Evaluation of the NHS Health Check Programme; Local and National Findings from the Early Stages of the Programme

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Chapter I: Abstract

**Background:** The NHS Health Check programme is one of the boldest commitments to primary prevention in cardiovascular disease (CVD) internationally. It offers risk assessment and management to the entire 40 to 74 year old population, without existing vascular disease. I aim to assess its early impact in general practice, and examine workload implications.

**Methods:** Modelling the population at high risk of CVD in England; comparing CVD risk prediction using two risk scores, and two methods of data imputation for missing risk factor data; the assessment of CVD risk factor recording before the programme and Health Check uptake, using patient-level medical record data from general practice in Ealing, London.

**Results:** Prior to the programme, in Ealing, there was good recording of blood pressure (85.6%) and smoking status (95.8%) in a general population; cholesterol recording was lower (55.6%). Uptake of the Health Check was lower than national estimates at 45% compared with 75% projections, and there were small increases in statin prescribing, reaching 45 percent of the eligible population. Health Check uptake were greater in south Asian patients (adjusted odds ratio=1.80 (1.37-2.36). The JBS2 CVD risk score generated overall higher estimates of risk than QRISK2 (mean of 13% compared with 11%); this was significantly greater in south Asian men, the group exposed to the JBS2 risk multiplication factor. Modelling, using QRISK2, predicts 2 million patients at high risk in England, with screening and management costing £176 million. Cost using the JBS2 risk score are estimated to be over two times higher JBS2.

**Conclusions:** Poor uptake of the NHS Health Check and interventions will severely limit the population-wide impact of the programme, Given this, and other limitations, I suggest a targeted approach to screening may be an appropriate alternative, and demonstrate from previous literature the complimentary use of population-wide prevention is likely to significantly improve CVD prevention.
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Chapter v: Abbreviations

AF- Atrial Fibrillation
AIC- Akaike Information Criterion
AOR- Adjusted Odds Ratio
AUROCS- Area under the Receiver Operating Characteristic Curve
BFHS- British Family Heart Study
BHF- British Heart Foundation
BME- Black and Ethnic Minority
BMI- Body Mass Index
CHD- Coronary Heart Disease
CI- Confidence Interval
CDC- Centers for Disease Control
CKD- Chronic Kidney Disease
CQC- Care Quality Commission
CRP- C-Reactive Protein
CVD- Cardiovascular Disease
DALY- Disability Adjusted Life-Year
EMR- Electronic Medical Record
FOBT- Faecal Occult Blood Test
FSA- Food Standards Agency
FTE- Full Time Equivalent
GMS- General Medical Services
GOF- Goodness-of-Fit
GP- General Practitioner
GPRD- General Practice Research Database
HDL- High-Density Lipoprotein
HHS- Department of Health and Human Services
HSFe- Health Survey for England
IFG- Impaired Fasting Glycaemia
IGT- Impaired Glucose Tolerance
IMD- Indices of Multiple Deprivation
IQR- Interquartile Range
JBS- Joint British Societies (the organisation)
JBS2- Joint British Societies 2 (the CVD risk score)
KPI- Key Performance Indicator
LDL- Low- Density Lipoprotein
LES- Locally Enhanced Service
LSOA- Lower Super output Area
MAR- Missing at Random
MCAR- Missing Completely at Random
MOR- Median Odds Ratio
MRFIT- Multiple Risk Factor Intervention Trial
NICE- National Institute of Clinical Excellence
NNT- Numbers Needed to Treat
NSC- National Screening Committee
NSF- National Service Framework
ONS- Office for National Statistics
OR- Odds Ratio
PAD- Peripheral Arterial Disease
PCT- Primary Care Trust
QALY- Quality-Adjusted Life Year
QOF- Quality and Outcome Framework
ROC- Receiver Operating Characteristic Curve
RRR- Relative Risk Reduction
SE- Standard Error
SELSS- South East London Screening Study
SEP- Socioeconomic Position
SHA- Strategic Health Authority
TIA- transient Ischaemic Attack
TTM- Trans-Theoretical Model of States of Change
VPC- Variance Partitioning Coefficient
VRA-LES- Vascular Risk Assessment Locally Enhanced Service
WHO- World Health Organization
Chapter vi; Acknowledgements

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Chapter vii; Statement of Contribution

This thesis represents my own work, with support from my supervisors Alex Bottle, Azeem Majeed and Chris Millett. Michael Soljak assisted me in the processing of data and analyses for the modelling using Health Survey for England data. Edgar Samarasundera provided advice on geographical mapping techniques.
Chapter 1; Introduction

In January 2008, a speech by the UK Prime Minister Gordon Brown outlined details of an ambitious national prevention programme for cardiovascular disease (CVD) in England. This programme, which became the NHS Health Check, aims to offer a cardiovascular risk assessment to the entire eligible population in England. The eligible population consists of those aged 40 to 74 years, with no prevalent CVD, diabetes or other related vascular conditions. The programme not only involves risk assessment, but also risk management. Those screened and found to have risk factors for CVD must be referred or signposted to appropriate risk lowering interventions, whilst those found to have undiagnosed clinical disease enter the relevant care pathway.

The NHS Health Check is widely regarded as an ambitious programme. There has never previously been such a large, universally implemented programme of this type in the UK, or indeed elsewhere in the world, merely small scale local projects and clinical trials. As a result, the effectiveness of such a large programme is untested, but is certain to generate a large expenditure. Its careful evaluation will be of paramount importance and findings from the programme certain to be of international interest.

1.1 Cardiovascular Disease

1.1.1 A definition of CVD

Cardiovascular disease comprises of a group of disorders which affect the heart and blood vessels.¹ Common diseases found within CVD are outlined in Table 1-1. CVD covers both acute and chronic forms of disease; events can be acute such as myocardial infarction (MI- a form of coronary heart disease (CHD)) or stroke. CVD can also be chronic, either once a patient has experienced an acute event or in purely chronic forms such as angina pectoris.
Table 1-1; common cardiovascular diseases

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary heart disease (CHD)-</td>
<td>disease of the blood vessels supplying the heart muscle</td>
</tr>
<tr>
<td>Cerebrovascular disease-</td>
<td>disease of the blood vessels supplying the brain</td>
</tr>
<tr>
<td>Peripheral arterial disease-</td>
<td>disease of blood vessels supplying the arms and legs;</td>
</tr>
<tr>
<td>Rheumatic heart disease-</td>
<td>damage to the heart muscle and heart valves from rheumatic fever, caused by streptococcal bacteria</td>
</tr>
<tr>
<td>Congenital heart disease-</td>
<td>malformations of heart structure existing at birth</td>
</tr>
<tr>
<td>deep vein thrombosis and pulmonary embolism-</td>
<td>clots in the leg veins, which can dislodge and move to the heart and lungs</td>
</tr>
</tbody>
</table>

Within CVD, CHD and cerebrovascular disease contribute the majority of events and the greatest disease burden. These two groups are frequently caused by blockages to the blood vessel supplying the heart or brain (ischaemic stroke) respectively. Acute blockages to the heart result in an MI, whereby a sudden interruption in blood supply can permanently destroy cardiac tissue. Less severe blockages result in angina pectoris, where a partial blockage reduces the oxygen supply to the heart causing chest pain on exertion.

Ischaemic stroke involves an acute blockage to cerebral arteries that destroys cerebral tissue. Transient ischaemic attack (TIA) is similar to ischaemic stroke. TIA is also caused by a blockage to the cerebral arteries, but is defined by the symptoms naturally abating within 24 hours. Patients who have suffered a TIA are at high risk of suffering a stroke. Stroke, in addition to ischaemic, can be caused by bleeding within the brain (haemorrhagic stroke accounts for approximately twenty percent of the total stroke cases). Blockages to arteries, both in the cardiac and cerebral tissue, are frequently caused by the process of atherosclerosis. Artery walls thicken due to build up of deposits, often cholesterol.

1.1.2 Secular trends in CVD

Over the last four decades there have been large reductions in levels of CVD in the UK and across the western world (Figure 1-1). The UK for example has seen a 60 percent reduction in CVD mortality between 1985-2005\(^2\) and a 62 percent reduction in a person's risk of a first MI between 1978 and 2003\(^3\) with 68,000 fewer deaths due to CVD in 2001 than in 1981.\(^4\)
Despite these reductions, CVD is still the leading cause of mortality in the UK. In 2008, combined cardiovascular and circulatory diseases (including diabetes) accounted for 33 percent of the total number of deaths in the UK and 25 percent of premature deaths (those aged under 75).\(^2\) CHD alone accounted for 15 percent of the total deaths. Using data from the Quality and Outcomes Framework (QOF) in 2009/10, out of a total of 54,800,000 patients registered with general practices in England, there were 1,890,000 (3.4%) with diagnosed CHD and 922,000 (1.7%) with stroke or TIA.

### 1.1.3 Economic impact of CVD

The large disease burden associated with CVD generates large health care costs. In 2006, an estimated £14,400 million was spent in the UK on CVD care, approximately 12 percent of total NHS spending.\(^2\) Estimates beyond purely healthcare expenditure suggest the cost of CVD to the UK reached €36,550 (~£26,010) million in 2003 (€169 billion across the whole of the EU).\(^5\) These high rates of spending are mirrored in other high-income countries. In the USA, CVD costs the nation $475.3 billion annually, 50 percent more than spending on the next highest disease group.\(^6\) CVD is increasing becoming a burden in low to middle income...
countries; in China for example spending has reached €30.8 billion, 4 percent of the gross national income.\(^7\)

### 1.1.4 Risk factors for CVD

Risk factors for CVD can be separated into two groups, modifiable and non-modifiable. The strongest predictor of CVD risk, age is in fact non-modifiable. CVD risk increases substantially with age. Gender and ethnicity are two further important non-modifiable risk factors. Men have a significantly greater risk of CHD than woman before menopause. Post-menopause, the risk profiles of the two sexes converge.

Ethnic origin plays a complex role in CVD risk. It impacts both on levels of other risk factors and directly upon risk. Family history of CVD is a final non-modifiable risk factor; having a first-degree relative experiencing premature CVD event increases one’s risk.

The second group of risk factors are considerably more important for the prevention of CVD, the modifiable risk factors. Modifiable risk factors can be separated into two groups; clinical and behavioural. The most significant, traditional clinical risk factors for CVD are blood pressure and lipid levels. Raised blood pressure increases CVD risk, especially of stroke. Blood lipid levels have a number of impacts on CVD risk; raised total cholesterol and low-density lipoprotein increase risk, whilst raised high-density lipoprotein is protective. Both obesity and adiposity are independent risk factors for CVD, and additionally affect risk factors including blood pressure and lipid levels. Clinical diabetes and poor blood glucose control both increase CVD risk. More recently a number of novel risk factors have been identified. These clinical markers of risk include C-reactive protein (CRP), fibrinogen and homocysteine, and are frequently markers of inflammation.

Behavioural risk factors have a complex relationship with CVD risk. They affect both CVD risk directly and other risk factors. Tobacco use increases CVD risk: risk increases with the levels of exposure. Diet is important for CVD risk, with many different elements. High fat (saturated) and salt diets will negatively affect lipid levels and raise blood pressure. Fibre,
fruit and vegetable consumption are independently protective. Physical activity and physical fitness are independently protective of CVD; both also modify other risk factors including blood pressure and lipid levels. Alcohol has a complex relationship with CVD risk, in moderation being protective whilst higher levels of intake are associated with increasing risk. Finally deprivation is a risk factor for CVD, the relationship modulated through a complex, multi-factorial pathway. Of the modifiable risk factors, three stand out for their relationship with risk; cholesterol, blood pressure and smoking alone predict up to 75 percent of CHD incidence.\(^8\)

*Secular trends in CVD risk factors*

Population level reductions in risk factors have been the major driving force behind reductions in CVD mortality.\(^4\)\(^9\)-\(^12\) They have made a larger contribution than improvements in healthcare and treatment of patients with established disease, with estimates between 60 and 75 percent of the total reduction.\(^10\)\(^12\) The reductions in mortality cannot, however, be solely be attributed to trends in risk factors. Medical improvement through the second half of the 20th century, often powered by the integration of new technologies, have contributed to the decline in mortality, and its part must not be forgotten.

Over the last 40 years the major cause behind these risk factor reductions appears to have been dynamic. Evidence is growing that initial large reductions were driven by population-wide behavioural changes.\(^13\) More recent reductions have, however, had drug therapy play a larger role.\(^13\) This could be interpreted in two ways: one could argue the greater use of effective drug therapies has allowed them to make a greater contribution to risk reductions in recent times. Alternately, as positive societal trends in health behaviours have slowed, drug therapies could have been allowed to play a larger relative role. If the latter is true, then although risk factor reductions currently play a smaller roll than in the past, this would still be the best method to pursue into the future. It is currently unclear which scenario is true; further clarification will be vital for the future direction of CVD prevention.
It has been more difficult to elucidate the underlying cause behind reductions in CVD incidence compared with mortality. To lower incidence, one must see reductions in CVD risk factors. These reductions are evident in the three CVD risk factors most strongly associated with incidence; blood pressure, lipid levels and smoking. Between 1980 and 2008, total cholesterol levels fell across Australasia, North America, and western Europe, with average rates of 0.2 mmol/L per decade. In the USA, mean population total cholesterol levels reduced from 5.50 mmol/l in 1980 to 5.16 mmol/l in 2000. Likewise, there have been reductions in blood pressure levels in high-income regions, peaking at rates of 3mmHg per decade. The USA has seen a reduction in the prevalence of raised blood pressure of 40 percent between 1960/2 and 1999/2000, although reductions in the prevalence of diagnosed hypertension have not always matched. Finally, a number of high-income countries have witnessed reductions in smoking prevalence since the 1970s. These smoking reductions, in fact, are likely to be the single greatest factor, responsible for over a third of the entire risk reduction. The UK has experienced a similar pattern in changing risk factors as other high-income countries; with reductions in the three risk factors.

Two major risk factors stand out as anomalies to these decreasing trends. There have been global increases in body mass index (BMI), with especially large changes in the high-income countries. Secondly, the prevalence of diabetes has seen recent dramatic increases, with for example a 54 percent increase in prevalence in the UK between 1996 and 2005 and a 69 percent increase in Ontario, Canada between 1995 and 2005.

Risk factor reductions have decreased CVD incidence. The question remains, however, as to what has been the major driver behind these changes. The two candidates are the increased use of pharmacological agents and background changes to population behaviour, especially through diet. The answer to this question will again have major implications for the future of CVD prevention. Studies demonstrating the importance of risk factor reductions have, at times, failed to establish this underlying cause. Often the detailed longitudinal data
required to answer this question are simply absent, leaving the current literature both minimal and conflicting.

One study, using Swedish data, has found major improvements in lipid levels across the population. Concomitantly they failed to find increases in pharmaceutical intervention; this implies there are further underlying changes, possibly in diet, which are responsible for the patterns in lipid levels.\textsuperscript{25} Similarly, trans-European data suggest that behaviour changes are likely to be crucial, with only minimal impacts of medication.\textsuperscript{26}

Contrary to this, analysis of a 20 year British cohort, entering the cohort aged 40 to 59, suggests blood pressure reductions were predominately found in medication users. Hence treatment was likely to be the major factor.\textsuperscript{27} These were older men and this finding may not be generalisable to the entire population. A recent Japanese study has also supported medication as the largest known factor to reduce blood pressure levels, especially in the older population. Notably, this study suggests that the largest driver behind these reductions is as yet unknown.\textsuperscript{19}

One further complexity was touched upon above. The relationship between CVD risk factors and both drug therapy and health behaviours is dynamic over time. The Japanese finding presents data from the tail-end of the CVD risk reduction, between 1986 and 2002. A better question for interventionary public health would be what drove risk factor reductions in the 1970s, when rates of CVD fell at their greatest.

The major cause of risk factor reduction is currently unknown; however, behavioural trends must have played at least some role. Prescribing levels in areas experiencing risk factor reductions do not meet those required to be the main causal factor.\textsuperscript{25} Even now, in high-income countries, prescribing levels do not reach levels required to exact the changes seen over the last four decades. One final piece of circumstantial evidence that secular change in behaviour is central to risk factor reductions is the timing of the changes. Many reductions
started before the widespread availability of therapeutic agents. Lipid reductions, for example, began before statins were commonplace and low-cost. 13

1.1.5 A watershed moment in CVD care?

Although the incidence of CVD has declined in high-income countries, we currently stand at a critical point in time in the interplay between CVD care and health system funding. Along with gender, the strongest predictor of CVD is age. In the UK, in the population aged 75 and older, 30 percent live with prevalent CVD compared with 10 percent in the general population. 2 If current patterns in the prevalence of CVD persist, an aging western population will dramatically increase the number of prevalent cases of CVD, which in turn will increase the costs of care. 28 29 Estimates from the USA indicate that CVD spending could triple in the next twenty years alone. 29

An equivalent projection in the UK would place the NHS under a catastrophic strain regardless of the organisation's financial situation. An increase in spending will inevitably deplete the organisation’s finite resources. This impact would be exacerbated in a health system which shows little sign of financial growth over the coming years, and may face real-term cuts in spending. 30 Evidence above documented how risk factor reductions have been central to CVD reductions through the second half of the 20th century. Further gains in cardiovascular risk factors, preventing the projected rise in disease burden, might be the only way to mitigate the threat of CVD spending destabilising the NHS, and the health systems of other high-income countries. 29

1.1.6 Inequalities in CVD

Not only do CVD and its risk factors have a large overall impact on health in the UK, they also play a significant role in generating health inequalities. 31 32 Socio-economic variation in CVD has been well documented since the Whitehall study in the 1970s. 33 These socioeconomic variations translate into large absolute differences across populations. In 2008, CHD mortality was nearly 1.5 times higher in the most deprived fifth of the UK
population compared with the least deprived.\textsuperscript{2} There are additional marked inequalities in incidence based upon individual level employment grade, again up to 1.5 times higher in the lowest versus highest grade.\textsuperscript{34} Both individual level measures of deprivation (income for example) and area level deprivation independently map inequalities in CVD.\textsuperscript{35}

In addition to socio-economic, there are ethnic disparities in CVD,\textsuperscript{36} with large differences in incidence in the UK.\textsuperscript{37} South Asian patients are at higher risk of CVD,\textsuperscript{36,37} despite lower levels of many risk factors, with an especially large burden of CHD.\textsuperscript{38-40} Men living in the UK born in Bangladesh have over twice the CHD mortality of those born in England\textsuperscript{41} and amongst men of a Pakistani ethnic background CVD accounts for 43 percent of the total mortality (compared with 33 percent in white British).\textsuperscript{2} Recent data demonstrate patients from a Pakistani background have a greater prevalence of angina, whilst mortality and hospital admissions related to chest pain were greater in men from an Indian background, and in both sexes from a Pakistani background.\textsuperscript{39} Similarly, longitudinal data demonstrate a greater incidence of angina and chest pain in south Asian groups.\textsuperscript{40} For stroke, patients from black ethnic backgrounds suffer the greatest morbidity and mortality.\textsuperscript{37,38} Men born in west Africa have over 2.6 times higher stroke mortality than English born.\textsuperscript{41}

Ethnic and social disparities in CVD are partially explained by differences in CVD risk factors.\textsuperscript{42,43} The raised stroke prevalence in the black population, especially in women, is largely attributable to raised blood pressure.\textsuperscript{36} Meanwhile the increased risk of CHD in the south Asian population is in part caused by increased insulin resistance.\textsuperscript{44,45} However, ethnic differences cannot currently be fully explained by the levels of known risk factors. Either unknown, intrinsic differences exist between the ethnic groups or less likely there are undiscovered risk factors that modulate the inequalities. Genetic influences will also play a role in ethnic differences.

These societal inequalities in CVD map into staggering regional differences across England. The age standardised mortality from CHD in men from 2001 to 2006 ranged from 145 per 100,000 in the South East to 196 in the North West. Differences between local authorities
were even more marked, from example 105 in Kensington and Chelsea compared with 237 per 100,000 in Barnsley.  

1.2 CVD prevention

1.2.1 Disease prevention

The field of disease prevention covers a wide range of processes; from reducing risk factors in a population free from clinical disease, to limiting the impacts of a condition once established.  

Traditionally, it has been seen as helpful to separate prevention into three separate processes.

*Primary prevention is [the] protection of health by personal and communal efforts... and elimination of environmental risks*

*Secondary prevention is a set of measures available to individuals and communities for the early detection and prompt intervention to control disease and minimise disability*

*Tertiary prevention consists of measures aimed to soften the impact of long-term disease and disability; ... minimise suffering and maximising potential years of useful life.*

*Tertiary and quaternary prevention*

I shall not discuss tertiary prevention any further. Although important, it is very different from the remaining areas of prevention, it relies mostly on rehabilitation services and lies more removed from public health and health promotion. Likewise a further term, *quaternary prevention* has been discussed in literature, but this has been used by different authors to encompass different ideas, has not been widely adopted and I shall not consider further.
**Primordial prevention**

More recently a fourth level, *primordial prevention* has become widely adopted after the concept was first discussed in 1978.\(^4^9\) This is defined as-

> preventing the emergence of predisposing social and environmental conditions that can lead to causation of disease\(^4^8\)

*Primordial* prevention is of vital importance; in fact it has the potential to be the most important preventative mechanisms. There has, however, been confusion in the literature over the concept of primordial prevention. At times, the term has been used differently from above. A second interpretation considers primordial prevention at an earlier stage in the life course, with authors implying that it requires the maintenance of a healthy cardiovascular risk profile through childhood and into adulthood.\(^5^0\)\(^5^1\)

The concepts discussed under the title of primordial prevention are of the highest importance to society. The question remains, however, whether there is the need for the discussion of *primordial* prevention and whether it in fact differs from the traditional concepts of primary prevention. Many would strongly argue that the two processes are fundamentally different.\(^5^2\)

This difference stems from primordial prevention acting more distal from disease outcomes than primary prevention. For example, changing the built environment to enable physical activity is considerably removed from reductions in CVD risk factors.

Defining the term primordial prevention may severely downplay the scope of primary prevention. Interventions well outside of the medical domain, town planning to promote physical activity for example, do lie within the bounds of primary prevention. This becomes especially apparent when considering the work of Geoffrey Rose, which is further discussed below.\(^5^3\) Rose described his mass or population primary prevention strategies as *radical*, a word which firmly emphasises that its scope is as wide as the primordial prevention now discussed. The added division of primordial prevention may therefore be redundant, and add unnecessary confusion to the discussion of prevention. In fact the phrase primordial prevention follows this, and is still used infrequently in literature.
The one area that the term primordial prevention may be important is in highlighting the implications that the life-course has on CVD risk.\textsuperscript{54} My evaluation of the NHS Health Check programme however covers the 35 to 74 age group, therefore a truly life-course approach to prevention lies outside its scope. Nonetheless, the emerging evidence clearly demonstrates the importance of life-course in CVD and this is an important area in prevention.\textsuperscript{55}

\subsection*{1.2.2 Primary and Secondary prevention of CVD}

\textit{Secondary prevention of CVD}

I outlined above the divisions within prevention in a general context. Now I shall consider both primary and secondary prevention specifically relating to CVD. In the context of CVD, secondary prevention involves the reduction of CVD risk factors in patients who have already experienced a CVD event. This event could be acute, a MI or stroke, or chronic such as diagnosed angina. The single goal of this risk factor control is to prevent further CVD events. Although unique to CVD, this process still sits within the broader definition above. It involves the control of disease, and less directly, minimising disability.

The rationale behind secondary prevention is that a large proportion of the total CVD events occur in patients with existing clinical disease, a relatively small population (approximately 6 percent of the total). In a cohort of 36,000 patients aged 30 to 74, Kerr et al.\textsuperscript{56} found that 42 percent of the total CVD events occurred in the 10 percent of the population with prior CVD. These patients are on average at a higher risk than the general population therefore have the greatest need for risk reduction.

Targeting CVD patients for preventative therapy creates a cost effective prevention strategy. One can cover a relatively large proportion of the total population risk in only a small group, getting the most out of limited resources. Secondary prevention has a further strength in its implementation. There is a clearly defined, mutually exclusive target population, i.e. patients with CVD. This clarity and lack of additional stratification can make the process simpler and therefore easy to implement.
In the UK, in 2004, the payment structure and contract for general practice within the NHS was significantly altered. The new general medical services (GMS) contract maintained a baseline, ‘global sum’, payment to practices based on their list size, although with greater weighting for need. It also, however, introduced a significant pay-for-performance framework, named the Quality and Outcomes Framework (QOF), which provides payment in addition to the baseline. Initially the QOF consisted of 146 health indicators, aimed to outline best practice care, which equate (not evenly) to 1050 ‘points’. Achievement of the indicators, within the practice population is translated to a number of points. Finally, the points are annually translated into payment for the practice.

Within the QOF there has been a structured secondary prevention programme for CHD and Stroke/TIA. Despite the earlier presence of national clinical guidance and the National Service Framework for Coronary Heart Disease (NSF-CHD), the QOF actively standardised care for the first time. The QOF aims to strengthen the clinical management of patients (secondary prevention) within general practice. Practices must maintain registers of diagnosed patients within the two disease areas, carry out a series of process measures for patients aimed to improve clinical care and control CVD risk factors to meet intermediate outcomes, clinical targets for risk factors designed to reduce the risk of a further disease event.

The secondary prevention of CVD is a vital process. Interventions are effective, reducing CVD risk in a population with great need. Secondary prevention can reduce overall health care costs. A further strength is that given the population have first-hand experience of CVD, they are likely to be well motivated to reduce their future risk.

The criticism of secondary prevention is over its scope. Although a large proportion of CVD events are secondary occurrences, the majority are first time. Using the data from Kerr et al. described above, 58 percent of CVD events occur in the population previously free from disease. Had interventions been focused solely on secondary prevention, the majority of people to have a CVD event would not have received any previous preventative care. Low to
moderate risk patients still experience CVD events. They do have a lower risk individually, yet given their vastly greater number, this sums to give a greater cumulative risk and hence more CVD events.

There is one other, slightly different concern in concentrating on a secondary prevention group, thereby omitting the wider population. Patients eligible for secondary prevention are at high risk of CVD. The question is whether these patients are necessarily at a higher risk than the whole of the remaining population. This was the long held dogma but has recently been overturned. An improved understanding of the complex relationship between CVD risk factors and global CVD risk has demonstrated that a patient yet to encounter clinical CVD can have a high overall risk. Underlying risk is simply due to the combination of risk factors present. Whether or not an individual has experienced a CVD event, cardiovascular aetiology, such as atheroma and arterial calcification can be present. Secondary prevention will fail to capture these high risk patients yet to experience CVD.

*Primary prevention of CVD*

Primary prevention of CVD involves lowering CVD risk factors, hence CVD risk, in the population yet to experience CVD. When including the general population, one captures both CVD events occurring in low risk patients and the exceptionally high risk patients yet to experience disease. There is far greater scope for benefit across the population.

There are weaknesses in primary prevention. Frequently these are opposite to weaknesses of secondary prevention. The population eligible is large leading to one of two difficulties. One either targets the entire population, which is likely to be more costly and less cost effective than secondary prevention. The alternate is to employ a method of targeting; this creates difficulties in implementation with the added work load of stratification and questions over how to target. Finally, whereas the secondary prevention group might be motivated to change, this is not the case for primary prevention. They are the general population, with no experience of disease and frequently when considering CVD present no signs or symptoms.
Motivation amongst this group can be difficult, for example there is evidence of poor uptake and adherence to statins for primary than secondary prevention.\(^{59}\)

Despite a group of very high risk individuals yet to experience CVD and a greater cumulative number of events in this group, there is still a greater concentration of risk in a secondary than primary prevention group. This makes primary prevention inherently less cost-effective. Despite these weaknesses, primary prevention is vitally important. In truth, both primary and secondary prevention must be carried out; the greater question is what form the primary prevention should take.

The extensive discussion of primary and secondary prevention can imply that the two patient groups are different, especially in terms of risk. Some authors have questioned whether one should in fact draw a distinction between the primary and secondary prevention of CVD. Since the disease and risk process is the same across all individuals, all CVD prevention is in fact the same. In conclusion, prevention across the whole population is vital. The secondary prevention group is, however, a natural high risk group to target. As long as secondary prevention does not exclude the remaining population from consideration then there is little harm in such a distinction.

1.2.3 High risk and Population approaches to prevention; the Rose debate

Aside from the distinction between primary and secondary prevention, a second division can be applied to prevention. This separation was crystallised in 1981 by Geoffrey Rose in his paper entitled *Strategy of prevention: lessons from cardiovascular disease*.\(^{53}\) Rose outlined two approaches for the prevention of CVD, the *high risk* and *mass*, frequently termed *population*, approaches.

A high risk approach targets only those deemed to be at the highest risk. It aims simply to reduce the risk in this sub-population or "truncate the risk factor distribution" (Figure 1-2).\(^{63}\) Implicit in high risk prevention is an initial phase of screening required to identify the high risk group. A high risk approach to prevention can be considered as more similar to the wider
process of clinical practice. Simplistically, in routine care a clinician encounters an ‘ill’ patient and must intervene to ‘cure’ them. Similarly with high risk prevention, a population sub group is defined then interventions are administered to lower the individual risk of each member of the group. The main difference with routine clinical care is simply that the object of the intervention does not have manifest symptoms of disease.

Figure 1-2; a demonstration of the high risk and population approaches to lower systolic blood pressure

Systolic blood pressure measurements taken from combining the Health Survey for England data from 2004 to 2007 inclusive; the population approach uses a 7 mmHg reduction in systolic blood pressure for the entire population, the high risk approach reduces the blood pressure of everyone over 140 mmHg by 15 mmHg
A population approach on the other hand does not target individuals. Instead it aims to lower the entire population’s risk. Imagining that CVD risk (or a specific risk factor) is a frequency distribution, the entire distribution is translated along the x-axis (Figure 1-2). Population prevention aims to control the determinants of disease at an earlier stage than the high risk approach, effectively preventing people from becoming at high risk. Although less popular in the clinical and indeed policy domain, population level prevention has been frequently advocated by the public health community. As we shall see however, this has not always led to a widespread implementation of this approach.

**High risk prevention**

Each strategy has a number of strengths and weaknesses (Table 1-2), many of which were outlined by Rose. His original work has generated considerable debate, which at times has lead to a polarised discussion. The main advantages of a high risk strategy surround the small(er) target population. Secondary prevention, although targeted at a different group from primary prevention, is nonetheless targeted. As a result the strengths of high risk primary prevention are analogous to the strengths of secondary prevention.

<table>
<thead>
<tr>
<th>Prevention by the 'high-risk strategy'</th>
<th>Prevention by the 'population strategy'</th>
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<tbody>
<tr>
<td><strong>Advantages</strong></td>
<td><strong>Advantages</strong></td>
</tr>
<tr>
<td>1. Intervention appropriate to individual</td>
<td>1. Radical</td>
</tr>
<tr>
<td>2. Subject motivation</td>
<td>2. Large potential for population</td>
</tr>
<tr>
<td>3. Physician motivation</td>
<td>3. Behaviourally appropriate</td>
</tr>
<tr>
<td>4. Cost-effective use of resources</td>
<td></td>
</tr>
<tr>
<td>5. Benefit: risk ratio favourable</td>
<td></td>
</tr>
<tr>
<td><strong>Disadvantages</strong></td>
<td><strong>Disadvantages</strong></td>
</tr>
<tr>
<td>1. Difficulties and costs of screening</td>
<td>1. Small benefit lo individual</td>
</tr>
<tr>
<td>2. Palliative and temporary— not radical</td>
<td>('Prevention Paradox')</td>
</tr>
<tr>
<td>3. Limited potential for (a) individual (b) population</td>
<td>2. Poor motivation of subject</td>
</tr>
<tr>
<td>4. Behaviourally inappropriate</td>
<td>3. Poor motivation of physician</td>
</tr>
<tr>
<td></td>
<td>4. Benefit: risk ratio worrisome</td>
</tr>
</tbody>
</table>

*Modified from tables 2, 3, 5, 6 in* Rose, G. *Sick Individuals and Sick Populations*. Int. J. Epi, 1985; 14(1):32-8
Many benefits of high risk prevention surround the finite, smaller target population. Interventions are targeted at those with the highest risk and they will generate the greatest overall gain per individual. This produces a strong ratio of costs to benefit and therefore a more cost effective strategy. The relatively small target population also allows a higher quality, more individually tailored intervention than if aiming to cover the whole population and will expose fewer people to any potential harms of treatment.

A final area outlined where high risk prevention has strengths is in motivation to participate. Interventions delivered explicitly aim to reduce the risk status of every person involved. Each person, therefore, has a tangible gain from their involvement in the process which can motivate them to comply. Even high risk approaches to CVD prevention, however, have questions over patient motivation. Patients at high risk possess no clinical disease, and often show no symptoms. There has been little research into levels of motivation in such patients, and how these compare to patients with diagnosed disease. Increased motivation when using high risk approaches may also affect the practitioners involved. They are firstly not diverging markedly from the routine care of patients. Secondly, because there is a real gain for every patient involved, they see value in the prevention and become more involved and motivated. Practitioner confidence is an important factor in their participation in health promotion.

In order to make significant improvements to a population’s health, a high risk primary prevention strategy must do three things. It must aim to modify a prevalent risk factor; have an effective mechanism to modify it and cover a large proportion of the population who possess that risk factor. For cardiovascular disease, the former is certainly true; raised blood pressure, cholesterol levels and BMI are all widespread risk factors in the UK population. The latter two requirements enter the debate between the efficaciousness and effectiveness of available interventions. There are efficacious ways to reduce cardiovascular risk; statins for example lower lipid levels in high risk patients with no prior disease and aspirins reduce CVD events. There are then effective mechanisms to modify risk.
The last element is crucial to the success of a high risk prevention programme for CVD. Despite being efficacious, in practice, the effectiveness of prevention regimens can be limited. Two factors, the uptake of the screening process or an intervention, and its continued adherence, both severely limit the impact of a high risk prevention process. Thinking specifically about high risk CVD prevention, there are a number of stages when uptake or adherence could limit outcomes. For example, poor attendance (uptake) at the initial risk assessment, poor attendance at a weight loss programme or poor adherence to statins once prescribed will all limit effectiveness.

Evidence from well provisioned clinical trials (Chapter 3.2.2) has found limited uptake and adherence in high risk CVD prevention. In three major UK based trials, the British Family Heart Study (BFHS) had a 73 percent initial response to screening with 86 percent of those returning after one year;\textsuperscript{70} the OXCHECK\textsuperscript{71} and South East London screening studies (SELSS)\textsuperscript{72} both had 73 percent attendance at the primary screen. Adherence to statins for primary prevention is as low as 25\textsuperscript{73} and 46 percent.\textsuperscript{74} In the WoSCoP study, non-adherence with medication ranged from 5 to 15 percent in year one; by the fifth year, 30 percent had completely withdrawn.\textsuperscript{75} Given clinical trials are better resourced than routine care; rates of non-compliance should be higher in routine settings. There is, therefore, a question over whether high risk CVD prevention will reach the entire population at need and be sufficiently far reaching to produce substantial gains in population health.

High risk prevention is compatible with and at times can rely largely on pharmacological interventions. As with secondary prevention, a defined cohort is identified who can be prescribed medication such as anti-hypertensive agents, statins and aspirin to lower risk. This may be effective in lowering risk amongst the targeted group, but does nothing to resolve the underlying societal causes that generated the risk. Having treated one high risk cohort with medication, another will be produced in the next generation. The populations risk factor distribution will remain intact without further future intervention; over time there is a treadmill of high risk patients requiring treatment.
The over-use of medication in a high risk group has one further flaw. The use of medication can create new ‘patients’. The high risk group can lose their self-efficacy, simply relying on their general practitioner (GP) and the medication to lower their risk. For the successful lowering of CVD risk the opposite is in fact required, a highly motivated, self-efficacious participant.

The last major weakness of high risk prevention echoes the major weaknesses from secondary prevention. Although there is a higher rate of cardiovascular disease in the high risk group, most events still occur in the low to moderate risk. This, again, is simply due to an accumulation of risk. There are significantly larger numbers at lower levels. This is exacerbated by the high prevalence of CVD. As pointed out by Rose, people at the lowest levels of CVD risk are still eventually highly likely to die from CVD. Everyone is at a relatively high risk of cardiovascular disease in their lifetime.

Population prevention
In order to overcome this final, major weakness of high risk strategies for prevention, the solution is to incorporate the entire population. There is one important consideration in population prevention. A programme covering an entire population does not need to make as large an individual change per patient, compared with a high risk method, to make significant improvements to the health of a population. A population approach to primary prevention will cover all levels of risk and can potentially intervene in the entire population that will proceed to experience CVD.

Aside from covering the entire population, population approaches also overcome a number of other weaknesses of high risk strategies. They firstly possess a greater potential for lasting change. Population interventions have the ability to alter social norms and change the environment. These systematic, fundamental changes will prevent the creation of high risk individuals, therefore stopping the need for continued intervention. This is best explained through an example. The largest recent population health intervention in the UK was the 2007 ban on smoking in public places. Having implemented the legislation in 2007, this will
shield the population from the health consequences of second hand smoke, not only when first implemented but for as long as the legislation is in place.

Rose quotes as a major strength of the population approach, that it has a large potential to do good within a population. I shall give examples of modelling data to support this below. One concern over population prevention is that they are inherently slow processes with a considerable lag in time between the interventions and any tangible health gain. Recent findings contrary to this have indicated considerably faster impacts, especially concerning dietary changes.

Population-wide interventions have a sound theoretical support, but flaws arise when implemented. A cluster of weaknesses surround what Rose called the *prevention paradox.* By its very nature, a population approach does not require a large risk reduction in every individual; instead it relies on an accumulation of small reductions over the whole population. Despite a great potential for gain in a population, no one individual will receive any great, possibly not even any perceptible benefit. This, combined with the fact the majority of participants are ‘well’ individuals and might not understand the need for health changes, means it can be difficult to motivate the population to partake. This would especially be true if the intervention involved harm or potential harm, a participant will be far less likely to accept the risk for so little gain.

Many population interventions require no direct public involvement. The intervention instead targets environmental change, making public involvement passive. For example, they can target the food industry, for instance limiting the salt content, to passively modify the population’s diet. Finally if individual changes are required, incentives and deterrents can be introduced to aid behaviour change.

Finally, with no tangible gain per patient, it can also be difficult to motivate a clinician to be involved. With population approaches one begins to break from medical practice. One no longer focuses on a single patient, and cannot expect an individual to ‘get better’. Crucially
however, population prevention frequently does not require clinical involvement; indeed it uses far broader public health and legislative changes. The motivation of individual clinicians for the process is therefore less of a concern.

*The Rose debate; 1981 and now*

At the core of high risk prevention is the ability to identify the high risk population. At the time of Rose, cardiovascular risk prediction was dependent on individual risk factors. Neither an understanding of the cumulative effect of risk factors nor of the continuous relationship between risk factors and risk had been fully elucidated. Since then there have been major advancements in CVD risk prediction. In other words, the *Rose debate* might not be the same debate in 2011 as it was in 1981.

Despite their flaws (see chapter 4.3), cardiovascular risk scores have become more established within practice and advanced in their methodologies. They possess a greater ability to quantify global risk, and therefore may dramatically increase the power of high risk CVD prevention. Some have indeed gone so far as to argue that the existence of CVD risk scores alleviates the need for the population-wide prevention of CVD. Higher quality risk scores will improve high risk prevention, with fewer CVD events missed in the screening process.

To say CVD risk scores nullify flaws in high risk prevention, stopping the need for population approaches, is however a dramatic overestimation of their power. Weaknesses come in two levels, the conceptual and the practical. CVD risk scores only accurately predict high risk. They have limited power to predict the moderate/low risk patients who will experience a CVD event, which as we saw above is common. A second problem is that the use of CVD risk score is still a highly reactive method of prevention; they do nothing to proactively stop the accumulation of risk. Even using a risk score with 100 percent predictive accuracy, there would be a need to address the whole population. From a practical view, risk scores, despite improvements, still do not predict risk perfectly. In reality risk scores will miss out genuinely high risk patients, therefore an over-reliance can limit the scope of prevention.
A second recent advance has impacted the Rose debate, with the potential to change the balance of the discussion through promoting the effectiveness of high risk prevention. The ability to intervene and react to risk is an integral component of CVD prevention. Since 1981 notable advances have been made in effective therapeutic agents. The most prominent example is statins. We now have widely available and effective agents for lowering lipid levels which has the potential to promote the power of high risk prevention.\textsuperscript{86}

**Modelling studies of CVD prevention**

Alongside theoretical support advancing the cause of high risk approaches, especially the two recent advances described previously, two influential articles showing, or that were interpreted as showing, the strengths of high risk prevention were published in the 2000s. Modelling by Murray et al.\textsuperscript{87} suggests that high risk interventions, especially multiple risk factor reduction based on global risk, had a far greater potential impact in saving disability-adjusted life years (DALYs) than population approaches including, the reduction of salt intake through legislation. They did, however, find population strategies considerably less costly and more cost effective. Modelling a situation of scarce resources, population interventions proved more effective than the high-risk. They further acknowledge that population approaches might have greater impact than modelled under their assumptions.

The second piece of work was even more supportive of the high risk approach and outspoken in its conclusions. Manuel et al.\textsuperscript{84} suggest high risk screening and risk management to again have a far greater potential impact than a population approach. The population-based approach modelled prevented 66 percent fewer deaths than high risk. This work has subsequently, however, received substantial criticism.\textsuperscript{88} The modelling both underestimated the impact of the population strategy and overestimated the compliance with high risk treatments in clinical practice, both of which promote the relative position of the high risk approach. A further recent article has again placed targeted screening as an overall more effective approach,\textsuperscript{89} but again their model assumptions do not account for poor compliance of high risk interventions.
Modelling has supported claims that a population-wide intervention can have a greater potential impact than high risk, not merely be more cost effective. Emberson et al. estimate that a 0.3 mmol/L reduction in total cholesterol and 7 mmHg in blood pressure (both 5 percent reductions from the population means) could lead to a 26 percent reduction in risk of MI across the population. In order to achieve similar health gains, a high risk approach would be required to prescribe statins, aspirin and an anti-hypertensive to the whole population at greater than 20 percent, ten year CHD risk. Further recent modelling confirmed that with more realistic assumptions there is a strong case for the use of population approaches.

Health inequalities
The debate over the merits of high risk and population approaches goes beyond solely their overall effectiveness. The choice of prevention strategy can impact heavily upon health inequalities. Aside from the previously documented inequalities in the prevalence of CVD and exposure to its risk factors, there are parallel inequalities in both the availability and utilisation of health care. In 1971 Julian Tudor-Hart postulated the inverse care law. This states that the use of medical care is inversely related to a population’s need. This relationship is especially apparent within prevention and screening, what Acheson termed the inverse prevention law.

A high risk approach to CVD prevention has the potential to increase health inequalities. As Capewell and Graham document, this can occur at every stage of the prevention process. There can be unequal attendance at the screen; with the success of screening programmes limited in the most deprived areas and varying between ethnic groups. Likewise, the final stage of the process, the risk lowering interventions, can have limited uptake and compliance in socially deprived areas with patients unwilling to alter health behaviour.

Population approaches to prevention conversely may be inherently more equitable. Mean levels of CVD risk factors correlate positively to the prevalence of CVD across a population. If a population approach reduces risk factor levels equally, then arithmetically, overall inequalities will be reduced. One of the main limitations with high risk prevention is
that the poorest health behaviours occur in the most needy.\textsuperscript{101} High risk prevention relies on the actions of a participant- \textit{attending} the screen, \textit{adhering} to the medication for example. These actions are less likely to be present in the neediest; therefore they are inherently likely to receive a poorer outcome, exacerbating inequalities. A number of population interventions, however, do not rely on active health behaviours. Instead they alter the whole environment and as described above, act passively on a population. Such interventions are much more likely to act equally on the entire population, therefore reduce inequalities.

Modelling by Kivimaki et al.\textsuperscript{91} applied the two prevention approaches to a large UK based cohort. They compared a population approach, uniform risk reduction, to a high risk approach using best estimates of compliance and risk reductions from the literature. The population approach produced both greater overall greater reductions in risk, and a greater reduction in social inequalities. There is currently strong theoretical and model based evidence that population approaches will reduce health inequalities, but as yet little empirical data to support this.\textsuperscript{64}

In 2008, Frohlich and Potvin\textsuperscript{104} released a notable critique of the population approach to prevention, specifically focusing on its relationship with inequalities. They conclude that population-wide prevention is inherently likely to \textit{increase} health inequalities unless specific measures targeting the least engaged members of society are taken. Frohlich and Potvin argue that the inverse care law acts equally within a population prevention intervention as it does within other aspects of healthcare. Those with poorest CVD health have the poorest health behaviour;\textsuperscript{101} this poor health behaviour in turn causes them to gain least from the population intervention. Individuals at the most risk gain the least, increasing inequalities.

A further criticism of the population approach by Frohlich and Potvin\textsuperscript{104} is that it fails to fully address the “fundamental causes” of disease. Current CVD risk status is the product of a life-course of exposures. Frohlich and Potvin argue that a population intervention will shift the population distribution of risk, but do nothing to alter the life-course risk trajectories of the
individuals. They finally state that Rose’s approach will concentrate on single risk factors, therefore doing nothing to address the clustering of risk factors in certain populations.

In order to overcome these concerns, Frohlich and Potvin\textsuperscript{104} postulate a third method of prevention, what they term the vulnerable population approach. This third-way is to be employed alongside a population approach, with the aim of reducing inequalities. The vulnerable population approach targets interventions, not at patients at high risk \textit{per se}, but at ones “at [a] higher risk of risk.” Examples of such target populations might include a geographic area known to be socially deprived, or an ethnic minority group which experiences poor health outcomes. Once the population has been identified, they then become the focus of interventions beyond those in the wider population. The overall goal is to remove or reduce the fundamental causes of risk, in our case CVD risk, from the population which will stop high risk individuals from being generated.

Frohlich and Potvin’s vulnerable population approach itself has weaknesses. As a concept it is not far removed from a high risk approach, it simply uses a different method to target a population. It will therefore face many of the same difficulties.\textsuperscript{105} Its success will be strongly modulated by the uptake of interventions, the identification and definition of the vulnerable population is likely to be troublesome and furthermore the approach may stigmatise those targeted as vulnerable.\textsuperscript{104} It is also limited by the ecological fallacy.\textsuperscript{106} An entire population does not possess the mean characteristics of that population. High risk individuals will be present in populations not designated as at high risk, hence being omitted from the most intensive prevention. Conversely non-high risk patients will be present in the vulnerable group; therefore focus on these will be wasteful of resources.

McLaren et al.\textsuperscript{105} issued a reply in 2010. Like them, I would argue that Frohlich and Potvin have not fully grasped the essence and potential breadth of Rose’s population approach. McLaren et al.\textsuperscript{105} draw a continuum along which all population measures sit. The continuum goes from the agentic- where individual choice is heavily involved in the success of an intervention, to the structural- where the environment is changed, passively addressing the
population. This continuum becomes useful in assessing the relationship that an intervention might have with health inequalities. Under the inverse care law, to increase inequalities an intervention must possess at least a modicum of patient choice or from the service side an inequality in provision. Assuming provision is homogeneous, inequalities in care stem from the poor health behaviours - choices. Theoretically therefore an intervention at the agentic end of the spectrum does have the potential to increase inequalities; a structural intervention on the other hand does not, as long as provision is equal.

The examples that Frohlich and Potvin draw upon as the evidence behind a population approach increasing inequalities lie at the agentic end of this spectrum. Two examples were cervical screening programmes and the provision of information to aid smoking cessation. The former relies on a decision by the patient to attend screening, whilst the latter relies on the ability to utilise information to enact behavioural change. Both of these are individual actions or choices. Their final example, that of the impacts of childhood health interventions in Brazil is inappropriately included. Although the interventions saw an initial widening of inequalities, in the long term there was an overall narrowing.\(^\text{107}\)

Frohlich and Potvin are correct that not all interventions applied to a whole population will inherently act positively upon health inequalities. Crucially however, not all population interventions will increase inequalities. Some do have real potential to decrease them. Rose describes his population strategy as radical.\(^\text{53}\) This implies a method of intervention much more akin McLaren et al.’s structural approach,\(^\text{105}\) a method likely to reduce inequalities.

High risk and population approaches are not mutually exclusive; in fact they should be complementary. The same was stated by Frohlich and Potvin about whole population and vulnerable population approaches.\(^\text{104}\) It is important to reduce risk in high risk patients. This improves individual's health and maintains the commitment of care that clinicians have for patients. Equally, if significant lasting reductions to CVD are to be made, in an equitable fashion, a population aspect to prevention is vital.\(^\text{76}\) The question should never be which the
most effective mechanism is. Instead, one must ask whether an appropriate balance between methods is reached. I shall try to answer this through the remainder of this work.

1.2.4 The changing face of high risk prevention

Global risk

The prevention of cardiovascular disease has been a constantly evolving field. Arguably the biggest single change was the paradigm shift that occurred through the 1990s. Focus moved from individuals with single raised CVD risk factors to those with high global risk. This has dramatically altered the face of CVD prevention and increased the effectiveness of the high risk strategy. This change was facilitated by two advances in knowledge. Firstly, understanding that a combination of risk factors increases a patients risk of a CVD event, and secondly that risk factors have a continuous linear relationship with risk. Patients continue to benefit in terms of risk reduction when levels of risk factors are reduced well below traditional clinical targets.

Modelling studies demonstrate the management of global risk has greater potential benefits to a population, than using lipid and blood pressure levels alone. There are, for example, greater reductions in life years lost due to CVD if blood pressure control is targeted at those with a high global risk, not simply high blood pressure. The notion of global risk is supported by an improved understanding of cardiovascular disease aetiology. Disease aetiology is in fact highly complex and is not linearly dependent on single risk factors.

Patients with the same blood pressure or cholesterol levels, can for example have up to a twenty fold difference in global CVD risk, dependant simply on other risk factors. Table 1-3 further demonstrates how overall CVD risk escalates as more risk factors are added. One risk factor alone (raised blood pressure in the first scenario) has little impact on overall risk, but levels of risk quickly rise with each additional risk factor. Clearly a single risk factor measurement (blood pressure) is not a good predictor of global risk.
Table 1-3; QRISK2 score of a 50 year old white woman with different CVD risk profiles

<table>
<thead>
<tr>
<th>Systolic blood pressure =180 mmHg</th>
<th>BMI = 23</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure =180 mmHg</td>
<td>Lipid ratio = 5</td>
</tr>
<tr>
<td>CVD risk = 8%</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Blood pressure =180 mmHg</th>
<th>BMI = 30</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure =180 mmHg</td>
<td>Lipid ratio = 8</td>
</tr>
<tr>
<td>CVD risk = 13%</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Blood pressure =180 mmHg</th>
<th>BMI = 30</th>
</tr>
</thead>
<tbody>
<tr>
<td>lipid ratio = 8</td>
<td>Diabetic</td>
</tr>
<tr>
<td>CVD risk = 30%</td>
<td></td>
</tr>
</tbody>
</table>

**BMI- Body Mass Index**

When using global risk to identify patients for prevention programmes, one must define a cut-off value at which an individual becomes eligible for intervention. A balance is required between a low cut-off generating high management costs and a high cut-off failing to capture the at risk population. When the Joint British societies (JBS) changed their guidance from stating a 40 percent cut off, to 20 percent ten year CVD risk, the number eligible for statins in the UK increased from 1 to 3 million. The UK currently uses a 20 percent ten year risk as the definition of high risk, and therefore as the threshold for a number of interventions. This has altered over recent years; JBS1 guidance used a 30 percent ten year CHD risk, which translates into approximately 40 percent CVD risk, the cut-off revised with JBS2 recommendations to 20 percent CVD risk.

