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REVIEW

Ethnicity and cardiovascular disease prevention in the United Kingdom: a practical approach to management

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The United Kingdom is a diverse society with 7.9% of the population from black and minority ethnic groups (BMEGs). The causes of the excess cardiovascular disease (CVD) and stroke morbidity and mortality in BMEGs are incompletely understood though socioeconomic factors are important. However, the role of classical cardiovascular (CV) risk factors is clearly important despite the patterns of these risk factors varying significantly by ethnic group. Despite the major burden of CVD and stroke among BMEGs in the UK, the majority of the evidence on the management of such conditions has been based on predominantly white European populations. Moreover, the CV epidemiology of African Americans does not represent well the morbidity and mortality experience seen in black Africans and black Caribbeans, both in Britain and in their native African countries. In particular, atherosclerotic disease and coronary heart disease are still relatively rare in the latter groups. This is unlike the South Asian diaspora, who have prevalence rates of CVD in epidemic proportions both in the diaspora and on the subcontinent. As the BMEGs have been underrepresented in research, a multitude of guidelines exists for the 'general population.' However, specific reference and recommendation on primary and secondary prevention guidelines in relation to ethnic groups is extremely limited. This document provides an overview of ethnicity and CVD in the United Kingdom, with management recommendations based on a roundtable discussion of a multidisciplinary ethnicity and CVD consensus group, all of whom have an academic interest and clinical practice in a multiethnic community.
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Introduction

The United Kingdom is a diverse society with 7.9% (4.6 million) of the population from black and minority ethnic groups (BMEGs) (Supplementary Table W1). The latter is a heterogeneous group residing in all parts of the UK but clustering in the major metropolitan areas.[1]

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Defining ethnicity

There has been considerable debate on the concepts of ethnicity and race both within health services and among social scientists.[2–4] Ethnicity derives from the Greek word ethnos, meaning a nation and is a complex, multidimensional social construct that reflects self-identification with cultural traditions and social identity and boundaries between groups. It embodies one or more of the following:[5]

- shared origins or social background,
- shared culture and traditions that are distinctive, maintained between generations, and lead to a sense of identity and group and
- common language or religious traditions.

It is also usually a shorthand term for people sharing a distinctive physical appearance (skin colour) with ancestral origins in Europe, Asia, Africa or the Caribbean. This definition reflects self-identification with cultural traditions and social identity and boundaries between groups. Several authors[3,5] have stressed the dynamic nature and fluidity of ethnicity as a concept.

Operationalizing ethnicity

Given the importance of ethnicity on health, there are pragmatic grounds for assigning people into ethnic groups. The benefit of collecting data on ethnic group is to help reduce inequalities in health and health-care delivery.[6] Many have used proxy measures to define ethnic, for example, skin colour; country of birth; name analysis, family origin etc. Each of these has disadvantages and general consensus is that self-identified ethnicity is the way forward using, for example, the 2001 Census Ethnic Group question.[1,2] It is also worth noting the Census 2001 question was significant as it asked questions on people of Irish descent and mixed parentage for the first time.

It follows that for research purposes, investigators should collect data on many factors to define ethnicity accurately.

Demographic profile

The largest minority ethnic group is the Asian or Asian British category (50.2%) followed by the Black or Black British (24.8%). For the first time, the 2001 census also recorded that there were 677 117 (1.2%) people belonging to the 'Mixed' ethnic group category – and this is nearly as big as the Pakistani category (see Supplementary Table W1). It is worth noting that the average age of minority ethnic communities are younger than the White European population.[1]

Epidemiology of CVD

Cardiovascular disease (CVD) is the most common and yet one of the most preventable causes of death in the Western World. Economic development in Asia and rapid urbanization in Africa are associated with rapid changes in lifestyle and environmental exposures, so that the burden of CVD is rising rapidly in developing countries.[7]

Relative to white European populations, people of African origin, both Caribbeans and West Africans, have a high incidence of stroke and end-stage renal failure, whereas coronary heart disease (CHD) is less common[8–10] – around half the rate found in the general population for men and two-thirds of the rate for women. On the other hand, South Asian Indians from the Indian subcontinent and from East Africa have a much higher incidence of CHD.[8–10] Further examination on the mortality from CHD in South Asian populations overseas indicate that the high risk appears to be a feature of these populations across the world, whether populations are indigenous or diaspora. Similarly, the mortality from stroke in African Americans is high, whether born in the US or in the Caribbean.[3,4] Indeed, South Asians living in the UK, have a 50% greater risk of dying prematurely from CHD than the general population.[1] Of note, the poorest groups, particularly Pakistanis and Bangladeshis, have the highest mortality rates. Also, the disparity between South Asians and the rest of the population may be increasing. This is because the death rate from CHD is not falling as fast in South Asians as it is in the rest of the population. From 1971 to 1991, the mortality rate for 20–69 year olds for the whole population fell by 29% for men and 17% for women, whereas in South Asians it fell by 20% for men and 7% for women.[11] However, a more recent analysis of secular trends in the past 15 years, death from acute myocardial infarction (AMI) among South Asians appears to have declined at a rate similar to that seen in white patients, largely caused by reductions in indices of infarct severity (segment on the ECG (ST) elevation, peak creatine kinase, Q wave development on the electrocardiograph and treatment with thrombolytic therapy).[12]

The two main ethnic groups within the UK, Blacks and South Asians, are at particularly high risk of CVD, although there are ethnic differences in the prevalence of CVD conditions and cardiovascular (CV) mortality.[1] It is also important to note that mortality and morbidity from end-stage renal disease varies by ethnic group, with a much greater burden in both people of African origin and of people from the Indian subcontinent, even among those living in the United Kingdom.[13] The prevalence of CV risk factors and CHD in the UK Chinese population has been reported to be low.[14]

With respect to gender, note that the patterns for CV morbidity and mortality in men and women differ, with Bangladeshi and Pakistani men being top of the league whereas in women, the Indian and Irish predominate.

In relation to cerebrovascular disease, this is highest among the African Caribbean (AC) populations and high rates are also seen in Chinese and South Asian groups. The major known risk factor for stroke, hypertension, is common in ACs but not in the Chinese or South Asian populations.

The causes of the excess CVD and stroke morbidity and mortality in BMEGs are incompletely understood though recent work[14–17] indicates that socioeconomic factors are important. However, the role of classical CV risk factors is clearly important despite the patterns of these risk factors varying significantly by ethnic group.[1,18] Irrespective of these inter-ethnic differences in vascular diseases, CV morbidity and mortality are still much higher among these BMEGs when compared to the white European population[1] (Supplementary Table W2).

Ethnicity and management of CVD: the evidence base
Despite the major burden of CVD and stroke among BMEGs in the UK, the majority of the evidence-base on the management of such conditions has been based on predominantly white European populations. For AC, some evidence is available from data in African Americans, or for Chinese, from far Eastern studies. Moreover, the CV epidemiology of African Americans does not represent well the morbidity and mortality experience seen in black Africans and black Caribbeans both in Britain and in their native African countries. In particular, atherosclerotic disease and CHD are still relatively rare in the latter groups. Many of the large randomized controlled clinical trials that have informed our everyday clinical practice and primary/secondary prevention guidelines have similarly been performed in North American or European populations. To date, the BMEGs have been under-represented in research.[19]

As a consequence, a multitude of guidelines exists for the 'general population.' However, specific reference and recommendation on primary and secondary prevention guidelines in relation to ethnic groups is extremely limited.

Scope and search strategy

This document provides an overview of ethnicity and CVD in the United Kingdom, based on a roundtable discussion of a multidisciplinary ethnicity and CVD consensus group, which included multidisciplinary representation from experts in cardiology, diabetes, vascular surgery, primary care, public health and CV prevention, all of whom have an academic interest and clinical practice in a multiethnic community. This document is intended to be evidence based, recognizing that where there is a paucity of evidence in relation to ethnicity, some consensus was needed, although justification should be provided for any recommendation. It was decided to limit discussion to the main UK ethnic groups, that is, South Asians and ACs, while recognizing that there is heterogeneity within each, for example, Indian vs Pakistani vs Bengali, or African vs West Indies. As there is no definitive evidence for genetically predetermined risk for CVD as opposed to propensity to diabetes mellitus (DM) and impaired glucose tolerance (IGT), this document does not include genetic influences on CVD owing to the extent of controversy and lack of evidence.

For each section, we performed a literature search by using electronic bibliographic databases (i.e. MEDLINE, EMBASE and DARE), scanning reference lists from included articles and hand searching abstracts from national and international CV meetings. For the search, we had used the term 'ethnic', 'ethnicity', 'race' etc, in relation to subtopics related to CV prevention and cardiac rehabilitation that are covered in this overview. Bibliographies of all selected articles and review articles were reviewed for other relevant articles. Finally, the supplements of major journals were used to identify relevant abstracts that had not been published as peer reviewed articles. Where necessary, study authors were contacted to obtain further data. Where there was no specific evidence in ethnic groups, some extrapolation from other data in general populations was made. Where recommendations are made, these are tabulated and graded for the general population and for BMEGs. The grades of recommendations used are summarized in Supplementary Table W3.

Current guidelines for ethnicity and CVD prevention

Primary prevention of CVD in ethnic groups
The objective of primary prevention of CVD is to improve quality of life and life expectancy, by virtue of reducing the risk of index and subsequent CV events and interventions. Primary prevention of CVD commences with identification of a population at risk of CVD and identification and acceptance of a threshold of risk for intervention with evidence-based strategies. The need for guidelines with respect to CVD prevention is based upon the observations that

- CVD is the major cause of mortality and morbidity worldwide and will continue to be for the foreseeable future.
- Risk factor modification may attenuate or prevent clinical manifestation of atheromatous disease.
- Risk factor modification is possible in most patients.

The Framingham cohort study identified seven major risk factors of age, sex, blood pressure (BP), glucose intolerance, total- and high-density cholesterol, smoking and left ventricular hypertrophy[20] and these are often used to calculate risk at population and individual level. However, the use of Framingham for risk assessment is fraught with problems in the UK. For example, Framingham models underestimate prevalence of CVD in UK, as the data (over 20 years old) is derived from a white European middle class North American population with virtually no ethnic representation or individuals from low socio-economic subgroups. Deprivation, a currently debated risk factor, may be inextricably linked with ethnicity and therefore the Framingham model is unlikely to serve these populations adequately and thus, explain to some extent the inter-ethnic differences.

More recent than any Framingham evaluation, the INTERHEART study revealed that over 90% of the global burden of myocardial infarction (MI) could be accounted for by nine classical risk factors.[18] In order of potency, these were apolipoprotein B:A ratio (a marker of blood low-density cholesterol

(LDL):high-density cholesterol (HDL)), smoking, diabetes, hypertension, abdominal obesity (designated by waist:hip ratio), diet (fruit, vegetable and alcohol consumption), physical activity and psychosocial stress. Such 'classical' risk factors form the basis for many local guidelines aiming to tackle prevention strategies at a grassroots level.

