 Socioeconomic position and CHD risk

Table 6.1 describes the relation between occupational social class and other measures of socioeconomic position. The mean age at leaving full-time education was lower in manual social classes. A much greater proportion of manual compared with non-manual social classes had left full-time education at the age of 14 years or less; 57% in social class V and only 7% in social class I. Among manual social classes (social class IIImanual, IV and V), the proportions of men who did not own a house or a car were greater than in non-manual social classes (social classes I, II, IIInon-manual). The proportion who only had a state pension was also higher than in manual social classes compared to non-manual social classes.

Table 6.2 shows the relationships of occupational social class and education with CHD risk. A graded relation between occupational social class and the risk of CHD incidence and mortality was observed, with the lowest hazard in social class I and the highest hazard in social class V. The hazard ratio for CHD incidence increased from 1.00 in social class I to 1.40 (95%CI 0.87, 2.26) in social class III manual and was highest in social class V [2.70 (95%CI 1.37, 5.35); age-adjusted p for trend 0.008]. Compared to those with the highest level of full-time education (up to >18 years), men with the lowest level of full-time education (up to ≤14 years of age) had a greater risk of CHD incidence (age-adjusted hazard ratio 1.70, 95%CI 1.01, 2.86). The increased CHD risk in those who completed full-time education between 15-18 years of age (age-adjusted hazard ratio 1.67, 95%CI 1.00, 2.70) was not substantially different from those in the lowest level of education (≤14 years); the test for trend across educational levels was not significant (p 0.11). The association of educational level with CHD incidence was attenuated when adjusted for occupational social class. After adjustment for occupational social class, the hazard ratios for CHD incidence were reduced to 1.56 (95%CI 0.92, 2.66) for those with full-time education up to 15-18 years and to 1.50 (95%CI 0.85, 2.65) for those in full-time education up to ≤14 years (p values for trend across educational levels=0.37). These levels of education were also not related to CHD mortality; compared to those with the highest level of education, age-adjusted hazard ratio for CHD mortality was 1.15 (95%CI 0.66, 1.98) for those with the lowest level of education, and 0.96 (95%CI 0.55, 1.67) for those who left full-time education between 15-18 years of age (p for trend across educational levels 0.42). These non-significant associations of educational levels with CHD mortality were attenuated on adjustment for occupational social class; hazard ratios were 0.99 (95%CI 0.54, 1.82) for those with the lowest level of education, and 0.90 (95%CI 0.50, 1.60) for those who left full-time education between 15-18 years of age (p for trend across educational levels 0.85). Fitted as a continuous variable, age at leaving full-time education did not appear to be associated with CHD risk; the age-adjusted hazard ratio for every one year increase in age at leaving full-time education was 0.98 (95% CI 0.94, 1.01; p for trend 0.17) for CHD incidence and 1.00 (95% 0.97, 1.03; p for trend 0.93) for CHD mortality. There was some attenuation in the relationship between social class and CHD risk when adjusted for education; age-adjusted hazard ratio for CHD mortality attenuated from 1.15 (95%CI 1.02, 1.28) to 1.11 (95%CI 0.96, 1.28) on adjustment for education

Table 6.3 shows the relationship between house and car ownership and CHD risk. Men who did not own their house/accommodation had a higher risk of CHD incidence and CHD mortality compared with those who owned a house; age-adjusted hazard ratios (95%CI) were 1.42 (1.03, 1.96) for CHD incidence and 1.55 (1.08, 2.24) for CHD mortality. These effects were attenuated when adjusted for occupational social class; hazard ratios (HR) with 95%CI for those who did not own a house weakened to 1.30 (0.93, 1.80) for CHD incidence and 1.42 (0.97, 2.08) for CHD mortality. Those who did not own a car had a higher CHD risk compared to those who owned a car; hazard ratio (95%CI) was 1.39 (1.04, 1.86) for CHD incidence and 1.64 (1.18, 2.27) for CHD mortality. After adjustment for occupational social class, the risk of CHD incidence among those who did not own a car was attenuated (HR 1.26; 95%CI 0.93, 1.71), while the risk of CHD mortality remained marginally significant, though it was weakened (HR 1.50, 1.06, 2.08; see Table 6.3). In addition to theses results in Table 6.3, the effect of occupational social class on CHD risk altered marginally and remained significant when adjusted for house ownership and car ownership; hazard ratio for CHD incidence for a unit increase in social class was 1.14 (95%CI 1.04, 1.25) in age-adjusted analysis and was 1.11 (95%CI 1.01, 1.23) when adjusted for house ownership and car ownership.

Table 6.3 also shows the relationship between pension arrangement and CHD risk. Men who only received a state pension had a greater risk of CHD compared with men who also had occupational/private pensions; age-adjusted hazard ratios (95%CI) were 1.48 (1.11, 1.99) for CHD incidence and 1.40 (0.98, 2.00) for CHD mortality. After adjustment for occupational social class, the association of pension arrangement with CHD incidence was reduced, though it remained marginally significant (HR 1.38; 95%CI 1.02, 1.88; p value 0.04). The relationship of pension arrangement with CHD mortality was further reduced after adjustment for occupational social class (HR 1.28; 95% 0.88, 1.86). In addition to these results in Table 6.3, there did not appear to be a significant difference in CHD risk between men who had occupational or private pensions; the age-adjusted hazard ratio

(95%CI) for CHD incidence was 0.93 (0.66, 1.30) for those with occupational pensions compared to those with private pensions.

6.5.2 Contribution of coronary risk factors to relative social class difference in CHD

As seen in Table 6.2 and Table 6.3, occupational social class appeared to be the indicator of socioeconomic position with the strongest association with CHD risk. In age-adjusted analyses, social class V had more than two and a half times increased risk of CHD incidence and mortality compared to social class I (Table 6.2). Combining social classes I+II and IV+V also revealed social class differences in CHD risk – the hazard ratios (95%CI) for men in social classes IV+V compared to social classes I+II were 1.60 (1.11, 2.31) for CHD incidence and 1.62 (1.05, 2.49) for CHD mortality.

Table 6.4 shows the effect of adjustment for different coronary risk factors on the relationship between occupational social class and CHD. For every unit increase in social class, the age-adjusted CHD risk increased by 1.14 (95%CI 1.04, 1.25) for CHD incidence and by 1.15 (95%CI 1.02, 1.28) for CHD mortality. Adjustment for behavioural risk factors (cigarette smoking, physical activity, BMI and alcohol consumption) reduced these increased risks of CHD incidence and mortality with lower social class. Behavioural risk factors explained 38% (95% bootstrap CI 12%, 166%) of the increased relative risk for CHD incidence and 39% (95%CI 8%, 236%) for CHD mortality in lower social class groups. Half of the attenuation in the effect of social class on CHD incidence and mortality after adjustment for behavioural factors was caused by cigarette smoking (20%). Further adjustment for biological risk factors (systolic blood pressure, triglycerides, LDL-cholesterol and HDL-cholesterol) did not materially alter the results after adjustment for behavioural factors. Individual adjustment for CRP, IL-6 or vWF in addition to behavioural risk factors further attenuated the effect of social class – together with behavioural factors, CRP accounted for 46%, IL-6 for 47% and vWF for 47% of the relative risk difference in CHD incidence between social class groups. All the behavioural, biological and novel coronary risk factors together explained 55% (95%CI 22%, 214%) of the relative risk for CHD incidence and 56% (95%CI 15% to 273%) of the relative risk for CHD mortality in lower social classes. After adjustment for behavioural, biological and novel risk factors an increased risk of CHD in social class V compared with I remained, although it was no longer significant (hazard ratio for CHD incidence 1.88; 95%CI 0.93, 3.81).

6.5.3 Contribution of coronary risk factors to absolute social class difference in CHD

Event probability for CHD incidence and mortality at 6.5 years was graded according to social class (see Table 6.5); social class I had the lowest event probability (4.74% for CHD incidence) and social class V had the highest (8.90% for CHD incidence). The absolute difference in the probability of CHD incidence between social class I and V was 4.16% and the corresponding figure for CHD mortality was 2.78%. Adjusting for behavioural risk factors explained 41% (95%CI 18%, 132%) of the absolute risk difference in the probability of CHD incidence between social classes I and V, and behavioural risk factors explained 45% of the absolute risk difference in CHD mortality. Further adjustment for biological risk factors (systolic blood pressure, triglycerides, LDL-cholesterol and HDL-cholesterol) did not make additional contributions to the absolute difference in CHD risk between social classes that was explained by behavioural factors. Additional adjustment for novel risk factors increased the proportions of risk explained slightly. When added to behavioural risk factors, adjustment for CRP explained 49%, IL-6 51% and vWF 51% of the absolute social class difference in the probability of CHD incidence. In combination, all the risk factors (behavioural, biological and novel) together accounted for 59% (95%CI 33%, 312%) of the absolute social class difference in risk of CHD incidence, and 63% (95%CI -153%, 162%) for CHD mortality. Limited power resulted in wide Bootstrap 95% confidence intervals for the analyses both for relative and absolute social class difference explained. Some confidence intervals reported give upper bounds of >100%, while other give lower bounds of <0% (negative). A value for percentage explained >100% implies that if the risk factors were equally distributed in the population, the CHD risk in manual social classes may be lower than that in non-manual social classes. A negative lower bound confidence limit suggests that the disadvantage of manual social class would be even more marked after adjustment for risk factors. At the 95% confidence level, the upper and lower bound indicate the best and worst possible contribution of risk factors; the point estimates give the best estimate of the contribution of risk factors obtained in these analyses.

6.5.4 Combining socioeconomic measures

Additional analyses in the present study revealed that combining different socioeconomic indicators was associated with increased CHD risk. A score combined [from 0 (highest socioeconomic position) to 5 (lowest socioeconomic position)] by giving one point each to manual social class, not owning a house, not owning a car, having state pension only and full-time education till ≤14 years, was associated with increased risk of CHD incidence. The age-adjusted hazard ratio for CHD incidence increased from 1.00 in the lowest score group (highest socioeconomic group) to 1.55 (95%CI 0.95, 2.60) in the lowest socioeconomic group (combined score >3). The age-adjusted hazard ratio for CHD incidence per unit increase in the score was 1.13 (95%CI 1.03, 1.24); p for trend 0.01. This was reduced to 1.08 (95%CI 0.98, 1.19) after adjustment for behavioural risk factors (smoking, physical activity, BMI and alcohol consumption). The magnitude of the association of the combined socioeconomic score and CHD risk was not greater than that observed between occupational social class and CHD risk. The strength of the association between the combined socioeconomic score and CHD risk (per unit increase in the score) was similar to that observed for occupational social class. Moreover, the contribution of behavioural risk factors to relative socioeconomic differences in CHD incidence using the combined score was similar (34%) to that observed for occupational social class.

6.5.5 Population attributable risk fractions

Table 6.6 shows the population attributable risk fractions (PARF) from manual social classes for CHD incidence and CHD mortality; these indicate the population risk for CHD incidence or mortality attributable to the excess risk in manual compared with non-manual social classes. Table 6.6 also shows the PARF for CHD adjusted for different risk factors and the contribution of these risk factors in reducing the PARF from manual social class. The age-adjusted population attributable risk fraction (PARF) for manual versus non-manual social classes was 12% for CHD incidence and 15% for CHD mortality. Adjustment for behavioural risk factors reduced the PARF to 7% for CHD incidence and 10% for CHD mortality, thus accounting for 41% of the PARF (manual versus non-manual groups) for CHD incidence and 34% for CHD mortality. Further adjustment for biological risk factors did not alter these attributable risk fractions. Adjusting for CRP, IL-6 and vWF individually in addition to behavioural factors further reduced the PARF slightly; all together these risk factors with behavioural factors explained 56% of the PARF from manual social class for CHD incidence and 52% for CHD mortality. These results are similar to the estimated contribution of risk factors to the relative and absolute social class differences reported in sections 6.5.2 and 6.5.3.

6.6 Discussion

In this prospective study of men aged 60-79 years, socioeconomic inequalities in CHD persisted in older age. The risk of CHD was almost threefold greater in the lowest compared with highest social class, and the absolute difference in risk of CHD incidence at 6.5 years was 4%. An appreciable proportion of the increased relative and absolute risk of CHD in lower social class groups was explained by behavioural factors, especially cigarette smoking, and also BMI, physical activity and alcohol consumption. Novel coronary risk factors including CRP, IL-6 and vWF also contributed to the inequalities in CHD in older age. Biological risk factors (blood pressure and lipids) made little contribution to these socioeconomic inequalities.

6.6.1 Strength and limitations of findings

A major strength of the results presented in this Chapter is that the findings are from a socioeconomically representative cohort of older British men with high rates of follow-up. Another strength of the findings is the wide range of socioeconomic measures used to assess the socioeconomic inequalities in CHD risk in older age. Occupational social class, education, house and car ownership, and pension arrangements were used to measure socioeconomic position and its association with CHD in older age. Socioeconomic inequalities in CHD risk in older age were present not only across occupational social class groups but also according to education, car and house ownership and pension arrangements. Those with state pensions only had a greater CHD risk than those who also had occupational or private pensions. Those who did not own a car or house had a higher risk of CHD compared with those who did. Car and house ownership have been used as proxy measures of income and wealth in previous studies.[365] Taken together with occupation, car ownership has been reported to reveal stronger mortality differentials in middle-aged populations.[97;362] However, in the above results, the effects of education, car or house ownership and pension arrangements were largely attenuated when adjusted for occupational social class. As seen in the present study, these measures are influenced by other indicators of socioeconomic position such as occupational social class. Car and house ownership in older age can also be influenced also by health status. Poor health can limit car usage, and housing arrangements can also change in the elderly. Education is often used as a marker of socioeconomic position due to its advantage of being easy to measure and its stability over adult life.[80;365] However, although previous studies in older populations have reported differences in CHD according to education,[22;29] the extent of differentiation in CHD risk by education appeared to be limited in the present study. There was no evidence of a consistent relationship across different levels of education and CHD risk. The increased risk of CHD in those who left full-time education at the age of ≤14 years was not substantially different from those who in stayed in full-time education till 15-18 years of age. A unit increase in year of full-time education also showed no evidence of an association with CHD risk in this older population. Occupational social class has been reported by other studies to be a better discriminator of mortality than education, with education being more a marker of early life socioeconomic position.[366] In the present study, occupational social class appeared to have the strongest associations with CHD risk compared with the other measures of socioeconomic position. Combining different socioeconomic measures did not reveal relative risks for CHD that were greater in magnitude than that observed across occupational social class groups. Therefore, it seemed appropriate to use occupational social class as the main measure of socioeconomic position to investigate the role of risk factors in socioeconomic inequalities in CHD in older age. The social class measure, based on the longest-held occupation during middle age (40-59 years), is a particularly stable indicator of socioeconomic position over most of adult life, which would also determine socioeconomic conditions in older age; a repeat assessment of social class before retirement indicated a very low proportion (9%) of marked social class change as reported in Chapter 3 (section 3.9.1 page 87).[28] The use of such a measure overcomes the difficulties of measuring socioeconomic position directly in later life.[365] However, since the study included only older men, the generalisability of the findings to younger subjects and to women may well be limited. Although limited numbers of events resulted in wide confidence intervals, it is nevertheless useful to have estimates to quantify the likely contribution of coronary risk factors to socioeconomic inequalities in CHD.

6.6.2 Comparison with previous studies

The persistence of socioeconomic inequalities in CHD in older age is consistent with previous studies.[22;118;119;121] In the present study CHD risk was nearly three-fold greater in the lowest compared with highest social class group, while other lower/manual social classes had about a 40-60% greater CHD risk. The absolute difference in CHD incidence risk between the highest and lowest social classes was 4%; for every 100 men followed-up for a mean period of 6.5 years in each of the highest and lowest social classes, 4 extra CHD events would be expected in the lowest social class group. Previous studies in older populations have not reported the magnitude of socioeconomic differences in CHD in absolute terms. A 20-50% greater relative risk of CHD in lower compared with higher socioeconomic groups has been previously reported in older age populations aged over 60 years.[22;26;118;119;121] These variations in the extent of inequalities previously reported could be due to the use of different socioeconomic measures including education and occupational social class.[22;118;119;121] Also different ways of classifying these measures and different groups for comparison have been used such as higher versus lower educational levels, highest versus lowest employment grade or social class, and non-manual versus manual social class.[22;26;118;119;121] In these studies the increased CHD risk in lower socioeconomic groups in older age was smaller than in middle-age.[22;118;119] It is nevertheless, clear that socioeconomic inequalities in CHD are present in older age and are not restricted to middle-age.

Explanations of socioeconomic inequalities in health have been widely researched in middle-aged populations. Studies with middle-aged participants mostly show that relative socioeconomic inequalities in CHD are not largely accounted by established coronary risk factors,[38;39;210] although some studies report that coronary risk factors such as cigarette smoking and blood pressure have a substantial influence on socioeconomic inequalities in CHD.[28;37;211] Recent studies in middle-aged populations suggest that coronary risk factors (smoking, blood pressure and cholesterol) can also explain a substantial proportion of absolute socioeconomic differences in CHD risk.[35;36;222] However, little is known about the determinants of socioeconomic inequalities in CHD in older age. A Swedish population-based cohort aged ≥65 years found adjustment for coronary risk factors (smoking, physical activity, BMI, hypertension and diabetes) to attenuate the increased risk in manual social class groups.[121] However, in a prospective study comprising older Danish men (mean age 63 years), established cardiovascular risk factors (blood pressure, smoking, lipids and physical activity) made only a small contribution to the relative social difference in CHD risk.[26] A study in South Korea showed much weaker socioeconomic inequalities in 55-64 year old men.[36] This study however, comprised public servants, a more homogenous group than the participants of the present study. The relative and absolute contributions of both established and novel risk factors to socioeconomic inequalities in CHD risk in older subjects (60-79 years) with a mean age over 65 years presented in this Chapter have not been previously reported.

Social class differences in behavioural coronary risk factors including cigarette smoking (the most important single factor), physical activity, BMI and alcohol consumption made an important contribution to the increased risk of CHD in lower social class groups, accounting for about 38% of the relative risk and over 40% of absolute risk. Another study has also observed that cigarette smoking was the largest contributor to socioeconomic inequalities in CHD.[36] Established coronary risk factors such as blood pressure, HDL-C, LDL-C and triglycerides made little contribution above that of the behavioural factors, possibly due to their weaker relationships with CHD with increasing age.[123;124] Novel cardiovascular risk factors including CRP, IL-6, and vWF explained about a further 10% of the relative socioeconomic inequalities in CHD risk in addition to behavioural risk factors. Taken together, both health behaviours and novel risk factors together explained about 55% of the relative and about 60% of the absolute social class inequalities in CHD. An increased CHD risk in the lowest social class group (social class V) remained after taking into account behavioural and novel risk factors, although it was not statistically significant. Studies examining the possible contribution of novel coronary risk factors such as inflammatory markers, to socioeconomic inequalities in CHD are limited and no previous study has investigated this in older populations. The Women's Health Study showed that CRP and fibrinogen explained little of the socioeconomic differences in cardiovascular disease beyond the effect of traditional coronary risk factors in middle-aged women.[212] The results of studies in middle-aged populations suggest that a greater proportion of absolute risk difference can be explained by established coronary risk factors even though the relative risk explained can be limited.[35;222] The limited contribution of established coronary risk factors such as blood lipids to relative socioeconomic inequalities may reflect the weak socioeconomic variations frequently observed in these risk factors.[35] In the present study, the contributions of risk factors to absolute social class differences were only slightly greater than contributions to relative inequalities. This difference in results between studies could be due to different methods used to quantify the contribution of risk factors. In the present study, average levels of risk factors in the cohort were applied to all participants to assess the influence of risk factors to absolute inequalities, whereas other studies have used a more optimistic scenario of complete removal of risk factors, or risk reductions achieved from best-practice interventions (smoking cessation, reduction of blood pressure and cholesterol).[35;36;222] Nonetheless, an appreciable substantial proportion of absolute social class inequalities (about 40%) was explained by established behavioural factors in the present study.

This was also reflected in the results on population attributable risk fractions – behavioural risk factors made the largest contribution to reducing the population risk for CHD attributable to manual social classes, while novel coronary risk factors made a modest additional contribution. If manual social classes had the same CHD risk as non-manual groups 12% of all CHD events could have been prevented. This population risk attributable to social class differences would be reduced to 7% if behavioural factors in manual social classes were similar to non-manual groups – implying a 41% contribution of behavioural risk factors.

6.6.3 Interpretation of findings

Findings of this Chapter suggest that marked socioeconomic inequalities in CHD persist in older British men. Occupational social class, based on longest-held occupation in middle-age, was a good indicator of these inequalities. CHD risk increased from the highest to the lowest social class. Behavioural coronary risk factors, particularly smoking, made a substantial contribution to these inequalities, both in relative and absolute terms (about 40%). The importance of these risk factors in terms of relative socioeconomic inequalities would be in understanding the aetiology of these inequalities, while the contribution of these risk factors to absolute socioeconomic differences implies their public health potential to reducing these inequalities in CHD in older age.[35]

The contribution of inflammatory/haemostatic markers, to the association between socioeconomic inequalities and CHD risk remains uncertain. The potential contribution of these markers to socioeconomic inequalities in CHD is based on the premise that these markers are related to increased CHD risk as well as to socioeconomic position. It is biologically plausible that this association may partly reflect the effects of psychosocial stresses on atherothrombosis,[12] and acute stress responses for IL-6 and vWF have been documented.[224;367] There is increasing evidence from prospective studies and from meta-analyses of prospective studies that inflammatory markers (CRP, IL-6 and vWF) are associated with increased risk of CHD.[12;13;188] However, the causal relations of inflammatory and haemostatic markers with CHD risk independent of established coronary risk factors and socioeconomic factors are not fully established.[12;368] Moreover, the relation of these markers with socioeconomic position has been reported to be confounded by behavioural risk factors such as smoking,[41;227] as also presented in Chapter 5. High levels of inflammatory markers in older age have been shown to be strongly related to morbidity, and to coronary risk factors including smoking, physical activity, blood pressure and dyslipidemia.[43;196;232;369] It is, therefore, possible that the contribution of inflammatory/haemostatic markers including CRP and IL-6 to socioeconomic inequalities in CHD reflect some underlying disease processes in the elderly, and the role of behavioural coronary risk factors. Even if these novel risk factors are considered to be independent contributors to socioeconomic inequalities, their contribution was less than that of behavioural risk factors in the present study.

Although the established and novel coronary risk factors together explained a substantial proportion of the socioeconomic inequalities, the full extent of inequalities in CHD in the above results was not accounted for by these factors. A possible explanation for this could be imprecision in measurement of risk factors such as smoking and blood pressure, leading to an underestimation of their contribution. Single measurements of these risk factors can fail to capture the cumulative effect of adverse lifestyle which is influenced by socioeconomic conditions over the life course.[46;261] It is also possible that psychosocial stress is playing a role. However, if it were important, it would need to be operating through pathways other than those involving behavioural and other established coronary risk factors.

6.6.4 Conclusions

Little is known about the extent and determinants of socioeconomic inequalities in CHD in later life. This Chapter investigated the extent of socioeconomic inequalities in CHD in older age. Data from the BRHS showed that socioeconomic inequalities persisted in 60-79 years old men who were followed up for at least 6 years – nearly a three-fold greater CHD risk was present in the lowest compared with highest social class, and a 4% absolute difference in CHD risk between these groups. Socioeconomic inequalities were seen for different measures including social class, house and car ownership and by pension arrangements. Behavioural risk factors (particularly cigarette smoking, and also physical activity, BMI and alcohol consumption) made substantial contributions to both the relative and absolute social class differences in CHD. Novel risk factors (inflammatory markers) made some additional contribution to social class inequalities in CHD in older men.

Table 6.1 Relationship between occupational social class and other measures of socioeconomic position in men aged 60-79 years in 1998-2000

Social class	**Age at leaving full-time education** Mean (SD)	**Proportion leaving full-time education ≤14 years** n (%)	**Not a house owner** n (%)	**Not a car owner** n (%)	**State only pension** n (%)
I	18 (6)	23 (7)	13 (4)	15 (4)	11 (3)
II	17 (5)	213 (22)	50 (5)	77 (8)	85 (9)
III non-manual	16 (5)	110 (31)	37 (10)	52 (14)	42 (12)
III manual	15 (5)	643 (50)	243 (16)	307 (21)	361 (27)
IV	15 (5)	155 (56)	82 (25)	109 (33)	86 (30)
V	15 (4)	50 (57)	32 (30)	37 (34)	31 (34)
p for trend	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

SD=standard deviation

Table 6.2 CHD (incidence and mortality) according to social class and education in men aged 60-79 years in 1998-2000

	n	CHD incidence			CHD mortality		
		Rate/1000 person years	Age-adjusted	Further adjusted for education/social class	Rate/1000 person years	Age-adjusted	Further adjusted for education/social class
Social Class			HR (95%CI)			HR (95%CI)	
I	372	8.1	1.00	1.00	5.6	1.00	1.00
II	1035	10.1	1.21 (0.74, 1.99)	1.33 (0.68, 2.61)	7.1	1.20 (0.66, 2.17)	1.24 (0.72, 2.13)
III non-manual	381	10.6	1.26 (0.71, 2.25)	1.22 (0.55, 2.71)	5.8	0.96 (0.47, 1.99)	1.40 (0.75, 2.61)
III manual	1525	11.6	1.40 (0.87, 2.26)	1.42 (0.71, 2.84)	8.2	1.39 (0.79, 2.46)	1.31 (0.75, 2.29)
IV	336	13.3	1.60 (0.90, 2.84)	1.38 (0.58, 3.30)	9.4	1.59 (0.80, 3.15)	1.25 (0.62, 2.52)
V	112	20.5	2.70 (1.37, 5.35)	3.10 (1.16, 8.31)	14.4	2.77 (1.23, 6.24)	2.57 (1.14, 5.79)
HR (95%CI) per unit social class			1.14 (1.04, 1.25)	1.08 (0.96, 1.22)		1.15 (1.02, 1.28)	1.11 (0.96, 1.28)
p for trend			0.008			0.02	
Age of leaving full-time education			HR (95%CI)	HR (95%CI)		HR (95%CI)	HR (95%CI)
>18 years	431	5.9	1.00	1.00	5.5	1.00	1.00
15-18 years	1697	9.1	1.67 (1.00, 2.80)	1.56 (0.92, 2.66)	4.8	0.96 (0.55, 1.67)	0.90 (0.50, 1.60)
≤14 years	1194	13.6	1.70 (1.01, 2.86)	1.50 (0.85, 2.65)	10.0	1.15 (0.66, 1.98)	0.99 (0.54, 1.82)
Trends across groups			0.11	0.37		0.42	0.85
HR (95%CI) per unit social class			0.98 (0.94, 1.01)	0.99 (0.95, 1.02)		1.00 (0.97, 1.03)	1.01 (0.98, 1.04)
p for trend per year of education			0.17	0.36		0.93	0.62

HR=hazard ratio; CI=confidence intervals

Table 6.3 CHD (incidence and mortality) according to house ownership, car ownership and pension arrangements in men aged 60-79 years followed-up from 1998-2000 to 2006

		CHD incidence			CHD mortality		
			Age-adjusted	Adjusted for social class		Age-adjusted	Adjusted for social class
	n	Rate/1000 person years	HR (95%CI)	HR (95%CI)	Rate/1000 person years	HR (95%CI)	HR (95%CI)
House ownership							
Yes	3201	10.4	1.00	1.00	7.0	1.00	1.00
No	457	16.4	1.42 (1.03, 1.96)	1.30 (0.93, 1.80)	12.7	1.55 (1.08, 2.24)	1.42 (0.97, 2.08)
p value			0.03	0.13		0.02	0.07
Car ownership							
Yes	3105	10.3	1.00	1.00	6.7	1.00	1.00
No	597	16.5	1.39 (1.04, 1.86)	1.26 (0.93, 1.71)	13.6	1.64 (1.18, 2.27)	1.50 (1.06, 2.11)
p value			0.03	0.13		0.003	0.02
Pension arrangement							
Occupational/private	2801	9.6	1.00	1.00	6.4	1.00	1.00
State only	616	15.3	1.48 (1.11, 1.99)	1.38 (1.02, 1.88)	10.0	1.40 (0.98, 2.00)	1.28 (0.88, 1.86)
p value			0.009	0.04		0.07	0.20

HR=hazard ratio; CI=confidence intervals

Table 6.4 Hazard ratios (95%CI) for CHD (incidence and mortality) according to social class and the effect of adjustment for established and novel coronary risk factors in men aged 60-79 years followed-up from 1998-2000

Social Class	Age-adjusted	Age and behavioural factors[1]	Age, behavioural and biological risk factors[2]	Age, behavioural factors and CRP	Age, behavioural factors and IL-6	Age, behavioural factors and vWF	All risk factors
				CHD incidence			
I	1.00	1.00	1.00	1.00	1.00	1.00	1.00
II	1.21 (0.74, 1.99)	1.13 (0.68, 1.86)	1.10 (0.67, 1.82)	1.09 (0.66, 1.80)	1.12 (0.68, 1.85)	1.12 (0.68, 1.85)	1.08 (0.65, 1.78)
III non-manual	1.26 (0.71, 2.25)	1.16 (0.65, 2.08)	1.12 (0.62, 2.01)	1.09 (0.61, 1.96)	1.13 (0.63, 2.02)	1.13 (0.63, 2.02)	1.05 (0.58, 1.88)
III manual	1.40 (0.87, 2.26)	1.23 (0.75, 2.00)	1.20 (0.74, 1.95)	1.16 (0.71, 1.89)	1.16 (0.71, 1.89)	1.19 (0.73, 1.95)	1.11 (0.68, 1.82)
IV	1.60 (0.90, 2.84)	1.22 (0.68, 2.21)	1.23 (0.68, 2.22)	1.17 (0.65, 2.11)	1.19 (0.66, 2.15)	1.17 (0.65, 2.11)	1.14 (0.63, 2.06)
V	2.70 (1.37, 5.35)	2.14 (1.06, 4.33)	2.08 (1.03, 4.19)	2.04 (1.01, 4.11)	2.08 (1.03, 4.18)	2.02 (0.97, 4.08)	1.88 (0.93, 3.81)
Per unit social class	1.14 (1.04, 1.25)	1.08 (0.98, 1.19)	1.08 (0.98, 1.19)	1.07 (0.97, 1.18)	1.07 (0.97, 1.18)	1.07 (0.97, 1.18)	1.06 (0.96, 1.17)
p for trend	0.008	0.11	0.11	0.16	0.17	0.17	0.25
% Attenuation in age-adjusted relative risk per unit social class after adjustment for risk factors*		38%	38%	46%	47%	47%	55%
				CHD mortality			
I	1.00	1.00	1.00	1.00	1.00	1.00	
II	1.20 (0.66, 2.17)	1.11 (0.61, 2.01)	1.09 (0.60, 1.98)	1.06 (0.58, 1.93)	1.11 (0.61, 2.03)	1.07 (0.60, 1.98)	1.07 (0.59, 1.95)
III non-manual	0.96 (0.47, 1.99)	0.86 (0.41, 1.79)	0.83 (0.40, 1.74)	0.78 (0.37, 1.64)	0.83 (0.40, 1.73)	0.81 (0.39, 1.70)	0.77 (0.37, 1.61)
III manual	1.39 (0.79, 2.46)	1.19 (0.66, 2.13)	1.18 (0.66, 2.11)	1.11 (0.62, 1.99)	1.12 (0.62, 2.01)	1.14 (0.64, 2.05)	1.07 (0.60, 1.92)
IV	1.59 (0.80, 3.15)	1.21 (0.60, 2.44)	1.23 (0.61, 2.48)	1.15 (0.57, 2.32)	1.20 (0.60, 2.43)	1.14 (0.56, 2.30)	1.14 (0.56, 2.31)
V	2.77 (1.23, 6.24)	2.13 (0.92, 4.91)	2.06 (0.90, 4.76)	2.01 (0.87, 4.65)	2.12 (0.92, 4.88)	2.00 (0.87, 4.63)	1.88 (0.81, 4.36)
Per unit social class	1.15 (1.02, 1.28)	1.09 (0.97, 1.22)	1.09 (0.97, 1.23)	1.08 (0.96, 1.21)	1.07 (0.95, 1.21)	1.07 (0.95, 1.21)	1.06 (0.94, 1.20)
p for trend	0.02	0.17	0.16	0.23	0.24	0.24	0.32
% Attenuation in age-adjusted relative risk per unit social class after adjustment for risk factors*		39%	37%	46%	46%	48%	56%

[1]Behavioural factors=smoking, alcohol consumption, physical activity and BMI; [2]Biological risk factors=systolic blood pressure, triglycerides, LDL-cholesterol, HDL-cholesterol;

*[(β0- β1)/ β0]*100; β0=age-adjusted log hazard ratio per unit increase in social class, β1=log hazard ratio per unit increase in social class additionally adjusted for risk factors

Table 6.5 Event probability (%) for CHD (incidence and mortality) according to social class at 6.5 years follow-up from 1998-2000 and the effect of adjustment for established and novel coronary risk factors on the absolute social class difference in event probability

Social Class	Age-adjusted	Age and behavioural factors[1]	Age, behavioural and biological risk factors[2]	Age, behavioural factors and CRP	Age, behavioural factors and IL-6	Age, behavioural factors and vWF	All risk factors
CHD incidence							
I	4.74	5.08	4.92	5.12	5.12	5.16	5.05
II	5.38	5.50	5.33	5.49	5.48	5.52	5.35
III non-manual	6.11	5.95	5.78	5.88	5.86	5.91	5.67
III manual	6.93	6.44	6.26	6.30	6.71	6.32	6.01
IV	7.86	6.96	6.78	6.75	6.55	6.75	6.37
V	8.90	7.53	7.34	7.23	7.17	7.22	6.75
% Attenuation in absolute difference between social class I and V after adjustment for risk factors*		41%	42%	49%	51%	51%	59%
CHD mortality							
I	2.85	2.91	2.81	2.91	2.87	2.95	2.83
II	3.27	3.17	3.07	3.13	3.08	3.17	3.01
III non-manual	3.75	3.45	3.34	3.37	3.32	3.40	3.20
III manual	4.30	3.75	3.65	3.64	3.56	3.66	3.40
IV	4.92	4.07	3.98	3.92	3.83	3.93	3.62
V	5.63	4.43	4.34	4.22	4.12	4.22	3.85
% Attenuation in absolute difference between social class I and V after adjustment for risk factors*		45%	45%	53%	55%	55%	63%

[1]Behavioural factors=smoking, alcohol consumption, physical activity and BMI; [2]Biological risk factors=systolic blood pressure, triglycerides, LDL-cholesterol, HDL-cholesterol; *(AD0-AD1)/AD0*100; AD0 is age-adjusted absolute difference in event probability between social class I and V; AD1 is absolute difference in event probability adjusted for risk factors

Table 6.6 Population attributable risk fraction (PARF) from socioeconomic differences between manual and non-manual social class for CHD (incidence and mortality)

	Population attributable risk fraction						
	Age-adjusted	Age and behavioural factors[1]	Age, behavioural and biological risk factors[2]	Age, behavioural factors and CRP	Age, behavioural factors and IL-6	Age, behavioural factors and vWF	All risk factors
PARF (%) - CHD incidence	12	7	7	6	5	6	5
% PARF explained by risk factors*		41	41	52	56	52	56
PARF (%) - CHD mortality	15	10	10	9	7	9	7
% PARF explained by risk factors*		34	34	43	52	43	52

*(Unadjusted PARF-adjusted PARF)/unadjusted PARF*100

Chapter 7

Relationship of childhood socioeconomic position with coronary heart disease risk in later life

7.1 Summary

The independent influence of childhood socioeconomic position on health in later life remains uncertain. The extent to which childhood socioeconomic position is related to risk of coronary heart disease (CHD) in older British men was examined, taking account of adult social class and adult behavioural risk factors. Childhood socioeconomic position, based on father's occupation and childhood household amenities, was assessed retrospectively in the subjects of the British Regional Heart Study in 1992 when the men were aged 52-73 years. The men were followed-up for CHD incidence (fatal and non-fatal) and CHD mortality for 12 years till 2004. Men whose childhood social class was manual had an increased hazard ratio (HR) of 1.34 (95%CI 1.11, 1.63) for CHD incidence, though there was no consistent graded association between childhood social class and CHD incidence (age-adjusted p for trend 0.18). This association was attenuated when adjusted for adult social class and adult behavioural risk factors (cigarette smoking, alcohol, physical activity and body weight) (HR 1.21; 95%CI 0.99, 1.48). Men whose family did not own a car in their childhood were at increased CHD incidence risk even after adjustments for adult social class and behaviours (HR 1.34, 95%CI 1.04, 1.74 for CHD incidence). Men with combined exposure to manual social class in both childhood and adulthood had the highest risk of CHD incidence (HR 1.51; 95%CI 1.19, 1.91). However, this was substantially reduced after adjustment for adult

behavioural risk factors (HR 1.25; 95%CI 0.98, 1.61). Similar results were observed for CHD mortality. Lower socioeconomic position in childhood may have a modest persisting influence on risk of CHD in older age.

7.2 Introduction

One of the potential pathways to the development of coronary heart disease (CHD) in adult life is through the environment in early life.[52] There is evidence supporting the association of childhood socioeconomic position with CHD independent of adult socioeconomic position.[54;55;267] However, many of the studies, which examined this issue (seven of the ten studies in a systematic review),[54] did not take into account the role of adult behavioural risk factors. Lower socioeconomic position in childhood is associated with adverse behavioural factors in adult life including cigarette smoking, greater physical inactivity and obesity.[180;259;262] Adult behavioural factors could, therefore, be important influences on the relationship between childhood socioeconomic position and risk of CHD in later life. Some studies show that the association of childhood socioeconomic position with CHD is attenuated or reduced when adult behavioural risk factors are controlled for.[56;58;106;370] Early life socioeconomic position is related to adult socioeconomic position,[262] which is itself related to CHD risk, and therefore, assessment of the association of childhood socioeconomic position with CHD needs to take adult socioeconomic position into account. Adjusting for adult socioeconomic position and adult behavioural factors assumes their role as confounders of the childhood socioeconomic position-CHD relationship. However, it is plausible that these factors mediate the influence of early life socioeconomic position on CHD in later life. It has previously been shown in the British Regional Heart Study that in middle age (mean age of 50 years), childhood socioeconomic position was related to

CHD, independent of adult socioeconomic position and behavioural risk factors.[272] Few previous studies have reported the extended influence of childhood socioeconomic position in older age.

7.3 Objectives

The objectives of this Chapter are:

i) To examine the relation of childhood socioeconomic position to CHD risk (incidence and mortality) in older men aged 52-74 years (mean age 62 years) over a 12 year follow-up period, using father's occupation and childhood household amenities as markers of childhood socioeconomic position.

ii) To assess the contribution of adult social class and adult behavioural risk factors to the associations between childhood socioeconomic position and CHD.

iii) To examine the combined effect of childhood and adult social class on CHD risk.

7.4 Methods

In 1992, information on childhood socioeconomic position in the British Regional Heart Study was collected by postal questionnaires in addition to information on lifestyle factors (response rate of 91%). The questionnaires were also used to collect information on adult behavioural factors. Adult social class was based on the longest-held occupation recorded at study entry when the men were aged 40-59 years.

