RELATIONSHIPS BETWEEN PERSONALITY TRAITS AND CARDIOVASCULAR DISEASES IN THE GENERAL POPULATION

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DECLARATION

I, Martha C. Whiteman, declare that this thesis is my own composition.

I administered personality questionnaires to the Edinburgh Artery Study participants in 1995 and prepared and analyzed the data pertaining to those questionnaires. I also carried out further analysis, not previously performed, on additional personality data collected earlier in the study. The Edinburgh Artery Study was already in progress when I undertook my project, and therefore, the earlier information was collected by others. Both parts of the analysis are included in the thesis.

Some of the findings shown in Chapter 8 were published during the time I was preparing this thesis (Whiteman et al, 1997). The paper is included in the appendices.

Signed....

Date.....25 November, 1997.....

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ABSTRACT

This thesis is an exploration of the relationships between personality and cardiovascular diseases. The established physical risk factors of hypertension, hypercholesterolaemia and smoking can only account for 50% of new disease. Accordingly, behavioural epidemiologists investigated the added impact of aggressive and competitive behaviour named Type A. Continued study suggested that the hostility component of this behaviour was the central element for cardiovascular disease risk. Inconsistent findings led to recommendations for clear definitions of psychological constructs, use of standard and reliable measures, and separation of objective and subjective disease endpoints. The emergence of the Five Factor model, which posits that personality can be described on the five broad dimensions of neuroticism, extraversion, agreeableness, openness to experience and conscientiousness, offered a way to standardize and amalgamate personality-health research.

The aims of this study were to apply the five-factor model to cardiovascular diseases, and to follow up previous research on hostility and anger, using objective measures of clinical and subclinical disease. Prospective analysis of hostility data helped to determine the causal directions of the relationship, and the use of standard, reproducible instruments to assess the five factors helped further elucidate the role of personality in cardiovascular disease risk.

The study is based on a cohort of 809 men and 783 women, aged 55-74 years, who were randomly sampled from the general population in Edinburgh. At the baseline medical examination in 1988, the Bedford-Foulds Personality Deviance

Scales, which elicit hostility and dominance-related traits, were administered. The study sample was followed up for five years to determine the incidence of fatal and nonfatal myocardial infarction, angina pectoris, and peripheral arterial disease. After the five-year follow-up, the NEO-FFI, a short inventory that measures the five factors, and the State-Trait Anger Expression Inventory were administered by post to assess their association with the prevalence of cardiovascular disease.

Submissiveness was protective of objectively-determined nonfatal myocardial infarction over five years, particularly in women. Traits associated with neuroticism were predictive of subjectively defined angina pectoris. Associations between anger-out and prevalent myocardial infarction in men, and neuroticism and prevalent intermittent claudication in women, were prominent. Different aspects of anger were inconsistently associated with other disease outcomes. Low agreeableness was not consistently independently associated with disease.

These results confirm the strength and direction of personality and cardiovascular disease associations previously observed, and can help improve our prediction of risk. They indicate that the five factor model's dimensions may be too heterogeneous for this type of research, and that they should be used alongside narrower measures. However, because of the important interaction between many personal factors, such as life stress, coping and especially socioeconomic status, studies examining only one of these elements may be too restrictive. Research into the biological mechanisms of the association is also important and should continue. Only by integrating the individual strands of research can we understand the complex effects of personality and other non-physical factors on cardiovascular diseases.

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LIST OF ABBREVIATIONS

ABPI	ankle brachial pressure index
BMI	body mass index
CHD	coronary heart disease
CI	confidence interval
CVD	cardiovascular disease
EAS	Edinburgh Artery Study
FFM	five-factor model
HDL-chol	high-density lipoprotein cholesterol
Ho scale	hostility scale of the MMPI
JAS	Jenkins Activity Survey
MI	myocardial infarction
MMPI	Minnesota Multiphasic Personality Inventory
NEO-FFI	NEO Five Factor Inventory
PAD	peripheral arterial disease
PDS	Bedford-Foulds Personality Deviance Scales
PH	Potential for Hostility
sd	standard deviation
SES	socioeconomic status
SI	Structured Interview
STAXI	State-Trait Anger Expression Inventory
TABP	Type A Behaviour Pattern

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CHAPTER ONE

Introduction

1.1 THE PROBLEM OF CORONARY HEART DISEASE

Each year, coronary heart disease (CHD) is named as the cause of death for over 180,000 people in the UK, and 500,000 in the USA (Durrington, 1993). This accounts for approximately one third to one half of all deaths in these countries (Tunstall-Pedoe, 1997). In Scotland, there are 17,000 deaths and 70,000 hospital admissions a year, at a cost of £140 million in Scottish NHS spending alone (PHPU, 1996). Nearly half of the 17,000 deaths are women, and half occur before the age of 75 (PHPU, 1996). In men, 40% of deaths in middle age are from CHD. Premature death is therefore a huge problem both in personal and economic terms (Durrington, 1993). In 60% of all fatal myocardial infarctions (MI), death occurs in the first hour of symptom onset, which is too quickly for any treatment to be of use (Marmot and Mann, 1987).

In the 20th century, in industrialized countries such as the USA and the UK, CHD overtook infectious diseases as the major cause of death (Marmot and Mann, 1987). CHD is still the biggest single cause of death in the UK, although there are differences even within the country (Central Health Monitoring Unit - CHMU - 1994). Mortality rates are higher in the north of England than the south, and higher in Scotland and Northern Ireland than in England and Wales (CHMU, 1994). There has been a recent decline, however, by as much as 50% in some developed nations (Henderson, 1996), and rates in Scotland have also been

declining (Tunstall-Pedoe 1997).

There are major differences even between industrialized countries in the prevalence of CHD: in Japan the occurrence of new cases each year was reported as 15 per 10,000 individuals, but it was 198 per 10,000 in Finland (Keys, 1980). Regional variations have provided important clues to researchers looking for causes, especially as people who migrate from a lower-risk country to a higher risk country start to experience CHD at nearly the same rate as the adopted country (Tunstall-Pedoe, 1997). This suggested that environmental and behavioural differences may have an important role in CHD aetiology, and has led to the identification of a number of risk factors (Marmot and Mann, 1987).

In this introductory chapter the main risk factors for CHD will be discussed, and the different syndromes of CHD will be described. This lays the foundation for the review, in chapters two and three, of studies investigating Type A behaviour and hostility as risk factors for CHD. The issue of quantifying personality is addressed in chapter four. The aims and objectives of this thesis, namely the further exploration of personality and CHD in the light of previous findings, are fully outlined in chapter five. The methods of the study are described in chapters six and seven. Chapters eight and nine present the results of the study, and the issues raised by the findings are discussed in chapter ten. In chapter eleven, the implications and recommendations for future research are considered.

1.2 MAJOR RISK FACTORS FOR CHD

The concept of risk factors developed out of research from the 1950s that first found cholesterol, smoking and blood pressure to be important for CHD (Stamler et al, 1959). The concept was further established in longitudinal epidemiological studies such as the Seven Countries Study (Keys, 1980) and the Framingham Study in the USA (Dawber, 1980). It became apparent through this work that there were multiple factors influencing the risk of CHD. Non-modifiable factors included increasing age, being male and having a family history of CHD, all of which were quite strong predictors of disease. Of the potentially modifiable factors, raised serum cholesterol levels (especially low-density lipoprotein), hypertension and smoking have been very consistently implicated in the aetiology of CHD (Stamler, 1992). Other factors such as fibrinogen levels, obesity, diabetes mellitus, lack of exercise, stress, socioeconomic deprivation and personality have also been investigated (Jenkins, 1976; Thelle et al, 1976, Logan et al, 1978; Dawber, 1980; Keys, 1980; Rose, 1985; Shaper et al, 1982, 1985; MRFIT Research Group, 1982; Martin et al, 1986; Marmot and Mann 1987; Shaper, 1988; Durrington 1993; CHMU, 1994). These risks act in a multiplicative fashion. For instance, CHD death rates in white male smokers were 2/1000 in non-smokers with low blood cholesterol and blood pressure, but were 17/1000 in smokers with hypertension and raised serum cholesterol levels (MRFIT, 1990). In addition, an individual with moderate increases in many of the factors can be at higher risk than a person with greatly increased levels of just one of the factors (PHPU, 1996; Kannel et al., 1986; Stamler, 1992). The risk factors

are discussed in turn below.

1.2.1 Age

In populations in which one or more risk factors are likely to be raised, both men and women experience at least a 15-fold increased mortality rate between the 35-44 ages and 55-64 ages (Shaper, 1988; Tunstall-Pedoe, 1997). The rate rises more dramatically in women, who, because they have a lower risk at first, experience a 30% increase in risk between these two age groups. In America, CHD is the main cause of death in the over-65 age group (Harlan and Manolio, 1992). One reason for higher risk at older ages may be the greater cumulative effect that the rest of the risk factors exert (Shaper, 1988). However, coronary deaths are not inevitable with age, as illustrated by the different rates that occur across populations (Tunstall-Pedoe, 1997), and there is evidence that modification of other risk factors can affect morbidity and mortality in older as well as younger age groups (Harlan and Manolio, 1992).

1.2.2 Sex

Women and men seem to experience mild CHD with similar frequency, but more severe CHD occurs more often in men (Smith et al, 1990). The excess risk ranges from two- to six-fold, with the difference greater in younger age groups (Khaw and Barrett-Connor, 1992). A man has a 5-6 times higher rate of CHD mortality than a woman when both are 35-44 years of age (Shaper, 1988). Female rates lag behind male rates by 10-15 years, although the gap narrows, but

does not completely close, in the over-85 age-group (Khaw and Barrett-Connor, 1992; Tunstall-Pedoe, 1997).

1.2.3 Cigarette smoking

Smoking is a large modifiable contributor to CHD deaths and a very important contributor to ill-health (Doll et al, 1994). It was shown in the Framingham Study that the rate of CHD in smokers was two to three times greater than in non-smokers (Larson, 1995). This was also true in the Copenhagen City Heart Study (Jensen et al., 1991), in the MRFIT study (Kannel et al., 1986) and in the Finnmark study (Njolstad, Arnesen and Lund-Larsen, 1996). And, when looking specifically at the risk of first acute myocardial infarction in the Copenhagen City Heart Study, Nyboe et al (1991) found that the risk was increased by two to three per cent for each gram of tobacco smoked daily. Importantly, ex-smokers had the same risk as those who had never smoked, regardless of how long they had smoked or how long it had been since they quit. However, data from the British Regional Heart Study showed an increased prevalence of CHD even in those who gave up smoking (Shaper, 1988). It is unclear how smoking affects the cardiovascular system, but the effect may be mediated through levels of plasma fibrinogen (Tunstall-Pedoe, 1997). The consistency of the relationship, its strength and its independent effect on CHD do suggest that the relationship is likely to be causal, although smoking does not appear to increase risk of CHD when population levels of other risk factors are very low, such as in Japan (Marmot and Mann, 1987).

1.2.4 Hypertension

Raised blood pressure is associated with hypertensive heart disease, cerebrovascular disease, CHD and renal failure (Shaper, 1988). In populations where CHD is not as prevalent (eg. Japan), blood pressure is a strong risk factor for stroke and renal failure; in the USA or UK, where CHD rates are high, mild hypertension is a risk factor for CHD (Tunstall Pedoe, 1997, PHPU, 1996). In Scotland approximately 35% of CHD deaths may be attributable to raised blood pressure (PHPU, 1996). In non-smokers in the MRFIT study, the relative risk of CHD in the highest versus lowest quintile of systolic blood pressure was 2.70 to 4.42, depending on serum cholesterol levels (Neaton et al, 1984; Stamler, Wentworth and Neaton, 1986). Research is consistent in finding increased risk of CHD with a rise in blood pressure (Dawber, 1980; Keys, 1980; Kannel et al, 1986; MacMahon et al, 1990), but a positive effect for reversibility has been difficult to show (Tunstall -Pedoe, 1997). However, in countries in which both atherosclerosis and CHD are widely prevalent, hypertension is a very important risk for CHD (Shaper, 1988).

1.2.5 Cholesterol

Total cholesterol level is a strong and specific risk factor for CHD (eg. Neaton et al, 1984; Stamler, Wentworth and Neaton, 1986), and it shows good evidence for reversibility of risk when levels are lowered (Tunstall-Pedoe, 1997). It is transported in the bloodstream by lipoproteins, some of which are very-low density (VLDL), some which are low density (LDL) and some which are high

density (HDL; Shaper, 1988). Research suggests that LDL cholesterol is the atherogenic element in total serum cholesterol, and that HDL cholesterol may have a protective effect (Gordon et al, 1977; Kannel, 1983, 1987; Kannel et al, 1986; Castelli et al, 1986; Jensen et al, 1991; Hargreaves et al, 1991; Larson, 1995; Njolstad et al, 1996). VLDL cholesterol seems to have no impact on CHD risk (Shaper, 1988).

There is no threshold of cholesterol level below which there is no risk, but the risk becomes much more pronounced at high levels: a rise from 5-6 mmol/l produces a much smaller increase in risk than a rise from 9-10mmol/l (Durrington, 1993). Based on findings in the MRFIT study in the USA, optimal levels seem to be <4.7mmol/l, and the relative risk of CHD in the highest versus lowest quintile of cholesterol levels ranged from 2.45 to 4.00, depending on smoking and blood pressure (Neaton et al, 1984; Stamler, Wentworth and Neaton, 1986).

Serum triglycerides have also emerged as a risk factor in large studies (Shaper, 1988). The relationship, however, may not be straightforward: triglyceride levels also correlate with body mass index and serum cholesterol levels (positively with LDL and negatively with HDL; Shaper, 1988; Durrington, 1993; Tunstall Pedoe, 1997). Levels may also be influenced by diabetes mellitus, alcohol consumption and oral contraceptives and any rise in triglycerides may be secondary to these factors (Shaper, 1988).

1.3 OTHER RISK FACTORS FOR CHD

Although cigarette smoking, hypertension and serum cholesterol levels

have consistently been found to contribute to the risk of CHD, there are many other related factors. These have often been studied alongside the major factors and the knowledge of how these elements affect CHD continues to evolve.

1.3.1 Diet

In general, populations consuming diets low in saturated fat and cholesterol have low rates of CHD, and those consuming high amounts have high rates of CHD (Keys, 1980; Shaper, 1988). Some researchers see the Western diet, which is rich in saturated fats, animal products, refined sugars and low in fibre and other nutrients, as the most essential element in coronary risk (Stamler, 1992). However, in individuals the relationship between diet and cholesterol is not direct: there are modifying factors, such as the degree of obesity, genetic factors and diabetes mellitus (Shaper, 1988). Although no other environmental factor influences cholesterol level more than diet does, in different individuals the response varies. That is, data collected on diet will not accurately predict cholesterol level, which is why a person's diet is not such a strong risk factor as his or her serum cholesterol level (Durrington, 1993). Yet, in animals atherosclerosis can be slowed or even halted following a long term dietary change (Shaper, 1988), and recently, the protective effects of fruit and vegetables have gained attention (Wood and Oliver, 1992). In Scotland, policy statements have consistently highlighted a diet high in saturated fat and salt, and low in antioxidants, as a major contributor to its very high rate of premature CHD deaths (PHPU, 1996).

1.3.2 Obesity

In populations where CHD is of high prevalence, overweight individuals have a two-fold excess of risk of CHD (Shaper, 1988). Because obesity is also so closely associated with high blood pressure, raised LDL-cholesterol and lowered HDL-cholesterol, raised triglycerides and lack of physical activity, in multivariate models of risk it sometimes has not shown an independent effect on CHD (Keys, 1980; Larsson, Bjorntrop and Tibblin, 1981; Barrett-Connor 1985; Hubert, 1986). Yet an overweight person has an increased likelihood of having many other risk factors also present, and therefore the obesity is an important indicator of risk (Shaper, 1988). Distribution of body fat has also been studied. The 'female' pattern of subcutaneous fat, often carried on the hips and thighs, appears to be less dangerous than the 'male' pattern of intra-abdominal fat (eg., Gillum, 1987).

1.3.3 Alcohol consumption

Much of the published research on alcohol and CHD has been interpreted as meaning that regular, moderate drinking (approx 2 drinks per day) reduces the risk of heart disease, especially when compared to non-drinking group or heavy drinking groups (Marmot et al, 1981; Kannel 1987; Rimm et al, 1991). Other studies have shown a similar relationship with the extent of peripheral arterial disease (Jepson et al, 1995). Shaper (1988) is careful to note that the teetotal comparison group may be a peculiar one, as they may have a high mortality rate for reasons unconnected with alcohol. When this group is discounted, the research meerely indicates that lighter drinkers have a lower risk of CHD than heavier

drinkers. However, other studies have shown that lifelong abstainers do have increased risk of CHD over moderate drinkers (Tunstall-Pedoe, 1997). Some of the benefit may be derived from increased HDL cholesterol level in moderate drinkers (Woodward and Tunstall Pedoe, 1995). Although the effect of alcohol on CHD is not a direct one, it at least does not appear that moderate, regular drinking is harmful.

1.3.4 Exercise

Epidemiologists have noted that frequent, relatively intense exercise may halve the risk of CHD (Berlin and Colditz 1990). The nature of its effect is not entirely understood, but since taking exercise helps reduce blood pressure, combat obesity and improve cardiovascular and pulmonary health, one possibility is that it exerts its effect through changing the risk factor profile (Shaper, 1988; Tunstall Pedoe, 1997). The risk of peripheral arterial disease, for example, was found to be inversely related to physical activity in middle age (Housley et al, 1993). Exercise also increases cardiac efficiency and may reduce the frequency of ectopic beats (Marmot and Mann, 1987). Some research suggests that there is a threshold of activity: that bouts of exercise nearing maximal output are more beneficial than the same total output at lesser intensity (Morris et al, 1980, 1990; Lee, Hsieh and Paffenbarger, 1995). Others assert that even regular walking is beneficial (Powell et al, 1987). Another possibility may be that those who exercise are healthier to begin with than those who do not. Even so, there are other benefits of exercise apart from CHD prevention: it reduces the risk of stroke and helps to preserve

bone mass and muscle function, in addition to helping control weight and blood lipid levels (Tunstall-Pedoe, 1997).

1.3.5 Diabetes

CHD risk is increased 2-5 times in those with either insulin-dependent or non insulin-dependent diabetes (IDDM, NIDDM; Kannel 1985; Barrett-Connor et al, 1991). Women lose their natural protection from CHD if they have diabetes (McKeigue and Keen, 1992). Cholesterol levels are raised in NIDDM compared to the general population (Durrington, 1993). Hypertension in diabetes sufferers is also common (Reaven 1988), and in IDDM particularly this may reflect kidney damage and proteinuria, and proteinuria increases the risk of CHD to 30-40 times that in general population (Durrington, 1993). Unfortunately, a diabetic who has a myocardial infarction is also more likely to die from it than one who is not diabetic, so watching for signs of CHD in a diabetic is particularly important.

1.3.6 Family influences

Men who develop CHD before the age of 55 are three to five times more likely to have had a first degree relative who also had CHD (Durrington, 1993). The family environment may also influence cholesterol levels, blood glucose levels, smoking habits and blood pressure. The effect may be in part genetic but it is very difficult to separate genetic from environmental influences. Some families have history of hyperlipidaemia (affecting between 1 in 50 and 1 in 200 people), which increases the risk of premature CHD (Shaper, 1988; Durrington,

1993). The family history is therefore very important for the physician in calculating risk, although some of the family influence can be countered with effortful modification of cigarette smoking or diet, for example.

Familial hypercholesteroaemia (FH) is an inherited genetic condition and occurs in about 1 in 500 people in the UK and USA; approximately the same as IDDM (Shaper, 1988; Durrington, 1993). Young men with it can have myocardial infarctions (MI) in their mid 20s if untreated, and less than 50% of affected men, if untreated, survive to the age of 60. Even the male survivors will have had either an MI or angina by the age of 50. The same applies to 50% of females with FH, although only 15% have died by the age of 60 (Durrington, 1993). For those with FH, treatment is essential and usually requires drugs to lower cholesterol.

1.3.7 Fibrinogen/coagulation factors

Haemostatic and thrombogenic factors such as fibrinogen and factor VII have increasingly been shown to be strong predictors of risk of CHD (Meade, 1992) and peripheral arterial disease (Kannel et al, 1987). It is possible that fibrinogen may be responsible for much of the effect of smoking (Meade, 1992; Ernst and Resch 1993). Raised levels of Factor VII, a coagulation factor, also increase risk, although the level can be lowered by a reduction in fat intake (Miller et al, 1989).

1.3.8 Socioeconomic deprivation

Socioeconomic status (SES) is a consistent and strong predictor of morbidity and premature mortality (Adler et al, 1993). The incidence of CHD is two to three times higher in social classes IV and V compared to I-III (Whitehead, 1992). The relationship is apparent regardless of what indicator is used for SES: income, education or occupational status. It does not appear to be an artifact of ill individuals drifting downward (Fox, Goldblatt, and Jones, 1986), nor is the disparity explained by the higher rates of smoking and hypertension observed in lower SES groups (Smith et al, 1990). High poverty populations also suffer from poorer diet, and their situation poses problems for getting access to healthier foods (Kaplan, 1995).

1.3.9 Psychosocial factors

Despite the increased knowledge concerning the risks of CHD, only about 50% of new cases can be predicted by serum cholesterol, blood pressure and cigarette smoking (Jenkins, 1976; Schmidt, 1983; Dembroski and Costa, 1987). Because of this, behaviour and personality were also measured in epidemiological studies of CHD (Schmidt, 1983). However, opinion about the effects of personality and stress on CHD is varied because results are not always consistent (Miller et al, 1996; Tunstall-Pedoe, 1997). In part this is because measurement of behaviour is complicated. Inconsistencies also arise because it is difficult to separate the effects of personality from the effects of the physical factors, although statistical analysis has shown an independent effect of personality factors on CHD

risk (eg., Johnston, 1993; Miller et al, 1996). There have been at least two main fields of enquiry: one into stress (an environmental demand or an individual's response to those demands; eg., Marmot et al., 1997; Everson et al, 1997), and the other into specific behaviours such as hostile or competitive behavoiur patterns (eg. Type A behaviour: Friedman and Rosenman, 1959). Other investigators have concentrated on social support, coping, or work stress. The general trend of findings has implicated an aetiological role for personality factors in CHD, with certain aspects of personality, for instance, hostility, increasing risk by approximately 50-90% (Miller et al, 1996).

1.4 CORONARY HEART DISEASE: THE SYNDROMES

Coronary heart disease (CHD) results from a narrowing of arteries that supply blood to the heart, and is an umbrella term for a group of syndromes including angina pectoris, acute myocardial infarction and sudden ischaemic cardiac death (Shaper, 1988; Henderson, 1996). There are also other variants: different manifestations of angina, acute myocardial insufficiency and chronic heart failure, for example (Maseri 1995). The term coronary heart disease (CHD) is nowadays used interchangeably with ischaemic heart disease (IHD; Maseri, 1995). The term CHD will be used throughout this thesis, although when referring to disease which may include the peripheral arteries, eg. intermittent claudication, the term cardiovascular disease (CVD) will be used. The underlying pathology of all the CHD syndromes is thought to be coronary atherosclerosis, although it is the myocardial *ischaemia* (insufficient oxygen supply to the cardiac muscle) that

causes the symptoms (Maseri, 1995).

"Atherosclerosis appears to provide the *necessary* background for the vast majority of CHD events, but it may not be *sufficient* cause in itself" (Shaper, 1988, p. 3; emphasis in original).

CHD events do sometimes occur with a background of minimal atherosclerosis, but are relatively rare and are usually associated with other specific phenomena such as severely increased thrombotic tendency, coronary artery embolism or coronary artery spasm (Shaper, 1988). The process of atherosclerosis and the syndromes of angina pectoris, myocardial infarction, sudden ischaemic cardiac death and chronic cardiac failure are described in further detail below.

1.4.1. Atherosclerosis

Atherosclerosis is a variable combination of changes of the lining of the arteries (intima), consisting of an accumulation of lipids, complex carbohydrates, blood and blood products, fibrous tissue and calcium deposits, concentrated at one site in the artery (Julian and Cowan, 1992; Henderson, 1996). These accumulations are known as plaques. Some plaques may be fatty, made up of a large pool of cholesterol and only separated from the lumen (the centre of the artery, through which the blood flows) by a thin fibrous cap. Other plaques are solid, made of smooth muscle cells and connective tissue, and may be calcified (Libby, 1996). The fibrous cap is susceptible to fracture, which allows the dead core of the plaque to ulcerate and attract deposits of fibrin and platelets. Repeated episodes of fracture and the buildup of fats and blood products progressively narrows the artery and may occlude it entirely (Fuster, Fallon and Nemerson,

1996).

Atherosclerosis is thought to begin in childhood with fatty streaks in the arterial wall (Tunstall-Pedoe, 1997; Libby, 1996). The fatty steaks probably progress to fatty plaques which then bulge into the artery, causing obstruction and narrowing of the lumen. The larger plaques grow in complexity, and may crack or rupture (Julian and Cowan, 1992; Henderson, 1996). A thrombus - a clot mainly composed of platelets and fibrin - may form and cause scarring at the site in the artery, or the thrombus or part of the plaque may break off, causing an embolus. A plaque that fissures but is not sufficient to cause occlusion or symptoms is still larger than before, causing greater obstruction and risk of later events. The coronary arteries are particularly vulnerable to atherosclerosis because they are often end-arteries with few collaterals. The narrowing of arteries may be progressive and chronic, causing pain or impaired function in daily activities, or there may be acute pain caused by a blockage, resulting in muscle death or fatal changes in cardiac rhythm (Tunstall-Pedoe, 1997).

1.4.2 Angina pectoris

Angina pectoris is a pain or discomfort in the chest, and sometimes in the jaw, arm or other adjacent areas, caused by a temporary shortage of blood supply to the heart (Julian and Cowan, 1992). The term refers to the symptoms, but the condition is only diagnosed if there is sufficient cause to believe the pain is related to myocardial ischaemia (Henderson, 1996). In most cases at least one of the major coronary arteries has a significant reduction in luminal diameter, and

two or three arteries may be involved. Although the threshold for exercise differs among sufferers, and even varies within individuals, the pain is almost always brought on by physical effort, as first noted by Heberden (1772, in Tunstall-Pedoe, 1997). Given the importance of symptoms in angina pectoris, the diagnosis is often based primarily on the patient's report of symptoms and the description of the onset and nature of the pain (Maseri, 1995). Clinical diagnosis may therefore not be reliable, and to standardize the diagnosis for epidemiological studies, Rose (1962) developed a questionnaire that was later adopted by the World Health Organization. In clinical practice, sometimes an exercise test is carried out to look for characteristic changes on the ECG, and, if the patient is being considered for coronary artery surgery or angioplasty, more invasive procedures such as coronary angiography must be performed to assess the state of the vasculature (Durrington, 1993). However, expensive and risky procedures such as these cannot be used in epidemiological surveys, unless they are being carried out for other reasons. More severe and persistent symptoms of angina indicate a greater risk for an acute CHD event (Tunstall Pedoe, 1997).

1.4.3 Myocardial infarction

A myocardial infarction (MI) is said to have occurred when myocardial tissue dies because of severe and acute interruption of the coronary blood supply (Julian and Cowan, 1992; Henderson, 1996), although its diagnosis is not always clear (WHO MONICA Project, 1994). Its main feature is severe chest pain, similar in location to angina pain, but it is more intense and may radiate widely

across the whole chest, into the jaw and the arms. In most cases the pain lasts for more than 20 minutes, glyceryl trinitrate will not relieve it, and the ECG is abnormal both during and after the infarction. These ECG changes happen in a sequence and therefore the recording of serial ECGs is very important (Tunstall-Pedoe, 1997; Julian and Cowan, 1992).

Enzymes present in the cardiac muscle are released upon necrosis, so their concentration in the serum at first rises, then falls, after the infarct; tests for new enzymes in serum are becoming increasingly sensitive and specific so that even mild infarcts are now recognized (Tunstall Pedoe, 1997). The amount of enzyme released is a rough indicator of the extent of myocardial damage. The initial diagnosis of MI is usually suspected on the basis of the nature, the location and the length of time of the chest pain. Death occurs in about 25% of cases a few minutes after onset of symptoms. Excluding these first deaths, the mortality rate is about 10%: the risk is greatest in the first hours and recedes as time goes on, and the risk of death is much higher in older patients (Julian and Cowan, 1992). Mortality rate for recurrent infarctions is also greater. If early enough, however, treatment with aspirin and thrombolytic drugs improves the survival rate (Tunstall-Pedoe, 1997).

1.4.4 Sudden ischaemic cardiac death (SICD)

Death may occur very suddenly in individuals with coronary atherosclerosis. In many cases, the death occurs without any warning, although in about 50% of cases there may be known angina or previous myocardial

infarction, or the history provided by a relative indicates that there was cardiac pain before the death (Julian and Cowan, 1992). Ventricular fibrillation often appears to be the cause in sudden deaths, but if defibrillation can be carried out promptly, resuscitation may possible (Fitzpatrick et al, 1992; Eisenberg, 1994). However, some deaths recorded as sudden ischaemic cardiac deaths may be misdiagnoses, especially if no further investigation takes place, and therefore accurate statistics for SICD are difficult to obtain (WHO MONICA Project, 1994).

1.4.5 Chronic cardiac failure (CCF)

Chronic cardiac failure is a recent addition to conditions falling under the heading CHD (Tunstall-Pedoe, 1997). For clinicians the presence of conditions such as pulmonary venous or systemic venous congestion indicate CCF. CCF results in inadequate blood supply to the body tissues and organs (Julian and Cowan, 1992). Heart failure may occur if there has been ischaemic damage, but the heart may also fail for other reasons. Diabetes mellitus, for instance, is strongly related to CCF. Heart failure admissions to hospital increase with the number of previous episodes of acute coronary events (Tunstall-Pedoe, 1997).

1.5 CHAPTER SUMMARY

CHD is collection of syndromes, some chronic and some acute, all of which seem to be strongly related to the buildup of atherosclerosis in the coronary arteries. Its great impact on morbidity and mortality has necessitated the search for its causes, in the hope of discovering better methods of treatment or

prevention. Epidemiological studies have identified three major potentially modifiable risk factors for the disease: smoking, serum cholesterol levels and hypertension. Nonmodifiable factors such as age, sex and family history, and other contributing factors such as diet, obesity, alcohol consumption and fibrinogen levels have also been investigated widely. Yet even with the knowledge about these risk factors, 50% of new cases cannot be explained. This incomplete understanding of the causes motivated many researchers to study personality in relation to CHD. Much of this personality research concentrated on the Type A behaviour pattern and hostility, and these studies are reviewed in the following two chapters.

CHAPTER TWO

Type A Behaviour and Coronary Heart Disease

2.1 INTRODUCTION

Research into personality and cardiovascular disease expanded in the 1960s because two physicians, Friedman and Rosenman, began to investigate links between psychological attributes and the risk of developing coronary heart disease (CHD). Initially, they conducted a survey to elicit the opinion of physicians and lay executives on the causes of clinical CHD (Friedman, Rosenman and Carroll, 1958). The consensus, that "chronic trauma" induced by excessive drive, competition, working to deadlines and "economic frustration" were to blame, spurred Friedman and Rosenman (1959) to conduct a preliminary study exploring the relationship between behaviour and the occurrence of CHD . They found a positive association, and this drove them and many others to investigate further the links between behaviour and CHD.

The particular behaviour in question, now known as the Type A behaviour pattern (TABP), was first described thus:

" 1) an intense, sustained drive to achieve self-selected but poorly defined goals; 2) profound inclination and eagerness to compete; 2) persistent drive for recognition and advancement; 4) continuous involvement in multiple and diverse functions constantly subject to time restrictions (deadlines); 5) habitual propensity to accelerate the rate of execution of many physical and mental functions, and 6) extraordinary mental and physical alertness" (Friedman and Rosenman, 1959, p. 1286).

Friedman and Rosenman were following not just their own instincts about the causes of CHD, but also those of earlier physicians such as Sir William Osler

(1910): "It is not the delicate neurotic person who is prone to angina, but the robust, the vigorous in mind and body, the keen and ambitious man, the indicator of whose engines is always at 'full speed ahead.'" It was not until the mid 1960s, however, through the work of Friedman and Rosenman, that other researchers developed an interest in systematically investigating relationships between personality and CHD. The remainder of the chapter outlines the history of Type A research, its main problems and the attempted solutions.

This chapter describes the main measures of Type A behaviour first, followed by a review of Type A/ CHD research from the 1960s. The review is divided into four sections: first, prospective population studies; second, studies of Type A and angiographically documented CHD; third, studies of groups already considered at high risk of CHD; and fourth, studies of the components of Type A.

2.2 MEASUREMENT OF TYPE A BEHAVIOUR

The Type A construct is a complex one, making standard, routine assessment difficult. There are four ways of measuring Type A behaviour that are the most commonly used: The Structured Interview, in which the subject is assessed by the interviewer, and the Jenkins Activity Survey, the Framingham Type A Scale and the Bortner Rating Scale, all three of which are self-report measures.

2.2.1 The Structured Interview

This method was devised by Friedman and Rosenman for use in the

Western Collaborative Group Study (WCGS; Rosenman et al., 1964). In the Structured Interview (SI) participants take part in a 30 minute tape recorded interview, and are assessed afterwards by a review of the tape and by behaviour noted during the interview. Questions address an individual's reactions to working at a slow pace or having to wait in queues, and are deliberately presented to draw out Type A speech patterns and behaviour (Matthews, 1982). For instance, a question with an obvious answer may be asked hesitantly, or the interviewer may express doubt about the accuracy of a reply. Inter-rater agreement rates range from 75%-90% (Rosenman, 1978), and in one study repeated interviews resulted in similar Type A/B decisions in 80% of cases (Rosenman et al., 1964). This interview is generally considered to be the best way of measuring Type A behaviour (Johnston, 1993).

2.2.2 Jenkins Activity Survey

The Jenkins Activity Survey (JAS) is a self-report measure, consisting of multiple-choice questions, precoded for ease and accuracy in keypunching (Jenkins, Rosenman and Zyzanski, 1974). Question content is similar to that in the Structured Interview (Matthews, 1982). There are three separate components scored: 'Speed and impatience', 'Job involvement', and 'Hard driving' (Jenkins, Rosenman and Zyzanski, 1974). Positive scores indicate the Type A direction, and negative scores the Type B direction. Test-retest reliability of the JAS ranged between 63%-73% agreement over four years (Jenkins, 1978), and reached a correlation of 0.79 over eight months (Johnston and Shaper, 1983). This is the

most frequently used measure of Type A behaviour (Matthews, 1982).

2.2.3 Framingham Type A Scale

The Framingham Type A scale is also a self-report measure. It was derived from combinations of items on a 300-item questionnaire administered to 3102 members of the Framingham cohort (Haynes et al., 1978). A panel of experts chose items which in their opinion measured Type A behaviour. The list of items was factor analyzed, and those with poor inter-item or factor loadings were discarded. Items which failed to obtain a correlation coefficient of 0.25 with the whole scale were also dropped. Ten year test-retest data for women showed 57%-80% agreement, although there were no data available for men (Matthews and Haynes, 1986). Haynes et al. (1978) reported that the Framingham Type A scale seemed to reflect the competitiveness, ambition, impatience and high need for achievement assessed by the SI.

2.2.4 Bortner Rating Scale

The Bortner scale has mainly been used in European epidemiologic studies (Bortner, 1969). Subjects are asked to mark a point on a line that reflects their behaviour on the dimension. It is scored by measuring the length of the line from the Type B end to the mark on each of the 14 items and then summing them for an overall score. A higher score indicates more Type A behaviour. The test-retest reliability coefficient was 0.71 between administrations eight months apart (Johnston and Shaper, 1983). Bortner emphasized that because the questionnaire

was self-administered, the verbal elements of Type A were impossible to evaluate, and that only part of the pattern was therefore being measured. Despite his reservations, he concluded that further work with the scale was justified, because of the relative advantage of its easy administration.

2.2.5 Problems

Self-report measures cannot challenge the respondent in the way the SI does, and therefore may be subject to 'social desirability' bias (Matthews, 1982). They are also unable to tap the characteristic speech patterns and aggressive responding that the SI assesses. Each of the measures has variable test-retest reliability coefficients, although they are comparable to reliability coefficients of other psychometric tests (Johnston and Shaper, 1983). However, the correlations between the measures are poor. Both the JAS and Framingham Type A scale agree with SI-assessed Type A/B classification in only 60%-70% of cases (10%-20% above chance levels; Matthews and Haynes, 1986). Correlations between the JAS and Bortner reached 0.71 (Johnston and Shaper, 1983). Haynes, Feinleib and Kannel (1980) reported that the SI and Framingham Type A Scale were concordant in 52%-68% of cases, and that SI-JAS correlations ranged from 0.38 to 0.64. This means that the four measures may not be assessing the same aspects of the Type A pattern, and this makes it very difficult to compare studies.

2.2.6 Section summary

There are difficulties in measuring type A behaviour. Friedman and

Rosenman designed the SI, which emphasizes speech style and reactions over actual question content. Others developed self-report questionnaires, which were easier to administer but which lack the challenging element. A universally used measure was never developed, so the literature contains research using many different Type A measures that do not intercorrelate highly, often making comparisons problematic. The following sections summarise studies using the different measures of Type A behaviour, starting with prospective population studies.

2.3 PROSPECTIVE POPULATION STUDIES

2.3.1 The Western Collaborative Group Study

Friedman and Rosenman's survey, along with the encouraging findings from their preliminary cross-sectional investigation (1959), led them to set up the Western Collaborative Group Study (WCGS). The WCGS was a longitudinal study of 3524 men, aged 39 to 59 years, recruited from 11 Californian companies (Rosenman et al., 1964). At intake in 1960/61, all were medically examined and assigned to a personality category: Type A or its antithesis, Type B. Each of the structured interviews was tape recorded and later played back and categorised.

Rosenman et al. (1964) found that 52% of the men were Type A and 48% were type B. All but 113 (3%) of the men were free of CHD. Over the years of follow-up, Friedman and Rosenman recorded the incidence of CHD, in the form of angina pectoris and fatal and non-fatal myocardial infarction (MI). The category of 'unrecognised MI' included those with ECG evidence of MI, which had been

either 'silent' or clinically unrecognised. After 8.5 years of follow-up, Type A men had experienced twice as much CHD as Type B men (Rosenman et al., 1975). The association was statistically significant in only the 50-59 year age group, but the same pattern held for the 39-49 year age group. Adjustment for risk factors did not affect the relationship between Type A and disease incidence.

Assessing the validity of Rosenman et al.'s (1964) results is difficult because 506 participants (14% of the study group), 282 Type As and 224 Type Bs, were lost to follow-up, with their CHD status unknown. For analysis, all were assumed to be *free of* CHD. Other assumptions should have been tested, such as assuming that those lost to follow up all *had* CHD. A second assumption could have been that all Type As had CHD, and that all Type Bs were free of CHD. Yet another alternative would have been that all Type As were free of CHD, but that all Type Bs did have CHD. Performing these sensitivity analyses would have allowed the robustness of the findings to be tested. As the 'true' status of the study group was unobtainable, extrapolation to the general population was made difficult. However, within the study group, the 2:1 ratio of CHD in Type As to Type Bs was a seminal finding.

After 22 years of follow-up in the WCGS, further analysis was carried out (Ragland and Brand, 1988a). During the first 8.5 year phase, 135 men developed MI or sudden coronary death, 71 silent MI and 51 angina pectoris (without MI): a total of 257 cases. In 1982-83 the vital status of 99% of the remaining 3154 men (excluding the 113 who had been found to have CHD at baseline and the 257 diagnosed with CHD during the first 8.5 year phase) was determined; cause of

death was obtained from the death certificate. Between 1960 and 1983 there were 214 CHD deaths.

Three types of survival analysis were conducted (Ragland and Brand, 1988a). In the first, each risk variable was categorized and the number of CHD deaths per 1000 person years was calculated by category. The second used a multivariate proportional hazards model to estimate the independent contribution of each variable. In the third, the total follow-up period was divided into four intervals, each for which separate multivariate proportional hazards analyses were conducted.

The results of Ragland and Brand's (1988a) univariate analysis showed significant trends of risk for blood pressure, cholesterol, smoking and age but not Type A/B behaviour. The multivariate analyses confirmed the univariate findings: no difference in mortality in Type A/B behaviour, in either of the age groups, 39-49 or 50-59; the age groups were defined by the participants' ages at baseline. The results for separate time intervals showed no effect of Type A/B behaviour, except for one finding in the second interval, in which Type Bs unexpectedly had higher CHD mortality than Type As.

Finally, to try to compare the 22-year results to those obtained after the first 8.5 year period, the 8.5-year data were re-analyzed using proportional hazards model (Ragland and Brand 1988a). They found serum cholesterol, cigarette smoking and age to be more strongly associated with 8.5 year mortality from CHD than with 8.5 year incidence of CHD (which included nonfatal events). These risk factors' 22-year relationship to CHD mortality was even stronger. Type

A/B behaviour showed no relationship with CHD mortality during 8.5 years or 22 years, yet there had been a significant relationship with 8.5 year CHD morbidity.

The interpretation of Ragland and Brand's (1988a) study could be either that any true association between Type A/B and CHD mortality was obscured by an artifact of design or conduct of the study, or that there really was no association. Ragland and Brand discussed bias and other factors in turn, but given all the evidence, decided that baseline Type A/B behaviour was *not* associated with CHD mortality in the WCGS. And, if Type A/B was truly associated with incidence of CHD, its effect was different from other risk factors (eg. cigarette smoking, serum cholesterol, age), which, unlike Type A, have stronger associations with mortality than morbidity. They concluded two things: (1) the results provided strong confirmatory evidence for the long-term importance of traditional risk factors, and (2) that although Type A/B behaviour *is* related to *nonfatal* CHD incidence, it has *no* long-term association with CHD *mortality*.

2.3.2 The Framingham Study

The excitement generated by the first phase of the WCGS incited researchers of another large prospective study to take an interest in the TABP. This was the Framingham Study, a longitudinal investigation of CHD and its risk factors in 5127 men and women in Framingham, Massachusetts (Haynes, Levine and Scotch et al., 1978). From 1965 to 1967, behaviour and life stress were assessed in the 3102 surviving members of the cohort, aged 45-77 years, who had been participants since the study's inception in 1949. The study team developed

their own, quite broad, behavioural questionnaire to allow for the exploration of many behavioural hypotheses. Part of the questionnaire was the 'Framingham Type A Scale'. The questionnaires were pre-tested on 670 participants, and, as several changes were made subsequently, the pre-tests were not included in the analyses. Two-hundred thirteen more were excluded because of incomplete questionnaires, and a further 397 were dropped from analysis because they were thought to have been subjected to interviewer bias. The final sample comprised 1822 men and women: just over half those originally eligible.

Cross-sectionally, the prevalence of CHD was significantly higher in Framingham Type A Scale-assessed Type A men and women than in Type B men and women (Haynes, Feinleib, Levine et al., 1978). In men, Type A behaviour was also specifically associated with MI. In multivariate analyses, the Framingham Type A scale remained significantly positively associated with the prevalence of CHD, but the relationship was attenuated by the addition of other emotional measures into the models. Age, in both men and women, aging worries in men, and emotional lability in women all were more strongly related to CHD than the Framingham Type A scale, in the cross-sectional analyses.

Results after a further eight years of follow-up suggested that women aged 45-64 years who developed CHD scored higher on Framingham Type A behaviour, suppressed hostility, tension and anxiety (Haynes, Feinleib and Kannel, 1980). Type A women developed twice as much CHD and three times the angina as Type B women. Framingham Type A Scale, work overload, suppressed hostility and frequent job promotions put men aged 55-64 years at increased risk

of CHD. Type A men aged 45-64 years showed a twofold risk of angina, MI and total CHD compared to Type Bs, independent of standard risk factors. In men, the association was found only in white-collar workers.

The excluded group of 397 participants, possibly subjected to interviewer bias, poses problems for interpretation of the results. Bias was suspected because of a threefold increase in 'socially desirable' responding in the presence of one of the interviewers (Haynes, Levine, Scotch et al., 1980). It was thought problematic that one interviewer could incite more socially desirable responses: it perhaps indicated that true responses were suppressed in this group and incorrect classification of subjects therefore was made. Haynes and colleagues did not indicate which items might have been affected by the 'social desirability' bias, so we are unable to judge if Type A responses would have been affected by this. However, if they were, those who were Type A presumably might have tried to hide their behaviour, thus *attenuating* the association of Type A and CHD in those with CHD. Thus the reported results may have reflected too large an effect of Type A on disease. It was also possible that the one group really was different from the others for reasons nothing to do with the interviewer. Consequently, the questionnaires from the 397 should have been either analyzed with the rest, or the analyses performed *with **and** without* the 397. This would have allowed the above alternatives to be explored and fully discussed. The scope of the study deliberately was broad, and therefore, results of the group of 397 would have been both interesting and relevant. Again, as with the WCGS, the results are difficult to extrapolate because it is unclear whether the results accurately reflected the whole

study population. However, because the study supported the previous WCGS findings, further incentive to investigate the Type A-CHD association was created.

2.3.3 The Honolulu Heart Program

A third large cohort was the Honolulu Heart Program: 2,187 men of Japanese descent were followed, again for eight years (Cohen and Reed, 1985). The researchers administered the Jenkins Activity Survey, a questionnaire designed to measure the TABP (Jenkins, Zyzanski and Rosenman, 1971). Baseline prevalence of total CHD was independently associated with the TABP, but the incidence of CHD over the next eight years showed no association with TABP. And, in 48 men who underwent autopsy, Type A was not correlated with evidence of MI or with severity of atherosclerosis. The study population did have a low incidence of both TABP and CHD, which would have reduced the statistical power to detect an association.

2.3.4 British Regional Heart Study

One large prospective British study began in the late 1970s (Johnston, Cook and Shaper, 1987). The participants were men between the ages of 40 and 59 and they were randomly selected from the age-sex registers of one group general practice in each of 24 towns. In 19 of these towns, the 6177 men who came for screening were asked to complete the Bortner questionnaire while waiting to be examined and assessed for baseline CHD. The questionnaire was

fully completed by 5936 men. They were followed up for between five and seven and a half years. The prevalence of baseline CHD, as assessed by questionnaire and ECG, was positively related to the Bortner score, though the relation to ECG evidence was weak (and not significant at the 5% level). After adjustment for demographic and standard risk factors, however, the trends across quintiles of the Bortner score were statistically significant for both questionnaire and ECG evidence. A man's recall of a doctor's diagnosis of CHD did not relate at all to the Bortner score.

Prospectively, the attack rates of new major CHD events, both fatal and non fatal, were higher in *Type B* men, although the relationship was not significant at the 5% level (Johnston, Cook and Shaper, 1987). The association was attenuated by adjustment for other risk factors. Hence, Type A behaviour, as measured by the Bortner questionnaire, did not relate to new major CHD events in a British, male, middle aged population. The authors suggested that perhaps only some aspects of the behaviour pattern, not tapped by the Bortner questionnaire, might be implicated in the risk of CHD.

2.3.5 Problems

Population based, longitudinal studies are invaluable when studying behaviour-disease relationships. However, they are costly and may be biased both through recruitment procedures, eg. only sampling certain sections of the community, and through losses to follow-up. The above studies are difficult to compare because they each used different type A measures; the same results using

the same measures would have meant a much stronger case for association.

2.3.6 Section summary

Two of the four large population-based studies reported longitudinal associations between Type A behaviour and CHD incidence. Each of the them used different measures of Type A behaviour, with only one, the structured interview of the WCGS, taking into account actual behaviour alongside answer content. The WCGS researchers, however, when assessing the long-term prognosis of Type A and B men, found no difference in CHD mortality between the two groups, although traditional risk factors showed a very strong relationship with mortality. The British Regional Heart study found that prevalence of CHD was associated with Type A behaviour, but did not find that Type A was predictive of new CHD events. However, before the long term follow-up results from the WCGS and other studies were available, researchers began to think that closer attention to the mechanisms linking Type A and CHD was necessary. They wanted to pinpoint how, and on which aspects of CHD, Type A was exerting its effect. For example, did Type A contribute to atherosclerosis only or to acute MI specifically? Therefore, mechanisms of the putative association between TAB and CHD were examined.

2.4 STUDIES OF TYPE A BEHAVIOUR AND ANGIOGRAPHICALLY DOCUMENTED CORONARY HEART DISEASE

2.4.1 The studies

The first of these studies was conducted in about 1975. The relatively new

technique of coronary angiography was used to measure CHD by assessing the extent of atherosclerosis in the coronary arteries. Zyzanski et al. (1976) administered the JAS to 94 men undergoing coronary angiography. There were statistically significant differences between men with more obstructed coronary arteries and men with fewer diseased vessels. Those with 2-4 obstructed vessels had higher Type A scores than those with 0-1 obstructed vessels.

Dimsdale et al. (1978) examined 109 patients (99 men, 10 women) undergoing coronary angiography. The participants were selected from a group awaiting cardiac catheterization at Massachusetts General Hospital. All patients had clinical evidence of CHD (eg. abnormal ECG) or chest pain of uncertain origin and were between 18 and 70 years of age. Before cardiac catheterization, patients completed the Jenkins Activity Survey Form B. There was no relationship between the number of diseased coronary vessels and JAS score, nor in comparisons of degree of severity in the vessels.

Another team of researchers tried to establish if prior arterial damage was accelerated by Type A behaviour, or if Type A behaviour induced atherosclerosis formation in the absence of previous damage (Blumenthal et al, 1978). It was the first coronary angiographic study to use the SI to assess Type A behaviour, although the JAS was also administered. Blumenthal and colleagues studied 80 men and 62 women referred for coronary angiography at Duke University Medical Centre. The participants were given the SI after catheterization, but before the results of the angiography were known. The SI scores *were* related to angiographically documented atherosclerosis, with a trend of a greater proportion

of Type As from mild, to moderate, to severe atherosclerosis. The authors suggested that SI-assessed Type A behaviour was therefore influencing risk over an extended period of time, and that modification of the behaviour should be attempted. No differences were found using the JAS.

A study of similar size, of 124 men and 23 women, was carried out at Columbia Presbyterian Medical Centre (Frank et al., 1978); Type A was assessed by the SI. Over half the group were found to be type As, and there was less severe disease found in Type Bs. Type Bs, in fact, were frequently found to have no significant disease. Type A behaviour was as strong an indicator as any other risk factor except for cholesterol, and the association persisted after adjustment for age, sex, blood pressure, smoking and cholesterol.

Unusually in angiographic studies, Williams et al. (1988) examined a large number of patients: data on 2289 patients undergoing diagnostic coronary angiography at Duke University Medical Centre were collected between 1974 and 1980. Type A behaviour was assessed both by the SI and the JAS, and complete data were available for 679 women and 1610 men. CHD severity was assessed by coronary angiography and extent of atherosclerosis was scored from 0 (no disease) to 5 (most diseased). The *number* of vessels significantly occluded was also recorded. The data were analyzed in multivariate models, with other risk factors as covariates. The goal of analysis was to identify characteristics that might have been involved in atherogenesis, rather than characteristics diagnostic simply of existing CHD.

The main finding was an age-dependent association between SI-assessed

Type A behaviour and angiographically documented CHD (Williams et al., 1988). In both men and women under 45 years, Type As had more severe CHD than Type Bs. This

"suggests, within the limitations of a cross-sectional study, that Type A behaviour may be primarily involved in the *premature* development of atherosclerosis" (Williams et al., 1988, p. 148).

Hyperlipidaemia and smoking also showed a weaker association with CHD severity with increasing age in this sample. The authors explained that it might be a survival effect: those Type A's who remained alive and healthy enough to be part of the study perhaps were less susceptible to other risk factors in the first place, and hardier than the Type Bs. The same effect, they pointed out, could explain the diminution of the Type A effect in the WCGS as the sample aged. It could also explain negative results in other angiographic studies that didn't test for age interactions, although inadequate statistical power could have been the problem in smaller studies (Williams et al., 1988). Their final recommendation was to take age effects into account, and to concentrate on aspects of Type A, such as anger and hostility, that might be responsible for the effect.

In a follow-up study of 1467 patients with angiographically documented CHD, Barefoot et al. (1989) discovered that Type A behaviour (assessed by the SI) did <u>not</u> predict the incidence of nonfatal MI or total coronary events. Indeed, in those considered to have poor prognoses owing to the extent of their CHD, Type As had <u>better</u> survival than Type Bs. In those with better prognoses, Type As had neither better nor worse survival than Type Bs. CHD severity alone, however, unlike Type A, was an excellent predictor of survival. Even when

CHD severity was taken into account, Type A behaviour could not distinguish between those who would have an overt CHD event (eg. MI) from those who remained free of such an event. Barefoot and colleagues did point out that because the patients were already diagnosed to have some degree of CHD, that the risk factors for CHD events in this study population might have been different from those in a randomly selected, healthier population (see also section 2.5.2).

2.4.2 Problems

There are problems with many of the above study designs. Many were retrospective, because the cost and risks of coronary angiography precludes the selection of a population sample to undergo the procedure without good reason. Therefore, assessment of the TABP cannot be made until referral for angiography has been made, and often is not assessed until after angiography has been carried out. Interpretation of findings is, then, always hampered by questions of causation: that behaviour may have been changed through illness or even through referral. The second major problem with these studies is that people are referred for angiography for suspected coronary disease, which means the sample may be biased. Chest pain is sometimes experienced in the absence of CHD. For instance, neuroticism has been linked to symptom reports of chest pain in the absence of CHD (Stone and Costa, 1990). It may be that people with non-CHD chest pain are being referred for angiography because they are willing to report their symptoms. Because subjects are often recruited for study before undergoing angiography, both those with and without true disease become part of the study

group. This reduces the numbers of subjects in angiography studies who have true disease, and therefore reduces statistical power to detect disease-risk factor associations. The third problem is that the studies allow for only one possible mechanism, atherosclerosis, linking Type A and CHD. Other mechanisms include thrombosis and plaque rupture, which angiography studies cannot address.

2.4.3 Section summary

Taken together the studies are equivocal. Assessment of the extent of coronary disease and Type A behaviour at first seemed it would be helpful in showing that the formation of atheroma was the mechanism linking Type A and CHD. However, the problems inherent in the study designs and the inconsistencies between self-report versus interview measurements of Type A made this very difficult.

Others took a different approach by studying populations already at high risk for CHD according to their risk factor or disease status. Their Type A behaviour was assessed to see if it could improve prediction of who would have a CHD event. These studies are discussed in the next section.

2.5 STUDIES IN HIGH RISK GROUPS

2.5.1 The studies

The Multiple Risk Factor Intervention Trial (MRFIT; Shekelle, Hulley, Neaton et al., 1985) was a very influential study, owing to the number of participants, the information obtained from them, its design and the length of

follow-up. It was a primary prevention trial designed to test the effects of a multifactor intervention on risk of death in 35-57 year old men who had no clinical evidence of CHD, but whose risk factor levels placed them in the upper 10-15% of the Framingham risk score. The men were randomized to special intervention or usual care: 12,866 were allocated to either group over three years. At entry, the SI and JAS were both administered; the SI at a limited number of participating clinics because of the cost involved.

The MRFIT interviews were tape recorded and sent to the coordination centre and assessed independently of the first interviewer's assessment (Shekelle, Hulley, Neaton et al., 1985). Discrepancies were referred to Dr Rosenman. The JAS was given to all the participants. None of the Type A information was held at the clinics or available to the clinic staff. CHD events and deaths were ascertained during the 7.1 year follow-up period by the clinic staff. The hypothesis, given that the Framingham study (Haynes, Feinleib and Kannel, 1980) found a Type A/CHD association only in white collar men, was that professional Type A men would have a greater incidence of first major coronary events than professional Type B men.

In neither the usual care nor treatment groups was Type A associated with increased incidence of first major coronary events (Shekelle, Hulley, Neaton et al., 1985). Predictors of events were age, blood pressure, cholesterol and cigarette smoking. Results were unchanged after excluding men taking beta-blockers. JAS scores were also not associated with incidence of first major coronary events.

Shekelle, Hulley, Neaton and colleagues (1985) discussed several possible

explanations for the findings. They dismissed most of them, but did cite imprecise measurement of Type A as a potential serious problem. They also pointed out that the MRFIT population was a highly selected sample of men, all of whom had agreed to participate in a clinical trial involving changes in diet, smoking and blood pressure treatment. The extrapolation of findings would therefore be very difficult. Therefore, the authors expressed reservations about the robustness of Type A as a risk factor.

A smaller study of recurrent CHD was the Multicenter Post-infarction Project (Case et al, 1985). It was a prospective, observational study of 516 patients who were assessed on the JAS within 2 weeks of an acute MI. The patients were followed-up for one to three years. There was no relationship between JAS score and total mortality, cardiac mortality, time to death for non survivors, or duration of stay in the coronary care unit. Physiologic factors were found to be the only significant prognostic factors.

The JAS was again the instrument used to measure Type A behaviour in a subset of the 4524 participants in the Aspirin Myocardial Infarction Study (AMIS; Shekelle, Gale and Norusis 1985). The study was carried out at 30 centres in the USA, and was a trial of aspirin in persons recovering from an MI. Subjects were monitored for fatal and nonfatal MI over three years. At 18 of the centres, participants completed the JAS. The hypothesis was that type A score would be positively related to risk of recurrent major coronary events such as definite nonfatal MI and coronary death.

JAS scores were completed for 2314 of 2698 AMIS participants who had

been invited to fill it in (Shekelle, Gale, and Norusis 1985). Data from the 18 centres were pooled, and so were data from both treatment groups. T-tests were carried out to evaluate differences in mean Type A score between those who had a recurrent event and those who did not. A proportional hazards regression model was used to adjust for potentially confounding variables. Type A score was forced into the equation and the rest of the variables were selected stepwise.

Level of type A score was not significantly related to risk of recurrent major coronary events in the 244 women, the 2070 men, or in the subgroup of men in professional, technical or managerial positions (Shekelle, Gale, and Norusis, 1985). The highest risks, in fact, were seen among persons with the lowest Type A scores. Results could have been biased by the 14% who refused or were unable to participate, and volunteers could have systematically differed from the general population. However, the negative results agreed both with MRFIT (Shekelle, Hulley, Neaton et al., 1985) and the Multicenter Post-infarction project (Case et al., 1985).

Shekelle, Gale and Norusis (1985) therefore concluded

> "that an association between the *JAS Type A score* and risk of recurrent coronary disease has not been adequately demonstrated. Using the JAS under the assumption that such an association does exist is not justified by the evidence" (p. 224).

In addition, because the JAS scores reflect competitiveness, achievement-focus and preference for a rapid pace of life, these traits appear not to be associated with risk of *recurrent* coronary events.

Ragland and Brand (1988b) conducted further analysis in the WCGS to test the association between Type A score and recurrent CHD. In the initial 8.5 year

study, conducted between 1960 and 1969, 257 men had developed CHD. One-hundred thirty-five had had symptomatic MI, of whom 26 died within 24 hours of symptom onset, 71 had a silent MI discovered by ECG at examination, and 51 had classic angina pectoris. The vital status of all but two of these men was ascertained in 1983, by which time there had been 91 deaths from CHD and 37 deaths from other causes. The 91 CHD deaths were divided into two groups: those who had died in under 24 hours, and those who survived longer than 24 hours.

Of those who died in less than 24 hours after the CHD event, the mortality rate for Type A and Type B subjects was nearly identical (Ragland and Brand, 1988). The analysis of long-term mortality in those who had survived longer than 24 hours showed that Type A subjects died of recurrent CHD at a rate 0.60 times that of Type B subjects. The association was strongest in those whose first coronary event was a *symptomatic* myocardial infarction. Ragland and Brand also reported other studies that showing an inverse association between Type A behaviour and secondary CHD events (Dimsdale et al., 1981; Shekelle, Gale and Norusis, 1985). In the first phase of the WCGS, however, a positive association between behaviour type and recurrent infarction had been found (Jenkins, Zyzanski and Rosenman, 1976).

2.5.2 Problems

The first difficulty with studies in high-risk groups is that the population is highly selected before the study begins. Their willingness to participate in

intensive study may set them apart from the general population. Analyses were often carried out on subsets of patients in larger studies, possibly further affecting representativeness. Also, the high risk group may be especially hardy, as other high risk individuals, all or many of whom may have been Type A, already could have died from CHD, thus leaving a non-representative group available for study (Miller et al., 1991). A further problem is the underlying assumption that Type A would operate in the same way in high-risk as in ordinary-risk persons, and in the same way after an MI as it did before. In addition, as with other investigations, various instruments were used to assess Type A, so the studies' results were not strictly comparable.

The final problem is that restricting the range of severity of CHD (ie. selecting those already at high risk) can attenuate observed correlations between Type A and CHD (Miller, 1996). This is because in studies of subjects with high physical risk factor levels, those without *overt* CHD will still have higher *subclinical* levels of CHD than truly healthy participants (Miller et al., 1991). This causes the 'disease-free' and 'diseased' groups in high risk studies to be more homogenous than the equivalent two groups in surveys of the healthy population. When comparing two such similar groups in high risk studies, it is perhaps is not surprising that no additional risk is observed for Type A individuals.

2.5.3 Section summary

Similar traditional risk factors are thought to be at work for secondary CHD events as for primary, yet for Type A this does not appear to be the case. Studies

in high risk groups generally showed that Type A is not a risk factor in people with elevated traditional risk factors or with a history of MI. Ragland and Brand's (1988b) study suggested the need for further research to discover whether Type A behaviour is actually *protective* of recurrent CHD mortality.

2.6 STUDIES OF THE COMPONENTS OF TYPE A BEHAVIOUR

As is clear from the original definition of Type A behaviour given at the beginning of the chapter, Type A behaviour is a mix of components, involving competitiveness, time pressure and aggression (Johnston, 1993). Perhaps inconsistent study results should have been expected, as it may be only one, or even a mixture of some of the components that is the true coronary prone behaviour. Because of this, studies emerged that examined the components of Type A behaviour separately.

2.6.1 The studies

As early as 1977, further analysis of Type A was being carried out in the WCGS. Matthews et al. (1977) attempted to discover a subset of factors in the SI that were related to CHD. Of the five primary factors recovered in factor analysis (competitive drive, past achievements, impatience, non-job achievement and speed), only two, competitive drive and impatience, were prospectively associated with coronary disease.

The analysis was performed on a sample of 186 men selected from the total WCGS population of 3524 (Matthews et al., 1977). There were 62 cases, each

with two matched controls. Type A/B classification had been made by SI in 1960-61. The interview comprised 44 items, and these were intercorrelated and factor analyzed. Competitive drive was composed of items pertaining to explosive voice, potential for hostility (as elicited by the challenge in the SI), and vigorous answers. The impatience element was almost solely reflected in the 'irritation at waiting in lines' item. The mean level of competitive drive in the CHD group was 1.90, and -1.23 for controls; on impatience the CHD group scored 2.44; the control group -1.26 (negative scores indicate the less type A direction). Matthews and colleagues concluded that the Type A pattern was possibly a response to the environment, in which Type As feel threatened, and therefore attempt to control.

