KWAME NKRUMAH UNIVERSITY OF SCIENCE AND TECHNOLOGY

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DEPARTMENT OF BIOCHEMISTRY AND BIOTECHNOLOGY

PREVALENCE OF CARDIOVASCULAR DISEASE RISK FACTORS AND DIETARY PATTERNS OF FOOTBALL PLAYERS OF SELECTED FOOTBALL CLUBS IN ASHANTI REGION OF GHANA

THIS THESIS IS PRESENTED TO THE DEPARTMENT OF BIOCHEMISTRY AND BIOTECHNOLOGY IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF MPhil DEGREE IN HUMAN NUTRITION AND DIETETICS

BY

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DECLARATION

I humbly and honestly declare that this study was wholly undertaken by me under the supervision of Prof. Kwabena Nsiah and Mr. Collins Afriyie Appiah, RD. This thesis, except portions where references were duly cited, is the outcome of my study.

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DEDICATION

I dedicate this work to my mother, Lucy Nkrumah for her love, care and support during my education and upbringing without whom I wouldn't have come this far. I also dedicate this work to my siblings, Patricia Gyamfua and Constance Afrifa for their love and support.

ABSTRACT

Physical activity promotes healthy living and prevents premature death. It provides increased benefits of health in individuals and can mitigate some chronic disease conditions. Cardiovascular disease risks during sports activities and exercises are low but in some instances of athletes who have undiagnosed pathological cardiac conditions, they could be at the mercy of tragic cardiovascular events. Healthy eating habits could be vital in the fight against cardiovascular diseases development. The aim of the study was to determine the dietary intake and the prevalence of cardiovascular disease risk factors among male football players of some selected football clubs in the Ashanti Region. A cross-sectional study was used and 95 football players of the premier, 1st and 2nd divisions teams were used for the study. The weight, height, BMI, percentage body fat, visceral fat and percentage muscle mass of the players were measured. The blood pressure and biochemical markers such as fasting blood glucose and lipid profiles of the players were also assessed. Dietary intakes of the players were collected using a 24-hour recall and food frequency questionnaire. SPSS version 23 was used to analyze the data collected. Out of the 95 players, 8 (8.4%) were goalkeepers, 30 (31.6%) were defenders, 38 (40.0%) were midfielders, while 19 (20.0%) were strikers. Generally, 8 (8.4%) players were overweight, 79 (83.2%) had a very high body fat and majority (92.7%) had high or very high muscle mass. All players had normal visceral fat. The prevalence of high blood pressure, high fasting plasma glucose among the study population was 8.4% and 10.5% respectively. Hypercholesterolemia, hypertriglyceridemia, low HDL and high LDL prevalence were 32.6%, 1.1%, 36.8% and 6.3% respectively. The goalkeepers had a higher mean total energy intake (2547.57±1198 kcal), carbohydrate intake (338.96±134.52g), protein intake (79.27±24.15g) and fat intake (99.32±80.01g). The dietary patterns of the players were poor as majority (63, 66.3%) either ate 2 meals or 1 meal a day. Poor dietary pattern could increase the risk of cardiovascular disease. Although the anthropometric parameters observed may not be an indication for the cardiovascular disease risk, the prevalence of high blood pressure, high fasting plasma glucose and hypercholesterolemia may put the players at an increased risk of cardiovascular disease.

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LIST OF ABBREVIATIONS

ADA: American Diabetes Association

AHA: American Heart Association

ASCVD: Atherosclerotic Cardiovascular Disease

BMI: Body mass index

BPLTT: Blood Pressure Lowering Treatment Trialist

CVD: Cardiovascular Disease

DALYs: Disability-adjusted life years

DASH: Dietary Approach to Stop Hypertension

DBP: Diastolic Blood Pressure

DM: Diabetes Mellitus

FFQ: Food Frequency Questionnaire

FPG: Fasting Plasma Glucose

GDP: Gross Domestic Product

HDL: High Density Lipoprotein

IFG: Impaired Fasting Glucose

IGT: Impaired Glucose Tolerance

KBTH: Korle Bu Teaching Hospital

KNUST: Kwame Nkrumah University of Science and Technology

LDL: Low Density Lipoprotein

MHO: Metabolically Healthy Obese

MONW: Metabolically Obese Normal Weight

NCEP: National Cholesterol Education Program

SBP: Systolic Blood Pressure

SCD: Sudden Cardiac Death

SIGN: Scottish Intercollegiate Guidelines Network

TC: Total Cholesterol

TG: Triglyceride

USDHHS: United States Department of Health and Human Services

WHO: World Health Organization

DEFINITION OF TERMS

Cardiovascular disease (CVD): It is a disease that affects the heart and blood vessels.

Diabetes mellitus (DM): It is the metabolic abnormality which is characterized by chronic high blood glucose levels which results from defects in either insulin action or insulin secretion or a combination of both.

Dietary Approach to Stop Hypertension (DASH) diet: It is a diet made up of fruits and vegetables and low fat dairy foods. It also comprises of fish, poultry, whole grains, nuts, and a low intake of sodium, sugary beverages and red meats

Disability-adjusted life years (DALYs): it is a measurement of deaths that occur at different age and disability.

Dyslipidemia: It is high levels of fasting blood total cholesterol (TC), Triglyceride (TG), low density lipoprotein (LDL) and a reduced high density lipoprotein (HDL).

Hypertension: It is the persistent high systolic blood pressure (≥ 140 mmHg) and/or diastolic pressure (≥ 90 mmHg in persons of 18 years of age and above.

Impaired fasting glucose (IFG): A fasting blood glucose level that ranges between 110mg/dL (6.1 mmol/l) and 125 mg/dL (6.9 mmol/L).

Impaired glucose tolerance (IGT): A 2-hour post-challenge glucose level ≥7.8 mmol/l (≥140 mg/dL) but < 11.1mmol/L (200 mg/dL)

Mediterranean diet: A diet made up of a high intake of complex carbohydrates, fruits and vegetables and legumes, in addition to a moderate intake of fish. It is normally eaten by people living around the Mediterranean Sea.

Risk factor: It is an assessable biological characteristic of an individual which comes before a well described outcome of the disease and can also predict that outcome and falls within the biological causative path.

Sudden Cardiac Death (SCD): Death which is of cardiac origin. It is unexpected and not associated to any organ disease or trauma.

CHAPTER 1

1.1 INTRODUCTION

Physical activity promotes healthy living and prevents premature death. It provides increased benefits of health in individuals and can mitigate some chronic disease conditions (Shiroma and Lee, 2010). Several benefits can be derived from physical activity and regular exercise. For example, according to Stern *et al.* (2011), cardiovascular disease risks during sports activities and exercises are low but in some instances of athletes who have undiagnosed pathological cardiac conditions, they could be at the mercy of tragic cardiovascular events (Thompson *et al.*, 2007).

According to Harmon *et al*. (2011), there has been increasing prevalence of sudden cardiac death and it was the primary contributing factor to death among student athletes of the National Collegiate Athletic Association in the United States (Harmon *et al*., 2011). Even though athletes are physically active, the preventive measures against the incidence of cardiovascular diseases do not rely on physical activity alone (Tucker *et al*., 2009). According to Selden *et al.* (2009) and Loucks (2004), there has been a continuous rise in the average weight of American football players. About 17.6% of the players are considered to be at risk of developing coronary heart diseases, while 35% are in the obesity range, through the assessment of body fat percentage and waist circumference (Loucks, 2004).

Healthy eating habits could be vital in the fight against cardiovascular diseases development, since cardiovascular diseases are the primary cause of death in the United States (Yu *et al.*, 2016). Obesity, high blood pressure, high cholesterol, insulin resistance, alcohol intake, smoking and physical inactivity are the risk factors for cardiovascular diseases. Fatty foods, particularly

saturated fats and trans fats are often implicated in the cause of obesity, hypertension and hypercholesterolemia (Elmer *et al*., 2006; Ogden *et al*., 2006,).

For the promotion of optimal performance and health, there has been the development of nutrition guidelines to help achieve such aims (Ray and Fowler, 2004). The requirements of athletes should be matched to their energy expenditure in their attempt to maintain healthy weight and muscle strength (Rodriguez *et al*., 2009; Shriver *et al*., 2013).

High lipid levels including serum cholesterol are related to high intake of saturated fat and cholesterol which predispose people to the future development of cardiovascular diseases (Reddy and Katan, 2004). This type of diet has been implicated in the development of hypertension and diabetes. It is thus important for athletes to have nutrient–dense and healthy foods so as to protect them from the risk of cardiovascular disease (Ridker *et al*., 2005; Rodriguez *et al.,* 2009).

1.2. PROBLEM STATEMENT

Worldwide, athletes and other sportsmen/women are viewed as a distinct group of people due to their exceptional physically active lifestyles. The possibility of athletes developing cardiovascular diseases or sudden death may seem contrary to expectation of society (Wasfy *et al.*, 2016).

Cardiac problems continue to occur among athletes often without symptoms and they often have considerable impact, both emotionally and socially on the athlete's immediate family and the public, at large (Schmied and Borjesson, 2014). In an attempt to understand, minimize or prevent sudden death in athletes, development of pre-participation screening strategies and formulation of disqualification criteria have been designed (Król *et al.,* 2016, Pelliccia and Corrado, 2017). Currently, the pre-participation screening program designed for athletes and specifically football

players in Ghana only focus on the medical assessment without emphasis on the importance of cardiovascular disease risk factors like diet and other lifestyles. There have been many studies which have assessed the cardiovascular disease risk in athletes in developed countries but such studies are very limited in Ghana, especially in football players at the premier league and lower division levels.

1.3. RESEARCH QUESTIONS

- What are the dietary intakes of the players?
- What is the prevalence of cardiovascular disease risk factors among football players?
- What are the lipid profiles and fasting blood glucose level of the players?
- Are players also susceptible to cardiovascular diseases due to their nutritional intake?

1.4. OBJECTIVES OF STUDY

1.4.1. MAIN OBJECTIVES:

To determine the dietary intake and the prevalence of cardiovascular disease risk factors among male football players of some selected football clubs in the Ashanti Region.

1.4.2. SPECIFIC OBJECTIVES

1. Assess the dietary intake of the players
2. Measure the anthropometric parameters (BMI, % body fat, % skeletal muscle mass and visceral fat) of the players
3. Assess the biochemical markers (Lipid profile and FPG) and blood pressure of the players.

1.5. SCOPE OF THE STUDY

This work basically, seeks to assess the prevalence of cardiovascular disease risk factors among football players. It will particularly focus on their anthropometrical, biochemical and dietary intake and how they affect their health and performance. The study focuses on football players from teams that are located in the Ashanti Region because of convenience and accessibility.

1.6. JUSTIFICATION

Cardiovascular diseases are the primary cause of sudden death among athletes. These deaths occur very often in football, basketball and rugby players in the United States. These sports involve intense levels of physical activity and highest levels of participation (Wasfy *et al*. 2016).

Because the incidence of deaths in athletes due to cardiovascular disease continues to increase and becoming common, it represents a substantive health problem and therefore the need for this study. Despite the concern on the cardiovascular health issues of football players, no study has been done on it in Ghana. This study will contribute to adding knowledge on the cardiovascular health status and the nutritional intake of football players.

CHAPTER 2

LITERATURE REVIEW

2.1. CARDIOVASCULAR DISEASES (CVDs)

2.1.1. GLOBAL BURDEN OF CVDs

Cardiovascular diseases (CVDs) are diseases that affect the heart and blood vessels. They include coronary heart diseases (CHD), peripheral arterial disease, stroke and aortic disease. The CVDs have become significant public health issue as the burden they cause has been increasing with time. CVDs killed about 17.5 million people in 2012, representing about 31% of all deaths globally (WHO, 2011). In 2012, ischemic heart disease was ranked as the number one cause of death and disability adjusted life years globally with an increase of 29% from 1990 (Lim, 2013).

CVDs are chronic diseases that develop throughout life. They usually get diagnosed at an advanced stage by the time symptoms had begun to appear. CVDs have been implicated as a major source of premature death worldwide and an estimated 80% of morbidities and mortalities occur in developing countries (Perk *et al.*, 2012). According to the WHO report on global health risks, CVD is ranked as the second cause of death after diarrheal diseases (WHO, 2009).

CVDs are estimated to raise the cause of disability-adjusted life years (DALYs) from 85 million in 1990 to more than 150 million by 2020 globally (SIGN, 2007). According to Bloom *et al.* (2012), the burden posed by CVD goes beyond mortality and morbidity to inflict a huge financial cost on households and countries at large. The Harvard School of Public Health and World Economic Forum, in their joint report had estimated the cost of non-communicable disease to be 47 trillion dollars over the period of 2010-2030 (Bloom *et al.*, 2012).

2.1.2. CARDIOVASCULAR DISEASE BURDEN IN AFRICA

In Africa, especially sub-Saharan Africa, CVD-associated deaths increased between 1990 and 2013 and it was the only region in the world where deaths that occurred as a result of CVD increased significantly (Roth *et al.*, 2015). In 2001, deaths that occurred as a result of CVD accounted for 9.2% of all deaths in Africa. It was also ranked as the leading cause of death among people aged over 45 years (Livesay, 2007). According to Mbewu and Mbanya (2006), the burden as a result of CVD will continue to rise and double by 2020 over the burden of 1990. According to Mendis and Alwan (2011), mortality in the world from non-communicable disease is likely to increase in all regions of the world in the next ten years of which the greatest will come from Africa.

