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# Cardiovascular Risk in First Degree Relatives of Patients with Premature Coronary Heart Disease 

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#### Abstract

Context: In most parts of the United Kingdom current cardiac services neglect assessment and primary prevention of cardiovascular disease in first degree relatives of patients with proven premature coronary heart disease. First degree relatives are at a higher risk than the general population by virtue of shared lifestyle risk and genetic factors to index cases.

Objectives: This study aimed to identify first degree relatives of patients with proven coronary heart disease and assess their cardiovascular risk, using various cardiovascular risk assessment tools. We also aimed to assess the effectiveness of cardiovascular risk reduction services on the cardiovascular risk of the individual.

Design, Setting, and Participation: A qualitative study was conducted at Sandwell Hospital. 43 participants aged 18-74years were recruited.

Results: The mean age of the cohort was $42( \pm 4)$. $66 \%$ were under the age of 40years. At the baseline appointment $30 \%$ of the cohort, had a systolic blood pressure greater than 140 mmHg , mean $140( \pm 14.8) \mathrm{mmHg}$ and $28 \%$ had a diastolic blood pressure greater than 90 mmHg , mean $94( \pm 2.12)$ mmHg. $82 \%$ of south Asians had a BMI greater than $23 \mathrm{Kg} / \mathrm{m}^{2} .63 \%$ of non south Asians had a BMI greater than $<25 \mathrm{Kg} / \mathrm{m}^{2} .37 \%$. $61 \%$ of the cohort's total cholesterol was greater than $5 \mathrm{mmol} / \mathrm{l}$, mean $7.1( \pm 1.8) \mathrm{mmol} / \mathrm{l} .64 \%$ had triglycerides greater than $2.0 \mathrm{mmol} / \mathrm{l}$, mean $2.75( \pm 0.49) \mathrm{mmol} / \mathrm{l}$. The high density lipoprotein for males, $11 \%$ had a level greater than less than $1.0 \mathrm{mmol} / \mathrm{l}$, mean 1.2( $\pm 0.2$ ) $\mathrm{mmol} / \mathrm{l}, 4 \%$ of females had a level less than $1.2 \mathrm{mmol} / \mathrm{l}$, mean $1.4( \pm 1) \mathrm{mmol} / \mathrm{l}$. The cardiovascular tools QRISK, ETHRISK CVD, Framingham CVD identified over 10\% of the cohort as high risk at the baseline appointment, and at the review appointment there was no change using QRISK. However, ETHRISK CVD and Framingham CVD demonstrated a risk reduction in the cohort. The tools varied in their selection of high risk, moderate risk and low risk. ETHRISK CHD and Framingham CHD and BNF identified $7 \%$ as high risk. Referral to specialist services was initiated with $14 \%$ referred for investigations, $21 \%$ commenced on medication or was altered. $12 \%$ of smokers were referred to a smoking cessation services. 25\% referred to weight management service. $32 \%$ were referred to Cardiologist or Lipidologist. 19\% referred to exercise on prescription.

Conclusions: The study identified risk factors in individuals who would not conventionally access the current National Health Service Health Checks programme and should therefore be seen as complementary to NHS Health Checks. 66\% were under the age of 40years who accessed the service. This population would not be able to access the systematic Health Checks programme provided by the National Health Service. This study therefore, illustrates the benefits of providing a tailored service for young individual's potentially high risk and susceptible to premature CVD. This service enabled first degree relatives to choose a healthier lifestyle to reduce their risk of cardiovascular event in the future.


## Declaration

This work is original and has not been previously submitted in support of Degree qualification or other courses.

Signed $\qquad$

Date $\qquad$

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## Chapter 1- Introduction

It is known that, cardiovascular disease (CVD) is still the leading cause of premature morbidity and mortality in the UK (British Heart Foundation, 2007), and therefore prevention is now becoming of significant importance. Primary prevention is highly cost effective and there is now a focus on primary prevention in the current National Health Service, with a multitude of recent strategic initiatives.

The Public Health White Paper, ‘Choosing Health’ (Department of Health, 2005), demonstrated a government commitment towards providing opportunity and support, which enabled individuals to lead healthier lives. This has been followed by recent initiatives such as the NHS Next Stage Review (2008), which identifies a need to change the emphasis of the NHS from being an illness management service to a health promoting and health preservation service. Linked to this vision is the recently launched NHS Health Checks Programme (Department of Health, 2008), which aims to offer access to cardiovascular risk assessment and a range of interventions, targeted to reduce cardiovascular risk at an individual level. None of these recent initiatives is new in terms of an aspiration for cardiovascular services.

Almost 10 years ago, the National Service Framework (NSF) for Coronary Heart Disease (Department of Health, 2000), in chapters 1 and 2, highlighted a need to deliver population and individual level cardiovascular prevention services. Whilst other chapters of the NSF have been delivered in an exemplary manner, with significant improvement in services across the spectrum of CVD, prevention has not
received the same magnitude of attention that downstream services have benefited from.

Embedded within the NSF was the recommendation that first degree relatives (FDRs) of patients with premature coronary heart disease (CHD) should be offered access to cardiovascular prevention. FDRs are defined as individuals such as the spouse, brothers, sisters and children. Studies, which will be discussed later, have demonstrated that this cohort of individuals is at a particularly high risk of CVD. Siblings have elevated risk due to the acquisition of risk factors from shared lifestyles and/or a genetic predisposition to premature heart disease. Offspring and partners of patients with premature disease also have increased risk for similar reasons (Chow, Pell, Walker, O'Dowd, Dominiczak \& Pell, 2007).

Studies such as the Heart Protection Study (Wilmshurst, 2002) and numerous other risk factor intervention studies, have shown how effective interventions, such as smoking cessation advice, dyslipidaemia therapy and physical activity, are in the primary prevention setting (these studies will be discussed in chapter 2). Taking into account that FDRs of patients with premature CHD (< 55 year old male, <65 year old females) which are considered a high risk population; one would predict a significant number of events could be avoided by targeting primary prevention at this group of individuals.

Since many FDRs are below the age of forty years, the recently launched Health Checks programme would not invite many of these individuals for cardiovascular risk assessment and therefore deny many of these high risk individuals access to
lifestyle risk management services and therapeutic interventions. Thus, there is a need to assess FDRs regardless of age. The rationale of this study is to initially assess FDRs over the age of eighteen and if they are identified as "at risk", then to provide intervention and support. As a result, this study would provide a primary prevention service for FDRs over the age of eighteen and would consequently complement the Health Checks programme and in addition deliver a key recommendation of the NSF.

However, there have been few examples of how systematic services for this group might be developed and delivered in a structured manner. Two sources of FDR exist; firstly, there are a considerable number of new cases of premature CHD diagnosed daily in England; therefore, there is an incident population of FDRs who should be offered access to primary prevention services. Secondly, there exists a prevalent population of FDRs, by virtue of CHD patients, who have had premature CHD diagnosed in the past and have been discharged from acute care to their general practitioner.

This study will report on the development and efficacy of services for the former group of FDRs who are identified from the acute sector. In addition, cardiovascular risk assessment will be quantified by the Framingham risk calculator and will be used to provide health promotion interventions to the FDR. It will go on to compare Framingham (Joint British Society 2, 2005) tools with other cardiovascular risk assessment tools, such as ETHRISK (Brindle, May, Gill, Cappuccio, D'Agostino, Fischbacher \& Ebrahim; 2006) and QRISK II (Hippisley-Cox, Coupland, Vinogradova, Robson, Minhas, Sheik \& Brindle, 2008).

In light of the key works as outlined above the objectives of this study are as follows:

1. To identify first degree relatives of patients with proven coronary heart disease and assess their cardiovascular risk
2. To compare different cardiovascular risk assessment tools
3. To assess the effectiveness of cardiovascular risk reduction services on the cardiovascular risk of the individual.

## Hypotheses

$\mathbf{H}_{0}$ Cardiovascular risk tools are not capable of predicting cardiovascular disease in FDRs of patients with premature CHD.
$H_{1}$ FDRs can be identified by proactive case finding
$\mathrm{H}_{2}$ Provision of lifestyle risk management services in FDR can lead to risk reduction and modify behaviour.

This study builds upon the existing literature that all FDRs should be detected early for the onset of CVD and the absolute risk calculated using cardiovascular algorithm and the risk communicated to the FDR. For the purpose of this study it is proposed that FDRs of patients admitted with CHD should be offered primary prevention service to assess CVD risk.

## Chapter 2 - Literature review

### 2.1 Definition of Cardiovascular Disease (CVD)

CVD is a general term used to describe disorders that can affect the heart and the body's system of arteries and veins. Atherosclerosis is defined as a gradual build of fatty deposits and debris in the arteries, which causes the narrowing of the arteries. The consequences of this may contribute to a myocardial infarction, angina or congestive heart failure. Therefore, risk factors for atherosclerosis need to be addressed to reduce the risk CVD in the general population.

CVD is the leading cause of premature death and a major cause of disability in the United Kingdom. It consumes a vast amount of resources in primary and secondary care, approximately £29.1 billion a year (Luengo-Fernandez, Leal, Gray, Petersen, \& Rayner, 2006). Comparison of mortality rates from CHD in England with those in countries such as Japan, France and Spain, suggest that 80\% of deaths in England could be avoided through improved treatment and active prevention (Kearney \& Chellaswamy, 2009., World Health Organization, 2005). According to the British Heart Foundation (2007), mortality rates from CHD have continued to decline steadily since the late 1960s. Unal, Critchley and Capewell (2004) suggest that the decline in mortality could be related to the improvements in population risk factors and in medical treatments for patients with coronary disease. These include secondary prevention strategies such as improvements in diet and the introduction of smoking cessation services, which have contributed substantially to the declines seen between 1981 and 2000. Furthermore, a $40 \%$ reduction in cardiovascular
deaths in people under the age of 75 years has been observed since the implementation of the NSF (Cardio \& Vascular Coalition 2008). Nevertheless, as indicated earlier, the NSF (Department of Health, 2000) has neglected to systematically deliver services which incorporate chapter 1 and chapter 2, which focus on primary prevention.

