KWAME NKRUMAH UNIVERSITY OF SCIENCE AND TECHNOLOGY, KUMASI, GHANA

COLLEGE OF HEALTH SCIENCES

SCHOOL OF MEDICAL SCIENCES

DEPARTMENT OF COMMUNITY HEALTH

CARDIOVASCULAR DISEASE RISK ASSESSMENT AMONG PATIENTS ATTENDING THE CARDIAC CLINIC AT THE KOMFO ANOKYE TEACHING HOSPITAL, KUMASI

BY

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NOVEMBER, 2014

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A THESIS SUBMITTED TO THE DEPARTMENT OF COMMUNITY HEALTH, SCHOOL OF MEDICAL SCIENCES, COLLEGE OF HEALTH SCIENCES, KWAME NKRUMAH UNIVERSITY OF SCIENCE AND TECHNOLOGY- KUMASI, IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF PUBLIC HEALTH (MPH) IN HEALTH EDUCATION AND PROMOTION

By

WAINDIM NYIAMBAM YVONNE

NOVEMBER, 2014

DECLARATION

I hereby declare that this submission is my own work towards the MPH and that, to the best of my knowledge, it contains no material previously published by another person, nor material which has been accepted for the award of any other degree of the University, except where due acknowledgement has been made in the text.

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6th November, 2014

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ABSTRACT

Over the last ten years, cardiovascular diseases (CVD) have risen to be among the top causes of admission and institutional deaths in Ghana. Assessment of an individual's "total" predicted risk of developing a CVD event in 5 or 10 years using risk scores has been identified as one of the ways to determine the burden of CVD risk and to guide treatment decisions. However, there is little information on the assessment of "total" cardiovascular disease risk in Kumasi. The objective of this study therefore was to assess the ten year cardiovascular disease risk among patients attending the cardiac clinic of the Komfo Anokye Teaching Hospital. A hospital based cross-sectional study was conducted among patients attending the cardiac clinic of the Komfo Anokye Teaching Hospital, Kumasi, aged 30 – 84years. Four hundred and forty one hospital records of patients without overt cardiovascular disease but attending the cardiac clinic were randomly selected and reviewed. Cardiovascular disease risk assessment was based on the most recent Framingham Risk Score by D'Agostino *et al.,* 2008. The prevalence of low, medium and high total CVD risk were 41.5% (95% C I 36.9 – 46.1), 28.1% (95% C I 24.0 – 32.3) and 30.4% (95% C I 26.1 – 34.7) respectively. More men were at moderate to total CVD high risk compared to females (64% vs 52.2%, p value for trend = 0.003). Increase in Body Mass Index, alcohol consumption and triglycerides did not show a significant trend with increase in total CVD risk (p= 0.492; p=0.820; p=0.057) respectively. Being male, aged, diabetic, a smoker and having high LDL-C levels coupled with low HDL-C levels were significantly associated with high total 10year CVD risk.

Key words: Total cardiovascular disease risk, Kumasi, Framingham Risk Score

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LIST OF ABBREVIATIONS

BP	Blood Pressure
CHD	Coronary Heart Disease
CVD	Cardiovascular Disease
FRS	Framingham Risk Score
GHS	Ghana Health Service
HDL-C	High Density Lipoprotein Cholesterol
IHD	Ischaemic Heart Disease
ISH	International Society of Hypertension
KATH	Komfo Anokye Teaching Hospital
LDL-C	Low Density Lipoprotein Cholesterol
WHF	World Heart Federation
WHO	World Health Organisation

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CHAPTER ONE

INTRODUCTION

1.1 Background

Cardiovascular disease (CVD) is a broad term for disorders of the heart and blood vessels which include; Coronary Heart Disease (heart attack), Cerebrovascular Disease (stroke), Peripheral Arterial Disease, Pulmonary Embolism, deep vein thrombosis, Congenital Heart Disease and Rheumatic Heart Disease, of which heart attack and stroke are the most common manifestations of (WHO, 2013). Current epidemiological evidence indicates that CVDs make a large contribution to the non-communicable disease burden in low and middle-income countries (Mendis and Alwan 2011). Globally, an estimated 17.3 million people died from CVDs in 2008 with over 80% of the deaths taking place in low and middle-income countries (WHO, 2013). In 2012, Ischaemic Heart Disease was identified as the leading cause of global death and disability adjusted life years (DALYs), with an increase of 29% from 1990 (Lim 2013). Furthermore, it is estimated that more than 23 million people will die annually from CVDs in 2030, with about two thirds of the deaths caused by stroke and occurring in people under 70 years old (Mensah 2008, Mendis *et al.*, 2011). In fact, projections indicate that while mortality from non-communicable diseases will increase in all regions of the world over the next ten years, the greatest increase is expected to occur in Africa (Mendis and Alwan, 2011). These figures all provide evidence that cardiovascular disease is no longer a disease of the "rich" or high income countries as initially thought. In his concluding remarks to the Forum on Global Health in 2009, the UN Secretary General, Ban Ki Moon described non-communicable diseases as no

longer diseases of the wealthy; rather "they hamper the people and economies of the poorest populations even more than infectious diseases, thus representing a public health emergency in slow action"(www.un.org)

The burden of CVD transcends mortality and morbidity to impose huge financial burdens on nations and households. According to a joint report by the Harvard School of Public Health and World Economic Forum, globally, non-communicable diseases pose a substantial economic burden that will evolve into a staggering loss of $47 trillion over the period 2010-2030 and cardiovascular disease is a major contributor to this economic burden (Bloom *et al.,* 2012). Cardiovascular diseases are among the most severe threats to global economic development (Narayan *et al.,* 2010). At the household level, non-communicable diseases especially cardiovascular disease pose heavy financial burdens more especially on poor households which deters many people affected from seeking care (Kankeu *et al.,* 2013).

In Sub-Saharan Africa, an epidemiological transition from predominantly infectious to non-communicable diseases has been reported with the age standardized death rates from non-communicable diseases being higher in at least 4 Sub-Saharan countries than in high income countries (Dalal *et al.,* 2011). As in other regions of the world, CVD also constitutes the predominant form of non-communicable diseases in this region. Drivers of this rising epidemic include; the changing demographic profile with a greater survival into adulthood and relative aging of the population; unplanned urbanization and changes in lifestyle associated with economic development which include diet, smoking, adiposity and alcohol use (Dalal *et al.,* 2011, Mendis and Alwan 2011).

In Ghana, CVD rose from being the seventh and tenth cause of death in Accra in 1953 and 1966 respectively, to the number one cause of death in 1991 and 2001 and it has continued as one of the major causes of mortality in the country since then (Agyei-Mensah and Aikins, 2010). The WHO (2011) reported that non-communicable diseases accounted for 34% of total deaths and 31% of total disability-adjusted-life years (DALYs) in Ghana with cardiovascular diseases being the leading cause of non-communicable disease deaths (15% of the total). Cardiovascular diseases are among the top causes of admission and institutional deaths. A study at the Komfo Anokye Teaching Hospital, Kumasi revealed that stroke constituted 9.1% of total medical adult admissions and 13.2% of all medical adult deaths, with the mean age of stroke patients being 63.7 years (Agyemang *et al.,* 2012).

The major modifiable risk factors for CVD are known and their impact is similar in most regions of the world (Tunstall-Pedoe, 2011). However, while many high income countries are experiencing a decrease in the prevalence of risk factors hence a decreased incidence of CVD, it is the contrary in many low and middle income countries (Anand and Yusuf, 2011) where a rise in the prevalence of risk factors has been reported by a number of studies (de-Graft Aikins, 2007, Mensah 2008, Bosu, 2010, Dzudie *et al.,* 2012, Dewhurst *et al.,* 2013, Owusu *et al.,* 2013). Risk factors have been shown to cluster and interact synergistically to promote CVD risk and epidemiological evidence indicates that combining risk factors into scores is capable of predicting an individual's total cardiovascular risk with reasonable accuracy (Conroy *et al.,* 2003, Hippisley-Cox *et al.,* 2007, Ridker *et al.,* 2007, D'Agostino *et al.,* 2008, Cooney *et al.,* 2010). Risk

scoring takes into account the co-existence in an individual of a range of risk factors and has thus been recommended by many guidelines on the prevention of CVD (Dugee *et al.*, 2013)

1.2 Statement of the Problem

A hospital based study at the Komfo Anokye Teaching Hospital – Kumasi, Ghana on the burden of stroke in Ashanti Region revealed that stroke was responsible for 9.1% of total medical adult admissions and 13.2% of all medical adult deaths, with the mean age of stroke patients being 63.7years (95% C I=62.8, 64.57) (Agyemang *et al.*, 2012) If these adults were aware of their risk status 10 years earlier on one hand, it is most likely they would have engaged in prevention strategies to remain at low risk or lower their risk for those in the high risk category. If Clinicians on the other hand were aware, it is most likely more aggressive treatment/prevention strategies would have been employed to reduce the risk of high risk patients while maintaining those at low risk at low risk. Unfortunately, because CVD events develop gradually and silently, most people are unaware of their risk status (WHO, 2007) and so may fail to optimize and individualize preventive/treatment strategies. This is evidenced in studies which reveal a poor control in CVD risk factors in Ghana (de-Graft Aikins,2007, Mensah, 2008, Bosu 2010, Agyemang *et al.*, 2012, Owusu *et al.*, 2013). Furthermore, given that CVD risk increases with age, as life expectancy increases in Ghana, it is expected that the number of people who will experience a CVD event will increase significantly in the near future (Agyemang *et al.*, 2012). It is therefore imperative that urgent measures be taken to know the burden of CDV risk so as to devise strategies to reduce these risks and in so

doing, optimize the health outcomes for stroke and other CVDs especially in people with a single raised risk factor. However, there is little information on the assessment of "total" cardiovascular disease risk and its consequential application in medical practice in Ghana.