More formal published guidelines accommodate a similar repertoire of risk factors to those evaluated by INTERHEART. Some guidelines use a more triaged quota of risk factors, for example, the European guidelines use only age, sex, smoking, total cholesterol and systolic BP (SBP).[21]

Although it is assumed that risk factors identified from studies on populations with established CHD correlate with risk factors for populations without established disease, this may not necessarily hold true. However, despite this reservation, Framingham is essentially one of the few risk prediction engines, which have promulgated risk factors from a true cohort study and the subsequent Framingham model is accepted to be a valid risk assessment tool. It is however now widely accepted that CV risk prediction models for identifying patients at increased risk of CV events (greater than 20% risk of CVD over 10 years) for high-risk populations are lacking. With regards to ethnicity, the current consensus opinion (http://www.sahf.org.uk/publications.html) reflects the lack of cohort studies in ethnic populations to guide prevention strategies.

Current risk prediction tools are neither sufficiently sensitive nor specific to recommend their widespread application in the UK population. The Framingham model underestimates risk in the UK South Asian population, an ethnic group with a documented high prevalence and mortality from CHD. Models such as FINRISK[22] provide similar predictions to Framingham, whereas applicability of the SCORE model,[21,23] even for the general population, is limited by the lack of inclusion of HDL and diabetes in this risk prediction tool. Recently a new web-based risk calculator, ETHRISK (available at http://www.epi.bris.ac.uk/CVDethrisk/), has been proposed which attempts to improve the estimates of risk in BMEGs for practical use in primary prevention.[24]

Population screening, whether opportunistic or systematic, requires the use of validated and reliable instruments for the quantification of CV risk in high-risk ethnic populations. Various recommendations exist from adding a 40–50% weighting to calculated Framingham risk in accordance with increased mortality rates seen in these patients or adding 10 years to the age of the patient.[25,26] Based upon this latter risk assessment, the calculated risk for non-diabetic South Asians is best adjusted by multiplying it by 1.79.

In addition to classical risk factors are the more controversial risk factors which to date, await widespread acceptance and subsequent inclusion in validated models of risk assessment. The metabolic syndrome, microalbuminuria, waist circumference, psychosocial stress, haemostatic factors and homocysteine name but a few of these more controversial factors.

Currently available guidelines with specific reference to ethnicity

NSF for CHD. Chapter 1 of the UK National Service Framework (NSF) for CHD (NSF) (www.doh.gov.uk/publications) focusing on reducing heart disease in the population, states that 'National Health Survey (NHS) and partner agencies should develop, implement and monitor policies that reduce the prevalence of coronary risk factors in the population and reduce inequalities in risk of developing heart disease.' With ethnic inequalities in the prevalence of CHD being of significance in the UK, such instruction is pertinent. Furthermore, Chapter 2 of the NSF, concerned with reducing heart disease in the population, proceeds to state that 'General practitioners and primary care teams should identify all people at significant risk of CVD but who have not yet developed symptoms and offer them appropriate advice and treatment to reduce their risk.' Although being far from a guideline, this reveals that there is a significant drive to target inequalities in CVD and that the targeting of high-risk ethnic groups is given importance within the NSF.[27] Targets of ethnicity coding within primary care within the UK Quality Outcomes Framework for Primary Care have only recently been introduced.

JBS-2. Published in December 2005, these revised guidelines suggest equitable prevention strategies are applied to individuals with established CVD, DM and those at high risk (>20% 10-year risk) of CVD.[25] Owing to the high prevalence of diabetes and IGT in South Asian populations, the impact of these guidelines will inevitably be far-reaching in this ethnic minority population. The Joint British Societies Guidelines (JBS) guidelines suggest ethnicity is included as part of the risk assessment exercise in all adults over the age of 40 years.

However, where this is factored into the JBS risk prediction charts is unclear, as the guidelines, despite suggesting adding 40% to a Framingham calculated risk score (accepting Framingham-based calculation as a reasonably valid assessment tool for the general population with respect to performance criteria) for South Asians proceed to state that 'CV risk prediction charts have not been validated in ethnic groups other than white Europeans and therefore they should be used with some caution. Unlike most guidelines on primary prevention however, awareness of select ethnic populations as high risk is specifically addressed with regards to risk prediction. The guidelines state that for South Asian patients, a factor of 1.4 is applied to the estimated 10-year risk derived from the JBS risk tables owing to the reported 40% increased risk of

CHD in this ethnic population when compared to the general population.[28–32]

This would mean that, a South Asian patient would require only a 14% 10-year risk to qualify for primary prevention according to JBS guidelines. On the contrary, the guidelines then state that after accounting for diabetes, there is no increased risk in South Asians. However, this statement requires further evaluation and these data are derived from a review[33] rather than a true epidemiological cohort study or risk modelling. One would be advised to adhere to the guidance elsewhere in the JBS document classifying South Asians as a high-risk ethnic group. The JBS guidelines also suggest that Asian males over 45 years and women over 55 years qualify for over-the-counter simvastatin use, by virtue of ethnic origin being a risk factor for CHD, though the evidence base for this is unvalidated.

AC populations are identified as being at lower risk of CHD but higher risk of hypertension and stroke in the JBS guidelines. Culturally appropriate lifestyle advice is advocated along with validated therapies in this population.

Joint European Societies Guidelines. The Joint European Societies Guidelines[21,34] have been promoted as the definitive pan-European CV prevention guidelines but fail to mention ethnicity at all. In fact, even in the section pertaining to metabolic syndrome, there is no documentation of the ethnically sensitive definitions for waist circumference or body mass index.

NZ guidelines. The most recent New Zealand (NZ) Census in 2002 found that the NZ ethnic population was dominated by the Mâori (14.5%), followed by people from the Pacific Islands (5.6%), Chinese (2.2%) and Indian (1.2%). Similar to South Asian populations elsewhere in the developed world, the NZ South Asian population has among the lowest rate of physical activity and the highest rates of obesity, treated dyslipidaemia and diabetes. In this guideline (http://www.nzgg.org.nz/guidelines/dsp_guideline_popup.cfm?guidelineCatID=53&guidelineID=35), recommended ages for initiating CV risk assessment incorporate some degree of ethnic specificity, as follows: (i) Maoris, Pacific peoples and South Asians: 35 years (M), 45 years (F); (ii) Known CV risk factors or people with diabetes: 35 years (M), 45 years (F); and (iii) Others at 45 years (M) and 55 years (F).

Because of this age stratification for screening the NZ guidelines are the most cost effective of national guidelines.[35] With low, moderate, high and very high risks being categorized as 5-year CVD risks of $<10\%$, 10–15%, 15–20% and $>20\%$, respectively, levels of risk are also defined at thresholds which inevitably facilitate the provision of preventative strategies for all high-risk populations.

Where the Framingham risk score is used in NZ, it is suggested that the following ethnic groups should be moved up one risk category (5%), as an acceptance that their CV risk is underestimated in the Framingham risk equation: Mäori; Pacific peoples or people from the Indian subcontinent; and people with the metabolic syndrome (which is more prevalent in those of South Asian descent).

Thresholds for specific interventions

It is now widely accepted that overall risk assessment should drive intervention strategies based upon overall CV risk (see http://www.dh.gov.uk/PolicyAndGuidance/HealthAndSocialCareTopics/CoronaryHeartDisease/fs/en). However, the normal ranges derived from studies in white European populations may be set so high that interventions driven by these ranges could result in undertreatment and inequity in high-risk groups. The high absolute risks of coronary, cerebrovascular and renal disease in some BMEG populations strongly suggest that there should be different thresholds for their use in these groups.

Let us take hypertension, as just one illustrative example. The British Hypertension Society (BHS) guidelines suggest that people with severe hypertension (BP consistently $\geqslant 160/100$ mm Hg) or those with mild-to-moderate hypertension (BP consistently between 140–159/90 and 99 mm Hg) and either diabetes, target organ damage or CV complications should be treated with antihypertensive therapy.[36] However, in the 1999 version of the guidelines,[37] those with mild-to-moderate hypertension without diabetes, target organ damage or CV complications, the decision to treat was made on the basis of a 10-year Framingham CHD risk estimate $\geqslant 15\%$ (assumed equivalent to a 10-year CVD risk $\geqslant 20\%$). Although the latter equivalence holds in white European populations, it does not for ethnic populations, as CHD risk underestimates CVD risk. Indeed the use of a 10-year risk of CHD $\geqslant 15\%$ to decide how to manage people with mild-to-moderate uncomplicated hypertension would identify for treatment 91% of white European people but only 81% of South Asians and people of African origin (Figure 1). Using a lower threshold for CHD risk, for example, 12 and 10% in South Asians and in AC respectively, with mild-to-moderate uncomplicated hypertension, delivers a higher probability of identifying and treating those with a 10-year CVD risk $\geqslant 20\%$ (Figure 1). However, the use of the CVD risk would be even better measurement.[38]

The latest BHS-IV guidelines have amended the criteria, now recommending the use of CVD risk as a guide to hypertension management in the mild-to-moderate group to avoid inequalities across hypertensive patients of different ethnic background.[36]

In summary, there is a general paucity of specific guidelines for the prevention of CVD in ethnic populations and of more concern, despite the ethnic minority population often harbouring high-risk populations, reference to ethnicity in existing guide-

lines is sparse. This pertains to the paucity of representation of these ethnic groups in major studies assessing the value of risk-reducing interventions. Consequently, individuals from ethnic groups may be inadequately targeted for risk-reduction strategies, including screening and treatment for dyslipidaemia, hypertension and diabetes etc.

Smoking cessation

Tobacco smoking is an established risk factor for CVD. The prevalence of smoking varies significantly in BMEGs and among subgroups owing to religion, customs, traditions, languages, education, economic status, social acceptance of smoking, attitudes and beliefs. In addition, the 'chewing' of tobacco and related products is common in certain communities.

Smoking cessation strategies include non-pharmacological and pharmacological tools. Non-pharmacological approaches to quitting smoking, such as physician advice, individual or group counselling, proactive telephone counselling or self-help, are generally more effective than no intervention, but the effects are modest.[39]

Pharmacotherapy for smoking cessation includes nicotine replacement therapy (NRT), bupropion and nortriptyline.[40] NRT is recommended first-line therapy as long acting patches or short acting gums, inhalers, nasal sprays and sublingual tablets/lozenges. The National Institute for Health and Clinical Excellence (NICE) endorses their use and most preparations are available in the NHS. Bupropion (mechanism of action is unknown), is also recommended as first-line therapy in the UK whereas nortriptyline, an antidepressant, is recommended as second-line therapy.[41]

The 1999 Health Survey for England reveals marked differences between ethnic groups and gender for smoking prevalence, with Bangladeshi males having the highest smoking prevalence (44%) and Black Caribbean males having a rate that is higher than the national average (35% compared to 27%). Observational studies indicate that quit attempts and sustained quit rates are lower among South Asians. Reasons may be found in low levels of awareness of the health risks associated to smoking, the addictiveness of cigarettes, the availability of management tools, poor socio-economic conditions and low self-esteem.