Mbewu and Mbanya (2006) estimated the cost of cardiovascular disease to be about 300 billion US Dollars annually, making it equal to the gross domestic product (GDP) of the entire African continent. They again debated that this could cause serious problems to the socio-economic development of the African continent. This, in a way, will lead to most people on the continent not being able to access health care services.

2.1.3. THE BURDEN OF CARDIOVASCULAR DISEASE IN GHANA

CVD cases became the leading cause of death in 1991 and 2001 from seventh in 1953 and tenth in 1966, in Accra, Ghana and this has continued in the country as the major cause of death since then (Agyemang *et al.*, 2012). In 2008, 14.5% of deaths reported were said to be caused by CVDs, as compared to 13.4% from malaria. It also became the leading cause of institutional death and most occurring cause of death among non-communicable disease (Bosu, 2013). A study done to review autopsy cases from 2006 to 2010 at Korle Bu Teaching Hospital (KBTH) in

Ghana revealed that among 19,289 deaths, 22.5% resulted from CVDs (Sanuade *et al.,* 2014*).* According to the WHO (2012), the likelihood of a person between the ages of 30 and 70 dying from CVD, diabetes, cancer or chronic respiratory disease is 20% in Ghana.

2.2. INCIDENCE OF CARDIOVASCULAR DISEASES IN ATHLETES

It is normally assumed that the cardiovascular health of sportsmen and women is good. This is because athletes partake in activities that promote their health, especially the cardiovascular system. However, studies done over the years have given the indication that there are many diseases; idiopathic/genetic or lifestyle-related, that may have an impact on an athlete's cardiovascular system which can lead to death. Many high profile incidents in recent years have drawn attention to the subject of sudden cardiac death (SCD) among young athletes (≤ 35 years) (Evans *et al.,* 2008). For many years, the reason why cardiac arrest was happening in vigorous athletes who had previously attained an exceptional exercise performance without any indicators was a mystery. The reason was generally attributed to myocardial infarction, although the proof of ischemic myocardial necrosis was hardly reported (Corrado *et al*., 2007). It is now evident that the most frequent cause of sudden death during sports is a sudden ventricular tachyarrhythmia, as a result of a widespread range of cardiovascular diseases; either inherited or acquired (Corrado *et al*., 2007)

According to Maron (1998), sudden cardiac death could be of cardiac origin. It is unexpected and not associated to any organ disease or trauma (Maron, 1998). The causes of SCD among athletes are many and different in younger athletes, compared to older athletes. Younger athletes have a greater probability of having congenital cardiac effect, while older athletes may have a coronary event or myocardial infarction (Evans and Cassady, 2010). At present, there are over 20

genetic or acquired disorders that contribute to SCD in younger athletes however, each of these disorders does not account for more than 30 to 40% of SCD cases (Spooner *et al.*, 2001).

The rate at which sudden death happens in athletes during organized competitions is low, compared with the general population. In a retrospective study, the incidence of fatal occurrences in high school and college athletes aged 12-24 years, was estimated to be lower than 1 in 100,000 per year in the United States (Corrado *et al*., 2006). A prospective population-based analysis in Italy reported a three times higher prevalence in competitive athletes 12 – 35 years old (Corrado *et al*., 2006; Maron *et al.*, 2009). The prevalence of SCD was 2.3 per 100,000 among athletes, a ratio which was higher than non-athletes (0.9 per 100,000) in Padua, Italy (Van Camp *et al*., 1995). From the year 1966 up to 2004, 1,101 incidents of SCD in athletes who were younger than 35 years were reported (Bille *et al*., 2006). Out of these, 90% were of cardiovascular origin with 50% being anatomic congenital cardiac diseases and cardiomyopathies and 10% being early coronary atherosclerotic disease. Majority of cases (30%) happened in soccer, 25% in basketball and 15% occurring in runners (Bille *et al*., 2006).

The causes of SCD among competitive athletes include exercise-associated sympathetic stimulation, widespread hemodynamic variations and acute myocardial ischemia which lead to life-threatening ventricular arrhythmias (Corrado *et al*., 2007). In younger athletes, there is a wide range of cardiovascular disorders such as congenital heart disease, even though atherosclerotic coronary artery diseases make up the greater part of events in older athletes (Basso *et al*., 2001; Corrado *et al*., 2006). Cardiovascular cases are related to vigorous activities (Brukner and Brown, 2005). According to the American Heart association and the American College of Sport Medicine, high intensity vigorous exercise can transiently increase the event of cardiac death and acute myocardial infarction especially among those with structural cardiac

disease such as genetic or congenital cardiovascular disorders and atherosclerotic disease (Thompson *et al*., 2007).

2.3. CARDIOVASCULAR DISEASE RISK FACTORS

A risk factor is an assessable biological characteristic of an individual which comes before a well described outcome of the disease and can also predict that outcome and falls within the biological causative path (Balagopal *et al*., 2011). The traditional risk factors of CVD and their mode of action are known and have been documented (D'Agostino *et al*., 2008). Risk factors that contribute to cardiovascular disease include age, sex, high blood pressure (hypertension), smoking, dyslipidemia, diabetes, overweight or obesity, alcohol, physical inactivity and family history of heart disease, psycho-social factors and drug abuse (Yusuf *et al*., 2004). For the purpose of this study, the researcher reviewed literature concerning hypertension, dyslipidemia, diabetes mellitus, overweight, obesity and nutrition.

2.3.1. DYSLIPIDEMIA

Lipids perform important functions in the body. In spite of their enormous contributions to the functions of the body, abnormally high levels of lipids and their lipoprotein carriers in the blood contribute greatly to the development of atherosclerosis (Leon and Bronas, 2009).

Dyslipidemia is high levels of fasting blood total cholesterol (TC), triglyceride (TG), low density lipoprotein (LDL) and a reduced high density lipoprotein (HDL) (Leon and Bronas, 2009). Dyslipidemia is a key CVD risk factor and is mostly seen in obese people, type 2 diabetics, people who are insulin resistant and also those who have metabolic syndrome.

2.3.1.1. LIPOPROTEINS

Lipoproteins are complexes of cholesterol, proteins and phospholipids (Sanossian *et al.*, 2007). They are classified as High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL) and Very Low Density Lipoprotein (VLDL) (NCEP ATP III, 2001). Elevated HDL, unlike LDL has a protective effect and evidence has shown that there is a 2-3% increased risk of CHD if there is a decrease in HDL by 1mg/dL, while a 1 mg/dL increase will lower the risk of death from CHD by 6% (Genest, 2008). A prospective study done in Quebec, Canada, showed that serum HDL concentration was an independent coronary heart disease and ischemic heart disease predictor. It further reported that reduced plasma HDL concentrations has a better impact on atherogenic index (total cholesterol/HDL cholesterol ratio) than raised LDL cholesterol (Després *et al.*, 2000)

Even though all the components of dyslipidemia have been shown to increase CVD risk, the strongest predictors are elevated LDL levels and total cholesterol (Miller, 2009; Musunuru, 2010). According to Musunuru (2010), this may be due to the fact that small dense LDL has a larger susceptibility to oxidation and can initiate the inflammatory process occurring in the vascular endothelium which underlies atherosclerotic diseases. While many studies have affirmed the contributory role of small dense LDL particles in the pathogenesis of CVDs (Toshima *et al.*, 2000), Cromwell and Otvos (2004) reported that classifying LDL cholesterol as small and large underrates and faults that perception since LDL Cholesterol is only a measure of the amount of the cholesterol in LDL.

2.3.1.2. TRIGLYCERIDES

Triglycerides (TG) are related to the atherosclerotic plaque formation and progression. Through indirect mechanisms, TG has been shown to drive atherogenesis especially those that bind to the

artery wall and those that are involved in lipolysis (Chapman *et al*., 2011). Postprandial hypertriglyceridemia occurs when there is a reduced breakdown and overproduction of triglyceride-rich lipoprotein as chylomicron. It is a result of the tendency of genetic disparities and conditions such as obesity and insulin resistance (Borén *et al*., 2014). Hypertriglyceridemia leads to a decrease in HDL and a buildup of small dense LDL. Furthermore, it may also promote the development of atherosclerosis by processes such as the release of free fatty acids and pro-inflammatory cytokines production (Tenenbaum *et al*., 2014). Independent relationships between high triglyceride and CVD risk are debatable (Miller *et al.,* 2011). The main problem in separating the effect of high triglyceride on CVD is the fact that high TG levels are mostly related with associated modifications in HDL and LDL, even though majority of studies reported a significant direct relationship between TG and opposing outcomes (Bansal *et al*., 2007). According to Schwartz *et al*. (2005), this relationship sometimes becomes insignificant after multivariate modification of other lipids and weight-associated parameters. For example, high TG levels were related to an increase in risk of CHD after modifications in age and sex but this relationship was gotten rid of after an added adjustment for HDL and non-HDL cholesterol in a meta-analysis (Schwartz *et al*., 2005).

2.3.1.3. TOTAL CHOLESTEROL

Cholesterol is a lipid which exists in cell membranes. It is the major precursor of sex hormones, bile acids, Vitamin D and cortisol (NCEP ATP III, 2002). High levels of plasma LDL cholesterol lead to the retention of cholesterol in the arterial walls and formation of foam cells at the intima of the arteries. Occlusive atherosclerosis is then developed with angina pectoris and/or plaque rupture with coronary heart disease (Nordestgaard *et al.,* 2013).

2.3.1.4 DYSLIPIDEMIA IN ATHLETES

Lipid profiles of athletes are likely to be good and may result in reduction of atherosclerotic cardiovascular disease (ASCVD) (Durstine *et al*., 2001). In general, compared to their sedentary counterparts, athletes have a 40-50% higher HDL-C, 5-10% lower LDL-C and a 20-40% lower TG, with a lower prevalence of hypercholesterolemia (total cholesterol >200mg/dL) and hypertriglyceridemia (TG >150mg/dL) (Thompson *et al*., 1980; Deshaies and Allard, 1981). Athletes like soccer players and rugby players endure intense physical training which frequently exceeds the energy expenditure requirements which is normally recognized to alter lipids and lipoproteins. According to Durstine *et al*. (2001), a relationship exists between the training intensity and blood lipids. This implies that exercising at low training volume can significantly change blood lipids levels (Durstine et al., 2001). Even though athletes undergo intense level of physical exercise with significant effect on lipid and lipoprotein carriers, evidence from a number of studies have reported an increase in CVD risks associated with dyslipidemia (Durstine *et al.*, 2001; Buell *et al.*, 2008; Chang *et al.*, 2009). Even though lipoprotein and lipid levels of athletes have shown decreased risk for the pathogenesis of CVD in comparison to their sedentary colleagues, there is still a limited evidence to show that physically active people have lower levels of LDL-C than those who are sedentary (Durstine *et al*., 2000).

2.3.2. HYPERTENSION

High blood pressure is a strong indicator of a risk to CVD (BPLTT, 2013). It can cause structural transformation of the heart such as cardiac fibrosis and left ventricular hypertrophy. These changes result in an increase in the stiffness of the left ventricles which can cause a dysfunction

in the diastole and also elevations in diastolic pressure of the left ventricles (Drazner, 2011; Owusu *et al*., 2014).

Hypertension is the persistent high systolic blood pressure (≥ 140 mmHg) and/or diastolic pressure (≥ 90 mmHg in persons of 15 years of age and above (Gibbons *et al.*, 2002). The presence of hypertensive retinopathy, the usage of anti-hypertensive drugs and a history of high blood pressure occurring alone or in combination can also be used to describe hypertension (Owusu *et al.*, 2014).

Hypertension has also been shown to be the major indicator to the risk of stroke (Jauch *et al*., 2013). Epidemiological data gathered show that a 10 mmHg rise in systolic blood pressure can result in a 40% increase in the risk of stroke. The risk doubles with every 7.5mmHg increase in diastolic pressure (Mensah, 2008).