Analysis of Type A components was also carried out in studies using the outcome of angiographically documented CAD. Dembroski et al. (1985) scored components of the SI to see which of the elements was associated with coronary disease severity. The patients in their study were a subgroup of 131 selected from the 2289 in Williams and colleagues' angiographic study at Duke University (Williams et al., 1988). The sample was chosen specifically to have either very minimal or severe coronary disease, to increase statistical power. Only potential for hostility and anger-in (an item recovered by Dembroski et al. in factor analysis) were significantly positively associated with disease severity, including angina and MIs. The two factors interacted: Potential for hostility was associated with disease only if patients were also high on anger-in. Although Potential for hostility was associated with ratings of Global type A, anger-in was independent

of Global Type A measures. As other factors in global Type A also were unrelated to disease endpoints, the authors' suggested that

"hostility, in conjunction with acquired coping mechanisms such as anger suppression (anger-in), may lie at the foundation of coronary-prone behavior" (Dembroski et al., 1985, p. 231).

In 1988 results from a larger subsample of the WCGS were published (Hecker et al., 1988). From the WCGS group, 250 CHD cases and 500 matched controls were selected, and the 8.5 year relationship between 12 facets of the Type A pattern and the incidence of CHD were studied. The original SI tapes were re-analyzed using coding rules developed for the 12 new factors. In multivariate analysis, when all 12 components were entered into the model, only hostility remained a significant risk factor (RR 1.93, $p<0.001$), and remained so when further adjusted for standard risk factors. The authors concluded that Type A is a mixture of benign and coronary-prone components, with the most important risk component being hostility.

The MRFIT trial was not to be left out of re-analysis of their data using components of Type A. Dembroski et al. (1989) broke down Type A into 8 facets: voice stylistics, anger-in, total potential for hostility, hostile content, intensity of hostility and stylistic hostility. A case-control study was performed on 192 cases and 384 matched controls. Based on previous findings, they hypothesized that total potential for hostility, dichotomized into high and low categories, would predict CHD incidence. It did, even after adjustment for standard CHD risk factors (RR 1.5, $p=0.032$). Neither Global type A nor speech style was related to CHD incidence. They also found an age interaction effect:

significant effects for potential for hostility and stylistic hostility were observed only in younger (< 47 years) participants. They too concluded that future work should focus on the narrower components of the TABP, in particular hostility.

The last paper discussed in this section was carried out in Finland (Julkunen et al., 1993). The role of the TABP and its component parts was examined prospectively in a study of first-year prognosis after MI. Patients were under 65 years and had suffered a recent MI, and 92 of 123 approached in hospital agreed to participate and provided complete data. Type A behaviour was measured by the JAS and its three components of job involvement, hard-driving competitiveness and speed and impatience. Achievement striving and impatience/irritability were also derived from the JAS responses, and other behavioural measures were administered. Although anger and irritability were associated with first year complications, they had been assessed using measures other than the JAS. Multivariate analyses were not carried out, and because of small numbers, cardiac deaths were not distinguished from non-fatal coronary events. Therefore, although the results of this study are in broad agreement with those previously discussed, caution is needed because of the differences in assessment of Type A and its facets, and methodological problems.

A review of research into Type A components was published by Dembroski and Costa (1987). They discussed the equivocal findings of research into Global Type A and the emerging evidence concerning separate components of the Type A pattern. They concluded that component scoring to find toxic components of Type A was a 'profitable research strategy' (p. 211), and noted that understanding the

specificity of coronary behaviours would allow research into risk of CHD to be much more efficient.

2.6.2 Problems

The main difficulty with the above studies is that post hoc analyses were carried out. The probability of finding a spurious association (Type I error) is therefore increased. Even in longitudinal studies there were so many elements to analyze that Type I error was likely. In some of the studies components were derived only because they related to CHD (eg. Matthews et al., 1977; Dembroski et al., 1985; Hecker et al., 1988); the items could have been specific to only one study population. These complications meant that it was difficult to apply the findings to a wider population.

2.6.3 Section summary

Despite obstacles created by study designs and analysis, discussed above, the investigation of the components of Type A behaviour was very useful. The findings showed that hostility, for instance, was an important element of Type A behaviour consistently related to the incidence of CHD. Dembroski and Costa (1987) were supportive of component scoring to find toxic elements of Type A, which would help clear the confusion of the diverse results of Type A/CHD studies. Importantly, these studies showed that prospective study of individual elements of Type A would probably be worthwhile.

2.7 REVIEW ARTICLES

2.7.1 The reviews

During the long time span of Type A/CHD research, intermittent reviews were written which helped consolidate findings and indicated useful directions for continuing research. The first of these was a report by the Review Panel on Coronary-prone behaviour and coronary heart disease, commissioned by the national Heart, Lung and Blood Institute (NHLBI) in the USA (Review Panel on Coronary Prone Behaviour and Coronary Heart Disease, 1981). They recognized the need for a comprehensive, impartial review of the data, and hoped to achieve it by holding a Forum on Coronary Prone Behaviour in 1977, and charging a panel of experts with the task of reviewing the vast literature on the topic. There were five panels each with their own remit:

> "(1) the association of behaviour and CHD; (2) the assessment of coronary prone behaviour; (3) the physiologic mechanisms underlying this presumed relation; (4) cultural and developmental patterns associated with the behaviour pattern; and (5) intervention strategies" (p. 1200).

In short journal papers reporting Type A/CHD results, the conclusion of the Review Panel (1981) was often recorded only as the following:

> "The review panel accepts the available body of scientific evidence as demonstrating that type A behaviour....is associated with an increased risk of clinically apparent CHD....This risk is greater than that imposed by age, elevated values of systolic blood pressure and serum cholesterol, and smoking..."(p.1200).

However, in addressing the first remit and having accepted the *association*, the panel stated their serious reservations concerning the understanding and assessment of Type A behaviour. These reservations, they said, limited the ability to make general conclusions about the *implications* of Type A for

cardiovascular disease. They strongly recommended improved Type A assessment techniques and investigation of mechanisms by which Type A affected disease, and decided that 'attempts to remedy the deficiency warrant high priority' (p. 1202).

Regarding the second remit, the Review Panel (1981) outlined the problems with Type A measurement, both in terms of the JAS/SI relationship and the predictive validity of these measures for CHD. Ideally they endorsed a single, brief, self-report scale that would be developed from existing measures, but would improve on them. Addressing the third remit, they expressed approval of the existing studies examining physiologic mechanisms, for example studies of arterial pressure responses. However, they also suggested the inclusion of cardiovascular dynamic measurements alongside personality characteristic measures, and emphasized the need for careful examination of all stages of CHD, to differentiate which stages were related to type A behaviour. Their advice concerning the fourth remit was to develop a more explicit definition of Type A. They recommended detailed study of its applicability to defined subsets of the population such as women, classes other than white, male middle classes, and different age groups. Finally, in looking at the fifth remit, intervention strategies, they advocated a concentration on the feasibility of behaviour change *before* looking for any change in CHD risk from the interventions. The caveats imposed here were that scientific rigor be maintained and that prior or simultaneous development of reliable, stable and reproducible instruments for Type A measurement take place.

Therefore, the support the Review Panel (1981) gave to Type A research was heavily qualified by calls for improved measurements, greater attention to physiologic mechanisms, cultural differences and circumspect intervention trials. Only when these areas were further explored did they think informed comments could be made regarding the implications of Type A behaviour on cardiovascular disease.

The second review was written at approximately the same time. Karen Matthews (1982) drew together the literature because she was examining Type A behaviour from a psychological perspective. Unlike the Review Panel, she began from the premise that the TABP was a firmly established risk factor for coronary heart disease, if poorly conceptually understood. Her paper was important because it reviewed, in depth, the Type A measurements and the psychological correlates of the measures. She discussed the contrast between SI-assessed TAB and self-reported measures, but was confident that all of them related to CHD and assessed at least some aspects of TAB. However, she stressed the necessity to measure comprehensively the multidimensional nature of Type A: that is, to measure the many components separately to find which was most salient for a particular person. She also encouraged use of several different measures because they had such little overlap. Additionally, she thought that Type A was not a continuum, but a typology, and advised that it should be analyzed as such. She pointed out, too, that other measures of life situation should be taken into account, as Type A is meant to be a 'response style' and not a trait. Reliability would also be enhanced if Type A could be measured during daily activities and not just in the

laboratory. Matthews (1982) hoped that psychologists would advance the understanding of Type A by taking account of previous shortfalls in conceptualization and measurement. In doing so, the behaviour could be better understood and would be much more useful in research.

Matthews and Haynes (1986) set out to review and evaluate accumulated data regarding Type A behaviour an CHD, particularly focusing on papers published after 1978, namely, after the meeting date of the Review Panel. In fact, they opened their paper by quoting the passage from the Review Panel that appeared to give unequivocal support to the TAB/CHD associations (see above). Because studies had been carried out since 1978 in slightly more diverse populations, Matthews and Haynes re-evaluated the evidence that, prior to 1978, they thought the review panel saw as uniformly supportive.

Matthews and Haynes' (1986) review was extremely comprehensive, touching on measurements, outlining and summarising a large number of studies, and also evaluating the Type A/CHD relationship according to Hill's (1965) nine epidemiologic criteria. They came to the conclusion that type A behaviour met some, but not all the epidemiologic criteria for causation:

"we suggest that Type A behaviour is associated with risk for coronary heart disease in initially healthy men, and that this association may be causal" (p. 955).

Their conclusions, in fact, echo those of the Review Panel, especially in recommending research in groups such as women, ethnic minorities and others, and in stressing the need for innovative adaptations of the existing measures of Type A. They re-emphasized the necessity of measuring individual aspects of Type A behaviour, and also thought that further investigation of the

pathophysiologic mechanisms was strongly warranted. Research in the years between 1978 and 1986, then, had shed no further light on the problems originally brought up by the Review Panel.

The next review update was in 1987, and it was a direct follow-on from the Review Panel's 1981 report (Costa et al., 1987). Its conclusions were remarkably similar: they pertained to studying more diverse groups, different CHD end points and components of Type A. Two recommendations, however, were new: (1) to maximize objectivity and reduce subjectivity in the SI method, and to make the measure exportable. This could include designing non-interview based measurements able to tap overt behavioural patterns; and (2) that particularly in angiography studies, factors influencing participation should be addressed. For example, since neuroticism is associated with both participation in studies and prevalence of symptoms, its potentially confounding effects should be considered. Broadly, however, the same areas of Type A research were of concern six years after the first review, although new and encouraging research was taking place.

Booth-Kewley and Friedman (1987) took a different approach to reviewing the research: meta-analysis, or a quantitative review. Meta-analysis is a statistical technique that allows pooling of results across several studies, helping to summarize and understand the wealth of data. Main problems with the technique include non-uniform outcomes of studies and the variability in study designs, quality and data. However, meta-analyses are meant to complement, not replace, qualitative reviews (Booth-Kewley and Friedman, 1987).

Booth Kewley and Friedman (1987) came to several conclusions: (1) Type

A behaviour is reliably, if modestly, related to CHD; (2) the SI is a much better predictor of CHD than the JAS; (3) the hard-driving, competitive and hostile elements of Type A are the only components to be related to CHD; (4) Effect sizes in prospective studies were much smaller than in cross-sectional studies; (5) other personality factors such as depression, neuroticism etc. may be both related to type A and also associated independently with CHD; and (6) for unclear reasons the strength of Type A relationships to CHD has decreased over time. Booth-Kewley and Friedman therefore recommended that the measurement of coronary-prone behaviour be broadened to include, for example, depression; but narrowed to eliminate job involvement and other elements which had been shown to be unimportant.

In 1988 Karen Matthews published an alternative to the Booth-Kewley and Friedman meta-analysis. She used different decision rules in choosing the studies for meta-analysis, segregated high risk from population studies and by Type A measure, and weighted the studies differently. With the new weighting, the type A-CHD association appeared only when type A was measured by the SI in population studies, not when measured by the JAS or in high-risk studies. Matthews' most important conclusion was that Type A was a reliable predictor of initial CHD events in population based studies, meaning that it might influence acute precipitating factors, such as coronary thrombosis, as well as possibly contributing to background atherosclerosis.

Miller et al., in their 1991 review, focused on the fact that the strength of the Type A/CHD relationship appeared to decrease from the first studies to the latest

studies. They tried to include all studies published before 1989 that had attempted for find a statistical relationship between TAB and CHD. They split the studies into one of four disease outcome groups: CHD, non fatal MI, coronary artery disease and fatal MI, and further classified them by Type A measure, study design and whether the study was 'early' (prior to 1978) or 'recent' (after 1978).

Miller and colleagues (1991) did indeed find a trend toward null findings, as Booth Kewley and Friedman (1987) had suspected. Studies published before 1978 were significantly more likely to have reported positive findings using both the SI and self-report measures. After 1978, studies were four times more likely to report null findings if assessment of Type A was made by the SI, and eight times as likely to have null findings if Type A was assessed by self-report. The analysis showed that three features of a study were associated with null findings: high risk design, self-report measures, and use of fatal MI as an outcome. All three of these features were more prevalent in later studies. High-risk studies (such as MRFIT; Shekelle et al., 1985) in particular showed attenuated or null findings, some reasons for which were discussed above (section 2.5.2). Self-report measures were favoured in more recent studies, too, which, as they had small correlations with CHD, led to increased null findings. Finally, null findings were almost always recorded when fatal MI was used as an outcome, which could reflect a hardiness of older Type As with CHD, who have survived long enough to be studied, unlike younger Type As, who may already have died from CHD. Alternatively, reliability of information on death certificates may have affected study outcomes.

The recommendations made by Miller and colleagues (1991) were to exercise caution in the interpretation of studies, paying particular attention to study design and methods; to carry out more healthy population studies assessing Type A behaviour by the SI; to try to develop continuous as opposed to dichotomous measures of Type A, which would give greater power to assess the strength of the relationship between Type A and CHD; to clarify the association between Type A and other risk factors; and to improve future meta-analyses by paying greater attention to the weighting of studies and to the problems particular to high risk or other study designs. With these cautions in mind, they hoped that future research would be valuable in determining the existence and strength of the Type A relationship to all types of CHD.

Johnston reviewed the expanding literature again in 1993. He summarized the main studies, which as we have seen had been quite mixed, and which were unable to show reliable Type A/CHD associations. He also reviewed studies of Type A components. He explained that

"the current consensus view is probably that some aspect of *hostility* (emphasis added) relates to CAD [coronary artery disease]...These findings should be regarded as suggestive rather than conclusive and we await clear findings from studies specifically designed to investigate the hostility hypothesis" (p. 407).

2.7.2 Problems

Reviews are helpful in pointing out areas of weakness and in suggesting ideas for future research. However, the conclusions can vary depending on the studies chosen for review, particularly in meta-analysis, where numerical results are combined. In meta-analyses, study weighting can make a big difference to the

overall result. These problems can nevertheless be diminished by careful reading and comparison of review papers.

2.7.3 Section summary

There were numerous reviews of the Type A literature over thirty years of research. As with the individual studies themselves, earlier reviews were more supportive of the status of Type A as a risk factor than later ones, which may have reflected the study designs and measures used in later investigations. On the whole, they seemed to highlight the same difficulties within the field and several made recommendations for continuing research. The most recent reviews supported the study of related personality factors, especially hostility, which emerged as important in the study of the components of the TABP.

2.8 TYPE A BEHAVIOUR IN WOMEN AND OTHER GROUPS

2.8.1 Women

There is a shortage of studies looking at Type A behaviour in women. A few early cross-sectional studies included women and reported differences between Type As and Bs (Rosenman and Friedman, 1961; Kenigsberg et al, 1974). Later studies examined both women and men (Dimsdale et al., 1978; Frank et al, 1978; Blumenthal et al, 1978; Dembroski et al., 1985; Shekelle, Gale and Norusis, 1985; Williams et al., 1988) but the number of women participating was often very small. In the Framingham study, however, the Framingham Type A Scale (FTAS) relationships with CHD were examined specifically in women.

In the cross-sectional phase of the Framingham study, 776 women aged 45 to 64 years with coronary disease scored significantly higher on the FTAS than women free of CHD (Haynes et al., 1978). This was apparent in the CHD categories of angina, both with and without MI. The number of women with a history of MI was too small to examine the category separately. No significant behaviour differences were apparent between CHD and non-CHD cases in women over 65 years. Working women had higher Type A scores than housewives, although angina was more prevalent in Type A women regardless of employment status. Prevalence of CHD among working women, however, was up to four times as great among Type As as Type Bs.

Twenty years on, follow-up analysis was carried out on the women in the Framingham study (Eaker, Pinsky and Castelli, 1992). The study included 749 women who were free of CHD at the baseline examination. Although some psychosocial factors (eg. low education level, perceived financial status, tension and anxiety, loneliness) were associated with the 20-year incidence of MI or coronary death, Type A behaviour was not among them. It was thought that the eight-year relationship observed between Type A and CHD in women may have been a consequence of including angina in the diagnosis of coronary heart disease. Thus, the findings may have been related to self-reported chest pain only and not to definite coronary disease.

Because so little Type A study had been focused on women, work in this area has been encouraged (Thoreson and Powell, 1992). Davidson and Hall (1995) also advocated further study in women, but warned that the SI may assess

different components, particularly different aspects of hostility, in men and women. They found that the Potential for Hostility element of the SI correlated with different behavioural measures in a sample of 45 males and 76 females. The differences have implications for what are considered to be risk behaviours for men and women. They recommended greater attention to exactly what is being measured, and to avoid making the assumption that the same instrument taps the same aspects of characteristics in men and women.

2.8.2 Other groups

There is virtually no Type A research specifically in groups other than white, predominantly middle-class males (Review Panel on Coronary Prone Behaviour, 1981; Matthews, 1982; Matthews and Haynes, 1986; Booth-Kewley and Friedman, 1987; Thoreson and Powell, 1992). Two cross-sectional studies found Type A to be equally prevalent in black and white populations (Anderson et al, 1986; Sprafka et al., 1990; in Thoreson and Powell, 1992), but also found that speech patterns in particular may tap different aspects than usually assumed in the SI. If research focused on these groups, more specific measures would be needed (Thoreson and Powell, 1992).

2.8.3 Problems and section summary

There is simply not enough research in women and other groups to be sure that the conclusions reached about Type A behaviour will apply to them. The research that has been done has assumed that Type A measures tap the same

phenomena in everyone, regardless of sex, race or ethnic background. Recommendations made in review articles argued for greater attention to what is measured, and to whether it is relevant to a group other than predominantly white, middle class men.

2.9 CHAPTER SUMMARY

Friedman and Rosenman pioneered the study of relationships between personality attributes and CHD by defining the Type A behaviour pattern, manifested by competitiveness, time urgency and aggression. They set up the WCGS in 1961 to examine the prospective association between Type A and CHD. They measured the TABP using the SI, a challenging structured interview in which speech style, attitude and behaviour were more important than answer content. The finding that CHD was twice as common in Type A as in Type B men was seminal.

Interest and research grew and it was hoped that Type A could help reliably predict the incidence of CHD. Different types of self-report measures such as the commonly used JAS were developed. At around the eight-year follow up of prospective studies, Type A seemed to be associated with CHD incidence and was nearly established as a risk factor. However, by the 20 year follow-ups, the relationship disappeared. In fact, in studies of recurrent CHD, Type A men had better survival than Type B men. And, in men with many other risk factors, Type A could add no further discrimination between those with or without CHD.

Coronary angiography studies attempted to show one mechanism of the Type

A/CHD relationship: atherosclerosis. It was hypothesized that greater extent of coronary disease would relate to Type A behaviour in patients referred for coronary angiography. Results were equivocal, and fraught with difficulties: the studies were unable to ensure bias-free samples, and could not properly assess causal relationships.

The discrepant findings led to studies investigating possible core elements of Type A behaviour. Only some components seemed to be predictors of CHD: hostility elements in particular were more reliably associated with CHD than global Type A. The focus of research from the mid 1980s has been on the hostility and aggression components of Type A. However, it is unclear whether the Type A or component relationships to CHD are the same in women and other groups, as research has concentrated on predominantly white, middle-class men.

Friedman and Rosenman laid the foundations of personality/CHD research and changed the outlook of the field to include psychological factors. Results have been mixed but the study of components of Type A, especially hostility/aggression, is promising. The next chapter enumerates the hostility/CHD studies and explains the path of that research as the focus changed from Type A to its components.

CHAPTER 3

Hostility and Coronary Heart Disease

3.1 INTRODUCTION

The interest in personality-CHD research never waned once it had gained momentum with the study of Type A behaviour. As the trend toward null findings with Type A was being documented (Miller et al., 1991), research into subcomponents of Type A, especially hostility, was growing rapidly.

Defining Type A behaviour, in spite of the many elements to the pattern, was straightforward. The concept had been developed and refined by two researchers (Friedman and Rosenman, 1959), and was centred on very specific, observable behaviours. In contrast, the definition of hostility is elusive. It cannot be restricted to a set of behaviours, because it also includes the unseen: thoughts and feelings. Accordingly, the literature holds examples of research using widely varying measures of hostility built around equally varying definitions. This makes the sheer amount of findings overwhelming, yet potentially very rich. The main body of this chapter will review the main hostility-CHD studies, their findings and implications; but first the definitions, construct, and measurement of hostility are discussed.

3.2 DEFINITIONS

Buss and Durkee (1957), when factor analyzing their new hostility inventory, found that there was a clear separation between the attitudinal component of hostility, involving resentment and suspicion, and a 'motor' aspect, involving assault, indirect hostility, irritability and verbal hostility. Subsequent research also consistently

identified the same two factors: one to do with the experience of hostility, the other dealing with the expression of hostility (Musante et al., 1989). The experience of hostility is subjective, perhaps involving angry feelings or suspicious, cynical thoughts; expressive or behavioural hostility refers to observable acts of aggression, which may be verbal (eg. insults), or physical (eg. punching) (Siegman, 1994).

The concept of hostility, even across the two factors, is broad. It includes anger, aggression or a chronic negative outlook; that is, it encompasses feelings, overt actions and thoughts or attitudes (Barefoot, 1992). The cognitive components (ie. thoughts) may include cynicism and mistrust, a desire to oppose others or to wish them harm (Smith, 1994; Miller et al., 1996). Spielberger et al. (1985) further broke down these elements, naming the entire complex the AHA! Syndrome: anger, hostility and aggression. Within the literature, anger usually referred to emotion of varying intensity, from irritation to fury or rage (Spielberger et al., 1983). Hostility included angry *feelings*, but also encompassed a person's hostile *attitudes*, which were likely to instigate aggressive behaviour. Aggression referred to destructive or punitive behaviours usually aimed at another person (Spielberger et al, 1983). The crossover of the concepts and their often logical concurrence (ie. angry feelings leading to aggressive behaviour) meant that measurement was difficult and the outcomes sometimes ambiguous, particularly if the measurement instrument did not clearly separate the components.

Because of this lack of consensus among researchers on the definitions, they have made different choices regarding instruments to measure hostility (Miller et al., 1995). Each measure has its own angle and focus; a selection is discussed below.

3.3 HOSTILITY ASSESSMENT

3.3.1 Cook-Medley Hostility Scale

Cook and Medley (1954) developed this hostility scale to be used as part of the Minnesota Multiphasic Personality Inventory, a 550 item true-false questionnaire which was designed to measure a person's ability to get along with other people. The proposed hostility (Ho) scale aimed to assess a teacher's rapport with pupils, and its first reported validity was thus based on its accuracy in doing so (Cook and Medley, 1954). A preliminary Ho scale of 77 MMPI items was administered to 200 graduate students, all of whom were experienced teachers. They also completed the Minnesota Teacher Attitude Inventory, which had been used previously to predict teacher-pupil rapport. The Ho scale correlated -0.45 with the MTAI, and after further refinement, to a 50 item Ho scale, the correlations with the MTAI remained quite similar. Internal consistency of the scale was 0.86. Higher scores indicated a person who disliked and distrusted others:

"he sees people as dishonest, unsocial, immoral, ugly and mean...hostility amounts to chronic hate and anger" (Cook and Medley, 1954; pp. 417-18).

Two items from this scale read:

"When someone does me a wrong I feel I should pay him back if I can, just for the principle of the thing," and " I have often met people who were supposed to be expert who were no better than I." (Cook and Medley, 1954, p. 417).

Greenglass and Julkunen (1989) factor analyzed the Ho scale in order to examine its coherence and structure, and reported an internal consistency of 0.84. Smith and Frohm (1985), in their validation study, also concluded that the Ho scale assessed cynical hostility, and that it demonstrated acceptable convergent and discriminant validity.

The Ho scale has been used widely in health research because of its ease of administration, and because in some samples the MMPI had been administered as part of a battery of tests. With the upsurge in hostility-CHD research, relevant scales of the MMPI, such as the Ho scale, could be re-analyzed using health as an outcome variable (Smith, Sanders and Alexander, 1990). Because cynicism was not usually thought of as an aspect of hostility in the way that rage and anger were (Greenglass and Julkunen, 1989), it is important to remember that scope of the Ho scale is "chronic hate and anger" when comparing results across studies (Miller et al, 1996).

3.3.2 Structured Interview: Potential for Hostility

One of the components of the Structured Interview (SI; Rosenman et al., 1964) was 'Potential for Hostility,' which was identified as a 'toxic' facet of the Type A Behaviour Pattern (TABP) in terms of CHD risk (Dembroski and Costa, 1987). Potential for Hostility (PH) seems to be a fairly stable attribute, and reflects a person's

> "tendency (a) to experience varying degrees and combinations of anger, irritability, resentment, and related negative affects in response to common, everyday events that are likely to arouse them in individuals who are prone to react in such ways, and/or (b) to react with expressions of antagonism, disagreeableness, rudeness, surliness, criticalness, and uncooperativeness" (Dembroski and Costa, 1987; p, 224; Dembroski, 1978; Dembroski et al, 1985).

The presence of these characteristics is gleaned from the content and intensity of responses, and style of interaction with the interviewer. For example, a high-scoring subject, if asked about his level of irritation when behind a slow-moving vehicle, is likely to admit to frustration; he might use strong language in his reply (eg. referring to the slow driver as a 'stupid bastard'), and he might also be antagonistic to the

interviewer by replying in a surly manner, such as (indignantly) saying 'Wouldn't you be angered by that?' (Dembroski and Costa, 1987). The total Potential for Hostility score is based on a subjective judgement by the interviewer, taking the above into account. Inter-rater reliability ranges from 0.70-0.85, and 6-18 monthtest-retest reliability was 0.55 (Dembroski and Costa, 1987).

Potential for Hostility ratings significantly correlate with Expression of Anger, rather than to the Experience of anger (the emotional and attitudinal aspects) (Musante et al., 1989). Potential for Hostility therefore more strongly represents the expressive side of hostility, unlike the Cook-Medley, which centres on the experiential, cynical aspects. It may be that this is picked up through the ratings on tone and intensity of voice and style of interaction, which are not easy to tap using a questionnaire (Musante et al, 1989). Potential for Hostility is not the only scoring system for the SI, but it has been widely used (Smith, 1992). However, there has been little work on construct validity of the PH scoring system.

3.3.3 Buss-Durkee Hostility Inventory

The Buss-Durkee Hostility Inventory (BDHI; Buss and Durkee, 1957) measures seven aspects of hostility: resentment, suspicion, assault, indirect hostility, irritability and verbal hostility. These can be divided into three broader dimensions: cognitive (suspicion), affective (irritability, resentment), and behavioural (assault, indirect, verbal and negativism) (Barefoot, 1992). Factor analysis revealed two underlying factors: experiential and expressive hostility, with the expressive factor mostly representing the behavioural components. The scales were developed, revised

and then re-administered to a sample of 173 college students (half were male, half were female). Each scale appeared to be identifying a partially independent behaviour, but always within an underlying two-factor structure. This inventory, although well-validated, is used rarely in hostility/health research (Miller et al, 1996).

3.3.4 Multidimensional Anger Inventory

This questionnaire assesses frequency, duration, and magnitude of anger, range of anger arousing situations, mode of anger expression and hostile outlook (Siegel, 1986). It was developed specifically for use in CHD research and designed to be psychometrically sound (Siegel, 1992). Factor analysis was carried out on two samples: one of 198 college students (males and females), the other 288 male factory workers. Three elements were recovered: anger arousal, hostile outlook and range of anger-eliciting situations. Anger-arousal included frequency, intensity and duration of anger items; hostile outlook and range of anger eliciting-situations were orthogonal (independent) to anger-arousal (Siegel, 1992). There also appeared to be two modes of anger expression, anger-in and anger-out.

The test-retest reliability over four weeks was 0.75 and internal consistency (Cronbach's alpha) was 0.84 and 0.89 in the two samples. There was adequate convergent validity with other similar scales (Siegel, 1992). This scale is less widely used in hostility-health research than the Ho scale or the Potential for Hostility scale of the SI.

3.3.5 State-Trait Anger Scale; State-Trait Anger Expression Inventory

The State-Trait Anger Scale (STAS) assesses both the intensity of anger and differences in anger proneness (Spielberger et al, 1985; Spielberger et al., 1983). Trait anger reflects a person's tendency to feel anger, which is partially related to a person's frequency of episodes of state anger (Spielberger et al, 1985). Administration of the scales to various samples allowed its refinement to ten items for each scale of trait and state anger (Spielberger, 1989). Internal consistency alphas were 0.93 in both sexes on state anger, and 0.87 for trait anger. Factor analysis revealed only one factor for state anger. There were two for trait anger: angry temperament, or the tendency to express anger generally; and angry reaction, which addresses specific situations, eg. frustration or unfair treatment.

The State-Trait Anger Expression Inventory (STAXI) incorporates anger expression scales in addition to the STAS's state and trait anger measures (Spielberger et al., 1985). After an initial administration to high-school students in the USA, factor analysis clearly showed two dimensions: anger-in and anger-out (Spielberger, 1989?) . Anger control was also found to be an aspect of anger expression: the final scales contain eight items for each scale of anger-out, anger-in and anger-control. Anger-in correlates with neither of the other two, but anger control has a strong negative correlation with anger-out (-0.59). These scales have been used in more recent research and results using them are not as widely reported as those using the Ho scale or PH ratings.

3.3.6 Problems

The above section briefly described two of the most common measures of hostility: the Cook-Medley Ho Scale and Potential for Hostility ratings of the SI, plus three others which have been used often enough to be mentioned here. There are many, many more instruments in existence: a meta-analysis of studies counted 63 (Miller et al., 1996). They are too numerous and sparsely used to discuss. This is one of the major problems in hostility-health research: diversity of measures. Even the two main measures have relatively poor inter-measure agreement; 0.29 between the PH and Cook-Medley (Swan, Carmelli and Rosenman, 1990; Dembroski et al., 1985; Smith, 1992) This probably reflects the differing rationale behind each scale's development, and makes it clear that the measures are not assessing the same construct. Therefore, it appears that results across studies are difficult, if not impossible, to interpret with confidence.

There are also difficulties particular to each measure. As with assessment of Type A, self-report questionnaires pose the problem of 'social desirability' bias and lack of objectivity by the respondent concerning his or her behaviour. However, they are convenient, quick and have reasonable internal consistency (eg. 0.84-0.89 for the MAI; Siegel, 1986). PH scales, on the other hand, have the advantage of allowing a neutral rater to judge whether the subject's behaviour and reactions are telling a different story from the respondent's words. It is, however, time consuming and the bias may arise from lack of objectivity on behalf of the rater, or perhaps from a quirk of the temporary relationship between the interviewer and responder.

These contrasts make study comparisons very difficult, but, if viewed

carefully, do allow us to see which type of measure correlates most strongly with specific health outcomes. However, because there is overlap among the definitions and measurements, it is not always clear what the results of a study mean. This is an ongoing problem which must be addressed if we are to move forward productively (Miller et al., 1991).

3.3.7 Section summary

Hostility is an amalgam of concepts including anger, aggression, disgust, suspicion, cynicism, etc. (Barefoot, 1992). The cognitive, affective and behavioural aspects interweave in a complex fashion: the cognitive component reflects a person's negative view of other people, which may influence affect (Barefoot, 1992). For instance, if one expects others to be antagonistic, one is more likely to feel resentment or anger at what might in reality be neutral behaviour. This in turn may lead to an aggressive response (Barefoot, 1992). However, it is possible for a person to be cynical yet not aggressive, or angry without being cynical. The cynical and emotional factors combine to make up the *experiential* component of hostility, with aggressive behaviour forming the *expressive* component (Miller et al., 1996). The measures of hostility are equally complex, each addressing different aspects of the concept, sometimes encompassing two of the three main components. Usually, however, an instrument focuses on either experiential or expressive aspects, but not both. The Cook-Medley Ho Scale (Cook and Medley, 1954) mainly reflects cynical aspects; other self -report inventories assess angry/hostile affect, such as the MAI (Siegel, 1986), and the STAS (Spielberger, 1983). The Potential for Hostility ratings

(Dembroski and Costa, 1987) allow assessment of the behavioural, aggressive component. The BDHI and STAXI attempt to include all three elements (cognitive, affective and behavioural), yet both are self-report scales, and still may not fully tap the behavioural, expressive element.

Self-report questionnaires may be subject to socially desirable responding, yet are easy to administer and thus useful in large epidemiological studies. PH ratings may be prone to interviewer bias, and are expensive and time consuming, yet tap the expressive elements that may be very important for CHD. Each study must therefore be considered carefully in light of other findings, but this is problematic given the lack of consensus among researchers and instruments. The research findings are quite extensive and are reviewed in the remainder of this chapter.

3.4 PROSPECTIVE STUDIES

3.4.1 The studies

Research on hostility/CHD relationships has taken many forms. These can be considered in three main groups: (1) prospective studies, (2) studies of groups at high risk of CHD, and (3) cross-sectional and case-control studies, often using angiography as an outcome. Prospective studies will be considered first.

The Western Collaborative Group Study was not only important for Type A/CHD epidemiology. It has also played a major part in the history of hostility/CHD research, through the development of component scoring methods for the SI which resulted in Potential for Hostility measures (Dembroski and Costa, 1987). The following two angiography studies, on subsamples of the WCGS, are considered in

this section because they were carried out within the ongoing longitudinal investigation. In the first, Dembroski et al. (1985) found that Potential for Hostility (PH), along with Anger-in, was significantly positively associated with disease severity, including angina and MIs, in a sample of 131 patients who had undergone coronary angiography. Because disease severity was not related to global Type A, the results suggested to them that hostility was at the foundation of coronary-prone behaviour. A second, retrospective, case-control study of 250 cases and 500 matched controls also found that hostility, assessed 12 years previously using the SI, to be the only element of the Type A pattern that was a significant predictive factor for CHD, defined as confirmed nonfatal MI, MI death, unrecognized 'silent' MI or angina (Hecker et al., 1988). The relationship remained statistically significant after adjustment for other coronary risk factors.

Other researchers studied the hostility/CHD relationship using the Cook-Medley Ho Scale (Cook and Medley, 1954). Barefoot, Dahlstrom and Williams (1983) examined the relationship between Ho scores and health 25 years later in 255 medical students who had completed the MMPI on their entrance to medical school. They found that the incidence density of CHD in those with Ho scores at or below the median (13) was 0.9 per 1000 person-years of follow-up, and 4.5 per 1000 for those with scores above the median. This, however, was unadjusted for age, sex, smoking or other factors. It is also unclear how many in the sample were women. The analysis was based on just 4 CHD deaths and 11 self-reported MIs or angina, so errors of misclassification could have seriously affected the results (Hearn, Murray and Luepker, 1989). Thus, the interpretation of the findings is problematic.

In the Western Electric Study, 1877 male employees at the Hawthorne Works of The Western Electric Company in Chicago, aged 40-55 years, were medically examined at baseline in 1957/58 (Shekelle et al, 1983). The MMPI was administered at that time, and the participants were followed up for 10 years by annual examination. Nonfatal MI was coded if there was diagnostic ECG evidence alone, or the ECG was not conclusive, but together with symptom history there was enough evidence for MI. In the event of death, underlying cause of death was obtained from death certificates, and then coded to an appropriate category: eg. fatal MI (as for nonfatal), CHD death (sudden death not attributable to another cause), other cardiovascular deaths, and deaths from other causes. The CHD groups plus sudden death were grouped together for analysis. The Ho scale was administered at the first and fourth exams, with a correlation of 0.84 between the two administrations. The Ho score did not have a straightforward relationship with CHD: it was significantly associated with the crude 10-year incidence of CHD, but incidence was lowest in the first quintile of Ho scores, highest in the middle, and intermediate in the other groups (a 'quadratic' association; Shekelle et al, 1983). Adjustment for age, cigarette smoking, alcohol consumption, blood pressure and serum cholesterol levels did not change this. The relative odds of CHD in a man with a Ho score ≤ 10 was 0.68: a decreased likelihood of CHD with a lower score. The Ho score was also significantly positively associated with all-cause 20-year mortality. In this study, definitions of cardiovascular disease and methods for ascertaining events were sound, but there was uncertainty about the 'quadratic' association present between hostility and CHD, which, because not reaching statistical significance, suggested that the quadratic part

of the finding was not a true one. The results, however, possibly indicated that Cook-Medley defined hostility does affect CHD and overall survival (Shekelle et al., 1983).

Analysis of the relationships between cynicism and mortality in the Western Electric Group Study was carried out by Almada and colleagues (1991). They looked at mortality 25 years after the first examinations in relation to baseline personality assessment. Cynicism, as assessed by the MMPI, was associated with relative risks of 1.4-1.6 for coronary death and total mortality, after statistical adjustment for age, cigarette smoking, alcohol consumption, blood pressure and serum cholesterol levels. A 20-point difference in cynicism was associated with a 50% increase in risk of coronary death over the 25 years. The cynicism scale was scored from 37 items focusing on distrust, and tapped the subject's belief in the selfishness of others (Costa et al., 1985). However, changes in the intervening years in other risk factors may have made a difference to the result, and if cynicism was related to these, then the true relationship will have been confounded. For instance, as Almada and colleagues (1991) noted, if more cynical men were likely to misrepresent alcohol consumption, for instance, because they distrusted the researchers, then statistical adjustment for alcohol consumption will have been incomplete. Therefore, some of the association with death could have been due to the actual effect of alcohol, not to cynicism. Although the cynicism scale is not really intended to measure the broader concept of hostility, the Cook-Medley Ho scale does include 22 of the 37 cynicism items, and the correlation between the two scales was 0.93. This shows again that the main aspect of Cook-Medley Hostility is cynicism.

McCranie et al. (1986) studied the relationship between the Ho scale and health status 25 years later in 478 physicians who completed the MMPI at the time of admission to medical school. They found no evidence of a statistical association between hostility scores, CHD incidence or total mortality. However, the medical school applicants may have tried to present themselves positively (thus generating falsely low Ho scores) and this could be one reason for the lack of association (Smith, 1992).

The MMPI was also administered to 280 men recruited in 1947 for the Cardiovascular Disease Project at the University of Minnesota (Leon et al., 1988). The researchers were interested in cynicism, depression and the Ho scale relationships with incident CHD. There were 43 MIs during the 30-year follow-up period, and there was no statistical evidence of a relationship between any of the scales and CHD in the sample, either before or after adjustment for other risk factors. The authors therefore questioned the consistency of the hostility-CHD relationship across populations.

In a Finnish sample of 3750 men, all twin pairs, Koskenvuo et al. (1988) reported that at baseline, prevalence of angina pectoris was positively and significantly associated with hostility. Hostility had been measured on a questionnaire assessing sociability, aggressiveness, self confidence and conscientiousness, from which a hostility score was derived. After three years there had been 65 deaths and 109 incident cases of CHD. Hostility did not relate to CHD incidence in healthy men, but higher hostility scores *were* significantly related to a new event in a man already diagnosed with CHD or hypertension at baseline. There

was also an increased risk of total mortality with higher scores. The hostility score was based on three items only, however, and its validity was untested (this could lead to an underestimation of effect). Although studying twin pairs may not have affected the outcome, it is possible that genetic confounders may have played a part. For instance, the relationship between heavy drinking and hostility was very strong, but this may have reflected many genetic factors which, in other studies, would have been distributed more randomly. While interesting, therefore, the results perhaps have limited application.

In a study similar in structure to the Barefoot, Dahlstrom and Williams (1983) investigation of 255 physicians, Hearn, Murray and Luepker (1989) ascertained the health status of 1400 male alumni of the University of Minnesota 33 years after their matriculation in 1953. A follow-up telephone survey was conducted in 1985-86 to establish the men's vital status and health. As part of their matriculation the students had completed the MMPI and other personality measures, and these baseline data were available for 1408 men. The 1985 survey incorporated questions on cigarette smoking (past and present habits), history of hypertension and hypercholesterolaemia, history of CHD and family history of CHD. Medical records were sought to verify reports of CHD events. A 'CHD event' was coded if there was a physician's documentation of MI, a positive angiogram or coronary artery bypass graft, with or without the corroborating evidence such as an ECG. Deaths were ascertained from death certificates and the underlying cause of death was coded according to ICD-9. Vital status was determined in 1313 (93.9%) of the men. In neither the analysis of the cohort nor in a concomitant case-control study was Ho score related to any CHD

outcome or to total mortality. Yet the baseline traditional risk factors, such as self-reported cigarette use, hypertension or family history of CHD, had been associated with incidence of CHD.

In a recently reported study of a Danish cohort, Barefoot et al. (1995) administered a shortened version of the Cook-Medley Ho scale (ACM), in 1954, to 436 men and 366 women aged 50 years at the time. They were followed up until 1991 for nonfatal and fatal CHD and all-cause mortality. Outcome groups were defined as acute MI (fatal and nonfatal) and other causes of death as ascertained from death certificates, autopsies and hospital records. The final sample included 409 men and 321 women for whom the ACM measures were available from either the first exam or the ten-year follow-up. Unusually, the analysis showed that hostility was a significant predictor of acute MI in a model adjusting for age, sex and other risk factors (RR 1.04-2.32), but was nonsignificant in the model adjusting for age and sex alone. The RR of all deaths excluding MI, in the fully-adjusted model, was 1.07-1.75. The authors noted that by including a relatively large number of women in the study, and also by examining the hostility-CHD relationship in an older sample, that the test of the association was in fact quite stringent. This, to them, underlined the importance of hostility to health in both sexes, in different cultures, and across the life span.

In the Normative Aging Study cohort, Kawachi et al. (1996) found that high levels of expressed anger were prospectively associated with coronary events. Anger was assessed using the MMPI-2, a revised scale of the MMPI intended to measure anger expression in a fashion similar to the STAXI (Spielberger, 1989). The

participants, 2280 men in the general population in Boston, were recruited in 1961, and in 1986 the MMPI-2 was sent to all the active cohort members. They then continued to be followed up by examination every 3-5 years. At these examinations, a medical history was obtained, potential events were identified and later verified against hospital records. For the analysis with the MMPI-2, there were 1305 men who were free of CHD at the time of its administration in 1986, and whose data were complete. During the follow-up, there were 30 nonfatal MIs, 20 cases of fatal CHD, and 60 cases of angina.

When divided into tertiles and analyzed using a test for trend, the RR of total CHD and angina given a score in the highest versus lowest tertile was 2.66 (95% C.I. 1.26-5.61; Kawachi et al., 1996). In no other outcome group was the association statistically significant once adjusted for other factors, for any level of score, although the direction of the association was always in the direction of more disease with higher scores. Cynicism, although moderately correlated with the anger score (0.50), did not appear to be related to any of the CHD endpoints. When the effect of anger was examined in relation to aspirin use, however, the relationship was markedly attenuated: in those using aspirin, the RR was 0.85 (0.53-1.34); in non-users the RR was 1.41 (1.16-1.72). This study's strengths were its prospective nature and definitions of cardiovascular events, which had been based on strict medical criteria. However, use of a combination of disease categories makes the results harder to interpret: if angina is also associated with traits such as neuroticism, but is included with MI as an outcome, then the meaning of the finding with anger is not completely clear. The mediating effect of aspirin may also be important, but requires a different

study to examine the question.

3.4.2 Problems

There are several possible alternatives for the discrepancies in findings: problems with the nature of the sample, statistical power, low base rates of disease, sample attrition, collection and specification of outcomes, distribution of Ho scores, personality testing procedures, stability of Ho over time, and construct validity of the Ho scale (Hearn, Murray and Luepker, 1988). Many of these explanations for disparate findings were explored by Hearn and colleagues (1988) in their own study, yet apart from various difficulties with the Ho scale or MMPI administration, these other explanations were not supported. However, in some studies low base rates and statistical power were problems, eg. the study of medical students conducted by Barefoot, Dahlstrom and Williams (1983), whose analysis centred on just four CHD deaths and 11 self-reported MIs and angina. However, Hearn , Murray and Luepker (1988) highlighted the need for investigation into the consistency of the Ho scale over time, standardization of testing conditions, re-assessment of the cut-points above which CHD risk is thought to be increased, and further information on reliability and validity of the scale. Most importantly, they pointed out that the Ho scale may not adequately assess the hostility construct, as it reflects mainly cynicism, and may also include items not relevant to hostility. Thus,

"a test of the predictive validity of the Ho scale for health outcomes does not, therefore, comprise a test of the predictive validity of the hostility construct" (p. 120).

They recommended administering a battery of hostility measures in future studies in order to develop and validate better techniques. Otherwise, even universally

consistent findings with the Ho scale would not provide any further information regarding hostility and CHD. Measurement of hostility is by far the biggest problem in prospective studies, although the usual cautions apply when assessing these studies: looking for accurate and complete ascertainment of events, low sample attrition and careful double-checking, and if possible, blind collection of data.

3.4.3 Section summary

Of the nine true prospective studies (excluding the two case control in the WCGS), six were positive and three had a null result. All the null findings were in studies assessing personality using the MMPI, either the Ho scale or a related scale. However, there were consistent positive associations between PH-rated hostility and CHD outcomes. This may indicate that only the type of hostility tapped by PH ratings is associated with CHD, or that there are complicated issues of measurement and design of studies that are causing the difference. Part of this regards the assessment of CHD: some studies had strict objective criteria for events, such as ECG or serum enzyme evidence, others used patient reports; some included angina, which is even more subjective, because it is based only on reports of symptoms. Many used combined categories including both objective and subjective endpoints. The difficulty, therefore, is to discover if hostility relates to the subjective or objective outcome, or both. Prospective studies are essential when examining causal relationships, and because of this the longitudinal findings between PH and CHD, and the weaker association between the Ho scale and CHD, are extremely valuable contributions to the body of research. The myriad of outcome measures used,

however, means that even the results of these prospective studies are incomplete.

3.5 CROSS SECTIONAL STUDIES

3.5.1 Positive findings

As early as 1980, Williams et al. published the results of a study examining coronary artery disease severity in relation to the Ho scale. In their 424 male and female patients, who underwent coronary angiography to assess the extent of atherosclerosis, high Ho scorers were more likely to have 'clinically significant arterial occlusion.' When bisected into high and low Ho scores, the relative risk associated with a high score was 1.46. The statistical significance of the association remained after multivariate adjustment.

In a well-designed case-control study carried out by Barefoot and colleagues (1994), a sample of aircrew of the United States Air Force was selected for study after taking part in their required annual flight physical. Those with an electrocardiogram giving concern were referred for further testing, which could include coronary angiography. Cases and controls were randomly selected from a larger pool of those referred for angiography, and the final sample consisted of 24 cases, who had evidence of coronary artery disease at angiography, and 25 controls, who had no evidence of coronary disease. On the afternoon before the coronary angiographic procedure was carried out, a structured interview was conducted by one of the members of the research team, and the Cook-Medley Hostility Scale was also administered.

The angiograms were independently evaluated by two cardiologists and

classified into case and control groups (Barefoot et al., 1994). The SI audiotapes were analyzed by two raters using yet another scoring system: the Interpersonal Hostility Assessment Technique (IHAT; Barefoot, 1992) giving scores on hostile withhold/evade, irritation, indirect challenge and direct challenge, which when summed form the Hostile Behaviour Index (HBI). Univariate analysis showed that more hostile scores on the HBI were significantly associated with the presence of coronary artery disease, but that scores of the Ho scale were not. Smoking was the only other risk factor to be significantly associated with disease status. When HBI scores and smoking history were entered into multiple logistic models, the HBI score became nonsignificant. Further analysis, however, showed that HBI scores discriminated between the diseased and nondiseased among nonsmokers, but not among smokers. No relationships were found with the Ho scale. The authors could not explain the interaction with smoking, but felt that it was important to keep in mind for future research.

The difference between the Barefoot et al. (1994) angiographic study and others lies in the fact that all the aircrew referred for angiography had been asymptomatic. This makes it harder to support the argument that hostility levels could have been changed by disease. In addition, the selection bias toward those willing to report symptoms, normally present in such studies, was not apparent; all the men were asymptomatic. The lack of association specifically with Ho scale scores was perhaps puzzling, but may have reflected the conditions under which the men filled in the questionnaires. Mean Ho scores were far below reported norms (Barefoot et al., 1994), and because the physical was testing for flight fitness,

responders might have downplayed 'unpleasant' aspects of themselves, thus lowering average hostility scores. They may have been unable to present such a favourable picture when they were interviewed. Alternatively, it may be that the type of hostility assessed by the Ho scale is not be associated with extent of CAD.

Meesters and Smulders (1994), however, found that Ho scores were related to MI in a case-control study of 249 men in the Netherlands. The cases were 81 men who had been hospitalized for their first documented MI. The controls were age-matched, lived on the same or adjacent street as the cases, and were free of MI or serious illness such as cancer. However, the association was only significant in the youngest age group of 35-49 year-old men (OR 1.09, p=0.04, also adjusted for smoking and blood pressure).

Mittelman and colleagues (1995) performed a study that they described as a case-crossover design, in which each study participant served as his or her own control. There were 1623 (501 women) subjects recruited in several Boston hospitals, who were interviewed, on average, four days after an acute MI. The interview explored the 'exposure' to anger and also sought information about other potential triggers of the MI such as heavy physical exertion. Subjects were asked to rate their level and frequency of anger over the past year (the control period) and during the preceding 26 hours (a second control period) before the infarction. For analysis, the usual frequency of anger and anger in the 26 hours before the infarction (but excluding the two hours prior the event) were compared to the two-hour 'hazard period' preceding the MI. They found that the relative risk of MI in the two hours after an episode of anger was 2.3 (95% C.I. 1.7-3.2), and that aspirin appeared to

reduce that risk: RR for the 'angry' aspirin users was 1.4 (0.8-2.6), and for angry nonusers it was 2.9 (2.1-4.1).

The Mittelman et al. (1995) study, however, may be subject to recall bias, as accuracy of reported anger may have differed between the annual frequency and period immediately preceding the MI. The authors argue that the case-crossover design minimizes this because they also included a control of the 26 hour period before the infarction, and the interviewers were unaware of the hypothesis concerning the two hour 'hazard period.' However, there is likely to be a substantial difference in the estimated annual frequency of anger, possibly underestimated, and memory for specific events immediately preceding the MI, perhaps overestimated given their possible subsequent importance. This would falsely increase the strength of the association, so the results must be treated with caution.

3.5.2 Null findings

In two Australian hospitals, 519 patients about to undergo coronary angiography were recruited (Tennant et al., 1987). Subjects provided information by questionnaire on demographic details, coronary risk factors and measures of 'coronary prone behaviour.' Subjects completed the Trait Anger scale (Spielberger et al 1983) and took part in the SI, as well as completing other measures. All the questionnaires and interviews were administered before the angiography was carried out. None of the personality scales showed any significant association with severity of coronary artery occlusion.

Helmer, Ragland and Syme (1991) also found no association between hostility

as assessed by the Ho scale or the HBI and the extent of coronary artery disease. They studied 158 subjects who had been hospitalized in California because of angina, recent MI, abnormal ECG or treadmill test and who were scheduled for coronary angiography. Subjects were asked to participate in the SI and to complete a questionnaire before the angiography. The angiograms were classified by the American Heart Association guidelines and two measures of coronary occlusion were used for analysis: significant occlusion in one artery and a mean occlusion score. None of the hostility measures, either in categories or treated as continuous variables, predicted coronary occlusion in one artery or predicted the mean occlusion score, although sex, history of hypertension and angina did predict mean occlusion score. There was minimal evidence of interaction between hostility score and sex but it was not statistically significant.

3.5.3 Problems

There are more methodological difficulties in cross-sectional studies than in longitudinal studies, particularly those using angiography to classify disease. For instance, the sample patients are often self-selected because they are presenting with symptoms; in prospective studies participants are usually sampled from the healthy general population. This means there may be consistent differences in the populations of cross-sectional studies that have nothing to do with actual disease, but would affect hostility scores. Psychological risk factor status, too, may be affected by the knowledge of one's diagnosis; subtle or even blatant changes in attitude may be apparent. This makes causality nearly impossible to determine.

Questions also arise over the use of coronary angiography both as a means to evaluate CHD (Pickering, 1985) and the inconsistencies in choosing a level of occlusion above which defines a case and below which defines a control (Fried and Pearson, 1987). Pickering (1985) argued that coronary angiography studies are not suitable for investigating the hostility-CHD hypothesis. There were several problems.

(1) The studies use different methods for scoring angiograms and arbitrary cutoffs for disease v. nondiseased.

(2) There is inherent bias toward a preponderance of patients in the studies who do have CHD (because people are referred for symptoms of CHD), and therefore, it is difficult to find a large enough true control group.

(3) There is not a strong association between blood pressure and smoking with extent of coronary atherosclerosis. If clear associations between angiographically determined coronary disease cannot be determined even with these traditional risk factors, then demonstrating an association with a less consistent risk factor such as hostility will be even more problematic.

(4) There may be confounding drug effects in studies evaluating hostility using the SI, as beta blockers are commonly not withdrawn before coronary angiography because of the risks involved in doing so, and taking them can influence behaviour.

(5) CHD involves more than just the developing atheroma in the coronary arteries, and therefore examining this endpoint alone may be insufficient.

(6) Even those with negative coronary angiograms cannot be seen as normal controls, because they are experiencing chest pain, and often other symptoms, that affect their lives even if they have no evidence of physical pathology.

Pickering (1985) also provided a practical solution to these problems: to select patients who are at the stage of noninvasive examinations for coronary disease, such as exercise testing or thallium scanning. Because these noninvasive tests are used for screening before coronary angiography, a much higher proportion of subjects will be found not to have significant disease at that stage, and the control group will therefore be more appropriate and larger. Others (eg. Miller et al, 1996) have said that the use of angiography patients attenuates the variance in CHD and makes Type II errors more likely. Ideally, further longitudinal studies such as the WCGS would take place, although given the expense and time, these will be less prevalent than other types of studies (Pickering, 1985).

The adequacy of the control group in angiography studies is also a problem. Fried and Pearson (1987) investigated how changing the definition of the control group could change the outcome of the study. They recruited a series of patients admitted consecutively to hospital for coronary angiography over 14 months. Prior to angiography information was collected on many risk factors, such as family history of CHD, history of hypertension, smoking habit, alcohol consumption, body mass index, blood cholesterol, etc. For analysis, three different control groups were defined and compared to a case group. The control groups varied from having as much as 49% maximum stenosis to only up to 24% stenosis. Significant differences were seen in the strength of the relationships depending on which comparison group was used. For example, the crude odds ratio for hypertension in males changed from 2.00 to 3.20 as the stringency, and therefore the specificity, of the control group was increased (ie. less stenosis was allowed). The relationships with diabetes, cholesterol

levels, cigarette smoking and family history of CHD showed the same pattern. By including those with more stenosis, who in some cases have enough to be classified as having subclinical disease, in the control group, the power to detect associations is reduced and our understanding of disease is confused (Fried and Pearson, 1987).

3.5.4 Section summary

Findings are inconsistent across studies. Most of those summarized above assessed CHD using coronary angiography, apart from Mittleman et al. who used MI as the outcome variable, and who reported the strongest effect size. Unlike prospective studies, however, null results were obtained even when hostility was assessed with the SI. Null results and weak associations were also found using the Ho scale as the instrument of choice.

Cross-sectional findings are fraught with difficulties of interpretation. They are useful in exploring hypotheses and often build very good foundations for prospective studies, but the prospective studies must always take place. It was not helpful to hostility-CHD research that the cross-sectional findings were so different, however, and yet more studies were being performed to try to arrive at a stronger conclusion. Unfortunately, the advice of those such as Pickering and Fried and Pearson could not be taken into account in studies already under way, so in some senses the research was not progressing at all.

3.6 STUDIES IN HIGH RISK GROUPS

3.6.1 The studies

The rationale behind these studies was the same as for Type A-CHD studies in similar groups (eg. MRFIT; Dembroski et al, 1989). If there is an association between hostility and CHD, then studying a group who are relatively uniform (ie. at high risk of CHD) on other risk factors will help show if hostility levels make a difference to the outcome.

A study in Australia examined survival after MI in relation to the SI and its components (Palmer et al., 1992). Participants who had been admitted with MI were recruited from the Coronary Care Unit of a large hospital in Sydney. One-hundred seventy men and 43 women were interviewed two to ten days after their admission, giving standard demographic information and taking part in a taped SI. The interviews were analyzed later for total Type A content but also for subfactors of Type A, including Potential for Hostility (PH). The Spielberger Trait Anger Scale (STAS) was also administered. Over the 12 month follow-up period, 13% had a nonfatal MI or died from a further cardiac event. None of the SI factors, the total SI score or the STAS score was related to mortality at 12 months.

Julkunen, Idänpään-Heikkilä and Saarinen (1993) found that in 123 patients who survived their first MI, irritability as measured by the Differential Personality Inventory (Jackson and Messick, 1970) was, even after adjustment for age, sex, social status and severity of MI, marginally significantly associated with poor prognosis over 12 months. Scores on the Anger Expression Scale (Spielberger et al, 1985) were also significantly higher in the group who had further complications (either reinfarction or death; 37% of the sample).

Dembroski et al. (1989) reported that in the Multiple Risk Factor Intervention

Trial (MRFIT), in 192 cases and 384 matched controls, only Potential for Hostility showed a significant relative risk with CHD (RR = 1.5; p=0.032). After stratification of the sample into older (>47) and younger (≤47) groups, they found that in the younger group, the association appeared to be quite strong, and was not affected by adjusting for traditional risk factors. However, Potential for Hostility was not related to CHD incidence in the older participants. Stylistic Hostility, too, was associated with incident CHD, after adjustment of other risk factors, in only in the younger group.

3.6.2 Problems

The most obvious problem with high-risk group studies, which often have the advantage of prospective design, is what Miller and colleagues call disease-based spectrum (DBS) bias (Miller et al., 1991). This occurs because in high risk populations the range of possible levels of disease is restricted, making it more difficult to pick up differences in risk factor levels, especially if more of the group as a whole is likely to have uniformly high levels of a risk factor. Therefore, if Type A or hostility is a risk factor for CHD, the studies of those who already are known to have CHD will contain more participants who have Type A characteristics or high hostility levels than occurs in the general population. There was some evidence of this: a greater percentage of the study groups who were at high risk had type A characteristics than in the healthy population samples (70% v. 46%; Miller et al, 1991). DBS bias is also a problem in angiography studies, as patients referred for such an invasive procedure invariably have clinical evidence suggesting CHD, and

therefore many of them are likely to be found to have substantial occlusion of the coronary arteries. Even those who do not have clinically significant stenosis still probably have higher levels of subclinical CHD than the general population (Miller et al, 1996). Therefore, the range of disease is restricted and comparisons on risk factor levels become difficult. To help avoid these difficulties, Miller et al. recommended that: (1) more healthy population studies should be carried out, and (2) that continuous rather than dichotomous measures of personality variables be used. This not only increases the statistical power to detect associations, but also allows the assessment of a possible dose-response relationship.

3.6.3 Section summary

Findings were mixed. The Australian study, using the SI and STAS, found no relationship between hostility and survival after MI (Palmer et al, 1992); in San Francisco, both irritability and anger expression seemed to worsen prognosis (Julkunen et al, 1992); in the MRFIT, PH was only associated with disease in the younger participants (Dembroski et al, 1989). Given the potential complication of DBS bias, plus standard uncertainties concerning measurement and disease/risk factor definitions, both the positive and null findings must be balanced extremely carefully, especially if trying to extrapolate to a wider population.

3.7 STUDIES IN OTHER GROUPS

3.7.1 Studies of women

There have been very few studies including women in their study groups in

this field, and even fewer of women exclusively. However, a small number of researchers set out to examine the hostility-heart disease hypothesis in women, to see if it differed from the relationship often observed in studies of men.

Hällström and colleagues (1986) followed a community sample of 795 women, who were either 38, 46, 50 or 54 years of age at baseline in 1968-69. This subsample, taken from a larger group of 1492 participants, was asked to participate in a psychiatric evaluation during which they completed the Eysenck Personality Inventory (EPI; Eysenck and Eysenck, 1964) and the Cesarec-Marke Personality Schedule (CMPS; Cesarec and Marke, 1968). The participants were followed up twice, once in 1974-75 and then again in 1980-81. Most of those who did not take part in the second follow-up were interviewed by telephone to establish their history of angina and MI. The relationships found were between angina and both neuroticism and passive dependency, and between a more severe grade of mental disorder (such as depression) and the incidence of angina. However, no associations were found with these variables and MI. Aggression as measured by these instruments was not predictive of MI. These results may reflect the measures used or it may be that the risk factors operate differently in men and women.

Adams (1994) analyzed data collected as part of the Mills Longitudinal Study. The final year classes of 1958 and 1960 at Mills College took part in a study of college women concerning their personalities and plans for the future. These women, most of whom were aged around 21 at the time, were predominantly white and of high socioeconomic status. They were followed up by postal questionnaire in 1963-64, 1981 and 1989, ie. when the women were 27, 43 and 52. Hostility was assessed

at baseline and at the first follow-up by the Cook-Medley Ho Scale and at all four ages using a scale derived from the California Psychological Inventory (CPI; Gough, 1987). General health was assessed at age 43 using a five-point scale, and as part of the same questionnaire, the women were asked about their cigarette smoking, alcohol consumption and body mass index.

The hostility scales showed adequate stability over time (from 0.38 to 0.90; Adams, 1994), and higher hostility at each age statistically significantly predicted the self-reported general health at age 52, with the Ho score showing slightly stronger associations than the CPI hostility scale (-0.18 for Ho v -0.14 for CPI at age 21; -0.24 for Ho, v. -0.21 for CPI at age 27). Ho scores were also related to cigarette smoking and marital satisfaction, but the correlations between Ho and general health were not affected when adjusted for these factors. Adams noted that the associations were modest, yet similar to effect sizes observed in other personality-health research and reported in Booth-Kewley and Friedman's (1987) review. This study cannot add much to the body of evidence concerning CHD because of its focus on the outcome of general health rather than strictly defined CHD endpoints, although it was important in establishing that the hostility-health relationship might differ in men and women.

3.7.2 Studies using different indicators of coronary disease

Two studies reported outcomes of peripheral arterial disease (PAD) and hostility. Joesoef et al. (1989) found that among 4462 young (31 to 46 years) male Vietnam veterans, the prevalence and odds ratio of PAD increased with an increase

in the Ho scale scores. The prevalence of PAD progressed from 0.7% in the lowest quartile of hostility to 1.6% in the highest quartile, a statistically significant trend. Odds ratios increased from 1.0 (reference) in the lowest quartile to 1.8 in the fourth quartile, after adjustment for age, race, cigarette smoking, hypertension, family history of CHD, diabetes and serum cholesterol levels. However, the 95% confidence limits of the odds ratios all included one, probably because of the small number of cases. Therefore, it is certainly possible the effect was due to chance.

Deary et al. (1994) also reported results using the outcome of PAD, and found that expressed hostility, measured on the Bedford-Foulds Personality Deviance Scales (Bedford and Foulds, 1978) was significantly associated with the severity of PAD in a random cross-sectional sample of 1592 men and women aged 55-74 years in the general population (a cross-sectional analysis of the Edinburgh Artery Study, the study sample further examined in this thesis). The mean score on the hostile acts scale was raised from 13.9 in normals to 14.6 in claudicants ($p<0.05$). In men, but not women, an increased risk of claudication was associated with an increase in hostile acts score: an odds ratio of 1.41 (95% C.I. 1.01-1.96) after adjustment of cigarette smoking, alcohol consumption, obesity and diabetes mellitus.