For this analysis, follow-up data on morbidity and mortality from 31st October 1992 until 1st June 2004 were used. The outcomes of interest were CHD incidence (fatal and non-fatal myocardial infarction) and CHD mortality. Information on morbidity and mortality has been routinely collected during the follow-up through general practice records and the National Health Service Central Register respectively (see sections 3.6.1 and 3.6.2). In accordance with the World Health Organisation criteria, non-fatal myocardial infarction was defined by the presence of at least two of – severe prolonged chest pain, ECG evidence of myocardial infarction, and cardiac-enzymes changes consistent with myocardial infarction.[283;284] Information from death certificates using the *International classification of diseases*, 9th revision (ICD-9) was used to identify fatal myocardial infarction cases as deaths with code 410-414 (equivalent to ICD 10th revision codes I20-I25).

7.4.1 Childhood socioeconomic position

As described in section 3.9.3 (page 90 of Chapter 3), the men were asked to report the longest-held occupation of their father in the 1992 questionnaire. Subjects were categorised into manual [3747 (72% of all subjects)] and non-manual [1434 (28%)] childhood social class groups. Based on the longest-held occupation of the father, the men were also classified into childhood social class groups using the Registrar General's Social Class Classification of 1931 (close to the mid-year of birth of study participants) – I (professional), II (intermediate), III (skilled), IV (partly skilled) and V (unskilled).[108;302] Social class III was further divided into III non-manual and III manual groups. Due lack of adequate information on father's occupation, a few subjects (n=120) were only classified into non-manual or manual groups. Information on childhood household amenities as a proxy for childhood socioeconomic position was also collected in 1992 to enable a better assessment of early life socioeconomic position. Subjects were asked if up to the age of 10 years their home had a bathroom, hot water supply and if their family owned a car.

7.4.2 Adult socioeconomic position

As described in section 3.9.1 (page 87) subjects' own adult social class was based on the longest-held occupation of each man recorded at the study entry when aged 40-59 years. Social classes I, II, III non-manual were categorised as 'non-manual social class' and social classes III manual, IV and V as 'manual social class'.

7.4.3 Adult behavioural risk factors

As described in sections 3.7.3 to 3.7.5 (page 82-83), detailed questions in 1992 were asked about cigarette smoking (number of cigarettes smoked and changes in smoking habits), alcohol consumption (frequency and number of alcoholic drinks), physical activity (frequency and type of activity), and body weight.[43;281;282] Body mass index (BMI) was calculated as body weight/(height)^2 using measures of body weight (in kilograms) and height (in meters, measured at the baseline examination). The men were classified into groups based on their alcohol intake – none, occasional (<1unit/week), light (1-15 units/week), moderate (16-42 units/week) and heavy (>42 units/week); 1 UK unit = 10g. In the questionnaire, subjects were also asked to report their pattern of physical activity including walking, cycling and other sporting activities. Physical activity scores were assigned on the basis of frequency and type of activity and were divided into 6 groups: none, occasional, light, moderate, moderately-vigorous and vigorous. Subjects in the categories of 'none' or 'occasional' activity were grouped together as 'inactive'.

7.4.4 Rationale for analyses

The relationship between childhood socioeconomic position and CHD (incidence and mortality) risk in older age was examined. Different measures of childhood socioeconomic position were used including childhood social class and childhood household amenities such as bathroom inside the house, hot water supply and family car ownership. Since childhood socioeconomic position is associated with adult socioeconomic position and adult behavioural factors which are also related to CHD,[19;180;259;262] the effect of the different measures of childhood socioeconomic position on CHD risk was adjusted for adult social class and for adult behavioural risk factors (cigarette smoking, physical activity, BMI and alcohol consumption). To assess the combined effect of childhood and adult social class on CHD risk, and to explore any interaction between childhood and adult social class, subjects were categorised into four groups according to both childhood and adult social class – both childhood and adult non-manual social class; childhood non-manual and adult manual social class; childhood manual and adult non-manual; and childhood and adult manual social class. The combined effect of childhood and adult social class on CHD risk was adjusted for adult behavioural factors. The relationship of adult behavioural risk factors with the combined childhood and adult social class groups was also explored. 268 (5%) men who did not report their father's occupation and 115 (2%) men whose fathers' longest-held occupation was the Armed Forces were excluded from the analyses.

7.4.5 Statistical methods

Cox proportional hazards model was used to calculate age-adjusted hazard ratios with 95% confidence intervals (CI) for CHD incidence and CHD mortality according to childhood social class across six social classes with social class I as the reference group, and separately also for manual (social classes IIImanual, IV, V) compared with non-manual (social classes I, II, IIInon-manual) childhood social class. The proportionality assumption for the Cox models was assessed by carrying out a test on the Schoenfeld residuals.[363] There was no strong evidence against the assumption. The model was then separately adjusted first for adult social class (all six social classes), second for adult behavioural risk factors, and finally both for adult social class and behavioural risk factors. Similar hazards ratios were calculated according to childhood household amenities. Hazard ratios for CHD risk were also calculated according to adult social class adjusted for childhood social class and then behavioural risk factors. Hazard ratios for CHD incidence and mortality were calculated for the combined childhood and adult social class groups - both childhood and adult non-manual social class (reference category); childhood non-manual and adult manual social class; childhood manual and adult non-manual; and childhood and adult manual social class. Formal tests of interaction between childhood and adult social class were also carried out. To further explore the relationship of adult behavioural risk factors with childhood and adult social class, the percentage of men who were current smokers, heavy drinkers, physically inactive and obese (BMI $\geq$30 kg/m^2) according to the groups of combined childhood and adult social class was calculated. For the adjustments, age and BMI were fitted as continuous variables. Childhood social class (six levels), adult social class (six levels), smoking (six levels), physical activity (five levels) and alcohol intake (five levels) were fitted as ordinal variables. Analyses were carried out using SAS version 8 and STATA version 9.

7.5 Results

7.5.1 Childhood social class, childhood amenities, and adult behavioural factors

Among 5181 men aged 52-74 years at 1992 who were followed over a 12 year period, 595 (1.12% per annum) CHD incident cases and 407 (0.76% per annum) CHD mortality had occurred. Table 7.1 describes the demographic characteristics of the subjects according to childhood social class. Table 7.1 shows that lower childhood social classes had a greater proportion of men who were in adult manual social class (social class IIImanual, IV, V). 85% of men of adult manual social class had a manual childhood social class. The proportions of men lacking childhood amenities (bathroom inside the house, hot water and family car) were higher in men of manual childhood social class (Table 7.1). Table 7.2 shows the differences in adult behavioural factors according to childhood social class. A greater proportion of men of manual childhood social class were current smokers, overweight, obese and physically inactive compared with men of non-manual childhood social class groups (I, II and III non-manual).

7.5.2 Childhood social class, childhood amenities and CHD incidence

Table 7.3 shows the number of CHD incident cases and hazard ratios for CHD according to childhood and adult social class. Across the 6 childhood social class groups, there was no strong evidence of a consistent association with CHD incidence (age-adjusted p for trend 0.18). However, men of manual childhood social class had a greater risk of CHD incidence compared with men of non-manual childhood social class (age-adjusted hazard ratio 1.34; 95%CI 1.11, 1.63). When adjusted for adult social class this effect, was weakened (hazard ratio 1.26; 95%CI 1.03, 1.55), though it remained statistically significant. In an age-stratified analysis, there was no evidence that this association differed between older (>63 years) and younger (≤63 years) men (p for interaction 0.82). The effect of childhood social class (non-manual vs. manual) was diminished when adjusted for adult behavioural risk factors (hazard ratio 1.21; 95%CI 0.99, 1.47. See Table 7.3). After adjustment for behavioural factors, additionally controlling for adult social class did not substantially alter these effect estimates (Table 7.3).

Adjustment for town of birth or region of residence in adult life (at the time of recruitment in the study) in addition to adult behavioural risk factors and adult social class made very little difference to the reported effect of childhood social class on CHD risk. In addition to adjustment for adult social class and behavioural factors, the hazard ratio for manual compared to non-manual childhood social classes was 1.17 (95%CI 0.96, 1.44) when adjusted for region of birth, and 1.19 (95%CI 0.97, 1.46) when adjusted for region of residence. In addition to adult behavioural risk factors and adult social class, adjustment for other biological risk factors measured at baseline screening including blood cholesterol, blood pressure and blood glucose also did not materially affect these results; the adjusted hazard ratio for manual childhood social classes was 1.21 (95%CI 0.98, 1.49).

Men of adult manual social class had a greater risk of CHD compared to non-manual groups, but the strength of this association was weaker than that seen for childhood social class (age-adjusted hazard ratio 1.24; 95%CI 1.05, 1.46. See Table 7.3). The effect of adult social class was slightly diminished when adjusted for childhood social class, and was attenuated when further adjusted for behavioural factors. Table 7.3 also shows hazard ratios for CHD incidence in relation to three different childhood household amenities. Those men whose family did not own a car in childhood had a higher CHD risk compared to those who did (age-adjusted hazard ratio 1.44; 95%CI 1.12, 1.85). This association remained statistically significant even after adjusting for adult social class and behavioural risk factors. Other childhood household amenities (bathroom inside the house and hot water supply) demonstrated weak non-significant associations with CHD incidence. Combining these childhood household amenities and childhood social class in a score (from 0 to 5), showed weak evidence of a higher CHD risk with increasing number of adverse childhood socioeconomic circumstances [hazard ratio for CHD incidence per unit increase in score was 1.07 (95%CI 1.01, 1.14); p for trend 0.02]. This association was, however, attenuated when adjusted for adult behavioural factors [hazard ratio for CHD incidence per score was 1.04 (95%CI 0.97, 1.10); p for trend 0.27].

7.5.3 Childhood social class, childhood amenities and CHD mortality

Table 7.4 shows the corresponding relationships of childhood social class, childhood household amenities and adult social class with CHD mortality. The associations of childhood social class and childhood amenities with CHD mortality were similar to those with CHD incidence described in the previous section. There was no gradient observed in the association with CHD mortality across the six childhood social classes (p for trend=0.24), although men of childhood manual social classes together had an increased risk of CHD mortality compared with non-manual groups (age-adjusted hazard ratio was 1.29; 95%CI 1.03, 1.63). The effect of manual childhood social class on CHD mortality was weakened on adjustment for adult social class and was attenuated when further adjusted for adult behavioural risk factors (hazard ratio 1.09; 95%CI 0.86, 1.40). Lack of family car ownership was associated with a greater risk of CHD mortality (age-adjusted hazard ratio 1.68; 95%CI 1.22, 2.33), which was independent of adult social class and behavioural risk factors. The effect of adult manual social class on CHD mortality was slightly weakened by adjustment for childhood social class, but was diminished when adult behavioural factors were taken into account.

7.5.4 Association of combined childhood and adult social class with CHD risk and with adult behavioural factors

The combined effects of social class in childhood and adulthood on risk of CHD were examined in Table 7.5 and Table 7.6, which show CHD incidence and CHD mortality rates per 1000 person years and age-adjusted relative risks according to childhood and adult social class with non-manual childhood and adult social class group as the reference group (group 1). CHD risk was lowest in this reference group and highest in those with both childhood and adult manual social class (group 4). Exposure to manual social class either in childhood or in adulthood (groups 2 and 3) was also associated with increased risk of CHD incidence and mortality.

Table 7.7 presents the relationship between adult behavioural factors and the combined childhood and adult social class marker. Higher levels of current smoking, physical inactivity and obesity were found in men who were of manual social class in either childhood or adulthood, with the highest levels in those exposed to both childhood and adult manual social class (group 4). Adult manual groups had higher levels of cigarette smoking and physical activity than all non-manual groups irrespective of childhood social class (groups 2 and 4 vs groups 1 and 3); childhood social class and adult social class had an influence on obesity. There was no evidence that the relation between childhood social class and these behavioural factors was different in adult non-manual and manual groups (p for tests for interaction for all behavioural factors >0.05).

Table 7.5 and Table 7.6 also show the relative risk for CHD incidence and mortality adjusted for these behavioural risk factors according to combined childhood and adult social class. Adjustment for adult behavioural risk factors substantially reduced the increased relative risks seen in those of both childhood and adult manual social class (group 4); the increased risk of CHD incidence was reduced from 1.51 (95%CI 1.19, 1.91) to 1.25 (95%CI 0.98, 1.61). The greater relative risks of CHD incidence and mortality in men of manual social class either in childhood or in adulthood (groups 2 and 3) were also attenuated when adjusted for adult behavioural risk factors. A test for interaction between childhood and adult social class showed no evidence that the effect of childhood social class (non-manual/manual) differed between those of adult non-manual and manual groups (p value 0.47 for CHD incidence and 0.41 for CHD mortality). The test for interaction using the extended childhood social class classification also showed no strong evidence of it being different across the six groups of adult social class (p values for tests for interaction were 0.71 for CHD incidence and 0.43 for CHD mortality).

7.6 Discussion

Manual childhood social class was associated with subsequent CHD risk in older age (52-74 years). The association diminished when adult behavioural risk factors were taken into account. The combination of manual childhood and adult social class increased the risk of CHD further but this was attenuated after adjustment for adult behavioural risk factors. Lack of family car ownership in childhood was associated with increased CHD risk, which was independent of adult social class and adult behavioural factors. A modest relationship of childhood social class with non-fatal CHD independent of adult social class and behavioural risk factors in middle-age (40-59 years) has been previously reported in the present cohort.[272] This Chapter extends the observations to CHD incidence and mortality in this population of older men, examines other measures of socioeconomic position in childhood as well as investigates the combined effect of social class in early life and adulthood with incident CHD.

7.6.1 Strengths and limitations of findings

The results of this Chapter are based on a population-based, socioeconomically representative sample of older men from across Britain, with high rates of follow-up for morbidity and mortality. The findings, however, are not necessarily generalisable to women and younger men. Since the British Regional Heart Study comprised largely white European men, the generalisability of the findings may be limited to other ethnic groups. Nevertheless, given the generic nature of childhood socioeconomic circumstances, their association with CHD risk may still be applicable to most population groups. This would be supported by a study in South Asian men which showed a greater risk of death from cardiovascular disease (CHD and stroke) in those from lower socioeconomic positions in childhood and adulthood.[371] In the present study, the childhood social class measure was based on the longest-held occupation of the father, which is likely to be a stable measure of childhood socioeconomic position, with social mobility in the father's generation probably less marked than among men in the generation of this study, who would have been more influenced by widened educational opportunities. A limitation of the results of this Chapter is that the measure of childhood social class is based on retrospective collection of information, raising the possibility both of random error and reporting bias. A particular tendency to overestimate childhood socioeconomic position by reporting a higher or more favourable father's social class than that recorded in early life has previously been demonstrated.[271] This would tend to result in underestimation of the effect of childhood social class on CHD risk. However, the validity of the father's social class measurement in the British Regional Heart Study is suggested by its agreement with the childhood household amenities data (Table 7.1) and its strong relationship with educational attainment of the subjects; a markedly lower proportion of subjects with fathers in manual occupations were educated after 18 years of age and a higher proportion left education at 14 years. Moreover, reporting bias is less likely for family household amenities in childhood, particularly car ownership, as participants are likely to recall such questions accurately. The accuracy of adult social class (based on occupation and used in adjusted analyses) is also important. This measure was based on longest-held occupation recorded at study entry in 1978-80 when the subjects were aged 40-59 years. It has already been established that this measure was stable over a 20-year period; as presented in Chapter 3 (section 3.9.1 page 87) only a small proportion (9%) of subjects changed from non-manual to manual social class. Moreover, the addition of other measures of adult social status (including car ownership and housing tenure) had little effect on the results observed in this Chapter (results not shown). The measures of socioeconomic position after retirement are however limited, allowing the possibility of some residual socioeconomic confounding.

7.6.2 Comparison with previous studies

The findings of this Chapter are consistent with previous studies on the relationship between childhood socioeconomic position and CHD risk.[54;55] These studies have indicated that lower childhood socioeconomic position is associated with increased CHD risk. The size of the associations were also mostly weak – increased CHD risk in lower childhood socioeconomic groups ranged from 8% to about 50%.[57;58;105;258;266;268;370;372;373] Some studies reported about a two-fold increased risk,[267;269;270] although the estimates for one study had wide confidence intervals.[267] Two studies, on the other hand, found no significant relationship between early life socioeconomic position and CHD risk in adulthood.[374;375] Only some of the earlier studies have taken into account the additional role of adult behavioural risk factors (which can be important influences on the relation between childhood socioeconomic position and CHD) and showed that adjustment reduces the influence of childhood socioeconomic position.[56-58;105;267;370] Socioeconomic position in childhood has also been found to be associated with increased accumulation of adult risk factors,[262;264;376] although some studies have reflected a difference in the influence of childhood and adult socioeconomic position on adult risk factors, with a stronger association of adult compared to childhood socioeconomic position with smoking and physical activity and a strong association between childhood socioeconomic position and BMI/obesity.[180;259;377] Previous studies also show a cumulative effect of childhood and adult socioeconomic position on CHD risk.[58;105;263] This is consistent with the findings of the present study in which manual social class in both childhood and adulthood was associated with a greater CHD risk, although the relationship was weakened when behavioural risk factors were taken into account. Most of the previous studies have, however, been carried out in middle-aged populations. A previous report from the British Regional Heart Study when the men were middle-aged (40-59 years), also observed a modest independent risk of non-fatal CHD in manual childhood social classes.[272] The absence of any relation between most childhood household amenities (access to bathroom and hot water) with CHD risk in the present study, which is consistent with findings from the British Women's Heart and Health Study, suggests that childhood infections as a result of poor household conditions are unlikely to be an important pathway to increased CHD risk in adulthood.[106] This would also be consistent with a previous finding in the British Regional Heart Study of the lack of an association between previous *Helicobacter Pylori* infection (acquired in childhood) and CHD risk in adult life.[378]

7.6.3 Interpretation of findings

The strength and statistical significance of the association of childhood socioeconomic position and CHD risk in this Chapter was strongly dependent on whether adjustment was made for adult social class and, particularly adult behavioural risk factors. The interpretation of these adjusted analyses depends on whether adult social class and adult behavioural risk factors are regarded as confounders of the childhood socioeconomic position–CHD association or mediators of it; if regarded as confounders then adjusted estimates are appropriate, and if regarded as mediators then unadjusted results would be a better guide. Childhood socioeconomic position is strongly related to adult socioeconomic position,[262] and the adult behavioural risk factors are themselves affected both by early life and adult socioeconomic conditions.[259;262] In the results presented in this Chapter, obesity in later life was related to childhood social class, while the other behavioural risk factors (smoking, physical activity) were more strongly influenced by adult social class. While the different effects of childhood and adult social class on behavioural risk factors have been explored to some extent, it was not possible in this Chapter to separate fully the issue of whether these risk factors are mediators or confounders, though the former remains a strong possibility, suggesting that unadjusted analyses may provide a truer indicator of the association. In the case of family car ownership in childhood, the association with CHD risk was however substantially independent of adjustment, though (like that for childhood social class) limited in strength. The persistence of this effect of childhood socioeconomic position in this older population is noteworthy. There are possible reasons why lack of family car ownership retained an independent relationship with CHD. First, the question on family car ownership may have been less prone to recall bias or misclassification compared with other questions on father's occupation or other childhood amenities. Second, family car ownership during the childhood of the subjects (approximately in the 1930s and 1940s) may be a better or stronger marker of material wealth or socioeconomic affluence. Thus, owning a car probably discriminated the very affluent from the rest, something that was not fully captured using the father's occupation-based social class distinction. It has been previously shown in this study population that material wealth such as car ownership discriminates mortality even within occupational social class groups.[362] A higher CHD risk in those lacking family car access, not necessarily indicating poverty as such, probably reflects the relative difference in wealth when compared to those who had a family car. It is possible that the effect of childhood socioeconomic position would be more apparent using more precise markers of resource income. This also highlights that the strength of the association of childhood socioeconomic position with CHD in later life in observational studies can differ according to the measures used to assess socioeconomic position in early life.[260]

Different mechanisms have been postulated to explain how childhood or early life factors affect health in later life.[46;254] Exposures acting during a specific period which influence the development of chronic diseases forms the basis of the 'critical period model' or 'critical period with later effect modifiers' if modified by exposures in later life. An example of this is the 'fetal origin of adult disease hypothesis' which proposes that low birth weight is associated with an increased coronary risk in adult life.[50] It is possible that in the present study, lower childhood socioeconomic position was associated with other early life exposures including low birth weight and fetal undernutrition, which are implicated in CHD risk.[52] The role of these early life exposures on CHD risk was not taken into account in the present study. The other potential pathway to chronic diseases is the 'accumulation of risk model'. According to this model adverse exposures accumulate over the life course gradually increasing the risk to worse adult health outcomes.[46;55] This accumulation of risk can either occur in a dose-response fashion or through clustering of exposures such as low birth weight, poor diet, lower educational attainment which are all associated with lower childhood socioeconomic position.[46] Adverse exposures can also accumulate by forming chains of risk where one exposure increases the risk of another. Although the data or results in this Chapter do not allow discrimination between these models, the results (particularly the combined influence of childhood and adult social class) would be consistent with a cumulative model of risk, with socioeconomic position at different stages of the life course contributing to overall CHD risk. However, it remains possible that socioeconomic exposures, particularly early in life, are critical in their timing. Large-scale cohorts with measures at a larger number of different stages of the life course are needed to fully substantiate the 'accumulation of risk' model and to distinguish it from the 'critical period' model.

7.6.4 Conclusions

The results of this Chapter show that the association of lower childhood socioeconomic position and CHD risk persists in a population of older men. Combined exposure to lower (manual) socioeconomic position in both childhood and adulthood is associated with the most unfavourable lifestyle behaviour. These findings add to the current literature in showing that the influence of socioeconomic position in childhood, though modest, persists in older age. Moreover, by this age, behavioural risk factors, which can have their origins in childhood and adulthood, play an important role in developing risk of CHD incidence and CHD mortality.

Table 7.1 Demographic characteristics of men aged 52-73 years in 1992 according to childhood social class

Childhood social class	n	**Age** (years) Mean (SD)	**Adult manual social class** n (%)	**No bathroom in childhood home** n (%)	**No hot water supply in childhood home** n (%)	**No family access to car in childhood** n (%)
I	208	61 (6)	29 (14)	30 (14)	24 (12)	99 (48)
II	767	62 (6)	259 (35)	268 (35)	253 (33)	471 (62)
III non-manual	555	61 (6)	180 (33)	155 (28)	160 (29)	410 (74)
III manual	2033	62 (6)	1133 (57)	1159 (57)	1113 (55)	1819 (90)
IV	833	62 (6)	592 (73)	539 (65)	523 (63)	785 (95)
V	665	61 (6)	477 (75)	444 (67)	436 (66)	635 (96)

n (%) = number of subjects (% of all those in that childhood social class)
SD=standard deviation

Table 7.2 Adult behavioural risk factors in men aged 52-73 years in 1992 according to childhood social class

Childhood social class	Current smokers n (%)	Heavy drinkers n (%)	Physically inactive n (%)	Overweight* n (%)	Obese† n (%)	**BMI** in kg/m^2 Mean (SE)
I	24 (12)	14 (7)	60 (29)	107 (51)	12 (6)	25.3 (0.23)
II	116 (15)	31 (4)	211 (28)	441 (58)	62 (8)	25.8 (0.12)
III non-manual	71 (13)	16 (3)	176 (32)	309 (56)	45 (8)	25.8 (0.14)
III manual	380 (19)	73 (4)	651 (32)	1216 (60)	215 (11)	26.1 (0.07)
IV	203 (24)	34 (4)	291 (35)	531 (64)	107 (13)	26.3 (0.12)
V	158 (24)	27 (4)	210 (32)	416 (63)	86 (13)	26.3 (0.13)
p for trend	<0.0001	0.55	0.02	0.0001	<0.0001	<0.0001

n (%) = number of subjects reporting health behaviour (% of all those in that childhood social class)
*BMI $\geq$25 kg/m^2
†BMI $\geq$30 kg/m^2
SE=-standard error

Table 7.3 CHD incidence according to childhood social class, adult social class and childhood household amenities in men aged 52-73 years followed-up from 1992 till 2004

		CHD incidence	Age-adjusted	Adjusted for age and adult social class	Adjusted for age and adult behavioural risk factors*	Adjusted for age, adult behavioural risk factors* and adult social class
Childhood social class	**n**	**n (%)**	**HR (95%CI)**	**HR (95%CI)**	**HR (95%CI)**	**HR (95%CI)**
I	208	18 (9)	1.00	1.00	1.00	1.00
II	767	86 (11)	1.26 (0.76, 2.10)	1.20 (0.72, 1.99)	1.14 (0.69, 1.90)	1.14 (0.68, 1.90)
III non-manual	555	51 (9)	1.03 (0.60, 1.76)	0.98 (0.57, 1.68)	0.91 (0.53, 1.57)	0.91 (0.53, 1.56)
III manual	2033	251 (12)	1.44 (0.89, 2.32)	1.31 (0.80, 2.13)	1.21 (0.75, 1.96)	1.19 (0.73, 1.94)
IV	833	94 (11)	1.34 (0.81, 2.21)	1.18 (0.70, 1.98)	1.06 (0.64, 1.77)	1.04 (0.62, 1.76)
V	665	77 (12)	1.34 (0.80, 2.34)	1.17 (0.69, 1.98)	1.07 (0.64, 1.80)	1.05 (0.62, 1.79)
p for trend			0.18	0.64	0.95	0.91
Non-manual	1434	136 (9)	1.00	1.00	1.00	1.00
Manual	3747	459 (12)	1.34 (1.11, 1.63)	1.26 (1.03, 1.55)	1.21 (0.99, 1.47)	1.21 (0.99, 1.48)
Adult social class						
Non-manual	2288	238 (10)	1.00	1.00	1.00	1.00
Manual	2757	338 (12)	1.24 (1.05, 1.46)	1.19 (1.00, 1.42)†	1.06 (0.89, 1.26)	1.04 (0.86, 1.25)†
Bathroom in childhood home						
Yes	2504	268 (11)	1.00	1.00	1.00	1.00
No	2660	327 (12)	1.07 (0.91, 1.26)	1.02 (0.86, 1.20)	0.99 (0.84, 1.16)	0.97 (0.82, 1.15)
Hot water supply in childhood home						
Yes	2587	279 (11)	1.00	1.00	1.00	1.00
No	2571	316 (12)	1.05 (0.90, 1.24)	1.00 (0.85, 1.18)	0.98 (0.83, 1.16)	0.97 (0.82, 1.15)
Family access to car in childhood						
Yes	839	70 (8)	1.00	1.00	1.00	1.00
No	4326	525 (12)	1.44 (1.12, 1.85)	1.37 (1.06, 1.77)	1.34 (1.04, 1.72)	1.34 (1.04, 1.72)

*Adult behavioural risk factors included smoking, alcohol consumption, physical activity and body mass index; HR=hazard ratio; CI=confidence intervals
† Adjusted for childhood social class

Table 7.4 CHD mortality according to childhood social class, adult social class and childhood household amenities in men aged 52-73 years followed-up from 1992 till 2004

	CHD deaths	Adjusted for age	Adjusted for age and adult social class	Adjusted for age and adult behavioural risk factors*	Adjusted for age, adult behavioural risk factors* and adult social class
Childhood social class	**n (%)**	**HR (95%CI)**	**HR (95%CI)**	**HR (95%CI)**	**HR (95%CI)**
I	16 (8)	1.00	1.00	1.00	1.00
II	53 (7)	0.79 (0.45, 1.38)	0.72 (0.41, 1.26)	0.70 (0.40, 1.24)	0.68 (0.38, 1.20)
III non-manual	34 (6)	0.74 (0.41, 1.35)	0.67 (0.37, 1.23)	0.64 (0.35, 1.17)	0.62 (0.34, 1.32)
III manual	172 (9)	1.04 (0.62, 1.73)	0.86 (0.51, 1.46)	0.84 (0.50, 1.41)	0.78 (0.47, 1.32)
IV	64 (8)	0.95 (0.55, 1.65)	0.75 (0.43, 1.32)	0.72 (0.41, 1.25)	0.66 (0.37, 1.17)
V	53 (8)	0.98 (0.56, 1.71)	0.77 (0.43, 1.37)	0.75 (0.42, 1.32)	0.68 (0.38, 1.23)
p for trend		0.24	0.99	0.94	0.63
Non-manual	95 (7)	1.00	1.00	1.00	1.00
Manual	312 (8)	1.29 (1.03, 1.63)	1.15 (0.90, 1.47)	1.14 (0.90, 1.44)	1.09 (0.86, 1.40)
Adult social class					
Non-manual	152 (7)	1.00	1.00	1.00	1.00
Manual	245 (9)	1.46 (1.19, 1.79)	1.43 (1.15, 1.78) †	1.23 (0.99, 1.52)	1.22 (0.97, 1.53) †
Bathroom in childhood home					
Yes	170 (7)	1.00	1.00	1.00	1.00
No	236 (9)	1.15 (0.94, 1.40)	1.06 (0.86, 1.30)	1.05 (0.86, 1.29)	1.02 (0.83, 1.25)
Hot water supply in childhood home					
Yes	179 (7)	1.00	1.00	1.00	1.00
No	227 (9)	1.10 (0.91, 1.35)	1.02 (0.84, 1.25)	1.03 (0.85, 1.26)	1.00 (0.82, 1.23)
Family access to car in childhood					
Yes	41 (5)	1.00	1.00	1.00	1.00
No	336 (8)	1.68 (1.22, 2.33)	1.54 (1.11, 2.14)	1.55 (1.12, 2.15)	1.51 (1.09, 2.11)

*Adult behavioural risk factors included smoking, alcohol consumption, physical activity and body mass index; HR=hazard ratio; CI=confidence intervals
† Adjusted for childhood social class

Table 7.5 CHD incidence according to childhood and adult social classes in men aged 52-73 years followed-up from 1992 till 2004

Group	Groups according to childhood and adult social class: Childhood	Adult	n	CHD incidence Rate per 1000 person years	Age-adjusted HR (95%CI)	Adjusted for age and adult behavioural risk factors* HR (95%CI)
1	Non-manual	Non-manual	989	8.5	1.00	1.00
2	Non-manual	Manual	406	10.9	1.25 (0.87, 1.79)	1.11 (0.77, 1.59)
3	Manual	Non-manual	1299	11.3	1.33 (1.02, 1.73)	1.27 (0.98, 1.66)
4	Manual	Manual	2351	12.8	1.51 (1.19, 1.91)	1.25 (0.98, 1.61)

*Adult behavioural risk factors included smoking, alcohol consumption, physical activity and body mass index
HR=hazard ratio; CI=confidence interval

[Permission to publish Table 7.5 has been obtained from the International Journal of Epidemiology]

Table 7.6 CHD mortality according to childhood and adult social classes in men aged 52-73 years followed-up from 1992 till 2004

Groups according to childhood and adult social class			CHD mortality Rate per 1000 person years	Age-adjusted	Adjusted for age and adult behavioural risk factors*
Group	Childhood	Adult			
				HR (95%CI)	HR (95%CI)
1	Non-manual	Non-manual	5.3	1.00	1.00
2	Non-manual	Manual	8.6	1.62 (1.07, 2.47)	1.48 (0.97, 2.27)
3	Manual	Non-manual	7.0	1.30 (0.93, 1.80)	1.27 (0.91, 1.77)
4	Manual	Manual	8.8	1.72 (1.28, 2.30)	1.41 (1.04, 1.93)

*Adult behavioural risk factors included smoking, alcohol consumption, physical activity and body mass index
HR=hazard ratio; CI=confidence interval

Table 7.7 Adult behavioural risk factors according to childhood and adult social classes in men aged 52-73 years followed-up from 1992 till 2004

Groups according to childhood and adult social class			Current smokers	Heavy drinking	Physically inactive	Obese (BMI≥30 kg/m^2)
Group	Childhood	Adult	n (%)	n (%)	n (%)	n (%)
1	Non-manual	Non-manual	122 (12)	46 (5)	281 (28)	72 (7)
2	Non-manual	Manual	65 (16)	13 (3)	131 (32)	38 (9)
3	Manual	Non-manual	172 (13)	44 (3)	380 (29)	112 (9)
4	Manual	Manual	577 (25)	98 (4)	801 (34)	311 (13)

n=number of subjects (% of all those in that group)

[Permission to publish Table 7.7 has been obtained from the International Journal of Epidemiology]

Chapter 8

Socioeconomic inequalities in disability among older men with coronary heart disease

8.1 Summary

Coronary heart disease (CHD) is an important contributor to disability, which has considerable public health implications in older people. However, the extent of socioeconomic inequalities in disability in people with CHD, particularly in older subjects, has not been well studied. This Chapter examines the extent of socioeconomic inequalities in disability and functional limitations in a socially representative sample of men aged 63-82 years in 2003 from 24 British towns. Disability, measured as problems with activities of daily living (ADLs) and instrumental activities of daily living (IADLs), and functional limitations were ascertained from questionnaires. Men with CHD had twice the prevalence of disability of those without CHD. Among men with CHD, lower social classes had higher risks of disability and functional limitation compared with higher social classes; odds ratios (95% CI) for social classes IV & V compared with I & II were 1.73 (1.00, 2.99) for ADL disability, 1.55 (0.88, 2.71) for IADL disability, and 2.33 (1.41, 3.84) for functional limitation. Behavioural risk factors (cigarette smoking, BMI, physical activity) attenuated these differences; the odds ratios (95%CI) in social classes IV & V reduced to 1.31 (0.67, 2.54) for ADL disability, 1.07 (0.54, 2.10) for IADL disability and 2.00 (1.08, 3.71) for functional limitations. Strong socioeconomic inequalities in disability were present amongst the elderly with CHD, which were considerably explained by behavioural factors.

8.2 Introduction

Disability is an important health outcome associated with coronary heart disease (CHD) in older populations.[379] Disability has been defined as limitation or loss of the ability to perform social roles and activities in relation to family, work or independent living.[380;381] Disability can originate from a pathology (disease) causing impairment (physiological or emotional), which in turn results in functional limitation, and finally in disability (limitation in carrying out social roles and activities).[382] The risk of disability increases with age and is a significant determinant of quality of life in the elderly.[60;383] Disability-free life expectancy is increasingly being used as a marker of population health in addition to life expectancy.[384;385] With increasing life expectancy, improving the quality of life is an important dimension of improving the health of the elderly population. The prevalence of disability has been observed to be 20-40% in people with CHD, which increases to 50-70% in older age (>75 years).[63;64;386] CHD, which is an important contributor to disability in older populations,[60;387;388] also shows strong socioeconomic gradients as presented in previous Chapters. Therefore, older people with CHD from lower socioeconomic positions might also have higher levels of disability than those in higher socioeconomic positions. Although previous studies have reported socioeconomic inequalities in disability and functional mobility or limitations,[273;276;277;389-392] the focus has largely been on functional mobility/limitations. While functional limitation and disability are related, they are not identical. Disability is an expression of functional limitation in a social context; functional limitations refer to problems in carrying out a task, whereas disability is difficulty in performing social roles.[381;393] Furthermore, little is known about the extent of socioeconomic differences in disability amongst older populations with CHD. The aim of this Chapter is to describe the extent of socioeconomic inequalities in disability in elderly men with CHD in Britain and their independence of health behaviours. Behavioural risk factors including smoking and physical activity, which vary by socioeconomic position, also have an important influence on disability.[180;394;395] Therefore, the extent to which socioeconomic inequalities in disability in those with CHD, were independent of behavioural factors was also investigated. Data from the British Regional Heart Study were used, specifically focusing on the questionnaire survey at which disability was assessed at age 63-82 years. Measures of disability in the form of problems in performing basic activities of daily living (ADLs) (such as eating and dressing) and problems in coping with instrumental activities of daily living (IADLs) (like shopping and managing money),[381;396;397] were used. These markers of disability not only form the core constructs of disability, but are also indicative of the quality of life in the elderly. In addition to disability, functional limitation, which is an important predictor of disability, was also measured.[398]

8.3 Objectives

The objectives of this Chapter are:

i) To examine the extent of socioeconomic inequalities in ADL disability, IADL disability and functional limitations in older British men aged 63-82 years with CHD.

ii) To investigate the impact of behavioural factors (cigarette smoking, physical activity, and body mass index) on the relationship between socioeconomic position and disability in older British men aged 63-82 years with CHD.

8.4 Methods

In 2003, when the men of the British Regional Heart Study were aged 63-82 years, information on disability and behavioural risk factors was sought through postal questionnaires, supplementing existing information on occupational social class (collected at baseline). Subjects reporting doctor-diagnosed heart attack or angina were classified as having CHD (the results were similar if the analyses were restricted to those only reporting heart attack). The longest-held occupation of each man, recorded at study entry when aged 40-59 years, was categorised using the Registrar Generals' Social Class Classification (I, II, III non-manual, III manual, IV and V). As in previous Chapters, occupational social class was used as the measure of adult socioeconomic position.

8.4.1 Assessment of disability

Disability was ascertained from responses to items in the 2003 postal questionnaire relating to problems in performing activities of daily living (ADLs) and instrumental activities of daily living (IADLs).[60;381;396;397] ADLs included performing the following activities unaided – walking across a room, getting in or out of bed, getting in and out of a chair, dressing and undressing, bathing or showering, feeding including cutting food, and getting to and using the toilet. IADLs included performing the following activities unaided – shopping for personal items such as toilet items or medicines, doing light housework such as washing up, preparing meals, using the telephone, taking medications, managing money (e.g. paying bills, etc), and using public transport. Reporting of some difficulty or inability/needing help to do one or more of the items was taken a having problem with ADLs or IADLs.

Functional limitation was ascertained through questions enquiring about the ability to walk without stopping or discomfort, to walk up and down stairs, and to bend and stand up. One or more of the following responses was taken as having a functional limitation – walking more than a few steps but less than 200 meters or only a few steps without stopping and without discomfort; unable to walk up and down a flight of 12 stairs without resting or only by holding and taking a rest; and unable to bend down when standing to pick up a shoe from the floor.

8.4.2 Behavioural factors

As described in sections 3.7.3 to 3.7.5 (page 82-83) detailed questions were asked in the 2003 questionnaire on smoking habits, physical activity and body weight. Subjects were asked to report their pattern of physical activity such as walking, cycling and other sporting activities. Physical activity scores were assigned on the basis of frequency and type of activity and the men were divided into six groups: none, occasional, light, moderate, moderately-vigorous and vigorous.[162] Scores of none and occasional were used to classify physically inactive subjects. Body mass index (BMI) was calculated as body weight/(height)^2 in kg/m^2. Obesity was defined as BMI of ≥30 kg/m^2.

8.4.3 Rationale for analyses

The relationship between social class and disability and functional limitations in those with CHD was examined. Adverse behavioural risk factors including cigarette smoking, greater physical inactivity and obesity can increase the risk of developing functional limitations and disability in older age.[399;400] These risk factors are also known to be associated with lower socioeconomic groups.[85;97;180] Therefore, the influence of behavioural risk factors on the relationship between social class and disability and functional limitations in those with CHD was also assessed. In this Chapter, which focuses on subjects with CHD, social classes I and II were combined, as were social classes IV and V, due to the small numbers of subjects in these groups.

8.4.4 Statistical methods

The prevalences of ADL disability, IADL disability and functional limitation were calculated in men with CHD and according to behavioural risk factors – current cigarette smoking, obesity and physical inactivity. Multiple logistic regression was used to assess the relation between social class and disability (problems with ADLs and IADLs) and functional limitation. Social classes I and II were treated as the reference category. Comparisons between non-manual and manual groups were also made. Non-manual groups included social classes I, II, III non-manual, and manual groups comprised social classes III manual, IV and V. After adjustment for age, behavioural risk factors (cigarette smoking, physical activity and BMI) were included in the models. For the adjustment, age and BMI were fitted as continuous variables; social class (three levels), cigarette smoking (six levels) and physical activity (five levels) were fitted as categorical variables.