Chronic aerobic exercise has been suggested as a primary prevention of hypertension and CVD events. The positive effects of exercise may be due to the mediation by a number of ways such as changes in lipid profile, carbohydrate metabolism, fibrinogen and neuro-hormonal release (Hagberg *et al*., 1989; Tolfrey *et al*., 2000). There is strong evidence that shows that ambulatory blood pressure (ABP) is more related to CVD events than clinical blood pressure. This is because ABP monitoring provides data on BP variability which is not available from an office measurement. A high blood pressure variability obtained from a 24 hour ABP monitoring has been related to an increase in CVD risk and a low night blood pressure fall has also been associated with poor prognosis (Schwartz and Hirth, 1995; Hoshide *et al*., 2003)

Chronic physical activities and training have been implicated in the reduction of blood pressure yet high blood pressure is one of the major abnormalities in athletes and continues to be the

major CVD condition found in the athletic populace (Leddy and Izzo, 2009). Though the general incidence of hypertension in persons who are physically active is 50% lower compared to the general population, yet black, obese, older, diabetic athletes or those who have chronic kidney disease more frequently develop essential hypertension than their counterparts who do not have such conditions (Lehmann *et al.*, 1990; Harmon *et al*., 2011). According to Hanson *et al.* (1994), the prevalence of hypertension in adolescent athletes is about 80% so it should be monitored closely regardless of the physical fitness level. The incidence of secondary hypertension is almost the same as in the general population, even though disabled athletes with spinal cord injuries may have intense, intermittent hypertension which may be due to autonomic or bladder dysfunction (Hanson *et al.*, 1994; Schmid *et al.*, 2001). Several studies have shown an increase in the mass of the left ventricles and atria of athletes and so there is a possibility that elevated blood pressure may be a causal factor that can also be implicated in the risk of atrial fibrillation in athletes (Manolis *et al*., 2012; Berge *et al.*, 2013)

2.3.3. DIABETES MELLITUS

Diabetes mellitus is a metabolic abnormality which is characterized by chronic high blood glucose level and a disorder in carbohydrates, protein and fats metabolism which results from disturbances in either insulin action or secretion or combination of both (Alberti and Zimmet, 1998). The American Diabetes Association also described diabetes mellitus as the metabolic disorder which is characterized by chronic high blood glucose which results from defects in either insulin secretion, insulin action or combination of both (ADA, 2010). Diabetes mellitus is noted as a risk factor for CHD and stroke (Spencer *et al.*, 2008). A similar relationship exists for non-diabetic persons who have elevated blood glucose levels which indicate glucose intolerance (Gerstein and Yusuf, 1996). The onset of diabetes whether early or late has a relation with a

significant increase in risk of CVD events. People who have mean diabetes duration longer than 8 years have been shown to be at higher risk, as compared to people with shorter duration of diabetes (Wannamethee *et al.*, 2011).

The chronic hyperglycemia of diabetes has been related to long-term impairment and malfunction of different organs such as kidneys, eyes, heart, nerves and the blood vessels. Diabetes is characterized by symptoms such as polyuria, weight loss, polydipsia, polyphagia and sometimes blurred vision (ADA, 2010). The increase in CVD risk among diabetics has been attributed to several mechanisms such as insulin resistance, poor glycemic control, oxidative stress, abnormalities in the endothelium of vascular smooth muscle cells, low grade inflammation and platelet function (Gomes, 2013; Paneni *et al.*, 2013). Diabetes mellitus is associated with defects of the autonomic nervous system. Persons who have diabetic autonomic nephropathy have an increased risk of sudden cardiac death, leading to an increased rate of cardiovascular mortality cases (Gomes, 2013)

The World Health Organization and the American Diabetes Association, in a consultation, identified impaired fasting glucose and impaired glucose tolerance as criteria for diagnosing diabetes mellitus (WHO/ADA, 1999). Impaired glucose tolerance (IGT) was explained as a 2-hour post-challenge glucose level < 11.1mmol/L (200 mg/dL) but ≥ 7.8 mmol/l (140 mg/dL). Impaired fasting glucose (IFG) was also defined as fasting glucose level between the range of 110mg/dL (6.1 mmol/l) and 125 mg/dL (6.9 mmol/L) (Gavin III *et al.*, 1997; WHO/ADA, 1999).

2.3.4. OVERWEIGHT AND OBESITY

Overweight and obesity are used to characterize abnormal or excess accumulation of fat (Marinou *et al.*, 2010). Obesity arises when caloric intake is higher than the energy required by

the body for growth and physical activity. Obesity could be as a result of genetic factors, physical inactivity and unhealthy eating habits (Escalona *et al.*, 2004). Worldwide, there has been 200% rise in the incidence of overweight and obesity (WHO, 2013). Nearly 2 billion adults were estimated to be overweight or obese in 2014 (WHO, 2013). Overweight and obesity were projected to have caused 3.9 million deaths and 3.8% of DALYs (WHO, 2013). In Ghana, 43% of adults were estimated to be either overweight or obese (Ofori-Asenso *et al.*, 2016). Obesity causes chronic conditions such as CVD, insulin resistance and diabetes mellitus. Obesity and overweight can also occur simultaneously with conditions such as dyslipidemia and metabolic syndrome (Singh *et al*., 2011). They can cause chronic stimulation of the innate immune system, resulting in the inflammation of white adipose tissue and then subsequently leading to diabetes and insulin resistance (Bastard *et al*., 2006).

Body mass index (BMI) classification of person into overweight and obese was proposed by WHO Expert Panel Committee in 1995. A BMI of <18.5kg/m^2 (Underweight), 18.5-24.9kg/m^2 (Normal) 25.0-29.9kg/m^2 (Overweight) and ≥30.0 kg/m^2 (obese) was proposed by the committee. Persons with a BMI 30.0-34.9 kg/m^2, 35.0-39.9 kg/m^2, ≥40.0 kg/m^2 were also classified into class I or mild obesity, class II or moderate obesity and class III or extreme obesity respectively (WHO, 1995). The use of BMI classifications presents valuable information about body fatness. It compares the weight of individuals in the population and the classification of persons and groups who have high risk of morbidity and mortality (Kopelman, 2000).

According to Prentice and Jebb (2001), classification of obesity should be based on excess body fat accumulation. A high body fat percentage and its distribution are shown to be closely related to increased mortality and morbidity. Remarkably, not all persons who are obese exhibit metabolic and cardiovascular risk factors and also not all lean persons do exhibit a metabolic

disease-free profile (Karelis *et al.*, 2004). Many studies began to look into the reality of the distinct obesity subtypes around the year, 1980 (Ruderman *et al.*, 1998). A unique group of obese persons have been classified who seem to be more resilient and protected from metabolic conditions related to obesity. One of these groups known as metabolically healthy obese (MHO), do exhibit normal to high levels insulin sensitivity and have no cardiovascular disease, even though they have large amounts of fat mass (Karelis *et al.*, 2004). Another group, metabolically obese, normal-weight (MONW) are those whose BMI are normal but show a collection of disorders related to obesity such as high blood pressure, high levels of triglycerides and hyperglycemia (Karelis *et al.*, 2004; St-Onge *et al.*, 2004).

2.4. NUTRITION AND CARDIOVASCULAR DISEASES

The link between cardiovascular disease and nutrition has been investigated intensively for more than a century now. An atherosclerosis in rabbits was produced by Ignatowski in 1908 with a diet which was high in saturated fat and cholesterol and similar lesions were produced when the rabbits were fed with cholesterol alone (Hu and Willett, 2002). Meanwhile, researches done after that period also found that an increase in serum cholesterol predicted an increase in the risk of coronary heart disease among humans. The findings brought about the classic diet-heart theory which implicates saturated fat and dietary cholesterol in the pathogenesis of atherosclerosis and coronary heart disease (Gordon, 1988).

According to Lichtenstein *et al.* (2006), many studies have concentrated on the individual nutrients in food, even though it is recognized that multiple dietary factors have effects on the risk of developing CVD. Epidemiological studies have also examined the health benefits of the whole diet; both clinical trials and observational studies. These studies have reported that healthy dietary habits are linked with a significant decrease in CVD risk factors (Knoops *et al.*, 2004).

The focus on whole diet also guarantees energy balance and nutrient adequacy. Therefore, instead of focusing on single food or nutrient, individuals must aim at enhancing their whole diet (Thompson and Veneman, 2005). In relation with this opinion, the American Heart Association (AHA) also recommended that people consume a range of fruits and vegetables, fat-free and low-fat dairy products, poultry, lean meats and fish especially oily fishes and whole grains (Lichtenstein *et al*., 2006).

2.4.1. FRUITS AND VEGETABLES

According to Belanger *et. al.*, (1976) in the Nurses' Health Study, high intake of fruits and vegetables, particularly green leafy vegetables are linked to reduction in the risk of major chronic diseases. It also recommended the intake of five or more servings of fruits and vegetables in a day (Hung *et al*., 2004). A meta-analysis of cohort studies showed that in comparison, individuals who consume less than 3 servings of fruits and vegetables have a lower risk reduction than individuals who consume more than 5 servings and 3-5 servings. Individuals with more than 5 servings have CVD risk reduction of approximately 17%, whereas those who consume 3-5 servings have a lesser and a marginal reduction of 7%. These outcomes present a strong support for the recommendation to eat more than 5 servings of fruits and vegetables (He *et al*., 2007) but a study in Japan reported no association between vegetable intake and CVD risk but confirmed a substantial inverse association between fruits and CVD risk (Takachi *et al.*, 2008).

2.4.2. WHOLE GRAINS

Current dietary recommendations propose an increase in the consumption of grain products to avoid CHD but they do not stipulate the amount of whole grains to be consumed (AHA, 1996). The effect of whole grain intake on the risk of CHD may probably depend on the presence or

absence of many components and their relationships. Furthermore whole grains provide essential fatty acids and protein and may contain some unique and useful combinations of antioxidants, phytochemicals, micronutrient and fiber (Slavin *et al*., 1997). More consumption of several components of whole grains like folate and vitamin E has been linked with CHD risk reduction independently but even after adjustment of these constituents, a significant inverse association of CHD risk to whole grain occurred (Rimm, 2001). The US Department of Agriculture's Food Guide Pyramid recommended the consumption of 6-11 servings of grain product but it was silent on the amount of whole grain to be taken (USDHHS, 1995). In a cohort study conducted in the United States, women who consumed an amount of 2.5 servings per day experienced more than 30% lower risk of CHD than those who consumed about 0.13 serving per day (Liu *et al.*, 1999). The association between low CHD risk and higher consumption of whole grain food in the cohort appeared to be continuous and so increasing the rate of whole grain food intake should be plausible and may result in substantial benefits in reducing the risk of CHD, even though several grains in the United States are refined and processed (Liu *et al.*, 1999).

2.4.3. FISH CONSUMPTION

An inverse association has been reported between omega-3 fatty acids, fish consumption and CHD risk. CHD risk in women was 29%, 31%, 34% and 21% lesser for fish intake once per week, 2-4 time per week, more than 5 times per week, 1-3 times per month respectively compared to women who seldom take fish (Hu *et al*., 2002). The consumption of fish has been found to reduce SCD. In a population-based, nested, case-control study, a negative association was shown between the risk for SCD and fish consumption (two fatty fish meal per week which is comparable to 5.5g of omega-3 fatty acids per month) and also linked to a reduction of 50% in primary cardiac arrest (Siscovick *et al.*, 1995). Omega-3 fatty acids can prevent or be used to

manage chronic diseases (dyslipidemia, type 2 diabetes, insulin resistance, hypertension, etc.). Eicosapentaenoic acid and docosahexaenoic acid present in fish oils and fish can change the membrane phospholipid components of the cells, control transcription factor activity when added to diet (Takahashi *et al.*, 2002)

2.4.4. MEDITERRANEAN DIET

The Mediterranean diet decreases the risk of chronic and non-communicable diseases. This diet is common in people living around the Mediterranean Sea. It includes a high consumption of complex carbohydrates, fruits and vegetables and legumes, in addition to a moderate intake of fish. It also includes the intake of olive oil as the key source of lipids and a consumption of low-to-moderate quantity of red wine (Bach *et al.*, 2006). According to Bach *et al.* (2006), the Mediterranean diet has a total fat ranging from 25% to 35%, with a saturated fat of 7-8%. According to Kris-Etherton *et al.* (2001) in the Lyon Diet Heart Study, there is a significant reduction of CVD when adherence to Mediterranean diet is increased. A Mediterranean diet with unrestricted energy which has either additional olive oil or nuts resulted in substantial decrease in the risk of three key cardiovascular events (Estruch *et al.*, 2013). Olive oil, a major constituent of the Mediterranean diet, is a rich source of monounsaturated fatty acids. All the lipophilic constituents of the fruits; alpha-tocopherol, and the phenolic compounds which have powerful antioxidant and anti-inflammatory properties are all retained in the virgin olive oil (Beauchamp *et al*., 2005). Mediterranean diet includes tree nuts, which also have a positive fatty acid profile and a rich supply of bioactive compounds such as fiber, antioxidants, phytosterols and folic acid that may positively reduce CHD risks. Increased nut intake has also been associated with a reduction in CHD rates in prospective studies (Kris-Etherton *et al*., 2001). According to Estruch *et al*. (2006), high risk individuals showed improved blood pressure levels, good lipid profile,

low concentrations of inflammatory molecules and reduced insulin resistance after they increased their intake for Mediterranean diet when given supplementation with virgin olive oils or mixed nuts and nutritional education.