Hostility was studied in relation to the progression of carotid atherosclerosis in 119 middle-aged Finnish men in the Kuopio Ischaemic Heart Disease Risk Factor Study (KIHD; Julkunen et al, 1994). The extent of carotid atherosclerosis in this subsample of men was assessed by ultrasonography in 1987 and again two years later. Each time, the mean of six intima-media thickness (IMT) measurements (three in each carotid artery at the site of the greatest IMT) was used to estimate 'true'

IMT. The difference between the two-year mean IMT and the baseline mean IMT was used as the measure of progression of carotid atherosclerosis. Several psychological measures were administered: to assess cognitive hostility, the cynical distrust scale of the MMPI was used; to assess the affective component of hostility, a score was derived from the impatience and irritability factors on the Finnish Type A scale (Jarivoski and Harkapaa, 1987); suppression and control of anger were evaluated using Spielberger's (1985) Anger Expression Scales. After two years, there appeared to be twice the progression of carotid atherosclerosis in the group with high cynical distrust and high anger control scores, after adjustment for traditional risk factors. The effects of these two variables seemed to be additive, and together they accounted for more than a third of the variance in the progression of carotid atherosclerosis. Explanations for why cynicism and anger control, but not anger-in, are related to carotid atherosclerosis are speculative, but it does appear that some form of hostility is related to other indicators of atherosclerosis in addition to coronary atherosclerosis.

3.7.3 Problems

Studies of women exclusively are rare, and that is the main problem. Caveats about study designs apply as usual, so the fewer studies there are, the harder it is to generalize. The remaining studies of different indicators of atherosclerosis also suffer from lack of frequency. Because of this, it is hard to tell if the finding will be reproducible, even if the study is well-designed and using common measures of hostility.

3.7.4 Section summary

The (two) studies in women were not dissimilar in design, but used quite different outcome measures. The first one described investigated aggression and MI, but found no relationship. The second found associations between hostility and health; but health was self-assessed and thus not comparable to other hostility-CHD research. There is a great need for further work in this area if we are to find out if this risk factor relationship differs between men and women.

It is the investigations using alternative measures of atherosclerosis that have had the most uniform findings. Of the three papers summarized, two assessing peripheral arterial disease of the legs, all found a relationship between extent of atherosclerosis and hostility level. Unfortunately, the first two were cross-sectional studies; the third, examining progression of carotid atherosclerosis, was prospective, and also found a relationship between hostility and disease. Comparisons with other studies of coronary heart disease are not straightforward, but these studies do provide additional results for the ever-accumulating research findings. Because these studies were so few, however, wider extrapolation would be unsound.

3.8 REVIEWS AND META-ANALYSES

The meta-analysis carried out by Booth-Kewley and Friedman (1987) was as important for hostility-CHD research as it was for Type A-CHD research, because it was the first review that was able to include the growing number of hostility studies alongside studies of Type A. Although its main focus was on SI-measured v. Jenkins Activity Survey-measured Type A behaviour, it also flagged up some pertinent

observations about other personality variables, often subcomponents of Type A, that were related to CHD. In fact, one of the questions posed by the authors was "Do other personality factors, such as anger and depression, relate reliably to CHD?" (p. 345). The meta-analysis included 83 studies, and when findings were collapsed across them, modest correlations were found for anger ($r=0.14$; $p=0.001$), hostility ($r=0.17$, $p<0.001$) and aggression ($r=0.06$, $p=0.10$) and MI, with hostility showing the strongest relationships of the three. For all CHD outcomes (which included angina and CHD deaths), only hostility showed a significant correlation ($r=0.19$, $p<0.001$). Effect sizes were almost always stronger in cross-sectional studies, especially between SI and disease, although they were not useful in determining causal relationships, a problem that can only be addressed in prospective studies. The overall conclusions of this meta-analysis were that anger and hostility, and also depression ($r=0.21$ with all CHD, $p<0.001$), appeared to be predictive of CHD and that the scope of research needed to be broadened to include them.

Following on from this, Matthews (1988) published an 'Update on and alternative to the Booth-Kewley and Friedman (1987) quantitative review.' Matthews also performed a meta-analysis, but used different decision rules for inclusion of papers and had access to results from four new prospective studies. Her conclusions, however, were quite similar to Booth-Kewley and Friedman's: she encouraged further research, especially prospective, into hostility and CHD, but urged caution given the extremely mixed results already published. She also did not think Type A research should be abandoned, and in line with Booth-Kewley and Friedman, found that SI measures of the Type A pattern and its subcomponents were the most valid.

Miller et al. (1991) examined the trend toward null findings specifically with Type A behaviour, but some of the same problems unearthed by their meta-analysis may also apply to hostility-CHD research. One of these was DBS bias, discussed above. They noted, too, that studies using fatal MI as a disease criterion invariably produced null findings with personality variables. This effect could possibly result from an acceleration of CHD in younger persons who are Type A or high hostility, and therefore the ones who are hardy enough to survive to take part in a study are in fact less likely to die than the Type Bs. This would also help explain the prolonged survival of Type As seen in studies of recurrent CHD (eg., Barefoot et al, 1989; Ragland and Brand, 1988). Alternatively, the effect could result from the misclassification of causes of death or other measurement errors (Miller et al, 1991), but this would not explain why traditional risk factors show strong relationships with CHD deaths in a way that personality variables do not.

An important enumerative review of hostility-health studies was undertaken by Smith (1992). He addressed the emerging problems of the definitions of hostility and its measurement, which had been obscured in previous reviews because of the continued focus on Type A measures. He made clear that the most common measures, the Ho scale and SI-assessed hostility, were not interchangeable and needed careful interpretation. A further important consideration was the slowly surfacing recognition that some of the Ho scale items overlapped with neuroticism, and the potentially very confusing effect that could have on research findings. His strongest guideline for future research was for the evaluation and refinement of hostility measurement, concentrating especially on construct validity and psychometric

properties; in addition, he stressed the importance of prospective studies if significant advances were to be made. His other unique suggestion was to study hostility in social contexts; his belief being that hostility may be more dangerous in some situations than others. Finally, he discussed the alternative trait models of personality and their positive application to health that could be used to great advantage in hostility-CHD research. Using trait-based measures, according to Smith, would allow easier comparisons of studies and would avoid the questions about construct validity inherent in other measures of hostility. As he explained:

"Current research on the topic of hostility and health has come to a crossroads. Additional conflicting findings and lingering interpretive ambiguities may lead to the slow abandonment of this area of research without satisfactory or lasting resolution of the basic issues. Alternatively, thoughtful application of the concepts and methods provided by current personality and health psychology may answer centuries-old questions regarding the impact of hostility on health" (p. 148).

More recently Miller and his colleagues reviewed the literature on hostility and health by performing a meta-analysis of all studies published before January 1995 (Miller et al., 1996). The search resulted in 45 studies that met their selection criteria. They classified these studies according to disease endpoints, by type of hostility assessed (eg. expressive, cynical), by study design and year of publication (to assess DBS bias).

As in previous reviews, they found that most case-control studies reported larger effect sizes (estimated using either the biserial 'r' or tetrachoric 'r', depending on outcomes) and that high-risk studies were more likely to have null findings. For instance, in studies using cognitive-experiential self-report measures of hostility, the case-control studies' r was 0.28, and the prospective studies' r was 0.07. Miller et al (1996) also found that studies using both MI *or* MI plus angina as an outcome had

similar effect sizes (r=0.07-0.08). And, in the 5 studies using fatal MI as an outcome, three reported zero-order correlations (Barefoot, Peterson et al, 1989; Koskenvuo et al, 1988; Maruta et al, 1993).

The most interesting outcome was that of the various hostility measures, SI assessed (expressive) hostility showed the strongest relationship with CHD (r=0.18): this was seen across eight different studies (Miller et al, 1996). And, overall, prospective studies in healthy population samples showed a very small but consistent association between Ho-assessed hostility and CHD (r=0.07-0.08).

In evaluating problems, Miller et al. (1996) drew attention to the poor construct validity of measures, especially the Ho scale. They were equally confused by the lack of conceptual distinctions drawn between aspects of hostility: definitions were adequate in theories but were not rigorously applied in research. To help overcome this, they recommended use of multiple measures, which would help separate effects of a particular measure from effects of a particular construct. They found that methodological difficulties abounded in many types of studies. For instance, angiography and other high-risk studies may be subject to DBS bias; cross-sectional studies may exclude many people who are ill and unable to come forward for study; case-control studies appear to have inflated effect sizes. It is therefore very important that prospective studies of the healthy population take place, using validated, multiple measures of hostility and objective indicators of disease. Miller and colleagues agreed with Smith (1992) that a more comprehensive assessment of personality, such as the five-factor model (Costa and McCrae, 1987), could be very important for discovering which traits are relevant to health.

3.9 GENERAL DIFFICULTIES

The issues complicating interpretation of the vast number of studies are many. The first is that of construct definition, the second, measurement. These alone are enough to cause ongoing problems for researchers and readers alike. Additionally, there are issues surrounding study design, outcome measures and study samples.

Apart from delineating exactly what is being measured using a particular instrument for assessing hostility, and/or trying to develop one that incorporates all aspects of hostility yet can still distinguish between different facets, there is no easy way to make studies more comparable. Various groups (Costa et al, 1986; Siegel, 1986; Musante et al, 1989; Bushman et al., 1991; Miller et al, 1995) have attempted to describe the underlying factor structure of different hostility inventories. Depending on the scales and the samples used in the factor analyses, these tended to identify different factors (Miller et al, 1995). Most did draw distinctions between behavioural, emotional and expressive aspects of hostility, but perhaps with different additional factors or slightly different definitions. Therefore, when trying to draw conclusions from disparate studies, great caution must be used, as factors found among one sample may not generalize to others (Miller et al, 1995). This, coupled with the fact that disease definitions, study designs and patient samples vary widely, means that in order to make sense of past findings, yet still progress, a different strategy will have to be adopted. Giving a battery of tests may be one solution (Miller et al, 1996), or, perhaps, using a more general measures of personality, such as personality traits (Smith and Williams, 1992).

A final problem in hostility-CHD research is the link between neuroticism and

disease. A person who is high on the trait of neuroticism has a tendency to experience negative, distressing emotions and concomitant behavioural and cognitive consequences such as fearfulness, low self-esteem, social anxiety, helplessness, poor control of impulses and irritability (Costa and McCrae, 1987). There is a great deal of research showing that those high in neuroticism report more medical problems, but as Costa and McCrae (1987) explained, well-controlled studies using objective measures of health failed to find direct links between neuroticism and disease. Self-reported complaints, however, were often associated with high neuroticism. This was true for chest pain and subsequent diagnosis of angina pectoris. Angiographic studies have shown that neuroticism, although it did associate with being referred for coronary angiography, did not correlate or had an inverse relationship with extent of coronary atherosclerosis (Blumenthal et al., 1979; Zyzanski et al, 1976; Elias et al., 1982; Schoken et al, 1985). Therefore, only prospective studies using objectively determined outcomes can help decipher the relationships between personality and disease; studies using symptom reports as proxies for disease will not do (Stone and Costa, 1990). By this logic, people high in neuroticism are diagnosed with angina because they are coming forward with symptoms of chest pain resembling angina, but may not actually have coronary atherosclerosis (Costa and McCrae, 1987).

Hostility-CHD studies, therefore, have a problem of interpretation if they use subjective outcomes, particularly if they are not separated from objective outcomes. They may be unable to distinguish whether the hostility measure correlates with the objective outcome (eg. MI) or to the subjective outcome (eg. angina), and therefore, to neuroticism and not to disease. For instance, in the Normative Aging Study

(Kawachi et al., 1996), disease outcomes were grouped together for analysis. By including angina with MI, it was impossible to determine whether anger was related to 'true' angina, MI, or to neuroticism reflected in the angina group. Angiography studies may be biased towards the null because high neuroticism patients are investigated for persistent symptoms, yet are often found to be free of disease (Costa and McCrae, 1987). It is most important in all types of studies, therefore, to use objective measures of disease outcomes, not to combine groups, and to measure neuroticism, which can be accomplished using instruments that assess the five-factor model of personality traits (Stone and Costa, 1990).

The five-factor model of personality traits is based on a growing international consensus and is a comprehensive classification of the basic personality traits of neuroticism, extraversion, openness, conscientiousness and agreeableness (McCrae and Costa, 1987; Deary and Matthews, 1993). The agreeableness dimension is of particular importance because it incorporates many aspects of hostility, yet it is part of a comprehensive system of traits (Deary and Matthews, 1993). According to Smith and Williams (1992),

"a large and potentially important research agenda can be articulated from the application of the five-factor model to issues of physical health...[and] would do much to facilitate progress in the field."

This was the same conclusion that Miller et al. (1995,1996), Smith (1992), Stone and Costa (1990) and Costa and McCrae (1987) had reached. Hence the problems of interpretation posed by the diversity of hostility studies outlined here can be addressed using this approach. Meta-analysis indicated that the strongest relationship was between expressive hostility and objectively determined outcomes (Miller et al.,

1996). Applying the five-factor model provides a way to standardize measures and simultaneously control for neuroticism.

3.10 CHAPTER SUMMARY

Hostility-CHD researchers began their investigations with enthusiasm, because of the strong indications from Type A-CHD findings that hostility was the toxic element in the pattern. Disappointingly, the pattern of positive and negative results that had descended on Type A research also afflicted hostility-CHD research. In fact, it was worse, because there were so many more ways to define and measure hostility than the Type A pattern. The broad concept of hostility, including the two halves of experience and expression of anger, could be further described as encompassing feelings, overt actions and thoughts or attitudes: that is, anger, aggression and chronic negativity (Barefoot, 1992). It was up to the researchers to choose a definition and measurement or, in many cases, capitalize on data that had been incidentally collected years before. The problem was compounded by poor inter-measurement agreement and sometimes questionable validity of the personality or outcome measures.

Despite this, in careful reviews common themes could be found: (1) prospective findings were of most value, (2) in these prospective studies, on aggregate, *expressive* aspects of hostility were associated with *objectively*-assessed CHD outcomes, and (3) in order to standardize results and also avoid confounding with neuroticism, a different, standard way to measure hostility should be considered.

This standard measurement is the five-factor model of personality traits (Costa and McCrae, 1987), and it assesses neuroticism, extraversion, openness, agreeableness

and conscientiousness. It is the agreeableness dimension which incorporates aspects of hostility. There are three advantages: sound psychometric properties, standardization, and a built-in control of neuroticism. Thus, it would allow more direct comparisons across studies. The background to and development of the five-factor model will be described in further detail in the following chapter.

CHAPTER 4

The Quantification of Personality

4.1 INTRODUCTION

Modern approaches to measuring personality, whether narrow aspects of behavior such as Type A or hostility, or wider dimensions such as extraversion, have developed within the context of ever-evolving psychological and personality theories. Trait theories of personality, postulating that underlying, stable traits have a direct impact on a person's behaviour, have come to prominence recently in the form of the 'five-factor model.' Measures to assess these five traits followed rapidly. Although the measures are new, the underlying thoughts are not. The trait concept can be seen in some of the philosophies of ancient Greece, for example. It is in contrast to some thinkers in this century, notably Freud, and later, the behaviourists, that trait theory is a marked deviation.

Before discussing the emergence of the 20th century concept of traits, I will sketch, very briefly, a history of philosophical thought that allowed the modern discipline of psychology and, as part of it, personality psychology, to develop. Following this, an outline of the course of personality psychology in the 1900s will be presented. The changes in thought processes over the centuries, and philosophies about personality in this century, provide the contextual background behind current trait theories. Finally, the trait measures themselves, in particular the five-factor model, and their applicability to personality-health research, and to the current study, will be discussed.

4.2 FROM PHILOSOPHY TO PSYCHOLOGY

The ancient Greeks were fascinated by human behaviour and its causes, and philosophers addressed themselves to questions about humans, their behaviour, and their place in the universe. Hippocrates, for instance, in about 300BC, put forward the theory of 'humours' - that the body's constituents of blood, yellow bile, black bile and phlegm - if not mixed in the correct proportions, resulted in recognizable indispositions (Brennan, 1986). Anaxagoras speculated that there was a 'world-mind, or nous' that ordered the world into the four elements of fire, water, air and earth. This nous also determined the nature of all people, whose individuality stemmed from biological differences (Brennan, 1986). These two philosophies, in addition to being theories about life, are early expositions of the idea of traits.

Over the next several centuries, philosophers continued to debate similar questions about the universe and human life. Much of this philosophical thought contributed to the emergence of psychology, which as an independent discipline, has existed for only about 100 years (Gleitman, 1995). For 'psychology' to be possible, there had to be a concept of 'mind' as separate from the body. Early Greek philosophers such as Plato and Aristotle (c. 300BC) began the thinking about the dualism of the body and soul (Brennan, 1986). This was a theme that recurred from then on, seen in Christianity, for example, and later, in the philosophical ideas developed in different European countries. In the Netherlands, Spinoza (c. 1650) nurtured the idea of a dynamic mind-body relationship, with the mind and body being different aspects of the same substance (Brennan, 1986). In France, also in the mid 1600's, Déscartes taught that the study of the body belonged to physiologists and that

the mind was a separate province; he also believed that the brain mediated between the spiritual mind and the physical body (Brennan, 1986). Of course, many others developed and expanded on these themes, and later, psychologists took for granted that the study of the mind was separate from the study of the body.

Twentieth century theories were grounded, either explicitly or implicitly, in the philosophies of earlier centuries, but also grew out of the 19th century intellectual climates of Europe and America. The rich diversity of thought seen in 19th century British science (such as evolutionary theory), German structuralism (such as Wundt and Titchener), and American functionalism (such as John Dewey and James Cattell), was reflected in 20th century psychological theories, too (Brennan, 1986). Below, I will first discuss Freud, because his influence on psychology in the 1900's was, and is, so pervasive.

4.3 DEVELOPMENTS IN PERSONALITY THEORY

4.3.1. Psychoanalytic theory

Sigmund Freud developed his ideas in the late 1890's and the early part of the 1900s. His basic belief was that human behaviour is importantly influenced by an unconscious part of the self, and that the goal of therapy is to uncover unconscious motivations and the resulting conflicts (Darley, Glucksberg and Kinchla, 1986). According to Freud, the basic structure of personality consists of the id, ego and superego (Hall and Lindzey, 1978; Feshback and Weiner, 1986). The origin of many unconscious desires is the id, which houses all a person's psychological energy, or libido. The libido is, in a sense, a set of biological drives, the fundamental one being

the sexual drive. The id demands that its wishes be immediately fulfilled, including sexual desires, and this can lead to great tension. It is the job of the ego to serve the desires of the id, but the ego is tempered by reality, and is able to plan, and to delay gratification. Actions governed by the ego are sometimes conscious, but often are unconscious. Finally, the superego emerges, and it has two functions, to reward the person for acceptable moral behaviour, but more often, to punish actions or thoughts it deems socially unacceptable. It accomplishes this by creating guilt; it serves as a conscience. The superego is very strict, and works to avoid ever allowing the id to obtain gratification. The interaction of these three elements may cause unacceptable desires, thoughts or memories to be repressed, and relegated to the unconscious. The unconscious then expresses itself in the ways it can, through dreams, for example, or slips of the tongue, or bodily symptoms. Freud's theory attempted to account for a great deal of human behaviour (Feshbach and Weiner, 1986) and left much argument and thought in its wake.

Freud's intellectual descendants were many. His daughter, Anna, Erik Erikson and Heinz Hartman, for instance, did not reject his theory, but expanded and modified psychoanalysis according to their own beliefs. Others departed more radically or formulated entirely new theories in reaction (Brennan, 1986); one of the first to develop an alternative was Carl Jung.

Jung was a student and contemporary of Freud, and he postulated that a person's main goal was self-actualization and growth (Feshbach and Weiner, 1986) He believed that the personality consisted of competing forces that a person learns to balance in the growth and self-actualization process (Hall and Lindzey, 1978). He,

like Freud, believed in the unconscious, but for Jung it was both personal and collective. The collective unconscious is a memory within each of us of our common ancestral history (Darley, Glucksberg and Kinchla, 1986). Jung also believed that a person was either an extravert or an introvert; although they coexisted in the person one was dominant. Extraversion-introversion was the main element, but there were other pairs of tendencies, too. The Myers-Briggs Type Indicator (Myers, 1962) was later developed to assess these domains, and it is still widely used (McCrae and Costa, 1990).

4.3.2 Social psychological theories

Alfred Adler, Karen Horney, Harry Stack Sullivan and Erich Fromm all believed that society influenced personality, although they stressed society's role to different degrees (Feshbach and Weiner, 1986). They departed from Freud on specific points regarding basic influences, but they still retained the basic idea of personality as an 'energy reduction system', with the outward manifestation of personality reflecting inner biological or spiritual forces (Brennan, 1986).

For Adler, unlike Freud, the basic motivation in human drive reduction was not negative, but a positive striving for self-improvement (Brennan, 1986). He also believed that human beings are naturally cooperative and interested in the welfare of others (Feshbach and Weiner, 1986). Horney, too, believed in the possibility of human growth and in the social motivation of behaviour, rather than sexual or biological motivations of behaviour that Freud postulated (Hall and Lindzey, 1978). Harry Stack Sullivan took the view that social relationships and situations were of

primary importance in the individual's development (Feshbach and Weiner, 1986). Erich Fromm's perception of society's influence was that the individual was a victim of a harmful society, and that only through changing social structures could a person's needs be met (Hall and Lindzey, 1978).

4.3.3 Interim summary

Psychoanalytic theory and its offshoots looked to the unconscious to explain behaviour. Expanders of Freud's theory such as Jung, Adler and Horney modified the theory to allow for cultural influences on personality, and theorists such Sullivan and Fromm studied the importance of human interactions within a particular societal context (Brennan, 1986).

Freud's psycholanalytic theory has been heavily criticized, much of it bitter (Hall and Lindzey, 1978). One of the main problems was that it did not generate testable hypotheses: data collection was unsystematic, the concepts were loosely defined, and the theory had little predictive value (Brennan, 1986). This left it vulnerable to both justified and, perhaps, unjustified attacks. Importantly, his theory generated furious debates, and led to the development and refinement of his ideas by followers and dissidents. In addition, few would deny the revolutionary nature of Freud's thinking, which awakened an immense interest in the intellectual pursuit of personality psychology, and which still pervades even lay ideas about personality and its development (Feshbach and Weiner, 1986).

4.3.4 Learning theories

The learning theory approaches to personality, in contrast to psychoanalytical theories, were grounded in the laboratory and in experimental analysis of learning, and rejected the idea of the unconscious as an underlying motive for behaviour. One of the common themes was that behaviours and thus, personality, are learned during the course of development, through experiences of punishment and reward (Feshbach and Weiner, 1986). The learning theorists believed that peronality was a set of learned behaviours (Darley, Glucksberg and Kinchla, 1986). Their theoretical focus was on observable stimuli and responses to those stimuli. The learning theories made few, if any, inferences as to what was happening within the person, and were based strictly on observable phenomena. For the behaviourist, the situation was of primary importance in determining behaviour.

The movement was formally introduced by Watson, an American psychologist, who believed that observable behaviour was the only legitimate subject matter in psychology (Brennan, 1986). His views developed from earlier work (c. 1900) by Thorndike, who developed 'connectionism', or the idea that learning was based on associations between sensing and acting, ie. habits that could be strengthened or weakened (Bower and Hilgard, 1981). Although Thorndike's ideas were about learning and not personality, they, along with the results of Pavlov's experiments on conditioning, were a strong influence on Watson (Bower and Hilgard, 1981). The ideas were further elaborated by Guthrie (1930), who advocated the principle of contiguous association to explain behaviour. After Watson and Guthrie, behaviourism evolved to encompass a wide range of human activity using many different

methodologies (Brennan, 1986). Hull (1943), for instance, used mathematics to explain his theory of learned responses, and Skinner (1938) depended heavily on animal and human experiments to explore his concept of behaviourism.

Hull believed that the current environment could only partly explain behaviour, and that prior training and biological needs were also influential (Bower and Hilgard, 1981). He believed that human beings were creatures of habit, and that habits were acquired and strengthened when a stimulus and response were immediately followed by a reduction in drive (Feshbach and Weiner, 1986). For example, if lunch is always served at 12.30, then it will become a habit to eat at 12.30. The biological drives also included thirst and sex, but there were other drives such as love, power, achievement or money (Feshbach and Weiner, 1986). Hull's theories were highly developed and carefully tested (Brennan, 1986) and generated a great deal of subsequent empirical work (Hall and Lindzey, 1978).

Dollard and Miller (1950) systematically extended Hull's theories to account for personality development, social behaviour and psychological illness. They developed the idea of conditioned anxieties that may explain why unhelpful patterns of behaviour persist (Darley, Glucksberg and Kinchla, 1986), such as a fear of the dentist because of one painful visit as a child, causing the adult with a toothache to avoid the dentist rather than seek relief. There was room in their theory for unconscious determinants of behaviour and for behaviour change. If the individual developed the ability to name these unconscious motivations (achieved insight), then the end result was more adaptive behaviour.

Skinner (1938), unlike Dollard and Miller, was concerned with whether stimuli

were rewarded or not (operant conditioning), and less with the type of associations that caused, for example, a fear of the dentist (classical conditioning; Darley, Glucksberg and Kinchla, 1986). Personality, for Skinner, was merely a collection of reinforced responses, and individual differences stemmed from the different behaviours that were reinforced and the variations in reinforcement schedules, with absolutely no attribution of behaviour to a inner force of any kind (Bower and Hilgard, 1981). The person was likened to a machine, which gives an output dependent on the input. For instance, if a person is observed to be eating, to say that he or she does so because of hunger tells us nothing more. However, if the situation is examined more closely, say to record the number of hours of food deprivation, or to see if it is lunchtime, or if it was particularly appetizing food, then we know the cause of the behaviour (Feshbach and Weiner, 1986). What is required is to determine which features of the environment are linked to which behaviours.

The learning theorists were closely allied to proponents of situationism such as Mischel (1968), who argued for a focus on the characteristics of the situation that were determining behaviour, rather than on characteristics of the person. He believed that theories of personality should centre on the way in which a person responds to any given environment. Two people may make different responses to similar stimuli if past reinforcements have taught them each to respond differently. Behaviours should, therefore, be amenable to change if the reinforcement of them is altered (Darley, Glucksberg and Kinchla, 1986).

4.3.5 Social learning theories

The social learning theorists, who included Rotter, Bandura and Walters, agreed with the behaviourists that patterns of responses are learned through various types of conditioning (Darley, Glucksberg and Kinchla, 1986), but also took account of the influence of cognitive factors on behaviour (Feshbach and Weiner, 1986). Rotter (1954) emphasized the importance of the type of reinforcement that would be salient for individuals. For some, money might be a potent reinforcer, but for others it might be status, sex, power, or affection. Bandura and Walters (1963), whose students included Mischel and Wolpe, also observed the importance of modelling in the behaviours individuals display. An adult showing violence to a blow-up clown doll, for instance, incited almost identical actions in the children who had observed this, compared to those not observing this behaviour (Feshbach and Weiner, 1986). The social learning theorists, like Mischel, stressed the importance of the situation on behaviour, and rejected the idea of behavioural consistency across situations.

4.3.6 Interim summary

The theoretical models of learning theory are diffuse and open to many interpretations and methodologies, although the guiding principle is of a system that recognizes the importance of observable behaviour (Brennan, 1986). The theories are based on testable hypotheses and rely on extensive observation and experiment, which is in direct contrast to the psychoanalytic tradition. Behaviouristic principles have wide applicability in areas such as education, military training, advertising and behaviour modification. However, behaviourism has been strongly criticized for its over-simplification, and thus trivialization, of human behaviour (Bower and Hilgard,

1981). The social learning theories, while recognizing the importance of cognitive influences on behaviour, also face difficulties in dealing with the issues of complex human relationships (Feshbach and Weiner, 1986).

4.3.6 Phenomenological or humanistic theories

Phenomenological theories centred on the person as is, and on the potential of the person (Hall and Lindzey, 1978). According to humanistic theory, it is individuals' responsibility to determine who they are and where they wish to go. Unlike behaviourism, little emphasis was placed on the impact of the environment. Humanistic psychologists studied human choice, creativity and self-actualization, in healthy individuals. Methodology, for them, was less important than understanding the individual, which the behaviourists would not have advocated (Feshbach and Weiner, 1986). They also placed great value on the dignity of the person, and saw the psychologist's role as to understand, not predict or control, others. These theorists recognized that having personal freedom forced individuals to be responsible for fulfilling their potential (Brennan, 1986).

Carl Rogers devised 'client-centred therapy' in the context of his humanistic approach (Hall and Lindzey, 1978). His theory stressed the importance of the personal and subjective relationship between the therapist and the client (Brennan, 1986). He believed that the main goal of a person is self-actualization; to develop talents to the fullest (Feshbach and Weiner, 1986). People are able to do this if they have a firm base of love and acceptance, which helps them realize their basic goodness. The therapist may need to provide previously lacked unconditional acceptance that is

necessary for further growth. In order to reach self-actualization, the actual image of oneself must nearly match the ideal image of oneself (Hall and Lindzey, 1978; Feshbach and Weiner, 1986).

Maslow developed the self-actualization theory in more detail, and proposed that there was a hierarchy of needs, of which the first must be met before the next can be attained (Feshbach and Weiner, 1986). These were physiologic, safety, love, esteem and self-actualization. A 'self-actualized' person was considered to be self-aware, creative, spontaneous, open and self-accepting (Brennan, 1986).

Humanism, with its focus on the person and potential for growth, overlaps heavily with existential philosophy and the 20th century writings of Kirkegaard, Sartre, and Camus (Brennan, 1986). Important issues were considered to be freedom, choice, anxiety, meaning, authenticity and struggle (Feshbach and Weiner, 1986). The central tenet was that people were responsible for their decisions, including behaviours, and that by confronting their anxieties, they eventually mature and gain authenticity (Hall and Lindzey, 1978). The humanistic or phemomenologocial psychologists shared the belief that mental processes were active and important in behaviour (Brennan, 1986), unlike the behaviourists, who rejected the role of thought in behaviour. This movement has had an impact on clinical, theraputic applications, although its applications were markedly different from those of behaviourism.

4.3.7 Comment/section summary

The pscyhoanalytic and humanistic theories of personality were philosophies of life rather than attempts to describe, classify and compare people according to

systematic differences in personality (McCrae and Costa, 1990). The behaviourists attempted to explain 'personality' as a collection of learned responses. All of these theorists could see that people were different, and their theories were constructed to explain the disparate personal or societal experiences that made people distinct from one another. There was an assumption that personality, or the person's cumulative collection of behaviours, could be changed given the right insight into unconscious motives (psychoanalysis), through manipulation of the environment (behaviourists) or building up of self-belief (humanistic). Some believed that there were basic types (Jung's extravert-introvert) that would continue to affect the person's reactions to the world even if self-actualization was achieved. These psychologists were looking for the reasons, either internal or external, behind the actions.

The trait theorists, discussed below, set out to describe and classify common patterns of behaviour (Gleitman, 1995). Unlike the behaviourists, they believed that the person, and not solely the situation, was central to behaviour. But the trait theorists were criticized for not being concerned with the causes of behaviour (Millon and Davis, 1994; Block, 1995; Butcher and Rouse, 1996; Widiger and Trull, 1997). They left that to the clinical psychologists and more recently, the geneticists. In their endeavours they identified traits, which were not new, but the use of powerful statistical techniques often showed that there appeared to be five basic traits: extraversion-introversion, neuroticism, openness to experience, agreeableness and conscientiousness (McCrae and Costa, 1990). These theorists believe that traits are common to everyone (nomothetic), and that people merely differ on the 'level' of each trait.

4.4 TRAIT THEORY AND EMERGENCE OF THE FIVE FACTOR MODEL

Most languages include personality trait terms (Goldberg, 1981; 1993), and thus, common sense indicates that people are stable enough to be described using standard terms (eg., shy; aggressive; gregarious). Goldberg's main assertion was that important personality traits will have been encoded in natural language. Traits are defined as "dimensions of individual differences in tendencies to show consistent patterns of thoughts, feelings and actions" (McCrae and Costa, 1990). These traits are found in different degrees in people, with perhaps a definite 'type' represented by the extremes on continuous dimensions (McCrae and Costa, 1989; Widiger and Frances, 1985; McCrae and Costa, 1990). Traits are general dispositions, propensities to behave in a certain way, not absolutes. Hence, a very shy person may in fact be quite open and relaxed among close friends; that is, the trait may be affected by situation and mood (Matthews and Deary, in press). However, trait theorists do assume and believe that personality traits have a direct impact on behaviour and that they are stable over time. In order to support this viewpoint, researchers must be able to quantify the trait and to test its stability, as well as to determine if there is a framework of traits on which any person can be described.

The science of trait theory developed recently, and involved systematic data collection, statistical analysis, and testable theories (Matthews and Deary, in press). The ideas behind current trait theory can, however, be traced back to the ancient Greeks, as discussed in the introduction to the chapter: Aristotle, for example, theorized about the traits of vanity, modesty and cowardice, and Hippocrates

postulated that there were four 'humours' (blood, phlegm, black and yellow bile) (Brennan, 1986). Galen then matched Hippocrates' humours to particular temperaments: melancholic, choleric, phlegmatic and sanguine (Matthews and Deary, in press). An imbalance of them led to physical or mental illness.

Sir Francis Galton proposed the idea that differences in personality will be apparent in language (known as the lexical hypothesis; Matthews and Deary, in press). The early scientific research, therefore, involved the study of trait terms in language (Allport and Odbert, 1936). Allport and Odbert undertook the task of extracting trait-relevant terms from the dictionary, and found 18,000, of which 4,000 clearly referred to personality traits (McCrae and Costa, 1990). Thirty-five clusters of these terms were later grouped by Raymond Cattell (1946), and of these 35 clusters, 12 dimensions were uncovered. These 12, plus four others factored from another series of questionnaires, formed the basis of the 16 Personality-Factor Questionnaire (16-PF; Cattell, Eber and Tatsuoka, 1970).

Tupes and Christal (1961, cited in McCrae and Costa, 1990) conducted a series of studies using Cattell's original 35 scales. They found five traits, not 16, across many samples. Norman (1963) replicated their findings. Goldberg (1981) went again to the dictionary, reduced the terms, and significantly, also came up with five factors. Many lexical studies that looked systematically at language terms have fairly consistently arrived at five broad factors (Digman, 1990; Goldberg, 1993; John, 1990, Widiger and Trull, 1997).

Costa and McCrae had originally settled on three dimensions for their model of personality: neuroticism, extraversion and openness to experience. However, when

they compared their three factors with Norman's (named extraversion/surgency, agreeableness, conscientiousness, emotional stability and culture), they saw that their three could be nested into his five (McCrae and Costa, 1990). Accordingly, they added agreeableness and conscientiousness to their inventory (Matthews and Deary, in press).

The earlier, widely used and accepted system of three traits developed by HJ Eysenck (Eysenck and Eysenck, 1975) included measures of psychoticism (P), extraversion (E), and neuroticism (N) (Costa and McCrae, 1995; Gleitman, 1995). An extravert on Eysenck's scales is sociable, likes excitement and takes chances, is unreliable and may lose his temper; the introvert is quiet, retiring, prefers books to people, controls feelings, is reliable and has high moral/ethical standards (Matthews and Deary, in press). A person who is high N tends to be anxious, depressed, worried, sleeps poorly, has psychosomatic problems, and lets emotion conquer rationality. Unsurprisingly, a low N is calm and unflappable, and is emotionally resilient. The P scale assesses emotional coldness, hostility, egocentricity and lack of impulse control; a high scorer may be susceptible to psychiatric disorders (Pritchard, 1991).

The five-factor model (FFM) has been widely used in more recent research. As noted, it includes neuroticism and extraversion, which are very similar to Eysenck's dimensions, except that the extravert of the five-factor model is not necessarily unreliable or temperamentally volatile, but is cheerful, lively and assertive, and craves excitement (Costa and McCrae, 1990; Matthews and Deary, in press). The three other factors are openness, which incorporates aesthetic sensitivity, intellectual

curiosity, a need for variety and broad-mindedness; and agreeableness, or trust, altruism, sympathy and friendliness (not cynical, self-centred or antagonistic); and conscientiousness- disciplined goal-striving, and adherence to principles (Avia et al, 1995; Costa and McCrae, 1992; Digman, 1990; John, 1990).

4.4.1 Measuring traits

Traits are among the easiest conceptions of personality to measure, because traits are generalized dispositions, and therefore evidence for them can be gathered by examining behaviour across situations (McCrae and Costa, 1990). These measures ought to agree over time, and each measure must allow a range of scores, to reflect individual differences on each trait. There has been debate concerning the best way to accurately assess traits, as investigators are aware that self-reports may not be totally accurate reports of behaviour (McCrae and Costa, 1990). Although repeated, objective *observation* of behaviour would be the ideal measurement, it is expensive. Fortunately for researchers, there has been much research showing that there is good agreement between peer ratings, observations, and self-reports of behaviour (McGowan and Gormly, 1976; Small, Zeldin and Savin-Williams, 1983). The highest correlations were reported between self and spouse ratings (McCrae and Costa, 1990). Validity scales, designed to try to assess the extent of socially desirable responding, have sometimes also been built into questionnaires, but the accuracy of the self reports seems not to be improved by them (Dicken, 1963; McCrae and Costa, 1983; McCrae et al, 1989). There have been a number of different inventories developed to measure personality traits, some explicitly for the FFM and others not; a selection

is described below.

4.4.1.1 Minnesota Multiphasic Personality Inventory (MMPI)

The MMPI has been widely used in medical, psychiatric and other settings, such as mass testing of soldiers or matriculating students (Lubin, Wallis and Paine, 1971). It was originally designed to measure abnormal personality, and thus each scale consisted of items that discriminated various diseased groups from a normal group (Feshbach and Weiner, 1986). It is a true-false questionnaire, made up of more than five-hundred items, and has ten scales, plus four validity scales. The scales measure hypochondriasis, depression, hysteria, psychopathic deviance, masculinity-femininity, paranoia, psychasthenia, schizophrenia, hypomania and social introversion. Further scales, such as the Cook-Medley Hostility Scale, were later added (Cook and Medley, 1954; discussed in previous chapter). Its test-retest reliability over the short-term is moderate, but for longer time periods, the scores are not stable (Dahlstrom and Welsh, 1960). This might be expected as it was supposed to measure psychopathology, which would be expected to fluctuate more than normal personality does (Feshbach and Weiner, 1986). This scale has not been used widely in the UK, however, and its structure has never been validated.

4.4.1.2 The 16 Personality Factors (16-PF)

Raymond Cattell developed the 16-PF using factor analytic methods. He added more descriptive terms to Allport and Odbert's (1936) list of 4500 trait terms,

and reduced it to 170 that captured the meanings of the broader array of terms. He then asked students to describe their friends using this list, and factor analyzed the results. Using this process he eventually arrived at 16 factors, or source traits (Cattell, 1957). Secondary factor analysis of these identified a smaller number of second-order factors, which included introversion-extraversion, anxiety, affectivity and freewill-resignation (Cattell, Eber and Tatsuoka, 1970). The introversion/extraversion dimensions have been found most often in other factor-analyses (Feshbach and Weiner, 1986). Short-term test-retest reliability is greater than that of the MMPI (Feshbach and Weiner, 1986). A study over ten years in 139 men, aged 25-82 years, reported that on the 16-PF, tension, adventurousness, liberal thinking, tender-mindedness and superego strength were stable over time (Costa and McCrae, 1978). In a separate study, eight-year stability was evident on outgoingness, emotional stability, assertiveness, being happy-go-lucky; conscientiousness, suspiciousness, imaginativeness, shrewdness, liberal thinking, independence, controlledness or tenseness (Siegler et al, 1979). This scale has since been further refined, now known as the 16PF 5, and it has five second order factors.

4.4.2 Measures assessing the five-factor model (FFM)

4.4.2.1 Goldberg Big Five Markers

Goldberg (1990; 1992) did extensive work in extracting trait terms from the dictionary and then refining the list to form his adjective checklists that measured the FFM. These take different formats (Widiger and Trull, 1997): one checklist comprises 100 unipolar adjectives (for example, rude, timid). Another is a list of 50

bipolar adjectives (for example, timid v. bold). The adjectives are rated on a nine-point scale by the respondent as to their relevance to that person. The terms are sometimes arranged alphabetically, and sometimes by domain. A major advantage of the lists is that they can be completed in 10-15 minutes, and that they adhere very closely to the five single terms of the FFM (Widiger and Trull, 1997). Some disadvantages may be that they don't address more maladaptive traits (Tellegen, 1993) or that they do not contain the right terms (Block, 1995; Tellegen, 1993).

4.4.2.3 The NEO Personality Inventory (NEO-PI)

The NEO-PI was developed by Costa and McCrae specifically to measure the five factors so often identified in their own and others' studies (McCrae and Costa, 1990). It comprised 181 items and had two forms- either in first person for self-reports, or in third person for peer or spouse ratings. The respondents reply to each item on a five-point scale, from strongly disagree to strongly agree, with a neutral option available. The replies are then summed to obtain the domain scores. In the early version, there were facet measures for the neuroticism, extraversion and openness dimensions, although not for the agreeableness and conscientiousness scales. For instance, neuroticism was broken down into anxiety, hostility, depression, self-consciousness, impulsiveness and vulnerability. To assess the stability of the measure, a six-year longitudinal study was conducted on 983 men and women aged 21-96 years (Costa and McCrae, 1988b). Self-reports and spouse ratings were obtained. The scales appeared to have good stability over time (McCrae and Costa, 1990).

A revised version of the NEO-PI, the NEO-PI-R, contains 240 items, and *each*

domain is split into six facets. The NEO-PI-R is now the predominant measure of the FFM (Widiger and Trull, 1997). There has been a great deal of reliability and validity research carried out, and it demonstrates consistent convergent and discriminant validity with adjective checklists (Costa and McCrae, 1992). Its advantages are the lesser ambiguity of full statements over single adjectives, its precise assessment of the domains and facets, its empirical support and its wide applicability (Widiger and Trull, 1997). Its disadvantages are common with the other measures of the FFM, in that it does not address more maladaptive personality (Widiger and Trull, 1997; Widiger and Costa, 1994) and that there is some argument over openness as a domain of personality (Clark and Livesley, 1994; Trull, 1992). A short version - 60 statements - of the NEO-PI-R is the NEO-FFI, which assesses only the main domains, not the facets.

4.4.2.4 Other measures of the Big Five

Some instruments have been developed with a precise theoretical background in mind. For instance, the Hogan Personality Inventory (Hogan, 1986) was designed to assess the five factors from a socioanalytic perspective (Widiger and Trull, 1997). It correlates reasonably with Goldberg's measures, although not as consistently as some other instruments. It may be most useful in business settings, because of its focus on career, job performance, leadership and competitiveness scales (Widiger and Trull, 1997).

The PSY-5 (Harkness, 1992) is related conceptually to the lexical FFM, but there is much more emphasis on abnormal personality (Harkness and McNulty, 1994).

For instance, the dimension of aggressiveness includes elements of cruelty, violence and enjoyment of frightening others (Harkness and McNulty, 1994). It is therefore useful in clinical settings (Widiger and Trull, 1997).

The Interpersonal Adjective Scales (IASR-B5) are an alternative to Goldberg's (1990; 1992) adjective checklists (Widiger and Trull, 1997). The IASR-B5 relate particularly to the interpersonal circumplex model of personality that centres on surgency (extraversion) and agreeableness. All eight of the IASR-B5 scales loaded highly on extraversion and agreeableness.

There are also other instruments, which will continue to develop and some will be more relevant in particular settings. Generally, the measures share the ability to classify a wide array of people on standard dimensions. The measures, however, may be subject to distortions by mood states in the subjects, and there is also the possibility that the facets are over-generalized to a domain covered by one word (Widiger and Trull, 1997). With careful attention to these matters, however, careful research will enable replicable studies and greater understanding of both personality traits and any associated health outcomes (Smith and Williams, 1992).

An important issue to determine about the NEO-PI, and the FFM generally, apart from measurement, is its comprehensiveness: can the model account for traits in other systems? It would seem to be so. Relationships were consistently found, across many questionnaires and dimensions: with the California Q-Set (McCrae and Costa, 1990), the revised Interpersonal Adjective Scales (Wiggins et al, 1988), Jackson's Personality Research Form (PRF; Jackson, 1984), the Guilford-Zimmerman Temperament Survey (Guilford et al, 1976), the Eysenck Personality Questionnaire

(Eysenck and Eysenck, 1975), and the Myers-Briggs Type Indicator (Myers and McCaulley, 1985) (McCrae and Costa, 1990). This led McCrae and Costa to conclude that:

> "the five factors appear to be both necessary and sufficient for describing the basic dimensions of personality; no other system is as complete and yet as parsimonious." (1990; p.51).

4.4.3 Stability of traits

Critics of trait theory have cited studies showing that behaviour varies widely across situations (Mischel, 1968). They have argued that the impact of the social setting - the situation - on behavoiur is much greater than the person's intrinsic qualities (Gleitman, 1995). However, proponents of trait theory have noted that behaviours that look different may, in fact, be a manifestation of the same underlying trait (Rorer and Widiger, 1983). For instance, aggression may takke different forms at different ages: boys may hit each other whereas men may shout (Kagan and Moss, 1962). Moreover, traits have been shown to be consistent in individuals who are measured at various points in time (Block, 1971). The longitudinal studies of the 16-PF and NEO-PI have provided strong evidence for the stability of the measures, and by implication, the traits, over time (McCrae and Costa, 1990). This does not mean that the state and situation are unimportant, however, as is discussed below.

4.4.4 Traits, states and health

A state is a temporary condition experienced by a person, such as a feeling of anger, perhaps, or happiness. Trait theorists do not deny the importance of states in the determination of behaviour, and most personality researchers are interactionists:

they believe that both the trait and the situation or state contribute to behaviour (Matthews and Deary, in press). This has been shown in the work of Spielberger on state and trait anxiety (Spielberger, Gorusch and Lushene, 1970), and in health research documenting the importance of person by situation interaction in cardiovascular reactivity (Siegman et al, 1992; Siegman, 1993). Wright and Mischel (1987) demonstrated that both traits and situations were predictive of behaviour. The situation itself may also in part be determined by the person: it has been shown that children with different temperaments elicit different behaviour in their parents, which in turn creates a separate set of circimstances for each child (Scarr and McCartney, 1983).

Fortunately, both traits and states can be measured, as is possible on the State-Trait Anger-Expression Inventory (Spielberger, 1989) and it is important to know which one is being measured before drawing conclusions about any relationship, say, between personality and health. The implications are different if a state, rather than a trait, is associated with disease. The widespread acceptance of the principle traits and states, though, has been important for health research. Type A behaviour, for instance, is a good example of an interactionist pattern: the person prone to competitive, aggressive behaviour displays it readily given the right situation. Hostility, too, has the interactionist bent: a cynical, mistrusting general attitude brings out instances of hostile behaviour toward others.

The epidemiologists studying CHD and the personality trait theorists developing the Big Five worked independently of one another. The Type A behaviour pattern definition grew out of observations, by medical doctors, of a pattern

of behaviours they noted in their cardiac patients. It had no theoretical basis, and the measures were designed with attention mainly to face validity (Matthews, 1992).

Some instruments to assess Type A, like the self-report Jenkins Activity Survey, were simple to administer and were included in many large-scale CHD studies, either new or already under way. By 1981 the evidence seemed strong that Type A was one of the risk factors for CHD and there was a huge interest it (eg, Review Panel of Coronary-Prone Behavior and Coronary Heart Disease, 1981). This led, in turn, to a more general interest in personality and CHD links, and research groups went back and looked at MMPI data, for instance, in relation to health (eg. Williams et al, 1980; Almada et al, 1991). MMPI hostility was inconsistently found to be associated with CHD (Miller et al, 1996). With the developments in factor analytic techniques and also new ways to measure Type A, its components were scrutinized and it seemed that hostility was the 'toxic' component of Type A (eg. Johnston, 1993). During this same time period, the personality theorists were working from a different angle. They had constructed an integrated system on which any person could be classified: the five factor model.

There was little overlap of between the psychological and medical disciplines, but with the FFM there was a way to integrate them and perhaps to advance personality-health research. For epidemiologists, the FFM is ideal. Epidemiology is concerned with patterns of disease in the population; if these patterns are also connected with specific, quantified patterns of personality, then we may be able to improve disease prediction and prevention. The FFM traits are precise, as are some medical outcomes, and any person can easily be classified on the trait system. Then

the patterns can be examined, to test which of the objective and subjective outcomes relate to the traits. If there is a true association between a trait and a disease, the experimental findings should readily replicable. The physiological underpinnings of these traits can be studied simultaneously and may even help unravel the 'why' of associations. Epidemiology and psychology may not seem compatible; the former is a study of disease patterns in populations; the latter a study of the individual. However, if the single unique person can be distinguished from another unique person on trait combinations, then each single person in a study can, too, and will allow the pattern of trait-disease associations to emerge.

For behavioural CHD epidemiology, assessment of traits and states as actual measurable quantities is essential. It is impossible to denote patterns of personality and disease without standard, reliable and valid measures of personality. If the trait can be measured then we can look for its relationship to CHD. Yet it is still vital than an acute problem brought about by a state (severe shock, for example, and sudden death) can be distinguished from a problem brought about by a basic, underlying disposition. Both approaches can be used in behavioural epidemiology. The five-factor model has already been productively used in studies examining both risk-taking and positive health behaviours (Booth-Kewley and Vickers, 1994), and it can help clarify redundant concepts within health psychology (Smith and Williams, 1992).

For the Edinburgh Artery Study (EAS), which is the population studied in this thesis, it became obvious that measuring the five factors to test the trait associations with disease prevalence was an essential step. It was also necessary and prudent to build on past findings by examining the narrower dimensions of hostility or anger-

proneness in relation to CHD. Anger and hostility may be aspects of the five factor dimension of agreeableness, but they are more like *facets* than traits, and of course can also be *states*. This strategy will allow the investigation of the effects of both broad and narrow traits on the prevalence of cardiovascular diseases in the EAS.

Looking at personality-health research from a philosophical viewpoint, it is interesting that the ancient theme of the mind-body dualism is reiterated. The distinct separation of the study of the mind and body has brought us to the point where we must re-investigate how they interact. This issue is also now being addressed by geneticists, who are discovering evidence for the biological bases of traits (eg. Plomin, Owen and McGuffin, 1994; Bouchard, 1994). However, the philosophical position of behavioural epidemiology is not important for the conduction of this research, I will leave the discussion there and summarize the chapter instead.

4.5 CHAPTER SUMMARY

Psychology grew out of the philosophical ideas about human behaviour and the distinction between the body and mind. Specific theories about personality developed in parallel with the societal changes of this century. Since the early 1900's, Freud's psychoanalytic theory has had a great impact on psychologists and also the lay public. Subsequent theorists expanded, built on or rejected his theories and many influenced people's thinking and the vocabulary they used (eg. ego, neurotic). However, personality theories were in essence philosophies of life, and it was not always easy, or even possible, to *measure* personality as conceptualized by the theorist. The behaviourist understood measurement, but could not tap the richness

of human behaviours. Recently, however, the historical concept of traits has become prominent and widely accepted. The modern trait theorists began the scientific study of traits by examining natural language, and through statistical techniques, were fairly consistently able to reduce important descriptive terms down to five main groupings. These dimensions are known as neuroticism, extraversion, openness or culture/intellectance, agreeableness and conscientiousness. One widely used and well-validated instrument designed to assess these traits was the NEO-Personality Inventory (Costa and McCrae, 1992). The ability to measure traits is essential for behavioural epidemiology, as it allows the connection between patterns of disease and patterns of personality to be studied, and opens another route for improved disease prediction and prevention.

CHAPTER 5

Aims and Objectives

Prevention of coronary heart disease and other manifestations of cardiovascular disease is a primary goal of health professionals. This is only possible if the natural history and all types of risk factors for the disease are known (McCrae and Costa, 1989). We already know the importance of educating people about smoking, cholesterol levels and hypertension. However, not all cases of coronary heart disease can be explained by the presence of those risk factors, and personality differences between people may help explain why some become diseased and others do not (Dembroski and Costa, 1987). Past studies have been conducted in isolation from one another (Marshall et al, 1994), using various instruments to measure Type A and hostility.

The emergence of the five factor model (FFM), however, has created new opportunities for health research. The NEO-Five Factor Inventory (Costa and McCrae, 1992), a short form used to elicit the five factors of neuroticism, agreeableness, openness, extraversion and conscientiousness, is a standard, self-administered, short questionnaire and can be used in widely varying study populations. Its reliability and validity make it a potentially useful tool in behavioural epidemiological research.

However, the specificity of expressed anger to CHD is becoming a more consistent finding (Miller et al, 1996). Therefore, standard and reliable measures of anger, as obtained on the State-Trait Anger Expression Inventory (Spielberger, 1989, STAXI) will be important in order to compare results across studies.

Recommendations for studies examining personality and CHD have called for a number of elements. These include conducting research in longitudinal studies, careful separation of subjective and objective disease endpoints, and clear definitions of the personality constructs being studied.

The aim of this study is to further elucidate the role of personality on the development of cardiovascular disease. In order to do this, a number of conditions must be met.

(1) Personality must be measured in a standard and reproducible manner. The five factor model (FFM) offers a way to do this, by applying a widely accepted model of personality to health.

(2) Previous developments concerning the role of hostility and anger with CHD, which have been inconsistent, need further follow up, and will be especially useful alongside broader dimensions of personality.

(3) Subjectively and objectively defined cardiovascular events must be clearly separable and analyzed independently so that a 'disease-prone personality' can be distinguished from the 'distress-prone personality' (Stone and Costa, 1990).

(4) The measurement of subclinical as well as clinical disease can help to distinguish objective versus subjective endpoints.

(5) Prospective analysis is necessary to help establish the causal directions of personality-disease relationships.

Bearing these conditions in mind, this study sample will be administered the NEO-FFI (McCrae and Costa, 1992) to measure the neuroticism, extraversion,

openness, conscientiousness and agreeableness traits. Their relationship to prevalent CHD will be examined. The outcomes are both subjective (angina) and objective (myocardial infarction), and include measures of subclinical disease such as the ankle brachial pressure index (ABPI; to measure arterial disease in the legs) and intima-media thickness (IMT; to measure carotid disease). This study is the first in the UK to examine these five core personality traits as well as hostility and anger with these objective measures of cardiovascular diseases in an older population.

At baseline, in 1988, measures of hostility-related variables were measured in this sample using the Bedford Foulds Personality Deviance Scales (Bedford and Foulds, 1978; PDS). These will be analyzed prospectively with incident CHD as well as with the ABPI and the change in ABPI over five years.

Both sets of personality variables will be studied in relation to risk factors for CHD such as hypertension, smoking and blood cholesterol. This will allow multivariate models to be adjusted for possible confounders and will allow examination of any independent relationship between the personality variables and the risk factors.

The objectives of the thesis, therefore, are to address the following questions:

(1) Are the hostility or dominance-related traits of the PDS associated with increased incidence of cardiovascular events?

(2) Do the PDS traits predict severity of subclinical disease as measured by the ABPI?

(3) Do the broader personality dimensions of neuroticism, extraversion, openness and agreeableness show a relationship with prevalent cardiovascular diseases such as

myocardial infarction, peripheral arterial disease or angina?

(4) Does the low pole of agreeableness relate to increased disease prevalence, and if so, is this a suitable standard way to measure hostility?

(5) Do higher anger scores, especially expressed anger, as would be expected from previous research, correlate with a higher prevalence of CHD, particularly myocardial infarction (MI)?

(6) Does neuroticism relate to increased prevalence of subjective events such as angina, and not to objective events such as MI?

(7) Are the observed associations independent of the effects of other risk factors, namely cigarette smoking, hypercholesterolaemia, hypertension, and rheological and haemostatic factors?

(8) What factors work in combination to increase or decrease the risk of CHD?

In sum, the aim of this study is to determine the personality profiles of a random sample of older people in the general population, who have had their health status monitored over a period of eight years, on the PDS, the NEO-FFI and the STAXI. The personality scores will then be examined statistically in relation to cardiovascular disease incidence and prevalence, and will be adjusted for established CHD risk factors. The subjective outcomes such as angina will be clearly separated from objective outcomes such as MI, so that the distinction between disease and distress is clear. This will help to advance our knowledge about personality factors that contribute to a person's risk of developing cardiovascular disease.

CHAPTER 6

Methods I: Edinburgh Artery Study from baseline to 5 year follow-up

6.1 INTRODUCTION

This chapter describes the methods of the baseline Edinburgh Artery Study (EAS) survey and the follow-up procedures over the subsequent five years, including the second medical examination. The chapter also explains the data analysis of the Personality Deviance Scales (PDS; Bedford and Foulds, 1978) and incident CHD. Shorter descriptions of the study methods for the baseline and follow-up have been published in papers reporting different results of the Edinburgh Artery Study (eg. Fowkes et al, 1991; 1992; Leng et al, 1996). The methods are fully outlined here as they are relevant to the analyses presented in chapter seven.

6.2 CROSS-SECTIONAL SURVEY

6.2.1 Study design

The first part of the Edinburgh Artery Study was a cross-sectional survey designed to measure the prevalence of peripheral arterial disease and its risk factors in the general population (see Appendix Y for a diagram of study design). The target population was Edinburgh residents aged 55-74 years. Participants were selected by age stratified random sampling from the age-sex registers of ten Edinburgh general practices serving the whole spectrum of socioeconomic areas in all parts of the city. The required sample size was calculated as 1500, enough to compare diseased and normal participants in relation to baseline characteristics in

the planned cohort study. Participants were randomly selected in sex-specific, five-year age groups to generate equal numbers in each age group (34 males and females in each group; 272 from each practice) . Those judged by the general practitioner to be unfit to participate (eg. through mental illness or terminal disease) were replaced by other randomly sampled patients (n=353; 13%).

Invitation letters to attend a university clinic for a medical examination, signed by the study director and by a partner in the relevant general practice, were sent out following publicity in the local media. Provision of transport or examination at home were available for any who would find it difficult to attend the clinic, and travelling expenses were also offered. Those whose invitation letters were returned by the post office were replaced by other randomly sampled subjects (n=163; 6%). Once an affirmative reply was received, each subject was send an appointment date, a map, and details about the examination. Those who did not respond at all were sent a second invitation letter. Affirmative responders who did not attend their appointment were offered second appointment date, usually by telephone. The study was approved by the (then) Lothian Health Board Ethics Committee, and informed consent was obtained from each participant before the medical examination (see Appendix A for the ethical approval, invitation, appointment letters and consent form).

6.2.2 Examination

The examinations were held on weekday mornings from August 1987 to September 1988 at the Edinburgh University Student Health Service. Occasional

out-of-hours sessions were arranged as required. Ten participants were invited for each appointment day. The participants were asked to fast from 11pm the previous evening if not diabetic, and to refrain from smoking for two hours prior to the examination. Each of the subjects underwent two sets of clinical procedures carried out by one of two teams of a nurse and a technician (see Appendix B for the data recording forms). A self-administered questionnaire (Appendix C) was completed by each subject and then checked by a member of the clinic team.

Quality of the measures was checked during the study by the study co-ordinator, who intermittently took repeat measurements. Each of the clinic team's measures were examined for drift, and variability studies of other measures has been assessed (Fowkes et al.,1992).

6.2.3 Clinical measurements

In the first set of procedures, a 20 ml sample of venous blood was withdrawn for subsequent analysis of biochemical, haemostatic and rheological factors. Standing height, without shoes, was measured to the nearest 5 mm using a free-standing metal ruler on a heavy base. Weight, without outer clothing or shoes, was measured to the nearest 100g on a digital Soehnle scale. A 12-lead ECG was taken using a Hewlett Packard 'Pagewriter' electrocardiograph. The ECGs were later coded according to the Minnesota code (Prineas, Crow and Blackburn, 1982) independently by two trained researchers, and a third team member checked the results. In the event of a disparity, the ECG was coded again by the third person. If this code agreed with neither of the other two, the

ECG was referred to a consultant cardiologist and a code assigned following a discussion among the coders.

The second set of procedures began with a ten-minute rest in the supine position, followed by the measurement of systolic and diastolic (Phase V) blood pressures in the right arm, using a Hawksley random zero sphygmomanometer. The femoral, posterior tibial and dorsalis pedis arteries were then palpated in both legs. Systolic ankle pressures were taken first in the right leg, then the left, using the random zero sphygmomanometer and a Sonicaid Doppler probe. The reactive hyperaemia test followed, in which ankle systolic pressure was measured in the right and left legs 15 seconds after the release of an above-the-knee cuff, which had occluded arterial flow for four minutes, at about 50 mm Hg above systolic pressure. This procedure was timed with an electronic timer.

6.2.4 Questionnaire

The self-administered questionnaire consisted of items regarding medical history, symptoms, smoking habits, alcohol consumption, diet and personal characteristics such as occupation and marital status (Appendix C). Medical history items addressed recall of a doctor's diagnosis of angina or myocardial infarction. Chest pain and leg pain symptoms were assessed by the WHO angina and intermittent claudication questionnaires (Rose, 1962). Smoking habits were self-reported and the information was converted into packyears for each person, obtained by calculating the number of 20-cigarette packs per day smoked, multiplied by number of years as a smoker. To obtain a measure of units of

alcohol consumed per week, participants were asked to record their alcohol intake over the past week by indicating the number of drinks they had consumed in each of three categories: beer (including later or cider), wine (including sherry, martini and fortified wine), and spirits (e.g. whisky, gin etc.). One unit of alcohol was defined as a half a pint of beer, one glass of wine or a single measure of spirits. They were also asked whether this represented a typical week's consumption, and if not, whether they usually drank more or less. These measures were judged to be sufficiently accurate because the self-reported levels correlated with the mean thiocyanate concentrations and gammaglutamyl transferase activities in the blood (Fowkes et al, 1992). Social classes I-V were later assigned according to the Registrar General's classification (OPCS, 1980): married women were classified by their husband's occupation, and the retired and unemployed were classified by their longest-held occupation. A deprivation score was also assigned to each participant, based on the postcode district classification from the 1981 census (Carstairs and Morris, 1989).

6.2.5 Personality measurement

The Bedford-Foulds Personality Deviance Scales (Bedford and Foulds, 1978; PDS) were administered as part of the whole questionnaire (Appendix D). They were originally designed to elicit three main traits: extrapunitiveness, by measurement on two primary scales, hostile thoughts and denigratory attitude towards others; intropunitiveness, by measuring lack of self-confidence and dependence on others; and dominance, by measuring hostile acts and a

domineering attitude. A series of 36 questions was answered, with six questions for each primary scale. Each question had four possible responses: very often (scoring 4), often (3), seldom (2) and never (1) (reverse scored when appropriate). The range of possible scores for each primary scale was therefore 6-24, and for each main scale, 12-48. The instructions to the questionnaire specifically asked participants to "choose the ***one*** which best describes you ***for most of your life***" (emphasis in original). This helps to ensure that the scales represent trait rather than state measures.