8.5 Results

3981 men aged 63-82 years responded to the questionnaire in 2003 (80% response rate). 793 men (23%) reported having a doctor-diagnosis of CHD. Figure 8.1 shows the prevalences of ADL and IADL disability and functional limitations in men with and without CHD. Among men with CHD, the prevalence of ADL was 24%, of IADL 23%, and functional limitations 32%. These prevalences were nearly half in those without CHD (ADL 12%, IADL 11% and functional limitations 15%).

The presence of disability and functional limitations according to adverse behavioural factors among men with CHD is presented in Table 8.1. Current smokers had a greater prevalence of disability (ADL and IADL) and functional limitations compared to non-smokers. The prevalences of disability and functional limitations were greater in men who were obese compared with men who were not obese. The proportions of disability and functional limitations were also much greater in men who were physically inactive than in those who were physically active.

Table 8.2 shows the prevalences and odds ratios for ADL and IADL disability and functional limitations according to social class in men with CHD. There were approximately graded relations of social class with disability and functional limitations. Men in manual social class groups had a much greater risk of disability and functional limitations compared with non-manual groups. The age-adjusted odds ratio for ADL disability for social classes IV & V was 1.73 (95%CI 1.00, 2.99) compared with social classes I & II. This increased relative risk was weakened to 1.31 (95%CI 0.67, 2.54) after adjustment for behavioural risk factors (cigarette smoking, physical activity and BMI). Similarly, the association of social class with IADL disability was weakened on adjustment for behavioural factors; age-adjusted odds ratio for social class IV & V was 1.55 (95%CI 0.88, 2.71), which reduced to 1.07 (95%CI 0.54, 2.10) on further adjustment for behavioural factors. The association of social class with functional limitations was also reduced when behavioural factors were taken into account; odds ratio for social class IV & V reduced from 2.33 (95%CI 1.41, 3.84) to 2.00 (95%CI 1.08, 3.71).

8.6 Discussion

In the present study, a considerable proportion of older men with CHD had disability and functional imitations. Amongst older men with CHD, strong social class differences were apparent both in disability and functional limitations; men from lower social class groups had almost a two-fold greater risk of having disability and more than a two-fold greater risk of functional limitations compared with non-manual social classes. These socioeconomic disparities were considerably explained by behavioural factors (cigarette smoking, physical activity and BMI).

8.6.1 Strengths and limitations of findings

The results highlight strong socioeconomic inequalities in disability in a socially and geographically representative sample of older British men with CHD. The generalisability of the findings to women, younger men and other Western populations is, however, limited. Since the study comprised mostly white European men, the applicability of the findings to other ethnic groups is uncertain. While CHD is known to be a major contributor to disability in older age, it is also possible that disability through increasing physical inactivity and obesity can increase CHD risk. However, since the results are based on cross-sectional data it is not possible to establish the extent to which disability was a consequence of CHD. It is also possible that physical inactivity was a result of disability, and therefore, taking physical activity into account in the association between social class and disability could be viewed as an over-adjustment. However, excluding physical activity from the model and limiting the adjustment to cigarette smoking and BMI did not markedly change the overall results reported. Presence of CHD was based on self-report of doctor-diagnosis. Although there may be potential for reporting bias, patient-recall was found to be a valid measure of CHD in the study participants;[401] if anything the subjects were likely to over-report heart disease. It can be argued that objective measures of physical functioning are more accurate than self-reported disability. However, self-report of disability is an important evaluation tool for the health of older populations,[60] and ADL and IADL assessments are widely used measures of disability.[60;273;402;403] Self-reported disability, using ADL and IADL measures, has been found to be reliable and valid, although it may not be consistent over an extended time due to change in disease status or use of interventions.[404] Objective measures may be better at capturing functional impairments or limitations but may not reflect the extent of disability, which is a manifestation of functional limitations in a social context.[381] In this analysis, functional limitation was also examined, since it is a key precursor of disability.[405] The measure of socioeconomic position in this Chapter was social class based on the longest-held occupation of the subjects at entry to the study. Social class measures based on occupation can be problematic in the elderly in post-retirement age, as discussed in Chapter 6. However, social class (based on the longest-held occupation assessed in middle-age when the men were 40-59 years) can provide a stable marker of socioeconomic conditions over most of adult life.

8.6.2 Comparison with other studies

The results presented in this Chapter are consistent with those of previous studies which have shown that lower socioeconomic position is associated with greater levels of disability.[273;276-278;389;392;406;407] However, only two of these studies focused on socioeconomic differences in physical functioning in those with CHD.[278;392] In the Whitehall II Study of London-based civil servants, functional limitations among those with CHD were found to be greater in lower compared with higher employment grades and this difference persisted over a 12-year period.[278] A Dutch study also showed that among those with heart disease, lower socioeconomic groups had about a two-fold greater risk of decline in mobility in comparison with those of higher socioeconomic groups.[392] While previous studies have mostly used functional limitations or mobility problems, in this Chapter the extent of inequalities in disability measured as problems with performing ADLs and IADLs was also explored. In the results of this Chapter, behavioural risk factors were observed to contribute to the socioeconomic inequalities in disability. Men with adverse behavioural risk factors, including cigarette smoking, physical inactivity and obesity had higher levels of functional limitation and disability, although given the cross-sectional nature of the data it is possible that physical inactivity and obesity are consequences of disability rather than causes of it. However, these behavioural risk factors have previously been shown to be strong predictors of developing mobility problems and disability in later life.[408;409]

8.6.3 Interpretation of findings

In the present study, older British men aged 63-82 years with CHD from lower socioeconomic positions had a greater risk of functional limitations and ADL and IADL disability compared to those in higher socioeconomic groups. Behavioural risk factors such as cigarette smoking, physical inactivity and obesity were observed to account for a large proportion, but not all of these socioeconomic differences. Apart from socioeconomic differences in behavioural factors, other pathways could be linking socioeconomic position and disability. People from lower socioeconomic groups have poorer access to services or resources, rehabilitation, and worse living conditions.[410-412] All of these can contribute to increased chances of developing disability, or can retard the process of recovering from or coping with functional decline or disability.[381;412] These resources are important in minimising the impact of disability, and in promoting recovery. In this analysis, however, due to limitations in the data, it was not possible to control for or take into account the availability of coping mechanisms (or the lack of them) on inequalities in disability.

8.6.4 Conclusions

Socioeconomic inequalities in disability exist in older men with CHD. The findings of this Chapter show that among older British men with CHD, lower compared to higher socioeconomic groups had about a two-fold greater risk of disability. These socioeconomic inequalities were to a large extent explained by behavioural risk factors. Just as disability reflects the overall impact of diseases such as CHD in older people,[60] these socioeconomic inequalities in disability in the elderly can be indicative of the overall extent of health inequalities in later life from CHD.

Figure 8.1 Prevalence (%) of disability and functional limitations in men with and without CHD aged 63-82 years in 2003

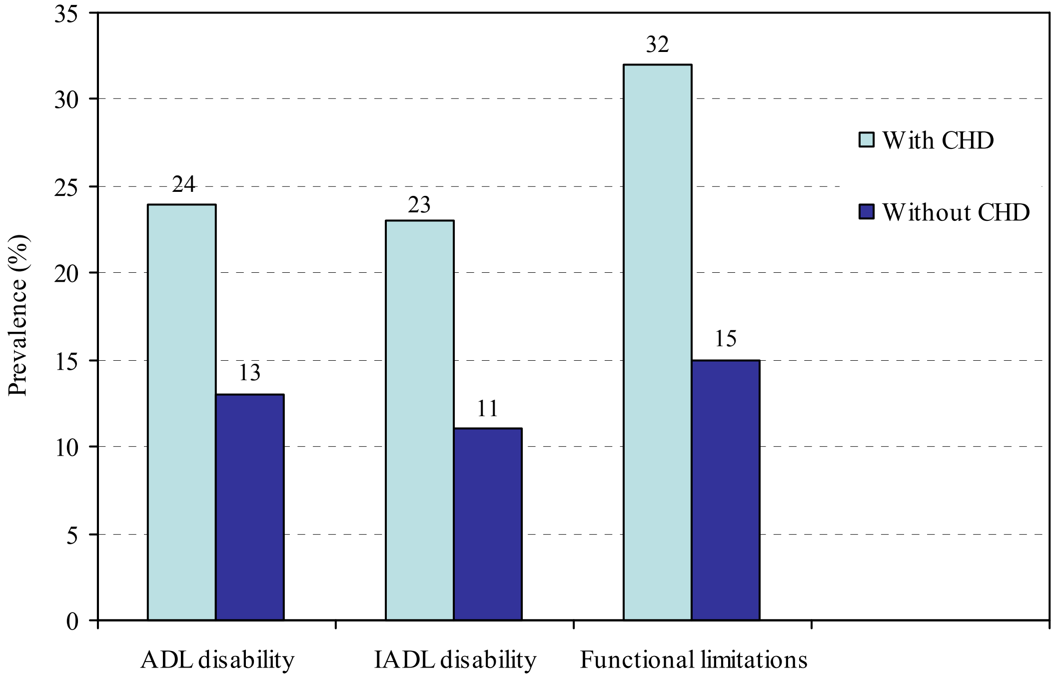


Table 8.1 Prevalence of disability and functional limitations according to behavioural risk factors in men with doctor-diagnosed CHD aged 63-82 years in 2003

Behavioural risk factors	n	**ADL disability** n (%)	**IADL disability** n (%)	**Functional limitations** n (%)
Cigarette smoking				
Current smokers	65	23 (36)	23 (36)	32 (49)
Non-smokers	711	165 (23)	158 (22)	217 (31)
Obesity				
Obese	203	72 (35)	66 (33)	86 (43)
Non-obese	573	116 (20)	115 (20)	163 (28)
Physically activity				
Inactive	382	151 (40)	154 (40)	193 (51)
Active	394	37 (9)	27 (7)	56 (14)

n (%)=number of subjects (% of all those in that group)

Table 8.2 Disability and functional limitations according to social class in men with CHD aged 63-82 years in 2003

		Social class					
		I & II	**III NM**	**III M**	**IV & V**	**Non-manual**	**Manual**
ADL disability	n (%)	47 (17)	24 (26)	91 (30)	26 (26)	71 (19)	117 (29)
	Odds ratio*	1.00	1.78 (1.01, 3.13)	2.15 (1.44, 3.21)	1.73 (1.00, 2.99)	1.00	1.74 (1.24, 2.43)
	Adjusted odds ratio†	1.00	2.20 (1.12, 4.32)	1.96 (1.21, 3.17)	1.31 (0.67, 2.54)	1.00	1.44 (0.95, 2.18)
IADL disability	n (%)	49 (18)	18 (20)	90 (30)	24 (24)	67 (18)	114 (28)
	Odds ratio*	1.00	1.23 (0.67, 2.26)	2.11 (1.41, 3.15)	1.55 (0.88, 2.71)	1.00	1.86 (1.31, 2.63)
	Adjusted odds ratio†	1.00	1.15 (0.56, 2.34)	1.92 (1.18, 3.10)	1.07 (0.54, 2.10)	1.00	1.61 (1.06, 2.45)
Functional limitations	n (%)	58 (21)	26 (28)	127 (42)	38 (37)	84 (23)	165 (41)
	Odds ratio*	1.00	1.56 (0.90, 2.68)	2.86 (1.97, 4.16)	2.33 (1.41, 3.84)	1.00	2.41 (1.76, 3.32)
	Adjusted odds ratio†	1.00	1.80 (0.92, 3.51)	2.86 (1.80, 4.54)	2.00 (1.08, 3.71)	1.00	2.25 (1.51, 3.34)

*Age-adjusted odds ratio
† Odds ratio adjusted for age and behavioural factors including cigarette smoking, physical activity and BMI

Chapter 9

Implications and conclusions

9.1 Summary

The implications of the findings of this thesis for public health and for epidemiological studies are reviewed in this Chapter. Findings from analyses conducted for this thesis demonstrate the extent of socioeconomic inequalities in coronary heart disease (CHD) in older age and the possible pathways to these inequalities. The particular findings of potential public health significance are: i) that further efforts are needed to reduce socioeconomic inequalities in CHD, which have not narrowed in Britain between the 1980s and the early part of the twenty-first century; ii) that socioeconomic inequalities in CHD, which persist in older age, need to be targeted; iii) that focusing on reducing socioeconomic differences in behavioural coronary risk factors (cigarette smoking, physical activity and obesity) will substantially reduce (probably by about a third) socioeconomic inequalities in CHD in older age; iv) that improving early life socioeconomic conditions is likely to have some modest effects on reducing CHD risk in older age; vi) that socioeconomic inequalities in disability in older populations with CHD needs to be reduced; and v) that reducing socioeconomic inequalities in behavioural risk factors can partially reduce theses inequalities in disability in the elderly with CHD. The results have implications for future epidemiological studies, which include (1) the need to include older populations when investigating socioeconomic inequalities in CHD, and (2) to explore other pathways to these inequalities including early life exposures, social integration and psychosocial conditions. A key general implication of this thesis is the need for understanding and reducing socioeconomic inequalities in CHD in older age.

9.2 Introduction

Previous Chapters have investigated the extent of socioeconomic inequalities in CHD in older men from 24 towns across Britain and possible pathways to these inequalities. Between the early 1980s and 2005, relative socioeconomic inequalities in CHD did not narrow. Relative inequalities narrowed as the men moved from middle to older age, although absolute inequalities increased with age. Nevertheless, a social class gradient in CHD persisted in older age and behavioural coronary risk factors contributed to at least a third of this gradient. Novel coronary risk factors (inflammatory/haemostatic markers) made some further contribution. Lower early life socioeconomic position was associated with increased CHD risk in older age. Marked socioeconomic inequalities in disability were also present in the elderly with CHD. In this Chapter the implications of these findings are discussed in more detail. Section 9.3 considers the potential public health implications of the results, and section 9.4 examines the implications for future epidemiological studies.

9.3 Public health implications of findings

9.3.1 Trends in socioeconomic inequalities in CHD

Monitoring trends in health inequalities makes it possible to detect whether disparities have reduced or increased over time. Reducing the gap in health inequalities has been a high priority of the Department of Health.[413] Documenting trends in socioeconomic differences in CHD has important implications for evaluating policies and informing decision-making for future policies to reduce health inequalities. The results of Chapter 4 showed that lower compared with higher social class groups in Britain continued to have a greater risk of CHD mortality throughout the period between the early 1980s to 2005, and these relative social class differences tended to have widened during this time. However, with the decline of overall CHD mortality rates during this period, the absolute difference in CHD mortality between social classes narrowed in Britain. The results suggest that policies in place during the 1980-2005 period have not led to a reduction in relative inequalities in CHD mortality. Although declining death rates have mitigated the consequences of this inequality, marked inequalities in absolute terms continued to persist. Relative socioeconomic inequalities express the strength of the association between social class and CHD mortality risk.[414;415] Absolute socioeconomic differences depict the magnitude of the inequality in CHD mortality, reflecting its public health importance.[414;415] Both these measures are important when monitoring inequalities over time as they indicate different, yet important, aspects of inequalities; absolute inequalities may change depending on the overall change in the mortality/disease patterns, while relative inequalities reflect the strength of the effect of socioeconomic position.[414] Chapter 4 also showed that with increasing age, relative social class inequalities in CHD had reduced from middle-age to older ages. The absolute social class difference in CHD, however, widened as the men became older, reflecting the higher disease mortality occurring at greater age. Socioeconomic inequalities in CHD are, therefore, likely to be a public health problem even in later life.

If reductions in relative inequalities in CHD are to be achieved, greater efforts are needed to devise and implement effective policies for inequality reduction. While the possible factors contributing to the observed trends in socioeconomic inequalities in CHD were not examined in the thesis, the actions required are likely to include both general measures to reduce income inequality and improve living standards of poorer households,[34] as well as more specific measures to reduce cigarette smoking prevalence, and improve physical activity and nutritional intakes among lower-income groups, by a combination of national and local action.[35] For example, policies and strategies at regional and local levels can plan the maintenance and development of towns and transport services to improve the physical environment,[416] while local services can encourage individually-focused interventions to promote physical activity such as exercise-referral schemes.[417] Ensuring equitable provision of health services, particularly preventive services, will also be important.[34] A recent National Institute of Health and Clinical Excellence (NICE) guidance focuses on reducing premature cardiovascular deaths in those who are disadvantaged including low-income families, those on benefits, people living in social housing or the homeless.[418] The guidance recommends identifying disadvantaged groups through primary care and community services to ensure provision of smoking cessation services and statin therapy, both of which are effective and cost effective interventions to reduce coronary risk. These actions need to be set in a broader policy framework ensuring that all government policies are monitored for their impact on inequality.[34;419] Monitoring the impact of policies on key risk factors as well as establishing targets for reducing inequalities in CHD will provide an important evidence base for the development and implementation of further policies and interventions. Several such issues have been highlighted in Department of Health publications such as Tackling Health Inequalities,[20] and more recently in Choosing Health.[420] The policy document on health inequalities published by the Department of Health in 2008 outlines measures to achieve the Public Service Agreement target of reducing inequalities in life expectancy by 10% by 2010, and to support improvements beyond 2010.[421] The report emphasises three domains where interventions will have the greatest impact on health inequalities: influences on health (the environment and wider social factors), the lives people lead (promoting healthy behaviours), and the services people use (ensuring equitable treatment and prevention). Suggestions for improvements in the wider influences on health included continued improvement in employment levels and education particularly for those in disadvantaged backgrounds, and encouragement of private sector employers to improve the health and well-being of their staff and their communities. Advice, support, preventive medication and appropriate treatment to reduce mortality and morbidity from CHD have also been outlined. However, the possibility of these initiatives increasing inequalities also needs to be addressed since those from higher socioeconomic groups or more motivated people may take more advantage from these initiatives than those at higher risk of disease. While these steps outlined in the Department of Health report[421] are based on evidence from local data of effectiveness and on evidence showing the relationship between socioeconomic conditions and health, more evidence is needed to support and evaluate effective policies. For example, the impact of policies such as the National Support Teams for Health Inequalities set up to support local areas to reduce health inequalities, need to be constantly evaluated to assess their effectiveness in reducing inequalities in disease such as CHD.[421] The use of health equity audits at local levels is an important step in monitoring patterns in provision of resources or services in relation to health needs of different groups.[113;422] More rigorous and determined steps need to be taken in implementing these reforms and policies if narrowing of socioeconomic inequalities is to be achieved.

9.3.2 Reducing socioeconomic inequalities in CHD in older age

Strong socioeconomic inequalities in CHD risk persisted in older age, as observed in Chapter 6. These inequalities were present in relation to occupational social class as well as other indicators of socioeconomic position including house and car ownership. Occupational social class differences appeared to have the strongest relationship with CHD; lower social classes had a greater CHD risk than higher social class groups. Given the increasing proportion of older people in the population and the greater CHD risk in the elderly, socioeconomic inequalities in CHD presents an important public health problem in the elderly. While the National Service Framework (NSF) for CHD highlighted reducing inequalities as a priority,[423] the focus was on individuals aged <70 years. The NSF for Older People concentrated largely on removal of age-related inequalities in all diseases, with little focus on socioeconomic disparities in heart disease in older age.[424]

9.3.2.1 Role of coronary risk factors

Associations between socioeconomic position and different coronary risk factors were examined in the older men (aged 60-79 years) of the British Regional Heart Study in Chapter 5. As with middle-aged populations, behavioural risk factors including cigarette smoking, physical inactivity and obesity were more common in lower social class groups in older age. Of the novel coronary risk factors, higher circulating levels of inflammatory/haemostatic markers were observed in lower social classes. However, the social class gradient in circulating inflammatory and haemostatic markers was to a large extent mediated by the higher levels of adverse behavioural risk factors (obesity and particularly cigarette smoking) in lower social class groups. Focusing efforts on understanding and reducing adverse behavioural factors and levels of obesity, which explained much of the social class variation in inflammatory/haemostatic markers, could therefore be particularly important in reducing socioeconomic inequalities in CHD.

The findings of Chapter 6 suggest that behavioural factors (particularly cigarette smoking) and to some extent novel coronary risk factors, are important determinants of socioeconomic inequalities in CHD in older age. The substantial contribution of these risk factors to the absolute risk difference between social classes indicates that their public health impact in reducing CHD inequalities in older people is potentially important. Novel coronary risk factors such as inflammatory and haemostatic markers made some contributions to socioeconomic inequalities, but these were modest in addition to that of behavioural risk factors. Socioeconomic inequalities in CHD in older age could potentially be narrowed by at least a third through reduction in levels of behavioural risk factors including cigarette smoking, BMI and physical inactivity. Of the behavioural risk factors, cigarette smoking made a particularly important contribution (about 20%) to these inequalities. Since there are socioeconomic differences in smoking initiation and cessation, both these aspects need to be targeted to reduce inequalities in smoking and in turn to reduce inequalities in CHD in later life. Policy efforts have included smoking bans in public places, higher prices and taxation policies, and restriction on advertisement and marketing.[425;426] Raising prices of cigarettes and tobacco products has been advocated as a useful way of reducing overall smoking levels as well as resulting in greater reductions of smoking in lower socioeconomic groups.[426-429] However, it has been argued that increased tobacco taxation is likely to result in imposing further economic hardship in lower socioeconomic groups who have greater levels of addiction, and could also result in increased illegal import of cigarettes.[430;431] Greater proportion of the tax revenue would be required for smoking cessation support especially for lower socioeconomic groups, alongside an increase in tobacco taxation.[431;432] Focusing on manual social class groups is one of the priorities outlined in the recent NHS Stop Smoking Services guidelines published by the Department of Health,[433] since lower quit rates in manual social classes compared with higher socioeconomic groups have been observed.[434;435] A narrowing of inequalities in smoking rates was reported from mid-1980s to the 1990s and continuing to 2000 in Britain.[436;437] These trends appear to be in the right direction to reduce inequalities in CHD.

In addition to smoking, improving levels of physical activity and reducing levels of obesity, particularly in lower socioeconomic groups, are also likely to reduce socioeconomic inequalities in CHD in later life. Food labelling to promote healthy eating, and environments (work and residential) conducive to encouraging physical activity, are some interventions aimed at reducing obesity.[421] Various interventions have been found to be effective in improving levels of physical activity and also in reducing body weight or adiposity.[438] These include: i) informational approaches aimed at increasing awareness about the benefits of physical activity and participation in community-based activities; ii) social approaches to improve physical activity such as school/college-based health education programmes, or community-based social support groups; and iii) policy interventions to improve environmental opportunities, resources and facilities to increase physical activity.[438] Improving physical activity even in older age has been found to be associated with a reduction in CHD risk.[166] Although the evidence associating reduction in body weight (intentional weight loss) with decreased CHD risk is limited, weight reduction is associated with an improvement in coronary risk factors such as decrease in blood pressure and LDL-cholesterol and increase in HDL-cholesterol levels.[439-443] Moreover, weight reduction has also been shown to reduce insulin resistance and clustering of metabolic risk factors,[444-447] which are associated with CHD risk. Individual-level interventions have shown modest sustained or long-term benefits for behavioural change, risk factor reduction, and reducing coronary risk.[448-453] Therefore, greater priority for fiscal and legislative changes is likely to result in further improvements in risk factors.

While targeting behavioural coronary risk factors is an important strategy to reduce socioeconomic inequalities in health outcomes such as CHD, ultimately reducing socioeconomic inequalities themselves must be addressed.[115] Although the proportion of pensioners living in low-income households has decreased over the last decade in the UK, income inequalities are present in older populations (19% of pensioners lived in low-income households in 2006/2007).[454;455] The wider social, cultural, political and material societal context along with disadvantaged socioeconomic conditions across the life course are known to be important in the origin of adverse health behaviours.[261;262;456] Higher socioeconomic groups have advantages in power, prestige and knowledge which enable them to avoid health hazards and adopt health-protective behaviours.[115] Therefore, these factors underlying the more proximal causes (coronary risk factors) of CHD also need to be tackled. Assessing the social context to identify and remove social, financial and environmental barriers to better health, including poverty, employment and education is one of the recommendations in the NICE guidance on behaviour change in disadvantaged groups.[457] Consequently, different sectors of public policy (including housing, transport and education) would play a crucial role in improving wider societal determinants to modify behavioural factors particularly in socioeconomically disadvantaged groups.

9.3.3 Impact of early life socioeconomic position on CHD risk in later life

The results from Chapter 7 show that the effect of lower childhood socioeconomic position on CHD risk persists even into older age. The influence of childhood social class on CHD, however, was not independent of behavioural risk factors (smoking, physical inactivity and BMI). Lack of family car ownership during subjects' childhood (a marker of lower socioeconomic position) was associated with a greater CHD risk even though the effect was diminished by adjustment for behavioural factors. Family car ownership was possibly a stronger discriminator of material wealth or affluence than childhood social class based on father's occupation. Men of manual childhood social class tended to have higher levels of cigarette smoking, physical inactivity and obesity in older age compared with those from non-manual childhood social classes. Combined exposure to manual socioeconomic position in both childhood and adulthood was associated with the most unfavourable adult lifestyle. Lower socioeconomic position (manual social class) in both childhood and adulthood was also associated with highest CHD risk in older age, although adult behavioural risk factors accounted for a lot of this increased CHD risk. Whether behavioural risk factors were mediators or confounders of the childhood socioeconomic position-CHD relation was not established in the results, although the former is a possibility. Nevertheless, behavioural risk factors, which can have their origins in childhood and adulthood, played an important role in developing risk of CHD in later life. As mentioned in the previous section (9.3.1), policies to reduce initiation of adverse health behaviours such as cigarette smoking and physical inactivity are important. However, these interventions need to be started early in life since health behaviours, such as smoking, adopted at an early age tend to extend into adulthood.[458;459] School-based interventions, mass media campaigns, and restrictions in advertising are known to reduce initiation of smoking in adolescents.[460-462] Increasing rates of obesity and lower levels of physical activity in children particularly in those from poorer socioeconomic backgrounds in Britain,[463;464] are likely to have an impact on the burden of CHD in later life. Recognition of the importance of early life socioeconomic conditions on health has led to the inclusion of a focus on early years and parenting in a recent Department of Health report on health inequalities.[421] Although childhood obesity was highlighted as one of the priority areas, there was little focus on improving physical activity in children and reducing the likelihood of taking up smoking in the report.[421] Specific interventions or policies are needed to improve early life socioeconomic conditions and health behaviours, which influence their association with CHD in later life. In addition to short-term gains from investing in socioeconomic conditions in early life such as better health of children, long-term gains can also be achieved in adult life with respect to improved adult health behaviours, and lower levels of CHD in older age, as observed in Chapter 7. Thus, as regards to public health policy, a dual approach aiming both to improve childhood socioeconomic conditions as well as to target socioeconomic disparities in behavioural risk factors in adult life will help reduce the burden of CHD in older people.

9.3.4 Socioeconomic inequalities in disability in the elderly with CHD

The findings of Chapter 8 show marked socioeconomic inequalities in disability in older men with CHD. CHD is an important contributor to disability in older age.[61;64] Therefore, among older populations with CHD, inequalities in disability could highlight socioeconomic inequalities in functional performance, fulfilling social roles, independent living in the elderly and ultimately in the quality of life, associated with CHD in the elderly.

In the British Regional Heart Study, older men with CHD had about twice the prevalence of disability compared with men without CHD. Furthermore, within men with CHD, lower compared with higher socioeconomic groups had a greater risk of disability. Disability was measured as difficulty in performing activities of daily living (ADL) and instrumental activities of daily living (IADL). Activities of daily living included tasks such as eating, bathing, dressing, and instrumental activities of daily living included shopping, preparing meals, taking medications. These measures, therefore, capture essential elements of disability, which has been defined as the limitation or loss of the ability to perform social roles and activities in relation to family, work or independent living.[465;466] A large proportion of the increased risk of ADL and IADL disability in manual social groups with CHD was accounted for by behavioural risk factors such as smoking, physical inactivity and BMI, which are known risk factors for disability.

An understanding of pathways underlying disability or the 'disablement process'[381] will inform health policy-makers in ways to reduce the burden of disability and inequalities in disability in those with CHD. First, continued efforts to reduce levels of behavioural risk factors such as smoking, physical inactivity and obesity in the elderly are needed. Although these may be regarded as 'individual' risk factors, they are influenced by the socioeconomic context,[261;467] and therefore policy plays a vital role in reducing these factors in the population. Improving these behavioural risk factors across all age groups would be likely to prevent disability as well as specific diseases and would reduce inequalities in disability in later life. Changes in lifestyle even later in life has been shown in the British Regional Heart Study to have the potential to not only reduce the onset of mobility limitations but also to improve recovery from disability in the elderly.[468] Smoking cessation and uptake of physical activity can reduce the onset of mobility limitations in older age, and improvements in physical activity in the form of walking or gardening can improve the likelihood of recovery from mobility problems.[469] Second, provision of adequate rehabilitation, interventions and care would be needed to cope with functional decline in old age. The ability to perform tasks for independent living and functioning in old age is not only dependent on the functional ability of older people but also on the facilities available in the physical or environmental context in which they live.[381;412;470] This implies that adequate provision for the needs of older people is important in housing and environmental policies. Trials have shown the effective prevention of disability from interventions including physical exercise, home visits, training in use of assistive devices and removal of environmental hazards.[471;472] However, more such evidence is needed to establish further means of reducing disability, particularly among the lower socioeconomic groups. Evaluation of the effectiveness and cost-effectiveness of interventions targeted at reducing inequalities in disability in the elderly are needed. Policy efforts are required to reduce the overall burden of disability in later life as well as to reduce the greater burden of disability experienced by those in lower socioeconomic groups with CHD.

9.4 Implications for future epidemiological studies

The implications of the findings of this thesis for future epidemiological studies will be discussed in the following sections. The particular areas that will be considered are the design and analysis of population-based studies to investigate 1) socioeconomic inequalities in CHD in older age; 2) pathways to these inequalities; and 3) the influence of early life socioeconomic position on CHD risk in later life. This will enable a better understanding of these important public health issues, and thus provide opportunities to reduce socioeconomic inequalities in CHD in later life.

9.4.1 Investigating socioeconomic inequalities in CHD in older age

In this thesis, *relative* socioeconomic inequalities in CHD did not appear to narrow over the 25 years between 1978-80 and 2005, although *absolute* socioeconomic inequalities in CHD had narrowed (Chapter 4). Previous work on the British Regional Heart Study showed that favourable changes (reductions) in smoking, blood pressure and cholesterol levels played an important role in the decline of overall CHD rates over the last 25 years.[473] Further research is needed to examine whether socioeconomic inequalities in these coronary risk factors changed during this period. In order to do this, changes in levels of coronary risk factors (smoking, physical activity, BMI, blood lipids and blood pressure) over this time according to socioeconomic groups would need to be studied and repeated information on these coronary risk factors would be required.

In order to investigate socioeconomic differences, it is important for epidemiological studies to be representative of the general population. An important strength of the data used in this thesis is that it comprises a population-based study of older men who were recruited in middle-age from socioeconomically representative general practices from towns representing all the major regions of Britain. In epidemiological studies in the elderly, there maybe a possibility of selection bias when recruiting older subjects. Non-response in epidemiological studies is often related to ill-health, and this factor can be more important in the elderly. Elderly non-responders tend to have more ill-health and are more likely to be in hospital or nursing homes,[474;475] thus resulting in an underestimation of disease prevalence and possibly even weakened associations between disease and exposures like socioeconomic position. To reduce this bias, subjects could be recruited in middle-age and followed-up thereafter, as was done in the present study. This would also provide opportunities to collect information on occupation, other socioeconomic indicators and coronary risk factors across adult life. Although attrition of the cohort with age remains an issue when studying older populations, follow-up can be maximised as much as is possible through careful tracking of subjects.

At the time of the inception of the British Regional Heart Study in the mid-1970s, it was realised that the lower risk of CHD in women in middle-age demanded a very large number of women subjects to ensure adequate number of endpoints, making for considerable logistic and financial problems. The study, therefore, comprised only men. Also, the towns selected at that time had relatively stable populations with a small proportion of ethnic minority groups. Therefore, the data presented in this thesis are based on men and do not include appreciable representation of British ethnic minority groups. It is now well-recognised that CHD is a major chronic disease in women,[6] and in ethnic minority groups such as south Asians, who have a much greater risk of CHD compared with white European population groups.[476;477] Therefore, it is important to extend investigations to include women and ethnic minority groups. While there was some advantage in the British Regional Heart Study in that it comprised a homogenous group of white European men, it was not possible to explore the extent of socioeconomic inequalities in other ethnic groups. Future studies including different ethnic minority groups would enable investigation of socioeconomic inequalities in CHD within and between ethnic groups in later life.

9.4.1.1 Ascertaining socioeconomic position in later life

A key challenge in future epidemiological studies investigating socioeconomic inequalities in older age is measuring socioeconomic position in old age, which is complex. Occupation-based measures are difficult to use in post-retirement age. In this thesis, the longest-held occupation of the men assessed in middle-age was used as the main marker of socioeconomic position. The advantage of using such as measure is that it is a relatively stable marker of socioeconomic position across adult life. The longest-held occupation is also likely to be related to socioeconomic conditions in later life, and can therefore, be an indicator of socioeconomic position in older age. Education did not appear to be related to CHD risk in later life in the results of this thesis (Chapter 6). Education, although commonly used as a marker of adult socioeconomic position, is to a large extent dependent on parental or early life socioeconomic position. This could mean that education is, conceptually, a weaker marker of socioeconomic position in old age. Other markers, such as house and car ownership, were associated with differences in CHD risk in this thesis, though they were not independent of occupational social class (Chapter 6). A limitation in using house and car ownership to measure socioeconomic position in old age is that these markers are likely to be influenced by ill-health and other processes of ageing, which weakens their validity as socioeconomic indicators in the elderly. People are likely to stop owning a car due to disability in old age, and housing status may also change with increasing age and ill-health (for example from owned home to nursing home/relatives' home). Combinations of different indicators, including social class, income, education, and house/car ownership, in the form of composite measures of socioeconomic position have also been used.[97;478] Combining such measures has been previously shown to indicate a greater magnitude of socioeconomic inequalities in all-cause and cardiovascular mortality in middle-age,[97] although combined measures (social class, car/house ownership, education and pension arrangements) in the present study of older men were not associated with a greater magnitude of CHD risk compared with that of occupational social class alone. However, caution should be exercised in choosing measures when combining indicators in older age; for example house/car ownership may not be particularly appropriate markers of socioeconomic position in old age. Alternatively, income, a powerful indicator of material wealth and socioeconomic conditions, can be used as an indicator of socioeconomic position. However, collecting information on income has been shown to reduce response rates.[479] Moreover, income has been shown to be more strongly related to health in younger rather than older ages, possibly due to lower income in old age.[479;480] Wealth in the form of financial assets, house ownership, and employment benefits instead of income has been proposed as a robust marker of socioeconomic position.[479] Wealth can be viewed as an indicator of income over the life course, which also reflects inherited assets and wealth. Retired and elderly individuals can have greater wealth (house value or measures of accumulated income or savings) even though they may have lower income due to reliance on pensions (a contemporaneous measure of income).[479] The English Longitudinal Study of Ageing in older populations has recently collected detailed information on wealth including financial, pension, housing and physical (assets, land, jewellery) wealth.[481] Although wealth offers an opportunity to capture socioeconomic position in older age, it has not been widely used. Its limited use could be due to the difficulty in collecting and combining information on different aspects of wealth including savings, inherited wealth, and household amenities.[479]

Therefore, attention needs to be paid to conceptual issues in measuring socioeconomic position in later life whether using education, occupational social class, house/car ownership, income or wealth. The use of different measures also depends on the feasibility of collection and appropriateness of the measure in the elderly. It is difficult to propose one indicator as the best measure over any other. However, it is important for studies to clarify why a particular indicator is chosen and what exactly it is used to measure. There may be merit in using more than one indicator to gain a better picture of the extent of socioeconomic inequalities in CHD in older age.

9.4.2 Pathways to socioeconomic inequalities in CHD in later life

9.4.2.1 Established and novel coronary risk factors

In this thesis the specific role of established and novel coronary risk factors in contributing to socioeconomic inequalities in CHD in older age was investigated (Chapter 6). The focus was on established risk factors including cigarette smoking, physical inactivity, BMI, blood pressure, and on novel risk factors such as inflammatory/haemostatic markers. The impact of these risk factors on relative inequalities is important in understanding their contribution to the relationship between socioeconomic position and CHD risk in older age. Previous studies in older populations have not investigated these pathways, particularly the role of novel risk factors to socioeconomic inequalities in CHD in later life; more evidence from future prospective studies is required to corroborate the findings of this thesis. Future studies could be improved by collecting more precise measurements of coronary risk factors. The risk factors used in the thesis were measured only once, and therefore lack long-term information on risk factors such as blood pressure and HDL-cholesterol. Imprecise measurement of these risk factors may contribute to residual confounding observed in relationships being studied, in this case of socioeconomic position and CHD.[482] Collecting information on risk factors more than once may reduce this bias. Further research is also needed to assess the effectiveness of change in behavioural factors in older age on reducing socioeconomic inequalities in CHD.