2.4.5. DIETARY APPROACHES TO STOP HYPERTENSION (DASH) DIET

According to Appel *et al.* (1997), the Dietary Approaches to stop Hypertension (DASH) diet is reported to show significant reduction in blood pressure among all populations of the world. It is made up of low fat dairy foods and fruits and vegetables. It also includes whole grains, fish, poultry, nuts, and a reduced intake of sugary beverages, sweets and red meats (Appel *et al.*, 1997). According to the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure, the DASH diet has undeniably shown that it is the overall dietary pattern that has the ultimate influence on blood pressure, instead of a single component of the diet (Chobanian *et al*., 2003). It has also been shown to decrease low density lipoprotein cholesterol (Obarzanek *et al*., 2001). When the DASH diet macronutrient component was altered by replacing the carbohydrate component with either unsaturated plant fat or protein in the Optimal Macronutrient Intake Trial to Prevent Heart Disease (OmniHeart) trial, it was found that there was a further decrease in blood pressure and improvement in lipid profile (Appel *et al*., 2005). The DASH diet again reduced the total cholesterol and LDL cholesterol by -0.16 and -0.33 mmol/L respectively but it also lowered HDL cholesterol by -4 mg/dL (Obarzanek *et al*., 2001, Sacks *et al*., 2001). According to Fung *et al.* (2008), even diets similar to DASH diet pattern which included a high intake of whole grains, fruit and vegetables, nuts, moderate legume and low-fat consumption and a reduction in processed and red meats and sodium resulted in a substantial decrease in CHD risk.

According to Lin *et al.* (2004), the DASH diet is rich in calcium, potassium, magnesium and fiber and low in sodium because it is high in fruits, vegetables, whole grains, nuts, low-fat dairy products, poultry, and fish, and low in red meat, fats, sugar containing beverages and sweets. Clinical trials have demonstrated that sodium reduction lowers blood pressure (Cutler *et al*., 1997). Sacks *et al*. (2001), reported that, when the sodium intake was reduced from high to low with the DASH diet, systolic blood pressure reduced significantly in people with hypertension. Moreover, several studies investigated in hypertensive rats have shown that a high sodium diet is related to pressure-independent changes that occur in nonmuscle myosin, proteoglycans and fibronectin of vascular smooth muscle cells. These changes are in harmony with the development of a primary secretory phenotype of smooth muscle cells (elastin and collagen) in the presence of a high sodium diet (Savolainen *et al*., 1996). According to Haddy (2006), increased dietary sodium intake increases plasma Na^+-K^+ pump inhibiting actions especially in people with low renin. An increase in dietary sodium also increases potassium excretion by the kidneys which results in a fall in plasma potassium levels (Haddy, 2006). When plasma potassium level decreases, vasoconstriction occurs in the vascular beds leading to hypokalemia. This is in part responsible for hypertension (Pamnani *et al*., 2000). The US Department of Health and Human Services and the US Department of Agriculture in their 2010 dietary Guidelines have recommended a daily sodium intake of less than 2300 mg per day and an even lesser aim of 1500 mg per day for specific subpopulations (Boon *et al*., 2010).

Population studies have indicated an inverse association between potassium intake and hypertension and stroke (Whelton, 2003). The INTERSALT study estimated that a reduction in potassium excretion by 50 mmol/day was related to an increase of 3.4 mmHg systolic blood pressure and 1.9 mmHg diastolic blood pressure (INTERSALT, 1994). According to Pratt

(2005), high dietary potassium intake as seen in the DASH diet has been shown to wield a dominant, dose-reliant inhibitory effect on sodium sensitivity and an increase in potassium intake can even eliminate sodium sensitivity in both hypertensive and normotensive subjects.

Metabolic and experimental studies have reported that magnesium has a role in the regulation of blood pressure (Resnick, 1999). *In vitro* studies have reported that magnesium has an effect on cell membrane sodium pump action, which affects the transport of sodium-potassium across membranes of cells and consequently vascular reactivity and tone (Song *et al*, 2006). According to Jee *et al.* (2002), clinical studies have also shown that there is significant decrease in blood pressure in eclampsia and glomerulonephritis patients with parenteral high dose magnesium and this indicates that an increased in dietary magnesium as found in the DASH diet can reduce blood pressure in healthy population. Magnesium reduces blood pressure by acting like natural calcium channel blocker and then contests for binding sites with sodium on vascular smooth muscles, binds in a supportive way to potassium, increases prostaglandin E, induces endothelial dependent vasodilation, improves hypertensive and diabetic patients' endothelial dysfunction, reduces intracellular sodium and calcium and subsequently decrease blood pressure (Barbagallo *et al*., 2010; Yamori *et al*., 2010).

Previous studies have indicated an inverse relationship between the intake of dairy products and cardiovascular disease, type 2 diabetes and metabolic syndrome (Bostick *et al.*, 1991; Choi *et al.*, 2005; Pereira *et al*., 2002). These prospective benefits of dairy intake are generally related to key nutrients such as calcium (Zemel, 1998). High intake of calcium reduces 1, 25-$(OH)_2$ Vitamin D and parathyroid hormone levels and thus decrease the influx of calcium extracellularly (Zemel, 2002). Decreased intracellular calcium may excite lipolysis and prevent lipogenesis, subdue the tone of vascular smooth muscle and endorse insulin motivated signal transduction (Zemel, 2002).

Population studies on the relationship between intake of dairy products and the prevalence of hypertension have been restricted to people in the Mediterranean countries (Alonso *et al*., 2005; Moore *et al*., 2005; Steffen *et al*., 2005). The DASH diet trial has proven that diet which is low in total and saturated fat and high in low-fat dairy products, fruits and vegetables efficiently decreases blood pressure in normotensive and hypertensive people (Wang *et al*., 2008). The blood pressure reducing effect of the DAH diet which stress on dairy intake were more reflective when compared with diets which were high in fruits and vegetables alone (Appel *et al*.,1997). Two prospective studies (Abbott *et al.*, 1996; Iso *et al.*, 1999) have demonstrated that milk or dietary calcium consumption decreases ischemic stroke risk but 2 other studies (Ness *et al.*, 2001; Elwood *et al.*, 2004) did not report any relationship.

2.4.6. DIETARY FAT

Dietary fat includes omega-3 and omega-6 polyunsaturated fats, monounsaturated fat and saturated fat (Appel *et al.*, 2006). Previous studies focused on the influence of total fats consumption on Blood pressure, even though there are possible biological foundations to postulate that certain kinds of fat such as omega 3 and omega 6 polyunsaturated fats might decrease blood pressure and that other kinds of fats such as saturated fat might increase blood pressure (Appel *et al.*, 2006).

Despite substantial evidence that the intake of saturated fats raises plasma LDL and causes atherosclerosis in humans, reports from several prospective cohort studies have not supported any substantial relationship between saturated fat and CVD risk (Mente *et al*., 2009; Siri-Tarino *et al*., 2010). Few clinical trials and many observational studies have evaluated the impact of saturated fat on blood pressure (Appel *et al*., 2006). Many studies including 2 prospective studies, the health professional follow up and the Nurses' health study, suggested that saturated

fat consumption was not related to hypertension (Ascherio *et al*., 1992; Ascherio *et al*., 1996). In the a small number of available trials, diet interventions that focused only on decreasing saturated fat had no substantial outcome on blood pressure (Morris, 1994). Previous cohort studies reported a relationship between higher saturated and trans fats consumption and increased CHD risk (Hu and Willet, 2002). As a significant measure to prevent CVD, the US Dietary Guidelines for Americans advocate a limit to the intake of saturated fat (USDA/USDHHS, 2010).

Consumption of dietary omega-6 polyunsaturated fat such as linoleic acid has little outcome on BP (Morris, 1994). In cross-sectional studies that correlated BP with omega-6 polyunsaturated fat plasma levels, there was no obvious association and prospective observational studies and clinical trials have also not supported such association (Morris, 1994; Ascherio *et al*., 1996; Pereira *et al*., 2004). Omega-6 Polyunsaturated fatty acids had anti-inflammatory properties which suppresses the development of adhesion molecules, interleukins and chemokines (these are vital intermediaries of the atherosclerosis development) in studies with vascular endothelial cells (De Caterina *et al.,* 2000). In randomized trials, metabolic studies and non-human primate studies, the intake of omega-6 polyunsaturated fatty acids reduced the risk of CHD by 10% to 21% (Harris *et al*., 2009).

Several epidemiologic studies have demonstrated people at risk for CHD benefits from the intake of omega-3 fatty acids. Prospective secondary prevention studies have also shown that the intake of omega-3 fatty acids from 0.5-1.8g/day substantially decreased mortality due to heart disease even though the quantity to take is not known (Kris-Etherton *et al*., 2003).

2.4.7. REFINED SUGARS

Added sugar was not a substantial aspect of the human diet until the inception of contemporary food processing techniques and since then, the intake of sugar has increase rapidly (Howard and Wylie-Rosett, 2002). Studies by Yudkin (1967 and 1978) indicated that high consumption of sugar was related to an increased risk of CVD. A recent study from the Nurses' Health Study demonstrated that women who consumes high intake of diets rich in sweets and highly processed starches had an increased risk of CHD (Liu *et al*., 2000). A few trials have also investigated the outcome of short term sugar intake on BP and many of such studies have demonstrated a direct association (Visvanathan *et al*., 2004).

CHAPTER THREE

MATERIALS AND METHODS

3.1. STUDY SITE

The study was carried out in the Ashanti Region of Ghana. It is the third largest region in Ghana. It occupies a total land surface of 24,389 km^2 (9,417 square miles) or 10.2% of the total land area of Ghana. It is the most populated region, according to the 2010 census, with a population of 4,780,380, accounting for 19.4% of Ghana's total population (Wikipedia, viewed 28/07/2017). Football (soccer) is the most popular sports in the region. Currently, the region has 2 Premier league clubs, 3 division 1 clubs and more than 50 division 2 clubs. The teams who participated in the study were located in Kumasi, the capital of Ashanti Region and Obuasi.

3.2. STUDY DESIGN

The study was cross sectional.

3.3. STUDY POPULATION

The subjects of the study consisted of players in the Premier, First and Second divisions' football teams. Teams whose players participated in the study were Ashanti Gold Sporting Club (Team 1), Anokye Stars Football Club (Team 2), S. K. Fresh Football Club (Team 3) and Sporting Times Football Club (Team 4).

3.4. SAMPLE SIZE

A sample size of 95 football players was recruited; 5 (Team 1), 41(Team 2), 21(Team 3) and 28 (Team 4) for the study. A convenience sampling technique was used in selecting the players for the study. All players registered by the teams for the season were included in the study. Each

team had at least 30 players who had registered for the season. For Team 1 especially, most of the players were not available during data collection.

3.5. ETHICAL APPROVAL

Ethical approval (CHRPE/AP/470/16) for the study was granted by the Committee on Human Research Publications and Ethics (CHRPE), School of Medical Science, Kwame Nkrumah University of Science and Technology (KNUST). Permission was also sought from the management of the teams whose players participated in the study. A consent form was signed by each of the players who participated in the study before they were recruited for the study.

3.6. INCLUSION CRITERIA

All players who were registered by the selected Clubs for the incoming season were included in the study. Players who agreed to allow their blood samples to be taken.

3.7. EXCLUSION CRITERIA

Players, who were on trials but had not been registered by the Clubs at the Ghana Football Association, were excluded from the study. Players who had eaten on the day of the data collection were also excluded.

3.8. DATA COLLECTION

Information on the players' demographics and playing roles were collected, using a pre-tested questionnaire. Pre-tested questionnaires were used to test the relevance of the questions being asked and the time used for answering the questionnaire.

3.8.1. ASSESSMENT OF DIETARY INTAKE

A food frequency questionnaire (FFQ) which contained about 133 foods, categorized into carbohydrates (tubers and grains), proteins (from both animal and plant sources), fats and oils (oils, fried foods, spreads and pastries), fruits, vegetables and others (beverages, soft drinks, energy drinks and alcohol) was used to assess the players' dietary patterns. The FFQ used for the study included eight (8) categories which ranged from "Daily" to "Never". The dietary intake of the players on two weekdays and one weekend were assessed, using a 24-hour dietary recall. Quantification of the amount of food eaten by participants was done, using handy measures of food items. The foods weight (in grams) was then recorded. This was done by using the household handy measures. The nutrients of the foods were then analyzed using the Nutrient Analysis Template (Tayie and Lartey, 1999; West African Food Composition Tables, 2012).

3.8.2. MEASUREMENT OF ANTHROPOMETRIC DATA

The heights (in centimeters) of players were measured, using stadiometer (Secca CE 0123). The weight (in kilograms), percentage body fat, percentage muscle fat, visceral fat and muscle mass of the players were measured using Omron Body Composition monitor (BF511). The BMI was then computed by dividing the weight by the height in meter square.

3.8.3. MEASUREMENT OF BIOCHEMICAL PARAMETERS

Five milliliters of venous blood samples of study participants were taken around 7 am in the morning by a phlebotomist through venipuncture. The blood samples collected were put into fluoride tubes and clot activator tubes.

3.8.3.1. FASTING PLASMA GLUCOSE

Sodium fluoride tubes were used to collect two (2) ml of blood samples and the blood samples in the tubes were centrifuged at 3000rpm for 5 minutes. Following the method of Randox Company Ltd, 1 ml of the supernatant was pipetted into a clean tube and 1 ml of Randox glucose reagent was then added. The solution was then incubated in a water bath for 10 mins at a temperature of 37°C and then transferred into a cuvette. A sample blank was run alongside and then reading taken at 550nm, using the Randox analyzer. The results were then recorded in mmol/L.