### 2.2 National policies and recommendations

Before a primary prevention service can be started, the target group for a service needs to be established. National strategies have evolved to identify individuals at the highest risk of a cardiac event and to provide cost-effective treatments and lifestyle advice to the individual so as to reduce the likelihood of an event (Kennedy 2008). In April 2008 the Department of Health announced plans to introduce a comprehensive vascular risk assessment and management programme, for all people aged forty to seventy four years old, based on recommendations by the National Screening Committee. It is estimated to cost around $£ 250$ million per year. (Department of Health, 2008). The key aims of the programme are as follows:

- To detect people at a high risk of CVD aged forty to seventy four years;
- Estimate cardiovascular risk using various algorithms;
- Enable the prevention of diabetes in many of those at increased risk of diabetes;
- To reduce premature mortality and to increase life expectancy;
- To reduce the overall population risk by reducing smoking rates, changing diets and increasing physical activity. In this way, identifying individuals at increased risk who could benefit from specific individual interventions;
- Reduction of inequalities arising from socio-economic, ethnic and gender inequalities (Khunti, Hiles and Davies 2008).

As stated, previously, the preliminary aim of the CVD programme is to identify people at high risk.

### 2.3 Calculating cardiovascular risk.

National guidelines for the prevention of CHD recommend the use of absolute risk profiles to guide decisions concerning treatment. This approach allows clinicians to target treatment at people who face the greatest risk of CVD (Joint British Society, 2005). Calculating cardiovascular risk enables clinicians to combine risk factor information and calculate the risk of a cardiovascular event within a specified time period. The National Institute of Clinical Excellence (NICE)(2008), advocates that high risk people should have their CHD/CVD risk estimated using a risk assessment tool. It has been suggested by Moon, Royston, Verguwe, Grobbee and Altmon (2009) that cardiovascular algorithms have a role in decision making and the future management of individuals.

There are numerous risk assessment algorithms available, which have been recommended to guide primary prevention strategies (Chauhan, 2007). The majority of cardiovascular algorithms use the Framingham equation. This equation was created using data from 5573 individuals who were followed up for 12 years. The traditional risk factors which are included in all of the algorithms include age, gender, blood pressure, cholesterol and smoking (Coleman, Stevens \& Holman 2007). The Framingham Heart Study was a public health study based in America. It began in 1948 and over 10,000 residents enrolled. The study's objective was to learn why people get CVD, how it evolves and results in death in the general population. The
study documented population characteristics such as blood pressure, diabetic status and smoking, together with the causes of death, over a period of 50 years. From these data, various risk factors for CHD, Stroke and total CVD have been defined and quantified (Dawber, Mealows \& Moore, 1951).

According to National Institute Clinical Excellence (2008), the current risk estimation is based upon the modified American Framingham equation which has limitations regarding the UK population. The Framingham equations can over / under estimate risk depending on the population being assessed. For example, the Framingham model does not take into account ethnicity and no allowances are made for family history of premature CHD. The CVD risk in south Asians is $40 \%$ higher than the general population (British Heart Foundation, 2007). NICE lipid modification (2008) recommended that the estimated CVD risk for men with south Asian background should be increased by 1.4. However, recently cardiovascular tools have been developed which take into consideration ethnicity. A recent report supports that the Framingham risk score may underestimates risk in women by $12 \%$ and overestimates that in men by $23 \%$, when compared to the QRISK score (HippisleyCox, Vinogradova, Robson, May \& Brindle, 2007).

### 2.4 Cardiovascular tools

QRISK: is a new cardiovascular disease risk calculator. It includes all the traditional risk factors, but also includes body mass index, family history of CVD, social deprivation and use of antihypertensive treatment ( Hippisley-Cox, Coupland, Vinogradora, Robson, Minhas, Sheik \& Brindle, 2008)

ETHRISK: A study that was conducted to recalibrate on the existing Framingham risk score to produce a web based tool for estimating ten year risk of CHD and CVD in black and ethnic minority groups. It consisted of 8,000 males and females, aged 35-54. The study concluded that in the absence of current cohort studies in the UK that includes significant numbers of black \& ethnic minority groups; this algorithm provides a pragmatic solution to include people from ethnic backgrounds (Brindle et al. 2006). Although this tool is not ideal it would have partially prevented inequalities in CVD prevention that are not easily overcome by subjective judgement (Cappuccio, 2008).

The Joint British Societies guidelines (2005) recommend risk stratification based on the probability of CVD, as opposed to coronary heart disease or other outcomes. The charts published in the British National Formulary are based on those given in the Joint British Society guidelines (2005)

### 2.5 Communicating cardiovascular risk.

This study looks at FDRs who are at risk, where the term 'risk' is defined as a situation or an event where something of human value is at stake and where the out come is uncertain (Rosa, 2003). Absolute risk refers to the risk of developing the disease over a period of time. CVD risk can be estimated for over a ten year period. Risk can be expressed in either percentage or pictorial format (Goodyear-Smith, Arroll, Chan, Jackson, Wells, \& Kenealy, 2008; Lindsay \& Gaw, 1997). The rationale for estimating CVD risk is based on the major risk factors because:
i) CVD is multi-factorial in origin;
ii) Risk factors tend to cluster;
iii) Co-existent risk factors tend to have multiplicative effect on CVD risk (JBS2);

According to O'Conner, Fiest, Rosta, Tetroe, Enwhistle, Llewellyn-Thomas Entwistle, Rostom, Fiset, Barry and Jones (2002) an 'open two-way' exchange of information and opinion about risk, leads to a better understanding about clinical decisions. This is supported by Edwards, Elwyn and Mulley (2002) who advocate that this definition moves away from the philosophy that information is communicated only from clinician to the individual and that the individual accepts the information without questioning the clinician further. The two-way exchange of information and opinion is important if decisions about treatment are to reflect the attitudes to risk of the people who will live with outcomes. Therefore, this is not a unilateral decision but a shared decision between patient and clinician (Goodyear-Smith et al. 2008; NICE lipid modification, 2008).

The NICE lipid modification (2008) guidelines support good communication skills between the clinician and individual. Recommendations of treatment / intervention should take into account the patient's needs and preferences. People at high risk of CVD or with established CVD should have the opportunity to make informed decisions about their care and treatment, in partnership with their healthcare professionals. Clinicians should always be aware that all CVD risk estimation tools can provide only an approximation of CVD risk. Interpretation of CVD risk scores should always reflect informed clinical judgement (Willots, 2009).

### 2.6 Risk Factors

The treatment and prevention of CVD focuses on reducing the development and destabilisation of coronary plaques. Modifiable risks such as smoking, physical inactivity, hypertension, diet, high plasma glucose level and the non-modifiable risk factors of age, gender and family history of premature CHD are epidemiologically strongly associated with CHD (Glick, 2002). Recent epidemiological studies such as EUROSPIRE II (2001), the Heart Protection Study (Wilmshurst, 2002) and EUROACTION (Wood, Kotseva, Connolly, Jennings, Mead, Holden, De Bacquer \& De Backer,2004) constantly report under utilization of evidence-based preventive treatment and inadequate management of risk factors in the overall population at risk of CVD. There is substantial evidence that indicates risk factor modification may reduce the possibility of developing CVD in the first place and improves outcomes in those who already had CVD (Joint British Society 2, 2005, Wang \& Widlansky, 2009).

In recent years, the development of CVD guidelines has acknowledged the multifactorial nature of CVD. Often, a treatment focuses on a single risk factor that might lower risk by up to $20 \%-30 \%$, but such a strategy leads to the "missing" of those patients who are at long-term risk of disease and consequently to chronic suboptimal management of the disease (Emberson, Whincup, Morris, Walker \& Ebrahim 2004). Multifactorial risk reduction has resulted in a positive shift towards managing a patients total cardiovascular risk, as demonstrated by the incorporation of tools for calculating cardiovascular risk into recent treatment guidelines (National Cholesterol Education Programme, 2001., De Backer, Ambrosioni, Borch-Johnsen, Brotons,

Cifkova, Dallongeville, Ebrahim, Faergeman, Graham, Mancia, Cats, Orth, Perk, Pyorala, Rodicio, Sans, Sansoy, Sechtem, Silber, Thomsen \& Wood, 2003,. Joint British Society 2, 2005)

### 2.7 Non-modifiable risk factors

### 2.7.1 Age and Gender

Although the majority of the emphasis is placed on modifiable risk factors, it is imperative that non-modifiable risk factors are not neglected. Age plays an important role in the risk equation. An increased risk of CVD complications and the prevalence of other risk factors regardless of gender are associated with increasing age. Glick (2002) suggested that the majority of new CHD events occur in both men and women after the age of 65 years. Therefore, the majority of all deaths which are due to CHD are recorded among people 65 years of age and over. However, the Framingham Heart Study calculates a lifetime risk of developing CHD. In a cohort of 7,733 study participants, the lifetime risk was estimated to be greater in men aged 40 years compared to women of the same age.

NICE (2008) the lipid modification report recommends that all individuals aged 75 or older should be considered at increased risk of CVD, particularly those who have other risk factors, such as hypertension. Assessment and treatment of these patients should be guided by the benefits and risks of treatment, informed preference and comorbidities.

A CHD event in the first two decades of life is rare, becoming more prevalent after the age of 30 and more evident in males than females below the age of 60 years.

Beyond the age of 60 years, CHD rates in females increase and the rate approaches that of males. The population data on CHD mortality indicates that CHD risk in females' increases with age. It has been argued that this is due to the natural menopause occurring at different ages in individuals. Studies have demonstrated that women are relatively protected against CHD whilst in premenopausal years (Colditz, Willett, Stampher, Rosner, Speizer \& Hennekens, 1987). The majority of observational epidemiological studies that examined the role of hormone replacement therapy in women without established heart disease have consistently demonstrated a lower incidence of cardiac events among users versus non-users. A recent meta-analysis showed an approximate 35\% reduction in CHD events among users of hormone replacement therapy (Mosca, Collins, Herrington, Mendelsohn, Pasternak, Robertson, Smith \& Wenger, 2001) (Grodstein, Stampfer, Colditz,,Willett, Manson, Joffe, Rosner, Fuchs, Hankinson, Hunter, Hennekens \& Speizer 1997)

### 2.7.2 Family History

Family history of premature disease contributes to an increased risk of CVD and risk factors in FDRs (Murabito, Pencina, Nam, D'Agostino, Wang, Lloyd-Jones \& O'Donnell 2005). Clinicians use self-reported information about family history to calculate the risk of CVD in their patients. The information available to the clinician, concerning family history is limited, due to the fact that patients themselves have inadequate information about their relative's medical history. Identifying a person with a positive family history is essential to reduce incidence of CVD.

