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**Ethnicity and cardiovascular disease prevention**

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**MBBS MPH MFPH**

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the Degree of Doctor of Philosophy**

**Institute of Health and Wellbeing  
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University of Glasgow**

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## **Related publications**

Baker J, Mitchell R, Pell J. Cross-sectional study of ethnic differences in the utility of area deprivation measures to target socioeconomically deprived individuals. *Social Science and Medicine* 2013;85:27-31.

Baker J, Mitchell R, Lawson K, Pell J. Ethnic differences in the cost-effectiveness of targeted and mass screening for high cardiovascular risk in the UK: cross-sectional study. *Heart* 2013;99(23):1766-71.

## **Abstract**

### **Background**

Public health interventions need to both improve health and reduce health inequalities, whilst using limited health care resources efficiently. Well-established ethnic differences in cardiovascular disease (CVD) raise the possibility that CVD prevention policies may not work equally well across ethnic groups. The aim of this thesis was to explore whether there are ethnic differences in the potential impact of two CVD prevention policy choices - the choice between mass and targeted screening for high cardiovascular risk, including the use of area deprivation measures to target screening, and the choice between population and high-risk approaches.

### **Methods**

Cross-sectional data from the Health Survey for England 2003 and 2004 were used. Three sets of analyses were carried out - first, calculation of ethnic differences in the utility of area deprivation measures to identify individual socioeconomic deprivation; second, investigation of ethnic differences in the cost-effectiveness of mass and targeted screening for high cardiovascular risk; third, analysis of ethnic differences in the potential impact of population and high-risk approaches to CVD prevention.

### **Results**

Area deprivation measures worked relatively effectively and efficiently at identifying individual socioeconomic deprivation in ethnic minority groups compared to the white group. In ethnic groups at high risk of CVD, cardiovascular risk screening programmes were a relatively cost-effective option, screening programmes targeted at deprived areas were particularly cost-effective, and population approaches were found to be an effective and equitable way of preventing CVD despite potential underestimation of their impact.

### **Discussion**

This thesis found that ethnic minority groups in the UK are unlikely to be systematically disadvantaged by a range of CVD prevention policies that have been proposed, or implemented, for the general population. Additional CVD prevention policies, in particular those based on the population approach, should be implemented.

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**Author's declaration**

I declare that the contents of this thesis are my own work and where the work of others has been used it has been indicated and appropriately referenced.

Dr. Jessica Baker

## 1 Chapter 1: Introduction

Health policy makers face continual decisions regarding how best to prevent disease and promote population health. Against a background of limited health resources and increasing demand and need for health services (1, 2), decision makers must assess the relative merits of public health interventions. The first aim of any public health intervention must be to improve health. Second, public health interventions should reduce, or at least not widen, health inequalities. In addition, the financial, resource and opportunity costs of interventions must be considered, to ensure that they offer good value for money and do not take up resources that could be better used elsewhere (3).

It can be difficult to ensure that these aims can and will be achieved in practice. Making this assessment requires detailed evaluation and evidence gathering regarding the effectiveness, cost and equity impact of potential and existing interventions. Evidence needs to be gathered both for the population as a whole and for population subgroups (4). This is because it is possible that public health interventions could work less effectively or efficiently in subgroups of the population, a difference that could create or exacerbate health inequalities or waste resources (5).

This thesis aims to explore this issue from the perspective of an important disease (cardiovascular disease) and important axis of health inequality (ethnicity). Cardiovascular disease (CVD) is the leading cause of premature mortality in the United Kingdom (UK) and, crucially, may be preventable through the modification of cardiovascular risk factors (6, 7). There is a well-established, though complex, association between CVD and ethnicity, with evidence of ethnic differences in CVD and many important determinants and causes of CVD (8-11). In particular, some of the largest ethnic minority groups in the UK experience a higher risk of CVD than the majority white population (12).

A number of high-profile decisions regarding CVD prevention policy have been made in recent years. In England, decision makers opted to implement a nationwide cardiovascular risk screening programme for all middle-aged adults (13), whilst in Scotland decision makers chose to target screening at socioeconomically deprived populations (14). The approach used by the English government to encourage healthy behaviours, and therefore reduce

cardiovascular risk factors, across the population has been controversial and continues to be debated (15-17), whilst national recommendations have been made for population wide measures to prevent CVD (18).

Alongside these developments in CVD prevention policy, the UK is becoming more ethnically diverse (19). This raises the increasingly important question of whether CVD prevention policies designed for the general population work equally well in different ethnic groups. If these policies worked less effectively or efficiently in these groups existing ethnic inequalities in CVD could be worsened and resources, which could be used elsewhere, wasted. Therefore, the purpose of this thesis is to explore whether there are ethnic differences in the potential effectiveness, cost and equity impact of a range of CVD prevention policy options designed for the general population.

## **1.1 Thesis structure**

In addition to this introductory chapter (chapter 1) this thesis is made up of 6 further chapters (chapters 2-7). Chapters 2 and 3 form the literature review; the first of these review chapters provides a broad overview of the literature on CVD prevention including the various approaches available, whilst the second of these chapters reviews the association between ethnicity and CVD and the implications of ethnicity for CVD prevention approaches. Following these literature review chapters, there are three chapters which each address separate, but linked, research questions (Chapters 4, 5 and 6). These questions relate to two policy choices in CVD prevention - the choice between mass and targeted screening for high cardiovascular risk, including the use of area deprivation measures to target screening, and the choice between population and high-risk approaches to CVD prevention. The specific research questions addressed are:

Are there ethnic differences in the utility of area deprivation measures to target socioeconomically deprived individuals? (Chapter 4)

Are there ethnic differences in the cost-effectiveness of targeted and mass screening for high cardiovascular risk? (Chapter 5)

Are there ethnic differences in the potential impact of population and high-risk approaches to CVD prevention? (Chapter 6)

Chapters 4, 5 and 6 each contain a brief introduction, plus methods, results and discussion sections. Finally, Chapter 7 provides a general discussion of the findings and their implications for CVD prevention policy makers.

## **2 Chapter 2: Cardiovascular disease prevention**

### **2.1 Overview**

This chapter forms the first part of the literature review and focuses on CVD prevention. This thesis is principally concerned with primary prevention of CVD - that is prevention in people who do not yet have a diagnosis of CVD. Therefore, this chapter focuses on the potential impact of primary prevention of CVD, alongside various methods of categorising preventative interventions, including population and high-risk approaches. The evidence for commonly used primary prevention interventions is reviewed. The chapter finishes by reviewing evidence of the potential impact of public health interventions on health inequalities, another core theme in this thesis.

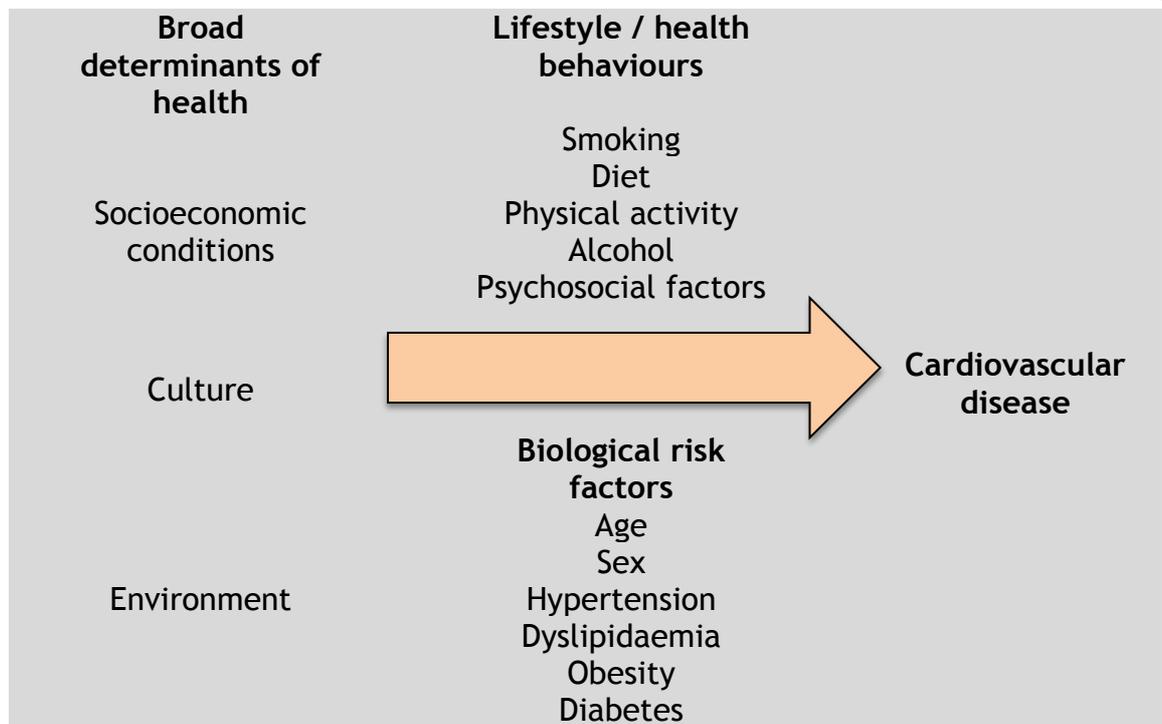
### **2.2 Definition of cardiovascular disease**

CVD is a term that encompasses a range of diseases affecting the heart and circulatory system (20). Whilst this can include diseases such as peripheral arterial disease and heart failure, this thesis is principally concerned with two of the most important diseases within this definition - ischaemic heart disease (IHD) (also known as coronary heart disease) and stroke. The main cause of CVD is atherosclerosis, a build up of fatty deposits (atheroma) in arteries (21). Atheroma in coronary arteries can result in partial blockages leading to angina or, if a clot (thrombosis) also forms, complete blockage leading to myocardial infarction (20); stroke can result from atheroma and thrombosis causing blocked arteries to the brain (ischaemic stroke), or from bleeding in the brain (haemorrhagic stroke) (22).

The Framingham study, a pivotal cohort study from North America, clearly demonstrated the importance of risk factors to the development of CVD (23), changing our understanding of CVD and how it could be prevented (24). These risk factors have subsequently been found to apply in different populations across the world, in both men and women and at all ages (7, 25). Cardiovascular risk factors can be unmodifiable or modifiable. Increased age and being male are important unmodifiable risk factors. Modifiable risk factors include health behaviours, such as smoking, and biological markers, such as high cholesterol concentrations (see Figure 2-1), risk factors that can be altered through lifestyle

changes and pharmacological interventions. Yusuf *et al* calculated a population attributable risk for first myocardial infarction of 90.4% associated with a combination of nine modifiable risk factors (smoking, fruit and vegetable consumption, physical activity, alcohol consumption, psychosocial factors, hypertension, dyslipidaemia, obesity and diabetes) (7). Population attributable risk is a statistic used in epidemiology to indicate the proportion of disease that could be prevented by eliminating exposure to risk factor(s) (26). When interpreting this value of 90.4% it is important to note that population attributable risks for multiple risk factors can exceed 100%, and that this value does not suggest that other risk factors can only account for 9.6% of disease (27). Despite this, Yusuf *et al*'s finding indicates that a very high proportion of CVD could be prevented if these risk factors were eliminated and, therefore, the enormous potential that exists for prevention of CVD.

In addition, broader determinants of health exist that influence the development of these individual risk factors and provide additional opportunities for CVD prevention (see Figure 2-1). Of particular relevance to this thesis is the well-established association between socioeconomic position and CVD, whereby lower socioeconomic position is associated with increased risk of CVD (28, 29). Socioeconomic position has been described as the social and economic factors that determine a person or group's position within society, which may in turn influence health, either positively or negatively (30); this definition indicates that socioeconomic position is a relative concept that will vary depending on the society considered. Socioeconomic position may act as an upstream determinant of health that influences the development of other cardiovascular risk factors or as an independent risk factor in itself (31, 32). The important role of socioeconomic position as a determinant of CVD and cardiovascular risk factors, and in CVD prevention, will be discussed further in this, and subsequent, chapters.



**Figure 2-1: Main determinants and causes of cardiovascular disease (derived from (33))**

### 2.3 Epidemiology of cardiovascular disease

CVD is the leading cause of death worldwide, as well as a major cause of disability (34, 35). It is the largest cause of premature mortality in the UK (6), where it is estimated that over 3 million people have CVD, with significant health service and societal costs amounting to around £30 billion per year and 21% of the NHS's overall expenditure (21, 36). The UK's record on CVD does not compare favourably with other high-income countries, with evidence that the age-standardised rate of years of life lost due to IHD is significantly higher in the UK than the mean rate in these countries (6).

The burden of CVD is changing around the world. The Global Burden of Disease project found that between 1990 and 2010 IHD and stroke moved from being the 4<sup>th</sup> and 5<sup>th</sup> leading causes of disease globally to being the 1<sup>st</sup> and 3<sup>rd</sup>, respectively (34). This change reflects the epidemiological transition, where non-communicable diseases, such as CVD, are becoming more prevalent whilst communicable diseases are declining. The burden of CVD is increasing in countries in the earlier stages of this transition (37), in contrast to the UK and other developed countries which are in the later stages of the transition and have reached a point where CVD is now declining (38-40). Compared to other Western European countries, whilst IHD mortality rates in the UK have been

high, the percentage reduction in mortality rates seen in the UK has been comparatively large (41). There is evidence that the decline in CVD is due to falling incidence, mortality and case fatality (38, 40), although there is uncertainty over the extent to which each of these contribute to the decline (38, 39, 42).

Falling rates of CVD may be partially accounted for by falls in cardiovascular risk factors, alongside better treatment (41, 43-45). A number of studies have attempted to identify the separate contributions made by treatment and risk factor changes to declines in CVD, although this is a difficult process considering recent favourable trends in both of these factors (46). The results vary but in general show that risk factor changes and treatment contributed similar proportions to the decline, with the contribution from risk factor changes ranging from approximately one-third to one-half depending on the country studied (47-52). In the UK it has been estimated that 46% of the reduction was due to risk factor changes, whilst a proportion of the decline in CVD remained unexplained (53). A number of these studies used the IMPACT model (47-50, 52), a mathematical model that uses local data on IHD mortality, risk factors and treatment. Whilst this model does not include nonfatal cases, and classifies prevention in people after a cardiovascular event (secondary prevention) as treatment, the results from different countries are largely consistent. However, it is possible that the contribution of risk factor changes may have been underestimated, as risk reductions due to risk factor changes may be more difficult to estimate accurately than those from treatment. This is because evidence of the effects of risk factor changes is more likely to come from observational studies, which are more likely to underestimate associations between exposures and risk factors than randomised controlled trials from which estimates of treatment effects are obtained. These important risk factor reductions are due to falls in blood pressure, cholesterol and smoking that have occurred across many developed countries. However, there is still room for improvement, with modelling studies suggesting that further, achievable risk factor reductions could halve the number of predicted IHD deaths in the UK and the USA (46, 54). However, evidence suggests that increases in the prevalence of obesity and diabetes may offset some of these recent gains and lead to rising levels of cardiovascular risk in younger age groups, with unknown consequences

as these groups get older (39, 41, 55-57). Therefore, it is crucial that recent declines in CVD are not taken for granted.

In addition to differences in the burden of CVD between countries, there are also differences within populations themselves. Differences in disease between populations and individuals can be described as health inequalities (58). Health inequalities can be both avoidable (e.g. due to lifestyle differences) or unavoidable (e.g. due to genetic differences) (58). Furthermore, some health inequalities may be viewed as being unfair, in which case they could be described as health inequities, a term that incorporates concepts of justice (59). However, this judgement is not necessary for the definition of health inequalities (58). This thesis uses the term health inequalities, however some of the differences described could also be considered health inequities depending on their cause and potential for reduction. Health inequalities can be measured in relative or absolute terms, complementary approaches that offer different information on the nature of or changes in inequalities (60). For instance, absolute measures provide evidence of the scale of differences in health between population groups, information that is particularly important to public health professionals (60). Socio-economic, geographic and ethnic inequalities in CVD are well established in the UK and other countries (21, 32, 61, 62). In fact, inequalities in CVD and cardiovascular risk factors are key drivers of overall socioeconomic health inequalities (61, 63). Although tackling health inequalities is a key aim of health and social policy, evidence suggests that despite falling prevalence of cardiovascular risk factors and declining mortality from CVD in the UK, relative and absolute inequalities may have widened or, at least, not improved (47, 61, 64-66). The issue of inequalities in CVD will be discussed further later in this chapter.

In summary, epidemiological evidence highlights that CVD carries a significant but changing burden, which affects population sub-groups unequally. Additionally, and crucially for this thesis, individual and population risk of CVD can be reduced through the modification of risk factors.

## **2.4 CVD prevention**

The importance of CVD prevention is widely accepted (67, 68). International guidelines highlight its potential, efficacy and the future gains that can be made (69). Effective preventative interventions can help to control escalating health-

care costs and promote the sustainability of health care services if they reduce overall resource use and demand (24, 67, 70-72). However, the argument for prevention is not entirely straightforward. As well as practical issues of implementation and evaluation (73, 74), there is a potential conflict between prevention and treatments used to cure disease or reduce symptoms in clinical medicine (75). A number of disadvantages of prevention have been described, including that it can create anxiety in otherwise healthy people, and that preventative interventions can be potentially harmful (75, 76). The opportunity costs of implementing preventative interventions must be considered, especially if they divert limited healthcare resources away from treatment. In fact, it has been argued that prevention should not be prioritised over providing basic medical care (75, 76). Despite these criticisms, it is important to note that clinical care can also be associated with disadvantages (side effects, polypharmacy, reduced quality of life, cost and so on) and it is hard to ignore the large potential health gains that prevention can bring.

Preventative interventions, like all health interventions, also need to ensure that they do not worsen health inequalities. This means that it is important to choose preventative approaches carefully and with consideration for the population involved. The next sections in the chapter will outline various approaches to CVD prevention and give examples of the types of interventions that can be used.

## **2.5 Primary prevention of CVD**

CVD can be prevented in a number of ways, depending on who the prevention is aimed at, which intervention is used and which risk factor is targeted. The four levels of prevention - primary, secondary, tertiary and primordial - provide a way of categorising preventative approaches (see Table 2-1) (77). Use of these levels is well established in public health practice although they have been criticised for a number of reasons. The levels divide preventative interventions according to whether an individual has developed the disease or not, but the progression of most diseases is unclear and cannot be divided neatly into groups (77, 78), and regardless of the categorisation used the actual interventions adopted may be similar (78). This is certainly the case for CVD, where atherosclerosis gradually progresses into disease (77), it is now possible to identify people with asymptomatic but established disease (68), and there is an

overlap in the interventions that are recommended for primary and secondary prevention (78).

**Table 2-1: Four levels of disease prevention (77, 79)**

Level of prevention	Primordial prevention	Primary prevention	Secondary prevention	Tertiary prevention
Definition	Prevention of risk factors before they occur in a population	Prevention of the onset of symptomatic disease	Prevention of recurrence or worsening of the disease after its initial occurrence	Reduction of the negative consequences of an incurable disease
Example from CVD prevention	Measures to prevent children being exposed to tobacco smoke	Use of cholesterol-lowering medication in a patient who has not developed symptomatic CVD	Prescription of lifelong aspirin in a patient who has had a heart attack	Management of heart failure in a patient with severe IHD

CVD cardiovascular disease, IHD ischaemic heart disease

Primary prevention has significant potential to reduce CVD (80). Evidence obtained from applying the IMPACT model suggests that primary prevention may account for 2 to 4 times more of the mortality reduction associated with reductions in risk factors seen in recent years than secondary prevention (52, 81, 82). In addition, Gemmell *et al* estimated that meeting government targets for cardiovascular risk factors through primary prevention could prevent more events than increasing treatment levels in secondary prevention (83). In contrast, data from the USA indicate that downward trends in age-standardised mortality and rates of hospital admission with recurrent myocardial infarction were not matched by reductions in incidence of admission, suggesting that recent declines in CVD may be due to treatment and secondary prevention rather than primary prevention (84). However, others have highlighted that it can be difficult to separate primary and secondary prevention in this way as better primary prevention may impact on mortality and recurrence indirectly through less severe presentations of disease (85).

Despite the potential effectiveness of primary prevention it is often underused, at both an individual and population level, and underfunded compared to secondary prevention (52, 72, 82). This could be because it is more challenging to implement effective primary preventative interventions. In primary

prevention it can be difficult to identify individuals who could benefit from interventions (86), and even in those who are positively identified there is evidence of poorer control of risk factors compared to secondary prevention patients (87, 88). Adherence to primary prevention medication was found to be only around 50% in a meta-analysis, a lower proportion than in secondary prevention (89). This contrasts with secondary prevention where it is easier to identify people who have the disease, the patients involved will be at high individual risk and therefore have greater potential to benefit from interventions, and there is a good range of evidence on the efficacy of preventative interventions in this group of patients (73). This is not to say that we have achieved all we can from secondary prevention, in fact uptake of secondary prevention drugs is far from complete (90). However, given the potential impact of primary prevention and evidence of its underuse it is an important area that needs further development.

## **2.6 Population and high-risk approaches**

The previous section described the four levels of disease prevention, with a focus on primary prevention. This section discusses another way of categorising approaches to prevention - population and high-risk approaches. Whilst the focus of this literature review remains on primary prevention, population and high-risk approaches can also include the other levels of disease prevention.

Geoffrey Rose compared two alternative approaches for disease prevention - a population approach in which risk across a whole population is reduced, and a high-risk approach where preventative action is focused on high-risk individuals (see Box 2-1 for examples of population and high-risk interventions) (91). This distinction was based on the premise that the causes of individual cases of disease may be different to the causes of incidence of disease at a population level, therefore requiring different interventions (92).

### Box 2-1: Examples of high-risk and population interventions in cardiovascular disease prevention

High risk	A screening programme that identifies people who are at high risk of developing CVD and offers them interventions, such as statins and lifestyle advice, to reduce their risk; smoking cessation for individuals.
Population	Legislation to reduce salt content of processed food at a national level; comprehensive tobacco control measures, including legislation, taxation and restriction.

CVD cardiovascular disease

Rose described a risk distribution that may exist for certain exposures in the population, where risk gradually increases as exposure to a normally distributed risk factor increases (see Figure 2-2 (a)) (91). Of note, he also highlighted that other exposure to risk relationships may exist that follow a different distribution. For instance, a J-shaped curve in which risk is also increased at low levels of exposure, such as that observed for alcohol and mortality (93). However, the risk distribution illustrated in Figure 2-2 (a) corresponds with many cardiovascular risk factors, such as cholesterol concentration and blood pressure. Within this distribution two key observations can be made - first, there are only a small number of people at the higher, more risky end of the distribution; and second, the majority of people lie in the middle of the distribution with a moderate risk.

Rose defined high-risk based on thresholds of single risk factors, such as cholesterol concentration or blood pressure. In the high-risk approach, individuals on the right-hand side of the distribution, above a predetermined threshold, would be targeted with risk reducing interventions (see Figure 2-2 (b)). The rest of the population would be unaffected. Rose outlined a number of advantages and disadvantages to the high-risk approach (see Table 2-2). One of these disadvantages - the difficulty of identifying high-risk individuals - is a key issue in this thesis.

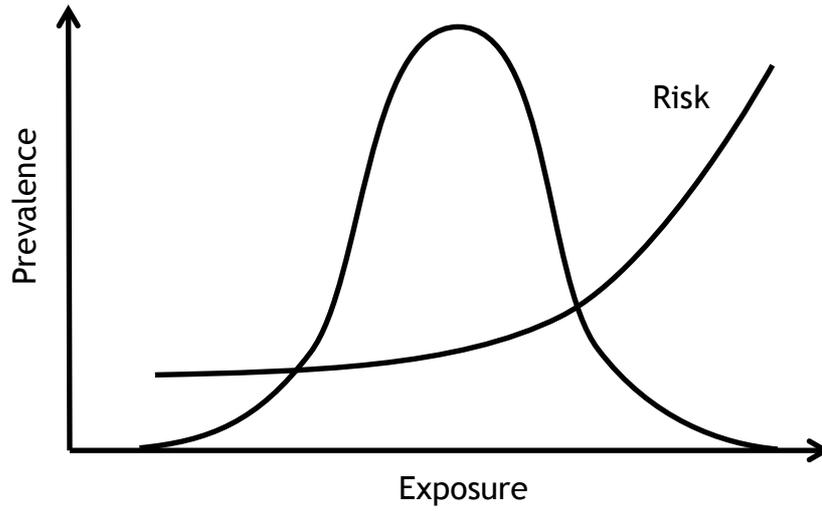
The alternative to this approach is to prevent disease at a population level by shifting the whole risk distribution to the left, i.e. to a lower overall level of risk for the whole population (see Figure 2-2 (c)) (91). This type of approach would be suitable for widespread diseases in which the risk is distributed throughout the population. Looking again at the risk distribution, it can be seen that most people lie in the middle of the distribution (see Figure 2-2). Rose described that

even though these individuals are not at high-risk most cases of disease will arise from this group. This group of people would not be identified, and therefore not benefit, from a high risk approach, so a more widespread intervention may be more appropriate. As with the high-risk approach, this approach has both advantages and disadvantages (see Table 2-2). The disadvantages include a key concept in disease prevention - the prevention paradox. This is the idea that while a population approach to disease prevention may offer a large benefit for the population, the benefit experienced by each individual may be small because most people are not at high risk of developing the disease.

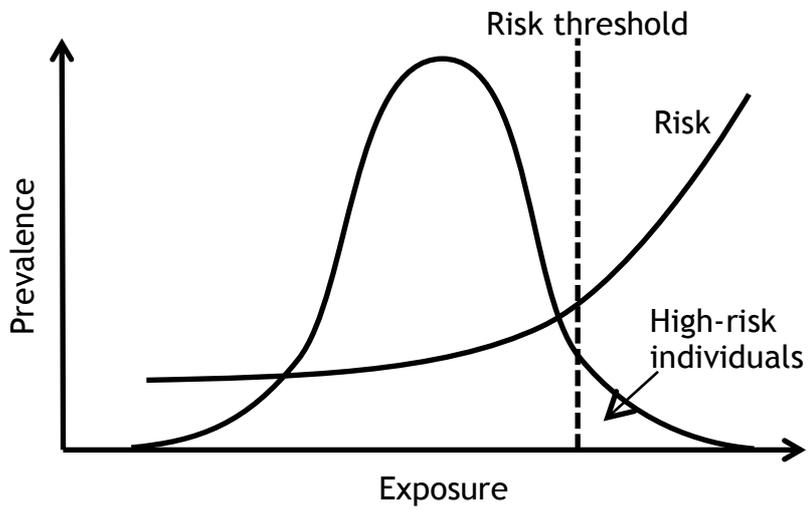
**Table 2-2: Strengths and weaknesses of population and high-risk approaches (based on (92) with additional points)**

	Strengths	Weaknesses
High-risk approach	<ul style="list-style-type: none"> <li>• Potentially large risk reduction for the individual</li> <li>• Intervention tailored to individual</li> <li>• Low risk individuals unaffected</li> <li>• Potentially cost-effective as target resources at high-risk individuals</li> <li>• Preferable benefit to risk ratio</li> <li>• Potential for greater motivation from patient and clinician</li> <li>• Easier to evaluate efficacy in clinical trials</li> </ul>	<ul style="list-style-type: none"> <li>• Medicalisation of otherwise healthy individuals</li> <li>• Risk reducing effect may not be sustainable in long-term</li> <li>• Difficulties identifying high-risk individuals and predicting their future risk</li> <li>• May only lead to small reductions in disease burden</li> </ul>
Population approach	<ul style="list-style-type: none"> <li>• Potential to effect change in underlying causes of disease, e.g. socioeconomic deprivation</li> <li>• May lead to large reductions in disease burden</li> <li>• Long-term and sustainable approach</li> <li>• May be a more efficient use of resources</li> </ul>	<ul style="list-style-type: none"> <li>• Prevention paradox</li> <li>• Small risk reduction for the individual</li> <li>• Sometimes unacceptable at an individual level</li> <li>• Difficult to implement</li> <li>• Poor benefit to risk ratio</li> <li>• Implementation may be influenced by non-health related priorities, e.g. from industry</li> <li>• More difficult to evaluate effectiveness, as evidence may need to come from e.g. natural experiments</li> </ul>

(a) Relationship between risk and exposure



(b) High-risk approach



(c) Population approach

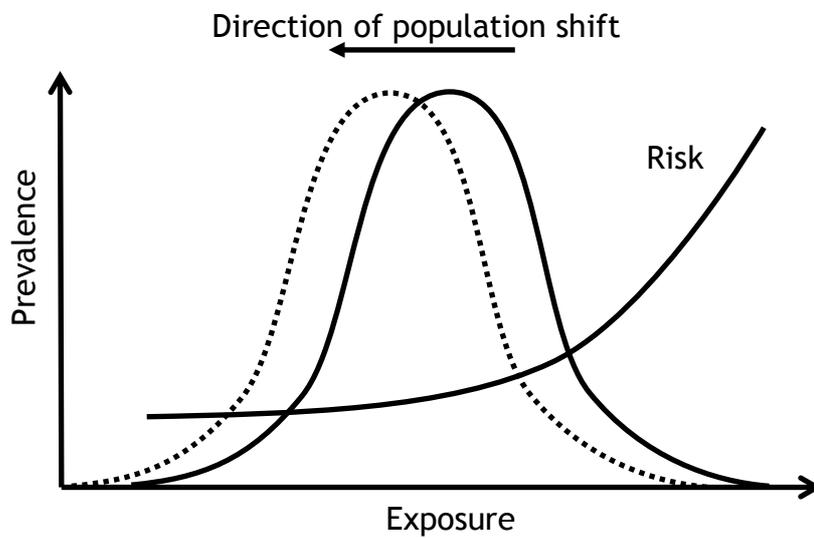


Figure 2-2: Population and high-risk approaches (based on (91))

Geoffrey Rose's ideas have been widely explored and debated. Rose advocated use of the population approach but Charlton criticised the lack of evidence on the relationship between risk and disease, and expressed concern over the potential for population interventions to lead to greater government control over people's lives (92, 94). Others have highlighted that developments in techniques to identify people at high risk of disease, for example cardiovascular risk calculators (see section 2.8.1), and the availability of safer and more effective interventions to reduce risk at an individual level may mean that the high-risk approach is a more favourable option than when Rose wrote his original work (95-97). However, the large potential gains that can be achieved from a population approach, evidence of its favourable cost effectiveness compared to high-risk options (98), and the long-term sustainability of this type of intervention, mean that it is an approach that is widely supported (97, 99, 100).

In addition to the theoretical debate on the relative merits of population and high-risk approaches, a variety of studies exist which quantify their impact on CVD. Many of these support the potential impact of population approaches (101, 102). Of particular interest is evidence from real-life populations, where significant changes in cardiovascular risk factors have occurred, associated with reductions in disease. The North Karelia project in Finland, in which a community wide programme of CVD prevention was introduced in response to high levels of CVD, led to a downward shift in the cholesterol levels of the population, in a similar way to that predicted by Rose (103). A comparable finding has also been made in Mauritius (104). Whilst evidence also exists of the potential effectiveness of high-risk approaches in real-life settings, largely from screening programmes, the scale of the changes achieved does not match those of the population approach (105, 106).

Other studies have directly compared the potential impact of population and high-risk approaches using modelling. Murray *et al* compared the costs and effects of population and high-risk approaches across a range of geographical regions (107). They found that population approaches were potentially very cost-effective; in contrast, individual interventions could prevent more disease but were less cost-effective. Cooney *et al* and Emberson *et al* both calculated the number of cardiovascular events that could be prevented using population versus high-risk approaches (108, 109). Their methods differ in terms of statistical techniques and populations studied, but both found that population

interventions could prevent more cardiovascular events than high-risk approaches. A particular strength of Emberson *et al*'s study is that they corrected for regression dilution bias, an inaccuracy in physical measurements that can occur when they are only taken once, not allowing for in person variability (108-110). In contrast to these findings, Manuel *et al* and Zulman *et al* found that high-risk approaches might be a more effective and efficient way to prevent CVD than population approaches (111, 112). The discrepancy in these studies' findings could be explained by methodological differences, including the populations studied, assumptions made about potential risk reductions and the age ranges included. For example, Manuel *et al* modelled an arguably conservative 2% reduction in cholesterol for the population approach (113), compared to 1-20% reductions by Cooney *et al*. However, Manuel *et al* argued that the high-risk approach may be more effective because, unlike previously thought, cardiovascular risk is not widely distributed in the population but is instead concentrated in certain individuals, who can now be more easily identified (111). This change in understanding may have arisen because recently developed cardiovascular risk calculators, which incorporate multiple risk factors (see section 2.8.1), allow more accurate prediction of risk, in contrast to Rose's consideration of single risk factors (114). Given this mix of evidence on the relative benefits of population and high-risk approaches, alongside the advantages and disadvantages of both approaches, it is not surprising that the general consensus is that a combination of both is needed. In the future, however, the boundary between population and high-risk approaches may become increasingly blurred through widespread use of individual level interventions, for example through use of fixed dose combination drugs ("Polypills") or personalised Smart health technology.

## **2.7 Primary prevention interventions**

A number of interventions can be used in the primary prevention of CVD (see Table 2-3). These include pharmacological and lifestyle interventions, acting at a population or individual level. Evidence in support of the effectiveness of pharmacological interventions appears more robust, although this may be because interventions of this type are easier to investigate using randomised controlled trials, whereas evaluating the effectiveness of population interventions, for example to improve diet, is more difficult.

Table 2-3: Summary of interventions for primary prevention of CVD

Intervention	Evidence of effectiveness	Nationally recommended in UK?	Comments	Population or high-risk
Statins	Two meta-analyses found significant reductions in CVD, but another found no benefit on all-cause mortality (115-117).	Yes	Cheap and generally well tolerated (96). Long-term adherence poor (118, 119).	High-risk
Treatment of high blood pressure	Recent systematic review and meta-analysis found significant reductions in CVD (120).	Yes	Choice of medication depends on age, ethnicity and comorbidity (121, 122).	High-risk
Smoking cessation	Evidence suggests 15% quitters abstinent after 1 year (123). Nicotine Replacement Therapy increases chance of quitting by 50% to 70% (124).	Yes	UK has a well-developed smoking cessation service (125).	High-risk
Tobacco control measures	Systematic review evidence suggests that bans on smoking in public places can reduce exposure to second-hand smoke and improve health outcomes (126, 127). Taxation a particularly effective way of reducing smoking (128, 129).	Yes	Various measures available, including taxation, sales and marketing restrictions, and bans on smoking in public places.	Population
Dietary salt reduction	Associated with reductions in blood pressure and cardiovascular events (130, 131). Evidence from UK of approximate 15% decrease in salt intake associated with salt reduction policy (132).	Yes	UK's Food Standard Agency previously ran a successful salt reduction programme, although this has been replaced by a new policy (133).	Population
Ban on trans fatty acids	Consumption associated with increased risk of CVD, with no nutritional benefit (134).	Yes	Bans in place in Denmark and New York (135).	Population

Table 2-3 continued...

Individual dietary changes	One systematic review found that dietary advice improved cardiovascular risk factors (136). Another systematic review found limited evidence of a beneficial effect (137). Individual interventions to reduce dietary salt intake may lead to small reductions in blood pressure (138). Mediterranean diet associated with 28-30% reduction in rate of cardiovascular events in a randomised controlled trial (139).	Yes	Evidence on best way of promoting healthy diets remains unclear. Size of relative risk reduction observed with a Mediterranean diet could exceed that from statins (117, 139).	Both
Promotion of physical activity	A systematic review of community wide interventions found no clear evidence that interventions were effective (140). Another systematic review found that individual interventions had moderate effects on exercise levels and fitness (141).	Yes	Limited evidence of the effectiveness of interventions for individuals.	Both
Aspirin	Associated with a significant, but small, reduction in cardiovascular events. However, this may be offset by an increase in gastrointestinal and extracranial bleeding (142).	Previously recommended, but unlicensed in UK (143, 144).	Unfavourable balance of risks and benefits illustrates Rose's discussion of the benefit to risk ratio.	High-risk
Multiple lifestyle interventions	Unhealthy behaviours have been found to cluster together (145). A systematic review of interventions did not find significant reductions in coronary heart disease mortality (146). Found to be the least cost-effective of a range of primary prevention strategies (98).	No	Limited evidence of effectiveness.	High-risk
"Polypill"	Recent Cochrane systematic review and meta-analysis found unclear evidence of effectiveness and concluded that further evidence is needed (147).	No	Proposal that all individuals >55 years should be offered a single pill, containing a statin, blood pressure lowering medication, aspirin and folic acid (148).	Both

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## 2.8 Identification of high-risk individuals

Earlier in this chapter one of the key challenges of the high-risk approach was highlighted - how to accurately identify high-risk people who will go on to develop disease. Two steps are required in this process - a screening test needs to be available which accurately distinguishes between low and high-risk individuals; and a strategy is needed to identify who should be invited for screening. The first of these steps relates to the use of cardiovascular risk calculators, the second to the screening strategy that is adopted.

### 2.8.1 Cardiovascular risk calculators

The Framingham cohort study led to the development of the Framingham equation - a multivariable cardiovascular risk calculator (149). This equation allowed information on an individual's risk factors, such as age, sex, cholesterol, blood pressure and smoking status, to be used to estimate the likelihood of them developing CVD in the future. This increased the accuracy of risk prediction compared to the use of single risk factors, such as high cholesterol or blood pressure, improving the identification of high-risk individuals and allowing interventions to be targeted appropriately (150, 151).

Use of the Framingham equation became widespread, and other cardiovascular risk equations were developed. In the UK, national guidelines recommend the use of cardiovascular risk calculators for the identification of high-risk individuals (121, 144). These guidelines set a threshold of risk - an individual with a risk score of  $\geq 20\%$  in 10 years (i.e. a 20% or greater chance of experiencing a cardiovascular event in the next 10 years) is classed as high-risk (121).

Despite this widespread use, cardiovascular risk calculators have a number of limitations. There is little evidence that the use of these risk scores actually improves clinical outcomes (152), and because age is such a powerful factor in these calculations they may perform less well in younger and older individuals (150). The accuracy of cardiovascular risk calculators is a key concern as any inaccuracy would mean that people who have potential to benefit from risk reducing interventions might be missed and others inappropriately targeted (151). When a cardiovascular risk calculator is used in a different population from the one in which it was developed it may be less accurate, a particular

issue relating to the use of the Framingham equation in the UK (150). The Framingham equation was derived from a now historical, largely white and affluent cohort in North America that had high rates of CVD (151). This limits its applicability to the UK population, which is more ethnically diverse, has greater variation in socioeconomic position and now experiences lower levels of CVD. Efforts have been made to improve the accuracy of cardiovascular risk prediction by recalibrating the original Framingham equations to different populations, adding new risk factors, and creating new UK specific calculators (153). Cardiovascular risk calculation will be discussed further in the next chapter in relation to ethnicity.

### **2.8.2 Screening strategies - mass and targeted screening**

Any CVD prevention programme seeking to identify high-risk individuals needs a strategy that specifies who will be screened. One approach is to offer screening to all members of the population (mass screening), another is to target screening at certain groups deemed to be at greatest potential risk (targeted screening). There is no consensus as to which of these two approaches is best, as illustrated by the national policy differences that exist in the UK - England has adopted a mass screening approach through the NHS Health Check programme, whereas Scotland's Keep Well programme targets screening at the most socioeconomically deprived areas.

#### **2.8.2.1 Mass screening**

Evidence on the effectiveness of mass health check strategies is mixed. In a Cochrane systematic review, Krogsboll *et al* found no evidence that general health checks improved health and concluded that programmes that systematically offered them to the general population should be avoided (154). Similarly, in a randomised controlled trial of mass screening and lifestyle interventions, Jorgensen *et al* found no significant difference in the incidence of cardiovascular events between the intervention and control groups (155). Likewise, recent evidence from a cluster-randomised trial of diabetes screening found no significant reduction in the relative risk of all-cause or cardiovascular mortality in the screened group compared to the control (156). This differs from Schuetz *et al* who modelled the cost effectiveness of health checks for CVD in Europe and found they had the potential to reduce incidence of CVD whilst being a cost-effective measure (157). Considering the strength of evidence arising

from these differing study designs - trial and systematic review evidence compared to evidence from modelling, which relies on assumptions of effectiveness - suggests that mass screening may not be an effective way to prevent CVD. However, a number of factors limit how generalisable some of this evidence may be to current high-risk CVD prevention programmes. For instance, Krogsboll *et al*'s review has been criticised for including older studies, where current drug prescribing guidelines would not have been followed, and considering general health checks as opposed to CVD checks (158). Similarly, Jorgensen *et al*'s study did not include use of preventative medications, such as statins. This means that it may not be appropriate to generalise these findings to programmes that use both pharmaceutical and lifestyle interventions.

Despite this mixed evidence, a mass screening programme involving health checks has been launched in England (159). Initial assessment suggested that this programme could prevent 9,500 heart attacks and strokes each year and would be highly cost-effective (159, 160). In the programme all 40 to 74-year-old adults without pre-existing CVD, chronic kidney disease, or diabetes, and who are not taking statins or antihypertensive medication, are invited for a health check every 5 years, where they are assessed for high cardiovascular risk and followed up as appropriate (159). Whilst the programme was developed centrally it is implemented locally (13). The programme is still in its early stages and awaits full evaluation, but evidence has started to emerge regarding its coverage (a measure of how many eligible people receive a health check), uptake (a measure of how many people who are invited for a health check subsequently attend), delivery and potential impact on CVD. Coverage of the programme has been found to vary widely, ranging in one study from 0% to 29.8% between primary care trusts (161). In a deprived area of London uptake in the first year of the programme was 44.8% (162). Early evidence suggests wide variation in how the programme is implemented across England (163, 164), an issue which has been cited as a key weakness of the programme (165). It would be particularly interesting to know whether a programme of this type can result in demonstrable reductions in cardiovascular risk. Two studies investigated this question by assessing changes in cardiovascular risk following health checks, with their findings suggesting small reductions in cardiovascular risk (166, 167). However, the studies did not have true control groups and did not account for secular reductions in risk. The Department of Health's initial impact assessment

has been criticised for providing an overly optimistic view of what could be achieved by the programme, for example they modelled uptake of 75% (159, 168). So far the evidence suggests that the programme is not meeting these levels, although further evidence is expected.

Mass screening programmes have the advantage of being able to offer assessment to all individuals in the population, without intentionally excluding any individuals or groups. However, mass screening has been criticised because it may not offer good value for money and may be an inefficient way to identify high-risk individuals (169); targeted screening of individuals who are likely to be at higher risk of CVD may be a more efficient and cost-effective alternative (168, 170).

#### **2.8.2.2 Targeted screening**

A variety of potential targeted screening approaches have been suggested. These include strategies in which an individual's cardiovascular risk is estimated prior to screening (pre-stratification) and strategies that target deprived areas.

##### **2.8.2.2.1 Pre-stratification**

Pre-stratification involves using existing individual patient information to determine who should be invited for screening, based on an assessment of whether that individual may be at high cardiovascular risk. Chamnan *et al* used prospective cohort data to model a variety of screening strategies including mass screening and pre-stratification, based on risk factors such as age, body mass index and estimated risk of diabetes (171). This study had a particular advantage of containing prospective data so providing information on actual, rather than estimated, cardiovascular events. They found that pre-stratification could prevent a similar number of events as mass screening but with fewer people needing to be screened. Similarly, with regards to diabetes screening, Harding *et al* found that pre-stratification by age, family history, physical activity and body mass index could provide an effective and efficient alternative to mass screening (172). Marshall and Rouse also found that pre-stratification could increase the efficiency of cardiovascular screening, although the treatments they modelled are now slightly dated (173).

#### 2.8.2.2.2 Targeting deprived areas

An alternative way of targeting cardiovascular screening is to use an area-based approach focusing on deprived areas. Before describing evidence of this approach in CVD prevention, it is worth considering what area deprivation is, how it can be measured and why it might be useful for this purpose.

Socioeconomic deprivation refers to a state in which individuals or groups do not have the resources necessary to achieve a normal standard of living, relative to the society they are living in (174). Socioeconomic deprivation overlaps with measures of poverty or low socioeconomic position, and can occur at an individual and area level. Area deprivation offers a potentially useful way of targeting CVD prevention interventions because socioeconomic deprivation is a risk factor for CVD and deprived areas have higher rates of CVD than less deprived areas (61, 64).