Smoking cessation services collect data on ethnicity in very broad ethnic groupings and from this it appears that ethnic minority smokers are under-

Current guidelines and applicability to ethnicity

Guideline	*Population*	*Grades of recommendations for general population*	*Grades of recommendations for BMEG applicability*
Joint British Societies	UK	A to D	D
Joint European Societies	Pan-European	A to D	D
New Zealand	New Zealand	A to D	D
National Service Framework for CHD	UK	A to D	D

Abbreviations: CHD, coronary heart disease.

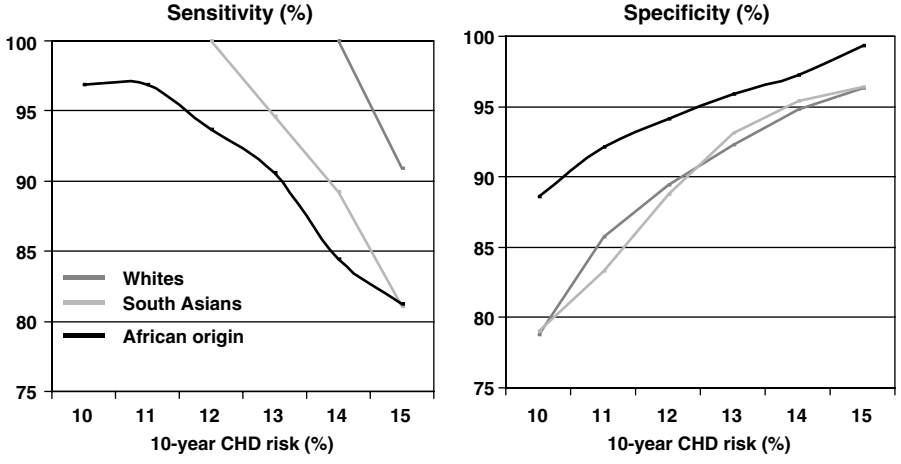

Figure 1 Sensitivity and specificity of thresholds for 10-year risk of CHD to identify 10-year risk of CVD $\geqslant$20% in different ethnic groups after exclusion of people with diabetes, target organ damage and CV complications. Adapted from Arabi and Jackson [26].

represented as attendees at these services. Although 2.3% of smokers in England could be categorized as 'Asian', only 1% of the users of the services are in this ethnic group.[42–44] There is some evidence that smoking cessation services to ethnic minority groups can be effective in the short term.

There is high-quality systematic review evidence (Cochrane reviews, see http://www.thecochranelibrary.com/) on the effectiveness of health care professional advice to stop smoking, individual behavioural counselling, group behaviour therapy programmes, NRT and antidepressants for smoking cessation. These reviews include studies from many different countries, but predominantly the USA, and the majority of included studies did not include a breakdown of participant ethnicity. Recent guidelines on brief interventions for smoking cessation issued by NICE (www.nice.org.uk) highlight the lack of evidence for effectiveness of interventions by ethnicity and the lack of information about effective interventions for smokeless tobacco use.[45]

Recommendations in BMEG subjects who smoke

	Recommendation	
	General	*BMEGs*
Quitting smoking Physician advice, individual or group counselling, proactive telephone counselling or self-help should be offered to all smokers	A	D
Nicotine replacement therapy should be offered to all smokers unless there is an absolute or relative contraindication.	A	D
Bupronion and nortriptyline should be offered to smokers who do not quit with the previous tools unless there is an absolute or relative contraindication.	A	D

Abbreviation: BMEG, black and minority ethnic group.

Hypertension

There is consensus that among people of African origin hypertension is three to fourfold more prevalent than in the UK white European population.[46–48] This is true for men and women and is present at any age, at least in adulthood. This observation fits with the excess risk of stroke and renal disease in these populations.[49] African Americans also show an increase in the prevalence and severity of hypertension,[50] as do AC[51] and urban[50] or rural[51] black African populations. Hypertension has also been associated with all-cause mortality in rural Africa[52] and with vascular and renal complications.[53]

The prevalence of hypertension appears to be significantly higher in some studies of South Asian immigrants in the UK compared to Europeans[54,55] and in both rural and urban populations living in India.[56,57] This contrasts with some reports both in the UK and in the South Asian groups living in Tanzania.[58,59] The high prevalence of hypertension in some South Asian groups and in people of African origin, particularly in view of their low smoking rates, is likely to contribute to the high mortality from stroke experienced by these groups in the UK.[11,60]

Detection

There is evidence to suggest that the 'rule of halves' by which only a small proportion of people with hypertension receive appropriate management and adequate control, has improved over the years.[61] People of Black African origin in the UK are more likely to have their hypertension detected in the community when compared to other ethnic groups.[54] This may indicate greater awareness among patients and doctors of the importance of controlling hypertension in black populations. However, black populations tend not to achieve a good BP control.[54] This may indicate either a greater severity of hypertension, inadequate drug therapy owing to individual sensitivity to different drugs, lack of concordance with therapy, doctors' perceptions and organizational pitfalls.

It is now established that the quality of hypertension control predicts mortality from stroke.[62] Furthermore, stroke-related events in people with known hypertension but with inadequate BP control might be regarded as unacceptable and potentially avoidable events, accepting that the majority of such individuals will not experience a stroke and hence the number needed to treat in order to prevent one such event is unknown but for that individual experiencing a stroke, it might have been potentially avoidable.[62]

Prevention and non-drug therapy

The first step for the prevention and management of high BP is non-pharmacological.[63] For hypertension, it is particularly important to aim at a reduction of sodium intake to about 50–80 mmol/day (equivalent to 3–5 g of salt per day). This is most useful in the populations of African origin who are particularly sensitive to the detrimental effect of high salt intake and, for the same reason, respond best to a reduction in sodium intake.[64,65] Reduction in sodium intake and an adequate potassium intake by increasing the amount of fruit and vegetable should, for instance, be considered for the prevention of hypertension and stroke particularly in communities of black African origin both in the UK and also in Africa[66] where urbanization is associated with an increase in salt intake and a reduction in potassium intake.

Drug therapy

Once non-pharmacological treatment has failed to reduce BP to target. There are many 'targets' dependent upon the guideline referred to (e.g. currently $\leqslant$140/85 and $\leqslant$130/80 mm Hg in diabetics according to the BHS IV guidelines[36]; 140/90 mm Hg according to recently updated NICE guidance, formulated jointly with the BHS (issued 28 June 2006); 145/85 mm Hg for diabetic patients and 150/90 mm Hg for non-diabetic patients in the Quality Outcomes Framework, to name but a few), pharmacological treatment is required. Most of the evidence from randomized controlled clinical trials relies on data in African Americans and in other American ethnic groups. The updated NICE guidance reflecting recommendations from the South Asian Health Foundation with respect to ethnicity highlights the need for specific areas of research pertinent to ethnicity and hypertension:

- the clinical effectiveness of antihypertensive therapy in minority ethnic groups particularly the African and Asian origin populations,
- the adoption of quality of life measures within future clinical trial protocols of antihypertensive therapy to allow measures of drug class utility,
- concordance with antihypertensive therapy

A recent systematic review and meta-analysis of antihypertensive drug therapy in black patients has evaluated BP lowering efficacy of monotherapy.[67] For systolic BP the results showed a significant reduction with all drug classes (except beta-blockers), whereas for diastolic BP all classes were efficacious, although diuretics had the greatest efficacy. Although drugs differed in their efficacy, there was no solid evidence that morbidity and mortality outcomes differed once patients achieved the BP goals. This meta-analysis did not include the Africa-American component of the ALLHAT study,[68] the largest trial yet of monotherapy of hypertension. The ALLHAT results suggested that among black participants, there was a detectable additional relative risk (RR) reduction for stroke and heart failure in the group treated with a diuretic compared to those treated with an angiotensin-converting enzyme (ACE) inhibitor. Likewise, an additional RR reduction for heart failure in the group treated with a diuretic compared to those treated with a calcium-channel blocker.

Large trials in British ethnic minority groups are lacking. However, small studies have been carried out over the years contributing both to the understanding of mechanisms and logical combination therapies.[69]

As BHS-IV,[36] newer evidence has come forth, with significant changes in our approach to hypertension, necessitating revision of the original AB/CD algorithm, which is now superseded by the new A/CD algorithm (Figure 2), as recommended in the joint NICE and BHS guidelines. In this guideline update, beta-blockers were regarded as not ideal as first-line monotherapy in uncomplicated essential hypertension and should only be used in the presence of associated heart disease, for example, atrial fibrillation, left ventricular systolic dysfunction and angina etc. Calcium-channel blockers or thiazide-type diuretics were still considered to be the most likely drugs to confer benefit as first-line agents for most patients aged 55 or older. In people younger than 55 years, initial therapy with an ACE inhibitor (or angiotensin receptor blocker) was considered better than initial therapy with a calcium-channel blocker or a thiazide-type diuretic. When using more than one drug, adding an ACE inhibitor to a calcium-channel blocker or a diuretic (or vice versa) was recommended as a logical combination, as commonly done in clinical trials. AC patients tend to have lower levels of renin than white Europeans and in whom salt sensitivity is often increased. They may therefore respond less well to drugs that act on the renin–angiotensin system, such as beta-blockers,

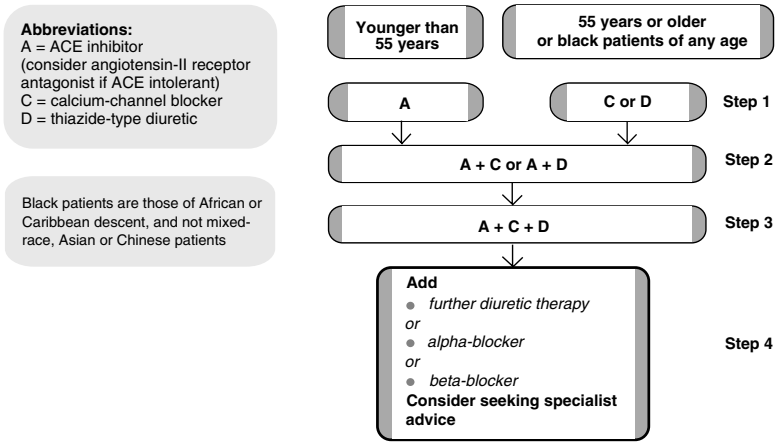

Figure 2 Choosing drugs for patients newly diagnosed with hypertension: The NICE/BHS Guidelines (see www.nice.org.uk/page.aspx?o=CG34).