**Lifetime risk; the future of high risk prevention?**

Since the adoption of global CVD risk and cardiovascular risk scores into clinical practice, there has been a reliance on medium term risk estimates, usually over ten years. Estimating risk over a relatively short time period has limitations, many of which will be outlined in chapter 4.3. The major criticism surrounds the prediction of global risk in the younger population. Put simply, no matter how high their levels of risk factors, young people do not have a high short term risk of CVD. Age is the strongest risk factor for CVD. In order to effectively reduce overall cardiovascular risk however, it may be beneficial to start risk lowering interventions at as early an age as possible. In order to overcome the
weaknesses of conventional risk scores in the young and successfully risk stratify them, lifetime CVD risk scores have been proposed as a more effective tool for screening.\textsuperscript{117}

Debate surrounding lifetime risk prediction has existed for nearly a decade, with calls for it to replace short term risk scoring growing in recent years. Lifetime risk prediction is not without criticism, which I discuss further elsewhere (chapter 4.5).\textsuperscript{118} The most notable weakness is that with the high prevalence of CVD across the entire population, everyone is in fact at a relatively high lifetime risk. As a result, lifetime risk scores produce homogeneously high risk predictions, making it difficult to target patients.

A danger may follow; a large high risk group, potentially the majority of the population can then become medicalised through drug based interventions to lower risk. This, although potentially bad for patients, may be an attractive proposition for pharmaceutical companies. A larger population eligible for medication creates a larger market to sell drugs too, therefore can increase profits. Clinical bodies, including those responsible for research and guidance, must resist pressure from pharmaceutical companies to promote health care suited to their vested interests. These pressure can be in the form of direct lobbying, of indirectly through industry sponsored research.\textsuperscript{119}

Lifetime risk estimation is a new development in risk prediction. Nonetheless it has already received backing to replace short term scores as the primary method of stratification.\textsuperscript{116} Others suggest that lifetime risk has a place alongside its shorter term relatives, with particular use in the communication of risk to patients.\textsuperscript{118} Crucially, lifetime risk scores have not been widely evaluated in practice; especially from a public health perspective when used to risk stratify populations. There are also unanswered questions surrounding their ability to discrimination between levels of risk.

If a dual approach, combining high risk and population prevention, is the most effective strategy for CVD prevention,\textsuperscript{87 90} lifetime risk may be further limited. Life time risk estimates produce greater numbers in high risk groups. As part of a dual approach to prevention, an
expanded high risk approach seems unlikely to be beneficial. The high risk arm is simply required to prevent immediate risk, whilst the population arm will make the telling population level changes. Based on the lack of current evidence, the widespread introduction of lifetime risk scoring currently must be treated with caution, although they have potential, especially for risk communication.
Chapter 2; UK policy and clinical guidance for CVD prevention

2.1 A history of CVD prevention policy

2.1.1 The UK

In the UK, there have been no previous primary prevention campaigns for CVD even close in scale to the NHS Health Check. Through the early history of the NHS, structured CVD prevention had never been a priority. Over the last twenty years, however, UK governments have begun to recognise its importance. In 1992 the white paper *the Health of a Nation* set out five key disease-areas, targets when looking to improve the nation's health.120 Amongst these priorities were both CHD and stroke. Guidance supporting the white paper discussed health promotion as a fundamental to improving CVD outcomes.121 This initial inclusion of CVD prevention in UK policy documentation did not, however, lead to a formal national strategy. Health care providers were not obliged by central government to act upon the guidance. Instead it was intended to help develop local services.

After the 1997 general elections, the 1998 white paper *Saving Lives: Our Healthier Nation*122 outlined CVD as a national priority. It included the explicit aim to cut CVD mortality in the UK 40 percent by 2010, stating that population-wide risk factor reductions, not treatment, was the central mechanism in reaching this goal. The white paper was quickly followed by the 2000 NSF-CHD,123 a document designed to improve the quality and equity of patient care throughout the NHS. The NSF-CHD set out as one of 12 standards of care the aim to;

*identify all people at significant risk of CVD but who have not developed symptoms

and offer them appropriate advice and treatment to reduce their risks.*123

Despite this explicit reference to primary prevention, it was still left to clinicians to implement existing clinical guidance.124
In 2004, *Choosing Health*\textsuperscript{125} outlined a range of public health goals, a number of which had implications for CVD. This white paper focused heavily on giving patients informed choice which in turn gives an ability to make good health decisions. Its aims were largely focused on primary prevention; strengthening smoking cessation services; reducing obesity, especially through increased opportunities for exercise and reducing salt intake through partnerships with industry. Choosing Health, importantly, paved the way for the *Smokefree* England, legislation which was formalised in the 2006 Health Act. This was a population level public health intervention, with the marquee initiative of banning smoking in public places, and was designed amongst other things to prevent CVD.

By 2007, CVD prevention became firmly embedded within the framework of the NHS with its inclusion in the Public Service Agreement,\textsuperscript{126} a document outlining the organisation’s broadest strategic aims over a three year spending cycle. However, the largest progress at a national level, and still the area of greatest focus within the NHS, was secondary prevention. Within the QOF, introduced in 2004, CHD and stroke/ TIA were two major disease areas within the clinical domain. Indicators within these disease groups give financial incentives to general practice, aiming to improve and standardise the care of patients with established disease. A major focus of the QOF is to promote good clinical practice (regular blood pressure monitoring for example) and control risk factor levels.

The QOF was a major investment in secondary disease prevention but initially did little to help reach the NSF-CHD target of identifying high risk individuals without clinical disease. The 2009/10 revision of the QOF added two ‘primary prevention’ indicators; however these involved newly diagnosed hypertensive patients and therefore arguably do not address *primary* prevention. The QOF does contain three indicators in the organisational domain which refer to patients without existing disease. One promotes the recording of smoking status in the population aged 16 years and over, and two indicators promote blood pressure recording in over 45s. A further indicator covers recording patients with a body mass index (BMI) over 30. These latter four indicators are all aspects of primary prevention, but do not
represent a coherent, comprehensive vascular risk assessment. Further, the number of points awarded within the QOF (and therefore the money available to GPs) is very small compared with the secondary prevention indicators.

2.1.2 Internationally

The Australian health system offers subsidised, universal health care across a number of settings. The publically funded body, Medicare, fully reimburses patients for all hospital activities. Co-payment is required for general practice and community activity (with Medicare funding approximately 75 and 85 percent respectively). There are means tested exemptions from co-payment but all are advised, if possible, to hold private medical insurance. Australia has a number of current schemes funded by Medicare which are analogous to a ‘health check.’ As a group they are named the Medicare Benefits Schedule health assessments.

The Aboriginal and Torres Strait Islander Adult Health Check is subsidised for all Aboriginal and Torres Strait Islanders aged 15 to 74 years. It includes the recording of a number of CVD risk factors, including blood pressure and waist circumference, although is targeted primarily at the risk of diabetes. Relatively few of these checks are carried out, with an estimate of 6.1 per 100 eligible population per year. A further diabetes risk assessment is available every three years, for the entire population aged 40 to 49 years.

A third health check in the general adult population is available at the age of 45 years. All patients considered to be at risk of a chronic disease (i.e. those who display one risk factor) are eligible. This assessment incorporates CVD risk; however does not focus solely on it. Outside of the Medicare health assessments, there have been audits and initiatives across the nation aimed to increase GP involvement in primary prevention. None of the health checks, currently offered in Australia, have universal coverage. Australia also has ongoing population initiatives. These include regulations on food labelling, the exemption of fresh produce from taxation, public awareness campaigns surrounding CVD risk, regulations for smoke-free areas, advertising restrictions and warning labels for tobacco.
After the 2006 federal election in Canada, the Heart Health Strategy was established. This task-force aimed to lead improvements in hypertension and CVD surveillance in Canada. The culmination of this group’s work came with the publication of the *Canadian Heart Health Strategy and Action Plan* in 2009.\(^{129}\) The document had great breadth, incorporating recommendations on many aspects of CVD care and prevention. It called for improvements in the environment, conducive to CVD health; reform of the health service to allow integrated CVD prevention; and the development of providers to carry out preventative care. It also explicitly called for the systematic, universal screening of the Canadian population aged 45 and over, with appropriate evidence based follow-up.

The Canadian health system is especially lacking in information systems required to undertake global screening. An early phase of the nation’s progress towards an efficient prevention strategy will therefore be the creation of a national CVD information strategy.\(^{129}\) The older Canadian Heart Health Initiative, running from 1986 to 1991 looked to support preventative policy and infrastructure but focused largely on community education.

The United States does not possess a formalised policy for CVD prevention, indeed some argue that its entire health system’s structure is not conducive to prevention.\(^{130}\) There is a large body of guidance produced by organisations including the American Heart Association (AHA) and the Centers for Disease Control (CDC),\(^{131,132}\) however this is not systematically implemented. There has been little federal organisation in prevention, although Medicare recipients are eligible for a CVD prevention check, the ‘annual wellness check’. A number of other parallel initiatives fringe on CVD prevention. The *Patient Protection and Affordable Care Act* looks to lower patient cost sharing for preventative therapies. This act may prove significant for preventative medicine in the US and begin a transition to a health system more amenable to prevention. The Diabetes Prevention Program looks to improve diet and physical activity in those at the highest risk of diabetes nationally.

In September 2011, the Department of Health and Human Services (HHS) launched the *Million Hearts initiative*, an effort to further formalise CVD prevention in the United
States. This initiative spans federal and local government; the Medicare and Medicaid services; private sector providers and other non-profit organisations, although is lead jointly by the CDC and HHS. It has two main goals; firstly, to improve the management of high risk patents in a clinical setting. This involves the promotion of an ‘ABCS’ strategy; namely aspirin prescription, blood pressure and cholesterol control and smoking cessation for all at the highest risk. It hopes to nationally standardise practice and incorporate CVD prevention into physician performance metrics. It will mandate preventative health checks within Medicare, and promote these across all private insurers. It secondly aims to strengthen community based prevention nationally and locally. Aims include producing a national menu labelling policy, further control of salt and trans-fat intake; and providing grants for local community prevention.

Million Hearts will not systematically screen an entire population. It is, nonetheless, a major step for the United States. It displays real targets (i.e. preventing one million events in five years). It also attempts to bring together often disparate elements of the health system, with a common goal of CVD prevention. It reinforces pre-existing AHA guidance, and will look to seize the opportunities within the Patient Protection and Affordable Care Act. Its strengths differ from the NHS Health Check, but suit the current US health system. It is less bold and novel; however may be effective in an organisational capacity, empowering CVD prevention.

Greater advances to date have been made at a state level. Examples include formal workplace screening in Kansas and CVD risk assessment in South Carolina. Areas, including California and New York City have lead the way in population interventions, with the early adoption of smoking and trans-fat control (Chapter 3.3.3/4).

Internationally, outside of the UK, New Zealand has made some of the greatest advances in the primary prevention of CVD. The New Zealand Ministry of Health set explicit targets of improving the proportion of patients having complete CVD risk assessment, and by 2012 hopes to have assessed 80 percent of the eligible population. The Ministry is also keen to
promote the compliance with the national guidance on prevention, a strong and thorough document. In 2006, the *Primary Health Organisation Performance Programme* was established in New Zealand. This offers financial payments to primary care for performance in a range of activities. More recently, the recording of global CVD risk has been added as an indicator. Although the programme aims to reduce health inequalities, it is voluntary and requires minimum quality standards before primary health organisations can enlist. As a result this does not give universal coverage. Further, although national, it does not have the depth of risk assessment and management offered by the NHS Health Check.

### 2.2 NHS Health Check programme

In April 2008, the Prime Minister, Gordon Brown, outlined a new national programme, establishing systematic vascular risk assessment and management in England. This built upon ideas first mooted by Sir Muir Gray and the National Screening Committee (NSC) two years earlier. The programme was heavily endorsed by the central Labour government, with subsequent suggestion that the evolution or at least the speed at which the programme was implemented was politically motivated. Further details of the programme quickly followed when the Department of Health published *Putting Prevention First*. The programme initially received the working title of *Vascular Checks*, later taking the name *NHS Health Check*. Over the following year, further guidelines outlined the programme's scope and best practice. The initial roll-out of the programme began in April 2009, with a gradual implementation. The expectation is that the programme will be fully implemented in the 2012/13 financial year. Being a five year rolling programme, this means that 20 percent of the eligible population must be invited for a Health Check in 2012/13.

The NHS Health Check programme seeks to offer a vascular risk assessment (the Health Check) to the entire population of England aged 40 to 74 years, who do not have established vascular disease. Valid exclusions consist of patients with CHD, stroke (and TIA), diabetes,
chronic kidney disease (CKD- stages III-V), atrial fibrillation, heart failure, diagnosed hypertension, hyperlipidaemia and peripheral arterial disease (PAD). The programme assumes that excluded patients (the majority of who lie on QOF registers) already receive adequate management of their vascular risk.

The programme is composed of two main stages; risk assessment and management. Organisation of the NHS Health Check is currently the responsibility of primary care trusts (PCTs), NHS organisations responsible for the development of primary and community health services and the commissioning of secondary care. PCTs have considerable scope to locally design and implement the programme. The Department of Health allowed this malleability to create services suited to local population needs.

The main area that guidance allows for variation is in the venue of the risk assessment. The Health Check must be offered to the entire eligible population but can occur across a range of settings, as long as minimum standards are met. It can further be offered to a wider target population, for example a larger age group. In practice the majority of the programme work load will be undertaken in general practice, with additional support from pharmacies and community activity. Many PCTs are using similar programme models; employing health care assistants to carry out risk assessment in general practice, with pharmacy and community projects designed to extend the Health Check to hard-to-reach populations.

The content of the Health Check is open to local variation; although the Department of Health requires a minimum body data to be collected (Table 2-1). Local providers can include additional tests and measurements where appropriate, for example some have incorporated alcohol screening. In addition to the minimum dataset (Table 2-1), blood glucose testing must be carried out for patients considered to be at high risk of diabetes and serum creatinine tests if at risk of CKD. Patients considered at risk of diabetes have a BMI of greater than 30 mmol/l (27.5 mmol/l if from any Asian ethnic group) or have a blood pressure greater than 140/90 mmHg, with the latter the sole indicator of CKD risk. All data within the Health Check is expected to be recorded at one clinical encounter.
Table 2-1: Core components of the NHS health Check minimum dataset

<table>
<thead>
<tr>
<th>Demographics</th>
<th>Health Check programme</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower Super Output Area (LSOA) of residence</td>
<td>Eligible population</td>
</tr>
<tr>
<td>Age at assessment</td>
<td>Invitation offer sent</td>
</tr>
<tr>
<td>Gender</td>
<td>Commissioner</td>
</tr>
<tr>
<td>Ethnic Category</td>
<td>Provider</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Personal observation</th>
<th>Information and Advice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Activity location</td>
<td>General lifestyle advice administered</td>
</tr>
<tr>
<td>BMI</td>
<td>Smoking cessation advice if smoker</td>
</tr>
<tr>
<td>Systolic/ diastolic blood pressure</td>
<td>Weight management advice</td>
</tr>
<tr>
<td>Cholesterol to HDL ratio</td>
<td>Physical activity brief intervention given</td>
</tr>
<tr>
<td>Total cholesterol level</td>
<td>Signposting to physical activity service</td>
</tr>
<tr>
<td>Smoking status</td>
<td>Signposting to stop smoking service</td>
</tr>
<tr>
<td>CVD risk score</td>
<td>Signposting to weight management service</td>
</tr>
<tr>
<td>Physical activity level</td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>Prescriptions</th>
<th>Referrals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Statins prescribed</td>
<td>Referrals to physical activity service/ stop</td>
</tr>
<tr>
<td>Anti-hypertensive prescribed</td>
<td>smoking service/ weight management/</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Diagnoses</th>
<th>Further Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Formal diagnosis of CKD (stage III-V)/ Type II diabetes/ hypertension/ non-diabetic hyperglycaemia</td>
<td>Assessment for- diabetes / Serum creatinine / hypertension / fasting lipid / IFG/IGT</td>
</tr>
</tbody>
</table>

Guidance outlining the composition of the Health Check was released before programme roll-out. There were, however, considerable delays in the publication of the minimum dataset, over two years after initial roll-out. This has created problems for local commissioners and service providers. Many have designed a local Health Check programme, only to find modifications are required to meet the Department of Health minimum standards.

The NHS Health Check is more than merely a risk assessment programme; risk management is an integral component. There are three broad flows of patient though the system (Figure 2-1). Firstly, as a result of a Health Check, patients will be diagnosed with clinical disease, hypertension or diabetes for instance. Patients found, during the Health Check, to show signs of disease are referred to general practice to receive formal diagnosis. Once diagnosed these patients become ineligible, leave the programme, and are placed on relevant disease registers for their continued care within general practice.
The second group of patients are those designated as at high risk of CVD, at greater than or equal to a 20 percent ten year risk using either the QRISK2 or JBS2 risk scores. These patients also leave the programme. They are referred to general practice where they join high risk registers and undergo annual follow up to modify their risk. Within general practice, under pre-existing clinical guidance, this population becomes eligible for the prescription of statins.

The remainder of the population are managed directly within the Health Check programme. The first line of management for a patient is the communication of risk, combined with a tailored brief lifestyle intervention bespoke to their level of risk. These are carried out at the time of the Health Check and are given to all participants. In addition, within the low and moderate risk groups (<20%) those with single raised risk factors are signposted or referred...
to more intensive interventions (Table 2-1). The options for referral are namely smoking cessation services, weight management, exercise interventions or impaired glucose tolerance (IGT) lifestyle interventions. The entire low to moderate risk cohort remains eligible for the programme and will enter a five year rolling recall.

Despite considerable local freedoms in the programme, there will be a degree of consistency across the country. All must comply to the same minimum standards, all are subject to the same clinical guidance and best practice is shared between areas through a national learning network. Across a number of areas in England, PCTs have commissioned local vascular screening services before the official roll-out of NHS Health Check. Most notably, larger programmes include the Sandwell Project, Heart MOTs in Birmingham, the Deadly Trio project in the Birmingham, the Big Bolton Health Check, the Healthy Heart programme in Knowsley and St. Helens and Test your Heart in Doncaster. Many of these commenced between 2007 and 2008, and have subsequently taken on the identity of NHS Health Check. There are further examples of the implementation of vascular screening in a trial setting, and the details of these will be outlined below (chapter 3.2.2).

The Department of Health has two overarching goals for the Health Check programme. They firstly aim to reduce the overall burden of CVD, with modelling suggesting a reduction of 650 CVD deaths and 1,600 fewer MIs and strokes. As outlined in chapter 1.1.4, this will not only have positive impacts for the nation’s health and well-being, but could lead to substantial cost savings for the health system. The second major goal of the programme is to narrow health inequalities in England.

If the programme universally reduces CVD risk factors across all societal groups, with the increased burden in disadvantaged groups, they will receive the greatest global risk reduction. The ability of the Health Check to reduce health inequalities has, however, divided opinion. If the programme’s uptake, adherence and therefore impact are equal, it will reduce inequalities. However, it is this equality that has been questioned. We have already seen the rationale behind how the inverse care law can negatively impact health inequalities.
If this relationship is maintained within the programme, it will increase inequalities. I shall further outline the evidence of inequalities in preventative medicine in chapter 5. Whether or not the programme actually exacerbates health inequalities, it may be necessary at the very least to provide extra resources to promote equitable uptake.

The NHS Health Check is a primary prevention programme, but there has been debate about whether it is a population or high risk strategy. Although this might seem a superfluous argument, it is important in terms of the extent of the programmes’ benefit and its relationship with inequalities. Some see it as simply a “screening and treatment” initiative. Although the entire population is eligible (i.e. the programme is *universal*), the most effective interventions are only available to those at high risk. Hence, it is a high risk programme.

The counter argument states that the entire eligible cohort receives intervention at the time of the Health Check. The Health Check is, therefore, a population level intervention, with the potential to modify risk universally. Realistically, interventions available to the entire population are minimal in scope. The use of brief interventions is supported by guidance from the national institute of clinical excellence (NICE). There are, however, questions over the extent of their effectiveness when implemented in a non-healthcare setting and by non-clinical staff. Experience from UK primary prevention trials shows the effectiveness of motivational interventions, for example, smoking cessation, can be severely limited when carried out by practitioners not experienced in the techniques. Evidence from smoking cessation points suggests limited belief, motivation and competence from clinicians not regularly providing the service. This may be equal across other lifestyle interventions. Research into the universal effectiveness of brief interventions is required.

A further oversight might be the effectiveness of risk communication. Effective risk communication is not a trivial process (chapter 3.2.4). Both its difficulty and importance may have been underestimated which will limit the extent of the population-wide intervention. There is therefore only limited intervention across the entire population; accounting for non-
attendance and adherence there is in fact none. The Health Check is fundamentally an extension of a high risk prevention approach, albeit with a universal reach.

CVD prevention has undergone a substantial paradigm shift in the last 20 years, moving focus from individuals with single raised risk factors to those with high global risk (chapter 1.2.4). Superficially, the Health Check programme is reliant on global CVD risk. All patients have their global risk recorded, with management tailored accordingly. In reality there are questions over the extent to which global risk is used. The brief lifestyle intervention is tailored to risk status and statin eligibility defined by global risk. The remaining programme outcomes are, however, defined by single risk factors. Blood pressure management, for example, is only available to those diagnosed with hypertension, despite evidence that CVD risk might be a more efficient marker for treatment. Ignoring global risk, despite growing evidence to support its efficiency, is confusing for health professionals and may prove to be a further weakness in the programme.

2.2.1 NHS Health Check programme in Ealing

The borough of Ealing

The borough of Ealing is located in North West London. It is the third largest borough in Greater London with an estimated population of 317,000 in 2009. The borough is noted for spanning the divide between inner city and outer London, and hence has a complex and varied set of health needs. The health care needs of the population are provisioned for by NHS Ealing, a PCT co-terminus to the borough. NHS Ealing acts as the provider of primary care through 85 general practices, and additionally commissions secondary care and specialist services. The PCT has a greater number of patients registered in general practice than reside within the borough, with 350,000 in 2009. This inflation is caused largely by the presence of ghost patients on practice registers (registered patients who have left the area). The PCT has made a concerted effort to remove ghost patients, with for example the registered population falling by 9,000 patients between April 2008 and 2009.
Ealing is highly ethnically diverse area, according to the 2001 census the fourth most diverse borough in England: Estimates from 2007 place 41 percent of the population in black and ethnic minority (BME) communities. The predominant BME community in Ealing is the Indian or British Indian, making up an estimated 15 percent the population, although overall the ethnic make-up is diverse (Table 2-2). The ethnic make-up is not only diverse, but also heterogeneous across the borough (Figure 2-2); at the Lower Super Output Areas (LSOA) level, some areas have 80 percent BME population whilst others fewer than 10 percent. The largest south Asian populations cluster in the south west of the borough in Southall. Indeed one area, Southall Broadway, is considered to be the most none-white area in England.

Ealing has highly deprived areas within the borough, and relatively few highly affluent areas, although overall the borough lies towards the centre of the socioeconomic gradient. Overall using the 2007 indices of multiple deprivation (IMD) it is the 75th most deprived of 354 English local authorities. Out of the 195 LSOAs in Ealing, 44 (approximately 23%) lie in the most deprived fifth nationally, with only 6 in the most affluent. The deprivation is relatively evenly distributed across the borough, with a suggestion of the most affluent clustering in the centre (Figure 2-3). Compared with England as a whole, Ealing has a marginally younger population; Ealing has a similar proportion of its population aged 19 and under, but fewer aged 65 and over (10.8% compared with 16.3%), hence a greater proportion of working age.
Figure 2-2: the percentage of the Ealing population of Asian or British Asian origin

Source: ONS, Super Output Area Boundaries. Crown copyright 2004. Crown copyright material is reproduced with the permission of the Controller of HMSO; Data from 2001 UK Census

Figure 2-3: the national fifths of IMD Score in Ealing by LSOA

Source: ONS, Super Output Area Boundaries. Crown copyright 2004. Crown copyright material is reproduced with the permission of the Controller of HMSO; Data from ONS (Neighborhood Statistics)
**NHS Health Check programme in Ealing**

*Table 2-3; Guidance for minimum Health Check from the Ealing VRA-LES*[^157]

<table>
<thead>
<tr>
<th>Screening</th>
<th>Follow-up for raised risk</th>
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</thead>
<tbody>
<tr>
<td>Family history of CHD in 1st degree relative</td>
<td>Blood pressure</td>
</tr>
<tr>
<td>Family history of diabetes in 1st degree relatives</td>
<td>BMI</td>
</tr>
<tr>
<td>BP measurement</td>
<td>Waist hip ratio if BMI &gt; 30</td>
</tr>
<tr>
<td>Smoking status</td>
<td>Review of smoking status &amp; family history</td>
</tr>
<tr>
<td>BMI</td>
<td>Repeat blood tests as appropriate.</td>
</tr>
<tr>
<td>Waist hip ratio if BMI &gt; 30</td>
<td>Confirm patient has attended any of 16 projects from Choosing Health Programme/ smoking cessation service if referred</td>
</tr>
<tr>
<td>Fasting venous plasma glucose to determine diabetic status</td>
<td></td>
</tr>
<tr>
<td>Total cholesterol and HDL</td>
<td></td>
</tr>
<tr>
<td>Ethnicity (including if patient declines)</td>
<td></td>
</tr>
<tr>
<td>Referral to Choosing Health Programme and/or smoking cessation counsellors where appropriate</td>
<td></td>
</tr>
<tr>
<td>A risk score obtained using either QRISK2 or JBS2</td>
<td></td>
</tr>
</tbody>
</table>

**Additions in 2011 revision**

| Lifestyle counselling as appropriate | |
| Physical activity levels as per the General Patient Activity Questionnaire (GPPAQ) | |
| Record information on the method of invitation | |
| Those identified with suspected impaired glucose tolerance or Chronic Kidney Disease | |

NHS Ealing started the Health Check programme in September 2008, six months before national roll-out. Initially the programme was provided entirely within general practice, with hope to include alternate providers once established. Practices received additional funding for the work undertaken in the programme, with remuneration organised through a locally enhanced service (LES; named the vascular risk assessment (VRA)LES- Table 2-3).[^157] The VRA-LES was ratified by the PCT and local GPs in June 2008, with a three year period of service (1st September 2008 to 31st August 2011). A year extension was subsequently agreed, starting 1st April 2011 to align the service to the national programme. The VRA-LES pays practices £15 per patient receiving the comprehensive vascular risk assessment, with a

[^157]: VRA-LES= Vascular risk assessment locally enhanced service
bonus of £200 per one thousand patients if more than 80 percent of the target population are screened. Components required for a risk assessment are outlined in Table 2-3.

Initially, in Ealing the age range for the programme was extended beyond the national minimum, from 35 to 74 years. With the large south Asian population in Ealing having raised CVD risk, the PCT felt starting screening at the age of 35 was important to capture enhanced risk early. The VRA-LES in Ealing had a number of further extensions to the national programme, notably a number of chronic disease groups excluded from the Health Check programme were to be invited for the initial risk assessment. These groups were namely hypertension, CKD and hyperlipidaemia. The PCT felt that despite their inclusion in the QOF, these patient groups would benefit from additional formal CVD risk assessment.

The NHS Health Check programme in Ealing, under the direction of the VRA-LES was to be implemented in a staggered fashion. Over each of the first five years, the PCT invited a different target group, summing to give the entire eligible population. The end of this roll-out period coincides with the Department of Health target for full roll-out, from when on the PCT will annually invite one fifth of its eligible population. The VRA-LES targeted ‘high risk’ (see below) and hypothyroidism patients in year one; patients on the QOF obesity register in year two; on the CKD and hypertension registers in year three and then equally dividing the remaining population between years four and five.

The high risk population targeted in year one was identified using the Oberoi primary prevention software. The Oberoi software, created for CVD risk assessment, was run in each general practice database, before the programme started. The software extracted Read coded patient data for those aged 35 to 74 years, without a diagnosis Read code for CVD (CHD, stroke/ TIA) or diabetes. The data extracted consisted of CVD risk factors, including the date and value of the last reading, and demographic variables. The data were namely blood pressure, total cholesterol and HDL measurements, BMI, smoking status, family CVD history, hypertensive status, age, sex and ethnicity. Additionally, the postcode of residence was extracted and substituted with LSOA codes. The software substituted missing risk factor
data, using default values from the Health Survey for England (HSfE), matched using age and sex. The software finally applied the Joint British Societies 2 (JBS2) CVD risk score to the data. Those with a risk score greater than or equal to twenty percent ten year risk, were designated as at high risk. These people were targeted for screening in year one.

Once a patient has been invited and successfully completed a risk assessment, a specific Read code is entered into the patient electronic medical records (EMRs). Additionally, if confirmed to be at high risk a further Read code is added; this group of high risk patients proceed to receive annual follow-up within the general practice. After the baseline run of the Oberoi software, the general practices are required to make annual data extractions. They must then provide the PCT with data outlining all of the patients eligible, the Health Checks completed and all other associated activity from the preceding year.

At the outset of the project, NHS Ealing envisaged offering the Health Check in a greater range of settings than general practice. One hope was to implement a formal network of pharmacies, under the supervision of the PCT, to carry out Health Checks. The selection of pharmacies would be targeted at areas of poorest uptake in general practice, with greatest health needs, and those able to meet the quality standards of the PCT. The main aim of this extension was to help the programme fully engage hard to reach populations. As yet there is no formal pharmacy based screening in Ealing; instead amendments were made to the LES in 2011 allowing GP surgeries to sub-contract work to pharmacies.

The PCT has undertaken some screening in alternate settings. In conjunction with the British Heart Foundation (BHF), a community based programme was carried out during year 2 of the VRA-LES. BHF nurses undertook CVD risk assessment at dedicated events in town halls, places of worship and work. Results were fed back to general practices, via the PCT, using paper records and were hoped to be entered into EMRs. In total, 561 were screened by the BHF, or whom 374 were eligible for the Health Check programme.
2.3 Health Check under the UK coalition government and the use of Nudging for health promotion

The NHS Health Check programme was designed under a Labour government and received considerable backing from senior party members.\(^{158}\) Around the 2010 general election, however, there was uncertainty over its future. There were indications any future Conservative government might review or even terminate the programme.\(^{159}\) A costly primary prevention programme seemed under particular threat in an NHS required to make cost savings. May 2010 saw the formation of a Conservative / Liberal Democrat coalition government. Over the early months of the new administration uncertainty remained. It was not until late 2010 that the government indicated its support for the programme, most notably through its inclusion in the 2011/12 NHS operating framework.\(^{160}\)

Despite early uncertainties, the NHS Health Check programme remained almost entirely unaltered after the change in UK government; the same cannot be said for the government’s outlook on population level prevention strategies in the UK. Discussion of the nature and effectiveness of population level measures to prevent CVD are currently very pertinent and timely in the UK. Across a range of areas within public health, the coalition government is keen to adopt a dual approach of intervention. They aim to make improvements in the population’s health by Nudging individuals and employing responsibility deals with industry, preferring the latter to formal legislation and regulation.

Nudging is a behavioural economics approach to altering social norms, first outlined in 2008 by Thaler and Sunstein.\(^{161}\) Simplistically it promotes the likelihood of positive behaviours, without using legislation or forbidding actions. It is a libertarian paternalist theory, encouraging choice to be governed by best interest, without placing restrictions. It is politically very appealing, especially for a Conservative led coalition. It inherently reduces the size of the State (i.e. through not legislating) and allows individuality, two themes at the core of Conservatism.
Discussion of nudging and more interventionary public health additionally enters the debate over what critics call the 'nanny state', an overly intrusive government. Recently, from New Labour and even more significantly under the Conservative /Liberal Democrat coalition, UK governments have shied away from intrusive policies. In their place are libertarian policies, designed to enable individual changes. Libertarian critics of a 'nanny state', frequently cite the erosion of individual freedom and 'rights' as the main concern. These reflect the wider principle at the core of right-wing politics, maximising individual freedom. Conversely, one night argue evidence showing the success of these interventions outweighs the relatively small reduction in freedom.

The responsibility deals set out in the 2010 public health white paper can be considered as corporate level nudging. Instead of placing legislation, on for example the food industry to improve the nation's diet, the government works with the industry to draw up bilateral agreements. These agreements aim to curb industries' behaviour for the benefit of the general population. The UK has made rapid steps towards the implementation of nudging, including the foundation of the behavioural insights team and the publication of documentation outlining the potential for future interventions.

Despite enthusiasm from the UK government, there are concerns in the field of public health over the widespread implementation and reliance on nudging in health. Although nudging is effective in certain settings, there is currently limited evidence when applied to health. Indeed it has been scarcely implemented outside of a small number of localised interventions. There is certainly no evidence of sustained population level improvements. Further criticisms of nudging include concerns of a limited scientific background; it has potential to do harm; and crucially that it may be more limited in terms of potential impact than traditional population level interventions. Responsibility deals and self regulation also have criticism, largely through past experience of working with the tobacco industry. Self regulation can deflect attention away from necessary government intervention and industry involvement can lead to malpractice to promote unhealthy products.
The debate surrounding nudging has major implications for the prevention of CVD, with nudging proposed as a strategy to address key risk factors. Nudging, for example, is currently being discussed as one of the main potential methods to control obesity.\textsuperscript{163} Nudging is a population level intervention, however applying McLaren et al.’s spectrum it lies well towards the agentic end; it is strongly reliant on individual choice.\textsuperscript{105} As a result its impact on reducing health inequalities may be limited, or even widen them if the deprived population respond less well to nudging. Those with the greatest need may be less receptive to being nudged- a further manifestation of the \textit{inverse care law.}

Any government embarking on widespread nudging may be unlikely to embrace structural population interventions; this is for two distinct reasons. Firstly, interventions have resource implications; investment will be made in one method of public health intervention not two. Secondly, any government which embraces nudging (both at an individual and corporate level) ideologically may not favour structural population interventions. Although there may be scope for both, a libertarian party may strongly favour the former. Elements of the two are in contrast; the former promotes individual choice, combined with a small State (little legislation). The latter requires legislation and is prescriptive. There is a growing evidence base behind the effectiveness of population measures (chapter 3.3), but many of the successful examples are structural. Marteau et al.\textsuperscript{166} note, using reductions in salt intake as an example, improvements made through legislation have gone well beyond the impacts of partnerships with industry. A widespread focus on nudging may stop investment in effective population interventions.

\section*{2.4 Clinical guidance}

The emergence of CVD prevention in clinical guidance preceded its appearance in UK policy. Nonetheless, it could be argued that guidance on the reduction of CVD risk \textit{per se} is also a recent advent. The prevention of CVD underwent a major shift around the turn of the
last millennium. Previously, to lower a patient’s risk of CVD, a clinician aimed to control a series of single risk factors. This centred around, and at times consisted entirely of, controlling blood pressure to below cut-off values.\textsuperscript{170} A body of work especially through the 1990s\textsuperscript{170} facilitated a change in clinical guidance which switched attention to controlling global cardiovascular risk.\textsuperscript{115 171 172}

Since then clinical guidance for the prevention of CVD, has evolved both in the UK and Europe.\textsuperscript{60 173} This guidance, specific to CVD, now has a place alongside the continually revised guidelines for the control of hypertension.\textsuperscript{174} Even current guidance for the control of hypertension, although concentrating on blood pressure levels, now incorporates broader aspects of CVD risk.\textsuperscript{174 175} Despite a widespread change in direction of prevention, clinical guidance remains complex and at times confusing. Multiple, overlapping guidelines often exist, which may be detrimental to patient care.\textsuperscript{176} Co-existing guidelines have little harmonisation, and at times can be inconsistent and conflicting.\textsuperscript{176}

In 2008 in the UK, NICE issued two guidance documents both firmly cementing global CVD risk assessment into clinical and public health practice. One outlined the value of using global cardiovascular risk in the reduction of CVD mortality in the deprived population;\textsuperscript{177} whilst the second called for the use of global risk, not lipid levels, when making clinical decisions concerning statin prescription.\textsuperscript{113}

Concomitant to the publication of Putting Prevention First in 2008, the UK NSC published in depth guidance on vascular risk assessment.\textsuperscript{144} This guidance, which extended earlier NSC work on screening for diabetes, was to act as a template for the Health Check programme and documents much of the best practice adopted. The most recent UK clinical guidance concerning CVD outlined a very different approach to prevention.\textsuperscript{64} In 2010, NICE published guidance which for the first time combined the evidence from population level interventions designed to lower CVD risk.\textsuperscript{64} This guidance was notable for its range of recommendations, and although containing some local level proposals, much of its content was aimed at national government.
Chapter 3; The evidence for cardiovascular prevention

3.1 Secondary Prevention

Secondary prevention of CVD is far more established in practice than primary prevention. Secondary prevention is commonplace in clinical practice, exemplified in the UK by its inclusion in the QOF. By placing secondary CVD prevention within a large pay-for-performance framework it was hoped that standardised, high quality clinical practice would be promoted across the country. Early evaluation of the QOF has indicated reductions in intermediate outcomes, such as blood pressure and lipid levels.\(^{178,179}\) Data on long-term CVD outcomes are less clear. Work has indicated a relationship between QOF achievement and practice level hospital admissions,\(^{180}\) whilst other data have failed to demonstrate such an association.\(^{181}\) Any impact of preventative measures, such as those within the QOF, on the rates of CVD events and mortality will lag behind the intervention. It is possible that it is still too early to fully evaluate the impact of the QOF on such CVD outcomes.

Evidence supporting the effectiveness of secondary prevention should not be confused with the evidence behind the QOF. The latter is a specific example of a quality improvement framework with a health system. Evidence behind secondary prevention is more established. The secondary prevention of CVD is widely regarded as a highly effective and cost-effective pursuit. Despite clear health benefits, secondary prevention can however be suboptimal when not fully supported within a health system.\(^{182}\)

Individual components and interventions used within secondary prevention have strong evidence of effectiveness from clinical trials. The provision of oral anti-platelet agents, including aspirin is effective in the prevention of further major coronary events;\(^{183}\) statins cut both the reoccurrence of CVD events and the need for further surgical procedures;\(^ {184}\) anti-hypertensive medication lowers blood pressure\(^ {185}\) and reduces secondary CVD events;\(^ {185,186}\) if CVD patients stop smoking they can see up to a 36 percent reduction in all-cause mortality;\(^ {187}\) and exercised based rehabilitation reduces vascular risk factors and mortality.\(^ {188}\)
Not only are the processes used within secondary prevention efficacious, secondary prevention is effective in practice. Secondary prevention trials demonstrate reductions in hospital readmissions, improved vascular risk factor profiles for patients, an improved likelihood of receiving effective treatment and an improvement in quality of life. A range of interventions; including exercise, education, counselling, and pharmaceutical can all be effective in practice. One element, an accurate register of all eligible patients, is essential in the implementation of secondary prevention.

There is a large body of evidence behind the effectiveness of secondary prevention; I shall only concentrate on two further UK trials. These were both before the QOF, therefore not concurrent to routine secondary prevention and were implemented in routine general practice, making them relevant to the Health Check. In the first trial in Scotland, nurses ran clinics for all CVD patients in general practice with the aim of monitoring symptoms and controlling risk factors for CVD. One year after starting the intervention, patients saw lower levels of all the risk factors measured (cholesterol, blood pressure, poor diet) except smoking. These translated into a reduction in hospital admissions, an improved quality of life and after four years lower CVD event rates and mortality. Reductions in mortality were not maintained after ten years of follow-up, but after such a time span there was a large amount of crossover, dilution and the power of the study was unlikely to detect significant changes. Although not cost saving, the intervention proved cost effective, with a cost of £1,097 per quality-adjusted life year (QALY) gained.

The second trial, carried out in Leicester, assigned trained nurses to all CHD and heart failure patients. The nurses managed the patients’ medication and appropriate referrals. Again, improvements in risk factor profiles were seen quickly after the start of the intervention, although the project was less cost effective at £13,158 per QALY. The intervention increased prescribing costs, the usage of practitioner time and hospital activity, all of which raised programme costs and lowered cost-effectiveness. The total follow-up time was only one year, too short to fully capture the savings made from CVD prevention.
These trials combined with the subsequent emerging evidence from the QOF, indicate secondary CVD prevention is effective when implemented in the contemporary UK health service. Evidence of its effectiveness however does not necessarily translate to primary prevention. As discussed in section 1.2.2, primary and secondary target different populations. Secondary prevention is aimed at a smaller, more easy to define group; who are on average at higher risk and may have a greater motivation to change behaviours.\(^{56}\)

### 3.2 High risk approaches to primary prevention

#### 3.2.1 Clinical evidence for the efficaciousness and effectiveness of primary prevention interventions

**Lipid reductions and statins**

Statins are effective in reducing lipid levels.\(^{198}\) Given that vascular risk, especially the risk of CHD, is continuously related to lipid levels and increased risk begins well below average lipid levels found in a population,\(^{199}\) there is strong theoretical support for their use in primary prevention. Despite early concerns about limited impact on vascular mortality,\(^{200}\) evidence has accumulated behind lipid reductions,\(^{201}\) and more specifically the actions of statins\(^{202}\) were able to reduce vascular deaths in a primary prevention population.

Statins reduce CHD mortality in patients with diagnosed CVD\(^{203}\) and are effective in other groups designated as at high CVD risk, including diabetic,\(^{204}\) hypertensive\(^{205}\) and hyperlipidaemia patients.\(^{86}\) Statins are effective primary prevention agents for cardiovascular disease in the high risk,\(^{206}\) although there is still heterogeneity in evidence concerning mortality.\(^{207}\) Recent evidence has, however, agreed that statins produce only a limited risk reduction in the low to moderate risk.\(^{65/68/208/209}\) It currently seems that statins are not an effective or cost-effective intervention for the whole population but can be highly effective in high risk groups eligible for primary prevention.
Anti-hypertensive agents

The relationship between blood pressure levels and CVD risk is continuous, with elevated vascular risk beginning at a systolic blood pressure as low as 115 mmHg. Blood pressure exerts an especially strong effect on stroke incidence, with CHD risk more multi-factorial; nevertheless both demonstrate strong relationships. As a result, blood pressure control reduces both CVD incidence and rates of mortality. There is no direct control of blood pressure within the NHS Health Check; instead the programme diagnoses hypertensive patients who are then transferred to primary care for management. There are a range of effective anti-hypertensive agents available, many of which are effective in reducing blood pressure levels and cardiovascular risk in populations with and without CVD.

Uptake and adherence to therapy in primary prevention

Aside from the clinical effectiveness of therapies and clinician prescribing patterns, the adherence to medication is a vital component of patients meeting risk factor targets. At a population level good adherence is required for the success of drug based high risk prevention and at the individual level it improves clinical outcomes. In patients with diagnosed CVD, the long term adherence to statins in the UK is around 74 percent in men and 81 in women. A study in the United States using patients discharged from hospital after an MI, found that only 66 percent of those prescribed aspirin, beta-blockers and statins adhered to all three after one month. 18 percent had discontinued the use of one therapy, but from one to twelve months there was no additional drop out. In stroke patients discharged from one US hospital, 39 percent discontinued statin therapy after one year; similarly 39 percent of diabetic patients in the UK discontinued statins after six months.

Adherence to drug therapy in patients with clinical disease can be limited, but is even poorer in non-disease groups. Evidence from the primary prevention of CVD has demonstrated this poor adherence. In Italy the one-year adherence rate to statins for the primary prevention of CVD was 46 percent, and in one study in Canada fell to 35 percent after three years. A longitudinal study from Canada assessed the adherence, defined as the
dispensing of a statin prescription every 120 days, over two years. For primary prevention after three months the adherence was 75 percent, falling to 25 percent after two years. In a small sample (n=77) of older patients in the United States eligible for primary prevention, 10 percent never commenced statin prescription and after six months there was 63 percent adherence. This was lower in those who perceived themselves to be at low risk.

Non-adherence is not only common, but is unequal across society. There is evidence of lower adherence in smokers, the younger population and those with the lowest income. This can have a significant impact on health inequalities. There are a number of causal factors behind non-adherence including forgetfulness, medication not being a patient’s priority in daily life, lack of information, side-effects especially ones potentially yet to be discovered, an addiction to therapy, the complexity of drug regimes and altering the drug formulation.

The only truly primary prevention drug therapy linked to the NHS Health Check, given newly-diagnosed patients leave the programme, is the prescription of statins to the high risk group (although technically these also leave the programme and are prescribed statins as part of routine primary care). This is not though, a truly general population. They are a high risk sub-group. It is not clear what levels of adherence are to be expected from this sub-group. Patients diagnosed with hypertension, like the high risk group, do not exhibit clinical symptoms yet have improved statin adherence. The high risk group, however, differ having no firm clinical diagnosis. It may be the diagnosis which motivates the patient to adhere. Difficulties in accurately conveying CVD risk information may further limit statin uptake in the NHS Health Check.

Adherence to drug therapy in primary prevention has been generally low, with additional evidence of limited adherence even in disease groups. There are a number of interventions that can be employed to improve adherence; these are however complex and require additional resources. The adherence to therapy is a major potential limitation to the
effectiveness of the Health Check programme, with the available evidence indicating that without additional resources adherence in primary prevention is limited.

*Interventions within the NHS Health Check*

The first two interventions discussed above will not be carried out directly through the NHS Health Check programme. Both hypertensive patients eligible for anti-hypertensive therapy and the high risk eligible for statins leave the programme and receive continued care within general practice. There is an additional suite of interventions and referrals that are carried out directly under the auspices of the Health Check programme.

*Brief lifestyle advice*

The brief intervention carried out during the Health Check is the one intervention open to individuals at all levels of CVD risk. Despite a number of primary prevention trials employing brief counselling during the vascular screen,\(^70\) there are little data to assess the impact of this intervention alone. Most of the use of brief interventions has been as components of complex interventions making it difficult to assess their individual contribution.

Brief interventions are effective for smoking cessation when carried out by clinicians, including nurses and pharmacists.\(^{146218154}\) There is, however, contradictory evidence when carried out by non-specialists in smoking cessation.\(^{151\ 226}\) Brief physical activity interventions may require follow-up to be effective, and have no evidence of an impact on CVD outcomes.\(^{227}\) Brief weight loss interventions have limited effect without the support of pharmacological intervention;\(^{228}\) and when used in secondary prevention, thorough lifestyle interventions are considerably more effective than brief interventions.\(^{229}\) There is currently not a sufficient evidence base to be confident that the brief intervention employed in the Health Check (i.e. one carried out by a non-clinician, potentially outside of a healthcare setting) can make sustained population level improvements.
Smoking cessation

Smoking is a major risk factor for CVD, with both an increased amount and duration through life heightening the effect.\textsuperscript{230, 231} Smoking cessation reduces vascular risk.\textsuperscript{187} Few studies have singled out the impact of smoking cessation upon CVD risk within primary prevention, instead the service is usually offered as one component of a complex intervention.\textsuperscript{232} However, with many trials generating reductions in smoking prevalence,\textsuperscript{232} and the strong link between smoking and CVD, smoking cessation is an effective component of primary prevention. Dedicated smoking cessation services are efficacious,\textsuperscript{226} and further the UK has an effective smoking cessation service.\textsuperscript{233, 234} Brief smoking cessation interventions carried out by clinicians are effective,\textsuperscript{151} but there is no evidence for the effectiveness of nurse (or other provider) lead brief interventions.\textsuperscript{151, 226}

Physical activity intervention

Increased levels of physical activity lower cardiovascular risk, with the greatest impact in the most sedentary.\textsuperscript{235-237} This is, however, a complex relationship. Physical activity leads to improvements in cardiovascular risk profile, including lipid levels, body weight and blood pressure, and has additional impacts beyond conventional risk factors.\textsuperscript{238} Physical activity and physical fitness may have unique rolls in CVD risk.\textsuperscript{239} A number of hypotheses have been made for the effect of physical activity, including the work of inflammatory markers but there is currently no consensus.\textsuperscript{240}

Exercise based interventions make significant reductions in vascular mortality in patients with CHD.\textsuperscript{188} Brief exercise interventions can produce prolonged increases to exercise levels, although follow-up may be required.\textsuperscript{227} Intensive exercise referral schemes can be effective in promoting activity,\textsuperscript{241, 242} with improved results the more intensive the intervention.\textsuperscript{243} Interventions to promote physical activity are cost effective,\textsuperscript{244} with a recent systematic review placing them on an equal footing with many available pharmacological interventions in terms of cost-utility.\textsuperscript{245} Despite this evidence, there is considerable uncertainty over the population level benefits of physical activity interventions, with questions
especially surrounding the referral from primary care.\textsuperscript{246} Many trials have failed to use intention-to-treat analysis, therefore have not accounted for non-attendance.\textsuperscript{242} Trials demonstrate high attrition, which is likely to be higher in routine practice.\textsuperscript{247} There are further uncertainties over their impact on cardiovascular outcomes, which have never been assessed in a primary prevention setting.

\textit{Weight loss intervention}

Body weight similarly exhibits a complex relationship with cardiovascular risk and risk factors. Weight loss reduces CVD risk factors, especially blood pressure and lipid levels,\textsuperscript{248} but is additionally beneficial as an independent element of risk.\textsuperscript{249} Increased CVD risk is not only related to body weight, but also independently to central adiposity.\textsuperscript{250} Commercial weight loss programmes, such as those available after referral from the Health Check, are effective in reducing body weight.\textsuperscript{251}{252} Studies have found that these, in turn, convey a reduction in CVD risk factors, including lipid and insulin levels.\textsuperscript{251}{253} This has, however, not been widely studied, and there are no links to CVD endpoints. There are further considerable social inequalities in the uptake and compliance of weight loss interventions.\textsuperscript{252} Weight loss is a highly efficacious goal to lower the risk of CVD, but interventions must be multi-faceted to bring about effective weight loss.\textsuperscript{254} There is currently insufficient evidence of the effectiveness of the available weight loss interventions relating to the reduction in CVD risk.\textsuperscript{255} Indeed, barring the UKs smoking cessation service, there are questions over all of the main interventions carried out within the Health Check programme.

\textbf{3.2.2 High risk primary prevention; evidence from the UK}

The UK has never implemented a national programme for the primary prevention of CVD, and therefore the majority of the evidence concerning its effectiveness stems for clinical and epidemiological trials. Two studies in the UK stand out for their size and subsequent influence, and currently encompass a large proportion of the UK’s evidence base.
Chapter 3; The evidence for cardiovascular prevention

The BFHS was a randomised control trial carried out across 13 towns in the UK. In each locale, two general practices were included in the study; one control, whilst one had half the population in the intervention group and the other half as a second, internal control. All men aged 40 to 59 registered in the practices were eligible for the study. Those in the intervention group were invited, along with their immediate family, to attend a health check at the practice. The health check, conducted by nurses, involved the assessment of cardiovascular risk followed by an agreement between the participant and nurse on a strategy of follow-up (tailored to the participants risk status). After one year of follow-up cholesterol levels and weight were reduced in the intervention group, as was CVD risk. All of these reductions were significantly greater in the population at highest baseline risk. With the family orientated structure of the interventions, improved risk profile were clustered within couples.

Based on levels of risk factor reduction achieved, the authors estimated a 12 percent reduction in global CHD risk. In those attending the initial health check there was an increased rate of clinical diagnosis, but there was no associated increase in prescribing levels or costs. The impact of the intervention on smoking was limited. Accounting for an increased drop-out rate amongst smokers, there was little change in smoking prevalence. Smoking cessation advice was administered by trial nurses, and the authors hypothesised that it might have been more effective if carried out by specialists in smoking cessation, with greater training in behaviour change and motivational techniques. The short term successes in risk factor reductions came at a cost; the intervention was costly in terms of nurse time, and although there was a reduction in GP visits in the intervention group there was an increase in outpatient care.

