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THE EFFECTIVENESS OF DIETARY AND LIFESTYLE INTERVENTION IN PERSONS WITH MAJOR LIFESTYLE RELATED CHRONIC DISEASES AT THE KORLE-BU TEACHING HOSPITAL

BY

REBECCA KISSIWA STEELE-DADZIE

(ID. NO. 10328188)

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DECLARATION

I declare that this thesis is the report of research undertaken by me under the supervision of Prof. A.G.B. Amoah, Prof. Margaret Armar-Klemesu and Prof. George Asare. All references to other people's work have been duly cited.

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REBECCA K. STEELE-DADZIE

…….....

PROF. ALBERT G.B. AMOAH

(PRINCIPAL SUPERVISOR)

……

PROF. MARGARET ARMAR-KLEMESU

…….

PROF. GEORGE ASARE

ABSTRACT

Background: The global prevalence of chronic non-communicable diseases (NCDs) continues to rise, accounting for over 71% of all global death and responsible for over 40% of pre-mature death (death occurring before the age 70 years). Low and middle income countries (LMIC) are expected to suffer the brunt of this epidemic, leading to high rates of disease, death and disability adjusted live years (DALYs). Most NCD's and their shared risk factors are preventable through lifestyle modifications, mainly regular physical activity, consumption of healthy diet, cessation of smoking and moderate use of alcohol. The main lifestyle indicator for the prevention of NCD's, however, is intake of healthy diet with emphasis on the regular intake of whole grains, fruits and vegetables, fish, nuts and seeds, low-fat dairy products, low saturated and trans-fat and high mono and polyunsaturated fat. Dietitians are the health care professionals specially trained to apply nutritional science in providing evidence based dietary intervention to prevent and manage various NCDs mainly through counselling. The dietetics profession and practice is evolving in Ghana, with a number of health care facilities currently resourced with dietitians. Patients are increasingly being referred by physicians to dietitians, to assist in the management of their various conditions. Information on the common conditions that get referred to dietitians in Ghana is, however, scanty. There is also paucity of information regarding patient's attitude towards such referrals and potential challenges they face in their effort to seek dietetic care. In addition, evidence is lacking on the effectiveness of the dietary intervention patients receive on their measurable health outcomes. These gaps in the practice of Dietherapy in Ghana informed this research.

Aim: The study had 2 phases. The primary aim for phase one was to identify the major lifestyle-related chronic diseases referred to the Dietherapy Department of the Korle-Bu Teaching Hospital (KBTH), the major referral tertiary facility in Ghana. The primary aim

for phase two was to audit the effectiveness of the routine dietary and lifestyle intervention given to patients at the Dietherapy Department of the KBTH on their dietary habits, alcohol intake, physical activity habits, smoking habits, anthropometry, body composition, blood pressure and biochemical indicators.

Method: Phase one was a cross-sectional study. Phase two was a clinical audit which followed a longitudinal study design. The Dietherapy Department of the Korle-Bu Teaching Hospital was the study site for both phases. Eligible participants were newly referred adult patients, referred by a medical doctor, who consented to participate. Nutrient and usual dietary intake were assessed using 24-hour dietary recall interviews for 2 days (1 weekday and 1 weekend) and a validated Food Frequency Questionnaire (FFQ) respectively. Anthropometry (height and weight for BMI, waist and hip circumference for waist-hip circumference ratio), body composition (percentage body fat, visceral fat, skeletal muscle, total body water) and blood pressure were measured. Biochemical indices (serum total cholesterol, low density lipoprotein cholesterol (LDLc), high density lipoprotein cholesterol (HDLc), triglycerides, C-reactive protein, fasting plasma glucose and 2-hour plasma glucose) were determined. All measurements were done at baseline and repeated at 3 and 6 months follow-up. Dietary and lifestyle intervention were based on the department's protocols developed from international guidelines for chronic disease management, especially National Cholesterol Education Programme and International Diabetes Federation, as well as the dietary approach to stop hypertension (DASH) and Mediterranean diets. All patients were counselled at baseline and reiterated at reviews. Statistical package for social sciences (SPSS) version 20 was used to analyze the data at a 95% confidence interval. Descriptive statistics (means, median, standard deviation, ranges) were used to summarize continuous variables such as anthropometric indices and blood pressure. Chi square was used to analyze categorical data. Differences between means at baseline and

follow-up were determined using analysis of variance (ANOVA) for repeated measures and independent sample t-test. Multiple linear and logistic regression analysis were done to determine the predictors of the variability of variables that significantly changed in the course of the study.

Results: A total of 339 patients participated in the cross-sectional study. A sub-sample of 132 were followed up for 6 months with 60 patients completing at 6 months. Hypertension (48.1%), was the major diet related chronic condition referred to the Dietherapy Department. It was followed by diabetes (44.8%), dyslipidemia (38.1%) and obesity (24.8%). Half (50.1%) of patients who were referred delayed in assessing dietary care mainly because the Department had closed when they got there on the same day of referral. Alcohol intake, smoking and physical activity levels of patients were all generally low at baseline and did not change markedly after 6 months. The proportion of patients who engaged in moderate physical activity, however, increased significantly at 6 months (61.7%; $P < 0.001$) compared to baseline (30.3%). Patients' body mass index (BMI) and educational level predicted 13% of the variability in physical activity habits. Normal weight and overweight patients had 55% ($p = 0.172$, CI: 0.235- 1.296) and 40% ($p = 0.015$, CI: 0.194 – 0.840) odds of not engaging in physical activity respectively, compared to obese patients. The odds of patients with no formal education, primary education or secondary education not engaging in moderate physical activity was 19 ($p < 0.001$, CI: 3.686 – 99.604), 3 ($p = 0.009$, CI: 1.316 – 6.924) and 2 ($p = 0.358$, CI: 0.629 – 3.608) times that of those with tertiary education respectively. Carbohydrate and dietary fibre intake increased significantly ($p = 0.010$, $p = 0.025$) respectively, between baseline and 6 months. This was attributed to the addition of whole grains and fruits to the usual diets of patients. Daily intake of total fat, protein and sodium decreased by 6 months but were not statistically significant ($p = 0.195$, $p = 0.132$, $p = 0.430$) respectively. Total daily energy intake did not change significantly

throughout the study (p = 0.344). Body mass index and visceral fat decreased significantly (p < 0.001, p = 0.005) respectively, between baseline and 3 months. The decreases were sustained at 6 months (p = 0.001, p = 0.003) respectively. Systolic and diastolic blood pressure remained unchanged despite pharmacological treatment and lifestyle modification (137 ± 26 mmHg; p = 0.867) and (88 ± 15 mmHg; p = 0.925) respectively. Fasting plasma glucose (FPG) significantly decreased (p = 0.044) between baseline and 3 months and was sustained at 6 months (p = 0.001). Two-hour glucose also significantly decreased at 6 months (p = 0.001). Both fasting and 2-hour glucose were predicted by waist-hip circumference ratio. High density lipoprotein (HDL) cholesterol increased significantly at 6 months (p = 0.045). The variability in HDL was explained by patients' percentage body fat, LDLc, triglyceride and serum total cholesterol in a multiple linear regression model.

Conclusion: The commonest NCDs seen at the Dietherapy Department were hypertension followed by diabetes mellitus, dyslipidaemia and obesity in decreasing order. Routine dietary and lifestyle intervention given to patients with NCDs at the Dietherapy Department of the KBTH led to improvement in a significant number of the measurable outcomes. These improvements have the potential to reduce their related complications if maintained on the long term. Physicians must therefore be encouraged to refer more patients for dietetic care.

DEDICATION

This thesis is dedicated to God Almighty, for all the great things He has done and continues to do in my life. Secondly, I dedicate this thesis to my entire family: husband, children, parents and siblings, for their numerous sacrifices, relentless support and patience for me throughout the pursuit of this programme. I am forever grateful.

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LIST OF ABBREVIATIONS

ACC - American College of Cardiology

ADA - American Diabetes Association

AHA - American Heart Association

AMDR - Acceptable Micronutrient Distribution Range

BIA - Bio-electrical Impedance Analysis

BMI - Body Mass Index

CHD - Coronary Heart Disease

CKD - Chronic Kidney Disease

COPD - Chronic Obstructive Pulmonary Diseases

CVD - Cardiovascular Diseases

DALYs - Disability Adjusted Life Years

DASH – Dietary Approach to Stop Hypertension

ESHA - Elizabeth Stewart Hands and Associates

FFQ - Food Frequency Questionnaire

GHS - Ghana Health Service

GSS - Ghana Statistical Service

IDF - International Diabetes Federation

ISH - International Society of Hypertension

KBTH – Korle-Bu Teaching Hospital

LMIC - Low and Middle Income Country

MNT - Medical Nutrition Therapy

NCDs - Non-Communicable Diseases

NCEP - National Cholesterol Education Program

NCP - Nutrition Care Process

NHANES - National Health and Nutrition Examination Survey

NHMRC - National Health and Medical Research Council

NICE - National Institute of Health and Care Excellence

OPD - Out Patient Department

RDA - Recommended Daily Allowance

WC - Waist Circumference

WHO- World Health Organization

WHR- Waist-Hip Circumference Ratio

CHAPTER ONE

1.0 INTRODUCTION

Non-communicable diseases (NCDs) are a group of diseases that are mostly chronic in nature. They have been widely defined in terms of their characteristic attributes including: their duration, their need for regular medical attention, their effect on body function, pathology (Dalal *et al.,* 2011; WHO, 2014; May *et al.,* 2015), being noncontagious in nature (Daar *et al.,* 2007), having multiple risk factors, and their incurable nature (Goodman *et al.,* 2013). A number of diseases are found in this group due to the heterogeneity of their definition. However, the main contributors to NCD deaths globally are cardiovascular diseases (CVD), cancers, diabetes and respiratory diseases, including asthma and chronic obstructive pulmonary disease (COPD) (Ekpenyong *et al.,* 2012; Sommer *et al.,* 2015).

In 2012, NCDs were responsible for the 68% of the world's 56 million deaths. About 42% (16 million) of these were premature, occurring below the age of 70 years (WHO, 2014). This figure reflects an increase compared to reported data in the year 2000, where 14.6 million premature deaths were attributed to NCDs (WHO, 2014). Low and middle income countries are most affected by this health menace. About 80% of global mortality attributed to NCDs occur in developing countries (Oti, van de Vijver and Kyobutungi, 2014). In these nations, it has added on to their problems with infectious diseases, resulting in a double burden of disease (Dalal *et al.,* 2011; Oti, van de Vijver and Kyobutungi, 2014). Their shared risk factors mainly obesity and hypertension, also continue to rise. The overall pooled prevalence of hypertension in Africa increased from 19.7% in 1990 to 27.4% in 2000 and 30.8% in 2010 (Adeloye and Basquill, 2014). The Overall prevalence of NCDs in Southern Nigeria is reported as 32.8% while disease specific prevalence is 25%, 14.4%, 12.7%, for obesity, hypertension and diabetes mellitus respectively (Ekpenyong *et al.,* 2012). Among

adult Ghanaians, prevalence of type 2 diabetes in the Greater Accra Region was 6% in 2000 (Amoah *et al.,* 2000). Nearly 43% of Ghanaian adults are either overweight or obese (Ofori-Asenso *et al.,* 2016). The national prevalence of overweight and obesity are estimated as 25.4% (95% CI 22.2–28.7%) and 17.1% (95% CI =14.7–19.5%), respectively. Urban dwellers are relatively more overweight and obese compared to rural dwellers (Amoah, 2003a; Ofori-Asenso *et al.,* 2016). Urban hypertension prevalence is reported as 32.3%, while rural prevalence is 27% (de-Graft Aikins *et al.,* 2014). A few decades ago, this was not the trend. Chronic diseases like hypertension were not considered as significant health threats especially in rural Ghanaian societies (Pobee *et al.,* 1977). Their harmful effects are feared to increase if steps are not taken to prevent or manage them (de-Graft Aikins *et al.,* 2010). Figure 1.0 below is the conceptual framework which describes the risk factors for non-communicable diseases and how they can be reversed through lifestyle interventions.

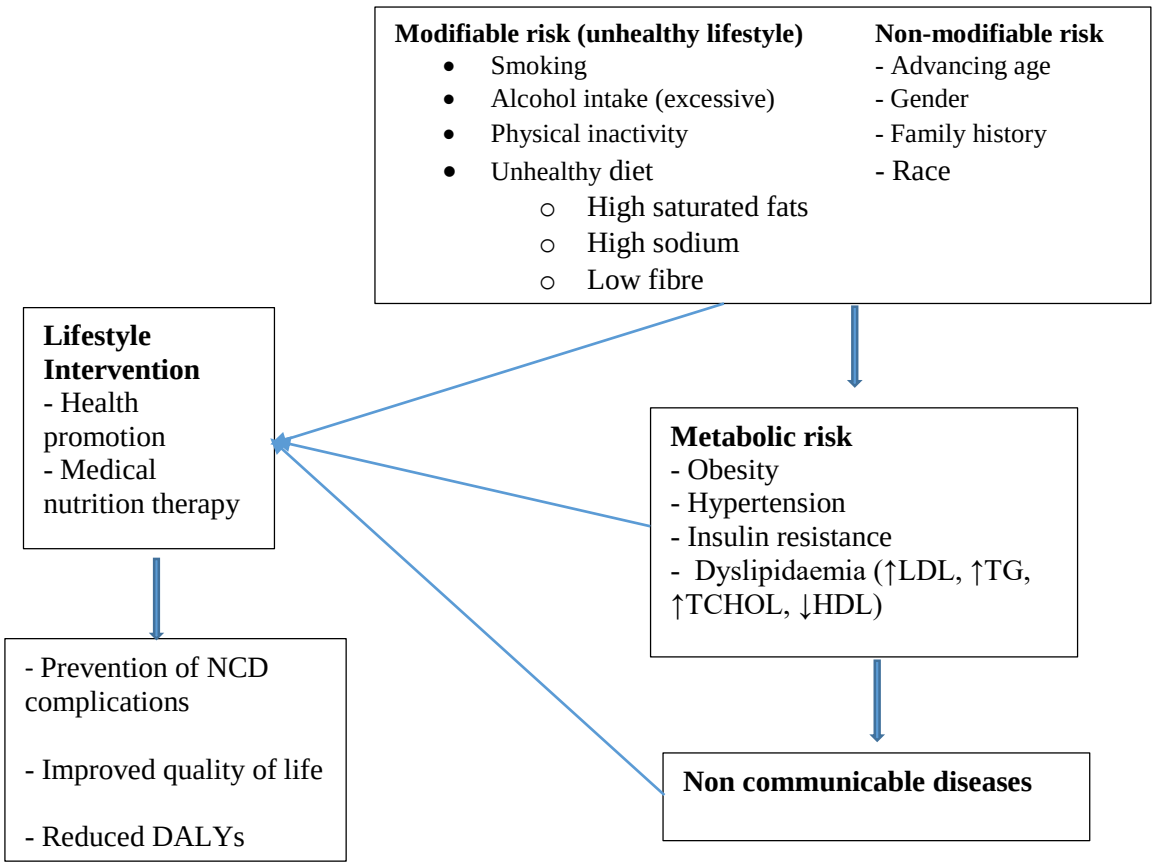


Fig 1.0: Conceptual framework of non-communicable diseases

The increasing prevalence of NCDs globally has been attributed to the significantly higher prevalence of preventable risk factors mainly, tobacco and alcohol use, low intake of fruits and vegetables and high rates of physical inactivity (Allen *et al.,* 2017). These risk factors in turn lead to four main metabolic/ physiologic changes, (raised blood pressure, overweight/obesity, raised blood glucose and raised cholesterol). These further increase the risk of developing NCDs (WHO, 2017b; Jamalizadeh *et al.,* 2016). as described in Fig 1.0 above. Other factors including changing lifestyle practices, increased life expectancy, poverty, globalization and urbanization have also been blamed (de-Graft Aikins *et al.,* 2010).

Randomized control trials and other studies have suggested that dietary and lifestyle interventions are effective in improving the health of most patients with NCDs (Appel *et al.,* 2006; Sarrafzadegan *et al.,* 2009). Adherence to dietary interventions that follow the dietary approach to stop hypertension (DASH) diet, improved blood pressure as well as reduced the risk of stroke and coronary heart disease (Fung *et al.,* 2008). A combined diet and physical activity intervention reduced body weight and improved fasting blood glucose and lipids in obese, glucose impaired adults in the Finnish diabetes prevention study (Lindström *et al.,* 2003). Like most cosmopolitan populations in recent times, Ghanaians, particularly those in urban areas, have very low physical activity levels, frequently consume high fat diets and also consume inadequate amounts of fruits and vegetables in their diets (Steele-Dadzie *et al.,* 2009; Hall *et al.,* 2009). Their risk of obesity and its associated health risk is therefore increased.

The Korle-Bu Teaching Hospital is the nation's biggest referral hospital. It is the third biggest hospital in Africa with a bed capacity of 2000 (KBTH, 2016a). The hospital has 17

clinical and diagnostic departments/units, an average daily attendance of 1,500 patients and about 250 patient admissions. The Dietherapy department provides medical nutrition therapy for all patients referred, both in and out patients (KBTH, 2016b). Patients referred from other health facilities across the nation also patronize the department.

Dietary intervention at the department typically takes the form of one-on-one counselling sessions. Counselling is evidence-based and is conducted with the aid of appropriate health communication tools such as diet sheets, food handy measures and food models.

Evidence-based dietary counselling interventions, especially with the aid of interactive health communication tools have been identified as effective in changing dietary intake habits such as reduction in the consumption of saturated fats and increased intake of fruits and vegetables. These behavior changes are more common among populations at risk of, or diagnosed with disease, than among general healthy populations (Ammerman *et al.*, 2002; Adisa and Fakeye, 2014). Among diabetes patients attending a community health centre at Moretele Sub-District (North West Province), South Africa, nutrition education significantly reduced the intake of starchy foods and overall energy intake after six and twelve months in the intervention group compared to control group (Muchiri *et al.,* 2016). Non adherence to dietary treatment among ambulatory patients with poorly controlled type 2 diabetes in southwestern Nigeria was largely attributed to inappropriate guidance (Adisa and Fakeye, 2014).

Such studies have not been carried out at the Dietherapy Department of the KBTH. To the knowledge of the author, there is no scientific data on the effect of routine dietary and lifestyle interventions given to patients on their measurable health outcomes in Ghana.

1.1 Problem Statement

In many low and middle income countries, the relentless rise in the prevalence of non-communicable diseases was left unchecked over some decades and this has resulted in avoidable loss of many lives (Ebrahim *et al.*, 2013). The four main NCDs; Cardiovascular disease, cancers, diabetes mellitus and chronic pulmonary disease, are now the leading causes of death globally, with about 80% of all CVD deaths occurring in developing countries (Fuster *et al.*, 2011). In 2015 a total of 56.4 million global deaths occurred, 39.5 million, or 70%, were due to NCDs (WHO, 2015a). The burden caused by these diseases is highest in LMIC where 48% of these deaths occur before the age of 70 years (WHO, 2015a). Equally so is the burden of disability adjusted life years (Duncan *et al.*, 2002), which is the sum of years of life lost and years lived with disability (Murray *et al.*, 2012). In Table 1.0, the top ten causes of admissions in 2013, at the Korle-Bu Polyclinic (KBPC), which is the primary health care wing of the KBTH is shown. Hypertension, with its complications and diabetes mellitus occupied the top 2 positions for both males and females. Cerebrovascular accident was the fourth and fifth cause of admission for males and females respectively.

Table 1.0: Top ten causes of admissions at the Korle-Bu Polyclinic

	Males	**Females**
1	Hypertension & complications	Hypertension & Complications
2	Diabetes mellitus	Diabetes Mellitus
3	Sickle cell disease	Sickle Cell disease
4	Cerebrovascular accident	Malaria
5	Respiratory tract infections	Cerebrovascular Accident
6	Malaria	Gastro-enteritis
7	Gastroenteritis	Respiratory tract infections
8	Anaemia	Urinary tract infections
9	Peptic ulcer/ gastritis	Anaemia
10	Alcoholism	Peptic ulcer/ gastritis

Source; KBTH annual report, 2013

These are all non-communicable in nature, attesting to the reported increasing contribution of NCDs to morbidity and mortality especially in developing countries (WHO, 2014).

Added to the death and ill-health caused by these NCDs is the major economic cost it presents on national healthcare budgets. The greater the severity of disease and the number of years lived with the disease, the higher the cost (Muka *et al.*, 2015). Most developing countries barely meet their national budgetary requirements and have to depend on international donors for support (Loser, 2004). The additional cost of NCDs therefore threatens their very economic survival. Of all NCDs, cardiovascular disease presents the highest economic impact on health care budget of between 12% and 16.5% annually (Muka *et al.*, 2015). Low and middle income countries (LMICs) are projected to save as much as 25 billion USD annually if efforts are made to reduce the mortality due to stroke and ischemic heart disease by 10% (Bloom *et al.*, 2013). At the household level, the economic cost of NCDs occur directly from the high cost of treatment and frequent visits to health care facilities to seek specialized care (Kankeu *et al.*, 2013). Between 5% and 24.5% of household incomes are spent on diabetes care in rural low-income households. For those on the minimum wage in rural Ghana, spending on insulin alone represents around 60% of monthly household incomes (de-Graft Aikins, 2005; Kankeu *et al.*, 2013). The indirect negative impact of NCDs on household incomes may occur from frequent absenteeism at work due to ill-health or disability, leading to loss of productivity and eventually loss of employment and reduced incomes (McIntyre *et al.*, 2006). Families of affected persons experience the additional social burden of caring for individuals with prolonged disability (de-Graft Aikins, 2005) and surviving on limited resources for the home due to the economic demands of the disease (Bloom *et al.*, 2013; McIntyre *et al.*, 2006). It further reduces productivity through loss of productive days spent caring for sick relatives (Alam and

Mahal, 2014) which sometimes may also lead to dropping out from school or loss of employment. A viscous cycle of poverty is therefore created (WHO, 2010; Alam and Mahal, 2014).

The negative social and economic impact of NCDs may well not have seen its worst as the age group most affected by them (age 60 years and above) is projected to increase due to increased life expectancy (Wang *et al.*, 2016)). The world population prospects report of the Department of Economic and Social Affairs of the United Nations, predicts an annual 1% growth rate of the population 60 years and above in the more developed countries, from now to 2050. After this period, it is projected to decline to 0.11% up to 2100 (United Nations, 2013). For the less developed countries, however, it reports that it is currently at its fastest growth ever of 3.7% between 2010-2015 and is projected to increase by 2.9% annually before 2050 and 0.9% annually from 2050 to 2100 (United Nations, 2013). These predictions suggest a rise in the number of people who will potentially be affected by NCDs and therefore likely to attend the nation's health facilities to seek medical care.

Out-patients attendance has already increased since the introduction of the National Health Insurance Scheme (NHIS) (Ghana Health Service, 2008). About 83% of all OPD attendants have been insured (Ghana Health Service, 2015). The number and spectrum of diet-related NCDs which would potentially be referred to the Dietherapy Unit of the KBTH is therefore likely to increase.

Most NCDs and their major risk factors, however, are preventable. Systematic review and meta-analysis have shown that dietary and lifestyle interventions can avert or delay the occurrence of NCDs, their secondary complications and death. Their major risk factors are

lifestyle related and include smoking, wrong use of alcohol, physical inactivity and poor dietary habits (WHO, 2010; WHO, 2014). Randomized control trials have indicated that smoking cessation reduces the risk of mortality from both lung cancer and cardiovascular diseases (Anthonisen *et al.,* 2005). Among high risk middle age men, smoking cessation coupled with reduction in the intake of cholesterol reduced the incidence of myocardial infarction and coronary heart disease by 47% in intervention group compared to control group (Hjermann *et al.,* 1981). Improvement in gastro esophageal reflux has also been associated with smoking cessation (Ness-Jensen *et al.,* 2014). In a population with a high burden of alcohol related diseases, the major NCD identified was chronic obstructive pulmonary disease (COPD) (n = 148, 45%) with most patients presenting with signs of liver cirrhosis (Amundsen *et al.,* 2016). Non-smoking, low BMI (BMI < 25.0 kg/m^2), being physically active, and adherence to a Mediterranean diet, significantly lowers disease burden (May *et al.,* 2015). Higher adherence to healthy dietary patterns is associated with a lower disease burden as assessed by DALYs (Struijk *et al.,* 2014).

The importance of dietary and lifestyle intervention for the prevention and management of NCDs have been recognized by several international expert panels for the development of guidelines for NCD management (International Diabetes Federation, 2012a; Stone *et al.,* 2014; National Cholesterol Education Programme, 2001; National Kidney Foundation, 2012). Together, these panels recommend that dietary and lifestyle interventions are crucial to the success or otherwise, of their NCD management guidelines. In populations that have not benefited from dietary and lifestyle interventions and education, the age onset of NCDs have progressively reduced (WHO, 2010). In such populations, poor control of these diseases is common and have resulted in higher rates of DALY (Oti, van de Vijver and Kyobutungi, 2014).

The Dietherapy Department of the KBTH employs evidence-based dietary intervention in managing their patients' conditions. Registered dietitians apply evidence from most international guidelines to provide individualized dietary care to patients. The Department has not carried out a well-designed scientific research to investigate and document the success or otherwise of the treatment given to their patients. The scope of conditions which are referred to the dietitians also remain to be investigated. Patients' dietetic care-seeking behaviour after being referred, and possible challenges they encounter in the course of accessing dietary care, also remains to be investigated. This information is critical for the advancement of dietetics and patient disease management in Ghana and formed the bases for carrying out this study.

1.2 Justification

Evidence of the effect of dietetic care on patient's measurable disease outcomes will be obtained. A positive outcome will form the basis to advocate for all health facilities in Ghana to be resourced with dietitians towards building a complete health care team in all primary, secondary and tertiary health facilities in Ghana. Currently, in addition to the generally few numbers of registered dietitians in the country, about 80% of them are employed in the Greater Accra Region (Aryeetey *et al.*, 2014), while some other regions have none. This compromises the standard of care that patients who patronize health facilities outside of Accra receive.

Evidence from this study will also inform public health policy on NCD prevention using lifestyle modification,

Patient dietetic care seeking behaviour after referral, and challenges they encounter when accessing dietetic care will also be identified. These findings will be made available to the various stakeholders such as the Ghana Health Service, to eliminate the barriers militating against easy access to dietetic care. In addition, the Ghana Dietetics Association, could organize continuous professional development programmes, to address areas in the dietetic practice which may be identified as needing improvement. These steps will significantly improve dietetic and general health care delivery in Ghana to combat the harmful effects of NCDs and other diseases.

The training of dietitians in Ghana has been ongoing for some years now, with institutions such as the School of Biomedical and Allied Health Sciences in the University of Ghana spearheading this move. Information obtained from this study will fill the knowledge gap of the common conditions that are referred to dietitians by medical doctors. This information is critical in the light of current consideration for specialist training in dietetics in Ghana .The long-term benefit would be improved dietetic care in the management of lifestyle-related chronic diseases in all health facilities in Ghana.

1.3 Aims and Objectives

Phase One, Cross-Sectional Study

Aims

To identify the major lifestyle-related chronic diseases referred to the Dietherapy Department of the Korle-Bu Teaching Hospital (KBTH).

Objectives

1. To identify and document the main conditions for which patients were referred to dietitians.
2. To obtain demographic and socioeconomic information of patients.
3. To identify the dietetic care-seeking behaviour of patients after referral to see a dietitian and the challenges they encounter when accessing dietetic care.
4. To determine patients' physical activity, smoking and alcohol intake habits.

Phase Two, Longitudinal Study (a clinical audit)

Aim

To audit the effectiveness of the routine dietary and lifestyle intervention given to patients at the Dietherapy Department of the KBTH on their dietary habits, alcohol intake, physical activity habits, smoking habits, anthropometry, body composition, blood pressure and biochemical indicators.

Objectives

1. To compare the mean nutrient, energy and dietary fibre intakes and the usual dietary intakes of patients at baseline to that at 3 months and 6 months.
2. To compare the mean blood pressure, anthropometry and body composition of patients at baseline to that at 3 and 6 months.
3. To compare blood biochemistry (fasting plasma glucose, 2-hour plasma glucose after a 75g glucose load, blood lipids and c-reactive protein) of patients at baseline to that at 3 and 6 months.
4. To determine changes in patients' physical activity, alcohol intake and smoking habits after routine dietary and lifestyle intervention.

5. To identify the predictors of the variability in all variables that underwent significant changes from baseline to 6 months.

1.4 Research questions

The study had the following research questions;

1. What is the common lifestyle-related chronic conditions referred by physicians to dietitians?
2. What are the demographic, socioeconomic and lifestyle characteristics of patients with these conditions?
3. What is the dietetic care-seeking behaviour of patients after referral to see a dietitian and the challenges they encounter when accessing dietetic care?
4. Does routine dietary and lifestyle intervention given by registered dietitians at the nation's major referral hospital have any effect on patients' dietary intake, alcohol intake, physical activity habits, smoking habits, anthropometry, body composition, blood pressure, plasma sugars, serum lipids and serum c-reactive protein?

1.5 Null hypothesis

Routine dietary and lifestyle intervention given by registered dietitians at the nation's major referral hospital, have no effect on patients' dietary intake, alcohol intake, physical activity habits, smoking habits, anthropometry, body composition, blood pressure, plasma sugars, serum lipids and serum c-reactive protein.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Obesity

Obesity is defined as "a condition of the accumulation of excess body fat to the extent that health is impaired" (WHO, 2008b). Globally the prevalence of obesity continues to rise even though obesity is preventable. In 2016, 39% of the world's population who were 18 years and above were overweight with 13% obese (WHO, 2017c). The national prevalence of overweight and obesity in Ghana are estimated as 25.4% and 17.1% respectively (Ofori-Asenso *et al.,* 2016). Obesity contributes to an increase in morbidity. It deceases an individual's quality of life, and increases their risk of mortality (Zhang *et al.,* 2014). Abdominal obesity, diabetes, hypertension and abnormal lipids were among the 9 risk factors which were identified to predict 90% of the population attributable risk of myocardial infarction in men and 94% in women in the INTERHEART study (Ajay and Prabhakaran, 2010). Many factors determine whether an individual becomes obese or not.

2.1.1 Pathophysiology and risk factors of obesity

The primary established cause of obesity is being in a prolonged state of positive energy balance, such that more energy is consumed than what is used up by the body either through physical or metabolic activities or both (Zhang *et al.*, 2014; WHO, 2000). Although obesity has multiple causes, some of which are not well understood, they can be grouped under the broad topics of genetic and environmental causes (Zhang *et al.,* 2014). Key among the environmental causes of obesity are excess food intake and physical inactivity. Others such as inadequate night sleep (Cassidy *et al.,* 2016), socioeconomic status, ethnicity, place of residence, whether urban or rural, have also been identified as potential causes of obesity (Jiang *et al.,* 2016; Amoah, 2003b).

2.1.1.1 Genetic causes of obesity

Twin studies reporting similarities in the BMI of twins raised in different environments, as well as adopted children whose BMI reflected that of their biological rather than their adopted parents, confirm the genetic influence on obesity (Stunkard *et al.*, 1990; Dubois *et al.*, 2012; Stunkard *et al.*, 1986; Longo *et al.*, 2017). Genetic studies have identified the association of single nucleotide polymorphisms with BMI, and reported that over 300 identified loci together account for less than 5% of the variation in BMI and adiposity of individuals (Longo *et al.*, 2017). Individuals who have one or two copies of the risk allele have a 1.2 kgm^{-2} or 3 kgm^{-2} increase respectively in BMI as compared with those without copies of the allele. Some genetic disorders also incite the consumption of obesity prone foods which are energy dense, and high in refined sugars and dietary fats (Zhang *et al.*, 2014; von Deneen and Liu, 2011). Other genetic disorders affect hormone secretion and metabolism which results in weight gain (McArdle *et al.*, 2013).

A recent inclusion into the causes of obesity which is not well understood is the intractable weight gain due to a damage to the hypothalamus (Lustig, 2008; Steele *et al.*, 2013).

2.1.1.2 Excess food intake as a cause of obesity

Frequent intake of food in excess of what the body requires results in a continuous state of positive energy balance. Food from sweetened sugars, sweetened beverages, refined carbohydrates, pastries, high fat meats, high fat dairy products are energy dense and can easily result in a state of positive energy balance (Bray and Barry, 1998). One gram of fat contains 9 kcal while one gram of protein and carbohydrate contributes 4 kcal. Regular intake of high fat diets will therefore increase the risk of obesity.

The quality of fat in a diet and not just the amount of energy is also reported to have greater impact. The structure of fatty acids is suggested to affect their degree of oxidation and deposition (Moussavi *et al.,* 2008). Some fatty acids are prone to oxidation and some others lead to fat storage when comparing isocaloric diets (Moussavi *et al.,* 2008; Schwab *et al.,* 2014). In that regard, monounsaturated fatty acids appear to favour weight loss compared to saturated fatty acids in human studies. Most of the energy obtained from fat is directly stored in adipose tissue and adds to body weight and width (Jiang *et al.,* 2016).

Alcohol intake also contributes to energy intake. One gram of alcohol contains 7 kcal and can therefore increase the state of positive energy balance. The excess energy is stored in the body as fat in adipose tissue. Adipose tissue which consist of adipocytes and connective tissue will continue to expand through the increase in the number and size of adipocytes if the state of positive energy balance continues leading to obesity (Longo *et al.,* 2017; Jiang *et al.,* 2016).

Foods that are high in unrefined carbohydrates, especially from cereals and other high fibre sources, such as fruits and vegetables, reduce the risk of obesity, because they are less energy dense. Additionally, low fat dairy products such as yoghurt, and foods rich in monounsaturated fats reduce the risk of obesity (Perez-Martinez *et al.,* 2011).

2.1.1.3 Physical inactivity as a cause of obesity

Obesity is also caused by prolonged physical inactivity (Ekelund *et al.,* 2011). The primary importance of physical activity in preventing obesity is through the increase of energy expenditure to create a negative energy balance to ensure weight loss in the case of overweight or obesity. In the EPIC (European Prospective Investigation into Cancer and

Nutrition) study, people who were more physically active showed a 7% to 10 % less likelihood of becoming obese (Ekelund *et al.*, 2011). Waist circumference among physically active individuals was also significantly lower compared to less physically active individuals, irrespective of whether they were normal weight, overweight or obese at baseline (Ekelund *et al.*, 2011; Shook *et al.*, 2015).

Beyond increasing energy expenditure, the type, duration and intensity of physical activity determines the amount of energy expended and the type of substrate expended as fuel, whether fat or glycogen (Manore *et al.*, 2017). The more intense the activity, the more energy expended. For instance, similar amount of time spent on jogging will expend more energy compared to walking. Moderate activities such as brisk walking will result in a shift to the greater use of fat as fuel (Sahlin *et al.*, 2008; Manore *et al.*, 2017). Physical activities that also focus on strengthening muscle mass such as aerobic exercises, leads to an increase in resting metabolic rate which eventually increases the amount of energy expended. The odds of gaining > 3% fat mass among the more physically active group in one study was between 1.8 to 3.8 less after one year of follow-up compared to the less physically active group (Shook *et al.*, 2015). Muscle mass and bone mass also increased resulting in a general improvement in body composition (Office of the Surgeon General, 2004; Manore *et al.*, 2017).

The impact of physical activity on appetite regulating hormones is also reported. Although not fully understood, physical activity is believed to stimulate or increase the concentrations of appetite suppressing or satiety hormones, notably peptide YY and glucagon-like peptide, while supressing or reducing the concentration of hunger hormones, mainly ghrelin (Stensel, 2010; King *et al.*, 2015). An association was also established between less physical activity

and cravings for savoury foods and obesity (Shook *et al.*, 2015). Other studies caution the association of physical activity and reduced appetite. They suggest more studies to establish this assertion, and report that if at all, initiation of physical activity rather induces compensatory energy intake which is similar among both males and females (Thackray *et al.*, 2016). Physical activity is reported to lower LDL and triglyceride levels, raise HDL cholesterol, improve insulin sensitivity, and lower blood pressure (National Cholesterol Education Programme, 2001).

2.1.2 Methods of estimating obesity

Common anthropometric indicators often used for the estimation of obesity are body mass index (BMI) waist circumference (WC) and waist hip circumference ratio (WHR).

2.1.2.1 Estimation of obesity using body mass index (BMI)

Body mass index is a simple index of weight-for-height used to classify underweight, overweight and obesity in adults. It is calculated as weight (in kg) / height (in m^2) (WHO, 2000) and categorized as follows:

Table 2.1: Classification of weight by BMI for adult Europids (applies also to Africans)

Classification	BMI (kg/m^2)	Risk of comorbidity
Underweight,	< 18.5	Low (but risk of other clinical problems increased)
Normal weight	18.5-24.9	Average
Overweight	≥ 25.0	
Pre-obese	25.0-29.9	Increased
Obese class I	30.0-34.9	Moderate
Obese class 11	35.0-39.9	Severe
Obese class Ill	≥ 40.00	Very severe

(World Health Organization, 1995, WHO, 2000).

Body mass index is the most widely used and simple measure of body size which has proved useful in the estimation of obesity among populations worldwide (Dalton *et al.*, 2003; Gu *et al.*, 2018). The categories apply to both males and females (Ranasinghe *et al.*, 2013). In its estimation of obesity, however, BMI does not differentiate between weight from muscle and weight from fat. Hence, a given BMI may not correspond to the same degree of fatness across populations partly due to differences in body proportions (WHO, 2000).

Body mass index's effectiveness in predicting obesity and risk of disease has been established for Caucasians and extrapolated to other ethnic groups such as Hispanics and black Africans (Misra, 2015). Among people of Asian descent, especially South Asians and Chinese, risk of chronic disease has been reported at lower BMI cut-offs (WHO, 2008b; Misra, 2015; He *et al.*, 2015). Among people of other ethnicities apart from the Asians, BMI category ≥ 25.0 kgm^2 is associated with increased risk of cardiovascular disease, diabetes

and other chronic diseases. Risk of chronic disease among Asians, however, occur at lower BMI's (Table 2.2).

Table 2.2: Proposed classification of weight by BMI for Asian adults

Classification	**BMI**	**Risk of comorbidity**
Underweight	< 18.5	Low (but risk of other clinical problems increased)
Normal range	18.5-22.9	Average
Overweight	≥ 23.0	
At risk	23.0-24.9	Increased
Obese I	25.0-29.9	Moderate
Obese II	≥ 30.0	Severe

(World Health Organization *et al.*, 2000; National Institute of Health and Care Excellence, 2012; Misra *et al.*, 2009).

These have been blamed on the relatively higher abdominal fat at lower BMI's among Asians compared to other ethnicities (World Health Organization *et al.*, 2000; WHO, 2008b).

2.1.2.2 Estimation of obesity using waist circumference and waist hip circumference ratio

Waist circumference and waist hip ratio are two anthropometric indices used widely in the estimation of abdominal, central or visceral obesity among adults (WHO, 2008b). Their importance became evident following the growing prevalence of obesity in both developed and developing countries worldwide. At the time, suggestions for a revision of BMI cut-off ranges for people of Asian descent were rife (WHO, 2000). The WHO Expert Consultation

on obesity's recognition that abdominal fat could vary even within a narrow range of total body fat and BMI, fuelled the advocacy for inclusion of WC and WHR in the estimation and management of obesity (WHO, 2008b). Various cut-off ranges have been proposed by various scientific bodies for the 2 different genders as follows:

Table 2.3a: WHO waist circumference and waist hip ratio cut-off ranges

Indicator	**Cut-off point**	**Risk of metabolic complications**
Waist circumference	> 94cm (M): > 80 cm (F)	Increased
Waist circumference	> 102 cm (M): > 88 cm (F)	Substantially increased
Waist hip ratio	≥ 0.90 (M): ≥ 0.85 (F)	Substantially increased

(WHO, 2000; WHO, 2008b)

Table 2.3b: IDF waist circumference cut-offs for different ethnic groups

Population	**Men**	**Women**
Europid	> 94cm	> 80 cm
Soutn Asians, Chinese and Japanese	> 90 cm	> 80 cm

(IDF, 2006)

Table 2.3c: NCEP waist circumference reference values based on NCEP

	Men	**Women**
Waist circumference	>102 cm	>88 cm

(National Cholesterol Education Programme, 2001)

A high WHR, >1.0 in men and > 0.85 in women is associated with high risk of chronic disease. Waist circumference alone, measured at the midpoint between the lower border of the rib cage and the iliac crest, was later identified as a more practical measure of abdominal fat distribution and also correlated well with associated ill health (WHO, 2000). Waist

circumference has the advantage over WHR of being a convenient and simple measurement that is unrelated to height, that correlates strongly with BMI and WHR and gives a very close estimate of intra-abdominal fat mass and total body fat. Changes in WC also reflected changes in cardiovascular and other chronic disease risk (WHO, 2000).

While some studies have found WC to be more effective in predicting the risk of chronic disease (Misra *et al.*, 2009; Gu *et al.*, 2018; Steele-Dadzie *et al.*, 2009 ; Lean *et al.*, 1995) others have found WHR as a better predictor and yet others have reported similar predictability of risk with WC and WHR (Vazquez *et al.*, 2007; Acuin and Duante, 2013).

2.2.2.3 Estimation of obesity using body composition measurements

Although BMI effectively predicts obesity (Ranasinghe *et al.*, 2013; WHO, 2000), its ability to predict adiposity has been questioned (WHO, 2000). Various methods for measuring adiposity include; underwater weighing (densitometry), bio-electrical impedance analysis (BIA), near infrared reactance (NIR), dual-energy X-ray absorptiometry (DEXA), and magnetic resonance imaging (MRI) (Akindele *et al.*, 2016; World Health Organization *et al.*, 2000). Using these methods, the total adiposity as well as the distribution of fat (subcutaneous or visceral fat) can be estimated. Together, subcutaneous fat and visceral fat constitute the fat mass. All the other body components, namely muscle mass, bone mass and body water together constitute fat-free mass (Mahan and Escott-Stump, 2008).

The obesity status of an individual as estimated by BMI, is therefore a summation of the fat mass and the fat-free mass. While the relationship between BMI and fat mass has been described as linear, that between BMI and percentage body fat has been described as curvilinear, with a weaker association at lower BMI (Akindele *et al.*, 2016; Ranasinghe *et*

al., 2013). The association is influenced by age and in some instances gender. This implies that, fat mass increases with increasing BMI. However, at a fixed BMI, percentage body fat also increases with increasing age (Meeuwsen *et al.*, 2010). The percentage body fat increases with increasing age up to age 60-65 years in both genders, but is higher among females compared to males (WHO, 2000). Lower testosterone levels in elderly men and fat redistribution in menopausal women together result in increased total and visceral fat among elderly men and women (Traish *et al.*, 2009; WHO, 2008b). Hence among the elderly and people of Asian descent, screening for risk of chronic disease at lower BMI ranges are advised (National Institute of Health and Care Excellence, 2012). A fat mass increase of 1.9 kg per decade and a percentage body fat increase of 1.1% - 1.4% / decade was reported in a study carried out in the UK to identify the relationship between BMI and percentage body fat (Meeuwsen *et al.*, 2010). A strong positive association (r = 0.81) was also reported between BMI and percentage body fat among adult Nigerians living in urban Nigeria. Age and gender predicted the association (Akindele *et al.*, 2016).

Till date, there are no universal cut-off ranges for percentage body fat to define obesity. This is because, unlike BMI which predicted increased risk of chronic disease at 25.0 kg/m^2 and above in many epidemiological studies among various populations, no such threshold has been established for percentage body fat (Ho-Pham and Campbell, 2011). Some studies have reported a percentage body fat cut off above 25% for men and 35% for women as predictive of obesity among Caucasians (He *et al.*, 2001; Deurenberg *et al.*, 1998; Chang *et al.*, 2003). These have not been substantiated or backed by any international institution involved in obesity research and are therefore subjective. In one study, 25% body fat corresponded to a BMI of 24.6 kg/m^2 and a waist circumference of 86 cm in males while a 35% body fat

corresponded to a BMI of 22.6 kg/m^2 and a waist circumference of 73.5 cm in females in an Asian population (He *et al.*, 2001).

2.1.3 Obesity as a risk factor for chronic diseases

Although obesity on its own is often regarded as a disease, it is also a major risk factor for most chronic diseases. The risk of diseases such as various cancers, coronary heart disease, dyslipidaemia, hypertension, gallbladder disease, and diabetes mellitus are significantly increased among obese individuals (Redinger, 2007; WHO, 2008c). Additionally, obesity causes and worsens co-morbid illnesses, decreases quality of life, and increases mortality (Zhang *et al.*, 2014). In addition to the excess accumulation of fat in obesity, the regional distribution of fat also determines the severity of the risk for chronic disease. Abdominal or visceral fat is blamed for most of the risk associated with obesity and chronic diseases (Goodfriend, 2008; Hall *et al.*, 2015). The increased risk of diseases including hypertension, diabetes mellitus, dyslipidaemia, and atherosclerosis associated with obesity is closely linked to visceral adiposity (Goodfriend, 2008; Hall *et al.*, 2015). Visceral adipose tissue is believed to secret a number of adipokines including tumour necrosis factor-α, interleukin-6, plasminogen activator inhibitor-1, angiotensinogen, C-reactive protein, and leptin, most of which are pro-inflammatory, and responsible for the associated risk of disease (Kawarazaki and Fujita, 2016). This notwithstanding excess total fat mass equally infer chronic disease risk.

2.1.3.1 Obesity and the risk of hypertension

The relationship between obesity and hypertension was earlier established prospectively by the Framingham Heart Study (Kannel *et al.*, 1967). Across all ages and ethnicities, a rise in BMI increases the risk of hypertension while a reduction of weight especially among obese

and overweight individuals reduces the risk (Landsberg *et al.*, 2013). Although aging naturally increases the risk of hypertension beyond age 25 years, studies have shown that, the effect of aging on systolic and diastolic blood pressure can be neutralized if a stable healthy BMI is maintained over a long period without increasing (Lloyd-Jones *et al.*, 2007). This was reported in the Coronary Artery Risk Development in Young Adults (CARDIA) study, where young adults (mean age 25 years at baseline) who maintained a stable BMI (within 2 kg/m^2 of baseline) at 6 examinations during 15 years had no significant changes in SBP or DBP, compared to substantial increase in blood pressure among those who had an increase in their BMI $\geq$ 2 kg/m^2 (Lloyd-Jones *et al.*, 2007).

The pathogenesis of obesity and hypertension is still not clear, howbeit, a number of pathways have been suggested (Goodfriend, 2008). Although a direct impact of excess fat on hypertension is not yet identified, indirect impact mainly through stimulation of the sympathetic nervous system is highly proposed (Goodfriend, 2008; Landsberg *et al.*, 2013; Ye, 2013; Jiang *et al.*, 2016). Sympathetic nervous system stimulation through increased levels of insulin and increased leptin production in obesity have been suggested as possible mechanisms by which increased BMI may increase blood pressure (Ye, 2013). Insulin secretion in obese individuals is increased, especially if carbohydrate is mostly obtained from refined simple sugars (Jiang *et al.*, 2016). Insulin activates sympathetic nervous system, thereby increasing blood pressure. Leptin is secreted in adipocytes. Increased adipose tissue in obesity therefore increases leptin secretion. Increased circulating leptin in plasma equally activates sympathetic nervous system, leading to increased blood pressure (Landsberg *et al.*, 2013). Adipose tissue also secrets angiotensinogen, which is converted to angiotensin II in the renin angiotensin aldosterone system, one of the major control systems for blood pressure and fluid control, thereby leading to increased blood pressure (Hall *et al.*,

2015; Zhang *et al.*, 2014). Obesity also predisposes the kidney to reabsorb sodium with corresponding fluid retention and a resultant increase in arterial blood pressure (Landsberg *et al.*, 2013; Jiang *et al.*, 2016). Other studies report of visceral adipose tissue being responsible for the secretion of adipokines that stimulate aldosterone release from adrenal cells, leading to increased blood pressure (Kawarazaki and Fujita, 2016).

2.1.3.2 Obesity and the risk of type 2 diabetes

The expanded adipose tissue associated with obesity is incriminated in most of the health issues associated with obesity including type 2 diabetes mellitus. Secretion of pro-inflammatory adipokines from expanded adipose tissue in obesity is reportedly associated with insulin sensitivity, with resultant hyperglycaemia in obesity related type 2 diabetes (Shin *et al.*, 2017; Ye, 2013). Insulin concentrations vary with adiposity, and the amount of visceral fat is negatively correlated with insulin sensitivity (Zhang 2014). Obesity also results in an increase in adipose tissue macrophages, which play a role in the induction of systemic insulin resistance, a precursor for diabetes mellitus (Shin *et al.*, 2017). Obesity affects insulin concentration such that fasting and postprandial insulin levels in obese individuals are higher compared to lean individuals. This can lead to a central insulin resistance resulting in diabetes (Zhang *et al.*, 2014).

In a Korean nationwide cohort study, a reduction in BMI was significantly correlated with lower risk of diabetes (Kim *et al.*, 2018). Among a group of obese type 2 diabetes patients who were randomly divided into 2 groups, the control group receiving regular care and the intervention group receiving dietary, exercise and psychological intervention for a period of 12 months, fasting plasma glucose, 2 hours postprandial blood glucose, haemoglobin A1c, and aldose reductase were all significantly decreased (all, $P<.05$) in intervention group

compared with the control group after 12 months follow-up (Jiang *et al.*, 2017). Body weight and BMI were also significantly reduced in the intervention group. Among a group of older adults in Ghana, obesity was associated with increased odds (AOR 4.81, 95% CI: 1.92–12.0) of diabetes mellitus among women (Gatimu *et al.*, 2016).