1.3 Rationale for the Study

Assessment of an individual's "total" predicted risk of developing a CVD event in 5 or 10 years using risk scores has been identified as one of the ways to determine the burden of CVD risk and to guide treatment decisions (Cooney *et al.,* 2010;Mendis and Alwan 2011;Dugee *et al.,* 2013). Risk scoring makes patients aware of their risk status and can therefore serve as enough motivation for engaging in activities to lower overall risk. Risk scoring also provides a rational means of making decisions about intervening in a targeted way, thereby making best use of resources available to reduce cardiovascular risk (Mendis and Alwan, 2011). Unfortunately, many people are not aware of their risk status and may go on to develop cardiovascular events in the nearest future. In addition, there is little information on the effect of the combination of these risk factors on total cardiovascular risk in Kumasi though some studies have assessed the prevalence of individual risk factors (Owusu *et al.,* 2013). The purpose of this study therefore was to assess total cardiovascular disease risk - the probability of an individual experiencing a cardiovascular event over a 10 year period using the most recent Framingham Risk Scoring Algorithm (D'Agostino *et al.,* 2008), among persons attending the Cardiac Clinic at the Komfo Anokye Teaching Hopital, Kumasi.

1.4 Conceptual Framework

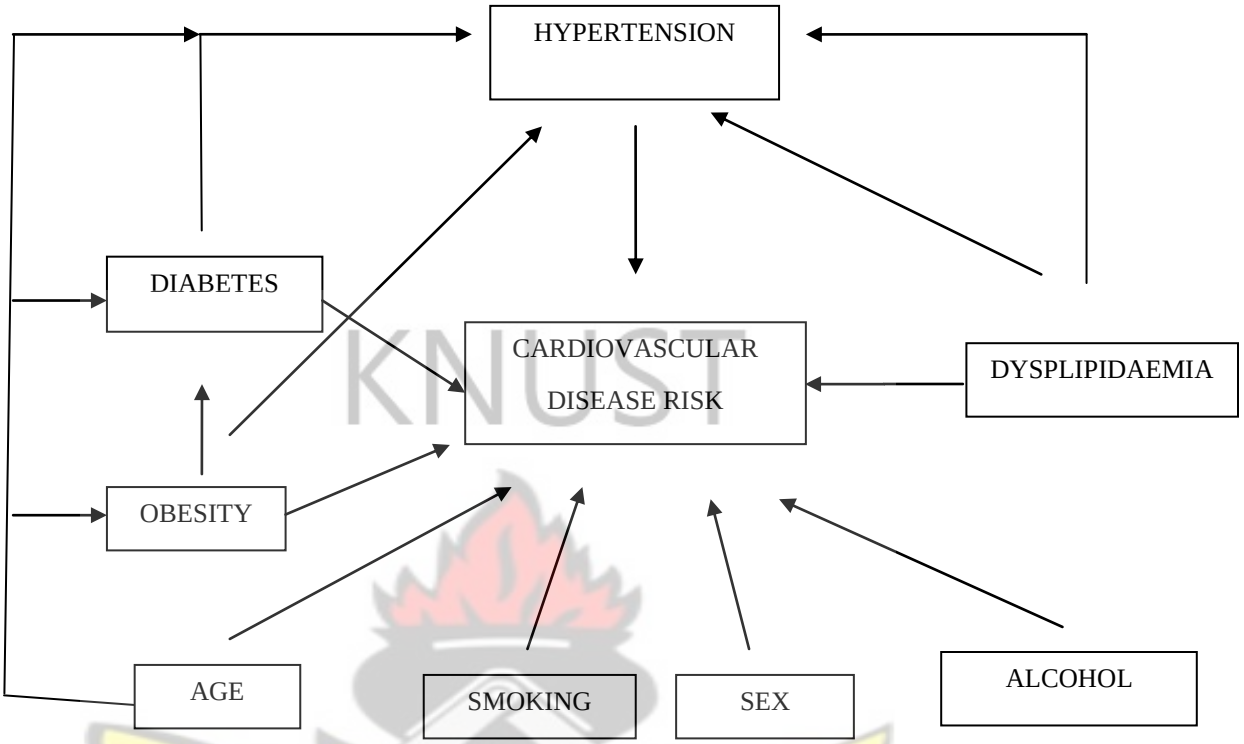

Figure 1: Conceptual Framework

All the risk factors illustrated above have a potential to increase the risk of cardiovascular disease either independently or in combination with others. Obesity for instance has been shown to increase CVD risk independently and has also been shown to cause diabetes and hypertension which are also risk factors for CVD.

1.5 Study Questions

The questions the study sought to answer were:

- Which are the prevailing risk factors of cardiovascular disease among patients attending the cardiac clinic at KATH?
- What is the distribution of total 10year CVD risk?

- What is the effect of each risk factor on total CVD risk?
- How sensitive and specific is the risk model in predicting CVD?
-

1.6 General Objective

To assess 10 year cardiovascular disease risk among persons attending the Cardiac Clinic at Komfo Anokye Teaching Hospital, Kumasi using the most recent Framingham Risk Scoring algorithm.

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1.7 Specific Objectives

1. To investigate the prevailing CDV risk factors among study participants
2. To determine the distribution of total 10year cardiovascular risk
3. To determine the effect of each risk factor on total CVD risk
4. To determine the sensitivity and specificity of the risk FRS in stratifying risk

CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction

A risk factor for a disease can be defined as a measurable biological characteristic of an individual that precedes a well-defined outcome of that disease, predict that outcome and is directly in the biological causal path (Balagopal *et al.*, 2011). The conventional risk factors of CVD are known (D'Agostino *et al.*, 2008) and some of their mechanisms of action as to how they cause CVD have been elucidated. CVD risk factors can be broadly classified into 2 groups: modifiable and non-modifiable. Whilst the non-modifiable risk factors are those which one has no control over and include age and sex the modifiable risk factors are the contrary and include; hypertension, diabetes, dyslipidaemia, obesity, smoking, alcohol, diet, psychosocial factors and physical exercise (Yusuf *et al.*, 2004). This chapter will focus on the CVD risk factors illustrated in the conceptual framework (figure 1) highlighting how they cause or contribute to CVD risk. There will also be a review on the rationale of risk scoring algorithms particularly the Framingham Risk Score and the importance of communicating CVD risk

2.2 Risk Factors of Cardiovascular Diseases

2.2.1 Age

The normal aging process is associated with progressive alterations in cardiovascular structure and function in healthy individuals even in the absence of major contributing factors to cardiac dysfunction like hypertension and atherosclerosis (Sung and Dyck, 2012). Age is the strongest determinant of stroke, which is less common before 40

years. However, from 55years on, the incidence of stroke doubles with each successive decade (Soler and Ruiz 2010). Age is also a very important and independent risk factor for Coronary Heart Disease (Soler and Ruiz, 2010). Thickening and dilatation of large arteries, large artery stiffness, endothelial dysfunction, increase in collagen content with the fraying of elastin fibers, cardiomyocyte apoptosis and blunted β-adrenergic response are amongst the several changes that occur with age even amongst healthy normotensive individuals (Fleg and Strait 2012, Sung and Dyck 2012). Insights from the Framingham Study reveal 3 haemodynamic phases in the changing pattern of ageing and blood pressure. Under the age of 50years, the progressive rise in DBP and SBP suggest the predominance of increased vascular resistance; the constancy of DBP for those in their 50s, together with the asymptotic leveling of mean arterial pressure and increased slope of pulse pressure suggest increased vascular resistance along with increased large artery stiffness and lastly, the fall in DBP during later ages signals preponderance(superiority) of large artery stiffness as the cause of further rise in SBP and, hence, dramatic widening of pulse pressure in the elderly (Franklin and Wong, 2013). These changes cause increases in systolic and pulse pressure, which in turn lead to left ventricular wall thickening and reduced early diastolic filling rate (Fleg and Strait 2012), exposing the elderly to hypertension, stroke, coronary heart disease, heart failure and other cardiovascular complications (Bosu 2010, Agyemang *et al.*, 2012, Berry *et al.*, 2012).

2.2.2 Sex

For a long time, coronary heart disease (CHD) has been considered as a disease predominantly affecting men and consequently women were not included in cardiovascular research programs (Roeters van Lennep *et al.*, 2002). Although women

and men share most classic CVD risk factors, there is often an incorrect perception that women are 'protected' against cardiovascular disease thus an underestimation of their risk of heart disease (Maas and Appelman 2010). This incorrect perception may be attributed to the observation that for women, up to 20% of coronary events occur in the absence of the major risk factors. Also, it has been reported that many women with traditional risk factors do not experience coronary events (Ridker *et al.,* 2007). Furthermore, the perceived "protection" may also stem from the fact that exposure to oestrogen during the fertile periods of life delays the manifestation of atherosclerotic disease in women (Maas and Appelman 2010). Oestrogen is known to have a regulatory effect on several metabolic factors such as inflammatory markers, lipids, and the coagulant system (Roeters van Lennep *et al.,* 2002, Merz *et al.,* 2003, Maas and Appelman 2010, Vaccarino *et al.,* 2011). Furthermore, data from the Women's Ischaemia Syndrome Evaluation (WISE) study show that young women with endogenous oestrogen deficiency have a more than sevenfold increase in coronary artery risk (Merz *et al.,* 2003). This "protection" however changes during menopause which is associated with a worsening CDV risk profile (Maas and Appleman, 2010). Following menopause, central obesity with an increase in visceral fat occurs more frequently in women compared with ageing men and this increase in obesity parallels an increase in the prevalence of type II Diabetes Mellitus, with women having a greater risk of CVD complications than their male counterparts (Maas and Appelman, 2010). Furthermore, systolic blood pressure rises more steeply in ageing women compared to men and at an older age (>75 years). Isolated systolic hypertension is 14% more prevalent in women and an important cause of left ventricular hypertrophy, (diastolic) heart failure and

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strokes (Maas and Appelman, 2010). Total cholesterol and LDL-cholesterol levels also rise by 10 and 14% respectively in women compared to men (Vaccarino *et al.,* 2011). All of these changes, therefore, increase the risk and makes post-menopausal women more vulnerable to CVD.