The OXCHECK study was the second major UK trial, similar in impact to the BFHS. The trial covered five general practices in Bedfordshire. The intervention was aimed at all 35 to 64 year old patients registered with the practices, and was again a nurse run health check, measuring cardiovascular risk factors and counselling participants on behaviour change. An internal control was employed in an attempt to minimise the effects of dilution between
groups. In clinical trials, participants allocated to the control group can adopt the behaviours they see exhibited by the intervention group and thus dilute the results of the trial. All participants were randomised to be screened in one of the four years of the study. The control for a given year group was the population to receive the intervention in the subsequent years, for example those with a health check in year one had the population to be screened in years two to four as a control. The control group were scheduled to receive the same intervention, merely at a later date, so may be less inclined to actively adopt the behaviours of the trial group.

A pilot study for the trial investigated the impact of providing practices with a primary prevention facilitator. Their role was to set up a screening programme and manage a register containing the population eligible for primary prevention. The provision of the facilitator led to an increase in recording of smoking status, blood pressure and BMI but after three years there were only minor, non-significant changes to the risk profiles of patients.

In the main trial there was a high baseline prevalence of cardiovascular risk factors in those eligible for primary prevention. One year after the initial health check, the intervention group saw reductions in cholesterol levels, blood pressure (although these were within the bounds of acclimatisation to testing) and improvements to aspects of diet, including reduced butter intake. After four years, there were significant reductions in BMI, blood pressure and cholesterol levels, and an increase in exercise. Based on these risk factor reductions, the population had an estimated 7 percent relative reduction in risk of myocardial infarction, 13 percent in women. The intervention group displayed the same usage of general practice, but used more practice nurse time outside of the health check. Like the BFHS the intervention was costly, with again a large burden from the time spent carrying out the intervention and additional increases in prescribing costs.

Results from the OXCHECK and BFHS were similar. Both saw reductions in cardiovascular risk factors in the short term, but were costly to implement and highly demanding on nurse time. Neither proved to be cost saving, and in an intermediate time frame increased the
workload to the wider health system. Although OXCHECK was less costly, both were relatively expensive to implement but may be cost-effective.\textsuperscript{263} In order to fully assess the impacts of cardiovascular prevention, longer term follow-up is required which in turn requires larger study populations than used in either of these studies.\textsuperscript{263} There is a strong theoretical argument that cardiovascular prevention can not only be cost effective, but also cost saving. Simple (potentially low cost) prevention services prevent the need for expensive future medical interventions after CVD events. These two trials, however, demonstrate the difficulty in establishing evidence of this saving in cost. The CVD endpoints studied, cardiovascular hospital admissions or deaths for example, are complex and influenced by a large range of factors. After the effect of dilution, it is very difficult to establish the impact of an intervention on such outcomes. It is in fact modelling studies, not interventional trials that provide the strongest evidence for the economic impact of CVD prevention.\textsuperscript{148} These two trials also raised questions, which remain unanswered, over how long the reductions in risk are maintained.\textsuperscript{232,263}

A handful of further trials have been carried out in the UK evaluating the impact of primary prevention. In the late 1980s in Stockport, Cheshire, the Stockport Cardiovascular Disease Risk Factor Screening programme was carried out. The trial involved screening for CVD risk in the population aged 35 to 60 in general practice. Similar to the previous trials, the intervention involved the assessment of risk factors, giving health advice to all participants and referring patients to general practitioners if above pre-defined hypertension and hyperlipidaemia thresholds.

After a mean of 4.8 years of follow-up, patients receiving the intervention saw a decline in cholesterol levels\textsuperscript{264} which was greater in more deprived participants.\textsuperscript{265} White participants saw reductions in smoking\textsuperscript{264} but there was an increase in BMI in all groups.\textsuperscript{97} The strongest predictor of the success of the interventions was the baseline risk of the participant. Reductions in blood pressure were largest in those at the highest risk; in fact there was an overall increase in blood pressure in the lowest risk.\textsuperscript{266} Similarly, reductions in alcohol
consumption, smoking cessation and lipid levels were all greatest in the highest risk,\textsuperscript{266} a finding found more broadly across multiple risk factor vascular prevention programmes.\textsuperscript{232}

The SELSS was the first major primary prevention trial in the UK. Starting in 1973, the trial took place in two London general practices. Patients aged 40 to 64 registered in the practices were randomly allocated to either a screening intervention or control group. The study lasted four years; with the initial intervention carried out by practice nurses. The nurses recorded CVD risk factors, gave smoking cessation and weight loss advice and made referral to GPs for diagnosis and intervention.\textsuperscript{267} There were only limited outcomes studied, many of which were distant from the intervention. After four years of follow-up there were no differences between groups in general practice activity, hospital admissions, CVD morbidity or mortality.

One more recent UK trial was the Healthy Hearts study in Wales.\textsuperscript{268} Set in three general practices; the entire 45 to 64 year old population without CVD were invited to attend risk factor screening. The nurse carrying out the risk assessment was able to make referrals to general practice (for medication and diagnoses), dieticians, an exercise scheme or smoking cessation. One year after the intervention, in those attending, there was a 6 percent relative fall in CVD risk (from a mean risk of 13.1% to 12.3%), as well as reductions in blood pressure levels, pulse rate and lipid ratios. Once again over the study period the mean BMI increased.

The largest risk reductions were found in participants with highest baseline risk. The presence of a single elevated baseline risk factor, which in turn generated referral to further intervention, was also strongly associated with the reduction of risk. The positive findings were tempered by levels of uptake achieved, only 29 percent of those initially contacted. Although only a small trial, the risk reductions achieved in attendees were encouraging, especially considering the speed at which these were seen. The uptake, however maintained to be a major concern over the effectiveness of universal primary prevention, and the cost implications or further cardiovascular endpoints were not assessed.
3.2.3 High risk primary prevention; international evidence

Internationally there have been a number of trials into the primary prevention of CVD, but overall the evidence is still limited. The lack of evidence stems largely from the difficulties, especially concerning costs, of establishing large primary prevention trials.269 A large proportion of the evidence has originated from Scandinavia. In the 1970s the Gothenburg Primary Prevention trial used two, three-year birth cohorts with mean ages of 51 and 61 respectively to investigate CVD prevention.270 Within the cohorts, intervention and control groups were allocated, the former of which were screened at the baseline. Smokers were referred to smoking cessation clinics, whilst those with raised blood pressure and lipid levels were given pharmacological interventions and dietary advice. After ten years of follow-up there were reductions in all three risk factors, smoking prevalence, blood pressure and lipid levels, but not significantly compared with control groups.

Similar results were found in the Malmo Prevention Project.271 The Malmo trial focused on more than solely CVD, encompassing wider aspects of health protection, but did have a cardiovascular component. This consisted of a baseline cardiovascular screen, followed by interventions for raised risk factors. After a 20 year follow-up, there were no differences between intervention and control in CVD mortality or morbidity. Through the 1970s, there were wide scale reductions in CVD risk factors at the population level.272 The impact of these changes was likely to be stronger, especially after a long follow-up, than any potential benefits of the interventions. This has generated questions over whether existing methods of prevention have the potential to overcome wider population trends in risk and risk factors.273

The Oslo Study invited all men in Oslo aged 40 to 49 for a vascular screen in 1972, those with controlled blood pressure, but raised lipid levels or CVD risk were included in a randomised control trial; the intervention aimed to lower lipid levels through diet. Follow-up lasted five years, at which time both risk factor levels and adherence to the diet regime were assessed.274 Although the intervention focused on dietetic advice, there was additional smoking cessation information producing large reductions in smoking.275 The intervention
group saw on average 2.5 to 3 kilogramme reduction in body weight,\textsuperscript{275} 13 percent lower mean total cholesterol values, with 90 percent of the population seeing lipid reductions.\textsuperscript{274} After 7.5 years, there were significantly fewer CHD events in the intervention group, which was greater in those with largest lipid reductions.\textsuperscript{274} Risk reductions were uniform across social class.\textsuperscript{276} The reduced event rates were maintained for up to eight years after the initial intervention, by which time there was also a lower mortality rate in the intervention group.\textsuperscript{277} The Oslo Study produced remarkable results. It achieved both marked risk reductions (especially lipid levels) and reductions in CVD events, without employing pharmacological interventions. This study stands out for its population’s adherence to diet advice, and it must be remember that although normotensive, it included a high risk population.

A randomised control trial in Denmark (The Ebeltoft Health Promotion Project) began in 1991. The Ebeltoft trial directly compared two methods of CVD prevention, differing in terms of the level of their resource use. The first intervention group received only a preliminary health check, consisting of CVD risk factor assessment and tailored feedback from health care assistants. The second intervention group received the same health check, with an additional 45 minute discussion with a general practitioner about cardiovascular risk and then annual follow-up checks for five years.

The two intervention groups did not differ in terms of the outcomes measured at the end of the study period. The additional GP consultation and follow-up did not produce any benefits above and beyond those of the health check alone. This is likely to be for practical reasons. The follow-up suffered from very high rates of drop out. Only 18 percent of participants for example attended three or more sessions.\textsuperscript{278} In terms of assessing the potential efficacy of follow-up in CVD risk reduction, the high attrition rate represents a weakness in the study. It does, however, splendidly show this major practical barrier to CVD prevention.

In the final analyses the two intervention groups were combined. There were significant reductions in BMI, lipid levels and CVD risk in the intervention group.\textsuperscript{278} Reductions were
greater for all risk factors in those who were smokers or obese at baseline with greater reductions in lipid levels in those at the highest global risk. Overall, there was a relative reduction of 8.6 percent in CVD risk. Six years after the health check there was better life expectancy in the intervention group (0.14 life years), with no significant difference in costs to the health system. In the early years after the intervention, there was an increase in rate of GP consultation however after 7 years this fell below the control group.

The other main body of evidence originates from the USA but again comes from a relatively small number of trials. Most notably, the multiple risk factor intervention trial (MRFIT), beginning in 1973, which included nearly 13,000 men, aged 57 to 85. Participants were given an intensive intervention, targeted at a triumvirate of risk factors; blood pressure, using medication; poor diet, aiming to control calorie and cholesterol intake and smoking. This was followed by at least four-monthly follow-up visits to assess progress towards pre-agreed risk factor goals. The MRFIT saw a marked reduction across all risk factors in the intervention group; this did not translate into a reduction in CVD or total mortality after 7 years of follow-up but after 10 years there was 8.3 percent lower CVD mortality in the intervention group. This trial demonstrated some of the largest risk factor reductions seen in primary prevention, however it was very resource intensive and notably the lowest risk men were excluded from participation. The Minnesota Heart Health Programme employed individual screening, had an uptake of 61 percent but reported no further outcomes.

3.2.4 Patient level consideration of high risk primary prevention

Harm

In any high risk prevention programme there must be an initial stage of screening to identify a target population. A patient’s decision to participate in screening or preventative medicine is governed largely by a balance between the benefits and associated harms from participation. No preventative intervention is without harm. It is therefore the magnitude of harm which modulates the decision to participate. Marshall describes the potential for harm within preventative medicine to fit into one of three levels:
The evidence for cardiovascular prevention

- at the initial screening process
- during further investigation of the results
- as part of the intervention if abnormality is detected

Generally, as one progresses through the levels the potential for harm increases. Running through all three levels, one can further classify harm into one of two categories, physical and psychological." Physical harms tend to be specific to screening or prevention procedures, but there has been little work on the potential for physical harm in CVD risk assessment. The immediate risk assessment and further investigations carry little potential for harm, beyond an adverse reaction to venepuncture during lipid tests. Of the interventions available, weight loss programmes possess potential for harm; they can increase all-cause mortality, reduce bone density and increase the incidence of eating disorders. Statins also possess a number of unintended side-effects. Overall, there is little scope for physical harm from the NHS Health Check, however, these must be considered when addressing uptake.

At the heart of the NHS Health Check programme will be a process of medicalisation. The programme takes disease-free individuals, and gives them either a label from being placed on the high risk register, or places them on medication. Medicalisation is not without harm, which is of greater concern if the medicalisation is unwarranted. Harms include, iatrogenic illness, creating poor treatment decisions in patients, loss of self-efficacy concerning health, economic waste and creating an unhealthy focus on illness in patients. The medicalisation of prevention is likely to be especially great if situated in general practice, therefore may impact of the Health Check programme. Over-medicalisation, allied with an increasingly medical view of prevention is likely to cause unnecessary harm and inefficiency.

Concerning the NHS Health Check, psychological harm is a greater threat the physical. There are a number of general psychological harms of prevention, many of which can be applied to the NHS Health Check. Participants can perceive discomfort or adverse effects from the screening procedure, which is exacerbated when venepuncture is involved. Participation in screening can increase general health concerns. The impact of increased
concern on the patient is unclear; it could either be negative due to increased anxiety or conversely a positive by promoting positive health actions. One study looked specifically into the impact of CVD risk assessment on patients’ general health status. Although knowledge of CVD risk increased, this did not impact on perceived health status. One final psychological harm is, whilst waiting for test results, patients can be exposed to increased anxiety.

Within the NHS Health Check, the process of risk stratification and communication of the results is the aspect most associated with patient harm. In CVD risk assessment, a positive screening outcome is generally considered as being classified as at high risk using a CVD risk score. CVD risk assessment is more prone to unnecessary anxiety from the risk assessment due to the low sensitivity of the screening tool (the CVD risk score). Participants may have increased anxiety levels after a positive result. If truly at high risk this can be considered to be a necessary consequence. If, however, the result is a false-positive, any increased anxiety is entirely unfounded. This can be considered to be a perverse, harmful outcome from the risk assessment process. The definition of a false-positive is, however, difficult in CVD risk assessment and as a result it has had little attention. CVD risk scores, unlike other screening tests, do not predict a binary outcome (i.e. disease present vs. absent). They instead offer an estimate of risk. Overestimation of risk is possible, but it is difficult to define as a false-positive.

A small number of studies have addressed psychological harms surrounding risk assessment. A meta-analysis across a range of areas of health, found that after receiving positive test results, patients increased levels of anxiety and depression in the short term. No long term or permanent effects where, however, maintained. A further review looked more specifically at the effect of CVD risk scores on psychological harm. There is no evidence (albeit from only four studies) of high risk groups suffering increased distress, although at times the moderate risk did. The authors conclude that scheduled follow-up for the high risk mitigates any harm from high risk labelling. Those at intermediate levels of risk, not
receiving the follow-up, therefore face raised anxiety. Qualitative work suggests patients leave CVD risk assessment with a broad suite of positive feelings. These include a willingness to change behaviour, being reflective about CVD and understanding the condition better. Positive outcomes were, however, reliant on a strong doctor-patient relationship, good communication of risk and the professionalism of the clinician.\textsuperscript{291}

The opposing situation to an increase in anxiety due to raised risk is reassurance gained from a negative screening result. In the case of CVD risk assessment, a participant designated as a low or moderate risk might receive false reassurance.\textsuperscript{285, 288} Low risk participants of the BFHS had a lower perceived risk of MI than a matched control group.\textsuperscript{287} Qualitative research has uncovered evidence of false reassurance. A focus group of participants confirmed to be at low risk in a CVD risk assessment found the common theme, participants no-longer felt it necessary to make lifestyle changes after the assessment. Even smokers, an overt CVD risk factor, felt no reason to change behaviour. The comprehensiveness of an assessment made participants feel confident of the results, therefore exacerbated the reassurance.\textsuperscript{292}

A further harm associated with prevention lies outside of the physical and psychological aspects. There is opportunity cost associated with attendance at the screening or prevention service.\textsuperscript{285} Time attending the service is lost to other activities which may in turn lead to a financial loss in earnings.\textsuperscript{293, 293} Participants can feel ‘annoyed’ by the inconvenience of the assessment and loss of the time.\textsuperscript{292}

There are a number of harms and potential harms from CVD risk assessment. As yet, however, there has been little work to fully quantify them. Greenland and Lloyd-Jones\textsuperscript{288} go so far as to say that there is currently insufficient research into the harms surrounding CVD prevention to justify its widespread implementation. Overall there is no suggestion of great harm stemming from a Health Check. Importantly, centrally organised programmes reduce the overall burden of harm, with standardised procedures and quality control.\textsuperscript{294} High quality work within the Health Check (for example strong clinical leadership and sufficient follow-up)
may mitigate for increased anxiety due to risk. The greatest paucity in evidence surrounds false-reassurance. All prevention poses harm, therefore it is vital to monitor the programme to minimise it, and more evidence concerning the psychological impact of CVD risk assessment is required.

**Vascular risk communication**

In order for a prevention strategy using risk stratification to be successful, a central component has to be the effective communication of patients' risk. In general, the population has a poor understanding of CVD risk. They systematically underestimate risk, and for example see cancer as a disproportionately greater threat. Patients have low agreement between perceived and actual CVD risk. For example, only 40 percent of a study population accurately predicted their CVD risk, with people twice as likely to underestimate their risk as to overestimate. Those with the highest levels of risk and those with overt CVD risk factors, such as smoking, show the most accurate prediction of risk. These high risk sub-groups present smaller systematic underestimation than the general population.

A patient’s accurate understanding of their CVD risk is important, and is associated with positive clinical outcomes. If a patient accurately estimates their own risk, there are improved prescribing patterns in clinicians managing their care and evidence of greater reductions in lipid levels. Given that even clinicians significantly and possibly systematically underestimate patients’ CVD risk, accurately conveying risk is going to be a challenging but crucial component of the NHS Health Check.

Health literacy is a significant barrier to the successful dissemination of health information, in this case of CVD risk, to a population. Health literacy can be defined as

*a constellation of skills, including the ability to perform basic reading and numerical tasks required to function in the health care environment*

Poor health literacy has a number of general implications; it can lead to the limited use of health services, poor health behaviours and poor outcomes. Health literacy can be broken
down into three stages; the functional reading of information, the interactive ability required to fully benefit from a clinical encounter and the critical ability to process and utilise information.\textsuperscript{301} A systematic review estimated that between 34 and 59 percent of the population had limited health literacy. This estimate was heavily dependent on the definition of ‘limited’ health literacy, which varied between studies. One cannot ascertain a true estimate, but it is clear that there is a high prevalence of suboptimal health literacy.\textsuperscript{302}

A patient’s level of health literacy (as well as general numeracy) dramatically affects their ability to assimilate health information. This relationship is also dependent on the method used to communicate information or data.\textsuperscript{303} Differences in health literacy impact upon health inequalities. Health literacy is co-linear with socioeconomic position (SEP). The deprived are more likely to have poor health literacy, but it also impact independently of SEP.\textsuperscript{300} Negative consequences of limited-health literacy will therefore exacerbate social health inequalities.

Considering the three components of health literacy outlined above,\textsuperscript{301} it is vital to present health information in a manner that supports each stage. The interactive ability of the patient cannot be modified. It therefore becomes vital to present CVD risk in the clearest, most perceptible format, to aid the reading and synthesis of information.

At the most basic level, when trying to make information accessible to patients, it is vital to keep information as simple as possible. Information presented must be restricted to only what is necessary.\textsuperscript{300} Using numeric measures of risk is preferential to a descriptive approach.\textsuperscript{304} Relative risks (or relative risk reductions (RRRs) when describing the impacts of interventions) are the preferred method by patients for expressing medical data.\textsuperscript{304 305} This is in preference to absolute risk and the numbers needed to treat (NNT). The RRR can have a impact on clinicians, for example they are more likely to prescribe drugs which have their impact presented as an RRR.\textsuperscript{306} One limitation of relative risk is it can lead to overestimates of risk. This, however, can be nullified by framing it against the baseline absolute risk.\textsuperscript{307} The numbers themselves used to present risk information are important. Decimals, for example, can be detrimental to a patient’s understanding of risk.\textsuperscript{307}
The graphical or pictorial representative of risk is highly effective, and improves understanding.\textsuperscript{304 307} When using graphs, it is vital to maintain a simple representation, otherwise understanding is hindered.\textsuperscript{307} The clinician has an important role in a patient’s understanding of risk. If they show a willingness to co-operate with the patient, for example including them in the decision making process, understanding will improve.\textsuperscript{308} Despite the information being designed to aid a patient’s decision, patients feel more comfortable when a clinician expresses their opinion along with the level of risk.\textsuperscript{304} Finally risk perception is improved if a patient is confident in and comfortable with a clinician.\textsuperscript{308}

A patient’s understanding of risk is central to the NHS Health Check programme. Risk communication, combined with a brief lifestyle intervention, are the sole interventions received by the majority of the eligible population. The evidence presented above demonstrates that risk communication is not a menial task. High standards must be maintained for it to be effective and not produce perverse outcomes. A number of recent advances in risk communication, including the concept of heart age and lifetime cardiovascular risk might aid communication, but as yet these have not been evaluated in practice.\textsuperscript{117 309} The majority of programme workload will be undertaken by health care assistants. Clinical trust is an important factor in risk communication,\textsuperscript{308} and therefore the monitoring of risk communication using these practitioners is vital.

### 3.2.5 High risk primary prevention; a summary

High risk primary prevention of CVD can lower risk factors.\textsuperscript{70 274} Evidence is more limited for endpoints such as morbidity and mortality, with data frequently failing to find an impact.\textsuperscript{267} Some argue that this is solely an artefact of study design and not due to the ineffectiveness of the interventions.\textsuperscript{310} Between an intervention such as an exercise referral and an endpoint such as a CHD death, there is a long chain of events. Over the course of this lag, individuals are exposed to many other related factors. These include CVD risk factors, other
interventions, the dissemination of behaviours between study groups and the wider societal changes in risk.\textsuperscript{311} These all have the effect of diluting the impact of an intervention in the study group. Given the rarity of CVD events in many trial settings, added to the impacts of dilution, sufficient statistical power is rarely if ever achieved.

As a result some argue that these hard-outcomes are never appropriate to assess the effectiveness of CVD prevention. Instead, given the strong relationship between intermediate outcomes and mortality, these should be used to measure effectiveness.\textsuperscript{310} Intermediate outcomes, such as blood pressure levels, are closer on the casual pathway an intervention, therefore face less dilution. They are also measured more numerously, therefore have greater statistical power.

The NHS Health Check is aimed universally at the English population. Although there is evidence of overall risk factor reductions in populations eligible for primary prevention, there is consistent evidence that benefits are strongly biased towards high risk groups.\textsuperscript{70 232 266 312} A recent Cochrane systematic review of multiple risk factor primary prevention interventions concluded there was no evidence of their benefit in the general population, although they were effective in the high risk.\textsuperscript{232} A final, general concern with high risk interventions is that they can prove unable to overcome underlying, population-wide changes in risk factors.\textsuperscript{270 271}

A number of aspects of the Health Check programme, especially within the interventions offered, have questions over effectiveness. Uptake and adherence may severely undermine the success of statin therapy and physical activity interventions. Brief interventions lack evidence of sustained gains, especially when implemented by non-clinicians. Commercial weight-loss programmes have scarcely been associated with improved risk factor profiles. A recent systematic review outlined the limited evidence supporting the referral to physical activity interventions from primary care, in fact calling for reduced investment in such interventions.\textsuperscript{246} Communication of CVD risk is a difficult process, possibly more difficult than the programme accounts for. Finally any potential harm from CVD prevention is largely unclear. Given the size of the commitment the Department of Health has made with the NHS
Health Check programme, there is perhaps insufficient evidence of effectiveness, and too many potential limitations to justify their confidence in such a universal approach. With the programme currently underway, this makes careful monitoring of outcomes vitally important.

3.3 Population prevention

3.3.1 Introduction

Modelling studies have endeavoured to quantify the benefits of population strategies for CVD prevention and compare these with other methods of prevention. Three studies of note stand out for findings of a limited impact of population strategies and demonstrating high risk approaches to be more effective or cost-effective.\textsuperscript{84 87 89} An article by Manual et al.\textsuperscript{84} is frequently cited as evidence promoting high risk over population strategies. Findings suggested that a population strategy for CVD prevention would save 5,160 deaths per ten years in Canada, compared with 35,800 with a targeted approach using CVD risk scores.

Methods employed by Manual et al.,\textsuperscript{84} however, have a number of limitations. They firstly employed only modest risk reductions in the population approach (simply a 2 percent lipid reduction). This is both modest in effect size and limited in scope, as population interventions can impact on more than just cholesterol alone. They also cite evidence from community education interventions as the evidence behind the population effect-size. Community education interventions are very different from other population strategies and are potentially more limited (chapter 3.3.6). Their high risk approach, using statins, assumes complete adherence, and they do not outline the size of the risk reduction produced. Finally, the costs of the two approached are not considered. These limitations systematically underestimate the impact of the population prevention, whilst simultaneously inflating the impact of the high risk. They do, however, suggest the best strategy might be to combine the approaches. They also show the merits of a targeted high risk approach, rather than the universal approach of the Health Check programme.
Murray et al.\textsuperscript{87} concentrate modelling on salt and blood pressure reductions, finding targeted approaches more effective but considerably less cost effect. Finally, Zulman et al.\textsuperscript{89} modelled two population strategies and a high risk approach. The two population approaches differed in intensity and resource use. More intense population intervention had greater gain than the high risk approach, which was superior to the less intense population. In terms of QALYs the high risk approach was superior to both. The limited impact in QALYs of the population interventions were largely governed by assumptions of large levels of patient harm, reducing quality of life. Evidence below will demonstrate that population prevention need not result in patient harm. A number other assumptions have also been criticised for overestimating the potential impact of the high risk approach.\textsuperscript{94}

\textit{Table 3-1; the estimated UK impacts of the CVD prevention strategies}

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Summary</th>
<th>CVD impact (reduction per annum)</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>NHS Health Check</td>
<td>Programme outlined in Chapter 2.2, with 75% uptake</td>
<td>650 CVD deaths, 1600 MI &amp; stroke combined</td>
<td>Ref. 139</td>
</tr>
<tr>
<td>Trans-fat ban</td>
<td>Reduce consumption of industrial trans-fats to 1% of total energy intake</td>
<td>7,000 CVD death, 11,000 MIs</td>
<td>Ref. 313</td>
</tr>
<tr>
<td>Salt reduction</td>
<td>3g. reduction in mean daily salt (to reach the FSA 6g per day target)</td>
<td>14-20,000 CVD deaths</td>
<td>Refs. 314 &amp; 64</td>
</tr>
<tr>
<td>Smoke free legislation</td>
<td>Smoking ban in all enclosed public spaces</td>
<td>620 CVD deaths\textsuperscript{1}</td>
<td>Ref. 315</td>
</tr>
<tr>
<td></td>
<td>Stop second hand smoke in all enclosed spaces</td>
<td>10,700 CVD deaths</td>
<td>Ref. 315</td>
</tr>
</tbody>
</table>

Modelling suggests population interventions have a large potential for gain in a population, potentially greater than high risk approaches.\textsuperscript{76 90 316} With more limited (possibly realistic) estimates of uptake, the effectiveness of a high risk approach is severely undermined. Using an attainable population reduction in risk factors, for example a 0.3 mmol/L reduction in total
Chapter 3; The evidence for cardiovascular prevention

cholesterol and 7mmHg in blood pressure (both 5 percent of mean levels) could lead to a 26 percent reduction in the risk of MI across the population. A 10 percent reduction in smoking, blood pressure and cholesterol would produce risk reductions equal to the best estimates of the high-risk strategies with complete adherence. Modelling suggests that population level interventions, such as reductions in salt intake and trans-fats (chapter 3.3.2/3), can both save a large number of deaths in the UK and are likely to be cost saving. Table 3-1 compared modelled impacts of population intervention with those of the Health Check programme; in comparison the Health Checks are predicted to do little at the population level. Aside from modelling, there has been work to study the impacts of applied population interventions. I shall continue by presenting evidence from these studies, focusing on salt intake, trans-fats, smoking, the Polypill and community education.

3.3.2 Salt intake

Elevated blood pressure levels are one of the major single causes of morbidity internationally and single biggest single cause of CVD mortality. Modelling studies suggest only small individual reductions in blood pressure are required to make significant impacts to CVD prevalence in a population.

Dietary salt intake has long been known as a key driver of blood pressure levels. Only small salt reductions are required to generate large reductions in blood pressure. A 4.5 grammes per day reduction, is estimated to decrease systolic blood pressure by five mmHg in hypertensive patients and two mmHg in normotensive. This translates into a nine and four percent reduction in CVD mortality respectively. A further review found greater impacts, estimating a 5 gramme reduction in salt intake could lower stroke risk by 23 percent and CVD by 17 percent. Beyond observation, Cook et al. provided interventional evidence that reductions in sodium intake lower both blood pressure and CVD risk.

Population-wide reductions in salt intake have great potential for reducing CVD risk. Modelling from the USA estimates a 3 gramme per day reduction would prevent between 44
and 92 thousand deaths per annum, saving between $10 and 24 billion.\textsuperscript{323} Even a smaller 1 gramme reduction would be more cost effective than prescribing medication nationally to hypertensive patients.\textsuperscript{323} A similar magnitude of impact has been demonstrated elsewhere. Although framed as the potential reduction from partnerships with industry in the USA, modelling by Smith-Spangler et al.\textsuperscript{324} estimated that a 9.5 percent reduction in dietary salt could save $32 billion in direct healthcare costs in the USA.

Since the late 1970s, Finland has experienced a near 80 percent reduction in CHD mortality. Over this period total salt intake was reduced by one third, generating a 10mmHg reduction in population mean blood pressure level. This is likely to have been the major contributing factor behind reductions in mortality.\textsuperscript{325} Over this time period, the Finnish government concentrated on population level interventions to reduce salt intake. The government, for example, demands that all food with a salt (sodium chloride) level above a certain threshold is visibly labelled as ‘high salt content.’ Controlling population salt intake through industry is especially attractive because in high-income countries the majority of salt consumed is from processed foods. In the UK, for example, this is estimated to be 80 percent.\textsuperscript{326}

The UK has started to address salt intake, but has taken a less formal approach than Finland.\textsuperscript{326} The UK Food Standards Agency (FSA) set goals for dietary salt intake; at their last revision this was an individual consumption of 6 grammes per day by 2012. Progress towards UK salt reduction targets has been almost entirely through partnerships and agreement with industries, not through legislation. There have been some successes; in the four years to 2008, there was a fall from 9.5 to 8.6 grammes.\textsuperscript{327} There are now concerns, however, FSA targets will not be met and progress will stall, especially now under a government keen to utilise the self-regulation of industry.\textsuperscript{328}

Finland has made great progress in salt reduction.\textsuperscript{329} Successes in Finland were founded on legislation and direct government intervention, not partnerships with industry. The only direct comparison between methods was modelling carried out by Cobiac et al.\textsuperscript{316} They compared four strategies, two high risk approaches to prevention using dietary advice and two
population approaches, one based on the best results from industry partnerships and one using legislation to control salt intake. Both population interventions were cost saving, whereas neither high risk were, but the legislative approach had far greater scope with 20 times greater cost saving. The food industry may also oppose reducing salt levels, which will limit the profitability of foods, however, to date, industry has been cooperative in the UK.

3.3.3 Trans-fats

A second major focus of a population strategy to combat CVD has been on trans-isomer fatty acids (trans-fats). Trans-fats are a type of unsaturated fat which contain at least one trans isomer double bond. This bond gives them a valuable characteristic for the food industry compared with their unsaturated brethren. They have greater solidity at room temperature, and are long lasting under freezing. As a result, trans-fats are added to other unsaturated fats to lengthen the shelf-life and improve the texture of food stuffs.

Trans-fats are highly concentrated in certain food items, especially fast-food, margarine, snacks and deep-fried food. Data from Denmark in 2001 showed that although the national average consumption was 1 gramme per day, it was possible to consume up to 30 grammes in one meal. Natural trans-fats exist, but industrial trans-fats, manufactured by partially hydrogenating vegetable oils, are far more common and harmful to health. They have one of the strongest positive relationships with CHD risk of any dietary factor. Estimates suggest a 2 percent increase trans-fat intake (as a proportion of the diet), produces a 23 percent increase in CHD risk CHD.

In 2004 the Danish government introduced legislation to restrict trans-fats to 2 percent of the total dietary fat content. Legislation has been highly effective. By 2005, there were large reductions in the trans-fat content of foods that had high levels before the ban. There was up to a 30 fold reduction in certain foodstuffs, resulting in Denmark having some of the lowest levels of trans-fat internationally.
New York City followed suit with trans-fat legislation. By 2008, 2 years after legislation, the average trans-fat content fell from 50 to 2 percent of the total fat content in fast food meals.\textsuperscript{335} After the example set by Denmark and New York a number of other areas, including cities in the USA and Switzerland have implemented restrictions. Once implemented, previously held concerns over the removal of trans-fats have not come to fruition. The price of food has remained constant, despite fears of an increase and taste was unaffected.\textsuperscript{334 335} Gains from legislation might even be greater than expected. Trans-fats have not been replaced solely by saturated fats, as was the fear. Instead a mixture of saturated, monounsaturated, and polyunsaturated fatty acids has been used.\textsuperscript{336}

In England, reducing the trans-fat content of food to less than one percent of the total fat content has been estimated to prevent up to 11,000 heart attacks and 7,000 deaths annually.\textsuperscript{337} Despite evidence above and support from NICE,\textsuperscript{64} previously the FSA and now the department of health, has not adopted a legislative approach to trans-fat. Instead they believe self regulation by the food industry will quickly eliminate trans-fat from the UK.\textsuperscript{338} Some have argued that this approach might not have the same scope as legislation, may place vulnerable groups at risk and put unnecessary pressure upon food retailers.\textsuperscript{313}

### 3.3.4 Smoking

Smoking is a major risk factor for CVD,\textsuperscript{231} amongst many other poor health outcomes. Inhalation of second hand smoke also increases CVD risk.\textsuperscript{339} Second hand smoke is a major health concern in the UK and is estimated to cause up to 7,000 vascular deaths annually.\textsuperscript{315} Smoking therefore is a strong candidate for population interventions to reduce CVD.

There are a range of interventions available for tobacco control, including restrictions on advertising, the control of pricing, health warnings on tobacco products and imposing bans on smoking in certain settings. A wide range of interventions have the potential to reduce the prevalence of smoking,\textsuperscript{340} many of which appear in the World Health Organization's (WHO)
Framework Convention on Tobacco Control.\(^ {341}\) Health warnings increase the knowledge of risks of smoking; however the impact on smoking prevalence is less clear.\(^ {342}\)

Outlawing smoking from certain settings, often public place and places of work (frequently called *smoke-free* legislation), has recently become more widely implemented internationally. The main aim of smoke-free legislation is to reduce the population’s exposure to second hand smoke, although it also hopes to reduce smoking prevalence. Smoking bans consistently reduce levels of passive smoking, reduce CVD outcomes.\(^ {343}\) Two meta-analyses found a 17 percent reduction in the incidence of MI,\(^ {344}\) an estimated 10 percent reduction in CVD events, with the impact of the legislation growing over time.\(^ {345}\) There is less evidence behind a reduction in active smoking, although systematic review points to a downward trend, with particular impacts in Ireland.\(^ {343}\)

Smoke-free legislation in Scotland, introduced in 2006, prohibited smoking in all enclosed public spaces and work-places. Comparing a 10 month period in 2005 with 2006, there was a 17 percent reduction in hospital admission for acute coronary syndrome compared with a 4 percent reduction in England. Non-smokers saw the greatest reduction, 21 percent compared with 14 percent in current smokers. An estimated 67 percent of the reduction in admissions was the result of reductions in passive smoke inhalation.\(^ {346}\)

Where cardiovascular disease is the single largest condition driving inequalities in health, smoking is the single largest risk factor affecting inequalities.\(^ {347}\) Jha et al.\(^ {348}\) assessed the contribution of smoking to health inequalities across five countries. There was a larger social gradient in smoking related diseases, with smoking accounting for 40 percent of the deaths in the most deprived. In total, Jha et al.\(^ {348}\) estimated smoking to account for between 51 and 65 percent of the difference in mortality between the most and least deprived groups. Although other studies have smoking to be less important for social gradients, it still is a major contributor.\(^ {349}\) Arithmetically it can be hypothesised that a blanket reduction in smoking will decrease health inequalities; although critics state that population interventions would preferentially benefit the affluent.\(^ {104}\)
There is preliminary, albeit tentative evidence, that a range of population smoking interventions reduce health inequalities.\textsuperscript{350} One review found no social gradient after workplace and school smoking restrictions; education levels did not vary the impact of health warnings and increased pricing reduces smoking more in low income groups, all positive for inequalities.\textsuperscript{340} One concern over changes to pricing is it promotes illicit activities, which might be greater in deprived groups.\textsuperscript{340} It is not solely interventions to reduce smoking prevalence that have implications for inequalities. Before the 2007 smoking ban, public houses in deprived areas of the UK had higher levels of second-hand smoke. Smoke-free legislation in the UK has had a significantly larger impact in deprived populations.\textsuperscript{351}

### 3.3.5 Polypill

In 2003, Wald and Law introduced what they termed the \textit{Polypill}.\textsuperscript{352} They proposed the use of pharmacological agents in an entirely new, population based manner. There are a number of effective agents for reducing cardiovascular risk: these include statins, folic acid (for serum homocysteine), aspirin as anti-platelet therapy and a range of anti-hypertensive agents. Folic acid has subsequently been removed from the Polypill formulation due to limited evidence of its impact on CVD outcomes.\textsuperscript{353}

Wald and Law argue that instead of using these in a conventional approach, high doses to the high risk, small amounts given to the entire population will be effective in lowering population CVD risk. The Polypill, containing the agents above, will shift the population distribution of CVD risk factors in the fashion described by Rose (chapter 1.2.3).\textsuperscript{53} Given risk factor reductions over the entire distribution are beneficial,\textsuperscript{109} this reduces population risk. Wald and Law first estimated that a Polypill given to the entire population aged 55 and over, would give an 88 percent reduction in CHD and 80 percent reduction in stroke mortality. They further advocate that the Polypill will have few detrimental effects and will be low cost, although no initial economic analysis was applied.
The claims made by Wald and Law were remarkable. The then editor of the *BMJ*, Richard Smith, suggested that the edition containing their article was the "most important *BMJ* for 50 years". Over the following years there has been substantial academic interest surrounding the Polypill, but as yet little progress made. The lack of progress has been for two reasons; criticisms over the concept and practical difficulties in trialling new drug formulations. There are, however, now a number of trials underway.

A number of criticisms followed the outlining of the concept; firstly there is debate over the price and cost-effectiveness of the strategy. Modelling has indicated that any medication used must be low cost for the strategy to be cost effective, although there are indications it might be more cost effective than other high risk strategies and would lie within WHO limits. If the Polypill is administered to the entire population the costs of dispensing will be low. There is still, however, cost associated with monitoring side-effects in a large population. Greater evaluation of the cost-effectiveness is required.

The major threat to the effectiveness of the Polypill approach is adherence. Once adherence falls, the population level impact will quickly diminish. Initial estimates by Law and Wald assumed a high compliance, for example only two percent of the population suffer contraindication. This was criticised for being over-optimistic, although two subsequent trials have shown relatively good adherence. The PolyCap study saw 16 percent of participants not completing the course of treatment, with only 3.4 percent due adverse effects. In a sample of 222 patients in the UK there was 17 percent non-adherence. Adverse effects were considerably higher in a recent trial. 23 percent of the Polypill group dropped after twelve weeks, 18 percent because of side effects. There is as yet only limited evidence from trials, and further, lower uptake can be expected in a routine setting.

There were initial fears that combining therapeutic agents would limit their individual effectiveness. The PolyCap trial, initially, failed to find drug interactions. The final results of the trial, however, differed. When comparing the Polypill to its individual components there was reduced effectiveness, for example a 20 percent lower effect of Simvastatin.
publication of the initial concept there have been questions over the use of statins in the low risk population. Although effective for primary prevention in the high risk, statins appear of limited use in the low/moderate risk groups which could further limit the scope of a Polypill.\textsuperscript{58}

There are two final concerns over the widespread use of a Polypill. Firstly it may over-medicalise the population, creating a dependency on drug therapy. Secondly, it might reduce focus on the care of high risk patients.\textsuperscript{361} This final point is important. Implicit in the Polypill is that it is the sole method of prevention.\textsuperscript{352} This maintains its cost-effectiveness. One must forgo the resources used for risk assessment and stratification, to give cost reductions. As a result there is no defined high risk group. The high risk group receives the same (small) risk reduction as the wider population, and therefore will maintain their status as ‘high risk’. Ethical concerns might result from not managing the immediate risk in this group, which will be of particular concern to the clinicians with a duty of care to patients.

There is as yet little evidence behind the effectiveness of the Polypill. It requires the \textit{de novo} formulation of drug combinations; therefore needs greater approval in order to enter clinical trial. This has delayed the progress of the Polypill. There have been greater advances in its use for secondary prevention and in the high risk,\textsuperscript{362} than in universal primary prevention.\textsuperscript{361}

The largest primary prevention study has been the PolyCap trial in India,\textsuperscript{357} although recruitment criteria still demanded one raised CVD risk factor. The intervention significantly reduced levels of all target risk factors, but as mentioned above, drug combinations limited their overall effect. The PILL collaborative group published results of an international trial in 2011. This placebo randomised control trial, produced substantial risk reductions; a 9.9 mmHg reduction in systolic blood pressure, a 0.8 mmol/L reduction in low-density lipoprotein (LDL) cholesterol, resulting in a 60 percent reduction in CHD risk.\textsuperscript{359} This trial was again not in a truly \textit{general} population. Its inclusion criteria were a 7.5 percent ten year risk of CVD. They further had considerable levels of drop out and side-effects (described above), even having excluded patients with severe contraindications from entry into the study.
The Polypill still has questions, especially concerning cost effectiveness, uptake and adherence in routine practice. Current evidence does not come from the general population that the Polypill concept was created for. There is currently, therefore, no evidence it can work cost-effectively in the population manner described by Rose. Finally the Polypill has never been directly compared with other population level interventions, for which there is currently greater supporting evidence. A large amount of work, answering a large number of concerns is required before the use of the Polypill for primary prevention.

3.3.6 Community Education

In the USA in the 1970s and early 1980s, there was great research interest into what can broadly be termed as community CVD interventions. These trials employed complex, multi-faceted interventions across communities to reduce CVD incidence. They frequently used education and awareness campaigns, but also contained a range of further activities including individual level screening.

The Stanford study, beginning in 1972, was the first such major trial in the USA. It delivered educational interventions, primarily using mass-media to disseminate health promotion messages. These messages were designed to teach skills to enable behaviour change related to CVD risk. Modes of delivery included television, radio, newspapers, billboards and leaflets. One intervention group were exposed solely to the media intervention. A second had additional individual education delivering the same messages on a one on one basis.

Both interventions reduced a range of outcomes; including CVD risk (a relative risk reduction up to 28%), cholesterol and blood pressure levels, an increase in knowledge of CVD risk but no change in weight. The most significant finding from the Stanford study was that the individual intervention, although increasing knowledge of CVD and smoking cessation rates, did not lead to greater reductions in blood pressure, cholesterol or global risk. Personal intervention reduced risk factor levels faster, peaking after only one year. By two years, however, mass media produced equal risk reduction. The Stanford study, together with the
North Karelia project,\textsuperscript{364} was significant in demonstrating that community-wide interventions, especially through mass education, were able to reduce cardiovascular risk. It showed community education could potentially match the impact of personal education.

Following Stanford, a triumvirate of trials quickly followed; the previously described MRFIT, the Minnesota Heart Health Programme and Pawtucket Heart Health programme. The Minnesota trial began in 1981, aiming to build on findings from Stanford.\textsuperscript{365} Interventions focused on a discreet number of CVD risk factors, but used a number of strategies, ranging from mass education to risk factor screening.\textsuperscript{284} Minnesota produced one common theme; despite gains in knowledge and reductions in risk factors, these were insignificant in the face of secular trends.\textsuperscript{366} \textsuperscript{367} Further, there were increases in BMI in all trial participants.

The Pawtucket programme added to community education, employing environmental changes. Interventions varied, including modifying shop layouts food to aid healthy choices, screening programmes and media coverage.\textsuperscript{368} Again, risk factor reduction could not outstrip secular changes,\textsuperscript{369} \textsuperscript{370} although mean BMI held constant in control cities.\textsuperscript{369} Pawtucket had a staggered roll-out, individual screening not being used until later in the trial. Worryingly, without individual screening, trial involvement was low.\textsuperscript{368} \textsuperscript{371} Exposure is key to community education; methods might be efficacious, but effectiveness is lost without exposure.\textsuperscript{366}

Work into community interventions continued into the 1980s, with further evidence of an inability to reduce risk factors,\textsuperscript{372} with even more limited impacts in deprived urban settings.\textsuperscript{373} One later trial did resurrect the successes of Stanford, showing mass media education, individual counselling and courses, to reduce risk factors.\textsuperscript{374} This trial over a number of sites was, however, marked by great heterogeneity in impact.

A systematic review of community interventions outlines another weakness in the literature. Meta-analysis, covering 36 studies produced a significant mean (absolute) risk reduction of 0.65 percent ten year CVD risk. The majority of trials produced some, small reduction in risk,
merely not statistically significant.\textsuperscript{375} Individually trials were under-powered to detect change. Although a relatively small risk reduction, this could be telling at a population level.

The decade of scrutiny of community interventions, especially in the USA, produced important messages. Despite the initial successes of Stanford, individually many trials were unable to alter secular trends in risk factors. Indeed one systematic review suggests community interventions are unable to do so.\textsuperscript{376} This group of trials exemplifies why Rose quoted \textit{radical} methods as necessary for risk reduction.\textsuperscript{53} Community interventions, although population-wide, rely on individual participation; for example attendance at screening or the uptake of education. They are highly \textit{agentic},\textsuperscript{105} often doing little to modify the environment. The inertia of secular risk factor trends is extremely difficult to overcome. They saw some success, but provided no evidence of a reduction in mortality.\textsuperscript{375} Community education might be a useful component in our arsenal against CVD, however, does not have the power to be used as the sole method of prevention. Current challenges of vascular disease may be too great\textsuperscript{29} with more \textit{radical} methods required.

\textbf{3.3.7 Population prevention; a summary}

Modelling using realistic, albeit challenging assumptions of the impact of population-wide primary prevention, outlines the potential for this public health strategy.\textsuperscript{78, 90} Across a population it may have greater potential than high risk interventions. If applying a continuum from the structural to agentic intervention,\textsuperscript{105} there appears be a divide in both effectiveness and impacts on inequalities. Agentic interventions, such as community education programmes, can be limited and fail to overcome secular trends in risk.\textsuperscript{366, 369} There is evidence, however, that more radical interventions can exert significant change. Focusing on salt intake, trans-fat and second hand smoke,\textsuperscript{310, 319, 346} changes in legislation produce gains in cardiovascular health and positively impact on health inequalities.\textsuperscript{351}

Evidence for population prevention remains limited. Interventions require large-scale, national commitments. They are strongly influenced by political pressures and need full
The evidence for cardiovascular prevention

government support. They are contrary to the wishes of industry, making political lobbying a threat. They demand state intervention in the population; limiting individual freedoms is distasteful to certain political viewpoints. Finally they require a major commitment, which may not give immediate reward. This places them beyond the often limited foresight of national governments, who demand successes in order to maintain political support. In short, their implementation is severely limited by a range of factors outside of the health care domain.

The implementation of population measures has focused on a discreet number of interventions. There are further risk factors for CVD, therefore population interventions which remain unstudied. Examples include the restriction of saturated fat, and wider contextual changes to society, such as the promotion of physically active transport. For all the limits in size of the evidence base, there are a number of examples showing great success. These example support calls by Geoffrey Rose and others, that to achieve the effective prevention of CVD we must follow a truly bipartite strategy. That is a strategy incorporating both high risk and population measures.
Chapter 4; CVD risk scores

4.1 Concept of the CVD risk score

If aiming to reduce CVD incidence, the ability to predict in which patients events are to occur would be useful. When adopting a high risk approach to screening, categorising patients based on risk is a vital first element. Once identified, high risk patients can proceed to risk lowering interventions. At the population level, up to 75 percent of CVD risk can be predicted by established risk factors, 85 percent in patients with diabetes.\(^8\) With the large predictive power of a relatively small number of anthropometric variables, it is plausible that these can be used to produce prospective, predictive models for CVD events.

These predictive models, termed \textit{CVD risk scores}, are derived from longitudinal data; simplistically baseline patient data for CVD risk factors are recorded. After follow-up, CVD outcomes are recorded and prediction models are built using baseline data. The models are usually proportional hazard or regression models. They initially calculate relative risks for each baseline risk factors at a population level. The model coefficients are then translated into the risk prediction models for the individual.\(^377\) The final risk score takes the form of a percentage chance of a CVD endpoint during a set time span, usually ten years.

4.2 History of CVD risk scores

4.2.1 Framingham; the beginnings

CVD risk scores have their origins in the 1950s. The first to use methods comparable to modern risk scores was derived from the Framingham Heart Study in 1967.\(^378\) The first risk score to enter routine practice in a near significant manner came in 1976, again from the Framingham group.\(^79\) This risk score was instrumental in demonstrating that CVD risk factors, for example systolic blood pressure, have a continuous relationship with CVD risk. Lowering blood pressure beyond traditional targets was beneficial to the patient.\(^379\)
The Framingham group, using longitudinal data from the Framingham Heart Study, quickly became pioneers in the methods used to produce risk scores. The early Framingham scores were periodically replaced, with the most notable development in the early 1990s. In this period the group produced a series of algorithms first predicting CHD events, and then going on to predict more outcomes including stroke and combined CVD.

The cluster of Framingham risk scores produced in the early 1990s, especially for combined CVD events (the Anderson risk score) became far more widely used than anything seen previously. These risk scores became embedded in clinical practice and are still in routine use over two decades later. The Anderson risk score uses age, sex, systolic blood pressure, smoking status, total cholesterol to HDL ratio, diabetic status and left ventricular hypertrophy to predict CVD outcomes. Systolic blood pressure and total cholesterol to HDL ratio are entered as continuous variables.

A further combined CVD risk score from the Framingham group, the Wilson score, attempted to use categorical data for risk prediction. The performance of the model was similar to the Anderson score, but is computationally simpler to use. Despite this the Wilson score has never reached the levels of usage as the Anderson score. The Framingham risk scores are known to have a number of flaws. Despite these flaws they remain widely used often the most frequently used in practice. Their continued use may, however, be due to clinicians’ familiarity, rather than a measure of the accuracy of risk estimation.

Weaknesses of the Framingham risk scores

The major weakness of the Framingham risk scores is in their ability to estimate risk in different populations. They generally overestimate risk in the UK. In a sample from the British Regional Heart Study, there were 47 percent more predicted deaths than observed and a 57 percent overestimate of CVD events. This overestimation is not homogeneous and accuracy varies dramatically between settings. At times, Framingham risk scores in fact underestimate risk in modern settings. The ratio of predicted to observed risk varies widely, from 0.43 to 2.87. Underestimation of risk is most frequently seen in deprived areas.
Estimation varies geographically in the UK; there is larger overestimation in the South East of England than in the North.\textsuperscript{384}

The overestimation has arisen from the population-wide changes in CVD risk over the latter half of the 20\textsuperscript{th} century. The Framingham cohort had risk factor data recorded in 1970s USA.\textsuperscript{381} Like the UK (chapter 1.1.2), the USA has seen large reductions in CVD incidence and the prevalence of risk factors. Indeed reductions have outstripped those in the UK.\textsuperscript{17} The Framingham population was at higher absolute risk than any contemporary population. Their risk scores therefore systematically overestimate risk.\textsuperscript{384}

In order to compensate for inadequacies in risk estimation, attempts have been made to recalibrate \textit{Framingham} risk scores. Recalibration involves taking a pre-existing risk score, comparing mortality rates between the derivation population and a population of interest, and then adjusting the risk score accordingly. Recalibration can be at a national level;\textsuperscript{387} 388 locally\textsuperscript{389} or within specific groups.\textsuperscript{390} Recalibration is attractive; it allows risk scores to be used across a range of settings without the costly and complex process of generating a \textit{de novo} algorithm. It is though, not without limitations. This is exemplified by an attempt to recalibrate a 1998 Framingham score to the Swiss population.\textsuperscript{387} After synthesising national mortality data and data from local cohort studies to recalibrate the algorithm, a significant overestimation of risk was maintained.

