BLOOD PRESSURE AND LIFESTYLE IN UK AFRICAN-CARIBBEANS

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12 ***Health of the Nation* 1992** Page 29

"A reduction in mean blood pressure in the population of 5 mmHg could be expected from success in achieving the obesity and alcohol target, together with the reduction in sodium intake. It has been estimated that such a reduction would result in a 10% reduction in mortality from coronary heart disease and stroke".

13 **Balarajan and Raleigh 1993** Page 30

"Information about lifestyle and diet (among Caribbeans), is scanty, and the findings are not always consistent".

14 **Balarajan and Raleigh 1993** Page 30

"The sub-groups of black people, (including African-Caribbeans), are not homogeneous and show considerable diversity in terms of culture, lifestyle, social class and economic status".

15 **WHO 1993** Page 38

"The dividing line between 'normotension' and 'hypertension' is arbitrary. The current definition is that this line is the level of blood pressure above which intervention has been shown to reduce the risk"

16 **Chaturvedi 1993** Page 50

"For the first time, gender differences in resting blood pressure in African-Caribbeans commensurated with mortality data".

17 **Health of the Nation 1992** Page 53

"All patients should be given advice to modify their lifestyle, as appropriate, by stopping smoking, reducing obesity, limiting alcohol and dietary saturated fat, and engaging in regular mild dynamic exercise... salt restriction may assist in lowering blood pressure"

18 **Health of the Nation 1992** Page 54

"Raised blood pressure is associated with obesity and overweight, excessive alcohol consumption, high sodium (principally from salt) and low potassium intakes"

19 **Health of the Nation 1992** Page 54

"The environments in which people live and work can have both favourable and adverse effects on their health and well being".

20 **Health of the Nation 1992** Page 54

"By the active promotion of physical environments conducive to health... by increasing knowledge and understanding about how the way people live affects their health"

21 **Ruskin 1956 (in)** Page 54

"If too much salt is used in food the pulse hardens".

22 **Health of the Nation 1992** Page 57
"To reduce the average percentage of food energy derived by the population from total fat by at least 12% by 2005, from about 40% in 1990 to no more than 35%"

23 **Health of the Nation 1992** Page 57
"A diet relatively low in fat and rich in vegetables and fruit"

24 **Health of the Nation 1992** Page 58
"The eating habits of the population play a significant part in the development and prevention of stroke, as blood pressure is affected by both obesity and sodium intake"

25 **Health of the Nation 1992** Page 59
"Sustained drinking in excess of these levels progressively increases the risk of raised blood pressure and stroke"

26 **Health of the Nation 1992** Page 60
"Smoking is estimated to account for up to 11% of stroke deaths".

27 **Health of the Nation 1992** Page 66
"Smoking prevalence is lower than the white population in people of African origin-Caribbean and Asian origin"

28 **Thesis definition of African-Caribbean** Page 69
"A person of African origin (racially mixed or unmixed), born in the Caribbean or had at least one parent of African origin who was Caribbean born Caribbean".

29 **Dometrius (1992)** Page 86
"Regression is robust. We could manufacture an artificial population and create a relationship between some variables that we know violates one or more regression assumptions. If we were then to take repeated samples from this 'population' and analyze the relationship with regression, the regression statistics would come pretty close to identifying the true population relationships. Obviously, a robust procedure has considerable value, and regression is such a procedure".

30 **Dometrius (1992)** Page 87
"In general, associations below .10 are not large enough to be important and may even be due to sampling error. Relationships between .10 and .20 are small but consequential. Relationships between .20 and .40 are moderate to strong, definitely large enough to be substantial and important. Any relationship above .40 can usually be considered quite strong"

31 **Stevens (1992)** Page 87

"The ideal situation in terms of obtaining a high R would be to have each of the predictors significantly correlated with the dependent variable and for the predictors to be un-correlated with each other, so that they measure different constructs and are able to predict different parts of the variance on the y. Of course in practice we will not find this because almost all variables are correlated to some degree. A good situation in practice then would be one in which most of our predictors correlate significantly with y and the predictors have relatively low correlations among themselves"

32 ***OPCS* 1993** Page 164

"Systolic less than 160 mmHg, not currently taking drugs (s) prescribed for high blood pressure" and hypertensive untreated as "systolic greater than 159 mmHg not currently taking drug(s) prescribed for high blood pressure"

33 **Cooper and Rotomi 1994** Page 169

"Not a single shred of direct (i.e. molecular) evidence exists to support the contention that higher rates of hypertension among Blacks have a genetic basis".

34 **Cruickshank 1993** Page 169

"The null hypothesis - that genetic differences account for little of the blood pressure differences - remains most likely".

35 **Cruickshank and Beevers 1989** Page 169

"Genetic factors within and between different ethnic groups do have powerful effects. Diabetes, hypertension and coronary heart disease tends to run in families; such familiarity many be partly genetic and partly due to shared environmental influences".

36 **Grim 1996** Page 170

"Debate continues as to whether it is their African ancestry or the Western environment that is more important in increasing the prevalence of hypertension in the African Diaspora above that of the indigenous Africans as well as of fellow inhabitants in the Western hemisphere"

37 **Marmot, Adelstein, Bulusu 1984** Page 170

"Mortality from respiratory disease is low in African-Caribbeans.... Part of the explanation for this pattern is the lower prevalence of heavy smokers among the Caribbean immigrants"

38 ***OPCS* 1993** Page 177

"There does not appear to be a clear or direct association between cigarette smoking status and raised blood pressure".

39 **Douglas 1989)** Page 181
"Traditional Caribbean diets can be recommended as being sound in terms of nutritional content and balance, being high in fibre and low in saturated fats".

40 **Douglas 1989** Page 181
"Health promotion messages should be based upon promoting traditional Caribbean diets while at the same time recommending areas for improvement in terms of lowering fats, sugar and salt intake".

41 **Beevers and Beevers 1992** Page 182
"There is every reason to believe that (a down ward) shift of blood pressure in the whole population could be achieved if the average sodium intake was reduced to below 100 mmol per day".

42 **Beevers and Beevers 1992** Page 185
"A population salt reduction strategy might prove particularly successful for African-Caribbeans".

43 **Chaturvedi 1992** Page 186
"Median blood pressures were as high in west African migrants as they were in Caribbean born migrants".

44 **OPCS 1993** Page 192
"Although the logistic regression did not show a smooth relationship, there was a general trend (age standardised), for those with higher blood pressure to report less stress.

45 **Pearson 1989** Page 193
"The stress from direct racist abuse, and direct structural discrimination also has an impact on health".

46 **Cooper 1984** Page 193
"The experience of being Black in a White racist society is the key stressor that accounts for the excess risk of hypertension among persons of African origin".

47 **Cooper and Rotomi 1994** Page 194
"The impact of racial antagonism on the individual psyche is not adequately captured by the concept of 'stress'".

48 **Cooper and Rotomi 1994** Page 194
"Living behind the veil", that *"connects racism to blood pressure control, rather than its ability to produce anxiety or other psychological discomfort"*

49 **Cooper and Rotomi 1994** Page 194
"Much of the difficulty with psychosocial research on hypertension in Blacks may reside in the paucity of theory-driven research that is embedded in the social context of communities".

50 **Cooper and Rotomi 1994** Page 195
"Unfortunately relatively little attention has been focused directly on this question and there are few tools available to study it at present".

51 **Cooper and Rotomi 1994** Page 195
"It remains entirely plausible, from a theoretical point of view, that the driving force underlying the differential in blood pressure between Blacks and Whites is the psychologic consequences of racial discrimination"

52 **Marmot 1989** Page 202
"Social class, as conventionally defined, cannot explain patterns of disease seen among immigrants ... forces determining mortality in immigrants are not well summarized by the conventional social class measure. Either this measure is not completely capturing the social position of the immigrants... or it is not reflecting the environmental and cultural/ethnic influence on disease risk.... While we should pay great attention to the social and economic position of immigrants, these are unlikely to be the only factors that determine the pattern of disease".

53 **Marmot, Adelstein, Bulusu 1984** Page 202
"Social class per se cannot account for many of the ethnic differences in disease... the SMRs from hypertension in West Indians ... is larger than the SMR in the most disadvantaged social class in England and Wales (Class V)"

54 **Marmot, Adelstein, Bulusu 1984** Page 204
"The disease patterns might be expected to be strongly influenced by those of the old country and by the process of migration itself"

55 **Senior and Bhopal 1994** Page 205
"Ethnicity's fluid and dynamic nature means that results of research may rapidly become out of date - results should not be generalised across time, generations, or populations with different histories of migration, except with great caution".

56 **Webster and Fox 1989** Page 205
"Does the term include solely immigrants, or also those born in the 'new' country to parents (or grandparents) born overseas. If the latter, is membership of a particular ethnic group restricted to those with both parents born in the same country; how are those of mixed parentage to be assigned?"

57 **Cruickshank and Beevers 1989** Page 206
"How many research workers have bothered to define the 'racial' group they study. To our knowledge only Miller has categorised black or Indian groups, whom he studied in Trinidad, by grand parental origin rather than by immediate appearance as a surrogate for 'race'".

58 **Chaturvedi et al 1993** Page 208

"We have demonstrated clear differences in blood pressure between the two ethnic groups (African-Caribbeans and Europeans*) and for the first time sex differences in resting blood pressure in Afro-Caribbeans commensurate with mortality data".*

59 **Cruickshank 1989** Page 209

"Contrary to the consistent findings in the USA, the average blood pressure levels among Black populations in the Caribbean, West Africa and Britain do not differ substantially from those in Whites".

60 **Stewart and Padfield 1994** Page 210

"known almost since the inception of the technique, yet the skill seems not to have been learnt by many practising medical and ancillary staff today"

61 **Pickering 1994** Page 211

"The measurement of blood pressure is much too serious to be left to physicians"

62 **Pickering and James 1989** Page 212

"One of the initiating factors may be the patient's view of the physician as a potential threatening authority figure, which results in the increased clinic pressure".

63 **Pearson 1989** Page 212

"In a society in which racism is deeply ingrained, ethnic minorities may have particularly bad experiences and low expectations of doctors and the NHS".

64 **Pearson 1989** Page 213

"People may become aware that they are labelled as 'awkward' if they challenge the submissive culture of being a patient"

65 **Cruickshank and Beevers 1989** Page 213

"An important challenge to doctors will be a change in attitude to being questioned. In particular... the 'informed-doctor-dispensing-to-ignorant-patient' approach will have to give way to an appreciation that many 'minority' patients will know more of recent developments in their condition than their doctors".

66 **Mancia and Zanchetti 1996** Page 213

"Even in the presence of a normal or low 24h average blood pressure, the possibility of an increased risk (of cardiovascular risk) possibly due to a high number of blood pressure peaks or increased blood pressure variability cannot be excluded".

67 **Cruickshank et al 1988** Page 216

"As yet, no ideal monotherapy exists for hypertension in Black patients"

68 **Cruickshank et al 1988** Page 216

"There are no ideal antihypertensive drugs for black patients".

69 **Marmot, Adelstein, Bulusu, 1984** Page 217

"The process of migration itself involves major changes that may affect disease risk. Economic and social strain, breaking of family ties, changes in smoking, drinking and dietary practices may all occur... it is possible that the stresses of migration may have contributed to hypertension in West Indians"

70 **Chaturvedi et al 1993** Page 217

"The reasons for these ethnic differences in blood pressure and the effectiveness of measures to prevent hypertension in people of African-Caribbean descent remain to be established"

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Abstract

Mortality statistics for the 1970s to 1980s have shown adult African-Caribbeans to be the highest risk group for hypertension and its most common outcome, stroke. The Department of Health has acknowledged hypertension in African-Caribbeans to be an important sub-priority area in the *Health of the Nation*'s systolic blood pressure reduction programme. To date, there has been very little researched aetiological data on blood pressure in this ethnic group. The significance of lifestyle related factors to hypertension within the general population, has been accepted by *The Health of the Nation*. These include diet, exercise, alcohol and smoking habits, obesity (body mass index), as-well-as socio-economic conditions. Health behaviour modifications are judged to offer an effective non-pharmacologic approach towards the prevention and management of hypertension. This study carried out 1994-1997, examined the association between systolic blood pressure and a number of lifestyle factors in a workforce group of 164 African-Caribbeans -138 women and 26 men.

Design: Information about lifestyle habits, and other related factors was collected from a survey, through face-to-face interviews, by a single observer, using pre-designed structured questionnaires. Blood pressure, heart rate, height, and weight were measured and recorded by the same (single), observer. Up to six repeated measurements and progress reports for subsets of the study group were done. Multivariate linear regression analysis was used to develop prediction models at significance levels of <0.05.

Results: Age related rise in blood pressure was evident, and age had the strongest independent linear relationship with systolic blood pressure (Pearson's $r = 0.43$). The most significant (positive) lifestyle factor was body mass index by group (Spearman's $r = 0.26$). Negative associations were found with alcohol and smoking habits. No association was found with levels of physical activity. Unexpectedly a negative association was found with adding salt, (Spearman's $r = -0.23$), but no association was found with dietary habits in general. Subjects who were least educated and had the lowest job grades were more predisposed to high blood pressure. Apart from brother's blood pressure history, familial hypertension was not evident. Negative associations were found with factors relating to racism. UK-born subjects generally recorded significantly lower blood pressures than their (usually older) non-UK-born African-Caribbeans colleagues, but this difference was marginalised with age adjusted analysis.

Conclusion: Apart from body mass index, lifestyle and other psycho-socio-economic factors examined generally did not contribute to high blood pressure in the subjects. Some significance was found in the 'wrong direction', that is, negative when positive was anticipated. There was no indication that lifestyle related factors were of any greater significance to the blood pressure values of African-Caribbeans than has been reported for the general population. Findings reflected those reported by the *OPCS* for the general population. However, there is an urgent need for much more research in the area.

1 First Chapter

Overview of the Research

1.1 OBJECTIVE

1.1a Research Goal

This study aimed to determine associations between a number of lifestyle-related factors and **systolic blood pressure** in a workforce group of African-Caribbeans. The emphasis on systolic blood pressure, (Korotkoff Phase 1), reflected the *Health of the Nation's* (1992, p.20) target: ***"to reduce mean systolic blood pressure in the adult population by at least 5 mmHg by the year 2005".*** Systolic blood pressure was also emphasised by the World Health Organisation (WHO1993) in its guidelines for the management of mild hypertension, in which it was stated: ***"Emphasis is also placed on systolic blood pressure as a criterion for decision making"*** (p.905).

1.1b Expected Outcome

National surveys and reports concerning the (general) British population have determined a significant association between lifestyle (health behaviour) and blood pressure, (*Health of the Nation* 1992; OPCS, 1993). African-Caribbeans currently have the highest hypertension mortality rates (Balarajan and Raleigh 1993). It was therefore, expected that the participants in this study would record mean systolic blood pressures above the national average. It is widely accepted that lifestyle factors have a significant impact on blood pressure. Given that African-Caribbeans have the highest rates of hypertension in the UK (Balarajan and Raleigh 1993), it was theorised that a significant number of the lifestyle factors examined in this study would be 'positively' associated with the blood pressure values of the subjects. The

exceptionally high rates of hypertension in the UK African-Caribbean population made it feasible to theorise that one or more of the lifestyle factors examined in this study could explain a significant percentage of the blood pressure variability among members of this group.

1.1c Why This Research

The government's White Paper, The *Health of the Nation* (1992, p.45), targeted five main priority areas for specific attention. Priority 'A' was coronary heart disease and stroke. The main risk factors associated with stroke were named as cigarette smoking, excessive dietary intake of saturated fatty acids resulting in raised plasma cholesterol levels, lack of physical activity, and raised blood pressure. In addition, it was stated that: ***"...Raised blood pressure is the most important risk factor for stroke."*** (p.46). In 1993, the *Health of the Nation*, through the Department of Health, published *Ethnicity and Health* - a guide for the NHS, (Balarajan and Raleigh 1993), which detailed priority areas with regards to ethnic (minority) groups, (see appendix A). In that manual, the morbidity and mortality statistics (1979-83) reviewed by Balarajan and Bulusu (1990), were used to highlight the significant rates of hypertension in the African-Caribbean population. The manual reiterated the findings that:

> ***"Mortality from hypertensive disease in England and Wales during the period 1979-83 was four, (4) times greater than the national average in Caribbean men, and seven times greater in Caribbean women"*** (p.18).

*See appendix A1

Essentially, the *Ethnicity and Health* publication spotlighted hypertensive disease in the African-Caribbean population as one of the sub-priority areas in the Government's *Health of the Nation* programme. Other specialist in the area, one of the strongest proponents being Cruickshank, have echoed this opinion. Cruickshank (1993) contended that the *Health of the Nation's* targets offered ethnic populations a major

opportunity for prioritising prevention strategies with regards to hypertension and stroke. He stated that:

> ***"For no group could this be more opportune than people of Caribbean origin for whom much of the stroke burden can be prevented because its major underlying risk factor of high blood pressure can be treated effectively - but only by controlling blood pressure to adequate level."*** (p. 28)

Although a general acknowledgement of the significance of hypertension in African-Caribbeans had been made in the *Health of the Nation* (1992, p.120), there was no information in the later OPCS (1993), report specific to this high-risk ethnic group. However, in the *Ethnicity and Health* (1993), publication in which hypertension within this ethnic group was given greater prominence, it was stated that: ***"the underlying aetiology and causation of stroke*** **(and hypertension)** ***is different*** (in African-Caribbeans) ***from that seen in the indigenous population"*** (p.54). It was further stated that although hypertension and stroke, ***"are encountered by all groups in the UK population, the causation and hence the modes of prevention may differ"*** (p.54). This statement was interpreted to mean that perhaps the significance of lifestyle related factors was markedly different in African-Caribbeans than in the indigenous (White), population. In the publication, the authors further noted that: ***"there are significant differences between the various ethnic minority groups in terms of socio-economic status, lifestyles, and genetic predisposition"*** (p.54). No indication as to the magnitude and direction of this difference was offered, but the authors opined that: ***"The strategy for the future needs to incorporate...further investigation of the aetiology of conditions that place ethnic minority groups at greater risk"*** (p.56). This proposed strategy related to a question posed earlier in the publication, namely, ***"How can we develop services for the early detection and management of hypertension among African-Caribbeans"*** (p.20).

Balarajan and Raleigh's observations, as outlined in *Ethnicity and Health* (and that of others with interest in the field), of the need for greater knowledge on the aetiology of hypertension in African-Caribbeans underscored the necessity for (aetiological), research relating to lifestyle on this group. Blaxter (1990) had earlier highlighted the sparsity of information on the lifestyles (health behaviour) of African-Caribbeans and other black populations in the UK.

It seems evident that accumulation of lifestyle related information would be of immense benefit to those organisations or persons formulating hypertension detection prevention, and management programmes in African-Caribbeans. It was with these postulates in mind that the author of this project was motivated to examine the association between blood pressure and lifestyle in a workforce group of African-Caribbeans. In so doing this study will contribute to the current sparse research pool.

1.1d Main Research Questions

The main question addressed in this study was:

> **Were the recorded blood pressures of the volunteers significantly associated with their lifestyle, in particular, health behaviour patterns?**

If, generally, positive significance was found, this could indicate that lifestyle related factors were important precursors to the incidence and prevalence of hypertension in African-Caribbean general population.

1.1e Value of the study

The need for further research on hypertension in African-Caribbeans has been recognised by bodies such as the *London Hypertension Network*, which grew out of the *City and Hackney Hypertension Reduction Project*, (CHHRP: Trenchard-Mabere

1992), as well as expert researchers within in the field. Among the most noted proponents was Cruickshank (1993), who wrote, ***"There can be no question of the public health importance of hypertension and stroke for this*** (African-Caribbean) ***community"*** (p.30). Generally, experts within the field agree that causes of hypertension in UK African-Caribbeans were unestablished. Cruickshank noted that while a few findings have indicated a partial link to body mass indices, the aetiological significance of other factors, genetic or environmental remains largely unknown. The scarcity of information on hypertension in African-Caribbeans, and the need for further investigation on the aetiology of the condition was also acknowledged in the Government's *Ethnicity and Health* (1993) publication.

This study's observational investigation into the aetiology of the disease with respects to lifestyle factors is in keeping with the *Health of the Nation's* initiative of aiming to reduce systolic blood pressure. In view of the shortage of aetiological information on the subject, it is hoped that the findings will become a valuable reference source for health professionals and administrators planning hypertension prevention and management programmes.

1.2 DESIGN

1.2a Eligibility

Any current (1994-95), employee of the Lambeth Social Services, London, who was Caribbean-born of African descent or any employee who had at least one parent of African descent born in the Caribbean was eligible. Excluded were persons currently taking blood pressure medication (treated hypertensives) because of the likely

confounding factors. Further the individual's 'true' blood pressure could not be ascertained, as this would be masked by the medication. 164 volunteers, comprising 138 women and 26 men, participated in the programme.

1.2b Research Design

The research design was a one-sample survey, through face-to-face interviews, using pre-designed structured 'close-ended' questionnaires, conducted on a workforce group of volunteers, (see appendix B). This retrospective design was selected because the study was essentially a general examination of the possible pre-existing effects of certain lifestyle factors on blood pressure, using a single sample. The research was designed to be exploratory, aiming to generate further investigations and research.

Self-reported information[1] was collected and recorded about alcohol consumption, cigarette smoking, dietary habits, physical activity levels, educational and job status, housing conditions, general health, perceived stress levels, and a number of other lifestyle related socio-economic factors, including family background and racism. The questions also related to the subject's own current and past blood pressure status and that of their closest blood relatives. Information was also collected on the subjects' 'ethnicity', which included country of birth. A single observer, (the author), measured blood pressure heart rate, height and weight (recorded as body mass index), during the individual's one hour interview. There were repeated measures of blood pressure, heart rate and body weight, as well as progress reports, for subsets of the sample population. These repeated measurements and progress reports occurred

[1] All information, except anthropometric measurement, was self-reported and given during the interview.

approximately every 8 weeks, for up to twelve months: a maximum of six measurements per volunteer.

1.2c Outline of Analysis

The 138 female and 26 male subjects were grouped as a single sample. The main sub-divisions for analytical purposes related to gender, age and UK-born versus Caribbean-born. Systolic blood pressure was the main dependent (continuous) variable, but other variables were designated dependent status when expedient, for example, diastolic blood pressure, and age. The independent variables excluding those derived from the anthropometric measurements, (body mass index and heart rate), were categorical, therefore, discrete. These non-continuous variables were converted to ordinal format, allowing for interval-scale (parametric) analysis. Preliminary statistical tests established the normality of the systolic blood pressure distribution curve.[2] This, coupled with the conversion of the ordinal independent variables, into *'dummy variables'* facilitated the use of linear multivariate analysis to develop the final statistical models. Following the convention of social science, the significance level (decision criterion) was set at Sig = <.05, (Dometrius 1992).

Data were analysed using the Statistical Programme for Social Science (SPSS). After the information from the questionnaires was entered, checked, and coded, frequency distributions and descriptive results were established. Simple exploratory tests were concluded, particularly stem and leaf analysis, which detailed percentiles and normality. These early stage analyses allowed for data sorting and familiarisation, and gave early indications of distributions. Early (first and second stage), exploratory analysis used both parametric and non-parametric tests, but cross-tabulation statistics

was the main analysis tool. Relevant cross-tabulation statistics were Chi-square, (two-way contingency tables), which examined the presence (or absence), of substantive significance between blood pressure and the (ordinal), independent variables. Gamma (γ), statistics were examined because it is deemed to be the simplest measure of the strength of ordinal data (Dometrius, 1992, p.309). Linear regression enter method (with the use of 'dummy variables' was carried out, and used to develop models. The final, prediction model, was derived from linear regression stepwise method. To facilitate the use of cross tabulations, the (continuous) dependent variable, systolic blood pressure was re-classified on an ordinal scale as shown in table 1.1 below.

Table 1.1 Systolic Blood pressure Classification

Category A	Below normal	= SBP	<107 mmHg
	Normal	= SBP	107-137 mmHg
	Above normal	= SBP	> 137 mmHg.
Category B	Normal	= SBP	< 138 mmHg
	High	= SBP	> 137 mmHg.

The results obtained from cross tabulations enabled the identification of the most probable variables for further (multi-way) analysis.

[2]Diastolic blood pressure was also normally distributed

2 Second Chapter

Introduction

2.1 THE PROBLEM

2.1a Hypertension in African-Caribbeans

Caribbeans of African descent have the highest mortality rate of hypertensive disease in the UK (Ethnicity and Health 1993). Early evidence of the predominance of hypertension in African-Caribbeans was derived indirectly from hospital admissions and mortality (death rate) figures for the period 1970-78, (Marmot, Adelstein, Bulusu 1984). The most recent recorded data dates back to the period 1979-83, (Balarajan and Bulusu 1990). These two reports, derived from death certificates, were the primary references in the Department of Health publication *Ethnicity and Health-a guide to the NHS*, (Balarajan and Raleigh 1993), in which the 'problem' of hypertension in the African-Caribbean population, was expounded. The *Ethnicity and Health* publication served to highlight hypertensive disease in African-Caribbeans as a priority area for the *Health of the Nation* initiative programme. However, the morbidity and mortality figures offered as reference for the high rates of hypertension in African-Caribbeans were between 15 to 25 years old, (Marmot, Adelstein, Bulusu 1984; Balarajan and Bulusu 1990). None-the-less, in the 1990s, they remain the official (primary) sources of information on hypertension in African-Caribbeans (Balarajan and Raleigh 1993; Cruickshank 1993).

These mortality reports did not include as a primary concern, any substantial aetiological data, that is, information on the causes of the disease. To date (approximately two decades later), there have been very few epidemiological and aetiological surveys on hypertension in African-Caribbeans. However, those that

were conducted have generally served to augment the mortality findings, further illuminating the 'problem' of hypertension in African-Caribbeans, (Cruickshank 1993).

2.1b Hypertension and Lifestyle Risk Factors

Within this dissertation, lifestyle is defined as a way of life or style of living that reflects the individual's social and economic environment and the attitudes and values of the person. This 'way of living' is variable, that is, changeable so that individuals can modify their way of living, and adapt to a new lifestyle. Lifestyle changes may impact on the individual's health, including blood pressure. Beilin (1988) calculated that the combined changes in diet, alcohol consumption and physical activity could produce a fall in population mean systolic blood pressure of 10-20 mmHg. British health authorities have acknowledged that lifestyle factors are important considerations when examining the pathogenesis of hypertensive disease, (*Health of the Nation* 1992; OPCS 1993). Risk factors for hypertension noted have been cigarette smoking, excessive consumption of alcohol; excessive consumption of sodium (common table salt); excessive energy intake; (obesity); lack of physical activity. In the *Health of the Nation* (1992), it was stated that:

> ***"A reduction in mean blood pressure in the population of 5 mmHg could be expected from success in achieving the obesity and alcohol target, together with the reduction in sodium intake. It has been estimated that such a reduction would result in a 10% reduction in mortality from coronary heart disease and stroke"*** (p.57).

The primary focus of attention in this study included lifestyle factors named in the *Health of the Nation* report (1992) which were reiterated in the OPCS report (1993). These are dietary habits, salt intake, alcohol consumption, cigarette smoking, physical

activity, obesity (body mass index), and perceived stress. Socio-economic factors, social status, and housing were examined.

2.1c Caribbean Lifestyle

A report by Blaxter (1990) concluded that there was insufficient researched (documented) information on the African-Caribbean lifestyle to determine the significance to their health. The deficiency of information on the aetiology of hypertensive disease in African-Caribbeans, including the importance of lifestyle factors, was further emphasised in the *Ethnicity and Health* (1993), publication. In it was stated ***"Information about lifestyle and diet*** (among Caribbeans) ***is scanty, and the findings are not always consistent"*** (p.18). Like Blaxter earlier, Balarajan and Raleigh (1993), noted that ***"the sub-groups of black people,*** (including African-Caribbeans), ***are not homogeneous and show considerable diversity in terms of culture, lifestyle, social class and economic status"*** (p.18). Balarajan and Raleigh offered references to demonstrate that findings relating to lifestyle in Caribbeans were not consistent, noting for instance that some studies showed a low prevalence of heavy smoking in Caribbean men, (Balarajan and Yuen 1986; Watson and Murray-Lyon 1989), while others reported a high prevalence, (Miller and Kotecha et al 1988). Again, the alcohol consumption of African-Caribbeans was reported by Balarajan and Yuen (1986), to be below the national average, but Cochrane (1989), found alcohol related morbidity to be rising in Caribbean men.

The shortage of researched information on the African-Caribbean lifestyle, and the inconsistencies in results of the few studies that have been carried out, has made it difficult to determine the possible or likely effects of health behaviour practices on the blood pressure patterns of that ethnic group. Yet, in the light of the significant effect

of lifestyle on the blood pressure of a wide cross section of populations world-wide, (WHO 1991; JNC V 1993), it is reasonable to infer that lifestyle habits could explain a significant proportion of the African-Caribbeans' seemingly manifest propensity to hypertension. The reduction of lifestyle related risk factors was a major prevention strategy of the *Health of the Nation*. Changes in pertinent lifestyle patterns may be one of the priority strategies that could to be effectively employed towards the prevention, reduction and control of hypertension in African-Caribbeans.

The scarcity of information on the aetiological significance of lifestyle habits to hypertension in African-Caribbeans makes it prudent at this time to contribute to this area. This was central to the purpose of this research project. The study was developed on the premise that lifestyle (health behaviour), has been established as a significant precursor to hypertension in the general population, (Health of the Nation 1992; OPCS 1993). It is then highly likely that the pertinent lifestyle factors would be strong contributory agents in the pathogenesis of hypertensive disease in high risk African-Caribbean population.

2.1d Thesis Hypothesis

As such the thesis **hypothesis** is stated as:

> **Lifestyle factors will explain a significant proportion of the variability in blood pressure within the group of African-Caribbeans being studied.**

SUMMARY OF CHAPTERS 1 AND 2

Morbidity and mortality statistics on hypertension (and stroke), derived in the 1970s and early 1980s, suggested that African-Caribbean (migrants), were the highest risk group in Britain. The *Health of the Nation* considers these figures pertinent to the

current UK African-Caribbean population. Certain lifestyle factors including cigarette smoking, diet, alcohol intake, and physical activity levels are acknowledged primary contributors to the risk of developing essential hypertension. It is therefore, probable that these lifestyle factors have aetiological significance to the pathogenesis of hypertension in the high risk African-Caribbean ethnic group. This study examined a number of lifestyle and other relevant variables which related to the blood pressure, in a workforce group volunteers of 164 African-Caribbeans, comprising 138 women and 26 men. The volunteers were drawn from the large Caribbean workforce of a government department in the inner city London Borough of Lambeth. Repeated blood pressure and heart rate measurements, as well as progress reports of subsets of the study population were mapped over a period of up to twelve months.

This study aimed to gain an insight into the general living and working environment of a group of African-Caribbeans and to determine possible associations between the participants' lifestyle their blood pressure levels. The essential question was: *Did those subjects in the higher blood pressure categories have less desirable lifestyle 'scores' than those in the lower blood pressure categories?* Rotated, the question becomes: *Did the subjects with the less desirable lifestyles have higher blood pressures than the subjects with more desirable lifestyles?*

The findings of this study will add to the sparse information pool on hypertension in African-Caribbeans. It is a contribution to the Government's *Health of the Nation* initiative, which seeks to establish methods of preventing and managing hypertension (and stroke) in the general population.

3 Third Chapter

Caribbeans in Britain

3.1 HISTORY

3.1a Caribbeans: Who They Are

The 1991 census reports that approximately one-percent, (1%), of the total population in England and Wales is African-Caribbean, making them the second largest ethnic minority group in Britain, behind the 1.7% Indian community. Ethnic minorities make up six-percent of the total population. There are approximately 500-thousand, (499,964), African-Caribbeans in Britain, fifty-percent of which were British-born. (Census, 1991).

UK-African-Caribbeans originated from the (former),[3] British West Indian Islands, (BWI), which were part of the old British Commonwealth. These islands were Barbados, Grenada, Jamaica, Montserrat, Trinidad, St Kitts, St Lucia, and others. (British) Guyana, which is on the South American mainland, was also a West Indian territory (see appendix C).

The ethnic composition of these islands is largely multi-racial to include people of African, European, Indian, and Chinese, origins. In some of the territories, (especially Trinidad and Guyana), there was almost an equal balance of people of African and Indian origin, with minute percentages of other ethnic groups. However, the majority of the islands, (including Jamaica), had a disproportionately

[3]Many of the Islands gained political independence in the 1960s, 1970s and 1980s

large percentage (up to 80-90%) of natives who were (primarily)[4] of African descent. These African descendants had been transported to the Caribbean from Sub-Sahara Africa, (Mainly West Africa) during the Atlantic Slave Trade, (Davidson, 1980).

3.1b Caribbeans Migration to UK

The 1991 census revealed that approximately fifty-percent (142,483) of the total Caribbean migrants originated from the island of Jamaica, the largest and most populated of these former British ruled West Indian Islands. The majority of the Jamaican immigrants, as well as those from the other West Indian territories were of African descent.[5] It is these migrants and up to four generations of their offspring, which comprised what is now African-Caribbean population of Britain.

Prior to World War II, there were relatively few Caribbeans in the UK. West Indians migrated to Britain in significant numbers in the 1950s and 1960s, post World War II when Britain was in the process of rebuilding, and there was a great demand for skilled labour, (Patterson 1963; Peach 1968). The British dependent territories (including the West Indian - Caribbean Islands) had been adversely affected by the war, as the 'Mother' country could no longer afford to offer them the economic support they needed. Jobs were scarce in these territories, and the Caribbean islanders responded to the bidding of the 'Motherland' and sought opportunities in Britain, (Patterson 1963; Peach 1967). Migration reflected the

[4]Integration and inter-marriage between different ethnic groups was common

[5]A substantial percentage of the West Indian Migrants, especially those from Trinidad and Guyana were Indian. There were also migrants from other West Indian ethnic groups.

needs in the job market. In the very early years of migration, there was a relatively high proportion of skilled workers, but this decreased over time, (Patterson 1963; Peach 1967). Jobs at the lower end of the scale were plentiful, and consequently, early West Indians (and migrants from other British dependent territories), were primarily recruited into jobs that the indigenous workers did not fill (Patterson 1963; Peach 1967). These were mainly low paid, low status jobs, which required these immigrant employees to work long and unsociable hours.

By the late 1950s to early 1960s the British economy had made substantial recovery resulting in greater stability, and diminishing need for immigrant labour. Immigration from the islands was now inversely related to unemployment rates in the new economy (Peach 1981). This was also evident as government enforced measures to curtail immigration from the West Indian territories with the passing of the 1962 Commonwealth Immigration Act. This Act allowed for only skilled workers, primarily in the health sector (especially nurses), as well as dependants of those migrants already settled in the UK, to be eligible for permanent residence. By the mid 1970s mass migration had been significantly reduced (Peach 1981).

3.2 CURRENT AFRICAN-CARIBBEAN POPULATION

3.2a Geographical Location

The African-Caribbean population is not evenly distributed across Britain, but most reside in a small number of inner city local authority areas, (Census1991). The vast majority, 94%, (468,979), of African-Caribbeans reside in the London boroughs.

Within the inner city London Borough of Lambeth, the borough within which this research was carried out, there were 30,789, African-Caribbeans recorded in the 1991 census, (OPCS 1991). This converts to 12.6% of Lambeth's population, making it among the largest concentration of African-Caribbeans in Britain.

3.2b Caribbeans in this Study

The participants of this study were Caribbean-born and UK-born African-Caribbeans employees of the Directorate of Social Services, Lambeth. During the field study period of this survey, (1994-1995) there were 1200 African-Caribbean employees 84%, of which were females. These 1200 African-Caribbeans formed the sample frame of the research. Table 3.2 below gives a breakdown.

Table 3.2 African-Caribbean Employees at Lambeth Social Service

Total workforce	Total Caribbeans	Caribbean males	Caribbean females
2744	1200	198 (16%)	1002 (84%)
Source: Directorate of Social Services – Lambeth, 1994-95			

Lambeth Social Services had a large African-Caribbean work force, the majority (84%) of which were female.

The researcher was informed by the Directorate of Lambeth Social Services that its African-Caribbean employees formed a comprehensive cross-section of the employed African-Caribbean population in South London, and London in general, (*Meeting with the Director of Equality and Race Division, Lambeth Social Services, February 1994*). Affirmation was also given that these employees were reasonably representative of the employed African-Caribbean population in London, as it was

felt that the distribution was reasonably proportionate of socio-economic groups of that ethnic group. On the strength to these assurances, it was concluded that results derived from the sample group of 164 subjects, could be generalised to include not only the Lambeth African-Caribbean employees, but also other London based employed persons of this ethnic group.

4 Fourth Chapter

Review of Previous Studies

4.1 HYPERTENSION

4.1a General Overview

Blood pressure varies across and within populations world-wide, (Intersalt 1988; WHO 1993). It is a continuum, with wide variations not only across individuals within a single population, but also within a single individual, over a given period of time which may be years, months or even within a 24-hour period (Swales 1995). Blood pressure that is maintained below a certain level is deemed 'normal', and is termed 'normotension'.[6] Persistently high blood pressure is termed hypertension, but: ***"the dividing line between 'normotension' and 'hypertension' is arbitrary. The current definition is that this line is the level of blood pressure above which intervention has been shown to reduce the risk"*** (WHO 1993, p.392). Individuals within a single population, or even a small subset of a population will have varying degrees of hypertension, ranging from very low blood pressure (hypotension), to normal (normotension), to high (hypertension). A single individual may have multiple variations in blood pressure within any given time frame, but even in 'borderline hypertension', blood pressure may not be persistently raised, (WHO 1993).

Hypertension can be secondary whereby a (single) cause for the raised blood pressure has been identified, or primary, in which case the hypertension very often cannot be attributed to a single cause, or no specific cause(s) have been identified.

Variations in the blood pressure of individuals within a society are largely explainable by genetic and environmental factors,[7] including age, gender, socio-economic conditions and ethnic background, (JNC V 1993; Swales 1995). Hypertensive disease is, therefore, multi-factorial. Unlike secondary hypertension, primary hypertension (also known as essential hypertension), usually involves complicated and diverse traits of both environmental and genetic factors, (Swales 1995).

4.1b Classification of Hypertension

The arbitrary nature of blood pressure has contributed to a number of non-standardised categorisations of hypertension. The British Government's *OPCS* (1993), categorisation is shown in table 4.3 below.

Table 4.3 OPCS Hypertension Classification

Normotensive untreated	SBP less than 160 mmHg and DBP less than 95 mmHg, not currently taking drug(s) prescribed for high blood pressure.
Normotensive treated	SBP less that 160 mmHg and DBP less than 95 mmHg, currently taking drug(s) prescribed for high blood pressure.
Hypertensive treated	SBP greater than 159 mmHg and/or DBP greater than 94 mmHg, currently taking drug(s) prescribed for high blood pressure.
Hypertensive untreated	SBP greater than 159 mmHg and/or DBP greater than 94 mmHg, not currently taking drug(s) prescribed for high blood pressure.

Hypertension is defined as systolic blood pressure greater than 159 mmHg.

The World Health Organisation's, (WHO 1993) has more stringent standards, as shown in table 4.4 below.

[6]However, blood pressure that is persistently very low it is termed ***hypotension***

[7]This study will only examine environmental factors.

Table 4.4 WHO Hypertension Classification

	SBP mmHg		DBP mmHg
Normotension	<140	and	<90
Mild hypertension	140-180	and/or	90-105
Subgroup: Borderline	140-160	and/or	90-95
Moderate/severe hypertension	≥ 180	and/or	≥ 105
Isolated systolic hypertension	≥ 140	and	<90
Subgroup: Borderline	140-160	and	<90

Source: WHO\SH Guidelines, 1993

Hypertension (Mild) is defined as systolic blood pressure greater than 139 mmHg.

The American body, the Joint National Committee on Blood Pressure, (JNC V 1993), considered values of 140 mmHg systolic or 90 mmHg diastolic to be worthy of treatment. The Committee's classification is shown in table 4.5 below. This classification was used as the main guideline for this research, primarily because blood pressure was measured by similar standards, and the sub-categorisations were most suitable for this study.

Table 4.5 JNC V Classification of blood pressure for adults (18 years +)

Category	SBP (mmHg)	DBP (mmHg)
Optimal	<120	<80
Normal	120-129	80-84
High Normal	130-139	85-89
Hypertension...		
Stage 1 (mild)	140-159	90-99
Stage 2 (moderate)	160-179	100-109
Stage 3 (severe)	180-209	110-119
Stage 4 (very severe)	≥ 210	120
Based on an average of two or more readings on two or more occasions in individuals not taking anti-hypertensive medications and not acutely ill. When average falls in different categories of systolic and diastolic blood pressure the higher category applies. (JNC V, 1993)		

Hypertension is defined as systolic blood pressure greater than 139 mmHg.

4.1c Hypertension World-wide

Populations described as 'isolated' have shown virtually no evidence of the 'normal' age-related change in blood pressure, found in more technologically developed countries, (Shaper 1974; Carvalho 1989) and hypertensive disease is rare or non existent in these non-industrialised communities, (Intersalt 1988). Arterial diseases, including hypertension, have repeatedly been shown to have a strong positive association with industrial and technological development, (Shaper, 1974; Intersalt 1989). This has been evident in some sub-Saharan African countries in which the blood pressure patterns of rural populations have been altered to resemble that of urban (more westernised), dwellers within a short period after migration, (Poulter et al 1990). Migrants generally adopt the pattern of disease in their new host environment, (He et al 1991). In general, members of societies appear to acquire a predisposition to age-related increases in blood pressure following adaptation of western lifestyles, (Carvalho et al 1989; WHO Statistics 1991). The World Health statistics suggested that the epidemiology of hypertension in developing countries is increasingly following the pattern of western industrialised countries.

4.1d Hypertension in the UK

Recent figures, (OPCS 1993), indicated that hypertension in Britain was in keeping with the general western phenomena. British categorisation of hypertension is a systolic blood pressure greater 159 mmHg and/or diastolic blood pressure greater than 94 mmHg. By these standards, 20% of adults were hypertensive in 1993. Twelve percent of men and 10% of women were classed as untreated hypertensive. Using the World Health Organisation standards, the number of hypertensives in Britain was closer to 40%. Mean systolic blood pressure in the UK population was

137 mmHg, and diastolic was 76 mmHg. Systolic pressure was higher for men, (139 mmHg), than for women, (136 mmHg). Men also had a higher mean diastolic blood pressure than women, (78 mmHg and 74 mmHg respectively). Both systolic and diastolic pressures increased with age, but diastolic pressure tailed off among the oldest group of men.

4.2 HYPERTENSION IN WESTERN BLACKS

4.2a Overview

Western Blacks originated from sub-Sahara Africa, where there is a wide variation in blood pressures among various ethnic groups (Intersalt 1988). The blood pressures of members of these societies have consistently been found to be markedly lower than that of their western kin, (Akinkugbe 1985; Intersalt 1988; Wilson 1990). Kenyans, (in East Africa), are amongst the lowest blood pressure populations in the world (Carvalho et al 1989). The West African country of Nigeria, reportedly has amongst the highest rates of hypertension in sub-Sahara Africa, but still significantly lower than that of comparable black populations in the USA and the Caribbean, (Akinkugbe 1985).

Ethnicity and blood pressure has been investigated in the USA since at least the 1930s, (Adams 1932; Saunders and Bancroft 1942). These early studies and numerous since, have served to established that African-Americans have the worst recorded rates of hypertensive related disease (Hypertension Detection & Follow-up programme 1977; National Center for Health Statistics 1988; Akinkugbe 1985).

Populations of African origin in South America exhibit the same trend as that of North America, (James et al 1991). Reports from Britain, (Cruickshank 1993), and the Caribbean, (Grell 1983; Akinkugbe 1985), have offered evidence of the similarity of blood pressure patterns in people of African origin to that found in their African-American kin. Western people of African origin therefore, appear to share similar high mortality rates from hypertensive disease, with African-Americans having the highest rates.

4.2b Explanations of the High Blood Pressure in Western Blacks

Multitudes of environmental explanations and theories have been propounded on the subject of high blood pressure in peoples of African descent, with most originating from the USA. Popular explanations are those concerning socio-economic status, (Syme et al 1974); educational status, (Dyer et al 1976); racism, (Armstead et al 1989); behavioural stress, (Johnson et al 1992); and general psycho-social factors, (Myers and McClure 1993). A genetic explanation, primarily linked to sodium intolerance has been among the most popular. Theories by proponents such as Wilson and Grim (1991); Law et al (1991) and Grim (1996), have suggested that the pressor effect of dietary sodium, which is probably genetically determined, is more pronounced in Blacks than in their White counterparts. In general adverse physiological response of black people to sodium has proved increasingly popular (Falkner 1990). While the numerous studies have resulted in a plethora of conclusions and hypothesis, the subject is still unresolved in the 1990s.

4.2c UK Caribbeans versus USA Blacks

Awareness of the possible predisposition to hypertension in persons of African ancestry in the USA began in the 1930s, (Adams 1932), but it only became the subject of focus in Britain in the 1970s. In the 1960s, the estimated prevalence of essential hypertension in African-Americans was approximately twice as high as that of whites, (McDonough 1964). Twenty years later, official government reports stated that death rates amongst Black Americans from hypertensive disease were more that three times (3.5 times), greater than the national average (National Centre for Health Statistics 1988).

Epidemiological patterns in hypertensive disease, between Blacks and Whites in the USA and Blacks and Whites in Britain have appeared similar (Akinkugbe 1985). Cruickshank (1993), pointed out that major blood pressure studies in the USA comparing Blacks and Whites such as the Evans County study, (Comstock 1957; Tyroler 1985), compared quite closely with the UK's Birmingham Factory study results, (Cruickshank et al 1985). Despite the close comparisons, levels in African-Caribbeans, both native and UK-based, have been found to be significantly lower than that of their North American kin (Akinkugbe 1985; Wilson et al 1991).

The quantity of researched material on the subject in America far exceeds that of the UK, where there has been relatively little research. Relative to the USA, the British are at a very early research stage. Theories as to the aetiology of hypertension in UK African-Caribbeans have to some extent depended on studies that have been carried out on African-Americans, as well as investigations in the Caribbean islands. Some of the conclusions and theories postulated with respect to African Americans

and other western Blacks have been imported as reference points for investigations of the problem in UK African-Caribbeans, (Cruickshank 1993). These studies may have significant relevance to UK African-Caribbeans, but the absolute relevance cannot be judged until more UK studies have been completed.

4.2d Hypertension in Native African-Caribbeans

Among the earliest recorded hypertension surveys carried out in the Caribbean[8] was that of Saunders and Bancroft (1942) in the Virgin Islands. Surveys were later carried out in other islands including: the Bahamas, (Moser et al 1959); St. Kitts, (Schneckloth et al, 1962); Jamaica, (Miall et al 1961; Ashcroft and Desai 1978); St. Lucia, (Khaw and Rose 1982); Trinidad (Miller and Kirkwood et al 1988) and quite recently in Barbados, (Foster et al 1993). These and other similar studies in the region sought to acquire epidemiological information in order to define the blood pressure distribution in the populations. The findings reported from these and other Caribbean studies have been mixed. Miall reported (1961) that hypertension did not appear to be particularly common among Jamaicans of African descent, but twenty-years later Grell (1983) reported a high prevalence. A recent (unpublished) summary report of studies in the Caribbean, (Forrester and Wilks 1995), discerned that overall the findings concerning native Caribbeans have been varied, and highlighted the non-conclusiveness of the evidence which suggest a high prevalence of hypertensive disease in the Caribbean.

[8]Including the non-British territories

4.3 UK African-Caribbeans' Hypertension Problem

4.3a Discovery of the Problem

Historically, medical records have been a primary source of information detailing the 'official' hypertension status of African-Caribbeans, (Tunstall-Pedoe et al 1975; Cruickshank et al 1980; Marmot, Adelstein, Bulusu 1984; Haines et al 1987; Balarajan and Bulusu 1990). Within fifteen to twenty years of the start of mass migration by Caribbeans to the UK (in the mid 1970s), medical records suggested that these relatively recent immigrants had higher rates of hypertension and stroke than was found in the indigenous, (White), population, (Tunstall-Pedoe et al (1975). Tunstall-Pedoe's report revealed the possibility of an epidemiological and significant problem, from which the need for further research could be justified. By the late 1970s, these findings had been somewhat 'verified' as a result of the Northwick Park Heart Study (Meade et al 1978) and a follow-up study by Sever et al (1979). The prevailing clinical impression going into the 1980s was that African-Caribbeans were more prone to high blood pressure than their indigenous, White, counterparts.

4.3b African-Caribbean Studies of the 1970s

Northwick Park Heart Study: 1978

Meade (1978) compared black and white day and night shift workers. In that study, which emphasised haemostatic risk factors, significant ethnic differences in mean systolic blood pressure were recorded. The female comparisons showed significantly higher blood pressures in the black women. No significant differences were detected between black and white male workers on the night shift, but there was a significant difference between the ethnic groups on the day shift.

Sever et al (1979) tested plasma renin and noradrenaline on a sub-group of the Northwick Park subjects. No significant differences in plasma noradrenaline were established between Blacks and Whites, but Blacks had 55% lower plasma renin than Whites. The differences observed were independent of body mass index.

BIRMINGHAM HOSPITAL STUDY: 1980

Information from a Birmingham hospital admissions rates between 1974 to 1978, (Cruickshank et al 1980), supported the Northwick Park findings. Hypertension was more common in African-Caribbeans than both Asians and Whites.

4.3c African-Caribbean Studies of the 1980s

The report of the studies by Meade, Sever, and Cruickshank in the 1970s set the stage for further investigations into hypertension in African-Caribbeans. Two major surveys in the 1980s, The Birmingham Factory Screening Project, (Cruickshank et al 1985) and The north-west London inner city general practice study, (Haines et al 1987), produced results which were somewhat concordant with the earlier findings that African-Caribbeans seemed prone to high blood pressure.

THE BIRMINGHAM FACTORY SCREENING PROJECT: 1985

Cruickshank et al (1985) screened 1384 multi-racial factory workers, comprising 274 African-Caribbeans (173 men and 101 women), compared with 439 white men and 164 white women, with the Asians being the other main ethnic group. Fourteen lifestyle and family history independent variables were measured from the multi-ethnic workforce. The lifestyle variables included cigarette smoking, and alcohol intake. Positive associations were found between age, body mass index, and alcohol intake in both systolic and diastolic blood pressures. No consistent differences were

found in the average blood pressures of the three main ethnic groups, but a modest excess prevalence of hypertension amongst the African-Caribbeans over the Whites was detected. There were no significant differences in mean blood pressure by age (decade) in the different male groups, but African-Caribbean women were found to have greater mean systolic and diastolic blood pressures. However, the African-Caribbean women over 35 years of age were more obese by an average of 4 kg/m^2, (2.5 to 5 kg/m^2). Cruickshank concluded that the differences in blood pressures between Caribbean and White women was significant with African-Caribbean women over 35 years and paralleled the differences in body mass index.

THE NORTH WEST LONDON INNER CITY GENERAL PRACTICE STUDY: 1987

Haines et al (1987) surveyed routine medical records of a north west London general practice. Although no statistically significant differences in age related blood pressure between Blacks and Whites were found, the records showed that more Blacks were being treated for hypertension.

AMBULATORY STUDY: 1982

Another study of the 1980s was that carried out by Rowlands et al (1982) who compared ambulatory blood pressure patterns of African-Caribbeans with that of Whites. No difference was found between the groups.

SUMMARY OF 1970S AND 1980S STUDIES

The major drawback to these 1970s and 1980s surveys were that their sample sizes (of African-Caribbeans) were relatively small. Further, in some of the studies, analysis made no distinction between black people who were born in Africa, and those who were Caribbean. In the 1978 comparative study by Meade and his team, there were less than 56 Black male subjects, but they were not all Caribbeans as

some were west Africans. The Birmingham Factory study was significantly larger, comprising 274 Blacks, but again not all were African-Caribbeans, some being African. Their shortcomings acknowledged, the relative value of each of these studies could not be undermined.

4.3d African-Caribbean Studies of the 1990s

In this decade, there have been two important surveys on adult African-Caribbeans, the Northwest London Survey (Cruickshank et al 1991), and the Brent Study, (Chaturvedi et al 1993). These studies differed from previous surveys on the subject, in that the sample sizes were significantly larger, and they both included fasting for glucose tolerance tests. These factors, especially the larger sample sizes, contributed to the greater statistical power of these surveys over earlier ones.

NORTHWEST LONDON SURVEY: 1991

Cruickshank et al (1991) investigated the mechanisms leading to ethnic differences in hypertension in 344 subjects, comprising 106 African-Caribbeans. The subjects were drawn from the practice list in north-west London health centres. Overall, blood pressure, especially diastolic blood pressure, was higher in African-Caribbeans than in Whites.

THE BRENT STUDY: 1993

Chaturvedi et al (1993), was among the most comprehensive on the subject, comprising a sample of 514 African-Caribbeans, (211 men and 303 women), 155 white men, and 156 white women. The subjects were drawn from the lists of six family practices in the inner London borough of Brent. The survey sought to determine the prevalence of hypertension in African-Caribbeans and whether diurnal

blood pressure patterns differed in African-Caribbeans and Europeans. A sub-sample was measured with 24-hour ambulatory monitoring, the results of which highlighted the importance of 24-hour control of blood pressure for both the Black and White subjects.

