Council Tax Valuation Banding as a surrogate marker of Socioeconomic Position in the Primary and Secondary Prevention of Coronary Heart Disease

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SUMMARY

Coronary Heart Disease (CHD) is the commonest cause of premature death in men and women in Scotland. Research has suggested that a significant proportion of incident CHD is attributable to modifiable risk factors such as level of physical activity, diet and smoking. This recognition that CHD is a largely preventable disease has focused health policy, both in the UK and elsewhere, on prevention strategies.

There is well established evidence of a socioeconomic gradient in CHD; where those of lowest socioeconomic position (SEP) experience the highest CHD burden and greatest exposure to cardiovascular risk factors. This presents distinct challenges for effective primary prevention (defined as the prevention of new-onset CHD) and secondary prevention (defined as the prevention of recurrent coronary events in patients with established CHD) of the disease. A key consideration in the implementation of CHD preventative strategies is thus the measure of SEP used in the allocation of preventative resources.

This study will investigate the predictive validity of Council Tax Valuation Banding (CTVB) in identifying high-risk sub-groups within both CHD primary and secondary prevention populations. CTVB is worthy of consideration as a marker of SEP in this context as it appears to have several appealing characteristics appropriate for use in CHD prevention. CTVB is based on the property value; theoretically reflecting both individual material circumstance and to an extent geographical area characteristics. Furthermore CTVB is objective, uncomplicated, universally available and sensitive to the household level. This study originated from an interest in developing practical and applicable methods of identifying highest risk individuals within CHD prevention populations. Gaps in existing research support a need for this.

Firstly a cohort of just under 2,000 men and women, aged between 45-60 years who participated in the Have a Heart Paisley (HaHP) CHD Primary Prevention Programme was examined. These individuals were enrolled in 2006 and underwent comprehensive cardiovascular risk screening. Secondly, in 2009, the HaHP Chronic Disease Register (CDR) was used to pool Secondary Prevention primary care data for just over 3,000 men and women, of all ages with established CHD.

Socioeconomic patterning of risk factors and absolute risk was examined in the primary prevention population. Socioeconomic inequalities were examined in risk-factor monitoring and therapies prescribing in the secondary prevention population. SEP for analyses in both populations was measured using the Scottish Index of Multiple Deprivation (SIMD) and CTVB- which was supplied by the

Renfrewshire Joint Valuation Board. Both measures of SEP were linked to these data using address information and postcodes.

The findings of this study demonstrate some potential for the use of CTVB as a surrogate marker of SEP in health research and cardiovascular preventative strategies. But that further research on this matter is required. CTVB showed significant association with few classical cardiovascular risk factors in the primary prevention population; body mass index in females, high-density lipoproteins (HDL) cholesterol in females, and rates of current smokers in both males and females (age and age-squared adjusted). However all associations with the exception of rates of current smokers (both males and females) became insignificant when SIMD was added into the statistical modelling. CTVB displayed association with Framingham risk scores in both men and women (age and age-squared adjusted) however added independent predictive power in men only.

The associations between SEP (as measured by CTVB) and classical risk factors in the present study are generally weaker than the literature reviewed using established measures of SEP. Particularly striking is the insignificant socioeconomic variance in blood pressure levels when using CTVB, which is at odds with the overwhelming majority of literature in this field to date. Aside from the CTVB analyses, in general the analysis undertaken adds to existing literature; re-enforcing the existence of socioeconomic inequalities in classical risk factors and absolute risk in an asymptomatic population.

When examining the secondary prevention population, significant socioeconomic (using CTVB as a measure of SEP) variance was identified in risk-factor monitoring and in some therapies prescribing. The analyses demonstrates that the removal of "exception reporting" from the Quality Outcomes Framework (QOF) records reveals some important inequalities in care and treatment within an established CHD population. The analyses did demonstrate that overall rates of risk factor monitoring and therapies prescribing have risen markedly over the past decade, especially post introduction of the QOF. These findings have important implications for the delivery of the QOF in Scotland and for Secondary Prevention of CHD in general.

Considerable methodological difficulty was encountered when using CTVB as a surrogate marker of SEP. Data linkage based on address and postcode data proved problematic, notable proportions within each population required matching "by hand" which proved time consuming. Furthermore use of CTVB in this study identified significant potential to misclassify the SEP of individuals who are renting properties; particularly homes of multiple occupation. Additionally the marked rise in housing price over the past two decades in the UK may further compromise CTVB's accuracy as a measure of SEP.

Such practical and theoretical limitations of the use of CTVB as a marker of SEP have not been reported in the literature to date. This supports the conclusions of the literature review within the present study which question the quality and scientific objectivity of studies examining CTVB as a marker of SEP undertaken thus far.

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AUTHOR'S DECLERATION

I declare that, except where explicit reference has been made to the contributions of others that this thesis is as a result of my own work and has not been submitted for any other degree at the University of Glasgow or any other institution.

Christopher Harkins.

CHAPTER 1: INTRODUCTION

1.1 Introduction to CHD and rationale of the study

Coronary Heart Disease (CHD) is the commonest cause of premature death in men and women in Scotland[1]. Research has suggested that a significant proportion of incident CHD is attributable to modifiable risk factors such as level of physical activity, diet and smoking[2; 3]. This recognition that CHD is a largely preventable disease has focused health policy, both in the UK and elsewhere, on prevention strategies[4].

Whilst largely preventable, CHD is a complex disease; its development is influenced by a variety of associated factors beyond that of exposure to risk factors. The development of CHD has been shown to be affected by social and environmental, as well as political and economic, factors[5].

As will be outlined later in this thesis, the evidence base for primary prevention (defined as the prevention of new-onset CHD) is weak. This is in contrast to that of secondary prevention (defined as the prevention of recurrent coronary events in patients with established CHD). However there is strong political support for both primary and secondary CHD prevention strategies. There is well established evidence of a socioeconomic gradient in CHD[6]; which presents distinct challenges for effective primary and secondary prevention of the disease. Moreover, beyond CHD, there is a lack of reliable evidence in relation to designing effective programmes to reduce health inequalities in general[7].

Despite emerging evidence in relation to novel risk factors for CHD[8], there remains a focus on reducing exposure to classical cardiovascular risk factors (behavioural and physiological) in areas of socioeconomic disadvantage[9]. A key consideration there in is the measure of socioeconomic position (SEP) [10] used to define such areas or identify high risk individuals when distributing preventative resources. Logically individual measures of SEP may appear more sensitive than area based measures as not all individuals of low SEP reside in areas of socioeconomic disadvantage[11]. However the use of individual measures of SEP is at odds with evidence which supports area contextual influences on health[12; 13] and the development of CHD[14].

1.2 Introduction to the study and organisation of the thesis

This study originated from an interest in developing practical and applicable methods of identifying individuals at highest risk within CHD primary and secondary prevention populations. Gaps in existing research support a need for this. Political will exists to address inequalities in health and a drive towards service redesign which shifts delivery to early intervention and prevention and away from treatment and emergency management. Despite these widely held ideals, many barriers remain for the complete acceptance of these principles within the treatment and prevention of CHD; principally treatment budgets greatly outweigh those of prevention. This balance is unlikely to shift in the near future considering the inherent difficulties in generating reliable scientific evidence in relation to effectiveness of CHD primary prevention. A particular aspect of primary prevention with a distinct paucity of evidence concerns the methods of targeting and indeed engaging high risk individuals or populations; although recent studies in this area are beginning to emerge [15;16]. In maximising CHD primary prevention resources it is generally recognised that programmes should positively discriminate resource allocation in favour of individuals and or households of highest risk.

Within secondary prevention there is a need to continue to monitor the quality and equity of care and treatment delivered within primary care to patients with established CHD. The General Medical Service (GMS) Quality Outcomes Framework (QOF) was introduced to universally improve the quality of care for CHD patients. Despite this there is evidence that inequalities in care and treatment continue to exist within secondary prevention. Thus it is important to characterise sub-groups of the CHD population for whom the introduction of the QOF has failed to ensure equitable care and treatment.

In improving equity in both the primary and secondary prevention of CHD, the measure of SEP adopted is a key consideration. In both instances the more sensitive the measure of SEP (in terms of its association to/with cardiovascular risk in asymptomatic populations and with levels of care and treatment for populations with established CHD) the greater the efficiency as to how CHD preventative resources can be utilised. As mentioned previously it would appear that individual measures of SEP have intrinsic advantage[17] over area based measures. However this is problematic firstly because individual level markers of SEP are not routinely available for entire populations (particularly in asymptomatic populations). Furthermore, as stated, such an approach ignores the evidenced influence of neighbourhood or area of residence characteristics on health.

This study will investigate the use of Council Tax Valuation Banding (CTVB) as a surrogate marker of SEP in CHD prevention. CTVB is worthy of consideration in this context because it is sensitive to the household level; every household in the UK, irrespective of tenure has a council tax band. CTVB is based on the market value of property which reflects, to an extent, the areas socioeconomic characteristics. Intuitively CTVB has inherent properties which relate to SEP- property value is associated to some degree with individual or household income, which is one of the most accurate measures of SEP within cardiovascular research and beyond

The utility of this study could be questioned given the quality of national health information collection in Scotland is high compared to other countries and SIMD is an established and validated national measure of SEP. The drive to 'improve' on the use of SIMD is born purely out of a desire to impact on Scotland's widening inequalities in CHD.

Using data from the Have a Heart Paisley (HaHP) Chronic Disease Register (CDR) the aim of this study is:

To assess the predictive validity of CTVB in identifying elevated risk within a primary prevention population and sub-optimal care and treatment within a secondary prevention population.

To address this aim, the specific objectives of the thesis are:

1. To examine the distribution of cardiovascular risk factors and Framingham absolute risk according to CTVB in an asymptomatic primary prevention population (men and women, aged 45-60) in Paisley, Scotland in 2006 and to establish the predicative validity of CTVB with these variables.

2. To explore the rates of risk factor monitoring and secondary prevention therapies prescribing according to CTVB in an established CHD population (men and women with CHD of all ages in Paisley, Scotland) in 2009 and to establish the predicative validity of CTVB with these treatment and care outcome variables.

As a precursor to these analyses the association between CTVB and SIMD, a validated national marker of SEP will be investigated.

The thesis is structured in the following way:

Chapter 1 provides an introduction, to the rationale and the aims and objectives of the study and also provides a guide as to the layout of the thesis.

Chapter 2 begins with a detailed account of the literature search strategy and literature inclusion criteria. The chapter then presents a literature review as a background to the thesis, including an introduction to the threads of research of which the thesis attempts to synthesise; CHD and atherosclerosis, global, European and UK perspectives of CHD and an introduction to CHD prevention strategies, this section of the chapter concludes with an introduction to some key considerations in addressing health inequalities.

The chapter continues with an in-depth review of different measures of SEP in health research including perspectives on the contextual and compositional influences on health in general and cardiovascular disease specifically, this section of chapter 2 concludes with a review of the literature on CTVB as a proxy measure of SEP in health research to date.

The chapter then goes on to outline perspectives on CHD primary prevention including an introduction to primary prevention, the historical basis of community based primary prevention, a review of evidence on the effectiveness of community-based primary prevention programmes, political support for primary prevention, an overview of the recent focus on classical versus novel risk factors in CHD and the distribution of classical risk factors according to SEP in asymptomatic populations. The chapter concludes with a brief overview of cardiovascular absolute risk measures.

The chapter continues with an outline of perspectives on CHD secondary prevention, outlining the Quality and Outcomes Framework (QOF) and exception reporting and a review of evidence in equity of risk factor monitoring and therapies prescribing in populations with established CHD. A summary of the key points of the entire literature review concludes chapter 2.

Chapter 3 describes the design and methodology of the HaHP project with a focus on aspects of the project relevant to this thesis. Primary care read codes and related data fields used to identify the target populations of interest and filter the data appropriately are described, as is the ethics approval process, General Practice consent and Caldicott Guardian approval of the study.

Chapter 4 is the first of two results chapters; primary prevention population demographics are followed by regression analysis of the association between CTVB and SIMD in the asymptomatic primary prevention population. The chapter also presents box plots of the distribution of cardiovascular risk factors according to CTVB using this population and regression analysis to assess the significance of these associations. The association between CTVB and absolute cardiovascular risk is also presented; further regression modelling is undertaken to ascertain whether CTVB has greater strength of association with absolute risk in comparison to the association between SIMD and absolute risk. Analyses are stratified by gender.

Chapter 5 begins with population demographics of the secondary prevention population in Paisley in 2009. The chapter then charts risk factor monitoring over the period 1999 to 2009; regression analysis is then presented to ascertain if inequalities in risk factor monitoring exist in the 2009 population only, the same analysis is then presented for secondary prevention prescribing. Furthermore analyses examines whether CTVB has greater association with risk factor monitoring and therapies prescribing in comparison to the associations with SIMD.

Chapter 6 assimilates the thesis findings and discusses, in the context of the literature reviewed, the implications for CHD prevention and the strengths of CTVB's predictive validity and its potential use as a proxy marker of SEP are considered and assessed. The strengths and weaknesses of the present study are described; recommendations from the study are offered. Chapter 6 concludes with a concise conclusion based on the present study.

Appendices provide detail of ethics-related documentation.

CHAPTER 2: LITERATURE REVIEW

2.1 Introduction to the literature review

In this chapter the approaches taken towards searching and reviewing the literature are outlined and a review of the literature relevant to this study is presented. The findings of the literature review are divided into 4 distinct topic areas:

- Introduction to CHD and key evidence supporting the rationale for the study
- Review of measures of socioeconomic position in health research
- Perspectives on the primary prevention of CHD
- Perspectives on the secondary prevention of CHD

2.2 Literature review methodology

2.2.1 Literature Search Strategy

The literature search began in November 2008 and was repeated throughout the duration of the study- most recently June 2010. The literature search strategy was developed with a medical librarian at the University of Glasgow, by means of three meetings to ensure the literature search was as comprehensive and robust as possible. Electronic databases were accessed through the University's online library resources. Databases used included Ovid Medline (R) 1950 to June 2010, Embase 1980 to 2010, Embase Classic 1947 to 1973, Health and Psychosocial Instruments 1985 to June 2010, ERIC 1965 to June 2010, Journals@OVID Full text, Books@OVID. Additionally searches were conducted directly within specialist cardiovascular journals - primarily Biomed Central, European Heart Journal, European Journal of Cardiovascular Prevention and Rehabilitation, Heart, British Medical Journal and Circulation. Furthermore seminal papers and experts within the respective fields were identified with study supervisors and these papers and references from these papers were explored. The search strategy was broken down into the four broad headings of the study detailed in the above sections and involved using Boolean operators and combinations of key words.

Key words used in the review for the Introduction to CHD and key evidence in the rationale for the study section included- “coronary heart disease”, “atherosclerosis”, “coronary artery disease”, “ischemic heart disease”, “heart disease”, “myocardial infarction”, “burden”, “global”, “UK”, “European”, “risk factors”, “classical”, “novel”, “absolute risk”, “high risk”, “prevention strategies”, “Scottish Health Policy”, “Scottish Social Policy Frameworks”, “CHD and Stroke Strategy”, “impact of CHD prevention”, “incidence”, “prevalence”, “primary prevention”, “secondary prevention”, “health inequalities”, “Equally Well”, “markers”, “proxy markers”, “novel markers”, “socioeconomic status”, “socioeconomic position”, “deprivation”, “social class”, “income”, “occupation”, “social stratification” and “council tax”.

Key words used in the review of measures of socioeconomic position in health included- “health”, “measures”, “indices”, “markers”, “proxy markers”, “novel markers”, “socioeconomic status”, “socioeconomic position”, “deprivation”, “social class”, “income”, “occupation”, “education”, “area-based”, “individual measures”, “social stratification” and “council tax”.

Key words used in the review of CHD primary prevention included “primary prevention”, “coronary heart disease”, “coronary artery disease”, “ischemic heart disease”, “heart disease”, “myocardial infarction”, “risk factors”, “classical”, “novel”, “absolute risk”, “high risk”, “Framingham”, “ASSIGN”, “Q-risk”, “socioeconomic status”, “socioeconomic position”, “deprivation”, “social class”, “total cholesterol”, “HDL cholesterol”, “systolic blood pressure”, “diastolic blood pressure”, “body mass index”, “diabetes” and “smoking status”.

Key words used in the review of CHD secondary prevention included “secondary prevention”, “coronary heart disease”, “coronary artery disease”, “ischemic heart disease”, “heart disease”, “myocardial infarction”, “risk factors”, “monitoring”, “management”, “review”, “established CHD”, “therapies”, “prescribing”, “ACE-inhibitor”, “anti-platelet”, “beta-blocker”, “statin”, “primary care”, “general practice”, “high risk”, “Quality Outcomes Framework”, “QOF”, “General Medical Services”, “socioeconomic status”, “socioeconomic position”, “deprivation”, “income”, “occupation”, “social position”, “social class” and “social stratification”

Equivalent or comparable terms identified through thesauruses or mesh browsers were used in specific aspects of searches where possible. Searches were limited to English language research papers, articles and discussions, however no other limits were imposed. Thorough consideration was given to the key word search and the development of the search terms used was an iterative and progressive process, building on the success or otherwise of search terms tried. Despite best efforts within the scope of this study, it should be recognised that electronic databases use a limited range of keywords that typically

describe general topic areas rather than the exact area of interest of the researcher, thus, it is possible that some relevant research papers were overlooked despite the comprehensive nature of this literature search.

2.2.2 Inclusion Criteria

The inclusion criteria were initially deliberately broad; any English language articles that were related to the areas of the literature review were included; including reports, review articles and editorial discussions. Aspects of the search became more focussed on UK studies to enable more accurate synthesis of the thesis findings and not least because CTVB is a tax used in the UK only. Excluded were abstracts from journals of which the University was not a subscriber (thus full text was not accessible), although this proved an infrequent occurrence, also excluded were student theses and conference abstracts.

In order to determine if articles met these criteria and were relevant to the study, the titles and abstracts of papers were scanned in the first instance followed by a more detailed consideration of the full text where doubt remained. All studies were subsequently read in entirety. As a result, in the region of 250 research papers were reviewed and are discussed under the four areas of the review.

The search and inclusion criteria were undertaken to ensure that as far as possible only quality studies were reviewed. However given the breadth of topics synthesised in the thesis- varied study designs and different socio-demographic strata have been considered.

2.3 Introduction to CHD

2.3.1 CHD: a continuum of atherosclerosis

From a biomedical perspective coronary heart disease (CHD) can be thought of as a continuum of a pathological process named atherosclerosis[18]. Atherosclerosis involves the coronary arteries thickening and hardening over time inhibiting blood flow, and the development of atherosclerotic plaques within the arteries, which further inhibit blood flow and risks rupturing[19]. Though typically asymptomatic for decades[20] the resultant effects of the atherosclerotic process are chronic, slowly progressive and cumulative[21]. The effects lead to clinical manifestations such as angina[22], and acute coronary events as a result of rupturing plaques; such as myocardial infarction, acute coronary syndromes and death[23].

Research has suggested that a significant proportion of incident CHD is attributable to modifiable classical risk factors such as smoking, diet, physical activity level and alcohol consumption[24]. This recognition that CHD is a largely preventable disease has focused health policy, both in the UK and elsewhere, on prevention strategies[25]. This perspective belies the challenges CHD presents to epidemiologists and public health practitioners in terms of the science of understanding the disease and the development of approaches to its prevention. Evidence suggests that CHD is caused by a hierarchy of associated factors, beyond the presence of modifiable behavioural risk factors; including social and environmental, as well as political and economic, factors[25]. CHD appears to be a complex disease demanding sustained, multi-faceted approaches to reduce its global burden.

2.3.2 CHD: Global, European and UK perspectives

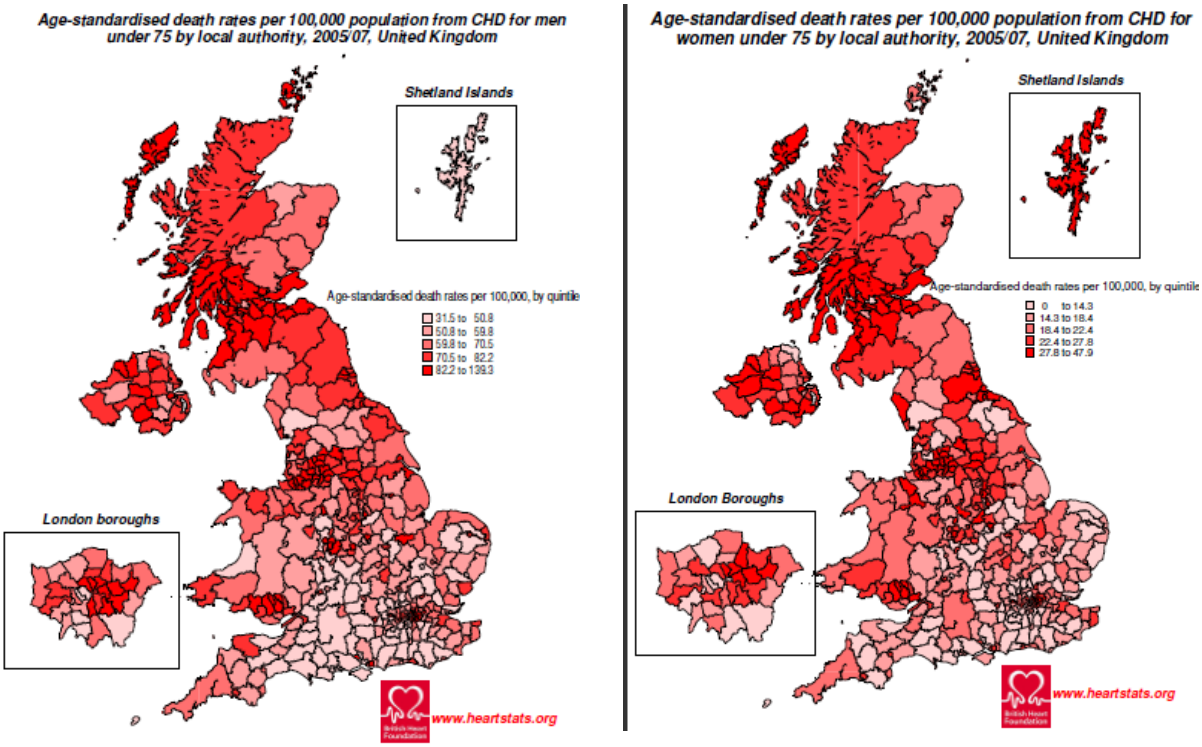
CHD is recognised as a leading cause of mortality and morbidity in both rich and poor countries[26]. In recent years CHD has been gathering unwelcome momentum in poorer developing countries [27]. The World Health Report, conducted in 2003[28], reported that cardiovascular diseases are now the leading cause of death in the majority of developing countries. Furthermore, twice as many deaths, as a result of cardiovascular diseases now occur in developing countries compared to developed countries, with CHD increasingly a key contributor to this observation.