Reporting of family history of premature CVD has been included in the National Screening guidelines (Department of Health, 2008). Consequently this highlights an
area where health professionals need to educate their patients on the presence and/or relevance of family history to a disease. There is a need to clarify within families the associated risk to an individual and in turn enhance primary prevention services (Murabito, Nam, D'Agostinao, Lloyd, O'Donnell, \& Wilson, 2004).

Premature death from CAD clusters in families, Eaton Bostom, Yanek, Laurino, McQuade, Hume and Selhub (1996) suggested that the risk of developing premature CHD was increased more than threefold when any FDR was affected, and almost by six fold when the FDR developed CHD before the age of 45 years or when at least two first degree relative had history of CHD. Therefore, primary prevention guidelines urge screening of FDR for the presence of cardiovascular risk factors. A study conducted by Becker, Yook, Moy, Blumenthal and Becker (1998) identified that among siblings of patients with premature CHD, $80 \%$ of black men and women and $70 \%$ of white men and women had treatable risk factors.

A recent study by Murabito et al. (2005) suggested that it still remains debateable whether family history alone is an independent risk factor and for whether the issue is a genetic rather than family behaviour. A positive family history seems attributable to the familial aggregation of genes or to shared environment; diet, living conditions, lifestyle habits, and occupation (Mortia \& Nagai, 2005).

Nevertheless, a case-control study conducted in Italy over a period of four years examined those people with premature CHD which was defined as documented myocardial infarction with a significant occlusion greater than $50 \%$ in a major coronary artery in people younger than 60 years of age. The number of acute myocardial infarctions according to family history was estimated using unconditional logistic regression, adjusting for other acute myocardial infarction risk factors and
family size. The results of the study indicated that by having one or more FDRs the odds ratio was doubled and for those with two or more FDRs the odds ratio was tripled. This was also similar to those with an affected parent or sibling, sex, age at diagnosis of the person or the relative. The study concluded that family history of ischemic heart disease is an independent risk factor for CHD in people younger than 60 years of age and indicated significant correlation between early onset of CHD and family history. As a result, it could be suggested that management and intervention of modifiable risk factors may be beneficial for those with family history at a younger age (Bertuzzi, Negri, Alessandra Tavani \& La Vecchia, 2003).

### 2.7.3 Ethnicity

South Asians (SA) originate from the Indian sub continent and represent one fifth of the world's population. It is widely accepted that SA have a $40-60 \%$ increased risk of developing CHD when compared with other populations (Bhopal, Fischbacher, Vartianinen, Unwin, White, \& Alberti 2005). Epidemiological studies showed that the migrant population of SA descent have a higher risk of ischaemic heart disease than the general population. They have the highest risk of developing CVD at a younger age in comparison to other ethnic groups (Ramaraj \& Chellappa 2008). The World Health Organisation, warned countries about the rising burden of heart disease among SA. The greatest global increase in diabetes is projected in India by 2025. In rural settings, the prevalence of diabetes is higher in the urban communities. In the UK, the prevalence of diabetes in SA is around $20 \%$, studies by Mather, Chaturvedi and Fuller (1998), Chaturvedi and Fuller (1996) showed that South Asian immigrants with diabetes have a three to four fold higher mortality rate due to CVD than people from other ethnic groups.

A systematic review (Kurian \& Cardarelli 2007) suggested that CVD is the leading cause of death in United States. To expand current understanding of the factors associated with ethnic disparities in CVD risk factors, sixteen studies carried out in America were reviewed, with the majority finding that diabetes was significantly higher within the minority groups. Also, hypertension was significantly higher in blacks than whites. No population group was found to be consistently of a higher or lower prevalence of obesity or hypercholesterolemia. Mexican Americans had a lower prevalence of smoking than whites and blacks. The American Indians had a significantly higher prevalence of smoking compared to whites. Regarding physical activity Mexican Americans had the highest prevalence of no leisure time. This review demonstrates that better understanding and awareness of the disparities of CVD risk factors by ethnicity may aid public health professionals and clinicians to develop culturally sensitive interventions, prevention programmes and services specifically targeted to address risk. The Euroaction (2008) study has demonstrated that standards of preventive cardiology can be improved. To achieve the results demonstrated in the Euroaction study a similar model needs to be appropriately adapted taking into consideration the medical, cultural and economic settings of the country.

Joint British Society guidelines (2005) have cautioned against the generalisation of risk predication methods as they may not be valid for SA. Ramaraj et al. (2008) conducted a review to discuss the existing data on the prevalence of CVD and its risk factors in South Asians and found that there are no published randomised controlled studies supplying robust information on risk factors outcomes in South Asians or to test the validity of cardiovascular risk predication

### 2.8 Modifiable Risk Factors

### 2.8.1 Smoking

Cigarette smoking has been established as a risk factor for CHD independent of any other risk factors. The prevalence of smoking has declined since the 1960s, but it still remains high in some groups. These groups include the under thirties, some ethnic minority groups and lower socioeconomic groups. Dawber (1980) suggested that there is a two to three fold increase of CHD risk in the whole population which is associated with cigarette smoking. The risk rises with number of cigarettes smoked per day. In the 1990s, smoking accounted for $28 \%$ of male and $26 \%$ of female all cause vascular death aged 35 to 69 (Peto , Lopez, Boreham, Thun \& Heath 1994).

To achieve the greatest population health benefit, in the United Kingdom the guidance has emphasised two forms of smoking cessation:

- Opportunistic, brief advice to encourage all smokers to quit and to direct them to effective treatment that can help.
- Treatment services for those who would like or need help to stop (West, 2005).

The Department of Health (2005), has implemented a comprehensive strategy to reduce tobacco use in the UK, leading to the development of NHS Stop Smoking Services which provided psychological support and medication for the individual. Cigarette smoking has been established as a risk factor for CHD independent of any other risk factor. Smoking cessation has shown to reduce CVD mortality in individuals with established CVD and those without.

A prospective study conducted for 40 years (Clarke, Emberson, Fletcher, Breeze, Marmot \& Shipley, 2009) assessed the life expectancy in relation to cardiovascular risk factors. The study recruited 18,000 men from the civil service aged 40 to 49 years. The initial assessment included a medical examination and a questionnaire. The participant completed a questionnaire regarding medical history demographical information, social situation, smoking habits, body mass index and cholesterol. All surviving participants were contacted. A questionnaire was sent regarding medical history and they were asked to visit their local surgery to ascertain information on blood pressure, cholesterol and smoking habits. The findings suggest that the presence of smoking, cholesterol and high blood pressure predicated a three fold higher rate of non-vascular mortality and a ten year shorter life expectancy compared to those with none of the risk factors.

This is supported by a further study, which compared the risk of smoking in men who formed their habits at different periods and the reduction in risk when smoking is stopped at different ages. British doctors participated in the study in 1951 and information concerning smoking status ascertained for 50 years. The findings demonstrated men born around 1920, who continued to smoking from early adulthood tripled their age-specific mortality rates but, cessation at the age of 50 halved the risk, and cessation at the age of 30 avoided almost all of it (Doll, Reto, Boreham \& Sutherland, 2004).

### 2.8.2 Physical Activity

Physical inactivity predisposes an individual to numerous health problems including: obesity, increased insulin resistance and metabolic risk factors (Fletcher, Balady \& Blair, 1996). Epidemiological studies have shown that a sedentary lifestyle will contribute to early onset and progression of CAD and is associated with doubling the risk of premature death (Paffenbarger, Hyde, Wing, Lee, Jung \& Kampert, 1993). Current public health statements recommend physical activity of 30 minutes of moderate-intensity activity each day. This provides substantial benefits across a broad range of health outcomes for sedentary adults. Individuals performing 30 minutes of moderate-intensity exercise per day are likely to achieve additional health benefits if they exercise more. In addition to aerobic exercise, people should engage in resistance training and flexibility exercises at least twice a week, which will promote the maintenance of lean body mass and improvements in muscular strength and endurance, all of which enable long-term participation in regular physical activity and promote quality of life (Blair, Lamont \& Nichaman, 2004). People should be advised to choose enjoyable activities which fit into their daily routine, preferably 30 to 45 minutes, 4 to 5 times weekly at $60-70 \%$ of the average maximum heart rate (De Backer et al. 2003).

Physically inactive middle aged women experienced a $52 \%$ increase all cause mortality and a doubling of cardiovascular related mortality (Hu, Willett \& Li, 2004). Furthermore, it appears that people who are fit and have other risk factors for CVD may be at a lower risk of premature death than people who are inactive with no other risk factors for CVD (Wessel, Arant, Olson, Johnson, Reis, Sharaf, Shaw, Handberg, Sopko, Kelsey, Pepine \& Merz 2004).

An increase in physical fitness will reduce the risk of premature death and a decrease in physical fitness will increase the risk. The amount of exercise increased or decreased appears to have a graded impact on risk. A study conducted by Eriksson (2001), demonstrated that participants with the highest levels of physical fitness at baseline, who maintained or improved their physical fitness over a prolonged period of time had the lowest risk of premature disease. However, modest enhancement in physical fitness, in previously sedentary people has been associated with a significant improvement in health status. A study by Myers, Prakash, Froelicher, Do, Partington \& Atwood (2002) observed that every 1-MET (metabolic equivalent task) increase in treadmill performance was associated with a 12 percent improvement in survival, therefore suggesting that health professionals should incorporate promoting physical activity into their practice strategies, in addition to the routine treatment of other modifiable risk factors.

Warburton, Nicol and Bredin (2006) conducted a review of observational studies that have been carried out to evaluate the current literature and to provide further insight into the role of inactivity in the development of disease and premature death. The review concluded that there is a linear relationship between physical activity and health status. Physical fitness has been associated with cardiovascular mortality and even small improvements in fitness may lower mortality (Vanhess, Lefevre, Philippaerts, Martens, Huygens, Troosters \& Beunen, 2005).

