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Ethnic group comparisons of variables associated with ischaemic heart disease

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SUMMARY Variables known or postulated as being associated with the onset of ischaemic heart disease have been measured in 553 male and female members, aged 18 to 49, of a working population in north-west London; 412 were white, and 141 were black. Cigarette and alcohol consumption were higher in white than in black men, and so were cholesterol, triglyceride, factor VII, carbon monoxide, and haemoglobin levels, and platelet and white cell counts; factor VIII and blood pressure levels, and fibrinolytic activity were lower. More white than black women smoked; the latter were more obese. Differences between black and white women in factor VIII, carbon monoxide, haemoglobin, and blood pressure levels, and in white cell count were similar to those seen in men; however, there were no significant differences in levels of cholesterol, triglycerides or factor VII, or in platelet count, or fibrinolytic activity. Though fibrinolytic activity was higher in the black men than in the white, the difference was not as distinct as in some other studies; the possibility that black men may be losing some of their natural protection against ischaemic heart disease should be borne in mind. On the basis of the ethnic group comparisons it is concluded that, in addition to variables already known to be associated with the onset of ischaemic heart disease, attention should be given to the possible effects of high factor VII levels, poor fibrinolytic activity, and high platelet counts; other considerations suggest that further haemostatic variables, such as factor VIII and fibrinogen, should also receive attention.

Contrasts between ethnic groups in their experience of clinically manifest ischaemic heart disease, and in variables associated with its incidence, may be valuable in studying the aetiology and pathogenesis of the disease. Blacks in Africa, especially those in rural surroundings, rarely develop ischaemic heart disease, and compared with whites in Africa they have lower levels of blood cholesterol (Bronte-Stewart *et al.*, 1955; Shaper, 1970). In the U.S.A., Cassel (1971) found an appreciably lower prevalence and incidence of ischaemic heart disease in blacks than in whites in Evans County, Georgia; however, only a small part of the differences could be explained in terms of contrasts in diet, smoking habits, blood pressure, blood cholesterol, obesity, or physical activity levels (the last of these being the single factor which is likely to have had the largest effect). Boyle (1974) summarises several other studies in the U.S.A. showing higher mortality from ischaemic heart disease in whites than blacks. The United States National Center for Health Statistics (1964a, b), on the other hand, found a similar prevalence of ischaemic heart disease in blacks and whites. In this country, Pedoe *et al.* (1975) found that men of Caribbean origin living in East London experienced an incidence of ischaemic heart disease only 8 per cent of that in all men in the same area.

Cassel (1971) concluded that there are mechanisms other than those so far studied epidemiologically that either protect blacks against clinical ischaemic heart disease, or increase the risk in whites, or both. This conclusion is supported by other considerations; the epidemiology of mural atheroma, for example, is not the same as that of lumen occlusion or of ischaemic heart disease itself (Morris, 1951; Morris and Crawford, 1958; Acheson, 1962). Thus, while atheroma is undoubtedly a necessary cause of ischaemic heart disease (in all but rather exceptional circumstances), it is not by any means always a sufficient one (Morris and Crawford, 1961), and it is necessary to consider mechanisms other than the formation of

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atheroma in order to explain much of the occurrence of clinically manifest ischaemic heart disease. Clearly, thrombogenesis may be one such mechanism, though for various reasons it has not so far received much attention from the epidemiological point of view. If there is an 'increased thrombotic tendency' which is partly determined by components of the haemostatic system, then by comparison with blacks, whites might be expected to have more adhesive or aggregable platelets, higher clotting factor levels, and poorer fibrinolytic activity. Several ethnic group comparisons of fibrinolytic activity have indicated higher levels in blacks than in whites (Gillman *et al.*, 1957; Franz *et al.*, 1961; Menon, 1967; Barr *et al.*, 1973). Walker (1961) suggested that urbanisation might be accompanied by a significant reduction in fibrinolytic activity in blacks. Shaper *et al.* (1966) compared Africans and Asians in Uganda, and found fibrinolytic activity to be significantly greater in the former. In addition to higher fibrinolytic activity, Merskey *et al.* (1960) found lower factor VII levels in Bantu migrant labourers, as compared with white South Africans, but the blacks had higher factor VIII levels, and generated more thromboplastin.

There are, however, virtually no systematic data comparing haemostatic findings in blacks and whites in the United Kingdom, or, indeed, findings on variables such as cholesterol, blood pressure and smoking levels, for which an association with ischaemic heart disease is well established. This paper presents such data on blacks and whites in north-west London; women, as well as men, have been studied.