Area deprivation can be measured using indices that capture information on socioeconomic deprivation gathered at small area levels. A variety of these indices have been created, including the English Index of Multiple deprivation (IMD) and its Scottish counterpart (SIMD), Townsend scores and the Carstairs index. Each index differs in terms of how it was developed and the information it is based on. These indices incorporate multiple aggregate indicators, on which data are gathered and then combined, rather than relying on a single measure of deprivation such as income (60). Take for example the IMD (175). This contains seven domains (income, employment, health and disability, education skills and training, housing and service barriers, living environment and crime) with a variety of indicators in each domain. Data in each of these domains is combined into a single score for each super output area in England (small areas with approximately 1,500 residents). These areas are then ranked so that areas can be compared in terms of their relative deprivation. This can then be used for targeting by, for example, selecting the most deprived 20% of areas. A particular advantage of area deprivation measures is that they can be used as practical and accessible proxies for individual socioeconomic position (176, 177), on which data may not be available or may be too resource intensive to collect.

Macintyre *et al* described three mechanisms by which area may be related to health - through its composition (the individuals who live in an area), its context (the environment itself) and its collective characteristics (the social and cultural

nature of the community) (178). These categories can be used to highlight the potential advantages of using area deprivation measures to target public health interventions. First, given the association between socioeconomic deprivation and CVD, deprived areas may have higher rates of CVD because there are relatively high numbers of socioeconomically deprived people living there, who are at increased risk of CVD. This concentration of socioeconomically deprived individuals occurs because the UK population is partially segregated by socioeconomic position, with distinct deprived and affluent areas (179). Indeed, individual socioeconomic position can influence a person's area of residence (177), for example because low income can restrict housing choices (180). Area deprivation measures could therefore be used to identify areas with high concentrations of individuals known to be at increased risk of CVD (181), allowing limited healthcare resources to be targeted at these individuals (182). Second, targeting interventions at deprived areas could allow modifiable area characteristics that have a detrimental effect on health to be improved (177, 182, 183). Studies have identified independent effects of individual and area level deprivation on health (and more specifically CVD), with poorer health in deprived areas over and above the individual socioeconomic characteristics of the population (31, 184, 185). For instance, Davey Smith *et al* analysed cohort data from Scotland and found that area deprivation and individual social class were independently associated with cardiovascular risk factors and all cause mortality (31). It has therefore been suggested that physical and social environmental characteristics, such as access to healthy food in shops or opportunities to exercise in good quality parks, may influence health, with these positive characteristics being less prevalent in deprived areas (177, 183). In addition, if unhealthy behaviours, such as smoking, are more common in deprived communities this could influence individual health behaviour and potentially worsen health (178).

Neither composition, context, nor collective characteristics completely explain the association between area and health (186), and these mechanisms for the association between area and health are likely to be inter-related (177, 183). Indeed, if an individual's socioeconomic circumstances influence their choice of where to live then area characteristics may lie on the causal pathway between individual socioeconomic position and health (and CVD) (177, 186). A further complication arises from the potentially long temporal association between area

and health that could occur if the effect of area or socioeconomic deprivation occurs intergenerationally, as suggested by Barker (178, 187); a long frame could also impact on the ability to influence this association in the short-term. Despite this complexity, from the practical perspective of designing public health interventions, this evidence suggests that area deprivation can be used as a means of identifying and targeting socioeconomically deprived individuals who are at increased risk of CVD, as well as providing an opportunity to create interventions that improve health related area characteristics.

There are a number of disadvantages in using area deprivation measures to target public health interventions. Choosing which areas to target, based on which measure of area deprivation, is a difficult and politically contentious issue (181). Evidence of the effectiveness of area-based programmes is limited (188, 189). For instance, evaluation of Health Action Zones, an area based programme involving the development of multi-agency working, found that the programme only had a small impact on health, although it has been suggested that the short time frame and complexity of the programme may have influenced this finding (188). Additionally, and crucially, most deprived people do not live in deprived areas and would be missed by an area deprivation based intervention (181). This potential for misclassification relates to the “ecological fallacy”. Macintyre *et al* described this as the issue of using area level information to make conclusions at an individual level, although individual or area level analysis could produce different results (178). Regarding the use of area deprivation measures to target socioeconomic deprivation, this could mean that information collated on a group of individuals may not accurately reflect the characteristics of all the individuals in that group. Tunstall and Lupton tested the accuracy of the IMD 2000 to correctly target deprived individuals (182). They found that area based initiatives had the potential to identify the majority of deprived individuals (defined by unemployment benefit receipt) but were not efficient at doing so, i.e. a large proportion of people living in target areas were not deprived by their definition. In addition, the role of area deprivation measures as a proxy for individual socioeconomic position has been tested by calculating agreement between these measures. Both Demissie *et al* and Hanley *et al* found low agreement and correlation between area and individual socioeconomic measures (190, 191), although the measures used in these Canadian studies may not be comparable to area deprivation measures used in the UK. This potential for

inaccuracy and misclassification is an important issue in the performance of area deprivation measures for targeting public health interventions, and will be explored further in Chapter 4.

Area deprivation measures have been used to target cardiovascular risk screening in Scotland's Keep Well programme, although the scope of the programme has since increased to include other population groups and targeting approaches (14). In Keep Well, general practices in some of the most deprived areas in Scotland offer health checks to their patients. Evaluation has found that individuals involved in the programme are generally supportive of this area deprivation based approach and that it may be an effective way of targeting people at high risk of CVD (14). However a lack of data has limited evaluation of the programme's effectiveness (14).

The use of area-based targeting for cardiovascular screening has also been assessed through modelling. Lawson *et al* used cross-sectional data from Scotland to model the cost effectiveness of targeted screening strategies based on area deprivation and family history compared to mass screening (192). They found that targeting the 20% most deprived areas would involve screening 15% of the population but identifying 25% of high-risk individuals. It has been highlighted that mass screening has been found to be cost-effective in comparison to no screening, but not when compared with targeted screening (193). Lawson *et al* used an incremental analysis to compare the cost effectiveness of screening strategies with each other rather than with no screening, and found that targeted screening was more cost-effective than mass screening (192). The implementation of Keep Well, alongside Lawson *et al*'s findings, demonstrate that targeting CVD prevention programmes at deprived areas as a means of identifying high-risk individuals is feasible and a potentially cost-effective alternative to mass screening. This is an important theme in this thesis and will be considered further in Chapter 5.

### **2.8.3 Section summary**

Mass and targeted screening have both been used in the UK and provide two alternative ways of identifying high-risk individuals. Each of these approaches has its own merits, such as the equal provision that comes from mass screening or increased efficiency from a targeted approach. The use of area deprivation

measures for targeting is an interesting, though complex, option that will be discussed further in the next chapter in relation to ethnicity.

## **2.9 Public health interventions and health inequalities**

Previous sections provided an overview of CVD prevention, including the types of preventative interventions and approaches that are available. A key issue that has not yet been discussed is the potential impact of these interventions on health inequalities. As highlighted in the introductory chapter, public health interventions share the dual goals of improving health whilst also tackling health inequalities. These aims are central to health policy and public health interventions, including Keep Well and the NHS Health Check programme (14, 159, 194-196).

Improving inequalities in CVD is particularly important because, whilst rates of CVD have declined in recent years, there is evidence that inequalities have increased. For instance, Asaria *et al* analysed routine, area level data from England and found that absolute inequalities decreased between the most to least deprived areas from the 1980s and 2000s but relative inequalities increased (61). Bajekal *et al* made a similar finding using a modelling approach, despite evidence that the uptake of treatment was equitable across socio-economic groups (47).

Although most public health interventions have these dual goals of improving health and reducing health inequalities, there can be a conflict between them (197). An intervention that makes positive health gains may increase inequalities in health, perhaps inadvertently. White *et al* suggested a range of potential sources of inequalities in public health interventions, ranging from low survey response rates impacting on assessment of need, to variations in the uptake of interventions and subsequent compliance with them (4). Similarly, Tugwell proposed that a “staircase effect” could occur whereby combined disadvantage from, for example, reduced access, lower screening rates, poorer diagnosis and lower adherence, in already disadvantaged populations could lead to greater health inequalities (198). The “inverse equity hypothesis” provides another way of considering how preventative interventions could lead to inequalities, by highlighting that new interventions tend to reach more socioeconomically affluent people first, thereby initially widening inequalities, before they narrow as lower socioeconomic groups catch up with the intervention (199). All of these

frameworks are relevant to CVD prevention where it is fairly easy to imagine the inverse equity hypothesis applying after the launch of the NHS Health Check programme with the “worried well” benefitting first.

There is an ongoing debate regarding the impact population and high-risk approaches may have on health inequalities. It has been suggested that population approaches may be less likely to increase health inequalities than high-risk approaches (5). Indeed, Kivimaki *et al* demonstrated, using modelling, that it is possible to greatly reduce absolute and relative socioeconomic inequalities in CVD if substantial cardiovascular risk factor reductions can be made equally across the population (200). However, population approaches may increase inequalities if some people benefit more from the shift in exposure than others. For instance, Frohlich and Potvin suggested that population interventions could increase inequalities if there are subgroups of the population who are both at higher baseline risk than the general population and less able to benefit from the intervention (201). Further, differences in the prevalence of the risk factor being addressed by a population intervention between population groups could influence health inequalities. For instance, inequalities in unhealthy behaviours, such as diet and salt intake, could mean that a population intervention that altered these risk factors could produce differential effects across the population (202, 203).

Evidence also suggests that the choice of population intervention used may influence its effect on health inequalities. Lorenc *et al* systematically reviewed the evidence for the impact of various population interventions on health inequalities and found evidence that mass media campaigns may increase inequalities whereas interventions based on price may reduce them (204). Similarly, Thomas *et al* found that fiscal measures may be a particularly effective way of reducing inequalities in smoking between social groups (205). This fits with Macintyre’s conclusion that structural, upstream interventions may be more beneficial for health inequalities than downstream ones (206).

High-risk approaches, which tend to rely on individual behaviour and change, may exacerbate health inequalities more than population approaches (207). This could be because some population groups are less able to access and benefit from the types of preventative services and advice that are typically offered in high-risk interventions, such as health checks and lifestyle advice (206). Indeed,

there is evidence that the uptake of health checks varies according to demographic characteristics and is lower in people with greater clinical need (208). There is also evidence of inequalities in the use of preventative interventions, such as statins and cholesterol screening, in Europe and the UK (209-211), and in the effectiveness of interventions in population subgroups, including by socioeconomic position and ethnicity (for ethnicity see sections 3.7.7.2 and 3.7.7.3) (212). However, the use of incentive based approaches in UK general practice may have reduced these types of inequalities, and there is evidence that smoking cessation services have the potential to reduce inequalities in smoking (213, 214).

There are two notable themes that emerge from the literature in this final section. First, it is crucial that the impact of public health interventions on inequalities is considered, and not just socioeconomic inequalities but other axes as well (4, 5, 207, 215). The type of intervention used, and the way it is implemented, will be key to whether it increases or decreases inequalities. Second, despite the importance of this issue there is a lack of evidence on the impact of interventions on health inequalities (205, 206, 216). Inequalities in CVD can arise from differences in socioeconomic position, lifestyle, geography, age and ethnicity (217). Whilst most of the research reviewed in this section considered socioeconomic inequalities, this thesis focuses on ethnic health inequalities.

## **2.10 Chapter summary**

This chapter outlined a number of key issues relevant to CVD prevention, including the various approaches available, and the types of interventions that could be used. Prevention has significant potential to reduce the burden of CVD and therefore improve health overall. However, it is important that the preventative approach used does not worsen health inequalities and instead reduces them. This thesis seeks to explore whether there are ethnic differences in the potential impact of a number of the CVD prevention interventions described in this chapter. Therefore, the next chapter builds on the topics reviewed in this chapter by focusing on ethnicity.

### **3 Chapter 3: Ethnicity and cardiovascular disease**

The purpose of this chapter is to provide a review of the association between ethnicity and CVD, and to outline how ethnicity may relate to the various aspects of CVD prevention discussed in the previous chapter. The chapter starts by discussing the definition of ethnicity and moves on to describe ethnic inequalities in health and CVD. A number of potential explanations for these inequalities are then reviewed, including the role of socioeconomic position, CVD prevention policies and interventions, and area.

#### **3.1 Ethnicity as an epidemiological variable**

Ethnicity is a widely used, but complex, epidemiological variable with no single, straightforward definition (218). It is generally considered to be a complex, multifaceted, context-dependent social construct (219-222), that represents a variety of characteristics, including ancestry, religion, culture, language, socio-economic position, biology, geography and race (218, 219, 221, 223, 224). These characteristics can either be shared, providing a common identity, or used as markers of distinction from other groups of the population (225). Karlsen suggested that ethnicity involves both internal and external forces, arising from the individual and from their context in society, which act to distinguish one individual or group from another (225). Ford and Harawa describe a similar view, highlighting the importance of the relationship between the individual or group and their society in determining ethnicity (223).

Despite its complexity, ethnicity is considered to be an important epidemiological variable because it can identify differences in disease and disease risk factors between populations, help improve understanding of the causes of disease, and potentially improve provision of healthcare and preventative services (222, 224). Indeed, Senior and Bhopal highlighted that good epidemiological variables should be accurate measures that can be used for these purposes (222). However, the use of ethnicity as an epidemiological variable has been criticised because it has a number of limitations, which could result in the misclassification of individuals and populations (223). First, it is difficult to measure accurately (222). A number of methods have previously been used to categorise populations in ways comparable to using ethnic group,

including race, nationality or country of birth. Race refers to a longstanding method of classifying people based largely on physical appearance or geographical origins (226). It is an arguably unsound construct with significant negative connotations (226), and it is important to differentiate it from ethnicity (222). Race was thought to be linked to biological and genetic differences, often based on skin colour, but has subsequently been shown to have little scientific basis (226). Race is still used as an epidemiological variable in some settings, especially the USA (227), where it is used to provide information on socioeconomic position and discrimination (223). Interestingly, routine data in the USA divides the population into both racial and ethnic groups, with ethnicity based on Hispanic or non-Hispanic origin (228), highlighting the different uses and interpretations of these terms that still exist (see Figure 3-3). Nationality or country of birth can be useful in certain settings, for example in studies of migration or as adjuncts to measurement of ethnicity, but are limited in the aspects of ethnicity that they can measure. For example, knowing that a person was born in India does not provide potentially significant information on their religion, language or cultural background; a broader measure of ethnic group may be better able to incorporate this information. Second, there is significant heterogeneity within ethnic groups, although research often makes the assumption of similarity between people in the same group (222). One particular example is South Asian populations, which are often grouped together and considered as one ethnic group for research purposes, but which represent populations that can be vastly different in terms of culture, language, religion and geographical origins (222). Third, because it is widely accepted that ethnicity should be self-reported, it is possible for a person to change their ethnic group during their life, affecting the consistency of the measure (219). This could be seen as a limitation of self-reported ethnicity, but the practical implications of this are unclear given that ethnicity by definition represents an individual's own perception of their identity. However, longitudinal data suggests fairly good consistency in self-reported ethnicity, although this may vary between ethnic groups (229).

Collection of data on ethnicity has increased in recent decades in the UK because of legislative requirements and the introduction of a question on ethnicity to the Census (221). This Census question, which was first added in 1991, measures self-reported ethnicity using tick boxes and free text responses

(230). The question has been developed and expanded upon in 2001 and 2011, with the addition of further categories, including mixed ethnicity options (see Box 3-1) (230). Aspinall has highlighted that this nationwide measure of ethnicity has helped improve our knowledge of the relationship between ethnicity and health (231). However, he has also questioned the continuing use of skin colour in the definitions used, and the limited range of white and Asian categories available (230). This approach of measuring ethnicity appears to have been accepted as reasonable and pragmatic (perhaps in the absence of alternative options) and has been used in other surveys and research, despite the fact that it was developed for administrative rather than research purposes (221). In addition, qualitative evidence suggests that individuals from ethnic minority groups consider recording of ethnicity to be important and acceptable in healthcare, as long as it is done for clearly explained reasons (232).

**Box 3-1: Ethnic categories in the 2001 Census of England and Wales (233)**

- |   |   |
|---|---|
| <ul style="list-style-type: none"> <li>• White               <ul style="list-style-type: none"> <li>▫ White British</li> <li>▫ Irish</li> <li>▫ Other white background</li> </ul> </li> <li>• Mixed               <ul style="list-style-type: none"> <li>▫ White and black Caribbean</li> <li>▫ White and black African</li> <li>▫ White and Asian</li> <li>▫ Other mixed background</li> </ul> </li> </ul> | <ul style="list-style-type: none"> <li>• Asian or Asian British               <ul style="list-style-type: none"> <li>▫ Indian</li> <li>▫ Pakistani</li> <li>▫ Bangladeshi</li> <li>▫ Other Asian background</li> </ul> </li> <li>• Black or black British               <ul style="list-style-type: none"> <li>▫ African</li> <li>▫ Caribbean</li> <li>▫ Other black background</li> </ul> </li> <li>• Chinese or other ethnic group               <ul style="list-style-type: none"> <li>▫ Chinese</li> <li>▫ Other</li> </ul> </li> </ul> |
|---|---|

Note: Categories from the 2001 Census are shown because they are most relevant to this thesis. The 2011 Census included similar categories with the addition of tick boxes for Gypsy or Irish Traveller and Arab.

Senior and Bhopal described a number of ways in which the use of ethnicity as an epidemiological variable could be improved, including acknowledgement of its complexity and limitations, clear statement of how it is defined in the research, and acknowledgement that its changeable nature may limit generalisability across time and populations (222). In addition to these suggestions, ways of improving the broader issue of research into ethnicity and

health have also been considered. In an exercise that involved gaining consensus from a group of researchers in this field, it was agreed that it was important to include ethnicity in research on health inequalities, that researchers should seek to reduce disadvantage and discrimination experienced by ethnic minority groups, that it was important to be transparent about how ethnicity is defined and to recognise the diversity that exists within groups, that ways of categorising ethnicity need to be meaningful, and that it is important to acknowledge social context (234).

### **3.2 Ethnic groups in the UK**

The UK is becoming increasingly ethnically diverse (19). The white British population is still very much in the majority in the UK, but the proportional size of this group has decreased (from 87.5% in 2001 to 80.5% in 2011) (19). Currently, the largest ethnic minority groups are other white, Indian and Pakistani (see Figure 3-1).

The size and composition of the ethnic minority population varies geographically, with the highest ethnic diversity in London (see Figure 3-2) (19). For instance, whilst the overall size of the Indian population is 2.5%, this ranges from 0.0% to 28.3% by local authority (19).

Two studies that calculated population projections by ethnicity in the UK found that the relative size of the white population is likely to fall in subsequent decades, alongside significant increases in the size of ethnic minority groups (235, 236). One of these studies projected that the white British population will decline to 56% in 2056 with increases in all ethnic minority groups (236). This ongoing trend of increasing ethnic diversity means that measurement of ethnicity and research into ethnicity and health has become, and will become, increasingly important (231).

The ethnic make-up of the UK contrasts with that of other countries, where different historical contexts and migration patterns have resulted in the formation of different ethnic minority groups. A number of studies in this review are based on populations in the USA and Holland so these countries have been selected to illustrate the differences in the ethnic make-up of populations in other countries (see Figure 3-3 and Figure 3-4).

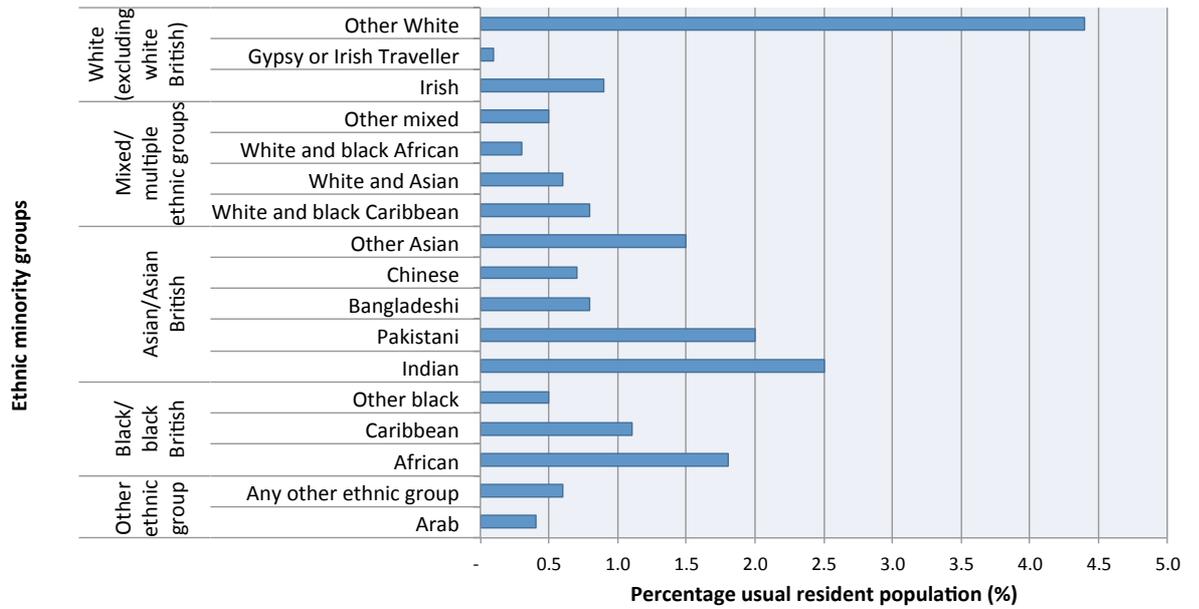


Figure 3-1: Proportion of population in ethnic minority groups in England and Wales, 2011 (19)

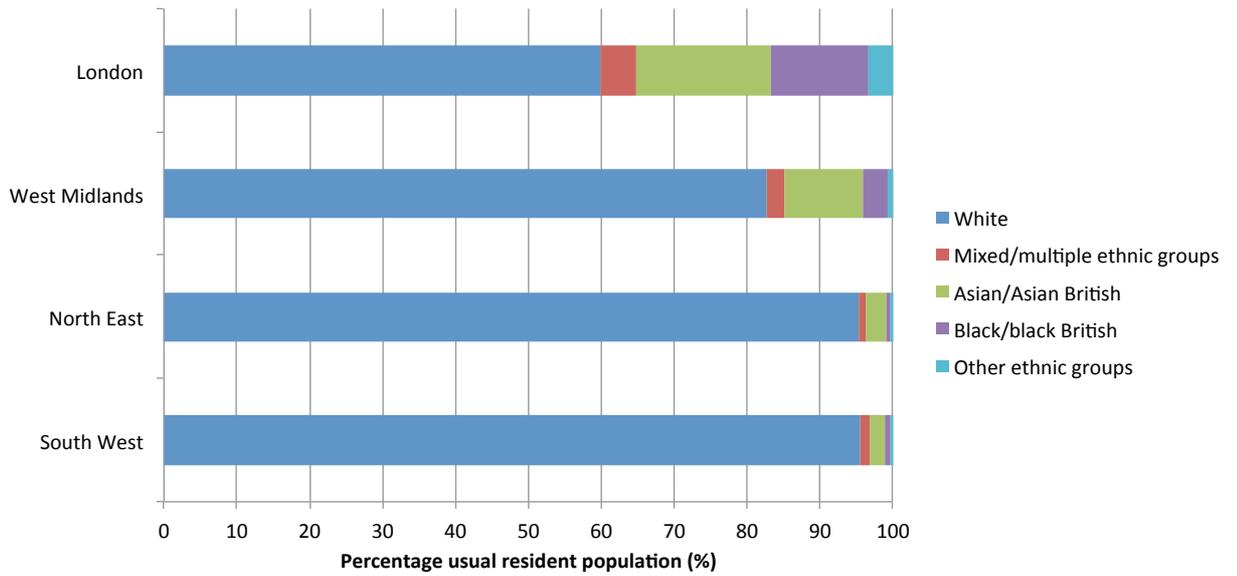
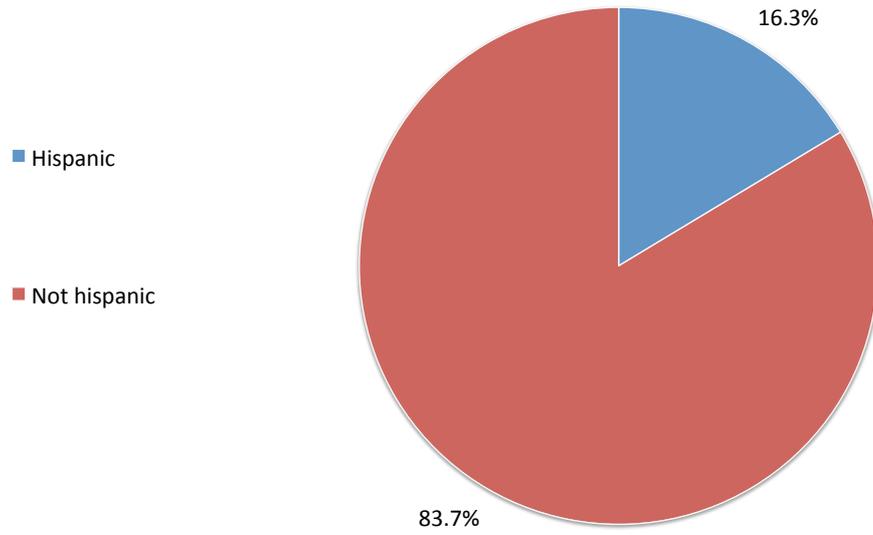


Figure 3-2: Ethnic groups in selected areas of high and low ethnic diversity in England, 2011 (19)

(a) Ethnic groups



(b) Racial groups

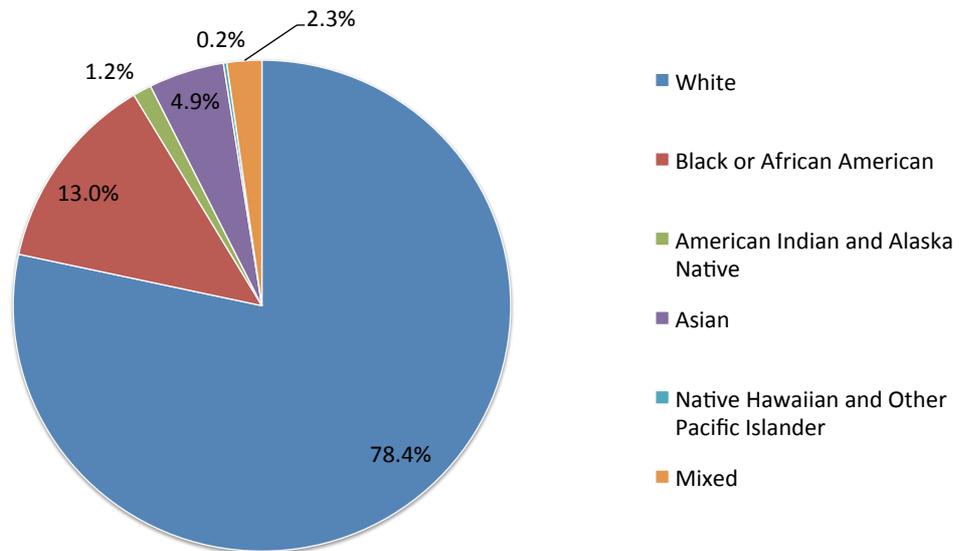
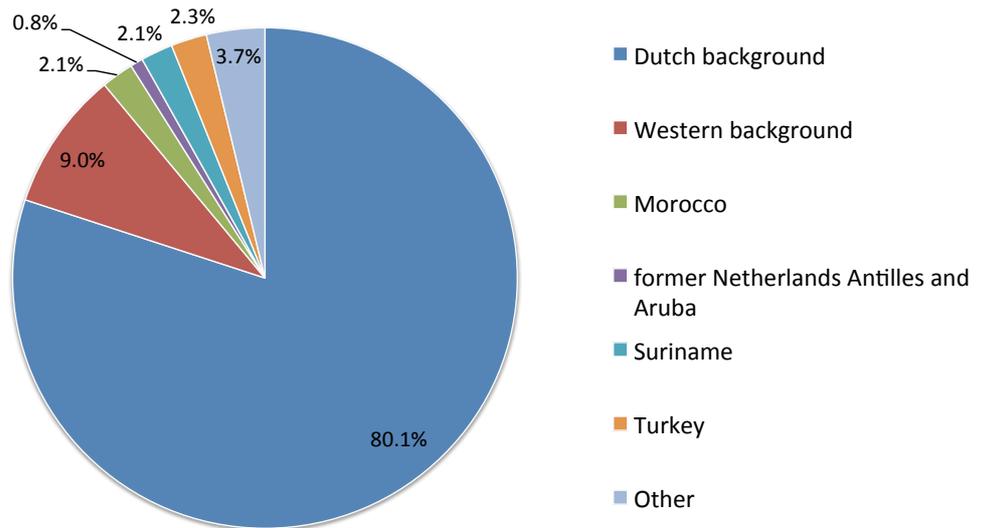


Figure 3-3: Racial and ethnic groups in the USA (228)



**Figure 3-4: Ethnic groups by origin in Holland (237)**

Note: Dutch routine statistics are based on country of birth

### 3.3 Ethnic inequalities in health

Examining the association between ethnicity and health reveals a range of ethnic inequalities. There is evidence of ethnic inequalities in mortality and morbidity from a variety of conditions, including CVD (238). However, these inequalities vary by ethnic group and context.

Early studies from the UK used country of birth to explore health inequalities, likely driven by data availability. Wild and McKeigue analysed routine death and census data from 1970-92 and found inequalities in mortality by country of birth (239). They found that all-cause mortality was higher in all immigrant groups than in the general population, except in immigrants from the Caribbean. Marmot *et al* and Harding and Maxwell also studied mortality according to country of birth and identified significant inequalities (240, 241). Whilst these studies identified and drew attention to important inequalities between immigrant groups, country of birth does not fully measure ethnicity according to the definition we understand today. However, subsequent research that used a broader measure of ethnicity has also identified inequalities in health. Using self-reported ethnicity data from the 1991 census, Harding and Balarajan found that the relative risk of limiting long-term illness was significantly higher in all ethnic minority groups than in the white group, with the exception of the Chinese group (242). Becares also used census data on limiting long-term illness

and reported similar findings, with higher prevalence of illness in Irish and black Caribbean men, and Pakistani and Bangladeshi women (243). Cooper analysed cross-sectional data from the Health Survey for England and identified poorer self-reported health in black Caribbean, Indian and Pakistani women (244). In addition to evidence on general health outcomes, ethnic inequalities in infant mortality and cancer have also been observed (9, 245).

Ethnic inequalities in health are seen around the world. A range of ethnic inequalities have been documented in the USA (246). For example, in the USA black and American Indian people are generally seen to have poorer health than white people (246). In Europe, a systematic review found an association between poorer self-reported health and most ethnic minority groups; however, many of the studies reviewed were from Sweden, limiting the applicability of the findings to other countries (247). Similar to Wild and McKeigue's findings in England and Wales, ethnic inequalities in mortality have been identified in the Netherlands from classifying people by country of birth (248). In New Zealand there are well-established ethnic inequalities in health, largely related to the indigenous Maori population (249), which has a very different historical background to ethnic minority populations in Europe.

### **3.4 Ethnic inequalities in cardiovascular disease**

In addition to the health outcomes described above, there are also well-established ethnic inequalities in CVD. Evidence of these inequalities comes from a variety of ethnic groups, countries and sources. In England and Wales Wild *et al* found notable ethnic differences in mortality from both IHD and stroke (239). For instance, between 1989 and 1992 they found that the standardised mortality ratio was higher in men born in South Asia and Ireland compared to the general male population (146 and 124, respectively), whereas it was lower in men born in the Caribbean (standardised mortality ratio 46). The standardised mortality ratio for stroke was higher in all immigrant groups than the general population - in South Asian men it was 155, in Caribbean men 168 and in Irish men 138. In a similar follow-up study, using more recent data, these ethnic inequalities were again noted (250). IHD mortality was significantly higher in men born in Ireland, Bangladesh, India and Pakistan compared to the general male population, and was significantly lower in men born in China and the West Indies. A similar pattern was noted for women. Stroke mortality was significantly

higher in men born in Ireland, the West Indies, Bangladesh, India, Pakistan, China, Eastern Europe and Scotland. These two studies indicate inequalities in cardiovascular mortality, including differences in the inequalities observed in IHD and stroke. However, the measure of ethnicity used was country of birth, which the authors note, may be an unreliable measure of ethnicity in younger people who are less likely to be migrants. This potential for misclassification means that these results may not fully reflect ethnic inequalities in CVD.

Despite this limitation, Wild *et al*'s findings are consistent with evidence from a range of other sources. First, evidence suggests that the risk of developing IHD varies by ethnicity. Some ethnic groups, in particular South Asian groups, have been found to be at higher risk than others, such as the black Caribbean and Chinese groups. Forouhi *et al* used prospective data from primary care patients in London to analyse inequalities in mortality between South Asian and European people, using a broader measure of ethnicity than country of birth (12). After adjusting for age they found that IHD mortality was 60% higher in South Asian people than European people, a difference that remained after adjustment for socioeconomic position and cardiovascular risk factors. Data linkage work from Scotland has also identified inequalities in IHD incidence between South Asian ethnic groups and the majority white population (251, 252). Ethnicity data from the Census 2001 were added to routine hospital admission and mortality data. Indian and Pakistani people were found to have higher rate ratios of chest pain and angina compared to white Scottish people, and there was a significantly increased incidence rate ratio of acute myocardial infarction in South Asian compared to non-South Asian people (251, 252). This approach demonstrates that data linkage may be a potentially useful way of adding ethnicity data to routine sources that lack it, although the lack of primary care data in this analysis limits the conclusions that can be drawn about less severe presentations of IHD. In addition to a higher risk of IHD, there is evidence that Asian people develop the disease at a younger age and may present with different symptoms (253, 254). However, South Asian ethnic groups may experience a better prognosis than white individuals, with evidence of better survival after acute myocardial infarction (251, 255). In contrast, mortality rates from IHD are lower in black men and women compared to white and South Asian people in England and Wales, consistent with Wild *et al*'s findings (239, 256). There is also evidence of ethnic inequalities in IHD from other countries. In the USA, mortality

rates from IHD have been found to be higher in black people than white people, although hospital admissions for myocardial infarction were highest in white individuals (257). In Canada, higher prevalence of IHD has been identified in South Asian people compared to European and Chinese people (258).

There are also ethnic inequalities in the epidemiology of stroke, although these are not entirely consistent with those observed for IHD. For instance, the risk of stroke has been found to be higher in Caribbean and Chinese populations compared to the general population, despite the lower risk of IHD in these groups (250). A range of studies has identified ethnic inequalities in stroke incidence, prevalence and mortality. In London, using data from a stroke register, higher age-adjusted incidence of stroke has been found in black African and black Caribbean people compared to white people (259, 260). Analysis of linked data from Scotland, as described above, found that the risk ratio for hospitalisation and mortality from stroke was significantly higher in African men than white Scottish men (261), although this study did not report results for black Caribbean people. Ethnic inequalities in stroke between black and white individuals have also been identified in the USA, and demonstrated higher incidence of and mortality from stroke in black people compared to white (262). Similarly to IHD, stroke has been found to occur at an earlier age in higher risk ethnic groups, with differences in presentation and survival. For instance, black stroke patients in London were found to be significantly younger than white patients, with ethnic differences noted in the types of stroke occurring, and evidence of better survival in black compared to white patients (263, 264).

### **3.5 Ethnic differences in cardiovascular risk factors**

The vast majority of cases of CVD can be accounted for by a group of modifiable risk factors, as described in the previous chapter (see section 2.2). The Framingham study demonstrated the importance of cardiovascular risk factors to the development of CVD (23), and subsequent research indicates that these traditional risk factors are also relevant across ethnic groups and in explaining ethnic differences in CVD (265, 266). This section outlines evidence of ethnic differences in the prevalence of important cardiovascular risk factors and considers whether there may be ethnic differences in the risk associated with these risk factors.

### 3.5.1 Cholesterol

A number of studies have found ethnic differences in cholesterol concentrations and lipid profiles. It has been suggested that South Asian people may have a higher risk lipid profile than white individuals (267), whilst individuals of African descent may have a lower risk profile (268). However, a systematic review of ethnic differences in cardiovascular risk factors found that evidence of ethnic differences in cholesterol was inconsistent (10). In a cross-sectional study, Bhopal *et al* found that South Asian people living in Newcastle had a lower HDL concentration, higher triglycerides, and a higher total cholesterol:HDL ratio than European people (269). Likewise, in an analysis of UK civil servants, Whitty *et al* found that South Asian people may have a more adverse lipid profile than white people (270). In contrast, age-adjusted prevalence of raised total cholesterol concentration was found to be highest in white people in an analysis of cardiovascular risk factors in London (271). There is evidence that African Caribbean people have a more favourable lipid profile than white people, with lower total cholesterol, lower triglycerides and higher HDL observed in a prospective study from London (272). In contrast, a different type of analysis, using cross-sectional data from the Health Survey for England 1999, found that cholesterol concentrations were similar between black Caribbean people and the general population (273).

### 3.5.2 Blood pressure

Raised blood pressure is the most important modifiable risk factor for stroke (274). Black people have a relatively increased risk of stroke, and evidence suggests that black populations may have higher levels of blood pressure and hypertension than white populations. Higher blood pressure and higher prevalence of hypertension in black populations has been observed in the UK and the USA (10, 267, 270, 275). For instance, age-adjusted prevalence of hypertension was found to be highest in individuals of African origin in a sample of general practice patients from London in the 1990s, with a prevalence ratio of 2.6 compared to the white group (271). In the USA, the prevalence of hypertension and risk of developing hypertension has been found to be higher in black compared to white populations (276, 277). Risk of stroke is also relatively high in Chinese populations. Prevalence of hypertension in Chinese adults in the UK has been found to be similar to the general population (11, 256), a finding

that contrasts with evidence from North America of increased risk and prevalence of hypertension in Chinese compared to White individuals (258, 277). Studies of South Asian populations suggest that they may have similar or lower blood pressure and prevalence of hypertension than white populations, although the evidence is mixed. On the one hand, Lyratzopoulos *et al* found that South Asian people had significantly lower blood pressure than white people, in a study that excluded people with known hypertension (278). Similarly, Bhopal *et al* found that hypertension was less common in South Asian people than European people in an analysis of cross-sectional survey data from Newcastle (269). In contrast, two studies carried out in London found increased prevalence of hypertension in South Asian people compared to white and European people (271, 279), and Whitty *et al* found that whilst South Asian men had lower mean systolic blood pressure, South Asian people had higher prevalence of hypertension than white people (270). These differences could be explained by variations in the populations studied and inclusion criteria used. In addition, a number of these studies are limited by the age of the data used, for example based on cohorts from the 1980s and 1990s (270, 279), and so may not reflect recent trends in blood pressure across ethnic groups.

### 3.5.3 Smoking

Smoking is a very important cardiovascular risk factor that carries a population attributable risk of over 35% for myocardial infarction (7). Prevalence of smoking is lower in many ethnic minority groups than in the white population in the UK, although this varies by gender. Smoking was found to be less common among black Caribbean, black African and South Asian individuals compared to white individuals in two studies from London (271, 272). Analysis of the Health Survey for England indicated that the prevalence of current smoking is highest in Bangladeshi men (43.5%), Irish men (38.0%) and Irish women (31.7%) (11). Bhopal *et al* also found a higher prevalence of smoking among Bangladeshi men compared to Indian, Pakistani and white men (269). In contrast, low levels of smoking have been observed in Pakistani (4.5%) and Bangladeshi (2.4%) women (11). However, it should be noted these very low levels of smoking in Bangladeshi women may conceal a higher proportion who consume tobacco in different ways, such as chewing it (256). Another analysis of the Health Survey for England reported similar findings, with higher prevalence of current smoking

in black Caribbean and Bangladeshi men compared to white men, and lower prevalence of current smoking in Pakistani, black African and Indian men (280). In this study, very low smoking levels were observed in Indian, Pakistani, Bangladeshi, black African and Chinese women. However, these findings were found to be largely influenced by socioeconomic position with reductions in ethnic differences after adjusting for area deprivation (280). Ethnic differences in smoking have also been noted in the USA, with consistently lower levels in Mexican-American people but conflicting results in comparisons of smoking in black versus white populations (10).

#### **3.5.4 Diet**

Eating an unhealthy diet is an important risk factor for CVD; indeed, evidence from the Global Burden of Disease study suggests that the proportion of disability-adjusted life-years from IHD attributable to poor diet may exceed those attributable to tobacco smoking, alcohol or physical inactivity (281). Dietary risk factors that would lead to lower levels of CVD, according to this study, include high consumption of nuts and seeds, fruit and vegetables, whole grains and fibre, and low consumption of trans fatty acids, sodium and processed meat (281). Evidence suggests that people from ethnic minority groups in the UK may consume more fruit and vegetables than the general population (256), a potentially positive lifestyle behaviour. For example, Bhopal *et al* found that Pakistani and Indian men consumed more fruit and vegetables each day than Bangladeshi and white men (269). In contrast, however, the InterHeart case-control study found that intake of fruit and vegetables was lower in South Asian cases and controls compared to individuals from other countries (254). However, this finding was based on South Asian individuals living in South Asian countries, rather than those who have migrated to other parts of the world. In addition to ethnic differences in fruit and vegetable consumption, there may also be ethnic differences in consumption of salt and trans fatty acids, two dietary behaviours that are particularly relevant to population CVD prevention policies (see section 3.7.7.3 for further details).

#### **3.5.5 Physical activity**

Physical inactivity is another important lifestyle risk factor for CVD. Similar to diet, there is evidence of ethnic differences in physical activity levels in the UK although this evidence generally suggests lower physical activity levels in ethnic

minority groups (282). Data from two separate analyses of the Health Survey for England indicated that South Asian individuals had the lowest levels of physical activity compared to other ethnic groups (11, 282). For instance, Williams *et al* found that higher proportions of Indian, Pakistani and Bangladeshi individuals reported taking no physical activity each week compared to white individuals (31.7% among Indian, 56.7% among Pakistani and Bangladeshi, and 28.1% among white people) (282). This is consistent with evidence from other countries, where for example InterHeart found a lower prevalence of physical activity in South Asian cases and controls (254), and lower levels of physical activity have been noted in Mexican-American women and black men and women in the USA (10).

### **3.5.6 Obesity**

Prevalence of obesity has been found to vary between ethnic groups, with evidence of differences using a variety of measures including body mass index and waist to hip ratio. Prevalence of having a high waist to hip ratio was found to be higher in Indian, Pakistani and Bangladeshi men compared to white men (269). Cappuccio *et al* found the highest age-adjusted prevalence of obesity in African women and the lowest in South Asian men in London in the 1990s (271). Analysis of the Health Survey for England showed the highest prevalence of obesity in black Caribbean men and women, Pakistani women and Irish men (11). Evidence from a cohort study in London showed higher mean waist circumference in Afro-Caribbean women than in European women (272), and in a separate analysis higher waist circumference and higher waist:hip ratios in South Asian people compared to the European group (279). Whilst there is evidence that black and Mexican-American populations in the USA may have higher body mass index than white populations, other studies have found no difference between these groups (10, 283). Ethnic differences in adiposity and the related cardiovascular risk factor of metabolic syndrome are discussed further in section 3.7.6.3.

### **3.5.7 Diabetes mellitus**

This important risk factor for CVD varies notably by ethnicity (10). Indeed, increased risk of insulin resistance in South Asian and African Caribbean groups has been suggested as a potentially important cause of ethnic inequalities in CVD (62). Higher prevalence of insulin resistance and diabetes has been found in

African and South Asian ethnic groups (12, 279). Similarly, black Caribbean, Indian, Pakistani, Bangladeshi and Chinese men have been found to be at increased risk of diabetes than the general population (11). Indeed, Bhopal *et al* found that the prevalence of diabetes was five times higher in South Asian groups than in white individuals (269), and Cappuccio *et al* observed the highest prevalence of diabetes in South Asian people, followed by African and then white individuals in London (271).

### **3.5.8 The association between ethnicity and cardiovascular risk factors**

The evidence outlined above illustrates that ethnic differences in cardiovascular risk factors are not straightforward, and vary by risk factor, ethnic group and context. Ethnic differences in cardiovascular risk factors mean studies that seek to investigate ethnic inequalities in CVD often attempt to control for these factors, in order to explore the role of cardiovascular risk factors in explaining ethnic differences in CVD. For instance, Howard *et al* found that classic cardiovascular risk factors accounted for around 40% of the excess risk of stroke in black compared to white people in the USA (284). In contrast, other studies have found that adjusting for risk factors made little difference to ethnic inequalities in CVD (272), or conversely that it eliminated all observed ethnic differences (285). Despite these discrepancies, it appears that ethnic differences in cardiovascular risk factors are important in understanding ethnic inequalities in CVD, although these differences cannot be fully explained by classic risk factors (286).