2.1.3.3 Obesity as a risk factor for dyslipidaemia

Dietary components that predispose to obesity may also have an impact on blood lipids. Intake of foods high in dietary fat, especially if the balance between saturated and unsaturated fat is skewed towards saturated fat, increases the risk of dyslipidaemia (Jiang *et al.*, 2016). Among a group of adults in rural South Africa, obesity was one of the factors that significantly determined dyslipidaemia in a multivariate logistic regression analysis (Reiger *et al.*, 2017). The odds of elevated total cholesterol were also higher among adults with high BMI in South Western Part of Uganda (Asiki *et al.*, 2015). In Ghana, prevalence of dyslipidaemia was higher among overweight and obese children of school age than that of normal weight children (Lartey *et al.*, 2018). Weight reduction in overweight and obese individuals effectively favourably modifies atherogenic dyslipidaemia especially when physical activity is enhanced (National Cholesterol Education Programme, 2001).

2.2 Hypertension

Hypertension describes a condition of persistently high arterial blood pressure defined as a systolic and diastolic blood pressure ≥ 130/80 mmHg (ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA, 2018). Essential or primary hypertension is hypertension that has no identifiable cause or its aetiology is unknown (Mahan and Escott-Stump, 2008). Hypertension is a major public health challenge worldwide and is the number one risk factor for chronic diseases globally (Aguilar, 2017,

WHO, 2017b; Holmes *et al.*, 2013). It is responsible for 12.8% of all global deaths, representing 3.7% of total DALYS and the most important cause of premature death worldwide (WHO, 2017a). Its harmful effect is attributed in part, to the absence of obvious symptoms of the condition (silent killer), resulting in its late detection at the time when it has advanced into serious health conditions that leads to high mortality (Mahan and Escott-Stump, 2008). A global hypertension prevalence of 31.1% has been reported, which is higher in low and middle income countries (31.5%) compared to those of higher income (28.5%) (Mills *et al.*, 2016). Its awareness, treatment and control show similar trends (Mills *et al.*, 2016). Additionally, low and middle income countries have seen an increase in blood pressure levels over the past 4 decades (Aguilar, 2017).

Hypertension is a major modifiable risk factor for most cardiovascular diseases, in particular, coronary artery disease, stroke and heart failure (Owolabi *et al.*, 2017). Apart from the danger it poses to the heart, it also causes serious damage to blood vessels such as the aorta, the kidneys (leading to chronic kidney disease), the eyes and the brain (Alexanderson-Rosas *et al.*, 2015). Blood pressure is classified as follows:

Table 2.4 Classification of hypertension

Blood Pressure Classification	**SBP* mmHg**	**DBP* mmHg**
Normal	<120	< 80
Elevated	120–129	< 80
Stage 1 Hypertension	130 -139	80-89
Stage 2 Hypertension	≥140	≥ 90

*; SBP, systolic blood pressure; DBP, diastolic blood pressure.
(ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA, 2018)

2.2.1 Pathophysiology and risk factors of hypertension

About 95% of global prevalence of hypertension is idiopathic with only 5% having a known cause (Sandberg and Ji, 2012). The pathogenesis of essential hypertension is complex and multifactorial. It involves multiple hormonal and organ systems such as the sympathetic nervous system and renin-angiotensin-aldosterone system, as well as other multiple organ systems (Hamrahian *et al.*, 2017). Blood pressure is a product of cardiac output and total peripheral vascular resistance (Hamrahian *et al.*, 2017). Factors which cause an increase in any of these variables therefore results in an increase in blood pressure. Major risk factors of hypertension include obesity, the wrongful use of alcohol, physical inactivity, tobacco use and other risks including population growth, high sodium intake, ageing and exposure to persistent stress (WHO, 2013). Men have higher risk than women before the age of 45 years (Ghosh *et al.*, 2016). This difference is, however, eliminated between the ages of 45 to 64 years, where both genders have equal risk, after which the tables turn and women become more at risk from 65 years of age (Ghosh *et al.*, 2016; Everett and Zajacova, 2015). Both biological and behavioural factors account for the gender differences between males and females (Everett and Zajacova, 2015). The protective effect of oestrogen ensures lower blood pressure levels among premenopausal women. After menopause, this protection is eliminated resulting in equal risk of high blood pressure between the genders (Sandberg and Ji, 2012). Smoking among males tend to be higher compared to females. Even though males also tend to be more physically active than females, the behavioural differences taken together either increases or reduces the gender variations in hypertension (Everett and Zajacova, 2015).

Population specific risk factors also occur. In Rural Cameroon, age (≥ 40), obesity, low educational status, and being married were identified to predispose members in the

community to hypertension. Compared to those less than 40 years old, a 3-fold increase in prevalence of hypertension was reported among 40 to 59 year olds, 8.3 fold among 60 to 79 year olds and 11.6 fold for those above 80 years (Arrey *et al.,* 2016). Obese individuals were 2.8 times more likely to be hypertensive compared to non-obese. An inverse association was also reported between educational level and occurrence of hypertension, with those who never went to school having 6.7 times the odds of being hypertensive compared to those with a minimum of high school education (Arrey *et al.,* 2016). Married people were 1.5 times more likely to be hypertensive than those unmarried.

The identified risk factors significantly associated with hypertension among urban slum dwellers in Lagos State were age, gender, education, religion, BMI, and marital status (Daniel *et al.,* 2013). In both rural and urban Ghana, the predominant risk were increasing body mass index, increased salt consumption, family history of hypertension and excessive alcohol intake (Addo *et al.*, 2012).

Across the Eastern and Mediterranean Region, in addition to the global risk factors for hypertension, the main risk factor for hypertension was identified as high salt intake (Arici, 2016). Among this group, sodium intakes of between 3.74g to 4.12 g per day, obtained from between 9.35g to 10.3 g of salt intake per day, were reported (Arici, 2016).

2.3 Diabetes mellitus

Diabetes mellitus is one of the main contributors to non-communicable disease death globally (Ekpenyong *et al.,* 2012; Sommer *et al.,* 2015; IDF, 2013). Although Africa's diabetes population is currently the least among the regions, it may be largely due to the estimated 66.7% of cases that are undiagnosed. That notwithstanding, diabetes prevalence in Africa is projected to double by 2035 (IDF, 2013).

Diabetes is one of the 4 main non-communicable diseases, together with cardiovascular diseases, chronic respiratory diseases and cancers, which reduction in prevalence by 25% is being targeted by many nations across the globe by the year 2025 (Kontis *et al.*, 2014). Diabetes occurs when the body cannot produce enough of the hormone insulin or cannot use insulin effectively or both (IDF, 2013). Of the 3 different types of diabetes, type 2 diabetes is the most common, accounting for about 90% to 95% of all diagnosed cases (Mahan and Escott-Stump, 2008). Among adult Ghanaians, prevalence of type 2 diabetes in the Greater Accra Region was 6% in 2000 (Amoah *et al.*, 2000). Its prevalence among adult Ghanaians 50 years and above based on self-reporting was 3.95% (Gatimu *et al.*, 2016). The main risk factors for diabetes include age above 40 to 45 years, obesity, increased waist circumference, hypertension and family history of diabetes (International Diabetes Federation, 2012b).

Improvement in any or all of these risk factors have shown improvement in diabetes prevalence in different populations. Diet and physical activity intervention reduced body weight and improved fasting blood glucose and lipids in obese, glucose impaired adults in the FINNISH diabetes prevention study (Lindström *et al.*, 2003). The diabetes remission clinical trial (DIRECT) investigating the effect of a low fat diet, a Mediterranean diet and a low carbohydrate diet among obese adults reported significant weight reduction with the Mediterranean diet 4 years after the intervention as well as favourable glycaemic control with the Mediterranean diet (Shai *et al.*, 2008). In the Chinese Da Qing Study the cumulative incidence of type 2 diabetes during 6 years follow-up was significantly lower in the three intervention groups, diet only, exercise only, diet and exercise compared to the control (Li *et al.*, 2014). Diagnosis of diabetes may be done using either of 4 standard test as follows:

Table 2.5: Diagnosis of diabetes mellitus

	Test	Prediabetes	Diabetes
1	Fasting glucose	100-125 mg/dL (6.1-7.0 mmol/L)	≥ 126 mg/dL (7.0 mmol/L)
2	2- hour glucose following ingestion of 75-g glucose load	140-199 mg/dL (7.8-11.0 mmol/L)	≥ 200 mg/dL (11.1 mmol/L)
3	Random plasma glucose in symptomatic patient		≥ 200 mg/dL (11.1 mmol/L)
4	HbA1c		≥ 6.5% (48 mmol/mol)

Source: (International Diabetes Federation, 2017)

2.3.1 Pathophysiology of diabetes

Diabetes mostly occurs from a combination of insulin resistance or □-cell failure such that insulin levels may be normal, low or high (Mahan and Escott-Stump, 2008; Ozougwu *et al.*, 2013). Insulin's ability to clear plasma glucose is impaired due in part to tissue insensitivity to insulin, leading to a state of chronic hyperglycaemia (IDF, 2013).

In addition, obesity, a major risk factor for type 2 diabetes, causes hypertrophy of adipose tissue leading to its dysfunction. Obesity further stimulates adipose tissue endocrine and immune responses that culminates in various disorders including insulin resistance (Capurso and Capurso, 2012). The expanded adipose tissue in obesity especially that of visceral adipose, releases free fatty acids into plasma and reduces fatty acid clearance, resulting in an increased free fatty acid concentration in plasma among obese individuals compared to the non-obese. Together with obesity induced inflammatory process, the elevated free fatty acids leads to insulin resistance and consequently to diabetes mellitus (Mook *et al.*, 2004; Capurso and Capurso, 2012).

2.4 Dyslipidaemia

Dyslipidaemia describes a complex disorder in lipid metabolism. It includes high concentrations of low-density lipoprotein cholesterol and/or triglycerides and/or low concentrations of high-density lipoprotein cholesterol and high total cholesterol either as mixed or pure disorders (Anagnostis *et al.*, 2018).

Like most chronic diseases, the prevalence of dyslipidaemia is on the increase, and in most cases, the awareness is very low. In the Health and Aging in Africa Longitudinal Study in rural South Africa, among 4247 subjects investigated, overall prevalence of dyslipidaemia was 67.3%, out of which 1.05% were aware of their condition and 0.73% were receiving treatment (Reiger *et al.*, 2017). Elevated total cholesterol prevalence was 37.66%, elevated LDL-c was 36.71%, low HDL-c was 40.10% and elevated triglyceride was 59.30% (Reiger *et al.*, 2017). In the South African national nutrition and health survey, among adults 18 years and above, elevated total cholesterol was found in 28% of women and 19% of men, while 52% of men and 44% of women had low levels of HDL (Shisana *et al.*, 2013). In a rural population in South Western Uganda, prevalence of elevated total cholesterol was 6.0%, while that of low HDL-c was 71.3% (Asiki *et al.*, 2015). In that population, factors independently associated with decreasing HDL included intake of less than 5 servings a day of fruits and vegetables, low education and alcohol intake. In a group of adults in semi urban Nigeria, elevated total cholesterol, high low-density lipoprotein-cholesterol, elevated triglyceride, and low high-density lipoprotein were seen in 5.3%, 19.3%, 4.4%, and 76.3% of the participants, respectively (Olamoyegun *et al.*, 2016). Among a group of school age children in urban Ghana, the proportion of children with elevated total cholesterol was 12.1%, elevated triglyceride was 4.5%, low HDL-c was 28.4% and elevated LDL-c was 9.2%. Overweight and obese children recorded higher levels of dyslipidaemia compared to

normal weight children. In addition, frequent fruit and vegetable intake was associated with low levels of dyslipidaemia (Lartey *et al.*, 2018). In contrast, among adults in the Cape Coast metropolis, dyslipidaemia of total cholesterol and LDL cholesterol was higher among normal weight adults compared to their overweight obese counterparts (Acquah *et al.*, 2017). Desirable levels of the various components of blood lipids are as follows:

Table 2.6 Definition of dyslipidaemia

Blood lipid component	Desirable levels
Total Cholesterol	< 5.2 mmol/L
LDL Cholesterol	< 3.4 mmol/L
HDL Cholesterol Males Female	 >1.0 mmol/L >1.3mmol/L
Triglyceride:	< 1.7 mmol/L

Source: (National Cholesterol Education Programme, 2001)

2.4.1 Pathophysiology of dyslipidemia

Two main classifications of dyslipidaemias are primary and secondary dyslipidaemias. While primary dyslipidaemias are mainly of genetic origin, secondary dyslipidaemia occurs secondary to an underlying condition, including diabetes mellitus, hypothyroidism, chronic kidney disease, nephrotic syndrome and human immunodeficiency virus (HIV) infection (Anagnostis *et al.*, 2018). There are no known causes of dyslipidaemia. However, some factors have been identified to increase their risk of development. Overweight / obesity, physical inactivity, cigarette smoking, excess alcohol intake, very high carbohydrate intake and some medications such as protease inhibitor for management of HIV and oestrogens, can increase the risk of dyslipidaemias (National Cholesterol Education Programme, 2001).

Obesity mediated dyslipidaemia is characterized by elevated fasting and postprandial triglyceride, together with the increased small dense LDL and low HDL-C (National Cholesterol Education Programme, 2001). Most of these originates from adipokines secreted by adipocytes in adipose tissue, especially visceral adipose tissue (Anagnostis *et al.,* 2018; Klop *et al.,* 2013)

2.4.2 Dietary management of dyslipidaemia

The management approach of dyslipidaemia is in two fronts; controlling dietary intake and reducing weight in overweight / obesity (Reiger *et al.,* 2017; Anagnostis *et al.,* 2018). It is generally ensured that all sources of secondary dyslipidaemia are eliminated, mainly through weight reduction in overweight/ obesity by 5% to 10% respectively (Anagnostis *et al.,* 2018). To achieve this, reducing energy intake by between 500–750 kcal/d or 30% of daily energy intake resulting in a daily energy intake of between 1200 to 1500 kcal/d for women and 1500 to 1800 kcal/d for men, may be necessary, coupled with regular physical activity (Jensen *et al.,* 2014). This suggest that fat and carbohydrate intake must both be reduced. Some studies suggest that reduction of carbohydrate intake alone to < 20% energy/day achieves greater weight reduction even though it also results in the elevation of both HDL and LDL cholesterol (Mansoor *et al.,* 2015). Other studies suggest that reducing carbohydrate intake alone leads to the loss of more weight than reducing fat intake, although the difference is lost by 12 months (Nordmann *et al.,* 2006). Also reducing fat intake gives more favourable lipid profile compared to reducing carbohydrate intake (Nordmann *et al.,* 2006; Hession *et al.,* 2009). The diabetes remission clinical trial (DIRECT) investigating the effect of a low fat diet, a Mediterranean diet and a low carbohydrate diet among obese adults reported significant weight reduction with the Mediterranean diet 4 years after the

intervention (Shai *et al.*, 2008) as well as favourable blood lipids with the low carbohydrate diet (Yu-Poth *et al.*, 1999).

Reduction of saturated fat intake to less than 10% of daily energy and avoiding trans fat are mandatory to achieve improvement in blood lipid profile (Anagnostis *et al.*, 2018). The best results are achieved when saturated fatty acids in the diet are replaced with mono and polyunsaturated fatty acids and not with carbohydrates (Reiger *et al.*, 2017). Substituting saturated fats with omega-3 fatty acids, eicosapentaenoic (EPA) and docosahexaenoic (DHA)], which are mainly found in fish oils and nuts, reduces the synthesis of very-low density lipoprotein in the liver and hence reduce serum triglyceride levels (Anagnostis *et al.*, 2018; Mansoor *et al.*, 2015).

2.5 Major risk factors of non-communicable diseases

Non communicable diseases share many common modifiable risk factors which are mostly lifestyle related. The main components of lifestyle implicated are unhealthy dietary intake, physical inactivity, smoking and alcohol intake.

2.5.1. Diet and non-communicable diseases

Different dietary components have different influence on health. While some components are reported to improve health and are therefore recommended for frequent intake, others are associated with risk to health when consumed too often or in large quantities and must therefore be restricted or avoided. Foods that are high in salt, saturated fats, cholesterol and refined sugars and sweetened beverages are best avoided. Those that contain high fibre, from cereals, legumes fruits or vegetables as well as fish and lean foods, low fat dairy products and nuts are recommended (Jiang *et al.*, 2016)

2.5.1.1 High sodium diets

Over the past few decades, it has become evident that high sodium intake increases the risk of cardiovascular diseases especially hypertension. The INTERSALT study. Which was a worldwide epidemiologic study tested the hypothesis on 24-h urine excretion and blood pressure and found a positive independent linear relationship between 24h urine excretion and systolic blood pressure (Stamler, 1997). Other studies have since confirmed the relationship between high salt intake and blood pressure (He and MacGregor, 2010; Kong *et al.,* 2016). The dietary approach to stop hypertension (DASH) sodium study further established that progressively reducing the amount of sodium intake progressively reduces systolic blood pressure (Sacks *et al.,* 2001). Based on this body of evidence, different institutions have recommended various levels of restricted sodium intake to maintain good health among normotensive, pre-hypertensive and hypertensive patients.

Table 2.7: Recommended sodium intake by different institutions for different target groups.

Recommended sodium intake	Target population	Recommending institution	Reference
≤ 2,300 mg/day (100 mmol/24 h) ≤ 1,500 mg/day (65 mmol/24 h)	General population Higher risk subgroups	The 2010 dietary guidelines for Americans	(US Department of Agriculture and US Department of Health and Human Services, 2010)
< 1,500 mg/day (65 mmol/24 h)	General population	American Heart Association (2010 guidelines	(Lloyd-Jones *et al.*, 2010)
< 2,000 mg/day (87 mmol/24 h)	For adults	WHO 2012 guidelines	(World Health Organization, 2012)
< 2,300 mg/day (100 mmol/24 h) < 2,000 mg/day (87 mmol/24 h)	Hypertensive or normotensive Patients with diabetes and symptomatic heart failure	The American Diabetes Association	(Bantle *et al.*, 2008)
< 90 mmol/24 h	Prevent progression of chronic renal disease	KDIGO, 2012	(Kidney Disease: Improving Global Outcomes CKD Work Group, 2013)

In addition to the increased risk of hypertension associated with the intake of large amounts of salt, high sodium intake is also reported to increase cardiovascular disease risk

independent of hypertension, through its association with left ventricular hypertrophy, an independent predictor of cardiovascular disease risk (Kong *et al.,* 2016).

2.5.1.2 High fat diets

Dietary fats are an important component of human diets. They are macronutrients which play very vital roles in the human body. They are useful in ensuring that energy needs are met due to their relatively higher caloric content (Mahan and Escott-Stump, 2008). They are needed for the digestion, transport and absorption of soluble vitamins and phytochemicals including carotenoids and lycopene (Mahan and Escott-Stump, 2008). They ensure the maintenance of the human structure by providing structural support for internal organs and also offers protection from injury (Duyff, 2002; Mahan and Escott-Stump, 2008). When stored as subcutaneous fat, they also provide insulation for the body in order to maintain the body's temperature (Mahan and Escott-Stump, 2008).

Due to their high caloric content excess fat intake in our diets leads to obesity due to storage of excess fat in adipose tissue, especially white adipose tissue (U.S. Department of Health and Human Services and U.S. Department of Agriculture, 2015). White adipose tissue produces adipokines that are responsible for chronic inflammation processes which are associated with obesity-related metabolic diseases including cardiovascular disease, diabetes, hypertension and dyslipidaemia (Narasimhan *et al.,* 2016; Mancini *et al.,* 2015).

Fat from diet immediately increases the blood lipid and cholesterol levels and can increase the risk of atherosclerosis and its concomitant complications (Jiang *et al.,* 2016). High fat intake is also suggested to diminish nitric oxide bioavailability, resulting in a disruption of

the nitric oxide mediated endothelium-dependent vasodilation, which can potentially increase the risk of cardiovascular diseases (Dow *et al.,* 2015).

A study carried out on adult Indians from the rural component of the Chennai Urban Rural Epidemiological Study, found that those who were in the highest quintile of dietary fat intake reported significantly higher prevalence of abdominal obesity, hypertension and impaired fasting glucose (Narasimhan *et al.,* 2016).

In addition to the quantity of fat consumed, the quality of fat consumed is also a health concern. Fats that have more unsaturated fatty acid fractions promote better health than those with higher saturated fatty acid fractions (Narasimhan *et al.,* 2016). Intake of diets high in saturated fatty acids are suggested to expanded saturated fatty acid depot in white adipose tissue, which stimulates pro-inflammatory mechanisms leading to the down-regulation of insulin-signalling in insulin target cells. This provides a possible saturated fatty acid-mediated inflammatory response resulting in insulin resistance (Mancini *et al.,* 2015; Jager *et al.,* 2007; Wen *et al.,* 2011). High saturated fat intake is also reported to alter gut microbiota components that determines lipid accumulation (Mancini *et al.,* 2015; Musa *et al.,* 2012). In contrast, total fat and types of fat were not associated with cardiovascular disease, in the Prospective Urban Rural Epidemiology (PURE) study (Dehghan *et al.,* 2017)

In a randomized controlled trial, individuals who were put on a low fat diet (23% of energy) with a high polyunsaturated/saturated ratio (1·0) showed the lowest reduction in systolic and diastolic blood pressure compared to those who were put on a low salt diet as well as the control group after 6 weeks of intervention (Puska *et al.,* 1983).

It is recommended that total fat should constitute 25% to 35% of total daily energy intake, comprising < 7% of saturated fats, up to 10% polyunsaturated fats, up to 20% monounsaturated fats and less than 200 mg of cholesterol per day (National Cholesterol Education Programme, 2001). The American healthy eating guidelines recommendation for oils at the 2,000-calorie level is 27 g (about 5 teaspoons) per day (U.S. Department of Health and Human Services and U.S. Department of Agriculture, 2015). For Ghanaians, the recommended contribution of fat to total daily energy intake is 10% -15% (Ministry of Health, 2010).

In most developing countries including Ghana, domestic production and use of various oil seeds is common (Popkin, 2002). Palm oil and palm kennel oil are common vegetable oils used in most Ghanaian diets, especially among commercial food vendors. They are both high in saturated fatty acids. Palm kennel oil contains 85% saturated fatty acids, mainly lauric and myristic acids, while palm oil contains 50% saturated fatty acids, mostly palmitic (44%) and lower amounts of stearic acid (5%), 40% monounsaturated fatty acids, mostly oleic acid, and 10% polyunsaturated fatty acids, mostly linoleic acids (Mancini *et al.,* 2015). Although palm oil is used in the preparation of most Ghanaian meals, the Ministry of Health in Ghana recommends the use of less saturated vegetable fats such as canola oil and soy oil over the more saturated ones such as palm oil, coconut oil and shea butter (Ministry of Health, 2010). Replacing saturated fats with unsaturated fats, especially polyunsaturated fats, is associated with reduced blood levels of total cholesterol and of low-density lipoprotein-cholesterol (U.S. Department of Health and Human Services and U.S. Department of Agriculture, 2015).

2.5.1.3 Excess carbohydrate intake

Carbohydrates represent a vital component of most human diets. They are the primary source of energy for all human activities providing over 50% of the total energy needs of humans (Leturque *et al.*, 2012). Among people in poorer and developing countries, it may provide as much as 90% of the energy in their diets (Macdonald, 2003). The recommended daily allowance (RDA) of carbohydrate is 130g/d (Wolfe *et al.*, 2017, Institute of Medicine of the National Academies, 2002), although for purposes of weight loss or management of some chronic diseases, a reduction in carbohydrate intake may be recommended (International Diabetes Federation, 2012b).

Weight loss targets of an initial 5-10% in 3 to 6 months is recommended (NICE, 2015; International Diabetes Federation, 2012b). Losing 1lb, approximately 2kg a week translates into losing 3,500 calories, hence restricting daily energy intake by 500 calories, is estimated as a realistic healthy target (British Dietetic Association, 2016). Energy restriction diets with carbohydrate as low as 50g-100g per day has been reported as safe for a short-term (up to 6 months) among obese type 2 diabetes patients. Reduction of daily energy intake by 500 to 600 calories is encouraged for such patients (International Diabetes Federation, 2017). In addition, limiting energy intake to 800 to1200 calories to achieve a 10kg weight reduction in 6 months is recommended (International Diabetes Federation, 2017).

High intake of carbohydrates is associated with atherogenic dyslipidaemia, characterized by elevated triglycerides, reduced HDL concentrations and elevated small density lipoprotein cholesterol, which endanger cardio metabolic health and increase cardiovascular diseases and total mortality (Siri-Tarino *et al.*, 2015). Elevated triglyceride and reduced HDL are part

of the criterial for the diagnosis of the metabolic syndrome (National Cholesterol Education Programme, 2001)

Excess intake of carbohydrate in the diet is converted into fat and stored in adipose tissue which can result in obesity (WHO, 2008b). Obesity, especially visceral obesity stimulates pro-inflammatory mechanisms leading to insulin resistance, diabetes as well as hypertension (Narasimhan *et al.*, 2016; Mancini *et al.*, 2015). Carbohydrate refined starches as well as sweetened beverages increases post prandial plasma sugar which is associated with insulin resistance and diabetes mellitus. Excess carbohydrate intake contributing more than 60% of total daily energy intake contributes to elevated serum triglyceride levels (National Cholesterol Education Programme, 2001)

2.5.1.4 Foods low in dietary fibre

Inadequate dietary fibre intake is a risk factor for many chronic diseases (Threapleton *et al.*, 2013; Doménech *et al.*, 2014; Sandberg *et al.*, 2017). Adequate dietary fibre intake is especially useful for maintaining good health and also for the management of conditions such as diabetes, hypertension, higher cholesterol and obesity (U.S. Department of Health and Human Services and U.S. Department of Agriculture, 2015; Ministry of Health, 2010). The American Diabetes Association recommends a dietary fibre intake of 14g / 1000 kcal for diabetes patients (American Diabetes Association, 2012). This translates to 25g of dietary fibre for a 1800 kcal diet. Between 20g to 30g of dietary fibre intake per day have also been recommended in lowering blood lipids to reduce coronary heart disease risk (National Cholesterol Education Programme, 2001; Huang *et al.*, 2015). Others recommend 30g per day intake of dietary fibre as part of lifestyle changes to reduce the risk of chronic disease for adults aged 18 years and above (The British nutrition foundation, 2018,

Australian National Health and Medical Research Council (NHMRC) and the New Zealand Ministry of Health (MoH), 2005). Daily dietary fibre intake of 25g for women and 38g for men has also been recommended (Institute of Medicine of the National Academies, 2002). The recommended dietary fibre intake for adult Ghanaians is at least 25g per day, which can be achieved by consuming at least 5 servings of fruits and vegetables a day, in addition to 6 servings of grain products daily, 3 servings of which should be whole grain products (Ministry of Health, 2010).

Increasing dietary fibre intake reduces the risk of cardiovascular disease and coronary heart disease (Threapleton *et al.*, 2013; Hajishafiee *et al.*, 2016; Mozaffarian *et al.*, 2003). Whole grains, fruits and vegetables are the main contributors of dietary fibre in our diet, contributing over 70% of daily dietary fibre intake (Storey and Anderson, 2014). The contributions by fruits and vegetables are, however, modest due to their higher moisture contents (Storey and Anderson, 2014). Mostly, all dietary sources of fibre contain a combination of both soluble and insoluble fibre. However, fruits, vegetables, legumes and oat bran contain a greater proportion of the soluble fibre while whole grains and cereals have a greater proportion of insoluble fibre (Hajishafiee *et al.*, 2016; McKee and Latner, 2000). Both soluble and insoluble dietary fibre have been associated with increasing insulin sensitivity with an associated improvement in blood glucose levels. The protective effect of dietary fibre on blood glucose was previously associated with soluble fibre. Their gel forming, viscous, easily fermentable nature facilitated interaction with fluids in the gastrointestinal tract, leading to the formation of a viscous solution barrier through which sugar and glucose journeyed on their way to the intestinal brush border for absorption (Schulze *et al.*, 2007; Weickert and Pfeiffer, 2008). This slowed glucose absorption with subsequent control of post-prandial blood glucose (Hajishafiee *et al.*, 2016). This blood

glucose control effect is increasingly showing stronger association with insoluble fibre rather than soluble fibre (Weickert and Pfeiffer, 2018; Johansson *et al.,* 2013; Sandberg *et al.,* 2017), while soluble fibre from fruit and vegetable sources shows moderate association with type 2 diabetes prevention. Higher intake of cereal fibre is also associated with reduced risk of CVD and all-cause mortality (Sandberg *et al.,* 2017; Johansson *et al.,* 2013). Soluble dietary fibre has, however, strong association with weight loss for the control of overweight and obesity while cereal fibre has shown moderate effect (Weickert and Pfeiffer, 2018).

The mechanism by which cereal fibre protects against all-cause mortality, CVD and diabetes are not well understood. It is conjectured to be related to the rich micronutrient content of foods rich in cereal fibre. Foods rich in cereal fibre are rich sources of magnesium, folate, selenium, zinc, copper, antioxidants, phenolic acids, phytochemicals and lignans. These components are believed to reduce inflammatory markers including C-reactive protein and tumour necrosis factor- α (TNF-α) receptor 2 (Weickert and Pfeiffer, 2008; Buyken *et al.,* 2010). The reduction of inflammatory markers in part provides a possible explanation to the observed inverse association of cereal fibre intake with all-cause mortality (Huang *et al.,* 2015; Wannamethee *et al.,* 2009; Ma *et al.,* 2006; Buyken *et al.,* 2010).

It is therefore recommended that patients increase their intake of fruits, vegetables as well as whole grain and cereals to increase their dietary fibre intake. A minimum of 5 servings of fruits and vegetables daily, with 6 servings of grain foods, preferably 3 servings being whole gain, is advised (Ministry of Health, 2010).

In a meta-analysis of six cohort studies including 286,125 participants and 10,944 cases, a two servings per day increment in whole grain consumption was associated with a 21%

(95% CI, 13–28%) decrease in risk of type 2 diabetes after adjustment for potential confounders. Other meta-analysis confirmed these results and also reported that compared with never/rare consumers of whole grains, individuals consuming 48 to 80 g of whole grains per day (3 to 5 serving/day) had a 21% lower risk of CVD (relative risk = 0.79; 95% CI, 0.74 –0.85). Inverse associations were also reported between intake of whole grains and incident hypertension (Huang *et al.*, 2015)

2.5.2 Excess alcohol intake

Alcohol intake is a risk factor for many chronic diseases. Its ability to contribute to disease risk depends on the amount consumed, the drinking patterns as well as the quality of alcohol consumed (Shield *et al.*, 2014). While it is entirely responsible for causing some conditions, for others, it plays a complimentary role as a risk factor. For conditions such as diabetes, stroke and ischemic heart disease, alcohol may even have a beneficial role as well as an adverse role depending on how much alcohol is consumed (Shield *et al.*, 2014). Alcohol is reported to have contributed significantly to the estimated 35 million deaths and 603 million disability-adjusted life-years (DALYs) which were lost to chronic diseases and conditions globally in 2004 (WHO, 2004). The WHO and other international bodies recommend a safe daily alcohol intake of 2 drinks for men and 1 drink for women (International Diabetes Federation, 2012b; American Diabetes Association, 2012; WHO, 2008c). A drink is defined as 120 ml of wine (½ medium glass of dry wine) or 285 ml of beer (half of large beer bottle or one full mini) or one bottle of Guinness or 30ml (one tot) of spirit, whisky, gin, *akpeteshie*, "*woba ada anaa*", other alcoholic bitters or 60ml of brandy, vermouth or aperitif (WHO, 2008a). People who do not drink alcohol were encouraged to continue abstaining from alcohol.

There are mixed reports on the effect of alcohol on cardiovascular and circulatory diseases. Alcohol is said to increase the risk of hypertension at all consumption levels for men and at higher consumption levels for women (Shield *et al.,* 2014). The likely explanation given is that alcohol enhances the activity of the sympathetic nervous system, which leads to a greater constriction of the blood vessels and makes the heart contract more strongly. Constriction of blood vessels results in the increase of blood pressure (Siddiqui, 2011). At higher intakes, alcohol consumption also increases the risk of ischemic stroke (Shield *et al.,* 2014). Alcohol intake additionally interferes with mechanisms involved in the generation and transmission of electrical signals that coordinate the heart, leading to conduction disorders and other dysrhythmias (Samokhvalov *et al.,* 2010).

Conversely alcohol is also reported to protect against cardiovascular disease when consumed in moderation (Djoussé *et al.,* 2009). Its protective effect is linked with its HDL raising effect as well as its ability to reduce platelet aggregation on arterial walls (Djoussé *et al.,* 2009; Shield *et al.,* 2014). A meta- analysis involving 28 prospective studies investigated the relationship between alcohol intake and coronary heart disease risk (CHD), and reported that 20 g of alcohol a day (equivalent to 1–2 standard drinks) was associated with a 20% (95% confidence index [CI] 17% to 22%) reduction in the relative risk of CHD. This protective effect was found to persist up to a consumption as high as 72 g/day and only became significantly harmful after 89 g/day (approximately 7 standard drinks a day) (Corrao *et al.,* 2000). Moderate alcohol intake is also associated with reduced risk of diabetes through the increase in insulin sensitivity (Shield *et al.,* 2014). The association is, however, dose dependent such that at higher intakes of alcohol, the risk of diabetes rather increases due in part to its contribution to weight gain. In a meta-analysis which investigated 20 cohort studies, a U-shaped relationship was found for both genders. Compared with lifetime

abstainers, the relative risk for type 2 diabetes among men was most protective at alcohol intake of 22 g/day (RR 0.87 [95% CI 0.76 –1.00]). Alcohol intake ≥ 60 g/day was, however, harmful, (1.01[0.71–1.44]). Among women, alcohol was at its highest protectiveness when 24 g/day was consumed (0.60 [0.52– 0.69]), but harmful at about 50 g/day (1.02 [0.83–1.26]) (Shield *et al.*, 2014; Baliunas *et al.*, 2009).

2.5.3 Physical inactivity

The effect of physical inactivity on chronic disease has earlier been reviewed in this report under causes of obesity. To maintain good health as well as manage chronic diseases, engaging in moderate physical activity for at least 150 minutes weekly is recommended (NICE, 2015; American Diabetes Association, 2012; International Diabetes Federation, 2017; Ministry of Health, 2010). This translates into a minimum of 30 minutes daily for at least 5 days in a week. Moderate physical activity include brisk walking, cycling, dancing, jogging or horse riding (WHO, 2008a). A dose response relationship between physical activity and hypertension has been reported (Diaz and Shimbo, 2013). Increasing physical activity through increase in intensity, frequency or duration results in additional health benefits (Warburton *et al.*, 2010). As long as similar amount of energy are expended, the intensity of physical activity, whether moderate or vigorous has similar health benefits (Diaz and Shimbo, 2013; Warburton *et al.*, 2010).

2.5.4 Smoking habits

Smoking is a major risk factor for most non-communicable diseases especially cardiovascular disease, diabetes and certain cancers (WHO, 2008c; National High Blood Pressure Education Program, 2004). It is also a risk factor for the major non-communicable disease risk factors such as obesity and hypertension. An association between smoking and

obesity has been reported. Among middle aged adults in the UK general population, those who currently smoked reported lower odds of obesity compared to those who never smoked as well as those who quit smoking, with obesity increasing with increasing years of quitting smoking (Dare *et al.,* 2015). It was explained that nicotine is an appetite suppressant as well as a metabolic stimulant. It therefore reduces the amount of food consumed while increasing energy expenditure from metabolic activities even when physical activity is unchanged, leading to weight loss (Courtemanche *et al.,* 2016; Watanabe *et al.,* 2016). Obesity among smokers, however, increased with increasing pack and number of sticks smoked in a day (Watanabe *et al.,* 2016; Dare *et al.,* 2015). Contrary reports suggests that, smoking increases the risk of obesity through the reduction of lung capacity which leads to a reduction in energy expenditure from physical activity (Courtemanche *et al.,* 2016).

Smoking is a major risk factor for cardiovascular diseases (Hozawa *et al.,* 2007). Smoking cessation is regarded as the single most effective lifestyle measure for the prevention of a many cardiovascular diseases (Virdis *et al.,* 2010). Smoking increases high blood pressure through the stimulation of the sympathetic nervous system. Its role in the alteration of antithrombotic and pro-thrombotic factors, endothelial function, arterial stiffness, inflammation and lipid modification further speeds up atherothrombotic processes that increases the risk of other cardiovascular diseases (Virdis *et al.,* 2010). People who smoke were advised to stop smoking and also avoid passive smoking (National Cholesterol Education Programme, 2001; National Institutes of Health, 2004; Ministry of Health, 2010).

2.5.5 C-Reactive Protein as a risk factor for non-communicable diseases

C- reactive protein is an acute phase reactant that is synthesized and released by various cells in response to microbial infection, tissue injury, inflammation stimulus or cell damage

(Haider *et al.*, 2006). Due to the direct role it plays in the development of atherosclerosis, it is regarded as an independent predictor of cardiovascular diseases (Smidowicz and Regula, 2015). Serum C-reactive protein level is therefore a suitable marker for assessing the effectiveness of therapeutic interventions to reduce CVD risk (Krajcovicova-Kudlackova and Blazicek, 2005; Avan *et al.*, 2018). C-reactive protein is produced in various parts of the body. In the liver, it is produced in response to the presence of interleukin-6. To a lesser extent, it is also produced by non-hepatic cells like neurons, atherosclerotic plaques, monocytes, Kupffer cells and lymphocytes (Chandrashekara, 2014). C- reactive protein plays a role in various immunological processes in the defence of the body against pathogen, especially bacteria, injury and inflammations (Chandrashekara, 2014; Haider *et al.*, 2006).

Due to its association with Il-6, conditions that result in increased blood levels of Il-6 also increases the secretion of C-reactive protein (Ma *et al.*, 2006). Diabetes mellitus, obesity and some cardiovascular diseases such as atherosclerosis are related to increased levels of pro-inflammatory cytokines, including Il-6 (Salazar *et al.*, 2014). Hence C-reactive protein levels are elevated in diabetes and cardiovascular diseases.

Dietary factors that increase the risk of these diseases also tend to increase the plasma levels of C - reactive protein. Low fibre, high sugar sweetened and refined carbohydrates increase the risk of obesity and diabetes which increases the pro-inflammatory cytokines plasma interleukin-6, tumour necrosis factor α, interleukin-18 and invariably, C-reactive protein (Ma *et al.*, 2006). Adequate intake of dietary fibre is associated with the decreased oxidation of lipids, which in turn is associated with decreased inflammation (Ma *et al.*, 2006). In a study to investigate CRP levels among people who practised different models of diet, CRP levels were lowest among vegetarians compared to non-vegetarians (Krajcovicova-

Kudlackova and Blazicek, 2005). Among the non-vegetarians, a significant positive correlation was found between age and CRP, while among the vegetarians, CRP levels were low and non-age dependent. High levels of anti-inflammatory compounds in fruits and vegetables were cited as the reason for the observed low CRP levels among vegetarians (Nanri *et al.*, 2007; Krajcovicova-Kudlackova and Blazicek, 2005). High intakes of carotenoids and vitamin C, were also associated with decreased levels of circulating CRP. Food models based on whole grains, fruits, nuts, and green leafy vegetables was significantly inversely correlated with serum concentrations of CRP (Smidowicz and Regula, 2015). Foods high in total fat as well as saturated and trans fat, e.g. processed meats, full-fat dairy products, French fries were also associated with increased serum CRP levels (Basu *et al.*, 2006).

Age and gender differences in serum CRP levels have also been reported. In their study, Khera *et al.*, 2005, reported high CRP levels among women compared to men and among black Americans compared to whites (Khera *et al.*, 2005). Significantly higher CRP levels were also reported among people of African, Latin American, and Asian descent compared to those of European ancestry (Aydin and Cesar, 2007). Modifiable risk factors, mainly BMI was reported to be responsible for most, but not all of the ethnic differences observed (Kelley-Hedgepeth *et al.*, 2008). The higher prevalence of obesity, hypertension and diabetes among African American, followed by Hispanic Women, compared to Chinese and Japanese women were some of the reasons given for the observed differences in their CRP levels (Kelley-Hedgepeth *et al.*, 2008). In addition, the higher prevalence of the metabolic syndrome in Hispanic women, with corresponding higher triglyceride, LDL levels and lower HDL levels potentially explained their relatively higher CRP levels compared to women of Asian or European descent (Kelley-Hedgepeth *et al.*, 2008). Additionally, lower

physical activity and education levels among Hispanic women and higher smoking prevalence among African American women contributed to their higher CRP levels. Significant inverse correlations were observed between CRP and HDL among African, Hispanic, Asian and Caucasian ancestry but systolic blood pressure, BMI and waist hip ratio showed significant linear correlations with CRP among all groups (Kelley-Hedgepeth *et al.*, 2008). In a cohort of Korean adults, a strong significant correlation was reported between serum CRP and various anthropometric indicators including body weight, BMI, WC, hip circumference, and the waist-hip ratio (Smidowicz and Regula, 2015).

C-reactive protein reference range of 0.2 mg /L to 3.0 mg/L has been cited as a healthy range (Fauci *et al.*, 2008).

2.5.6 Socioeconomic Status

The common socioeconomic indicators often investigated in relation with health are education, occupation and income (Duncan *et al.*, 2002). In a study to investigate the impact of each of these indicators on CVD risk factors, Winkleby et at., (1992) concluded that higher education status was the best predictor of good health. Although all paired correlation were positive, correlation between education and income was the lowest. They explained that education was not the primary predictor of wage. A higher correlation occurred between education and occupation suggesting that the skills acquired in education to a large extent, determined an individual's occupation (Winkleby *et al.*, 1992), and subsequently their health, through the work conditions they are exposed to and their levels of consumptions (Duncan *et al.*, 2002). In their study, participants with lower education consistently exhibited higher risk for all CVD risk factors while income and occupation were less consistent (Winkleby *et al.*, 1992). Higher educational levels have also been associated with health enhancing and health maintenance behaviours, while health endangering behaviour

among adults have been associated with low education, defined as less than a high school education (Franks *et al.,* 2011, Ettner and Grzywacz, 2003), from poor childhood background or having blue collar jobs (Lynch *et al.,* 1997). People with low education are less likely to stop smoking. For those who may be on antihypertension medication, their inability to afford these medications may result in discontinuing their use leading to serious complications (Franks *et al.,* 2011). In general, a low socioeconomic status, especially low education increases an individual's exposure to environmental hazards due to poor sanitation and pollution of their residential areas (e.g. being close to mining and other industrial areas), overcrowding, high smoking rates and polluted sources of water compared to their counterparts of higher socioeconomic status. This increases their risk of developing various chronic and other infectious diseases (Evans and Kantrowitz, 2003). In contrast, some studies have also found high socioeconomic status to be associated with increased disease risk. A significant linear association between diabetes and higher education and wealth has also been reported (Gatimu *et al.,* 2016). In their study, respondents who had university and secondary education were 5 and 2 times more likely to have diabetes than those who had never been to school respectively, largely due to the nutrition transition towards the intake of more refined foods with less dietary fibre and more sedentary lifestyle among the educated compared to the uneducated (Gatimu *et al.,* 2016).

2.6 Dietary counselling approaches used in the hospital

Traditionally, the work of a dietitian has been considered as that of a teacher, an adviser or a guide (Gable, 2007). They provide service to the sick, people who may be living with some chronic condition as well as apparently healthy people who are concerned with maintaining and promoting their health. Their role is to provide unbiased food and nutrition information that is culturally sensitive, scientifically accurate, medically appropriate and

feasible for that particular client. In short, their work involve recommending dietary behaviour change through brief advice-giving (Martins and McNeil, 2009). The challenge is that, after they have given off their best to inform, educate and facilitate dietary behaviour change, some patients still refuse to comply (Gable, 2007). The proportion of patients who completely adhere to dietary advice is often minimal. In one study conducted in Egypt, as low as 2.2% of respondents followed dietary advice (Ibrahim *et al.,* 2010), while in another at the USA, 52% of diabetic patients followed dietary advice (Anderson and Gustafson, 1998). About 25.2% of patients with type 2 diabetes are reported to have strictly adhered to dietary advice while 63.2 % adhered sometimes with 11 % completely ignoring dietary advice (Al-Sinani *et al.,* 2010). In their study, Parajuli *et al.,* (2014), reported that 82.5% of type 2 diabetes patients were nonadherent to dietary advice while 12.5% poorly adhered. The determinants of nonadherence to dietary advice was identified in a study as female gender, increasing age, joint or extended family members (members in a nuclear family are more economically secure to afford recommended foods compared to those in extended families), farther distance of residence from hospital, poor knowledge about diabetes mellitus and advice given by health personnel other than physicians. Determinants of nonadherence to physical activity were identified as negative family history of diabetes mellitus, divorce status and lower socioeconomic status (Parajuli *et al.,* 2014). Literate groups have also been reported to significantly adhere to dietary guidelines ($p < 0.0005$) compared to their illiterate counterparts (Gundala *et al.,* 2016).

A qualitative study to identify the factors why patients do not adhere revealed the different levels of influence on participants' dietary behaviour (Ebrahim *et al.,* 2014). Beginning from individual level, to small group family and friends, organizational or health systems as well as community and policy level. Individual level factors included motivation, individual

knowledge, perceptions of moderation, self-responsibility, taste concept or cravings, and temptations. Family relations were the main support system for managing their conditions while factors at the health system level included long waiting times and seeing different doctors. Culture and cost of food were the main factors affecting patient's adherence to dietary advice at the community level (Ebrahim *et al.*, 2014).

Common dietary counselling approaches used in the hospital include directive, guided or motivational counselling.

2.6.1 Directive Counseling

This approach is often employed and suitable in acute care setting when an urgent dietary change may make the difference as to whether a patient lives or dies. The primary objective is to bring a patient out of a critical, emergency or high-risk situation and stabilize them. It is not preferred if the objective is to achieve a permanent behavioral change for developing a new habit pattern. It primarily involves the use of instructional language leaving little room for discussion, with high risk of non-compliance (Cant and Aroni, 2008).

2.6.2 Guided Counseling

Guided counselling allows room for discussion between patient and dietitian with the dietitian staying in control of the discussion. It often employs the use of more open ended questions. Through the open discussion, issues that may be the root problem of the eating problem may be easily identified and addressed. The dietitian does a lot of listening and reflects on what is being said to offer appropriate counsel. The patient in-turn hears what is said and may be given the chance to alter it if that will not alter their goals. There is less resistance to dietary change as both parties can agree on an acceptable dietary modification.

The main components may include; the use of open- ended questions, asking permission, listening, reflecting, summarizing, assessing interest and confidence about dietary change as well as discussing the pros and cons of a dietary change (Cant and Aroni, 2008).

2.6.3 Motivational interviews (MI) and counselling

Motivational interview (MI) has been defined as "a client-centered, directive method for enhancing intrinsic motivation to change by exploring and resolving ambivalence" (Miller and Rollnick, 2002). This approach is a guided counselling style designed to achieve behaviour change through helping the patient to uncover his/her internal motivation for change (Brodie *et al.*, 2008). It is used to identify and resolve disagreements between desired behaviour and actual behaviour and to increase motivation for behaviour change (Miller and Rollnick, 2002; Martins and McNeil, 2009).

Motivational interviews therefore challenge traditional dietary intervention delivery methods with suggestions that patients know what they want, and must therefore be more involved in their own dietary intervention (Martins and McNeil, 2009). When adopted in dietary counselling, the work of the dietitians now becomes more as a facilitator for a change in dietary intake behaviour rather than a teacher or an adviser (Gable, 2007). The thoughts of the client is stimulated and supported through a decision making process which will eventually lead to behaviour change (Brodie *et al.*, 2008). It is considered to be a promising counseling model for health promotion and disease management (VanWormer and Boucher, 2004).

Dietitians who received basic training in motivational interviewing were reportedly more emphatic, often showed more reflection during consultation and were more likely than

control dietitians to make their patients talk for the majority of the consultation. Their patients consequently showed significantly lower saturated fat intake at post-test compared to patients of control dietitians. However, no effects on HbA_{1c}, BMI and waist circumference were found (Brug *et al.*, 2007).

Motivational interview is founded on four main principles; expression of empathy, development of discrepancy, rolling with resistance and the support of self-efficacy (Miller and Rollnick, 2002). Discrepancies usually develop between desired behaviour and actual behaviour. The patient may provide their own reasons for change and receive the support of the dietitian. However, if the patient is resistant to change, the dietitian will have to go along with them, instead of fighting or insisting on change, until such a time that the patient will be ready for change, where the dietitian continues to offer support (Martins and McNeil, 2009). Some studies have compared the effectiveness of MI and traditional dietary interventions in effecting a change in dietary behaviour and found it more effective.

In a systematic review to investigate the use of motivational interview's efficacy in medical health care, a statistically significant result in favour of MI was reported odds ratio = 1.55 (CI: 1.40 – 1.71), $z = 8.67$, $p < 0.001$. This suggests that patients who received MI were 1.55 times more likely to improve than those who did not. Among the areas where MI showed great promise were alcohol and tobacco use, body weight and sedentary behaviour (Lundahl *et al.*, 2013). Similarly, an earlier randomized control trial (RCT) comparing the effectiveness of a standard dietary intervention to a motivational dietary intervention among patients with hyperlipidemia reported that both methods resulted in statistically significant changes in dietary knowledge and behaviour which led to a reduction in body weight, but not serum cholesterol after 3 months of follow-up (Mhurchú *et al.*, 1998). In a review to

examine the effectiveness of MI for weight loss and exercise, MI showed significant improvement in diet, exercise behaviour and weight loss, (Van Dorsten, 2007).