3.8.3.2 SERUM LIPID PROFILE

The lipid profile test consists of the analyses of High Density Lipoproteins (HDL), Low Density Lipoproteins (LDL)), triglycerides (TG) and Total Cholesterol (TC). The blood samples were centrifuged at 4000rpm for 5 mins to obtain the serum.

3.8.3.2.1. TEST FOR TRIGLYCERIDES

One (1) milliliter of the serum was measured into a test tube. 1 ml of TG reagent was measured and added to the test tube. A sample blank was prepared by measuring the reagent but not the sample. Both tubes were incubated in a water bath at a temperature of 37°C for 10 mins. The sample was then transferred into a cuvette for the concentration of TG to be determined, against the blank, using the Kenza Biochemistry analyzer, the result was recorded in mmol/L at an absorbance of 505nm. The procedure used is as recommended by Medsource Ozone Biomedical Limited, India.

3.8.3.2.2. ASSAY OF TOTAL CHOLESTEROL

One (1) ml of the serum was measured into a test tube and then 1 ml of TC reagent subsequently added to it. A sample blank was prepared by measuring the TC reagent but not the sample. They were then incubated at 37^0C for 5 mins. The sample concentration was determined against the blank using the Kenza Biochemistry analyzer after being transferred into a cuvette. At 505nm, the results were recorded in mmol/L. The protocol followed was as recommended by Medsource Ozone Biomedical Limited, India.

3.8.3.2.3. ASSAY OF HIGH DENSITY LIPOPROTEIN (HDL) AND LOW DENSITY LIPOPROTEINS (LDL)

HDL was analyzed by measuring 500µl of the serum into a clean dry tube. One (1) ml of the HDL precipitant was measured and added. It was mixed and incubated for 10 mins at a temperature of 25°C. After that, it was centrifuged at 4000rpm for 10 mins. About 100µl of the supernatant was pipetted into a new empty test tube. One (1) ml of the cholesterol reagent was added to the supernatant and incubated at 37°C for 5 mins. A reagent blank was prepared by measuring 100µl of distilled water and 1 ml of cholesterol reagent and incubated similarly. After the said time, reading was taken using the Kenza Biochemistry analyzer, against the reagent blank. The result for HDL was recorded in mmol/L. The HDL was analyzed following the procedure by Fortress Diagnostics, United Kingdom.

The LDL level was calculated using Friedewald's formula; (LDL) = (TC) – (HDL) - (TG/2.2)

3.9. MEASUREMENT OF BLOOD PRESSURE

Players were asked to rest for 30 minutes prior to the measurement of the blood pressure. They were then asked to sit on a chair upright with the back straight and the feet flat on the floor. The blood pressure was measured with the Omron automatic blood pressure monitor (Model M2 Basic). The arm and the back of the player were supported by the arms and back of the chair. The arm cuff was placed on the arm at the same level of the heart. The start/stop button was then pressed to measure the blood pressure. Blood pressure was recorded once because players were about going for their morning training.

3.10. DATA ANALYSIS

Data collected were analyzed using Statistical Package for Social Sciences (SPSS) version 23 of IBM Corporation, United States. For continuous variables, mean and standard deviations were calculated, while percentages were calculated for categorical variables. ANOVA was used to compare the means of the players of particular teams and their biochemical data and their food intake. A cross-tabulation analysis was also done to assess the players and their teams against their respective biochemical and anthropometry data. A p value of ≤ 0.05 was reckoned to be statistically significant.

CHAPTER 4

RESULTS

4.1. INTRODUCTION

This chapter presents the results of data collected from the participants (football players) in the Ashanti Region.

4.2. AGE PROFILE, PLAYING POSITIONS AND TEAMS OF PLAYERS

Ninety-five (95) players were recruited for the study. Out of the total number of players recruited, more than half of them (51.6%) were between 20-25 years. Majority of the players (40.0%) were also midfielders and Team 2 had the highest number of players (43.2%) recruited for the study, while Team 1 had the lowest number (Table 4.1).

Table 4.1: Age profile, playing positions and teams of players

VARIABLE	N	PERCENTAGE (%)
AGE (YEARS)		
15 – 19	31	32.6
20 -25	49	51.6
26 – 30	15	15.8
Total	95	100.0
PLAYING POSITION		
Goalkeepers	8	8.4
Defenders	30	31.6
Midfielders	38	40.0
Strikers	19	20.0
Total	95	100
TEAMS		
Team 1	5	5.3
Team 2	41	43.2
Team 3	21	22.1
Team 4	28	29.5
Total	95	100.0

4.3. DISTRIBUTION OF ANTHROPOMETRIC PARAMETERS OF PLAYERS

From Table 4.2, majority of the players fell within the normal weight, with none of the players being obese, and 5.3% of them being underweight. The players with normal body fat predominated (83.2%), and all the players had normal visceral fat. For the muscle mass, players with very high level was the most prominent, followed by those with high level.

Table 4.2: Distribution of anthropometric parameters of players

CHARACTERISTICS	N	PERCENTAGE (%)
BMI		
Underweight	5	5.3
Normal	82	86.3
Overweight	8	8.4
Obese	0	0
Total	95	100.0
% BODY FAT		
Low	10	10.5
Normal	79	83.2
High	5	5.3
Very High	1	1.1
Total	95	100.0
% MUSCLE MASS		
Normal	7	7.4
High	43	45.3
Very high	45	47.4
Total	95	100
VISCERAL FAT		
Normal	95	100
High	0	0
Very high	0	0
Total	95	100

Underweight BMI- <18.5kg/m^2, Normal weight – 18.5 – 24.9kg/m^2, Overweight BMI- 25.0-29.9kg/m^2, Obese BMI - ≥30kg/m^2, Low Body Fat- <8.0%, Normal Body fat – 8.0-19.9%, High – 20.0-24.9%, Very High - ≥25.0%, Normal % Muscle Mass – 33.3-39.3%, High % Muscle Mass – 39.4-44.0%, Very High % Muscle Mass - ≥44.1%, Normal Visceral fat – 1-9, High Visceral fat- 10-14, Very High Visceral Fat – 15-30

4.4. ANTHROPOMETRIC DISTRIBUTION BY AGE, PLAYING POSITIONS AND TEAMS OF PLAYERS

According to Table 4.3, all the anthropometric parameters were dependent on age as those between 26-30 years showed a significantly higher mean BMI (23.57±2.26 kg/m^2), mean percentage body fat (17.00±4.00%) and mean visceral fat (5.60±2.35). Anthropometric parameters were also dependent on teams, as Team 1 showed a significantly higher mean BMI, mean percentage body fat and mean visceral fat than the other teams. Playing positions did not have any effect on the anthropometric variables of the players.

Table 4.3: Anthropometric distribution by age, playing positions and teams of players

	N	BMI (Mean±SD)	p-value	%BF (Mean±SD)	P-value	%MM (Mean±SD)	P-value	VF (Mean±SD)	p-value
AGE (YEARS)									
15-19	31	20.03±1.95[a,]	**0.000**	10.71±3.51[a]	**0.000**	43.60±3.15[a]	**0.002**	2.90±1.49[a]	**0.000**
20-25	49	22.43±2.01[b,c]		13.71±4.05[b]		44.41±2.56		4.26±1.58[b]	
26-30	15	23.57±2.26[c,b]		17.00±4.00[c]		41.49±2.52[c]		5.60±2.35[c]	
PLAYING POSITION									
Goalkeepers	8	24.00±2.91	0.056	16.35±5.84	0.202	42.03±3.92	0.072	5.75±3.10	0.062
Defenders	30	21.73±2.27		13.04±4.37		44.45±2.70		3.90±1.71	
Midfielders	38	21.47±1.88		13.12±3.49		43.90±2.69		3.79±1.54	
Strikers	19	21.81±2.97		12.52±5.14		42.73±2.93		4.00±2.05	
TEAMS									
Team 1	5	25.56±2.94[a]	**0.000**	18.68±4.31[a,d]	**0.000**	41.06±2.87	0.400	6.00±1.41[a,c,d]	**0.000**
Team 2	41	20.43±1.96[b]		11.05±3.45[b,c]		44.05±3.09		3.00±1.43[b]	
Team 3	21	22.36±1.30[c,d]		13.14±3.70[c,b,d]		44.64±2.34		4.38±1.12[c,a,d]	
Team 4	28	22.81±2.25[d,c]		15.58±4.22[d,a,c]		42.90±2.73		4.93±2.28[d,a,c]	

*Mean values with different superscripts are significantly different at $p < 0.05$ level. BMI: Body Mass Index, % BF: Percentage Body Fat, % MM: Percentage Muscle Mass, VF: Visceral Fat

4.5. RELATIONSHIP AMONG ANTHROPOMETRY AND AGE, PLAYING POSITIONS AND TEAMS

Table 4.4 shows a cross tabulation of age, playing positions, teams and anthropometry. The BMI and muscle mass of the players were dependent on age and the teams. The 5 underweight players were found in the youngest year group, while the overweight were in the 20-25 and 26-30 year groups. The visceral fat was normal for all the year groups.

On the other hand, percentage body fat of the players was dependent on age, playing position and teams. Players of the age group 20-25 years constituted the majority of those who had normal body fat. Midfielders predominated the players with normal body fat, followed by defenders. According to teams, Team 2 had the highest percentage of players with normal body fat, followed by Team 4.

Table 4.4: Relationship among anthropometry and age, playing positions and teams of players

	AGE				PLAYING POSITIONS					TEAMS				
	15-19 (%)	**20-25 (%)**	**26-30 (%)**	**P value**	**GLKS (%)**	**DFDS (%)**	**MDFS (%)**	**STRK (%)**	**P Value**	**1 (%)**	**2 (%)**	**3 (%)**	**4 (%)**	**P value**
BMI														
Underweight	5 (5.3)	0 (0.0)	0 (0.0)	**0.004**	0 (0)	2 (2.1)	2 (2.1)	1 (1.1)	0.629	0 (0)	5(5.3)	0(0.0)	0(0.0)	**0.012**
Normal	26 (27.4)	44 (46.3)	12(12.6)		6(6.3)	27 (28.4)	33 (34.7)	16 (16.8)		3 (3.2)	35(36.8)	20 (21.1)	24(25.3)	
Overweight	0 (0)	5 (5.3)	3(3.2)		2 (2.1)	1 (1.1)	3 (3.2)	2 (2.1)		2(2.1)	1(1.1)	1(1.1)	4(4.2)	
Obese	0 (0)	0 (0)	0(0.0)		0 (0)	0 (0.0)	0 (0)	0 (0.0)		0 (0)	0(0.0)	0(0.0)	0(0.0)	
% BODY FAT														
Low	7(7.4)	3(3.2)	0 (0.0)	**0.022**	0(0.0)	4(4.2)	2(2.1)	4(4.2)	**0.048**	0(0.0)	8(8.4)	2(2.1)	0(0.0)	**0.000**
Normal	24(25.3)	42(44.2)	13(13.7)		6(6.3)	24(25.3)	35(36.8)	14(14.7)		2(2.1)	33(34.7)	19(20.0)	25(26.3)	
High	0(0.0)	4(4.2)	1 (1.1)		1(1.1)	2(2.1)	1(1.1)	1(1.1)		3(3.2)	0(0.0)	0(0.0)	2(2.1)	
Very High	0(0.0)	1(1.1)	1(1.1)		2(2.1)	1(1.1)	0(0.0)	0(0.0)		0(0.0)	0(0.0)	0(0.0)	1(1.1)	
VISCERAL FAT														
Normal	31(32.6)	49(51.6)	15(15.8)		8(8.4)	30(31.6)	38(40.0)	19(20.0)		5(5.3)	41(43.2)	21(22.1)	28(29.5)	
High	0(0.0)	0(0.0)	0(0.0)		0(0.0)	0(0.0)	0(0.0)	0(0.0)		0(0.0)	0(0.0)	0(0.0)	0(0.0)	
Very High	0(0.0)	0(0.0)	0(0.0)		0(0.0)	0(0.0)	0(0.0)	0(0.0)		0(0.0)	0(0.0)	0(0.0)	0(0.0)	
%MM														
Low	0 (0.0)	0(0.0)	0(0.0)	**0.017**	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0.298	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0.410
Normal	4(4.2)	2(2.1)	1(1.1)		2(2.1)	1(1.1)	2(2.1)	2(2.1)		1(1.1)	4(4.2)	0(0.0)	2(2.1)	
High	13(13.7)	18(18.9)	12(12.6)		3(3.2)	12(12.6)	17(17.9)	11(11.6)		3(3.2)	17(17.9)	8(8.4)	15(15.8)	
Very High	14(14.7)	29(30.5)	2(2.1)		3(3.2)	17(17.9)	19(20.0)	6(6.3)		1(1.1)	20(21.1)	13(13.7)	11(11.6)	

GLKS: Goalkeepers; DFDS: Defenders; MDFS: Midfielders; STRKS: Strikers; %MM: Percentage Muscle Mass; P values (Pearson chi-square) are significant at $p < 0.05$

4.6. BLOOD PRESSURE AND BIOCHEMICAL MARKERS OF PLAYERS

From Table 4.5, 8 of the players (8.4%) had hypertension, while 10 (10.5%) of the players were diabetics. More than a third of the players (36.8%) had lowered HDL levels, almost a third of the players (31, 32.6%) had high TC, while 1 (1.1%) had a high TG and 6.3% of the players had high LDL.