The decline in CHD rates in developed countries has been an emerging trend in epidemiological research over the past three decades[29; 30]. However, these trends have been far from equitable. Across Europe, as CHD declining in the Western countries it was increasing in the former communist countries of Central and Eastern Europe; creating a profound East-West gap[31]. However there is evidence of this gap decreasing in some Eastern European countries[32]. Within European countries, including those of Western Europe, where CHD rates have been falling, some groups in society have not benefitted to the same extent as others[33;34]. Most notably, considerable socio-economic variances in CHD have emerged; whereby those in more favoured socio-economic positions have seen steeper declines in CHD[35]. As a result, socio-economic inequalities in CHD within developed countries[36], including Scotland[37] have actually risen in the past two to three decades.

In the United Kingdom (UK), CHD mortality is higher in Scotland than anywhere else in the country[38]. The 2007 premature death rate (individuals dying aged between 35-74 years) from CHD for men living in Scotland is almost double that of men living in the South West of England and is exactly double for women in the same comparison. The socioeconomic inequalities in CHD observed in other European

countries are apparent within Scotland[37]. Moreover Scotland appears to have its own East-West divide where the post de-industrialised West-central Scotland has markedly higher rates of CHD than the East and North[38]. The map below of the UK illustrates these two points; CHD premature death rates for men (left hand chart) and women (right hand chart) being the highest in local authority areas in Scotland compared to the majority of the rest of the UK (darker colouring), and within Scotland CHD rates are notably higher in West-central Scotland compared to the rest of the country (darker colouring):

Figure 1: Age standardised death rates from CHD form men and women under 75 by local authority



Source: British Heart Foundation Statistics Website

2.3.3 Introduction to Strategies for preventing CHD

The theory that CHD can be prevented has become increasingly popular in cardiovascular medicine in recent decades[39]. For years there have been many prominent detractors from prevention in the field, however gradually prevention has come to the fore. Primary prevention can be defined as the prevention of new-onset CHD[40]. Acceptance of the concept of primary prevention in cardiovascular medicine was arguably achieved through the evidenced success of secondary prevention[41]- defined as the prevention of

recurrent coronary events in patients with established CHD[42]. Secondary prevention appears to straddle two ideological perspectives; some cardiologists argue that secondary prevention is simply the treatment of coronary artery disease; others might argue that the focus is on the prevention of recurrent events[43].

2.3.4 Estimating impact of CHD prevention strategies

The prevalence (number of current cases) of CHD is influenced by a range of associated factors. To illustrate, a decrease in the number of new CHD cases (incidence) due to primary prevention efforts would reduce prevalence, however higher survival rates resulting from secondary prevention efforts, coupled with an aging population would actually increase prevalence[44]. CHD mortality rates are also influenced in a similar fashion[45]. Major advances in care and treatment have undoubtedly contributed to falling CHD mortality rates, however falling incidence rates must also be contributing to the overall reductions in mortality. The incidence of CHD is determined by complex interwoven factors acting over the life course. The contributions to reductions in mortality rates of improving these risk factors and of improvements in care and treatments are not completely clear. In a seminal paper Capewell and colleagues estimate that changes in risk factors equate to approximately a 50% reduction in mortality rates and improvements in care and treatments equate to a further 40%, leaving 10% un-attributable[46].

2.3.5 Approaches to addressing health inequalities: CHD and beyond

Beyond CHD, there is a paucity of reliable evidence relating to the most effective and cost effective methods or approaches when addressing socioeconomic inequalities in health. Specifically there are challenges regarding what approach is most successful in making sure resources are targeted at the highest risk populations. Area based approaches are convenient for practitioners because established national measures of socio-economic position tend to be area based. However identifying deprived areas is not the same as targeting deprived households or individuals, because not all deprived people live in deprived areas. It was estimated using the 1991 census that if the most deprived quintile of Scottish postcode sectors were targeted only 41% of unemployed individuals would be captured and only 34% of low income households would be captured[11].

Furthermore the importance of this point is recognised by the Scottish Government. Within the *Equally Well* policy document:

'Area based initiatives need to be complemented by approaches which specifically target disadvantaged individuals or households.'[37]

2.4 Review of measures of socioeconomic position in health

This section of the review summarises literature in relation to measures of socioeconomic position (SEP) in health. The literature surrounding the concept of SEP is virtually immeasurable; with much debate continuing out with the scope of this thesis. The review describes the nature of the measures; what they intend to quantify, the types of data used and the strengths and weaknesses of the measures are assessed. The term SEP is used in the review however the terms socioeconomic status, social position, social class and social stratification are used in the literature summarised. It became apparent that these terms are however not interchangeable and refer to differing theoretical constructs and may represent differing interpretations; whilst this will be touched on within the review it is beyond the scope of the study to explore these constructs in the detail, thus to a degree these terms will be considered as having similar meaning.

SEP can be broadly defined as the social and economic circumstances of an individual or group relative to the rest of society[47; 48].. In terms of health, SEP is associated with exposures to risk, behaviours affecting risk, access to resources and general susceptibility to disease or illness[49]. Different measures of SEP have their own strengths and limitations; particularly in the context of the research field[50].

During the review it became apparent that one of the key considerations is the differing nature of the theorised link between SEP and the health outcome of interest. SEP is often described as a confounding or explanatory factor within studies (often controlled for in analysis) but is also the core exposure or risk factor (and its influence on health or disease) investigated within studies. Increasingly dominant in recent literature is the influence or association of SEP and stage of life. It is also clear that the majority of measures of SEP have theoretical correlations across the stages of life, for example educational attainment has a clear bearing on occupation as an adult and income and specific to this study; the value of the house that can be purchased and thus the CTVB that the house falls under. Consistent with the aims of the present study, the review organises the measures of SEP in the following ways- firstly individual measures are considered followed by area level measures, subsequently arguments for and against individual and area level measures within the context of CHD and cardiovascular risk are discussed.

2.4.1 Education

Education is a widely used measure of SEP particularly in epidemiological studies. The history of the use of education as a measure of SEP is associated with Weberian theory[51]; which describes education as an attempt to capture the 'knowledge related assets of a person'. Educational is influenced by parental characteristics and to an extent reflect the overall circumstances of a person in these early years[52].

Education is typically measured as a categorical variable by assessing educational achievements such as completion of primary school, high school, college diplomas or university degree[53]. Education reflects the circumstance of parental SEP and influences in early life but also captures the development of an individual into adulthood where their own socioeconomic identity begins to be realised[54]. Education is a strong predictor of adult occupation and income[55]. Thus education reflects the well evidenced association between early life circumstance and adult health outcomes. Indeed several studies have concluded that SEP across the life course influences CHD risk with childhood SEP and adult SEP both contributing to risk independently and cumulatively[56].

Furthermore educational attainment has been cited as pivotal in understanding health education messages[57]. Education may also play a role in the ability of individuals to access health services[58]. Interestingly poor health in early years could limit educational attendance affecting adult health outcomes- perhaps suggesting a selective influence within health inequalities[59].

Education is a widely used measure of SEP in health research; furthermore educational attainment is easy to gather and is non-intrusive to record. However educational opportunity for some sub-groups of society has varied greatly over the generations. Thus older individuals may be classified as less educated in some studies[60]. Generally it proved difficult to compare and synthesise evidence using education as a measure of SEP due to the large variance in its recording.

2.4.2 Housing

Of particular interest to this study are measures of SEP which quantify housing circumstance and characteristics. Housing based indicators of SEP vary across the literature and often refer to localised measures of housing, particularly within rural or non-industrialised studies or countries[61]. A number of quality studies have examined housing tenure in relation to CHD[62; 63], the former study being a comprehensive systematic review and the latter from a large scale Scottish cohort study. Characteristics within the household (access to a toilet, hot water, telephone or heating) are also used as a measure of SEP in some studies[64].

Car access is a characteristic of housing or household which is used frequently throughout UK studies as an indicator of SEP[65; 66].. Car access is related to the notion of socioeconomic gradients in access to health services[67] or retailers selling fresh fruit and vegetables[68]; however it is less appropriate in rural populations as even the most deprived households have a car out of immediate requirement[69].

The number of individuals living in a house commonly termed as crowding or overcrowding has been used as a marker of SEP[70].Links to poor health exist as a result of overcrowding[71]. Cultural variances in numbers of family members residing in a house may not be representative of SEP.

Housing data has strong features as a marker of SEP with clear links to income[72]. Housing standard is often referred to as a characteristic of wealth, which can be regarded as comprising of income, financial and physical assets[73]. Wealth has been shown to be associated with access to healthcare services, provide environments (residence and work) conducive to good health and allow the consumption of health promoting commodities (healthy diet, exercise) which has an important effect on health[74]. The limitations of the housing measures of SEP reviewed are the inherent dependence on the context of the study; in terms of geography and study population of interest. Thus the literature was difficult to synthesise and generalise there on.

2.4.3 Income

From the literature reviewed it is apparent that income is generally regarded as the most accurate measure of SEP available[75]. Income as an indicator of SEP directly measures material resources and circumstance[76]. There is a correlation between increasing income and better health outcomes[77; 78]. The association between income and health is accepted across the literature yet the causal mechanism is rarely explored, representing a gap in evidence. Few studies reviewed directly consider income expenditure on health promoting commodities.

Income is usually measured in categories[79]. Household income is less frequently reported and in some studies this is considered by family size or dependants [80]- this theoretically provides a more accurate reflection of the resources available to the household[81].

Personal income may be a sensitive subject and people may be reticent in providing it[82]. No studies reviewed acknowledged the possibility of the over reporting of income. Income is strongly associated with educational attainment and occupation[83; 84]. Arguably disposable income would be the best measure of SEP, which over and above costs of living would reflect available resource to consume commodities

relating to health; this could be further enhanced by considering family size[85]. Less often income is considered over the life course[86]; this is important as income tends to increase with age, thus SEP will continually rise over the life course making the interpretation of SEP somewhat variable[87].

2.4.4 Occupation

Occupation based measures of SEP are widely used in the literature [88-90]. Similar to education, occupation has historical links to Weber's theory that SEP should reflect a person's standing in society. Thus occupation reflects educational attainment, intellectual capacity and the underlying social class[91]. Similar to income, stage of life is a consideration in using occupation as a marker of SEP[92], some studies refer to parental occupation as a measure of early years SEP relating to adult health outcomes[93]. Thus occupation reflects the transferability of material circumstances from one generation to the next. Occupation of the head of the household is often used to reflect the SEP of the family or household[94].

Occupation is usually referred to in categories in the literature (manual, professional etc) and was the measure of SEP used in the Whitehall study[95; 96] where the occupational grades of civil servants were categorised and the distribution of cardiovascular risk factors and outcomes were analysed accordingly revealing stark occupational inequalities in the disease.

Similar to income and housing, occupation can be considered to have an indirect bearing on health in terms of the degree to which health promoting commodities can be purchased[97]. Occupation type also has interesting characteristics relating to health; lower income manual jobs may have greater exposure to environmental hazards and higher physical demands[98]. Lower income jobs are also categorised as repetitive, unsupportive and having little autonomy resulting in poorer overall health outcomes[99].

Occupation has been well recorded for a long time, notably in British death records and in census data collection[100]. The most appropriate method of recording the occupation of unemployed, retired, carers or those in illegal jobs is problematic; health inequalities may be under-represented in studies which do not included such individuals[101]. Furthermore inaccuracies are inevitable when attempting to quantify occupation across a limited range of categories. Moreover, temporal analysis using occupation as a measure of SEP is problematic due to the changing nature of the modern workforce[102].

2.4.5 Area-level measures

Area level measures of SEP are aggregated from individual data and small area characteristics, usually from census and government administration data sources. They characterise areas on a scale from most to least deprived. Area-based measures represent only a proxy measure of SEP for the individuals living in those areas. In the UK there has been a relative proliferation of composite area based measures in recent years, such as the Townsend deprivation index[103], the Carstairs deprivation index[104], the Jarman or underprivileged area (UPA) score[105] and the Scottish Index of Multiple Deprivation (SIMD)[106]. The SIMD is used in subsequent analysis in this study. The SIMD comprises of 37 indicators of SEP in seven domains: current income, employment, health, education, skills and training, geographic access to services (including public transport travel times), housing tenure and crime levels. SIMD is based on aggregated data within defined geographical parameters called data zones which on average contain approximately 800 individuals. Data zones are then refined into deciles or quintiles; a continuum of socioeconomic deprivation[107].

The key strengths of area based measures such as SIMD is that they are official and available for the entire nation and easily linked to study populations[108]. Area measures of SEP are complete, clean datasets and are unobtrusive and cheap to gather and use[109]. Furthermore area based measures are less prone to misclassification than individual measures[110].

Area based measures the socioeconomic conditions of an area to some extent; thus whilst area based measures are derived from aggregated individual level data and other sources the relationship to health is primarily described as the area's influence on health[111].

2.4.6 Discourse in the literature; contextual and compositional influences on health; area versus individual measures of SEP

There is an ongoing debate within the literature regarding the merits of area-based versus individual measures of SEP and which are most appropriate to use in health research in general and for particular disease and study types[112-118]. The focus of the debate in relation to CHD has been to describe the wider influences on CHD mortality and morbidity which are not fully explained by classical risk factor analysis at the individual level[119;120]. Thus, the scope of studies has expanded to explore how the socioeconomic environment of an area affects the health behaviours and outcomes of those living there[121; 122]. Geographic variance in cardiovascular risk and health behaviours is often described within the literature as the

compositional (i.e. how many people residing in the area smoke, lead a sedentary existence etc) or the contextual influences (i.e. transport, access to fresh fruit and vegetables, quality of housing, access to safe environments to exercise etc) on[123].

Uncertainty regarding the balance between compositional and contextual influences and the interplay with SEP on the development of CHD limits preventative strategies in important ways[124]. If there are primarily compositional explanations for the socioeconomic inequalities in CHD then prevention strategies should focus on the individual; thus utilising individual measures of SEP where possible[125; 126]. On the other hand a contextual explanation would mean action on the wider living and working conditions in an area; targeting geographical areas or neighbourhoods; thus the measures of SEP used in resource allocation should be area-level indicators[127;128].

From the evidence reviewed it appears that both contextual and compositional factors influence behaviours, exposure to risk factors and cardiovascular outcomes. It is interesting to note within the literature reviewed that many studies report associations between either contextual and compositional influences or area-based and individual measures of SEP to the detriment of the association with the other. In other words it appears that the theoretical construct which underpins many papers in this field is that contextual or compositional influences on health cannot co-exist.

A recent study which defies this construct found independent effects of both individual SEP and residential area deprivation on classical behavioural risk factors - smoking, exercise and diet. The study concluded that although community SEP and individual SEP may affect each other, they also operate through separate pathways to affect health behaviours[129] and that this is what is important. An important influence explored by Mitchell[130; 131] has been proximity and access to natural green space, straddling both contextual and compositional ideologies of health behaviour influence.

2.4.7 CTVB as a proxy household-level measure of SEP

The Council Tax was introduced by the British Government in 1992 and is based on the market value of British homes as at 1st April 1991 (for Wales the valuation date is 1st April 2003). The valuation then places the home into one of eight bands- A-H with band A being the lowest and band H the highest. Houses built or modified since 1991 are valued in the present day and then devalued to 1991 levels for banding. Council tax banding data is accessible information, at a household level, under the freedom of information act, either online or through local valuation boards. The range of property values according to council tax for Scotland and England are shown in the below two tables:

Table 1: Council Tax Valuation Bandings for Scotland

Council Tax valuation bands	Ranges of property values in Scotland
A	up to £27,000
B	over £27,000 and up to £35,000
C	over £35,000 and up to £45,000
D	over £45,000 and up to £58,000
E	over £58,000 and up to £80,000
F	over £80,000 and up to £106,000
G	over £106,000 and up to £212,000
H	over £212,000

There is one Council Tax bill for every property in the UK. Usually the person living in the property has to pay the Council Tax Bill. Spouses and partners who live together are jointly responsible for paying the Council Tax bill. The below list represents a hierarchy of responsibility for paying Council Tax, the bill payer being the individual who:

• lives in the property and owns it

• lives in the property and has a lease (this includes 'assured tenants' under the Housing Act 1988)

• lives in the property and is a 'statutory' or 'secure' tenant

• lives in the property and is not a tenant but has permission to live there

• lives in the property (for example a squatter)

• has a lease of six months or more on the property, but does not live there

• owns the property but does not live there

The use of CTVB as a surrogate marker of SEP in health research has been limited to date and has been investigated in small studies, primarily within primary care. Beale has led the use of CTVB as a marker of SEP. In a 2001 study, Beale et al began exploration of CTVB as a marker of SEP by establishing its

association with the 'Jarman Index'[133]. The Jarman index or Under-privileged area 8 (UPA8) score is an established marker of SEP developed in the 1980s and is based on 8 socioeconomic factors available from UK census returns. Beale et al conclude that CTVB and the Jarman index are highly correlated but that CTVB is a stronger predictor of GP workload. Unlike the Jarman index, Beale argues that CTVB is simple, objective, and free of the problems of Census data. Furthermore CTVB, being household-based, can be aggregated at will.

In 2002, Beale et al undertook a study to determine the association between the CTVB of residence and mortality risk using the death registers of a UK general practice[134]. The study findings from analysis of 856 deaths were that consistent and significant differences in death rates between CTVBs exist. Above average mortality was identified in bands A and B residents; below average for other band residents. The study concludes that CTVB of final residence appears to be a surrogate marker of mortality risk and could be a worthwhile indicator of health needs resource at a household level.

In 2005 Beale et al investigated the costs of daily clinical activities within a general practice by gender, age and SEP as measured by CTVB[135]. The results of the study were strong- CTVB was as strong a predictor of patient care cost as patient gender and age. The study concludes that NHS planning and resource allocation could be simplified and enhanced by using CTVB as a marker of SEP.

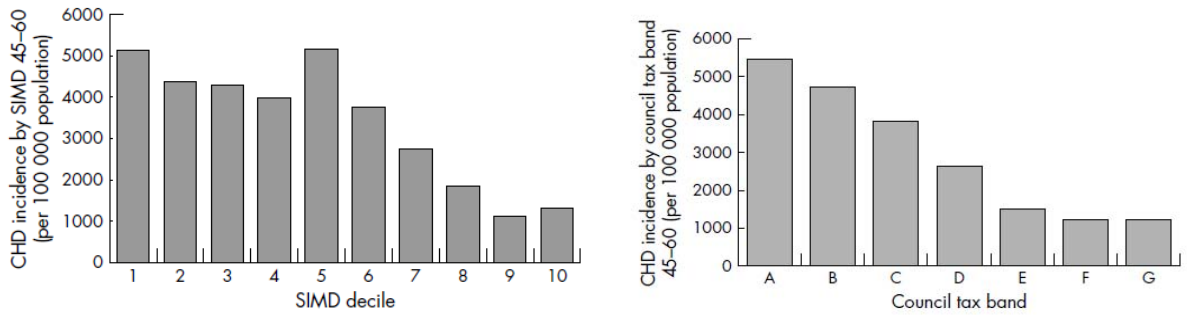
In 2006 Beale et al used data from the ALSPAC sample (Avon Longitudinal Study of Parents and children) to investigate the association between CTVB and breast-feeding rates[136]. The study concludes that CTVB predicts breast-feeding rates and that CTVB could be used for accurate resource allocation within community paediatric services.

Whilst Beale et al's work has been innovative; the study populations used (with the exception of the ALSPAC breast feeding study) have exclusively been the general practice from which the lead author works. These studies have small sample sizes and give little insight into the socio-demographics of the population. The accuracy of primary care records can be questioned as their primary purpose is administrative rather than for official recording. Furthermore Beale et al make no reference to housing tenure as having a possible confounding effect on the theorised link between income, material circumstance and CTVB. The paper makes no reference to the data linkage of CTVB. Thus the accuracy of CTVB as a measure of SEP is arguably not properly critiqued, theoretically or practically in these studies. Indeed Beale's discussion in each of the published papers appears to lack scientific objectivity. The conclusions are so strikingly in unequivocal support of CTVB as a marker of SEP above all other measures that it detracts from the credibility of the research.

In a larger 2006 study[137], Fone et al looked to assess CTVB as a measure of SEP by comparing the strength of the associations between selected health and lifestyle outcomes. The study found that there were significant trends in odds ratios across the CTVB categories for all outcomes, most marked for smoking and mental and physical health status. The associations with CTVB were higher than that of the established measures of SEP considered in the study. The study concludes that CTVB deserves further consideration as a proxy for SEP in epidemiological studies in Great Britain. Unlike Beale, Fone et al acknowledge that there were anomalies in data linkage and that CTVB does not distinguish between owner-occupied and rented accommodation.