The results of the Brent study showed age standardised medial systolic blood pressure by ethnic groups to be 7 mmHg higher in African-Caribbean men than in white men, (129 mmHg and 122 mmHg respectively), and 17 mmHg higher is African-Caribbean women than in white women, (135 mmHg and 118 mmHg respectively). Black subjects also tended to have higher circadian (day and night), blood pressure, with the rise being lowest at the peak morning period. The researchers interpreted this as being significant, noting that most of the previous studies on this group were carried out in the mornings, and could explain the low significant differences in blood pressure between the Black and White groups of these former studies. Unlike the Cruickshank findings in the Birmingham study, in the blood pressures of those in the Brent study were higher in African-Caribbeans at all levels of body mass index. Like Haines (1987), earlier Chaturvedi found that African-Caribbeans with hypertension were also more likely to be taking medication for the control of hypertension than Whites were.

The Chaturvedi team maintained that their results not only supported the findings of previous studies that hypertension was more prevalent in African-Caribbeans, but in addition showed that the resting blood pressure variances between African-Caribbeans and Whites were larger than had been demonstrated in previous studies. Further, Chaturvedi and colleagues maintained that: ***"For the first time, gender***

differences in resting blood pressure in African-Caribbeans commensurated with mortality data" (p.94). The researchers further opined that such confirmation offered strong arguments to support the view that there was a continued rise in the incidence of hypertension in African-Caribbeans. They concluded that in women, (but not in men), these differences could be large enough to explain the excess stroke mortality in African-Caribbeans. Reasons for the ethnic differences that were observed were not established.

4.3e Other African-Caribbean Studies in the 1990s

THE WEST LAMBETH STUDY: 1993

Regan and Parry-Cooke (1993) examined causes and risks of coronary heart disease in 146 African-Caribbeans. A small proportion of the respondents, 10 men and 13 women, had raised blood pressure, and almost all suffers were currently taking medication.

THE HACKNEY HYPERTENSION REDUCTION PROJECT, (HHRP): 1993

The HHRP (Trenchard-Mabere1992) involved 602 subject, with 262 African-Caribbeans. The survey results showed that raised blood pressure was most prevalent amongst African-Caribbeans, at approximately double the risks of the other ethnic groups, including Black Africans.

4.3f Current Ongoing Studies

The Manchester Study

The Epidemiology Unit at the University of Manchester, is part of a large longitudinal international study of high blood pressure and diabetes in Black populations. This study is comparing people in the Cameroon and Nigeria in West Africa, Barbados and Jamaica in the Caribbean, Black Americans in Chicago, USA, and the Manchester Black population. Dr. K. Cruickshank leads the Manchester project (Cruickshank 1996). The aim of this international project is to examine blood pressure and related factors in a precisely standardised manner across these geographical sites. Among other things, the project will follow the subjects over several years to initiate a controlled trial to delay the onset of hypertension by dietary lifestyle interventions. To date, the Manchester project reports a greater prevalence of hypertension in African-Caribbeans (Cruickshank 1996).

The St. George's Hospital Study

At the Blood Pressure Unit in **St George's Hospital** south-west London, there is a longitudinal controlled study, looking at the effect of salt alteration on blood pressure in ethnic groups. Professor G. MacGregor leads the research.

Summary of Hypertension in Caribbeans

Research results suggests that the blood pressure of African-Caribbeans, both native and British, lies between the low rates in rural sub-Sahara African populations and the high rates of urban African Americans. Generally, blood pressure among people of African ethnic populations is lowest among inhabitants of sub-Sahara Africa, higher in the people living in the Caribbean islands, higher still in those African-Caribbeans who settled in Britain, and highest in African-Americans. It appears that

as Africans and people of African descent adopt and integrate into western cultures their propensity to hypertension increases.

4.4 BLOOD PRESSURE AND LIFESTYLE

4.4a Overview of Lifestyle Factors in Hypertension

The known risks of (primary) hypertension are well documented to include environment considerations, which may broadly be interpreted as lifestyle related factors. The significance of lifestyle to age-related increases in blood pressure, and the importance of health behaviour modifications in non-pharmacologic interventions have been internationally ratified, (JNC V 1993; WHO 1993). Among the several life-style factors listed by the JNV.C (1993), as important contributors to age-related increases in blood were a high sodium intake, an excessive consumption of calories, physical inactivity, excessive alcohol consumption, and a low intake of potassium. The World Health Organisation (WHO 1993) guidelines for the management of mild hypertension, stated that before labelling a subject hypertensive:

> ***"All patients should be given advice to modify their lifestyle, as appropriate, by stopping smoking, reducing obesity, limiting alcohol and dietary saturated fat, and engaging in regular mild dynamic exercise...salt restriction may assist in lowering blood pressure"*** (p.394).

The findings of British government surveys (OPCS, 1992,1993), have largely concurred with these and other global reports. In 1993 the OPCS reported that blood pressure was positively associated, in varying degrees in the adult population, with alcohol intake, cigarette smoking, dietary habits, (fat intake), body mass index,

(obesity), and perceived social support. In the earlier *Health of the Nation* (1992) report it was stated that ***"Raised blood pressure is associated with obesity and overweight, excessive alcohol consumption, high sodium (principally from salt) and low potassium intakes"*** (p.57). The *Health of the Nation* further stated that ***"The environments in which people live and work can have both favourable and adverse effects on their health and well being"*** (p.12). Its targets included working towards the creation of healthy surroundings and a healthy lifestyle, ***"By the active promotion of physical environments conducive to health... by increasing knowledge and understanding about how the way people live affects their health"*** (pp. 12 & 13).

A brief summary of the lifestyle factors in this study: salt intake, general diet, physical activity level, alcohol consumption, cigarette smoking; coffee consumption, social class, stress level, and perceived social support, is given below.

4.4b Dietary Salt

A relationship between salt intake and blood pressure was recognised at least four thousand years ago in the Chinese Yellow Emperor's classic on internal medicine - 2698-2598 BC, (in Ruskin 1956, pp X-XII). In that Emperor's Classic was written: ***"If too much salt is used in food the pulse hardens"***. Since that time a plethora of studies have examined the association between sodium intake and (high) Blood pressure in populations. From the huge volume of studies have been derived mixed and often inconclusive results, (Law et al 1991). However, a number of trials on sodium reduction, including those reported by Elliot (1989); and Cutler et al (1991),

have shown the significant effect that such reduction could have in reducing elevated blood pressure.

The *Health of the Nation* acknowledged the possible blood pressure elevating effect of excessive sodium intake, and recommended that programmes for salt reduction in the daily diet should be instituted. The Ministry of Agriculture, Fisheries and Foods, (MAFF 1994) ratified this when it acknowledged the positive association between salt intake and blood pressure. It concluded that the association of sodium with elevated diastolic and systolic blood pressures might be apparent at intakes of 3,200-4,700 mg/d. The Ministry reported that the average daily intake of sodium was 2,858 mg, with the intake being greater in men, and set the *Recommended Reference Nutritional Intake*, (RNA), at 1600 milligrams per day (mg/d), with the *Lower Reference Nutritional Intake*, (LRNA), being 575 mg/d. It was also revealed that a high proportion of the sodium intake was from *'hidden'* salt in such forms as cereals, bakery products, snacks, and beverages.

4.4c *INTERSALT*

Intersalt (1988) was arguably the most intensive and extensive investigation into the significance of the sodium/potassium blood pressure relationship. The survey involved over ten thousand, (10,079), persons, 52 populations, across 32 nations worldwide. Investigations concentrated on the relations of urinary electrolyte and other factors with blood pressure. Only four low salt populations were observed, two in Brazil, one, in New Papua New Guinea and one, in Kenya. Highest salt intake among the four low salt populations was found in the Kenyan population, where the median intake was less than 3 grams per day (g/day) and the median level

of the other 48 higher salt intake *Intersalt* populations was 9g/d (Carvalho et al 1989). Residents of the low salt areas also had very low blood pressure, and hypertension in these communities was rare or non-existent.

Carvalho et al (1989) detailed the differences in salt intake and blood pressure within the low salt consumption communities. The Kenyans were reported to have both the greatest salt intake of the four low salt groups, and greater sodium-to-potassium ratio than the other three communities. Paralleled with these differences, the Kenya sample generally showed both higher systolic and diastolic blood pressures, as well as a wider variation of distribution. There was five-percent hypertension in the Kenya sample, compared to zero-to-one percent, in the other three, low salt populations.

These *Intersalt* results confirmed earlier findings by Page et al (1974), and others, that within populations with low salt intake hypertension is virtually non-existent, and that the greater the salt intake the greater the prevalence and incidence of hypertension. *Intersalt's* overall conclusion was that there was an age related positive linear association between sodium intake and blood pressure in 48 of the populations studied which had not been detected in the four low salt communities. This observation by Intersalt was consistent with the theory that a certain minimum amount of salt is required for age-related rise in blood pressure in adults (Law et al 1991). Some researchers in the area have concluded that the relationship between sodium intake and blood pressure was significantly stronger than that indicated by the *Intersalt* results, (Elliott et al 1988; Stamler et al 1991; Law et al 1991). The Law team also concluded that although blood pressure was higher on average in the

more developed communities, the association with sodium intake was similar to that of the less developed populations.

Sodium reduction continues to be deemed an important intervention factor in blood pressure national management programmes, including the *Health of the Nation.*

4.4d General Diet - Dietary fats

The importance of a well balanced diet, especially the low intake of dietary fats, in the control of hypertension is acknowledged globally, (WHO 1993; JNC V 1993). In The *Health of the Nation* (1992), it was reported that total cholesterol was significantly associated with both systolic and diastolic blood pressures. A target was set to ***"To reduce the average percentage of food energy derived by the population from total fat by at least 12% by 2005, from about 40% in 1990 to no more than 35%"*** (p.52). To lower the risk of stroke, (and blood pressure), ***"a diet relatively low in fat and rich in vegetables and fruit"*** (p.64) was recommended. The importance of dietary fats in hypertension control was reiterated in the later OPCS report (1993) where it was stated that persons with the higher cholesterol levels tended to have higher mean blood pressure.

4.4e BMI - Obesity

Body mass index, (weight in kilograms divided by height in metres squared, that is, kg/m^2), has been found over many decades to be independently significantly related to blood pressure, (Stamler 1991). Of the five variables examined in the *Intersalt* study body mass index was found to be the most consistently related to blood pressure (Dyer and Elliott 1989). The OPCS (1993) defined overweight as body

mass index greater than 25 kg/m^2 and obesity as body mass index of greater than 30 kg/m^2. Using this definition, the report stated that obesity was significantly associated with an increased prevalence raised blood pressure. According to the OPCS (1993) report, age-standardised ratios showed the obese more likely to have high blood pressure than would be expected from their age-distribution alone. The underweight, (body mass index of 20 kg/m^2 or less), and those of 'desirable' weight (body mass index 21-25), were less likely to have high blood pressure after controlling for age. This is a reiteration of the earlier *Health of the Nation* (1992), report which stated that ***"The eating habits of the population play a significant part in the development and prevention of stroke, as blood pressure is affected by both obesity and sodium intake"*** (p.52).

4.4f Physical Activity

Two major longitudinal surveys: Paffenbarger et al (1983), and Blair et al (1984), identified the effects of regular physical activity on blood pressure. Paffenbarger followed 15,500 Harvard Alumni students for approximately ten years, while Blair assessed the physical fitness of 6,039, normotensive men and women and recorded their blood pressure variations for an average of four years. Both studies concluded that the sedentary and less active subjects had a significantly greater risk of developing hypertension. This risk was independent of factors such as age, gender, baseline blood pressure and body mass index. The UK (national) *Allied Dunbar National Fitness Survey* (1992), reported that the overall, the fitness levels of the population in England were below the acceptable level for the achievement and maintenance of good health. The OPCS (1993), survey found that physical activity

was independently (inversely), associated with the systolic blood pressure, but in men only.

4.4g Alcohol

The relationship between blood pressure and alcohol consumption has been comprehensively researched with a wide range of samples, including one with over 80,000 subjects, (MacMahon 1984). The relationship has also been consistently established, (World Hypertension League 1991). In the *Intersalt* survey, alcohol consumption level was one of the few lifestyle-related factors to show a strong positive relationship with blood pressure. This relationship has also been found independent of age, gender, ethnicity, and socio-economic factors, (Intersalt 1988; JNC V 1993). Examination of a number of cross-sectional studies enabled Friedman et al (1982) to estimate that in approximately five-percent for females and double that for males, the overall prevalence of hypertension could be attributed to alcohol intake that exceeded two units per day. Friedman and colleagues deduced that the highest blood pressure levels were evident when alcohol consumption exceeded 5/6 units per day. The OPCS (1993) reported that alcohol consumption was independently associated with diastolic blood pressure for both men and women, but with systolic blood pressure for men only. A weekly intake of less than 21, units for men, and 14 units for women, was recommended by the *Health of the Nation* (1992), which stated that, ***"Sustained drinking in excess of these levels progressively increases the risk of raised blood pressure and stroke"*** (p.58)

4.4h Cigarette Smoking

The negative effect of cigarette smoking (including passive smoking), on the cardiovascular system is no longer disputed in the medical community, as evidenced by the ban on smoking in most public places. Despite this, a number of epidemiological studies have revealed that smokers do not necessarily have higher blood pressure than non-smokers (JNC V 1993). Within the UK, conflicting findings have been reported at the most accredited levels. The *Health of the Nation*, reported that "***Smoking is estimated to account for up to 11% of stroke deaths***" (p.51). A year later the OPCS (1993), concluded were that there did not appear to be a clear or direct association between cigarette smoking status and raised blood pressure, describing the relationship as *"complex"*. According to the *OPCS*, the age-standardised ratios suggested that non-smokers were no more likely to have high blood pressure than expected from their age distribution, while all current smokers appeared to be significantly less likely to suffer high blood pressure than would be expected on the basis of their age. However, men who had never regularly smoked were less likely to have high blood pressure. The issue remains a complex one.

4.4i Coffee

Investigations into the adverse effect of coffee or caffeine on blood pressure have been recorded since the turn of the century (Pierce 1909). Subsequently, there have been many studies, but overall, randomised trials of coffee consumption have not revealed consistent increase in resting blood pressure as a result of coffee intake, (Ammon et al 1983; Stensvold et al 1989). However, ambulatory blood pressure measurements have detected daylong blood pressure effects that were not apparent on resting blood pressure, (Myers et al 1987). It has been found that tolerance to

caffeine develops rapidly, with adaptation occurring in just a few days, (Robertson et al 1984; Myers et al 1987).

4.4j Stress - Psycho-social

Long-term stress may play an important role in the development and maintenance of hypertension (Pieper et al 1989; Schnall et al 1990). Johnson and Hall (1988) reported that job related stress resulting from high demands and employees perceived ability to cope was associated with elevated blood pressure. Workers who had been exposed to stressful situations, but lacked the decision-making ability to control their exposure and response to environmental stress, were most likely to be at special risk. The workers' perception of social support was also associated with blood pressure. Generally, people in the lower income brackets were likely to be exposed to higher levels of stress than their better off counter-parts. James (1987) concluded that the less socio-economically well off, and least empowered members of society had greater difficulty coping, making them more prone to stress related hypertension. The OPCS (1993) reported that, among men, there was a weak inverse relationship between blood pressure levels and perceived social support, but this association was not detected in women.

4.4k Socio-economic Situation

The inverse association between social class and mortality from circulatory disease, documented in the *Black Report* (1980), and similar papers, is generally not disputed. Marmot and Shipley et al (1984) reporting on the Whitehall Study of civil servants, found that there was an inverse relation between job grade and mortality. Wilson et al (1991) stated that in the USA the higher systolic blood pressure among

rural Blacks also indicated an environmental aetiology, perhaps related to education and socio-economic status.

SUMMARY OF BLOOD PRESSURE AND LIFESTYLES

Independent associations have been established between blood pressure and the lifestyle factors examined in this project. Further, virtually all the variables are inter-related, contributing to the multi-factorial nature of hypertension, (JNC V 1993). Some lifestyle factors, however, appear to have stronger independent associations with blood pressure than others, in particular sodium and body mass index. No single lifestyle factor alone can account for a significant variation in blood pressure in any one individual or even any one population.

4.5 CARIBBEAN LIFESTYLE AND BLOOD PRESSURE

4.5a Overview of Caribbean Lifestyle and Blood Pressure

Information on the lifestyle of African-Caribbeans is scarce, and even less is known about the blood pressure raising effect (if any) of their lifestyle. The area is generally unresearched. Current researchers including Cruickshank et al (1985) Beevers and Beevers (1992), and Chaturvedi (1993), have conducted comparative studies on risk factors of vascular diseases in different ethnic groups, with some attention paid to the relationship between lifestyle factors and hypertension in African-Caribbeans. However, lifestyle aetiological factors have not been priority areas in most of the relatively few studies on blood pressure in this ethnic group. Chaturvedi et al study was one of the most recent to investigate the relationship,

which included ambulant blood pressure monitoring of African-Caribbeans. The team concluded that the prevalence of smoking, heavy drinking and low levels of exercise were no higher in African-Caribbeans than in Whites. An outline of the general findings with regard to African-Caribbeans on the main lifestyle factors in this project follows.

4.5b African-Caribbeans General Diet

There are very few published data on the dietary habits and nutrient intake of African-Caribbeans. Information on the African-Caribbean dietary practices has largely been anecdotal, rather than resulting from structured research. Since the 1990s, the interest has grown, but studies have been small scale, and generalisations have proven difficult because, as Douglas (1989) pointed out, there is wide variation of eating habits among the diverse African-Caribbean population. Douglas also noted that there had been varying degrees of adaptation to the British dietary culture, which compounded the problem of standardising the nutrient intake of African-Caribbeans. She felt it reasonable to assume that African-Caribbeans had modified their dietary habits, adapting to the conditions and lifestyle of their adopted country. Douglas reported that in general the younger, mainly British-born African-Caribbeans had maintained only a small percentage of their parents, and grandparents' dietary preferences. A generalisation about the regular dietary habits of African-Caribbeans was difficult.

Wide variations notwithstanding, in general older African-Caribbeans[9] have tended to eat traditional foods at home, combined with British foods outside the home,

[9]The migrants as opposed to the UK-born African-Caribbeans.

mainly at work. A survey of a sample of 40 African-Caribbeans two-day food diary, by Cade and Sharma (1994) concluded that traditional Caribbean meals were eaten daily by a large percentage of the families. Cade and Sharma also reported that the group's fat intake as a percentage of energy was 38%, which was lower than the 40%, national average reported by the *Health of the Nation.* The traditional African-Caribbean diet is reportedly balanced, with staples, meat, fish and vegetables, cooked in a variety of styles, including a lot of frying, with possible dietary restrictions for religious reasons, such as abstinence from pork or alcohol, (Douglas 1989).

4.5c Caribbeans and Salt

The relationship between sodium and blood pressure in people of African origin has arguably been the topic of the bulk of the detailed investigations in the ethnic blood pressure problem in the USA, the Caribbean and Britain. None-the-less, the pressor effect of sodium in African-Caribbean is largely unresearched, and therefore, no firm conclusions have been derived. The subject is still wide open. Beevers and Beevers (1992) concluded that it was highly probable that there were no major differences between African-Caribbeans and the indigenous (White) population at the same levels of sodium intake. Interest in the subject, as it relates to African-Caribbeans appears to be growing, as demonstrated in longitudinal trials of the type being undertaken by the St George's Hospital, (London) group.

For the purposes of this study, it will be useful to outline the general findings on salt in Blacks, that is, people of African origin. Studies by researchers such as Luft et al (1979) have largely sought to establish whether information on levels of sodium

intake could offer aetiological explanations for the observed racial differences in blood pressure between Blacks and Whites. Luft found the blood pressure of African-Americans to be highly sensitive to increases of dietary salt, showing a greater tendency of retaining sodium than their white counterparts. It has also been demonstrated that Black hypertensives were more likely to normalise their blood pressures with salt-excreting diuretics than Whites, (Freis et al 1988). The findings of both Luft and Freis, and that of other similar findings, have been challenged by a number of researchers, including Madhavean and Alderman (1994) who studied a homogeneous biracial cohort of New York workers (in the USA). From this group, Madhavean and Alderman concluded that Blacks at the same sodium intake did not have higher blood pressures than Caucasians. They observed no significant ethnic differences for either systolic or diastolic blood pressure after adjusting for age and body mass index, and concluded that any apparent differences were not independently attributable to ethnicity. Although there have been a wealth of research in the area, the significance of sodium (salt intake) to the blood pressure trends in of people of African descent largely remains an enigma

4.5d Caribbeans and Body Mass Index

Studies involving UK African-Caribbeans have in general found that African-Caribbean women have higher body mass indexes than their White counterparts, (Cruickshank et al 1985; Chaturvedi et al 1993; Cade and Sharma 1994). In the Birmingham Factory Screening Project (Cruickshank et al 1985), African-Caribbean women were found to have greater mean systolic and diastolic blood pressures. However, the African-Caribbean women over 35 years of age were more obese by an average of 4 kg/m^2, (2 .5 to 5 kg/m^2). The differences in blood pressures between

Caribbean and White women paralleled the differences in body mass index. A comparative study to the Birmingham survey, carried out in the Caribbean Island of Jamaica, between 1979-80, (Cruickshank et al 1985), revealed lower levels systolic blood pressure in the native Jamaican men maintained until the over age 45 years. This was closely associated with lower body mass indices and with the much greater exercise taken routinely by the Jamaican men. After reviewing both the Birmingham and the Jamaican findings, Cruickshank that, the variability in females could be explained by the greater body mass index in African-Caribbean women, especially those over age 35 years.

The Birmingham findings were only partially supported in a later study by that of the Brent study (Chaturvedi et al 1993). Chaturvedi reported a mean difference of 3 kg/m^2 between African-Caribbean and European women, but no significant difference in within in the male groups. Blood pressure was higher in African-Caribbeans at all levels of body mass index in women, but body mass index only partially explained the differences. In summary, blood pressure variations in African-Caribbean women may be explained by levels of obesity (body mass index), but it is an area which needs much investigating.

4.5e Caribbeans and Alcohol and Smoking

There have been very few studies on the cigarette smoking and alcohol habits of African-Caribbeans, and results have been widely varied, especially with regards to men. The *Health of the Nation* (1992) stated that ***"Smoking prevalence is lower than the white population in people of African origin-Caribbean and Asian origin"*** (p.120). A low prevalence of heavy smoking and drinking has been found

in African-Caribbean men and women, (Jackson et al 1981; Balarajan and Yuen 1983), but Miller and Kotecha et al (1988) reported higher levels of alcohol consumption and smoking in African-Caribbean men. In the Brent study (Chaturvedi et al 1993), both African-Caribbean men and women smoked less, and consumed less alcohol than the Europeans. A survey by Cade and Sharma (1994), found that the African-Caribbean men had higher levels of alcohol intake, (10%), compared to the 7% national average. However, African-Caribbean women had lower intakes, (1%), compared to the 2.8% national average for females generally. There has been no evidence that alcohol consumption has a greater or lesser effect on African-Caribbeans blood pressure than other populations, but Cochrane and Bal (1989) reported that alcohol-related morbidity appeared to be rising in African-Caribbean men.

SUMMARY OF CARIBBEAN LIFESTYLE

The lifestyle of UK African-Caribbeans is relatively unresearched. Very little is known about the African-Caribbean diet and the degree and type of adaptation to the dietary practices of their adopted home as the group becomes more integrated and ingrained in the British culture. The average sodium intake of African-Caribbeans is not known. Findings on high-risk blood pressure factors such as alcohol consumption and cigarette smoking, and sodium intake have produced mixed results and are inconclusive. Levels of obesity (body mass index) may explain variations in blood pressure in African-Caribbean women, but it is an area in need of much investigating. African-Caribbeans are not homogeneous, and there are wide variations in health behaviour as it relates to hypertension risk.

SUMMARY OF CHAPTER FOUR

Chapter four gave a review of the literature regarding this research project. The UK African-Caribbean blood pressure status was also shown in the wider perspective as it relates to other Black people of African origin. Similarities in blood pressure patterns were evident across western Black populations, especially between African-Americans and UK African-Caribbeans. However, African-Caribbeans did not show the high levels of hypertension found in the very high-risk group of African-Americans.

The reviewed documents suggest that excessive hypertension in African-Caribbeans existed shortly after mass migration began in the 1950s, and that it is currently deemed by the British health authorities to be of urgent concern. A major reason for the urgency is the high stroke morbidity and mortality resulting from hypertension in that ethnic group. The problem was exacerbated by indications that the current situation will persist beyond the 1990s. A review of the literature revealed that the subject of hypertension in African-Caribbeans is relatively unresearched, and that most studies relating to the group were comparative. There was particularly a sparsity of researched information on the association between blood pressure and the lifestyle practices of this ethnic group. Existing information was sparse and generally conflicting, highlighting the need for more research. In general, despite a mountain of research in the USA, the pathophysiology of hypertension in western peoples of African descent is still very inconclusive. The aetiology of the disease in western Blacks remains an enigma, and is somewhat bemusing in the African-Caribbean population in the UK.

5 Fifth Chapter

The Survey Environment

5.1 SUBJECTS

5.1a Eligibility

Subjects were London-based African-Caribbean volunteers, employed in a government agency. The definition for the term 'African-Caribbean' used was "**A person of African origin (racially mixed or unmixed), born in the Caribbean or had at least one parent of African origin who was Caribbean born Caribbean".** Currently treated hypertensives were excluded, defined to mean that no respondent should be currently taking medication to lower blood pressure.

5.1b Social Services Lambeth

Subjects were employees of the Directorate of Social Services, Lambeth. This government agency was selected as the borough of Lambeth had among the highest proportion of African-Caribbeans in the UK, (census 1991). Further, the Director of the Equalities Service Development Division of Lambeth Social Services confirmed that:

- Lambeth Social Services had among the largest workforce of African-Caribbean in London, (see appendix D).
- The Caribbean employees of the Directorate were diverse, with representatives from all five social classes, (see appendix E).
- The African-Caribbeans employed by Lambeth Social Services could be considered representative of the Caribbean community in London with regards to ethnicity, general lifestyle and life experiences.
- The Lambeth Social Services encouraged research of this nature as part of its health initiative programme, evidenced by its support of this project.

Following initial face to face approach and proposal of the project to the head of the Equality and Race Division of the agency, the thesis proposal was submitted to the Directorate (head) of Lambeth Social services, (see appendix F). Approval for the survey to be conducted on the African-Caribbean employees of that institution was granted, (see appendix G). It was understood that no staff member was made to feel obliged or pressured to participate. Once information about the project was filtered to the staff, decision to participate was to be totally voluntary, and at participants' initiative. Information about the project was given to department heads, who were requested to disseminate the information to their African-Caribbean staff, (see appendix H). There was also a briefing session to inform managers in detail about the programme, (see appendix I). The researcher of this project had agreed to organise and run a series of seminars relating to blood pressure during the 18-month data collection, and the managers enthusiastically encouraged this, (see appendix J).

5.1c Initial Contact with Subjects

Direct contact between the researcher and prospective subjects was made, initially, through the organisation's internal communication system. Subjects completed a response slip, to confirm their willingness to participate in the research, (see appendix K). There were also outreach programmes which included chaperoned visits by the researcher to some (outside) Social Services sites, (see appendix L).. This was necessary, as some departments within the organisation were located throughout the borough. Subjects were also recruited by word of mouth, as some of the early volunteers encouraged their colleagues to participate. The highest volume

of the response came in the first month, through the response slips, along with telephone enquiries. Every employee from every department had the opportunity to participate.

5.1d Total Number of Subjects

There were 164 eligible volunteers, comprising 138 females and 26 males. The average (mean) age of the subjects was 38 years, the youngest subject was aged 20 years, while the eldest was 61 years, a range of 41 years, (see appendix M). Both the age and gender proportions were reasonably representative of the workforce, which was largely in the thirties age range and more than 80% female. Volunteers came from all sectors within the Directorate. The majority of subjects who volunteered for the programme stated that they did so primarily because they wanted to contribute towards the study, and improve their general health awareness. Curiosity as to what the study entailed and the use to which it would be put was also given as a reason.

5.1e Non-Participants

Feed-back primarily from other participants, suggested that:

- Some people were sceptical of the confidentiality aspect, they were afraid that their employer might have access to the information given.
- Some felt they did not have the time, or they could not or would not make the time, despite the fact that their employer allowed them the time to participate.
- Some offered no specific reason, but just did not want to participate. It was speculated that many were unsure as to whether the survey would benefit African-Caribbeans.

Females responded significantly more enthusiastically than the males. Most of the 26 male participants responded only after encouragement from a registered participant, (colleague), or after chance conversations with the researcher.

5.2 DATA COLLECTION: PREPARATION AND PROCEDURES

5.2a Social Services Supervision

The researcher received primary assistance from the Directorate through the Equality and Race Division, in making contact with potential participants. The participants' first line of communication with the researcher was through the Directorate's internal mail or telephone. When an eligible member of staff indicated interest to the researcher, an appointment was made for the individual to attend an interview with her. The researcher made all appointments (directly). If the prospective subject required greater detailed explanation on the telephone before making an appointment, the researcher gave this.

5.2b Confidentiality

The Directorate was offered no information concerning the participant and researcher interaction. No employee of the Directorate had access to any information concerning the participant that was collected by the researcher. All aspects of a volunteer's relationship with the researcher adhered to strict codes of confidentiality. No information pertaining to any participant was left or stored at the Directorate, not even overnight. No information pertaining to any participant was documented on any of the Directorate's computers. At the end of each working day,

the researcher took all material concerning the participants' home with her. Information concerning any particular participant was only taken to the office if there was a scheduled appointment for that participant on that day. If the participant failed to keep an appointment the documents (questionnaires and progress reports), were returned to the researcher's home, and re-filed there. The name of a participant was not required or recorded on the questionnaire. A number, which was known only to the participant and the researcher, identified each participant. Subjects' personal details, that is, name and telephone numbers, and identification number were recorded on a separate form, (see Appendix N).

5.2c Interview Venue(s) - room

The researcher was allocated the first-aid rest room, situated on the first floor of Mary Seacole House, the head office of the Directorate of Social Services, Lambeth. The room was available to the researcher Monday to Fridays, from 08.00 to 18.00 for the duration of period needed to complete interviews and monitoring, which was approximately 18 months, from May 1994 to August 1995. The room had one window, which was never totally closed, except if there was heavy rain. On cold days, the researcher ensured that there was at least a crack, to allow fresh air to enter. Apart from the central heating, there was a slim line heater, so the room was adequately heated, and a fan was provided during the two (hot) summers. The room, which offered adequate privacy, was equipped with a single bed, desk, two chairs, direct line telephone and stationary. A key was given to the researcher, so that she could lock the door if she had to leave the room. At the end of her working day, the researcher locked away the blood pressure measuring equipment to prevent tampering from the cleaners. When the researcher left the building for long periods

of time, and at the end of her working day, the key to the room was placed in the Office Managers quarters.

5.2d Interview Conditions

Interviews were conducted with only the subject and the researcher in the room. On three occasions participants asked to have a second person present. Once was a male subject's female friend, another was the subject's daughter, and the other was female subject's female friend. Some interviews were conducted at the researcher's home as there was a division of the Directorate less than 100 metres from her habitat, thus it was convenient for employees of this division to be interviewed there. Also, there were a few subjects who resided in the same area as the researcher and found it convenient to be interviewed there. These interviews were conducted in the living room of a two-bedroom flat, in complete privacy. The researcher's couch was used for the recumbent blood pressure and heart rate measurements. In two instances, interviews were conducted at the subject's homes. Conditions were similar to those of the researcher's home environment. All interviews were pre-arranged, usually by telephone. Prior to the subjects arriving for their interviews, the researcher pre-prepared the room as follows:

- Bed was made tidy, and a clean disposable sheet slip was laid on the pillow.
- Measuring equipment were checked and made ready
- Forms were made ready.

5.3 DATA COLLECTION: INTERVIEWS AND MEASUREMENTS

5.3a First Interview

At the first interview the researcher introduced herself and invited the subject to be seated. She[10] was given an introductory letter to read and asked if she understood what the research involved, (see appendix O). Additional information was given on request. The subject was reassured of the confidentiality standards. The independence of the research from her employer (Lambeth Social Services), was reiterated whenever this reinforcement was needed. The repeated measurement nature of the research was explained to subjects, and most said they had no problem with that, work and personal circumstances allowing.

Having confirmed a desire to participate in the project, personal communication details were taken as follows: Full name, communication address at work, and telephone numbers. Subjects who did not wish to give their home telephone numbers were not required to do so. However, the vast majority of subjects willingly did so after the researcher explained the possible need to contact them at home if for any reason they could not be reached at work. These personal data were not entered on the questionnaire, but kept by the researcher (at her home). The participant was then given an identification (ID) number, which was allocated according to stage of joining the programme. Thus the first subject to be interviewed was allocated the number 001; the second subject 002 and so on. The questionnaire was dated, and the venue (where the interview took place) was noted. Gender and age were recorded. Height was measured to the nearest 0.5 inch using a

[10] Since the vast majority of subjects were women, the female gender will be used to refer to individual subjects

vertical ruler placed on the wall (a carpenter's ruler was used for the away subjects). The height was later converted to metres. Weight was recorded to the nearest 0.5 kilograms on a Hanson portable (accurately calibrated), scale. At this point, the subject was invited to 'use the bathroom' if needed before commencement of blood pressure measurements.

Before commencing with the questionnaire and measurements of blood pressure and heart rate, the researcher tried to ensure that the subject felt comfortable and at ease. There were no notable cases of discomfort from any of the 164 subjects. However, if the researcher detected any indication of apprehension she did not proceed until the subject was 'ready'. There were also a number of instances in which individual subjects seemed 'rushed', and they usually explained that they had hurried to get to the interview, or had been rushing around on the job just before coming. In such instances, the subject was given a few minutes extra time to 'relax'.

5.3b Blood Pressure/Heart Rate Measuring

A trained and experienced observer, the researcher, took measurements. Blood pressure and heart rate were measured on the dominant arm, in accordance with the guidelines from the World Health Organisation, (WHO 1993), and the guidelines of Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC V 1993). Guidelines for accurate blood pressure measurements were also referenced from Petrie et al (1986) and Stolt et al (1993). The subject's dominant arm was recorded on the questionnaire. This first of three seated blood pressure and heart rate measurements were taken approximately 4-6 minutes after the subject had been seated on a comfortable chair, (with back support). Resting

heart rate was measured via the radial pulse for 15 seconds. Blood pressure was measured using either an Accoson mercury sphygmomanometer, and/or an Accoson aneroid sphygmomanometer. The mercury machine had been calibrated prior to the commencement of the project at a reputable medical supplies shop in London. The aneroid machine was newly purchased from the same outlet. A standard adult Accoson cuff was used for the majority of participants, and a larger one used for individuals whose upper arm circumference were too large for the standard cuff, (Stolt et al 1993). The cuffs were newly purchased.

The subject's dominant arm was placed on the desk (or equivalent for home visits), with a folded bed-sheet under the elbow for comfort. Some subjects had the 'natural' tendency to tense the arm muscles, and this was corrected. With the arm relaxed the cuff was wrapped around the upper arm encircling the biceps/triceps, with the lower end of the cuff placed approximately one-inch above the elbow. On the first reading, the minimum-maximum inflation levels were determined before actual measurement of blood pressure. This was done either by feeling for the pulse on the brachial artery in the area of the elbow joint during initial inflation, or listening through the stethoscope, in cases where there was a weak brachial pulse. Inflation was taken to approximately 30 mmHg above the anticipated systolic blood pressure, which was recorded as the appearance of Korotkoff sound (phase I). The pressure was deflated at approximately 2-3 mmHg per second and diastolic pressure was recorded as the disappearance of sound (phase V). Where sounds continued to zero or near zero mmHg, Korotkoff phase V was used. Such readings were recorded with a minus sign to indicate no fifth phase, for example, -40. However, the minus

was not included in analysis, thus -40 mmHg was for purposes of analysis treated as 40 mmHg.

On a few occasions measurements were taken over thin clothing because female subjects wore garments that had to be completely removed in order to expose the arm. If the individual was not comfortable with removing such clothing, she was not required to do so. On such occasions, the interviewer took special care to ensure that the pulse was clearly audible. Such measurements were taken using two different sphygmomanometers for verification. If the researcher detected a significant difference between the two readings, the measurement was taken a third time and the average was used as the recorded value. However, instances needing a third reading (verification) were rare.

5.3c Questionnaire Completion

After recording the first blood pressure and heart rate readings, the researcher began the completion of the questionnaire, which was placed on the desk so that the questions were visible to the subject. Each question was asked in the order in which it appeared on the form. If a definite answer was given to a question that coincided with an option on the form, that answer was circled. When individuals seemed unsure of either the question, or how to respond, they were asked to look more closely at the options and to indicate which of them best represented the most accurate answer. Subjects usually responded to this 'show-card' method quite definitely. However, there were instances in which subjects thought their preferences were not adequately represented by one of the options. The questions on racism proved to be the most difficult for subjects to answer definitively, (see

section 11 on questionnaire, appendix B). Respondents who preferred to give explanatory answers, make comments, or give views, were encouraged to do so. The researcher considered it important to give such latitude as this could assist the subject in determining the most appropriate answer to circle from the options on the questionnaire. Further, the researcher herself would learn much from it, which would enhance the research. Such expansions to answers enabled her to modify the phrasing of some questions and answers, in the very early stage (first week) of the research.

After about five to eight minutes into the questionnaire, the second seated blood pressure and heart rate measurements were taken. The researcher then resumed completing the questionnaire. The third seated blood pressure and heart rate readings were taken at approximately the same time interval. On completion of the three seated readings, the subject was asked to lie down, and completion of the questionnaire continued. At this point the questionnaire was usually about two-thirds completed. Recumbent (supine position) blood pressure and heart rate readings were taken in the same sequence as the seated ones.

Time intervals for the blood pressure and heart rate readings were pre-approximated at about five minutes. However, this was not rigidly adhered to, although the time intervals were never less than five minutes in apart. Thus, the time between say the first and second readings may have been different to the time between the second and third readings. The main reason for the time variations was the subjects varied approaches to answering questions. Some subjects answered quite concisely, while others chose to be more detailed about some things. Participants generally seemed

appreciative of the opportunity to relate their particular situations or views. For example, very often elaborate answers were given to the questions on stress at work, and supportiveness (usually lack of it) of partners. It was generally not expedient to stop the subject in 'mid-air' in order to take a measurement. The interviewer welcomed the variety of responses, as it also helped her to detect inconsistencies and allow the subject to clarify them. Further, it offered her an insight into the personality of participant. This was important because of the repeated measures design, where the researcher could have several meetings with that subject over a ten to twelve months period. It was, therefore, important to use such opportunities to build relationships with individual participants. In some cases, therefore, subjects interview times went beyond the standard 55 minutes allocated, which was acceptable to the researcher if subsequent appointments allowed. In principle, if the subject demonstrated that level of confidence in the researcher, she did her best to accommodate it. The average interview time was however, 50 minutes. Blood pressure and heart rate measurements were taken over a period of about 35-45 minutes.

5.3d Procedure at the End of First Interview Session

Participants were given a copy of their blood pressure and heart rate readings, as well as information literature on blood pressure and related subjects. They were also instructed that (if they agreed), they would be required to have their second readings in approximately seven to eight weeks. If possible an appointment was made for this upcoming visit, otherwise participants were contacted about ten days before an appointment was due. Reminders were given about the series of workshops to be held as part of their blood pressure education programme.

5.3e Subsequent Interviews and Measurements

Subsequent interviews involved weighing the subjects, and measuring blood pressure and heart rate, using the same methods as in the first session. Whereas the measurements had been taken in between questions on the original questionnaire for the first session, the measurements at these follow-up sessions were taken during the completion of the progress report, (see appendix P). The progress report outlined relevant changes that might have occurred since the previous visit, such as work pressure, stress level generally, dietary changes, activity level, alcohol intake. These sessions allowed for casual conversations, and a much more relaxed atmosphere than that of the first session. The first blood pressure measurement was taken within the first five, minutes of sitting down, and subsequent measurements were taken on three to five minute intervals. These follow-up sessions lasted on average twenty-five minutes. At the end of the session, arrangements were made for the next visit. During the final session (those subjects who had completed all 6 sessions) there were no formal time constraints. After the measurements were taken, the researcher used this opportunity to extract feedback from the participant, and to engage in light conversation if time allowed.

5.3f Verification and Validation of Measurements

Since a single observer took all measurements in this survey, at least two modes of validation were established.

- The multiple readings (6) for each subjects meant there was not a dependence on one measurement, and reduced the chance of error.
- The repeated measurements, (up to six 6 for each subject), served as an additional checks-and-balance

- Subjects were asked to, where possible, obtain independent assessments from their general practitioners, or other suitably qualified medical practitioner. This could be compared to the readings derived from the observer in this study.

- Subjects were asked, where possible, to bring the figures of their latest readings to be attached to or recorded on their questionnaires.

Note: There was no occasion on which the blood pressure values recorded by the observer in this study were found incorrect.

Summary Comments

Interviews were conducted in a closed private room, generally proceeding uninterrupted except by the telephone, with only the subject and the researcher in the room. The researcher tried not to convey a feeling of a clinic or office setting, but rather one of informality and ease. She also aimed to measure the subjects' blood pressures as close to their 'normal' resting behaviour pattern as possible. For instance, respondents who liked to talk were not discouraged from doing so, except while the actual measurement was in progress. After the first interview, many subjects developed a rapport with the researcher, and conversation was often natural. The researcher felt that conversing on matters of their choice would make them feel more at ease. It was also important that the participants not feel that they were just 'numbered subjects'.

Interviews went smoothly. Volunteers were, without exception, extremely co-operative and willing. There was never any indication by any participant of unwillingness to answer the questions. The interviewer informed subjects that they should feel free to question the reason for any question asked, but this 'privilege' was very rarely used. Wherever it was deemed necessary, the reason for the research was explained, emphasising the researcher's personal interest in the topic

and her intention to continue working in this area after the completion of this project, which virtually all subjects said they were pleased to hear. A certain amount of scepticism had been anticipated, but none was detected, although many had expressed a fear that this research might have been purely an academic endeavour, with no positive outcome to the African-Caribbean community.

Workshops

The researcher designed, organised and facilitated a number of workshops for the participants, where experts in the field gave presentations, see appendix Q). They were well received, and proved very beneficial to the participants.

6 Sixth Chapter

Structure of Analysis

6.1 OVERVIEW

6.1a Aim of Analysis

Analysis was designed to detect significant associations between the blood pressure levels of the subjects and their reported lifestyle. In essence, did those subjects with the lower blood pressures appear to have more desirable health behaviour practices than those with the higher blood pressures? Repeated measures analysis was used primarily to:

- To determine the range of variation within and across visits
- To determine the range of variation within and across subjects
- To determine if the blood pressure graph changed across sessions
- To observe changes in blood pressure with changing circumstances of the subject's life/lifestyle over the monitoring period.

6.1b First Stage Analysis

The data were first entered, checked 'cleaned' and coded into ordinal format. The following analysis were then completed prior to multivariate regression analysis:

- **Repeated measures analysis**: to examine changes in blood pressure and heart rate over time, within and between subjects. Of particular importance was the determination of differences in blood pressure and heart rate readings between session one and subsequent sessions. Body mass index stability was also analysed.
- **Frequency and descriptive analysis:** to determine the distributions (frequency, percentages, skew, kurtosis), Averages (mean), dispersions (standard deviation, range).

- **Exploratory statistics:** primarily stem and leaf graphs and statistics for more detailed distribution information, including percentiles and to determine levels of normality in the continuous independent variables (heart rate, body mass index) and the main independent variable systolic blood pressure. Outliers were identified, with a view to making necessary adjustments during analysis.

- **Cross tabulations** - Chi square and Gamma tests were used to derive early indications of associations. From the Gamma results, an indication of the strength of the association would also be derived.

- **Spearman and Pearson correlations:** to determine strength of correlated variables, and to examine multi-collinearity.

- **Scatter plots:** for graphical observations of correlations and linearity

* See Appendix Titled Statistical Analysis – Table of Contents- for detailed results..

6.1c Cross-tabulations plus

Chi-square analysis via contingency tables, (cross-tabulations), were used in the early stages of the analysis to determine associations, with the full knowledge that the chi-square 'test of independence', only offered indications of associations, and gave no substantive information about the strength of the relationships. The contingency tables were largely developed from ordinal data, (including blood pressure grouped. This facilitated the use of Gamma, Kendall's Tau and Spearman's statistics, which gave early indication of the strength of associations. Particular attention was given to wide variations in results, possibly resulting from non-monotonic relationships, especially as ordinal measures of association generally only detects monotonic relationships, (Dometrius 1992). A strong non-monotonic relationship between two ordinal variables, (that is, positive over some values of the independent variable and negative over others), would not usually be detected by ordinal measures of association, such as Gamma.

Statistical controls were used to eliminate the effect possible of suspected confounding independent variables. The results of these partial correlations (primarily first, and second order statistics), were important in identifying associations in sub-groups which were not otherwise evident. The most consistent control variable was age, but other independent variables were used as controls during this early exploratory stage of analysis.

The information derived from various cross-tabulation statistics allowed for the filtering of 'irrelevant' variables, and the further non-processing of those variables that showed no association with systolic blood pressure. Although some of the cross-tabulation statistics gave indications as to strength of associations, the results of these statistics were expressed and recorded (at this early stage), categorically, that is, reporting simply that there was or was not a significant relationship. Determination of the strength of the relationships was not of main concern at this stage as cross tabulations were the first-stage in the analysis process.

6.1d Linear Regression

On determining the normality of the distribution of the main dependent variable (systolic blood pressure), linear regression was selected as a suitable statistical method to develop the final predictive, (parsimonious), models. As Dometrius (1992) observed:

> *"Regression is robust. We could manufacture an artificial population and create a relationship between some variables that we know violates one or more regression assumptions. If we were then to take repeated samples from this 'population' and analyze the relationship with regression, the regression statistics would come pretty close to identifying the true population relationships. Obviously, a robust procedure has considerable value, and regression is such a procedure"*. (p. 437).

In order to be exhaustive various other non-parametric tests, including logistic regression, were carried out. This 'extra' analysis served to help identify questionable variables allowing for closer examination of variables concerned.

6.1e Linear Regression Prediction Standards

In social science research, an acceptable ratio of predictors to number of subjects in a regression analysis for is about 15 subjects per predictor. It is generally accepted that this ratio gives a reliable equation, that is, an equation that will cross-validate with little loss in predictive power, (in Stevens 1992 p.72). For this study 164/15 = 11, therefore, a maximum of eleven predictors would be acceptable in the models. Ultimately, the R^2, with close attention to confidence intervals, would determine the level of association between systolic blood pressure the variables in an equation. Guidelines on the minimum value that constitutes an acceptable R^2 were taken from Dometrius (1992) who stated:

> ***"In general, associations below .10 are not large enough to be important and may even be due to sampling error. Relationships between .10 and .20 are small but consequential. Relationships between .20 and .40 are moderate to strong, definitely large enough to be substantial and important. Any relationship above .40 can usually be considered quite strong"*** (p 314).

Much attention was paid to Multicollinearity, that is, moderate to high inter-correlations among the predictors. Stevens (1992) recognised that some inter-correlation should be expected when he wrote:

> ***"The ideal situation in terms of obtaining a high R would be to have each of the predictors significantly correlated with the dependent variable and for the predictors to be un-correlated with each other, so that they measure different constructs and are able to predict different parts of the variance on the y. Of course in practice we will not find this because almost all variables are correlated to some degree. A good situation in practice then would be one in which most of our predictors correlate significantly with y and the predictors have relatively low correlations among themselves"*** (p. 75).

6.1f The Lifestyle Variables categorised

The main lifestyle variables in this research were subdivided into categories as follows:

- Dietary Behaviour
- Alcohol Habits
- Smoking Habits
- Physical Activity Levels
- Body Mass Index (BMI)
- Socio-economic Factors

The only independent lifestyle variable measured on an interval scale, body mass index, was also used as a control, along with the main control, age. Both body mass index and age were independently correlated with systolic and diastolic blood pressures.

6.1g Blood Pressure Ordinal Categorisation

To facilitate analysis in an ordinal format, systolic blood pressure was divided into two categorises, as shown in table 1.1 on page 27 above.

6.1h Linear Regression Method

Stage one linear regression analysis involved entering those independent variables which had been shown to have an association with systolic blood pressure, even a weak one, into the univariate regression analysis. Thus, each variable was analysed within its group using 'dummy' variables, which is acceptable in regression analysis, (Dometrius 1992). Analysis using dummy variables was also relevant to this type exploratory statistical analysis as (when using dummy variables) the B-coefficient represents the difference in averages between the groups in the variables. Thus, this type of analysis would serve the main aim of this research, which was to ascertain the significance of differences in average systolic blood pressures between the different groups within a variable. Close attention was given to the results of

collinearity diagnostics, residual statistics, the F-statistics (test of linearity) and confidence intervals in determining the variables to be included in the final 'best' models. Enter method was used throughout the early regression analysis. This stage of the regression was primarily to determine the most likely candidates for the more complex multiple regression analysis from which regression equations could be derived. Stage two regression involved manipulation of those variables that had graduated to the multiple regression analysis. From these variables the final (prediction) model would be developed, using stepwise (forward) method.

Descriptive Results

7.1 GENDER, AGE, HEART RATE, BMI

7.1a Subjects and Gender

The survey comprised 164 African Caribbean subjects, comprising 138, (84%), females and 26 males (16%)

7.1b Age Distribution

Age was normally distributed, with a skew of .68, and kurtosis of .005, see fig. 7.1. below. Subjects were mainly in their thirties and early forties. The mean age was 38.2, SD ± 8.7, with a range of 41 years (range 20 - 61), for both males and females. Two percent of the subjects were under age 25; and 13% were under age 30; while 62% under age 40. Eighty-seven percent of the subjects were under age 50 years, 13% over age forty-nine years. The frequency distribution of analysis by age group by decade and five-year increments are shown in table.7.6 and table7.7.

Table 7.6 Age by Decade

Age group	<30	30-39	40-49	50 plus	Total
No. Subjects	22	80	40	22	164
% Total	13%	49%	24%	14%	100%

The majority of subjects were aged 30-39 years.

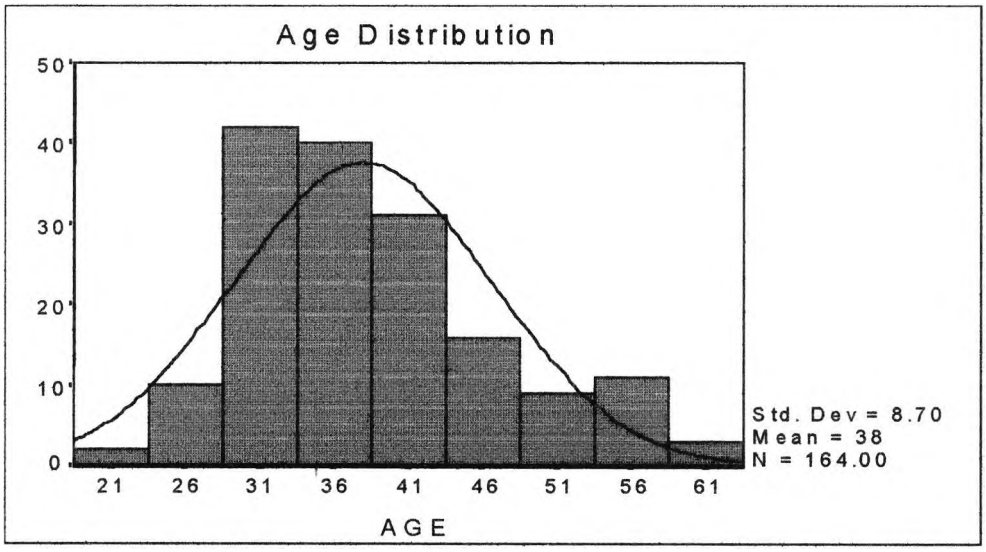
Table 7.7 Age by Five Year Incremental Grouping

Age group	<30	30-34	35-39	40-44	45-49	>50
No. Subjects	22	43	37	25	11	22
% Total	13	26	23	18	6	14

The majority of subjects were under age 40.

As the above tables show, for all three categorisations, the majority of subjects were in their thirties with a fairly balanced proportion on either side.

Fig. 7.1 Age Distribution



The histogram shows that age was normally distributed.

7.1c Drop-Outs

Of those persons who 'dropped-out' at any stage after the first session, four persons were excluded from participation in repeated measures at (different stages), because they were subsequently placed on blood pressure medication.[11] However, only one of the four subjects who were excluded had initial blood pressure in the very high blood pressure range (that is, SBP >159). Not all the persons who were placed on medication had blood pressure in the super hypertensive range, and not all those in the super hypertensive range were subsequently placed on medication.

[11] Once on medication, the blood pressure measurements would not give the individual's 'true readings'.

SUMMARY: GENDER AGE AND BODY MASS INDEX HEART RATE

A disproportionate proportion of the study population was female (84%). Exploratory test determined that both the male and female distributions were not significantly different for the variables age, body mass index, and heart rate. Males and females were therefore, treated as single population comprising 164 subjects. Where necessary analysis by gender could however, be carried out as the need arose.

7.2 BLOOD PRESSURE DISTRIBUTION

7.2a Repeated Measures

Analysis began with the determination of homogeneity of blood pressure across sessions, using Anova repeated measures design. The findings served to establish the validity of using the initial measurements of the 164 subjects (as the base readings) for all analysis and statistical modelling. This confirmation of homogeneity allowed for the analysis of the data to be modelled as a single (average), measurement on a single sample. A single systolic and diastolic blood pressure measurement was defined as the average of three seated and three lying readings taken at a single session, that is, the first session. The information attained from the first three of the six repeated measures was used to analyse changes in blood pressure, heart rate, and body mass index measurements over time. The distributions for the repeated measures for systolic blood pressure is outlined in table 7.8 below.