Three additional considerations arise from examining the relationship between ethnicity and cardiovascular risk factors. First, it is important to consider overall risk profiles as well as prevalence of individual risk factors, i.e. whether individuals have multiple cardiovascular risk factors. A conclusion from some studies is that, for example, South Asian ethnic groups have a more adverse risk profile than white individuals (269). However, other studies have drawn a different conclusion. For instance, Lyratzopoulos *et al* concluded that South Asian people did not exhibit an adverse risk profile compared with white people (278). These differences may be due to differences in study design and inclusion criteria. For example, the latter study did not include people with hypertension and diabetes. In the USA, black people have been found to have a higher total

number of cardiovascular risk factors compared to white and Mexican-American people, a factor that is associated with a higher risk of developing CVD (287). Second, the association between cardiovascular risk factors and disease may itself vary by ethnicity. A difference of this kind has also been suggested for other epidemiological variables, such as sex and socioeconomic position (288, 289). Forouhi *et al* found that diabetes is associated with a higher risk of mortality in South Asian than in European people (12). Similarly, Bellary *et al* found that South Asian people with diabetes were more likely to develop premature CVD, with higher incidence rates of cardiovascular events at younger ages compared to white people, although there were few events in the follow-up period of this study (290). In a longitudinal cohort analysis from the USA, Howard *et al* found that the increased risk of stroke associated with increasing systolic blood pressure varied between black and white people - a 10 mmHg increase was associated with an 8% increased risk of stroke in white people and 24% in black people (291). Indeed, it has been suggested that some cardiovascular risk factors may be associated with greater risk in ethnic minority individuals (292), and that different thresholds for common risk factors, such as cholesterol and body mass index, may be needed in higher risk ethnic groups (292, 293). For instance, a recent study using data from the UK Biobank found that the risk of diabetes mellitus was higher in non-white than white individuals at lower body mass index values (294). The National Institute for Health and Care Excellence (NICE) has previously considered lowering the thresholds for defining overweight and obesity in black and Asian populations in the UK, however a lack of evidence meant that the thresholds were not changed (293).

In contrast, other evidence suggests that the relationship between risk factors and disease are similar across ethnic groups. Forouhi *et al* found that the hazard ratios for IHD mortality associated with smoking, blood pressure and cholesterol were similar in South Asian and European people, despite ethnic differences in the hazard ratio associated with diabetes (12). An international cohort study of middle-aged men found that there was little evidence of differences in the strength of association between cardiovascular risk factors and coronary mortality across countries and populations (295). Likewise, InterHeart found that the odds ratios for various cardiovascular risk factors were similar by country, including between South Asian and other countries (254), and by ethnicity (7). It therefore remains unclear whether there are ethnic differences in the

association between cardiovascular risk factors and disease. Differences may depend on context, other risk factors or confounders. Evidence from prospective cohort studies that include large samples of ethnic minority individuals would help to address this question, but this would be a significant undertaking.

Third, another interesting conclusion from the InterHeart study is that the impact of risk factors on population levels of disease, as measured by population attributable risk, may vary (7, 254). These differences would be driven, at least partially, by variations in the prevalence of risk factors and would be important from a public health perspective. For instance, Joshi *et al* found that South Asian populations had a higher population attributable risk associated with high waist to hip ratio, but lower population attributable risk associated with hypertension and stress (254). However, despite these differences Yusuf *et al* found that the nine main risk factors together still accounted for a similar proportion of the population attributable risk in each ethnic group (86% European, 90% Chinese, 92% South Asian, 92% black African) (7).

### **3.6 Trends in ethnic inequalities in health**

Ethnic inequalities in health and CVD have persisted and may be widening (239, 243, 296-298). With regards to general health, Becares found persistent ethnic differences in rates of limiting long-term illness between 1991 and 2011 in England and Wales (243). In New Zealand, Blakely *et al* analysed routine data and found that relative and absolute inequalities in mortality between Maori and non-Maori people increased between 1981 and 1999 (296). Evidence also suggests that whilst CVD is declining, the rate of decline may be different in some ethnic groups. In Birmingham, admissions for stroke declined between 1997 and 2005 but the fall was smaller in South Asian individuals (299). Overall falls in stroke incidence seen in London were not observed in black men, although the relative inequality between black and white men and women reduced (260). Similarly, in the USA mortality rates from CVD have not declined as much in black compared to white populations (257, 262).

It has been recommended that steps should be taken to tackle ethnic health inequalities at a national level in the UK whilst public organisations have a legal obligation to tackle racial discrimination and promote equality (300, 301). However, recent work on health inequalities has been criticised for its lack of attention to ethnic health inequalities (302). This is concerning given evidence

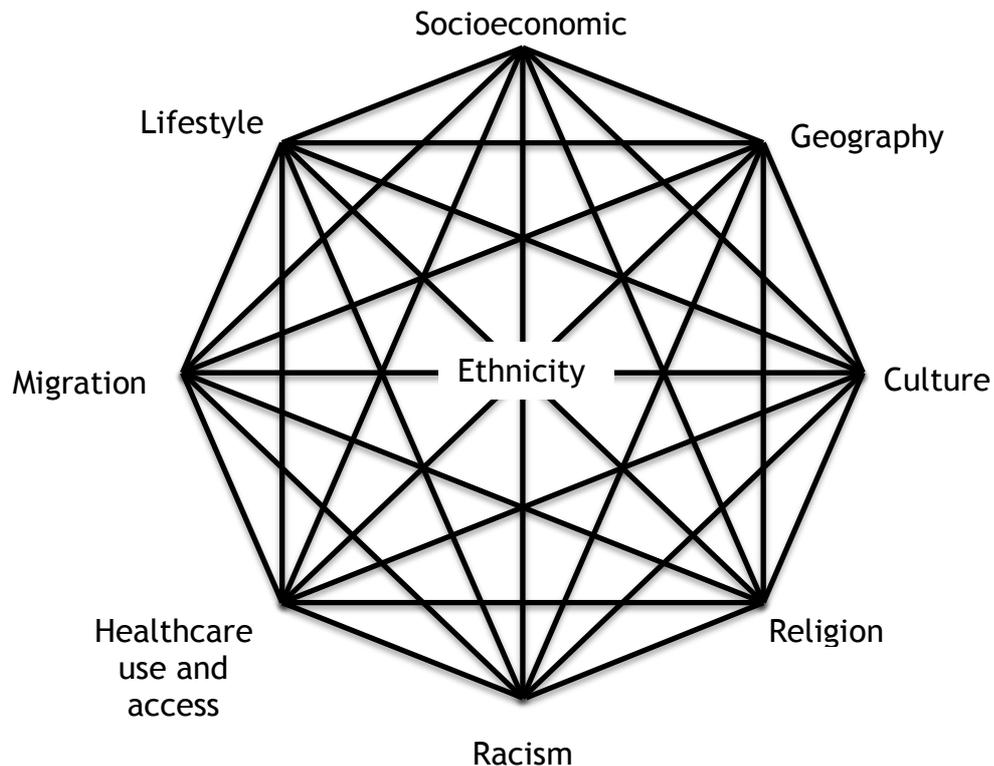
that ethnic minority groups are accounting for an increasing proportion of the population and ethnic inequalities in health may be widening.

### **3.7 Explanations for ethnic inequalities in health**

The next section of this review considers a range of explanations for ethnic inequalities in health. These explanations include artefact, socioeconomic position, migration, racism, cultural and behaviour, biology, healthcare access and effectiveness, and area effects (9, 238, 303).

Explanations for ethnic inequalities in health do not operate in isolation but are linked by complex and changing relationships (see Figure 3-5). For example, socioeconomic position may change following migration (304); racism may influence socioeconomic position, perhaps through widespread structural discrimination (305); language may influence ability to access healthcare services and education; religion and culture may affect health behaviour and attitudes towards education and employment; where a person lives may influence their employment and educational opportunities, and so on.

These complex relationships make interpreting causes of ethnic inequalities in health complicated and mean that interventions to reduce these inequalities are unlikely to be straightforward.



**Figure 3-5: Illustration of the complex inter-relationships between explanations for ethnic inequalities in health (idea based on (306))**

### 3.7.1 Artefact

Ethnic inequalities in health could be due to artefact, arising from inaccuracies or bias in the data analysed (238). For instance, Davey Smith *et al* highlight that artefactual differences in health could arise if there are ethnic differences in how individuals respond to questions on self-reported health (9). Given the complex definition of ethnicity and variety of methods used to measure it, artefact should be considered when interpreting evidence of ethnic inequalities in health.

### 3.7.2 Socioeconomic position

There is a well-established relationship between ethnicity, socioeconomic position and health. However, this complex relationship can depend on context, and has been interpreted and measured in different ways (307). There are two key reasons to suppose that socioeconomic position is important in the relationship between ethnicity and health. First, there are differences in the socioeconomic position of different ethnic groups. Second, evidence suggests that at least some of the observed ethnic differences in health can be accounted for by socioeconomic position. However, the measurement of socioeconomic

position in different ethnic groups can be problematic and may affect the results of research in this area.

In the UK, socioeconomic deprivation is more common in ethnic minority groups than in the majority white population (308). Higher proportions of the ethnic minority population live in deprived areas (308), although this varies between ethnic groups and geographical areas (309). For instance, Pakistani and Bangladeshi groups are particularly concentrated in deprived areas, although a slightly smaller proportion of the Pakistani group live in the most deprived areas in London (309). In contrast, the Chinese and Irish groups are less likely to live in the most deprived areas, and the white British group are more likely to live in less deprived areas (309). Wages have been found to be lower in Bangladeshi and Pakistani men compared to Chinese men (310). Household wealth is lower in Bangladeshi and Pakistani groups compared to Indian and white groups - £15,000 and £97,000 for Bangladeshi and Pakistani respectively, compared to £200,000 or more for Indian and white (310). There are ethnic differences in educational achievement, with greater achievement in many ethnic minority groups compared to the white majority (308). In Scotland, socio-economic deprivation is also more prevalent in ethnic minority groups, although there are some differences compared to England such as the Indian group having lower levels of poverty in Scotland (311).

As well as this evidence of increased socioeconomic disadvantage in ethnic minority groups, it is important to note the variation that exists within ethnic groups. For instance, there are large income inequalities within the Chinese group compared to low inequalities within the Bangladeshi group (312). Socioeconomic disadvantage associated with being a member of an ethnic minority group also exists in other countries, such as the USA and New Zealand. In the USA, black and Hispanic populations have been found to have poorer socioeconomic position than the white group (313, 314). In New Zealand the Maori population are socioeconomically disadvantaged compared to the non-Maori population (296).

A number of studies have found that socioeconomic position is an important explanation for ethnic inequalities in general health and all-cause mortality. Early studies in this area, examining mortality by country of birth, found no socioeconomic gradient for immigrants from the Indian subcontinent and a

possible reverse gradient in people from the Caribbean (241). However, further research has suggested that this is no longer the case. For instance, Nazroo found socioeconomic gradients in self-reported health within ethnic groups with poorer general health associated with lower socioeconomic position and has suggested that socioeconomic factors are a "fundamental cause" of ethnic inequalities in health (p.277) (315). This discrepancy between earlier and later studies could be accounted for by a cohort effect of the largely immigrant populations studied in the 1970s compared to more recent studies in which ethnic minority people are more likely to have been born in the UK and to have had different socioeconomic experiences (316, 317). In fact, longitudinal evidence from 1971 to 1981 suggests the emergence of clearer socioeconomic gradients in mortality among immigrants to the UK, with socioeconomic circumstances improving for these groups (317). In the USA, increased life expectancy is associated with higher income in both white and black groups, despite being lower overall in black people (318). In the Netherlands, absolute and relative socioeconomic inequalities in mortality were identified within ethnic groups, but varied in size and direction according to the cause of death (319).

Socioeconomic inequalities in CVD within ethnic groups have been identified, and found to be changing. Scottish data showed an association between cardiovascular risk and various measures of socioeconomic position in most ethnic groups, although there was variation in the strength of association (320). In the UK, Harding analysed deaths by country of birth and identified socioeconomic gradients in IHD mortality among South Asian immigrants, although these gradients were less consistent in Pakistani and Bangladeshi compared to Indian immigrants (321). This finding is perhaps consistent with Bhopal *et al*'s finding that the European pattern of socioeconomic gradients in CVD (higher levels of risk and disease in lower socioeconomic groups) is developing in Indian populations, and perhaps also amongst Pakistani and Bangladeshi people in the UK (8). There is also evidence of socioeconomic gradients in CVD within ethnic minority groups in other countries. For instance, in the Netherlands, Agyemang *et al* analysed national routine data and found a higher incidence of myocardial infarction in the lowest income tertile in each ethnic group studied (322). Analysis of cross-sectional survey data from the USA showed inverse socioeconomic gradients in IHD risk in most ethnic groups studied

(323). In addition, cardiovascular mortality in the USA has been found to be associated with socioeconomic position in both black and white people, with particularly high mortality in blue-collar black men (313). Changing patterns in socioeconomic gradients have also been observed in India, where individuals from urban and non-urban areas exhibit differences in the association between socioeconomic position and CVD, with an inverse relationship seen in men from urban areas in contrast to a positive relationship in non-urban areas (324).

The causes of changes in socioeconomic gradients in ethnic minority groups are likely to be complex, but could be related to changing socioeconomic position or acculturation leading to lifestyle changes. The diffusion theory suggests that the CVD epidemic affects individuals in higher socioeconomic groups first as they can afford to adopt unhealthy behaviours such as smoking and high saturated fat consumption (28). As the epidemic progresses high levels of CVD then start to affect lower socioeconomic groups as they also adopt unhealthy behaviours. Disease rates then decline in higher socioeconomic groups, as they are the first to adopt healthier behaviours, leading to an inverse socioeconomic gradient (28). This theory could explain the changing socioeconomic gradients in CVD seen in ethnic minority groups, with different groups at different stages in the process.

Studies have shown that adjusting for socioeconomic position attenuates the observed relationship between ethnicity and health (325). In the UK, Chandola analysed cross-sectional data for an association between ethnicity, health and socioeconomic position (316). Although, the socioeconomic gradient observed in Pakistani and Bangladeshi people was weaker than that in Indian and white people, ethnic differences in self-reported health became non-significant after adjusting for a range of socioeconomic factors. Similarly, Davey Smith *et al* found a reduction in the increased relative risk of all-cause mortality in black compared to white men in the USA after adjusting for an area based income measure (326). Another study from the USA, which benefited from large samples of black and white individuals, found that adjusting for income reduced the hazard ratio for cardiovascular deaths in black compared to white people (from 1.35 to 1.09) (327). These findings emphasise the importance of considering socioeconomic position when studying ethnic differences in health. However, this approach has often led to the potentially incorrect conclusion that after adjusting for socioeconomic position any remaining differences in health must be

due to factors inherent to the ethnic groups themselves, such as genetics or culture (307).

Examining the relationship between ethnicity, socioeconomic position and health is problematic for a number of reasons. First, socioeconomic measures may not be equally applicable to different ethnic groups and may not fully reflect socioeconomic disadvantage within ethnic minority groups (307). Take for instance common socioeconomic measures such as income, education, occupation and housing tenure. Income has been found to be lower in ethnic minority individuals in the same occupational class as white people in the UK (303), and similarly in the USA median income in black and Hispanic people has been found to be lower than in white people with the same educational level (318). Educational achievement may be higher in many ethnic minority groups in the UK, but this may translate into poorer long-term socioeconomic outcomes than in the majority population (307, 308). Occupational status may be adversely affected by migration and people from ethnic minority groups may be exposed to more work-related hazards and poorer quality employment (307, 308). There are ethnic differences in housing tenure, with high levels of home ownership in Indian and Pakistani groups (308). This may appear to be a positive socioeconomic circumstance but may not reflect differences in housing quality, such as overcrowding or lack of modernisation (307). This could mean that ethnic minority individuals are more likely to be misclassified socioeconomically, and the real effects of socioeconomic deprivation will not be fully accounted for. This could mean that residual ethnic differences in health after adjustment socioeconomic position could be due to socioeconomic differences that have not been measured rather than being due to ethnicity itself (307).

The choice of measure of socioeconomic position can influence the observed association between socioeconomic deprivation and health (328, 329).

Furthermore, it may influence the observed association between ethnicity and health. Kelaher *et al* found that the size of ethnic differences in health varied depending on which socioeconomic measure was adjusted for (330). Similarly, Fischbacher *et al* analysed linked routine data from Scotland, with various measures of socioeconomic position, including education, occupation, area deprivation, housing tenure and car access (320). They found that the association between socioeconomic position and incident CVD within ethnic groups varied according to which measure of socioeconomic position was used.

The association between ethnicity and CVD changed slightly after adjustment for the various measures of socioeconomic position, with the largest change seen after adjustment for education. It has therefore been suggested that multiple rather than single measures of socioeconomic position should be used (303, 320).

Second, given the complex relationship between ethnicity and socioeconomic position, controlling for the latter may lose some of the explanation of ethnic inequalities in health (303). Socioeconomic position is likely to moderate the effect of ethnicity on health, illustrated by the fact that ethnic inequalities in health vary according to socioeconomic position (326), although this would also be true for confounding factors. Brancati *et al* used cross-sectional survey data from the USA to analyse the relationship between diabetes, ethnicity and socioeconomic position (331). Although they used data from the 1970s, which is somewhat dated, they found that the association between ethnicity and diabetes differed by socioeconomic position, with a stronger association seen in lower socioeconomic groups. Again from the USA, local mortality data showed that older black men living in poor neighbourhoods had a higher rate of cardiovascular mortality than older white men also living in poor neighbourhoods, whereas cardiovascular mortality was similar in black and white individuals living in more affluent areas (332). Likewise, Huxley *et al* found that ethnic differences in stroke rates between black and white adults in the USA were smaller at higher income levels (333). Conversely, analysis of different data from the USA has shown that ethnic differences in cardiovascular mortality did not differ according to income (327). However, this range of evidence suggests that treating socioeconomic position as a confounding factor may not be appropriate. It is also possible that socioeconomic position acts as a mediating factor on the causal pathway between ethnicity and health, another reason why controlling for it may not be appropriate.

Third, standard socioeconomic measures may not reflect life course circumstances. Whilst this issue applies to use of these measures in the general population as well, it may be particularly relevant to people from ethnic minority groups who have experienced events such as migration (315). Evidence suggests that life course socioeconomic position affects CVD in both black and white individuals, although adult socioeconomic position may have a greater effect on ethnic differences in stroke risk than childhood socioeconomic position (334). In the UK, Tillin *et al* found that both child and adulthood socioeconomic

position was associated with cardiovascular mortality in South Asian men (335). Their analysis, which was based on migrants, also showed that for many men good childhood socioeconomic circumstances still led to manual occupations, suggesting that migration may be associated with negative effects on socioeconomic trajectory (335).

These three reasons mean that caution is required when interpreting evidence on the relationship between ethnicity, socioeconomic position and health. However, any research in this area must consider socioeconomic differences and the complex relationship with ethnicity.

### **3.7.3 Migration**

Migration gives people exposure to at least two different environments (the place(s) of origin and destination(s)) plus the experience of migration itself, all of which could influence health (9). Evidence suggests that the effect of migration on CVD can vary according to a person's origin and destination, and may be driven by acculturation or changes in socioeconomic position. The evidence reviewed in this section relates to migration between countries, however migration within countries, such as between rural and urban areas, may also affect health. For instance, in India there is evidence that the prevalence of stroke is higher in urban compared to rural areas (336).

Researching the association between migration and health can be challenging because of complex relationships between ethnicity, migration, CVD, and adaptation to new and different environments (337). In addition, selection bias can influence interpretation of the effect of migration on health. This is because migrant populations may be selected on the basis of health, with healthier people being more likely to migrate, destination countries imposing varying health requirements for migrants, and the possibility that unhealthy people may return to their home country (9). For example, a retrospective analysis of health insurance data in Canada indicated that the new migrants had a hazard ratio for acute stroke of 0.69 compared to long-term residents after adjusting for potential confounders, suggesting a potential healthy migrant effect (338). Furthermore, migration patterns around the world are changing (339). This means that the composition of ethnic minority populations within countries may change significantly, as existing communities become more established and new

communities arrive, creating a particular challenge for research and timely provision of appropriate health services.

CVD mortality can vary within migrant groups depending on the destination country. A European analysis of cardiovascular deaths by country of birth showed differences in mortality rates depended both on country of origin and destination (340). Gray *et al* investigated cardiovascular mortality differences by country of birth using routine data in Australia (337). They found that cardiovascular mortality decreased with increased duration of residence in Australia in some migrant groups, whereas in others mortality increased. Interestingly, they found that CVD mortality was lower than the national average in migrants from India and Sri Lanka. This contrasts with findings from England and Wales where all-cause and cardiovascular mortality of migrants from the Indian subcontinent increased with increasing duration of residence (341), although results of a similar study on Caribbean migrants did not show such mortality changes (342). It is possible that changes in socioeconomic position could explain some of these findings, however these studies report similar results before and after controlling for socioeconomic position (337, 341, 342).

Acculturation, leading to changes in health behaviours and cardiovascular risk factors, could be a mechanism by which migration influences CVD. Moran *et al* found that people born outside the USA had a lower prevalence of hypertension than people born in the USA, and that living in the USA for longer was associated with a higher prevalence of hypertension, although their sample was unrepresentative with regards to ethnicity (343). Using the Health Survey for England, with the sample divided into people born overseas or in the UK, Smith *et al* found that the risk of obesity in most ethnic minority groups converged with that of the white population (344). For instance, Chinese and Indian people born in the UK were more likely to be obese than those born overseas after controlling for demographic factors. Indeed, there is evidence that migrants to the UK may have worse cardiovascular risk factor profiles than those who have not migrated. Smeeton *et al* found that Barbadian stroke patients had a generally more favourable risk factor profile than black Caribbean stroke patients in London (345), and Patel *et al* reported a similar finding when comparing community samples of the Gujarati community in the West Midlands and India (346). These findings are consistent with evidence that mortality rates in some migrants are converging with mortality rates of people born in England

and Wales (347). Whilst acculturation could explain changes in cardiovascular risk factors in childhood and adulthood, this explanation contrasts with Barker's hypothesis that fetal undernutrition can lead to increased risk of CVD (348). If this were the case, risk of developing CVD could be at least partially determined before migration takes place, and would be increased in individuals from countries where maternal undernutrition is more prevalent.

#### **3.7.4 Racism and psychosocial experiences**

Racism, racial discrimination and harassment may have a negative impact on health. Racism could affect health through direct physical or psychological consequences, or indirectly through the creation of socioeconomic disadvantage (349). It can occur at an individual or institutional level (349), in fact it has been suggested that health services may be institutionally racist (350). Karlsen and Nazroo examined cross-sectional survey data and found a statistically significant association between poor self-reported health and experiencing racism (349). Becares *et al* also used cross-sectional data, from a more recent survey, to assess the association between racism and health and found an association with limiting long-term illness (351). In an adolescent population racism has been found to be associated with poorer psychological well-being (352). These findings are important because they suggest that racism, which is widespread (315), can have a negative impact on general health. However, a challenge for studies of this type is how to accurately measure racism and its impact on an individual (349).

More specifically, there is evidence of an association between racism and cardiovascular risk factors. Cozier *et al* asked black women in the USA about their experiences of racism and discrimination, alongside their self-reported weight and height (353). They found an association between incidence of obesity and experience of racism, which was stronger in the women who had experienced racism over a longer period of time. Although they acknowledge that their sample was not representative and they used self-reported weight and height, this suggests a potential relationship between racism and cardiovascular risk factors.

In addition, evidence suggests that there may be ethnic differences in the association between negative psychosocial experiences and CVD. For instance, Williams *et al* used a validated measure of hostility in a sample of South Asian

and white adults receiving cardiovascular screening in London (354). They found significantly higher levels of hostility in South Asian people compared to white people, with ethnic differences in the association between hostility and various cardiovascular risk factors. Negative psychosocial experiences could impact health if they lie on the causal pathway between socioeconomic position or racism and health or CVD. For instance, in a prospective cohort study of African American adults in the USA, the association between socioeconomic position and hypertension and diabetes reduced after adjustment for stress (355).

### **3.7.5 Culture and behaviour**

Ethnic differences in health may arise from variations in cultural practices, religion and health behaviours (9). The previous section on cardiovascular risk factors described ethnic differences in various health behaviours (see sections 3.5.3, 3.5.4 and 3.5.5).

The nature and definition of ethnicity means that it is integrally related to a person's culture, a factor that is likely to influence health behaviour. The mechanisms by which ethnicity influences health behaviour are likely to be complex and relate to factors such as socioeconomic circumstances and religion. For instance, qualitative research with South Asian individuals in focus groups in Edinburgh suggested that ethnicity may impact on lifestyle choices because of ethnic differences and ethnic specific barriers in social norms, working practices, food choices, and perceptions of health (356).

Despite descriptive evidence of ethnic differences in health behaviour, interpreting these differences and forming health policy based upon them can be problematic. In particular, attributing ethnic inequalities in health to cultural and behavioural differences requires caution because of the risk of stereotyping heterogeneous groups and making assumptions that certain cultural behaviours are responsible for poor health (9).

### **3.7.6 Biological**

It has been suggested that ethnic inequalities in health could be due to biological differences. A number of potential biological mechanisms for ethnic inequalities in CVD have been suggested. These include vascular, metabolic and genetic differences, as described below. In considering the role of physical differences as a cause of ethnic health inequalities it is important to remember the inter-relationships that exist between genetics, biological traits and the

social and physical environment - the relationship between socioeconomic position, low birthweight and chronic disease is one example of this (9).

#### **3.7.6.1 Genetic**

Genetics may play a role in explaining ethnic inequalities in health, although its role may have been overstated in the past (218). Indeed, it has been suggested that environmental and social exposures are more important determinants of CVD (357). Whilst there are a small number of conditions, predominately specific inherited genetic diseases, that are associated with certain ethnic groups, these would have a very limited impact on broader ethnic health inequalities (9). In addition, genetic variation between ethnic groups is smaller than that seen within groups (9, 218).

Development of CVD and certain risk factors, such as diabetes, is associated with the presence of certain genetic traits, the prevalence of which can vary between ethnic groups (358). The thrifty gene hypothesis is one potential explanation for the origin of genetic differences; this hypothesis suggests that being predisposed to insulin resistance may protect individuals during periods of food restriction, and may have developed in populations such as those in South Asia and Africa (358, 359). Indeed, it has been suggested that this genotype is common in African populations but its expression may be driven by exposure to Westernised lifestyles, i.e. an epigenetic phenomenon (359). However, the origin of this genotype has been questioned as areas such as South Asia are agriculturally productive and capable of supporting large numbers of people (360).

#### **3.7.6.2 Vascular**

There may be ethnic differences in the development and presentation of atherosclerosis. Budoff *et al* found a significantly lower prevalence of coronary artery calcification, a sign of atherosclerosis, in black and Hispanic patients in the USA, with Asian people having a similar prevalence to white people (361). This finding was based on scans performed on patients undergoing coronary angiography, a potential source of selection bias. However, a prospective study from Canada also identified ethnic differences in atherosclerosis, with the highest levels seen in European people followed by Chinese and then South Asian people, although the South Asian group still had higher rates of CVD (258). Furthermore, there is evidence of ethnic differences in the distribution of

atherosclerosis (362), illustrated by Chaturvedi *et al*'s finding that South Asian men have less peripheral (i.e. lower limb) atherosclerosis than European men within categories of similar coronary artery atherosclerosis (363). Additionally, differences in renin activity may lead to ethnic differences in hypertension (364). Renin activity has been found to be lower in black compared to white people, potentially accounting for ethnic differences in hypertension between these groups (364).

### **3.7.6.3 Metabolic**

Ethnic differences in metabolic syndrome have been suggested as a potentially important driver of ethnic inequalities in CVD (62). Metabolic syndrome is characterised by the presence of abdominal obesity, insulin resistance, hypertension and raised triglyceride:HDL ratio (365), and is associated with an increased risk of CVD (366). Prevalence of metabolic syndrome has been found to be higher and increasing in South Asian people (267, 365), and has been suggested as a potential cause of increased levels of CVD in this group (366). The African Caribbean group has also been found to have higher levels of insulin resistance, but have atypical associations with lipids and obesity compared to other ethnic groups (62). In addition, there are ethnic differences in fat deposition and distribution, with high levels of abdominal adiposity seen in South Asian groups, including evidence of ethnic differences in adiposity from infancy (367), and evidence of ethnic differences in visceral deposition of fat, a type of fat that may be underestimated by body mass index measurements (368). It has been suggested that current thresholds used to define metabolic syndrome may underestimate the prevalence of the condition in South Asian people and may need to be adapted with ethnic specific cut-offs, for example in body mass index, waist circumference and glucose measurement (also see section 3.5.8) (365).

### **3.7.7 Prevention and health care access and availability**

Ethnic inequalities in health could arise from differences in the impact of healthcare policies and interventions. There are many potential sources of these differences but for the purposes of this review four key areas, which are particularly relevant to ethnic inequalities in CVD, have been identified. These are ethnic inequalities in access to prevention and healthcare, in the effectiveness of preventative interventions and more specifically the

effectiveness of population approaches, plus ethnic differences in the performance of cardiovascular risk calculators. The previous chapter described that public health interventions can lead to health inequalities if they do not work equally well in population subgroups and particularly if disadvantage accumulates through various stages of the intervention (see section 2.9). This mechanism is potentially relevant for this section, where it is possible that ethnic differences in the impact of the healthcare policies and interventions described could combine in such a way.

#### **3.7.7.1 Ethnicity and access to prevention and healthcare**

Ethnic inequalities in health could arise from differences in access to healthcare, including to CVD prevention interventions. Access is a broad concept that includes service availability, timely uptake, and quality (369); alongside this, access should also be based on health needs and aim to ensure equity (369, 370). This section includes examples from across this broad definition of access, although much of the evidence relates to uptake.

There is strong evidence of ethnic inequalities in access to health care (370). Ethnic inequalities have been identified in access to health care generally, and to preventative interventions, including those for CVD specifically. However, evidence regarding the nature and direction of these ethnic differences is mixed and depends on the intervention, ethnic group and context studied.

Mixed evidence of ethnic differences in uptake of preventative interventions has been identified. For instance, Bansal *et al* found that women from many ethnic minority groups in Scotland, including Pakistani, African and Indian women, had a higher risk of non-attendance for breast cancer screening than white Scottish women, including after adjustment for socioeconomic position (371). In contrast, uptake of childhood vaccinations has been found to be highest in Asian children and lowest in black Caribbean children, with intermediate uptake in white children, in a study from Birmingham (372). Additionally, a study from the USA on uptake of preventative health services found that black individuals were equally or more likely to receive these services than white or Hispanic people (373), although this telephone survey evidence may have been subject to selection bias owing to potential differences in telephone access or availability for interview.

Uptake of preventative lifestyle interventions, such as smoking cessation, may also vary by ethnic group. Whilst ethnic minority individuals are no less willing to quit smoking than the rest of the population fewer attempt to quit using professional services (374). An evaluation of the NHS stop smoking services in England found that between 2001 and 2011 the proportion of people attending the service who were from an ethnic minority group increased from 4 to 7% (125). This figure is likely to underrepresent the proportion of the population who come from an ethnic minority group, and therefore may indicate lower uptake in these populations although ethnic differences in smoking will also be relevant.

There is also evidence of ethnic differences in the uptake of cardiovascular screening programmes, including England's NHS Health Check programme. In contrast to the evidence cited above on cancer screening, evidence suggests that there may be higher uptake of cardiovascular risk screening in ethnic minority groups than in the white population. For instance, both Artac *et al* and Dalton *et al* found higher uptake of NHS Health Check appointments among South Asian and black individuals (162, 163). However, these studies were based on uptake in the earliest years of the programme, which may not reflect ongoing attendance (162, 163). Uptake of cardiovascular screening has also been found to be higher in South Asian and black groups in local screening programmes in Birmingham (375, 376). Whilst the generalisability of these findings may be limited because of the specific design of the programmes, these findings demonstrate the potential to achieve good uptake of cardiovascular risk screening among ethnic minority individuals. Ethnic differences in the uptake of cardiovascular risk factor screening have also been found in the USA, where self-reported uptake of cholesterol screening was initially found to be lower among Hispanic individuals compared to white non-Hispanic (377). However, adjustment for confounding factors including health insurance and socioeconomic position reversed this finding.

Ethnic differences in the diagnosis and treatment of health conditions would be another potential source of health inequalities. However, Nazroo *et al* analysed cross-sectional data from the Health Survey for England and found little evidence to suggest that chronic conditions, such as hypertension or high cholesterol, were less well treated or diagnosed in ethnic minority groups (378). Indeed, evidence from the UK suggested that uptake of cardiac investigations

was higher in South Asian than white civil servants after adjustment for differences in need (379). Ben-Schlomo *et al* investigated the healthcare seeking behaviour of South Asian and white patients admitted to hospital with acute coronary syndrome, and found that whilst there were ethnic differences in how patients arrived at the hospital and whether they received thrombolysis, this did not equate with inequitable care for South Asian patients (380).

The use of preventative medications may vary across ethnic groups. An ecological study from the UK found that in areas with a high South Asian population primary care patients were less likely to be prescribed lipid-lowering medications (381). Similarly, Ashworth *et al* found that prescribing of statins was lower in areas with high proportions of African Caribbean or South Asian people, although the size of the association was small (382). Aspirin use was found to be lower in African-American and Hispanic individuals in the USA compared to white individuals, although this study made no adjustment for health insurance status (383). Adherence to medication has also been found to vary by ethnicity. In a systematic review and meta-analysis, Lewey *et al* compared adherence to statins in non-white and white individuals in the USA (384). They found a crude odds ratio of non-adherence in non-white compared to white people of 1.53, with higher odds of non-adherence also observed in studies that controlled for socio-economic and health insurance status. In contrast, willingness to take antihypertensive medication has been found to be similar in South Asian and white individuals in the UK (385), whilst reported use of statins has been found to be higher in South Asian compared to white people (386). Studies that have assessed uptake of medication for the secondary prevention of CVD in South Asian people have found it to be higher than in white individuals (379, 387), but lower in black patients (387). These differences between the USA and UK could reflect variations in availability, funding or routine recommendations. In addition, adherence to and uptake of medication can be difficult to measure as it often relies on self-report or prescription data that may not reflect actual intake.

Szczepura has postulated that ethnic differences in access to health care could arise from individual factors, such as culture, healthcare seeking behaviour and language, or organisational factors, such as staff training or location of services (370). For instance, Nazroo *et al* explored ethnic differences in primary care and hospital attendance in England (378), indicators of healthcare seeking behaviour

as well as need. They found that black Caribbean, Indian, Pakistani and Bangladeshi adults were significantly more likely to have visited their general practitioner, but that Pakistani, Bangladeshi and Chinese adults were significantly less likely to have attended hospital than white adults. These differences remained after adjusting for self-reported health, although this measure may not fully capture ethnic differences in health need.

There is evidence of ethnic differences in the awareness of the presence of high cardiovascular risk factors, a factor that could reduce uptake of recommended risk reducing interventions. Analysis of general practice data from the UK suggests that patients in areas with a high ethnic minority population may be less likely to have variables such as blood pressure and cholesterol recorded, suggesting possible variations in the quality of primary care services (388). In the USA, awareness and treatment of hypertension was found to be significantly lower in Mexican American people than in non-Hispanic white people after adjustment for confounding factors, including health insurance status (389); awareness and treatment of dyslipidaemia was also been found to be significantly lower in African-American compared to white people (390).

Qualitative research has been used to identify specific barriers that could affect adoption of healthy behaviours and access to health interventions in ethnic minority communities. For example, Horne *et al* found a number of similarities between older South Asian and white individuals in their attitudes towards physical activity, but also identified a number of issues specifically affecting South Asian people (391). These included language barriers, religious requirements for fasting, and attitudes towards modesty and gender segregation; issues that could have implications for provision of health promoting activities. In another qualitative study, Grace *et al* carried out focus groups with the Bangladeshi community in Tower Hamlets to explore attitudes towards diabetes prevention (392). They also found concerns related to gender segregation and language barriers, in addition to the important role of hospitality in influencing food choices. This qualitative evidence highlights a number of potential explanations for ethnic differences in access to healthcare, although these findings may be relevant only to the particular populations studied. Stereotyping by health professionals has also been identified as a barrier to accessing CVD prevention interventions (393), and it is important to remember the heterogeneity that exists within and between ethnic groups.

In addition to the ethnic differences outlined in this section, access to healthcare has also been found to vary by gender, age and socioeconomic position (208). Given that demographic characteristics and socioeconomic position are known to vary by ethnicity these factors may also contribute to ethnic inequalities in access.

### **3.7.7.2 Ethnicity and effectiveness of cardiovascular disease prevention interventions**

Ethnic differences in the response to CVD prevention interventions could lead to ethnic inequalities in CVD. This could arise from ethnic differences in the response to commonly used medications or lifestyle interventions. Evidence for these ethnic differences is mixed and much of it comes from comparisons between black and white individuals in the USA.

Ethnicity may impact on drug response, for example to antihypertensive or lipid-lowering medication (394). Evidence for ethnic differences in drug response comes from a range of study types, including large randomised controlled trials, although the availability of evidence is limited by lack of reporting of ethnicity in some trials (395, 396). The outcomes reported vary - some studies report changes in risk factor levels following medication whilst others also report cardiovascular events, a more clinically relevant outcome. A key example is the difference between black and white individuals in response to antihypertensive medication. A number of studies have evaluated the efficacy of different types of antihypertensive medication in black and white individuals. A general finding is that calcium channel blockers and diuretics are more effective in black individuals, whilst beta-blockers and ACE inhibitors are more effective in white individuals (397-400). In the UK, Gupta *et al* compared blood pressure changes from a beta-blocker (atenolol) and calcium channel blocker (amlodipine) in European, black and South Asian hypertensive patients (398). They identified no ethnic differences in blood pressure reductions from taking amlodipine, but blood pressure did not fall with atenolol in black participants while it decreased in white and South Asian people. In a separate analysis of a large antihypertensive medication trial (ALLHAT) the impact of ACE inhibitors and calcium channel blockers on blood pressure and cardiovascular outcomes were compared in black and non-black participants (399). The calcium channel blocker was found to lower blood pressure more in black individuals than the ACE inhibitor, a difference that was not observed in non-black individuals.

However, despite these differences in blood pressure the relative risk of cardiovascular outcomes was broadly similar between black and non-black individuals. In contrast, a systematic review and meta-analysis of randomised controlled trials of antihypertensive medication in white and African-American women found that the use of these medications resulted in a greater risk reduction for cardiovascular events in African-American compared to white women (401). However, in this systematic review most of the evidence from African-American women came from a single trial, limiting its generalisability (401). Given the increased risk of hypertension and stroke in black individuals effective treatment of hypertension is particularly important in this group. However, it has been highlighted that there is a lack of evidence regarding cardiovascular outcomes in this area (397).

Whilst much of the literature focuses on differences between black and white people in response to antihypertensive medication (364), there may also be differences between other ethnic groups. Although it has been suggested that response to antihypertensive medication is similar in South Asian and white people (402), there is also evidence of potential ethnic differences. The PROGRESS trial, a randomised controlled trial of the ACE inhibitor perindopril in patients with cerebrovascular disease in Asian (Chinese and Japanese) and Western locations, found larger reductions in blood pressure in Asian compared to Western patients (403). This trial observed a 38% reduction in major cardiovascular events in Asian participants compared to a 20% reduction in Western participants, although the confidence intervals for these risk reductions overlapped and the difference could partially be explained by differences in the prescribed dose by bodyweight (403).

There may be ethnic differences in response to statins, although the evidence is less clear than for antihypertensive medication. A randomised controlled trial of rosuvastatin found ethnic differences in lipid profile changes with the medication, for instance a smaller relative reduction in LDL-cholesterol in non-white individuals (404). However, the hazard ratios for cardiovascular events were similar in white and non-white individuals, although small numbers of ethnic minority individuals limited the ability to undertake further subgroup analyses (404). In contrast, a randomised controlled trial that included the use of atorvastatin found no statistically significant differences in the effect of the medication on the lipid profile of white, black and South Asian individuals (405).

In a different type of study, in this case a retrospective cohort study of diabetic patients, Brunner *et al* found that statin prescription was associated with similar benefits in all cause mortality across South Asian, Chinese and white groups (406). Whilst there is evidence that the effectiveness of statins may be similar across ethnic groups, it has been suggested that Asian individuals may achieve these benefits at a lower dose than Western individuals (407). A prospective cohort study from Japan in which patients with high cholesterol were prescribed low-dose simvastatin identified changes in cholesterol concentrations comparable to Western studies that used higher doses of medication (408).

Many of the explanations suggested for these ethnic differences in drug response relate to biological or genetic differences. Differences between black and white people in renin activity and nephron mass, which affect the pathophysiology of hypertension, have been identified (364, 402). Renin activity has been found to be lower in black hypertensive people compared to white, a factor that could influence the relative efficacy of different types of antihypertensive medication (364). Genetic differences may also play a role if they influence the response to or metabolism of medication (394, 407). A number of potential genetic differences have been identified (394), although there is also evidence that the pharmacokinetics of statins may be similar in white, Asian, black and Hispanic individuals (409).

Ethnic differences in drug response may influence prescribing practices and drug development. Indeed, UK guidelines for the prescription of antihypertensive medication advise different medications for black individuals (122). In the USA, a drug for heart failure, BiDil, has been developed only for use in African-American individuals (410). However, the complex relationship between ethnicity, biological differences and social determinants of health is an important consideration in determining the appropriateness of this type of approach. Whilst ethnicity will reflect some biological and genetic differences between populations, it is a largely social construct, and may therefore be a poor predictor of biological determinants of drug response (410). Ethnic differences in drug response could arise from other differences for which ethnicity as a marker, such as environmental or lifestyle differences, which could potentially impact on biological response to medication. Genetic characteristics may directly influence response to medication, but if not all members of an ethnic group have those characteristics, ethnically determined

use of medication may be less effective or appropriate for certain individuals (227). In fact, Sehgal found a notable overlap in the response to antihypertensive medications in black and white individuals, suggesting that this indicated similarity rather than difference between the ethnic groups and highlighting that ethnic differences in drug response are often smaller and less significant than differences observed within ethnic groups themselves (400).

Perhaps related to ethnic differences in drug response is the common observation that control of cardiovascular risk factors varies by ethnicity. In the UK, black general practice patients known to have hypertension have been found to be less likely to have a blood pressure at or below recommended treatment targets than white or South Asian patients (411), although there is evidence that this difference may have improved following changes to primary care contracts (412). Likewise, Schofield *et al* analysed primary care data from London and found that black patients were significantly less likely to have controlled hypertension than Asian and white patients (413). In contrast, Nazroo *et al* found that black Caribbean adults had similar levels of blood pressure control as white adults, and Indian, Pakistani and Bangladeshi adults were less likely to have uncontrolled cholesterol levels than white adults (378). These differences could reflect variations in study design and data used, including the use of self-reported information in Nazroo *et al*'s study and primary care data in Schofield *et al*'s, both of which will be subject to their own limitations. In the USA, black and Mexican-American hypertensive patients have been found to be less likely to have their hypertension controlled than white patients (276, 277, 389), and African-American people less likely to have lipid concentrations controlled (390). There are likely to be multiple explanations for these differences in control. Whilst differences in drug response are a possible explanation, they could also be due to differences in prescribing, uptake, adherence and availability of medication.

The impact of lifestyle interventions, including smoking cessation and diet, may also vary by ethnicity. Smoking cessation is associated with a sizeable reduction in the risk of CVD and evidence suggests that this benefit may be similar across ethnic groups. Analysis of a USA cohort study, which benefitted from a large sample size, found that the cardiovascular risk reduction associated with smoking cessation was similar in African-American and white people (414). Routine data from England's stop smoking services indicates that the proportion

of smokers who set a quit date and then successfully quit (at 4 weeks) is similar across most ethnic groups, but lower in black Caribbeans (51% of white smokers, compared to 52% of Asian smokers and 44% of black Caribbean smokers) (415). Another method of reducing smoking rates is the use of health warning labels on cigarette packaging. A web based experimental study from the USA tested the impact of text and pictorial warnings on white, African-American and Hispanic smokers and found that all ethnic groups responded more to pictures rather than text warnings, and that Hispanic and African-American smokers had greater responses to both types of warning than white smokers (416). Ethnically adapted smoking cessation interventions have been developed and may have a role in ensuring good and equitable outcomes, although evidence of their added benefit in terms of effectiveness is limited (417).