Findlay et al were the first to relate CTVB to cardiovascular outcomes[138]. This 2006 study used data from the HaHP CDR to investigate the association of CHD incidence had a closer association with CTVB than with SIMD The correlation coefficient between SIMD score and CHD incidence for all ages was 0.71 and for CTVB was 0.89. The correlation coefficient for those aged 45–60 was 0.90 and 0.98 respectively. The below figure chart the latter analyses; the chart on the left shows the distribution of CHD incidence by SIMD and the chart on the right shows the distribution of CHD incidence by CTVB:

Figure 2: Findlay et al, Distribution of CHD incidence according to SIMD and CTVB in 45-60 year old men and women in Paisley 2005



The study concludes that CTVB should be explored as a simple measure to individualise the correction that needs to be applied to standard risk calculators to account for the influence of deprivation on CHD risk. This paper has been pivotal to the present study; it is arguably the highest quality study of CTVB as a marker of SEP as it uses accurate hospital discharge data required for national morbidity and mortality records.

Theoretically CTVB incorporates many of the characteristics of the measures of SEP covered thus far. CTVB is a measure of housing value; this has theorised links to material circumstance relating to education, occupation and income. The value of the house purchased must reflect to an extent these characteristics of SEP. CTVB being sensitive to the household level is close to an individual measure of SEP. However housing value is influenced also by area characteristics and the socio-demographic compositions of areas. For example, identical tenement housing stock varies dramatically in value within the present study; as the areas vary from most to least deprived according to SIMD. Crucially CTVB is available for entire populations and requires no collection.

2.5 Perspectives on Primary Prevention of CHD

2.5.1 Introduction to the primary prevention of CHD

The Scottish National CHD and Stroke strategies of the past decade state that effective primary prevention is a key priority in reducing the burden of CHD[37]. Despite this, resource allocated for care and treatment far outweigh that of prevention[139; 140]. Evidence as to the effectiveness of primary prevention is

mixed[141; 142]. The difficulty in generating quality evidence from primary prevention programmes is however recognised[143; 144].

In 1995 the WHO European Working Group on Health Promotion Evaluation[145] state that using randomised control trials (RCTs) to evaluate such programmes is inappropriate. Thus CHD primary prevention research often falls short of scientific approval. Most CHD primary prevention programmes are community-based programmes[146-148]; in contrast to the financial might of the pharmaceutical industry arguably these programmes may appear colloquial and underfunded[149].

2.5.2 The historical basis of community based CHD Primary Prevention

Since the 1970s there have been countless CHD primary prevention interventions and programmes. The nature of primary prevention programmes delivered has varied, involving risk screening drug therapies, educational and media programmes as well as community development activities. Wider focus has also been on health related legislation and policy - such as food retailing and cigarette advertising[150].

Some of the earlier primary prevention interventions such as the North Karelia Project[151] and the Stanford Three City Projects[152] have been influential to similar large-scale community based trials- the Stanford Five City Project[153], the Minnesota Heart Health Project[154] and the Pawtucket Heart Health Project[155]

2.5.3 Assessing the impact of CHD primary prevention

In 1999 Lundvall et al[156] published a systematic review of CHD primary prevention programs. The authors included only high quality studies involving a control group. Eight studies were included in this review:

Table 2: CHD primary prevention interventions considered in Lundvall et al, systematic review

Community based CHD prevention programme	**Nation of study and Year**	**Authors**
North Karelia	Finland 1972–1977	Pushka et al[157]. Vartiainen et al[158].
The Stanford Three City Projects	US – California 1972-1975	Farquhar et al[159].
The Stanford Five City Project	US – California 1980-1986	Farqhuhar et al[160].

Community based CHD prevention programme	**Nation of study and Year**	**Authors**
Minnesota Heart Health Program	US -Minnesota 1981-1988	Luepker et al[161].
Pawtuckett Heart Health Program	US -New England 1981-1994	Carleton et al[162].
German Cardiovascular Prevention Programme	Germany 1984-1991	Hoffmeister et al[163]
Swiss National Research Programme	Switserland 1985-	Gutzwiller, et al[164]
Kilkenny Project	Ireland 1985-1992	Shelley et al[165]

The Lundvall review concluded that outcomes were insignificant and that the differences seen between intervention and control areas were negligible in terms of reductions in classical risk factors or CHD incidence. They summarised that:

> *'There is no conclusive scientific evidence that would support starting new large scale community intervention programmes – such as those assessed here – aimed at preventing cardiovascular disease. The eight large community intervention projects reviewed in this report have not demonstrated any significant effects on risk factor levels or disease incidence beyond those observed in populations at large'*

There have been some studies of primary prevention interventions where classical risk factors have shown significant reductions[166; 167], however, it remains unclear if these reductions are large enough to impact on CHD morbidity or mortality.

In 1997, Ebrahim and Davey-Smith conducted a comprehensive systematic review and meta-analysis[168] which aimed to assess the effect of multiple risk factor primary prevention interventions in reducing total mortality, CHD mortality. All the trials and interventions included in the analysis were randomised designs. The paper considered interventions from 1966 to 1995..

Davey-Smith concluded that these interventions had no effect on mortality given that the pooled effect for both CHD and total mortality was 0.97:

'Multiple risk factor interventions comprising counselling, education, and drug treatments were ineffective in achieving reductions in total mortality or mortality from cardiovascular disease when used in general or workforce populations of middle aged adults. The pooled effects of intervention were insignificant, but a potentially useful benefit of treatment (about a 10% reduction in mortality from coronary heart disease) may have been missed'.

Furthermore, the risk factor changes that occurred were modest; the authors suggested that they may even have been overestimated due to issues of measurement, analysis and study design:

'The changes in risk factors associated with interventions were modest but are probably optimistic estimates as changes could be measured only in those remaining in the trials. Habituation to blood pressure measurement, regression to the mean and self reports of smoking will also tend to exaggerate the changes observed'.

2.5.4 Political support for CHD primary prevention

In the face of convincing evidence to the contrary policy makers continue to support CHD primary prevention. The role of scientific evidence in policy making is a complex and nuanced paradigm[169].

2.5.5 Focus of primary prevention: classical versus novel cardiovascular risk factors

In recent years epidemiological research into CHD risk-factors has reached a crossroads. One route suggests there is already convincing evidence to take effective preventative action against CHD in relation to classical behavioural and physiological risk factors [170]. The other route argues that classical risk factors only explain a proportion of the socio-economic gradient in CHD[171; 172]. The latter path has thus seen a drive to identify new risk factors to further the understanding of CHD aetiology; lipoprotein, C-reactive protein, fibrinogen, homocysteine, microalbuminuria, inflammation, anti-oxidant intake, fish intake, air pollution, personality types, oral hygiene and gene-environment interactions to name but a few[173].

The literature suggests that research into emerging risk factors should be continuous[174]; however focus on classical risk factors should remain the key priority in CHD prevention. The American Heart Association's (AHA) position is clear; classifying major risk factors as those that research has shown significantly increase the risk of CHD, these include risk factors that cannot be changed (increasing age,

male gender and heredity) and those which can be modified or controlled through lifestyle change or taking medicine (tobacco smoking, high blood cholesterol, high blood pressure, physical inactivity, obesity and diabetes). The AHA recognises novel risk factors but states that they have not yet been precisely determined[175].

Individuals of lower socio-economic position continue to have significantly greater exposure to classical risk factors[176]. Research suggests that a proportion of increased risk in areas of socioeconomic deprivation is due to coping behaviours[177; 178].

Whilst the exact interaction of classical risk factor exposure, SEP and heredity susceptibility are not completely understood in the development of CHD, a progressive step in recent literature has been to at least view SEP as an independent risk factor. A Scottish study thus incorporated SEP as a risk factor when calculating absolute cardiovascular risk in screening[179]. This inclusion increased the predictive power of the risk calculator. Whether SEP is considered as an independent risk factor[180] or termed as a contributing risk factor[175] is inconsequential; to maximise the use of CHD preventative resources SEP is a core consideration.

The focus on classical risk factors and SEP is further underlined when exposure is considered over the life-course[181; 182]. It is argued that the explanatory power of classical risk factors can be massively underplayed[183; 184] by a single measurement in mid-life. This viewpoint casts doubt over the need to 'explain' why measurements of classical risk factors explain only a proportion of the socioeconomic gradient in CHD[185]. Thus a key priority for CHD preventative programmes continues to be the focus on reducing exposure to classical risk factors targeting individuals or areas of low SEP[186].

2.5.6 Cardiovascular risk factors in CHD asymptomatic populations and SEP

SEP, measured by occupation, educational level and income is related to mortality and morbidity from CHD[187]. Evidence suggests that the most marked improvements in cardiovascular health have occurred among higher SEP populations, whilst progress among lower SEP populations has been slower[188]. Overall evidence points to lower SEP populations having greater exposure to risk factors; such as smoking, unhealthy diets, sedentary lifestyles and have worsened psychological profiles[189].

The literature review will now focus on the distribution of selected classical risk factors; gender and age, diabetes, cholesterol, blood pressure, body mass index and smoking status according to measures of SEP.

2.5.6.1 Gender and age

There are differences in CHD risk between the sexes, CHD is between 2 to 5 times more likely to develop in men than women[190]. In both men and women the risk of CHD increases significantly with age but the rate of increase is sharper for women. Differences in classical risk factors explain a substantial proportion of the gender differences in CHD risk[191]. The sharp increase in CHD risk seen in women in later life is associated with the decrease in oestrogen production post menopause and the effect this hormonal change has on lipid metabolism[192].

2.5.6.2 Diabetes

The presence of non-insulin dependent (Type II) diabetes is associated with increased risk of CHD and excessive CHD mortality and morbidity. The INTERHEART study estimates this elevated risk as being increased 3 fold[193]. Furthermore diabetes rates increase as SEP decreases[194;195], particularly amongst women and ethnic groups[196-198], however this relationship is not reported across all the literature[199;200].

2.5.6.3 Cholesterol

CHD risk is related to blood cholesterol levels[201]. The World Health Organization report 2002 estimates that 8% of all disease burden in developed countries can be attributed to raised cholesterol levels[28]. Furthermore the report estimates that 60% of incident CHD and 40% of stroke is due to raised cholesterol.

The relationship between SEP and total cholesterol level is less clear. Many studies report an inverse relationship between SEP and serum cholesterol[202; 203], but not all[204].

High density lipoprotein (HDL) cholesterol is commonly referred to as 'good' cholesterol because it removes cholesterol from the blood via the liver[205]. Low levels of HDL cholesterol are associated with increased CHD risk. Evidence exists of socioeconomic variance within lipids- with lower SEP individuals having higher levels of total cholesterol and lower levels of HDL cholesterol[206].

2.5.6.4 Blood Pressure

Raised blood pressure (BP) levels or hypertension is a CHD risk factor. An inverse relationship between BP and SEP is reported throughout the literature[207-209]. Irrespective of the measure of SEP adopted the literature consistently reports that lower SEP individuals have higher rates of elevated BP (both systolic and diastolic)[210].

Lower rates of education and awareness of hypertensive risk have been identified in deprived communities[211]. The combination of genetic susceptibility and job strain (physically demanding/low autonomy/decision making authority roles) has been shown to contribute towards raised blood pressure[212]. Foetal mal-nutrition has also been shown to play a role in increasing the susceptibility for hypertension in adulthood[213].

2.5.6.5 Smoking

Smoking significantly increases the risk of CHD[214]. It has been estimated that one fifth of all cardiovascular related deaths are attributed to smoking[28]. Passive, second-hand smoke is also a cardiovascular risk factor[215].

Smoking is consistently related to SEP. The key theme emerging is that individuals of lower SEP are more likely to smoke[216;217]; this association is consistent across all ages and gender[218] and is irrespective of the measure of SEP[219] . Overall smoking rates have been decreasing but have been decreasing at a significantly slower rate in areas of lower SEP[11].

2.5.6.6 Body Mass Index

Obesity is a CHD risk factor. Obesity is an independent CHD risk factor and is also a major contributor towards raised blood pressure, increased blood cholesterol, impaired glucose tolerance and diabetes[220]. 7% of all disease burden in developed countries is due to raised body mass index (BMI) and one third of CHD and stroke and almost 60% of hypertensive disease is as a result of being overweight (BMI from 25-30 kg/m^2) and obese (BMI $\geq$ 30 kg/m^2)[28]. Increased cardiovascular risk is posed when excess weight is concentrated around the lower abdominal area. Waist to hip ratio is used to measure this phenomenon and literature refers to it as central or abdominal obesity[221; 222].

The literature reviewed concerning the relationship of SEP to obesity concludes that women of lower SEP are more likely to be obese than the rest of the socioeconomic strata[223], but that in men the association is far less clear[224;225].

2.5.6.7 Identifying high risk- screening strategies and measures of absolute cardiovascular risk

National, structural, health improvement policy aims to lessen the population distribution of risk through appropriate legislation- smoking bans, reduced salt/fat in food etc[226]. Such legislation is an important aspect of primary prevention. The majority of evidence reviewed suggests that primary prevention should be targeted at those with the highest risk. Some argue however that this may actually widen inequalities[227]. Others advocate a duel approach; both legislative and highest risk targeting[228-230].Engaging lower SEP communities in primary prevention demands nuanced approaches[16]

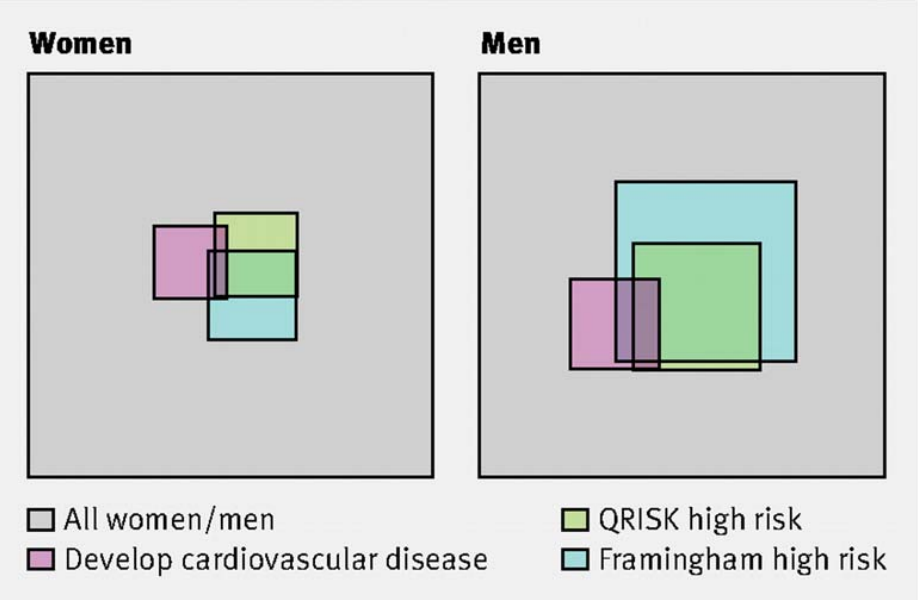
CHD risk factors affect each other cumulatively tending to cluster in high risk populations. This has seen the development of multivariable risk prediction algorithms which allow rapid assessment of absolute cardiovascular risk. Absolute measures of risk have been advocated to guide treatment of risk factors. The Framingham CHD risk assessment tool uses age, sex, family history of CHD, systolic blood pressure, total and HDL cholesterol, diabetes status, and smoking status to generate the chance (expressed as a %) of developing CHD in the next 10 years[231]. Individuals with a Framingham score ≥ 20% are considered to be high risk.

The Framingham score has been criticised because it does not encompass SEP as a risk factor. In 2005 Tunstall-Pedoe and co-workers[232] added SEP (as measured by SIMD) into the Framingham equation to derive the ASSIGN score. The ASSIGN score increased the predictive power of cardiovascular outcomes ahead of Framingham. Soon after Hippsley-Cox et al[233] derived QRISK, which in addition to classical risk factors used in Framingham also includes BMI, family history of cardiovascular disease, social deprivation (Townsend score) and the use of antihypertensive treatment. The study concludes that QRISK was better calibrated to the UK population than either the Framingham model or ASSIGN.

Jackson et al[234], strike a sobering note as to the accuracy of risk prediction tools, stating that all tools yield modest results and that QRISK is no different classifying 10% of men in the UK as high risk however only 30% of subsequent cardiovascular events in men occurred in this high risk group. Framingham classifies twice as many men in the UK as high risk, although this larger group does not include twice as many of the men who had a cardiovascular event during follow-up (it included only 50%). Thus the

margin for improvement is small; indeed Framingham predicts a larger quantity of high risk individuals if not as accurately. The below figure illustrates this point; representing the predictive power of QRISK and Framingham:

Figure 3: Jackson et al, Accuracy of QRISK and Framingham absolute risk measures



2.6 CHD mortality and SEP

Critical to primary prevention strategies (and secondary prevention) has been the association between CHD mortality and SEP. CHD mortality is higher in the most deprived; this has been reported in the literature for over 50 years[235]. A recent Scottish study investigating CHD mortality using SIMD as the measure of SEP concluded that premature death from CHD remains a major contributor to social inequalities. Furthermore, the plateau in the decline in mortality for CHD among younger adults of lowest SEP is worrying[236]. Similar findings have been observed in other Scottish studies[237; 238]. The studies reviewed have not controlled for the effects of risk-factors on mortality.

2.7 CHD Incidence and SEP

One large study (2.6 million people) examined the entire Swedish population (aged 40-64) and found an association between CHD incidence and income; although only sex and age were adjusted for in the

analysis[239]. However in another study which did adjust for classical risk factors, income was associated with increased incidence in both men and women[240].

2.8 Perspectives on Secondary prevention of CHD

2.8.1 Introduction to Secondary Prevention of CHD in Primary Care

In comparison to primary prevention of CHD, secondary prevention of the disease has established guidelines and stronger evidence. The at risk population are clearly identified. The care and treatment of individuals with CHD is well established and the impacts of secondary prevention are significant[241; 242]. That said there remains evidence of inequalities in the delivery of secondary prevention[243]. The focus of secondary prevention strategies in the UK literature has been on exposure to risk factors, both behaviour risk (stopping smoking, adopting a healthy, balanced diet and cardiac-rehabilitation) and physiological risk (cholesterol, blood pressure, body mass index). The appropriate treatment through secondary prevention medications (anti-platelet therapy, statins, ACE inhibitors and beta-blockers, if there are no specific clinical contraindications) is also pivotal[244; 245]. The importance of secondary prevention drugs cannot be underplayed; they are estimated to account for 10% of the reduction in CHD mortality rates in the UK and are thought to be an inexpensive and effective prevention method[246].

Most patients with CHD are cared for in primary care[247]. In recent years there has been much research into secondary prevention treatment and practice among patients with CHD in primary care[248]. In 1999 the UK CHD National Service Framework recognised that 100% uptake of secondary prevention therapies in primary care is unrealistic and set national targets of 80%[249]. Yet evidence suggests that even these targets remained challenging[250; 251]. In 2004 the General Medical Services (GMS) introduced the Quality Outcomes Framework (QOF)[252], in which scores attained are now directly linked to general practitioner remuneration. The QOF contract has set national standards for quality CHD care based on a variety of indicators including regular classical risk factor monitoring, advice and referral on smoking and prescribing appropriate secondary prevention therapies. Figure 4 below details the targets set in the QOF for the secondary prevention of CHD, this figure is extracted from a 2007 study[253].

Figure 4: GMS QOF indicators and targets in secondary prevention of CHD

Secondary Prevention in Coronary Heart Disease (Description of Indicators)	Points*	Target**
CHD 2. The percentage of patients with newly diagnosed angina (diagnosed after 01/04/03) who are referred for exercise testing and/or specialist assessment	7	90%
CHD 3. The percentage of patients with coronary heart disease, whose notes record smoking status in the past 15 months, except those who have never smoked where smoking status need be recorded only once	7	90%
CHD 4. The percentage of patients with coronary heart disease who smoke, whose notes contain a record that smoking cessation advice has been offered within the last 15 months	4	90%
CHD 5. The percentage of patients with coronary heart disease whose notes have a record of blood pressure in the previous 15 months	7	90%
CHD 6. The percentage of patients with coronary heart disease, in whom the last blood pressure reading (measured in the last 15 months) is 150/90 or less	19	70%
CHD 7. The percentage of patients with coronary heart disease whose notes have a record of total cholesterol in the previous 15 months	7	90%
CHD 8. The percentage of patients with coronary heart disease whose last measured total cholesterol (measured in the last 15 months) is 5 mmol/l or less	16	60%
CHD 9. The percentage of patients with coronary heart disease with a record in the last 15 months that aspirin, an alternative anti-platelet therapy, or an anti-coagulant is being taken (unless a contraindication or side effects are recorded)	7	90%
CHD 10. The percentage of patients with coronary heart disease who are currently treated with a beta blocker (unless a contraindication or side-effects are recorded)	7	50%
CHD 11. The percentage of patients with a history of myocardial infarction (diagnosed after 1 April 2003) who are currently treated with an ACE inhibitor	7	70%
CHD 12. The percentage of patients with coronary heart disease who have a record of influenza vaccination in the preceding 1 September to 31 March	7	85%

2.8.2 Exception reporting within the QOF contract

General practitioners are remunerated for providing quality CHD care. Payment is equally weighted across the socio-demographic strata. Arguably this was designed to eradicate the potential for inequalities in care across CHD populations. The QOF contract does not positively discriminate in favour of or seek to identify individuals who are receiving sub-optimal care or disease management. The QOF contract contains 'exception reporting' which allows general practitioners to receive care payment when they have not seen patients face-to-face. The general practitioners select patients for exclusion against set criteria.