Table 7.8 Systolic Frequency Distribution Over Six Sessions

	Session Number							
Avg. SBP Subjects	**1- (164)**	**2 - (141)**	**3 - (117)**	**4 - (90)**	**5 - (67)**	**6 - (47)**	**Overall Mean**	**Overall Range**
Mean	122	121	123	121	121	122	**121**	**2**
Range	88	76	73	68	54	65	**71**	**34**
Minimum	93	92	94	93	99	92	**94**	**7**
Maximum	181	168	167	161	153	157	**165**	**28**

Mean systolic blood pressure remained reasonably constant across visits. The range decreased across visits, as the number of subjects decreased.

There was a **significant Pearson's r correlation** between the blood pressure readings of adjacent session, that is, over repeated measurements. The overall correlation between the first average systolic blood pressure results and those of the other five sessions reads was **r = 0.78**, and between the first (average) diastolic blood pressure measurements and the other five measurements was r = **0.62**. The mean systolic and diastolic blood pressures did not vary significantly over time, but there were marked differences in the range of systolic and diastolic blood pressures over six visits, with the range decreasing with time. It is important to note that the reduction in range of blood pressure for each measurement session was partly due to the decreasing number of subjects over time. In addition, some extreme hypertensive subjects were excluded early in the programme.

7.2b Systolic and Diastolic Blood Pressure

This research concentrated on systolic blood pressure, but where relevant, the diastolic blood pressure findings are reported and discussed. A strong positive correlation was found between average systolic and diastolic blood pressures:

Pearson's r = **0.79**. This indicated that subjects who tended to have higher levels systolic blood pressures, also tended to have higher levels diastolic blood pressures.

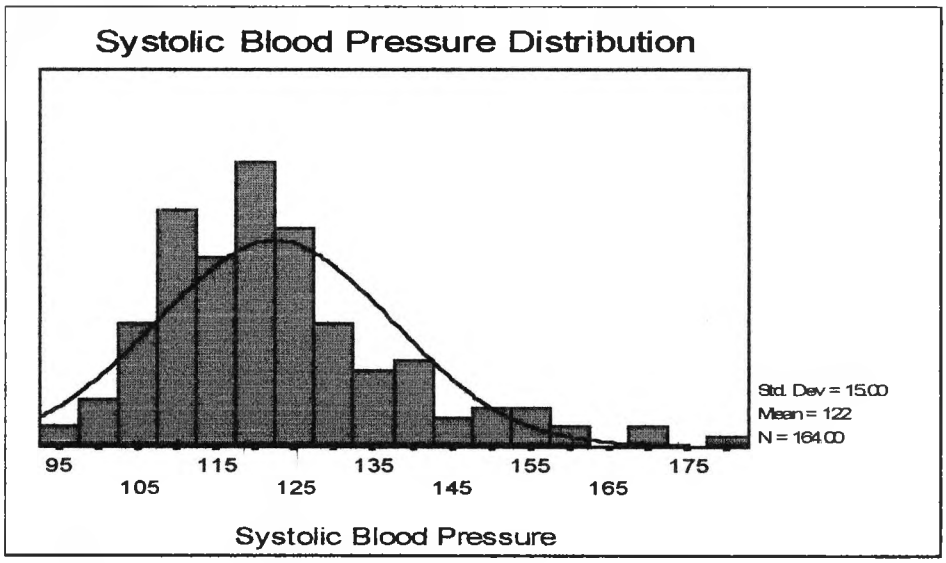
7.2c Diastolic Blood Pressure

Diastolic blood pressure was normally distributed, with a mean of 76.9 mmHg, SD ± 11.8, range 72. The majority of subjects (87.8 %) had diastolic blood pressure below 90 mmHg, and therefore, 12.2% were above 89 mmHg, with 105 subjects (64%) recording an average diastolic blood pressure of under 80 mmHg. Five subjects, (3%), had average diastolic blood pressure greater than 99 mmHg. Average diastolic pressure curve veered closer to normality than the systolic pressure curve.

7.2d Systolic Blood Pressure Distribution

Systolic blood pressure was normally distributed, see fig 7.2 below. Average systolic blood pressure (average of 3 seated and 3 lying) was 122.4 mmHg, SD ± 15.1, range 88.0, a maximum of 181 mmHg and a minimum of 93 mmHg. The majority, 127 subjects (77.4%), had systolic blood pressure below 130 mmHg, while 74 persons (45.7%) recorded an average systolic blood pressure of less than 120 mmHg. Fifty-three persons (32%), recorded systolic blood pressure in the mid or normal range of 120 to 129 mmHg, four persons. Thirty-seven subjects (23%), recorded mean systolic blood pressure above of 130 mmHg or greater. Twelve percent of the subjects recorded systolic blood pressure of 140 mmHg or greater.

Fig. 7.2 Systolic Blood Pressure Distribution



The histogram shows that systolic blood pressure followed a normal distribution.

When the subjects with systolic blood pressure greater than 139 mmHg were excluded leaving normotensive, the mean systolic blood pressure of those in the lower range (SBP <140; N=146), was 118.5 mmHg, SD ± 9.9, range 46.5. Approximately fifty percent, (50.7%) of these 'normotensives' recorded average systolic blood pressure of less than 120 mmHg. Thirteen percent, of this lower blood pressure group had systolic blood pressure in the high of normal range, (130 - 139 mmHg). Overall, seated blood pressure readings were significantly higher than the lying ones.

SUMMARY: BLOOD PRESSURE DISTRIBUTION

Both the systolic and diastolic blood pressure curves showed a normal distribution, but with a slight positive skew. Diastolic blood pressure had a more normal distribution than systolic blood pressure. Mean systolic blood pressure was 122.4

mmHg, while mean diastolic blood pressure was 77 mmHg. Just under 90% of the subjects had systolic blood pressure below what was categorised as the hypertensive range, that is, systolic blood pressure <140 mmHg.

7.3 INDEPENDENT VARIABLES

7.3a History of Raised Blood

A distinction was made between high blood pressure and hypertension.

- History of high blood pressure indicated that the individual had been told at some point, by a qualified medical practitioner that her blood pressure was high, (for instance, during pregnancy: pre-eclampsia). However, the individual was not diagnosed as hypertensive, as the high blood pressure was not sustained.
- History of hypertension related to medical diagnosis of sustained high blood pressure, with or without drug treatment.

The subjects' histories of raised blood pressures were self-reported. Sixty-six percent of the 164 subjects reported having no history of high blood pressure, therefore, 34% had been told that their blood pressure was high on at least one occasion, prior to undertaking this study.

A total of 153 persons, (93%) reported that they had never been diagnosed as hypertensive, therefore, eleven persons (7%), had been previously diagnosed hypertensive. Table 7.9 below shows the distribution.

Table 7.9 History of High Blood Pressure and History Of Hypertension

All	High BP History		Hypertension History	
No	109	65.6%	153	93.3%
Yes	55	33.5%	11	6.7%
Females N =138				
No	85	61.6%	128	92.8%
Yes	53	38.4%	10	7.2%
Males N = 26				
No	24	92.3%	25	96.2%
Yes	2	7.7%	1	3.8%

Females were more likely to have had high blood pressure than males, but the figures were the same for hypertension.

History Of Elevated Blood Pressure By Gender: Sixty-two percent of the 138 female subjects said they had no history of high blood pressure, compared to 92% of the 26 male subjects. Thus, 38% of females had a history of high blood pressure, compared to 8% males. The difference was proved significant, using Chi-Square cross tabulation analysis, (Pearson's and Mantel-Haenszel's test showed significance of = 0.002). The results were different for history of hypertension, as the proportions were similar for both genders.

7.3b Family Blood Pressure History

Family blood pressure history was examined with regards to the subjects' grandparents, parents, and siblings. The results are charted in table 7.10 below. The information in the table shows that a large percentage (56%), of the subjects' mothers had a history of elevated blood pressure, and that the percentage was significantly greater for mothers (double), than it was for fathers, (28%). Mothers also had higher percentage of stroke (12%), than fathers (4%). The percentages

were relatively similar for the subjects' siblings, with 20% of sisters and 14% of brothers reportedly having a history of hypertension.

Table 7.10 Family Blood Pressure History

Family BP History	Subject	Grand-parents	Mother	Father	Sister	Brother
% High Blood pressure	**34**	31	56	28	20	14
% Don't Know	-	52	6	4	0	8
% Yes Stroke	-	14	12	4	-	-

Subjects' mothers were more likely to have a history of raised blood pressure that their fathers, and sisters more than brothers.

The majority of subjects did know their parents and siblings histories of elevated blood pressures, but more than half did not know that of their grandparents. The respondents who knew grandparent's history tended to be those who had at least one grandparent with a history of high blood pressure.

7.3c Ethnicity and Duration in the UK

Forty-three percent of the subjects were Jamaican-born, and 16% percent were from the combined Eastern-Caribbean islands, making the total Caribbean-born 59%. The other 41% of subjects were UK-born. Ninety-eight percent of the subjects' parents (mothers and fathers) were born in the Caribbean, with the vast majority being Jamaican. A summary of subjects' birthplace is given in table 7.11 below.

Table 7.11 Subjects Birth Place

Birth Place	No.	Percentage
Jamaica	70	43
Eastern-Caribbean	27	16
UK	67	41
Totals N=164	164	100%

The percentage UK-born subject was similar to Caribbean-borns.

Table 7.12 Parents and Subjects Duration in the UK

Duration in UK	Parents (mother)		Subjects	
	No.	Percentage	No.	Percentage
Never	34	20	0	0
<21 years	9	6	15	9
21-30 years	33	20	43	26
>31 years	83	51	39	24
UK-born	5	3	67	41
Totals	164	100%	164	100%

M ost of the subjects and their parents had resided in the UK for over 30 years

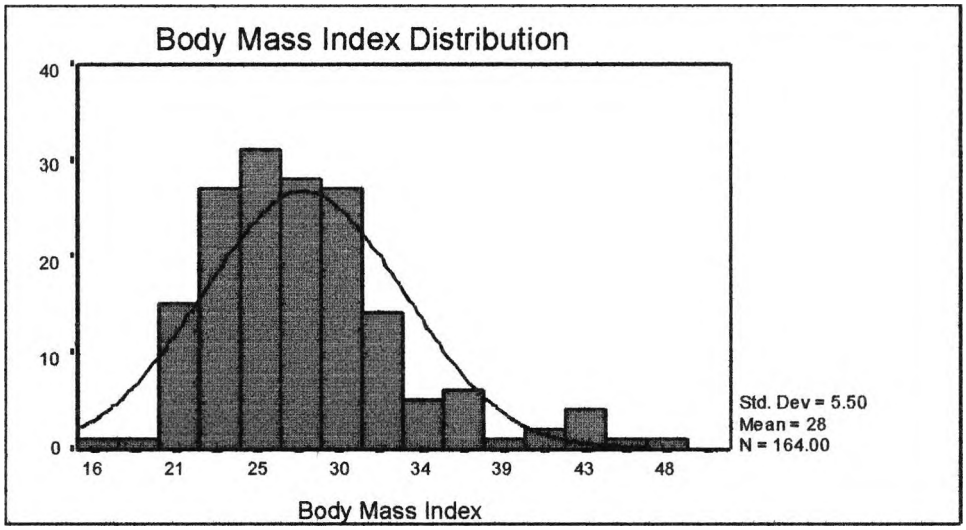
Table 7.12 above shows that while 20% of the subjects parents had never lived in the UK, the majority of parents (over 50%) had lived in the UK for more than thirty years. More than 72% of (non UK-born) parents had resided in the UK for over twenty years. Most of the subjects, (91%) had lived in the UK for more than 20 years, with thirty 41% being UK born.

7.3d Body Mass Index Distribution

The subjects mean body mass index was 27.6 kg/m^2, SD ± 5.5, with minimum being 16 kg/m^2 and maximum being 48 kg/m^2, a range of 31 kg/m^2. Body mass index was normally distributed, with slight positively skew of 1.1, with a 1.9 kurtosis, see fig.7.3 below. Fewer than twenty percent (19.5%) of the subjects had a body mass

index below 23 kg/m^2, while over fifty percent (52.4%) were less than 27 kg/m^2. Over eighty percent (81.1%) of the subjects had Body mass index below 31 kg/m^2. Body mass indices below 20 kg/m^2 (two persons), and above 40 kg/m^2 (seven persons) were considered extreme cases.

Fig. 7.3 Body Mass Index Distribution



Body Mass Index was normally distributed, with a mean of 28 kg/m^2.

Females had higher body mass indices than males, a mean of 27.8 kg/m^2, while the 26 male subjects had a mean of 26.2 kg/m^2. There was no statistically significant difference in body mass index between the different age groups.

7.3e Heart Rate

Heart rate was reasonably normally distributed, with an overall mean of 72.8 beats per minute, (bpm), SD ± 8.6, range 45. The minimum average heart rate was 52 bpm and the maximum was 98 bpm. Most subjects had heart rate in the low 70s, with values above 90 bpm and below 60 bpm being extreme. Seated heart rate

readings were higher than lying ones and readings taken earlier in the session were higher than those taken later. Forty-three percent of the subjects had average heart rate of 70 bpm or less and 81% had heart rate eighty beats per minute or less under. Nineteen percent of the subjects had heart rate greater than 80 beats per minute. Male subjects had overall lower mean heart rates (70 bpm, SD ± 8.5), than the female subjects (74 bpm, SD ± 8.5). The male range was 28 beats per minute while the female range was 44 beats per minute. Although males generally had lower heart rates than females, the difference was not statistically significant. There was no statistically significant difference between heart rates across age groups.

7.3f Accommodation

Adequate. The majority of the subjects (67%), reported that their accommodation was very adequate, and at least 75% lived in what could be classed low crowding conditions. Table 7.13 below summarises the frequencies.

Table 7.13 Accommodation Adequacy

Accommodation	Number	Percentage
Adequate	110	67%
Not Adequate	54	33%

Most subjects reported that their accommodation was adequate, that is, satisfactory.

There was a significant age difference between subjects who reported that their accommodation was very adequate (40.2 years, SD ± 8.9), and whose accommodation fell below this level (34.2 years, SD ± 6.8). Analysis of variance test of the difference in means derived an f statistics of 19.2, Sig = 0.000.

Crowding: Table 7.14 below shows that most respondents (46.3%) lived in medium crowding housing, with approximately equal percentages in highest and lowest crowding accommodation. The age difference between subjects in the three crowding groups was significant. Analysis of variance found the association to be linearly significant, Sig = 0.0009. The mean age of subjects reduced as the level of crowding reduced. The average age of those with the highest crowding was 35.5 years, SD ± 6.0 compared to 37.4, SD ± 8.9, for medium crowding, and 42.1, SD ± 9.3 for highest crowding.

Table 7.14 Accommodation Crowding

Crowding	Highest	Medium	Lowest
Number	42	76	46
Percentage	26%	46%	28%

Most subjects lived in low crowding housing.

7.3g Education

Education (based on academic qualifications) was grouped by four levels: University (highest), professional (second highest), high school/tertiary, and none. The majority (51%), of subjects had a high school/or tertiary education. The combined group of university and professionals made up 33% of the subjects. The distribution is shown in table 7.15 below.

Table 7.15 Education levels

Education	University	Professional	High School	None	Total
Frequency	28	26	83	27	164
Percentage	17%	16%	51%	16%	100%

Most subjects had high school level qualifications, that is, GCSE 'O and A levels.

There was no discernible age relationship with qualifications. The average age of those respondents with no qualification was the same as those with professional qualifications, 41 years.

7.3h Job Grade

Categorisation of job grades was carried out with the guidance of the organisation based primarily on rate of pay. Although this classification was not a strict adherence of the national guidelines, it offered a practical, working categorisation of the employees by ascending job grade. The subjects were categorised into five job grade groups, the highest being executives and top managers. In the second highest were middle managers, highly qualified social workers, and other top professionals. The middle group members were partially qualified social workers, secretarial and administration assistant staff. Subjects in the second lowest were generally clerical and in the lowest were clerical assistants, home helps, janitors and messengers. Table 7.16 below shows the frequency distributions.

Table 7.16 Job Grade

Job Grade	Highest	2nd Highest	Middle	2nd Lowest	Lowest
Frequency	23	74	28	19	20
Percentage	14%	45%	17%	12%	12

The largest percentages of respondents were in the second highest job grade, that is, middle managers and highly qualified social workers. Overall, greater proportions of the respondents were in the higher grades.

Most subjects, (59%), were in the top two job grades, ranging from top executives within the organisations, to managers and qualified social workers. Seventeen percent were in the middle grades, comprising middle managers and administration staff. Twenty-four percent of the subjects fell in the lowest grades, ranging from

administration assistants to drivers and care assistants. While the mean age of those subjects in the lowest job grade was higher than the other groups (42.5 years), the difference in ages across job grades was not significant.

7.3i Racism: Anger

A large percentage (46%), of subjects were at least sometimes angered by racism. Fifteen percent said that they felt angry because of racism almost on a daily basis, while 29% were angered on average once a week. Ten percent said they were very rarely angered because of racism. The distributions are shown in table 7.17 below.

Table 7.17 Anger from Racism

Anger From Racism	**Daily**	**Weekly**	**Sometimes**	**Rarely**
Frequency	24	48	76	16
Percentage	15%	29%	46%	10%

Anger from racism was most often felt sometimes, and quite often felt weekly.

7.3j Racism: Colleagues and Career

Career: The greater majority of the 164 subjects (43%) felt that their career development had been stymied by racism quite a bit. Twenty-nine percent felt that racism had little or no effect on their career development, and 28% were not sure.

Colleagues: The majority (66%) said that they had suffered little or no racism from their colleagues. The other 34% reported having suffered quite a bit of racism from colleagues. Table 7.18 below gives a breakdown for racism from colleagues and effect of racism on career.

Table 7.18 Effect of Racism on Career and Racism from Colleagues

Racism Effect	None/Very little		Quite a Bit		Not sure	
	Subjects	%	Subjects	%	Subjects	%
Career	47	29%	71	43%	46	28%
Colleagues	109	66%	55	34%	00	00%

A large percentage of subjects felt that racism had affected their careers quite-a-bit. Most subjects had suffered little of no racism from colleagues.

7.3k Dietary Habits

Dietary habits analysed using cross tabulation were: General diet (vegetarian to non-vegetarian status), and habits relating to the general consumption of coffee, fried food, red meat, heavily seasoned foods, cooking with salt, and adding salt.

General diet was categorised by vegetarian status. Vegetarian was defined as non-meat and non-fish eaters, but included those persons who occasionally or rarely ate meat or fish; part vegetarians ate mainly vegetarian diets, but did eat meat or fish at least once, but not generally more than three times per week. The majority (70%) of the 164 subjects reported that they were non-vegetarians; 27% said they were part vegetarians; and 3% reported that they were full vegetarians. Although, a greater percentage of the under 35 years of age group were non-vegetarians (75%), the differences between age groups were not significant. Overall, the older age group ate less meat than the younger age group.

Healthy meals: A healthy meal was nutritious, vegetarian or otherwise. The majority (59%), of the subjects ate on average one healthy meal daily. Thirty

percent ate approximately two healthy meals each day and 5% reported eating three healthy meals each day on a regular basis. Five percent did not eat a healthy meal on a daily basis.

Fried Foods: Most subjects reported that they did not eat fried foods on a regular basis. Sixty-three percent reported that they ate fried foods at most once per week, 26% of which ate fried foods very rarely. Thirty-seven-percent said they ate fried foods regularly and 18% ate it most days of the week.

Red Meat: The majority of subjects (71%) ate red meat as part of their regular normal diet, with 29% who never or only rarely ate it. Sixty-four-percent ate red meat at least once per week, and 7% ate read meat most days of the week

Seasoned Food: Most subjects, (73%), included heavily seasoned or spicy foods in their regular diet. Thirty-four percent ate seasoned foods as part of their daily diet, 39%, ate it on a regular basis, and 27% did not generally eat heavily seasoned foods.

Table 7.19 below shows the distribution of fried foods, red meat and seasoned foods.

Table 7.19 Fried Foods, Red Meat, Seasoned Foods: Dietary Habits Salt

	Fried food	Red Meat	Seasoned Food
Daily	18%	7%	34%
Regularly	19%	64%	39%
Rarely/Never	63%	29%	27%
Total	100%	100%	100%

Most respondents rarely ate fried foods, but most ate red meat. The distribution for seasoned foods was generally equal.

7.3l Salt

Cook with Salt: Most (83%), subjects always cooked with salt. The other 17% either sometimes cooked with salt, (6%), or never or rarely did (11%).

Add Salt: An almost equal proportion of subjects reported never or rarely adding salt (48%) to those who added salt at least sometimes, (52%). Most (42%), of those who added salt did so sometimes, thus 10%, of the subjects always added salt.

Cooked with and Added Salt: Table 7.20 below shows the distribution of salt use.

Table 7.20 Cooking with Salt and Adding Salt Habits

Salt Habit	Never/Rarely		Sometimes		Always	
	Frequency	%	Frequency	%	Frequency	%
Cook with	18	11%	10	6%	136	83%
Add	79	48%	68	42%	17	10%

The vast majority of subjects always cooked with salt, while very few always added salt. Most subjects would either very rarely add salt or sometimes add salt.

Cross-tabulation analysis derived a significant relationship (Mantel-Haenszel test Sig = 0.003) between cooking with salt and adding salt, revealing that generally persons who added salt also cooked with salt. However, the reverse was not necessarily true, as only 13%, of those who habitually cooked with salt would habitually add salt at the table. Of the 135 subjects (86% of the total 164) who always cooked with salt, almost half (44%), would at least sometimes add salt. In contrast, the subjects that never or only sometimes cooked with salt would never or

very rarely add salt, (79%). Four subjects reported that they sometimes added salt (mainly to salad dishes) but never cooked with salt.

7.3m Coffee

Seventy percent of the subjects never drank coffee. Thirty-four percent drank 1 to 7 cups per week, and 23% drank 8 or more per week. Coffee drinking habits were similar across age groups.

7.3n Alcohol

The large majority (72%), of subjects drank alcohol, thus 28% generally did not. Thirty-eight-percent of the alcohol drinkers were occasional drinkers, 47%, consumed less than 8 units per week, and 15%, consumed more than 7 units per week. The distribution is shown in Table 7.21 below. Thirty-three percent of alcohol drinkers started before age 18 years of age, 42% started between age 18-21 years, and 25%, at various ages after age 21. Forty-four percent participants consumed less alcohol weekly than they had in the past, 37% consumed the same and had not changed their alcohol habits. Nineteen percent of the respondents said that their weekly consumption rate had risen over the past two years.

Table 7.21 Weekly Alcohol Intake Levels of the Subjects

Alcohol Intake	Occasional	Under 8 Units	Over 7 Units
No. o f Subjects	38%	47%	15%

Eighty five percent of subjects consumed less than the recommended weekly limit of alcohol

7.3o Smoking

The majority (75%), of the subjects were non-smokers, thus 25% smoked. Sixty-percent reported that they had never smoked, therefore, 15% of the non-smokers had smoked in the past. Of the forty-one subjects who were smokers, half, (51%), started before age 18 years, and with the other half starting to smoke at an older age. More persons from the younger age groups started smoking before age 18, than from the older groups. Sixty-six-percent of smokers smoked less than 10 per day. A large percentage of these relatively 'light' smokers would not generally smoke more than one or two cigarettes daily. Thirty-four-percent of the smokers generally smoked more than 10 daily. Forty-two percent of the smokers said they smoked less now than two years ago and thirty-32 % smoked more. Twenty-six-percent said their smoking habits had not changed in recent years.

Passive Smokers: Fifty-five percent of the respondents said they were regular passive smokers. More than half of these passive smokers was exposed at least three times per week.

7.3p Physical Activity

Physical activity was defined as any type of structured activity, that is, exercise, or other daily physical activities such as walking to work, taking the stairs instead of the lift, and gardening. The terms exercise and physical activity were used interchangeably. When total activity was calculated, 33% of the group never exercised. Members of this non-exercise group generally did not walk more than they needed to, and made no effort to include physical activity in their daily or weekly routine. Twenty-nine percent of the total subjects said they did make the effort to get some physical activity each week, but conceded that generally were not

very active, while 26% reported that they were fairly active. Only12% of the group fell into the very active category. The majority of subjects said they were active at home and on weekends. This type of activity was largely centred around domestic chores and outdoor pursuits. Fifty percent of the group reported being very active at work, while 24% felt they were reasonably active and 26% said they have very little physical activity on the job.

Summary of Descriptive Results

Of the 164 subjects, 138 (84%) were females, and 26 (16%) were males. Age was normally distributed, the average being 38 years, ranging from 20 to 61. Average heart rate was 74 beats per minute. Body mass index was normally distributed, with a mean of 28 kg/m^2, ranging from 16 kg/m^2 to 48 kg/m^2. Male subjects had lower heart rates and body mass indices than females, but the differences were not highly significant. Blood pressure followed a normal distribution, with diastolic blood pressure being more bell-shaped than systolic blood pressure. Average systolic blood pressure was 122.4 mmHg and average diastolic blood pressure was 77 mmHg. Both systolic and diastolic blood pressures were positively correlated with age thus the younger participants tended to have lower blood pressures than their older colleagues. Blood pressure was associated with body mass index (when grouped). Blood pressure was not associated with heart rate. The vast majority of the 164 subjects (88%) were normotensive, that is, recorded systolic blood pressure less than 140 mmHg. The correlation between systolic and diastolic blood pressures was Pearson's $r = 0.79$.

The majority (66%), of the subjects had no known history of high blood pressure, and a larger majority, (93%), had no known history of hypertension. A greater percentage of females (38%), had history of high blood pressure than males (8%), making the difference statistically significant, but there was no significant difference in the male and female percentages of history of hypertension. History of raised blood pressure was positively correlated with current blood pressure. There was no significant difference with either high blood pressure history, or hypertension history across the age groups. More of the subjects' mothers had a history of hypertension than their fathers, 56% compared to 27%. The differences were also significant for stroke history: 12% for mothers and 4% for fathers. Family's history of hypertension was not significantly associated with the subjects' current blood pressure.

The general dietary habits of the group was (normal) healthy. Consumption of the least 'desirable' foods such as red meat, and spicy foods was not excessive. Salt intake was did not appear to be excessive. Overall, the subjects appeared to maintain reasonably (normal) healthy lifestyles. The majority the group was non-smokers, and did not consume low to moderate amounts of alcohol each week. Many alcohol drinkers were only occasional drinkers. The group's overall physical activity levels were about average, based on national standards.

Results of Analysis

8.1 Simple Relationships

The relationships between blood pressure and the independent (lifestyle) variables, were first explored using cross tabulations and stem and leaf graphs, with the aim of establishing bivariate relationships. To facilitate the use of contingency tables (cross-tabulations), systolic blood pressure was grouped by two methods.

Category A: The subjects were divided into three groups based on the mean systolic blood pressure and the standard deviation from the mean, as shown in table 8.22 below. Respondents whose blood pressure fell within one standard deviation, (15 mmHg), of the mean (122.4 mmHg) were classed as *average* or normal. Those subjects whose blood pressures were below or above one standard deviation of the mean were classed as *low* or *high* respectively. Comparative analysis of lifestyle was carried out mainly based on this triadic systolic blood pressure categorisation.

Table 8.22 Categorisation A Based on Mean Systolic Blood Pressure

	Low	Average	High
SBP	93-106 mmHg	107-137 mmHg	138-181 mmHg
Frequency	16	123	25
Mean systolic blood pressure = 122.4 mmHg SD = ± 15 = mmHg			

Average systolic blood pressure is categorised as ± 1 standard deviation of the mean. Low and High represents blood pressures lesser or greater than 1 standard deviation of the mean.

The vast majority of subjects had average blood pressure centred around the mean, with an even distribution on either side.

Category B: This second method was based on the range of blood pressures found among the subjects, as outlined in table 8.23 below. With this classification, there were two groups comprising those subjects at the lower end of the range, and those at the higher end. The mid-point of the range, (137 mmHg) was used as the demarcation line. Differences in lifestyle were compared between the highs and the lows. The distribution of **Category B** showed a disproportionate number of highs (24) to lows (140).

Table 8.23 Categorisation B - Based on Range of Systolic Blood Pressure

Category	Low	High
SBP	93-137 mmHg	138-181 mmHg
Frequency	140	24
Range = 93 mmHg to 181 mmHg : Mid Point of Range 137 mmHg		

The vast majority of respondents recorded systolic blood pressure in the lower of the two ranges.

8.1b Age Blood and Pressure

There was a significant positive correlation between age and both systolic and diastolic blood pressures: Pearson's r =0 43 (p= 0.000). Analysis of variance of age by decade, and age grouped by *OPCS* categories yielded F-statistics of 12.4, and 14.6 (Sig = < 0.05) respectively. Table 8.24 below shows that younger subjects therefore, generally had lower blood pressures than their older colleagues.

Table 8.24 Age by decade mean systolic blood pressure

Age Group	Mean SBP	Minimum	Maximum	Range
Under 30	117.6	101	141	40
30-39	117.8	93	170	77
40-49	127.3	103	160	57
over 49	135.4	108	181	73
All Ages	122.4	93	181	88
Mean age = 38.2, SD ± 8.7				

Systolic blood pressure increased (linearly) with age.

8.1c Blood Pressure History and Blood Pressure

There was an (expected), association of blood pressure with both history of high blood pressure and history of hypertension and present systolic blood pressure status. Both variables yielded approximately the same correlation: Pearson's r = 0.37; Spearman's r = 0.32; and Kendal's r = 0.25. The correlation with diastolic blood pressure was lower (approximately r= 0.21)

8.1d Family Blood Pressure and Blood Pressure

The blood pressure and stroke history of the subjects' grandparents, parents, and siblings was examined. Table 8.25 below shows that while a positive association was found between the subjects recorded blood pressure their brother's pressure history, no association was found with any other family member.

Table 8.25 Blood Pressure and Family Blood Pressure History

Family Member	Parents	Grand-Parents	Sisters	Brothers
Association	No	No	No	Yes

Only brothers' blood pressure was (positively) associated with the subjects own blood pressure

8.1e Heart Rate and Blood Pressure

No significant relationship was found, using both Pearson's and Spearman's correlation coefficients, between blood pressure and heart rate. Subjects' blood pressure could not be predicted from their heart rates

8.1f Body Mass Index and Blood Pressure

Subjects whose body mass index fell in the lowest range (< 23 kg/m^2), also had blood pressure in the lower range. There was, however, no significant difference between subjects at the highest end of the body mass index scale and those in the group just below them. However, those subjects in the group just below the highest, (27-30 kg/m^2), recorded lower mean systolic blood pressure than their heavier colleagues. Analysis of variance F-statistics found the overall differences in means between the different body mass index groups to be significant, when body mass index was analysed according to the grouping outlined in table 8.26 below, as significance of <0.05. There was a significant Pearson's correlation of r = **0.221** (Sig <0.05) between blood pressure and body mass index.

Table 8.26 Blood Pressure by Body Mass Index Grouped

Body Mass Index - kg/m^2	Mean SBP	SD	Cases
BMI – Under 23	116.8	11.9	32
BMI – 23-26	120.7	15.1	54
BMI – 27-30	126.3	15.9	47
BMI – Over 30	125.4	14.5	31
Mean body mass index = 27.6, SD ± 5.5: *Mean systolic blood pressure = 122.4, SD ± 15.0*			

The range of body mass indices was reasonably evenly distributed across subjects. Systolic blood pressure was positively associated with body mass index, but there was little difference between the two highest groups.

Systolic blood pressure increased linearly as body mass index increased. Subjects with the lowest body mass index (under 23 kg/m^2), recorded an average systolic blood pressure 8-9 mmHg lower than lower than the highest body mass index groups. The second lowest group (23-26 kg/m^2) recorded mean systolic blood pressure of approximately 5 mmHg lower than the groups above it. However, when body mass index was analysed as a continuous variable, an independent significant Pearson's correlation was not found between body mass index and blood pressure, with the Pearson's r = 0.151 Sig = 0.054.

Gender and body mass index: When the 138 female subjects were analysed separately, the independent association between body mass index and systolic blood pressure was less significant than for the whole group. The same was true for the 26 males. Thus, body mass index was not significantly associated with blood pressure when both males and females were analysed separately.

8.1g Diet and Blood Pressure

General diet, (vegetarian vs. non-vegetarian), did not have a significant association with average systolic and diastolic blood pressures. For all blood pressure groups, the greater percentage of participants were not vegetarians, and there was no indication that those persons were at the lower end of the blood pressure continuum were more likely to favour vegetarian diets. Table 8.27 below shows the associations found between blood pressure and each of the dietary variables.

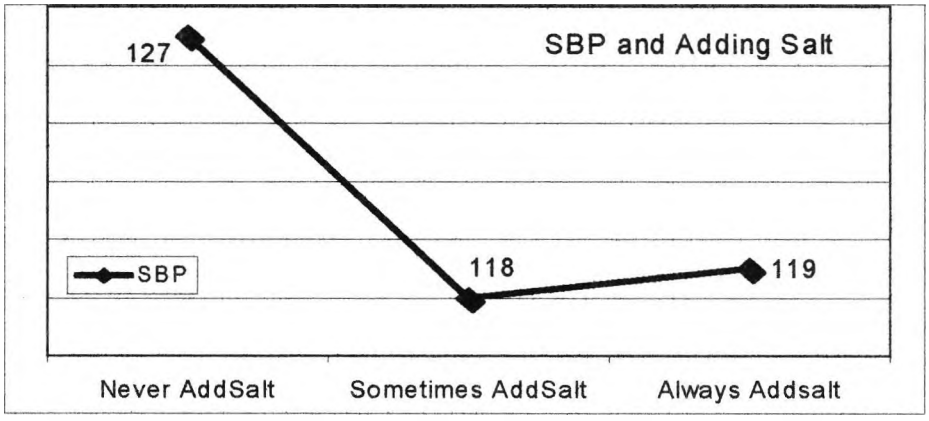
Table 8.27 Average SBP Association with Dietary Habits

	Add Salt	Coffee	Cook Salt	Diet	Fried Food	Healthy	Red Meat	Season Food
Spearman	**-0.23**	0	0.12	0.02	0.03	0.09	0	0
Result	Neg.	Nil	Nil	nil	nil	Nil	nil	nil

Except for adding salt, dietary habits were not associated with systolic blood pressure. The association with adding salt was an inverse one.

Adding Salt: The only dietary variable that had a significantly, but negative association with blood pressure was adding salt. This suggested that participants whose blood pressure were in the higher ranges, were less likely to add salt to their meals than their colleagues who had lower levels of blood pressure. The group that never or rarely added salt recorded the highest mean systolic blood pressure, 127 mmHg, SD±17.7. The sometimes added salt and always added salt groups had similar mean systolic blood pressures, (118 mmHg, SD±10.4 and 119 mmHg, SD± 9.8 respectively). The sometimes-added salt group was therefore, 1 mmHg lower. Since the mean systolic blood pressure was similar for both sometimes added salt and always added salt, the adding salt variable was re-grouped nominally, into yes (add salt), and no (do not add salt). This converted to 48%, who never generally added salt, and 52%, who added salt at least sometimes. There was a difference in mean systolic blood pressure of 9 mmHg between subjects who did not generally add salt, (127 mmHg) and those who did, (118 mmHg). A graphical representation is shown in fig. 8.4 below.

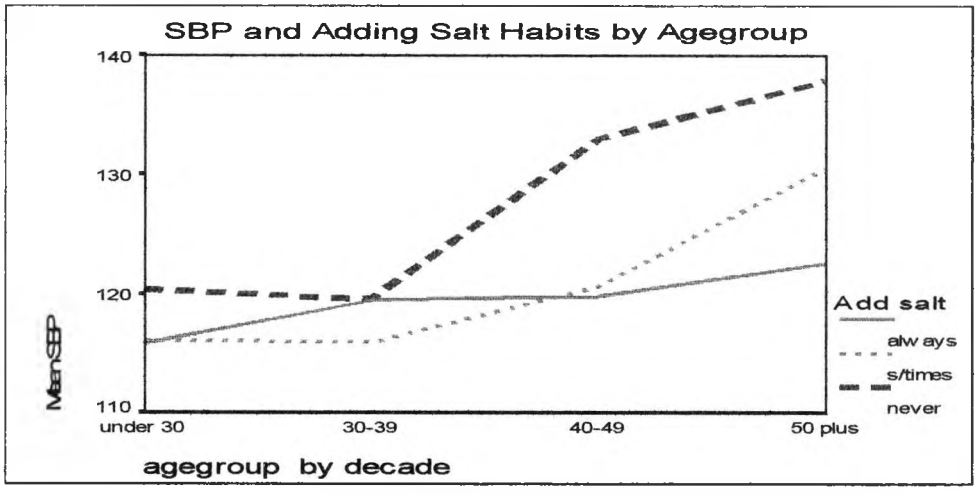
Fig. 8.4 Systolic Blood Pressure with Adding Salt



Respondents who never added salt recorded significantly higher systolic blood pressures than those who sometimes or always did.

Adding salt and Age: Cross-tabulation of blood pressure and adding salt by **age group** showed a significant negative relationship, but only for the 35-44 age group, (Spearman r = -0.29). Figure 8.5 below graphically represents the relationship between salt habits and systolic blood pressure by age group.

Fig. 8.5 Systolic Blood pressure and Adding Salt Habits by Age-group



Older respondents who never added salt recorded higher mean systolic blood pressures than the younger never add salt groups. At the same time, the older age groups who always added salt also recorded higher systolic blood pressures than their younger always-added salt colleagues did.

Thus, the association referred to one age group. The negative relationship indicates that those subjects, (in the age 35 to 44 bracket), who were most likely to add salt were the ones with lower levels blood pressure. Members of the older age groups that were likely never to add salt were also more likely to have blood pressure in the higher range than members of their group that sometimes or always added salt. Thus, older people with higher levels of blood pressure did not tend to add salt.

SUMMARY OF DIETARY VARIABLES AND BLOOD PRESSURE

Generally, the subjects' dietary habits did not appear have a significant association with their blood pressure. The only dietary variable that was (negatively), associated with blood pressure was adding salt and indications were that the association related to only one age group, the 30s to 40s age group. Generally, the blood pressure values of those who tended towards healthy eating habits, was not significantly different from the less healthy eaters.

8.1h Alcohol and Blood Pressure

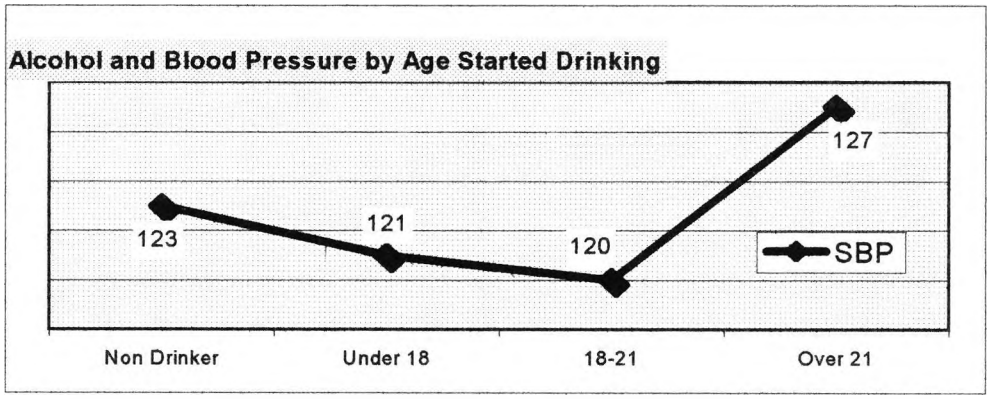
No significant association was found between systolic blood pressure and alcohol habits. Alcohol drinkers and non-alcohol drinkers had the same average systolic blood pressure of 122.3 mmHg, *SD ± 14.1* and 122.9 mmHg, *SD ± 17.3* respectively. Non-alcohol drinkers did not have significantly lower systolic blood pressure than alcohol drinkers. No association was found between age and drinking habits, hence the alcohol consumption habits were similar across age groups.

Amount Drunk Weekly: Subjects who rarely consumed alcohol had lower mean systolic blood pressure than those who drank more often: 121.4 mmHg, *SD ± 14.5*

and approximately 123.5 mmHg, *SD ± 16.0*, (approximately), respectively, but the difference was not statistically significant.

***Age started drinking*:** Subjects who reported starting drinking at age over 21 years, recorded higher blood pressures than their counterparts who began drinking at an earlier age, see fig. 8.6 below. However, the difference in blood pressure between the earlier starters and the later starters was not statistically significant.

Fig. 8.6 Systolic Blood Pressure and Age Started Drinking Alcohol



Respondents who started drinking later in life recorded higher systolic blood pressures than their colleagues who started earlier and also than teetotallers.

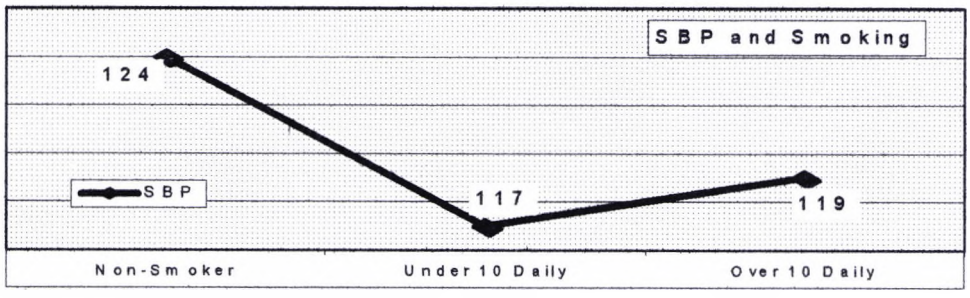
8.1i Smoking and Blood Pressure

Smokers recorded significantly lower mean systolic blood pressure than non-smokers: 117.7 mmHg, SD ± 11.1, and 124.0 mmHg, SD ± 15.8 respectively. Thus a significant inverse association was found between blood pressure and smoking.

Amount Smoked: Subjects who smoked the most (ten or more cigarettes daily), recorded on average lower systolic blood pressure (116.8 mmHg, *SD ± 7.7)*, than

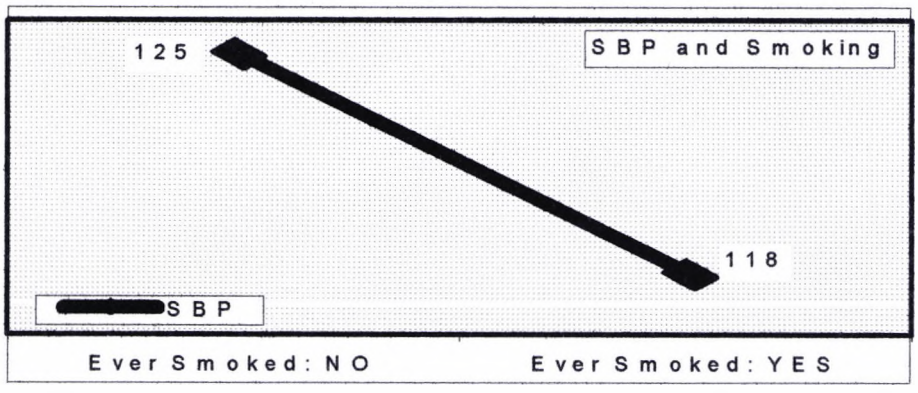
Amount Smoked: Subjects who smoked the most (ten or more cigarettes daily), recorded on average lower systolic blood pressure (116.8 mmHg, *SD ± 7.7)*, than fellow smokers who smoked less than 10 daily, (118.2 mmHg, *SD ± 12.5)*, and also lower than non-smokers (124.0 mmHg, *SD ± 15.8)*. The relationship is shown in fig. 8.7 below. However, the differences were not statistically significant, and even less significant when non-smokers were left out of the analysis.

Fig. 8.7 Systolic Blood Pressure and Amount Smoked Daily



Non-smokers recorded significantly higher systolic blood pressures than smokers. Heavier smokers recorded higher blood pressures than lighter smokers.

Fig. 8.8 Systolic Blood pressure and Ever Smoked



Respondents with no previous history of smoking recorded higher systolic blood pressures than those who were past (or present) smokers.

Ever Smoked: Figure 8.8 above shows that there was also an inverse association between blood pressure and history of smoking. Those members of the group who did have a history of smoking, (ever smoked) even if no longer a smoker, recorded

lower mean blood pressure 117.9 mmHg, *SD ± 12.0* than those persons who had never smoked - 125.4 mmHg, SD *± 16.0*.

***Age started Smoking*:** Smokers who started before age 18 years, had a lower overall mean systolic blood pressure (116.4 mmHg, *SD ± 13.4*), than those who started smoking after age 18 years (118.8 mmHg, *SD ± 10.1*). When non-smokers were included in the analysis the difference was significant, but lost significant when non-smokers were excluded. Thus, within the smokers category, age started smoking did not significantly impact on systolic blood pressure.

***Passive smoking*:** No association was found between passive smoking and blood pressure, with mean blood pressure being similar across levels of exposure to smoking environments.

8.1j Exercise and Blood Pressure

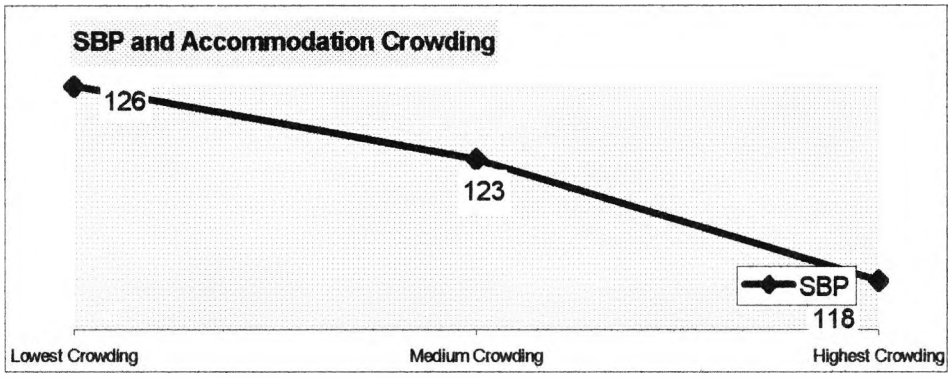
No association was found between blood pressure and any of the exercise and activity variables. Activity level did not seem to affect the subjects' systolic blood pressures. The results were similar across age groups.

8.1k Accommodation and Blood Pressure

Adequate: The mean systolic blood pressure of 116.4 mmHg, SD ± 13.1 of those subjects who felt that their accommodation was inadequate was lower at significant <.05, than those who reported that their accommodation was adequate, who had a mean systolic blood pressure of 125.4 mmHg, SD ± 15.1. Analysis of variance found the difference to be linearly significant at Sig = 0.0003.

Crowding: Respondents who lived in the most crowded conditions (number of people per rooms), had lower systolic blood pressure (118 mmHg, SD ± 10.5), than those who had the second lowest levels of crowding, (123 mmHg, SD ± 13.6).

Fig. 8.9 Systolic Blood Pressure and Accommodation Crowding



The relationship between crowding and systolic blood pressure was negatively linear.

Figure 8.9 above shows that the most crowding group also had lower mean systolic blood pressure that those who had the lowest levels of crowding, whose mean systolic blood pressure was 126 mmHg, SD ± 19.3. Analysis of variance statistics found the difference to be linearly significant at Sig = 0.036.

8.11 Education and Blood Pressure

Analysis of variance tests of a linear relationship between education level and blood pressure showed non-significance. Both the group with no qualifications (none), and that with the highest qualifications, (university level), recorded higher mean blood pressures than those in the middle categories of education. However, the 27 subjects who were the least educated group, (no qualifications), recorded the overall

highest mean systolic blood pressures, of 127 mmHg, SD ±16.7. Table 8.28 below shows the distribution.

Table 8.28 Qualifications and Systolic Blood Pressure

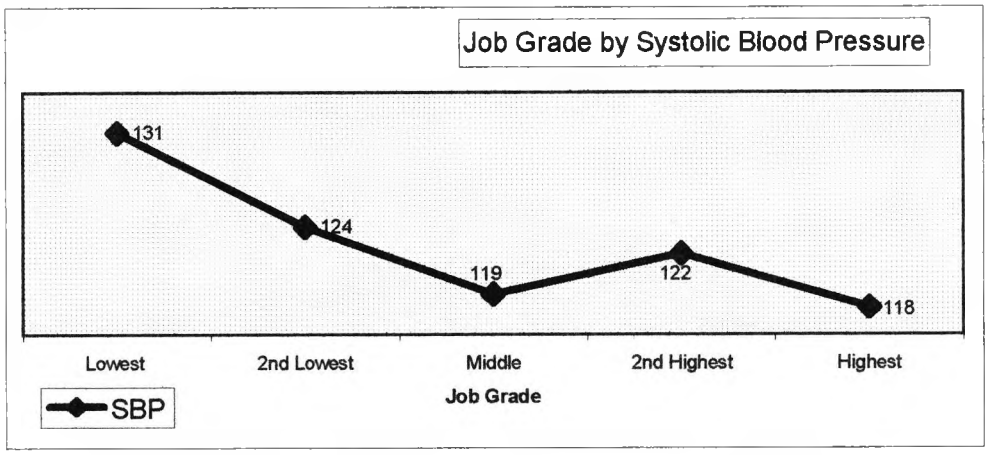
Qualification	Mean SBP	SD	Cases
None	127.7	16.7	27
High School	121.0	13.0	83
Professional	120.7	17.5	26
University	123.2	15.9	28

Those subjects with no qualifications recorded the highest systolic blood pressure, the most educated (university level), recorded the second highest mean systolic blood pressure.

8.1m Job Grade and Blood pressure

The relationship between blood pressure and job grade was somewhat negatively linear between the three lowest grades, that is, lowest, second lowest and middle. Respondents in the second lowest and lowest job grades recorded the highest mean systolic blood pressures, 124 mmHg, SD ± 14.8 and 131 mmHg, SD ± 19.7. Subjects in the highest job grade had the lowest systolic blood pressure, 118 mmHg, SD ± 11.6. Although the relationship between blood pressure and job grade was (overall) negatively linear between the three, lowest grades, that is, lowest, second lowest and middle, the linearity did not extend across all the groups. Analysis of variance found a linear significant F-statistic of 2.8, Sig = 0.028, between the different job grades. A graphical representation is shown in fig. 8.10 below.

Fig. 8.10 Systolic Blood Pressure and Job Grade



Subjects in the lowest job grades recorded significantly higher systolic blood pressures than their colleagues in the highest job grades

8.1n Blood Pressure and Family's Blood Pressure History

Family blood pressure history was not significantly associated with subjects recorded blood pressure. Only brother's blood pressure history had a significant (positive), relation to the subjects' own blood pressure. Subjects who had a brother with high blood pressure had significantly higher mean systolic blood pressure (132 mmHg SD ± 17.6), than subjects who did not (120 mmHg SD ± 13.6) or those who did not know, (119 mmHg SD ± 16.8). Sisters' history of high blood pressure showed some association, but the relationship was not significant. Parents history of high blood pressure was not significant.

8.1o Blood Pressure and Ethnicity

There were significant differences in mean systolic blood pressures relating to subjects' place of birth and duration living in the UK.

Birth Place: Table 8.29 below shows that Jamaican-born subjects had the highest mean systolic blood pressures (128 mmHg SD ± 15.1). Eastern-Caribbean born subjects had a mean of 124 mmHg, SD ± 16.5. The British born had the lowest mean systolic blood pressure of 116 mmHg SD ± 11.2.[12] The difference in mean systolic blood pressure between the Jamaican born and the Eastern Caribbean born was not significant, but the difference in means was significant between the combined Caribbean-born and British-born.

Table 8.29 Systolic Blood Pressure by Birth Place

Birth place	Systolic Blood Pressure	SD	Case
UK-born	116.3	11.2	67
Eastern Caribbean	124.0	16.5	27
Jamaican	127.7	15.6	70

Jamaican-born subjects had significantly higher systolic blood pressure than those who were born in the eastern Caribbean and those who are UK-born.

Duration in Britain: Excluding UK-born subjects, there was appeared to be a linear relationship between length of duration in the UK and blood pressure: the longer the duration in Britain the higher the mean systolic blood pressure. Respondents who had been living in the UK for under 21 years had a mean systolic blood pressure of 120 mmHg, SD ± 10.1, compared to 126 mmHg, SD ± 15.9 subjects who had resided for 21-30 years. Those immigrants who had resided in the UK for more than 30 years recorded mean systolic blood pressure of 130 mmHg, SD ± 17.0. Overall, subjects who had not lived in the UK all their lives (non-UK born) had significantly higher blood pressure than their (younger) UK born colleagues.

[12]Note: British born subjects mainly fell in the youngest age group.

8.1p Blood Pressure and Racism

Three racism variables were significantly associated with systolic blood pressure, at Sig = <0.05: anger from racism, effect on career, and racism from colleagues. Overall subjects who reported being most (negatively), affected by racism, had lower mean systolic blood pressure than those subjects who reported that racism had little or no effect.

Racism and anger: Subjects who said that they were most angered by racism, most often, had significantly lower mean systolic blood pressure than those who were not so angered. Table 8.30 below shows that those subjects who were angered on a daily basis, had mean systolic blood pressure of 115 mmHg, SD ± 9.2, while those subjects were angered on a weekly basis had a mean systolic blood pressure of 122 mmHg, SD ± 12.6. Subjects who felt angered only sometimes had a mean systolic blood pressure of 123 mmHg SD ± 16. The members of the group that rarely felt angered by racism had the lowest mean systolic blood pressure of 131 mmHg, SD ± 16.6.

Table 8.30 Anger from Racism and Systolic Blood Pressure

Anger Level	Systolic Blood Pressure	SD	Cases
Rarely	130.7	16.6	16
Sometimes	123.1	16.5	76
Weekly	122.5	12.6	48
Daily	114.7	9.2	24

Anger from racism was inversely associated with systolic blood pressure.