9.4.2.2 Exploring other pathways

Further research is required to explore other possible pathways to socioeconomic inequalities in CHD in older age, which were not investigated in this thesis. Studies have shown that lower socioeconomic groups have greater case fatality from CHD than higher socioeconomic groups, implying inequities in CHD treatment and management.[483-485] It is possible that socioeconomic differences in case fatality or differences in treatment may partly underlie the relationship between socioeconomic position and CHD mortality. A Finnish study of middle-aged subjects found that socioeconomic differences in case fatality contributed to a large proportion of socioeconomic inequalities in CHD mortality.[483] To explore this, detailed information on procedures including coronary bypass and revascularisation, and other aspects of quality of care such as time to treatment, use of statins and referral patterns would need to be collected and explored. Further research is also needed to investigate whether the relationship of socioeconomic position with CHD risk in older age is influenced by area-level deprivation. Socioeconomic conditions at area or neighbourhood level have been found to be associated with CHD risk independent of individual socioeconomic measures such as socioeconomic position.[486-489]

Another postulated mechanism to link socioeconomic position with CHD in middle-aged populations concerns psychosocial factors. Studies have shown that psychosocial factors including, job control, depression, social isolation and poor coping mechanisms, can contribute to socioeconomic inequalities in CHD.[25;38;490] These factors are found to be more prevalent in lower socioeconomic groups and have also been observed to be related to coronary risk.[491-495] One hypothesis is that psychosocial factors associated with low socioeconomic position can influence smoking, lack of physical activity, and in turn have an impact on haemostatic and lipid profiles;[491-493] a stressful psychosocial environment without adequate coping resources can lead to negative emotions, and in turn to adverse health behaviours such as smoking, poor diet and lack of exercise. Thus, psychosocial factors can have an indirect impact on CHD. Another hypothesis is that psychosocial factors may exert more of a 'direct' effect on coronary risk independent of health behaviours. Psychosocial stresses can trigger pathophysiologic processes through their influence on neuroendocrine systems and result in increased adiposity, hypertension, and activation of platelets and inflammatory markers such as C-reactive protein and interleukin-6.[491] While the role of job stress or low job control may be limited after retirement in older populations, psychosocial factors related to social support and network could be important in the elderly. Social networks represent formal or informal relationships with friends, family, clubs or groups, and indicate the extent to which individuals are engaged or integrated within societies.[496;497] From these relationships, individuals draw social support which can be emotional or functional or informational.[496;497] Previous studies have shown that low levels of social support and social network are associated with increased coronary risk.[493;498-500] In the elderly, who are more likely to experience social exclusion or isolation, it is important to ascertain whether levels of social support or network contribute to socioeconomic inequalities in CHD in older age. Future studies need to investigate this further. However, care is needed in investigating this issue since social support is closely related to socioeconomic position and also to behavioural risk factors.[501] Also, care needs to be taken in measuring social relationships; social support is mostly ascertained as perceived support,[493] which can be influenced by disease status. Other quantitative aspects of social relationships such as social network or ties are less subjective and are measured as number of friends/contacts, membership of clubs, and frequency of contact with friends and family.[493;496]

9.4.3 Childhood socioeconomic position and CHD risk in older age

In this thesis, information on early life socioeconomic position was collected retrospectively for one period of the life course (early childhood). Lower childhood socioeconomic position appeared to have some association with CHD risk in later life (Chapter 7). However, from these findings important implications arise for future studies. Ascertaining childhood socioeconomic position retrospectively in adult life may result in inaccurate recording of information. Recall bias is likely in the form of subjects, particularly from lower socioeconomic positions, over-estimating the social class of their father.[271] This can result in less marked childhood socioeconomic variations and a weakened effect of childhood socioeconomic position on adult disease.[271] Studies such as the British birth cohort studies, which follow-up subjects from birth have the advantage of recording parental occupation and other measures of childhood socioeconomic position more accurately.[502-504] Following-up a cohort from birth also provides advantages in the possibility of gaining information on socioeconomic position at different stages of life from birth, early childhood, later childhood, adolescence, early adult life, middle-age and old age. Along with information on socioeconomic position across the life course, other information on behavioural risk factors (smoking, physical activity and BMI) and CHD outcomes at different life stages would enable investigation of the temporal relationship between early life factors and adult behaviours. This information might help explore whether adult behavioural factors are mediators or confounders of the relation between early life socioeconomic position and CHD in old age. It would also be possible to investigate the cumulative effect of socioeconomic position across the life course on CHD risk in older age. Path analysis could be used to explore the effect of early life socioeconomic position on coronary risk in later life taking into account detailed information on intermediary factors including health behaviours and socioeconomic position in adolescence and middle-age. Despite these advantages, birth cohort studies involve substantial costs and are of prolonged duration when studying long-term outcomes such as CHD in old age. An alternative would be to follow-up subjects from early adult or middle-age to old age, and rely on accurate sources of data for information on early life factors such as birth or medical records, or school records for parental occupation and anthropometric measures, provided high retrieval rates for such records can be achieved. These data can complement adult recall information and provide valuable information on childhood socioeconomic position.

While in this thesis the association between childhood socioeconomic position and CHD risk in older age was investigated (Chapter 7), particular areas of further research are required to confirm this finding. First, in addition to behavioural risk factors, early life socioeconomic position is also known to be associated with biological coronary risk factors such as blood pressure, blood lipids and BMI, which can influence the relationship between childhood socioeconomic position and CHD in older age.[180;259;333;505] Lower childhood socioeconomic position has been observed to be associated with higher blood pressure in adult life, with the possibility of increased blood pressure tracking from childhood to adolescence and onto adulthood.[265;506-508] Similar associations of lower early life socioeconomic position with increased BMI,[333] and tracking of BMI from early to adult life have been reported.[509] The extent to which these coronary risk factors influence the relation between early life socioeconomic position and CHD risk in later life needs to be investigated. Second, some studies also show a relationship between childhood socioeconomic position and novel coronary risk factors such as fibrinogen in adulthood.[223;231] This relationship needs to be further explored with other inflammatory markers. Third, the influence of other early life exposures such as fetal undernutrition or low birth weight, postnatal growth and breastfeeding, which have been implicated in increasing CHD risk in adult life,[52] were not taken into account in this thesis. The extent to which these early life exposures are associated with CHD risk in older age independent of childhood socioeconomic position, or their influence on the association between childhood socioeconomic position and CHD risk in older age warrants further investigation.

9.5 Recommendations

Results of this thesis demonstrate that *relative* socioeconomic inequalities in CHD have not narrowed over the last 25 years in Britain. While social class inequalities in CHD narrowed with age (in relative terms), absolute social class differences increase with age. Marked socioeconomic differences in CHD persist in older British men. The proportion of older people in the UK population, as in most developed countries, is growing rapidly; the proportion of the UK's population aged over 65 years has doubled since the 1930s.[424] Coronary risk also increases with age.[6] Therefore, socioeconomic inequalities associated with CHD in the elderly pose an important public health problem. Current policies place little emphasis on reducing inequalities in CHD in older populations. In 2002, the Government announced a target to reduce inequalities in health (as measured by infant mortality and life expectancy) by 10% by 2010.[510] Similarly, specific targets to reduce socioeconomic inequalities in CHD in older age are also required. A substantial proportion of socioeconomic inequalities in CHD in older age can be reduced by targeting established coronary risk factors particularly cigarette smoking, and also physical activity and BMI. Reducing overall levels as well as socioeconomic differences in these risk factors in older age would be important for narrowing current socioeconomic inequalities in CHD in older age. Understanding the origins of these risk factors across the life course would also be valuable in preventing these inequalities. Individual-level interventions have shown limited benefits in long-term improvement of health behaviours or risk factors.[451;511] Population-wide approaches to reducing risk factor levels with specific efforts targeted at lower socioeconomic groups would be more effective.[451;511] Some of these interventions include restricting smoking advertisements, improving food labelling, improving the environment to promote physical activity, and other fiscal and legislative changes.[451;511] Addressing the fundamental social, economic and material context in order to influence the causes of socioeconomic inequalities through public policy is essential if socioeconomic inequalities in CHD in older men are to be reduced.

Appendix I Social class distribution of subjects at twenty-year follow-up according to social class measured at baseline

Social class at baseline (1978-80) based on longest-held occupation at 40-59 years	**Social class at follow-up** (1998-2000) at age 60-79 years, based on most-recent or last occupation before retirement								
	I	II	III non-manual	III manual	IV	V	Total	Non-manual	Manual
I	223 (56%)	110 (28%)	22 (6%)	28 (7%)	11 (3%)	0	394 (100%)		
II	82 (9%)	661 (61%)	182 (17%)	118 (11%)	30 (3%)	7 (1%)	1080 (100%)	1624 (86%)	269 (14%)
III non-manual	19 (5%)	123 (29%)	202 (48%)	38 (9%)	29 (7%)	8 (2%)	419 (100%)		
III manual	33 (2%)	170 (11%)	95 (6%)	974 (62%)	248 (16%)	60 (4%)	1580 (100%)		
IV	2 (1%)	16 (5%)	19 (5%)	100 (29%)	176 (52%)	27 (8%)	340 (100%)	346 (17%)	1684 (83%)
V	0	2 (2%)	9 (8%)	44 (40%)	24 (22%)	31 (28%)	110 (100%)		
Total	359 (9%)	1082 (28%)	529 (14%)	1302 (33%)	518 (13%)	133 (3%)	3923 (100%)		

Appendix II

Publications from this thesis

1. Ramsay SE, Morris RW, Whincup PH, Lennon LT, Wannamethee SG. Are social inequalities in mortality in Britain narrowing? Time trends from 1978 to 2005 in a population-based study of older men. J Epidemiol Community Health 2008; 62:75-80.
2. Ramsay S, Lowe GDO, Whincup PH, Rumley A, Morris RW, Wannamethee G. Relationships of inflammatory and haemostatic markers with social class: Results from a population-based study of older men. Atherosclerosis 2008; 197:654-661.
3. Ramsay SE, Whincup PH, Morris R, Lennon L, Wannamethee SG. Is socioeconomic position related to the prevalence of metabolic syndrome? Influence of social class across the life-course in a population-based study of older men. Diabetes Care 2008; 31:2380-2382.
4. Ramsay SE, Whincup PH, Morris RW, Lennon LT, Wannamethee SG. Are childhood socio-economic circumstances related to coronary heart disease risk? Findings from a population-based study of older men. Int J Epidemiol 2007; 36:560-566.
5. Ramsay SE, Whincup PH, Morris RW, Lennon LT, Wannamethee SG. Extent of Social Inequalities in Disability in the Elderly: Results From a Population-based Study of British Men. Ann Epidemiol 2008; 18:896-903.

Appendix III BRHS questionnaires

The subsequent pages include the following British Regional Heart Study questionnaires which are relevant to this thesis, and the general practice medical record review sheet:

1. Baseline questionnaire in 1978-80
2. Postal questionnaire in 1992
3. Questionnaire in 1998-2000 at twenty-year follow-up
4. Dietary questionnaire in 1998-2000
5. Postal questionnaire in 2003
6. General practice medical record review sheet used for two-yearly update of morbidity data

Baseline questionnaire (1978-80)

1

Serial Number	☐☐☐☐☐☐☐☐	1
Card Number	0 1	9
Date of Screening	☐☐☐☐☐☐	11
Time of Screening	☐☐☐☐	17

1. GENERAL

What is your date of birth? Day ☐☐ 21
Month ☐☐ 23
Year 19 ☐☐ 25

Where were you born?
Town ……………………………………
County ……………………………………
Country ……………………………………

1.2 How many years have you lived within 10 miles of this town? ☐☐ 27 years
If you have moved to this area within the last five years, where did you move from?
……………………………………………………………

1.3 What is your marital status?

Single	1	☐	29
Married	2		
Widowed	3		
Other	4		

1.4 How many children do you have?

	M	F	
<5 yrs	☐	☐	30
5-10 yrs.	☐	☐	32
11-16 yrs.	☐	☐	34
> 16 yrs.	☐	☐	36

2 YOUR FATHER

2.1 Where was your Father born?
Town ……………………………………
County ……………………………………
Country ……………………………………

2.2 Is your father alive? (Y/N) ☐ 38

2.3 How old is he now? / How old was he when he died? ☐☐ 39 years

2.4 If your father has died, what were you told was the cause of his death?

Heart trouble	1		
High blood pressure	2		
Stroke	3		
Respiratory disease	4	☐	41
Cancer of lung	5		
Other cancer	6		
Accident or injury	7		
Other	8		
Don't know	9		

3 YOUR MOTHER

3.1 Where was your mother born?
Town ……………………………………
County ……………………………………
Country ……………………………………

3.2 Is your mother alive? (Y/N) ☐ 42

3.3 How old is she now? / How old was she when she died? ☐☐ years 43

3.4 If your mother has died, what were you told was the cause of her death?

Heart trouble	1		
High blood pressure	2		
Stroke	3		
Respiratory disease	4	☐	45
Cancer of breast	5		
Other cancer	6		
Accident or injury	7		
Other	8		
Don't know	9		

4. OCCUPATION

4.1 What is your present job? ……………………………………………

If employed go to question 4.4

4.2 If you are unemployed, for how long has this been?

<6weeks	1		
6wk.-5mo.	2		
6mo. –1yr.	3	☐	46
> 1 year	4		

4.3 Is this because of ill health? (Y/N) 47

....

4.4 What kind of work have you done for the longest period of time?

....

4.5 What business or industry is this?

....

4.6 How many years have you done this kind of work? years 48

4.7 Are / were you:

SELF-EMPLOYED	with 25 or more employees	1	
	with less than 25 employees	2	
	without employees	3	50
MANAGER	of 25 or more people	4	
	of less than 25 people	5	
FOREMAN	....	6	
ORDINARY EMPLOYEE	....	7	
ARMED SERVICES	....	8	

5 <u>SEVERE CHEST PAIN</u>

5.1 Have you <u>ever</u> had a <u>severe</u> pain in your chest lasting for half an hour or more? (Y/N) 51
<u>If NO, go to question 6.</u>

5.2 Where did you get this severe pain? 52
(Show chart.)

5.3 Did you see a doctor because of this pain? (Y/N) 55

6 <u>CHEST PAIN</u>

6.1 Do you ever have any pain or discomfort in your chest? (Y/N) 56
<u>If NO, go to question 7.</u>

6.2 When last did you get the pain? 57

Within 1 month	1	
1-5 months ago	2	
6-12 months ago	3	
Over 1 year ago	4	
Occasionally	5	

6.3 How often do you get it? 58

Daily	1
Weekly	2
Monthly	3
Once only	4
Occasionally	5

6.4 Where do you get this pain or discomfort? 59
(Show chart.)

6.5 When you walk at an ordinary pace on the level, does this produce the pain? (Y/N) 62

6.6 When you walk uphill or hurry, does this produce the pain? (Y/N) 63

6.7 When you get any pain or discomfort in your chest on walking, what do you do? 64

Stop	1
Slow down	2
Continue at the same pace	3

6.8 Does the pain or discomfort in your chest go away if you stand still? (Y/N) 65

6.9 How long does it take to go away? 66

10 minutes or less	1
more than 10 minutes	2

7.0 <u>PHLEGM, COUGH AND BREATHING</u>

7.1 Do you usually bring up phlegm (spit) from your chest first thing in the morning in the winter? (Y/N) 67
<u>If NO, go to question 7.4</u>

7.2 Do you bring up phlegm like this on most days for as much as 3 months in the winter each year? (Y/N) 68

7.3 In the past 3 years have you ever had a period of increased cough and phlegm lasting 3 weeks or more? 66

Yes, once	1
Yes, twice or more	2
Never	3

7.4 Does your chest sound wheezy or whistling on most days (or nights)? (Y/N) 70

7.5 Does the weather affect your breathing?
And if so, at what season of the year is it most affected?

Not affected	1	71
Winter	2	
Summer	3	
Both	4	

8 BREATHLESSNESS

8.1 Do you get short of breath walking with people your own age on level ground? (Y/N) 72

8.2 On walking up hills or stairs, do you get more breathless than people you on age? (Y/N) 73

8.3 Do you ever have to stop walking because of breathlessness? (Y/N) 74

S.E.G. 75

Social class 77

Activity score 78

Serial Number

Card Number 0 2

9 LEG PAIN

9.1 Do you ever get pain in your calf muscles on walking at an ordinary pace, on the level? (Y/N) 11

9.2 Do you get pain in your calf muscles when you walk uphill or hurry? (Y/N) 12

10 MEDICAL HISTORY

10.1 Have you ever been told by a doctor that you have, or have had, any of the following?

Angina	(Y/N)	13
Heart attack	(Y/N)	14
Coronary thrombosis	(Y/N)	15
Myocardial infarction	(Y/N)	16
Other heart trouble	(Y/N)	17
High blood pressure	(Y/N)	18
Stroke	(Y/N)	19
Diabetes	(Y/N)	20
Peptic ulcer	(Y/N)	21
Gout	(Y/N)	22
Gall bladder disease	(Y/N)	23
Thyroid disease	(Y/N)	24
Arthritis	(Y/N)	25
Bronchitis	(Y/N)	26
Asthma	(Y/N)	27
Other condition (s) including surgery……………………………………	(Y/N)	28

10.2 Are you on any regular medical treatment from a doctor for any condition? (Y/N) 29

If NO, go to question 10.3

Do you know if the pills / medicines /injections are:-

Tranquillizers	Y/N	30
Pain killers	Y/N	31
Antihypertensive drugs	Y/N	32
Anti coagulants	Y/N	33
Lipid lowering drugs	Y/N	34

	Oral antidiabetics	Y/N	35
	Injection of insulin	Y/N	36
	Any others	Y/N	37
	Don't know	Y/N	38
10.3	Have you taken any of these in the last 48 hours?		
	Tranquillizers	Y/N	39
	Pain killers	Y/N	40
	Antihypertensive drugs	Y/N	41
	Anti coagulants	Y/N	42
	Lipid lowering drugs	Y/N	43
	Oral antidiabetics	Y/N	44
	Injection of insulin	Y/N	45
	Any others	Y/N	46
	Don't know	Y/N	47

11 DIET & ALCOHOL

11.1 How many times during an average week would you have the following foods?

Meat (including beef, lamb, pork, bacon in any form)	48
Chicken	50
Fish	52
Eggs - how many eggs do you eat in a week	54
Cheese – how often do you eat cheese, including cheese dishes?	56
Breakfast cereals – how often do you eat these (porridge included)? State kind …………………………………………	58

11.2 What kinds of bread do you eat ?

White	Y/N	60
Brown	Y/N	61
Wholemeal	Y/N	62
Other	Y/N	63

11.3 Spreading fats: What kinds do you use at home?

Butter	Y/N	64
Margarine ……………………	Y/N	65

(State kind or brand name.)

11.4 Do you take sugar?

In tea	Y/N	66
In coffee	Y/N	67
In other drinks	Y/N	68

11.5 Do you use milk?

On cereals	Y/N	69
In tea	Y/N	70
In coffee	Y/N	71
As a milk drink	Y/N	72

11.6 (i) Would you describe your present alcohol intake as:

None	1	
On special occasions only	2	73
Once or twice a month	3	
Weekends	4	
Daily / most days	5	

If NONE, go to question 12

(ii) What type of drink do you usually take?

Beer	1	
Spirits	2	
Wine/sherry	3	74
Mixed beer & spirits	4	
Mixed beer, spirits, wine and sherry	5	

(iii) How much do you usually take?

2 drinks a day or less	1	
3-6 drinks a day	2	
more than 6 drinks a day	3	75

(One drink is a single whisky, gin or brandy, a glass of wine, sherry or port or half a pint of beer.)

Serial Number
Card Number 0 3

12 SMOKING

12.1 (i) Do you smoke at present?

Yes, regularly	1		
No	2		11
Occasionally	3		

If NO, go to question 12.6

(ii) How old were you when you started? years 12

(iii) Have you ever given up smoking? (Y/N) 14

(iv) If yes, what is the maximum time for which you have given up smoking? years 15

12.2 (i) Do you smoke cigarettes now?

Yes regularly	1		
No	2		17
Occasionally (<1 day)	3		

If NO, or OCCASIONALLY go to question 12.3

(ii) How many cigarettes do you usually smoke a day? 18

(iii) If hand rolled, how much tabacco do you use a week? (ozs.) ozs. 20

Now proceed to 12.4

12.3 (i) Were you previously a regular cigarette smoker? (Y/N) 22

(ii) If Yes, how many cigarettes did you usually smoke a day? 23

(iii) At what age did you change to a pipe and / or cigars? years 25

12.4 (i) Do you smoke a pipe now?

Yes regularly	1		
No	2		27
Occasionally	3		

If NO or OCCASIONALLY go to question 12

(ii) If YES, how many ozs. a week do you smoke? ozs. 20

12.5 (i) Do you smoke a pipe now?

Yes regularly	1		
No	2		30
Occasionally	3		

(ii) If YES, how many cigars do you smoke a day?

Large 31
Small 32

If you smoke ANYTHING currently, go to question 13.

12.6 (i) Have you ever smoked for a more than 1 month ? (Y/N) 35

How much did you usually smoke

Cigarettes (per day)		36
Pipe (ozs) (per week)		38
Cigars (per day)	Large	40
	Small	42

If NO, go to question 13.

(ii) At what age did you start smoking? years 44

(iii) At what age did you finally stop smoking? years 46

(iv) What was the maximum time between these two ages for which you gave up smoking? years 48

13 EXERCISE

13.1 (i) Do you usually walk or cycle in the course of your journeys to or from work each day?

No	1	
Walk	2	50
Cycle	3	

If YES, how many minutes do these journeys take? mins 51

(ii) Apart from your journeys to or from work, do you usually walk or cycle on weekdays?

No	1	
Walk	2	50
Cycle	3	

If YES, how many minutes do you walk/cycle each day? mins 51

(iii) Would you say that in your occupation you are physically :

Very active	1	
Fairly active	2	
Average	3	56
Fairly inactive	4	
Very inactive	5	

13.2 On average, a man of your age spends 4 hours on most weekends on some of the following activities: walking, gardening, household chores, DIY projects. Compared to such a man, how physically active do you consider yourself?

Very active	1	
Fairly active	2	
Average	3	57
Fairly inactive	4	
Very inactive	5	

13.3 Apart from these activities, do you take active physical exercise, e.g. running, digging, swimming, tennis, golf, sailing, etc.

No	1		
Occassionally	2		
Frequently	3	[ ]	58

<u>If NO or Occasionally – stop here.</u>

13.4 Please state type of activity...

13.5 How many years have you been involved in this activity? [ ][ ] years 59

13.6 How many times a month (on average) do you undertake these activities?

Winter	[ ][ ]	61
Summer	[ ][ ]	63

Administrator	[ ]	65
Coder	[ ]	66

BRITISH REGIONAL HEART STUDY QUESTIONNAIRE

1992

Royal Free Hospital School of Medicine
Department of Public Health and Primary Care
Rowland Hill Street
London NW3 2PF

Health

Please answer the following questions by filling in the appropriate box with a tick or an answer in the space provided.

office use 0 1

1.0 Please write your date of birth here ☐ day ☐ month ☐ year

2.0 How would you describe your health at present?

Excellent	☐	1
Good	☐	2
Fair	☐	3
Poor	☐	4

3.0 Have you ever been told by your doctor that you have, or have had any of the following?

	Yes	No
Heart attack, coronary thrombosis or myocardial infarction	☐	☐
Angina	☐	☐
Other heart trouble	☐	☐
High blood pressure	☐	☐
Stroke	☐	☐
Diabetes	☐	☐
Aortic aneurysm	☐	☐
Gastric, peptic or duodenal ulcer	☐	☐
Gout	☐	☐
Gall bladder disease	☐	☐
Thyroid disease	☐	☐
Arthritis	☐	☐
Bronchitis	☐	☐
Asthma	☐	☐
Cancer	☐	☐

If you have ever had cancer please state what kind of cancer

office use

4.0 Have you ever had any of the following ?

	Yes	No
A referral to a heart specialist	☐	☐
An ECG Exercise test - bicycle	☐	☐
- treadmill	☐	☐
X-ray or angiogram of your coronary arteries	☐	☐
Angioplasty of coronary arteries	☐	☐
Coronary artery by-pass graft	☐	☐
Surgery to aorta for aneurysm	☐	☐
Other heart surgery - Valves	☐	☐
- Pacemaker	☐	☐
Any other major surgery?	☐	☐

If you have had any other major surgery please give details

office use

5.0 Are you on any regular treatment from a doctor for any condition? Yes ☐ No ☐

If you answered YES

5.1 Please tick all those you are on and give the name of tablets if possible

	Yes	No	Name of tablet
Tablets to lower blood pressure	☐	☐	________
Diuretics (water tablets)	☐	☐	________
Tranquillisers	☐	☐	
Anti-depressants	☐	☐	
Sleeping tablets	☐	☐	
Insulin injections	☐	☐	
Diet for diabetes	☐	☐	
Other regular treatment	☐	☐	

Please specify other treatments

5.2 Do you take aspirin on a regular of daily basis? Yes ☐ No ☐

If you answered YES,
How many tablets of aspirin do you usually take each week

If you have diabetes

5.3 What year was your diabetes first diagnosed? 19

5.4 What year did you begin regular treatment with diet or drugs for diabetes? 19

5.5 Do you currently attend a diabetic clinic?

- No not at all ☐ 1
- Yes, at the hospital ☐ 2
- Yes, at the GP surgery ☐ 3

6.0 Family History

6.1 Is your father alive Yes ☐ No ☐

6.2 How old is he now? or How old was he when he died?

6.3 Did / does he ever suffer from: If dead, did he die from

	Yes	No	Yes	No
Heart trouble	☐	☐	☐	☐
High blood pressure	☐	☐	☐	☐
Stroke	☐	☐	☐	☐
Diabetes	☐	☐	☐	☐
Cancer	☐	☐	☐	☐

office use

0 2

6.4 Is your mother alive Yes ☐ No ☐

6.5 How old is she now? or How old was she when she died?

6.6 Did / does she ever suffer from: If dead, did she die from

	Yes	No	Yes	No
Heart trouble	☐	☐	☐	☐
High blood pressure	☐	☐	☐	☐
Stroke	☐	☐	☐	☐
Diabetes	☐	☐	☐	☐
Cancer	☐	☐	☐	☐

Family history continued

6.7 How many older brothers or sisters do you have? (include any who have died) ☐

6.8 How many younger brothers or sisters do you have? (include any who have died) ☐

	Yes	No
6.9 Are you one of twins or triplets	☐	☐

6.10 Have any of your brothers and sisters ever suffered from

	Yes	No	How many of them have this problem
Heart trouble	☐	☐	☐
High blood pressure	☐	☐	☐
Stroke	☐	☐	☐
Diabetes	☐	☐	☐
Cancer	☐	☐	☐

	Yes	No
6.11 Did any of your brothers or sisters die at birth or in their first year of life?	☐	☐

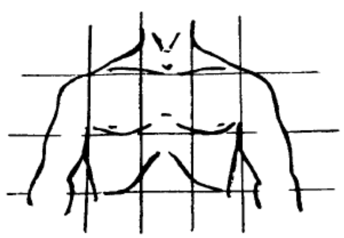
7.0 Chest pain

	Yes	No
7.1 Do you ever have any pain or discomfort in your chest?	☐	☐

7.2 Where do you get this pain or discomfort?

Please mark X on the appropriate places

YOUR RIGHT SIDE



YOUR LEFT SIDE

office use ☐ ☐ ☐

Chest pain continued

	Yes	No
7.3 When you walk at an ordinary pace on the level, does this produce the pain?	☐	☐
7.4 When you walk uphill or hurry, does this produce the pain?	☐	☐

7.5 When you get any pain or discomfort in your chest on walking, what do you do?

- Stop ☐ 1
- Slow down ☐ 2
- Continue at the same pace ☐ 3

	Yes	No
7.6 Does the pain or discomfort in your chest go away if you stand still?	☐	☐

7.7 How long does it take to go away?

- 10 minutes or less ☐ 1
- more than 10 minutes ☐ 2

8.0 *Severe chest pain*

	Yes	No
8.1 Have you ever had a severe pain across the front of your chest lasting for half an hour or more ?	☐	☐

If you answered No please go to section 9.0

If you answered Yes,

	Yes	No
8.2 Did you see a doctor because of this pain?	☐	☐

8.3 What year(s) did this happen? 19 ☐ and 19 ☐

9.0 Leg Pain

	Yes	No
9.1 Do you ever get pain in your calf when walking at an ordinary pace on the level?	☐	☐
9.2 Do you get pain in your calf muscle when you walk uphill or hurry?	☐	☐

If YES,

	Yes	No
9.3 Does the pain go away if you stop or stand still?	☐	☐

9.4 How long does it take to go away?

- 10 minutes or less ☐ 1
- more than 10 minutes ☐ 2

10.0 Breathlessness

		Yes	No
10.1	Do you get short of breath walking with other people of your own age on level ground?	☐	☐
10.2	On walking uphill or stairs do you get more breathlessness than people of your own age?	☐	☐
10.3	Do you ever have stop walking because of breathlessness?	☐	☐

11.0 Cough and Wheeze

		Yes	No
11.1	Do you usually bring up phlegm (spit) from your chest first thing in the morning in the winter?	☐	☐
	If you answered No then go to question 11.4		
11.2	Do you bring up phlegm like this on most days for as much as 3 months in the winter each year?	☐	☐

11.3 In the past 5 years have you ever had a period of increased cough and phlegm lasting 3 weeks or more?

- Yes, once ☐ 1
- Yes, twice or more ☐ 2
- Never ☐ 3

		Yes	No
11.4	Does your chest sound wheezy or whistling on most days or nights?	☐	☐

11.5 Does the weather affect your breathing and if so what season of the year is it most affected?

- Not affected ☐ 1
- Winter ☐ 2
- Summer ☐ 3
- Both ☐ 4

12.0 *Weight*

12.1 Has your weight changed in the last five years?

- No change ☐ 1
- Increased ☐ 2
- Decreased ☐ 3
- Don't Know ☐ 4

12.2 What is your present weight? ☐ stones ☐ pounds or ☐ kilos

(Indoor clothes, no shoes)

If you have no scale, please fill in an estimate.

13.0 *Personal Circumstances*

office use 0 3

13.1 Are you

- Married ☐ 1
- Single ☐ 2
- Widowed ☐ 3
- Divorced or separated ☐ 4
- Other ☐ 5

13.2 Please describe your accommodation. Are you

- an owner occupier ☐ 1
- renting privately ☐ 2
- renting from the council ☐ 3
- other (please specify below) ☐ 4

office use

13.2 How many cars are there available for use in your household?

- None ☐ 1
- One ☐ 2
- Two or more ☐ 3

14.0 *Smoking*

		Yes	No
14.1	Do you regularly smoke cigarettes at present?	☐	☐

If YES,

14.2 How many cigarettes do you smoke a day? ☐

14.3 Have you changed your smoking habits over the last 5 years?

- No ☐ 1
- Yes increased ☐ 2
- Yes decreased ☐ 3
- Yes given up ☐ 4

		Yes	No
14.4	Do you currently smoke a pipe or cigars?	☐	☐

If No,

		Yes	No
14.5	Have you ever regularly smoked a pipe or cigars?	☐	☐

For those not smoking at present

		Yes	No
14.6	Were you previously a regular cigarette smoker?	☐	☐

If YES,
14.7 At what age did you give up? ☐

14.8 Why did you give up?

Personal choice	☐	1
Doctor's advice	☐	2
Definite illness	☐	3

15.0 *Drinking Alcohol*

15.1 Would you describe your present alcohol intake as

Daily / most days	☐	1
Weekends only	☐	2
Once or twice a month	☐	3
None	☐	4

15.2 *One drink is HALF a pint of beer, a SINGLE whisky, gin etc or a glass of wine or sherry.*

How much do you usually drink?

More than 6 drinks a day	☐	1
3-6 drinks a day	☐	2
2 drinks a day or less	☐	3
None	☐	4

		Yes	No
15.3	Have you ever been a regular drinker of more than 6 drinks daily?	☐	☐

15.4 What type of drink do you usually take?

Beers, lagers	☐	1
Wines, sherry	☐	2
Spirits	☐	3
Variety of beers, wines or spirits	☐	4
Low alcohol drinks	☐	5

Drinking continued

		Yes	No
15.5	Have you reduced your alcohol intake in the last five years?	☐	☐

If NO, go to question 16.0

If YES
15.6 Was this due to

Personal choice	☐	1
Doctor's advice	☐	2
Definite illness	☐	3

16.0 *For drinkers and ex drinkers*

		Yes	No
16.1	Have you ever felt that you should cut down on your drinking?	☐	☐
16.2	Have other people annoyed you by criticising your drinking habits?	☐	☐
16.3	Have you ever felt guilty about drinking?	☐	☐
16.4	Have you ever taken a drink in the morning to steady your nerves or get rid of a hangover?	☐	☐

17.0 *For people who do not drink at present*

17.1 Why do you not drink at present?

Personal choice	☐	1
Doctor's advice	☐	2
Definite illness	☐	3

		Yes	No
17.2	Did you drink in the past?	☐	☐

If YES
17.3 For how long have you given up?

Less than 5 years	☐	1
5-10 years	☐	2
11-20 years	☐	3
more than 20 years	☐	4

18.0 *Physical activity*

18.1 Do you usually walk or cycle in the course of your journey to or from work each day?

- No ☐ 1
- Walk ☐ 2
- Cycle ☐ 3
- Not applicable ☐ 4

If YES,

18.2 How many minutes do these journeys take in total each day? ☐ minutes

18.3 Apart from any journeys to or from work, do you usually walk or cycle on weekdays?

- No ☐ 1
- Walk ☐ 2
- Cycle ☐ 3

If YES,

18.4 How many minutes do these journeys take in total each day? ☐ minutes

18.5 Would you say that in your occupation you are or were physically

- Very active ☐ 1
- Fairly active ☐ 2
- Average ☐ 3
- Fairly inactive ☐ 4
- Very inactive ☐ 5

18.6 *On average a man of your age spends 4 hours on most weekends on some of the following activities:*
walking, gardening, household chores, DIY projects.

Compared to such a man, how physically active do you consider yourself?

- Very active ☐ 1
- Fairly active ☐ 2
- Average ☐ 3
- Fairly inactive ☐ 4
- Very inactive ☐ 5

Physical activity continued

18.7 How many hours a week do you spend gardening

	In the spring/summer	In the autumn/ winter?
Hours of light gardening work per week	☐	☐
Hours of moderate gardening work per week	☐	☐
Hours of heavy digging gardening work per week	☐	☐

18.8 Do you take active physical exercise such as running, swimming, golf, tennis, squash, jogging, bowls, cycling etc.?

- No ☐ 1
- Occasionally (less than once a month) ☐ 2
- Frequently (once a month or more) ☐ 3

If you ticked **No** or **Occasionally** then please go to question 19.0

If you ticked **Frequently** (once a month or more),

18.9 Please state type of activities :

☐ *office use* ☐☐

18.10 How many years have you been involved in this activities ? ☐

18.11 How many **times** a month (on average) do you take part in this activities in

- Winter ☐
- Summer ☐

19.0 *Disability*

Do you currently have difficulty carrying out any of the following activities on your own as a result of a long term health problem?

	Yes	No
Going up or down stairs	☐	☐
Bending down	☐	☐
Straightening up	☐	☐
Keeping your balance	☐	☐
Going out of the house	☐	☐
Walking 400 yards	☐	☐

office use 0 4

If you ticked No in all cases then please go to question 20.0

19.2 Is your present state of health causing problems with any of the following

	Yes	No
Job at work (paid employment)	☐	☐
Household chores	☐	☐
Social life	☐	☐
Sex life	☐	☐
Interests and hobbies	☐	☐
Holidays and outings	☐	☐
Family relationships	☐	☐

19.3 If you have ticked YES in questions 19.1 or 19.2 please give details of the condition that you have which causes you these difficulties?

office use

20.0 *Falls*

20.1 Have you had a fall in the last year? Yes ☐ No ☐

If NO please go to question 21.0

If YES,

20.2 How many times?

20.3 Did you have medical attention for any of these falls? Yes ☐ No ☐

21.0 *Present Employment*

21.1 At present are you

Employed full time	☐	1
Employed part time	☐	2
Unemployed, seeking work	☐	3
Unemployed, not seeking work	☐	4
Retired	☐	5

21.2 If unemployed, is this due to

Redundancy	☐	1
Illness/ disability	☐	2
Other reasons	☐	3

If you ticked Other reasons please give details below

office use

21.3 If retired, is this due to

Normal retiring age	☐	1
Early retirement opportunity	☐	2
Illness/ disability	☐	3
Redundancy	☐	4
Other reasons	☐	5

Employment continued

21.4 What job have you done for the longest period of time?

office use

21.6 Would you describe this work as

Manual	☐	1
Non-manual	☐	2

21.5 What job did you father do for the longest period of his working life?

office use

21.6 Would you describe this work as

Manual	☐	1
Non-manual	☐	2

22.0 *When you were a child (up to 10 years old)*

		Yes	No
22.1	Did you have a bathroom in your house?	☐	☐
22.2	Did you have a hot water tap in the house?	☐	☐
22.3	Did you share a bedroom with brothers or sisters?	☐	☐
22.4	Did your family own a car?	☐	☐

23.0 *At present*

		Yes	No
23.1	Do you have access to a telephone in your house?	☐	☐
23.2	Have you made a personal phone call in the last week?	☐	☐
23.3	Have you written a personal letter in the last week?	☐	☐
23.4	Do you take a weekly or monthly magazine or journal?	☐	☐
23.5	Do you attend religious services or meetings?	☐	☐
23.6	Did you vote in the last general or local elections?	☐	☐
23.7	Have you been on holiday in the last year?	☐	☐
23.8	Are you planning to go on holiday next year?	☐	☐
23.9	Do you use the public library?	☐	☐
23.10	Are you a member of any club, society or group?	☐	☐
23.11	If YES, In the past month have you attended a meeting of a club, society or group?	☐	☐

Thank you for you help

All your answers will be treated in complete confidence and will not be identifiable. Please you would check that you have answered all the questions you can, and then return the form in the envelope provided, NO STAMP IS NEEDED.

University of London Senate House Printing Services 44789 10m 10/92

Study Number : ☐☐☐☐☐☐☐☐

BRITISH REGIONAL HEART STUDY

20 YEAR FOLLOW-UP SURVEY

Thank you for attending this follow-up survey. It would be very helpful if you could complete this questionnaire, which will bring us up to date with your health and lifestyle.

Most questions can be answered simply by ticking the correct box ☑

All information will be treated as **strictly confidential**.

The Research Nurse will help you with any problems.

Thank you for your help.

Conditions affecting the heart or circulation

1.0 Have you **ever** been told by a doctor that you have or have had any of the following conditions ?

		Yes	No	If after 1996, please give year
(a)	Heart attack (coronary thrombosis or myocardial infarction)	☐	☐	19_______
(b)	Heart failure	☐	☐	19_______
(c)	Angina	☐	☐	19_______
(d)	Other heart trouble	☐	☐	19_______
(e)	High blood pressure	☐	☐	19_______
(f)	Aortic Aneurysm	☐	☐	19_______
(g)	Narrowing or hardening of the leg arteries (including claudication)	☐	☐	19_______
(h)	Deep Vein Thrombosis (clot in the deep leg vein)	☐	☐	19_______
(i)	Pulmonary Embolism (clot on the lung)	☐	☐	19_______

Treatment for heart trouble

2.0 Have you **ever** had any of the following **TREATMENTS** for chest pain or heart disease ?

		Yes	No	If **Yes,** please give year of treatment	
(a)	Angioplasty of coronary arteries ('balloon treatment')	☐	☐	19_______	19_______
(b)	Coronary artery bypass graft (CABG) operation	☐	☐	19_______	19_______

Stroke

		Yes	No	Year of first diagnosis
3.0	Have you **ever** been told by a doctor that you have had a stroke ?	☐	☐	19_______
(a)	**If Yes,** did the symptoms last for more than 24 hours ?	☐	☐	

Cancer

		Yes	No
4.0	Have you **ever** been told by a doctor that you have or have had Cancer ?	☐	☐

If Yes, please give the following information:-

OFFICE USE

(a) Cancer Site_________________ ☐☐☐ Year first diagnosed 19________

Diabetes

Please answer all the questions

		Yes	No
5.0	Have any of your close 'blood' relatives (your parents, brothers or sisters) **ever** had diabetes ?	☐	☐

If Yes, please list any of these relatives who have had diabetes and if possible their age when they were first diagnosed:

OFFICE USE

(a) Mother ____ ☐☐

(b) Father ____ ☐☐

(c) Brothers ____ ☐ ☐☐

(d) Sisters ____ ☐ ☐☐

		Yes	No
5.1	Have you **ever** been told by a doctor that you have (or have had) diabetes?	☐	☐

(a) **If Yes,** in what year was your diabetes first diagnosed ? 19_________

Chest pain

6.0 Do you ever have any pain or discomfort in your chest ?

Yes ☐

No ☐ ➔ If **No**, go to Question 7.0 on the next page

		Yes	No
6.1	Do you know the cause of the pain ?	☐	☐

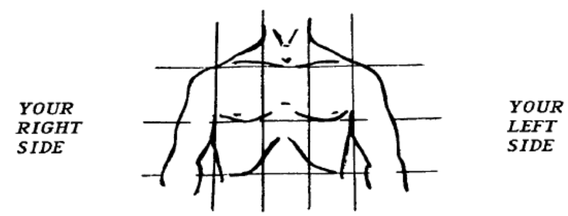
OFFICE USE

(a) **If Yes**, please state:

____ ☐

(b) Where do you get this pain or discomfort ?

Please mark **X** on the appropriate places



OFFICE USE

☐☐☐

(c) When you walk at an ordinary pace on the level does this produce the chest pain ?

Yes ☐1
No ☐2
Unable to walk on level ☐3

(d) When you walk uphill or hurry does this produce the chest pain ?