Exception reporting was included in the QOF in order that practices would not be penalised for the characteristics of the patient socio-demographic they serve. Reasons why a patient might be exception reported include:

- patients who have been recorded as refusing to attend review;
- patients who have been invited on at least three occasions during the preceding twelve months;
- patients for whom it is not appropriate to review the chronic disease parameters due to particular circumstances, e.g. terminal illness or extreme frailty;
- patients newly diagnosed within the practice, or who have recently registered with the practice;

- patients who should have measurements made within three months and delivery of clinical standards within nine months, e.g. blood pressure or cholesterol measurements within target levels;
- patients who are on maximum tolerated doses of medication whose levels remain sub-optimal;
- patients for whom prescribing a medication is not clinically appropriate, e.g. those who have an allergy, another contraindication or have experienced an adverse reaction,
- where a patient has not tolerated medication,
- where a patient does not agree to investigation or treatment (informed dissent), and this has been recorded in their medical records,
- where the patient has a supervening condition, which makes treatment of their condition inappropriate, e.g. cholesterol reduction where the patient has liver disease,
- where an investigative or secondary care service is unavailable.

2.8.3 Equity of CHD secondary prevention care and treatment in primary care

Primary care studies have shown that more affluent communities experience a higher standard of care. Failing to match quality and inclusive care to the needs of communities may lead to the inverse care law described by Tudor Hart where communities with the highest prevalence of CHD and other chronic diseases[254; 255] are the least likely to access healthcare services. A 2006 study[256] concludes that exception reporting within the QOF does not incentivise the additional work required to engage and care for individuals of lower SEP in the management of CHD. Furthermore Downing et al's[253] (2007) findings suggest that target-based remuneration of primary care dampens sensitivity to inequalities and will do little to improve the health of disadvantaged groups.

2.8.4 Equity of risk factor monitoring in primary care under the QOF

The quality of primary care studies, which are predominantly based on administrative databases, can be questioned, especially in contrast to studies analysing national health outcomes records. However the limitations of the data sources are acknowledged within the field.

The literature points to the introduction of the QOF in 2004 as having a substantial increase in the proportion of individuals with CHD having regular risk factor reviews[257]; however findings in relation to the socioeconomic, gender and age equity in QOF risk factor review delivery are mixed. In 1998 (pre-QOF) Campbell et al conducted a study within 89 general practices. One of the main findings of the study was that nearly two thirds of the study population had at least two aspects of their health behaviour that would benefit from increased levels of monitoring, however there was little socioeconomic variance in these findings[258].

A 2006 study reported practice-level CHD prevalence was associated with deprivation but that there was no socioeconomic difference in risk factor monitoring[259]. In a 2008 study[260] using data from all general practices in England, no significant socioeconomic inequality risk factor monitoring was found, however neither of these studies considered exception reporting. A study comparing rates of risk factor monitoring between QOF practices in Northern Ireland and non-incentivised primary care in Ireland concluded that cholesterol and blood pressure monitoring was higher in the QOF practices[261].

A 2007 primary care study of 55,522,778 patients in England and Scotland concluded that there were clear socioeconomic differences in risk factor monitoring (particularly where further investigation was required) was higher in GP surgeries in more affluent areas[254]. Furthermore, McLean et al was one of the first studies to identify that exception reporting played a part in masking socioeconomic inequalities in care and treatment within 17 of 33 QOF indicators; including smoking status, blood pressure and cholesterol recording. This seminal study and others[262] conclude that whilst 'payment quality' in isolation suggests no socioeconomic variance in risk factor monitoring. However, the removal of exception reporting, leaving actual 'delivered quality' demonstrates that inequalities in monitoring of these key cardiovascular risk factors persists[256].

2.8.5 Equity of secondary prevention therapies in primary care under the QOF

The literature reviewed demonstrates that secondary prevention therapies prescribing has increased over the past 10 years and as a result of the introduction of the QOF[263;264]. However analysis of the impact of the QOF introduction in 2004 on prescribing demonstrates an already increasing prescription rate of secondary prevention therapies before 2004. It could be argued that this increase in prescribing paralleled the pre- QOF improvements in clinical care and was influenced by national guidelines or local managed clinical networks[265].

In a 2006 seminal paper, Capewell et al[266] highlight that in 2000 barely half of individuals with CHD in England and Wales were receiving optimum secondary prevention and that if 80% had being receiving appropriate therapies then 20,000 deaths may have been prevented or postponed over this period. The paper generally supports the introduction of the QOF.

A 2006 study demonstrated that there were no socioeconomic variances in secondary prevention prescribing[267]. Studies prior to the implementation of the QOF have shown significant socioeconomic inequalities in rates of statin prescribing[268; 269]. However in a 2007, post-QOF study, Ashworth et al[255] demonstrated using a cross-sectional survey of all general practices in England, that socioeconomic inequalities in statin prescribing were not apparent; however older individuals were less likely to be prescribed statin therapies. Similar age inequalities in statin prescribing and other secondary prevention therapies has been an ever emerging theme in the literature in recent years and is supported by other studies[270;271]. Indeed age and gender differences in secondary prevention therapies emerged in a 2003 study where older men and women were less likely to be prescribed optimum therapy combinations and women generally were less likely to receive optimal prescribing compared to men[272].

A randomised control trial within primary care demonstrates the overall improvements that can still be made within therapies prescribing for patients with CHD. This study highlights that even after the introduction of QOF sub-optimal prescribing is still apparent in primary care[273]. A 2006 study using data extractions from 201 UK general practices reported that Prescribing of anti-platelet and statin drugs is at a high level. However, the study noted that there is still scope for improvement in secondary prevention by increasing use of beta-blockers, ACE-inhibitors and other blood pressure lowering drugs in patients who can tolerate them. This and other studies conclude that there are strong age inequalities in secondary prevention prescribing in general and especially amongst individuals with less severe symptoms[274;275].

2.9 Literature Review Summary

The scope and diversity of the literature reviewed in this study is ambitious. This concluding section of the literature review will attempt to briefly synthesise and summarise the key themes and discourse in the literature. Additionally this section will illustrate where the present study fits with the literature reviewed and how the interpretation of this study's findings will add value.

2.9.1 Introduction to CHD

CHD is a complex, multi-faceted disease and its development is influenced by many factors over the life-course. CHD can be thought of as a continuum of the pathological process atherosclerosis. CHD is recognised as a leading cause of mortality and morbidity in both rich and poor countries. Overall rates of CHD have been declining; however the decline has been far from equitable. The decline in rates of CHD has been far less pronounced in individuals or groups of lower SEP. This presents real challenges to effective prevention of CHD; which has strong political support within the UK and other countries. When approaching CHD prevention SEP and thus the measure of SEP is a key consideration.

2.9.2 Measures of Socioeconomic Position

The concept of SEP is fundamental to the thrust of preventative medicine as there are profound socioeconomic gradients across many diseases, care and treatments. The literature search identifies many measures of SEP; all of which have strengths and weaknesses- both theoretically and in practical terms. The quality of national health information collection in Scotland is high and SIMD is an established and validated national measure of SEP. The drive to 'improve' on SIMD in terms of CTVB's potential increase in predictive validity is born purely out of a desire to impact on Scotland's widening inequalities in CHD.

CTVB is worthy of consideration as a surrogate marker of SEP as it has appealing characteristics in comparison to other markers of SEP. The current literature assessing CTVB as a marker of SEP is limited and the quality of the studies is questionable. Only one study has used CTVB in cardiovascular research. The use of CTVB as a marker of SEP in the current study is completely novel. Current discourse in the literature surrounds the influences on health and health behaviours; contextual or compositional. This debate theoretically merges somewhat with evidence exploring the merits of area-based or individual measures of SEP. The quality of health information in Scotland is high

2.9.3 Primary Prevention of CHD

Evidence and policy review suggests it is generally accepted within CHD primary prevention that the highest risk individuals should receive the greatest amount of resource. Given the socioeconomic gradient in CHD mortality, morbidity and exposure to most classical risk factors; individuals or groups of lowest

SEP represent elevated risk. The evidence base for effective primary prevention of CHD is however weak. Particularly there is little evidence relating to identifying and engaging high risk individuals from the wider population. Despite the weaknesses in primary prevention evidence, political support appears unwavering. The use of CTVB as a marker of SEP to identify and target high risk individuals from the wider population is appealing both theoretically and in terms of practical ease-of-use in programme delivery. The present study will assess if CTVB has a stronger association with cardiovascular risk factors and absolute risk than SIMD in an asymptomatic population; this, the literature review suggests, is completely novel research. Due to the characteristics of SIMD and CTVB, the findings of this analysis can be related to the debate within the literature concerning the contextual/compositional influence on health. However within the limits of this study this is a theorisation only. Aside from the interest in CTVB as a proxy marker of SEP, the literature reviewed suggests there remains value in the exploration of socioeconomic inequalities in classical cardiovascular risk factors and absolute risk in this asymptomatic population. Debate within the literature concerns the limited power of classical risk factors in explaining the socioeconomic gradient in CHD; it is beyond the scope of this study to add significantly to this debate. The accuracy of absolute risk measures has been criticised in the literature.

2.9.4 Secondary Prevention of CHD

The evidence base for secondary prevention of CHD is strong, particularly in contrast to that of primary prevention. The QOF was introduced to improve the quality of CHD patients' care and disease management within primary care. The QOF financially incentivises the monitoring of risk factors and appropriate therapies prescribing within CHD populations. The evidence reviewed suggests there are mixed reports as to the equity of QOF implementation in relation to risk factor monitoring and therapies prescribing in established CHD populations. One such focus in this area has been on the notion of "exception reporting"; essentially where a GP receives remuneration when they have not actually seen the patient. Exception reporting is recorded against pre-defined criteria, one of which concerns patients who have not responded to invites for risk and medication review. Limited evidence suggests that this area of exception reporting creates socioeconomic inequalities in those actually accessing review; the QOF does not recognise the extra effort required to engage individuals of low SEP. The present study records individuals who have not responded to invites for risk and medication review (but QOF payment has been made) as having not had risk factor and medication review. With the review data thus filtered, socioeconomic inequality in risk factor monitoring and therapies prescribing rates are assessed using CTVB and SIMD. Evidence reviewed suggests this is novel research. Aside from the exploration of CTVB as a marker of SEP this analyses is important, characterising individuals who are underserved by

current QOF arrangements may have strong implications for the future delivery of CHD secondary prevention within primary care.

CHAPTER 3: METHODS

3.1 Introduction

This chapter gives an overview of Phase 2 of the Have a Heart Paisley (HaHP) project which ran from 2006 to 2008. It provides a detailed explanation of how data used in this thesis was collected in the HaHP project and the subsequent redevelopment of the HaHP CDR in 2009. Also included are details as to the strict ethical approval process adhered to by the study, with relevant documentation included in the appendices.

3.2 Have a Heart Paisley

3.2.1 Phase One

HaHP began as a partnership between NHS Argyll & Clyde, Renfrewshire Council, voluntary and community organizations and the people of Paisley in October 2000. HaHP aimed to provide a uniting focus for action across a broad front to prevent CHD, promote good health and reduce health inequalities in Paisley, Scotland's largest town. Phase One of HaHP took a population wide approach. The aim of this approach was to raise awareness of CHD and its risk factors as well as design interventions targeting those at high risk and to change the risk profile of the whole population. Independent evaluation of Phase 1 was not positive. The evaluation of HaHP and other community health initiatives suggests that targeting the whole population is over-ambitious and the timescales on which it expected to deliver were unrealistic.

3.2.2 Phase Two

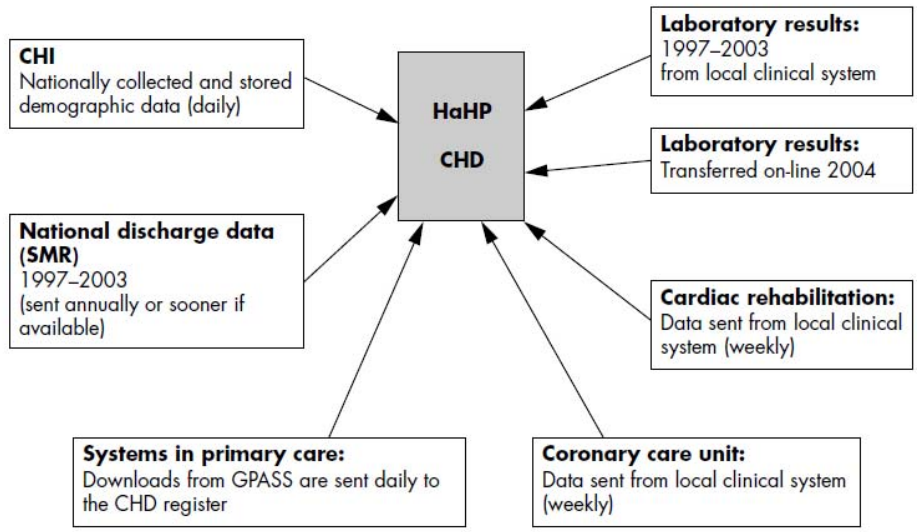
In 2003, The Scottish Government signaled commitment to a second phase of HaHP. In Phase Two, HaHP moved from a population approach to a method that focused support for those most at risk of developing heart disease. The vision for Phase 2 was to deliver, through the combined efforts of its community, voluntary, local authority and NHS partners; primary and secondary preventive interventions that would improve heart health by tackling classical risk factors, with a particular focus on those in deprived communities.

3.2.3 The Have a Heart Paisley Chronic Disease Register

A development in Phase 2 of HaHP that is fundamental to this study is the Chronic Disease Register (CDR). The CDR was set up primarily to identify the primary and secondary prevention populations within Phase 2 of the project, but laterally broadened its scope to be a comprehensive hub of CHD related data for the town of Paisley, with links to lab results, coronary care units, Scottish Morbidity Records and General Practice (GP) systems. Figure 5 below is an extract from a 2005 published editorial describing the CDR[276]. The CDR performed regular electronic extractions of patient data from Paisley GPs. Using primary care read codes the CDR was able to identify individuals aged 45 to 60 who were free from cardiovascular disease and diabetes (primary prevention) and those with CHD of all ages (secondary prevention population).

This study uses risk factor data from the original HaHP primary prevention cohort as identified using the CDR in 2006, and is thus linked to SIMD and CTVB data. The present study does not however use the original secondary prevention population identified in 2006 using the CDR. Instead an updated secondary prevention population has been identified using 2009 data extracted from the CDR. All CDR development, administration and data linkage carried out there in is undertaken by the NHS Greater Glasgow & Clyde Health Information and Technology Department at Westward House, Paisley.

Figure 5: Clark et al, CDR data sources interface



3.3 Read codes used to define primary and secondary prevention populations

Read codes are used to record clinical summary information, their main benefit is that they allow some standardisation of the way information is recorded in primary care. Read codes were developed within a framework of disease areas or chapters. Read codes are 5 characters long and if there is no character after the initial disease area character then the remaining characters are represented by dots. Read codes are organised as a hierarchy; the higher up the hierarchy the less specific the code is, for example:

G…. Circulatory system diseases

G3… Ischaemic Heart Disease

G30.. Acute Myocardial Infarction

G30y, Other Acute Myocardial Infarction

G30y2 Acute Septal Infarction

Exploratory analysis of read code usage in the CDR shows variation between general practices in Paisley. In terms of clinical accuracy the more characters that are present- the more accurately defined the patient's condition is, however this is time consuming for practice staff to ensure this accuracy. Furthermore in terms of audit and disease registers it is more efficient to group disease types using fewer characters within the read codes.

All methods adopted in designing the GP data extractions within the CDR to meet the needs of the study were heavily influenced by the Health Information and Technology Development Department of NHS GG&C and reviewed by the cardiologist and consultant in public health medicine attached to the study.

To identify the primary prevention population, read codes were used in reverse- that is an "is not" operator was used in the query within the CDR to filter patients without cardiovascular (CHD and cerebrovascular diseases) and diabetes (Type 1 and 2) read codes (Read codes- G3***, G6***, C10E, C10F). Patient age was derived from date of birth recorded under "PAT_DOB" field and thus filtered down to individuals aged 45 to 60 years old as of 1st of February 2006.

To identify the secondary prevention population queries were set up within the CDR that enabled the CHD heading to be captured as well as all sub categories of disease there in- G3*** (* denotes 'wildcard'-query returning all read codes beginning with G3). This method has been validated in similar

studies[256]. The data was captured at 31st December 2009 (and at this date for each preceding year of the recording period, 1999-2009, for risk factor recording and therapies prescribing trend analysis only); risk factor monitoring and therapies prescribing are considered for this population in the preceding 12 months (a minimum of 1 recording of the risk factor or review and prescription of therapies was recorded as a 1, where none had occurred over the 12 month period a 0 was recorded).

To establish the rate of risk factor monitoring the following fields within the GP administration system were required to be populated: BP_DAT, (date of blood pressure measure) CHOL_DAT, (date of cholesterol measure) SMOK_DAT, (date of smoking status recording) and BMI_DAT (date of BMI recording). For these data the CDR query was also designed to ensure a valid value was associated with the read code of each risk factor and the date of recording. An important point to note in the analysis of risk factor monitoring is that where an exception code (recorded against any of the four risk factors), which described a patient who had been invited to attend for secondary prevention review on 3 occasions but had not attended; this was recorded within the CDR query as no risk factor monitoring had occurred within the recording year. This decision was taken in line with other studies which aim to highlight the potential for socio-economic inequalities under the existing exception reporting within the QOF contract[256]. As advised by the cardiologist attached to the study, this is the only exception code that was removed as the remaining codes stray into clinical judgement which was deemed unsuitable to comment on within the scope of this study[277-279]. To review QOF implementation datasets and business rules including read codes and exception reporting codes in full, links to electronic resources are provided[280-285] in the references section of the thesis.

To establish the rate of secondary prevention prescribing the following fields within the GP administration system were considered for individuals with an existing CHD diagnoses read code - BB_COD, BB_DAT (beta-blocker prescribed and date of prescription) ACE_COD, ACE_DAT (ACE-inhibitor prescribed and date of prescription), CLO_COD, CLO_DAT (Clopidogrel; anti-platelet prescribed and date of prescription). Querying statin prescribing was more difficult as it is not actually a QOF target in its own right; other than through QOF cholesterol level targets. Statin prescribing was identified using British National Formulary (BNF) drug therapies codes[286] held within GP records. Indeed the Health Information and Technology Development Department of NHS GG&C recommended cross referencing the read code query for anti-platelet, ACE-inhibitor and beta-blocker therapies by using BNF drug therapies codes to identify secondary prevention therapies prescribing. Variance between the data returned using the two methods for these three therapies was negligible. Exception reporting within therapies prescribing was filtered in the exact same manner as risk factor monitoring, described above.

3.4 Linkage of socio-economic indices within the CDR

CTVB data was originally linked, using postcode and address, within the CDR in 2006. A more recent download of Paisley council tax data was requested from the Renfrewshire Joint Valuation Board (RJVB) in February 2009. The RJVB have an obligation to provide council tax data under the freedom of information act however requesting data for the entire town represented a non standard request incurring a £100 administration charge. This charge was paid from the study's budget.

SIMD data zone data from 2006 was held within the Health Information and Technology Development Department of NHS GG&C. SIMD data was linked to CDR using patient postcode. At the time of conducting the analysis (over 2009 and early 2010) the 2006 SIMD data was the most recent SIMD data available. However at the time of writing the new SIMD data gathered in 2009 is now in the public domain.

3.5 CDR Ethics, patient consent and data-linkage

A detailed ethics application including a description of the study with particular attention to the use of patient data and patient consent for this data usage was submitted to the South Greater Glasgow and Clyde (SGG&C) Local Research Ethics Committee (LREC) via the Integrated Research Application System (IRAS) on October 24th 2008. The application supported that the CDR should be updated and data linkage redeveloped in order to ensure accuracy of study findings. The application also explicitly stated that all data extracted from the CDR to be used in this study will be completely anonymous and non-identifiable. The SGG&C LREC then considered the application during their November 2008 meeting and ethics approval was granted on the letter marked 28th November 2008 (Appendix A).

The ethics application submitted recognised that it would be an inefficient use of time and resources to attempt to gather informed consent from the Paisley populations of interest. The application described how awareness raising of the CDR and the option for Paisley citizens to opt out (opt out form is included as Appendix D) of the use of their records in the CDR was posted to all individuals in Paisley with CHD (secondary prevention population) and those deemed to be at risk (the primary prevention population- aged between 45- 60 years old and currently free from cardiovascular disease and diabetes) in 2006. The ethics application recognised that only 2 opt-out forms were received from a target population in the region of 15,000 individuals. Thus it was deemed that reasonable attempt to gather informed patient

consent at a population level had been undertaken[287]. The SGG&C ethics committee were in agreement with this.

In order for data linkages within the CDR to be updated explicit approval was sought from the NHS Greater Glasgow & Clyde (GG&C) Caldicott Guardian. The Caldicott Guardian was thus written to in October 2008 and approval was granted via a hand signed letter in December 2008 (Appendix B). NHS GG&C Research and Development (R&D) sponsorship and approval was also sought and received in December 2008; all supporting documents outlined were forwarded on to R&D at this time.

Additionally consent was sought from Paisley General Practices in order that the CDR could extract their patient records for analysis. The letter sent to Paisley GPs is in Appendix C. Of the 13 practices contacted, 1 practice refused to allow the study access to their patients' records. The reason for refusal was not directly related to concerns over the study or patient confidentiality, rather it was misgivings the GP and practice manager had, based on a prior negative experience of the data extraction method. The practice in question used the EMIS GP electronic records system whereas the rest of the Paisley GPs used the GPASS system. The GP in question stated that a prior extract had created problems in the EMIS system and that the method of extraction was not suitable for the EMIS system. This was regrettable as this meant the study had incomplete data as regards the entire Paisley population. That said the GP's views were respected and data from the practice was not included in any of the analysis.