Table 4.5: Frequency distribution of blood pressure and biochemical markers of players

VARIABLE	N	PERCENTAGE (%)
BLOOD PRESSURE (mmHg)		
Normal	62	65.3
Pre-Hypertension	25	26.3
Hypertension	8	8.4
Total	95	100.0
FASTING PLASMA GLUCOSE		
Normal	74	77.9
Prediabetes	11	11.6
Diabetes	10	10.5
Total	95	100.0
TOTAL CHOLESTEROL		
Normal	64	67.4
High	31	32.6
Total	95	100.0
TRIGLYCERIDES		
Normal	94	98.9
High	1	1.1
Total	95	100.0
HIGH DENSITY LIPOPROTEINS		
Low	35	36.8
Optimal	60	63.2
Total	95	100.0
LOW DENSITY LIPOPROTEINS		
Normal	89	93.7
High	6	6.3
Total	95	100.0

Normal BP - <120/80 mmHg, Prehypertension - 120-139/80-89mmHg, Hypertension - ≥140/90mmHg, Normal FBS - <6.1mmol/L, Prediabetes – 6.1-6.9mmol/L, diabetes- ≥7mmol/L, Low TC-<5.2mmol/L, High TC-≥5.2mmol/L, Low TG-<1.69mmol/L, High TG- ≥1.69mmol/L, Low HDL- <1.42mmol/L, High HDL - ≥1.42mmol/L, low LDL- <3.88mmol/L High LDL-≥3.88 mmol/L

4.7. BLOOD PRESSURE AND BIOCHEMICAL MARKERS BY AGE, PLAYING POSITION AND TEAMS OF PLAYERS

According to Table 4.6, Systolic Blood Pressure (SBP) did not depend on age, position and team. On the other hand, Diastolic Blood Pressure (DBP) increased with age and teams; the highest mean DBP was found in the oldest age group; Team 4 recorded the highest mean level and Team 1, the lowest. The mean FPG did not show any difference, based on age and playing positions, but it was dependent on the teams, as Team 2 showed the highest mean level, while Team 3 showed the lowest. The mean TC also depended on age and teams; it decreased with age, as the youngest age group, 15-19 years had the highest mean TC and Team 1 also had the highest and Team 4 the lowest.

The mean TG was also dependent on playing positions as goalkeepers showed the highest, while strikers showed the lowest. The mean HDL did depend on teams and Team 3 and Team 4 showed the highest and lowest respectively. The mean LDL was dependent on age, playing positions and teams; it decreased with age, defenders had the highest LDL and goalkeepers had the lowest. Team 1 had the highest mean LDL whereas Team 4 had the lowest

Table 4.6: Blood pressure and biochemical markers by age, playing positions and teams of players

	N	SBP (Mean±SD)	P-value	DBP (Mean±SD)	P-value	FPG (Mean±SD)	P-value	TC (Mean±SD)	P-value	TG (Mean±SD)	P-value	HDL (Mean±SD	P-value	LDL (Mean±SD	P-value
AGE (YEARS)															
15-19	31	120.68±11.63	0.085	70.87 ±11.09[a,]	**0.030**	6.28±2.53	0.199	4.97±0.84[a]	**0.044**	0.77±0.27	0.369	1.59±0.24	0.612	2.96±0.67[a]	**0.036**
20-25	49	126.59±12.82		75.16 ±8.99		5.49±1.20		4.60±0.90		0.70±0.19		1.53±0.22		2.70±0.81	
26-30	15	124±12.17		79.19±12.02[b]		5.55±0.49		4.31±0.81[b]		0.81±0.46		1.55±0.24		2.35±0.64[b]	
PLAYING POSITIONS															
Goalkeepers	8	133.63 ±12.29	0.158	81.63±7.71	0.095	5.40±0.51	0.475	4.09±0.73	0.057	1.03± 0.57[a]	**0.016**	1.46±0.12	0.321	2.11±0.69[a, c,d]	**0.034**
Defenders	30	124.97 ±12.01		73.17±9.64		5.99±1.73		4.85±0.82		0.70±0.21[b,c, d]		1.58±0.20		2.91±0.70[b, c,d]	
Midfielders	38	123.47 ±12.44		72.63±9.89		5.89±2.12		4.80±0.98		0.75±0.25[c,b, d]		1.59±0.23		2.82±0.84[c, a,b,d]	
Strikers	19	122.74 ±11.04		76.84 ±12.77		5.29±0.84		4.39±0.73		0.68±0.17[d,b, c]		1.50±0.29		2.54±0.59[d, a,b,c]	
TEAM															
Team 1	5	133.40±5.18	0.297	69.80±14.10[a,b, c,d]	**0.003**	5.08±0.22[a,b,c ,d]	**0.007**	**5.14±0.90[a,b, c]**	**0.000**	0.72±0.16	0.113	1.50±0.10[a, b,c,d]	**0.008**	**3.30±1.01[a, b,c]**	**0.000**
Team 2	41	125.56 ±13.23		**70.90±10.55[b,a ,c]**		**6.39±2.41[b,a, d]**		**5.01±0.86[b,a, c]**		0.77±0.33		**1.60±0.22, ba,c**		**3.01±0.73[b, a,c]**	
Team 3	21	123.00 ±10.24		75.04±8.12[c,a,b, d]		**4.91 ±0.39[c,a,d]**		**5.00±0.58[c,a, b]**		0.83±0.21		**1.64±0.20[c, a,b]**		**2.94±0.53[c, a,b]**	
Team 4	28	123.00 ±12.43		**79.86±9.31[d,a,c]**		5.60±0.50[d,a, b,c]		**3.85±0.54[d]**		0.65±0.22		**1.44±0.23[d, a]**		**2.07±0.46[d]**	

*Mean values with the different superscripts are significantly different at $p < 0.05$ level. SBP: Systolic Blood Pressure, DBP: Diastolic Blood pressure, FPG: Fasting Plasma Glucose, TC: Total Cholesterol, TG: Triglycerides, HDL: High Density Lipoproteins, LDL: Low Density Lipoproteins

4.8. RELATIONSHIP AMONG BIOCHEMICAL MARKERS AND AGE, PLAYING POSITIONS AND TEAM

From Table 4.7, FPG and TC were dependent on age and teams; pre-diabetes was found in Teams 2 and 4; all the diabetics were from Team 2. The 20-25 year age group showed the highest number of high TC while high TC was predominantly found in Team 2. On the other hand, TG was dependent on playing positions. The midfielders showed the highest number of normal TG with high TG found in the Goalkeepers. Whether a player would be pre-diabetic or diabetic was dependent on age; these were found in ages 15-19 and 20-25, but not 26-30 years. Players with high total cholesterol levels were found in 15-25 years, but not 26-30 years. The distribution of pre-hypertension and hypertension did not depend on age, playing positions and teams. Also, age, playing positions and team did not show any significant effect on HDL and LDL of the players.

Table 4.7: Relationship among blood pressure and biochemical markers and age, playing positions and teams

	AGE				PLAYING POSITIONS					TEAMS				
	15-19 (%)	20-25 (%)	26-30 (%)	P value	GKS (%)	DFDS (%)	MDS (%)	STKS (%)	P value	1 (%)	2 (%)	3(%)	4 (%)	P value
BLOOD PRESSURE														
Normal	24(25.3)	29(30.5)	9(9.5)	0.188	2(2.1)	21(22.1)	26(27.4)	13(13.7)	0.119	2(2.1)	26(27.4)	15(15.8)	19(20.0)	0.294
Pre-hypertensive	7 (7.4)	13(13.7)	5(5.3)		5(5.3)	5(5.3)	9(9.5)	6(6.3)		3(3.2)	9(9.5)	6(6.3)	7(7.4)	
Hypertension	0 (0.0)	7(7.4)	1(1.1)		1(1.1)	4(4.2)	3(3.2)	0(0.0)		0(0.0)	6(6.3)	0(0.0)	2(2.1)	
FPG														
Normal	21(22.1)	41(43.2)	12(12.6)	0.075	7(7.4)	21(22.1)	29(30.5)	17(17.9)	0.688	5(5.3)	25(26.3)	21(22.1)	23(24.2)	**0.002**
Pre-diabetes	3(3.2)	5(5.3)	3(3.2)		1(1.1)	4(4.2)	5(5.3)	1(1.1)		0(0.0)	6(6.3)	0(0.0)	5(5.3)	
Diabetes	7(7.4)	3(3.2)	0(0.0)		0(0.0)	5(5.3)	4(4.2)	1(1.1)		0(0.0)	10(10.5)	0(0.0)	0(0.0)	
TC														
Normal	17(17.9)	33(34.7)	14(14.7)	**0.033**	7(7.4)	18(18.9)	23(24.2(	16(16.8)	0.140	2(2.1)	20(21.1)	14(14.7)	28(29.5)	**0.000**
High	14(14.8)	16(16.9)	1(1.1)		1(1.1)	12(12.7)	15(15.8)	3(3.2)		3(3.2)	21(22.1)	7(7.4)	0(0.0)	
TRIGLYCERIDE														
Normal	31(32.6)	49(51.6)	14(14.7)	0.068	7(7.4)	30(31.6)	38(40.0)	19(20.0)	**0.012**	5(5.3)	40(42.1)	21(22.1)	28(29.5)	0.722
High	0(0.0)	0(0.0)	1(1.1)		1(1.1)	0(0.0)	0(0.0)	0(0.0)		0(0.0)	1(1.1)	0(0.0)	0(0.0)	
HDL														
Low	8(8.4)	23(24.2)	4(4.2)	0.109	3(3.2)	11(11.6)	10(10.5)	11(11.6)	0.143	2(2.1)	12(12.6)	5(5.3)	16(16.8)	0.057
High	23(24.2)	26(27.4)	11(11.6)		5(5.3)	19(20.0)	28(29.5)	8(8.4)		3(3.2)	29(30.5)	16(16.8)	12(12.6)	
LDL														
Optimal	29(30.5)	46(48.4)	14(14.7)	0.996	8(8.4)	27(28.4)	35(36.8)	19(20.0)	0.446	4(4.3)	37(38.9)	20(21.1)	28(29.5)	0.224
High	2(2.1)	3(3.2)	1(1.1)		0(0.0)	3(3.2)	3(3.2)	0(0.0)		1(1.1)	4(4.2)	1(1.1)	0(0.0)	

FPG: Fasting Plasma Glucose; TC: Total Cholesterol; HDL: High Density Lipoproteins; LDL: Low Density Lipoproteins; GKS: Goalkeeper; DFDS: Defenders; MDS: Midfielders; STKS: Strikers; P values (Pearson chi-square) are significant at $p<0.05$

4.9. DIETARY INTAKE DISTRIBUTION BY AGE, PLAYING POSITIONS AND TEAMS OF PLAYERS

Shown in Table 4.8 is the dietary intake by age, playing positions and teams of players. Players of age group 20-25 had the highest mean total energy intake, as well as highest mean intakes of carbohydrates, proteins and fats, except that these were not significantly different ($p > 0.05$) from the other age groups.

According to playing positions, the goalkeepers had the highest mean total energy intake, as well as highest mean intakes of carbohydrates, proteins and fats even though they were not statistically significant. Team 4 also showed the highest mean intakes of total energy, carbohydrate, proteins and fat but these were not significantly different from the other teams ($p>0.05$).

Table 4.8: Dietary intake distribution by age, playing positions and teams of players

	N	TEI (Mean±SD)	p-value	CHO (Mean±SD)	p-value	PROTEIN (Mean±SD)	p-value	FAT (Mean±SD)	p-value
AGE									
15-19	31	2107.16±621.95	0.204	311.50±109.95	0.634	70.62±20.96	0.524	66.71±21.08	0.082
20-25	49	2342.96±848.18		324.18±106.35		75.54±23.94		83.00±50.76	
26-30	15	1999.89±692.26		294.12±119.96		70.63±26.14		61.17±22.89	
PLAYING POSITION									
Goalkeeper	8	2547.57±1198	0.437	338.96±134.52	0.788	79.27±24.15	0.814	99.32±80.01	0.184
Defender	30	2070.64±542.03		301.26±94.16		71.81±21.78		66.68±22.49	
Midfielder	38	2216.26±803.87		315.74±121.02		74.23±26.35		71.59±34.32	
Striker	19	2284.61±767.75		326.62±99.87		70.58±19.41		80.88±48.02	
TEAMS									
Team 1	5	1693.55±357.72	0.220	269.87±84.45	0.809	55.72±11.06	0.206	44.33±22.86	0.071
Team 2	41	2159.82±596.43		319.34±102.61		70.96±22.25		69.06±20.56	
Team 3	21	2173.74±962.06		312.11±133.72		74.94±25.64		72.57±54.71	
Team 4	28	2409.15±836.08		319.86±105.24		78.14±23.51		88.41±48.04	

TEI: Total Energy Intake, CHO: Carbohydrate; P-values are significant at $p < 0.0$

4.10. DIETARY HABITS OF PLAYERS

Shown in Table 4.9 is the dietary pattern of participants. Only about a third of the players ate 3 times in a day. The remaining ate either once or two times in a day, with the majority (44.2%) eating two times.