Racism and Career: Respondents who reported that racism had the least effect on their careers had the highest mean systolic blood pressure (126 mmHg, SD ± 17.6). Those subjects who were not sure of the effect of racism on their careers, recorded overall mean systolic blood pressures of 124 mmHg, SD ± 12.2. Subjects who had

the lowest mean systolic blood pressure (118 mmHg, SD ± 10.9), felt that racism had adversely affected their careers quite a bit.

Racism from Colleagues: Respondents who said they had experienced the least amount of racism from colleagues had higher mean systolic blood pressure (124 mmHg, SD ± 16.3) than those who had experienced a greater amount of racism from their work colleagues, whose mean was 118 mmHg SD ± 11.3.

SUMMARY: BLOOD PRESSURE AND INDEPENDENT VARIABLES

Age had the strongest association with blood pressure. When analysed as a continuous variable, **body mass index** was not significant, but when grouped it was. **Heart rate** was not significant. There was a negative association with **adding salt**. No significant association was found with **cooking with salt**. Generally, **dietary habits** did not have significant associations with blood pressure. The association with **alcohol** was not significant. There was a significant negative association with **smoking**. Although smokers had significantly lower systolic blood pressure than non-smokers, **heavy smokers** had a higher mean systolic blood pressure than their fellow smokers who smoked less heavily. However, the higher systolic blood pressure of heavier smokers was lower than that of their non-smoking colleagues. **Physical activity** was not significant. **Job grade** and **education** had negative associations, whereby the lowest job grade and the lowest education levels had the highest blood pressures. **Accommodation satisfaction** had a positive association, whereby those who were most satisfied had the highest blood pressure. **Accommodation crowding** showed significant inverse association, such that those subjects with the highest levels of crowding recorded the lowest blood pressures. Of

the **Family blood pressure** history variables, only **brother's** history of high blood pressure was (positively), significant. **Ethnicity** was significant, in that where **subjects were born** and **duration living in the UK** revealed that UK born subjects had lowest blood pressure, while Caribbean-born subject who had resided in the UK longest had highest blood pressure. **Racism** was overall negatively significant, as those who reported being most (negatively) affected by racism, generally had lower blood pressure than those on whom racism had a lesser effect

8.1q Adding Salt and Other Variables

The relationship of adding salt with other independent variables was examined.

ADDING SALT AND BLOOD PRESSURE HISTORY

There was a weak **negative** (Spearman's) correlation between adding salt and history of high blood pressure, r = **-0.16,** Sig 0.03). The Spearman's correlation of adding salt with history of hypertension was stronger, r = **-0.20**, Sig 0.01). Cross-tabulation analysis showed that a significant number of participants, who reported having had a history of high blood pressure, or a history hypertension, also reported that they generally never added salt.

Adding Salt and Low Blood Pressure: **No** significant association was found between adding salt and history of low blood pressure. Respondents who reported having had a history of low blood pressure had very similar adding salt habits to those who did not have a history of low blood pressure. However, a greater percentage of the no low blood pressure history subjects never added salt (48% versus. 20%), but the difference was not significant.

Adding Salt and Parents' Blood Pressure: No significant association was found between adding salt and subjects' parents history of hypertension. However, a

greater percentage (14%), of the subjects whose mother had a history of hypertension always added salt, than the subjects whose mother did not have a history of hypertension (5%). Just under half, (44%) of the subjects whose mother had a history of hypertension never added salt.

Adding Salt and General Diet: Persons who generally ate healthy meals did not show significantly different adding salt habits to individuals who did not habitually eat healthy meals. No significant association was found between red meat eating habits and adding salt. A higher percentage of people ate heavily seasoned foods than did not, but the adding salt habits were similar (that is, no significant difference) between the two groups. However, generally, those respondents who did not eat heavily seasoned foods also tended not to add salt

Adding Salt and Body Mass Index: A weak significant negative correlation was found in both Pearson's and Spearman's correlation of r = **-0.16**: Sig=0.03, between body mass index and adding salt. Participants in the higher body mass index category were slightly less likely to always add salt than their counterparts in the lower groups. Fifty-five percent of those in the heaviest body mass index group never added salt, compared to 34.4% of those who were in the lightest group.

Adding Salt and Alcohol: There was a weak association between adding salt and alcohol habits. Approximately half (56%), of the non- drinkers never added salt, compared to one-third (33%), of the heaviest drinkers. Seven-percent (6.6%), of the non-drinkers always added salt, compared to eleven-percent (11%), of the heavy drinkers. In general, alcohol drinkers were more likely to add salt than non-alcohol drinkers were or those who drank only occasionally.

Adding Salt and Smoking: A greater percentage of the smokers than the non-smokers always added salt, but the difference was not significant.

Adding Salt and Physical Activity: The subjects physical activity level was not associated with their adding salt habits. There was no significant difference between the salt adding habits of physically active persons and those of low physical activity levels.

SUMMARY: SALT WITH OTHER HEALTH BEHAVIOUR VARIABLES

Associations were found between adding salt subjects' hypertension history, body mass index, and alcohol consumption. Other associations found were not significant.

Note: *It is worth noting that a greater percentage of Jamaican born subjects never added salt than subjects born in the other islands, or subjects born in the UK. Generally, the UK born African-Caribbeans seemed to consume more salt than non-UK born subjects.*

8.2 MULTIVARIATE ANALYSIS: STAGE ONE

Stage one multivariate analysis involved more detailed analyses than those carried out earlier. The aim was to pin-point more accurately the independent variables, in particular the lifestyle factors, that were the potential partial regression coefficients to be used in the construction of multiple linear regression equations. Variables, which, in earlier analysis, had shown no significance, were not included in the detailed regression analysis. However, in an effort to be exhaustive, variables that were just outside the required significance of Sig= 0.05 were included.

8.2a Linear Regression and 'Dummy Variables'

Descriptive and exploratory statistics had confirmed that the dependent variable (systolic blood pressure) was normally distributed, which validated the use of the robust linear regression analysis. Where necessary independent variables were converted into ordinals, and further into 'dummy variables' to facilitate use in linear regression analysis. Coding was ordinal, that is, from lowest to highest, or vice versa. For most cases, when using dummy variables for analysis, the group within the variable that had appeared to have the lowest risk, based on the earlier analysis was used as the baseline group. Otherwise, the group with the lowest 'expected' risk, based on existing literature, was used as the baseline group.

8.2b Body Mass Index

Body mass index had been measured on a continuous scale, and a univariate analysis was carried out. The results represented in output table 8.31 below shows that when

body mass index was analysed in its original state, that is, as a continuous variable, it was not a significant predictor of systolic blood pressure in the subjects.

Table 8.31 Regression: Systolic Blood Pressure and Body Mass Index

Regression output of Systolic Blood Pressure and Body Mass Index

		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	111.086	5.951		18.665	.000	99.333	122.84
	BMI	.412	.212	.151	1.945	.054	-.006	.830

Body mass index as a continuous variable was not significantly related to systolic blood pressure.

However, as seen in table 8.32 below, body mass index was a significant predictor of systolic blood pressure when it was analysed as a grouped variable. The Beta-value of **0.221**, which converts to a R^2 of **0.049**, suggests that 5% of the variability in the systolic blood pressures of the subjects could be explained by their body mass indices, at Sig = 0.005.

Table 8.32 Regression: Systolic Blood pressure and Body mass index – Grouped

Regression Output of Systolic Blood Pressure and Body Mass Index - Grouped

		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	114.361	3.030		37.740	.000	108.377	120.344
	BMI - Grouped	3.270	1.136	.221	2.879	.005	1.027	5.513

Body mass index – grouped was significantly related to systolic blood pressure, Beta value = .221.

When age was entered into the regression analysis, body mass index, on the as a continuous variable was significant. This is shown in output table 8.33 below. The result with age was similar, but more significant when it was combined with body mass index grouped, yielding a Multiple R of 0.46, and 0.48 respectively.

Table 8.33 Regression: Systolic Blood Pressure and Body Mass Index with Age

Regression Output of Systolic Blood Pressure with BMI and Age

Model		Unstandardized Coefficients	Standardized Coefficients	t	Sig.	95% Confidence Interval for B	
		Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	7.233		11.159	.000	66.429	94.997
	BMI	.191	.172	2.453	.015	.091	.845
	AGE	.121	.437	6.248	.000	.516	.992

With age as a control, body mass index – grouped remained significant.

8.2c Adding Salt

Adding salt had proved to the only significant dietary factor, but indications were that the relationship was negative. Univariate regression analysis confirmed this negative association, as shown in table 8.34 below.

Table 8.34 Regression: Systolic Blood Pressure with Adding Salt

Systolic Blood Pressure with Adding Salt

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	127.034	1.621		78.36	.000	123.832	130.235
	Sometimes	-9.031	2.384[a]	-.298	-3.789	.000	-13.739	-4.324
	Always	-4.115	1.926	-.168	-2.136	.034	-7.919	-.311

a. R Square = .08 Baseline Group is: Never add Salt

Adding salt negtively associated with systolic blood pressure.

The regression output table 8.34 revealed that the predicted systolic blood pressure for a respondent in the baseline (never added salt) was 127 mmHg. The b-coefficient in 'dummy variables' represents the difference in average systolic blood pressures between the groups. Thus, compared to the reference group, those respondents who sometimes added salt, and those who always added salt had predicted systolic blood pressures of 9 mmHg, and 4 mmHg (respectively) lower. Correspondingly, the Beta-values for both sometimes and always adding salt were negative, indicating a negative correlation between adding salt and blood pressure. When analysis was carried out with age as a control, the results did not change significantly, in that adding salt remained negatively significant with systolic blood pressure.

8.2d Alcohol

Although earlier analysis did not suggest a relationship between alcohol intake and systolic blood pressure, regression analysis was carried out in the interest of exhaustive probing. The alcohol variables examined were: drink alcohol; amount drink weekly; age started drink. Both univariate and multivariate analysis found that overall, no alcohol variable was independently significant. The closest indications of probable associations between blood pressure and alcohol, was with age started drinking, but it was not statistically significant. Controlling for age did not significantly change the results.

8.2e Smoking

The relevant variables were: smoker, ever smoked, amount smoke, age started smoking. Univarate analysis was carried out on each smoking variable, using non-smoker as the baseline.

Smoker: A significant (negative) relationship was found with those subjects who were smokers (past and present). Controlling for age did not significantly change the results. Output table 8.35 below shows that compared to subjects who did not smoke, smokers had lower mean systolic blood pressures by 6 mmHg, C.I.= -11.55 to –1.01, at .Sig =0 02.

Table 8.35 Regression: Systolic blood pressure and smoking

Regression Output of Systolic Blood Pressure and Smoking

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	124.005	1.334		92.969	.000	121.371	126.639
	Smoker	-6.278	2.668	-.182	-2.353	.020	-11.546	-1.010

a. R Square = .033. Baseline Group = non Smoker

Univariate analysis derived a negative Beta value between smoking and systolic blood pressure.

Ever smoked: A negative Beta value, and the B-coefficient indicated that subjects who had never smoked (baseline group) had an average systolic blood pressure of 7 mmHg higher than those subjects with a smoking history. Output table 8.36 below gives the details, showing that this result was significant at 0.001, C.I.= -12.1 to –2.98.

Table 8.36 Regression: Systolic blood pressure and ever smoked

Regression Output of Systolic Blood Pressure with Smoking - Ever Smoked

Model		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	125.436	1.465		85.630	.000	122.543	128.329
	Smoked Ever	-7.569	2.327	-.248	-3.253	.001	-12.164	-2.975

a. R Square = .06 Baseline group = Never smoked

Univariate analysis derived a negative Beta value between ever smoked and systolic blood pressure.

Age started smoking: Subjects who started smoking at the youngest age, had on average systolic blood pressure 8 mmHg (significantly) lower than those who were non smokers. Those who started at older had lower systolic blood pressure than non smokers by 2, a difference which not statistically significant. Output table 8.37 below shows the significance of the margin between the non-smokers and those who started at the youngest age to be Sig = 0.011, at C.I. = -14.3 to –7.89.

Table 8.37 Regression: Systolic Blood Pressure and Age Started Smoking

Regression Output of Systolic Blood Pressure and Smoking: Age Started Smoking

Model		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	124.536	1.373		90.714	.000	121.825	127.247
	Under 18	-8.104	3.148	-.201	-2.574	.011	-14.322	-1.887
	Over 18	-2.855	1.713	-.130	-1.667	.097	-6.238	.527

a. R Square = .048. Baseline Group = non smoker

Starting smoking at a young age did not contribute to raised systolic blood pressure.

Table 8.38 Regression: Systolic Blood Pressure With Amount Smoked Daily

Regression Output of Systolic Blood Pressure and Smoking: Amount Smoked Daily

Model		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	124.248	1.368		90.801	.000	121.546	126.950
	< 10 Daily	-7.115	3.029	-.184	-2.349	.020	-13.096	-1.133
	> 10 Daily	-2.462	1.921	-.100	-1.282	.202	-6.255	1.331

a. R Square = .038 Baeline group = non smoker

Univariate analysis derived a negative Beta value between amount smoked daily and systolic blood pressure.

Amount Smoke Daily: Table 8.38 above shows that subjects who smoked less than 10 cigarettes daily had lower mean blood pressures by 7 mmHg than non smokers, (Sig = 0.020, C.I.= -13.1 to –1.1). Those who smoked ten or more had lower mean systolic blood pressure than non-smokers did, but the difference was not significant (Sig = 0.202). Subjects who smoked more than 10 cigarettes daily had higher mean systolic blood pressure than their fellow smokers who smoked less.

When age was entered into the analysis, amount smoked lost significance with systolic blood pressure.

8.2f Physical Activity

Regression analysis confirmed that no physical activity variable was independently significant. With age as a control, the results were not significantly different.

8.2g General Health

Confirmations of earlier findings were made, as regression analysis did not fine health to be significant, with or without age in the analysis.

8.2h Accommodation

Adequate: Output table 8.39 below confirms earlier findings that respondents who thought their accommodation was very adequate had an average systolic blood pressure 9 mmHg higher that the less than adequate group. This difference was highly significant at Sig = 0.000, C.I.= 4.2 to 13.7. With age in the analysis the difference remained significant, but reduced to Sig = 0.03. However, the F-statistics remained relatively high at 20.7, Sig = 0.000).

Table 8.39 Regression: Systolic Blood Pressure and Accommodation Adequacy

Regression Output of Systolic Blood Pressure and Accommodation Adequate

Model		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	116.441	1.965		59.265	.000	112.562	120.321
	Adequate	8.937	2.399	.281	3.725	.000	4.200	13.675

a. R Square = .079. F-Statistic = 13.8, Sig = .000 Baseline Group = Very adequate

Univariate analysis derived a positive Beta value between accommodation adequacy and systolic blood pressure.

Table 8.40 Regression: Systolic Blood Pressure and Accommodation Crowding

Regression Output of Systolic Blood Pressure and Accommodation Crowding

Model		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	117.976	2.281		51.725	.000	113.472	122.480
	Medium Crowding	2.328	1.421	.155	1.638	.103	-.479	5.134
	Lowest Crowding	2.736	1.052	.247	2.602	.010	.660	4.813

a. R Square = .040 F-Statics = 3.40,Sig = .36. Baseline group = Highest Crowc

The difference between levels of crowding was significant between the baseline highest-crowding group and the lowest-crowding group, but not with the medium-crowding group.

Crowding: Subjects who lived in the most crowded accommodation had significantly lower systolic blood pressure, by 3 mmHg, Sig = 0.010, C.I = 0.660 to 4.81, than their colleagues in less crowded homes, which is reflected by the positive Beta values, shown in table 8.40 above. However, with age in the analysis, the crowding was no longer significant.

When both accommodation adequacy and accommodation crowding were placed into the regression analysis, only accommodation adequacy proved significant, at Sig = 0.003, C.I.= 2.69 to 12.67. This is shown in output table 8.41 below.

Table 8.41 Regression: systolic blood pressure and Accommodation

Regression Output of Systolic Blood Pressure with Housing Accommodation

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	122.212	4.232		28.876	.000	113.854	130.570
	Adequacy	7.677	2.526	.241	3.039	.003	2.689	12.665
	Crowding	-2.493	1.621	-.122	-1.538	.126	-5.695	.709

a. R Square = .092. F-Statistic = 8.2, Sig = .000. Baseline Groups, Least Adequate & Most Crowding

Accommodation adequacy proved to be of more significance than crowding

8.2i Education

The output table 8.42 below shows that the no qualification baseline group had the highest mean systolic blood pressure of 128 mmHg. Although the more educated subject had lower mean systolic blood pressures than the baseline group, in all cases the differences were not statistically significant. With age in the analysis the results did were not significantly different.

Table 8.42 Regression: Systolic Blood Pressure and Education

Regression Output of Systolic Blood Pressure and Education

Model		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	127.716	2.872		44.463	.000	122.043	133.389
	High School	-6.700	3.307	-.224	-2.026	.044	-13.231	-.169
	Professional	-3.531	2.051	-.173	-1.722	.087	-7.581	.518
	University	-1.503	1.342	-.113	-1.120	.265	-4.153	1.148

a. R Square = .028. F Static = 1.52, @ Sig .120. Baseline group = No education.

Univariate analysis of the qualifications variables revealed that compared to the least educated baseline group (no qualifications), the much better qualified groups did not have significantly different systolic blood pressure.

8.2j Job grade

With lowest job grade as the baseline group, output table 8.43 below shows that the higher job grades had significantly lower blood pressures than 131 mmHg of this baseline group. However, the relationship was not linear as the middle job grade had the most significant difference, (4 mmHg, Sig = 0.005, C.I.= -6.9 to –1.2).

Table 8.43 Regression: Systolic Blood Pressure and Job Grade

Regression Output of Systolic Blood Pressure and Job Grade

Model		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	131.217	3.282		39.984	.000	124.735	137.698
	2nd Lowest	-3.714	2.351	-.159	-1.580	.116	-8.357	.929
	Middle	-4.108	1.432	-.310	-2.868	.005	-6.937	-1.279
	2nd Highest	-2.183	.925	-.291	-2.361	.019	-4.009	-.357
	Highest	-2.675	.897	-.311	-2.981	.003	-4.448	-.903

*. R Square = .066. F-Statistic = 2.80, Sig = .03. Baseline Group = Lowest Job Grade

Univariate analysis of the job grade variables revealed that compared to the least lowest job grade baseline group, the systolic blood pressures of the higher grades were overall, significantly higher.

With age in the analysis, the results were still significant, but with different groups and to different degrees. This is shown in output table 8.44 below.

Table 8.44 Regression: Systolic Blood Pressure with Job Grade and AGE

Regression Output of Systolic Blood Pressure with Job Grade and AGE

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	100.594	6.018		16.716	.000	88.708	112.479
	2nd Lowest	-1.694	2.165	-.073	-.783	.435	-5.970	2.582
	Middle	-2.830	1.320	-.214	-2.143	.034	-5.438	-.222
	2nd Highest	-1.248	.856	-.166	-1.459	.147	-2.939	.442
	Highest	-2.302	.818	-.267	-2.813	.006	-3.919	-.686
	AGE	.721	.123	.418	5.860	.000	.478	.963

Univariate analysis of the job grade variables with AGE, emphasised the significance of the difference in mean systolic blood pressure between the highest and lowest job grades

Table 8.45 Regression: Systolic Blood Pressure, Education and Job Grade

Regression Output of Systolic Blood Pressure and Qualification & Job Grade

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	130.855	3.415		38.315	.000	124.110	137.599
	Qualifications	1.298	1.513	.082	.858	.392	-1.690	4.285
	Job Grade	-3.011	1.177	-.245	-2.559	.011	-5.334	-.687

a. R Square = .043. F-statics - 3.61, Sig = .029. Balseline groups = Lowest Qualifications/Job Grade

Education (qualifications), lost significance when combined with job grade in the regression analysis.

Table 8.45. above shows that when both educational qualifications and job grade were placed into the regression analysis, only job grade was significant, at Sig = 0.011, C.I.= -5.33 to 0.687. Age in the analysis did not change the result.

8.2k Brothers' Blood Pressure History

Of all the family members, that is, parents, grandparents, siblings, only brothers blood pressure history proved to be of significant (F statistic Regression Sig = .003) in regression analysis. Subjects who had a brother with high blood pressure had an average systolic blood pressure of 5.5 mmHg higher had no brothers with high blood pressure. Table 8.46 below shows that this difference was significant at 0.001, C.I.= 2.3 to 8.7. With age in the analysis no significant there were no changes

Table 8.46 Regression: Systolic Blood Pressure with Brothers' Blood Pressure

Regression Output of Systolic Blood Pressure and Brothers with High Blood Pressure

Model		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	120.948	1.291		93.671	.000	118.398	123.497
	Don't Know	-1.537	4.237	-.028	-.363	.717	-9.905	6.831
	YES	5.502	1.619	.260	3.398	.001	2.304	8.700

a. R Square = .07 Baseline Group = No Brother with high BP

Univariate analysis of brothers blood pressure with systolic blood pressure revealed that compared to the baseline group who did not have a brother with high blood pressure, the Yes group had significantly higher systolic blood pressure.

8.2l Birth Place

Compared to UK-born subjects, Caribbean born respondents recorded significantly higher systolic blood pressure, by 4 mmHg. The significance was stronger with Jamaican born respondents, Sig = 0.000, C.I.= 2.21 to 5.39. However, with **age** in the analysis the significance was lost. This is shown in table 8.47 below.

Table 8.47 Regression: Systolic Blood Pressure and Birth Place

Regression Output of Systolic Blood Pressure by Birth Place

Model		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	116.296	1.726		67.381	.000	112.888	119.704
	East Caribbean	3.877	1.610	.192	2.408	.017	.697	7.057
	Jamaican	3.798	.805	.377	4.719	.000	2.209	5.388

* . R square = .124. F-Statistics = 11.3, Sig = .000. Baseline Group = UK-Born

Univariate analysis of birthplace with systolic blood pressure revealed that compared to the baseline UK-born group, Caribbean-born subjects, especially Jamaicans had significantly higher systolic blood pressures

Duration Lived in UK: The period of residence in the UK also yielded the same results as the country of birth factor. Thus, the difference was only significant when age was not a considered factor.

8.2m Racism

Anger: The regression output in table 8.48 below shows a significant difference in mean systolic blood pressure between those subjects who were most angered by racism and those who were least angered, F-Statistics = 4.1 at Sig = 0.009. A subject who was most angered by racism (daily) could be expected to have on average systolic blood pressure of 5 mmHg lower than a colleague who was least angered (rarely). When **age** was a considered factor, significance remained only between the two extremes, that is, least and most angered, with a C.I. of –7.25 to –1.56, Sig = 0.003.

Table 8.48 Regression: Systolic Blood Pressure and Racism: Anger

Regression Output of Systolic Blood Pressure with Anger from Racism

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	130.698	3.650		35.808	.000	123.490	137.906
	Angry Sometimes	-7.619	4.016	-.254	-1.897	.060	-15.550	.312
	Angry Weekly	-4.085	2.107	-.249	-1.939	.054	-8.247	.077
	Angry - Daily	-5.330	1.571	-.378	-3.393	.001	-8.432	-2.228

a. R-square = .07. F-statistics = 4.1, Sig = .009. Baseline group = rarely angered

Univariate analysis of anger from racism with systolic blood pressure revealed that compared to the baseline rarely angered group, those who were most angered would be predicted to have significantly lower systolic blood pressure.

Career: Table 8.49 below shows that subjects who felt that their careers had been most adversely affected by racism (quite a bit) had and average systolic blood pressure of 4 mmHg, Sig = 0.002, C.I.= -7.0 to –1.5. The result was the same with **age** in the analysis

Table 8.49 Regression: Systolic Blood Pressure and Racism: Career

Regression Output of Systolic Blood Pressure and Racism: Career

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	126.918	2.132		59.534	.000	122.708	131.128
	Not Sure	-2.748	3.031	-.083	-.907	.366	-8.734	3.238
	Quite a Bit	-4.287	1.374	-.284	-3.119	.002	-7.000	-1.573

a. R-Square = .062. F-Statistic = 5.3, Sig = .006. Baseline group = Little or No Effect

Univariate analysis of effect of racism on career with systolic blood pressure revealed that compared to the baseline little or no effect group, those whose careers were most affected would be predicted to have significantly lower systolic blood pressure.

Colleagues: Similar results to those found on the effect of racism on career were derived for racism from colleagues. Thus, subjects who reported that they had suffered the most racism from colleagues recorded on average 6 mmHg (Sig = 0.016) lower systolic blood pressure than those respondents who had suffered little or no racism from colleagues. However, the significance was lost when age was entered into the regression equation.

When all the racism variables, anger from racism, effect of racism on career, and racism from colleagues were placed into a single regression analysis, racism from colleagues was not significant. Table 8.50 below shows the effect of racism on career had the strongest significance, with C.I.= -6.1 to –0.60.

Table 8.50 Regression: Systolic Blood Pressure and Racism

Regression Output of Systolic Blood Pressure with Racism Variables[a]

Model		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	131.473	2.512		52.3	.000	126.512	136.434
	Angry	-2.866	1.409	-.165	-2.0	.044	-5.649	-.082
	Career	-3.360	1.396	-.188	-2.4	.017	-6.117	-.603
	Colleagues	-2.750	2.569	-.087	-1.1	.286	-7.823	2.323

[a] R Square =.101. F-Statistics = 6.0, Sig = .001. Baseline Groups = least affect by racism

Multivariate analysis of racism with systolic blood pressure revealed that compared to the baseline (least affected) groups, those who were most affected by racism would be predicted to have significantly lower systolic blood pressures.

When **age** was also added to the analysis, only effect of racism on career proved to be significant, Sig = 0.012, C.I.= -5.71 to -0.71.

SUMMARY: FIRST STAGE REGRESSION ANALYSIS

Results from the first stage multivariate analysis of key variables from the different categories in this study revealed that:

Dietary habits in general were not significantly associated with blood pressure. The association with ***salt intake*** was significant, but negative.

Alcohol intake was not significantly associated with blood pressure.

Smoking was significantly associated with blood pressure, but negatively. Smokers did not have higher blood pressures than non-smokers but rather generally had lower average systolic blood pressure. However, heavy smokers had higher systolic blood pressure than those who smoked less, but the difference was no significant. ***Smoking history*** (ever smoked) also had a significant negative association with blood pressure, whereby those subjects with a smoking history had lower blood pressure than those without.

Physical activity level did not have a significant effect on blood pressure.

Apart from past history of ***raised blood pressure, Health factors*** were not associated with blood pressure.

Family blood pressure: only brothers' history of high blood pressure was significantly associated with the subjects own blood pressure.

Birthplace and duration living in the UK were significant. Caribbean born subjects had higher blood pressures than the UK-born participants, and that Caribbeans who had resided in the UK longest, had the highest blood pressure. However, **age** was a significant, (confounding) factor, which reduced or eliminated the difference.

Accommodation: The association was negatively significant. Subjects in ***more crowded*** or ***less than adequate*** tended to record lower mean blood pressures than

their more comfortably house colleagues. The association with blood pressure was strongest with accommodation adequacy.

Qualifications and Job grade: The most qualified and the lowest qualified subjects had higher blood pressure than those did who fell in the mid range, with the least qualified having the highest average blood pressure. ***Job grade*** was significantly associated with blood pressure. Subjects at lower job grades were more likely to have higher blood pressure than respondents in the higher grades. Job grade had a stronger association with blood pressure than qualifications.

Racism: Significant negative associations were found. In general, subjects who reported that they were most (negatively) affected by racism, recorded lower blood pressures than those who said that racism had very little adverse effect.

8.3 FINAL REGRESSION ANALYSIS

This final stage analysis aimed to derive a final predictive model from those variables that had proven significant to the systolic blood pressure values of the respondents. Prior analysis had revealed that age was the strongest predictor variable (Pearson's r=4.4, Sig = 0.000). Age is standardly used as a control in social science research, and earlier analysis qualified age in this respect.

ADDING SALT AND BODY MASS INDEX

When modelling was carried out using the independent variables adding salt and body mass index (grouped), a Multiple R of 0.317 was established, with both variables showing significance at less than the required 0.05. Adding salt was negatively significance, at Sig = 0.003, C.I. being –8.55 to –1.83. Body mass index was positively significant, at Sig = 0.017, C.I.= .496 to 4.93. The regression analysis output is shown in table 8.51 below.

Table 8.51 Regression: SBP with Adding Salt with Body Mass Index

Regression Output of Systolic Blood Pressure and Adding Salt with BMI

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	118.961	3.319		35.843	.000	112.406	125.515
	Addsalt	-5.188	1.703	-.231	-3.047	.003	-8.550	-1.825
	BMI	2.714	1.123	.183	2.417	.017	.496	4.931

a. R Square = .010. F-Statistics = 9.10, Sig = .000. Baseline Groups = Rarely Addsalt/Lowest BMI

Multivariate analysis with adding salt and body mass index revealed that both were significant predictors of systolic blood pressure.

When age was placed in the analysis, body mass index gained a stronger significance than adding salt, at Sig = 0.005. C.I.= 0.896 to 4.95.

ADDING SALT, SMOKING, BODY MASS INDEX

The regression analysis with adding salt, ever smoked and body mass index, revealed that all three factors were significant when combined. Output table 8.52 below shows that of the three variables, body mass index was the least significant, with a t-value of Beta-value of .166, Sig = 0.027.

Table 8.52 Regression: SBP with Add Salt, Body Mass Index and Smoking

Regression Output of Systolic Blood Pressure with Addsalt, BMI & Smoking

Model		Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	121.657	3.405		35.729	.000	114.933	128.382
	Addsalt	-4.608	1.684	-.205	-2.736	.007	-7.934	-1.282
	BMI	2.462	1.105	.166	2.227	.027	.279	4.645
	Ever Smoked	-6.145	2.272	-.201	-2.705	.008	-10.633	-1.658

a. R Square = .141. F-Statistic = 8.67, Sig = .000. Basline Groups = Rarely Addsalt, Lowest BMI. Never Smoked

Adding salt, body mass index and ever smoked together were all significant regressors to systolic blood pressure.

However, with age in the analysis, body mass index was the most significant, with a Beta-value of 0.185, Sig = 0.008. At the same time, ever smoked became less significant, at Sig = 0.047, and adding salt was no longer significant.

ADDING SALT, SMOKING, BODY MASS INDEX ACCOMMODATION

When accommodation ***crowding*** was included with adding salt, ever smoked, and body mass index all variables in were significant. Body mass index had the lowest

significance at Sig = 0.019. When was entered into the analysis, body mass index became the most significant variable, Sig = 0.004, but ever smoked lost significance.

Table 8.53 Regression: SBP with Add salt, BMI, Smoking and Crowding

Regression Output of Systolic Blood Pressure and Adding Salt, BMI, Smoking, & Crowding

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	112.989	4.541		24.880	.000	104.020	121.958
	Addsalt	-4.632	1.649	-.206	-2.809	.006	-7.889	-1.376
	BMI	2.565	1.083	.173	2.369	.019	.426	4.704
	Eever Smoked	-5.952	2.226	-.195	-2.674	.008	-10.348	-1.556
	Crowding	4.126	1.468	.202	2.811	.006	1.227	7.024

a. R Square = .181. F-Statitics = 8.76. Baseline groups = Rarely addsalt, lowest BMI, never smoked, highest level of crowding.

All the variables were significant regressors.

Accommodation adequacy was added to the analysis (with adding salt, ever smoked, and body mass index). All variables, except ever smoked, were significant, with accommodation adequacy having the strongest Beta-value of 0.26, Sig T = 0.000. Age in the analysis made body mass index the most significant, Beta-value of 0.199, Sig = 0.004 and ever smoked lost significance.

With both accommodation adequacy and accommodation crowding in the analysis, crowding lost significance, and ever smoked was on the crux, at Sig = 0.050.

When age was entered into the analysis, both accommodation variables lost significance, along with ever smoked. Body mass index and adding salt maintained significance.

The variables add salt, body mass index, ever smoked, accommodation adequacy, accommodation crowding, and job grade were placed into a single regression analysis. Together all variables were significant, yielding an F-statistics of 10.0, Sig = 0.000. Job grade was the most significant, with a Beta value of 0.25, Sig = 0.001, followed by accommodation adequacy.

Table 8.54 Regression: Salt, BMI, Smoking, Accommodation, Job and AGE

Regression Output of Systolic Blood Pressure with Adding salt, BMI, Accommodation, Job Grade and AGE

Model		Unstandardized Coefficients		Standard Coeffic.			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	86.272	6.240		13.827	.000	73.947	98.597
	Addsalt	-3.200	1.529	-.142	-2.093	.038	-6.220	-.180
	BMI	2.725	.990	.184	2.752	.007	.769	4.682
	Ever Smoked	-3.863	2.099	-.126	-1.841	.068	-8.009	.283
	Accom. Adequate	5.359	2.344	.168	2.286	.024	.729	9.989
	Accom. Crowding	2.281	1.467	.112	1.555	.122	-.617	5.179
	Job Grade	2.487	.838	.202	2.968	.003	.832	4.143
	AGE	.476	.126	.276	3.764	.000	.226	.726

Ever smoked and accommodation crowding were non-significant.

Table 8.54 above shows the output when age was added to the analysis, Ever smoked and accommodation crowding lost significance. Job grade and body mass index were the most significant variables.

BROTHERS' BLOOD PRESSURE AND BIRTH PLACE

Added to the model, brothers' blood pressure subjects' birth place were significant. This is shown in table 8.55 below.. However, when age was included in the analysis, birth place, along with a number of other variables (adding salt, ever smoked, and crowding) lost significance.

Table 8.55 Regression: Lifestyle Variables With Brothers' Blood Pressure and Birthplace.

Regression Output of Systolic Blood Pressure with Adding salt, BMI, Accommodation, Job Grade and Brothers' BP and Birth Place

Model		Unstandardized Coefficients		Standard Coeffic.			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	93.490	5.344		17.494	.000	82.933	104.046
	Addsalt	-2.749	1.544	-.122	-1.781	.077	-5.799	.300
	BMI	2.630	.991	.177	2.653	.009	.671	4.588
	Ever Smoked	-4.107	2.085	-.134	-1.970	.051	-8.225	.011
	Accom. Adequate	5.950	2.305	.187	2.581	.011	1.397	10.503
	Accom. Crowding	2.768	1.436	.136	1.927	.056	-.069	5.604
	Job Grade	2.715	.827	.221	3.283	.001	1.082	4.349
	Brothers' BP	3.063	1.374	.149	2.229	.027	.349	5.777
	Birthplace	3.913	1.137	.238	3.443	.001	1.668	6.158

a. R Square = .346. F-Statistic = 10.24, Sig = .000 Baseline groups = rarely add salt, lowest BMI, never smoked, accommodation less than adequate, highest crowding, lowest job grade, Brother high BP = No, UK-born.

Both brothers' blood pressure and the subjects' birth place were significant to the model, when age was excluded.

RACISM

When the ***Racism*** variables, anger from racism and effect of racism on career were entered they were significant to the group. The result with the racism variables, along with all the other significant variables is shown in the regression output table 8.56 below.

Table 8.56 Regression: All Significant Variables in the Enter Method Model

Regression Output of Systolic Blood Pressure with Adding salt, BMI, Accommodation, Job Grade and Brothers' BP, Birth Place and Racism[a]

Model		Unstandardized Coefficients		Standard Coeffic.			95% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	114.495	5.268		21.734	.000	104.088	124.903
	Addsalt	-2.871	1.486	-.128	-1.932	.055	-5.806	.064
	BMI- grouped	2.634	.958	.178	2.750	.007	.742	4.526
	Ever Smoked	-3.720	1.999	-.122	-1.861	.065	-7.669	.229
	Accom. adequate	5.603	2.214	.176	2.531	.012	1.229	9.976
	Accom.Crowding	2.708	1.375	.133	1.969	.051	-.009	5.425
	Job Grade	-1.789	.835	-.145	-2.143	.034	-3.437	-.140
	Brothers BP	3.283	1.317	.159	2.492	.014	.680	5.885
	Birthplace	3.897	1.091	.237	3.572	.000	1.742	6.053
	Racism:Angry	-2.650	1.173	-.152	-2.260	.025	-4.967	-.334
	Racism: Career	-3.211	1.160	-.179	-2.767	.006	-5.503	-.919

a. CR Square = .415. F-Statistic = 9.81 Sig = .000 Baseline groups = rarely add salt, lowest BMI, never smoked, accommodation less than adequate, highest crowding, lowest job grade, Brother high BP = No, UK-born, Least affected by Racism

All the significant variables in the regression model, enter method, but without age. Add salt, ever smoked and crowding were not significant regressors. The most significant variable was birthplace, followed by anger from racism and body mass index.

With age in the analysis, see table 8.57 below, adding salt, ever smoked, accommodation crowding remained non-significant, along with job grade. The only remaining significant variables along with age, were body mass index, accommodation adequate, brothers' blood pressure history, birthplace, and anger from racism, and effect of racism on career. Of these significant variables, body mass index was the most significant, with a Beta value of 0.179, Sig = 0.006, C.I.= 0.784 to 4.52. The effect of racism on career was the second most significant variable. Age lost the strength of significance that it had shown in earlier stages of the analysis, now showing a Beta value of 0.183, Sig = 0.022, C.I.= 0.046 to 0.585.

Table 8.57 Regression: All Significant Variables with AGE – Enter Method

Regression Output of Systolic Blood Pressure with Adding salt, BMI, Accommodation, Job Grade and Brothers' BP, Birth Place Racism, and AGE.

Model		Unstandardized Coefficients		Standard Coeffic.	t	Sig.	95% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	105.710	6.439		16.417	.000	92.989	118.432
	Addsalt	-2.526	1.473	-.112	-1.715	.088	-5.435	.384
	BMI- grouped	2.650	.944	.179	2.806	.006	.784	4.516
	Ever Smoked	-3.333	1.978	-.109	-1.684	.094	-7.241	.576
	Accom. adequate	4.706	2.217	.148	2.123	.035	.326	9.087
	Accom.Crowding	2.062	1.385	.101	1.489	.139	-.674	4.798
	Job Grade	-1.527	.831	-.124	-1.839	.068	-3.169	.114
	Brothers BP	3.175	1.300	.154	2.443	.016	.607	5.743
	Birthplace	2.506	1.233	.153	2.032	.044	.069	4.942
	Racism:Angry	-2.600	1.156	-.149	-2.248	.026	-4.885	-.315
	Racism: Career	-3.102	1.145	-.173	-2.709	.008	-5.364	-.839
	AGE	.315	.137	.183	2.309	.022	.046	.585

With age in the analysis with all the 'final' variables, showed body mass index and effect of racism on career to be the most significant variables. However, both the racism variables yielded negative Beta values.

8.3b The Final Model

The final model was developed using Forward Stepwise Method regression analysis, with selection criteria for entry and removal of set at 0.05 and 0.06 respectively. This method ensured that only the strongest significant variables would contribute to the model as the partial regression coefficients. Stepwise regression output table 8.58 below shows the coefficients (variables) that made up the multiple linear regression equation with **age** being the strongest with a R-Square of 0.18. Age is followed by effect of **racism on career**, **body mass index**, **anger from racism**, **accommodation adequacy**, and **adding salt**. The model resulted in a Multiple R of 0.601, R Square = 0.361, adjusted R Square = 0.336.

Table 8.58 The Variables that made up the Final Model.

Regression - Stepwise Method of final Significant Variables

Model	Variables Entered	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	AGE	.429	.184	.179	13.59
2	Racism: Career	.485	.235	.226	13.20
3	BMI- grouped	.527	.277	.264	12.87
4	Racism:Angry	.554	.307	.289	12.64
5	Accom. adequate	.580	.337	.316	12.41
6	Addsalt	.601	.361	.336	12.22

The table shows the final model variables resulting from Stepwise Analysis. The variables are listed by strength order. Brothers' BP was not included in the analysis.

The variables that made up the final model following stepwise analysis. The variables are listed by strength order, therefore, Age proved to be the strongest regressor, and adding salt, the weakest. Brothers' blood pressure was (deliberately) not included in the analysis.

Table 8.59 below gives further details of the final model from which an algorithm can be derived. From the algorithm will be derived a prediction formula, based on the factors (regressors) in the model.

The standardised Beta coefficients confirms **age** to as the strongest predictor variable, with a value of 0.313, at Sig = 0.000, C.I.= 0.304 to 0.775. **Body mass index** is second in strength, adding salt being the lowest. **Racism** and **adding salt** resulted in negative B-coefficients. Since in each of these variables, the variables were ordinal, with absence of a factor, or the lowest level of the factor as the baseline that is, starting value, the negative B coefficients indicated inverse relationships.

Table 8.59 The Final Model Regression:

Variable	Unstandardised Coefficients		Standardised Coefficients			95% Confidence Interval for B	
	B	Std. Err	Beta	t	Sig	Lower Bound	Upper Bound
Age	.540	.119	.313	4.518	.000	.304	.775
Racism:career	-3.133	1.184	-.175	-2.647	.009	-5.471	-.795
BMI – Grouped	3.062	.970	.207	3.158	.002	1.147	4.978
Racism:angry	-3.126	1.154	-.180	-2.709	.008	-5.405	-.846
Accom. Adequate	6.063	2.165	.191	2.801	.006	1.787	10.338
Add Salt	-3.626	1.494	-.161	-2.427	.016	-6.577	-.674
Constant	100.67	5.839		17.243	.000	89.142	112.206

From the final model, derived by stepwise linear regression analysis, a prediction of subjects blood pressure can be made if the values of the variables within the model are known. Thus, knowing subjects' age, body mass index, adding salt habit, accommodation adequacy, how angered they were from racism, and the effect of racism on their careers, could help to make a substantial prediction of their blood pressure. With this prediction (model) algorithm, approximately 36% of the variability in an individual's blood pressure could be accounted, that is, explained. The algorithm is shown fig. 8.11 below.

Fig. 8.11 Algorithm

Predicted systolic blood pressure =

Constant: 100.7 + (age: * .540) + (Racism: career * -3.1) + (BMI *3.1) + (Racism: angry * -3.2) + (Accommodation Adequate* 6.1) + (Add salt * -3.7).

9 Chapter 9

Discussion of the Findings

9.1 Summary Overview of the Research

The inspiration to embark on this research project came from reading the Department of Health's publication *Ethnicity and Health* - A guide for the NHS, (Balarajan and Raleigh 1993). That publication highlighted the high rates of hypertension (and stroke) in the African-Caribbean population based on mortality statistics for the period 1970 to 1983. It was evident from this 'Guide for the NHS' that African-Caribbeans in general were (still) considered to be a high risk group in this the current decade. Some of the foremost researchers on the topic, including Cruickshank (1993, 1996), and Chaturvedi (1993), have endorsed this position, maintaining that the prevalence of hypertension in African-Caribbeans should be of much concern in the 1990s. Implicit to this conclusion is the view that both the older and the younger generations of African-Caribbeans are to be considered high-risk groups. General indications from the current (although sparse) literature, were that high blood pressure in African-Caribbeans was not perceived as a past generation disease which was only prevalent in the first generation immigrants who arrived in Britain in the 1950's and 1960's. Rather high blood pressure and stroke were deemed current (and future), generation problems. Balarajan and Raleigh (1993), Cruickshank (1993) and others have emphasised the urgent need for further research on hypertension in African-Caribbeans in order to effectively address the problem.

It was on the strength of the informed opinions of the experts in this area of research, and the importance of the subject at government level, (*Health of the Nation*), that this research project was undertaken. An exhaustive literature search on the subject of hypertension in African-Caribbeans revealed that the majority of past (sparse) research did not emphasise the aetiological aspects of the disease. Rather, they were inclined to be epidemiological comparative studies, seeking to examine the differences in blood pressure patterns of African-Caribbeans and other ethnic groups in the UK population. Most frequently, the comparison was made with the indigenous UK (white) population, as part of an examination of the differences in blood pressure patterns between Whites and Blacks.

The concentration of this research on lifestyle aetiological considerations offered a causal-relationship approach, seeking to unearth associations between the blood pressure patterns of a group of African-Caribbeans and their corresponding lifestyles, particularly relating to health behaviour.

A fundamental premise of this research was that, as with other populations, lifestyle could explain a significant amount of the variability in blood pressure in African-Caribbeans, and therefore, offer feasible explanations for the higher rates of hypertension in that ethnic group. Existing researched conclusions from well known studies, such as the JNC V (1993), and the OPCS (1993) reports concerning the independent (positive) association between the variables in this research and blood pressure, formed the basis of the hypothesis, and the expected outcomes. The most recent (at the commencement of this study) national statistics on the aetiological significance of health behaviour in the British population (OPCS 1993) presented

the results of the relationship between alcohol, smoking, BMI, and physical activity on systolic and diastolic blood pressures.[13] These OPCS results were validated indicators of the blood pressure distribution of the British population and possible effects of the named lifestyle factors on blood pressure, and were used as a hallmark reference for the findings of this study. Since the majority of subjects in this study were females, (84%), the OPCS female findings were used as the comparative reference group.

The primary objective of the study was to determine the extent to which lifestyle, in particular health behaviour appeared to influence the blood pressures of the participants of this research. Pre-analysis expectations were that, overall, positive relationships between the degree of risk factor indulgence and blood pressure would be found in the group of African-Caribbeans being researched. The lifestyle factors included in this research were general dietary habits, with special emphasis on salt intake, alcohol consumption, cigarette smoking, and exercise habits. Other factors examined were general health including the subjects' history of high blood pressure, accommodation, job status, education, and racism, among others. A significant (positive) outcome would be indicative of the importance of lifestyle factors to the blood pressure patterns of the group. Negative associations, on the other hand, would be interpreted to mean that health behaviour was not of particular aetiological significance to the blood pressure trends found in the group.

Information for this project was obtained from 164 normotensives defined here to include those persons who were found when measured to have elevated blood

[13] other lifestyle factors analysed by OPCS were not relevant to this study

pressures, (systolic blood pressure >139 mmHg) but who were not taking any form of medication for hypertension. A disproportionate number of the study population were female (84%. The largely female sample was not deemed a disadvantage as hypertension was reported to be significantly more prevalent in the African-Caribbean females than the males, (*Ethnicity and Health* 1993). However, exploratory test determined that both the male and female blood pressure distributions were normally distributed, and differences in the independent variables were not significant enough to be confounding. Males and females were therefore treated as a single population, of 164 subjects, with differences between the groups being highlighted as deemed necessary.

9.1a Repeated measurements

The study involved repeated measurements of blood pressure. Comparison of the repeated measurements showed that the mean blood pressure of the subjects taken at the first session were overall higher than those recorded at session two, but not higher than measurements taken at the third session. However, there was a strong correlation between the (initial) measurements (first session), and the subsequent measurements at sessions two, Pearson's r = 0.740, and session three, Pearson's r = 0.755. This was an important validation tool, and justified use of the blood pressure measurements taken at the first session, (N = 164) for the analysis and model development.

It was reasonable to conclude that those subjects who had repeated measures felt more relaxed with successive measurements, and this could account for lower readings in some subjects. This blood pressure lowering response to successive

measurements is supported by a number of studies, including Rosner and Polk (1981) and MacMahon et al (1990). Further, a number of subjects informed the observer that they did in fact feel more at ease on successive measurements and sessions. Another likely significant factor was increased awareness by the subjects of hypertension risk factors, following consultations with the researcher after the first (initial) session. Some of the respondents might have adjusted their health behaviours, which could have been reflected in progressively lower blood pressures. Research provides evidence that changes in blood pressure following health behaviour modifications could be significant over a relatively short period of time, (JNC V 1993). Cross-referencing of information provided by participants with regard to their progress between sessions, indicated that subjects were positively influenced to improve their health behaviour, with a view to lowering their risks of high blood pressure.

Generally, the group within a variable that would normally be expected to have the lowest relative risk was used as the baseline group during regression analysis. The other groups within the variable were compared to this lowest expected risk (baseline) group. This baseline group would most often be the group with an absence of the factor, or the lowest level within the factor. For instance, in the adding salt category the groups were, never/rarely-added salt, sometimes-added salt, and always-added salt were given ordinal values of 0, 1, and 2 respective. Of these, the rarely added salt group would be expected to have the lowest risk and was used as the baseline group. With the lowest expected risk group as the baseline group, the findings in a number of instances were negative Beta values. These negative results were tantamount to negative associations. In the case for instance, of adding salt,

the negative Beta value meant that compared to the baseline group, never/rarely added salt, the sometimes added salt, and the always added salt groups had, on average, lower blood pressures.

Regression analysis was tiered whereby the simplest univariate analysis were conducted at the earlier stages of analysis, with progression to multivariate modelling. The end result of exhaustive analysis, culminating in multiple regression analysis, and the development of a prediction model, showed that associations found in the simple, early stages analysis, (including cross tabulation analysis) were largely verified.

The robust, but stringently accurate regression analysis, served to examine the variables and their interrelations, and to determine how far from zero the B-coefficients of each independent variable veered. Use of the B-coefficient to test the null hypothesis of this study was prudent, as it would not only establish the existence or non-existence of a relationship, but also calculates the rate of change between the dependent variable (systolic blood pressure), and the independent variables. In the final analysis variables that 'survived' were used to develop the final (predictive) model of the effect or predictive value of the lifestyle factors on the blood pressures of the subjects in this study. The (standardised) Beta coefficient revealed the ultimate strengths of each variable in the model, as this Beta reports the change in standardised value of systolic blood pressure for every one standard deviation change in the standardised value of each of the independent variables.

9.2 THE BLOOD PRESSURE FINDINGS DISCUSSED

Overall, blood pressure was low in this study group. The average systolic blood pressure of this group was 122.4 mmHg, SD ± 15. The figures for the normotensives in the *OPCS* were to 128 mmHg for women and 130 mmHg for men OPCS report (1993). The OPCS defined (systolic), normotensives untreated as: ***"systolic less than 160 mmHg, not currently taking drugs (s) prescribed for high blood pressure"*** and hypertensive untreated as ***"systolic greater than 159 mmHg not currently taking drug(s) prescribed for high blood pressure***" (p.11). In light of the *Ethnicity and Health* (1993), report, and the findings of recent studies on African-Caribbeans, such as the Brent Study (Chaturvedi et al 1993), the relatively low mean blood pressure of the African-Caribbeans in this study was unexpected. It was anticipated that the mean blood pressure of this 'non-medication' group would be markedly higher than that of the corresponding national group, and that the percentages of subjects in the hypertensive range would be higher. In this study, an age related rise in blood pressure was very evident, but generally, the blood pressures of the subjects were relatively low, even taking into consideration the exclusion of diagnosed hypertensives on medication.

The hypertension mortality figures referenced in this study, (Marmot, Adelstein, Bulusu 1984; Balarajan and Bulusu 1990) suggested that the generally low blood pressure of the African-Caribbeans in this study could be considered unrepresentative. However, the low blood pressures found in the subjects of this study are not unique. In the **Birmingham Factory Study**, (Cruickshank et al 1985; Cruickshank 1993) the mean systolic blood pressure for African-Caribbean women

under age 25 was 114 mmHg, and approximately 125 mmHg in the age 20-30 years group. An age related rise in the blood pressure of African-Caribbeans was also found in that study. The **West Lambeth Study,** (Regan and Parry-Cooke 1993) of African-Caribbeans found that the proportion of respondents suffering from high blood pressure was small, (approximately 7% men, and 9% women). Further the vast majority of persons in that survey who recorded above normal blood pressures, were aged over 50 years. In the City and Hackney Hypertension Reduction Project (Trenchard-Mabere 1992), 43% of the subjects were African-Caribbeans. Sixty percent of the African-Caribbeans were over aged 50 years and no African-Caribbean aged 30 and below was found to have raised blood pressure. An age-related rise in blood pressure was also found, with the mean age for people found to have normal blood pressure being 49 years. The mean age of those with raised blood pressure was 61 years. In the report on Coronary Heart Disease: Beliefs Among Caribbeans in West Lambeth (Regan and Parry-Cooke 1993), only a small proportion of respondents (mainly women), had raised blood pressure, and most were over aged 50 years. The Brent study (Chaturvedi et al 1993) reported that African-Caribbeans had significantly higher blood pressures than the Europeans, but subjects in that study were all first generation African-Caribbeans aged 40-64 years.

The OPCS (1993) report did not offer epidemiological statistics on the status of hypertension in this the highest risk group. This was surprising in light of the official government acknowledgement that African-Caribbeans were the highest risk group in the nation. Figures for the high rates of hypertension in African-Caribbeans were derived from mortality statistics for the period 1972-1983. These statistics formed the brunt of the official justification at government level for the

classification of African-Caribbeans as the highest risk group in the UK. In these statistics African-Caribbean were classified based on country of birth (Marmot, Adelstein, Bulusu 1984) and was therefore constrained as they excluded British born African-Caribbeans.

9.3 DEMOGRAPHIC FACTORS

9.3a Healthy Subjects

The self reported information on the subjects' health, showed that they were generally in good health. Twenty-seven percent said they were in very good health; 38% said their health was good; and 35%, felt that their health was fair. Similar percentages were reported from the African-Caribbean samples in the SHARP (Thompson et al 1993) and the West Lambeth (Regan and Parry-Cooke 1993) studies, whose findings will be referred to in this discussion.

9.3b High blood pressure history

Most of the subjects (81%) reported having had their blood pressure checked in the last 2 years. When high blood pressure history was defined to mean having been informed by a medical practitioner that blood pressure was high (including isolated occasions), 34% of the subjects responded positively. Thus, approximately one third of the group had elevated blood pressure at some time in the past. With a distinction being made between high blood pressure and hypertension, the latter being defined to mean sustained high blood pressure, 7% of subjects reported having a history of hypertension. A significantly greater percentage of females than males reported having a history of high blood pressure (34% versus 7%), but the percentages were

largely the same for history of hypertension, that is, 7%. Although the definitions may not be the same as that of this study, the SHARP Study (Thompson et al 1993) reported similar findings. In that study, 13% of its sample of 60 of African-Caribbeans reported having a history of raised blood pressure, most also having had their blood pressure checked in the last two years. This is somewhere between the figure reported for high blood pressure and that for hypertension in this study.

