IMPACT OF PEANUT SUPPLEMENTATION ON CARDIOVASCULAR DISEASE MARKERS IN HIV-INFECTED ADULTS WITH DYSLIPIDEMIA ATTENDING NYERI LEVEL- 5 -HOSPITAL, KENYA

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APRIL, 2016

DECLARATION

I declare that this thesis is my original work and has not been presented for a degree in any other university or for any other award.

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DEDICATION

This work is a dedication to my lovely late father Mr Joseph Kamuhu Murage and my sons Victor Kamuhu and Rafael Wanjuki.

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OPERATIONAL DEFINITION OF TERMS

HIV- associated dyslipidemia- lipid disorder that is found in HIV infected patient who are on ARV and is characterized by increased triglyceride, high total cholesterol, low High density lipoprotein and elevated low density lipoprotein.

Nutritional status - this includes the weight, BMI, blood glucose, waist to hip ratio and body composition.

Healthy diet- this is diet that includes complex carbohydrate, a lot of fruits and vegetables, low in saturated fat and its sources and contains all the food groups**.**

ABBREVIATIONS AND ACRONYMS

ANOVA	Analysis of variance
ART	Anti-retroviral therapy
ATP III	Adult Treatment Panel III
BF	Body fat
BMI	Body Mass Index
CAD	Coronary Artery Disease
CCC	Comprehensive care clinic
CEPT	Cholesteryl-ester transfer protein
CHD	Coronary heart disease
CVD	Cardiovascular Disease
D4T	Stavudine
DBP	Diastolic Blood pressure
DHA	Docosahexaenoic acid
eNOS	Endothelial nitric-oxide synthetase
EPA	Eicosapentaenoic acid
FBG	Fasting blood glucose
FFA	Free fatty acids
HAART	Highly active anti-retroviral therapy
HDL- C	High Density Lipoprotein cholesterol
HIV	Human Immune deficiency Virus
HL	Hepatic lipase
KDHS	Kenya demographic and health survey
LDL- C	Low Density Lipoprotein cholesterol
LDL-P	Low density lipoprotein particle
MI	Myocardial infarction
MUFA	Mono unsaturated fatty acids
NCEP	National Cholesterol Education Program
PCM	Protein calorie malnutrition
PI	Protease inhibitors
PUFA	Poly unsaturated fatty acids
SBP	Systolic blood pressure
SES	Social Economic Status
SFA	Saturated fatty acids
SFA	Saturated fatty acids
SSA	Sub Saharan Africa
TC	Total Cholesterol
TG	Triglycerides
TNF	Tumour necrosis factor
VLDL	Very low density lipoprotein
WHO	World Health Organization
WHR	Waist hip ratio
ZDV	Zidovudine

ABSTRACT

Cardiovascular diseases (CVD) is currently second, after cancer, as the most frequent cause of death among HIV-positive subjects in areas of the world where Highly active anti-retroviral therapy (HAART) is widely available. Dyslipidemia is an important adaptable cardiovascular risk factor that is a widespread clinical feature of HIV-infected patients in the present era of HAART. Peanuts are a rich source of magnesium, folate, fibre, α- tocopherol, copper, arginine and resveratrol. These compounds have been shown to reduce the CVD risk in various ways and this suggests that peanut consumption might benefit those at risk of CVD. The purpose of this study was to investigate the effect of peanut supplementation on cardiovascular disease markers in HIV-infected adults with normal and hyperlipidemia attending comprehensive care clinic in Nyeri Level- 5-Hospital. The study design was a randomized cross-over clinical trial. The study duration was 22 weeks. The eligible participants were randomly assigned to a two arm study. In treatment I, the participants consumed their regular diet supplemented with 80g of peanuts; while in treatment II, the participants were counseled on healthy diet and supplemented it with 80g of peanut. The participants then crossed over to respective treatments. Each treatment took 8 weeks, with a six weeks washout period between treatments. Descriptive statistics were used to analyze all study variables. Relationships between all and individual CVD risk factors were analyzed using Spearman's correlation coefficient, single line linkage and ward's cluster method. A paired T- test was used to compare subject differences in markers at baseline and at the end of each treatment. Multiple regression analysis was used to determine the effect of peanut supplementation on CVD markers. Criterion for statistical significance was at $p < 0.05$ and 90% power of test. The sample comprised of 18 (21.2%) males and 67 (78.8%) females. Peanut supplementation significantly increased intake of total fat ($p < 0.05$), poly unsaturated fatty acid ($p < 0.05$), Vitamin E ($p < 0.05$) and mono unsaturated fatty acids ($p < 0.05$) while carbohydrate intake decreased significantly ($p < 0.05$) between baseline and the two treatments. There was no significant change in weight, BMI, waist circumference, hip circumference, body fat, body muscle, systolic and diastolic blood pressure and fasting blood glucose after consumption of peanut with regular diet and consumption of peanut combined with nutritional counseling on healthy diet. There was a significant decrease ($p < 0.05$) in total cholesterol, triglycerides and Low density lipoprotein in both treatments while High density lipoprotein increased significantly ($p < 0.05$). Also reduced significantly, was the estimated 10-year risk of coronary heart disease between baseline and end of study ($p = 0.03$). Peanut consumption was found to lower cardiovascular risk significantly due to reduction in total cholesterol, triglyceride and Low Density Lipoprotein Cholesterol. The policy makers should promote the incorporation of this food-based supplement in order to lower the economic burden cardiovascular diseases are imposing on the economy.