Table 4.9: Frequency distribution of dietary patterns of participants

DIETARY PATTERN	N	PERCENTAGE (%)
3 MEALS A DAY	32	33.7
2 MEALS A DAY	42	44.2
1 MEAL A DAY	21	22.1
TOTAL	95	100.0

4.11. BLOOD PRESSURE AND BIOCHEMICAL MARKERS IN RELATION TO DIETARY PATTERN OF PLAYERS

Table 4.10 shows the blood pressure and biochemical markers by the dietary patterns of the players. The players who ate two meals showed the highest mean systolic and diastolic BP even though this is not significantly different ($p>0.05$).

Players who ate 3 meals a day also showed the highest means FPG, TC and HDL, except that they were not statistically different ($p>0.05$). The mean TG increased as the number of meals also increased with those eating 3 meals a day showing the highest and those who ate once a day showing the lowest but it was not significantly different.

The players who ate 3 meals a day showed the highest mean LDL while those who ate 2 times and 1 meal showed the same mean LDL, although they were not statistically significant ($p> 0.05$).

Table 4.10: Blood pressure and biochemical markers by dietary pattern

	3 MEALS A DAY (Mean±SD)	2 MEALS A DAY (Mean±SD)	1 MEAL A DAY (Mean±SD)	P value
Systolic (mmHg)	124.53±13.29	125.86±11.77	122.43±11.40	0.577
Diastolic (mmHg)	72.59±10.78	75.40±9.81	75.14±11.52	0.493
FPG (mmol/L)	6.08±2.44	5.54±1.03	5.70±1.46	0.402
Total Cholesterol (mmol/L)	4.95±0.83	4.52±0.86	4.56±0.98	0.099
Triglyceride (mmol/L)	0.69±0.24	0.75±0.28	0.80±0.31	0.348
HDL (mmol/L)	1.58±0.17	1.54±0.25	1.56±0.27	0.757
LDL (mmol/L)	2.97±0.71	2.61±0.77	2.61±0.77	0.093

FBS: Fasting Plasma Glucose, TC: Total Cholesterol, TG: Triglycerides, HDL: High Density Lipoproteins, LDL: Low Density Lipoproteins. P-values are significant at $p<0.05$

4.12. RELATIONSHIP AMONG DIETARY PATTERN, PERCENTAGE MUSCLE MASS AND BIOCHEMICAL MARKERS.

According to Table 4.11, FPG did depend on muscle mass; all prediabetics were having normal muscle mass with no high or very high muscle mass. For the diabetics, equal numbers were found to have low and normal muscle mass, but like the pre-diabetics, none of them had high or very high muscle mass.

Total Cholesterol (TC) was also dependent on dietary pattern; those who ate 3 meals a day showed the highest number of high TC and those who ate 1 meal a day showed the lowest. The distribution of blood pressure did not depend on the dietary pattern and the percentage muscle mass of the players. Also, the dietary pattern and percentage muscle mass of the players did not show any significant effect on their TG, HDL and LDL.

Table 4.11: Relationship among dietary pattern, percentage muscle mass, blood pressure and biochemical markers

	DIETARY PATTERN				PERCENTAGE MUSCLE MASS				
	3 MEALS A DAY (%)	2 MEALS A DAY (%)	1 MEAL A DAY (%)	P value	LOW (%)	NORMAL (%)	HIGH (%)	V.HIGH (%)	P value
BLOOD PRESSURE									
Normal	21(22.1)	26(27.4)	15(15.8)	0.909	8(8.4)	50(52.6)	4(4.2)	0(0.0)	0.530
Pre-hypertensive	9(9.5)	12(12.6)	4(4.2)		2(2.1)	21(22.1)	1(1.1)	1(1.1)	
Hypertensive	2(2.1)	4(4.2)	2)2.1)		0(0.0)	8(8.4)	0(0.0)	0(0.0)	
FPG									
Normal	25(26.3)	35(36.8)	18(18.9)	0.260	5(5.3)	67(70.5)	5(5.3)	1(1.1)	**0.003**
Prediabetes	2(2.1)	5(5.3)	0(0.0)		0(0.0)	7(7.4)	0(0.0)	0(0.0)	
Diabetes	5(5.3)	2(2.1)	3(3.2)		5(5.3)	5(5.3)	0(0.0)	0(0.0)	
TOTAL CHOLESTEROL									
Low	16(16.8)	33(34.7)	15(15.8)	**0.031**	4(4.2)	55(57.9)	4(4.2)	1(1.1)	0.218
High	16(16.8)	9(9.5)	6(6.3)		6(6.3)	24(25.3)	1(1.1)	0(0.0)	
TRIGLYCERIDES									
Normal	32(33.7)	41(43.2)	21(22.1)	0.529	10(10.5)	78(82.1)	5(5.3)	1(1.1)	0.977
High	0(0.0)	1(1.1)	0(0.0)		0(0.0)	1(1.1)	0(0.0)	0(0.0)	
HDL									
Low	8(8.4)	18(18.9)	9(9.5)	0.234	2(2.1)	30(31.6)	3(3.2)	0(0.0)	0.392
High	24(25.3)	24(25.3)	12(12.6)		8(8.4)	49(51.6)	2(2.1)	1(1.1)	
LDL									
Optimal	30(31.6)	40(42.1)	19(20.0)	0.765	10(10.5)	74(77.9)	4(4.2)	1(1.1)	0.508
High	2(2.1)	2(2.1)	2(2.1)		0(0.0)	5(5.3)	1(1.1)	0(0.0)	

FBS: Fasting Plasma Glucose HDL: High Density Lipoprotein, LDL: Low Density Lipoprotein, P value < 0.00

CHAPTER FIVE

DISCUSSION

5.1. AGE PROFILES, PLAYING POSITIONS AND TEAMS OF PLAYERS

This study was aimed at finding how the dietary pattern of some football players, predispose them to some cardiovascular disease risk factors. It involved using some 95 players of four teams from the Ashanti Region. It was a cross-sectional study in which anthropometric measurements of the players were done, blood pressure measured and blood samples taken to determine FBS and lipid profile.

According to Table 1, majority of players fell within the age range of 20 to 25 years. This was similar to a study by Abdulai (2015), which reported that 58% of soccer players in the division 1 teams in Tamale, Ghana were between 20-29 years of age. This means that the players were young and have more playing days ahead of them in their career. It also revealed that the defenders and midfielders were the playing positions that were very dominant (71.6%). This was in contradiction to a study of Ofori *et al*.(2012), which reported that strikers (attackers) formed the majority of football players (45%) in Ghana but similar to the findings of Iglesias-Gutierrez's study (2012), which reported that 72% of football players are either defenders or midfielders.

5.2. ANTHROPOMETRICAL PARAMETERS AND CARDIOVASCULAR DISEASES

From Table 2, majority of the players were of normal BMI and that the prevalence of overweight and obesity among the football players was 8.4%. This is in contradiction to the findings of Abdulai (2015), that there was 39% of overweight and obesity in division one players in Tamale,

Ghana. It is also lower, compared to a similar study done by Stiefel *et al*. (2016), which reported a 19.8% of overweight and 10.1% obesity among student soccer players.

It was also observed that all the players had normal visceral fat and most of the players had a high and very high muscle mass. This is in agreement with similar studies where a high muscle mass has been found in athletes (Prentice and Jebb, 2001; Kreider *et.al,* 2010). This also underscores the study by Davidson et al. (2002), which reported that a high muscle mass, correlates to a low total body fat.

From Table 3, it is observed that BMI, percentage body fat and visceral fat increased as age increased, with players between 26-30 years having the highest. This was similar to the work done by Meeuwsen *et al.* (2010), who also reported a direct relationship between age and BMI and percentage body fat. This means that as one grows older, his or her BMI increases. Moreover, it was also observed that Team 1 had a higher BMI, percentage body fat and visceral fat than the other teams. This could be due to the low number of players who participated in the study from the team, compared to the other teams.

According to Table 4, all the underweights (5, 5.3%) were between the ages of 15-19 years and were all found in Team 2. It was also observed that majority of the players who were overweight were also found in Team 4. Very high percentage body fat was more prevalent among the goalkeepers (2.1%), compared to other playing positions.

5.3. RELATIONSHIP AMONG BLOOD PRESSURE, BIOCHEMICAL MARKERS AND CARDIOVASCULAR DISEASE

According to Table 5, 8.4% of the players were hypertensives. This is a little higher than the 7% reported by Berge *et al*. (2013), even though lower than the 13.8% and 19.2% reported by

Tucker *et al.* (2009), and Karpinos *et al*. (2013), respectively. The high rate of hypertension among the players could be due to over-training and poor dietary intake. Over-training is thought to have a negative impact on the autonomic cardiovascular control (Baumert *et. al*., 2006). Overtraining results when there is an imbalance between training and competition and recovery. In Ghana, most teams train at least 3 hours a day and 5 times in a week. This could affect the heart muscles as they are not able to get enough recovery between their training and competition. The training and exercise routine of the players could mask the symptoms of hypertension which can lead to a sudden cardiac death on the field. Healthy dietary patterns are related to significant reduction in cardiovascular disease risks factors (Lichtenstein *et. al.*, 2006). Poor dietary pattern which were reported by most (66.3%) of the players as in Table 9 (most of them not getting 3 meals a day) could increase their risk to cardiovascular disease.

The prevalence rate of prediabetes and diabetes were 7.4% and 10.5% respectively. The high prevalence of prediabetes and diabetes among the players could be due to inadequacy of their dietary intake. This could also be a case of type 1 diabetes as majority of the diabetics were below the age of 20 years. Moreover, the pattern of dyslipidemia in the players as observed; 36.8% reduced HDL, 32.6% high levels of cholesterol and 1.1% of high triglyceride were contradictory to the study by de Araujo *et al*. (2012), which found prevalence of hypercholesterolemia (29.2%), hypertriglyceridemia (16.7%), low HDL (4.2%) and High LDL (20.8%) among soccer players in Brazil. Thus the two key components of the dyslipidemia among the players were reduced HDL and increased cholesterol.

According to Table 6, there was a direct association between age and diastolic blood pressure. Players between the ages of 26-30 had a higher diastolic blood pressure, compared to the other age groups. This finding supports the work by Sharma, (2003) which reported that age affects the

size of the heart muscles of athletes. Total cholesterol and low density lipoprotein also reduced as age increases. This was in contradiction to reported studies which reported that as age increases, total cholesterol and LDL increase, thereby increasing risk of cardiovascular disease of a person (Conroy *et al.,* 2003). It was also revealed that goalkeepers had a higher mean TG than the other playing positions. Defenders also showed a higher mean LDL than other playing positions.

From Table 7, the pre-diabetes and diabetes are more prevalent among the younger age group (15-25 years). All the 10 players who were diabetics were found to be in Team 2. It was also observed that majority of the players who were having high TC were between the age groups of 15-19 years and 20-25 years. Players with high TC were also common in Team 2 (21, 22.1%).

Hyperglycemia is one of the prevalent risk factors among the players who participated in the study. Chronic hyperglycemia destroys the endothelial cells of blood vessels which can further develop into atherosclerosis and increase the players' risk of cardiovascular disease (Bhutto *et. al*., 2017). This accounts for why some of the players may be susceptible to cardiovascular disease.

Several investigations conducted have reported a relationship between hypertriglyceridemia and coronary artery disease (Sarwar *et. al*., 2007; Shai *et al*., 2004). The difference in the lipid profiles of the players by their playing positions could be due to the playing styles and system of play of the team since their dietary intake did not have any significant effect on their lipid profiles (Table 10). Dyslipidemia is a major cardiovascular disease risk factor (Porez *et al.*, 2012). The reduced HDL but increased levels of cholesterol are noteworthy, as these can contribute to atherosclerosis in the players. There is induction of inflammatory response and

extensive vascular remodeling when plasma lipids are accumulated in the anterior intima. This results in the development of atherosclerotic plaques which increases the risk factor for cardiovascular diseases (Libby *et al*., 2011).

5.4. NUTRITIONAL INTAKE AMONG THE PLAYERS

According to playing positions, the goalkeepers had the highest mean total energy intake, as well as highest mean intakes of carbohydrates, proteins and fats, even though they were not statistically significant (Table 8). Team 4 also showed the highest mean intakes of total energy, carbohydrate, proteins and fat, except that these were not significant ($p>0.05$). The energy and nutrient intakes in this study are in contradiction to the total energy intake reported by a study done by Iglesias-Gutiérrez *et al*., 2012. They reported a total energy intake of 2600±641, 2766±452, 2855±475 and 2779±659 for Goalkeepers, Defenders, Midfielders and Strikers respectively. The dietary intakes observed in this study were lower as compared to studies done by Rico-Sanz (1998), Leblanc *et al.* (2002) and Iglesias-Gutiérrez *et al.*, (2012). This shows that the Ghanaian players were having low energy intake, compared to other soccer players in other countries. This could be due to sub-optimal dietary intake, as most of the players were eating either once or two meals in a day. Sub-optimal dietary intake could lead to poor performance in competitions (Caccialanza *et al*., 2007).