3.6 Gathering physiological risk data in the primary and secondary prevention population

As described, the primary prevention population was identified using GP read codes to exclude patients with a history of cardiovascular disease and diabetes mellitus. Some 11,270 patients met these criteria. Patients thus selected were invited to participate in the study. They were informed about the study through local media and mass mailings. Of the 11,270 eligible individuals, 1,894 individuals attended screening at a convenient community location and were asked to give informed consent. The location was designed in order to overcome barriers to recruitment faced by those of lower SEP. A questionnaire recorded family history of CVD and some behavioural risk factors including smoking status. In addition, patients underwent a physical examination by a qualified nurse. The examination consisted of blood pressure, cholesterol, weight and height. Blood pressure was measured in a sitting position with a validated sphygmomanometer. Two measurements were taken separated by at least 10 minutes; the mean of these readings was used in the analysis. Cholesterol was measured with a portable 'Cholistech' (Cholistech Corp, Hayward, California) machine; blood samples were taken using a finger pin prick. Height and weight were measured by standard procedures. As recommended in British national guidelines

patients with a Framingham score ≥ 20% were considered high risk[180]. These patients' results were communicated to their GP and a further blood sample was taken and sent to the laboratory to measure fasting glucose, haemoglobin and lipid profiles as part of a more thorough investigation.

Using the Framingham equation[20], each patient's 10 year risk (%) of developing a CVD event was calculated. The formula for Framingham risk includes the following independent variables: gender (male/female), age (in years), systolic blood pressure (mm Hg), serum total cholesterol, and high density lipoprotein (HDL) cholesterol, diabetes mellitus (yes/no), body mass index (kg/m2) and current smoking status (yes/no).

In the secondary prevention population all physiological measures were taken by a qualified practice nurse or the GP themselves.

3.7 Statistical techniques

All data used in analysis was anonymous and non-identifiable in line with ethics approval. All analyses of the primary prevention population were stratified by sex. All analyses were explored initially through box-plots. Linear regression was then used throughout whereby Council Tax Band A was the control from which differences in the distribution of the risk factor or Framingham score were measured against in the other Council Tax Bands (i.e. Bands B to G). Regression analyses were undertaken to:

1) Test the association between SIMD and CTVB.

2) Test the associations between cardiovascular risk factors and CTVB (except for association between current smoking status and CTVB for which logistic regression was used).

3) Test the association between Framingham risk score and CTVB. Likelihood ratio tests were used to test the significance of the associations at a significance level of 0.05.

Analyses in the secondary prevention section of the results were not stratified by sex. Linear regression was used throughout whereby Band A was the control from which differences in risk factor monitoring and therapies prescribing were measured against in the other Council Tax Bands (i.e. Bands B to G) or SIMD quintiles (where quintile 1 was the control to measure against quintiles 2 to 5). The effects of age and sex are adjusted for within the regression analyses to ensure that findings are not skewed by circumstantial variation between council tax bands or SIMD quintiles. Likelihood ratio tests were used to

test the significance of the associations at a significance level of 0.05. Regression analyses were undertaken to:

1) Test the significance of differences between levels of risk factor monitoring (cholesterol, blood pressure, smoking status and body mass index) between council tax bands.

2) Test the significance of differences between levels of secondary prevention therapies (ACE-inhibitor, anti-platelet, beta-blocker and statin) prescribing between council tax bands.

3) To establish if CTVB has an independent contribution over and above SIMD in the above two analyses.

Before adjustment for age was carried out, the linearity of effect with respect to the particular outcome was tested by adding an age squared term into the model. If significant this term was retained in the adjustment to account for the curvi-linear association between age and the outcome in question.

AIC (The Akaike information criterion) are also calculated in multiple regression models. This additional test is included as it is used to compare models. The better fitted model is the one having the smaller AIC value. Differences between models are used to illustrate the degree of preference[288].

The c-statistic is also calculated for analyses involving binary outcomes within the secondary prevention cohort. The c-statistic equals the area under a receiver operating characteristic (ROC) curve and is commonly used to measure the performance of models predicting dichotomous outcomes[289].

All analyses were undertaken using Stata (version 10) statistical analysis software.

CHAPTER 4: RESULTS, THE PREDICTIVE VALIDITY OF CTVB AS A MEASURE OF SEP IN THE PRIMARY PREVENTION OF CHD

4.1 Primary Prevention Population Demographics and Risk factor summary

Table 3: Baseline characteristics and summary of cardiovascular risk factors in asymptomatic men and women aged 45-60 years of age, Paisley, West of Scotland, 2006

	Males	Females
Total	815 (44.1)	1,079 (55.9)
Age (years)	52.4 (4.8)	52.9 (4.6)
Current Smoker†	213 (19.7%)	186 (22.8%)
Family History of CVD†	439 (53.9%)	669 (62.0%)
Systolic BP (mm Hg)	141.9 (16.6)	132.1 (17.4)
Diastolic BP (mm Hg)	88.3 (11.0)	84.4 (11.2)
Serum total cholesterol (mmol/l)	5.4 (0.9)	5.4 (0.9)
Serum HDL cholesterol (mmol/l)	1.2 (0.4)	1.5 (0.4)
Body Mass Index (kg/m^2)	27.6 (4.2)	27.3 (5.1)
Waist (cms)	99.1 (12.0)	89.0 (12.8)
Obesity rate (BMI=30 kg/m^2)	209 (25.6%)	272 (25.2%)
10 year CVD absolute risk (%)	13.5 (7.0)	6.6 (4.5)
Values are mean (SD) or number (%) †Based on patients' self reports BP, blood pressure; CVD, cardiovascular disease; HDL, high density lipoprotein		

Cases utilised: 1,894

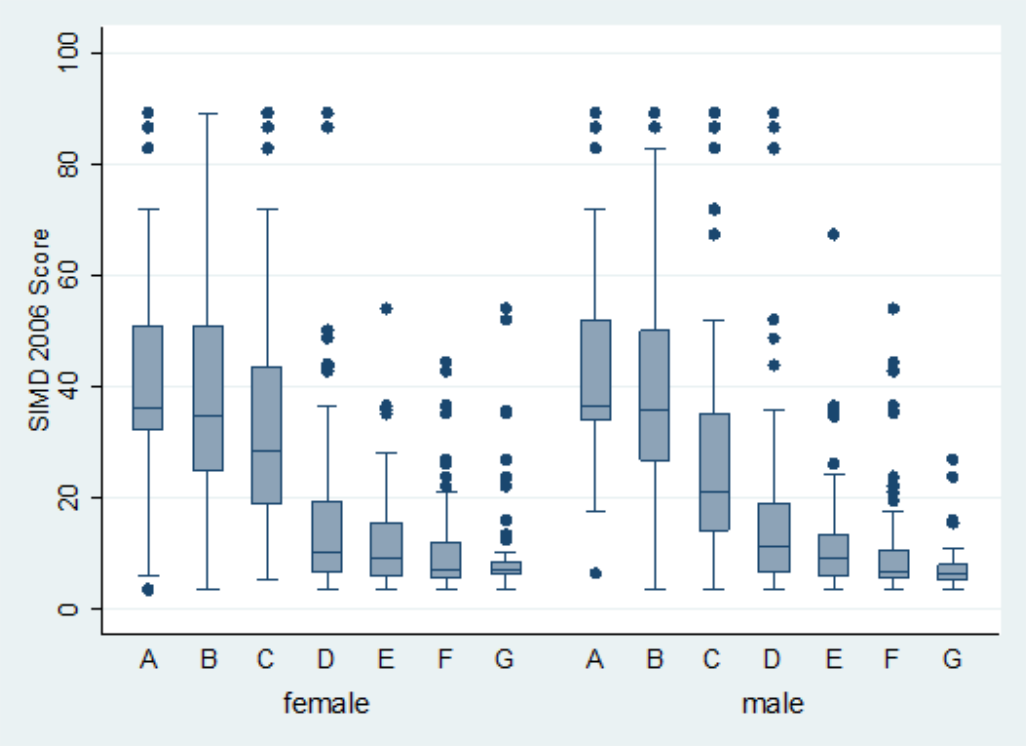
The above table lists the variables of interest in this part of the study. The total sample size is 1,894 and 100% of risk factor data fields are complete i.e. there are no missing cases. All data were linked to CTVB successfully. It is understood that having 100% of data is unusual within health-related research data, however it should be recognised that this data was gathered by a dedicated, specialist evaluation team within a national health demonstration project and is not a secondary data source extract.

Almost two thirds of the study population were women (55.9%).The mean age was: 52.4 years in men and 52.9 in women. Around a fifth were current smokers; 19.7% of men compared to 22.8% of women. More than a half of all men (53.9%) and almost two thirds of the women (62%) reported a family history of CVD.

4.2 Distribution of SIMD Score according to CTVB

This analysis plots the distribution of SIMD score according to CTVB. This is undertaken to establish the association between SIMD and CTVB and assess whether CTVB can be thought of as a marker of SEP. The below box plot charts the distribution of SIMD scores (y-axis) according to CTVB (x-axis):

Figure 6: box plot, distribution of SIMD score according to CTVB by gender



Cases utilised: 1,894

From the box plot it is clear there is an association between SIMD and CTVB. This association will now be formally established using linear regression analysis. As can be seen in the below regression output table there was a highly significant association ($p<0.0001$ and $R^2=0.40$) between CTVB and SIMD Score. As the CTVB increased the mean SIMD and hence average level of socioeconomic deprivation decreased so that the mean SIMD in the lowest value housing was 42.02 for males and 41.2 for females compared to 9.30 for males and 10.32 for females in the highest value housing:

Table 4: Regression of SIMD according to CTVB by gender in Primary Prevention population

CTVB	Male			Female		
	Mean SIMD	95% CI	p-Value	Mean SIMD	95% CI	p-Value
A	42.02	(39.99 to 44.04)	-	41.21	(38.53 to 44.01)	-
B	-5.09	(-8.74 to -1.44)	<0.001	-2.58	(-5.96 to 0.79)	0.13
C	-14.44	(-18.83 to -10.06)	<0.001	-8.25	(-12.28 to -4.22)	<0.001
D	-26.41	(-30.43 to -22.39)	<0.001	-25.09	(-28.79 to -21.39)	<0.001
E	-31.54	(-35.38 to -27.70)	<0.001	-29.69	(33.27 to -26.12)	<0.001
F	-32.51	(-36.62 to -28.41)	<0.001	-29.54	(-33.46 to -25.62)	<0.001
G	-35.51	(-41.08 to -29.94)	<0.001	-30.94	(-35.69 to -26.18)	<0.001

Cases utilised: 1,894

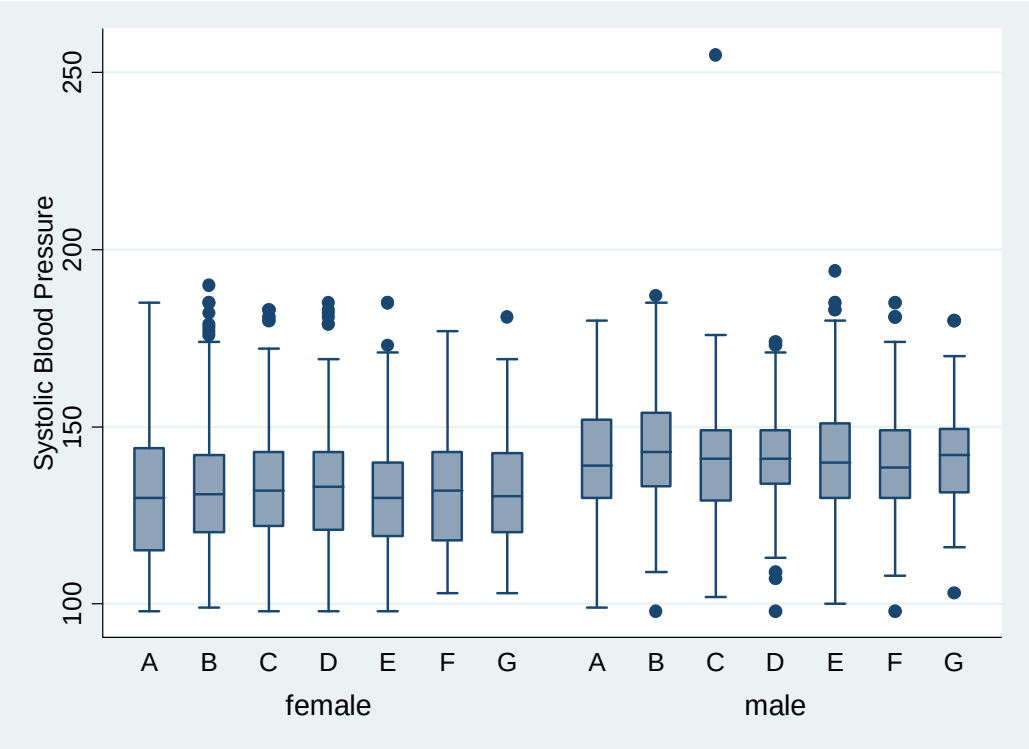
4.3 Distribution of classical risk factors according to CTVB and gender

The distribution of each classical CHD risk factor according to CTVB and gender will now be plotted using a box-plot.

4.3.1 Distribution of systolic blood pressure according to CTVB by gender

Figure 7 below shows the distribution of systolic blood pressure by CTVB for females and males in the primary prevention population:

Figure 7: box plot, distribution of systolic blood pressure according to CTVB in asymptomatic population, by gender



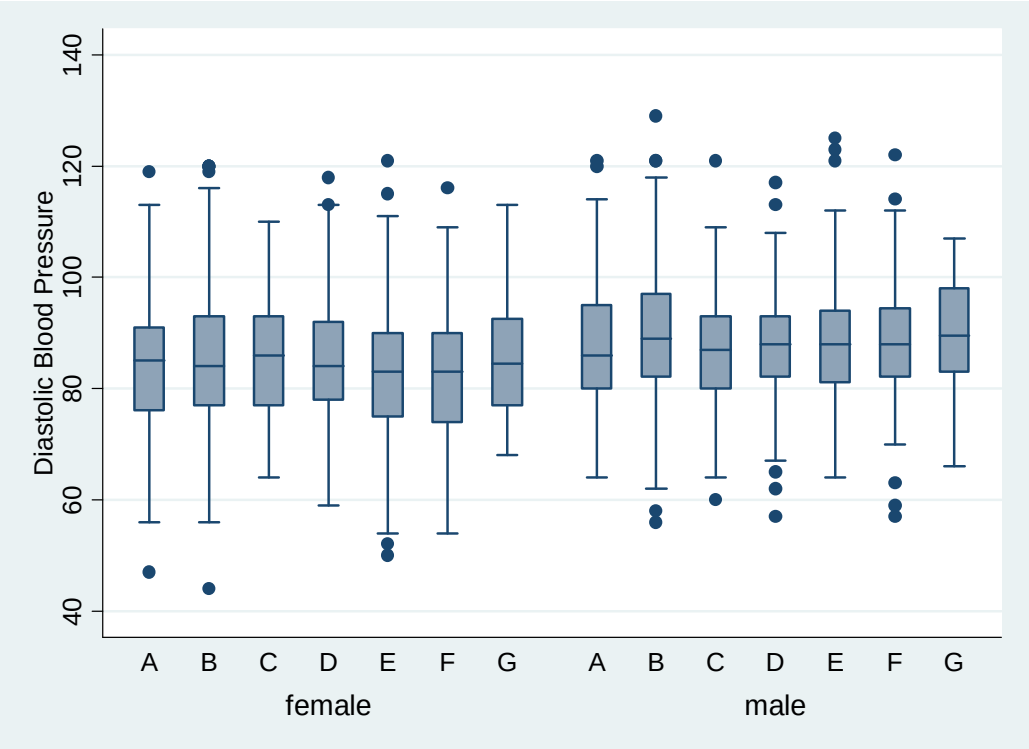
Cases utilised: 1,894

The distribution of systolic blood pressure appears relatively evenly distributed across the CTVB banding for both men and women.

4.3.2 Distribution of diastolic blood pressure according to CTVB by gender

Figure 8 below shows the distribution of diastolic blood pressure according to CTVB for females and males in the primary prevention population:

Figure 8: box plot, distribution of diastolic blood pressure according to CTVB in asymptomatic population, by gender



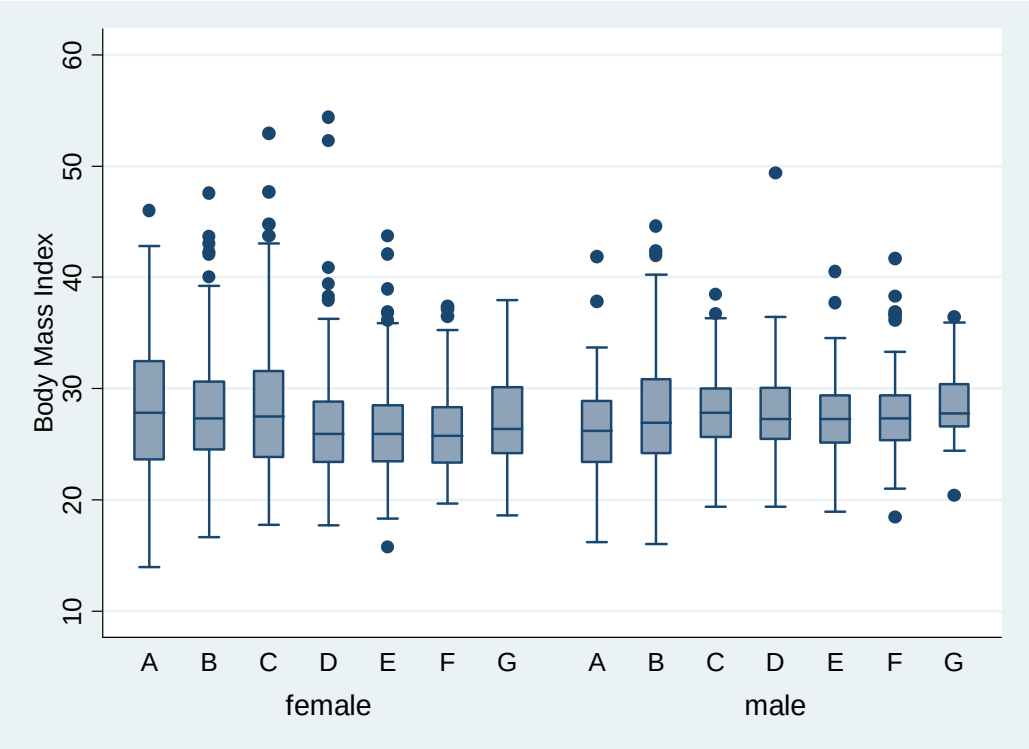
Cases utilised: 1,894

Similarly the distribution of diastolic blood pressure appears evenly distributed across the CTVB; there is a slight gradient in males where diastolic blood pressure in band G seems higher than band A.

4.3.3 Distribution of body mass index according to CTVB by gender

Figure 9 below shows the distribution of body mass index (weight in kilograms divided by height in metres squared) according to CTVB for females and males in the primary prevention population:

Figure 9: box plot, distribution of body mass index according to CTVB in asymptomatic population, by gender



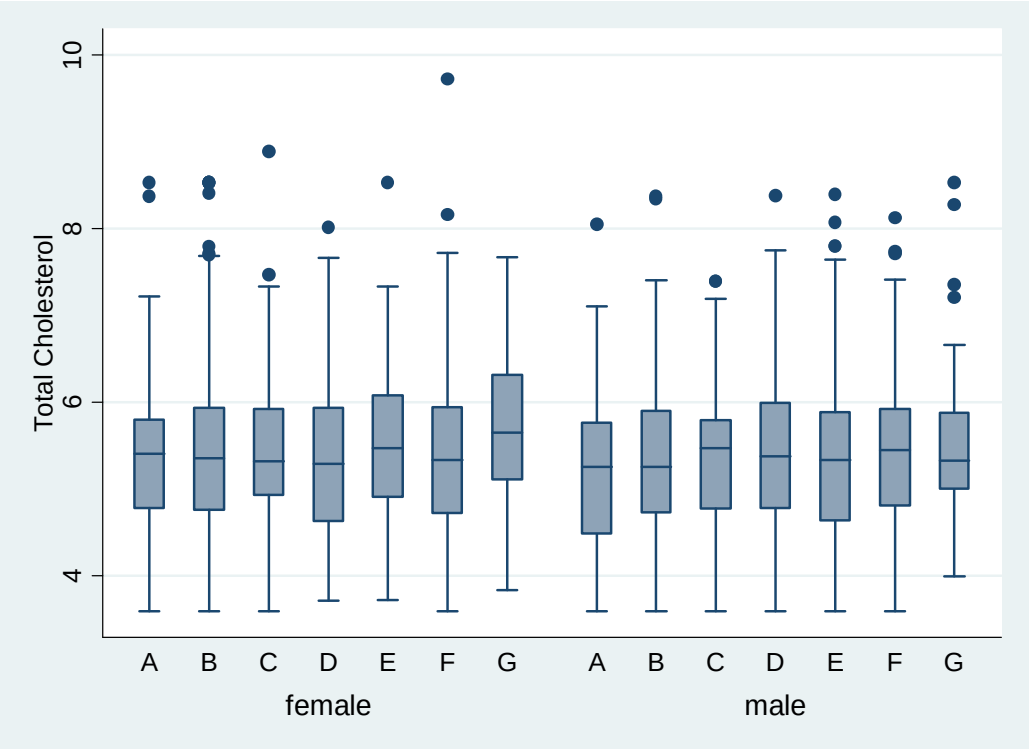
Cases utilised: 1,894

The distribution of BMI across the CTVB is interesting. It would appear from the box plot that females in the lower CTVB (bands A, B and C) have higher BMI than those in higher bands (noticeable E and F). Whilst for males the reverse seems true, BMI appears to increase as CTVB increases.