Yes ☐1
No ☐2
Unable to walk on level ☐3

Chest pain continued

(e) When you get any pain or discomfort in your chest on walking, what do you do?

- Yes ☐1
- No ☐2
- Continue at the same pace ☐3

(f) Does the pain or discomfort in your chest go away if you stand still? Yes ☐ No ☐

(g) How long does it take to go away ?

- 10 minutes or less ☐1
- More than 10 minutes ☐2

(h) Overall is the chest pain

- Becoming more frequent ☐1
- Staying about the same ☐2
- Becoming less frequent ☐3

Previous Chest Pain

7.0 Have you previously had chest pain, which has stopped because of an operation? Yes ☐ No ☐

(a) If **Yes,** please give details:

____

OFFICE USE ☐

Severe chest pain

8.0 Have you **ever** had a **severe** pain across the front of your chest lasting for half an hour or more ?

Yes☐ No☐ ➔ If **No**, go to question 9.0 on the next page

(a) If **Yes**, what year did this happen ? 19________

(b) Did you see a doctor because of this pain? Yes ☐ No ☐

(c) If **Yes**, what were you told was the cause_______________________________

OFFICE USE ☐

Leg pain

9.0 Do you get pain or discomfort in your leg (or legs) when you walk?

- Yes ☐1
- No ☐2
- Unable to walk ☐3 ➔ If **No** or **Unable to walk**, go to question 10.0, on the next page

9.1 Do you know the cause of the pain ? Yes ☐ No ☐

(a) If **Yes,** please state: -

____

OFFICE USE ☐

(b) Does this pain ever begin when you are standing still or sitting ? Yes ☐ No ☐

(c) Do you get the pain if you walk uphill or hurry?

- Yes ☐1
- No ☐2
- Unable to walk ☐3

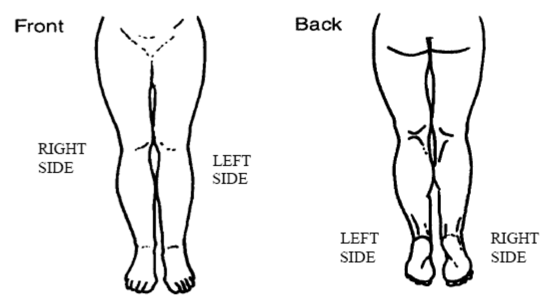
(d) Do you get the pain walking at an ordinary pace on the level?

- Yes ☐1
- No ☐2
- Unable to walk ☐3

(e) What happens to the pain if you stand still?

- Usually continues more than 10 minutes ☐1
- Usually disappears in 10 minutes or less ☐2

(f) Please mark on the diagram below where you get the pain.



OFFICE USE ☐ L ☐ R

Smoking

10.0 Have you **ever** smoked cigarettes regularly (at least 1 a day) ?

Yes ☐1
No ☐2 ➔ **If No**, go to question 10.3 below

10.1 Do you smoke cigarettes at present?

Yes ☐1
No ☐2

(a) **If Yes**, how many cigarettes do you smoke a day at present ? ☐☐

(If hand-rolled, how much tobacco do you use a week ? ☐☐ oz / ☐☐☐ grams)

(b) **If No**, at what age did you give up ? ☐☐ years

10.2 Have you changed your cigarette smoking habits over the last three years ?

No ☐1
Yes, increased ☐2
Yes, decreased ☐3
Yes, given up ☐4

Pipe & Cigar Smoking

10.3 Have you **ever** regularly smoked a pipe ? Yes ☐ No ☐

(a) **If Yes**, do you currently smoke a pipe ? Yes ☐ No ☐

(b) **If Yes,** how much tobacco do you smoke per week? ☐☐ oz / ☐☐☐ grams

10.4 Have you **ever** regularly smoked cigars ?

Yes ☐1
No ☐2 ➔ **If No**, go to question 10.5 below

(a) **If Yes**, do you currently smoke cigars ? Yes☐ No☐

(b) **If Yes,** how many cigars do you smoke per week ? ☐☐

Other exposure to Cigarette smoke

10.5 Does your wife / partner smoke cigarettes ?

Yes ☐1 ➔Number per day ☐☐
Ex -Smoker ☐2
No ☐3
Does not apply ☐4

10.6 For about how many hours each day are you exposed to other people's cigarette smoke ?

(a) at home ☐☐ (hours)

(b) outside the home ☐☐ (hours)

(c) Tick here if rarely exposed to cigarette smoke ☐1

Alcohol

11.0 Would you describe your present alcohol intake as

- Daily/most days ☐1
- Weekends only ☐2
- Occasionally (once or twice a month) ☐3
- Special occasions only ☐4
- None ☐5

One drink is **HALF** a pint of beer /cider, a **SINGLE** whisky, gin, etc. or **ONE GLASS** of wine or sherry

11.1 How much do you usually drink on the days when you drink alcohol ?

- More than 6 drinks ☐1
- 3-6 drinks ☐2
- 1-2 drinks ☐3
- None ☐4

11.2 How many alcoholic drinks do you have during an average week ? ☐☐☐

11.3 What type of drink do you usually take?

- Beers, Lagers ☐1
- Wines, Sherry ☐2
- Spirits ☐3
- Variety of Beers, Wines or Spirits ☐4
- Low alcohol drinks ☐5

		Yes	No	If **Yes**, glasses per week
(a) Do you drink	white wine ?	☐	☐	☐☐
	red wine ?	☐	☐	☐☐

11.4 Is the alcohol which you drink usually taken (tick whichever applies) :-

- before meals ☐1
- with meals ☐1
- after meals ☐1
- separate from meals ☐1

11.5 Have you changed your alcohol intake in the last three years?

- No ☐1
- Yes, increased ☐2
- Yes, cut down ☐3
- Yes, given up ☐4

11.6 If you have **CUT DOWN** or **GIVEN UP**
Was this due to (tick which ever apply):-

- Personal choice ☐1
- Doctor's advice ☐1
- Illness or ill health ☐1
- Health precaution ☐1
- Being on medication ☐1
- Financial reasons ☐1
- Other ☐1

Physical Activity

12.0 Do you make regular journeys every day or most days either walking or cycling ?

- No ☐1
- Walk ☐2
- Cycle ☐3
- Both ☐4

12.1 How long do you spend on all forms of walking in an average week ? ☐☐ hours

12.2 Which of the following best describes your usual walking pace

- Slow ☐1
- Steady average ☐2
- Fairly brisk ☐3
- Fast (at least 4 mph) ☐4

12.3 How long do you spend cycling in an average week ? ☐☐ hours

12.4 Compared with a man who spends four hours on most weekends on activities such as: walking, gardening, household chores, DIY projects, how physically active would you consider yourself?

- Much more active ☐1
- More active ☐2
- Similar ☐3
- Less active ☐4
- Much less active ☐5

12.5 Do you take active physical exercise such as running, swimming, dancing, golf, tennis, squash, jogging, bowls, cycling, hiking, etc.?

- No ☐1
- Occasionally (less than once a month) ☐2
- Frequently (once a month or more) ☐3

(a) If you ticked **frequently** please state type of activities : OFFICE USE

____ ☐☐

(b) How many years have you been engaged in these sort of physical activities ? ☐☐

(c) How many times a **month** (on average) do you take part in these activities (give overall total)?

In winter ☐☐ In summer ☐☐

Your Health Overall

Please indicate which statements best describe your health **TODAY**
(Do not tick **more than one** box in each group)

13.0 General Health:-
- Excellent ☐1
- Good ☐2
- Fair ☐3
- Poor ☐4

13.1 Pain / Discomfort:-
- I have no pain or discomfort ☐1
- I have moderate pain or discomfort ☐2
- I have extreme pain or discomfort ☐3

13.2 Usual Activities (e.g. work, study, housework, family or leisure activities):-
- I have no problems with performing my usual activities ☐1
- I have some problems with performing my usual activities ☐2
- I am unable to perform my usual activities ☐3

13.3 Self Care:-
- I have no problems with washing and dressing ☐1
- I have some problems with washing and dressing myself ☐2
- I am unable to wash or dress myself ☐3

13.4 Mobility:-
- I have no problems in walking about ☐1
- I have some problems in walking about ☐2
- I am confined to a chair / wheelchair ☐3

13.5 Anxiety /Depression:-
- I am not anxious or depressed ☐1
- I am moderately anxious and /or depressed ☐2
- I am extremely anxious and /or depressed ☐3

13.6 Your Memory:- compared to five years ago, is your memory
- improved ☐1
- the same ☐2
- almost as good ☐3
- worse ☐4
- much worse ☐5

13.7 Health Scale

We have drawn a health scale (rather like a thermometer) on which perfect health is 100 and very poor health is 0. Please put a cross (**X**) on the scale to reflect how good or bad your health is today.

Worst Imaginable Health State — Best Imaginable Health State

0 • 10 • 20 • 30 • 40 • 50 • 60 • 70 • 80 • 90 • 100

OFFICE USE

Disability

		Yes	No
14.0	Do you have any long-standing illness, disability or infirmity ?	☐	☐

('long-standing' means anything which has troubled you over a period of time or is likely to do so)

	If Yes,	Yes	No
(a)	Does this illness or disability limit your activities in any way?	☐	☐
(b)	Do you receive a disability allowance ?	☐	☐

14.1 Do you currently have difficulty carrying out any of the following activities on your own as a result of a long term health problem?

		Yes	No	Date started	Cause of problem	OFFICE USE
(a)	Difficulty going up / down stairs	☐	☐	19_____	__________	
(b)	Difficulty bending down / straightening up	☐	☐	19_____	__________	
(c)	Falling or having great difficulty keeping balance	☐	☐	19_____	__________	
(d)	Difficulty walking for a quarter of a mile on the level	☐	☐	19_____	__________	

14.2 Is your present state of health causing problems with any of the following ?

		Yes	No	Cause of problem	OFFICE USE
(a)	Job at work (paid employment)	☐	☐	__________	
(b)	Household chores	☐	☐	__________	
(c)	Social life	☐	☐	__________	
(d)	Interests and hobbies	☐	☐	__________	
(e)	Holidays and outings	☐	☐	__________	
(f)	Family relationships	☐	☐	__________	

Contact with relatives and friends

15.0 How often do you see or speak to :-

	Every week 1	Every month 2	Every few months 3	Every Year 4	Rarely or Never 5	Does not apply 6
Your Children						
Brothers / Sisters						
Friends						
Neighbours						

15.1 Is the amount of contact you have with each of these:-

	Too little 1	About right 2	Too much 3	Does not apply 4
Your Children				
Brothers / Sisters				
Friends				
Neighbours				

Present Circumstances

16.0 Are you at present :- Please give year

single ☐1
married ☐2 ➘
widowed ☐3 ➔ 19 _________
divorced or separated ☐4 ➚
other ☐5

16.1 Are you at present :-

living alone ☐1
living with a partner or spouse ☐2
living with other family member(s) ☐3
living with other people ☐4

16.2 Your accommodation

Are you :- an owner occupier ☐1
renting from the local authority ☐2
renting privately ☐3
other (please give details)_________________ ☐4

OFFICE USE

Present Circumstances continued

		Yes	No
16.3	Do you have a car available for your own use ?	☐	☐
16.4	Do you have a pet ?	☐	☐

OFFICE USE

(a) **If Yes**, what kind of pet do you own :- _______________________

16.5 **Heating**
Please tick the fuels you use to heat your home:-

Natural gas ☐1 Oil ☐1 Wood ☐1
Calor gas ☐1 Coal ☐1
Electricity ☐1 Other ☐1 please specify _____________

OFFICE USE

16.6 Does your home have:-

	Yes	No	
Central heating	☐1	☐2	
Open fires	☐1	☐2	
Double Glazing	☐1	☐2	In part ☐3

16.7 Please tick the fuels you use for cooking:-

Natural gas ☐1
Electricity ☐1
Other ☐1 (Please specify) ___________

OFFICE USE

Work and Retirement

17.0 At present are you :-

retired ☐1 age at retirement ☐☐
employed, full time ☐2
employed, part time ☐3
unemployed, seeking work ☐4
unemployed, not seeking work ☐5

(a) If you are **retired**, did you retire because of:-

normal retiring age ☐1
early retirement, voluntary ☐2
early retirement, compulsory ☐3
retirement, medical grounds ☐4
other reasons ☐5

17.1 Please give details of your current occupation **or** the last job you held before retiring: -

(a) What kind of work do you / did you do_______________________________

(b) Type of business or industry_______________________________________

(c) How many years have you done or did you do that kind of work ? ___________

OFFICE USE

18.0 Are you on any regular medication ?

Yes ☐

No ☐ ➔ If **No**, go to question 18.3 on the next page

For Research Nurse use only	
Actual medications	☐1
Prescription Card (repeat)	☐2
Other list	☐3
No formal documentation	☐4

18.2 Which medications (including tablets, medicines, inhalers, sprays, injections) you are taking ?
Please list medications below:

OFFICE USE

Medication	Dose	Frequency	Reason for taking	BNF CODE	ICD CODE

Aspirin

18.3 Do you take aspirin regularly ? Yes ☐ No ☐ ➔ If **No**, go to question 18.3(b) below

(a) If Yes , year started 19 ☐☐

Dose ☐☐☐ mg

Frequency / week ☐

OFFICE USE

Reason for use ________________________________

On Prescription Yes ☐ No ☐

18.3 (b) If **No**, have you taken aspirin regularly in the past ? Yes ☐ No ☐

If Yes , year started 19 ☐☐

year stopped 19 ☐☐

OFFICE USE

Reason for taking ________________________________

On Prescription Yes ☐ No ☐

Warfarin

18.4 Have you taken warfarin regularly at any time ? Yes ☐ No ☐

If Yes , year started 19 ☐☐

Duration in months ☐☐

OFFICE USE

Reason for taking ________________________________

GTN

18.5 Have you ever taken GTN tablets under the tongue (or spray) to relieve pain in the chest ?

Yes ☐ No ☐

(a) **If Yes,** when was the last time you used them ? ☐☐☐ mths ago

Vitamins & Minerals

18.6 Do you regularly take any vitamin or mineral tablets? Yes ☐ No ☐

(a) **If Yes**, please give details :-

Name of vitamin / mineral	Daily Dose	Year Started
		19________
		19________
		19________
		19________

Blood Cholesterol Test

19.0 Have you ever had your blood cholesterol measured ? Yes ☐ No ☐

(a) **If Yes**, were you told that the result was
High ☐$_1$
Normal ☐$_2$
Low ☐$_3$
Not told ☐$_4$

(b) **If High**, have you been advised to take any particular action ? (please give details)

Diet ☐$_1$
Drugs ☐$_1$

Eating and drinking

20.0 What time did you last have something to eat or drink other than water ?

☐☐ . ☐☐ hours If yesterday please tick ☐$_1$

21.0 **Consent to follow up studies**

An important part of this study is to observe the future health of the people taking part. We are therefore seeking your permission to receive specific information related to heart disease and stroke, particularly from the records held by your general practitioner. All these details would be treated in **absolute confidence** by the Research Team.

Do you agree to us following your future health through your health records ?

☐$_1$ Agreed ☐$_2$ Not Agreed

We will arrange to have your blood sample checked for cholesterol and other factors which are important for heart disease risk. The results of these tests will be sent back to your doctor in the next four to five weeks. If any of the results give cause for concern, you will be asked to make an appointment with your doctor.

Do you agree to us passing the test results to your doctor ?

☐$_1$ Agreed ☐$_2$ Not Agreed

Part of your blood sample will be frozen and kept for special scientific studies of factors affecting heart disease risk, which may help us to understand how to prevent heart disease in the future. Among the factors we may need to study will be the way in which genetic factors affect heart disease risk.

Would you allow us to use your sample in this way ?

☐$_1$ Agreed ☐$_2$ Not Agreed

I agree to allow the Research Team to continue to study my health in accordance with the criteria above. I understand that any details recorded will be treated in complete confidence.

Signed: ____

Date: ____________________

Study Number : | | | | | | | | |

BRITISH REGIONAL HEART STUDY

20 YEAR FOLLOW-UP SURVEY

QUESTIONNAIRE ON PHYSICAL ACTIVITY AND DIET

We should be very grateful if you would complete this questionnaire which asks about your physical activities and diet. Please return it to us with your appointment reply card in the reply paid envelope provided. You may wish to seek help from others with some of the questions on diet, especially if you do not do your own cooking. If you have any difficulties in completing this questionnaire, please phone us on 0171 830 2335 and leave your telephone number so that we can call you back and answer your queries.

All information will be treated as **strictly confidential**.

Thank you for your help.

British Regional Heart Study
Department of Primary Care & Population Sciences
Royal Free Hospital School of Medicine
Rowland Hill Street
London NW3 2PF

PART I : PHYSICAL ACTIVITY

These questions are designed to find out how physically active you are in everyday life, both inside and outside your home. Please try to answer all questions, describing your usual activities **OVER THE LAST YEAR**.

Getting About

1.0 Which of the following forms of transport do you use most often? (tick only one box)

- Car ☐ 1
- Public transport ☐ 2
- Walking or Cycling ☐ 3

Walking

1.1 How many miles do you walk in total in an average week ? __ __ miles / week

1.2 How many journeys of at **least a mile** do you walk each week ? __ __ journeys
(Please write '0' if none)

2.0 **Household Activities**

About how many hours each week do you usually spend on the following household activities ?
(please tick one box for each question)

	None 1	Less than 1 hour a week 2	1 to 3 hours a week 3	3 to 6 hours a week 4	6 to 10 hours a week 5	More than 10 hours a week 6
Light Activities (eg preparing food, cooking, washing up, dusting)						
Moderate Activities (eg cleaning, sweeping, hoovering washing floors, shopping)						
Heavy Activities (eg scrubbing floors, walking with heavy shopping)						

3.0 **Climbing Stairs**

How many flights of stairs do you climb up each day ? (a flight of stairs = 10-15 stairs)

	None 1	1 to 5 flights 2	6 to 10 flights 3	11 to 15 flights 4	More than 15 flights 5
On a weekday					
On a weekend day					

4.0 **Other Activities in the past year**

Please indicate how often you did these activities **during the past year.**
If you didn't do a particular activity at all, simply write 'X' in the first column.

	How many times each month ?		How many months of the year?		Average time on each occasion? Hours	Minutes
	☐☐		☐☐		☐☐	☐☐
Walking on specific journeys (eg to shops, errands)		→		→		
Rambling / Hiking		→		→		
Cycling		→		→		
Light gardening (eg watering the lawn/garden)		→		→		
Moderate gardening (eg planting, cutting grass)		→		→		
Heavy gardening (eg digging, shovelling)		→		→		
DIY (eg home / car maintenance, carpentry)		→		→		
Swimming		→		→		
Jogging		→		→		
Exercises (stretching, bending, keep fit, etc)		→		→		
Dancing		→		→		
Bowling (indoor, lawn, tenpin)		→		→		
Golf		→		→		
Tennis / Badminton		→		→		
Fishing		→		→		
Other exercises (please specify)		→		→		
		→		→		

OFFICE USE ☐ ☐

5.0 Did you do any of these activities vigorously enough to cause sweating, breathlessness or fast heartbeat ? Yes ☐ No ☐

5.1 **If Yes,** for about how many minutes did you do such vigorous activities each week ? __ __ __ (mins)

5.2 Compared with your level of activity three years ago, are you doing

more ☐1
about the same ☐2
less ☐3

5.3 **If less**, please give the reason

____ OFFICE USE ☐

PART II : YOUR DIET

1. Are you on any special diet (eg vegetarian , low fat, diabetic) ? Yes ☐ No ☐ OFFICE USE

If Yes, please give details ____________________________________ ☐

How to fill in the diet questionnaire

The following questions are mostly about how often you **USUALLY** eat different sorts of food each week.

If you usually eat a food **every day**, ring **7** days a week

If you usually eat a food on **three days a week**, ring **3**, and so on

For foods which you eat **less than once a week** :-

Ring **M** if you eat it at **least** once a month

Ring **R** if you eat it **less than** once a month, or if you **never** eat it at all

Please ring **one** answer for each of the foods listed. Remember to circle **R** if you never eat a food.

Example

	Number of days each week							Monthly	Rarely/ Never
Food eaten every day (7 days a week)	7	6	5	4	3	2	1	M	R
Food eaten on three days a week	7	6	5	4	3	2	1	M	R
Food eaten less often than once a week but at least once a month	7	6	5	4	3	2	1	M	R
Food eaten never or less than once a month	7	6	5	4	3	2	1	M	R

<u>Please ring the correct number or letter for every food item</u> <u>(one circle only per line)</u>

		Number of days each week							0 Monthly	8 Rarely/ Never
2.	**Meat**									
(a)	Beef (including minced beef, beef burgers)	7	6	5	4	3	2	1	M	R
(b)	Lamb	7	6	5	4	3	2	1	M	R
(c)	Pork, bacon, ham, salami	7	6	5	4	3	2	1	M	R
(d)	Chicken, turkey, other poultry	7	6	5	4	3	2	1	M	R
(e)	Tinned meat (all types, corned beef, etc)	7	6	5	4	3	2	1	M	R
(f)	Pork Sausages	7	6	5	4	3	2	1	M	R
(g)	Beef Sausages	7	6	5	4	3	2	1	M	R
(h)	Meat Pie, Pasties	7	6	5	4	3	2	1	M	R
(i)	Liver, kidney, heart	7	6	5	4	3	2	1	M	R
3.	**Fish**									
(a)	White fish (cod, haddock, hake, plaice, fish fingers, etc)	7	6	5	4	3	2	1	M	R
(b)	Kippers, herrings, pilchards, tuna, sardines, salmon, mackerel (including tinned)	7	6	5	4	3	2	1	M	R
(c)	Shellfish	7	6	5	4	3	2	1	M	R
4.	**Vegetables (fresh, tinned, dried, frozen)**									
(a)	Potatoes: boiled, baked, mashed	7	6	5	4	3	2	1	M	R
(b)	Potatoes									
	(i) chips or fried (from shop)	7	6	5	4	3	2	1	M	R
	(ii) chips, fried or (cooked at home)	7	6	5	4	3	2	1	M	R
	(iii) roast potatoes	7	6	5	4	3	2	1	M	R
(c)	Green vegetables, salads	7	6	5	4	3	2	1	M	R
(d)	Carrots	7	6	5	4	3	2	1	M	R
(e)	Parsnips, swedes, turnips, beetroot, and other root vegetables	7	6	5	4	3	2	1	M	R
(f)	Baked or butter beans, lentils, peas, chickpeas, sweetcorn	7	6	5	4	3	2	1	M	R
(g)	Onions (cooked, raw, pickled)	7	6	5	4	3	2	1	M	R
(h)	Garlic	7	6	5	4	3	2	1	M	R
(i)	Spaghetti and other pasta	7	6	5	4	3	2	1	M	R
(j)	Rice (all types except pudding rice)	7	6	5	4	3	2	1	M	R
(k)	Tomatoes (fresh, tinned, pureed)	7	6	5	4	3	2	1	M	R
	How often do you eat fresh vegetables in :									
(l)	**summer**	7	6	5	4	3	2	1	M	R
(m)	**winter**	7	6	5	4	3	2	1	M	R

Please remember to circle ® if you never eat a food

		Number of days each week							0 Monthly	8 Rarely/ Never
5.	**Fresh fruit**									
	How often do you eat fresh fruit in :									
(a)	**summer**	7	6	5	4	3	2	1	M	R
(b)	**winter**	7	6	5	4	3	2	1	M	R

(c) Number of apples eaten a week ___ ___

(d) Number of pears eaten a week ___ ___

(e) Number of oranges or grapefruit eaten a week ___ ___

(f) Number of bananas eaten a week ___ ___

(g) Number of other fruits eaten a week (please give name and quantity)

Name	Quantity	Name	Quantity
....	....	....	....
....	....	....	....
....	....	....	....

OFFICE USE

		Number of days each week							0 Monthly	8 Rarely/ Never
6.	**Cheese**									
	Full- fat cheese (eg Cheddar, Leicester, Stilton, Brie, soft cheeses)	7	6	5	4	3	2	1	M	R
	Low-fat cheese (eg Edam, Cottage cheese, reduced fat cheeses)	7	6	5	4	3	2	1	M	R
7.	**Bread**									
(a)	White bread	7	6	5	4	3	2	1	M	R
(b)	Brown bread	7	6	5	4	3	2	1	M	R
(c)	Wholemeal	7	6	5	4	3	2	1	M	R
(d)	Bread rolls	7	6	5	4	3	2	1	M	R
(e)	Crispbread (Ryvita, cream crackers, etc)	7	6	5	4	3	2	1	M	R

please give name of crispbread etc...

(f) Further details about your bread

	How many slices or rolls a day ?	Are the slices thick, medium or thin? Circle your answer.		
(i) White Bread	___ ___	thick $_1$	medium $_2$	thin $_3$
(ii) Brown Bread	___ ___	thick	medium	thin
(iii) Wholemeal Bread	___ ___	thick	medium	thin
(iv) Bread Rolls	___ ___	large	medium	small

Please remember to circle ® if you never eat a food

8. **Breakfast Cereals**

		Number of days each week							0 Monthly	8 Rarely/ Never
(a)	Grapenuts, Porridge, Ready Brek, Special K, Sugar Puffs, Rice Crispies	7	6	5	4	3	2	1	M	R
(b)	Cornflakes, Muesli, Shredded Wheat, Sultana Bran, Weetabix	7	6	5	4	3	2	1	M	R
(c)	Bran Flakes, Puffed wheat	7	6	5	4	3	2	1	M	R
(d)	All Bran, Wheat Bran	7	6	5	4	3	2	1	M	R
(e)	Another Cereal	7	6	5	4	3	2	1	M	R
	please give name									
9.	**Biscuits, puddings and sweets**									
(a)	Digestive biscuits, plain biscuits	7	6	5	4	3	2	1	M	R
(b)	Sweet biscuits, sponge cakes, scones, buns	7	6	5	4	3	2	1	M	R
(c)	Ice cream, sweet yoghurts, trifle	7	6	5	4	3	2	1	M	R
(d)	Fruit cake, fruit bread, plum pudding	7	6	5	4	3	2	1	M	R
(e)	Fruit tart, jam tart, fruit crumble	7	6	5	4	3	2	1	M	R
(f)	Milk puddings (rice, tapioca)	7	6	5	4	3	2	1	M	R
(g)	Tinned fruit, jellies	7	6	5	4	3	2	1	M	R
(h)	Sweet sauces (chocolate, custard)	7	6	5	4	3	2	1	M	R
(i)	Chocolate, chocolate bars, sweets (all types)	7	6	5	4	3	2	1	M	R
10.	**Eggs**									
(a)	Eggs (boiled, poached, fried, scrambled)	7	6	5	4	3	2	1	M	R
(b)	Eggs in baked dishes (eg flans, quiches, soufflés, egg custard, etc)	7	6	5	4	3	2	1	M	R
11.	**Other foods**									
(a)	Soups (all kinds, home-made, tinned, packet)	7	6	5	4	3	2	1	M	R
(b)	Nuts, nut butter (eg salted or unsalted peanuts)	7	6	5	4	3	2	1	M	R
(c)	Savoury snacks (eg potato crisps, corn chips, crackers)	7	6	5	4	3	2	1	M	R
(d)	Chutney, brown sauce, tomato sauce	7	6	5	4	3	2	1	M	R
(e)	Sweet spreads (eg jam, honey, marmalade, chocolate spread)	7	6	5	4	3	2	1	M	R
12.	**Drinks and Juices (non-alcoholic)**									
(a)	Natural fruit juices (including tomato juice)	7	6	5	4	3	2	1	M	R
(b)	Fizzy drinks and Non-diet squashes	7	6	5	4	3	2	1	M	R
(c)	**Low calorie** (diet) squashes and fizzy drinks	7	6	5	4	3	2	1	M	R

Please remember to circle ® if you never eat a food

13. **Milk**

(a) Roughly how much milk do you drink a day in tea, coffee, milky drinks or cereals ? (Tick only one box)

1 ☐ none at all
2 ☐ half pint or less
3 ☐ between half and one pint
4 ☐ more than one pint

(b) What kind of milk do you usually use? (Tick only one box)

1 ☐ full fat milk, fresh or dried
2 ☐ semi-skimmed milk, fresh or dried
3 ☐ fully skimmed milk, fresh or dried
4 ☐ other kinds of milk, eg condensed, evaporated

14. **Fats**

(a) What do you usually spread on bread ? OFFICE USE

1 ☐ butter Give brand name ....
1 ☐ full-fat soft margarine Give brand name .... ☐
1 ☐ low-fat soft margarine Give brand name ....
1 ☐ hard margarine Give brand name .... ☐

(b) How do you normally spread the fat ?

1 ☐ thinly 2 ☐ average 3 ☐ thickly

(c) How often do you eat home-fried food (including chips), cooked with :-

	Number of days each week							0 Monthly	8 Rarely/ Never	
Lard, dripping, solid vegetable oil	7	6	5	4	3	2	1	M	R	
Give brand name and type										☐
Liquid vegetable oil	7	6	5	4	3	2	1	M	R	
Give brand name and type										☐

15. **Salt**

(a) How much salt is added to your food, on cooking ?
1 ☐ a lot 2 ☐ a little 3 ☐ none

(b) How much salt is added to your food on your plate ?
1 ☐ a lot 2 ☐ a little 3 ☐ none

Please remember to circle ® if you never eat a food

16. **Your household**

How many people normally eat in your household ?

Number of adults (including yourself) ___ ___ Number of children 1 to 4 years old ___ ___

Number of children 5 to 16 years old ___ ___ Number of babies under 1 year old ___ ___

17. How much of the following foods does **your household** use on average **each week** (including cooking and baking)? If you live on your own, please give the amounts which you yourself eat a week.

	If rarely or never used tick here			
Butter	☐1	___ ___ lbs	___ ___ozs	or ___ ___ ___ grams
Margarine (all types)	☐1	___ ___ lbs	___ ___ozs	or ___ ___ ___ grams
Lard and solid vegetable oil	☐1	___ ___ lbs	___ ___ozs	or ___ ___ ___ grams
Liquid vegetable oil (eg Sunflower, Corn, Groundnut oil)	☐1		___ ___ozs	or ___ ___ ___ ml
Olive Oil	☐1		___ ___ozs	or ___ ___ ___ ml
Cream	☐1		___ ___ozs	or ___ ___ ___ ml
Full- fat cheese (eg Cheddar, Leicester, Stilton, Brie, and soft cheeses)	☐1	___ ___ lbs	___ ___ozs	or ___ ___ ___ grams
Low-fat cheese (eg reduced fat cheddar, reduced fat soft cheeses, Edam)	☐1	___ ___ lbs	___ ___ozs	or ___ ___ ___ grams
Sugar	☐1	___ ___ lbs	___ ___ozs	or ___ ___ ___ grams

18. **Hot drinks**

Coffee

(a) How many cups of **coffee** do you have a day ? ___ ___ cups a day

Is this ☐1 ground coffee ☐2 instant coffee

Is it decaffeinated ? ☐1 Yes ☐2 No

(b) How many teaspoons of sugar do you take in each cup ? ___ teaspoons
(Do not count artificial sweeteners)

Tea

(c) How many cups of **tea** do you have a day ? ___ ___ cups a day

(d) How many teaspoons of sugar do you take in each cup ? ___ teaspoons
(Do not count artificial sweeteners)

Other Hot Drinks

(e) How many cups of other hot drinks (eg drinking hot chocolate, malted milk, Horlicks) do you have a day ? ___ ___ cups a day

19. **Alcoholic drinks**

		Yes	No	Seldom
(a)	Have you ever consumed alcoholic drinks ?	☐1	☐2	
(b)	Do you take alcoholic drinks at present ?	☐1	☐2	☐3

(c) Think back carefully over the last seven days.
Please write the number of alcoholic drinks you have consumed on each day during the past week. It may help if you try to remember where you were and who you were with on each day.

For each day, write in how much you have drunk:

(i) the **number of pints** of non-alcoholic beer, lager, etc
(ii) the **number of pints** of low-alcohol beer, lager, etc
(iii) the **number of pints** of beer, lager, shandy, cider, stout, etc
(iv) the **number of single glasses** of whisky, vodka, gin, rum, etc
(v) the **number of single glasses** of wine, sherry, martini, port, etc

	(i) Pints of Non-alcoholic Beer ☐☐	(ii) Pints of Low-alcohol Beer ☐☐	(iii) Pints of Beer, Lager, Shandy ☐☐	(iv) Single glasses of Spirits ☐☐	(v) Single glasses of Wine ☐☐
Monday					
Tuesday					
Wednesday					
Thursday					
Friday					
Saturday					
Sunday					

		Yes	No
(d)	Would you say last week was fairly typical of what you usually have to drink in one week ?	☐1	☐2

		More	Less
(e)	If last week was not typical, would you normally drink more or less in a week ?	☐1	☐2

20. **Birth Weight**

Recent research has suggested that circumstances around the time of birth, and particularly birthweight, may influence the heart and circulation many years later.

If you can tell us about your birthweight and the birthweight (s) of your children (asking other family members if necessary) this would be very helpful :-

(a) **Your birth weight:** ___ ___ lb ___ ___ oz Not known ☐ 1

(b) **The birthweight of your children:-**

			Boy	Girl		
First Child	___ ___ lb	___ ___ oz	☐ 1	☐ 2	Not known Does not apply	☐ 1 ☐ 1
Second Child	___ ___ lb	___ ___ oz	☐ 1	☐ 2		
Etc	___ ___ lb	___ ___ oz	☐ 1	☐ 2		
	___ ___ lb	___ ___ oz	☐ 1	☐ 2		
	___ ___ lb	___ ___ oz	☐ 1	☐ 2		

Thank you for your help with this questionnaire.

Please check that you have answered all questions and
return the questionnaire to us in the envelope provided.

No stamp is required.

For comments:

Study Number: ☐☐☐☐☐☐☐☐

BRITISH REGIONAL HEART STUDY

2003 QUESTIONNAIRE

Thank you very much for taking the time to complete this questionnaire, which will bring us up to date with your present state of health. All the information will be treated as **strictly confidential** and will only be seen by the Research Team.

Most questions can be answered by ticking the correct box ☑

Please check that you have answered as many questions as you can and return it to us in the envelope provided – you do not need to use a stamp.

If you have any trouble answering the questions, or would like a large-print copy, please phone us on 020 7830 2335 and give us your telephone number. We will then call you back to answer your query.

THANK YOU FOR YOUR HELP

Department of Primary Care & Population Sciences
Royal Free & University College Medical School,
Rowland Hill Street, London NW3 2PF

1.0 Date of birth ________ day ________ month 19________ year

(This information is necessary for us to ensure that you are the correct recipient)

Conditions affecting the heart or circulation

2.0 Have you **ever** been told by a doctor that you have or have had any of the following conditions?

If you tick **Yes**, please give the year of **last occurrence**

		Yes	No	Year of last occurrence
(a)	Heart attack (coronary thrombosis or myocardial infarction)	☐	☐	________
(b)	Heart failure	☐	☐	________
(c)	Angina	☐	☐	________
(d)	Other heart trouble	☐	☐	________
(e)	High blood pressure	☐	☐	________
(f)	High blood cholesterol	☐	☐	________
(g)	Aortic Aneurysm	☐	☐	________
(h)	Narrowing or hardening of the leg arteries (including claudication)	☐	☐	________
(i)	Deep Vein Thrombosis (clot in the deep leg vein)	☐	☐	________
(j)	Pulmonary Embolism (clot on the lung)	☐	☐	________

Stroke	Yes	No	Year of last occurrence
3.0 Have you **ever** been told by a doctor that you have had a stroke?	☐	☐	________
If Yes,			
3.1 Did the symptoms last for more than 24 hours?	☐	☐	
3.2 Have you made a complete recovery from your stroke?	☐	☐	
3.3 Following your stroke, do you still need any help in carrying out everyday activities?	☐	☐	

Investigations and special treatment for conditions affecting the heart and circulation

4.0 Have you **ever** had one of the following?

		Yes	No	Year of last occurrence
4.1	A referral to a heart specialist	☐	☐	_______
4.2	A referral to a chest pain clinic	☐	☐	_______
4.3	An exercise ECG ("stress" or "treadmill") test	☐	☐	_______
4.4	Angiogram or X-ray of coronary arteries (using a dye)	☐	☐	_______
4.5	Angioplasty (balloon treatment of coronary artery for angina)	☐	☐	_______
4.6	Coronary artery bypass graft operation ("heart bypass" or "CABG")	☐	☐	_______
4.7	Other tests, investigations or operations on the heart, arteries or veins?	☐	☐	_______

If **Yes**, please give details:

____ OFFICE USE ☐

Diabetes

		Yes	No	
5.0	Have you **ever** been told by a doctor that you have or have had diabetes?	☐	☐	

If **Yes**,

5.1 In what year was it first diagnosed? ________ (Year)

			Yes	No	
5.2	Do you have any complications of diabetes affecting………..	your feet?	☐	☐	
		your nerves?	☐	☐	
		your kidneys?	☐	☐	
		your eyes?	☐	☐	
5.3	Have your eyes been checked for signs of diabetes? (Please give year of last check)		☐	☐	_______ (Year)

Cancer

6.0 Have you **ever** been told by a doctor that you have or have had cancer? Yes ☐ No ☐

If **Yes**, please give:

(a) Year first diagnosed __________ (b) Cancer Site ____________________ OFFICE USE ☐☐☐☐

Arthritis

7.0 Have you **ever** been told by a doctor that you have or have had arthritis? Yes ☐ No ☐

If **Yes**,

7.1 Type of arthritis (if known), (eg. osteoarthritis, rheumatoid arthritis, other): OFFICE USE ☐

____

7.2 Year first diagnosed ________

7.3 Joint(s) affected:
please tick the relevant box(es)

Knees ☐
Hips ☐
Feet ☐
Hands and/or wrists ☐
Other (please specify) ____________________ OFFICE USE ☐

Other Medical Conditions

8.0 Have you **ever** been told by a doctor that you have or have had any of the following conditions?

If **Yes**, please give the year when **first** diagnosed, if possible

		Yes	No	Year			Yes	No	Year
(a)	Asthma	☐	☐	_____	(b)	Bronchitis	☐	☐	_____
(c)	Cataract	☐	☐	_____	(d)	Depression	☐	☐	_____
(e)	Emphysema	☐	☐	_____	(f)	Gall bladder disease	☐	☐	_____
(g)	Gastric, peptic or duodenal ulcer	☐	☐	_____	(h)	Glaucoma	☐	☐	_____
(i)	Gout	☐	☐	_____	(j)	Osteoporosis	☐	☐	_____
(k)	Parkinson's disease	☐	☐	_____	(l)	Pneumonia	☐	☐	_____
(m)	Prostate trouble	☐	☐	_____					

(n) Other conditions, please give details: OFFICE USE

______________________________ _______ (year) ☐☐☐☐

______________________________ _______ (year) ☐☐☐☐

Joint pain, swelling or stiffness

9.0 During **the past year** have you had pain, aching, stiffness or swelling on most days for at least one month, in your…

	Yes	No	
(a) Hands or wrists	☐	☐	
(b) Knees	☐	☐	
(c) Hips	☐	☐	
(d) Feet	☐	☐	OFFICE USE
(e) Other joint	☐	☐	(please specify)______________________ ☐

Lower back pain

	Yes	No
10.0 Have you **ever** had pain in your lower back on most days for at least one month?	☐	☐
10.1 If **Yes**, have you had this in the last year?	☐	☐

Fractures and falls

	Yes	No	Please give year
11.0 Have you **ever** fractured your hip?	☐	☐	___________
11.1 Have you **ever** fractured your wrist?	☐	☐	___________
11.2 Have you had a fall in the last 12 months?	☐	☐	

If **Yes**,

(a) how many times? ☐☐ times

(b) Did you receive medical attention for any of these falls? ☐ ☐

Chest pain

	Yes	No
12.0 Do you **ever** have any pain or discomfort in your chest?	☐	☐

If **Yes**,

	Yes	No	Unable to walk on level
(a) When you walk at an ordinary pace on the level, does this produce the pain?	☐	☐	☐

	Yes	No	Unable to walk uphill
(b) When you walk uphill or hurry, does this produce the pain?	☐	☐	☐

Breathlessness

	Yes	No
13.0 Do you **ever** get short of breath walking with other people of your own age on level ground?	☐	☐
13.1 On walking up hill or stairs do you get more breathless than people of your own age?	☐	☐
13.2 Do you **ever** have to stop walking because of breathlessness?	☐	☐
13.3 In the **past twelve months** have you at any time been awoken at night by an attack of shortness of breath?	☐	☐

Weight

14.0 What is your present weight (indoor clothes, without shoes)?

________Stones _______ Pounds / or _______ Kilograms

(If you have no scales and have made an estimate please tick here ☐)

	Yes	No
14.1 Have you tried to lose weight in the last four years?	☐	☐
If **Yes**, did you:		
Change your diet?	☐	☐
Take more exercise?	☐	☐

Other (please give details) ________________________ OFFICE USE ☐

	Yes	No
14.2 Have you been advised by a doctor or other health professional to lose weight in the last four years?	☐	☐

14.3 Has your weight changed in the last four years?

Not changed	☐1
Increased	☐2
Decreased	☐3
Both increased and decreased)	☐4
Don't know	☐5

	Yes	No
14.4 **If your weight has changed** -was this change intentional?	☐	☐

-was it the result of:-

Personal choice	☐1
Medical advice	☐1
Illness or ill health	☐1

14.5 Do you consider your present weight to be:-

about right	☐1
too high	☐2
too low	☐3

Disability

	Yes	No
15.0 Do you have any long-standing illness, disability or infirmity?	☐	☐

("long-standing" means anything which has troubled you over a period of time or is likely to do so)

If **Yes**,

	Yes	No
(a) Does this illness or disability limit your activities in any way?	☐	☐
(b) Do you receive a disability allowance?	☐	☐

15.1 Do you currently have difficulty carrying out any of the following activities on your own as a result of a **long term** health problem?