2.7 Non pharmacological management of non-communicable diseases

By their chronic nature, non-communicable diseases require a lifetime of management which involve continuous care and treatment associated with high cost and often with limited improvement (Allam and Ortiz-Arjona, 2013). Drug resistance as well as harmful side effects in some cases may also occur. Most NCDs are idiopathic but are associated with unhealthy lifestyle, with emphasis on unhealthy diet, physical inactivity, smoking and wrongful use of alcohol (Lachat *et al.,* 2013). Few are also associated with degenerative processes associated with aging (Allam and Ortiz-Arjona, 2013). Lifestyle modification are therefore fundamental aspects of NCD prevention and management (Gillett *et al.*, 2012; American Diabetes Association, 2017). Effective lifestyle modification alone are able to control plasma glucose efficiently such that pharmacological management can be delayed (Gillett *et al.*, 2012). Some studies have shown that the best diabetes management outcomes are achieved when lifestyle management is combined with pharmacological treatment (Marín-Peñalver *et al.,* 2016). Prevention and reversal of conditions such as hypertension, dyslipidaemia and elevated blood sugars, as well as their risk factors such as obesity, have also been reported following lifestyle modification (Arena *et al.,* 2015). Two servings per day increment in whole grain consumption was associated with a 21% (95% CI, 13–28%) decrease in risk of type 2 diabetes after adjustment for potential confounders and BMI (Huang *et al.,* 2015). About 30% of hypertension patients were estimated to control their blood pressures through the lifestyle modification such that they did not require pharmacological treatment (Allam and Ortiz Arjona, 2013). Inverse associations were also reported be-tween intake of whole grains and incident hypertension (Huang *et al.,* 2015).

Adoption of the DASH diet significantly reversed systolic and diastolic blood pressure among hypertension patients (Steinberg *et al.*, 2017). Healthy behaviour involving frequent intake of whole grains, fruits, vegetable, low-fat dairy products, fish and nuts as well as reduction in the intake of saturated fat and refined sugars as described in the Mediterranean diet, resulted in significant weight reduction as well as glycaemic control 4 years after the intervention (Shai *et al.*, 2008). Favourable blood lipids were also reported with a low carbohydrate diet (Yu-Poth *et al.*, 1999). Lifestyle modification involving weight control, regular physical activity and smoking cessation have been suggested as protective against NCDs (WHO, 2018). Weight loss strategies using healthy diet and physical activity significantly reduced weight and the incidence of diabetes among pre-diabetes patients in a systematic review involving different populations (Norris *et al.*, 2005).

2.8 Recommended healthy eating dietary patterns

Healthy eating dietary patterns are those in agreement with the DASH or the Mediterranean eating plan (Sacks *et al.*, 2009; Sacks *et al.*, 2001). They are rich in vegetables, fruits, low-fat dairy products, whole grains, poultry, fish, beans, nuts, potassium, calcium, and magnesium, as well as dietary fibre and protein. They are low in sweets, sugar-sweetened beverages, red meats and saturated fats. Their sodium levels are also low ranging from 1,500 mg per day to 2,300 mg/day (U.S. Department of Health and Human Services and U.S. Department of Agriculture, 2015). Healthy dietary patterns are essential for all populations, both healthy and diseased, and is effective for both prevention and management of various diseases. The same principle of healthy eating is applied for the management of most diseases with marginal modifications where necessary.

Intake of more whole grains, fruits and vegetables, coupled with reduced intake of sweetened and refined sugars are recommended (NICE, 2015). Additionally, reduced total fat (cholesterol, saturated fat and trans fat) are advised (NICE, 2015; American Diabetes Association, 2012; International Diabetes Federation, 2012b). In Ghana, as part of healthy eating for adults, the recommended contribution of unrefined carbohydrate and starchy foods to total energy in a meal is 30%-50%, fruit and vegetables 10%-15%, protein if animal based 5%-10%, if plant based 20%-30% and total fat 10%-15% (Ministry of Health, 2010).

Table 2.8: Nutrient Composition of the therapeutic lifestyle changes diet

Nutrient	**Recommended Intake**
Saturated fat	Less than 7% of total energy
Polyunsaturated fat	Up to 10% of total energy
Monounsaturated fat	Up to 20% of total energy
Total fat	25-35% of total energy
Carbohydrate	50-60% of total energy
Fibre	20-30 g/day
Protein	Approximately 15% of total energy
Cholesterol	Less than 200 mg/day
Total energy	Balance energy intake and expenditure to maintain desirable body weight/prevent weight gain

Source: (National Cholesterol Education Programme, 2001; Ministry of Health, 2012)

A maximum salt intake of 2.4 mg/d, the equivalent of one teaspoon of salt in a day, is recommended (National Institutes of Health, 2004; National Cholesterol Education Programme, 2001). This may be achieved by avoiding the addition of salt at table, cooking with very little salt and reducing the intake of salty snacks. For individuals who have multiple risk of heart disease apart from being hypertensive, further lowering of sodium

intake to 1.5 g/d - 2.0 g/day is recommended (National Cholesterol Education Programme, 2001). Reduction of the intake of canned and processed foods and in some cases, adding salt only to sauces and soups but not starches and staples may help to achieve this. In a recent study to investigate the association of dietary factors with mortality from heart disease, stroke, and type 2 diabetes in the United States, it was concluded that dietary factors were indeed associated with substantial proportion of death from heart disease, stroke and diabetes. The largest numbers of estimated diet-related cardio-metabolic deaths were related to high sodium (66, 508 deaths in 2012; 9.5% of all cardio-metabolic deaths), low nuts/seeds (59 374; 8.5%), high processed meats (57 766; 8.2%), low seafood omega-3 fats (54 626; 7.8%), low vegetables (53 410; 7.6%), low fruits (52 547; 7.5%), and high sugar sweetened beverages (51 694; 7.4%) (Micha *et al.*, 2017). Frequent adequate intake of nuts and seeds, sea foods containing omega-3 fats, vegetables and fruits coupled with a reduction in the intake of sodium, processed meat and sugar sweetened beverages will therefore improve the health associated with food.

2.9 Commonly referred conditions by doctors to dietitians

Appropriate and timely nutrition management is well documented as necessary to improve patient health and reduce cost of health care (Tappenden *et al.*, 2013; Avelino-Silva and Jaluul, 2017). Conditions that are often referred to dietitians and the rate of referral, however, vary in different populations. The most common conditions referred to dietitians in a study carried out in Guateng South Africa, were hyperlipidaemia, diabetes mellitus and obesity (Barron, 2006). In another study, diabetes, heart disease and kidney disease were the most commonly referred conditions (Barnes *et al.*, 2018).

Some studies have suggested demographic factors of the referring doctor, mainly age and years of practise as the primary influence of their decision to refer patients. Older and longer serving general practitioners were less likely to refer patients to dietitians compared to younger ones (Barron, 2006). Other studies have found no association between gender, age and hours of work and the decision to refer (Pomeroy and Cant, 2010). Instead, poor clarity of the responsibilities of a community health dietician in chronic disease management, insurance-imposed constraints, perceived patient readiness to change, and service inaccessibility were the identified barriers to referrals to both community health and specialty care dietitians (Barnes *et al.,* 2018). General practitioners in one study also identified cost to the patient as the main barrier to referring to a dietitian, while the dietitians interviewed considered a lack of knowledge of where to refer, as the key issue (Nicholas *et al.,* 2003). Most of these studies applied qualitative methods of assessment, mainly by the use of focus groups and in-depth interviews. While some general practitioners formed the main study participants in some of the studies, general practitioners and dietitians formed that of others. A good working relationship between doctors and dietitians has been suggested as a possible remedy to improve patients' timely referral for nutrition management (Mitchell *et al.,* 2012).

2.10 The effectiveness of dietetic care provided by dietitians on patient health outcomes

The effectiveness of dietetic care provided by dietitians on patient health outcomes have been investigated in some studies (Agee *et al.,* 2017; Willaing *et al.,* 2004; Howatson *et al.,* 2015). Patients with conditions especially obesity, diabetes and cardiovascular diseases have mostly been investigated. Outcome indicators in most of these studies included glycated haemoglobin, waist circumference, body mass index, blood lipids mainly total

cholesterol as well as LDL cholesterol. The effect of dietetic care in most studies were estimated as changes in the outcome indicators from the beginning of the study to periods ranging from 6 months to 24 months of follow-up.

Among a group of type 2 diabetes patients randomly assigned to dietetic counselling by a dietitian in one group and general care in a second group, weight (Frondelius *et al.,* 2017), BMI (WHO, 2000) waist circumference (Misra *et al.,* 2009) and HbA1c (-0.3%, 0 = 0.04) significantly improved in the dietetic group after 24 months of follow-up, compared to the general care group (Battista *et al.,* 2012; Howatson *et al.,* 2015). In a cohort study involving adults with type 2 diabetes mellitus from a low-income background, patients who received medical nutrition therapy (MNT) by dietitians every 3 months for a period of one year were compared to those who received primary care alone. Significant improvement in systolic blood pressure, diastolic blood pressure and HbA1c were reported for the MNT group compared to the primary care alone group (Agee *et al.,* 2017). HbA1c reduction was -0.8% ($P < 0.01$), systolic blood pressure reduction was -8.2 mmHg ($P < 0.01$) and diastolic blood pressure reduction was -4.3 mmHg ($P < 0.05$). Systematic reviews of longitudinal data involving obese individuals who received dietary and lifestyle intervention over 1 to 3 years follow-up showed modest reduction in weight with corresponding reductions in lipid profiles. A 1kg decrease in weight resulted in a 1.3% decrease in serum total cholesterol and a 1.6% decrease in triglyceride. Weight loss that was sustained beyond 3 years, however, was not associated with any beneficial lipid changes. This suggested that other lifestyle changes apart from weight needed to be maintain (Aucott *et al.,* 2011).

In a systematic review to investigate the use of dietetic intervention using motivational interview's efficacy in medical health care, a statistically significant result in favour of the intervention group was reported, odds ratio = 1.55 (CI: 1.40 – 1.71), $z = 8.67$, $p < 0.001$.

This suggests that patients who received dietetic care based on motivational interviews were 1.55 times more likely to improve than those who did not. Improvement was seen as a positive change in areas such as alcohol and tobacco use, body weight and sedentary behaviour (Lundahl *et al.*, 2013). Similarly, a randomized control trial (RCT) comparing the effectiveness of a standard dietary intervention to a motivational dietary intervention among patients with hyperlipidaemia reported that both methods resulted in statistically significant changes in dietary knowledge and behaviour which led to a reduction in body weight, but not serum cholesterol after 3 months of follow-up (Mhurchú *et al.*, 1998).

The effectiveness of a class-based lifestyle intervention programme in a hospital, designed to mitigate the physical and fiscal consequences of chronic diseases, was tested on patients with various chronic conditions. It comprised an initial 48 hours of curriculum taught twice per week, in four-hour sessions over a 6-week period. The sessions involved physical activity, cooking and nutrition classes as well as stress management classes. The follow-up phase included three components; on-site classes at weeks 10, 18 and 30 that followed the same four-hour structure as earlier descried**,** a weekly e-mail newsletter and the buddy system where patients were paired to check on and encourage each other. Anthropometric, biomedical and blood pressure measurements were taken at baseline, 6 weeks and 30 weeks. After 30 weeks of follow-up, the proportion of patients with the metabolic syndrome dropped from 54% at baseline to 37% at 30 weeks. Means of all metabolic measures were significantly lower at 6 weeks compared with baseline (all $P < 0.025$). Mean blood glucose, total cholesterol, triglycerides, LDL, HgbA1c, insulin and US-CRP, were also significantly lower at 30 weeks compared with baseline, while mean HDL levels rose 3.7 ± 8.4 mg/dL (0.10 ± 0.22 mmol/L) ($P < 0.001$). In general, changes at 30 weeks were sustained, but sometimes less than at 6 weeks (Ricanati *et al*., 2011).

In a review to examine the effectiveness of dietary counselling using motivational interviews for weight loss and exercise, significant improvement in diet, exercise behaviour and weight loss occurred in the intervention group compared to the control group (Van Dorsten, 2007).

The similarities in the positive effect of dietetic care on these outcome indicators in the various studies is in spite of the differences in study designs as well as the follow-up periods and study participants, suggesting a true positive effect of dietetic care on disease outcomes.

CHAPTER THREE

3.0 METHODOLOGY

3.1 Study design

The design for the first phase of the study was cross-sectional. Cross sectional design often assesses a population, through a select sample at a single point in time (Thiese, 2014; Rubin and Babbie, 2009). Patients who were recruited were interviewed only once. Cross sectional studies are also useful in investigating the percentage of the population who may be exposed to a variable under investigation and the associated outcome of this exposure. It achieves this, either through observational, explanatory, descriptive or exploratory approach (Rubin and Babbie, 2009). In this study, an observational approach was followed. Cross sectional designs are also useful in the estimation of prevalence (Carlson and Morrison, 2009), as was the objective of this phase. The main aim of this phase was to identify the major diet and lifestyle-related chronic diseases which are referred to the Dietherapy Department of the Korle-Bu Teaching Hospital (KBTH).

The study design for the second phase of the study was a longitudinal follow-up study design. Patients were interviewed and measured at baseline. Interviews and measurements were repeated at three months and at six months follow-up. Three months was defined as 13 weeks from the date of recruitment while 6 months was defined as 26 weeks from the day of recruitment into the study.

3.2 Study site and area

The study site for the entire study was the Dietherapy Department of the Korle-Bu Teaching Hospital (KBTH) in Accra. The KBTH is the 3rd largest hospital in Africa and the leading national referral centre in Ghana. Patients who seek medical care in this hospital are

therefore referred from various health care facilities across the entire nation and may therefore be representative of the patient population nationwide. It is located in the Ablekuma South Constituency of the Greater Accra Region. The total reported outpatient attendance in 2013 was 365,387 (KBTH, 2013). The Dietherapy Department is responsible for the provision of optimal nutritional care for patients through quality therapeutic counselling. The reported total number of patients seen at the department's outpatient for 2011, 2012 and 2013 were 10,423, 12,021 and 12,005 respectively. In-patients seen during the same period totaled 32,964, 34,329 and 38,707 respectively (KBTH, 2013). Dietetic outpatient consultations occur on Mondays to Fridays, from 8.00 am to 4.00 pm daily (KBTH, 2016b). Treatment approach at the OPD is purely through counselling with the use of various educational tools (e.g. diet sheets) and materials such as food models and handy measures. Newly referred patients to the department are as a matter of principle attended to by registered dietitians only.

3.3 Study population

Eligible participants for the study were patients who had been referred by a medical doctor for the first time, to see a dietitian to assist in the management of their condition.

3.3.1 Inclusion criteria

All patients 18 years and above, who were referred to the study site for the first time were eligible. Only those who presented a referral letter signed by the referring medical doctor were considered. The referral letter needed to clearly state the condition for which they had been referred to seek dietetic care with supporting clinical measurements or laboratory test results associated with the condition e.g. blood pressure and fasting plasma glucose. Patients who met these criteria and consented to participate in the study were recruited.

3.3.2 Exclusion criteria

Patients who were less than 18 years old (such as children under 5 years old who were referred for malnutrition related issues) and pregnant women were excluded. Patients who were too ill to participate or who refused consent to participate in the study, as well as regular patients to the department who reported for review were also excluded.

3.4 Sample size determination

The OpenEpi software for calculation of sample size was used. For the cross-sectional study, estimation of the frequency in a population was used. The following information were imputed:

- Population of Accra *(GSS, 2012)* = 4,010,054
- Prevalence of hypertension in central Ghana = 28.7%

  (Cappuccio *et al.*, 2004)
  - A confidence interval = 95%
  - A statistical power = 80%

Prevalence of hypertension in central Ghana was used in estimating sample size because it is the condition with the highest reported prevalence among Ghanaians. The calculated sample size was 315. This was adjusted upwards to 331 to cater for a 5% non-response rate. A total of 331 patients were therefore needed to participate in the study. However, 339 eligible patients were recruited and participated in the study.

For the follow-up study, sample size calculation for cohort studies was used. Using similar parameters as above, a total of 80 patients were needed for the study. A 25% drop out rate and a 15% non-response rate were considered. Hence, a minimum of 116 patients were needed for participation in the study. A total of 132 patients were, however, recruited.

3.5 Sampling technique

For phase 1, all consecutive patients who met the eligibility criteria, who consented to participate were recruited. Daily visits were paid to the study site. With the assistance of the records officer at the department's OPD, eligible patients were identified. The details of the study were explained to them and their written consent obtained before they were recruited into the study. They were then interviewed and measured. Recruitment continued until the total number of patients needed was obtained.

For phase 2, a systematic sampling technique was used to sub-sample patients from the cross-sectional study to participate in the follow-up study. To obtain the ideal interval for the systematic sampling, (the K^{th} number), the total calculated sample size of the cross-sectional study (331) was divided by that of the follow-up study (116) to obtain 2.8. An interval of 2 was decided on, taking into consideration the fact that some patients who consented to participate in the cross-sectional study may refused consent for the follow-up study. Hence, every other patient recruited for the cross-sectional study was invited to participate in the follow-up study. A total of 132 patients were therefore interviewed and measured at baseline for the follow-up study. By the 3^{rd} and 6^{th} months, however, their numbers had reduced to 80 and 60 respectively, with the others lost to attrition.

3.6 Data collection tools

3.6.1 Questionnaires used

In total, 3 questionnaires were used for the entire study. These were the cross-sectional questionnaire, follow-up baseline questionnaire and the follow-up questionnaire. They were all interviewer administered.

3.6.1.1 The cross-sectional questionnaire

The core WHO STEPSwise approach for non-communicable disease risk factor surveillance instrument, with minimal modifications was used for data collection. The instrument had 52 main questions. Some of these questions had follow-up questions. The questionnaire had 3 main sections as follows:

1. Demographic information and socioeconomic information (e.g. age, gender, ethnicity, marital status highest educational attainment, employment status and current occupation).
2. Clinical information (e.g. diagnosis for which referred, other comorbidities, date of referral to the Dietherapy Department, actual date of accessing dietetic care and reasons for delays if any).
3. Lifestyle (e.g. smoking habits, alcohol intake and physical activity habits).

The questionnaire combined both pre-coded questions as well as open ended questions. They were all interviewer administered.

3.6.1.2 The follow-up baseline questionnaire

The baseline questionnaire was similar to the cross-sectional questionnaire. It adopted the core WHO STEPS approach for non-communicable disease risk factor surveillance instrument (WHO, 2008d), with some modifications. In addition, was a food frequency questionnaire, a 24-hour dietary recall interview guide and a questionnaire to obtain information on the interventions given to patients.

A standard 24-hour dietary recall interview guide was attached to determine the macro nutrient, energy and dietary fibre intake of patients. It obtained information on all the foods which patients had eaten in the immediate past 24-hours, and estimated the amounts

consumed. The time at which each food was consumed was also noted. It had 4 columns. The first column required information on the time of day at which food had been eaten. This was followed by what type of food the patient had eaten in the next column. Next was the amount of food eaten. Patients estimated this with the assistance of food models and handy measures, and the final column was the metric weight (in grams) of the food eaten. It was administered before patients saw the dietitian. It could not be repeated on a second day because patients received dietary counselling from registered dietitians that same day they were recruited, interviewed and measured. Their subsequent dietary intake may therefore have been influenced by the counselling intervention they had received and was no longer a true reflection of their previous dietary intake habits.

A validated food frequency questionnaire (FFQ) adopted from Steele-Dadzie *et al.*, (2009) was attached. It was used to determine patient's usual dietary intake. A food frequency questionnaire is a dietary intake assessment tool useful for providing information on the usual dietary intake of individuals. It has the advantage of providing long-term (weekly, monthly or yearly) dietary intake habits. Weekly estimation was chosen over monthly or yearly estimations in this study because, estimations over longer periods are often associated with higher bias, as people are less likely to remember and estimate correctly over such long periods. It may be argued that such shorter estimations may fail to capture foods that may be consumed rarely, such as monthly or bi-monthly etc. However, such rarely consumed foods may not impart significantly on the individuals' health, just by virtue of it being so rarely consumed. Dietary intake habits that markedly impart on the health of an individual are mainly the routine daily or weekly intakes which constitute their habitual intake. These habitual intakes often do not change and get repeated week after week. Occasional, ceremonial diets may not make much impact on the health of an individual. A knowledge

of the routine usual dietary intake of study participants was necessary in this study due to the association of diet with the development of NCDs. Unhealthy dietary habits over a period has been associated with risk of the development of conditions such as obesity, diabetes mellitus and hypertension (WHO, 2008c).

The questionnaire consisted of 88 listed food items categorized and discussed under 9 main groups based on similarities in nutrient and dietary fibre profiles. The 9 groups were informed by the Department's diet sheets which are given to patients to assist them to keep to their diet regimens. The diet sheets are designed based on international guidelines for the management of various diet-related chronic diseases such as the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III), IDF 2012 and NICE, 2011. The dietary components defining each of these 9 groups were either promoters of good health, which were therefore recommended for frequent intake or a health risk when consumed too frequently, and were therefore to be consumed less frequently in small quantities or completely avoided. The groups were; foods made with whole grains, other starches, fatty foods, salted foods, lean foods, fruits, vegetables, sweetened beverages and alcoholic beverages.

Dietetic advice (intervention) given to patients for the management of the conditions for which they were referred were also documented. Patients received 30 to 45 minutes of one-on-one counselling sessions with qualified dietitians on their first visit to the department. Interventions were individualized following a nutrition assessment of the patient and a nutrition diagnosis as described in the nutrition care process (NCP) of the Academy of Nutrition and Dietetics (https://www.ncpro.org//default.cfm). The use of NCP at the time of

data collection was at its infantile stage in most hospitals in Ghana, as it had just been introduced. As a result, there was lack of uniformity of the information which dietitians provided in patients folders at the time. The study therefore limited information on interventions given to patients to the type of dietary regimen (intervention) that the patient had been put on, and the recommended total daily energy intake. This was because, those were the information which patients had in common in their folders. Patients were put on different dietary regimens depending on the condition of referral, its severity and comorbidities. Most patients were therefore put on more than one dietary regimen.

These dietary interventions are all evidence based and are backed by various international guidelines. Most of these diet regimens were marked by restrictions of the intakes of various dietary components. The type of diet regimen invariably implied the avoidance or reduced intake of certain foods, or the increased intake of others. These were explained in detail on their diet sheets. Each patient was therefore issued with the diet sheet commensurate with the recommended diet regimen, to be taken home and used as reference material towards enhancing compliance to the diet regimen. Table 3.I below shows the diet regimens which were recommended for patients and the dietary interpretation. Appendix XIII also shows a sample of diet sheets given to patients with hypertension at the department.

Table 3.1: Diet interventions recommended for patients

Type of diet regimen	Dietary interpretation
High fibre diet	> 25 g of fibre daily (At least 5 servings of fruits and vegetables daily. Reduction of refined carbohydrate intake and inclusion of more whole grains in routine diet).
Weight reducing diet	Energy restriction such that energy expenditure exceeds energy intake. Patients were advised to reduce intake of high calorie foods, include low-calorie unrefined carbohydrates, fruits and vegetables in their diet and also to be more physically active.
Low sodium diet	Patients were required to limit their intake of salt to 1500 - 2300/day i.e. a maximum of 1 tea spoon per day, or less. They were to avoid addition of salt at table, cook with less salt, and significantly reduce their intake of salted processed foods.
Low total fat, cholesterol and saturated fat diet	Patients were required to reduce their intake of fatty meat, fried foods, organ meat, high fat dairy products and some pastries. They were to cook with less oil and to choose less fat dairy products such as skimmed milk. Total fat was supposed to contribute to 25% - 35% of their total daily energy intake. Their diet was to provide the following; Cholesterol = < 200mg / day, PUFA = up to 10% of total calories, MUFA = up to 20% of total calories Saturated fat < 7% of total daily calories.

Subsequently, patients were to report for reviews. The number of reviews differed for each patient and was dependent on the severity of their condition, the nutritional targets that had been set and the patient's readiness for change. Reviews were mainly used to monitor the progress of patients, address particular challenges they were facing in their effort to meet the prescribed dietary regimen and re-emphasize or clarify areas where patients may have forgotten or misunderstood.

3.6.1.3 Follow-up questionnaire

This was similar to the follow-up baseline questionnaire. It adopted the core WHO STEPSwise approach for non-communicable disease risk factor surveillance instrument. It further included a 24-hour dietary recall interview guide and a food frequency questionnaire. The 24-hour dietary recall here, however, was administered on two separate days; 1 weekday and 1 weekend day. The first was done when patients reported at the Department on their 3 or 6 month scheduled date. The second was done by phone interview within the same week of follow-up. Their mean macronutrient, energy and dietary fibre intakes for the two days were determined and used in analysis. The food frequency questionnaire was the same as for follow-up baseline questionnaire. The same follow-up questionnaire was administered at 3 and 6 months.

3.7. Training of field workers for data collection

Field workers comprising 2 research assistants and 2 laboratory technicians were taken through a one-week training. The training was as follows:

a. A general overview of the study including explaining the study objectives.
b. Systematically going through each instrument step by step to acquaint themselves with the nature of the data to be captured.
c. Field workers were taught how to take anthropometric measurements (height, weight, waist circumference and hip circumference) using the NHANES III anthropometric procedure videos. Field workers took turns to practice on each other to sharpen their skills.
d. Field workers were also trained to use the Omron digital blood pressure monitor to measure blood pressure as well as the flat Tanita body composition monitor for measuring body composition.

e. Field workers engaged in role plays as patients and interviewers, to expose themselves to potential challenges they may encounter. Challenges that ensued were all addressed by the researcher to ensure accuracy of data that were collected.

3.7.1 Equipment used

Standing height of respondents was measured using a Seca stadiometer (Model 213, Germany) to the nearest 0.1 cm. Weight, percentage body fat, visceral fat, muscle mass and total body water were also measured using the Tanita BC-533 InnerScan Body Composition Monitor. Waist and hip circumference were measured using an inelastic measuring tape to the nearest 0.1cm. Blood pressure in mmHg was measured on the right arm using the Omron Digital Blood Pressure Monitor, Model No: Hem-907XL by Omron Health Care Inc. Fasting plasma glucose, 2-hour plasma glucose, serum total cholesterol, triglycerides and high density lipoprotein were all measured using the Bio-Systems Chemical Analyzer with Biosystem reagents. C-reactive protein was determined using the RX Series CP 3847 and the RX Daytona analyzer by Crumlin, United Kingdom.

3.7.2 Quality control

Quality control measures undertaken included the following:

1. All equipment were checked before they were used daily.
2. Batteries for operating these equipment were replaced every week to ensure efficiency.
3. Weight measuring equipment were calibrated routinely using objects of known weight.
4. At the end of each day, all filled questionnaire were double-checked for completeness.

5. Patients were called on phone to obtain any missing information to complete any questionnaires that was found to be incomplete.

3.8. Data collection techniques

All data were collected by the researcher, together with the trained research assistants and laboratory technicians through face to face interviews. For patients recruited into the cross-sectional study, administration of the cross-sectional questionnaire by the interviewers ended the process. Patients were never contacted again.

For the follow-up study, however, following the administration of consent at baseline, patients fasting blood samples were taken. Two EDTA tubes and 1 serum separator tube were pre-labeled for each patient with their respective identity numbers. Fasting blood samples were taken between 7am and 8am on each day. No fasting samples were taken after 8.00am to prevent over-fasting. Patients had fasted between 8 to 12 hours before blood samples were taken. For each patient, 10 ml of venous blood was taken using 10 ml syringes. Two milliliters was emptied into 1 of the EDTA tubes for fasting plasma preparation while the remaining was emptied into the serum separator tube for serum samples.

Patients were then given a glucose drink (75g anhydrous glucose dissolved in 300 ml purified water), and the time at which drinking was commenced was noted (McMillin, 1990). Anthropometry, body composition and blood pressure were then measured. Patients then sat while the follow-up baseline and food frequency questionnaire were administered and 24-hour interviews conducted during the 2-hour wait period. Exactly 2 hours from the time glucose drink was taken, 2ml of blood was again drawn into the second EDTA tube to perform their 2-hour glucose test. This marked the end of the process and patients went on

to see the dietitian for counselling. All blood samples were kept on ice packs in an ice chest until the last sample was drawn. Samples were then immediately transported to the laboratory for preparation. For patients who had already had breakfast, the various questionnaires were administered and their measurements were taken. They were then scheduled to report early the next morning for fasting blood samples to be taken. They were counselled what time to eat their last meal in the evening and what time to report in the morning.

The 24-hour recall interview for the second day (weekday or weekend day) was obtained through telephone interview within that very week. The entire procedure described above, with the exception of administration of consent, was repeated at 3 and 6 months follow-up. Patients were called on phone on the 11^{th} and 12^{th} weeks as well as the 21^{st} and 22^{nd} weeks to remind them of their follow-up meetings for the 13^{th} week (3 months) and 26^{th} week (6 months) respectively. Fig 3.1 below shows the flow diagram describing the sequence of activities which were involved in the data collection.

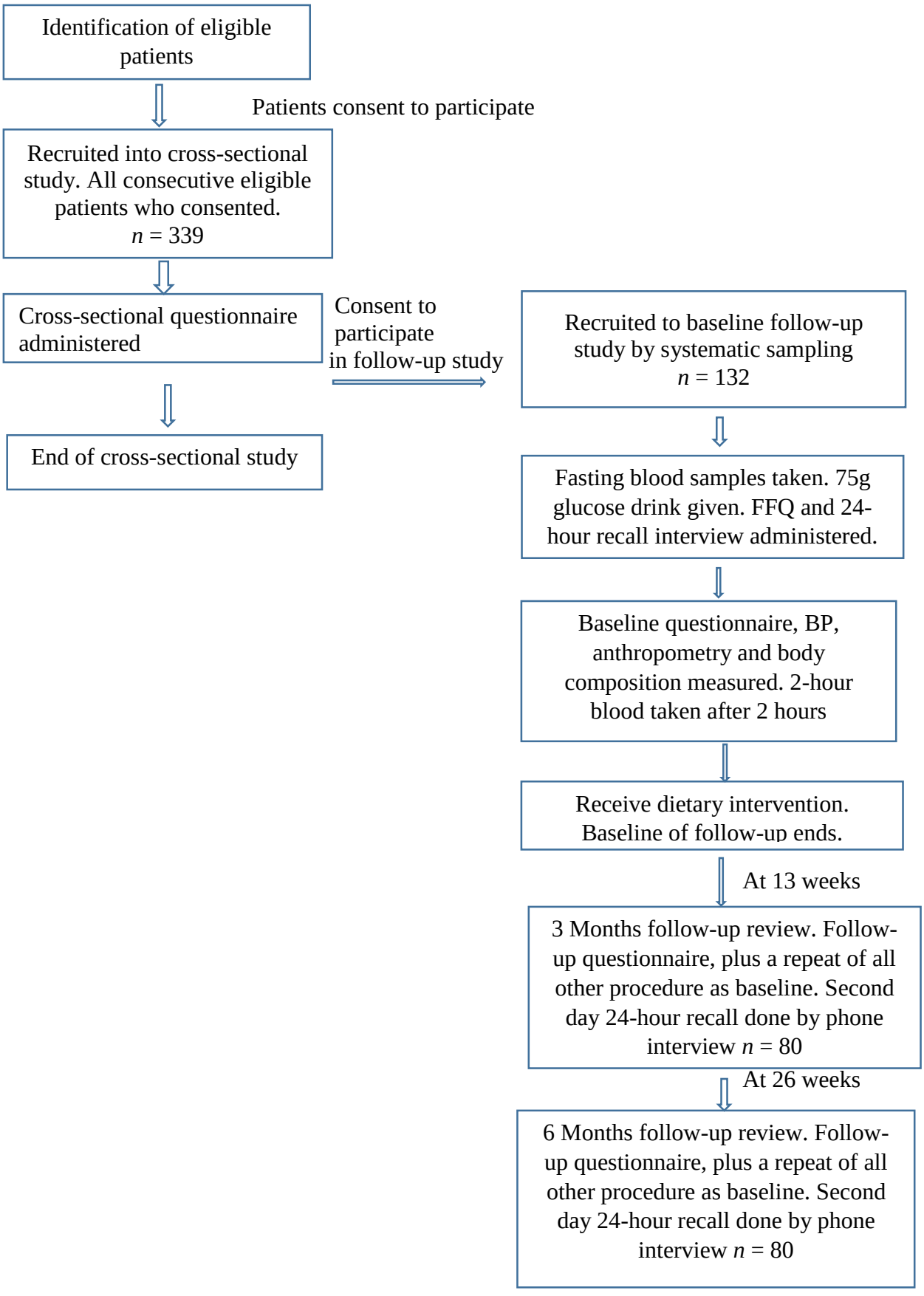


Fig 3.1. Flow diagram outlining the sequence of procedures involved in data collection

3.8.1 Data collection procedure; cross-sectional study

The cross-sectional questionnaire was administered. All information was obtained once, on their first day of recruitment into the study. Patients were not contacted again after that.

3.8.2 Data collection procedure; follow-up study

The respective questionnaire, whether follow-up baseline, or follow-up questionnaire as well as food frequency questionnaire were administered at follow-up. Twenty-four hour recall interviews were also done. Nutritional status was described based on BMI and WHR. Height was measured with a Seca stadiometer to the nearest 0.1cm. Patients stood flat on the level platform of the stadiometer with barefoot, with their knees straightened. The lower eye socket to the ear called the Frankfurt Plane, was ensured to be perpendicular to the vertical board of the stadiometer. The sliding piece of the stadiometer was moved gently to the level of the crown of their head, before readings were taken.

Weight and body composition were measured using the Tanita BC-533 InnerScan Body Composition Monitor. The equipment was turned on and patient's height and gender imputed. Patients stood barefooted with straightened knees and back on the platform of the monitor looking straight ahead. It was ensured that each foot was placed on the foot and heel electrodes. Percentage body fat, muscle mass, visceral fat and total body water were recorded. BMI was calculated as wt (kg)/ ht(m^2). It was classified with the following cut-offs based on WHO, 2008.

i. < 18.5 = underweight
ii. 18.5 - 24.9 = Normal weight
iii. 25.0 – 29.9 = Overweight
iv. ≥ 30 = Obese

Body composition reference values were based on manufacturer's guide in APPENDIX XI.

Waist and hip circumference were measured in duplicate with a non-elastic plastic measuring tape to the nearest 0.1cm to evaluate the relative distribution of fat at different locations of the body. Waist circumference (WC) was measured by encircling the body horizontally at the visible waist or at the narrowest part of the torso when viewed from the front. Hip circumference (HC) was measured in a similar way, but at the level of maximal protrusion in the hip region, including the buttocks. Waist hip ratio (WHR) was calculated as WC/HC. (WHO, 2008c) was used as reference data and illustrated in Table 3.3 below. Waist circumference was interpreted following IDF definition shown in Table 3.2 below.

Table 3.2: IDF definition of waist circumference

Gender	Waist circumference	Definition
Males	< 94 cm ≥ 94 cm	Healthy Central obesity
Females	≤ 80 cm > 80 cm	Healthy Central obesity

Source: (IDF, 2006)

Table 3.3: WHO definition of waist to hip ratio

Gender	WHR	Definition
Males	≥ 0.90	Central obesity
Females	≥ 0.85	Central obesity

Source: (WHO, 2008b)

Patients were allowed to rest for 10 minutes before blood pressure was measured in the right arm while seated. Systolic blood pressure and diastolic blood pressure were measured using the Digital Blood Pressure Monitor, Model No: Hem-907XL by Omron Health Care Inc. Patients were allowed to sit comfortably and their arm was rested on a table at the level of their arms. Patients were asked to loosen or remove completely any clothing that covered the upper arm where measurements were taken. The cuff was connected to the equipment

and wrapped around the upper arm with the lower part of the cuff about an inch above the elbows, the antecubital fossa. The tube of the cuff was also ensured to align to the brachial artery. The equipment was then started and measurement recorded in duplicate. The mean systolic and diastolic measurement were then recorded. Hypertension was defined as systolic and diastolic blood pressure ≥130/ 80 mmHg (ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA, 2018),

To assess physical activity, patients were asked questions on 2 activity domains: vigorous intensity activity (e.g. football and sprint activities) or moderate intensity actives (e.g. brisk walking). They were asked to indicate the number of times they perform these activities in a typical week and the number of minutes each activity lasted. Activities needed to last at least 10 minutes per session to be regarded. Patients who engaged in some physical activity for at least 30 minutes daily, 5 or more days a week, were classified as physically active.
Smoking status was determined by identifying patients who had never smoked, those who were current smokers and those who had smoked in the past but had stopped. Questions such as “Are you currently smoking (cigarettes, pipe or cigar)?” if yes how often do you smoke in a day, week or month” were asked. Individuals who smoked at least 1 stick a week were categorized as current smokers.

Alcohol intake was assessed by asking patients if they had taken any alcohol within the past 12 months and the immediate past 7 days. The frequency of intake in the past 12 months as well as the number of drinks taken at a sitting, on a typical day were also asked. One drink was defined as 120 ml of wine (½ medium glass of dry wine) or 285 ml of beer (half of large beer bottle, one full mini) or One bottle of Guinness or 30ml (one tot) of spirit, whisky, gin, akpeteshie, “woba ada anaa”, other alcoholic bitters or 60ml of brandy, vermouth, aperitif

etc. Men who consumed more than two drinks in a day and women who consumed more than one drink in a day were classified as heavy drinkers.

3.8.2.1 Preparation of plasma samples

On arriving at the laboratory with the samples on ice, fasting blood, 2-hour blood (both in separate EDTA tubes) and the blood in an 8 ml serum separator tube, were centrifuged at 3000 rpm for 10 minutes. Two aliquots each for fasting and 2-hour plasma samples per patient, and eight aliquots of serum for each patient, were stored in pre labelled Eppendorf tubes at -80^{o}C.

3.8.2.2 Determination of serum HDL concentration by direct method

Serum HDL concentration was determined using the enzymatic colorimetric method. The method involved two stages; the precipitation and the colorimetry stages. The Biosystems chemical analyzer with ready to use Biosystems reagent was used.

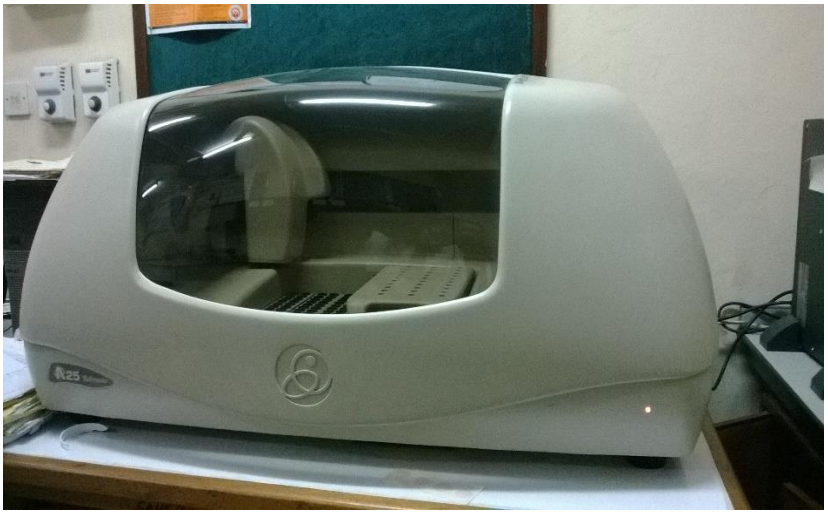


Fig 3: Biosystems chemical analyzer (Biosystems Model No: SN 8310141030 08030, Barcelona, Spain.

At the precipitation stage, Serum stored at -80^{o}C was thawed to room temperature. Serum sample (0.2 ml) was then pipetted into a tube and 0.5 ml of reagent containing phosphotungstate magnesium chloride added. They were thoroughly mixed and allowed to stand for 10 mins at room temperature. Very low density lipoprotein, low density lipoprotein

cholesterol and chylomicrons precipitated in the presence of phosphotungstate and magnesium ions. The mixture was then centrifuged at 4000 rpm for 10 mins. The HDL containing supernatant was pipetted into another pre-labelled tube.

At the colorimetry stage 50 µl of supernatant at room temperature was pipetted into a tube. Similarly, 50 µl of distilled water and HDL cholesterol standard were each pipetted into a separate tube. Reagent B (.0 ml), at room temperature, was added to each of the three tubes. Each tube was mixed thoroughly and incubated for 30 mins at room temperature. The absorbance of the sample as well as the standard were measured at 500 nm against the blank. The HDL cholesterol concentration in the sample was determined as;

$$\frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times \text{Concentration of standard} \times \text{sample dilution factor} = \text{HDL conc. of sample}$$

The colorimetry stage was carried out entirely using the chemical analyzer. HDL concentration of samples in mmol/L were therefore printed out from the analyzer. All test were done in duplicate and the mean value determined and recorded.

$$\text{Cholesterol esters} + H_2O \xrightarrow{\text{Cholesterol esterase}} \text{Cholesterol} + \text{Fatty acids}$$

$$\text{Cholesterol} + \tfrac{1}{2} O_2 + H_2O \xrightarrow{\text{Cholesterol oxidase}} \text{Cholestenone} + H_2O_2$$

$$2\ H_2O_2 + \text{4-Aminoantipyrine} + \text{DCFS} \xrightarrow{\text{Peroxidase}} \text{Quinoneimine} + 4\ H_2O$$

Reference values for HDL were that of the National Cholesterol Education Programme, 2001, and are shown in Table 3.4 below.

Table 3.4a: reference values for HDL based on NCEP, 2001

HDL Cholesterol	**Healthy Range**
Males	>1.0 mmol/L
Female	>1.3mmol/L

Source: (National Cholesterol Education Programme, 2001)

3.8.3 Determination of serum total cholesterol

Serum total cholesterol concentration was determined using the enzymatic colorimetric method. Cholesterol esters in the sample are hydrolyzed by cholesterol esterase. The released free cholesterol is further oxidized by cholesterol oxidase to form cholestenone and peroxide. A measurable red quinoneimine derivative, that has an absorbance at 500 nm, is formed from hydrogen peroxide (H_2O_2) and 4-amino-antipyrine in the presence of phenol and peroxidase

$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{Cholesterol esterase}} \text{Cholesterol} + \text{Fatty acids}$$

$$\text{Cholesterol} + 1/2O_2 + H_2O \xrightarrow{\text{Cholesterol oxidase}} \text{Cholestenone} + H_2O_2$$

$$2\,H_2O_2 + \text{4-Aminoantipyrine} + \text{Phenol} \xrightarrow{\text{Peroxidase}} \text{Quinoneimine} + 4\,H_2O$$

The Biosystems chemical analyzer with ready to use Biosystems reagent was used. Serum stored in a -80^{o}C freezer was removed and thawed to room temperature. Reagent standard, sample and distilled water (10 µL), was each pipetted into separated pre-labelled tubes. The ready to use reagent (1.0 mL) was added to each tube. The mixtures were mixed thoroughly and incubated for 10 mins at room temperature. The absorbance of sample and standard were measured against the blank at 500 nm by spectrophotometry. The triglyceride concentration was computed as follows:

$$\frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times \text{concentration of standard} = \text{Conc. of total cholesterol in the sample}$$

All measurements were done in duplicate and the mean concentration determined. The results were printed from the Biosystems analyzer. The reference values below were used:

Table 3.4b: Reference values for serum total cholesterol based on NCEP, 2001.

Serum total cholesterol range	Classification
< 5.2 mmol/L	Desirable

Source; (National Cholesterol Education Programme, 2001)

3.8.4 Determination of serum triglyceride

Serum triglyceride concentration was determined using the enzymatic colorimetric method. Triglyceride undergoes enzymatic hydrolysis to produce glycerol. The resulting glycerol is then enzymatically determined. It is oxidized and peroxide, one of the by-products of the oxidation reaction is measured quantitatively in a peroxidase catalyzed reaction that produces a colour. The colour intensity is proportional to the triglyceride concentration.

$$\text{Triglycerides} + H2O \xrightarrow{\text{Lipase}} \text{Glycerol} + \text{fatty acids}$$

$$\text{Glycerol} + ATP \xrightarrow{\text{Glycerokinase}} \text{Glycerol-3-phosphate} + ADP$$

$$\text{Glycerol-3-phosphate} + O2 \xrightarrow{\text{Glycerol-3-phosphate oxidase}} \text{Dihydroxyacetone phosphate} + H_2O_2$$

$$2\ H_2O_2 + \text{4-aminoantipyrine} + \text{4-chlorophenol} \xrightarrow{\text{Peroxidase}} \text{Quinoneimine} + 4\ H_2O.$$

The Biosystems chemical analyzer with ready to use Biosystems reagent was used. Serum stored in a -80^{o}C freezer was removed and thawed to room temperature. An amount (10 µL) of reagent standard and sample were each pipetted into separated pre-labelled tubes. The ready to use reagent (1.0 mL) was added to each tube with an additional 1.0 mL into a blank tube. The mixtures were mixed thoroughly and incubated for 15 mins at room temperature. The absorbance of sample and standard were measured against the blank at 500 nm by spectrophotometry. The triglyceride concentration was computed as follows:

$$\frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times \text{concentration of standard} = \text{Concentration of triglyceride.}$$

All measurements were done in duplicate and the mean concentration determined. The results were printed from the Biosystems analyzer. The reference values used are shown in Table 3.4c below.

Table 3.4c: Reference values of serum triglycerides based on NECP, 2001

Serum triglyceride range	Classification
< 1.7 mmol/L	Desirable

Source: (National Cholesterol Education Programme, 2001).

3.8.5. Estimation of fasting serum LDL (Low density lipoprotein cholesterol)

In this study, fasting serum LDL concentration was calculated using the Friedewald Formula (Friedewald *et al.*, 1972) as follows;

LDL = Total cholesterol – (Total triglyceride ÷ 2.2) - HDL cholesterol.

The reference below (Table 3.4d) was used to determine healthy levels and vice versa.

Table 3.4d: Reference values for serum LDL based on NCEP, 2001

Serum LDL range	Classification
< 3.4 mmol/L	Desirable

Source: (National Cholesterol Education Programme, 2001)

3.8.6 Determination of serum C-reactive protein

Serum C-reactive protein was determined using Randox full range CRP Immunoturbidimetric assay, RX Series CP 3847 and the RX Daytona analyzer by Crumlin, United Kingdom.

Fig 10: RX Daytona analyzer by Crumlin, United Kingdom.

The assay comprised an assay buffer (2 ×11ml) and an antibody-latex reagent (2 × 11ml). All components were supplied ready-to-use. The assay operates on the principle that, serum samples will react with the buffer and the anti-CRP coated latex to form an antibody-antigen complex. This complex results in an increase in the turbidity which can be measured as the amount of light absorbed at 570 nm. A standard curve of the absorbance versus the CRP concentration of the standards is then used to determine the concentration of the samples. Normal CRP values ranged from 0 to 5 mg/L.

The assay was placed in the equipment following manufacturer's instruction. Serum samples in Eppendorf tubes stored at -80^{o}C were thawed to room temperature. They were then mounted in their holders in the analyzer which had already been turned on, and the analysis were run. C-reactive protein results were displaying on the computer of the analyzer after 15 to 20 minutes. This was entered into Microsoft excel spreadsheets and analyzed.

3.8.7. Determination of fasting and 2-hour plasma glucose using glucose oxidase test

Fasting and 2-hour glucose were determined using glucose oxidase enzyme test. Glucose oxidase is specific to glucose and not any other saccharide, thereby making it an accurate and preferred method for determination of plasma glucose concentration. Glucose oxidase catalyzes the oxidation of glucose present in the plasma. The hydrogen peroxide liberated in the reaction is then reacted with peroxidase enzyme and a suitable chromogenic substrate. The development of substrate colour thus is proportional to the amount of H_2O_2 released which is in turn related to the amount of glucose originally present, and is measured using colorimetry. Biosystems reagent and analyzer was used.

$$\text{Glucose} + \tfrac{1}{2}\,O_2 + H_2O \xrightarrow{\text{Glucose oxidase}} \text{Gluconate} + H_2O_2$$

$$2\,H_2O_2 + \text{4-Aminoantipyrine} + \text{phenol} \xrightarrow{\text{Peroxidase}} \text{Quinoneimine} + 4\,H_2O$$

Fasting samples were removed from the freezer and thawed to room temperature. 10 µL of sample was then pipetted into each of 2 pre-labelled tube (test was duplicated). Similar volume of glucose standard and distilled water (blank) were also pipetted into separate tubes. 1.0 mL of reagent was added to each of the tubes, mixed and incubated for 10 mins at room temperature. The absorbance of the sample and standard were measured against that of the blank at 500 nm. The glucose concentration in the sample was then calculated as follows;

$$\frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times \text{concentration of standard} = \text{Glucose concentration.}$$

The results were all generated by the Biosystems chemical analyzer and printed out. Both fasting and 2-hour glucose test were done following similar procedure. They were done in

duplicate and their means computed and used in analysis. The reference values shown in Table 3.5 were used.

Table 3.5: Reference values for fasting and 2-hour plasma glucose based on WHO, 2006

Plasma glucose	**Normal glucose tolerance**	**Impaired glucose tolerance**	**Diabetes mellitus**
Fasting plasma glucose (National Cholesterol Education Programme)	< 5.5mmol/L	5.5 – 6.9 mmol/L	≥ 7.0 mmol/L
Two-hour after glucose load (National Cholesterol Education Programme)	< 7.8 mmol/L	≥ 7.8 – 11.0 mmol/L	≥ 11.1 mmol/L

(World Health Organization, 2006)

3.8.8 Assessment of patient's macronutrient energy and dietary fibre intake

The actual amounts of nutrients consumed by patients was determined by the use of a 2-day 24-hour dietary recall interview. Patients were required to recall and estimate, using handy measures (e.g. cups, kitchen spoons and plates) all foods consumed within the last 24 hours immediately preceding the day of interview. The weights of all foods consumed were then determined using the Food Weights/ Handy Measures Tables of the Dietetic Group of The Dreyfus Health Foundation, Ghana. ESHA Food Processor (version 6.02) as well as nutrient composition tables of the Food Research Institute of Ghana (Eyeson and Ankrah, 1975) were used to convert food intake into nutrient intake. Ministry of Health, 2010, U.S. Department of Health and Human Services and U.S. Department of Agriculture, 2015, were used as reference data.