Methods

The data in this report come from a subgroup in a prospective study of ischaemic heart disease in just over 3400 participants. Further details, including the composition of the total study population, are given elsewhere (Meade and North, 1977). The findings presented here deal with those participants who work at the production site of a food-processing company in north-west London, this being the occupational group in the study with an appreciable proportion of black employees. There are very few blacks over the age of 50 working at the site concerned, and the comparisons are therefore confined to those (white and black) aged 18 to 49. As the use of oral contraceptives leads to substantial changes in many of the variables discussed in this paper (Meade *et al.*, 1976a) the findings in the women (34 white; 4 black) using these preparations have been omitted; the numbers of women given in the text and the results shown in the Tables therefore refer only to women not on oral contraceptives at the time of recruitment into the study. The 412 whites (351 men; 61 women) included are all of British nationality, all but a few having also been born in the United Kingdom. The 141 blacks (86 men; 55 women) are nearly all of British nationality; all were born outside the United Kingdom, 132 coming from the West Indies, and 9 from Africa.

On day shifts, men (283 white, 48 black) and women (61 white, 55 black) are seen on two consecutive days between 09·00 and 11·00, that is at the start of their working day. Blood is taken without venecompression except when unobtainable other wise. Most of the tests are carried out on non-fasting blood samples taken on the first day, and include an estimation of serum triglycerides. On the second day, however, blood samples are taken after an overnight fast (broken only by 50 g glucose taken by mouth 1½ to 2 hours before the blood is taken); these samples are used for blood sugar and serum triglyceride estimations. Men on the night shift (68 white, 38 black) are seen on one day only, between 07·00 and 08·30, that is at the end of their working day, and in a non-fasting state. (There are no women on the night shift.) Clotting factor assays measure biological activity. Further details of procedures and methods are given in Meade *et al.* (1976a,b) and Meade and North (1977).

As already reported (Meade and North, 1977) many of the variables in this study alter with age. However, within each of the three main groups of the population described here (day men, night men, and women), the mean ages of the whites and blacks are very similar (Table 1), and unadjusted mean values for the variables in question are, therefore, shown where the three groups are considered separately (Table 2). In the case of the men, however, there are some variables (e.g. cholesterol) for which a significant difference is only apparent in one of the two shifts, or for which (e.g. fibrinolytic activity) the difference is not significant at the 5 per cent level within either shift, though in the same direction. In these cases, the data for the two shifts have been pooled, and analysed by the technique of stepwise multiple regression in order to test for an ethnic group effect that is independent of effects caused by shift or age (since night men are, on average, older than the day men and contain a higher proportion of blacks).

Results

Table 1 compares the whites and blacks in terms of several personal and social characteristics. There are appreciably fewer blacks in social classes I and II and a higher proportion in classes IV and V. As

Table 1 *Characteristics of whites (W) and blacks (B)*

	Day men W (283)	B (48)	*Night men* W (68)	B (38)	*Women** W (61)	B (55)
Mean age (y)	34·7	35·5	40·5	40·0	36·9	38·6
Social class						
% I and II	15·2	0	7·4	0	16·3	3·8
% III	60·0	47·9	61·7	42·1	63·6	45·3
% IV and V	24·8	52·1	30·9	57·9	20·1	50·9
No. with past history of 'definite' ischaemic heart disease†	6	0	1	0	0	0
Smoking						
Per cent current cigarette smokers	47·9	47·9	70·6	44·7	59·0	7·3
Mean cigarettes/day in current smokers	18·0	11·9	21·3	9·6	15·5	12·3
Alcohol intake						
Mean (g/w)	126	93	94	58	27	22
Obesity						
Weight/height[a]	24·4	24·5	25·2	25·8	23·6	27·4
Analgesics						
Per cent having taken any in last week	17·9	20·0	23·5	18·4	30·0	19·2
Blood group (sexes and shifts combined)		*White*		*Black*		
% A		46·2		22·4		
% B		10·3		25·6		
% O		41·6		49·6		
% AB		1·9		2·4		

*Those on oral contraceptives omitted.
†'Definite' myocardial infarction (WHO criteria) and/or Q wave on electrocardiogram, and/or 'definite' angina pectoris.