### 2.8.3 Dyslipidaemia

Dyslipidaemia means that abnormal levels of lipids are in the body. These lipids include cholesterol and triglycerides. There are three main lipids of lipoproteins in the body: low density lipoproteins (LDL), high density lipoprotein (HDL) and
triglycerides. These are essential for our bodies to function, but elevated lipids can contribute to CVD/CHD. This can be caused by genetic, environmental and lifestyle factors.

Elevated LDL contributes to atherosclerosis over a period. On the other hand, having elevated HDL protects the heart by absorbing the build up of LDL from the arteries. Low levels of HDL and triglycerides can contribute to atherosclerosis and CHD, especially in people who are obese or have diabetes. According to the National Cholesterol Education Programme Guidelines (2001), healthy adults should be screened once every five years starting at age twenty. Those with a family history of hyperlipidaemia or other risk factors may need earlier or more frequent screening.

Raised cholesterol has been rated by the World Health Organisation (2002) as the leading cause of mortality world wide as it leads to an increased risk of CVD. Management of lipids with statins can provide a relative reduction in CVD of around 30\%. However, Kosteva, Wood, Backer De Bacquer, Pyorala and Keil (2009) argue that many patients do not achieve the desirable target on statins alone; lifestyle management is an essential element and should not be ignored

West of Scotland Coronary Prevention Group (WOSCOP) was a randomized clinical trial comparing Pravastatin with a placebo, in men aged 45-64 with hypercholesterolemia and no history of a cardiac event. The participants were followed up for 5 years and the analysis concluded treatment with Pravastatin was associated with significant reduction in coronary events. Statin treatment for an
average of 5 years provided an ongoing reduction in risk for up to 10 years (Ford, Murray, Packard, Shepherd, Macfarlene, \& Cobbe .2007)

It has been estimated that every $0.03 \mathrm{mmol} / /$ increase in HDL reduces the risk of cardiac event by $2 \%$ in men and $3 \%$ in women (Gordon, Probstfield, Garrison et al. 1989)

The INTERHEART study (Yusuf, Hawken, Ounpuu et al. 2004) conducted in 52 countries investigated the association of risk factors regarding coronary heart disease. 15,152 cases and 14,820 controls were enrolled. The findings suggested that the risk factors discussed in this paper, plus diabetes and psychosocial factors account for most of the risk of myocardial infarction worldwide in both sexes and in all ages. The study recommended that approaches to prevention can be based on similar principles worldwide and have the potential to avert most premature cases of CHD.

### 2.8.4 Obesity

Overweight and obesity are defined as abnormal or excessive fat accumulation that may lead to serious health consequences. According to the World Health Organisation (2006), there are about 400 million overweight adults worldwide. The fundamental cause of obesity and overweight is an imbalance between calorie consumption and expenditure. The Global increase in the prevalence of obesity is attributable to a number of factors including increased sedentary lifestyle, modes of transportation and increasing urbanization which all lead to decreased physical activity.

The body mass index (BMI) is a simple tool used to distinguish between overweight and obesity in the adult population. It is defined as the weight in kilograms divided by the square of the height in metres $\left(\mathrm{kg} / \mathrm{m}^{2}\right)$. The risk of CVD increases progressively as body mass index increases. Raised body mass index is a major risk factor for chronic disease including CVD, which is already the world number one cause of death, killing 17 million people each year (World Health Organisation, 2006). A meta-analysis conducted using data from the prospective studies in the South Asian region which integrated 33 cohort studies, including over 300,000 participants. It concluded, that was a relationship between high body mass index and CVD. Therefore, decreasing an individual's BMI would reduce the risk of CVD event and every unit of sustained decrease in BMI, HDL increases by $0.02 \mathrm{mmol} / \mathrm{l}$ (Mhurchu, Rodgers, Pan, Gu \& woodward, 2004., Berns, de Vries \& Kata, 1998)

Conventional body mass index classifications are: overweight $\left(25.0 \mathrm{~kg} / \mathrm{m}^{2}\right.$ to $<30.0 \mathrm{~kg} / \mathrm{m}^{2}$ ) and obese ( $\geq 30.0 \mathrm{~kg} / \mathrm{m}^{2}$ ). These cut off points were derived primarily from the European population to represent the thresholds for the risk of CVD. Due to the differential body mass index thresholds relating to ethnicity, the South Asian population has had thresholds defined separately to the rest of the population, using thresholds of BMI for obesity of $25 \mathrm{~kg} / \mathrm{m}^{2}$ for South Asians. The BMI of $23-25 \mathrm{~kg} / \mathrm{m}^{2}$ defines overweight for the South Asian population (WHO, 2004). The NICE guidelines of obesity (2006) dispute this and suggest there is limited evidence that for a given BMI morbidity risk in South Asians is higher than the general population. However, it recommends that the parameters which have been suggested by WHO should generate action for public health.

The OXCHECK study was a randomised control trial to assess the effectiveness of health checks by nurses in reducing risk factors for cardiovascular disease in patients from the general practices. General health checks by nurses are ineffective in helping smokers to stop smoking, but they help patients to modify their diet and total cholesterol concentration. The public health importance of this dietary change depends on whether it is sustained (Muir, Mant, Jones \& Yudkin,1994). Even though in this OXCHECK study nurses were ineffective in smoking cessation, the individual had access to a service which enabled the individual to make a lifestyle choice.

### 2.8.5 Blood Pressure and Hypertension

The outcomes of clinical trials have determined the definition of hypertension. Hypertension is classified as the level of blood pressure (BP) where evidence implies that a reduction of the parameters benefits the individual (Joint British Society 2, 2005). The CVD risk associated with blood pressure is determined by both diastolic and systolic blood pressure and the presence of other risk factors. Epidemiological studies demonstrate a close relationship between blood pressure and risk of CVD and CHD. A meta-analysis suggests that the association between systolic and diastolic blood pressure and CVD is more apparent than previously considered (Mancia, De Backer, Dominiczak, Cifkova et al. 1990).

As blood pressure is an independent risk factor, a reduction in BP by $6-12 \mathrm{mmHg}$ can be estimated to reduce risk of CVD by $40 \%$ CHD by $20 \%$ (Prospective Studies Collaboration, 2002). According to the British Hypertension Society (2004) classification of systolic blood pressure of $\geq 140-159 \mathrm{mmHg}$ and/or diastolic blood pressure $\geq 90 \mathrm{mmHg}$ is considered to be Grade 1 hypertension (mild). Lifestyle
modification such as, weight reduction, moderation of alcohol, salt intake and physical activity has been shown to reduce blood pressure (Berchthold et al 1982, Macmohan \& Norton 1986). Long term pharmacological trials have demonstrated the benefits of anti hypertensive therapy in reducing the risk of stroke. Therefore, it is imperative that lifestyle modification and intervention for example pharmacological therapies are initiated earlier.

### 2.9 Summary

To summarise, damage to the vascular system increases with age and progresses faster in men than women, especially in those with a family history of vascular disease and of certain ethnic groups. The rate of vascular damage progression is determined by modifiable factors. Changing these can reduce the probability of premature disease. These risk factors are, smoking, physical inactivity, raised blood pressure, raised blood cholesterol and obesity which have already been discussed at some length.

Several published guidelines have recommended screening of FDRs. This would involve an assessment of an individuals' risk over ten years (Pearson, Blair, Daniels, Eckel, Fair, Fortmann, Franklin, Goldstein, Greenland, Grundy et al. 2002, Chow, Pell, Walker, O'Dowd, Dominiczak \& Pell 2007). Studies such as the Heart Protection study (Wilmshurst 2002) and numerous other risk factor invention studies have shown how effective interventions such as smoking cessation advice, dyslipidaemia therapy etc. are in the primary prevention setting. Coupled with the fact that the FDRs of patients with premature CVD are a high risk population, it is
anticipated that a significant number of events can be avoided by targeted primary prevention of this group of individuals.

EUROASPIRE I, II, and III (Kotseva, Wood, De Backer, De Bacquer, Pyorala, \& Keil (2009) are cross-sectional studies and included the same selected geographical areas and hospitals in eight European countries. Patients of both sexes under the age of seventy were identified after revascularisation or after a hospital admission because of a cardiac event and were interviewed six months later. Over 8,000 patients were interviewed in the three studies. The results indicated that the proportions of patients who smoke have remained virtually the same, but there has been an increase in women smokers aged less than 50 years. The occurrence of obesity over the three studies has increased by $13 \%$. The percentage of patients with hypertension was comparable, whereas the proportion with raised total cholesterol ( $\geq 4.5 \mathrm{mmol} / \mathrm{L}$ ) decreased by $48 \%$.

The EUROACTION (2004) study followed up all patients with heart disease, high risk individuals, their partner and FDRs of patients with premature heart disease. The aim of the study was to raise the standards of preventive cardiology in Europe by demonstrating that national lifestyles, risk factors and therapeutic goals in CVD prevention are achievable and sustainable in everyday clinical practice (Wood, Kotseva, Connolly, Jennings et al. 2004). The EUROACTION preventive cardiology programme reduced the risk of CVD, through lifestyle changes by families who together made healthier food choices and became more physically active than before the intervention. These changes consequently led to some central obesity weight reduction in high risk patients. In a systematic review of ten trials with
outcome data, no significant effect on total or coronary mortality was evident (Ebrahim, Beswick, Burke \& Davey Smith 2006). However, EUROACTION was more effective than usual care because lifestyle intervention was combined with cardio-protective drugs that together reduced the CVD event.

Neil, Perera, Armitage, Farmer, Mant and Durrington (2008), describe four steps which are involved in disease prevention, these include:

1. Collection of clinical data: ethnic and socioeconomic determinants of health need to be accounted for as part of the cardiovascular risk assessment;
2. The measurement of risk factors;
3. Interpretation of risk related data;
4. Intervention to minimise risk disease risk and prevent risk factor development.

The clinician is best positioned to interpret and integrate the collected data and therefore needs to be familiar with all its limitations (Vuljoen 2008).

## Chapter 3 Methods

Primary prevention refers to interventions designed to educate and modify risk factors in individuals who have not yet been diagnosed with CHD. The risk of developing heart disease can be minimised by using two broad strategies, firstly to try to reduce the prevalence of heart disease within the entire population. The second important element of primary prevention is identifying and supporting those individuals at high cardiovascular risk. In most parts of the United Kingdom current services in both primary and secondary care neglect targeted risk assessment and primary prevention of CVD in FDRs of patients with proven premature CHD (Patel, Minhas, Lincoln \& Dhillon 2008).