Aside from systematic overestimation of risk, a second weakness of the \textit{Framingham} risk scores is the omission of risk factors known to be independent predictors of CVD. Such risk factors include obesity, family history of CVD, deprivation and ethnicity. Whereas the first weakness impacted the entire population equally, the omission of independent risk factors is likely to alter the relative ranking of patients by risk. \textit{Post hoc} modifications of the risk scores have been made to account for this. Most notably in the UK, the Joint British Societies modified the \textit{Anderson score} to account for family history\textsuperscript{115} and ethnicity,\textsuperscript{60} creating the \textit{JBS} and \textit{JBS2} risk scores.
4.2.2 Deprivation and the CVD risk score

Ever since the Whitehall Study, deprivation has been considered an independent risk factor for CVD. Deprivation has a marked impact on risk. The difference between the highest and lowest fifth of deprivation in the UK is equal to the difference between a diabetic and non-diabetic patient. Differential estimation between socioeconomic groups has a perverse consequence. The large overestimation of risk in the affluent and underestimation in the deprived leaves the risk score more sensitive to CVD events in the former (although less specific). In deprived communities in the UK the Anderson risk score underestimated risk, with the most deprived receiving only half of the care relative to their need.

The inclusion of deprivation to a CVD risk score can compensate for observed differences in risk between social groups. Its inclusion may not improve overall predictive accuracy in the population; it will however improve the equity in prediction. The ASSIGN risk score, developed from 30 to 74 year old participants of the Scottish Heart Health Study and Scottish MONICA Project, incorporated deprivation to using the Scottish Index of Multiple Deprivation. ASSIGN has an improvement in discrimination compared with Anderson but this was small considering the two independent risk predictors added (the other being family history). Crucially prediction was more equitable; using the Anderson risk score the observed to expected ratio of risk varied with deprivation, but not using ASSIGN. ASSIGN shows greater discrimination in Scottish data, but still overestimates risk. Again the derivation data are not contemporary, recorded between 1984 and 1995, since when the prevalence of CVD in the UK has further declined.

4.2.3 Novel CVD biomarkers and the CVD risk score

A large number of cardiovascular risk scores have been developed. They use different vascular endpoints, different methods and are derived in different populations. Many recent developments, especially in the USA, have focused on incorporating newly discovered risk factors for CVD including CRP. CRP is a significant independent predictor
of CVD, although thorough assessment of the evidence using the Bradford–Hill criteria, place it as non-causative marker of inflammation. Although a significant independent predictor, when added to a risk score however, as when adding SEP to ASSIGN, CRP fails to add a demonstrable improvement in discrimination of risk. Some now question whether there is any use for CRP, or other novel CVD biomarkers in the prediction of CVD risks.

It is not only SEP and novel biomarkers that seemingly add little predictive power to risk scores. Long established risk factors, even lipid levels, can also appear to add little extra accuracy to models. Two opposing explanations have been proposed for the apparent inability of additional risk factors to improve risk prediction. Firstly, it might simply be an artefact caused by the large predictive power of age and sex. Compared with these, any further risk factors give relatively little improvement in discrimination and therefore add little to the model.

The second potential reason is a flaw in the tools used to measure discrimination. Discrimination is most commonly measured using the area under the receiver operating characteristic curve (AUROC). The AUROC was developed to discriminate between binary outcomes and may therefore be inadequate when comparing continuous data such as CVD risk. Some also suggest that discrimination- the ability to separate events from non-events, is not the most appropriate comparator of risk scores. Instead calibration, a comparison between predictive probabilities and observed risk has been suggested as a better measure, with a number of alternates to the AUROC proposed.

4.2.4 The QRESEARCH risk scores

Two further risk scores warrant discussion. These, both developed from the QRESEARCH database, are significant both in the UK and internationally. They are the most contemporary UK risk scores and are internationally important for the derivation data used. Before QRISK, risk scores had been developed using data from cohort studies, for example the Framingham and Scottish Heart Health Extended cohorts. Cohort data have a number of
strengths, especially surrounding data quality. Both baseline risk factors and CVD endpoints are well recorded; there are few missing data. Quality, however, comes at a price; a financial cost and a time commitment. The foundation and management of cohorts are resource intensive. A subsequent problem can then be sample size; with the costs of recruitment the cohort data can be under-powered for a risk score.

The QRESEARCH database, from which QRISK and QRISK2 are derived, sources data differently. The database is created by longitudinal extraction of data from routine primary care records in the UK. It currently includes over 13 million patients. The characteristics of such a dataset are almost entirely the reverse of bespoke cohort data. The data are far more numerous because there is little cost associated with data extraction and no management of participants. The weakness is that datasets are filled with missing risk factor data and incomplete follow-up. The QRISK2 data for example had lipid data for only 33 percent of the dataset and only a mean of seven years follow-up extrapolated to ten.

The first offering from QRESEARCH, QRISK was developed in 2007 using data from 3.4 million EMRs. The population, aged 35 to 74, excluded patients with established diabetes or CVD. QRISK modelled a first CVD event, using Cox proportional hazard models. The final model, in addition to Framingham risk factors, included deprivation (the Townsend deprivation score), anti-hypertensive therapy, family history of CVD and BMI. In the validation dataset, QRISK produced a lower estimate of risk than both the ASSIGN and Anderson scores and better discrimination. Validation in external datasets continued to suggest QRISK was superior to Anderson in the UK. QRISK exhibits improvements in discrimination and calibration in nationally representative data; with generally a small underestimation of risk. QRISK does have weaknesses and criticisms, centred on the quality of the derivation dataset described above.

QRISK was superseded in 2008 by QRISK2. There were a number of additions to QRISK variables, most notably ethnicity. QRISK2 further incorporated a series of chronic conditions shown to increase CVD risk, including rheumatoid arthritis and atrial fibrillation (AF). Within
the derivation data, QRISK2 has improved discrimination and calibration compared with QRISK, but as yet is untested in an external dataset.

The choice of CVD risk score used in the NHS Health Check programme has proved to be an area of considerable debate. Following NICE guidance, initially JBS2 was recommended, despite criticism of Framingham scores. There were, however, calls to allow the use of QRISK or QRISK2, which showed signs of improved risk prediction. NICE were unable to approve a clinical tool without validation independent of its authors. After the publication of independent validation and pressure on NICE to review the decision, the choice of either JBS2 or QRISK/QRISK2 was permitted. After this, the Department of Health followed suite allowing the use of either within the Health Check programme. The Department of Health and NICE are unlikely to give their sole support to QRISK2 because it is not currently available universally free of charge. It, instead, is available under license from EMIS, and NICE therefore will not make QRISK2 their sole recommendation.

4.3 Weaknesses of CVD risk scores

CVD risk scores demonstrate general weaknesses; weaknesses not in individual algorithms but in the concept per se. Before commenting on these flaws, one should bear in mind that they are only models. Models are simplifications of reality, designed to aid understanding. By their nature they are not perfect. CVD risk models are required because a patient’s true CVD risk is highly complex and impossible to measure. The question therefore is not are CVD risk scores inaccurate, but do the inaccuracies prevent them from being a useful tool in clinical and public health practice?

CVD risk scores are not diagnostic tools in the manner of many screening tests. Instead they are prognostic tools. They predict the likelihood of an event, not its presence. This is not a weakness, but one must acknowledge the scope of risk scores when using them. The likelihood of an event is derived by translating population-level relative risks to an individual.
Risk scores are therefore exposed to the ecological fallacy. Not everyone in a population possesses the mean characteristics of that population; in this case, everyone with a given risk factor profile will not have the same risk. The impact of this is borne out by Figure 4-1, both risk scores have limited discrimination, for example using QRISK2 only 30 percent of CVD events occur in the high risk group.

CVD risk score possesses a number of technical and mathematical limitations. Statistical modelling requires careful balance between parsimony and predictive accuracy. CVD risk scores are no different. Risk scores containing more risk factors can predict risk more accurately. Each additional variable, however, brings measurement error. Individual errors sum to give a larger total error of the model. Most statistical procedures, the estimation of a population mean for example, explicitly accounts for the error in estimation. Error is, however, rarely considered in CVD risk scores; they rarely quote confidence intervals. The magnitude, even the very presence of error is therefore forgotten. Estimates of the level of uncertainty place confidence intervals for a 20 percent ten year risk at ±3 percent.
Increasing the number of model variables produces a further weakness. The more risk factors included in a prediction model the greater the likelihood of missing data in the derivation dataset, especially if using routine data. This creates inaccuracy. Large numbers of risk factors also affect the use of the risk score. The more variables, the greater amount of data need collecting. This is self-evident, but nonetheless important. It increases the workload of the practitioner carrying out the risk assessment and may limit its usage.

The importance of simple CVD risk scores is growing. The global burden of CVD is quickly spreading to low and middle income countries. Simple risk scores will be vital settings, where there are limited facilities to measure a full suite of risk factors. Laboratory measurements, for example lipids, will be a particular burden and restrict risk scores' use. Recent evidence has suggested risk scores not requiring laboratory data give strong risk prediction, and might be effective alternatives in low-income settings. The use of existing risk scores, simply with partial data is also possible and may provide an alternate strategy.

The accuracy of a prediction model is dependent on the number of endpoints observed in the derivation dataset. If there are too few CVD endpoints, power is severely undermined. In CVD risk scores this manifests itself as a different accuracy between sexes. Risk prediction is poorer in women than in men. Women experience a lower incidence of CVD, especially excluding the population aged 75 and over, therefore fewer CVD events. Finally, when entering patient data into a CVD risk score, the number of measurements impacts on the accuracy; the mean of multiple measurements decreases measurement error.

Risk scores should ideally be applied to populations with disease profiles similar to the population in which they were derived; otherwise accuracy is poor. This is true for Framingham (4.2.1), and indeed all risk scores. The PROCAM risk score, derived in Germany, for example overestimates risk in a UK population. Risk scores predicting the same CVD endpoint produce very different risk estimates in the same patients; even concordance of high risk status can be low. When there is agreement between mean levels of risk in a population, this may not translate to the individual patient.
comparing risk scores, the high risk population mimics the population in which each score was derived. A high risk population from a risk score derived in data from a lipid clinic (the GREAT risk score), for example, has higher lipid levels than other risk scores.\textsuperscript{418}

There are a number of weaknesses and limitations to CVD risk scores, many of which are unavoidable. A risk scores must be judged on the quality of derivation, for example appropriate methodology and sufficient CVD endpoints are vital. Risk scores must be used in populations similar to their derivation data. In using risk scores, two important considerations should, but frequently are not made. Firstly they are prognostic not diagnostic models, and have large errors in discrimination. Secondly, although rarely expressed, CVD risk scores inherently contain error. As long as these limitations are fully understood, they need not limit the use of CVD risk scores.

### 4.4 Clinical effectiveness of CVD risk scores

Compared with the attention given to producing CVD risk scores, the number derived and their discussion in academic literature, there is surprisingly little evidence of their effectiveness in practice.\textsuperscript{383,421} Even their presence in national clinical guidance appears to outstrip their evidence base.\textsuperscript{383} A recent systematic review examined the impact of providing patients with their CVD risk score.\textsuperscript{421} Risk scores failed to improve a patient’s perception of risk or willingness to commence drug therapy. There are, however, consistent weaknesses in the literature. Studies often have no control group, with the comparator rarely receiving no-treatment. Instead there is frequently intervention within the control group; judging the effect of the risk score is therefore difficult.

Another major difficulty is risk scores are used as components of complex interventions, for example combined with education or counselling.\textsuperscript{422} It is, therefore, difficult to attribute improvements in care to the risk score alone; indeed they are never likely to be employed alone in practice. They are unlikely to single-handedly improve patient outcomes, but may
help as part of an overall intervention, especially if used repeatedly. There is equally little evidence of whether clinician knowledge of risk scores translates into better outcomes and it is not known how to promote their use in clinical practice.

A strong rationale behind the routine use of risk scores is that clinicians can be poor judges of CVD risk using risk factors data alone. By assimilating the elements of risk, the clinician might better utilise the data. The presence of a risk score in medical records can increase the prescribing of anti-hypertensive and lipid lowering drugs in high risk patients; can improve the attendance at screening and promote risk factor reductions, again largely in the high risk group.

There are risks in the use of CVD risk scores. Patients can fail to understand the concept of a CVD risk score and GPs may not find it easy to explain. Even clinicians may not fully understand the concept of global risk or may not find numeric outputs useful in clinical decision making. Importantly, as with their effectiveness, there is very little evidence of patient harms. One study found the use of a CVD risk score does not increase a patient’s anxiety over risk status, but more research is needed into patient harm.

The evidence of clinical effectiveness is limited; this relates to an individual’s CVD outcomes. Risk scores are not, however, used solely as clinical tools. They also have an important role in public health. They are used for risk stratification and as inclusion criteria for preventative care. The evidence presented above does not apply to this latter role. There is far stronger evidence that the targeting of interventions using risk scores is effective at the population level when compared with both universal methods and the use of single risk factors. Data published since the NHS Health Check programme have indicated that through targeting with CVD risk scores, one can capture all prospective CVD events in a targeted population, without screening the entire population. This of course saves costs. Even significantly more simple targeting methods, using for example deprivation, can cost-effectively identify a large proportion of high risk patients.
Indeed, support for a targeted high risk strategy, compared with universal, is not new in the UK. For nearly a decade, there has been evidence that the use of CVD risk scores with pre-recorded data from EMRs is an efficient process, preventing more CVD events for a given expenditure. It is significantly less costly to identify patients eligible for therapy using pre-selection, indeed up to 85 percent of patients eligible for anti-hypertensive therapy lie within the 20 percent estimated to be at highest risk. One great attraction of the pre-selection of patients with CVD risk scores is that complete data are not required. Although complete data are ideal, partial data produces strong estimates.

### 4.5 The future of CVD risk scores

There are three main avenues of current and future work surrounding CVD risk scores; the recalibration of existing risk scores to new populations; incorporating novel risk factors and the prediction of lifetime risk estimates instead of medium term. The recalibration of risk scores to match the cardiovascular disease profile of different populations has frequently focused on *Framingham* scores. Other risk scores have been recalibrated, for example *SCORE* has had iterations generated for a number of European countries. Recalibration is less costly than the *de novo* creation of a risk score; however despite considerable statistical effort, can maintain inaccuracies in prediction.

The inclusion of new risk factors has taken different paths in the UK and USA. Recent American risk scores have included newly discovered biomarkers for CVD risk; with for example the Reynolds’s risk score incorporating CRP. As described previously, despite being an independent marker of CVD risk, the inclusion of CRP produced limited improvements in prediction. UK developments in risk scores have, attempted to include risk factors outside of the clinical domain. Notable examples of these include ASSIGN and QRISK both incorporating SEP, whilst QRISK2 modelled ethnicity.
CVD risk scores do not effectively predict risk in a younger population. In patients aged under 50, only 10 percent have a JBS2 risk score over 20 percent but over 20 percent have risk factors.\textsuperscript{439} Using the SCORE risk chart, there is no combination of risk factors by which a 40 year old patient will be classified as at high risk.\textsuperscript{403} Similarly, using a *Framingham* based score, in non smokers no men aged under 45 years or women under 65 could be classified as at high risk.\textsuperscript{440} This is not an *inaccuracy,* they are 10 year risk scores and these patients do have low medium term risk. The problem again lies with the scope of most risk scores. They aim to predict *medium-term* risk, therefore are of limited use in the young. Within a high risk prevention programme for CVD this can be detrimental. The early identification of risk, followed by early risk factor control is likely to be beneficial to patients.\textsuperscript{441}

The simplest method to improve risk prediction in the young is to use the same short term risk scores, but have age dependent thresholds to define ‘high risk.’ Such a strategy was implemented in Norway. Drug therapy is recommend if greater than one percent ten year risk when aged 40 to 49 years, but greater than five percent if aged 50 to 59.\textsuperscript{442} This approach has one major weakness; risk scores do not have the precision to discriminate between low levels of risk.\textsuperscript{441} In a longitudinal study, the 10 year *Framingham* risk scores showed little ability to differentiate between levels of lifetime risk in the younger populations.\textsuperscript{443} Also given their inherent error described above (chapter 4.3), there is no evidence that the use of a risk score is valid at this level of accuracy.

A second option is to use a comparative approach to risk prediction.\textsuperscript{444} Instead of absolute risk, one can calculate risk comparative to the average person of the same age.\textsuperscript{445} This method is again heavily limited by the poor discrimination of risk score at the lowest levels of risk. The third option is when generating the CVD risk score, to enter the patient’s current risk factor profile, but to simply increase their age. This gives an estimate of what their risk will be in the future if they did not alter their risk profile. This method is unpopular, and could be dangerous for patients. Patients can easily misunderstand, take the score literally, unnecessarily increasing their concern.\textsuperscript{403}
The final and most popular solution is to shift from the measurement of medium-term risk to life time risk. Lifetime risk prediction has been considered in academic literature for over a decade,\textsuperscript{446} largely motivated by calls to begin the management of CVD risk at younger age.\textsuperscript{441, 447} Even at the youngest age groups, CVD risk factors begin to detrimentally affect physiology. One study, for example found, in patients aged 33 to 50 that there was a significant prevalence of coronary artery calcification, a sub-clinical marker of CVD, even after excluding those with the highest 10 year CVD risk.\textsuperscript{448} Through the use of lifetime CVD risk, one hopes to identify at risk patients at a younger age, therefore preventing the accumulation of harmful changes.

The concept of life time risk estimation has one main criticism; lifetime risk estimates will be homogenously high risk in a population. The incidence of CVD is high in the whole population; therefore a large proportion will have high lifetime risk. More people will die from CVD than any other cause. Early studies into the use of lifetime risk suggest some variation in the levels of risk scores.\textsuperscript{447, 449} For example, at the age of 40 there was a two-fold difference in lifetime risk across the spectrum of lipid levels.\textsuperscript{449} This implies they have some ability to discriminate between levels of risk. There are, nonetheless, concerns that life time risk scores will vary sufficiently across a population to be a useful tool for targeting.\textsuperscript{118}

In addition to problems with risk stratification, once identified as ‘at high risk’ at an early age, there is a question over how best to manage risk. As when using the short term risk scores, once found to be at high risk there will be a temptation to start medication. This treatment regimen does not prevent the underlying causes of risk,\textsuperscript{427} and could begin a lifetime of over-medicalisation, which itself has associated harms.\textsuperscript{119, 442} As discussed above, lifetime risk prediction is a strong candidate to receive support from pharmaceutical companies selling preventative medication.

Current evidence behind the clinical effectiveness of lifetime risk scores is even more limited than medium-term risk. A clinician’s knowledge of lifetime risk can increase prescribing levels, going beyond the levels recommended in clinical guidance.\textsuperscript{298} This may be an
implication of the generally high risk scores produced. As clinical tools they may be useful in conjunction with medium-term risk scores, especially in the young. Strengths of lifetime risk estimates may be in risk communication and demonstrating the long term implications of modifying risk factors to motivate change.\textsuperscript{118}

Lifetime risk score are a recent addition to CVD risk prediction, and considerably more data are needed on their effectiveness. Questions persist, especially over their ability to stratify patients and there is no evidence behind the cost-effectiveness of targeting patients through lifetime risk. They will identify a large population at high risk. With large associated cost implications, one concern would be that if implemented prevention programmes using lifetime risk will be costly, medicalise patients and draw attention away from other strategies, for example the combined use of high risk and population prevention (chapter 3). Pressures to implement lifetime risk prediction from pharmaceutical companies, despite the lack of evidence base, must also be resisted.
Chapter 5: Evidence from the uptake of screening and health promotion

5.1 Introduction

The NHS Health Check programme is not the first national, systematic screening programme. In the adult population in England for example, there are currently national breast, cervical, colon cancer, diabetic retinopathy and abdominal aortic aneurism screening programmes. There is great experience and evidence in the UK and internationally, with a number of programmes underway for many years. The UK’s national breast screening programme was, internationally, the first programme of its kind, starting in 1988. Cervical screening has been carried out in the UK since 1964, but was only formalised with a national call-recall system in 1988. The UK national bowel screening programme began more recently, in 2006, but even this has generated a large body of research. I shall review evidence from the wider screening literature, and that more closely akin to the NHS Health Check, from CVD prevention and general practice usage.

Screening is an action applied to a population with the goal of selecting people at risk of adverse health outcomes. The selection process then enables further investigation, monitoring, advice or treatment.\(^6\)\(^4\)\(^5\)\(^0\) Screening is fundamentally different from the usual processes of clinical care. It does not stem from a specific patient request or need.\(^4\)\(^5\)\(^1\) For a health intervention to be viable, benefits to a patient must outweigh harm. Balancing these is critical in a screening process, where there is relatively little immediate gain. A screening programme will become a viable option in a population if:\(^4\)\(^5\)\(^1\)

- Gains for an individual outweigh the cost; costs are not merely financial, but include negative patient outcomes.
- The condition is a major health concern to the population, with impact governed both by severity and prevalence.
Within the health system, due to finite resources, it is cost-effective when compared with other work undertaken.

The natural history of the condition screened is well understood. The ‘disease’ is never a measurable entity. A proxy or downstream element must instead be measured, and this must reliably map the outcome.

There is evidence of effective interventions to follow the screening test.

A screening programme must be equitable and be able to reach an entire population.

The screening literature covers many different programmes and processes. When reviewing this, it is important in the first instance to understand that some patterns in uptake are unique to procedures. A good example is flexible sigmoidoscopy, a technique used in colorectal screening. This procedure is considerably more invasive than other screening methods, therefore is likely to invoke a different patient response (Chapter 5.2.1). There are, however, general patterns in the usage of preventative medicine and all aspects of health care.

As a result, lessons for the literature are transferable to the NHS Health Check.

5.2 Screening uptake; a review

5.2.1 Difference in uptake between screening programmes

Despite many similarities, there are inevitable differences in uptake of different screening procedures. Each has different characteristics, and therefore will invoke different responses from patients. Moser et al. directly compared the uptake of breast and cervical in the same population of British women. Predictors of uptake differed, ethnicity and education were important in cervical screening, whilst economic factors were influential in breast screening. Exact reasons for these differences are unclear. Practical barriers may be important, for example cervical screening occurs more locally therefore car ownership less of a barrier.

There are likely to be more complex factors, including the perception of screening, that impact differentially between societal groups. Perceptions of flexible sigmoidoscopy differ
across ethnic groups, especially concerning barriers to attendance. The method of screening, as opposed to the condition screened for, is also important. Attendees at two methods of prostate screening significantly differ in characteristics.

5.2.2 Socioeconomic position (SEP)

An introduction to SEP

SEP is commonly used to study the uptake and usage of health care. Deprivation, the negative extreme of SEP can be defined as

\[ \text{a state of observable and demonstrable disadvantage relative to the local community} \]
\[ \text{or wider society or nation to which an individual, family or group belongs} \]

Examining the relationship between SEP and a function of health there are two important factors to be considered-

- The proximity of SEP to the health factor; there must be a credible pathway by which an individual's SEP can act upon the health behaviour.
- The components of SEP associated with the health behaviour; SEP is not a single factor exerting a force upon behaviour. It is highly complex, with many different components.

Both are frequently ignored, leading to evidence being miss-interpreted and spurious causal pathways drawn. The latter point should be considered. From a practical approach, however, it is impossible to capture all of the aspects of SEP. This creates a paradox. SEP is not a single entity, therefore cannot be measured as one. Equally one cannot measure (or even know) all its contributing factors. A proxy or range of proxy measures must therefore be found. Further adding to complexity when using multiple proxy measures, is co-linearity between components. Although different factors exert their own influence on SEP, they are also highly inter-related. Discovering causal relationships is therefore difficult.

Many different constructs have been used to measure individual SEP. Three main areas include material elements (income, car ownership, housing, etc.); education and
employment. Further aspects include health status and social inclusion. The former is inappropriate when studying health because the independent variable controls for differences in the outcome. Summary measure of SEP, incorporating many individual constructs are now available and frequently applied to research.\textsuperscript{460}

\textit{Summary measure of SEP}

Socioeconomic position can be summarised by a single measure, incorporating a number of elements. Two examples, commonly used in the UK, are the Townsend deprivation score\textsuperscript{406} and the Indices of Multiple Deprivation (IMD).\textsuperscript{460} Across a range of screening procedures, poor uptake has been found in the deprived when using summary measures; in breast screening,\textsuperscript{461-463} diabetic retinopathy\textsuperscript{464-466} and colorectal screening.\textsuperscript{467-469} At times, however, summary measures are incorrectly specified as continuous measures where categories should be used.\textsuperscript{469}

Summary measures of SEP represent an average level in a geographical area, at a single point in time. They are frequently used as the sole measure of SEP. For practical reasons, this is often the only measure researchers have for SEP. Medical data rarely contain individual SEP measures. Their use does, however, bring weaknesses to analysis. They firstly do not fully capture the complexity of SEP. Secondly they are an area level measure. Not all individuals in an area share the same characteristics.\textsuperscript{106} Finally, they measure a single time point, not accounting for the levels of social position over the life course.

An individual’s entire life-course of social position is important to health. Low childhood socioeconomic position increases the likelihood of CVD in middle-age men,\textsuperscript{470,471} with effects extending into older age.\textsuperscript{472} In women, life course SEP predicts CVD mortality, possibly more strongly than current socioeconomic position.\textsuperscript{473} CVD risk accumulates over the life course; greater exposure to low socioeconomic position generates greater risk.\textsuperscript{474} Life-time exposure to deprivation also impacts on health behaviours, including diet and physical activity.\textsuperscript{475-477} Smoking is less clear and possibly more strongly driven by current SEP.\textsuperscript{475,476}
The use of area level measures as a proxy for SEP must not be confused with using area level measures *in addition* to individual factors. Two aspects of a population affect its health, its *context* and its *composition*. Composition covers traditional, individual components of SEP. Context, however, is unrelated to the individual, comprising features of the area in which the population resides. Examples of context include housing, sanitation and shops.

Controlling for composition, there is growing evidence that context leads to poor health. Studies have found increased CVD mortality,\(^{478}\) atherosclerosis\(^{479}\) and CHD\(^{474}\) prevalence in deprived areas. The same relationship is seen for CVD risk factors, including BMI, smoking, poor diet and physical inactivity.\(^{477-482}\) Adding further to the complexity, those with greatest individual deprivation may suffer a greater impact from their context.\(^{483}\) The question remains over whether living in a deprived context is causal or a consequence of poor health.

Little work has applied both compositional and contextual elements to the uptake of health services. One study found poor breast screening uptake in deprived areas,\(^{484}\) and a recent study from Singapore found this relationship across a range of preventative procedures.\(^{485}\) Pruitt et al.,\(^{486}\) in a review of cancer screening found no significant evidence of an additional area level effect, however from limited studies. There is currently no evidence of an area level effect of deprivation on uptake, but this has been seen for other health variables. It cannot be discounted, especially given the different intervention required to overcome compositional and contextual deprivation.

*Education*

Education is one construct of social deprivation frequently applied to the analyses of uptake and many health outcomes. Greater levels of education promote colorectal screening,\(^{468-487}\) both for flexible sigmoidoscopy\(^{488}\) and faecal occult blood test (FOBT).\(^{489}\) Education improves interest in screening,\(^{490}\) attendance once invited\(^{491-492}\) and belief in its worth.\(^{490}\)

Educated women are more likely to attend breast\(^{487-493}\) and cervical screening,\(^{494-495}\) although the later is not always clear.\(^{98}\) One study suggested education has a non-linear
relationship. Women not only with the lowest, but also the highest levels were less likely to attend. Two newer screening procedures, abdominal aortic aneurysm and prostate cancer both show greater uptake in the educated. Accounting for other constructs of SEP, such as economic factors, education is significantly related to uptake.

Education is often employed in analyses as a cross sectional measure of the highest education attained. Sabates and Feinstein claim this is a major weakness in research. Education has a far more complex relationship with patient characteristics. It increases self efficacy and patience, which can in turn encourage health promotion. Complexity is added, however, because both also predict consumption of education. This is why, at times, previously attained education does not predict health behaviours. Current education, such as continued adult learning might be key. Current education, when included in analysis with previous education, became the strongest measure of cervical screening uptake. It may improve social inclusion or awareness of screening, therefore promote uptake.

**Employment**

Employment is the second frequently used component of SEP. This has been analysed in two ways, as a binary variable of employed/ unemployed and as a graded level of occupation. Unemployed women have been found less likely to attend breast screening, although attendance also decreased in those with highest grades of employment. Increasing grade of employment was also a predictor of continued attendance at colorectal screening. Conversely, the unemployed were more likely to attend new patient health check in general practice, screening for undiagnosed diabetes in Denmark, and more likely to access a range of health promotion services. Further, employment also shows insignificant in analyse. Overall there is no clear evidence that employment directly influences screening behaviours.

**Material SEP**

A third individual-level construct of SEP used is material wealth. The most direct measures are income or wealth, whilst other proxy measures include car and home ownership. Women
in the UK with higher incomes have shown higher uptake of mammography, with increased colorectal screening in both sexes. Higher levels of car and home ownership predict interest in colorectal screening, attendance once invited and uptake of breast and cervical screening. Finally, in the UK, there is evidence of greater attendance at abdominal aortic aneurysm screening with higher income. In the USA there is evidence of higher uptake of breast, cervical and colorectal screening.

Material aspects of deprivation may act through direct economic barriers. Even in health systems where care is free at the point of delivery, the economically deprived may still suffer greater additional costs. They may, for example, face higher travel costs. Women from lower social classes are more likely to use public transport to attend screening which is more costly and time consuming. Low income groups may suffer a greater opportunity cost, for example, having to forgo pay or holiday to attend screening.

There are also barriers beyond the economic; social disparities are maintained in self administered tests carried out at a participant’s home. One study included the cost of attendance in analysis of breast and cervical screening uptake. The impact of income on uptake was reduced, however, was not totally attenuated. Income impacts on health behaviours by more than direct barriers, and for example might have psychological consequences.

SEP and uptake; the causal pathways

Mechanisms through which social deprivation limits participation in preventative medicine are unclear, although many have been postulated. Theories include deprived groups having; a limited locus of control, the degree to which individuals believe they control their lives; low future salience, the extent to which individuals contemplate the future; or lower awareness of current health status. Time orientation is likely to be important. This affects processing health information; Future orientation promotes positive health behaviours, whilst present orientation high risk behaviour. Future orientation is generally greater in the more
affluent and present orientation in the deprived.\textsuperscript{509} Finally the deprived have lower expectations of screening.\textsuperscript{506}

There is strong, but not universal evidence that the deprived use less preventative medicine. This is in conflict with general health care, where controlling for need, they use more services.\textsuperscript{510} Income and education both have strong evidence for impacting on uptake. They further have plausible pathways, the former in part through direct economic barriers and the latter through social inclusion, patience and self-efficacy. Ideally deprivation should be considered in its true complexity; its multiple components, its effect over the life course and its complex interactions.\textsuperscript{511} Pragmatically, however, this is often not possible.

\textbf{5.2.3 Ethnicity}

Ethnicity is a second demographic characteristic frequently studied in relation to screening uptake. Defining ethnicity proves difficult, and frequently becomes confused with race.\textsuperscript{512} Concerning health, an ethnic group can be defined as

\begin{quote}
\textit{a group that people belong to because of shared characteristics, including ancestral and geographical origins, cultural traditions, and languages.}
\end{quote}

Crucially, members of an ethnic groups require a consciousness of belonging to that group.\textsuperscript{513}

The presence of ethnic inequalities in screening uptake remains unclear, despite dogma to the contrary. Many studies show ethnic differences. In the UK, there is evidence of lower uptake of breast, colorectal and cervical screening in south Asian patients.\textsuperscript{459} \textsuperscript{514-516} Perceived appropriateness of procedures further differs by ethnic background.\textsuperscript{517} One review concluded that south Asian patients have the poorest uptake across all preventative services in London.\textsuperscript{518} Within the south Asian diaspora, there is evidence of further differences, with the Muslim population having poorer colorectal screening attendance than other south Asian groups.\textsuperscript{468} There is evidence that white patients have highest colorectal screening uptake,\textsuperscript{468}
with the poorest FOBT returns in ethnically diverse areas.\textsuperscript{469} \textsuperscript{505} In the USA there are ethnic differences in the uptake of mammography, cervical and colorectal screening.\textsuperscript{519}

There are questions over these differences, especially over BME groups \textit{per se} having poor uptake. One common problem with the evidence is confounding from SEP. Univariate analyses frequently demonstrate ethnic variation; once adjusting for SEP, this is lost.\textsuperscript{520} \textsuperscript{517} \textsuperscript{521} Indeed, black populations in the UK can show greater uptake than white.\textsuperscript{455} \textsuperscript{521} \textsuperscript{523} Looking broadly at the use of health promotion, one review concluded that ethnic differences do not exist outside of the relationship with SEP.\textsuperscript{454} A second, methodological problem is in the groupings chosen as units of study.\textsuperscript{511} These can be too broad, for example using south Asian not the constituent backgrounds such as Bangladeshi, Pakistani and Indian. The ethnic categories include a highly heterogeneous population; therefore can mask differences between the constituent groups.

If ethnicity \textit{per se} does not impact on uptake, closely related factors might. Being a non-English speaker can limit the use of health promotion\textsuperscript{454} and lower the awareness of national screening programmes.\textsuperscript{524} When incorporated, overseas birth, not ethnicity, better explains differences in uptake.\textsuperscript{522} Again, this could be through language or cultural barriers.

Yet more difficulties stem from a heterogeneous effect of ethnicity. Webb et al.\textsuperscript{525} found increased uptake of cervical screening in south Asian patients as the proportion in the general practice increased. Once more than 50 percent of a practice was of south Asian background, uptake was in fact higher than in the white population. Uptake may also improve with a clinician from the same ethnic group, especially if both are female.\textsuperscript{525} Finally, but of great importance to public health, there is evidence of the poorest uptake in ethnic minority women, possibly due to low literacy levels.\textsuperscript{521}

\textbf{5.2.4 Age}

Age is an important factor in the uptake of screening,\textsuperscript{455} although the relationship is not simple and linear, and may differ between services. There is evidence older patients have
greater participation, for example in diabetic retinopathy screening, mammography and FOBT. There is equally strong evidence, however, that in the very oldest patients uptake decreases, for example in breast screening and FOBT. Poor uptake in the oldest patients may be greater in more invasive screening procedure such as flexible sigmoidoscopy, which have greater discomfort and require greater fitness. Further, older patients may have a lower perceived risk of disease, which limits uptake.

5.2.5 Sex

There is some evidence of differences in screening uptake based on a patient’s sex; however, there has been limited opportunity for study with the majority of the work on cervical and breast screening. The introduction of colorectal screening programmes has allowed a greater evaluation of sex differences. Women have a higher use of FOBT, This is not, however, true for flexible sigmoidoscopy, with significantly greater uptake in men. Men consider discomfort as less of a barrier than women, which in part might explain the latter.

5.2.6 Marriage

Marriage stands out as a prominent factor associated with screening uptake. The major problem is, however, there is no indication of whether the relationship is causal or from a selection effect, whereby screening uptake and marriage share a common behavioural root. Married women were more likely to attend cervical and breast screening, although there is evidence contrary to the latter. Married patients have greater interest in colorectal screening and are more likely to attend. Finally, men had higher uptake of prostate specific antigen testing if living with a partner but not digital rectal examination. Marriage increases uptake in both sexes, with further gains if patients are jointly invited.

There are several mechanisms by which marriage could affect health behaviour. Marriage can cause an individual to give a greater control of their health to a partner. This control can either be active, through direct organisation or through passively absorbing health
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behaviours. Marriage has other potential implications. It could increase a patient’s responsibility for their own health because they feel part of a partnership. It can also have the more practical impact of increasing organisation.

5.2.7 Transience

There are strong theoretical reasons as to why a transient population have poor screening uptake. Transience can act in two distinct ways, one on the supply and one on the demand side of screening organisation. From the patient perspective, a transient population suffers from greater social exclusion and less cohesion. They are, therefore, both less likely to know about screening and less likely to attend when invited.

From the organisational perspective of screening, accurate medical registers- frequently in the UK derived from general practice EMRs- are essential. Records can be incorrect in two ways, either inflated (with more patients than actually present) or under recorded. These inaccuracies can be for a number of reasons. Inflation can be caused by patients registering twice, or patients dying or leaving a practice going unrecorded. Under recording simply happens when patients in a catchment area do not register with general practice. Transience is strongly linked to list errors. It creates a large practice turn-over which, based on the definitions above, could produce either list inflation or under reporting. In reality this manifests as list inflation, especially in young transient populations.

In the UK, practice registers are mostly inflated. In London, for example, there is an average of 15 percent inflation, with up to 18 percent in the inner city. Greater list inflation will theoretically decrease screening uptake. A rapidly changing practice register will make it impossible to maintain accurate invitation. A still greater impact of list inflation is not on actual screening uptake but on the level of uptake measured. If a practice has 15 percent list inflation, only 87 percent of the register actually attends. In other words, 13 percent of the registered population are simply not there to be screened; they are ghost patients. These patients can never be screened; therefore one could never reach an uptake greater than 87
percent. In order to reach an apparent uptake of 80 in the population above, 89 percent would need to be screened.\textsuperscript{533}

There is strong theoretical support as to why list inflation and therefore transience decrease screening uptake, but less empirical evidence. For list inflation itself, there is evidence of decreased cervical\textsuperscript{532 534} and breast screening uptake\textsuperscript{535} with greater list inflation, however others have failed to find a relationship.\textsuperscript{520} Transience has even less evidence; however there are frequently major flaws in the measures used.\textsuperscript{536} When stronger measures of mobility are used, including patient change of address\textsuperscript{520} and change in practice register,\textsuperscript{537} both find transience to reduce uptake.

Population mobility in London is the highest in the UK; with for example twice as much cross-boundary movement between boroughs as elsewhere. Some boroughs see up to a 35 percent population turnover per annum.\textsuperscript{530} There is also large list inflation,\textsuperscript{532 533} therefore any work carried out in London should, if possible, account for population mobility.

### 5.2.8 Health and health behaviours

Patients frequently exhibit poor health behaviours; for example smoking, physical inactivity, low seatbelt use, and low quality diet.\textsuperscript{453 538} Poor health behaviours cluster within patients and within lower socioeconomic groups.\textsuperscript{453} General poor health behaviours can be strong predictors of screening uptake.\textsuperscript{455} They are not, however, causal of poor uptake. Instead they may arise from the same underlying patient characteristics.

The uptake of cervical and breast screening are highly correlated at a patient and practice level\textsuperscript{454} and previous attendance at breast screening is one of the strongest predictors of future attendance.\textsuperscript{523} Smoking,\textsuperscript{491 500} a dislike of medical tests,\textsuperscript{490 539} infrequent dentistry\textsuperscript{491 500} and general practice attendance\textsuperscript{490} have all been associated with low colorectal screening attendance. In other screening procedures, there is evidence that low alcohol intake and frequent dentistry are predictive of cervical screening uptake; smokers are less likely to attend diabetic retinopathy screening\textsuperscript{465} and a wide range of good health behaviours promote
mammography.\textsuperscript{540} There is even evidence of a dose-response. One study found that each additional good health behaviour increased odds of screening uptake.\textsuperscript{541}

There are two further health factors which have been assessed in relation to screening uptake. Subjective health has a relationship with the perceived risk of disease.\textsuperscript{500} From here, one might expect a relationship with screening uptake, but evidence is mixed.\textsuperscript{490, 491, 500} Poor subjective health has been demonstrated as marker of poor colorectal,\textsuperscript{491, 492, 500} abdominal aortic aneurysm\textsuperscript{497} and breast\textsuperscript{493} screening attendance. Difficulties come when controlling for general practice attendance, the relationship is then attenuated.\textsuperscript{459} Finally there is limited evidence that a family history of a condition will increase uptake, with evidence from colorectal,\textsuperscript{492, 500} prostate\textsuperscript{457} breast cancer screening.\textsuperscript{504, 540} If true, this is likely through an increased perception of risk.\textsuperscript{527}

5.2.9 Informed choice

Informed choice has become increasingly important in healthcare, especially in prevention and screening. Greater individual choice in health care has followed wider increases in consumer choice.\textsuperscript{542} Traditionally public health has had a solitary aim; to reach the largest possible population in order to achieve the greatest good. In screening, high uptake was considered to outweigh any harm from limited patient information.\textsuperscript{542} Patients were not required to decide whether they would attend, therefore did not need information about the screening procedure.

This traditional, paternalistic, approach to public health is now being replaced by informed choice. The UK national screening committee state,

\textit{There is a responsibility to ensure that people who accept an invitation (to screening) do so on the basis of informed choice, and appreciate that in accepting an invitation or participating in a programme to reduce their risk of a disease there is a risk of an adverse outcome}\textsuperscript{450}
*Informed consent* is the positive decision made by a patient involved in informed choice. To give informed consent, a patient must have “*both knowledge and comprehension*” of the procedure and “*give consent freely without duress or undue influence*.”47 The decision must be based on good and relevant information, and be dependent on and applicable to the patient’s views.543

An array of information is needed for informed consent. This includes the risk of disease, harms of the procedure, test accuracy and implications of a positive test. Provision of information is a vital component of informed choice, the format of information is therefore important.544 Information is not, however, the sole ingredient. An autonomous, unbiased decision must be made, with different options available, each without barriers.

Some argue that informed choice is the antithesis of traditional public health and therefore of efforts to improve uptake.542 There is, though, no evidence that informed choice reduces uptake.545 In addition to reductions in overall uptake, some fear that informed choice is inherently bad for health inequalities. Processing of information is central to informed choice. This may vary based on SEP, for example due to differences in education and time orientation.506 Finally the patients themselves might not be supportive; some prefer to follow clinician advice, feeling they ‘know best’.546

Informed choice presents an ethical dilemma. It is fundamentally considered a good thing; however, there is strong theoretical basis for it increasing health inequalities. A more holistic view of inequalities might change this view. Informed choice itself does good, increasing psychological wellbeing and improving self-efficacy. These in turn improve physical health and further health behaviours.506 The deprived have the poorest baseline psychological health; informed choice will therefore do them the greatest good. If considering this wider view of health one could argue inequalities are inevitably reduced.506 A consideration of informed choice can be sculpted by ones beliefs. Traditional public health is fundamentally utilitarian but one must decide whether such a paternalistic approach is appropriate. In
aspiring for equality one must decide what exactly is to be made equal; one specific aspect of health or a more holistic health.

5.2.10 Structural factors of screening

The distance travelled to attend a screening procedure seems a likely candidate to influence uptake. Greater distance requires greater effort, hence reduces attendance. The cost to benefit ratio for the participant is altered.\textsuperscript{451} Again, however, when included in analyses findings are mixed. Studies on breast,\textsuperscript{462, 523} diabetic retinopathy\textsuperscript{165} and abdominal aortic aneurysm\textsuperscript{497} screening have all failed to find a significant effect. Conversely remoteness has predicted cervical screening uptake\textsuperscript{536} and distance of travel reduced the use of mobile breast screening units.\textsuperscript{547} It is possible there is a minimum distance, beyond which further proximity is no longer beneficial. Null findings in the UK may reflect this.

For screening programmes situated in general practice (mainly cervical screening), practice list size has a differential relationship with screening uptake. Larger practices demonstrate improved uptake,\textsuperscript{523, 525, 534, 548} although only one study used patient level data.\textsuperscript{525} One study, conversely, gives greatest uptake in smallest practices. The number of GPs per practice may not attenuate this relationship.\textsuperscript{520} One might, therefore, conclude larger practices gain from improved organisation, with the ability to employ more additional staff and use better recall systems.\textsuperscript{520}

The characteristics of a patient’s GP or the practitioner involved in screening are important in screening programmes, especially ones targeted at women. Both breast\textsuperscript{461} and cervical\textsuperscript{520} screening rates are higher in women with a female GP. The introduction of male mammographers in the Republic of Ireland resulted in a large drop-out of women from the service.\textsuperscript{549} Outside of sex, practices with GPs of south Asian origin were found to have lower cervical screening uptake.\textsuperscript{525} Although the GP is not directly involved in the screening process, they can influence uptake. If a GP sees screening as advantageous, they can promote it during consultations or facilitate its organisation.\textsuperscript{67}
Centralised screening programmes possess a number of desirable characteristics. They have better quality assurance, improved patient follow-up and recall, clear eligibility criteria, better control of budget and evaluation.\textsuperscript{294} They are believed to offer better value. The main practical difference between a centralised programme and locally implemented projects is the source of invitation.\textsuperscript{294} They, as a result, tend to have fixed appointments whereas localised programmes rely on opportunistic screening. Fixed appointments increase uptake,\textsuperscript{550} whilst after opportunistic screening patients are left less well informed.\textsuperscript{546} Opportunistic screening can also annoy patients who are unhappy to discuss additional issues outside of the primary reason for consultation.\textsuperscript{551}

In the UK, response rates to cervical screening increased once the programme became centralised in 1988.\textsuperscript{536, 552} Some consider centralised programmes inherently more able to reduce inequalities in screening uptake.\textsuperscript{294} Despite a lag, national cervical screening in the UK did improve inequalities.\textsuperscript{537} In Belgium, like the UK, a disparate series of provincial cervical projects was moulded into a national programme. After becoming centralised, however, there was no immediate reduction in inequalities.\textsuperscript{553}

5.3 General practice usage and ‘health check' uptake

5.3.1 General practice

Across much of England, the NHS Health Check programme will be carried out in general practice. The underlying rates of general practice attendance are therefore an important factor in Health Check attendance.

Women have higher rates of attendance at general practice than men.\textsuperscript{455, 510, 554-556} They are both more likely to be highly frequent attendees and less likely to be highly infrequent.\textsuperscript{557, 558} Reproductive health care is believed to contribute to difference in consultation rates.\textsuperscript{559, 560} Especially amongst young adults, women attend general practice regularly for contraception.
and other reproductive needs. Even accounting for reproductive needs however, women still see excess attendance. Additional explanations include women displaying more symptoms, especially psychological; having greater morbidity; being more predisposed to seek health care or with lower employment rates having greater opportunity to attend.

General practice consultation rates are bi-modal with regards to age. Children and older patients have the highest rates of attendance. Differences with age are widely regarded as ‘legitimate’, therefore little further work has been done in this area. Observed differences are mediated by genuine health care needs. Therefore although the usage is unequal, it is not a health inequality.

There is an interaction between age and sex in general practice attendance. There is a greater difference between the sexes in young adults (Figure 5-1). This is in part caused by the reproductive needs described above, however other factors influence usage. Women have relatively constant attendance with age whilst men increase (Figure 5-1).
There is evidence that ethnic minority groups in the UK have higher levels of GP attendance than the white population,\(^{454}\) although there is less recent evidence. There is especially strong evidence of a high usage of general practice in Indian, Pakistani and Black patients.\(^{563-565}\) Having controlled for health care need, ethnic differences are reduced but still evident, especially in south Asian groups.\(^{454}\) Not all ethnic minority groups have increased attendance; patients of Chinese ethnic origin and young Pakistani women have low attendance.\(^{454} 556\) The latter finding is in part caused by a preference for female GPs restricting usage, although they may face wider barriers to access.\(^{556}\) Chinese patients, with both lower registration at general practice and attendance, may have greater language barriers than other ethnic groups.\(^{454}\)

Quality of care received may be a driver of ethnic differences in general practice consultation rates.\(^{454}\) Despite more frequent attendance, ethnic minority groups are less likely to leave general practice with a referral to further care.\(^{564}\) If need for referral is equal between ethnic groups, the white population reaches the desired outcome more easily. One can therefore imply the consultation was of greater quality.

Language and understanding are potential drivers of quality. Ethnic minority patients can have a poor mutual understanding with their GP, which leads to a poor relationship.\(^{566}\) Non-English speaking patients have longer consultation times.\(^{567}\) This may be partially causal of difference between ethnic groups. BME groups may require more time to reach a desired outcome because of language, therefore having lower consultation quality. In some BME groups, notably from black and Chinese backgrounds, actual quality measures, including waiting times and continuity of care, explain the poorer perceptions of quality compared with white.\(^{568}\) There may, however, be differences in reported quality beyond actual differences. South Asian patients for example, may have higher expectations of care which further affects reported quality.\(^{568} 569\) Increased GP attendance in minority groups might not entirely be unnecessary. A greater need, especially from non-specific symptoms, may be present.\(^{564}\)
Using a range of measures of SEP, there is lower general practice consultation in affluent groups.\textsuperscript{454, 555, 563, 570} Much of this is driven by need, however like minority ethnic groups there is some question over consultation quality. Despite low SEP groups seeking primary health care more quickly when ill,\textsuperscript{571} they see lower referral rates from primary to secondary care.\textsuperscript{572} One study looked at differences in type of referral by SEP. The excess in the affluent was entirely due to referrals to a consultant.\textsuperscript{573} Given it unlikely the deprived have less need for a consultant, they may be less able to achieve the best outcome from a GP consultation. It is not however clear whether the affluent are over-referred.

5.3.2 Health check uptake

There have been several trials examining the effectiveness of the primary prevention of CVD in primary care, the majority of which have employed a ‘health check’ approach (Chapter 3.2.2/3). Major trials including the BFHS\textsuperscript{70} and SELSS\textsuperscript{72} have not described patterns in uptake. A third major trial in the UK, the OXCHECK study,\textsuperscript{574} found greater attendance in women, married participants, higher social classes and those who owned cars and houses. Higher uptake of health checks in women has also been demonstrated elsewhere.\textsuperscript{575, 576}

Two trials in the 1980s carried out opportunistic health checks in general practice. Attendance decreased with deprivation\textsuperscript{576, 577} and in patients with lower education.\textsuperscript{576} New patient health checks in general practice also found poorest attendance in the lowest social classes.\textsuperscript{501} A recent health check project, situated in both general practice and pharmacy found higher uptake in both south Asian and black patients, and older patients.\textsuperscript{147}

A small number of studies have assessed the relationship between health behaviours and health check attendance. Infrequent general practice attendance,\textsuperscript{574, 575} smoking,\textsuperscript{147, 501, 574, 577} drinking and poor diet\textsuperscript{577} are markers of poor uptake. Age and ethnic differences have not frequently been assessed in the UK; although poorer uptake in black patient,\textsuperscript{501} and older non-attendees\textsuperscript{576} have been found. Internationally, in the Malmo Prevention Trial attendees
were more likely to be co-habiting with a partner, but no further inequalities were apparent. Other major studies, including MRFIT, failed to assess uptake.

5.4 Summary of screening uptake

There is relatively little work around inequalities in the uptake of cardiovascular risk assessment in a healthy population. From research undertaken, there is some evidence of lower uptake in men and deprived patients. Differences in usage of general practice are considerably more established. Differences by age, sex, ethnic and SEP are all present. With the NHS Health Check frequently situated in general practice; these inequalities will be important in Health Check uptake. Community screening will potentially overcome this barrier, although this is not without weaknesses.

There is wider evidence of inequalities in the uptake of screening and preventative medicine. Summary measures of SEP and education have links to uptake, with economic factors showing some importance. Having controlled for SEP, there is no strong evidence of overall ethnic variations in health promotion, however there may be some differences between programmes. Language, not ethnicity is a plausible risk factor for poor uptake; with efforts to overcome language barriers important. Married people frequently have improved usage of health promotion. This might not be considered an important health inequality, but efforts to reach single patients might improve uptake. Screening programmes do differ in their barriers to uptake, but some commonalities exist and previous inequalities give important evidence for the implementation of a novel screening programme.