Table 2 *(a) Biochemical, (b) coagulation, (c) haematological, and (d) blood pressure findings in whites (W) and blacks (B); means (and standard deviations)*

	Day men W (283)		B (48)	*Night men* W (68)		B (38)	*Women†* W (61)		B (55)
(a) Cholesterol (mmol/l)	5·4 (1·2)	*	5·1 (1·0)	5·6 (1·0)		5·2 (0·9)	5·5 (1·0)		5·3 (1·2)
Triglycerides (g/l)									
Fasting	1·1 (0·9)	*	0·7 (0·3)	—		—	0·8 (0·4)		0·9 (0·4)
Non-fasting	1·4 (0·8)	**	0·9 (0·4)	1·9 (1·4)	**	1·2 (0·6)	1·0 (0·5)		1·0 (0·5)
Blood sugar, fasting (mmol/l)	3·4 (0·9)		3·7 (0·8)	—		—	3·3 (0·9)		3·6 (0·6)
Uric acid (μmol/l)	353·2 (67·9)		355·2 (65·7)	346·2 (70·4)		324·0 (68·0)	257·7 (48·6)		267·1 (51·6)
Carbon monoxide (%)	2·7 (3·8)	*	1·5 (1·7)	3·3 (2·6)	**	1·9 (2·2)	2·7 (2·5)	**	0·8 (1·0)
(b) Factor V	116·2 (30·1)		110·6 (23·4)	115·6 (31·9)		104·7 (26·5)	117·0 (20·8)		114·6 (22·5)
Factor VII } % of standard	93·3 (26·2)	*	84·7 (25·5)	111·2 (29·2)	**	95·5 (25·1)	92·7 (25·5)		94·2 (28·3)
Factor VIII }	90·4 (32·8)	**	105·7 (48·4)	85·1 (31·2)	**	104·2 (41·4)	89·5 (31·3)	**	121·6 (61·3)
Fibrinogen (mg/100 ml)	251·8 (56·6)		251·2 (53·1)	272·9 (61·8)		271·6 (58·2)	266·7 (63·7)		282·5 (48·3)
Platelets (10^3/μl)	222·9 (65·2)	*	187·0 (53·0)	211·5 (49·7)		195·6 (81·3)	216·6 (62·2)		202·1 (63·7)
Platelet adhesiveness (%)	40·7 (16·5)		41·7 (18·4)	39·5 (17·5)		41·9 (11·1)	38·4 (15·5)		37·2 (18·0)
Fibrinolytic activity, 100/lysis time (h)	30·8 (14·7)		33·5 (16·3)	24·1 (13·4)		27·2 (13·1)	28·6 (15·0)		27·0 (10·4)
(c) White cell count (10^3/μl)									
All	7·2 (2·1)	**	6·0 (1·6)	7·8 (1·7)	**	6·4 (1·9)	6·7 (1·6)	**	5·8 (1·4)
Non-smokers	6·2	*	5·4	7·1	**	5·8	6·2		5·8
Haemoglobin g/dl	15·1 (0·9)	**	14·7 (1·0)	14·9 (1·0)		14·9 (0·8)	13·4 (1·2)	*	12·8 (1·2)
(d) Blood pressure (mmHg)									
Systolic	125·6 (16·8)	**	133·4 (17·6)	134·9 (18·4)		139·3 (18·9)	119·1 (15·5)	*	125·9 (23·9)
Diastolic	75·5 (12·2)	**	81·5 (13·8)	82·2 (12·7)		87·3 (15·6)	70·2 (10·3)	*	74·8 (14·5)
Pulse rate/minute	76·1 (9·5)		76·4 (10·1)	76·2 (10·1)		75·0 (9·9)	76·1 (9·7)		77·4 (8·9)

*$P < 0.05$, between W and B.
**$P < 0.01$, between W and B.
†Those on oral contraceptives omitted.

expected, such events of ischaemic heart disease as have occurred in men of the age range under consideration are confined to the whites. In day men, the proportions of current smokers are much the same, but among the smokers, daily cigarette consumption is greater in the whites than blacks. Among night men, a substantially higher proportion of whites smoke compared with blacks, and they do so at a much higher level of consumption. It is of particular note that in this population, proportionately more white women (who are all day workers) smoke cigarettes than white day men. Very few black women smoke. Alcohol consumption is higher in white than in black men; it is similar in the two groups of women, though considerably lower than in the men. Black women are, on average, more obese than white women. The proportion of whites of blood group A is twice that of blacks, this

difference being balanced mainly by the much higher proportion of blacks of group B.

Table 2 compares the biochemical, coagulation, haematological, and blood pressure findings. In the two shifts of men, levels of non-fasting triglycerides, factor VII, and carbon monoxide, and white cell counts are consistently and significantly higher, and factor VIII levels lower, in the whites than in the blacks. There is also evidence that cholesterol and haemoglobin levels and platelet counts are higher, and blood pressure levels lower, in white than in black men, the differences being in the same direction in both shifts, though significant in only one. There is a suggestion that fibrinolytic activity may be lower in whites than in blacks, and that factor V levels may be higher, but there are no obvious differences in fibrinogen or blood sugar levels, or platelet adhesiveness.