There is mixed evidence of ethnic differences in response to dietary interventions. He *et al* carried out a study in the USA in which 71 white and 33 black patients with hypertension were put onto short-term high and low sodium diets and had their blood pressure monitored (418). Blood pressure levels fell more following the switch from high to low sodium intake in black participants, who also had a smaller change in renin activity, than in white participants. Adjustment for changes in renin activity eliminated the ethnic differences in blood pressure response, suggesting that this was an important driver of the observed differences. This suggests that dietary interventions based on reduced salt intake could have differential effects across ethnic groups. Dietary salt reduction may also lead to ethnic differences in other physiological markers, such as urinary albumin (419). Weight loss is also an important way to reduce cardiovascular risk and prevalence of overweight and obesity is known to vary by ethnicity. Analysis of a USA prospective cohort study examined whether there were differences between African-American and white individuals in the impact of weight loss on hypertension (420). No clear ethnic differences were observed, with weight loss leading to similar reductions in levels of hypertension in both groups. Although this study had the benefit of observing the impact of weight loss in a large sample outside of an interventional setting, the analysis did not account for any differences in co-morbidity or physical activity.

### **3.7.7.3 Ethnicity and effectiveness of population approaches to CVD prevention**

Ethnic differences in the effectiveness of population approaches to CVD prevention could impact ethnic health inequalities. It has been suggested that population approaches may be more beneficial for health inequalities than high-risk approaches (5), however there is mixed and limited evidence for the effect of population approaches on ethnic health inequalities (421, 422).

One source of evidence is studies on the impact of folic acid interventions in the USA. Dowd *et al* found that folic acid fortification reduced absolute inequalities but increased relative inequalities in folate levels between black and white individuals (421). A systematic review of the effect of increasing folic acid intake found mixed evidence of the impact of population and high-risk approaches on ethnic inequalities, although the review suggested that population approaches may be more likely to reduce inequalities (422).

Ethnic differences in the impact of population approaches could arise from differences in the baseline exposure being targeted or in response to the policy. Two examples of exposures that could be addressed with population approaches are salt intake and dietary fat intake (18), both of which may vary between ethnic groups. Millett *et al* analysed data from the Health Survey for England and found ethnic differences in the addition of salt to food (423). Indeed, it has been suggested that South Asian and black individuals may get a higher proportion of their salt intake from cooking and table added salt, compared to the general population who get a higher proportion as hidden salt in processed food (424). Therefore a population intervention to reduce overall salt intake by reducing the amount of hidden salt in processed food could lead to ethnic differences in overall salt consumption. Similarly, there may be ethnic differences in the consumption of harmful dietary fats such as trans fats that could lead to differential impact of eliminating them from the food chain (425). There are sources of artificial ghee available in northern India that contain 50% trans fats; in the UK high levels of trans fats have been found in takeaway food, which may be consumed more frequently by people from ethnic minority groups (135). Therefore, ethnic minority groups could potentially benefit more from the elimination of trans fats from takeaway food.

Ethnic differences could also arise from variations in the response to population approaches. For instance, evidence suggests ethnic differences in blood pressure

response following salt reduction (426). As described previously larger blood pressure falls in response to a low sodium diet had been found in black compared to white individuals (418). Bibbins-Domingo *et al* modelled the impact of salt reduction in black and white individuals in the USA and found larger reductions in CVD in black people (131). Their model included a larger blood pressure reduction in black individuals but they highlighted that even without this difference there would still be a greater percentage reduction in CVD incidence from salt reduction in black individuals because of a higher baseline prevalence of hypertension. Conversely, a recent Cochrane systematic review and meta-analysis found that blood pressure falls after modest salt reduction were similar in white, black and Asian individuals (427). The relevance of these findings to the question of ethnic differences in the impact of population approaches may be limited as this evidence is based on response to individual dietary interventions. One exception to this is a study by Millett *et al* who investigated differences in salt intake between 2003 and 2007, when a population salt reduction strategy was being implemented in the UK (423). They did not identify ethnic differences in the absolute decrease in salt intake during this period, with higher intake among ethnic minority individuals remaining.

The implementation of smoke-free legislation in many countries in recent years has enabled investigation of ethnic differences in response to this population intervention. In the USA, there is evidence that smoke-free legislation may be associated with smaller falls in current smoking and cotinine concentrations in ethnic minority groups, with more research needed to understand this difference (428). Indeed, the prevalence of exposure to second-hand smoke was found to be higher in non-Hispanic black people than non-Hispanic white and Mexican American people in a cross-sectional study from the USA (429), although this study did not specifically compare ethnic differences in secondhand smoke exposure between areas with and without smoke-free legislation. Qualitative research from the UK suggests that ethnic minority individuals may have responded differently to the introduction of smoke-free legislation. Lock *et al* interviewed Turkish, Somali and white smokers before and after the introduction of legislation in England (430). They found ethnic differences in the impact of the legislation on smoking habits at home. For instance, Somali women found that they had to hide their smoking or smoke at home rather than in public following the legislation. In another qualitative study, Hight *et al* interviewed

and carried out focus groups with Bangladeshi smokers (431). The participants discussed the importance of respecting elders in their community, the use of different types of tobacco, and the impact of working environments on smoking practices, factors which are likely to vary by ethnicity.

In summary, these examples suggest potential mechanisms by which population approaches could perform differently across ethnic groups. The evidence is mixed, with no suggestion that commonly proposed population approaches would perform systematically worse or better in ethnic minority groups. Rather, any ethnic differences that did occur are likely to be variable in nature and direction, and would depend on other factors such as socioeconomic position and environment.

#### **3.7.7.4 Ethnicity and cardiovascular risk calculation**

The performance of cardiovascular risk calculators can be impaired if they are used in different populations from the one in which they were derived, particularly if the background incidence of CVD is different in that population or has changed over time since the original study that derived the risk calculator (432). Given that rates of CVD vary between ethnic groups, and cardiovascular risk calculators incorporate populations' baseline cardiovascular risk, it is unsurprising that the performance of cardiovascular risk calculators has been found to vary by ethnicity (433). D'Agostino *et al* analysed the performance of the Framingham equation in an ethnically diverse range of cohort studies and found that discrimination (the ability of the equation to distinguish between those who will and will not develop CVD) was similar by ethnicity but calibration (a measure of the agreement between actual and predicted outcomes) varied (434). Discrimination is a measure that is particularly relevant to this thesis as it relates to questions of whether the individuals at highest cardiovascular risk are accurately identified. In this study, observed and predicted rates of IHD events were similar in black and white individuals but Framingham overestimated the risk of IHD events amongst Japanese and Hispanic men. Quirke *et al* compared risk predictions from the Framingham equation with published mortality data across ethnic groups in England (266). They found that the pattern and direction of ethnic differences in IHD risk was consistent with those seen in mortality data, but that the Framingham equation underestimated cardiovascular risk in higher risk ethnic groups and overestimated it in lower risk groups. This scenario

was also observed in New Zealand where the Framingham equation was found to underestimate cardiovascular risk in a combined group of Maori, Pacific and Indian individuals, and overestimate risk in lower risk European individuals, although data limitations prevented analysis of individual ethnic groups (435). Likewise, in Australia, the Framingham equation was found to significantly underestimate CHD risk amongst an Aboriginal community where the risk of CVD is known to be particularly high (436). This evidence suggests that the Framingham equation underestimates risk in high-risk ethnic groups and overestimates it in low-risk ethnic groups (266). Systematic inaccuracies in cardiovascular risk calculation across ethnic groups such as this could reduce the cost effectiveness of cardiovascular screening (437), lead to ineffective allocation of health care resources (438), and worsen existing health inequalities by reducing access to preventative interventions in higher risk groups when treatment decisions are made using cardiovascular risk thresholds (437, 439). This could have a significant impact on public health if ethnic groups with a higher risk of CVD form a large proportion of the population (151).

Ethnic differences in the association between cardiovascular risk factors and outcomes could affect the performance of cardiovascular risk calculators (432). There is mixed evidence as to whether this type of difference occurs (see section 3.5.8), however studies that have explored the performance of Framingham risk factors suggest that the associations are consistent across populations. Hurley *et al* used cross-sectional data from the USA, with follow-up for mortality outcomes, to assess whether there were ethnic differences in the association between cardiovascular risk factors and mortality (265). They found that the relative risk associated with Framingham risk factors was consistent across ethnic groups and that these risk factors were able to produce accurately calibrated models in different ethnic groups. In addition, consistency between estimated cardiovascular risk scores and mortality rates, despite issues of relative over or underestimation, indicates that traditional cardiovascular risk factors play a role in ethnic differences in CVD (266).

Inaccuracies in cardiovascular risk calculation in deprived socioeconomic groups may be particularly relevant to ethnic minority groups. The Framingham equation does not include any measure of socioeconomic position, although this is a recognised risk factor for CVD. It has been suggested that this could lead to a systematic underestimation of cardiovascular risk in deprived individuals (440,

441), and therefore in ethnic minority groups in which socioeconomic deprivation is over-represented. Two studies from the UK have assessed the performance of the Framingham equation in different socioeconomic groups. Brindle *et al* compared observed cardiovascular mortality rates with those estimated by the Framingham equation across socioeconomic groups in a relatively deprived sample of the Scottish population (440). They found that the Framingham equation consistently under predicted the risk of cardiovascular death, and more so in more deprived individuals. Whilst Ramsay *et al* also found that the Framingham equation underestimated the risk score in deprived individuals compared to affluent ones, overall they found that Framingham overestimated cardiovascular risk in their study of British men (441). This difference could be accounted for by differences in the baseline incidence of CVD between the studies. In these studies the social gradient in observed CVD was greater than that predicted by the Framingham equation (441, 442). Given the use of a single threshold of cardiovascular risk in determining treatment decisions, this means that affluent individuals may be more likely to receive CVD prevention interventions than deprived individuals, potentially exacerbating socioeconomic health inequalities (442). One response to this issue has been the development of the ASSIGN risk score in Scotland (443). ASSIGN incorporates area deprivation in its risk calculation, and whilst its overall performance has been found to be very similar to that of the Framingham equation, including in black populations in the UK (444), it may perform more equitably (443). Given higher levels of socioeconomic deprivation in ethnic minority groups any systematic inaccuracy by socioeconomic position, in addition to that from ethnicity, would add to the potential for cardiovascular risk calculation to perform poorly in deprived ethnic minority individuals.

A number of approaches have been used to improve the accuracy of cardiovascular risk estimation in different ethnic groups, including recalibration of existing equations, simple adjustment approaches, and the creation of new equations that include ethnicity. Recalibration can occur by updating the baseline incidence rates on which the equation is based along with prevalence of cardiovascular risk factors. In the UK, Brindle *et al* adopted a pragmatic approach to recalibrate the Framingham equation to ethnic minority groups (445). Prospective data from ethnic minority groups containing incidence rates of CVD were not available so they used cross-sectional data instead, substituting

prevalence for incidence. This resulted in the creation of an updated, though unvalidated, equation that allowed the calculation of cardiovascular risk by ethnic groups - "Ethrisk". Barzi *et al* recalibrated the Framingham equation to a Chinese population and found that this improved the accuracy of risk prediction, so much so that they concluded that there was no need to develop a new equation for this Asian population (432). A number of simple adjustment approaches have also been suggested that do not require the more complicated recalibration process. National UK guidelines have previously suggested that the Framingham score in South Asian men should be multiplied by 1.4 (121, 144). However, this straightforward adjustment does not account for differences in risk in women or other ethnic minority groups. Aarabi and Jackson suggested that the age of South Asian individuals should be increased by 10 years when calculating cardiovascular risk, although this solution was suggested at a time when paper-based tools were more commonly used and a simple adjustment process was needed (446). Cappuccio *et al* suggested that lower thresholds should to be used to define high-risk individuals in high-risk ethnic groups (447). This particular conclusion was made at a time when it was more common to estimate risk of CHD and then multiply it in order to calculate CVD risk. Given that the risk of stroke varies by ethnicity this approach could underestimate risk of CVD in some ethnic groups, however it is now recommended practice to calculate CVD rather than CHD risk.

An arguably more significant step to improving the accuracy of cardiovascular risk calculation in ethnic minority groups is the creation of new calculators that include ethnicity. Indeed, it has been suggested that models that incorporate ethnicity may be necessary in ethnically diverse populations in order to accurately discriminate between people who will or will not develop CVD (151). One key example of this in the UK is the QRISK2 calculator (438). This updated version of QRISK contains both ethnicity and socioeconomic deprivation as independent risk factors of CVD. It was derived from a prospective cohort of primary care patients in England and Wales, taken from a large electronic database. Independent validation of this score found that it performs better than Framingham in a general UK population, with better accuracy, discrimination and calibration (448, 449). However, a limitation of QRISK2 is that the original dataset contained a large proportion of missing data. In particular, ethnicity was recorded in only 27.1% of women and 23.8% of men with the

remaining individuals assumed to be white (438). This assumption may have led to an underrepresentation of ethnic minority individuals in the cohort (438). Two recent studies have assessed the performance of QRISK2 among ethnic minority groups in the UK. Tillin *et al* compared the performance of QRISK2 and Framingham in an ethnically diverse London cohort (450). They found that calibration of the scores varied by ethnicity and gender; for instance they found that both QRISK2 and Framingham under predicted cardiovascular risk in South Asian women whilst both scores were fairly accurate in South Asian and European men. Correct classification of individuals as high or low risk was poor in African Caribbeans and classification by QRISK2 was also poor in South Asian women. The authors concluded that there was no evidence that either QRISK2 or Framingham performed better than the other score. However, the baseline data on which their risk calculations were based were over 20 years old and may not reflect individual changes in incidence and prevalence of cardiovascular risk factors. Schofield *et al* also used data from an ethnically diverse area of London to compare the performance of a variety of cardiovascular risk scores in the black population, including QRISK2 and Framingham (444). QRISK2 was the only equation that did not appear to over predict cardiovascular risk when compared with national data. The authors concluded that QRISK2 might provide the most accurate estimation of cardiovascular risk in black individuals in the UK (444).

#### **3.7.7.5 Section summary**

This section has outlined how a variety of interventions used in CVD prevention could impact on ethnic health inequalities. Whilst evidence for ethnic differences in access to healthcare, or the performance of individual and population interventions is at times mixed or limited, there is stronger evidence that cardiovascular risk calculators need to consider ethnicity.

### **3.8 Ethnicity and area of residence**

The previous section of this chapter reviewed the evidence for a variety of potential explanations for ethnic health inequalities. This section now moves on to consider another potential explanation - area of residence. This is because, as the evidence reviewed here suggests, there are ethnic differences in the areas people live in and in the association between area and health, differences that could lead to ethnic inequalities in health (303).

Ethnic minority populations have been found to be concentrated in deprived areas and segregated from other parts of the population (451, 452). This pattern of residence occurs in the UK, USA and other countries including New Zealand and Holland (451, 453-455). Concentration of ethnic minority groups in deprived urban areas in the UK stems from historical migration patterns and socioeconomic opportunities (451, 452). Post-war migration from former colonies to the UK produced concentrations of ethnic minority groups, driven by geographically centered employment opportunities, such as in the textile industry in the North of England, alongside limited and discriminatory housing choices (452). Movement of white populations to suburbs of cities added to the concentration of these communities, although some ethnic minority populations have now started to spread into other parts of the country (452, 456). Broadly speaking this distribution of population applies to many of the largest ethnic minority groups in the UK, although there are ethnic and geographical differences in the degree of concentration in deprived areas (308, 309). For example, Pakistani and Bangladeshi groups are most concentrated in deprived areas (457); the Chinese population is not concentrated into deprived areas in the same way as other ethnic minority groups (309); Indian, Pakistani and Bangladeshi people have been found to be more concentrated into areas populated by people of their own ethnicity compared to black Caribbean and Chinese people, who are less concentrated (456, 458); and greater concentration of ethnic minority communities is seen in cities in the North West of England (179). These ethnic variations in population distribution reflect differences in the factors which determine where individuals and communities are located, for instance the influence of migration, housing availability, employment opportunities, racism, living within a community, religion, and language skills (451, 457). It should be noted, however, that in the UK segregation by socioeconomic position is greater than segregation by ethnic group (179).

Segregation of ethnic minority groups is also seen in the USA, to an even greater extent than in the UK. Black people in the USA are the most segregated group (459, 460), and are concentrated into areas of greater deprivation than white individuals (453). Areas populated by the poorest black people are more deprived than those populated by the poorest white people (461). Like the UK, segregation in the USA is related to discrimination, including in the housing system, and socioeconomic differences (318, 459, 462). However, the

relationship between socioeconomic position and segregation is not straightforward. Segregation does not just affect poorer ethnic minority people but is also seen in more affluent groups, although to a lesser extent (459, 462); in addition, there are ethnic differences in the relationship between segregation and socioeconomic position (462).

It is possible that the concentration of ethnic minority groups into deprived areas or residential segregation of these groups could impact on health. Indeed, segregation has been suggested as being a key cause of ethnic inequalities in health (460). Health could be affected by differences in the quality of areas that different ethnic groups live in, the impact of living in segregated areas on socioeconomic position, or through ethnic density effects.

Area can influence health through the quality of physical environments, i.e. contextual effects (see section 2.8.2.2.2). It has been suggested that areas populated by high numbers of people from ethnic minority groups may have a physical environments that are more detrimental to health. Whilst an unhealthier physical environment could also be related to socioeconomic deprivation, this may not account for all the differences seen. For instance, areas populated by high numbers of black individuals in the USA have higher levels of pollution and industry, with low quality housing, fewer services, and more fast-food shops than white areas of comparable socioeconomic deprivation (459, 460). In the UK, Molaodi *et al* found that areas with high concentrations of ethnic minority populations had more fast food shops (463). However, they also found that some of these areas had more supermarkets and facilities for physical activity, although this ecological study was not able to look at differences in the quality of food or facilities available.

In addition to being a possible cause of the concentration or segregation of ethnic minority groups, socioeconomic position may also be worsened by segregation. Areas with high proportions of ethnic minority people have been found to be more deprived than areas with low proportions, with lower levels of car access, education and central heating (464). Whilst it has been suggested that areas with high concentrations of ethnic minority populations may benefit socioeconomically, for example because of fewer barriers from discrimination or language, there may be detrimental socioeconomic effects (464). Clark and Drinkwater found that male unemployment was higher in more ethnically

concentrated areas, although they also highlighted that this finding could have been affected by bias if employment status influenced a person's decision about where to live (464). Income and employment opportunities may be relatively worse for ethnic minority individuals living in deprived areas compared to ethnic majority groups (451). In addition, segregation in the USA is associated with differences in education, employment opportunities and access to good quality healthcare, to the detriment of ethnic minority groups living in deprived areas (318, 460).

Whilst areas in which ethnic minority groups are concentrated may have poorer physical environment, and living in these areas may exacerbate socioeconomic inequalities, it is also possible that living in an area with people of the same ethnicity may be beneficial for health - otherwise known as the ethnic density effect (461, 465, 466). Evidence on whether such an effect exists is mixed. Some studies have found a positive association between ethnic density and improved health. For example, Stafford *et al* found that increased ethnic density was significantly associated with lower levels of limiting long-term illness in white and Bangladeshi people in the UK (467). Similarly, increased ethnic density has been found to be associated with improved self-rated health in Maori individuals in New Zealand after adjustment for area and individual deprivation (455). Conversely, other studies have found no association between ethnic density and health (458, 468). In the UK, analysis of cross-sectional survey data from the 1990s did not find an association between ethnic density and self-reported health in Indian, Pakistani, Bangladeshi or black Caribbean people (468). Indeed, there is some evidence from the USA that ethnic density could have a negative effect on health, with the finding by Kirby *et al* that living in an area with a high Hispanic population was associated with higher body mass index for Hispanic individuals (469). The conclusions of narrative and systematic reviews reflect this mixed evidence (461, 466). For instance, Becares *et al* systematically reviewed evidence for an ethnic density effect on physical health, and found some evidence that living in an area of high same ethnic density may be beneficial for Hispanic people in the USA, although the opposite may be true for black people (466); evidence from the UK showed no clear ethnic density effect, although small sample sizes in the studies reviewed may have limited these findings (466).

These results refer to people living in areas with a high proportion of people of the same ethnicity but there is also evidence that high ethnic density of one group may influence the health of people from other ethnic groups. For instance, white individuals living in areas with high density of ethnic minority populations have been found to have better general health, and vice versa (468). In addition, analysis of the Health Survey for England indicated that living in an area with a high density of non-white individuals is associated with lower alcohol intake in people from all ethnic groups, including white people (470). It has been suggested that religion may play a role in this relationship between ethnicity and alcohol consumption, supported by evidence from Holland that the proportion of Muslims living in an area can have a small impact on alcohol consumption in Dutch individuals (454).

Ethnic density may affect health because it influences levels of discrimination or social capital. Becares *et al* used cross-sectional data to assess the association between racism, ethnic density and health (471). Their findings indicated that racism occurred less often in areas with high ethnic density and that ethnic density may affect the association between racism and mental health, although a lack of statistical power may have limited the strength of their findings (471). In the USA, Borrell *et al* were able to use prospective cohort data to investigate the association between discrimination, ethnic density and health behaviours, and found that ethnic density did not impact on the relationship between discrimination and health behaviour (472). In contrast, another study from the USA found that discrimination and neighbourhood stressors were positively associated with hypertension, with ethnic minority groups experiencing greater levels of stress than white individuals (473). This study did not specifically investigate ethnic density but the findings suggest that area characteristics related to racism and stress may impact on health. Whilst discrimination may have a negative effect on health, it has been suggested that increased social support and capital may explain the positive ethnic density effect on health (468). Becares and Nazroo investigated this association using mixed methods (458). They found a strong association between social capital measured at an area level and ethnic density. Their qualitative research also suggested ethnic differences in perceived importance and strength of social networks. However, their measurement of the association between ethnic density and mental health,

with or without adjustment for social capital, was generally non-significant across ethnic groups.

There are methodological challenges that effect the investigation of ethnic density effects. These include how ethnic density is measured, such as whether it is based on individual perception or quantitative measures (467). Many studies in this area are ecological or cross-sectional, leading to difficulty in establishing the direction of the association between area characteristics and health (461). There is uncertainty as to what defines an area as being ethnically dense, with arbitrary thresholds used in some studies (461, 469). Also, as with other studies on the relationship between area and health, it is unclear which geographical level of analysis is most appropriate to use and whether small areas correspond to meaningful neighbourhoods (474). Another complicating factor in investigating the association between ethnic density and health is the role of socioeconomic position. As described above, socioeconomic position may drive ethnic minority concentration and segregation as well as potentially being worsened by it. If there were a positive association between ethnic density and health, and ethnically dense areas are more deprived, the effect of one may cancel out that of the other - i.e. the benefits of ethnic density may be cancelled out by the negative impact of socioeconomic deprivation (461, 468). This makes disentangling the effects of socioeconomic position and ethnic density important but difficult in practice, especially given the limitations of the measures available for both of these variables.

Aside from the impact of ethnic concentration and segregation into deprived areas, there may be ethnic differences in the impact that area characteristics have on health. Evidence of this comes from Holland, where Agyemang *et al* identified ethnic differences in the association between area characteristics and blood pressure (475). Using multilevel modelling they found that the association between area characteristics, such as green space and neighbourhood stressors, and blood pressure was greater in ethnic minority compared to Dutch individuals, although many of the associations observed were not significant. Cross-sectional evidence from the UK suggests ethnic differences in the association between area deprivation and self-reported health (476). In this study the gradient observed between increasing area deprivation and poorer self-reported health in ethnic minority groups was notably shallower than the gradient for white individuals. However, the authors discuss that the area

deprivation measure used (the Index of Multiple Deprivation) may not have accurately reflected the greater degree of socioeconomic deprivation seen in ethnic minority groups (476). In the USA, Diez Roux *et al* found that the association between area characteristics and smoking in young adults was not as strong in black compared to white individuals, although the diversity of area characteristics observed in the black group was limited as they were concentrated into deprived areas (477). In contrast, another study from the USA found comparable associations between area socioeconomic characteristics and IHD in black and white individuals (478). However, the generalisability of these findings may be limited because all of the black participants in this study came from a single area.

Clark and Drinkwater suggest that the fact that ethnic minority populations are concentrated into deprived areas means that policies which target deprived areas could be particularly useful for ethnic minority groups (464). Indeed, by targeting programmes at socioeconomically deprived areas in the UK, good coverage of ethnic minority populations may have been achieved even though they were not specifically targeted (300). In a US context, Williams and Collins suggest that policies to tackle racial health inequalities should target segregated areas populated by minority populations, given the nature and impact of racial segregation (460).

This evidence suggests that area based interventions may be a useful and appropriate way of tackling ethnic health inequalities. However, there are practical questions regarding how programmes select areas to target. One approach is to use area measures of socioeconomic deprivation, such as the Index of Multiple Deprivation, however there are a number of reasons why these measures may perform differently across ethnic minority groups. First, if area is used as a proxy for individual socioeconomic position it may misclassify people, and this misclassification may vary by ethnicity. Diez Roux *et al* used data from three large, American studies to analyse the association between area and individual socioeconomic measures (479). They found that misclassification of individual socioeconomic position by area varied between black and white individuals with black people more likely to live in areas with lower median household income irrespective of their individual income. However, odds ratios for the association between area and individual measures were similar. Second, measures such as the Index of Multiple Deprivation are based on the aggregation

and ranking of data from a majority white population (175), and may not fully reflect the extent of socioeconomic deprivation within ethnic minority groups. As described previously ethnic minority individuals in the same socioeconomic grouping, for example occupational class, as white majority individuals may be comparatively worse off. This means that these socioeconomic classifications, which may be constituent parts of area deprivation measures, may not accurately reflect ethnic differences in socioeconomic position (307). This would limit the accuracy of area deprivation measures in research involving ethnic minority groups (476), and in targeting deprived communities. Third, people from ethnic minority groups may choose to remain in deprived areas, where their community is established, rather than move to more affluent areas. This has been observed to be the case in Leeds, with middle class families from ethnic minority groups living in deprived urban areas because of social ties and protection from discrimination, rather than moving out to the suburbs as white families may do (480). Conversely, it was also observed that less affluent ethnic minority individuals were able to move to affluent areas because of resources arising from larger extended families (480). Having reasons to live in deprived areas that are not related to socioeconomic position could reduce the accuracy of area deprivation as a proxy for individual socioeconomic position, and its usefulness in targeting deprived ethnic minority individuals. Fourth, the Index of Multiple Deprivation has been criticised for potentially underestimating socioeconomic deprivation in urban areas (481). One criticism is that it measures access to services - this may appear better in urban areas despite other non-physical barriers that may be especially relevant for ethnic minority groups, such as those arising from cultural differences (481). Given that ethnic minority populations are concentrated into deprived urban areas (309), this criticism could be particularly relevant. Most of these issues relate to individual characteristics, and the implications of them will depend on the relative importance of area versus individual characteristics for health and socioeconomic deprivation.

### **3.8.1 Section summary**

This section highlights that area may cause ethnic differences in health because of variations in the areas people live in plus potential ethnic differences in the association between area and health. However, it also raises practical issues relating to methodological challenges for research and achievement of effective

and accurate targeting of public health interventions for ethnic minority groups; in particular, the issue that area based measures of deprivation may not work equally well across ethnic groups (307). This latter point is the subject of the next chapter.

### **3.9 Chapter summary**

This chapter has reviewed evidence relating to ethnicity and CVD, including potential causes of ethnic inequalities in CVD. It can be seen that ethnicity is an important, though complex, epidemiological variable. With increasing ethnic diversity in the UK and well-established ethnic inequalities in health and CVD, it is becoming increasingly important to understand the relationship between ethnicity and health and to ensure that healthcare interventions promote, and do not exacerbate, ethnic health inequalities. Many of the issues raised here, and in the previous chapter, are relevant to the rest of this thesis, in particular the use of area based measures of deprivation to target public health interventions, the performance of cardiovascular risk calculators across ethnic groups, and the effectiveness of population approaches.

### **3.10 Questions arising from the evidence**

The aim of this thesis is to explore whether there are ethnic differences in the potential effectiveness, cost and equity impact of CVD prevention policies designed for the general population. From the literature review in this, and the previous chapter, two policy choices have been identified for investigation - the choice between population and high-risk approaches to CVD prevention, and, within the high-risk approach, the choice between mass and targeted screening for high cardiovascular risk. Ethnic differences in CVD and its risk factors and determinants suggest that there may be ethnic differences in the potential impact of these policy options, however it is unclear from the evidence whether this is the case.

Chapter 6 investigates whether there are ethnic differences in the potential impact of population and high-risk approaches to CVD prevention in terms of prevention of cardiovascular events and changes in ethnic health inequalities. Prior to this, Chapter 5 explores whether there are ethnic differences in the cost-effectiveness of mass and targeted screening for high cardiovascular risk. However, these analyses involve the use of area deprivation measures for

targeting deprived individuals. Therefore, given the evidence that suggests that area deprivation measures may not be equally applicable across ethnic groups, Chapter 4 first investigates whether there are ethnic differences in the utility of area deprivation measures to target individual socioeconomic deprivation.

## **4 Chapter 4: Cross-sectional study of ethnic differences in the utility of area deprivation measures to target socioeconomically deprived individuals**

### **4.1 Introduction**

This chapter forms the first of three sections of analysis in this thesis, each of which addresses a separate, though related, research question. The purpose of this introduction is to briefly reiterate a number of issues raised in Chapters 2 and 3 in order to explain why the research question was investigated.

Socioeconomic position is a well-established determinant of health and health inequalities, whereby lower individual socioeconomic position is associated with poorer health and, specifically, increased risk of CVD (29, 64, 180, 482). This means that targeting public health interventions at socioeconomically deprived individuals has the potential to both improve overall health and reduce health inequalities. However, it would be resource intensive and rather impractical to measure socioeconomic position at an individual level across the whole population for this purpose. Therefore, as discussed in Chapter 2, area deprivation measures are often used to identify those geographical areas in which socioeconomically deprived individuals are more likely to live (181, 182, 483). These measures have the advantages of being accessible and of including multiple aspects of deprivation (182, 484), through the aggregation of a variety of indicators from small areas (175). However, they are subject to the “ecological fallacy” and may misclassify the socioeconomic position of individuals (178, 181). Nonetheless, area deprivation measures may act as an effective proxy of individual socioeconomic deprivation and therefore as a useful tool for targeting interventions if the proportion of deprived individuals living in deprived areas is sufficiently high and the proportion of non-deprived individuals living in these areas is sufficiently low.

Ethnic minority groups in the UK experience higher levels of individual socioeconomic deprivation and higher proportions live in deprived areas compared to the general population (238, 303, 308, 451). This coincides with higher risk of associated diseases, including CVD, than the white population (8, 9, 315). Chapter 3 outlined a number of reasons why area deprivation measures,

which are derived from a majority white population, may not be equally applicable across ethnic minority groups. These included evidence of ethnic differences in the misclassification of individual socioeconomic position by area deprivation measures (479), the questionable ability of area deprivation measures to reflect the extent of socioeconomic deprivation in ethnic minority groups (307), and potential ethnic differences in reasons for remaining in or moving from deprived areas (480). Given these limitations it is plausible that area deprivation measures may not work equally well in ethnic minority groups as a tool for targeting socioeconomically deprived individuals. However, there is a lack of evidence as to whether this is the case. Previous studies from the UK on the effectiveness of area deprivation measures in targeting socioeconomically deprived individuals were based on the general population (182, 485), and the evidence cited of ethnic differences in misclassification comes from the US (479), which is substantially different from the UK in terms of ethnic and socioeconomic characteristics.

This chapter therefore addresses the following research question:

Are there ethnic differences in the utility of area deprivation measures to target socioeconomically deprived individuals?

This question is divided into three parts. First, are there ethnic differences in the extent to which area deprivation measures agree with individual socioeconomic measures? Second, are there ethnic differences in the proportion of socioeconomically deprived individuals that are correctly identified by area deprivation measures? Third, are there ethnic differences in the extent to which people without individual socioeconomic deprivation are inappropriately included using area deprivation measures? It is worth emphasising that the intention of this analysis is to explore the practical use of area deprivation measures in the context of interventions aimed at the deprived general population rather than in interventions that specifically target ethnic minority groups.

## 4.2 Methods

### 4.2.1 Data

Cross-sectional data from the Health Survey for England (HSE) 2004 were used (486). The HSE is a large, annual survey that collects data on common health conditions and factors that influence health (487). These data are obtained from nationally representative samples and allow comparisons between population subgroups, monitoring of trends, and calculation of prevalence estimates (487). Data from the HSE were used throughout this thesis so will be described in further detail here. Subsequent chapters add further information as relevant. Both adults and children are included in the HSE but as this thesis only included adults aspects of the survey that are only relevant to children are not discussed. Unless otherwise specified the information in this sub-section comes from the HSE 2004 survey documentation (488, 489).

A range of alternatives data sources were considered, including the Census and surveys such as Understanding Society. The Individual Controlled Access Microdata Sample from the 2001 Census includes IMD 2004 data alongside various individual socioeconomic measures, excluding income (490). Another potential source of data was Understanding Society, otherwise known as the UK Household Longitudinal Study (491). Whilst longitudinal data were not required for these analyses, this survey also provides socioeconomic data from a boosted sample of ethnic minority individuals. However, neither of these datasets contained the full range of data that were required for this thesis, in particular measures of cardiovascular risk factors, which are included in the HSE. However, carrying out similar analyses on these alternatives datasets, in particular on Census data, could strengthen the findings of this chapter's analyses.

#### 4.2.1.1 Design of HSE 2004

The HSE 2004 is a cross-sectional survey that used a "clustered, stratified multi-stage sample design" (p34) (489). The HSE 2004 was used in preference to more recent years as it contained a boosted sample of people from the largest ethnic minority groups in England - black Caribbean, black African, Indian, Pakistani, Bangladeshi, Chinese and Irish. It therefore contained much higher numbers of participants from ethnic minority groups than are usually present in HSE

samples. In addition to the boosted sample data were also collected from a core sample of the general population.

The HSE 2004 received ethical approval from the London Multi-centre Research Ethics Committee.

#### **4.2.1.2 Sampling**

The HSE 2004 used multi-stage stratified probability sampling. For the core sample 312 census wards were randomly selected from a list of wards ordered by geographical location and an area level measure of socioeconomic position based on occupation (proportion of households with a non-manual head of household). Each ward was then split into two with one-half randomly selected as the primary sampling unit. The Postcode Address File was used as the sampling frame to randomly select 21 addresses from each primary sampling unit. This meant that 6,552 addresses were selected. In addresses with multiple households up to 3 households were selected, and in households with multiple residents up to 10 adults and 2 children were randomly selected.

Broadly speaking the sampling approach for the boost sample was similar, although there were differences in how areas were stratified and focused enumeration was used in some areas. The boost sample was designed so that additional black Caribbean, black African, Indian, Pakistani, Bangladeshi, Chinese and Irish participants would be included. Participants of mixed ethnicity from these ethnic groups were also included. Census wards were stratified by various definitions of ethnic density into 13 groups using 2001 Census estimates. 408 wards were then selected and divided into primary sampling units as in the core sample. Between 40 and 115 addresses were then selected from the Postcode Address File for each primary sampling unit depending on its stratum. Focused enumeration was used in areas with the lowest density of people from Asian and black backgrounds (2-10% of residents) - 80 "seed" addresses were selected at which individuals were asked about their own eligibility for inclusion (i.e. belonging to a targeted ethnic minority group) and about addresses adjacent to their household. This approach was not used to identify Irish participants as it relied on visual assessment of ethnicity; Irish participants were only sampled from the original "seed" addresses in these areas. Up to 4 adults and 3 children from the specified ethnic minority groups were eligible for inclusion from each household. Additional sampling was used to obtain Chinese

participants. In this case the electoral register was used to identify areas with residents with “Chinese sounding” names.

The design of the HSE is such that only individuals living in private households are eligible for inclusion. People living in institutions, such as care homes or prisons are excluded. As discussed in the HSE documentation, this may have an impact on the assessment of ethnic inequalities in health. This is because ethnic minority groups are generally younger and may have different caring arrangements for those in need, so excluding people living in institutions may have a lower impact on estimates of health in these groups.

#### **4.2.1.3 Data collection**

Data were collected in two stages. First, computer-assisted interviews were carried out with participants at their home. The interview covered topics such as general health, health behaviours, socioeconomic position and ethnicity. Height and weight were measured by the interviewer. Participants from ethnic minority groups were also asked about CVD. All participants in the ethnic minority groups listed above were then invited to participate in a nurse visit. At the nurse visit information was collected on prescribed medication, and physical measurements were taken such as a blood sample and blood pressure. White participants were not asked about CVD nor invited to participate in a nurse visit.

Steps were taken to ensure that people who did not speak English could take part in the survey. Survey materials were available in seven languages (Urdu, Punjabi, Gujarati, Hindi, Bengali, Mandarin and Cantonese). Whenever possible, interviews and nurse visits were carried out in the participant’s own language.

#### **4.2.1.4 Survey response**

Survey response was calculated at both a household and individual level. In 2004, 72% of households eligible for the core sample participated in the survey, compared to 69% of households eligible for the boost sample. In the core sample 6,704 adults were interviewed. This included 876 adults from ethnic minority groups. These individuals were invited for a nurse visit in the same way as participants in the boost sample and were combined with the boost sample for calculation of response rates. In the boost sample 5,940 adults were interviewed but lower numbers agreed to a nurse visit or had a blood sample taken (3,540 and 2,325 respectively).

The HSE calculates individual survey response by estimating the total number of adults in eligible households for use as the denominator as no data are available on non-responders to the survey interview. Using this approach the individual interview response rate for adults was 66% in the general population core sample and 63% in the boost sample. However, response rates varied geographically and by type of dwelling, and were lower in men and younger adults. Response rates also varied by ethnicity (see Table 4-1).

**Table 4-1: Estimated response to HSE 2004 interview by ethnic group and sex (489)**

Ethnic minority group	Estimated individual response to survey interview (%)	
	Men	Women
Black Caribbean	57	64
Black African	62	64
Indian	60	65
Pakistani	57	63
Bangladeshi	66	71
Chinese	55	57
Irish	64	70

#### **4.2.1.5 Weights**

Survey weights are used to ensure that samples are representative of their intended population by addressing issues such as selection and non-response bias that would make the sample unrepresentative (492). The HSE uses weights to address differences in the probability of being selected for or of responding to the survey. In 2004, slightly different weights were used for the core and boost samples, reflecting the differences in sampling between these groups. For the core sample, household and individual weights were derived that addressed selection and non-response bias. Demographic and geographical information was used to calibrate weights for household non-response. In the boost sample different weights were used in areas in which focused enumeration was used and selection weights were used to account for the fact that probability of selection depended on ethnicity. Weighting variables were also calculated to ensure the sample was consistent with estimated national populations of each ethnic group. This process resulted in three weighting variables that could be applied - an interview weight plus nurse visit and blood sample weights. The interview weight was applied in this analysis.

#### 4.2.2 Participants

Adults aged 16-64 years old from four ethnic groups (black Caribbean, Indian, Pakistani and white) were included in these analyses.

#### 4.2.3 Variables

Ethnicity was self-reported, based on questions about cultural background. It is recommended that research that includes ethnicity as a variable reports how this information was collected and categorised (493). In the HSE participants were asked whether they considered themselves to belong to one of the following groups - white, mixed, black, black British, Asian, Asian British or other (489). Depending on their response they were then asked to further specify their cultural background as Caribbean, African, Indian, Pakistani, Bangladeshi, Chinese and so on. The exception to this approach was in the definition of the Irish group. This was done based on country of birth or parental origin (488). The survey design meant that participants reporting mixed ethnicity were assigned to an ethnic minority group rather than being classified as having mixed ethnicity. The categories used corresponded to those in the 2001 Census (see Box 3-1) (488).

The HSE measures area deprivation using the Index of Multiple Deprivation (IMD) 2004. The IMD was discussed in Chapters 2 and 3, including some of the limitations of its derivation and application, and its use in identifying and targeting deprived areas. Briefly, IMD is a composite measure of multiple aspects of deprivation in which individual level data on seven domains of deprivation (income; employment; health deprivation and disability; education, skills and training; barriers to housing and services; crime; and living environment) are aggregated for Super Output Areas (small areas of approximately 1,500 residents) (175). The domains are combined using weights, with the highest weights given to the income and employment domains. All Super Output Areas in England are then ranked by increasing area deprivation and grouped into quintiles. Each household in the HSE 2004 was assigned to an IMD 2004 quintile based on its postcode (494). The purpose of the analyses was to assess the utility of area deprivation as a tool for targeting interventions. This type of approach would require areas to be selected based on a cut-off of area deprivation so the IMD 2004 quintiles were then divided into two groups - more deprived (quintile 5) and less deprived (quintiles 1-4).

Individual socioeconomic position was measured using self-reported information on education, occupation, car access, income and housing tenure. As there is no single gold standard measure of individual socioeconomic position multiple measures were selected to represent a range of socioeconomic circumstances. Each of these measures has previously been used in the study of the association between socioeconomic position and health (28, 29, 180, 326). Education was based on highest qualification achieved. Housing tenure was based on the circumstances by which the household occupied their current accommodation. Occupation was categorised using the UK's National Statistics Socio-economic Classification (NSSEC) for the household reference person (the householder with the highest income, or the oldest householder in the case of equal incomes). The NSSEC has been recommended as a replacement for older occupational classifications, such as social class or socioeconomic group, as it incorporates broader aspects of employment status and better reflects women's employment status (495). Education, housing tenure and occupation had multiple categories and were dichotomised (see Figure 4-1, which also includes categories used in the sensitivity analysis (see section 4.2.4.3)). Car access was based on a yes or no response to whether a car or van was normally available for use by the respondent or their household. Income was based on equivalised annual income, a measure of total household income that accounts for the number of people living in the household (488). Income quintiles were calculated based on the whole sample, and converted into a binary variable of lower income (quintile 5) and higher income (quintiles 1-4).

Education	None	Foreign / other	NVQ1	NVQ2 / GCSE	NVQ3 / A-level	Higher education	Degree
Narrower definition	Lower	Higher					
Main analysis	Lower			Higher			
Broader definition	Lower				Higher		

Occupation	Never worked and long-term unemployed	Routine	Manual	Intermediate	Managerial and professional
Narrower definition	Lower		Higher		
Main analysis	Lower			Higher	
Broader definition	Lower				Higher

Car access	No	Yes
Narrower definition	No further divisions possible	
Main analysis	Lower	Higher
Broader definition	No further divisions possible	

Income	Quintile 5	Quintile 4	Quintile 3	Quintile 2	Quintile 1
Narrower definition	Lower	Higher			
Main analysis	Lower	Higher			
Broader definition	Lower		Higher		

Housing tenure	Rent free	Rented	Rent and mortgage (shared ownership)	Own through mortgage	Owned outright
Narrower definition	No further divisions possible (insufficient numbers)				
Main analysis	Lower		Higher		
Broader definition	No further divisions possible (insufficient numbers)				

**Figure 4-1: Dichotomisation of individual socioeconomic variables in main analysis and with narrower and broader definitions**

#### 4.2.4 Analyses

##### 4.2.4.1 *Descriptive statistics*

Demographic and socioeconomic characteristics were explored using descriptive statistics. Age was the only continuous variable. The mean and standard deviation were calculated for each ethnic group and an independent-samples t-test was used to compare the age of each ethnic minority group with the white group. The remaining variables were categorical. Proportions were calculated and chi-squared tests used to compare each ethnic minority group with the white group.

##### 4.2.4.2 *Agreement, sensitivity and positive predictive value*

Ethnic differences in the association between area deprivation and individual socioeconomic position were investigated by comparing percentage agreement. Percentage agreement signifies the proportion of individuals in whom the category of area deprivation and individual socioeconomic position matched (see Figure 4-2). Sensitivity and positive predictive value are calculations that are commonly used in epidemiological practice to assess the accuracy and efficiency of screening tests to identify individuals with disease (496). In this analysis sensitivity was used to calculate the proportion of socioeconomically deprived individuals correctly identified by the area deprivation measure, a marker of accuracy. Positive predictive value was used to investigate the extent to which the area deprivation measure inappropriately included people with higher socioeconomic position, a marker of efficiency.

95% confidence intervals for percentage agreement, sensitivity and positive predictive value were calculated using the formula for calculating the confidence intervals of proportions:

$$p \pm 1.96\sqrt{p(1-p)/n}$$

where p is the proportion and n is the sample size.

Agreement between area deprivation and individual socioeconomic position could also have been assessed using alternative statistical techniques, such as correlation. However, the approach described above was selected because it would provide results consistent with how variables are used for targeting interventions, i.e. using thresholds of area deprivation, and because it would provide a measure of efficiency.