The primary benefit of information on the subjects past history of hypertension was to ascertain their previously 'known' propensity to hypertension, and to determine the correlation between their blood pressure history and their present blood pressure status as recorded in this survey. As expected, subjects' history of raised blood pressure was associated with their current blood pressure status, although there was no association with their general health history. Subjects with a history of raised blood pressure recorded significantly higher blood pressure, than those who had no such history. The mean systolic blood pressure of those subjects with no history of **high** blood pressure was 118 mmHg, compared to 130 mmHg for respondents who reported having had elevated blood pressure at some time in the past. This converted to a (significant) difference of 12 mmHg. The difference in systolic blood pressure for **history of hypertension** was even wider, 121 mmHg for the no history subjects and 142 mmHg for those with a history, a significant difference of 21 mmHg. Despite the significant differences, raised blood pressure history was not used in the multivariate analysis as an independent variable since it was not strictly a lifestyle risk factor. Further, these variables would serve to confound the analysis.

9.3c Familial Hypertension

In general, subjects' family blood pressure was not associated with their own blood pressure. A large percentage of the subjects' mothers (56%) had a history of high blood pressure, but this 'trait' was not generally found in their offspring, the subjects. A significantly lower number of fathers (28%) had a history of high blood pressure. The blood pressures of subjects whose parent(s) had high blood pressure were widely varied. Parents' history of stroke and grandparents' history of hypertension and/or stroke were also not significantly associated with the subjects' own blood pressures. An interesting sibling finding was that while 20% of the subjects had sisters, and 14% had brothers with high blood pressure history, an association (positive) was found only with brothers blood pressure history. It is noteworthy that the association the **brothers' history** of high blood pressure was only significant with the female subjects. Of the 26 male subject, the five who had a brother with a history of high blood pressure, did not have higher blood pressure than the 21 male subjects whose brother(s) had no such history. Instead, the mean systolic blood pressure of the subjects whose **brother(s)** had high blood pressure was lower (118 mmHg) than those whose brothers had no history (120 mmHg). The findings in this study then was that a female subject with a brother who had a history of high blood pressure was more likely to have high blood pressure herself, than if she had a sister with high blood pressure.

A large volume of literature has deemed plausible the familial, usually interpreted as hereditary, link in hypertension, including in western people of Africa descent, (Moll et al 1983; Kaplan 1994, among many others). In light of this, the non-association with family blood pressure history found in this study was surprising. A

strong association with parents' blood pressure history had been anticipated. However, although researchers such as Barley et al (1991) have found links between polymorphism and raised blood pressure in blacks, they have not found any conclusive evidence to support the hereditary/genetic explanation for hypertension in Blacks. Cooper and Rotomi (1994) have opined that such a link has yet to be established, stating that: "***Not a single shred of direct (i.e. molecular) evidence exists to support the contention that higher rates of hypertension among Blacks have a genetic basis"*** (p. 222). Cruickshank (1993) has contended that: ***"The null hypothesis - that genetic differences account for little of the blood pressure differences - remains most likely"*** (1993 p.34). Harrap (1994) pointed out that, families share both genes and environment, and that similarities in blood pressure may result from either. He argued that important environmental differences could alter estimates of heritability because of gene-environment interaction. In the same vein, Cruickshank and Beevers (1989) noted that:

> "***Genetic factors within and between different ethnic groups do have powerful effects. Diabetes, hypertension and coronary heart disease tends to run in families; such familiarity many be partly genetic and partly due to shared environmental influences"*** (p.6).

The results of this study has not positively contributed to the belief that hypertension has an important genetic origin, especially relating to salt sensitivity, in African-Caribbeans. However, the notion, based on a plethora of studies that have found positive associations in western Blacks (Grim 1996), is considered to offer sound explanations, and is in no way dismissed. The views of Swales (1995) and Cooper and Rotimi (1994) and a host of others, that the origin of hypertension may be either environmental or genetic, or most probably some combination of both are duly accepted. It is therefore, possible for a clear familial (genetic), association to be

minimal or not evident in a survey group such as that studied in this research. The author of this thesis does however, consider the environmental influences to be of paramount importance. Such an influence on a particular ethnic group was made evident from the results of the Kenyan Luo Migration study (Poulter et al 1990). This study showed the magnitude of the effect of migration on blood pressure from a low blood pressure rural population that moved to an urban area. Differences in blood pressure between the stable and migrant group were noticeable within a month of the migrant group settling in the urban environment. Poulter and company concluded that the new environment had a significant effect on the shift to the right of the migrants' blood pressure. As one of the 'salt sensitivity' theory proponents noted:

> ***"Debate continues as to whether it is their African ancestry or the Western environment that is more important in increasing the prevalence of hypertension in the African Diaspora above that of the indigenous Africans as well as of fellow inhabitants in the Western hemisphere"*** (Grim 1996, abstract).

9.3d Ethnicity Factors

An African-Caribbean in this study was a person of African origin, born in the Caribbean, or with at least one parent born in the Caribbean. In this study, ethnicity was largely defined by country of birth that is, **Caribbean-born** and **UK-born** subjects. In some instances, Caribbean-born subjects were sub-divided into Eastern-Caribbeans and Jamaican (western Caribbeans) as there was a disproportionate number of Jamaican respondents compared to the other (Eastern) Caribbean islands. This distinction proved useful, as significant differences were noted between the two types of Caribbean-born subjects. Although self reported information was collected on the subjects' 'racial' mixture, this was not found useful to this project. The

simple definition of ethnicity used was adequate in clear recognition that ethnicity is a complex and controversial concept, (Webster and Fox 1989). Senior and Bhopal (1994) noted that ethnicity is, among other things, a socially constructed phenomenon, which may be difficult to encapsulate. This was recognised by Marmot (1989) who wrote that ethnic boundaries were imprecise and fluid, emphasising that the definition of ethnicity to be used for a particular research needs to be clearly defined. Within the definition of ethnicity used in this study, it was recognised that Caribbean was not synonymous with African-Caribbean, as there are many different 'ethnic' or 'racial' groups, as well as admixtures of the different groups. Effort was made to distinguish African-Caribbeans from other racial groups, by excluding from the programme Caribbeans who had no known grandparent who was at least partly of African origin. Thus, whether the subject was Caribbean-born or UK-born, at least one grandparent had Black-African origin.

The distribution of Caribbean-born (59%) to UK-born (41%) was relatively even, but there was a significant difference in blood pressures between the groups. The UK-born subjects recorded statistically significantly lower systolic blood pressures, (116 mmHg) than their Caribbean-born colleagues whose mean was 126 mmHg. However, this difference was marginalised, and no longer statistically significant, as it was primarily age related, based on the much younger UK-born group. The mean age of the Caribbean-born subjects was 43 years compared to 32 years for the UK-born, and this difference was statistically significant. Similar outcomes were found when duration that Caribbean-born subjects had lived in the UK was examined in relation to blood pressure. Most (82%) had resided in the UK for more than 20

years, but the significance of duration of residence was lost when adjustment was made for age, as older subjects tended to have had longer residence.

In conclusion, ethnicity as defined in this study did not explain any significant percentage of the variability in blood pressure within the group. The major difference in blood pressure with respect to ethnic considerations resulted from the age-related rise in blood pressure.

9.3e Age

Age proved to be the most significant factor in the subject's blood pressure values, Pearson's r = 0.43. This result was in keeping with pre-analysis expectations as an age related rise in blood pressure is one of the most established aetiological 'facts' with respects to the western world, and western lifestyle. The importance of the effect of age on the respondents' blood pressures was borne out, when controlling for age in analysis reduced or eliminated the significance of some significant variables, most notably, country of birth. Most of the subjects were in their thirties, (mean age 38 years, SD ± 8.7, range 41 –20-61 years.), but the normal distribution of age meant that there was proportional representation of the age groups.

With respect to age related rise in blood pressure, the blood pressure demography was not significantly different to that found in the UK population (OPCS, 1993) and the populations of most western societies, (WHO 1993, Intersalt 1988). Rodriguez et al (1994), using data from Intersalt, computed the blood pressure values at age 20, 40, and 60, years for a number of populations world-wide. Among the findings included the results for a Birmingham, (UK) population, which showed the median

systolic blood pressure, (in Birmingham), according to the age groups to be 108 mmHg, 118 mmHg, and 128 mmHg respectively, with a median systolic blood pressure of 118 mmHg.

Body mass index proved to be the most 'positively' significant, of the lifestyle variables. This was also a pre-analysis expectation based on the prevailing literature Body mass index on its own, without age, resulted in a Multiple R of 0.25. However, the findings were somewhat complex, as the relationship was only significant when body mass index was grouped. When body mass index was analysed as a continuous variable, the relationship with systolic blood pressure was not significant.

The positive association between blood pressure and body mass index in Blacks, especially women, has perhaps been one of the most consistent findings relating to the aetiological significance of lifestyle related factors on hypertension in this ethnic group. This has been found applicable to the African-Caribbeans. Cruickshank (1993) reported that body mass index could account for the higher blood pressures found in African-Caribbeans. However, Chaturvedi et al (1993), concluded from their findings that body mass index did not sufficiently explain the higher blood pressure in African-Caribbeans in their study.

In this study, subjects with the lowest body mass index were least likely to have high blood pressures by an average systolic blood pressure 1.8 mmHg. However, respondents who recorded the highest body mass index, (greater than 30 kg/m^2) did not have higher systolic blood pressure than their lighter counterparts immediately

below them, but there was no significant difference in body mass index between these top two groups. The body mass index results of this study compared favourably with those of the OPCS (1993) survey where a positive linear relationship found. However, in the OPCS finding, the blood pressure continued its upward (linear), curve through to the highest-level body mass index. Another, (significant) difference in the findings of this study was that the blood pressure for all body mass indices groups, was lower in the African-Caribbeans of this study than in the general population represented through the OPCS survey.

When age and body mass index were entered into a linear regression equation, the two variables were significant, with Multiple R = .421.

Conclusion: Body mass index has partially explained the differences in blood pressure between African-Caribbean women and European women, but the findings are not universal. Body mass index may be a very significant factor in the pathophysiology of hypertension in African-Caribbeans, especially females, as supported by the findings in this study.

9.3f How Significant Were the Lifestyle Variables

Very early in the analysis it was evident that findings were not 'positive' for most of the lifestyle-related variables. **General diet, alcohol, physical activity** did not appear to have any significant impact on blood pressure.

The majority (75%), of respondents were currently non-smokers, while 60% had never smoked. Thus 25% of the (largely female), subjects smoked. These figures compared favourably with the 1990 findings on regional trends in smoking in the South east, reported by Church (1993). In this regional survey 31% of men and 28% of women smoked. The OPCS (1993), figures for women also revealed a similar smoking pattern to that found in this study group, whereby the vast majority of subjects were non-smokers, occasional smokers, or very mild smokers. However, as shown in table 9.60 below the percentages of heavy smokers were higher in the OPCS group (18%), compared to the heavy smokers in this group (9%), with the reverse being true for light smokers.

Table 9.60 Smoking Habits of This Study Group and OPCS 1993

	This Study Group	OPCS 1993 (Women)
Non Smokers/ex-smoker	75%	74%
Very Light Smokers	16%	8%
Regular to Heavy smokers	9%	18%

Smoking habits of the African-Caribbeans in this study were similar to that of the subjects in the national OPCS survey.

A number of surveys have reported on the comparative smoking habits of whites and blacks in the UK. The results on smoking found in this study concord with a most of them. In an OPCS report (Marmot, Adelstein, Bulusu 1984) it was stated that: "***Mortality from respiratory disease is low in African-Caribbeans.... Part of the explanation for this pattern is the lower prevalence of heavy smokers among the Caribbean immigrants***" (p. 69). The *Health of the Nation* (1992, p.120) also reported that smoking prevalence was lower in African-Caribbeans. Reports by Jackson et al, (1981) and Balarajan and Yuen (1983), concluded that overall African-Caribbeans smoked less than the national average. The HHRP

(Trenchard-Mabere1992) found that among all the ethnic groups, including Whites, Caribbeans had the lowest rates of smoking. The SHARP (Thompson et al 1993), reported that White women had the highest prevalence of smoking (47%), among all ethnic groups, which was more than double that of African-Caribbean women, (20%). However, in the SHARP study a similar prevalence existed among African-Caribbean men (47%), and to that of white men, (43%). In the Birmingham study, (Cruickshank et al 1985) Black respondents smoked less than whites. The results of the Brent study, (Chaturvedi et al 1993) were similar, although the smaller quantity of cigarettes smoked weekly by African-Caribbeans was not statistically significant different to that of the whites.

Within this study, it was found that UK-born Caribbeans were more likely to smoke than their Caribbean-born colleagues were. Thirty-four percent of the UK-born subjects smoked compared to 19% of Caribbean-born (generally older) respondents. A similar finding was reported by the West Lambeth study (Regan and Parry-Cooke, 1993), in which it was found that 28% of Caribbean-born subjects (male and female) smoked, compared to 51% of African-Caribbeans who were UK-born.

In this study, associations found between smoking and blood pressure were inverse, that is, non smokers had higher blood pressures than smokers. However, the differences were largely non-significant. Cigarette smoking at any level did not contribute to raised blood pressure. Subjects who smoked less 10, per week (lighter smokers), had lower blood pressures than those who smoked more, (heavy smokers), but the result was also not statistically significant. In addition, compared to non-smokers, subjects who started smoking before age 18 years had significantly lower

blood pressure. Respondents who started smoking at a later age also had lower blood pressure then non-smokers, but the difference was not statistically significant. Since the majority of smokers were in the younger age group, this could have had a possible impact on the non-positive association with blood pressure. The smoking factor that had the most statistically significant impact on the blood pressure was **smoking history**, (ever smoked), where a significant, but negative, association was established.

The findings in this study that smoking did not contribute to raised blood pressure are not unique as similar results have emerged from a number of other studies in various populations, including the *OPCS* (1993). The findings of *OPCS* were that cigarette smoking among women was not associated with systolic blood pressure. The OPCS also reported a higher mean blood pressure level among those who had never regularly smoked than the rest. It concluded that overall, for both men and women: ***"There does not appear to be a clear or direct association between cigarette smoking status and raised blood pressure"*** (p. 23). In both this study and the *OPCS* survey, non-smokers generally had higher systolic blood pressure than smokers, but in this study, the heaviest smokers had higher systolic blood pressure than both non smokers and light smokers.[14]

The relationship between smoking and blood pressure has scarcely been examined in African-Caribbeans. However, one major study, the City and Hackney

[14] It is worth noting that for a heavy smoker who has not smoked for some time before the measurement is made, the blood pressure may be underestimated (Mann et al 1991).

Hypertension Reduction Project (Trenchard-Mabere 1992), found no consistent relationship between smoking and blood pressure in African-Caribbeans surveyed.

In conclusion, compared to non-smokers smokers did not appear to have a greater risk of hypertension. However, smoking above a certain level (continuous heavy smoking), could place the African-Caribbean individual (like any other individual within the society) at greater risk of developing hypertension.

Physical Activity

The Allied Dunbar Survey (1992), in computing levels of physical activity in the general population, included everyday activities such as walking, shopping, DIY, gardening, and housework. Physical activity levels of the subjects in this study were computed by taking into consideration all types of physical activities, including physical activity at work, and structured exercise. Sixty-two percent of the subjects had little or no physical activity, while 26% were 'fairly' active. Only twelve percent could be categorised as 'very' active. These figures reflected those of Allied Dunbar, which found that the majority of adults (70% men and 91% women), did not have up to the recommended level of exercise per week. The findings in this study also concord with the OPCS (1993). Table 9.61 below shows that approximately the same percentages (62% and 64%) of subjects were in the lower half for both this study and the OPCS group. In both groups physical activity level could be considered below the nationally recommended levels (*Health of the Nation*, 1992).

Table 9.61 Physical Activity Levels of This Study Group and OPCS

Physical Activity Level	This Study Group	OPCS 1993 (women)
None	33%	20%
Not very	29%	44%
Fairly	26%	32%
Very	12%	04%

Both groups show the same patterns in physical activity levels.

Like many of the other lifestyle factors in this study, the general physical activity levels of African-Caribbeans as a group are relatively unknown. However, there have been a few studies which have included information on physical activities in their surveys, and in general they have reported comparable findings to those of this study. In the HHRP (Trenchard-Mabere1992), where the majority were of the age group over 50 years, 80% of respondents for all ethnic groups, reported that they did not participate in any regular form of exercise. In the West Lambeth study (Regan and Parry-Cooke 1993), 26% of respondents described themselves as very active, 49% as fairly active, and 25% as not very active. The Smethwick Heart Action Research Project - SHARP (Thompson et al 1994), concluded that based on levels of physical activity (not including structured exercise), African-Caribbeans, were overall, no less physically active than any of the other ethnic groups. Chaturvedi, (1993), also found no significant difference in physical activity levels between African-Caribbeans and the indigenous White group.

Within this study, it was apparent from very early into the analysis that no significant association would be found between blood pressure and physical activity. This compared with the OPCS findings in women, where no significant association

was found between levels of physical activity and blood pressure. However, in that survey there was an association found in the male respondents. Subjects in this study who were calculated as being most active had the highest systolic blood pressure, with those who had just above zero activity having the lowest values. It is postulated that this could be associated with the type of physical activity, that is, how stress related it was. For, instance, if the subjects in this range were deriving most of their physical activity from work related tasks, compared to leisure pursuits.

DIET

The study sought to determine, generally, if the subjects eating habits could be considered healthy, in relation to hypertension risk. Generally, the respondents were found health conscious, with a high level of awareness of healthy diets. Only 6% of subjects reported that they did not take particular care to eat healthy meals on a daily basis although most, (70%), ate primarily non-vegetarian meals.

There are very few surveys on the African-Caribbeans' dietary practices, but the few than have been conducted have largely concluded that a significant proportion of they show a strong preference for Caribbean foods, and that generally the African-Caribbean diet is well balanced. The findings of a survey in the 1960s by McKenzie and Mumford's (1964) were that African-Caribbeans tended to prefer Caribbean foods. This was echoed more that 20 years later by Kemm et al (1986), who reported that the English food eaten as part of the Caribbean diet were lighter meals and snacks. Douglas (1989) reiterated these findings, when she concluded that although the dietary habits of African-Caribbeans is not well known, a large

proportion of the African-Caribbeans diet comprised Caribbean dishes. She also reported that the African-Caribbean diet was nutritious and balanced, stating that: ***"Traditional Caribbean diets can be recommended as being sound in terms of nutritional content and balance, being high in fibre and low in saturated fats"*** (p.254).

In this study preferences for Caribbean versus UK dishes was not investigated, but the observation was that the subjects dietary habits were mixed and that 'undesirable' foods were not consumed at greater levels in this study group than the general population. This position is supported by the SHARP study (Thompson et al 1993) which concluded that from a nutritional standpoint, African-Caribbeans had similar dietary habits to whites. Thus whether the foods eaten were traditional or English or more likely a combination, the nutrient value was deemed to be within the recommended standards.

Healthy eating habits, in particular avoidance of high cholesterol foods, is one of the major promotional tools in coronary heart disease risk prevention, and this applies to hypertension (*Health of the Nation* 1992). The OPCS survey found total cholesterol level to be significantly associated with blood pressure, whereby those with higher cholesterol levels tended to have higher mean blood pressure. In this study, there was no direct measurement of cholesterol levels, with estimations being made from information on general diet, including red meat. No significant association between dietary habits and blood pressure was found. Douglas (1989) opined that ***"Health promotion messages should be based upon promoting traditional Caribbean diets while at the same time recommending areas for improvement in terms of lowering***

fats, sugar and salt intake (p.254). Based on the findings in this study, the author concluded that dietary habits were of no more importance in regard to hypertension risk in this study group than was to be found in their counterparts within the general population. The author considers that it would not be necessary to offer 'special' dietary advice to African-Caribbeans similar to those that participated in this study, over and above what is recommended to the general population.

SALT

Of all the dietary variables, salt/sodium has had the most attention as a hypertension risk factor, evidenced by the Intersalt study (1988). Excess salt intake (salt intake above a certain level) is generally considered to have the greatest blood pressure raising effect, particular in western societies, (Luft et al, 1979; Intersalt 1988; Law et al 1991). The British hypertension society guidelines for the management of mild hypertension in clinical practice include restriction of salt intake (Sever et al 1993), a position supported by both the *Health of the Nation* (1992) and the Ministry of Agriculture and Fisheries, (MAFF 1994). According to Beevers and Beevers, (1992), ***"There is every reason to believe that (a down ward) shift of blood pressure in the whole population could be achieved if the average sodium intake was reduced to below 100 mmol per day***"(p.129). In modern societies, such as the UK, much of the intake of sodium is from **'hidden salt'**, and food companies have been urged to reduce the salt in their products (Godlee 1996). The MAFF (1994) confirmed this 'hidden' salt in the foods when it reported that a high proportion of the 2,86 mg sodium intake in the UK diet was from 'hidden' salt in such forms as cereals, bakery products, snacks, and beverages estimated that.

High salt intake has been deemed to be particularly relevant to people of Africa origin, (Luft et al 1979; Law et al 1991). With the salt intake in the UK African-Caribbeans being largely unresearched, the blood pressure raising effect of salt is in that group is also unknown. As such postulates as to the special importance of low salt diets in African-Caribbeans are based on 'foreign' studies of other peoples of African descent, especially African-Americans.

Much attention was given to the information on (reported) salt intake habits of the subjects in this study based on the prevailing literature its importance as a risk factor to western populations (Intersalt 1988), but more specially because of its even greater importance to Blacks. Responses indicated that the salt habits of the group may not have been significantly different from that of the general population as reported by the MAFF, (1994). Most (94%) of the subjects cooked with salt. Very early in the analysis, it was evident that there was no difference in blood pressures based on frequency of cooking with salt, categorised as never, sometimes, or always. Thus, no significant association was found between cooking with salt and blood pressure in this study group. The HHRP, (Trenchard-Mabere1992), also reported that no significant difference was found between salt use and blood pressure in African-Caribbeans. However, in that survey only 33% of the African-Caribbean participants said that they generally cooked with salt. The findings of HHRP were that, there was a stronger association between reported salt use and blood pressure in the non-Caribbean group, than was found in Caribbeans.

Anecdotal evidence suggests that African-Caribbeans have a general preference for spicy or highly seasoned foods, which are generally high in 'hidden' salt. No

association was found between frequency of consumption of spicy foods and blood pressure. Indications from this group were that consumption of heavily seasoned food was not excessive, as only one third (34%) of respondents indicated that heavily seasoned foods was part of their normal daily diet. Thirty nine percent of the subjects said they ate spicy foods regularly at least on a weekly basis, and the remaining 27% did not generally or only rarely ate seasoned foods. It could be concluded that with regard to heavily seasoned food, the African-Caribbeans in this study did not deviate significantly form the general population. The results of the Hackney Hypertension Reduction Project (Trenchard-Mabere 1992) support this view, as the differences between ethnic groups in that project, including Whites, in reported salt use were negligible.

Add Salt: A negative and rather bemusing association was found between blood pressure and adding salt from very early in the analysis. Subjects who were more likely to add salt (always or nearly always added salt), had significantly lower systolic blood pressure than those who were less likely to or never added salt. The B-coefficient revealed that the sometimes-added salt group (42% of subjects) had systolic blood pressure 9 mmHg (significantly) lower than the never added salt (baseline) group, which comprised 48% of the subjects. Those who always-added salt (which were only 10% of the subject), had systolic blood pressure of 4 mmHg lower than the baseline (never added salt) group. The (negative) significance of adding salt survived the rigours multivariate regression analysis, and was part of the final model. Salt habit was not statistically significant across age groups, or across ethnicity based on country of birth. However, subjects in and around the 40s age group contributed most to the (negative) significance.

The results of this study suggest that the salt intake (cooking with salt, adding salt, and hidden salt combined) of the subjects, reflected that of the general population. If this were correct, then, it would seem that a relatively high intake of salt (the national level is higher than recommended) did not result in higher than normal blood pressure in this group. In other words, African-Caribbeans in this study, who had the 'normal' salt intake of the population, did not appear to be at increased risk of hypertension. This finding reflects that of the Madhaven and Alderman (1994) homogeneous workforce hypertension control programme (USA) study in America. They reported that the Black and Caucasian subjects had similar blood pressures at the same sodium intake, and that apparent differences were attributable to the influence of weight and age, rather than race. Madhaven and Alderman also opined that no study had offered convincing evidence that a difference exists.

Beevers and Beevers (1992) recommendation that ***"a population salt reduction strategy might prove particularly successful for African-Caribbeans"*** (p126). From the results of this study, and that of numerous other studies, particularly on African-Americans, the usefulness of this recommendation is questionable.

Salt and Genetics: Intrinsic to the sodium retaining, salt-sensitivity explanations for the high prevalence of hypertension in Western Blacks, (including African-Caribbeans) is a genetic explanation, such as that offered by Wilson and Grim, (1991), and many others. Proponents of this theory maintain the likelihood that renin release is suppressed in some way by a genetic tendency in Blacks to conserve sodium. The salt conserving tendency is not thought to be relevant to Blacks in

Africa, and Wilson and Grim offered a possible explanation. They suggested that the nightmarish conditions associated with the capture or purchase of slaves in Africa, and their transport to the New World in the holds of sailing ships where the heat was intense and diarrhoeal illness almost universal, led to a high mortality in transit. However, those slaves with the best (well) developed renal capacity to conserve sodium were the most likely to survive. The theory presupposes that the slaves and their descendants in the Americas (including the Caribbean) represent a subset of Africans who were most able to retain sodium. Given the high salt intake of western societies, the ability ceased to be an advantage, but instead led to an increased tendency to develop hypertension.

The hypothesis may be plausible but it is difficult, and perhaps impossible to prove. The theory has been dismissed in relation to UK Blacks when Chaturvedi et al (1993) stated that they could not support the hypothesis as: ***"median blood pressures were as high in west African migrants as they were in Caribbean born migrants"*** (p.95). The sodium retaining theory, remains at best a viable theory, yet to be scientifically established.

ALCOHOL

Most subjects in this study consumed less than the recommended maximum weekly intake, of 14 units for women and 21 units for men (*Health of the Nation*, 1992). Fifty five percent of the largely female subjects did not drink alcohol, or drank it very rarely. Thirty-five percent consumed less than 7 units per week. Only 11% drank more than 8 units per week, with the greater majority consuming less than 21

units per week. The alcohol consumption rates found in this study group were lower than that found in the female group of the national population, (OPCS 1993), but the trends were similar as shown in table 8.62 below.

Table 9.62 Alcohol Consumption in this Study and the OPCS

Consumption Weekly	This Study	OPCS 1993 (women)
None-rare	55%	33%
0.5 to 7	34%	39%
Over 7 units	11%	9%

Although alcohol consumption patterns were similar, the respondents in this study consumed less alcohol per week than found in subjects of the national study.

The findings of this study corresponded to that of the SHARP survey (Thompson et al 1993), which reported that Whites, both men and women had the highest proportion of alcohol drinkers (87%). Seventy percent of African-Caribbean men and 57% of African-Caribbean women were alcohol drinkers. The alcohol consumption levels of the African-Caribbeans in this study also reflected other findings on the alcohol habits of African-Caribbeans, (Jackson et al 1981; Balarajan and Yuen 1983). A survey by Cade and Sharma (1994) found that African-Caribbean women consumed low alcohol compared to the national level, but that the African-Caribbean men were higher than the national level. In the Brent study (Chaturvedi et al 1994) it was concluded that African-Caribbeans consumed (overall) less alcohol than the indigenous population, but that the difference was not statistically significant.

Reports, both national and international, on the effect of alcohol on blood pressure are mixed. The OPCS (1993) results added to the non-conclusive 'evidence' as it found an independent association with systolic blood pressure in men, but no association in women. An association with diastolic blood pressure was established in both women and men. With regard to females in that survey, it was the non or occasional drinkers who were more likely to have high blood pressure, than expected for their age distribution alone, while moderate drinkers (7-14 units per week), were less likely to do so. This study found no significant association between alcohol and systolic blood pressure. Non-alcohol drinkers did not have significantly lower average seated blood pressure than alcohol drinkers did and amount of alcohol intake weekly did not appear to affect blood pressure.

It is concluded that the respondents of this African-Caribbean study group generally had a low alcohol consumption rates, and that this finding harmonies with others that have examined this health behaviour in African-Caribbeans. Perhaps the low association found between blood pressure and alcohol could be partially explained by the relatively low alcohol consumption within the group it. However, since such an association has not been consistently found in the general population, and in many other populations the findings of this study may be representative of a wider African-Caribbean population. This is reflected in the opinions expressed at government level in 1995 that the safe limits set by the *Health of the Nation* (1992) may be extended. The results of this study, coupled with those found in other research are taken as indicators that high alcohol consumption need not be deemed a high risk factor in hypertension in African-Caribbeans.

9.4 SOCIO ECONOMIC FACTORS

The Black Report, which highlighted inequalities in health, has served as a solid reference guide of the relationship between health and social circumstances measured by occupation, education, accommodation, and general living environment. Generally, the lower the social 'standing' the greater the risk of resultant poor health, (Blaxter 1990). The findings of the *Black Report* (1980) are also considered relevant to hypertensive disease. The higher systolic blood pressure among African Americans has been linked to education, social circumstances and socio-economic status, (Wilson et al 1991; Cooper 1984). The *Black Report* (1980) and the *Health of the Nation* (1992) acknowledged that Ethnic Minorities and Blacks do suffer from the inequalities in health. In light of the prevailing position with regards to importance of socio-economic factors on in health, the higher blood pressure in Blacks, including African-Caribbeans could reflect the (generally) lower socio-economic status and a lower standard of health care.

In **this study**, social circumstances was calculated by examining education level, job grade in the organisation, accommodation, and other factors including car ownership. As subjects comprised representatives from top management to unskilled employees, a reasonable comparison was possible. Many of the respondents were various grades of social workers, including both administrative and field workers. The only socio-economic factor to remain significant following rigorous analysis was job grade..

ACADEMIC QUALIFICATIONS AND JOB GRADE

The study group was representative of most grades of academic qualifications and job grades. Virtually equal percentages of subjects were in the top two education levels, (university and professional) as in the bottom two levels (high school and no qualifications.). However, in the job grade distribution a higher percentage (59%) was in the top two levels compared to the lower two levels (24%). For the purpose of the study, job grade categorisation was defined according to the organisations grading system, as opposed to the national Class 1-IV system, with top management executives being the highest ranked. The distribution indicated that employees were promoted based on performance, as well as qualification, as job grade did not always mirror qualifications. From observations it appeared that the groups were representative of 'typical' social service civil servants, working in a relatively safe and comfortable environment. However, during the process of the study, the organisation underwent major policy changes, which resulted in much job insecurity. This did not, however, impact on the blood pressure of the subjects, as no difference was found in the blood pressures of those subjects who said they felt most insecure and those who felt less.

The inverse associations found between blood pressure and qualifications and job grade were in keeping with expectations based on prevailing literature. The least qualified (no qualifications), and lowest job grade groups, recorded significantly higher blood pressures than their more qualified and higher grades colleagues. This reflects the reports from a number of studies including the Whitehall study (Marmot and Shipley et al (1984) of an inverse relationship between job grade, which is a reflection of education level, and CHD related diseases. However, the linear

significance of job grade and blood pressure in this study on African-Caribbeans was weakened by the relatively low mean blood pressure found in the middle grade employees.

The association found in this study between education level and blood pressure did not survive the rigorous linear regression tests. A significant (linear) association could not be established primarily because the highest qualified group, (university level), recorded the second highest mean blood pressures. The middle educated levels recorded blood pressures lower than both the groups below and above them. However, despite the non-significance, the results veered towards the expected inverse relationship, that is, the least educated individuals had higher blood pressures than the more educated.

It is perhaps significant that the relatively low blood pressures found in the middle job grade group somewhat corresponded to the relatively low pressure of the middle education group.

ACCOMMODATION

Most (67%) of the subjects reported that they were very satisfied with their accommodation, and generally lived in conditions of low crowding, calculated as number of rooms to occupants ratio. In general, subjects were well accommodated, with the gap between those at the highest end of the continuum and those at the lowest end being narrow. Significant, but unexpected negative associations were derived for crowded accommodation and dissatisfaction. Somewhat surprisingly, subjects with the higher levels of crowding had the lowest levels of blood pressure,

and subjects who reported being most satisfied with their accommodation recorded higher levels of blood pressure than their colleagues who were not as happy with their dwellings. The relationship was linear with blood pressure rising as levels of crowding fell. The results suggested that living in crowded accommodation, as number of rooms divided by number of occupants, did not contribute to high blood pressure. It was the subjects who lived in the least crowded accommodation that recorded the highest mean blood pressure. Similar results were found with regards to satisfaction with accommodation. Respondents who reported being least satisfied with their accommodation, recorded significantly lower blood pressure than their colleagues who lived in more comfort.

Stress

Stress is one of those conditions that, intuitively, would be deemed a precursor to hypertension, evidenced by the numerous examinations of the relationship, which have yielded a conglomerate of findings. Avoidance of stress and stress-related factors remains a promotional tool in hypertension prevention and management.

A significant positive association was anticipated, but none was found, rather the most stressed individuals (58% of subjects), recorded lower mean systolic blood pressures (121 mmHg), than their less stressed counterparts which comprised 42% of subjects, mean systolic blood pressure being 124 mmHg. This skew towards negative significance corresponded to the OPCS (1993), findings where no significant association was found for women, and there was an independent negative association with blood pressure of males. The report stated: "***Although the logistic regression did not show a smooth relationship, there was a general trend (age***

standardised), for those with higher blood pressure to report less stress (p. 149). The *OPCS* report described the result as ***'counter-intuitive'***, a view which is shared by this author.

RACISM

The nature of, and the adverse consequences of racism, makes it almost self-evident that racism would adversely affect the victim's health, (Krieger and Sidney. 1996). Pearson (1989), contended that it was essential that the impact of racism on the health of ethnic minorities in a white society, be examined, and stated that: ***"the stress from direct racist abuse, and direct structural discrimination also has an impact on health"*** (p. 75). High on the list of racism related conditions is hypertension (Krieger and Sidney 1996). Cooper (1984), believed that it was reasonable to associate racism with hypertension, as ***"The experience of being Black in a White racist society is the key stressor that accounts for the excess risk of hypertension among persons of African origin"*** (p 219.

The expectation prior to embarking on this study was a positive outcome, whereby those who were adversely affected by racism would exhibit higher blood pressures than their colleagues on whom racism had affected less adversely. Contrary to this 'natural' expectation, the bemusing results showed exactly the opposite, with negative significance. The majority of the subjects reported being adversely affected by racism at some time or other, both within and outside the work environment. Anger from racism was examined as an indicator of the (mental) effect and most respondents (46%) reported that they felt angered from racism at least sometimes. Although one third of the respondents (29%) said they felt angered on a weekly

basis and 15% on a daily basis, no adverse effect on their blood pressure was found, but rather the opposite. Very similar percentages were found for effect of racism on career, and racism from colleagues, but again with no adverse effect on blood pressure.

The results of the study somewhat support the findings of the Committee for Racial Equality (CRE 1983), that significant racial discrimination in employment existed, which led to the approval by Parliament of a Code of Practice for its elimination. However, despite the large percentage of subjects who felt they had suffered at least some adverse effects from racism, and who were angered from it, there was no indication that racism contributed to high blood pressure.

Cooper and Rotomi (1994) offered an explanation for results such as those of this study, stating that: "***the impact of racial antagonism on the individual psyche is not adequately captured by the concept of 'stress',***". Their explanation encompassed the concept of "***Living behind the veil***", that ***"connects racism to blood pressure control, rather than its ability to produce anxiety or other psychological discomfort"*** (p.219). Cooper and Rotomi further opined that: ***"much of the difficulty with psychosocial research on hypertension in Blacks may reside in the paucity of theory-driven research that is embedded in the social context of communities"*** (p. 219). The author interprets this to mean that because racism is a way of life for Black people in western societies, adaptation has occurred such that the 'natural' stress responses are no longer evident or even measurable. Rather, a certain amount of blood pressure control is the result. If Cooper and Rotomi are correct, then it is almost impossible to scientifically ascertain the true impact of

racism on the 'sufferer'. This was borne out when they wrote that: ***"unfortunately relatively little attention has been focused directly on this question and there are few tools available to study it at present"*** (p. 220). Cooper and Rotimi concluded that: ***"it remains entirely plausible, from a theoretical point of view, that the driving force underlying the differential in blood pressure between Blacks and Whites is the psychologic consequences of racial discrimination"*** (p. 220). The findings of the recent major study in the USA (Krieger and Sidney. 1996) support this standpoint.

9.5 SUMMARY OF OVERALL FINDINGS

The final model included only the variables that were accepted into the equation using regression stepwise method. These were age, body mass index accommodation adequacy, adding salt, anger from racism, and effect of racism on career. Table 9.63 below summarises the outcome detailing actual versus expected results, and shows that a number of variables did not yield the expected results, but rather the opposite.

The variables in the final model resulted in a Multiple R of 0.601, R Square = 0.361, adjusted R Square = 0.336, and F-statistics with 157 df of 14.77, Sig = 0.000. Thus, based on the final model, approximately 34% to 36% of the variation in blood pressure found in the subjects could be explained by the model. Age was the most significant factor. The model derived suggested that if information was provided on

all the factors within the model, a reasonably accurate prediction of the systolic blood pressure of an individual of similar overall lifestyle to the subjects in this study could be made.

Table 9.63 The Variables in the Final Model

Risk Factor	Actual	Expected	Comments
Age	Positive	Positive	Age had the strongest independent association. The independent Pearson's correlation coefficient was r= 0.45. Within the final model, the standardised Beta-value was 0.313.
BMI	Positive	Positive	In the final model, body mass index-grouped had a standardised Beta value of 0.207.
Add Salt	Inverse	Positive	A very suprising result. Although included in the final model, it's (negative) significance diminished at more stringent analysis level, to a standardised Beta value of –0.161.
Accommodation *-Adequate*	Positive	Inverse	Lower level accommodation status did not contribute to high blood pressure, but perhaps the opposite, with a positive standardised Beta of 0.191.
Racism: Anger	Inverse	Positive	The higher the levels of anger from racism the lower the mean blood pressure found. The (negative) standardised Beta value being -0.180
Racism: Career	Inverse	Positive	The greater the adverse effect of racism on career, the lower the lower the mean blood pressure found. The (negative) standardised Beta value being -0.175.

The Final Model shows that in some instance negative (inverse) associations were found where positive ones were expected, and vice versa.

From the model a predictive **algorithm** was derived:

ALGORITHM: PREDICTED SYSTOLIC BLOOD PRESSURE =
Constant: 100.7 + (age: * 0.540) + (Racism: career * -3.1) + (BMI *3.1) + (Racism: angry * -3.2) + (Accommodation Adequate* 6.1) + (Add salt * -3.7).

9.5b Significant Variables Outside of the Model

Lifestyle factors that were found statistically significant but did not make it into the model were **Smoking** (negative significance), **Education** (inverse relationship), **Job Grade** (inverse relationship), **and Birthplace** (Caribbeans higher blood pressure). It should be noted that **Brothers' BP** and **subjects history of raised blood pressure** were eligible for entry into the final model, but were deliberately excluded because they were not 'strictly' lifestyle related factors, and would have confounded the analysis to derive the final model.

9.5c Conclusion

Age related rise in blood pressure was unquestioningly evident. In general, health behaviour lifestyle factors were not significantly associated with systolic blood pressure. 'Undesirable' health behaviour did not appear to predispose the subjects to high blood pressure. The only health behaviour factor that showed this kind of association was body mass index. Dietary factors along with alcohol and smoking habits were generally not positively associated with blood pressure, but significant inverse associations were found, most notably with adding salt, and smoking. Similar results were found for the socio-economic lifestyle factors. Where significant associations were found, they too were often not adverse, such as in factors relating to levels of accommodation. However, significant negative associations were found in factors relating to education and job grade, although the significance of these factors was marginalised at the final model development stage of the analysis. Non-associations or adverse associations were also unearthed from the psychosocial factors, most evident in stress (no association), and racism, adverse associations. On first examination, it appeared that there was a significant difference

in blood pressure patterns based on whether the subject was UK-born or Caribbean-born, but this later shown to be non-significant. An interesting result was that brothers' history of high blood pressure was positively significant to the subjects own blood pressure, but no other significant familial association was found.

10 Tenth Chapter

Interpretation of the Findings

10.1 Was the Hypothesis Proved

The main research question posed on page 23 and the related questions on page 24 concerned whether respondents at the higher end of the blood pressure continuum would generally be seen to have less desirable lifestyles than their colleagues at the lower end of the scale.

Subjects with the more desirable lifestyles (overall) did not have significantly lower systolic blood pressures than their colleagues with less desirable health behaviour. Except for body mass index, there was no clear indication to suggest that health behaviour and lifestyle in general could account for the higher blood pressures found in some subjects. Although there were significant indications that education and job grade were positively associated with blood pressure, these factors did not withstand the rigid linear regression analysis, and did not graduate into the final model. None-the-less, based on the numerous findings of significant associations, including within this study, their importance cannot be determined. The (surprising) negative results, whereby lifestyle factors that were expected to yield positive outcomes, recorded exactly the opposite helped to support the conclusion of the non-effect of lifestyle on the blood pressures of the African-Caribbeans in the study. Prime examples of these related to smoking, accommodation, stress, and racism. The non-significance of familial association (except with brothers) was also surprising.

It must be concluded that the only lifestyle variables that were hypertension risk factors in these subjects were body mass index and job grade (although job grade was not included in the final model). Body mass index in particular warrants further examination, especially as this lifestyle factor has been consistently found to be associated with raised blood pressure, not only in UK-African-Caribbeans (Cruickshank 1985), but also in native Caribbeans (Foster et al 1993). In addition body mass index has been cited as an important risk factor in UK-whites (OPCS, 1993). The body mass indices of the subjects of this study were relatively low, generally below the population mean. Given a wider spread to include more subjects in the higher body mass index categories, there was a strong likelihood that the relationship with blood pressure would have been significantly stronger.

The Final Model is Significant

The results suggested that adverse lifestyle was not necessarily a precursor to high blood pressure. A number of negative outcomes indicated that adherence to a 'desirable' lifestyle may not be beneficial to blood pressure. These negative findings not-withstanding, the final (predictive) model derived from the study was statistically significant, at Multiple R = 0.601, indicating that 35% of the variation in systolic blood pressure could be explained by the variables within it. Multivariate models, including all known exposures, seldom 'explain' more than about one third of blood pressure variance (Dometrius 1992; Stamler et al 1975; Criqui et al 1982). If this is accepted, then the model derived from this study could be deemed of strong predictive value to a hypertension risk programme. However, it is noted that measurements of the hypertension risk factors are imprecise. As Marmot (1989) noted, the examination of lifestyle factors on hypertension is greatly stymied, as any

one factor is likely to result from an interaction of personal situations coupled with social economic, cultural and environmental influences.

Along with **age**, the model comprised a combination of health behaviour (body mass index, add salt), socio-economic (accommodation), and psychosocial (racism) factors, with age having the strongest predictive value. The algorithm derived from the model suggests that the highest risk individuals would be those who were most advanced in age, who were overweight, and tended never to add salt, while living in the most comfortable accommodation, and in whom racism had the least adverse effect. Although deliberately excluded from the model, if individuals also had a brother with high blood pressure, their risk was increased. Other important predisposing factors were having no qualifications and being in the lowest job grade.

The negative Beta values of some of the variables in the model have rendered the model perplexing. However, there could be plausible explanations, such as those offered earlier on the discussion of racism, (page 193). With regard to accommodation, which was broadly categorised as a socio-economic factor, there have been other indications that social stratification may not be an accurate indicator of high blood pressure risk in African-Caribbeans. Chaturvedi (1993) found that as an independent variable, housing tenure did not account for any significant variability in blood pressure among African-Caribbeans. It is worth noting that most of the subjects in this study who lived in what may ordinarily be termed the relatively crowded conditions, were not dissatisfied with their accommodation situation. Some expressed the preference of living as part of an extended family unit.

Social class may not explain blood pressure variability in African-Caribbeans based on the traditional standards. Marmot (1989) expressed this view, stating that:

> ***"Social class, as conventionally defined, cannot explain patterns of disease seen among immigrants ... forces determining mortality in immigrants are not well summarized by the conventional social class measure. Either this measure is not completely capturing the social position of the immigrants... or it is not reflecting the environmental and cultural/ethnic influence on disease risk…. While we should pay great attention to the social and economic position of immigrants, these are unlikely to be the only factors that determine the pattern of disease"*** (p.15).

Marmot also recognised that conventional stratification may not offer much insight into blood pressure trends in African-Caribbeans as: ***"Social class per se cannot account for many of the ethnic differences in disease... the SMRs from hypertension in West Indians ... is larger than the SMR in the most disadvantaged social class in England and Wales (Class V)"*** (p.73).

From a sociological perspective, a positive association with racism should have been forthcoming, and perhaps even welcomed. An identifiable association between racism and (ill) health helps to strengthen the position of organisations such as the Council for Racial Equality (CRE). The explanation offered by Cooper and Rotimi, quoted on page 194, though largely theoretical, has a certain amount of plausibility, and is one that psychologists and sociologist could easily relate to. As Cooper and Rotomi pointed out, it may be a monumental task to study the effect of racism on blood pressure as the tools are not readily available.

From a purely scientific standpoint, results such as those found in this study are much more 'cut-and-dry', based on the respondents own perceptions, and the

corresponding blood pressure measurements. The 'scientific' conclusion would have to be that no association between racism and blood pressure has been established.

10.2 Closing Discussion

Two important findings of this study were, firstly, the age-related rise in blood pressure independent of all other factors, in both Caribbean-born and UK-born subjects. Secondly, that blood pressure, especially among the younger African-Caribbeans, was relatively low. The second point is expressed cognisant of the exclusion of treated hypertensives from the study, and in awareness of the mean systolic blood pressures of normotensives and non-treated hypertensives in the general population.

The *OPCS* (1993) defined untreated hypertension as systolic blood pressure greater than 159 mmHg not controlled by drugs prescribed for high blood pressure. With treated hypertensives excluded from this study, based on the national standards, at least 10%-12% of the group screened were expected to fall within the category of untreated hypertensives, (*OPCS* 1993). In this study, only four persons fell within that category, which converts to 2.4% of the group. Even when the more rigid standard of systolic blood pressure greater than 139 mmHg for hypertensive untreated is used, only 12% were untreated hypertensives. Considering that African-Caribbeans are the highest risk group nationally, it was anticipated that a higher

percentage of persons with high blood pressure than that reported at the national level would be found. With respect to the age-related rise in blood pressure found in this study group, the pattern reflected that of the general population. That is, blood pressure rose with age in African-Caribbeans as it does in the general population. However, African-Caribbeans in this group appeared to have overall lower systolic blood pressures than the corresponding national population group.

The results have led to ponderation of the following questions:

ARE THE MORTALITY DATA TOO OLD TO BE RELEVANT?

How relevant to the 'new', younger, and changing African-Caribbean population are the findings of 1970s-80s mortality data on which much of the conclusions concerning the prevalence of hypertension were based. As Cruickshank (1993) pointed out mortality rates in the British African-Caribbean community from hypertensive disease and its commonest direct outcome stroke, are still not published for the later 1980s and 1990s. The 1993 *OPCS* report gave no details on this high-risk group. However, in an earlier report (Marmot, Adelstein, Bulusu 1984) having noted that African-Caribbean migration to the UK was quite recent, it was stated that: ***"the disease patterns might be expected to be strongly influenced by those of the old country and by the process of migration itself"*** (p. 69).

African-Caribbeans have never been an homogeneous group, and are even less so in the 1990s. Like most other ethnic groups, the African-Caribbean population is dynamic, therefore, second, third, and fourth generation members of this group may be just a dim reflection of their immigrant parents and fore parents. Writing in the

1980s Webster and Fox (1989) noted that the UK-born Black population differs in important ways from that which first came to Britain 30 years ago. Unlike second generations born in a 'new' country, immigrants may adopt a new lifestyle, while retaining elements of their country of origin, with a wide contrast in social circumstances. As Senior and Bhopal (1994) noted ***"Ethnicity's fluid and dynamic nature means that results of research may rapidly become out of date - results should not be generalised across time, generations, or populations with different histories of migration, except with great caution".*** It is now over 40 years since the mass migration of African-Caribbeans to Britain began. It seems evident that the generation gap and differences in disease patterns would have expanded.

This leads to a further question.

WHO IS AN AFRICAN-CARIBBEAN IN THE 1990S?

In the 1970s and 1980s reports on hypertension mortality, African-Caribbeans were defined based on country of birth only. Even back in the 1970s when the Caribbean population was more 'pure' the definition was inaccurate, since not all people born in the Caribbean (West Indian) islands were of African origin. Now in the 1990s to define an African-Caribbean is even more difficult. Webster and Fox (1989) pointed out that the concept of ethnicity is neither simple nor precise and asked:

> ***"does the term include solely immigrants, or also those born in the 'new' country to parents (or grandparents) born overseas. If the latter, is membership of a particular ethnic group restricted to those with both parents born in the same country; how are those of mixed parentage to be assigned?"*** (p.7).

It is important to be aware that those African-Caribbeans who were found (based on medical records) to be high risk, may not be the socially and ethnically the same types of 'African-Caribbeans' who currently reside in the UK, that is, in the 1990s.

Prior to 1991, ethnicity was not a factor in routine health statistics, but rather place of birth. Thus, before the 1990s there were no independent health statistics data on UK-born African-Caribbeans, that is, offspring of first and second generation Caribbeans. General mortality statistics were therefore, unable to provide data on this group. Past mortality findings may have little or no relevance to present day African-Caribbeans.

The importance of 'accurate' definitions of African-Caribbeans is paramount to research on the group. Marmot (1989) pointed out that the imprecise and fluid nature of ethnic boundaries makes it imperative that a definition must be made explicit prior of embarking on research. If studies conducted on African-Caribbeans have adhered to Marmot's standards it is clear that the definitions and parameters have differed. Cruickshank and Beevers (1989) asked:

> ***"How many research workers have bothered to define the 'racial' group they study. To our knowledge only Miller has categorised black or Indian groups, whom he studied in Trinidad, by grand parental origin rather than by immediate appearance as a surrogate for 'race'*** (p. Vii-Preface).

In surveys, such as the Birmingham Factory Study (Cruickshank et al 1985) and the Brent study (Chaturvedi et al 1993) persons born in sub-Sahara Africa were categorised as African-Caribbeans, (although sub-categorisations were made within the group). Standardisation on categorisation of African-Caribbeans needs to be addressed. Webster and Fox (1989) opined that to a great degree, the definitions rest upon the requirements and orientation of the researcher.

Senior and Bhopal (1994) argued that ethnicity has not always been a valuable and sound variable. According to them, the main tools used to measure ethnicity, skin

colour, country of birth and voluntary self classification are subjective and therefore, unscientific. They pointed out that a person's ethnicity cannot be determined by skin colour, and that while country of birth is objective it is crude, especially relating to the offspring of immigrants. Voluntary self-classification guided the classification in the 1991 census, but this is unreliable primarily, because self assessed ethnicity is changeable over short periods of time. The opinion of Senior and Bhopal that populations identified by current methods of measuring ethnicity are often too diverse to provide useful information is deserving of in-depth discussion.

INCONSISTENCY IN RESEARCH FINDINGS

Surveys of hypertension by ethnic group in Britain have not all shown higher rates in African-Caribbeans, (Cruickshank 1993). The findings in the main UK studies on blood pressure in African-Caribbeans (Meade et al 1978; Cruickshank et al 1985; Haines et al 1987; Cruickshank et al 1991; Chaturvedi et al (1993) have not always corresponded. For instance, the two studies measuring ambulatory blood pressure although a decade apart had significantly different results. No difference was found between African-Caribbeans and Whites in the ambulatory study carried out by Rowlands et al (1982). However, 10 years later results of the Brent study (Chaturvedi et al 1993) showed an age standardised systolic blood pressure difference of 6 mmHg in men and 17 mmHg in women.

The authors of the Brent Study acknowledged the general inconsistency in findings of studies on hypertension in African-Caribbeans. They pointed out that differences in average systolic blood pressure between African-Caribbeans and Europeans have ranged from 1 mmHg lower in African-Caribbean men to 9 mmHg higher in

African-Caribbean men, and 3 mmHg lower in African-Caribbean women to 7 mm Hg higher in African-Caribbean women. However, the Brent study team reported: ***"We have demonstrated clear differences in blood pressure between the two ethnic groups*** (African-Caribbeans and Europeans) ***and for the first time sex differences in resting blood pressure in Afro-Caribbeans commensurate with mortality data"***(p94). It is important to point out that the African-Caribbeans in Brent study were first generation migrants, aged between 40-64 years, thus a group of 'older' African-Caribbeans. Further, the members of the group termed 'Afro-Caribbean' in that study were not comprised entirely of Caribbeans, but included 'unknown Blacks' and West Africans. However, the authors reported no difference in median blood pressure between Caribbeans and West Africans. Prior to the Brent study, only in the Northwick Park Study (Meade et al 1978) was a 'highly' significant difference found between African-Caribbeans and other groups.