Table 3 *Multiple regression analysis on pooled data from 331 day and 106 night men; significance levels for independent age, shift (day or night), and ethnic group effects on variables specified**

Dependent variables	*Independent variables*		
	Age	*Shift*	*Ethnic group*
Cholesterol	<0·001	<0·05	<0·05
Factor V	<0·001	<0·05	NS
Fibrinogen	<0·001	NS	NS
Platelet count	NS	NS	<0·001
Platelet adhesion	NS	NS	NS
Fibrinolytic activity	<0·001	<0·001	<0·05
Blood pressure			
Systolic	<0·001	<0·01	<0·01
Diastolic	<0·001	<0·01	<0·001

*For details of variables included in the table, see text. The directions of the shift and ethnic group effects are as in Table 2. The age effects are of an increase with rising age on cholesterol, factor V, fibrinogen, and blood pressure, and of a decrease in fibrinolytic activity.

Table 3 summarises the results of the multiple regression analysis carried out where the results in Table 2, while suggestive of a difference between the two ethnic groups, do not reach a conventional level of statistical significance in both the day and night men. This analysis confirms ethnic group effects, in the directions indicated in Table 2, on cholesterol and blood pressure levels, in platelet count, and in fibrinolytic activity, though in this last case the difference is barely significant. The multiple regression analysis does not support an ethnic group effect on factor V levels and it also confirmed, as already indicated, that there is no ethnic group effect on fibrinogen or on platelet adhesiveness. The variables on which Table 2 indicates statistically significant and consistent differences between white and black men in both shifts—that is non-fasting triglycerides, factors VII and VIII, carbon monoxide and white cell count—have not been included in the results shown in Table 3, though they have also been analysed by multiple regression. In addition to the ethnic group effects on factors VII and VIII and on triglycerides, shown in Table 2, there are significant increases with age; in the case of factor VII, there is also a significant shift effect, night men having higher values than day men. The apparent shift effect on triglycerides seen in Table 2 almost certainly results from the differences between shifts in food intake at the time of blood sampling. Haemoglobin has not been included in Table 3; there are particularly complex interactions between ethnic group and shift effects, making interpretation difficult.

Table 2 shows that among the women, there are ethnic group differences in factor VIII, carbon monoxide, haemoglobin, and blood pressure levels, and in white cell count (smokers and non-smokers combined) similar to those seen in men; there are, however, no significant differences in cholesterol or factor VII levels, platelet count, or fibrinolytic activity (blood sugar), or, despite the relative obesity of the black women, in triglycerides.

Discussion

These results should be interpreted bearing in mind the several opportunities for self-selection that have occurred, i.e. into the particular factory concerned, into the study itself and, in the case of the blacks, into this country. Nevertheless, the response rate in this study makes it likely that our population is fairly representative of the factory as a whole, and to the extent that the factory is a major employer in the area, our results are likely to be a useful index of the probable ethnic group differences in the part of London in question.

Further multiple regression analysis of our data has shown that the differences in social class, smoking, alcohol consumption, and obesity account for only part of the differences between whites and blacks shown in Tables 2 and 3, and that, with the possible exception of fibrinolytic activity in the men, the ethnic group effects persist when these other variables have been taken into account. Differences in the types of work done in the factory by the whites and blacks are reflected in the social class differences shown in Table 1. However, there are obviously likely to be other contrasts which we cannot allow for. We have only limited data on dietary intake, which as far as it goes nevertheless suggests substantially lower levels of total calorie, carbohydrate, fat, and protein intake in the blacks than in the whites. (Data on family history are available, and in the whites the relationship between

family history and the variables in this report will be described in due course; in the blacks, the virtual absence of ischaemic heart disease in the study participants and in members of their families more or less precludes similar analyses.)