2.2.3 Obesity

Obesity is a nutritional disorder that is traditionally defined in humans by measure of body mass index (BMI $\geq 30 kg/m^2$). Other less commonly used indexes but possibly with more predictive power include; waist circumference, waist-to-hip ratio, hip circumference, body fatness and weight-to-height ratio (Goh *et al.,* 2014). Obesity is known as one of the major risk factors for hypertension, heart failure and coronary heart disease. Even without arterial pressure and age, obesity increases the risk of left ventricular hypertrophy (LVH),, as well as other structural abnormalities which not only increase the risk of heart failure but also affect systolic and diastolic pressure as well (Poirier *et al.,* 2006;Lavie *et al.,* 2009). Central obesity especially is associated with low-grade inflammation which has been implicated in the pathogenesis of diabetes, and both conditions are associated with significant increase in morbidity and mortality due to CVD (Matheus *et al.,* 2013). Obesity is also known to cause insulin resistance which is largely responsible for the development of type 2 diabetes in adults (Cheung and Li 2012). However, despite the adverse effects of obesity on health in general, an obesity paradox has been reported wherein overweight and obese patients with established cardiovascular disease tend to have a more favourable short and long term prognosis (lower all cause and cardiovascular mortality rates) (Oreopoulos *et al.,* 2008;Lavie *et al.,* 2009;Sung and Dyck 2012)

2.2.4 Diabetes Mellitus

Increased cardiovascular morbidity and mortality in persons with type II diabetes has been well established (Bulugahapitiya *et al.*, 2009). Late and early onset of diabetes has been associated with significantly increased risk of CVD events even after adjustment for conventional and novel risk markers. People with a mean diabetes duration longer than 8 years have been shown to be at more risk compared to those with early onset (Evans *et al.*, 2002, Wannamethee *et al.*, 2011). Though some studies have identified diabetes as a coronary heart disease equivalent (people with diabetes without prior myocardial infarction having a similar risk of coronary heart disease compared to those without diabetes who have had a myocardial infarction) (Haffner *et al.*, 1998, Dagenais *et al.*, 2009, Wannamethee *et al.*, 2011), others do not support the claim (Bulugahapitiya *et al.*, 2009). Several mechanisms have been proposed to explain the increased CVD risk in diabetics some of which include; oxidative stress, insulin resistance, poor glycaemic control, low-grade inflammation and abnormalities in endothelial, vascular smooth muscle cell, and platelet function (Creager *et al.*, 2003, Matheus *et al.*, 2013, Paneni *et al.*, 2013). Diabetes mellitus also contributes to defects in the autonomic nervous system and patients with diabetic autonomic nephropathy have increased rates of sudden cardiac death as well as a higher overall cardiovascular mortality rate (Matheus *et al.*, 2013). All of these abnormalities in turn contribute to the cellular events that cause atherosclerosis and subsequently increase the risk of the adverse cardiovascular events that occur in patients with diabetes(Creager *et al.*, 2003).

2.2.5 Smoking

Smoking is estimated to cause nearly 10 per cent of cardiovascular disease (CVD) and is the second leading cause of CVD after high blood pressure. It is also the most preventable cause of cardiovascular morbidity and mortality. The risk of a non-fatal heart attack increases by 5.6 per cent for every cigarette smoked and persists even at only one to two cigarettes per day (WHF, 2014). There are over 4000 known components in cigarette smoke which is divided into 2 phases; a tar (particulate) phase which contains over 10^{17} free radicals and the gas phase which contains more than 10^{15} free radicals (Ambrose and Barua, 2004). Cigarette smoking predisposes an individual to several different clinical atherosclerotic syndromes including; stable angina, acute coronary syndromes, sudden death, and stroke (Ambrose and Barua, 2004). Cigarette smoking has been found to impair endothelium vasodilation (vasomotor dysfunction), increase the level of multiple inflammatory markers and modify lipid profile, increased levels of total cholesterol, triglycerides and low density lipoprotein cholesterol but lower levels of high density lipoprotein cholesterol. Smoking also increases the oxidative modification of LDL-C, which together with the aforementioned lipid changes are integral components for the initiation and progression of atherosclerosis and thus may explain the increased risk of CVDs among smokers (Ambrose and Barua, 2004). These effects of smoking go beyond the active smoker to affect even those who do not smoke. In 2004, exposure to second-hand smoke is estimated to have caused 603 000 deaths, 379 000 of which were due to Ischaemic Heart Disease accounting for about 1% worldwide mortality (Öberg *et al.,* 2011). Second hand smoke results from the combination of 85% side stream smoke (smoke emitted from the burning ends of a

cigarette) and a small fraction (15%) of exhaled mainstream smoke from smokers. It (second-hand smoke) contains a relatively higher concentration of the toxic gaseous component than mainstream cigarette smoke and is believed to cause CVD in much the same way as in active smokers (Ambrose and Barua, 2004). Furthermore, there is evidence of sex-related differences in CVD morbidity and mortality in relation to smoking, with women being more vulnerable. Women who smoke have been reported to have a 25% greater relative risk of coronary heart disease than do male smokers, independent of other cardiovascular risk factors (Huxley and Woodward, 2011). Women have also been reported to be more exposed to second hand smoke and suffer the largest effects on death than men (Öberg *et al.*, 2011)

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2.2.6 Hypertension

Hypertension is a powerful independent risk factor of many cardiovascular events. Hypertension causes left ventricular hypertrophy and structural remodeling of the heart including cardiac fibrosis. These structural changes contribute to an increase in left ventricular stiffness which in turn leads to diastolic dysfunction and elevations in left ventricular end diastolic pressure (Drazner, 2011, Sung and Dyck 2012, Owusu *et al.*, 2014). Three types of hypertensive heart failures have been described: type I wherein the decompensated myocardium is still strong enough to sustain a high blood pressure and patients with this type present with high blood pressure on admission; type II wherein patients may present with a low or normal blood pressure initially but following treatment of the heart failure, the decompensated myocardium may recover sufficiently enough for the blood pressure to rise again few days after the commencement of treatment; type III wherein the decompensated myocardium has been irreversibly

damaged by the chronic uncontrolled hypertension that despite adequate treatment of the heart failure, the myocardium is not strong enough to sustain a high blood pressure any longer (Owusu *et al*., 2014). Hypertension has also been implicated as the most powerful, independent, highly prevalent, modifiable risk factor of stroke at the population level (Mensah, 2008). Epidemiological data show that in middle-aged people, a 10 mm Hg increase in systolic BP is associated with about 40% more stroke and that the risk of stroke approximately doubles for every 7.5 mm Hg increase in diastolic BP (Mensah, 2008). While many developed countries have begun to reduce hypertension in their populations through strong public health policies like; widely available diagnosis and treatment that deal with hypertension and other risk factors together coupled with salt reduction in processed food, developing countries are rather experiencing many more people who suffer from strokes and heart attacks due to undiagnosed and uncontrolled hypertension (WHO, 2013).

2.2.7 Dyslipidaemia

Dyslipidemia is recognized as a prominent risk factor for cardiovascular disease and is characteristically seen in patients with obesity, insulin resistance, type 2 diabetes mellitus and the metabolic syndrome (Leon and Bronas 2009, Miller 2009, Musunuru 2010). Dyslipidaemia is defined as elevated fasting blood levels of Total Cholesterol, Low Density Lipoprotein (LDL) cholesterol, Triglycerides and reduced levels of High-Density Lipoprotein (HDL) cholesterol, alone or in combination (Leon and Bronas 2009). Although each component of dyslipidaemia has been shown to increase the risk of CVD, evidence supporting causality is strongest for elevated levels of LDL-C and total cholesterol (Miller 2009, Musunuru 2010). This probably is because small dense

LDL-C has a greater susceptibility to oxidation and thus is more likely to instigate the inflammatory process in vascular endothelium that underlie atherosclerotic disease (Musunuru 2010). HDL-C on the other hand also has a strong epidemiologic relationship with CVD but unlike LDL-C, increased HDL-C levels are rather protective against CVD. Evidence from major a prospective cohort study show that each 1-mg/dL decrement in HDL-C is associated with a 2% to 3% increased risk of CHD, whereas each l-mg/dL increase is associated with a 6% lower risk of death from a coronary event (Genest, 2008)

Triglycerides have been implicated to contribute directly to atherosclerotic plague formation and progression and also to drive atherogenesis via indirect mechanisms particularly those involving lipolysis and binding at the artery wall (Chapman *et al.*, 2011). The role of triglycerides in atherogenesis is, however, often complicated by the inverse relationship that exists between the plasma concentrations of triglycerides and HDL cholesterol. Because cholesterol esters are transferred from HDL-C to Very Low Density Lipoprotein (VLDL) - a triglyceride rich molecule, higher levels of VLDL promote this transfer and so lead to lower HDL cholesterol concentrations. Any association between triglycerides and CVD risk thus, has often been attenuated by taking into account serum HDL-C concentrations (Jones, 2013). Though some studies have reported a statistically significant relationship between fasting triglyceride concentrations and the risk of cardiac death and there exist a basic biological plausibility as to its aetiology in CVD, available clinical literature however is inconsistent and complicated (Jones, 2013)

2.2.8 Alcohol

While the harmful effects of alcohol on conditions such as, injuries, liver cirrhosis and cancers of the liver, colorectum, breast, to name a few have been firmly established (Rehm *et al.,* 2010), cohort studies have pointed out that light-to-moderate alcohol consumers (less than 3 drinks a day, or 1 to 2 glasses of wine a day) have an increased survival compared to abstainers (Ronksley *et al.,* 2011). Current evidence also suggests the protective effects of moderate drinking on cardiovascular events including Coronary Heart Disease (CHD), Ischemic Stroke, Peripheral Arteriopathy and congestive heart failure (Arranz *et al.,* 2012). However, while some studies have described the relationship between alcohol consumption and coronary heart disease as J shaped or U shaped suggesting an increased risk among drinkers of greater amounts of alcohol (Arranz *et al.,* 2012, Holmes *et al.,* 2014), others have described an L shape, suggesting no increase in coronary heart disease risk associated with higher alcohol consumption (Ronksley *et al.,* 2011). Moderate alcohol consumption has also been associated with lower stroke incidence and mortality though this association differs by stroke sub-type with a slightly lower risk of Ischaemic Stroke but higher risk of Haemorrhagic Stroke (Ronksley *et al.,* 2011). The cardioprotective effects of moderate alcohol drinking have mainly been attributed to an increase in antioxidant capacity, changes in lipid profiles (raises HDL-C levels), improved insulin sensitivity, and the anti-inflammatory effects produced (Arranz *et al.,* 2012, Holmes *et al.,* 2014). However, despite these effects of moderate alcohol consumption, there are discrepancies regarding the specific effects of different types of beverages (wine, beer and spirits) on the cardiovascular system and also whether the possible protective effects of alcoholic beverages are due to their

alcoholic content (ethanol) or to their non-alcoholic components (mainly polyphenols) (Arranz *et al.,* 2012). Furthermore, one study has suggested that the perceived benefits of moderate alcohol consumption could rather be explained by the elevated cardiovascular risk from underlying poor health in non-drinkers, or confounding by lifestyle or social factors associated with light to moderate drinking (Holmes *et al.,* 2014)

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2.3 Distribution of Cardiovascular Disease Risk Factors

Most cardiovascular risk factors share a similar pathophysiology thus there is a tendency for them to cluster and interact together to promote CVD risk for instance;

Hypertension is about 6 times more frequent in obese subjects than in lean men and women (Poirier *et al.,* 2006). Activation of the sympathetic nervous system leading to primary sodium retention and thus extracellular fluid volume expansion; high plasma renin activity; angiotensinogen; angiotensin II and aldosterone levels; insulin resistance and inflammation have all been identified as some of the mechanisms of obesity induced hypertension (Kotsis *et al.,* 2010).