In line with the Department of Health (2000), primary prevention of CHD should be targeted at people with high overall risk and FDR's of patients with premature CHD who are at higher risk than the general population. Premature CHD was defined as below the age of 55 years in men and 60 years in women so relatives of patients with premature onset are at a greater risk than those of patients with late onset of disease.

### 3.1 Ethical Approval

Ethical approval to conduct this study was granted on the $4^{\text {th }}$ August 2008 from the Ethical Committee (appendix 1) and the Research and Development department at Sandwell Hospital (appendix 2). Verbal consent was gained from the index case during their admission to contact their FDRs (as part of standard NHS care) and FDRs gave written consent at their first hospital visit (appendix 3).

## Participants

Total of 43 Participants, both female and male aged from $18-74$ years old were involved in the study. The study was carried out at the Outpatient Department at Sandwell Hospital, which consisted of one cohort of FDRs. The sample population included all FDRs seen over a period of 6 months on two occasions in the primary prevention service.

### 3.2 Inclusion and exclusion criteria

## Inclusion criteria

- FDR of patients with proven coronary heart disease.
- Index cases were men under the age 60 and women under the age of 65 .


## Exclusion Criteria

- Individuals with dementia
- Under the age of18
- Over the age of 75
- Individual declining participation in the primary prevention service


### 3.3 Procedure

For the purposes of this study, premature CHD was defined as below the age of 60 years in males and below the age of 65 years in females. Index cases were deemed to have established and proven CHD by virtue of a diagnosis of acute coronary syndrome (including myocardial infarction) and /or requiring coronary angioplasty or bypass surgery on acute admission to hospital. These index cases were identified on a daily basis by the cardiac rehabilitation nursing staff at an acute trust in Sandwell
and West Birmingham. During structured delivery of phase 1 of cardiac rehabilitation, Index cases were invited to participate in the study following direct discussion and informed consent by a specialist nurse. Index cases were therefore consented to analysis of their own CV risk factors and also agreed to invite their FDR to a primary prevention service established to support this cohort of FDR. Therefore, direct contact with FDR was not made. Access to primary prevention was offered either in the acute hospital clinic or in primary care with their own GP or a nurse led community service. Only individuals accessing the secondary care service were assessed in this study.

Index Cases underwent conventional cardiovascular risk assessment as part of Phase 1 of cardiac rehabilitation which included documentation of and where necessary interventions in terms of both behavioural advice, access to lifestyle services or therapeutic interventions for smoking status, obesity, dyslipidaemia, physical (in)activity, hypertension and diabetic status. During phase 1 assessment, a detailed family history would be obtained to identify the number of FDR at risk of CVD. Index cases were then provided invitation letters (appendix 4) and information sheets (appendix 5) to cascade to their FDRs.

The FDR would then contact the cardiac rehabilitation team administrator and a member of the cardiac rehabilitation team nursing staff provided an explanation of the service offered. The FDR was able to access primary prevention in either primary or secondary care. If acceptable, an appointment was made and a request form to have blood tests taken for full blood count, full lipid profile, renal and liver biochemistry and blood glucose. This enabled results to be available at the time of clinic visit.

The FDR, at appointment underwent cardiovascular risk assessment and received advice and interventions as necessary from the researcher. Therapeutic interventions were guided by guidelines from the National Institute for Health and Clinical Excellence (NICE, 2008) following cardiovascular risk assessment using the modified Framingham equation also advocated by NICE (2008). If a FDR declined initial referral to lifestyle risk management services for example smoking cessation, they were given contact numbers to access the service at a later date if required.

Prior to the consultation the health care assistant would record the height and weight of the FDRs this was recorded in their medical notes. At the first consultation the subject was

- Given verbal information about the consultation.
- Given written information and consent form regarding the study.
- Informed that they may withdraw from the study at anytime and this would not impact on any future treatment.
- During consultation the FDR was given information regarding CVD risk in line with NICE guidelines lipid modification (2008)(Glick,2000)


### 3.4 Smoking Status

The smoking status of FDR was ascertained, European guidelines (2003) emphasise that smoking status must be addressed and documented by health professionals. (Manso, Tosteson, Ricler et al 1992)

- Advice and written literature given regarding the benefits of stopping
- Offer nicotine replacement patches
- Referred to smoking cessation service.

The above intervention is in line with the NICE guidelines for Smoking Cessation (2007).

### 3.5 Body mass index

Lending from the literature, the parameters for body mass index were set according to the World Health Organisation (2004) and NICE guidelines for obesity (2006). The weight was recorded using the Avery E101 serial number 00002047 scales. Height measured using Seca wall mounted metre. The intervention below is based on recommendation by Department of Health, (2000) and the Health Checks Programme (2008):

- Ascertain current eating habits and offer written literature;
- Refer to dietician or weight management team for further support;
- Initiate medical therapy if $\mathrm{BMI}>40 \mathrm{Kg} / \mathrm{m}^{2}$;
- Refer to GP to initiate medical therapy.


### 3.6 Blood cholesterol

A blood Sample was taken prior to clinic appointment. These would be analysed within the Pathology Department at the hospital to ascertain cholesterol profile renal and liver biochemistry. Depending upon the lipid profile and calculated CVD risk, the intervention was either:

- Written information to improve dietary intake;
- Medical therapy was initiated and a written prescription was given to the individual if clinically indicated.

The above intervention is supported by the NICE guidelines for Lipid modification (2008).

### 3.7 Blood pressure and hypertension

Blood pressure was measured according to the guidelines recommended by Mancia, De Backer, Dominiczak, Cifkova, Fagard, Germano et al. (2007). The FDRs were informed of their blood pressure reading. Blood pressure was measured using an OMRON electronic blood pressure monitor which meets the British Hypertension Society (2004).

- According to the BHS (2004) recommendation standard and large adult cuff sizes were used;
- The blood pressure was repeated if the diastolic was 90 mmHg or more, or if the systolic pressure was 160 mmHg or more and the lowest of the two readings were recorded;
- Medical therapy would be initiated by the Consultant if indicated prior to discharge from clinic;
- If blood pressure was above the recommended target they were advised to be reviewed by the General Practitioner or Practice Nurse.

If therapeutic intervention was required the guiding recommendations stipulated by NICE (2006) were adhered to.

### 3.8 Cardiovascular calculators

A number of models for coronary heart disease and cardiovascular risk calculators have been developed and for the purposes of this research the following calculators were used:-

1. QRISK II, - this was developed from routinely collected data from the general practice clinical computer systems. The algorithm uses existing risk factors, smoking status, lipid profile, age, blood pressure but also incorporates deprivation and ethnicity (Hippisley-Cox, Coupland, Vinogradova, Robson, Minhas, Sheik \& Brindle, 2008).
2. FRAMINGHAM - Calculates the absolute risk of coronary heart disease event for patients with no previous history of coronary heart disease, stroke and peripheral vascular disease (Sheridan, Pignone \& Mulrow 2003).
3. ETHRISK - Ethnic groups within Britain have differing risks of coronary heart disease and CVD at the same level of risk factors. This calculator adjusts for ethnicity and is based on a re-calibration of the Framingham risk equations. It also provides the standard Framingham estimates. The method uses the prevalence ratios for coronary heart disease and CVD for each ethnic group compared to the general population, and adjusts for differences in mean risk factor levels and prevalence of smoking between each ethnic group (Brindle, May, Gill, Cappuccio, D'Agostino, Fischbacher \& Ebrahim 2006).
4. Modified Framingham - this method involves prior estimate of cardiovascular risk. This is derived from risk factors data that are routinely held in electronic medical records with unknown blood pressure and /or cholesterol levels replaced by default values derived from the national survey data (Marshall, 2008)

### 3.9 Method of data collection

Data was collected on two occasions, on their first appointment then six months later at their review appointment. All the data was entered onto an Excel spreadsheet which was password protected. The FDR identified goals which needed to be achieved prior to their next appointment (appendix 6). The FDR was then reviewed in clinic six months later to assess the impact of the service and discuss any concerns they may have had (NICE, 2008).

### 3.10 Method of data analysis

Appropriate descriptive statistics were calculated for each variable following a check of normality using the Shapiro-Wilk statistic (Coakes \& Steed, 2004). The mean and standard deviation for each variable was calculated for each variable. All data was analysed using SPSS (version 16.0) and the significance level was set at 0.05 .

The assumption of normal distribution homogeneity was violated for each variable. The data was ordinal consequently a non-parametric approach. The Wilcoxan signed rank test was used to analyse the data because the participants were seen on two occasions and the same tests were repeated. The mean and standard deviation for each variable was calculated.

## Chapter 4: Results

A total of 463 letters were given to 201 patients who accessed the Coronary care unit. 113 (24\%) FDR accessed the service since September 2008 - June 2009. Only $43(44 \%)$ were eligible, $57 \%$ were not eligible because they failed to attend the clinic on two occasions or they were still waiting for their review to take place.

### 4.1 Basic Demographic data

The following results are based on FDRs who attended both first and review appointments. The information was gathered at their first appointment at the Primary Prevention Service.
4.1. Illustrates demography of the FDRs

|  |  |
| :--- | ---: |
| List of Variables | Value |
| Mean Age (years) | 42 |
| Males (\%) | 44 |
| Females (\%) | 56 |
| South Asian (\%) | 63 |
| Non-South Asian (\%) | 37 |

Table 4.1: Demography of subjects

The table shows the mean age of the FDRs was 42 years. $44 \%$ (19) were male and 56\%(24) were female. $63 \%(27)$ were south Asians and $37 \%$ (16) were non south Asians

### 4.2 Summary of results

### 4.2.1 Dependent and independent variables

| DEPENDENT VARIABLES | BASELINE | REVIEW | PERCENTAGE CHANGE | $P$ <br> VALUE |
| :---: | :---: | :---: | :---: | :---: |
| Systolic Blood Pressure ( mmHg ) | 140( $\pm 14.8$ ) | $137( \pm 4.94)$ | -2 | 0.56 |
| Diastolic Blood Pressure (mmHg) | 94( $\pm 2.12)$ | $92( \pm 11.31$ | -2 | 0.58 |
| Total Cholesterol (mmolli) | 7.1( $\pm 1.8)$ | $6.1( \pm 0.56)$. | -7.4 | 0.34 |
| High Density Lipoprotein males (mmolll) | 1.2( $\pm 0.2)$ | 1.1( $\pm 0.1$ ) | -8 | 0.31 |
| High Density Lipoprotein females (mmolll) | 1.4( $\pm 1$ ) | 1.3( $\pm 0)$ | -8 | 0.31 |
| Triglycerides (mmolli) | $2.75( \pm 0.49$ | $2.45( \pm 0.49)$ | -11 | 0.41 |
| Body mass index ( $\mathrm{Kg} / \mathrm{m}^{2}$ ) <br> South Asians | 31( $\pm 0$ ) | 31( $\pm 1.8)$ | 0 | 0.09 |
| Body mass index ( $\mathrm{Kg} / \mathrm{m}^{2}$ ) <br> Non South Asians | 27( $\pm 1$ ) | 26( $\pm 2.8)$ | -3 | 0.08 |

Table 4.2: Summary of results for all dependent and independent variables measured at baseline and review appointments for First Degree Relatives ( $n=43$ ).