The findings suggest that in addition to the role in ischaemic heart disease of blood pressure, blood lipid levels, and smoking habits, attention should also be given to other, less familiar, variables. If high clotting factor levels favour thrombogenesis, this study indicates a possible role for factor VII; this particular hypothesis is further supported by the consistent finding in many studies of substantially raised factor VII levels in women on oral contraceptives (e.g. Meade *et al.*, 1976a), and by the association between the latter and increased risks of thromboembolic episodes, including ischaemic heart disease (Mann *et al.*, 1976). Our finding of higher platelet counts in whites than in blacks complements the data of Shaper *et al.* (1966) who showed lower platelet counts in Africans than in Asians. The possibility that platelet counts might be used as a simple epidemiological index of platelet function should be considered further in view of the dearth of practicable methods in this notoriously difficult field. The carbon monoxide results reflect the differences in smoking. It is doubtful whether high leucocyte counts contribute directly to the aetiology of ischaemic heart disease, but they have been reported in smokers and in subjects who have subsequently had myocardial infarcts (Friedman *et al.*, 1974); higher levels are seen in the whites than in the blacks in our study, even in the non-smokers. Another finding in our data, reported in fuller detail elsewhere (Slack *et al.*, 1977), is that 34·7 per cent of total fasting lipoproteins are carried as α-lipoproteins in the black day men in our study, compared with 28·5 per cent in the white day men ($P < 0{\cdot}001$).

Our blood pressure data are similar to those from studies in the U.S.A. (Stamler, 1962; United States National Center for Health Statistics, 1964a, b; Cassel, 1971) in showing higher levels in blacks than in whites. While, in spite of their higher blood pressure levels, blacks appear to be less prone to ischaemic heart disease than whites, there is little doubt that they experience a considerable excess over whites of hypertensive and cerebrovascular disease (Boyle, 1974). The similarity of pulse rates in blacks and whites makes it unlikely that the differences in blood pressure are the result of differences in levels of anxiety and nervousness, as has sometimes been suggested.

By comparison with the blacks, the white men are not only possibly more prone to atheroma (e.g. higher lipid levels) and perhaps to 'hypercoagulability' (e.g. higher factor VII levels), they are also fibrinolytically less active; they are also, of course, more prone to clinical events of ischaemic heart disease. However, the difference in fibrinolytic activity between the blacks and whites in our study seems to be less obvious than in other studies, based on smaller numbers in those living in Africa. One possible explanation for the small difference in fibrinolytic activity that we have found, and which should at least be borne in mind, is that while blacks in this country still have low ischaemic heart disease rates, they may gradually be losing some of their natural protection against the disease. In a similar context, the differences in cholesterol levels, while significant in the day men (Table 2) and in all men combined (Table 3), are considerably less than the differences found in comparisons of blacks and whites in Africa, for example.

Our study is one of only a small number on ischaemic heart disease that has included women. There are clearly fewer differences between white and black women than between white and black men, the main respects in which the two groups of women seem to resemble one another, in contrast to the men, being cholesterol, triglyceride, and factor VII levels, and fibrinolytic activity. The absence of significant differences in factor VII levels and fibrinolytic activity between black and white women, both with a low incidence of ischaemic heart disease, makes an interesting contrast with the differences in the same variables between black and white men. (In the case of fibrinolytic activity, however, it should be noted that Walker (1961) found significantly lower fibrinolytic activity in rural and urban Bantu women than in the white women studied; our finding is in the same direction, though the difference is not at all obvious.) Our data also suggest potentially interesting and valuable comparisons between white men and women; of particular note in this group of workers is that proportionately more white women smoke cigarettes than white day men. [The lower proportion of women smokers in a previous report (Meade *et al.*, 1976a) was derived from a group which included women at the administrative headquarters of the company concerned, and which was of a different social class composition.] It is also clear that there are several substantial differences between day and night men. These comparisons will be described in greater detail in due course.

The observation which most obviously runs counter to the hypothesis on thrombogenesis discussed earlier is the higher factor VIII levels in blacks than in whites; this result is not the result of differences in the distributions of blood groups since our data show that it occurs within each ABO

group in both ethnic groups (Meade and North, 1977). This finding does not, however, necessarily mean that high factor VIII levels play no part in the pathogenesis of ischaemic heart disease: Egeberg (1962) has reported clinical observations which indicate that they may, and Nilsson (1974) and Poller (1973) have discussed the possibility. The same kind of consideration applies to fibrinogen, the white and black men in our study having almost identical levels. There is accumulating evidence (e.g. Dormandy *et al.*, 1973a, b) that high fibrinogen levels are associated with the clinical manifestations and prognosis of intermittent claudication, for example. The main value in ischaemic heart disease research of comparisons *between* different ethnic groups is to suggest hypotheses which, together with hypotheses from other sources (whether or not they accord with those derived from ethnic group comparisons), should then be tested *within* ischaemic heart disease endemic populations. From the comparisons presented in this paper, we conclude that, in addition to variables already known to be associated with the onset of ischaemic heart disease, attention should be given to the possible effects of high factor VII levels, poor fibrinolytic activity, and high platelet counts; considerations other than conclusions from ethnic group comparisons suggest that further haemostatic variables (e.g. factor VIII and fibrinogen levels) should also receive attention.

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