Two revised scales were later derived from item-level factor analysis of the PDS carried out on the Edinburgh Artery Study participants' questionnaires: hostility and submissiveness/low self confidence (Deary, Bedford and Fowkes, 1995). These revised scales were statistically independent of one another (orthogonal). The revised hostility scale was calculated from items originally assessing hostile acts and hostile thoughts, and was based on 8 questions, allowing a range of 8-32. The submissiveness/low self confidence scale (hereafter referred to as submissiveness) was based on 9 questions drawn from a combination of the domineering attitude and lack of self confidence scales, and the range of scores was therefore 9-36. The questions that make up these two scales are shown in Appendix E.

The original scales were found to be valid and reproducible (Bedford and Foulds, 1978), although they had never been compared with other widely used measures of hostility such as the Cook-Medley hostility scale (Cook and Medley, 1954) of the Minnesota Multiphasic Personality Inventory (Deary et al., 1994).

The 1995 factor analysis, however, was the first report of the item level factor structure of the PDS questionnaire, and the resulting solution of two main scales is psychometrically sound. Therefore, analysis using the two revised scales will be particularly useful, because reliable, validated scales are important for health and personality research, and use of the original primary scales is also warranted if narrower traits are of interest (Deary, Bedford and Fowkes, 1995). Thus, the two sets of scales are used in the current analysis because both wide and narrow traits are of interest.

6.3 FIVE-YEAR FOLLOW-UP

Following the baseline examination, the participants were followed up to detect cardiovascular events. At the end of a five-year period, they were invited to attend a second medical examination.

6.3.1 Identification of cardiovascular events

Information about the following cardiovascular events was obtained during the follow-up period: myocardial infarction, angina and intermittent claudication. Data on stroke, transient ischaemic attacks, critical limb ischaemia, thrombo-embolism, vascular surgery, angioplasty and coronary artery bypass grafting were also collected, but these definitions are not included below because these events were not used as endpoints in this study (see Appendix F for the study guideline sheet and event recording forms). Criteria to define the events were adapted from the American Heart Association (Gillum et al., 1983), and an event was recorded

only if the following criteria were fulfilled :

Myocardial infarction

1. *Non fatal*

a. Definite myocardial infarction was coded in two of the following three criteria were fulfilled:

i. presence of prolonged (>20 min) cardiac pain, anywhere in anterior chest, left arm or jaw (possibly also involving back, shoulder, right arm or abdomen) and lasting at least 20 minutes.

ii. Diagnostic ECG codes: Minnesota codes (Prineas, Crow and Blackburn, 1982) 1.1.1-1.2.5; 1.2.7; or 9.2 plus 5.1 or 5.2.

iii. Elevated serum enzyme levels: creatine phosphokinase greater than twice the upper limits of normal, and one of the following also greater than twice the upper limits of normal: lactate dehydrogenase, glutamic oxalo-acetic transaminase, or the MB isoinzyme of creatine phosphokinase. The enzymes must have been measured within 72 hours of an acute event.

b. Possible myocardial infarction was coded if:

i. One of the above criteria was present, plus either:

aa. equivocal ECG codes: 1.2.8-1.3.6; 4.1-4.3; 5.1-5.3; or 9.2.

bb. equivocal enzyme levels: above normal but not twice normal, or one was above twice normal but could be

attributed to another cause.

ii. Equivocal ECG codes and equivocal enzyme levels

2. *Fatal*

a. coded if there was a post mortem finding of acute myocardial infarction,

OR

b. criteria for definite myocardial infarction were met in the four weeks prior to death,

OR

c. ICD-9 (International Classification of Diseases, version 9) death certificate codes were 410-414 plus there was a history of definite or possible myocardial infarction; or 410-414 plus criteria for possible myocardial infarction immediately preceding death; or ICD codes 410-414 plus post mortem evidence of severe coronary atherosclerosis or previous myocardial infarction

d. Possible fatal myocardial infarction was coded if death certificate codes were 410-414 but no other evidence of myocardial infarction could be found.

3. *Silent*

Coded if ECG Minnesota codes were 1.1.1-1.2.5; 1.2.7; or 9.2 plus 5.1 or 5.2, in the absence of elevated enzymes levels or cardiac pain, and the ECG at baseline was coded as normal

Angina pectoris

Angina was recorded if there was no evidence of angina on the WHO questionnaire (Rose, 1962) at baseline, plus one of the following:

a. WHO angina questionnaire during follow-up was positive plus recall of a doctor's diagnosis of angina,

OR

b. WHO angina questionnaire positive plus diagnostic ECG codes

OR

c. Clinical diagnosis of angina investigated by the general practitioner or in hospital

Intermittent claudication

Intermittent claudication was diagnosed using the WHO Questionnaire (Rose, 1962). Grade 1 was recorded if calf pain occurred walking uphill or hurrying, and grade 2 if the pain also occurred while walking at ordinary pace on the level. 'Probable' claudication was defined as calf pain present on exercise but not at rest that otherwise did not fully meet the WHO criteria.

The data on the cardiovascular events were ascertained in a number of different ways:

1. *General practitioners*

At the start of the study, a card was prepared with the participant's details, which was then attached, together with a business reply envelope addressed to the

Edinburgh Artery Study, to the front of the subject's records (Appendix G). This card was to be returned following a cardiovascular event, a change of address or death. The card was also returned if the patient left the practice, in which case the subject was traced through the Primary Care Division of Lothian Health.

2. *National Health Service Central Registry (NHSCR)*

Each participant's record was flagged at the NHSCR to ensure that death certificates would be automatically forwarded for deaths occurring within the UK. All deaths recorded as cardiovascular on the death certificate were further investigated using hospital or general practitioner records to determine if the study criteria were fulfilled.

3. *Information and Statistics Division of the Scottish Office Home and Health Department (ISD)*

ISD provided annual computer printouts of all hospital discharges in Scotland for the previous year. In the first instance, *all* discharges with ICD-9 (International Classification of Diseases, 9th revision) codes 410-414; 430-438; 440-444; 785; 250.6; 342, 344.3, 344.4; 781.2, 781.4; 784.3, 784.5; 798, 798.1, 798.2, 798.9 were provided and the list was checked to identify Edinburgh Artery Study participants. Following a systems change in 1991 it became possible to link the Edinburgh Artery Study participant list to the database of hospital discharges. Printouts thereafter were listed by participant name rather than by ICD code. These were matched with the ISD file on name, date of birth, sex and

Edinburgh Artery Study members. In the former case, once the patients were identified, their records were obtained at the relevant hospital to check the details of admission and progress, and only if study criteria were fulfilled was an event recorded. In the latter case, those discharges with relevant ICD codes were identified, and then the records were followed up at the hospitals. Hospital notes were reviewed and events were coded by the study co-ordinators.

4. *Royal Infirmary of Edinburgh*

Lists of new referrals to the peripheral vascular clinic and lists of vascular operations coded according to a system designed by the Surgical Audit Committee (Surgical Audit Committee Lothian-Scotland, 1988) were supplied by the Royal Infirmary. These were checked by the study co-ordinator to identify EAS participants and follow up of records was carried out as required.

5. *Annual questionnaires to participants*

Each study member was sent an annual questionnaire enquiring about the development of the following symptoms or disease in the previous year: heart attack, stroke, chest and leg pain, loss of power in arms or legs, and hardening of the arteries (Appendix H). Participants were also asked about hospital admissions and attendances, and visits to their general practitioner. Follow-up depended on the nature of the replies and included sending a WHO angina or intermittent claudication questionnaire, contacting the general practitioner or investigating hospital records to check the criteria for events.

6.3.2 Five-year follow-up examination

6.3.2.1 Invitation procedure

After five years of follow-up, participants were invited to a second medical examination (Appendix I). They were offered the opportunity to be examined at home if they were not able or did not wish to attend the clinic. Study members who had moved outside the area were offered overnight accommodation and travelling expenses, but not home visits. Those who did not wish to attend, who missed their appointments or who had not responded to three invitations were sent the same questionnaire given to attenders. If the questionnaire was not returned, they were telephoned. If they were not contactable by telephone, they were visited at home and asked to complete the questionnaire. Participants whose letters were returned by the post office were traced through the Primary Care Division of Lothian Health Board, and the invitation procedure was repeated.

6.3.2.2 Medical examination

The examinations were performed by three specially trained nurses and were held between November 1992 and March 1994 at the Vascular Studies Unit in the Royal Infirmary of Edinburgh (see Appendix J for consent and recording forms). Participants were asked to complete a self-administered questionnaire (Appendix K) which included the annual questions about cardiovascular events, plus marital status, employment status, the WHO angina and intermittent claudication questionnaires (Rose, 1962), an update of their smoking habits, their medications (including aspirin) and age at menopause. After a rest of 5 minutes,

blood pressures were measured in the right arm using the same Hawksley random zero sphygmomanometer that had been used at baseline. The femoral, posterior tibial and dorsalis pedis arteries were then palpated in both legs. Ankle pressures were measured using a Sonicaid Doppler ultrasound probe. Height and weight were measured in the same way as at baseline, and on the same equipment. Ultrasound scans of the abdomen and neck were then performed to detect aortic aneurysm and carotid artery disease. Carotid scanning was performed using an ATL UM9, HDI Duplex Scanner (Bothwell). The carotid arteries were examined first in a transverse plane and then longitudinally. A note was taken as to which segments were identifiable on each side (internal, external, common carotid and bulb). A measurement of intima-media thickness was made at the far wall of the common carotid artery, 2 cm proximal to the bifurcation. Once the correct point was located, the magnified image of the far wall of the common carotid artery was frozen on the display screen of the unltrasound scanner. Two cursors were positioned on the boundaries of the intima-media, and the distance between them was recorded to the nearest 0.1mm and taken as the intima-media thickness. All the scan images were videotaped and those in which measurements were not obtained but suggested significant disease were reviewed by the consultant radiologist and a member of the study team. A decision was then made as to whether the participant should be re-scanned by the consultant. At least one of the variables recorded was later amended by the consultant in forty-one participants.

A 12-lead ECG was recorded using the Hewlett Packard portable

'pagewriter.' The ECGs were coded using the Minnesota coding system (Prineas, Crow and Blackburn, 1982) by two independent researchers. In the case of discrepancy, the final decision was made by a consultant cardiologist. A 30 ml sample of blood was also taken for later analysis of haemostatic, rheological and genetic factors.

6.4 DATA ANALYSIS

Data on the questionnaires and recording forms from the examinations were checked by the clinic staff, coded and entered onto a DBASE III database at examination one, or onto a DBASE IV database at examination two. All forms were entered dually to control error rates, and discrepancies were checked by referring to original records. The data were then transferred to the Edinburgh University mainframe computer for analysis. The Personality Deviance Scales (PDS) were coded by the research staff, and the data were then entered directly onto the University mainframe computer and validated by the University Data Preparation Services.

The data were analysed using the statistical packages SPSS-X (SPSS Reference Guide, 1990) and SAS (SAS/STAT User's Guide, 1988). Descriptive statistics were calculated for the PDS (original and revised scales) and the 5-year follow-up cardiovascular data in men and women. Further analyses were carried out separately in men and women because of the differences in both personality trait levels and cardiovascular data. The distributions of the personality scores were examined for normality. For the remaining analyses, those with a history of

angina or MI at baseline (142 men, 89 women) were excluded. Missing values were deleted on a pairwise basis. The significance of differences in the event and non-event groups in both men and women was then assessed using Student's t-test. Outcome categories were defined as (a) total myocardial infarction (MI), which included nonfatal and fatal MI's; (b) fatal MI, (c) nonfatal MI (both nonsilent and silent); and (d) new angina pectoris (see above for definitions). The personality scales were then entered into multiple logistic regression equations (backward stepwise elimination; criteria for removal, $p \leq 0.05$, were based on the likelihood ratio test) for each outcome category as the dependent variable. The independent variables were age, degree of baseline vascular disease (measured using the ankle-brachial pressure index), and the baseline risk factors: social class, systolic and diastolic blood pressure, total serum cholesterol (and HDL-cholesterol), triglycerides, body mass index, smoking and alcohol consumption. Square root transformations of the packyear and alcohol consumption data were used because their distributions were significantly skewed. The original and revised PDS scales were kept in separate equations to avoid redundancy in the personality variables.

The relationship between extent of vascular disease and risk factors for CHD was also investigated. Of particular interest was the association between ankle-brachial pressure index (ABPI), which is an indicator of extent of peripheral atherosclerosis (Fowkes et al, 1991) and personality, especially given the previous finding in the study of a positive relationship between intermittent claudication and a higher hostile acts score in men (Deary et al, 1994). Pearson correlation coefficients were calculated between each of the Bedford-Foulds personality scales

and measures of ABPI at the follow-up examination, and two variables estimating change in ABPI over the five years. One of these was a calculation of the first measure subtracted from the second. The second measure of change was calculated by regressing the second measure on the first, and saving the residuals as a measure of change. Pearson correlation coefficients were then generated between the personality variables and the two measures of change in ABPI. Those personality scales showing the strongest correlations with the follow-up ABPI measures and measures of change were used as independent variables in subsequent multiple linear regression models, along with the covariates of baseline risk factors: age, social class, social deprivation, HDL and total serum cholesterol, triglycerides, body mass index, smoking and alcohol consumption. Systolic and diastolic blood pressure were not included as covariates as the arm pressure forms part of the fraction of ABPI. Backward stepwise multiple linear regression analysis was carried out, with criteria for entry set at $p<0.05$, and for removal at $p<0.06$.

Finally, prospective relationships between personality and the risk factors for CHD of blood pressure, smoking and body mass index were examined. Again, Pearson correlations were calculated, then backward stepwise multiple linear regression models were generated, using the same covariates as above (except where blood pressure, ABPI or BMI was the dependent variable), and using the same entry and removal criteria as previously.

CHAPTER 7

Methods II: Procedures, Coding and Analysis of Data on Core Personality Traits

7.1 INTRODUCTION

This chapter reviews the methods of the administration of the NEO-Five Factor Inventory (NEO-FFI; Costa and McCrae, 1992) and the State-Trait Anger Expression Inventory (STAXI; Spielberger, 1989). The procedures for the data analysis of prevalent cardiovascular disease and the NEO-FFI and STAXI are also described.

7.2 STUDY POPULATION

The study population comprised all members of the Edinburgh Artery Study cohort who were still participating in the study in April, 1995 (aged 62-81 years at that time). There were 269 deaths and 27 who had dropped out because they were unable or no longer wished to participate, leaving a sample size of 1296. All these participants were sent a personality questionnaire along with their annual medical questionnaire (see Appendix Y for diagram of study design).

7.3 METHODS

7.3.1 Administration procedure

Ethical approval to administer the personality questionnaires was granted by Lothian Health Medicine and Clinical Oncology Ethics Sub-Committee (Appendix L). A general update about the study was sent to the general practices and information about the planned administration of the personality questionnaires

was included (Appendix M). The general practitioners were encouraged to contact us if they had any queries or reservations about sending the personality questionnaires to their patients who were Edinburgh Artery Study participants. We were not contacted by any of them.

Personality questionnaires were sent out in batches by general practice (Appendix N). They were sent with a letter informing the participants about the questionnaires and asking if they would fill them in and return them, together with their annual medical questionnaire, in a prepaid envelope (Appendix O). An information/update sheet about the study generally was also sent with the questionnaires (Appendix P). If there was no reply after 6 weeks, a reminder letter was sent. If there was no reply following this, a second questionnaire was sent out with a repeat request to fill it in. Questionnaires that were returned with a complete medical section but omitting the personality questionnaires were taken to be refusers and not contacted again. Participants whose letters were returned by the post office were traced through the Primary Care Division of Lothian Health, where possible, although 16 could still not be found by the end of the administration period. The 1996 follow-up letter included thanks for filling in the personality questionnaires the year before (Appendix Q).

7.3.2 Measurements

7.3.2.1 The NEO Five-Factor Inventory (NEO-FFI)

The NEO-FFI is a shorter form (60 statements) of the NEO Personality Inventory- Revised (NEO-PI-R) which assesses neuroticism (N), extraversion (E),

openness (O), agreeableness (A) and conscientiousness (C) (Costa and McCrae, 1992); Appendix N; pp. N-2-N-5).. Correlations between the short and full versions were 0.92, 0.90, 0.91, 0.77 and 0.87 for N, E, O, A and C, respectively (Costa and McCrae, 1992). Internal consistency for the NEO-FFI scales were calculated using coefficient alpha and were 0.86 (N), 0.77 (E), 0.73 (O), 0.68 (A) and 0.81 (C); slighly smaller than those for NEO-PI-R (Costa and McCrae, 1992). Correlations between the NEO-FFI self-reports and the person's spouse's rating of them on the NEO-PI-R also correlate well: 0.93 (N), 0.90 (E), 0.94 (O), 0.88 (A); and 0.89 (C). The shorter scales of the NEO-FFI appear to account for approximately 15% less of the variance in measures of convergent validity than the full scales, and therefore some precision is lost (Costa and McCrae, 1992). For the EAS participants, the convenience of the 60-item version was preferred to the possible irritation or fatigue that may have been caused by administration of the 240-item scale.

The five domains are: (1) neuroticism, which measures the tendency to experience negative emotions and to have corresponding upsets in both thinking and actions (Vestre, 1984); (2) extraversion, or a tendency to prefer company and social interaction over solitude, and a liking for spirited, lively activity; (3) openness, a willingess to entertain new ideas or experiences, such an openness to trying foreign food or exotic holidays; (4) agreeableness, which assesses the tendency to be helpful rather than competitive, and also reflects a generous, concerned and trusting nature; and (5) conscientiousness, or the tendency to be purposeful, strong-willed, dutiful, self-disciplined or ambitious (Costa and McCrae,

1987, 1992). Subjects are asked note their level of agreement with the statements: strongly disagree, disagree, neutral, agree, or strongly agree. An example of one of the statements, from the extraversion scale, is "I like to have a lot of people around me." There are 12 statements pertaining to each domain, and the items are scored from 0-4, leaving a possible range of 0-48 for each of the five factors.

Instructions for scoring dictate that if ten or more items have been left blank, then the whole test is invalid and should not be formally scored. If nine or fewer items are blank, then the blank items are scored as if the neutral response had been selected. However, if 4 or more items are missing from any one domain scale, the scores need interpreting with caution (Costa and McCrae, 1992). Scale scores in this sample were not calculated where there were missing values. There are normally three questions, to check validity of the questionnaire, following the end of the scale statements. These are: "Have you responded to all the statements?"; "Have you entered your responses in the correct boxes?"; and "Have you responded accurately and honestly?" In the Edinburgh Artery Study we were given permission by the publishers (Appendix R) to slightly alter the format of the NEO-FFI to suit the sample. We provided boxes to tick immediately to the right of the statements rather than using a separate answer booklet. We also altered the end statement to read "please check that you have responded to all the statements" (p. N-5). The ticking of boxes provided continuity format-wise with our own annual questionnaire. Given that the sample also had been cooperative and was still participating nearly 10 years after recruitment, and providing accurate medical data, the validity check questions seemed inappropriate. These

questions would have invalidated some questionnaires if the respondent indicated he or she had not answered honestly. The extremely small number of deliberately dishonest questionnaires that may possibly be included is unlikely to bias our results. The benefit of maximizing our response rate and retaining our participants outweighed the cost of possibly including a rogue questionnaire.

7.3.2.2 State-Trait Anger Expression Inventory (STAXI)

The STAXI (Spielberger, 1989) consists of 44 items to allow measurement of the experience and expression of anger (Appendix N; pp. N-5-N-7). The state anger section (10 items) was omitted for the purposes of the EAS (with permission from the publishers, see Appendix R), as it was not relevant to the analysis and would have lengthened the amount of time needed to fill in the questionnaire. The remaining trait section contains several scales:

(1) A *trait anger* scale (10 items), which also contains two subscales:

(a) *angry temperament* (4 items), or a general propensity to experience anger (not necessarily with specific provocation), and

(b) *angry-reaction* (4 items), which measures anger as a response to criticism or unfairness (Spielberger, 1989).

(2) There are three scales measuring

(a) *anger-out* (8 items), which is the expression of anger toward other people or things;

(b) *anger-in* (8 items), or the suppression of angry feelings, and

(c) *anger-control* (8 items), the extent to which people try to

control their angry feelings.

(3) The remaining scale is calculated from the anger-out, anger-in and anger-control scales. It results in an anger expression scale, which gives a general index of how often anger is expressed, regardless of the mode of expression.

For all questions, the respondent replies to a statement on a four-point scale, from almost never (scoring 1) to almost always (scoring 4). For instance, one statement is "I have a fiery temper." Scale scores were not calculated where there were missing values.

Correlations of the trait-anger scale with the total score from the Buss-Durkee Hostility Inventory ranged from 0.66 to 0.73, and with the Ho scale of the MMPI from 0.43 to 0.59, thus demonstrating concurrent validity (Spielberger, 1989). The anger expression scales correlate reasonably well with responses to Harburg , Blakelock and Roeper's (1979) "Teacher" and "Movie" vignettes describing anger-provoking situations (ranging from -0.26 between 'movie queue' and anger-in to 0.49 between 'angry teacher' and angry expression). Evidence of divergent validity was also present, as the correlations between a 'curiosity' scale and the anger scales were essentially zero. Test-retest reliability has been shown (Jacobs et al, 1988). The scale has been used successfully in many areas of health research, including investigations of blood pressure (Harburg and Hauenstein, 1980) and coronary heart disease (Tennant et al., 1987.)

7.3 DATA ANALYSIS

The questionnaires were entered directly onto disk by the University Data Preparation Services. They were then keyed in for a second time and discrepancies were checked with the original questionnaires. The data were transferred into SPSS-X and SAS files for analysis with the EAS data.

Mean levels of NEO-FFI and STAXI scale scores for men and women were calculated, and their distributions checked for normality. Logarithmic transformations of the anger-out, anger expression, and angry-reaction scales were used because the distributions were skewed. All remaining analyses were conducted in men and women separately because of the differences in cardiovascular disease and personality scores apparent between the sexes.

To obtain a measure of 'prevalent' blood pressure, the mean of the baseline and follow up measures of both systolic and diastolic measures was calculated. This value was used in all analyses of the NEO-FFI and STAXI data. Smoking information was converted into packyears, which were calculated using the updated information from the follow-up questionnaire. A square-root transformation was used in analyses, given the skewed distribution of the data. Logarithmic transformation was used for the follow-up intima-media thickness (IMT) measures, which assess the extent of atherosclerosis in the carotid arteries.

Student's t-test was used to explore the mean levels of each personality variable in different disease categories: (1) All prevalent cardiovascular disease (CVD); (2) prevalent CHD: myocardial infarction (MI), including nonfatal, nonsilent and nonfatal, silent; and angina; (4) all prevalent peripheral arterial

disease (PAD), including asymptomatic disease; and (5) prevalent intermittent claudication. Cutoff dates for prevalent events were set at the five-year follow-up. Disease categories were defined as follows:

(1) All prevalent vascular disease. Included any evidence of vascular disease at baseline, during follow-up or at the five-year follow-up, and therefore included history of MI or angina at baseline, history of intermittent claudication at baseline or diagnosed during follow-up, major or minor asymptomatic peripheral arterial disease diagnosed at baseline or during follow-up, nonfatal (nonsilent and silent) MI or new angina diagnosed during or at five-year follow-up (see previous chapter for definitions).

(2) Prevalent CHD. MI: nonfatal MI, both definite and possible, and both nonsilent and silent MI. Angina pectoris: included those with angina at baseline or meeting criteria for new angina over five-year follow-up.

(3) All prevalent PAD. Included intermittent claudication, major and minor asymptomatic PAD, either at baseline or during follow-up.

(4) Prevalent intermittent claudication only.

Multiple logistic regression models were then generated for each of the prevalent disease categories and the personality variables. Forward stepwise regression was used because it allowed the anger variables to be entered one at a time. Criteria for entry were set at $p \leq 0.05$, and criterial for removal were set at $p \leq 0.06$. In order to obtain an odds ratio for a one standard deviation increase in a personality scale, the personality variable used in the equation was the scale score divided by its sex-appropriate standard deviation. Covariates in the models were

age, social class, social deprivation, total serum and HDL-cholesterol, triglycerides, body mass index, ABPI, blood pressure, smoking and alcohol consumption (except where ABPI was the dependent variable). Updated information was not available for alcohol consumption, so the baseline values were used.

The personality variables were examined in relation to smoking, blood pressure, body mass index, cholesterol, ABPI and IMT measures. Univariate Pearson correlations were followed by analysis using stepwise multiple linear regression models. Criteria for entry and removal were the same as above. Covariates were also as above, except where ABPI, smoking, blood pressure or cholesterol were not covariates when analyzed as the dependent variables. These equations did not include measures of *disease* as covariates.

Lastly, the correlations between all the personality measures - the PDS, the NEO-FFI and the STAXI - were examined. This allowed us to get some idea of shared variance of the personality measures.

CHAPTER 8

Results I: Relationships between baseline personality and incident cardiovascular events

8.1 INTRODUCTION

The first step in the analysis was to determine the relationships between the Bedford-Foulds Personality Deviance Scale scores (PDS; Bedford and Foulds, 1978), which were administered at baseline, and the five-year incidence of cardiovascular events. The methods of the data analysis were described in chapter six (section 6.3).

The first two objectives of the thesis (see also chapter five) are addressed by the analyses presented in this chapter, as are objectives seven and eight:

(1) Are the hostility or dominance-related traits of the PDS associated with increased incidence of cardiovascular events?

(2) Do the PDS traits predict severity of subclinical disease as measured by the ABPI?

(7) Are the observed associations independent of the effects of other risk factors, namely cigarette smoking, hypercholesterolaemia, hypertension, and rheological and haemostatic factors?

(8) What factors work in combination to increase or decrease the risk of CHD?

In this chapter, the response rates and descriptive data will be presented first. Univariate relationships between the PDS variables, incident events and risk factors are presented second, and finally, the multivariate analyses will be described.

8.2 RESPONSE RATES

The Edinburgh Artery Study sample was fairly representative of the target population, although women in the 70-74 age group and men in the 55-59 age group were slightly under-represented (21.3% and 22.5% instead of 25%). Social classes IV and V made up 13% of the respondents, which is lower than the estimated 19% in the Edinburgh population according to the 1981 census.

8.3 CUMULATIVE INCIDENCE OF CARDIOVASCULAR EVENTS

The cumulative incidence of events over 5 years of follow-up is shown in Table 8.1 (tables begin p. 176). Nearly 10% (n=80) of men and 4.3% (n=34) women experienced a myocardial infarction of some kind. Angina and intermittent claudication showed similar rates in both sexes, with 5.9% (n=48) men and 5.2% (n=41) women having new angina, and 4.2% (n=34) men and 4.7% (n=37) women experiencing new intermittent claudication over the five years. Men died of noncardiovascular causes at nearly twice the rate of women, 11.9% (96) compared with 6.6% (52).

8.4 PERSONALITY SCORES

8.4.1 PDS scores in men and women

In Table 8.2, the means and standard deviations are shown for the PDS in men and women. Men scored higher on the dominance (29.6 v. 27.8), hostile acts (14.3 v. 13.5) and domineering attitude (15.3 v. 14.3) scales, but lower on the submissiveness (18.8 v. 20.6) and lack of self confidence scales (11.2 v. 12.5). The

distributions of the PDS scores approximated the normal curve and none required transformation (Appendix S, figures s1-s9).

8.4.2 Original and revised PDS dimensions

Because the revised PDS measures of hostility and submissiveness are psychometrically sounder (Deary, Bedford and Fowkes, 1995) than the original scales, greater weight can be assigned to results using these scales. Their correlations with the original scales are shown in Table 8.3. Submissiveness was highly positively correlated with intropunitiveness ($r=0.79$; $p\leq0.01$) and negatively with dominance ($r=-0.69$; $p\leq0.01$). New hostility was positively correlated with both hostile thoughts ($r=0.87$; $p\leq0.01$) and hostile acts ($r=0.68$; $p\leq0.01$).

8.5 BASELINE RISK FACTORS

The majority of risk factors were normally distributed (Appendix T, figures t1-t7), but the smoking and alcohol data were skewed, and were therefore adjusted using square-root transformations (figures t8-t11). Mean cholesterol levels (7.03 mmol/l) were slightly higher than reported in other Scottish studies (6.34 mmol/l; Hargreaves et al, 1991).

There were 69.2% (560) men who had accumulated more than one packyear, and of these, the median number of packyears was 30. In women, the median number of packyears was 20, in the 49.2% (385) who had accumulated more than one packyear. Smoking was more common in men than women, and the younger women tended to smoke more than older women. Alcohol consumption was lower

in women than in men: 58.5% (458) women drank one unit or more per week, and of these, the median units per week was 4. In men, 76.8% (621) drank one or more units per week, and they consumed a median of 11 units per week.

8.6 UNIVARIATE ANALYSIS

Univariate associations were carried out first in order to explore the direction and nature of the relationships between the personality factors and incident disease. There were no statistically significant differences on the revised PDS between those who died of a noncardiovascular event and those who did not. In men, for instance, those who died from a noncardiovascular event had a mean score of 18.8 on submissiveness, and those who did not also had a mean score of 18.8 (p=0.69). The respective scores for submissiveness in women were 20.4 and 20.6 (p=0.58). For hostility, men who died from a noncardiovascular event had a mean score of 17.5, and so those who did not (p=0.48); and the scores for women were 17.0 and 17.4 (p=0.25). Therefore, no further analysis was performed on noncardiovascular deaths, and the remaining results are for the cardiovascular outcomes only.

8.6.1 PDS and Incident Disease categories

Mean levels of each of the personality variables across the disease groups (nonfatal, fatal and total MI, angina, and intermittent claudication) are shown in tables 8.4-8.6. In both men (table 8.4) and women (table 8.5), submissiveness scores were significantly higher in those avoiding a non-fatal MI compared to those who had a non-fatal MI. In men, for instance, the mean score in those who had had a

nonfatal MI (n=57) was 17.7, and in those who had not (n=611) it was 18.9 ($p \leq 0.05$). In women the corresponding values were 18.2 (n=28) and 20.8 (n=642; $p \leq 0.01$).

Both men and women who had a nonfatal MI had higher domineering attitude and dominance scores than those who did not. The mean dominance score in men (table 8.4) with a nonfatal MI was 31.0, whereas in men without, it was 29.4 ($p \leq 0.05$). This also was seen in the domineering attitude scores, which were 15.2 in men without a nonfatal MI, and 16.0 in men who had an MI ($p \leq 0.05$). In women, the differential was greater (table 8.5): the mean dominance score was 29.8 in those with a nonfatal MI, and 27.7 in those without ($p \leq 0.05$). The MI group had a mean domineering attitude score of 15.7, versus 14.2 in those without ($p \leq 0.05$). Women also showed differences in denigratory attitude, for both nonfatal and total MI. Mean scores in the event-free group were higher (11.8 versus 10.6; $p \leq 0.01$). The same direction of difference was seen in their extrapunitiveness scores. In the angina group, in men (table 8.4), dependence scores were higher (14.4 versus 13.6; $p \leq 0.05$), and in women (table 8.5), lack of self confidence scores were higher (13.7 versus 12.4; $p \leq 0.01$).

The patterns of the mean scores for intermittent claudication in men and women were similar to the CHD groups (Table 8.6). For instance, dominance in women was higher in the diseased group (29.0 v. 27.7). However, no statistically significant differences were apparent.

There are redundancies in the PDS. For instance, the submissiveness scale includes some items from the lack of self-confidence and domineering attitude scales, as shown in their correlations (table 8.3). There is also overlap between the MI

categories, as participants may appear in both the nonfatal and fatal MI groups, if they experienced a nonfatal MI first and a fatal MI later.

8.6.2 PDS and ABPI at follow-up and change in ABPI over 5 years

The PDS did not strongly correlate with levels of ABPI at follow-up in either men or women (table 8.7). However, in men, the crude differences between ABPI at baseline and follow-up (table 8.8) were statistically significantly, although not strongly, correlated with dominance ($r=-0.09$; $p\leq 0.05$), domineering attitude ($r=-0.09$; $p\leq 0.05$) and submissiveness ($r=0.10$; $p\leq 0.05$). That is, higher dominance and domineering attitude were associated with a worsening ABPI over five years, and submissiveness was correlated with an improved ABPI (table 8.8). Another method of obtaining a measure of change in ABPI over the five years was to regress the second measurement on the first and to save the residuals as a variable. This variable was then correlated with the risk factors (table 8.9). The residuals were significantly correlated with dominance (men: $r=-0.09$; $p\leq 0.05$; women: $r=-0.11$; $p\leq 0.05$) and hostile acts (men: $r=-0.09$; $p\leq 0.05$; women: $r=-0.12$; $p\leq 0.05$) in both sexes, and also with extrapunitiveness in women ($r=-0.09$, $p\leq 0.05$). All these correlations indicated that ***higher*** PDS scores were associated with a worsening ABPI, including the association between submissiveness and ABPI in women.

8.7 MULTIVARIATE ANALYSIS

Associations evident in the univariate analyses required testing in multivariate models, to allow for adjustment of confounding factors and to see how various

factors might be acting together to increase or reduce risk. Multivariate analysis can also help determine if some of the predictor variables are redundant. For the disease outcomes, multiple logistic regression was used, and for the continuous measures such as ABPI, multiple linear regression models were generated.

8.7.1 PDS scales and CHD outcomes

The multiple logistic models generated for the original PDS and covariates, using disease as the dependent variable, reinforced the value of risk indicators such as ABPI in the prediction of disease. Backward logistic regression was used in this part of the analysis, and the final models, including all the risk factors that emerged as independent predictors of disease, are shown in table 8.10. For the myocardial infarction outcomes a higher ABPI (indicating less disease) was very protective: for total MI in men the RR associated with a change of +0.1 in the ABPI was 0.21 (95% CI 0.07-0.59), and in women it was 0.09 (0.01- 0.55). This translates into a 41-93% decrease in risk for men, and a 45-99% decrease in risk for women associated with a higher ABPI. In women, a one standard deviation increase in denigratory attitude also appeared to be protective of myocardial infarction, both nonfatal (0.59; 0.40-0.86) and total (0.57; 0.40-0.81). In addition, in women, higher lack of self confidence scores were associated with a RR of 0.60 (0.40-0.90) for nonfatal MI. However, for fatal MI, the only independent predictor for men was ABPI (0.10; 0.02-0.50); and in women it was systolic blood pressure (1.04; 1.01-1.06).

For angina, the predictors were different: in men, the PDS primary scale of over-dependence was associated with increased risk (1.42; 1.05-1.93), as was smoking

(1.14; 1.02-1.26; Table 8.10). Higher levels of HDL-cholesterol were protective against angina in men, with a RR of 0.24 (0.08-0.70). For women, lack of self confidence and increased systolic pressure were both associated with an increase in risk (LSC 1.52; 1.09-2.11; Systolic 1.02; 1.00-1.03). The only predictor for the development of intermittent claudication over the five years was ABPI, with a 0.1 increase in the ratio associated with an approximately 100% decrease in risk, in both men and women (Men, RR 0.014; women, RR 0.009 - no table).

A second set of models were calculated for the revised PDS scales of hostility and submissiveness and disease outcomes, also using backward logistic regression. The final models are shown in Table 8.11. As with the original scales, ABPI also emerged as an independent predictor- a higher ABPI was associated with a decreased risk of total MI in both men and women (Men: RR 0.21; 0.07-0.59; Women: 0.11; 0.02-0.63). Hostility was not associated with risk at all, but submissiveness, in women only, was associated with a decreased risk of both nonfatal and total MI (nonfatal: 0.59; 0.40-0.85; total: 0.69; 0.27-0.96). Again, angina had a different 'risk profile,' with neither ABPI nor the revised scales emerging as independent predictors of risk. Only HDL-cholesterol and smoking were associated with angina in men, and systolic pressure in women. Neither of the revised PDS scales was associated with intermittent claudication in multivariate models.

8.7.2 PDS scales and risk factors/indicators of disease

The multiple linear regression models using follow-up ABPI, change in ABPI or residuals of ABPI as the dependent variables showed that age and smoking were

the most consistent predictors of a lower ABPI (Tables 8.12 and 8.13). However, in men (table 8.12), total and HDL cholesterol, and body mass index (BMI), added to the model for follow-up ABPI, and the five factors together could account for 5% of the variance in ABPI (adjusted R^2 0.05). In models 'adjusting' for systolic and diastolic blood pressure at baseline, these factors emerged as clearly important (data not shown); but as the brachial pressure forms part of the ratio of the ABPI, this seemed an inappropriate adjustment, and the remaining predictors in the models in any case remained largely unaffected. Deprivation score was the only variable left in the final model for change in ABPI, but on its own accounted for 1% of the variance in the change in ABPI (Table 8.12). The residual change in ABPI was predicted by smoking, HDL cholesterol and deprivation score, and these factors together accounted for slightly more of the variance (7%). For women (table 8.13), age and smoking were the only factors included in the final models. Age, together with smoking, with age providing a slightly stronger contribution, accounted for 14% of the variance in follow-up ABPI. Age alone was responsible for 6% of the variance in change in ABPI. Smoking alone accounted for 2% of the variance in the residual change in ABPI.

8.8 CHAPTER SUMMARY

The findings can now be applied to the objectives listed in the introduction to the chapter:

(1) Are the hostility or dominance-related traits of the PDS associated with increased incidence of cardiovascular events? Briefly, yes: In the univariate analysis there were

differences in mean levels of the PDS scores in the different CHD categories. The nonfatal MI groups, both men and women, had higher dominance and domineering attitude scores, and lower submissiveness scores. Women with MI had decreased denigratory attitude scores. Lack of self confidence (in women) and over-dependence (in men) were associated with increased incidence of angina.

(2) Do the PDS traits predict severity of subclinical disease as measured by the ABPI? Again, yes: Higher dominance and lower submissiveness (in men), and higher dominance and higher submissiveness (in women) were associated with a worsening ABPI over five years.

(7) Are the observed associations independent of the effects of other risk factors, namely cigarette smoking, hypercholesterolaemia, hypertension and rheological and haemostatic factors? Statistically, yes: in women, for instance, denigratory attitude was still exerting an independent effect on the risk of MI after adjustment for other factors, as was submissiveness; in both sexes neuroticism-related dimensions were associated with increased angina. However, other factors also had an effect on risk which was sometimes stronger than that of the personality variable. This was particularly true for the influence of ABPI on future risk.

(8) What factors work in combination to increase or decrease the risk of CHD? The personality variables were not the sole predictors of risk, as indicated above. They had an *additional* influence on risk of MI, which, although not confounded with other risk factors, was still acting in conjunction with them. The risk factors themselves also acted together. For instance, age, smoking and cholesterol levels all influenced the ABPI, which was itself an important indicator of risk.

There are numerous issues to consider in the interpretation of these findings, including how deaths, the age of the cohort, and methodological and statistical techniques may affect the results. These will be discussed in the first part of chapter ten. First, in chapter nine, the results of the cross-sectional analyses of the NEO-FFI and STAXI with prevalent disease will be presented.

Cumulative incidence of coronary heart disease and non cardiovascular deaths over five years of follow-up in men and women

	MEN (n=809)	WOMEN (n=783)
TOTAL MYOCARDIAL INFARCTION	9.9% (80)	4.3% (34)
NONFATAL	7.0% (57)	3.6% (28)
FATAL	3.1% (25)	1.0% (8)
ANGINA	5.9% (48)	5.2% (41)
CLAUDICATION	4.2% (34)	4.7% (37)
NON CARDIOVASCULAR DEATHS	11.9% (96)	6.6% (52)

A subject may appear in more than one category.
Those with baseline MI or angina (142 men, 89 women) are excluded.

Table 8.2

Means (s.d.) of Bedford-Foulds Personality Deviance Scales in men and women

	MEN n=806	**WOMEN** n=780
ORIGINAL SCALES		
Extrapunitiveness	25.4 (3.9)	24.1 (4.1)
Hostile thoughts	12.3 (2.4)	12.4 (2.6)
Denigratory attitude	13.1 (2.5)	11.8 (2.5)
Intropunitiveness	24.9 (3.9)	26.0 (4.5)
Lack of Self Confidence	11.2 (2.5)	12.5 (3.0)
Over-dependence	13.7 (2.3)	13.6 (2.5)
Dominance	29.6 (4.8)	27.8 (5.1)
Hostile acts	14.3 (2.8)	13.5 (2.8)
Domineering attitude	15.3 (3.0)	14.3 (3.2)
REVISED SCALES		
Hostility	17.5 (3.2)	17.3 (3.4)
Submissiveness	18.8 (3.7)	20.6 (4.3)

Table 8.3

Correlations between revised and original Bedford-Foulds Personality Deviance Scales

	Submissiveness	Hostility
Submissiveness	1.00	-0.07
Extrapunitiveness	0.02	0.71*
Hostile thoughts	0.09	0.87*
Denigratory attitude	-0.05	0.28*
Intropunitiveness	0.79*	0.05
Lacks self confidence	0.85*	0.03
Over-dependent	0.40*	0.05
Dominance	-0.69*	0.53*
Hostile acts	-0.36*	0.68*
Domineering attitude	-0.79*	0.24*

*$p \leq 0.01$

Table 8.4

Means (s.d.) of Bedford-Foulds Personality Deviance Scales by coronary heart disease category in men

	MYOCARDIAL INFARCTION						ANGINA	
	Nonfatal		Fatal		Total			
ORIGINAL SCALES	Yes n=57	No n=611	Yes n=25	No n=643	Yes n=80	No n=588	Yes n=48	No n=620
Extrapunitiveness	25.3 (4.3)	25.3 (3.9)	26.0 (3.8)	25.3 (3.9)	25.6 (4.1)	25.3 (3.9)	25.8 (4.3)	25.3 (3.9)
Hostile thoughts	12.4 (2.6)	12.2 (2.4)	12.3 (2.3)	12.2 (2.4)	12.4 (2.5)	12.2 (2.4)	12.3 (2.7)	12.2 (2.4)
Denigratory attitude	12.9 (2.3)	13.1 (2.5)	13.7 (2.0)	13.1 (2.5)	13.2 (2.2)	13.1 (2.3)	13.5 (2.8)	13.1 (2.4)
Intropunitiveness	24.7 (3.8)	24.9 (4.0)	26.0 (4.9)	24.9 (3.9)	25.1 (4.2)	24.9 (3.9)	25.9 (4.6)	24.8 (3.9)
Lack of self confidence	10.8 (2.4)	11.3 (2.6)	11.9 (3.1)	11.2 (2.5)	11.1 (2.7)	11.2 (2.5)	11.5 (3.1)	11.2 (2.5)
Over dependence	14.0 (2.3)	13.7 (2.3)	14.1 (2.7)	13.7 (2.3)	14.0 (2.4)	13.6 (2.3)	14.4 (2.2)	13.6 (2.3)*
Dominance	31.0 (4.0)	29.4 (5.0)*	28.9 (5.2)	29.6 (4.9)	30.3 (4.5)	29.4 (5.0)	30.4 (4.8)	29.5 (4.9)
Hostile acts	15.0 (2.7)	14.2 (2.8)	14.1 (2.5)	14.3 (2.8)	14.7 (2.6)	14.2 (2.8)	14.8 (3.1)	14.3 (2.8)
Domineering attitude	16.0 (2.7)	15.2 (3.0)*	14.8 (3.3)	15.3 (3.0)	15.6 (3.0)	15.2 (3.0)	15.6 (2.6)	15.2 (3.1)
REVISED SCALES								
Hostility	18.0 (3.5)	17.4 (3.2)	17.5 (3.2)	17.7 (3.3)	17.9 (3.4)	17.4 (3.2)	17.8 (3.5)	17.5 (3.2)
Submissivenes	17.7 (3.0)	18.9 (3.8)*	19.3 (3.4)	18.8 (3.8)	18.2 (3.3)	18.7 (3.8)	18.6 (3.7)	18.8 (3.8)

*p<0.05,**p<0.01 Subjects were excluded if all items not completed (leaving 774 men). Those with history of angina or MI at baseline are excluded (142 men); a subject may appear in more than one category.

Table 8.5

Means (s.d.) of Bedford-Foulds Personality Deviance Scales by coronary heart disease category in women

	MYOCARDIAL INFARCTION						ANGINA	
	Nonfatal		Fatal		Total			
ORIGINAL SCALES	Yes n=28	No n=642	Yes n=8	No n=662	Yes n=34	No n=636	Yes n=41	No n=629
Extrapunitiveness	22.2 (3.9)	24.1 (4.1)*	23.3 (4.1)	24.1 (4.1)	22.3 (4.0)	24.1 (4.1)**	24.0 (4.3)	24.1 (4.1)
Hostile thoughts	11.6 (2.7)	12.4 (2.6)	12.3 (2.8)	12.3 (2.6)	11.6 (2.7)	12.4 (2.6)	12.3 (2.7)	12.3 (2.6)
Denigratory attitude	10.6 (2.2)	11.8 (2.5)*	11.0 (1.7)	11.8 (2.5)	10.6 (2.1)	11.8 (2.5)**	11.6 (2.5)	11.7 (2.5)
Intropunitiveness	24.1 (4.4)	26.1 (4.5)*	27.8 (4.4)	26.0 (4.5)	24.9 (4.4)	26.1 (4.5)	27.3 (4.9)	26.0 (4.4)
Lack of self confidence	10.8 (2.7)	12.5 (3.0)**	13.4 (2.9)	12.4 (3.0)	11.3 (2.7)	12.5 (3.0)*	13.7 (3.0)	12.4 (2.9)**
Over dependence	13.3 (2.5)	13.6 (2.5)	14.4 (2.4)	13.6 (2.5)	13.6 (2.5)	13.6 (2.5)	13.6 (2.9)	13.6 (2.5)
Dominance	29.8 (5.1)	27.7 (5.1)*	25.9 (2.7)	27.8 (5.1)	29.1 (5.0)	27.7 (5.1)	28.0 (5.4)	27.7 (5.1)
Hostile acts	14.1 (2.3)	13.5 (2.8)	12.3 (1.8)	13.5 (2.8)	13.8 (2.3)	13.5 (2.8)	13.9 (2.5)	13.8 (2.8)
Domineering attitude	15.7 (3.6)	14.2 (3.2)*	13.6 (1.7)	14.3 (3.2)	15.3 (3.4)	14.2 (3.2)	14.1 (3.5)	14.3 (3.2)
REVISED SCALES								
Hostility	16.7 (3.2)	17.3(3.4)	17.0 (3.3)	17.3 (3.4)	16.7 (3.2)	17.3 (3.4)	17.6 (3.5)	17.2 (3.4)
Submissivenes	18.2 (4.5)	20.8(4.3)**	22.1 (4.2)	20.6 (4.4)	18.9 (4.5)	20.8 (4.4)*	21.6 (4.5)	20.6 (4.4)

*p<0.05,**p<0.01

Subjects were excluded if all items not completed (leaving 740 women).

Those with history of angina or MI at baseline are excluded (89 women); a subject may appear in more than one category.

Table 8.6

Means (s.d.) of Bedford-Foulds Personality Deviance Scales for incident intermittent claudication over 5-years of follow-up in men and women

	MEN		WOMEN	
	YES (n=34)	NO (n=772)	YES (n=37)	NO (n=744)
ORIGINAL SCALES				
Extrapunitiveness	25.3 (3.6)	25.4 (3.9)	24.2 (4.8)	24.1 (4.1)
Hostile thoughts	12.0 (2.2)	12.3 (2.4)	12.7 (2.9)	12.3 (2.6)
Denigratory attitude	13.3 (2.2)	13.1 (2.5)	11.5 (2.8)	11.8 (2.5)
Intropunitiveness	24.6 (3.5)	24.9 (3.9)	25.2 (4.4)	26.1 (4.5)
Lacks self confidence	11.0 (2.7)	11.3 (2.5)	11.9 (3.0)	12.5 (3.0)
Over-dependent	13.6 (2.1)	13.7 (2.3)	13.4 (2.8)	13.6 (2.5)
Dominance	29.1 (5.0)	29.7 (4.8)	29.0 (4.4)	27.7 (5.2)
Hostile acts	13.9 (3.1)	14.3 (2.7)	14.2 (3.0)	13.4 (2.9)
Domineering attitude	15.2 (2.9)	15.3 (3.0)	14.8 (2.8)	14.2 (3.2)
REVISED SCALES				
Hostility	17.5 (2.9)	17.5 (3.2)	18.0 (3.7)	17.3 (3.4)
Submissiveness	18.5 (3.6)	18.8 (3.7)	19.5 (4.3)	20.7 (4.3)

Those with a history of intermittent claudication at baseline were excluded.

Correlations of Follow-up Ankle Brachial Pressure Index (ABPI) with Bedford-Foulds Personality Deviance Scales

	Follow-up ABPI	
	MEN	WOMEN
ORIGINAL SCALES		
Extrapunitiveness	0.04	-0.01
Hostile thoughts	0.05	0.00
Denigratory attitude	0.02	-0.03
Intropunitiveness	-0.02	-0.04
Lack of self confidence	-0.05	-0.05
Over-dependence	0.03	-0.01
Dominance	0.04	-0.05
Hostile acts	-0.01	-0.08
Domineering attitude	0.07	-0.02
REVISED SCALES		
Submissiveness	-0.07	-0.02
Hostility	0.00	-0.03

Table 8.8

Correlations of Change in Ankle Brachial Pressure Index (CABPI) over five years with Bedford-Foulds Personality Deviance Scales

	Change in ABPI (CABPI)	
	MEN	WOMEN
ORIGINAL SCALES		
Extrapunitiveness	-0.05	-0.05
Hostile thoughts	-0.06	-0.04
Denigratory attitude	-0.02	-0.03
Intropunitiveness	0.05	0.01
Lack of self confidence	0.08	0.02
Over-dependence	-0.02	-0.01
Dominance	-0.09*	-0.04
Hostile acts	-0.06	-0.03
Domineering attitude	-0.09*	-0.04
REVISED SCALES		
Submissiveness	0.10*	0.03
Hostility	-0.07	-0.05

*p≤0.05; **p≤0.01

Correlations of residuals of follow-up Ankle Brachial Pressure Index (ABPI) regressed on baseline ABPI (RABPI) with Bedford-Foulds Personality Deviance Scales in men and women

	Residuals (RABPI)	
	MEN	WOMEN
ORIGINAL SCALES		
Extrapunitiveness	-0.04	-0.09*
Hostile thoughts	-0.05	-0.06
Denigratory attitude	-0.02	-0.06
Intropunitiveness	0.05	-0.03
Lack of self confidence	0.07	-0.02
Over-dependence	0.00	-0.03
Dominance	-0.09*	-0.11*
Hostile acts	-0.09*	-0.12**
Domineering attitude	-0.06	-0.07
REVISED SCALES		
Submissiveness	0.07	-0.10*
Hostility	-0.10*	0.03

*p≤0.05; **p≤0.01

Table 8.10

Multiple logistic regression of one standard deviation increase in Bedford-Foulds Personality Deviance Scales (original scales) plus covariates on the risk of coronary heart disease over 5 years in men and women

	MYOCARDIAL INFARCTION RR (95% CI)						**ANGINA** RR (95% CI)	
	Nonfatal		**Fatal**		**Total**			
MEN	Intercept	No independent predictors	Baseline ABPI	**0.10** (0.02, 0.50)	Baseline ABPI	**0.21** (0.07, 0.59)	Over-dependence HDL-chol Smoking	**1.42** (1.05, 1.93) **0.24** (0.08, 0.70) **1.14** (1.02, 1.26)
WOMEN	Denigratory attitude Lacks self confidence Baseline ABPI	**0.59** (0.40, 0.86) **0.60** (0.40, 0.90) **0.07** (0.01, 0.49	Systolic	**1.04** (1.01, 1.06)	Denigratory attitude Baseline ABPI Diastolic	**0.57** (0.40, 0.81) **0.09** (0.01, 0.55) **1.03** (1.00, 1.05)	Lacks confidence Systolic	**1.52** (1.09, 2.11) **1.02** (1.00, 1.03)

ABPI=ankle brachial pressure index; systolic=systolic blood pressure measured in the arm; diastolic=diastolic blood pressure; Smoking measured in packyears: number of 20-cigarette packs smoked per day, multiplied by number of years as a smoker; HDL - high-density lipoprotein cholesterol

Table 8.11

Multiple logistic regression of one standard deviation increase in Bedford-Foulds Personality Deviance Scales (revised scales) and covariates on the risk of CHD over 5 years in men and women

	MYOCARDIAL INFARCTION RR (95% CI)						**ANGINA** RR (95% CI)	
	Nonfatal		**Fatal**		**Total**			
MEN	Intercept	No independent predictors	Baseline ABPI	**0.11** (0.54, 0.02)	Baseline ABPI	**0.21** (0.07, 0.59)	HDL-chol Smoking	**0.30** (0.11,0.83) 1.12 (1.01,1.23)
WOMEN	Submissive-ness Systolic	**0.59** (0.40, 0.85) **1.02** (1.00, 1.03)	Systolic	**1.02** (1.01, 1.06)	Submissive-ness Baseline ABPI	**0.69** (0.27, 0.96) **0.11** (0.02, 0.63)	Systolic	**1.02** (1.00, 1.03)

ABPI=ankle brachial pressure index; systolic blood pressure measured in the arm; HDL-chol=high density lipoprotein cholesterol; smoking measured in packyears: number of 20-cigarette packs smoked per day, multiplied by number of years as a smoker.

Table 8.12

Multiple linear regression of Bedford-Foulds Personality Deviance Scales and baseline risk factors on follow-up ABPI, change in ABPI and residuals of ABPI in men

	B	(SE) B	p-value	Adjusted R^2 for model
Follow-up ABPI				
BMI	0.01	0.00	0.03	0.05
Total Chol	-0.02	0.01	0.02	
Age	-0.01	0.00	0.01	
Smoking	-0.02	0.01	0.01	
HDL-chol.	0.07	0.03	0.03	
Change in ABPI				
Deprivation	-0.00	0.00	0.06	0.01
Residuals of ABPI				
Smoking	-0.09	0.03	0.00	0.07
HDL-chol.	0.49	0.16	0.00	
Deprivation	-0.34	0.01	0.03	

ABPI=ankle brachial pressure index; **BMI**= body mass index; **Deprivation**-measured so that higher score indicates greater deprivation; **HDL-chol**=high density lipoprotein cholesterol. **Total chol**=total serum cholesterol. **Smoking** measured in packyears: number of 20-cigarette packs per day smoked, multiplied by number of years as a smoker.

Table 8.13

Multiple linear regression of Bedford-Foulds Personality Deviance Scales and baseline risk factors on follow-up ABPI, change in ABPI and residuals of ABPI in Women

	B	(SE) B	p-value	Adjusted R^2 for model
Follow-up ABPI				
Age	-0.01	0.00	0.00	0.14
Smoking	-0.01	0.01	0.02	
Change in ABPI				
Age	0.01	0.00	0.00	0.06
Residuals of ABPI				
Smoking	-0.10	0.04	0.01	0.02

ABPI=ankle brachial pressure index; **Smoking** measured in packyears: number of 20-cigarette packs smoked per day, multiplied by number of years as a smoker

CHAPTER 9

Results II: Cross-sectional associations between core personality traits, anger, disease and risk factors

9.1 INTRODUCTION

The second phase of the analysis was to determine the cross-sectional relationships between the NEO-Five Factor Inventory (NEO-FFI) Costa and McCrae, 1992) and the State-Trait Anger Expression Inventory (STAXI; Spielberger, 1989) and cardiovascular diseases. This allowed the associations between cardiovascular disease prevalence and neuroticism, extraversion, openness, agreeableness and conscientiousness as measured on the NEO-FFI, and anger as measured by the STAXI, to be examined. The methods of data collection and analysis were described in chapter seven.

Objectives three to six of the thesis are addressed by this part of the analysis, as are objectives seven and eight (see also chapter five):

(3) Do the broader personality dimensions of neuroticism, extraversion, openness and agreeableness show a relationship with prevalent cardiovascular diseases such as myocardial infarction, peripheral arterial disease or angina?

(4) Does the low pole of agreeableness relate to increased disease prevalence, and is this a suitable standard way to measure hostility?

(5) Do higher anger scores, especially anger-out, as would be expected from previous research, correlate with a higher prevalence of CHD, particularly MI?

(6) Does neuroticism relate to increased prevalence of subjective events such as angina, and not to objective events such as MI?

(7) Are the observed associations independent of the effects of other risk factors,

namely cigarette smoking, hypercholesterolaemia, hypertension, and rheological and haemostatic factors?

(8) What factors work in combination to increase or decrease the risk of CHD?

In this chapter, the response rates and descriptive data on disease prevalence and personality scores are presented first. The univariate analysis of the personality-disease and risk factor associations are presented next, followed by the multivariate analysis of the same data. Finally, the correlations between all three of the personality scales - the Bedford-Foulds Personality Deviance Scales (Bedford and Foulds, 1978), the NEO-FFI and the STAXI - are shown.

9.2 RESPONSE RATE

There were 1101 responders to the personality questionnaire from the eligible sample of 1296, a response rate of 85%. Eighty-two per cent (n=903) of the responders returned valid NEO-FFI questionnaires (questionnaires with no items missing). Valid numbers varied slightly for each scale, but the lowest was 82% (n=903) on the neuroticism scale, and the highest was 85% (n=933), on the agreeableness scale. Eighty per cent had valid STAXI scores: the range was from 80% (n=881) on the anger expression scale to 89% (n=983) on the total anger scale. Anger expression is calculated over a number of items, so if some are missing the scale cannot be scored.

9.3 DISEASE PREVALENCE

The prevalence of disease at the five-year cutoff date is shown in Table 9.1 -

(tables begin p. 203). The percentages of disease shown are calculated minus the deaths, but the percentage of deaths is shown out of the original 1592 participants. Prevalence of peripheral arterial disease reaches 25.6% in men and 33.5% in women when defined using a low ABPI as well as a doctor's diagnosis or a positive response on the WHO intermittent claudication questionnaire. The prevalence of intermittent claudication only was approximately 8.5% in both men and women. However, men had almost twice the rate of prevalent MI as women (13.8% v. 6.7%), twice the death rate from noncardiovascular causes (16.9% v 8.4%), and a higher prevalence rate of angina (17.4% v 13.1%). Overall prevalence of any form of cardiovascular disease (CVD) was approximately 40.5% in men and 43.4% in women.

9.4 DESCRIPTIVE STATISTICS OF NEO-FFI AND STAXI

The means and standard deviations of the NEO-FFI and STAXI in men and women are shown in Table 9.2. Women had slightly higher neuroticism (20.9 v. 17.3) and agreeableness (33.6 v. 31.5) scores than men, but mean levels of the other NEO-FFI and STAXI scales were roughly equivalent. For instance, the mean extraversion score for men was 25.0, and for women in was 24.9; conscientiousness in men was 33.1, and in women it was 33.2. On the STAXI, too, men and women had similar mean scores: total anger in men was 16.4, and in women was 16.0; angry reaction was 7.9 in men and 7.7 in women, and anger control was 25.3 in men and 24.8 in women.

The distributions of the NEO-FFI scales are shown in Appendix U (figures u1-u5), and it can be seen that they approximated the normal curve. However, some of

the anger measures (Appendix V; figures v1-v11) were highly skewed (anger-out - figure v5, anger-control- figure v6 and angry-temperament- figure v2) and were therefore adjusted using logarithmic transformations (figures v9, v11 and v8 respectively). The anger-control scale distribution, even transformed (figure v6), was problematic, but it was decided to use the nontransformed variable in further analyses. The anger-expression scale was used with the constant of 100 added (figure v10) in order to make interpretation of the direction of correlations and regression clearer (this avoided negative scores).

Distributions of 'prevalent' systolic and diastolic blood pressure (obtained by averaging the baseline and follow-up readings), updated smoking information and intima-media thickness (IMT) measures are shown in Appendix W (figures w1-w7). As the smoking and IMT data were skewed, both were transformed: the smoking data by square-root (figures w1 and w2), and the IMT data by logarithmic transformation (w6 and w7). ABPI measures taken at follow-up were used for this part of the analysis.

9.5 UNIVARIATE ANALYSIS

A similar strategy was used to analyze the cross-sectional data as was used for the longitudinal data. Mean levels of the personality scores were calculated for the groups with or without prevalent disease, and correlations were calculated between the personality variables and continuous data, such as blood pressure and smoking.

The numbers of events in each category for the remaining analysis is different

from the prevalence of events shown in table 9.1. This is because in the t-tests, only those with valid NEO-FFI and STAXI scores could be included. There were a further 4% of men (calculated against all those eligible who had not died) and 2% of women who had prevalent MI but no cross-sectional personality scores; and a further 9% of men and 5% of women who had prevalent angina; a further 3% of men and women had intermittent claudication; and a further 8% of men and 13% of women had some form of peripheral arterial disease, but no personality scores.

9.5.1 NEO-FFI and disease prevalence

The mean levels of the NEO-FFI in the various prevalent disease categories are shown in tables 9.3-9.6. In men (table 9.3), mean level differences were apparent between neuroticism in those with prevalent MI (19.1) and those without (17.1; $p \leq 0.05$), and those with angina (19.1) versus those without (17.0; $p \leq 0.05$). Openness scores were higher in the group without peripheral arterial disease (24.1) compared to those with some form of PAD (22.8; $p \leq 0.05$).

In women (table 9.4), there were differences in neuroticism scores in the MI (22.3 in the diseased v. 20.8 in the non-diseased). Statistically significant differences were apparent on neuroticism in the angina group (24.2 in the diseased v. 20.4 in the non-diseased; $p \leq 0.05$). On the agreeableness scale, higher scores were seen in those without angina (33.7 versus 32.3; $p \leq 0.05$), and on the conscientiousness scale, women without angina scored higher (33.4 versus 31.5; $p \leq 0.05$).

9.5.2 STAXI and disease prevalence

The mean STAXI scores by disease category are shown in tables 9.5 and 9.6. The anger-out and angry temperament scales were logarithmically transformed for analysis, so their transformed means are shown in the table. The means of the anger expression scale are shown with the constant of 100 added (explained in methods in chapter seven). In men, lower scores were apparent in those without an MI on the anger-out (1.10 v. 1.14; $p \leq 0.01$), anger expression (86.8 v. 89.3; $p \leq 0.05$) and total anger scales (16.3 v. 16.9; $p \leq 0.05$ - table 9.5). In the outcome of all CVD, anger-out scores were statistically significantly lower in those with CVD (1.10 v. 1.11; $p \leq 0.05$), as were anger-expression scores (86.5 v. 88.2; $p \leq 0.05$). Anger control scores, however, were lower in those men with CVD (24.7 v. 25.7; $p \leq 0.05$).

In women (table 9.6), the statistically significant differences appeared in the angina group. Anger control scores were lower in those with angina (23.6 versus 25.0; $p \leq 0.05$), but anger expression scores were higher (90.1 versus 87.2; $p \leq 0.05$). Anger expression scores were also higher in the group with CVD (88.7) versus those without (86.8; $p \leq 0.05$).

9.5.3 NEO-FFI and risk factors

The correlations between the NEO-FFI and physical factors in men and women are shown in Tables 9.7 and 9.8. In both sexes, statistically significant negative correlations were apparent between systolic and diastolic blood pressure and agreeableness (men, systolic, $r=-0.09$, $p \leq 0.05$; diastolic, $r=-0.13$, $p \leq 0.01$, table 8.7, systolic, women $r=-0.14$, $p \leq 0.01$; diastolic, $r=-0.11$, $p \leq 0.01$, table 8.8). In men, total cholesterol was also negatively correlated with agreeableness ($r=-0.10$, $p \leq 0.05$), and

openness was correlated -0.22 with systolic pressure ($p \leq 0.01$; table 9.7). In women, HDL cholesterol correlated positively with agreeableness ($r=0.16$, $p \leq 0.01$) and openness ($r=0.16$, $p \leq 0.01$; table 9.8). In addition, in women, conscientiousness was associated with systolic ($r=-0.10$, $p \leq 0.05$) and diastolic ($r=-0.13$, $p \leq 0.01$) blood pressure. Neither the ABPI nor IMT measures were significantly correlated with the NEO-FFI.