Non-standardisation of the definition of African-Caribbeans across studies could have contributed to the inconsistent findings. Further, most of the studies on African-Caribbean have generally been small scale, with low numbers of subjects who were African-Caribbeans. These include studies of the 1990s such as the SHARP study (Thompson et al 1993) with 60 subjects, the West Lambeth study (Regan and Parry-Cooke 1993) with 146 African-Caribbeans and the CHHRP (1992) with 262 African-Caribbeans. It is noteworthy that while many of the earliest studies carried out in the Caribbean islands found significantly higher blood pressure in Blacks than in Whites (Moser et al 1959; Schneckloth et al 1962), more recent studies suggest that Blacks in the Caribbean did not have a greater prevalence of hypertension (Ashcroft and Desai 1978; Khaw and Rose 1982). A comparative

study (to the Birmingham survey) was carried out in Jamaica between 1979-80, (Cruickshank et al 1981), the findings of which showed that variances in both systolic and diastolic blood pressures were not explained by ethnicity. A review of studies carried out in the Caribbean highlighted the inconsistencies in findings in the region (Forrester and Wilks 1995)

Morbidity and Mortality Discussed

The mortality findings reported by Marmot Adelstein, Bulusu (1984) and Balarajan and Bulusu (1990) have been fundamental to the perception of blood pressure and African-Caribbeans, and researchers with serious interest in the area would have consulted these authoritative documents. The later *Health of the Nation* publication, *Ethnicity and Health* (1993) served as acknowledgement at government level of the importance of these mortality statistics. It is from these 'primary' documents that investigations of the nature of this research project are justified. However, there is an inherent problem. As shown earlier in this discussion, surveys and other studies relating to morbidity have not always supported the mortality findings. One of the foremost authorities in the field, Cruickshank, stated quite clearly that: ***"Contrary to the consistent findings in the USA, average blood pressure (BP) levels among Black populations in the Caribbean, West Africa and Britain do not differ substantially from those in Whites"***(Cruickshank 1989 p. 268). If this is accepted, then explanations must be sought concerning the high mortality rates, which have not always been reflected in other (independent) research findings. A few possible 'explanations' are considered below.

A Questioning of the 'Evidence' of Hypertension in African-Caribbeans

It has been found that African-Caribbeans with hypertension were more likely to be on treatment that their White counterparts (Haines et al, 1987; Chaturvedi 1993). The north west London general practice survey of routine medical records conducted by Haines and colleagues, was unable to establish any significant differences in age related blood pressure between Blacks and Whites, even though the records showed that more Blacks were being treated for hypertension. The question is posed as to whether ***GPs were more likely to prescribe medication to African-Caribbeans than to Whites with similar hypertension symptoms.*** While an answer to this question is not forthcoming, it is deemed likely that many GPs may have prematurely classified African-Caribbeans as hypertensive and placed them on medication unnecessarily. The possible outcomes of such actions are considered further in this discussion. Questions surrounding accuracy of blood pressure measurements are also pertinent.

Blood Pressure Measurements - Not Dependable

Stewart and Padfield (1994) carried out an audit of blood pressure measuring techniques in hospitals and general practices. They observed that although the need for careful blood pressure measurements to minimise error and obtain accurate results has been ***"known almost since the inception of the technique, yet the skill seems not to have been learnt by many practising medical and ancillary staff today"*** (p.422). Problems with blood pressure measuring equipment in common usage have been found widespread. Burke et al (1982) reported that as many as half of hospital mercury sphygmomanometers were found defective and/or poorly maintained. The Burke team, and later, Mckay et al (1990), also found that the aneroid sphygmomanometers which are in common use in general practice, were more likely to be defective. Few would disagree that multiple blood pressure

readings, within and across visits, are required to estimate an individual's blood pressure (Pickering and James 1989). Rosner and Polk (1981) concluded that at least 2 readings obtained on each of 3 visits are necessary before classifying an individual as hypertensive. Pickering (1994) felt that this was not the practice of most general practitioners, as he concluded, ***"the measurement of blood pressure is much too serious to be left to physicians"***(p.34).

WHITE COAT (CLINIC) HYPERTENSION

Closely related to the inaccuracies in blood pressure measurements is 'white coat' hypertension. White-coat' hypertension is persistently raised clinic pressure together with a normal ambulatory pressure, (Pickering 1994) and affects about 20% of borderline hypertensives (Hoegholm et al 1992; Pickering et al 1994). The effect of the physician's white coat the on blood pressure was first recorded by Ayman and Goldshine (1940), who reported that blood pressures of hypertensive patients measured in their own home were substantially lower than that recorded in the clinic. A number of researchers including Mancia (1987) Hoegholm et al (1992); and Pickering et al (1990), have since confirmed what is known as the 'alerting reaction' associated with an increase in blood pressure. These investigators have concluded that 'white-coat' hypertension affects both normotensives and hypertensives of any age or gender, and that the effect can, and very often persists with repeated measures over time. Pickering et al (1990) postulated that white-coat hypertension becomes perpetuated through 'classical conditioning'. This habituation leads to a diminution of the defence response, while conditioning leads to a perpetuation of it as the white-coat effect.

It is within the GP/clinic environment that African-Caribbeans, like other members of the society, are most likely to have been measured and ultimately diagnosed hypertensive. The unreliability of blood pressure measurements based on either inaccurate measuring or the 'white-coat' effect, or most likely a combination of both, is a real issue. Pickering and James (1989) noted that anxiety associated with a clinic visit is a plausible explanation for white coat hypertension. They also suggested that ***"one of the initiating factors may be patient's view of the physician as a potential threatening authority figure, which results in the increased clinic pressure"*** (p.S67).

In discussing the attitudes and general approach of doctors, Pearson (1989) has pointed to strong evidence that patients' relationships to their physicians are generally determined by their social, economic and cultural situation. Patients from the lowest socio-economic background are less likely to have a positive and active input in their consultations. Immigrant ethnic minority groups, including African-Caribbeans, would normally tend to have even less communication. Low socio-economic status coupled with racism and discrimination makes them less likely to be trusting and confident in the medical practitioners. Pearson wrote, ***"In a society in which racism is deeply ingrained, ethnic minorities may have particularly bad experiences and low expectations of doctors and the NHS"*** (p.76).

There have been numerous direct and indirect (anecdotal) reports from immigrant African-Caribbeans stating that they were generally not given the opportunity to relate to their GPs and other NHS medical personnel in a manner satisfactory to

them. Doctors were most often deemed aloof, and lacking in empathy, and many have found 'going to the doctor' to be stressful rather than comforting. In general these anecdotal reports suggest disempowerment. Pearson supports these points when she pointed out particularly with respect to ethnic minorities: ***"people may become aware that they are labelled as 'awkward' if they challenge the submissive culture of being a patient"*** (p.77). This point is somewhat acknowledged in the preface of the Publication *'Ethnic Factors in Health and Disease'* edited by Cruickshank and Beevers (1989). The editors wrote:

> ***"An important challenge to doctors will be a change in attitude to being questioned. In particular...the 'informed-doctor-dispensing-to-ignorant-patient' approach will have to give way to an appreciation that many 'minority' patients will know more of recent developments in their condition than their doctors"*** (p.viii Preface).

The above discussion points to the likelihood that many of the (first generation) African-Caribbean immigrants on whom the mortality figures of the 1970s to1980s were based, may have been more predisposed to 'white coat' hypertension, than their White counterparts. The prognostic importance of isolated clinic hypertension (with regards to white-coat hypertensives generally) was recognised by Mancia and Zanchetti (1996). They pointed out that repeated occurrences of brief increases in blood pressure might have some clinical significance because brief blood pressure increases in animals have been shown to precede established hypertension and lead to organ damage. Mancia and Zanchetti that:

> ***"Even in the presence of a normal or low 24h average blood pressure, the possibility of an increased*** (cardiovascular) ***risk possibly due to a high number of blood pressure peaks or increased blood pressure variability cannot be excluded",*** (p. 1050).

If African-Caribbeans were more predisposed to isolated clinic (white-coat) hypertension, then this may also have led to increased cardiovascular risk. Perhaps

the origin of the high mortality rate from hypertension related conditions, especially stroke, found in the 1970s-1980s could be found thorough further probing in this direction..

Anecdotally, there is evidence to suggest that many, if not most, African-Caribbeans who are eventually diagnosed hypertensive and subsequently placed on medication did not initially go for consultation on an hypertension related illness. Often it was during 'routine' blood pressure checks that the elevated blood pressure was detected. It is reasonable to question how often these cases are simply white-coat effects. How often might it have been the case that blood pressure was temporarily raised based simply on not being well, or on the anxiety associated with being ill and the myriad of adverse effects that may result, for example, time off work? African-Caribbeans could have been showing what has been described as the 'alerting response', which can cause a substantial rise in pressure of some individuals, (Pickering et al 1988). It is further likely that being told, 'alarmed' that one's blood pressure was high could create further anxiety, which would perpetuate the high readings, both within and across visits. A non-empathetic GP or medical practitioner would only serve to increase the 'white-coat' effect. Pickering reported that a substantial number of white-coat hypertensives would continue to give unrepresentative values at clinic readings no matter how many measurements are taken. This is probably more likely in what is recognised often to be 'undesirable' environments within the NHS system, especially for ethnic minority groups such as Caribbeans.

There is also a likelihood that many of the African-Caribbean 'hypertensives' should have been more properly diagnosed as labile hypertensives, as opposed to 'true' hypertensives.

WERE AFRICAN-CARIBBEANS 'VICTIMS' OF THE J-CURVE PHENOMENON

Early studies have reported that African-Caribbeans with hypertension were more likely to be taking medication for the control of hypertension than Whites were (Haines 1987). The 1990s Brent study Chaturvedi et al (1993) also found this to be the case. This suggests that African-Caribbeans could have been placed on hypertension drug treatment more readily than Whites, and consequently more unnecessarily. Misdiagnosed (African-Caribbean) hypertensives placed on medication could have developed a dependency and may have continued to show high readings. In this situation the over-treatment could have exaggerated and perpetuated the high blood pressure, leading to J-curve, (Hanson 1991) whereby medication lowered the blood pressure initially, but then contributed to continued elevation, which then became controllable only with continued use.

Most, if not all, pharmaceutical drugs have side effects, and this applies to drugs used in the therapy of hypertension (Swales 1995). The side effects of long-term use of the variety of medication prescribed for the lowering of blood pressure may not be fully known. Research has shown that African-Caribbeans and other Blacks (especially African-Americans) do not respond in the same (favourable) way to hypertension medication as Whites. For instance, Cruickshank et al (1988) reported that their study confirmed the limited effect of β-blockers and Ace-inhibitor drugs for treating hypertension in Blacks. The commonly used Beta-blocker metoprolol

failed to lower blood pressure in the Black subjects at all despite heart rates being significantly reduced; but the drug was effective in the Whites. The Cruickshank team concluded that ***"as yet, no ideal monotherapy exists for hypertension in Black patients"***(p 1155), and that ***"there are no ideal antihypertensive drugs for black patients***" (p. 1159). Materson et al (1993) reported that the entire class of calcium antagonists, including nifedipine and diltiazem, were relatively more effective than other classes of second line drugs in Blacks.

General information of the ineffectiveness of certain drugs on African-Caribbeans and the resultant adverse effects of these contraindicated drugs has only been widely published in the past 10-15 years. This was after the mortality figures that highlighted African-Caribbeans to be of exceptionally high-risk were computed. The side effects of the drugs on deceased hypertensive African-Caribbeans will never be known. It is also unlikely that their full hypertensive history, including pre-diagnostic conditions will be unearthed. Therefore, the largely hypothetical postulations made in this discussion are likely to remain just that, that is, pure speculation. None-the-less based on the inconsistencies of the post 1970s-80s findings, the issues raised here are pertinent. African-Caribbeans may have been misdiagnosed, wrongly prescribed, and over treated. A point of issue here is that (anecdotally), a number of Caribbeans who have resettled in their native lands, mostly after retirement from their UK jobs, have reported normalisation of their blood pressures, even after many years of taking prescribed blood pressure medication.

In this discussion, the possibility that the high mortality rates from hypertension related disease in the 1970s-1980s may not have represented a true reflection of the 'essential' hypertension prevalence rates in African-Caribbeans was explored. Many African-Caribbeans could have been wrongly diagnosed initially, and their 'recorded' high blood pressure resulting from a combination of poor measuring practices and the 'white-coat' effect. Perhaps also some were prone to *labile* hypertension, defined here to mean unstable blood pressure, liable to change over short periods. Labile hypertension could be triggered by such unsettling factors as migration. This was recognised in an *OPCS* report, (Marmot, Adelstein, Bulusu 1984) in which the authors stated that:

> ***"The process of migration itself involves major changes that may affect disease risk. Economic and social strain, breaking of family ties, changes in smoking, drinking and dietary practices may all occur... it is possible that the stresses of migration may have contributed to hypertension in West Indians"*** (p.73).

These possibilities can only be examined by further in-depth studies of the African-Caribbean population.

The aetiology of hypertension in African-Caribbeans remains an enigma, and the results of this study could be said to contribute to the puzzle. The conclusion by the authors of the Brent Study (Chaturvedi et al 1993) who stated that ***"the reasons for these ethnic differences in blood pressure and the effectiveness of measures to prevent hypertension in people of African-Caribbean descent remain to be established"*** (p.95), is considered sound. This conclusion would also apply to the most common, and perhaps most plausible explanations, such as *salt intake* and *sodium insensitivity*. Information on the salt intake in African-Caribbeans is lacking, but there is no evidence to suggest that there is any major difference to that of the

general population, that is, an average daily consumption of 150-200 mmol. Also, as shown earlier in the discussion (page 185-186) there is also no conclusive evidence that African-Caribbeans are more salt sensitive.

Blood pressure is multifactorial. That the pathogenesis of hypertension encompasses varied combinations of environmental and genetic factors is generally accepted and perhaps one of 'informed common-sense'. None-the-less it seems reasonable that, as with many other diseases, some people may be (genetically), more predisposed to hypertension than others are. The effect of environmental factors on blood pressure, while preventable, could be unavoidable in individuals who have the greatest genetically pre-determined susceptibility. However, the view that hypertension in African-Caribbeans (and Blacks) is primarily genetic is not accepted, if only because hypertension is very rare in primitive rural societies in Africa. Further, efforts to prove this overwhelming genetic component has not been scientifically validated, including 'salt sensitivity' theories, such as those proposed by Wilson and Grim (1991).

The wide concern about the 'problem' of hypertension in African-Caribbeans is acknowledged and not deemed to be unfounded, based on the past mortality records. However, the 'problem' may not be as severe in current African-Caribbeans, and thus the 'alarming' concern may not be justified. Younger Black British persons of Caribbean origin, may not have a greater predisposition to hypertensive disease than their European counterparts, and as such the 'problem' may have been minimised or phased out as new generations of Black British Caribbeans emerged. The possibility of an association between the high rates of hypertension in African-

Caribbeans and the effects of migration into an alien racist and hostile environment during the 1950s to 1960s exists. Younger generations of African-Caribbeans would not have had such an experience, or at least significantly less of it. One can conclude (controversially no doubt), that perhaps the problem is diminishing to the level whereby African-Caribbeans can no longer be considered a 'special' population with regards to hypertension. A 1980s study of African-Caribbean and European school Children in Birmingham found no differences in blood pressures between the two ethnic groups, (De Giovanni et al 1983).

The hypertension prevention and management programmes geared at the general population, promoting healthy lifestyles, might be just as applicable to African-Caribbeans. This is supported by a number of (adult) studies cited within this thesis which found no differences in blood pressures between African-Caribbeans and Europeans, or did not find a high prevalence of hypertension in African-Caribbeans. Where differences were found, they were generally with older African-Caribbeans. There may be no need for any 'extra' or 'different' programmes for African-Caribbeans.

In Conclusion: outside of body mass index, the African-Caribbeans 'undesirable' lifestyle factors, particularly health behaviour, of the subjects in this study did not appear to have a marked adverse effect on their blood pressure. As discussed earlier, **body mass index** has provided the most consistent explanation for any differences found in blood pressure between African-Caribbeans (especially middle-aged women), and Europeans (Cruickshank 1993). The findings of this study support this

position. It is recommended that this be an area of emphasis on future research on hypertension in African-Caribbeans.

A note on the 'Competing Cause for Death': Cruickshank's (1993), competing cause for death position with respect to African-Caribbeans and stroke mortality was made based on the low mortality from ischaemic heart disease (IHD) in that ethnic group. Cruickshank calculated that this 'deficit' in deaths from ischaemic heart disease meant that more African-Caribbeans would become available to die from stroke. He opined that further evidence of the relevance and importance of this 'competing cause for death' was that the average age at death from stroke was higher than from ischaemic heart disease.

This argument appears to be sound and supports the position that hypertension in African-Caribbeans most probably reflects the age-related rise in blood pressure that is found in most westernised populations. The competing cause for death theory with regards to African-Caribbeans and hypertensive related mortality only serves as to highlight further that, since African-Caribbeans are not dying at a relatively young age from ischemic heart disease, then they are more likely to die of other vascular diseases related to 'old age'. Hypertensive related conditions, especially stroke, fall into this category. More investigations are needed into blood pressure and age in African-Caribbeans. For instance, and important question is whether there is a greater prevalence of hypertension in young African-Caribbeans than is found in a corresponding European population.

10.3 RECOMMENDATIONS FOR FUTURE RESEARCH

The subject of hypertension in African-Caribbeans is relatively unresearched. Further research is needed in all areas of the topic. With regard to health behaviour there would appear to be a need for more studies which pay particular attention to body mass index. More information is needed on the general lifestyle of African-Caribbeans across generations, especially UK-born versus Caribbean-born. Particular focus could be placed on dietary habits, exercise habits, and alcohol and smoking habits. Until there is a greater number of diverse studies on the lifestyle of African-Caribbeans, it will be difficult to make 'conclusive' judgements concerning the impact of their lifestyle on their blood pressures.

There was a relative significance of education level and job grade to blood pressure, whereby those at the lower end tended to have higher blood pressures. Perhaps a useful study might be one of direct comparison between a group of highly educated subjects in executive level jobs, compared to and with a group of subjects, say factory workers who were less educated. Long term unemployed persons could also be included in the study.

A study on white-coat hypertension in African-Caribbeans could be valuable, with particular attention to the older African-Caribbeans' perception of the their general practitioners, and other medical practitioners who may have measured their blood pressures. Questions relating to blood pressure measurement techniques used would be very important.

Finally, a controlled study by age group would be very useful, especially with regard to the theory of 'competing cause for death'.

The author of this thesis hopes to continue research within this field, in one of the above areas.

-End -

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Appendices

STATISTICAL ANALYSIS APPENDICES

SH **Heart rate**: frequency distribution, descriptive and exploratory statistics, mean SBP

SI **BMI**: frequency distribution, descriptive and exploratory statistics, mean SBP

SJ **Subjects' and Subjects' family blood pressure history**: frequency distribution, descriptive statistics, mean SBP

SK **Qualifications and Job Grade**: frequency distribution, descriptive statistics, mean SBP

SL **Dietary habits**: frequency distribution, descriptive statistics, mean SBP, cross tabulations

SM **Alcohol**: frequency distribution, descriptive statistics, mean SBP

SN **Smoking**: frequency distribution, descriptive statistics, mean SBP

SO **Accommodation**: frequency distribution, descriptive statistics, mean SBP

SP **Racism**: frequency distribution, descriptive statistics, mean SBP

SQ **Sessions completed**: descriptive statistics, mean SBP

A

THE HEALTH OF THE NATION

ETHNICITY AND HEALTH

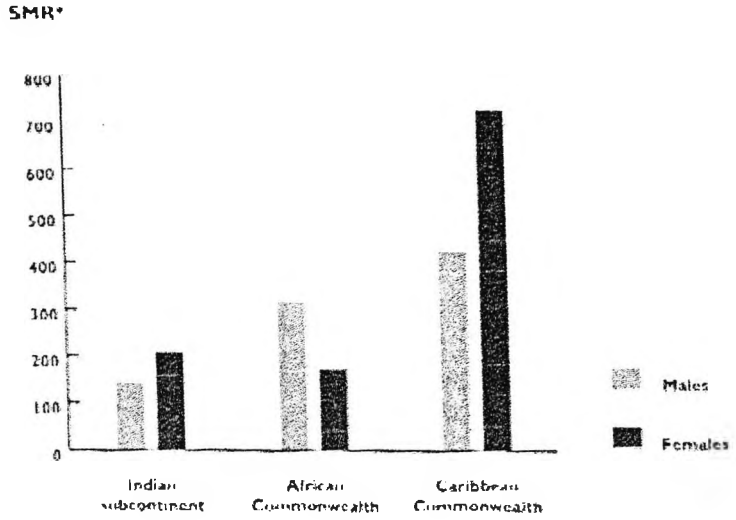
A GUIDE FOR THE NHS

HYPERTENSION

African-Caribbeans and Asians - particularly the former - have a higher than average propensity for hypertension[1,2,3]. Mortality from hypertensive disease in England and Wales during 1979-83 was four times greater than the national average in Caribbean men, and seven times greater in Caribbean women[4] (Figure 8). Persons born in Africa and the Indian subcontinent also experienced large excesses.

Mortality from hypertension by country of birth (ages 20-69)
England and Wales, 1979-83

Figure 8



*Standardised mortality ratios with England and Wales = 100

RISK FACTORS

In addition to the mortality reduction targets, the White Paper also specifies targets for risk factors associated with CHD and stroke, namely smoking, drinking, fat intake, obesity, and blood pressure levels. Information about lifestyle and diet among ethnic minorities is scanty, and the findings are not always consistent. The latter is not surprising since the sub-groups of Asian and black people are not homogeneous, and show considerable diversity in terms of culture, lifestyle, social class and economic status.

HYPERTENSION IN AFRO-CARIBBEANS IN THE U.K. - PHD PROJECT

QUESTIONNAIRE

I.D.#_____ DATE_____ VENUE_____

MEASUREMENTS (SECT. 1)

GENDER _____ AGE_____ HGT_____ WGT_____ BMI_____

ARE YOU: [RIGHT] [LEFT] HANDED

SBP (S)	(1)_____	(2)_____	(3)_____	LOWEST_____
DBP (S)	(1)_____	(2)_____	(3)_____	LOWEST_____
RHR (S)	(1)_____	(2)_____	(3)_____	LOWEST_____
SBP (L)	(1)_____	(2)_____	(3)_____	LOWEST_____
DBP (L)	(1)_____	(2)_____	(3)_____	LOWEST_____
RHR (L)	(1)_____	(2)_____	(3)_____	LOWEST_____
MBP (S)	(1)_____	(2)_____	(3)_____	LOWEST_____
MBP (L)	(1)_____	(2)_____	(3)_____	LOWEST_____
MBP (S)	(1)_____	(2)_____	(3)_____	LOWEST_____
MBP (L)	(1)_____	(2)_____	(3)_____	LOWEST_____

COMMENTS_____

_____

_____

I.D._____

BIRTH (SECT. 2)

1. MOTHER'S BIRTH PLACE_____ 2. HOW LONG LIVE UK_____

3. FATHER'S BIRTH PLACE_____ 4. HOW LONG LIVE UK_____

5. YOUR BIRTH PLACE_____ 6. HOW LONG LIVE UK_____

7. MOTHERS'S RACE AFR_____ WHITE_____ INDIAN_____ OTHER_____

8. MOTHER'S PROFESSION _____

9. FATHERS'S RACE AFR_____ WHITE_____ INDIAN_____ OTHER_____

10. FATHER'S PROFESSION _____

EMPLOYMENT (SECT. 3)

11. WHAT IS YOUR HIGHEST ACADEMIC QUALIFICATION_____

12. WHAT IS YOUR HIGHEST PROFESSIONAL QUALIFICATION_____

13 ARE YOU EMPLOYED [YES] [NO]

14. MORE THAN 2 YEARS [YES] [NO]

15. HOW MANY YEARS HAVE YOU WORKED WITH LAMBETH SOCIAL SERVICES____

16. PRES. JOB TITLE_____ 17. HOW LONG_____

18. PREV. JOB TITLE_____ 19. HOW LONG_____

20. HOW MUCH DID YOU LIKE YOUR PREVIOUS JOB [V.MUCH] [LIKED IT]
[FAIRLY WELL] [NOT VERY MUCH] [NOT AT ALL]

21. HOW MUCH DO YOU LIKE YOUR PRESENT JOB [V.MUCH] [LIKE IT]
[FAIRLY WELL] [NOT VERY MUCH] [NOT AT ALL]

I.D._____

22. DID YOU WORK <20 H/P/W IN YOUR PREVIOUS JOB [YES] [NO]

23. DO YOU WORK <20 H/P/W IN YOUR PRESENT JOB [YES] [NO]

24. DID YOU REGULARLY WORK >45 H/P/W IN YOUR PREV. JOB [YES] [NO]

25. DO YOU REGULARLY WORK >45 H/P/W IN YOUR PRES. JOB [YES] [NO]

26 HOW OFTEN DID YOU FEEL STRESSED IN YOUR PREVIOUS JOB

[EVERY DAY] [2-3 P.W] [1 P.W.] [S/TIMES] [RARELY] [NEVER]

27. HOW OFTEN DO YOU FEEL STRESSED IN THIS JOB.

[EVERY DAY] [2-3 P.W] [1 P.W.] [S/TIMES] [RARELY] [NEVER]

28. HOW HAPPY WERE YOU IN PREV. JOB [VERY] [HAPPY] [FAIRLY] [NOT]

29. HOW HAPPY ARE YOU IN THIS JOB [VERY] [HAPPY] [FAIRLY] [NOT]

30. HOW OFTEN DO YOU TAKE WORK HOME [EVERY DAY] [2-3 P.W]

[1 P.W] [S/TIMES] [RARELY] [NEVER]

31. DO YOU FORGET ABOUT WORK ONCE YOU LEAVE THE WORK ENVIRONMENT

[I DEFINATELY DO] [MORE OFTEN THAN NOT I DO]

[GENERALLY I DON'T] [I DEFINATELY DON'T] [NOT SURE]

32. HOW SECURE DO YOU FEEL IN YOUR PRESENT JOB [VERY SECURE]

[SECURE] [FAIRLY SECURE] [NOT VERY] [NOT AT ALL]

I.D._____

HEALTH (SECT 4.)

33. HAVE YOU SUFFERED FROM ANY OF THE FOLLOWING IN THE PAST 2 YRS:

HYPERTENSION_____DIABETES_____ARTHRITIS_____ASTHMA_____

FATIGUE_____HEADACHES_____DIZZINESS_____ULCER_____

INSOMNIA_____STRESS_____ANXIETY_____BLURRING_____

FAINTING_____CANCER_____ANAEMIA_____LUNG-ROBLEMS_____

HEART-PROBLEM_____EPILEPSY_____DEPRESSION_____KIDNEY-PROBLEM_____

BLOOD-DISORDER_____BOWEL-PROBLEMS_____BREATHLESSNESS_____

OTHER_____

34. HOW WOULD YOU DESCRIBE YOUR GENERAL HEALTH OVER THE PAST 2 YEARS: [EXCELLENT] [VERY GOOD] [GOOD] [FAIR] [POOR]

35. ARE YOU NOW ON THE CONTRACEPTIVE PILL [YES] [NO] [N/A]

36. HAVE YOU BEEN ON THE PILL IN THE PAST 2 YRS [YES] [NO] [N/A]

37. ARE YOU TAKING ANY PRESCRIBED MEDICATION [YES] [NO]

38. WHAT FOR _____

39. ARE YOU TAKING ANY UNPRESCRIBED MEDICATION [YES] [NO]

40. WHAT FOR _____

41. HAVE YOU HAD A FULL MEDIC. CHECK IN THE LAST 2 YRS [YES] [NO]

42 HAVE YOU HAD A BP CHECK IN THE LAST 2 YEARS [YES] [NO]

43. HAVE ANY OF YOUR GRAND-PARENTS HAD HIGH BP

[0] [1] [2] [DON'T KNOW]

44. HAVE ANY OF YOUR GRAND-PARENTS HAD A STROKE

[0] [1] [2] [DON'T KNOW]

I.D._____

45. HAVE ANY OF YOUR **PARENTS** HAD HIGH **BP**

[MOM] [DAD] [DON'T KNOW] [NO]

46. HAVE ANY OF YOUR **PARENTS** HAD A STROKE

[MOM] [DAD] [DON'T KNOW] [NO]

47. HAVE YOU EVER BEEN PRESCRIBED MEDICATION FOR BP [YES] [NO]

48. HAVE YOU EVER HAD **HIGH BP** [YES] [NO]

49. WHEN_____

50. HAVE YOU EVER HAD **LOW BP** [YES] [NO]

51. WHEN_____

52. DO YOU KNOW WHAT IS CONSIDERED TO BE NORMAL BP [YES] [NO]

53. DO YOU KNOW WHAT YOUR BP MEASUREMENTS ARE [YES] [NO]

54. HAVE YOU EVER CHECKED YOUR BP YOURSELF [YES] [NO]

55. WOULD YOU CHECK YOUR BP YOURSELF IF TAUGHT [YES] [NO]

56 DO YOU KNOW WHAT A PULSE RATE IS [YES] [NO]

57. DO YOU KNOW HOW TO TAKE THE PULSE [YES] [NO]

58. DO YOU KNOW WHAT NORMAL PULSE IS [YES] [NO]

59. DO YOU KNOW YOUR NORMAL PULSE RATE [YES] [NO]

60. HOW MANY **SISTERS**_____ DO YOU HAVE

61. HOW MANY **BROTHERS**_____ DO YOU HAVE

62. HOW MANY OF YOUR SISTERS HAVE HAD HIGH BP _____ [D/N]

63. HOW MANY OF YOUR BROTHERS HAVE HAD HIGH BP _____ [D/N]

I.D._____

SMOKING HABITS (SECT. 5)

64. DC YOU NOW SMOKE CIGARETTES [YES] [NO]

65. WERE YOU EVER A SMOKER [YES] [NO]

66. HOW MANY YEARS HAVE YOU STOPPED SMOKING _____ [N/A]

67. HOW MANY CIGARETTES DID/DO YOU SMOKE PER DAY _____ [N/A]

68. AGE STARTED SMOKING [<18] [18-21] [22-25] [>25] [N/A]

69. DO YOU SMOKE MORE NOW THAN 2 YRS AGO

[MORE] [LESS] [SAME] [NOT SURE] [N/A]

70. HOW FREQUENTLY ARE YOU IN A ROOM WHERE SOMEONE ELSE IS SMOKING

[EVERY DAY] [2-3 P.W] [1 P.W] [RARELY] [VERY RARELY]

71. DOES ANYONE IN YOUR HOUSEHOLD SMOKE AROUND YOU [YES] [NO]

ALCOHOL HABITS (SECT. 6)

72. DO YOU NOW DRINK ALCOHOL [YES] [NO]

73. DID YOU EVER DRINK ALCOHOL [YES] [NO]

74. DO YOU DRINK WHEN YOU GO OUT [YES] [NO] [N/A]

75 DO YOU DRINK AT HOME [YES] [NO] [N/A]

76. HOW MANY YEARS AGO DID YOU STOP DRINKING_____ [N/A]

77. HOW MANY UNITS PER WK. DO YOU DRINK _____ [N/A]

78. WHAT AGE DID YOU START DRINKING

[<18] [18-21] [22-25] [OVER 25] [N/A]

I.D._____

79. DO YOU DRINK MORE NOW THAN 2 YRS AGO.

[MORE] [LESS] [SAME] [DON'T KNOW] [N/A]

80. DO YOU EVER DRINK BECAUSE YOU ARE DEPRESSED

[YES] [NO] [SOMETIMES] [HAVE DONE]

FITNESS (SECT. 7)

81. ARE YOU NOW PARTICIPATING IN AN EXERCISE ACTIVITY [YES] [NO]

82. HOW MANY YEARS HAVE YOU BEEN EXERCISING REGULARLY _____

83. WHAT EXERCISE ACTIVITIES DO YOU USUALLY DO_____

84. HOW OFTEN PER WEEKS [<1] [1] [2] [3] [4] [5] [6] [7]

85. HOW MANY MINUTES [<10] [15] [20] [30] [45] [60] [90] [>120]

86. WERE YOU EVER A COMPETITIVE ATHLETE [YES] [NO]

87. ARE YOU NOW A COMPETITIVE ATHLETE [YES] [NO]

HOW PHYSICALLY ACTIVE ARE YOU AT:

88 WORK - [VERY] [FAIRLY] [NOT VERY] [NOT AT ALL]

89. HOME - [VERY] [FAIRLY] [NOT VERY] [NOT AT ALL]

90. WK/ENDS - [VERY] [FAIRLY] [NOT VERY] [NOT AT ALL]

91. HAVE YOU LOST WEIGHT WITHIN THE PAST 2 YEARS

[YES] [NO] [NOT SURE]

92. HAVE YOU LOST WEIGHT AND KEPT IT OFF IN THE LAST 2 YEARS

[YES] [NO] [NOT SURE]

I.D._____

93. HAVE YOU LOST WEIGHT AND REGAINED IT IN THE LAST 2 YEARS

[YES] [NO] [NOT SURE]

94. HAVE YOU GAINED WEIGHT IN THE PAST 2 YEARS

[YES] [NO] [NOT SURE]

95. HAVE YOU GAINED WEIGHT AND KEPT IT ON IN THE LAST 2 YEARS

[YES] [NO] [NOT SURE]

96. HOW CONCERNED ARE YOU ABOUT YOUR WEIGHT

[VERY] [CONCERNED] [FAIRLY] [NOT VERY] [NOT AT ALL]

DIET (SECT.8)

97. HOW MANY HEALTHY MEALS DO YOU USUALLY EAT P/DAY _____

98. ARE YOUR MEALS COOKED WITH SALT

[YES] [NO] [SOMETIMES] [RARELY]

99. DO YOU ADD SALT TO COOKED FOOD

[ALWAYS] [SOMETIMES] [RARELY] [NEVER]

100. WOULD YOUR DESCRIBE YOUR NORMAL DIET AS

[VEGETARIAN] [PART VEGETARIAN] [NOT VEGETARIAN]

101. HOW OFTEN DO YOU EAT RED MEAT P/WK

[0] [1] [2] [3] [4] [DAILY] [S/TIMES] [RARELY]

102. HOW OFTEN DO YOU EAT RED FRIED FOODS P/WK

[0] [1] [2] [3] [4] [DAILY] [S/TIMES] [RARELY]

103. HOW OFTEN DO GENERALLY EAT HEAVILY SEASONED FOODS P/WK

[0] [1] [2] [3] [4] [DAILY] [S/TIMES] [RARELY]

104. HOW MANY CUPS OF COFFEE DO YOU DRINK W/KLY _____

I.D._____

FAMILY (SECT.9)

105 DO YOU HAVE A PARTNER [YES [NO]

106. HOW LONG HAVE YOU HAD THIS PARTNER _____ YEARS

107. WOULD YOU LIKE TO HAVE A PARTNER [YES] [NO] [NOT SURE] [N/A]

108. DID YOU HAVE A PARTNER (IMMEDIATELY BEFORE THIS ONE) [YES] [NO]

109. DO YOU LIVE WITH YOUR PARTNER [YES] [NO] [N/A]

110. DID YOU LIVE WITH YOUR MOST RECENT EX. PARTNER [YES] [NO] [N/A]

111. HOW SUPPORTIVE IS YOUR PARTNER

[VERY] [SUPPORTIVE] [FAIRLY] [NOT VERY] [NOT] [N/A]

112. HOW SUPPORTIVE WAS YOUR EX. PARTNER

[VERY] [SUPPORTIVE] [FAIRLY] [NOT VERY] [NOT] [N/A]

113. HOW MANY CHILDREN DO YOU HAVE _____

114. THE AGE RANGE OF YOUR CHILDREN _____

115. ARE ALL YOUR CHILDREN ALSO YOUR PARTNERS CHILDREN

[YES] [NO] [N/A]

116. HOW MANY CHILDREN DOES YOUR PARTNER HAVE_____ [N/A]

117. THE AGE RANGE OF YOUR PARTNER'S CHILDREN _____[N/A]

118. HOW MANY OF YOUR CHILDREN LIVE WITH YOU _____[N/A]

119. HOW MANY OF YOUR PARTNER'S CHILDREN LIVE WITH YOU _____[N/A]

120. HOW MANY CHILDREN ARE YOU RESPONSIBLE FOR _____

121. ARE YOU A LONE PARENT [YES] [NO] [N/A]

122 WOULD YOU PREFER SHARED PARENTAGE [YES] [NO] [NOT SURE] [N/A]

I.D._____

123. DO YOU CONSIDER YOUR IMMEDIATE FAMILY TO INCLUDE: [PARENT(S)]
[SIBLINGS] [NIECES/NEPHEWS] [AUNTS/UNCLES] [INLAWS] [COUSINS]

124. HOW HAPPY ARE YOU WITH YOUR FAMILY SITUATION
[VERY HAPPY] [HAPPY] [FAIRLY] [NOT HAPPY] [NOT SURE]

125. ARE YOU MORE HAPPY WITH FAMILY NOW THAN IN THE PAST 2 YRS.
[YES] [NO] [SAME] [NOT SURE]

126. DO PREFER A [NUCLEAR] [EXTENDED] [AS IS] FAMILY

127. HAS THERE BEEN ANY TRAGIC EVENTS IN YOUR LIFE IN THE PAST 2 YRS
[YES] [NO]

128. HOW MUCH DID THIS EVENT AFFECT YOUR HEALTH:
[V/MUCH] [QUITE A BIT] [V/LITTLE] [NOT] [NOT SURE]

129. ALL THINGS CONSIDERED HOW CONTENDED ARE YOU WITH LIFE
[VERY] [CONTENDED] [FAIRLY] [NOT CONTENDED]

COMMUNITY (SECT. 10)

130. HOW ADEQUATE IS YOUR LIVING ACCOMMODATION FOR YOUR NEEDS
[VERY] [ADEQUATE] [FAIRLY] [INADEQUATE]

131 HOW MANY YRS HAS IT BEEN ADEQUATE
[<1] [1] [3] [5] [10] [15] [>20] [N/A]

132. HOW MANY YRS HAS IT BEEN INADEQUATE
[<1] [1] [3] [5] [10] [15] [>20] [N/A]

133 WAS YOUR LIVING ACCOMMODATION EVER VERY BAD [YES] [NO]

I.D._____

134 HAVE YOU EVER BEEN HOMELESS [YES] [NO]

135. HAVE YOU BEEN HOMELESS IN THE PAST 2 YRS: [YES] [NO]

136. HOW SAFE DO YOU FEEL IN YOUR COMMUNITY

[VERY] [SAFE] [FAIRLY] [NOT SAFE]

137. DO YOU FEEL MORE SAFE NOW THAN 2 YRS AGO [YES] [NO] [NOT SURE]

138. HAVE YOU LIVED IN A VERY UNSAFE COMMUNITY [YES] [NO]

139. HOW MANY PEOPLE LIVE IN YOUR HOUSEHOLD _____

140. HOW MANY ROOMS (EX. KITCHEN/BATH/WC) ARE THERE_____

141. DO YOU OWN AT LEAST ONE CAR [YES] [NO]

RACISM (SECT. 11)

142. HOW MUCH DOES RACISM NEGATIVELY AFFECT YOUR DAY TO DAY LIVING:

[V. MUCH] [QUITE A BIT] [NOT MUCH] [NOT AT ALL]

143. HOW MANY YRS HAVE YOU NOTICED THIS EFFECT _____

144. HOW MUCH DID RACISM AFFECT YOUR LIFE IN YOUR FIRST YRS IN THE UK [V. MUCH] [QUITE A BIT] [NOT MUCH] [NOT AT ALL]

145. HOW MUCH DID RACISM NEGATIVELY AFFECT YOU AS A CHILD

[V. MUCH] [QUITE A BIT] [A LITTLE] [NOT AT ALL]

146. HAS RACISM AFFECTED YOU ALL YOU LIFE (IN THE UK) [YES] [NO]

147. HOW UNSAFE DOES RACISM MAKE YOU FEEL

[VERY] [QUITE A BIT] [A LITTLE UNSAFE] [NOT AT ALL]

I.D._____

148. HOW MUCH DO YOU SUFFER FROM RACIAL DISCRIMINATION FROM WORK COLLEAGUES [V.MUCH] [QUITE A BIT] [A LITTLE] [NOT AT ALL]

149. HOW MUCH DO YOU SUFFER RACIAL DISCRIMINATION FROM CLIENTS [V.MUCH] [QUITE A BIT] [A LITTLE] [NOT AT ALL]

150 HOW MUCH DO YOU SUFFER RACIAL DISCRIMINATION FROM THE PUBLIC AT WORK: [V.MUCH] [QUITE A BIT] [A LITTLE] [NOT AT ALL]

151. HOW MUCH DO YOU THINK RACISM HAS AFFECTED YOUR CAREER DEVELOPMENT: [V.MUCH] [QUITE A BIT] [A LITTLE] [NOT AT ALL] [NOT SURE]

152. HOW OFTEN DOES RACISM MAKE YOU FEEL TENSE [DAILY] [2-3 P.W] [1 P.W] [S/TIMES] [RARELY] [NEVER]

153. HOW OFTEN DOES RACISM MAKE YOU FEEL INSECURE [DAILY] [2-3 P.W] [1 P.W] [S/TIMES] [RARELY] [NEVER]

154. HOW OFTEN DOES RACISM MAKE YOU FEEL ANGRY [DAILY] [2-3 P.W] [1 P.W] [S/TIMES] [RARELY] [NEVER]

155 HOW MUCH DO YOU THINK THAT RACISM HAS AFFECTED YOUR HEALTH: [V.MUCH] [QUITE A BIT] [A LITTLE] [NOT AT ALL] [NOT SURE]

156. HOW MUCH DO YOU FEEL AT HOME IN ENGLAND: [VERY MUCH] [QUITE A BIT] [NOT MUCH] [NOT AT ALL]

157. DO YOU FEEL MORE AT HOME NOW THAN 2 YRS. AGO [MORE] [LESS] [SAME] [NOT SURE]

158. DO YOU CONSIDER THE U.K. TO BE YOUR HOME [YES [NO] [NOT SURE]

159. WOULD YOU PREFER TO LIVE IN YOUR (PARENTS) COUNTRY OF ORIGIN [YES] [NO] [NOT SURE]

I.D._____

160. DO YOU PLAN TO (RE) SETTLE IN YOUR (PARENTS) COUNTRY OF ORIGIN

[YES] [NO] [NOT SURE]

161. WOULD YOU LIKE TO SETILE IN A CARIBBEAN COUNTRY

[YES] [NO] [NOT SURE]

REASON FOR PARTICIPATION (SECT. 12)

DID YOU CHOOSE TO PARTICIPATE IN THIS STUDY BECAUSE OF:

162.	CONCERN ABOUT YOUR GENERAL HEALTH	[YES]	[NO]
163.	CONCERN ABOUT YOUR BLOOD PRESSURE	[YES]	[NO]
164.	BECAUSE BLOOD PRESSURE RUNS IN YOUR FAMILY	[YES]	[NO]
165.	TO IMPROVE YOUR HEALTH AWARENESS GENERALLY	[YES]	[NO]
166.	TO CONTRIBUTE TOWARDS THE STUDY	[YES]	[NO]
167.	CURIOSITY	[YES]	[NO]

IN YOUR OWN WORDS_____

_____

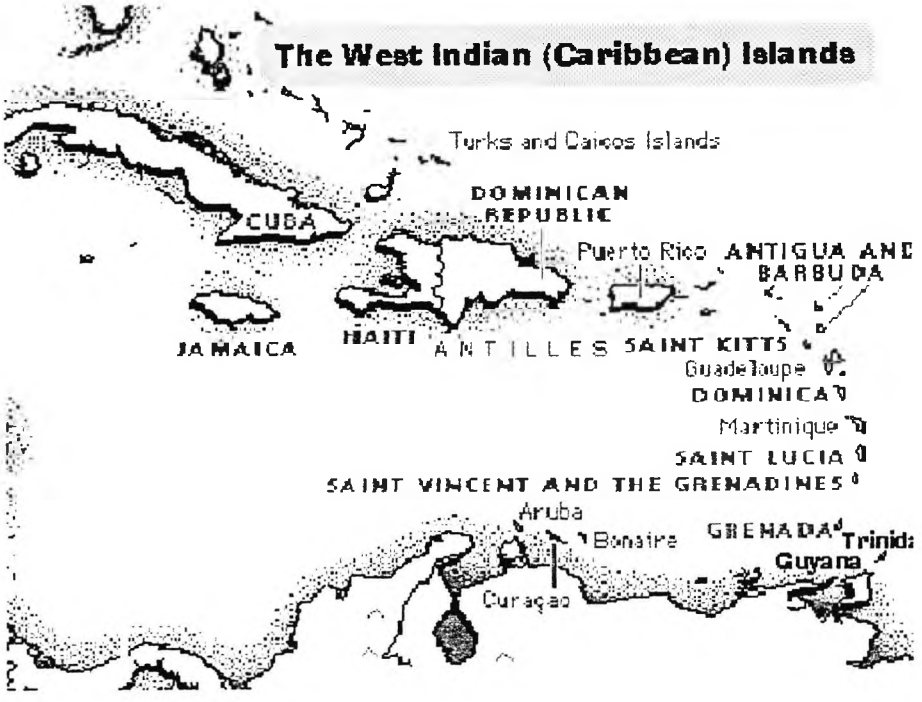
_____

_____

_____

C

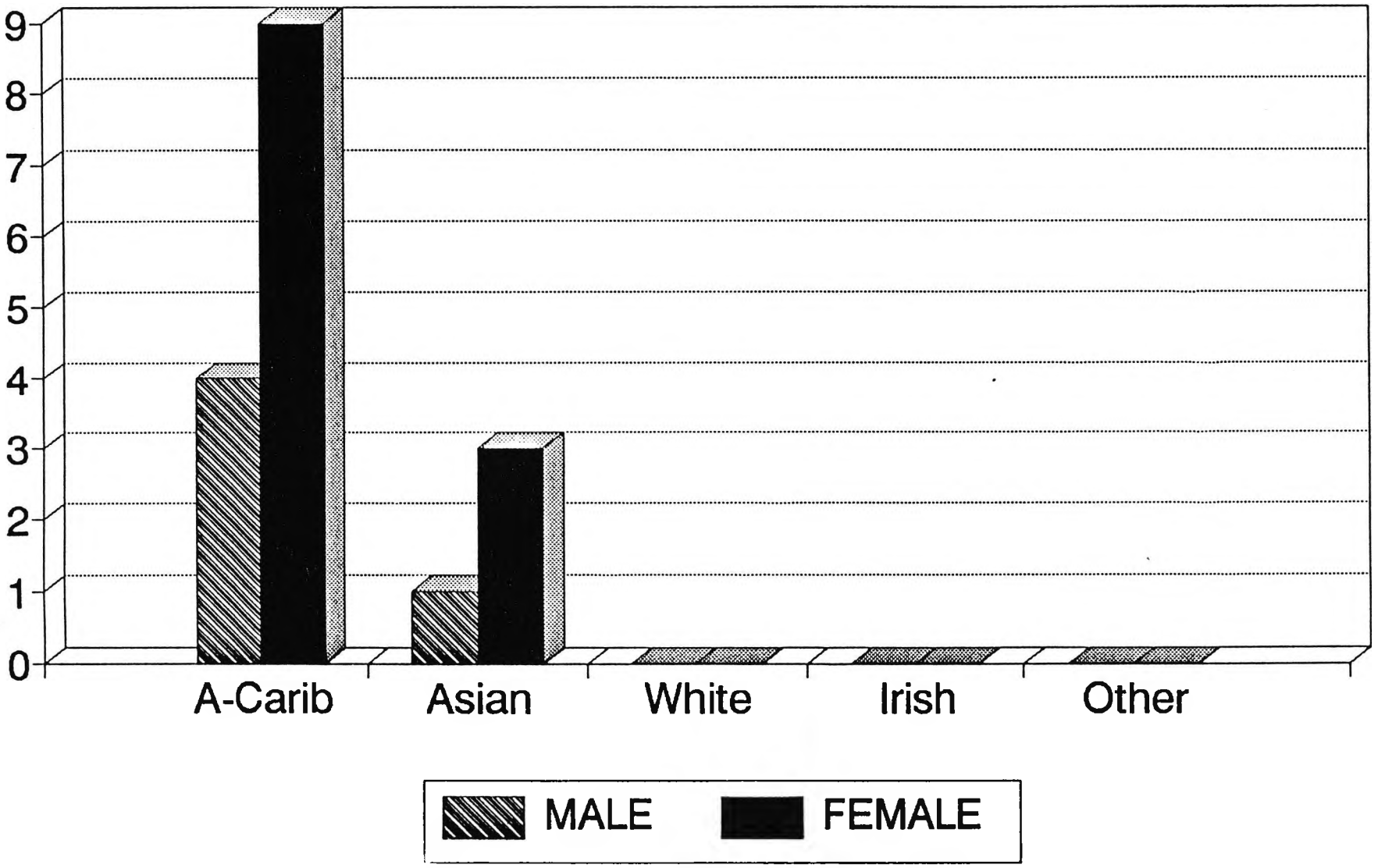
The West Indian (Caribbean) Islands


The West Indian (Caribbean) Islands
Turks and Caicos Islands
DOMINICAN REPUBLIC
CUBA
Puerto Rico
ANTIGUA AND BARBUDA
JAMAICA
HAITI
ANTILLES
SAINT KITTS
Guadeloupe
DOMINICA
Martinique
SAINT LUCIA
SAINT VINCENT AND THE GRENADINES
Aruba
Bonaire
GRENADA
Trinida
Guyana
Curaçao

D

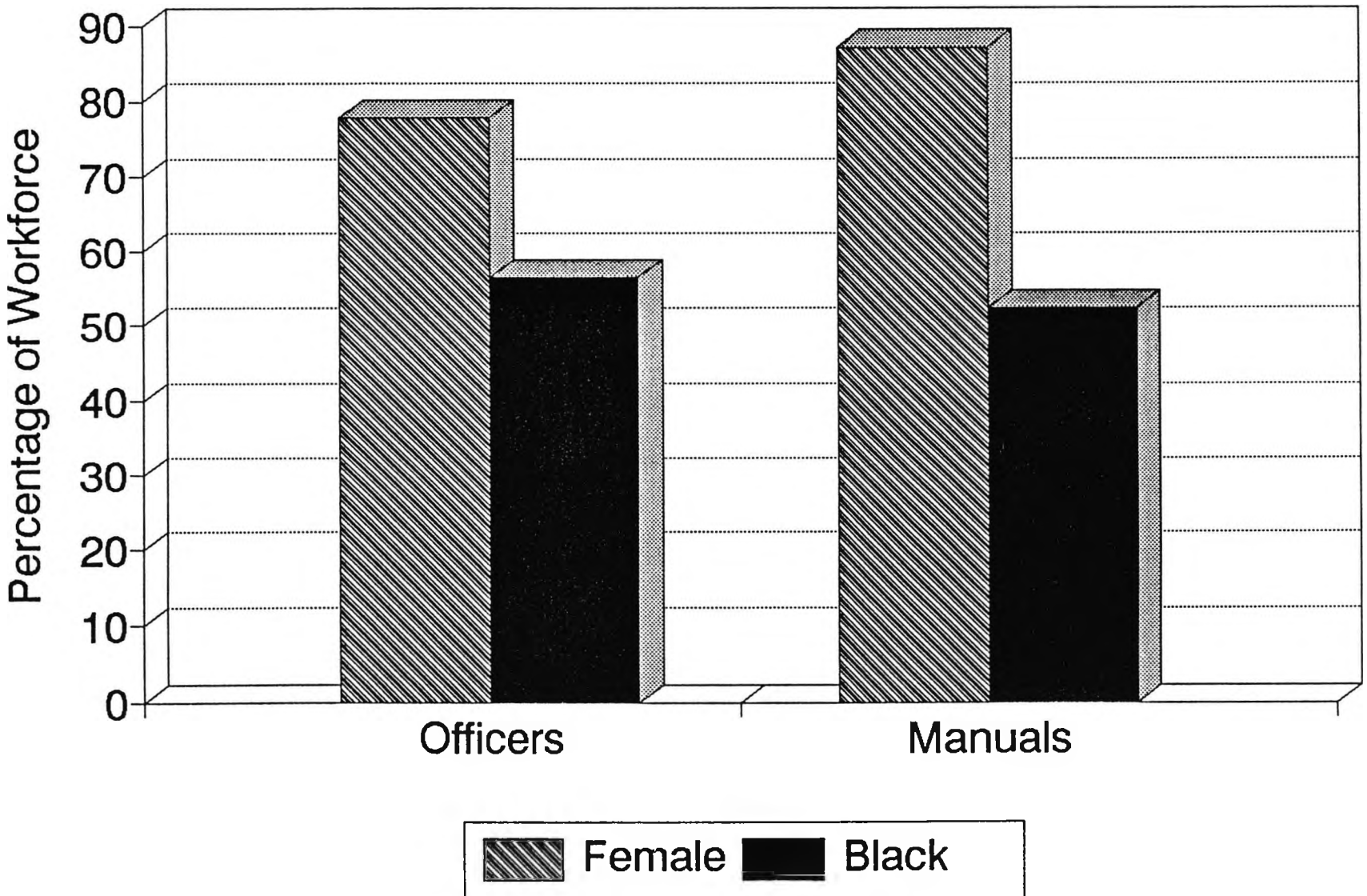
RACE 1993

RACE/GENDER MAKEUP - OFFICERS



DIRECTORATE OF SOCIAL SERVICES 1993

FEMALE/BLACK STAFF - PERCENTAGE IN POST



F

Flat 23, Century House
245 Streatham High Rd.
London SW16 6ER

Tel: 081-677-5975

Mr. David Pope
Director, Social Services
Lambeth Council
Mary Seacole House
91 Clapham High S.
London SW4 7TF

Dear Mr Pope

I am about to begin Phd Studies, researching in the area of **HYPERTENSION IN AFRO-CARIBBEANS IN THE U.K.**, at City University, London.

I have had discussions with Pat Bell, adn Nigel Goldie, concerning my proposal to include Afro-Caribbean employees of your organisation as the main study population of my research project.