Area deprivation	Individual socioeconomic variable		Total
	Lower socioeconomic position	Higher socioeconomic position	
More deprived	a	b	a + b
Less deprived	c	d	c + d
Total	a + c	b + d	a + b + c + d

Percentage agreement	$(a + d) / (a + b + c + d) \times 100$
Sensitivity	$a / (a + c)$
Positive predictive value	$a / (a + b)$

**Figure 4-2: Calculation of percentage agreement, sensitivity and positive predictive value**

#### **4.2.4.3 Sensitivity analysis**

The effect of using different approaches to dichotomise the measures of individual socioeconomic position was investigated in further analyses. Narrower and broader definitions of lower individual socioeconomic position were set and the analysis repeated using each of these definitions (see Figure 4-1). This allowed the robustness of the conclusions from the main analysis to be tested.

#### **4.2.5 Software used**

SPSS 19.0 and Microsoft Excel were used for the analyses.

## **4.3 Results**

### **4.3.1 Demographic and socioeconomic characteristics**

The unweighted sample comprised 7,208 participants, of whom 4,377 (60.7%) were white, 1,070 (14.8%) Indian, 874 (12.2%) Pakistani and 887 (12.3%) black Caribbean (see Table 4-2). All ethnic minority groups were significantly younger than the white group; the lowest mean age was observed in the Pakistani group (mean age 34.6 years compared to 39.9 years in the white group). All ethnic minority groups had a significantly lower proportion of men than the white group, with the lowest proportion in the black Caribbean group.

The prevalence of area deprivation was significantly higher in all ethnic minority groups than in the white group (see Table 4-2). In particular, 52.2% of the Pakistani group and 45.1% of the black Caribbean group lived in the more deprived quintile compared to 14.9% in the white group. Individual socioeconomic position, measured by occupation, car access and income, was significantly lower in all ethnic minority groups than in the white group. The exceptions to this pattern were housing tenure and education. There was no significant difference between the Indian and white group in housing tenure, although significantly higher proportions of the Pakistani and black Caribbean groups lived in rented or rent free housing. There were no significant differences between the Indian or black Caribbean and white groups in education, but a significantly higher proportion of the Pakistani group had lower education than the white group.

More deprived areas had higher proportions of individuals with lower socioeconomic position (see Table 4-3). This association was observed across all ethnic groups and all individual socioeconomic measures. Within less deprived areas, the proportion of individuals who had higher socioeconomic position was generally greater in the white group and lower in the Pakistani and black Caribbean groups. The proportion of individuals with lower socioeconomic position who lived in the more deprived areas was more variable and depended on the individual socioeconomic measure used.

### **4.3.2 Agreement, sensitivity and positive predictive value**

Ethnic differences were observed in agreement and sensitivity but not in positive predictive value (see Table 4-4). Agreement was generally highest in the white

group (ranging from 67.2% to 82.4%), with the exception of education where it was highest in the Indian group. In contrast, agreement was lower in the Pakistani (50.9% to 63.4%) and black Caribbean (61.0% to 70.1%) groups across all individual socioeconomic measures. Intermediate results, closer to the white group than the Pakistani and black Caribbean groups, were observed in the Indian group. Sensitivity was lowest in the white group and highest in the Pakistani and black Caribbean groups across all individual socioeconomic measures. Sensitivity ranged from 0.56 to 0.64 in the Pakistani group and from 0.59 to 0.66 in the black Caribbean group, whereas it ranged from 0.24 to 0.38 in the white group. Similar to the results for agreement, sensitivity results in the Indian group were intermediate and more similar to the white group than to the Pakistani and black Caribbean groups. 95% confidence intervals for sensitivity in the Pakistani and black Caribbean groups did not overlap with those in the white and Indian groups. Positive predictive value was similar across the ethnic groups, with no consistent ethnic differences observed. For occupation, positive predictive value ranged from 0.60 to 0.64 across the ethnic groups; for car access it ranged from 0.22 to 0.56 across the ethnic groups.

#### **4.3.3 Sensitivity analysis**

Calculation of agreement, sensitivity and positive predictive value using narrower and broader definitions of individual socioeconomic position produced results that were generally consistent in direction with the main analysis (see Table 4-5). Sensitivity remained lower in the white and Indian groups than in the Pakistani and black Caribbean groups using both the narrower and broader definitions. Results for positive predictive value were similar across the ethnic groups, again with no consistent ethnic differences observed. Agreement was higher in the white and Indian groups than in the Pakistani and black Caribbean groups using the narrower definition. However, this pattern was less clear using the broader definition, where greater similarity in agreement was observed across the ethnic groups with relatively lower agreement in the white and Indian groups than in the main analysis.

Table 4-2: Characteristics of participants by ethnic group

	White	Indian		Pakistani		Black Caribbean	
Unweighted base	4,377	1,070		874		887	
Weighted base	64,771	1,784		858		973	
	mean (SD) <sup>a</sup>	mean (SD)	<i>p Value</i> <sup>b</sup>	mean (SD)	<i>p Value</i>	mean (SD)	<i>p Value</i>
Age (years)	39.9 (13.8)	38.3 (12.7)	<0.001	34.6 (12.2)	<0.001	38.2 (13.0)	<0.001
Male	n <sup>c</sup> (%) 32,513 (50.2)	n (%) 801 (44.9)	<0.001	n (%) 386 (45.0)	0.002	n (%) 391 (40.2)	<0.001
Area deprivation							
Quintiles 1-4	55,138 (85.1)	1,428 (80.0)	<0.001	410 (47.8)	<0.001	534 (54.9)	<0.001
Quintile 5 <sup>d</sup>	9,633 (14.9)	357 (20.0)		448 (52.2)		438 (45.1)	
Missing	0	0		0		0	
Education							
NVQ <sup>e</sup> 2 and above	47,092 (72.9)	1,331 (74.9)	0.062	466 (54.8)	<0.001	718 (74.7)	0.221
NVQ1, other and no qualifications	17,464 (27.1)	445 (25.1)		385 (45.2)		243 (25.3)	
Missing	215	8		7		11	
Occupation							
Managerial, professional and intermediate	40,873 (63.3)	1,032 (58.2)	<0.001	394 (46.8)	<0.001	525 (54.5)	<0.001
Routine, manual and none	23,688 (36.7)	742 (41.8)		447 (53.2)		438 (45.5)	
Missing	210	10		17		10	
Car access							
Access	57,540 (88.8)	1,500 (84.1)	<0.001	704 (82.1)	<0.001	591 (60.7)	<0.001
No access	7,232 (11.2)	284 (15.9)		154 (17.9)		382 (39.3)	
Missing	0	0		0		0	
Income							
Quintiles 1-4	45,650 (81.7)	939 (70.5)	<0.001	264 (42.0)	<0.001	494 (63.6)	<0.001
Quintile 5 <sup>f</sup>	10,231 (18.3)	393 (29.5)		365 (58.0)		283 (36.4)	
Missing	8,891	451		229		197	
Housing tenure							
Owner occupier	49,442 (76.5)	1,380 (77.7)	0.233	593 (69.7)	<0.001	498 (51.6)	<0.001
Rent or rent free	15,162 (23.5)	395 (22.3)		258 (30.3)		467 (48.4)	
Missing	168	9		7		8	

<sup>a</sup> SD standard deviation; <sup>b</sup> *p Value* indicates difference between ethnic minority group and white group; <sup>c</sup> n weighted base; <sup>d</sup> Quintile 5 for area deprivation represents more deprived areas; <sup>e</sup> NVQ National Vocational Qualification; <sup>f</sup> Quintile 5 for income represents lower income

**Table 4-3: Individual socioeconomic position for each area deprivation category by ethnic group**

		White		Indian		Pakistani		Black Caribbean	
		Quintile 5 <sup>a</sup>	Quintiles 1-4	Quintile 5	Quintiles 1-4	Quintile 5	Quintiles 1-4	Quintile 5	Quintiles 1-4
		n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Education	NVQ1 <sup>b</sup> , other and no qualifications	4,213 (44.0)	13,251 (24.1)	161 (45.4)	284 (20.0)	234 (52.3)	151 (37.4)	150 (34.8)	94 (17.7)
	NVQ 2 and above	5,356 (56.0)	41,735 (75.9)	194 (54.6)	1,138 (80.0)	213 (47.7)	253 (62.6)	281 (65.2)	437 (82.3)
Occupation	Routine, manual and none	6,045 (63.3)	17,643 (32.1)	225 (63.7)	517 (36.4)	267 (60.5)	180 (45.0)	258 (60.0)	179 (33.7)
	Managerial, professional and intermediate	3,505 (36.7)	37,369 (67.9)	128 (36.3)	904 (63.6)	174 (39.5)	220 (55.0)	172 (40.0)	352 (66.3)
Car access	No access	2,738 (28.4)	4,493 (8.1)	124 (34.8)	160 (11.2)	99 (22.1)	55 (13.4)	244 (55.6)	138 (25.8)
	Access	6,894 (71.6)	50,645 (91.9)	232 (65.2)	1,268 (88.8)	349 (77.9)	355 (86.6)	195 (44.4)	396 (74.2)
Income	Quintile 5 <sup>c</sup>	3,196 (38.0)	7,035 (14.8)	131 (52.2)	262 (24.2)	235 (70.1)	130 (44.2)	168 (49.4)	115 (26.4)
	Quintiles 1-4	5,219 (62.0)	40,430 (85.2)	120 (47.8)	820 (75.8)	100 (29.9)	164 (55.8)	172 (50.6)	321 (73.6)
Housing tenure	Rented or rent free	4,540 (47.1)	10,622 (19.3)	137 (38.4)	258 (18.2)	144 (32.1)	114 (28.3)	308 (70.5)	159 (30.2)
	Owner occupier	5,093 (52.9)	44,349 (80.7)	220 (61.6)	1,161 (81.8)	304 (67.9)	289 (71.7)	129 (29.5)	368 (69.8)

<sup>a</sup> Quintile 5 for area deprivation represents more deprived areas; <sup>b</sup> NVQ National Vocational Qualification; <sup>c</sup> Quintile 5 for income represents lowest income

**Table 4-4: Results for agreement, sensitivity and positive predictive value calculations for each individual socioeconomic measure by ethnic group**

		White	Indian	Pakistani	Black Caribbean
Education	Agreement (%)	71.2	73.1	57.2	61.0
	95% CI	70.8-71.5	71.0-75.2	53.9-60.6	57.9-64.1
	Sensitivity	0.24	0.36	0.61	0.61
	95% CI	0.23-0.25	0.32-0.41	0.56-0.66	0.55-0.68
	PPV	0.44	0.45	0.52	0.35
	95% CI	0.43-0.45	0.40-0.51	0.48-0.57	0.30-0.39
Occupation	Agreement (%)	67.2	63.7	57.8	63.4
	95% CI	66.9-67.6	61.4-65.9	54.6-61.2	60.4-66.5
	Sensitivity	0.26	0.30	0.60	0.59
	95% CI	0.25-0.26	0.27-0.34	0.55-0.64	0.54-0.64
	PPV	0.63	0.64	0.61	0.60
	95% CI	0.62-0.64	0.59-0.69	0.56-0.65	0.55-0.65
Car access	Agreement (%)	82.4	78.0	52.9	65.8
	95% CI	82.1-82.7	76.1-80.0	49.6-56.3	62.8-68.8
	Sensitivity	0.38	0.44	0.64	0.64
	95% CI	0.37-0.39	0.38-0.49	0.57-0.72	0.59-0.69
	PPV	0.28	0.35	0.22	0.56
	95% CI	0.28-0.29	0.30-0.40	0.18-0.26	0.51-0.60
Income	Agreement (%)	78.1	71.3	63.4	63.0
	95% CI	77.8-78.4	68.9-73.8	59.7-67.2	59.6-66.4
	Sensitivity	0.31	0.33	0.64	0.59
	95% CI	0.30-0.32	0.29-0.38	0.59-0.69	0.54-0.65
	PPV	0.38	0.52	0.70	0.49
	95% CI	0.37-0.39	0.46-0.58	0.65-0.75	0.44-0.55
Housing tenure	Agreement (%)	75.7	73.1	50.9	70.1
	95% CI	75.3-76.0	71.0-75.2	47.5-54.2	67.2-73.0
	Sensitivity	0.30	0.35	0.56	0.66
	95% CI	0.29-0.31	0.30-0.39	0.50-0.62	0.62-0.70
	PPV	0.47	0.38	0.32	0.70
	95% CI	0.46-0.48	0.33-0.43	0.28-0.36	0.66-0.75

CI confidence interval

PPV positive predictive value

**Table 4-5: Results of analyses with narrower and broader definitions of individual socioeconomic position for agreement, sensitivity and positive predictive value by ethnic group**

	White		Indian		Pakistani		Black Caribbean	
	Narrower definition	Broader definition						
<b>Education</b>								
Agreement (%)	75.1	51.4	73.6	61.1	56.6	61.8	60.6	60.5
95% CI	74.8-75.5	51.0-51.8	71.5-75.6	58.9-63.4	53.3-60.0	58.6-65.1	57.5-63.7	57.4-63.6
Sensitivity	0.25	0.18	0.35	0.29	0.61	0.62	0.63	0.55
95% CI	0.24-0.25	0.18-0.19	0.30-0.40	0.26-0.32	0.56-0.67	0.57-0.66	0.57-0.70	0.51-0.59
PPV	0.33	0.66	0.37	0.64	0.47	0.72	0.29	0.63
95% CI	0.32-0.34	0.66-0.67	0.32-0.42	0.59-0.69	0.42-0.52	0.68-0.77	0.24-0.33	0.59-0.68
<b>Occupation</b>								
Agreement (%)	80.1	52.4	74.9	49.7	54.7	57.3	61.9	61.9
95% CI	79.8-80.4	52.0-52.8	72.8-76.9	47.4-52.0	51.3-58.1	54.0-60.7	58.8-65.0	58.8-65.0
Sensitivity	0.30	0.21	0.35	0.25	0.62	0.56	0.67	0.55
95% CI	0.29-0.31	0.20-0.21	0.30-0.40	0.23-0.28	0.56-0.68	0.52-0.60	0.60-0.74	0.51-0.59
PPV	0.27	0.78	0.30	0.80	0.35	0.88	0.29	0.79
95% CI	0.26-0.28	0.77-0.79	0.26-0.35	0.76-0.84	0.30-0.39	0.85-0.91	0.25-0.33	0.75-0.83
<b>Income</b>								
Agreement (%)	82.6	67.0	77.2	55.1	55.5	61.6	61.0	64.9
95% CI	82.3-82.9	66.6-67.4	74.9-79.5	52.4-57.7	51.6-59.4	57.8-65.4	57.6-64.4	61.6-68.3
Sensitivity	0.36	0.26	0.38	0.27	0.63	0.59	0.60	0.57
95% CI	0.35-0.38	0.26-0.27	0.32-0.45	0.24-0.30	0.57-0.70	0.55-0.63	0.53-0.66	0.53-0.62
PPV	0.21	0.67	0.35	0.82	0.39	0.90	0.35	0.77
95% CI	0.20-0.22	0.66-0.68	0.29-0.41	0.77-0.86	0.34-0.45	0.87-0.93	0.30-0.40	0.73-0.82

CI confidence interval

PPV positive predictive value

Narrower definition of lower socioeconomic position is educational level of no qualifications, occupation of routine or no employment, and income in the lowest decile. Broader definition of lower socioeconomic position is educational level of NVQ2 level and below, occupation of intermediate, routine, manual or no employment, and income in quintiles 4 and 5.

## **4.4 Discussion**

### **4.4.1 Principal findings**

These analyses identified ethnic differences in the performance of area deprivation as a tool for targeting socioeconomically deprived individuals. Despite lower agreement between area and individual measures of socioeconomic position in the Pakistani and black Caribbean groups, sensitivity was consistently higher compared to the white group. In the Indian group results for both agreement and sensitivity were intermediate but most similar to those in the white group. There were no consistent ethnic differences in positive predictive value and, in particular, positive predictive value was no worse in the ethnic minority groups.

If area deprivation measures were ineffective at identifying deprived individuals in ethnic minority groups, then at-risk individuals could be missed and ethnic health inequalities could increase. Conversely, if area deprivation measures performed more effectively in ethnic minority groups then interventions targeted on the basis of area deprivation would tend to reduce ethnic health inequalities. If area deprivation measures were inefficient because they identified higher numbers of non-deprived individuals in ethnic minority groups this would reduce the value for money and increase the opportunity costs of interventions targeted at deprived areas.

In this analysis area deprivation correctly identified higher proportions of socioeconomically deprived Pakistani and black Caribbean individuals than white or Indian individuals, whilst at the same time performing comparably across the ethnic groups at excluding individuals with higher socioeconomic position. Compared to the white population, in the context of an area based intervention this would lead to increased coverage of deprived Pakistani and black Caribbean populations, and comparable coverage of deprived Indian populations, without changing the efficiency of the targeted intervention.

### **4.4.2 Strengths and limitations**

The HSE 2004 provided cross-sectional data from a boosted sample of ethnic minority participants. The boosted sample provided sufficient numbers of participants from the largest ethnic groups in England. Indeed, the ethnic

minority sample sizes in this survey are greater than those in many other general population surveys, and certainly in subsequent years of the HSE. Individuals from the four largest ethnic groups in England were included in the analyses in this chapter and the related publication (497). Extending the analyses to other ethnic groups would provide further information to inform the use of area deprivation measures for targeting socioeconomically deprived individuals and it is possible that the findings may vary in other ethnic groups, for example lower levels of socioeconomic deprivation could mean that area deprivation measures perform comparably in Chinese and white groups. Survey response rates varied by ethnicity and sex leading to a potential for response bias if those who did not respond were systematically different from those who did, although a lack of information on non-responders makes the extent and direction of this potential bias difficult to assess. HSE weighting variables that account for selection and non-response bias were applied.

The HSE had the advantage of including a broad range of individual socioeconomic measures, including income, a variable that is not available in alternative data sources such as the Census (see section 4.2.1). This enabled the performance of the IMD 2004 to be tested against a variety of individual socioeconomic measures, thereby strengthening the findings through the observation of consistent results across the range of measures. All of the individual socioeconomic measures were based on self-reported information, raising the possibility of reporting or recall bias. An alternative approach could have been for socioeconomic position to be measured using objective information, such as tax or housing records. However, this would be impractical approach that could potentially put individuals off participating in the survey. Bias is arguably most likely to have affected the measurement of income. There was a high proportion of missing data on income, which varied between ethnic groups (ranging from 13.7% in the white group to 26.7% in the Pakistani group). Bias may have been introduced if this non-response was also related to income. One option for addressing the missing data would have been to use a statistical technique such as multiple imputation. However, given that the results for income were consistent with those from the other individual socioeconomic measures studied, where levels of missing data were much lower, it was decided not to carry out multiple imputation at this stage.

Area and individual socio-economic measures were dichotomised. An alternative approach to using dichotomised variables could have been to analyse the socioeconomic variables using their full range of categories, or continuously in the case of income, and to apply different statistical techniques, such as correlation. This alternative approach could have added additional information on possible gradients in the agreement between individual and area based measures of socioeconomic deprivation. Although the conversion into binary variables may lose some of this information, it was appropriate for these analyses as it reflects the design and practical delivery of many public health interventions, where populations are dichotomised into those who are included or excluded from the intervention based on a predetermined threshold (e.g. the most deprived 20% of areas). However, the cut-offs chosen may be seen as somewhat arbitrary and may not reflect an individual's actual experienced socioeconomic position. Indeed, the cut-offs used to define lower individual socioeconomic position may not equate to the cut-off used to define greater area deprivation. This may be particularly relevant for individuals from ethnic minority groups for whom certain categories of socioeconomic position, such as having a higher education, may not translate into equally advantageous socioeconomic circumstances as in white individuals. A lack of empirical evidence quantifying this ethnic inequality meant that it could not be included in the analysis. However, the likely impact of considering this inequality would have been to increase the number of ethnic minority individuals with lower individual socioeconomic position, which would not affect the results for sensitivity and could improve them for positive predictive value in these groups. Sensitivity analysis was used to explore narrower and broader definitions of lower socio-economic position and reassuringly the results were broadly consistent with those from the main analysis.

This study focused on the identification of individual socio-economic deprivation. However, as previously discussed in chapter 2, area itself may independently influence health, beyond the impact of the characteristics of individuals living in an area (177, 178). This could occur through health effects of the physical environment or community influence (178). This means that targeting public health interventions at deprived areas can potentially address two separate risk factors since it identifies individuals subject to both area and individual deprivation. However, IMD 2004 is derived from aggregated data on

individuals and includes limited measures of area characteristics (air quality is an exception to this) (175). Therefore, whilst it will identify areas occupied by higher proportions of deprived individuals it may not reflect the full range of area characteristics that can influence health. The implications of this for ethnic health inequalities are unclear, because whilst addressing area characteristics that are detrimental to health will affect all residents regardless of ethnic group, there is evidence of ethnic differences in the association between area and health (see section 3.8).

#### **4.4.3 Relations to other studies**

Previous studies have compared the agreement between area and individual measures of socioeconomic deprivation. Two studies from Canada found low agreement between area and individual measures (190, 191). However, methodological differences prevent direct comparisons with this analysis as these studies calculated agreement in quintiles or deciles plus with correlation coefficients, and did not investigate ethnic differences. In contrast, Diez-Roux *et al* compared the association between area and individual measures in black and white individuals in the USA (479). They also found ethnic differences in agreement between area and individual measures of income and education, accounting for this by the higher proportion of black people living in deprived areas irrespective of their socioeconomic position. Despite differences between the two countries this may reflect similarities in the relationships between ethnicity, socioeconomic deprivation and area of residence in the USA and UK.

Targeting public health interventions at deprived areas can be an efficient way of identifying socioeconomically deprived individuals and focusing limited resources on people with the greatest need (181). The geographical clustering of socioeconomic deprivation in the UK and availability of area deprivation measures make this approach feasible (175, 181). Tunstall and Lupton investigated the effectiveness of area deprivation measures in targeting deprived individuals in the general population (182). They calculated two measures, completeness and efficiency, that correspond with the measures of sensitivity and positive predictive value used in this analysis. Their analysis included various definitions of areas to be targeted and they found that approximately 54-64% of employment benefits claimants would be identified by targeting these areas, with 13-17% of adults in these areas being benefits

claimants. The former is equivalent to sensitivity in this analysis but the results were higher than those observed here for the white group; the latter is equivalent to positive predictive value with lower results compared to this analysis. These differences could be accounted for by variations in the definitions of deprived areas and of individual socioeconomic position between the studies. For instance, Tunstall and Lupton used a narrower definition of lower individual socioeconomic position (Job Seeker's Allowance and Income Support claimants) compared to this analysis. Results from the narrower definition explored here in the sensitivity analysis gave lower positive predictive values that correspond more closely with those in Tunstall and Lupton's analysis. In a similar analysis, Batey and Brown noted high levels of inefficiency and incompleteness in the area targeting used in the Sure Start programme (485). Whilst these studies did not investigate ethnic differences, Tunstall and Lupton suggested that spatial patterning of population sub-groups could affect the performance of area deprivation measures as tools for targeting deprived individuals (182). This conclusion would fit with the findings of this analysis given that ethnic minority groups are known to cluster in deprived areas (309, 451, 452, 464). In fact, geographical clustering of ethnic minority groups into deprived areas alongside higher levels of socioeconomic deprivation than in the white population are likely to account for the ethnic differences observed in this analysis.

A key criticism of the use of area deprivation measures to target interventions is the fact that the majority of deprived people do not live in deprived areas (181, 190). This example of the ecological fallacy is well established (178), and is consistent with findings from this analysis that only 24-38% of deprived white individuals lived in deprived areas, although the results indicate that this key criticism may not apply to Pakistani and black Caribbean individuals. Despite this fundamental drawback and previous conclusions that area deprivation measures are unlikely to ever give high completeness and efficiency (182), area based initiatives have been widely adopted in the UK. These initiatives include the New Deal for Communities initiative and Scotland's Keep Well CVD prevention programme (14, 189). Evidence for the effectiveness of these programmes is limited (188), though emerging (498). However, given the drawbacks of area deprivation measures, interventions to tackle inequalities may need to adopt wider measures beyond targeting areas alone.

#### **4.4.4 Implications**

This analysis indicates that area deprivation measures perform relatively well in certain ethnic minority groups compared to the white population as a tool for targeting deprived individuals, in that higher proportions of deprived individuals from ethnic minority groups would be identified without higher inadvertent inclusion of non-deprived individuals. This finding is particularly relevant to CVD prevention programmes. It has been suggested that targeting CVD prevention interventions at deprived areas may be an acceptable and cost-effective alternative to mass coverage (192, 443), although this conclusion is based on evidence from the general population and it is unclear as yet whether it applies to ethnic minority groups (the next chapter addresses this question). Given the increased risk of CVD and of socioeconomic deprivation in many ethnic minority groups in the UK, it would be particularly important for CVD prevention programmes to achieve good coverage of deprived ethnic minority populations in such a way that did not waste limited resources. These results therefore provide reassuring evidence that the use of area deprivation measures for targeting socioeconomically deprived individuals may be appropriate for ethnic minority populations, with no evidence to suggest that these groups would be systematically disadvantaged. Rather, the evidence suggests systematic advantage in these groups with regards to targeting, which could potentially reduce ethnic health inequalities.

These findings may be generalisable to other settings that have ethnic minority populations with similar socioeconomic characteristics. However, this would need to be done with caution given likely differences in ethnic demographics and geographical distribution of socioeconomic deprivation and ethnic minority populations.

#### **4.4.5 Next steps**

A number of questions regarding the use of area deprivation as a means of targeting public health interventions remain unanswered, including whether there are ethnic differences in the potential for area deprivation to identify individuals at high risk of disease. Therefore, the next stage in this thesis is to investigate ethnic differences in the cost-effectiveness of area based targeting of cardiovascular risk screening.

## **5 Chapter 5: Ethnic differences in the cost-effectiveness of targeted and mass screening for high cardiovascular risk in the UK**

### **5.1 Introduction**

Having established in the previous chapter that the use of area deprivation measures may be appropriate for identifying deprived individuals from ethnic minority groups, this chapter builds on that work by investigating ethnic differences in the cost-effectiveness of targeted screening for high cardiovascular risk in deprived areas. Chapters 2 and 3 described a number of key issues that are pertinent to this chapter; these are summarised here in order to explain why this particular analysis was carried out.

Primary prevention, through risk factor modification, is key to successfully reducing the burden of CVD, the leading cause of premature mortality in the UK (6, 53, 80). Current UK guidance recommends that individuals with an estimated cardiovascular risk score  $\geq 20\%$  are offered interventions, such as statins, to reduce their risk (although at the time of writing this threshold is set to change to  $\geq 10\%$ ) (121, 499, 500). Previously the choice of cardiovascular risk calculator was left to the practitioner's discretion (121), however this looks set to change to a recommendation in England to use QRISK2 (501), a calculator that may be particularly suitable for ethnic minority populations (121, 438, 444). As described in Chapter 2, mass or targeted screening can be used to identify individuals with high cardiovascular risk. Whilst mass screening has the potential to identify all high-risk individuals in the population, targeting screening at deprived areas might be a more cost-effective strategy that identifies the majority (192). Current policy varies across the UK, with mass screening through NHS Health Checks in England (although local implementation means there is variation in programme delivery, including additional targeting of higher risk population sub-groups in some areas (502)) and targeted screening of the most deprived areas through the Keep Well programme in Scotland (14, 159).

Public health interventions have the potential to both improve overall health but may paradoxically widen health inequalities if people with better baseline health benefit more from the intervention than those with worse health (4,

5, 199). The UK's population is becoming increasingly ethnically diverse (19), with many of the largest ethnic minority groups at increased risk of CVD compared to the white population (12, 239, 250-252, 259, 260). This means that it is particularly important to assess the effectiveness of public health interventions, including CVD prevention programmes, in different ethnic groups. This is to ensure that these interventions work equally, if not more, effectively in these groups, in order to reduce health inequalities as well as improve overall health.

Against a background of limited financial resources and the need to tackle health inequalities, the relative merits of mass and targeted CVD prevention policies continue to be discussed (159, 169). However, it is unclear what the impact of a choice between screening the whole population or focusing on deprived communities would be on ethnic minority populations. Ethnic minority groups, particularly black Caribbean, Pakistani and Bangladeshi groups, are more likely to live in deprived areas than the white population (308, 309, 451, 452, 457). Therefore, it is likely that there will be ethnic differences in the cost-effectiveness of targeted screening, and how this compares with mass screening.

This chapter therefore addresses the following research question:

Are there ethnic differences in the cost-effectiveness of targeted and mass screening for high cardiovascular risk?

Similar to the previous chapter, the intention of these analyses was to explore the potential impact of policies designed for the general population rather than to suggest specific screening strategies for different ethnic groups.

## **5.2 Methods**

### **5.2.1 Data**

This analysis used data from the HSE 2003 and 2004 (486, 503). Details of the HSE 2004 were outlined in the previous chapter. The HSE 2004 was used for analysis of ethnic minority groups as it contained a boosted sample of the ethnic minority population in England. Whilst white individuals were included in the core sample of the HSE 2004 the design of the survey meant that only ethnic minority participants received a nurse visit at which physical measurements, such as cholesterol and blood pressure, were taken. Therefore, the HSE 2003 was used for analysis of the white group and general population.

#### **5.2.1.1 HSE 2003**

The HSE 2003 was similar to the 2004 survey in terms of its design, sampling and use of weights (504). The focus of the 2003 survey was CVD and cardiovascular risk factors in the general population; it did not include a boosted sample of any particular population sub-group. The 2003 survey therefore predominantly included white participants, although there were small numbers of ethnic minority individuals who are included in the general population group. Like the 2004 survey, ethnicity was self-reported from a question on broad ethnic grouping with a follow up question on cultural background.

In 2003, a similar stratified multistage probability sampling approach was used to that in the 2004 core sample. The primary sampling unit was postcode sectors of which 720 were selected after stratification by geography and socioeconomic position. The Postcode Address File was then used to select 13,680 addresses from these postcode sectors.

Response rates were estimated in a similar way to the 2004 survey. In 2003, 14,836 adults were interviewed, of whom 11,408 received a nurse visit and 8,552 gave a blood sample. Using estimated denominators the HSE documentation provides the following adult response rates - 66% interviewed, 51% received a nurse visit, 50% had blood pressure taken, and 38% gave a blood sample (504). The corresponding estimates from the 2004 ethnic boost sample were 63% interviewed, 36% received a nurse visit, 32% had blood pressure taken and 21% gave a blood sample (489).

The HSE 2003 included household and individual nonresponse weights. Logistic regression was used to calculate the probability of response for individuals, with the model including variables such as age, sex, geography, household type and social class. Three individual weights were then calculated to correspond to response to the interview, nurse visit or blood sample.

Ethical approval for the HSE 2003 was obtained from the London Multi-centre research ethics committee.

#### **5.2.1.2 Use of the HSE 2003 and 2004**

The HSE 2003 and 2004 were analysed separately and then the results compared. These analyses used data collected from the interview, nurse visit and blood sample components of the surveys. Whilst separate weights are available for these sections in both 2003 and 2004 the interview weights were applied in these analyses.

#### **5.2.2 Participants**

Adults aged 40-74 years without CVD were included. The ethnic groups included were black Caribbean, Indian, Pakistani/Bangladeshi, Irish and white. The Pakistani and Bangladeshi groups were initially analysed separately but then merged in the final analysis because of small numbers. The black African and Chinese groups were excluded because the sample sizes in these groups were considered to be too small for the analyses (n = 291 and 313, respectively).

#### **5.2.3 Variables**

Ethnicity was self-reported and based on 2001 Census categories, as previously described (see sections 4.2.3 and 5.2.1.1). CVD was defined as self-reported diagnosis of angina, myocardial infarction or stroke. This definition did not include conditions such as heart murmur, irregular heart rhythm or high blood pressure. Area deprivation was measured using IMD 2004 quintiles (see section 4.2.3).

QRISK2-2012 was used to estimate each individual's cardiovascular risk score. QRISK2-2012 was selected as it includes ethnicity and area deprivation as independent risk variables and may be more accurate in ethnic minority groups in the UK than alternative risk calculators (438, 444). QRISK2-2012 contains a broader range of variables than alternative cardiovascular risk calculators such as Framingham and Ethrisk (see Table 5-1). HSE variables on age, sex, ethnicity,

family history of CVD, systolic blood pressure, total and HDL cholesterol, body mass index, treated hypertension, self-reported diabetes and self-reported smoking status were used. QRISK2-2012 defines family history of CHD as ischaemic heart disease in a first degree relative less than 60 years old, whereas the HSE measures it as death of a parent less than 65 years old from CVD, hypertension or diabetes. Smoking status was split into five categories in the QRISK2-2012 calculation (non-smoker, former smoker, light smoker, moderate smoker and heavy smoker) so HSE variables on current smoking and amount smoked was recoded to fit these categories. QRISK2-2012 only considers type 2 diabetes so the small number of individuals with type 1 diabetes were included in the same category as non-diabetics.

**Table 5-1: Summary of variables in QRISK2-2012, Framingham and Ethrisk cardiovascular risk calculators**

Variable	Cardiovascular risk calculator		
	QRISK2-2012 (438)	Framingham (149)	Ethrisk (445)
Age	✓	✓	✓
Sex	✓	✓	✓
Ethnicity	✓	X	✓
Smoking	✓	✓	✓
Systolic blood pressure	✓	✓	✓
Diastolic blood pressure	X	✓	X
Total: HDL cholesterol ratio	✓	✓	✓
Body mass index	✓	X	X
Family history of CHD	✓	X	X
Socioeconomic deprivation	✓	X	X
Treated hypertension	✓	X	X
Type 2 diabetes	✓	✓	X
Atrial fibrillation	✓	X	X
Chronic kidney disease	✓	X	X
Rheumatoid arthritis	✓	X	X

### 5.2.3.1 Estimation of variables for QRISK2

#### 5.2.3.1.1 Atrial fibrillation, rheumatoid arthritis and chronic kidney disease

The HSE 2003 and 2004 do not include data on atrial fibrillation, rheumatoid arthritis and chronic kidney disease. The prevalence of these conditions is generally low and previous studies have followed an approach suggested by QRISK2 of assuming that these conditions are absent when data are not available (437, 505). However, exploratory analysis indicated that these conditions had a large effect on the cardiovascular risk score when present. In addition, whilst overall prevalence is low it increases with age (e.g. approximately 17% of women aged 70-74 have chronic kidney disease). Therefore, a microsimulation approach was used to apply prevalence estimates to the HSE samples. These variables could not be imputed (see section 5.2.4.2) as they were absent rather than containing missing data. Age, sex and IMD 2007 specific prevalence estimates for atrial fibrillation and chronic kidney disease were obtained from The Health Improvement Network database (THIN), a large primary care database that covers 6.2% of the UK population (see Table 5-2 and Table 5-3) (506, 507). Similar data were not available for rheumatoid arthritis so age and sex specific data from a prevalence survey were used (see Table 5-4) (508). These data indicated the important of age in the prevalence of these conditions, so although ethnic group specific data were identified these data were not used

as they did not include age (438). A random number simulation approach, based on Monte Carlo simulation, was used to apply these prevalence estimates to the sample. Stata's random number function was used to generate random numbers between zero and one. On the basis of these numbers individuals were designated to a binary variable of presence or absence of each condition depending on their age and sex, and also their IMD 2004 quintile for atrial fibrillation and chronic kidney disease. The impact of this approach on the results was assessed by repeating the analyses (described in section 5.2.5.3) with the prevalence of these conditions assumed to be zero.

**Table 5-2: Estimated prevalence (%) of atrial fibrillation by age, sex and IMD 2007 quintile (507)**

Sex	Age group (years)	IMD 2007 quintile				
		1 - least deprived	2	3	4	5 - most deprived
Men	40-44	0.37	0.34	0.24	0.32	0.25
	45-49	0.45	0.52	0.54	0.41	0.36
	50-54	0.83	0.76	0.84	0.88	0.77
	55-59	1.23	1.42	1.22	1.30	1.46
	60-64	2.22	2.50	2.55	2.64	2.52
	65-69	4.11	4.25	3.96	3.79	4.24
	70-74	6.81	6.55	6.70	6.56	5.94
Women	40-44	0.06	0.06	0.12	0.09	0.07
	45-49	0.14	0.11	0.13	0.13	0.12
	50-54	0.24	0.30	0.29	0.32	0.33
	55-59	0.51	0.51	0.58	0.59	0.61
	60-64	0.94	1.08	1.09	1.06	1.24
	65-69	1.93	1.93	1.94	2.23	2.50
	70-74	3.51	4.00	4.34	4.00	4.71

**Table 5-3: Estimated prevalence (%) of chronic kidney disease by age, sex and IMD 2007 quintile (507)**

Sex	Age group (years)	IMD 2007 quintile				
		1 - least deprived	2	3	4	5 - most deprived
Men	40-44	0.20	0.20	0.24	0.33	0.42
	45-49	0.40	0.42	0.46	0.46	0.62
	50-54	0.73	0.66	0.78	1.11	1.07
	55-59	1.56	1.50	1.58	1.84	2.03
	60-64	3.03	3.06	3.08	3.65	3.68
	65-69	5.67	6.08	6.08	7.22	7.74
	70-74	10.77	11.44	11.48	11.76	13.51
Women	40-44	0.31	0.28	0.39	0.44	0.47
	45-49	0.55	0.55	0.70	0.99	1.07
	50-54	1.13	1.23	1.30	1.69	1.83
	55-59	2.35	2.35	2.47	3.12	3.24
	60-64	4.09	4.54	4.65	5.49	6.58
	65-69	7.41	8.13	8.68	9.50	11.06
	70-74	12.10	13.40	13.95	15.67	17.35

**Table 5-4: Estimated prevalence (%) of rheumatoid arthritis by age and sex (508)**

Sex	Age group (years)		
	16-44	45-64	65-74
Men	0.02 <sup>a</sup>	0.58	1.14
Women	0.12	1.67	2.56

<sup>a</sup>Estimated by Symmons *et al* from male:female ratio as this age group was not included in the survey

### 5.2.3.1.2 Townsend deprivation score

QRISK2-2012 uses Townsend scores, a deprivation index that uses Census data on four variables - car access, unemployment, overcrowding and housing tenure (60). A positive score indicates greater deprivation, and vice versa (509).

However, IMD 2004 quintiles rather than Townsend score were available in the HSE 2003 and 2004. Therefore, individuals were assigned an estimated Townsend score based on their IMD 2004 quintile. Data on Townsend scores for England were obtained (510). The Townsend scores were divided into quintiles and the mean score calculated in each quintile (see Table 5-5). This mean score was then assigned to the corresponding IMD 2004 quintile in the HSE datasets. In order to assess the robustness of this approach, the analyses (described in section 5.2.5.3) were repeated using the minimum and maximum Townsend scores in each quintile, rather than the mean score.

Hippesley-Cox *et al* adopted a similar approach in an analysis of the performance of the original QRISK calculator in a dataset that contained quintiles of Townsend score rather than individual scores (511). In that study, each individual within a given Townsend score quintile was assigned the median Townsend score for that quintile. In this analysis, median scores were also calculated for each quintile and were found to be similar to the mean score (see Table 5-5).

**Table 5-5: Results for calculated mean and median Townsend score by quintile**

Townsend score quintile	Mean Townsend score	Median Townsend score
1 - least deprived	-4.05	-3.95
2	-2.55	-2.57
3	-0.79	-0.81
4	1.70	1.66
5 - most deprived	5.69	5.34

#### 5.2.4 Missing data

The HSE 2003 and 2004 contain a high proportion of missing data on two variables required for the calculation of QRISK2-2012, namely blood pressure and cholesterol (39.8% and 44.1%, respectively), with fewer missing data on body mass index and family history of CVD (12.1% and 8.0%, respectively). The unweighted sample contained 4,051 complete cases (88 black Caribbean, 215 Indian, 72 Pakistani, 36 Bangladeshi, 228 Irish and 3,412 general population of whom 3,180 were white). The presence of missing data is consistent with the design of the HSE where body mass index and family history of CVD were measured at the interview stage and blood pressure and cholesterol were measured at the nurse visit, a component of the survey with a lower response rate than the interview stage (see sections 4.2.1.4 and 5.2.1.1).

##### 5.2.4.1 Options for addressing missing data

Missing data may be problematic because it can create unrepresentative samples and may lead to biased results (512). A number of approaches for dealing with missing data exist, the choice of which depends on why it is missing. Missing data have been described as being ignorable or non-ignorable (513), where the missingness of ignorable data does not depend on its value whereas it does for non-ignorable data (514). Ignorable data can also be classified as missing at random or missing completely at random (514). Data that are missing at random can be explained by other measured variables (512), in contrast to data that are

missing completely at random where chance rather than other variables accounts for the missing data. Non-ignorable data can be described as missing not at random, where the missing data are dependent on variables that have not been measured (512). In practice, it can be difficult to clearly classify missing data into one of these categories, and a mixture often occurs (512). In the case of the HSE 2003 and 2004, it seems reasonable to assume that the missing data are most likely missing at random. The design of the survey where all participants were offered a nurse visit but some declined, suggests that data collected at the nurse visit would not be entirely randomly missing but could depend on other measured characteristics that would determine a person's likelihood of participating, such as age and sex. There is little evidence to suggest that these missing data fit the definition of being missing not at random. Given an understanding of the nature of the missing data, a number of approaches for addressing it can be considered. One commonly used approach is complete case analysis, where cases with missing data are not included in the analysis. This is a simple approach but it can produce biased results if the cases with missing data are not representative of the overall sample and the unrepresentative variables matter for the analysis (512). Indeed, it has been suggested that complete case analysis only gives valid results when the data are missing completely at random (513). Alternative approaches for dealing with missing data include multiple imputation, mean substitution, regression imputation and hot deck substitution (514). The performance of these techniques has been found to vary, with evidence of particularly poor performance from complete case analysis (514).

#### **5.2.4.2 Multiple imputation**

A widely accepted approach for dealing with missing data is multiple imputation (515). This approach may be superior to the alternative options, and in the case of data that are missing at random has been found to produce unbiased results (516). In multiple imputation, missing values are estimated from existing values in other variables in the dataset (512). This process is repeated a number of times to create multiple imputed datasets that can then be combined for the final analysis (515). This approach has the advantage of maintaining variability within the data and acknowledging uncertainty in the estimations by repeating the process multiple times (512).

Given the limitations of complete case analysis and the assumption that the missing data were most likely missing at random, multiple imputation was used to estimate the missing values for the variables required for calculation of QRISK2-2012 and subsequent analyses. A multiple imputation model was built using Stata's Imputation by Chained Equations (ICE) command. The model included systolic blood pressure, diastolic blood pressure, total cholesterol concentration, HDL cholesterol concentration, body mass index and family history of CVD, plus age, sex, ethnicity, IMD 2004, diabetes, smoking status, whether taking lipid lowering medication and treated hypertension. The QRISK2 calculation did not require the lipid lowering medication variable but it was added to the imputation model because the cholesterol measurements included people taking these medications and were therefore influenced by this variable. Prior to running the model, the small number of cases with missing data on smoking, diabetes and antihypertensive medication were excluded, along with individuals not aged 40-74 years and those with CVD, and survey weights were specified.

Initial analysis of the imputed data indicated that certain values had been imputed that would be highly improbable, for example a body mass index of nine. Therefore, intervals were set so that imputed values did not fall outside the existing ranges of blood pressure, cholesterol or body mass index observed in the HSE. This was done by specifying a lower and upper limit for each of these variables in the multiple imputation model (517).