Transience is an important consideration surrounding the uptake and provision of any population based public health intervention. Transient populations make patient registers less accurate. This severely undermines any structured call-recall system. Transience is further important in measuring uptake. The presence of ghost patients drastically limits the uptake that can be achieved. Poor health behaviours do not cause poor uptake, but they are
markers of it. Since poor health behaviours cluster together, those with the greatest need for an intervention (the NHS Health Check) are the least likely to attend. Although these inequalities may be governed by underlying social inequalities, efforts to promote uptake in these groups is vital.
Chapter 6; Aims, scope and justification of work

Summary of the literature

- Despite a large reduction over the last four decades, CVD remains the greatest cause of morbidity, mortality and health spending in high-income countries.
- Due to the projected aging population, high-income countries face a pivotal period in CVD care; unless incidence is reduced through primary prevention, health systems may not be able to maintain CVD spending.
- Dramatic social and ethnic disparities exist in CVD which are the single greatest underlying contributor to health inequalities in high income countries.
- Despite growing evidence for a number of population approaches to the primary prevention of CVD, high-risk strategies remain the most common in practice.
- The NHS Health Check programme is a major UK spending commitment to the primary prevention of CVD; there are, however, a number of outstanding questions over the strategy employed and its implementation.
- CVD risk scores are effective in the population level stratification of risk and may prove useful in a targeted primary prevention of CVD.
- A number of social inequalities are common, both in the uptake of screening and attendance at general practice.

Justification of thesis

The NHS Health Check programme is a major commitment of spending for the UK government and time for health care professionals. Given this commitment, however, there is limited international evidence of the successful implementation of universal high risk primary prevention. The literature uncovers a number of questions over methodologies employed. These questions include the extent to which primary prevention interventions
benefit those outside of the high risk groups; the effectiveness of brief lifestyle interventions;
the ability of non-clinicians to effectively and safely communicate CVD risk and the presence
of psychological harms, most notably through inappropriate reassurance, in CVD prevention.
Further, there is evidence of a number of effective alternatives, including population
strategies and targeted high risk approaches. Evaluation of the programme is therefore vital
from the outset to ensure a safe, effective and cost-effective service.

The success of high risk prevention relies heavily upon uptake. Incomplete uptake and
adherence quickly diminishes the population impact of interventions. With no previous
experience of a national CVD prevention programme, one must draw on experiences from
clinical trials. Trials have suffered deficiencies in uptake, most notably Healthy Hearts study-
the trial most transferable to contemporary English general practice, achieved only 29
percent attendance. The absolute levels of uptake of the Health Check programme must be
constantly monitored to ensure an effective programme; this will be the first work to do so.
Similarly, to be effective, high risk prevention strategies must modify risk in the target
population. There are currently no data concerning the uptake of interventions following a
Health Check. Using one high profile exemplar, the prescription of statins to high risk
patients, I assess the uptake of intervention following the Health Check.

More than merely reducing the national burden of CVD, the Department of Health hopes the
Health Check programme will reduce well established social inequalities in cardiovascular
health. These are the core of wider English health inequalities. High risk interventions, most
notably screening programmes, and general practice consultation experience unequal
uptake. Frequently, these inequalities run parallel with health care need; those with greatest
need exhibit poorest attendance. For this reason, any application of high risk prevention has
the potential to exacerbate health inequalities and must be monitored. I shall therefore
examine patient and practice level patterns in uptake.

Central to the Health Check programme is the definition of high risk patients, those at greater
than or equal to 20 percent ten year risk of CVD. This population, once defined, becomes
eligible for more intensive risk-lowering intervention. The size of this population will therefore impact on the workload and costs generated by the programme. Further, given that this population is central to the programme, monitoring its size will be a useful metric of success. There are, however, no national estimates of its size, with even less known about the prevalence of CVD risk factors within this population.

There is growing support behind the effectiveness of targeted high risk prevention compared with universal approaches. Such methods can capture the vast majority of CVD risk within a population, in a highly cost effective manner. The more complete the medical record data used to target screening, however, the more accurate prediction. With no recent assessment of the completeness of CVD risk factor data in primary care medical records in the general population, I aim to address this, and additionally assess inequalities in recording.

A targeted approach to high risk CVD prevention, based on medical record data, will inevitably encounter missing risk factor data. There are different methods available for substituting these missing data. Methods vary in terms of complexity, and may differ in the accuracy of estimation. I therefore look to compare methods of data imputation when risk-stratifying patients using incomplete medical record data.

The CVD risk score is central to both the NHS Health Check programme and any alternate, targeted approach to CVD prevention. Despite varying evidence of accuracy of different risk scores, the Health Check programme permits the use of two, QRISK2 and JBS2. Aside from the previously documented differences in overall risk prediction, the two risk score account for the ethnic variance in CVD risk differently. Given marked ethnic disparities in the burden of CVD, there is no evidence how a risk score’s consideration of ethnicity will impact on risk prediction, and therefore the ethnic equity of prevention. Differences in prediction may further affect inequalities in work load dependent on the ethnic composition of a population. I shall examine this in data from a highly ethnically diverse area of England.
Aim

To assess the early impact of the NHS Health Check programme in general practice, and to examine the workload implications.

Objectives

- To assess ethnic group differences in risk scoring, using the two CVD risk scores permitted within the NHS Health Check programme.
- To assess methods of dealing with missing data in the risk stratification of patients, using data from electronic medical records.
- To estimate the number of high risk patients (greater than 20 percent risk) identified in England from the Health Check programme and the prevalence of risk factors in this population.
- To determine the levels of CVD risk factors recorded in electronic medical records before the NHS Health Check programme.
- To provide the first estimate of the levels of patient uptake of the Health Check programme and to examine variation by patient and practice characteristics.
Chapter 7; Ethnic group differences in cardiovascular risk estimates using JBS2 and QRISK2 risk scores: national cross-sectional study

7.1 Introduction

Cardiovascular risk scores have become widely incorporated into guidance for the prevention of CVD. Despite this, they are often not routinely applied to primary care practice in many countries. In England, they currently lie at the heart the NHS Health Check programme.

The primary aim of the NHS Health Check programme is to lower the burden of CVD in England. A major secondary aim, concurrent to this, is to reduce overarching population wide health inequalities, of which CVD is a major causal factor (Chapter 1.1.5). A socio-economic gradient has been apparent since the 1970s and ethnic disparities in both incidence and mortality have persisted in the UK and worldwide. South Asian patients in the UK have up to 1.8 times higher mortality from ischaemic heart disease than their white counterparts and black patients a 1.5 to 2.5 times higher stroke mortality.

Risk scores must accurately reflect ethnic difference in risk if they are to reduce health inequalities. The two risk scores permitted for use in the NHS Health Check, JBS2 and QRISK2, account for ethnicity in different ways. Ethnicity is directly incorporated as a component of QRISK2, although there were relatively few ethnic minority individuals in the derivation dataset from which risk estimates were calculated.

The Framingham Heart study algorithm, on which JBS2 was based, did not incorporate ethnicity. It was, therefore, adapted for use in the UK by adding a simple multiplication factor of 1.4 for south Asian men. This adjustment factor based on CVD prevalence data, however, has little evidence to support it and does not account for raised risk in south Asian women.
If a risk score over-estimates risk, the programme will potentially expose patients to increased harm, with unnecessary therapy and increased anxiety over their risk status. Further, the programme is likely to be less cost effective, with interventions administered to a greater number of patients with less relative gain in lower risk patients. Conversely, under-estimating risk will exclude high risk patients from necessary treatment.

Given the potential consequences of poor risk estimation, including widening of inequalities, increased exposure to iatrogenic harm and reduced cost-effectiveness, I aimed to assess the difference in risk classification in a more ethnically diverse population than used previously drawn from national health survey data, using the QRISK2 and JBS2 risk scores. I concentrate on differences between black, south Asian and white populations, with emphasis on the impact of the risk multiplier applied to south Asian men in the JBS2 score.

### 7.2 Methods

**Data source**

I conducted analyses using data from the HSfE. The HSfE is an annual survey of individuals living in private households and a primary mechanism for monitoring population health in England. The methodology of data collection is described in detail elsewhere. Small geographical areas are randomly selected, and within these a number of households. All adults within selected households are eligible for survey. Interviewers record socio-economic, personal details, information on health and health service use from respondents. Trained nurses record anthropometric measurements including blood pressure, BMI, take blood specimens and ask respondents about prescribed medications.

I used data from the 2003 and 2004 HSfEs. The 2003 survey was conducted with a representative sample of the general population, whilst the 2004 survey focused on ethnic minority communities. It contained a general population sample (approximately half the size of that in the previous year) and an ‘ethnic boost’ sample involving stratified multistage
probability sampling. I only used data from the ethnic boost sample in 2004 because interviews with general population sample did not include cardiovascular disease questions.

I selected respondents aged between 35 and 74, the age range in which the risk scores are valid, and excluded patients with self reported diagnosed CVD. In addition to the standard HSfE data, I obtained 2001 Townsend deprivation and IMD 2007 scores for each informant based on their postcode of residence.\textsuperscript{406, 460} Table 7-1 lists the variables from the dataset used to produce the risk scores. In the HSfE there are no data recorded for Rheumatoid Arthritis, heart failure or AF although they were present in the QRISK2 score.

### Table 7-1: the variables included in QRISK2 and JBS2 score, and levels of missing data

<table>
<thead>
<tr>
<th></th>
<th>QRISK2</th>
<th>JBS2</th>
<th>% Missing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age‡</td>
<td>x</td>
<td>x</td>
<td>-</td>
</tr>
<tr>
<td>Sex‡</td>
<td>x</td>
<td>x</td>
<td>-</td>
</tr>
<tr>
<td>Ethnicity‡</td>
<td>x</td>
<td>†</td>
<td>0.7%</td>
</tr>
<tr>
<td>Deprivation*‡</td>
<td>x</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>x</td>
<td>x</td>
<td>21.8%</td>
</tr>
<tr>
<td>Hypertension‡</td>
<td>x</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes‡</td>
<td>x</td>
<td>x</td>
<td>-</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>x</td>
<td>x</td>
<td>29.4%</td>
</tr>
<tr>
<td>Cholesterol/ HDL</td>
<td>x</td>
<td>x</td>
<td>45.5%</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>x</td>
<td></td>
<td>11.4%</td>
</tr>
<tr>
<td>Family History of CVD</td>
<td>x</td>
<td>†</td>
<td>7.2%</td>
</tr>
<tr>
<td>Heart Failure</td>
<td>x</td>
<td></td>
<td>ALL</td>
</tr>
<tr>
<td>Chronic Kidney Disease‡</td>
<td>x</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Rheumatoid Arthritis</td>
<td>x</td>
<td></td>
<td>ALL</td>
</tr>
<tr>
<td>Atrial Fibrillation</td>
<td>x</td>
<td></td>
<td>ALL</td>
</tr>
</tbody>
</table>

| † Not included within the algorithm, but adjustment factors are added |
| ‡ Variables used in the multiple imputation of missing data |
*Townsend Score for QRISK2/ IMD2007 for other analysis

I included three ethnic groups in the study; in the survey data these were coded as white, Asian or British Asian and Black or Black British (hereafter referred to as white, south Asian and black respectively). The data contained missing values for a number of variables (Table 7-1). I excluded patients with missing ethnicity records (473 (4.0%)) and those not from White, Black and south Asian ethnic groups (600 (5.0%)). I used multiple imputation to estimate values for missing blood pressure, total cholesterol, high-density lipoprotein (HDL)
cholesterol, BMI and smoking status.\textsuperscript{14} Using the \textit{MI} suite of commands in Stata, I generated 10 imputed datasets (\textit{m}), running linear imputation models (logistic regression for smoking status) to impute missing values.

I built the regression models used in the imputation by backwards stepwise selection, using the Akaike Information Criterion (AIC) to assess the model fit.\textsuperscript{580} I took variables with complete recording as candidates for inclusion in the models, these are shown in Table 7-1; in addition I used interaction terms between age and sex, and ethnicity and sex. The final models selected for each imputed variable are shown in \textit{appendix}, Table viii-1. I calculated the fraction of missing information (\(\gamma\)) for each imputed variable assessing whether \(m\) was sufficiently large.\textsuperscript{581} For each variable imputed, \(m=10\) gave adequate power of imputation. Values in the imputed dataset outside the range valid for the QRISK2 algorithm were dropped. For the remaining variables, including Rheumatoid Arthritis, heart failure and AF, I assumed the condition to be absent if missing, in line with the guidance for QRISK2.\textsuperscript{83} In total, I had data for 11,468 respondents.

\textit{Analysis}

I compared CVD risk factors between white, black and south Asian ethnic groups separately in men and women, applying t-tests to assess differences. I present the age and deprivation breakdowns of the population, plus age-standardised risk factor summaries. I used the direct method of standardisation, with eight equal age groups, using the total population as the standard. I applied the QRISK2\textsuperscript{83} (after the 2010 update\textsuperscript{582}) and JBS2\textsuperscript{60} risk scores to each imputed copy of the dataset, calculating the mean score across imputations to give a single score per respondent. I summarised levels of the two risk scores and differences between them, in each sex/ethnicity group, and the proportion designated as at high risk (\(\geq20\%\)) - again using direct age standardisation. I ranked respondents in each sex/ethnicity group based on each risk score, comparing using Spearman’s correlation coefficient and calculated Cohen’s kappa to assess agreement between high risk classifications. I assessed agreement between risk scores using a Wilcoxon signed-rank test, due to skewed data. To
compare the risk scores over different levels of risk, I plotted the running mean of the JBS2 score over the range of QRISK2 score. The running mean reduces small scale fluctuation.

I assessed changes in risk classification [high (≥20%), moderate/ low (<20%)] by changing the risk score used to stratify patients, both in the total population, and separately in ethnic groups. I summarised levels of CVD risk factors in high risk groups defined by each risk score, and examined changes in risk score across levels of risk factors by plotting running means. I compared differences between risk scores across population tenths of deprivation, testing using a Wilcoxon rank-sum test. I carried out a sensitivity analysis, re-running all analyses using only the complete data. I present asymmetric confidence intervals at a level of 95%, using a logit transformation. All analyses were carried out using StataSE 11.1.

7.3 Results

Characteristics of the population are in Table 7-2. The majority of respondents with recorded ethnicity were white (71.4%); 13.8% were of south Asian and 9.6% of black ethnicity. Black men had higher mean systolic blood pressure (t-test p<0.001 compared with white men). Black women had a higher mean BMI and levels of obesity (p<0.001 compared with white for both). There was a greater prevalence of diabetes in south Asian respondents, especially men (p<0.001 compared with white), and higher total cholesterol in the white population (in both sexes p<0.001 compared with south Asian and black).

The mean JBS2 risk score in the total population was 13.0% [95% confidence interval (CI) = 12.8-13.2], median= 9.3% [interquartile range (IQR) 14.5] and mean QRISK2 score was 11.0% [10.8-11.2], median= 6.6% [14.4]. There was a statistically significant difference between mean risk scores of 2.0% [1.9- 2.1] (median of 1.5% [4.2]) which was statistically significant (p<0.001 using a Wilcoxon signed-rank test).
The mean QRISK2 score was significantly lower than the mean JBS2 score in men, with a significantly larger difference in south Asian compared with other respondents. In women, the mean QRISK2 score was on average lower in all except black respondents, though the difference was smaller than for men (Table 7-3).

There was a lower proportion of the population at ≥20 percent risk with QRISK2 compared with JBS2 in men in all ethnicities, with the greatest difference in the south Asian group (19.1% [16.2- 22.0, compared with 8.8% [5.9-9.8] in white). When assessing agreement of high risk status, the kappa values demonstrate significantly poorer agreement in south Asian men. Significantly more in all three ethnic groups were classified as ≥20% risk using QRISK2 than JBS2.
Table 7-3: the age adjusted risk scores, and proportion at >20% risk in ethnic groups

<table>
<thead>
<tr>
<th></th>
<th>Mean risk score</th>
<th>Proportion high risk</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>White</td>
<td>South Asian</td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mean risk score</strong></td>
<td>JBS2</td>
<td>9.3</td>
</tr>
<tr>
<td></td>
<td>QRISK</td>
<td>8.7</td>
</tr>
<tr>
<td>Difference (95% CI)</td>
<td>0.6 (0.5 to 0.7)</td>
<td>0.3 (0.1 to -0.4)</td>
</tr>
<tr>
<td>Spearman†</td>
<td>0.94</td>
<td>0.94</td>
</tr>
<tr>
<td><strong>Proportion high risk</strong></td>
<td>JBS2</td>
<td>12.3</td>
</tr>
<tr>
<td></td>
<td>QRISK</td>
<td>14.0</td>
</tr>
<tr>
<td>Difference (95% CI)</td>
<td>-1.7 (-1.1 to -2.4)</td>
<td>-2.5 (-0.5 to -4.5)</td>
</tr>
<tr>
<td>Kappa (se) ‡</td>
<td>0.69 (0.01)</td>
<td>0.75 (0.03)</td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mean risk score</strong></td>
<td>JBS2</td>
<td>16.7</td>
</tr>
<tr>
<td></td>
<td>QRISK</td>
<td>13.4</td>
</tr>
<tr>
<td>Difference (95% CI)</td>
<td>3.3 (3.1 to 3.4)</td>
<td>9.7 (9.2 to 10.3)</td>
</tr>
<tr>
<td>Spearman†</td>
<td>0.93</td>
<td>0.91</td>
</tr>
<tr>
<td><strong>Proportion high risk</strong></td>
<td>JBS2</td>
<td>32.7</td>
</tr>
<tr>
<td></td>
<td>QRISK</td>
<td>25.9</td>
</tr>
<tr>
<td>Difference (95% CI)</td>
<td>8.8 (5.9 to 9.8)</td>
<td>19.1 (16.2 to 22.0)</td>
</tr>
<tr>
<td>Kappa (se) ‡</td>
<td>0.72 (0.02)</td>
<td>0.60 (0.03)</td>
</tr>
</tbody>
</table>

*se= standard error*

Table 7-4: high risk status using the JBS2 and QRISK2 scores in white and south Asian populations; number (percentage of total)

<table>
<thead>
<tr>
<th></th>
<th>Low/ Moderate</th>
<th>High</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Male</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>JBS2</td>
<td>Low/ Moderate</td>
<td>2267 (62.5)</td>
<td>81 (2.2)</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>356 (9.8)</td>
<td>923 (25.4)</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>2623 (72.3)</td>
<td>1004 (27.7)</td>
</tr>
<tr>
<td>QRISK2</td>
<td>Low/ Moderate</td>
<td>445 (58.9)</td>
<td>6 (0.8)</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>131 (17.3)</td>
<td>174 (23)</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>576 (76.2)</td>
<td>180 (23.8)</td>
</tr>
<tr>
<td><strong>Female</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>JBS2</td>
<td>Low/ Moderate</td>
<td>3687 (80.9)</td>
<td>233 (5.1)</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>131 (2.9)</td>
<td>509 (11.2)</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>3818 (83.7)</td>
<td>742 (16.3)</td>
</tr>
<tr>
<td>QRISK2</td>
<td>Low/ Moderate</td>
<td>723 (87.8)</td>
<td>24 (2.9)</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>13 (1.6)</td>
<td>63 (7.7)</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>736 (89.4)</td>
<td>87 (10.6)</td>
</tr>
<tr>
<td><strong>Black</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>JBS2</td>
<td>Low/ Moderate</td>
<td>338 (71.9)</td>
<td>7 (1.5)</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>31 (6.6)</td>
<td>94 (20)</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>369 (78.5)</td>
<td>101 (21.5)</td>
</tr>
<tr>
<td>QRISK2</td>
<td>Low/ Moderate</td>
<td>554 (87.7)</td>
<td>24 (3.8)</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>4 (0.6)</td>
<td>50 (7.9)</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>558 (88.3)</td>
<td>74 (11.7)</td>
</tr>
</tbody>
</table>
Chapter 7: Ethnic group differences in cardiovascular risk estimates using JBS2 and QRISK2 risk scores

In changing from using the JBS2 to QRISK2 score to risk stratify patients (Table 7-4), 9.6% of the sample would have their risk status reclassified. 26.5% of the high risk JBS2 group would be reclassified as at low risk using QRISK2 and 17.6% of the high risk QRISK2 group would be reclassified as at low risk using JBS2. There is greater total risk reclassification in men (12.6% [11.7- 13.5]) than in women (7.1% [6.5- 7.8]). In men, the south Asian group has the greatest risk reclassification, 18.1% [15.4- 20.9] compared with 12.0% [11.0-13.1] and 8.1% [5.6-10.6] in the white and black groups respectively. The majority of this reclassification in south Asian men is the downgrading of high risk JBS2 patients, with 43.0% [37.4-48.5] of the total high risk JBS2 downgraded compared with 27.8% [25.4-30.3] in white patients and 24.8% [17.1- 32.5] in black respondents.

![Figure 7-1](image)

Figure 7-1: the running mean of the JBS2 risk score over QRISK2 in white and south Asian patients

Figure 7-1 presents mean JBS2 score over levels of QRISK2 in the three ethnic groups, for men and women. Mean JBS2 scores were higher than QRISK scores over all levels of risk in south Asian and black men, especially the former. In white men the JBS2 score is higher up to a risk score of approximately 40, above which the QRISK score is higher (Figure 7-1a). In women the two risk scores were comparable in all ethnicities up to a score of approximately 35, beyond which QRISK2 estimates a higher level of risk (Figure 7-1b).
There were differences in the characteristics of high risk populations defined using the two scores (Table 7-5). The high risk population defined using JBS2 was younger on average, especially in men. In south Asian and black women, and white men there was higher systolic blood pressure using JBS2 and more patients with blood pressure over 140/90 mm Hg. There was a consistently higher prevalence of diabetes in the population defined using QRISK2, and frequently a higher BMI and levels of obesity.

Table 7-5: The CVD risk factors in the high risk populations defined by JBS2 and QRISK2 scores

<table>
<thead>
<tr>
<th></th>
<th>White</th>
<th>JBS2</th>
<th>QRISK2</th>
<th>South Asian</th>
<th>JBS2</th>
<th>QRISK2</th>
<th>Black</th>
<th>JBS2</th>
<th>QRISK2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Male</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years; se)</td>
<td>61.6 (0.21)</td>
<td>64.1 (0.21)</td>
<td>57.8 (0.50)</td>
<td>61.3 (0.68)</td>
<td></td>
<td></td>
<td>62.6 (0.75)</td>
<td>64.9 (0.75)</td>
<td>62.6 (0.75)</td>
</tr>
<tr>
<td>Systolic BP (mean; IQR)</td>
<td>142.2 (18.5)</td>
<td>141.3 (19.2)</td>
<td>135.7 (14.3)</td>
<td>137.7 (15.5)</td>
<td>147.5 (17.1)</td>
<td>147 (17.1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BP &gt; 140/90 (%; SE)</td>
<td>51.8 (1.4)</td>
<td>52.1 (1.6)</td>
<td>30.2 (2.6)</td>
<td>35 (3.6)</td>
<td>66.4 (4.2)</td>
<td>65.3 (4.8)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Cholesterol (mean; IQR)</td>
<td>6 (0.9)</td>
<td>6 (0.9)</td>
<td>5.5 (0.8)</td>
<td>5.5 (0.9)</td>
<td>5.6 (0.9)</td>
<td>5.6 (0.9)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Chol &gt;5 (%; SE)</td>
<td>90.2 (0.8)</td>
<td>89 (1)</td>
<td>77.7 (2.4)</td>
<td>76.7 (3.2)</td>
<td>82.4 (3.4)</td>
<td>81.2 (3.9)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI (mean; IQR)</td>
<td>28.3 (4.9)</td>
<td>28.4 (5.1)</td>
<td>26.4 (4.2)</td>
<td>26.4 (4.1)</td>
<td>28 (4.3)</td>
<td>28.4 (4)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese (%; SE)</td>
<td>29.1 (1.3)</td>
<td>30.8 (1.5)</td>
<td>13.4 (2)</td>
<td>14.4 (2.6)</td>
<td>27.2 (4)</td>
<td>25.7 (4.4)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current smoking (%; SE)</td>
<td>45.7 (1.5)</td>
<td>35.3 (1.6)</td>
<td>53.6 (3.4)</td>
<td>47.2 (4.5)</td>
<td>51.3 (5.6)</td>
<td>38.5 (6.1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes (%; SE)</td>
<td>8.6 (0.8)</td>
<td>10.8 (1)</td>
<td>27.2 (2.6)</td>
<td>37.8 (3.6)</td>
<td>16.8 (3.4)</td>
<td>19.8 (4)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Female</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years; se)</td>
<td>65.7 (0.26)</td>
<td>67.8 (0.20)</td>
<td>63.6 (0.71)</td>
<td>65.2 (0.69)</td>
<td></td>
<td></td>
<td>66.8 (0.79)</td>
<td>66.5 (0.74)</td>
<td>66.5 (0.74)</td>
</tr>
<tr>
<td>Systolic BP (mean; IQR)</td>
<td>149.8 (21.8)</td>
<td>146 (20.3)</td>
<td>145.9 (15.1)</td>
<td>142.2 (13.7)</td>
<td>143.6 (9.6)</td>
<td>139.6 (15)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BP &gt; 140/90 (%; SE)</td>
<td>68.1 (1.8)</td>
<td>60.6 (1.8)</td>
<td>63.2 (5.6)</td>
<td>54 (5.4)</td>
<td>72.2 (6.2)</td>
<td>62.2 (5.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Cholesterol (mean; IQR)</td>
<td>6.6 (1)</td>
<td>6.6 (1)</td>
<td>5.7 (1.4)</td>
<td>5.8 (1.3)</td>
<td>5.7 (0.8)</td>
<td>5.7 (1.1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Chol &gt;5 (%; SE)</td>
<td>95.5 (0.8)</td>
<td>96 (0.7)</td>
<td>84.2 (4.2)</td>
<td>88.5 (3.4)</td>
<td>87 (4.6)</td>
<td>85.1 (4.2)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI (mean; IQR)</td>
<td>28.5 (6.2)</td>
<td>28.6 (6.4)</td>
<td>28.8 (4.9)</td>
<td>28.6 (5.2)</td>
<td>31.6 (5)</td>
<td>31.5 (5.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese (%; SE)</td>
<td>33.4 (1.9)</td>
<td>33 (1.7)</td>
<td>34.2 (5.5)</td>
<td>33.3 (5.1)</td>
<td>68.5 (6.4)</td>
<td>59.5 (5.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current smoking (%; SE)</td>
<td>53.4 (2.2)</td>
<td>35.1 (1.9)</td>
<td>11.9 (5.1)</td>
<td>8.9 (4.3)</td>
<td>30.8 (9.2)</td>
<td>25 (7.3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes (%; SE)</td>
<td>12.8 (1.3)</td>
<td>10.6 (1.1)</td>
<td>57.9 (5.7)</td>
<td>46 (5.4)</td>
<td>63 (6.6)</td>
<td>55.4 (5.8)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Levels of the two risk scores changed across the spectrum of the six risk factors (Figure 7-2). JBS2 was higher than QRISK2 until approximately the age of 67, when patients’ have a higher mean QRISK2 score; the JBS2 score is higher across all levels of systolic blood pressure, total cholesterol, BMI and Townsend deprivation score. The scores diverge with higher systolic blood pressure, but converge with extreme levels of the later three. In the most affluent tenth of the population JBS2 is on average 1.22 times higher than QRISK2, but 1.15 times higher in the most deprived tenth (p<0.001; Wilcoxon rank-sum test).
Chapter 7: Ethnic group differences in cardiovascular risk estimates using JBS2 and QRISK2 risk scores

Figure 7-2: The running mean of JBS2 and QRISK2 across 6 cardiovascular risk factors

Sensitivity analysis

Compared with the sub-sample of the study population with complete data, the study population had different general characteristics; it was for example younger, had a higher proportion of white respondents and a higher prevalence of diabetes. However, my findings were the same in respondents with complete data recording.
7.4 Discussion

Main findings

QRISK2 mostly produces lower estimates of CVD risk than JBS2. This is the case in both sexes, although larger in men and across ethnic groups, with the exception of black women. In white and south Asian women despite a lower mean QRISK2 score than JBS2 there was a larger proportion of the population classified as at high risk using QRISK2. The largest difference between risk scores was seen in south Asian men, with 19.1 percent more of the population classified at high risk using JBS2. The excess was seen over all levels of risk and results in a large number of respondents identified as at high risk using JBS2 downgraded in risk status if using QRISK2. The two high risk groups have different profiles of CVD risk factors, with for example greater levels of obesity and diabetes if using QRISK2.

There are important ethnic differences in CVD incidence and outcomes in many countries. In the UK, patients of south Asian ethnic origin suffer from a higher burden of CVD than the white population. The JBS2 risk score attempts to account for the increased risk by using a multiplication factor of 1.4 in south Asian men, but there have been concerns over the accuracy of this estimate. QRISK2 also incorporates ethnic differences, but does so by directly including it as a CVD risk factor within the algorithm. There are also concerns over inaccuracies within QRISK2 due to limited numbers of ethnic minorities in the derivation data. My work identified a difference between risk scores, which was significantly larger amongst south Asian men.

Amongst men, there are fewer designated at high risk using the QRISK2 score, with a significantly larger difference in south Asian respondents. In practice, fewer patients would be exposed to the potential harms of screening, including unnecessary therapy (for example the prescription of statins) and increased anxiety over their risk status. Despite smaller numbers of patients at high risk, QRISK2 better predicts CVD events in the UK population. In men, a programme using QRISK2 is likely to be more cost effective in capturing CVD risk, especially in areas with large number of south Asian patients, and will have lower costs.
A larger numbers of women are designated at high risk using the QRISK2 score, making more eligible for interventions. Recent reductions in CVD in the western world have been driven by population level reductions in conventional CVD risk factors, including blood pressure and levels of smoking. Women traditionally see lower levels of CVD risk factors than men, with a greater reduction in recent years. Future CVD risk is likely to be driven by obesity and diabetes, two risk factors rising in prevalence in both sexes. Unlike traditional risk factors, obesity is not predominantly found in men. QRISK2 is more sensitive to levels of obesity, therefore may better predict risk in women. Its use for risk stratification might provide a prevention programme which is more equitable between the sexes.

*What is already known?*

Framingham based risk scores, including JBS2, over-estimate CVD risk in most western populations, although this differs in the most deprived. The more recent QRISK2 risk score and its predecessor QRISK produce lower estimates of CVD risk and more accurately predict CVD outcomes in the UK. Previous studies found, when comparing Framingham and QRISK risk scores, considerable reclassification of risk status. Collins and Altman found, using the QRISK score, 11.6 percent of men would be reclassified with 1.8 percent of Framingham low risk upgraded and 45.4 percent of high risk downgraded. In women 5.2 percent were reclassified. Using QRISK2, 41.1 percent of the high risk Framingham group were downgraded.

CVD risk scores have become embedded in clinical guidance internationally. A large number of CVD prediction tools exist; however there is still considerable work carried out around risk prediction. Outside recalibration, advances in new risk scores can be summarised into two main streams; the incorporation of novel risk factors and the prediction of lifetime risk (chapter 4.5). The introduction of novel risk factors has largely taken different paths internationally. The USA has focused on new biomarkers for vascular risk, with for example the Reynolds's risk score incorporating CRP. In the UK recent risk scores have included non-clinical risk factors, for example SEP and ethnicity.


**Strengths and limitations**

Data presented here are nationally representative, derived from a large national survey. Unlike previous comparisons between *Framingham* and the QRISK or QRISK2 risk scores, I used data which better represent BME patients. Further, unlike previous work, I focus analysis on the differences between ethnic groups. I use the most recently updated version of the QRISK2 algorithm.\(^{582}\) Although previous studies have assessed *Framingham* risk scores, few have applied the JBS2 ethnicity and family history multiplication factors, despite their frequent use in England.

My study did not compare or validate the predictive accuracy of the two risk scores. Using data from a national survey, I did not have access to follow-up data concerning CVD endpoints. In order to capture the BME populations, I used HSFEs from 2003 and 2004. The data are not the most recent and may not fully represent current populations risk profile; however I aimed to compare risk scores for which the data are adequate. The survey data contained missing data on a variety of CVD risk factors, however I used multiple imputation, a powerful method to deal with missing data,\(^{586}\) which will reduce bias. I finally did not have access to data concerning heart failure, rheumatoid arthritis or AF, all components of the QRISK2. They are not core components of the risk score and the algorithm allows them to be treated as absent if missing.\(^{83}\)

**Implications for practice**

Across ethnic groups, in all global settings there are established inequalities in CVD burden. If cardiovascular risk scores are to be central to prevention programmes, such as the NHS Health Check, it is vital they capture fully these ethnic differences. The recalibration of risk scores involves the adjustment of pre-existing risk scores to match mortality rates from a different setting or population. This is possible, but can lead to inaccuracy.\(^{387}\) Recalibration could also lead to the use of multiple risk scores simultaneously in clinical practice, adding further confusion to risk assessment. The JBS2 score in the UK attempts to adjust a *Framingham* risk score to capture the raised risk in south Asian men. Work presented here
suggests that the calibration factor creates a large significant difference in risk prediction compared with a risk score that directly models ethnic differences. In areas with large ethnic minority groups this will have large implications for screening costs, and may expose patients to unwarranted risk. JBS2 also fails to account for increased risk amongst south Asian women.\textsuperscript{36}

QRISK2 may under estimate risk in ethnic minorities,\textsuperscript{83} possibly due to under-representation in the derivation dataset. The QRISK2 algorithm will, however, undergo annual revisions. With overall improvements in the recording of medical data in the UK (Chapter 10), especially ethnicity recording (following the ethnicity recording directly enhanced service), the predictive accuracy is likely to improve. Despite the UK commencing on a universal CVD screening programme, recent evidence has suggested that a targeted approach might be worthwhile, for example using CVD risk scores to target patients.\textsuperscript{430 431} The predictive accuracy of risk scores will be vital in targeted programmes, and to avoid ethnic inequalities in health outcomes or costs, the risk scores must fully capture ethnic differences in risk.

Conclusions
Reducing ethnic group inequalities in CVD incidence and outcomes is a priority in many countries. With CVD risk scores becoming more systematically used in primary prevention, it is important that these accurately account for the ethnic differences in risk. Recalibration of risk scores is possible; however, data presented here suggests risk multiplication factors can create differences in risk prediction. Risk scores derived from large, national routine datasets allow ethnicity to be directly included in CVD risk scores, especially with improvements in ethnicity recording in EMRs.
Chapter 8; The comparison of cardiovascular risk scores using two methods of substituting missing risk factor data in patient medical records

8.1 Introduction

Despite the UK’s national commitment to a large, universal primary prevention programme for CVD, a targeted approach might be equally effective and more cost effective. Calls to prioritise treatment and prevention efforts are not new. Modelling has demonstrated that risk stratification using existing risk factor data from patient medical records is worthwhile. Risk stratification leads to the efficient case finding of high CVD risk. The accuracy of stratification improves with increased data recording; however the completeness of EMRs in the UK appears good for a number of risk factors (Chapter 10).

One method of risk stratification involves applying a CVD risk score to pre-existing risk factor data from medical records. A frequent problem is missing data; despite improvements in data recording, there is still incompleteness (Chapter 10). When data are incomplete default values must be entered to produce a risk score. I hypothesize that a more sensitive method used to replace missing data will lead to improved risk prediction.

I compare two methods of data imputation; firstly using risk factor estimates derived from national survey data. This method is computationally simple but may create inaccuracies if local profiles of risk factors differ from the national survey data and has no evidence base. It may lead to systematic errors in risk estimation in certain locales and affect the impact a prevention programme has upon health inequalities. A second option is to generate estimates from local data sources from which the target population is derived.

Multiple imputation when used to replace missing biomedical data can improve the accuracy of analysis. It is most effective when missing data are unrelated to observation characteristics (missing completely at random (MCAR)). Multiple imputation can however be
effective when the pattern of missing data are dependent on other patient characteristics (Missing at Random (MAR)); especially if related to variables collected in the dataset.\textsuperscript{587} Missing data patterns can be predicted by patient characteristics (Chapter 10). Although multiple imputation is more computationally difficult, it is likely to be sensitive to patient differences and maintain the local risk profile within the imputed data and has been effectively used in the analysis of large primary care data.\textsuperscript{83}

I have previously demonstrated the patient level implications of the ethnicity risk multiplication factor used in JBS2 (Chapter 7). I further compare differences in risk stratification using the two risk scores in an ethnically diverse population, concentrating on practice level work load implications.

I aim to compare risk stratification using the JBS2\textsuperscript{60} and QRISK2\textsuperscript{83} risk scores using two methods to substitute the missing data in EMRs; firstly using default values from nation survey data and secondly multiple imputation.

\textbf{8.2 Methods}

\textit{Data source}

I obtained patient level data from the EMRs in 70 of the 85 general practices in Ealing. Data were extracted before the NHS Health Check, using the Oberoi primary prevention software (Chapter 2.2.1). I used data from the baseline of the programme, between December 2008 and December 2009. Briefly, data consist of physical characteristics and anthropometric data relating to cardiovascular risk, for patients registered in general practice, aged 35 to 74 years without diagnosed coronary heart disease or stroke; Table 8-1 lists variables extracted. I did not have data recorded for Rheumatoid Arthritis. Two deprivation scores, the 2007 IMD and Townsend Score were applied based on the patient's postcode of residence. The data represent a patients' latest record at the time of extraction. I discounted data older than 15 years, using a liberal cut-off because the analysis compared the risk scores, not patients;
therefore complete data were more important than timely, however, the majority of data records were from the previous five years.

### Table 8-1: the variables included in QRISK2 and JBS2 score, and levels of missing data

<table>
<thead>
<tr>
<th>Variable</th>
<th>QRISK2</th>
<th>JBS2</th>
<th>% Missing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age‡</td>
<td>x</td>
<td>x</td>
<td>-</td>
</tr>
<tr>
<td>Sex‡</td>
<td>x</td>
<td>x</td>
<td>-</td>
</tr>
<tr>
<td>Ethnicity‡</td>
<td>x</td>
<td>†</td>
<td>34.3</td>
</tr>
<tr>
<td>Deprivation (Townsend Score)‡</td>
<td>x</td>
<td>†</td>
<td>11.9</td>
</tr>
<tr>
<td>Smoking</td>
<td>x</td>
<td>x</td>
<td>3.2</td>
</tr>
<tr>
<td>Hypertension‡</td>
<td>x</td>
<td>†</td>
<td>-</td>
</tr>
<tr>
<td>Diabetes‡</td>
<td>x</td>
<td>†</td>
<td>-</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>x</td>
<td>x</td>
<td>11.1</td>
</tr>
<tr>
<td>Cholesterol/ HDL</td>
<td>x</td>
<td>x</td>
<td>44.6</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>x</td>
<td></td>
<td>15.7</td>
</tr>
<tr>
<td>Family History of CVD‡</td>
<td>x</td>
<td>†</td>
<td>28.6</td>
</tr>
<tr>
<td>Heart Failure</td>
<td>x</td>
<td></td>
<td>18.5</td>
</tr>
<tr>
<td>Chronic Kidney Disease‡</td>
<td>x</td>
<td></td>
<td>18.5</td>
</tr>
<tr>
<td>Rheumatoid Arthritis</td>
<td>x</td>
<td>ALL</td>
<td></td>
</tr>
<tr>
<td>Atrial Fibrillation</td>
<td>x</td>
<td></td>
<td>18.5</td>
</tr>
</tbody>
</table>

† Not included within the algorithm, but adjustment factors are added
*Townsend Score for QRISK2/ IMD2007 for other analysis
‡ Variables used in the multiple imputation of missing data

The data contained missing values for a number of variables (Table 8-1). For both methods of imputation, I entered patients with missing ethnicity records as a subgroup in the analysis; for deprivation I replaced the missing Townsend score used in the QRISK2 algorithm with the PCT median. For Rheumatoid Arthritis, I assumed the condition to be absent if missing, in line with guidance for the QRISK2 score. Similarly, CKD and a family history of CVD were assumed absent with missing data. I used two methods to estimate values for missing blood pressure, total cholesterol, HDL, BMI and smoking status.

Firstly I used multiple imputation, using the `uvvis` commands from Stata and generated 10 imputed datasets (m). I built multi-level linear imputation models (logistic regression for smoking status) to impute missing values by backwards stepwise selection, using AIC to assess the best fitting model. Level one of the model was the patient and level two the general practice. Variables entered as candidates for the models can be seen in (Table 8-1),
with the addition of interaction terms between sex and age, and sex and ethnicity. The
variables selected for each model are shown in the appendix (Table viii-2). I calculated the
fraction of missing information (γ) for each imputed variable. For each variable imputed, 
\( m=10 \) gave adequate power of imputation, i.e. less than a 5% deviation from the power of 
using \( m=100 \). Any values in the imputed dataset outside the range valid for the QRISK2 
algorithm were dropped.

For the second method of substitution, I took the mean risk factor values from the 2008 HSfE 
in each year of age and sex group. For patients with missing risk factor data I entered the 
matching mean value from the HSfE based on the age and sex of the patient and assumed 
patients with missing smoking data to be non-smokers.

Analysis

I compared CVD risk factors between ethnic groups using t-tests (Mann-Whitney U test for 
IMD), using the following five categories (missing, white, south Asian, black and other). I 
present age and deprivation breakdowns, plus age-standardised risk factor summaries for 
both men and women. I used the direct method of standardisation, using eight equal age 
groups and the complete dataset as the standard. I applied the QRISK2\(^{83} \) (after the 2010 
update\(^{582} \)) and JBS2\(^{60} \) risk scores to each imputed copy of the dataset, calculating mean 
score across imputations for each patient to give a single score for each. I summarised 
levels of the two risk scores and differences overall, in each sex/ ethnicity group, and the 
proportion designated as at high risk (≥20%) - again using direct age standardisation.

I then compared methods of data imputation. For the two risk scores, I calculated the mean 
risk score and percentage of the population designated as high risk using the multiple 
imputation and health survey data. I assessed the agreement of high risk status between 
imputation methods using \textit{Cohen's Kappa}, quoting 95% confidence intervals. \(^{589} \) Outside of 
the population characteristics, I present the results for those patients with incomplete 
(n=63,607) data recording.
Table 8-2; variables included in QRISK2 and JBS2 score, and levels of missing data

<table>
<thead>
<tr>
<th></th>
<th>Missing</th>
<th>White</th>
<th>Ethnicity</th>
<th>South Asian</th>
<th>Black</th>
<th>Other</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No (%)</td>
<td>22108 (17.3)</td>
<td>15855 (12.4)</td>
<td>13733 (10.8)</td>
<td>4586 (3.6)</td>
<td>6533 (5.1)</td>
<td>62815 (49.2)</td>
<td></td>
</tr>
<tr>
<td>Median IMD</td>
<td>23.2</td>
<td>20.6</td>
<td>26.2</td>
<td>29.5</td>
<td>21.4</td>
<td>23.7</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>49.1</td>
<td>50.7</td>
<td>49.3</td>
<td>48.9</td>
<td>50.4</td>
<td>49.7</td>
<td></td>
</tr>
<tr>
<td>Systolic BP</td>
<td>131.5 (131.4-131.7)</td>
<td>131.0 (130.8-131.2)</td>
<td>130.5 (130.3-130.8)</td>
<td>131.4 (131-131.9)</td>
<td>130.3 (130.0-130.6)</td>
<td>131 (130.9-131.1)</td>
<td></td>
</tr>
<tr>
<td>BP&gt; 140/90</td>
<td>27.6 (27-28.2)</td>
<td>29.2 (28.5-29.9)</td>
<td>30.7 (30-31.5)</td>
<td>31.1 (29.8-32.5)</td>
<td>27.1 (26.1-28.2)</td>
<td>28.9 (28.5-29.2)</td>
<td></td>
</tr>
<tr>
<td>Chol</td>
<td>5.27 (5.26-5.28)</td>
<td>5.22 (5.21-5.23)</td>
<td>5.01 (4.99-5.02)</td>
<td>5.05 (5.03-5.07)</td>
<td>5.11 (5.09-5.13)</td>
<td>5.16 (5.16-5.17)</td>
<td></td>
</tr>
<tr>
<td>Chol &gt; 5</td>
<td>71.5 (70.9-72.1)</td>
<td>68.3 (67.6-69)</td>
<td>55.6 (54.8-56.4)</td>
<td>58.3 (56.8-59.7)</td>
<td>62.2 (61.1-63.3)</td>
<td>65.2 (64.8-65.6)</td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>26.5 (26.5-26.6)</td>
<td>27.1 (27.1-27.1)</td>
<td>26.7 (26.6-26.7)</td>
<td>26.8 (26.7-26.8)</td>
<td>27.3 (27.3-27.4)</td>
<td>26.8 (26.8-26.8)</td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td>12.8 (12.3-13.2)</td>
<td>20.8 (20.2-21.4)</td>
<td>17.5 (16.9-18.2)</td>
<td>19.8 (18.7-21)</td>
<td>22.8 (21.8-23.8)</td>
<td>17.4 (17.1-17.7)</td>
<td></td>
</tr>
<tr>
<td>Smoking (%)</td>
<td>20 (19.4-20.5)</td>
<td>27.4 (26.7-28.1)</td>
<td>16.5 (15.9-17.1)</td>
<td>24.8 (23.5-26.1)</td>
<td>26.5 (25.4-27.6)</td>
<td>22 (21.7-22.4)</td>
<td></td>
</tr>
<tr>
<td>Diabetes (%)</td>
<td>3.2 (3-3.5)</td>
<td>6.6 (6.3-7)</td>
<td>19.7 (19.1-20.4)</td>
<td>15.2 (14.1-16.3)</td>
<td>11.3 (10.6-12.1)</td>
<td>9.4 (9.2-9.7)</td>
<td></td>
</tr>
</tbody>
</table>

Male

<table>
<thead>
<tr>
<th></th>
<th>No (%)</th>
<th>Median IMD</th>
<th>Age</th>
<th>Systolic BP</th>
<th>BP&gt; 140/90</th>
<th>Total Chol</th>
<th>Chol &gt; 5</th>
<th>BMI</th>
<th>Obese</th>
<th>Smoking (%)</th>
<th>Diabetes (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>16943 (13.3)</td>
<td>20.6</td>
<td>50.6</td>
<td>124.7 (124.4-125)</td>
<td>21.9 (21.3-22.6)</td>
<td>5.00 (4.98-5.01)</td>
<td>50.5 (49.7-51.3)</td>
<td>26.4 (26.3-26.4)</td>
<td>21.1 (20.5-21.7)</td>
<td>21.2 (20.6-21.9)</td>
<td>4.2 (3.9-4.5)</td>
</tr>
<tr>
<td>South Asian</td>
<td>13331 (10.4)</td>
<td>26.0</td>
<td>50.6</td>
<td>124.6 (125.1)</td>
<td>21.9 (21.3-22.6)</td>
<td>5.00 (4.98-5.01)</td>
<td>50.5 (49.7-51.3)</td>
<td>26.4 (26.3-26.4)</td>
<td>21.1 (20.5-21.7)</td>
<td>21.2 (20.6-21.9)</td>
<td>4.2 (3.9-4.5)</td>
</tr>
<tr>
<td>Black</td>
<td>13507 (10.6)</td>
<td>26.0</td>
<td>50.6</td>
<td>124.6 (125.1)</td>
<td>21.9 (21.3-22.6)</td>
<td>5.00 (4.98-5.01)</td>
<td>50.5 (49.7-51.3)</td>
<td>26.4 (26.3-26.4)</td>
<td>21.1 (20.5-21.7)</td>
<td>21.2 (20.6-21.9)</td>
<td>4.2 (3.9-4.5)</td>
</tr>
<tr>
<td>Other</td>
<td>16943 (13.3)</td>
<td>20.6</td>
<td>50.6</td>
<td>124.7 (124.4-125)</td>
<td>21.9 (21.3-22.6)</td>
<td>5.00 (4.98-5.01)</td>
<td>50.5 (49.7-51.3)</td>
<td>26.4 (26.3-26.4)</td>
<td>21.1 (20.5-21.7)</td>
<td>21.2 (20.6-21.9)</td>
<td>4.2 (3.9-4.5)</td>
</tr>
</tbody>
</table>

Female

<table>
<thead>
<tr>
<th></th>
<th>No (%)</th>
<th>Median IMD</th>
<th>Age</th>
<th>Systolic BP</th>
<th>BP&gt; 140/90</th>
<th>Total Chol</th>
<th>Chol &gt; 5</th>
<th>BMI</th>
<th>Obese</th>
<th>Smoking (%)</th>
<th>Diabetes (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>12.7 (12.4-125)</td>
<td>19.9 (19.3-20.5)</td>
<td>21.9 (21.3-22.6)</td>
<td>27.7 (26.6-28.9)</td>
<td>20.4 (19.5-21.3)</td>
<td>20.7 (20.4-21)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>South Asian</td>
<td>124.9 (124.4-125.1)</td>
<td>19.2 (18.7-19.7)</td>
<td>21.9 (21.3-22.6)</td>
<td>27.7 (26.6-28.9)</td>
<td>20.4 (19.5-21.3)</td>
<td>20.7 (20.4-21)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>124.9 (124.4-125.1)</td>
<td>19.2 (18.7-19.7)</td>
<td>21.9 (21.3-22.6)</td>
<td>27.7 (26.6-28.9)</td>
<td>20.4 (19.5-21.3)</td>
<td>20.7 (20.4-21)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>124.9 (124.4-125.1)</td>
<td>19.2 (18.7-19.7)</td>
<td>21.9 (21.3-22.6)</td>
<td>27.7 (26.6-28.9)</td>
<td>20.4 (19.5-21.3)</td>
<td>20.7 (20.4-21)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Chol. = cholesterol
Finally, I examined the effect at general practice level of changing the risk score used to risk stratify patients. I calculated differences in mean risk scores and the median difference in number and proportion of a practice at high risk between the two risk scores. I compared these between practices dependent on the proportion of south Asian patients in the 35-74 population with recorded ethnicity. All analyses were carried out using Stata 11.1, quoting confidence intervals at a level of 95%. I obtained ethical approval for the use of anonymised patient level data from the London research ethics committee.

### 8.3 Results

Table 8-2 shows characteristics of the study population; the white population was more affluent than other ethnicities (Mann-Whitney U p<0.001 compared with all ethnic groups), suffered higher lipid levels and higher smoking prevalence in women (t test p<0.001 compared with all ethnic groups). The south Asian and black population have higher levels of diabetes (t test p<0.001 compared with all ethnic groups) and in the female population, black patients have the highest obesity levels (t test p<0.001 compared with all ethnic groups).