3.8.9. Assessment of patient's usual dietary intake

Patients' usual dietary intake was assessed using the food frequency questionnaire, earlier described under data collection tools. For each of the 88 food items listed under 9 groups,

patients were asked to indicate the number of times they consumed it in a typical week. Frequencies were categorized as never (for foods that were never consumed), low intake (for foods that were consumed only once in a week) and moderate to high intake (for foods consumed 2 or more times in a week). Usual food intake was assessed at baseline and repeated at 3 and 6 months follow-up. The proportion of patients who consumed each food item at the various frequencies were compared between the three periods to determine any changes in usual dietary intake, which could be attributed to the interventions they received.

3.8.10. Documentation of Intervention

Intervention given to patients were documented at baseline of the follow-up study. Dietary interventions at the Dietherapy Department of the KBTH took the form of one-on-one counselling sessions between patients and dietitian. In situations where a patient was brought by a care giver, the care giver would also be present. The interventions were decided based on the nutrition assessment (BMI and diet history). It took the form of modifications to patients' diet in agreement to the recommended guidelines for the management of the condition which patient presented, as well as their clinical evidence (e.g. results of blood pressure, FPG, lipid profile etc.). Any of the 5 registered dietitians who worked at the Dietherapy Department at the time provided dietary intervention following the department's standard operating protocols. Interventions were individualized and the goals agreed between patient and dietitian. Two key information was obtained from patients folders to represent the intervention they received. These were; the type of dietary regimen recommended for patients (earlier described under data collection tools) and the recommended total daily energy intake.

3.9 Data Management

All questionnaires were kept under lock at the Dietherapy Department, KBTH, and was accessible to the research team only during the period of collecting data. Data were entered on a password protected laptop and accessible to only the research team. Patient confidentiality was assured in all reports from the study.

3.10 Data Analysis

Data from phase one and two were entered manually into Microsoft Excel. Data was cleaned using the data filter function of Microsoft Excel. Each variable was filtered to check for errors and inconsistencies resulting from data entry. For any identified error, the related questionnaire was pulled out to check and correct all such anomalies. The cleaned data were then exported to Statistical Package for Social Sciences (SPSS vs 20.0) for analysis. Data from the cross-sectional study were analyzed and reported using mainly descriptive statistics; means ± standard deviation, frequencies and percentages. The main outcome indicators were the demographic information (age, gender, marital status and ethnicity), socioeconomic information (highest educational attainment, employment status and occupation), and lifestyle, especially smoking status (never smoked, previous smoker or current smoker), alcohol intake (teetotaler, moderate drinker, heavy drinker) and physical activity habits (physically active or physically inactive). Others included clinical information (diagnosis for which referred, comorbidities, family history of NCD, date of referral to the Dietherapy Department, actual date of accessing dietetic care and reasons for delays if any). Others were anthropometry (BMI, waist circumference, WHR), body composition (percentage body fat, visceral fat, muscle mass,) and blood pressure (systolic blood pressure, diastolic blood pressure and pulse). The independent variables were those variables that did not depend on others. They included age, gender and ethnicity. All other

variables were dependent. Anthropometric indicators were all presented first as continuous variables for the estimation of means and standard deviations, and then coded into categorical variables based on their cut-off ranges. Differences in means between male and females were tested using the independent sample T-test. Significance was set at a $p \leq 0.05$. Pearson's Chi Square was used to test for associations between variables as well as to test for significant differences between categorical variables.

For the follow-up study, the main outcome indicators were the nutrient, energy and dietary fibre intake at baseline, 3 months and 6 months, nutritional status and body composition (BMI, WHR, WC, percentage body fat, visceral fat, muscle mass) at baseline, 3 months, and 6 months and blood biochemistry (fasting plasma glucose, 2-hour glucose, serum total cholesterol, triglycerides, high density lipoprotein cholesterol, low density lipoprotein cholesterol and C-reactive protein at baseline, 3 months and 6 months. In addition, were alcohol intake habits, physical activity status and smoking habits at baseline, compare to 3 and 6 months. Chi square test were used to analyze association in categorical data such as BMI classification, percentage body fat classification as well as frequency of intake of different foods within a week, at baseline, 3 months and 6 months follow-up. Significance was set at a $p \leq 0.05$. Differences in mean macronutrient, energy and dietary fibre intake at baseline, 3 months and 6 months were determined using analysis of variance for repeated measures. Similarly, differences in mean blood biochemistry, blood pressure, nutritional status and body composition at baseline, 3 months and 6 months were also determined using analysis of variance for repeated measures. For variables which were skewed, median values at baseline, 3 and 6 months were reported. The non-parametric test for related samples, Friedman's two-way analysis of variance, was then used to determine significant differences between median values at baseline 3 and 6 months follow-up. Variables which showed

significant changes in both analyses were defined as dependent variables. Dunnett Post Hoc test (One-way ANOVA) was conducted. Using baseline means/medians as constants, mean/median values at 3 and 6 months were compared to baseline values to establish the stage at which the observed significant changes may have occurred within the study.

Variables that changed significantly over the period were defined as dependent variables, and all others as predictor variables in a multiple linear regression equation. Those that made significant predictions of each dependent variable were maintained in the model. Care was taken to ensure that there was no violation of the assumptions of regression, mainly of normality, linearity and multicollinearity. Skewed variables were log-transformed to ensure normality before entry into the model. Predictor variables were first correlated with the dependent variable and those that showed significant correlations with the dependent variable were then entered into the model, adjusting for stage of the study in each case.

3.11 Ethical issues

The study conformed to the Helsinki Declaration on Human Experimentation of 1975, revised in 2013. The proposal was submitted to the University of Ghana, School of Allied Health Sciences Ethics Review Committee, for ethics approval, before the study was carried out. Permission was also sought from the Dietherapy Department of the Korle- Bu Teaching Hospital. Consent was also sought from patients before being recruited into the study.

The purpose of the study was explained to each eligible study participant. It was explained that participation was entirely voluntary and that refusal to participate was not going to affect or influence the care they received at the Dietherapy Department. Patients were also informed that they had the right to withdraw consent at any time after agreeing to take part

in the study. It was explained that, they will go through the inconvenience of spending time to answer questions about themselves. Their height, weight, blood pressure, waist and hip circumference as well as body composition was also going to be measured. The additional risk of having 12 ml of blood (Less than 3 tea spoons) drawn to measure substances in the bloodstream that may increase cardiovascular risk was to be experienced. However, it was explained that the body contains the equivalent of five large beer bottles of blood. Therefore 3 tea spoon full of blood was nothing compared to the amount of blood in their body and would therefore not affect their health. Secondly the amount of blood needed was similar to what was routinely demanded at the laboratories for regular medical checkup and would not harm them. Patients were made to understand that the processes would be obtained at baseline and repeated after 3 months (13 weeks) of recruitment and at 6 months (26 weeks) after recruitment.

The confidentiality of their personal information was assured. They were not going to be identified by name in any scientific reporting of the study. Their information was only going to be known by the research team. All their questions were answered there, and in addition, a contact phone number was also provided for any further questions they may have. A consent form was then signed or thumb printed by each subject before their participation in the study.

CHAPTER FOUR

4.0 RESULTS

Cross-sectional study

4.1. Specific objective 1: To determine the demographic and socioeconomic characteristics of patients.

The demographic information of all patients is summarized in Table 4.1.1a and 4.1.1b. A total of 339 patients participated in this study. They comprised 104 (30.7%) males and 235 (69.3%) females. Their mean age was 49.3 ± 11.9 years. This was significantly higher among males compared to females ($p < 0.001$).

Table 4.1.1a: Age, of study participants. Mean ± SD

Variable	Male *n* = 104	Female *n* = 235	Total *n* = 339	P-value
Age (years)	50.7 ± 12.9	48.7 ± 11.4	49.3 ± 11.9	P < 0.001*
Range	(20 – 80)	(19 – 81)	(19 -81)	

*Independent sample T test: Significant at $p < 0.05$

In Table 4.1.1b, the majority of study participants were married. This was significantly higher among males compared to females. Akans formed the major ethnic group. Christianity was the most common religion. These did not differ significantly between males and females.

Table 4.1.1b: Demographic information of the study participants. Frequency (%)

Variable	Male *n* = 104	Female *n* = 235	Total *n* = 339	P- values
Marital status				
Divorced	2 (1.9)	18 (7.7)	20 (5.9)	P = 0.043*
Married	77 (74.0)	149 (63.4)	226 (66.7)	
Single	21 (20.2)	39 (16.6)	60 (17.7)	
Others[a]	4 (3.9)	29 (12.3)	33 (9.7)	
Total	**104 (100.0)**	**235 (100.0)**	**339 (100.0)**	
Ethnicity				
Akan	45 (43.3)	109 (46.4)	154 (45.4)	P = 0.63
Ewe	19 (18.3)	41 (17.4)	60 (17.7)	
Ga	29 (27.8)	71 (30.2)	100 (29.5)	
Others[b]	11 (10.6)	14 (6.0)	25 (7.4)	
Total	**104 (100.0)**	**235 (100.0)**	**339 (100.0**)	
Religion				
Christian	95 (91.3)	217 (92.3)	312 (92.0)	P = 0.747
Others[c]	9 (8.7)	18 (7.7)	25 (8.0)	
Total	**104 (30.7)**	**235 (69.3)**	**339 (100.0)**	

* Pearson's Chi square: significant at $p \leq 0.05$.

[a] Others were: separated (0.3%), widowed (6.2%) and not disclosed (3.2%)

[b] Others were: Nigerians (0.9%) and Northerners (6.5%)

[c] Others were: Muslims (7.4%), Buddhist (0.3%) and Naturalist (0.3%)

In Table 4.1.2, the socioeconomic characteristics of all the participants are described. The highest educational attainment for the majority was the primary level. This was significantly higher among females compared to males. The majority of all participants were employed. Significantly more females were unemployed compared to males. Among the employed, a significant majority of females were technicians/ drivers/ artisans/ traders/ security persons compared to males, while significantly more males were in the higher skilled professions such as doctors/ dentist/ administrator/ certified accountants/ engineers.

Table 4.1.2: Socioeconomic characteristics of the study participants. Frequency (%)

Variable	Male *n* = 104	Female *n* = 235	Total *n* = 339	P-value
Education				
At most primary	35 (33.7)	123 (52.3)	158 (46.6)	P = 0.001*
Secondary	30 (28.8)	65 (27.7)	95 (28.0)	
Tertiary	39 (37.5)	47 (20.0)	86 (25.4)	
Total	**104 (100.0)**	**235 (100.0)**	**339 (100.0)**	
Employment				
Employed	81 (77.9)	181 (77.0)	262 (77.3)	P = 0.004*
Unemployed	9 (8.6)	42 (17.9)	51 (15.1)	
Pensioner	14 (13.5)	12 (5.1)	26 (7.6)	
Total	**104 (100.0)**	**235 (100.0)**	**339 (100.0)**	
Occupation				
Unskilled labourer/ orderly/watchman	6 (5.8)	4 (1.7)	10 (2.9)	P < 0.001*
Technician/Driver/Artisan/ Tradesperson/Security	32 (30.8)	129 (54.9)	161 (47.5)	
Clerical/Audit/Accounts/Messenger/ Office worker	13 (12.5)	12 (5.1)	25 (7.3)	
Nurse/Dietician/Technologist/ Secretary/Transport or Estates	13 (12.5)	13 (5.5)	26 (7.6)	
Doctor/Dentist/Administrator/ Certified Accountant/Engineer	12 (11.5)	8 (3.4)	20 (5.9)	
Others	5 (4.8)	15 (6.4)	20 (5.9)	
Total	**81 (77.9)**	**181 (77.0)**	**262 (77.3)**	

*: Pearson's Chi square: significant at $p \leq 0.05$. Others = Teachers, Rev. Minister, House wife

4.2. Results for specific objective 2: To determine the major diet related chronic diseases which were referred to the dietitians

In Fig 1, the major diet related chronic diseases which were referred to the dietitians are shown. Hypertension was the major condition referred. It was followed by diabetes mellitus, dyslipidaemia and obesity. Most patients referred had more than one chronic condition and, in some cases, other co-morbidities. The detailed list of referred conditions are shown in Appendix VIII.

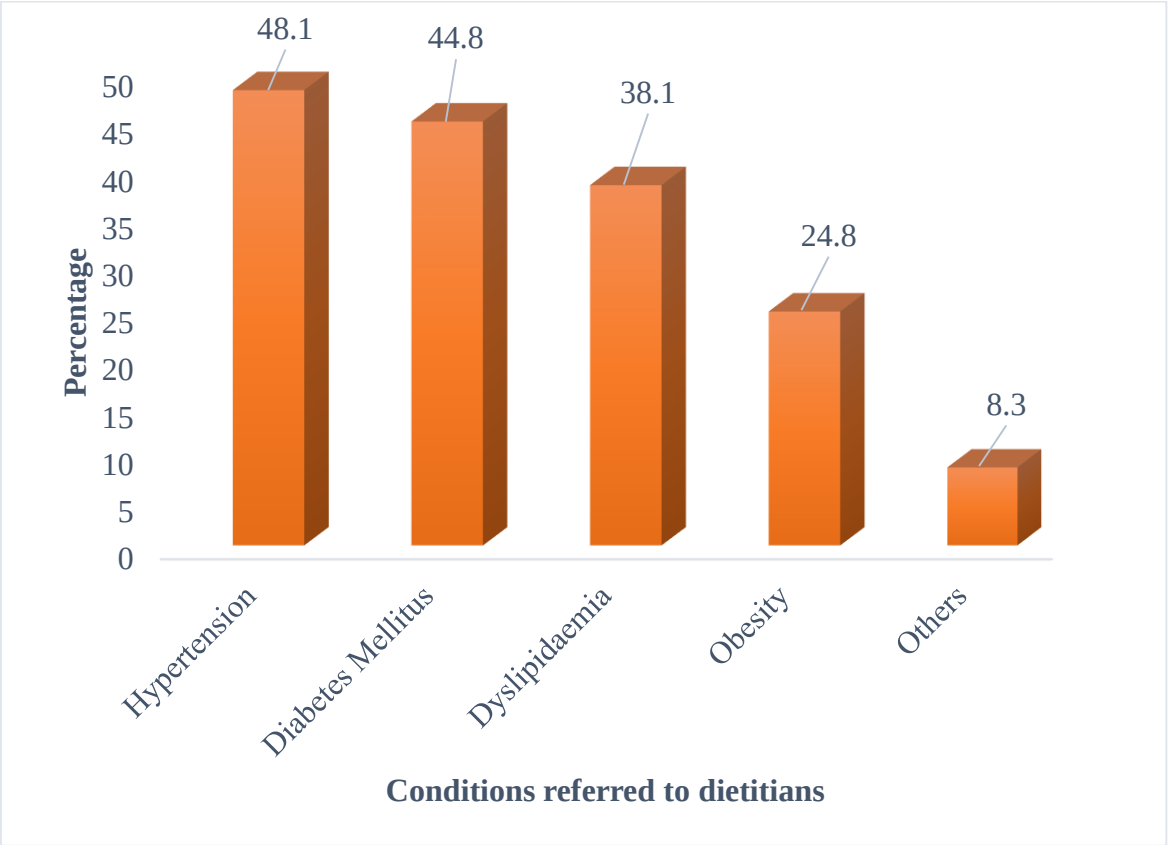


Fig 4.1: Major diet related chronic diseases that were referred to the dietitians.

4.3. Results for specific objective 3; To identify the dietetic care-seeking behaviour of patients after referral to see a dietitian and the challenges they encounter when accessing dietetic care.

4.3.1 Determination of dietetic care-seeking behaviour of patients after referral and associated challenges.

In Table 4.3.1, the dietetic care-seeking behaviour of patients after referral and associated challenges are reported. Care-seeking behaviour was categorized as prompt (seeking care on the same day of referral) or delayed (not seeking care on the same day of referral). In total, 50.1% of patients delayed in accessing dietetic care compared to 49.9% who sought and obtained dietetic care on the same day that they were referred by a medical doctor. Males and females equally delayed in accessing dietetic care. The reasons for their delays as well as the length of delay were also comparable. Among those who delayed, the main reason for delay was due to late reporting at the Dietherapy Department, on the same day they were referred, when it had already closed. Additional reasons for delay were given and included lack of time, patient being too sick and too weak to seek dietetic care on the same day of referral and travels to attend to business and family related issues. The majority delayed between 1 - 3 days while a few delayed more than 30 days.

Table 4.3.1: Patients' dietetic care-seeking behaviour after referral.

Variable	Male *n* = 104	Female *n* = 235	Total *n* = 104	P- value
Delay in seeking dietetic care				
Yes	56 (53.8)	114 (48.5)	170 (50.1)	P = 0.410
No	48 (46.2)	121 (51.5)	169 (49.9)	
Total	**104 (100.0)**	**235 (100.0)**	**339 (100.0)**	
Reasons for delay				
Time restraint	9 (8.6)	15 (6.4)	24 (7.1)	P = 0.610
The Dietherapy Dept. had closed	15 (14.4)	27 (11.5)	42 (12.4)	
Travelled	8 (7.7)	8 (3.4)	16 (4.7)	
Poor health	6 (5.8)	11 (4.7)	17 (5.0)	
Others [a]	18 (17.3)	53 (22.5)	71 (21.0)	
Total	**56 (53.8)**	**114 (48.5)**	**170 (50.1)**	
Length of delay				
1-3 days	27 (26.0)	45 (19.1)	72 (21.2)	P = 0.860
4 -7 day	9 (8.7)	20 (8.5)	29 (8.6)	
8-14 days	7 (6.7)	20 (8.5)	27 (8.0)	
15 -30 days	7 (6.7)	19 (8.1)	26 (7.7)	
More than 30 days	6 (5.7)	10 (4.3)	16 (4.7)	
Total	**56 (58.8)**	**114 (48.5)**	**170 (50.1)**	

*; Pearson's Chi square; significant at $p \leq 0.05$. Others[a] = Family responsibilities, misplaced folder/referral letter, caregiver not present to accompany them to the dietitian, didn't know they had to see a dietitian, did not get permission from work, forgetfulness, strike action by doctors, wanted to wait till my next visit to the doctor, lack of funds.

4.4. Results for specific objective 4: To assess the lifestyle of study participants.

4.4.1 Alcohol intake habits of study participants

The alcohol intake habits of patients are reported in Table 4.4.1. More than half of the participants had not consumed any alcohol for over 12 months preceding the study. The frequency of alcohol intake differed significantly between males and females, with more males consuming alcohol daily compared to females. The number of drinks consumed on a

typical day, also differed significantly between males and females. Significantly more males consumed more than 2 drinks daily compared to females.

Table 4.4.1: Alcohol intake habits of study participants. Frequency (%)

Variable	Male *n* = 104	Female *n* = 235	Total *n* = 339	P-value
Alcohol consumed within the past 12 months				
Yes	48 (46.2)	88 (37.4)	136 (40.1)	P = 0.150
No	56 (53.8)	147 (62.6)	203 (59.9)	
Total	**104 (100.0)**	**235 (100.0)**	**339 (100.0)**	
Frequency of intake				
Daily	8 (7.7)	2 (0.9)	10 (2.9)	P = 0.001*
1-6 days per week	13 (12.5)	12 (5.1)	25 (7.4)	
1-3 days per month	10 (9.6)	15 (6.4)	25 (7.4)	
Less than once a month	17 (16.4)	59 (25.1)	76 (22.4)	
Total	**48 (46.2)**	**88 (37.4)**	**136 (40.1)**	
Alcohol consumed within the past 7 days				
Yes	16 (15.4)	33 (14.1)	49 (14.5)	P = 0.740
No	88 (84.6)	202 (85.9)	290 (85.5)	
Total	**104 100.0)**	**235 (100.0)**	**339 (100.0)**	
Number of drinks on a typical day				
1 to 2 drinks	26 (25.0)	66 (28.0)	92 (27.1)	P < 0.001*
More than 2 drinks	17 (16.4)	8 (3.4)	25 (7.4)	
Could not estimate number of drinks	5 (4.8)	14 (6.0)	19 (5.6)	
Total	**48 (46.2)**	**88 (37.4)**	**117 (40.1)**	

*: Pearson's Chi square Analysis: Significant at $p \leq 0.05$.

4.4.2. Smoking habits of study participants

The smoking habits of study participants are reported in Table 4.4.2. Although in general, majority of participants never smoked, this was significantly higher among

females compared to males. Additionally, significantly more males previously smoked compared to females. Only a few patients currently continued to smoke.

Table 4.4.2: Smoking habits of study participants.

Variable	Male *n* = 104	Female *n* = 235	Total *n* = 339	P-value
Smoking habits				
Never smoked	80 (76.9)	229 (97.5)	309 (91.1)	$P^{*} = 0.01$
Previously smoked but has stopped	19 (18.3)	5 (2.1)	24 (7.1)	
Currently smokes	5 (4.8)	1 (0.4)	6 (1.8)	
Total	**104 (100.0)**	**235 (100.0)**	**339 (100.0)**	

*: Pearson's Chi square Analysis: Significant at $p \leq 0.05$.

4.4.3 Physical activity habits of study participants

The physical activity habits of patients are reported in Table 4.4.3. In general, majority of participants never engaged in moderate-intensity sports, fitness and recreational activity. Moderate-intensity sports, fitness or recreational activity was significantly associated with gender. Significantly more males engaged in these activities compared to females. The number of days activities were performed in a week was, however, not significantly associated with gender. Similarly, the time spent on these activities was not significantly associated with gender. Only few patients engaged in these activities for 30 minutes or more in a day.

Table 4.4.3: Physical Activity habits of study participants. Frequency (%).

Variable	Male *n* = 104	Female *n* = 235	Total *n* = 339	P-value
Moderate-intensity sports, fitness or recreational (leisure) activities continuously for at least 10 minutes				
Yes	33 (31.7)	46 (19.6)	79 (23.3)	P = 0.018*
No	71 (68.3)	189 (80.4)	260 (76.7)	
Total	**104 (100.0)**	**235 (100.0)**	**339 (100.0)**	
Number of days moderate activities are done in a week				
1-4 days in a week	18 (17.3)	29 (12.3)	47 (13.9)	P = 0.338*
≥ 5 days in a week	15 (14.4)	17 (7.3)	32 (9.4)	
Total	**33 (31.7)**	**46 (19.6)**	**79 (23.3)**	
Time spent on moderate activities				
< 30 mins	11 (10.6)	19 (8.1)	30 (8.8)	P = 0.472
≥ 30 mins	22 (21.1)	27 (11.5)	49 (14.5)	
Total	**33 (31.7)**	**46 (19.6)**	**79 (23.3)**	

*: Pearson's Chi square Analysis: Significant at $p \leq 0.05$.

4.4.3.1 Reasons why patients either engaged in physical activity or not.

The reasons why some patients did not engage in physical activity and why others did are illustrated in Fig 4.2a and Fig 4.2b respectively. From Fig 4.2a, among the over 76% of patients who did not engage in physical activity, the main reason for the majority was due to lack of time. It was followed by laziness, having an already active career and the body aches associated with physical activity, Fig 4.2a.

For the over 23% who did engage in some physical activity, the main reason was to stay fit and healthy. Others wanted to lose weight, exercised as a means of transport and because they had been advised by a doctor to do so, Fig 4.2b.

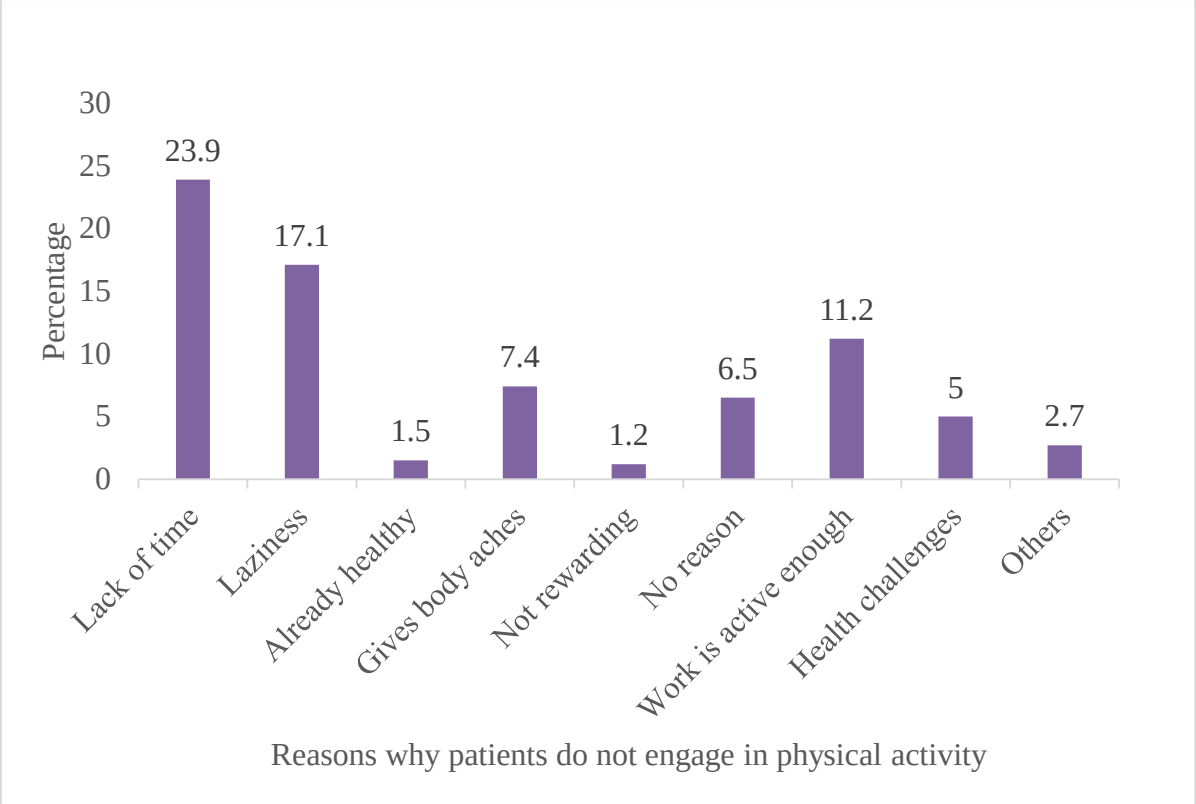


Fig 4.2a: Reasons why some patients did not engage in physical activity

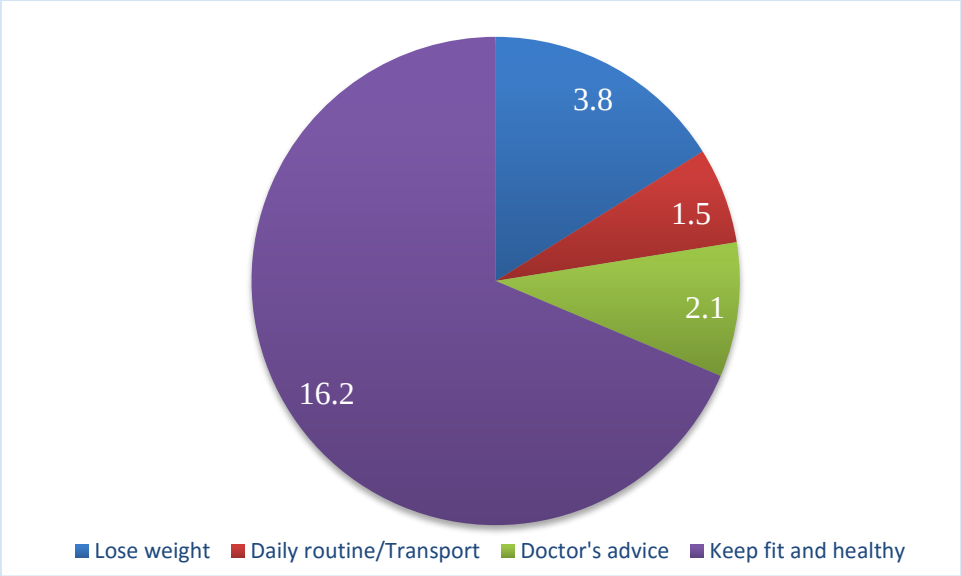


Fig 4.2b: Reasons why some patients engaged in physical activity

4.5 Results for follow-up study

Results of the follow-up study, is presented in this section. The primary aim of this study was to audit the effectiveness of the routine dietary and lifestyle intervention given to patients at the Dietherapy Department, on their dietary habits, alcohol intake, physical activity habits, smoking habits, anthropometry, body composition, blood pressure and biochemical indicators. This phase had 5 specific objectives. The results are reported below. It begins with the background characteristics of study participants.

4.5.1 Background information of follow-up patients

A total of 132 patients were sampled from the cross-sectional study and followed up for 6 months. They included 97 females and 35 males. In Table 4.5.1 below, there was no significant difference in the mean ages of males compared to females.

Table 4.5.1: Age profile of follow-up patients

	N	**Mean Age (years) ± SD**	**Range**	**P value**
Male	35	50.7 ± 11.8	27 - 68	P = 0.458
Female	97	49.2 ± 9.6	20 - 72	
Total	132	49.6 ± 10.2	20 - 72	

*; Independent sample T-test; significant at $p \leq 0.05$.

4.5.2. Medication intake habits of patients

A summary of the medications taken by patients is reported in Table 4.5.2a. below. Antihypertensive agents were taken by the majority of patients. Other medications included oral hypoglycaemic agents, statins and anti-platelets.

Table 4.5.2a: Summary of medications taken by patients

Type of medication	Frequency (%)
Antihypertensive agents	84 (63.6)
Oral hypoglycaemic agents	55 (41.7)
Statins	50 (37.9)
Antiplatelet	11 (8.3)
Insulin	4 (3.0)
Antidepressants	4 (3.0)
Diuretics	3 (2.3)
Antacids	2 (1.5)
Proton pump inhibitors	1 (0.8)
Antibiotics	1 (0.8)

Table 4.5.2b below details the medication intake habits of patients. At baseline, 85.6% were on medication while 14.4 were not (only required to undergo lifestyle modification). The proportions of those who regularly took their medication as prescribed progressively decreased at 3 and 6 months. Main reason why patients failed to take their medication regularly was because it was finished and due to negative side effects.

Table 4.5.2b: Medication intake habits of patients. Frequency (%)

Variable	Baseline *n* = 132	3 Months *n* = 80	6 Months *n* = 60
Patient was put on medication			
Yes	113 (85.6)	71 (88.7)	55 (91.7)
No	19 (14.4)	9 (11.3)	5 (8.3)
Total	**132 (100.0)**	**80 (100.0)**	**60 (100.0)**
Regularly takes medication as prescribed			
Yes	-	61 (76.2)	43 (71.6)
No	-	10 (12.5)	12 (20.0)
Total	-	**71 (88.7)**	**55 (91.6)**
Reasons for not taking medication as prescribed			
Medication got finished	-	6 (7.5)	7 (11.7)
Because I feel better now	-	2 (2.5)	-
Stopped taking them due to side effects	-	1 (1.25)	3 (5.0)
Could not afford the medicines	-	-	2 (3.3)
No reason	-	1 (1.25)	-
Total	-	**10 (12.5)**	**12(20.0)**

4.5.3 Patients attendance at dietetic reviews

In Table 4.5.3 below, the number of dietetic reviews attended by patients between baseline and 3 months and between 3 and 6 months are reported. A significant majority of patients attended 3 or more reviews between baseline and 3 months compared to between 3 and 6 months. Significantly more patients did not attend any review between 3 and 6 months compared to between baseline and 3 months. Three and six months reports are based on 80 and 60 patients respectively as the remaining were lost to attrition.

Table 4.5.3: Number of dietetic reviews attended by patients between baseline, 3 months and 6 months. Frequency (%)

Number of reviews	Between baseline and 3 months	Between 3 and 6 months	P value
0	13 (16.2)	14 (23.3)	P = 0.009*
1	12 (15.0)	18 (30.0)	
2	18 (22.5)	19 (31.8)	
3	19 (23.7)	5 (8.3)	
4 and more	18 (22.6)	4 (6.6)	
TOTAL	**80 (100.0)**	**60 (100.0)**	

*: Pearson's Chi square; significant at $p \leq 0.05$

In Fig 4.5.3 below, a significant association was observed between gender and the number of reviews attended in favour of females ($\chi = 16.6$; $p = 0.011$).

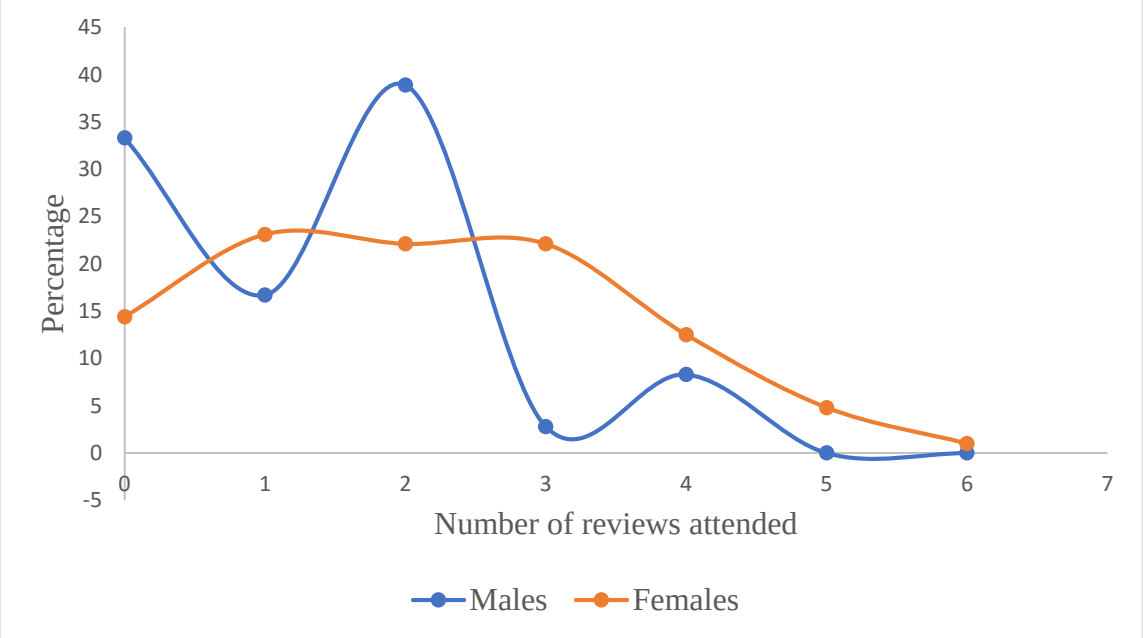


Fig 4.2c: Association between gender and number of dietetic reviews attended

4.6. Summary of interventions given to patients

Interventions given to patients covered both dietary and other lifestyle interventions.

4.6.1. Dietary interventions given to patients at the Dietherapy Department of KBTH

In Table 4.6.1 below, a summary of the interventions which was given to patients is shown. All patients were asked to increase their dietary fibre intake. A low sodium diet was prescribed for 78.8% of patients. Another (62.1%) of patients were put on a low fat, low cholesterol and saturated fat diet. Weight-reducing diet was next and recommended for 28.8% of patients.

Table 4.6.1: Summary of the dietary interventions which were given to patients.

Intervention	Frequency (%)	Goal
High fibre diet	132 (100.0)	> 25 g of fibre daily (At least 5 servings of fruits and vegetables daily and inclusion of more whole grains in routine diet).
Weight reducing diet	38 (28.8)	Energy expenditure must exceed energy intake. Individualized daily energy intake recommended for patients ranged from 1500 kcal to 1800 kcal.
Low sodium diet	104 (78.8)	Reducing daily salt intake to ≤ 5g of salt (i.e. about 1 tea-spoon of salt).
Low fat, cholesterol and saturated fat diet	82 (62.1)	25% to 35% of total calories Cholesterol = < 200mg / day PUFA = up to 10% of total calories MUFA = up to 20% of total calories Saturated fat < 7% of total daily calories

*: Goals are supported by various international guidelines such as the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III), IDF 2012, UK NICE, 2011

4.6.2. Other lifestyle interventions

The lifestyle intervention given to patients is summarized in Table 4.6.2 below. They covered the areas of physical activity, alcohol intake and smoking habits. All patients were advised to carry out moderate-intensity physical activities for at least 150 minutes weekly, i.e. ≥ 30 minutes, 5 days in a week. Male and female patients who consumed alcohol were advised to limit their intake to at most 2 drinks and 1 drink respectively in a day. Patients who smoked were advised to stop smoking.

4.6.2: Lifestyle intervention given to patients

Intervention	Group of patients	Goal
Physical activity	All patients	At least 30 minutes of moderate-intensity physical activity (e.g. brisk walking) 5 or more days weekly
Alcohol	Males who drink	At most 2 drinks daily
	Females who drink	At most 1 drink daily
	Teetotalers	Continue to abstain
Smoking	For current smokers	Stop smoking
	All others	Continue to abstain

4.7. Results for specific objective 1: To compare the mean / median nutrient, energy and dietary fibre intakes and the usual dietary intakes of patients at baseline to that at 3 months and 6 months.

The differences in mean / median nutrient, energy and dietary fibre intake of patients at baseline compared to 3 and 6 months are shown in Table 4.7.1a and 4.7.1b below. Although the intakes of some nutrients such as protein, fat and sodium decreased, and energy increased over the period, the only significant changes were an increase in the intake of carbohydrate and dietary fibre between baseline and 6 months.

Table 4.7.1a: Mean / *Median* nutrient, energy and fibre intake of patients at baseline compared to 3 months and 6 months follow-up (Mean ± SD)

Variables	Baseline	3 Months	6 Months	P value
		(*n* = 60)		
Carbohydrate (g)	202.4 ± 110.5	220.1 ± 87.1	251.3 ± 95.4	P = 0.010*
Protein (g)	49.8 ± 24.4	42.3 ± 22.6	46.9 ± 18.0	P = 0.132
Fat (g)	41.2 ± 31.5	35.7 ± 18.1	34.5 ± 16.9	P = 0.195
Energy (Kcal)	1403 ± 647	1367 ± 544	1497 ± 482	P = 0.344
Fibre (g)	16.6 ± 10.8	18.4 ± 9.1	20.8 ± 7.9	P = 0.025*
Saturated fat (g)	5.0 ± 14.8	2.9 ± 7.2	1.7 ± 1.8	P = 0.177
Median	*1.3*	*1.5*	*1.1*	*P = 0.649*
Total cholesterol (mg)	43.7 ± 76.7	23.6 ± 45.1	25.1 ± 48.3	P = 0.079
Median	*14.7*	*7.8*	*7.4*	*P = 0.074*
Sodium (mg)	954.1 ± 2311.6	534.7 ± 433.2	498.6 ± 398.7	P = 0.168
Median	*452.5*	*432.3*	*390.5*	*P = 0.430*
Potassium (mg)	746.1 ± 784.8	780.0 ± 628.3	741.0 ± 511.3	P = 0.907
Median	*395.0*	*487.8*	*605.3*	*P = 0.394*

*; Significant at P ≤ 0.05; Greenhouse Geisser Test of within-Subject Effect, (repeated measures ANOVA).

The significant increase in the intake of carbohydrates occurred among females, but not males. Conversely the significant increase of dietary fibre within the period occurred among males but not among females, Table 4.7.1b.

Table 4.7.1b: Mean / *Median* nutrient, energy and fibre intake of male and female patients at baseline compared to 3 and 6 months follow-up (Mean ± SD)

Variables	Baseline	3 Months	6 Months	P value
Males (*n* = 15)				
Carbohydrate (g)	183.1 ± 83.4	237.5 ± 119.7	235.2 ± 60.0	P = 0.171
Protein (g)	59.6 ± 19.9	46.7 ± 30.5	49.5 ± 17.5	P = 0.326
Fat (g)	48.7 ± 35.6	32.2 ± 14.3	36.8 ±17.5	P = 0.135
Energy (Kcal)	1447 ± 543	1445 ± 750	1454 ± 325	P = 0.993
Fibre (g)	13.1 ± 8.8	18.2 ± 7.5	19.8 ± 6.0	P = 0.032*
Saturated fat (g)	4.6 ± 12.1	1.9 ± 1.9	2.1 ± 2.3	P = 0.431
Median	*0.9*	*0.9*	*1.1*	*P = 0.903*
Total cholesterol (mg)	40.7 ± 80.3	11.7 ±17.2	33.1 ± 66.2	P = 0.432
Median	*7.4*	*9.6*	*16.9*	*P = 0.286*
Sodium (mg)	894.8 ± 1099.9	486.2 ± 265.7	634.1 ± 478.6	P = 0.285
Median	*627.0*	*430.5*	*527.5*	*P = 0.584*
Potassium (mg)	481.4 ± 524.7	647.6 ± 600.0	850.5 ± 529.0	P = 0.140
Median	*380.5*	*388.0*	*720.0*	*P = 0.062*
Females (*n* = 45)				
Carbohydrate (g)	208.8 ± 118.3	214.3 ± 74.0	256.6 ± 104.6	P = 0.020*
Protein (g)	46.5 ± 25.1	40.8 ± 19.4	46.1 ±18.3	P = 0.282
Fat (g)	38.7 ± 30.1	36.9 ± 19.2	33.8 ± 16.8	P = 0.505
Energy (Kcal)	1388 ± 684	1341 ± 463	1512 ± 527	P = 0.245
Fibre (g)	17.7 ± 11.3	18.4 ± 9.7	21.1 ± 8.5	P = 0.162
Saturated fat (g)	5.2 ± 15.7	3.2 ± 8.3	1.6 ± 1.6	P = 0.261
Median	*1.4*	*1.6*	*0.9*	*P = 0.607*
Total cholesterol (mg)	44.7 ± 70.9	27.5 ± 50.7	22.4 ± 41.3	P = 0.101
Median	*14.7*	*7.8*	*4.5*	*P = 0.134*
Sodium (mg)	971.2 ± 2567.3	548.7 ± 472.2	459.4 ± 369.4	P = 0.247
Median	*382.0*	*434.0*	*386.5*	*P = 0.656*
Potassium (mg)	828.4 ± 837.4	821.2 ± 637.7	706.9 ± 506.9	P = 0.526
Median	*419.0*	*608.5*	*602.0*	*P = 0.595*

*; Significant at P ≤ 0.05; Greenhouse Geisser Test of within-Subject Effect, (repeated measures ANOVA). *P; ≤ 0.05; Non parametric test for related samples, Friedman's two-way analysis of variance.*

Post-hoc analysis to establish the period during which the significant increases occurred is also shown in Table 4.7.1c below. The increment in the intake of both dietary fibre and carbohydrate was significant in the long term (between baseline and 6 months), but not in the short term (between baseline and 3 months).

Table 4.7.1c: Post hoc analysis showing significant variation in the mean macronutrients intakes at baseline compared to 3 months and 6 months (p values).

Variable	Baseline vs 3 Months	Baseline vs 6 Months
Dietary fibre	0.472	0.028*
Carbohydrate	0.511	0.013*

*; Significant at $P \leq 0.05$; Dunnett Post Hoc test (One-way ANOVA).

In Table 4.7.1d below, macronutrient and fibre intakes at baseline, 3 and 6 months are compared to recommended daily allowance (RDA) and acceptable macronutrient distribution ranges (AMDR). Carbohydrate intake exceeded RDA at all 3 stages of the study with the least intake at baseline. It also exceeded the AMDR at 6 months. Protein intake was similarly above the RDA at all stages except among females at 3 months. Males reported the highest protein intake at baseline. Total fat intake progressively decreased from baseline through to 6 months, though at each stage, intake was within the AMDR. The least total fat intake (20.7%) was at 6 months. Dietary fibre intake at all stages was below the RDA for both males and females with lowest intakes at baseline (pre-intervention).

Table 4.7.1d: Macronutrients and dietary fibre intake at baseline, 3 and 6 months compared to RDA and AMDR

Nutrient	RDA	AMDR USA	AMDR GHANA	AMDR Baseline	AMDR 3 Months	AMDR 6 Months
CHO		45% – 65%	40% - 65%	57.7%	64.4%	67.1%
	130 g/d			202g	220g	251g
Protein		10% – 35%	5% -10% or 20% - 30%	14.2%	12.4%	12.5%
Males	0.8/kg/d			1.2 g/kg	0.9 g/kg	1.0 g/kg
Females				0.9 g/kg	0.8 g/kg	0.9 g/kg
Total				1.0 g/kg	0.9 g/kg	0.9g/kg
Total fat	NA	20% – 35%	10% - 15%	26.4%	23.5%	20.7%
Fibre			>25g/d			
Males	38 g/d			13.1 g/d	18.2 g/d	19.8 g/d
Females	25 g/d			17.7 g/d	18.4 g/d	21.1 g/d
Total				16.6 g/d	18.4 g/d	20.8 g/d

CHO = carbohydrate, RDA = Recommended daily allowance, AMDR = Acceptable macronutrient distribution range, NA = Not available, USA = United States of America.

Ref.: (Ministry of Health, 2010, U.S. Department of Health and Human Services and U.S. Department of Agriculture, 2015).

Fig 4.3 below shows the proportion of patients who consumed foods made from whole grains in their usual diets, from baseline through to 6 months. Generally, significant increases occurred in the proportions of patients who included foods made from whole grains in their usual diets from baseline through 3 to 6 months APPENDIX Xa. The most frequently consumed whole grain foods by the majority of the people were *banku* followed by k*enkey* and wheat. At the third and sixth month, the majority consumed these foods at least two times in a week, APPENDIX IXa.

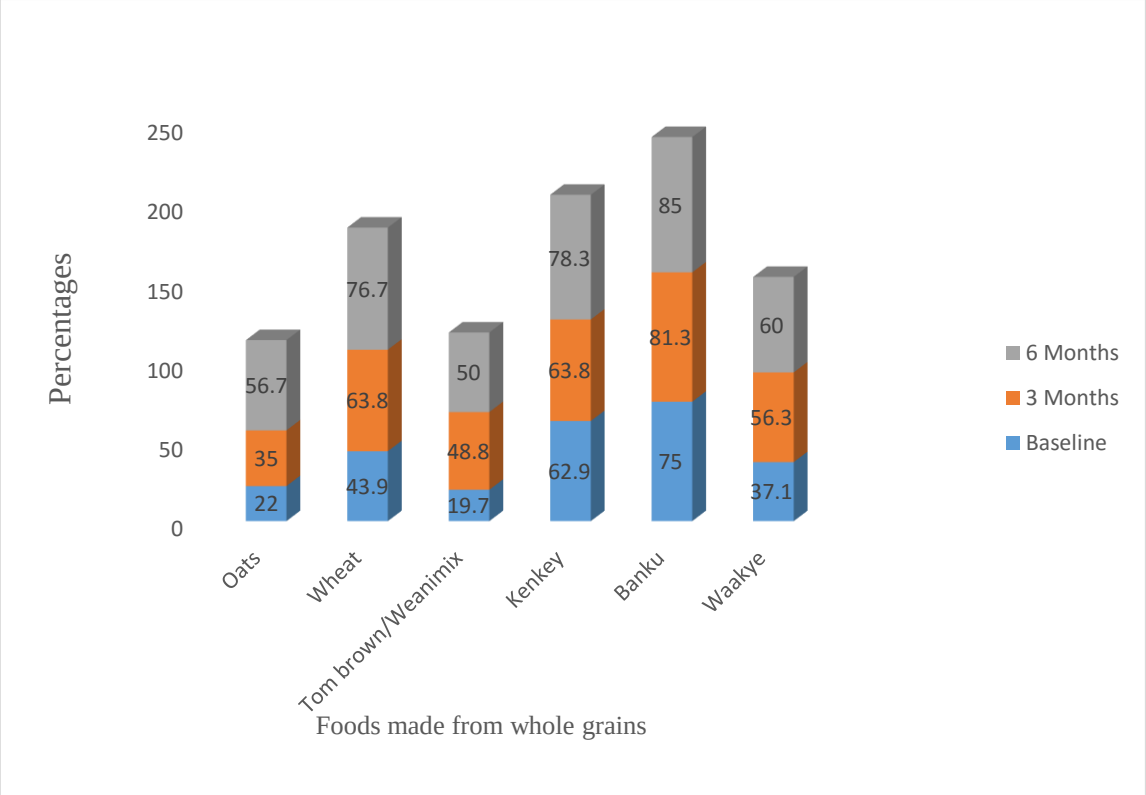


Fig 4.3 Proportion of patients who included foods made from whole grains in their usual diets at baseline, 3 months and 6 months

The percentage of patients who included common Ghanaian starchy foods in their usual diets at baseline, 3 months and 6 months are show in Fig 4.4 below. The most frequently consumed starches by the majority of patients were plain rice, boiled yam, boiled plantain, fufu and tea bread. They were mostly consumed two or more times weekly, Appendix IXb. There were no significant differences in the proportions of patients who consumed these foods between baseline and 6 months in most cases, APPENDIX Xb.