### 4.2.2 CV risk scores

| DEPENDENT <br> VARIABLES | BASELINE | REVIEW | PERCENTAGE | $\boldsymbol{P}$ |
| :--- | :---: | :---: | :---: | :---: |
| CHANGE | VALUE |  |  |  |
| QRISK (\%) | $7.5( \pm 6.4)$ | $6( \pm 5.6)$ | -20 | 0.31 |
| ETHRISK CVD (\%) | $14.5( \pm 9.1$ | $7.5( \pm 7)$ | -48 | 0.1 |
| ETHRISK CHD (\%) | $12.5( \pm 6.3)$ | $6.5( \pm 6.3)$ | -48 | 0.1 |
| FRAMINGHAM BNF (\%) | $12.4( \pm 12.1)$ | $7.3( \pm 6.7))$ | -41 | 0.68 |
| FRAMINGHAM CVD (\%) | $14.8( \pm 14.2)$ | $8.3( \pm 6.57)$ | -43 | $0.04^{*}$ |
| FRAMINGHAM CHD (\%) | $10.5( \pm 10.5)$ | $6( \pm 5.5)$ | -43 | 0.31 |
| MODIFIED FRAMINGHAM | $18.5( \pm 23.3)$ | $9.5( \pm 4.9)$ | -48 | $0.0 .2^{*}$ |
| *denotes significant difference $\ll 0$ |  |  |  |  |

*denotes significant difference $\mathrm{p}<0.05$
Table 4.3: CV Risk Scores in First Degree Relatives ( $\mathrm{n}=43$ )

### 4.2.3 Gender and age distribution



Figure 4.1: Distribution of gender and age of First Degree Relatives ( $n=43$ )

The mean age of the FDRs was 42 ( $\pm 4.24$ years).
$56 \%(n=24)$ of FDRs were females and $44 \%(n=19)$ were males.
$66 \%(n=28)$ of patients who accessed the service were less than 40 year old.

### 4.3.4 Systolic blood pressure measurement



Figure 4.2: Systolic Blood Pressure (SBP) of First Degree Relatives

At the baseline appointment $30 \%$ ( $n=13$ )of FDRs had a systolic blood pressure greater than 140 mmHg and $70 \% \mathrm{I}(30)$ less than 139 mmHg . The mean SBP of FDRs was at $140( \pm 14.8) \mathrm{mmHg}$.

At the review appointment $26 \%(\mathrm{n}=11)$ of FDRs had a systolic blood pressure greater than 140 mmHg and $74 \%(\mathrm{n}=32)$ less than 139 mmHg . The mean was 137 $( \pm 4.94) \mathrm{mmHg}$.

A Wilcoxon test revealed that these differences were not statistically significant ( $p=0.56$ ).

### 4.2.5 Diastolic blood pressure measurement



Figure 4.3: Diastolic Blood Pressure of First Degree Relatives

At the baseline appointment $28 \%(\mathrm{n}=28)$ of FDRs had a diastolic blood pressure greater than 90 mmHg and $72 \%(\mathrm{n}=31)$ less than 90 mmHg . The mean DBP of FDRs was at $94( \pm 2.12) \mathrm{mmHg}$.

At the review appointment 14\% (n=6) of FDRs had a diastolic blood pressure greater than 90 mmHg and $86 \%(n=37)$ was less than 90 mmHg . The mean was $92( \pm 11.31)$ mmHg .

A Wilcoxon test revealed that these differences were not statistically significant ( $p=$ 0.58)

### 4.2.6 South Asian body mass index



Figure 4.4: Distribution of Body Mass Index of
First Degree Relatives in South Asians
$18 \%(n=8)$ of FDRs of South Asian background at first appointment had a BMI less than $23 \mathrm{Kg} / \mathrm{m}^{2} .82 \%(\mathrm{n}=35)$ of FDR had a BMI greater than $23 \mathrm{Kg} / \mathrm{m}^{2}$.
$26 \%(n=11)$ of FDRs South Asian background at review appointment had a BMI less than $23 \mathrm{Kg} / \mathrm{m}^{2} .74 \%(\mathrm{n}=32)$ of FDRs had a BMI greater than $23 \mathrm{Kg} / \mathrm{m}^{2}$.

### 4.2.7 Non-South Asian body mass index



Figure 4.5: Distribution of Body Mass Index of First Degree Relatives in Non South Asians
$38 \%(\mathrm{n}=16)$ of FDRs of non-South Asian background at first appointment had a BMI less than $25 \mathrm{Kg} / \mathrm{m}^{2}$. $63 \%\left(\mathrm{n}=27\right.$ ) of FDRs had a BMI greater than $25 \mathrm{Kg} / \mathrm{m}^{2}$.

44\% (n=19) of FDRs non-South Asian at review appointment had a BMI less than 25 $\mathrm{Kg} / \mathrm{m}^{2} .50 \%(\mathrm{n}=22)$ of FDRs had a BMI greater than $25 \mathrm{Kg} / \mathrm{m}^{2}$.

### 4.2.8 Distribution of high density lipoproteins in males



Figure 4.6: Distribution High Density Lipoprotein of First Degree Relatives in males

In $11 \%(n=5)$ of male FDRs HDL had less than $1.0 \mathrm{mmol} / \mathrm{l}$ and $89 \%(n=38)$ greater than $1.0 \mathrm{mmol} / \mathrm{l}$ at baseline appointment. The mean was $1.2( \pm 0.2) \mathrm{mmol} / \mathrm{l}$.
$6 \%(n=3)$ of male FDRs had HDL less than $1.0 \mathrm{mmol} / \mathrm{l}$ and $94 \%(\mathrm{n}=40)$ greater than $1.0 \mathrm{mmol} / \mathrm{l}$ at their review appointment. The mean was $1.1( \pm 0.1) \mathrm{mmol} / \mathrm{l}$.

A Wilcoxon test revealed that these differences were not statistically significant. ( $\mathrm{p}=0.31$ )

### 4.2.9 Distribution of high density lipoproteins in females



Figure 4.7: Distribution High Density Lipoprotein of First Degree Relatives in females
$4 \%(n=2)$ of females FDRs had HDL less than 1.2mmol/l and 96\% ( $n=41$ ) greater than1.2mmol/I at baseline appointment. The mean was $1.4( \pm 1) \mathrm{mmol} / \mathrm{l} .16 \%(\mathrm{n}=7)$ of females FDR had HDL less than $1.2 \mathrm{mmol} / \mathrm{I}$ and $84 \%(n=36)$ greater than1.2mmol/I at their review appointment. The mean was $1.3( \pm 0) \mathrm{mmol} / \mathrm{l}$.

A Wilcoxon test revealed that these differences were not statistically significant ( $p=0.31$ ).

### 4.2.10 Distribution of cholesterol levels



Figure 4.8: Distribution of Total Cholesterol of the population
$37 \%(n=16)$ of FDRs had total cholesterol less than 5mmol/l and 61\% (n=26) of FDR had total cholesterol greater than $5 \mathrm{mmol} / \mathrm{I}$ but less than $7.5 \mathrm{mmol} / \mathrm{l}$. $2 \%(\mathrm{n}=1)$ of FDRs at their baseline appointment had a total cholesterol greater than $7.5 \mathrm{mmol} / \mathrm{l}$. The mean of the total cholesterol was $7.1( \pm 1.8) \mathrm{mmol} / \mathrm{l}$.

54\% ( $n=23$ ) of FDRs had total cholesterol less than 5mmol/l and 44\% ( $\mathrm{n}=19$ ) had total cholesterol greater than $5 \mathrm{mmol} / \mathrm{l}$ but less than $7.5 \mathrm{mmol} / \mathrm{I} .2 \%(\mathrm{n}=1)$ of the FDRs had a total cholesterol greater than $7.5 \mathrm{mmol} / \mathrm{I}$. The mean of the total cholesterol was $6.1( \pm 0.56) \mathrm{mmol} / \mathrm{l}$.

A Wilcoxon test revealed that these differences were not statistically significant $\mathrm{p}=0.34$ (not significant).

### 4.2.11 Distribution of triglycerides



Figure 4.9: Distribution of Triglycerides in first degree relatives

In 64\% ( $\mathrm{n}=28$ ) of FDRs triglycerides were less than $2.0 \mathrm{mmol} / \mathrm{I}$ and $36 \%(\mathrm{n}=15$ ) greater than $2.0 \mathrm{mmol} / \mathrm{I}$ at baseline appointment. The mean of the triglycerides was $2.75( \pm 0.49) \mathrm{mmol} / \mathrm{l}$.

In $68 \%(n=29)$ of FDRs triglycerides were less than $2.0 \mathrm{mmol} / \mathrm{l}$ and $32 \%(n=14)$ greater than $2.0 \mathrm{mmol} / \mathrm{l}$ at their review appointment. The mean of the triglycerides was $2.45( \pm 0.49) \mathrm{mmol} / \mathrm{l}$.