9.5.4 STAXI and risk factors

Three of the anger measures on the STAXI correlated positively with blood pressure in men (table 9.9): angry temperament ($r=0.10$ systolic, $p \leq 0.05$; $r=0.16$ diastolic, $p \leq 0.01$), angry reaction ($r=0.13$ diastolic, $p \leq 0.01$), and total anger ($r=0.19$ diastolic, $p \leq 0.01$). Angry reaction and total anger also were positively correlated with smoking ($r=0.13$, $p \leq 0.05$; $r=0.16$, $p \leq 0.01$).

Three STAXI scales correlated negatively with blood pressure in women (table 9.10): anger control with systolic ($r=-0.12$; $p \leq 0.05$) and diastolic ($r=-0.15$; $p \leq 0.01$), and angry reaction with systolic ($r=-0.10$, $p \leq 0.05$). Neither the ABPI nor the IMT measures were correlated with any of the anger scales, in either men or women.

9.6 MULTIVARIATE ANALYSIS

9.6.1 Multiple linear regression - risk factors

After controlling for the effects of the remaining risk factors in multivariate models, openness in men remained in the final model when systolic pressure was the

dependent variable (p=0.04), with BMI (p=0.00), age (p=0.02) and social class (p=0.04) also contributing to the 18% of the variance that was explained by the model (Table 9.11). Total anger (p=0.00) and BMI (p=0.00) explained 17% of the variance in diastolic pressure. Models generated for packyears and follow-up ABPI accounted for less of the variance than those for blood pressure: 7% of the variance in smoking was accounted for by total anger (p=0.01), deprivation score (p=0.02) and total cholesterol (p=0.05). Follow-up ABPI was most strongly associated with smoking and alcohol (p=0.01 for both), but only 4% of the variance in the measure was explained by the two variables together.

A somewhat similar picture was true for women (Table 9.12), but for systolic pressure, the model included age (p=0.00) and conscientiousness (p=0.04), and accounted for 24% of the variance. The proportion of variance (7%) explained by agreeableness (p=0.02) and BMI (p=0.03) in diastolic pressure was much smaller. Age was the only variable contributing independently to follow-up ABPI in women, and accounting for 4% of its variance. For smoking, total anger (p=0.02) and social class (p=0.00) remained in the final model, which explained 13% of the variance in smoking.

9.6.2 Multiple logistic regression - cardiovascular disease

Interestingly, although there had been only a hint in the univariate analysis of the effects, anger scores were still associated with prevalence of events in multivariate models. For instance, the multiple logistic model for prevalent myocardial infarction in men, using forward stepwise selection (Table 9.13), included anger-out, which was

associated with a higher risk of prevalent myocardial infarction (OR 1.90, 95% CI 1.28-2.80). Age (OR 1.08), agreeableness (1.10) and HDL cholesterol (0.14) were also significantly associated with prevalent myocardial infarction. The risk of prevalent intermittent claudication, in men, was associated only with a lower ABPI (indicating greater disease) (OR 0.002). However, the risk of having any kind of PAD, which included measures of asymptomatic disease, was associated with *lower* total anger scores (0.70; 0.52-0.95), but higher age (1.06), lower social class (1.27) and lowered ABPI (0.001). The variables associated with total CVD reflected the large proportion of this group made up by those with asymptomatic peripheral arterial disease, and higher total anger scores were associated with reduced risk (OR 0.74; 0.57- 0.95), as were higher HDL-cholesterol levels (0.42), and a higher ABPI (0.01). Older age (1.07) and lower social class (1.26) were associated with greater risk of being in the prevalent CVD category.

A different set of factors seemed to be of importance for women (Table 9.14). Age (OR 1.09) and diastolic pressure (OR 1.05) were associated with prevalent MI. For angina, higher anger-control scores were associated with decreased risk (OR 0.63; 0.42-0.94), as were higher HDL cholesterol levels (OR 0.09). Increased diastolic pressure was also associated with increased risk of angina (OR 1.04). Variables important for intermittent claudication were higher anger-in scores, which *decreased* risk (0.54; 0.32-0.90), greater number of packyears, which increased risk (OR 1.19), higher neuroticism scores, which also increased risk (OR 2.01), and a higher ABPI, which decreased risk (OR 0.01). For all peripheral vascular disease, however, only age (OR 1.11) and ABPI (OR 0.001) were included in the final model. Finally, for

all disease groups combined, it appeared that anger-control was associated with reduced risk (OR 0.72; 0.55-0.95), along with higher HDL cholesterol (0.49) and ABPI (0.01); factors increasing risk were age (1.09) and systolic pressure (1.02).

9.7 CORRELATIONS OF PDS, NEO-FFI AND STAXI

9.7.1 PDS and NEO-FFI

Many of the PDS and NEO-FFI dimensions were correlated, often statistically significantly (table 9.15). For instance, neuroticism was correlated positively with intropunitiveness ($r=0.46$; $p\leq0.01$), lack of self confidence ($r=0.49$; $p\leq0.01$) and submissiveness ($r=0.43$; $p\leq0.01$), and negatively with dominance ($r=-0.22$; $p\leq0.01$) and domineering attitude ($r=-0.25$; $p\leq0.01$). Agreeableness was negatively correlated with hostile acts ($r=-0.35$; $p\leq0.01$), dominance ($r=-0.34$; $p\leq0.01$), extrapunitiveness ($r=-0.38$; $p\leq0.01$), hostile thoughts ($r=-0.31$;$p\leq0.01$) and denigratory attitude ($r=-0.31$; $p\leq0.01$).

9.7.2 PDS and STAXI

There were also correlations between the PDS and STAXI measures of anger (table 9.16). Anger-out was correlated most strongly with PDS-hostility ($r=0.47$; $p\leq0.01$), hostile acts ($r=0.43$; $p\leq0.01$), hostile thoughts ($r=0.39$; $p\leq0.01$) and dominance ($r=0.39$; $p\leq0.01$). Angry temperament, too, was correlated with PDS-hostility ($r=0.48$; $p\leq0.01$), hostile acts ($r=0.40$; $p\leq0.01$), hostile thoughts ($r=0.40$; $p\leq0.01$) and dominance ($r=0.36$; $p\leq0.01$). STAXI total anger and anger expression also showed correlation coefficients of similar magnitude with PDS-hostility, hostile

acts, hostile thoughts and dominance. Anger-in and angry reaction in general showed slightly smaller correlation coefficients. STAXI anger control was correlated negatively with the PDS dimensions.

9.7.3 NEO-FFI and STAXI

STAXI anger-out was negatively correlated with NEO-agreeableness ($r=-0.50$; $p\leq0.01$; table 9.17). NEO-agreeableness was also negatively correlated with anger expression ($r=-0.50$), angry temperament ($r=-0.45$) and total anger ($r=-0.45$), and postively correlated with anger control ($r=0.38$; all at $p\leq0.01$). Neuroticism was significantly correlated with anger-in ($r=0.42;p\leq0.01$) and anger-expression ($r=0.42;p\leq0.01$); anger-out, angry temperament, angry reaction and total anger were slightly less strongly positively correlated with neuroticism, and anger-control was negatively correlated with neuroticism ($r=-0.27;p\leq0.01$). Other correlations were weaker, such as between higher conscientiousness and lower anger expression ($r=-0.22$; $p\leq0.01$) or higher extraversion and lower anger-in ($r=-0.24$; $p\leq0.01$). The NEO facet of openness was not correlated with any of the STAXI anger measures.

9.8 CHAPTER SUMMARY

The findings of this chapter can now be applied to thesis's objectives, which were listed at the beginning of the chapter.

(3) Do the broader personality dimensions of neuroticism, extraversion, openness and agreeableness show a relationship with prevalent cardiovascular diseases such as myocardial infarction, peripheral arterial disease or angina? Yes, although the

strength of the relationship was not immediately clear on performing the univariate analysis. There were differences in neuroticism in the MI and angina categories in both men and women, and in agreeableness and conscientiousness, for angina, in women. There were also differences in mean levels of the anger measures in men who had an MI, and in women who had angina. However, the associations needed further exploration in multivariate analysis, to allow statistical adjustment for other risk factors.

(4) Does the low pole of agreeableness relate to increased disease prevalence, and is this a suitable standard way to measure hostility? In the univariate analysis, low agreeableness was associated with increased prevalence of angina in women. In multivariate analysis, low agreeableness was independently associated with prevalent MI, in men. The anger measures on the STAXI, however, were associated with the increased prevalence of disease much more consistently, although not always in the expected directions. In multivariate analysis, in men, higher anger-out scores were associated with a 90% increase in the risk of having had an MI. Other anger scales were associated with a lower prevalence of disease: higher total anger scores were related to lower prevalence of PAD in men, and higher anger-control and anger-in scores were associated with lower prevalence of angina and intermittent claudication in women.

Lower agreeableness was moderately correlated with higher anger-out (r=-0.50), anger expression (r=-0.50), and total anger (r=-0.45), and positively correlated with anger-control (r=0.38). Although the domains of anger appear to overlap, agreeableness does not seem to be specific enough, for CHD at least, to use as the

only measure of anger or hostility in behavioural epidemiological studies.

(5) Do higher anger scores, especially anger-out, as would be expected from previous research, correlate with a higher prevalence of CHD, particularly MI? Part of this question was considered above: higher anger-out scores, in men, *were* associated in multivariate analysis with an increased prevalence of MI. However, other anger measures were associated with a reduced prevalence of angina and intermittent claudication, in both men and women.

(6) Does neuroticism relate to increased prevalence of subjective events such as angina, and not to objective events such as MI? This relationship does not appear to be a simple one. In the univariate stage of the analysis, NEO-FFI neuroticism scores were higher in both men and women who had an MI or angina. In the multivariate analyses, however, neuroticism was associated with the increased prevalence of intermittent claudication in women only - not to angina or MI, and not in men. Intermittent claudication may be diagnosed on only the reporting of symptoms, so this does not contradict expectations. However, NEO-neuroticism was positively ($r=0.42$) correlated with the STAXI dimension of anger in, but anger-in was associated with a *decreased* risk of intermittent claudication in women. Therefore, although the anger-in and neuroticism may tap some similarities, they are not measuring the same construct.

(7) Are the observed associations independent of the effects of other risk factors, namely cigarette smoking, hypercholesterolaemia, hypertension, and rheological and haemostatic factors? As in the longitudinal analysis, it was found that some of the personality measures were exerting an independent effect on the risk of disease. This

effect, in fact, was more apparent after adjusting for other risk factors: the univariate analysis had not shown many differences in mean levels on the NEO-FFI or STAXI across the disease categories. Their influence may have been masked by the complicated nature of the relationship. On multivariate analysis the anger measures emerged as having an independent effect on the risk of MI and PAD in men, and on angina and intermittent claudication in women. Nevertheless, these dimensions were also independently associated with the risk factors, especially blood pressure and smoking with anger, so they may be exerting their effects in complicated ways.

(8) What factors work in combination to increase or decrease the risk of CHD? The traditional risk factors were important in the multivariate models of risk: a higher HDL-cholesterol, for instance, was associated with a reduction in MI prevalence in men of 86%, and a reduction in risk of angina in women of 91%. Each of the disease outcomes was associated with multiple factors. They appeared in slightly different combinations, but age, blood pressure and HDL-cholesterol were consistently associated with disease prevalence, in the expected directions. The ABPI was again shown to be an important and strong indicator of risk. The personality variables were *adding* information, allowing a more precise calculation of risk to be made. The issues and complications raised by these results, and the interpretation of them, are discussed in the second section of chapter ten.

Table 9.1

Prevalence of cardiovascular disease after 5 years of follow-up in men and women

	MEN n=672	WOMEN n=717
CHD		
Myocardial infarction	13.8% (93)	6.7% (48)
Angina	17.4% (117)	13.1% (94)
PAD		
Claudication	8.3% (56)	8.5% (61)
All PAD	25.6% (172)	33.5% (240)
ALL CVD	40.5% (272)	43.4% (311)
ALL DEATHS	16.9% (137)	8.4% (66)

Deaths calculated as percentage of 1592; prevalent disease as percentage of 1592 minus the deaths during the five years.
CHD - coronary heart disease; **PAD** - peripheral arterial disease; **CVD** - cardiovascular disease

Table 9.2

Means (s.d.) and median of NEO-Five Factor Inventory (NEO-FFI) and State-Trait Anger Expression Inventory (STAXI) in 447 men and 452 women

	MEN	**WOMEN**
NEO-FFI		
	Mean	Mean
Neuroticism	17.3 (7.25)	20.9 (7.96)
Extraversion	25.0 (5.26)	24.9 (5.59)
Openness	23.8 (5.90)	24.7 (7.00)
Agreeableness	31.5 (5.18)	33.6 (4.69)
Conscientiousness	33.1 (5.83)	33.2 (5.74)
STAXI		
Total anger	16.4 (4.05)	16.0 (3.75)
Angry-temperament	5.8 (1.90)	5.6 (1.70)
Angry-reaction	7.9 (2.29)	7.7 (2.15)
Anger-out	13.0 (3.14)	12.6 (2.89)
Anger-in	15.5 (3.52)	15.8 (3.59)
Anger-expression	-12.9 (8.35)	-12.4 (8.06)
Anger-control	25.3 (4.97)	24.8 (4.95)

Means (s.d.) of NEO Five Factor Inventory (NEO-FFI) and prevalent cardiovascular disease in men

	CHD				**PAD**				**ALL CVD**	
	MI		ANGINA		CLAUD		ALL PAD			
	yes n=57	no n=394	yes n=71	no n=376	yes n=33	no n=414	yes n=99	no n=348	yes n=162	no n=285
Neuroticism	19.1 (6.5)	17.1* (7.3)	19.1 (7.6)	17.0* (7.1)	18.0 (8.6)	17.3 (7.1)	17.6 (6.9)	17.3 (7.4)	18.1 (7.1)	16.9 (7.3)
Extraversion	24.8 (4.4)	25.0 (5.4)	25.0 (5.7)	24.9 (5.2)	24.1 (5.4)	25.0 (5.2)	24.4 (5.1)	25.1 (5.3)	24.7 (5.2)	25.1 (5.3)
Openness	24.5 (6.4)	23.7 (5.8)	24.3 (6.2)	23.7 (5.9)	22.6 (6.5)	23.9 (5.8)	22.8 (5.2)	24.1* (6.0)	23.6 (5.8)	23.9 (6.0)
Agreeableness	31.4 (5.2)	31.5 (5.2)	31.9 (5.7)	31.4 (5.1)	32.3 (4.8)	31.4 (5.2)	31.5 (5.3)	31.5 (5.1)	31.7 (5.5)	31.4 (5.0)
Conscientiousness	32.3 (5.5)	33.2 (5.9)	33.3 (6.3)	33.0 (5.8)	32.8 (6.5)	33.1 (5.8)	33.3 (5.2)	33.0 (6.0)	33.1 (5.3)	33.0 (6.1)

*p<0.05; **p<0.01; **CHD** - coronary heart disease; **PAD** - peripheral arterial disease; **CVD** - cardiovascular disease (includes all other categories); **MI** - myocardial infarction; **CLAUD** - intermittent claudication

Means (s.d.) of NEO Five Factor Inventory (NEO-FFI) and prevalent cardiovascular disease in women

	CHD				PAD				ALL CVD	
	MI		ANGINA		CLAUD		ALL PAD			
	yes n=31	no n=425	yes n=55	no n=401	yes n=38	no n=418	yes n=148	no n=308	yes n=191	no n=265
Neuroticism	22.3 (9.1)	20.8 (7.9)	24.2 (7.2)	20.4* (8.0)	22.9 (8.4)	20.7 (7.9)	20.9 (7.5)	20.8 (8.2)	21.3 (7.6)	20.5 (8.2)
Extraversion	26.0 (7.2)	24.8 (5.5)	24.1 (5.0)	25.0 (5.7)	24.9 (5.3)	24.9 (5.6)	24.7 (5.6)	25.0 (5.6)	24.8 (5.6)	25.0 (5.6)
Openness	24.8 (4.6)	24.7 (5.7)	23.5 (6.0)	24.9 (5.5)	23.4 (5.3)	24.8 (5.6)	24.2 (5.3)	25.0 (5.7)	24.3 (5.4)	25.1 (5.7)
Agreeableness	32.5 (4.3)	33.6 (4.7)	32.3 (3.8)	33.7* (4.8)	32.7 (3.8)	33.6 (4.7)	33.2 (4.7)	33.7 (4.7)	33.1 (4.6)	33.9 (4.8)
Conscientiousness	32.9 (5.3)	33.2 (5.8)	31.5 (4.5)	33.4* (5.8)	32.8 (6.9)	33.7 (5.6)	33.1 (5.8)	33.3 (5.7)	32.9 (5.6)	33.5 (5.8)

*p<0.05; **p<0.01; **CHD** - coronary heart disease; **PAD** - peripheral arterial disease; **CVD** - cardiovascular disease (includes all other categories); **MI** - myocardial infarction; **CLAUD** - intermittent claudication

Table 9.5

Means (s.d.) of State Trait Anger Expression Inventory (STAXI) dimensions and prevalent cardiovascular disease in men

	CHD				PAD				All CVD	
	MI		ANGINA		CLAUD		ALL PAD			
	yes n=60	no n=408	yes n=73	no n=378	yes n=36	no n=417	yes n=108	no n=360	yes n=167	no n=284
Anger out	1.14 (0.1)	1.10** (0.1)	12.12 (0.1)	1.10 (0.1)	1.10 (0.1)	1.10 (0.1)	1.10 (0.1)	1.10 (0.1)	1.11 (0.1)	1.10* (0.1)
Anger in	15.8 (3.3)	15.50 (3.6)	15.4 (3.6)	15.6 (3.5)	16.2 (3.7)	15.5 (3.5)	15.9 (3.6)	15.4 (3.5)	15.7 (3.5)	15.5 (3.5)
Anger control	24.2 (4.8)	25.45 (5.0)	25.0 (5.0)	25.4 (5.0)	25.9 (4.9)	25.2 (5.0)	24.9 (5.0)	25.4 (5.0)	24.7 (5.0)	25.7* (4.9)
Anger expression	89.3 (8.6)	86.8* (8.3)	87.6 (8.9)	87.0 (8.2)	87.3 (8.9)	87.1 (8.3)	90.0 (8.7)	86.8 (8.2)	88.2 (8.8)	86.5* (8.0)
Angry temperament	0.78 (0.1)	0.74 (0.1)	0.76 (0.1)	0.74 (0.1)	0.75 (0.1)	0.74 (0.1)	0.75 (0.1)	0.74 (0.1)	0.75 (0.1)	0.74 (0.1)
Angry reaction	7.8 (2.1)	7.88 (2.3)	7.7 (2.2)	7.9 (2.3)	8.2 (2.3)	7.8 (2.3)	7.6 (2.1)	8.0 (2.3)	7.7 (2.1)	8.0 (2.4)
Total anger	16.9 (4.2)	16.3* (4.0)	16.5 (4.1)	16.4 (4.0)	16.9 (4.1)	16.3 (4.0)	16.2 (4.1)	16.4 (4.1)	16.5 (4.0)	16.3 (4.1)

*p<0.05; **p<0.01; CHD - coronary heart disease; PAD - peripheral arterial disease; CVD - cardiovascular disease (includes all other categories); MI - myocardial infarction; CLAUD - intermittent claudication

Table 9.6

Means (s.d.) of State Trait Anger Expression Inventory (STAXI) dimensions and prevalent cardiovascular disease in women

	CHD				**PAD**				**All CVD**	
	MI		ANGINA		CLAUD		ALL PAD			
	yes n=32	no n=437	yes n=51	no n=418	yes n=40	no n=435	yes n=150	no n=325	yes n=196	no n=279
Anger out	1.11 (0.1)	1.09 (0.1)	1.10 (0.1)	1.09 (0.1)	1.11 (0.1)	1.09 (0.1)	1.10 (0.1)	1.09 (0.1)	1.10 (0.1)	1.08 (0.1)
Anger in	15.2 (3.7)	15.9 (3.6)	16.5 (3.5)	15.7 (3.6)	16.3 (4.0)	15.8 (3.6)	15.9 (3.8)	15.8 (3.5)	16.0 (3.8)	15.7 (3.4)
Anger control	23.4 (5.8)	24.9 (4.9)	23.6 (4.7)	25.0 (5.0)	24.0 (5.1)	24.9 (4.9)	24.5 (4.9)	24.9 (5.0)	24.3 (4.9)	25.2 (4.9)
Anger expression	88.7 (8.9)	87.5 (8.0)	90.1 (7.2)	87.2 (8.1)	89.6 (8.1)	87.4 (8.0)	88.2 (8.3)	87.3 (8.0)	88.7 (8.1)	86.8 (7.9)
Angry temperament	0.75 (0.1)	0.73 (0.1)	0.75 (0.1)	0.73 (0.1)	0.74 (0.1)	0.73 (0.1)	0.74 (0.1)	0.73 (0.1)	0.74 (0.1)	0.72 (0.1)
Angry reaction	7.7 (2.2)	7.7 (2.2)	7.6 (2.2)	7.6 (2.1)	7.7 (2.2)	7.7 (2.1)	7.6 (2.1)	7.7 (2.2)	7.6 (2.1)	7.7 (2.1)
Total anger	16.4 (4.2)	16.0 (3.7)	16.4 (3.7)	16.0 (3.8)	16.6 (4.2)	16.0 (3.7)	16.1 (3.8)	16.0 (3.7)	16.1 (3.8)	15.9 (3.7)

*p<0.05; **p<0.01; **CHD** - coronary heart disease; **PAD** - peripheral arterial disease; **CVD** - cardiovascular disease (includes all other categories); **MI** - myocardial infarction; **CLAUD** - intermittent claudication

Table 9.7

Correlations of NEO Five Factor Inventory (NEO-FFI) dimensions with physical risk factors measured at follow-up in 447 men

	Neuroticism	Extraversion	Openness	Agreeableness	Conscientiousness
Total Cholesterol	0.03	-0.03	0.01	-0.10*	-0.03
HDL-chol	-0.03	-0.02	0.04	0.01	-0.03
Systolic	0.02	0.01	-0.22**	-0.09*	0.05
Diastolic	0.07	0.08	-0.07	-0.13**	0.04
Smoking	-0.10	-0.03	-0.05	0.02	-0.06
ABPI	-0.03	-0.01	0.05	0.11	-0.05
IMT	-0.06	0.00	-0.08	-0.03	0.05

*p<0.05; **p<0.01; **HDL-chol** - high density lipoprotein cholesterol; **smoking** is calculated in packyears - by calculating the number of 20-cigarette packs smoked per day multiplied by number of years as a smoker; **ABPI** -ankle-brachial pressure index**IMT** - intima-media thickness; **Systolic** and **diastolic** blood pressures refer to arm measurements

Table 9.8

Correlations of NEO Five Factor Inventory (NEO-FFI) dimensions with physical risk factors measured at follow-up in 452 women

	Neuroticism	Extraversion	Openness	Agreeableness	Conscientiousness
Total cholesterol	-0.06	0.05	0.05	-0.08	-0.01
HDL-chol	-0.07	-0.01	0.16**	0.16**	0.09
Systolic	0.00	-0.06	-0.08	-0.14**	-0.10*
Diastolic	0.01	0.00	-0.02	-0.11*	-0.13**
Smoking	-0.10	0.05	-0.12	-0.03	-0.02
ABPI	-0.01	0.08	-0.01	0.02	0.04
IMT	-0.01	-0.06	-0.06	0.03	0.01

*p<0.05; **p<0.01

HDL-chol - high density lipoprotein cholesterol; **smoking** measured in packyears - are calculated by calculating the number of 20-cigarette packs smoked per day multiplied by number of years as a smoker; **ABPI** - ankle brachial pressure index **IMT** - intima-media thickness; **systolic** and **diastolic** blood pressures refer to are measurements

Table 9.9

Correlations of State-Trait Anger Expression Inventory (STAXI) anger measures and physical risk factors in 447 men

	Anger-out	Anger-in	Anger-control	Anger expression	Angry temperament	Angry reaction	Total anger
Total cholesterol	0.06	0.09*	-0.02	0.07	0.02	0.01	0.02
HDL-chol	-0.08	0.02	0.05	-0.05	-0.03	-0.01	-0.01
Systolic	0.10	-0.02	-0.04	0.07	0.10*	0.04	0.07
Diastolic	0.11	0.03	-0.08	0.09	0.16**	0.13**	0.19**
Smoking	0.05	0.05	-0.03	0.07	0.08	0.13*	0.16**
ABPI	-0.04	-0.07	0.02	-0.07	-0.03	0.09	0.03
IMT	0.06	-0.05	-0.04	0.02	0.06	0.03	0.04

*p<0.05; **p<0.01; **HDL-chol** - high density lipoprotein cholesterol; **smoking** measured in packyears - are calculated by calculating the number of 20-cigarette packs smoked per day multiplied by number of years as a smoker; **ABPI** - ankle brachial pressure index; **IMT** - intima-media thickness; systolic and diastolic pressures refer to arm measurements

Table 9.10

Correlations of State-Trait Anger Expression Inventory (STAXI) anger measures and physical risk factors in 452 women

	Anger out	Anger-in	Anger control	Anger expression	Angry temperament	Angry reaction	Total anger
Total chol	0.04	0.03	0.04	0.01	0.02	0.00	0.00
HDL chol	0.00	-0.06	0.06	-0.07	0.00	-0.02	-0.03
Systolic	-0.07	-0.07	-0.12*	0.02	0.01	-0.10*	-0.06
Diastolic	-0.10*	-0.08	-0.15**	0.02	0.01	-0.06	-0.04
ABPI	-0.06	0.02	0.03	-0.02	-0.11*	0.00	-0.06
Smoking	0.07	0.04	-0.11	0.08	0.01	-0.07	0.07
IMT	0.06	-0.05	0.00	0.02	0.06	0.03	0.04

*p<0.05; **p<0.01; **HDL-chol** - high density lipoprotein cholesterol; **smoking** measured in packyears are calculated by calculating the number of 20-cigarette packs smoked per day multiplied by number of years as a smoker; **ABPI** - ankle brachial pressure index **IMT** - initma-media thickness; **systolic** and **diastolic** pressures refer to arm blood pressures

Table 9.11

Multiple linear regression of NEO Five Factor Inventory (NEO-FFI) and State-Trait Anger Expression Inventory (STAXI) dimensions plus covariates on physical risk factors in 447 men

	B	SE B	p-value	R^2 for model
Systolic pressure				
Body mass index	2.0	0.42	0.00	0.18
NEO-Openness	-0.5	0.24	0.04	
age	0.6	0.25	0.02	
social class	2.2	1.08	0.04	
Diastolic pressure				
Body mass index	1.2	0.21	0.00	0.17
STAXI-Total anger	0.3	0.15	0.03	
Smoking				
STAXI-Total anger	0.1	0.03	0.01	0.07
Deprivation score	0.1	0.04	0.02	
Total cholesterol	-0.2	0.12	0.05	
Follow-up ABPI				
Packyears (baseline)	0.0	0.01	0.01	0.04
Alcohol	0.0	0.01	0.01	

Higher **social class** and **deprivation** scores were indicative of lower social class and greater deprivation; **smoking** measured in packyears - calculated by calculating the number of 20-cigarette packs smoked per day multiplied by number of years as a smoker; **ABPI** - ankle brachial pressure index.

Table 9.12

Multiple linear regression of NEO Five Factor Inventory (NEO-FFI) and State-Trait Anger Expression Inventory (STAXI) dimensions plus covariates on physical risk factors in 452 women

	B	SE B	p-value	R2 for model
Systolic pressure				
Age	1.47	0.31	0.00	0.24
Body mass index	1.25	0.37	0.00	
NEO-Conscientiousness	-0.59	0.29	0.04	
Diastolic pressure				
NEO-Agreeableness	-0.45	0.19	0.02	0.07
Body mass index	0.48	0.22	0.03	
Smoking				
Social class	0.56	0.15	0.00	0.13
STAXI-Total anger	0.18	0.08	0.02	
Follow-up ABPI				
Age	-0.01	0.00	0.01	0.04

Higher **social class** and **deprivation** scores were indicative of lower social class and greater deprivation; **smoking** measured in packyears - calculated by calculating the number of 20-cigarette packs smoked per day multiplied by number of years as a smoker; **ABPI** - ankle brachial pressure index.

Multiple logistic regression of one standard deviation increase in the NEO-Five Factor Inventory (NEO-FFI) and State-Trait Anger Expression Inventory (STAXI) plus covariates on the risk of prevalent CHD in 447 men

	OR (95% CI)
CHD	
Myocardial infarction	
STAXI Anger-out	1.90 (1.28, 2.80)
age	1.08 (1.01, 1.15)
NEO agreeableness	1.10 (1.01, 1.20)
HDL-chol	0.14 (0.04, 0.51)
Angina	
HDL-chol	0.15 (0.04, 0.48)
ABPI	0.12 (0.02, 0.64)
PAD	
Claudication	
ABPI	0.002 (0.0002, 0.02)
All PAD	
STAXI Total anger	0.70 (0.52, 0.95)
age	1.06 (1.01, 1.12)
social class	1.27 (1.01, 1.58)
ABPI	0.001 (0.00, 0.007)
Total CVD	
STAXI Total anger	0.74 (0.57, 0.95)
age	1.07 (1.02, 1.13)
social class	1.26 (1.03, 1.54)
HDL-chol	0.42 (0.18, 0.98)
ABPI	0.01 (0.001, 0.06)

CHD - coronary heart disease; **PAD** - peripheral arterial disease; CVD - cardiovascular disease; **ABPI** - ankle brachial pressure index; **HDL**-chol - high density lipoprotein cholesterol

Table 9.14

Multiple logistic regression of one standard deviation increase in the NEO-Five Factor Inventory (NEO-FFI) and State-Trait Anger Expression Inventory (STAXI) plus covariates on the risk of prevalent CHD in 452 women

	OR (95% CI)
CHD	
Myocardial infarction	
age	1.09 (1.00, 1.18)
diastolic pressure	1.05 (1.01, 1.09)
Angina	
STAXI anger-control	0.63 (0.42, 0.94)
HDL-chol	0.09 (0.02, 0.32)
diastolic pressure	1.04 (1.00, 1.07)
PAD	
Claudication	
STAXI Anger-in	0.54 (0.32, 0.90)
smoking	1.19 (1.01, 1.40)
NEO neuroticism	2.01 (1.23, 3.27)
ABPI	0.01 (0.0005, 0.20)
All PAD	
age	1.11 (1.05, 1.17)
ABPI	0.001 (0.00, 0.006)
Total CVD	
anger-control	0.72 (0.55, 0.95)
age	1.09 (1.04, 1.15)
HDL-chol	0.49 (0.26, 0.95)
systolic pressure	1.02 (1.00, 1.03)
ABPI	0.01 (0.0007, 0.06)

CHD - coronary heart disease; **PAD** - peripheral arterial disease; CVD - cardiovascular disease; **ABPI** - ankle brachial pressure index; HDL-chol - high density lipoprotein cholesterol

Table 9.15

Correlations between the Bedford-Foulds Personality Deviance Scales (PDS) and NEO Five Factor Inventory (NEO-FFI) in 447 men and 452 women

	Neuroticism	Extraversion	Openness	Agreeableness	Conscientiousness
Extrapunitiveness	0.13**	-0.08*	0.00	-0.38**	-0.11**
Hostile thoughts	0.20**	-0.04	0.06	-0.31**	-0.15**
Denigratory attitude	0.01	-0.08*	-0.06	-0.31**	-0.03
Intropunitiveness	0.46**	-0.26**	-0.08**	0.06	-0.27**
Low self confidence	0.49**	-0.33**	-0.05	0.04	-0.28**
Over dependence	0.23**	-0.06	-0.09**	0.06	-0.15**
Dominance	-0.22**	0.22**	0.07*	-0.34**	0.11**
Hostile acts	-0.10**	0.13**	0.00	-0.35**	0.06*
Domineering attitude	-0.25**	-0.08*	0.11**	-0.21**	0.12**
Hostility	0.15**	0.01	0.02	-0.38**	-0.08*
Submissiveness	0.43**	-0.35**	-0.08**	0.11**	-0.26**

*p<0.05; **p<0.01

Table 9.16

Correlations between the Bedford-Foulds Personality Deviance Scales (PDS) and the State Trait Anger Expression Inventory (STAXI) scales in 447 men and 452 women

	Anger out	Anger in	Anger control	Anger expression	Angry temperament	Angry reaction	Total anger
Extrapunitiveness	0.34**	0.24**	-0.21**	0.37**	0.32**	0.29**	0.39**
Hostile thoughts	0.39**	0.24**	-0.24**	0.40**	0.40**	0.29**	0.43**
Denigratory attitude	0.17**	0.16**	-0.10**	0.21**	0.13**	0.19**	0.21**
Intropunitiveness	0.02	0.18**	-0.12**	0.17**	0.06	0.00	0.04
Low self confidence	0.03	0.24**	-0.14**	0.21**	0.04	0.01	0.05
Over dependence	0.01	0.03	-0.04	0.06	0.05	-0.02	0.01
Dominance	0.39**	-0.11**	-0.23**	0.23**	0.36**	0.19**	0.32**
Hostile acts	0.43**	-0.06*	-0.25**	0.28**	0.40**	0.18**	0.33**
Domineering attitude	0.24**	-0.12**	-0.15**	0.12**	0.23**	0.14**	0.21**
Hostility	0.47**	0.17**	-0.29**	0.43**	0.48**	0.29**	0.47**
Submissiveness	-0.08*	0.23**	-0.01	0.09**	-0.07*	-0.05	-0.05

*p<0.05; **p<0.01

Table 9.17

Correlations between NEO Five Factor Inventory (NEO-FFI) dimensions and State Trait Anger Expression Inventory (STAXI) scales in 447 men and 452 women

	Anger out	Anger in	Anger control	Anger Expression	Angry temperament	Angry reaction	Total anger
Neuroticism	0.21**	0.42**	-0.27**	0.42**	0.27**	0.21**	0.32**
Extraversion	-0.03	-0.24**	0.15**	-0.22**	-0.03	-0.01	-0.06
Openness	0.01	-0.05	0.06	-0.06	-0.01	-0.05	-0.04
Agreeableness	-0.50**	-0.23**	0.38**	-0.50**	-0.45**	-0.26**	-0.45**
Conscientious-ness	-0.12**	-0.10**	0.21*	-0.22**	-0.11**	0.08*	-0.05

*p<0.05; **p<0.01

CHAPTER 10

Discussion

10.1 INTRODUCTION

In this chapter the longitudinal findings presented in chapter eight - the associations between the Bedford-Foulds Personality Deviance Scales (PDS; Bedford and Foulds, 1978) incident coronary heart disease - are discussed first. The cross-sectional results relating to the NEO-Five Factor Inventory (McCrae and Costa, 1990) and the State-Trait Anger Expression Inventory (Spielberger, 1989), which were shown in chapter nine, are addressed in the second part of the chapter. Both sets of findings are considered in relation to other studies, and the general issues of the methodology and the analysis are also examined. In the third part of the chapter, the hypotheses about the biological mechanisms of personality-disease relationships are reviewed.

10.2 LONGITUDINAL FINDINGS

The main findings of the longitudinal analysis are that submissiveness scores were higher in men and women who remained free of a non-fatal MI over the five-year follow-up period, compared to those who did not. On further adjustment for baseline risk factors and indicators of disease, submissiveness still appeared to exert an independent effect in women, but not in men. A standard deviation increase in the womens' submissiveness score was associated with a 41% decrease in risk of nonfatal myocardial infarction (MI). Lack of self confidence in women, and over-dependence in men, aspects of neuroticism, were associated

with increased incidence of angina. None of the baseline personality factors, however, was associated with five-year incidence of peripheral arterial disease (PAD), either clinical or subclinical, in multivariate analysis. In all of the longitudinal analyses, a low ankle brachial pressure index (ABPI) was a strong predictor of future CHD incidence. This was expected, since the ABPI measures the extent of peripheral arterial disease (PAD).

The result that submissiveness appears to be protective against non-fatal MI concurs with findings from the Western Collaborative Group Study, which showed that men who were more submissive experienced a reduced mortality rate over 22 years (Houston et al, 1997). However, in the Whitehall II study, London civil servants (both men and women), with greater job control, apparently *less* submissive, were at decreased risk of self-reported CHD (Bosma et al, 1997; Marmot et al, 1997). Moreover, in a study of Olive baboons, Sapolsky (1995) found a possible deleterious health effect for submissiveness: subordinate male baboons had poorer cardiovascular responses to stress (introduction of a new dominant troop member) in comparison with dominant male baboons. In cynomolgous macaque monkeys, Kaplan et al (1996) found that dominant females developed little atherosclerosis, whereas subordinate females resembled males in the extent of atherosclerotic lesions. The subordinate females also had other markers of poor health, including hypercortisolaemia, behavioural dysfunction and impaired ovarian function. It is possible that the resulting low concentrations of oestrogens accounted for their accelerated atherosclerosis (Kaplan et al, 1996).

Interpretation of the EAS findings is therefore complicated. For example,

the PDS measure of submissiveness may not be comparable to observations of social subordinance or to job control: job control focuses on the person's environment, and PDS-personality trait measures focus on the person. In addition, one feature of the PDS-submissiveness scale items is the stress on contentment with the role. For example, two of the items read "When in a group, I have been quite content to be led", and " I have been content to be dominated by someone else." The protective effect may therefore be apparent in submissives because they are content to be so. In the Whitehall II study, those in positions of low job control, who are at greater risk, may in fact not be submissive (a personal characteristic), but are forced to be subordinate (an environmental demand). This also may be happening in the troops of baboons and monkeys, where being forcefully subdued is stressful and adversely affects cardiovascular health.

Sapolsky (1995) also observed that subordinate individuals who avoided conflict with dominant animals during times of conflict were not adversely affected, and that dominant baboons showed a marked cardiovascular response if involved in an interaction which challenged their dominance. His interpretation was that

"social instability is not intrinsically a stressor - it appears to depend on whether one is fortunate enough to remain a spectator during such instability."

There is further support for this from a study of captive female macaque monkeys being fed an atherogenic diet: females who were normally dominant, but who became subordinate when switched to a different social group, and subordinate females who became dominant when switched, both experienced a significant excess of atherosclerosis compared with those who remained in their original

social position.

The PDS-submissiveness scale, although not necessarily related to the concept of job control, does modestly correlate with the personality trait of neuroticism (Deary, Bedford and Fowkes, 1995). Neuroticism has been linked to symptom reporting behaviour, including reports of chest pain that may result in a diagnosis of angina (Watson and Pennebaker, 1989; Costa and McCrae, 1987; Miller et al, 1991; Stone and Costa, 1990), which agrees with the slightly higher scores we observed in the participants with angina. However, the use of objective criteria to measure disease helps to avoid confusion between personality factors related to symptom reporting rather than true disease (Miller et al, 1996; Stone and Costa, 1990). The statistically significant association found between the objectively-assessed outcome of nonfatal MI and submissiveness indicates that the relationship is different from the neuroticism and angina association.

Over dependence and lack of self-confidence are also aspects of neuroticism (Deary, Bedford and Fowkes, 1995). Both of these PDS traits were related to the increased incidence of angina in the EAS. New angina was defined as a positive response on the WHO angina questionnaire, which only takes account of symptoms, or a diagnosis by a doctor, which may not involve investigation of the coronary vasculature. Angina may, of course, be organic in origin, but on the whole it is not a good proxy for objective health (Costa and McCrae, 1987). Therefore, angina as an outcome must be examined on its own. The finding in the EAS was therefore in accordance with previous research (Stone and Costa, 1990; Booth-Kewley and Friedman, 1987).

Another of the objective outcome measures was ABPI. Although the relationships between the PDS and the change in ABPI over five years were attenuated by adjustment for other factors, this does not necessarily indicate that personality factors are not related to the ABPI. If there is a large proportion of shared variance between two of the covariates in the model, and both are related to ABPI, they may each cancel out the effect of the other, and both will drop out of the equation. Associations have been found elsewhere between personality and objective measures of disease. A study of carotid disease progression in Finnish men, assessed using measures of intima-media thickness, showed that men who had high levels of hopelessness at baseline were more likely to have accelerated atherosclerosis over a four-year period than men with lower levels of hoplessness (Everson et al, 1997). With ABPI, like carotid disease, being a clear marker of disease (Allan et al, 1997), it is important that these associations are tested further.

The final objectively assessed outcome, fatal MI, did not show significant differences on the personality variables. The direction of mean differences for fatal MI was opposite to that seen in nonfatal MI, but the magnitude of the differences and the numbers of fatal MI were very small. A higher base rate of fatal MI would be required in order to examine properly its relationship with personality.

Some bias may have been introduced because the comparison group for each outcome was not necessarily disease-free. For instance, the comparison group for fatal MI included those who had angina diagnosed during follow-up (n=88). In addition, in this age group, even those who did not have a diagnosed

event may have had significant atherosclerosis. In short, the comparison groups may have been contaminated with another effect of the same underlying problem, such as atherosclerosis. This 'dilution' would have made differences between the groups less extreme, biasing the results toward the null and leading to possible under-estimation of effect sizes.

The usefulness of the submissiveness scale scores in this context is pragmatic: to add to the predictive power of models incorporating established risk factors. The 0.5 of a standard deviation lower submissiveness mean scores in women who had a nonfatal MI compared to those who did not is a medium effect size (Cohen, 1992). The RR of 0.59 with a one standard deviation increase in submissiveness, the magnitude of which is commensurate with other personality-CHD studies (Miller et al, 1996), reinforces the additional predictive value of the measure.

It was interesting that the submissive-dominance dimension was associated with cardiac events, and not hostility, especially as previous analyses of peripheral arterial disease and the PDS in the EAS showed that a higher hostile acts score was significantly related to increase severity of peripheral arterial disease (Deary et al, 1994). However, self-reported hostility measures generally show weaker relationships with CHD than interview measures (Miller et al, 1996). For instance, in an investigation of self-reported versus examination-determined hostility, 12 of 21 patient reported themselves to be non hostile on a self-report questionnaire (Cook Medley Hostility Scale), but on examination 20 of 21 were found to be severely hostile (Friedman, 1996). The PDS are self-reported, and

therefore if subjects underestimate their hostility, and hostility is related to CHD, the magnitude of the association will be attenuated. Alternatively, it could be an age effect: hostility has been shown to be a stronger risk factor in younger age groups (Miller et al, 1996; Barefoot et al, 1995), and the EAS is an older cohort.

It is also possible that submissiveness is not protective of MI in younger age groups: the contribution of risk may shift with age, as it does with hostility. The group may be comprised of 'survivors,' if some of the potential submissive participants had died. However, within the 55-74 year age group, the findings can be extrapolated to the relevant wider population with reasonable confidence, because the cohort was a random sample of the general population. It was sampled from all parts of Edinburgh and in all social class groups.

Given the age of the participants, it cannot be ruled out that their submissiveness and hostility scores have been shaped by their social class or working history or medical history. However, the PDS-scale scores were trait measures (Deary et al, 1994), and there is good evidence that personality traits are stable in adulthood (McCrae and Costa, 1990). In addition, because participants with a history of CHD at baseline were excluded, personality changes were not likely to have been caused by the disease or diagnosis. On the other hand, by age 55 most adults have at least moderate atherosclerosis, and it is possible that subclinical disease may have as yet unknown effects on personality. Social class has been shown to have a substantial influence on health (eg., Whitehead, 1992). For instance, a person's experiences throughout life influence many biological variables, including hormonal stress mechanisms which are particularly important

for coronary disease (Brunner, 1997). Therefore, statistical adjustment may not fully account for the intricate ways in which social class is intertwined with home environment, working environment, personality and health. The direction of any bias is difficult to assess, however, and these are complex research problems that are common to most behavioural epidemiological studies. Ideally, personality would need to be assessed in a young study group and continue to be reassessed as the cohort aged and CHD events were being recorded.

10.3 CROSS-SECTIONAL FINDINGS

In the multiple logistic analyses of the NEO-FFI and STAXI scales with CHD, anger was clearly related to the prevalence of events. In men, higher anger-out scores were related to increased prevalence of myocardial infarction: the OR was 1.9. Low agreeableness was not as strongly associated with prevalent MI: the OR was 1.08. To complicate matters, higher anger scores were associated with a lower prevalence of all PAD; and to lower prevalence of all cardiovascular disease (CVD; of which a large proportion had asymptomatic PAD).

The results for women were different from those of the men. Higher anger-control scores were related to a reduced prevalence of angina in women. Raised anger scores, in women's case anger-in, were related negatively to prevalent intermittent claudication. Higher neuroticism scores were positively related to prevalent intermittent claudication. For all CVD categories together, higher anger control scores were related to a lower prevalence of events.

Many of the risk factors were also correlated with both the NEO-FFI and

STAXI, notably NEO-agreeableness with blood pressure and cholesterol; plus STAXI-anger measures with blood pressure in both men and women, and with cigarette smoking in men. Factors such as age, social class and body mass index were also related to blood pressure, as was smoking. In both men and women, systolic and diastolic blood pressure, often cholesterol and ABPI, and sometimes smoking, were associated with the prevalence of CHD; confirming the ubiquitousness of these elements as risk factors, if confirmation were needed.

Why were some of the anger measures positively related to prevalent MI, and others to decreased prevalence of PAD? Other studies of anger and CVD have suggested that the relationship is not simple. For instance, Siegman and Snow (1997) reported that a recalled anger-inducing episode brought on high cardiovascular reactivity levels only when the event was described in a loud and fast voice. Neither the inward reliving of the event nor describing it in a soft, slow voice caused changes in reactivity. This would indicate that only complete expression of anger, with its associated speech patterns, is pathogenic (Siegman and Snow, 1997). In the EAS, anger-out scores *were* associated with prevalent MI in men, although speech style when angry was not assessed, so it is difficult to compare the findings directly.

In the Boston Area Health Study (O'Connor et al, 1995), suppressed anger, as in the EAS (STAXI anger-in), was not associated with nonfatal MI. Type A behaviour as measured on the Framingham Type A scale was related to increased risk of nonfatal MI, but this was markedly reduced after controlling for HDL-cholesterol levels. In men in the EAS, HDL cholesterol was associated with a 49-

96% reduction in risk of prevalent MI with each 1mmol/l rise; in the same model, anger-out had an OR of 1.90 (CI 1.28-2.80). O'Connor and colleagues (1995) stressed the necessity of including HDL cholesterol as a measure. The EAS findings showed its importance too, although adjusting for HDL did not attenuate anger's relationship with prevalent MI, it merely highlighted the fact that *both* factors were contributory.

A similar pattern for anger was observed in the Normative Aging Study, a cohort of older men (Kawachi et al, 1996). Men reporting the highest levels of expressed anger compared to men with lowest appeared to be at three times the increased risk of fatal and nonfatal MI (although confidence intervals were wide and included one: 0.94-10.5). Additionally, however, there appeared to be a dose-response relationship between the level of anger and risk of all CHD - but this outcome included angina.

Differences between men and women in the effect of anger on cardiovascular risk were highlighted by Burns (1995). In men, anger suppressors with high trait anger showed the largest cardiovascular reactivity when harassed, although anger expressors did have generally high reactivity. The interaction between expression style and experience of anger was statistically significant only in men. However, in both men and women, anger expression style interacted with appraisal of the situation. If negative affect concerning the situation was high, cardiovascular reactivity was greater in anger suppressors. The findings suggest that the *interaction* of traits, gender and situation may affect the extent to which anger and anxiety contribute to coronary risk (Burns, 1995). This, too, is shown

in the EAS differences between the factors associated with prevalent CVD in men and women.

Other traits may not be the only mediating factors: social class is also an important consideration. People with lower socioeconomic status score higher on the Cook-Medley hostility scale (Scherwitz et al, 1992; Barefoot et al, 1991). Higher hostile subjects, when involved in a task in which they are harassed, show exaggerated cardiovascular reactivity (Suarez and Williams, 1989; Weidner et al, 1989; Mittelman et al, 1997). If those of higher hostility are also of lower social class, then the relationship between hostility and cardiovascular reactivity may be mediated by socioeconomic status. Mittelman and colleagues (1997) evaluated the influence of years in education (as a measure of social class/status) on the risk of an MI triggered by anger. Those with less education were at increased risk of having an MI that seemed to have been brought on by anger. The difficulty in interpretation is that if episodes of anger were more frequent and MI was also more frequent in the lower social class groups, then the observed association between an anger episode and MI could have been spurious. However, the importance of socioeconomic status for CHD and on anger is undisputed, and it is a factor requiring close attention. Although adjusting for social class in the EAS did not attenuate the relationship between anger and MI, there is a possibility that socioeconomic status had a wider impact - perhaps lifelong - than could be corrected by statistical adjustment.

Thus, there may be confounding factors that mediate the association between anger and CHD risk that cannot be fully accounted for by statistical

adjustment. There also appear to be important interactions between a person's propensity to feel anger (the trait) and situations that increase risk. Consistent findings across epidemiological surveys may therefore be hard to find, if these factors are not taken into account. However, the association between anger-out and prevalent MI, as in the EAS, has been more consistently found than other types of anger with MI (Miller et al, 1996). Findings with cynical hostility have been more mixed. Use of objective disease outcomes, such as the objective definitions used in the EAS, is therefore very important, so that factors associated with 'true' disease and factors associated with symptoms are quite clearly separate.

10.3.1 Other findings with peripheral arterial and carotid disease

There has been very little previous research into personality and indicators of atherosclerosis such as PAD or carotid disease. Previous findings in the EAS suggested that there was some association between severity of PAD and higher PDS hostile acts score, after adjustment for confounding factors, in men (Deary et al, 1994). Joesoef et al. (1989) observed a small univariate association between PAD and hostility, but this was attenuated by adjustment for other factors. Carotid artery disease, like ABPI, as discussed previously, a good indicator of general atherosclerosis (Craven et al, 1990; Salonen and Salonen, 1993; Miller et al, 1996), and the two measures are also strongly related to each other (Allan et al, 1997). A Finnish study found that hostility predicted carotid disease progression over a two-year period (Julkunen et al, 1994), and Stevens et al (1984) also reported an association between carotid disease and hostility. There is reason,

therefore, to expect that higher anger levels would be related to greater prevalence of PAD (including a lower ABPI) in the EAS. This was not the case: in men, *higher* total anger scores were significantly associated with a *lower* risk of being in the prevalent PAD group (OR 0.70), and in women, the odds of having prevalent intermittent claudication *decreased* as anger-in scores went *up* (OR 0.54). In both men and women, higher anger scores were associated with lower risk of having any type of CVD, although this probably reflected the large proportion of subjects with PAD in this category.

10.3.2 Methodological issues

Methodological difficulties may account for the incongruent results. It is possible not only that the cohort when recruited were 'survivors,' but that five years later, those available to complete the second set of personality questionnaires were not a representative group. If people with more severe disease died, and also had higher anger scores, those that were left would have represented neither the most severe disease nor the most extreme anger scores. Alternatively, if anger is more closely associated with the plaque rupture/thrombosis of MI, then we would see that association (which we did, with anger-out and MI in men), but would not necessarily find an association with chronic atherosclerosis such as PAD.

As so many statistical tests were performed, the findings may be due to chance despite 'statistical significance' (Type I error); this is true for both the longitudinal and cross-sectional results. In the longitudinal analysis, the univariate findings with submissiveness were confirmed in the multivariate analysis,

indicating that the result was reasonably consistent. Cross-sectionally, in both sexes some form of increased anger, but none of the NEO-FFI factors, was associated with a lower prevalence of either intermittent claudication specifically (in women, although this was anger-in, which is correlated 0.42 with neuroticism) or any kind of PAD (total anger, in men). This suggests that this observation was not purely chance, but that there may be an unseen confounder or a relationship we do not understand. The associations between anger and prevalent CVD, however, were not consistent between the univariate and multivariate analyses; the associations became *clearer* when adjusted for other risk factors. This may indicate that the associations either were observed by chance, or that the risk relationships are complex, and mediated by other factors.

It is conceivable that people who already knew they had disease would be more reluctant to report higher anger levels, and thus a spurious protective effect of higher anger, such as total anger and all CVD in men, was apparent in those without disease. This, however, cannot account for relationships observed between personality and subclinical PAD.

The five factor and anger measures and prevalent disease were not measured at precisely the same time, which meant that the medical data were more complete than the personality data. This may have led to bias. There were men and women who had disease but did not provide NEO-FFI or STAXI data (for example, 4% of men and 2% of women had prevalent MI, but did not provide cross-sectional personality data; and 9% of men and 5% of women had prevalent angina, but no NEO-FFI or STAXI scores). This may be particulary

relevant for the unexpected direction of some of the observed associations with anger. If those who refused or were unable to fill in a questionnaire would have scored more highly on anger, then the results presented here will have been biased towards the null - underestimating effect sizes - or possibly even shown associations in the opposite direction. However, there may have been bias in the other direction: if the missing anger scores were lower, then any observed effect may have been inflated. The missing scores, though, would have to have been quite extreme to greatly affect the findings, particularly associations that were consistent and statistically significant, such as that between MI and anger-out in men, and neuroticism and intermittent claudication in women. Nonetheless, the direction of the possible bias is difficult to assess, and therefore makes it more important that the cross-sectional findings are interpreted cautiously, with careful attention to the results of other studies.

The anger-in and anger-control scales were calculated on four items each. This may have led to errors in measurement. The classification of intermittent claudication, made by the WHO questionnaire, had fairly low specificity and thus may have included a proportion who do not have substantial disease, possibly attenuating the strength of associations. However, the measurement of subclinical disease by ABPI is objective, so there is less likelihood of misclassification, and may have increased the likelihood of revealing the effect of personality on subclinical disease.

Studies examining PAD and personality are rare, and each finding is very important for establishing patterns. These EAS results are unexpected given the

limited previous studies, including findings in the EAS itself, but without further investigation we will not know if this was chance or an indication of a relationship we don't yet understand. Findings such as Siegman and Snow's (1997) demonstrating the importance of voice volume and speed on physiological reactivity highlight the importance of trait by situation interactions. We could not examine interactions in this phase of the EAS, and it may be necessary to do so before we fully understand the anger findings.

The associations between blood pressure, anger and agreeableness were also confusing. In men, there were positive correlations between anger and blood pressure, but in women, the correlations were negative. There are no reports yet concerning agreeableness, but a meta-analysis of the effects of anger on essential hypertension found that study results were inconsistent (Suls, Wan and Costa, 1995). The authors recommended that studies should not simply look at associations between resting blood pressure and trait anger, and that prospective studies were needed, preferably with ambulatory monitoring of blood pressure. The EAS analysis of blood pressure in this case was none of these things. Therefore, the findings should be interpreted with caution, and may not reflect 'best practice' for this particular association. Over-interpretation could be very misleading. The correlations, too, although statistically significant because of the size of the sample, were not strong (from 0.10-0.20). It may be better to complete longitudinal analysis in this sample later before coming to any conclusions regarding the specific association between trait anger and blood pressure in the EAS.

10.3.3 Interim summary

The personality trait of submissiveness, in the EAS, appeared to protect against incident MI, particularly in women, over a five-year period. Although the pattern in men was similar, statistical adjustment for confounding factors attenuated the association. Trait anger and (low) agreeableness were inconsistently associated with prevalent CVD in this sample. The relationship between anger-out and prevalent MI in men was among the strongest personality/disease relationships, with an OR of 1.90 for each standard deviation increase in anger-out score. However, the relationship was not apparent for women, who showed decreased prevalence of angina and intermittent claudication with higher anger-in and higher neuroticism scores, respectively. Neither men nor women provided evidence that higher anger or low agreeableness related to increased risk of PAD.

In sum, the findings are not straightforward. There is a precedent for the submissiveness and protection from MI found in the longitudinal analysis (in Type A studies looking at the dominance in the TABP, and in animal models), and for the anger-out and risk of MI for men in the cross-sectional analysis, but it appears that conclusions regarding PAD must await further research. Difficulties in interpretation of the cross-sectional findings also show the need for longer-term study. What the results indicate most strongly is that research must continue to include both men and women, given that different factors appear to be important for them. It will be especially important to investigate younger women, who have been somewhat ignored because they seem at low risk until menopause. The

findings also suggest that the five factors, at least in CHD research, may not be as essential to measure as it will be to continue to examine anger and hostility and their specific effects on a person's risk. Further work should also continue into the likely biological mechanism of this association, which would not only help in the understanding of prevention, but which may be important in discovering if there are different personality factors implicated in chronic versus acute CHD. Previous studies examining the biological plausibility of personality/CHD link are discussed below.

10.4 BIOLOGICAL PLAUSIBILITY

An important question for epidemiological studies of the association between personality and CHD is how the connection works. For hostility/anger and CHD, there are a number of plausible biological routes. The hostility may be a marker of an 'inborn structural weakness' that in turn raises the risk of CHD (Suls and Sanders, 1989). It may be part of a general pattern of hyperresponsivity to stressors which accelerates atherosclerosis (Suls and Sanders, 1989; Williams, Barefoot and Shekelle, 1985; Smith and Christensen, 1992). Perhaps it increases a person's vulnerability to stress and disease (eg. Barefoot et al, 1983), or creates a more dangerous personal environment, through a delay in seeking medical attention (Suls and Sanders, 1989), through reducing the number and quality of social supports (Smith and Christensen, 1992), or through increasing unhealthy behaviours such as smoking or alcohol consumption (Leiker and Hailey, 1988; Scherwitz et al, 1992; Siegler et al, 1992; Whiteman et al, 1997). Alternatively, there may be reciprocal relationships between

the cognitive, behavioural and social environments that act in a cycle to increase risk. This 'transactional model' posits that the 'high hostile' persons' belief that others will harm them causes them to act in an antagonistic manner, which elicits retaliatory behaviour, undermining social support and confirming the hostile person's cynical view, while at the same time causing an enhanced physiological response to stressors (Smith and Christensen, 1992). Finally, the relationship may be mediated through hostility's relationship with other classic risk factors, such as hypertension or blood lipid levels (Siegler, 1994). These alternatives are discussed below, after a brief account of the sympathetic system and its functions.

10.4.1 The sympathetic system

The sympathetic division of the nervous system helps control arousal functions (Carlson, 1986). It co-ordinates the body's acute response to stressors and is therefore designed to react rapidly (Ely, 1995). It controls the physiological changes to the stressor that maintain the body's homeostasis (Christensen and Jensen, 1995; Stratakis and Chrousos, 1995). These physiological changes help to divert oxygen and nutrients to the stressed site and to the central nervous system, where they are needed most (Dom and Chrousos, 1993). The reaction is coordinated by the central nervous system (Chrousos, 1992). For instance, in the brain, the hypothalamus, medulla, locus ceruleus and pons may all be involved. They communicate with the peripheral organs such as the adrenal glands, and together they control the release of hormones such as cortisol, epinephrine, norepinephrine and insulin (Chrousos, 1992). These hormones (hereafter referred to as 'stress hormones' or neuroendocrines) induce a

wide range of effects, including the raising of blood pressure and heart rate, and after repeated episodes, structural thickening of the blood vessel wall (Ely, 1995; Folkow, 1982).

Stress, as defined by Selye (1957) "is the state manifested by a specific syndrome which consists of all the nonspecifically induced changes within a biologic system."(p.54). The *state of stress* may therefore be induced by *stressors*, some of which are salient for only one individual (eg., a phobia) and some which are more general (for example, being attacked). The pattern of reaction, however, is common to everyone: the 'emergency reaction' involving the central and peripheral nervous systems (Cannon, 1914), which is often now known as the "fight or flight" response (Selye, 1957).

Sympathetic activity can be assessed indirectly by recording heart rate and pressure. Direct assessment of sympathetic activity may include measuring norepinephrine in plasma or the recording impulses in sympathetic nerves (Christensen and Jensen, 1995). All stressors have a measurable physiological effect: if the reaction is either consistently inadequate or excessive, it may result in diseases (Stratakis and Chrousos, 1995; Levine, 1985) such as hypertension (Johnston et al, 1993; Ely, 1995; Jorgensen et al, 1996) and other forms of CHD (Henry, 1986). This is true for either psychological stressors (Jorgensen et al, 1996) or physiological stressors such as electric shock (Turkkan, Harris and Goldstein, 1989).

Clinical and epidemiological studies have found that the stress hormone cortisol was related to lipid metabolism, hypertension and severity of coronary heart disease (Stout, 1985; Krakoff, Nicholis and Amsel, 1975; Troxler et al, 1977; Herd,

1986). High insulin levels, which are also associated with obesity, hypertriglyceridaemia and diabetes mellitus of adult onset, may cause atherosclerosis, and hyperinsulinaemia is a risk factor for CHD in those even without these concomitant factors (Herd, 1986).

Psychological stress, therefore, may promote atherosclerosis because the associated increases in heart rate and blood pressure (Clarkson, Manuck and Kaplan, 1986) and the secretions of cortisol, epinephrine or norepinephrine (Herd, 1986). For instance, Davis, Gass and Bassett (1981) found that serum cortisol levels were higher in a group of subjects *new* to an exercise test, compared with those who were experienced, when both groups had the same level of fitness and oxygen uptake during the test (that is, the newness was stressful). Increases in serum cortisol were 59% in the experienced, versus 138% in the inexperienced. A study of trainee parachute jumpers found a drop in cortisol, epinephrine and norepinephrine secretions over successive training days, perhaps reflecting the effects of both the improved performance and reduced fear coming with practice (Ursin, Baade and Levine, 1978). Differences in these neuroendocrines have also been shown under a series of laboratory stressors: physical stressors (a hand in cold water) and psychological stressors (mental arithmetic) both caused increases in norepinephrine and epinephrine responses (LeBlanc et al, 1979). Increases in plasma cortisol, greatest in the first sessions of laboratory tasks, were likewise correlated with error rates (Brandenberger et al, 1980). Hospital patients with essential hypertension had similar epinephrine and norepinephrine rises following a mental arithmetic test, whereas there were no differences between hypertensive and normal subjects in the stress hormone levels

(Januszewicz et al, 1979). Longer term differences were also evident: comparisons of two groups of factory workers, one group on a payment-by-results schedule, and the other on a fixed salary payment schedule showed that the non-fixed payment workers had greater urinary excretion of epinephrine and norepinehrine (Timio and Gentili, 1976). This detrimental effect was seen also in assembly-line workers versus non-assembly line workers. The differences between the groups were in the same direction when the workers were switched between conditions: the effect was for the work, not for the worker (Timio, Gentili and Pede, 1979).

The sympathetic response to stress involves the central and peripheral nervous systems and associated hormones. These responses vary in intensity depending on both the person and the stressor. Stressors may be physical or psychological, and reactions to them that are chronically disordered may increase the risk of different manifestations of CHD. There are thus may routes by which psychological factors could influence the risk of CHD, and there have been a number of models proposed to try to make the pathways explicit. These models are discussed below.

10.4.2 Models of biological pathways

10.4.2.1 Structural weakness hypothesis

The issue of an 'inborn structural weakness' (Suls and Sanders, 1989) has been discussed in the context of Type A behaviour. According to this hypothesis, Type A's may have arteries that are more conducive to lesion formation. The behaviour, then, is harmless, but is merely a marker for the genetic predisposition to atherosclerosis (Suls and Sanders, 1989). However, Type A remains a significant risk factor even

after controlling for parental CHD history, and family history does not account for the association between hostility and coronary artery occlusion (MacDougall et al, 1985). Nonetheless, some aspects of Type A behaviour appear to be heritable, although these tend to be related to cardiovascular hyperreactivity (Matthews and Rakaczny 1983), which would incicate that cardiovascular reactivity may be to blame (Suls and Sanders, 1989).