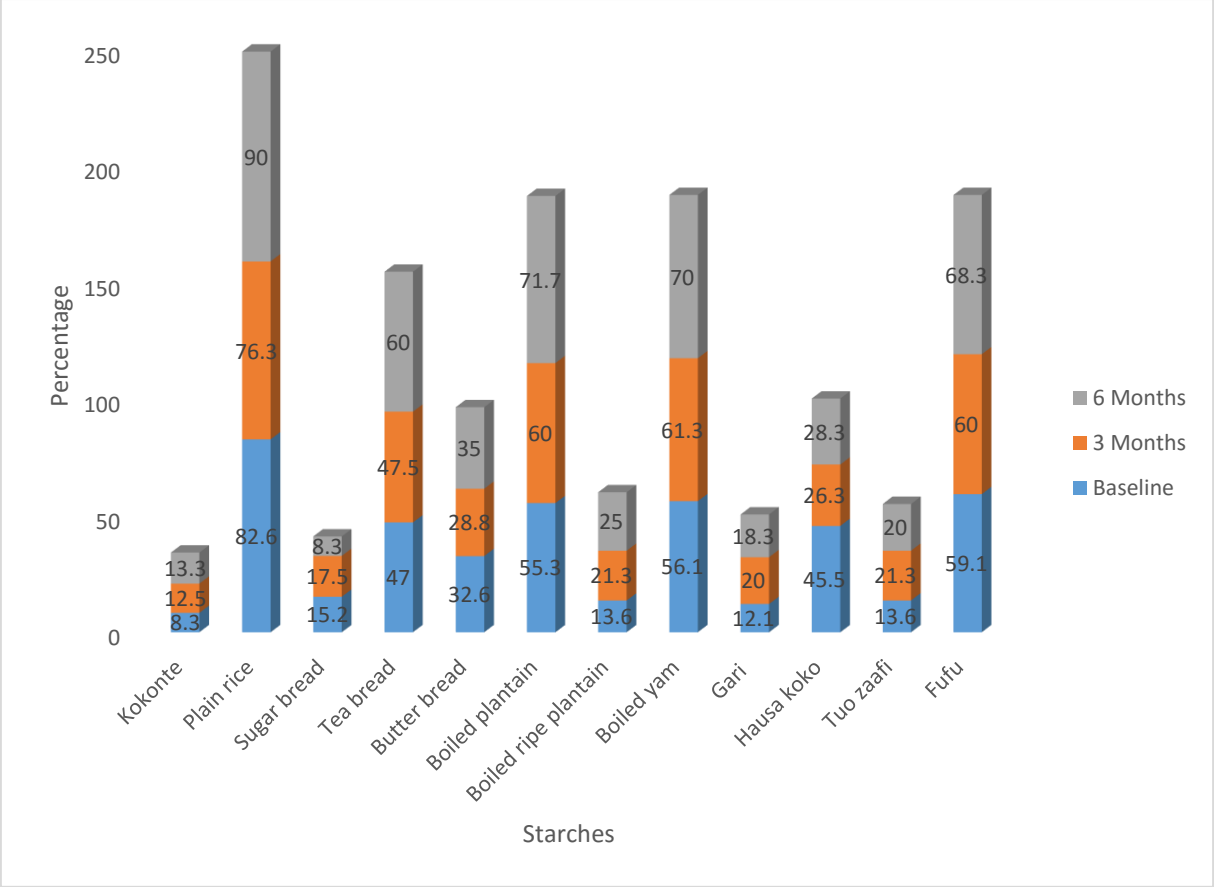


Fig 4.4: Percentage of patients who consumed common Ghanaian starchy foods at baseline 3 months and 6 months

The proportions of patients who included fruits as part of their usual diet at baseline, 3 months and 6 months is shown in Fig. 4.5 below. The most frequently consumed fruits by the majority of patients were pawpaw, orange, banana, water melon and apple. They were mostly consumed two or more times weekly, APPENDIX IXc. Generally, there were significant increases in the proportions of patients who consumed fruits as part of their usual diet between baseline and 6 months APPENDIX Xc.

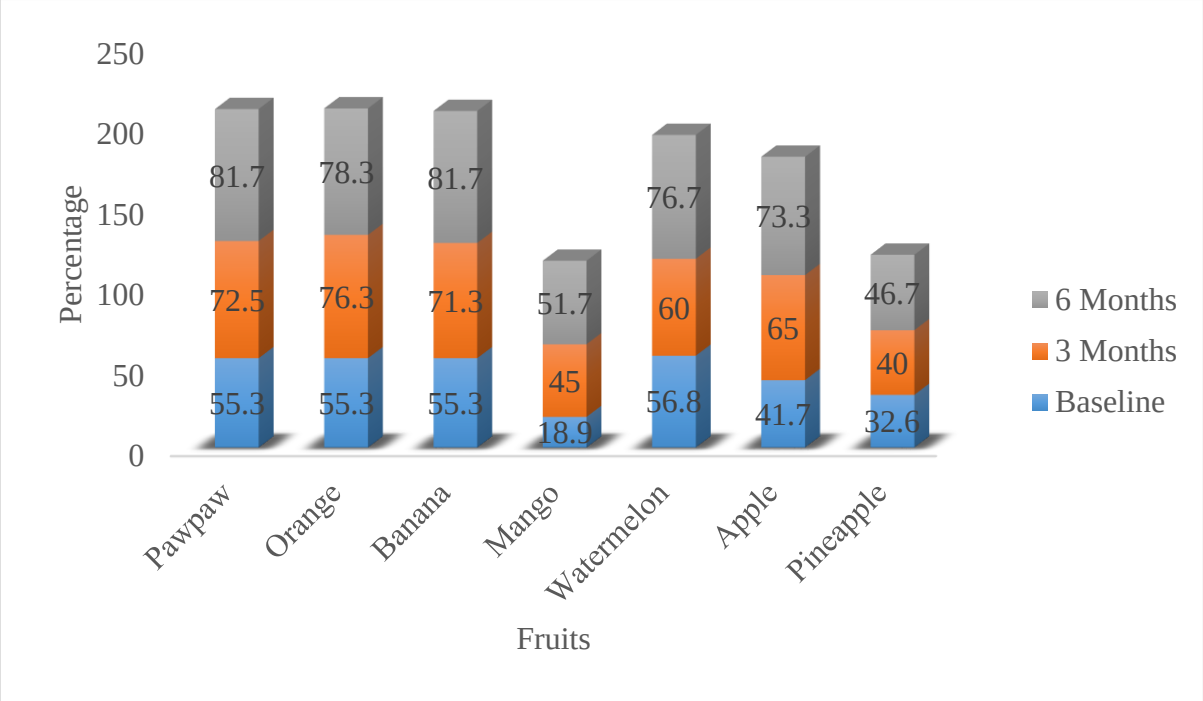


Fig 4.5: Proportions of patients who included common fruits in their usual diets at baseline compared to 3 months and 6 months.

In Fig 4.6 below, the proportion of patients who ate vegetables as part of their usual diet at baseline, 3 months and 6 months is shown. With the exception of cucumber, over 50% of patients ate various vegetables at baseline, and this figure increased significantly by 6 months APPENDIX Xd. All the vegetables studied were equally consumed with the most frequently consumed by the majority of patients being fresh tomatoes, carrot, *kontomire* and garden eggs. They were mostly consumed 2 or more times a week. APPENDIX IXd.

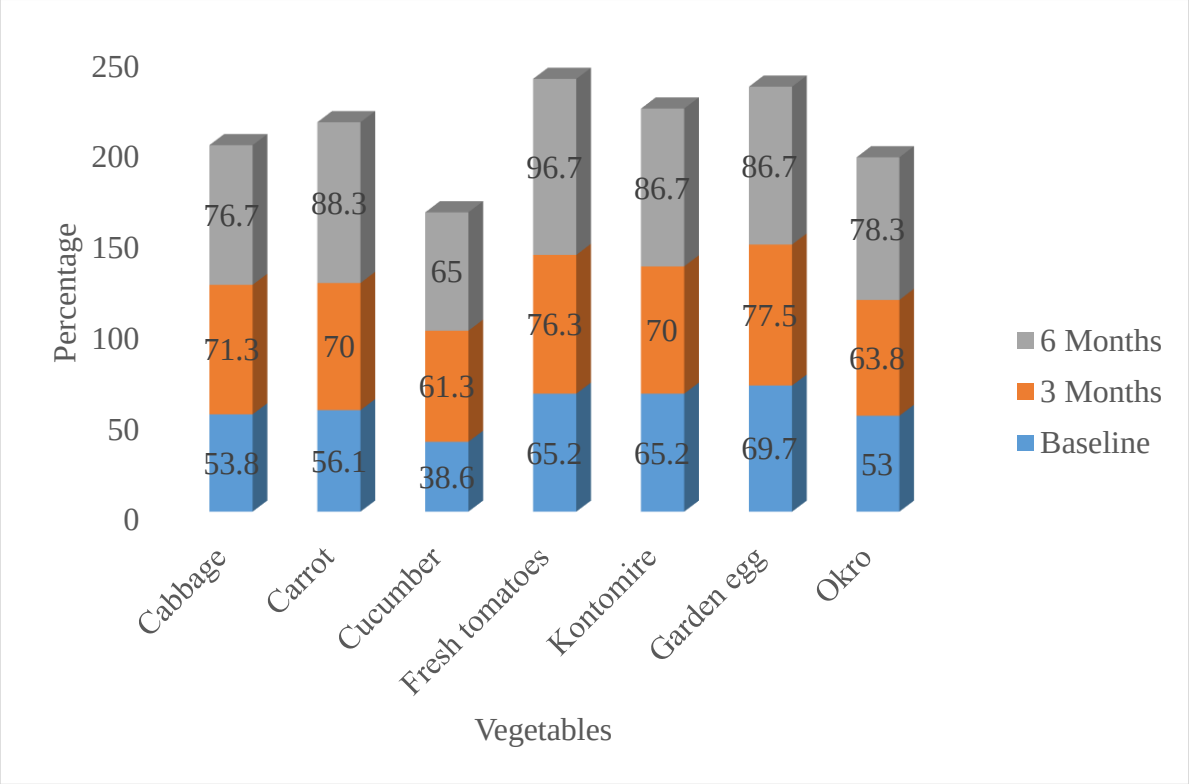


Fig 4.6: Proportion of patients who ate vegetables in their usual diets at baseline, 3 months and 6 months.

The proportions of patients who consumed fatty foods as part of their usual diets are illustrated in Fig 4.7 below. There were no significant changes in the proportions of patients who consumed fatty foods throughout the study APPENDIX.Xe. The most frequently consumed fatty foods by the majority of patients was fried fish followed by jollof rice, palm soup and groundnut soup.

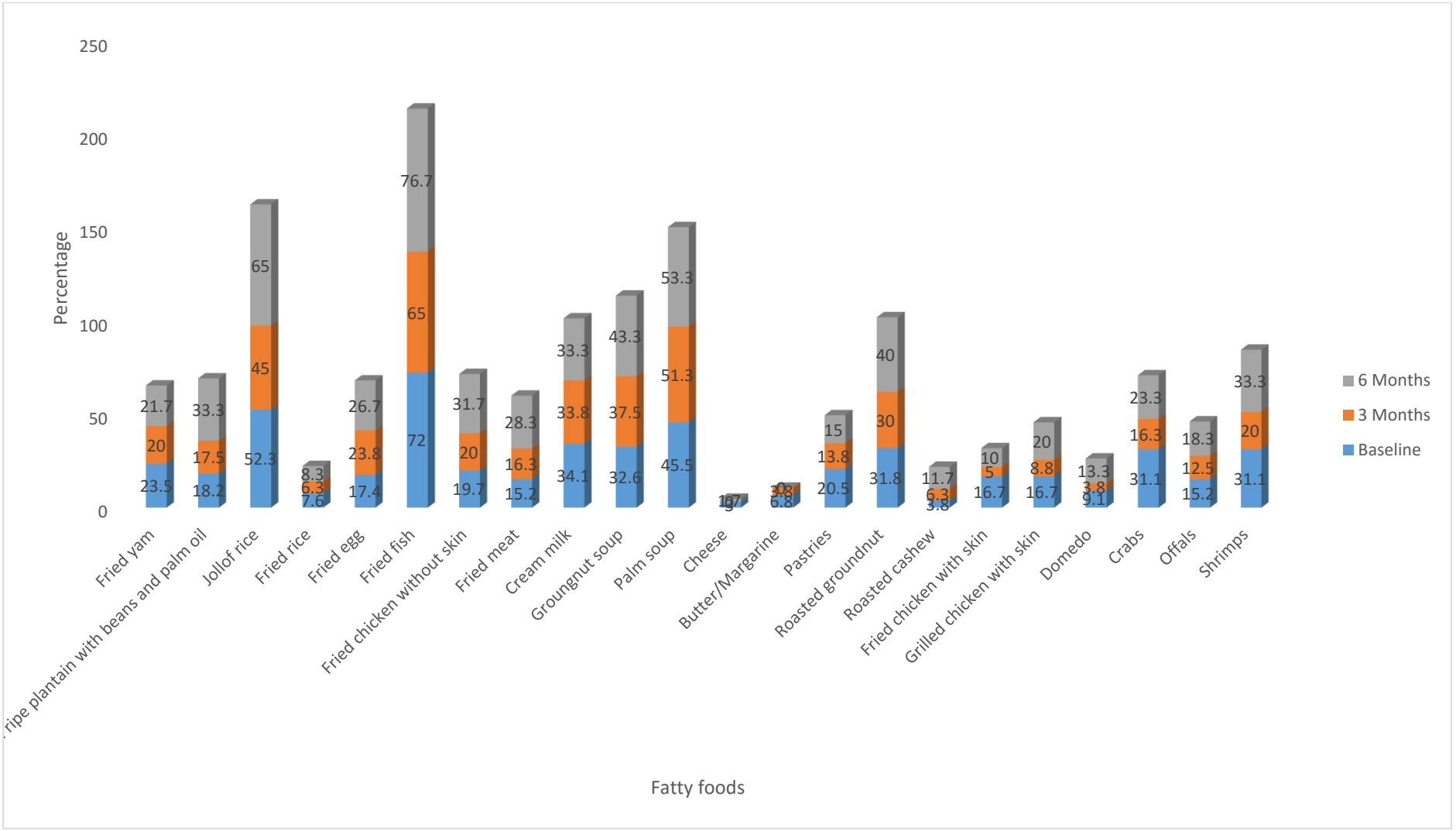


Fig 4.7: Percentage of patients who consumed fatty foods in their usual diet at baseline, 3 months and 6 months

The proportions of patients who consumed lean foods as part of their usual diets are illustrated in Fig 4.8 below. Most of these proportions did not change significantly throughout the study, APPENDIX Xf. The most commonly consumed lean food by the majority of patients was smoked fish. They mostly consumed them 2 or more times in a week, APPENDIX IXf.

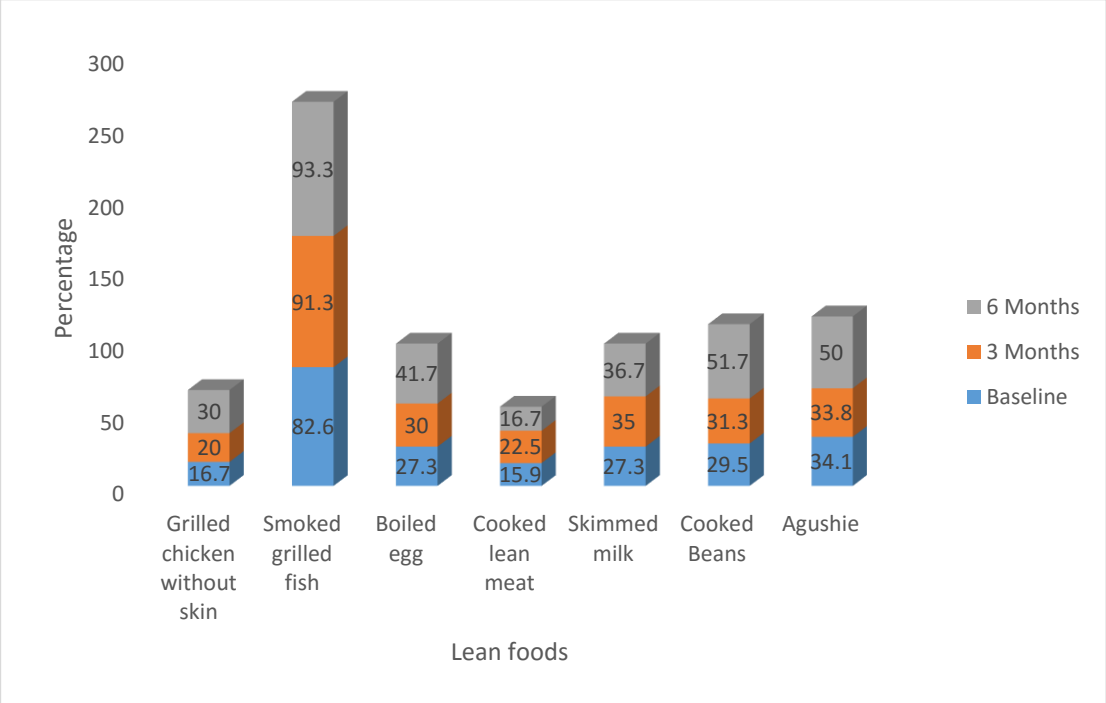


Fig 4.8 Proportion of patients who consumed lean foods at baseline, 3 months and 6 months

In Fig 4.9 below, the proportion of patients who included salted foods in their regular diet are illustrated. In general, not many patients consumed the common salted foods. With the exception of *dzomi*, *momoni*, *koobi* and maggi, less than 30% of patients consumed these foods from baseline to 6 months with no significant changes throughout the study period, APPENDIX Xg. The salted food mostly consumed by the majority of patients was *Dzomi*, followed by *maggi*, *momoni* and *koobi*, Fig 4.9.

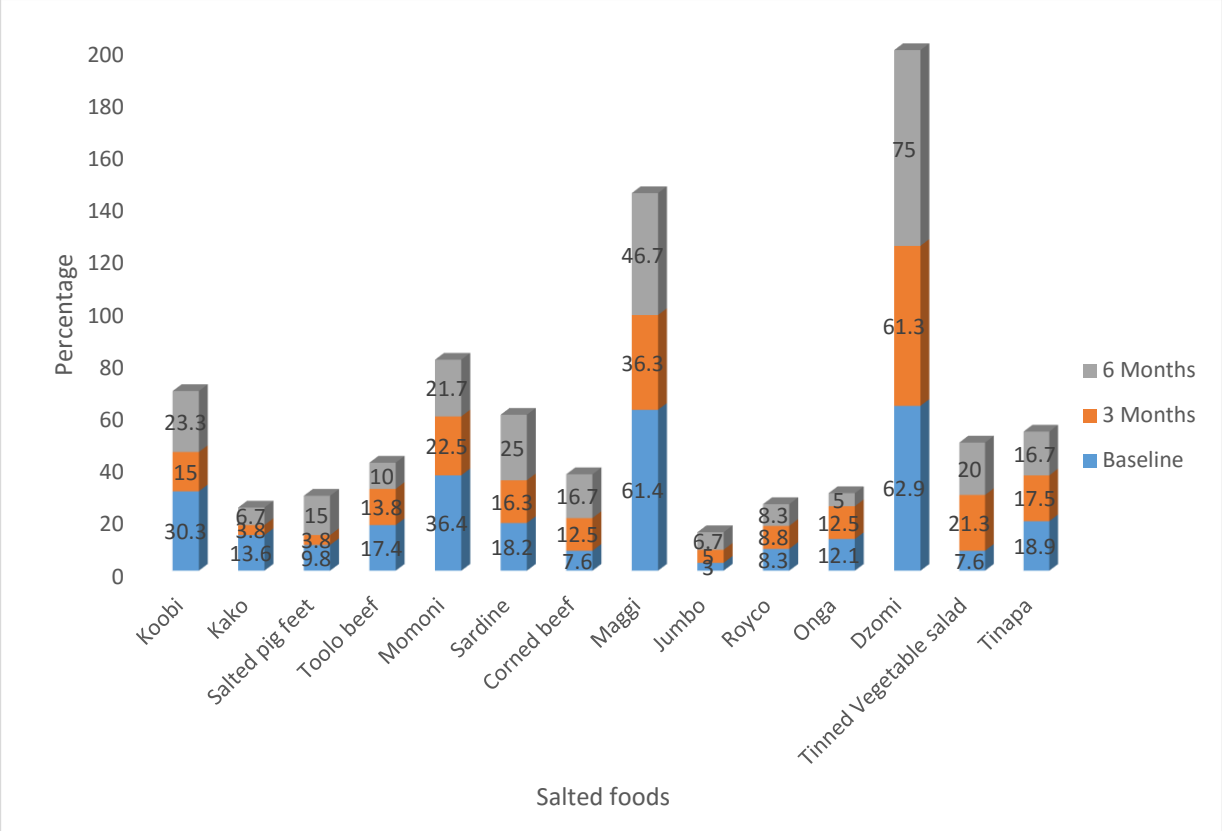


Fig 4.9 Proportion of patients who consumed salted foods at baseline, 3 months and 6 months

In Fig 4.10 below, the proportion of patients who consumed sweetened foods in their regular diet are illustrated. Over two thirds of patients did not include sweetened foods in their usual diets. Among those who did, the most commonly consumed sweetened food was biscuit. It was followed by fruit juices and malt drink. The proportions of patients who consumed these foods were comparable from baseline through to 6 months, APPENDIX Xh.

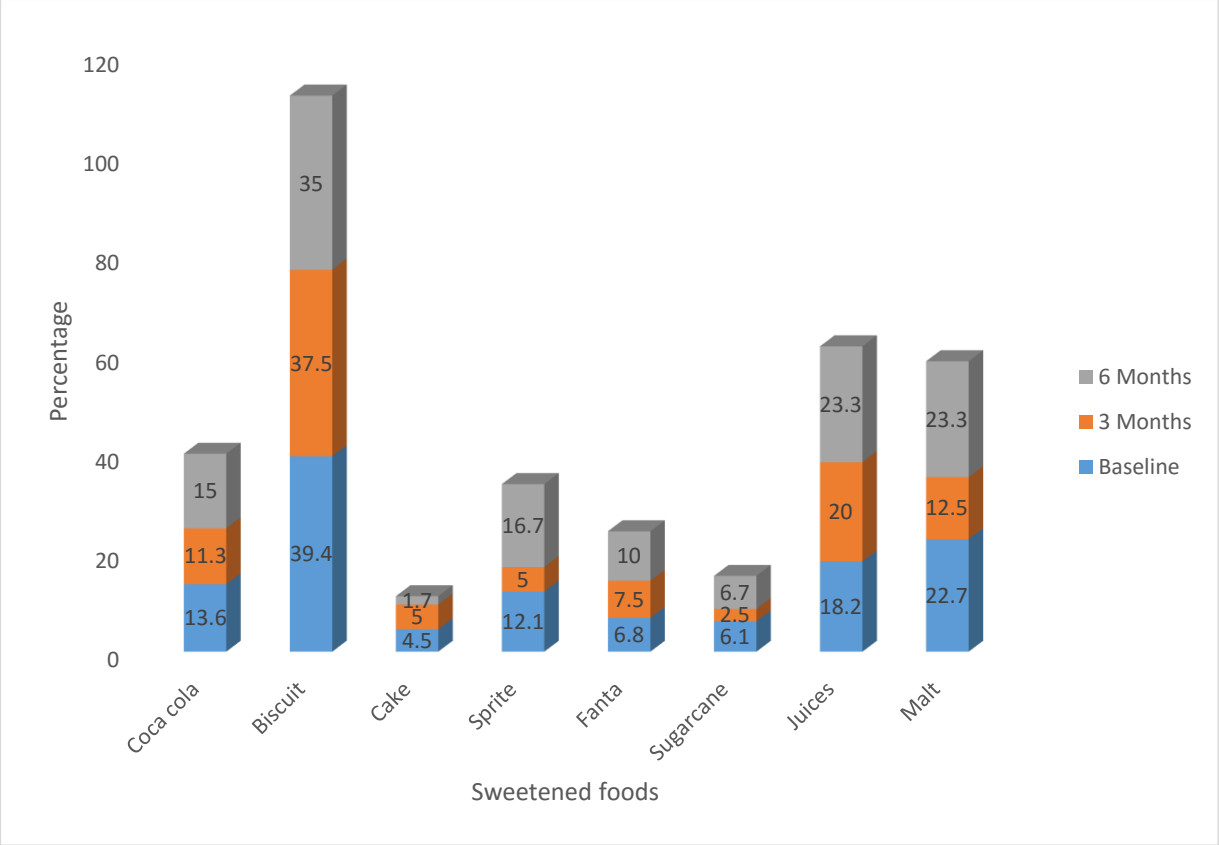


Fig 4.10: Proportion of patients who consumed Sweetened foods at baseline, 3 months and 6 months

In Fig 4.10 below, the proportion of patients who usually consumed alcoholic beverages are illustrated. Less than 15% of patients consumed alcoholic beverages throughout the study with no significant changes in their proportions over the perio, APPENDIX Xh. Beer, wine and Guinness were the most commonly consumed alcoholic beverage, Fig 4.10.

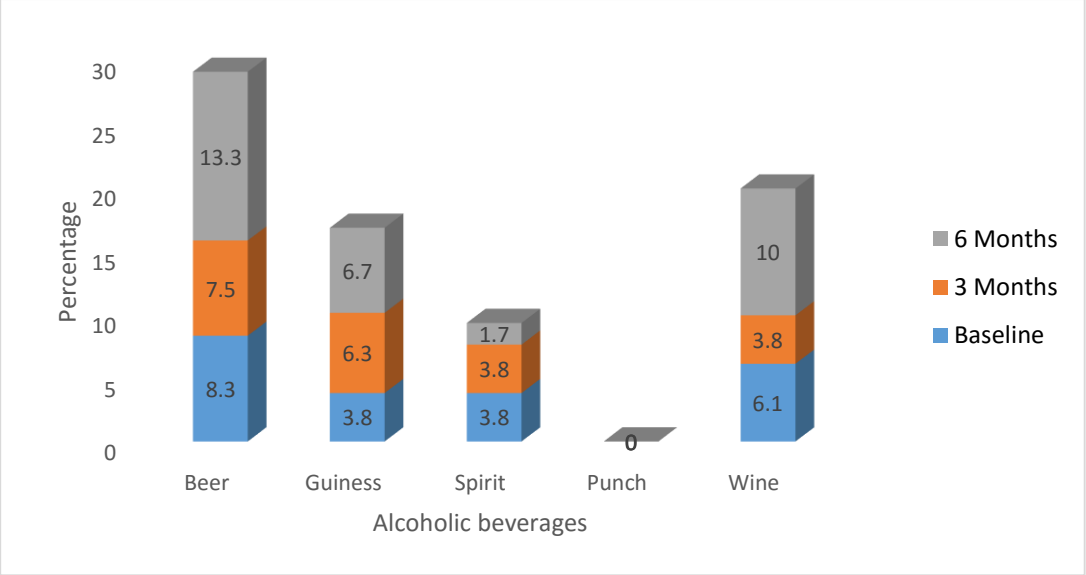


Fig 4.11: Proportion of patients who consumed alcoholic beverages at baseline, 3 months and 6 months

4.8 Results for specific objective 2; To compare the mean blood pressure, anthropometry and body composition of patients at baseline to that at 3 and 6 months.

Table 4.8a below shows the variations in blood pressure, anthropometric and body composition indicators of all patients from baseline through to 6 months. Body mass index and visceral fat were the only indicators that reduced significantly from baseline to 6 months. No significant changes in blood pressure occurred.

Table 4.8a: Variation in anthropometry, body composition and blood pressure of patients, at baseline compared to 3 and 6 months follow-up (Mean ± SD)

Variable	Baseline	3 Months	6 Months	P value
		***n* = 60**		
Body mass index (kgm^{-2})	32.3 ± 6.7	31.7 ± 6.5	31.5 ± 6.5	P < 0.001*
Waist circumference (cm)	103.9 ± 13.0	102.9 ± 12.6	102.9 ± 13.4	P = 0.076
Waist-hip ratio	0.92 ± 0.06	0.91 ± 0.07	0.92 ± 0.06	P = 0.198
Percentage body fat (%)	38.7 ± 9.5	38.0 ± 9.5	37.7 ± 9.2	P= 0.059
Visceral fat	11.2 ± 3.2	10.7 ± 3.1	10.4 ± 3.1	P = 0.003*
Muscle mass	47.4 ± 8.5	47.8 ± 7.8	47.8 ± 7.9	P = 0.678
Total body water	43.4 ± 6.2	43.9 ± 6.2	44.0 ± 6.0	P = 0.168
SBP (mmHg)	137 ± 20	138 ± 20	137 ± 26	P = 0.867
DBP (mmHg)	88 ± 14	89 ± 14	88 ±15	P = 0.925

*; Significant at $P \leq 0.05$; Greenhouse Geisser Test of within-Subject Effect, (repeated measures ANOVA). SBP = Systolic blood pressure. DBP = Diastolic blood pressure

Table 4.8b below illustrates the changes in anthropometry and body composition between the genders. From this table, it is observed that the significant changes in BMI and visceral fat that were shown in Table 4.8a did occur only among females while that of males did not change markedly. Also percentage body fat and total body water improved significantly among females but not in males. Males recorded no significant changes in all the anthropometry and body composition indicators.

Table 4.8b: Gender variations in anthropometry and body composition indicators at baseline compared to 3 and 6 months. (Mean ± SD).

Variables	Baseline	3 Months	6 Months	P value
Males (*n* =15)				
Body mass index (kgm^{-2})	26.8 ± 4.7	26.8 ± 4.6	26.8 ± 4.7	P = 0.920
Waist circumference (cm)	93.7 ± 12.6	93.7 ± 12.3	93.4 ± 11.6	P = 0.915
Waist-hip ratio	0.92 ± 0.06	0.92 ± 0.07	0.92 ± 0.06	P = 0.920
Percentage body fat (%)	25.6 ± 6.8	25.2 ± 8.1	25.9 ± 8.2	P = 0.902
Visceral fat	11.6 ± 4.2	10.9 ± 3.5	10.3 ± 3.9	P = 0.181
Muscle mass	53.0 ± 7.9	53.2 ± 8.1	52.9 ± 8.1	P = 0.931
Total body water	52.6 ± 4.3	52.5 ± 5.2	52.2 ± 5.3	P = 0.933
SBP (mmHg)	144 ± 22	143 ± 21	145 ± 34	P = 0.778
DBP (mmHg)	92 ± 14	90 ± 9	95 ± 18	P = 0.304
Females (*n* = 45)				
Body mass index (kgm^{-2})	34.1 ± 6.3	33.3 ± 6.2	33.0 ± 6.4	P < 0.001*
Waist circumference (cm)	107.4 ± 11.3	105.9 ± 11.2	106.0 ± 12.6	P = 0.07
Waist-hip ratio	0.92 ± 0.06	0.91 ± 0.07	0.92 ± 0.06	P = 0.076
Percentage body fat (%)	43.1 ± 5.1	42.3 ± 5.1	41.7 ± 5.4	P = 0.001*
Visceral fat	11.0 ± 2.9	10.6 ± 2.9	10.4 ± 2.9	P = 0.001*
Muscle mass	45.5 ± 8.0	46.0 ± 6.9	46.I ±7.2	P = 0.607
Total body water	40.4 ± 2.9	41.0 ± 3.0	41.3 ± 3.0	P = 0.005*
SBP (mmHg)	136 ±19	137 ± 19	135 ± 22	P = 0.632
DBP (mmHg)	87 ± 14	88 ± 15	86 ± 14	P = 0.461

*; Significant at P ≤ 0.05; Greenhouse Geisser Test of within-Subject Effect, (repeated measures ANOVA).

Changes in BMI and visceral fat were all significant in both the short term (between baseline and 3 months) and in the long term (between baseline and 6 months) (Table 4.8c).

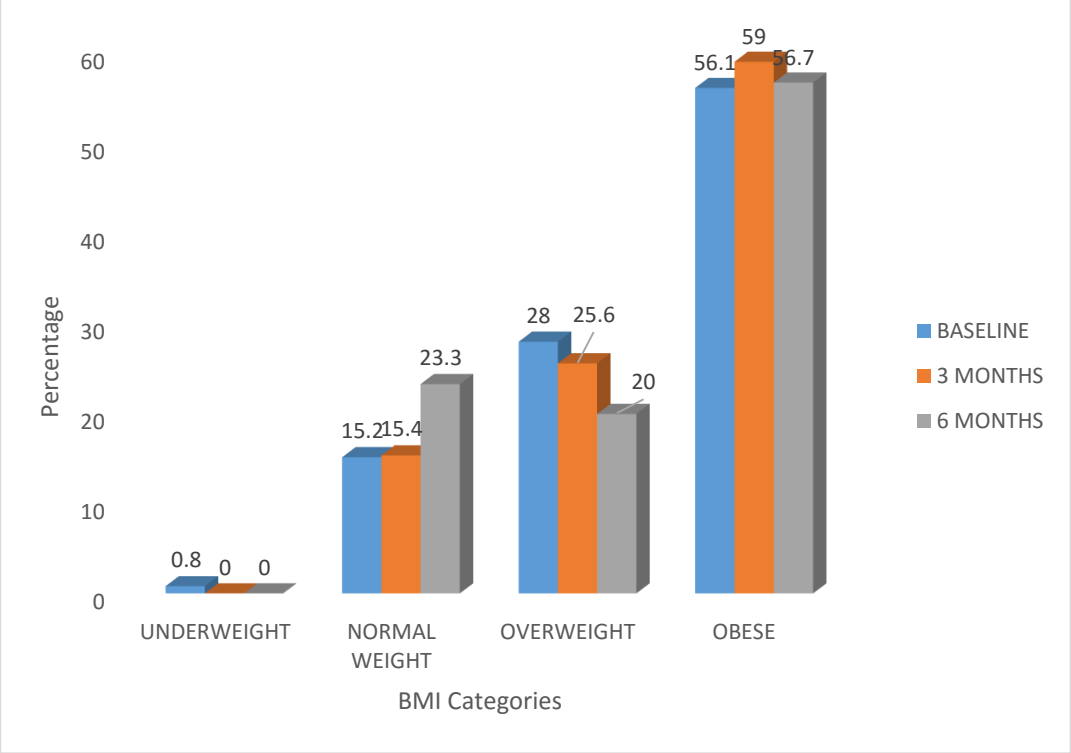
Table 4.8c: Post hoc analysis showing the point at which significant variations in mean BMI, visceral fat and pulse occurred (p values).

Variable	Baseline vs 3 Months	Baseline vs 6 Months
Body mass index	P < 0.001*	P < 0.001*
Visceral fat	P = 0.005*	P = 0.004*

*; Significant at P ≤ 0.05; Dunnett Post Hoc test (One-way ANOVA)

4.8.1 Obesity status of patients at baseline, 3 months and 6 months

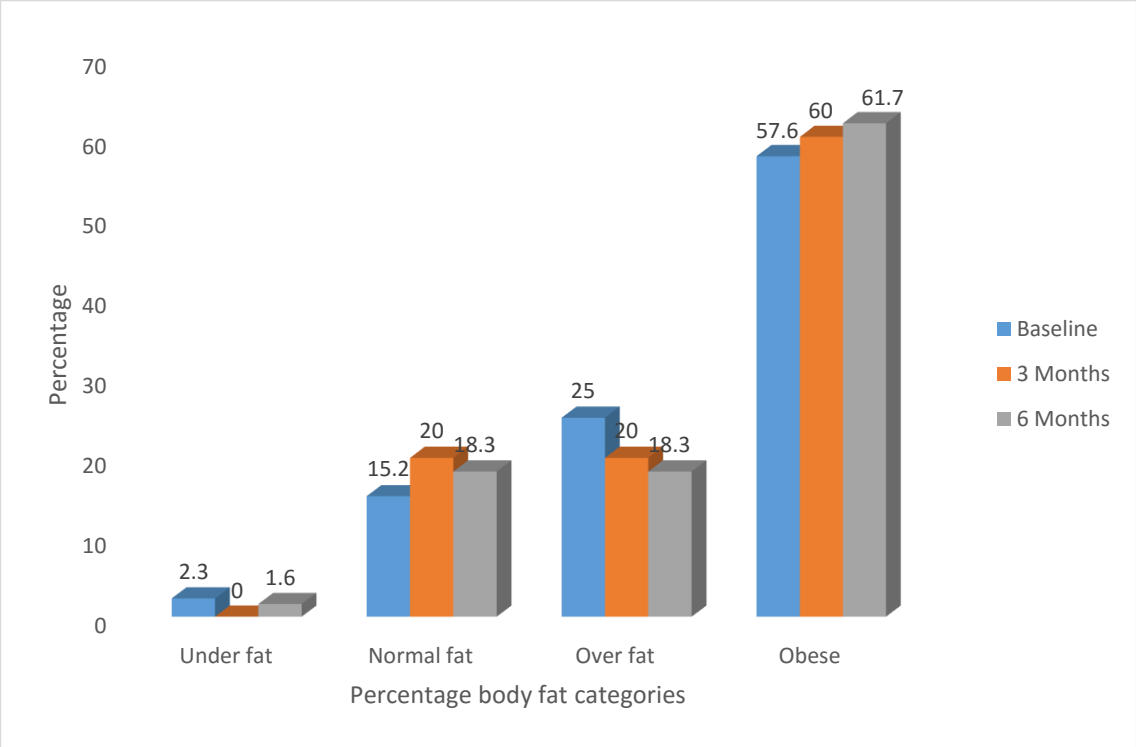
In Fig 4.8.1 below, the proportion of underweight, normal weight, overweight and obese patients at baseline, 3 and 6 months are shown. The proportion of normal weight patients remained unchanged between baseline and 3 months but increased at 6 months. That of overweight individuals reduced from 28% at baseline to 20% after 6 months. Proportion of obese patients remained unchanged at 6 months compared to baseline, Fig 4.8.1.



P = 0.705: Pearson's Chi square, significant at p ≤ 0.05.

Fig 4.8.1: Proportion of patients in the different BMI categories at baseline, 3 and 6 months

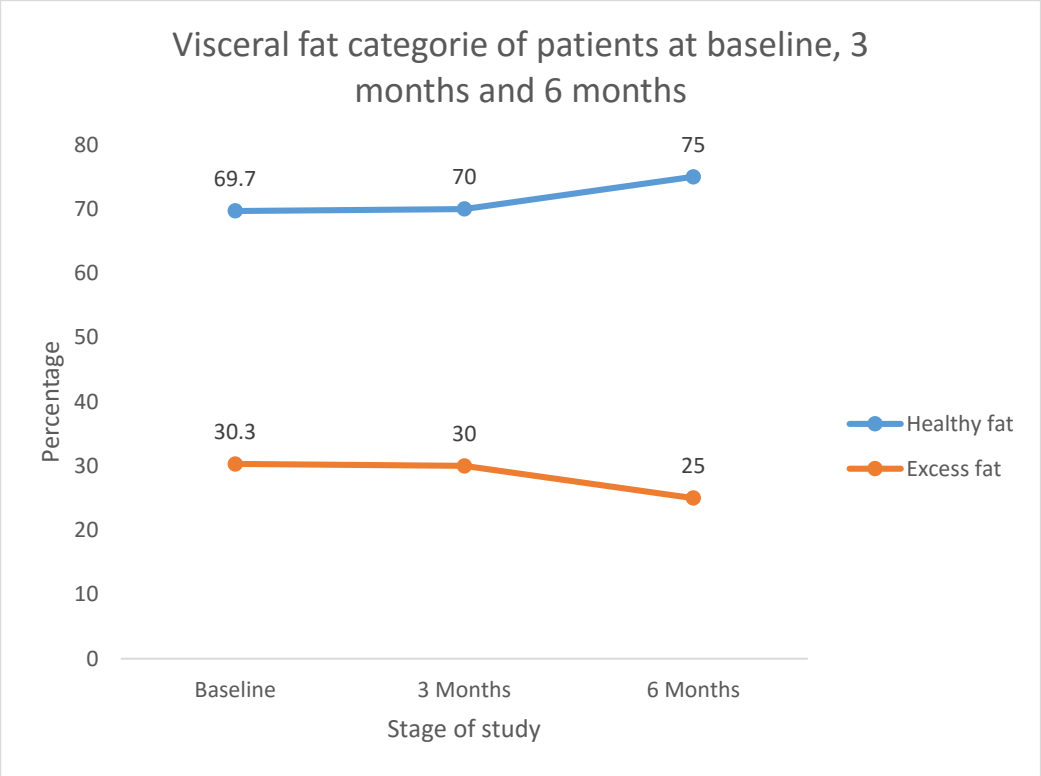
In Fig 4.8.2 below, the proportion of patients in the different body fat categories are shown. The proportion of overfat patients progressively decreased from baseline to 6 months while those who were obese progressively increased during the same period. Those who had healthy percentage body fat at baseline increased after 3 months but not statistically significant.



P = 0.721

Fig 4.8.2: The proportion of patients within the various percentage body fat categories at baseline, 3 months and 6 months

In Fig 4.8.3 below, the visceral fat category of patients is shown. A 5% decline in the proportion of patients with excess visceral fat occurred between baseline and 6 months with a corresponding 5% increase in the proportion of patients with healthy visceral fat within the same period.



P = 0.736

Fig 4.8.3: The proportion of patients within the various visceral fat categories at baseline, 3 months and 6 months.

4.9. Results for specific objective 3; To compare blood biochemistry of patients at baseline to that at 3 and 6 months.

The differences in patients' blood biochemistry from baseline compared to 3 and 6 months is illustrated in Table 4.9 below. Fasting plasma glucose (FPG) and 2-hour plasma glucose decreased significantly during the period. This was evident among both males and females for FPG and females only for 2-hour blood glucose. HDL cholesterol also increased significantly after 6 months. Among females, triglyceride levels significantly increased after 6 months, Table 4.9.

Table 4.9.1 Variation in blood biochemistry of patients, at baseline compared to 3 and 6 months follow-up (Mean ± SD)

Variable	Baseline	3 Months	6 Months	P value
Total (*n* = 60)				
Fasting plasma glucose(mmol/L))	8.3 ± 3.9	6.6 ± 4.4	6.4 ± 3.0	P = 0.001*
2-hour plasma glucose (mmol/L)	14.9 ± 7.8	12.9 ± 6.7	12.0 ± 7.4	P = 0.001*
HDL Chol (mmol/L)	1.2 ± 0.3	1.3 ± 0.3	1.4 ± 0.5	P = 0.021*
LDL Chol (mmol/L)	3.5 ± 1.2	3.2 ± 1.1	3.3 ± 1.1	P = 0.257
Triglycerides (mmol/L)	1.2 ± 0.6	1.1 ± 0.4	1.2 ± 0.5	P = 0.320
Total cholesterol (mmol/L)	5.3 ± 1.4	5.1 ± 1.2	5.3 ± 1.3	P = 0.243
C-reactive protein (mg/L)	5.1 ± 6.3	4.7 ± 6.1	5.9 ± 8.8	P = 0.366
(Median)	*3.1*	*2.9*	*2.0*	*P = 0.129*[a]
Males (*n* = 15)				
Fasting plasma glucose (mmol/L)	7.6 ± 3.5	5.3 ± 1.4	5.7 ± 2.0	P = 0.018*
2-hour plasma glucose (mmol/L)	13.7 ± 6.7	10.4 ± 5.2	9.5 ± 6.4	P = 0.059
HDLcholesterol (mmol/L)	1.2 ± 0.4	1.3 ± 0.5	1.7 ± 0.8	P = 0.119
LDLcholesterol (mmol/L)	3.0 ± 1.1	2.8 ± 1.0	3.1 ±1.1	P = 0.518
Triglycerides (mmol/L)	1.1 ± 0.9	1.0 ± 0.4	1.0 ± 0.4	P = 0.681
Total cholesterol (mmol/L)	4.7 ± 1.4	4.6 ± 1.0	5.2 ± 1.3	P = 0.178
C-reactive protein (mg/ L)	5.5 ± 7.5	2.8 ± 6.2	4.2 ± 8.5	P = 0.325
(Median)	*2.2*	*1.3*	*0.8*	*P = 0.159*
Females (*n* = 45)				
Fasting plasma glucose (mmol/L))	8.5 ± 4.1	7.2 ± 5.0	6.7 ± 3.3	P = 0.005*
2-hour plasma glucose (mmol/L)	15.2 ± 8.2	13.7 ± 7.2	12.8 ±7.6	P = 0.011*
HDL cholesterol (mmol/L)	1.2 ± 0.3	1.3 ± 0.3	1.3 ± 0.2	P = 0.112
LDL cholesterol (mmol/L)	3.7 ± 1.2	3.4 ± 1.1	3.4 ± 1.1	P = 0.222
Triglycerides (mmol/L)	1.2 ± 0.6	1.1 ± 0.4	1.3 ± 0.5	P = 0.045*
Total cholesterol (mmol/L)	5.5 ± 1.5	5.2 ± 1.3	5.3 ± 1.3	P = 0.301
C-reactive protein (mg/ L)	5.0 ± 5.9	5.3 ± 6.0	6.6 ± 8.9	P = 0.294
(Median)	*3.3*	*3.7*	*2.8*	*P = 0.466*

*; Significant at P ≤ 0.05; Greenhouse Geisser Test of within-Subject Effect, (repeated measures ANOVA).
[a]: Significant at P ≤ 0.05; Non parametric test for related samples, Friedman's two-way analysis of variance.

Post hoc analysis showed that the change in fasting plasma glucose occurred in the short term (between baseline and 3 months) and was sustained in the long term (between baseline and 6 months). Significant change in HDL occurred after 6 months.

Table 4.9.2: Post hoc analysis showing significant variation in blood biochemistry at baseline compared to 3 months and 6 months (p values).

Variable	Baseline vs 3 Months	Baseline vs 6 Months
Fasting plasma glucose (mmol/L))	P = 0.044	P = 0.017
2-hour plasma glucose (mmol/L)	P = 0.258	P = 0.062
HDL Chol (mmol/L)	P = 0.911	P = 0.045

*; Significant at $P \leq 0.05$; Dunnett Post Hoc test (One-way ANOVA).

4.10 Results of specific objective 4: To determine changes in patients' physical activity, alcohol intake and smoking habits after routine dietary and lifestyle intervention.

The physical activity habits of patients at baseline was compared to that at 3 and 6 months and is shown in Table 4.10.1 below. A significant increase in the proportion of patients who engaged in moderate activity occurred between baseline and 3 months. Although the proportion decreased between 3 and 6 months, the difference between baseline and 6 months was still significant. There was, however, no significant changes in the frequency (the number of physically active days) as well as the time spent on moderate physical activity.

Table 4.10.1: Physical activity habits of patients at baseline compared to 3 months and 6 months (p values).

Physical activity habits	**Baseline N = 132**	**3 months N = 80**	**6 months N = 60**	**P value**
Moderate activities done continuously for at least 10 minutes				
Yes	40 (30.3)	61 (76.3)	37 (61.7)	P < 0.001*
No	92 (69.7)	19 (23.7)	23 (38.3)	
Total	132 (100.0)	80 (100.0)	60 (100.0)	
Number of days moderate activities are done in a week	22 (16.7)	33 (41.3)	16 (26.7)	P = 0.478
1-4 days in a week	18 (13.6)	28 (35.0)	21 (35.0)	
≥ 5 days in a week	40 (30.3)	61 (76.3)	37 (61.7)	
Total				
Time spent on moderate activities				
< 30 mins	16 (12.1)	17 (21.3)	13 (21.7)	P = 0.433
≥ 30 mins	24 (18.2)	44 (55.0)	24 (40.0)	
Total	40 (30.3)	61 (76.3)	37 (61.7)	

*; Significant at P ≤ 0.05: Pearson's Chi Square.

In Table 4.10.2 below, the proportion of males who engaged in moderate physical activity is compared to females who did likewise. Significantly more males engaged in moderate physical activity at baseline and at 6 months compared to females.

Table 4.10.2: Comparison of proportion of males and females who engaged in moderate physical activity at baseline 3 and 6 months follow-up. Frequency (%)

Variable	Yes	No	Total	P-value
Moderate-intensity sports, fitness or recreational (leisure) activities continuously for at least 10 minutes				
Baseline (*n* = 132)				
Males (*n* = 35)	16 (45.7)	19 (54.3)	35 (100.0)	P = 0.021*
Females (*n* = 97)	24 (24.7)	73 (75.3)	97 (100.0)	
3 Months (*n* = 80)				
Males (*n* = 21)	17 (81.0)	4 (19.0)	21 (100.0)	P = 0.555
Females (*n* = 59)	44 (74.6)	15 (25.4)	59 (100.0)	
6 Months (*n* = 60)				
Males (*n* = 15)	13 (86.7)	2 (13.3)	15 (100.00)	P = 0.021*
Females (*n* = 45)	24 (53.3)	21 (46.7)	45 (100.00)	

*; Significant at P ≤ 0.05: Pearson's Chi Square.

Table 4.10.3 below compares the alcohol intake habits of patients at baseline to that at 3 and 6 months follow-up. No significant changes in the frequency and amount of alcohol consumed occurred from baseline to 6 months.

Table 4.10.3: Alcohol intake habits of patients at baseline compared to 3 months and 6 months (p values).

Alcohol intake	Baseline	3 Months	6 Months	P - value
Frequency of alcohol intake within the past 3 months				
Daily	4 (3.0)	-	-	P = 0.571
1-6 days per week	5 (3.8)	1 (1.35)	-	
1-3 days per month	6 (4.5)	3 (3.8)	4 (6.7)	
Less than once a month	32 (24.2)	21 (26.3)	12 (20.0)	
Total	47 (35.6)	25 (31.3)	16 (26.7)	
Alcohol consumed within the past 7 days				
Yes	18 (13.6)	16 (20.0)	13 (21.7)	P = 0.294
No	114 (86.4)	64 (80.0)	47 (78.3)	
Total	132 (100.0)	80 (100.0)	60 (100.0)	
Number of drinks consumed on a typical day				
< 2 drinks	18 (13.6)	12 (15.0)	9 (15.0)	P = 0.631
≥ 2 drinks	13 (9.8)	5 (6.3)	7 (11.7)	
Total	31 (23.4)	17 (21.3)	16 (26.7)	

*; Significant at P ≤ 0.05: Pearson's Chi Square.

Only 2 patients recruited into the follow-up study smoked at baseline. They were, however, lost to follow-up. No patients smoked at 3and 6 months follow-up.

4.11. Results for specific objective 5; To identify the predictors of the variability in all variables that underwent significant changes from baseline to 6 months.

Dietary fibre intake, dietary carbohydrate intake, body mass index, visceral fat, fasting plasma glucose, 2-hour plasma glucose, high density lipoprotein cholesterol and engaging in moderate physical activity, were significantly changed from baseline to 6 months in this study. They were defined as the dependent variables in a multiple linear or binary logistic regression model. All other variables assessed in this study were considered as their independent predictor variables. Regression analysis were also performed for C-reactive protein because it is established as an independent predictor of CVD risk, yet little is known about its predictors among Ghanaians.