CHAPTER ONE INTRODUCTION

1.1 Background to the study

Sub-Saharan Africa (SSA) has been reported to have the highest burden of Human immune deficiency virus (HIV) in the world, with approximately 22.9 million prevalent cases and 1.9 million new infections recorded in 2010 (WHO, 2011). It is estimated that 1.3 million people died of HIV-related illnesses in SSA in 2009; this figure comprised 72% of the global mortality attributable to the epidemic (WHO, 2010). The 2008-09 Kenya Demographic and Health Survey (KDHS) indicate HIV prevalence among women aged between ages 15-49 was 8.0 percent, while 4.3% was recorded among men of ages 15-49. This female-to-male ratio of 1.9 to 1 was higher than that found in most population-based studies in Africa.

Anti-retroviral therapy (ART) coverage in the region has had a rapid increase over the past ten years, with 49% of eligible cases receiving treatment in 2010 (WHO, 2011). Expansion in the use of ART, has resulted in a notable decline of HIV-related morbidity and death in SSA (UNAIDS, 2010). While the life expectancy among HIV-infected people improves, it is critical to appreciate the long-term impact of HIV and its treatment in SSA (Negin, Barnighausen, Lundgren & Mills, 2012).

Alongside the changing landscape of HIV care, the burden of cardio metabolic diseases in SSA is rising, (de-Graft, Boynton &Alanga, 2010) with deaths attributable to cardiovascular disease expected to double to 2.4 million in 2030 relative to reports from 2000 (WHO, 2009). The data suggests that cardio metabolic illnesses would become a

major health problem in SSA, competing with infectious diseases for limited health resources (MaherWaswa, Baisle, Karabarind, Unwin &Grosskurth, 2011). Cardiovascular diseases (CVD) are presently the second most common cause of death (after cancer) among people living with HIV in regions of the world where highly active anti-retroviral therapy (HAART) is extensively available (High, Effros, Fletcher & Gebo, 2008).

Factors linked to increased risk of CVD development have been classified as modifiable and non-modifiable. Included among the modifiable, traditional factors are; smoking, elevated LDL and non-HDL cholesterol and lack of exercise. Other factors comprising of the metabolic syndrome including insulin resistance, hypertension, elevated waist circumference, elevated triglycerides and low HDL-cholesterol, are also modifiable. Non-modifiable factors comprise of age, sex and genetic predisposition (Grundy, Pasternak, Greenland, Smith & Fuster, 1999; Hackam & Anand, 2003). Furthermore, other emerging CVD risk factors have been identified in recent years; these include homocystinuria and high levels of C-reactive protein (Grundy, 1999; Hackam & Anand, 2003). Latest data indicate that HIV infection and components of HAART may unfavorably affect multiple risk factors for CVD.

A large global level case-control study comprising over twenty nine thousand cases and controls, which examined cardiovascular risk and other related outcomes across continents found a link between hypertension, diabetes and abnormal lipids to poor CVD outcomes that included stroke and myocardial infarction (MI) in Africa and in the rest of the world. (Poulter,Khaw, Hopwood, Mugambi , Peart, & Sever, 1985). All over the

world, the prevalence of cardiovascular risk factors has been steadily increasing. The modifiable risk factors for cardiovascular disease (CVD), which include hypertension, smoking, abdominal obesity, high levels of cholesterol and diabetes; are the major contributors to cardiovascular morbidity and mortality (Dzau, Antman, Black, Hayes, Manson & Plutzky, 2006; Yusuf, Hawken, Ounpuu, Dans, Avezum & Lanas, 2004). Recently, the World Health Organization (WHO) reported that hypertension affected 972 million people worldwide in 2000 and this number is expected to rise by roughly 60% to 1.56 billion people by 2025 (Kearney, Whelton, Reynolds, Muntner, Whelton & He, 2005). In addition, hypertension is now the leading cause of global mortality (Ezzati, Lopez, Rodgers, Vander Hoorn & Murray, 2002). It has been established that CVD risk factors show a continuous association with overall cardiovascular risk, with no minimum threshold for disease (Kannel, 2000; Stamler, Wentworth & Neaton, 1986). In addition, risk factors hardly occur in isolation as they tend to cluster, acting synergistically to increase an individual's total risk of CVD, from 4-fold with 1 risk factor to 60-fold in the presence of 5 risk factors (Wilson, Kannel, Silbershatz, & D'Agostino 1999)

Moreover, since age and sex are the major non-modifiable cardiovascular risk factors, the risk for CVD in this population is likely to increase in the near future. Estimates were that by the year 2015, more than 50% of all HIV-positive patients would be more than 50 years old; a situation resulting from the significant decline in mortality due to HAART (High, et al, 2008). Hypertension has emerged as an important medical and public health issue in SSA, having been identified as a major risk factor for CVD. This is occurring despite the ravage being perpetuated by tuberculosis, HIV and malaria (Gill, 2009).