From Table 9, most of the players (66.6%) were eating either once or two times in a day and so could not meet their dietary goals. Good dietary intake is an important component of a soccer player's health and sporting future (Rogol *et al*., 2000). Despite this, the soccer players are not meeting their dietary goals. This could be due to players having low financial status and so could not afford 3 meals a day.

CHAPTER 6

6.1. CONCLUSION

The dietary pattern of the players indicated that they had a poor dietary intake, as most of them were eating once or twice a day. Poor dietary pattern could increase a player's cardiovascular disease risk. Anthropometric parameters showed a high incidence of overweight (8.4%), normal body fat percentage (83.2%), high muscle mass (92.7%) and normal visceral fat. The normal body fat percentage observed in majority of the players could be due to high muscle mass and normal visceral fat showed in the study. The prevalence rate of pre-diabetes and diabetes were 11.6% and 10.5% respectively. The other factors revealed in the study include hypertension (8.4%), hypercholesterolemia (32.6%) and low HDL (35.8%). Although, the anthropometric parameters observed may not give an indication of the cardiovascular disease risk, the prevalence of high blood pressure, high blood glucose, reduced HDL and hypercholesterolemia may put the players at an increased risk of cardiovascular disease.

6.2. LIMITATIONS

Blood pressure was measured once. The 24-hour recall and food frequency questionnaire were personal responses from the players. This means that there was a high possibility of over/under reporting. The sample size and inability to recruit majority of players from Team 1.

6.2. RECOMMENDATIONS

1. Players should be counselled on healthy eating pattern and their dietary intake.

2. Biochemical markers of players should form part of their usual pre-participation screening before the beginning of each league season.

3. Dieticians should form part of the technical team of each team so they could educate the playing body and the entire team staff about the importance of healthy eating.

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APPENDIX

APPENDIX A

Participant Information Leaflet and Consent Form

This leaflet must be given to all prospective participants to enable them know enough about the research before deciding to or not to participate

Title of Research: Assessing the prevalence of cardiovascular disease risk factors among football players of the premiership clubs in Ashanti Region of Ghana

(For example: Phase III randomized clinical trial of "New Toothpaste" for Treatment of Fetororis)

Name(s) and affiliation(s) of researcher(s): Daniel Afrifa

Background (Please explain simply and briefly what the study is about): the study is about assessing the cardiovascular disease risk factors among football players using their nutritional intake

Purpose(s) of research: the study is to find out the nutritional and dietary intake and the prevalence of cardiovascular disease risk factors among the football players of the Premier League Clubs in the Ashanti Region

Procedure of the research, what shall be required of each participant and approximate total number of participants that would be involved in the research:

Risk(s): The risks that will be associated with this study will be the pain and discomfort during the drawing of venous blood (5ml of blood).

Benefit(s): The participants will be educated on their nutritional and dietary intake. Their biochemical indices such as lipid profile and fasting blood glucose will also be an added advantage to the Participants as this is not routinely done by their clubs. (For example:

Confidentiality: Participants' names will be recorded on the questionnaires. This will help when giving feedback to study subjects about their laboratory results and nutritional status, so that they can seek medical help. All information collected will be known by the members of the research group only. No name will be used in any publication or reports of this study. However, as part of our responsibility to conduct this research properly, we may allow officials from ethics committees to have access to our records such as the names.

Voluntariness: Taking part in this study should be out of your own free will. You are not under obligation to participate. Research is entirely voluntary

Alternatives to participation: if you choose not to participate, this will not affect you in any way.

Withdrawal from the research: You may choose to withdraw from the research at any time without having to explain yourself. You may also choose not to answer any question you find uncomfortable or private.

Consequence of Withdrawal There will be no consequence, loss of benefit or care to you if you choose to withdraw from the study. Please note however, that some of the information that may have been obtained from you without identifiers (name etc.), before you chose to withdraw, may still be modified or used in analysis reports and publications. We do promise to make good faith and effort to comply with your wishes as much as practicable.

Costs/Compensation: For your time/inconvenience, we will compensate you with a snack to show our appreciation for your participation.

Contacts: If any question concerning this study, you can contact Daniel Afrifa on 0248793527

Further, if you have any concern about the conduct of this study, your welfare or your rights as a research participant, you may contact:

The Office of the Chairman
Committee on Human Research and Publication Ethics
Kumasi
Tel: 03220 63248 or 020 5453785

CONSENT FORM

Statement of person obtaining informed consent:
I have fully explained this research to _____ and have given sufficient information about the study, including that on procedures, risks and benefits, to enable the prospective participant make an informed decision to or not to participate.

DATE: _____ NAME: _____

Statement of person giving consent:
I have read the information on this study/research or have had it translated into a language I understand. I have also talked it over with the interviewer to my satisfaction.

I understand that my participation is voluntary (not compulsory).

I know enough about the purpose, methods, risks and benefits of the research study to decide that I want to take part in it.

I understand that I may freely stop being part of this study at any time without having to explain myself.

I have received a copy of this information leaflet and consent form to keep for myself.

NAME: _____

DATE: _____ SIGNATURE/THUMB PRINT: _____

Statement of person witnessing consent (Process for Non-Literate Participants):

I _____ (Name of Witness) certify that information given to _____ (Name of Participant), in the local language, is a true reflection of what l have read from the study Participant Information Leaflet, attached.

WITNESS' SIGNATURE (maintain if participant is non-literate): _____

MOTHER'S SIGNATURE (maintain if participant is under 18 years): _____

MOTHER'S NAME: _____

FATHER'S SIGNATURE (maintain if participant is under 18 years): _____

FATHER'S NAME: _____

APPENDIX B: QUESTIONNAIRE: ***ASSESSING THE CARDIOVASCULAR DISEASE RISK FACTORS AMONG SOCCER PLAYERS***

A. SOCIO-DEMOGRAPHIC DATA

I.NAME: …… II. AGE: ……. yrs. CODE:

III. MARITAL STATUS: [ ] SINGLE [ ] MARRIED IV. TEAM: ……..
V. POSITION: ……..

B. ANTHROPOMETRIC DATA

I. HEIGHT: …… cm II. WEIGHT: …… Kg III. BMI: ……kg/m^2

IV. BLOOD PRESSURE (BP): ……mmHg V. SKELETAL MUSCLE MASS: ……%

VI. TOTAL BODY FAT: …… % VII. VISCERAL FAT: …….. VIII. RM: …… kcal

C. BIOCHEMICAL DATA

I. FBS: ……mmol/L REFERENCE: < 7.0

ii. TOTAL CHOLESTEROL: …….. mmol/L REFERENCE: 3.6 – 6.4

iii. TRIGLYCERIDE: ……mmol/L REFERENCE: 0.34 – 1.82

iv. HDL: …….. mmol/L REFERENCE: 0.90 – 3.00

v. LDL: …… mmol/L REFERENCE: 0.00 – 3.88

D. FOOD FREQUENCY QUESTIONNAIRE

FOODS **A. CARBOHYDRATES**	**DAILY**	**ONCE IN A WEEK**	**2-3 TIMES IN A WEEK**	**>3 TIMES A WEEK**	**ONCE A MONTH**	**> 1 A MONTH**	**SELDOM (IRREGULAR IN THE YEAR)**	**NEVER**
a. TUBERS								
Cocoyam								
Plantain								
Sweet potatoes								
Yam								
Cassava (fufu, Konkonte,)								
Gari								
Others (specify)								

FOODS	DAILY	ONCE IN A WEEK	2-3 TIMES IN A WEEK	>3 TIMES A WEEK	ONCE A MONTH	> 1 A MONTH	SELDOM (IRREGULAR IN THE YEAR)	NEVER
b. GRAINS AND CEREALS								
Plain Rice (White)								
Plain Rice (Brown)								
Waakye								
Banku								
Fante kenkey								
Ga kenkey								
Spaghetti/Macaroni								
Akple								
Tuo Zaafi								
Omotuo								
Mpotompoto								
Maize Porridge								
Hausa Koko (Millet Porridge)								
Tom brown/weanimix								
Rice Porridge								
Oats Porridge								
Wheat Porridge								
Ekwegbemi								
Others (specify)								
B. PROTEINS								
a. ANIMAL BASED PROTEINS								
I. MEAT AND MEAT PRODUCTS								
Beef								
Chicken								
Boiled Eggs								
Sausages								
Cow's skin (Wele)								
Offals (Liver, intestines, tripes)								
Pork								
Goat Meat								
Snails								
Sheep Meat								
Grass cutter meat								
Cat meat								
Dog meat								
Duck meat Rabbit Meat								

FOODS	DAILY	ONCE IN A WEEK	2-3 TIMES IN A WEEK	>3 TIMES A WEEK	ONCE A MONTH	> 1 A MONTH	SELDOM (IRREGULAR IN THE YEAR)	NEVER
ii. FISH								
Tilapia								
Amane (Fresh or smoked)								
Red fish								
Edoie 'barracuda'(Fresh or smoked)								
Mud fish (Fresh or smoked)								
Cattle fish								
Others specify								
iii. MILK AND MILK PRODUCTS								
Full fat milk								
Low fat milk								
Semi skimmed milk								
Skimmed milk								
Condensed Milk								
Brukina								
Wagashie/cheese								
Atadwe Milk								
b. PLANT SOURCE PROTEINS								
Baked Beans								
Groundnuts								
Groundnut soup								
Beans Stew								
Soybean								
Groundnut paste								

FOODS	DAILY	ONCE IN A WEEK	2-3 TIMES IN A WEEK	>3 TIMES A WEEK	ONCE A MONTH	> 1 A MONTH	SELDOM (IRREGULAR IN THE YEAR)	NEVER
C. FATS AND OILS								
a. OILS								
Palm oils								
Sunflower oil								
Coconut oil								
Soybean oil								
Fish oils								
b. FRIED FOODS								
Ball float								
Fried yams								
Beans cake (Koose)								
Maasa								
Fried plantains								
Kelewele								
Plantain chips								
Pizza								
Potato chips								
Fried Eggs								
Fried Rice								
Jollof								
Fried fishes								
SPREADS AND PASTRIES								
Margarine								
Mayonnaise								
Meat pie								
Cake								
Spring roll								

FOODS	DAILY	ONCE IN A WEEK	2-3 TIMES IN A WEEK	>3 TIMES A WEEK	ONCE A MONTH	> 1 A MONTH	SELDOM (IRREGULAR IN THE YEAR)	NEVER
D.FRUITS AND VEGETABLES								
a. FRUITS								
Oranges								
Banana								
Watermelon								
Pineapple								
Apple								
Pawpaw								
Mango								
Coconut								
Grapes								
Tangerine								
Avocado pear								
Guava								
blackberries								
Africa apple								
Fruit juices (Fresh)								
Others specify								
b. VEGETABLES								
Cabbage								
Carrot								
Lettuce								
Cassava Leaves								
Green Peppers								
Pumpkin Leaves								
Green beans								
Moringa								
Dandelion								
Agusi								
Garden eggs								
Kontomire								
Okro								
Tomatoes								
Pepper								
Others specify								

FOODS	DAILY	ONCE A WEEK	2-3 TIMES A WEEK	> 3 TIMES A WEEK	ONCE A WEEK	> 1 A MONTH	SELDOM (IRREGULAR IN THE YEAR)	NEVER
E. OTHERS								
a. BEVERAGES								
Chocolate drink								
Tea								
Coffee								
Millet drink (Pito)								
b. SOFT DRINKS AND ENERGY DRINKS								
soft drink (Bottled and canned(								
Energy drinks								
Sobolo								
c. ALCOHOL								
Beer								
Spirits								
Gin								
Wine								
MENU PLAN AND FREQUENCY								
Breakfast (7:00 – 8:00 am)								
Mid-Morning snack (10:00 – 11:00 am)								
Lunch (1:00 - 2:00 pm)								
Mid-Afternoon snack (3:00 – 4:000 pm)								
Supper (5:30 – 6:30 pm)								
Bedtime snack (8:00pm)								

E. 24-HOUR RECALL

WEEK DAY 1

TIME	MEAL/FOOD	QUANTITIES (HANDY MEASURE)	WEIGHT (g)
Breakfast			
Time:			
Mid-morning snack			
Time:			
Lunch			
Time:			
Mid-afternoon snack			
Time:			
Supper			
Time:			
Bedtime snack			
Time:			

WEEK DAY 2

TIME	MEAL/FOOD	QUANTITIES (HANDY MEASURE)	WEIGHT (g)
Breakfast			
Time:			
Mid-morning snack			
Time:			
Lunch			
Time:			
Mid-afternoon snack			
Time:			
Supper			
Time:			
Bedtime snack			
Time:			

WEEKEND 1

TIME	MEAL/FOOD	QUANTITIES (HANDY MEASURE)	WEIGHT (g)
Breakfast			
Time:			
Mid-morning snack			
Time:			
Lunch			
Time:			
Mid-afternoon snack			
Time:			
Supper			
Time:			
Bedtime snack			
Time:			