10.4.2.2 Cardiovascular reactivity

The cardiovascular hyperresponsivity model has been researched and discussed in depth. This hypothesis suggests that hostile people have higher levels of cardiovascular and sympathetic system arousal and heightened response to stressors, partly through vigilant attention to their environment and frequent anger episodes that increase levels of atherosclerosis or lead to greater likelihood of cardiac arrhythmias or thrombosis (Suls and Sanders, 1989; Smith and Christensen, 1992; Williams, Barefoot and Shekelle 1985).

The assumption behind this hypothesis is that the exaggerated sympathetic response to stressors increases endothelial injury, accumulation of atheroma and incidence of cardiac arrhythmias (Krantz and Manuck, 1984). The evidence has been gathered using several different measures of anger/hostility: neurotic hostility, hostility, aggression, expressive hostility, and inhibition versus expression of anger (Houston, 1994.). Central to the studies is the environment in which the experiment is conducted, given that anger-related variables almost invariably interact with other personality or situational influences (Houston, 1994). Suarez and Williams (1990)

found that neurotic hostility scores were significantly positively related to increased blood flow in the arm when the subjects were performing a task during which they were harassed by the researcher. There were no increases in forearm blood flow if the task was performed without harassment. Studies using the Cook-Medley Ho Scale (Cook and Medley, 1954) also uncovered the person by situation importance. Hardy and Smith (1983) found that men high in hostility performing a role play that was high-conflict had greater diastolic responses than how Ho scorers, and the effect was not seen in the low-conflict role-plays. A current events debate, too, highlighted this effect (Smith and Allred, 1989). A study of husband-wife interactions showed that the high Ho men had increased systolic blood pressure responses only when trying to influence their wives, rather than when having a simple discussion with them (Smith and Brown, 1991).

Self-disclosure, thought to be stressful for high Ho scorers because it goes against their suspicious nature (Smith 1992), has been shown to increase systolic and diastolic blood pressure responses in high Ho men (Christensen and Smith, 1993). A field observational study using ambulatory blood pressure monitoring in male paramedics showed that the highest diastolic pressure responses were in High Ho subjects during conflicts at work (Jamner et al, 1991). Although there have been some conflicting studies (Kamarck, Manuck and Jennings 1990); Allred and Smith 1991), results generally indicated that high Ho subjects have increased cardiovascular responses only to tasks that were either conflict-oriented, personally revealing, or harassing (Houston, 1994).

Studies examining expressive hostility, such as Potential for Hostility (PH) as

measured on the Structured Interview (SI), have had less consistent findings. This may be because fewer of the PH studies used interpersonal stressors, such as harassment, to test the association (Houston, 1994). Yet expressive hostility as measured on the Buss-Durkee Hostility Inventory (BDHI) had conflicting results even for harassing tasks (Jorgensen and Houston 1988; Suarez and Williams, 1990).

Equally confusing are studies assessing the expression or repression of anger, which sometimes have found that expressors had lower heart rate reactivity to a mental arithmetic task (Mills, Schneider and Dimsdale, 1989); and sometimes found no association at all (Smith and Houston, 1987; Haynes et al, 1978). Provoked or angered men high in expressive hostility carrying out a mental arithmetic task have also been found to have greater systolic and diastolic blood pressure reactivity (Siegman et al, 1992). Siegman (1993; 1997) found that only the full expression of anger, as shown through speech volume, pitch and speed, was associated with cardiovascular reactivity. Neither the experience of anger alone nor its repression had the same negative effects on cardiovascular reactivity. Elaborate theories have been postulated regarding the dependence of increased cardiovascular reactivity on provocation, and on whether the individual is allowed to cope with the provocation in his 'preferred' way (that is, expressive if he is expressive, or repressive if he is repressive; Engebretson, Matthews and Scheier 1989), but this is so complicated that testing is very difficult. The numerous inconsistencies and the methodological complications mean that the cardiovascular reactivity hypothesis may equally be correct, incorrect, or, more likely, interacting with other hypotheses. Although in the meta-analysis of Suls and Wan (1993) the importance of the person-situation

interaction emerged clearly, the cardiovascular reactivity hypothesis alone cannot fully account for the hostility/CHD association (Suls and Wan 1993).

10.4.2.3 Psychosocial vulnerability

This model suggests that as high hostility scores are associated with reports of fewer, less satisfactory social supports and more conflicts, that this increased vulnerability could be leading to the development of disease (Smith and Christensen, 1992; Barefoot et al, 1983; Blumenthal et al, 1987; Hardy and Smith 1988; Houston and Kelley, 1989; Smith and Frohm, 1985; Smith et al, 1988). It is not exclusive of the hyperresponsivity model, in that hostile people may both react more strongly to stressors and also experience them more often. (Smith and Christensen 1992). It may be that coronary-prone individuals actually acquire damaged systems as a result of their reaction to the trauma and their attempts to maintain control (Henry, 1986), as trauma can cause permanently disturbed emotional responses (Krystal, 1978). These disturbed responses may cause an increase in stress hormone levels, which damages the arteries and perhaps even related brain areas such as the hippocampus (Sapolsky, Drey and McEwen, 1984), causing a further worsening in function.

10.4.2.4 Dangerous personal environment

Leiker and Hailey (1988) proposed that hostile people may be at increased risk because of poorer health habits, such as smoking or poor diet (Smith and Christensen, 1992). Type A behaviour, for instance, has been associated with greater smoking (Forgays et al, 1993); Kreitler et al, 1990). Hostility has also been associated

independently with cholesterol intake (Musante et al, 1992); poorer physical fitness, greater likelihood of driving under the influence of alcohol or drugs (Leiker and Hailey, 1988; Houston and Vavak, 1991); smoking and alcohol consumption (Koskenvuo et al, 1988; Shekelle et al, 1983; Smith and Christensen 1992; Siegler et al, 1992; Scherwitz et al, 1992; Musante et al, 1992; Whiteman et al, 1997) and a higher BMI (Siegler et al, 1992; Scherwitz et al, 1992). However, hostility has also been correlated with greater vigorous physical activity and lower resting systolic pressure in men (Musante et al, 1992). This theory could also include other behaviours that influence risk, such as avoiding or delaying seeking treatment because of mistrust of the medical profession, and even receiving poor treatment because of aggressive behaviour towards the health workers (Suls and Sanders, 1989).

Hostility levels have not only been correlated with low social support, greater reactivity and poor health habits, but also to higher total and LDL-cholesterol levels (Dujovne and Houston, 1991), greater platelet reactivity (Markovitz et al, 1996) and increased stress hormone levels (Suarez et al, 1991). These physiological changes can also be considered part of the dangerous personal environment. The pathophysiological consequences of high cholesterol and stress hormones reacting together may be especially dangerous for accelerated atherosclerosis (Williams, 1994). For instance, when sand rats were fed a high cholesterol diet and administered exogenous norepinephrine, atherosclerosis reached the same level in two months as that achieved by the diet alone, in six to eight months (Mikat et al, 1992). The long term studies of Manuck and Kaplan (Manuck et al, 1983) that showed that chronic emotional arousal in monkeys, and the concomitant upset of the sympathetic system,

together with dietary hyperlipidaemia, was extremely effective in inducing atherosclerosis. The stress-lipid combination was a much stronger potentiator than lipids alone.

In studies of humans, the work of Ely and colleagues on police officers in the USA has shown that an individual's predominant behaviour and coping pattern influenced his or her perception of a situation and therefore the physiological response (Ely and Mostardi, 1986). The officers who were hostile also had higher 'life-change' scores, lower coping scores and higher diastolic blood pressure than either the tolerant group or the overall police average. This work, plus the complicated nature of the relationships, highlights the need for studies of stress, behaviour patterns and coping; especially longitudinal prospective studies in high stress occupational groups, which would help assess the health effects of prolonged stress (Ely, 1995). The research question would be whether the behavioural and/or stress hormone factors accurately *predict* cardiovascular disease (Ely, 1995).

10.4.2.5 Brain serotonin

Some studies have indicated that diminished brain serotonin function may mediate the biology/behaviour link between hostility and CHD (Williams, 1994) Serotonin is a neurotransmitter that affects mood, sleep, eating and pain regulation as well as blood vessel constriction (Carlson, 1986). Decreased levels of a metabolite of serotonin have been found in the cerebro-spinal fluid in men with a history of aggression (Brown et al, 1979) and in normal subjects who scored highly on a scale measuring the wish to 'act-out' hostility (Roy, Adinoff and Linnoila, 1988)

Depressed responses to fenfluramine, which increases serotonin release, were inversely correlated with the assault and irritability scales of the BDHI (Coccaro et al, 1989). Evidence from animal studies suggests that a lowered serotonin level in male monkeys increases aggression (Raleigh et al, 1991).

Serotonin affects many factors that relate to CHD. Depletion of brain serotonin increases eating, body weight and adiposity, and increases in serotonin decrease the appetite, food intake and facilitate weight loss in humans (Waldbillig, Bortness and Stanley, 1981; Levine et al, 1989; Williams, 1994). Increasing serotonin in animals causes reduction in their self-regulated alcohol intake (Sellers and Naranjo, 1986), and some studies have shown that enhancing brain serotonin function can help reduce nicotine craving in smokers (Williams, 1994). The above evidence suggests

"that all the harmful biobehavioural characteristics found in hostile persons - the hostility syndrome consisting of increased aggression/irritability, increased sympathetic function, decreased parasympathetic function, increased eating, drinking and smoking - are the result of a single 'lesion,' diminished brain serotonin function" (Williams, 1994, p. 123).

The hypothesis needs further testing, but the implications for CHD prevention are important. Documenting the neurochemical basis of coronary-risk behaviour would be a major step for behavioural medicine, as it would potentially allow preventive pharmacological treatments to be developed (Williams, 1994).

10.4.2.6 Transactional model

This theory expands on the previous models. The logic dictates that the high conflict and low social support experienced by high hostile people may result from

their cynical expectations of others, causing them to act in a confrontational manner, which, in turn, elicits similar behaviour in others (Suls and Sanders, 1989). This, then, confirms the hostile person's cynical view of others and further undermines the hope of social support (Houston, 1994). Within this framework, physiological responses are also exaggerated, and since stressful situations occur more often, health is further affected. As with the other models, testing it is formidably challenging, and research findings therefore cannot fully support or refute the theory (Houston, 1994).

The diagram on page 250 shows putative pathways among personality and CHD risk, and the 'location' of models in relation to their strongest focus - either inside or outside the person. For instance, the structural weakness and brain serotonin hypotheses focus on the internal causes of CHD. Psychosocial vulnerability concentrates on how the external environment may influence risk. The models regarding dangerous personal environment and cardiovascular reactivity include influences from both sides, and the transactional model postulates that all the systems work together to increase risk.

10.4.3 Connections between animal and human studies

There are enough similarities between humans and some non-human primates in gestures and behaviours indicating combative or aggressive mood that groups of animals have been very useful for modelling human anger/hostility (Kaplan, Botchin and Manuck, 1994). For instance, a characteristic threat gesture in macaque monkeys may represent hostility, and if the threat is carried forward into some form of attack, it may be characterized as aggression, and the intensity of the attack as 'anger'

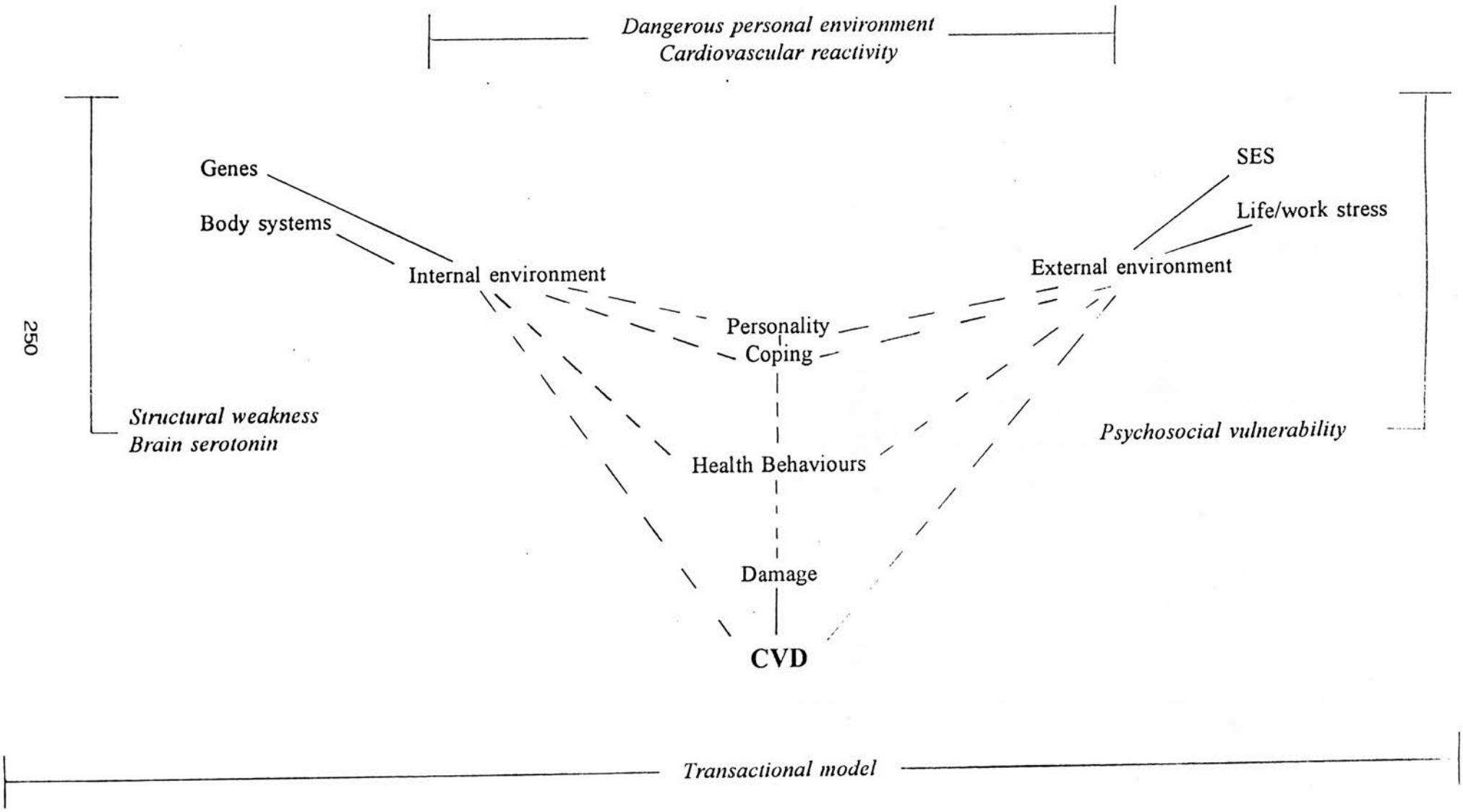

Diagram: Pathways of risk for cardiovascular disease (CVD) and the 'location' of different models of biological mechanisms

SES - socioeconomic status

(Kaplan, Botchin and Manuck, 1994). Macaques also live in complex social groups involving much social interaction (Kaplan et al, 1985; Sade, 1967; 1972; Seyfarth, 1977; Kaplan, Botchin and Manuck, 1994). In addition, they develop atherosclerosis fairly rapidly when fed diets high in saturated fat and cholesterol, and it results in coronary lesions and myocardial infarctions, much the same as it does in humans (Kaplan, Botchin and Manuck, 1994). Premenopausal females, again, like humans, are relatively protected from atherosclerosis (Kaplan et al, 1991).

The social hierarchy of macaque troops is normally relatively stable, and is therefore easily subject to experimental manipulation. This allows the study of physiological consequences of the manipulations for individuals in different hierarchical positions. In three experimental manipulations, it was found that atherosclerosis was accelerated in habitually aggressive (ie. more dominant) animals (Kaplan et al, 1982; Kaplan, Botchin and Manuck, 1994). The atherosclerosis was partly related to adjustments of the sympathetic nervous system made during aggressive encounters, illustrated by the attenuating effect of beta-blockade on the sympathetic response and also the atherosclerosis.

The effect for beta blockers has also been observed in human subjects. Two groups of 16 subjects, one of which was classified as hyperreactive and one hyporeactive, on the basis of heart rate and systolic blood pressure responses to a self-paced, reaction time task, had their heart rate monitored in the field over an eight-hour period (Johnston et al, 1994). Participants were given either a placebo or bisoprolol (a cardio-selective beta-blocking agent). The hyperreactors on placebo were found to have a markedly more variable heart rate than hyperresponders after

adjustment for physical activity. The difference was negligible between the groups who were taking beta-blockers.

Some monkeys were susceptible to atherosclerosis because they were hyperresponsive to stress, regardless of their hierarchical position (Manuck, Kaplan and Clarkson, 1983). Support for this has again been found in human subjects: two studies have shown that hyperreactive heart rate and blood pressure responses to active coping tasks in the lab predicted hyperreactions in the field (Johnston, Anastasiades and Wood 1990; Anastasiades et al, 1991). In short, monkeys appear to be at risk if either they have frequent aggressive encounters in socially unstable conditions, or if they are overly-reactive to stressors (Kaplan et al, 1991; Manuck, Kaplan, Adams and Clarkson, 1988; Kaplan, Botchin and Manuck, 1994); a situation that also seems true for humans. The sympathetic activation may be the mediator between behaviour and atherosclerosis (Kaplan, Botchin and Manuck, 1994). This in turn links to the serotonin hypothesis of autonomic arousal, which could be activated by either frequent encounters or individual differences in brain serotonin levels.

Koudy Williams et al (1991) experimentally manipulated four groups of monkeys, to examine the effect of chronic psychological disruption and diet on dilator responses in the coronary arteries. They found that a group fed a high-cholesterol diet and housed in an unstable group had larger plaques and relative constriction of the coronary arteries in response to acetylcholine, compared with non-atherosclerotic controls. Low cholesterol-diet, unstable groups had smaller plaques, but similar vascular responses. The low cholesterol, high-stability group had both small plaques

and vascular responses, similar to the control group (which was left alone). Differences between the stable and unstable groups persisted despite adjustment for total and HDL-cholesterol concentrations, plaque size, baseline heart rate and blood pressure. Therefore, the study shows that chronic stressors may impair vascular responses of the coronary arteries. The authors concluded that the chronic social disruption may lead to intermediate bodily changes that underlie CHD.

In a Finnish study (Everson et al, 1997), men who showed stress induced blood pressure reactivity and who reported high job demands had greater carotid atherosclerotic progression over four years than men who were less reactive and had fewer demands. The men with at least 20% stenosis or a non-stenotic plaque at baseline, who were also reactive and had high job demand, had 46% greater atherosclerotic progression than the others.

Other studies examined coronary arteries to determine the effect of anger on coronary vasoconstriction. Twelve men with clinical signs of coronary artery disease, who had elected to undergo cardiac catheterization, took part (Boltwood et al, 1993). During the procedure, in which one narrowed and two non-narrowed coronary artery segments were visualized, the subjects were asked to recall an event that had made them angry. It was found that only high levels of anger induced vasoconstriction, and that the vasoconstriction only occurred in the narrowed segments, not the non-narrowed segments. Anger recall has also been shown to reduce left ventricular ejection fraction to a greater extent than exercise or other stressors such as mental arithmetic. This was true for both men with and without CAD, although the anger seemed to be particularly potent in those with CAD (Ironson et al, 1992). Together,

the physiological findings

"both demonstrate the potential pathogenicity of behaviour and identify sympathoadrenal activation as one responsible mechanism in these associations" (Manuck et al, 1995; pp. 279-280), for both monkeys and humans.

10.5 CHAPTER SUMMARY

The EAS analysis of personality and CHD data suggests two main findings: a protective effect on nonfatal MI, particularly in women, of submissiveness over a five-year follow-up; and an increased risk of prevalent MI in men with higher anger-out scores. Both of these factors added to models showing that the ABPI was a strong predictor of future disease, and after that, blood pressure.

The protective effect of submissiveness in women is a new result, although placid and non-hostile men in the Western Collaborative Group Study (Houston et al, 1997) were at reduced risk of death over a 22-year period. As expressive anger has been found fairly consistently to correlate with MI, the EAS finding that this was true for men, at least with prevalent MI, was not surprising. It was notable that low agreeableness did not correlate consistently with prevalent CHD in men or women. It may be that the domain is too heterogeneous.

There are many theories for how a relationship between personality and CHD would be mediated, including the physiological effects of the experience of anger, the deleterious effect of hostility on health choices or on social supports. Transactional models postulate, sensibly, that there is likely to be interaction of many systems that work together to increase risk. Animal models have provided evidence of the effects on the body of circulating stress hormones, and the harmful changes in arteries occurring in response to chronic stress. All the models involve the sympathetic

response system: through hormone/risk factor associations, through the increased heart rate or blood pressure reactivity, through increased reactivity to hostility in others, or through effects on mood or behaviours such as eating or smoking.

The relationship between personality and CHD, therefore, is biologically plausible, and almost certainly involves the sympathetic nervous system. However, there may be social influences, such as poverty or social class, that contribute to risk in a myriad of ways that are not fully understood. Other types of studies will need to continue research into the biological mechanisms, but epidemiological studies can be improved by taking into account a wide range of factors, and using standard measures. In the final chapter, the implications and recommendations for future research will be discussed, based on the review of past research and on the present findings of this study.

CHAPTER 11

Implications, Recommendations and Conclusions

11.1 IMPLICATIONS

The implications of this and other risk factor research in CHD are that the knowledge should be used eventually to help improve prediction and prevention of disease. What the findings about submissiveness and anger in this thesis can do *now* is to help improve prediction of disease, *as long as the status of other risk factors is also known.*

The knowledge may, in future, be extended to prevention. Type A behaviour, for instance, has been researched at the prevention stage: treatment trials have been conducted to test the effect of behaviour modification on the incidence of secondary myocardial infarctions (MI's). Friedman and colleagues (1986), in the Recurrent Coronary Prevention Project, recruited about 1000 patients who had had an MI and who expressed an interest in joining a stress reduction programme. Treatment groups were randomly allocated to a Type A reduction programme plus cardiological care or cardiological care alone. Cognitive and behaviour change, reaction to stress, The Type A pattern itself, and the basic assumptions underlying Type A behaviour were assessed. Reductions in Type A behaviour occurred reliably with intervention; 14% more in this group showed a reduction than in the non-treatment group. Global Type A and hostility were affected and both spouse and laboratory assessments of the participants changed after the intervention. Over four and a half years of follow-up, the

reinfarction rate was nearly 5% in the controls group and about 3% in the behaviour change group, and the two groups did not differ on potentially confounding factors such as medication use or serum cholesterol levels. This indicated that Type A behaviour could be altered and that it led to a reduced rate of reinfarction in cardiac patients. Other studies have demonstrated that the behaviour pattern can change, but did not follow the patients to determine prognosis (Blumenthal et al, 1980; Howard et al, 1986; Gill et al, 1985).

The findings of this thesis, especially concerning submissiveness, do not yet provide enough evidence for consistency of effect for behaviour change programmes to be implemented. It is also unclear whether submissiveness may have a different impact on the risk of other diseases. However, the findings do add valid, objective data to the body of research already in existence. This type of data is very important for behavioural epidemiology. The information on the occurrence of cardiovascular events was collected systematically and exhaustively, leading to a near-complete data set. Subjective events such as angina pectoris were analyzed separately. Therefore, if the same personality questionnaires were administered in a different population, they could be used to improve prediction beyond what would be expected from physical risk factor data alone. The results show that the dimension of submissiveness/dominance predicts future risk of CHD, and that it warrants further study. They also show, in common with previous work, that different aspects of anger are important in disease pathology. They hint that low agreeableness might play a part in risk of CHD, but that the five factor dimensions should be used in conjunction with more specific measures

of anger. Overall, the results showed that there was a small-to-moderate effect of some personality traits on objective measures of cardiovascular health. This is undoubtedly useful knowledge for such a widespread disease. The risk factors were different between the sexes, and both men and women need to be included in future studies, as this has implications for treatment trials and recommendations for disease prevention.

Yet there are difficulties in the area of personality-CHD research. One problem is that epidemiologists have disparate interests and limited funding. Therefore, in epidemiologic and experimental studies of CHD, this has led to a repetition, within discrete subject areas, of the findings from the 1960s and 1970s; that is, researchers have conducted similar research repeatedly (Salonen, 1988). This is partly because it is difficult to obtain funding for large-scale, more inclusive studies, and partly because of separate interests. For example, a study such as this one may examine personality in relation to heart disease, while another is examining life stress, and a third, employment demands. In addition, different aspects of personality have been studied independently, such as Type A, hostility, anger, dominance, neuroticism or depression. Other studies have focussed on personal resources for dealing with stress, such as coping skills or social support. Even studies of the same constructs may not have used the measured them in the same way, leading to inconsistent results and a requirement for yet more research (Greenwood et al, 1996). Therefore, there are two complications of the disparate interests and limited funding: the first is that the many studies examining just one behaviour-related risk (hostility, for instance, or

job stress) do not replicate each other because they have not used the same behavioural measures or disease endpoints. The second complication is that these same studies are unable to test complex models of risk, because they measured only one behavioural element (just life stress, for instance, and not personality, when the two may both have an impact on risk).

A second problem in the field stems from the interpretation of findings:

> "The popular media, stirred by occasional reports in the medical literature, remind us incessantly of the hazards of certain personality types. We are told that Type A people are vulnerable to heart attacks....the hard-driving executive has a heart attack BECAUSE he is pushing for promotion..." (Angell, 1985; p. 1570, emphasis in original).

This study could be considered one of the "occasional reports in the medical literature." The trouble with such findings is that the conclusions drawn by the media, for instance, may be both over-interpretations and over-simplifications of the evidence. Unfortunately, what is often implied when personality or behaviour correlations with disease are reported is that the sick are responsible for having 'bad' lifestyles or personalities (Kaplan, 1995). Evidence regarding the main risk factors is temporarily forgotten. Issues such as the social class gradient in health are also easily sidelined, yet in reality may be inextricably linked to psychological risks. For instance, social class gradient may affect health directly through lack of basic necessities or because patterns of hostility, distrust, loneliness and depression thrive in deprived communities (Kaplan, 1995). Moreover, a person's physiology may be altered through adverse experiences starting in infancy and continuing throughout a lifetime; hormonal stress mechanisms may be particularly important in the social gradient seen in the risk of coronary disease (Brunner, 1997). The

ankle brachial pressure index (ABPI), for instance, was associated with greater deprivation in this study, and indicates that this may be an important source of risk in this population. Social deprivation itself did not emerge as an *independent* risk factor, but it the relationship could conceivably have been mediated by the ABPI. Social standing may have many effects: studies of wild baboons illustrated the adverse biological consequences that are associated with being subordinate (Sapolsky and Mott, 1987). In this thesis the personality/CHD analyses were adjusted for social class and other factors, but this may not have accounted fully for the lifetime of interactions between social class, personality or behaviour.

However, careful interpretation of these 'occasional reports' can still provide us with important information. Researchers need not downplay the importance of social class or physical risk factors such as cholesterol levels, hypertension or smoking, and most do not. Even armed with a substantial amount of data about a person, predicting CHD is inexact (Dembroski and Costa, 1987) and therefore, the extra knowledge is important. This is the real foundation of epidemiological research: to establish the links, to try to replicate findings in different study groups, to find out if certain risks are associated with certain diseases, and if even more information is required, to keep looking. The role of epidemiologists is then to disseminate their findings to allow their clinical colleagues to develop treatment or prevention trials. However, as seen above, some changes in the carrying out of behavioural epidemiology research may be prudent if we are to maximise our knowledge about risk factors for CHD.

11.2 RECOMMENDATIONS

In the first section below, a number of general recommendations for future research on personality and CHD are discussed. In the second section, recommendations are made for future work within the EAS.

11.2.1 General recommendations

(1) To define the constructs, to use standard measures for personality, and to assess objective disease endpoints, preferably in longitudinal studies.
This would allow studies to be more easily compared and causal directions to be better understood. If theory is used effectively, appropriate, rather than convenient, but perhaps inappropriate, personality measures can be chosen (Sheier and Bridges, 1995). This recommendation was made for Type A behaviour when it was being studied widely (Review Panel on Coronary-Prone Behavior and Coronary Heart Disease, 1981) and for hostility (Miller et al, 1996), and is a recommendation that is still relevant and important. Dembroski and Costa (1987) and Miller and colleagues (1996) have shown the importance of assessing disease endpoints objectively. Others have also stressed the importance of being able to accurately quantify socioeconomic status (SES) as well as psychological factors (Greenwood et al, 1996; Scheier and Bridges, 1995; Lenfant, 1996). Careful longitudinal studies examining psychosocial factors are important for establishing basic, sound evidence that will form the foundation for more extensive research.

(2) To test more complex models to find the associations among differnt

behaviour/personality elements.

Researchers such as Steptoe (1989) have highlighted the importance of looking for interactions between factors such as coping, personality, life stress, cardiovascular reactivity and CHD. Although these elements are often closely interrelated, it is impossible to discover whether one measure is better able to predict risk than another unless both are applied in the same study. This would also allow us to discover if measuring two or three elements concomitantly improves prediction of risk even further.

(3) To keep the implications of the research in mind.

When dealing with personality in relation to disease, researchers need to be careful about what they are claiming, as the claims may have an unjustified widespread social impact (Holroyd and Coyne, 1987). Proponents of a behaviour-disease link may find themselves implying, or being interpreted as implying, that the disease is the sole responsibility of the individual . Holroyd and Coyne (1987) suggested a number of guidelines for researchers: if, as in the first recommendation above, the disease endpoints and psychological constructs are clearly defined, and, as in the second, more complex interactions among variables are tested, we may eventually be able to answer the question:

"Under what circumstances, what aspects of health or health-related behavior, and with what practical implications does personality affect health?" (p. 373).

If the answers turn out to be inconsistent or of very limited practical application, then it would be best to suspend investigation of the factors and focus efforts on the practical issue of helping to prevent illness in the ways we know we can, and

treating it once it has arisen.

(4) To examine the effect of personality on other diseases.

The specificity or non-specificity of the factors to a disease is very important. For instance, the EAS suggested that submissiveness is protective against non-fatal MI. This is interesting and may be important, but as soon as it is practicable in terms of numbers, it must also be examined in relation to fatal MI. There was no indication that submissiveness or hostility affected overall mortality in the EAS, but other study groups may be better placed to consider this question. If this is not investigated, we may end up making recommendations for one disease without regard for the impact a factor may have on other diseases.

(5) To continue the investigation of biological mechanisms between personality/CHD associations.

This is also a suggestion that has been advocated elsewhere. For instance, Salonen (1988) recommended that research continue into mechanisms that may link psychosocial and behaviour-related risk factors to CHD, such as coagulation factors, platelet function or lipoproteins. The link between psychological risks for CHD and SES, too, is one that requires a great deal of attention, in order to understand its possible biological underpinnings such as hormonal stress mechanisms (Brunner, 1997; Lenfant, 1996; Anderson and Armstead, 1995), as well as the social and behavioural pathways (Kaplan, 1995; Lenfant, 1996; Anderson and Armstead, 1995). If we can pintpoint where the links are, then there

is hope that they can be broken.

Geneticists have found that a combination of genes may influence a wide range of behaviours (eg., Plomin, Owen and McGuffin, 1994; Bouchard, 1994). Eventually it may be possible to link the genes for behaviour with the genes for disease, leading to improved prevention and treatment. This is the kind of hope expressed by Williams (1994) for serotonin: genetic levels of the neurotransmitter are predetermined, and these levels lead to certain behavioural propensities, exacerbating both the initial effect of the serotonin and influencing risk of CHD. Yet with appropriate drug treatment, this set of complex interactions could be controlled, and their effect on CHD risk attenuated.

(6) To study both men and women, and diverse populations. This recommendation has been made again and again in reviews (eg. Review on Coronary-Prone Behavior and Coronary Heart Disease, 1981; Matthews, 1988) and meta-analyses (eg. Booth-Kewley and Friedman, 1987; Miller et al, 1996). This will be especially important if a consistent effect is found in one population, which would provide both a reason and impetus for studying the association in more diverse groups. If findings are different between study populations, this has important implications for prevention and treatment.

In short, there is a need for a close integration between disciplines and for co-operation in finding and selecting appropriate, standard and reliable measures. This may help us get away from focussing on aspects of behaviour or personality that are either too narrow, too wide or poorly defined (Sheier and Bridges, 1995). If consistent effects continue to be found, or factors are found to interact, this will

form a sound basis for improving the prevention and/or treatment for CHD. Only with a collective effort can research results have a meaningful impact on our knowledge of how to predict and prevent CHD. The diagram in chapter 10 (p. 249) shows some of the ways that the factors may interact, and there may be many more. The numerous putative pathways and their inter-connections reinforce the need for collective, cross-disciplinary effort.

11.2.2 Recommendations for EAS

Within the EAS itself, there are ways in which the behavioural research may be taken forward:

(1) The medical follow-up and extensive collection of CHD data must contine. This information is important, and essential for the future analysis of both physical and psychological risk factors for CHD.

(2) The ABPI is very important assessment. It is objective and a good indicator of generalized vascular disease. The change in ABPI over ten years, and its association with psychological factors, will be very interesting; statistical power will be stronger at the analysis of the 10-year follow up.

(3) Other psychosocial measures could be applied to the study group, including measures of social support, coping and life stress. More complex modelling of these psychological factors and their relationship both to disease and to other risk factors would be instructive, especially as the EAS is longitudinal and uses well-defined, objective measures of disease.

(4) Longitudinal analysis of NEO/STAXI and events should be carried out.

This will help uncover if there is evidence for temporal associations as well as cross-sectional associations between these factors and objective measures of disease. Long-term follow-up of the PDS-disease associations will also be of great interest, especially because the PDS were administered at baseline instead of at a later point in the study.

11.3 SUMMARY OF THESIS

CHD is an important cause of morbidity and mortality in this country and worldwide. The established risk factors cannot fully account for incidence of the disease, but Type A behaviour and its hostility element have been linked to increased risk. However, there are many different ways to measure Type A, hostility and anger, and this may account for inconsistent research findings.

The Type A concept was developed through observations by doctors, not from theory by psychologists: the groups worked independently. The theory of personality has changed a great deal since the beginning of the century, and personality measurement has been further developed and has been a strong focus in recent research. The five factor model (FFM), which identified five basic personality dimensions, was refined using factor analytic techniques that were developed in the second half of this century. Any person can be described on the continuum of each of the five factors: neuroticism, extraversion, openness, agreeableness and conscientiousness. This model has great applicability to epidemiology, where precise measurement is vital. It can be used effectively in personality and health research, especially in studies like the EAS.

The FFM dimensions are, however, broad. Past behavioural research in CHD found a link between the narrower dimensions of anger/hostility and increased risk. Therefore, research addressing these narrower facets in relation to disease is also necessary.

In this thesis, dimensions of hostility and dominance/submissiveness were studied prosepectively in relation to CHD incidence in both men and women. The five factors, along with specific anger measures, were examined in relation to prevalent CHD.

The results showed that personality trait of submissiveness in the 55-74 year age group, particulary in women, was a protective factor in the incidence of MI. Dominance, although not a completely opposite measure, appeared to increase risk.

Lack of self-confidence and over-dependence, which were highly correlated with neuroticism, predicted a higher incidence of angina in both men and women. This highlighted the importance of the separation of subjective outcomes, which are often associated with neuroticism, and objective outcomes, which are associated with different personality variables. Higher neuroticism scores were linked with prevalent intermittent claudication in women.

Higher anger scores as measured on the STAXI were linked with a variety of prevalent CHD outcomes: particularly anger-out with myocardial infarction in men. Increased total anger was related to greater deprivation and smoking in both men and women. Increased anger scores (anger-in and anger-control in women, and total anger in men), however, were associated with a decreased likelihood of

prevalent peripheral arterial disease and angina.

The FFM measure of agreeableness appeared to have some links with CHD in the EAS, but more specific measures of anger had more relevance. PDS measures did not predict the ankle brachial pressure index (ABPI) or change in ABPI over five years, once tested in multivariate models.

Therefore, despite adjustment for confounders, there were still modest independent links between personality and prevalent and incident disease. These findings indicate that careful measurement of personality may help predict who is at greater at lesser risk of CHD, if their status on other risk factors is also known.

Future research would benefit by including a broader range of factors within each study. This would avoid a large number of similar, but not exactly the same, studies taking place in parallel, each providing a limited amount of information. In particular, the influence of SES on health throughout the life span is important, as SES may have an impact on many biological factors that predispose to CHD. Thus, research would benefit from the testing of more complex models, and further study of the biological mechanisms of the association. This requires continued co-operation between disciplines such as epidemiology and psychology, use of standard measures and careful definition of disease endpoints.

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APPENDIX A: DOCUMENTATION RELATING TO THE BASELINE SURVEY

Northern General Hospital

FERRY ROAD, EDINBURGH EH5 2DQ

Telephone: 031 332 2525

Ref: MF/GS - MCO/229

15th October, 1986.

Doctor F.G.R. Fowkes,
University Department of Community Medicine,
Medical School,
Teviot Place,
Edinburgh,
EH8 9AG.

Dear Dr. Fowkes,

I write with reference to your recent application to the Ethics of Medical Research Sub-Committee for Medicine and Clinical Oncology entitled "Epidemiology of Peripheral Arterial Disease. I Cross Sectional Study".

I write to inform you that this application was given ethical approval at the last meeting of the Sub-Committee.

Yours sincerely,

Martin Farrar,
SECRETARY.

Northern General Hospital

FERRY ROAD, EDINBURGH EH5 2DQ

Telephone: 031 332 2525

Ref: JW/GS MCO/146/87.

26th January, 1988.

Doctor F.G.R. Fowkes,
Department of Community Medicine,
University of Edinburgh,
Medical School,
Teviot Place,
Edinburgh,
EH8 8AG.

Dear Doctor Fowkes,

I write with reference to your recent application to the Ethics of Medical Research Sub-Committee for Medicine and Clinical Oncology entitled "5 year Cohort Study of Epidemiology of Peripheral Arterial Disease".

I write to inform you that this application was given ethical approval at the last meeting of the Sub-Committee.

Yours sincerely,

Miss J. Jieckowicz,
Secretary.

st Invitation Letter

EDINBURGH ARTERY STUDY

A–2

ART FOUNDATION

Department of Community Medicine
Medical School
Teviot Place
Edinburgh EH8 9AG

Tel: 031–667 1011 Ext: 2489
Tel: 031–668 3848 (24hr Answering Service)

University of Edinburgh

itle& &firstnames& &surname&
ddln1&
ddln2&
ddln3&
ddln4&

date&

ear &title& &surname&,

EDINBURGH ARTERY STUDY

ou may know that heart disease, strokes and hardening of the rteries in the legs are very common in Scotland causing many eaths and pain for a lot of people. The British Heart Foundation as recently given some money to the University of Edinburgh to tudy these diseases and we are writing to ask if you would be repared to help with the research. We are working with onsultants in the Edinburgh Royal Infirmary. About 2000 people n Edinburgh will be taking part.

ou may already have read about this project in the Evening News, his being the first large study of its kind in the United ingdom. There is an extract from the Evening News on the back of his letter.

hould you wish to volunteer to take part in the project, you ould have a medical examination at a clinic in the University on he morning only, after which refreshments will be served. The esults of the examination will be sent in confidence to your octor. If you have trouble getting time of work please telephone r write to the above address.

e do hope you will be able to help us with this research. you ill benefit by having a comprehensive medical check-up articularly for heart disease, high blood pressure, diabetes and ardening of the arteries. You will also be helping to combat iseases which are particularly serious in Scotland.

ours sincerely,

r. F G R Fowkes
enior Lecturer

&practice&
&paddln1&
&paddln2&
&paddln3&
&paddln4&

ease fill in and return the slip in the prepaid envelope and we
ll send you more details of the study and your involvement.

you have any queries please contact Pam Farquhar (668 3848).

&recno&

me: &title& &firstnames& &surname&

ldress: &addln1& &addln2& &addln3& &addln4&

am willing to participate in the research study. Please send me
rther details and an appointment for my medical examination.

ease tick:

| I will be travelling by private car, or bus, to and from the clinic (Bristo Square). I understand that I will have my expenses reimbursed at the clinic.

OR

| I would like voluntary transport arranged if possible.

OR

| I am physically unable to attend the clinic (Bristo Square) and want to have the examination at home.

EDINBURGH ARTERY STUDY

HEART FOUNDATION

Department of Community Medicine
Medical School
Teviot Place
Edinburgh EH8 9AG

Tel: 031–667 1011 Ext: 2489
Tel: 031–668 3848 (24hr Answering Service)

University of Edinburgh

title& &forename& &surname&
addln1&
addln2&
addln3&
postcode&

date&

ear &title& &surname&,

EDINBURGH ARTERY STUDY

hank you for volunteering to participate in our study. We should be rateful if you would attend the Richard Verney Health Centre, Bristo quare, ground floor at:

.00 a.m. on &day&

efreshments will be served from 11.30 a.m. at the end of the roceedings.

our medical examination:

hen you arrive at the clinic a nurse will tell you about the morning's xaminations. A blood sample will then be taken, after which you will be iven a sweet drink (if you are not diabetic). You will be given a uestionnaire which you can fill out in the Lounge Area between xaminations. You will have an ECG (to check your heart), and easurements of height, weight and blood pressure. You will also have a pecial test for hardening of the arteries in your legs. Each leg in urn will have a cuff wrapped firmly round it just above the knee. This ill be inflated for four minutes, then released and the blood pressure aken at the ankle. You will also be asked to give a sample of urine. A mall blood sample (to test for diabetes) will be taken two hours after he sweet drink.

hat happens to the results.

he results of your medical examination will be sent in confidence to our doctor. Unless there is any urgency (in which case you will be ontacted), you should ask your doctor in about 3 months time if you ant to discuss these.

reparation for the examination.

The diabetes test does not work if you have eaten any food or drunk certain liquids, so please do not eat or drink anything from 11 p.m. the previous night except for water, or a cup of tea or coffe in the morning without milk or sugar. If you are a diabetic this does not apply.

Please do not smoke for 2 hours before the examination.

Please write down the names of any medicines you are taking, or bring them with you to the examination.

If you wear spectacles for reading, please bring them with you.

Since you will have an ECG machine attached for a few seconds to your chest, ladies should wear a skirt and blouse/jumper rather than a dress.

If you don't think you will be able to produce a small urine sample while at the clinic, could you please bring a recent sample in a small clean container and hand it in as soon as you arrive.

f you asked for voluntary transport please be ready for a minibus to ick you up from 8.00 a.m. You will be returned home by minibus, leaving ne clinic at about 12.30 p.m.

f you have any queries please do not hesitiate to phone Pam on 668 348.

e are very grateful for your co-operation.

ours sincerely,

G R Fowkes
enior Lecturer

EDINBURGH ARTERY STUDY

CONSENT FORM

PURPOSE OF THE RESEARCH

The purpose of this research is to measure the state of your arteries using special blood pressure techniques, and to find out how this is affected by the make-up of your blood, the condition of your heart, your diet, alcohol consumption, and smoking habits. By finding out what harms arteries, we can take steps to prevent disease in the future.

YOUR MEDICAL EXAMINATION

This will begin with an explanation by a team member of the morning's proceedings. A blood sample will then be taken and you will be given a sweet drink (if you are not diabetic). You will be given a questionnaire which you can complete during the morning. You will have an E.C.G. (to check your heart), measurements of height, weight and blood pressure, and a special test for hardening of the arteries in your legs, during which a cuff will be wrapped very firmly around each leg just above the knee for four minutes. You will also be asked to give a sample of urine. A small blood sample will be taken after two hours. There will be refreshments at the end of the morning.

WHAT HAPPENS TO THE RESULTS

The results of your medical examination will be sent in confidence to your doctor. You should contact your doctor in about 3 months time if you want to discuss these.

The results of the research will be published in medical journals and will appear only in the form of statistics from which it will be impossible to identify you as an individual.

CONSENT

I have read the above and understand what is involved in my participation in this research. I know that I can readily withdraw from the medical examination at any point if I so wish. I also understand that the study has been given ethical approval by a Medical Ethics Sub-Committee of the Lothian Health Board. I realise that no liability is accepted by the research team in the proper execution of their work.

I give my consent to the research team carrying out a medical examination on me as described.

Name (Capitals) .....

Address .....

Date .....

Signature .....

9.20

EDINBURGH ARTERY STUDY.

HEIGHT AND WEIGHT RECORDING FORM & VENEPUNCTURE

SUBJECT NAME: .....

RECORDER: 1: M.A. 2: E.K. 3: F.S. 4: A.R. 5: J.D. 6: E.C.

	Yes	No
Did the patient fast overnight?	☑	☐

VENEPUNCTURE (1):

	Yes	No
Has patient had jaundice in the last year?	☐	☑
Has patient had serum jaundice?	☐	☑
Is patient a diabetic?	☐	☑

Was venepuncture normal? (1) 18 mls. Larm

Was venepuncture difficult/slow? 2

Was venepuncture not possible? 3

HEIGHT: (without shoes) 166.0 cm

WEIGHT: (without coat and shoes) 62.9 kg

VENEPUNCTURE (2):

Was venepuncture normal? (1) Larm. 12 mls. Dawes + Lowe

Was venepuncture difficult? 2

Has urine sample been given?

EDINBURGH ARTERY STUDY

BLOOD PRESSURE AND PERIPHERAL PULSE PALPATION RECORDING FORM

SUBJECT NAME: ...../...../.....

RECORDER: 1: M.A. 2: E.K. 3: F.S. 4: A.R. (5: J.D) 6: E.C.

PALPATION OF PULSES:	Present	Absent
right dorsalis pedis	✓	
left dorsalis pedis	✓	
right posterior tibial	✓	
left posterior tibial		✓
right femoral	✓	
left femoral	✓	

RESTING PRESSURE (Couch flat, 1 pillow)		Observed	Zero
RIGHT or LEFT arm	systolic mm Hg	158	
	diastolic	90	24

RESTING SYSTOLIC ANKLE PRESSURES	Observed	Zero
right ankle mm Hg	187	37
left ankle	204	57

REACTIVE HYPERAEMIA		
right ankle	180	36
left ankle	183	40

COMMENTS; Any deviation from standard procedures:

E D I N B U R G H A R T E R Y S T U D Y

QUESTIONNAIRE

E INFORMATION IN THIS QUESTIONNAIRE IS HIGHLY CONFIDENTIAL AND IS PART OF MEDICAL RESEARCH STUDY

e information you give in this personal health record will be treated as rictly confidential and will be available only to your own doctor and the udy team. The results of the research will appear only in the form of neral statistics from which it will be impossible to identify you as an dividual.

ease complete the following:

RNAME: .....

RENAMES: .....

TE: .....

you have any difficulties in answering some questions you will have a ance to discuss these later with a member of the study team.

ANK YOU FOR YOUR CO-OPERATION IN THIS STUDY. THE FINDINGS WILL HELP TO PROVE HEALTH IN SCOTLAND.

IS IMPORTANT TO ANSWER ALL THE QUESTIONS CAREFULLY. PLEASE TAKE YOUR ME.

ere is some evidence of a relationship between health and other factors ch as exercise, occupation, education, diet etc. In order to compare our ta with national figures and other research work, we are interested to ve the following details about yourself.

RSONAL HISTORY

Please tick one box: Male ☐ 1 Female ☐ 2

Enter your date of birth: Day ☐☐ Month ☐☐ Year ☐☐

Please tick the box showing your present marital status:

- Married (or equivalent) ☐ 1
- Single ☐ 2
- Widowed ☐ 3
- Divorced or separated ☐ 4

UCATION

What is the HIGHEST level of education you and your spouse or ex-spouse have completed? Please tick boxes as appropriate.

	Yourself	Spouse or Ex-spouse
University/college degree course	☐ 1	☐ 1
Other professional or technical qualification after leaving school	☐ 2	☐ 2
Secondary School	☐ 3	☐ 3
Primary School	☐ 4	☐ 4

ID EMPLOYMENT

What is your employment status at the moment? Please tick boxes as appropriate.

- Employed, full-time ☐ 1
- Employed, part-time ☐ 2
- Unemployed ☐ 3
- Retired ☐ 4
- A housewife (full-time) ☐ 5
- Other, please specify ☐ 6

lease complete questions 6 and 7 as appropriate for yourself and your pouse or ex-spouse.

YOURSELF	YOUR SPOUSE or EX-SPOUSE

) Please give the name of your present job and describe what you do as fully as possible. If unemployed or retired, do not complete this question, BUT PROCEED TO QUESTION 7.

.....	.....
.....	.....
.....	.....
.....	.....

) What business or industry is this in?

.....	.....

) In this job are you?

self-employed	☐	foreman	☐	self-employed	☐	foreman	☐
manager	☐	other	☐	manager	☐	other	☐

) In this job do you supervise/employ?

25 or more people	☐	25 or more people	☐
fewer than 25 people	☐	fewer than 25 people	☐
no-one	☐	no-one	☐

YOURSELF	YOUR SPOUSE or EX-SPOUSE

a) Please give the name of the job you have done for the longest period of your life, and describe what you did as fully as possible. (If the answer is the same as in Question 6 above, write SAME)

.....	.....
.....	.....
.....	.....
.....	.....

b) What business or industry was this in?

.....	.....

c) In this job were you?

self-employed	☐	foreman	☐	self-employed	☐	foreman	☐
manager	☐	other employee	☐	manager	☐	other employee	☐

d) In this job did you supervise/employ?

25 or more people	☐	25 or more people	☐
fewer than 25 people	☐	fewer than 25 people	☐
no-one	☐	no-one	☐

OR OFFICE USE ONLY

C.

SMOKING

Smoking has been linked with many health problems. It is important that you answer the following section as accurately as possible. Please tick appropriate boxes.

8(a) Do you smoke at present? Yes ☐ No ☐
IF NO, PROCEED TO QUESTION 8(f)

(b) What do you usually smoke now?

cigarettes Yes ☐ No ☐

pipe Yes ☐ No ☐

cigars Yes ☐ No ☐

(c) How many do you usually smoke now?

cigarettes per day ..... cigarettes

oz. tobacco per week ..... oz.

cigars per week ..... cigars

(d) For how many years during your life have you smoked cigarettes? ..... years

(e) How many cigarettes have you smoked on average per day during the period you have smoked? ..... cigarettes
NOW PROCEED TO QUESTION 8(k)

(f) Have you ever smoked regularly? Yes ☐ No ☐
IF NO, PROCEED TO QUESTION 8(k)

(g) What did you usually smoke?

cigarettes Yes ☐ No ☐

pipe Yes ☐ No ☐

cigars Yes ☐ No ☐

(h) How much did you smoke on average while you were a smoker?

cigarettes per day ..... cigarettes

oz. tobacco per week ..... oz.

cigars per week ..... cigars

(i) For how many years did you smoke cigarettes? ..... years

(j) If you smoked cigarettes, how long is it since you finally gave up? ..... years .....months

(k) Is any other member of your household a smoker? Yes ☐ No ☐

EDICAL HISTORY

e should now like to ask you questions about your health, illnesses you ave had in the past, and how you are feeling now. Please tick appropriate oxes.

. Have you ever been told by a doctor that you have or have had any of the following?

	Yes	No
Hardening of the arteries in the legs	☐	☐
Angina	☐	☐
Heart attack (coronary thrombosis, myocardial infarction)	☐	☐
High blood pressure	☐	☐
Stroke	☐	☐
Diabetes (sugar disease)	☐	☐
Bronchitis	☐	☐
Tuberculosis	☐	☐
Asthma	☐	☐

0. Are you on any regular medical treatment from a doctor as follows?

	Yes	No
Drugs to lower blood pressure	☐	☐
Diuretics (water tablets)	☐	☐
Insulin injections	☐	☐
Tablets for diabetes	☐	☐
Other treatments? Give names if possible.	☐	☐

.....

.....

.....

.....

HEST PAIN

11(a) Do you ever get pain or discomfort in your chest?
IF NO, PROCEED TO QUESTION 12

Yes ☐ No ☐

(b) Do you get this pain or discomfort when you walk uphill or hurry?
IF NO, PROCEED TO QUESTION 11g

Yes ☐ No ☐

(c) Do you get it when you walk at an ordinary pace on the level?

Yes ☐ No ☐

(d) When you get any pain or discomfort in your chest what do you do?

Stop ☐

Slow down ☐

Continue at the same pace ☐

(e) Does it go away when you stand still or sit down?

Yes ☐ No ☐

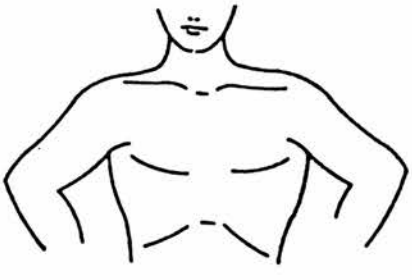
(f) How soon?

10 minutes or less Yes ☐ No ☐

more than 10 minutes Yes ☐ No ☐

(g) Where do you get this pain or discomfort? Mark the place(s) with 'X' on the diagram.

RIGHT



LEFT

12(a) Have you ever had a severe pain across the front of your chest lasting for half an hour or more?

Yes ☐ No ☐

(b) What was the cause? .....

FOR OFFICE USE ONLY A: GRADE MI:

UGH

(a) Do you usually cough several times first thing in the morning in the winter? (Ignore clearing throat or single cough)

Yes ☐ No ☐

b) Do you usually cough during the day or night in winter? (Ignore the occasional cough)

Yes ☐ No ☐

c) If yes to (a) or (b), do you cough on most days for at least three months each winter?

Yes ☐ No ☐

LEGM (SPIT)

(a) Do you usually bring up any phlegm (spit) from your chest first thing in the morning in the winter?

Yes ☐ No ☐

b) Do you usually bring up any phlegm from your chest during the day, or at night, in the winter?

Yes ☐ No ☐

c) If yes to (a) or (b), do you bring up phlegm like this on most days for as much as three months each year?

Yes ☐ No ☐

EATHLESSNESS

(a) Are you troubled by shortness of breath when hurrying on level ground or walking up a slight hill?

IF NO, GO TO QUESTION 16

Yes ☐ No ☐

b) Do you get short of breath walking with other people of your own age on level ground?

Yes ☐ No ☐

c) Do you have to stop for breath when walking at your own pace on level ground?

Yes ☐ No ☐

OR OFFICE USE ONLY

GRADE

HEEZING

5(a) Have you had attacks of wheezing or whistling in your chest at any time in the last 12 months?

Yes ☐ No ☐

b) Have you ever had attacks of shortness of breath with wheezing?

Yes ☐ No ☐

c) If yes to (b), is/was your breathing absolutely normal between attacks?

Yes ☐ No ☐

d) Have you at any time in the last 12 months been woken at night by an attack of shortness of breath?

Yes ☐ No ☐

LEG PAIN

17(a) Do you get a pain in either leg on walking?
IF NO, GO TO QUESTION 18

Yes ☐ No ☐

(b) Does this pain ever begin when you are standing still or sitting?

Yes ☐ No ☐

(c) Do you get this pain in your calf (or calves)?

Yes ☐ No ☐

(d) Do you get it when you walk uphill or hurry?

Yes ☐ No ☐

(e) Do you get it when you walk at an ordinary pace on the level?

Yes ☐ No ☐

(f) Does the pain ever disappear while you are still walking?

Yes ☐ No ☐

(g) What do you do if you get it when you are walking?

Stop 1 ☐

Slow down 2 ☐

Continue at same pace 3 ☐

(h) What happens to it if you stand still?

Usually continues for more than 10 minutes 1 ☐

Usually disappears in 10 minutes or less 2 ☐

18. Have you ever had surgery on the arteries of your legs other than for varicose veins?

Yes ☐ No ☐

Please specify .....

19. Have you ever had surgery to remove

toes? Yes ☐ No ☐

leg below the knee? Yes ☐ No ☐

leg above the knee? Yes ☐ No ☐

FOR OFFICE USE ONLY

I.C. GRADE

OTHER MEMBERS OF YOUR FAMILY

20. Please tick the appropriate boxes for other members of your family if they have been diagnosed as having any of the illnesses below:

Illnesses	Father	Mother	Any brother or sister	Any son or daughter
Angina	☐	☐	☐	☐
Stroke	☐	☐	☐	☐
High blood cholesterol level	☐	☐	☐	☐
Diabetes (sugar disease)	☐	☐	☐	☐
Hardening of the arteries in the leg/claudication	☐	☐	☐	☐
Thrombosis/embolism	☐	☐	☐	☐
High blood pressure	☐	☐	☐	☐
Heart attack	☐	☐	☐	☐
If died from heart attack, at what age?	....Yrs	....Yrs	....Yrs	....Yrs

PHYSICAL ACTIVITY

The following section gives examples of the sort of activities you might do or may have done REGULARLY.

LIGHT activity	MODERATE activity	STRENUOUS activity
Ballroom dancing	Badminton	Basketball
Bowling	Cricket	Competitive cycling
Light do-it-yourself	Cycling (include to and	Competitive swimming
Light gardening	from work, to shops etc)	Competitive running
Horse riding	Heavy do-it-yourself	Field sports (such as
Sailing	Golf	rugby, soccer, hockey)
Walking (include to and	Jogging	Training for strenuous
from work, to shops etc)	Swimming	sport
Yoga	Tennis	Squash
And other activities of similar intensity. Please specify others you have done.	And other activities of similar intensity. Please specify others you have done.	And other activities of similar intensity. Please specify others you have done.
.....	.....	.....
.....	.....	.....

21. In a typical week during the last year, on how many occasions would you take part FOR MORE THAN 20 MINUTES EACH TIME:

Insert 'None' if appropriate

in LIGHT physical activity?	in summer	..... times
	in winter	..... times
in MODERATE physical activity?	in summer	..... times
	in winter	..... times
in STRENUOUS physical activity?	in summer	..... times
	in winter	..... times

22. In a typical week, when you were 35-45 years old, on how many occasions would you take part, FOR MORE THAN 20 MINUTES EACH TIME:

Insert 'None' if appropriate

in LIGHT physical activity?	in summer	..... times
	in winter	..... times
in MODERATE physical activity?	in summer	..... times
	in winter	..... times
in STRENUOUS physical activity?	in summer	..... times
	in winter	..... times

23. Which of the following best describes your daily work or other daytime activity at the present time?
Please tick one box only.

I am usually sitting during the day and do not walk about much	☐	eg. office workers, drivers
I stand or walk about quite a lot during the day, but do not have to carry or lift things very often	☐	eg. housewives, shop assistants
I usually lift or carry light loads and have to climb stairs and/or hills often	☐	eg. postmen, packers
I do heavy work and carry heavy loads	☐	eg. building, mining workers, agricultural workers

24. Which of the following best described your daily work or other daytime activity WHEN YOU WERE 35-45 YEARS OLD?

I usually sat during the day and did not walk about much	☐	eg. office workers, drivers
I stood or walked about quite a lot during the day, but did not have to carry or lift things very often	☐	eg. housewives, shop assistants
I usually lifted or carried light loads and had to climb stairs and/or hills often	☐	eg. postmen, packers
I did heavy work and carried heavy loads	☐	eg. building, mining heavy workers, agricultural workers

36. SALT

(a) How much salt is added in your cooking? (please tick one)

None	1 ☐
A little	2 ☐
A lot	3 ☐

(b) Do you add salt to your meals at the table?

No	1 ☐
When the food is not salty enough	2 ☐
Almost always before tasting	3 ☐

ALCOHOL

36(a) Think back carefully over the last seven days. Please write in exactly what alcoholic drinks you have consumed on each day during the past week and enter in table below:

1. the number of pints of beer, lager, shandy, cider, stout etc.
2. the number of single glasses of whisky, vodka, gin, rum etc.
3. the number of single glasses of Martini, port, sherry or wine etc.

(Try to remember where you were and who you were with on each day. This may help you remember what you have had to drink).

	1 Pints of beer etc	2 Single Glasses of whisky etc	3 Single Glasses of martini etc
Monday	☐	☐	☐
Tuesday	☐	☐	☐
Wednesday	☐	☐	☐
Thursday	☐	☐	☐
Friday	☐	☐	☐
Saturday	☐	☐	☐
Sunday	☐	☐	☐

(b) Would you say that last week was fairly typical of what you usually have to drink in a week?

Yes ☐ No ☐

(c) If last week was not typical, would you normally drink more or less in a week?

More ☐ Less ☐

37. Think about the last time you had a drink. Exactly how much did you drink on that occasion? Please insert the numbers in the boxes.

How many pints of lager, cider, shandy, stout etc. did you drink? ..... Pints

How many single glasses of whisky, vodka, gin, rum or other spirits did you drink? ..... Glasses

How many single glasses of martini, port, sherry or wine did you drink? ..... Glasses

26. The next section contains descriptions of how you may have felt, thought, or acted during <u>most of your life.</u> Below each statement there are four words or phrases; choose the <u>one</u> which best describes you <u>for most of your life</u> and draw a circle round it.

<u>EXAMPLE</u>

(a) I have enjoyed being with other people.

Nearly always (Often) Seldom Never

The first example would mean that most of your life you have often enjoyed being with other people.

(1) I would have liked to get my own back on someone. (HT) *

Very often Often Seldom Never

(2) I have been content to act in a very humble way. (MIN)

Never Seldom Often Nearly always

(3) I have thought that people will tell the truth, even if it gets them into trouble. (DO)

Nearly always Often Seldom Never

(4) I have felt as capable as other people. (LSC)

Never Seldom Often Nearly always

(5) When I've wanted to have a row with someone, I have done so. (HA)

Nearly always Often Seldom Never

(6) I have preferred to take a lot of advice before doing anything. (DEP)

Never Seldom Often Nearly always

(7) I have felt like telling people to go to blazes. (HT)

Nearly always Often Seldom Never

(8) When in a group I have been content to be led. (MIN)

Never Seldom Often Nearly always

(9) When someone has been particularly helpful, I've wondered what real reason lay behind it. (DO)

Nearly always Often Seldom Never

(10) I have had confidence in myself. (LSC)

Never Seldom Often Nearly always

* See back page for key

(11) When I've disliked someone, I have shown it. (HA)

Nearly always Often Seldom Never

(12) I have wanted plenty of support from people. (DEP)

Never Seldom Often Nearly always

(13) I have felt the urge to smash things. (HT)

Very often Often Seldom Never

(14) I have been content to be dominated by someone else. (MIN)

Never Seldom Often Nearly always

(15) I have believed that people are pretty reliable. (DO)

Nearly always Often Seldom Never

(16) I have been very unsure of myself. (LSC)

Never Seldom Often Nearly always

(17) When I've been angry with someone, I've bottled it up. (HA)

Nearly always Often Seldom Never

(18) I have liked to be told what needs doing. (DEP)

Never Seldom Often Nearly always

(19) I have wanted to give someone a piece of my mind. (HT)

Very often Often Seldom Never

(20) I have preferred to let people have their own way. (MIN)

Never Seldom Often Nearly always

(21) I have felt that people would tell lies to get ahead. (DO)

Nearly always Often Seldom Never

(22) I have given up doing something because I thought too little of my own ability. (LSC)

Never Seldom Often Very Often

(23) Even when crossed, I've let people get away with it. (HA)

Nearly always Often Seldom Never

(24) I have been content to lean on other people for emotional support. (DEP)

Never Seldom Often Nearly always

(25) I would have liked to pick a quarrel with someone. (HT)

Very often Often Seldom Never

(26) I have been happy to play second fiddle. (MIN)

Never Seldom Often Nearly always

(27) I have felt that people are out for what they can get. (DO)

Nearly always Often Seldom Never

(28) I have felt that even when difficulties were piling up I would overcome them. (LSC)

Never Seldom Often Very often

(29) When I've thought I was justified in losing my temper, I have done so in no uncertain terms. (HA)

Very often Often Seldom Never

(30) I have preferred to find out for myself what's to be done. (DEP)

Never Seldom Often Nearly always

(31) I have felt like blaming others when things have gone wrong. (HT)

Nearly always Often Seldom Never

(32) I have preferred to stay in the background. (MIN)

Never Seldom Often Nearly always

(33) I have thought one can safely trust people. (DO)

Nearly always Often Seldom Never

(34) I have felt pretty useless. (LSC)

Never Seldom Often Nearly always

(35) When I've felt like blaming someone to their face for something that has gone wrong, I have done so. (HA)

Nearly always Often Seldom Never

(36) I have needed a lot of help from other people. (DEP)

Never Seldom Often Very often

Antipunitiveness:
Hostile thoughts (HT)
Negativistic attitude (DO)

Intropunitiveness:
Lacks self confidence (LSC)
Over-dependence (DEP)

Dominance:
Hostile acts (HA)
Domineering attitude (MIN)

Revised Scales on the Bedford-Foulds Personality Deviance Scales

Revised Hostility Scale
(numbered according to item on original questionnaire)

1. I would have liked to get my own back on someone.
5. When I have wanted to have a row with someone, I have done so.
7. I have felt like telling people to go to blazes.
11. When I've disliked someone, I have shown it.
13. I have felt the urge to smash things.
19. I have wanted to give someone a piece of my mind.
25. I would have liked to pick a quarrel with someone.
29. When I've though I was justified in losing my temper, I have done so in no uncertain terms.