A Wilcoxon test revealed that these differences were not statistically significant. ( $p=0.41$ )

### 4.2.12 Cardiovascular risk using QRISK



Figure 4.10: Cardiovascular tool QRISK
$77 \%(n=33)$ of FDRs were low risk, $9 \%(n=4)$ medium risk and $14 \%(n=6)$ high risk at baseline appointment. The mean was 7.5(土6.4) \%.
$84 \%(n=36)$ of FDRs were low risk, $2 \%(n=1)$ medium risk, $14 \%(n=6)$ high risk at their review appointment. The mean was 6( $\pm 5.6) \%$.

A Wilcoxon test revealed that these differences were not statistically significant ( $p=0.31$ ).

### 4.2.13 Cardiovascular risk using ETHRISK CVD



Figure 4.11: Cardiovascular calculator ETHRISK CVD

81\% ( $n=35$ ) of FDRs were low risk, $5 \%(n=2)$ medium risk and $14 \%(n=6)$ high risk at baseline appointment. The mean of the Cardiovascular calculator ETHRISK CVD was $14.5( \pm 9.1) \%$.
$88 \%(n=38)$ of FDRs were low risk, $2 \%(n=1)$ medium risk, $9 \%(n=4)$ high risk at their review appointment. The mean of the Cardiovascular calculator ETHRISK CVD was $7.5( \pm 7) \%$.

A Wilcoxon test revealed that these differences were not statistically significant $(p=0.10)$.

### 4.2.14 Cardiovascular risk using ETHRISK CHD



Figure 4.12: Cardiovascular calculator ETHRISK CHD
$86 \%(n=37)$ of FDRs were low risk, $12 \%(n=5)$ medium risk and $2 \%(n=1)$ high risk at baseline appointment. The mean of the Cardiovascular calculator ETHRISK CHD was 12.5( $\pm 6.3) \%$.

93\% ( $n=40$ ) of FDRs were low risk, $5 \%(n=2)$ medium risk, $2 \%(n=1)$ high risk at their review appointment. The mean of the Cardiovascular calculator ETHRISK CHD was $6.5( \pm 6.3) \%$.

A Wilcoxon test revealed that these differences were not statistically significant ( $p=0.10$ ).

### 4.2.15 Cardiovascular risk using Framingham BNF



Figure 4.13: Cardiovascular calculator Framingham BNF
$86 \%(n=37)$ of FDRs were low risk, $7 \%(n=3)$ medium risk and $7 \%(n=3)$ high risk at baseline appointment. The mean of the Cardiovascular calculator Framingham BNF was 12.4( $\pm 12.1)$ \%.
$98 \%(n=42)$ of FDRs were low risk, $0 \%$ medium risk, $2 \%(n=1)$ high risk at their review appointment. The mean Cardiovascular calculator Framingham BNF was 7.3( $\pm 6.7) \%$.

A Wilcoxon test revealed that these differences were not statistically significant $(p=0.68)$.

### 4.2.16 Cardiovascular risk using Framingham CVD



Figure 4.14: Cardiovascular calculator Framingham CVD
$86 \%(n=37)$ of FDRs were low risk, $2 \%(n=1)$ medium risk and $12 \%(n=5)$ high risk at baseline appointment. The Cardiovascular calculator Framingham CVD mean was 14.8( $\pm 14.2)$ \%.
$88 \%(n=38)$ of FDRs were low risk, $7 \%(n=3)$ medium risk, $5 \%(n=2)$ high risk at their review appointment. The mean of the cardiovascular calculator Framingham CVD was 8.3( $\pm 6.5) \%$.

A Wilcoxon test revealed that these differences were statistically significant ( $p=0.046$ ).

### 4.2.17 Cardiovascular risk using Framingham CHD



Figure 4.15: Cardiovascular calculator Framingham CHD
$93 \%(n=40)$ of FDRs were low risk, $7 \%(n=3)$ medium risk and $0 \%$ high risk at baseline appointment. The mean of the cardiovascular calculator Framingham CHD was 10.5( $\pm 10.5) \%$.

98\% ( $n=42$ ) of FDRs were low risk, $2 \%(n=1)$ medium risk, $0 \%$ high risk at their review appointment. The mean of the cardiovascular calculator Framingham CHD was 6( $\pm 5.5) \%$.

A Wilcoxon test revealed that these differences were not statistically significant ( $p=0.317$ ).


Figure: 4.16. Cardiovascular calculator Modified Framingham
$70 \%(n=30)$ of FDR were low risk, $9 \%(n=4)$ medium risk and $21 \%(n=9)$ high risk at base appointment. The mean of the Modified Framingham was 18.5( $\pm 23.3$ ) \%
$79 \%(n=34)$ of FDR were low risk, $9 \%(n=4)$ medium risk, $12 \%(n=5)$ high risk at their review appointment. The mean of the Modified Framingham was 9.5( $\pm 4.9) \%$

A Wilcoxon test revealed that these differences were statistically significant $(P=0.02)$

### 4.2.19 Referral to specialist services



Figure 4.16: Intervention during Primary Prevention Service
$14 \%$ ( $n=6$ ) of FDRs were referred for other investigations. $21 \%$ ( $n=9$ ) were commenced on medication or medication was altered. $12 \%(n=9)$ of smokers were referred to a smoking cessation clinic. $25 \%$ ( $n=11$ ) were referred to a weight management service. $32 \%$ ( $n=14$ ) were referred to Consultant Cardiologist or Lipidologist. $19 \%$ referred to exercise on prescription.

## Chapter 5 - Discussion

This study, attempts to evaluate a FDRs screening programme to identify those at cardiovascular risk and support the concept that a prevention and intervention programme can be effectively implemented in the acute sector. The objective of the FDRs study was to identify and assess cardiovascular risk of FDRs with proven CHD. Furthermore, we assessed the effectiveness of cardiovascular risk reduction services on the individual.

Since $85 \%$ of CV risk is modifiable and premature CHD constitutes a significant burden of morbidity and mortality in England, it is prudent to reduce lifetime risk by targeted primary prevention in high risk populations such as FDR, many of whom would not be able to access conventional Health Checks in England. Delivery of a service which systematically identifies and offers primary prevention to FDR of patients with documented premature CHD, such as this one discussed, would enable complementary strategies to address absolute risk and lifetime risk to be delivered in tandem.

## Demographics

The FDR who accessed the service were younger 40 years of age (66\%) (figure 4.1), suggesting the younger FDR are more motivated and took up the opportunity to modify their cardiovascular risk. However, the service was predominately accessed by female (56\%) and south Asians (63\%) (Table 4.1). This study identified risk factors in individuals who would not conventionally be accessed by the NHS Health Checks programme. It could be argued that the age criteria of the National Programme should be lowered to include FDR's under the age of 40. It could also be
recommended that this service could be seen as complementary to NHS Health checks. South Asians group are considered to be hard to reach groups. For this study the majority of FDR that accessed the service were from a south Asian background. In addition the Joint British Society 2 (2005), suggests that asymptomatic people should have an opportunistic cardiovascular risk check over the age Of 40. But also recognises that younger people will be at low total risk over the short term, although they may be at very high risk relative to peers of the same age.

The primary objective of this study was to identify and assess FDRs which was successful and consequently the hypothesis $\left(\mathrm{H}_{1}\right)$ that FDR can be identified by proactive case finding was substantiated. This enables the recommendations in Joint British Society 2 guidelines, that FDR should always be screened for cardiovascular risk if they have a strong family history of CVD. As discussed earlier, Eaton et al (1996) suggested that there is a threefold increase risk of developing premature CVD when any FDRs was affected and sixfold increase when FDR developed CVD before the age of 45 years.

Both the Joint British Society 2 (2005), Department of Health, (2000) and the task force define family history of premature CHD men $<55$ and women $<65$. For the purpose of this study the author recruited men $<60$ and women $<65$, this enabled the researcher to capture a larger cohort.

The second objective of the study was to identify FDRs of patients with proven CHD, this was effectively accomplished as results clearly illustrate. This cohort of young high risk individuals would not conventionally be accessed by the NHS Health

Checks programme. Hence, demonstrates the benefits of providing a tailored service for young individuals potentially high risk and susceptible to premature CVD. 53\% of the FDR accessing the service were under the age of 40 years. This population would not be able to access the systematic Health Checks programme provided by the National Health Service. This study is supported by research conducted by Bertuzzi et al. (2003) suggesting that family history of ischemic heart disease is an independent risk factor and correlation between early onset of CHD and family history is evident therefore early intervention and management is imperative.

## Systolic and Diastolic Blood Pressure

$30 \%$ of FDR's at their baseline appointment had a systolic blood pressure greater than 140 mmHg (figure 4.2). The Wilcoxon signed rank test showed there was no significant difference $(\mathrm{p}=0.56)$ between baseline and review visit. However, the cohort of FDRs' demonstrated a percentage change - $2 \%$ reduction in systolic blood pressure reading.

Figure 4.3 illustrates that diastolic blood pressure showed a percentage change -2\% improvement over a period of 6 months even though these differences were not significant ( $p=0.56$ ). It can be argued that primary prevention intervention has an impact on the FDRs lifestyle choices. The objective of the study regarding blood pressure has been achieved and the null hypotheses can be rejected, because cardiovascular tools were able to risk stratify as, low, medium and high. The literature supports the parameters used in this study regarding systolic and diastolic (British Hypertension Society, 2004 and, Joint British Society 2 005). The result of this study is dissimilar to the EUROASPIRE I, II, \&III (2009), which demonstrated no change in blood pressure, but emphasises the compelling need for more effective lifestyle management which this study exemplifies. It could be recommended that the
time frame this study was to short and perhaps similar to EUROSPIRE study it could extended for a number of years.

The results of this study mirror the Euroaction (2008) study discussed earlier. However, the main variation is that the Euroaction study consisted of a physiotherapist and dietician. The relatives were invited to attend a particular group every week to address the risk factor and behavioural modification. It could be recommended that the service model of this study and elements of the Euroaction study (2008) could expand into the existing cardiac rehabilitation programme and provide an entire programme under one speciality.

