High blood pressure appears commoner in black people than in whites. Studies in the United States and Britain have shown that the average blood pressure of black populations was higher than that of whites living in the same area [1-3]. Early comparative surveys in the West Indies also reported higher blood pressure in black than in white residents, although in these studies income levels and social class differed in the two races [4, 5]. Surprisingly, however, when Miall and co-workers compared the blood pressures of Jamaicans and whites in South Wales, no differences were found [6, 7]. Mortality studies in Britain and the United States have also shown that blacks have higher overall standardized mortality rates than white [8, 9].

Some important biochemical differences are also found when black and white hypertensive subjects are compared. It is tempting therefore to assume that these apparent contrasts in both quantity and quality of hypertension are due to some major genetic differences between blacks and whites. One fact, however, casts immediate doubt on this view; groups of black people in tribal areas in Africa do not have high blood pressures, and hypertension is rare [10]. It follows that other non-genetic factors including social class, environmental stress, nutrition and access to medical care must play an important part in high blood pressure of black people in urban communities.

Morbidity and mortality from hypertension

As high blood pressure is a prime risk factor for coronary heart disease and stroke, it would be expected that both heart attack and stroke would be correspondingly commoner in black people [11, 12]. Some authors have stated that not only is high blood pressure commoner in blacks, but also, for a given level of blood pressure, the prognosis for a black hypertensive patient is worse than for a comparable white [13]. However, Langford [14] has pointed out that there is little to support this view and the opposite may be the case. One of the few long-term epidemiological follow-up studies of blacks and whites, in Evans County, Georgia, reported that black males with raised blood pressure had a lower individual attributable risk of death than white males, even though the average blood pressure of the black population was higher. When the higher prevalence of hypertension was taken into account, blacks did have a higher population attributable risk of death [15, 16]. The use of arbitrary dividing lines for defining hypertension and subsequent mortality (for example the WHO criteria of 160/95 mmHg) will over-estimate individual risk when comparing black and white populations with different blood pressure distributions. Thus if white men had blood pressures as high as their black counterparts they would be expected to suffer an even higher mortality. Further evidence of differences in the risk of high blood pressure in the two ethnic groups comes from Jamaica. In a 13 year follow-up study of 1065 black West Indians, no correlation was found between blood pressure and total mortality until pressures of more than 180/110 mmHg were reached [17]; thus the blood pressure and mortality relationship is quite different from that seen in the long-term epidemiological studies in whites.

There are also some major differences in the nature of the vascular complications of hypertension suffered by the two ethnic groups. Reports of mortality, morbidity and hospital admission rates of blacks in Britain, the United States and the West Indies, as well as Africa, have all shown that heart attack is significantly less common.

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than in whites or Asians [9, 18-21]. In Birmingham admission for heart attack occurred with half the expected frequency in blacks whereas admission for stroke was much commoner [22]. In Trinidad and in Britain, Asians have been found to have more heart disease than blacks or whites [18, 23]. These sharp contrasts in the type of cardiovascular complications of hypertension, with blacks being relatively immune to heart attack, are hard to explain. One possibility is that black people with their high frequency of premature stroke do not survive long enough to develop myocardial infarction. This simple explanation is unlikely, because even below the age of 50, heart attack is a much commoner cause of death than stroke in all ethnic groups [19]. Another possibility is that the higher blood pressures in blacks are offset by lower levels of other coronary risk factors. Blacks in Britain, the U.S.A. and South Africa have been found to have lower levels of plasma triglycerides and cholesterol and higher high-density-lipoprotein cholesterol levels than whites or Asians [3, 24, 25]. Some authors have suggested that coronary risk in black populations may be better estimated by HDL status alone, rather than from a combination of the other major risk factors [26].

Cigarette smoking in the West Indians in Britain is almost as common as in whites, although average consumption in smokers is lower. However, these differences are probably not great enough to explain the major ethnic differences in coronary heart disease. In Britain where there is a large Asian community an even more complex picture emerges. Asians have lower blood pressures than whites or blacks, they smoke less commonly but they appear to have more coronary heart disease [18, 27]. Whereas the frequency of stroke in the three ethnic groups is more nearly related to their blood pressures, there may be some differences in pathological type of stroke. In whites, strokes are predominantly thrombotic [28], whereas in Jamaican black populations strokes are more often haemorrhagic [29] and are thereby more closely related to the height of the blood pressure. Little is known about the pathology of strokes in the three ethnic groups in Britain. Blacks are not, however, totally protected from the development of atheroma. The International Atheroma Project was a detailed autopsy study of 19 ethnic groups in 14 countries, where amounts and types of arteriosclerosis were measured by standardized techniques. This project did demonstrate that fatty streaks and raised lesions in arterial vessels were significantly less common in black subjects than in age-matched whites. However, blacks in New Orleans, Jamaica and Puerto Rico had more extensive arterial damage than several South American white groups as well as South African blacks, although less than United States whites [30]. Hypertension and diabetes were shown to exaggerate these arterial lesions independently but with a similar geographic and ethnic pattern as was seen in subjects without these conditions [31].