		Yes	No
(a)	Going up or down stairs	☐	☐
(b)	Bending down	☐	☐
(c)	Straightening up	☐	☐
(d)	Keeping your balance	☐	☐
(e)	Going out of the house?	☐	☐
(f)	Walking 400 yards	☐	☐

15.2 Is your present state of health causing problems with any of the following:-

		Yes	No
(a)	Job at work (paid employment)	☐	☐
(b)	Household chores	☐	☐
(c)	Social life	☐	☐
(d)	Sex life	☐	☐
(e)	Interests and hobbies	☐	☐
(f)	Holidays and outings	☐	☐

Eyesight

	Yes	No
16.0 Using glasses or corrective lenses if needed, can you see well enough to recognise a friend at a distance of 12 feet/ four yards (across a road)?	☐	☐
If **No**, can you see well enough to recognise a friend at a distance of one yard?	☐	☐

Hearing

	Yes	No
17.0 Do you use a hearing aid?	☐	☐
17.1 Using a hearing aid if needed, is your hearing good enough to follow a TV programme at a volume others find acceptable?	☐	☐
If **No**, can you follow a TV programme with the volume turned up?	☐	☐

Your Health Overall

Please indicate which statements best describe your health **TODAY.** (Please tick **only one box**)

18.0	General Health:-	Excellent	☐1
		Good	☐2
		Fair	☐3
		Poor	☐4
18.1	Pain/Discomfort:-	I have no pain or discomfort	☐1
		I have moderate pain or discomfort	☐2
		I have extreme pain or discomfort	☐3
18.2	Mobility:-	I have no problems in walking about	☐1
		I have some problems in walking about	☐2
		I am confined to a chair/wheelchair	☐3
18.3	Anxiety/Depression:-	I am not anxious or depressed	☐1
		I am moderately anxious and/or depressed	☐2
		I am extremely anxious and/or depressed	☐3

Sleep:-

18.4 On average, how many hours' sleep do you have each night? ☐☐ hours

18.5 On average, how much sleep (if any) do you have during the daytime? ☐☐ hours

18.6	Do you snore while asleep?	Yes, regularly	☐1
		Yes, occasionally	☐2
		No, never	☐3
		Don't know	☐4

18.7 **Health Scale**

We have drawn a health scale (rather like a thermometer) on which perfect health is 100 and very poor health is 0. Please put a cross (**X**) on the scale to reflect how good or bad your health is today.

Worst Imaginable Health State — Best Imaginable Health State

0 • 10 • 20 • 30 • 40 • 50 • 60 • 70 • 80 • 90 • 100

OFFICE USE ☐☐☐

Physical activity

19.0 Do you make regular journeys every day or most days either walking or cycling?

No	☐1
Walk	☐2
Cycle	☐3
Both	☐4

(a) How many hours do you normally spend walking (e.g. on errands or for leisure) in an average week? ☐☐ hours

19.1 Which of the following best describes your usual walking pace?

Slow	☐1
Steady average	☐2
Fast	☐3

19.2 How long do you spend cycling in an average week? ☐☐ hours

19.3 Compared with a man who spends four hours on most weekends on activities such as: walking, gardening, household chores, DIY projects, how physically active would you consider yourself?

Much more active	☐1
More active	☐2
Similar	☐3
Less active	☐4
Much less active	☐5

19.4 Do you take active sporting physical exercise such as running, swimming, dancing, golf, tennis, squash, jogging, bowls, cycling, hiking, etc.?

No	☐1
Occasionally (less than once a month)	☐2
Frequently (once a month or more)	☐3

(a) If you ticked **frequently** please state type of activities:

OFFICE USE ☐☐

____

(b) How many times a **month** (on average) do you take part in these activities? (give overall total)

In winter ☐☐ times

In summer ☐☐ times

19.5 Do you engage in exercises to increase muscle strength and endurance such as lifting weights, doing push-ups, using exercise machines?

Yes	☐
No	☐

If Yes, on average how many hours per week do you engage in these exercises? ☐☐ hours per week

Cigarette smoking

	Yes	No
20.0 Do you smoke cigarettes at present?	☐	☐

If Yes, please answer the following questions:

20.1 How many cigarettes do you smoke a day at present? ☐☐

20.2 If hand-rolled, how much tobacco do you use a week? ☐☐ oz / ☐☐☐ grams

	Yes	No
20.3 Do you want to give up smoking?	☐	☐
20.4 Have you tried to stop smoking?	☐	☐

20.5 Have you been offered any of the following to help you stop smoking?

	Yes	No
(a) Advice from a health professional (e.g. doctor or nurse)	☐	☐
(b) Referral to a stop-smoking clinic	☐	☐
(c) Nicotine replacement treatment (including sprays, patches etc)	☐	☐
(d) Zyban tablets	☐	☐

(e) Other treatment (please specify) ______________________

OFFICE USE ☐

21.0 Have you changed your cigarette smoking habits during the past four years?

No	☐1
Yes, increased	☐2
Yes, cut down	☐3
Yes, given up	☐4

21.1 **If you have given up smoking** in the last four years, were any of these factors important?

	Yes	No
(a) Advice from a health professional (e.g. doctor or nurse)	☐	☐
(b) Referral to a stop-smoking clinic	☐	☐
(c) Nicotine replacement treatment (including sprays, patches etc)	☐	☐
(d) Zyban tablets	☐	☐
(e) Illness or ill-health	☐	☐
(f) Cost of cigarettes	☐	☐

(g) Other factors (please specify) ______________________

OFFICE USE ☐

Pipe and cigar smoking

	Yes	No
22.0 Do you currently smoke a pipe?	☐	☐
22.1 Do you currently smoke cigars?	☐	☐

Alcohol intake

23.0 Would you describe your present alcohol intake as

- Daily/most days ☐1
- Weekends only ☐2
- Occasionally (once or twice a month) ☐3
- Special occasions only ☐4
- None ☐5

One drink is **HALF** a pint of beer/lager/cider, a **SINGLE** whisky, gin, etc. or **ONE GLASS** of wine or sherry

23.1 How much do you usually drink on the days when you drink alcohol?

- More than 6 drinks ☐1
- 5-6 drinks ☐2
- 3-4 drinks ☐3
- 1-2 drinks ☐4

23.2 How many alcoholic drinks do you have during an average week? ☐☐

23.3 What type of drink do you usually take?

- Beers, Lagers ☐1
- Wines, Sherry ☐2
- Spirits ☐3
- Combination of Beers, Wines or Spirits ☐4
- Low alcohol drinks ☐5

23.4 What is your usual consumption of these alcoholic beverages? Please tick boxes

	PER WEEK					
Type of drink	Never/ hardly ever	Less than 1	1-6	7-13	14-20	21+
Beer or lager (pints)	☐	☐	☐	☐	☐	☐
Red wine (single glass)	☐	☐	☐	☐	☐	☐
White wine (single glass)	☐	☐	☐	☐	☐	☐
Spirits (1 drink/shot)	☐	☐	☐	☐	☐	☐

23.5 Is the alcohol which you drink usually taken (tick whichever applies):-

- before meals ☐1
- with meals ☐1
- after meals ☐1
- separate from meals ☐1

Alcohol Intake continued

23.6 Have you changed your alcohol intake in the last four years?

- No ☐1
- Yes, increased ☐2
- Yes, cut down ☐3
- Yes, given up ☐4

23.7 If you have **CUT DOWN** or **GIVEN UP**, was this due to (tick whichever applies):-

Personal choice	☐1	Being on medication	☐1
Doctor's advice	☐1	Financial reasons	☐1
Illness or ill health	☐1	Other	☐1
Health precaution	☐1		

Preventive Health Care

24.0 In what **year** did you last consult a GP about a health problem? ___________

24.1 Have you ever had any of the following

	Yes	No	If **Yes**, year of most recent
(a) Blood pressure check	☐	☐	_______
(b) Blood cholesterol check	☐	☐	_______
(c) Flu vaccination	☐	☐	_______
(d) Dental check	☐	☐	_______
(e) Foot care from a chiropodist	☐	☐	_______

24.2 Approximately, how many times in the **last twelve months** have you consulted your GP about a health problem? ☐☐ times

Questions about medicines

	Yes	No	
25.0 Do you take any regular medication?	☐	☐	
If **Yes**, do you take any of the following medicines regularly?			Year started
(a) Aspirin tablets	☐	☐	_______
(b) Treatment for any form of heart disease	☐	☐	_______
(c) Treatment to lower blood pressure	☐	☐	_______
(d) Treatment to lower blood cholesterol	☐	☐	_______

25.1 If you are on treatment to lower your blood cholesterol:-

OFFICE USE

(a) Please give the name of this medicine: ______________________________ ☐☐

(b) Please give the amount you take each day: ______________________________ ☐☐☐
(details of the amount in each tablet should be on the bottle)

Details of ALL medicines

26.0 Please write down details of all medicines – including tablets, injections, inhalers, eye-drops etc – which you take regularly. Please also include any medications which you buy for yourself.

	Name of medicine	Reason for taking (if you know)	Date started	Is this prescribed? Yes	No	OFFICE USE
1				☐	☐	
2				☐	☐	
3				☐	☐	
4				☐	☐	
5				☐	☐	
6				☐	☐	
7				☐	☐	
8				☐	☐	
9				☐	☐	
10				☐	☐	

Present circumstances

27.0 Are you at present:-

- single ☐1
- married ☐2
- widowed ☐3
- divorced or separated ☐4
- other ☐5

(a) If you are widowed or divorced/separated, please give the year when this occurred:- ________

27.1 Are you at present:-

- living alone ☐1
- living with a partner or spouse ☐2
- living with other family member(s) ☐3
- living with other people ☐4

27.2 Your accommodation
Are you:-

- an owner occupier ☐1
- renting from the local authority ☐2
- renting privately ☐3
- living in a residential home ☐4
- living in a nursing home ☐5
- other (please give details) ☐6

____

27.3 During the winter, is your accommodation usually:

- Very warm ☐1
- Warm ☐2
- Medium ☐3
- Cold ☐4
- Very cold ☐5

		Yes	No
27.4	Do you have a car available for your own use?	☐	☐
27.5	Are you currently in full-time paid employment?	☐	☐
27.6	Do you have private medical insurance?	☐	☐
27.7	Have you ever had private medical treatment?	☐	☐

Activities of daily living

The following questions will help us to understand difficulties people may have with various everyday activities

28.0 What is the furthest you can walk on your own without stopping and without discomfort?

- 200 metres or more ☐1
- More than a few steps but less than 200 metres ☐2
- Only a few steps ☐3

28.1 Can you walk up and down a flight of 12 stairs without resting?

- Yes ☐1
- Only if I hold on and take a rest ☐2
- Not at all ☐3

28.2 Can you, when standing, bend down and pick up a shoe from the floor?

- Yes ☐
- No ☐

29.0 Please indicate if you have difficulty doing any of the following activities:	No difficulty	Some difficulty	Unable to do or need help
Reaching or extending your arms above shoulder level	☐	☐	☐
Pulling or pushing large objects like a living room chair	☐	☐	☐
Walking across a room	☐	☐	☐
Getting in and out of bed on your own?	☐	☐	☐
Getting in and out of a chair on your own?	☐	☐	☐
Dressing and undressing yourself on your own?	☐	☐	☐
Bathing or showering?	☐	☐	☐
Feeding yourself, including cutting food?	☐	☐	☐
Getting to and using the toilet on your own?	☐	☐	☐
Lifting and carrying something as heavy as 10 lbs, for example a bag of groceries	☐	☐	☐
Shopping for personal items such as toilet items or medicine by yourself	☐	☐	☐
Doing light housework such as washing up	☐	☐	☐
Preparing your own meals by yourself	☐	☐	☐
Using the telephone by yourself	☐	☐	☐
Taking medications by yourself	☐	☐	☐
Managing money (e.g. paying bills etc)	☐	☐	☐
Using public transport on your own	☐	☐	☐
Driving a car on your own	☐	☐	☐

Time spent on various activities

30.0 Approximately how many **hours each week** (if any) do you spend:

			Tick box if you never do
Looking after wife/partner?	☐☐	hours per week	☐
Looking after other adult family member or friend?	☐☐		☐
Looking after grandchildren?	☐☐		☐
In paid work?	☐☐	hours per week	☐
In voluntary work?	☐☐		☐
On housework?	☐☐	hours per week	☐
On gardening?	☐☐		☐
In a pub or club?	☐☐	hours per week	☐
Attending religious services?	☐☐		☐
Playing cards, games, or bingo?	☐☐	hours per week	☐
Visiting the cinema/restaurants/sporting events?	☐☐		☐
Watching television/videos?	☐☐	hours per week	☐
Reading?	☐☐		☐
Attending class or course of study?	☐☐		☐

31.0 Do you go on day or overnight trips…

- Never ☐1
- Sometimes ☐2
- Often ☐3

	Yes	No
31.1 Have you been on holiday in the last year?	☐	☐

Thank you very much for completing the questionnaire.

Please return it to us, along with the blue consent form, in the envelope provided.

No stamp is needed.

References

(1) Ness A, Davey Smith G. The epidemiology of ischaemic heart disease. Oxford Textbook of Medicine. Fourth ed. Oxford: Oxford University Press; 2003. Volume 2, 15.4.1.2.

(2) Henderson A. Coronary heart disease: Overview. Lancet 1996; 348:S1-S4.

(3) Shaper AG. Coronary Heart Disease: Risks and Reasons. London: Current Medical Literature Ltd; 1988.

(4) Tyroler HA. Coronary Heart Disease Epidemiology in the 21st Century. Epidemiol Rev 2000; 22:7-13.

(5) Hampton JR. Chest Pain. Oxford Textbook of Medicine. Fourth ed. Oxford: Oxford University Press; 2003. Volume 2, 15.2.1.

(6) Allender S, Peto V, Scarborough P, Boxer A, Rayner M. Coronary heart disease statistics 2007. British Heart Foundation: London; 2007.

(7) Petersen S, Peto V, Scarborough P, Rayner M. Coronary heart disease statistics 2005. London: BHF; 2005.

(8) Allender S, Peto V, Scarborough P, Boxer A, Rayner M. Coronary heart disease statistics 2008. British Heart Foundation: London; 2008.

(9) Jousilahti P, Vartiainen E, Tuomilehto J, Puska P. Sex, Age, Cardiovascular Risk Factors, and Coronary Heart Disease : A Prospective Follow-Up Study of 14 786 Middle-Aged Men and Women in Finland. Circulation 1999; 99:1165-1172.

(10) Emberson JR, Whincup PH, Morris RW, Walker M. Re-assessing the contribution of serum total cholesterol, blood pressure and cigarette smoking to the aetiology of coronary heart disease: impact of regression dilution bias. Eur Heart J 2003; 24:1719-1726.

(11) Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet 2004; 364:937-952.

(12) Lowe GDO. Circulating inflammatory markers and risks of cardiovascular and non-cardiovascular disease. J Thromb Haemost 2005; 3:1618-1627.

(13) Whincup PH, Danesh J, Walker M, Lennon L, Thomson A, Appleby P et al. von Willebrand factor and coronary heart disease. Prospective study and meta-analysis. Eur Heart J 2002; 23:1764-1770.

(14) Brunner EJ, Marmot MG, Nanchahal K, Shipley MJ, Stansfeld SA, Juneja M et al. Social inequality in coronary risk: central obesity and the metabolic syndrome. Evidence from the Whitehall II study. Diabetologia 1997; V40:1341-1349.

(15) Gami AS, Witt BJ, Howard DE, Erwin PJ, Gami LA, Somers VK et al. Metabolic Syndrome and Risk of Incident Cardiovascular Events and Death: A Systematic Review and Meta-Analysis of Longitudinal Studies. J Am Coll Cardiol 2007; 49:403-414.

(16) Link BG, Phelan J. Social Conditions As Fundamental Causes of Disease. J Health Soc Behav 1995; 35:80-94.

(17) Waitzkin H. The Social Origins of Illness: A Neglected History. In: Krieger N, editor. Embodying Inequality. Epidemiologic Perspectives. First ed. New York: Baywood Publishing Company, Inc.; 2005. 21-50.

(18) Antonovsky A. Social Class, Life Expectancy and Overall Mortality. Milbank Q 1967; 45:31-73.

(19) Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. Circulation 1993; 88:1973-1998.

(20) Department of Health. Tackling Health Inequalities: A Programme for Action. London: Department of Health Publications; 2003.

(21) Mackenbach JP, Cavelaars AEJM, Kunst AE, Groenhof F. Socioeconomic inequalities in cardiovascular disease mortality. An international study. Eur Heart J 2000; 21:1141-1151.

(22) Avendano M, Kunst AE, Huisman M, Lenthe FV, Bopp M, Regidor E et al. Socioeconomic status and ischaemic heart disease mortality in 10 western European populations during the 1990s. Heart 2006; 92:461-467.

(23) Kivimaki M, Lawlor DA, Davey Smith G, Kouvonen A, Virtanen M, Elovainio M et al. Socioeconomic Position, Co-Occurrence of Behavior-Related Risk Factors, and Coronary Heart Disease: the Finnish Public Sector Study. Am J Public Health 2007; 97:874-879.

(24) Liu K, Cedres LB, Stamler J, Dyer A, Stamler R, Nanas S et al. Relationship of education to major risk factors and death from coronary heart disease, cardiovascular diseases and all causes, Findings of three Chicago epidemiologic studies. Circulation 1982; 66:1308-1314.

(25) Marmot MG, Bosma H, Hemingway H, Brunner E, Stansfeld S. Contribution of job control and other risk factors to social variations in coronary heart disease incidence. Lancet 1997; 350:235-239.

(26) Suadicani P, Hein HO, Gyntelberg F. Strong mediators of social inequalities in risk of ischaemic heart disease: a six-year follow-up in the Copenhagen Male Study. Int J Epidemiol 1997; 26:516-522.

(27) Turrell G, Mathers C. Socioeconomic inequalities in all-cause and specific-cause mortality in Australia: 1985-1987 and 1995-1997. Int J Epidemiol 2001; 30:231-239.

(28) Emberson JR, Whincup PH, Morris RW, Walker M. Social class differences in coronary heart disease in middle-aged British men: implications for prevention. Int J Epidemiol 2004; 33:289-296.

(29) Huisman M, Kunst AE, Bopp M, Borgan JK, Borrell C, Costa G et al. Educational inequalities in cause-specific mortality in middle-aged and older men and women in eight western European populations. Lancet 2005; 365:493-500.

(30) Kunst AE, Groenhof F, Mackenbach JP, EU Working Group on Socioeconomic Inequalities in Health. Occupational class and cause specific mortality in middle aged men in 11 European countries: comparison of population based studies. BMJ 1998; 316:1636-1642.

(31) United Nations Population Division. World Population Prospects: The 2006 Revision. UN [2007 [cited 2008 July 4]; Available from: URL:http://esa.un.org/unpp/

(32) Huisman M, Kunst AE, Andersen O, Bopp M, Borgan JK, Borrell C et al. Socioeconomic inequalities in mortality among elderly people in 11 European populations. J Epidemiol Community Health 2004; 58:468-475.

(33) Marmot MG, Shipley MJ. Do socioeconomic differences in mortality persist after retirement? 25 Year follow up of civil servants from the first Whitehall study. BMJ 1996; 313:1177-1180.

(34) Sir Donald Acheson. Independent Inquiry into Inequalities in Health. London: The Stationary Office; 1998.

(35) Lynch J, Davey Smith G, Harper S, Bainbridge K. Explaining the social gradient in coronary heart disease: comparing relative and absolute risk approaches. J Epidemiol Community Health 2006; 60:436-441.

(36) Khang YH, Lynch J, Jung-Choi K, Cho HJ. Explaining Age Specific Inequalities in Mortality from All Causes, Cardiovascular Disease and Ischaemic Heart Disease Among South Korean Male Public Servants: Relative and Absolute Perspectives. Heart 2008; 94:75-82.

(37) Pocock SJ, Cook DG, Shaper AG, Phillips AN, Walker M. Social class differences in ischaemic heart disease in British men. Lancet 1987; 330:197-201.

(38) Lynch J, Kaplan G, Cohen R, et al. Do known risk factors explain the relation between socioeconomic status risk of all-cause mortality, cardiovascular mortality and acute myocardial infarction? Am J Epidemiol 1996; 144:934-942.

(39) Rose G, Marmot MG. Social class and coronary heart disease. Heart 1981; 45:13-19.

(40) Jousilahti P, Salomaa V, Rasi V, Vahtera E, Palosuo T. Association of markers of systemic inflammation, C reactive protein, serum amyloid A, and fibrinogen, with socioeconomic status. J Epidemiol Community Health 2003; 57:730-733.

(41) Koster A, Bosma H, Penninx BWJH, Newman AB, Harris TB, van Eijk JT et al. Association of Inflammatory Markers With Socioeconomic Status. J Gerontol A Biol Sci Med Sci 2006; 61:284-290.

(42) Loucks EB, Rehkopf DH, Thurston RC, Kawachi I. Socioeconomic Disparities in Metabolic Syndrome Differ by Gender: Evidence from NHANES III. Ann Epidemiol 2007; 17:19-26.

(43) Wannamethee SG, Lowe GDO, Whincup PH, Rumley A, Walker M, Lennon L. Physical Activity and Hemostatic and Inflammatory Variables in Elderly Men. Circulation 2002; 105:1785-1790.

(44) Wannamethee SG, Lowe GDO, Shaper AG, Rumley A, Lennon L, Whincup PH. Associations between cigarette smoking, pipe/cigar smoking, and smoking cessation, and haemostatic and inflammatory markers for cardiovascular disease. Eur Heart J 2005; 26:1765-1773.

(45) Wannamethee SG, Shaper AG, Whincup PH. Modifiable Lifestyle Factors and the Metabolic Syndrome in Older Men: Effects of Lifestyle Changes. J Am Geriatr Soc 2006; 54:1909-1914.

(46) Lynch J, Davey Smith G. A life course approach to chronic disease epidemiology. Annu Rev Public Health 2005; 26:1-35.

(47) Kermack WO, McKendrick AG, Mckinlay PL. Death-rates in Great Britain and Sweden. Some general regularities and their significance. Lancet 1934; 223:698-703.

(48) Forsdahl A. Are poor living conditions in childhood and adolescence an important risk factor for arteriosclerotic heart disease? Br J Prev Soc Med 1977; 31:91-95.

(49) Barker DJ, Winter PD, Osmond C, Margetts B, Simmonds SJ. Weight in infancy and death from ischaemic heart disease. Lancet 1989; 334:577-580.

(50) Barker DJ. Fetal origins of coronary heart disease. BMJ 1995; 311:171-174.

(51) Barker DJ, Forsen T, Uutela A, Osmond C, Eriksson JG. Size at birth and resilience to effects of poor living conditions in adult life: longitudinal study. BMJ 2001; 323:1273-1276.

(52) Lawlor DA, Ben-Shlomo Y, Leon DA. Pre-adult influences on cardiovascular disease. In: Kuh D, Ben-Shlomo Y, editors. A life course approach to chronic disease epidemiology. 2 ed. Oxford: Oxford University Press; 2004. 41-76.

(53) Galobardes B, Lynch JW, Davey Smith G. Is the association between childhood socioeconomic circumstances and cause-specific mortality established? Update of a systematic review. J Epidemiol Community Health 2008; 62:387-390.

(54) Galobardes B, Lynch JW, Davey Smith G. Childhood Socioeconomic Circumstances and Cause-specific Mortality in Adulthood: Systematic Review and Interpretation. Epidemiol Rev 2004; 26:7-21.

(55) Davey Smith G, Lynch J. Life course approaches to socioeconomic differentials in health. In: Kuh D, Ben-Shlomo Y, editors. A life course approach to chronic disease epidemiology. 2 ed. Oxford: Oxford University Press; 2004. 77-115.

(56) Hart CL, Davey Smith G. Relation between number of siblings and adult mortality and stroke risk: 25 year follow up of men in the Collaborative study. J Epidemiol Community Health 2003; 57:385-391.

(57) Notkola V, Punsar S, Karvonen MJ, Haapakoski J. Socio-economic conditions in childhood and mortality and morbidity caused by coronary heart disease in adulthood in rural Finland. Soc Sci Med 1985; 21:517-523.

(58) Davey Smith G, Hart C, Blane D, Hole D. Adverse socioeconomic conditions in childhood and cause specific adult mortality: prospective observational study. BMJ 1998; 316:1631-1635.

(59) Burden of Disease Network Project. Disability in Old Age. The Finnish Centre for Interdisciplinary Gerontology, University of Jyväskylä Finland [2004 [cited 2006 Sept. 7]; Available from: URL:http://www.jyu.fi/BURDIS/FinalReport.pdf

(60) Guralnik JM, Fried LP, Salive ME. Disability as a Public Health Outcome in the Aging Population. Annu Rev Public Health 1996; 17:25-46.

(61) Stewart AL, Greenfield S, Hays RD, Wells K, Rogers WH, Berry SD et al. Functional status and well-being of patients with chronic conditions. Results from the Medical Outcomes Study. JAMA 1989; 262:907-913.

(62) Ebrahim S, Wannamethee SG, Whincup P, Walker M, Shaper AG. Locomotor disability in a cohort of British men: the impact of lifestyle and disease. Int J Epidemiol 2000; 29:478-486.

(63) Kattainen A, Koskinen S, Reunanen A, Martelin T, Knekt P, Aromaa A. Impact of cardiovascular diseases on activity limitations and need for help among older persons. J Clin Epidemiol 2004; 57:82-88.

(64) Pinsky JL, Jette AM, Branch LG, Kannel WB, Feinleib M. The Framingham Disability Study: relationship of various coronary heart disease manifestations to disability in older persons living in the community. Am J Public Health 1990; 80:1363-1367.

(65) Grundy E, Glaser K. Socio-demographic differences in the onset and progression of disability in early old age: a longitudinal study. Age Ageing 2000; 29:149-157.

(66) Melzer D, McWilliams B, Brayne C, Johnson T, Bond J. Socioeconomic status and the expectation of disability in old age: estimates for England. J Epidemiol Community Health 2000; 54:286-292.

(67) Sigerist HE. The Wesley M. Carpenter Lecture: "Historical Background of Industrial and Occupational Diseases". Bull N Y Acad Med 1936; 12:597-609.

(68) Omran AR. The Epidemiologic Transition: A Theory of the Epidemiology of Population Change. Milbank Q 1971; 49:509-538.

(69) Black D, Morris JN, Smith C, Townsend P. Inequalities in health: report of a Research Working Group. London: Department of Health and Social Security; 1980.

(70) Nazroo JY. The Structuring of Ethnic Inequalities in Health: Economic Position, Racial Discrimination, and Racism. Am J Public Health 2003; 93:277-284.

(71) Davey Smith G, Neaton JD, Wentworth D, Stamler R, Stamler J. Socioeconomic differentials in mortality risk among men screened for the Multiple Risk Factor Intervention Trial: I. White men. Am J Public Health 1996; 86:486-496.

(72) Shaw M, Davey Smith G, Dorling D. Health inequalities and New Labour: how the promises compare with real progress. BMJ 2005; 330:1016-1021.

(73) Macintyre S, Hunt K, Sweeting H. Gender differences in health: Are things really as simple as they seem? Soc Sci Med 1996; 42:617-624.

(74) Dalstra JA, Kunst AE, Borrell C, Breeze E, Cambois E, Costa G et al. Socioeconomic differences in the prevalence of common chronic diseases: an overview of eight European countries. Int J Epidemiol 2005; 34:316-326.

(75) Davey Smith G, Hart C, Manning GC, Lantz PM, House JS. Socioeconomic Factors and Determinants of Mortality. JAMA 1998; 280:1744-1745.

(76) Davey Smith G, Dorling D, Mitchell R, Shaw M. Health inequalities in Britain: continuing increases up to the end of the 20th century. J Epidemiol Community Health 2002; 56:434-435.

(77) Leon D, Vagero D, Olausson PO. Social class differences in infant mortality in Sweden: comparison with England and Wales. BMJ 1992; 19:687-691.

(78) Kunst AE, Groenhof F, Andersen O, Borgan JK, Costa G, Desplanques G et al. Occupational class and ischemic heart disease mortality in the United States and 11 European countries. Am J Public Health 1999; 89:47-53.

(79) Galobardes B, Shaw M, Lawlor DA, Lynch JW, Davey Smith G. Indicators of socioeconomic position (part 1). J Epidemiol Community Health 2006; 60:7-12.

(80) Krieger N, Williams DR, Moss NE. Measuring Social Class in US Public Health Research:Concepts, Methodologies, and Guidelines. Annu Rev Public Health 1997; 18:341-378.

(81) Wright EO, Perrone L. Marxist Class Categories and Income Inequality. Am Sociol Rev 1977; 42:32-55.

(82) Wright EO. The Shadow of Exploitation in Weber's Class Analysis. Am Sociol Rev 2002; 67:832-853.

(83) Bartley M. Measuring Socio-economic Position. In: Bartley M, editor. Health Inequality. An introduction to theories, concepts and methods. First ed. Cambridge: Polity Press; 2004. 22-34.

(84) Lynch J, Kaplan G. Socioeconomic position. In: Berkman LF, Kawachi I, editors. Social Epidemiology. First ed. New York: Oxford University Press; 2000. 13-35.

(85) Davey Smith G, Blane D, Bartley M. Explanations for socio-economic differentials in mortality: Evidence from Britain and elsewhere. Eur J Public Health 1994; 4:131-144.

(86) Strong PM. Black on class and mortality: theory, method and history. J Public Health 1990; 12:168-180.

(87) Blane D, Davey Smith G, Bartley M. Social selection: what does it contribute to social class differences in health? Sociol Health Ill 1985; 15:1-15.

(88) Bartley M, Plewis I. Increasing social mobility: an effective policy to reduce health inequalities. J Roy Stat Soc A Sta 2007; 170:469-481.

(89) Szreter SRS. The Genesis of the Registrar-General's Social Classification of Occupations. Br J Sociol 1984; 35:522-546.

(90) Jones IG, Cameron D. Social class analysis--an embarrassment to epidemiology. Community Med 1984; 6:37-46.

(91) Leete R, Fox J. Registrar General's social classes: origins and uses. Population Trends 1977; 8:1-7.

(92) Prandy K. The Revised Cambridge Scale of Occupations. Sociology 1990; 24:629-655.

(93) Bartley M, Carpenter L, Dunnell K, Fitzpatrick R. Measuring inequalities in health: an analysis of mortality patterns using two social classifications. Sociol Health Ill 1996; 18:455-475.

(94) Elo IT, Preston SH. Educational differentials in mortality: United States, 1979-1985. Soc Sci Med 1996; 42:47-57.

(95) Winkleby MA, Jatulis DE, Frank E, Fortmann SP. Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. Am J Public Health 1992; 82:816-820.

(96) Hadden WC. The use of educational attainment as an indicator of socioeconomic position. Am J Public Health 1996; 86:1525-1526.

(97) Davey Smith G, Shipley MJ, Rose G. Magnitude and causes of socioeconomic differentials in mortality: further evidence from the Whitehall Study. J Epidemiol Community Health 1990; 44:265-270.

(98) Bucher HC, Ragland DR. Socioeconomic indicators and mortality from coronary heart disease and cancer: a 22-year follow-up of middle-aged men. Am J Public Health 1995; 85:1231-1236.

(99) Health Inequalities. Decennial Supplement. London: The Stationary Office; 1997.

(100) Marmot MG, Rose G, Shipley M, Hamilton PJ. Employment grade and coronary heart disease in British civil servants. J Epidemiol Community Health 1978; 32:244-249.

(101) Yarnell JW, Baker IA, Sweetnam PM, Bainton D, O'Brien JR, Whitehead PJ et al. Fibrinogen, viscosity, and white blood cell count are major risk factors for ischemic heart disease. The Caerphilly and Speedwell collaborative heart disease studies. Circulation 1991; 83:836-844.

(102) Baker IA, Sweetnam PM, Yarmell JWG, Bainton D, Elwood PC. Haemostatic and Other Risk Factors for Ischaemic Heart Disease and Social Class: Evidence from the Caerphilly and Speedwell Studies. Int J Epidemiol 1988; 17:759-765.

(103) Woodward M, Shewry MC, Cairns S.Smith, Tunstall-Pedoe H. Social status and coronary heart disease: Results from the Scottish heart health study. Prev Med 1992; 21:136-148.

(104) Woodward M, Oliphant J, Lowe G, Tunstall-Pedoe H. Contribution of contemporaneous risk factors to social inequality in coronary heart disease and all causes mortality. Prev Med 2003; 36:561-568.

(105) Power C, Hypponen E, Davey Smith G. Socioeconomic Position in Childhood and Early Adult Life and Risk of Mortality: A Prospective Study of the Mothers of the 1958 British Birth Cohort. Am J Public Health 2005; 95:1396-1402.

(106) Lawlor DA, Ebrahim S, Davey Smith G. Adverse socioeconomic position across the lifecourse increases coronary heart disease risk cumulatively: findings from the British women's heart and health study. J Epidemiol Community Health 2005; 59:785-793.

(107) Keppel K, Pamuk E, Lynch J, et al. Methodological issues in measuring health disparities. National Center for Health Statistics. Vital Health Statistics 2005; 2.

(108) The Registrar General's Decennial Supplement (England & Wales) Part IIa. London: His Majesty's Stationary Office; 1938.

(109) Marmot MG, Adelstein AM, Robinson N, Rose GA. Changing social-class distribution of heart disease. BMJ 1978; 2:1109-1112.

(110) Morris JN. In Search of Causes. Uses of Epidemiology. Third ed. New York: Churchill Livingstone; 1975. 142-249.

(111) Reddy KS. Cardiovascular diseases in the developing countries: dimensions, determinants, dynamics and directions for public health action. Public Health Nutr 2002; 5:231-237.

(112) Harding S, Bethune A, Maxwell R, Brown J. Mortality trends using the Longitudinal Study. In: Drever F, Whitehead M, editors. London: The Stationary Office; 1997.

(113) Department of Health. Tackling Health Inequalities: Status Report on the Programme for Action. London: Department of Health Publications; 2005.

(114) Beckett M. Converging Health Inequalities in Later Life-An Artifact of Mortality Selection? Journal of Health and Social Behavior 2000; 41:106-119.

(115) House JS, Kessler RC, Herzog AR. Age, Socioeconomic Status, and Health. Milbank Q 1990; 68:383-411.

(116) Mishra GD, Ball K, Dobson AJ, Byles JE. Do socioeconomic gradients in women's health widen over time and with age? Soc Sci Med 2004; 58:1585-1595.

(117) Robert S, House JS. SES Differentials in Health by Age and Alternative Indicators of SES. J Aging Health 1996; 8:359-388.

(118) van Rossum CTM, Shipley MJ, van de Mheen H, Grobbee DE, Marmot MG. Employment grade differences in cause specific mortality. A 25 year follow up of civil servants from the first Whitehall study. J Epidemiol Community Health 2000; 54:178-184.

(119) McMunn A, Hyde M, Janevic M, Kumari M. Health. In: Marmot M, Banks J, Blundell R, Lessof C, Nazroo J, editors. Health, wealth and lifestyles of the older population in England: The 2002 English Longitudinal Study Of Ageing. London: Institute of Fiscal Studies; 2003.

(120) Banks J, Marmot M, Oldfield Z, Smith JP. Disease and Disadvantage in the United States and in England. JAMA 2006; 295:2037-2045.

(121) Sundquist K, Johansson SE, Qvist J, Sundquist J. Does occupational social class predict coronary heart disease after retirement? A 12-year follow-up study in Sweden. Scand J Public Health 2005; 33:447-454.

(122) Suadicani P, Hein HO, Gyntelberg F. Do physical and chemical working conditions explain the association of social class with ischaemic heart disease? Atherosclerosis 1995; 113:63-69.

(123) Prospective Studies Collaboration. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. Lancet 2002; 360:1903-1913.

(124) Prospective Studies Collaboration. Blood cholesterol and vascular mortality by age, sex, and blood pressure: a meta-analysis of individual data from 61 prospective studies with 55 000 vascular deaths. Lancet 2007; 370:1829-1839.

(125) Stamler J. Established major coronary risk factors. In: Marmot M, Elliott P, editors. Coronary heart disease epidemiology. From aetiology to public health. First ed. Oxford: Oxford University Press; 1994. 35-66.

(126) Doll R, Peto R, Wheatley K, Gray R, Sutherland I. Mortality in relation to smoking: 40 years' observations on male British doctors. BMJ 1994; 309:901-911.

(127) Neaton JD, Kuller LH, Wentworth D, Borhani NO. Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol concentration, and diastolic blood pressure among black and white males followed up for five years. Am Heart J 1984; 108:759-769.

(128) Reid DD, Mccartney P, Hamilton PJS, Rose G, Jarrett RJ, Keen H. Smoking and other risk factors for coronary heart-disease in British civil servants. Lancet 1976; 308:979-984.

(129) Shaper AG, Pocock SJ, Walker M, Phillips AN, Whitehead TP, Macfarlane PW. Risk factors for ischaemic heart disease: the prospective phase of the British Regional Heart Study. J Epidemiol Community Health 1985; 39:197-209.

(130) Jajich CL, Ostfeld AM, Freeman DH, Jr. Smoking and coronary heart disease mortality in the elderly. JAMA 1984; 252:2831-2834.