## Body mass index

In terms of body mass index differences between the south Asians (Figure 4.4) and non south Asians (Figure: 4.5) levels of obesity were high in both populations. In the non south Asians 63\% were considered to be overweight or obese and at the review appointment $40 \%$ were categorised overweight or obese the overall percentage change was $-3 \%$. In south Asians $82 \%$ of FDR had a $\mathrm{BMI}>23 \mathrm{~kg} / \mathrm{m}^{2}$ at baseline appointment and the review it was $64 \%$ the overall percentage rate indicated no change. Taken as a whole cohort $25 \%$ were referred to weight management services $19 \%$ were referred to exercise on prescription to assist in weight management and to reduce their CVD risk (figure: 4.14). However, it's unclear whether the FDR engaged with the weight management or the exercise on prescription service at baseline or at review. This would suggest that interventions to tackle the obesity epidemic are warranted at a general population level but should perhaps be reinforced and tailored to address obesity in the high risk south Asian population, where premature CHD occurs at a significantly younger age than for other populations and results in a greater socioeconomic impact at the level of the family unit as well as the overall
economy by virtue of increasing potential years of life lost and increased disability adjusted life years. The studies discussed in Chapter 2, highlight that sedentary lifestyle is a predisposing factor to obesity. This study enabled FDRs to access services to improve their physical activity the finding of the study conducted Eriksson (1998) demonstrated that those that maintained physical fitness reduced CVD risk. Future recommendations for this study that FDRs referred to exercise on prescription need to be followed up to for a longer period to establish whether exercise actually reduced their BMI levels. Waist circumference also could be measured to define obesity as Joint British Society guidelines (2005) and Department of Health (2008) Health programme have recommended.

## Dyslipidaemia

$11 \%$ of males had an HDL less than $1.0 \mathrm{mmolll}, 89 \%$ was over $1.0 \mathrm{mmol} / \mathrm{l}$ at baseline appointment. At the review appointment $6 \%$ of males had an HDL less than $1.0 \mathrm{mmolll}, 94 \%$ was over $1.0 \mathrm{mmol} / \mathrm{l}$ at baseline appointment The overall percentage change was $-8 \%$ from a mean of $1.2 \mathrm{mmol} / \mathrm{l}$ to $1.1 \mathrm{mmol} / \mathrm{I}$ (table 4.2). Although, this demonstrates an improvement (Figure: 4.6.), these differences were not statistically significant ( $\mathrm{p}=0.31$ ).

In the females the results illustrates a contrasting trend (Figure: 4.7) 4\% of females HDL was less than $1.0 \mathrm{mmolll}, 96 \%$ was over $1.0 \mathrm{mmol} / \mathrm{I}$ at the baseline appointment. At the review appointment this showed, $16 \%$ of females HDL was less than 1.0mmolll; $84 \%$ was over $1.0 \mathrm{mmol} / \mathrm{l}$. Consequently, there has been an opposite impact on the findings. The overall percentage shows -8 from a mean $1.4 \mathrm{mmol} / \mathrm{l}$ to $1.3 \mathrm{mmol} / \mathrm{l}$, but these differences were not statistically significant ( $p=0.31$ ).
$61 \%$ of FDRs cholesterol was between the level of $5-7.5 \mathrm{mmol} / \mathrm{I}$ (figure: 4.8 ) at review appointment his level was reduced to $44 \%$. Overall, table 4.2 , shows a percentage change of $-7.4 \%$, although this was not statically significant it shows a positive shift towards cholesterol reduction. Similar results were apparent with the triglycerides (figure: 4.9.). At baseline, $64 \%$ had triglycerides less than $2 \mathrm{mml} / I$ at review $68 \%$ had triglycerides less than $2 \mathrm{mml} / \mathrm{l}$, demonstrating percentage change $-11 \%$ (table 2) from a mean of $2.75 \mathrm{mmol} / /$ to $2.45 \mathrm{mmol} / \mathrm{l}$. Figure 4.16 , exemplifies that $21 \%$ of the FDRs where commenced on medication or there current medication was optimized. However, it is indistinguishable whether FDR were commenced on medication during baseline or review appointment. Recommendation for future study could include collecting data regarding when the intervention was first commenced and follow the FDR to observe whether lifestyle or pharmacological intervention actually reduced CVD risk. The INTERHEART study (Yusuf et al, 2004) identified cholesterol as a risk factor associated with CHD. The study was a large study performed in 52 countries simultaneously. The FDRs study could be extended into varies acute sectors throughout England, recruiting FDRs from the age of 18 years and the collect data which actually reflects the population of England. The Euroaction study (2008), to a degree performs this however, the index cases were less than 80 years whereas in this study the age was younger for proven CHD, the Framingham study concluded that life time risk was greater in men aged 40 years. It would also have been of benefit to identify FDR with low HDL and to distinguish whether they were referred to exercise on prescription and repeat blood sample to see whether their HDL had improved. Research discussed earlier suggested that HDL increase with physical activity within a year of commencing moderate exercise.

## Cardiovascular risk predication tools

QRISK tool (figure 4.10) identified $77 \%$ of FDRs as low risk, $9 \%$ at medium risk and $14 \%$ at high risk at baseline. At the review appointment $84 \%$ were categorised as low risk, $2 \%$ medium risk and $14 \%$ high risk. Overall, table 4.3 shows percentage change of $-20 \%$, from a mean of $7.5 \%$ to $6 \%$, but this difference was not statically significant ( $p=0.31$ ).

ETHRISK CVD (figure 4.11) calculated $81 \%$ as low risk, $5 \%$ medium risk and $14 \%$ high risk at baseline. At the review appointment $88 \%$ calculated as low risk, $2 \%$ medium risk and $9 \%$ high risk. Overall, table 4.3 shows percentage change of $-48 \%$, from a mean of $14.5 \%$ to $7.5 \%$, but this difference was not statically significant ( $p=0.1$ ).

ETHRISK CHD (figure 4.12) calculated $88 \%$ as low risk, $12 \%$ medium risk and $2 \%$ high risk at baseline. At the review appointment $93 \%$ calculated as low risk, $5 \%$ medium risk and $2 \%$ high risk. Table 4.3 , shows percentage change of $-48 \%$, from a mean of $12.5 \%$ to $6.5 \%$, but this difference was not statically significant ( $p=0.1$ ).

Framingham BNF (figure 4.13) calculated $86 \%$ as low risk, $7 \%$ medium risk and $7 \%$ high risk at baseline. At the review appointment $98 \%$ calculated as low risk, $0 \%$ medium risk and $2 \%$ high risk. Overall, table 4.3 shows percentage change of $-41 \%$, from a mean of $12.4 \%$ to $7.3 \%$, but this difference was not statically significant ( $p=0.68$ )

Framingham CVD (figure.4.14) calculated 86\% as low risk, 2\% medium risk and 12\% high risk at baseline. At the review appointment $88 \%$ calculated as low risk, $7 \%$ medium risk and 5\% high risk. Overall, table 4.3 shows percentage change of $-43 \%$, from a mean of $14.8 \%$ to $8.3 \%$, but this difference was not statically significant. $(p=0.04)$

Framingham CHD (figure 4.15) calculated $93 \%$ as low risk, $7 \%$ medium risk and $0 \%$ high risk at baseline. At the review appointment $98 \%$ calculated as low risk, $2 \%$ medium risk and $0 \%$ high risk. Overall, table 4.3 shows percentage change of $-43 \%$, from a mean of $10.5 \%$ to $6 \%$, but this difference was not statically significant ( $p=0.317$ ).

Modified Framingham (figure 4.16) calculated 70\% as low risk, 9\% medium risk and 21\% high risk at baseline. At the review appointment 79\% calculated as low risk, 9\% medium risk and $12 \%$ high risk. Overall, table 4.3 shows percentage change of $43 \%$, from a mean of $23.3 \%$ to $4.9 \%$, but this difference was statically significant ( $p=0.02$ ).

All the tools accessed were able to risk stratify FDRs if they were over a particular age, but the age criteria for each tool was different. The QRISK (Hippisley-Cox et al. 2008) and ETHRISK (Brindle et al. 2006) age criteria was $35-74$ years of age, Framingham BNF, CHD, CVD (Joint British Society, 2005) is 30-75 years of age and for the Modified Framingham (Marshall, 2008) age from 18 years of age. For the benefit of the study those that were under the age 30, modified Framingham was able to communicate risk to the FDR over the age 18 especially if they possessed all three risk factor Dyslipidaemia, hypertension and was smoker. The calculator was
adjusted according to age and risk factor changed to show the FDR how lifestyle modification could reduce the impact of CVD. In this study at the review appointment the calculator was used again to assess risk, it evident in the guidelines discussed that once treatment has been commenced tool should be used again to illustrate CVD risk (Joint British Guidelines, 2005., Department of Health Health Checks Programme, 2008). However the FDR were informed of this, but still sought to observe if their risk had decreased. As Goodyear-Smith et al (2008) suggest calculating risk is not a unilateral decision but decision which shared between clinician and individual. For the purpose of this study, the risk calculated and communicated to the FDRs was based on the Modified Framingham as the NICE lipid modification guidelines (2008).

## Primary prevention service

$14 \%$ of FDRs were referred for other investigations. $21 \%$ were commenced on medication or medication was altered. $12 \%$ of smokers were referred to a smoking cessation clinic. $25 \%$ were referred to a weight management service. $32 \%$ were referred to Consultant Cardiologist or Lipidologist. 19\% referred to exercise on prescription. With this service the FDRs had access to lifestyle risk management services which they were able to access at time of appointment whether at baseline or review. $32 \%$ had to be reviewed by the cardiologist or lipidologist and further investigations were initiated. It can be suggested these individual would not have had access to a specialist service at such early age if they never took the initiative to contact the researcher. As proposed previously it is only the motivated FDRs that actually access the service. However a limitation to the study is that those that had investigation performed the outcomes of these were not collated, future recommendation would be gather the data. Furthermore, when collecting the data it
would have been useful to have categorised which speciality the FDR was referred to.

## Summary

Overall, this evaluation demonstrates how one might establish a service to identify individuals with significant risk factors for CHD at a young age by taking advantage of an opportunity when these families might be a more captive audience at the time of an acute event in a relative. In order to provide not only cardiovascular risk assessment, but also provision of upstream lifestyle risk management services and behavioural change. This demonstrates the philosophy of the NHS Next Stage Review that prevention is everybody's business and requires collaborative working and partnerships across care pathways between primary and secondary care to provide for identification and management of individuals at high cardiovascular risk and thereby contribute to the reduction in premature CVD burden and health inequalities overall.

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