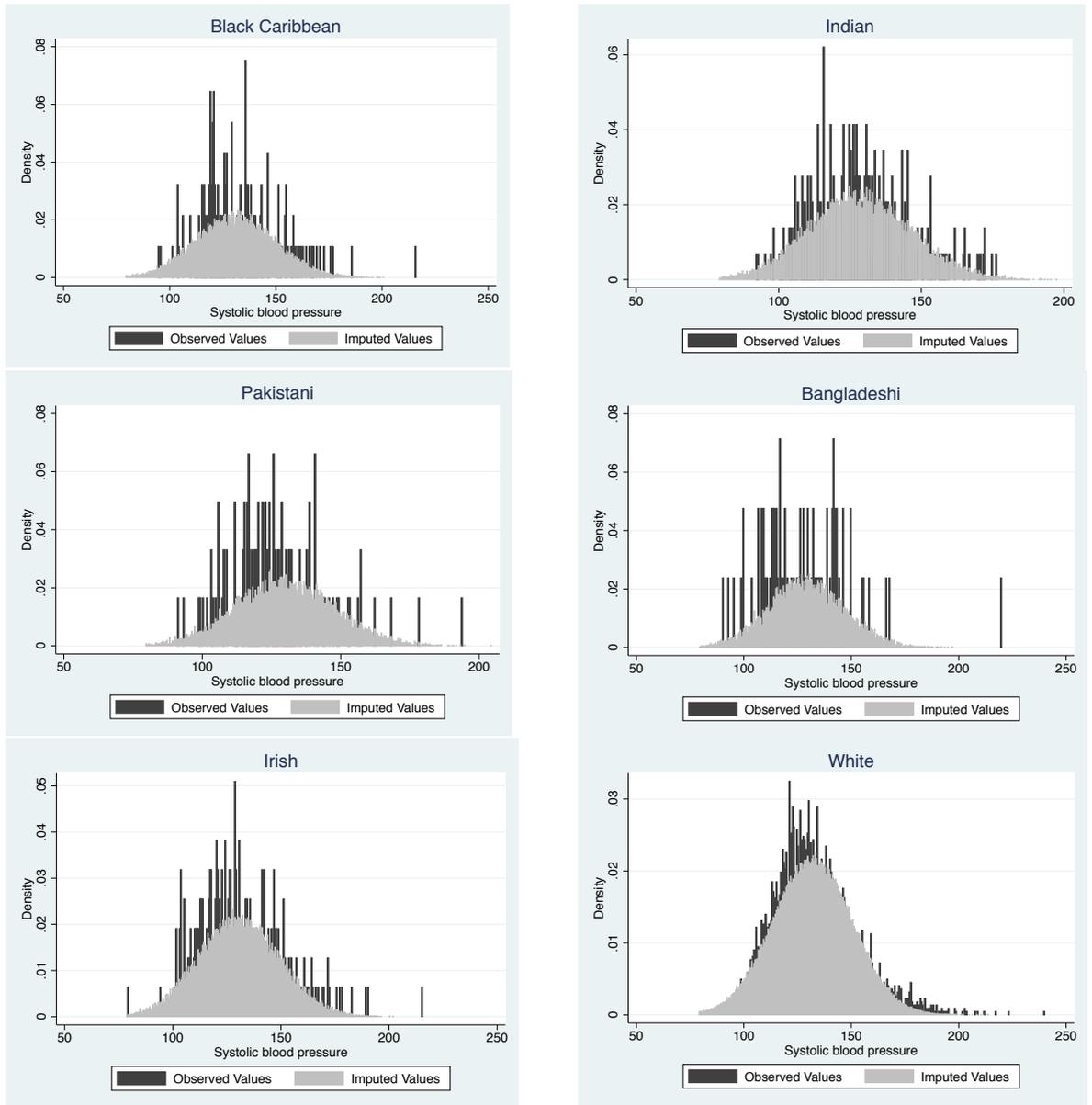
The multiple imputation model was run with 100 imputations. This number was determined by calculating the fraction of missing information ( $\gamma$ ) for each variable to be imputed. The  $\gamma$  values obtained were systolic blood pressure 0.49, diastolic blood pressure 0.54, total cholesterol 0.68, HDL cholesterol 0.59, and family history of CVD 0.11. On the basis of previous literature this suggested that 100 imputations would be required (518).

Descriptive statistics and histograms were used to compare the distribution of the original and imputed data (see Table 5-6, Figure 5-1 and Figure 5-2). The distributions were generally similar across the continuous imputed variables. Histograms for HDL cholesterol concentration and body mass index showed a similar picture to those for systolic blood pressure and total cholesterol concentration.

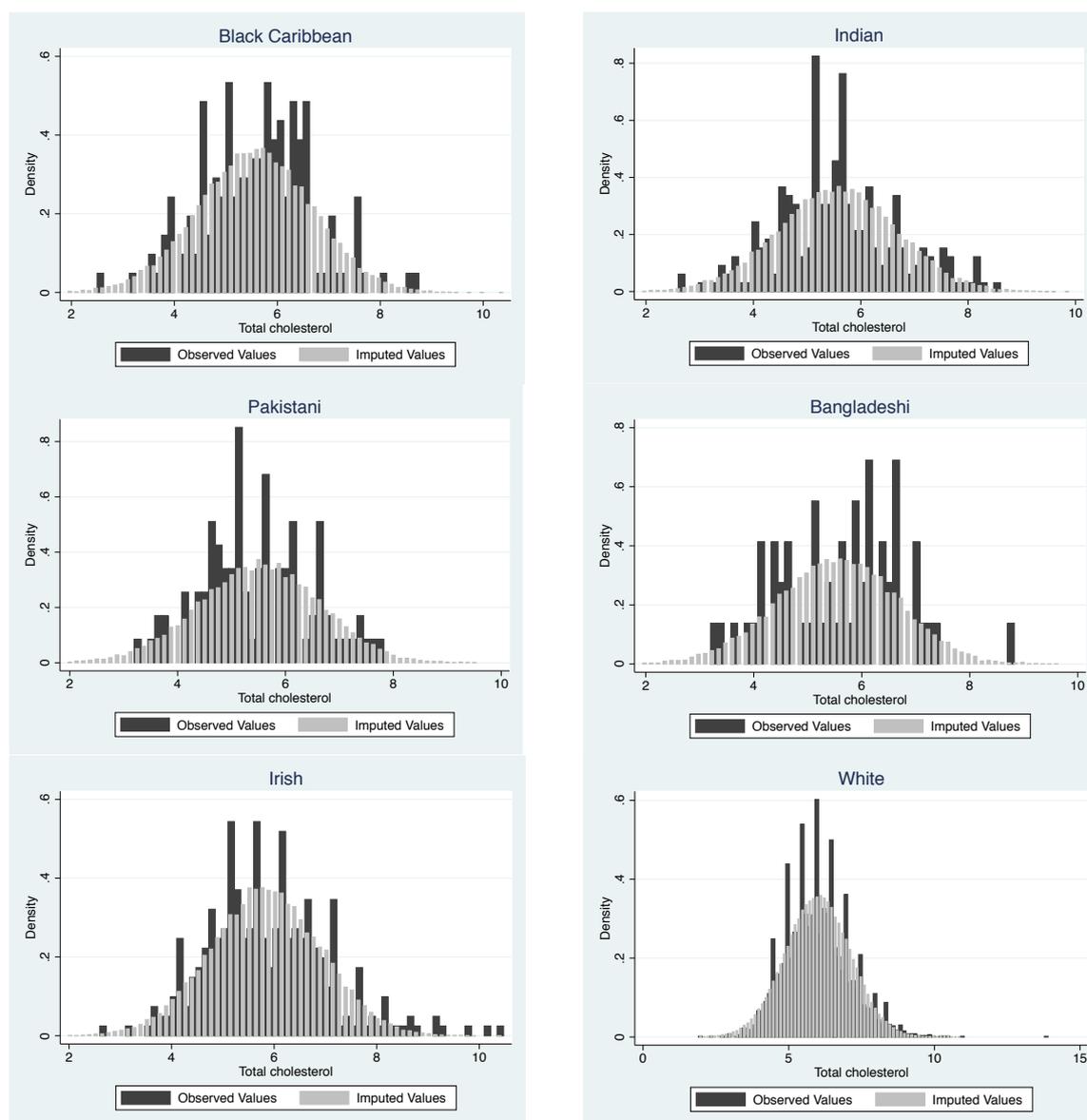
**Table 5-6: Mean and standard errors of continuous cardiovascular variables from complete cases and imputed data**

		Systolic blood pressure		Total cholesterol		HDL cholesterol		Body mass index	
		Mean	SE	Mean	SE	Mean	SE	Mean	SE
Black Caribbean	Complete cases	132.74	0.77	5.57	0.05	1.48	0.01	28.94	0.24
	Imputed data	131.95	1.18	5.57	0.08	1.44	0.02	28.88	0.26
Indian	Complete cases	127.83	0.72	5.55	0.05	1.37	0.02	27.12	0.19
	Imputed data	128.21	0.94	5.56	0.06	1.41	0.02	27.21	0.21
Pakistani / Bangladeshi	Complete cases	126.01	0.77	5.52	0.05	1.26	0.01	28.08	0.21
	Imputed data	128.13	1.00	5.53	0.07	1.37	0.02	28.01	0.23
Irish	Complete cases	129.92	0.75	5.79	0.04	1.58	0.02	27.33	0.21
	Imputed data	130.14	0.95	5.82	0.06	1.55	0.02	27.22	0.23
White	Complete cases	132.05	0.23	5.98	0.01	1.54	0.00	27.72	0.06
	Imputed data	132.01	0.26	5.98	0.02	1.54	0.01	27.75	0.06

HDL high density lipoprotein, SE standard error



**Figure 5-1: Histograms comparing original and imputed data on systolic blood pressure by ethnic group**



**Figure 5-2: Histograms comparing original and imputed data on total cholesterol concentration by ethnic group**

The QRISK2-2012 calculator, obtained from QRISK open source software (519), was then applied to each imputed dataset. The QRISK2-2012 calculator only accepts values within a specified range for each variable. Most notably, only body mass index values between 20 and 40 were valid. Therefore any body mass index values outside of this range were recoded as 20 or 40 as appropriate. A mean QRISK2-2012 score was then calculated for each individual from his or her 100 calculated values, consistent with a method used elsewhere (505). High cardiovascular risk was defined as a QRISK2-2012 score  $\geq 20\%$ . Descriptive statistics were used to compare the QRISK2-2012 score calculated on complete cases and from the imputed data, and indicated slightly higher mean scores from the imputed data (see Table 5-7).

In addition to the use of imputed data, the analyses described in section 5.2.5.3 were repeated with complete cases only in order to test the robustness of the results from the multiple imputation model.

**Table 5-7: Comparison of QRISK2-2012 scores between complete cases and imputed data**

	Complete cases		Imputed data	
	Mean	SD	Mean	SD
Black Caribbean	8.60	8.69	10.04	10.03
Indian	10.63	11.97	11.46	11.69
Pakistani/Bangladeshi	15.45	13.64	18.14	16.17
Irish	8.85	8.93	10.70	9.76
White	10.68	10.09	11.22	10.12

SD standard deviation

#### **5.2.4.3 Repeated analyses using Framingham and Ethrisk cardiovascular risk calculators**

The analyses were repeated using the Framingham and Ethrisk cardiovascular risk calculators, in order to explore whether the use of different calculators influenced the findings. The Framingham cardiovascular risk score was calculated using Anderson *et al's* approach (149). Adjustments recommended by NICE for South Asian males (multiply score by 1.4) and a positive family history (multiply score by 1.5) were applied (121). Ethrisk was calculated based on the published method, with diabetic individuals excluded as recommended (445).

### **5.2.5 Analysis**

#### **5.2.5.1 Demographic and cardiovascular characteristics**

Ethnic differences in demographic and cardiovascular characteristics were assessed. The independent samples t-test was used for continuous variables and a chi-squared test for categorical variables, with the general population as the reference group.

#### **5.2.5.2 Unit cost**

The unit cost of a screening appointment was set at £23.70. This was based on a previous Department of Health estimation, and included the costs of administration, nurse and healthcare assistant time, and blood tests (160). The one-off costs of programme start-up, were not included. The costs of subsequent investigations and interventions were not included as the aim of this analysis was to evaluate the cost-effectiveness of identifying high-risk individuals only.

### 5.2.5.3 Coverage, effectiveness and cost-effectiveness

A similar analytical approach to that used by Lawson *et al* for the general population was applied (192). The coverage, effectiveness and cost-effectiveness of targeting screening at the most deprived areas were calculated in each ethnic group (see Figure 5-3). This was then compared with mass screening using an incremental approach, which assessed the additional costs and benefits of extending the targeted programme to the whole population.

The targeted screening strategy was based on screening the most deprived IMD 2004 quintile. The overall proportion screened; the proportion of high-risk individuals screened (and therefore identified); the proportion screened who would be found to be at high-risk and the number needed to screen to identify one person at high cardiovascular risk were calculated in each ethnic group. The proportion of the high-risk population identified is a measure of how many of the high-risk individuals in each ethnic group would be identified by targeting screening at the most deprived areas and is therefore a measure of coverage; the proportion screened found to be at high-risk demonstrates how many of the individuals living in the most deprived areas are found to have high cardiovascular risk and is therefore an indicator of the efficiency of the screening strategy. This latter value is equivalent to the positive predictive value (PPV) and was used to calculate the number needed to screen (NNS) to identify one individual at high cardiovascular risk ( $NNS=1/PPV$ ). The mean cost to identify one high-risk individual was calculated by multiplying the number needed to screen by the estimated unit cost.

An incremental analysis was then carried out to assess the additional costs and benefits from extending targeted screening to the whole population (mass screening). The additional proportion of each ethnic group screened, additional proportion of the high-risk population identified, proportion of the additional screened population found to be at high-risk and number needed to screen to identify one additional person at high cardiovascular risk were calculated for each ethnic group. An incremental cost-effectiveness ratio was then calculated from the additional number needed to screen multiplied by the estimated unit cost. This type of analysis, in which the cost-effectiveness of one intervention is compared with another rather than with no intervention, is consistent with the approach recommended by NICE (3).

	High risk CVD	Low risk CVD	Total
More deprived areas	a	b	a+b
Less deprived areas	c	d	c+d
Total	a+c	b+d	a+b+c+d

### Targeted screening

Coverage =  $(a+b)/(a+b+c+d)$

Proportion of high-risk population identified =  $a/(a+c)$

Proportion of screened population at high-risk =  $a/(a+b)$

### Incremental analysis

Additional coverage =  $(c+d)/(a+b+c+d)$

Additional coverage of high-risk population =  $c/(a+c)$

Proportion of additional screened population at high-risk =  $c/(c+d)$

**Figure 5-3: Values used in targeted screening and incremental analysis calculations**

### 5.2.6 Sensitivity analyses

Throughout this methods section a number of alternative methodological approaches have been described that were used to test the robustness of the results. In summary these were:

- Analysis without simulation of atrial fibrillation, chronic kidney disease and rheumatoid arthritis variables (prevalence assumed to be zero)
- Analysis using minimum and maximum Townsend scores within each quintile, rather than mean score
- Complete case analysis
- Analysis using Framingham and Ethrisk cardiovascular risk calculators instead of QRISK2

Sensitivity analyses of cost and uptake were also considered. However, varying the costs between a minimum and maximum value would not have affected any ethnic differences observed because the cost would be varied uniformly across the ethnic groups. For instance, although unit costs of £10 or £40 would produce different mean costs to identify one high-risk individual, the numbers needed to screen, and any ethnic differences in them, would be unchanged. This type of analysis would have been appropriate if the costs were to be scaled to a population level in order to describe the potential overall costs of a cardiovascular risk screening programme, but this was not the aim of this study. Likewise, varying uptake uniformly across the ethnic groups would not have

altered any ethnic differences observed, and evidence for ethnic differences in screening uptake is unclear (see section 3.7.7.1).

#### **5.2.7 Software used**

All analyses were carried out using Stata V.12 and Microsoft Excel.

## 5.3 Results

### 5.3.1 Demographic and cardiovascular characteristics

The sample comprised 493 black Caribbean, 532 Indian, 516 Pakistani/Bangladeshi, 617 Irish and 7,249 people from the general population of whom 6,633 were white (see Table 5-8). Demographic characteristics varied by ethnicity. Compared to the general population, the Indian and Pakistani/Bangladeshi groups were significantly younger, there were significantly fewer men in the black Caribbean and Irish groups, and significantly higher proportions of the black Caribbean, Pakistani/Bangladeshi and Irish groups lived in the most deprived quintile.

Cardiovascular characteristics also varied by ethnicity (see Table 5-8). The prevalence of diabetes was significantly higher in the black Caribbean, Indian and Pakistani/Bangladeshi groups compared to the general population. The prevalence of a family history of CVD was also significantly higher in Indian and Pakistani/Bangladeshi groups, although it was significantly lower in the black Caribbean group, compared to the general population. In contrast, lower proportions of the black Caribbean, Indian and Pakistani/Bangladeshi groups were current smokers than the general population. Mean systolic blood pressure was significantly lower in the Indian and Pakistani/Bangladeshi groups than in the general population, with significantly higher proportions of the black Caribbean, Indian and Pakistani/Bangladeshi groups taking antihypertensive medication. Total cholesterol was significantly lower in the black Caribbean, Indian, Pakistani/Bangladeshi and Irish groups compared to the general population, although HDL cholesterol was also significantly lower in the black Caribbean, Indian and Pakistani/Bangladeshi groups. Compared to the general population, mean body mass index was significantly higher in the black Caribbean group but significantly lower in the Indian and Irish groups.

### 5.3.2 Targeted screening

There were ethnic differences in the effectiveness and cost-effectiveness of targeted screening in detecting individuals at high cardiovascular risk.

Targeted screening of the most deprived quintile would result in higher proportions of ethnic minority groups being screened compared to the general population and white group (see Table 5-9). Overall, 58.2% of the

Pakistani/Bangladeshi group and 44.1% of the black Caribbean group would be screened, compared with only 14.1% of the general population. Targeted screening would also result in greater coverage of high-risk individuals among ethnic minority groups than in the general population or white group; 68.7% of high-risk Pakistani/Bangladeshi individuals and 69.8% of high-risk black Caribbean individuals would be identified in contrast to only 19.2% of high-risk individuals from the general population. Among ethnic minority groups, higher proportions of the screened populations would be found to be at high risk compared to the general population. For instance, 39.9% of screened Pakistani/Bangladeshi individuals and 34.9% of screened Irish individuals would be found to be at high risk compared to 24.2% of the general population. This would result in a lower number needed to screen to detect one high-risk individual in ethnic minority groups than in the general population and white group. The lowest number needed to screen was observed in the Pakistani/Bangladeshi group where only 2.5 people would need to be screened to detect one person at high cardiovascular risk incurring a cost of £59. In comparison, in the general population 4.1 people would need to be screened at a cost of £98.

### **5.3.3 Comparison of mass and targeted screening**

In all ethnic groups, targeted screening was more cost-effective than mass screening (see Table 5-9). If targeted screening were expanded to mass screening there would be a lower prevalence of high-risk individuals among the additional screened population with a higher cost to identify additional high-risk individuals. However, the actual figures varied by ethnicity. The proportion of the additional screened population found to be at high risk was lowest in the black Caribbean and Irish groups, and therefore the number needed to screen to identify an additional high-risk individual and the associated cost of doing so was highest in these groups (additional numbers needed to screen 11.3 and 6.8 at costs of £269 and £161, respectively). In contrast, the additional number needed to screen was lowest in the Pakistani/Bangladeshi group (4.0) with a lower associated cost (£94). Incremental results for the Indian group were similar to the general population and white group.

### 5.3.4 Sensitivity analyses

#### 5.3.4.1 *Alternative cardiovascular risk calculators*

Repeating the analysis using the Framingham and Ethrisk cardiovascular risk calculators produced results that were broadly comparable with the main analysis (see Table 5-10 and Table 5-11). With respect to targeted screening, the number needed to screen to detect one high-risk individual and the associated cost of doing so were lowest in the Pakistani/Bangladeshi and Irish groups with both Framingham and Ethrisk. Similar to the main analysis there were ethnic differences in the cost-effectiveness of expanding targeted to mass screening, and targeted screening was more cost-effective than mass screening in all ethnic groups. Whilst, generally speaking, the direction of observed ethnic differences was consistent with the main analysis, the size of the differences calculated using Framingham and Ethrisk varied.

#### 5.3.4.2 *Complete case analysis*

Analysis of complete cases only gave results that were consistent with the main analysis (see Table 5-12). For targeted screening, the lowest number needed to screen to detect one person at high cardiovascular risk and the cost of doing so were lowest in the Indian and Pakistani/Bangladeshi groups. Targeted screening was more cost-effective than mass screening across the ethnic groups, although it was not possible to calculate this result in the black Caribbean group due to limited numbers in the sample.

#### 5.3.4.3 *Estimated variables in QRISK2*

Analyses using alternative estimated Townsend scores, and atrial fibrillation, rheumatoid arthritis and chronic kidney disease variables produced results that were consistent with the main analysis. Varying the estimated Townsend deprivation scores from the minimum to the maximum value within each quintile shifted the number needed to screen higher and lower, respectively. The effect was consistent across the ethnic groups and reflected lower proportions of the population being at high cardiovascular risk using the minimum quintile value, which denotes lower socioeconomic deprivation, and vice versa. The effect of assuming that the prevalence of atrial fibrillation, rheumatoid arthritis and chronic kidney disease was zero was to slightly reduce the proportion of the sample at high cardiovascular risk (see Table 5-13). This marginally increased the number needed to screen to detect one person at high cardiovascular risk,

and the associated cost, for targeted screening and in the incremental analysis. This small reduction in number needed to screen occurred across all ethnic groups, although it was slightly larger in the black Caribbean group.

Table 5-8: Demographic and cardiovascular characteristics by ethnic group

	General population <sup>a</sup>	Black Caribbean	Indian	Pakistani / Bangladeshi	Irish	White <sup>a</sup>
n	7,249	493	532	516	617	6,633
Age (Mean (SD))	54.2 (9.6)	53.5 (10.7)	51.7 (8.7)	51.4 (9.3)	54.1 (9.4)	54.4 (9.6)
<i>p-value</i>		0.150	<0.001	<0.001	0.952	0.174
Male (%)	48.1	41.5	44.3	47.7	42.3	48.0
<i>p-value</i>		0.004	0.096	0.855	0.006	0.659
Most deprived quintile (%)	14.2	44.1	15.5	58.0	18.0	13.0
<i>p-value</i>		<0.001	0.368	<0.001	0.012	0.053
Smoking status <sup>b</sup>						
Non-smoker (%)	42.3	59.2	76.7	72.4	37.5	40.7
Ex-smoker (%)	34.9	21.5	10.9	9.4	36.6	36.2
Light smoker (%)	4.9	10.2	6.4	7.1	5.7	4.6
Moderate smoker (%)	9.0	6.2	4.8	7.4	9.7	9.1
Heavy smoker (%)	8.9	2.9	1.2	3.7	10.5	9.4
<i>p-value</i>		<0.001	<0.001	<0.001	0.177	0.268
Family history CVD (%)	14.9	11.2	18.6	16.8	12.7	14.9
<i>p-value</i>		<0.001	<0.001	<0.001	<0.001	0.180
Taking anti-hypertensive medication (%)	15.9	27.1	21.3	21.7	17.3	15.4
<i>p-value</i>		<0.001	0.002	0.001	0.331	0.513
Diabetes (%)	3.9	13.2	11.1	17.8	3.0	3.5
<i>p-value</i>		<0.001	<0.001	<0.001	0.382	0.244
Systolic blood pressure (Mean (SE))	131.8 (0.25)	132.0 (1.18)	128.2 (0.94)	128.1 (1.00)	130.1 (0.95)	132.0 (0.26)
<i>p-value</i>		0.845	<0.001	<0.001	0.074	0.488
Total cholesterol (Mean (SE))	5.9 (0.02)	5.6 (0.08)	5.6 (0.06)	5.5 (0.07)	5.8 (0.06)	6.0 (0.02)
<i>p-value</i>		<0.001	<0.001	<0.001	0.031	0.153

**Table 5-8 continued...**

HDL cholesterol (Mean (SE))	1.5 (0.01)	1.4 (0.02)	1.4 (0.02)	1.4 (0.02)	1.6 (0.02)	1.5 (0.01)
<i>p-value</i>		<0.001	<0.001	<0.001	0.411	0.427
BMI (Mean (SE))	27.7 (0.06)	28.9 (0.26)	27.2 (0.21)	28.0 (0.23)	27.2 (0.23)	27.7 (0.06)
<i>p-value</i>		<0.001	0.023	0.226	0.017	0.850

Reference group for p-values is general population. <sup>a</sup> General population and white data from HSE 2003; <sup>b</sup> Light smoker is less than 10 cigarettes per day, moderate smoker 10-19 cigarettes per day, and heavy smoker 20 or more cigarettes per day. SD standard deviation, CVD cardiovascular disease, SE standard error, HDL high-density lipoprotein, BMI body mass index

**Table 5-9: Cost-effectiveness of targeted and mass screening for high cardiovascular risk by ethnic group**

	General population <sup>a</sup>	Black Caribbean	Indian	Pakistani / Bangladeshi	Irish	White <sup>a</sup>
<b>Targeted screening at most deprived quintile</b>						
Proportion of ethnic group screened	14.1%	44.1%	15.5%	58.2%	18.0%	13.0%
Proportion of high-risk population identified	19.2%	69.8%	23.3%	68.7%	34.2%	17.9%
Proportion of screened population at high-risk	24.2%	25.9%	30.0%	39.9%	34.9%	24.8%
NNS <sup>b</sup>	4.1	3.9	3.3	2.5	2.9	4.0
Mean cost to detect one person at high cardiovascular risk	£98	£92	£79	£59	£68	£96
<b>Incremental analysis of mass screening</b>						
Additional coverage of ethnic group	85.9%	55.9%	84.5%	41.8%	82.0%	87.0%
Additional coverage of high-risk population	80.8%	30.2%	76.7%	31.3%	65.8%	82.1%
Proportion of additional screened population at high-risk	16.8%	8.8%	18.2%	25.3%	14.7%	17.0%
Additional NNS <sup>b</sup>	6.0	11.3	5.5	4.0	6.8	5.9
Mean cost to detect one additional person at high cardiovascular risk <sup>c</sup>	£141	£269	£130	£94	£161	£139

<sup>a</sup> White and general population data from HSE 2003; <sup>b</sup> NNS number needed to screen; <sup>c</sup> This number is also the incremental cost-effectiveness ratio

**Table 5-10: Cost-effectiveness of targeted and mass screening for high cardiovascular risk by ethnic group using Framingham cardiovascular risk calculator**

	General population <sup>a</sup>	Black Caribbean	Indian	Pakistani / Bangladeshi	Irish	White <sup>a</sup>
Targeted screening at most deprived quintile						
Proportion of ethnic group screened	14.1%	44.1%	15.5%	58.0%	18.0%	13.0%
Proportion of high-risk population identified	16.8%	71.3%	16.5%	71.4%	29.0%	15.9%
Proportion of screened population at high-risk	22.6%	27.3%	23.5%	29.3%	31.6%	23.2%
NNS <sup>b</sup>	4.4	3.7	4.2	3.4	3.2	4.3
Mean cost to detect one person at high cardiovascular risk	£105	£87	£101	£81	£75	£102
Incremental analysis of mass screening						
Additional coverage of ethnic group	85.9%	55.9%	84.5%	42.0%	82.0%	87.0%
Additional coverage of high-risk population	83.2%	28.7%	83.5%	28.6%	71.0%	84.1%
Proportion of additional screened population at high-risk	18.4%	8.7%	21.9%	16.3%	16.9%	18.4%
Additional NNS <sup>b</sup>	5.4	11.5	4.6	6.2	5.9	5.4
Mean cost to detect one additional person at high cardiovascular risk <sup>c</sup>	£129	£273	£108	£146	£140	£129

<sup>a</sup> White and general population data from HSE 2003; <sup>b</sup> NNS number needed to screen; <sup>c</sup> Also the incremental cost-effectiveness ratio

**Table 5-11: Cost-effectiveness of targeted and mass screening for high cardiovascular risk by ethnic group using Ethrisk cardiovascular risk calculator**

	General population <sup>a</sup>	Black Caribbean	Indian	Pakistani / Bangladeshi	Irish	White <sup>a</sup>
Targeted screening at most deprived quintile						
Proportion of ethnic group screened	13.9%	42.1%	14.0%	56.3%	17.4%	12.9%
Proportion of high-risk population identified	16.4%	63.2%	18.3%	65.0%	26.1%	15.8%
Proportion of screened population at high-risk	20.1%	21.4%	20.7%	21.6%	30.8%	21.1%
NNS <sup>b</sup>	5.0	4.7	4.8	4.6	3.2	4.7
Mean cost to detect one person at high cardiovascular risk	£118	£111	£115	£110	£77	£112
Incremental analysis of mass screening						
Additional coverage of ethnic group	86.1%	57.9%	86.0%	43.7%	82.6%	87.1%
Additional coverage of high-risk population	83.6%	36.8%	81.7%	35.0%	73.9%	84.2%
Proportion of additional screened population at high-risk	16.6%	9.0%	15.0%	15.0%	18.4%	16.6%
Additional NNS <sup>b</sup>	6.0	11.1	6.7	6.7	5.4	6.0
Mean cost to detect one additional person at high cardiovascular risk <sup>c</sup>	£143	£262	£158	£158	£129	£143

<sup>a</sup> White and general population data from HSE 2003 with cardiovascular risk calculated using Framingham risk calculator; <sup>b</sup> NNS number needed to screen; <sup>c</sup> This number is also the incremental cost-effectiveness ratio

**Table 5-12: Complete case analysis results for cost-effectiveness of targeted and mass screening for high cardiovascular risk by ethnic group**

	General population <sup>a</sup>	Black Caribbean	Indian	Pakistani / Bangladeshi	Irish	White <sup>a</sup>
Targeted screening at most deprived quintile						
Proportion of ethnic group screened	10.9%	48.4%	14.6%	48.9%	13.2%	10.1%
Proportion of high-risk population identified	16.7%	100.0%	40.6%	57.7%	24.2%	15.9%
Proportion of screened population at high-risk	23.0%	12.9%	41.5%	32.6%	18.8%	23.9%
NNS <sup>b</sup>	4.4	7.8	2.4	3.1	5.3	4.2
Mean cost to detect one person at high cardiovascular risk	£103	£184	£57	£73	£126	£99
Incremental analysis of mass screening						
Additional coverage of ethnic group	89.1%	51.6%	85.4%	51.1%	86.8%	89.9%
Additional coverage of high-risk population	83.3%	0.0%	59.4%	42.3%	75.8%	84.1%
Proportion of additional screened population at high-risk	14.1%	0.0%	10.4%	22.9%	8.9%	14.2%
Additional NNS <sup>b</sup>	7.1	§	9.6	4.4	11.2	7.0
Mean cost to detect one additional person at high cardiovascular risk <sup>c</sup>	£169	§	£228	£103	£265	£167

<sup>a</sup> White and general population data from HSE 2003; <sup>b</sup> NNS number needed to screen; <sup>c</sup> This number is also the incremental cost-effectiveness ratio; § No value as there were no black Caribbean individuals with high cardiovascular risk in less deprived areas

**Table 5-13: Cost-effectiveness of targeted and mass screening for high cardiovascular risk by ethnic group with prevalence of atrial fibrillation, rheumatoid arthritis and chronic kidney disease assumed to be zero**

	General population <sup>a</sup>	Black Caribbean	Indian	Pakistani / Bangladeshi	Irish	White <sup>a</sup>
Targeted screening at most deprived quintile						
Proportion of ethnic group screened	14.1%	44.1%	15.5%	58.2%	18.0%	13.0%
Proportion of high-risk population identified	19.6%	69.4%	22.1%	68.4%	34.9%	18.1%
Proportion of screened population at high-risk	22.7%	23.0%	27.2%	38.1%	33.5%	22.9%
NNS <sup>b</sup>	4.4	4.3	3.7	2.6	3.0	4.4
Mean cost to detect one person at high cardiovascular risk	£105	£103	£87	£62	£71	£103
Incremental analysis of mass screening						
Additional coverage of ethnic group	85.9%	55.9%	84.5%	41.8%	82.0%	87.0%
Additional coverage of high-risk population	80.4%	30.6%	77.9%	31.6%	65.1%	81.9%
Proportion of additional screened population at high-risk	15.3%	8.0%	17.6%	24.6%	13.7%	15.5%
Additional NNS <sup>b</sup>	6.5	12.5	5.7	4.1	7.3	6.5
Mean cost to detect one additional person at high cardiovascular risk <sup>c</sup>	£155	£297	£134	£96	£173	£153

<sup>a</sup> White and general population data from HSE 2003; <sup>b</sup> NNS number needed to screen; <sup>c</sup> This number is also the incremental cost-effectiveness ratio

## **5.4 Discussion**

### **5.4.1 Principal findings**

Higher proportions of ethnic minority groups would be screened using a targeted screening strategy than in the general population or white group. In particular, high proportions of the Pakistani/Bangladeshi and black Caribbean groups would be identified by targeted screening of the most deprived areas. Coverage of high-risk individuals by targeted screening was good in the general population but was higher in ethnic minority groups. Targeted screening was more efficient and cost-effective at identifying high-risk individuals in all ethnic minority groups than in the general population, especially in the Pakistani/Bangladeshi group. Despite similar coverage of high-risk individuals, targeted screening performed relatively well in the Indian group where it was more efficient and cost-effective than in the general population.

In comparison with targeted screening, mass screening was less cost-effective in all ethnic groups. The cost per additional high-risk individual identified was lowest in the Indian and Pakistani/Bangladeshi groups. However, there was a greater difference between the Pakistani/Bangladeshi group and the general population than between the Indian group and general population. This suggests that both strategies would be more cost-effective in South Asian groups, particularly for the Pakistani/Bangladeshi group, than in the general population. In contrast, the cost per additional high-risk individual identified was highest in the black Caribbean group, suggesting that extending targeted screening to the whole population would be less cost-effective in this group than in the general population.

### **5.4.2 Strengths and limitations**

This analysis used the QRISK2-2012 cardiovascular risk calculator. QRISK2-2012 was chosen as it may be a more accurate predictor of cardiovascular risk in the UK than the Framingham calculator, and may be particularly relevant for ethnic minority and socioeconomically deprived individuals (444, 448). However, the use of QRISK2-2012 required estimation of a number of variables that were not available in the HSE 2003 and 2004, namely Townsend score, atrial fibrillation, chronic kidney disease and rheumatoid arthritis. This may have led to inaccuracies in the QRISK2-2012 scores, although sensitivity analyses using different estimated Townsend scores and with zero prevalence of atrial

fibrillation, chronic kidney disease and rheumatoid arthritis produced results that were consistent with the main analysis. In addition, the estimations were carried out consistently across all ethnic groups so are unlikely to have introduced systematic errors between the groups. This is relevant as the focus of this analysis was to investigate ethnic differences rather than produce estimations of cost-effectiveness to be used at a population level, for which inaccuracies in absolute estimated values would be important. The same prevalence estimates for atrial fibrillation, chronic kidney disease and rheumatoid arthritis were used across the ethnic groups. Whilst there is evidence of ethnic differences in the prevalence of these conditions (520-523), ethnic group specific prevalence estimates by age group could not be found. QRISK2 provides prevalence estimates by ethnicity but not stratified by age (438). Given that the prevalence of these conditions increases markedly with age it was decided that age specific estimates were most appropriate. However, incorporating ethnic differences in the prevalence of these conditions could have strengthened this analysis, and potentially added to the ethnic differences observed. Family history was defined differently in the HSE and by QRISK2-2012. The use of the HSE definition may have underestimated cardiovascular risk as it only included parental mortality rather than morbidity or mortality in any first degree relative, although with a higher age threshold and a broader inclusion of CVD conditions. Individuals taking lipid-lowering medications were not excluded from this analysis. Whilst QRISK2 recommends the exclusion of these individuals this was not possible given the design of the survey - there was a high proportion of missing data on this variable, which was measured at the nurse visit, meaning that it would not be possible to accurately exclude these individuals without potentially biasing the results of the multiple imputation model. Including these individuals may have led to an underestimation of the cost-effectiveness of both targeted and mass screening, as people taking lipid-lowering medication may have iatrogenically lowered cholesterol levels that could reduce their QRISK2-2012 score.

There was a high proportion of missing data on variables such as cholesterol and blood pressure. A widely accepted method of dealing with missing data - multiple imputation - was therefore used. Steps were taken to ensure that the imputed data were consistent with the original data. The multiple imputation model used 100 imputations. Whilst this is a higher number of imputations than

has been used in previous studies, this approach was based on literature that suggests that high numbers of imputations should be used (518). An alternative approach to multiple imputation would have been to analyse complete cases only. However, this could have biased the results, especially if the representativeness of individuals with complete data varied by ethnicity, and would have further limited the sample size. Despite this, sensitivity analysis including complete cases only produced results that were broadly consistent with the main analysis.

The survey data used were limited by the age of the data and the sample size. These data from 2003 and 2004 will not reflect recent changes in the demographic or cardiovascular characteristics of the general population or ethnic minority groups. For instance, there is evidence of increasing movement of some ethnic minority populations to more affluent areas (524), a change that could reduce the cost-effectiveness of targeted screening of the most deprived areas. Small sample sizes precluded the inclusion of some ethnic groups in the analysis. In particular, the Chinese and black African groups were excluded based on a judgement of what sample size was reasonable for inclusion in the analysis. The Pakistani and Bangladeshi groups were merged because of small numbers; however, separate analyses of these groups produced consistent results. The Chinese group may have been an important group to include given the relatively low prevalence of CVD among Chinese populations, which may have led to contrasting findings compared to the other ethnic groups. Merging additional years of the HSE with the 2004 survey, in order to increase the sample sizes, was considered but not done as the additional benefit in terms of increased numbers of ethnic minority participants was small and differences in sampling procedures between the years meant there were technical difficulties in accurately weighting the merged sample. Despite these limitations, it was not possible to identify an alternative source of more recent data with a sufficiently large sample of ethnic minority individuals. The alternative sources discussed in the previous chapter (Census data and Understanding Society) would not have been appropriate as data on physical measurements, in particular cholesterol and blood pressure, were needed. Another option would have been to use UK Biobank data (525). At the time of this analysis cholesterol concentrations from this large survey were unavailable, however in future UK Biobank will provide a

useful source of up-to-date data on sufficiently large numbers of ethnic minority individuals.

This analysis did not consider socioeconomic or ethnic differences in the uptake of cardiovascular risk screening. Whilst evidence from the NHS Health Check programme suggests that there may be reduced uptake in deprived areas (526), there is no consistent evidence on the nature or direction of ethnic differences in the uptake of public health interventions (see section 3.7.7.1). Nevertheless, differences in uptake would need to be considered in the implementation and evaluation of any targeted or mass cardiovascular risk screening programme, as they could impact on the effectiveness of screening and on socioeconomic or ethnic health inequalities.

This analysis only considered costs of screening, rather than additional costs of subsequent treatment and follow-up. These additional costs would be particularly important if the costs were to be scaled to a population level in order to provide an estimated total cost for a mass or targeted screening programme. However, this was not the aim of this analysis, which was instead concerned with investigating relative ethnic differences in the cost-effectiveness of identifying high-risk individuals.

The statistical significance of the observed ethnic differences in cost-effectiveness was not assessed in this analysis. This means that it is not possible to state whether targeted or mass screening would be significantly more or less cost-effective in one ethnic group compared to another. It has been argued that consideration of uncertainty in economic analysis may be helpful for decision makers, particularly if it provides information about whether further data are needed to reduce uncertainty (527). However, the approach used in this analysis is consistent with other economic theory, whereby Claxton argues that the results of statistical tests of significance or precision, such as confidence intervals, are not relevant to decision making in health where a choice between interventions needs to be made at a particular time (528). This reflects the choices that policy makers continue to make between cardiovascular risk screening strategies, where one approach must be chosen over another regardless of the statistical significance of differences in cost-effectiveness. Whilst the decision was made not to include tests of statistical significance in

this analysis on this basis, future analyses could benefit from exploration of statistical uncertainty.

#### 5.4.3 Relations to other studies

Mass screening for high cardiovascular risk has been found to be less cost-effective than a range of alternative strategies in the general population. Lawson *et al* found that targeted screening based on area deprivation and family history was more cost-effective than mass screening in a Scottish population (192). Their analysis indicated that targeting deprived areas would identify 25% of high-risk individuals with a number needed to screen of 3.0. This contrasts with the results from the general population in this analysis, where 19.2% of high-risk individuals would be identified with a higher number needed to screen of 4.1. These differences could be explained by variations in levels of socioeconomic deprivation and cardiovascular risk between England and Scotland, alongside the use of different cardiovascular risk calculators in these analyses. Chamnan *et al* and Marshall and Rouse also found that mass screening was potentially less cost-effective and efficient than alternative strategies such as pre-stratifying individuals by cardiovascular risk (171, 173). However, these studies did not investigate the use of targeted screening of deprived areas limiting further comparisons that can be made with this analysis. Moreover, this analysis differs from this previous evidence as it explores ethnic differences rather than studying the general population.

The ethnic differences observed in deprivation and the prevalence of cardiovascular risk factors were broadly consistent with previous studies. The significantly higher proportion of the black Caribbean, Pakistani/Bangladeshi and Irish groups living in the most deprived areas in this analysis corresponds to knowledge of the concentration of these ethnic minority groups into deprived areas (308, 309, 452, 457). Similar to this analysis, a range of previous studies have demonstrated a higher risk of diabetes mellitus (11, 269, 271), and lower prevalence of smoking (271, 272), in South Asian and black Caribbean ethnic groups. The lower mean systolic blood pressure in the Indian and Pakistani/Bangladeshi groups in this analysis is consistent with previous evidence (269, 270), although a significantly higher systolic blood pressure was not observed in the black Caribbean group despite evidence of increased blood pressure in this ethnic group (270, 275). This discrepancy could be accounted for

by differences in the specific populations studied or in the age of the data (Lane *et al* and Whitty *et al*'s measurements were carried out 20-30 years earlier than the HSE 2003 and 2004). Also, compared to White individuals, higher proportions of black Caribbean, Indian and Pakistani/Bangladeshi individuals were taking anti-hypertensive medication, which may have lowered the observed blood pressure values. Evidence of ethnic differences in cholesterol concentrations is mixed; the observation in this analysis of lower total cholesterol but lower HDL cholesterol in the black Caribbean, Indian and Pakistani/Bangladeshi groups partially corresponds with findings by Bhopal *et al* and Tillin *et al* (269, 272), but contrasts with evidence of there being limited ethnic differences in cholesterol concentrations (10, 273). Ethnic differences in mean body mass index in this analysis are consistent with previous evidence of a higher prevalence of obesity in black Caribbean populations (11), and lower prevalence in South Asian populations (271). However, South Asian individuals may also have a higher prevalence of central obesity (269, 279), a measurement that was not included in this analysis.

Area deprivation measures may provide an efficient and effective way of focusing limited public health resources on at risk populations (181, 182). The previous analyses in this thesis found that area deprivation measures are better at identifying individual socioeconomic deprivation in ethnic minority groups in England. This suggests that area deprivation measures may be a sufficiently effective and efficient tool for targeting cardiovascular risk screening at deprived ethnic minority populations. The purpose of this would be to identify areas in which high concentrations of socioeconomically deprived individuals live because socioeconomically deprived individuals are known to be at increased risk of CVD, either through the presence of other cardiovascular risk factors or because of the independent effect of deprivation itself (see section 2.2) (28, 29, 31). However, area deprivation measures are subject to the "ecological fallacy" (178, 181), meaning that not all individuals screened will be socioeconomically deprived, and deprived individuals living outside of the most deprived areas will not be offered screening. This could reduce the efficiency of CVD prevention programmes targeted at deprived areas, as this is influenced by the concentration of risk in the target population, which may not be as high as expected. In addition, if socioeconomically deprived individuals living in less

deprived areas are systematically excluded, this could decrease the potential for the intervention to reduce health inequalities.

Further to the potential to identify and influence individuals, CVD prevention programmes targeted at deprived areas also have the potential to improve health through the modification of area characteristics. This is because of the independent association between individual and area socioeconomic deprivation and CVD (178, 184), and provides another reason for supporting the use of area-based interventions in CVD prevention. However, promoting healthy environments, for example by creating easier access to physical activity, would require a different type of intervention to one based on individual cardiovascular risk screening, such as that analysed here.

#### **5.4.4 Implications**

This analysis suggests that both mass and targeted cardiovascular risk screening are more efficient and cost-effective in high-risk ethnic minority groups compared to the general population. Whilst targeted screening may be more efficient and cost-effective than mass screening, especially in ethnic groups with high levels of cardiovascular risk and socioeconomic deprivation, mass screening may also be a relatively cost-effective option in these groups. This is reassuring as it suggests that both approaches have the potential to reduce ethnic health inequalities. For decision makers choosing between targeted and mass screening for high cardiovascular risk in the population as a whole, this analysis indicates that either strategy is likely to work well in ethnic minority groups with little evidence that any of the ethnic groups studied here would be systematically disadvantaged.

These findings could potentially be generalisable to ethnic minority groups with similar demographic and cardiovascular characteristics in other settings. Ethnic minority groups in countries such as the USA and New Zealand are known to be at increased risk of CVD and to be concentrated in socioeconomically deprived areas (257, 296, 453, 455), characteristics that suggest that cardiovascular screening could be useful in these groups, especially if it is targeted at deprived areas. However, given international differences in the geographical distribution of socioeconomic deprivation and ethnic minority populations any generalisation would need to be done with caution.

#### **5.4.5 Next steps**

These analyses focused on ethnic differences in the application of a high-risk approach to CVD prevention. However, as discussed in Chapter 2, population approaches may offer greater potential to prevent CVD than high-risk approaches. Therefore, a further policy choice needs to be considered - the choice between population and high-risk approaches. This raises the question of whether there are ethnic differences in the potential impact of population and high-risk approaches to CVD prevention. The next chapter addresses this question.