(131) Ben-Shlomo Y, Davey Smith G, Shipley MJ, Marmot MG. What determines mortality risk in male former cigarette smokers? Am J Public Health 1994; 84:1235-1242.

(132) Critchley JA, Capewell S. Mortality risk reduction associated with smoking cessation in patients with coronary heart disease: a systematic review. JAMA 2003; 290:86-97.

(133) Wilhelmsson C, Elmfeldt D, Vedin JA, Tibblin G, Wilhelmsen L. Smoking and myocardial infarction. Lancet 1975; 305:415-420.

(134) He J, Vupputuri S, Allen K, Prerost MR, Hughes J, Whelton PK. Passive Smoking and the Risk of Coronary Heart Disease -- A Meta-Analysis of Epidemiologic Studies. N Engl J Med 1999; 340:920-926.

(135) Law MR, Morris JK, Wald NJ. Environmental tobacco smoke exposure and ischaemic heart disease: an evaluation of the evidence. BMJ 1997; 315:973-980.

(136) Whincup PH, Gilg JA, Emberson JR, Jarvis MJ, Feyerabend C, Bryant A et al. Passive smoking and risk of coronary heart disease and stroke: prospective study with cotinine measurement. BMJ 2004; 329:200-205.

(137) Tunstall-Pedoe H, Brown CA, Woodward M, Tavendale R. Passive smoking by self report and serum cotinine and the prevalence of respiratory and coronary heart disease in the Scottish heart health study. J Epidemiol Community Health 1995; 49:139-143.

(138) Blood Pressure Lowering Treatment Trialists' Collaboration. Effects of ACE inhibitors, calcium antagonists, and other blood-pressure-lowering drugs: results of prospectively designed overviews of randomised trials. Lancet 2000; 356:1955-1964.

(139) Staessen JA, Wang JG, Thijs L. Cardiovascular protection and blood pressure reduction: a meta-analysis. Lancet 2001; 358:1305-1315.

(140) Law MR, Wald NJ, Morris JK, Jordan RE. Value of low dose combination treatment with blood pressure lowering drugs: analysis of 354 randomised trials. BMJ 2003; 326:1427.

(141) Staessen JA, Gasowski J, Wang JG, Thijs L, Hond ED, Boissel JP et al. Risks of untreated and treated isolated systolic hypertension in the elderly: meta-analysis of outcome trials. Lancet 2000; 355:865-872.

(142) Elliott P, Stamler J, Nichols R, Dyer AR, Stamler R, Kesteloot H et al. Intersalt revisited: further analyses of 24 hour sodium excretion and blood pressure within and across populations. BMJ 1996; 312:1249-1253.

(143) Law MR, Frost CD, Wald NJ. By how much does dietary salt reduction lower blood pressure? I--Analysis of observational data among populations. BMJ 1991; 302:811-815.

(144) Law MR, Frost CD, Wald NJ. By how much does dietary salt reduction lower blood pressure? III--Analysis of data from trials of salt reduction. BMJ 1991; 302:819-824.

(145) Neter JE, Stam BE, Kok FJ, Grobbee DE, Geleijnse JM. Influence of weight reduction on blood pressure: a meta-analysis of randomized controlled trials. Hypertension 2003; 42:878-884.

(146) Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. N Engl J Med 1997; 336:1117-1124.

(147) Whelton PK, He J, Cutler JA, Brancati FL, Appel LJ, Follmann D et al. Effects of oral potassium on blood pressure. Meta-analysis of randomized controlled clinical trials. JAMA 1997; 277:1624-1632.

(148) Barker DJ, Bull AR, Osmond C, Simmonds SJ. Fetal and placental size and risk of hypertension in adult life. BMJ 1990; 301:259-262.

(149) Law MR, Wald NJ, Thompson SG. By how much and how quickly does reduction in serum cholesterol concentration lower risk of ischaemic heart disease? BMJ 1994; 308:367-372.

(150) Cholesterol Treatment Trialists' (CTT) Collaborators. Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of data from 90, 056 participants in 14 randomised trials of statins. Lancet 2005; 366:1267-1278.

(151) Clarke R, Frost C, Collins R, Appleby P, Peto R. Dietary lipids and blood cholesterol: quantitative meta-analysis of metabolic ward studies. BMJ 1997; 314:112-117.

(152) Hegsted DM, Ausman LM, Johnson JA, Dallal GE. Dietary fat and serum lipids: an evaluation of the experimental data. Am J Clin Nutr 1993; 57:875-883.

(153) Keys A, Anderson J, Grande F. Prediction of serum-cholesterol responses of man to changes in fats in the diet. Lancet 1957; 270:959-966.

(154) Morris JN, Kagan A, Pattison DC, Gardner MJ, Raffle PAB. Incidence and prediction of ischaemic heart-disease in London busmen. Lancet 1966; 288:553-559.

(155) Morris JN, Clayton DG, Everitt MG, Semmence AM, Burgess EH. Exercise in leisure time: coronary attack and death rates. Br Heart J 1990; 63:325-334.

(156) Berlin JA, Colditz GA. A meta-analysis of physical activity in the prevention of coronary heart disease. Am J Epidemiol 1990; 132:612-628.

(157) Kushi LH, Fee RM, Folsom AR, Mink PJ, Anderson KE, Sellers TA. Physical activity and mortality in postmenopausal women. JAMA 1997; 277:1287-1292.

(158) Leon AS, Connett J, Jacobs DR, Jr., Rauramaa R. Leisure-time physical activity levels and risk of coronary heart disease and death. The Multiple Risk Factor Intervention Trial. JAMA 1987; 258:2388-2395.

(159) Manson JE, Hu FB, Rich-Edwards JW, Colditz GA, Stampfer MJ, Willett WC et al. A prospective study of walking as compared with vigorous exercise in the prevention of coronary heart disease in women. N Engl J Med 1999; 341:650-658.

(160) Rodriguez BL, Curb JD, Burchfiel CM, Abbott RD, Petrovitch H, Masaki K et al. Physical activity and 23-year incidence of coronary heart disease morbidity and mortality among middle-aged men. The Honolulu Heart Program. Circulation 1994; 89:2540-2544.

(161) Sesso HD, Paffenbarger RS, Ha T, Lee IM. Physical activity and cardiovascular disease risk in middle-aged and older women. Am J Epidemiol 1999; 150:408-416.

(162) Shaper AG, Wannamethee G, Weatherall R. Physical activity and ischaemic heart disease in middle-aged British men. Heart 1991; 66:384-394.

(163) Hakim AA, Curb JD, Petrovitch H, Rodriguez BL, Yano K, Ross GW et al. Effects of Walking on Coronary Heart Disease in Elderly Men : The Honolulu Heart Program. Circulation 1999; 100:9-13.

(164) LaCroix AZ, Leveille SG, Hecht JA, Grothaus LC, Wagner EH. Does walking decrease the risk of cardiovascular disease hospitalizations and death in older adults? J Am Geriatr Soc 1996; 44:113-120.

(165) Paffenbarger RS, Hyde RT, Wing AL, Lee IM, Jung DL, Kampert JB. The Association of Changes in Physical-Activity Level and Other Lifestyle Characteristics with Mortality among Men. N Engl J Med 1993; 328:538-545.

(166) Wannamethee SG, Shaper AG, Walker M. Changes in physical activity, mortality, and incidence of coronary heart disease in older men. Lancet 1998; 351:1603-1608.

(167) Leon AS, Connett J. Physical activity and 10.5 year mortality in the Multiple Risk Factor Intervention Trial (MRFIT). Int J Epidemiol 1991; 20:690-697.

(168) Jousilahti P, Tuomilehto J, Vartiainen E, Pekkanen J, Puska P. Body Weight, Cardiovascular Risk Factors, and Coronary Mortality : 15-Year Follow-up of Middle-aged Men and Women in Eastern Finland. Circulation 1996; 93:1372-1379.

(169) Rosengren A, Wedel H, Wilhelmsen L. Body weight and weight gain during adult life in men in relation to coronary heart disease and mortality: A prospective population study. Eur Heart J 1999; 20:269-277.

(170) Shaper AG, Wannamethee SG, Walker M. Body weight: implications for the prevention of coronary heart disease, stroke, and diabetes mellitus in a cohort study of middle aged men. BMJ 1997; 314:1311-1317.

(171) Asia Pacific Cohort Studies Collaboration. Body mass index and cardiovascular disease in the Asia-Pacific Region: an overview of 33 cohorts involving 310 000 participants. Int J Epidemiol 2004; 33:751-758.

(172) Bogers RP, Bemelmans WJE, Hoogenveen RT, Boshuizen HC, Woodward M, Knekt P et al. Association of Overweight With Increased Risk of Coronary Heart Disease Partly Independent of Blood Pressure and Cholesterol Levels: A Meta-analysis of 21 Cohort Studies Including More Than 300 000 Persons. Arch Intern Med 2007; 167:1720-1728.

(173) Whitlock G, Lewington S, Mhurchu C. Coronary Heart Disease and Body Mass Index: A Systematic Review of the Evidence from Larger Prospective Cohort Studies. Semin Vasc Med 2002; 02:369-382.

(174) Inelmen EM, Sergi G, Coin A, Miotto F, Peruzza S, Enzi G. Can obesity be a risk factor in elderly people? Obes Rev 2003; 44:147-155.

(175) Rimm EB, Stampfer MJ, Giovannucci E, Ascherio A, Spiegelman D, COLDITZ GA et al. Body Size and Fat Distribution as Predictors of Coronary Heart Disease among Middle-aged and Older US Men. Am J Epidemiol 1995; 141:1117-1127.

(176) Silventoinen K, Jousilahti P, Vartiainen E, Tuomilehto J. Appropriateness of anthropometric obesity indicators in assessment of coronary heart disease risk among Finnish men and women. Scand J Public Health 2003; 31:283-290.

(177) Yusuf S, Hawken S, Ôunpuu S, Bautista L, Franzosi MG, Commerford P et al. Obesity and the risk of myocardial infarction in 27 000 participants from 52 countries: a case-control study. Lancet 2005; 366:1640-1649.

(178) Emberson JR, Whincup PH, Morris RW, Wannamethee SG, Shaper AG. Lifestyle and cardiovascular disease in middle-aged British men: the effect of adjusting for within-person variation. Eur Heart J 2005; 26:1774-1782.

(179) Stampfer MJ, Hu FB, Manson JE, Rimm EB, Willett WC. Primary Prevention of Coronary Heart Disease in Women through Diet and Lifestyle. N Engl J Med 2000; 343:16-22.

(180) Blane D, Hart CL, Davey Smith G, Gillis CR, Hole DJ, Hawthorne VM. Association of cardiovascular disease risk factors with socioeconomic position during childhood and during adulthood. BMJ 1996; 313:1434-1438.

(181) Kopelman PG. Obesity as a medical problem. Nature 2000; 404:635-643.

(182) Corrao G, Rubbiati L, Bagnardi V, Zambon A, Poikolainen K. Alcohol and coronary heart disease: a meta-analysis. Addiction 2000; 95:1505-1523.

(183) Rehm J, Sempos CT, Trevisan M. Alcohol and cardiovascular disease--more than one paradox to consider. Average volume of alcohol consumption, patterns of drinking and risk of coronary heart disease--a review. J Cardiovasc Risk 2003; 10:15-20.

(184) Shaper AG. Alcohol and mortality: a review of prospective studies. Br J Addict 1990; 85:837-847.

(185) Murray RP, Connett JE, Tyas SL, Bond R, Ekuma O, Silversides CK et al. Alcohol Volume, Drinking Pattern, and Cardiovascular Disease Morbidity and Mortality: Is There a U-shaped Function? Am J Epidemiol 2002; 155:242-248.

(186) Rosengren A, Wilhelmsen L, Pennert K, Berglund G, Elmfeldt D. Alcoholic intemperance, coronary heart disease and mortality in middle-aged Swedish men. Acta Med Scand 1987; 222:201-213.

(187) Danesh J, Collins R, Peto R, Lowe GDO. Haematocrit, viscosity, erythrocyte sedimentation rate: meta-analyses of prospective studies of coronary heart disease. Eur Heart J 2000; 21:515-520.

(188) Danesh J, Kaptoge S, Mann AG, Sarwar N, Wood A, Angleman SB et al. Long-Term Interleukin-6 Levels and Subsequent Risk of Coronary Heart Disease: Two New Prospective Studies and a Systematic Review. PLoS Med 2008; 5:e78.

(189) Danesh J, Collins R, Appleby P, Peto R. Association of Fibrinogen, C-reactive Protein, Albumin, or Leukocyte Count With Coronary Heart Disease: Meta-analyses of Prospective Studies. JAMA 1998; 279:1477-1482.

(190) Danesh J, Whincup P, Walker M, Lennon L, Thomson A, Appleby P et al. Fibrin D-Dimer and Coronary Heart Disease : Prospective Study and Meta-Analysis. Circulation 2001; 103:2323-2327.

(191) Danesh J, Wheeler JG, Hirschfield GM, Eda S, Eiriksdottir G, Rumley A et al. C-Reactive Protein and Other Circulating Markers of Inflammation in the Prediction of Coronary Heart Disease. N Engl J Med 2004; 350:1387-1397.

(192) Fibrinogen Studies Collaboration. Collaborative meta-analysis of prospective studies of plasma fibrinogen and cardiovascular disease. Eur J Cardiovasc Prev Rehab 2004; 11:9-17.

(193) Lowe GDO, Danesh J, Lewington S, Walker M, Lennon L, Thomson A et al. Tissue plasminogen activator antigen and coronary heart disease: Prospective study and meta-analysis. Eur Heart J 2004; 25:252-259.

(194) Kritchevsky SB, Cesari M, Pahor M. Inflammatory markers and cardiovascular health in older adults. Cardiovasc Res 2005; 66:265-275.

(195) Fantuzzi G. Adipose tissue, adipokines, and inflammation. J Allergy Clin Immunol 2005; 115:911-919.

(196) Wannamethee SG, Lowe GDO, Shaper AG, Whincup PH, Rumley A, Walker M et al. The effects of different alcoholic drinks on lipids, insulin and haemostatic and inflammatory markers in older men. Thromb Haemost 2003; 90:1080-1087.

(197) Wannamethee SG, Lowe GDO, Shaper AG, Rumley A, Lennon L, Whincup PH. Insulin resistance, haemostatic and inflammatory markers and coronary heart disease risk factors in Type 2 diabetic men with and without coronary heart disease. Diabetologia 2004; 47:1557-1565.

(198) Casas JP, Shah T, Cooper J, Hawe E, McMahon AD, Gaffney D et al. Insight into the nature of the CRP-coronary event association using Mendelian randomization. Int J Epidemiol 2006; 35:922-931.

(199) Davey Smith G, Harbord R, Milton J, Ebrahim S, Sterne JAC. Does Elevated Plasma Fibrinogen Increase the Risk of Coronary Heart Disease?: Evidence from a Meta-Analysis of Genetic Association Studies. Arterioscler Thromb Vasc Biol 2005; 25:2228-2233.

(200) Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. Diabetes 1998; 37:1595-1607.

(201) Kahn R, Buse J, Ferrannini E, Stern M. The Metabolic Syndrome: Time for a Critical Appraisal: Joint statement from the American Diabetes Association and the European Association for the Study of Diabetes. Diabetes Care 2005; 28:2289-2304.

(202) Sarwar N, Sattar N, Gudnason V, Danesh J. Circulating concentrations of insulin markers and coronary heart disease: a quantitative review of 19 Western prospective studies. Eur Heart J 2007; 28:2491-2497.

(203) James PT, Rigby N, Leach R. The obesity epidemic, metabolic syndrome and future prevention strategies. Eur J Cardiovasc Prev Rehab 2004; 11:3-8.

(204) Sattar N, McConnachie A, Shaper AG, Blauw GJ, Buckley BM, de Craen AJ et al. Can metabolic syndrome usefully predict cardiovascular disease and diabetes? Outcome data from two prospective studies. Lancet 2008; 371:1927-1935.

(205) Yarnell JWG, Patterson CC, Bainton D, Sweetnam PM. Is metabolic syndrome a discrete entity in the general population? Evidence from the Caerphilly and Speedwell population studies. Heart 1998; 79:248-252.

(206) Willett W. Diet and coronary heart disease. In: Willett W, editor. Nutritional Epidemiology. Second ed. New York: Oxford University Press; 1998. 414-466.

(207) Khaw KT, Bingham S, Welch A, Luben R, Wareham N, Oakes S et al. Relation between plasma ascorbic acid and mortality in men and women in EPIC-Norfolk prospective study: a prospective population study. Lancet 2001; 357:657-663.

(208) MRC/BHF Heart Protection Study of antioxidant vitamin supplementation in 20, 536 high-risk individuals: a randomised placebo-controlled trial. Lancet 2002; 360:23-33.

(209) Sesso HD, Buring JE, Christen WG, Kurth T, Belanger C, MacFadyen J et al. Vitamins E and C in the Prevention of Cardiovascular Disease in Men: The Physicians' Health Study II Randomized Controlled Trial. JAMA 2008; 300:2123-2133.

(210) Hein HO, Suadicani P, Gyntelberg F. Ischaemic heart disease incidence by social class and form of smoking: the Copenhagen Male Study--17 years' follow-up. J Intern Med 1992; 231:477-483.

(211) Laaksonen M, Talala K, Martelin T, Rahkonen O, Roos E, Helakorpi S et al. Health behaviours as explanations for educational level differences in cardiovascular and all-cause mortality: a follow-up of 60 000 men and women over 23 years. Eur J Public Health 2008; 18:38-43.

(212) Albert MA, Glynn RJ, Buring J, Ridker PM. Impact of Traditional and Novel Risk Factors on the Relationship Between Socioeconomic Status and Incident Cardiovascular Events. Circulation 2006; 114:2619-2626.

(213) Loucks EB, Sullivan LM, Hayes LJ, D'Agostino RB, Sr., Larson MG, Vasan RS et al. Association of Educational Level with Inflammatory Markers in the Framingham Offspring Study. Am J Epidemiol 2006; 163:622-628.

(214) Bartley M, Sacker A, Firth D, Fitzpatrick R. Understanding social variation in cardiovascular risk factors in women and men: the advantage of theoretically based measures. Soc Sci Med 1999; 49:831-845.

(215) Marmot MG, Stansfeld S, Patel C, North F, Head J, White I et al. Health inequalities among British civil servants: the Whitehall II study. Lancet 1991; 337:1387-1393.

(216) Regidor E, Gutierrez-Fisac JL, Banegas JR, Dominguez V, Rodriguez-Artalejo F. Association of adult socioeconomic position with hypertension in older people. J Epidemiol Community Health 2006; 60:74-80.

(217) Svetkey LP, George LK, Burchett BM, Morgan PA, Blazer DG. Black/White Differences in Hypertension in the Elderly An Epidemiologic Analysis in Central North Carolina. Am J Epidemiol 1993; 137:64-73.

(218) Avendano M, Kawachi I, Van Lenthe F, Boshuizen HC, Mackenbach JP, Van den Bos GAM et al. Socioeconomic Status and Stroke Incidence in the US Elderly: The Role of Risk Factors in the EPESE Study. Stroke 2006; 37:1368-1373.

(219) Winkleby MA, Fortmann SP, Barrett DC. Social class disparities in risk factors for disease: Eight-year prevalence patterns by level of education. Prev Med 1990; 19:1-12.

(220) Lawlor DA, Ebrahim S, Davey Smith G. Socioeconomic position in childhood and adulthood and insulin resistance: cross sectional survey using data from British women's heart and health study. BMJ 2002; 325:805.

(221) Hebert PR, Buring JE, O'Connor GT, Rosner B, Hennekens CH. Occupation and risk of nonfatal myocardial infarction. Arch Intern Med 1992; 152:2253-2257.

(222) Kivimaki M, Shipley MJ, Ferrie JE, Singh-Manoux A, Batty GD, Chandola T et al. Best-practice interventions to reduce socioeconomic inequalities of coronary heart disease mortality in UK: a prospective occupational cohort study. Lancet 2008; 372:1648-1654.

(223) Brunner E, Marmot M, Canner R, Beksinska M, Davey Smith G, O'Brien J. Childhood social circumstances and psychosocial and behavioural factors as determinants of plasma fibrinogen. Lancet 1996; 347:1008-1013.

(224) Steptoe A, Kunz-Ebrecht S, Rumley A, Lowe GDO. Prolonged elevations in haemostatic and rheological responses following psychological stress in low socioeconomic status men and women. Thromb Haemost 2003; 89:83-90.

(225) Lubbock LA, Goh A, Ali S, Ritchie J, Whooley MA. Relation of Low Socioeconomic Status to C-Reactive Protein in Patients With Coronary Heart Disease (from the Heart and Soul Study). Am J Cardiol 2005; 96:1506-1511.

(226) Hemingway H, Shipley M, Mullen MJ, Kumari M, Brunner E, Taylor M et al. Social and psychosocial influences on inflammatory markers and vascular function in civil servants (the Whitehall II study). Am J Cardiol 2003; 92:984-987.

(227) Myllykangas M, Pekkanen J, Rasi V, Haukkala A, Vahtera E, Salomaa V. Haemostatic and Other Cardiovascular Risk Factors, and Socioeconomic Status

among Middle-Aged Finnish Men and Women. Int J Epidemiol 1995; 24:1110-1116.

(228) Rosengren A, Wilhelmsen L, Welin L, Tsipogianni A, Teger-Nilsson AC, Wedel H. Social influences and cardiovascular risk factors as determinants of plasma fibrinogen concentration in a general population sample of middle aged men. BMJ 1990; 300:634-638.

(229) Kumari M, Marmot M, Brunner E. Social Determinants of von Willebrand Factor: The Whitehall II Study. Arterioscler Thromb Vasc Biol 2000; 20:1842-1847.

(230) Yarnell JWG, Sweetnam PM, Rumley A, Lowe GDO. Lifestyle factors and coagulation activation markers: the Caerphilly Study. Blood Coagul Fibrinolysis 2001; 12:721-728.

(231) Kivimaki M, Lawlor DA, Juonala M, Davey Smith G, Elovainio M, Keltikangas-Jarvinen L et al. Lifecourse Socioeconomic Position, C-Reactive Protein, and Carotid Intima-Media Thickness in Young Adults: The Cardiovascular Risk in Young Finns Study. Arterioscler Thromb Vasc Biol 2005; 25:2197-2202.

(232) Ferrucci L, Corsi A, Lauretani F, Bandinelli S, Bartali B, Taub DD et al. The origins of age-related proinflammatory state. Blood 2005; 105:2294-2299.

(233) Silventoinen K, Pankow J, Jousilahti P, Hu G, Tuomilehto J. Educational inequalities in the metabolic syndrome and coronary heart disease among middle-aged men and women. Int J Epidemiol 2005; 34:327-334.

(234) Abraham NG, Brunner EJ, Eriksson JW, Robertson RP. Metabolic Syndrome. Psychosocial, Neuroendocrine, and Classical Risk Factors in Type 2 Diabetes. Ann N Y Acad Sci 2007; 1113:256-275.

(235) Brunner EJ, Hemingway H, Walker BR, Page M, Clarke P, Juneja M et al. Adrenocortical, Autonomic, and Inflammatory Causes of the Metabolic Syndrome: Nested Case-Control Study. Circulation 2002; 106:2659-2665.

(236) Dallongeville J, Cottel D, Ferrieres J, Arveiler D, Bingham A, Ruidavets JB et al. Household Income Is Associated With the Risk of Metabolic Syndrome in a Sex-Specific Manner. Diabetes Care 2005; 28:409-415.

(237) Park MJ, Yun KE, Lee GE, Cho HJ, Park HS. A cross-sectional study of socioeconomic status and the metabolic syndrome in Korean adults. Ann Epidemiol 2007; 17:320-326.

(238) Park YW, Zhu S, Palaniappan L, Heshka S, Carnethon MR, Heymsfield SB. The metabolic syndrome: prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988-1994. Arch Intern Med 2003; 163:427-436.

(239) Park HS, Oh SW, Cho SI, Choi WH, Kim YS. The metabolic syndrome and associated lifestyle factors among South Korean adults. Int J Epidemiol 2004; 33:328-336.

(240) Paek KW, Chun KH, Jin KN, Lee KS. Do Health Behaviors Moderate the Effect of Socioeconomic Status on Metabolic Syndrome? Ann Epidemiol 2006; 16:756-762.

(241) Parker L, Lamont DW, Unwin N, Pearce MS, Bennett SMA, Dickinson HO et al. A lifecourse study of risk for hyperinsulinaemia, dyslipidaemia and obesity (the central metabolic syndrome) at age 49-51 years. Diabet Med 2003; 20:406-415.

(242) Prescott E, Godtfredsen N, Merete O, Schnohr P, Barefoot J. Social gradient in the metabolic syndrome not explained by psychosocial and behavioural factors: evidence from the Copenhagen City Heart Study. Eur J Cardiovasc Prev Rehab 2007; 14:405-412.

(243) Langenberg C, Kuh D, Wadsworth MEJ, Brunner E, Hardy R. Social Circumstances and Education: Life Course Origins of Social Inequalities in Metabolic Risk in a Prospective National Birth Cohort. Am J Public Health 2006; 96:2216-2221.

(244) Perel P, Langenberg C, Ferrie J, Moser K, Brunner E, Marmot M. Household Wealth and the Metabolic Syndrome in the Whitehall II Study. Diabetes Care 2006; 29:2694-2700.

(245) Bolton-Smith C, Smith WCS, Woodward M, Tunstall-Pedoe H. Nutrient intakes of different social-class groups: results from the Scottish Heart Health Study (SHHS). Br J Nutr 1991; 65:321-335.

(246) Fehily AM, Phillips KM, Yarnell JW. Diet, smoking, social class, and body mass index in the Caerphilly Heart Disease Study. Am J Clin Nutr 1984; 40:827-833.

(247) Rimm EB, Ascherio A, Giovannucci E, Spiegelman D, Stampfer MJ, Willett WC. Vegetable, fruit, and cereal fiber intake and risk of coronary heart disease among men. JAMA 1996; 275:447-451.

(248) Billson H, Pryer JA, Nichols R. Variation in fruit and vegetable consumption among adults in Britain. An analysis from the dietary and nutritional survey of British adults. Eur J Clin Nutr 1999; 53:946-952.

(249) Morgan M, Heller RF, Swerdlow A. Changes in diet and coronary heart disease mortality among social classes in Great Britain. J Epidemiol Community Health 1989; 43:162-167.

(250) Gregory J, Foster K, Tyler H, Wiseman M. The Dietary and Nutritional Survey of British Adults. London: H.M. Stationery Office; 1990.

(251) Braddon FE, Wadsworth ME, Davies JM, Cripps HA. Social and regional differences in food and alcohol consumption and their measurement in a national birth cohort. J Epidemiol Community Health 1988; 42:341-349.

(252) Dubios L, Girard M. Social position and nutrition: a gradient relationship in Canada and the USA. Eur J Clin Nutr 2007; 55:366-373.

(253) Ben-Shlomo Y, Kuh D. A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. Int J Epidemiol 2002; 31:285-293.

(254) Kuh D, Ben-Shlomo Y. Introduction. In: Kuh D, Ben-Shlomo Y, editors. A lifecourse approach to chronic disease epidemiology. 2nd ed. Oxford: Oxford University Press; 2004. 3-14.

(255) Elo IT, Preston SH. Effects of Early-Life Conditions on Adult Mortality: A Review. Popul Index 1992; 58:186-212.

(256) Barker DJ, Osmond C. Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. Lancet 1986; 1:1077-1081.

(257) Ben-Shlomo Y, Davey Smith G. Deprivation in infancy or in adult life: which is more important for mortality risk? Lancet 1991; 337:530-534.

(258) Strand BH, Kunst A. Childhood Socioeconomic Position and Cause-specific Mortality in Early Adulthood. Am J Epidemiol 2007; 165:85-93.

(259) Brunner E, Shipley MJ, Blane D, Davey Smith G, Marmot MG. When does cardiovascular risk start? Past and present socioeconomic circumstances and risk factors in adulthood. J Epidemiol Community Health 1999; 53:757-764.

(260) Kauhanen L, Lakka HM, Lynch JW, Kauhanen J. Social disadvantages in childhood and risk of all-cause death and cardiovascular disease in later life: a comparison of historical and retrospective childhood information. Int J Epidemiol 2006; 35:962-968.

(261) Lynch JW, Kaplan GA, Salonen JT. Why do poor people behave poorly? Variation in adult health behaviours and psychosocial characteristics by stages of the socioeconomic lifecourse. Soc Sci Med 1997; 44:809-819.

(262) Power C, Matthews S. Origins of health inequalities in a national population sample. Lancet 1997; 350:1584-1589.

(263) Loucks EB, Lynch JW, Pilote L, Fuhrer R, Almeida ND, Richard H et al. Life-Course Socioeconomic Position and Incidence of Coronary Heart Disease: The Framingham Offspring Study. Am J Epidemiol 2009; 169:829-836.

(264) Kivimaki M, Davey Smith G, Juonala M, Ferrie JE, Keltikangas-Jarvinen L, Elovainio M et al. Socioeconomic position in childhood and adult cardiovascular risk factors, vascular structure, and function: cardiovascular risk in young Finns study. Heart 2006; 92:474-480.

(265) Hardy R, Kuh D, Langenberg C, Wadsworth ME. Birthweight, childhood social class, and change in adult blood pressure in the 1946 British birth cohort. Lancet 2003; 362:1178-1183.

(266) Frankel S, Davey Smith G, Gunnell D. Childhood Socioeconomic Position and Adult Cardiovascular Mortality: The Boyd Orr Cohort. Am J Epidemiol 1999; 150:1081-1084.

(267) Galobardes B, Davey Smith G, Jeffreys M, McCarron P. Childhood socioeconomic circumstances predict specific causes of death in adulthood: the Glasgow student cohort study. J Epidemiol Community Health 2006; 60:527-529.

(268) Lawlor DA, Sterne JAC, Tynelius P, Davey Smith G, Rasmussen F. Association of Childhood Socioeconomic Position with Cause-specific Mortality in a Prospective Record Linkage Study of 1,839,384 Individuals. Am J Epidemiol 2006; 164:907-915.

(269) Naess O, Strand BH, Smith GD. Childhood and adulthood socioeconomic position across 20 causes of death: a prospective cohort study of 800 000 Norwegian men and women. J Epidemiol Community Health 2007; 61:1004-1009.

(270) Naess O, Claussen B, Davey Smith G. Relative impact of childhood and adulthood socioeconomic conditions on cause specific mortality in men. J Epidemiol Community Health 2004; 58:597-598.

(271) Batty GD, Lawlor DA, Macintyre S, Clark H, Leon DA. Accuracy of adults' recall of childhood social class: findings from the Aberdeen children of the 1950s study. J Epidemiol Community Health 2005; 59:898-903.

(272) Wannamethee SG, Whincup PH, Shaper G, Walker M. Influence of fathers' social class on cardiovascular disease in middle-aged men. Lancet 1996; 348:1259-1263.

(273) Jagger C, Matthews R, Melzer D, Matthews F, Brayne C, MRC CFAS. Educational differences in the dynamics of disability incidence, recovery and mortality: Findings from the MRC Cognitive Function and Ageing Study (MRC CFAS). Int J Epidemiol 2007; 36:358-365.

(274) Breeze E, Fletcher AE, Leon DA, Marmot MG, Clarke RJ, Shipley MJ. Do socioeconomic disadvantages persist into old age? Self-reported morbidity in a 29-year follow-up of the Whitehall Study. Am J Public Health 2001; 91:277-283.

(275) Adamson J, Hunt K, Ebrahim S. Socioeconomic position, occupational exposures, and gender: the relation with locomotor disability in early old age. J Epidemiol Community Health 2003; 57:453-455.

(276) Melzer D, Izmirlian G, Leveille SG, Guralnik JM. Educational Differences in the Prevalence of Mobility Disability in Old Age: The Dynamics of Incidence, Mortality, and Recovery. J Gerontol B Psychol Sci Soc Sci 2001; 56:S294-S301.

(277) Minkler M, Fuller-Thomson E, Guralnik JM. Gradient of Disability across the Socioeconomic Spectrum in the United States. N Engl J Med 2006; 355:695-703.

(278) Sacker A, Head J, Bartley M. Impact of Coronary Heart Disease on Health Functioning in an Aging Population: Are There Differences According to Socioeconomic Position? Psychosom Med 2008; 70:133-140.

(279) Matthews RJ, Smith LK, Hancock RM, Jagger C, Spiers NA. Socioeconomic factors associated with the onset of disability in older age: a longitudinal study of people aged 75 years and over. Soc Sci Med 2005; 61:1567-1575.

(280) Schoeni RF, Martin LG, Andreski PM, Freedman VA. Persistent and Growing Socioeconomic Disparities in Disability Among the Elderly: 1982-2002. Am J Public Health 2005; 95:2065-2070.

(281) Shaper AG, Pocock SJ, Walker M, Cohen NM, Wale CJ, Thomson AG. British Regional Heart Study: cardiovascular risk factors in middle-aged men in 24 towns. BMJ 1981; 283:179-186.

(282) Walker M, Whincup PH, Shaper AG. The British Regional Heart Study 1975-2004. Int J Epidemiol 2004; 33:1185-1192.

(283) Tunstall-Pedoe H, Kuulasmaa K, Amouyel P, Arveiler D, Rajakangas AM, Pajak A. Myocardial infarction and coronary deaths in the World Health Organization MONICA Project. Registration procedures, event rates, and case-fatality rates in 38 populations from 21 countries in four continents. Circulation 1994; 90:583-612.

(284) Nomenclature and criteria for diagnosis of ischemic heart disease. Report of the Joint International Society and Federation of Cardiology/World Health Organization task force on standardization of clinical nomenclature. Circulation 1979; 59:607-609.

(285) Wannamethee SG, Shaper AG. Lifelong teetotallers, ex-drinkers and drinkers: mortality and the incidence of major coronary heart disease events in middle-aged British men. Int J Epidemiol 1997; 26:523-531.

(286) Office of Population Censuses and Surveys.Social Survey Division. General Househol Survey 1978. London: HMSO; 1980.

(287) The World Health Organization MONICA Project (monitoring trends and determinants in cardiovascular disease): a major international collaboration. WHO MONICA Project Principal Investigators. J Clin Epidemiol 1988; 41:105-114.

(288) Bolton-Smith C, Casey CE, Gey KF, Smith WC, Tunstall-Pedoe H. Antioxidant vitamin intakes assessed using a food-frequency questionnaire: correlation with biochemical status in smokers and non-smokers. Br J Nutr 1991; 65:337-346.

(289) Holland B, Welch A, Unwin I, Buss D, Paul A, Southgate D. McCance and Widdowson's the Composition of Foods. 5th ed. Cambridge: Royal Society of Chemistry; 1991.

(290) Wannamethee SG, Shaper AG, Morris RW, Whincup PH. Measures of adiposity in the identification of metabolic abnormalities in elderly men. Am J Clin Nutr 2005; 81:1313-1321.

(291) Whincup PH, Bruce NG, Cook DG, Shaper AG. The Dinamap 1846SX automated blood pressure recorder: comparison with the Hawksley random zero sphygmomanometer under field conditions. J Epidemiol Community Health 1992; 46:164-169.

(292) Bruce NG, Cook DG, Shaper AG. Differences between observers in blood pressure measurement with an automatic oscillometric recorder. J Hypertens Suppl 1990; 4:S11-S13.

(293) Emberson J, Whincup PH, Walker M, Thomas M, Alberti KGMM. Biochemical measures in a population-based study: effect of fasting duration and time of day. Ann Clin Biochem 2002; 39:493-501.

(294) Siedel J, Hagele EO, Ziegenhorn J, Wahlefield AW. Reagent for the enzymatic determination of serum total with improved lipolytic efficiency. Clin Chem 1983; 29:1075-1080.

(295) Sugiuchi H, Uji Y, Okabe H, Irie T, Uekama K, Kayahara N et al. Direct measurement of high-density lipoprotein cholesterol in serum with polyethylene glycol-modified enzymes and sulfated alpha-cyclodextrin. Clin Chem 1995; 41:717-723.

(296) Friedewald WT, Levy.R.I., Fredrickson DS. Estimation of the concentration of low-density lipoprotien cholesterol in plasma, without use of the preparative centrifuge. Clin Chem 1972; 18:499-502.

(297) Trinder P. Determination of glucose in blood using glucose oxidase with an alternative oxygen acceptor. Ann Clin Biochem 1969; 6:24-27.

(298) Jennings PE, Chirico S, Jones AF, Lunec J, Barnett AH. Vitamin C metabolites and microangiopathy in diabetes mellitus. Diabetes Res 1987; 6:151-154.

(299) Rice-Evans CA, Diplock AT, Symons MCR. Techniques in free radical research. In: Burdon RH, Vanknippenberg PH, editors. Laboratory techniques in biochemistry and molecular biology. Amsterdam: Elsiever; 1991. 185-206.

(300) Classification of Occupations 1970. London: HM Stationary Office; 1970.

(301) Duffy SW, Rohan TE, Day NE. Misclassification in more than one factor in a case-control study: a combination of Mantel-Haenszel and maximum likelihood approaches. Stat Med 1989; 8:1529-1536.

(302) Classification of Occupations. Census 1931. London: His Majesty's Stationery Office; 1929.

(303) Walker M, Shaper AG, Cook DG. Non-participation and mortality in a prospective study of cardiovascular disease. J Epidemiol Community Health 1987; 41:295-299.

(304) Altman DG. Practical statistics for medical researchers. London: Chapman & Hall; 1991.

(305) Barber JA, Thompson SG. Analysis of cost data in randomized trials: an application of the non-parametric bootstrap. Stat Med 2000; 19:3219-3236.

(306) Efron B, Tibshirani RJ. An Introduction to the Bootstrap. London: Chapman and Hall; 1993.

(307) Mackenbach JP, Bos V, Andersen O, Cardano M, Costa G, Harding S et al. Widening socioeconomic inequalities in mortality in six Western European countries. Int J Epidemiol 2003; 32:830-837.

(308) Marang-van de Mheen P, Davey Smith G, Hart CL, Gunning-Schepers LJ. Socioeconomic differentials in mortality among men within Great Britain: time trends and contributory causes. J Epidemiol Community Health 1998; 52:214-218.

(309) Lampe FC, Morris RW, Walker M, Shaper AG, Whincup PH. Trends in rates of different forms of diagnosed coronary heart disease, 1978 to 2000: prospective, population based study of British men. BMJ 2005; 330:1046-1049.

(310) Diggle PJ, Heagerty P, Liang K-Y, Zeger SL. Analysis of longitudinal data. 2 ed. New York: Oxford University Press; 2002.

(311) Collett D. Modelling Survival Data in Medical Research. 2nd ed. London: Chapman & Hall; 2003.

(312) Avendano M, Kunst AE, van Lenthe F, Bos V, Costa G, Valkonen T et al. Trends in Socioeconomic Disparities in Stroke Mortality in Six European Countries between 1981-1985 and 1991-1995. Am J Epidemiol 2005; 161:52-61.

(313) Unal B, Critchley JA, Capewell S. Explaining the Decline in Coronary Heart Disease Mortality in England and Wales Between 1981 and 2000. Circulation 2004; 109:1101-1107.

(314) Age specific death rates per 100,000 population from CHD by sex, 1968 to 2005, United Kingdom. British Heart Foundation Statistics Website [2007 [cited 2007 Dec. 7]; Available from: URL:http://www.heartstats.org/temp/Tabsp1.4spweb07.xls

(315) Breeze E, Sloggett A, Fletcher A. Socioeconomic and demographic predictors of mortality and institutional residence among middle aged and older people: results from the Longitudinal Study. J Epidemiol Community Health 1999; 53:765-774.