<table>
<thead>
<tr>
<th></th>
<th>Missing</th>
<th>White</th>
<th>South Asian</th>
<th>Black</th>
<th>Other</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>JBS2</strong></td>
<td>6.6</td>
<td>7.6</td>
<td>8.1</td>
<td>7.6</td>
<td>7.7</td>
<td>7.4</td>
</tr>
<tr>
<td><strong>QRISK</strong></td>
<td>5.4</td>
<td>6.4</td>
<td>7.3</td>
<td>6.4</td>
<td>8.1</td>
<td>6.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Difference (95% CI)</th>
<th>1.25 (1.22 to 1.29)</th>
<th>1.21 (1.17 to 1.27)</th>
<th>0.81 (0.76 to 0.87)</th>
<th>1.21 (1.12 to 1.29)</th>
<th>-0.43 (-0.52 to -0.33)</th>
<th>0.95 (0.92 to 0.97)</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th><strong>Proportion high risk</strong></th>
<th>JBS2</th>
<th>QRISK</th>
<th>South Asian</th>
<th>Black</th>
<th>Other</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Female</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>JBS2</strong></td>
<td>4.6</td>
<td>7.4</td>
<td>9.6</td>
<td>8.2</td>
<td>7.9</td>
<td>7.1</td>
</tr>
<tr>
<td><strong>QRISK</strong></td>
<td>3.8</td>
<td>6.7</td>
<td>9.8</td>
<td>6.9</td>
<td>11.7</td>
<td>7.1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Difference (95% CI)</th>
<th>0.84 (0.59 to 1.1)</th>
<th>0.65 (0.34 to 0.96)</th>
<th>-0.2 (-0.56 to 0.17)</th>
<th>1.38 (0.78 to 1.97)</th>
<th>-3.85 (-4.4 to -3.29)</th>
<th>0.02 (-0.14 to 0.17)</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th><strong>Mean risk score</strong></th>
<th>JBS2</th>
<th>QRISK</th>
<th>South Asian</th>
<th>Black</th>
<th>Other</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Male</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>JBS2</strong></td>
<td>13.1</td>
<td>14.2</td>
<td>20.4</td>
<td>13.8</td>
<td>14.4</td>
<td>15.1</td>
</tr>
<tr>
<td><strong>QRISK</strong></td>
<td>8.9</td>
<td>10.5</td>
<td>11.7</td>
<td>9.5</td>
<td>13.5</td>
<td>10.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Difference (95% CI)</th>
<th>4.11 (4.05 to 4.17)</th>
<th>3.66 (3.59 to 3.74)</th>
<th>8.71 (8.59 to 8.82)</th>
<th>4.35 (4.21 to 4.50)</th>
<th>0.93 (0.78 to 1.08)</th>
<th>4.63 (4.58 to 4.67)</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th><strong>Proportion high risk</strong></th>
<th>JBS2</th>
<th>QRISK</th>
<th>South Asian</th>
<th>Black</th>
<th>Other</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Male</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>JBS2</strong></td>
<td>21.0</td>
<td>24.4</td>
<td>40.6</td>
<td>23.9</td>
<td>25.6</td>
<td>26.7</td>
</tr>
<tr>
<td><strong>QRISK</strong></td>
<td>12.4</td>
<td>17.1</td>
<td>21.4</td>
<td>15.2</td>
<td>24.7</td>
<td>17.2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Difference (95% CI)</th>
<th>8.66 (8.23 to 9.08)</th>
<th>7.32 (6.83 to 7.81)</th>
<th>19.2 (18.5 to 19.8)</th>
<th>8.68 (7.67 to 9.68)</th>
<th>0.91 (0.12 to 1.7)</th>
<th>9.6 (9.3 to 9.8)</th>
</tr>
</thead>
</table>
In the whole population (n=127,724), using the multiple imputation data, the mean QRISK2 score (8.4 [95% CI 8.4-8.4]) was significantly lower than JBS2 (11.1 [11.1-11.2]), with fewer patients designated as at high risk (n=15,258; 11.9% [11.8-12.1] and n=21,377; 16.7 [16.5%-16.9] respectively (Table 8-3)). The QRISK score was lower in both sexes, and over most ethnic groups with the largest difference in men of south Asian ethnic origin. Patients not of white, south Asian or black ethnicity had a higher QRISK score than JBS2 in women.

In the population with missing risk factor data (n=63,607), the multiple imputation method produces lower estimates of risk scores and lower proportions of the population at high risk; differences are, however, relatively small. Using multiple imputation (Table 8-4), the QRISK2 risk score was 0.1 percent lower, with 0.6 percent less of the population at high risk, (JBS2 risk score is shown in appendix, Table viii-3).

<table>
<thead>
<tr>
<th>Risk Score</th>
<th>Missing White</th>
<th>South Asian</th>
<th>Black</th>
<th>Other</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Male</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HSE</td>
<td>8.0 (7.9-8.0)</td>
<td>8.7 (8.6-8.8)</td>
<td>9.4 (9.1-9.6)</td>
<td>7.8 (7.5-8)</td>
<td>11.2 (11.0-11.5)</td>
</tr>
<tr>
<td>IMP</td>
<td>7.9 (7.9-8.0)</td>
<td>8.4 (8.3-8.5)</td>
<td>9.0 (8.8-9.2)</td>
<td>7.4 (7.1-7.6)</td>
<td>11.1 (10.8-11.3)</td>
</tr>
<tr>
<td><strong>Female</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HSE</td>
<td>4.8 (4.8-4.9)</td>
<td>5.3 (5.2-5.3)</td>
<td>5.8 (5.6-6.0)</td>
<td>5.0 (4.8-5.2)</td>
<td>6.7 (6.5-6.9)</td>
</tr>
<tr>
<td>IMP</td>
<td>4.7 (4.7-4.8)</td>
<td>5.1 (5.0-5.2)</td>
<td>5.6 (5.5-5.8)</td>
<td>4.8 (4.7-5.0)</td>
<td>6.5 (6.3-6.7)</td>
</tr>
</tbody>
</table>

There were differences in agreement between methods of imputation by sex and ethnic group. White men and women, south Asian men and black women had greater estimates of

Table 8-4; Age standardised QRISK2 estimates using multiple imputation and HSE data to replace missing data in patients with missing data

HSE: Missing data replaced with Health Survey default values
IMP: Missing data replaced using multiple imputation
Kappa- Cohen's Kappa
the high risk population using the HSfE data, but these differences were small. In designating patients to be at high risk, the two methods have strong agreement with high \textit{kappa} values across all sex and ethnic groups. In black men, there is a significantly higher agreement between methods to other ethnic groups.

There were 70 general practices in the study; with a mean of 1,824 patients aged 35-74 without diagnosed CVD (median=1,609), ranging from 508 to 5,429. Using JBS2, there was a median of 265 patients at high risk per practice (range = 107-754; IQR= 188), 183 (range = 71-507; IQR=134) using QRISK2. The choice of risk score impacts practices to different degrees. The difference in proportion of the population designated as at high risk ranges from 0.3- 10.3\% less using QRISK2, a range of 10-317 patients.

The practice differences between the two scores were associated with the ethnic make-up of practices (Table 8-5;Figure 8-1); in practices with less than 25 percent of the population of south Asian origin, the mean difference in risk scores was 2.43 (2.29-2.56) with 3.86 percent (3.46-4.26) fewer patients designated at high risk. In practices with more than 75\% of the
population of south Asian ethnicity, the QRISK2 score was 3.79 (3.38-4.20) lower, with 7.14% (5.60-8.69) fewer patients at high risk.

<table>
<thead>
<tr>
<th>Proportion of practice south Asian</th>
<th>Total</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>&lt;25%</td>
<td>25-75%</td>
<td>&gt;75%</td>
</tr>
<tr>
<td>Mean JBS2</td>
<td></td>
<td>8.2 (7.8-8.6)</td>
<td>9.2 (8.7-9.7)</td>
<td>9.5 (8.8-10.2)</td>
</tr>
<tr>
<td>Mean QRISK</td>
<td></td>
<td>10.6 (10.2-11)</td>
<td>12.4 (11.8-12.9)</td>
<td>13.3 (12.4-14.1)</td>
</tr>
<tr>
<td>Difference</td>
<td></td>
<td>2.4 (2.3-2.6)</td>
<td>3.2 (2.9-3.4)</td>
<td>3.8 (3.4-4.2)</td>
</tr>
<tr>
<td>Median diff in prop'n high risk</td>
<td></td>
<td>3.9 (0.2-6.6)</td>
<td>6.1 (2.6-10.9)</td>
<td>7.7 (2.0-10.3)</td>
</tr>
<tr>
<td>Median diff in no high risk</td>
<td></td>
<td>69 (10-158)</td>
<td>74 (31-157)</td>
<td>120 (46-317)</td>
</tr>
</tbody>
</table>

8.4 Discussion

Main findings

I compared cardiovascular risk scores generated for patients using two methods to substitute missing factor data. One approach was methodologically and computationally simple using national health survey data, whilst the second more complex using multiple imputation. Using multiple imputation the risk score estimates were marginally lower, but often not significantly so and there was a small but significantly lower prevalence of high risk status. Ethnic differences were seen between methods, although these were again only small. Multiple imputation is regarded as an accurate method of dealing with missing data and is robust to large amounts of missing data. It is, however, a more time consuming and complicated process than the use of default values from survey data, and less transparent to non-statistically trained people. The simple method produced different risk scores, but these were small and unlikely to be clinically significant.

The JBS2 and QRISK2 score account for ethnic differences in CVD using different methods, which produces ethnic variation in risk prediction (Chapter 7). Once again, using data from EMRs, the largest difference between risk scores is in south Asian men. The raised risk estimates caused by the multiplication factor in south Asian men have consequences for general practice workload. Compared with using QRISK2, JBS2 creates a disproportionately
large work load in general practices with large south Asian populations. If the multiplication factor is inaccurate, over-estimating the CVD risk in south Asian men, then this difference in work load and cost may be unjustified.

What is already known?
Targeted screening for CVD is likely to be an effective and cost effective approach. Interrogating general practice data, Marshall et al. found a large proportion of patients eligible for primary prevention therapy lie in those with the highest estimated risk. Recent data suggest those estimated to be at high risk to possess a high prevalence of CVD risk factors. Modelling further suggests that targeted screening is more cost effective than a universal approach. CVD risk factor recording is not complete in EMRs (Chapter 10), but partial data can give accurate risk prediction. In order to produce a CVD risk score, default values must be generated to replace missing data, with multiple imputation a sensitive and effective method.

Strengths and limitations
My data were extracted from the EMRs of a large number of patients. They cover an ethnically diverse and socio-economically deprived population, different from those previously used to compare CVD risk scores in the UK. They cover a large proportion of registered patients in one English PCT. I used the recently updated version of the QRISK2 algorithm, and unlike many previous studies used the modified Framingham (JBS2) risk score recommended in UK national clinical guidance. My data are timely and accurately represent the level of cardiovascular risk in a deprived, ethnically diverse population, although may not represent levels of risk in other settings.

I cannot compare the predictive accuracy of the risk score as I was unable to link data to CVD outcomes. The QRISK2 score, used as a comparator of risk prediction, may underestimate risk, especially in ethnic minority groups. This is likely to be due to the small ethnic minority populations in the derivation dataset. QRISK2 and its predecessor QRISK do however predict risk more accurately in the UK than Framingham risk scores. Aside from
the missing data imputed in the study, my dataset contained no data on rheumatoid arthritis and had missing data for AF, CKD and heart failure, all of which are variables in the QRISK2 score. These are not core components of the algorithm and the risk score allows them to be treated as absent if missing.\textsuperscript{83} There were further missing data for the Townsend deprivation score which were replaced with median score from the PCT. Neither method of data imputation will fully account for patient level differences; it is likely those with missing data will differ from remainder of the population.

\textit{Implications for practice}

In the UK, there is currently a large national expenditure on a primary prevention programme for CVD; with Department of Health estimates of programme spending of between £180 and £240 million per year.\textsuperscript{139} Department of Health economic modelling, however, only compared universal approaches to screening, without looking at targeted approaches.\textsuperscript{139} Since the NHS Health Check roll out there has been evidence that a targeted approach to screening might be more cost effective than the universal approach pursued by the Department of Health.\textsuperscript{430, 431} The call for targeted screening is not a new one, with growing evidence to support this approach (Chapter 4.4).

If using existing medical record data for targeting screening, missing data are inevitable. My analysis suggests a simple method of substituting missing data can be effective in producing a CVD risk score. There is currently a need to reduce spending across the NHS. A targeted approach to CVD prevention will be less costly in total and may be more cost effective than a universal approach and I have demonstrated that it does not have to use complex methods to overcome missing data.
Chapter 9; Prevalence of CVD risk amongst the population eligible for NHS Health Check in England; a modelling study using QRISK2 and JBS2 scores

9.1 Introduction

Before the roll out of the NHS Health Check, the Department of Health carried out economic modelling for the programme.\textsuperscript{139} This presented the costs and cost-effectiveness of screening the total eligible population and offering risk reduction interventions to everyone found with a CVD risk factor, whether at high risk or not. A universal approach was the only strategy modelled, with no comparison made to a targeted high risk strategy. As a result, the numbers and costs of screening and treating patients at high risk were not explicitly considered. Recent work suggests an approach targeted on those likely to be at high risk may offer better value (Chapter 4.4).\textsuperscript{430}

There are no national estimates of the size of the population at high risk of CVD in England, only estimates from comparatively small populations,\textsuperscript{83} or estimates of the prevalence of risk factors within this high risk population. The baseline prevalence of high CVD risk and subsequent local changes should be a key indicator of the performance of the Programme. The expectation should be that, with good risk presentation,\textsuperscript{290} intensive intervention and annual review, CVD risk should fall faster in the high risk sub-population. Although NHS Health Check started in April 2009 and general practices will be capturing data electronically, there is as yet no national coverage data.

Using HSfE data, I aimed to model the number of patients with greater than or equal to 20 percent ten year risk of developing CVD in every general practice in England, using QRISK2 and JBS2 risk scores. I compared prevalence of high risk status and the number of patients with CVD risk factors and costs of the programme in the high risk group.
9.2 Methods

Data sources

I appended data from the 2003 to 2006 HSfE inclusive (see chapter 7); of the 71,717 informants interviewed, 25,319 were aged 40 to 74 years. I excluded patients with diagnosed CVD, diabetes and hypertension (n=5,186) i.e. those not eligible for the Health Check. In addition to the standard HSfE data, I obtained 2001 Townsend deprivation scores and IMD 2007 scores based on respondent’s postcode of residence.

Table 9-1; the variables included in QRISK2 and JBS2 score, and levels of missing data

<table>
<thead>
<tr>
<th>Variable</th>
<th>QRISK2</th>
<th>JBS2</th>
<th>% Missing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age‡</td>
<td>x</td>
<td>x</td>
<td>0.0%</td>
</tr>
<tr>
<td>Sex‡</td>
<td>x</td>
<td>x</td>
<td>0.0%</td>
</tr>
<tr>
<td>Ethnicity‡</td>
<td>x</td>
<td>†</td>
<td>0.0%</td>
</tr>
<tr>
<td>Deprivation (Townsend Score) ‡*</td>
<td>x</td>
<td></td>
<td>0.0%</td>
</tr>
<tr>
<td>Smoking‡</td>
<td>x</td>
<td>x</td>
<td>0.0%</td>
</tr>
<tr>
<td>Hypertension</td>
<td>x</td>
<td></td>
<td>0.0%</td>
</tr>
<tr>
<td>Diabetes</td>
<td>x</td>
<td>x</td>
<td>0.0%</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>x</td>
<td>x</td>
<td>38.0%</td>
</tr>
<tr>
<td>Cholesterol/ HDL</td>
<td>x</td>
<td>x</td>
<td>60.5%</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>x</td>
<td></td>
<td>11.5%</td>
</tr>
<tr>
<td>Family History of CVD</td>
<td>x</td>
<td>†</td>
<td>29.7%</td>
</tr>
<tr>
<td>Heart Failure</td>
<td>x</td>
<td></td>
<td>ALL</td>
</tr>
<tr>
<td>Chronic Kidney Disease</td>
<td>x</td>
<td></td>
<td>ALL</td>
</tr>
<tr>
<td>Rheumatoid Arthritis</td>
<td>x</td>
<td></td>
<td>ALL</td>
</tr>
<tr>
<td>Atrial Fibrillation</td>
<td>x</td>
<td></td>
<td>ALL</td>
</tr>
</tbody>
</table>

† Not included within the algorithm, but adjustment factors are added
*Townsend Score for QRISK2/ IMD2007 for other analysis
‡ Variables used in the multiple imputation of missing data

I applied the QRISK2 (after the 2010 update) and JBS2 risk scores to each informant’s data. Both models use age, sex, ethnicity, smoking status, family history of CVD, systolic blood pressure and lipid ratios (total cholesterol and high density lipoprotein) (Table 9-1). The HSfE contained complete data with the following exceptions (Table 9-1). I assumed no family history of CVD in the 5,986 (29.7%) with missing data. Data were missing for other required risk factors: 7,641 (38.0%) had blood pressure missing, 2,322 (11.5%) BMI and 12,187 (60.5%) total cholesterol to high density lipoprotein (lipid) ratios (Table 9-1). I used
multiple imputation to estimate missing values, building linear regression models using backward stepwise selection to predict the four outcomes within the complete data. Multiple imputation is most applicable when data are MCAR, however the methodology is robust to MAR data; based upon the HSfE methodology I had no reason to assume a strong violation of MAR. I included variables with complete recording as model candidates. For each variable the final imputation model contained age, sex, ethnicity, smoking status, deprivation and age/sex interaction term. Using the \texttt{uvis} command in Stata, I produced ten imputed copies of the dataset ($m$). I calculated the fraction of missing information ($\gamma$) for each imputed variable, using this to assess whether $m$ was sufficiently large. I determined that $\gamma = 0.61$, $0.45$ and $0.25$ for cholesterol, blood pressure and BMI respectively give an acceptable power of imputation with $m=10$ (a 5%, 4% and 3% loss of power respectively compared with $m=100$).

In addition to JBS2 variables, the QRISK2 algorithm requires data on BMI, Townsend deprivation score, CKD, rheumatoid arthritis, heart failure and AF. As there are no data in the HSfE for CKD, heart failure, AF or rheumatoid arthritis, and the prevalence is low, these were assumed negative. Both JBS2 and QRISK2 require ethnicity data. The JBS2 risk score differs from the Framingham combined CVD score (Anderson) by employing a multiplication factor of 1.4 for men with a south Asian background and 1.5 if a first-degree relative has a history of premature CVD.

\textit{High risk models}

I partitioned the population based on age (40-49, 50-54, 55-64, 65-74 years); sex; ethnicity (White, south Asian (Asian or Asian British in the HSfE), black (Black or Black British in the HSfE), Chinese or Other) and deprivation (national thirds of the IMD), generating 96 data cells. I applied the risk algorithms to each imputed dataset and calculated a mean risk score between imputations for each observation. I calculated proportions at high risk ($\geq 20\%$) in each cell; combining any cell with fewer than 10 entrants with the adjacent (the next highest age group).
I obtained age and sex breakdowns of every general practice in England and subtracted the numbers of patients on the CHD, Stroke/ TIA, diabetes and hypertension registers using estimates from QOF data. Using HSfE data, I estimated an age distribution of the population with each condition, and hence the proportion of the total within the 40 to 74 age group. I adjusted the prevalence for patients with multiple conditions, deriving estimates of co-morbidity from the HSfE data; 40% of diabetes patients appear on a CVD register and 37% of the hypertension register have CVD or diabetes. I obtained estimates of the ethnic breakdown of each practice from the Care Quality Commission (CQC), which were derived by assuming the proportions admitted to hospital reflected the ethnic composition of LSOA resident populations. I applied the ethnicity proportions to the population data assuming the ethnic breakdown was uniform over age/ sex groups. I used IMD scores for each practice, derived from the postcode of residence of the registered population, and assigned each practice to a national third. Each practice was broken down into the same 96 cells as the risk proportions and I applied the risk proportions, giving the number of patients with a ≥20% score for JBS2 and QRISK2; these were summed to give PCT level estimates. I present median values with skewed data. I used Wilcoxon sign rank tests to assess the difference in national ranking of practices and PCTs by risk prevalence.

Managing CVD risk factors in high risk group

I modelled the distribution of individual CVD risk factors in patients at high risk. These were BMI greater than 25 kg/m²; undiagnosed hypertension (assuming 50% of patients with a raised blood pressure proceed to be diagnosed with hypertension); physical inactivity (fewer than five 30 minute bouts of exercise per week); unrecorded impaired fasting glycaemia (IFG) (fasting plasma glucose between 6.1 and 6.9 mmol/l), smoking and the prevalence of undiagnosed diabetes (fasting plasma glucose ≥7mmol/l). Taking the high risk population, I found the proportion in the HSfE data of patients in each age/ sex category with risk factors, and transposed the proportions onto the PCT high risk estimates.
Costs

I estimated the costs of the basic NHS Health Check, initial interventions and referrals (related to the risk factors above) in the high risk group. I assume one Health Check encounter per patient. Most subsequent assumptions were those used in the Department of Health impact assessment\(^{139}\) (appendix; Table viii-4 and Table viii-5). I assumed 75% attendance at the initial health check.\(^{139}\) However, one of the key Department of Health assumptions is that 50% of Health Check activity already happens as part of routine clinical practice. Once the programme is underway, GPs are likely to switch preventative CVD care to within the Health Check activity; the NHS will be funding the majority of CVD prevention through the programme, hence I modelled the entire costs of activity. I estimated the costs of the basic Health Check; including time, diagnostic tests and resultant referrals, but not additional time for the diagnoses. All data analyses were carried out using Microsoft Excel and Stata 11.1SE.

9.3 Results

Prevalence of high CVD risk

I estimate approximately 2,012,000 people in England aged 40 to 74 years have a QRISK2 score $> 20\%$, out of 19,388,700 eligible for a Health Check (10.4\%) (Table 9-2). The median per PCT was 10,838 (range= 3,799- 42,258). As a percentage of the eligible population, the PCT median was 10.7 (range = 6.4- 13.7). There was a median of 206 patients at high risk per practice, ranging from zero to 1,693. Using the JBS2 algorithm there was a larger number classified as at high risk; approximately 4,268,000 aged 40-74 years (22.0\%), with a median of 23,136 (range=8,796-96,342) per PCT.
### Table 9-2: PCT and practice level estimates of the numbers and proportion of high-risk patients

<table>
<thead>
<tr>
<th>Risk Score</th>
<th>Total</th>
<th>Median</th>
<th>Min - Max</th>
<th>IQR</th>
</tr>
</thead>
<tbody>
<tr>
<td>QRISK2</td>
<td>Number</td>
<td>2,012,151</td>
<td>10,838</td>
<td>3,799-42,258</td>
</tr>
<tr>
<td></td>
<td>Prevalence</td>
<td>10.4%</td>
<td>10.7%</td>
<td>6.4-13.7%</td>
</tr>
<tr>
<td>JBS2</td>
<td>Number</td>
<td>4,267,415</td>
<td>23,136</td>
<td>8,796-96,342</td>
</tr>
<tr>
<td></td>
<td>Prevalence</td>
<td>22.0%</td>
<td>22.4%</td>
<td>17.2-26.4%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Practice level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
</tr>
<tr>
<td>QRISK2</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>JBS2</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

**Prevalence of risk factors in the high risk population**

The distribution of individual CVD risk factors in patients with high risk scores is presented in Table 9-3. Within the high risk population defined by the QRISK2 score, there were large proportions of physically inactive (82.6%) and overweight or obese (65.1%) patients. I estimate 23.9% of the eligible population (479,200 patients) will have undiagnosed hypertension and 11.3% (228,800 patients) will have impaired fasting glucose. Screening will detect 114,400 patients with undiagnosed diabetes, 5.7% of the screened population, with a median of 604 [range =222-2,463] per PCT.

The distribution of risk factors identified using JBS2 differ from that estimated from QRISK2 (Table 9-3) The JBS2 population had an especially large proportion of patients who smoke (42.7%), with undiagnosed hypertension (27.2%), and larger numbers of high risk in younger age groups.
<table>
<thead>
<tr>
<th>High risk</th>
<th>QRSK2</th>
<th>JBS2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. in England</td>
<td>Median per PCT [Range]</td>
</tr>
<tr>
<td>40-49 years</td>
<td>6,943</td>
<td>39 [1-167]</td>
</tr>
<tr>
<td>50-54 years</td>
<td>19,790</td>
<td>116 [13-385]</td>
</tr>
<tr>
<td>55-64 years</td>
<td>255,565</td>
<td>1,517 [361-4,293]</td>
</tr>
<tr>
<td>65-74 years</td>
<td>1,729,853</td>
<td>9,087 [3,423-38,720]</td>
</tr>
<tr>
<td>Smoking</td>
<td>558,641</td>
<td>3,134 [966-10,540]</td>
</tr>
<tr>
<td>Undiagnosed Hypertension</td>
<td>479,150</td>
<td>2,601 [898-9,985]</td>
</tr>
<tr>
<td>Physically inactive</td>
<td>1,663,779</td>
<td>8,958 [3,151-35,163]</td>
</tr>
<tr>
<td>Overweight/ obese</td>
<td>1,310,354</td>
<td>7,041 [2,479-27,589]</td>
</tr>
<tr>
<td>Unrecorded IFG</td>
<td>228,817</td>
<td>1,207 [445-4,928]</td>
</tr>
</tbody>
</table>
Figure 9-1 shows the geographical distribution of high risk status in England using both risk scores. The highest prevalence of high risk status is centred on the major urban areas in northern England. The absolute prevalence of high risk status changes with risk score and there is a change in relative distribution of prevalence. The national ranking of both PCT (Wilcoxon signed rank, p=0.017) and practice (p= <0.001) prevalence varies significantly upon changing risk score.

Cost estimates

In the 2,012,151 patients estimated at high risk using QRISK2, with a total of 1,509,113 (75%) screened, I estimate the total cost of the basic Health Check for the target population to be £30,814,000 (Table 9-4) The median cost per PCT is £166,000, ranging from £58,000 to £647,000. The total cost using QRISK2, including interventions for the six risk factors, was £176,057,000, with a median per PCT £936,000, ranging from £335,000 to 3,725,000. In comparison, the total costs including the six risk factor interventions, using JBS2 is £378,383,000 (PCT median=£2,047,000), ranging from £776,000 to £8,534,000.
### Table 9-4: Costs of the NHS Health Check

<table>
<thead>
<tr>
<th></th>
<th>JBS2</th>
<th>QRISK2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total Cost</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>National Total</td>
<td>£378,382,784</td>
<td>£176,057,856</td>
</tr>
<tr>
<td>PCT Median</td>
<td>£2,489,360</td>
<td>£936,465</td>
</tr>
<tr>
<td>(Range)</td>
<td>(£776,450-8,533,836)</td>
<td>(£335,596-3,724,891)</td>
</tr>
<tr>
<td><strong>Screening</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>National Total</td>
<td>£65,556,741</td>
<td>£30,814,363</td>
</tr>
<tr>
<td>PCT Median</td>
<td>£355,458</td>
<td>£165,998</td>
</tr>
<tr>
<td>(Range)</td>
<td>(£135,172-1,479,937)</td>
<td>(£58,116-647,036)</td>
</tr>
<tr>
<td><strong>Interventions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical activity</td>
<td>£39,168,904</td>
<td>£20,278,365</td>
</tr>
<tr>
<td>Smoking</td>
<td>£23,025,134</td>
<td>£7,177,940</td>
</tr>
<tr>
<td>Hypertension</td>
<td>£4,953,264</td>
<td>£2,045,088</td>
</tr>
<tr>
<td>Statin</td>
<td>£82,321,639</td>
<td>£38,815,898</td>
</tr>
<tr>
<td>Weight management</td>
<td>£33,891,765</td>
<td>£16,273,074</td>
</tr>
<tr>
<td>IFG lifestyle</td>
<td>£129,465,337</td>
<td>£60,653,128</td>
</tr>
</tbody>
</table>

### 9.4 Discussion

**Main findings**

Using the QRISK2 risk score, the model estimates approximately 2 million patients aged 40 to 74 at high risk of CVD in England, with geographic variation in prevalence. JBS2 results in twice as many high risk (4.3 million people), and changes the relative prevalence between PCTs. I identified a high prevalence of cardiovascular risk factors in this population; for example 65 percent of high risk patients will be overweight or obese (BMI >25 kg/m²), 83 percent physically inactive and over 220,000 will have unrecorded impaired fasting glucose.

The levels of CVD risk factors captured by the high risk population were dependent on the risk score used; with relatively higher levels of smoking and raised blood pressure with JBS2. I estimate the cost of screening and initial interventions in the high risk population using the QRISK2 score was approximately £176 million, whilst using JBS2 the cost would be £378 million.

CVD risk scores were derived as clinical tools to assist treatment, although they can be used to compare levels of risk between populations. There is geographic variation in the
distribution of high CVD risk across England, with prevalence highest in the urban centres of the north. Adding social deprivation to CVD risk scores is believed to improve the equity in their use.\textsuperscript{393} The QRISK2 algorithm alters the relative high risk status between PCTs and practices nationally, possibly due to the inclusion of deprivation and ethnicity.

Profiles of CVD risk factors differ between high risk populations defined by the two risk scores, for example there is a significantly higher smoking prevalence when defined using JBS2. The most common risk factors in the derivation dataset are the most prevalent in a high risk population defined by that risk score.\textsuperscript{418} JBS2, based on the \textit{Framingham} risk score (\textit{Anderson}), was produced using data from the USA in the 1970s where smoking prevalence was higher than today.\textsuperscript{20} The QRISK2 dataset covers the UK between 1993 and 2003, a time with higher levels of inactivity and obesity, with for example a threefold increase in the prevalence of obesity from 1980 to 1998.\textsuperscript{593}

The programme’s main focus is CVD risk, although the programme may have wider impacts. My data estimate that within the high risk population there is a large amount of both undiagnosed diabetes and unrecorded IFG. Early diagnosis of diabetes leads to earlier treatment and reduces complications.\textsuperscript{594} For IFG, interventions to reduce the progression to diabetes are costly but effective,\textsuperscript{595,596} and benefits over a long period.\textsuperscript{597} Slowing progression to diabetes is beneficial both to patients by increasing life expectancy and a health system in saving cost.\textsuperscript{598}

I could not directly compare estimates of the cost of Health Checks programme with those generated by the Department of Health (£180-243 million annually) because the latter include interventions in non-high risk patients and the methods used are different, notably our omission of the assumption that 50 percent of costs are not attributable to the programme. The analysis suggests that screening and treating the high risk group alone will cost at least the amount of the annual Department of Health cost estimates. This is for two main reasons; firstly the high prevalence of risk factors seen in my high risk cohort increases the cost of patient management. Secondly, and perhaps crucially, there were differences in
the modelling methods. As noted previously, Department of Health estimates eliminate half of the potential costs on the grounds that these Health Checks are already being carried out. Based on Department of Health figures this equates to nearly 1.1 million health checks carried out annually before 2009. Evidence for this level of risk assessment in general practice is unclear, and the assumption seems unfounded, especially with its implication on reducing modelled programme costs. NHS Health Check once fully implemented will be a five year rolling programme, with 20 percent of the eligible population screened annually. The high risk cohort identified here is fewer in number than the annual target for screening, although they possess increased levels of risk factors, increasing the cost of management.

**What is already known?**

Modelled data presented here are the first estimates of the prevalence of high CVD risk status in England, using a common definition from national guidance.\textsuperscript{113} Using *Framingham* risk scores in small populations there have been estimates of 15.8\textsuperscript{145} and 19.1 percent\textsuperscript{83} in the 35 to 74, 15.5 percent in the 30 to 74 and 17.8 percent in the 40 to 70 age groups;\textsuperscript{145} with 13.3 percent of the 35 to 74 population for QRISK\textsuperscript{2}\textsuperscript{83}. The evaluation of a pilot CVD risk assessment service in pharmacies found 70 percent of the population screened required referral to general practice through raised risk factors.\textsuperscript{146} This population was not at high risk, but nevertheless, as here, had a large need for referral.

**Strengths and limitations**

The NHS Health Check, although a national programme, is being implemented locally by service providers. With, as yet, no national call-recall system or minimum data set, the programme is likely to remain fragmented. There has been no coordinated collection of NHS Health Check data across England to date, especially at a patient level, although this was planned from April 2011. This makes modelling a vital element in the programme evaluation.

My modelling estimates CVD risk factors using prevalence estimates of risk factors derived from the high risk population, instead of using general population estimates which are likely
to be lower. I also use practice population data which captured local level differences in the population make-up.

Limitations include missing data for some HSfE variables used. This is common when using large population based datasets, for instance in the derivation of QRISK2. Blood pressure, BMI and lipid ratio recording was incomplete; I used multiple imputation to replace missing data. Although more complete data would be preferred, multiple imputation can give unbiased estimates even with a large proportion of missing data, and I generated a sufficient number of imputed data datasets. Data were more likely to be missing in deprived respondents and those of black ethnicity (Table viii-1), but multiple imputation used these as covariates. Estimates of predicted CVD risk, and therefore the prevalence of high risk may be more accurate in the affluent white population. Differences in risk factors between deprived and affluent, and white and black respondents may be open to dilution.

A problem with HSfE sampling, based on households, is there is no information on unresponsive households. Even the number of non-responders is unknown. There is an estimated response rate of 61 percent in the 2006 survey, 88 percent for individuals within compliant households. There are no data on the ethnic patterns of non-response. We used fasting plasma glucose as the diagnostic criteria for diabetes, since the analysis HbA1c can be used for diagnosis, however this has limited impact on prevalence of undiagnosed diabetes.

I did not have access to data concerning CKD or rheumatoid arthritis, two components of the QRISK2 score. Neither, however, are core components of the algorithm and the risk score allows them to be assumed absent if data are missing. Family history of CVD was further assumed null when missing; sensitivity analysis using only complete data, however gives comparable model outputs. Similarly a sensitivity analysis was carried out in those have complete data overall (barring CKD, heart failure, AF or rheumatoid arthritis) and again model outputs were similar.
One aim was to estimate the cost of Health Checks in the high CVD risk population; I did not consider costs in the low risk population. Some of the estimates used in this assessment, such as programme uptake of 75 percent, have been questioned (Chapter 11). Lower uptake will reduce programme costs but in turn decrease programme effectiveness. In practice, with staffing a major element of programme cost, the effectiveness might be reduced for no great reduction in spending. I constrained modelling to the invitation and intervention uptake assumptions made in the Department of Health economic assessment.

My evaluation uses only the predicted uptake from the Department of Health, but I explored variation in level of uptake and found workload and cost both varied in a linear fashion. I did not conduct any further formal sensitivity analysis, as there are many assumptions which could be varied, for example the uptake for each intervention. The modelling only examined costs, and should form the basis for a full cost effectiveness analysis focussed specifically on the high risk population, properly comparing QRISK2 and JBS2, and considering the health outcomes of the programme.

Implications for practice

Evidence demonstrates the population impact of healthcare interventions in high CVD risk groups. However, these must be complemented by population-based interventions to reduce the burden of CVD, for example legislation to reduce salt content of food. Preventative strategies in the UK, including the NHS Health Check, have focused on high risk approaches. In the current financial situation NHS resources must be allocated carefully and given population strategies are effective and cost saving there is a strong case for greater utilisation of this approach.

I have highlighted the considerable work load implications of the Health Check programme in primary care, entailing the screening and ongoing management of high risk patients. The work load of risk management will vary enormously between practices: with some practices estimated to have over 1,693 high risk patients using QRISK2. Local commissioners must account for these differences when allocating resources to achieve an equitable service.
Many local commissioners remunerate general practices for Health Check work through LESs. Although providing funding, this method of payment does not help practices with organisational and capacity limitations. Further, with geographic variation, national funding might be better targeted using prevalence of high risk, instead of population size alone.

Lifestyle and community based interventions to address risk factors such as obesity and low levels of physical activity remain underdeveloped. Commissioners must ensure there is sufficient capacity for referral to manage the CVD risk unearthed by the programme.

Health Checks include the offer of a statin to all patients at high risk of CVD; this will generate large prescribing costs, with over two million patients eligible for therapy. There is strong evidence for the ability of statins to reduce lipid levels, with a resultant reduction in CVD events. This is found in patients with existing CVD and those at high risk.

Recent evidence has questioned the effectiveness of statins in primary prevention in groups at lower risk. This is even after common statins, including Simvastatin and Pravastatin lost patent, becoming low cost. They are not completely ineffective in low to moderate risk groups; the question is whether they produce enough of an absolute risk reduction to be beneficial and cost effective. Although increasing age improves cost effectiveness, even patients aged 65 require a 10 percent ten-year risk to have favourable cost effectiveness.

The evidence for the use of statins in a high risk group, however, remains strong. From the age of 45, in patients with a risk of greater than 15 percent, the incremental cost effectiveness ratio fell to £22,000 per QALY. This is in line with previous work suggesting cost effectiveness is strongly linked to initial levels of risk. With evidence of their effectiveness in this group, provisions must be made for the large expenditure.

With global CVD risk promoted as a method to guide and target intervention, it seems illogical not to also use it to prioritise interventions at the time of screening. Targeted screening may be a more effective approach than a universal method like the NHS Health
Risk prediction using data from patients’ EMRs can be efficient in prioritising treatment, and I previously demonstrate this need not use complex methods (Chapter 8).

The Department of Health and NICE have specified the use of either the QRISK2 or JBS2 risk scores in both the NHS Health Check and statin prescribing. Modelling suggests the choice of risk score is important. If the QRISK2 score outperforms Framingham scores at risk prediction in England, it may be the preferential risk score. Further, data presented here find lower costs by approximately half, largely due to the smaller high risk cohort, making QRISK2 attractive.

A major deficiency in the Health Check Programme has been the lack of definition of the size of the high CVD risk population. The progress of the Programme should be judged by outcome measures, not solely on simple metrics such as the number of Checks offered. Possibilities include the size of the high risk population, or the individuals’ global CVD risk. This study has provided a baseline estimate against which the success of the Programme can be monitored.

Conclusions

There is a high prevalence of patients eligible for intensive intervention through the NHS Health Check and within this population there is a high prevalence of CVD risk factors; especially obesity and physical inactivity. Unless adequate local access to effective interventions is provided, there is a risk the Health Checks Programme will fail to meet its objectives. The choice of CVD risk score will affect the nature, size and costs of the high risk population identified by the Programme. To create a standardised, cost-effective programme across England, the NHS should consider using the same risk score nationally.
Chapter 10; Implementation of the NHS Health Check programme: baseline assessment of risk factor recording in an urban, culturally diverse setting

10.1 Introduction

The potential work load implications of the NHS Health Check programme for primary care are considerable. The Health Check is expected to be carried out in one encounter, with risk factor data recorded before the date of the Health Check invalid. A single consultation focusing on CVD is hoped to enhance the patient’s concept of vascular risk and in turn improve the likelihood of positive action. Even if people have data recorded this should be re-measured. A more pragmatic approach is for GPs not to retake recent measurements already in the person’s records, and there is anecdotal evidence suggesting that in areas this is happening. If this is so, the work load created by the NHS Health Check will be dependent on the completeness of medical records before the programme.

Incomplete CVD risk factor data may have other implications. Practices may call the population to be screened based upon estimated CVD risk. NHS Ealing, for example, targeted patients with an estimated risk score of ≥20 percent in the first year of the programme. Risk stratification can be achieved with incomplete records, but more complete data give better sensitivity and specificity. Complete and rich primary care data is beneficial to patient care, and secondary health surveillance, but there are variations in quality. For example, prescribing data are more complete than lifestyle and demographic data, and recording of consultations greater than morbidity. There is high practice variation in medical records and before the QOF, variation between disease groups.

Since the introduction of the QOF to general practice in 2004, there has been an improvement in the recording of risk factor data in areas covered by indicators. A number of studies have demonstrated that in the early stages of the QOF there were improvements in
patient records. Diabetic patients saw improved blood pressure, cholesterol, HbA1c, weight\textsuperscript{607,608} and smoking status\textsuperscript{609} recording; and patients with CVD had improvements in recording across a number of risk factors\textsuperscript{178}.

It is not certain whether the QOF is entirely causal in these improvements\textsuperscript{610}, or whether incentives provided payment for changes already underway\textsuperscript{611}. Further the QOF might distort care, focusing on areas that are incentivised, not on areas at the core of clinical care\textsuperscript{611}. There may have been trends of improvement before 2004. The recording of blood pressure increased continuously from 1997 to 2007 in patients with diagnosed CVD\textsuperscript{612}. CVD patients also saw improved recording of lipid levels over a similar time period\textsuperscript{613}. In patients with diabetes, between 1997 and 2005 the recording of blood pressure, lipid levels and HbA1c all improved\textsuperscript{614}. Whatever the cause, there are clear improvements in the completeness of medical records in chronic disease groups, and many are now largely complete.

Recording of risk factors in the population without established disease appears poorer, although recent evidence is sparse, especially when considering patient level differences. Between 1997 and 2007 in Wandsworth, as well as the increase in blood pressure recording in CVD patients, there were increases in the general population\textsuperscript{612}. By 2007, in the population aged 45 and over, 37 percent of the population had a blood pressure measurement recorded in the previous year.

The recording of blood pressure is likely to be high. Using a cross section of general practices, Ashworth et al.\textsuperscript{615} found 88 percent of the population aged 45 and over to have a blood pressure measurement from the proceeding five years. Unlike other CVD risk factors in the general population, however, there is a QOF indicator covering the recording of blood pressure. Achievement in this indicator has improved since the start of the QOF\textsuperscript{615}. Even before the incentive payment, blood pressure saw considerably better recording than other CVD risk factors including BMI and lipid levels\textsuperscript{435}. 
Less still is known about differences in CVD risk factor recording between patients and practices. Ashworth et al. found poorer blood pressure recording in more deprived areas in 2005, although these small differences were largely abolished by 2007, possibly due to the QOF payments. Practices with a lower list size to GP ratio have been found to have poorer recording, as have practices with a higher proportion of black and south Asian patients; and women have more complete records than men.

In the 1980s, a greater amount of work studied the completeness of patient medical records. Although the levels of recording will be out of date, these data may give important lessons concerning patterns in recording. In 1982, 35 percent of all patients in three general practices had a blood pressure record from the previous five years, 11 percent for smoking status. A subsequent intervention to facilitate health checks improved recording to 59 and 49 percent respectively. In 1987, across 24 Scottish practices, 68 percent of men aged 35 to 64 had a blood pressure record and 47 percent smoking. There was considerable practice variation, with ranges of 53 to 99 and 1 to 100 percent respectively.

A patient survey between 1993 and 1994 found smoking records in 75 percent of patients, although this finding is weakened by returner bias in the questionnaire. Fleming et al. found blood pressure recording improved in practices with smaller list sizes, although this was not found for smoking status. Recording improved in practices which made an explicit attempt to audit medical records.

Internationally, in the Netherlands, there was large variation between general practices in blood pressure recording (12 to 76%), with blood pressure the most complete CVD risk factor. Likewise in Finland, blood pressure was the best recorded in general practice, and the introduction of structured risk factor recording forms increased recording.

The aim was to determine the level of risk factor recording and variation with patient characteristics in Ealing before the NHS Health Check programme. I further sought to quantify work load for primary care teams in England of data recording in the programme.
10.2 Methods

Data

I obtained data from 14 practices participating in the NHS Health Check programme in Ealing, extracting data before the programme between December 2008 and January 2009 using the Oberoi Primary Prevention Extraction Software (Chapter 2.2.1). Briefly, the software extracted patient data for all those aged 35 to 74 years and not already on a CVD (CHD, stroke, TIA) or diabetes register. Data extracted include CVD risk factor data, patient characteristics and the date that the last record was made. My analysis concentrates on the recording of five CVD risk factors, namely blood pressure, total cholesterol values, BMI, smoking status and ethnicity. Analysis covers only the 14 practices who were the earliest adopters of the Health Check programme.

As the Health Check programme requires the up to date measurement of risk factors, I only reported the proportion of patients with a measurement taken within the last 5 years and also examined the proportion of patients with a blood pressure reading from within the last two years. For analysis, I split age into ten-year bands, from 35 to 74 inclusive. Each patient was assigned a deprivation (IMD 2007) score based on postcode of residence and assigned to a population weighted deprivation fifth for England. In addition, I assigned each practice a deprivation score as follows. From the mean of the deprivation scores of patients aged 35 to 74 in the practice, I found the English fifth of deprivation the practice was located in, analogous to the method described by Strong et al.\textsuperscript{622} I obtained two further practice characteristics, practice list size\textsuperscript{590} and number of full time equivalent (FTE) GPs; then calculated the average number of patients per FTE GP by dividing the former by the latter.

Analysis

I examined overall levels of data recording, variation between practices and effects of patient characteristics. Using median practice level of recording, I estimated the work load implications of the programme for a practice with an average list size in England in 2007.
I examined the recording of blood pressure, total cholesterol, BMI and smoking using multilevel logistic regression analysis. I used a random effects model with the patient at level one and practice at level two. I compared the random effects model to an unstructured model using a log-likelihood test, and found the former to offer better fit. All independent variables were added into the model with fixed slopes. I built the model using backward stepwise selection, a common method of modelling. The initial full model contained the variables in Table 10-1. I added the interaction term age*sex into the model, and tested its statistical significance using a log-likelihood test. Consultation rates have an age and sex interaction, with the largest gap between women and men in the young adults; this relationship could persist into risk factor recording. I removed independent variables during model building based on both likelihood-ratio tests and the AIC, the most parsimonious model was obtained when there was no longer a fall in AIC and p value for the likelihood-ratio test was below 0.2. The final parameters in each model are those shown in Table 10-3.

The Hosmer-Lemeshow test, normally used to test Goodness of Fit (GOF) of logistic regression models was not used. In large samples the test is oversensitive to small deviation. Instead, observations with large standardised Pearson's residuals (>3) were examined and residuals were checked for relationships to independent variables. Analyses were carried out using Stata version 10.1, using the `xtmelogit` command for the regression. Ethical approval was obtained from the London Research Ethics Committee.
10.3 Results

A summary of the patient characteristics is in Table 10-2. There were 21,510 individuals aged 35 to 74 years within the 14 practices; 11,377 male and 10,133 female. The mean age was 50.2 years. In one practice, data were not extracted on ethnicity. Excluding this practice, the percentage of patients with ethnicity recorded was 55.6%. Of those with a valid record for ethnicity, 38.5% were from a white ethnic background, 39.6% from south Asian and 10.4% from a black ethnic background. The total ethnic makeup of the population (Table 10-2) differed markedly between practices. The median deprivation score for patients’ area of residence was 25.5, compared with 17.1 for England as a whole and 25.1 in Ealing.

Table 10-2; Characteristics of the patient population and the recording of blood pressure, total cholesterol, BMI and smoking within the subgroups

<table>
<thead>
<tr>
<th></th>
<th>Number of patients (%)</th>
<th>Blood pressure (% recorded)</th>
<th>Total cholesterol (% recorded)</th>
<th>BMI (% recorded)</th>
<th>Smoking (% recorded)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>10,133 (41.1%)</td>
<td>91.6</td>
<td>62.3</td>
<td>67.6</td>
<td>97.8</td>
</tr>
<tr>
<td>Male</td>
<td>11,377 (52.9%)</td>
<td>80.2</td>
<td>52.0</td>
<td>79.3</td>
<td>93.9</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>35-44</td>
<td>8,352 (38.8%)</td>
<td>79.5</td>
<td>42.0</td>
<td>70.4</td>
<td>94.7</td>
</tr>
<tr>
<td>45-54</td>
<td>6,559 (30.5%)</td>
<td>87.2</td>
<td>59.8</td>
<td>72.1</td>
<td>96.0</td>
</tr>
<tr>
<td>55-64</td>
<td>4,250 (19.8%)</td>
<td>90.4</td>
<td>68.9</td>
<td>75.1</td>
<td>96.2</td>
</tr>
<tr>
<td>65-74</td>
<td>2,349 (10.9%)</td>
<td>93.8</td>
<td>79.5</td>
<td>79.6</td>
<td>97.8</td>
</tr>
<tr>
<td><strong>Deprivation (IMD</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>third, 3 least</td>
<td>11,461 (53.3%)</td>
<td>85.1</td>
<td>59.8</td>
<td>71.3</td>
<td>95.4</td>
</tr>
<tr>
<td>deprived )</td>
<td>8,504 (39.5%)</td>
<td>86.0</td>
<td>55.0</td>
<td>74.7</td>
<td>96.3</td>
</tr>
<tr>
<td>2</td>
<td>1,545 (7.2%)</td>
<td>86.9</td>
<td>45.4</td>
<td>74.3</td>
<td>95.7</td>
</tr>
<tr>
<td><strong>Ethnic group†</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>3,646 (20.6%)</td>
<td>93.4</td>
<td>51.7</td>
<td>87.3</td>
<td>99.0</td>
</tr>
<tr>
<td>Mixed</td>
<td>555 (3.1%)</td>
<td>94.4</td>
<td>60.9</td>
<td>91.9</td>
<td>99.6</td>
</tr>
<tr>
<td>Black</td>
<td>984 (5.6%)</td>
<td>93.7</td>
<td>61.5</td>
<td>87.9</td>
<td>99.2</td>
</tr>
<tr>
<td>South Asian</td>
<td>3,754 (21.2%)</td>
<td>95.9</td>
<td>75.0</td>
<td>91.9</td>
<td>98.7</td>
</tr>
<tr>
<td>Other</td>
<td>539 (2.5%)</td>
<td>93.9</td>
<td>56.2</td>
<td>86.8</td>
<td>99.1</td>
</tr>
<tr>
<td><strong>Hypertension</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>8,242 (55.7%)</td>
<td>78.5</td>
<td>52.1</td>
<td>59.6</td>
<td>93.2</td>
</tr>
<tr>
<td>Yes</td>
<td>3,245 (15.1%)</td>
<td>99.3</td>
<td>91.5</td>
<td>89.7</td>
<td>99.9</td>
</tr>
<tr>
<td>No</td>
<td>18,265 (84.9%)</td>
<td>82.6</td>
<td>50.9</td>
<td>69.8</td>
<td>95.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>21,510</td>
<td>85.6</td>
<td>55.6</td>
<td>72.8</td>
<td>95.8</td>
</tr>
</tbody>
</table>

† this excludes data from a practice where there were no ethnicity data extracted

Patient and practice level variation in recording of risk factors

There was high recording of smoking status, 95.8% of the population, and a small, though statistically significant, variation between practices (range 91.7% - 100% p<0.001) (Table
Recording of blood pressure was high; overall 85.6% of patients had a valid blood pressure measurement recorded in the last 5 years, and 69.0% in the last two years. BMI was less well recorded at 72.8%, whilst lipids were even less so; 59.9% of patient records having a total cholesterol value.

Risk factor recording varied with patient characteristics (Table 10-2). There was higher risk factor recording in women (odds ratio (OR) = 2.89 [2.28-3.32] for blood pressure and 1.66 [1.56-1.78] for cholesterol) and with increasing age (OR = 1.91 [1.78-2.05] for 45-54, 2.86 [2.62-3.13] for 55-64 and 4.11 [3.64-4.65] for 65-74 all compared with 35-44 years old in blood pressure recording. Recording of cholesterol and blood pressure was higher in patients living in the most deprived areas. Blood pressure was more likely to be recorded in south Asian (AOR = 1.38 [1.09- 1.75]) than in white patients and less so in those without ethnicity recorded (AOR = 0.29 [0.25- 0.34]). For cholesterol and BMI, there was better recording in south Asian patients and patients with mixed ethnicity than in white patients and poorest if the ethnicity was missing, the later was true for smoking status.

There was a large between-practice variation in the risk factor recording (Figure 10-1). Blood pressure (81.5% - 95.2%); total cholesterol (33.6% - 78.0%); HDL (30.9% - 77.4 %) and BMI (52.0%-94.0%) recording all varied by practice. Recording of ethnicity also varied considerably between practices (9.8% - 95.9%).

Model Building

The multilevel logistic models derived are in Table 10-3. None of the models had a relationship between standardised Pearson residual and the model variants. In examining the magnitude of the residuals, eight observations were large in the model for blood pressure and were removed. The odds ratios and the associated confidence intervals for models, for recorded blood pressure, cholesterol, BMI and smoking are in Table 10-3. Hypertensive status was the strongest predictor of risk factor recording, with patients having better recording of the three variables (blood pressure AOR = 36.3 [21.0-62.9]; cholesterol AOR = 7.8 [6.78-8.87]; BMI = 3.23 [2.84-3.68]; smoking AOR= 30.3 [11.3-81.2])
Large univariate practice variation found did not persist in three of the regression analyses, and the intra-class correlation coefficients ($\rho$) from the models (nested within practices) were small ($\rho=0.017, 0.0079$ and $0.031$ respectively). These small values indicate most of the variation between practices is accounted for by other variables included in the models. Practice level variation remained an important explanatory variable for recording of smoking status after adjustment ($\rho=0.22$).