Diabetes and hypertension are also known to frequently occur together and there is a substantial overlap in their aetiologies and pathopysiology mechanisms. Genetic predisposition, autonomic derangements, endothelial dysfunction, premature aterial stiffening, inflammation, obesity, insulin resistance, and oxidative stress are thought to be the common pathways linking both conditions (Campbell *et al.,* 2011, Cheung and Li 2012).

Diabetes has also been reported to be associated with an altered lipid profile; high triglycerides, low HDL and a predominance of the small dense form of LDL. In fact up to 97% of diabetics are dyslipidaemic (Dokken 2008). In patients with diabetes, LDL particles can also become glycated (just like haemoglobin) causing a lengthening of its half-life and by so doing increase the ability of LDL to promote atherogenesis (Dokken, 2008)

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In the light of dyslipidaemia and hypertension, Angiotensin I over expression and endothelial dysfunction have been implicated to be the link between both conditions. Dyslipidaemia causes endothelial damage leading to loss of the vasomotor activity which may become manifested as an increase in blood pressure (Halperin *et al.*, 2006;Tuñón *et al.,* 2007). Furthermore, lipid lowering drugs have been shown to reduce incident hypertension lending more weight to a shared pathophysiology between dyslipidaemia and hypertension (Halperin *et al.,* 2006, Tuñón *et al.,* 2007)

2.4 Risk Scoring

Atherosclerosis which underlie most CDV is rarely the result of a single risk factor and the knowledge that CVD risk factors tend to cluster and interact multiplicatively to promote vascular risk has led to the development of many multivariable risk prediction algorithms that are being advocated for use to estimate absolute or total cardiovascular risk and to guide treatment of risk factors (D'Agostino *et al.,* 2008;Cooney *et al.,* 2010). While the term "total risk estimation" is perhaps a misnomer because no risk estimation algorithm accommodates all known risk factors, it refers however to the fact that CVD in most people is a product of several risk factors that may interact to greatly increase

risk (Cooney *et al.*, 2009). Because all atherosclerotic diseases of the vascular tree share almost the same risk factors, total or global CVD risk is thus generally considered the most appropriate outcome for risk estimation systems (Cooney *et al.*, 2010). The estimation of global CVD risk facilitates matching of the intensity of risk factor lowering with the estimated likelihood of disease, thereby rendering treatment to be most cost-effective. Furthermore, multivariable risk assessment also avoids overlooking high risk CVD candidates with multiple marginal risk factors and avoids needlessly alarming persons with only 1 isolated risk factor. For a risk estimation system to be clinically useful it has to fulfill the following criteria (Cooney *et al.*, 2009):

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- Appropriate statistical methods for the derivation of the function
    - Sufficient power
    - Accepted statistical methods
    - The end point predicted by the function should be defined in such a way that it is easily standardized across populations and relevant to the outcomes of randomized controlled trials of preventive measures
- Performance of the function
    - Internal and external validity
    - Discrimination - the ability of the function to separate those who will develop the end point from those who will not - often assessed using Area under receiver operating characteristic curve (AUROC)

- Calibration - a measure of how closely predicted outcomes agree with actual outcomes often assessed using either Hosmer-Lemeshow goodness of fit testing or Predicted to observed ratios

- Usability of the function
- Inclusion of appropriate risk factors
- measurable health gains in the use of the system

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Several CVD risk estimation systems are in existence including but not limited to: the Framingham Risk score (D'Agostino *et al.,* 2008), SCORE (Systematic COronary Risk Evaluation) system (Conroy *et al.,* 2003), ASSIGN score (Woodward *et al.,* 2007), QRISK (Hippisley-Cox *et al.,* 2007), QRISK2 (Hippisley-Cox *et al.,* 2008), PROCAM (Prospective Cardiovascular Münster) score (Assmann *et al.,* 2002), Reynold's score (Ridker *et al.,* 2007) and the WHO/ISH risk scoring charts. The total risk scores are based upon multivariate risk analyses of longitudinal cohorts that ascribe values to different risk factors (Dugee *et al.,* 2013) While they all aim at estimating CVD risk, different approaches were used for their derivations. For instance, the PROCAM study was based on a sample of industrial employees and is considered underpowered for risk estimation in women; the QRISK1 and QRISK2 are based on databases of general practice attendees and are therefore not random representative samples of the population and the Reynolds risk score is based on the prospective follow-up of 2 separate randomized controlled trials. Whereas the Framingham Risk Score, SCORE, QRISK 1 and 2 and PROCAM risk scores have been externally validated in other populations (than those from which they were generated) with good discrimination, the ASSIGN-

SCORE, WHO/ISH and Reynold's risk scores have not been assessed (Cooney *et al.*, 2009).

2.4.1 Framingham Risk Scoring Algorithm

The Framingham Risk Score is the best known and most commonly used risk score internationally and has been recommended by a number of national and international guidelines for CVD prevention (Genest *et al.*, 2009, Perk *et al.*, 2012, Goff *et al.*, 2013). It is a sex specific multivariable risk assessment tool that enables primary health care givers to identify high risk candidates for any and all initial atherosclerotic CDV events using measurements readily available at the clinic or office (D'Agostino *et al.*, 2008). It calculates the 10 year risk of CVD events and has sex, age, HDL- cholesterol, total cholesterol, systolic blood pressure, diabetic status, smoking status, and hypertension treatment status as the variables incorporated (Appendix A – D). Various sub-categories of each variable have sex specific points assigned to them. These points are then given to individuals based on their individual values for the various variables. The points for each person are then summed up to obtain a final score which is matched against the corresponding CVD risk to obtain that individual's total CVD risk. The total CVD risk refers to the likelihood of a CVD event occurring in the next ten years, expressed as a percentage. CVD risk is stratified as low risk (<10%), moderate risk (10 – 20%) and high risk (≥ 20%).

Regarding internal validity, the Framingham Risk Score performed well in terms of discrimination (ability to separate those who develop the endpoint from those who will not) and calibration (a measure of how closely the predicted outcomes agree with the actual outcomes). The c statistics ranged from 0.763 (95% confidence interval [C I],

0.746 to 0.780) in men to 0.793 (95% CI, 0.772 to 0.814) in women and the calibration X^2 statistics for the CVD prediction models were 7.79 for the women and 13.48 in men, indicating excellent goodness of fit (D'Agostino *et al.,* 2008). Furthermore, the Framingham Risk Score has also shown good discrimination in a number of external validation studies; the PRIME study (Empana *et al.,* 2003), the Dutch Study (Scheltens *et al.,* 2008), Physicians Health Study (D'Agostino *et al.,* 2013). The FRS has also been shown to apply well to African American populations (D'Agostino *et al.,* 2013). Unfortunately, information on the use of or transportability of this risk score in a purely African population is scarce. It is for these reasons that this risk score was chosen over the others for use in this study.

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2.5 CDV Risk Communication to Patients

CVD risk communication constitutes a key component in the risk prediction process. For any risk scoring algorithm to reach its full potential in the primary prevention of CVD, there must be effective communication of the implications of the predicted risk to patients as this improves patients' risk perception and preventive intentions. Various communication strategies have been described some of which include: the "positive strategy", "scare tactic" and "indirect strategy" (Bonner *et al*., 2014). The "positive" strategy aims at reassuring and motivating patients with a focus on achievable changes they can do to lower their risk. This strategy is usually used for patients who are concerned about their health, are willing to negotiate goals about lifestyle change and are at lower risk of CVD. The "scare tactic" aims at "scaring" people to take action and is thus used for people at high CVD risk particularly males and smokers who are

dismissive about their health and are unmotivated to change their lifestyle. The "indirect" strategy is used for people for whom CVD risk is not the main focus but who have some lifestyle risk factors and a high resistance to change (Bonner *et al*., 2014). In addition to the strategy, gain-framed messages (e.g presenting a survival benefit) as opposed to lost-framed messages (presenting a potential damage) have been shown to enhance understanding of risk and to increase the self-ability to prevent CVD (Stefan *et al*., 2011). The strategy of risk communication to be used within our Ghanaian context should therefore be informed by patient characteristics such as age, cultural background, education, psychosocial aspects and literacy as these go a long way to influence patients' understanding and perception of risk. Furthermore, discussing CVD risk with patients make them feel involved in the decision making process and this has been shown to have positive effects in terms of patient satisfaction, adherence to treatment and even better health outcomes (Stefan *et al*., 2011). Improving comprehension and effective risk communication is thus an important aspect in CVD risk prediction and prevention.

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CHAPTER THREE

MATERIALS AND METHODS

3.1 Study Methods and Design

A hospital based cross-sectional design was used. Hospital records of all patients who attended the Cardiac Clinic of KATH from January 2012 to July 2014 were reviewed. Information on sex, age, height, weight, diastolic blood pressure, systolic blood pressure, diabetic status, alcohol consumption (yes/no), smoking (yes/no), treatment of hypertension (yes/no), duration of hypertension, clinical diagnosis and laboratory results of the following tests: Total cholesterol, High Density Lipoprotein Cholesterol (HDL-C), Low Density Lipoprotein Cholesterol (LDL-C), Triglycerides and Fasting Blood Sugar, as at the last visit were collected and recorded on data capture sheets. The information obtained was then used to calculate 10year total cardiovascular risk using the most recent Framingham Risk Scoring algorithm (D'Agostino *et al*., 2008).