As the standard cardiovascular risk factors, hypertension, cigarette smoking and hypercholesterolaemia, do not explain the ethnic differences in atheromatous disease [32], several groups have investigated other biochemical and haematological indices. Meade and co-workers studying a working population in north-west London confirmed that blood pressures were higher in blacks, and blood cholesterol and triglyceride levels were higher in whites [3]. However, in addition, factor VII, haemoglobin and platelet counts were higher in whites. In Evans County, white people had lower fibrinolytic activity than blacks [33]. It may be that these other variables which are related to the development of arterial disease may help to explain the relative protection from atheroma in blacks, where the main risk factors have failed.

Whether the black/white differences in risk factors and complications of hypertension are primarily of genetic or environmental origin, or a combination of both, is not known but differences in blood levels of haemostatic proteins imply a major genetic contribution.

Environmental stress

The concept of environmental stress as an important risk factor for both hypertension and coronary heart disease is attractive. Unfortunately, attempts to measure stress or personality type are fraught with difficulties. Type A (stressed) personalities have been reported to be more prone to heart disease [34, 35] although this has not been universally confirmed [36]. A recent well conducted study from Scotland failed to show any relationship between blood pressure and personality [37]. Although hypertensive patients may exhibit signs of stress once they have been diagnosed this cannot be causally related to the elevation of blood pressure. A recent review of six studies, in which psychological testing was conducted before the diagnosis of hypertension, reported two demonstrating a positive relationship between blood pressure and personality, two with no link and two with the opposite results [38]. All these studies were well conducted and this type of
approach seems unlikely to be fruitful in the future.

It remains possible that socio-economic factors, including social class, urban stress and deprivation, are related to high blood pressures. Epidemiological studies in the United States have rarely employed any assessment of social class, similar to that of the Registrar General in Britain; and this may explain the apparent excess of hypertension which is usually found in blacks. However, in Charleston, South Carolina, correction for social class almost abolished the racial differences in blood pressure and attenuated the reported correlation between blood pressure and depth of skin pigmentation in blacks [40, 41]. One study of four urban areas in Detroit, U.S.A. [39] showed that black people living in areas with high crime rates, residential instability and high population density had higher blood pressures than comparable black groups living in low stress areas. These differences in blood pressure between ‘stressor areas’ were as great as the difference between blacks and whites; low stress blacks had slightly lower blood pressures than high stress whites. Educational level has also been found to be inversely associated with hypertension in black and in white Americans [42]. Further support for the stress hypothesis comes from Africa, where tribesmen living in rural areas have lower blood pressures than genetically identical groups living in towns [43–45]. Recruits to the Kenyan army had low blood pressures on enlistment but these rose sharply over the subsequent years [46]. In general populations which migrate take some of their health characteristics with them but also take on some features of the host population [47]. Japanese migrants in California have more coronary heart disease than those in Hawaii, who in turn have more than those who remained in Japan [48]. The marked changes which occur in migrants might be explained by increased stress of migration, but there are many other possible explanations, particularly changes in nutrition. The two main migrant communities in Britain, as already stated, differ enormously and it is difficult to be sure to what extent the difference are due to variation in diet, or to different responses to urban stress. Both groups tend to live in poor areas of inner cities, and both are employed in relatively less remunerative and unattractive occupations. It is of interest that Miall et al. [7] did not find higher blood pressures in the semi-rural area of Lawrence Tavern, Jamaica, in comparison with those of whites in South Wales [6], when blood pressures were measured by the same observer using identical techniques and equipment.

Alcohol

High alcohol consumption has recently been recognized as an important factor in high blood pressure [49, 50]. In the Kaiser Permanente study of America [51] the close relationship between alcohol intake and blood pressure was confirmed for all ethnic groups separately, and blacks with comparable drinking habits to whites still had higher blood pressures. In Britain, Asians consume less alcohol than whites or blacks and this might in part explain their slightly lower blood pressures, (L. T. Bannan, S. H. D. Jackson & D. G. Beevers, unpublished work).

Nutrition

Differences in weight or body mass index are not likely to explain ethnic differences in blood pressure. Black men are generally no more obese than whites, although black women frequently are [2]. Salt may be a more important dietary factor in the aetiology of hypertension. The concept that excessive salt intake is a major cause of hypertension dates from ancient times [52], and it has long been known that severe salt restriction causes a fall in blood pressure [53]. Tribal societies which consume virtually no salt have lower blood pressures and less increase in blood pressure with age than advanced societies with high salt intakes [54]. Many of these primitive societies are in Africa. There are, however many other variables, including malnutrition and increased frequencies of infective and parasitic disease [55, 56] which may explain these trends, and this, of course, also includes lack of urban stress. The hypertension salt hypothesis depends largely on comparisons of primitive and urban societies; usually no differences in salt intake or excretion are found when comparing hypertensives or normotensive subjects in a single population group [57]. Estimates of salt intake of blacks and whites in urban Britain or America are identical [58], so it is unlikely that dietary salt excess alone is the cause of high blood pressure in blacks.