## **6 Chapter 6: Ethnic differences in the impact of population versus high-risk approaches to primary prevention of cardiovascular disease**

### **6.1 Introduction**

The previous chapter explored ethnic differences in the cost-effectiveness of a high-risk approach to CVD prevention. This chapter moves on to investigate both high-risk and population approaches to CVD prevention, and in particular whether there are ethnic differences in the relative effectiveness of these approaches. Population and high-risk approaches to CVD prevention were introduced and reviewed in Chapter 2, whilst ethnic differences in CVD were reviewed in Chapter 3. Relevant points arising from these chapters are summarised here.

A population approach to CVD prevention involves reducing cardiovascular risk factors by a small amount across the whole population (91). This could occur by, for example, reducing the salt content of processed food to lower blood pressure, or by introducing comprehensive tobacco control measures to reduce smoking prevalence and exposure. In contrast, the high-risk approach targets preventative interventions on individuals deemed to be at high-risk of developing CVD (91). Examples of high-risk approaches include cardiovascular screening and risk reduction programmes, such as the NHS Health Check programme (159). Population approaches may lead to a large reduction in CVD at a population level but with small risk reduction for individuals; in contrast high-risk approaches may produce a greater risk reduction for individuals but a smaller reduction in CVD in the population (91).

Health policy makers need to decide on the extent to which they invest in population or high-risk approaches to primary prevention. Evidence on which approach has the greatest potential to prevent CVD at a population level is mixed (108, 109, 111), as described in Chapter 2. The general consensus is that a combination of both approaches is needed, although the ideal balance is not known. Current CVD primary prevention policy includes both approaches, but tends to favour high-risk interventions. The NHS Health Check programme, estimated by the Department of Health to cost over £200m per year to the NHS

(although this may be an underestimate of the cost (529)) (159), demonstrates a commitment to a high-risk strategy. Population approaches, such as elimination of trans fatty acids from the food chain and minimum pricing for alcohol, have also been recommended but not yet acted upon (18, 530).

Given that public health policies need to both prevent disease and reduce health inequalities, it is important that their potential impact on important population subgroups, such as ethnic minorities, is considered (4, 5, 199). There are well-established ethnic differences in CVD and cardiovascular risk factors, as described in Chapter 3. For instance, individuals from South Asian groups are known to be at increased risk of CVD (239), there are ethnic differences in smoking prevalence (11), and blood pressure may be higher in black populations (10). This could impact the effectiveness of population and high-risk approaches if the prevalence of the risk factor to be reduced varies by ethnicity, or if a higher proportion of an ethnic group is categorised as being at high cardiovascular risk. However, it is unclear whether the effectiveness of population and high-risk approaches to CVD prevention differs across ethnic groups.

This chapter therefore addresses the following research question:

Are there ethnic differences in the potential impact of population and high-risk approaches to CVD prevention?

In this chapter potential impact is assessed in terms of both effectiveness and impact on health inequalities. Similar to the previous two chapters, the intention of these analyses was to explore the potential impact of policies designed for the population as a whole rather than policies for specific ethnic groups.

## **6.2 Methods**

### **6.2.1 Data and participants**

Cross-sectional data from the Health Survey for England (HSE) 2003 and 2004 were used (486, 503), as described in Chapters 4 and 5. Adults aged 35-74 years were included in these analyses. Population approaches to CVD prevention may impact on a wider age range than cardiovascular screening programmes so a broader age group was selected than in the previous analyses in order to capture this. Participants with a self-reported history of CVD (angina, myocardial infarction or stroke) were excluded. Black Caribbean, Indian, Pakistani, Bangladeshi, Irish and white participants were included. Similar to the previous chapter, the Pakistani and Bangladeshi groups were merged because of small numbers. The black African and Chinese groups were excluded due to small numbers. Interview weights, provided by the HSE, were applied.

### **6.2.2 Cardiovascular risk calculation**

QRISK2-2012 was used to estimate the 10-year risk of a cardiovascular event (angina, myocardial infarction, stroke or transient ischaemic attack) for each individual using the same method as the previous chapter (see Chapter 5).

### **6.2.3 Missing data**

The dataset contained large proportions of missing data on variables required for the calculation of QRISK2-2012, namely on cholesterol (45.1%) and blood pressure (40.6%), with smaller amounts on body mass index (12.1%) and family history of CVD (7.5%). Therefore, multiple imputation was used to impute values for these missing data in the same way as in the previous chapter (see section 5.2.4.2). The open source QRISK2-2012 calculator was applied to each imputed data set and a mean QRISK2-2012 score calculated for each individual (see section 5.2.4.2) (519).

### **6.2.4 Population approach**

Models were developed to simulate the potential risk factor changes that would result from a population approach to primary prevention of CVD. These models were based on reductions in cholesterol, blood pressure, body mass index and smoking. These modifiable risk factors were selected to reflect changes that could occur after implementation of national guidelines on population

approaches to CVD prevention. For instance, NICE guidance recommends a range of interventions aimed at reducing these risk factors through reductions in salt intake, saturated and trans fat consumption, and smoking, plus encouragement of physical activity (18, 531).

Plausible reductions in these risk factors were identified from the literature (see Table 6-1). A variety of risk factor reductions have been documented, and applied in similar modelling studies. Therefore, a range of reductions was defined for cholesterol, blood pressure and body mass index, designed to reflect increasing levels of impact plus uncertainty in the size of risk factor reductions that could be achieved. Initially the impact of separately reducing each risk factor was modelled and then the risk factor reductions were applied simultaneously in an overall model (see Table 6-2).

**Table 6-1: Evidence for reductions in cardiovascular risk factors following a population approach and reductions applied in models**

Risk factor	Possible interventions	Risk factor changes identified in literature	Reduction in models
Total cholesterol	<ul style="list-style-type: none"> <li>• Elimination of trans fatty acids from food chain.</li> <li>• Reduced saturated fat content in processed food.</li> </ul>	<p>Reductions of 20% in Finland and Sweden following CVD prevention programmes and changes in dietary fat intake (532, 533). 15% fall in Mauritius after changes in cooking oil (104). Reduction of 0.15 mmol/L with dietary advice (136). Previous modelling studies comparing population and high-risk CVD prevention approaches contained reductions ranging from 1-20% (108, 109, 111).</p>	0.1, 0.5 and 1 mmol/L
Systolic blood pressure	<ul style="list-style-type: none"> <li>• Reduced salt content in processed food</li> </ul>	<p>6g salt reduction per day led to reduction of 5.8mmHg; authors recommended aiming for a larger reduction in salt intake (427). Reduced salt intake led to 3.39mmHg reduction (130). Previous modelling studies comparing population and high-risk CVD prevention approaches contained reductions ranging from 1-20% (108, 109, 534).</p>	2.5, 5 and 7.5 mmHg
Body mass index	<ul style="list-style-type: none"> <li>• Physical environments that encourage physical activity</li> <li>• Taxation of sugar sweetened drinks</li> </ul>	<p>Prevalence of overweight and obesity has increased and needs to be reduced (535). Recommendation in USA for a 10% reduction in proportion of adults who are obese, plus a 10% increase in proportion with a normal weight by 2020 (536). Food price changes can affect weight (537). Evidence of whether physical activity by itself can reduce body mass index is uncertain (538, 539).</p>	0.5, 1.5 and 2.5 kg/m <sup>2</sup>
Smoking status	<ul style="list-style-type: none"> <li>• Extended smoking bans</li> <li>• Fiscal measures</li> </ul>	<p>English smoking ban decreased proportion of heavy smokers (540). Comprehensive tobacco control policies could reduce smoking prevalence by 20-40% in next 1-30 years (541).</p>	Down 1 category <sup>a</sup>

<sup>a</sup> Smoking status reduced by moving each smoker down one smoking category, i.e. heavy smokers moved to being moderate smokers, moderate smokers to light smokers, light smokers to former smokers, with no change for former and non-smokers.

**Table 6-2: Combined models of risk factor reductions in population approach**

	Risk factor reductions		
	Model 1	Model 2	Model 3
Total cholesterol	0.1 mmol/L	0.5 mmol/L	1.0 mmol/L
Blood pressure	2.5 mmHg	5 mmHg	7.5 mmHg
Body mass index	0.5 kg/m <sup>2</sup>	1.5 kg/m <sup>2</sup>	2.5 kg/m <sup>2</sup>
Smoking	Down 1 category	Down 1 category	Down 1 category

### 6.2.5 High-risk approach

Models were developed that simulated the potential impact of targeting those at high-risk with nationally recommended risk reducing interventions. Mean total cholesterol, systolic blood pressure, and body mass index were calculated for each individual from the imputed data sets. Individuals were defined as high-risk if they had a QRISK2 score  $\geq 20\%$ , total cholesterol  $\geq 7.5$  mmol/L or blood pressure  $\geq 150/95$  mmHg (or isolated systolic hypertension of  $\geq 160$  mmHg).

Results of published studies were used to identify estimates of the relative risk of CVD following use of statins and anti-hypertensive medication, and after smoking cessation (see Table 6-3). Aspirin was not included in these analyses, as it is not currently licensed for primary prevention of CVD in the UK. In accordance with national guidelines, it was assumed that hypertension would be treated using ACE-inhibitors in non-black Caribbean individuals under 55 years of age and calcium channel blockers in the remainder of individuals (122).

**Table 6-3: Relative risks used in model of high-risk approach with sources**

Risk factor	Intervention	Estimated relative risk of CVD following intervention	Source(s)
Total cholesterol	Statin medication	0.75	Taylor <i>et al.</i> (117)
Anti-hypertensive medication			Both from Wright <i>et al.</i> (120) (also broadly consistent with Law <i>et al.</i> (542))
Age $\geq 55$ years or black Caribbean	Calcium-channel blocker	0.71	
Age <55 years and not black Caribbean	ACE-inhibitor	0.76	
Smoking	Smoking cessation	0.50	Derived from various sources: Doll <i>et al.</i> (543), Kenfield <i>et al.</i> (544), and Cao <i>et al.</i> (545)

ACE-inhibitor Angiotensin converting enzyme inhibitor

Risk reductions were applied multiplicatively to each individual's QRISK2-2012 score depending on their risk factor status and in accordance with national guidelines (see Table 6-4). This multiplicative approach is consistent with similar studies (109), plus it would not have been appropriate to sum the risk reductions as the total reduction could have exceeded 1. These guidelines recommend that individuals with a high cardiovascular risk score (currently  $\geq 20\%$ ) are offered statins, plus antihypertensive medication if their blood pressure is 135/85mmHg or over (121, 122); individuals with blood pressure of 150/95mmHg are offered anti-hypertensive medication regardless of their cardiovascular risk score (122). In addition, all smokers should be offered individual smoking cessation advice and interventions (546). For example, a non-smoker with a high QRISK2 score and no hypertension would receive risk reduction from statins only, a black Caribbean non-smoker with hypertension and a low QRISK2 score would receive risk reduction from a calcium channel blocker only, and an Indian smoker aged 50 years old with a high QRISK2 score and hypertension would receive risk reductions from statins, an ACE inhibitor and smoking cessation. This model included individuals who were already taking lipid-lowering medication. They received a risk reduction from statins if their total cholesterol was  $\geq 7.5$ mmol/L, with the assumption that they still had the potential to benefit from further cholesterol reduction.

Models were developed with three different uptake levels: model A assumed an uptake of 25%, model B 50% and model C 75%. These values were chosen to reflect the range of estimates of uptake of high-risk CVD prevention programmes. For instance, the Department of Health assumed that 75% of people would accept the offer of a cardiovascular screening appointment in their modelling for the NHS Health Check programme (159); in contrast, evidence from the early stages of the NHS Health Check programme found uptake of appointments of approximately 43-45% with a lower proportion taking up risk reducing interventions (162, 526).

Smoking cessation was assumed to be successful in 15% of smokers, meaning that only 15% of high-risk smokers received the relative risk reduction from smoking cessation. This assumption was based on evidence from NHS smoking cessation services, where the 1 year quit rate has been found to be in the region of 13-23%, with an estimate of 15% based on carbon monoxide measurement (123, 547).

A microsimulation approach, using random number generation, was used to allocate uptake of risk reduction interventions. Stata's random number function was used to generate a random number between 0 and 1. Each individual was then allocated to a variable of uptake / no uptake based on this number and the relative risk reductions applied accordingly (i.e. if categorised as no uptake then no risk reductions were applied). The same method was used to allocate smoking cessation success.

**Table 6-4: Possible combinations of high-risk conditions and corresponding risk reducing intervention(s)**

High-risk conditions	Possible combinations of high-risk conditions																		
High QRISK2 score ( $\geq 20\%$ )	✓	✓	✓	✓	✓	✓					✓	✓	✓	✓	✓	✓			
Hypertension																			
$\geq 135/85\text{mmHg}$ (or SBP $\geq 160\text{mmHg}$ )																			
Age $\geq 55$ years or black Caribbean			✓	✓									✓	✓					
Age $< 55$ years and not black Caribbean					✓	✓									✓	✓			
$\geq 150/95\text{mmHg}$ (or SBP $\geq 160\text{mmHg}$ )																			
Age $\geq 55$ years or black Caribbean						✓	✓									✓	✓		
Age $< 55$ years and not black Caribbean								✓	✓								✓	✓	
High total cholesterol ( $\geq 7.5\text{mmol/L}$ )								✓	✓	✓							✓	✓	✓
Smoker and successful quit attempt											✓	✓	✓	✓	✓	✓	✓	✓	✓
Eligible for statin	✓		✓		✓		✓	✓	✓	✓	✓		✓		✓		✓	✓	✓
Corresponding risk reducing intervention(s):																			
Statin	✓		✓		✓		✓	✓	✓	✓		✓		✓		✓	✓	✓	✓
Calcium channel blocker			✓	✓		✓		✓				✓	✓			✓		✓	
ACE-inhibitor					✓	✓		✓	✓					✓	✓		✓		✓
Smoking cessation											✓	✓	✓	✓	✓	✓	✓	✓	✓

SBP Systolic blood pressure; ACE-inhibitor Angiotensin converting enzyme inhibitor.

Note: Relative risk reductions applied multiplicatively; individuals not eligible for statin if taking lipid lowering drug and “low” total cholesterol.

## 6.2.6 Analyses

### 6.2.6.1 *Descriptive statistics*

Ethnic differences in demographic and cardiovascular characteristics were assessed using the independent samples t-test for continuous variables and a chi-squared test for categorical variables. In the previous chapter the general population was used as the reference group, but this group was largely made up of white people and findings from the two groups were very similar. Therefore, in these analyses only the white group was included and was used as the reference group.

Graphs were plotted to allow visual assessment of ethnic differences in the distribution of the continuous cardiovascular risk factors. The distribution of total cholesterol, systolic blood pressure and body mass index was plotted for each ethnic group, and compared to the white group. In addition, graphs were produced to compare the distribution of these risk factors in high-risk individuals compared to the overall population in each ethnic group.

### 6.2.6.2 *Impact of population and high-risk approaches*

The potential impact of the population and high-risk models described above was calculated with the sample divided by ethnic group and sex. Mean QRISK-2012 score with current risk factors was calculated for each ethnic group and sex. The population and high-risk models were then applied in turn and the mean QRISK2-2012 score recalculated. For the high-risk model this process was repeated 10 times and the results averaged in order to address variability in the results arising from the use of random number generation in determining uptake.

The QRISK2-2012 score was used to estimate the number of cardiovascular events that would occur in 10 years, scaled to a population of 100,000. For example, a mean QRISK2-2012 score of 12% would equate to 12,000 predicted cardiovascular events per 100,000 population in 10 years. This approach of using a cardiovascular risk calculator to estimate future cardiovascular events, which has been used elsewhere (108), was chosen as it was possible to apply a risk calculator to the cross-sectional data in the HSE. An alternative approach could have been to use prospective cohort data, like Emberson et al (109). This would have allowed specific risk factor coefficients to be calculated from the study population using baseline risk factor and cardiovascular incident data, however a

suitable study with sufficient numbers of ethnic minority individuals could not be identified.

Comparison of the demographic characteristics of the HSE sample with 2001 Census data revealed small differences in age distribution. In particular, the mean age of HSE participants tended to be lower than the mean age from 2001 Census data, especially for Pakistani and Bangladeshi males. Unlike the analyses in Chapters 4 and 5, these differences were particularly relevant to these analyses because they involved scaling the results to a population level to make inferences about the number of cardiovascular events that could occur. Therefore, direct age standardisation was used to adjust the results. The sample was divided into 5-year age categories and the mean QRISK2-2012 score was calculated for each age group, ethnic group and sex. 2001 Census data for England and Wales, broken down by ethnic group, age and sex (548), was used as the reference population. The number of predicted cardiovascular events, calculated from the mean QRISK2-2012 scores, was then directly age standardised against the reference population (see Figure 6-1).

Age group (years)	Number of predicted events per 100,000 population (A)	Percentage of reference population in age group (B)	A × B
35-39	100	20	2,000
40-44	1,000	20	20,000
45-49	2,000	18	36,000
50-54	3,000	16	48,000
55-59	4,000	14	56,000
60-64	5,000	12	60,000
65-69	6,000	10	60,000
70-74	7,000	8	56,000
		Total/100	338,000/100
→ Age standardised number of predicted events per 100,000 population			3,380

**Figure 6-1: Illustration of method of direct age standardisation (based on (549))**

### **6.2.6.3 Impact of population and high-risk approaches on health inequalities**

Changes in absolute inequalities between the ethnic groups following application of the population and high-risk models were assessed. The absolute difference in the number of predicted cardiovascular events between each ethnic minority

group and the white group was calculated at baseline and then following each population and high-risk model.

#### **6.2.7 Statistical software**

Analyses were carried out using Stata 12.1 and Microsoft Excel.

## 6.3 Results

### 6.3.1 Demographic and cardiovascular characteristics

These analyses were based on 7,929 white, 639 black Caribbean, 659 Indian, 704 Pakistani/Bangladeshi and 741 Irish individuals (see Table 6-5). Compared with the white group, the black Caribbean, Indian and Pakistani/Bangladeshi groups were significantly younger and there were significantly fewer men in the black Caribbean and Irish groups. Ethnic differences in cardiovascular characteristics were similar to those observed in the previous chapter. Compared to the white group, systolic blood pressure was significantly lower in the Indian, Pakistani/Bangladeshi and Irish groups, total cholesterol was significantly lower in all the ethnic minority groups, and body mass index was significantly higher in the black Caribbean group but lower in the Indian and Irish groups. Lower proportions of the black Caribbean, Indian and Pakistani/Bangladeshi groups were current smokers.

Visual assessment of ethnic differences in the distribution of continuous cardiovascular risk factors revealed slight differences. Compared to the white group, the distribution of body mass index was shifted slightly to the right in the black Caribbean group but was similar in the other ethnic minority groups (see Figure 6-2). The distribution of total cholesterol values was shifted slightly towards the left in all of the ethnic minority groups compared to the white group (see Figure 6-3). The distribution of systolic blood pressure was similar between the white and ethnic minority groups. Visual assessment of the data from complete cases only produced similar observations to those from the imputed data.

In each ethnic group, the distribution of systolic blood pressure, total cholesterol and body mass index values in high-risk individuals was shifted to the right in comparison with the overall distribution (Figure 6-4 shows systolic blood pressure; similar findings were noted for total cholesterol and body mass index).

### 6.3.2 Population approach

With current risk factors, the number of predicted cardiovascular events in the next 10 years per 100,000 population was highest in Pakistani/Bangladeshi, Irish and Indian men (19,939, 14,593 and 14,161 events, respectively) (see Table 6-6). Applying each of the population based reductions in cardiovascular risk factors

separately prevented varying numbers of cardiovascular events. Reducing total cholesterol by 1.0 mmol/L prevented the highest number of events, particularly in Pakistani/Bangladeshi, Indian and Irish men. Reducing smoking status by one category prevented few cardiovascular events in Indian and Pakistani/Bangladeshi women, whilst preventing a higher number of events in men from these ethnic groups. In contrast, systolic blood pressure reduction prevented similar numbers of events in men and women from all ethnic groups. A combined population approach could prevent up to 2,071 events per 100,000 white men and 1,176 events per 100,000 white women (see Table 6-7). The highest number of events could be prevented in the Pakistani/Bangladeshi group where up to 3,487 events per 100,000 men and 1,753 events per 100,000 women could be prevented. Higher estimated numbers of events prevented were also observed for Indian men (up to 2,491 events per 100,000 population) and Irish men (up to 2,374 events per 100,000 population). Fewer events would be prevented in the black Caribbean group than the white group where up to 1,515 events per 100,000 men and 1,037 events per 100,000 women could be prevented. The lowest number of events prevented was in Indian women (up to 989 events per 100,000 population). A combined population approach with moderate risk factor reductions (model 2) would prevent a similar number of events in the Pakistani/Bangladeshi group than an approach with higher risk factor reductions (model 3) in the white group. In all ethnic groups, fewer events were prevented in women than men.

### **6.3.3 High-risk approach**

The high-risk approach was estimated to prevent up to 1,638 events per 100,000 white men and 751 events per 100,000 white women over ten years (see Table 6-7). The highest estimated numbers of events prevented were for Pakistani/Bangladeshi men (up to 2,698 events per 100,000 population) and Irish men (up to 1,979 events per 100,000 population). The lowest number of cardiovascular events prevented was observed in the black Caribbean group, where up to 935 events per 100,000 men and 469 events per 100,000 women could be prevented. A high-risk approach with moderate uptake (model B) would prevent a similar number of events in the Pakistani/Bangladeshi group than an approach with higher uptake (model C) in the white group. Similar to the

population approach, fewer events were prevented in women than men in all ethnic groups.

#### **6.3.4 Population versus high-risk approaches**

In all ethnic groups, and in both men and women, population approaches prevented more events than high-risk approaches (see Table 6-7). The largest difference in the number of cardiovascular events that could be prevented by the population and high-risk approaches occurred in Pakistani/Bangladeshi individuals and Indian men. In women from all ethnic groups and black Caribbean men, a high-risk approach with 75% uptake (model C) prevented fewer cardiovascular events than a population approach with moderate risk factor reductions (model 2). The number of events prevented by a high-risk approach with 75% uptake (model C) was intermediate between that prevented by population approaches with moderate and high risk factors reductions (models 2 and 3) in Indian, Pakistani/Bangladeshi, Irish and white men.

#### **6.3.5 Health inequalities**

In Indian, Pakistani/Bangladeshi, and Irish men both the population and high-risk approaches reduced the absolute difference in the predicted number of events per 100,000 population compared to the white group (see Table 6-8 and Figure 6-5). However, the population approach reduced this difference to a greater extent than the high-risk approach in Pakistani/Bangladeshi and Indian men. In black Caribbean men the population and high-risk approaches also reduced the absolute difference in the predicted number of events per 100,000 population compared to the white group, although this occurred in the opposite direction to the other ethnic minority groups.

Reductions in the absolute difference in the predicted number of events between the ethnic minority and white group were more modest in women compared to men. In Pakistani/Bangladeshi and Irish women both the population and high-risk approaches reduced the absolute difference in the predicted number of events per 100,000 population compared to the white group, although to a slightly greater extent following the population approach. In black Caribbean and Indian women the population and high-risk approaches resulted in similar, small reductions in the difference in the predicted number of events compared to the white group, but in the opposite direction to Pakistani/Bangladeshi and Irish women.

**Table 6-5: Demographic and cardiovascular characteristics by ethnic group**

	White n=7,929	Black Caribbean n=639	Indian n=659	Pakistani / Bangladeshi n=704	Irish n=741
Age	Mean (SE) 51.5 (0.1)	Mean (SE) 49.8 (0.5)	Mean (SE) 48.7 (0.4)	Mean (SE) 47.4 (0.4)	Mean (SE) 51.2 (0.4)
p-value	-	<0.001	<0.001	<0.001	0.524
Systolic blood pressure	130.2 (0.2)	129.4 (0.9)	126.4 (0.8)	125.6 (0.9)	128.0 (0.9)
p-value	-	0.364	<0.001	<0.001	0.009
Total cholesterol	5.9 (0.0)	5.4 (0.1)	5.5 (0.1)	5.5 (0.1)	5.7 (0.1)
p-value	-	<0.001	<0.001	<0.001	0.001
Body mass index	27.6 (0.1)	28.6 (0.2)	27.1 (0.2)	27.7 (0.2)	27.0 (0.2)
p-value	-	<0.001	0.009	0.809	0.002
Male	n (%) 3,806 (48.0)	n (%) 263 (41.2)	n (%) 297 (45.1)	n (%) 342 (48.6)	n (%) 306 (41.4)
p-value	-	0.001	0.155	0.783	<0.001
Smoking status					
Non-smoker	3,305 (41.7)	373 (58.4)	507 (77.0)	511 (72.6)	292 (39.4)
Ex-smoker	2,680 (33.8)	134 (21.0)	70 (10.7)	62 (8.7)	258 (34.9)
Light smoker	387 (4.9)	72 (11.3)	47 (7.1)	54 (7.6)	47 (6.3)
Moderate smoker	787 (9.9)	45 (7.0)	28 (4.2)	54 (7.7)	75 (10.1)
Heavy smoker	770 (9.7)	15 (2.3)	7 (1.1)	24 (3.4)	69 (9.3)
p-value	-	<0.001	<0.001	<0.001	0.395

SE standard error

Reference group for p-values is white group

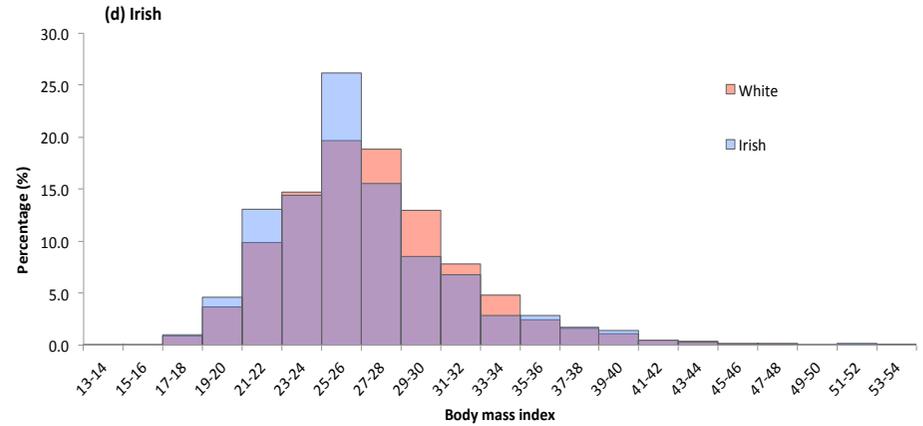
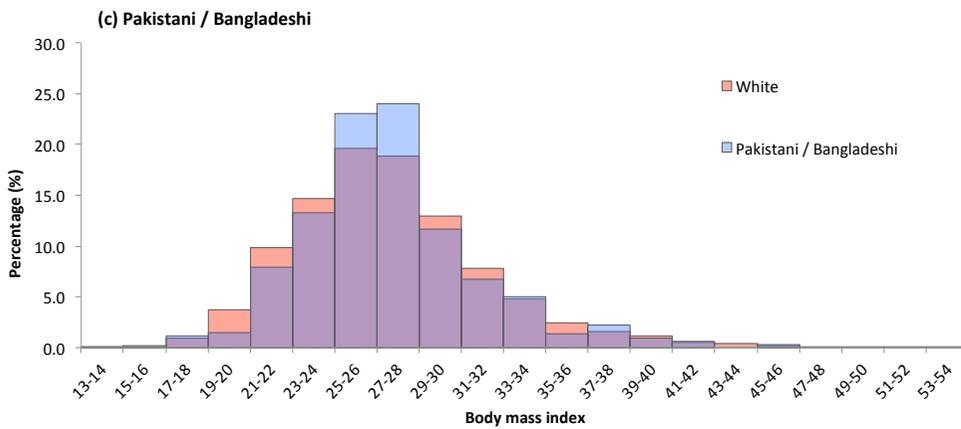
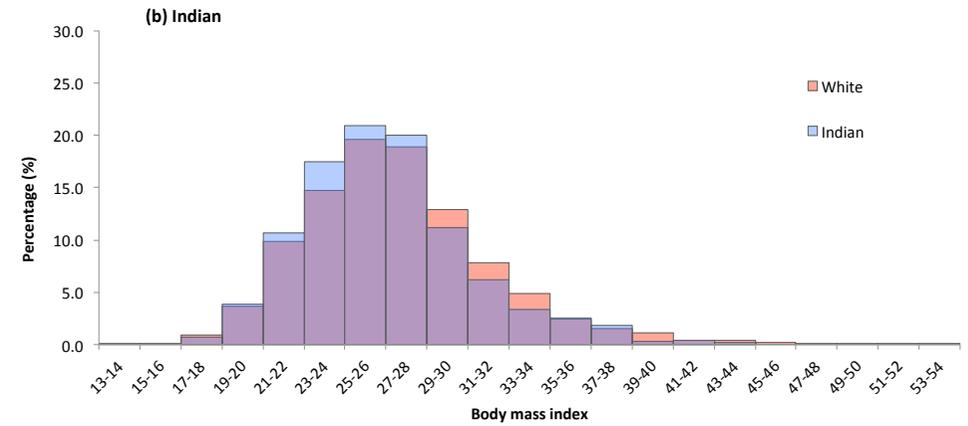
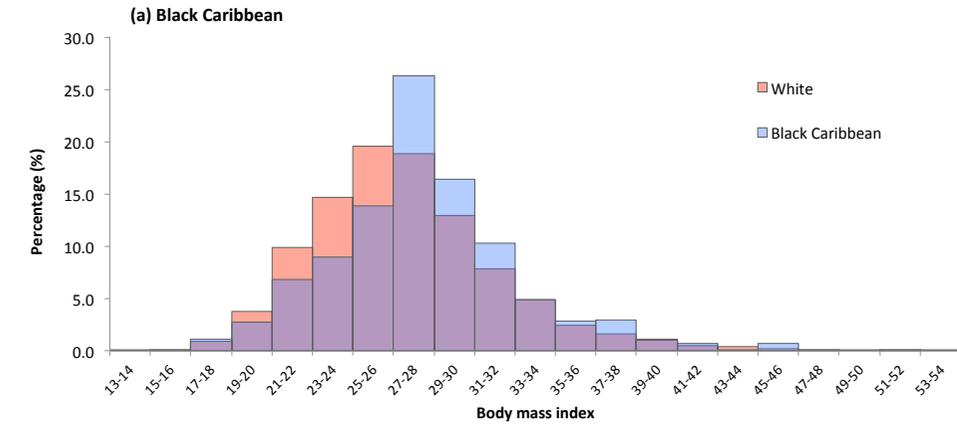
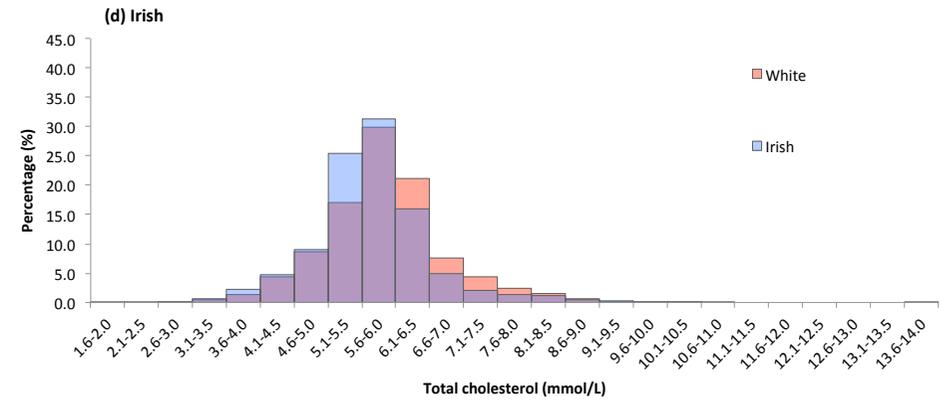
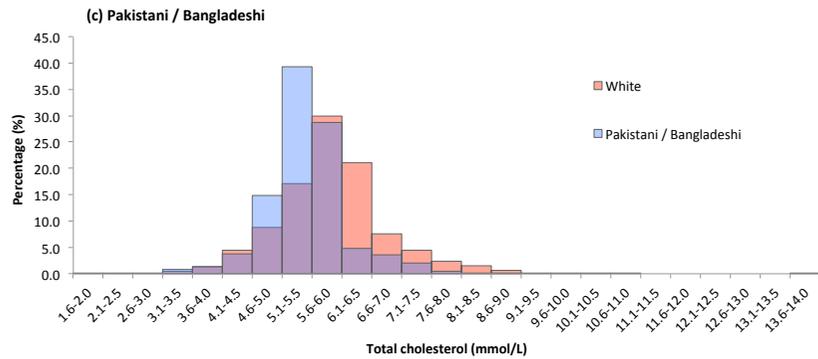
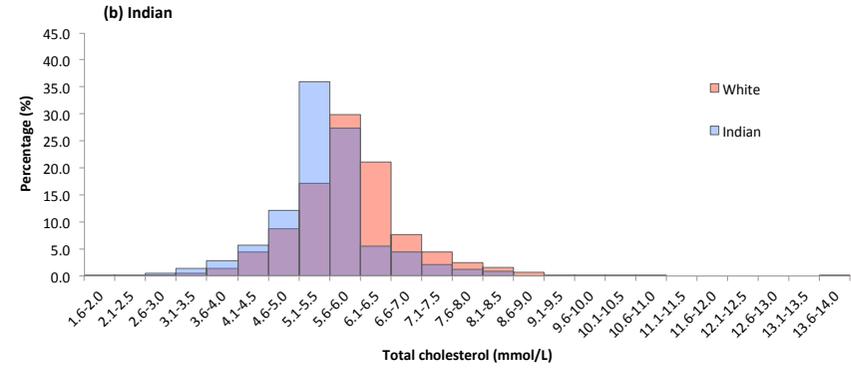
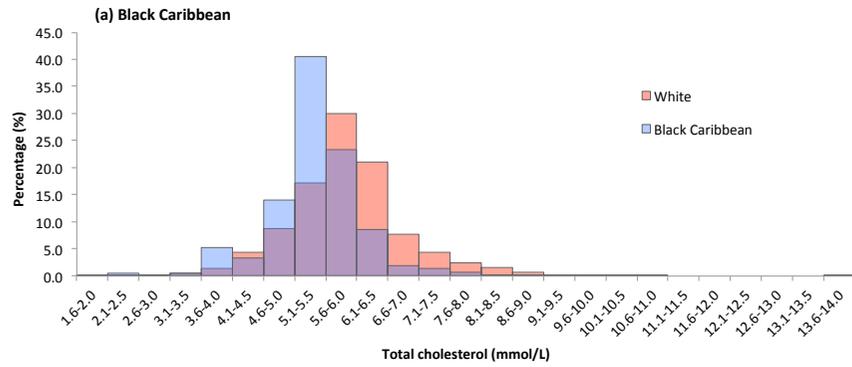
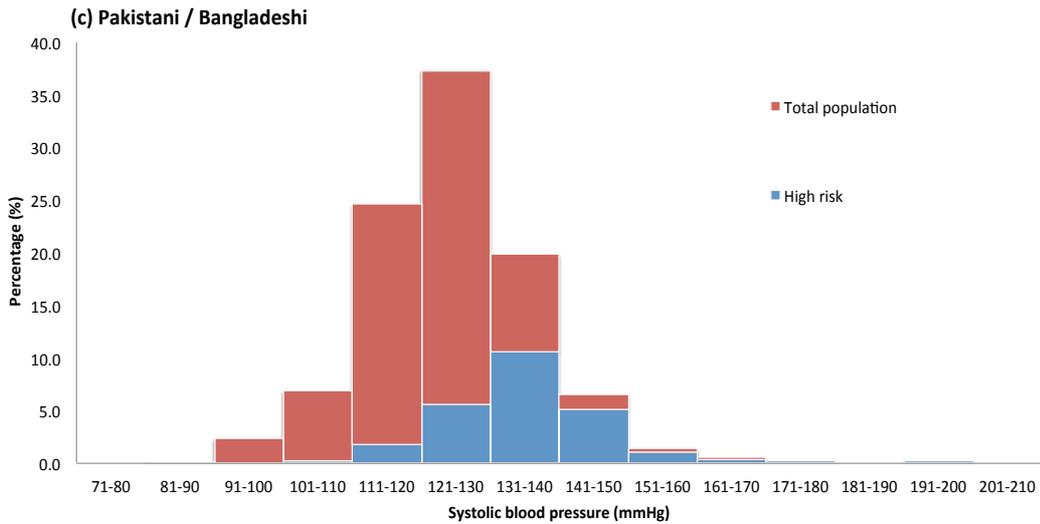
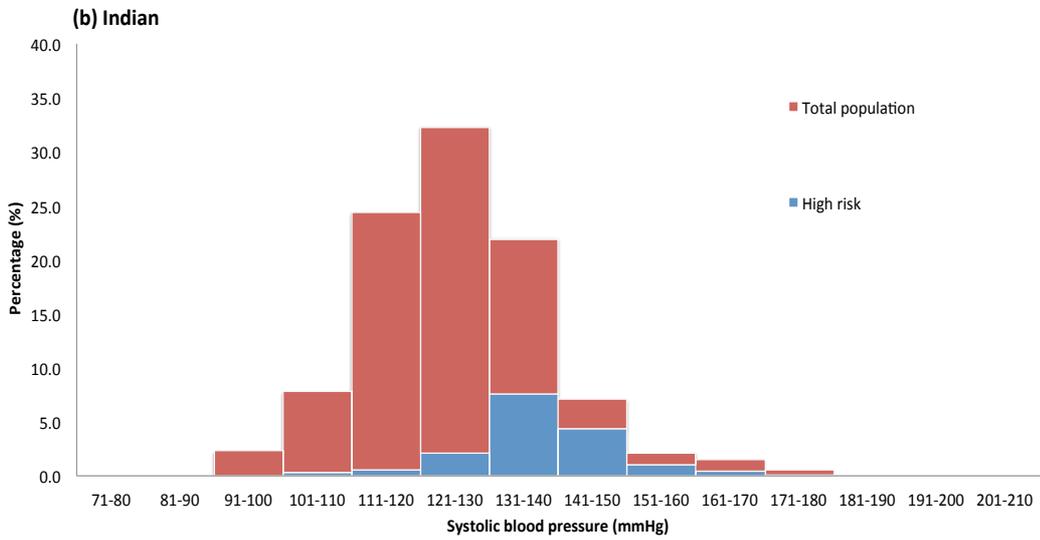
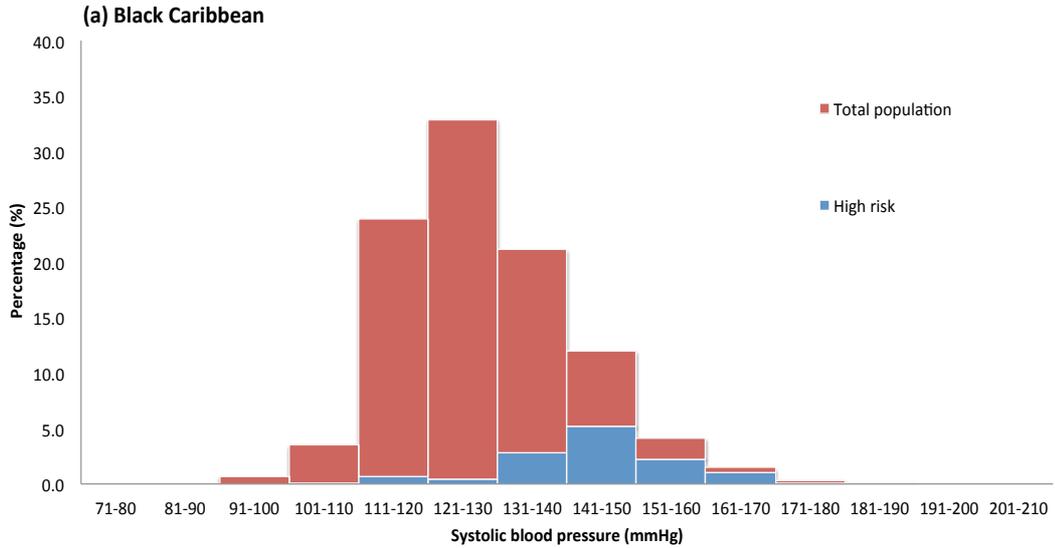
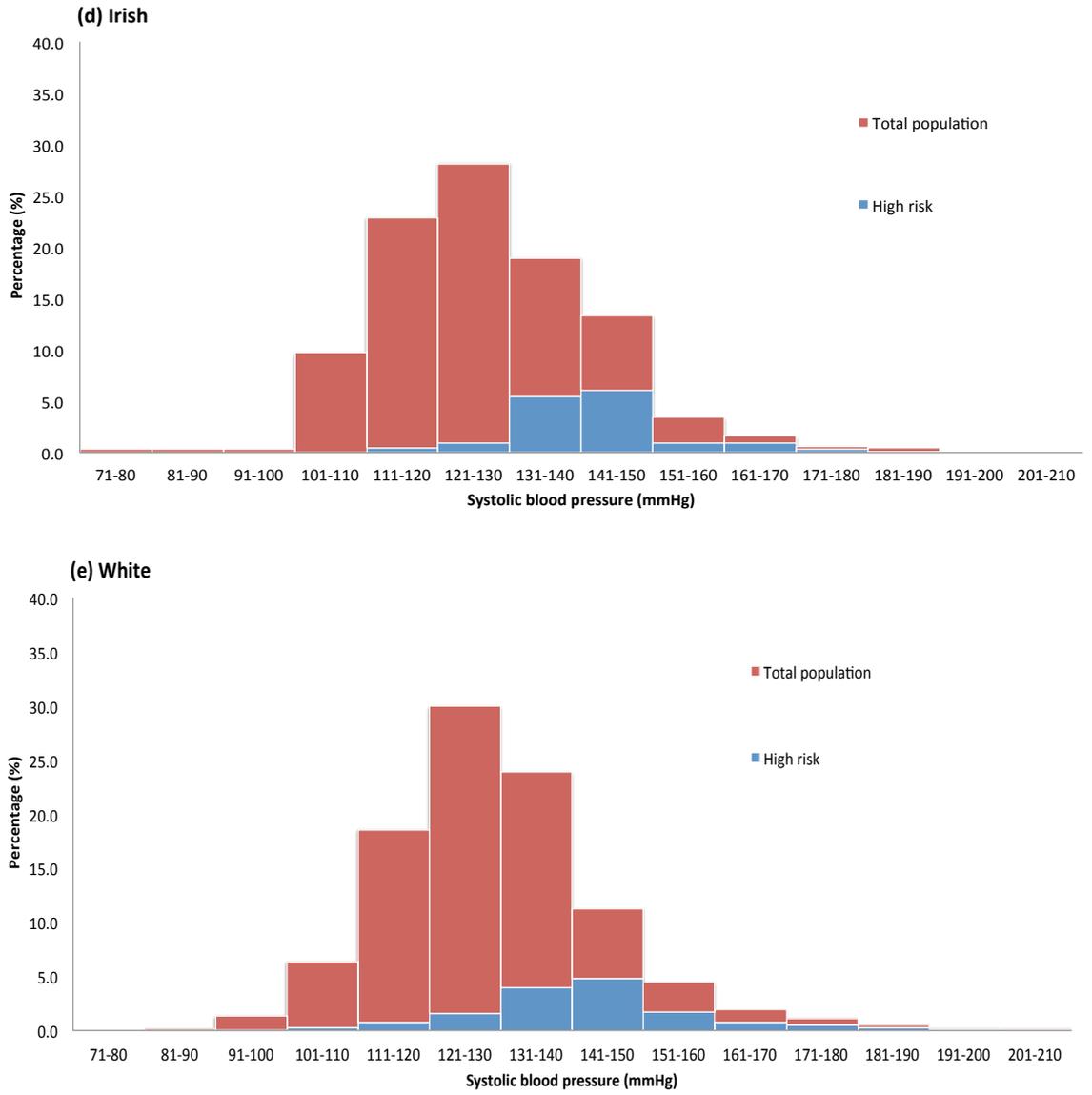


Figure 6-2: Distribution of body mass index in ethnic minority group compared to white group



**Figure 6-3: Distribution of total cholesterol concentration in ethnic minority group compared to white group**





**Figure 6-4: Distribution of systolic blood pressure in total population and high-risk individuals by ethnic group**

**Table 6-6: Age standardised number of cardiovascular events and events prevented following cholesterol, systolic blood pressure, body mass index and smoking reductions**

	Black Caribbean		Indian		Pakistani / Bangladeshi		Irish		White	
	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
Number of cardiovascular events predicted over 10 years per 100,000 population based on current risk factors	9,280	7,073	14,161	6,832	19,939	12,188	14,593	9,372	12,722	7,948
Number of cardiovascular events prevented over 10 years per 100,000 population										
Cholesterol reduction										
0.1 mmol/L	81	58	139	58	180	99	140	73	122	61
0.5 mmol/L	444	291	681	285	907	487	679	361	603	303
1.0 mmol/L	879	568	1,341	553	1,797	956	1,328	709	1,180	596
Systolic blood pressure reduction										
2.5 mmHg	100	109	169	113	229	202	152	128	141	113
5.0 mmHg	199	216	335	225	455	399	303	254	280	227
7.5 mmHg	287	321	500	334	678	594	452	365	418	333
Body mass index reduction										
0.5 kg/m <sup>2</sup>	42	17	78	20	106	40	63	16	60	17
1.5 kg/m <sup>2</sup>	125	52	236	61	317	123	185	48	181	51
2.5 kg/m <sup>2</sup>	209	86	391	101	523	205	302	77	301	83
Smoking reduction										
Decrease of 1 category	229	120	416	48	701	87	416	342	287	230

**Table 6-7: Age standardised number of cardiovascular events and events prevented following population and high-risk approaches**

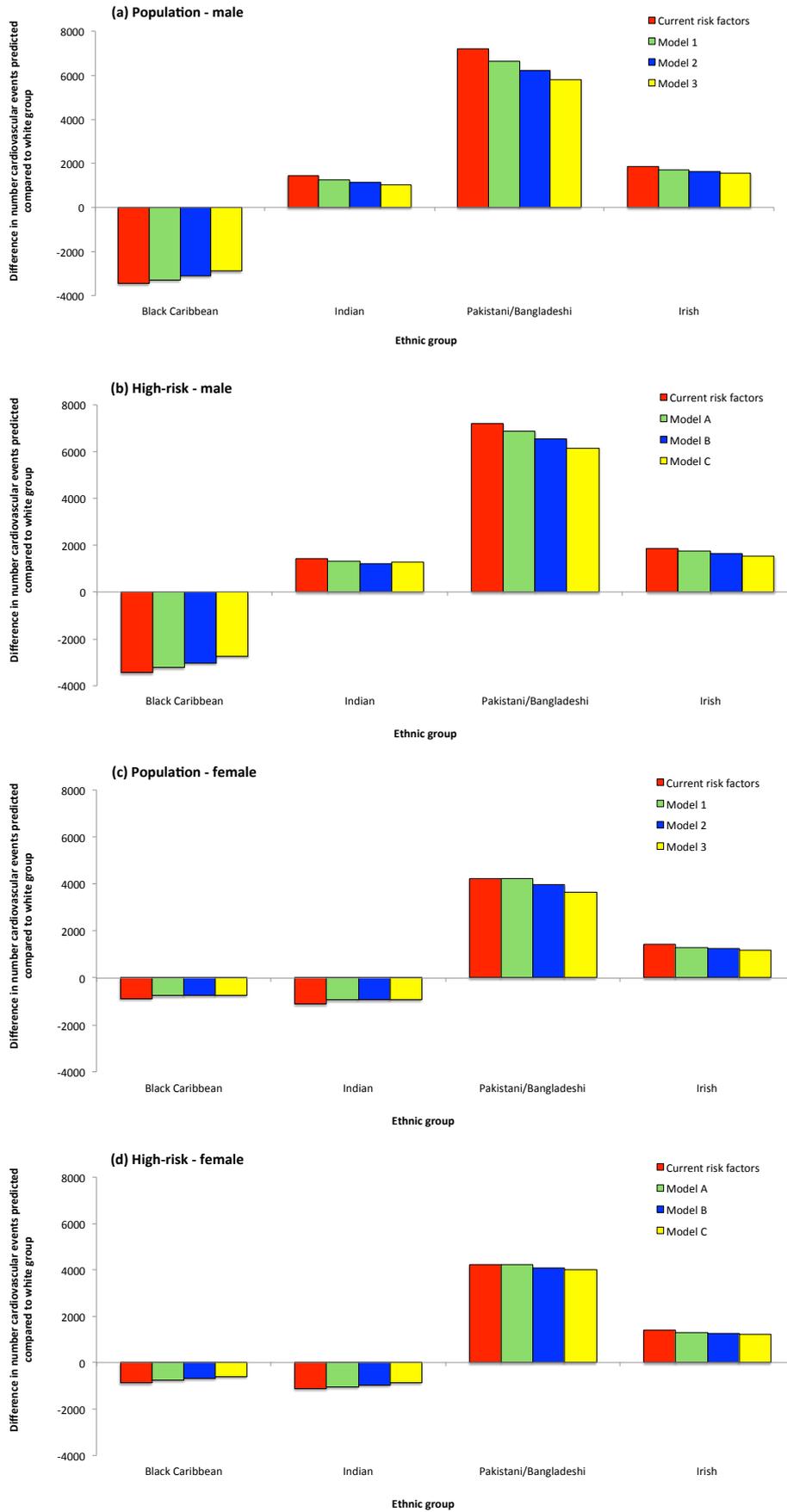
	Black Caribbean		Indian		Pakistani / Bangladeshi		Irish		White	
	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
Number of cardiovascular events predicted over 10 years per 100,000 population based on current risk factors										
	9,280	7,073	14,161	6,832	19,939	12,188	14,593	9,372	12,722	7,948
Number of cardiovascular events prevented over 10 years per 100,000 population										
Population approach										
Model 1	443	298	786	237	1,193	421	759	550	599	414
Model 2	958	653	1,600	601	2,284	1,062	1,529	969	1,302	781
Model 3	1,515	1,037	2,491	989	3,487	1,753	2,374	1,417	2,071	1,176
High-risk approach										
Model A	330	129	658	180	898	275	675	383	553	256
Model B	662	302	1,295	339	1,766	653	1,291	657	1,085	498
Model C	935	469	1,802	497	2,698	982	1,979	956	1,638	751

Population approach: model 1 applied reductions of 2.5 mmHg systolic blood pressure, 0.1 mmol/L total cholesterol, and 0.5 kg/m<sup>2</sup> body mass index; model 2 applied reductions of 5.0 mmHg systolic blood pressure, 0.5 mmol/L total cholesterol, and 1.5 kg/m<sup>2</sup> body mass index; and model 3 applied reductions of 7.5 mmHg systolic blood pressure, 1.0 mmol/L total cholesterol, and 2.5 kg/m<sup>2</sup> body mass index. In each model smoking status was also reduced by one category. High-risk approach: 25% uptake in model A; 50% uptake in model B; and 75% uptake in model C.