3.2 Study Area

The study was conducted at the Cardiac Clinic of the Komfo Anokye Teaching Hospital (KATH), where patients with cardiovascular related conditions like hypertension, stroke and heart failure are attended to and followed up. This site was used solely for its strategic position – serving almost all Ghanaians and especially those in the northern and middle belts of Ghana. KATH is a thousand bed capacity tertiary medical institution that receives referrals from eight of the ten regions of the country owing to its strategic location at the confluence of the country's transportation network otherwise known as the economic nerve of Ghana (www.kath.org). Kumasi is the capital of the Ashanti

region and the indigenous people are the Ashantes who constitute the largest of the various subgroups of the Akans. The dominant religion is Christianity; illiteracy levels are high; traditional healing facilities are commonly found in the localities; agriculture and trading are the main economic activities and "fufu" is the staple meal (www.ghana.gov.gh)

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3.3 Study Population

It consisted of persons 20 years and above attending the Cardiac Clinic who either were first registered at the clinic as at January 2012 onwards or were registered earlier and their hospital records showed regular attendance from January 2012 up to the time of data collection – July 2014.

3.4 Inclusion Criteria

- Adult 20 years and above
- Hospital record showed regular attendance as at January 2012 to date of data collection
- Patients whose records showed no established or history of CVD

3.5 Exclusion Criteria

- Patients whose records showed established or a history of any cardiovascular disease e.g all forms of stroke, heart failure, peripheral arterial disease
- Patients with missing values for any covariate
- Patients whose records failed to show regular attendance as from January 2012

3.6 Sampling

Simple random sampling was used. The following process was used. The hospital records of patients who met the inclusion criteria were selected and numbered. This constituted the sampling frame (800). Random numbers were generated using Microsoft Excel. Hospital records whose numbers matched those generated by Microsoft Excel were selected to be part of the study. The patients whose records were selected thus constituted the study population.

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3.7 Sample Size

A sample size of 441 was calculated using the formula below

$$n = \frac{Z^2 (pq)}{d^2} = 384$$

Where,

p (proportion of patients at risk of CVD)= 0.5

d (precision) = 0.05

Z (95% level of confidence) = 1.96

A non-response rate (absence of complete information) of 15% was calculated and added to the sample size to give a total of 441 subjects.

3.8 Data Collection

A data capture sheet (Appendix E) was used to extract information on sex, age, weight, height, systolic blood pressure, diastolic blood pressure, diabetic status, alcohol consumption (yes/no), smoking (yes/no), treatment of hypertension (yes/no) and duration of hypertension. Other information collected were; clinical diagnosis and laboratory results of the following tests: Fasting Blood Sugar, Total cholesterol, High Density Lipoprotein Cholesterol (HDL-C), Low Density Lipoprotein Cholesterol (LDL-C) and Triglycerides as at the last attendance.

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3.9 Study Variables

3.9.1 Dependent Variable

The outcome variable was 10 year cardiovascular disease risk

3.9.2 Independent Variables

The independent variables or predictors included: sex, age, body mass index (BMI), systolic blood pressure and diastolic blood pressure. Other predictors were; diabetic status, alcohol consumption, smoking, treatment of hypertension and duration of hypertension. The rest were; clinical diagnosis, Fasting Blood Sugar, Total cholesterol, High Density Lipoprotein Cholesterol (HDL-C), Low Density Lipoprotein Cholesterol (LDL-C) and Triglycerides

Table 3. 1 Study Variables

Specific Objective	**Dependent Variable**	**Independent variable(s)**	**Conceptual Definition of dependent variable**	**Scale of measurement**	**Indicators**	**Data Collection Method**	**Type of statistical analysis**
To investigate the prevalence of CVD risk factors	CVD risk factor prevalence	Sex Age Systolic blood pressure Diastolic pressure Body mass index Fasting blood sugar Total cholesterol LDL-C HDL-C Triglycerides	Factors that increase the risk of CVD	Percentage	Proportions, Frequency	Review of hospital records	Descriptive
To determine the distribution of CVD risk	CVD risk	Sex Age Hypertension Diabetes Dyslipidaemia Obesity Smoking Alcohol consumptio	probability of experiencing a CVD event in 10 years	Nominal: Low Moderate High	Proportions, frequency	Review of hospital records	descriptive test for trend

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		n Duration of hypertension Treatment of hypertension					
To determine the effect of each risk factor on CVD risk	CVD risk	Sex Age Hypertension Diabetes Dyslipidaemia Obesity Smoking Alcohol consumption Duration of hypertension Treatment of hypertension	probability of experiencing a CVD event in 10 years	Nominal: low Moderate High	Odds ratios	-	Univariate Ordered logistic regression Multivariate ordered logistic regression
To assess the sensitivity and specificity of the model	Sensitivity Specificity	-	Correct classification of CDV risk	Percentage	Area under the ROC curve	-	ROC analysis

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3.10 Risk Scoring using the Framingham Risk Score

Individuals were placed in the various categories for each variable as stipulated by the FRS (Appendix A and C) and assigned the corresponding sex specific risk score for that category. The various risk scores for each patient were then summed up to obtain the total risk score. This total risk score was matched against the corresponding CVD risk (Appendix B and D) to obtain the total CVD risk. This risk was then classified as low if the total risk was less than 10%, moderate if the total risk was between 10 – 20% and high if the total risk was greater than 20%

To test the sensitivity, specificity and discrimination of the model in stratifying risk, the model was subjected to post estimation statistics. The recommended cutoff of 20% (Selvarajah *et al*., 2014) was used for the analysis - wherein people were classified as either being at high risk (>20%) or moderate/low risk (< 20%).

3.11 Data Handling

Numbers were used to identify hospital records so as to ensure anonymity and confidentiality. Double entry of the data into Microsoft Excel was done to minimise errors. The database was password protected to prevent third parties from having access. The database was then exported to STATA version 11.1 for further cleaning and analysis.

The following international definitions and cutoffs (which are also consistent with Ghana Health Service standards) were used during the analysis:

- Hypertension was defined as the presence of persistent elevated systolic blood pressure ≥ 140mmHg and/or diastolic blood pressure ≥ 90mmHg and/or use of antihypertensive drugs and/or past medical history of hypertension (Chobanian *et al.*, 2003)

- Diabetes mellitus was defined as a random blood glucose level ≥11.1mmol/l and/ or fasting blood glucose level ≥ 7.0 mmol/l and/or use of insulin or an oral hypoglycaemic agent (WHO, 2006)

- Dyslipidaemia was defined as low levels of high density lipoprotein (HDL) cholesterol (≤ 1.03mmol/l) and/or high levels of total cholesterol (≥ 5.2mmol/l) and/or high levels of low density lipoprotein (LDL) cholesterol (≥ 3.6mmol/l) and/or high levels of triglycerides (≥ 1.7mmol/l) (GHS, 2014)

- Obesity was defined as a BMI ≥ 30kg/m^2 (GHS, 2014)

- Smoking was defined as "no" for never smokers and "yes" for ever and current smokers

- Alcohol consumption was defined as "no" for never drinkers and "yes" for ever and current drinkers

- Treatment of hypertension was defined as "no" for those not on any antihypertensive drug and "yes" for those taking any type of antihypertensive drug

- Age (years) which was collected as a continuous variable was transformed into 5year age groups and each age group was given a score as stipulated by the Framingham Risk Score (Appendix A, C).

- Body mass index (kg/m^2) was computed by dividing weight (kg) by the square of height in meters.

- Systolic blood pressure (depending on whether patient was on treatment or not), total cholesterol and HDL-C were also grouped in categories and each category was assigned a score. Subjects were also grouped according to their smoking and diabetic status and assigned scores (Appendix A, C).

- The total risk score (the likelihood for a CVD occurring in 10 years expressed as a percentage) for a subject was obtained by summing the points for the various covariates in the model. This total risk score was then stratified either as low risk <10%, moderate risk (10 – 20)% or high risk ≥20%

3.12 Data Analysis

Data was entered in Microsoft Excel and then exported to STATA (version 11) where it was cleaned and managed. Percentages were used to describe categorical variables while mean ± standard deviation were used for continuous variables. For comparison of risk factors between men and women, the chi square test was used for categorical variables and independent t test for continuous variables. The test for trend was used to determine the trend of the relationship between covariates and CVD risk. Univariate and multivariate ordered logistic regression was used to determine the effects of covariates

on CVD risk. The sensitivity and specificity of the model was assessed at the recommended cutoff point of > 20%. The discrimination of the model was assessed using the area under the ROC curve (AUROC). A value greater than 0.75 was considered good discrimination. All analyses were performed using STATA version 11.1 (STATA CORPORATION, College Station, Texas, USA) and p-values less than 0.05 were considered statistically significant.

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3.13 Ethical Considerations

Ethical approval to carry out the study was obtained from the Committee on Human Research Publication and Ethics (CHRPE) of the School of Medical Sciences, Kwame Nkrumah University of Science and Technology, Kumasi and Komfo Anokye Teaching Hospital, Kumasi – Ghana. Permission was also sought from the Head of the Medicine Directorate and the Head of the Cardiac Clinic at KATH to review patients' hospital records.

3.14 Limitations of Study

This present study had several limitations which include;

- It was a hospital based study so the findings cannot be generalized to the general population
- The study design solely involved the review of hospital records which for the majority of study participants failed to capture information on socio-economic status like level of education, occupation or demographic information like

marital status and place of residence. As a consequence therefore, these factors were not considered in the analysis

- The small numbers observed for some risk factors like smoking and alcohol consumption made their confidence intervals to be quite wide hence less precise estimates

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3.15 Strengths of Study

- The use of hospital records as opposed to self-reporting enabled the objective exclusion of persons with already established or a history of cardiovascular disease.
- The study population included those seen by physicians in routine practice as they come for follow up so the misclassification of persons as to their disease status e.g hypertension or diabetes such as is seen when measurements are only taken during one visit as is the case in surveys was minimized.
- Given that the study participants are already seeing a physician, it is easier to communicate their risk status to them hence making it more probable that the physicians will engage more aggressive strategies to reduce risk and the patients on their part may more likely personalize medical advice like physical activity, healthy diet or more drug compliance so as to either remain at low risk or reduce their risk which is unlike the case if the study was population based.