Sodium and potassium transport

Although dietary intake of salt cannot alone explain ethnic differences in blood pressure, there remains much evidence that black people tend to metabolize salt differently from whites. In one study normotensive blacks were found to have a reduced capacity to excrete a sodium load when salt intake was increased tenfold, and this was associated with a small rise in blood pressure
A very marked difference is seen in the cellular transport of sodium and potassium in blacks. The erythrocytes of both hypertensive and normotensive blacks have a significantly reduced ouabain-insensitive sodium/potassium pump activity when compared with normotensive whites, and this may be interpreted as a reduced cellular capacity to eliminate sodium [60]. This cellular abnormality may be the key to hypertension in blacks, as abnormalities of erythrocyte sodium transport do appear to have a genetic basis [61].

If black people do have a genetic difference in cellular handling of sodium ions, then it seems likely that hypertension occurs only in the presence of a relatively high salt Western style diet. Evidence that genetic differences in sodium handling may produce hypertension comes from animal models, as seen in salt-sensitive rats [62]. This species develops hypertension only when given salt in the diet, although the amount of salt needed to do this is enormous. By contrast salt-resistant rats do not develop hypertension when receiving similar dietary sodium loads.

Although no differences are found in urinary sodium when comparing blacks and whites, several studies have reported differences in urinary sodium/potassium ratio [54]. These are due to higher urinary potassium excretions in normotensive subjects. It is possible that high blood pressure is related less to dietary sodium excess but rather to potassium deficiency. Most studies of electrolytes and blood pressure have failed to examine potassium, but one study from Scotland found no difference in potassium excretion in hypertensive and normotensive subjects, although average 24 h urinary potassium levels were low in both groups in an area with a high prevalence of hypertension. However, dietary potassium loading has been found by some workers to lead to a fall in blood pressure [63]. Further work on potassium and blood pressure is needed, as this may provide a dietary link which has been missing until now. Langford has commented that high potassium foods, including fruit and vegetables, are relatively more expensive than other sources of calories and thus would be less readily available to poorer black populations [14].

Low-renin hypertension

One major and consistent biochemical difference between blacks and whites has been found in practically every published report. Blacks have lower plasma renin levels than whites and so-called ‘low-renin hypertension’ is commoner [64]. As renin is suppressed in response to salt loading it is tempting to regard the lower renin levels in blacks as being due to higher dietary salt. This is clearly untenable as blacks do not eat or excrete more salt [59], and furthermore when renin secretion is expressed in relation to concurrent dietary or urinary sodium, the differences between blacks and whites remain. The reason for the lower renin levels in blacks is therefore unknown. Two other possible mechanisms for renin suppression have been advanced but neither appears to fit the bill. Renin suppression may occur as a consequence of increasing nephrosclerosis [65, 66], and, in hypertensive patients, lower renin levels are found with advancing age [67]. The low-renin hypertension/nephrosclerosis hypothesis does not account for the lower levels of renin in relatively mild hypertensive young blacks when compared with older severer hypertensive whites. Renin release is also under sympathetic nervous control, so it seems possible that renin suppression may occur as a consequence of failure of sympathetic stimulation. The relationship between plasma levels of catecholamines and sympathetic activity is unsure, but despite the marked renin suppression in blacks, no consistent differences are found in plasma levels of noradrenaline [58] although in the Evans County and Bogulasa studies blacks were found to have lower levels of dopamine β-hydroxylase [68, 69]. Noradrenaline rises with advancing age whilst renin falls. The relationship between plasma renin and noradrenaline levels and hypertension has only rarely been examined in epidemiological studies, as the assays are technically difficult and cannot be applied to large populations. However, one study of 115 factory workers demonstrated no ethnic differences in plasma noradrenaline, but did confirm that blood pressures were higher, and renin levels lower, in blacks than in whites. No correlation was found between the height of the blood pressure and either plasma renin or noradrenaline levels [58].

Response to treatment

Good control of blood pressure leads to prevention of death from heart attack and stroke in both blacks and whites [70]. In this respect blacks do not differ from whites. As antihypertensive therapy is still provided on only a very sporadic basis, the higher mortality of blacks may be due in part to less reliable medical care and reduced compliance with drug therapy. When blacks receive better medical care, comparable with that of their white neighbours, their blood pressures
and their mortality fall. There is evidence that blacks respond less well to β-receptor-blocking drugs [71] and respond better to thiazide diuretics [72], although good blood pressure control is achieved in both blacks and whites when these drugs are used in combination [73].

At the moment therefore it seems that there are genuine differences between the races; blood pressures are lower in blacks when they live in tribal or rural societies, but in response to urbanization into similar environmental influences as whites, their blood pressures rise to as high or higher levels. This effect could be due to a genetically increased sensitivity to environmental factors. It remains likely, however, that much of the black/white differences in blood pressure may be accounted for by differences in social class.

References