4.11.1: Identification of the predictors of patients' dietary fibre intake

In Tables 4.11.1a and 4.11.1b below, results of multiple linear regression analysis to identify the predictors of patients' dietary fibre intake are shown. A significant equation was found, $F\ (2,\ 217) = 139.48$, $p < 0.001$, $R^2 = 0.562$ based on mean daily carbohydrate intake. Carbohydrate intake significantly accounted for 56.2% of the variability in dietary fibre intake. The association was stronger ($R^2 = 58.3\%$) after correcting for age and gender.

Table 4.11.1a: Model summary for predictors of dietary fibre intake

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.750[a]	.562	.558	6.39244

Table 4.11.1b: Significance of linear regression equation for dietary fibre

Model	Sum of Squares	df	Mean Square	F	Sig.
Regression	11398.868	2	5699.434	139.476	.000
Residual	8867.322	217	40.863		
Total	20266.190	219			

*: Significant at $p \leq 0.05$: ANOVA

In Table 4.11.1c below, patients' estimated dietary fibre intake was equal to 0.530 + 0.069 (daily carbohydrate intake in g). Patients' dietary fibre intake increased by 0.069 g for each 1g increase in carbohydrate intake.

Table 4.11.1c: Coefficient of predicting variables for dietary fibre intake

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B	
	B	Std. Error	Beta			Lower Bound	Upper Bound
(Constant)	.530	1.335		.397	.692	-2.101	3.160
Daily CHO intake	.069	.004	.728	15.890	.000	.060	.077

*; Significant at $p \leq 0.05$: multiple linear regression.

4.11.2; Identification of the predictors of patients' dietary carbohydrate intake.

In Tables 4.11.2a and 4.11.2b below, results of multiple linear regression analysis to identify the predicters of patients' dietary carbohydrate intake are shown. A significant equation was found, $F(5, 48) = 179.83$, $p < 0.001$, $R^2 = 0.95$ based on dietary protein intake, total fat intake, daily energy intake and the number of alcoholic drinks consumed daily. As a group, they significantly accounted for 94.9% of the variability in patients' dietary carbohydrate intake, Table 4.11.2a.

Table 4.11.2a: Model summary for dietary carbohydrate intake

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.974	.949	.944	21.24454

Table 4.11.2b: Significance of linear regression equation for carbohydrates

Model	Sum of Squares	df	Mean Square	F	Sig.
Regression	405819.665	5	81163.933	179.833	.000*
Residual	21663.857	48	451.330		
Total	427483.521	53			

*: Significant at $p \leq 0.05$: ANOVA

In Table 4.11.2c below, patients' estimated dietary carbohydrate intake was equal to 27.594 - 1.985 (total dietary fat intake in g) – 0.997 (dietary protein intake in g) + 0.228 (total daily energy intake in kcal) – 7.951 (daily alcohol intake). The patients estimated carbohydrate intake decreased by 1.985g for every 1 g increase in total dietary fat intake. It decreased by 0.997 g for every 1.0 g increase in protein intake, increased by 0.228 g for every 1 kcal increase in energy, but decreased by 7.951g for every 1 drink increase in alcohol intake. The unique contribution of each variable to the variance observed was statistically significant. The significant associations were stronger $R^2 = 95\%$, after correcting for age and gender.

Table 4.11.2c: Coefficients of predicting variables for dietary carbohydrate intake

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B	
	B	Std. Error	Beta			Lower Bound	Upper Bound
(Constant)	27.594	11.701		2.358	.022	4.066	51.121
Daily total fat intake	-1.985	.163	-.577	-12.176	.000	-2.312	-1.657
Daily protein intake	-.997	.261	-.273	-3.817	.000	-1.523	-.472
Daily energy intake	.228	.012	1.488	19.641	.000	.204	.251
Average number of drinks daily	-7.951	2.917	-.090	-2.726	.009	-13.817	-2.086

*; Significant at $p \leq 0.05$: multiple linear regression.

4.11.3: Identification of the predictors of the variability in patients' body mass index

In Tables 4.11.3a and 4.11.3b below, results of multiple linear regression analysis to identify the variables that predicted patients' body mass index are shown. A significant equation was found, F (5, 214) = 352.61, $p < 0.001$, $R^2 = 0.89$ based on patients' visceral fat, percentage body fat, gender and age. Together, they significantly accounted for 89.2% of the variability in patients' body mass index,

Table 4.11.3a: Model summary for patients' body mass index

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.944	.892	.889	2.1558

Table 4.11.3b: Significance of linear regression equation for body mass index

Model	Sum of Squares	df	Mean Square	F	Sig.
Regression	8193.750	5	1638.750	352.607	.000*
Residual	994.569	214	4.648		
Total	9188.320	219			

*: Significant at $p \leq 0.05$: ANOVA

In Table 4.11.3c below, patients' estimated body mass index was equal to 15.36 + 1.43 (visceral fat) + 0.16 (percentage body fat in %) + 3.71 (gender) - 0.24 (age in years), where gender was coded 1= males and 2 = females. The patients estimated body mass index increased by 1.43kgm^{-2} for every unit increase in visceral fat, increased by 0.16 kgm^{-2} for every percentage increase in total body fat and decreased by 0.24 kgm^{-2} for every one-year increase in age. Females had 3.71 kgm^{-2} more BMI than males.

Table 4.11.3c: Coefficients of predicting variables for patients' body mass index

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B	
	B	Std. Error	Beta			Lower Bound	Upper Bound
(Constant)	15.360	1.214		12.650	.000*	12.967	17.753
Visceral fat	1.434	.077	.692	18.571	.000*	1.281	1.586
Percentage body fat	.158	.041	.232	3.833	.000*	.077	.240
Gender	3.705	.775	.251	4.781	.000*	2.177	5.232
Age	-.235	.019	-.323	-12.632	.000*	-.272	-.198

*; Significant at $p \leq 0.05$: multiple linear regression.

4.11.4: Identification of the predictors of the variability in patients' visceral fat

In Tables 4.11.4a and 4.11.4b below, a multiple linear regression analysis to identify the variables that predicted patients' visceral fat is shown. A significant equation was found, ($F(4, 215) = 93.8$, $p < 0.001$), $R^2 = 0.636$ based on patients' percentage body fat, gender and age. Together, they significantly accounted for 63.6% of the variability in patients' visceral fat, Table 4.11.4a and 4.11.4b.

Table 4.11.4a: Model summary for patients' visceral fat

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.797	.636	.629	1.905

Table 4.11.4b: Significance of linear regression equation for visceral fat

Model	Sum of Squares	df	Mean Square	F	Sig.
Regression	1361.109	4	340.277	93.812	.000
Residual	779.850	215	3.627		
Total	2140.959	219			

*: Significant at $p \leq 0.05$: ANOVA

In Table 4.11.4c below, patients' estimated visceral fat was equal to 2.212 - 7.157 (gender) + 0.105 (age in years) + 0.422 (percentage body fat), where gender was coded 1= males and 2 = females. The patients estimated visceral fat increased by 0.422 for every percentage increase in body fat. Males had 7.157 higher visceral fat than females. Visceral fat further increased by 0.105 for every year increase in age. Percentage body fat, age and gender were all significant predictors of visceral fat, Table 4.11.4c.

Table 4.11.4c: Coefficients of predicting variables for patients' visceral fat

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B	
	B	Std. Error	Beta			Lower Bound	Upper Bound
(Constant)	2.212	1.062		2.082	.038	.118	4.305
Gender	-7.157	.480	-1.005	-14.911	.000	-8.103	-6.211
Age	.105	.015	.299	7.092	.000	.076	.134
Percentage body fat	.422	.022	1.282	18.793	.000	.377	.466

*; Significant at $p \leq 0.05$: multiple linear regression

4.11.5; Identification of the predictors of the variability in patients' fasting plasma glucose

Tables 4.11.5a and 4.11.5b below show results of multiple linear regression analysis to identify the predicters of fasting plasma glucose among study participants. A significant equation was found, ($F(3, 216) = 11.67$, $p < 0.001$), $R^2 = 0.139$ based on patients' waist-hip circumference ratio and gender. Together, they significantly accounted for 13.9% of the variability in patients' fasting plasma glucose, Table 4.11.5a and 4.10.5b.

Table 4.11.5a: Model summary for patients' fasting plasma glucose

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.373	.139	.127	3.8580136

Table 4.11.5b: Significance of linear regression equation for fasting plasma glucose

Model	Sum of Squares	df	Mean Square	F	Sig.
Regression	520.989	3	173.663	11.668	.000
Residual	3215.002	216	14.884		
Total	3735.992	219			

*: Significant at $p \leq 0.05$: ANOVA

In Table 4.11.5c below, patients' estimated fasting plasma glucose was equal to -12.260 + 20.294 (waist-hip circumference ratio) + 1.423 (gender). Gender was coded as Males = 1 and Female = 2. Fasting plasma glucose increased by 20.294 mmol/L for every (0.1) increase in waist-hip circumference ratio. Females had 1.423 mmol/L higher fasting plasma glucose than males. Waist-hip circumference ratio and gender were significant predictors of patients' fasting plasma glucose, Table 4.11.5c.

Table 4.11.5c: Coefficients of predicting variables for patients' fasting plasma glucose

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B	
	B	Std. Error	Beta			Lower Bound	Upper Bound
(Constant)	-12.260	4.343		-2.823	.005	-20.821	-3.700
Waist – hip circumference ratio	20.294	4.527	.283	4.483	.000	11.371	29.218
Gender	1.423	.594	.151	2.396	.017	.252	2.593

*; Significant at $p \leq 0.05$: multiple linear regression

4.11.6; Identification of the predictors of the variability in patients' 2-hour plasma glucose

In Table 4.11.6a and 4.11.6b below, results of multiple linear regression analysis to identify the predictors of patients' 2-hour plasma glucose yielded a significant equation, ($F(3, 216) = 95.78$, $p < 0.001$), $R^2 = 0.57$, based on a fasting plasma glucose and waist hip

circumference ratio. Together, they significantly accounted for 57% of the variability in patients' 2- hour plasma glucose, Table 4.11.6a and 4.11.6b.

Table 4.10.6a: Model summary for patients' 2-hour plasma glucose

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.756[a]	.571	.565	4.7956076

Table 4.11.6b: Significance of linear regression equation for 2-hour plasma glucose

Model	Sum of Squares	df	Mean Square	F	Sig.
Regression	6608.135	3	2202.712	95.779	.000
Residual	4967.536	216	22.998		
Total	11575.672	219			

*: Significant at $p \leq 0.05$: ANOVA

In Table 4.11.6c below, patients' estimated 2-hour plasma glucose was equal to - 13.674 + 1.229 (fasting plasma glucose mmol/L) +19.131 (waist hip circumference ratio). Two-hour plasma glucose increased by 1.229 mmol/L for every 1 mmol/L increase in fasting plasma glucose and also increased by 19.131 mmol/L, for every 0.1 increase in waist hip circumference ratio. Both variables significantly predicted 2-hour plasma glucose Table 4.11.6c.

Table 4.11.6c: Coefficients of predicting variables for patients' 2-hour plasma glucose

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B	
	B	Std. Error	Beta			Lower Bound	Upper Bound
(Constant)	-13.674	5.342		-2.560	.011	-24.203	-3.144
Mean FPG	1.229	.083	.698	14.719	.000	1.064	1.393
WHR	19.131	5.882	.152	3.252	.001	7.537	30.724

*; Significant at $p \leq 0.05$: multiple linear regression. FPG = fasting plasma glucose, WHR= waist hip circumference ratio

4.11.7; Identification of the predictors of the variability in patients' HDL Cholesterol

Tables 4.11.7a and 4.11.7b below show results of multiple linear regression analysis to identify the predictors of patients' high density lipoprotein (HDL) cholesterol. A significant equation, ($F(5, 214) = 27.00$, $p < 0.001$), $R^2 = 0.39$, based on percentage body fat, serum triglyceride, low density lipoprotein cholesterol and serum total cholesterol was obtained. Together, they significantly accounted for 38.7% of the variability in patients' HDL, Table 4.11.7a and 4.11.7b.

Table 4.11.7a: Model summary for patients' HDL Cholesterol

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.622	.387	.372	.3026054

Table 4.11.7b: Significance of linear regression equation for HDL cholesterol

Model	Sum of Squares	df	Mean Square	F	Sig.
Regression	12.362	5	2.472	27.001	.000*
Residual	19.596	214	.092		
Total	31.958	219			

*: Significant at p ≤ 0.05: ANOVA

In Table 4.11.7c below, the estimated serum HDL cholesterol was equal to 1.14 – 0.01 (percentage body fat in %) – 0.28 (serum triglyceride in mmol/L) + 0.25 (serum total cholesterol in mmol/L) – 0.16 (LDL cholesterol in mmol/L. High density lipoprotein cholesterol reduced by 0.01 mmol/L for every percentage increase in percentage body fat. It further reduced by 0.28 mmol/L for every 1 mmol/L increase in serum triglyceride but increased by 0.25 mmol/L for every 1 mmol/L increase in serum total cholesterol. HDL also reduced by 0.16 mmol/L for every 1 mmol/L increase in LDL.

Table 4.11.7c: Coefficients of predicting variables for patients' serum HDL cholesterol

Model	Unstandardized Coefficients		Standardized Coefficients	T	Sig.	95.0% Confidence Interval for B	
	B	Std. Error	Beta			Lower Bound	Upper Bound
(Constant)	1.14	.131		8.672	.000	.878	1.394
Percent body fat	-.01	.002	-.140	-2.580	.011	-.010	-.001
Serum triglyceride	-.28	.045	-.366	-6.146	.000	-.366	-.188
Serum total cholesterol	.25	.024	.851	10.232	.000	.202	.299
Serum LDL cholesterol	-.16	.029	-.566	-5.459	.000	-.215	-.101

*; Significant at p ≤ 0.05: multiple linear regression. LDL = Low density lipoprotein

4.11.8; Identification of the predictors of the variability in patients' C-reactive protein

Tables 4.11.8a and 4.11.8b below show results of multiple linear regression analysis to identify the predictors of patients' C-reactive protein. A significant linear equation was obtained, ($F(4, 215) = 15.309$, $p < 0.001$), $R^2 = 0.222$, based on the total daily dietary fibre intake, BMI and fasting plasma glucose. They significantly accounted for 22.2% of the variability in patients' C-reactive protein, Table 4.11.8a and 4.11.8b.

Table 4.11.8a: Model summary for patients' C-reactive protein

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.471	.222	.207	.48753

Table 4.11.8b: Significance of linear regression equation for C-reactive protein

Model	Sum of Squares	df	Mean Square	F	Sig.
Regression	14.554	4	3.639	15.309	.000*
Residual	51.101	215	.238		
Total	65.656	219			

*; Significant at $p \leq 0.05$:

In Table 4.11.8c below, the estimated serum C- reactive protein was equal to - 0.656- 0.009 (total daily dietary fibre intake in grams) + 0.034 (body mass index kg/m^2) + 0.021 (fasting plasma glucose in mmol/L). Serum C- reactive protein decreased by 0.009 mg/L for every 1 g increase in daily dietary fibre intake. It increased by 0.034 mg/L for every 1 kg/m^2 increase in BMI and also increased by 0.021 mg/L for every 1 mmol/L increase in fasting plasma glucose.

Table 4.11.8c: Coefficients of predicting variables for patients' C-reactive protein

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B	
	B	Std. Error	Beta			Lower Bound	Upper Bound
(Constant)	-.656	.210		-3.131	.002	-1.069	-.243
Total daily fibre intake (g)	-.009	.004	-.162	-2.616	.010	-.016	-.002
Body mass index	.034	.005	.402	6.647	.000	.024	.044
Fasting plasma glucose	.021	.008	.160	2.608	.010	.005	.037

*; Significant at $p \leq 0.05$: multiple linear regression.

4.11.9: Identification of the determinants of engaging in moderate physical activity among patients

Table 4.11.9 below shows the results of a binary logistic regression to identify the predictors of whether patients engaged in moderate physical activity. A significant model ($p < 0.001$), $R^2 = 13.0$ was calculated based on patients' educational level and body mass index. Together, they accounted for 13% of the variability in patients' engaging in moderate physical activity.

Engaging in moderate physical activity was coded as Yes = 0, No = 1. No patient was underweight. Considering being obese as the point of reference, patients with BMI in the normal range had 0.551 odds of not engaging in moderate physical activity compared to those who were obese, although having normal BMI did not significantly contribute to the regression model ($p = 0.172$). Being overweight, however, contributed significantly to the model ($p = 0.015$). Patients who were overweight had 0.404 odds of not exercising compared to those who were obese. Hence the odds of overweight and normal weight patients not engaging in moderate physical activity was progressively higher (40.4% and 55.1%) respectively, compared to that of the obese patients. The lower the BMI, the higher the odds of not engaging in moderate physical activity.

Table 4.11.9: Variables in the logistic regression model to determine patients' moderate physical activity habits.

Variables	B	S.E.	Wald	df	Sig.	Odds ratio	95% C.I. for odds ratio	
							Lower	Upper
Body mass index			6.551	2	**.038***			
Normal BMI	-.595	.436	1.863	1	.172	.551	.235	1.296
Overweight	-.907	.374	5.880	1	**.015***	**.404**	.194	.840
Educational level			16.926	3	**.001***			
No formal education	2.953	.841	12.327	1	**.000***	**19.160**	3.686	99.604
Completed primary	1.105	.424	6.807	1	**.009***	**3.019**	1.316	6.924
Completed secondary	.409	.446	.843	1	.358	1.506	.629	3.608
Constant	-.815	.362	5.064	1	.024*	.443		

*; Significant at $p \leq 0.05$: Binary logistic regression.

Having no formal education and completing primary education both contributed significantly to the logistic regression model, ($p < 0.001$ and $p = 0.009$) respectively. Considering having tertiary education as the reference point, the odds of patients with no formal education not engaging in moderate physical activity was 19 times that of those with tertiary education. In addition, patients who had completed primary education had 3 times the odds of not engaging in moderate physical as those who had tertiary education. The odds of not engaging in moderate physical activity among patients who had completed secondary education was approximately 2 times that of those with tertiary education, even though having completed secondary education did not contribute significantly to the logistic regression model ($p = 0.358$). The lower the educational level, the higher the odds of not engaging in moderate physical activity, Table 4.11.9.

Table 4.12: Summary of predictive equations determined by linear regression

Equation	R^2 (%)
Dietary fibre intake = 0.530 + 0.069 (a)	56.2
Carbohydrate intake = 27.594 - 1.985 (b) - 0.997 (c) + 0.228 (d) - 7.951 (e)	94.9
Body mass index = 15.36 + 1.43 (f) + 0.16 (g) + 3.71 (h) - 0.24 (i)	89.2
Visceral fat = 2.212 - 7.157 (h) + 0.105 (i) + 0.422 (g)	63.6
Fasting plasma glucose = -12.26 + 20.294 (j) +1.423 (h)	13.9
Two-hour plasma glucose = - 13.674 + 1.229 (k) + 19.131 (j)	57.1
High density lipoprotein cholesterol = 1.14 - 0.01 (g) - 0.28 (l) + 0.25 (m) - 0.16 (n)	38.7
Serum C-reactive protein = - 0.656 - 0.009 (p) + 0.034 (q) + 0.021 (k)	22.2

Where:
a = Mean daily carbohydrate intake (g)
b = Mean daily total fat intake (g)
c = Mean daily protein intake (g)
d = Mean daily energy intake (Kcal)
e = Mean daily alcohol intake (drink)
f = Visceral fat
g = Percentage body fat (%)
h = Gender
I = Age (years)
J = Waist-hip circumference ratio
K = Fasting plasma glucose (mmol/L)
L = Serum triglyceride (mmol/L)
M = Serum total cholesterol (mmol/L)
N = Serum low density lipoprotein cholesterol (mmol/L)

CHAPTER FIVE

5.0 DISCUSSION

5.1. Cross-sectional study

5.1.1 Demography of study participants

A total of 339 patients participated in the cross-sectional study. A significant majority of this number were female compared to male. The Ghana demography and health survey (2014), reports of a progressive decline in the male to female ratio among the Ghanaian population from 1960 to 2010. From 1.022 in 1960 to the current 0.952 (Ghana Statistical Service, 2014). The male to female ratio among the study participants was, however, still lower (0.443) than the national ratio. Evidence generally suggests that females are more likely to attend health facilities than males (Thompson *et al.,* 2016) with the greatest difference occurring between the ages of 16 to 60 years (Wang *et al.,* 2013). They therefore had a better chance of being recruited into the study. In addition, some of the conditions such as type 2 diabetes, which are often referred to the dietitian are reported to be more common among females than males in some populations (Hilawe *et al.,* 2013) and may have contributed to the study population being more female than male.

Their mean age was 49.3 ± 11.9 years. A decline in the age of onset of chronic diseases in Ghana has been suggested, with ages as low as 15 years having conditions such as high blood pressure and peaking between 45 and 49 years (Ghana Statistical Service, 2014). With the majority of the study participants being Akan, Christian and a significant majority being married, the demography of the study participants was a close reflection of the national statistics (Ghana Statistical Service, 2014).

5.1.2 Socioeconomic profile of study participants

Educational attainment nationwide remains low with 19.1% of adult females and 9.4% of adult primary and middle school level while 22% female and 34.5% males have been educated beyond the secondary level (Ghana Statistical Service, 2014). Similar to the national profile, among the participants of this current study, significantly more females than males had no formal education or had been educated up to the primary level. People living in rural areas, older age and wealth differences were some of the factors that explained the variation in educational attainment at the national level (Ghana Statistical Service, 2014). Such gender disparities in educational attainment are common in other countries in Sub Saharan Africa. A combination of social, economic and cultural factors have been cited as contributing factors to these disparities (Dube, 2015).

Similar proportions of male and female patients were employed which contradicts the general gender disparities in employment among Ghanaians, where 60.3% of the employed are males and 39.7 are females (Ghana Satistical Service, 2015). The significantly lower male to female ratio among study participants could have accounted for this contradiction. Significantly more females were, however, unemployed while more males were retired. The majority of participants (47.5%) were also in the less skilled occupations mainly traders, artisans and drivers, a situation that could be attributed to the general low educational attainment.

5.1.3 Commonly referred conditions to the Dietherapy Department

The most common condition for which patients were referred to see the dietitian was hypertension (48.1%). It was followed by type 2 diabetes (44.8%) and dyslipidaemia (38.1%). Referral for obesity formed only 24.8%. The rather low referral for obesity is

alarming, considering that obesity is a major risk factor for most NCDs (WHO, 2008). If care is not taken to address obesity early, it can lead to higher NCD incidence than is currently being recorded, with its attendant health, social and economic challenges. The current prevalence of obesity among adult Ghanaians is 17.1%, making it of public health concern. A possible reason for the low referral for obesity may be because, culturally, Ghanaians prefer large body sizes and associate it with wealth and blessings (Afriyie-Appiah, Otoo and Steiner-Asiedu, 2016). It is therefore not regarded as something to curb or worry about. Additionally, the generally low referral rate of patients to dietitians could have accounted for the low obesity referrals. In 2013, out of a total of 365,387 OPD attendance at the KBTH, only 12,005 attended the Dietherapy Department, constituting 3.3% of total OPD attendance. Physicians need to refer more patients especially obesity related cases, for dietetic management.

These findings are similar to a study carried out in Gauteng, South Africa, which identified hyperlipidaemia, diabetes and obesity as the conditions which doctors would most likely refer to dietitians (Barron, 2006). The main reason why doctors referred patients to dietitians in that study was because patients presented with specific disease states. In another study carried out in Australia, doctors referred when patients had complicated dietary needs (Nicholas *et al.,* 2003). Patients who were considered as able to commit to dietary change and were willing to attend a dietetic consultation were also more likely to be referred (Pomeroy and Cant, 2010). The high current national prevalence of hypertension among adult Ghanaians (urban hypertension prevalence = 32.3%, rural prevalence = 27%) (de-Graft Aikins *et al.*, 2014) could have accounted for it being the most referred condition. Due to the silent nature of the condition, and earlier reports of its rather low prevalence among Ghanaians (Pobee *et al.,* 1977), it probably did not receive enough public health attention

until recent out-patient and mortality reports in some health facilities in Ghana have indicated its devastating impact on health (KBTH, 2013). Diabetes and associated risk factors, mainly obesity, were among the chronic conditions which received relatively early attention in the early days of the double burden of disease (Amoah *et al.*, 2000). At the time when its prevalence began to rise among adult Ghanaians, through the efforts of the National Diabetes Control Programme, diabetes prevention and management was incorporated into the national health policy (Asante *et al.*, 2014). This may have increased the awareness of general practitioners of the management requirements for the condition and the need to involve dietitians. Early association of diabetes with diet, due to its characteristic elevated blood sugar, may also have contributed to doctors referring them to dietitians, even in the early challenging days of the dietetic profession in Ghana (Aryeetey *et al.*, 2014). Additionally, the clear recognition of diet in the management of hypertension, dyslipidaemia and diabetes in various international guidelines (National Cholesterol Education Programme, 2001; International Diabetes Federation, 2012b; National Institutes of Health, 2004) may have contributed to doctors referring these conditions to dietitians.

5.1.4 Patients' dietetic care-seeking behaviour after referral to see a dietitian

The majority of patients delayed between 1 to 3 days from the day they were referred by the doctor to see the dietitian, till when they actually saw the dietitian. Delays ranged from a day to over a month. In all 50.1% of patients delayed in assessing dietetic care. Delays in assessing dietetic care by male patients was comparable to that of females. Numerous reasons explained their delays in seeking dietetic care. Some reasons were patient centred while others related to the hospital service delivery. The main reason for the delay was because the Dietherapy Department had closed at the time they reported on the same day they were referred. Some patients had had to go to the laboratory to conduct some blood

test. Others had earlier gone to the pharmacy to procure the medications which had been prescribed for them by their doctors. At each of these units, they may have had to join long queues to await their turn due to the large numbers of people who visit the hospital daily (KBTH, 2016a). Their visit to the Dietherapy Department was therefore delayed, and by the time they finally got there, it was already past the closing time of 4 pm (KBTH, 2016b). Strike actions by health personnel during the period of data collection also accounted for delays by some patients as they mistakenly thought that all hospital staff were on strike, even though only medical doctors were on strike. Those patients therefore waited until the end of the strike was announced in the media, before they sought dietetic care. A lack of funds to afford dietetic service explained the delay in only a few (1.2%) patients.

Some patients, due to busy work schedules, travel and other family responsibilities (e.g. funerals, caring for other sick relatives and babysitting) could not find the time to see the dietitian. With the majority of patients being in their middle and elderly ages, these family responsibilities were to be expected. Fleetwood, (2015), in a study identified that difficultly in keeping appointments and a lack of referral by medical personnel were the main barriers to accessing prompt nutrition care among HIV patients (Fleetwood, 2015).

5.1.5 Lifestyle of patients

Participants lifestyle including alcohol intake, smoking and physical activity were assessed and are discussed below. Dietary and nutrient intake are however discussed in the follow-up study.

5.1.5.1 Alcohol intake habits of patients

For one year preceding the study, close to 60% of patients had not consumed alcohol. The Ghana Demography and Health Survey, 2008 reported that the percentage of adult Ghanaians who consume alcohol increased with age peaking among the 45 to 49-year groups (Ghana Statistical Service, 2008). Although the mean age of participants in this study was within this age range, most of them did not consume alcohol one year prior to this study. A potential changing trend in alcohol consumption among the Ghanaian population, where more youth now consume alcohol compared to adults is a possible explanation (Osei-Bonsu *et al.*, 2017).

5.1.5.2. Smoking habits of patients

Smoking was very rare among both male and female participants. A significant majority of patients had never smoked. Significantly more men had had some smoking experience in the past compared to women, although most of them had stopped smoking long before the start of this study. This compares with the national smoking statistics where almost all women and 93% of men reported never being involved in tobacco smoking, with only 6 % of men who currently smoked (Ghana Statistical Service, 2008). In this current study, 4.8% of males and 0.4% of females currently smoked. Over the years, cigarette smoking has seen a gradual decline among the adult Ghanaian population (Tagoe and Dake, 2011). Pockets of smoking incidence, however, remain to be addressed, especially among the youth who now indulge in smoking of hard drugs, fuelled mostly by peer influence (Osei-Bonsu *et al.*, 2017).

5.1.5.3 Physical activity habits of patients

Physical activity habits of all patients were very poor. Only 23.3% of all patients did engage in some form of moderate physical activity. Out of that proportion, only 9.4% were physically active for 5 or more days in a week. The WHO recommends a minimum of 30 minutes of moderate physical activity daily for at least 5 days in a week, to reduce the risk of chronic disease and ensure good health (WHO, 2015b). Many non-communicable diseases, such as cardiovascular disease, cancer, diabetes and their risk factors, obesity and hypertension have been associated with physical inactivity (WHO, 2013). In this current study even though only few participants engaged in moderate physical activity, significantly fewer females than males (19.6 % vs 31.7%; p = 0.018) engaged in moderate physical activity. This finding agrees with what is reported in literature, that males at any age are more physically active than females (Azevedo *et al.*, 2007; Hands *et al.*, 2016).

The number of days activities were done as well as the time spent on an activity was, however, comparable between the genders. Lack of time and laziness as well as having an already active career and body aches associated with physical activity were the four main reasons fuelling physical inactivity among patients. Similar findings of lack of time and work/family obligations was reported among a group of Ghanaian women in urban Ghana (Tuakli-Wosornu *et al.*, 2014).

Significant inverse correlations occurred between engaging in moderate physical activity and educational level, employment and occupation, Appendix XII. Patients with at most a primary education were less physically active compared to those with secondary or tertiary education. Those who were employed were also less physically active compared to the unemployed. Also, those involved in less skilled occupations (such as traders, artisans,

security men and drivers) were also less physically active, compared to those in more skilled jobs (such as engineers, accountants, administrators and medical doctors). All the socioeconomic indicators therefore pointed to the fact that patients in the low socioeconomic bracket were less physically active compared to those of the higher SES. Similar as well as contradicting results have been reported by other studies. While in some cases, individuals of high SES could afford leisure physical activity and were therefore more physically active (Talaei *et al.*, 2013; Azevedo *et al.*, 2007), in others, individuals with high SES were too busy and could not find the time to be physically active (Allen *et al.*, 2017). Most of these studies were, however, carried out on apparently healthy populations, while this current study involved patients with chronic diseases. Some patients had indicated that they engaged in physical activity due to doctor's advice, while others wanted to lose weight. Their physical activity habits may therefore have been influenced by their current health state and may therefore not reflect that of the general adult population in Ghana.

A significant linear correlation occurred between the time spent on moderate physical activity and the amount of alcohol intake ($r = 0.454$; $p = 0.012$). The more time spent on moderate physical activity, the higher the intake of alcohol. Anecdotal evidence among Ghanaian adults, especially those in the middle and high socioeconomic brackets, suggest a growing culture of engaging in bouts of drinking following work-outs and leisure sporting activities. This growing culture undermines the health benefits for which regular physical activity was intended and poses a threat to the health of such individuals (Parry *et al.*, 2011).

5.2 The follow-up Study

5.2.1 Patient's attendance at reviews

A smaller fraction of patients absented themselves from reviews between baseline and 3 months compared to between 3 and 6 months. In addition, more patients attended 3 or more reviews between baseline and 3 months compared to between 3 and 6 months. Endevelt and Gesser-Edelsburg, (2014), similarly reported that within a period of two years, 40% to 50% of patients who started nutrition therapy did not return for review after their first visit, even though they were required to do so for free. For chronic conditions, however, it is established that short-term nutrition counselling is of little use compared to continuous nutrition therapy. There is scanty information on how many follow-up visits are required until a patient is discharged. About 70% of chronic disease patients are, however, reported to benefit from continuous nutrition therapy over a long period of time (Pastors *et al.*, 2003; Franz, 2004). A number of factors inform the decision to discharge, these include; meeting the agreed goals set by both patient and dietitian, the patient compliance to dietherapy and the nature of the condition being managed. Nutrition intervention is reported to have the largest statistically significant impact on metabolic control among the various lifestyle interventions (Pastors et al, 2003). The perception of some patients that their visit to the dietitian was of little use has been reported (Spikmans *et al.,* 2003). In their study, the two main reasons explaining why diabetes patients missed their appointment with the dietitian were a health locus of control, (a feeling that their well-being depended on the health worker) and feeling obliged to attend reviews. The solution suggested by the researchers were that, patients should be educated on their role in contributing to their own health. Additionally, their responsibility in ensuring that their appointments with the dietitian were kept, needed to be emphasized (Spikmans *et al.*, 2003). Dietitians at the Dietherapy Department, KBTH, may benefit from educating patients on the importance of attending

reviews and also involve them in setting review dates (Endevelt and Gesser-Edelsburg, 2014).

The counselling style of dietitians is also reported to influence patient's attendance for reviews. Patients were discouraged from continuing with dietetic care and were more likely to miss subsequent reviews when their encounter with the dietitian was short-term and followed an educational and informative approach rather than an individualized therapeutic counselling approach (Endevelt and Gesser-Edelsburg, 2014). Interactions between dietitians and patients at the Dietherapy Department largely follows a personalized therapeutic counselling approach, although, on days when very large numbers of patients attend clinic, or during reviews, a more educational and informative approach may be employed. Review sessions with patients are also often very short and a repetition of set goals, unless in situations where patients had peculiar needs that needed to be addressed. These factors may partly have accounted for the progressive drop in the numbers of patients who attended reviews at 3 and 6 months in this current study.

The absence of private insurance coverage to enable more patients to attend review sessions is also reported to affect attendance at reviews (Alameddine *et al.*, 2013). At the Korle-Bu Teaching Hospital, the National Health Insurance covers the cost of care in most departments but not the Dietherapy Department. First consultations currently cost an amount of GH₵ 48.00 (equivalent to $10.00) while reviews cost GH₵ 32.00 (equivalent to $ 7.00). Patients therefore have to pay this amount every time they attend reviews. Most of these patients additionally visit their general practitioners periodically for routine checks and also need to purchase medications for their chronic conditions as well as carry out routine laboratory checks. All of these have cost implications. In most cases, they may be

nearing their retirements or may already be retired, and may therefore have no regular source of income to pay for all their medical bills. The visit to the dietitian, with its attendant cost implications may therefore be avoided in difficult times, as was observed in this current study.

Long waiting periods between dietetic appointments due to patients being put on long waiting list also causes reduced review attendance rates (Mayre- Chilton and Joyce, 2011). Patients scheduled for dietetic review at the Dietherapy Department are not put on waiting list. After their first appointment, patients are given appointments for review ranging from 2 weeks to one month, based on the severity of their condition, the agreed treatment goals and other factors. When patients report on their review date, they are attended to on the same day without too much waiting time. Other factors which may have accounted for the decline in review attendance are patient familiarity with their treatment regimens as their conditions begin to improve, as well as fatigue from the repetition of information and goals at reviews. On the other hand, dietitians may have given patients longer intervals between reviews due to good progress in their health which therefore required less monitoring.

5.2.2 Changes in patients' energy, nutrient and fibre intake following routine dietary and lifestyle intervention at the KBTH

The changes in patients' energy, nutrient and fibre intake after receiving routine dietetic counselling are hereby discussed. Food frequency questionnaire and repeated 24-hour interviews were used to obtain usual food intake information as well as actual nutrient intakes respectively. Food frequency questionnaire, though subjective, is cost-effective and time-saving; suitable for epidemiological studies (Jee-Seon, Kyungwon and Hyeon 2014). Knowledge of patients' usual food intake over a prolonged period was necessary because

of the established association of dietary intake with risk of NCD development (Appel *et al.*, 2006; Sarrafzadegan *et al.*, 2009). Food frequency questionnaire allowed the assesment of patients routine weekly intakes over the three time lines, baseline, 3 months and 6 months and allowed for comparisn of frequency of intakes at the differenct time lines using chi square analysis. The use of 24-hour recalls were also necessary because it provided additional detailed data such as food preparation methods and ingredients used in mixed dishes. Even though it is also a subjective method of dietary intake assessment, it provided information of actual nutrient intakes which were needed to make the meaningful inferencial ananlysis as the study required.

5.2.2.1 Changes in patients' mean daily energy intake after routine dietary and lifestyle intervention at the KBTH.

The estimation of mean daily energy requirement for each patient was individualized based on patient's particular health situation, physical activity level, treatment goals and other factors. Individualized energy recommended for patients ranged from 1,500 kcal to 1,800 kcal. The allowable daily distribution range (40% to 65%) for energy is expected to be provided from carbohydrates. Total fat is also recommended to contribute an additional 20% to 35% of total daily energy intake and protein between 5% and 35% (US Department of Agriculture and US Department of Health and Human Services, 2010; Ministry of Health, 2010). Mean daily energy intake at baseline was comparable to that at 3 and 6 months with no significant changes. Notwithstanding, the contributions of carbohydrate, fat and protein recorded some changes. Mean daily carbohydrate intake increased significantly resulting in its increased contribution to daily energy from 57.7% at baseline to 67.1% at 6 months. This therefore reduced the contributions of protein and fat from 14.2% and 26.4% to 12.5% and 20.7% at 3 and 6 months respectively, although the amount intake did not change. Mean

daily total fat intake was within recommended ranges throughout the study. A situation that could enhance good health among study participants. A similar situation was observed for mean daily protein intake. Some studies have reported significant improvement in daily energy intakes in the intervention groups following dietary interventions (Lin *et al.*, 2007; Hajira *et al.*, 2017). Their study designs were, however, randomized controlled trials and participants' dietary intakes were closely monitored by researchers. This current study was, however, a clinical audit, adopting a longitudinal study design. Patients self-reported their dietary intakes following the interventions they routinely received in the Dietherapy department. The researchers therefore did not influence the patients' dietary intakes in any way.

5.2.2.2 Changes in patients' carbohydrate intake after receiving dietetic counselling.

A significant increase in carbohydrate intake occurred among all patients. The change occurred in the long term (between baseline and 6 months) but not in the short term (between baseline and 3 months). Also, it occurred among females but not males. Carbohydrate intake pre and post intervention were higher than the RDA of 130g/d (Wolfe *et al.*, 2017; Institute of Medicine of the National Academies, 2002). This recommended amount has been ascertained as sufficient to meet the energy requirement of nearly all (97% to 98%) of the healthy individuals in a population (Institute of Medicine, 1998). In the event of weight loss or the management of a chronic diseases, which was the case for most participants in this current study, carbohydrate restriction to as low as 50g-100g per day, has been reported as safe for a short-term (up to 6 months). Obese patients especially, often require a reduction in daily energy intake by 500 to 600 calories (International Diabetes Federation, 2017). Intakes exceeding the needed amount without a corresponding physical activity to burn off the excess energy leads to overweight and obesity. The observed excess carbohydrate intake

before and after dietetic advice suggest that, excess carbohydrate intake formed part of the usual dietary habit among this group. Carbohydrate intake among people in poorer and developing countries is estimated to provide as much as 90% of the energy in their diets in most cases (Macdonald, 2003). The average Ghanaian diet comprise mostly of tubers and starches as well as fermented cereal products which are high in fibre and low in energy (Ministry of Health, 2010). Evidence is accumulating regarding the westernization of most Ghanaian diets especially among those in urban areas, resulting in the intake of relatively higher energy diets (Galbete *et al.*, 2018; Frank *et al.*, 2014).

The dietetic intervention given to patients recommended the shift from the more refined starches and sweetened beverages which are higher in energy, to the more unrefined, high fibre options such as whole grain and cereals which are relatively low in energy. In addition, more vegetables and fruits intake were encouraged. This, however, did not reduce their carbohydrate intakes. Changes in health behaviour, including dietary intake habits have been suggested to be ineffective when addressed at the individual level or community level. Addressing dietary behaviour change at the national levels, involving policy change, such as higher taxation of unhealthy food options, have been reported as more effective (National Research Council, 2004; Kelly and Barker, 2016).

Demographic and socioeconomic factors of the individual e.g. age and educational levels, in some cases, have facilitated a positive change in dietary behaviour (Franks *et al.*, 2011; Ettner and Grzywacz, 2003). The counselling approach, whether educational or motivational have also been suggested to influence the success of the intervention, with motivational counselling approach showing more success (Lundahl *et al.*, 2013). In this study, carbohydrate intake did not correlate significantly with any demographic or

socioeconomic indicator. Instead, the variables that predicted carbohydrate intake among patients were their total dietary fat, protein, alcohol and total daily energy intake. A reduction in the intakes of total fat, protein and alcohol resulted in an increase in carbohydrate intake while the reverse occurred for total daily energy intake. Together, they predicted 94.5% of the variability in carbohydrate intake.

A possible explanation to the observed increase in carbohydrate intake at 3 and 6 months may be that, instead of patients exchanging or replacing the high calorie starches in their diet with high fibre starches, they maintained their regular intakes and additionally included high fibre starches as well as fruits in their diet. In so doing, they exceeded their healthy carbohydrate limits. This is deduced from the fact that the frequency of intake of the commonly consumed starches, such as plain rice, boiled yam, boiled plantain and fufu, did not change significantly over the study period, The frequency of intake of whole grains, such as oats, wheat and *tombrown* (a mixed whole cereal porridge) in a week, however, increased, although not significantly. The proportion of patients who never consumed these cereals progressively decreased at 3 and 6 months compared to baseline, with corresponding increases in the proportions whose intakes were low or moderate at 3 and 6 months. It was therefore evident that patients were making the effort to include whole grains and fruits in their usual diet. Banku and *kenkey*, (both prepared from fermented whole maize dough), remained the most frequently consumed whole grain food products. Plain rice, followed by boiled plantain, boiled yam and fufu were the most frequently consumed starches.

High intake of carbohydrates is associated with atherogenic dyslipidaemia, characterized by elevated triglycerides, reduced HDL concentrations and elevated small density lipoprotein cholesterol. These endanger cardio metabolic health and increase cardiovascular diseases as

well as total mortality (Siri-Tarino *et al.*, 2015). Elevated triglyceride and reduced HDL are part of the criterial for the diagnosis of the metabolic syndrome (National Cholesterol Education Programme, 2001). Excess intake of carbohydrate in the diet is converted into fat and stored in adipose tissue which can result in obesity (WHO, 2008b). Obesity, especially visceral obesity stimulates pro-inflammatory mechanisms leading to insulin resistance, diabetes as well as hypertension (Narasimhan *et al.,* 2016; Mancini *et al.,* 2015). Increased carbohydrate intake, especially from simple sugars, increases norepinephrine turnover in peripheral tissues, which is an indirect measure of sympathetic nervous system activity. Activation of sympathetic nervous system leads to an increased blood pressure (Jiang *et al.*, 2016; Landsberg *et al.,* 2013). Continuous excess carbohydrate intake among study participants therefore poses a great threat to their long-term health especially if it is without a commensurate increase in physical activity. The danger may be more among females because their carbohydrate intake increased significantly in the long term but not the males.

5.2.2.3 Changes in patients' dietary fibre intake after receiving dietetic counselling.

The mean daily dietary fibre intake at baseline was 16.6 ± 10.8 g which significantly increased to 20.8 ± 7.9 g at 6 months (CI:95%, P = 0.025). Similarly, daily dietary fibre intake of 17g was reported among American adults aged 20 years and above (Storey and Anderson, 2014). Similar to carbohydrates, the significant increase was between baseline and 6 months but not between baseline and 3 months. Carbohydrate and dietary fibre share many common sources. Most fibre are found in carbohydrate rich foods such as whole grains, cereals, some fruits as well as root vegetables such as potatoes (Australian National Health and Medical Research Council (NHMRC) and the New Zealand Ministry of Health (MoH), 2005). An increased intake of one may easily result in a corresponding increase in

the other and vice versa. The increment in the proportions of patients who consumed foods made from whole grains may have resulted in the significant increase in dietary fibre intake. Although the frequency of intake of most fruits did not change significantly throughout the study period, the proportions of patients who consumed fruits progressively increased from baseline through to 6 months. At 6 months, over 73% of patients were consuming most of the common fruits such as oranges, pawpaw, banana, watermelon and apples compared to over 41% at baseline, with the majority consuming them at least twice a week. Fruit and vegetable intake among adult Ghanaians are generally low. About 36.6% of males and 38% of females consume less than the minimum recommended 5 servings of fruit and vegetables a day (Hall *et al.,* 2009). Seasonal variation in the consumption of fruits and vegetables, where frequency and amount of fruit intake is increased during their bumper season and vice versa, have been reported in some studies (Locke *et al*., 2009; Toorang *et al*., 2013). However, seasonal variation may not have affected reported fruit and vegetable intake much, among participants of this current study. This is because, recruitment and follow-up of study participants lasted over 18 months, spanning both the lean and bumper seasons of most fruits and vegetables repeatedly. Patients reported fruit and vegetable intake were therefore a good reflection of their usual intake all year round. Dietary intervention therefore succeeded in convincing most patients who hitherto did not include fruits in their regular diets, to begin to eat them at least twice a week.

Similar trends were observed in the intake of vegetables. For most vegetables, the frequency of their intake by patients did not change significantly, but the proportions of patients who consumed them within a week increased markedly. At 6 months over 76% of patients were consuming them compared to over 53% at baseline, with the exception of cucumbers. By the 6^{th} month most patients ate vegetables at least 2 times a week. Cabbage and carrot were

the only vegetables which reported significant increase in the frequency of their intake at 3 and 6 months compared to baseline. The proportion of patients who never consumed these vegetables significantly reduced at 3 and 6 months while those whose intake were low and moderate increased significantly over the same period. The increased proportions of patients who consumed both fruits and vegetables in their weekly diets may also have contributed to the significant increase in dietary fibre intake observed. This significant increase in dietary fibre intake, however, occurred among males but not females. A reasonable explanation would be that, the pre-intervention (baseline) daily dietary fibre intake was so much below the recommended intake among males than females. As males approached the recommended intakes therefore, the difference was significant. Females were already consuming dietary fibre close to recommended daily intakes pre-intervention. Their increased intake post-intervention towards recommended intake was therefore not significantly different from what they previously consumed. Similar report was made by Leblanc *et al.,* (2015), who observed that men seemed to benefit more from nutritional intervention to promote the Mediterranean diet, as their metabolic profile showed more significant improvement than women, due to the more deteriorated profiles of men at baseline compared to women. A contrary report was made by Storey and Anderson, (2014). In their study, males significantly consumed more dietary fibre than females (Storey and Anderson, 2014). They also found a significant linear correlation between dietary fibre intake and income levels and explained the higher dietary fibre intake among men to be associated with their higher income levels compared to women (Storey and Anderson, 2014). Among a group of university students in Ibadan Nigeria, mean dietary fibre intake of both males and females exceeded the recommended daily allowance of 30g/day (Adegoke *et al.*, 2006). Mean daily dietary fibre among males was 54.2 ± 13.7g and that of female was 40.5 ± 8.5g. Most of the fibre in their diets were contributed by cereals which are rich in

Nigerian diets. They are cheaper and accessible and together with tubers and roots, formed the main staples of Nigerian diet. They are also cheaper than the refined foods. Fruits and vegetables, however, made modest contributions to dietary fibre intake (Adegoke *et al.*, 2006).

Despite their significant increase in dietary fibre intake after 6 months, the mean daily fibre intake among males was still lower than that of females. Mean daily carbohydrate intake predicted dietary fibre intake among patients in a multiple linear regression model. An increase in carbohydrate intake corresponded to an increase in dietary fibre intake. Most fibre are found in carbohydrate rich foods such as whole grains, cereals, some fruits as well as root vegetables such as potatoes (Australian National Health and Medical Research Council (NHMRC) and the New Zealand Ministry of Health (MoH), 2005). Females recorded the highest carbohydrate intake at 6 months while males recorded the highest cholesterol intake at the same period. This could explain why dietary fibre intake was higher among females compared to males. At the end of 6 months, though dietary fibre intake had significantly increased among the group, their mean daily intake still fell below the recommended 25g -30g intake which is estimated to reduce the risk of obesity and other chronic disease and their complications. Dietary intervention therefore significantly increased dietary fibre intake of patients, although their level of intake still fell below recommended intakes.

5.2.2.4 Changes in patients' dietary fat intake after receiving dietetic counselling.

Daily total fat intake reduced progressively from baseline through 3 to 6 months although the difference was not statistically significant. This trend occurred among females although among males their daily total fat intake decreased at 3 months but increased again at 6

months. Their intake at 6 months was, however, lower than that at baseline. This suggests a general decreasing trend among the group. The progressive decrease in the median intake of total cholesterol from baseline to 6 months and the decrease in median intake of saturated fat at 6 months may have contributed in part to the general decrease in total fat intake. The observed increase in the proportions of patients who never consumed some fatty foods by the 6 month may also have added to the decrease. Foods like pastries, butter, margarine and crabs showed more people abstaining from them at 6 months compared to baseline. It is possible that, these were not very common in the diets of patients to start with, and were therefore easily dispensable from their diets.

More patients also included some lean foods in their regular diets at 6 months compared to baseline. Skimmed milk, cooked beans, *agushie* and boiled egg all showed marked decreases in the proportions of patients who never consumed them at 6 months compared to baseline, with corresponding increase in those who consumed them at least once a week. The most commonly consumed lean food at 6 months was smoked fish. Over 50% of patients consumed them 2 or more times a week. Smoked fish, however, has most of its natural fish oils lost during the smoking process and therefore may not be a good source of the omega 3 fatty acids which are protective against cardiovascular disease.

While the decreases in saturated fat and cholesterol were markedly evident among females, males conversely showed marked increase especially in their total cholesterol intake and a marginal increase in saturated fats intake. Saturated fat and cholesterol are all found in animal source foods (O'Sullivan *et al.,* 2013). Anecdotal evidence in Ghana suggest that due to the relatively higher cost of animal proteins, people of higher socioeconomic status are more able to patronize them. Ghanaian men tend to have higher education and income

levels compared to their female counterparts (Ghana Statistical Service, 2014). In addition, culturally, men are served with the choicest parts of meats in a meal because mostly they are the bread winners in most African households (FAO, 2013). It is possible that their higher median intakes of saturated fat and total cholesterol were as a result of their relatively higher intake of animal proteins.