**Predicted work load for general practice in England**

The mean list size for general practices in England during 2007-8 was 6,511. By applying the 2007 age structure of the population of England and assuming levels of missing data similar to Ealing (within the population aged 40-74, not 35-74 as reported above), I estimate that an average practice with 2,660 registered patients aged 40-74 years would need to undertake 356 blood pressure, 721 BMI and 1,018 cholesterol measurements to fill missing risk factor data.
Table 10-3: multiple logistic regression analysis of blood pressure, cholesterol and BMI recording controlling for practice level clustering

<table>
<thead>
<tr>
<th></th>
<th>Blood pressure AOR</th>
<th>95% CI</th>
<th>Cholesterol AOR</th>
<th>95% CI</th>
<th>BMI AOR</th>
<th>95% CI</th>
<th>Smoking AOR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>age/ sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>female 35-44</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>male 35-44</td>
<td>0.34**</td>
<td>0.30-0.38</td>
<td>0.66**</td>
<td>0.61-0.73</td>
<td>0.46**</td>
<td>0.41-0.51</td>
<td>0.39**</td>
<td>0.31-0.49</td>
</tr>
<tr>
<td>female 45-54</td>
<td>1.74**</td>
<td>1.47-2.06</td>
<td>2.28**</td>
<td>2.05-2.53</td>
<td>1.01</td>
<td>0.89-1.15</td>
<td>1.40*</td>
<td>1.01-1.93</td>
</tr>
<tr>
<td>male 45-54</td>
<td>0.57**</td>
<td>0.50-0.65</td>
<td>1.10</td>
<td>1.00-1.22</td>
<td>0.47**</td>
<td>0.42-0.52</td>
<td>0.48**</td>
<td>0.37-0.61</td>
</tr>
<tr>
<td>female 55-64</td>
<td>2.06**</td>
<td>1.66-2.56</td>
<td>3.04**</td>
<td>2.68-3.46</td>
<td>0.94</td>
<td>0.82-1.09</td>
<td>1.25</td>
<td>0.86-1.82</td>
</tr>
<tr>
<td>male 55-64</td>
<td>0.73**</td>
<td>0.62-0.86</td>
<td>1.81**</td>
<td>1.61-2.04</td>
<td>0.55**</td>
<td>0.48-0.62</td>
<td>0.40**</td>
<td>0.31-0.53</td>
</tr>
<tr>
<td>female 65-74</td>
<td>2.13**</td>
<td>1.58-2.87</td>
<td>3.55**</td>
<td>2.99-4.22</td>
<td>0.98</td>
<td>0.81-1.17</td>
<td>1.43</td>
<td>0.84-2.46</td>
</tr>
<tr>
<td>male 65-74</td>
<td>1.07</td>
<td>0.83-1.37</td>
<td>3.15**</td>
<td>2.66-3.75</td>
<td>0.68**</td>
<td>0.57-0.81</td>
<td>0.55**</td>
<td>0.37-0.82</td>
</tr>
<tr>
<td>ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>white</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>mixed</td>
<td>1.17</td>
<td>0.79-1.73</td>
<td>1.23</td>
<td>1.00-1.51</td>
<td>1.77**</td>
<td>1.27-2.46</td>
<td>1.98</td>
<td>0.60-6.53</td>
</tr>
<tr>
<td>black</td>
<td>1.13</td>
<td>0.84-1.51</td>
<td>1.20*</td>
<td>1.02-1.42</td>
<td>1.06</td>
<td>0.83-1.34</td>
<td>1.18</td>
<td>0.54-2.60</td>
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<td>1.38**</td>
<td>1.09-1.75</td>
<td>1.47**</td>
<td>1.30-1.66</td>
<td>1.13</td>
<td>0.92-1.39</td>
<td>1.27</td>
<td>0.79-2.04</td>
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<td>0.25-0.34</td>
<td>0.88**</td>
<td>0.80-0.96</td>
<td>0.31**</td>
<td>0.28-0.35</td>
<td>0.19**</td>
<td>0.13-0.28</td>
</tr>
<tr>
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<td>1.03-1.20</td>
<td>1.16**</td>
<td>1.07-1.27</td>
<td>-</td>
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</tr>
<tr>
<td>deprived)</td>
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<td>3</td>
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<td>0.54-0.70</td>
<td>0.42**</td>
<td>0.38-0.46</td>
<td>0.84*</td>
<td>0.72-0.98</td>
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<td>0.05-1.25</td>
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<tr>
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<td>1.02-1.15</td>
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<td>1.0001</td>
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<tr>
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<td>36.3**</td>
<td>21.0-62.9</td>
<td>7.76**</td>
<td>6.78-8.87</td>
<td>3.23**</td>
<td>2.84-3.68</td>
<td>30.3**</td>
<td>11.3-81.2</td>
</tr>
</tbody>
</table>

* p<0.05
** p<0.01
† The UK fifth that the practice falls into
10.4 Discussion

Main findings

In this culturally and socio-economically diverse area of London, the recording of smoking status and blood pressure was very high in the EMRs of patients aged 35 to 74 years without existing CVD or diabetes. The recording of BMI and cholesterol was considerably lower. Ethnicity recording was highly variable and was very low in some practices. There were large differences in the levels of recording by patient characteristics.

The main determinant of risk factor recording was hypertensive status. For all risk factors, patients on the hypertension register were more likely to have a risk factor recorded. Hypertensive patients are annually invited for a review of their condition, and practices receive QOF points for recording their blood pressure. The regular attendance in general practice gives opportunity to maintain their medical records, and they are a group known to be at high risk of CVD therefore get more attention to the risk factors.

There was better recording of all risk factors in women than in men, despite men being at higher risk of CVD and its complications. This may reflect higher consultation rates among women (Chapter 5.3.1). There was increased recording with age, except for BMI in females, again likely to be a result of increased general practice attendance. The older population are also at higher CVD risk; therefore have improved risk factor recording. Younger women could potentially be receiving BMI measurements due to increased contact with primary care when receiving contraception.

South Asian patients had better blood pressure and total cholesterol recording than white patients, whilst those with no recorded ethnicity had poorest data records. South Asian patients may have high attendance at general practice, giving more opportunity to measure risk factors. With a high number of South Asian GPs in Ealing, there may be better care with ethnic concordance between patient and GP, or South Asian patients may have better risk factor recording because they are perceived to be at greater risk of CVD.
Patients with missing ethnicity records are likely to have little contact with general practice, therefore have poor recording across all risk factors. This group could further consist of a large number of *ghost patients*, for whom it is impossible to record risk factors. Within Ealing up to 11 percent of patients on GP registers could be no longer actively using the practice.\(^5\)

Recording of risk factors was found to be significantly lower in more deprived practices, even after adjustment for a patient deprivation measure; such practices may provide poorer quality care.\(^6\) The larger the practice size the better the recording of cholesterol and BMI. Larger practices generally have more practice staff, such as nurses and health care assistants, and may also have better organisation and performance.\(^6\) My findings contrast with those from practice-level studies with no differences in risk factor recording in patients with cardiovascular disease and diabetes by practice size.\(^6\) This likely reflects the impact of financial incentives within QOF which focus primarily on improving secondary prevention. Although there are three QOF indicators concerning CVD risk factors in those without current disease (covering smoking status and blood pressure recording), the size of the financial incentive is small. Nonetheless, recording of these risk factors was found to be considerably higher in this study than cholesterol or BMI.

*What is already known?*

My findings are consistent with previous research which has found that blood pressure recording in people aged over 45 years without a chronic disease was high in the UK, and had improved since the introduction of the QOF.\(^\)\(^6\)\(^1\)\(^5\) Previous work, even before the introduction of financial incentives for blood pressure recording, suggested cholesterol and BMI were more poorly recorded than blood pressure.\(^\)\(^4\)\(^3\)\(^5\) Recording of blood pressure was poorer in deprived areas, but the introduction of financial incentives may have abolished this.\(^6\)\(^1\)\(^5\) A recent study examined the completeness of data records after a CVD risk assessment. Data were more complete in white attendees than south Asian, poorest when no ethnicity was recorded, and poorer in single handed practices and smokers.\(^\)\(^1\)\(^4\)\(^7\)
My findings differ from previous studies which found poorer recording of risk factors in practices with a higher proportion of black or south Asian patients. However, the association found by Ashworth et al. was an examined using ecological analysis with no access to patient level information. My findings confirm previous studies which suggest that risk factor recording is higher in women. There is large practice level variation in general measures of quality, and in the completeness of primary care records.

**Strengths and limitations**

This was a population-based study conducted in a culturally and socio-economically heterogeneous part of the UK. I obtained data on all patients aged 35 to 74 years without existing CVD and diabetes in 14 practices. Practices in Ealing are all computerised and, through the QOF, have invested time and resources improving the recording of risk factors, particularly for patients with diseases included in the QOF.

A weakness of the study is not all practices took part in the pilot, although practices that did so were broadly representative of practices in Ealing. I assigned an area-based deprivation score to patients and practices using postcodes. It would be preferable to employ patient level measures of socio-economic circumstances, such as income or education status, but these are not routinely recorded in UK general practice. It may have been informative to examine the impact of other practice and practitioner characteristics, such as the age and nationality of the GP and whether they were trained in the UK or abroad. Finally, I had no data concerning blood glucose measurement, another element of the programme.

**Implications for practice**

I found some CVD risk factors, smoking status and blood pressure, are well recorded in general practice. Others, such as BMI and lipids, are less well recorded. For an average practice in England (with a list size of 6,500 and 2,660 patients aged 40 to 74 years), I estimate that over 1,018 lipid, 721 BMI and 356 blood pressure measurements will be required for the Health Check Programme.
Programme workload will extend beyond screening alone. Risk communication and the ongoing management and follow up of high risk patients are central to the programme. Maximising adherence to primary prevention medications and lifestyle advice is likely to be challenging, particularly in deprived areas, because many high risk patients will be men with no associated morbidity and poor health literacy.530

Findings provide a snapshot of recording before the introduction of the NHS Health Check. Levels of recording found here may be similar to other urban, culturally diverse areas, with a high CVD burden but may differ to other areas. The UK has considerable inequalities in cardiovascular disease outcomes between ethnic and socio-economic groups. My findings suggest that while risk factor recording was higher in south Asian patients than whites, it was lower in patients attending practices in more deprived areas than those in affluent areas. It will be important to ensure the Health Check programme addresses this inequality by achieving high coverage within target populations, particularly in deprived communities. Low recording of ethnicity in certain general practices, despite financial incentives in the QOF and a national Directly Enhanced Service, needs to be addressed to ensure that commissioners are able to monitor whether local programmes are delivered equitably.

National monitoring of the Health Check programme may be problematic in the short term because although the Department of Health requires that all adults aged 40 to 74 years are invited for a minimum battery of tests by 2013, PCTs have been granted considerable autonomy to determine their own timetable for programme implementation and which population groups are prioritised for screening locally.

Conclusions

Recording of risk factors for CVD has improved considerably in UK primary care among patients with established chronic diseases. However, there remain considerable gaps in risk factor recording required for the NHS Health Check programme among patients without CVD and diabetes and marked variations in recording between practices and between age, gender, ethnic and socio-economic groups.
Chapter 11; Uptake of NHS Health Check programme in a deprived, culturally diverse setting: cross sectional study

11.1 Introduction

Health policy in the UK has often emphasised high risk rather than population approaches to prevention, despite evidence that a combination of strategies are required to reduce the burden of CVD. This is reflected in the NSFs for CHD\textsuperscript{123} and Older People,\textsuperscript{631} the increased investment in secondary prevention in primary care through the 2004 General Practitioner contract and the NHS Health Check programme.

The success of the Health Check programme, as with all high risk prevention, is strongly reliant on high uptake. Cost effectiveness modelling assumes a 75 percent uptake will be achieved. Evidence from established preventative services in the UK (Chapter 5), suggest that uptake may be lower among socio-economically deprived, and possibly ethnic minority groups, particularly in the early stages of programme development.\textsuperscript{537} Evidence from the more recently introduced colorectal suggests lower uptake among male patients.\textsuperscript{469}

I aimed to examine the uptake of the Health Check programme in the first year of implementation and explore whether participation in the programme differed with patient and practice characteristics. A secondary aim was to examine prescribing of lipid lowering medication among individuals identified to be at high risk of a future cardiovascular event.

11.2 Methods

Data

I obtained data on patients estimated to be high risk of developing CVD, the primary prevention registers, from 29 of the 86 general practices in Ealing, representing some 57,240 out of a target population of approximately 163,000. The programme and data are more fully
described in Chapter 2.2.1. Briefly, the Oberoi software extracted CVD risk factor data from patient EMRs of all patients aged 35 to 74 years without CVD (CHD, stroke/TIA) or diabetes. The JBS2 risk score\(^6^0\) was applied to the data, as recommended at the time by national clinical guidance, and those at greater than or equal to 20 percent risk were placed on primary prevention registers and invited for a Health Check between 1\(^{st}\) September 2008 and 31\(^{st}\) August 2009.

In October 2009, after the first year of programme, practices produced a list of patients who attended. I matched these records to primary prevention registers to determine programme uptake. I added further general practice level data; practice list size and number of general practitioners per practice, and a variable representing the proportion of south Asian patients registered per practice (≥/<50\%). Webb et al.\(^5^2^5\) found south Asian patients had improved cervical screening uptake when not the minority group in a practice. With a large south Asian population in Ealing I aimed to investigate this relationship in the Health Check programme.

As discussed (Chapter 5.2.7), transience is an important factor in screening uptake. I was unable to obtain a direct measure of list turnover in practices. As proxy I generated a variable comprising of the difference in practice list size between 2008 and 2009. I split these into the following three categories based on the distribution of the sample; an absolute decrease in list size; a rise below 5 percent and a rise greater than or equal to 5 percent.

The uptake of cervical screening and primary immunisations in children can be used as markers of general practice quality.\(^6^3^2\) High cervical screening uptake, for example, is found in more organised practices which have better maintained practice registers and more practice nurses.\(^5^3^7\) I included the percentage uptake in practices of both cervical screening and primary immunisations in 2008-9 in the analysis.

Area level deprivation can impact on health above and beyond individual measures (Chapter 5.2.2). I generated a general practice level measure of deprivation, analogous to the method described by Strong et al.\(^6^2^2\) For each practice I took the individual IMD scores for the
population aged 35 to 74, and being non-normally distributed took the median score to represent the practice. For analysis I separated these by national quintiles.

Additional data extractions were undertaken in 16 out of the 29 practices during January 2010. I assessed the clinical measurements taken during the Health Check and the impact of the programme on prescribing of lipid lowering drugs. All additional data were considered valid if recorded on the date of Health Check attendance, or later.

My primary outcome measure was attendance for screening and secondary outcome measure the prescription of a statin. Predictor variables included age, sex, ethnicity, deprivation, smoking and hypertensive status, and practice list size.

Analysis

I generated categorical variables for all explanatory variables (Table 11-1). I assessed levels of attendance in subgroups and used two-tailed z-tests for proportions to test differences in levels of attendance. I assessed the univariate relationship of each independent variable and screening uptake, using chi squared tests for categorical and logistic regression models for continuous data, and reported the p-value for each.

I used multi-level logistic regression to analyse Health Check attendance, building mixed models with patient variables at level 1 and practice at level 2. I included variables listed above, plus the proportion of south Asian patients, number of general practitioners per practice and baseline CVD risk score of the patient. First, I tested each variable for whether a naïve (no level 2 structure), random effect or random slope model fitted best. Each variable was entered individually into the model with the dependent variable under each model structure. Using the AIC, the lowest showing improved fit. I selected the most suitable structure to be put forward for model building; ethnicity was modelled with a random slope, with the remaining variables as random effects.

I built two models, firstly using individual level variables and secondly adding in the practice level measures. In addition to the variables described above I included interaction terms in
Chapter 11; Uptake of NHS Health Check programme

the model building: between age and sex; ethnicity and sex (both found more widely in healthcare\textsuperscript{555 556}); the proportion of south Asian patients per practice and ethnicity;\textsuperscript{522} as well as smoking with both age and sex. Model selection using automated or mechanistic model selection processes have been criticised,\textsuperscript{533} especially when models are not hypothesis driven such as here. I therefore entered all a variables into the model, barring the interaction terms which were tested for inclusion using log-likelihood tests.

Goodness of fit was assessed using a Hosmer- Lemeshow Chi squared test, a p-value of <0.05 showing poor fit, by plotting the receiver operator characteristic (ROC) curve and assessing the c-statistic. I examined poor fitting models using their standardised Pearson and Pregibon leverage residuals.\textsuperscript{634} I calculated the median odds ratio (MOR)\textsuperscript{535} which transposes the variance of the random effects onto the scale of the odds ratio. The MOR is analogous to the median change in odds of attendance if a patient were to move between practices at random. Further, I present the variance partitioning coefficient (VPC), to demonstrate the amount of model variance held at the practice level.

In the subset of 16 practices with data obtained after year one I calculated mean values for cardiovascular risk factors and the sensitivity of using incomplete baseline data to risk stratify patients. I examined changes in statin prescribing in patients at low and high risk of developing CVD based on complete risk factor data. I assessed the sensitivity of the risk score and statin prescribing in different demographic groups. All analyses were conducted using Stata version 11.0 SE. Ethical approval for the study was granted from the London Research Ethics Committee.

11.3 Results

In the 29 practices, 5,294 patients aged 35 to 74 years with an estimated cardiovascular risk \( \geq 20 \) percent were invited for a Health Check, out of a total population of 57,240. Table 11-1 displays the summary characteristics of the population invited and attending a Health Check.
As anticipated for a sample at high risk of developing CVD, the population was a largely older, male with high smoking and hypertension prevalence.

44.8% of patients invited for a Health Check during 2008/09 attended, with considerable variation by patient and practice characteristics (Table 11-1). Attendance was significantly lower among younger patients (41.0% in 35-54), and smokers (40.1%); whilst significantly higher among patients from south Asian (53.0%) or mixed (57.8%) ethnic backgrounds, those with diagnosed hypertension, and patients registered with smaller practices (61.6, 44.9, 37.1% for <3000, 3000-5999 and ≥6000 respectively. Many variables have a univariate relationship with screening uptake (Table 11-2).
Table 11-2; the significance of univariate relationship between screening uptake and independent variables

<table>
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<tr>
<th>Variable</th>
<th>p value</th>
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</tr>
<tr>
<td>Sex</td>
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<tr>
<td>Ethnicity</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Smoking status</td>
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<tr>
<td>Deprivation</td>
<td>0.001</td>
</tr>
<tr>
<td>Hypertension</td>
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</tr>
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<td>Baseline CVD risk score*</td>
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<td>Practice</td>
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<tr>
<td>Proportion south Asian</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>List size</td>
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<td>Single handed</td>
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<td>Primary immunisation uptake*</td>
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<tr>
<td>Cervical screening uptake*</td>
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<tr>
<td>Deprivation</td>
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<tr>
<td>List inflation</td>
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</table>

* Continuous data tested using univariate logistic regression, all other variables use Chi² tests

Variation in uptake

Both models (Table 11-3) had a satisfactory goodness of fit, (Hosmer-Lemeshow p ≤0.05). The model with patient level variables alone had better model fit, as demonstrated by the likelihood ratio test p-value of 0.193. Age significantly predicted attendance in men (AOR 1.33 [1.08-1.63] aged 65-74 compared with 35-54), but there was no impact of age in women. Likewise women were more likely to attend in the 35-54 year old age groups (AOR=1.69 [1.02-2.80]). South Asian (AOR 1.80 [1.40-2.42]), black (AOR 1.59 [1.09-2.32]) and patients of mixed ethnicity (AOR=3.08 [1.91- 4.96]) were significantly more likely to attend than white; as were those diagnosed with hypertension. Women who smoked were less likely to attend (AOR=0.66 [0.48-0.91]), although smoking had no effect in men. Patient level deprivation was not found to predict Health Check uptake. In model 2, the only
Table 11-3: Associations between patient and practice characteristics and Health Check attendance; multivariate analysis

<table>
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<tr>
<th>Fixed effects</th>
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<td>Male 55-64</td>
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<td>1.12-1.64</td>
<td>1.36</td>
<td>0.002</td>
<td>1.12-1.64</td>
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<tr>
<td>Male 65-74</td>
<td>1.33</td>
<td>0.007</td>
<td>1.08-1.63</td>
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<tr>
<td>Female 55-64</td>
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<td>0.907</td>
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<td>(PCT fifth where 1 is the</td>
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<td>most deprived)</td>
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<td>1 most deprived</td>
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<td>1.21</td>
<td>0.170</td>
<td>0.92-1.59</td>
</tr>
<tr>
<td>5 least deprived</td>
<td>1.18</td>
<td>0.651</td>
<td>0.58-2.41</td>
<td>1.17</td>
<td>0.670</td>
<td>0.57-2.39</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>(with age interaction)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Male 35-54</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
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</tr>
<tr>
<td>Male 55-64</td>
<td>1.69</td>
<td>0.042</td>
<td>1.02-2.80</td>
<td>1.68</td>
<td>0.044</td>
<td>1.01-2.79</td>
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<tr>
<td>Female 55-64</td>
<td>1.21</td>
<td>0.223</td>
<td>0.89-1.66</td>
<td>1.20</td>
<td>0.243</td>
<td>0.88-1.65</td>
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<tr>
<td>Male 65-74</td>
<td>0.96</td>
<td>0.736</td>
<td>0.76-1.21</td>
<td>0.96</td>
<td>0.711</td>
<td>0.76-1.21</td>
</tr>
<tr>
<td>Female 65-74</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>(with sex interaction)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male No</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male Yes</td>
<td>0.91</td>
<td>0.23</td>
<td>0.78-1.06</td>
<td>0.91</td>
<td>0.246</td>
<td>0.78-1.07</td>
</tr>
<tr>
<td>Female No</td>
<td>1</td>
<td></td>
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<td>1</td>
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<td></td>
</tr>
<tr>
<td>Female Yes</td>
<td>0.66</td>
<td>0.011</td>
<td>0.48-0.91</td>
<td>0.67</td>
<td>0.014</td>
<td>0.49-0.92</td>
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<td>Hypertension</td>
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</tr>
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<td></td>
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</tr>
<tr>
<td>Yes</td>
<td>1.30</td>
<td>&lt;0.001</td>
<td>1.13-1.49</td>
<td>1.30</td>
<td>&lt;0.001</td>
<td>1.13-1.49</td>
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<tr>
<td>Table 11-3 continued</td>
<td>Model 1</td>
<td>Model 2</td>
<td></td>
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<tr>
<td>----------------------</td>
<td>---------</td>
<td>---------</td>
<td></td>
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</tr>
<tr>
<td>Practice list size</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;3000</td>
<td>-</td>
<td>5.10</td>
<td>0.019</td>
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</tr>
<tr>
<td>3000-5999</td>
<td>-</td>
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<td></td>
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<td></td>
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<td>≥6000</td>
<td>-</td>
<td>1.22</td>
<td>0.688</td>
<td>0.46-3.22</td>
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<td>Single handed practice</td>
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</tr>
<tr>
<td>Yes</td>
<td>-</td>
<td>0.63</td>
<td>0.442</td>
<td>0.19-2.06</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>-</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practice deprivation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>-</td>
<td>0.72</td>
<td>0.756</td>
<td>0.09-5.88</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>-</td>
<td>1.12</td>
<td>0.861</td>
<td>0.30-4.18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proportion of the practice south Asian</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;50%</td>
<td>-</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥50%</td>
<td>-</td>
<td>1.50</td>
<td>0.434</td>
<td>0.54-4.12</td>
<td></td>
<td></td>
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<tr>
<td>List inflation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;0%</td>
<td>-</td>
<td>0.39</td>
<td>0.053</td>
<td>0.15-1.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-5%</td>
<td>-</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥5%</td>
<td>-</td>
<td>0.62</td>
<td>0.257</td>
<td>0.27-1.42</td>
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<td></td>
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<td>Cervical Screening</td>
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</tr>
<tr>
<td>Primary IMMs</td>
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<td>0.97</td>
<td>0.539</td>
<td>0.86-1.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practice list size</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Random Effects</td>
<td>Var</td>
<td>(SE)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Practice intercept</td>
<td>1.75</td>
<td>0.553</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>MOR</td>
<td>3.52</td>
<td>2.69</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethnicity (slope)</td>
<td>0.06</td>
<td>0.02</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VPC (p)</td>
<td>0.347</td>
<td>0.248</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VPC</td>
<td></td>
<td>0.347</td>
<td>0.248</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Model Fit</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Hosmer Lemeshow p-</td>
<td></td>
</tr>
<tr>
<td>value</td>
<td>0.019</td>
</tr>
<tr>
<td>AIC</td>
<td>6305.4</td>
</tr>
<tr>
<td>ROC c-statistic</td>
<td>0.762</td>
</tr>
</tbody>
</table>

\[ ^a \text{VPC}= \text{variance partitioning coefficient} \]

\[ ^b \text{Note: Odds ratios are adjusted for all variables in the Table} \]
practice level variable found to be significantly associated with Health Check uptake was practice size; practices with the smallest list sizes (<3,000) had significantly higher attendance than larger practice (AOR= 5.10 [1.31-19.88] compared with a list size of 3,000-5,999). Practices with a reduction in list size between 2008 and 2009 were of borderline significance of having lower uptake.

There was considerable variance in attendance between practices. The MOR was large, 3.52 when not controlling for practice level factors and 2.69 when controlling for them, demonstrating that if moving between practices there was a large odds of change in uptake. From the variance partitioning coefficient, controlling for the practice level factors available, 25% total variance in uptake is due to unexplained practice level factors.

**Risk factor levels after Health Check attendance**

Table 11-4 presents a summary of four major CVD risk factors in attendees. 50.6% of patients had a blood pressure of 140/90 mm Hg or greater, with 31.6% prescribed anti-hypertensive therapy, 66.5% had a total cholesterol of 5 mmol/L or greater, and 26.0% had a BMI of 30 kg/m² or greater. Diagnosed hypertension rose from 32.0% to 39.2%.

**Table 11-4: Cardiovascular risk factors† in patients who attended a NHS Health Check (n=1,033)**

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>Mean systolic BP</th>
<th>138.0 [137.1-139.0]</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean diastolic BP</td>
<td>80.9 [80.3-81.5]</td>
</tr>
<tr>
<td>BP&gt; 140/90</td>
<td></td>
<td>50.6% (43.2%‡)</td>
</tr>
<tr>
<td>Diagnosed Hypertension</td>
<td>39.2%</td>
<td></td>
</tr>
<tr>
<td>Prescribed Anti-Hypertensive</td>
<td>31.6%</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lipids</th>
<th>Mean</th>
<th>5.27 [5.20-5.34]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol ≥ 6 mmol/l</td>
<td>36.9%</td>
<td></td>
</tr>
<tr>
<td>Cholesterol ≥ 5 mmol/l</td>
<td>66.5%</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>BMI</th>
<th>Mean</th>
<th>27.6 [27.3-27.9]</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI ≥ 30 kg/m²</td>
<td>26.0%</td>
<td></td>
</tr>
<tr>
<td>BMI ≥ 25 kg/m³</td>
<td>69.2%</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Smoking</th>
<th>Prevalence</th>
<th>37.1%</th>
</tr>
</thead>
</table>

†Within the last two years
‡ In those without diagnosed hypertension
Of the patients designated at high risk from pre-existing EMR data, 74.5% were confirmed to have a ≥20% risk score after their risk factor data were completed during the Check. The predictive accuracy varied by sub-group; with south Asian (82.4% [p=0.003]); the oldest (65-74: 82.4% [p=0.009]); and most deprived (88.0% [p<0.001]) having most accurate, whilst white patients (67.2% [p=0.022]), and women (61.7% [p<0.001]) had poorer prediction.

Figure 11-1 shows statin prescribing in patients who received a Health Check. Before the health check, 24.9% were prescribed statins (Table 11-5) a further 1.8% had a valid exclusion in their EMR- these were either drug contraindications or refusal of therapy. After the intervention, 43.4% were prescribed statins, with 6.1% excluded; a relative increase of 74%. Statin prescribing increased from 27.0% to 39.6% in patients assessed to be at low risk of CVD as a result of the Health Check. In those low risk patients prescribed statins after the Health Check the mean total cholesterol was 4.78 (4.52-5.03); 75 (72.1%) had a total cholesterol greater than 4 mmol/L, 39 (37.5%) greater than 5 mmol/L and 59 (56.7%) were diagnosed with hypertension. There was variation between subgroups prescribing (Table 11-5). South Asian patients, women, and those at the highest risk saw markedly larger increases in statin prescribing, while white patients the lowest increase.
Table 11-5: Differences in statin prescribing before and after the Health Check in patient subgroups

<table>
<thead>
<tr>
<th></th>
<th>Statin prescription</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre Health Check</td>
<td>Post Health Check</td>
<td>Relative increase</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>21.1%</td>
<td>42.5%</td>
<td>2.01</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>25.4%</td>
<td>57.9%</td>
<td>2.28</td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>23.0%</td>
<td>33.4%</td>
<td>1.45</td>
<td></td>
</tr>
<tr>
<td>South Asian</td>
<td>24.9%</td>
<td>54.0%</td>
<td>2.17</td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>29.4%</td>
<td>47.1%</td>
<td>1.60</td>
<td></td>
</tr>
<tr>
<td>Mixed</td>
<td>25.5%</td>
<td>46.0%</td>
<td>1.80</td>
<td></td>
</tr>
<tr>
<td>Deprivation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>third†</td>
<td>1 deprived</td>
<td>22.0%</td>
<td>40.1%</td>
<td>1.82</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>26.3%</td>
<td>46.8%</td>
<td>1.78</td>
</tr>
<tr>
<td></td>
<td>3 affluent</td>
<td>27.3%</td>
<td>48.9%</td>
<td>1.79</td>
</tr>
<tr>
<td>CVD Risk</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20%</td>
<td>27.0%</td>
<td>39.5%</td>
<td>1.46</td>
<td></td>
</tr>
<tr>
<td>20-29.9%</td>
<td>23.1%</td>
<td>41.9%</td>
<td>1.81</td>
<td></td>
</tr>
<tr>
<td>30-39.9%</td>
<td>29.9%</td>
<td>47.3%</td>
<td>1.58</td>
<td></td>
</tr>
<tr>
<td>≥40%</td>
<td>22.4%</td>
<td>53.3%</td>
<td>2.38</td>
<td></td>
</tr>
</tbody>
</table>

†Local third, where 1 = most deprived

11.4 Discussion

Main findings

Uptake of the NHS Health Check programme was lower (45%) than predicted in Department of Health modelling (75%) and was significantly lower among younger men, and female smokers but higher among patients from south Asian or mixed ethnic backgrounds, those with diagnosed hypertension, and patients registered with smaller practices. The percentage of patients confirmed to be at high risk of CVD who were prescribed a statin increased from 25 percent to 45 percent, compared with the Department of Health estimate of 85 percent statin coverage after the Health Check.

What is already known?

Few countries have introduced large scale cardiovascular risk assessment programmes and literature evaluating the performance of such schemes in routine care settings is sparse. Evaluations of earlier local CVD risk assessment programmes in the UK have found uptake from 29 to 66 percent, with lower participation among the materially deprived, men, and smokers (Chapter 5.3.2). A recent screening project, before the Health Check, based across
general practice and pharmacy found a 24.3 percent screening attendance, with attendance higher in south Asian, older patients and non-smokers. In the OXCHECK trial, there was poor uptake in deprived patients, and women. However an evaluation of a pilot CVD risk screening programme in Stockport identified only modest differences in uptake (solely in men) by deprivation group. Horgan et al. assessed the risk status of patients given a health check analogous to, but again predating the national programme, carried out in pharmacies. The population screened had relatively low levels of risk, although screening did not target those at high risk.

*What this study adds*

My finding of higher attendance in south Asian patients contrasts previous research which has found lower uptake of preventative services when compared with white British. This may be because the Health Check programme is more firmly rooted in general practice, where patients of south Asian origin can have a higher attendance. A recent general practice based project also found improved uptake in both south Asian and Black patients. Another possible explanation is that Ealing has many general practitioners of south Asian origin and there is often cultural concordance between patient and physician. Cultural concordance may improve patient satisfaction and make consultations more participatory for the patient. This may improve attendance; however concordance may not impact quality clinical care.

Patients in practices with the smallest list sizes saw high rates of attendance. Practices with smaller list sizes have greater perceived physician availability, and longer consultation time, these can both improve patient satisfaction and in turn compliance with interventions. Lambert et al. found improved uptake of CVD risk assessments in single-handed practices, not having controlled for list size. They hypothesise that the funding structure, as here a LES, was more beneficial to single handed practices. Compared with the global budget of the practice, they had a greater relative gain from the LES payment than a multi-
GP practice. Likewise, smaller practices could gain relatively more from LES payments, therefore making a greater effort in screening.

In the youngest age group, women were more likely to attend a Health Check than men. This may be because younger women have greater health care utilisation, through consultation for sexual and reproductive health needs. In men, older patients had highest attendance; older patients have high attendance at general practice and have a greater risk of vascular disease.

A large proportion of patients were confirmed to be at high risk after risk factor assessment. Previous modelling and analysis suggest estimation of vascular risk using existing medical record data is efficient. The accuracy of risk prediction improved in south Asian, older patients, and the most deprived; groups with the most complete data (Chapter 10), more complete data improve prediction. However, risk prediction was incorrect in 33 percent of the white population and 38 percent of women.

The population, although estimated to be at high risk, did not have especially high levels of some risk factors; for example the mean blood pressure was 138/81 mmHg. Cardiovascular risk scores capture universal risk, not relying on individual risk factors. Current guidelines for vascular risk management utilise universal risk, which is more effective than reliance on single risk factors. The JBS2 risk score uses a risk multiplier for patients of south Asian origin, the majority in the population, which further increases the risk score (Chapter 7).

Although prescribing of statins appeared to increase as a result of the Health Check programme, only 45 percent of patients confirmed to be at high risk of CVD were prescribed a statin at the end of the study period. Given the population included patients already taking statins and diagnosed with hypertension before the Health Check, prescribing may be lower when targeting patients with no previous vascular management. The low rate of prescribing may be due to a number of factors, including patient and practitioner beliefs about the risks, and benefits of prescribing medications in persons without established disease. The
prescription rate was higher in south Asian patients, possibly due to their greater perceived risk of cardiovascular disease by GPs and patients, or greater willingness to accept prescribed medication. Prescribing was lower in the most deprived. The increase in statin prescribing seen in patients identified at low CVD risk is of concern and suggests that programmes should be monitored for inappropriate prescribing.

**Strengths and limitations**

This study is the first to examine the uptake of the Health Check programme in England. I used patient level data, derived from patients’ EMRs in general practice and used methods appropriate to nested data. Although the population studied is not representative of the whole country, this evaluation is important given that the overall effectiveness of the programme and its impact on inequalities in cardiovascular health will be largely determined by attendance in deprived areas. Further, little national data are available to evaluate the programme, especially at a patient level, making local evaluation vital.

I was only able to obtain data from 29 of the 86 practices in Ealing due to incompatible software and low data returns. However, the demographic profile of patients participating was similar to those in Ealing as a whole. I presented data on statin prescribing, but could not assess whether prescriptions were collected by patients or examine drug adherence. Medication use carries a perception of adverse outcomes and patients may be unclear about benefits of medication in the absence of a diagnosed condition. Previous studies have suggested the one-year adherence rate to statins for primary prevention of CVD was 46 percent, and can fall to 35 percent after three years. I was unable to determine whether patient’s received and adhered to lifestyle interventions, including weight management, and exercise promotion that might be more appropriate than pharmacological interventions for primary prevention.

The data did not cover all patients eligible for a Health Check, rather focusing on those estimated to be at high risk of CVD. The uptake in the remaining population may differ, although it is unclear how. Being labelled as high risk might motivate patients to attend.
conversely membership of the high risk group might be associated with poor health behaviours and low attendance.\textsuperscript{574} I did not have access to data on consultations and was unable to determine the extent to which Health Checks were carried out opportunistically.

Ethnicity data were missing in 30 percent of the sample which may be a source of bias. The Health Check offered in Ealing differs slightly from national guidance, with the inclusion of a younger age group and patients diagnosed with hypertension and prescribed statins. Regression analysis controlled for hypertension and I have presented baseline statin prescribing and hypertension prevalence. The younger age group might in theory be a weakness because one might assume the 35 to 39 year age group to have especially low attendance. This, however, will not be a problem. As an artefact of the risk stratification process, in the first year (high risk) data analysed, there were only 16 patients aged 35 to 39.

The transience of the population has strong founding to be important for screening uptake (Chapter 5.2.7). Principally, transience leads to inaccurate practice registers through list inflation, which makes it difficult to achieve strong uptake. I did not have access to data on patient registration and deregistration in practices, and hence had to use what is likely to be a weak proxy measure of transience. Finally, the measure of social deprivation, found not to influence attendance, was an area level measure from the patient’s postcode. I described above (Chapter 5.2.2) how individual elements of SEP can impact upon health behaviours, however, did not have access to any such measures. The study setting further contained few affluent areas, so findings that attendance did not vary with socio-economic status should be interpreted with some caution.

\textit{Implications for policy}

Despite the coalition government’s apparent commitment to the NHS Health Check, many areas appear likely to withdraw direct funding for the service.\textsuperscript{643} The universal nature of the programme has been criticised,\textsuperscript{432} \textsuperscript{644} and the programme has a number of barriers to overcome to be effective.\textsuperscript{599} In terms of policy options, it is not the only choice available.\textsuperscript{644}
Work load implications of NHS Health Check for primary care are considerable and the cost-effectiveness of the programme has been questioned.\textsuperscript{94, 427} Modelling undertaken by the Department of Health was based on a number of key assumptions; 75 percent attendance (based on uptake of the NHS Breast Screening Programme), 85 percent of high risk patients will take up statins and 70 percent adherence in those patients prescribed. The findings of 45 percent attendance and 43 percent take up of statins in high risk individuals suggest that these assumptions may have been over-optimistic. Previous research suggests that adherence to statins for primary prevention at two years was 46 percent.\textsuperscript{74}

Options include maintaining current mass screening; my findings suggest that current programme goals of 75 percent attendance and high adherence to lifestyle and therapeutic interventions are probably unachievable without additional resources to primary care. I build on the well resourced OXCHECK and British Family Heart Study trials which achieved 80 and 73 percent uptake respectively, compared with the 45 percent found here.\textsuperscript{152}

A second option is to adopt a targeted screening approach; several studies have demonstrated targeted screening approaches are likely to be more cost-effective than mass screening.\textsuperscript{430, 431} Options include targeting the programme in areas with high CVD prevalence, in individuals estimated to be high risk from existing clinical data and/or individuals with familial risk.\textsuperscript{645} This study provides further evidence that the use of existing medical record data could be efficient in targeting high risk groups for intervention. Targeting limited resources to increase uptake, improve risk communication, and adherence to interventions in high risk populations is likely to increase the cost-effectiveness and population benefits of this programme.

A final option is to reinvest resources into population wide strategies to increase physical activity, and reduce smoking, salt\textsuperscript{87}, and transfat intake.\textsuperscript{646} Estimates from observational and randomised trial data suggest lowering population level blood pressure and cholesterol by 10 percent would reduce CVD events by 45 percent compared with a 34 percent reduction associated with aggressive pharmacological treatment (with statins, beta blockers, ace
inhibitors, and aspirin) of individuals with a 20 percent risk of a CVD event in the next ten years.\textsuperscript{76} Population approaches may not only be effective, but might prove cost saving.\textsuperscript{317}

Despite low uptake overall, I found no evidence of poorer uptake among deprived and ethnic minority groups. However, there was considerable variation in attendance by practice and attendance was significantly lower among younger men and female smokers. Ongoing local and national monitoring and evaluation is, therefore, essential to ensure the programme is delivered equitably.

\textit{Conclusions}

Uptake of the NHS Health Check programme in Ealing was considerably lower than projected, despite financial incentive for general practices. Targeting limited resources to increase uptake, improve risk communication and adherence to interventions in high risk populations may be more cost effective and increase population gain from the programme. However, a wider question remains about whether £250 million annual costs would be better spent on population-wide strategies to reduce obesity, smoking and improve diet.
Chapter 12; Discussion and Conclusions

12.1 Introduction

The aging population in many high-income countries makes the primary prevention of CVD of paramount importance to the financial stability of health systems. My thesis sets out to explore and to evaluate the English NHS Health Check programme, during its early stages of implementation. I focus on a number of key areas of the programme; namely the work load for general practice, early uptake of the programme, the choice of CVD risk score and the programme’s potential impact on health inequalities. From reviewing the literature on the primary prevention of CVD, a number of concerns arise over the programme. From my findings and through the review of the literature, I further explore the feasibility of implementing alternate policy approaches. These included a more targeted high risk approach or population-wide methods of prevention.

12.2 Summary of main findings

Work load

Guidance for the NHS Health programme requires the recording of a range of demographic and CVD risk factor data for all attendees. Strictly speaking, the guidance requires the *de novo* recording of all data. My findings, however, demonstrate that patients have a substantial amount of pre-existing CVD risk factor data in their EMRs, much of which was recently recorded. For example, 96 percent of patients have a record of smoking status, 86 percent blood pressure and 73 percent BMI recorded within the last five years, and 69 percent of patient blood pressure from within the last two. The recording of total cholesterol was significantly lower, with 56 percent in the previous five years.

A relaxation of the programme regarding the *de novo* recording of data will produce a smaller work load for providers. Even this scenario, however, would generate a substantial work
load. The average English general practice, for example, would have to undertake an additional 356 blood pressure, 721 BMI and 1,018 cholesterol measurements in order to achieve the complete recording of CVD risk factors in the population eligible for the programme.

At a national level, I estimate, using the QRISK2 risk score, 2.0 million patients to be at high risk of CVD in England (greater than or equal to 20 percent risk of a CVD event in 10 years). This equates to over 10 percent of the population eligible for the Health Check programme. At the general practice level, this translates into a median of 206 patients, with a maximum of 1,693 in England. Using the JBS2 score there were 4.3 million at high risk. The work load will have considerable cost implications for the NHS. Using QRISK2, the risk assessment in the high risk population will cost £31 million, with an additional £176 million for managing risk factors. Total costs using JBS2 will be nearly double at £378 million, with £66 million for screening.

Within the population identified to be at high risk of CVD, there is a high prevalence of CVD risk factors. My modelling, using national survey data, estimates that within the high risk population, 83 percent are physically inactive, 65 percent obese, 24 percent have undiagnosed hypertension and 114,400 have undiagnosed diabetes. Using local data from NHS Ealing, there was also evidence of a large follow-up work load from the programme (stemming from CVD risk factors). In the high risk cohort attending the Health Check, 43 percent (excluding diagnosed hypertensive patients) had a blood pressure record greater than 140/90 mmHg; 37 percent cholesterol over 6 mmol/l; 69 percent were obese and 37 percent smoke. Within the high risk population identified by each CVD risk score, JBS2 and QRISK2, the profiles of CVD risk factors differ.

There are geographic inequalities in prevalence of high risk status, hence in the work load created by the programme. In general, there is a greater prevalence of high risk status in the industrial centres in the North of England, and lower prevalence in the South, especially surrounding the Home Counties. This pattern was broadly similar using the JBS2 and
QRISK2 risk scores; however prevalence did significantly vary nationally at both the general practice and PCT level.

**Programme effectiveness**

The overall uptake of Health Checks during the first year in Ealing was 45 percent. This was lower in a number of socio-demographic groups, for example 41 percent in those aged 35 to 54 years, 40 percent in smokers, 38 in patients with no recorded ethnicity and 37 percent in the largest general practices (with greater than 6,000 patients). One year after a NHS Health Check, prescribing of statins in eligible patients increased from 25 to 43 percent; a relative increase of 1.74. Post-Health Check, prescribing was lower in some groups, for example reaching only 33 percent in patients of a white ethnic background and 40 percent in the most deprived. In patients discovered not to be eligible for statin prescriptions after a Health Check, there was also an increase in statin prescribing, from 27.0 to 39.6 percent; a relative increase of 1.46.

**Inequalities**

Over the course of my work, from prior to the Programme into its earliest stages, I discovered a number of inequalities in care and service provision. Before the Health Check, the completeness of CVD risk factor data in EMRs varied by patient and practice characteristics. Hypertensive status was the single greatest predictor of risk factor recording. There was increased blood pressure recording in south Asian patients (compared with white) and poorest recording of all risk factors in those with no ethnicity record. There was poor risk factor recording in deprived practices, and better in larger practices. Finally there was greater recording in women and in older patients.

Health Check uptake was higher in older men, but, there was no effect of age in women. Similarly, there was greater uptake in women than men in the youngest age group. South Asian, black and mixed ethnic groups had higher attendance than white patients, as did hypertensive patients. There was poorer uptake in women who smoke, but not in men. One
year after the NHS Health Check, statin prescribing had seen significantly greater increases in south Asian patients and women.

There were marked differences in the completeness of risk factor data and Health Check uptake between general practices. The recording of ethnicity, for example, varied between 10 and 96 percent, and HDL between 31 and 77. For blood pressure, total cholesterol, and BMI recording, the patient and practice characteristics controlled for in my analyses accounted for the majority of practice variation. 22 percent of the total variation in smoking status records, however, remained unexplained at the general practice level. Variation in screening uptake was not explained by patient and practice characteristics. The MOR at the practice level was 2.69. In moving randomly from one general practice to any other with a higher uptake, one can expect an odds ratio of 2.69 for the change in screening uptake (the median of all the potential moves). Overall, 25 percent of the total unexplained variance was at the general practice level.

*Improving risk prediction*

The Department of Health permit the use of two CVD risk scores for the Health Check programme, QRISK2 and JBS2. In line with much previous evidence, JBS2, an Anderson-Framingham based risk score, gives a higher estimation of risk than QRISK2 overall. There were higher mean risk scores in all, and up to a twofold larger number at high risk in, social groups studied using JBS2 and QRISK2 in NHS Ealing and nationally representative survey data. The main exception was black women who, despite a lower mean QRISK2 score, had a larger proportion at high risk using QRISK2.

The choice of risk score will potentially have an important role in the programme’s impact on health inequalities. Although QRISK2 gives universally lower estimates of CVD risk, my findings suggest this difference is significantly greater in south Asian men. The difference in proportion of the population designated as at high risk using JBS2 and QRISK2 is 19 percent in south Asian, but only 9 percent in white. If changing between risk scores, there is greater reclassification (between high and low/moderate risk) in south Asian men. 43 percent of the
high risk JBS2 group would be downgraded in risk status using QRISK2 in south Asian men, compared with 28 percent in white.

At a general practice level, the increased risk estimates in south Asian men produce significant differences in work load, dependent upon the ethnic make-up of its population. Practices with the largest south Asian population, have the greatest work surplus created by JBS2. A second inequality results from the risk factor profile of the high risk populations defined by each risk score. Blood pressure levels, undiagnosed hypertension and smoking are greater using JBS2, with diabetes and obesity greater using QRISK2. A high risk population identified using JBS2 is on average younger.

A computationally and methodologically simple approach to substituting missing risk factor data produces accurate estimates of CVD risk scores when using incomplete medical record data. Compared with a complex, theoretically more sensitive method (multiple imputation), national default values produce comparable risk scores. In data from Ealing; the estimated risk score and proportion at high risk were lower using multiple imputation but only marginally so. Agreement in high risk classification proved strong.

Using risk stratification prior to the Health Check proved effective in Ealing. Following a Health Check, 75 percent of patients estimated to be at high risk of CVD were confirmed as such after complete data recording. This proved more accurate in a number of social groups. South Asian, the oldest and most deprived patients had improved risk prediction. This reflects the more complete medical record data at baseline.

12.3 Strengths, limitations and consideration of the methods

Within each of chapters 7 to 11, I address the limitations of the methods specific to the analyses within each study. In addition to these concerns, here, I shall address the limitations that apply to this body of work as a whole. A large amount of my work uses data
from Ealing, London. This creates two limitations concerning the generalisability of my findings. Firstly, the Health Check offered by NHS Ealing was modified to suit local needs. The main differences with national minimum standards were an extended age range (beginning at 35 years) and the inclusion of some groups exempt from the Health Check programme (hypertensive patients for example). I overcome much of this limitation in the analyses, for example, by using multivariate methods. Also, given the programme’s structure, there is going to be no area in England that offers a *typical* Health Check. The malleability allowed by the Department of Health will create differences in the service; therefore the study of local variation will be vital.

The population in Ealing differs from England as a whole. Most notably it is considerably more ethnically diverse, one of the most diverse in the country. This may limit the application of the results, and my findings might not be valid in areas with different population profiles. Ealing does, however, represent a community with considerable health need. Being highly ethnically diverse, it suffers a disproportionately high burden of CVD. It is especially in areas such as Ealing that the Health Check must be successful if the programme is to reduce the CVD burden and lessen health inequalities.

Any work concerning the NHS Health Check is likely to be of interest globally. This is the first population-wide CVD prevention programme of its type therefore will provide valuable data for other countries. Given this, one must account for aspects of the timing and setting of the programme when considering its implications internationally. The UK health system has strong general practice, with a saturation of EMRs and offers publically-funded universal health care. These all impact on the ability to offer such a screening programme. Indeed, it is unlikely that health systems with weaker primary care or more limited information technology could offer such a programme. Findings may not, for example, be directly transferred to the USA and the *Million Hearts* initiative with the relatively under-developed primary care.

The Health Check in Ealing was, during the time period studied, carried out through general practice. Nationally, the programme can be carried out across a variety of settings. My work
does not address the programme’s implementation in alternate settings where the uptake and programme outcomes may differ. A general practice-centric scheme might fail address hard to reach populations. It will omit patients not registered with general practice, a population with considerable health needs. Both the programme and its evaluation must encompass this group. Despite evidence of the effective involvement of pharmacies in CVD prevention, its implementation can encounter problems. These include information technology deficiencies, a large requirement for training and set-up cost, problems in data transfer and a limited through-flow of patients. These were not addressed here.

My findings are from data from the first year of the programme in Ealing. Uptake may increase as the programme becomes embedded. The early Ealing data also focused on a population estimated to be at high risk, not a strictly general population. However, this population has greatest need and is one critical to the programme’s success. The uptake in the remaining population may differ, although it is unclear how. Being labelled as high risk might motivate patients to attend; conversely membership of the high-risk group might be associated with poor health behaviours and low attendance.

The aim of my thesis was to assess the early implications of the Health Check programme. As such a number of important questions lie outside of the timeframe of my work. I did not have data on the impacts of the programme on CVD risk factors, risk, or endpoints. Nor was I able to assess continued adherence to interventions, or compliance of the high risk population to follow-up in general practice. All of these require data from further into the programme’s implementation. Follow-up data on statin adherence will be vital, as will the uptake of community interventions.

By focusing on the Health Check programme, I did not consider all aspects of CVD prevention. Chronic disease groups and patients aged over 74 are not included in the programme. These high risk groups are covered by separate prevention programmes, including the QOF and have been evaluated elsewhere. It further omits the youngest. There is growing support for early intervention to lower CVD risk. The entire life-course is
implicit in CVD risk.\textsuperscript{475} As a result primordial prevention and a focus on maintaining a favourable CVD risk profile over the entire life course is important.

Strengths of this work include timing. My analysis of Health Check uptake is among the first of its kind, carried out only one year into the programme. It is vital in shaping the programme over the coming years. It clearly documents the importance of uptake; what should be the central area of focus within the programme. Estimating the numbers of patients at high risk of CVD in the UK filled an important gap in data, a gap left by considerable delays in both a national dataset and call-recall system. For the first time, it documents need in the population and outlines who might benefit most from the programme. My work may be useful in the future management of the programme. When comparing risk scores and assessing the high risk population, I use nationally representative data for the Health Survey for England. In the immediate prelude to such a large, national programme it was also important to review the literature surrounding CVD prevention, and to document future research needs.