Revised Submissiveness/Low Self Confidence Scale

4. I have felt as capable as other people.
8. When in a group, I have been quite content to be led.
10. I have had confidence in myself.
14. I have been content to be dominated by someone else.
16. I have been very unsure of myself.
22. I have given up doing something because I thought too little of my own ability.
26. I have been happy to play second fiddle.
32. I have preferred to stay in the background.
34. I have felt pretty useless.

CRITERIA FOR DIAGNOSIS OF CORONARY EVENTS & STROKES
Fatal and Non-Fatal

A. CORONARY EVENT - FATAL

1. Definite Fatal Coronary Event

(i) Post Mortem: acute M.I.
OR
(ii) Death Certificate Codes (I.C.D. 410-414)
+ possible history of M.I.
or possible criteria for M.I.
or definite criteria for M.I. more than 4 weeks before death
or post mortem evidence of severe coronary atherosclerosis/M.I.
OR
(iii) definite criteria for M.I. within 4 weeks of death

2. Possible Fatal Coronary Event

Death certificate codes I.C.D. 410-414 and no supplementary evidence

3. Sudden Death

No evidence of definite or possible fatal coronary event and death occuring within 1 hour of cardiac symptoms within 1 hour of being seen symptom free

B. CORONARY EVENT - NON-FATAL

1. Definite Myocardial Infarction - at least 2 of the following:

(i) Prolonged cardiac pain - anywhere in the anterior chest, left arm, or jaw (which may also involve back, shoulder, right arm, abdomen) and last at least 20 minutes

(ii) Diagnostic E.C.G. - Minnesota codes 1.1.1 - 1.2.5, 1.2.7, or 9.2 + 5.1 or 5.2

(iii) Abnormal enzymes - CPK > twice upper limits of normal
and LDH (USLD) " " " "
or SGOT (AST) " " " "
or CPK-MB " " " "
Enzymes measured within 72 hours of admission or acute event

2. Possible Myocardial Infarction

(i) One of the above criteria plus none AND more unusual record of the other parameters or at least one of the following equivocal criteria:

(a) equivocal E.C.G. - Minnesota codes 1.2.8 - 1.3.6
or 4.1 - 4.3
or 5.1 - 5.3
or 9.2

(b) equivocal enzymes - at least one of the following above upper limit of normal but not twice normal
CPK
LDH
SGOT
CPK MB
or one above twice normal and there is a non-ischaemic cause present e.g. defibrillation, surgery, liver disease

3. Primary Cardiac Arrest with Successful Resuscitation

No evidence of myocardial infarction as above and no obvious non-atherosclerotic cause of cardiac arrest

C. STROKE - FATAL

1. Definite Fatal Stroke

(i) Post mortem: cerebral infarction or haemorrhage
OR
(ii) Criteria of definite stroke within 6 weeks of death

2. Possible Fatal Stroke

Death certificate code of underlying or immediate cause (I.C.D. 431-437).

D. STROKE - NON FATAL

1. Definition non-fatal stroke

History of rapid onset (<48 hours) AND clinical confirmation of signs of focal (or global) disturbance of cerebral function lasting > 24 hours or confirmation by C.T. scan of cerebral infarction or haemorrhage

2. Possible non-fatal stroke

Discharge diagnosis with primary or secondary codes of I.C.D. 431, 432, 434, 436, 437.

N.B. No evidence of other non-atherosclerotic disease causing any of the above

DEFINITIONS

RANSIENT ISCHAEMIC ATTACK (T.I.A.)

"History of rapid onset of clinical signs of focal (or global) disturbance of cerebral function lasting less than 24 hours".

NGINA PECTORIS

"pain or discomfort in the centre of chest or (L) anterior chest and (L) arm when walking up hill or hurrying requiring the person to stop or slow down for 10 minutes or less whereupon the pain is relieved"

Grade 1 Angina - the pain or discomfort is not evident when walking at an ordinary pace on the level.

Grade 2 Angina - the pain or discomfort is evident when walking at an ordinary pace on the level.

INTERMITTENT CLAUDICATION

"Pain in the calf of either leg which does not begin when standing still or sitting, but occurs when walking uphill, hurrying, or at an ordinary pace on the level. The pain does not disappear while walking but is relieved in 10 minutes or less if the person slows down or stops".

Grade 1 Intermittent Claudication - no pain when walking at an ordinary pace on the level.

Grade 2 Intermittent Claudication - pain when walking at an ordinary pace on the level.

THROMBOSIS/EMBOLISM

Clinical diagnosis confirmed by laboratory, radiological or surgical evidence.

AMPUTATION

Amputation of any part of the lower limb due to diabetes or vascular causes only.

STUDY NO._____

CHECK LIST FOR CARDIOVASCULAR EVENT RECORDING FORM

Name_____ Date of Birth_____

Address_____

_____

Event No._____ E.A.S. Appt._____

Date of Event_____

G.P. Name_____

Address_____

HOSPITAL ADMISSION

Hospital_____ O.P._____

Ward/Dept_____ Diagnosis_____

Consultant_____

Record no._____ CCU No._____

Date(s) of Admission/Attendance_____/_____/_____

Date(s) of Discharge_____/_____/_____

DIAGNOSIS 1._____
2._____
3._____

PAST MEDICAL HISTORY
1._____
2._____
3._____

ON ADMISSION _____

_____

E.C.G. CODES _____

_____

CARDIAC ENZYMES DATE_____ DATE_____ DATE_____
AST (10-35 RIE)
USLD (100-300 RIE)
CK (30-200 MALES RIE)
(30-150 FEMALES RIE)

CT SCAN _____

COMMENTS_____

PTO for further remarks

Cardiovascular Event Recording Form (1) Alive

No ☐☐☐☐☐ F-5

ess. _____ GP Name _____

GP Address _____

/ /

iovascular Event Number ☐☐ Date / /

Number _____

rmation Source

lent/Relative ☐ 1
☐ 2
C ☐ 3
A ☐ 4
ner _____ ☐ 5
known ☐ 6

visional Diagnosis

ocardial Infarction ☐ 1
oke ☐ 2
gina ☐ 3
nsient Ischaemic Attack ☐ 4
ermittent Claudication ☐ 5
rombosis/Embolism ☐ 6
putation ☐ 7
ner CV _____ ☐ 8
ner NonCV _____ ☐ 9
known ☐ 10

nfirmation Source

atient/Relative ☐ 1
P Record ☐ 2
spital Record ☐ 3
ther _____ ☐ 4
known ☐ 5

4. Final Diagnosis

Myocardial Infarction Definite	☐ 1
Myocardial Infarction Possible	☐ 2
Primary Cardiac Arrest	☐ 3
Stroke Definite	☐ 4
Stroke Possible	☐ 5
Angina	☐ 6
Transient Ischaemic Attack	☐ 7
Intermittent Claudication	☐ 8
Rest Pain/Ulcer/Gangrene	☐ 9
Thrombosis/Embolism	☐ 10
Vascular Surgery (not amp)	☐ 11
Amputation	☐ 12
Other CV _____	☐ 13
Other NonCV _____	☐ 14
Unknown	☐ 15
Angioplasty	☐ 16
C.A.B.G.	☐ 17

5. Confirmatory Criteria for M.I.

Pain	☐ 1
ECG	☐ 2
Enzymes	☐ 3
Equivocal ECG	☐ 4
Equivocal Enzymes	☐ 5

6. Confirmatory Criteria for Stroke

Clinical Criteria	☐ 1
CT Scan Positive	☐ 2
Discharge Diagnosis	☐ 3

7. Final Diagnosis Confirmation

Study Criteria	☐ 1
Clinical Impression	☐ 2

Master copy

07563

tudy No ☐☐☐☐☐

ame _____ GP Name _____

ddress _____ GP Address _____

_____ _____

_____ _____

oB / /

ardiovascular Event Number ☐☐ Place of Death _____

HS Number _____ Date of Death / /

Information Source

Patient's Relative	☐ 1
GP	☐ 2
NHSCR	☐ 3
CSA	☐ 4
Vascular Surgery/PVC	☐ 5
Other _____	☐ 6
Unknown	☐ 7

Provisional Cause of Death

Myocardial Infarction	☐ 1
Stroke	☐ 2
Thrombosis/Embolism	☐ 3
Amputation	☐ 4
Other CV _____	☐ 5
Other Non CV _____	☐ 6
Unknown	☐ 7

Confirmation Source

Death Certificate	☐ 1
Patient's Relative	☐ 2
GP Rec	☐ 3
Hospital Record	☐ 4
Hospital Post Mortem	☐ 5
Procurator Fiscal PM	☐ 6
Other _____	☐ 7
Unknown	☐ 8

4. Final Cause of Death

Myocardial Infarction	
- Definite	☐ 1
- Possible	☐ 2
- Sudden Death	☐ 3
Stroke	
- Definite	☐ 4
- Possible	☐ 5
Other	
- CV	☐ 6
- Non CV	☐ 7

5. Confirmatory Criteria for Definite Fatal MI

Post Mortem	☐ 1
Death Certificate	☐ 2
Definite criteria of MI within 4 weeks	☐ 3

6. Confirmatory Criteria for Definite Fatal Stroke

Post Mortem	☐ 1
Clinical criteria within 6 weeks	☐ 2

EDINBURGH ARTERY STUDY

Please complete card and return in pre-paid envelope if New Diagnosis, Change of Address or Death:

PTO

NEW DIAGNOSIS	Definite	Possible
Myocardial Infarction	☐	☐
Angina	☐	☐
Stroke	☐	☐
Transient ischaemic Attack	☐	☐
Intermittent Claudication	☐	☐
Arterial Thrombosis/Embolism	☐	☐

CHANGE OF ADDRESS .....

.....

RECALL OF NOTES BY LOTHIAN HEALTH BOARD ☐

DEATH (Cause) ..... (Date) .....

Signed ..... Date .....

EDINBURGH ARTERY STUDY

EART FOUNDATION

Department of Community Medicine
Medical School
Teviot Place
Edinburgh EH8 9AG
Tel: 031-650 3246 (direct line)
Tel: 031-668 3848 (24hr Answering Service)

University of Edinburgh

Dear

Many thanks for returning your health questionnaire which we sent to you last year. We are now enclosing another questionnaire to find out if you have developed any of these conditions for the first time since we last contacted you.

This information is helping our research team find out more about why people get heart attacks, strokes and artery disease. It is most important that we find out each year about the health of everyone in the study.

We should therefore be most grateful if you would take just a few minutes of your time to answer the enclosed questionnaire, and return it in the prepaid envelope. We should like to hear from you even if you have been perfectly well during the past year, and please note that we are not asking you to attend for an examination.

With many thanks for your continuing co-operation.

With best wishes.

Yours sincerely,

Mrs. Janet Dunbar
Research Sister

Dr. F.G.R. Fowkes
Study Director

encl:

1990 QUESTIONNAIRE

PLEASE COMPLETE AND RETURN IN PREPAID ENVELOPE

SINCE COMPLETING THE PREVIOUS QUESTIONNAIRE HAVE YOU HAD ANY OF THE FOLLOWING WHICH YOU HAVE NOT EXPERIENCED BEFORE:	Please tick Yes	No	Not sure
1. Severe pain in your chest? (Excluding any pain which was treated as an infection)	☐	☐	☐
2. Heart Attack?	☐	☐	☐
3. Angina?	☐	☐	☐
4. Stroke?	☐	☐	☐
5. Sudden loss of power in either leg or arm?	☐	☐	☐
6. Pain in back of either leg below the knee when walking? (excluding pain due to varicose veins or arthritis)	☐	☐	☐
7. Blood clot or hardening of the arteries in either leg?	☐	☐	☐
8. Outpatient attendance or admission to hospital with any of the above?	☐	☐	☐
If yes, which hospital?			
9. Seen your G.P. about any of the above?	☐	☐	
10. Have you changed your G.P.?	☐	☐	

If yes —

New G.P. Name .....

Address .....

.....

11. Have you changed your address?

If yes —

New Address .....

.....

.....

MANY THANKS FOR ANSWERING THESE QUESTIONS

1991 QUESTIONNAIRE

PLEASE COMPLETE AND RETURN IN PREPAID ENVELOPE

SINCE COMPLETING THE 1990 QUESTIONNAIRE HAVE YOU EXPERIENCED ANY OF THE FOLLOWING FOR THE FIRST TIME:	Please tick Yes	No	Not sure
. Severe pain in your chest? (Excluding any pain which was treated as an infection)	☐	☐	☐
. Heart Attack?	☐	☐	☐
. Angina?	☐	☐	☐
. Stroke?	☐	☐	☐
. Sudden loss of power in either leg or arm?	☐	☐	☐
. Pain in back of either leg below the knee when walking? (excluding pain due to varicose veins or arthritis)	☐	☐	☐
. Blood clot or hardening of the arteries in either leg?	☐	☐	☐
. Do you take aspirin daily?	☐	☐	☐
. Have you attended hospital as an outpatient with any of the above?	☐	☐	☐
0. If yes, which hospital? ..... date of attendance?	☐	☐	☐
1. Have you been admitted to hospital with any of the above?	☐	☐	☐
2. If yes, which hospital? ..... date of admission?	☐	☐	☐
3. Have you changed your G.P.?	☐	☐	☐

– If yes – New G.P. Name .....

Address .....

.....

14. Have you changed your address?

– If yes –

New address .....

.....

.....

MANY THANKS FOR ANSWERING THESE QUESTIONS

1992 QUESTIONNAIRE

LEASE COMPLETE AND RETURN IN PREPAID ENVELOPE

Please tick

INCE COMPLETING THE 1991 QUESTIONNAIRE
IAVE YOU EXPERIENCED ANY OF THE FOLLOWING IN SECTION A
'OR THE FIRST TIME:

	Yes	No	Not sure
1. Severe pain in your chest? (Excluding any pain which was treated as an infection)	☐	☐	☐
2. Heart Attack?	☐	☐	☐
3. Angina?	☐	☐	☐
4. Stroke?	☐	☐	☐
5. Sudden loss of power in either leg or arm?	☐	☐	☐
6. Pain in back of either leg below the knee when walking? (excluding pain due to varicose veins or arthritis)	☐	☐	☐
7. Blood clot or hardening of the arteries in either leg?	☐	☐	☐
8. Do you take aspirin daily?	☐	☐	☐
9. Have you attended your G.P. with any of the above?	☐	☐	☐
10. Have you attended hospital as an outpatient with any of the above?	☐	☐	☐
11. If yes, which hospital? ..... date of attendance?	☐	☐	☐
12. Have you been admitted to hospital with any of the above?	☐	☐	☐
13. If yes, which hospital? ..... date of admission?	☐	☐	☐
14. Have you changed your G.P.?	☐	☐	☐

– If yes – New G.P. Name .....

Address .....

.....

15. Have you changed your address?
– If yes –

New address .....

.....

.....

Telephone No .....

MANY THANKS FOR ANSWERING THESE QUESTIONS

EDINBURGH ARTERY STUDY

ART FOUNDATION

Department of Public Health Sciences
Medical School
Teviot Place
Edinburgh EH8 9AG
Tel: 031-650 3246 (direct line)

THE UNIVERSITY OF EDINBURGH

Edinburgh Artery Study: Invitation

Many thanks for returning your health questionnaires which we have sent to you over the last few years. We have greatly appreciated the time you have spent in completing and returning these to us. The information from the medical examination which you had nearly five years ago and from your annual health questionnaires, has been helping our research team to find out more about why people get heart attacks, strokes and artery disease.

We would now like to invite you to have another medical examination to be carried out in the Vascular Studies Unit at the Royal Infirmary of Edinburgh. From this examination we can find out how your health has progressed.

This examination will be shorter and less onerous than your first examination. When you arrive at the Unit we shall ask you to complete a brief questionnaire. You will have your height and weight recorded and your blood pressure checked. You will also have an electrocardiogram of your heart and an ultrasound scan of your abdomen and neck. These tests are quite straightforward and do not cause any discomfort. We should also like to take one small blood sample.

We should be very grateful if you would complete the enclosed form and return it to us in the prepaid envelope.

Thank you again for your participation in the Edinburgh Artery Study and for your continuing support. We look forward to hearing from you.

Yours sincerely,

Janet T Dunbar
Research Sister

Dr F G R Fowkes
Reader

EDINBURGH ARTERY STUDY

Please complete and return in Prepaid Envelope

Name: .....

Address: .....

.....

..... Postcode .....

Telephone No. .....

Please tick

	Yes	No
1. I would like to participate in the medical examination of Edinburgh Artery Study.	☐	☐

2. **TRANSPORT**

Please tick

a) I will travel to and from Royal Infirmary by private car, or bus (expenses can be reimbursed from Clinic Staff) ☐

b) I am physically unable to attend the Royal Infirmary and want to be examined at home. ☐

EDINBURGH ARTERY STUDY

ART FOUNDATION

Department of Public Health Sciences
Medical School
Teviot Place
Edinburgh EH8 9AG
Tel: 031-650 3246 (direct line)

Edinburgh Artery Study: Invitation

Many thanks for returning your health questionnaires which we have sent to you over the last few years. We have greatly appreciated the time you have spent in completing and returning these to us.

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This examination will be shorter and less onerous than your first examination. When you arrive at the Unit we shall ask you to complete a brief questionnaire. You will also have your height and weight recorded and your blood pressure checked. You will also have an electrocardiogram of your heart and an ultrasound scan of your abdomen and neck. These tests are quite straightforward and do not cause any discomfort. We should also like to take one small blood sample.

We appreciate that you have moved away from Edinburgh but we would still like to extend this invitation to you. We can make arrangements for you to be reimbursed for your travelling expenses equivalent to the 2nd class rail return fare. If you require to spend the night in Edinburgh because of the distance travelled, we can, if you wish, make arrangements for you to stay free of charge in a family run Guest House. If you are planning to visit Edinburgh anyway during the next few months, we can make an appointment at the time of your intended visit.

EDINBURGH ARTERY STUDY

Please complete and return in Prepaid Envelope

Name: .....

Address: .....

.....

..... Postcode .....

Telephone No. .....

	Please tick Yes	No
I would like to participate in the medical examination of Edinburgh Artery Study.	☐	☐

TRANSPORT AND ACCOMMODATION

I intend to stay overnight in Edinburgh
If staying overnight, your appointment will be scheduled for the following morning.

	Please tick Yes	No
a) I would like the Edinburgh Artery Study to book overnight accommodation for me the night before the examination.	☐	☐
b) I will be in Edinburgh within the next 3 months and would like an appointment during this time.	☐	☐

If answering 'Yes' to **(b)** :

Date of arrival in Edinburgh .....

Date of departure from Edinburgh .....

EDINBURGH ARTERY STUDY

H HEART FOUNDATION

Department of Public Health Sciences
Medical School
Teviot Place
Edinburgh EH8 9AG
Tel: 031-650 3246 (direct line)

THE UNIVERSITY OF EDINBURGH

Dear

Appointment Letter - Home Visit

Many thanks for your reply indicating you are willing to have an examination but that you are unable to come to the Vascular Studies Unit in the Royal Infirmary of Edinburgh.

We intend to visit you at your home on:

Date: _____ **at Time:** _____

We hope this date and time is convenient. If this is not a convenient time for you, please telephone:

Mrs J. T. Dunbar on (031) 650-3246 to arrange a more suitable time.

A small questionnaire is enclosed which we would like you to complete. The nurse will check it and answer any queries when she visits you.

We look forward to seeing you.

Yours sincerely,

Janet T. Dunbar
Research Sister

Encl.

EDINBURGH ARTERY STUDY

CONSENT FORM

Purpose of Research -

The purpose of this research is to measure the state of your arteries using blood pressure techniques and scanning techniques, and to find out how these are affected by the make up of your blood, the condition of your heart and your smoking habits. By finding out what harms arteries we can take steps to prevent disease in the future.

Your examination - Time approximately 1 hour 15 minutes.

An explanation by a member of clinic staff will begin the proceedings. You will have an E.C.G. (to check your heart); your abdomen and neck will be scanned; measurements of height, weight and blood pressure will be taken; a small blood sample will be taken at the end, and your questionnaire will be checked.

SCANNING

Abdomen

Some people develop a swelling of their main blood vessel as they grow older. Such a swelling is called an aortic aneurysm. The main blood vessel (the aorta) is normally less than 1 inch (or about 2 cm) wide as it brings blood down from the heart to the abdomen and legs. Swellings of the aorta in the abdomen can easily be detected and measured accurately by ultrasound examination. Very occasionally, large swellings are detected and treatment may be advisable. The ultrasound examination is painless and without risk to your health.

Neck

Just as the development of a swelling of the aorta can occur due to age, the blood vessels in your neck can become narrower as you grow older. The blood vessels in your neck can be identified and an accurate measurement of the blood vessel taken by ultrasound examination. Very occasionally, severe narrowing is detected and treatment may be advisable. The ultrasound examination is painless and without risk to your health.

If during your ultrasound we find a swelling of the aorta, or a narrowing of the blood vessels in your neck, we shall inform your G.P. and would recommend that you discuss this with him or her.

CONSENT

I have read the above, which has also been explained to me by a member of staff, and understand what is involved in my participation in this research. I know that I can readily withdraw from the medical examination, or any part of it, at any point if I so wish. I also understand that the study has been given ethical approval by a Medical Ethics Sub-Committee of the Lothian Health Board. I realise that no liability is accepted by the research team in the proper execution of their work.

Signature: .....

Name in Capitals: .....**Date:** .....

EDINBURGH ARTERY STUDY

VENEPUNCTURE

ECT NAME: .....

RDER: 1: M.C. 2: A.C. 3: J.D.

VENEPUNCTURE

	Yes	No
1. Has patient had jaundice in the last year?	☐	☐
2. Has patient had serum jaundice?	☐	☐
3. Is patient a diabetic?	☐	☐
4. Was venepuncture normal?	☐	☐
5. Was venepuncture difficult/slow?	☐	☐
6. Was venepuncture not possible?	☐	☐

RE STUDY PARTICIPATION

	Yes	No	Not Sure
rticipant willing to return for blood test?	☐	☐	☐

and date for return to be arranged later.

ipant's Telephone No. _____

GHT (WITHOUT SHOES) ☐☐☐ . ☐ CM

GHT (WITHOUT COAT & SHOES) ☐☐☐ . ☐ KG

J-3

EDINBURGH ARTERY STUDY

BLOOD PRESSURE PERIPHERAL PULSE PALPATION RECORDING FORM

UBJECT NAME: .....

ECORDER: 1: M.C. 2: (A.C.) 3: J.D.

ALPATION OF PULSES:

	Present	Absent
right dorsalis pedis	✓	
left dorsalis pedis	✓	
right posterior tibial	✓	
left posterior tibial	✓	
right femoral	✓	
left femoral	✓	

ESTING BLOOD PRESSURE (Couch flat, 1 pillow)

	Observed	Zero
RIGHT or ~~LEFT~~ arm systolic mm Hg	166	
diastolic	123	49

OPPLER MEASUREMENTS

	Observed	Zero
RESTING SYSTOLIC PRESSURE - BRACHIAL	174	50

ESTING SYSTOLIC ANKLE PRESSURES

	Observed	Zero
right ankle mm Hg	200	49
left ankle	148	60

? Very hard to detect.

OMMENTS: Any deviation from standard procedures:

	Yes	No
E C G RECORDED	✓	

EDINBURGH ARTERY STUDY

CAROTID SCANNING RECORD

: .....

rder : 1 MC 2 AC 3 JD

	RIGHT	LEFT
ification of Common Carotid	☐	☐
ification of Internal Carotid	☐	☐
ification of External Carotid	☐	☐
ification of Bifurcation	☐	☐

MAL MEDIAL THICKNESS
SUREMENTS (IMT)

2cms below bifurcation ☐.☐☐ cm ☐.☐☐ cm

OF PLAQUE

	RIGHT	LEFT
mon Carotid	☐	☐
nal Carotid	☐	☐
rnal Carotid	☐	☐
rcation	☐	☐

test thickness of plaque ☐.☐☐ cm ☐.☐☐ cm

iameter stenosis ☐☐ % ☐☐ %

Systolic Velocity (PSV) at stenosis ☐☐☐.☐ cm/sec. ☐☐☐.☐ cm/sec.

☐☐☐.☐ cm/sec. ☐☐☐.☐ cm/sec.

ments : Any deviation from standard practice_____

.....

OFFICIAL USE Normal ☐ Abnormal ☐

informed Yes ☐ No ☐ Date __ __ /__ __ /__ __

eference only

of disease

al medial thickness(IMT)
cm **normal**
- 0.8 cm
cm **probable disease**

% of Carotid Stenosis

> 50% stenosis
Symptomatic - European Carotid Surgery Trial
30 - 70% randomised surgery or observation
Asymptomatic
>70% stenosis randomised to surgery or no surgery

EDINBURGH ARTERY STUDY

I-5

AORTIC SCANNING RECORD

: .....

der : 1 (MC) 2 AC 3 JD

- Mode External AP of Aorta (max. systole) [2] . [5][6] cms

2.56
2.04
2.00
2.11

diameter > 2.8 cm [ ] . [ ][ ] cms
[ ] . [ ][ ] cms

nts : Any deviation from standard practice

_____

_____

.....

OFFICIAL USE Normal [ ] Abnormal [ ]

formed Yes [ ] No [ ] Date __ /__ /__

ortic Aneurysm Criteria - reference only

cms observe
cms TRIAL
ms surgery if fit

EDINBURGH ARTERY STUDY

QUESTIONNAIRE

THE INFORMATION IN THIS QUESTIONNAIRE IS HIGHLY CONFIDENTIAL AND IS PART OF A MEDICAL RESEARCH STUDY.

The information you give in this personal health record will be treated as strictly confidential and will be available only to your own doctor and the study team. The results of the research will appear only in the form of general statistics from which it will be impossible to identify you as an individual.

Please complete the following:

SURNAME: .....

FORENAMES: .....

DATE: .....

If you have any difficulties in answering some of the questions you will have a chance to discuss these later with a member of the study team.

Please bring this questionnaire with you when you attend your medical examination.

THANK YOU FOR YOUR CO-OPERATION IN THIS STUDY. THE FINDINGS WILL HELP TO IMPROVE HEALTH IN SCOTLAND.

IT IS IMPORTANT TO ANSWER ALL THE QUESTIONS CAREFULLY.

PLEASE TAKE YOUR TIME.

PERSONAL HISTORY

1. Please tick one box: Male ☐ 1 Female ☐ 2

2. Enter your date of birth: Day ☐☐ Month ☐☐ Year ☐☐

3. Please tick the box showing your present marital status:

Married (or equivalent)	☐ 1
Single	☐ 2
Widowed	☐ 3
Divorced or separated	☐ 4

4. What is your employment status at the moment? Please tick boxes as appropriate:

Paid Employment, full-time	☐ 1
Paid Employment, part-time	☐ 2
Unemployed	☐ 3
Retired	☐ 4
Housewife (full-time)	☐ 5
Other, please specify	☐ 6

5. Have you changed your address in the last year? Yes ☐ No ☐

If yes - New Address . .....

.....

6. Have you changed your G.P.? Yes ☐ No ☐

If yes - New G.P. Name .....

Address .....

7. Have you experienced any of the following for the first time in the last year

Please tick

	Yes	No	Not Sure
(i) Severe pain in your chest? (excluding any pain which was treated as an infection)	☐	☐	☐
(ii) Heart attack?	☐	☐	☐
(iii) Angina?	☐	☐	☐
(iv) Stroke?	☐	☐	☐
(v) Sudden loss of power in either leg or arm?	☐	☐	☐
(vi) Pain in back of either leg below the knee when walking? (excluding pain due to varicose veins or arthritis)	☐	☐	☐
(vii) Blood clot or hardening of the arteries in either leg?	☐	☐	☐

	Yes	No	Not Sure
8. Have you attended your G.P. with any of the above?	☐	☐	☐
9. Have you attended hospital as an outpatient with any of the above?	☐	☐	☐

If yes, which hospital? .....

Date of attendance? .....

10. Have you been admitted to hospital with any of the above?	☐	☐	☐

If yes, which hospital? .....

Date of admission? .....

11. Do you take aspirin daily?	☐	☐	☐

12. Are you on any regular medical treatment from a doctor as follows:

	Yes	No
Drugs to lower blood pressure	☐	☐
Diuretics (water tablets)	☐	☐
Insulin injections	☐	☐
Tablets for diabetes	☐	☐
Other treatments?	☐	☐

Give names if possible:

.....

.....

.....

13. **For Female Participants Only**

What age were you when you went through the menopause? .....

14. SMOKING

Smoking has been linked with many health problems. It is important that you answer the following section as accurately as possible.

Please Tick

	Yes	No
(a) Do you smoke at present	☐	☐

If no, proceed to Question 14(f)

(b) What do you usually smoke now?	Yes	No
Cigarettes	☐	☐
Pipe	☐	☐
Cigars	☐	☐

(c) How many do you usually smoke now:

Cigarettes per day ..... Cigarettes

Oz. tobacco per week ..... Oz.

Cigars per week ..... Cigars

(d) For how many years during your life have you smoked cigarettes? ..... Years

(e) How many cigarettes have you smoked on average per day during the period you have smoked: ..... Cigarettes

Now proceed to question 14(k)

	Yes	No
(f) Have you ever smoked regularly?	☐	☐

If no, proceed to question 14(k)

(g) What did you usually smoke?

	Yes	No
Cigarettes	☐	☐
Pipe	☐	☐
Cigars	☐	☐

(h) How much did you smoke on average while you were a smoker?

Cigarettes per day	..... Cigarettes
Oz. tobacco per week	..... Oz.
Cigars	..... Cigars

(i) For how many years did you smoke cigarettes? ..... Years

(j) If you smoked cigarettes, how long is it since you finally gave up? ..... Years ..... Months

(k) Is any other member of your household a smoker? Yes ☐ No ☐

CHEST PAIN

		Yes	No
15. (a)	Do you ever get pain or discomfort in your chest?	☐	☐

IF NO, PROCEED TO QUESTION 16

(b)	Do you get this pain or discomfort when you walk uphill or hurry?	☐	☐

IF NO, PROCEED TO QUESTION 16

(c)	Do you get it when you walk at an ordinary pace on the level?	☐	☐

(d) When you get any pain or discomfort in your chest what do you do? Tick One

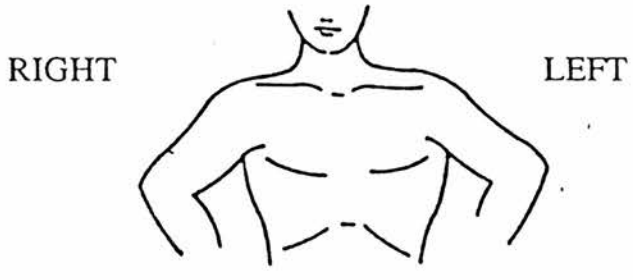
- Stop ☐
- Slow down ☐
- Continue at the same pace ☐

		Yes	No
(e)	Does it go away when you stand still or sit down?	☐	☐

(f) How soon? Tick One

- 10 minutes or less ☐
- more than 10 minutes ☐

(g) Where do you get this pain or discomfort? Mark the place(s) with 'X' on the diagram.



		Yes	No
16. (a)	Have you ever had a severe pain across the front of your chest lasting for half an hour?	☐	☐

(b) What was the cause? .....

FOR OFFICE USE ONLY A: GRADE MI:

17. LEG PAIN

	Yes	No
1. (a) Do you get a pain in either leg on walking?	☐	☐

IF NO, GO TO QUESTION 18

(b) Does this pain ever begin when you are standing still or sitting?	☐	☐
(c) Do you get this pain in your calf (or calves)?	☐	☐
(d) Do you get it when you walk uphill or hurry?	☐	☐
(e) Do you get it when you walk at an ordinary pace on the level?	☐	☐
(f) Does the pain ever disappear while you are still walking?	☐	☐

(g) What do you do if you get it when you are walking? Tick One

Stop	1	☐
Slow down	2	☐
Continue at same pace	3	☐

(h) What happens to it if you stand still? Tick One

Usually continues for more than 10 minutes	1	☐
Usually disappears in 10 minutes or less	2	☐

	Yes	No
18. Have you ever had surgery on the arteries of your legs other than for varicose veins?	☐	☐

Please specify .....

19. Have you ever had surgery to remove		
toes?	☐	☐
leg below the knee?	☐	☐
leg above the knee?	☐	☐

FOR OFFICE USE ONLY

I.C. GRADE

LOTHIAN health

OUR REF: 1702/93/4/51 YOUR REF:

Mr Les Malone
Ext 9028

7 June 1995

Prof F G R Fowkes
Wolfson Unit for the Prevention of
Peripheral Vascular Disease
The University of Edinburgh
Teviot Place
Edinburgh EH8 9AG

Dear Prof Fowkes,

Research Protocol : Peripheral Vascular Epidemiology Research Group : further work on Edinburgh Artery Study.

Thank you for your letter dated 24 May 1995 asking for permission to administer slightly longer annual questionnaires to the Edinburgh Artery Study participants. The Chairman of the Medicine and Clinical Oncology Research Ethics Sub-Committee has agreed to approve these questionnaires.

Yours sincerely

Les Malone.

Mr Les Malone
Secretary
Medicine and Clinical Oncology
Research Ethics Sub-Committee

LOTHIAN HEALTH BOARD

DEACONESS HOUSE
148 PLEASANCE EDINBURGH EH8 9RS
TELEPHONE: 0131 536 9000 DIRECT DIAL: 0131 536 FAX NUMBER: 0131 536

27th February, 1995

Dr M Morrison
Crewe Medical Centre
135 Boswall Parkway
Edinburgh
EH5 2NT

Dear Dr Morrison,

Re: **EDINBURGH ARTERY STUDY**

Thank you very much for your co-operation with the above named study over the last four years. Within the next few weeks we will be sending out our annual health questionnaire to the Edinburgh Artery Study participants.

This year, because of some previous findings that certain aspects of personality and the severity of peripheral arterial disease are associated in this population, we should like to ask the participants to fill in two personality inventories. These questionnaires have been widely used in previous studies and we do not believe that any of the questions are too sensitive or contentious. They comprise statements such as "I like to have a lot of people around me," and the respondent is asked to indicate one of five options from "strongly disagree" to "strongly agree." The participants will also be receiving an update sheet about the study, which is enclosed for your information.

You and your colleagues are also cordially invited to attend an inaugural lecture "Arterial Mysteries in Edinburgh Legs" by Gerry Fowkes, in which he will be discussing some findings from the Edinburgh Artery Study. Yours support and co-operation is very much appreciated and will be acknowledged in the lecture. It will take place on Tuesday, March 7th, 1995 in the Anatomy Lecture Theatre at the Medical School, Teviot Place, at 5.15 p.m.

With best wishes.

Yours sincerely,

Martha Whiteman
Research Coordinator, Edinburgh Artery Study

Ref:1~;9~

2~4~
5~
6~
7~
8~

Dear 2~4~

Many thanks for your co-operation with the Edinburgh Artery Study over the last several years. We are now enclosing another questionnaire to find out if you have developed any new occurrences of these conditions since we last had contact with you.

It is very important that we find out each year about the health of everyone in the study, as it is your information that is helping our research team find out more about why people get strokes, heart attacks and artery disease. We would like to hear from you even if you have been perfectly well during the last year.

This year we have also included a personality questionnaire, similar to the one you completed at your first examination in 1987-88. This should help us to continue to find out more about how personality might be related to these conditions. We hope you will find it both interesting and fun to do. We would therefore be very grateful if you would take 20 minutes or so to fill it in, **if you wish to do so.** It can then be returned with your health questionnaire in the prepaid envelope.

With many thanks and best wishes.

Yours sincerely,

Mrs Martha Whiteman
Research Associate

Professor F.G.R. Fowkes
Study Director

P.S. We also enclose an update sheet about some of the research team's important findings from the information you have given us so far.

PERSONALITY QUESTIONNAIRE

Please complete Parts 1 and 2 of the Personality Questionnaire and return in the same prepaid envelope as the Edinburgh Artery Study 1995 Questionnaire. Your responses will be kept in the strictest confidence, and entered into our computer without any personal identification.

PART 1: THE NEO FIVE FACTOR INVENTORY

INSTRUCTIONS

Carefully read all of the instructions before beginning.This questionnaire contains 60 statements. Please read each statement carefully. For each statement please tick the box that best represents your opinion.

Tick **strongly disagree** if you strongly disagree or the statement is definitely false.

Tick **disagree** if you disagree or the statement is mostly false.

Tick **neutral** if you are neutral on the statement, you cannot decide or the statement is about equally true and false.

Tick **agree** if you agree or the statement is mostly true.

Tick **strongly agree** if you strongly agree or the statement is definitely true.

For example, if you strongly disagree or believe that a statement is definitely false, you would tick the strongly disagree box for that statement.

STRONGLY DISAGREE	DISAGREE	NEUTRAL	AGREE	STRONGLY AGREE
☑	☐	☐	☐	☐

Fill in only one response for each statement. Please respond to all of the statements, making sure that you fill in the correct response. If you need to change an answer, cross out the incorrect response and tick the correct box.

	STRONGLY DISAGREE	DISAGREE	NEUTRAL	AGREE	STRONGLY AGREE
1. I am not a worrier.	☐	☐	☐	☐	☐
2. I like to have a lot of people around me.	☐	☐	☐	☐	☐
3. I don't like to waste my time daydreaming.	☐	☐	☐	☐	☐
4. I try to be courteous to everyone I meet.	☐	☐	☐	☐	☐
5. I keep my belongings clean and neat.	☐	☐	☐	☐	☐
6. I often feel inferior to others.	☐	☐	☐	☐	☐
7. I laugh easily.	☐	☐	☐	☐	☐
8. Once I find the right way to do something I stick to it.	☐	☐	☐	☐	☐
9. I often get in to arguments with my family and co-workers.	☐	☐	☐	☐	☐
10. I'm pretty good at pacing myself so as to get things done on time.	☐	☐	☐	☐	☐
11. When I'm under a great deal of stress, sometimes I feel like I'm going to pieces.	☐	☐	☐	☐	☐
12. I don't consider myself especially "light-hearted".	☐	☐	☐	☐	☐
13. I am intrigued by the patterns I find in art and nature.	☐	☐	☐	☐	☐
14. Some people think I'm selfish and egotistical.	☐	☐	☐	☐	☐
15. I am not a very methodical person.	☐	☐	☐	☐	☐

	STRONGLY DISAGREE	DISAGREE	NEUTRAL	AGREE	STRONGLY AGREE
16. I rarely feel lonely or blue.	☐	☐	☐	☐	☐
17. I really enjoy talking to people.	☐	☐	☐	☐	☐
18. I believe letting students hear controversial speakers can only confuse and mislead them.	☐	☐	☐	☐	☐
19. I would rather co-operate with others than compete with them.	☐	☐	☐	☐	☐
20. I try to perform all the tasks assigned to me conscientiously.	☐	☐	☐	☐	☐
21. I often feel tense and jittery.	☐	☐	☐	☐	☐
22. I like to be where the action is.	☐	☐	☐	☐	☐
23. Poetry has little or no effect on me.	☐	☐	☐	☐	☐
24. I tend to be cynical of others' intentions.	☐	☐	☐	☐	☐
25. I have a clear set of goals and work towards them in an orderly fashion.	☐	☐	☐	☐	☐
26. Sometimes I feel completely worthless.	☐	☐	☐	☐	☐
27. I usually prefer to do things alone.	☐	☐	☐	☐	☐

		STRONGLY DISAGREE	DISAGREE	NEUTRAL	AGREE	STRONGLY AGREE
28.	I often try new and foreign foods.	☐	☐	☐	☐	☐
29.	I believe that most people will take advantage of you if you let them.	☐	☐	☐	☐	☐
30.	I waste a lot of time before settling down to work.	☐	☐	☐	☐	☐
31.	I rarely feel fearful and anxious.	☐	☐	☐	☐	☐
32.	I often feel as if I an bursting with energy..	☐	☐	☐	☐	☐
33.	I seldom notice the moods or feelings that different environments produce.	☐	☐	☐	☐	☐
34.	Most people I know like me.	☐	☐	☐	☐	☐
35.	I work hard to accomplish my goals.	☐	☐	☐	☐	☐
36.	I often get angry at the way people treat me.	☐	☐	☐	☐	☐
37.	I am a cheerful, high-spirited person.	☐	☐	☐	☐	☐
38.	I believe we should look to our religious authorities for decisions on moral issues.	☐	☐	☐	☐	☐
39.	Some people think of me as cold and calculating.	☐	☐	☐	☐	☐
40.	When I make a commitment, I can always be counted on to follow through.	☐	☐	☐	☐	☐
41.	Too often, when things go wrong, I get discouraged and feel like giving up.	☐	☐	☐	☐	☐

		STRONGLY DISAGREE	DISAGREE	NEUTRAL	AGREE	STRONGLY AGREE
42.	I am not a cheerful optimist.	☐	☐	☐	☐	☐
43.	Sometimes when I am reading poetry or looking at a work of art, I feel a chill or wave of excitement.	☐	☐	☐	☐	☐
44.	I'm hard-headed and tough-minded in my attitudes.	☐	☐	☐	☐	☐
45.	Sometimes I'm not as dependable or reliable as I should be.	☐	☐	☐	☐	☐
46.	I am seldom sad or depressed.	☐	☐	☐	☐	☐
47.	My life is fast-paced.	☐	☐	☐	☐	☐
48.	I have little interest in speculating on the nature of the universe or the human condition.	☐	☐	☐	☐	☐
49.	I generally try to be thoughtful and considerate.	☐	☐	☐	☐	☐
50.	I am productive person who always gets the job done.	☐	☐	☐	☐	☐
51.	I often feel hopeless and want someone else to solve my problems.	☐	☐	☐	☐	☐
52.	I am a very active person.	☐	☐	☐	☐	☐
53.	I have a lot of intellectual curiosity.	☐	☐	☐	☐	☐

		STRONGLY DISAGREE	DISAGREE	NEUTRAL	AGREE	STRONGLY AGREE
54.	If I don't like people, I let them know it.	☐	☐	☐	☐	☐
55.	I never seem to be able to get organised.					
56.	At times I have been so ashamed I just wanted to hide.	☐	☐	☐	☐	☐
57.	I would rather go my own way than be a leader of others.	☐	☐	☐	☐	☐
58.	I often enjoy playing with theories or abstract ideas.	☐	☐	☐	☐	☐
59.	If necessary, I am willing to manipulate people to get what I want.	☐	☐	☐	☐	☐
60.	I strive for excellence in everything I do.	☐	☐	☐	☐	☐

Please check that you have responded to all the statements.

PART 2: THE SELF-RATING QUESTIONNAIRE (STAXI)

This part of the questionnaire concerns your feelings and behaviour. It is divided into two parts, HOW I GENERALLY FEEL and WHEN ANGRY OR FURIOUS. Read each statement and then give the answer that describes you best. If you need to change your answer, cross out the incorrect response and tick the correct box. The first section contains ten statements and begins on the next page. The second section contains 24 statements.

Examples:

ALMOST NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
☒	☐	☑	☐

HOW I GENERALLY FEEL

A number of statements that people use to describe themselves are given below. Read each statement and then tick the box that indicates how you generally feel. Remember that there are no right or wrong answers. Do not spend too much time on any one statement, but give the answer which seems to best describe how you generally feel.

		ALMOST NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
1.	I am quick tempered.	☐	☐	☐	☐
2.	I have a fiery temper.	☐	☐	☐	☐
3.	I am a hotheaded person.	☐	☐	☐	☐
4.	I get angry when I'm slowed down by others' mistakes.	☐	☐	☐	☐
5.	I feel annoyed when I am not given recognition for doing good work.	☐	☐	☐	☐
6.	I fly off the handle.	☐	☐	☐	☐
7.	When I get mad, I say nasty things.	☐	☐	☐	☐
8.	It makes me furious if I am criticised in front of others.	☐	☐	☐	☐
9.	When I get frustrated, I feel like hitting someone.	☐	☐	☐	☐
10.	I feel infuriated when I do a good job and get a poor evaluation.	☐	☐	☐	☐

WHEN ANGRY OR FURIOUS

Everyone feels angry or furious from time to time, but people differ in the ways that they react when they are angry. A number of statements are listed below which people use to describe their reactions when they feel angry or furious. Read each statement and then tick the box which indicates how often you generally react or behave in the manner described when you are feeling angry or furious. Remember that there are no right or wrong answers. Do not spend too much time on any one statement.

WHEN ANGRY OR FURIOUS . . .

		ALMOST NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
1.	I control my temper.	☐	☐	☐	☐
2.	I express my anger.	☐	☐	☐	☐
3.	I keep things in.	☐	☐	☐	☐
4.	I am patient with others.	☐	☐	☐	☐
5.	I pout or sulk.	☐	☐	☐	☐
6.	I withdraw from people.	☐	☐	☐	☐

		ALMOST NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
7.	I make sarcastic remarks to others.	☐	☐	☐	☐
8.	I keep my cool.	☐	☐	☐	☐
9.	I do things like slam doors.	☐	☐	☐	☐
10.	I boil inside, but I don't show it.	☐	☐	☐	☐
11.	I control my behaviour.	☐	☐	☐	☐
12.	I argue with others.	☐	☐	☐	☐
13.	I tend to harbour grudges that I don't tell anyone about.	☐	☐	☐	☐
14.	I strike out at whatever infuriates me.	☐	☐	☐	☐
15.	I can stop myself from losing my temper.	☐	☐	☐	☐
16.	I am secretly quite critical of others.	☐	☐	☐	☐
17.	I am angrier than I am willing to admit.	☐	☐	☐	☐
18.	I calm down faster than most other people.	☐	☐	☐	☐
19.	I say nasty things.	☐	☐	☐	☐
20.	I try to be tolerant and understanding.	☐	☐	☐	☐
21.	I'm irritated a great deal more than people are aware of.	☐	☐	☐	☐
22.	I lose my temper.	☐	☐	☐	☐
23.	If someone annoys me, I'm apt to tell him or her how I feel.	☐	☐	☐	☐
24.	I control my angry feelings.	☐	☐	☐	☐

THANK YOU VERY MUCH FOR COMPLETING THIS QUESTIONNAIRE

EDINBURGH ARTERY STUDY

1995 QUESTIONNAIRE

PLEASE COMPLETE AND RETURN IN THE PREPAID ENVELOPE

Since we last had contact with you, have you had any new occurrences of the following?

	Please tick YES	NO	NOT SURE
1. Severe pain in your chest?	☐	☐	☐
2. Heart attack?	☐	☐	☐
3. Angina?	☐	☐	☐
4. Stroke?	☐	☐	☐
5. Sudden loss of power in either leg or arm?	☐	☐	☐
6. Pain in either leg below the knee when walking? (Excluding any pain due to varicose veins or arthritis)	☐	☐	☐
7. Blood clot or hardening of the arteries in either leg?	☐	☐	☐

	YES	NO
8. Have you attended your G.P. with any of the above conditions? If yes, which condition?	☐	☐
9. Have you attended hospital as an outpatient with any of the above conditions?	☐	☐

If yes, which condition? .....

Which hospital? .....

Date of attendance .....

	YES	NO
10. Have you been admitted to hospital with any of the above conditions?	☐	☐

If yes, which condition? .....

Which hospital? .....

Date of attendance .....

	YES	NO
11. Do you take aspirin daily?	☐	☐

THE EDINBURGH ARTERY STUDY

1995 UPDATE SHEET

The research team has been able to find out several things from the information you have given us over the years. Since the study began in 1987, we have discovered that:

* Cigarette smoking is likely to cause even more disease in the leg blood vessels than it does in the heart.

* People who have artery disease in the legs are also more likely to get heart disease.

*Simple measurement of disease in the legs, such as the measurements performed at your examinations, can help identify apparently healthy people who are at increased risk of heart disease.

*Smoking may contribute to disease by damaging artery walls. It also may affect clotting of blood and increase certain fats in the blood.

*People with certain genetic structures that affect the clotting of the blood are more at risk of artery disease.

*Certain personality types are more likely to have artery disease, and your personality may possibly affect the fats in your blood.

* More men than women are likely to have artery disease, and this may be related to different clotting and stickiness of the blood in men and women.

*Moderate alcohol consumption and moderate exercise may offer slight protection against artery disease in the legs and in the heart, although as you probably know, there is some debate about what is considered to be moderate alcohol consumption!

*All the findings are helping us to build up a picture of who gets the disease and why. This should lead to advances in treatment and prevention.

We are now continuing this work over another five years, so you will be receiving yearly questionnaires until at least the year 2000. We are very grateful for all your help and support for the study so far, and we hope to continue to find out about the different factors that contribute to heart attacks, strokes and artery disease. Without your assistance, all of this research would have been impossible.

With our sincere thanks and very best wishes,

The Edinburgh Artery Study Research Team.

Ref:1~;9~

2~4~
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Dear 2~4~

Many thanks for returning last year's Edinburgh Artery Study Questionnaire. We are now enclosing another questionnaire to find out if you have developed any of these conditions for the **first time** since we last contacted you. This year we have enclosed a one-page health questionnaire only, much shorter than the forms we sent you last year.

This information is helping our research team find out more about why people get heart attacks, strokes and artery disease. It is very important that we find out each year about the health of everyone in the study.

We should therefore be most grateful if you would take a few minutes of your time to complete the enclosed questionnaire, and return it in the prepaid envelope. We should like to hear from you even if you have been perfectly well during the past year, and please note that we are not asking you to attend for an examination.

Many thanks for your continuing co-operation.

With best wishes.

Yours sincerely,

Mrs Martha Whiteman
Research Associate

Professor F.G.R. Fowkes
Study Director

encl:

cement sent
1995

Psychological Assessment Resources, Inc.
Mailing Address: P.O. Box 998/Odessa, Florida 33556
Street Address: 16204 N. Florida Ave./Lutz, Florida 33549
Telephone (813) 968-3003
Telefax (813) 968-2598

cheque to follow

February 20, 1995

CHECK # 226430077
P.O. # _____
DUE DATE _____
AMOUNT $1495.00
DATE PAID 3/22/95
INITIALS BV

Martha C. Whiteman
Research Associate
University of Edinburgh
Public Health Sciences
Medical School
Teviot Place
Edinburgh, Scotland LH8 9AG
UNITED KINGDOM

Dear Ms. Whiteman:

In response to your recent request, permission is hereby granted to you to modify the NEO-Five Factory Inventory and the State-Trait Anger Expression Inventory (State Anger section only) and incorporate them in your own questionnaire format and reproduce up to 1,300 copies of each instrument for use in your medical research project at Edinburgh University.

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Sincerely,

R. BOB SMITH, III, Ph.D.
President

RBS/bv

ACCEPTED AND AGREED:

BY: Martha C. Whiteman
MARTHA C. WHITEMAN

DATE: 13 March 1995

ACCEPTED AND AGREED:

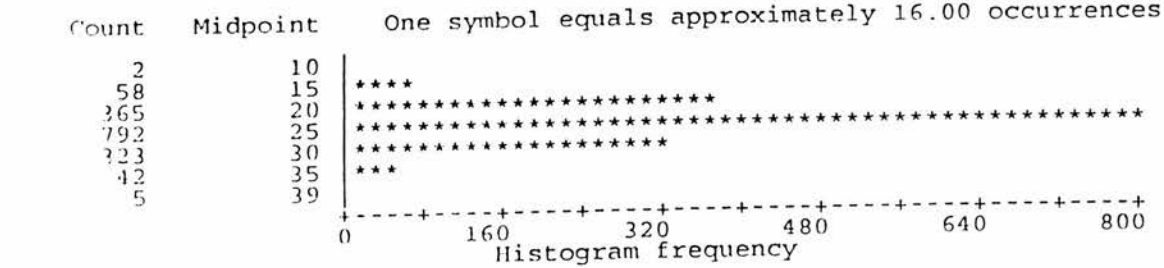
BY: R. Bob Smith
R. BOB SMITH III, Ph.D.

DATE: 3/22/95

APPENDIX S: DISTRIBUTIONS OF BEDFORD FOULDS PERSONALITY DEVIANCE SCALE (PDS) SCORES IN MEN AND WOMEN

EXTP26 extrapunitive scale

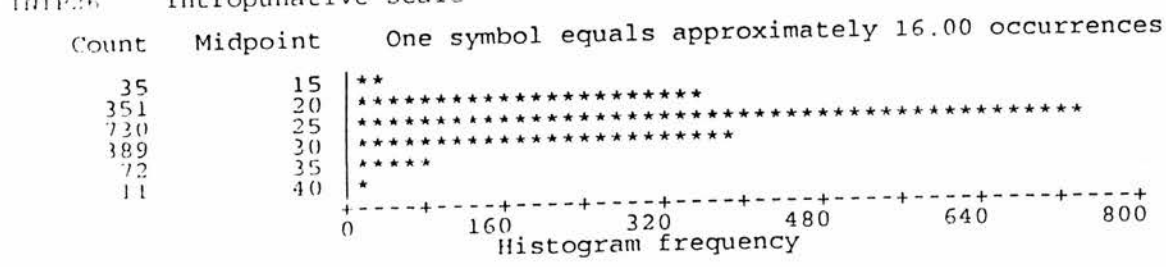
Figure s1. Extrapunitiveness



Valid cases 1587 Missing cases 9

INTP26 intropunative scale

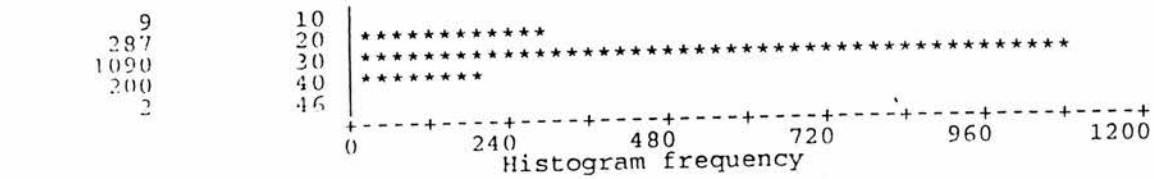
Figure s2. Intropunitiveness



Valid cases 1588 Missing cases 8

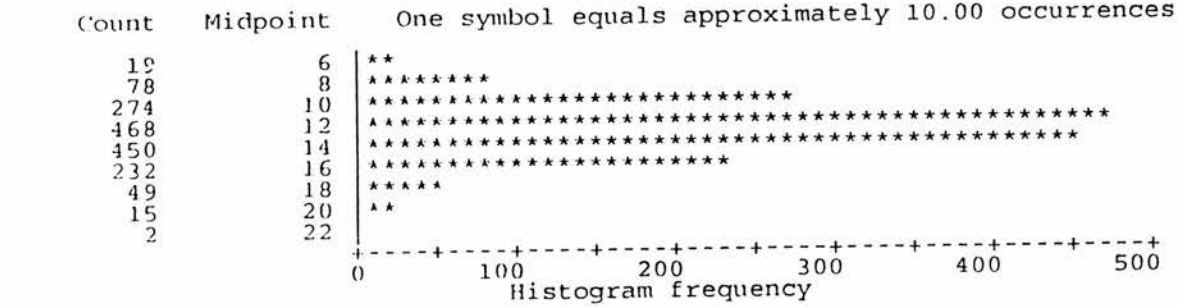
DOM26 dominance scale

Figure s3. Dominance



Valid cases 1588 Missing cases 8

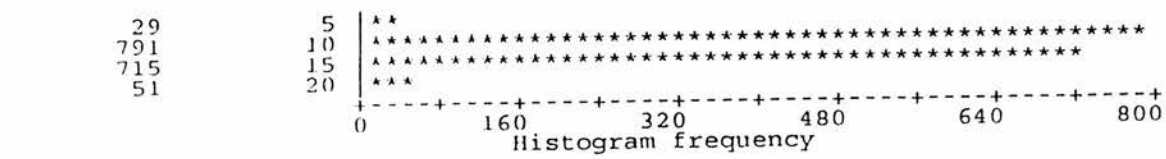
HT26 hostile thoughts



Valid cases 1587 Missing cases 9

Figure s4. Hostile thoughts

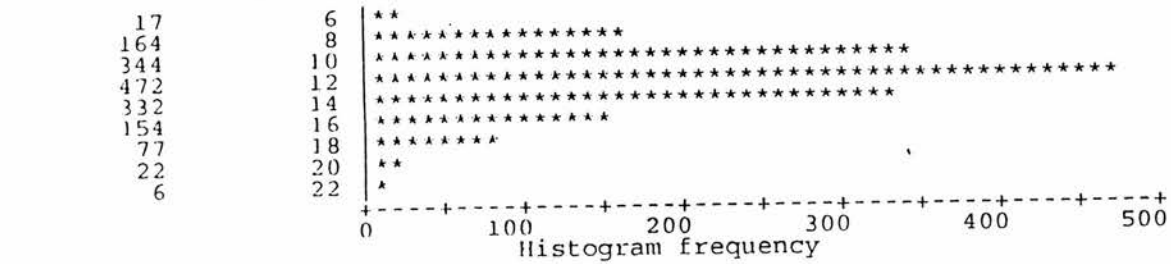
DO26 denigratory towards others



Valid cases 1586 Missing cases 10

Figure s5. Denigratory attitude

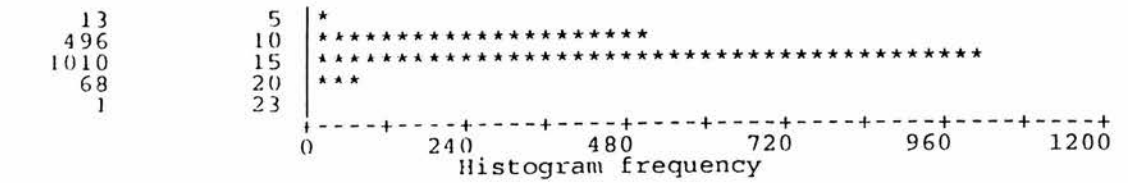
LSC26 lack of selfconfidence



Valid cases 1588 Missing cases 8

Figure s6. Lacks self confidence

DEP26 over-dependent

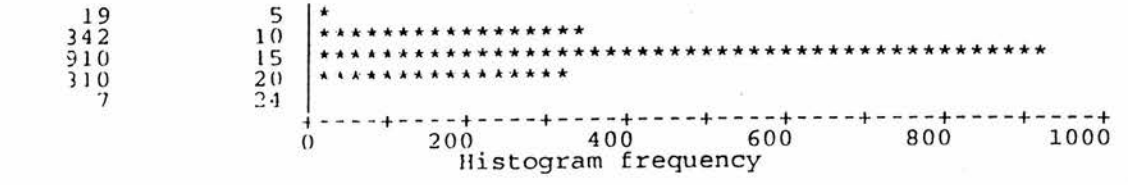


Valid cases 1588 Missing cases 8

Figure s7. Over-dependence

- -

DOMN26 domineering attitude

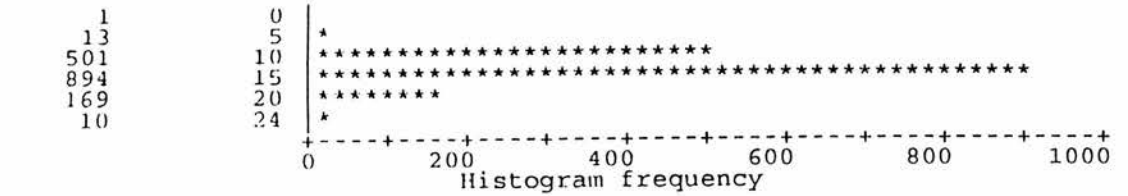


Valid cases 1588 Missing cases 8

Figure s8. Domineering attitude

- -

HOA26 hostile acts



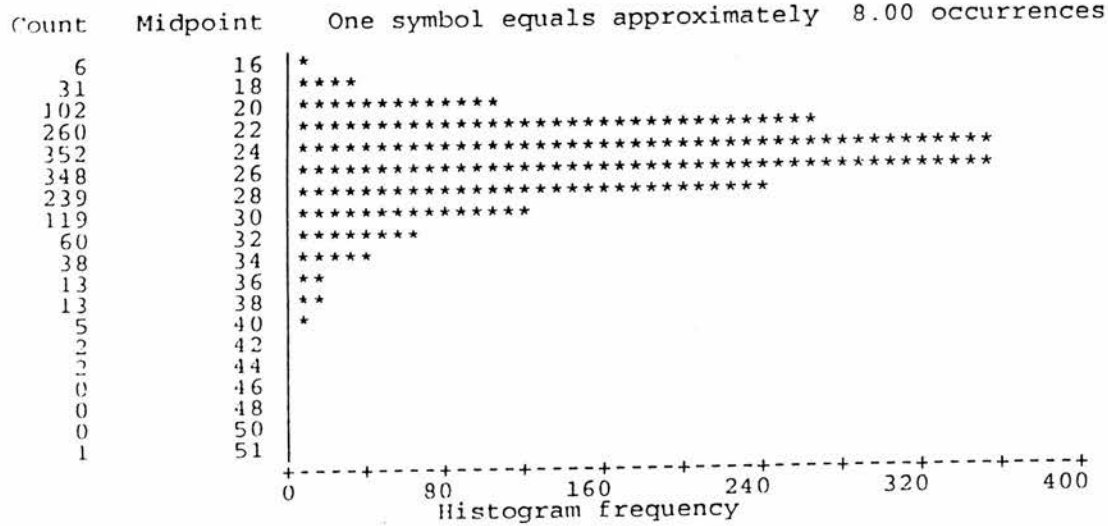
Valid cases 1588 Missing cases 8

Figure s9. Hostile acts

APPENDIX T: DISTRIBUTIONS OF CORONARY RISK FACTORS MEASURED AT BASELINE

BMI

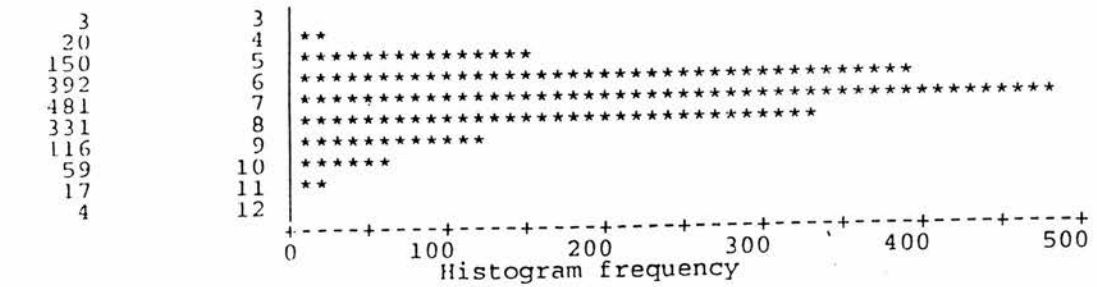
Figure t1. Body mass index



Valid cases 1591 Missing cases 5

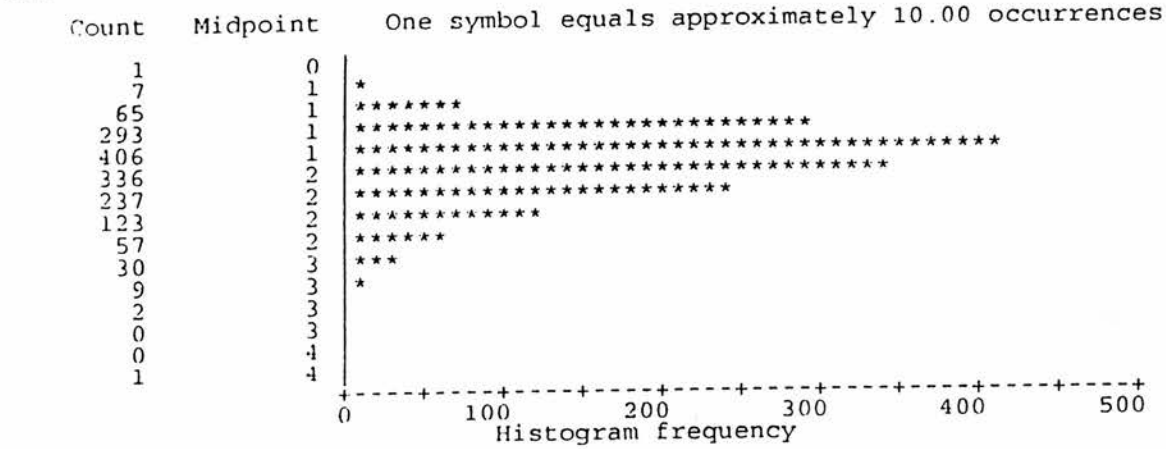
CHOLEST

Figure t2. Total serum cholesterol



Valid cases 1573 Missing cases 23

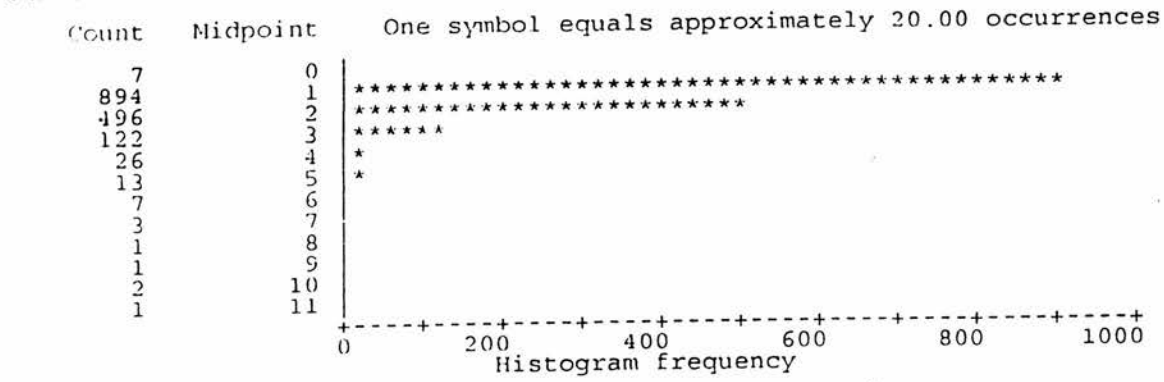
HDL



Valid cases 1567 Missing cases 29

Figure t3. High-density lipoprotein cholesterol

TGLYC



Valid cases 1573 Missing cases 23

Figure t4. Triglycerides

ANKMIN min ankle ratio

One symbol equals approximately 10.00 occurrences

Count	Midpoint	
3	.23	
2	.33	
17	.43	**
21	.53	**
36	.63	****
45	.73	*****
112	.83	**********
233	.93	**********************
452	1.03	**
388	1.13	**
205	1.23	*********************
53	1.33	*****
9	1.43	*
4	1.53	
0	1.63	
1	1.73	
0	1.83	
0	1.93	
0	2.03	
1	2.13	