Please see attached brief outlines as follows:

- Profile of the researcher
- Why this study
- Aim of the research
- Method

Also Ms. Bell has a copy of the detailed research proposal, and the draft questionnaire.

I hope that you will give the proposal your approval, and look forward to commencing the study.

Yours Sincerely,

Pauline Hylton

March 22, 1994

(For Managers)

9

LAMBETH

Memorandum

TO	ALL AFRO-CARIBBEAN STAFF
Attn	
Your ref	
FROM	DAVID POPE - DSS
Our Ref	DSS/HESD/PB/SM
Tel	EXT 64511
Date	18.4.94

RE: RESEARCH INTO BLOOD PRESSURE IN AFRO-CARIBBEANS

Ms. P. Hylton is undertaking research in the area of Hypertension in Afro-Caribbeans in the UK, leading to a PhD.

Afro-Caribbeans have significantly higher risks of hypertension and stroke than the rest of the U.K. population. This study aims to establish the factors that place Afro-Caribbeans at this greater risk.

I am privileged that Ms. Hylton has selected Afro-Caribbean employees of Lambeth Social Services as her study population. The results of her study will benefit not only Afro-Caribbeans generally, but specifically those who participate in this pilot study, as participants will gain valuable information about blood pressure, as well as management of their own blood pressure.

Please find attached, an outline of the procedure for the participants.

A briefing has been arranged for managers, to give more detailed information of the programme. The briefing will be held on **Friday 13th May 1994**.

I am hoping that as many Afro-Caribbean staff as possible will volunteer for this programme, as I believe it will be a useful exercise for the individual and will benefit the Afro-Caribbean community.

I have agreed for interviews to take place in working hours, subject to the agreement of managers.

Please complete the attached slip and return immediately.

DAVID POPE
DIRECTOR OF SOCIAL SERVICES

srm/docs/memo/HYPERTNSN.

H

L A M B E T H

Memorandum

TO	ALL MANAGERS
Attn	
Your ref	
FROM	PAT BELL - HESD
Our Ref	DSS/HESD/RG/SM
Tel	EXT 64511
Date	20.4.94

URGENT

RE: RESEARCH INTO BLOOD PRESSURE IN AFRO-CARIBBEANS

Please find attached, memo from David Pope and correspondence relating to the above, which is self-explanatory.

Could you please note the enclosed information, giving further details about a briefing day for managers on the **15th May 1994.**

Could you also co-ordinate **urgently**, the circulation of the additional information enclosed to Afro-Caribbean staff in your Division/section.

Please complete the attached slip and return to either Sandra Mills or Pauline Huggan by **10th May 1994**.

Thank you for your assistance.

PBell

PAT BELL
HEAD OF EQUALITIES SERVICE DEVELOPMENT

srm/docs/memo/HYPERTNSN.

(For Managers)

LAMBETH

Memorandum

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DIRECTOR OF SOCIAL SERVICES

srm/docs/memo/HYPERTNSN.

L A M B E T H

Memorandum

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Attn	
Your ref	
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DAVID POPE
DIRECTOR OF SOCIAL SERVICES

srm/docs/memo/HYPERTNSN.

WORKING TOGETHER FOR BETTER HEALTH

RESEARCH INTO
BLOOD PRESSURE
IN THE
AFRO-CARIBBEAN COMMUNITY

A CHALLENGE FOR THE AFRO-CARIBBEAN COMMUNITY TOWARDS CONTROLLING HYPERTENSION AND STROKE

The Directorate of Social Services, Equalities Service Development Division, has developed 'Healthy Alliance' with an Afro-Caribbean Researcher, researching into Hypertension in the Afro-Caribbean community.

The results of this study will not only benefit Afro-Caribbeans generally, but specifically those who participate in this pilot study.

Participants will gain:

* **valuable information about BLOOD PRESSURE**
* **knowledge of individual prevention strategies**
* **information on the management of blood pressure**

* **we would like 150 females and 150 males to participate***

IF YOU ARE OVER AGE 18 AND WORK WITHIN SOCIAL SERVICES, YOU ARE MOST WELCOMED TO PARTICIPATE.

INTERVIEWS ARE PRIVATE AND CONFIDENTIAL, AND MAY BE CONDUCTED AT PARTICIPANTS CONVENIENCE, INCLUDING DURING WORKING HOURS

This research is supported by the Directorate of Social Services as part of its 'HEALTHY ALLIANCE INITIATIVE'

FOR FURTHER INFORMATION, PLEASE CONTACT:

SANDRA MILLS OR PAULINE HUGGAN ON: 071-926 4511

RESPONSE SLIP

RESEARCH INTO BLOOD PRESSURE IN

AFRO-CARIBBEANS

I will/will not be able to attend the Management Briefing Day on 15th May 1994. (Please delete, as appropriate)

I would like more information on the Blood Pressure in Afro-Caribbeans Research Project

[ ] Yes [ ] No (Please tick)

NAME _____

Establishment/address _____

_____

_____

_____

CONTACT NUMBER _____

- -

Please complete and return to:

Sandra Mills, Directorate of Social Services, Equalities Service Development, Room 603, Mary Seacole House, 91 Clapham High Street London SW4 7TF

WORKING TOGETHER FOR BETTER HEALTH

BRIEFING ON

BLOOD PRESSURE IN AFRO-CARIBBEANS

FRIDAY, MAY 13TH , 2PM-4PM

VENUE: 7th floor, Training & Staff Development Unit, Mary Seacole House

INTRODUCTION: Pat Bell - HESD

PRESENTERS:

Ms. P. Hylton - researcher
Dr. E. Ddumba - lecturer

The BRIEFING Forms part of the 'Healthy Alliance' developed between the Directorate of Social Services Equalities Service Development Division and Afro-Caribbean Researcher, Ms. P Hylton.

The briefing is the first in a series on BLOOD PRESSURE in the AFRO-CARIBBEAN COMMUNITY, and is part of the alliance's HYPERTENSION & STROKE PREVENTION STRATEGY.

P.T.O.

Researcher, Ms. P. Hylton and her medical consultant, Dr. E. Ddumba, will lead discussions on:

* **PREVENTION STRATEGIES**
* **MANAGEMENT PROGRAMMES**
* **B.P. MONITORING**

Ms. Hylton will give details of her research programme and outline the role of the Alliance in the project.

Participants will have the opportunity to question Ms. Hylton and to gain a greater insight into the research programme and the contribution that employees of Social Services are being asked to make.

For further information contact:

SANDRA MILLS OR PAULINE HUGGAN ON:

071-926 4511

SOCIAL SERVICES
Director: David A. Pope

LAMBETH

London Borough of Lambeth
Mary Seacole House
91 Clapham High Street
LONDON SW4 7TF

071-926 4563
926 4511

Your Ref:

Our Ref:

Date: 7th June 1994

Dear

RE: RESEARCH INTO BLOOD PRESSURE IN THE CARIBBEAN COMMUNITY

Please find attached, details of the programme for the first of a number of monthly workshops for registered participants of the above project. The date of this workshop is as follows:

DATE **Friday 8th July 1994**

TIME **a) Morning Session 9.30am to 12.30pm**

b) Afternoon Session 1.30pm to 4.30pm

VENUE **Room 719, Mary Seacole House, 91 Clapham High Road, SW4**

Particpants can choose to attend either the morning session or the afternoon session. Please complete and return the attached slip immediately.

I Look forward to seeing you.

A. HILTON (Ms)

I will/will not, be attending Workshop No. 1. I will attend the morning/afternoon session. (Please delete as appropriate)

Name/Establishment_____

_____ **Tel:**_____

Please return to: Sandra Mills, Equalities Service Development, Room 603, Mary Seacole House, 91 Clapham High St. SW4 7TF.

RESEARCH INTO BLOOD PRESSURE IN THE CARIBBEAN COMMUNITY

PARTICIPANTS WORKSHOP NO.1

DATE Friday July 8th

TIME A) Morning Session 9.30am to 12.30pm
B) Afternoon Session 1.30pm to 4.30pm

VENUE Room 719, Mary Seacole House, 91 Clapham High Road, SW4

Particpants can choose to attend either the morning or afternoon session.

THEME:

WHY CARIBBEANS NEED TO TAKE UP THE CHALLENGE

PROGRAMME

AM	PM	
9.30-9.45	1.30-1.45	Introduction, Ms. Pat Bell
9.45-10.00	1.45-2.00	Report from Ms. A. Hilton
10.00-10.30	2.00-2.30	The need for this research
10.30-11.00	2.30-3.00	Break/Discussion
11.00-11.30	3.00-3.30	Taking control of your Blood Pressure.
11.30-12.15	3.30-4.15	Open Discussion
12.15-12.30	4.15-4.30	Closing remarks

PARTICIPANT'S WORKSHOP NO.1

THEME

WHY CARIBBEANS NEED TO TAKE UP THE CHALLENGE

PROGRAMME

AM	PM	
9:30-9.45	1.30-1.45	Introduction, Mr. John Knowles
9.45-10.00	1.45-2.00	Researcher's Report, Ms. Hylton
10.00-10.30	2.00-2.30	The need for this research, Ms. Hylton
10.30-11.00	2.30-3.00	Questions/discussion over coffee
11.00-11.30	3.00-3.30	Taking control of your blood pressure. AM - Ms Anna Minihane (Stroke Assoc.) PM - Ms. Susan Stanford (Stroke Assoc.)
11.30-12.15	3.30-4.15	OPEN DISCUSSION
12.15-12.30	4.15-4.30	Closing remarks, John Knowles

Thank you for your participation

SOCIAL SERVICES
Director: David A. Pope

LAMBETH

London Borough of Lambeth
Mary Seacole House
91 Clapham High Street
LONDON SW4 7TF

071-720 0220
ext.

Your Ref:

Our Ref:

Date:

Dear participant

Details for the next workshop are as follows:

DATE **THURSDAY, AUGUST 25**

VENUE Room 719, Mary Seacole House, 91 Clapham High Rd.

TIME Morning Session 9.30am to 12.30pm
Afternoon Session 1.30pm to 4.30pm

Participants can choose to attend either the morning sesson or the afternoon session. Please complete and return the attached slip immediately.

RETURN TO: Sandra Mills, Equalities

Look forward to seeing you

A. Hylton (Ms)

I **WILL / WILL NOT** be attending workshop **NO. 2.** I will attend the **MORNING / AFTERNOON** session.

Name_____Tel_____

Please return to: **SANDRA MILLS, Directorate of Social Services, Equalities Services Development, Room 603, Mary Seacole House, 91 Clapham High Street, London SW4 7TF**

TEL. 071 926 4511

BLOOD PRESSURE WORKSHOP MEMO

The next Blood Pressure Workshops will be held on:

Thursday February 23.

The topic for the workshop is:

STRESS MANAGEMENT

The workshops will be facilitated by **Ms. Diana Hsu** whose techniques have been found to be very effective. It is an honour to have Ms. Hsu volunteer her services to this programme.

Ms. Hsu's session will cover **meditation, relaxation** and **visualization**. If possible, please wear casual garments that are easy to move in.

The workshop will run from **9:30 am to 12:30 pm**. The session will be repeated in the afternoon from **1:30 to 4:30**. Thus you may attend either one.

I look forward to seeing you at what promises to be an enlightening and enjoyable workshop.

Antoinette

J

** BLOOD PRESSURE WORKSHOP **

Dear Participant

The next and **FINAL** workshop will be held on:
THURSDAY, JUNE 8TH, 7th Floor, Mary Seacole. Room 719

The workshop will be **ONE** session only from 10 am to 3 pm, with a lunch break from **12 to 1pm.**

I hope that you will be able to attend this final workshop, which promises to be very intersting.

However, should you not be able to attend this workshop, you will have the opportunity to learn the results of this research and the implications thereof, at a **mini-conference** to be held early next year.

My contact number is: 0181 677 5975. It would be nice to hear from you, anytime.

Antoinette
RESEARCHER

K

WORKING TOGETHER FOR BETTER HEALTH

RESEARCH INTO
BLOOD PRESSURE
IN THE
AFRO-CARIBBEAN COMMUNITY

A CHALLENGE FOR THE AFRO-CARIBBEAN COMMUNITY TOWARDS CONTROLLING HYPERTENSION AND STROKE

The Directorate of Social Services, Equalities Service Development Division, has developed 'Healthy Alliance' with an Afro-Caribbean Researcher, researching into Hypertension in the Afro-Caribbean community.

The results of this study will not only benefit Afro-Caribbeans generally, but specifically those who participate in this pilot study.

Participants will gain:

* **valuable information about BLOOD PRESSURE**
* **knowledge of individual prevention strategies**
* **information on the management of blood pressure**

* **we would like 150 females and 150 males to participate***

IF YOU ARE OVER AGE 18 AND WORK WITHIN SOCIAL SERVICES, YOU ARE MOST WELCOMED TO PARTICIPATE.

INTERVIEWS ARE PRIVATE AND CONFIDENTIAL, AND MAY BE CONDUCTED AT PARTICIPANTS CONVENIENCE, INCLUDING DURING WORKING HOURS

This research is supported by the Directorate of Social Services as part of its 'HEALTHY ALLIANCE INITIATIVE'

FOR FURTHER INFORMATION, PLEASE CONTACT:

SANDRA MILLS OR PAULINE HUGGAN ON: 071-926 4511

PARTICIPANTS

A minimum of 100 **males and 100 females,** ages 18 and over will be drawn from the Social Services workforce.

Participants should be healthy and not be taking any form of prescribed medication for hypertension

FORM OF INTERVIEW

Applicants will be required to complete a questionnaire during an interview with the Researcher. The interview will take approximately one, (1) hour. The questionnaire will generally seek to derive information about the participant's lifestyle, heritage, general health, exercise habits, dietary habits, and their social and pyhsical environment.

During the interview, the participant's blood pressure and heart rate will be measured. Blood pressure will be measured, using the standard sphymomanometer, (the instrument used by the nurse or doctor). Heart rate will be measured via the radial pulse, (i.e. at the wrist). **THERE IS NO INVASIVE METHODS OF MEASUREMENTS.**

At the end of the interview, the participant will be asked if he or she wishes to participate in the full one year programme. The blood pressure and heart rate of those participants in the full programme will be monitored every eight (8) weeks for twelve (12) months, thus a total of six (6) readings.

WHERE INTERVIEWED

Participants will be able to choose whether they wish to be interviewed during work hours or outside of work hours, including weekends.

CONFIDENTIALITY

All interviews will be conducted by the Researcher personnally. Interviews will be private and completely confidential in accordance with the relevant ethics regulations. Questionnaires are so designed, that the names of the participants are not known. Completed questionnaires will not be made available to any Council employee, but will be made available to the University's examiners. The Council will however, get a copy of the statistical findings at the end of the project. The Researcher will keep a separate listing of participants' names, to match questionnaires. This is necessary for the monitoring purposes.

K

RESPONSE SLIP

RESEARCH INTO BLOOD PRESSURE IN AFRO-CARIBBEANS

I would like to participate in the above pilot study

yes [ ] no [ ] (Please tick)

I would like more information on the Blood Pressure in Afro-Caribbeans Research Project

yes [ ] no [ ] (Please tick)

NAME _____

ESTABLISHMENT/ADDRESS _____

_____

_____

_____

CONTACT NUMBER _____

- -

Please complete and return to:

Sandra Mills, Directorate of Social Services, Equalities Service Development, Room 603, Mary Seacole House, 91 Clapham High Street London SW4 7TF

L

CALLING ALL
CARIBBEAN STAFF

MORE PARTICIPANTS ARE NEEDED FOR THE

RESEARCH IN BLOOD PRESSURE IN CARIBBEANS

YOU CAN HELP YOURSELF AND THE RESEARCH BY VOLUNTEERING

TODAY

CONTACT:
MS. SANDRA MILLS 071-926-4511
MS. P.A. HILTON 071-926-4563

Appendix M Frequencies of Gender and Age

GENDER

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	female	138	84.1	84.1	84.1
	male	26	15.9	15.9	100.0
	Total	164	100.0	100.0	

AGE

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	20	1	.6	.6	.6
	22	1	.6	.6	1.2
	24	2	1.2	1.2	2.4
	25	3	1.8	1.8	4.3
	26	1	.6	.6	4.9
	27	3	1.8	1.8	6.7
	28	1	.6	.6	7.3
	29	10	6.1	6.1	13.4
	30	9	5.5	5.5	18.9
	31	7	4.3	4.3	23.2
	32	5	3.0	3.0	26.2
	33	11	6.7	6.7	32.9
	34	11	6.7	6.7	39.6
	35	9	5.5	5.5	45.1
	36	7	4.3	4.3	49.4
	37	4	2.4	2.4	51.8
	38	9	5.5	5.5	57.3
	39	8	4.9	4.9	62.2
	40	8	4.9	4.9	67.1
	41	6	3.7	3.7	70.7
	42	8	4.9	4.9	75.6
	43	1	.6	.6	76.2
	44	6	3.7	3.7	79.9
	45	4	2.4	2.4	82.3
	46	2	1.2	1.2	83.5
	47	1	.6	.6	84.1
	48	3	1.8	1.8	86.0
	49	1	.6	.6	86.6
	50	2	1.2	1.2	87.8
	51	3	1.8	1.8	89.6
	52	2	1.2	1.2	90.9
	53	1	.6	.6	91.5
	54	2	1.2	1.2	92.7
	55	2	1.2	1.2	93.9
	56	3	1.8	1.8	95.7
	57	2	1.2	1.2	97.0
	58	2	1.2	1.2	98.2
	59	1	.6	.6	98.8
	61	2	1.2	1.2	100.0
	Total	164	100.0	100.0	

N

QUESTIONNAIRE APPENDIX

ID #_____ NAME_____

DEPARTMENT_____

TEL. WK._____ HOME_____

COMMENTS_____

_____

_____

QUESTIONNAIRE APPENDIX

ID #_____ NAME_____

DEPARTMENT_____

TEL. WK._____ HOME_____

COMMENTS_____

_____

_____

_____

Dear _____

Thank you for volunteering to be a participant in this research. Without you, and the many other volunteers, this research project could not materialise.

As a participant you will be making an invaluable contribution towards the knowledge of blood pressure patterns in your fellow Afro-caribbeans, both within and outside of the UK .

Integral to this research project is that it will help to build awareness through education. Your participation in this project means that you are now a member of the team. This means that you will be offered telephone awareness support, on issues concerning blood pressure, for the duration of your monitoring.

If I the researcher do not have the ready answer, I will find it for you.

Not only is this the least that we can do as a form of thanks for your contibution, but by educating you the partcipant, we will be educating many more people, as you will pass on what you have learnt to your family, friends and associates.

THE STAGES OF OF YOUR PARTICIPATION ARE :

INTERVIEW STAGE This will take place now. You will complete the questionnaire,and a total of 6 BP and HR measurements will be taken, (3 sitting down and 3 lying down.

MONITORING STAGE

After today, your BP & HR will be monitored for 12 months, approximately every 8 weeks, a total of 6 monitoring sessions.

**NOTE

Over the 12 months that monitoring is being conducted, it is possible that ammedments may be made to the questionnaire, as based on information gathered from participants from the original questionnaire, it may be necessary to qualify or elaborate on certain topics, in order to facilitate more accurate analysis.

CONFIDENTIALITY

Please be assured that your participation in this study adheres to the highest standards of confidentialty, and that no one outside of the research team is privy to the completed questionnnaires, which does not identify you the participant by name.

Thank you again for your commitment, and I know that at the end of this study, you will be glad that you participated.

Sincerely,

P. Antointette Hylton

RESEARCHER

P

AFRO-CARIBBEAN PARTICIPANTS BP/HR REPORT

ID_____ READ NO._____ DATE_____

SITTING	SBP	1_____	2_____	3_____	LOW_____
	DBP	1_____	2_____	3_____	LOW_____
	RHR	1_____	2_____	3_____	LOW_____
LYING	SBP	1_____	2_____	3_____	LOW_____
	DBP	1_____	2_____	3_____	LOW_____
	RHR	1_____	2_____	3_____	LOW_____

WEIGHT - KG _____

NEXT READING DATE IS BEFORE _____

PROGRESS

OVERALL _____

WORK PRESSURE _____

STRESS LEVEL _____

HEALTH _____

ALCHOL/SMOKE _____

EXERCISE _____

DIET _____

COMMENTS _____

_____

_____

_____

_____

RESEARCHER: ANTOINETTE 64563 OR 64511

Q

PARTICIPANT'S WORKSHOP NO.1

THEME

WHY CARIBBEANS NEED TO TAKE UP THE CHALLENGE

PROGRAMME

AM	PM	
9:30-9.45	1.30-1.45	Introduction, Mr. John Knowles
9.45-10.00	1.45-2.00	Researcher's Report, Ms. Hylton
10.00-10.30	2.00-2.30	The need for this research, Ms. Hylton
10.30-11.00	2.30-3.00	Questions/discussion over coffee
11.00-11.30	3.00-3.30	Taking control of your blood pressure. AM - Ms Anna Minihane (Stroke Assoc.) * * PM - Ms. Susan Stanford (Stroke Assoc.)
11.30-12.15	3.30-4.15	OPEN DISCUSSION
12.15-12.30	4.15-4.30	Closing remarks, John Knowles

Thank you for your participation

PARTICIPANT'S WORKSHOP NO. 2

THEME

WHY AND HOW BLOOD PRESSURE IS MEASURED

PROGRAMME

AM	PM	
9:30-9.45	1.30-1.45	Introduction, Raj Gupta
9.45-10.00	1.45-2.00	Researcher's Report, Ms. Hylton
10.00-10.30	2.00-2.30	Video Viewing on Blood Pressure
10.30-11.00	2.30-3.00	Questions/discussion over coffee
11.00-11.30	3.00-3.30	Presentation by: AM - P.A. Hylton (Researcher) * PM - Annabella Franco(District Nurse)
11.30-12.15	3.30-4.15	Hands-on & Open Discussion
12.15-12.30	4.15-4.30	Closing remarks, Raj Gupta

Thank you for your participation

BLOOD PRESSURE WORKSHOP MEMO

The next Blood Pressure Workshops will be held on:

Thursday February 23.

The topic for the workshop is:

STRESS MANAGEMENT

The workshops will be facilitated by **Ms. Diana Hsu** whose techniques have been found to be very effective. It is an honour to have Ms. Hsu volunteer her services to this programme.

Ms. Hsu's session will cover **meditation, relaxation** and **visualization**. If possible, please wear casual garments that are easy to move in.

The workshop will run from **9:30 am to 12:30 pm**. The session will be repeated in the afternoon from **1:30 to 4:30**. Thus you may attend either one.

I look forward to seeing you at what promises to be an enlightening and enjoyable workshop.

Antoinette

SA

- - Correlation Coefficients - -

	SBP	DBP	AGE	BMI	RHR	GENDER
SBP	1.0000	.7865	.4291	.1510	.0914	-.0823
	(164)	(164)	(164)	(164)	(164)	(164)
	P= .	P= .000	P= .000	P= .054	P= .245	P= .295
DBP	.7865	1.0000	.3331	.1153	-.0358	-.0842
	(164)	(164)	(164)	(164)	(164)	(164)
	P= .000	P= .	P= .000	P= .142	P= .649	P= .284
AGE	.4291	.3331	1.0000	-.0472	.0245	.0054
	(164)	(164)	(164)	(164)	(164)	(164)
	P= .000	P= .000	P= .	P= .548	P= .756	P= .945
BMI	.1510	.1153	-.0472	1.0000	.1260	-.1057
	(164)	(164)	(164)	(164)	(164)	(164)
	P= .054	P= .142	P= .548	P= .	P= .108	P= .178
RHR	.0914	-.0358	.0245	.1260	1.0000	-.1851
	(164)	(164)	(164)	(164)	(164)	(164)
	P= .245	P= .649	P= .756	P= .108	P= .	P= .018
GENDER	-.0823	-.0842	.0054	-.1057	-.1851	1.0000
	(164)	(164)	(164)	(164)	(164)	(164)
	P= .295	P= .284	P= .945	P= .178	P= .018	P= .

- - - S P E A R M A N C O R R E L A T I O N C O E F F I C I E N T S - - -

DBP	.7746				
	N(164)				
	Sig .000				
AGE	.4351	.4136			
	N(164)	N(164)			
	Sig .000	Sig .000			
BMI	.2628	.2042	-.0165		
	N(164)	N(164)	N(164)		
	Sig .001	Sig .009	Sig .834		
RHR	.1500	-.0208	.0143	.0441	
	N(164)	N(164)	N(164)	N(164)	
	Sig .055	Sig .791	Sig .856	Sig .575	
GENDER	-.0533	-.0970	-.0129	-.0908	-.1499
	N(164)	N(164)	N(164)	N(164)	N(164)
	Sig .498	Sig .217	Sig .870	Sig .248	Sig .055
	SBP	DBP	AGE	BMI	RHR

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--- SPEARMAN CORRELATION COEFFICIENTS ---

	SBP	AGE	BMI	ACOM.CRWD	ACOM.OK	ADDSALT
AGE	.4351 N(164) Sig .000					
BMI	.2628 N(164) Sig .001	-.0165 N(164) Sig .834				
ACOM.CRWD	.1451 N(164) Sig .064	.2579 N(164) Sig .001	-.0420 N(164) Sig .593			
ACOM.OK	.3233 N(164) Sig .000	.3451 N(164) Sig .000	-.0776 N(164) Sig .324	.3245 N(164) Sig .000		
ADDSALT	-.2306 N(164) Sig .003	-.1950 N(164) Sig .012	-.1373 N(164) Sig .080	.0031 N(164) Sig .968	.0039 N(164) Sig .960	
BP.BROS	.1727 N(164) Sig .027	.0456 N(164) Sig .562	.1461 N(164) Sig .062	.0427 N(164) Sig .588	-.0013 N(164) Sig .987	-.1679 N(164) Sig .032
DRINK.AMT	.0497 N(164) Sig .528	.0643 N(164) Sig .413	-.0455 N(164) Sig .563	.0211 N(164) Sig .789	.0113 N(164) Sig .886	.1913 N(164) Sig .014
BORN	.3555 N(164) Sig .000	.6183 N(164) Sig .000	-.0221 N(164) Sig .779	.1644 N(164) Sig .035	.2470 N(164) Sig .001	-.1980 N(164) Sig .011
UK.LIVE	.3864 N(164) Sig .000	.7595 N(164) Sig .000	.0225 N(164) Sig .775	.2539 N(164) Sig .001	.2376 N(164) Sig .002	-.1424 N(164) Sig .069
QUALIF	-.0935 N(164) Sig .234	-.0119 N(164) Sig .879	-.0567 N(164) Sig .471	.1450 N(164) Sig .064	.0564 N(164) Sig .473	-.0133 N(164) Sig .866

SA

--- SPEARMAN CORRELATION COEFFICIENTS ---

	SBP	AGE	BMI	ACOM.CRWD	ACOM.OK	ADDSALT
JOBGRADE	-.1541	-.0079	-.0753	.2043	.1665	.0665
	N(164)	N(164)	N(164)	N(164)	N(164)	N(164)
	Sig .049	Sig .920	Sig .338	Sig .009	Sig .033	Sig .397
R.ANGRY	-.2184	-.0593	.0428	.0237	.0370	.0756
	N(164)	N(164)	N(164)	N(164)	N(164)	N(164)
	Sig .005	Sig .451	Sig .587	Sig .763	Sig .638	Sig .336
R.CLIENT	-.0240	-.0164	-.0578	-.0197	-.0151	-.0761
	N(164)	N(164)	N(164)	N(164)	N(164)	N(164)
	Sig .760	Sig .834	Sig .463	Sig .803	Sig .848	Sig .333
R.COLEGU	-.1873	-.0223	-.0941	.0285	-.0519	.0183
	N(164)	N(164)	N(164)	N(164)	N(164)	N(164)
	Sig .016	Sig .777	Sig .231	Sig .717	Sig .509	Sig .816
SMOKEAGE	-.2001	-.2217	-.1554	-.1038	-.2456	.0687
	N(164)	N(164)	N(164)	N(164)	N(164)	N(164)
	Sig .010	Sig .004	Sig .047	Sig .186	Sig .002	Sig .382
SMOKE.AMT	-.1711	-.2216	-.0871	-.1426	-.2239	.0824
	N(164)	N(164)	N(164)	N(164)	N(164)	N(164)
	Sig .028	Sig .004	Sig .268	Sig .069	Sig .004	Sig .294
SMOKER	-.1712	-.2144	-.1044	-.1346	-.1948	.0668
	N(164)	N(164)	N(164)	N(164)	N(164)	N(164)
	Sig .028	Sig .006	Sig .183	Sig .086	Sig .012	Sig .395
SMOKE.EVER	-.2433	-.1973	-.1305	-.0267	-.2281	.1509
	N(164)	N(164)	N(164)	N(164)	N(164)	N(164)
	Sig .002	Sig .011	Sig .096	Sig .735	Sig .003	Sig .054

SB

AVERAGE SYSTOLIC BLOOD PRESSURE - Grouped: Below and above 131 mmHg

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
bp <131 - low	0	130	79.3	79.3	79.3
bp >130 - high	1	34	20.7	20.7	100.0
	Total	164	100.0	100.0	

AVERAGE SYSTOLIC BLOOD PRESSURE - Grouped: normal(under 121 mmHg) or above normal

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
bp <121 normal	0	82	50.0	50.0	50.0
bp >120 above normal	1	82	50.0	50.0	100.0
	Total	164	100.0	100.0	

AVERAGE SYSTOLIC BLOOD PRESSURE - Grouped

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
<120	1	74	45.1	45.1	45.1
120 - 129	2	53	32.3	32.3	77.4
130 & over	3	37	22.6	22.6	100.0
	Total	164	100.0	100.0	

AVERAGE SYSTOLIC BLOOD PRESSURE - Grouped: CATEGORY A

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
below normal < 107 m	1	18	11.0	11.0	11.0
normal 107 - 137 mmh	2	122	74.4	74.4	85.4
above normal > 137 m	3	24	14.6	14.6	100.0
	Total	164	100.0	100.0	

AVERAGE SYSTOLIC BLOOD PRESSURE - Grouped: CATEGORY B

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
normal < 138 mmhg	1	140	85.4	85.4	85.4
high > 137 mmhg	2	24	14.6	14.6	100.0
	Total	164	100.0	100.0	

SYSTOLIC BLOOD PRESSURE - DISTRIBUTION

Mean	122.436	Std err	1.171	Median	120.083
Std dev	14.997	Variance	224.922	Kurtosis	1.723
S E Kurt	.377	Skewness	1.125	S E Skew	.190
Range	88.000	Minimum	93.333	Maximum	181.333

Valid cases	164	Missing cases	0

SYSTOLIC BLOOD PRESSURE - FREQUENCY DISTRIBUTION - DETAILED

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
	93	1	.6	.6	.6
	96	1	.6	.6	1.2
	101	1	.6	.6	1.8
	101	1	.6	.6	2.4
	101	1	.6	.6	3.0
	102	2	1.2	1.2	4.3
	103	2	1.2	1.2	5.5
	103	2	1.2	1.2	6.7
	103	1	.6	.6	7.3
	106	3	1.8	1.8	9.1
	106	1	.6	.6	9.8
	107	1	.6	.6	10.4
	107	1	.6	.6	11.0
	107	2	1.2	1.2	12.2
	108	1	.6	.6	12.8
	108	2	1.2	1.2	14.0
	108	1	.6	.6	14.6
	108	1	.6	.6	15.2
	110	1	.6	.6	15.9
	110	3	1.8	1.8	17.7
	111	3	1.8	1.8	19.5
	111	2	1.2	1.2	20.7
	111	3	1.8	1.8	22.6
	112	2	1.2	1.2	23.8
	112	3	1.8	1.8	25.6
	112	2	1.2	1.2	26.8
	112	1	.6	.6	27.4
	113	3	1.8	1.8	29.3
	113	1	.6	.6	29.9
	114	1	.6	.6	30.5
	114	1	.6	.6	31.1
	114	1	.6	.6	31.7
	114	2	1.2	1.2	32.9
	114	2	1.2	1.2	34.1
	114	1	.6	.6	34.8
	115	1	.6	.6	35.4

SYSTOLIC BLOOD PRESSURE - FREQUENCY DISTRIBUTION

116	1	.6	.6	36.0
117	1	.6	.6	36.6
117	1	.6	.6	37.2
117	2	1.2	1.2	38.4
117	1	.6	.6	39.0
117	1	.6	.6	39.6
118	1	.6	.6	40.2
118	4	2.4	2.4	42.7
118	1	.6	.6	43.3
119	1	.6	.6	43.9
119	1	.6	.6	44.5
119	1	.6	.6	45.1
120	1	.6	.6	45.7
120	4	2.4	2.4	48.2
120	1	.6	.6	48.8
120	2	1.2	1.2	50.0
120	1	.6	.6	50.6
121	2	1.2	1.2	51.8
121	1	.6	.6	52.4
121	2	1.2	1.2	53.7
121	1	.6	.6	54.3
122	2	1.2	1.2	55.5
122	3	1.8	1.8	57.3
122	1	.6	.6	57.9
123	2	1.2	1.2	59.1
123	2	1.2	1.2	60.4
123	2	1.2	1.2	61.6
124	1	.6	.6	62.2
124	1	.6	.6	62.8
124	1	.6	.6	63.4
124	2	1.2	1.2	64.6
125	1	.6	.6	65.2
125	2	1.2	1.2	66.5
125	2	1.2	1.2	67.7
125	1	.6	.6	68.3
125	2	1.2	1.2	69.5
126	1	.6	.6	70.1
126	2	1.2	1.2	71.3
126	1	.6	.6	72.0
128	2	1.2	1.2	73.2
128	1	.6	.6	73.8
128	1	.6	.6	74.4
128	1	.6	.6	75.0
128	1	.6	.6	75.6
129	1	.6	.6	76.2
129	2	1.2	1.2	77.4
130	3	1.8	1.8	79.3
132	1	.6	.6	79.9
133	1	.6	.6	80.5
134	2	1.2	1.2	81.7
134	1	.6	.6	82.3
134	1	.6	.6	82.9
136	1	.6	.6	83.5
137	2	1.2	1.2	84.8
138	1	.6	.6	85.4
138	1	.6	.6	86.0
138	1	.6	.6	86.6
139	1	.6	.6	87.2
140	1	.6	.6	87.8
140	1	.6	.6	88.4
140	1	.6	.6	89.0
141	1	.6	.6	89.6

SB

SYSTOLIC BLOOD PRESSURE - FREQUENCY DISTRIBUTION

141	1	.6	.6	90.2
144	1	.6	.6	90.9
145	1	.6	.6	91.5
147	1	.6	.6	92.1
149	1	.6	.6	92.7
150	1	.6	.6	93.3
151	1	.6	.6	93.9
152	1	.6	.6	94.5
153	1	.6	.6	95.1
154	1	.6	.6	95.7
155	1	.6	.6	96.3
155	1	.6	.6	97.0
158	1	.6	.6	97.6
160	1	.6	.6	98.2
170	2	1.2	1.2	99.4
181	1	.6	.6	100.0
Total	164	100.0	100.0	

SB

SYSTOLIC BLOOD PRESSURE - EXPLORATORY STATISTICS

Valid cases:	164.0	Missing cases:	.0	Percent missing:	.0		
Mean	122.4360	Std Err	1.1711	Min	93.3333	Skewness	1.1254
Median	120.0833	Variance	224.9224	Max	181.3333	S E Skew	.1896
5% Trim	121.4530	Std Dev	14.9974	Range	88.0000	Kurtosis	1.7231
95% CI for Mean	(120.1235, 124.7485)			IQR	16.6250	S E Kurt	.3769

Frequency		Stem &	Leaf
1.00		9 *	3
1.00		9 .	6
10.00		10 *	0112222223
14.00		10 .	55566677777889
32.00		11 *	00000000111111111122223333444444
22.00		11 .	6667777788888999999999
29.00		12 *	00000011111222222333333444444
21.00		12 .	555555556777888899999
6.00		13 *	124444
10.00		13 .	6777889999
3.00		14 *	004
4.00		14 .	5789
3.00		15 *	122
8.00	Extremes		(154), (155), (155), (158), (160), (170), (181)

Stem width: 10
Each leaf: 1 case(s)

Percentiles

	5.0000	10.0000	25.0000	50.0000	75.0000	90.0000
Percentiles						
Haverage	102.5000	106.5000	111.6667	120.0833	128.2917	142.3333
Tukey's Hinges			111.6667	120.0833	128.2500	

	95.0000
Percentiles	153.2917

Extreme Values

5	Highest	Case #	5	Lowest	Case #
	181	Case: 154		93	Case: 138
	170	Case: 148		96	Case: 116
	170	Case: 118		101	Case: 91
	160	Case: 85		101	Case: 122
	158	Case: 158		101	Case: 21

SC.

DIASTOLIC BLOOD PRESSURE - EXPLORATORY STATISTICS

Valid cases:	164.0	Missing cases:	.0	Percent missing:	.0		
Mean	76.9035	Std Err	.9185	Min	44.8333	Skewness	.5321
Median	75.8333	Variance	138.3493	Max	116.5000	S E Skew	.1896
5% Trim	76.5831	Std Dev	11.7622	Range	71.6667	Kurtosis	.9958
95% CI for Mean	(75.0898, 78.7171)			IQR	13.7500	S E Kurt	.3769

Frequency		Stem &	Leaf
1.00	Extremes		(45)
2.00		5 *	03
6.00		5 .	667899
13.00		6 *	0122233334444
24.00		6 .	555566666677788889999999
26.00		7 *	00011111111222233333444444
33.00		7 .	555555555556666777777777788889999
24.00		8 *	000000111112222223333344
15.00		8 .	555666677789999
5.00		9 *	00112
10.00		9 .	5566777899
2.00		10 *	11
3.00	Extremes		(110), (116)

Stem width: 10
Each leaf: 1 case(s)

Percentiles

Percentiles	5.0000	10.0000	25.0000	50.0000	75.0000	90.0000
Haverage	59.0833	63.4167	69.3750	75.8333	83.1250	91.8333
Tukey's Hinges			69.4167	75.8333	83.0833	
Percentiles	95.0000					
	97.8333					

Extreme Values

	Highest	Case #		Lowest	Case #
5	117	Case: 118	5	45	Case: 79
	116	Case: 73		50	Case: 86
	110	Case: 81		54	Case: 103
	101	Case: 148		56	Case: 67
	101	Case: 128		56	Case: 121

SD

AGE: FREQUENCY DISTRIBUTION - DETAILED

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
	20	1	.6	.6	.6
	22	1	.6	.6	1.2
	24	2	1.2	1.2	2.4
	25	3	1.8	1.8	4.3
	26	1	.6	.6	4.9
	27	3	1.8	1.8	6.7
	28	1	.6	.6	7.3
	29	10	6.1	6.1	13.4
	30	9	5.5	5.5	18.9
	31	7	4.3	4.3	23.2
	32	5	3.0	3.0	26.2
	33	11	6.7	6.7	32.9
	34	11	6.7	6.7	39.6
	35	9	5.5	5.5	45.1
	36	7	4.3	4.3	49.4
	37	4	2.4	2.4	51.8
	38	9	5.5	5.5	57.3
	39	8	4.9	4.9	62.2
	40	8	4.9	4.9	67.1
	41	6	3.7	3.7	70.7
	42	8	4.9	4.9	75.6
	43	1	.6	.6	76.2
	44	6	3.7	3.7	79.9
	45	4	2.4	2.4	82.3
	46	2	1.2	1.2	83.5
	47	1	.6	.6	84.1
	48	3	1.8	1.8	86.0
	49	1	.6	.6	86.6
	50	2	1.2	1.2	87.8
	51	3	1.8	1.8	89.6
	52	2	1.2	1.2	90.9
	53	1	.6	.6	91.5
	54	2	1.2	1.2	92.7
	55	2	1.2	1.2	93.9
	56	3	1.8	1.8	95.7
	57	2	1.2	1.2	97.0
	58	2	1.2	1.2	98.2
	59	1	.6	.6	98.8
	61	2	1.2	1.2	100.0
	Total	164	100.0	100.0	

AGE - DESCRIPTIVE STATISTICS

Mean	38.238	Std err	.679	Median	37.000
Std dev	8.699	Variance	75.667	Kurtosis	.005
S E Kurt	.377	Skewness	.680	S E Skew	.190
Range	41.000	Minimum	20.000	Maximum	61.000

Valid cases 164 Missing cases 0

AGE: GROUPED BY DECADE

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
under 30	1	22	13.4	13.4	13.4
30-39	2	80	48.8	48.8	62.2
40-49	3	40	24.4	24.4	86.6
50 plus	4	22	13.4	13.4	100.0
	Total	164	100.0	100.0	

AGE: GROUPED BY OPCS CATEGORISATIONS

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
<25	1	4	2.4	2.4	2.4
25-34	2	61	37.2	37.2	39.6
35-44	3	66	40.2	40.2	79.9
>44	4	33	20.1	20.1	100.0
	Total	164	100.0	100.0	

AGE AGE

Valid cases: 164.0 Missing cases: .0 Percent missing: .0

Mean	38.2378	Std Err	.6793	Min	20.0000	Skewness	.6800
Median	37.0000	Variance	75.6670	Max	61.0000	S E Skew	.1896
5% Trim	37.9160	Std Dev	8.6987	Range	41.0000	Kurtosis	.0053
95% CI for Mean (36.8965, 39.5791)				IQR	10.0000	S E Kurt	.3769

Frequency	Stem &	Leaf
1.00	2 *	0
1.00	2 t	2
5.00	2 f	44555
4.00	2 s	6777
11.00	2 .	89999999999
16.00	3 *	0000000001111111
16.00	3 t	2222233333333333
20.00	3 f	44444444444555555555
11.00	3 s	66666667777
17.00	3 .	88888888899999999
14.00	4 *	00000000111111
9.00	4 t	222222223
10.00	4 f	4444445555
3.00	4 s	667
4.00	4 .	8889
5.00	5 *	00111
3.00	5 t	223
4.00	5 f	4455
3.00	5 s	666
7.00	Extremes	(57), (58), (59), (61)

Stem width: 10
Each leaf: 1 case(s)

- - Description of Subpopulations - -

Summaries of SBP
By levels of age grouped by OPCS CATEGORIES

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
AGE.GRP	1	<25	127.5000	8.9267	4
AGE.GRP	2	25-34	115.5601	11.0509	61
AGE.GRP	3	35-44	122.4066	13.8710	66
AGE.GRP	4	>44	134.5909	16.5149	33

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable A.SBP SBP
By levels of AGE.GRP age grouped by opcs standard

Value	Label	Mean	Std Dev	Sum of Sq	Cases
1	<25	127.5000	8.9267	239.0556	4
2	25-34	115.5601	11.0509	7327.2796	61
3	35-44	122.4066	13.8710	12506.2294	66
4	>44	134.5909	16.5149	8727.7273	33
Within Groups Total		122.4360	13.4165	28800.2918	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	7862.0637	3	2620.6879	14.5592	.0000
Linearity	5959.4410	1	5959.4410	33.1077	.0000
Dev. from Linearity	1902.6227	2	951.3114	5.2850	.0060
	R = .4032	R Squared = .1625			
Within Groups	28800.2918	160	180.0018		
	Eta = .4631	Eta Squared = .2144			

5.1

- - Description of Subpopulations - -

Summaries of SBP
By levels of agegroup by decade

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
AGE.DEC	1	under 30	117.5606	9.6101	22
AGE.DEC	2	30-39	117.7729	12.6027	80
AGE.DEC	3	40-49	127.2958	14.4432	40
AGE.DEC	4	50 plus	135.4318	18.4096	22

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of agegroup by decade

Value	Label	Mean	Std Dev	Sum of Sq	Cases
1	under 30	117.5606	9.6101	1939.4192	22
2	30-39	117.7729	12.6027	12547.3469	80
3	40-49	127.2958	14.4432	8135.5826	40
4	50 plus	135.4318	18.4096	7117.2033	22
Within Groups Total		122.4360	13.6335	29739.5520	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	6922.8035	3	2307.6012	12.4150	.0000
Linearity	6028.3115	1	6028.3115	32.4326	.0000
Dev. from Linearity	894.4920	2	447.2460	2.4062	.0934
	R = .4055	R Squared = .1644			
Within Groups	29739.5520	160	185.8722		
	Eta = .4345	Eta Squared = .1888			

CROSSTABULATION OF SYSTOLIC BLOOD PRESSURE BY AGE GROUPED BY DECADE

SBP	Count / Exp Val / Row Pct / Col Pct / Tot Pct / Residual	AGE BY DECADE under 30 (1)	30-39 (2)	40-49 (3)	50 plus (4)	Row Total
<120	1	13	46	12	3	74
		9.9	36.1	18.0	9.9	45.1%
		17.6%	62.2%	16.2%	4.1%	
		59.1%	57.5%	30.0%	13.6%	
		7.9%	28.0%	7.3%	1.8%	
		3.1	9.9	-6.0	-6.9	
120 - 129	2	7	25	13	8	53
		7.1	25.9	12.9	7.1	32.3%
		13.2%	47.2%	24.5%	15.1%	
		31.8%	31.3%	32.5%	36.4%	
		4.3%	15.2%	7.9%	4.9%	
		-.1	-.9	.1	.9	
130 & over	3	2	9	15	11	37
		5.0	18.0	9.0	5.0	22.6%
		5.4%	24.3%	40.5%	29.7%	
		9.1%	11.3%	37.5%	50.0%	
		1.2%	5.5%	9.1%	6.7%	
		-3.0	-9.0	6.0	6.0	
	Column Total	22 13.4%	80 48.8%	40 24.4%	22 13.4%	164 100.0%

Chi-Square	Value	DF	Significance
Pearson	28.27481	6	.00008
Likelihood Ratio	29.06657	6	.00006
Mantel-Haenszel test for linear association	24.27633	1	.00000

Minimum Expected Frequency - 4.963
Cells with Expected Frequency < 5 - 2 OF 12 (16.7%)

Statistic	Value	ASE1	Val/ASE0	Approximate Significance
Kendall's Tau-b	.33410	.06148	5.30607	
Kendall's Tau-c	.32760	.06174	5.30607	
Gamma	.48747	.08420	5.30607	
Somers' D :				
symmetric	.33403	.06147	5.30607	
with A.AVSPG1 dependent	.32764	.06041	5.30607	
with AGE.DEC dependent	.34068	.06321	5.30607	
Pearson's R	.38592	.06790	5.32444	.00000 *4
Spearman Correlation	.38010	.06909	5.23050	.00000 *4

SE

- - Description of Subpopulations - -

Summaries of SBP
By levels of GENDER

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
GENDER	1	female	122.9698	15.5984	138
GENDER	2	male	119.6026	11.1009	26

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of GENDER

Value	Label	Mean	Std Dev	Sum of Sq	Cases
1	female	122.9698	15.5984	33333.5686	138
2	male	119.6026	11.1009	3080.7265	26
Within Groups Total		122.4360	14.9927	36414.2951	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	248.0604	1	248.0604	1.1036	.2950
With fewer than three groups, the relationship is linear					
Within Groups	36414.2951	162	224.7796		

Eta = .0823 Eta Squared = .0068

SE

CROSSTABULATION OF SYSTOLIC BLOOD PRESSURE BY GENDER

SBP	Count / Exp Val / Row Pct / Col Pct / Tot Pct / Residual	GENDER female 1	GENDER male 2	Row Total
<120	1	63 62.3 85.1% 45.7% 38.4% .7	11 11.7 14.9% 42.3% 6.7% -.7	74 45.1%
120 - 129	2	42 44.6 79.2% 30.4% 25.6% -2.6	11 8.4 20.8% 42.3% 6.7% 2.6	53 32.3%
130 & over	3	33 31.1 89.2% 23.9% 20.1% 1.9	4 5.9 10.8% 15.4% 2.4% -1.9	37 22.6%
	Column Total	138 84.1%	26 15.9%	164 100.0%

Chi-Square	Value	DF	Significance
Pearson	1.71387	2	.42446
Likelihood Ratio	1.71912	2	.42335
Mantel-Haenszel test for linear association	.09336	1	.75995

Minimum Expected Frequency - 5.866

Statistic	Value	ASE1	Val/ASE0	Approximate Significance
Kendall's Tau-b	-.01349	.06912	-.19499	
Kendall's Tau-c	-.01115	.05720	-.19499	
Gamma	-.03259	.16767	-.19499	
Somers' D :				
symmetric	-.01229	.06297	-.19499	
with A.AVSPG1 dependent	-.02090	.10716	-.19499	
with GENDER dependent	-.00870	.04459	-.19499	
Pearson's R	-.02393	.07161	-.30469	.76099 *4
Spearman Correlation	-.01424	.07297	-.18121	.85643 *4

SF

SUBJECTS BIRTHPLACE

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
UK-born	1	66	40.2	40.2	40.2
Eastern Caribbean	2	28	17.1	17.1	57.3
Jamaican	3	70	42.7	42.7	100.0
	Total	164	100.0	100.0	

SUBJECT'S DURATION LIVING IN THE UK

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
UK -born	1	66	40.2	40.2	40.2
under 21 yrs	2	15	9.1	9.1	49.4
21 to 30 yrs	3	44	26.8	26.8	76.2
over 30 years	4	39	23.8	23.8	100.0
	Total	164	100.0	100.0	

SUBJECT'S MOTHER'S BIRTHPLACE

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
jamaica	1	112	68.3	68.3	68.3
other island	2	47	28.7	28.7	97.0
UK born	3	5	3.0	3.0	100.0
	Total	164	100.0	100.0	

SUBJECT'S FATHER'S BIRTHPLACE

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
jamaica	1	116	70.7	70.7	70.7
all other islands	2	47	28.7	28.7	99.4
UK	3	1	.6	.6	100.0
	Total	164	100.0	100.0	

5F

- - Description of Subpopulations - -

Summaries of SBP
By levels of Birthplace

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
BIRTHPLACE	1	UK-born	116.1439	11.2347	66
BIRTHPLACE	2	Eastern Caribbean	124.1310	16.1592	28
BIRTHPLACE	3	Jamaican	127.6905	15.5954	70

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of Birthplace

Value	Label	Mean	Std Dev	Sum of Sq	Cases
1	UK-born	116.1439	11.2347	8204.2159	66
2	Eastern Caribbean	124.1310	16.1592	7050.2421	28
3	Jamaican	127.6905	15.5954	16781.8492	70
Within Groups Total		122.4360	14.1061	32036.3072	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	4626.0483	2	2313.0242	11.6242	.0000
Linearity	4512.2741	1	4512.2741	22.6767	.0000
Dev. from Linearity	113.7742	1	113.7742	.5718	.4507
	R = .3508	R Squared = .1231			
Within Groups	32036.3072	161	198.9833		
	Eta = .3552	Eta Squared = .1262			

SF

- - Description of Subpopulations - -

Summaries of SBP
By levels of how long lived in UK

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
DURATION.UK	1	UK -born	116.1439	11.2347	66
DURATION.UK	2	under 21 yrs	119.7333	10.0855	15
DURATION.UK	3	21 to 30 yrs	126.2348	15.7409	44
DURATION.UK	4	over 30 years	129.8376	16.9253	39

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of how long lived in UK

Value	Label	Mean	Std Dev	Sum of Sq	Cases
1	UK -born	116.1439	11.2347	8204.2159	66
2	under 21 yrs	119.7333	10.0855	1424.0444	15
3	21 to 30 yrs	126.2348	15.7409	10654.4066	44
4	over 30 years	129.8376	16.9253	10885.6382	39
Within Groups Total		122.4360	13.9571	31168.3051	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	5494.0504	3	1831.3501	9.4011	.0000
Linearity	5448.1343	1	5448.1343	27.9676	.0000
Dev. from Linearity	45.9161	2	22.9580	.1179	.8889
	R = .3855	R Squared = .1486			
Within Groups	31168.3051	160	194.8019		
	Eta = .3871	Eta Squared = .1499			

GENERAL HEALTH

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
fair	1	57	34.8	34.8	34.8
good	2	63	38.4	38.4	73.2
v.good	3	44	26.8	26.8	100.0
	Total	164	100.0	100.0	

HAD MEDICAL CHECK IN PAST 2 YEARS

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO	0	105	64.0	64.0	64.0
YES	1	59	36.0	36.0	100.0
	Total	164	100.0	100.0	

TAKING ANY PRESCRIBED MEDICATION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO	0	107	65.2	65.2	65.2
YES	1	57	34.8	34.8	100.0
	Total	164	100.0	100.0	

TAKING ANY UNPRESCRIBED MEDICATION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO	0	131	79.9	79.9	79.9
YES	1	33	20.1	20.1	100.0
	Total	164	100.0	100.0	

SUFFERING FROM STRESS

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO	0	68	41.5	41.5	41.5
YES	1	96	58.5	58.5	100.0
	Total	164	100.0	100.0	

- - Description of Subpopulations - -

Summaries of SBP
By levels of SUFFERING FROM STRESS

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
STRESS	0	NO	123.8333	16.9926	68
STRESS	1	YES	121.4462	13.4122	96

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of SUFFERING FROM STRESS

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	NO	123.8333	16.9926	19346.1667	68
1	YES	121.4462	13.4122	17089.3608	96
Within Groups Total		122.4360	14.9970	36435.5275	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	226.8280	1	226.8280	1.0085	.3168
With fewer than three groups, the relationship is linear					
Within Groups	36435.5275	162	224.9107		

Eta = .0787 Eta Squared = .0062

- - Description of Subpopulations - -

Summaries of SBP
By levels of medical check past 2 yrs

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
MED.CHK	0	NO	121.8841	15.4484	105
MED.CHK	1	YES	123.4181	14.2354	59

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of medical check past 2 yrs

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	NO	121.8841	15.4484	24820.0069	105
1	YES	123.4181	14.2354	11753.4652	59
Within Groups Total		122.4360	15.0254	36573.4720	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	88.8835	1	88.8835	.3937	.5312
With fewer than three groups, the relationship is linear					
Within Groups	36573.4720	162	225.7622		

Eta = .0492 Eta Squared = .0024

SH

AVERAGE HEART RATE - DESCRIPTIVE STATISICS

Mean	72.811	Std err	.672	Median	71.917
Std dev	8.600	Variance	73.958	Kurtosis	.004
S E Kurt	.377	Skewness	.195	S E Skew	.190
Range	45.333	Minimum	51.667	Maximum	97.000

AVERAGE HEART RATE - grouped

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
under 71 bpm	1	71	43.3	43.3	43.3
71 - 80 bpm	2	62	37.8	37.8	81.1
over 80 bpm	3	31	18.9	18.9	100.0
	Total	164	100.0	100.0	

SH

HEART RATE - EXPLORATORY STATISTICS

Valid cases: 164.0 Missing cases: .0 Percent missing: .0

Mean	72.8110	Std Err	.6715	Min	51.6667	Skewness	.1945
Median	71.9167	Variance	73.9579	Max	97.0000	S E Skew	.1896
5% Trim	72.7261	Std Dev	8.5999	Range	45.3333	Kurtosis	.0040
95% CI for Mean (71.4849, 74.1370)				IQR	11.1667	S E Kurt	.3769

Frequency		Stem &	Leaf
3.00		5 *	&
6.00		5 .	59&
19.00		6 *	0112234
28.00		6 .	5666777888999
49.00		7 *	0000000111112222333444
23.00		7 .	5566778899
21.00		8 *	00122344
9.00		8 .	5557&
5.00		9 *	13&
1.00	Extremes		(97)

Stem width: 10
Each leaf: 2 case(s)

& denotes fractional leaves.