ACE inhibitors and angiotensin receptor blockers (ARBs).[70–72] All drug management strategies should be implemented on a background of non-pharmacological management.

BP control in diabetes
The NICE guidelines for people with type II diabetes recommend a target BP of: $<140/80$ or $\leqslant 135/75$ mm Hg if they also have microalbuminuria or proteinuria.[70] The BHS guidelines are more aggressive with targets of: $<130/80$ mm Hg (or $<125/75$ mm Hg if proteinuria is present).[36]

Recommendations in BMEG subjects with hypertension

	Recommendation	
	General	*BMEGs*
Blood pressure control All patients with hypertension should undergo non-pharmacological management with focus on weight control, reduction in salt intake and increase in potassium intake.	A	B
All patients with hypertension should have CVD risk assessment carried out.	A	B
All patients with hypertension should have blood pressure reduced to below 140/85 mm Hg, or 140/80 mm Hg if patients also have type II diabetes.	A	A
In AC patients, thiazide diuretics or calcium-channel blockers should be used for initial treatment unless there is an absolute or relative contraindication.	A	B
A thiazide-type diuretic with a beta-blocker is not recommended as an initial therapy.	A	B

Abbreviations: AC, African Caribbean; BMEG, black and minority ethnic group; CVD, cardiovascular disease.

Dyslipidaemia

Serum cholesterol on the other hand, is not an obvious risk factor among migrant South Asians, especially when mean concentrations are compared with those among European populations. The concept of a putative 'metabolic syndrome' or 'insulin resistance' is thought to drive coronary risk long before the onset of clinically diagnosed diabetes,[73,74] and is characterized by dyslipidaemia (that includes moderately raised triglyceride (TG), low HDL cholesterol (HDL-C)), disordered glucose and insulin metabolism, raised BP and central obesity.

Hypercholesterolaemia among South Asians
Serum cholesterol levels among South Asian migrants appear lower or no different compared to the general UK population[75] and this information has been used to dispute whether LDL cholesterol (LDL-C) could explain the high rates of CHD mortality among Indian migrants. However, LDL-C levels among South Asian migrants living in Trinidad (4.2 mmol/l), were actually higher than those for the indigenous population.[76] Furthermore, in addition to diabetes, hypercholesterolaemia (serum cholesterol $\geqslant 6.5$ mmol/l) and raised TG were all significantly associated with a positive CHD history among South Asians in South Africa.[77] Certainly, unique migration studies have reported higher serum cholesterol among migrant groups compared to reference populations still living in regions of origin in India.[78,79] A large body of evidence from primary and secondary prevention trials have established that lowering LDL-C levels will lead to a substantial reduction in the risk of CVD events, though there is a paucity of data in ethnic populations.[80] Even with normal cholesterol levels, increased numbers of small dense LDL particles will increase the risk of CHD.[81] Small dense LDL may contribute to the heightened CHD risk among South Asians, as it usually occurs in the presence of raised TG and low HDL-C.

Low HDL-C and elevated serum TG
An independent and inverse relationship exists between CHD risk and HDL-C.[82,83] Low HDL-C and raised TG are common dyslipidaemic characteristics of South Asian populations[84–91] and appear to be more pronounced on migration from the Indian subcontinent.[78] By contrast, black children have been found to have mean TG levels which are lower and HDL-C levels which are higher than their white European counterparts.[85,86] This phenomenon is continued through adolescence with both sexes continuing to show similar patterns of lipoproteins, perhaps explaining the lower incidence of CHD in the black population.

There are some studies comparing black populations with their white counterparts as adults, which have been conducted across a number of populations. However, epidemiological surveys in the Caribbean comparing lipoprotein profiles in people of African descent with age-matched adults of white European background showed black people having lower TC and LDL-C than white European people.[87]

The term '*isolated low HDL-C*' has been adopted to describe the appearance of a normal serum or LDL-C together with low HDL-C and its prevalence, with and without the presence of elevated TG (>1.7 mmol/l), was greater among Indians living in Singapore compared to Chinese (odds ratio (OR) 2.88, $P<0.0001$) and Melanesian contemporaries.[92] In the absence of environmental differences to explain the ethnic prevalence, it was viewed that Indians had a genetic predisposition to isolated low HDL-C.

Although raised TG have been shown to be an independent risk factor for CHD, evidence from fibrate therapy trials is less convincing.[93] Raised TG may be a synergistic risk factor for CHD risk. A meta-analysis of 17 population-based studies reported that elevated TG were associated with a 30% increase in CVD in men and a 75% increase in women. When other risk factors such as HDL-C were controlled, the association was diminished but not to insignificance.[94] Overall, it is increasingly apparent that TG have an independent and synergistic influence on CHD.

Basis of elevated CHD risk and dyslipidaemia

There is some evidence of a genetic basis of CHD risk through other lipoproteins in South Asians. South Asians have high serum concentrations of Lipoprotein(a) (Lp(a)) in comparison to general populations of the US and UK, which might generate a synergistic risk to serum cholesterol.[95,96] Lp(a) is similar in lipid consistency to LDL, and high plasma concentrations of Lp(a) have been associated with an increased incidence of CHD.[97]

Management options

The South Asian population present a management challenge in the amelioration of risk factors contributing to their high prevalence of CVD. Although statins have been demonstrated to reduce CV events, it is notable that such reductions have been less when baseline HDL was lower.[98] However, there is a suggestion that the uptake of statin therapy among South Asian patients is lower than in other populations, suggesting inequalities in access to appropriate therapy.[99]

Current therapeutic options that may be considered in addressing low HDL and high TG include nicotinic acid and fibrate therapy, the former also having a useful Lp(a)-lowering effect. Although outcome data are limited with these drugs, the reduction of TG and increase of HDL-C has a beneficial effect on CHD events,[100] and this could be extrapolated to predict significant benefit in South Asian populations. For example, in the Veterans Affairs High-density lipoprotein cholesterol Intervention Trial (VA-HIT) enrolled men with existing CHD and a diabetic-like dyslipidaemia and randomized them to the fibric-acid derivative gemfibrozil or placebo.[100] At 5-year follow-up, the fibrate was associated with a 22% reduction in non-fatal MI or death from coronary causes. In those with significant hypertriglyceridaemia and low HDL, the combination of a statin and fibrate may be necessary. Other interventions such as insulin sensitizing agents and omega-3 fatty acids may in future play a part in the reduction of the huge CV burden of disease in this population but current evidence is lacking.

The recently published Fenofibrate Intervention and Event Lowering in Diabetes (FIELD) study has provided data on the use of fibrates in the management of people with type II diabetes.[101] The aim of this large intervention study (9795 patients) was to assess the effect of fenofibrate on CV events in people with type II diabetes; 78% of the study population was without prior CVD. Fenofibrate was associated with 11% fewer major coronary events (the primary outcome of the study), but this reduction was not statistically significant. Treatment with fenofibrate significantly reduced total CV events, mainly owing to a 24% reduction in the risk of non-fatal MI and a 21% reduction in the need for revascularizations, but there was no significant reduction in CV mortality or total mortality. Current guidelines, published before the results of the FIELD trial, recommend fibrates as an option for secondary, but not primary prevention.

Nicotinic acid is the most effective agent for raising HDL-C levels. In addition, it also has significant LDL-C- and TG-lowering effects.[102,103] The extended-release formulation of nicotinic acid, is promoted as an adjunct to statins in patients with low HDL-C, or as monotherapy in patients intolerant to statins. Extended-release nicotinic acid is a treatment option for patients with type II diabetes who exhibit the typical dyslipidaemia of high TG, small dense LDL-C and low HDL-C. Patients may experience a dose-related rise in blood glucose and frequent monitoring of blood glucose and liver function in all patients treated with extended-release nicotinic acid is advised by the manufacturer. Flushing is a common side effect with nicotinic acid, but symptoms can be ameliorated if aspirin is taken 30 min before treatment.

The JBS-2 guidelines discussed above suggest targets of TG (<1.7) and HDL target >1.0 mol/l for male and >1.2 mol/l for female subjects but there is no outcome evidence to support this at present. Of note, these JBS targets differ from those advocated in the GP Quality and Outcomes Framework and NICE guidelines.

Key areas for future research

There is an urgent need for outcome studies on lowering LDL-C and prevention of CVD in ethnic groups, particularly in South Asians as the major trials so far completed have failed to recruit sufficient of these populations to allow meaningful conclusions to be drawn. There is a particular need to undertake research into the effects of increasing HDL-C on the incidence of CHD particularly with regard to South Asians and black female subjects. With new drugs such as cholesterol ester transfer protein inhibitors and cannabinoid receptor antagonists on the horizon, such data is essential to guide appropriate therapy. Utilization of TG-lowering drugs such as nicotinic acid (which also increases HDL-C) in outcome trial in these populations should be undertaken.

Recommendations for dyslipidaemia in BMEG subjects

	Recommendation	
	General	*BMEGs*
Dyslipidaemia All patients at high risk should be treated with a statin	A	B
Achieve a target LDL-C of <2.0 mmol/l and TC of <4.0 mmol/l	D	D
Achieve a target HDL-C of 1.0 mmol/l for men and 1.2 mmol/l for women, utilizing fibrates or nicotinic acid as appropriate.	D	D
Achieve a target TG level of 1.7 mmol/l	D	D
If significant dyslipidaemia remains consider statin/fibrate of stain/nicotinic acid combination	C	D

Abbreviations: LDL, low-density lipoprotein; HDL, high-density lipoprotein; TC, total cholesterol; TG, triglyceride.

DM and metabolic syndrome

The prevalence of type II DM is increased in AC and South Asians compared with the general population and, in South Asians at least, tends to develop around 5 years sooner.[104] After standardizing for age, individuals of South Asian descent are 3–6 times more likely to have diabetes; for AC, the risk ratios are 2.5 for men and 4.2 for women.[104] Recent estimates for prevalence rates indicate that 20% of the South Asian community and 17% of the AC community has type II diabetes in contrast to 3% of the general population.[13]

In South Asians, there is evidence that a pattern of insulin resistance and associated metabolic abnormalities, the metabolic syndrome, might be responsible. Indeed, South Asians commonly have a characteristic clustering of risk factors that are representative of the metabolic syndrome, which may include three or more of the following: increased waist circumference, reduced HDL-C, increased TG, hypertension, and impaired fasting glucose or diabetes.[105,106] In addition to a lack of glucose regulation, it is very likely that non-esterified fatty acid (NEFA) metabolism is also disordered among South Asians, with poor NEFA suppression among South Asians, in both CHD patients and normal participants.[107,108] UK AC subjects, while having a tendency for insulin resistance and a high risk of diabetes, have a more favourable lipid profile and low risk of CHD and thus do not fit the typical definition of the metabolic syndrome.