CHAPTER FOUR

RESULTS

4.1 Introduction

A total of 441 participants were enrolled for the study of which 53.5% were males. The average age was 54.35(12.9) years with women having a significantly higher mean age than men (p=0.004). Majority of study participants (30.8%) were in the age group (50 – 59) years and there was no significant difference in the age distribution according to sex (p=0.076). Overall, the study population was obese with a mean BMI of 30.26(5.94) kg/m^2. More males were overweight compared to females while more females were obese than males and this association was significant (p<0.001; p<0.001) respectively. The majority (61%) of study participants were on hypertension treatment and there was no significant association between treatment and sex (p=0.32). The mean systolic and diastolic blood pressure of the study population was 137.6(21.44) mmHg and 76.38(14.09) mmHg respectively. For the lipid profile, only total cholesterol and LDL-C showed a significant difference between males and females (p=0.02; p=0.02) respectively. The mean values of most of the main CVD risk factors were slightly raised or borderline compared to clinical thresholds (Table 4.1)

Table 4. 1 Characteristics of study Participants

Variable	Overall(n=441	Male(n=236)	Female(n=205)	P value
Age (years), mean (sd)	54.35(12.90)	52.69(12.47)	56.26(13.15)	0.004
Age group (years), n(%)				0.076
< 39	61(13.8)	41(17.4)	20(9.8)	
40 – 49	89(20.2)	49(20.8)	40(19.5)	
50 – 59	136(30.8)	72(30.5)	64(31.2)	
60 – 69	88(19.9)	46(19.5)	42(20.5)	
≥ 70	67(15.2)	28(11.8)	39(19.0)	
BMI(kg/m^2), mean(sd)	30.26(5.94)	28.44(5.07)	32.36(6.17)	<0.001
BMI category, n(%)				<0.001
Normal weight	76(17.2)	56(23.7)	20(9.8)	
Overweight	163(37.0)	105(44.5)	58(28.3)	
Obese	202(45.8)	75(31.8)	127(61.9)	
Hypertension duration, years, n(%)				0.846
<10	334(75.7)	179(53.6)	155(46.4)	
10 - 19	73(16.5)	41(56.2)	32(43.8)	
20 - 29	26(6.0)	12(46.2)	14(53.8)	
≥ 30	08(1.8)	4(50.0)	4(50.0)	
Treatment of hypertension,n(%)	269(61)	149(55.4)	120(44.6)	0.32
Systolic BP(mmHg), mean(sd)	137.66(21.44)	138.02(21.73)	137.23(21.15)	0.69
Diastolic BP(mmHg), mean(sd)	76.38(14.09)	77.25(15.09)	75.38(12.75)	0.16
TC (mmol/l), mean(sd)	5.19(1.25)	5.05(1.20)	5.34(1.29)	0.02
Triglycerides(mmol/l),mean(sd)	1.35(0.62)	1.32(0.63)	1.39(0.61)	0.26
HDL-C(mmol/l),mean(sd)	1.23(0.37)	1.22(0.36)	1.23(0.37)	0.65

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LDL-C(mmol/l), mean(sd)	3.32(1.04)	3.21(0.99)	3.44(1.08)	0.02
FBS(mmol/l), mean(sd)	5.81(1.88)	5.75(1.78)	5.87(2.00)	0.50

BP indicates blood pressure; TC – Total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; FBS, fasting blood sugar

4.2 Prevalence of Cardiovascular Risk Factors

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Hypertension was the most prevalent risk factor (72.3%) followed by obesity (45.8%) and diabetes (17.0%). While the occurrence of hypertension and diabetes was comparable between men and women (p=0.625), women were however significantly more obese than men (p<0.001). With regards to dyslipidaemia, most participants had low HDL-C (80.7%) and high LDL-C (64.4%) levels. Only total cholesterol was significantly different between the sexes with women having higher mean levels. Only men smoked and consumed alcohol (Table 4.2)

Table 4. 2 Prevalence of CVD Risk Factors by Sex

Variable	Overall (n=441)	Men (n=236)	Women (n=205)	P value
Hypertension, n(%)	319 (72.3)	173(73.3)	146(71.2)	0.625
Diabetes, n(%)	75(17.0)	36(15.3)	39(19.0)	0.293
Obesity, n(%)	202(45.8)	75(31.8)	127(61.9)	<0.001
Smoking, n(%)	11(2.5)	11(4.7)	0(0.0)	0.002
Alcohol, n(%)	19(4.3)	19(8.1)	0(0.0)	<0.001
High cholesterol	204(46.3)	98(41.5)	106(51.7)	0.032
High triglycerides	122(27.7)	61(25.9)	61(29.8)	0.36

High LDL-C	284(64.4)	147(62.3)	137(66.8)	0.321
Low HDL-C	356(80.7)	187(79.2)	169(82.4)	0.395

4.3 Prevalence of Hypertension Co-morbidities

Many study participants with the other risk factors also had hypertension. There was a significant association between hypertension and diabetes; hypertension and obesity; and hypertension and low HDL-C (p=0.002; p=0.001; p=0.02) respectively. Though the co-morbidity of hypertension with the other risk factors was common, the association was not significant (Table 4.3)

Table 4. 3 Prevalence of Hypertension Co-morbidities

Risk factor	**Overall**	**Hypertension n(%)**		**P-value**
		Yes (n=319)	**No (n=122)**	
Diabetes	75	65 (86.6)	10 (13.3)	0.002
Obesity	202	161(79.7)	41(20.3)	0.001
Smoking	11	7(63.6)	4(36.4)	0.51
Alcohol	19	13(68.4)	6(31.6)	0.69
High cholesterol	79	63(79.7)	16(20.3)	0.22
High triglyceride	28	22(78.6)	6(21.4)	0.27
High LDL-C	171	125(73.1)	46(26.9)	0.65
Low HDL-C	85	69(81.2)	16(18.8)	0.02

4.4 Total 10year CVD Risk Prediction

More than half of study participants (58.5%) had either moderate or high risk. (Table 4.2)

Table 4. 4 Ten-year Total CVD Risk Prediction

10year CVD Risk	n	Percentage (95% C I)
<10%	183	41.5 (36.9 – 46.1)
10 – 20%	124	28.1 (24.0 – 32.3)
>20%	134	30.4 (26.1 – 34.7)

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4.5 Distribution of CVD Risk

More men were at moderate to high risk and this trend was significant (p=0.003).There was also a significant trend in the distribution of CVD risk with hypertension, diabetes and smoking, wherein the majority of people who were hypertensive, diabetic and smoked were at moderate to high CVD risk compared to those who were or did not (p<0.001; p<0.001; p=0.027) respectively. As age increased so did CVD risk and the trend was significant (p<0.001). No one 44years and below was at high CVD risk but from 45years and above, increasingly more people were at high CVD risk. There was no significant increase in CVD risk with increasing BMI (p=0.492) and alcohol consumption (p=0.82). Nonetheless, there was an increase in CVD risk as the levels of the lipids increased and this trend was significant but for triglycerides for which the trend was only marginal (p=0.057). As the duration of hypertension increased so did CVD risk and this trend was significant (p<0.001). Many people who were on hypertension treatment were at moderate to high CVD risk compared to those who were not and this trend was significant (p<0.001) (Table 4.5)

Table 4. 5 Distribution of 10year CVD Risk

Variable	CVD RISK			p value for trend
	Low	**Moderate**	**High**	
Hypertension				P<0.001
No	92(75.4)	19(15.6)	11(9.0)	
Yes	91(28.5)	105(32.9)	123(38.6)	
Sex				P=0.003
Male	85(36.0)	66(28.0)	85(36.0)	
Female	98(47.8)	58(28.3)	49(23.9)	
Age group (years)				P<0.001
< 30	9(100.0)	0(0.0)	0(0.0)	
30 - 34	23(100.0)	0(0.0)	0(0.0)	
35 - 39	25(96.5)	1(3.5)	0(0.0)	
40 - 44	22(73.3)	8(26.7)	0(0.0)	
45 - 49	40(67.8)	18(30.5)	1(1.7)	
50 - 54	29(36.3)	38(47.5)	13(16.2)	
55 - 59	17(30.4)	21(37.5)	18(32.1)	
60 - 64	5(10.2)	14(28.6)	30(61.2)	
65 - 69	7(18.0)	11(28.2)	21(53.8)	
≥ 70	3(4.5)	13(19.4)	51(76.1)	
Diabetes				P<0.001
No	180(49.2)	107(29.2)	79(21.6)	

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Yes	3(4.0)	17(22.7)	55(73.3)	
Body Mass Index(kg/m^2)				P=0.492
Normal weight	37(48.7)	18(23.7)	21(27.6)	
over weight	64(39.3)	44(27.0)	55(33.7)	
obese	82(40.6)	62(30.7)	58(28.7)	
Total Cholesterol				p=0.001
Optimal	111(60.7)	63(26.6)	63(26.6)	
Borderline High	50(40.0)	40(33.6)	33(26.4)	
High	22(27.8)	19(24.1)	38(48.1)	
Triglycerides				p=0.057
Optimal	148(46.9)	78(24.4)	93(29.2)	
Borderline High	25(26.6)	34 (36.2)	35(37.2)	
High	93(29.2)	35(37.2)	6(21.4)	
Low Density Cholesterol				p=0.002
Optimal	74(47.1)	46(29.3)	37(23.6)	
Borderline High	51(45.1)	32(28.3)	30(26.6)	
High	58(33.9)	46(26.9)	67(39.2)	
High Density Cholesterol				p=0.018
Optimal	50(52.1)	26(27.1)	20(20.8)	
Borderline low	102(39.2)	73(28.1)	85(32.7)	
Low	31(36.5)	25(29.4)	29(34.1)	
Smoking				p=0.027

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No	181(42.1)	122(28.4)	127(29.5)	
Yes	2(18.2)	2(18.2)	7(63.4)	
Alcohol				p=0.820
No	174(41.2)	122(28.9)	126(29.8)	
Yes	9(47.40	2(10.5)	8(42.1)	
Treatment of hypertension				p<0.001
No	113(65.7)	38(22.1)	21(12.2)	
Yes	70(26.0)	86(32.0)	113(42.0)	
Hypertension duration(years)				p<0.001
< 10	168(50.3)	92(27.5)	74(22.2)	
10 -19	12(16.5)	19(26.0)	42(57.5)	
20-29	3(11.5)	10(38.5)	13(50.0)	
≥ 30	0(0.0)	3(37.5)	5(62.5)	

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4.6 Univariate and Multivariate Ordinal logistic regression Between CVD Risk Factors And 10year CVD Risk

Women were about 41% less likely to be at high CVD risk compared to moderate risk, or moderate risk compared to low risk than men without controlling for other covariates. For every 5years increase in age, the odds of having high risk compared to moderate risk or moderate risk compared to low risk increased by about 2 when uncontrolled for other

covariates. Without controlling for other covariates, the odds of having high CVD risk compared to moderate CVD risk or moderate CVD risk compared to low CVD risk was some 11 times greater for those who were diabetic compared to those who were not. For all lipids but for triglycerides, the odds of having high CVD risk compared to moderate CVD risk, or moderate CVD risk compared to low CVD risk was not significantly greater (or lower for HDL-C) for those who had borderline levels compared to those whose levels were optimal. People who smoked were roughly 4 times more likely of having high CVD risk compared to moderate CVD risk, or moderate CVD risk compared to low CVD risk than those who did not, without adjusting for other covariates (Table 4.6). After multivariate analysis, women were 94% less likely to be at high CVD risk compared to moderate risk, or moderate risk compared to low risk than men holding all other predictors constant. For every 5 year increase in age, the odds of having high risk compared to moderate risk, or moderate risk compared to low risk increased to about 3.7 adjusting for other covariates. The odds of having high CVD risk compared to moderate, or moderate compared to low risk increased for hypertension, diabetes and smoking adjusting for other covariates. However, though the odds increased, the very wide confidence intervals suggest less precise estimates. After controlling for other covariates, total cholesterol and triglyceride were no longer significant but were included in the final model because they are known important predictors of CVD risk.