Percentiles

Percentiles	5.0000	10.0000	25.0000	50.0000	75.0000	90.0000
Haverage	59.0833	61.7500	67.6667	71.9167	78.8333	84.6667
Tukey's Hinges			67.6667	71.9167	78.6667	
Percentiles	95.0000					
	87.0833					

Extreme Values

	Highest	Case #		Lowest	Case #
5	97	Case: 111	5	52	Case: 152
	93	Case: 97		54	Case: 161
	93	Case: 34		55	Case: 141
	93	Case: 78		55	Case: 13
	92	Case: 40		56	Case: 28

SH

- - Description of Subpopulations - -

Summaries of SBP
By levels of average heart rate - grouped

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
RHR	1	under 71 bpm	120.2770	15.2972	71
RHR	2	71 - 80 bpm	124.2043	15.5896	62
RHR	3	over 80 bpm	123.8441	12.7419	31

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of average heart rate - grouped

Value	Label	Mean	Std Dev	Sum of Sq	Cases
1	under 71 bpm	120.2770	15.2972	16380.2191	71
2	71 - 80 bpm	124.2043	15.5896	14825.1900	62
3	over 80 bpm	123.8441	12.7419	4870.6631	31
Within Groups Total		122.4360	14.9691	36076.0721	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	586.2834	2	293.1417	1.3082	.2732
Linearity	420.4612	1	420.4612	1.8764	.1726
Dev. from Linearity	165.8222	1	165.8222	.7400	.3909
	R = .1071	R Squared = .0115			
Within Groups	36076.0721	161	224.0750		
	Eta = .1265	Eta Squared = .0160			

BODY MASS INDEX - FREQUENCY DISTRIBUTION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
	16	1	.6	.6	.6
	19	1	.6	.6	1.2
	20	1	.6	.6	1.8
	20	1	.6	.6	2.4
	20	1	.6	.6	3.0
	20	1	.6	.6	3.7
	21	1	.6	.6	4.3
	21	1	.6	.6	4.9
	21	1	.6	.6	5.5
	21	1	.6	.6	6.1
	21	1	.6	.6	6.7
	21	1	.6	.6	7.3
	21	1	.6	.6	7.9
	21	1	.6	.6	8.5
	22	1	.6	.6	9.1
	22	2	1.2	1.2	10.4
	22	1	.6	.6	11.0
	22	1	.6	.6	11.6
	22	1	.6	.6	12.2
	22	1	.6	.6	12.8
	22	1	.6	.6	13.4
	22	1	.6	.6	14.0
	22	1	.6	.6	14.6
	22	1	.6	.6	15.2
	22	1	.6	.6	15.9
	22	1	.6	.6	16.5
	22	1	.6	.6	17.1
	23	1	.6	.6	17.7
	23	1	.6	.6	18.3
	23	1	.6	.6	18.9
	23	1	.6	.6	19.5
	23	1	.6	.6	20.1
	23	1	.6	.6	20.7
	23	1	.6	.6	21.3
	23	1	.6	.6	22.0
	23	1	.6	.6	22.6
	23	1	.6	.6	23.2
	24	1	.6	.6	23.8
	24	1	.6	.6	24.4
	24	1	.6	.6	25.0
	24	1	.6	.6	25.6
	24	1	.6	.6	26.2
	24	1	.6	.6	26.8
	24	1	.6	.6	27.4
	24	1	.6	.6	28.0
	24	1	.6	.6	28.7
	24	1	.6	.6	29.3

BODY MASS INDEX - FREQUENCY DISTRIBUTION

25	2	1.2	1.2	30.5
25	1	.6	.6	31.1
25	1	.6	.6	31.7
25	2	1.2	1.2	32.9
25	1	.6	.6	33.5
25	1	.6	.6	34.1
25	1	.6	.6	34.8
25	2	1.2	1.2	36.0
25	2	1.2	1.2	37.2
25	1	.6	.6	37.8
25	1	.6	.6	38.4
25	1	.6	.6	39.0
26	2	1.2	1.2	40.2
26	2	1.2	1.2	41.5
26	1	.6	.6	42.1
26	2	1.2	1.2	43.3
26	1	.6	.6	43.9
26	1	.6	.6	44.5
26	1	.6	.6	45.1
26	1	.6	.6	45.7
26	1	.6	.6	46.3
26	1	.6	.6	47.0
27	1	.6	.6	47.6
27	1	.6	.6	48.2
27	1	.6	.6	48.8
27	1	.6	.6	49.4
27	1	.6	.6	50.0
27	1	.6	.6	50.6
27	1	.6	.6	51.2
27	1	.6	.6	51.8
27	1	.6	.6	52.4
27	1	.6	.6	53.0
27	1	.6	.6	53.7
27	1	.6	.6	54.3
27	1	.6	.6	54.9
27	1	.6	.6	55.5
27	1	.6	.6	56.1
28	1	.6	.6	56.7
28	1	.6	.6	57.3
28	1	.6	.6	57.9
28	1	.6	.6	58.5
28	1	.6	.6	59.1
28	1	.6	.6	59.8
28	1	.6	.6	60.4
28	1	.6	.6	61.0
28	1	.6	.6	61.6
28	1	.6	.6	62.2
28	1	.6	.6	62.8
29	1	.6	.6	63.4
29	2	1.2	1.2	64.6
29	1	.6	.6	65.2

BODY MASS INDEX - FREQUENCY DISTRIBUTION

29	1	.6	.6	65.9
29	1	.6	.6	66.5
29	1	.6	.6	67.1
29	1	.6	.6	67.7
29	1	.6	.6	68.3
29	1	.6	.6	68.9
29	1	.6	.6	69.5
29	1	.6	.6	70.1
29	1	.6	.6	70.7
30	1	.6	.6	71.3
30	1	.6	.6	72.0
30	1	.6	.6	72.6
30	1	.6	.6	73.2
30	1	.6	.6	73.8
30	1	.6	.6	74.4
30	1	.6	.6	75.0
30	1	.6	.6	75.6
30	1	.6	.6	76.2
30	1	.6	.6	76.8
30	1	.6	.6	77.4
30	1	.6	.6	78.0
30	1	.6	.6	78.7
31	1	.6	.6	79.3
31	1	.6	.6	79.9
31	1	.6	.6	80.5
31	1	.6	.6	81.1
31	1	.6	.6	81.7
31	1	.6	.6	82.3
32	1	.6	.6	82.9
32	1	.6	.6	83.5
32	1	.6	.6	84.1
32	1	.6	.6	84.8
32	1	.6	.6	85.4
32	1	.6	.6	86.0
33	1	.6	.6	86.6
33	1	.6	.6	87.2
33	1	.6	.6	87.8
33	2	1.2	1.2	89.0
34	1	.6	.6	89.6
34	1	.6	.6	90.2
34	1	.6	.6	90.9
36	1	.6	.6	91.5
36	1	.6	.6	92.1
36	1	.6	.6	92.7
36	1	.6	.6	93.3
37	1	.6	.6	93.9
37	1	.6	.6	94.5
39	1	.6	.6	95.1
40	1	.6	.6	95.7
42	1	.6	.6	96.3
43	1	.6	.6	97.0
43	1	.6	.6	97.6
44	1	.6	.6	98.2
44	1	.6	.6	98.8
45	1	.6	.6	99.4
48	1	.6	.6	100.0
Total	164	100.0	100.0	

BODY MASS INDEX - DESCRIPTIVE STATISTICS

Mean	27.566	Std err	.430	Median	26.702
Std dev	5.501	Variance	30.259	Kurtosis	1.893
S E Kurt	.377	Skewness	1.188	S E Skew	.190
Range	31.534	Minimum	16.125	Maximum	47.659
Valid cases	164	Missing cases	0		

BODY MASS INDEX - grouped

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
<23	1	32	19.5	19.5	19.5
23-26	2	54	32.9	32.9	52.4
27-30	3	47	28.7	28.7	81.1
>30	4	31	18.9	18.9	100.0
	Total	164	100.0	100.0	

BODY MASS INDEX - EXPLORATORY STATISTICS

Valid cases:	164.0	Missing cases:	.0	Percent missing:	.0		
Mean	27.5660	Std Err	.4295	Min	16.1247	Skewness	1.1878
Median	26.7024	Variance	30.2592	Max	47.6587	S E Skew	.1896
5% Trim	27.1339	Std Dev	5.5008	Range	31.5340	Kurtosis	1.8929
95% CI for Mean	(26.7178, 28.4141)			IQR	6.5071	S E Kurt	.3769

Frequency		Stem &	Leaf
1.00		1 s	6
4.00		1 .	9999
15.00		2 *	000001111111111
24.00		2 t	222222222222333333333333
29.00		2 f	44444444444445555555555555555
26.00		2 s	66666666666667777777777777
22.00		2 .	8888888888889999999999
19.00		3 *	0000000000001111111
7.00		3 t	2222333
5.00		3 f	44555
3.00		3 s	667
2.00		3 .	89
7.00	Extremes		(42), (43), (43), (44), (48)

Stem width: 10
Each leaf: 1 case(s)

Percentiles

Percentiles	5.0000	10.0000	25.0000	50.0000	75.0000	90.0000
Haverage	20.7755	21.6713	23.6333	26.7024	30.1404	34.1361
Tukey's Hinges			23.6666	26.7024	30.1323	

Percentiles	95.0000
	39.5621

Extreme Values

5	Highest	Case #	5	Lowest	Case #
	48	Case: 78		16	Case: 50
	45	Case: 75		19	Case: 157
	44	Case: 79		20	Case: 16
	44	Case: 113		20	Case: 17
	43	Case: 112		20	Case: 6

- - Description of Subpopulations - -

Summaries of SBP
By levels of BMI- grouped

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
BMI	1	<23	116.8385	11.9728	32
BMI	2	23-26	120.6759	15.0513	54
BMI	3	27-30	126.3050	15.9902	47
BMI	4	>30	125.4140	14.4938	31

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of BMI- grouped

Value	Label	Mean	Std Dev	Sum of Sq	Cases
1	<23	116.8385	11.9728	4443.7491	32
2	23-26	120.6759	15.0513	12006.6343	54
3	27-30	126.3050	15.9902	11761.5177	47
4	>30	125.4140	14.4938	6302.1039	31
Within Groups Total		122.4360	14.6872	34514.0051	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	2148.3505	3	716.1168	3.3198	.0214
Linearity	1784.1171	1	1784.1171	8.2708	.0046
Dev. from Linearity	364.2333	2	182.1167	.8443	.4318
	R = .2206	R Squared = .0487			
Within Groups	34514.0051	160	215.7125		
	Eta = .2421	Eta Squared = .0586			

CROSSTABULATION OF SYSTOLIC BLOOD PRESSURE BY BODY MASS INDEX

SBP	Count / Exp Val / Row Pct / Col Pct / Tot Pct / Residual	BMI <23 (1)	23-26 (2)	27-30 (3)	>30 (4)	Row Total
<120	1	21	26	19	8	74
		14.4	24.4	21.2	14.0	45.1%
		28.4%	35.1%	25.7%	10.8%	
		65.6%	48.1%	40.4%	25.8%	
		12.8%	15.9%	11.6%	4.9%	
		6.6	1.6	-2.2	-6.0	
120 - 129	2	5	15	16	17	53
		10.3	17.5	15.2	10.0	32.3%
		9.4%	28.3%	30.2%	32.1%	
		15.6%	27.8%	34.0%	54.8%	
		3.0%	9.1%	9.8%	10.4%	
		-5.3	-2.5	.8	7.0	
130 & over	3	6	13	12	6	37
		7.2	12.2	10.6	7.0	22.6%
		16.2%	35.1%	32.4%	16.2%	
		18.8%	24.1%	25.5%	19.4%	
		3.7%	7.9%	7.3%	3.7%	
		-1.2	.8	1.4	-1.0	
	Column Total	32 19.5%	54 32.9%	47 28.7%	31 18.9%	164 100.0%

Chi-Square	Value	DF	Significance
Pearson	14.48177	6	.02469
Likelihood Ratio	14.42459	6	.02524
Mantel-Haenszel test for linear association	4.34697	1	.03707

Minimum Expected Frequency - 6.994

Statistic	Value	ASE1	Val/ASE0	Approximate Significance
Kendall's Tau-b	.15798	.06483	2.44548	
Kendall's Tau-c	.16274	.06655	2.44548	
Gamma	.22701	.09244	2.44548	
Somers' D :				
symmetric	.15761	.06467	2.44548	
with A.AVSPG1 dependent	.14748	.06010	2.44548	
with BMIGR.R1 dependent	.16924	.06999	2.44548	
Pearson's R	.16331	.07409	2.10682	.03667 *4
Spearman Correlation	.18423	.07539	2.38573	.01820 *4

PHYSICAL ACTIVITY LEVEL - FREQUENCY DISTRIBUTION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
not very active	1	48	29.3	29.3	29.3
fairly active	2	46	28.0	28.0	57.3
very active	3	70	42.7	42.7	100.0
	Total	164	100.0	100.0	

SJ

HISTORY OF HIGH BLOOD PRESSURE

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
no	0	109	66.5	66.5	66.5
yes	1	55	33.5	33.5	100.0
	Total	164	100.0	100.0	

HISTORY OF HYPERTENSION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO	0	153	93.3	93.3	93.3
YES	1	11	6.7	6.7	100.0
	Total	164	100.0	100.0	

MOTHER WITH HISTORY OF HYPERTENSION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
no	0	63	38.4	38.4	38.4
don't know	1	10	6.1	6.1	44.5
yes	2	91	55.5	55.5	100.0
	Total	164	100.0	100.0	

FATHER WITH HISTORY OF HYPERTENSION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
no	0	112	68.3	68.3	68.3
yes	2	45	27.4	27.4	95.7
	3	7	4.3	4.3	100.0
	Total	164	100.0	100.0	

BROTHER WITH HIGH BLOOD PRESSURE

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
No	0	127	77.4	77.4	77.4
Don't Know	1	13	7.9	7.9	85.4
Yes	2	24	14.6	14.6	100.0
	Total	164	100.0	100.0	

SISTER WITH HIGH BLOOD PRESSURE

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
no	0	129	78.7	78.7	78.7
yes	1	35	21.3	21.3	100.0
	Total	164	100.0	100.0	

GRAND PARENTS WITH HIGH HISTORY OF HYPERTENSION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
none	0	28	17.1	17.1	17.1
don't know	1	84	51.2	51.2	68.3
at least one	2	52	31.7	31.7	100.0
	Total	164	100.0	100.0	

SJ

- - Description of Subpopulations - -

Summaries of SBP
By levels of ever had high bp

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
BP.HIGH	0	no	118.4633	11.4240	109
BP.HIGH	1	yes	130.3091	17.9702	55

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of ever had high bp

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	no	118.4633	11.4240	14094.7977	109
1	yes	130.3091	17.9702	17438.0788	55
Within Groups Total		122.4360	13.9516	31532.8764	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	5129.4791	1	5129.4791	26.3527	.0000
Within Groups	31532.8764	162	194.6474		

With fewer than three groups, the relationship is linear

Eta = .3740 Eta Squared = .1399

S1

- - Description of Subpopulations - -

Summaries of SBP
By levels of ever hypertensive

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
HYP.HIST	0	NO	120.9989	13.7893	153
HYP.HIST	1	YES	142.4242	17.4632	11

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of ever hypertensive

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	NO	120.9989	13.7893	28901.9165	153
1	YES	142.4242	17.4632	3049.6313	11
Within Groups Total		122.4360	14.0439	31951.5478	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	4710.8077	1	4710.8077	23.8846	.0000
With fewer than three groups, the relationship is linear					
Within Groups	31951.5478	162	197.2318		

Eta = .3585 Eta Squared = .1285

S1

- - Description of Subpopulations - -

Summaries of SBP
By levels of mother hypertensive

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
BP.MOMHI	0	no	122.2063	15.5456	63
BP.MOMHI	1	don't know	124.6000	17.7919	10
BP.MOMHI	2	yes	122.3571	14.4451	91

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of mother hypertensive

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	no	122.2063	15.5456	14983.3175	63
1	don't know	124.6000	17.7919	2848.9556	10
2	yes	122.3571	14.4451	18779.3651	91
Within Groups Total		122.4360	15.0798	36611.6381	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	50.7174	2	25.3587	.1115	.8945
Linearity	.3564	1	.3564	.0016	.9685
Dev. from Linearity	50.3610	1	50.3610	.2215	.6386
	R = .0031	R Squared = .0000			
Within Groups	36611.6381	161	227.4015		
	Eta = .0372	Eta Squared = .0014			

SJ

- - Description of Subpopulations - -

Summaries of SBP
By levels of dad hypertensive

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
BP.DADHI	0	no	121.8661	14.6341	112
BP.DADHI	2	yes	124.1222	15.6606	45
BP.DADHI	3		120.7143	17.8636	7

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of father hypertensive

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	no	121.8661	14.6341	23771.4355	112
2	yes	124.1222	15.6606	10791.1889	45
3		120.7143	17.8636	1914.6508	7
Within Groups Total		122.4360	15.0521	36477.2752	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	185.0803	2	92.5402	.4084	.6654
Linearity	79.6137	1	79.6137	.3514	.5542
Dev. from Linearity	105.4666	1	105.4666	.4655	.4960
	R = .0466	R Squared = .0022			
Within Groups	36477.2752	161	226.5669		
	Eta = .0711	Eta Squared = .0050			

SJ

- - Description of Subpopulations - -

Summaries of SBP
By levels of Sister with high BP

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
high BP.SIS	0	no	121.0284	13.2839	129
high BP.SIS	1	yes	127.6238	19.4617	35

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of Sisters hypertensive

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	no	121.0284	13.2839	22587.0624	129
1	yes	127.6238	19.4617	12877.7413	35
Within Groups Total		122.4360	14.7959	35464.8037	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	1197.5518	1	1197.5518	5.4703	.0206
Within Groups	35464.8037	162	218.9185		

With fewer than three groups, the relationship is linear

Eta = .1807 Eta Squared = .0327

SJ

- - Description of Subpopulations - -

Summaries of SBP
By levels of Brother with high BP

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
BP.BROS	0	No	120.9475	13.6740	127
BP.BROS	1	Don't Know	119.4103	16.8987	13
BP.BROS	2	Yes	131.9514	17.5735	24

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of Brothers BP

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	No	120.9475	13.6740	23559.0945	127
1	Don't Know	119.4103	16.8987	3426.8120	13
2	Yes	131.9514	17.5735	7103.0266	24
Within Groups Total		122.4360	14.5510	34088.9331	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	2573.4224	2	1286.7112	6.0771	.0029
Linearity	2018.6001	1	2018.6001	9.5337	.0024
Dev. from Linearity	554.8224	1	554.8224	2.6204	.1075
	R = .2346	R Squared = .0551			
Within Groups	34088.9331	161	211.7325		
	Eta = .2649	Eta Squared = .0702			

SK

QUALIFICATIONS

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
none	0	27	16.5	16.5	16.5
high school	1	83	50.6	50.6	67.1
professional	2	26	15.9	15.9	82.9
university	3	28	17.1	17.1	100.0
	Total	164	100.0	100.0	

JOB GRADE

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
lowest	1	20	12.2	12.2	12.2
2nd lowest	2	19	11.6	11.6	23.8
middle	3	28	17.1	17.1	40.9
2nd highest	4	74	45.1	45.1	86.0
highest	5	23	14.0	14.0	100.0
	Total	164	100.0	100.0	

JOB - LIKE

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
no/not much	0	28	17.1	17.1	17.1
like	1	70	42.7	42.7	59.8
very much	2	66	40.2	40.2	100.0
	Total	164	100.0	100.0	

JOB: HAPPY IN

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
not happy	0	36	22.0	22.0	22.0
fairly	1	45	27.4	27.4	49.4
happy	2	52	31.7	31.7	81.1
very	3	31	18.9	18.9	100.0
	Total	164	100.0	100.0	

SK

HAPPY IN JOB

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
not happy	0	36	22.0	22.0	22.0
fairly	1	45	27.4	27.4	49.4
happy	2	52	31.7	31.7	81.1
very	3	31	18.9	18.9	100.0
	Total	164	100.0	100.0	

JOB SECURITY

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
not secure	0	34	20.7	20.7	20.7
fairly	1	88	53.7	53.7	74.4
secure	2	42	25.6	25.6	100.0
	Total	164	100.0	100.0	

JOB STRESS

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
no/rare	0	20	12.2	12.2	12.2
fairly regularly	1	59	36.0	36.0	48.2
every day	2	85	51.8	51.8	100.0
	Total	164	100.0	100.0	

YEARS IN THE JOB

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
< 2 yrs	1	45	27.4	27.4	27.4
2-5 yrs	2	76	46.3	46.3	73.8
> 5 yrs	3	43	26.2	26.2	100.0
	Total	164	100.0	100.0	

SK

- - Description of Subpopulations - -

Summaries of SBP
By levels of academic qual

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
QUALIF	0	none	127.7160	16.7036	27
QUALIF	1	high school	121.0161	13.0197	83
QUALIF	2	professional	120.6538	17.5142	26
QUALIF	3	university	123.2083	15.8932	28

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of academic qual

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	none	127.7160	16.7036	7254.2675	27
1	high school	121.0161	13.0197	13899.9786	83
2	professional	120.6538	17.5142	7668.7179	26
3	university	123.2083	15.8932	6820.0347	28
Within Groups Total		122.4360	14.9254	35642.9987	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	1019.3568	3	339.7856	1.5253	.2100
Linearity	144.7414	1	144.7414	.6497	.4214
Dev. from Linearity	874.6154	2	437.3077	1.9631	.1438
	R = -.0628	R Squared = .0039			
Within Groups	35642.9987	160	222.7687		
	Eta = .1667	Eta Squared = .0278			

5X

- - Description of Subpopulations - -

Summaries of SBP
By levels of Job Grade

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
JOB.GRADE	1	lowest	131.2167	19.6592	20
JOB.GRADE	2	2nd lowest	123.7895	14.8473	19
JOB.GRADE	3	middle	118.8929	10.3088	28
JOB.GRADE	4	2nd highest	122.4842	15.3036	74
JOB.GRADE	5	highest	117.8406	11.6210	23

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of Job Grade

Value	Label	Mean	Std Dev	Sum of Sq	Cases
1	lowest	131.2167	19.6592	7343.2278	20
2	2nd lowest	123.7895	14.8473	3967.9357	19
3	middle	118.8929	10.3088	2869.3452	28
4	2nd highest	122.4842	15.3036	17096.6205	74
5	highest	117.8406	11.6210	2971.0266	23
Within Groups Total		122.4360	14.6764	34248.1558	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	2414.1998	4	603.5499	2.8020	.0277
Linearity	1411.1851	1	1411.1851	6.5515	.0114
Dev. from Linearity	1003.0147	3	334.3382	1.5522	.2032
	R = -.1962	R Squared = .0385			
Within Groups	34248.1558	159	215.3972		
	Eta = .2566	Eta Squared = .0658			

SK

- - Description of Subpopulations - -

Summaries of SBP
By levels of job stress

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
JOB.STRESS	0	no/rare	128.5583	23.6409	20
JOB.STRES	1	fairly regularly	121.4802	12.5681	59
JOB.STRES	2	every day	121.6588	13.8130	85

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of job stress

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	no/rare	128.5583	23.6409	10618.9597	20
1	fairly regularly	121.4802	12.5681	9161.4492	59
2	every day	121.6588	13.8130	16027.0503	85
Within Groups Total		122.4360	14.9133	35807.4592	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	854.8963	2	427.4482	1.9219	.1497
Linearity	448.4496	1	448.4496	2.0164	.1575
Dev. from Linearity	406.4467	1	406.4467	1.8275	.1783
	R = -.1106	R Squared = .0122			
Within Groups	35807.4592	161	222.4066		
	Eta = .1527	Eta Squared = .0233			

SL

DIET: COOK WITH SALT

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
nev./rare	0	18	11.0	11.0	11.0
s/time	1	10	6.1	6.1	17.1
always	2	136	82.9	82.9	100.0
	Total	164	100.0	100.0	

DIET:ADD SALT

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
Never/rarely	0	79	48.2	48.2	48.2
Sometimes	1	68	41.5	41.5	89.6
Always	2	17	10.4	10.4	100.0
	Total	164	100.0	100.0	

DIET: COFFEE INTAKE WEEKLY

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
none	0	70	42.7	42.7	42.7
1 - 7	1	56	34.1	34.1	76.8
over 7	2	38	23.2	23.2	100.0
	Total	164	100.0	100.0	

DIET: FIRED FOODS WEEKLY

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
rare	0	42	25.6	25.6	25.6
once pw	1	60	36.6	36.6	62.2
2 - 3	2	31	18.9	18.9	81.1
> 3	3	31	18.9	18.9	100.0
	Total	164	100.0	100.0	

DIET: HEALTY MEALS EATEN PER DAY

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
	0	9	5.5	5.5	5.5
	1	97	59.1	59.1	64.6
	2	49	29.9	29.9	94.5
	3	9	5.5	5.5	100.0
	Total	164	100.0	100.0	

DIET: RED MEAT WEEKLY

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
nev/rare	0	48	29.3	29.3	29.3
1-3 times pw	1	105	64.0	64.0	93.3
> times pw	2	11	6.7	6.7	100.0
	Total	164	100.0	100.0	

DIET: SPICY FOODS WEEKLY

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
not much	1	45	27.4	27.4	27.4
regularly	2	64	39.0	39.0	66.5
daily	3	55	33.5	33.5	100.0
	Total	164	100.0	100.0	

DIET: NORMAL DIET

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
non-vegetarian	0	114	69.5	69.5	69.5
part vegetarian	1	45	27.4	27.4	97.0
vegetarian	2	5	3.0	3.0	100.0
	Total	164	100.0	100.0	

SL

- - Description of Subpopulations - -

Summaries of SBP
By levels of Add salt

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
ADDSALT	0	Never/rarely	127.0338	17.7532	79
ADDSALT	1	Sometimes	118.0025	10.4536	68
ADDSALT	2	Always	118.8039	9.7664	17

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable A.SBP SBP
By levels of ADSALT Addsalt

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	Never/rarely	127.0338	17.7532	24583.7433	79
1	Sometimes	118.0025	10.4536	7321.5829	68
2	Always	118.8039	9.7664	1526.1242	17
Within Groups Total		122.4360	14.4100	33431.4504	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	3230.9051	2	1615.4525	7.7797	.0006
Linearity	2488.9286	1	2488.9286	11.9862	.0007
Dev. from Linearity	741.9765	1	741.9765	3.5732	.0605
	R = -.2606	R Squared = .0679			
Within Groups	33431.4504	161	207.6488		
	Eta = .2969	Eta Squared = .0881			

SL

- - Description of Subpopulations - -

Summaries of SBP
By levels of cook with salt

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
COOK SALT	0	nev./rare	120.0278	11.9024	18
COOK SALT	1	s/time	117.4667	17.1370	10
COOK SALT	2	always	123.1201	15.2013	136

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of cook with salt

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	nev./rare	120.0278	11.9024	2408.3472	18
1	s/time	117.4667	17.1370	2643.1000	10
2	always	123.1201	15.2013	31195.9273	136
Within Groups Total		122.4360	15.0046	36247.3745	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	414.9810	2	207.4905	.9216	.4000
Linearity	269.2099	1	269.2099	1.1957	.2758
Dev. from Linearity	145.7712	1	145.7712	.6475	.4222
	R = .0857	R Squared = .0073			
Within Groups	36247.3745	161	225.1390		
	Eta = .1064	Eta Squared = .0113			

SL

- - Description of Subpopulations - -

Summaries of SBP
By levels of eat heavily seasoned foods

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
SPICY	1	not much	123.9259	14.8317	45
SPICY	2	regularly	121.6016	16.8787	64
SPICY	3	daily	122.1879	12.8288	55

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of eat heavily seasoned foods

Value	Label	Mean	Std Dev	Sum of Sq	Cases
1	not much	123.9259	14.8317	9679.0309	45
2	regularly	121.6016	16.8787	17948.2010	64
3	daily	122.1879	12.8288	8887.2808	55
Within Groups Total		122.4360	15.0598	36514.5126	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	147.8429	2	73.9214	.3259	.7223
Linearity	65.5132	1	65.5132	.2889	.5917
Dev. from Linearity	82.3297	1	82.3297	.3630	.5477
	R = -.0423	R Squared = .0018			
Within Groups	36514.5126	161	226.7982		
	Eta = .0635	Eta Squared = .0040			

SL

- - Description of Subpopulations - -

Summaries of SBP
By levels of normal diet type

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
DIET	0	non-vegetarian	122.1082	14.9114	114
DIET	1	part vegetarian	123.4556	15.4969	45
DIET	2	vegetarian	120.7333	14.9711	5

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of normal diet type

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	non-vegetarian	122.1082	14.9114	25125.5546	114
1	part vegetarian	123.4556	15.4969	10566.7444	45
2	vegetarian	120.7333	14.9711	896.5333	5
Within Groups Total		122.4360	15.0751	36588.8324	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	73.5232	2	36.7616	.1618	.8508
Linearity	17.8841	1	17.8841	.0787	.7794
Dev. from Linearity	55.6391	1	55.6391	.2448	.6214
	R = .0221	R Squared = .0005			
Within Groups	36588.8324	161	227.2598		
	Eta = .0448	Eta Squared = .0020			

CROSSTABULATION OF SYSTOLIC BLOOD PRESSURE BY ADD SALT

SBP	Count / Exp Val / Row Pct / Col Pct / Tot Pct / Residual	ADDSALT Never/rarely 0	Sometimes 1	Always 2	Row Total
<120	1	27	39	8	74
		35.6	30.7	7.7	45.1%
		36.5%	52.7%	10.8%	
		34.2%	57.4%	47.1%	
		16.5%	23.8%	4.9%	
		-8.6	8.3	.3	
120 - 129	2	24	22	7	53
		25.5	22.0	5.5	32.3%
		45.3%	41.5%	13.2%	
		30.4%	32.4%	41.2%	
		14.6%	13.4%	4.3%	
		-1.5	.0	1.5	
130 & over	3	28	7	2	37
		17.8	15.3	3.8	22.6%
		75.7%	18.9%	5.4%	
		35.4%	10.3%	11.8%	
		17.1%	4.3%	1.2%	
		10.2	-8.3	-1.8	
	Column Total	79 48.2%	68 41.5%	17 10.4%	164 100.0%

Chi-Square	Value	DF	Significance
Pearson	16.09508	4	.00289
Likelihood Ratio	16.55866	4	.00235
Mantel-Haenszel test for linear association	9.63938	1	.00190

Minimum Expected Frequency - 3.835
Cells with Expected Frequency < 5 - 1 OF 9 (11.1%)

Statistic	Value	ASE1	Val/ASE0	Approximate Significance
Kendall's Tau-b	-.23065	.06792	-3.39130	
Kendall's Tau-c	-.21193	.06249	-3.39130	
Gamma	-.36722	.10471	-3.39130	
Somers' D :				
symmetric	-.23041	.06785	-3.39130	
with A.AVSPG1 dependent	-.24139	.07216	-3.39130	
with ADSALT dependent	-.22039	.06419	-3.39130	
Pearson's R	-.24318	.07229	-3.19099	.00170 *4
Spearman Correlation	-.25425	.07417	-3.34603	.00102 *4

SL

CROSSTABULATION OF SYSTOLIC BLOOD PRESSURE BY ADDING SALT

SBP	Count / Exp Val / Row Pct / Col Pct / Tot Pct / Residual	ADD SALT no 0	ADD SALT yes 1	Row Total
<120	1	27 35.6 36.5% 34.2% 16.5% -8.6	47 38.4 63.5% 55.3% 28.7% 8.6	74 45.1%
120 - 129	2	24 25.5 45.3% 30.4% 14.6% -1.5	29 27.5 54.7% 34.1% 17.7% 1.5	53 32.3%
130 & over	3	28 17.8 75.7% 35.4% 17.1% 10.2	9 19.2 24.3% 10.6% 5.5% -10.2	37 22.6%
	Column Total	79 48.2%	85 51.8%	164 100.0%

Chi-Square	Value	DF	Significance
Pearson	15.43501	2	.00044
Likelihood Ratio	15.96451	2	.00034
Mantel-Haenszel test for linear association	13.74047	1	.00021

Minimum Expected Frequency - 17.823

Statistic	Value	ASE1	Val/ASE0	Approximate Significance
Kendall's Tau-b	-.26470	.06987	-3.75962	
Kendall's Tau-c	-.29952	.07967	-3.75962	
Gamma	-.44775	.10870	-3.75962	
Somers' D :				
symmetric	-.26265	.06933	-3.75962	
with A.AVSPG1 dependent	-.29993	.07976	-3.75962	
with D.ADSLTG dependent	-.23362	.06131	-3.75962	
Pearson's R	-.29034	.07271	-3.86178	.00016 *4
Spearman Correlation	-.27943	.07391	-3.70412	.00029 *4

SM

ALCOHOL: AGE STARTED DRINKING

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
non-drinker	0	46	28.0	28.0	28.0
under 18	1	39	23.8	23.8	51.8
18 to 21	2	49	29.9	29.9	81.7
>21	3	30	18.3	18.3	100.0
	Total	164	100.0	100.0	

ALCOHOL: ALCOHOL DRINKING STATUS

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
no	0	46	28.0	28.0	28.0
yes	1	118	72.0	72.0	100.0
	Total	164	100.0	100.0	

ALCOHOL: EVER DRANK

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
no	0	38	23.2	23.2	23.2
yes	1	126	76.8	76.8	100.0
	Total	164	100.0	100.0	

SM

- - Description of Subpopulations - -

Summaries of SBP
By levels of alcohol drinker

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
DRNKER	0	no	122.8696	17.3076	46
DRNKER	1	yes	122.2669	14.0726	118

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of alcohol drinker

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	no	122.8696	17.3076	13479.8285	46
1	yes	122.2669	14.0726	23170.5078	118
Within Groups Total		122.4360	15.0412	36650.3363	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	12.0193	1	12.0193	.0531	.8180
With fewer than three groups, the relationship is linear					
Within Groups	36650.3363	162	226.2366		

Eta = .0181 Eta Squared = .0003

SM

- - Description of Subpopulations - -

Summaries of SBP
By levels of age started drink

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
DRINK.AGE	0	non-drinker	122.8696	17.3076	46
DRINK.AGE	1	under 18	121.3504	16.0642	39
DRINK.AGEE	2	18 to 21	120.2619	12.9064	49
DRINK.AGE	3	>21	126.7333	12.5127	30

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of age started drink

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	non-drinker	122.8696	17.3076	13479.8285	46
1	under 18	121.3504	16.0642	9806.2108	39
2	18 to 21	120.2619	12.9064	7995.6111	49
3	>21	126.7333	12.5127	4540.4778	30
Within Groups Total		122.4360	14.9629	35822.1282	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	840.2273	3	280.0758	1.2510	.2932
Linearity	90.4474	1	90.4474	.4040	.5259
Dev. from Linearity	749.7799	2	374.8899	1.6745	.1907
	R = .0497	R Squared = .0025			
Within Groups	35822.1282	160	223.8883		
	Eta = .1514	Eta Squared = .0229			

SM

- - Description of Subpopulations - -

Summaries of SBP
By levels of amtount drink weekly

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
DRINK.AMT	0	none/ v. rare	121.4249	14.5225	91
DRINK.AMT	1	<8 units	123.9152	14.8039	55
DRINK.AMT	2	>8 units	123.0278	18.1934	18

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of amtount drink weekly

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	none/ v. rare	121.4249	14.5225	18981.2369	91
1	<8 units	123.9152	14.8039	11834.4374	55
2	>8 units	123.0278	18.1934	5627.0139	18
Within Groups Total		122.4360	15.0450	36442.6881	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	219.6674	2	109.8337	.4852	.6165
Linearity	137.7535	1	137.7535	.6086	.4365
Dev. from Linearity	81.9139	1	81.9139	.3619	.5483
	R = .0613	R Squared = .0038			
Within Groups	36442.6881	161	226.3521		
	Eta = .0774	Eta Squared = .0060			

SM

- - Description of Subpopulations - -

Summaries of SBP
By levels of ever a alcohol drinker

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
DRINK EVER	0	no	124.3465	18.5109	38
DRINK EVER	1	yes	121.8598	13.7996	126

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of ever a alcohol drinker

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	no	124.3465	18.5109	12678.1323	38
1	yes	121.8598	13.7996	23803.6896	126
Within Groups Total		122.4360	15.0065	36481.8219	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	180.5336	1	180.5336	.8017	.3719
With fewer than three groups, the relationship is linear					
Within Groups	36481.8219	162	225.1964		

Eta = .0702 Eta Squared = .0049

SN

SMOKING STATUS

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
no	0	123	75.0	75.0	75.0
yes	1	41	25.0	25.0	100.0
	Total	164	100.0	100.0	

AMOUNT SMOKED DAILY

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
non-smoker	0	123	75.0	75.0	75.0
<10	1	27	16.5	16.5	91.5
>10	2	14	8.5	8.5	100.0
	Total	164	100.0	100.0	

AGE STARTED SMOKING

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
non-smoker	0	115	70.1	70.1	70.1
<age 18	1	27	16.5	16.5	86.6
>age 18	2	22	13.4	13.4	100.0
	Total	164	100.0	100.0	

EVER SMOKED

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
no	0	99	60.4	60.4	60.4
yes	1	65	39.6	39.6	100.0
	Total	164	100.0	100.0	

PASSIVE SMOKING

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
very rarely	0	48	29.3	29.3	29.3
sometimes	1	57	34.8	34.8	64.0
daily	2	59	36.0	36.0	100.0
	Total	164	100.0	100.0	

SN

- - Description of Subpopulations - -

Summaries of SBP
By levels of SMOKER

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
SMOKER	0	no	124.0054	15.8291	123
SMOKER	1	yes	117.7276	11.0476	41

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of smoker

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	no	124.0054	15.8291	30568.4964	123
1	yes	117.7276	11.0476	4881.9864	41
Within Groups Total		122.4360	14.7929	35450.4828	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	1211.8727	1	1211.8727	5.5380	.0198
With fewer than three groups, the relationship is linear					
Within Groups	35450.4828	162	218.8301		

Eta = .1818 Eta Squared = .0331

SN

- - Description of Subpopulations - -

Summaries of SBP
By levels of age started smoking

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
SMOKE.AGE	0	non-smoker	124.5362	15.7064	115
SMOKE.AGE	1	<age 18	116.4321	13.3739	27
SMOKE.AGE	2	>age 18	118.8258	10.0522	22

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of age started smoking

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	non-smoker	124.5362	15.7064	28122.7101	115
1	<age 18	116.4321	13.3739	4650.4033	27
2	>age 18	118.8258	10.0522	2121.9710	22
Within Groups Total		122.4360	14.7221	34895.0844	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	1767.2711	2	883.6356	4.0769	.0187
Linearity	1222.5132	1	1222.5132	5.6405	.0187
Dev. from Linearity	544.7580	1	544.7580	2.5134	.1148
	R = -.1826	R Squared = .0333			
Within Groups	34895.0844	161	216.7397		
	Eta = .2196	Eta Squared = .0482			

SN

- - Description of Subpopulations - -

Summaries of SBP
By levels of amt. smoke daily

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
SMOKE.AMT	0	non-smoker	124.0054	15.8291	123
SMOKE.AMT	1	<10	118.2037	12.5388	27
SMOKE.AMT	2	>10	116.8095	7.7277	14

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of amt. smoke daily

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	non-smoker	124.0054	15.8291	30568.4964	123
1	<10	118.2037	12.5388	4087.7407	27
2	>10	116.8095	7.7277	776.3254	14
Within Groups Total		122.4360	14.8350	35432.5625	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	1229.7930	2	614.8965	2.7940	.0641
Linearity	1144.4799	1	1144.4799	5.2003	.0239
Dev. from Linearity	85.3131	1	85.3131	.3876	.5344
	R = -.1767	R Squared = .0312			
Within Groups	35432.5625	161	220.0780		
	Eta = .1831	Eta Squared = .0335			

SN

- - Description of Subpopulations - -

Summaries of SBP
By levels of Ever Smoked

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
SMOK.EVER	0	no	125.4360	16.0141	99
SMOK.EVER	1	yes	117.8667	12.0428	65

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of Ever Smoked

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	no	125.4360	16.0141	25132.3726	99
1	yes	117.8667	12.0428	9281.8444	65
Within Groups Total		122.4360	14.5751	34414.2171	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	2248.1385	1	2248.1385	10.5828	.0014

With fewer than three groups, the relationship is linear

Within Groups	34414.2171	162	212.4334

Eta = .2476 Eta Squared = .0613

ACCOMODATION - CROWDING : FREQUENCY DISTRIBUTION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
highest crowding	1	42	25.6	25.6	25.6
medium crowding	2	76	46.3	46.3	72.0
lowest crowding	3	46	28.0	28.0	100.0
	Total	164	100.0	100.0	

ACCOMODATION - ADEQUACY : FREQUENCY DISTRIBUTION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
not/reasonably	0	54	32.9	32.9	32.9
very adequate	1	110	67.1	67.1	100.0
	Total	164	100.0	100.0	

- - Description of Subpopulations - -

Summaries of SBP
By levels of Accommodation adequate

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
ACOM.OK	0	not/reasonably	116.4414	12.9929	54
ACOM.OK	1	very adequate	125.3788	15.0905	110

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable A.SBP SBP
By levels of AC.OK Accom. adequate

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	not/reasonably	116.4414	12.9929	8947.2865	54
1	very adequate	125.3788	15.0905	24821.9394	110
Within Groups Total		122.4360	14.4379	33769.2259	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	2893.1296	1	2893.1296	13.8791	.0003
Within Groups	33769.2259	162	208.4520		

With fewer than three groups, the relationship is linear

Eta = .2809 Eta Squared = .0789

- - Description of Subpopulations - -

Summaries of SBP
By levels of Accommodation Crowding

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
ACOM.CRWDG	1	highest crowding	117.9762	10.4492	42
ACOM.CRWDG	2	medium crowding	122.6316	13.5956	76
ACOM.CRWDG	3	lowest crowding	126.1848	19.3437	46

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable A.SBP SBP
By levels of AC.CRWDG Accom.Crowding

Value	Label	Mean	Std Dev	Sum of Sq	Cases
1	highest crowding	117.9762	10.4492	4476.5873	42
2	medium crowding	122.6316	13.5956	13862.9620	76
3	lowest crowding	126.1848	19.3437	16838.0682	46
Within Groups Total		122.4360	14.7816	35177.6175	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	1484.7380	2	742.3690	3.3977	.0359
Linearity	1472.3647	1	1472.3647	6.7387	.0103
Dev. from Linearity	12.3733	1	12.3733	.0566	.8122
	R = .2004	R Squared = .0402			
Within Groups	35177.6175	161	218.4945		
	Eta = .2012	Eta Squared = .0405			

SP

EFFECT OF RACISM ON CAREER

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
very little	0	47	28.7	28.7	28.7
not sure	1	46	28.0	28.0	56.7
quite a bit	2	71	43.3	43.3	100.0
	Total	164	100.0	100.0	

RACISM FROM CLIENTS

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
none/little	0	133	81.1	81.1	81.1
quite a bit	1	26	15.9	15.9	97.0
very much	2	5	3.0	3.0	100.0
	Total	164	100.0	100.0	

RACISM FROM WORK COLLEAGUES

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
none	0	109	66.5	66.5	66.5
quite a bit	1	55	33.5	33.5	100.0
	Total	164	100.0	100.0	

RACISM FROM THE GENERAL PUBLIC WHILE ON THE JOB

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
not at all	0	68	41.5	41.5	41.5
a little	1	65	39.6	39.6	81.1
quite a bit	2	31	18.9	18.9	100.0
	Total	164	100.0	100.0	

ANGERED BY RACISM

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
rarely	0	16	9.8	9.8	9.8
sometimes	1	76	46.3	46.3	56.1
weekly	2	48	29.3	29.3	85.4
daily	3	24	14.6	14.6	100.0
	Total	164	100.0	100.0	

FEEL TENSE BECAUSE OF RACISM

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
nev/rare	0	80	48.8	48.8	48.8
fairly often	1	8	4.9	4.9	53.7
v. often	2	76	46.3	46.3	100.0
	Total	164	100.0	100.0	

FREQUENCY RACISM CAUSES FEELING OF INSECURITY

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
nev/rare	0	96	58.5	58.5	58.5
s/times	1	45	27.4	27.4	86.0
regularly	2	23	14.0	14.0	100.0
	Total	164	100.0	100.0	

UNSAFE BECAUSE OF RACISM

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
not at all	0	55	33.5	33.5	33.5
a little	1	48	29.3	29.3	62.8
quite a bit	2	61	37.2	37.2	100.0
	Total	164	100.0	100.0	

SP

EFFECT OF RACISM ON HEALTH

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
none/little	0	104	63.4	63.4	63.4
v. much	1	60	36.6	36.6	100.0
	Total	164	100.0	100.0	

FREQUENCY OF NEGATIVE EFFECT OF RACISM

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
none/little	0	90	54.9	54.9	54.9
v. regularly	1	74	45.1	45.1	100.0
	Total	164	100.0	100.0	

HAS RACISM AFFECTED YOU ALL LIFE -UK

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO	0	42	25.6	25.6	25.6
YES	1	122	74.4	74.4	100.0
	Total	164	100.0	100.0	

SP

- - Description of Subpopulations - -

Summaries of SBP
By levels of Racism:Angry

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
RACISM.ANGRY	0	rarely	130.6979	16.6443	16
RACISM.ANGRY	1	sometimes	123.0789	16.5227	76
RACISM.ANGRY	2	weekly	122.5278	12.6448	48
RACISM.ANGRY	3	daily	114.7083	9.2307	24

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of Racism:Angry

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	rarely	130.6979	16.6443	4155.5122	16
1	sometimes	123.0789	16.5227	20475.0263	76
2	weekly	122.5278	12.6448	7514.9074	48
3	daily	114.7083	9.2307	1959.7361	24
Within Groups Total		122.4360	14.5999	34105.1820	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	2557.1735	3	852.3912	3.9989	.0089
Linearity	2055.8941	1	2055.8941	9.6450	.0022
Dev. from Linearity	501.2794	2	250.6397	1.1758	.3112
	R = -.2368	R Squared = .0561			
Within Groups	34105.1820	160	213.1574		
	Eta = .2641	Eta Squared = .0697			

SP

- - Description of Subpopulations - -

Summaries of SBP
By levels of Racism: Career

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
RACISM.CAREER	0	very little	126.9184	17.6530	47
RACISM.CAREERR	1	not sure	124.1703	16.2026	46
RACISM.CAREER	2	quite a bit	118.3451	10.8516	71

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of Racism: Career

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	very little	126.9184	17.6530	14334.9374	47
1	not sure	124.1703	16.2026	11813.5272	46
2	quite a bit	118.3451	10.8516	8242.9624	71
Within Groups Total		122.4360	14.6154	34391.4270	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	2270.9286	2	1135.4643	5.3156	.0058
Linearity	2193.5206	1	2193.5206	10.2687	.0016
Dev. from Linearity	77.4080	1	77.4080	.3624	.5480
	R = -.2446	R Squared = .0598			
Within Groups	34391.4270	161	213.6113		
	Eta = .2489	Eta Squared = .0619			

SP

- - Description of Subpopulations - -

Summaries of SBP
By levels of racism from clients

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
RACISM.CLIENT	0	none/little	122.8759	15.8720	133
RACISM.CLIENT	1	quite a bit	119.1474	8.3946	26
RACISM.CLIENT	2	very much	127.8333	17.2812	5

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of racism from clients

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	none/little	122.8759	15.8720	33253.4808	133
1	quite a bit	119.1474	8.3946	1761.7404	26
2	very much	127.8333	17.2812	1194.5556	5
Within Groups Total		122.4360	14.9968	36209.7767	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	452.5788	2	226.2894	1.0062	.3679
Linearity	26.0921	1	26.0921	.1160	.7338
Dev. from Linearity	426.4867	1	426.4867	1.8963	.1704
	R = -.0267	R Squared = .0007			
Within Groups	36209.7767	161	224.9054		
	Eta = .1111	Eta Squared = .0123			

SP

- - Description of Subpopulations - -

Summaries of SBP
By levels of Racism from colleagues reversed

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
RACISM.COLG	0	quite a bit	118.4879	11.2628	55
RACISM.COLG	1	none	124.4281	16.2511	109

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of Racism from colleagues reversed

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	quite a bit	118.4879	11.2628	6849.9364	55
1	none	124.4281	16.2511	28522.5204	109
Within Groups Total		122.4360	14.7766	35372.4568	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	1289.8988	1	1289.8988	5.9075	.0162
Within Groups	35372.4568	162	218.3485		

With fewer than three groups, the relationship is linear

Eta = .1876 Eta Squared = .0352

SP

- - Description of Subpopulations - -

Summaries of SBP
By levels of racism make insecure

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
RACISM.INSECUR	0	nev/rare	124.4757	17.2219	96
RACISM.INSECUR	1	s/times	120.9926	11.5855	45
RACISM.INSECURR	2	regularly	116.7464	7.8114	23

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable A.SBP SBP
By levels of R.NSECUR racism make insecure

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	nev/rare	124.4757	17.2219	28176.3877	96
1	s/times	120.9926	11.5855	5905.8864	45
2	regularly	116.7464	7.8114	1342.3816	23
Within Groups Total		122.4360	14.8334	35424.6558	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	1237.6997	2	618.8499	2.8126	.0630
Linearity	1233.6212	1	1233.6212	5.6066	.0191
Dev. from Linearity	4.0785	1	4.0785	.0185	.8919
	R = -.1834	R Squared = .0336			
Within Groups	35424.6558	161	220.0289		
	Eta = .1837	Eta Squared = .0338			

SP

- - Description of Subpopulations - -

Summaries of SBP
By levels of racism - tense

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
RACISM.TENSE	0	nev/rare	125.1354	17.0301	80
RACISM.TENSE	1	fairly often	118.7917	9.5114	8
RACISM.TENSE	2	v. often	119.9781	12.6326	76

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of racism tense

Value	Label	Mean	Std Dev	Sum of Sq	Cases
0	nev/rare	125.1354	17.0301	22912.0052	80
1	fairly often	118.7917	9.5114	633.2639	8
2	v. often	119.9781	12.6326	11968.7412	76
Within Groups Total		122.4360	14.8521	35514.0103	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	1148.3452	2	574.1726	2.6030	.0772
Linearity	1040.4743	1	1040.4743	4.7169	.0313
Dev. from Linearity	107.8709	1	107.8709	.4890	.4854
	R = -.1685	R Squared = .0284			
Within Groups	35514.0103	161	220.5839		
	Eta = .1770	Eta Squared = .0313			

- - Description of Subpopulations - -

Summaries of SBP
By levels of Sessions completed - REPEATED MEASURES

Variable	Value	Label	Mean	Std Dev	Cases
For Entire Population			122.4360	14.9974	164
SESSIONS	1		123.6667	20.4031	22
SESSIONS	2		123.3467	17.5051	25
SESSIONS	3		121.1410	11.7903	26
SESSIONS	4		122.2361	15.0257	24
SESSIONS	5		118.0750	12.7521	20
SESSIONS	6		124.0496	13.3571	47

Total Cases = 164

- - Analysis of Variance - -

Dependent Variable SBP
By levels of Sessions completed

Value	Label	Mean	Std Dev	Sum of Sq	Cases
1		123.6667	20.4031	8742.0000	22
2		123.3467	17.5051	7354.2733	25
3		121.1410	11.7903	3475.2607	26
4		122.2361	15.0257	5192.7731	24
5		118.0750	12.7521	3089.6931	20
6		124.0496	13.3571	8206.9953	47
Within Groups Total		122.4360	15.1074	36060.9955	164

Source	Sum of Squares	d.f.	Mean Square	F	Sig.
Between Groups	601.3600	5	120.2720	.5270	.7556
Linearity	1.5543	1	1.5543	.0068	.9343
Dev. from Linearity	599.8057	4	149.9514	.6570	.6228
	R = -.0065	R Squared = .0000			
Within Groups	36060.9955	158	228.2341		
	Eta = .1281	Eta Squared = .0164			