Fried fish followed by jollof rice, palm soup and cream milk were the most commonly consumed fatty foods by the majority of patients. Over 20% of patients consumed them 2 or more times weekly by the 6th month. Additionally, the proportion of patients who never consumed fried meat and *domedo* significantly reduced by the 6th month with corresponding increase in those who consumed them at least once a week. Frying introduces inferior oils to the food and a loss of the natural healthy fish oils (Przybylski and Aladedunye, 2012). In most cases, especially among those who fry fish or meat for commercial purposes, palm kennel oil is used, due to its characteristic aroma and low cost. Palm kennel oil is high in saturated fat, containing about 85% saturated fatty acids, mainly lauric and myristic acids (Mancini *et al.*, 2015). The process of deep frying is also characterized by water loss and oil uptake by food, resulting in a high oil content of the deep-fried food (Ma *et al.*, 2016). The oils used in frying are also used repeatedly to fry batch after batch. Repeated heating of oils reduces the amount of polyunsaturated fats due to oxidative degradation, thereby reducing the health benefits associated with the oil (Przybylski and Aladedunye, 2012). Consuming fried fish 2 or more times in a week suggest intake of additional oils which will only increase the energy content of food and lead to obesity with its associated risk of CVDs and other chronic diseases. This needless risk can be avoided by replacing fried fish intake with intake of minimally grilled or steamed fish, especially oily fish, at least twice a week (Ministry of Health, 2012), to improve blood lipid profile as well as maintain healthy weight.

5.2.2.4 Changes in patients' dietary sodium intake after receiving dietetic counselling.

Daily sodium intake progressively decreased from baseline to 6 months with the least median intake occurring at 6 months. Median sodium intake at baseline and 6 months among males was higher than that of females. Generally, daily sodium intake at all periods of the study were markedly below the recommended intake of 2,300mg (National Cholesterol Education Programme, 2001). The 24-hour recall used to investigate sodium intake did not investigate addition of salt at table or amounts of salt added to food when cooking. Further, the ESHA food processor used to convert food intake into nutrient intake is based on foods prepared using standard healthy recipes. Most people may not necessarily follow these standard recipes in their cooking. Sodium intake may therefore have been underestimated. This notwithstanding, the decreasing trend of daily sodium intake among all patients may suggest that dietary intervention to limit salt intake was effective. Most patients never consumed many of the known salted foods such as *koobi, momoni, kako*, salted pig feet and *toolo-beef* etc. Only about 30% of patients consumed *maggi, koobi, dzomi and momoni* mostly once a week. This suggests that these foods may not necessarily be the main sources of high sodium intake in patients' regular diet. Other cooking and eating behaviour such as amount of salt used in cooking and adding salt at table may possibly be a more likely source of high sodium intake in the diet than the intake of these salted foods. The daily salt intake of adult Ghanaians has been estimated as 9g (Ministry of Health, 2012). Practices such as amount of salt used in cooking (including the use of stock cubes, soy sauce and spices) and adding salt to food at table, were cited as the main sources of salt intake in our meals (Ministry of Health, 2012). Patient education and counselling will therefore need to firmly investigate and address these habits. The food intake frequency assessment showed that those who managed to reduce their intake or abstained from eating some salted foods between baseline and 3 months only reversed to their usual intake by 6 months. This is

indicative of short-term, but not long-term effectiveness of dietary intervention to reduce salt intake.

5.2.3 Changes in patients' lifestyle indicators after receiving lifestyle intervention.

Counselling on healthy lifestyle impacted the different lifestyle indicators to varying extents, and are discussed below.

5.2.3.1 Changes in patients' physical activity habits following counselling on healthy lifestyle.

Significant changes in patient's physical activity habits were observed after the dietary and lifestyle intervention. The proportion of patients who engaged in moderate physical activity significantly increased at 3 months. The proportion dwindled at 6 months although it was still significantly higher compared to baseline. The number of days in a week on which physical activity occurred as well as the amount of time spent on physical activity did not change. It suggests that lifestyle intervention may be effective in increasing the numbers of people who engage in moderate physical activity, but not the time spent on physical activity or the frequency of physical activity. The significant increase in numbers may also be guaranteed in the short term but not in the long term. Poor adherence to recommended lifestyle interventions is widespread, particularly over the long-term (Middleton *et al.*, 2013). Common reasons given for not adhering to physical activity is stress, fatigue, lack of time and lack of facilities in the community to enhance physical activity (Middleton *et al.*, 2013). These are similar to reasons which patients in this current study gave for not engaging in regular physical activity.

An inherent resistance to physical activity, particularly those that are programme centred, as well as vigorous intensity activities, has been suggested (Laitakari *et al.,* 1996). People are reportedly more responsive and committed to activities that form part of daily routine e.g. exercising as a means of transport, house chores etc. In general, activities that are moderate in intensity and that are easily integrated into an individuals' lifestyle is more likely to be maintained (Laitakari *et al.,* 1996)

Individuals having various health challenges are reportedly more susceptible to lifestyle modifications than apparently healthy individuals (Ammerman *et al.,* 2002). In this study, some patients admitted that they engaged in moderate physical activity because their doctors had advised them to. The elderly age of most patients and their busy work schedules may also have prevented them from increasing their number of days and the time spent on physical activity per session. Similar results were reported by Pate *et al.,* (2015), who reported that medium to vigorous physical activity was lowest among the older adult group of their study participants compared to the younger adult group due to their older age (Pate *et al.,* 2015). The health status of study participants may also have played a role in their lower physical activity levels. Individuals with perceived poor health are likely to reduce or avoid physical activity even after they have been taken through a physical activity intervention programme (Droomers *et al.,* 2001). Males were generally more physically active than females, with significantly more males engaging in moderate physical activity at baseline and 6 months that females.

Patient's body mass index and educational level were the 2 variables that predicted whether they engaged in moderate physical activity or not in a binary logistic regression model. Together, they predicted 13% of the variability of engaging in moderate physical activity.

Compared to obese patients, patients with normal BMI had 55% odds of not engaging in moderate physical activity while overweight patients had 40% odds of not engaging in moderate physical activity. Hence patients who were less obese were more likely not to engage in physical activity. This may be because, they may have the erroneous impression that physical activity is only useful for weight reduction. Some participants did indicate that their motivation for engaging in moderate physical activity was to lose weight. Therefore, when patients considered that they had a healthy weight, they did not see the need to be physically active.

Compared to patients with a tertiary education, the odds of patients with no formal education not engaging in moderate physical activity was 19 times that of those with tertiary education. Patients who had completed primary education had 3 times the odds of not engaging in moderate physical as those who had tertiary education while the odds of not engaging in moderate physical activity for those who had completed secondary education was approximately 2 times that of those with tertiary education. Higher education therefore enhanced a positive physical activity culture. People of lower educational levels may fail to appreciate the important role that physical activity plays in improving health. They may also fail to invest time and other resources necessary for physical activity. This may be because their major preoccupation is how to acquire their basic needs, especially because they often belong to the lower socioeconomic bracket. Droomers *et al.,* (2001) similarly reported significantly higher odds of decreasing physical activity during follow-up among people of low socioeconomic status. The main reason identified for this outcome was a low perceived control among this group. Their belief that they had little control over their inside state and behaviours, the place, people, things, feelings or activities surrounding them, led to higher

odds of decreasing physical activity during follow-up, compared to those of higher educational levels.

5.2.3.2 Changes in patients' alcohol intake habits following lifestyle intervention.

Alcohol intake habits of patients did not change throughout the follow-up period. Both the frequency and the amount intake remained comparable throughout the period. At all stages of follow-up, the majority of patients who consumed alcohol did so less than once a month and less than 2 drinks in a day. Their alcohol intake frequency was therefore generally low and at levels that are not risky to their health. This may have accounted for the no significant changes in alcohol intake as for the majority of them, their intakes were already below thee recommended healthy level, hence there wasn't much to change.

5.2.3.3 Effect of lifestyle intervention on patients' smoking habits

No patient smoked at 3 or 6 month follow-up. The only 2 patients who smoked at baseline were lost to follow-up. Among the non-smokers, there were no incident smoking during the study.

5.2.4. Changes in patients' blood pressure following dietary and lifestyle intervention

Mean systolic and diastolic blood pressures did not change significantly from baseline through to 6 months. For all patients, the mean systolic and diastolic blood pressure at baseline, 3 and 6 months remained at stage 1 hypertension. This was also seen among females. Male values at all stages of the study were, however, higher, indicating stage 2 hypertension, with the highest record at 6 months. Females recorded their lowest at 6 months. Although men are reported to have higher risk of hypertension than women, the

risk is eliminated between the ages of 45 to 64 years, where both genders have equal risk, due in part to the loss of oestrogen protection at menopause (Ghosh *et al.*, 2016). The mean age of patients in this study was 49.6 ± 10.2 years, suggesting that both males and females were at equal risk of hypertension. Although sodium intake of all patients was below the recommended daily intake, male intake was marginally higher compared to females. Males generally had better blood lipid profile than females. However, their dietary saturated fat and total cholesterol intake increased over the study period, with the highest intakes of both nutrients recorded at 6 months. This was in contrast to that of females whose intakes of the two nutrients rather decreased over the period.

It is suggested that both behavioural and biological factors affect blood pressure levels among males and females. The most crucial behavioural factors identified are obesity status, smoking and physical activity with sex hormones and chromosomal differences accounting for the biological factors (Everett and Zajacova, 2015; Sandberg and Ji, 2012). More males had preciously smoked compared to females. Although the dietary fibre intake of males significantly increased, the overall mean dietary fibre intake at 6 months was higher among females compared to males. High intake of dietary fibre has been associated with a reduction in CVD risk as well as incident hypertension (Huang *et al.*, 2015). Patients who consumed between 48g and 80 g (3 to 5 serving/day) of dietary fibre daily had a 21% lower risk of CVD (relative risk = 0.79; 95% CI, 0.74 –0.85) (Huang *et al.*, 2015). Hence, relatively higher fibre intake among females must have conferred better control of their blood pressure compared to males. Females recorded significantly higher BMI compared to males, while males were significantly more physically active compared to females. The biological and behavioural factors put together therefore determined whether the gender gap in hypertension levels were further widened or eliminated. In this study, the gender variation

persisted in the face of a combination of positive and negative health behaviour among both genders.

5.2.5 Changes in patients' anthropometry and body composition indicators following routine dietary and lifestyle intervention at the KBTH.

Dietary and lifestyle intervention was effective in significantly reducing body mass index and visceral fat of all patients but not the other anthropometric and body composition indicators.

5.2.5.1 Changes in patient's anthropometry and body composition indicators following dietary and lifestyle intervention

The significant reduction in body mass index was evident among females but not males. It occurred in the short term and was sustained through to the end of the study. By the 3rd month of follow-up, there were no underweight patients, suggesting that the few underweight patients at the beginning of the study now had normal healthy BMI. The proportion of patients who had normal healthy BMI increased at 6 months but not at 3 months. This suggest that, between the 3rd and 6th month, some patients who may have been overweight or obese, experienced a decrease in their BMI to attain normal healthy BMI. Fig 4.7.1 shows that the prevalence of both overweight and obesity among patients experienced its major decrease between the 3rd and 6th months while at the same period the prevalence of normal weight experienced its major rise. The significant increase in dietary fibre intake together with that of the proportion of patients who engaged in moderate physical activity may have accounted for this observed change (Sims *et al.,* 2013). In spite of these encouraging results, the fact that throughout the study, over 50% of patients were obese should be a course for concern, due to the heightened risk of chronic disease and

complications associated with obesity (Zhang *et al.*, 2014). These results affirm the evidence that obesity is a major risk factor for non-communicable diseases and may have contributed to their current health state to start with (Kawarazaki and Fujita, 2016; Hall *et al.*, 2015; Goodfriend, 2008).

The alarming prevalence of obesity among the study participants throughout the study period notwithstanding, there was a significant reduction in visceral fat among the entire group between baseline and 3 months, which remained significantly low at 6 months. Reduction in percentage body fat was borderline significant while marginal increases in muscle mass and total body water also occurred. Among females, the reduction in percentage body fat and the increase in total body water were significant. These changes suggest that, although the prevalence of obesity remained above 50% throughout the study, the various components of the body which contributed to the excess weight were changed by the end of the study. Some reviews have drawn attention to the fact that, weight loss should not be the only target to cardiovascular disease risk reduction (Ross and Janiszewski, 2008) and physical activity interventions (Chaput *et al.*, 2011). The improvement in body composition parameters needs to be highlighted because it confers cardiorespiratory fitness, which is evidence of a reduction in the risk of cardiovascular and other chronic diseases (Chaput *et al.*, 2011; Ross and Janiszewski, 2008). The findings in this study further endorses the limitation of BMI in differentiating between weight from muscle and weight from fat (Ranasinghe *et al.*, 2013; WHO, 2000). Hence, a given BMI may not correspond to the same degree of fatness across populations partly due to differences in body proportions (WHO, 2000).

The significant decrease in BMI among patients most likely contributed to the improvements in some biochemical indicators such as fasting and 2-hour glucose as well as HDL which are later discussed.

The factors that predicted patients' body mass index in a linear regression model were their percentage body fat, visceral fat, gender and age. Increasing percent body fat and visceral fat resulted in a corresponding increase in BMI. Females had (3.71 kgm^{-2}) higher BMI than males while a 1year increase in age corresponded to a 0.24 kgm^{-2} decrease in BMI. This result agrees with evidence from obesity research which has established the linear association of fat mass with BMI (Akindele *et al.*, 2016; Ranasinghe *et al.*, 2013). A strong positive association (r = 0.81) was also reported between BMI and percentage body fat among adult Nigerians living in urban Nigeria. Age and gender predicted the association (Akindele *et al.*, 2016).

5.2.5.2 Factors that contributed to the changes in patients' visceral fats

Visceral fat of all patients, decreased significantly in both the short and long term. The proportion of patients who had healthy visceral fat remained the same between baseline and 3 months but increased markedly between 3 and 6 months. By similar amount of increase, the proportion of patients with unhealthy visceral fat also decreased between 3 and 6 months after showing a plateau between baseline and 3 months. This suggests that, some patients with unhealthy visceral fat had lost some fat, through healthy dietary modification and physical activity to attain healthy visceral fat. These results suggest that dietary and lifestyle intervention resulted in the lowering of patients' visceral fat in the long term but not in the short term.

The multiple linear regression model which accounted 64 % of the variability in patient's visceral fat estimated the visceral fat in males to exceed that of females by 7. Visceral fat further increased by 0.106 for every year increase in age. Physiologically, excess fat deposits among males is reported to occur mainly around the abdominal area and may lead to high visceral fat (WHO, 2008b). Evidence of high truncal fat was reported at pre-puberty, with pre-puberty girls having 5% less ($P = 0.027$) and adult women having 48% less ($P < 0.0001$) waist fat than males (Taylor *et al.*, 2010). Men tend to have a more central fat pattern compared with the more peripheral fat distribution typically observed in adult women. The action of sex hormones is reported to account for the differences in body composition between the genders which becomes evident from the onset of puberty (Traish *et al.*, 2009; Taylor *et al.*, 2010). With aging, testosterone levels in men reduce, it results in a reduction in muscle mass and a corresponding increase in fat mass. Testosterone levels are therefore inversely associated with obesity in men (WHO, 2008b; Traish *et al.*, 2009). Among females, from late middle age to the 80's, there is a decline in the volume of subcutaneous fat and a redistribution of fat from subcutaneous to visceral regions. This is accompanied by accumulation of fat outside adipose tissue (in muscle, liver and bone marrow) and loss of lean body mass (WHO, 2008b). Additional results from the linear regression model in this current study showed that increasing percentage body fat resulted in a corresponding increase in visceral fat. Percentage body fat is the sum of visceral fat and subcutaneous fat. Increment in percentage body fat can therefore result from increases in both subcutaneous and visceral fat and vice versa (Mahan and Escott-Stump, 2008). Excess dietary intake without a corresponding increase in physical activity, can also result in an increase in percentage body fat and subsequently visceral fat. In this current study, patients' carbohydrate and protein intakes both exceeded the recommended daily allowance at all stages of the study. The main protein rich food in the diets of patients were fried/ smoked

fish, cream milk and skimmed milk. Protein rich foods, especially protein from animal sources such as milk, meat and egg are also good sources of saturated fat and dietary cholesterol (O'Sullivan *et al.,* 2013). High intake of these foods can therefore result in increased intake of saturated fat and cholesterol, which can contribute to increases in visceral fat in the absence of adequate physical activity (Narasimhan *et al.*, 2016). Duration and frequency of moderate physical activity remained effectively unchanged throughout the study period. Incidentally, these are the very attributes of physical activity which have been associated with loss of body fat and storage energy (Sahlin *et al*, 2008; Manore *et al.,* 2017). Although protein intake was higher than the recommended daily allowance for all patients at all stages of the study, its contribution to total daily energy intake was markedly below the allowable macronutrient distribution range (AMDR). This was mostly due to the exaggerated intake of carbohydrate intake, which resulted in the relative reduction in the contribution of protein to total daily energy intake.

5.2.6 Changes in patients' blood sugar following dietary and lifestyle intervention.

Baseline fasting plasma glucose reduced significantly by 3 months and was sustained through to the end of the study at 6 months. The change was evident among both males and female. Two-hour glucose likewise decreased significantly at the end of 6 months. The difference was evident only among females. In this study, 41.7% of patients were on oral hypoglycaemic agents while 3.0% were on insulin. A total of 71.6% of all patients regularly took their medication as prescribed by a medical doctor at 6 months. A number of factors may therefore have accounted for the observed differences in patients' fasting and 2-hour plasma glucose in addition to the intake of oral hypoglycaemic agents. Dietary fibre intake among patients significantly increased. Dietary fibre especially soluble fibre from fruits vegetables, oats etc. regulates the absorption of sugar into the blood postprandial, by

forming gels through which glucose passes on its way to the intestines for absorption, thereby controlling post prandial plasma glucose (Goff *et al.,* 2018). A meta-analysis of six cohort studies including 286,125 participants and 10,944 cases, a two servings per day increment in whole grain intake was associated with a 21% (95% CI, 13–28%) decrease in risk of type 2 diabetes (Huang *et al.,* 2015). Increased dietary fibre intake coupled with the significant increase in the proportion of patients who engaged in moderate physical activity, contributed to a significant decrease in patients' BMI and visceral fat (Walsh *et al.,* 2018). Moderate weight loss up to about 5% loss is estimated to improve insulin action and control plasma glucose (American Diabetes Association, 2017). Improvement in fasting blood glucose is estimated to be directly related to the relative amount of weight lost (UK Prospective Diabetes Study 7, 1990). Over 13% of the variability in fasting plasma glucose was explained by WHR, which is an index of obesity. In one study, blood glucose was significantly associated with BMI, an increase of 1 mmol/L in glucose was associated with 0.33 additional unit increase in BMI (Walsh *et al.,* 2018). In this study waist-hip circumference ratio and gender were the two predictors of fasting plasma glucose among patients. The two variables predicted 13.9% of the variability in fasting plasma glucose. FPG increased by 20.3 mmol/L for every unit increase in WHR. Females as well had a 1.42mmol/L higher fasting plasma glucose than males. A high waist-hip ratio is suggestive of the accumulation of adipose tissue in the abdominal region relative to the hip area. Similarly, 2-hour plasma glucose was also predicted by the fasting plasma glucose and waist-hip circumference ratio in a linear regression equation. Two-hour plasma glucose increased by 1.23 mmol/L for every 1 mmol/L increase in fasting plasma glucose and also increased by 19.13 mmol/L, for every unit increase in waist hip circumference ratio. Adipose tissue acts as a hormone which secrets substances that predispose to insulin insensitivity, therefore increasing the risk of type 2 diabetes (Kawarazaki and Fujita, 2016).

Most of the risk associated with obesity and chronic disease is blamed on central obesity (Goodfriend, 2008; Hall *et al.,* 2015). WHR remained unchanged among patients in this study. Mean WHR among females was, however, above the normal healthy limit of 8.5, above which risk of chronic conditions increases (WHO, 2000). Female gender has also been documented as a risk factor for type 2 diabetes (Alotaibi *et al.,* 2017). These findings therefore corroborate with that of other studies.

5.2.7 Changes in patients' serum lipids following dietary and lifestyle intervention.

There was no significant change in serum lipids throughout this study with the exception of HDL cholesterol. HDL significantly increased in the long term, between baseline and 6 months but not in the short term. A total of 37.9% of patients were on statins at baseline. Dietary and lifestyle intervention together with statin treatment was therefore effective in improving serum HDL cholesterol in the long term, but not the other blood lipids. The change was evident among the total group of patients but not among males or females. The numbers in the individual gender groups may have been too small to show any significant changes in HDL or longer follow-up periods may have been required to show any significant change. Contrary results were reported of a decreased in HDL and an increase of all other components of the lipid profile after 30 days of a dietary intervention based on a low-fat plant base diet (Kent *et al.,* 2013). HDL is an important component of the blood lipids. It is documented as an independent risk factor for CVD, especially coronary heart disease (Wesnigk *et al.,* 2016). Low HDL is one of the criteria for diagnosing the metabolic syndrome (Menotti *et al.,* 2011). The main lifestyle interventions which have been shown to positively modify HDL are smoking cessation, weight loss and physical activity (Escolà-Gil *et al.,* 2015). In this current study, significant reduction in patients' BMI and visceral fat occurred. Significantly more patients started engaging in moderate physical activity. By

the 6th month, no patient smoked. These factors could have contributed to the significant increase in HDL at 6 months.

The factors that explained 39% of the variability in patient's HDL levels were percentage body fat, serum triglyceride, total cholesterol and low density lipoprotein cholesterol. A unit increase in percentage body fat, serum triglyceride and LDL resulted in a reduction of HDL to different extents. Only a unit increase in serum total cholesterol resulted in a 0.25 mmol/L increase in HDL. Percentage body fat is a marker of obesity and share similar risk factors as that of unfavourable HDL levels e.g. physical inactivity. Serum total cholesterol, triglyceride and LDL cholesterol recorded minor changes, though not significant, throughout the study. In most cases, they remained within healthy limits. Total cholesterol and triglyceride levels were, however, mostly higher among female patients than their male counterparts. A possible explanation may be the significantly higher carbohydrate intake among females compared to males at baseline and at 6 months. High intake of carbohydrates has been associated with atherogenic dyslipidaemia, characterized by elevated triglycerides, reduced HDL concentrations and elevated small density lipoprotein cholesterol which endanger cardio metabolic health and increase cardiovascular diseases and total mortality (Siri-Tarino *et al.*, 2015).

CHAPTER SIX

6.0 CONCLUSIONS, RECOMMENDATIONS AND LIMITATIONS

6.1 Conclusions

The study concluded that hypertension was the major diet related chronic condition referred to the Dietherapy Department of the KBTH. It was followed by diabetes, dyslipidaemia and obesity. Referral for obesity was not as high as anticipated despite the relatively high national prevalence.

The demography and socioeconomic profile of patients referred to the Dietherapy Department were reflective of the national population as reported in national research studies and census reports. Close to 50% of participants had no formal education or had attained primary education and were mostly artisans. The majority were married and were employed.

Half of patients who were referred to see the dietitian delayed in assessing dietary care. The main reason for the delay was because even though patients had sought care immediately, on the same day they were referred, the Department had closed when they eventually got there later in the day. Long queues at the OPD, the hospital's Central Laboratory Department as well as Pharmacy, where patients mostly report for the requested laboratory test and prescribed medications, were all crowded with patients and therefore it took a longer time for them to be attended to. Others could not find the time, had to travel and therefore waited until their return, or were too sick to move around and seek dietetic care. Other reasons such as lack of funds or carers to take them to the hospital and the loss of their folders or referral letters, were also reported.

Alcohol intake, smoking and physical activity levels of patients were all generally low. Following dietary and lifestyle intervention, no significant change occurred in the alcohol intake of patients. The frequency and duration of physical activity additionally did not change. The proportion of patients who engaged in moderate physical activity, however, significantly increased. The main reason why patients did not engage in moderate physical activity was due to lack of time. For those who did, their main reason was to keep fit. BMI and education level predicted physical activity habits of patients. The odds of overweight and normal weight patients not engaging in moderate physical activity was progressively higher (40.0% and 55.1%) respectively, compared to that of the obese patients. The odds of not engaging in moderate physical activity by patients with no formal education, primary education or secondary education was 19 times, 3 times and 2 times respectively, that of those with tertiary education.

Following routine dietary and lifestyle intervention given by registered dietitians at the nation's major referral hospital, patients dietary intake, anthropometry, body composition, blood pressure and biochemical indicators changed to various extents.

While some nutrient intakes increased significantly, others marginally changed. Mean daily energy intake did not change significantly throughout the study. The contributions from the macronutrients, however, changed. Carbohydrate intake increased significantly between baseline and 6 months mostly due to the inclusion of whole grains and fruits to the usual diets of patients. The main sources of whole grains included in the diets were oats, wheat and *tombrown* (a mixed cereal porridge). *Banku* and *kenkey* were already commonly consumed by majority of patients at baseline. Commonly consumed starches, mainly plain

rice, boiled yam, boiled plantain and fufu at baseline continued to by consumed by the majority of patients at least 2 times in a week, throughout the study.

Marginal but progressive decreases in patients total dietary fat, saturated fat and cholesterol intake occurred throughout the study. This was mostly evident among females but not males. The most common fatty food consumed by the majority of patients was fried fish. It was followed by *jollof* rice, cream milk, groundnut soup and palm soup. Although fish intake was frequent among majority of patients, the associated benefits may be lost due to the methods of preparation, mostly frying and smoking. Both methods are associated with the loss of natural healthy fish oils which are protective against CVDs. Protein intake reduced marginally at 6 months compared to baseline.

Contrary to what was earlier believed, common salted foods, mostly *koobi*, *momoni*, *kako*, salted pig feet and *toolo beefe*, were not consumed frequently by the majority of patients, and could not have contributed significantly to dietary sodium intake. Sodium intake progressively decreased while potassium intake progressively increased throughout the study, though not significantly.

Dietary fibre intake increased significantly between 3 and 6 months mainly from the significant increases in the proportions of patients who consumed whole cereals like oats, wheat and fermented maize products as well as vegetables like carrot and cabbage. Most of these patients consumed them at least twice in a week. Mean daily carbohydrate intake predicted 56.5% of the variability in dietary fibre intake.

Body mass index and visceral fat were the two anthropometric indices which decreased significantly between baseline and 3 months. The significant decrease was sustained at 6 months. Systolic and diastolic blood pressure remained unchanged despite pharmacological treatment and lifestyle modification.

Among the biochemical indicators, fasting plasma glucose significantly decreased between baseline and 3 months. The significant decrease was sustained at 6 months. Two-hour glucose also significantly decreased at 6 months. Both fasting and 2-hour glucose were predicted by waist-hip circumference ratio. High density lipoprotein cholesterol also increased significantly at 6 months. The variability in HDL was explained inversely by patients' percentage body fat, LDL and triglyceride but linearly by serum total cholesterol in a multiple linear regression model.

The null hypothesis of this study is therefore rejected. The conclusion follows that, routine dietary and lifestyle intervention given to patients with lifestyle related chronic diseases, by registered dietitians at the KBTH, led to improvement in a significant number of the measurable outcomes. The improvement in the outcomes has the potential to reduce the related complications of the NCDs if maintained on the long term.

6.2 Recommendations

The following are some recommendations informed by the finding from this study.

1. Dietetic care is recommended for all patients who are diagnosed with lifestyle related chronic diseases for improved health.
2. At least all regional and district health facilities across the nation should be resourced with registered dietitians to improve patient care.

3. The Dietherapy Department of KBTH should develop a standard protocol for patient counselling to include:
    a. the information to give (e.g. recommended sources of carbohydrate and the portions to eat at a meal).
    b. how to present the information (counselling style to adopt)
4. Physicians need to increase referral of patients for dietetic care, especially obese patients who attend the hospital's OPD, with or without NCDs for weight management, to control the increasing NCD prevalence.
5. The Dietherapy Department at the KBTH may consider running a full day's clinic ending at 5 pm.
6. Public health promotion programmes needs to take advantage of low alcohol and smoking levels among Ghanaians to promote their continuous low levels to prevent escalating prevalence of NCDs.
7. National health promotion programmes such as the Regenerative Health and Nutrition Programme needs to be revived. Such programmes should emphasize the need for daily intake of fruits and vegetables and frequent and longer lasting physical activities, especially among the obese and the less educated communities.
8. Dietitians need to explore the use of mhealth (e.g. sms) to send periodic reminders of lifestyle targets to patients, to sustain improved health outcomes in the long term.
9. Further research should investigate the effect of different counselling styles by dietitians on patient health outcomes.
10. Further research to test regression equations for validity are also recommended.
11. Patients counselling on salt intake should place more emphasis on reduction of salt used in cooking and avoiding salt added at table, than on avoidance of the intake of common salty foods.

12. Patients should be encouraged to prepare fish using processes such as steaming, which does not result in much loss of natural fish oils.
13. Replacement of refined carbohydrates in usual diet with whole grains, and not the addition of whole grains to usual food intake, should be well explained during patient counselling to prevent excess carbohydrate intake.
14. Activities at the hospital's main pharmacy and laboratory needs to be streamlined to reduce the amount of time patients spend when patronizing their services.
15. Feather research involving a larger sample also needs to investigate the effect of dietetic care on patient's blood pressure. This can involve both patients who may or may not be receiving pharmacological treatment.

6.3 Contribution to Knowledge

- The common conditions which medical doctors refer to dietitians for dietary management, which was previously unknown, has now been determined.
- Patients' dietetic care seeking behaviour after referral, and the major factors which contribute to delays in seeking prompt care is now known.
- The evidence of the effect of routine dietetic care given to patients at the nation's major referral hospital, on some of their important clinical and lifestyle indicators, has now been scientifically determined.
- Conditions, e.g. high blood pressure, which may require closer monitoring and evaluation when being managed has been identified.
- Areas in dietetic counselling of patients which require better clarity has been identified.

6.4 Limitations

Certain confounding factors which could not be effectively controlled in this study and may have influenced the findings are outlined below.

1. Different registered dietitians at the Dietherapy Department counselled patients as they reported. Their different counselling approaches could have affected patient's ability to comply with dietary recommendations.
2. Changes in patients' medication, including changes in the type or dosage was not investigated at follow-up. Further, the influence of the medication on the measurable outcomes cannot be ruled out. However, withholding the medication of the patients to assess the impact of the dietary and lifestyle intervention would have been unethical.
3. Twenty-four-hour recall interviews were based on patient's ability to recall and may have influenced their estimation of food portions consumed. In addition, 24- hour recall at baseline was done for only one day, in order to assess patients' dietary intake before they received dietary counselling. It could not be repeated as required because, routinely, at the Dietherapy department, patients are programmed to receive dietary counselling on the same day they visit. Their dietary intake subsequently was therefore no longer reflective of their pre-counselling intake and could therefore not be considered as baseline dietary intake.
4. ESHA FPRO nutrition software used in nutrient analysis are based on foods prepared using standard recipes and may have over or underreported some nutrients consumed by patients.

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APPENDICES

APPENDIX I: Ethical Clearance

Please always quote the ethical identification number in all future correspondence in relation to this protocol.

Thank you.

Yours sincerely,

Dr. George Asare
(Chairman, Ethical and Protocol Review Comm

cc Ag. Dean
Co-ordinator, Dept. of Dietetics
Senior Assistant Registrar

SCHOOL OF ALLIED HEALTH SCIENCES
COLLEGE OF HEALTH SCIENCES
UNIVERSITY OF GHANA
ACADEMIC AFFAIRS

Phone: +233-0302-687974/5
Fax: +233-0302-688291
My Ref. No. SAHS/PSM/DT/04
Your Ref. No.

P. O .Box KB 143
Korle Bu
Accra
Ghana

29th March, 2012

Mrs. Rebecca K Steele-Dadzie,
Dept. of Dietetics,
SAHS.
Korle Bu.

Dear Mrs. Steele-Dadzie,

TEMPORARY ETHICAL CLEARANCE

Ethical Identification Number: SAHS – ET./SAHS/PSM/DT/04/AA/26A/2012-2013.

Following a meeting of the Ethical and Protocol Review Committee of the School of Allied Health Sciences held on Thursday 29th March, 2012, I write on behalf of the Committee to approve your research proposal as follows:

TITLE OF RESEARCH PROPOSAL: **"The effectiveness of dietary and lifestyle intervention in persons with major lifestyle related chronic diseases at the Korle Bu Teaching Hospital".**

This approval requires that you submit six-monthly review reports of the protocol to the Committee and a final full review to the Committee on completion of the research. The Committee may observe the procedures and records of the research during and after implementation.

Please note that any significant modification of the research must be submitted to the Committee for review and approval before its implementation.

You are required to report all serious adverse events related to this research to the Committee within seven (7) days verbally and fourteen (14) days in writing.

As part of the review process, it is the Committee's duty to review the ethical aspects of any manuscript that may be produced from this research. You will therefore, be required to furnish the Committee with any manuscript for publication.

APPENDIX II: Participant Information Sheet

DEPARTMENT OF NUTRITION AND DIETETICS, SCHOOL OF BIOMEDICAL AND ALLIED HEALTH SCIENCES

PARTICIPANT INFORMATION SHEET

Title of Research: The Effectiveness of Dietary and Lifestyle Intervention in Persons with Major Lifestyle Related Chronic Diseases at The Korle-Bu Teaching Hospital.

Participant Name: _____ Participant ID_____

There is evidence of increasing trends in the prevalence of diet-related chronic diseases among adult Ghanaians. The impact of dietary and lifestyle intervention given to patients on their disease outcomes however, remains to be investigated. We are therefore carrying out a research to determine the effectiveness of dietary and lifestyle intervention given to patients at the Dietherapy Department of the KBTH on their dietary intake, anthropometry, lifestyle, body composition, blood pressure and metabolic indices. You are being invited to take part in this research.

When you agree to take part in the research you will spend a few minutes answering questions about your personal medical health. We will also measure your blood pressure, height, weight, waist circumference, hip circumference and body composition. The information we ask about yourself and family may make you feel uncomfortable. This risk is, however, no more than you will normally be exposed to when you report for counselling at the department.

In addition, 12 ml, the equivalent of 2 and a half tea-spoonful of blood will also be drawn to measure your fasting and 2-hour plasma glucose, blood lipids and C-reactive protein, all of which determine cardiovascular risk. This will not affect your health negatively in any way, besides, a similar amount of blood is what the laboratories normally demand when you report for such medical check-up. Please understand that taking part in the research is entirely voluntary. You are further to note that you may refuse to take part or withdraw from the study at any time without anyone objecting or refusing you care at the department.

Please be assured that all information you give us in this research will be kept confidential and secure. The information will only be available to the researchers conducting this study. You are further assured that if a report of this study is prepared for the scientific and medical community you will not be identified by name.

Please note that there may be no personal benefit to you. However, your taking part in the study will help us assess the impact of dietary and lifestyle intervention on disease outcome to improve Dietetic care in Ghana. Do you have any questions or concerns about this study?" Should you later wish to have any matter or question relating to this research clarified, please contact Mrs. Rebecca Steele-Dadzie (024 6242805), Department of Nutrition and Dietetics, School of Biomedical and Allied Health Sciences, University of Ghana. Thank you.

APPENDIX III: Participant Consent Form

PARTICIPANT CONSENT FORM

I have fully explained to _____ the nature and purpose of the above described procedure and risks that are involved in its performance. I have answered and will answer to the best of my ability, all questions relating to the study.

_____	_____	_____
Signature	Name of Research Team Member	Date

I _____ have read (or have had read to me in a language that I fully understand) the proposed research and that I have understood what is going to be done. Also, any questions/concerns I have, have fully been explained to me by _____. My signature or thumbprint below indicates that I have understood what is going to be done and that I agree to take part in the study voluntarily.

_____ Date:_____
(Signature/thumbprint of Study Participant)

_____ Date:_____
(Signature: Witness)

APPENDIX IV: Cross-Sectional Questionnaire

DEPARTMENT OF NUTRITION AND DIETETICS, SCHOOL OF BIOMEDICAL AND ALLIED HEALTH SCIENCES

COLLEGE OF HEALTH SCIENCES

CROSS-SECTIONAL QUESTIONNAIRE

EFFECTIVENESS OF DIETARY AND LIFESTYLE INTERVENTION IN PERSONS WITH MAJOR LIFESTYLE RELATED CHRONIC DISEASES AT THE KORLE-BU TEACHING HOSPITAL.

DEMOGRAPHIC AND SOCIOECONOMIC QUESTIONNAIRE

1	**Date of completion of the instrument**	⌊_⌋⌊_⌋ ⌊_⌋⌊_⌋ ⌊_⌋⌊_⌋⌊_⌋⌊_⌋ dd mm year	I5

	Participant Id Number ⌊_⌋⌊_⌋⌊_⌋⌊_⌋⌊_⌋⌊_⌋	
Consent, Interview Language and Name	**Response**	**Code**

2	Consent has been obtained Yes=1 No=2 If no, do not proceed	_____	
2a	Name of Hospital ……..		

3	Name of Participant	
4a	Contact phone number:	
4b	Specify whose phone _____	
5.	Where do you stay? _____	

6. Gender (Record Male / Female as observed) Male=1; Female=2	_____	
7. How old are you?	Years ___ ___	

8.	What is your ethnic background	
9.	What is your religious affiliation?	

10.	What is the highest level of education you have completed?	

11.	What work do you do?	

CLINICAL INFORMATION

12. Condition for which patient was referred	Code
13. Date of referral by doctor to see the dietitian	
14. Date of reporting to dietitian	
15. Reason for delay if dates for 13 and14 are different …… …… ……	
16. Date of receiving dietary counseling by the dietitian	
17. Reason for delay if dates 14 and 16 are different …… ……	
18. Does any relative of yours have your condition? If yes please specify……..	

LIFESTYLE INFORMATION

19. Do you currently smoke any tobacco products, such as cigarettes, cigars or pipes? Yes =1 No =2; If No, go to Q. 23	_____	LSTYL19
20. If Yes, Do you currently smoke tobacco products daily? Yes =1 No =2;	_____	LSTYL20
21. How old were you when you first started smoking daily? If cannot remember, write 777 in the spaces	___ ___ ___ Age in years	LSTYL 21
22. On average, how many of the following do you smoke each day? (PLEASE RECORD EACH TYPE) Manufactured cigarettes Hand-rolled cigarettes Pipes full of tobacco Other If other, please specify type(s) _____	 ___ ___ ___ ___ ___ ___ ___ ___ ___ ___ ___ ___ ___ ___ __	 LSTYL 22a LSTYL 22b LSTYL 22c LSTYL 22d LSTYL 22e

23. In the past, did you ever smoke daily? Yes =1 No =2; If No, go to Q. 24	_____	LSTYL 23
24. If Yes, how old were you when you stopped smoking daily?	___ ___ ___ (Age in years)	LSTYL 24
25. Have you consumed alcohol such as beer, wine, brandy, aperitif, spirits, akpeteshie, palm wine, pito, "woba ada anaa" other alcoholic bitters, fermented cider or any other drink within the past 12 months? Yes =1 No =2; If No, go to Q. 28	_____	LSTYL 25
26. In the past 12 months, how frequently have you had at least one drink? _____	_____	LSTYL26
27. Have you consumed alcohol (such as beer, wine, spirits,		LSTYL27

akpeteshie, palm wine, pito, "woba ada anaa" other alcoholic bitters, fermented cider or any other drink within the past 7 days? Yes =1 No =2;		
28. When you drink alcohol, on average, how many drinks do you have during one day? <u>Please note that one drink is :</u> 120 ml of wine (½ medium glass of dry wine) or 285 ml of beer (half of large beer bottle, one full mini) or One bottle of Guinness or 30ml (one tot) of spirit, whisky, gin, akpeteshie, "woba ada anaa", other alcoholic bitters or 60ml of brandy, vermouth, aperitif etc_____	___ ___ ___ ___ ___ ___ Number of drinks	LSTYL28

29. Do you do any vigorous-intensity sports, fitness or recreational (leisure) activities that cause large increases in breathing or heart rate like [running or football] for at least 10 minutes continuously? Yes =1 No =2 <u>If No, go to Q. 32</u>	_____	LSTYL 29
30. In a typical week, on how many days do you do vigorous-intensity sports, fitness or recreational (leisure) activities?	___ ___ Number	LSTYL 30
31. How much time do you spend doing vigorous-intensity sports, fitness or recreational activities on a typical day?	__ __ __ __ Hrs Min	LSTYL 31
32. Do you do any moderate-intensity sports, fitness or recreational (leisure) activities that causes a small increase in breathing or heart rate such as brisk walking, (cycling, swimming, volleyball) for at least 10 minutes continuously? Yes =1 No =2	_____	LSTYL 32
33. In a typical week, on how many days do you do moderate-intensity sports, fitness or recreational (leisure) activities?	___ ___ Number	LSTYL 33
34. How much time do you spend doing moderate-intensity sports, fitness or recreational (leisure) activities on a typical day?	__ __ __ __ Hrs Min	LSTYL 34

APPENDIX V: Follow-Up Baseline Questionnaire

DEPARTMENT OF NUTRITION AND DIETETICS, SCHOOL OF BIOMEDICAL AND ALLIED HEALTH SCIENCES

COLLEGE OF HEALTH SCIENCES.

FOLLOW-UP BASELINE QUESTIONNAIRE

EFFECTIVENESS OF DIETARY AND LIFESTYLE INTERVENTION IN PERSONS WITH MAJOR LIFESTYLE RELATED CHRONIC DISEASES AT THE KORLE-BU TEACHING HOSPITAL.

DEMOGRAPHIC AND SOCIOECONOMIC INFORMATION

Participant Id Number _____
Date of recruitment …….

1a	Consent has been obtained Yes=1 No=2 If no, do not proceed ……..	----------

2	Name of Participant	
3a	Contact phone number if available:	
3b	Specify whose phone	
4	Where do you stay?	

5. Gender (Record Male / Female as observed) Male=1; Female=2	_____

6. Age	Years ___ ___

7.	What is your ethnic background	
8a.	What is your religious affiliation?	
8b.	Marital status	
9.	What is the highest level of education you have completed?	
10.	Are you employed? **Yes =1: No = 2**	
10 b	If Yes, what work do you do?	

CLINICAL INFORMATION

11. Condition for which patient was referred	
12. Date of referral by doctor to see the dietitian	
13. Date of reporting to dietitian	
14. Reason for delay if dates for 12 and13 are different	
15. Date of receiving dietary counseling by the dietitian	
16. Reason for delay if dates 13 and 15 are different	

18. Does any relative of yours have your condition? Yes = 1; No = 2; Don't know = 3		
18b. If **yes** please specify	**Relative**	**Disease**
19a. In which year were you first told you have this condition?		
19b. How long have you had this condition (years: months)		
20. Do you have any other medical conditions? Yes = 1; No = 2; Don't know = 3 If No go to Q. 22		
21. If yes, please tell me the CONDITION	Duration YEARS	 MONTHS
21a. Are you on any medication? YES=1 NO = 2		
IF yes please tell me the names of your medication.		
22. Have you seen a dietitian before in the past? Yes =1: No = 2		
23. If Yes why, if No why?		

ANTHROPOMETRIC AND MEDICAL INFORMATION

Measurement	First Reading	Second Reading	Unit
Anthropometry			
Weight			
Height			
Waist circumference			
Hip circumference			
Blood Pressure	First Reading	Second Reading	Unit
Systolic blood pressure			
Diastolic blood pressure			

Body Composition	Reading	Unit
Percentage body fat		
Visceral fat		
Skeletal muscle		
Body water		

LIFESTYLE INFORMATION

24. Do you currently smoke any tobacco products, such as cigarettes, cigars or pipes? Yes =1 No =2; If No, go to Q. 28	_____	LSTYL19
25. If Yes, Do you currently smoke tobacco products daily? Yes =1 No =2;	_____	LSTYL20
26. How old were you when you first started smoking daily? If cannot remember, write 777 in the spaces	___ ___ ___ Age in years	LSTYL 21
27. On average, how many of the following do you smoke each day? (PLEASE RECORD EACH TYPE) Manufactured cigarettes	___ ___ ___ ___ ___ ___	LSTYL 22a

Hand-rolled cigarettes Pipes full of tobacco Other If other, please specify type(s) _____	___ ___ ___ ___ ___ ___ ___ ___ __	LSTYL 22b LSTYL 22c LSTYL 22d LSTYL 22e

28. In the past, did you ever smoke daily? Yes =1 No =2; <u>If No, go to Q. 30</u>	_____	LSTYL 23
29. If Yes, how old were you when you stopped smoking daily?	___ ___ ___ (Age in years)	LSTYL 24
30. Have you consumed alcohol such as beer, wine, brandy, aperitif, spirits, akpeteshie, palm wine, pito, "woba ada anaa" other alcoholic bitters, fermented cider or any other drink within the past 12 months? Yes =1 No =2; <u>If No, go to Q. 34a</u>	_____	LSTYL 25
31. In the past 12 months, how frequently have you had at least one drink? _____	_____	LSTYL26
32. Have you consumed alcohol (such as beer, wine, spirits, akpeteshie, palm wine, pito, "woba ada anaa" other alcoholic bitters, fermented cider or any other drink within the past 7 days? Yes =1 No =2;		LSTYL27
33. When you drink alcohol, on average, how many drinks do you have during one day? <u>Please note that one drink is :</u> 120 ml of wine (½ medium glass of dry wine) or 285 ml of beer (half of large beer bottle, one full mini) or One bottle of Guinness or 30ml (one tot) of spirit, whisky, gin, akpeteshie, "woba ada anaa", other alcoholic bitters or 60ml of brandy, vermouth, aperitif etc_____	___ ___ ___ ___ ___ ___ Number of drinks	LSTYL28

34a .Is exercises a part of your usual routine? Yes =1 No =2		
34b. Why?		
35. Do you do any vigorous-intensity sports, fitness or recreational (leisure) activities that cause large increases in breathing or heart rate like [running or football,] for at least 10 minutes continuously? Yes =1 No =2 If No, go to Q. 36	_____	LSTYL 29
34. In a typical week, on how many days do you do vigorous-intensity sports, fitness or recreational (leisure) activities?	___ ___ Number	LSTYL 30
35. How much time do you spend doing vigorous-intensity sports, fitness or recreational activities on a typical day?	__ __ __ __ Hrs Min	LSTYL 31
36. Do you do any moderate-intensity sports, fitness or recreational (leisure) activities that causes a small increase in breathing or heart rate such as brisk walking, (cycling, swimming, volleyball) for at least 10 minutes continuously? Yes =1 No =2	_____	LSTYL 32
37. In a typical week, on how many days do you do moderate-intensity sports, fitness or recreational (leisure) activities?	___ ___ Number	LSTYL 33
38. How much time do you spend doing moderate-intensity sports, fitness or recreational (leisure) activities on a typical day?	___ __ __ Hrs Min	LSTYL 34

INTERVENTIONS GIVEN

1	Daily energy prescribed	
2	Type of diet given	
3	Advice on alcohol intake	
4	Advice on smoking	

5	Advice on physical activity	
6	Advice on fruit and vegetable intake	

APPENDIX VI: Follow-Up Questionnaire

DEPARTMENT OF NUTRITION AND DIETETICS, SCHOOL OF BIOMEDICAL AND ALLIED HEALTH SCIENCES

COLLEGE OF HEALTH SCIENCES.