Accepting the paucity of ethnic specific data for much of the management of patients with diabetes, there are many studies, which one must assume are applicable to all patients with diabetes, regardless of ethnic origin. The following interventions can improve CV outcomes in people with diabetes, although the cost effectiveness of such intervention on such a scale had not been established to date.

BP control in patients with DM

Recommendations for BP control have been summarized earlier. The use of ACE inhibitors to prevent CV events in patients with type II diabetes and established CHD, but with no evidence of heart failure was demonstrated in the diabetes subgroup of the Heart Outcomes Prevention Evaluation (HOPE) trial[109] and in the PERindopril SUbstudy in CHD and diabetes (PERSUADE) study,[110] which was a substudy of the large EUropean trial on Reduction Of cardiac events with Perindopril in stable CHD (EUROPA) trial.[111] Analysis of the diabetes cohort in the HOPE study demonstrated a statistically significant 25% RR reduction in the combined primary end point of MI, stroke and CV death with ramipril.[109] In the PERSUADE study, perindopril was associated with a nonsignificant 19% RR reduction in the primary end point of CV death, non-fatal MI and resuscitated cardiac arrest in patients with diabetes.[110] In both studies, the benefits appeared to be independent of BP lowering.

Evidence for the benefits of ARBs in reducing CV events in a high-risk population of diabetic patients with hypertension and left ventricular hypertrophy is also available. The Losartan Intervention For End point reduction in hypertension (LIFE) trial demonstrated a significantly greater reduction in the primary composite end point of CV morbidity and mortality for losartan compared with atenolol.[111,112]

Both hypertension and diabetes are leading risk factors for chronic kidney disease. ACE inhibitors and ARBs have also been shown to exert a renoprotective role, beyond what can be attributed to BP reduction. Activation of the renin–angiotensin–aldosterone system (RAAS), which results in increased angiotensin II production, raises systemic BP, a major contributor to renal disease initiation and progression. For this reason, ACE inhibitors and ARBs that target the RAAS should logically be the antihypertensive agents of choice in patients with diabetic nephropathy. Although ACE inhibitors and ARBs act on the RAAS in different ways, both types of agent prevent the progression from microalbuminuria to clinical proteinuria in type II diabetes.[109,113] ARBs may, however, provide better renoprotection in patients with overt nephropathy,[114–116] although equivalence between the two agents was reported in a recent study.[117] Ideally, therefore, all patients with diabetes and renal or CVD should be treated with ACE inhibitors or ARBs.

Up to one-third of patients with diabetes will require three or more agents to achieve effective BP control and the consequent CV benefit, which may include treatment with an ACE inhibitor or ARB, beta-blocker, calcium channel blocker or a diuretic. Initial monotherapy is generally selected according to age and ethnic group, as discussed above.[36] Beta-

blockers and thiazide diuretics may have adverse effects on glycaemic control in some patients and hence, their use may be limited. In the ALLHAT trial, new-onset diabetes was higher in patients receiving the diuretic, chlorthalidone, than in the other groups after 2 and 4 years of follow-up.[68] In the Anglo-Scandinavian Cardiac Outcomes Trial – Blood Pressure Lowering Arm (ASCOT-BPLA),[118] more patients developed new-onset diabetes in the atenolol group than in the amlodipine group. In patients with diabetes, ACE inhibitor/ARB–calcium channel blocker or ACE inhibitor/ARB–diuretic regimens are preferred when more than one agent is required.

Management of dyslipidaemia in patients with diabetes
For secondary prevention, there is therefore little debate about the benefits of statin therapy, whether or not patients have diabetes. For primary prevention, the NICE guidelines[70] recommend lipid-lowering drugs for people with type II diabetes based on an assessment of their absolute risk of a coronary event and whether or not they have adverse lipid profiles (defined as total cholesterol $\geqslant 5$ mmol/l, or LDL-C $\geqslant 3.0$ mmol/l or TG $\geqslant 2.3$ mmol/l). For patients with an adverse lipid profile, statins are recommended if they are at higher risk (history of CVD or 10-year coronary event risk $>15\%$).

The results of the Heart Protection Study (HPS)[119] and the Collaborative AtoRvastatin Diabetes Study (CARDS)[120] support statin use for primary prevention in people with diabetes regardless of their baseline cholesterol level. Many now argue that all patients with type II diabetes should be prescribed a statin. The BHS considers patients with type II diabetes who are aged 50 years or over (or who have been diagnosed for at least 10 years) as having the same risk as those with manifest CVD and recommend statins in all these patients if they have total cholesterol >3.5 mmol/l.[36] If the 50% increased risk of CHD in South Asians is taken into consideration, the corresponding threshold for statin therapy should be a 10-year risk of $>10\%$.

Fibrates and niacin may also play a part in managing the characteristic dyslipidaemia of the metabolic syndrome and/or diabetes, which is typical of the lipid profile of many South Asians. In the VA-HIT,[100] the benefit among patients with diabetes was similar to that observed in the total cohort.

Aspirin in patients with diabetes
There again is a paucity of data in relation to ethnicity but in high-risk patients, the Antithrombotic Trialists' Collaboration meta-analysis of randomized trials of antiplatelet therapy for prevention of death, MI and stroke revealed that the absolute benefits of antiplatelet therapy substantially outweighed the absolute risks of major extracranial bleeding.[121] Aspirin was the most widely studied antiplatelet agent and daily doses of 75–150 mg appeared to be as effective as higher doses for long-term treatments. Subjects with diabetes had risk reductions that were comparable to non-diabetic individuals. For example, the Hypertension Optimal Trial (HOT) demonstrated a 15% risk reduction for CV end points in patients with type II diabetes treated with low-dose aspirin, albeit with increased risk of (non-fatal) haemorrhage.[122] In practice, the evidence suggests that all patients with type II diabetes over 50 years of age should be treated with low-dose aspirin (usually 75 mg) once BP is controlled (systolic <150 mm Hg). The SBP level of <150 mm Hg was based on the Medical Research Council (MRC) trial, where aspirin was shown to be more beneficial in patients with lower SBP at entry.[123] If aspirin is given to people with higher BP, they may be exposed to the risks of bleeding without deriving benefits in the reduction of CV events.

Recommendations in BMEG subjects with diabetes

Blood pressure control	*Recommendation*	
	General population	*UK South Asian/AC*
All patients with diabetes should have blood pressure reduced to below 140/80 mm Hg, or 135/75 mm Hg if patients also have renal disease.	A	B
ACE inhibitors or ARBs should be considered as first choice agents in patients with type II diabetes	A	B[a]
A thiazide-type diuretic with a beta-blocker is not recommended as an initial combination for patients at increased risk of developing type II diabetes.	B	C

Abbreviations: AC, African Caribbean; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blockers.
[a]In AC patients, ACE inhibitors or ARBs may be less effective. A diuretic should be used for initial treatment unless there is an absolute or relative contraindication.

All patients with diabetes should be treated with statin therapy regardless of baseline cholesterol level.	B	C
All patients with CVD and very high TG levels (>2.3), but normal TC and LDL-C levels, should be offered a fibrate.	A	B
In patients with diabetes and a dyslipidaemia characteristic of the metabolic syndrome, consider a statin in combination with a fibrate.	B	C
Low doses of extended-release nicotinic acid may be considered as an alternative to statins or fibrates for patients with diabetes in whom these agents are not tolerated or fail to sufficiently correct low HDL-C levels or very high TG levels.	B	C

Abbreviations: CVD, cardiovascular disease; LDL, low-density lipoprotein; HDL, high-density lipoprotein; TC, total cholesterol; TG, triglyceride.

Aspirin	*Recommendation*	
	General population	*UK South Asian/AC*
Aspirin should be given routinely and continued long-term in patients with diabetes and CHD.	A	B
Aspirin should be considered for people with type II diabetes with a 10-year CHD risk >15%, providing systolic blood pressure ⩽145 mm Hg.	B	C
Aspirin should be considered for people <50 years of age with type II diabetes and well-controlled hypertension if there is a history of CVD or at least one other CV risk factor.	B	C

Abbreviations: AC, African Caribbean; CHD, coronary heart disease; CVD, cardiovascular disease.

Ischaemic heart disease

Many previous studies have documented sex and ethnic disparities in outcomes after AMI,[124] but the explanation of these disparities remains limited – although social, personal, and medical factors could explain sex and ethnic disparities in prognosis after AMI. For example, in a prospective cohort study of 20 263 men and 10 061 women of an integrated health-care delivery system in northern California who had experienced an AMI, age-adjusted analyses demonstrated that black men (hazard ratio (HR) relative to White European men, 1.44; 95% confidence interval (CI), 1.26–1.65), black women (HR, 1.47; 95% CI, 1.26–1.72) and Asian women (HR, 1.37; 95% CI, 1.13–1.65) were at increased risk of AMI recurrence.[125] However, multivariate adjustment for sociodemographic background, comorbidities, medication use, angiography and revascularization procedures effectively removed the excess risk of AMI recurrence in these ethnic groups. Older data from North America suggest that African Americans and White Europeans had a similar presentation and natural history of AMI and similar access to most medical care and cardiac procedures; however, the rate of coronary artery bypass procedures was much lower among African Americans than among White Europeans.[126] Indeed, the possibility has been raised that some of the ethnic disparity in management can be accounted for by the specific hospital to which patients were admitted, in contrast to differential treatment by ethnicity inside the hospital.[127]

In a UK study, South Asian patients had a higher risk of admission with AMI and a higher risk of death over the ensuing 6 months than their white European counterparts.[128] The higher case fatality among South Asians was largely attributable to diabetes. There may be issues regarding to how BMEG subjects present with coronary events. For example, Bangladeshi patients with AMI often present with atypical symptoms, which may lead to slower triage in the casualty department and delay in essential treatment.[129] This cultural difference in symptom presentation needs to be recognized by clinicians if mortality rates in this high-risk group are to be reduced. In addition, design of services to improve access to services must recognize dieferential presentation according to ethnicity. This was highlighted by a UK study using the Rose Angina questionnaire, which has been extensively used in different cultural settings and epidemiological studies of CHD. In this study, Fischbacher *et al*.[130] found that definite Rose angina showed lower sensitivity for other measures in South Asians than in Europeans: sensitivity for a doctor's diagnosis was 21% in South Asian and 37% in European men. Thus the performance of the Rose angina questionnaire was sufficiently inconsistent to warrant further work to achieve greater cross-cultural validity.