4.3.4 Distribution of total cholesterol according to CTVB by gender

Figure 10 below shows the distribution of total cholesterol according to CTVB for females and males in the primary prevention population:

Figure 10: box plot, distribution of total cholesterol according to CTVB in asymptomatic population, by gender



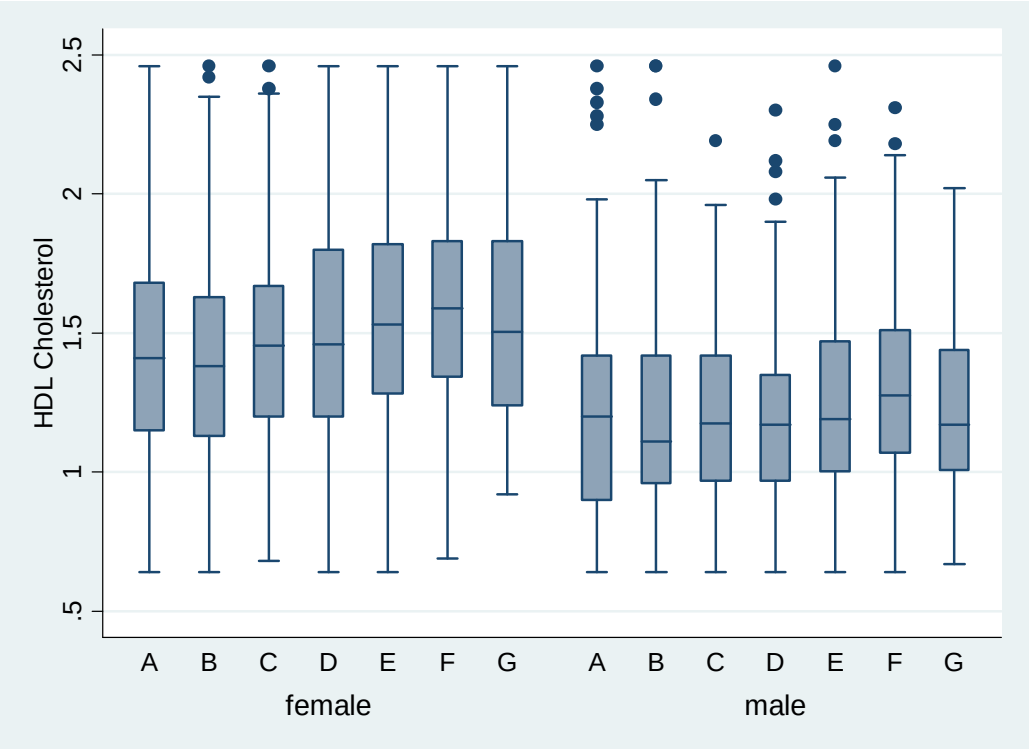
Cases utilised: 1,894

The distribution of total cholesterol appears to slightly increase as CTVB increases, particularly in females. In general the distribution of total cholesterol in each CTVB is quite wide.

4.3.5 Distribution of HDL cholesterol according to CTVB by gender

Figure 11 below shows the distribution of high-density lipoprotein (HDL) cholesterol according to CTVB for females and males in the primary prevention population:

Figure 11: box plot, Distribution of HDL cholesterol according to CTVB, by gender



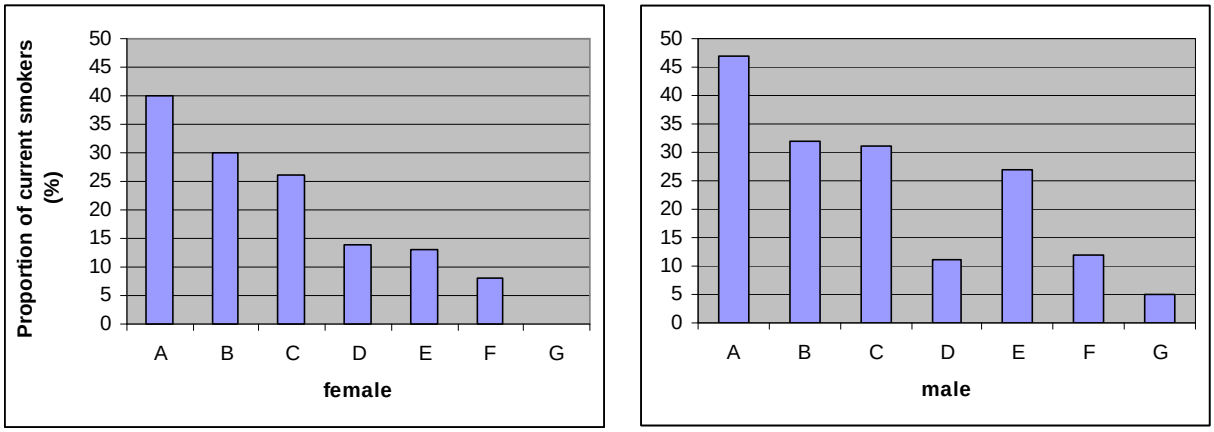
Cases utilised: 1,894

The distribution of HDL cholesterol across CTVB is quite striking in both females and males. It appears that as CTVB increases so too does HDL cholesterol. Females have a broader range of HDL cholesterol values within CTVBs compared to males and have markedly higher values in general.

4.3.6 Distribution of current smokers according to CTVB by gender

Figure 12 below shows the distribution of current smokers according to CTVB for females and males in the primary prevention population:

Figure 12: Distribution of current smokers according to CTVB, by gender



Cases utilised: 1,894

The distribution of current smokers by CTVB is striking with a near perfect socioeconomic gradient in both males and females; where individuals of lower CTVB had much higher rates of smoking.

4.4 Multiple Linear Regression: Distribution of classical cardiovascular risk factors according to CTVB

This regression analysis is undertaken to establish the association between cardiovascular risk factors and CTVB in the primary prevention population. Table 5 however begins by detailing the distribution of risk factors across the council tax bands:

Table 5: Distribution of classical risk factors in asymptomatic primary prevention population according to CTVB

	CTVB	A		B		C		D		E		F		G	
	Value §	Up to £27,000		£27,000 to £35,000		£35,000 to £45,000		£45,000 to £58,000		£58,000 to £80,000		£80,000 to £106,000		£106,000 to £212,000	
Cardiovascular risk factors	Gender														
Systolic Blood Pressure (mm Hg)	M	139.88	1.68	143.99	2.05	142.28	2.46	141.19	2.25	142.5	2.15	139.73	2.3	142.13	3.12
	F	133.59	1.49	133.05	1.84	133.16	2.19	133.1	2.01	130.5	1.94	131.68	2.13	131.8	2.59
Diastolic Blood Pressure (mm Hg)	M	86.88	1.1	89.58	1.35	87.38	1.62	87.81	1.49	87.84	1.42	88.38	1.52	89.88	2.06
	F	84.11	0.96	85.03	1.18	85.69	1.41	85.04	1.29	83.1	1.25	82.7	1.37	85.29	1.67
Body Mass Index (kg/m^2)	M	26.4	0.43	27.74	0.52	27.82	0.62	27.85	0.57	27.33	0.55	27.79	0.58	28.69	0.79
	F	28.09	0.44	27.92	0.54	28.51	0.64	26.82	0.59	26.45	0.57	26.2	0.62	27.01	0.76
Serum total Cholesterol (mmol/l)	M	5.19	0.09	5.32	0.11	5.35	0.14	5.44	0.13	5.35	0.12	5.45	0.13	5.54	0.17
	F	5.37	0.08	5.44	0.1	5.44	0.12	5.36	0.11	5.5	0.1	5.41	0.11	5.71	0.14
Serum HDL Cholesterol (mmol/l)	M	1.22	0.04	1.17	0.05	1.22	0.05	1.2	0.05	1.26	0.05	1.29	0.05	1.34	0.07
	F	1.42	0.03	1.39	0.04	1.45	0.05	1.5	0.04	1.54	0.04	1.61	0.05	1.56	0.06
Current Smokers (%) †	M	46.94	0.5	32.02	0.48	31.4	0.47	10.57	0.31	13.07	0.34	11.61	0.31	5	0.22
	F	40.14	0.49	29.66	0.5	26.27	0.46	14.37	0.44	26.72	0.35	7.63	0.27	0	0

Cases utilised: 1,894

§ based on 1991 housing value

† based on self reporting

Table 6: Output of regression, distribution of classical risk factors by CTVB in the asymptomatic primary prevention population

		From regression, un-adjusted		From regression, adjusted for age and age-squared*		From regression, adjusted for age, age-squared* and SIMD	
Cardiovascular risk factors	Gender	P-value	AIC	P-value	AIC	P-value	AIC
Systolic Blood Pressure (mm Hg)	M	0.32	6902.1	0.4	6894.7	0.33	6896
	F	0.61	9237	0.48	9217.6	0.52	9219.6
Diastolic Blood Pressure (mm Hg)	M	0.39	6224.4	0.48	6223.3	0.49	6225.3
	F	0.17	8288.3	0.16	8290.4	0.32	8291.1
Body Mass Index (kg/m^2)	M	0.056	4668.2	0.053	4672	0.12	4672.5
	F	<0.001	6583.4	<0.001	6587	0.13	6584
Serum total Cholesterol (mmol/l)	M	0.3	2192.04	0.26	2194.6	0.82	2192.8
	F	0.17	2888.4	0.4	2837	0.28	2837.6
Serum HDL Cholesterol (mmol/l)	M	0.14	671.4	0.13	670.3	0.34	672.1
	F	<0.001	1017.9	<0.001	1015.4	0.05	1009.2
Current Smokers (%) †	M	<0.001	809	<0.001	806.8	<0.001	800.1
	F	<0.001	1020	<0.001	996.1	0.03	944.3
*age-squared term proved to have significant association with all therapies prescribing and was thus retained in adjusted models to account for the curvi-linear association between age and the therapy prescribing outcome in question							

Cases utilised: 1,894

Table 6 above details the output of risk factor regression analysis. The table contains p-values as well as AIC values. The values are presented under the three models were the regression distribution of the risk factor variables are undertaken in unadjusted, adjusted for age and age-squared and adjusted for age, age-squared and SIMD models. BMI (females, marginal significance in males), HDL Cholesterol (females) and rates of current smokers (males and females) all proved to have significant association with CTVB in the unadjusted model.

These associations remained relatively unchanged when adjusting for age and age-squared in the second model. However based on the p-values, the addition of SIMD into the third model tends to weaken the

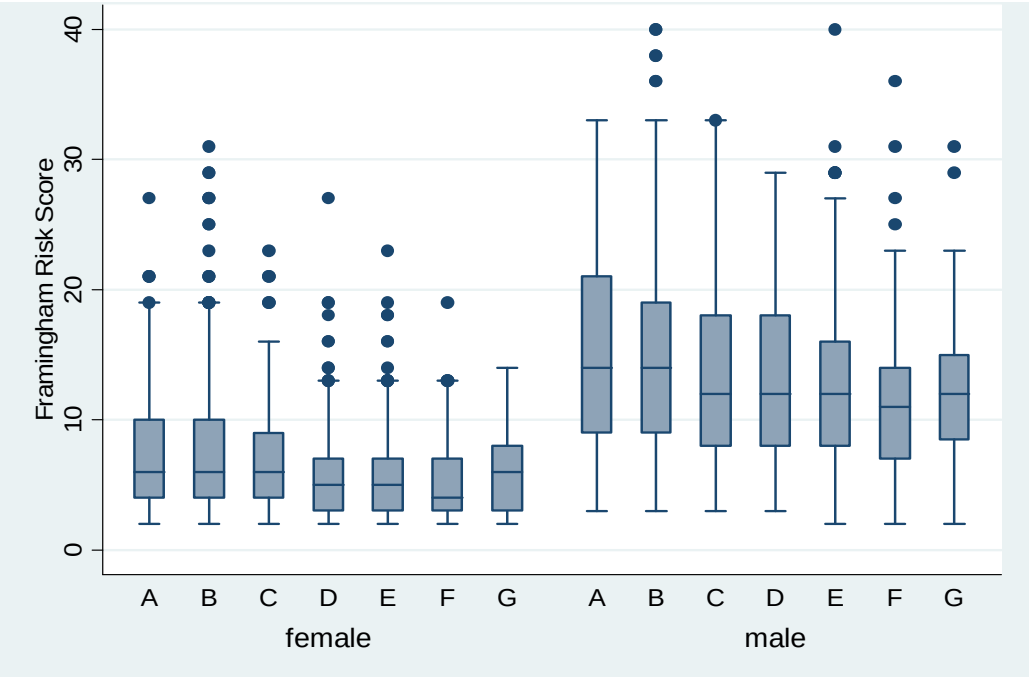
independent contribution of CTVB with BMI (males and females) HDL cholesterol (females) and current smokers (females).

The smaller AIC of 6584 with a model that includes age, sex, CTVB, SIMD compared with AIC of 6587 with a model that includes age, sex, CTVB indicates that SIMD is improving model fit; the difference in models being 3. Hardin and Hilbe[288] state that a difference in AIC values of greater than 2 and less than 8 between models is a 'positive' degree of preference. Similar positive preferences in differences between AIC values for the same model comparison are evidenced in HDL Cholesterol in females (6.2 difference in AIC value) and rates of current smokers in males (6.7 difference in AIC value). However the difference in AIC values for the same model comparison in female current smoker rates is 51.8, indicating a very strong preference for the model with SIMD included.

4.5 Regression modelling: Distribution of Framingham absolute cardiovascular risk according to CTVB

The following analysis concludes this results chapter and is undertaken to assess the strength of association between Framingham risk and CTVB. Firstly a box-plot is used to explore the association and finally regression modelling is used to quantify the association.

Figure 13: box plot, distribution of absolute cardiovascular risk (measured by Framingham Risk Score) according to CTVB in asymptomatic primary prevention population, by gender



Cases utilised: 1,894

There appears to be a socioeconomic gradient in Framingham risk score in both men and women; Framingham score is higher in both men and women in the lower value housing (Bands A, B and C) compared to the Framingham risk scores in the higher value housing (Bands E, F and G), however Band G (the highest value properties) seems to go against this gradient somewhat as it would appear that in both men and women the Framingham risk tends to increase going from Band F to Band G.

Table 7: Regression analysis, association between Framingham risk and CTVB in asymptomatic primary prevention population, by gender

Model 1. From regression of CTVB and Framingham Risk, adjusted for age and age squared*						
	Males			Females		
CTVB	Reg Coeff	95% CI	p-value	Reg. Coeff	95% CI	p-value
			<0.001			<0.001
B	-0.39	(-1.93 to 1.15)	0.617	0.31	(-0.56 to 1.17)	0.486
C	-1.42	(-3.27 to 0.42)	0.131	-0.34	(-1.37 to 0.69)	0.52
D	-2.46	(-4.15 to -0.77)	0.004	-1.51	(-2.45 to -0.56)	0.002
E	-2.55	(-4.16 to -0.93)	0.002	-1.94	(-2.85 to -1.03)	<0.001
F	-3.39	(-5.12 to -1.66)	<0.001	-2.4	(-3.41 to -1.40)	<0.001
G	-2.95	(-5.30 to -0.61)	0.014	-1.96	(-3.18 to -0.74)	0.002
Model 1	R^2=0.17, AIC=5340.0			R^2=0.13, AIC=6198.7		
Model 2. From regression of CTVB and Framingham Risk, adjusted for age, age squared*and SIMD						
	Males			Females		
CTVB	Reg Coeff	95% CI	p-value	Reg Coeff	95% CI	p-value
			0.01			0.04
B	-0.38	(-1.93 to 1.17)	0.629	0.41	(-0.45 to 1.26)	0.349
C	-1.39	(-3.28 to 0.51)	0.151	0	(-1.03 to 1.03)	0.998
D	-2.4	(-4.26 to -0.54)	0.012	-0.51	(-1.52 to 0.50)	0.326
E	-2.47	(-4.33 to -0.61)	0.009	-0.77	(-1.78 to 0.25)	0.138
F	-3.31	(-5.28 to -1.34)	0.001	-1.23	(-2.46 to 0.55)	0.027
G	-2.87	(-5.44 to -0.30)	0.028	-0.76	(-2.05 to 0.53)	0.247
Model 2	R^2=0.17, AIC=5342.0			R^2=0.15, AIC=6129.0		

Cases utilised: 1,894 *

age-squared term proved to have significant association with all therapies prescribing and was thus retained in adjusted models to account for the curvi-linear association between age and the therapy prescribing outcome in question

Model 1.of table 7 tests the association between Framingham cardiovascular risk score and CTVB in men and women adjusting for age and age squared. Model 2 tests the association between Framingham cardiovascular risk score and CTVB in men and women adjusting for age, age squared and SIMD. Each regression co-efficient (B through to G) represents the difference in Framingham score between the given band and band A. The p-value in the first row of each table represents the significance of the overall associations.

The analysis found that CTVB was a significant predictor of Framingham cardiovascular risk score in both men and women (Model 1) but according to the R-squared values that the addition of SIMD into the model (Model 2) improved its predictive value in women. In men CVTB alone was an independent predictor of cardiovascular risk and the model did not improve on addition of SIMD (Model 2). The AIC values support this also where the addition of SIMD, moving from model 1 to model 2 increased the AIC value by 2 for men; thus demonstrating a weakening model fit. The AIC values for women within the same model comparison demonstrate massively improved model fit with the addition of SIMD. CTVB thus adds predictive power (over and above SIMD) of cardiovascular risk in men but not in women.

CHAPTER 5: RESULTS, THE PREDICTIVE VALIDITY OF CTVB AS A MEASURE OF SEP IN THE SECONDARY PREVENTION OF CHD

5.1 Secondary prevention population demographics

Table 8 details the demographics of the secondary prevention population. The majority of individuals with CHD in Paisley at the recording point in 2009 were male (54.9%) and the male population was significantly younger than the female population. The majority of individuals with CHD are from council tax bands A and B. There is no other demographic information available on this population for ethical reasons. Based on an estimated population size the approximate prevalence of CHD in Paisley is 3.7%, which is similar to CHD prevalence recorded in primary care in other UK studies[243] (however this estimate is based on data from 12 of 13 GP practices in Paisley and with thus be higher if this data were available).

Table 8: Secondary prevention population demographics

	Male	Female
Total	1,739 (54.86)	1,431 (45.14)
Mean age	67.7 (2.91)	72.6 (3.12)
CTVB A	398 (22.9)	314 (21.9)
CTVB B	873 (50.2)	669 (46.8)
CTVB C	199 (11.4)	158 (11.0)
CTVB D	115 (6.6)	123 (8.6)
CTVB E	85 (4.9)	108 (7.5)
CTVB F	38 (2.2)	34 (2.4)
CTVB GH	31 (1.8)	25 (1.7)

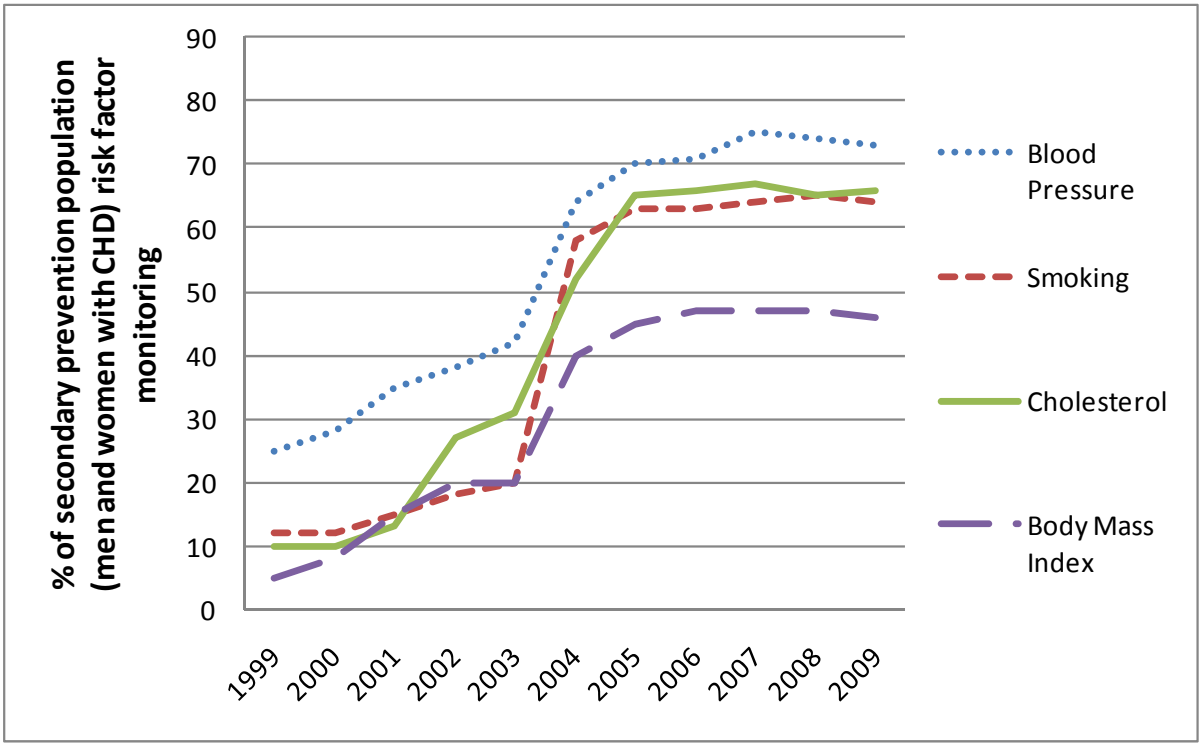
Cases utilised: 3,170

Assessing if there are any missing cases within the secondary prevention primary care extract is problematic. The extract is entirely of a binary nature, i.e. a "1" represents the presence of an appropriate value within the primary care risk factor recording field; thereby meaning that the risk factor review had taken place. Similarly for the therapies prescribing a "1" represents appropriate values in the therapies prescribed and review date primary care fields; thereby meaning that the therapies review had taken place and the given medication was prescribed. Where a "0" is returned, in both instances, it means that the relevant primary care fields did not contain any values and therefore the risk-factor review or therapies review had not taken place. It is indeterminate from the format of the extract whether a proportion of the "0" values are potentially missing cases; where the review had taken place but the values were not entered within the practice. However every effort was taken to ensure that this extract is accurate and the potential for missing cases (and determining the quantity of missing cases) is out with the control of the study. Given that the recording of risk factors and prescribing is financially incentivized the potential for missing cases is small.