FOLLOW-UP UESTIONNAIRE

EFFECTIVENESS OF DIETARY AND LIFESTYLE INTERVENTION IN PERSONS WITH MAJOR LIFESTYLE RELATED CHRONIC DISEASES AT THE KORLE-BU TEACHING HOSPITAL

	Participant Id Number └ ┴ ┴ ┴ ┴ ┴ ┘
1	Name of Participant
2	Date of review…… Stage of follow-up……

2	Total number of reviews since first visit	_____
3	Total number of reviews patient should have attended.	
4	Did patient miss any review appointment/ **YES = 1 NO = 2**	
5	If Yes, how many appointments were missed?	
6	What were the reasons for missing appointments?	a…… …… b……

		……
7	How many meals do you take in a day?	
8	How many snacks do you take in a day?	
9	What time do you take the following in a typical day? a. Morning meal b. Snack c. Afternoon meal d. Snack e. Evening meal f. other	 ……. ……. ……. ……. …….. ……
10	How many times do you Eat fruits in a day?	
11	Are you on any medication?	
12	Do you take your medication as prescribed by the doctor? **YES =1 NO = 2**	
13	If No, what is your reason for this?	
14	Please tell me the names of your medication	

ANTHROPOMETRY, BLOOD PRESSURE AND BODY COMPOSITION INFORMATION

Measurement	**First Reading**	**Second Reading**	**Unit**
Anthropometry			
Weight			
Height			
Waist circumference			
Hip circumference			

Blood Pressure	First Reading	Second Reading	Unit
Systolic blood pressure			
Diastolic blood pressure			

Body Composition	Reading	Unit
Percentage body fat		
Visceral fat		
Skeletal muscle		
Body water		

LIFESTYLE INFORMATION

24. Do you currently smoke any tobacco products, such as cigarettes, cigars or pipes? Yes =1 No =2; If No, go to Q. 28	_____	LSTYL19
25. If Yes, Do you currently smoke tobacco products daily? Yes =1 No =2;	_____	LSTYL20
26. How old were you when you first started smoking daily? If cannot remember, write 777 in the spaces	___ ___ ___ Age in years	LSTYL 21
27. On average, how many of the following do you smoke each day? (PLEASE RECORD EACH TYPE) Manufactured cigarettes Hand-rolled cigarettes Pipes full of tobacco Other If other, please specify type(s) _____	___ ___ ___ ___ ___ ___ ___ ___ ___ ___ ___ ___ ___ ___ __	LSTYL 22a LSTYL 22b LSTYL 22c LSTYL 22d LSTYL 22e

28. In the past, did you ever smoke daily? Yes =1 No =2; If No, go to Q. 30	_____	LSTYL 23
29. If Yes, how old were you when you stopped smoking daily?	___ ___ ___	LSTYL 24

	(Age in years)	
30. Have you consumed alcohol such as beer, wine, brandy, aperitif, spirits, akpeteshie, palm wine, pito, "woba ada anaa" other alcoholic bitters, fermented cider or any other drink within the past 3 months? Yes =1 No =2; <u>If No, go to Q. 34a</u>	_____	LSTYL 25
31. In the past 3 months, how frequently have you had at least one drink? _____	_____	LSTYL26
32. Have you consumed alcohol (such as beer, wine, spirits, akpeteshie, palm wine, pito, "woba ada anaa" other alcoholic bitters, fermented cider or any other drink within the past 7 days? Yes =1 No =2;		LSTYL27
33. When you drink alcohol, on average, how many drinks do you have during one day? <u>Please note that one drink is :</u> 120 ml of wine (½ medium glass of dry wine) or 285 ml of beer (half of large beer bottle, one full mini) or One bottle of Guinness or 30ml (one tot) of spirit, whisky, gin, akpeteshie, "woba ada anaa", other alcoholic bitters or 60ml of brandy, vermouth, aperitif etc_____	___ ___ ___ ___ ___ ___ Number of drinks	LSTYL28
34a .Is exercises a part of your usual routine? Yes =1 No =2		
34b. Why?		
35. Do you do any vigorous-intensity sports, fitness or recreational (leisure) activities that cause large increases in breathing or heart rate like [running or football,] for at least 10 minutes continuously? Yes =1 No =2 <u>If No, go to Q. 36</u>	_____	LSTYL 29

34. In a typical week, on how many days do you do vigorous-intensity sports, fitness or recreational (leisure) activities?	___ ___ Number	LSTYL 30
35. How much time do you spend doing vigorous-intensity sports, fitness or recreational activities on a typical day?	__ __ __ __ Hrs Min	LSTYL 31
36. Do you do any moderate-intensity sports, fitness or recreational (leisure) activities that causes a small increase in breathing or heart rate such as brisk walking, (cycling, swimming, volleyball) for at least 10 minutes continuously? Yes =1 No =2	_____	LSTYL 32
37. In a typical week, on how many days do you do moderate-intensity sports, fitness or recreational (leisure) activities?	___ ___ Number	LSTYL 33
38. How much time do you spend doing moderate-intensity sports, fitness or recreational (leisure) activities on a typical day?	___ __ __ Hrs Min	LSTYL 34

APPENDIX VII: Food Frequency Questionnaire and 24-Hour Diet Recall Interview Guide

24 –HOUR DIET RECALL GUIDE (WEEKDAY)

Time	Type Of Food	Portion Size (Use Handy Measure)	Weight (g)

24 –HOUR DIET RECALL GUIDE (WEEK-END DAY)

Time	Type Of Food	Portion Size (Use Handy Measure)	Weight (g)

FOOD FREQUENCY QUESTIONNAIRE

I am going to read out a list of various foods. Please tell me how many times you eat them on average every week.

HIGH FIBER STARCHES	**Number of times per week**	**Never**
Oats		
Tom brown / Weanimix/ pokponsu		
Wheat bread / wheat		
Kenkey		
Others		
LOW FIBER STARCHES		
Banku		
Fufu		
Konkonte		
Plain rice / rice balls		
Waakye		
Sugar Bread		
Tea bread		
Butter bread		
Boiled plantain		
boiled ripe plantain		
Boiled yam		
Gari		
Housa koko		
Tuo zaafi		
Others		
FATTY FOODS	**Number of times per week**	**Never**
Fried yam		
Fried ripe plantain with beans and oil		
Jollof rice		

Fried rice		
Fried egg		
Fried fish		
Fried chicken without skin		
Fried meat		
Cream milk		
Groundnut soup		
Palm soup		
Cheese		
Butter/margarine		
Meat pie / doughnut / rock buns		
Others		
FOODS HIGH IN SATURATED FATS AND CHOLESTEROL		
Fried chicken with skin		
Grilled / boiled chicken with skin		
Domedo		
Crabs		
Offals		
Shrimps		
Others		
FOODS LOW IN SATURATED FATS AND CHOLESTEROL	**Number of times per week**	**Never**
Grilled / boiled chicken without skin		
Smoked / grilled fish		
Boiled egg		
Cooked lean meat		

Skimmed milk		
Light soup		
Others		
NUTS AND SEEDS		
Cooked beans		
Roasted Groundnut		
Roasted cashew nut		
Agushie		
Others		
HIGH SODIUM FOODS		
Koobi		
Kako		
Salted pig feet		
Toolo beef		
Momoni		
Sardine		
Corned beef		
Maggi		
Jumbo		
Royco		
Onga		
HIGH SODIUM FOODS	**Number of times per week**	**Never**
Dzomi		
Tinned vegetable salad		
Tinapa		
Others		
FRUITS		

Pawpaw		
Orange		
Banana		
Mango		
Water melon		
Pineapple		
Apple		
Others		
VEGETABLES		
Cabbage		
Carrot		
Cucumber		
Fresh tomatoes		
Kontomire/ayoyo/alefu		
Garden eggs		
Okro		
Others		
FOODS HIGH IN REFINED SUGAR	**Number of times per week**	**Never**
Coca cola		
Biscuits		
Cakes		
Sprite		
Fanta		
Sugar cane		
Don simon / ceres / minute maid etc		
Malt		
others		

ALCOHOLIC BEVERAGES		
Beer		
Guinness		
Spirit		
Wine		
Punch		
Others		

APPENDIX VIII: Conditions for Which Patients were Referred to See the Dietitian and Their Co-Morbidities. Frequency (%)

Diagnosed condition	**Male**	**Female**	**Total**
Type 2 diabetes mellitus	34 (10.0)	49 (14.5)	83 (24.5)
Hypertension and dyslipidemia	18 (5.3)	52 (15.3)	70 (20.6)
Type 2 diabetes mellitus and hypertension	13 (3.8)	27 (8.0)	40 (11.8)
Dyslipidemia	13 (3.8)	26 (7.7)	39 (11.5)
Hypertension	10 (2.9)	21 (6.2)	31 (9.1)
Obesity	2 (0.6)	30 (8.8)	32 (9.4)
Type 2 diabetes mellitus, hypertension and dyslipidemia	6 (1.8)	10 (2.9)	16 (4.7)
Others[a]	8 (2.3)	20 (6.0)	28 (8.3)
Total	**104 (30.7)**	**235 (69.3)**	**339 (100.0)**
Presence of co-morbidity			
Yes	38 (11.2)	100 (29.5)	138 (40.7)
No	60 (17.7)	115 (33.9)	175 ((51.6)
Don't know	6 (1.8.)	20 (5.9)	26 (7.7)
Total	**104 (30.7)**	**235 (69.3)**	**339 (100.0)**
Comorbidities of patients			
Arthritic pains	2 (0.6)	16 (4.7)	18 (5.3)
Benign prostate hyperplasia/ enlarged prostate/ pains in the testes	5 (1.5)	0	5 (1.5)
Infertility	0	12 (3.5)	12 (3.5)
Asthma	1 (0.3)	4 (1.2)	5 (1.5)
Enlarged heart	1 (0.3)	2 (0.6)	3 (0.9)
Eye problems, cataract, glaucoma, diabetes retinopathy	3 (0.9)	7 (2.1)	10 (2.9)
Cervical or thoracolumbar spondylosis	1 (0.3)	2 (0.6)	3 (0.9)
Type 2 diabetes mellitus, impaired fasting glucose	2 (0.6)	4 (1.2)	6 (1.8)
Dyslipidemia			
Gastritis, reflux oesophagitis	1 (0.3)	2 (0.6)	3 (0.9)
Hypertension	2 (0.6)	1 (0.3)	3 (0.9)
Migraines	14 (4.1)	30 (8.8)	44 (13.0)
Ulcers	0	2 (0.6)	2 (0.6)
Sickle cell anaemia	1 (0.3)	7 (2.1)	8 (2.4)
HIV	1 (0.3)	1 (0.3)	2 (0.6)
Others [b]	2 (0.6)	1 (0.3)	3 (0.9)
Total	2 (0.6)	9 (2.7)	11 (3.2)
	38 (11.2)	**100 (29.5)**	**138 (40.7)**

Others[a]: Hypertension together with type 2 diabetes mellitus and Obesity = 8 (2.4), Hyperuricemia = 2 (0.6), Kidney related conditions (Acute renal failure / polycystic kidney / chronic kidney disease / impaired renal function) = 10 (2.9) Spondylosis = 3 (0.9), diseases of the liver (Chronic liver disease/ Hepatitis B/ Cirrhosis/ Fatty liver) = 4 (1.2) Stroke = 1 (0.3).

Others[b]: stroke, oedema, ear problem, thyroid malfunction, hepatitis B, jaundice

APPENDIX IX: FREQUENCY OF INTAKE OF FOODS FROM THE VARIOUS FOOD GROUPS AT BASELINE COMPARED TO THREE AND SIX MONTHS

APPENDIX IXa: Frequency of intake of foods made from whole grains at baseline 3 months and 6 months follow-up

Foods made from whole grains	Never 0x/wk N (%)	Low (1x/wk) N (%)	Moderate (≥2x/wk) N (%)	P-value
Oats				0.566
Baseline (N = 132)	103 (78.0)	12 (9.1)	17 (12.8)	
3 Months (N = 80)	52 (65.0)	6 (7.5)	22 (27.6)	
6 Months (N = 60)	26 (43.3)	13 (21.7)	21 (35.0)	
Wheat				0.840
Baseline	74 (56.1)	7 (5.3)	51 (38.6)	
3 Months	29 (36.3)	4 (5.0)	47 (58.8)	
6 Months	14 (23.3)	6 (10.0)	40 (66.6)	
Tom brown				0.077
Baseline	106 (80.3)	14 (10.6)	12 (9.1)	
3 Months	41 (51.3)	9 (11.3)	30 (37.5)	
6 Months	30 (50.0)	10 (16.7)	20 (33.4)	
Kenkey				0.209
Baseline	49 (37.1)	16 (12.1)	67 (50.8)	
3 Months	29 (36.3)	13 (16.3)	38 (47.6)	
6 Months	13 (21.7)	17 (28.3)	30 (45.0)	
Banku				0.310
Baseline	33 (25.0)	21 (15.9)	78 (59.1)	
3 Months	15 (18.8)	16 (20.0)	49 (62.3)	
6 Months	8 (13.3)	13 (21.7)	39 (55.3)	
Waakye				
Baseline	83 (62.9)	22 (16.7)	27 (20.4)	0.394
3 Months	35 (43.8)	14 (17.5)	31 (33.8)	
6 Months	24 (40.0)	15 (25.0)	21 (35.0)	

APPENDIX IXb: Frequency of intake of starches at baseline three months and 6 months follow-up. Frequency (%)

Starches	Never 0x/wk N (%)	Low (1x/wk) N (%)	Moderate (≥2x/wk) N (%)	P-value
Konkonte				
Baseline (N =132)	121 (91.7)	5 (3.8)	6 (4.5)	P = 0.107
3 Months (N= 80)	70 (87.5)	9 (11.3)	1 (1.3)	
6 Months (N = 60)	52 (86.7)	4 (6.7)	4 (6.7)	
Plain rice / rice balls				
Baseline	23 (17.4)	13 (9.8)	96 (72.7)	P = 0.110
3 Months	19 (23.8)	3 (3.8)	58 (72.5)	
6 Months	6 (10.0)	6 (10.0)	48 (80)	
Sugar Bread				
Baseline	112 (84.8)	4 (3.0)	16 (12.1)	P = 0.071
3 Months	66 (82.5)	8 (10.0)	6 (7.5)	
6 Months	55 (91.7)	3 (5.0)	2 (3.3)	
Tea bread				
Baseline	70 (53.0)	15 (11.4)	47 (35.6)	P = 0.663
3 Months	42 (52.5)	10 (12.5)	28 (35.0)	
6 Months	24 (40.0)	12 (20.0)	24 (40.0)	
Butter bread				
Baseline	89 (67.4)	9 (6.8)	34 (25.7)	P = 0.002*
3 Months	57 (71.3)	11 (13.8)	12 (15.1)	
6 Months	39 (65.0)	13 (21.7)	8 (13.3)	
Boiled plantain				
Baseline	59 (44.7)	19 (14.4)	54 (40.9)	P = 0.562
3 Months	32 (40.0)	13 (16.3)	35 (43.8)	
6 Months	17 (28.3)	17 (28.3)	26 (43.3)	
Boiled ripe plantain				
Baseline	114 (86.4)	8 (6.1)	10 (7.6)	P = 0.230
3 Months	63 (78.8)	13 (16.3)	4 (5.0)	
6 Months	45 (75.0)	11 (18.3)	4 (6.7)	
Boiled yam				
Baseline	58 (43.9)	24 (18.2)	50 (37.8)	P = 0.454
3 Months	31 (38.8)	15 (18.8)	34 (42.5)	
6 Months	18 (30.0)	19 (31.7)	23 (38.3)	
Gari				
Baseline	116 (87.9)	5 (3.8)	11 (8.3)	P = 0.258
3 Months	64 (80.0)	8 (10.0)	8 (10.0)	
6 Months	49 (81.7)	8 (13.3)	3 (5.0)	
Housa koko				
Baseline	72 (54.5)	13 (9.8)	47 (35.6)	P = 0.026*
3 Months	59 (73.8)	6 (7.5)	15 (18.8)	
6 Months	43 (71.7)	8 (13.3)	9 (15.0)	
Tuo zaafi				
Baseline	114 (86.4)	11 (8.3)	7 (5.3)	P = 0.632
3 Months	63 (78.8)	9 (11.3)	8 (10.0)	
6 Months	48 (80.0)	5 (8.3)	7 (11.7)	
Fufu				
Baseline	54 (40.9)	38 (28.8)	40 (30.3)	P = 0.546
3 Months	32 (40.0)	27 (33.8)	21 (26.3)	
6 Months	19 (31.7)	22 (36.7)	19 (31.7)	

APPENDIX IXc: Frequency of intake of fruits at baseline three months and 6 months follow-up. Frequency (%)

Fruits	Never 0x/wk N (%)	Low (1x/wk) N (%)	Moderate -High (≥2x/wk) N (%)	P-value*
Pawpaw				
Baseline (N=132)	59 (44.7)	21 (15.9)	52 (39.4)	P = 0.219
3 Months (N =80)	22 (27.5)	18 (22.5)	40 (50.0)	
6 Months (N= 60)	11 (18.3)	12 (20.0)	37 (61.7)	
Orange				
Baseline	59 (44.7)	20 (15.2)	53 (40.2)	P = 0.721
3 Months	19 (23.8)	18 (22.5)	43 (53.8)	
6 Months	13 (21.7)	14 (23.3)	33 (55.0)	
Banana				
Baseline	59 (44.7)	18 (13.6	55 (41.7)	P = 0.310
3 Months	23 (28.8)	13 16.3)	44 (55.0)	
6 Months	11 (18.3)	17 (28.3)	32 (53.3)	
Mango				
Baseline	107 (81.1)	10 (7.6)	15 (11.4)	P = 0.393
3 Months	44 (55.0)	15 (18.8)	21 (26.3)	
6 Month	29 (48.3)	15 (25.0)	16 (26.7)	
Water Melon				
Baseline	57 (43.2)	18 (13.6)	57 (43.2)	P = 0.665
3 Months	32 (40.0)	14 (17.5)	3 (42.5)	
6 Months	14 (23.3)	11 (18.3)	35 (58.3)	
Pineapple				
Baseline	89 (67.4)	18 (13.6)	25 (19.0)	P = 0.841
3 Months	48 (60.0)	10 (12.5)	22 (27.5)	
6 Months	32 (53.3)	10 (16.7)	18 (30.0)	
Apple				
Baseline	77 (58.3)	24 (18.2)	31 (23.5)	P = 0.660
3 Months	28 (35.0)	20 (25.0)	32 (40.0)	
6 Months	16 (26.7)	22 (36.7)	22 (36.7)	

*: Significant at $P \leq 0.05$: Pearson's Chi Square

APPENDIX IXd: Frequency of intake of vegetables at baseline three months and 6 months follow-up. Frequency (%)

Vegetables	Never 0x/wk N (%)	Low (1x/wk) N (%)	Moderate - High (≥2x/wk) N (%)	P-value
Cabbage				
Baseline (N=132)	61 (46.2)	30 (22.7)	41 (31.1)	P = 0.008*
3 Months (N=80)	23 (28.8)	10 (12.5)	47 (58.8)	
6 Months (N=60)	14 (23.3)	10 (16.7)	36 (60.0)	
Carrot				
Baseline	58 (43.9)	28 (21.1)	46 (43.8)	P = 0.005*
3 Months	24 (30.0)	7 (8.8)	49 (61.3)	
6 Months	7 (11.7)	15 (25.0)	38 (63.3)	
Cucumber				
Baseline	81 (61.4)	21 (15.9)	30 (22.7)	P = 0.132
3 Months	31 (38.8)	12 (15.0)	37 (46.3)	
6 Months	21 (35.0)	15 (25.0)	24 (40.0)	
Fresh tomatoes				
Baseline	46 (34.8)	3 (2.3)	83 (62.9)	P = 0.091
3 Months	19 (23.8)	1 (1.3)	60 (75.0)	
6 Months	2 (3.3)	3 (5.0)	55 (91.7)	
Kontomire				
Baseline	46 (34.8)	27 (20.5)	59 (44.7)	P = 0.635
3 Months	24 (30.0)	16 (20.0)	40 (50.0)	
6 Months	8 (13.3)	12 (20.0)	40 (66.7)	
Garden egg				
Baseline	40 (30.3)	28 (21.2)	64 (48.5)	P = 0.192
3 Months	18 (22.5)	17 (21.3)	45 (56.3)	
6 Months	8 (13.3)	13 (21.7)	39 (65.0)	
Okra				
Baseline	62 (47.0)	31 (23.5)	39 (29.5)	P = 0.616
3 Months	29 (36.3)	17 (21.3)	34 (42.5.)	
6 Months	13 (21.7)	15 (25.0)	32 (53.3)	

*: Significant at P ≤ 0.05: Pearson's Chi Square

APPENDIX IXe: Frequency of intake of fatty foods at baseline three months and 6 months follow-up. Frequency (%)

Fatty foods	Never 0x/wk N (%)	Low (1x/wk) N (%)	Moderate - High (≥2x/wk) N (%)	P-value
Fried yam				
Baseline (N=132)	101 (76.5)	14 (10.6)	17 (12.9)	P = 0.256
3 Months (N=80)	64 (80.0)	9 (11.3)	7 (8.8)	
6 Months (N=60)	47 (78.3)	8 (13.3)	5 (8.3)	
Red red				
Baseline	108 (81.8)	17 (12.9)	7 (5.3)	P = 0.469
3 Months	66 (82.5)	8 (10.0)	6 (7.5)	
6 Months	40 (66.7)	14 (23.3)	6 (10.0)	
Jollof rice				
Baseline	63 (47.7)	34 (25.8)	35 (26.5)	P = 0.161
3 Months	44 (55.0)	17 (21.3)	19 (23.8)	
6 Months	21 (35.0)	23 (38.3)	16 (26.7)	
Fried rice				
Baseline	122 (92.4)	7 (5.3)	3 (2.3)	P = 0.392
3 Months	75 (93.8)	4 (5.0)	1 (1.3)	
6 Months	55 (91.7)	5 (8.3)	0 (0)	
Fried egg				
Baseline	109 (82.6)	8 (6.1)	15 (11.4)	P = 0.056
3 Months	61 (76.3)	13 (16.3)	6 (7.5)	
6 Months	44 (73.3)	12 (20.0)	4 (6.7)	
Fried fish				
Baseline	37 (28.0)	14 (10.6)	81 (61.4)	P = 0.872
3 Months	28 (35.0)	10 (12.5)	42 (52.5)	
6 Months	14 (23.3)	10 (16.7)	36 (60.0)	
Fried chicken without skin				
Baseline	106 (80.3)	6 (4.5)	20 (15.2)	P = 0.060
3 Months	64 (80.0)	7 (8.8)	9 (11.3)	
6 Months	41 (68.3)	12 (20.0)	7 (11.7)	
Fried meat				
Baseline	112 (84.8)	7 (5.3)	13 (9.8)	P = 0.044*
3 Months	67 (83.8)	5 (6.3)	8 (10.0)	
6 Months	43 (71.7)	10 (16.7)	7 (11.7)	
Cream milk				
Baseline	87 (65.9)	8 (6.1)	37 (28.0)	P = 0.101
3 Months	53 (66.3)	10 (12.5)	17 (21.3)	
6 Months	40 (66.7)	6 (10.0)	14 (23.3)	
Groundnut soup				
Baseline	89 (67.4)	18 (13.6)	25 (18.9)	P = 0.079
3 Months	50 (62.5)	18 (22.5)	12 (15.0)	
6 Months	34 (56.7)	19 (31.7)	1 (11.7)	

APPENDIX IXe: Frequency of intake of fatty foods at baseline three months and 6 months follow-up cont. Frequency (%)

Fatty foods	Never 0x/wk N (%)	Low (1x/wk) N (%)	Moderate - High (≥2x/wk) N (%)	P-value
Palm soup				
Baseline (N=132)	72 (54.5)	25 (18.9)	35 (26.5)	P = 0.332
3 Months (N=80)	39 (48.8)	23 (28.8)	18 (22.5)	
6 Months (N=60)	28 (46.7)	16 (36.7)	16 (26.7)	
Butter/margarine				
Baseline	123 (93.2)	1 (0.8)	8 (6.1)	P = 0.144
3 Months	77 (96.3)	2 (2.5)	1 (1.3)	
6 Months	60 (100)	0 (0)	0 (0)	
Pastries				
Baseline	105 (79.5)	13 (9.8)	14 (10.6)	P = 0.546
3 Months	69 (86.3)	7 (8.8)	4 (5.0)	
6 Months	5 (85.0)	6 (10.0)	3 (5.0)	
Roasted groundnut				
Baseline	90 (68.2)	22 (16.7)	20 (15.2)	P = 0.393
3 Months	56 (70.0)	16 (20.0)	8 (10.0)	
6 Months	36 (60.0)	15 (25.0)	9 (15.0)	
Roasted cashew nut				
Baseline	127 (96.2)	1 (0.8)	4 (3.0)	P = 0.131
3 Months	75 (93.8)	2 (2.5)	3 (3.8)	
6 Months	53 (88.3)	6 (10.0)	1 (1.7)	
Fried chicken with skin				
Baseline	110 (83.3)	8 (6.1)	14 (10.6)	P = 0.612
3 Months	79 (95.0)	2 (2.5)	2 (2.5)	
6 Months	54 (90.0)	4 (6.7)	2 (3.3)	
Grilled / boiled chicken with skin				
Baseline	110 (83.3)	8 (6.1)	14 (10.6)	P = 0.097
3 Months	73 (91.3)	6 (7.5)	1 (1.3)	
6 Months	48 (80.0)	8 (13.3)	4 (6.7)	
Domedo				
Baseline	120 (90.9)	6 (4.5)	6 (4.5)	P = 0.935
3 Months	77 (96.3)	2 (2.5)	1 (1.3)	
6 Months	52 (86.7)	5 (8.3)	3 (5.0)	
Crabs				
Baseline	91 (68.9)	25 (18.9)	16 (12.1)	P = 0.906
3 Months	67 (83.8)	8 (10.0)	5 (6.3)	
6 Months	46 (76.7)	9 (15.0)	5 (8.3)	
Offals				
Baseline	112 (84.8)	10 (7.6)	10 (7.6)	P = 0.054*
3 Months	70 (87.5)	2 (2.5)	8 (10.0)	
6 Months	49 (81.7)	8 (13.3)	3 (5.0)	
Shrimps				
Baseline	91 (68.9)	18 (13.6)	23 (17.4)	P = 0.178
3 Months	64 (80.0)	5 (6.3)	11 (13.8)	
6 Months	40 (66.7)	9 (15.0)	11 (18.3)	

*; Significant at P ≤ 0.05: Pearson's Chi Square.

APPENDIX IXf: Frequency of intake of lean foods at baseline three months and 6 months follow-up. Frequency (%)

Lean foods	Never 0x/wk N (%)	Low (1x/wk) N (%)	Moderate - High (≥2x/wk) N (%)	P-value*
Grilled chicken without skin				
Baseline (N=132)	110 (83.3)	10 (7.6)	12 (9.1)	P = 0.641
3 Months (N=80)	64 (80.0)	8 (10.0)	8 (10.0)	
6 Months (N=60)	42 (70.0)	11 (18.3)	7 (11.7)	
Smoked/grilled fish				
Baseline	23 (17.4)	15 (11.4)	94 (71.2)	P = 0.145
3 Months	7 (8.8)	9 (11.3)	64 (80.0)	
6 Months	4 (6.7)	12 (20.0)	44 (73.3)	
Boiled egg				
Baseline	96 (72.7)	17 (12.9)	19 (14.4)	P = 0.212
3 Months	56 (70.0)	16 (20.0)	8 (10.0)	
6 Months	35 (58.3)	16 (26.7)	9 (15.0)	
Cooked lean meat				
Baseline	111 (84.1)	9 (6.8)	12 (9.1)	P = 0.513
3 Months	62 (77.5)	10 (12.5)	8 (10.0)	
6 Months	50 (83.3)	7 (11.7)	3 (5.0)	
Skimmed milk				
Baseline	96 (72.7)	7 (5.3)	29 (22.0)	P = 0.920
3 Months	52 (65.0)	5 (6.3)	23 (28.5)	
6 Months	38 (63.3)	6 (10.0)	16 (26.7)	
Cooked beans				
Baseline	93 (70.5)	20 (15.2)	19 (14.4)	P = 0.785
3 Months	55 (68.8)	12 (15.0)	13 (16.3)	
6 Months	29 (48.3)	19 (31.7)	12 (20.0)	
Agushie				
Baseline	87 (65.9)	27 (20.5)	18 (13.6)	P = 0.785
3 Months	53 (66.3)	13 (16.3)	14 (17.5)	
6 Months	30 (30.0)	15 (25.0)	15 (25.0)	

*: Significant at P ≤ 0.05: Pearson's Chi Square.

APPENDIX IXg: Frequency of intake of salted foods at baseline three months and 6 months follow-up. Frequency (%)

Salted foods	Never 0x/wk N (%)	Low (1x/wk) N (%)	Moderate - High (≥2x/wk) N (%)	P-value
Koobi				
Baseline (N=132)	92 (69.7)	22 (16.7)	18 (13.6)	P =
3 Months (N=80)	68 (85.0)	7 (8.8)	17 (21.3)	0.423
6 Months (N=60)	46 (76.7)	11 (18.3)	17 (28.3)	
Kako				
Baseline	114 (86.4)	11 (8.3)	7 (5.3)	P =
3 Months	77 (96.3)	2 (2.5)	1 (1.3)	0.967
6 Months	56 (93.3)	3 (5.0)	1 (1.7)	
Salted pig feet				
Baseline	119 (90.2)	7 (5.3)	6 (4.5)	P =
3 Months	77 (96.3)	2 (2.5)	1 (1.3)	0.765
6 Months	51 (85.0)	7 (11.7)	2 (3.3)	
Toolo beef				
Baseline	109 (82.6)	15 (11.4)	8 (6.1)	P =
3 Months	89 (86.3)	9 (11.3)	2 (2.5)	0.776
6 Months	54 (90.0)	5 (8.3)	1 (1.7)	
Momoni				
Baseline	84 (63.6)	23 (17.4)	25 (18.9)	P =
3 Months	62 (77.5)	12 (15.0)	6 (7.5)	0.292
6 Months	47 (78.3)	8 (13.3)	5 (8.3)	
Sardine				
Baseline	108 (81.8)	14 (10.6)	10 (7.6)	P =
3 Months	67 (83.8)	10 (12.5)	3 (3.8)	0.538
6 Months	45 (75.0)	12 (20.0)	3 (5.0)	
Corned beef				
Baseline	122 (92.4)	7 (5.3)	3 (2.3)	P =
3 Months	70 (87.5)	10 (12.5)	0 (0)	0.186
6 Months	50 (83.3)	8 (13.3)	2 (3.3	
Maggi				
Baseline	51 (38.6)	15 (11.4)	66 (50.0)	P =
3 Months	51 (63.8)	9 (11.3)	20 (25.0)	0.133
6 Months	32 (53.3)	10 (16.7)	18 (30.0)	
Jumbo				
Baseline	128 (97.0)	0 (0)	4 (3.0)	P =
3 Months	76 (95.0)	1 (1.3)	3 (3.8)	0.091
6 Months	56 (93.3)	0 (0)	4 (6.7)	
Royco				
Baseline	121 (91.7)	0 (0)	11 (8.3)	P =
3 Months	73 (91.3)	1 (1.3)	6 (7.5)	0.275
6 Months	55 (91.7)	0 (0)	5 (8.3)	
Onga				
Baseline	116 (87.9)	5 (3.8)	11 (8.3)	P =
3 Months	70 (87.5)	5 (6.3)	5 (6.3)	0.653
6 Months	57 (95.0)	1 (1.7)	2 (3.3)	
Dzomi				
Baseline	49 (37.1)	21 (15.7)	62 (47.0)	P =
3 Months	31 (38.8)	13 (16.3)	36 (45.0)	0.869
6 Months	15 (25.)	12 (20.0)	31 (51.7)	

Frequency of intake of salted foods at baseline three months and 6 months follow-up Cont. Frequency (%)

Salted foods	Never 0x/wk N (%)	Low (1x/wk) N (%)	Moderate (≥2x/wk) N (%)	P-value
Tinapa				
Baseline	107 (81.1)	15 (11.4)	10 (7.6)	P = 0.803
3 Months	66 (82.5)	8 (10.0)	6 (7.5)	
6 Months	50 (83.3)	7 (11.7)	3 (5.0)	

APPENDIX IXi: Frequency of intake of sweetened foods at baseline three months and 6 months follow-up. Frequency (%)

Sweetened foods	Never	Low frequency	Moderate – High frequency	P Value
Coca cola				
Baseline (N=132)	114 (86.4)	4 (3.0)	14 (10.6)	P = 0.119
3 Months (N=80)	71 (88.8)	5 (6.3)	4 (5.0)	
6 Months (N=60)	51 (85.0)	6 (10.0)	3 (5.0)	
Biscuit				
Baseline	80 (60.6)	21 (15.9)	31 (23.5)	P = 0.006*
3 Months	50 (62.5)	10 (12.5)	20 (25.0)	
6 Months	39 (65.0)	10 (16.7)	11 (18.3)	
Cakes				
Baseline	126 (95.5)	4 (3.0)	2 (1.5)	P = 0.600
3 Months	76 (95.0)	1 (1.3)	3 (3.8)	
6 Months	59 (98.3)	1 (1.7)	0 (0)	
Sprite				
Baseline	116 (87.9)	11 (8.3)	5 (3.8)	P = 0.028*
3 Months	76 (95.0)	0 (0)	4 (5.0)	
6 Month	50 (83.3)	7 (11.7)	3 (5.0)	
Fanta				
Baseline	123 (93.2)	7 (5.3)	2 (1.5)	P = 0.378
3 Months	74 (92.5)	4 (5.0)	2 (2.5)	
6 Months	54 (90.0)	6 (10.0)	0 (0)	
Sugar cane				
Baseline	124 (93.9)	6 (4.5)	2 (1.5)	P = 0.769
3 Months	78 (97.5)	1 (1.3)	1 (1.3)	
6 Months	56 (93.3)	3 (5.0)	1 (1.7)	
Paper pack juices				
Baseline	108 (81.8)	12 (9.1)	12 (9.1)	P = 0.347
3 Months	64 (80.0)	12 (15.0)	4 (5.0)	
6 Months	46 (76.7)	9 (15.0)	5 (8.3)	
Malt				
Baseline	102 (77.3)	19 (14.4)	11 (8.3)	P = 0.699
3 Months	70 (87.5)	7 (8.8)	3 (3.8)	
6 Months	46 (76.7)	11 (18.3)	3 (5.0)	

*: Significant at P ≤ 0.05: Pearson's Chi Square.

APPENDIX IXj: Frequency of intake of alcoholic beverages at baseline three months and 6 months follow-up. Frequency (%)

Alcoholic beverages	Never	Low frequency	Moderate frequency	P value
Beer				
Baseline (N=132)	121 (91.7)	7 (5.3)	4 (3.0)	P = 0.303
3 Months (N=80)	74 (92.5)	3 (3.8)	3 (3.8)	
6 Months (N=60)	52 (86.7)	7 (11.7)	1 (1.7)	
Guinness				
Baseline	127 (96.2)	5 (3.8)	0 (0)	P = 0.200
3 Months	75 (93.8)	3 (3.8)	2 (2.5)	
6 Months	56 (93.3)	2 (3.3)	2 (3.3)	
Spirit				
Baseline	127 (96.2)	2 (1.5)	1 (0.8)	P = 0.620
3 Months	77 (96.3)	2 (2.5)	1 (1.3)	
6 Months	59 (98.3)	1 (1.7)	0 (0)	
Wine				
Baseline	124 (93.9)	6 (4.5)	2 (1.5)	P = 0.849
3 Months	77 (96.3)	2 (2.5)	1 (1.3)	
6 Month	54 (90.0)	5 (8.3)	1 (1.7)	

Baseline (N = 132), 3 Months (N = 80), 6 Months (N = 60). Low frequency = once a week, moderate frequency = 2-3 times a week, high frequency = 4 or more times a week

APPENDIX X: Test of Proportions of Patients Who Consumed Foods from the Various Food Groups at Baseline Compared to Three and Six Months

APPENDIX Xa: Chi Square test of proportions of patients who included whole grains in their usual diet baseline compared to 3 months and 6 months. Chi square value (p-value)

Foods made from whole grains	Baseline vs 3 Months	Baseline vs 6 months	3 months vs 6 months
Oats	1.164 (0.2807)	7.683 (0.0056)[a]	2.857 (0.0910)
Wheat	4.277 (0.0386) [a]	11.252 (0.0008) [a]	1.894 (0.1688)
Tom brown	5.570 (0.0183) [a]	5.456 (0.0195) [a]	0.010 (0.9218)
Kenkey	0.011 (0.9168)	3.271 (0.0705)	2.460 (0.1168)
Banku	0.888 (0.3461)	1.974 (0.1600)	0.274 (0.6007)
Waakye	3.441 (0.0636)	4.320 (0.0377) [a]	0.111 (0.7390)
Cooked beans	0.023 (0.8793)	3.516 (0.0608)	2.313 (0.1283)

[a] Significant at $P \leq 0.05$: Fisher's Exact Test

APPENDIX Xb: Chi Square test of proportions of patients who included starches in their usual diet at baseline compared to 3 months and 6 months. Chi square value (p-value)

Starches	Baseline vs 3 Months	Baseline vs 6 months	3 months vs 6 months
Kokonte	0.095 (0.7576)	0.118 (0.7316)	0.002 (0.9609)
Plain rice	0.977 (0.3229)	1.546 (0.2138)	3.731 (0.0534)[a]
Sugar bread	0.031 (0.8597)	0.154 (0.6952)	0.231 (0.6310)
Tea bread	0.002 (0.9614)	1.526 (0.2167)	1.146 (0.2845)
Butter bread	0.099 (0.7529)	0.036 (0.8496)	0.190 (0.6627)
Boiled plantain	0.259 (0.6108)	3.044 (0.0810)	1.359 (0.2436)
Boiled ripe plantain	0.351 (0.5534)	0.676 (0.4110)	0.060 (0.8071)
Gari	0.359 (0.5491)	0.193 (0.6602)	0.012 (0.9141)
Tuo zaafi	0.351 (0.5534)	0.210 (0.6464)	0.007 (0.9334)
Fufu	0.010 (0.9207)	0.960 (0.3272)	0.652 (0.4193)
Boiled yam	0.325 (0.5687)	2.160 (0.1417)	0.747 (0.3873)

[a] Significant at $P \leq 0.05$: Fisher's Exact Test

APPENDIX Xc: Chi Square test of proportions of patients who included fruits in their usual diet at baseline compared to 3 months and 6 months. Chi square value (p-value).

Fruits	Baseline vs 3 Months	Baseline vs 6 months	3 months vs 6 months
Pawpaw	4.067 (0.0437)[a]	9.019 (0.00027) [a]	1.247 (0.2642)
Orange	6.382 (0.0115) [a]	6.535 (0.0106) [a]	0.060 (0.8069)
Banana	3.462 (0.0628)	9.019 (0.0027) [a]	1.552 (0.2128)
Mango	4.387 (0.0362) [a]	6.269 (0.0123) [a]	0.295 (0.5870)
Watermelon	0.122 (0.7268)	4.882 (0.0271) [a]	2.987 (0.0839)
Apple	5.771 (0.0163) [a]	9.794 (0.0018) [a]	0.757 (0.3843)
Pineapple	0.432 (0.5112)	1.409 (0.2353)	0.269 (0.6042)

[a] Significant at $P < 0.05$: Fisher's Exact Test

APPENDIX Xd: Chi Square test of proportions of patients who included vegetables in their usual diet at baseline compared to 3 months and 6 months. Chi square value (p-value)

Vegetables	Baseline vs 3 Months	Baseline vs 6 months	3 months vs 6 months
Cabbage	4.061 (0.0439) [a]	6.213 (0.0127) [a]	0.379 (0.5379)
Carrot	2.596 (0.1071)	14.997 (0.0001) [a]	5.427 (0.0198) [a]
Cucumber	5.099 (0.0239) [a]	6.093 (0.0136) [a]	0.126 (0.7226)
Fresh tomatoes	2.072 (0.1500)	19.818 (0.0001) [a]	10.341 (0.0013) [a]
Kontomire	0.351 (0.5533)	7.599 (0.0058) [a]	4.347 (0.0371) [a]
Garden eggs	1.132 (0.2874)	5.203 (0.0225) [a]	1.587 (0.2078)
Okro	1.397 (0.2372)	7.670 (0.0056) [a]	2.460 (0.1168)

[a] Significant at P < 0.05: Fisher's Exact Test

APPENDIX Xe: Chi Square test of proportions of patients who included fatty foods in their usual diet at baseline compared to 3 months and 6 months. Chi square value (p-value)

Fatty foods	Baseline vs 3 Months	Baseline vs 6 months	3 months vs 6 months
Fried yam	0.073 (0.7870)	0.016 (0.8981)	0.012 (0.9122)
Fried ripe plantain with beans and palm oil	0.003 (0.9573)	1.294 (0.2553)	1.017 (0.3132)
Jollof rice	0.499 (0.4797)	1.623 (0.2026)	2.990 (0.0838)
Fried rice	0.008 (0.9292)	0.002 (0.9633)	0.013 (0.9082)
Fried egg	0.257 (0.6121)	0.476 (0.4904)	0.038 (0.8460)
Fried fish	0.772 (0.3796)	0.349 (0.5545)	1.590 (0.2073)
Fried chicken without skin	0.001 (0.9813)	0.830 (0.3624)	0.595 (0.4404)
Fried meat	0.007 (0.9333)	0.918 (0.3380)	0.577 (0.4474)
Cream milk	0.001 (0.9794)	0.004 (0.9502)	0.001 (0.9717)
Groundnut soup	0.185 (0.6672)	0.788 (0.3748)	0.191 (0.6617)

Palm soup	0.325 (0.5686)	0.503 (0.4782)	0.028 (0.8661)
Cheese	0	0.004 (0.9492)	0
Butter/margarine	0.033 (0.8566)	0	0
Pastries	0.226 (0.634)	0.128 (0.7202)	0.006 (0.9408)
Roasted Groundnut	0.023 (0.8802)	0.446 (0.5043)	0.516 (0.4723)
Roasted cashew nut	0.029 (0.8640)	0.217 (0.6416)	0.091 (0.7628)
Agushie	0.001 (0.9794)	1.864 (0.1722)	1.501 (0.2205)
Fried chicken with skin	0.351 (0.5334)	0.158 (0.6912)	0.073 (0.7865)
Grilled / boiled chicken with skin	0.254 (0.6144)	0.254 (0.6144)	0.393 (0.5305)
Domedo	0.085 (0.7705)	0.084 (0.7724)	0.187 (0.6653)
Crabs	1.063 (0.3024)	0.302 (0.5826)	0.199 (0.6553)
Offals	0.038 (0.8448)	0.048 (0.8259)	0.128 (0.7206)
Shrimps	0.691 (0.4057)	0.029 (0.8636)	0.769 (0.3806)

[a] Significant at P ≤ 0.05: Fisher's Exact Test. Jollof rice = refined rice cooked in oily tomatoes sauce mostly with meat. *Domedo* = grilled pork. Offals = mostly intestines and other internal organs of goat and other domestic animals.

APPENDIX Xf: Chi Square test of proportions of patients who included lean foods in their usual diet at baseline compared to 3 months and 6 months. Chi square value (p-value)

Lean foods	**Baseline vs 3 Months**	**Baseline vs 6 months**	**3 months vs 6 months**
Grilled/boiled chicken without skin	0.066 (0.7968)	0.974 (0.3238)	0.435 (0.5095)
Smoked / grilled fish	2.748 (0.0974)	3.546 (0.0597)	0.174 (0.6764)
Boiled egg	0.051 (0.8217)	1.357 (0.2441)	0.713 (0.3985)
Cooked lean meat	0.268 (0.6048)	0.003 (0.9556)	0.128 (0.7202)
Skimmed milk	0.432 (0.5109)	0.556 (0.4560)	0.015 (0.9019)
Cooked beans	0.023 (0.8793)	3.516 (0.0608)	2.313 (0.1283)
Agushie	0.001 (0.9794)	1.864 (0.1722)	1.501 (0.2205)

[a] Significant at P ≤ 0.05: Fisher's Exact Test

APPENDIX Xg: Chi Square test of proportions of patients who included salted foods in their usual diet at baseline compared to 3 months and 6 months. Chi square value (p-value)

Salted foods	Baseline vs 3 Months	Baseline vs 6 months	3 months vs 6 months
Koobi	1.081 (0.2985)	0.245 (0.6207)	0.273 (0.6013)
Kako	0.220 (0.6394)	0.137 (0.7108)	0.027(0.8770)
Salted pig feet	0.104 (0.7473)	0.131 (0.7177)	0.242 (0.6231)
Toolo beef	0.069 (0.7931)	0.188 (0.6642)	0.042 (0.8259)
Momoni	1.134 (0.2870)	0.979 (0.3223)	0.003 (0.9585)
Sardine	0.020 (0.8862)	0.252 (0.6154)	0.307 (0.5796)
Corned beef	0.126 (0.7224)	0.369 (0.5438)	0.067 (0.7955)
Maggi	5.382 (0.0203)[a]	1.824 (0.1768)	0.624 (0.4296)
Jumbo	0.018 (0.8926)	0.052 (0.8198)	0.009 (0.9237)
Royco	1.001 (0.9712)	0.000 (1.00)	0.001 (0.9767)
Onga	0.001 (0.9763)	0.123 (0.7253)	0.125 (0.7240)
Dzomi	0.033 (0.8551)	1.922 (0.1657)	1.997 (0.1576)
Tinned vegetable salad	0.837 (0.3602)	0.651 (0.4198)	0.007 (0.9334)
Tinapa	0.011 (0.9149)	0.022 (0.8808)	0.003 (0.9600)

[a] Significant at $P < 0.05$: Fisher's Exact Test

APPENDIX Xh: Chi Square test of proportions of patients who included sweetened foods in their usual diet at baseline compared to 3 months and 6 months. Chi square value (p-value)

Sweetened foods	Baseline vs 3 Months	Baseline vs 6 months	3 months vs 6 months
Coka cola	0.027 (0.8687)	0.009 (0.9229)	0.051 (0.8214)
Biscuit	0.029 (0.8657)	0.121 (0.7279)	0.033 (0.8566)
Cake	0.001 (0.9723)	0.015 (0.9037)	0.017 (0.8969)
Sprite	0.161 (0.6886)	0.105 (0.7461)	0.314 (0.5753)
Fanta	0.003 (0.9601)	0.046 (0.8296)	0.022 (0.8834)
Sugar cane	0.037 (0.8482)	0.001 (0.9692)	0.039 (0.8834)
Paper pack juices	0.020 (0.8882)	0.140 (0.7087)	0.047 (0.8293)
Malt	0.473 (0.4917)	0.002 (0.9652)	0.427 (0.5134)

APPENDIX Xi: Chi Square test of proportions of patients who usually consumed alcoholic beverages at baseline compared to 3 months and 6 months. Chi square value (p-value)

Alcoholic beverages	Baseline vs 3 Months	Baseline vs 6 months	3 months vs 6 months
Beer	0.003 (0.9551)	0.118 (0.7316)	0.111 (0.7390)
Guinness	0.029 (0.8640)	0.034 (0.8529)	0.001 (0.9818)
Spirit	0.000 (1.000)	0.009 (0.9236)	0.008 (0.9295)
Wine	0.020 (0.8868)	0.068 (0.7949)	0.094 (0.7597)

APPENDIX XI: Tanita Reference Ranges for Percentage Body Fat, Body Water and Visceral Fat for Standard Adults**.**

A) Percentage body fat ranges for standard adults

Age (years)	% Body fat range			
	Underfat	**Healthy**	**Overfat**	**Obese**
Male				
18	1-9	10-19	20-23	24-50
19	1-8	9-19	20-23	24-50
20 - 39	1-7	8-19	20-24	25-50
40 - 59	1-10	11-21	22-27	28-50
≥ 60	1-12	12-24	25-29	30-50
Female				
18	1-16	17-30	31-35	36-50
19	1-18	19-31	32-36	37-50
20 - 39	1-20	21-32	33-38	39-50
40 - 59	1-22	23-33	34-39	40-50
≥ 60	1-23	24-35	36-41	42-50

B) Body water ranges for standard adults

Gender	% Body water range		
	Low	**Healthy**	**High**
Male	<50	50-65	> 65
Female	<45	45-60	>60

C) Visceral fat ranges for standard adults

Visceral fat range	
Healthy level	**Excess level**
1-12	12-59

Source: *www.tanita.com/data/Manuals/HealthyLifeEducationalBro_.pd*

APPENDIX XII: Bivariate Correlations Between Demographic, Socioeconomic and Lifestyle Indicators

	AGE	MS	EDU	EMP	OCC	AL12MO	FRQ	AMT	TMOD
AGE	.								
MS	.026 .705	.							
EDU	-.008 .904	.016 .815	.						
EMP	.245** .000	-.019 .781	.067 .324	.					
OCC	.023 .763	-.138 .069	.345** .000	.219** .004	.				
AL12MO	-.133 .051	.055 .420	-.137* .046	-.114 .097	.031 .690	.			
FRQ	-.157 .196	-.116 .338	.182 .131	-.024 .847	.045 .753	-.030 .810	.		
AMT	-.436** .001	-.277* .042	.167 .226	-.078 .576	-.026 .880	.166 .248	.262 .072	.	
TMOD	-.068 .446	-.292** .001	.002 .978	-.012 .896	.033 .749	-.015 .868	-.175 .280	.454* .012	.

*: Significant at $p < 0.05$: Spearman's correlations

MS = Marital status
EDU = Education
EMP = Employment
OCC = Occupation
AL12MO = Alcohol consumed in the past 12 months
FRQ = Frequency of alcohol intake in a week
AMT = Amount of alcohol intake at a sitting (number of drinks)
TMOD = Time spent on moderate physical activity (mins)

APPENDIX XIII: Diet Sheet for the Management of Hypertension at The Dietherapy Department, KBTH

DIETHERAPY DEPARTMENT
KORLEBU TEACHING HOSPITAL

Name:……

DIETARY MANAGEMENT AND PREVENTION OF HYPERTENSION

1. Buy lean meat instead of fatty ones. Trim off all excess fat form meat. Remove skin form poultry meats e.g. chicken.

   Do not stop eating. Take meat at least 3 *times a week. Meat intake should be about* 2 *match box sizes or* 1 *joint of chicken or* 1/2 *palm size of fish or large snail per meal.*

2. Eat staples like corn and all corn foods, brown or white rice, yam, plantain. cocoyam, sorghum, millet, cassava and all its products.

3. Eat more vegetables ofall colors e.g. tomatoes, garden eggs, okro, spinash and all green leafy vegetables including salad vegetables. Include beans in the diet more often.

4. Eat fish more often than meat, be it boiled, smoked or fried.

5. Use only 1 teaspoon of evaporated milk o teaspoon of powdered milk in a cup of milo or tea. Take a maximum of 2 cups a

6. Reduce your intake of all oils i.c. you can have 1 fried fish or thickened soups c.g. ,palm or groundnut soup 3 times/week.

7. Choose from these oils-palm oil, groundnut oil, frytol, soya oil, corn oil and olive oil. Use 114 of small tomato puree tin for a meal per person.

8. Palm kernel oil, coconut oil and all solid fats should be taken in moderation.

9. Cut down drastically on cakes, meat and fish pies, rich biscuits, cream crackers, sugar and all sugary foods e.g. toffees, chocolate, ice cream.

10. limit the intake of eggs to two whole eggs per week. If you require more eggs, remove the yolk and eat only the white.

11 . Use little salt when cooking and avoid adding extra salt after the food is served.

12. Avoid all foods preserved with salt e.g. koobi, kako, saIted beef e.g. pig feet, bacon, sausages, salami and all sodium containing foods and flavor boosters like monosodium glutamate e.g. Maggi.A 1 ,Onga,etc.

13. Soups like light, okro, and kontonmire soup can be taken daily,

14. Exercise at least for *30-45* minutes daily.