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Table 4. 6 Univariate and Multivariate Ordinal logistic regression Between CVD Risk Factors And 10year CVD Risk

Variables	Crude OR (95%CI)	P value	Adjusted OR (95% C I)	P value
Sex				
Male	1.00		1.00	
Female	0.59(0.42 – 0.84)	0.003	0.06(0.03 – 0.13)	<0.001
Age group (years)	2.18(1.94 – 2.45)	<0.001	3.70(2.99 – 4.57)	<0.001
Hypertension				
No	1.00		1.00	
Yes	7.43 (4.66 – 11.87)	<0.001	20.53(9.53 – 44.25)	<0.001
Diabetes				
No	1.00		1.00	
Yes	11.02(6.32 – 19.21)	<0.001	68.94(27.38 – 173.6)	<0.001
Body Mass Index (kg/m2)				
Normal weight	1.00			
Over weight	1.43(0.86 – 2.38)	0.17		
Obese	1.25(0.76 – 2.05)	0.38		
Total cholesterol				
Optimal	1.00		1.00	
Borderline high	1.18(0.79 – 1.76)	0.42	0.94(0.38 – 2.37)	0.905
High	2.5(1.54 – 4.05)	<0.001	2.95(0.87 – 10.03)	0.083
Triglycerides				
Optimal	1.00		1.00	
Borderline high	1.84(1.21-2.8)	0.004	1.21(0.61 – 2.36)	0.585
High	1.11(0.56 – 2.21)	0.75	1.67(0.55 – 5.00)	0.361
LDL-C				
Optimal	1.00		1.00	
Borderline high	1.11(0.71 – 1.74)	0.64	3.69(1.70 – 8.01)	0.001
High	1.88(1.26 – 2.82)	0.002	8.96(3.03 – 26.56)	<0.001

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HDL-C				
Optimal	1.00		1.00	
Borderline low	0.91(0.58 – 1.43)	0.69	0.53(0.25 – 1.09)	0.087
Low	0.52(0.30 – 0.91)	0.021	0.12(0.05 – 0.30)	<0.001
Smoking				
No	1.00		1.00	
Yes	3.96(1.17 – 13.44)	0.027	17.57(2.48 – 124.09)	0.004
Alcohol				
No	1.00			
Yes	1.12(0.45 – 2.78)	0.81		
Hypertension Treatment				
No	1.00			
Yes	5.37(3.63 – 7.96)	<0.001		
Hypertension duration	2.57(1.91 – 3.44)	<0.001		

OR indicates Odds ratio; C I-confidence interval; LDL-C indicates Low-Density lipoprotein cholesterol; HDL-C-high-density lipoprotein cholesterol

4.7 Sensitivity and Specificity of the Framingham Risk Score

Overall, the model showed good discrimination at the cutoff of >20% with an AUC of 0.93; 0.95 for men and 0.92 for women. It correctly classified 85.3% of study participants with an overall sensitivity of 85.8% and specificity of 84.7% (Table 4.7)

Table 4. 7 Sensitivity, specificity and discrimination of the Framingham Risk Score

	Correctly classified (%)	Sensitivity (%)	Specificity (%)	AUC
Overall	85.3	85.8	84.7	0.93
Men	86.8	87.1	86.4	0.95
Women	86.0	81.6	89.7	0.92

CHAPTER FIVE

DISCUSSION

5.0 Introduction

Cardiovascular disease remains one of the topmost public health menaces in developing countries (Agyemang *et al*., 2012, Bosu, 2010, Mensah 2008 and de-Graft Aikins, 2007). Unfortunately, the condition is either lately recognized or poorly assessed. In most developing countries including Ghana, information on cardiovascular disease risk assessment is very scanty. This study sought to assess 10year cardiovascular disease risk among patients attending the cardiac clinic at Komfo Anokye Teaching Hospital, Kumasi using the most recent Framingham Risk Scoring algorithm.

5.1 Prevalence of cardiovascular disease risk factors

Hypertension, obesity and diabetes were found to be highly prevalent in the study population while smoking and alcohol consumption were the least. The high prevalence of hypertension in this study is similar to the finding of Owusu *et al* (2013) in Kumasi. Despite the high prevalence of hypertension, there was no significant difference in occurrence between men and women. The similar occurrence of hypertension in both men and women as seen in this study has also been reported in other studies in Africa (Addo *et al.*, 2013). However, the high prevalence of hypertension and hypertension co-morbidities as seen in this study is a cause for concern as it increases the total risk for CVD events such as seen in this present study. Never the less, awareness of hypertension has been reported to be better in Ghana than other African countries like Cameroon, Erithea, Burkina Fasso and Gambia (Bosu, 2010). This is supported by the

finding that about 61% of study participants were on treatment for hypertension. However, analysis revealed that those on treatment rather had a significantly higher mean systolic blood pressure than those not on treatment indicating poor control. This poor control may be attributed to probably low compliance to treatment and subsequent default from treatment due to high cost of drugs, ready access to herbal treatment or misconceptions that hypertension is curable.

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5.2 Prediction and Distribution of 10year total cardiovascular disease risk

The study revealed that more than half of the studied population of patients attending the Cardiac Clinic at KATH were at moderate to high 10 year CVD risk. This is similar to the findings of a hospital based study in Malaysia which also used the Framingham Risk Score where about 69% of patients attending the outpatient clinic at the USM hospital in Kelantan were at moderate to high risk (Norhayati *et al.*, 2013), and in Bulgaria where about 70% of patients being attended to by a general practitioner were at moderate to high risk, using the SCORE risk algorithm (Dyakova *et al.,* 2008). The finding of this study is however contrary to findings reported in Cuba, Mongolia, Cambodia and Jamaica where; 89.7%, 89.6%, 97% and 89.3% respectively of the general population were reported to be at low risk (Dugee *et al.*, 2013, Nordet *et al.,* 2013, Tulloch-Reid *et al.,* 2013). This discrepancy is probably because these studies besides being population based rather used the WHO/ISH risk scoring charts to estimate total CVD risk. Apart from not being calibrated and validated in most countries (Cooney *et al.,* 2010), the WHO/ISH risk scoring charts have been shown in comparative studies to classify the majority of individuals as low risk despite the high prevalence of CVD risk factors in

those populations (Norhayati *et al.,* 2013, Tulloch-Reid *et al.,* 2013, Selvarajah *et al.,* 2014). Furthermore, the present study was a hospital based study and so it is possible that those attending the Clinic already had a single raised or multiple CVD risk factors accounting for the values of the moderate and high 10year CVD risk observed.

There was a significant sex difference in the distribution of 10 year CVD risk, with males being more likely to be in the moderate and high risk categories despite the fact that women were significantly older than men. This sex difference in risk observed in this present study is confirmed by a recent study at KATH where more males died of stroke than females (53.5% vs 46.5%) (Agyemang *et al.,* 2012). While this finding is contrary to that of Tulloch-Reid *et al.,* (2013) in Jamaica where there was no sex difference in the distribution of 10year CVD risk, it however agrees with that of Dyakova *et al* (2013) in Bulgaria where men had a significantly higher risk than women. The decreased likelihood for women to be at moderate or high risk compared to men observed in this present study may be explained by the fact that the life time risk for developing a CVD event is higher for men than women and this is translated into the Framingham risk score, with men having a higher percentage of risk for the same risk score as women. Furthermore, CVD has been reported to develop 7 to 10 years later in women than men and that exposure to endogenous oestrogen during the fertile period of life delays the manifestation of atherosclerotic disease in women (Maas and Appelman 2010). Another probable reason for the observed low risk for women is the obesity paradox, where obese people have been reported to have more favorable cardiovascular outcomes for instance a decreased stroke risk than normal weight or lean people (Lavie *et al.,* 2009). Results from the present study show that more men were normal weight

than women (23.7% vs 9.8%) respectively while more women were obese than men (61.9% vs 31.8%) respectively. Furthermore, the observation that women did not smoke maybe another reason for their decreased risk as reported by this present study. Smoking is a recognized independent risk factor for CVD. Even though the 95% confidence intervals for smoking seen in this study were very wide implying less precise estimates, the effects of smoking on CVD risk were still significant compared to those who did not smoke. Given that all women did not smoke, it is thus probable that this translated into a lower risk for them.

As expected, the 10 year risk of a CVD event increased with age, with only 4.5% of people 70 years and over having a predicted 10 year incidence of CVD of <10% (low risk). This result is similar to those of Dyakova *et al* (2013) in Bulgaria, Dugee *et al* (2013) in Asia and Tulloch-Reid *et al* (2013) in Jamaica. This increased CVD risk with age can be explained by the physiologic changes that come with age such as thickening and dilatation of large arteries, large artery stiffness, and endothelial dysfunction that occur even in healthy people (Fleg and Strait 2012;Sung and Dyck 2012). These changes cause haemodeynamic alterations that make the old vulnerable to hypertension, stroke and Coronary heart disease (Soler and Ruiz 2010;Franklin and Wong, 2013). However, the moderate and high total CVD risk seen in the productive age groups of (50 -54) and (55 – 59) years is worrisome due to its negative economic and social impact. This implies if nothing is done to reduce the risk in these age groups, we can be sure of many more CDV events in the near future with its accompanying economic loss both in terms of income and production for those affected directly or those affected indirectly as care givers

The finding of a lack of association between increasing BMI and CVD risk is consistent with that of Tulloch-Reid *et al* (2013) in Jamaica. This lack of association between obesity and CVD risk may be due to the effects of obesity being mediated through other CVD risk factors like hypertension, diabetes and dyslipidaemia in the short term. It is for this reason obesity is frequently omitted from most CVD risk scoring algorithms (Berger *et al.*, 2010), despite it being an important CVD risk factor.