5.2 Distribution of risk factor monitoring in primary care by both CTVB and SIMD

Figure 13 below shows that overall risk factor recording has risen dramatically over 1999-2009, especially after the introduction of the QOF in 2004:

Figure 14: Risk factor monitoring in men and women in Paisley with CHD excluding exception reporting of failure to attend review over 1999 to 2009



As described in the methods chapter this analysis does not include patients who have been recorded as not attending review who have been invited on at least three occasions during the reporting period and have been recorded under exception reporting. Analysis of the risk factor monitoring rates in 2009 showed that after exception reporting is removed none of the monitoring levels reached the QOF targets (detailed in figure. 4). Blood pressure recording shows that 72.8% of the secondary prevention population had at least one measure of this risk factor taken in 2009- 17.2% below the 90% QOF target. Cholesterol recording in 2009 was 66.1%, some 23.9% below the QOF target. Smoking status monitoring was at 64% within the target population; 26% below the QOF target. The proportion of the target population having body mass index recorded in 2009 was 46%.

Rates of risk factor monitoring for cholesterol, blood pressure, smoking status and body mass index within the secondary prevention population will now be analysed according to CTVB. Due to low numbers (as evidenced in the table within section 5.2) Bands G and H are combined throughout this analysis.

Table 9: Rates of Risk factor monitoring by CTVB in men and women in Paisley with CHD excluding exception reporting of failure to attend review in 2009

CTVB	A	B	C	D	E	F	GH
Value of Housing*	Up to £27,000	£27,000 to £35,000	£35,000 to £45,000	£45,000 to £58,000	£58,000 to £80,000	£80,000 to £106,000	£106,000 +
Cholesterol	490 (68.8)	919 (59.6)	280 (78.4)	168 (70.6)	147 (76.2)	62 (86.1)	45 (80.4)
Blood Pressure	485 (68.1)	1168 (75.8)	267 (74.8)	181 (76.1)	142 (73.6)	57 (79.2)	41 (73.2)
Smoking Status	440 (61.8)	984 (63.8)	217 (60.8)	147 (61.8)	132 (68.4)	54 (75.0)	44 (78.6)
Body Mass Index	287 (40.3)	696 (45.1)	180 (50.4)	116 (48.7)	79 (40.9)	47 (65.3)	32 (57.1)

Cases utilised: 3,170

Table 10: Regression output for risk factor monitoring by CTVB in men and women in Paisley with CHD excluding exception reporting of failure to attend review in 2009

	From regression, un-adjusted			From regression, adjusted for age, age-squared* and sex			From regression, adjusted for age, age-squared*, sex and SIMD		
	P-value	AIC	C-stat	P-value	AIC	C-stat	P-value	AIC	C-stat
Cholesterol	0.006	2854.9	0.55	0.007	2820.5	0.59	0.12	2814.3	0.6
Blood Pressure	0.05	2906.3	0.54	0.01	3293.2	0.56	0.09	3276.5	0.58
Smoking Status	0.03	3297.7	0.54	0.04	2802	0.63	0.04	2806.8	0.63
Body Mass Index	0.03	3049.4	0.53	0.045	3047.1	0.55	0.4	3034.6	0.58
*age-squared term proved to have significant association with all therapies prescribing and was thus retained in adjusted models to account for the curvi-linear association between age and the therapy prescribing outcome in question									

Cases utilised: 3,170

Table 6 above details the output of risk factor recording regression analysis. The table contains p-values, AIC values and C-statistic values. The values are presented under the three models like that of table 6. The findings of this analysis are that (using CTVB) socioeconomic inequalities in risk factor recording

are evident in the first two models but when SIMD is introduced in third model on smoking status remains significant.

According to the p-values all risk factor proved to have significant association with CTVB in the unadjusted model. These associations tended to weaken slightly (but remained significant) when adjusting for age, age-squared and sex in the second model, with the exception of blood-pressure, where its p-value reduced from 0.05 (unadjusted) to 0.01 (adjusted).

However based on the p-values, the addition of SIMD into the third model tends to weaken the independent contribution of CTVB with all risk factor recording (making the contribution insignificant) with the exception of smoking status, where it's p-value remained un-changed after the introduction of SIMD into the regression model and still significant (0.04).

The smaller AIC values in model 3 which includes age, sex, CTVB, SIMD compared to that of model 2 including age, sex and CTVB indicates that SIMD is improving model fit. The exception to this is smoking status where AIC value increases from 2802.0 to 2806.8 moving from model 2 to 3. This demonstrates that CTVB has an independent contribution to smoking status recording. There is no increase in the c-statistic for smoking status recording moving from model 2 to 3, demonstrating that the addition of SIMD into the model adds no predictive power for smoking status recording. The c-statistic for the rest of the risk factors increase slightly over the same comparison further demonstrating the addition of SIMD improves predictive power.

5.3 Distribution of secondary prevention therapies prescribing in men and women in Paisley with CHD excluding exception reporting of failure to attend review

Figure 15 below shows secondary prevention therapies prescribing in primary care amongst the secondary prevention population over the period 1999 to 2009. All therapies have significantly increased over the recording period particularly at 2004. However all therapies prescribing are below QOF targets once selected exception reporting as detailed in the method section are removed. Analysis of 2009 data reveals that anti-platelet prescribing was at 72.3%, some 17.7% below the QOF target, beta-blocker prescribing was at 45%, 5% below the QOF target, ace-inhibitor prescribing was at 62.9%, some 7.1% below the QOF target, statin prescribing was at 53%- there are no specific targets for statin prescribing other than the 60% target of patients with cholesterol of 5.0 mmol/l.

Figure 15: secondary prevention therapies prescribing in men and women in Paisley with CHD excluding exception reporting of failure to attend review over the period 1999 to 2009

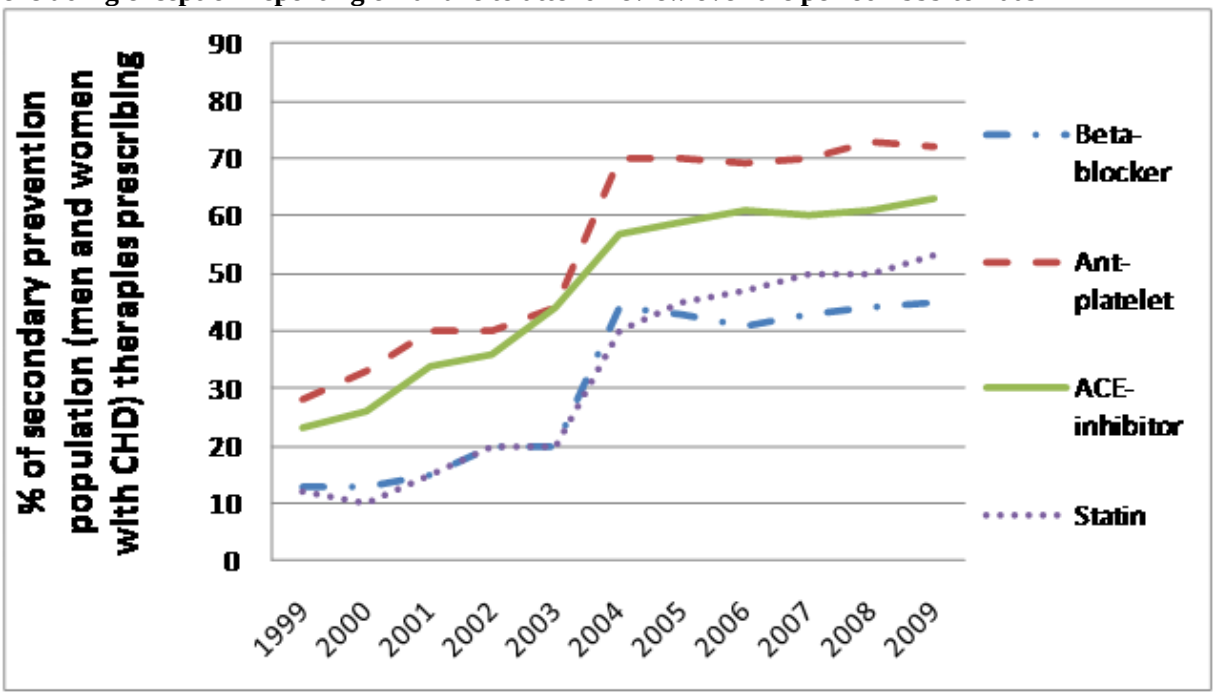


Table 11: Rates of secondary prevention therapies prescribing by CTVB in men and women in Paisley with CHD excluding exception reporting of failure to attend review in 2009

CTVB	A	B	C	D	E	F	GH
Value of Housing*	Up to £27,000	£27,000 to £35,000	£35,000 to £45,000	£45,000 to £58,000	£58,000 to £80,000	£80,000 to £106,000	£106,000 +
Ace-inhibitor	444 (62.4)	955 (61.9)	219 (61.3)	164 (68.9)	124 (64.2)	40 (55.6)	42 (75.0)
Anti-platelet	533 (74.9)	1045 (67.8)	247 (69.2)	184 (77.3)	149 (77.2)	52 (72.2)	43 (76.8)
Beta-blocker	324 (45.5)	646 (41.9)	201 (56.3)	94 (39.5)	105 (54.4)	40 (55.6)	37 (66.1)
Statin	362 (50.8)	830 (53.8)	161 (45.1)	147 (61.8)	104 (53.9)	48 (66.7)	35 (62.5)

Cases utilised: 3,170

*based on 1991 value

Table 12: Regression output for secondary prevention therapies prescribing by CTVB in men and women in Paisley with CHD excluding exception reporting of failure to attend review in 2009

	From regression, un-adjusted			From regression, adjusted for age, age-squared* and sex			From regression, adjusted for age, age-squared*, sex and SIMD		
	P-value	AIC	C-stat	P-value	AIC	C-stat	P-value	AIC	C-stat
Ace-inhibitor	0.39	1247.6	0.55	0.7	1217.3	0.63	0.58	1214.5	0.65
Anti-platelet	0.56	1544.4	0.53	0.51	1463.7	0.67	0.73	1463.8	0.68
Beta-blocker	0.07	1281.9	0.56	0.11	1220.2	0.66	0.12	1226.2	0.69
Statin	0.04	1539.4	0.55	0.04	1437.1	0.69	0.02	1438.8	0.69
*age-squared term proved to have significant association with all therapies prescribing and was thus retained in adjusted models to account for the curvi-linear association between age and the therapy prescribing outcome in question									

Cases utilised: 3,170

Table 11 above details the output of therapies prescribing regression analysis. The table contains p-values, AIC and C-statistic values. The values are presented under the three models like that of tables 6 and 9. The findings are that only Statin prescribing displayed significant socioeconomic (using CTVB as measure of SEP) variance, this was evidenced throughout the three models.

According to the p-values, all therapies prescribing with the exception of statins (although beta-blockers were marginal (p=0.07)) proved to have insignificant associations with CTVB in the unadjusted model. These associations tended to remain unchanged when adjusting for age and age-squared in the second model, with the exception of ace-inhibitor, where its p-value increased from 0.39 to 0.70.

Based on the p-values, the addition of SIMD into the third model tends to weaken the independent contribution of CTVB within anti-platelet and beta-blockers prescribing, but increases the contribution with ace-inhibitor and Statins. The independent contribution of CTVB with statin prescribing remained significant (p=0.02) in the third model adjusting for age, age-squared*, sex and SIMD, but was insignificant for ace-inhibitors (p=0.58). This indicates that CTVB has an independent contribution to statin prescribing, but has not for the rest of the therapies.

The smaller AIC values in model 3 for ace-inhibitor prescribing indicates that SIMD is improving model fit for this therapy prescribing rate. However the AIC values for the rest of the therapies tend to increase very slightly indicating that the addition of SIMD adds nothing to model fit. The c-statistic for therapies

prescribing remained relatively unchanged moving from model 2 to 3 indicating little predictive power with the addition of SIMD to the model.

CHAPTER 6: DISCUSSION

6.1 Revisiting the study aims

The aims of the study were to examine the socioeconomic distribution of absolute cardiovascular risk and classical risk factors in an asymptomatic population and to explore inequalities in risk factor monitoring and therapies prescribing within primary care in an established CHD population. In both population analyses the overarching aim of the study concerns the predictive validity of CTVB as a surrogate marker of SEP and its strength of association within these analyses will be compared to SIMD- an established measure of SEP in Scotland. The analysis began by examining the association between CTVB and SIMD.

6.2 Main findings of the study

6.2.1 The predictive validity of CTVB as a marker of SEP in the primary prevention population

The association between CTVB and SIMD was undertaken using data from the primary prevention population to establish the extent to which CTVB- the hypothesized marker of SEP correlated with a nationally established measure of SEP. The results of this analysis (p-value<0.0001, R^2=0.40) are almost identical to the association explored in another study between CTVB and the Jarman Index[133] (p-value<0.0001, R^2= -0.42); CTVB increased as the Jarman Index reduces, thereby both moving in the direction of reducing deprivation. Thus findings from the present study support those of the Jarman study; that it is reasonable that CTVB is considered as a marker of SEP.

Similar to well established literature in the field[187-225], the distribution of some cardiovascular risk factors in this study demonstrated significant socioeconomic variance; where individuals of lower SEP had worse risk factor profiles than those of higher SEP. Using CTVB as surrogate marker of SEP both HDL cholesterol and BMI levels displayed statistically significant socioeconomic variance in women but not men; however the association proved insignificant once the effects of SIMD were adjusted for. These findings are somewhat consistent with literature reviewed; whereby the inverse association between SEP and cholesterol is demonstrable but varied and somewhat unclear[202-206], However, the association between SEP and obesity in women but not men is consistent with the literature reviewed[223-225].

Remarkably similar to another study[134] investigating CTVB as a surrogate marker of SEP in general practice, the socioeconomic distribution of current smokers according to CTVB was striking; representing a near perfect gradient where both men and women (p-value < 0.001) in the lowest value council tax band

(band A) had significantly higher rates of current smokers (49.94% and 40.14% respectively) compared to men and women in the highest value council tax band; band G (5.00% and 0% respectively). The association between CTVB and rates of current smokers remained after adjustment for both age, age-squared and SIMD. The socioeconomic gradient seen in rates of current smokers in this study is consistent with well established literature in the area[216-219].

Blood pressure, both systolic and diastolic did not vary significantly between council tax bands in either men or women in the present study. Interestingly, this is not consistent with the vast majority of literature in the field where blood pressure and SEP have a definite inverse relationship[207-210].

Comparing the strength of association between CTVB and Framingham risk and SIMD and Framingham risk revealed some noteworthy results. Consistent with all studies reviewed Framingham risk has an inverse relationship with SEP[20; 179; 229; 232]. In the present study CTVB had a significant association with Framingham scores in both men and women. However it was concluded that in women CTVB did not add predictive power over the association evidenced between SIMD and Framingham risk score. However in men adding CTVB did add predictive power over the association between SIMD and Framingham risk score. Hence, CTVB proved to have an independent association with Framingham risk in asymptomatic men (but not women) aged between 45 to 60 years.

There are no studies examining the association of CTVB and asymptomatic cardiovascular risk factors or absolute risk with which to compare the findings of the present study. However CTVB's independent association with Framingham risk in men and smoking rates in men and women are somewhat supportive with the conclusions of limited literature in the field; that CTVB is worthy of consideration as a marker of SEP in health research and may have utility ahead of aggregated or area-based measures of SEP[133-138]. The present study's findings are however generally less convincing than those summarised in the literature review. The questionable accuracy of absolute measures of cardiovascular risk[234] casts caution on these results.

Overall, the associations with cardiovascular risk factors and CTVB in the present study are weaker than the associations seen in other studies using established markers of SEP; income, education, occupation and other housing markers[61;64-66;83;91;92;102;103;187-225]. Importantly the lack of significant variance in blood pressure between bands[207-210] casts doubt over CTVB as reliable marker of SEP in cardiovascular research.

6.2.2 The predictive validity of CTVB as a marker of SEP in the secondary prevention population

Similar to literature reviewed, risk factor (cholesterol, blood pressure, BMI and smoking status) monitoring has increased dramatically over the period 1999 to 2009 in the present study[248;252]. Again similar to the evidence reviewed; a marked rise is observed around 2004 when the QOF contract was introduced- thereby rewarding general practices for monitoring CHD patients' risk factors and attaining targets within levels of risk factors. When removing exception reporting from the totals of risk factor recordings it is clear that actual risk factor monitoring levels seen in 2009 were below the QOF target levels. These methods adopted in the present study are identical to that of McLean et al[256] whereby the concepts of 'payment quality' versus 'actual delivery quality' in secondary prevention under the QOF are explored. By adopting this method the present study highlights a number of inequalities that may have been potentially 'masked' through current QOF exception reporting arrangements.

Using CTVB as of the measure of SEP, significant socioeconomic variances in cholesterol, blood pressure, body mass index and smoking status were observed in model 2 of the regression analysis within the secondary prevention population. Socioeconomic inequalities in risk-factor monitoring are consistent with some[254; 256] but not all[258-260] of the literature reviewed. CTVB's independent contribution to smoking status monitoring remained significant even when SIMD was introduced to the regression modelling in model 3.

CTVB proved to have an independent contribution to the likelihood of Statin prescribing within the secondary prevention population. This contribution remained significant over the three models. CTVB did not have a significant independent contribution to prescribing rates within the remaining therapies.

Reviewing the distribution of secondary prevention outcome variable rates across CTVB is crucial in interpreting the regression modelling analyses. Where CTVB's significant independent contribution is established with secondary prevention outcomes variables it appears that higher (more affluent) council tax bands have higher rates of the outcome measures in question thus experiencing better care and treatment than the lower (more deprived) council tax bands.

There are no studies with which to directly compare the use of CTVB as a marker of SEP in analyses exploring inequalities in risk factor recording and therapies prescribing in secondary prevention populations.

6.3 Implications of the main findings of the study for CHD prevention

The synthesis of evidence in the literature review of the present study has raised important considerations within CHD primary prevention. In particular there is a paucity of reliable research or evaluation relating to the process of effective CHD primary prevention delivery. Reviewing the evidence identified a number of issues that make primary prevention studies challenging to compare and findings difficult to generalise. These include differences in the reporting of recruitment, enrolment, and retention information; inconsistencies in the use of terminology and reporting of physiological and behaviour measures and variations within comparable measures of SEP across studies, and the complexity of the literature which covers disparate samples of socio-demographic compositions representing different risks, different sub categories of cardiovascular disease and risk factors, definitions of risk and study types.

Findings of this study support evidence that socioeconomic inequalities in asymptomatic cardiovascular risk persist. Socioeconomic inequalities in asymptomatic cardiovascular risk seen in this study are weaker than the established evidence but this is perhaps as a result of using CTVB as the marker of SEP as opposed to a validated measure of SEP. Overall, the socioeconomic inequalities in classical risk factors within the present study highlight the need for primary prevention interventions to effectively target and positively discriminate resource allocation in favour of deprived communities or individuals of lower SEP. This requires a substantial improvement in current evidence; specifically in identifying and engaging low SEP communities and individuals and improving intervention engagement, efficacy and outcomes within prevention strategies[16]. Furthermore the socioeconomic gradients in risk factors (particularly smoking in the present study) and absolute risk suggest that the thrust of primary prevention activity in low SEP areas should remain on classical risk factors. This is especially true when considering the life course exposure to such risk within deprived communities[181; 182]. A key challenge to public health is perhaps to remain focussed on effectively applying what is known already; in terms of reducing exposure to classical risk factors whilst continuing to further develop understanding of the disease through research into novel risk factors and the wider influences and determinants of CHD development.

Interestingly female absolute risk showed equal association with CTVB and SIMD. This suggests that the female risk profile within the current study is influenced by contextual and compositional factors in equal measures. Male risk profiles, which show better model fit with CTVB than SIMD, in this study, appear to be more influenced by compositional, individual behavioural risk than on neighbourhood or area, contextual factors. This finding is consistent with the recently published 2011 Scottish Health Survey[290].

These gender differences have potentially interesting implications for the design of primary prevention interventions. Research to date has not heavily distinguished between gender differences in contextual and compositional influences on risk.

Implementation of the QOF has received mixed reviews within the literature as to its impacts on CHD secondary prevention and equity of service delivery. The very existence of exception reporting acknowledges some individuals with CHD are potentially harder to engage in secondary prevention care and treatment than others. A criticism of the QOF is thus that it does not recognise this in terms of remuneration.

A fairer system would be to allocate additional remuneration for those sub-groups of CHD populations which are less likely to be optimally managed or cared for. This revision would mean that the greater effort on the part of the GP to engage with such sub-groups is rewarded with greater QOF payment. This decision may be unpopular with GPs serving affluent areas, for whom this could potentially mean less payment than peers serving more deprived areas. However equity of care as a principle must surely be of a higher priority for the GMS than the individual financial gain of GPs. It is questionable whether health care should ever be incentivised[253]. However the massive increases in risk factor monitoring and secondary prevention therapies prescribing evidenced in this (and other studies[257;261;263;264]) after the introduction of the QOF are striking- the QOF has improved levels of care overall.

The findings of this study are important in that they accurately characterise sections of the CHD population within the sample to the household level that are being underserved by current QOF arrangements. By adopting the methods outlined in the present study demonstrates that *actual* (versus paid) QOF risk factor monitoring and prescribing targets were not met and some inequalities in care and treatment persist in lower values council tax bands.