(316) Graham H. Building an inter-disciplinary science of health inequalities: the example of lifecourse research. Soc Sci Med 2002; 55:2005-2016.

(317) Bowling A. Socioeconomic differentials in mortality among older people. J Epidemiol Community Health 2004; 58:438-440.

(318) Emberson JR, Shaper AG, Wannamethee SG, Morris RW, Whincup PH. Alcohol Intake in Middle Age and Risk of Cardiovascular Disease and Mortality: Accounting for Intake Variation over Time. Am J Epidemiol 2005; 161:856-863.

(319) Lantz PM, House JS, Lepkowski JM, Williams DR, Mero RP, Chen J. Socioeconomic Factors, Health Behaviors, and Mortality: Results From a Nationally Representative Prospective Study of US Adults. JAMA 1998; 279:1703-1708.

(320) Lowe GDO, Rumley A, Whincup PH, Danesh J. Hemostatic and rheological variables and risk of cardiovascular disease. Semin Vasc Med 2002; 2:429-440.

(321) Markowe HL, Marmot MG, Shipley MJ, Bulpitt CJ, Meade TW, Stirling Y et al. Fibrinogen: a possible link between social class and coronary heart disease. BMJ 1985; 291:1312-1314.

(322) Lindblad U, Langer RD, Wingard DL, Thomas RG, Barrett-Connor EL. Metabolic Syndrome and Ischemic Heart Disease in Elderly Men and Women. Am J Epidemiol 2001; 153:481-489.

(323) Pyorala M, Miettinen H, Halonen P, Laakso M, Pyorala K. Insulin Resistance Syndrome Predicts the Risk of Coronary Heart Disease and Stroke in Healthy Middle-Aged Men : The 22-Year Follow-Up Results of the Helsinki Policemen Study. Arterioscler Thromb Vasc Biol 2000; 20:538-544.

(324) Santos A, Ebrahim S, Barros H. Gender, socio-economic status and metabolic syndrome in middle-aged and old adults. BMC Public Health 2008; 8:62-69.

(325) Fehily A.M., Yarnell J.W.G., Sweetnam P.M., Elwood P.C. Diet and incident ischaemic heart disease: the Caerphilly Study. Br J Nutr 1993; 69:303-314.

(326) Esrey KL, Joseph L, Grover SA. Relationship between dietary intake and coronary heart disease mortality: Lipid Research Clinics Prevalence Follow-Up Study. J Clin Epidemiol 1996; 49:211-216.

(327) Erkkila A, de Mello VDF, Riserus U, Laaksonen DE. Dietary fatty acids and cardiovascular disease: An epidemiological approach. Prog Lipid Res 2008; 47:172-187.

(328) Kiechl S, Muigg A, Santer P, Mitterer M, Egger G, Oberhollenzer M et al. Poor Response to Activated Protein C as a Prominent Risk Predictor of Advanced Atherosclerosis and Arterial Disease. Circulation 1999; 99:614-619.

(329) Zahn R, Beeck H, Winkelmann BR, Seidl K, Schneider S, Hellstern P et al. Prospective cross-sectional study of haemostatic factors in patients with and without coronary artery disease. Blood Coagul Fibrinolysis 2002; 13:81-87.

(330) Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA 2001; 285:2486-2497.

(331) Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985; 28:412-419.

(332) Risérus U, Ärnlöv J, Berglund L. Long-Term Predictors of Insulin Resistance: Role of lifestyle and metabolic factors in middle-aged men. Diabetes Care 2007; 30:2928-2933.

(333) Power C, Atherton K, Strachan DP, Shepherd P, Fuller E, Davis A et al. Life-course influences on health in British adults: effects of socio-economic position in childhood and adulthood. Int J Epidemiol 2007; 36:532-539.

(334) McEntegart A, Capell HA, Creran D, Rumley A, Woodward M, Lowe GDO. Cardiovascular risk factors, including thrombotic variables, in a population with rheumatoid arthritis. Rheumatology 2001; 40:640-644.

(335) Wannamethee SG, Lowe GDO, Shaper AG, Rumley A, Lennon L, Whincup PH. The metabolic syndrome and insulin resistance: relationship to haemostatic and inflammatory markers in older non-diabetic men. Atherosclerosis 2005; 181:101-108.

(336) Patrick DL, Cheadle A, Thompson DC, Diehr P, Koepsell T, Kinne S. The validity of self-reported smoking: a review and meta-analysis. Am J Public Health 1994; 84:1086-1093.

(337) Emberson JR, Whincup PH, Morris RW, Walker M, Lowe GD, Rumley A. Extent of regression dilution for established and novel coronary risk factors: results from the British Regional Heart Study. Eur J Cardiovasc Prev Rehabil 2004; 11:125-134.

(338) Emberson JR. Within-person variation in coronary risk factors: implications for aetiology and prevention of coronary heart disease. PhD Thesis. University of London; 2004.

(339) Gaillard TR, Schuster DP, Bossetti BM, Green PA, Osei K. The impact of socioeconomic status on cardiovascular risk factors in African-Americans at high risk for type II diabetes. Implications for syndrome X. Diabetes Care 1997; 20:745-752.

(340) Yarnell J, Yu S, McCrum E, Arveiler D, Hass B, Dallongeville J et al. Education, socioeconomic and lifestyle factors, and risk of coronary heart disease: the PRIME Study. Int J Epidemiol 2005; 34:268-275.

(341) O'Reilly DSt, Upton MN, Caslake MJ, Robertson M, Norrie J, McConnachie A et al. Plasma C reactive protein concentration indicates a direct relation between systemic inflammation and social deprivation. Heart 2006; 92:533-535.

(342) de Maat MPM, Bladbjerg EM, von Bornemann Hjelmborg J, Bathum L, Jespersen J, Christensen K. Genetic Influence on Inflammation Variables in the Elderly. Arterioscler Thromb Vasc Biol 2004; 24:2168-2173.

(343) Perry IJ, Wannamethee SG, Walker MK, Thomson AG, Whincup PH, Shaper AG. Prospective study of risk factors for development of non-insulin dependent diabetes in middle aged British men. BMJ 1995; 310:560-564.

(344) Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet 2005; 365:1415-1428.

(345) Haffner MD, Miettinen MD. Insulin Resistance Implications for Type II Diabetes Mellitus and Coronary Heart Disease. Am J Med 1997; 103:152-162.

(346) Hayes L, Pearce MS, Unwin NC. Lifecourse predictors of normal metabolic parameters in overweight and obese adults. Int J Obes 2006; 30:970-976.

(347) Lawlor DA, Davey Smith G, Ebrahim S. Life Course Influences on Insulin Resistance: Findings from the British Women's Heart and Health Study. Diabetes Care 2003; 26:97-103.

(348) Lawlor DA, Ebrahim S, Davey Smith G. The metabolic syndrome and coronary heart disease in older women: findings from the British Women's Heart and Health Study. Diabet Med 2004; 21:906-913.

(349) Wannamethee SG, Shaper AG, Whincup PH, Walker M. Role of risk factors for major coronary heart disease events with increasing length of follow up. Heart 1999; 81:374-379.

(350) Cade J, Barker D, Margetts B, Morris J. Diet and inequalities in health in three English towns. BMJ 1988; 296:1359-1362.

(351) Morland K, Wing S, Roux AD. The Contextual Effect of the Local Food Environment on Residents' Diets: The Atherosclerosis Risk in Communities Study. Am J Public Health 2002; 92:1761-1768.

(352) Rose D, Richards R. Food store access and household fruit and vegetable use among participants in the US Food Stamp Program. Public Health Nutr 2004; 7:1081-1088.

(353) Baranowski T, Cullen KW, Baranowski J. Psychosocial correlates of dietary intake: Advancing Dietary Intervention. Annu Rev Nutr 1999; 19:17-40.

(354) Kromhout D, Bosschieter EB, de Lezenne CC. Dietary fibre and 10-year mortality from coronary heart disease, cancer, and all causes. The Zutphen study. Lancet 1982; 320:518-522.

(355) Kushi LH, Lew RA, Stare FJ, Ellison CR, el Lozy M, Bourke G et al. Diet and 20-year mortality from coronary heart disease. The Ireland-Boston Diet-Heart Study. N Engl J Med 1985; 312:811-818.

(356) Mozaffarian D, Kumanyika SK, Lemaitre RN, Olson JL, Burke GL, Siscovick DS. Cereal, Fruit, and Vegetable Fiber Intake and the Risk of Cardiovascular Disease in Elderly Individuals. JAMA 2003; 289:1659-1666.

(357) Pereira MA, O'Reilly E, Augustsson K, Fraser GE, Goldbourt U, Heitmann BL et al. Dietary Fiber and Risk of Coronary Heart Disease: A Pooled Analysis of Cohort Studies. Arch Intern Med 2004; 164:370-376.

(358) Streppel MT, Ocke MC, Boshuizen HC, Kok FJ, Kromhout D. Dietary fiber intake in relation to coronary heart disease and all-cause mortality over 40 y: the Zutphen Study. Am J Clin Nutr 2008; 88:1119-1125.

(359) Davey Smith G, Brunner E. Socio-economic differentials in health: the role of nutrition. Proc Nutr Soc 1997; 56:75-90.

(360) Singh-Manoux A, Nabi H, Shipley M, Gueguen A, Sabia S, Dugravot A et al. The role of conventional risk factors in explaining social inequalities in coronary heart

disease: the relative and absolute approaches to risk. Epidemiology 2008; 19:599-605.

(361) Strand BH, Tverdal A. Can cardiovascular risk factors and lifestyle explain the educational inequalities in mortality from ischaemic heart disease and from other heart diseases? 26 year follow up of 50 000 Norwegian men and women. J Epidemiol Community Health 2004; 58:705-709.

(362) Wannamethee SG, Shaper AG. Socioeconomic status within social class and mortality: a prospective study in middle-aged British men. Int J Epidemiol 1997; 26:532-541.

(363) Schoenfeld D. Partial residuals for the proportional hazards regression model. Biometrika 1982; 69:239-241.

(364) Breslow N, Day N. Statistical Methods in Cancer Research. Vol. 1 The Analysis of Case-Control Studies. Lyon: International Agency for Research on Cancer; 1980.

(365) Grundy E, Holt G. The socioeconomic status of older adults: How should we measure it in studies of health inequalities? J Epidemiol Community Health 2001; 55:895-904.

(366) Davey Smith G, Hart C, Hole D, MacKinnon P, Gillis C, Watt G et al. Education and occupational social class: which is the more important indicator of mortality risk? J Epidemiol Community Health 1998; 52:153-160.

(367) Steptoe A, Willemsen G, Owen N, Flower L, Mohamed-Ali V. Acute mental stress elicits delayed increases in circulating inflammatory cytokine levels. Clin Sci (Lond) 2001; 101:185-192.

(368) Lawlor DA, Davey Smith G, Rumley A, Lowe GDO, Ebrahim S. Associations of fibrinogen and C-reactive protein with prevalent and incident coronary heart disease are attenuated by adjustment for confounding factors British Women's Heart and Health Study. Thromb Haemost 2005; 93:955-963.

(369) Fredrikson GN, Hedblad B, Nilsson JA, Alm R, Berglund G, Nilsson J. Association between diet, lifestyle, metabolic cardiovascular risk factors, and plasma C-reactive protein levels. Metabolism 2004; 53:1436-1442.

(370) Gliksman MD, Kawachi I, Hunter D, Colditz GA, Manson JE, Stampfer MJ et al. Childhood socioeconomic status and risk of cardiovascular disease in middle aged US women: a prospective study. J Epidemiol Community Health 1995; 49:10-15.

(371) Tillin T, Chaturvedi N, Forouhi NG, Smith GD, McKeigue PM. Cardiovascular disease mortality in relation to childhood and adulthood socioeconomic markers in British South Asian men. Heart 2008; 94:476-481.

(372) Gillum RF, Paffenbarger RSJ. Chronic disease in former college students: XVII. Sociocultural mobility as a precursor of coronary heart disease and hyptertension. Am J Epidemiol 1978; 108:289-298.

(373) Vagero D, Leon D. Effect of social class in childhood and adulthood on adult mortality. Lancet 1994; 343:1224-1225.

(374) Bobák M, Hertzman C, Škodová Z, Marmot M. Own education, current conditions, parental material circumstances, and risk of myocardial infarction in a former communist country. J Epidemiol Community Health 2000; 54:91-96.

(375) Eriksson JG, Forsen T, Tuomilehto J, Winter PD, Osmond C, Barker DJP. Catch-up growth in childhood and death from coronary heart disease: longitudinal study. BMJ 1999; 318:427-431.

(376) van de Mheen H, Stronks K, Looman CWN, Mackenbach JP. Does childhood socioeconomic status influence adult health through behavioural factors? Int J Epidemiol 1998; 27:431-437.

(377) Power C, Graham H, Due P, Hallqvist J, Joung I, Kuh D et al. The contribution of childhood and adult socioeconomic position to adult obesity and smoking behaviour: an international comparison. Int J Epidemiol 2005; 34:335-344.

(378) Whincup P, Danesh J, Walker M, Lennon L, Thomson A, Appleby P et al. Prospective Study of Potentially Virulent Strains of Helicobacter pylori and Coronary Heart Disease in Middle-Aged Men. Circulation 2000; 101:1647-1652.

(379) Stuck AE, Walthert JM, Nikolaus T, Bula CJ, Hohmann C, Beck JC. Risk factors for functional status decline in community-living elderly people: a systematic literature review. Soc Sci Med 1999; 48:445-469.

(380) Nagi SZ. An Epidemiology of Disability among Adults in the United States. Milbank Q 1976; 54:439-467.

(381) Verbrugge LM, Jette AM. The disablement process. Soc Sci Med 1994; 38:1-14.

(382) Nagi SZ. An Epidemiology of Disability among Adults in the United States. Milbank Q 1976; 54:439-467.

(383) Guralnik JM, LaCroix AZ, Abbott RD, Berkman LF, Satterfield S, Evans DA et al. Maintaining Mobility in Late Life. I. Demographic Characteristics and Chronic Conditions. Am J Epidemiol 1993; 137:845-857.

(384) Jagger C, Matthews R, Matthews F, Robinson T, Robine JM, Brayne C. The burden of diseases on disability-free life expectancy in later life. J Gerontol A Biol Sci Med Sci 2007; 62:408-414.

(385) Murray CJ, Lopez AD. Regional patterns of disability-free life expectancy and disability-adjusted life expectancy: global Burden of Disease Study. Lancet 1997; 349:1347-1352.

(386) Ebrahim S, Wannamethee SG, Whincup P, Walker M, Shaper AG. Locomotor disability in a cohort of British men: the impact of lifestyle and disease. Int J Epidemiol 2000; 29:478-486.

(387) Ebrahim S, Wannamethee SG, Whincup P, Walker M, Shaper AG. Locomotor disability in a cohort of British men: the impact of lifestyle and disease. Int J Epidemiol 2000; 29:478-486.

(388) Stuck AE, Walthert JM, Nikolaus T, Bula CJ, Hohmann C, Beck JC. Risk factors for functional status decline in community-living elderly people: a systematic literature review. Soc Sci Med 1999; 48:445-469.

(389) Coppin AK, Ferrucci L, Lauretani F, Phillips C, Chang M, Bandinelli S et al. Low Socioeconomic Status and Disability in Old Age: Evidence From the InChianti Study for the Mediating Role of Physiological Impairments. J Gerontol A Biol Sci Med Sci 2006; 61:86-91.

(390) Rautio N, Heikkinen E, Heikkinen RL. The association of socio-economic factors with physical and mental capacity in elderly men and women. Arch Gerontol Geriatr 2001; 33:163-178.

(391) Schoeni RF, Martin LG, Andreski PM, Freedman VA. Persistent and Growing Socioeconomic Disparities in Disability Among the Elderly: 1982-2002. Am J Public Health 2005; 95:2065-2070.

(392) Koster A, Bosma H, Kempen GI, van Lenthe FJ, van Eijk JT, Mackenbach JP. Socioeconomic inequalities in mobility decline in chronic disease groups (asthma/COPD, heart disease, diabetes mellitus, low back pain): only a minor role for disease severity and comorbidity. J Epidemiol Community Health 2004; 58:862-869.

(393) Disability in America: Towards a national agenda for prevention. In: Pope AM, Tarlov AR, editors. Division of Health Promotion and Disease Prevention ed. Washington, D.C.: National Academy Press; 1991.

(394) Ebrahim S, Wannamethee SG, Whincup P, Walker M, Shaper AG. Locomotor disability in a cohort of British men: the impact of lifestyle and disease. Int J Epidemiol 2000; 29:478-486.

(395) Vita AJ, Terry RB, Hubert HB, Fries JF. Aging, Health Risks, and Cumulative Disability. N Engl J Med 1998; 338:1035-1041.

(396) Katz S, Ford AB, Moskowitz RW, Jackson BA, Jaffe MW. Studies of illness in the aged. JAMA 1963; 185:914-919.

(397) Lawton PM, Brody EM. Assessment of older people: self-maintaining and instrumental activities of daily living. Gerontologist 1969; 9:179-186.

(398) Fried LP, Bandeen-Roche K, Chaves PH, Johnson BA. Preclinical mobility disability predicts incident mobility disability in older women. J Gerontol A Biol Sci Med Sci 2000; 55:M43-M52.

(399) Ebrahim S, Wannamethee SG, Whincup P, Walker M, Shaper AG. Locomotor disability in a cohort of British men: the impact of lifestyle and disease. Int J Epidemiol 2000; 29:478-486.

(400) Vita AJ, Terry RB, Hubert HB, Fries JF. Aging, Health Risks, and Cumulative Disability. N Engl J Med 1998; 338:1035-1041.

(401) Walker MK, Whincup PH, Shaper AG, Lennon LT, Thomson AG. Validation of Patient Recall of Doctor-diagnosed Heart Attack and Stroke: A Postal Questionnaire and Record Review Comparison. Am J Epidemiol 1998; 148:355-361.

(402) Freedman VA, Martin LG, Schoeni RF. Recent Trends in Disability and Functioning Among Older Adults in the United States: A Systematic Review. JAMA 2002; 288:3137-3146.

(403) Schoeni RF, Martin LG, Andreski PM, Freedman VA. Persistent and Growing Socioeconomic Disparities in Disability Among the Elderly: 1982-2002. Am J Public Health 2005; 95:2065-2070.

(404) Rathouz PJ, Kasper JD, Zeger SL, Ferrucci L, Bandeen-Roche K, Miglioretti DL et al. Short-term Consistency in Self-reported Physical Functioning among Elderly Women: The Women's Health and Aging Study. Am J Epidemiol 1998; 147:764-773.

(405) Fried LP, Bandeen-Roche K, Chaves PH, Johnson BA. Preclinical mobility disability predicts incident mobility disability in older women. J Gerontol A Biol Sci Med Sci 2000; 55:M43-M52.

(406) Rautio N, Heikkinen E, Heikkinen RL. The association of socio-economic factors with physical and mental capacity in elderly men and women. Arch Gerontol Geriatr 2001; 33:163-178.

(407) Schoeni RF, Martin LG, Andreski PM, Freedman VA. Persistent and Growing Socioeconomic Disparities in Disability Among the Elderly: 1982-2002. Am J Public Health 2005; 95:2065-2070.

(408) Ebrahim S, Wannamethee SG, Whincup P, Walker M, Shaper AG. Locomotor disability in a cohort of British men: the impact of lifestyle and disease. Int J Epidemiol 2000; 29:478-486.

(409) Vita AJ, Terry RB, Hubert HB, Fries JF. Aging, Health Risks, and Cumulative Disability. N Engl J Med 1998; 338:1035-1041.

(410) Morris RW, Whincup PH, Papacosta O, Walker M, Thomson A. Inequalities in coronary revascularisation during the 1990s: evidence from the British regional heart study. Heart 2005; 91:635-640.

(411) Robert SA. Socioeconomic Position and Health: The Independent Contribution of Community Socioeconomic Context. Annu Rev Sociol 1999; 25:489-516.

(412) Kennedy J, Minkler M. Disability theory and public policy: Implications for critical gerontology. In: Krieger N, editor. Embodying Inequality. Epidemiologic Perspectives. First ed. New York: Baywood Publishing Company, Inc.; 2005. 273-292.

(413) Department of Health. Spending Review 2002 Public Service Agreement. Department of Health; 2002.

(414) Low A, Low A. Importance of relative measures in policy on health inequalities. BMJ 2006; 332:967-969.

(415) Shaw M, Davey Smith G, Dorling D. Health inequalities under New Labour: Authors' reply. BMJ 2005; 330:1507-150b.

(416) National Institute for Health and Clinical Excellence. Physical activity and the environment. London: 2008.

(417) National Institute for Health and Clinical Excellence. Four commonly used methods to increase physical activity. London: 2006.

(418) National Institute for Health and Clinical Excellence. Reducing the rate of premature deaths from cardiovascular disease and other smoking-related diseases: finding and supporting those most at risk and improving access to services. London: 2008.

(419) Whitehead M, Petticrew M, Graham H, Macintyre SJ, Bambra C, Egan M. Evidence for public health policy on inequalities: 2: Assembling the evidence jigsaw. J Epidemiol Community Health 2004; 58:817-821.

(420) Department of Health. Choosing health - Making healthy choices easier. London: The Stationary Office; 2004.

(421) Department of Health. Health Inequalities: Progress and Next Steps. London: Department of Health Publications; 2008.

(422) Department of Health. Delivering choosing health: making healthier choices easier. London: Department of Health Publications; 2005.

(423) Department of Health. National Service Framework for Coronary Heart Disease. London: 2000.

(424) Department of Health. National Service Framework for Older People. London: Department of Health Publications; 2001.

(425) Sorensen G, Barbeau E, Hunt MK, Emmons K. Reducing social disparities in tobacco use: a social-contextual model for reducing tobacco use among blue-collar workers. Am J Public Health 2004; 94:230-239.

(426) Hopkins DP, Briss PA, Ricard CJ, Husten CG, Carande-Kulis VG, Fielding JE et al. Reviews of evidence regarding interventions to reduce tobacco use and exposure to environmental tobacco smoke. Am J Prev Med 2001; 20:16-66.

(427) Jha P, Chaloupka FJ. Curbing the Epidemic: Government and the economics of tobacco control. Washington DC, The World Bank; 1999.

(428) Farrelly MC, Bray JW, Office on Smoking and Health (CDC). Response to increases in cigarette prices by race/ethnicity, income, and age groups--United States, 1976-1993. Morb Mort Wkly Rep 1998; 47:605-609.

(429) Townsend J, Roderick P, Cooper J. Cigarette smoking by socioeconomic group, sex, and age: effects of price, income, and health publicity. BMJ 1994; 309:923.

(430) Hill SE, Blakeley TA, Fawcett JM, Howden-Chapman P. Could mainstream anti-smoking programs increase inequalities in tobacco use? New Zealand data from 1981-96. Aust N Z J Public Health 2005; 29:279-284.

(431) Wilson N, Blakely T, Tobias M. What potential has tobacco control for reducing health inequalities? The New Zealand situation. Int J Equity Health 2006; 5:14.

(432) Wilson N, Thomson G. Tobacco taxation and public health: ethical problems, policy responses. Soc Sci Med 2005; 61:649-659.

(433) Department of Health. NHS Stop Smoking Services: service and monitoring guidance, October 2007/08. London: Department of Health Publications; 2007.

(434) Janzon E, Engstrom G, Lindstrom M, Berglund G, Hedblad B, Janzon L. Who are the "quitters"? a cross-sectional study of circumstances associated with women giving up smoking. Scand J Public Health 2005; 33:175-182.

(435) Schaap MM, Kunst AE, Leinsalu M, Regidor E, Ekholm O, Dzurova D et al. Effect of nationwide tobacco control policies on smoking cessation in high and low educated groups in 18 European countries. Tob Control 2008; 17:248-255.

(436) Bartley M, Fitzpatrick R, Firth D, Marmot M. Social distribution of cardiovascular disease risk factors: change among men in England 1984-1993. J Epidemiol Community Health 2000; 54:806-814.

(437) Giskes K, Kunst AE, Benach J, Borrell C, Costa G, Dahl E et al. Trends in smoking behaviour between 1985 and 2000 in nine European countries by education. J Epidemiol Community Health 2005; 59:395-401.

(438) Kahn EB, Ramsey LT, Brownson RC, Heath GW, Howze EH, Powell KE et al. The effectiveness of interventions to increase physical activity: A systematic review and. Am J Prev Med 2002; 22:73-107.

(439) Dattilo AM, Kris-Etherton PM. Effects of weight reduction on blood lipids and lipoproteins: a meta- analysis. Am J Clin Nutr 1992; 56:320-328.

(440) MacMahon S, Cutler J, Brittain E, Higgins M. Obesity and hypertension: epidemiological and clinical issues. Eur Heart J 1987; 8 Suppl B:57-70.

(441) National Task Force on the Prevention and Treatment of Obesity. Overweight, Obesity, and Health Risk. Arch Intern Med 2000; 160:898-904.

(442) Pi-Sunyer FX. Short-Term Medical Benefits and Adverse Effects of Weight Loss. Ann Intern Med 1993; 119:722-726.

(443) Pi-Sunyer FX. A review of long-term studies evaluating the efficacy of weight loss in ameliorating disorders associated with obesity. Clin Ther 1996; 18:1006-1035.

(444) Esposito K, Pontillo A, Di Palo C, Giugliano G, Masella M, Marfella R et al. Effect of Weight Loss and Lifestyle Changes on Vascular Inflammatory Markers in Obese Women: A Randomized Trial. JAMA 2003; 289:1799-1804.

(445) Kelley DE, Kuller LH, McKolanis TM, Harper P, Mancino J, Kalhan S. Effects of Moderate Weight Loss and Orlistat on Insulin Resistance, Regional Adiposity, and Fatty Acids in Type 2 Diabetes. Diabetes Care 2004; 27:33-40.

(446) Orchard TJ, Temprosa M, Goldberg R, Haffner S, Ratner R, Marcovina S et al. The effect of metformin and intensive lifestyle intervention on the metabolic syndrome: the Diabetes Prevention Program randomized trial. Ann Intern Med 2005; 142:611-619.

(447) Wilson PW, Kannel WB, Silbershatz H, D'Agostino RB. Clustering of metabolic factors and coronary heart disease. Arch Intern Med 1999; 159:1104-1109.

(448) Corrigan M, Cupples ME, Stevenson M. Quitting and restarting smoking: cohort study of patients with angina in primary care. BMJ 2002; 324:1016-1017.

(449) Cupples ME, McKnight A. Randomised controlled trial of health promotion in general practice for patients at high cardiovascular risk. BMJ 1994; 309:993-996.

(450) Tang JL, Armitage JM, Lancaster T, Silagy CA, Fowler GH, Neil HA. Systematic review of dietary intervention trials to lower blood total cholesterol in free-living subjects. BMJ 1998; 316:1213-1220.

(451) Ebrahim S, Smith GD. Systematic review of randomised controlled trials of multiple risk factor interventions for preventing coronary heart disease. BMJ 1997; 314:1666.

(452) Muir J, Mant D, Jones L, Yudkin P. Effectiveness of health checks conducted by nurses in primary care: results of the OXCHECK study after one year. BMJ 1994; 308:308-312.

(453) Wood DA, Kinmonth AL, Davies GA, Yarwood J, Thompson SG, Pyke SDM et al. Randomised controlled trial evaluating cardiovascular screening and intervention in general practice: principal results of British family heart study. BMJ 1994; 308:313-320.

(454) Adams N, Johnson G, Matejic P, Murray C, Toufexis N, Whatley J. Households Below Average Income An analysis of the income distribution 1994/95 – 2006/07. Department of Work and Pensions; 2008.

(455) Palmer G, MacInnes T, Kenway P. Monitoring poverty and social exclusion 2007. York: Joseph Rowntree Foundation; 2007.

(456) Carroll D, Bennett P, Davey Smith G. Socio-economic health inequalities: Their origins and implications. Psychol Health 1993; 8:295-316.

(457) National Institute for Health and Clinical Excellence. Behaviour change. London: 2007.

(458) Chassin L, Presson CC, Sherman SJ, Edwards DA. The natural history of cigarette smoking: predicting young-adult smoking outcomes from adolescent smoking patterns. Health Psychol 1990; 9:701-716.

(459) Janson H. Longitudinal patterns of tobacco smoking from childhood to middle age. Addictive Behaviors 1999; 24:239-249.

(460) Kunst A, Giskes K, Mackenbach J, EU Network on Interventions to Reduce Socio-economic Inequalities in Health. Socio-economic inequalities in smoking in the European Union. Applying an equity lens to tobacco control policies. 2004.

(461) Lovato C, Linn G, Stead LF. Impact of tobacco advertising and promotion on increasing adolescent smoking behaviours. Cochrane Database of Systematic Reviews 2003; 4:CD003439.

(462) Sowden AJ, Arblaster L. Mass media interventions for preventing smoking in young people. Cochrane Database of Systematic Reviews 1998; 4:CD001006.

(463) Stamatakis E, Primatesta P, Chinn S, Rona R, Falascheti E. Overweight and obesity trends from 1974 to 2003 in English children: what is the role of socioeconomic factors? Arch Dis Child 2005; 90:999-1004.

(464) Brodersen NH, Steptoe A, Boniface DR, Wardle J, Hillsdon M. Trends in physical activity and sedentary behaviour in adolescence: ethnic and socioeconomic differences. Br J Sports Med 2007; 41:140-144.

(465) Nagi SZ. An Epidemiology of Disability among Adults in the United States. Milbank Q 1976; 54:439-467.

(466) World Health Organisation. International Classification of Impairments, Disabilities, and Handicaps. Geneva: World Health Organisation; 1980.

(467) Krieger N. Introduction: Embodiment, Inequality and Epidemiology: What are the Connections? In: Krieger N, editor. Embodying Inequality. Epidemiologic perspectives. First ed. New York: Baywood Publishing Company, Inc.; 2005. 1-10.

(468) Wannamethee SG, Ebrahim S, Papacosta O, Shaper AG. From a postal questionnaire of older men, healthy lifestyle factors reduced the onset of and may have increased recovery from mobility limitation. J Clin Epidemiol 2005; 58:831-840.

(469) Wannamethee SG, Ebrahim S, Papacosta O, Shaper AG. From a postal questionnaire of older men, healthy lifestyle factors reduced the onset of and may have increased recovery from mobility limitation. J Clin Epidemiol 2005; 58:831-840.

(470) Patla AE, Shumway-Cook A. Dimensions of mobility: Defining the complexity and difficulty associated with community mobility. J Aging Phys Act 1999; 7:7-19.

(471) Gill TM, Baker DI, Gottschalk M, Peduzzi PN, Allore H, Byers A. A Program to Prevent Functional Decline in Physically Frail, Elderly Persons Who Live at Home. N Engl J Med 2002; 347:1068-1074.

(472) Stuck AE, Egger M, Hammer A, Minder CE, Beck JC. Home Visits to Prevent Nursing Home Admission and Functional Decline in Elderly People: Systematic Review and Meta-regression Analysis. JAMA 2002; 287:1022-1028.

(473) Hardoon SL, Whincup PH, Lennon LT, Wannamethee SG, Capewell S, Morris RW. How much of the recent decline in the incidence of myocardial infarction in British men can be explained by changes in cardiovascular risk factors? Evidence from a prospective population-based study. Circulation 2008; 117:598-604.

(474) Rockwood K, Stolee P, Robertson D, Shillington ER. Response Bias in a Health Status Survey of Elderly People. Age Ageing 1989; 18:177-182.

(475) Hoeymans N, Feskens EJM, van Den Bos GAM, Kromhout D. Non-response bias in a study of cardiovascular diseases, functional status and self-rated health among elderly men. Age Ageing 1998; 27:35-40.

(476) McKeigue PM, Miller GJ, Marmot MG. Coronary heart disease in South Asians overseas: A review. J Clin Epidemiol 1989; 42:597-609.

(477) Forouhi N, Sattar N, Tillin T, McKeigue P, Chaturvedi N. Do known risk factors explain the higher coronary heart disease mortality in South Asian compared with European men? Prospective follow-up of the Southall and Brent studies, UK. Diabetologia 2006; 49:2580-2588.

(478) Libertos P, Link BG, Kelsey JL. The measurement of social class in epidemiology. Epidemiol Rev 1988; 10:87-121.

(479) Stewart J, in collaboration with the Social Environment working group. Economic Status. John D and Catherine T MacArthur Research Network on Socioeconomic Status and Health [2002 [cited 2008 Nov. 7]; Available from: URL:http://www.macses.ucsf.edu/Research/Social%20Environment/notebook/economic.html

(480) McDonough P, Duncan GJ, Williams D, House J. Income dynamics and adult mortality in the United States, 1972 through 1989. Am J Public Health 1997; 87:1476-1483.

(481) Banks J, Karlsen S, Oldfield Z. Socio-economic position. In: Marmot M, Banks J, Blundell R, Lessof C, Nazroo J, editors. Health, wealth and lifestyles of the older population in England: The 2002 English Longitudinal Study Of Ageing. London: Institute of Fiscal Studies; 2003.

(482) Davey Smith G, Phillips A. Declaring independence: why we should be cautious. J Epidemiol Community Health 1990; 44:257-258.

(483) Salomaa V, Miettinen H, Niemela M, Ketonen M, Mahonen M, Immonen-Raiha P et al. Relation of socioeconomic position to the case fatality, prognosis and treatment of myocardial infarction events; the FINMONICA MI Register Study. J Epidemiol Community Health 2001; 55:475-482.

(484) Lang T, Ducimetiere P, Arveiler D, Amouyel P, Cambou JP, Ruidavets JB et al. Incidence, case fatality, risk factors of acute coronary heart disease and occupational categories in men aged 30-59 in France. Int J Epidemiol 1997; 26:47-57.

(485) Morrison C, Woodward M, Leslie W, Tunstall-Pedoe H. Effect of socioeconomic group on incidence of, management of, and survival after myocardial infarction and coronary death: analysis of community coronary event register. BMJ 1997; 314:541.

(486) Diez Roux AV, Merkin SS, Arnett D, Chambless L, Massing M, Nieto FJ et al. Neighborhood of residence and incidence of coronary heart disease. N Engl J Med 2001; 345:99-106.

(487) Eames M, Ben-Shlomo Y, Marmot MG. Social deprivation and premature mortality: regional comparison across England. BMJ 1993; 307:1097-1102.

(488) Steenland K, Henley J, Calle E, Thun M. Individual- and Area-Level Socioeconomic Status Variables as Predictors of Mortality in a Cohort of 179,383 Persons. Am J Epidemiol 2004; 159:1047-1056.

(489) Sundquist K, Winkleby M, Ahlen H, Johansson SE. Neighborhood Socioeconomic Environment and Incidence of Coronary Heart Disease: A Follow-up Study of 25,319 Women and Men in Sweden. Am J Epidemiol 2004; 159:655-662.

(490) Bosma H, Van Jaarsveld CHM, Tuinstra J, Sanderman R, Ranchor AV, Van Eijk JT et al. Low control beliefs, classical coronary risk factors, and socio-economic differences in heart disease in older persons. Soc Sci Med 2005; 60:737-745.

(491) Strike PC, Steptoe A. Psychosocial factors in the development of coronary artery disease. Prog Cardiovasc Dis 2004; 46:337-347.

(492) Wamala SP, Mittleman MA, Schenck-Gustafsson K, Orth-Gomer K. Potential explanations for the educational gradient in coronary heart disease: a population-based case-control study of Swedish women. Am J Public Health 1999; 89:315-321.

(493) Eng PM, Rimm EB, Fitzmaurice G, Kawachi I. Social Ties and Change in Social Ties in Relation to Subsequent Total and Cause-specific Mortality and Coronary Heart Disease Incidence in Men. Am J Epidemiol 2002; 155:700-709.

(494) Rosengren A, Orth-Gomer K, Wilhelmsen L. Socioeconomic differences in health indices, social networks and mortality among Swedish men. A study of men born in 1933. Scand J Public Health 1998; 26:272-280.

(495) Kuper H, Marmot M, Hemingway H. Systematic Review of Prospective Cohort Studies of Psychosocial Factors in the Etiology and Prognosis of Coronary Heart Disease. Sem Vasc Med 2002; 2:267-314.

(496) Mookadam F, Arthur HM. Social Support and Its Relationship to Morbidity and Mortality After Acute Myocardial Infarction: Systematic Overview. Arch Intern Med 2004; 164:1514-1518.

(497) Seeman TE. Social ties and health: The benefits of social integration. Ann Epidemiol 1996; 6:442-451.

(498) Barefoot JC, Gronbaek M, Jensen G, Schnohr P, Prescott E. Social Network Diversity and Risks of Ischemic Heart Disease and Total Mortality: Findings from the Copenhagen City Heart Study. Am J Epidemiol 2005; 161:960-967.

(499) Burg MM, Barefoot J, Berkman L, Catellier DJ, Czajkowski S, Saab P et al. Low Perceived Social Support and Post-Myocardial Infarction Prognosis in the Enhancing Recovery in Coronary Heart Disease Clinical Trial: The Effects of Treatment. Psychosom Med 2005; 67:879-888.

(500) Kaplan GA, Salonen JT, Cohen RD, Brand RJ, Syme SL, Puska P. Social Connections And Mortality From All Causes And From Cardiovascular Disease: Prospective Evidence From Eastern Finland. Am J Epidemiol 1998; 128:370-380.

(501) Ford ES, Ahluwalia IB, Galuska DA. Social Relationships and Cardiovascular Disease Risk Factors: Findings from the Third National Health and Nutrition Examination Survey. Prev Med 2000; 30:83-92.

(502) Kuh D, Wadsworth M. Parental Height: Childhood Environment and Subsequent Adult Height in a National Birth Cohort. Int J Epidemiol 1989; 18:663-668.

(503) Power C, Manor O, Matthews S. The duration and timing of exposure: effects of socioeconomic environment on adult health. Am J Public Health 1999; 89:1059-1065.

(504) Smith K, Joshi H. The Millennium Cohort Study. Popul Trends 2002; 107:30-34.

(505) Poulton R, Caspi A, Milne BJ, Thomson WM, Taylor A, Sears MR et al. Association between children's experience of socioeconomic disadvantage and adult health: a life-course study. Lancet 2002; 360:1640-1645.

(506) Lawlor DA, Davey Smith G. Early life determinants of adult blood pressure. Curr Opin Nephrol Hypertens 2005; 14:259-264.

(507) Kivimäki M, Lawlor DA, Smith GD, Keltikangas-Järvinen L, Elovainio M, Vahtera J et al. Early Socioeconomic Position and Blood Pressure in Childhood and Adulthood: The Cardiovascular Risk in Young Finns Study. Hypertension 2006; 47:39-44.

(508) Whincup PH, Cook DG, Geleijnse JM. A life course approach to blood pressure. In: Kuh D, Ben-Shlomo Y, editors. A life course approach to chronic disease epidemiology. Second ed. New York: Oxford University Press; 2004. 218-239.

(509) Gillman MW. A life course approach to obesity. In: Kuh D, Ben-Shlomo Y, editors. A life course approach to chronic disease epidemiology. Second ed. New York: Oxford University Press; 2004. 189-217.

(510) Department of Health. Improvement, expansion and reform: The next three years. Priorities and planning framework 2003-2006. 2002.

(511) Orleans CT. Promoting the maintenance of health behavior change: recommendations for the next generation of research and practice. Health Psychol 2000; 19:76-83.