\textbf{Methodological implications}

Some of the methods that I used within the analysis warrant consideration over their impacts on findings, and I shall discuss the major issues here. My work is heavily reliant on multiple imputation. This is a powerful method to overcome missing data, and can reduce the bias caused by missing data\textsuperscript{586} in order not to introduce bias, however, care is required.

The first consideration is of the number of imputations (m) used. My analyses all use m=10. For each imputation model I tested the power of m=10, given the fraction of missing data (Ɣ) for each imputed variable. To do this, I calculated Ɣ, then compared this with findings published by Graham et al.\textsuperscript{581} For each Ɣ, assuming m=10 I was able to ascertain the loss of power compared with m=100, assumed to be a gold-standard for imputation. I allowed a 5 percent loss of power, and for all imputation models m=10 lay within these bounds. A larger m, may add precision, however this will be minimal and does not want the addition
computational power. Using data from Chapter 10 I reran the imputation using \( m=20 \), and found no significant difference in the risk factor estimates produced.

I used model building, stepwise selection using AIC, for multiple imputation. The alternate, using as many predictors as possible may be superior. As mentioned above, however, I conducted sensitivity analysis using the data from Chapter 10, which also compared full imputation models. This produced no overall significant difference in the imputed risk factors, nor differences within population subgroups. I only imputed clinical risk factor data and smoking, but for disease state risk factors for CVD, such as diagnosed atrial fibrillation, I simply assumed these missing if absent. The risk score, QRISK2, to which I applied the imputed data states in its guidance that such variables should be assumed null if missing. This zero imputation will systematically underestimated the derived risk score; however all the conditions are rare therefore this error will be minimal.

Throughout this work missing ethnicity was a problem with data. This is a widespread problem with health data, a number of methods have been used to deal with it, however with no overall consensus. Frequently missing data are included in the ‘other ethnic group’ category or can be excluded from analysis. When analysing the primary care data from Ealing, I included a specific ‘missing’ ethnicity group. This was for theoretical reasons, although has not been widely used elsewhere. Through the missingness, it is evident patients are infrequent attendees at general practice. Given this strong tie, I assumed although heterogeneous in terms of ethnicity, this is an important subgroup when considering the quality of care received. Finally, in Chapter 8 when directly imputation methods, my simple imputation i.e. cold decking health survey data to fill the missing. This has no great evidence to support its accuracy as a method of imputation \textit{per se}, and I used it as the most simple, practical exemplar of imputation that could be used in practice.
12.4 Implications for policy and practice

Choice of CVD risk score

**Framingham** based CVD risk scores over-estimate risk in many UK populations. My findings replicate this, with higher estimates of risk using JBS2 in both data from Ealing and national survey data. Despite a wealth of evidence of differences in risk estimation, UK national guidelines permit the use of both JBS2 and QRISK2. The initial decision to use JBS2 in the Health Check programme was *in lieu* of evidence that QRISK/QRISK2 have improved risk prediction in the UK and largely for practical reasons. Firstly, QRISK/QRISK2 are not freely available to all. Their use is permitted either within the EMIS clinical system or through an additional license from QRESEARCH. In the UK, NICE will not solely recommend a product or service which requires additional expenditure, therefore cannot solely advocate QRISK2. In addition to this, familiarity with the **Framingham** scores, may also promote their continued use.

JBS2 produces higher mean risk prediction, but with variations in the high risk group by sex and ethnicity. In the UK one of the most apparent cardiovascular inequalities occurs in men from a South Asian ethnic background, who despite displaying fewer risk factors, have a high burden of CVD. JBS2 accounts for this through a simple multiplication factor. My data suggest compared with QRISK2, which directly incorporates ethnicity; JBS2 has a significantly greater difference in risk prediction in South Asian men than in other ethnic groups. I had no data to compare with CVD outcomes, however, if this proves to be an over estimate in risk, this has major implications for the Health Check programme in South Asian men.

Over estimation in CVD risk will expose more South Asian men to the iatrogenic harms of screening including exposure to side-effects from statins and general psychological harms. The multiplication factor used within JBS2 has faced previous criticism. Upon its
initial implementation, there were calls that the factor was lacking in an evidence base and somewhat arbitrary.\textsuperscript{583}

It also equates to substantial practice level differences in workload and cost. Using JBS2, the work load and screening costs (especially from follow-up risk management) will be especially inflated in areas with large south Asian populations. The greater number classified as at high risk will, for example, generate greater numbers eligible for statins. Again, if the risk multiplication is inaccurate, the inflation in cost will be unwarranted and will impact on inequalities in service provision. Raised spending will run parallel to those with the greatest health care need.\textsuperscript{37, 38} Health Checks offered in areas with a large south Asian population might be of lower quality and exacerbate health inequalities.

A greater proportion of black women were designated as at high using QRISK2 in both national and local data. White women have lower cardiovascular risk than black women, with especially strong evidence from the United States.\textsuperscript{39, 131} QRISK, a CVD risk score which incorporates ethnicity, may capture this ethnic variation in risk. If so, QRISK2 will provide a method of CVD risk assessment more sensitive to patient need and more able to reduce health inequalities.

There are concerns surrounding the accuracy of CVD risk prediction in women.\textsuperscript{82} The predictive accuracy of any model improves as the number of outcomes increases. Frequently in the derivation datasets of CVD risk scores there are fewer CVD events in women, therefore risk prediction is poorer.\textsuperscript{381, 382} The larger derivation dataset used for the QRESEARCH risk scores may allow improved risk prediction in women. Also traditional CVD risk factors, for example blood pressure and cholesterol levels, are less important mediators of CVD risk in women.\textsuperscript{82} Framingham based risk scores are more strongly driven by traditional risk factors. A wider range of risk factors, for example BMI influence CVD risk in women,\textsuperscript{131} with obesity affecting both sexes equally.\textsuperscript{549} QRISK2 incorporates BMI as a risk factor for CVD; as a result it may be more able to capture CVD risk in women.
More generally within the NHS Health Check, the use of two CVD risk scores has the potential to generate inequalities in service provision. There is a likelihood of a split in the service between settings. A large proportion of general practices have access to QRISK2. EMIS currently has 53 percent coverage of GP systems in the UK. All of these practices have QRISK2 integrated within their clinical software. Additionally, many PCTs are likely to opt to purchase the relatively inexpensive licence to use QRISK2 across their entire area.

Alternate providers, for example community venues and pharmacies, will have more limited access to QRISK2. The license cost is likely to be more inhibitive for a single user than for a PCT. As a result, alternate providers will be left to use JBS2. Due to its higher estimates of CVD risk, this creates a potential scenario, whereby after a high risk group is defined by an alternate provider; these patients will be sent to general practice, only to be found ineligible for a high risk register using QRISK2. This creates confusion and concern amongst patients, and unnecessary work for general practice. I find this effect will be exacerbated in areas with large south Asian populations. Finally, the two risk scores give a different profile of risk factors in the high risk group. This might affect the planning for and provision of interventions following the Health Check.

The choice of CVD risk score has implications for the NHS Health Check; affecting work load and the potential for ethnic inequalities. Rational consideration of the two risk score also supports QRISK2. It was produced in England and most recently updated in 2011. JBS2, meanwhile, has its origins in a cohort established over 40 years ago in the USA; a time and place with very different CVD risk profiles to contemporary England. The minimum dataset for the Health Check programme requires the recording of patient’s global CVD risk. It does not, however, allow the recording of the risk score used. Given the enormous disparities in predicted risk this, at the very least; within the current programme the risk score used must be recorded. Findings presented here, in addition to evidence of improved risk prediction, suggest that efforts must be made to secure QRISK2 as the sole risk score used in the Health Check programme.
Findings related to CVD risk scores have implications beyond the NHS Health Check programme alone. QRISK/ QRISK2, despite improvements in prediction compared with *Framingham* scores,\(^{408}\) have limitations. Generally QRISK/ QRISK2 under-predicts risk, most notably in BME groups.\(^{83}\) Crucially, however, being the product of a continually evolving database, the algorithm itself is continually revised. With the completeness of a number of CVD risk factors in primary care EMRs presented here, the accuracy of CVD risk prediction using large routine datasets is likely to improve. This further strengthens the case for using routine datasets, over cohort data for risk prediction, indeed they may have a growing role in wider research.\(^{651}\)

My work, finally, demonstrates the importance of non-clinical elements within CVD risk prediction tools. The failure to accurately capture ethnic differences impacts on work load, costs, patient harm and health inequalities. The UK, through the ASSIGN and QRISK / QRISK2 risk scores has, internationally, been the vanguard in this field. Elsewhere, however, recent work concentrates on clinical aspects of risk.\(^{82}\) The consideration of non-clinical aspect of CVD risk may be vital in maintaining equitable CVD risk prediction.

**Programme workload and funding**

*Work load*

The NHS Health Check programme will generate an enormous work load for English general practice. Risk assessment and data recording alone will prove an enormous task. My data, for example, suggest that simply completing CVD risk factor data in EMRs will give a significant number of risk factor measurements. The *de novo* data recording, required under programme guidance, will be significantly greater.

The NHS Health Check, even in general practice, will not be undertaken by GPs. Practice nurses and more commonly health care assistants will carry out the work. These practitioners are less costly to employ and are likely to increase the capacity of primary care to manage the programme.\(^{652}\) Nonetheless, general practice still has a finite capacity. There
is little academic evidence surrounding general practice’s capacity outside of the GP, or more importantly its ability to expand capacity to meet need. Anecdotal evidence suggests that members of general practice teams are already placed under significant strain during the ‘flu vaccination season. Given potentially limited capacity, the Health Check work load may prove unmanageable for some. There is, in fact, early evidence that practices have failed to begin implementation, well over two years into the programme. In some areas progress across entire PCTs remains limited.

NHS Health Check work load does not stem entirely from risk assessment. Considerable efforts will be required in the management of cardiovascular risk. These efforts are a vital stage in CVD prevention, and without them the risk assessment process becomes entirely futile. Modelling identifies a large population eligible for statins under clinical guidance, 2 million patients using QRISK2, at a cost of £39 million per annum. Statins are efficacious agents for the control of lipid levels across all levels of CVD risk. Despite questions over their cost-effectiveness in low risk groups, there is growing evidence that they are cost-effective when used for primary prevention in the high risk, groups including those currently eligible. The offer of a statin remains one of the major elements of intervention within the programme, therefore sufficient funding must be made available to cover increases in prescribing generated by the NHS Health Check programme. Further funding may also be required to promote adherence, which has the potential to severely limit programme gains.

The prevalence of two CVD risk factors stands out within the high risk population. Using QRISK2 to define the cohort, my modelling suggests 83 percent of the high risk cohort will be physically inactive and 65 percent overweight or obese. These patients are eligible for intervention; a community physical activity intervention or weight management programme respectively, a large work load for these services. Across many PCTs, community referral services remain dramatically under-developed, with significant variation in the availability and capacity offered. There is no indication that there is currently sufficient capacity to manage the work load outlined above. The prevalence of these two risk factors has two main
implications. Firstly, if one deems these interventions effective, sufficient funding must be available for interventions.

The second implication is whether this is the correct strategy to pursue at all. Firstly, even with further funding and development one has to question whether these services can ever match the growing need generated by physical inactivity and obesity. Secondly, previous high risk interventions, even when successful in reducing some CVD risk factors, have failed to curb major population-wide trends in risk factors. Added to this the weaknesses outlined in these interventions, for example their limited uptake, and widely heterogeneous effects, these high risk interventions may simply not have the power to exert the required changes. Instead structural changes to the population may be the only way to manage the global rise in obesity and physical inactivity. Intervention incorporating the food industry, transport systems, town planning and recreation are likely to be required.

Programme funding
The size of the work load produced by the programme has links to another important element, it’s funding. The greater the work load generated by risk assessment and management, the greater the cost of the programme. My data, for example, indicate a cost of £176 million to assess risk and manage the high risk population, using the QRISK2 risk score. Further estimates have indicated an annual programme cost of between £180 and 240 million, with some PCTs currently allocating up to £910,000 per year for the programme. If pursuing the Health Check programme, it must be adequately provisioned for, with finances available.

The work load, and resultant costs, will prove especially difficult to manage in the changing landscape of NHS finances. In line with wider reductions in NHS spending, there is evidence that PCTs may begin to withdraw explicit funding for the programme. Already, there is up to a 20 fold difference in the funding available to general practices between PCTs. Without extra funding, general practices have to manage the programme work load
within their existing budget. This will create a greater burden, with more practices likely to withdraw.

Uncertainty surrounds whether financing alone will be sufficient to overcome limitations in general practice capacity. Even with additional financing, general practice might not have the time, space or staffing required managing the work load. Some areas have gone beyond purely financial support, providing project workers to support screening. Previous trials have demonstrated the value of central project facilitators in CVD prevention.

Wider questions also exist over Health Check remuneration. The preferred method of funding, through LES payment has limitations, and should be considered a short-term solution. Finally, with changes to NHS architecture, the funding structures will significantly alter. Funding a largely general practice based scheme through a local authority’s public health budget is almost certain to create challenges. Boundaries between what constitutes Health Check and routine care will be difficult to define. The annual follow of high risk patients, for example, could be seen to be either. Whether general practices receive additionally funding from the public health budget, or have to fund this through their core clinical budget will be open to debate.

Alternate service providers might make up for the limitations in capacity within general practice. A large work load and potentially limited funding might, however, prove a significant barrier to alternate providers. Two major alternate Health Check settings are pharmacy and community venues, such as places of worship and town halls. Community screening requires funding and is often implemented directly by PCTs. Pharmacy providers also need funding. They are profit making organisations, whose Health Check involvement is, at least to some degree, financially motivated. Alternate providers also present a challenge in the source of funding from within PCTs. Frequently, as in Ealing, PCTs use LES
contracts to remunerate general practice for the Health Check. LES money has to be used to fund additional services within general practice and cannot, therefore, fund alternate providers. The combination of weaknesses and a greater sensitivity to funding limitations might make alternate providers unable to fill gaps in capacity left by general practice.

In addition to the funding of general practice by PCTs, my work has implications for the allocation of finances to PCTs. Resource allocation for the Health Check goes from the Department of Health, through Strategic Health Authorities (SHAs), to PCTs. Currently resources are allocated based on the population size. My research suggests considerable variation in the prevalence of high risk patients between PCTs, ranging from 6.4 to 13.7 percent of the eligible population, using QRISK2. For equitable service provision, funding must better account for the risk profile of a population which will vary dependent on a number of demographic factors including the age, deprivation and ethnic profile of a population.

A second limitation is structure of the remuneration. The Health Check programme is funded within PCTs annual global budget, the programme has no ring fenced budget. This is how most health care activities are funded, and is employed by the Department of Health to allow PCTs freedom to shape local services, managing and partitioning their own budgets. Early experiences have suggested this might be detrimental to the Health Check programme. Currently, given growing financial restrictions, the merits of primary prevention appear low in the priorities of NHS finance departments. Being difficult to present evidence of immediate health gains from the programme, financial support has been lacking. Without sufficient funding, and high-level support within PCTs, the programme may be severely limited.

My data outlining may have a role in the current programme’s performance management. Performance management, undertaken by SHAs, focuses heavily on process measures. Even ‘outcome data,’ comprise merely of baseline risk profiles. The reliance on process data percolates through from SHAs to the PCT’s management of providers. Service providers are largely funded for risk assessment, not risk management. Focusing programme performance on outcome measures is likely to be central if the programme is to
be effective. In the interim levels of global CVD risk within the high risk population receiving intervention may be a useful metric to assess performance. With, as yet, no nationally collated data, data presented here provide a baseline measure of CVD risk in England.

**Threats to the success of the NHS Health check programme**

*Evidence base behind the implementation*

Findings above discuss the implications of my work given a continuation of the current structure of the programme. I shall now, however, summarise why this *status quo* might not be the best policy option for England. The NHS Health Check programme is one of England’s foremost public health initiatives, generating a large work load and spending. For such a great commitment, however, the existing literature raises questions about the programme’s current design and implementation. Strikingly, there is little previous evidence of the successful implementation of a universal high risk approach to CVD prevention.

Firstly, there have not been a large number of trials surrounding universal CVD prevention. From the existing literature, there is evidence of reductions in CVD risk factors in participants.\(^7^0\) There are, however, a number of limitations; firstly there is only limited evidence of a positive effect on CVD outcomes, although this is could be due to methodological deficiencies.\(^3^1^0\) Secondly, despite overall successes of trials, much of the risk factor reductions are within high risk patients.\(^7^0\)\(^2^3^2\)\(^2^6^6\)\(^2^7^9\)\(^3^1^2\) Thirdly, even when successful, high risk prevention may not be able to alter wider secular trends in risk factors; trends caused by wider behavioural and environmental changes.\(^2^7^0\)\(^2^7^1\) Finally, although successful in attendees, the population-wide impact of high risk prevention can be severely limited by poor uptake.\(^2^6^8\)

I also describe concerns over a number of interventions within the programme. The NHS Health Check offers risk assessment to all. Interventions, however, are focused on high risk groups, often at patients displaying risk factors. The impact of the programme on the low to medium risk groups is questionable. Only two interventions are available to the entire
population attending a Health Check, namely the communication of risk and a brief lifestyle intervention.

CVD risk communication is central to risk reduction. Accurate perception of risk by a patient, which relies on strong communication, improves patient and population level outcomes.\(^{298, 299}\) Equal to the positive aspects of good communication are negative consequences of poor communication. If CVD risk is poorly understood, a patient can feel increased levels of anxiety or alternatively complacency over their cardiovascular health.\(^{288, 290}\) There is evidence that clinical trust is important in risk communication.\(^{308}\) In many areas, the Health Check is undertaken by health care assistants. There is no indication of how their relationship with, and the confidence of patients will impact on the dissemination of risk. Given major implications of CVD risk communication, it receives little attention in programme guidance.\(^{114}\) Strong communication should be central to the current programme, however may prove a significant limitation.

Brief lifestyle interventions, similarly, have concerns surrounding their effectiveness when undertaken in a Health Check settings. Brief interventions are effective, however, evidence remains limited for their implementation by non-clinical staff or staff lacking motivation, belief and expertise in the methods.\(^{151, 153}\) Pharmacists can effectively give brief interventions, but again not when inexperienced or untrained.\(^{154}\) Brief interventions may require follow-up to be successful, something that will not happen for the majority of the Health Check population.\(^{227}\)

Weaknesses in the implementation of both risk communication and brief interventions may limit the population-wide impact of the programme and expose patients to unwarranted harm. So limited is the intervention in the whole population that one has to consider the programme a high risk approach to prevention. The nature of the programme is important. Being high risk may limit the overall impact of the programme within the population\(^{63, 90}\) and impact on its relationship with inequalities.\(^{94}\) Population prevention might be more able to reduce inequalities,\(^{91}\) whilst high risk approaches can exacerbate them.
All medical enterprises possess an element of patient harm. There are harms associated with screening, for example, a perception of adverse reactions and anxiety over the results. Harms associated with CVD prevention remain under studied. There may be limited scope for physical harm, although statins possess a number of side-effects as can medicalise patients. Nonetheless, psychological aspects of harm are likely to be of greatest concern. One potential perverse outcome in particular remains under studied; excessive reassurance gained from risk assessment in those defined at lowest levels of risk. This may result in patient complacency surrounding their cardiovascular health, and a loss of self efficacy. Calls not to implement cardiovascular screening because of our current gaps in knowledge seem excessive. We must not, however, discount patient harm. High standards of practice are needed to mitigate risks and harm studied as part of the programme evaluation.

CVD prevention has undergone a major change in recent years, shifting focus from management based upon individual risk factors to global risk. Concentrating on patients with raised global risk is, for example, the most cost effective method of preventing CVD events. Clinical guidance has begun to adopt global risk. In Europe, the European guidelines on CVD prevention, and recent guidance from the European Medicines Agency on the use of drugs for CVD prevention both support global risk. In the UK, NICE has promoted global risk, most notably to define statin eligibility for primary CVD prevention.

Superficially, the programme is reliant on global CVD risk, with the CVD risk score central. All patients screened must have their global risk score calculated and recorded. Brief lifestyle intervention should then be tailored to the patient’s risk. Global risk is further used to define a high risk cohort, who receive management in general practice, should undergo intensive follow-up and become eligible for statins.

The remaining interventions, however, are initiated by the presence of single risk factors. Any patient within the low to moderate risk group presenting a single CVD risk factor should be referred or signposted to further intervention. Blood pressure management, for example, is only available to those who proceed to be diagnosed with hypertension. This is
despite evidence that blood pressure control might be better targeted using global risk for CVD prevention.\textsuperscript{112} Management of patients raised risk factors within low to moderate risk groups is a large aspect of intervention, and will amount to considerable spending. Ignoring global risk, in spite of growing evidence to support this approach, may not give the most efficient and cost-effective resource allocation.\textsuperscript{111} Again, considering a revised prevention strategy, fully embracing global risk might be warranted.

\textit{Uptake}

The uptake achieved by a high risk prevention programme is the single greatest driver of success. Low uptake means a programme has minimal impact on the population at large.\textsuperscript{90} Specific to CVD prevention, both the uptake of the initial risk assessment and of risk lowering interventions are important; as is adherence to interventions. At only 45 percent, this is considerably lower than the 75 percent estimated by the Department of Health.\textsuperscript{139} Similarly, only 43 percent were prescribed a statin, compared with Department of Health estimates of 85 percent.

Comparing my findings with previous modelling demonstrates the impact of poor uptake. As the uptake of statins decrease, the population impact falls;\textsuperscript{90} falling from 100 to 40 percent, for example makes a 60 percent reduction in the impact on CVD mortality. In absolute terms, a 40 percent uptake of statins in those at high risk or with total cholesterol greater than 5 mmol/l was estimated to reduce CVD mortality by only 0.16 percent. My findings are from the first year of the programme, therefore uptake might improve. However, Health Check attendance does not differ from previous, comparable CVD prevention trials.\textsuperscript{147 312} The suboptimal uptake to the Health Check and statins will seriously diminish the success of the programme. This must either be improved, or else alternatives sought.

Full uptake of statin therapy will never be achieved. Patients suffer drug contraindications and therefore have valid reasons not to begin treatment.\textsuperscript{66} Contraindications do not, however, reach the magnitude of patients found defaulting from statin use in Ealing.\textsuperscript{66} The beliefs and behaviours of GPs and patients are likely to play an important role in statin
prescribing. GPs may have a limited understanding of the role of statins in primary prevention, although this may have changed since their wider availability and greater dissemination of clinical guidance.\textsuperscript{661} Perceptions of side-effects may be a major obstacle to prescribing, particularly as a preventative medication.\textsuperscript{224}

Other factors influence GP prescribing patterns; the same patient will not evoke the same prescribing response across clinicians.\textsuperscript{662,663} Clinicians may see an array of barriers to prescribing for primary prevention, including concerns over cost (to both their general practice and the health system); over the increased workload; the over-medicalisation of patients and loss of self-efficacy.\textsuperscript{662,663} Clinicians feel strongly about a shared and informed decision making process in primary prevention, which also impacts on prescribing.\textsuperscript{662,664} GPs may also hold doubts over primary prevention \textit{per se}.\textsuperscript{664} They see limitations in the evidence base and only minimal impact on the individual patient-to-whom they have ultimate responsibility.\textsuperscript{664} A large number barriers to prescribing and primary prevention exist, although adherence to clinical guidance should help overcome these.\textsuperscript{113}

Patients face barriers to primary prevention medication. If, as recent qualitative research suggests, patients play a major role in the decision making process,\textsuperscript{662,664} then these become obstacles to therapy. Obstacles for patients include concerns over side-effects and not wanting to be over reliant on medication.\textsuperscript{223,224} Good quality information can dispel unwarranted fears, with accurate communication of CVD risk also improving prescribing.\textsuperscript{298}

In addition to uptake, the adherence to interventions is vital for programme success. Although my analyses did not touch upon this, previous work suggests limited adherence to drug therapy for primary CVD prevention. The adherence to statins for primary prevention, in fact, seldom reaches 50 percent,\textsuperscript{59,74} let alone the 75 percent estimated by the Department of Health.\textsuperscript{139} Efforts must be made to promote the adherence to statins (and uptake of interventions) if the programme is to continue; otherwise if not improved this stands as such a limitation that alternative strategies are required.
Programme cost-effectiveness

Strong uptake and adherence to Health Check interventions are needed, not only to ensure programme effectiveness, but also cost-effectiveness. To be truly cost-effective, potentially cost saving, a prevention programme must \textit{prevent} disease events. The premise is simple; spend more on low cost prevention to save on high cost clinical care after disease occurs. Cost-effectiveness is intrinsically linked to the absolute impact which is in turn limited by poor uptake.\textsuperscript{90} Reductions in cost effectiveness will be exacerbated because the infrastructure and capacity for intervention must be developed and commissioned before screening. If patients do not attend, spending will not go towards reducing CVD risk and cost-effectiveness further reduced.

One further finding has implications for programme cost-effectiveness. In Ealing, after the Health Check, there was a 45 percent relative increase in statin prescribing in patients. not eligible.\textsuperscript{113} The cost of prescribing within the Health Check programme will be great. If GPs deviate from the clinical guidance based upon global CVD risk, then the programme’s cost-effectiveness is likely to be further undermined.\textsuperscript{111}

\textit{Inequalities}

A number of inequalities have been identified in uptake in preventative medicine and screening, with evidence of socioeconomic differences,\textsuperscript{469} and some, albeit still contentious, evidence of ethnic variation.\textsuperscript{514} I did not find lower uptake of the NHS Health Check in deprived or BME groups. Indeed patients of black, south Asian and mixed ethnic groups had higher attendance than the white group. This is a positive finding. The NHS Health Check aims to address overarching cardiovascular inequalities in England, of which ethnic and socioeconomic variation is a significant component.\textsuperscript{34,38} There has been debate over whether the Heath Check, indeed all high risk prevention, can reduce inequalities, or whether it will exacerbate them through low uptake in those with greatest need.\textsuperscript{94} This was not found here, in fact patterns in Health Check uptake broadly follow wider patterns in general practice attendance,\textsuperscript{454} the location of Health Checks in Ealing
Although all patients were invited to the Health Check via letter from general practice, it is unclear whether there was any opportunistic activity. If eligible and attending general practice, in addition to the letters, patients may be either reminded to attend their Health Check, or receive one opportunistically. It is plausible Health Check uptake will, to some degree, follow general practice attendance. Further work is required to assess to what degree (if at all) the NHS Health Check was opportunistic, and how many attendees responded to the letter of invitation. Providing access to a Health Check in additional to pre-booked appointments may improve attendance.\(^{657}\) Understanding the best methods of invitation and service delivery will be important if continuing with mass CVD screening.

One of the greatest inequalities found was between general practices. There were significant differences in both the completeness of CVD risk factor data in EMRs and in screening uptake between practices. The latter, was still evident having controlled for patient and practice characteristics. There are a number of potential underlying reasons behind this practice level variation. The organisational capacity of general practice can impact on both screening uptake and clinical recording. Additional support staff, including administration and clinical assistance improves organisation and invitation to preventative services, and can improve clinical outcomes.\(^{665} \quad 666\) In both the analysis of clinical recording and of uptake, I controlled for the number of FTE GPs per practice, however, had no data concerning wider practice staff. This might account for practice level differences. The beliefs and commitment of a GP to the area of prevention can also impact on the quality of service offered. This is maintained even when the service is not offered directly by the GP, and may be the result of the promotion screening or its facilitation by the GP.\(^{67}\)

In Ealing, practices carrying out the NHS Health Check received additional funding through a LES agreement, with payment proportional to the number of Health Checks conducted. Unexplained variation might be from differential response to payment. The system of remuneration may also be implicit in the finding that the smallest general practices had highest Health Check uptake. One recent study of cardiovascular risk assessment, also
funded though a LES, found greatest uptake in single-handed practices, having not control for practice size. They hypothesise, that single-handed practices have a smaller global budget and LES payments for cardiovascular risk assessment add relatively more to their overall income. Promoting the service, therefore, becomes more appealing. A similar situation may occur in the small practices in Ealing.

There is evidence of longer consultation time in smaller general practices, better perceived clinician availability by patients and greater patient satisfaction. These might improve compliance with the Health Check. My findings regarding uptake, along with previous work, counter recent moves to reconfigure primary care, towards larger general practices. Larger practices in Ealing did, however, have greater baseline recording of cholesterol and blood pressure. This may reflect traditional wisdom that larger practices are more organised with greater staffing, therefore produce higher quality care.

Work is required to fully establish the relationship between general practice size and quality of care, and therefore optimal practice size. Unwarranted practice level variation is clearly detrimental to programme effectiveness. Efforts must be made to establish equitable health care across all practices. This might require greater consideration of the funding structure of the NHS Health Check or additional support to poorly performing practices.

**Policy options for CVD prevention**

The first of the three major policy options in the UK for primary prevention of CVD is to maintain the *status quo*. That is, carry on employing a universal NHS Health Check programme. Through both reviewing the existing literature, and through work presented here, I show a number of flaws to the programme may severely limit its impact, and that the alternate options must be considered. However, even if continuing with a universal programme, my findings point to potential changes which may improve the equity and cost-effectiveness of prevention.
CVD risk scores can be used to prioritise patients for screening. Within a universal programme, patients estimated to be at the highest levels of risk, using existing EMR data, could be invited for screening first. Pre-selection demonstrates that one could identify the majority of CVD risk factors in those invited the earliest to screening. My data from Ealing suggest a number of CVD risk factors are largely complete, therefore prioritisation will be efficient. Secondly, having pre-stratified a high risk cohort in year one of the programme, a large proportion of this population were confirmed at high risk. Targeting the Health Check will improve equity, for example, overcoming a situation, whereby a 40 year old woman with no risk factors attends CVD risk assessment before a 70 year old man who smokes.

My data also question the necessity for de novo recording of all CVD risk factor data during a Health Check. Patient medical records had a large amount of recorded CVD risk factor data prior to the programme, with much of these data recent. Other work suggests many Health Check attendees have had recent previous contact with primary care, again pointing to recent data recording. De novo data recording in all Health Check attendees may not significantly improve risk assessment, and will lead to unnecessary programme spending. Another option is to allow the use of recent, pre-recorded risk factor data within the NHS Health Check, particularly for data requiring more costly blood testing.

These two options would modify the current programme; however, from evidence prior to the NHS Health Check, I identify a number of potential weaknesses. These include evidence that brief lifestyle interventions and risk communication can be ineffective, even harmful; limited evidence behind effective and cost-effective universal prevention. My finds add poor uptake as a major threat to effectiveness. If these are not dispelled, then it will be important to seek alternative policy options. Even without improvements in the current programme, I show evidence that an alternate English strategy may be required.

The first alternative approach to CVD prevention is to more fully embrace a population approach. I outline the growing evidence behind population approaches to prevention in chapter 3. There is now evidence supporting population level inventions for a number of CVD
risk factors, including smoking, salt and trans-fats. There are also further interventions available, including the recently adopted Danish approach to tax high-fat food; the taxation of high-sugar foods or more far reaching interventions supporting active transport or shaping the built environment to aid physical activity. Table 3-1, spells out a clear advantage of population prevention; Compared with Health Check predictions, they have the potential for a significantly greater impact. Banning trans-fats in England, for example, will have over a ten times greater reduction in CVD mortality.

Not only is population prevention are effective, but they may be more cost effective than high risk approaches, and may even be cost-saving. They can also be effective without being dependent on the explicit individual level behaviour change, structural interventions. Despite these strengths, and despite the evidence, the UK has focused largely on high risk strategies for prevention. There is now sufficient evidence available to more fully utilise population approaches to CVD prevention.

A more novel population approach is the Polypill. Despite growing evidence of its efficacy, there is currently insufficient evidence that this is cost-effective or even effective, especially when used in low risk groups. There is no indication of the implications of placing an entire population on medication, for example on patient self efficacy. Finally, implicit in a Polypill approach to maintain cost effectiveness, is to drop other prevention efforts. Ethical questions remain over not giving explicit consideration to the high risk group. In short, there are currently a large number of unanswered questions surrounding the Polypill; however, it remains a potential option for CVD prevention in the future.

The second option for CVD prevention is to maintain a high risk primary prevention programme, but to significantly overhaul it, making the programme targeted at high risk patients, not universal. Work presented here has a number of implications for targeted, high risk CVD prevention. CVD risk scores stand out as an effective method of selection. Pre-stratification involves taking existing risk factor data from EMRs, substituting missing data,
and then applying the CVD risk score. The programme would then invite only those estimated to be at the highest risk for CVD risk assessment.

Without additional data collection, CVD risk factors, especially blood pressure and smoking status are well recorded in general practice. Improved data recoding allows efficient targeting, however complete data are not required and may provide for accurate pre-selection. The use of EMR data to risk stratify patients in general practice will encounter missing risk factor data, which must be substituted to apply a CVD risk score. Chapter 8 demonstrates a simple method, not requiring statistical expertise, is effective in generating default values. A single national method for missing data, such as the one discussed here, could be incorporated into bespoke software designed for the Health Check programme and allow the widespread use of targeting.

Calls for targeted screening are not new to the UK, with evidence growing in support, especially of greater cost effectiveness compared with universal approaches. There is little incremental gain from undertaking risk assessment in low risk groups, and much previous CVD prevention has found greatest gains in highest risk subgroups. Further, recent evidence showing lipid data has only limited use in those estimated to be at the lowest risk, demonstrates the potential for targeting.

Targeted high risk prevention has a number of advantages over a universal method. Most notably it generates considerably smaller workload from risk assessment. Instead of screening the entire population, one merely screens a sub-section. This has the obvious benefit of reducing the time spent undertaking risk assessment; the number of tests carried out and hence the costs. The reduction in screening workload will be directly proportional to the reduction in number screened. Using data covering 29 general practices in Ealing, my estimates suggest a universal programme would screen 57,200 patients aged 35 to 74 years (see chapter 11). Pre-selection of the predicted high risk reduces this to 5,300 (9.3 percent).
The large work load generated by a universal NHS Health Check may have limitations for the quality of service provided. Within a closed health system, spending decisions possess an opportunity cost. Currently, within the NHS, there is a growing need to ‘deliver more with less.’ The work load of universal screening channels a large amount of spending into risk assessment, diverting resources away from the intervention and active promotion of uptake. The alternate, targeted screening, will allow greater investment in the service provided.

Low uptake is catastrophic for high risk prevention. Further, inequalities in screening uptake are widely documented. My work, demonstrates lower Health Check uptake in men than women, and there is additional evidence that inequalities can run parallel to patient need. High risk prevention, such as the NHS Health Check has the potential to increase health inequalities. Using targeted prevention, with greater resources available for risk assessment, one can pursue more sustained invitation and provide wider access. In general practice, one can extend the hours of access, provide additional drop-in screening or have greater patient contact during invitation. This may boost overall uptake and can also be targeted at those with greatest need, reducing inequalities.

Greater resources also allow the commissioning of additional services, designed specifically to target segments of the population. Targeted community events, for example, can be effective in both increasing total uptake and in promoting it in hard-to-reach populations. Finally better resourced risk assessment and intervention allow a higher quality service and might mitigate weaknesses of the programme, including the limitations of brief interventions and the risk communication.

A universal approach has some benefits. The Health Check programme might galvanise English primary care to the cause of CVD prevention. Preventative medicine, often set in primary care, has great potential for population gain, especially when part of an organised, well motivated programme. A targeted programme does not, however, ignore prevention, merely uses a different approach. In Chapter 9, I describe the potential for the Health Check programme to benefit diabetes prevention and management. It will indentify a large
prevalence of undiagnosed diabetes and IGT / IFG. The early management of these reduces CVD risk and prevents wider poor health outcomes. Case finding for diabetes, however, does not currently justify a universal programme. Universal screening can be inefficient, and questions remain over how best to screen for diabetes.594 669

The universal approach theoretically captures all of the CVD risk in a population. The whole population is assessed; therefore all risk factors identified. A targeted approach, conversely, may not. This reasoning has two flaws; firstly the question must revolve around cost-effectiveness, not total gain. The most cost-effective method to make the greatest risk reduction must be found, not the greatest risk reduction per se, considering what the universal approach adds incrementally to the targeted.435 The second question is whether, in practice, there actually are significant differences in coverage. With the uptake demonstrated here, one could hypothesise that a well provisioned targeted approach (able to better promote uptake) might be able to match a universal approach,

The case for using a targeted approach becomes even stronger in the current financial situation of the NHS. Over recent years, the NHS has benefited from continued, unprecedented, increases in funding. Between 1997 and 2007, government expenditure on the NHS increased from £56,414 to £97,218 million (at 2007 prices).670 At times, annual real terms growth reached 9.8 percent.670 The roll out of the programme has, however, coincided with a stalling in growth, which might well manifest itself as real term cuts in spending.30

The Health Check programme was devised during a period of immense financial growth but has to be implemented in more austere times. The strengths, or perceived strengths, of a universal programme might have been attractive when the programme was devised. These no longer warrant the increased spending, with a more cost effective targeted approach becoming even more attractive.

The initial decision to follow a universal Health Check might not have been entirely evidence based. A universal programme is politically appealing. It includes the entire general
population, therefore gives something tangible to all of the voting public. From the outset, the central UK government, not just Department of Health showed interest in the programme. The programme was, for example first announced by the Prime Minister. When undertaking modelling that underpinned the programme, a targeted approach was not compared, merely different universal methods. The decision to offer universal screening appears to have been made before adequate modelling took place.

Political involvement is also likely to have affected the programme’s time frame. Implementation began before a number of key elements, including national information technology capabilities, the minimum dataset and the call-recall system, were in place. Political influences appear to have hastened the programme’s introduction. This has created difficulties in implementation and has lead to duplication in local planning.

The two alternative methods of CVD prevention are not mutually exclusive. In fact, as first stated by Geoffrey Rose, high risk and population approaches should be used in combination. Recent academic texts have promoted a duality within prevention; however, this has not always been transferred into practical public health. Either a targeted or universal approach could partner population measures to prevent CVD, however given the discussion above, and evidence of the political and not evidence based introduction of the Health Checks, targeted screening stands out as superior option. The population interventions will make major changes to the mean risk across a population, and make significant reductions in mortality, whilst the targeted high risk approach will address those with greatest immediate clinical need.

12.5 Future research and programme evaluation

Given the evidence surrounding population primary prevention, modelling to compare these approaches with the current Health Check strategy will be vital to shape future policy, as will consideration of more targeted strategies which were omitted by Department of Health
modelling. My thesis examines the NHS Health Check in its infancy, with data covering the first year of the programme. It will be necessary to continually monitor uptake as the programme becomes more established. If uptake remains low, efforts must be made to find effective methods and interventions to increase it, or else redirect the programme spending.

I analysed levels of statin prescribing; it will be important to study compliance with statin prescriptions, incorporating pharmacy prescribing data into analyses. Little is known about the uptake or adherence to life-style and community interventions, or the resultant behaviour change, especially after referral from general practice. These interventions are heavily employed in the programme, and constitute a large proportion of the available intervention. Being central to the programme, strong uptake is crucial and more research into this is required.

The programme can be implemented across a range of settings; its effectiveness and uptake must be studied outside of general practice. When carried out by alternate providers, one key aspect is for newly recorded risk factor data to be added to patients’ primary care records. Without this, risk assessment will be of limited use and create a duplication in workload.\textsuperscript{671} It is important to monitor this secondary recording of risk factor data, with again very little currently known about the efficiency of this process. Although the provision of the Health Check through pharmacy is an attractive proposition, early work demonstrates a number of barriers.\textsuperscript{648} A direct comparison, in terms of effectiveness and cost-effectiveness, between providers is required.

Data from Ealing, in the first year of the programme, covered a population estimated to be at high risk. The bulk of Health Check workload will, however, stem the more general population; uptake and outcomes must be studied here. Ealing is also a unique setting, with a large ethnic minority (especially south Asian) population. Ealing may be atypical of England as a whole; therefore rates of attendance must be monitored elsewhere. Data from across the entire nation must be monitored and evaluated to fully understand the programme’s impact on health. Nationally representative datasets, including the General
Practice Research Database (GPRD) and QRESEARCH will be important for this, as will extracts from the Health Check dataset.¹⁴³

There is limited evidence behind the effectiveness of universal high risk CVD prevention. As the programme progresses CVD outcomes must be monitored. Initially medium-term measures, including CVD risk factors (blood pressure, lipid levels etc.) and global CVD risk are important. Rates of disease diagnosis and for diabetes, disease progression, are further important medium-term outcomes. It will also be important to consider the impact of the programme on hard outcomes, including CVD events, hospital admissions and mortality.

As outcome data become available, the thorough analysis of the programme’s cost-effectiveness is central to any continued support and funding of the programme. Similarly, the effectiveness of the two CVD risk scores, QRISK2 and JBS2, can be more closely scrutinised using longitudinal data containing CVD outcomes. As well as identifying high risk patients, the programme will discover a significant amount of undiagnosed disease. This provides a significant opportunity to assess the impact of the early diagnosis of conditions including CKD, diabetes and hypertension on clinical outcomes.

A number of potential harms surrounding CVD risk assessment were raised before the programme was implemented. General harms of CVD risk assessment and the effectiveness of both risk communication and brief interventions must be continually addressed to minimise perverse outcomes. One area of harm of great importance is the programme’s potential to excessively reassure those found to be at the lowest CVD risk. Little is currently known about the scope for false reassurance, and this could have great implications for patient care. Finally, assessment of the patients’ response to the programme and the acceptability of primary prevention are needed.
12.6 Conclusions

The NHS Health Check programme is a significant commitment in England to the primary prevention of CVD. Although open to the entire population, the programme is fundamentally a high risk strategy. A number of weaknesses identified prior to the programme, including limitations of the evidence base, a potential for patient harm, and a reliance on single risk factors, not global CVD risk, brought about concerns over the current English primary prevention policy.

My findings indicate that the NHS Health Check will generate a significant work load to the English health service, especially through CVD risk assessment and intervention in the high risk cohort. The early uptake of the Health Check and of statins, one of the major interventions in the high risk, was poor. Such levels of uptake will render the programme ineffective in reducing the CVD burden in England.

There were no negative socioeconomic or ethnic gradients in uptake which alleviates fear the programme will exacerbate health inequalities. This, however, must be studied in different settings. Considerable general practice variation warrants concern. Support to practices and possibly changes to funding structures may be required for prevention services.

Given these, especially the poor uptake, there is a need to consider alternate methods of CVD prevention. Current levels of data recording in primary care medical records, combined with simple methods to impute missing data, make the pre-selection of patients an extremely viable and attractive proposition. Added to this, evidence grows supporting large-scale, population prevention measures, with data suggesting they will outstrip high risk approaches in terms of overall impact. These must have a greater presence in national policy; a presence commensurate with the evidence.

For health systems, both in the UK and in many other high income countries, the primary prevention of CVD is currently more critical than ever before. Aging populations mean CVD
will cause greater and greater morbidity, threatening to financially destabilise them. We equally stand at a point in time where more cost-effective, greater value services are demanded. As such, this stands as a moment in time CVD prevention. Addressing this with a national programme is admirable. If maintaining the current programme structure, efforts must be made to strengthen funding, and to give the Health Check support to allow it to navigate the coming structural changes within the English health system. I, however, conclude that population and targeted high risk approaches, not the current Health Check strategy should be pursued if we are to successfully reduce the burden of CVD.

Despite weaknesses, the NHS Health Check is certain to create international interest for public health and policy-makers. It is the first such programme in the world, therefore will provide significant insights into the prevention of CVD. In summary, however, I show an insufficient evidence base, limitations in methods employed and general restrictions in high risk prevention undermined the programme from its outset. Poor uptake further compounds these concerns, and will cripple the programme’s population impact. Reinvestment in population intervention stands out as a great alternative. This in conjunction with a reduced size targeted Health Check programme, managing those at the immediate risk should be considered within national prevention policy.
Appendix I; additional tables

Table viii-1; the variables selected for the multiple imputation models in chapter 7

| Blood pressure | sex, age, ethnicity, hypertension, family history, age*sex, ethnicity*sex |
| Cholesterol    | sex, age, ethnicity, deprivation, family history, age*sex, ethnicity*sex |
| HDL            | sex, age, ethnicity, deprivation, diabetes, hypertension, family history, age*sex, ethnicity*sex |
| BMI            | sex, age, ethnicity, deprivation, diabetes, hypertension, age*sex, ethnicity*sex |
| Smoking        | sex, age, ethnicity, deprivation, diabetes, hypertension, family history, age*sex, ethnicity*sex |

Table viii-2; the variables selected for the multiple imputation models in chapter 8

| Blood pressure | sex, age, ethnicity, deprivation, family history, diagnosed hypertension, chronic kidney disease, sex*age, sex* ethnicity |
| Cholesterol    | Sex, age, ethnicity, deprivation, diabetes, family history of CVD, diagnosed hypertension, chronic kidney disease, sex*age, sex* ethnicity |
| HDL            | Sex, age, ethnicity, deprivation, diabetes, family history of CVD, chronic kidney disease, sex*age, sex* ethnicity |
| BMI            | Sex, age, ethnicity, deprivation, diabetes, family history of CVD, diagnosed hypertension, chronic kidney disease, sex*age, sex* ethnicity |
| Smoking        | Sex, age, ethnicity, deprivation, diabetes, family history of CVD, diagnosed hypertension, chronic kidney disease, sex*age, sex* ethnicity |

Table viii-3; Age standardised JBS2 estimates using multiple imputation and HSfE data to replace missing data in patients with missing data

| Risk Score | Missing | White | South Asian | Black | Other | Total |
|           |        |       |            |       |       |       |
| Male | HSE | 12.8 | 13.8 | 19.2 | 13.7 | 13.8 | 14.0 |
|       | IMP | 12.6 | 13.3 | 18.6 | 12.7 | 13.4 | 13.6 |
| Female | HSE | 6.4 | 7.0 | 6.9 | 6.6 | 7.0 | 6.7 |
|       | IMP | 6.2 | 6.7 | 6.8 | 6.2 | 6.7 | 6.4 |
| Male | HSE | 21.7 | 24.7 | 37.3 | 23.8 | 23.5 | 24.5 |
|       | IMP | 20.1 | 21.4 | 36.2 | 21.1 | 22.6 | 22.7 |
| Female | HSE | 0.892 | 0.885 | 0.902 | 0.898 | 0.901 | 0.894 |
|       | IMP | 0.892 | 0.892 | 0.892 | 0.892 | 0.892 | 0.892 |

| High Risk | Missing | White | South Asian | Black | Other | Total |
|           |        |       |            |       |       |       |
| Male | HSE | 3.4 | 5.2 | 6.5 | 5.0 | 5.5 | 4.5 |
|       | IMP | 2.8 | 2.6 | 5.2 | 3.6 | 4.6 | 3.6 |
| Female | HSE | 0.889 | 0.848 | 0.826 | 0.884 | 0.883 | 0.868 |
|       | IMP | 0.889 | 0.896 | 0.856 | 0.834 | 0.891 | 0.861 | 0.875 |
Table viii-4: General modelling assumptions from the Department of Health

Screening uptake = 70%
Screening- 50% practice nurse, 50% health care assistant paid £18 and £10 per hour respectively
Patient contact = 80%
Clinician has 80% patient contact
38% proceed to have FPG test
cholesterol test= £4.20
administration cost per vascular check= £4.70
Fasting Plasma Glucose test cost= £6.10
OGTT test cost= £12.30

Table viii-5: Department of Health model assumptions for the risk factor interventions

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Uptake</th>
<th>Compliance</th>
<th>Attribution‡</th>
<th>RRR of CVD</th>
<th>Cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking</td>
<td>19%</td>
<td>15%</td>
<td>51%</td>
<td>0.36</td>
<td>£ 176.80</td>
</tr>
<tr>
<td>Anti-Hypertensive</td>
<td>40%</td>
<td>87%</td>
<td>24%</td>
<td>0.24</td>
<td>£ 29.64 (£70†)</td>
</tr>
<tr>
<td>Exercise</td>
<td>77%</td>
<td>23%</td>
<td>63%</td>
<td>0.14</td>
<td>£ 33.50</td>
</tr>
<tr>
<td>IGT lifestyle</td>
<td>85%</td>
<td>90%</td>
<td>90%</td>
<td>0.09</td>
<td>£ 462.00</td>
</tr>
<tr>
<td>Statin prescription</td>
<td>85%</td>
<td>70%</td>
<td>50%</td>
<td>0.31</td>
<td>£ 60.52</td>
</tr>
<tr>
<td>Weight management</td>
<td>85%</td>
<td>68%</td>
<td>47%</td>
<td>0.36</td>
<td>£ 51.00</td>
</tr>
</tbody>
</table>

† if aged 70 or over
‡ attribution is the proportion of referral that will is attributed to the NHS Health Checks and would not have already occurred in general practice

Table viii-1; comparison of the population characteristics of the total population and those with recorded CVD risk factor data

<table>
<thead>
<tr>
<th></th>
<th>Age (mean; SE)</th>
<th>% Male (SE)</th>
<th>% South Asian (SE)</th>
<th>% Black (SE)</th>
<th>Median IMD (IQR)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total population</td>
<td>54.3 (0.07)</td>
<td>44.4 (0.35)</td>
<td>5.74 (0.16)</td>
<td>4.31 (0.14)</td>
<td>3 (2)</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>54.8 (0.11)‡</td>
<td>44.6 (0.56)</td>
<td>5.65 (0.26)</td>
<td>3.47 (0.21) ‡</td>
<td>3 (3) ‡</td>
</tr>
<tr>
<td>BP recorded</td>
<td>54.1 (0.87)†</td>
<td>44.6 (0.44)</td>
<td>5.21 (0.20)†</td>
<td>3.52 (0.17) ‡</td>
<td>3 (2) ‡</td>
</tr>
<tr>
<td>BMI recorded</td>
<td>54.3 (0.74)‡</td>
<td>44.5 (0.37)</td>
<td>5.56 (0.17)</td>
<td>3.83 (0.14) ‡</td>
<td>3 (2) ‡</td>
</tr>
<tr>
<td>FH recorded</td>
<td>53.0 (0.78)‡</td>
<td>43.6 (0.42)</td>
<td>7.14 (0.22)‡</td>
<td>4.40 (0.17) ‡</td>
<td>3 (2)</td>
</tr>
</tbody>
</table>

†p <0.05 (t-test* comparing those with recorded data to the total population)
‡p<0.01 (t-test* comparing those with recorded data to the total population)
*Chi squared test for categorical data
Appendix II Publications and Outputs from thesis

Journal Articles


ARHD obtained data from NHS Ealing, constructed and maintained the dataset; carried out all statistical analyses drafted the paper and interpreted the data


ARHD planned the paper; obtained data from NHS Ealing, constructed and maintained the dataset; carried out all statistical analyses drafted the paper and made the significant contribution to interpretation of data


ARHD planned the paper; obtained data from NHS Ealing, constructed and maintained the dataset; carried out all statistical analyses drafted the article and interpreted all findings


ARHD managed all HSfE data, carried out the modelling, data analysis and mapping; drafted the article and made the significant contribution to interpretation


ARHD planned and wrote the paper, and made the major contribution to content


ARHD planned the paper; managed all HSfE data and undertook all analyses; drafted the article and made the significant contribution to interpretation

ARHD planned the paper; obtained data from NHS Ealing, constructed and maintained the dataset; carried out all statistical analyses drafted the article and interpreted findings

**Presentations**


Dalton ARH. NHS Health Check programme; early findings from NHS Ealing and national lessons. NHS Ealing public health seminar, September 2011.


Chapter ix: References


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