Current recommendations for drugs used in the primary prevention of CVD include the use of antiplatelet therapy (aspirin/clopidogrel), beta-blockers, ACE inhibitors and statins. For secondary prevention, the previous list is extended to include omega-3 fatty acids. In the setting of acute coronary syndromes and revascularization, additional drugs used acutely in the management of CHD include aspirin–clopidogrel combination therapy, glycoprotein IIb/IIIa inhibitors, thrombolytic therapy (for ST elevation MIs) and heparin (unfractionated or low-molecular weight). Statins have been discussed in detail in the sections on hyperlipidaemia and diabetes, where beta-blockers are contraindicated, limited data are available for the use of non-dihydropyridine calcium channel blockers (i.e., verapamil, diltiazem) post-MI, where left ventricular function is preserved.

In general, there are limited data in relation to differential effectiveness in the ethnicity, although there are some data in relation to different usage of these drugs, or uptake or primary/secondary prevention measures. For example, an audit of primary care angina management in Sandwell, England[131] reported smoking cessation advice in 97.1% of White Europeans, compared to 46.0% of non-white subjects. Beta-blocker use, as well as weight and exercise advice, was less common among non-white subjects. More data are available in the non-UK setting. Brown *et al*.[132] reported that African Americans and Hispanics are less likely to take aspirin than their White European counterparts, and this was not accounted for by differences in sociodemographic characteristics and CVD risk factors among ethnic minorities.

Aspirin

In high-risk patients, the Antithrombotic Trialists' Collaboration meta-analysis of randomized trials of antiplatelet therapy for prevention of death, MI

and stroke revealed that the absolute benefits of antiplatelet therapy substantially outweighed the absolute risks of major extracranial bleeding as previously discussed.[12] In primary prevention, the evidence base for aspirin use is not as good as for statins, as identified in clinical trials, such as the Physicians' Health Study.[133] Following acute MI, the early use of aspirin results in a significant reduction in early mortality (death rate ratio 0.78) in the first 35 days.[134] Between day 36 and the end of year 1, the use of aspirin during the first month (i.e., before day 36) was not associated with any significant additional difference in the death rate. Hence, all of the survival benefit of an early, 1 month course of oral aspirin seemed to accrue during the first month, with little further benefit or loss during subsequent years.

Clopidogrel

Clopidogrel, a thienopyridine derivative, is an inhibitor of platelet aggregation induced by adenosine diphosphate. In the CAPRIE trial,[135] long-term administration of clopidogrel to patients with atherosclerotic vascular disease was marginally more effective than aspirin in reducing the combined risk of ischaemic stroke, MI, or vascular death. The overall safety profile of clopidogrel was also as good as that of medium-dose aspirin. However, $>95\%$ of the participants in CAPRIE were white European. Nonetheless, current guidelines recommend clopidogrel as an alternative to aspirin, especially in high-risk subgroups with vascular disease (e.g. peripheral artery disease) or where aspirin is not tolerated.

In the setting of acute coronary syndromes and revascularization, the use of aspirin-clopidogrel combination therapy has become well established. In the CURE trial,[136] aspirin–clopidogrel combination therapy for 9 months significantly improved outcomes compared to aspirin alone, but the ethnicity breakdown was not stated. However, the COMMIT (ClOpidogrel and Metoprolol in Myocardial Infarction Trial; also Second Chinese Cardiac Study (CCS-2)) was conducted in a Chinese population, and was a randomized placebo-controlled trial of the emergency treatment of patients with suspected acute MI.[137] This trial found that in acute MI, adding clopidogrel 75 mg daily to aspirin and other standard treatments (such as fibrinolytic therapy) safely reduced mortality and major vascular events in hospital. In patients with acute coronary syndromes receiving aspirin, a strategy of clopidogrel pretreatment followed by long-term therapy for up to 12 months, was found to be beneficial in reducing major CV events, compared with placebo.[138]

Beta-blockers

In a systematic review published in 1999, the use of beta-blockers reduced the odds of death by 23% (95% CI 15–31) among approximately 25 000 patients included in 31 long-term trials of MI.[139] Limited data in this setting are available in relation to ethnicity, or in the post-thrombolytic and percutaneous coronary intervention (PCI) era. However, this class of drugs are commonly used for the treatment of angina, as well as secondary prevention post-MI.

ACE inhibitors

Both early and late administration of ACE inhibitor therapy are associated with lower mortality following MI, with the largest benefits observed with long-term mortality.[140–142] Data in stable CHD are also emerging for RAAS blockade.[109,111,112] Note however, not all ACE inhibitors may have similar benefits, as a broadly similar population in the PEACE trial[143] using trandolapril did not show a reduction in mortality from CV causes, MI, or coronary revascularization, though one might argue that the patient characteristics were different to other trials. Limited data in the setting of CHD are available in relation to ethnicity, with (e.g., 92% of participants in PEACE being of white European ethnicity).

Omega-3 fatty acids

The recent GISSI-Prevention study[144,145] of 11 324 patients showed a marked decrease in risk of sudden cardiac death as well as a reduction in all-cause mortality in the group taking a highly purified form of omega-3 fatty acids, despite the use of other secondary prevention drugs, including beta-blockers and lipid-lowering therapy. However, this was a trial conducted in an Italian population, with a high background usage of the so-called 'Mediterranean diet'. Some epidemiological data also suggest that the regular consumption of fish or dietary supplementation of fish oils rich in long chain omega-3 polyunsaturated fatty acids (*n*-3) significantly lowers the risk of CHD.[146] The data in BMEGs are limited.

Glycoprotein IIb/IIIa inhibitors

Large randomized trials have established the use of this class of drugs (abciximab, tirofiban, epitifibatide) in the setting of acute coronary syndrome patients. Of these three agents, only abciximab is licensed in the UK for use in the setting of 'high risk' patients during PCIs. In 2002, the NICE guidelines on Abciximab were published,[147] recommending its use in those patients considered 'high risk' undergoing PCI, including all patients who present with an acute coronary syndrome (unstable angina or a non-ST elevation MI); elective patients with Diabetes Mellitus; and patients undergoing complex angioplasty (namely, those involving two or more vessels; PCI with deployment of two or more intracoronary stents; PCI in saphenous vein grafts; and PCI involving bifurcation stenoses (on the junction of two vessels)). Clinical trials with these agents have

either only included a minority of BMEG subjects,[148] or ethnic breakdown was unstated.[149,150] Case series have suggested that abciximab performs safely and efficiously, for example, in a multiethnic non-white European population in Singapore.[151] In a Hawaiian cohort, Pacific Islanders and Far Eastern Asians had a substantially higher burden of comorbidities than White European Caucasians, but ethnicity did not appear to influence PCI procedural success or procedure-related complications.[152]

Thrombolytic therapy

Thrombolytic therapy is well established as systemic reperfusion therapy in the setting of acute ST elevation MI.

Limited data are available on differences in responses to thrombolytic therapy in different ethnic populations. In a substudy from the Thrombolysis and Angioplasty in MI (phase 1) study, the patency rate of the infarct-related artery at 90 min was 91% for blacks and was 72% for white Europeans ($P=0.051$). Major clinical outcomes including survival until time of hospital discharge (92% black vs 93% white European, $P=0.68$) were not significantly different, although blacks received more transfusions.[153] In the global utilization of streptokinase and tPA for occluded coronary arteries trial, black race was an adverse feature for 1-year survival after thrombolysis for AMI on multivariate analysis.[154] No data were available for the UK.

Revascularization

There is a widespread perception that coronary arteries are smaller in south Asians, and smaller coronary arteries may give rise to technical difficulties during bypass graft and intervention procedures such as percutaneous transluminal coronary angioplasty, stents and atherectomy.[155] On smaller arteries, atheroma may also give an impression of more severe disease than on larger diameter arteries. In one study, the smaller coronary arteries in South Asian patients were explained by body size alone and were not due to ethnic origin *per se.*[156]

The Whitehall II prospective cohort study found no evidence that low social position or South Asian ethnicity was associated with lower use of cardiac procedures or drugs, independently of clinical need.[157] This is echoed by an ecological study, which actually found that general practices with a higher proportion of South Asian patients had higher rates of angiography, challenging the widely held belief that access may be inequitable.[158] However, the ACRE study[159] suggested access to revascularization may be inequitable.

Implications for management

The available data in CHD and revascularization in relation to ethnicity are limited, and clinical trials have only included a small minority of BMEGs, although some large randomized controlled trials (RCTs) have recently been conducted in China. Until the availability of more data, recommendations for pharmacological therapy for primary or secondary prevention, and during acute coronary syndromes or revascularization, should be based on current published guidelines.

Recommendations in BMEG subjects with ischaemic heart disease

Recommendation	*Grade of evidence in the general population*	*Grade of evidence in BMEG*
Primary prevention Drugs used for primary prevention should be part of a holistic approach to CV risk management, where all risk factors are addressed, including smoking cessation, weight reduction, exercise, etc.	A	D
Aspirin 75 mg daily is recommended for all people over the age of 50 years who have a total CVD risk >20%, and in selected people with diabetes (>50 years, or who are younger but have had the disease for more than 10 years, or who are already receiving treatment for hypertension), once the blood pressure has been controlled to at least the audit standard of <150 mm Hg systolic and <90 mm Hg diastolic. If aspirin is contraindicated, or not tolerated, then clopidogrel 75 mg daily is appropriate.	A	D
Beta-blockers, ACE inhibitors and statins should be used, as per recommended guidelines for primary prevention.	A	D
Secondary prevention Aspirin 75 mg daily is recommended for life for all people with CHD or PAD. If aspirin is contraindicated, or not tolerated, then clopidogrel 75 mg daily is appropriate.	A	D
Beta-blockers, ACE inhibitors and statins should be used, as per recommended guidelines for secondary prevention.	A	D

Abbreviations: ACE, angiotensin-converting enzyme; CV, cardiovascular; CVD, cardiovascular disease; CHD, coronary heart disease; PAD, peripheral arterial disease.