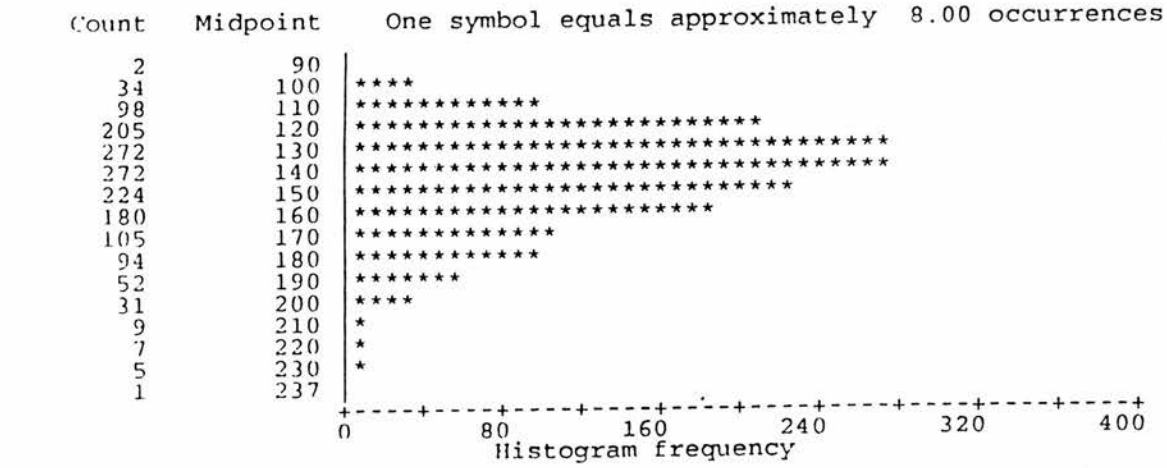
0 100 200 300 400 500

Histogram frequency

Valid cases 1582 Missing cases 14

Figure t5. Ankle brachial pressure index

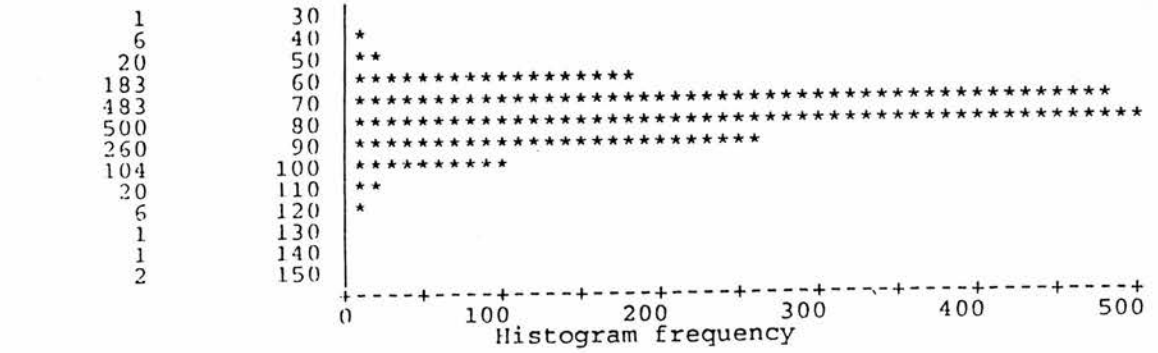
SYSTOLIC



Valid cases 1591 Missing cases 5

Figure t6. Systolic blood pressure

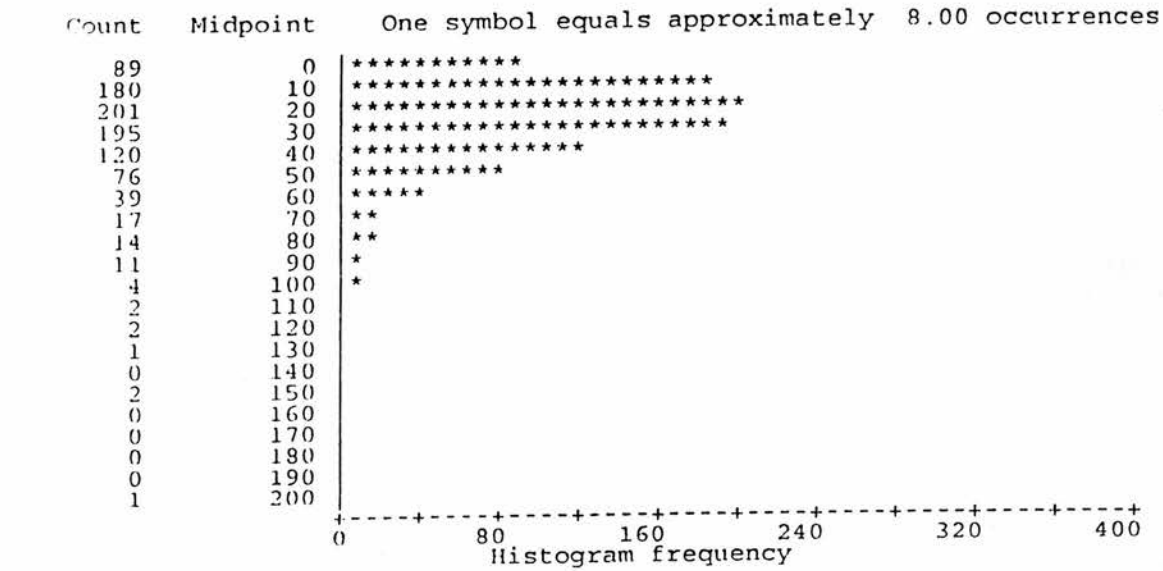
DIASTOLI



Valid cases 1587 Missing cases 9

Figure t7. Diastolic blood pressure

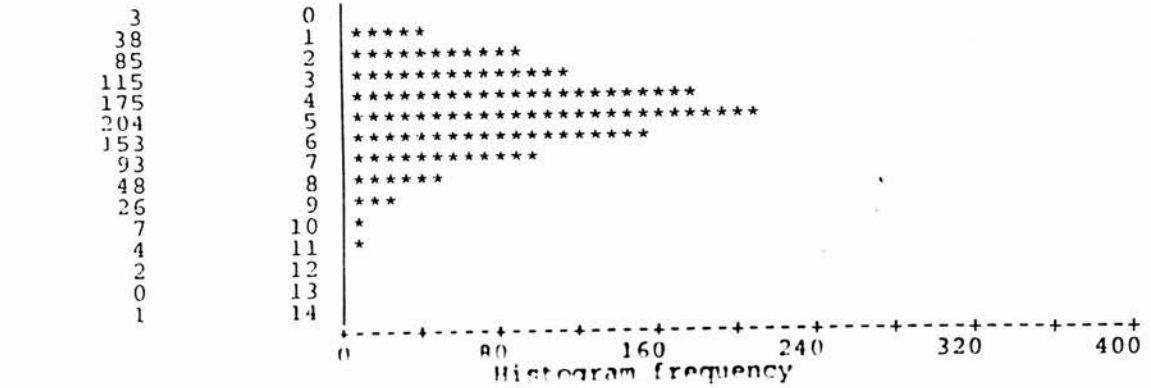
PACKYEAR



Valid cases 954 Missing cases 642

Figure t8. Smoking-packyears raw values

POOTPACK



Valid cases 954 Missing cases 642

Figure t9. Smoking- packyears Transformed by square-root

```
TOTALC1   total units in week

   Count   Midpoint   One symbol equals approximately 20.00 occurrences

    895        0   |*********************************************
    411       10   |*********************
    156       20   |********
     69       30   |***
     31       40   |**
     14       50   |*
      9       60   |
      2       70   |
      3       80   |
      1       90   |
      1       96   |
                   +----+----+----+----+----+----+----+----+----+----+
                   0        200       400       600       800      1000
                                 Histogram frequency

Valid cases     1592       Missing cases      4
- - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - -
```

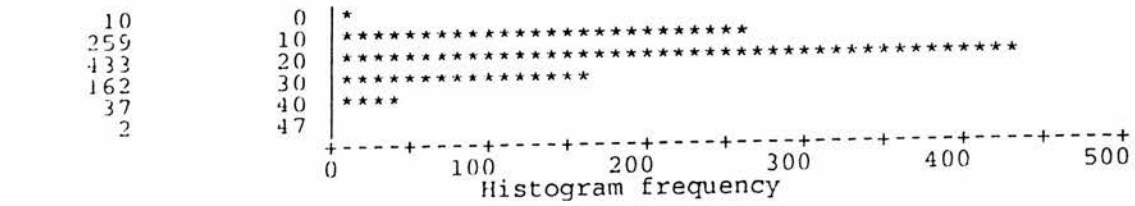
Figure t10. Alcohol units-
raw values

```
ROOTALC

   Count   Midpoint   One symbol equals approximately 12.00 occurrences

    513        0   |*******************************************
    236        1   |********************
    263        2   |**********************
    238        3   |********************
    159        4   |*************
    106        5   |*********
     44        6   |****
     21        7   |**
      7        8   |*
      4        9   |
      1       10   |
                   +----+----+----+----+----+----+----+----+----+----+
                   0        120       240       360       480       600
                                 Histogram frequency

Valid cases     1592       Missing cases      4
```

Figure t11. Alcohol units-
transformed by
square root

APPENDIX U: DISTRIBUTIONS OF NEO FIVE FACTOR INVENTORY SCALE SCORES

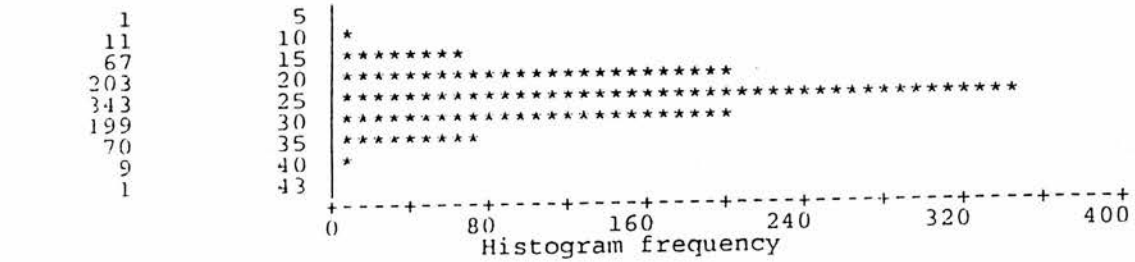
NEUR neuroticism scores



Valid cases 903 Missing cases 693

Figure u1. Neuroticism

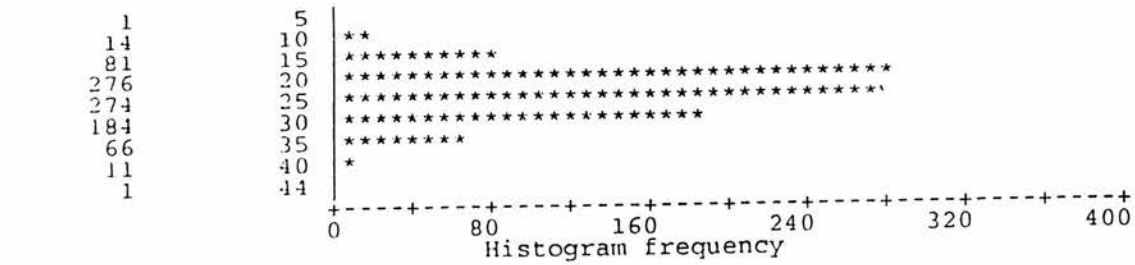
EXTRA extraversion scores



Valid cases 904 Missing cases 692

Figure u2. Extraversion

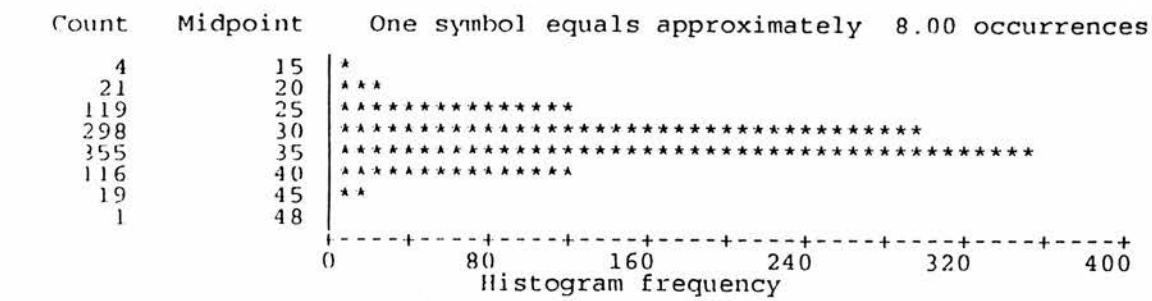
OPEN openness scores



Valid cases 908 Missing cases 688

Figure u3. Openness

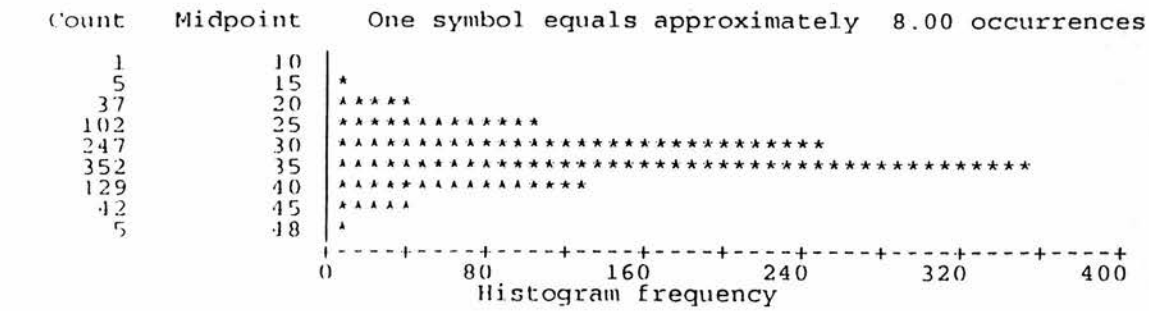
AGREE agreeableness scores



Valid cases 933 Missing cases 663

Figure u4. Agreeableness

CONSC conscientiousness scores



Valid cases 920 Missing cases 676

Figure u5. Conscientiousness

APPENDIX V: DISTRIBUTIONS OF STATE TRAIT ANGER EXPRESSION INVENTORY SCALE SCORES

TAIG total anger scores

Figure v1. Total anger

Count	Midpoint	One symbol equals approximately 12.00 occurrences
149	10	**********
503	15	**
240	20	********************
54	25	*****
10	30	*
1	33	

0 120 240 360 480 600
Histogram frequency

Valid cases 957 Missing cases 639

- -

TAIGT total anger T scores

Figure v2. Angry temperament

Count	Midpoint	One symbol equals approximately 8.00 occurrences
332	4	**
378	6	**
203	8	**************************
53	10	*******
12	12	**
3	14	
2	16	

0 80 160 240 320 400
Histogram frequency

Valid cases 983 Missing cases 613

- -

TAIGR total anger R scores

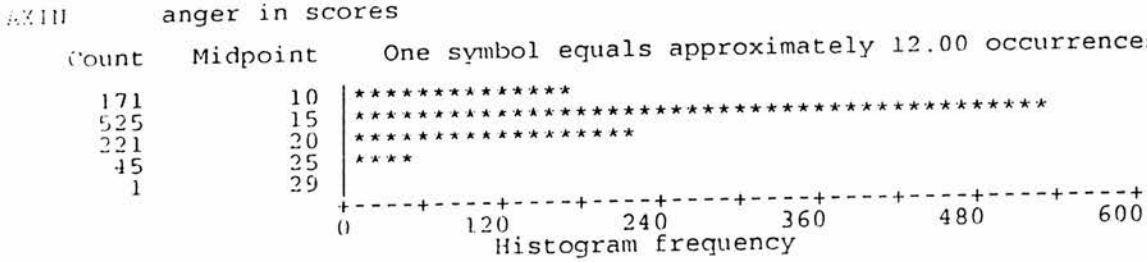
Figure v3. Angry reaction

Count	Midpoint	One symbol equals approximately 10.00 occurrences
62	4	******
210	6	*********************
430	8	**
164	10	*****************
80	12	********
24	14	**
8	16	*

0 100 200 300 400 500
Histogram frequency

Valid cases 978 Missing cases 618

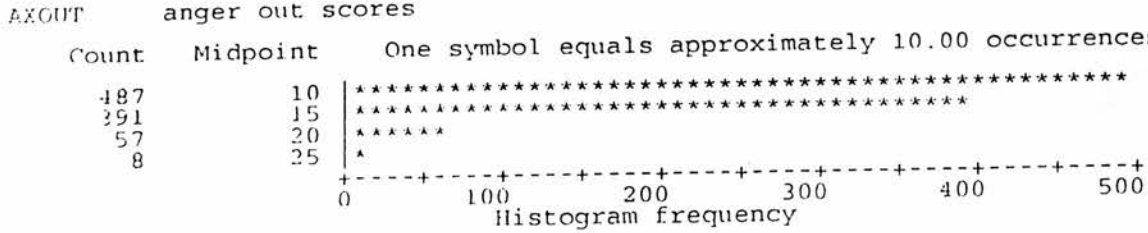
AXIN anger in scores



Valid cases 963 Missing cases 633

Figure v4. Anger-in

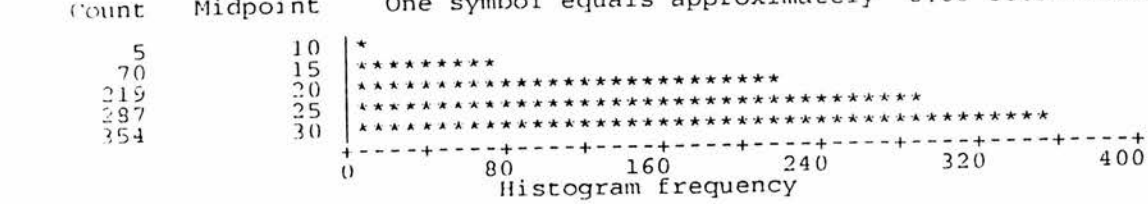
AXOUT anger out scores



Valid cases 943 Missing cases 653

Figure v5. Anger-out

AXCON anger control scores



Valid cases 935 Missing cases 661

Figure v6. Anger control

AXEX total anger expression scores

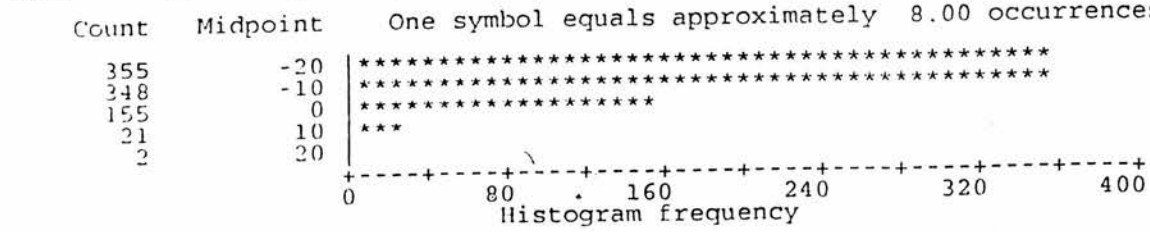
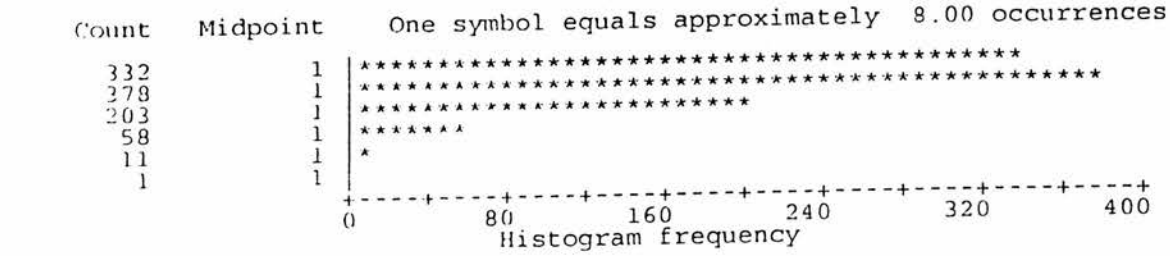


Figure v7. Anger expression

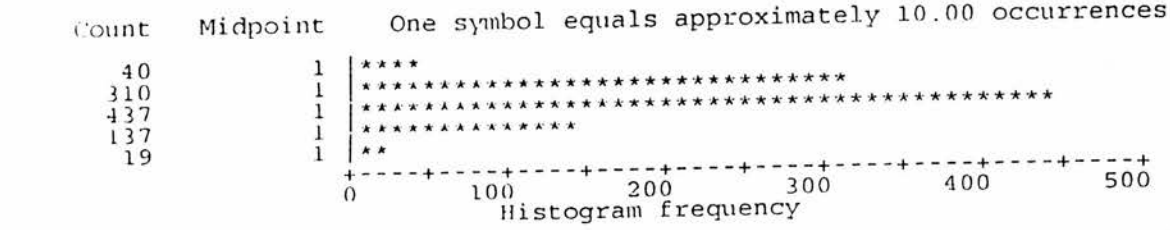
LGTANGT



Valid cases 983 Missing cases 613

Figure v8. Angry temperament–logarithmically transformed

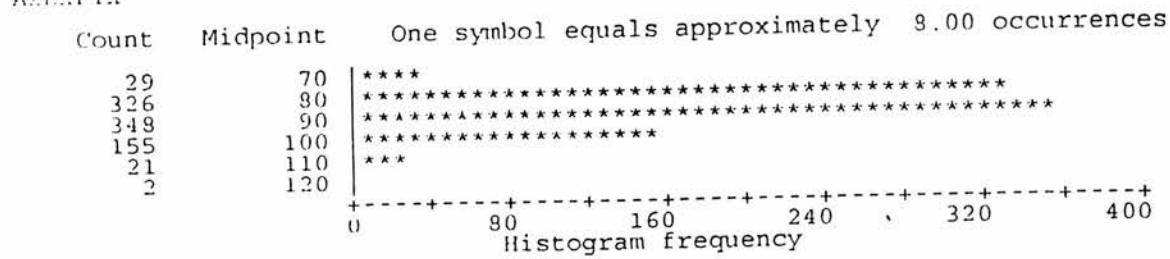
LGAXOUT



Valid cases 943 Missing cases 653

Figure v9. Anger–out–logarithmically transformed

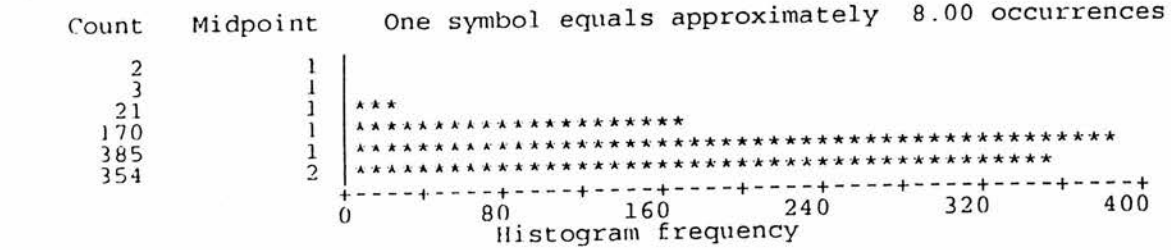
AXEXFIX



Valid cases 891 Missing cases 715

Figure v10. Anger expression–constant of 50 added

LGAXCON



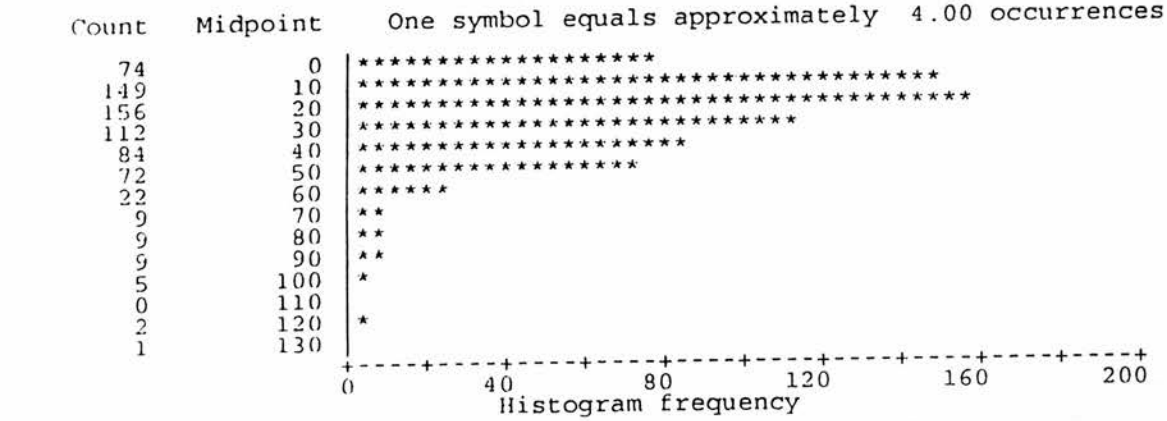
Valid cases 935 Missing cases 661

End of job.

Figure v11. Anger control–logarithmically transformed

APPENDIX W: DISTRIBUTIONS OF CORONARY RISK FACTORS MEASURED AT FOLLOW?UP

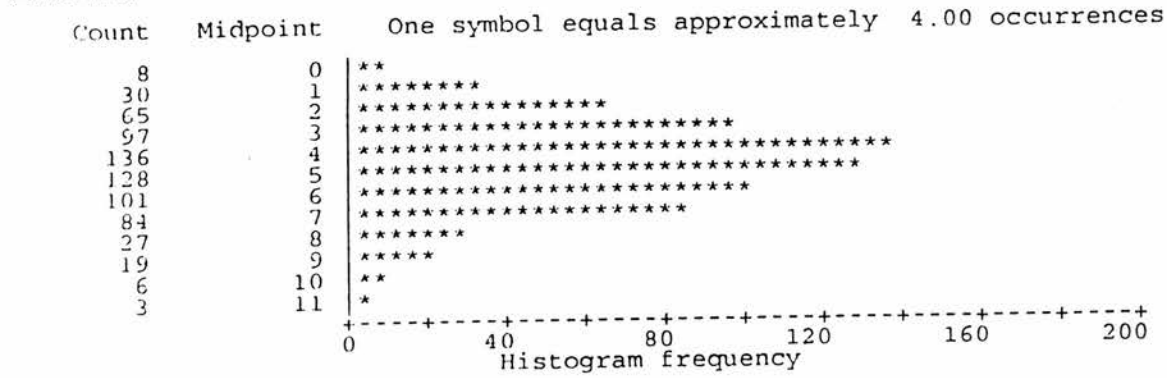
FUPPACK



Valid cases 704 Missing cases 892

Figure w1. Smoking-packyears raw values

SFUPPACK



Valid cases 704 Missing cases 892

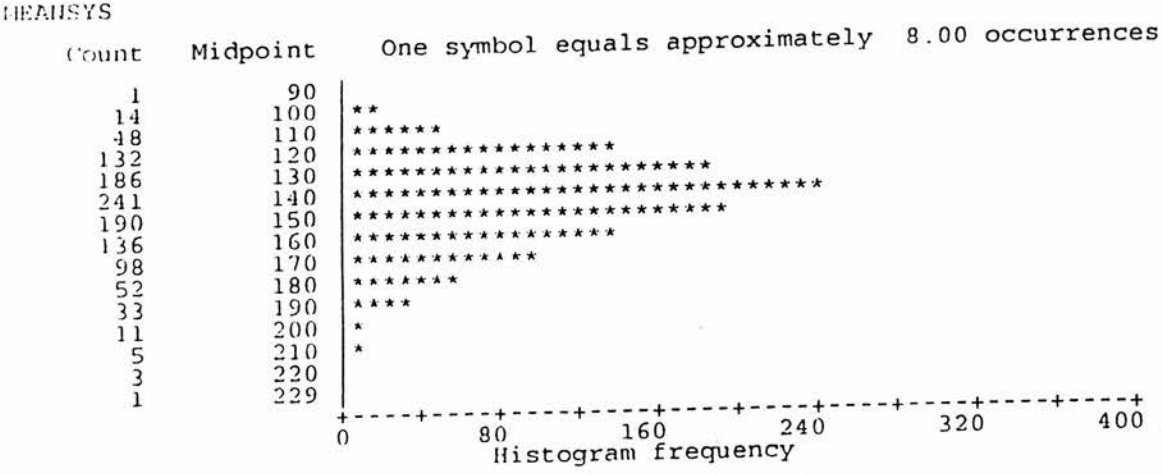
Figure w2. Smoking-packyears transformed by square root

FUABPI

```
   Count    Midpoint     One symbol equals approximately  8.00 occurrences
       4         .30 |*
       4         .40 |*
       9         .50 |*
      24         .60 |***
      43         .70 |*****
      53         .80 |*******
     166         .90 |**********************
     263        1.00 |***********************************
     272        1.10 |************************************
     167        1.20 |**********************
      56        1.30 |*******
      13        1.40 |**
       2        1.50 |
       1        1.60 |
       1        1.70 |
       1        1.80 |
       0        1.90 |
       0        2.00 |
       1        2.10 |
       1        2.18 |
                     +----+----+----+----+----+----+----+----+----+----+
                     0         80        160       240       320       400
                                Histogram frequency

Valid cases     1081      Missing cases     515
```

Figure w3. Anklè brachial pressure index

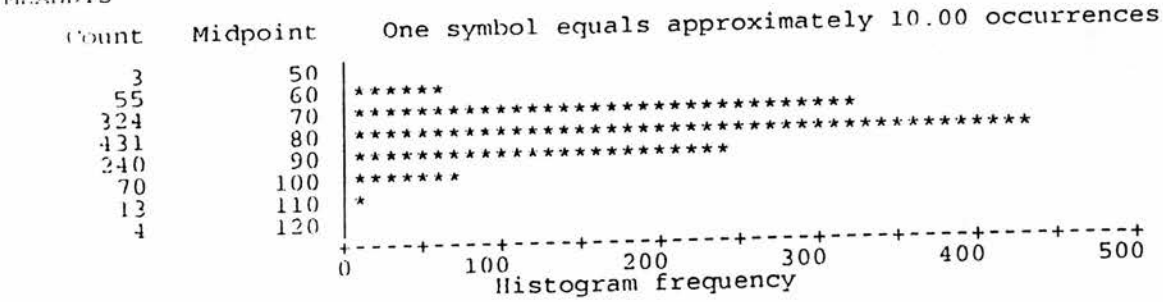
MEANSYS



Valid cases 1151 Missing cases 445

Figure w4. Systolic blood pressure

MEANDYS



Valid cases 1140 Missing cases 456

Figure w5. Diastolic blood pressure

MAXIMT

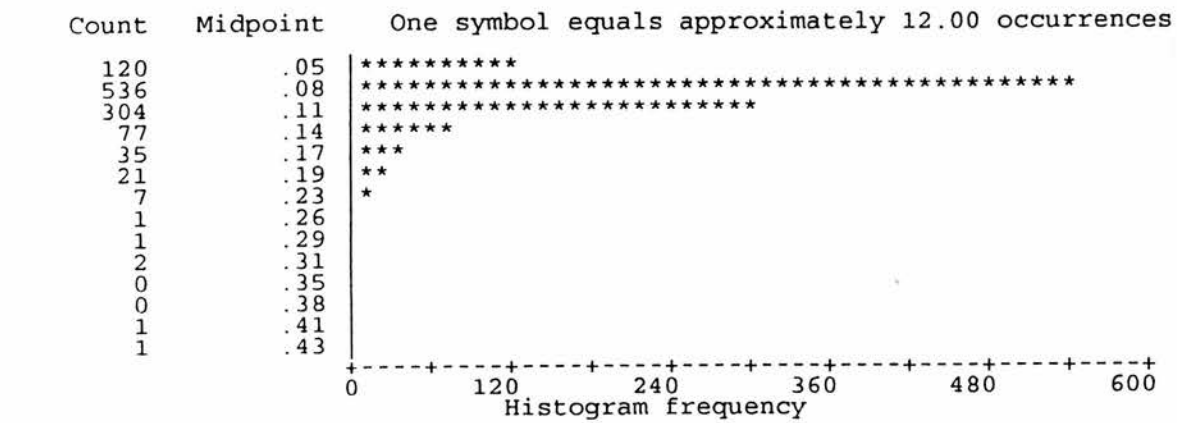


Figure w6. Intima-media thickness; non transformed

LGMAXIMT

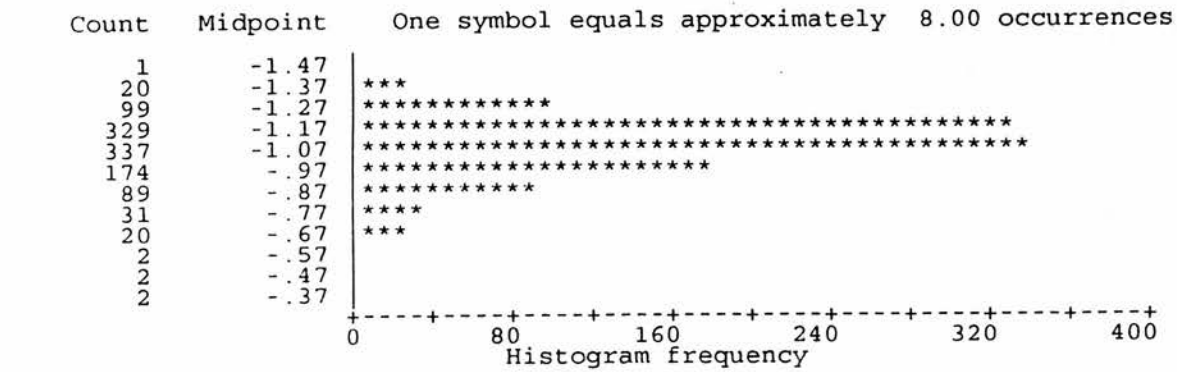


Figure w7. Intima-media thickness; logarithmically transformed

Valid cases 1106 Missing cases 486

THE LANCET

Submissiveness and protection from coronary heart disease in the general population: Edinburgh Artery Study

M C Whiteman, I J Deary, A J Lee, F G R Fowkes

Summary

Background Type A behaviour and, more specifically, hostility and anger have been associated with increased risk of coronary heart disease (CHD). But less attention has been paid to other features of personality. Our aim was to assess whether a submissiveness trait, which is independent of hostility, was related to future risk of CHD in the general population.

Methods The Edinburgh Artery Study is a cohort study of a random sample of 809 men and 783 women aged 55 to 74 years. At the baseline examination in 1988, we administered the Bedford-Foulds Personality Deviance Scales. The participants were followed up for 5 years for cardiovascular events. Criteria to define events were adapted from the American Heart Association. Events were ascertained from the Information and Statistics Division of the Scottish Office Home and Health Department, general practitioners, the UK National Health Service Central Register, annual questionnaires to the participants, and the second examination at the end of follow-up.

Findings During follow-up, 57 (7·0%) men and 28 (3·6%) women had non-fatal myocardial infarctions; 25 (3·1%) men and 8 (1·0%) women had fatal myocardial infarctions; and 48 (5·9%) men and 41 (5·2%) women developed angina pectoris. We found that mean submissiveness scores were significantly higher in men and women who did not have a non-fatal myocardial infarction than in those who did (18·88 [SE 0·15] *vs* 17·70 [0·40], p=0·023 in men; 20·76 [0·17] *vs* 18·18 [0·86], p=0·002 in women). In multiple logistic-regression models, submissiveness remained independently associated with risk of myocardial infarction in women only; a decreased risk of both non-fatal myocardial infarction (relative risk 0·59 [95% CI 0·40–0·85]) and, to a lesser extent, total myocardial infarction (0·69 [0·27–0·96]), was associated with an increase of 1 SD in submissiveness.

Interpretation The personality trait of submissiveness may be protective against non-fatal myocardial infarction, particularly in women. A better understanding is required of the complicated effects of personality on CHD development.

Lancet 1997; **350:** 541–45

Wolfson Unit for the Prevention of Peripheral Vascular Diseases, Department of Public Health Sciences (M C Whiteman MSc, A J Lee PhD, Prof F G R Fowkes FRCPE), **and Department of Psychology** (Prof I J Deary FRCPE) **University of Edinburgh, Edinburgh EH8 9JZ, UK**

Correspondence to: Prof I J Deary

Introduction

The type A behaviour pattern and its relation to coronary heart disease (CHD) have been studied extensively since the 1960s. Type A individuals are characterised by their wish to do too much in too little time, competitiveness, frustration, and aggression.[1] Prospective studies of general population samples have found that the pattern is associated with increased risk of CHD,[2,3] although studies on people at high risk and on disease diagnosed by coronary angiography have not consistently shared this finding.[4–6] There has also been much interest in discovering the adverse components of the heterogeneous type A behaviour pattern—in particular, whether hostility has a role.[7,8] Quantitative and descriptive reviews of these studies concluded that some aspects of hostility were related to CHD, but that further research was necessary.[9–11] Meta-analysis of the relation between hostility and heart disease suggested that this relation was strongest when objective indicators of CHD were used in prospective population studies.[12]

There has been less investigation of features of personality other than type A/B or hostility. We found that the trait of submissiveness/low self-confidence (hereafter denoted as submissiveness) was independent of hostility in the Edinburgh Artery Study.[13] A submissive person, as assessed by the Bedford-Foulds Personality Deviance Scales (PDS)[14] which were used in the study, prefers to stay in the background and to let others lead and dominate. The scale also contains items related to low self-confidence and lack of self-assurance.

The PDS-submissiveness trait is not merely a surrogate for type B behaviour. The type B person has been defined as somebody who is not impatient, competitive, or hostile; he or she is modest, enjoys being relaxed, feels secure, and has adequate self-esteem.[1] A person who scores highly on PDS-submissiveness may stay in the background (neither competitive nor domineering), unlike a type A person, but is not strictly type B, since the submissive person does not have the sense of security that defines a type B person. The latter is non-hostile and forgiving (though they will defend themselves if necessary);[1] but a high scorer on PDS-submissiveness may or may not be hostile, because submissiveness and hostility are uncorrelated (orthogonal) dimensions.[13] The Western Collaborative Group Study found that submissive men had a lower 22-year mortality rate than dominant men.[15] In our study, we specifically aimed to assess whether this independent trait of submissiveness was related to the risk of developing CHD.

Methods

Study population

The first phase of the Edinburgh Artery Study began in 1988 as a cross-sectional survey of 1592 men and women aged 55 to 74 years. The population was selected by age-stratified random sampling from the age-sex registers of ten general practices with catchment areas in a wide range of socioeconomic and

geographical districts of Edinburgh. The response rate was 65%, and follow-up of a sample of non-responders showed no substantial bias. Details of the study population and recruitment have been reported previously.[16] The participants were followed up for 5 years for cardiovascular events, and death, and were invited to attend a second medical examination at the end of that period. The study was approved by the Lothian Health Ethics Committee, and informed consent was obtained from each participant.

Examinations

Baseline examinations were done in 1988 and follow-up examinations in 1993.[17] At both examinations, specially trained nurses recorded an electrocardiogram (ECG), measured each participant's height and weight, and also withdrew 30 mL venous blood for analysis of haemostatic and rheological factors. All ECGs were later coded with the Minnesota code[18] separately by two observers. A consultant cardiologist made a final decision if there was a discrepancy. Each participant completed a questionnaire at baseline and follow-up that included personal characteristics, occupation, smoking, alcohol consumption, and medical history, such as recall of a doctor's diagnosis of angina, and the WHO angina questionnaire.[19]

Personality measurement

The Bedford-Foulds PDS[14] were administered at the baseline examinations as part of the questionnaire. The PDS contain 36 items and elicit three secondary traits: extrapunitiveness, which comprises two primary scales, hostile thoughts, and denigratory attitude towards others; intropunitiveness, which is the sum of the two primary scales of lack of self-confidence and over-dependence on others; and dominance, which comprises outright hostile acts and a domineering attitude. Two revised scales were later derived from item-level factor analysis: hostility and submissiveness/low self-confidence (submissiveness).[13] These revised scales were statistically independent of one another (orthogonal; $r=-0{\cdot}07$). Since the original scales were not retrieved in factor analysis, only the two—psychometrically sounder—revised scales are used in this analysis. To allow for comparisons with previous studies, we acknowledge that submissiveness is most strongly correlated with the PDS primary scales of lack of self-confidence ($r=0{\cdot}85$) and domineering attitude ($r=-0{\cdot}79$). Revised hostility showed the strongest correlations with hostile thoughts and hostile acts ($r=0{\cdot}87$ and 0·68, respectively). The revised hostility score is derived from eight questions, allowing a range of 8–32. One question from this scale is: "Most of my life, when I've thought I was justified in losing my temper, I have done so in no uncertain terms." The submissiveness scale is based on nine questions and the range, therefore, is 9–36. One statement from this scale is "Most of my life, I have preferred to stay in the background."

Identification of cardiovascular events

Information on fatal and non-fatal myocardial infarction, angina, and deaths from all causes was obtained over the 5-year follow-up period. Criteria to define cardiovascular events were adapted from the American Heart Association,[20] and an event was recorded only if these criteria were met.

Non-fatal non-silent myocardial infarction was defined as at least two of: sustained (>20 min) cardiac pain; ECG, coded according to the Minnesota coding system[18] as 1.1.1.–1.2.5, 1.2.7–1.3.6, 4.1–4.3, 5.1–5.3, or 9.2; raised serum concentrations of creatine phosphokinase, lactate dehydrogenase, aspartate aminotransferase, or creatine phosphokinase MB isoenzyme, not attributable to another cause.

Non-fatal, silent myocardial infarction was defined as Minnesota ECG codes[18] 1.1.1.–1.2.5, 1.2.7, or 9.2 plus 5.1 or 5.2, in the absence of raised enzyme concentrations and cardiac pain, as long as the ECG at baseline was coded as normal.

The requirements for fatal myocardial infarction were that acute myocardial infarction was found at necropsy; that the criteria for definite myocardial infarction were met in the 4 weeks before death; or that the International Classification of Diseases, version 9, death certificate codes were 410–414 with a history of MI or 410–414 plus necropsy evidence of severe coronary atherosclerosis or previous myocardial infarction. We defined ICD 9 codes 410–414 with no other evidence of myocardial infarction as possible fatal myocardial infarction.

New angina pectoris was recorded if there was no evidence of angina on the WHO questionnaire[19] at baseline, plus one of a positive WHO angina questionnaire[19] during follow-up, plus recall of a doctor's diagnosis of angina; a positive WHO angina questionnaire plus ECG Minnesota codes 1.3, 4.1–4.4, 5.1–5.3, or 7·1; and clinical diagnosis of angina investigated by the general practitioner or in hospital.

To identify deaths in the study cohort, each participant's record was flagged at the UK National Health Service Central Registry, which ensured that the death certificates were automatically forwarded. All cardiovascular deaths were investigated further through hospital or general-practitioner records or both, to check that study criteria were met.

Details of non-fatal events were sought from hospitals, the Information and Statistics Division of the Scottish Office Home and Health Department, and by annual questionnaires to study participants. A card was also given to the participant's general practitioner at the start of the study, to be returned after a cardiovascular event, death, or change of address.

Data analysis

Data on the questionnaires and recording forms from the examinations were checked by clinic staff, coded, and entered onto a DBASE-III database (baseline) or DBASE-IV database (follow-up). All forms were entered twice to control error rates; discrepancies were checked by referral to original records. The data were then transferred to the University of Edinburgh mainframe computer for analysis. The PDS were coded by research staff, and the data entered onto the computer and validated by the University Data Preparation Services.

Data were analysed with the statistical packages SPSS-X (Release 5·0) and SAS (Release 6.03). Participants with a history of angina or myocardial infarction at baseline (142 men, 89 women) were excluded from all analyses. The significance of differences in personality scales in the event and non-event groups was assessed in both men and women by Student's t test. Outcome categories were defined as: all myocardial infarctions (both non-fatal and fatal); fatal myocardial infarction only; non-fatal myocardial infarction (non-silent and silent); and new angina pectoris. The personality scales were then entered into multiple logistic-regression equations for each outcome category, in which each scale was adjusted for age, degree of baseline vascular disease (measured with the ankle-brachial pressure index), and baseline risk factors (social class, systolic and diastolic

	Men (n=809)	Women (n=783)	p
All MI	80 (9·9%)	34 (4·3%)	<0·001
Non-fatal MI	57 (7·0%)	28 (3·6%)	<0·001
Fatal MI	25 (3·1%)	8 (1·0%)	<0·001
Angina	48 (5·9%)	41 (5·2%)	0·391

MI=myocardial infarction.
*A participant may appear in more than one category.

Table 1: **Cumulative CHD events during 5 years of follow-up**

Age-group	Hostility		Submissiveness	
	Men	Women	Men	Women
55–59	17·7 (3·2)	17·7 (3·4)	18·2 (3·1)	20·5 (4·1)
60–64	17·6 (3·4)	17·6 (3·1)	18·6 (3·7)	20·7 (4·4)
65–69	17·6 (3·1)	17·4 (3·5)	18·8 (3·7)	20·8 (4·4)
70–75	17·2 (3·3)	16·4 (3·6)	19·5 (3·3)	20·6 (4·4)
All ages	17·5 (3·2)	17·3 (3·4)	18·8 (3·7)	20·6 (4·3)

*Table adapted from Deary and colleagues.[13]

Table 2: **Mean (SD) PDS hostility and submissiveness scores by age in 774 men and 740 women***

	Myocardial infarction						Angina	
	Non-fatal		Fatal		All		Yes	No
	Yes	No	Yes	No	Yes	No		
Men								
Number of men	57	611	25	643	80	588	48	620
Submissiveness score	17·70 (0·40)	18·88 (0·15)*	19·32 (0·69)	18·76 (0·15)	18·19 (0·37)	18·86 (0·16)	18·63 (0·54)	18·80 (0·15)
Hostility score	17·98 (0·46)	17·38 (0·13)	17·48 (0·64)	17·73 (0·13)	17·88 (0·38)	17·37 (0·13)	17·79 (0·50)	17·46 (0·12)
Women								
Number of women	28	642	8	662	34	636	41	629
Submissiveness score	18·18 (0·86)	20·76 (0·17)†	22·12 (1·47)	20·64 (0·17)	18·94 (0·77)	20·75 (0·17)‡	21·59 (0·71)	20·58 (0·17)
Hostility score	16·71 (0·60)	17·29 (0·13)	17·00 (1·18)	17·27 (0·13)	16·68 (0·55)	17·29 (0·13)	17·56 (0·53)	17·26 (0·13)

Individuals were excluded from scale calculations if all items not completed (which left 774 men and 740 women); those with history of angina or myocardial infarction at baseline are excluded (142 men, 89 women); an individual may appear in more than one category.
*p=0·023, †p=0·002, ‡p=0·019 for differences between those with and without relevant CHD event.

Table 3: **Mean (SE) submissiveness and hostility scores by CHD category in men and women**

blood pressure, serum cholesterol, serum triglycerides, body-mass index, smoking, and alcohol consumption).

For each regression equation, a unit increase in the personality scale was taken as 1 SD. Backward stepwise logistic regression was done, and criteria for removal ($p \geq 0{\cdot}05$) were based on the likelihood-ratio test.

Results

Cumulative CHD event rates were significantly higher in men than in women for all categories except angina, in which the rate was similar for both sexes (table 1).

Women had higher submissiveness scores than men, though hostility scores were similar in both sexes (table 2). Mean scores varied little with age; in men aged 70–75, however, submissiveness scores were slightly higher than at younger ages.

The mean scores of submissiveness and hostility in each CHD category are shown in table 3. There were no differences in personality scores for non-cardiovascular deaths (data not shown), so this outcome is not included in the table. There were significant differences in submissiveness scores between participants with and without various CHD events. Women with non-fatal myocardial infarctions had a lower mean submissiveness score than those without this event (p=0·002; effect size 0·5 SDs). Men with a non-fatal myocardial infarction had a lower mean score than men without (p=0·023; table 3). For all myocardial infarction, those with the event had lower submissiveness scores than those without, but the differences were not significant. For fatal myocardial infarction, the differences in scores were very small, but in the opposite direction; numbers of events, however, were small. The difference in submissiveness in angina groups was also reversed from the non-fatal myocardial infarction groups; there were slightly higher scores in those with angina than in those without, but, again, the differences were small and not significant. Hostility scores were higher in both men and women who had a non-fatal myocardial infarction than in those who did not, but the differences were not significant.

	Relative risk (95% CI)*	
	Men	Women
Non-fatal myocardial infarction		
Submissiveness	0·84 (0·63–1·11)	0·59 (0·40–0·85)†
Hostility	1·15 (0·88–1·48)	1·02 (0·71–1·46)
Fatal myocardial infarction		
Submissiveness	0·98 (0·72–1·63)	1·46 (0·74–2·89)
Hostility	1·04 (0·66–1·44)	1·01 (0·47–2·17)
Total myocardial infarction		
Submissiveness	0·89 (0·70–1·13)	0·69 (0·27–0·96)‡
Hostility	1·13 (0·89–1·41)	1·03 (0·70–1·39)
Angina		
Submissiveness	1·03 (0·75–1·41)	1·25 (0·89–1·76)
Hostility	1·00 (0·74–1·35)	1·06 (0·74–1·53)

*Adjusted for age, degree of baseline vascular disease, social class, systolic and diastolic blood pressure, serum cholesterol, serum triglycerides, body mass index, smoking, and alcohol consumption.
†p≤0·01, ‡p≤0·05.

Table 4: **Relative risks of CHD for 1 SD increase in submissiveness and hostility scores in men and women**

The backward stepwise logistic regression resulted in models that showed that in nearly all the disease categories, baseline degree of vascular disease was an important contributor to risk (data not shown). Baseline vascular disease was estimated with the ankle brachial pressure index, with a low value indicative of more severe disease. In the models, an increase in ankle brachial pressure index was associated with an 80–90% reduction in risk. For brevity, only the risks associated with the personality variables are shown in table 4. In women, the PDS submissiveness score remained independently associated with non-fatal myocardial infarction; an increase of 1 SD in the submissiveness score was associated with a relative risk of 0·59 (95% CI 0·40–0·85), a 41% decrease in risk. In the category of all myocardial infarction in women, an increase in submissiveness of 1 SD was also associated with a decrease in risk of 31% (relative risk 0·69 [0·27–0·96]). These relations were independent of age, degree of baseline vascular disease, social class, systolic and diastolic blood pressure, serum cholesterol, triglycerides, body mass index, smoking, and alcohol consumption. In men, neither personality factor (hostility nor submissiveness) remained independently associated with any of the cardiovascular outcomes.

Discussion

The main finding of this study is that the personality trait of submissiveness seems to confer protection against non-fatal myocardial infarction. Mean submissiveness scores were significantly lower in both men and women who had experienced a non-fatal myocardial infarction than in those who had not. After adjustment for the potential confounding factors, submissiveness remained independently associated with non-fatal myocardial infarction only in women.

The Western Collaborative Group study found a reduced mortality rate among men who were more submissive.[15] In the Whitehall II study, however, both male and female civil servants in London with greater job control—who were apparently less submissive—were at decreased risk of self-reported CHD.[21] In a study of Olive baboons, moreover, Sapolsky[22] found a possible adverse health effect for submissiveness: subordinate male baboons had poorer cardiovascular responses to stress

(caused by the introduction of a new dominant troop member) than dominant baboons. In cynomolgus macaque monkeys, dominant females developed little atherosclerosis, whereas subordinate females resembled males in the extent of atherosclerotic lesions.[23]

Thus, the finding that submissiveness may protect against myocardial infarction is complicated, and difficult to interpret. For example, the PDS measure of submissiveness may not be comparable with observations of social subordinance or with job control; the latter focuses on a person's environment, whereas PDS personality-trait measures focus on the person. In addition, one feature of the items on the PDS-submissiveness scale is the emphasis on contentment with personal role. The protective effect may therefore be apparent in submissive people because they are content to be this way. In the Whitehall study, people in positions of low job control, who are at greater risk, may not be submissive (a personal characteristic), but are forced to be subordinate (an environmental demand). Sapolsky[22] observed that subordinate baboons who avoided conflict with dominant animals during times of confrontation were not adversely affected, and that dominant baboons showed a marked cardiovascular response if involved in an interaction that challenged their dominance. His interpretation was that "social instability is not intrinsically a stressor—it appears to depend on whether one is fortunate enough to remain a spectator during such instability".[22] This interpretation is further supported by a study of captive female macaque monkeys fed an atherogenic diet.[30] In most cases, dominant females who became subordinate when switched to a different social group, and subordinate females who became dominant, experienced a significant excess of atherosclerosis compared with those who remained in their original social position.

Although not necessarily related to the concept of job control, the PDS-submissiveness scale does modestly correlate with the personality trait of neuroticism.[13] This trait has been linked to symptom-reporting behaviour, including reports of chest pain that may lead to a diagnosis of angina,[10,25] which accords with the slightly higher scores we observed in the participants with angina. However, the use of objective criteria to measure disease helps to avoid confusion between personality factors related to symptom reporting rather than true disease.[12,25] The statistically significant association found in this study was with the objectively assessed coronary outcome of non-fatal myocardial infarction. The other objectively assessed outcome, fatal myocardial infarction, did not show significant differences in personality variables. The direction of mean differences for fatal myocardial infarction was opposite to that in non-fatal myocardial infarction, but the magnitude of differences and the numbers of fatal myocardial infarctions were very small. A higher base rate of fatal myocardial infarction would be required to examine properly its relation to personality. Also, some bias may have been introduced because the comparison group for each outcome was not necessarily disease-free. For instance, the comparison group for fatal myocardial infarction included 88 participants who had angina diagnosed during follow-up; and 27 people who died of non-cardiovascular causes, but who also had angina, were included in the angina outcome (none of the participants who had non-fatal myocardial infarction died from a non-cardiovascular cause). In this age-group, even those who did not have a diagnosed event may have significant atherosclerosis. We were thus examining the relation of personality to the specific outcome (ie, non-fatal myocardial infarction) compared with not having that outcome (ie, not having non-fatal myocardial infarction but possibly having new angina). This slight dilution of the comparison groups would have made differences between the groups less extreme, and biased our results towards the null. This, in turn, could lead to underestimation of effect sizes.

The difference in submissiveness scores is difficult to translate into meaning for day-to-day life. Extreme scores on the original scales have been associated with various psychiatric illnesses,[13] but the mean scores in the outcome groups here are not extreme. The usefulness of the submissive-scale scores in this context is therefore pragmatic, in that they enhance the predictive power of models that incorporate established risk factors. The effect size of the difference in submission scores between women with and without non-fatal myocardial infarction is medium.[31] The relative risk of 0·59 with an increase in submissiveness of 1 SD reinforces the additional predictive value of the measure.

Although the PDS have been widely used in Europe, the psychometric properties of the scales were not assessed until 1995.[13] The revised submissiveness scale is derived from items originally used to calculate the primary PDS scales of lack of self-confidence and domineering attitude. The new hostility scale includes items from the hostile-acts and hostile-thoughts primary scales. The two new scales were uncorrelated (orthogonal); that is, they measure separate constructs. A person's scores on the submissiveness and hostility dimensions, therefore, vary independently, with no bearing on each other. Interestingly, it is the submissive-dominance dimension, rather than hostility, that is associated with cardiac events in this study. Previous analyses of peripheral arterial disease and PDS in the Edinburgh Artery Study showed that a higher hostile-acts score was significantly related to increased severity of disease.[27] However, self-reported hostility measures generally show weaker relations to CHD than interview measures.[10] In Friedman's study,[1] 12 of 21 patients reported themselves to be non-hostile (Cook-Medley Hostility Scale[28]), but on examination 20 of 21 were found to be severely hostile. The PDS are self-reported, so if individuals underestimate their hostility—which in turn is related to CHD—the magnitude of the association will be attenuated. Alternatively, age could have an effect: hostility is a stronger risk factor in younger age-groups,[12,29] whereas the Edinburgh Artery Study is an older cohort.

Submissiveness might not protect against myocardial infarction in younger age-groups; the contribution of risk may shift with age, as it does with hostility. We could have examined a group of survivors if some of the potential "submissive" participants had died. However, the findings for the age-group can be extrapolated to the larger relevant population with reasonable confidence, since the study cohort was a random sample of the general population, and is representative of the population aged 55–74.

Given the ages of the participants, we cannot exclude the possibility that their submissiveness and hostility scores have been shaped by their social class or working history. Experience of CHD in others may also affect personality, since individuals might try to change themselves or their lifestyles to protect against CHD. However, the

structions for the PDS specifically state "describe how you may have felt, thought, or acted during *most of your life*" (emphasis in original). The scale scores, which are based on the participants' reflections on their personalities throughout their lives, are trait measures. Type A behaviour, by contrast, was defined as a response style,[32] not a trait, and thus strongly influenced by situation. There is good evidence that personality traits are very stable in adulthood.[24] In addition, because participants with a history of CHD at baseline were excluded, personality changes were not likely to have been caused by the disease or diagnosis. However, by age 55, most adults have at least moderate atherosclerosis, and subclinical disease may have effects on personality that are so far unknown. Social class also has a substantial influence on health.[26] For instance, a person's experiences throughout life influence many biological variables, including neuroendocrine stress mechanisms, which are particularly important for coronary disease.[33] Statistical adjustment, therefore, may not fully account for the intricate ways in which social class is bound up with home environment, working environment, personality, and health. The direction of any bias is difficult to assess, however, and these are complex research problems that are common to most behavioural epidemiological studies. Ideally, personality would have to be assessed in a young study group, and continue to be reassessed as the cohort aged and CHD events were being recorded.

The possibility that submissiveness confers protection against non-fatal myocardial infarction in this cohort indicates that, in CHD research, investigation of a wide range of psychological factors is very important. This extra psychological information, especially when linked with our knowledge of physical risk factors, is not trivial; even a small effect size can have a large impact when disease prevalence is high.[9] The known risk factors of hypercholesterolaemia, hypertension, and smoking are undoubtedly important elements in the assessment of risk of CHD; measurement of the extent of atherosclerosis is also essential. However, personality factors may help to improve our prediction of who is at risk of a cardiac event. The "cardiac protectiveness" of submissiveness in our study indicates that a better understanding is required of the various effects of personality traits on CHD risk. Since the finding was particularly evident in women, it is clear that both sexes need to be included in studies of CHD.

Contributors

M C Whiteman carried out the personality literature review, statistical analysis, and collected follow-up data. I J Deary advised on personality testing, analysis, and background literature. A J Lee was the study statistician who organised the data and advised on analysis. F G R Fowkes was the principal investigator and designed the study. All authors contributed to the interpretation of results, and the writing and editing of the paper.

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Appendix Y: Design of Edinburgh Artery Study and Personality Studies within it