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5.3 Effect of risk factors on total cardiovascular disease risk

The findings of this study indicate that body mass index, alcohol consumption, borderline levels of total cholesterol, LDL-C, HDL-C and high levels of triglycerides did not have a significant effect on total 10year cardiovascular disease risk. The lack of a significant effect of body mass effect on total cardiovascular disease risk has also been reported by other studies (Tulloch-Reid *et al*., 2013, Berger *et al*., 2010), and has been attributed to the effects of obesity being mediated through other CVD risk factors. In as much as dyslipidaemia is a known risk factor of CVD, the lack of a significant effect of borderline levels of the lipids on total CVD risk suggest that lipid levels have to be "high" to be able to contribute to cardiovascular disease risk. The very wide 95% confidence intervals observed for most of the variables especially after multivariate analysis can partly be explained by the small numbers for some of the variables. For instance only 11(2.9%) of the study population smoked and 75(17.0%) were diabetic. However despite the wide confidence intervals implying less precise estimates, the accompanying p-values suggest a significant association of these variables with CVD

risk. Thus there is a need for a study with a larger sample size to be done so that the actual effects of these risk factors on CVD risk can be elucidated with more certainty.

5.4 Sensitivity, Specificity and Discrimination of the Framingham Risk Score

The sensitivity of the FRS to stratify risk as seen in this present study has also been reported in other studies. In Malaysia the Framingham risk score had an AUC of 0.75(95% C I 0.71 – 0.79) for men and 0.76(95% C I 0.70 – 0.82) for women (Selvarajah *et al.*, 2014); 0.73(95% CI 0.69 – 0.77) for men and 0.76(95%CI 0.72 – 0.80) for women in Australia (Carroll *et al.*, 2014); 0.79(95% CI 0.72 – 0.86) for men and 0.78(95% CI 0.71 – 0.85) in Spain (Artigao-Rodenas *et al.*, 2013) and 0.77(95% CI 0.74 – 0.81) for men and 0.82(0.79 – 0.85) in Tehran (Bozorgmanesh *et al.*, 2011). Unfortunately information on the discrimination or calibration of the Framingham risk score from Africa is scarce. The AUC obtained in this present study is higher than that reported by other studies. This can be partly explained by the fact that this study was a hospital based study that focused on people attending a cardiac clinic thus a higher likelihood for them to have a higher CVD risk. The majority of studies that have evaluated the discrimination of the Framingham risk score are either population based or if hospital based, the study population is patients attending the outpatient clinic/department and not specifically those attending a cardiac clinic.

CHAPTER SIX

CONCLUSION AND RECOMMENDATIONS

6.1 Conclusions

6.1.1 Prevalence of cardiovascular disease risk factors

Hypertension, obesity and diabetes mellitus were the most prevalent CVD risk factors while smoking and alcohol consumption were the least.

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6.1.2 Distribution of 10 year CVD risk

Men were significantly at moderate to high risk than women.

10year total CVD risk increased with increasing age, hypertension, diabetes, low density lipoprotein cholesterol and High density lipoprotein cholesterol in a significant trend.

Body mass index, triglycerides and alcohol consumption did not show a significant trend with CVD risk.

6.1.3 Effect of risk factors on total CVD risk

Sex, age, diabetes mellitus, LDL-C, HDL-C and smoking all had a significant effect on total 10 year CVD risk. Therefore, being male, aged, diabetic, a smoker and having high LDL-C levels coupled to low HDL-C levels increases total 10year CVD risk.

6.1.4 Sensitivity, Specificity and Discrimination of the Framingham Risk Score

The Framingham Risk Score showed good sensitivity, specificity and discrimination in this study group.

6.2 Recommendations

6.2.1 Ministry of Health and Ghana Health Service

10year total CVD risk estimation should be incorporated into routine practice in all hospitals in Ghana as stipulated by most guidelines on the prevention and treatment of CVDs, to enable consulting physicians know the risk status of their patients as this will improve patient care and management and in so doing better off treatment outcomes

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6.2.2 Health Facilities

Culturally sensitive techniques in communicating the risk status of patients by health professionals should be devised as this may aid the patients to optimize prevention/treatment strategies against CVD given that they often strike silently.

6.2.3 Areas for further research

Given that the existing total cardiovascular risk scores have been derived from predominantly white cohorts, there is a need for a large African based cohort study to either establish a risk score suited to our context or calibrate the existing risk scores to fit our context such that its usage will not underestimate or overestimate total CVD risk and in so doing mislead patients and health professionals.

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APPENDICES

APPENDIX A: FRAMINGHAM RISK SCORE: CDV RISK POINTS FOR WOMEN

Points	Age, y	HDL	Total cholesterol	SBP not treated	SBP treated	Smoker	Diabetic
-3				<120			
-2		60+					
-1		50 – 59			<120		
0	30 – 34	45 – 49	<160	120 – 129		No	No
1		35 – 44	160 – 199	130 - 139			
2	35 – 39	<35		140 – 149	120 – 129		
3			200 – 239		130 – 139	Yes	
4	40 – 44		240 – 279	150 – 159			Yes
5	45 – 49		280+	160+	140 - 149		
6					150 - 159		
7	50 – 54				160+		
8	55 – 59						
9	60 – 64						
10	65 - 69						
11	70 - 74						
12	75+						
Points allotted							Total

SBP indicates systolic blood pressure; HDL high-density lipoprotein cholesterol

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APPENDIX B: CDV RISK FOR WOMEN

Points	Risk, %
≤ -2	<1
-1	1.0
0	1.2
1	1.5
2	1.7
3	2.0
4	2.4
5	2.8
6	3.3
7	3.9
8	4.5
9	5.3
10	6.3
11	7.3
12	8.6
13	10.0
14	11.7
15	13.7
16	15.9
17	18.5
18	21.5
19	24.8
20	28.5
21+	>30

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APPENDIX C: FRAMINGHAM RISK SCORE: CVD RISK POINTS FOR MEN

Points	Age, y	HDL	Total cholesterol	SBP not treated	SBP treated	Smoker	Diabetic
-2		60+		<120			
-1		50 - 59					
0	30 – 34	45 – 49	<160	120 – 129	<120	No	No
1		35 – 44	160 – 199	130 - 139			
2	35 – 39	<35	200 - 239	140 – 159	120 – 129		
3			240 – 279	160+	130 – 139		Yes
4			280+		140 - 159	Yes	
5	40 – 44				160+		
6	45 - 49						
7							
8	50 – 54						
9							
10	55 - 59						
11	60 - 64						
12	65 - 69						
13							
14	70 - 74						
15	75+						
Points allotted							Total

SBP indicates systolic blood pressure; HDL high-density lipoprotein cholesterol

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APPENDIX D: CDV RISK FOR MEN

Points	Risk, %
≤ -3 or less	<1
-2	1.1
-1	1.4
0	1.6
1	1.9
2	2.3
3	2.8
4	3.3
5	3.9
6	4.7
7	5.6
8	6.7
9	7.9
10	9.4
11	11.2
12	13.2
13	15.6
14	18.4
15	21.6
16	25.3
17	29.4
18+	>30

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APPENDIX E: DATA CAPTURE SHEET

TOPIC: CARDIOVASCULAR DISEASE RISK ASSESSMENT AMONG PATIENTS ATTENDING THE CARDIAC CLINIC AT KOMFO ANOKYE TEACHING HOSPITAL, KUMASI – GHANA.

SECTION A: DEMOGRAPHIC INFORMATION

1. Sex: male ☐ female ☐
2. Age (completed years): _____
3. Highest level of education completed:
   No formal education ☐ primary school ☐ JHS ☐ SHS ☐ University
4. Marital status :
   Never married ☐ currently married ☐ separated ☐ divorced ☐ widowed ☐
5. Employment status
   Employed ☐ volunteer ☐ student ☐ retired ☐ unemployed
6. If employed occupation_____

SECTION B: LIFESTYLE PATTERNS

SMOKING

7. Do you **currently** smoke any tobacco products, such as cigarettes, cigars or pipes?
   Yes☐ no☐
8. In the past did you ever smoke any tobacco products?
   Yes☐ no☐
9. How long ago did you stop smoking _____

ALCOHOL CONSUMPTION

10. Do you currently consume any form of alcohol (beer, wine spirits, locally brewed)?

    Yes ☐ no ☐

11. Did you use to consume alcohol in the past?

    Yes ☐ no ☐

12. Have you stopped because of health reasons e.g negative impact on health or advice from doctor or other health worker

    Yes ☐ no ☐

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PHYSICAL ACTIVITY

13. Do you do any kind of physical work or exercise?

    Yes ☐ no ☐

14. Does your work involve vigorous-intensity activity that causes large increases in breathing or heart rate like (carrying or lifting heavy loads, digging or construction work) for at least 10 minutes?

    Yes ☐ no ☐

15. Does your work involve moderate-intensity activity, that causes small increases in breathing or heart rate such as brisk walking *[or carrying light loads]* for at least 10 minutes continuously

    Yes ☐ no ☐

16. Do you walk for at least 10 minutes continuously to get to and from places?

    Yes ☐ no ☐

17. How much time do you spend walking on a typical day?_____

CLINICAL INFORMATION

18. Use of any antihypertensive medication? Yes ☐ no ☐
19. Use of any oral hypoglycaemic drugs? Yes ☐ no ☐
20. Use of insulin? Yes ☐ no ☐
21. Use of any lipid lowering drug? Yes ☐ no ☐

PHYSICAL MEASUREMENTS

Variable	Measurement
Blood pressure (mmHg) Systolic BP Diastolic BP	
Weight (Kg)	
Height (cm)	
Body mass index (Kg/m^2)	

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BIOCHEMICAL MEASUREMENTS

Variable	Results
Fasting or random blood glucose(mmol/l)	
Total cholesterol (mmol/l)	
High density lipoprotein (HDL) cholesterol (mmol/l)	
Low density lipoprotein (LDL) cholesterol (mmol/l)	
Triglycerides (mmol/l)	
Uric acid (mmol/l)	