Peripheral artery disease

The epidemiology of peripheral arterial disease (PAD) in the white European population of developed countries is relatively well defined. By comparison, much less is known about the prevalence and characteristics PAD in the BMEG population. As a result, the evidence base for the treatment of PAD is

founded almost exclusively on white European patients and it is possible that these treatments may be clinically and cost ineffective, even harmful, in patients from other ethnic backgrounds.[160,161]

Lower limb arterial disease

On the basis of numerous population-based studies, it widely accepted that 20% of white European men aged over 65 years of age living in developed countries have lower limb PAD as defined by the absence of pulses or an ankle-brachial pressure index of less than 0.9. Approximately, one quarter of affected individuals (5% of the total population) is symptomatic with the commonest presentation being intermittent claudication. It is generally taught that the disease is less common in women of similar age but owing to the increase in smoking among women and their greater longevity, the overall burden of disease in white male and female subjects is likely to be similar.[162,163]

By contrast, there have been no large population-based studies specifically describing the prevalence of PAD in BMEGs. Retrospective hospital-based series from developing countries suggest that the overall prevalence of PAD may be lower than that seen in Western series[163–168] (Supplementary Table W4). By contrast, data from developed countries, notably the USA, suggest that non-white Europeans may at risk of accelerated atherosclerosis and lower limb ischaemia.[169] The anatomical distribution of PAD may also vary with ethnic origin with Asians and blacks possibly having a higher prevalence of more distal disease,[170–172] especially with thrombo-angiitis obliterans (Buerger's disease).[173,174] These data suggest that the underlying pathophysiology of arterial disease is affected by ethnicity and race, presumably as a result of poorly understood genetic factors.[175] However, it is difficult to define to what extent these difference are due to nature or nurture as other factors such as socio-economic factors, lifestyle and variable health provisos are also likely to be important. For example, there are numerous data, mainly from the US, to suggest that the availability and outcome of treatment for lower limb PAD is inferior in non-whites.[170,172,176] ACs appear more likely to undergo infra-inguinal than aorto-iliac bypass, supporting the notion that they have a greater tendency to develop distal disease.[176,177] This may also explain why US blacks appear less likely to undergo endovascular as opposed to open surgical revascularization and are at increased risk of amputation. However, life-style, socio-economic status, levels of insurance cover and thus unmet health-care need may be equally important[176,177–187] (Supplementary Table W5). A study from South Africa also indicated that non-whites tend to present with more advanced disease, are less likely to have reconstruction and more likely to have amputation.[188] By contrast, a recent US single centre VA study showed no relationship between ethnicity and amputation level[189] and a UK study based in London actually suggested the risk of amputation was lower in male AC subjects than in the white population.[190]

AAA

Numerous population-based screening studies have shown that, in developed countries, approximately 5% of white European men in their seventh decade of age have an abdominal aortic aneurysm (AAA), that this prevalence increases to over 10% in those over the age of 80 years and that, age-for-age the disease is approximately one-third less prevalent in women.[191] By contrast, there are few data on the prevalence and distribution of aneurysmal disease in the BME population. A study from Japan found a prevalence of only 0.3% among 1591 residents of rural farming population of median age 68 years.[192] A large hospital-based study from the US found that the odds ratio for AAA surgery in black, as opposed to white European men, was 0.29 (95% CI 0.07–1.23, $P=0.09$), despite comparable levels of CV risk factors.[176] Data from South Africa also suggest that the typical 'atherosclerotic' infra-renal AAA seen in whites is uncommon in the black population.[193] With regard to Asians, a study form Bradford, UK, where south Asians comprise 14% of the population, found that none of 233 patients undergoing AAA repair between 1990 and 1997 were south Asian.[194] As with lower limb PAD and rates of lower limb revascularization, it is difficult to know to what extent these data reflect lifestyle, socio-economic factors, unmet health care need or true ethnic and racial differences in the disease processes or access to services.[171,195–197] With regard to the latter there are data to suggest that non-atherosclerotic aneurysms related to syphilis, tuberculosis and immunosuppression are commoner in the black population and that this may account for their unusual presentation and distribution.[171,196]

Cerebrovascular disease

Across Europe, the incidence of stroke and transient ischaemic attacks (TIAs) varies more than two-fold.[198–200] Even within developed countries, such as the UK, there are significant differences in disease prevalence between different regions.[201] Once again, whether this reflects differences in ethnicity, lifestyle, heath care provision or reporting is unclear. A US study based in the VA hospitals indicated that blacks have a higher incidence stroke, a lower incidence of TIAs and were less likely to undergo imaging of their carotid arteries and carotid endarterectomy. Although this might reflect differences in stroke aetiology, race was also an important independent factor.[202,203] Other groups have also found differences in surgical outcomes between different racial and ethnic groups.[203] It is difficult to determine to what extent these data may reflect the fact that intracerebral haemorrhage, subarachnoid haemorrhage, small vessel ischaemic stroke

and cardio-embolism, as opposed to athero-embolism are commoner among blacks.[204–209]

Fewer studies have specifically assessed the epidemiology of cerebrovascular disease and its treatment in other ethnic groups such as south Asians, Hispanics and indigenous peoples. UK data suggest the incidence of stroke in south Asians is higher than that seen in white Europeans (particularly within the Bangladeshi community) but lower than in ACs.[11] As with the black population, this may be partially accounted for by the higher incidence and mortality from intracerebral haemorrhage and subarachnoid haemorrhage. Ischaemic stroke secondary to carotid atherosclerosis may be less common in Hispanics and south Asians than in the white European population.[210,211] However, as with lower PAD and aneurysmal disease it is very difficult to disentangle the effects of nature (genetics), nurture and relative social disadvantage in integrating, mutually adaptive, but still multicultural, societies such as exist in North America and Western Europe).

In summary, surprisingly little is known about the influence of ethnicity on the prevalence, distribution, natural history, treatment and outcome of PAD, including AAA and cerebrovascular disease. Without such data, it is impossible to plan and implement effective health-care strategies within our increasingly multiethnic Western societies, or indeed in the 'developing' world, where vascular disease is rapidly becoming a major cause of morbidity and mortality as deaths from communicable diseases reduce and risk factors for vascular disease, especially tobacco consumption, increase. The majority of published studies analysing the epidemiology of PAD in ethnic groups are hospital-based series that are subject to bias as a result of unequal access to health care.

The link between ethnicity, vascular risk factors and arterial disease remains unclear. South Asians appear to have less atherosclerosis than white Europeans and yet suffer from a higher prevalence of CHD, whereas blacks appear to suffer from a predisposition for infra-genicular disease and intracranial atherosclerosis. AAA appears to be a predominantly white European disease, reinforcing the belief that AAAs are a result of a different aetiological process to arterial occlusive disease. The epidemiological, pathophysiological and anatomical differences observed in relation to the many manifestations of atherosclerosis and other vascular diseases between different ethnic and racial groups are, therefore, the result of a complex and poorly understood interplay of environmental, cultural, psychosocial, health economic and, probably, genetic factors.

Management differences, in relation to medical therapy and surgery

Black ethnicity can now be considered a consistent and independent risk factor for PAD with a magnitude similar to other established risk factors.[212] The excess prevalence of PAD is not explained by the excess of diabetes, hypertension or other CVD risk factors. Although there appears to be a consistently greater ratio of amputation:revascularization in the black to white European population (Supplementary Table W5), currently there are a paucity of data to explain the differences in relation to either medical therapy or surgery. Further research is indicated to determine the differences. Further data on the prevalence of PAD in other non-white populations are required in order to better understand the reason(s) why race/ethnicity is independently associated with poor outcomes in PAD.

Until further data are available on both primary and secondary prevention, and also endovascular or open interventions should be based on existing published guidelines.

Recommendations for peripheral arteria disease in BMEG

Recommendation	*Grade of evidence in the general population*	*Grade of evidence in BMEG*
PAD		
Drugs used for primary prevention should be part of a holistic approach to CV risk management where all risk factors are addressed including lipid control, smoking cessation and exercise etc.	A/B	D
Supervised exercise training improves intermittent claudication symptoms in the medium term.	A/B	D
There is an advantage in first time successful femorodistal bypass over primary amputation.	B/C	D
AAA		
Population AAA screening becomes increasingly beneficial as screening continues over the longer term. Benefits continue to increase after screening has ceased.	A/B	D
Aneurysms >5.5 cm in transverse diameter should be considered for open or endovascular repair.	A	D
Cerebrovascular disease (primary prevention)		
Drugs used for primary prevention should be part of a holistic approach to CV risk management where all risk factors are addressed including lipid control, smoking cessation, control of blood pressure and exercise etc.	A/B	D
There is a small benefit to carotid endarterectomy plus maximal medical therapy over medical therapy alone in low-risk patients under 75 with asymptomatic critical internal carotid artery stenosis	A	D

Recommendation	*Grade of evidence in the general population*	*Grade of evidence in BMEG*
Cerebrovascular disease (secondary prevention)		
There is benefit to carotid endarterectomy plus maximal medical therapy over medical therapy alone in patients with symptomatic critical internal carotid artery stenosis.	A	D
The benefit is greatest if surgery is undertaken within 2 weeks of the index event.	A	D

Abbreviations: AAA, abdominal aortic aneurysm; ACE, angiotensin-converting enzyme; CV, cardiovascular; PAD, peripheral arterial disease.

Cardiac rehabilitation and public health issues

Effectiveness of cardiac rehabilitation
The effectiveness of cardiac rehabilitation following MI has been evaluated by a large number of RCTs and several systematic reviews. The recent Cochrane systematic review concluded that exercise-based cardiac rehabilitation reduced total mortality by 27% and cardiac mortality by 31%.[213] In patients receiving comprehensive cardiac rehabilitation the reductions in total mortality were less (OR 0.87, 95%CI 0.71, 1.05), but a significant reduction in cardiac mortality of 26% was reported. In addition there were improvements in cardiac risk factors. The beneficial effect of cardiac rehabilitation is consistent across different diagnostic groups (MI and revascularization) and with different doses of exercise.[214]

Effectiveness of cardiac rehabilitation in ethnic minorities
There is a paucity of information on the ethnic background of participants in clinical trials of cardiac rehabilitation, so evidence is lacking about effectiveness, but no mechanism has been put forward suggesting that rehabilitation should differ in effectiveness in ethnic minority groups.

Uptake and adherence to cardiac rehabilitation
Despite the evidence for its effectiveness, uptake rates to cardiac rehabilitation programmes are low. Surveys from the UK show levels of participation between 14 and 43%.[215–219] There is relatively little information about attendance at cardiac rehabilitation by ethnic group. A study in the USA reported a higher drop-out rate of black women from a cardiac rehabilitation programme compared to white European women, despite a greater prevalence of risk factors in the black women.[220] Non-English speaking patients were reported to be less likely to attend cardiac rehabilitation in a Canadian survey.[221] An audit in the UK found that South Asian patients less likely to have attended cardiac rehabilitation largely owing to communication difficulties.[222] A systemic review of determinants of referral to cardiac rehabilitation in 30 333 participants in ten observational studies from the US, Australia and Canada found that speaking English was strongly associated with referral (RR 9.56, (95% CI 2.18, 41.93).[223] Of note, the studies had a very low overall rate of participants from an ethnic minority group (approximately 2%).

Diet: primary prevention
The diets of people from South Asian communities are very diverse, ranging from vegetarianism, which is common in Hindu Gujaratis, to the Bangladeshis who consume beef and lamb and fish. With the high prevalence of insulin resistance among South Asians an important element of dietary advice is to avoid obesity. However general advice to reduce total fat intake and increase carbohydrates may be less appropriate for this population, as this may exacerbate insulin resistance. Low levels of B_{12} and Vitamin D are dietary deficiencies of particular issue for the South Asian community.[42,43,224] Recommendations from the American Heart Association for dietary change for the primary prevention of CHD advocate consumption of fruits, vegetables, grains, low-fat or non-fat dairy products, fish, legumes, poultry and lean meat, with the matching of energy intake with energy needs to control weight.[225] A Cochrane review[226] provides a good evidence base for dietary advice in reducing serum cholesterol, LDL-cholesterol, BP and urinary sodium excretion. The changes were of modest size, and only measured in the short term. However, only three of the included 23 studies included an ethnically diverse population.