The methods adopted in the present study may have seen individuals exempted for reasons of frailty recorded and counted as a non-attendance; the difference in coding being inconsequential to QOF payment. Frailty increases with age, thus perhaps explaining the apparent age inequalities. This theory was supported by NHS IT support staff working with QOF data within the present study, but has not been explored within the literature. On further consideration this does not detract from the findings; if the patient is too frail to attend the practice then under duty of care the GP must perform the review by some other means.

The socioeconomic inequalities evidenced in risk-factor monitoring and statin prescribing within the present study is striking and is an important finding. In pragmatic terms there is no room for

complacency; individuals of lower SEP and higher risk of a recurrent cardiovascular event are being sub-optimally managed. Greater socio-demographic scrutiny must be placed on exception reporting. In the longer-term, based on the findings of this study (and others[247]) the current arrangements of the QOF are likely to see Tudor-Hart's inverse care law persist.

6.4 The utility of CTVB as a surrogate marker of SEP

Irrespective of whether CTVB can be considered as a satisfactory surrogate marker of SEP it has many appealing characteristics which validate its consideration. Beale et al[133-136] argue that CTVB is official, is 'instituted and maintained' by the Government for its own discreet purpose- it can therefore be considered as independent of any health debate and therefore a truly objective measure.

Beale further argues that CTVB is also 'universal, comprehensive and stable'- council tax data is readily available online and is complete, without fail for every UK property; all property values (including new and extended properties) are generated as of 1991 values ensuring consistency and reliability of values across all of the country.

As council tax data is available to the household level it could be argued that it is free of the 'ecological fallacy'[133;135] which has been described as inherent in aggregated geographically defined data which can never be truly representative of all individuals residing there in. CTVB theoretically is not prone therefore to the underestimation of deprivation influence as is often argued is the case for ecological measures. CTVB can also be obtained and accessed without intrusion to the study participants which has great advantage, saving time and money, and increasing data quality, especially when working on large scale projects. Council tax bands are also incredibly easy to work with in epidemiological studies; a simple categorical variable, easily summarized and aggregated and trouble-free for use in statistical analysis.

Whilst CTVB appears to have many strengths as a marker of SEP, data linkage within this study proved problematic for approximately 18% in both the primary and secondary prevention populations. One of the main reasons for this was because in the region of 40% of Paisley properties are flats and thus up to 12 individual properties may share the same postcode, but vary in council tax band. For example a block of flats built in Paisley's West-end vary in accommodation size from one bedroom flats (band A) to three bedroom flats (band D), thus the full address was utilized in the data linkage which was entirely dependant on how, or how well address data were recorded in health records (there are several ways to record 'Flat 2/1', i.e. '2/1', '2-1', 'Apt 2/1', 'Flat 2, 1st floor' etc). Algorithms developed by the Health Information and Technology Development team performing extracts for this study enabled the majority of

the records to be linked electronically, even when the full address did not merge initially. This scaled down the unmerged data to around 11% which still took approximately 40 hours to match records manually.

It also became apparent that CTVB actually has significant shortcomings as a measure of SEP in terms of its 'universality' and 'completeness' as described by Beale et al[133-136]; a fundamental issue was encountered during data linkage. Measures of SEP are assessed on their ability to measure material resource and circumstance; thus impacting on one's ability to purchase health promoting commodities and avoid exposure to risk factors. Using council tax bands as a marker of SEP fails to recognise the inherent difficult of accurately assigning a suitable CTVB to individuals who are renting properties. By way of a real-life example from the present study; a tenement town-house in Paisley town centre was assigned council tax band H (based purely on the value of this substantial property) when on further investigating it transpires that the property is in fact a five bedroom bedsit with five tenants residing there. Thus all five tenants were allocated council tax band H- which is highly unlikely to be representative of their material resource and circumstance. Even in individual properties which are rented it is unlikely that the CTVB will provide an accurate measure of SEP for tenants, perhaps tending to overestimate SEP, compared to its utility as a marker of SEP for home owners. The potential for anomalous allocation of CTVB in the rental housing market represents a serious shortcoming of CTVB as a measure of SEP. This finding was briefly mentioned by Fone et al[137] but was not discussed. Furthermore this finding has not been highlighted in the rest of the studies outlined in the literature exploring the potential of CTVB as a marker of SEP[133-136; 138].

The predictive validity of CTVB is further diminished when considering the price increase of property in recent years. Whilst CTVB attempts to recalculate housing value to 1991 levels (ensuring equity of CTVB classification over time) it does not take cognisance of the proportion of household income which is outgoing on mortgage within the same council tax band. Due to exorbitant price increases in the housing market since the late 1990s it is likely that the CTVB of a home purchased in 1995 represents significantly less proportionate mortgage outgoings for the same home purchased in 2010. Thus the amount of disposable income under the same CTVB might be hugely varied, thereby leading to inaccurate or incomparable allocation of SEP using CTVB.

An appealing characteristic of CTVB is that it is available at a household level and thus is close to an individual level measure of SEP. The independent contribution of CTVB to absolute risk, some risk factors, risk factor monitoring rates and statin prescribing rates suggests some merit in seeking to move from aggregated forms of SEP. When contextualising the findings from this study in the current literature

it could be argued that the debate concerning area-based/individual measures of SEP has reached theoretical saturation. From the papers reviewed[112-132] it would appear that either compositional or contextual influences on health are supported to the detraction of the other. Furthermore the term 'ecological fallacy' appears over-used and misused in the literature whenever compositional influences on health come to the fore in a given study.

To this end perhaps the challenge to epidemiological research is to acknowledge that both compositional and contextual influences on health and disease co-exist; thus, instead the challenge is to better understand or conceptualise the causal mechanisms or pathways which underpin and reinforce these influences on health behaviours and outcomes, particularly amongst individuals of lower SEP where inequalities there in exist. This greater understanding and insight into why and how health damaging behaviours are adopted would enable public health in general and prevention strategies in particular to develop more effective, nuanced approaches to prevention intervention or programme design.

The findings of this study suggest that the potential for CTVB to be immediately used as a surrogate marker of SEP in health research is limited. The most applicable finding from this study is perhaps the independent contribution CTVB has in predicting current smoking rates and smoking status monitoring. CTVB may add value to existing measures of SEP in the design and coordination of population based smoking cessation campaigns or services especially amongst asymptomatic populations where smoking status is not known across the complete population.

6.5 Strengths of the present study

The present study is completely novel and the findings are potentially useful and interesting to a wide range of health professionals, from health improvement officers to policy makers, planners and epidemiologists. The scope of the study is extremely ambitious; synthesising many strands of, at times, diverse evidence and literature in a coherent, structured way.

The analysis covers both primary and secondary prevention populations and the findings highlight persistent and important inequalities which are of burning relevance to the development and delivery of CHD prevention services and interventions in Scotland and beyond. The findings and discussion sections highlight important issues in relation to the use of CTVB as a surrogate marker of SEP. Current evidence in this field has barely touched on the issues highlighted in the present study.

6.6 Limitations of the present study

It should be recognized that the SIMD contains a health domain which in part may be confounding in terms of its co-linearity with cardiovascular risk variables. Ideally the health domain should have been removed from the SIMD prior to the analysis; however this was not possible from the original data extract. Similarly the multi-co linearity in the primary prevention regression model (SIMD and CVTB are reasonably strongly correlated) is potentially a limitation within the analyses.

The validity of the Framingham score as well as total and HDL cholesterol levels recorded in the primary prevention population is compromised by the cholesterol measure being taken in a non-fasting state. Whilst some studies have shown that lipid profiles change only slightly during fasting versus non-fasting measurement, it is recognized that the most accurate tests are performed under fasting conditions.

Both the primary and secondary prevention populations' sampling framework was based entirely on patients registered with a Paisley GP. As such an indeterminate proportion of patients fitting the selection criteria, but not registered with a GP will have been excluded from this study.

6.7 Recommendations from the study

Findings from this study are important and have relevant implications for current approaches to prevention of CHD in the UK. Findings from the study support:

- That in terms of CHD prevention policy; the potential of CTVB as a surrogate marker of SEP should be noted and explored in further research.
- That CHD primary prevention resources should continue to be weighted towards areas or individuals of lower SEP where exposure to classical risk factors are higher over the life-course.
- That future research should recognise that both compositional and contextual influences on health co-exist; for CHD prevention arguably the priority is not to establish which has the greater influence but to investigate and conceptualise the causal mechanisms of both influences which underpin and reinforce cardio-damaging behaviours particularly amongst individuals of lower SEP

- That the potential of exception reporting to 'mask' socioeconomic and other inequalities in CHD secondary prevention care and treatment must be recognised and investigated on a larger scale and in more detail by the GMS.
- Further QOF investigation and research should adopt the methods outlined in the present study where exception reporting is removed leaving actual care and treatment delivery as opposed to care and treatment payments.

6.8 Conclusions

Based on the findings of the present study CTVB has limited scope as a surrogate marker of SEP in the primary and secondary prevention of CHD in the UK. Some findings of the study are noteworthy however the reliability of CTVB as a marker of SEP must be investigated further. Contrary to CTVB literature to date, this study encountered difficulties in the linkage of CTVB to health records and identified a major concern in relation to CTVB misclassifying the SEP of individuals who are not home owners; potentially overestimating individual SEP of those renting, especially within homes of multiple occupancy. The consistency and accuracy of CTVB as a measure of SEP is also questionable given the increase in housing price in recent years.

The present study demonstrates that socioeconomic disparities and inequalities exist within risk profiles of the asymptomatic population (primary prevention) and the care and treatment of the established CHD population (secondary prevention). The former reinforcing approaches which target the reduction of classical risk factors in high risk populations, the latter having strong implications for the delivery of secondary prevention under the GMS QOF within Primary Care in Scotland.

Appendix A Ethics Committee Study Approval

Primary Care Division

Research Ethics
South Glasgow & Clyde REC
R&D Directorate
1st Floor – The Tennent Institute
Western Infirmary
38 Church Street
Glasgow G11 6NT
www.nrscotland.gov.uk

NHS Greater Glasgow and Clyde

Dr Kate MacIntyre
Senior Clinical Lecturer in Chronic Disease Epidemiology/Honorary Consultant in Public Health Medicine
University of Glasgow
Section of Public Health & Health Policy
1 Lilybank Gardens
Glasgow G12 8RZ

Date 26 November 2008
Your Ref
Our Ref
Direct line 0141 211 2123
Fax 0141 211 2811
E-mail Liz.Jamieson@ggc.scot.nhs.uk

Dear Dr MacIntyre

Full title of study: **Identifying unmet need in Coronary Heart Disease care in Paisley, Renfrewshire using 'Have Heart Paisley's' Chronic Disease Register- NHS Greater Glasgow & Clyde funded research & PhD study.**

REC reference number: **08/S0710/87**

The Research Ethics Committee reviewed the above application at the meeting held on 25 November 2008.

Ethical opinion

Members of the Committee present gave a favourable ethical opinion of the above research on the basis described in the application form, protocol and supporting documentation, subject to the conditions specified below.

1) Please clarify what is meant by 'Unmet Needs'. Will the research identify 'Unmet Needs' and are they likely to be addressed?

2) Please formally confirm that the original consent form included consent for future research to be carried out on the database. The original consent form enclosed was difficult to read.

Ethical review of research sites

The favourable opinion applies to the research sites listed on the attached form.

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission at NHS sites ("R&D approval") should be obtained from the relevant care organisation(s) in accordance with NHS research governance arrangements. Guidance on applying for NHS permission is available in the Integrated Research Application System or at http://www.rdforum.nhs.uk.

Approved documents

The documents reviewed and approved at the meeting were:

Document	Version	Date
Protocol	-	
Investigator CV	-	
Application	IRAS 1.1	11 November 2008
Study Protocol Diagram	-	
Dr P D MacIntyre's CV	-	
Mr J D Lewsey's CV	-	
Mr C Hankins' CV	-	
Covering Letter	-	
Letter from Renfrewshire CHP confirming funding for the project	-	17 January 2008
Participant Consent Form: Participant Info and Consent combined	1	01 November 2005
GP/Consultant Information Sheets	1	
Compensation Arrangements	-	

Membership of the Committee

The members of the Ethics Committee who were present at the meeting are listed on the attached sheet.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Now that you have completed the application process please visit the National Research Ethics Website > After Review

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

The attached document "After ethical review – guidance for researchers" gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

We would also like to inform you that we consult regularly with stakeholders to improve our service. If you would like to join our Reference Group please email referencegroup@nres.npsa.nhs.uk

08/S0710/87	**Please quote this number on all correspondence**

With the Committee's best wishes for the success of this project

Yours sincerely

Liz Jamieson

Liz Jamieson
Interim Committee Co-ordinator
On behalf of Mr Gerald F Belton OBE, Chair

Enclosures:	List of names and professions of members who were present at the meeting "After ethical review – guidance for researchers" [Site approval form (SF1)
Copy to:	Mr Brian Rae, R&D

Appendix B Caldicott Guardian Study Approval

Health Information & Technology

Dalian House
PO Box 15329
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GLASGOW
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Tel. 0141 201 4444
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www.nhsggc.org.uk

NHS
Greater Glasgow and Clyde

Dr Kate MacIntyre
Senior Clinical Lecturer in Chronic Disease Epidemiology
Department of Public Health & Health Policy
Division of Community-Based Sciences
University of Glasgow
1 Lilybank Gardens
GLASGOW
G12 8RZ

Date 3 December 2008
Your Ref
Our Ref RWC/AMMcC

Enquiries to Richard Copland
Direct Line 0141 201 4994
E-mail richard.copland@ggc.scot.nhs.uk

Dear Dr MacIntyre

PAISLEY CHRONIC DISEASE REGISTER

Thank you for your recent e-mails seeking Caldicott Guardian approval for recreation of and access to the Paisley Chronic Disease Register.

As I mentioned in my e-mail to you, I am sorry that I did not respond to you earlier. I have now received comments from Dr Linda de Caestecker and Dr Brian Cowan, who assist me in my role as Caldicott Guardian and I am happy to grant approval for you to proceed.

Yours sincerely

RICHARD W COPLAND
Director of Health Information & Technology
and Caldicott Guardian

Appendix C: GP consent letter

Greater Glasgow and Clyde NHS Board

Development Department

Westward House

13 St James Street

Paisley

Dear Practice

IMPLEMENTATION OF CORONARY HEART DISEASE UNMET NEED PROJECT

As part of Phase 2 of Have a Heart Paisley (HaHP) it was agreed that data relating to Coronary Heart Disease (CHD) would be routinely extracted from your practice to populate HaHP's Chronic Disease Register (CDR). Building on the work of Have a Heart, Greater Glasgow and Clyde NHS Board in collaboration with the department of Public Health and Health Policy at the University of Glasgow are conducting research into unmet need in CHD within Paisley.

The purpose of the study is to explore and describe the determinants of unmet need in Coronary Heart Disease (CHD) risk, care and provision of services within Paisley. This study has been approved by the South Greater Glasgow & Clyde Research Ethics Committee and the NHS Greater Glasgow & Clyde Caldicott Guardian. The study is funded by NHS Greater Glasgow & Clyde.

As part of this research the CDR is being upgraded and updated. The data items required for the CDR are the same as those already routinely extracted from your practice for Keep Well and Local Enhanced

Services (LES). All patient data analysed at the University of Glasgow will be at the population level and the data will be made anonymous and non-identifiable.

We would be grateful if you could please complete the attached form if you agree that data from your practice can be used in this study.

If you have any questions in relation to the unmet need research please contact either Dr Iain Findlay, Consultant Physician and Cardiologist at the Royal Alexandra Hospital, Paisley, email: iain.findlay@rah.scot.nhs.uk or Dr Kate MacIntyre, Senior Clinical Lecturer in Chronic Disease Epidemiology/Honorary Consultant in Public Health Medicine, at the University of Glasgow, Section of Public Health and Health Policy, email: k.macintyre@clinmed.gla.ac.uk

Unmet Need Study

I have read the accompanying letter and wish to approve the use of Keep Well and LES data extracted from my practice for the stated purpose of the Unmet Need research study.

Practice Code:

Practice Address:

Telephone No:

Signature: ……

NAME (IN BLOCK) ……

Position ……. Date ……..

---

1. ***Planned Date of Installation: TBC. No user intervention is required.***

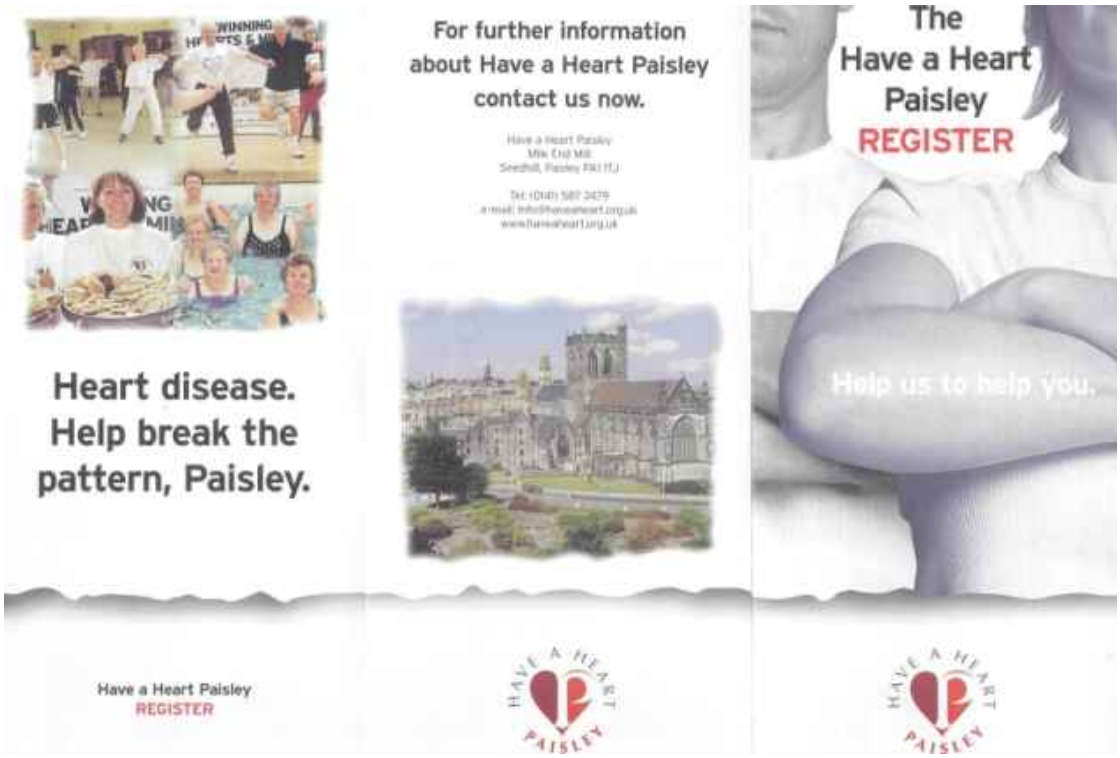
2. ***We will telephone the practice following the install to confirm that the server installation has been successful.***

   Who would you prefer us to ask for: ……

Please fax back to: **Development Dept.** **FAX #:**

Westward House **0141 843 2762**

Appendix D: Have a Heart Paisley CDR awareness raising and 'Opt out' form



Have a Heart Paisley
Have a Heart Paisley aims to reduce heart disease and promote healthier, longer lives for the people of Paisley.

Awarded £5million by the Scottish Executive, the project brings together the community, health care professionals and local organisations to work for a lasting improvement in the health of everyone in the town. In order to achieve this, it is vital that we know the extent of heart disease in Paisley, what affects it, and why. Have a Heart Paisley is therefore proposing a unique register that will not only help direct activities now, but point the way forward for Paisley and the rest of Scotland.

Have a Heart Paisley Register
The aim of the register is to help doctors ensure that people affected by heart disease, and those with a significant risk of developing it, receive appropriate, up-to-date care. To do this we need to create a register of all people who have a GP in the Paisley area.

Health-related information will be added to this register to identify those with heart disease or who are at risk of developing it. This information will come from existing records held by GPs, community nurses, hospitals, the Health Board and hospital laboratories. Details from cardiac screening and questionnaires will also be added to the register, which will be held on a computer **within the Primary Care Trust.** Information about the type of data held on the register will be available in GP surgeries. An example of the kind of information collected on the register can be viewed on the Have a Heart Paisley website.

www.haveaheart.org.uk
How will the register benefit patients?
The register will help to make sure that you receive the best up-to-date treatment. If you already have heart disease or if you become affected by it, your GP can make sure that you are taking all the right medicines. Up-to-date treatments will reduce your chances of developing further heart problems. You can have a check up to see if these are helping. For example, you can have your blood pressure and cholesterol level measured.

In addition, new treatments are being developed all the time to benefit people with heart disease. Your GP will be able to discuss these with you so that these benefits are made available to you as soon as possible.
The register will help to reduce your risk of having further heart problems. It will make a difference to your health.

Who will have access to the register?
Your GP and consultant will have full access. Other healthcare professionals will have access **only** to information required to administer your healthcare. Medical researchers who are gathering information to help in the fight against heart disease will be able to request use of the register, but will not have access to details that could identify you.

The Health Service is legally required to comply with the Data Protection Act (1998).

Can I Opt Out?
We hope you see how valuable this information will be in improving patient care in Paisley. However, if you do not wish your details to be held on the register, please complete the Opt Out form attached and return it to the Freepost address. Opting out will have no effect on any other healthcare you receive.

OPT OUT FORM

I do **not** wish my details to be recorded in the Have a Heart Paisley Register.

NAME

ADDRESS

POSTCODE

SIGNATURE

OPT OUT

Please return to the following Freepost address:
Royal Alexandra Hospital, Project Room Cardiology Dept, FREEPOST SCD6673, Paisley PA2 9BR

If you have any questions regarding participation in the Have a Heart Paisley Register, please don't hesitate to contact us at the number below.

0141 580 4919

If you are happy to take part, you need do nothing.

Have a Heart Paisley
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