**Table 6-8: Difference in predicted number of cardiovascular events over 10 years per 100,000 population between ethnic minority and white group**

	Black Caribbean		Indian		Pakistani / Bangladeshi		Irish	
	Men	Women	Men	Women	Men	Women	Men	Women
Current risk factors	-3,443	-875	1,438	-1,116	7,217	4,240	1,870	1,424
Population approach								
Model 1	-3,287	-759	1,251	-939	6,623	4,233	1,711	1,289
Model 2	-3,099	-747	1,140	-936	6,235	3,959	1,644	1,236
Model 3	-2,886	-736	1,018	-929	5,801	3,663	1,568	1,183
High-risk approach								
Model A	-3,220	-748	1,333	-1,040	6,871	4,221	1,748	1,297
Model B	-3,020	-678	1,228	-957	6,536	4,085	1,665	1,266
Model C	-2,740	-593	1,274	-862	6,157	4,009	1,530	1,219

Population approach: model 1 applied reductions of 2.5 mmHg systolic blood pressure, 0.1 mmol/L total cholesterol, and 0.5 kg/m<sup>2</sup> body mass index; model 2 applied reductions of 5.0 mmHg systolic blood pressure, 0.5 mmol/L total cholesterol, and 1.5 kg/m<sup>2</sup> body mass index; and model 3 applied reductions of 7.5 mmHg systolic blood pressure, 1.0 mmol/L total cholesterol, and 2.5 kg/m<sup>2</sup> body mass index. In each model smoking status was also reduced by one category. High-risk approach: 25% uptake in model A; 50% uptake in model B; and 75% uptake in model C.



**Figure 6-5: Difference in predicted number of cardiovascular events over 10 years per 100,000 population between ethnic minority and white group following population and high-risk approaches**

## 6.4 Discussion

### 6.4.1 Principal findings

Both population and high-risk approaches to primary prevention of CVD were found to have the potential to prevent significant numbers of cardiovascular events, although there were ethnic differences in the number of cardiovascular events that could be prevented. Higher estimated numbers of events could be prevented in Pakistani/Bangladeshi individuals, and Indian and Irish men, reflecting the higher cardiovascular risk and number of high-risk individuals in these groups and, therefore, their higher baseline number of predicted events. In ethnic groups with comparatively lower cardiovascular risk, such as the black Caribbean group, the population and high-risk approaches prevented fewer events than in ethnic groups with higher cardiovascular risk. In Pakistani/Bangladeshi individuals, population or high-risk approaches with moderate risk factor reduction or moderate uptake could prevent similar numbers of events than approaches with high risk factor reduction or high uptake in the white group. When population risk factor reductions were modelled separately there were ethnic differences in the number of cardiovascular events prevented. In particular, compared to white women smoking reduction prevented fewer events in Indian and Pakistani/Bangladeshi women. This could reflect lower prevalence of smoking in women from these ethnic minority groups.

In all ethnic groups the population approach prevented a larger number of events than the high-risk approach, especially in Pakistani/Bangladeshi individuals and Indian men. Indeed a population approach that achieved only moderate risk factor reduction could prevent a similar number of events as a high-risk approach with high uptake in women from all ethnic groups and in black Caribbean men.

Both population and high-risk approaches showed the potential to reduce inequalities in the number of cardiovascular events compared to the white group, but the results varied by ethnic group and sex. Health inequalities fell more noticeably among men than women, particularly following the population approach. However, in the black Caribbean group and in Indian women this was not in a favourable direction for either approach. This could be explained by the

lower baseline cardiovascular risk in these groups compared to the white group, in which higher numbers of events would be prevented. Despite this, these findings suggest that comprehensive CVD prevention programmes have the potential to reduce ethnic health inequalities and the reduction will be greatest for population approaches.

#### **6.4.2 Strengths and limitations**

The strengths and limitations related to the use and calculation of QRISK2-2012, use of multiple imputation to address missing data, and the sample size and age of the HSE data that were discussed in the previous chapter are also relevant to these analyses (see Chapter 5). Similar to the previous analyses, it was not possible to accurately exclude individuals who were taking lipid-lowering medication. Whilst it may have been preferable to exclude these individuals, the high-risk model was adapted to apply appropriate risk reductions to this group.

This modelling study required a number of assumptions to be made that might have affected the size of impact estimated. Assumptions were made regarding the size of potential risk factor reductions following population approaches, relative risk reductions from use of statins, anti-hypertensives and smoking cessation, and of uptake in the high-risk approach. Wherever possible these assumptions were based on existing evidence. A number of the assumptions, in particular the risk reductions that could be achieved by statins and anti-hypertensive medications or from salt reduction, were based on high quality evidence from systematic reviews and meta-analyses (117, 120, 427). However, it should be noted that risk reductions from high-risk interventions achieved in clinical trial settings might not be achievable in practice. In contrast, evidence for the size of population reductions in cholesterol came from real life scenarios where population wide changes had been achieved (104, 532, 533). Evidence for achievable population changes in body mass index was limited. Body mass index differs from the other cardiovascular risk factors modelled in that it is currently increasing at a population level - this could explain the relative lack of evidence on beneficial population changes in this risk factor and make the reductions modelled even more challenging. The risk distribution of body mass index may follow a J-shaped curve (550), a difference that may not have been fully considered given the way in which the QRISK2-2012 calculator deals with body mass index values. In addition, the categorical nature of smoking status limited

the changes that could be made to this variable. Nevertheless, the assumptions made regarding risk factor changes and risk reductions were consistent with those used in comparable modelling studies (108, 109, 111).

The uncertainty inherent in estimating the size of these risk changes was addressed by modelling a range of risk factor reductions for the population approach. For the high-risk approach a range of uptake levels was assessed, whilst the risk reductions remained constant. These levels were chosen to reflect the uncertainty surrounding the uptake that can be achieved in high-risk CVD prevention programmes. In this analysis the estimated uptake, e.g. of 25%, represented the uptake of screening and interventions but then assumed 100% adherence to the risk reducing interventions. Therefore, whilst a 25% uptake level may appear low, if considered against less than optimal adherence it may reflect a realistic level of take up of risk reducing interventions in high-risk individuals. Indeed, Cochrane *et al* found that 29.8% of individuals invited for an NHS Health Check took up risk reducing interventions (526), although this figure is based on the performance of the programme in a single area of England and estimates of uptake, including by socioeconomic position and ethnic group, remain uncertain. Similar to the previous chapter, estimates of uptake and assumptions regarding adherence were not varied between ethnic groups, given that evidence for ethnic differences in both is mixed (see section 3.7.7.1). Uptake (alongside access to healthcare) is an essential component of a high-risk approach. If uptake was reduced in ethnic minority groups compared to the white group these analyses could have overestimated the potential of high-risk approaches to reduce cardiovascular events and health inequalities in these groups. However, evidence from the NHS Health Check programme suggests that uptake of screening may not be reduced in ethnic minority groups (162), whilst other evidence indicates that use of primary care services is not lower in ethnic minority groups compared to white individuals (378). Whilst low uptake would limit the effectiveness of high-risk interventions, the high-risk approach enables programmes to be tailored to the needs of local populations, such as ethnic minority communities (551), potentially improving the uptake of appropriately designed programmes. It was beyond the scope of these analyses to incorporate ethnic differences in the design of the high-risk approaches, and as the aim was to model the differential impact of programmes for the general population this would not have been relevant.

The results of this analysis are presented in terms of absolute changes in cardiovascular events and ethnic inequalities. The use of calculations of relative changes was considered, for example calculation of percentage reductions in events or ratios for changes in ethnic inequalities. However, it was decided to focus on absolute changes as these reflect the potential public health impact of population and high-risk approaches. Future analyses could benefit from the addition of calculations of relative changes.

Previous analysis of population and high-risk approaches to CVD prevention corrected for regression dilution bias, and emphasised the importance of doing this so that the impact of the population approach is not underestimated (109). Regression dilution bias can occur when single measurements of cardiovascular risk factors, such as blood pressure and cholesterol, are used but are affected by random measurement error and within-person variability (109, 110, 552). This can lead to a reduction in the observed association between exposure and outcome, and in Emberson *et al*'s analysis to an underestimation of the impact of population (but not high-risk) approaches (109). Emberson *et al* were able to correct for regression dilution bias using prospective data. However, this was not possible in these analyses owing to the use of cross-sectional, i.e. single measurement, data. Therefore, it is possible that the potential impact of the population approach has been underestimated in these analyses.

Further underestimation of the impact, particularly of the population approach, could have arisen from the age range that was included in this analysis (35-74 years old). This range was used as it reflects the age range that QRISK2 was derived from and can therefore be applied to. However, this means that the potential benefits of the population approach in both younger and older ages, and the high-risk approach in older ages (it is highly unlikely that younger individuals would be classed as high-risk), would have been missed from these results.

#### **6.4.3 Relations to other studies**

These findings are consistent with previous studies, in terms of population approaches having the potential to prevent more events than a high-risk strategy (108, 109). Evidence suggests that population approaches have the potential to prevent large numbers of CVD events and save millions of pounds for the NHS (101), whilst high-risk approaches may also have a positive, but smaller, impact

on health (105, 106). Modelling studies have sought to directly compare the potential for population and high-risk approaches to prevent CVD, using similar methods to these analyses. These studies have produced mixed results. On the one hand, Emberson *et al* and Cooney *et al* both found that population approaches could prevent more CVD events than high-risk approaches (108, 109). In contrast, studies by Zulman *et al* and Manuel favoured high-risk approaches (111, 112). As discussed in Chapter 2, these discrepancies may be accounted for by methodological differences, for instance in the size of the risk reductions modelled. Manuel compared a population wide reduction in cholesterol of 2% with a high-risk approach with 100% adherence to statins; Emberson *et al* modelled population reductions of 5-15% in cholesterol and blood pressure against a high-risk approach using statins, aspirin and anti-hypertensive medication. The risk reductions modelled in these analyses are more consistent with those used by Emberson *et al* than by Manuel, perhaps explaining why these analyses also found that population approaches had greater potential to prevent CVD events than the high-risk approach. However, whilst utilising similar methods to these studies, these analyses differed because they explored ethnic differences in the potential impact of these programmes.

There is evidence of a range of ethnic differences (and similarities) in cardiovascular risk factors (see section 3.5). The findings of these analyses suggest that differences in the prevalence of certain risk factors, in particular smoking, may affect the potential impact of population approaches to CVD prevention. If it were the case that health policy solely focused on smoking cessation, groups such as Pakistani/Bangladeshi women, who are otherwise at high cardiovascular risk, may benefit less in terms of overall CVD prevention than groups with higher prevalence of smoking. However, in the combined population model, the results of which would have reflected ethnic differences in smoking status, Pakistani/Bangladeshi women still showed greater potential to benefit from a population approach than a high-risk approach. In contrast, the models did not incorporate ethnic differences in health behaviours, such as salt intake and fat consumption. Evidence suggests that there may be ethnic differences in salt intake, with higher consumption of salt from processed food among white people (424). In addition, there may be ethnic differences in blood pressure response to salt reduction, with evidence of larger falls in black compared to white people (131). A population wide policy of reducing hidden

salt in processed food could therefore have a differential effect across ethnic groups, for example by lowering blood pressure more in black individuals through increased physiological response or in white individuals who consume more processed food. Likewise, ethnic differences in the consumption of industrially produced trans fatty acids could lead to differences in the impact of eliminating them from the food chain, with potentially greater benefit for ethnic minority groups who consume them more (425). Despite this evidence, it was difficult to quantify these ethnic differences to the extent that they could be incorporated into these analyses. However, it is possible that population policies could lead to additional ethnic differences in impact beyond those considered here.

Ethnic minority groups in the UK experience higher levels of socioeconomic deprivation than the majority white population (308, 309). Socioeconomic position is a risk factor for CVD and is incorporated into the QRISK2-2012 calculator, so some of these socioeconomic differences will be reflected in the results. In addition, however, it has been suggested that high-risk approaches may systematically disadvantage socioeconomically deprived populations and may lead to an increase in inequalities (4, 5), perhaps because of lower access to or uptake of high-risk interventions in socioeconomically deprived populations (206, 208). Population strategies have been favoured from an equity perspective (5), although the type of population intervention adopted could affect its impact on health inequalities (204). Given higher levels of socioeconomic deprivation, the impact of population and high-risk approaches on socioeconomic health inequalities is likely to be particularly applicable to ethnic minority populations. The complex relationships that determine the impact of a particular population or high-risk intervention on socioeconomic health inequalities will not have been fully considered in these analyses and it remains possible that both approaches may have additional impacts on health inequalities; in particular, the benefits of the population approach on health inequalities may have been underestimated in this study.

#### **6.4.4 Implications**

Deciding on the optimum balance between population and high-risk approaches to primary prevention of CVD continues to be challenging for academics, policy makers and politicians. Despite evidence that both of these approaches are

needed, and that the population approach may be a particularly effective way of preventing disease, recent policy has favoured the high-risk approach. These findings reinforce the need to supplement high-risk CVD prevention with comprehensive and effective population approaches, such as salt reduction legislation. Although this may be a difficult political choice, this approach has the potential to prevent significant numbers of cardiovascular events and to reduce ethnic inequalities in health.

These results may be partially generalisable to ethnic minority populations in other settings, if they have comparable cardiovascular characteristics. The finding that comprehensive population and high-risk approaches to CVD prevention can prevent significant amounts of CVD and reduce health inequalities, particularly in ethnic groups at high risk of CVD, is applicable to many populations. However, the absolute benefits of these approaches will depend on the population's baseline risk. Further, alternative cardiovascular risk calculators will need to be used in other populations because of differences in baseline risk of CVD.

#### **6.4.5 Next steps**

The need to tackle health inequalities means that it is essential that the impact of public health interventions on high-risk sections of the population be considered. These analyses investigated ethnic differences in the potential impact of population and high-risk approaches to disease prevention, but the impact may also vary across other axes of inequality, particularly socioeconomic position.

## 7 Chapter 7: General discussion

### 7.1 Overview

This thesis explored ethnic differences in two CVD prevention policy choices - the choice between mass and targeted screening for high cardiovascular risk, including the use of area deprivation measures to target screening, and the choice between population and high-risk approaches. The findings suggest that area deprivation measures may be both effective and efficient at identifying individual deprivation in ethnic minority groups, and cardiovascular screening programmes targeted at deprived areas may be particularly cost-effective in ethnic minority groups that have a high risk of CVD. In addition, both population and high-risk approaches to CVD prevention, especially the population approach, were found to have significant potential to prevent CVD and reduce ethnic health inequalities, particularly in ethnic groups at high risk of CVD.

These findings could be explained by ethnic differences in individual and area deprivation, alongside differences in CVD and cardiovascular risk factors. For instance, the black Caribbean population in the UK experiences relatively high levels of individual socioeconomic deprivation, and is concentrated into socioeconomically deprived areas but has a lower overall risk of CVD than the white population, despite an increased risk of stroke (250, 259, 308, 309, 438). This accounts for why area deprivation measures performed effectively at identifying individual deprivation in the black Caribbean group, but targeting cardiovascular risk screening at socioeconomically deprived areas was no more cost-effective than in the general population whilst mass screening was less cost-effective. In contrast, in the Indian group levels of individual and area socioeconomic deprivation are more comparable to the white population, but overall risk of CVD is higher (12, 309, 310, 438). For the Indian group this explains why the performance of area deprivation measures in identifying individual socioeconomic deprivation was similar to the white group, but both mass and targeted screening for high cardiovascular risk were more cost-effective. Ethnic differences in the impact of population and high-risk approaches to CVD prevention, and in the cost-effectiveness of mass and targeted screening, could be explained by ethnic differences in baseline

cardiovascular risk, with greater impact and cost-effectiveness seen in the higher risk ethnic groups.

Chapters 4, 5 and 6 contained discussion sections that outlined the principal findings, and strengths and limitations of each analysis, alongside discussion of how the findings related to other relevant studies. Briefly, key strengths related to the use of survey data with a boosted sample of ethnic minority participants, use of QRISK2 as arguably the most appropriate cardiovascular risk calculator available at present and the use of evidence in informing modelling assumptions. Limitations arose from the age and sample size of the data used, estimations required for the calculation of QRISK2, the presence of missing data and steps required to address this, and the potential for the impact of the population approach to have been underestimated. This chapter builds on these previous discussion sections by examining issues and implications related to three core themes of this thesis, namely cardiovascular risk calculation, the use of ethnicity as a variable in research and health service planning, and CVD prevention policy. This is followed by conclusions and recommendations.

## **7.2 Cardiovascular risk calculation**

Cardiovascular risk calculation was used in two ways in this thesis. First, it was used to designate individuals as being at high or low cardiovascular risk (see Chapter 5). This application reflects the use of cardiovascular risk calculators in clinical practice and in cardiovascular risk screening programmes, where risk estimations are used to categorise individuals according to predetermined thresholds and to guide future interventions or advice. This use would be the same whether a mass or targeted screening programme was adopted. Indeed, cardiovascular risk calculators are integral to high-risk approaches to CVD prevention, and are a cornerstone of current national guidelines and screening programmes for primary prevention of CVD (13, 121). They can improve the accuracy of predictions of future risk beyond consideration of single risk factors, increasing the chances of risk reduction interventions being targeted at appropriate individuals (150, 151).

Despite the advantages and widespread use of cardiovascular risk calculation, it is important to recognise that they are imperfect tools that are often inaccurate predictors of risk. For instance, Jackson *et al* highlighted that in one study only 30% of cardiovascular events happened in individuals identified as being high-risk

by QRISK (a predecessor to QRISK2) (553). This misclassification of individuals as high or low risk could reduce the effectiveness and efficiency of even the best designed and implemented cardiovascular screening programme, as individuals who are subsequently going to have a cardiovascular event will not be correctly identified and therefore not offered risk reducing interventions, which instead may be given to people who would not have gone on to develop CVD. This limitation would have impacted the findings of the mass and targeted screening analysis in this thesis, and could mean that the cost to identify one high-risk individual may not be equivalent to the cost to detect one person who will actually develop CVD. Furthermore, the performance of cardiovascular risk calculators is known to vary by ethnicity (266, 434). Indeed, whilst the correct prediction of future events by cardiovascular risk calculators is far from perfect, it may be even lower in ethnic minority groups (450). This could further reduce the effectiveness and efficiency of cardiovascular screening in ethnic minority groups beyond the measures considered in these analyses and could adversely impact ethnic health inequalities. Further prospective analyses, based on sufficient numbers of ethnic minority individuals, could help to establish the significance of this issue and further improve the accuracy of cardiovascular risk prediction across ethnic groups. Whilst it may not be possible for cardiovascular risk calculators to ever accurately predict all cardiovascular events, it may at least be possible to predict them with similar accuracy across population subgroups.

In addition, there is limited evidence that the use of cardiovascular risk calculators has a positive impact on health outcomes (152, 554). Whilst short-term measures of the success of cardiovascular screening programmes, such as uptake or the measures of screening programme performance used in this thesis, may appear favourable, they do not provide evidence for positive clinical outcomes. Further evidence is needed to determine the long-term impact of cardiovascular risk calculation in cardiovascular risk screening programmes (152). Evidence from the NHS Health Check programme has the potential to inform this question, although it may be difficult to differentiate the effects of risk estimation from other aspects of the programme.

The second use of cardiovascular risk calculation in this thesis was in the estimation of average cardiovascular risk in population subgroups before and after implementation of population and high-risk interventions (see Chapter 6).

This illustrates a research application of cardiovascular risk calculators, similar to that adopted elsewhere (108, 534), where risk estimation is used to model future cardiovascular events. Whilst this use would not be subject to the misclassification that can arise when categorising individuals as high or low risk, limitations of cardiovascular risk estimation could mean that estimates of future cardiovascular events made using this method will be subject to inaccuracies, although the direction of these inaccuracies is unclear. From a policy perspective, although Manuel *et al* suggested that risk prediction algorithms could also be useful for planning and prioritising population interventions (114), population interventions would not rely on the use of cardiovascular risk calculators or be subject to their limitations.

A number of changes to the current definition of high cardiovascular risk and the tools used to predict cardiovascular risk have been suggested. These changes have the potential to alter how high-risk CVD prevention is delivered, by expanding the number of people eligible for individual risk reducing interventions or by altering the types of interventions offered. For instance, simpler methods of determining high cardiovascular risk have been suggested, such as using age alone (555), whilst NICE has recently proposed that the threshold for defining high cardiovascular risk be lowered from  $\geq 20\%$  to  $\geq 10\%$  (500). This latter change could result in higher numbers of cardiovascular events being prevented in all ethnic groups, as the number of people who are classified as being at high cardiovascular risk, and therefore offered individual risk reducing interventions would increase across the population. However, this would also increase both the financial and opportunity costs of this high-risk CVD prevention approach. In contrast, new guidelines recommend the use of lifetime risk prediction (556, 557), an approach that provides an estimate of an individual's risk of developing CVD during the rest of their life, rather than in just the next 10 years (558). This change is said to better reflect the progressive course of atherosclerosis and improve the identification of high cardiovascular risk in younger and ethnic minority individuals (556, 557). For ethnic minority groups in which CVD develops at younger ages lifetime risk prediction may be particularly beneficial (254, 556). This change has the potential to alter how CVD is prevented in the future as lifestyle rather than pharmaceutical interventions are likely to be more appropriate for younger people who are found to be at high lifetime, but low 10 year, cardiovascular risk (557).

However, further evidence is needed to establish the usefulness of lifetime risk measures, the effectiveness of long-term risk reduction interventions, and whether a threshold of high or low lifetime cardiovascular risk can be set (559).

Despite developments in cardiovascular risk prediction, such as new calculators adapted to specific populations and the addition of new variables, improvements in the accuracy of risk estimation have been incremental and small. Whilst evidence for the association between cardiovascular risk factors and the development of CVD is substantial, the fact that these risk factors do not predict higher proportions of cardiovascular events indicates complexity in the pathway to developing CVD that has not been accounted for or that may not be fully understood. Ethnic differences in cardiovascular risk factors, and potential differences in the association between these risk factors and disease, may add further complexity when considering the application of cardiovascular risk calculators across ethnic groups. New approaches to cardiovascular risk prediction continue to be developed and widely applied, however, unless the accuracy of cardiovascular risk estimation significantly improves the effectiveness of these tools in preventing CVD will continue to be limited. Perhaps the addition of epigenetic information could improve accuracy of risk assessment for individuals or, alternatively, these limitations could be minimised through greater use of population approaches to CVD prevention.

### **7.3 Ethnicity**

This thesis used ethnicity data to explore population subgroup differences in the impact of CVD prevention policies designed for the general population. A one-off self-reported measure of ethnicity, based on standard Census categories, was used. This approach is comparable to how ethnicity has been measured and used in other research and health service planning in the UK, and is perhaps the best method that is currently available. However, there are a number of issues to consider when interpreting evidence of ethnic differences in health or health policy performance.

Ethnicity is a multifaceted and complex variable (219-222), which is measured for a number of reasons, including for epidemiological, political and legal purposes. From an epidemiological point of view, ethnicity data can help develop understanding of patterns of disease between populations, provide appropriate and effective health care services, and tackle inequalities in health

(220, 222, 224). However, its complex meaning must also be considered. For instance, it is important to recognise the heterogeneity that exists within ethnic groups. There is likely to be important heterogeneity within the ethnic groups studied in this thesis, particularly if there are differences in other determinants of health such as migration status, socioeconomic position or cultural background within these groups. Indeed, whilst inequalities in socioeconomic position within ethnic groups were considered in these analyses, differences in migration status were not. A specific criticism of the use of ethnicity data is the aggregation of South Asian ethnic groups, often for reasons of sample size (222). Whilst the Indian group was analysed separately from the Pakistani and Bangladeshi groups in this thesis, and indeed the findings differed between these groups, again limited sample sizes meant that the groups could not be fully separated. Heterogeneity will also exist within categories of other commonly used epidemiological variables, such as socioeconomic position, and full consideration of heterogeneity within ethnic groups would be difficult using currently available ethnicity data. However, heterogeneity has implications for the development of health policy because, whilst evidence may suggest that a policy may be effective in a particular ethnic group, these findings may not apply equally to all individuals within that group. This limitation could be addressed by ensuring that ethnicity is not considered or analysed in isolation, but alongside other related variables such as socioeconomic position, migration status, experience of racism, and so on. In the context of secondary data analysis, this would require availability of data with sufficiently large sample sizes by ethnic group and other relevant variables, so that valid sub-group analyses could be performed. In a related issue, it is important to ensure that health policy decisions are not based on stereotypical perceptions of different ethnic groups, for instance on assumptions of the health behaviours of people with certain cultural or religious backgrounds. Indeed, it has been suggested that stereotypical assumptions made by health care professionals could lead to bias in the provision of healthcare and exacerbate ethnic inequalities in health (350).

Senior and Bhopal highlighted the issue of ethnocentricity that can arise when ethnicity is used in epidemiological research, whereby there is a tendency for researchers to compare minority groups with the majority leading to limited set of interpretations and conclusions about the nature of ethnic differences in

health (222). However, the implications of ethnocentricity for health policy development may differ. For decision makers developing CVD prevention policy for the population as a whole, comparisons against the ethnic majority may be necessary and may allow appropriate adaptations to population wide policy decisions. For instance, if the health needs of one ethnic minority group are found to be relatively high compared to the majority population, for whom the policy is being principally developed, a potential source of inequalities could be identified and the policy adapted as appropriate.

Ethnic inequalities in CVD and cardiovascular risk factors were evident in this thesis, consistent with existing evidence (see sections 3.4 and 3.5). Given the range of possible causes of ethnic inequalities in CVD (see section 3.7), a tension arises in how to interpret or tackle these differences. At one extreme, it is possible that some ethnic inequalities in CVD are unavoidable, arising from biological or genetic differences. In the way that differences in risk of CVD between men and women seem to have been accepted as inevitable, perhaps this is also true for some ethnic inequalities in CVD. In contrast, ethnic inequalities in CVD could be due to deeply embedded and troubling social issues such as racism and institutional discrimination. These starkly contrasting explanations of ethnic health inequalities would need to be addressed using very different actions. For example, the biological explanation of ethnic inequalities in CVD may suggest that steps are needed to optimise the effectiveness of pharmacological interventions in different ethnic groups or that knowledge of biological differences should be used to set ethnic specific thresholds of risk. Alternatively, racism and discrimination would need to be tackled through structural, legal and social changes. Biology and racism were selected, and separated, here to illustrate the contrast between explanations of ethnic inequalities in CVD. However, explanations of ethnic inequalities in health and CVD are not independent of each other but are likely linked through complex mechanisms. Indeed, some explanations may lie on the causal pathway between ethnicity and CVD, mediating this association. For instance, being a member of an ethnic minority group could lead to poorer socioeconomic position because of the negative impact of discrimination, resulting in increased risk of CVD; this pathway would suggest that a policy to reduce socioeconomic deprivation across the whole population could also improve ethnic inequalities in CVD. Therefore, whilst this complexity further increases the difficulties of choosing appropriate

and effective measures to reduce ethnic inequalities in health, understanding these associations is important because they may influence the choice and effectiveness of policies to improve health and reduce ethnic health inequalities.

Changes in the ethnic composition of populations present a further challenge for research and health service development in this area. As previously highlighted, the UK is becoming more ethnically diverse (19). Migration to the UK, key to the formation of ethnic minority groups, is increasing, and the countries that people migrate from have changed in recent years, particularly following the expansion of the European Union (560). At the same time, the experience of populations who migrated to the UK in previous decades will be developing as communities become established, future generations grow and socioeconomic circumstances change. For instance, UK projections suggest that higher proportions of ethnic minority populations will move to more affluent areas and become less segregated over forthcoming decades (561). Changing ethnic minority and migrant populations have implications for determining the health needs of populations and providing appropriate services (one high profile example of this is the impact of immigration on maternity services in the UK (562)). Therefore, public health professionals and researchers working in this area need to find ways to adapt to changes in populations.

A challenge in carrying out the analyses in this thesis was finding suitable and recent data with sufficient numbers of ethnic minority individuals. The data that were used are now 10 years old and may not reflect recent demographic and socioeconomic changes in ethnic minority populations. In particular, movement of ethnic minority populations out of deprived areas may make targeted screening for high cardiovascular risk based on area deprivation relatively less cost-effective in these groups. The data were further limited by incomplete response rates that also varied by ethnicity. This raises the possibility that the samples studied may not be representative of the intended populations. Whilst survey weights were used to account for non-response, it is possible that some response bias may remain. Given that the analyses in this thesis focused on investigating ethnic differences, this bias would be particularly important if it also varied by ethnic group - i.e. if the populations that responded varied by ethnicity. Whilst this limitation must be considered when interpreting the results, its scale and direction are unclear.

The routine collection of ethnicity data is improving (563), and data linkage may further increase the availability of large scale datasets that include ethnicity (251, 252, 261, 564), as well as economic and migration data. The availability of these new data will be helpful for both research and health service planning. These issues will also be relevant to other countries that are also experiencing changes in the ethnic composition of their populations (565).

#### **7.4 Implications for CVD prevention policy**

Current UK CVD prevention policy appears to favour high-risk approaches, based on cardiovascular risk screening. English policy makers have opted for a mass screening strategy in the NHS Health Check programme, a programme that comes with significant financial and opportunity cost. This is despite unclear evidence for the effectiveness of health checks on clinical outcomes (although the NHS Health Check programme has the potential to add to the evidence base in this area) (155, 566-569), alongside evidence that population approaches may be a more effective and equitable way of preventing CVD (5, 108, 109), and targeted screening may be a more cost-effective alternative to mass screening (171, 192). This thesis further adds to these findings by indicating that population approaches may be particularly effective in ethnic groups at high-risk of CVD, and targeted screening may be particularly cost-effective in these groups. Given existing ethnic health inequalities and the fact that health policy needs to reduce inequalities as well as improve overall health, these findings lend further support to the use of population rather than high-risk approaches, and to a strategy of targeted rather than mass screening in the prevention of CVD in the general population.

Despite evidence on effectiveness, cost and the equity impact of these CVD prevention policies, there are other factors that will influence the policy choices that are made. These factors include questions of politics and economics, and may explain the current direction of CVD prevention policy. For instance, population approaches have been criticised because they involve changes that affect the whole population, arguably infringing individual's rights to determine their own health choices (94). Whether this is seen as a reason for avoiding population approaches may depend on political viewpoint. Development of the NHS health check programme may reflect a political preference for a high-profile programme that is seen to be available for the whole population and that

encourages individual behaviour change. Furthermore, population approaches may also involve compulsory regulation of products produced by private industries, such as the food industry (18). However, these industries may prefer the choice of voluntary regulation or a focus on individual lifestyle change, which may influence the policy choices that are made.

This thesis focused on the choices between population and high-risk approaches, and between mass and targeted screening for high cardiovascular risk. However, there are other policy options available that may provide further opportunities for CVD prevention. First, an area-based approach could be taken to prevent CVD. This thesis explored the use of area deprivation measures as a means of targeting socio-economically deprived individuals, and subsequently as a tool for targeting cardiovascular risk screening programmes. However, this was an approach based on individuals and not area itself. Physical and social environments can influence health and CVD (177, 178, 184), an association that may be particularly relevant to ethnic minority groups owing to issues of segregation, and concentration in socioeconomically deprived areas and areas with unhealthy environmental characteristics (see section 3.8). Therefore, in addition to using area as a means of targeting at risk individuals, interventions could be developed which make areas themselves better for cardiovascular health, for example through urban design enabling easier access to active travel or green space to increase levels of physical activity and reduce stress (570, 571). This type of intervention could potentially lead to lasting improvements in upstream determinants and causes of CVD, with the co-benefit of improving health outcomes for other chronic diseases. If the purpose of an intervention were to prevent CVD by improving area characteristics, this would raise the question of which areas would be selected - socioeconomic deprivation and the ethnic make-up of the population may be factors that would influence this decision. Second, whilst this thesis considered primary prevention of CVD, health policy could be based on achieving primordial prevention - that is prevention of cardiovascular risk factors before they occur rather than reduction in existing risk factors (572). This is perhaps an idealistic scenario, which is most likely to be achieved in populations that currently have a low baseline risk using comprehensive population interventions and environmental change (79), reflecting our understanding of the development of cardiovascular risk factors over the lifecourse (573). However, it holds significant potential to improve

population health across all ethnic groups (572, 574). Indeed, one approach could be to achieve primordial prevention of CVD by stopping the development of cardiovascular risk factors in children through the kind of upstream area intervention described above. Third, another, quite different, option is the widespread use of a pill that contains a combination of medications known to reduce cardiovascular risk, i.e. the “polypill” (148). This could be a relatively simple approach to deliver, although evidence of its effectiveness is mixed (148, 575). Furthermore, its effectiveness in different ethnic groups would need to be assessed, as it is possible that the same combination of medications may not be equally effective across ethnic groups.

Whilst the changing ethnic composition of populations has implications for research and service provision, the evolving CVD epidemic will also influence which CVD prevention policy options are most appropriate. The burden of CVD is declining in the UK and other developed countries, however, prevalence of obesity and diabetes are increasing (39, 41, 57). These risk factors are particularly important in many ethnic minority groups in the UK, and increases in them may further increase ethnic inequalities in health (576). Therefore, CVD prevention policy will need to increasingly focus on the prevention of obesity and diabetes, including in high-risk ethnic minority groups.

## **7.5 Conclusions and recommendations**

### **7.5.1 For policy makers**

The findings of this thesis lead to four conclusions. First, area deprivation measures work relatively effectively and efficiently at identifying individual socioeconomic deprivation in the largest ethnic minority groups in England, with reassurance that these groups would not be systematically disadvantaged by the use of these measures. This finding is of relevance to policy makers seeking to use area deprivation measures to target public health interventions at socioeconomically deprived individuals but needing to ensure that population subgroups would also be appropriately targeted. Whilst the findings support the use of area deprivation measures for this purpose from an ethnicity perspective, the age of the data analysed and potential changes in the geographical distribution of ethnic minority populations are important caveats to this conclusion. In addition, further evidence would be needed to extend this

conclusion and recommendation to other population subgroups and smaller or new ethnic minority groups.

Second, cardiovascular risk screening programmes are a relatively cost-effective option in ethnic groups that have a high risk of CVD, and screening programmes targeted at deprived areas could be a particularly cost-effective option in these groups. Given limited health care resources and the need to use existing resources efficiently, this further supports the use of area based targeted cardiovascular risk screening programmes in the general population. Although the cardiovascular risk screening strategies adopted in the UK may be unlikely to change in the near future, this finding may be of relevance to health professionals delivering programmes locally and could be relevant to discussions about the relative merits of the existing approaches.

Third, comprehensive population and high-risk approaches have the potential to prevent a significant amount of CVD across all ethnic groups, with population approaches being particularly effective and equitable in ethnic groups at high-risk of CVD. Indeed, the potential impact of population approaches may be greater than estimated in this thesis. The relative merits of population and high-risk approaches to CVD prevention have been debated for many years, with a general consensus that a balance of both approaches is needed. However, current CVD prevention policy in the UK tends to favour high-risk rather than population approaches, despite the existence of feasible and successful population policies (for example, salt reduction programmes and smoke free legislation). Therefore, current high-risk policies should be supplemented with further population policies in order to maximize the health gain arising from CVD prevention, narrow health inequalities and create a more sustainable health service in the long-term.

Finally, the fact that this thesis found ethnic differences in the potential effectiveness, costs and equity impact of various CVD prevention policies, highlights the importance of assessing the impact of public health interventions in subgroups of the population. This should be done for both new and existing policies and interventions. Modelling can be used to predict the potential cost-effectiveness and impact on health inequalities of new policies within population subgroups, whilst monitoring and evaluation can be used for existing policies. This thesis considered ethnicity, but other important groupings would be those based on socioeconomic position, sex, age and geographical area of residence,

including urban or rural location. Although this would be a significant undertaking, and some of this work is already undertaken (e.g. in health inequalities impact assessments), a more systematic approach across a wide range of population subgroups could help to maximize the ability of public health interventions to improve health and reduce health inequalities.

### **7.5.2 For researchers**

The conclusions and recommendations described above will also be of relevance to researchers. In particular, researchers will need to continue to be involved in assessing the impact of public health interventions in population subgroups and in further investigation of the utility of area deprivation measures to target socioeconomically deprived individuals in other population subgroups. The availability of appropriate and sufficiently large datasets is key to achieving these recommendations and informing the implementation of effective and equitable public health policies. Whilst no secondary data sources are perfect, a number of data limitations were encountered in this thesis. This highlights the need for researchers in this field to continue to expand the data available. Datasets should contain ethnicity data alongside other relevant social and economic measures that are relevant to health and to the pathway between ethnicity and health. Increasing the recording of ethnicity data in routine health datasets alongside data linkage are two key approaches that can provide large, flexible datasets that could be adapted to the changing demographics of the population.

Considered together these conclusions provide reassurance that ethnic minority groups in the UK are unlikely to be systematically disadvantaged by CVD prevention policies that have been suggested, and implemented, for the general population. Furthermore, ethnic minority groups at high risk of CVD may benefit more from these CVD prevention policies, leading to a reduction in ethnic health inequalities. General speaking, this lends further support to the argument in favour of the implementation of and investment in comprehensive CVD prevention policies in the UK, using a range of approaches.

## **7.6 Reflections on this and future research**

Reflecting on my experience of carrying out this research has allowed me to consider alternative approaches for addressing similar research questions in the future. At various points in this thesis the potential for using alternative data

sources was considered. If I were to start this research again I would look again to see what new data sets were available, in particular exploring the potential for data linkage between Census and primary care data as this would provide the information required for these analyses. Another alternative approach, which was considered at the outset of this research, would be the use of microsimulation to create a large, ethnically diverse hypothetical population. This approach could have allowed the inclusion of additional ethnic groups, for example individuals from Eastern Europe, and could have led to the production of a model that could be applied to alternative research questions in the future, although its development would still have had to rely on existing lifestyle and health data from across ethnic groups.

Moving forward from the analyses in this thesis, it would be useful to investigate ethnic differences in the potential impact of specific population CVD prevention policies. For example, whether there would be ethnic differences in the impact of eliminating trans fatty acids from the food chain in the UK. An analysis of this type would require ethnic group specific data on trans fatty acid intake. Furthermore, it would be interesting to look again at the relationship between area, health and ethnicity and consider the use of population wide, area-based interventions that could prevent CVD or reduce health inequalities through systematic changes in areas themselves.

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