The Cochrane reviews have reported a modest fall in BP (SBP reduced 1.1 mm Hg) following comprehensive dietary and behavioural change programmes to reduce sodium intake and a small reduction in CV events following long-term interventions to reduce dietary fat consumption.[225–227] Only weak evidence exists for the benefits of low-glycaemic index diets owing to short-term, small trials.[228] The ability to implement these dietary change programmes into a non-trial based setting and in ethnic minority populations is not established. However, some small-scale community projects in ethnic minority or ethnically diverse populations have reported changes in self-reported diet following cook and taste sessions, and changes in BP, cholesterol and body weight in a family-based intervention.[229]

Physical activity: primary prevention
Evidence from the 1999 Health Survey for England identified that South Asian adults are

generally less active than white European British, with Bangladeshis reporting the least physical activity among South Asian groups and Indians the highest levels.[229] Only 18% of Bangladeshi men and 7% of Bangladeshi women would meet the current government recommended physical activity levels.[230] Women reported lower levels than men.

Reasons for the lower levels of physical activity in South Asian groups have been explored in the Health Education Authority (HEA) Second Health and Lifestyles Survey of black and ethnic minority groups in England (1994)[231] whereas 60% of the ethnic minority women reported ethnically specific reasons for non-participation in recreational physical activity, such as an unwillingness to attend mixed-sex facilities, only 30% of ethnic minority men gave these reasons.

A Cochrane review identified that physical activity interventions have a moderate effect on self-reported physical activity and cardiorespiratory fitness[232] but no information about the ethnicity of participants of included studies was provided. There is insufficient evidence to identify which components of interventions are most effective. More recent guidance from NICE supports brief interventions in primary care, but restricts the use of exercise referral schemes, pedometers, walking and cycling schemes to research studies at present.[233] This guidance does highlight the need to pay attention to the cultural needs of minority ethnic groups when developing services to promote physical activity and points to the lack of evidence about the effects of physical activity interventions in BMEGs.[233]

Improving uptake and adherence of cardiac rehabilitation

There is no good quality research evidence about improving uptake and adherence to cardiac rehabilitation in ethnic minority groups. Small uncontrolled observational studies involving a range of approaches including interpreters, bilingual staff, provision of minority language materials and a community nurse may have improved uptake (Supplementary Table W6).[234–236]

Evidence for improving uptake of primary preventive interventions

Only two studies have been published that have evaluated interventions to improve the primary prevention of CHD, both in South Asian communities[42,43,224] (Supplementary Table W7). Both were multifaceted community development studies, uncontrolled with measurement of processes, but little measurement of risk factor outcomes.

From evidence to recommendations

The NSF for CHD states that cardiac rehabilitation provision should be culturally and linguistically sensitive, but research evidence for how this should be done comes from community development, observational and uncontrolled studies.

Key areas for future research can be summarized as follows:

- Robust comparative studies of interventions designed to improve uptake and adherence to cardiac rehabilitation in ethnic minority groups;
- Risk prediction algorithms are required for the South Asian population for primary prevention of CHD;
- Outcomes based research required to evaluate community education programmes for CHD prevention in ethnic minority groups.

The health-care system perspective

As over 90% of health contacts occur within primary care and recent health policy is shifting health care from secondary to primary care, primary care is central to the management of CVD within the population. In general the overwhelming majority of the BMEGs are registered with a general practitioner and do not under use these services. The introduction of the new General Medical Services contract within general practice provides many opportunities and challenges for the prevention and management of CVD within primary care. A number of general issues in the management of CVD among the BMEGs need addressing including

Recommendations for improving uptake and adherence to cardiac rehabilitations in BMEG

Recommendation	*Grade of evidence*
Translation of materials used in CR patients into main languages used by ethnic minorities.	D
Racial and cultural differences should be taken into account in the promotion of diet and exercise.	D
Recording of ethnicity needs to be improved to enable monitoring to take place.	D

Language barriers. It is obvious that high-quality heath care requires effective communication between the patient and health professional; and we know that UK is a linguistically diverse with over 300 languages spoken by children in London alone! It is estimated that there are over $\frac{1}{2}$ million people from the four established communities (Indian, Pakistani, Bangladeshi and Chinese) unable to converse adequately with their health professional (Gill, personal communication). This does not include the recent migrants including refugees and asylum seekers. This raises many challenges for the provision of effective services and a number of

solutions have been suggested to overcome these including telephone and telemedicine interpreting. In addition, translation of materials for use with these minority communities needs to be undertaken.

Ethnic profiling. Ethnic profiling has been mandatory in secondary care since 1995 but it is still not so within primary care. A partial solution has been reached with only *new* registrants in general practice completing an ethnic profile form under the new Quality and Outcomes Framework. There is still a need to increase coverage for all the practice population in line with current guidelines not only to improve access and monitoring of services but also for research.

Cultural competence. Owing to the heterogeneity of BMEGs there is a need for health services to be culturally competent. This is defined as a set of values, behaviours, attitudes and practices within a system, organization, programme or among individuals which enables them to work effectively cross-culturally.[1] All staff should undertake training to deliver effective, culturally competent service to all their patients.

Access to health care

Disparities in health and health care clearly exist among the BMEGs.[1] Note that there are some general comments to note on research to date on access to healthcare, namely:

- studies to date have been mainly descriptive with few evaluative studies,
- few trials studies have included BMEGs
- involving small numbers from one centre only,
- heterogeneity of BMEGs is often overlooked and
- adjustment for social class/deprivation has not been undertaken

The cause of inequalities in access to services is due to a complex interplay of factors including health beliefs and knowledge; knowledge of and attitudes to health; provision of health resources; language barriers; racism in service delivery and quality of care. Further studies are therefore needed to address these.

It is important to remember that providing an equal service is *not* about giving people the same service, it is about them receiving a *comparable* service. For this we need a general framework, as follows[1]:

- to tailor *effective* services to patients NEEDS;
- to systematically monitor services and ensuring that it is part of performance management; and
- to have a culturally competent workforce hence ensure that training of ALL staff is undertaken
- services for BMEGs should be part of 'mainstream' health care provision
- to provide appropriate bilingual services for effective communication
- to involve the local community engagement in the development and implementation of local services and
- to systematically capture and use ethnic data in the planning of services

Primary prevention of CVD is quite rightly addressed in the main, in primary care. It is a concept rarely encountered by most physicians in the secondary care setting, except by those with a particular interest in primary prevention *per se*, lipidology, diabetes, hypertension or cardiac rehabilitation. All diabetics should be treated according to strategies identified for patients with established CVD. Primary prevention strategies should ideally cross the interface between primary and secondary care, with common guidelines, protocols and pathways easily identifiable. Any strategies must incorporate both lifestyle factors and therapeutic interventions, with regular review. The strength of community-based programmes is also potentially significant as a mechanism to improve access to appropriate services. Many such examples exist both in the UK (e.g. 'Heart disease and South Asians, Department of Health 2004') and abroad, but formal evaluation of such strategies is lacking.

Although the majority of primary prevention inevitably occurs in primary care, secondary care is an opportunity to identify first degree relatives (male subjects <55 years and female subjects <65 years) at high risk of CVD, by virtue of taking a careful family history from patients presenting with established CVD, for example, angina, stroke, MI etc. Often lacking is the infrastructure and resources to provide an equitable service to this high-risk population. Whether primary prevention here is the role of primary or secondary care is often debated, to the detriment of potential patients. Although the new General Medical Services contract encourages audit and provision of primary prevention in primary care, no such incentives exist in secondary care. Indeed, despite mandatory ethnic monitoring in secondary care, the identification of ethnicity remains poor in secondary care and limits the provision of primary prevention strategies. One hopes that this situation improves with cardiac networks developing strategies to deliver on chapters 1 and 2 of the NSF for CHD in the near future.

One possible mechanism to facilitate effective primary prevention strategies might be effective application of standardized CV risk estimation in specialty secondary care clinics such as lipidology, hypertension. In addition, cardiac rehabilitation programmes engaging family members might serve a potential route for providing primary prevention lifestyle advice to direct and extended family members, although such an approach and its efficacy is yet to be validated and costed.

Overall, the majority of primary prevention strategies remain within the remit of primary care

as stated earlier, though the development of CV risk clinics could span the interface of primary and secondary care in order to make delivery of primary prevention care more comprehensive in future.

Conclusion

This overview has highlighted that there are many gaps in knowledge on CVD and ethnicity. Many general areas for further research are needed, for example:

- how individuals from BMEGs negotiate their way *into* and *within* health care
- the need for incidence data on CVD hence a cohort study to produce a validated risk prediction model for all ethnic groups
- the evidence base by ethnic group on health status, access to services, health outcomes and cost-effectiveness of interventions is poor and needs to be addressed by all national commissioning bodies
- further evaluation of different models of providing bilingual services, such as physically present interpreters and advocates compared to telephone and telemedicine interpreting
- assessing the effect of race on health and health care, lastly
- ensure that we do not exclude the BMEG groups from research.

Competing interests

Sanofi-Aventis provided an unrestricted educational grant for honoraria and logistic support to allow meetings of the group to take place. No member of the pharmaceutical industry was present during discussions of the group. Sanofi Aventis had no input into the discussions or subsequent content of the publication arising from these discussions.

GYHL has received funding for research, educational symposia, consultancy and lecturing from different manufacturers of drugs used for the treatment of hypertension, atrial fibrillation and thrombosis. He is Clinical Adviser to the Guideline Development Group writing the United Kingdom NICE Guidelines on atrial fibrillation management (www.nice.org.uk), and is a contributor to the American College of Chest Physicians Consensus Guidelines on Antithrombotic Therapy.

KCRP has received fees and honoraria for consultation and speaking at educational meetings supported by Sanofi-Aventis, Takeda, Astra-Zeneca, Pfizer, MSD and Merck. He has received travel support for meetings from Takeda, Novartis, NICE and The Lancet. He is also Chairman of the South Asian Health Foundation and has provided input for NICE guidelines as both a member of the guideline development groups and an independent expert.

KJ is PI of a cardiac rehabilitation trial in a multiethnic population.

PSG is a Regional Patron of SAHF and member of NICE Lipid Modification Clinical Guideline Group.

EH has received honoraria for speaking, consultancy and research from MSD, Astra Zeneca, Pfizer, Merck and Sanofi Aventis.

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