Abubakari, & Bhopal, (2008) and Kimokoti & Hamer, (2008) have suggested that communities in SSA currently live with multiple psychosocial stressors. In previous studies conducted elsewhere, chronic poverty-related stressors, such as sanitation, environmental conditions, low education, unemployment, job insecurity, crime, inadequate housing, water, crowding, air pollution, and transportation needs, were found to be potent predictors of poorer perceived health status (Abubakari, Lauder, Agyemang, Jones, Kirk, & Bhopal, 2008; Longo-Mbenza, Mambune, Kasiamm, Vita, Fuele, Nsenga, Mabwa, & Nzuzi, 2008). These stressors may contribute significantly to the rising burden of cardiovascular morbidity and mortality rates in SSA

Other major and well-known modifiable risk factors for CVD are overweight and obesity whose prevalence is growing in SSA is despite the existing competing epidemic of malnutrition (Holdsworth, Gartner, Landais, Maire, & Delpeuch, 2004) and substance use whose disorders and CVD are often co-morbid. Alcohol consumption and tobacco use remains among the most serious epidemiological risk factors for prevalence of coronary artery disease (Verdier & Fourcade, 2007).

Highly active anti-retroviral therapy (HAART) associated dyslipidemia is characterized by hypertriglyceridemia with low plasma concentrations of high-density lipoprotein (HDL) cholesterol and increased total cholesterol, with or without increased low-density (LDL) cholesterol (Penzak & Chuck, 2000). Dyslipidemia refers to hypercholesterolemia (high serum total cholesterol) and hypoalphalipoproteinemia (low serum alpha lipoprotein) which refers to an abnormal metabolism of plasma lipids. Genetic, dietary or

secondary disease factors can cause this abnormal metabolism. The major classes of plasma lipids are cholesterol, cholesterol esters, triglyceride and phospholipids (Ghose, 2006). Dyslipidemia, consisting of hypertriglyceridemia together with depressed concentrations of high density lipoprotein cholesterol and elevated low-density lipoproteins cholesterol, is increasing among people living with HIV. The use of Highly Active Anti-Retroviral Therapy (HAART) has converted HIV infection into a chronic manageable disease (Mocroft, Ledergerber & Katlama, 2003).

A cross sectional study of 295 HIV-infected patients conducted by Manuthu, Joshi, Lule and Karari **(**2008), between January and April 2006 at Kenyatta National Hospital, compared the lipid profile among patients on HAART and those not on HAART. The study revealed that 83% of the HAART group was on Stavudine based regimens and 17% were on Zidovudine based regimen. Among the HAART group, the prevalence of hypercholesterolemia (total cholesterol (TC) > 5.17mmol/L) was 39.2% compared to 10% in the non-HAART group. Patients who were on HAART were more than five times likely to have high TC compared to the HAART naive patients. The prevalence of elevated LDL-C in the HAART group compared to the non-HAART group was 40% versus 11%.

Dyslipidemia associated with HAART is a prevalent condition in patients living with HIV. Above 85% of people living with HIV, receiving highly active antiretroviral therapy are presently surviving for more than 10 years after acquiring the infection (Mocroft et al., 2003). On the other hand, the significant decreases in morbidity and

mortality and increase in life expectancy resulting from the use of HAART have been accompanied by an increase in a number of clinical and metabolic complications. These metabolic complications include dyslipidemia, hyperinsulinemia, and adipose tissue distribution (Carr, Samaras & Burton, 1998). Dyslipidemia is a major modifiable cardiovascular risk factor that is becoming a common clinical feature of HIV-infected patients in the current era of HAART (Grinspoon & Carr, 2005). In addition to metabolic changes, traditional cardiovascular risk factors also appear quite prevalent in the HIV infected population (Friis-Moller, Reiss & Sabin, 2007).

Non- pharmaceutical interventions like diet and exercise should always be the first line of intervention in management of dyslipidemia (Schambelan, Benson, Carr, Currier, Dube, & Gerber 2002). Other interventions should include reduced caloric intake, attaining ideal bodyweight and increased physical activity. These initial steps may yield added health benefits in HIV related dyslipidemia (Henry, Melroe, Huebesch, Hermundson & Simpson, 1998). In a HIV- uninfected population, dietary advice may achieve a 11% and 22% decline in cholesterol and triglyceride levels, respectively, while in HIV- infected individuals, these figures range from 4 to 17% and from 21 to 26%, respectively (Moyle, Sabin, Cartledge, 2006). Barrios, Blanco and García-Benayas (2002), prospectively evaluated the effectiveness of a low fat diet in the reduction of cholesterol and triglycerides in 230 HIV -infected individuals. Those with good adherence to diet had a reduction of 11% and 10% in the levels of cholesterol and 12% and 23% in the levels of triglyceride after 3 and 6 months, respectively.

Nuts and peanuts are food sources that are a composite of numerous cardio protective nutrients and if they are routinely incorporated in a healthy diet, a distinct decrease in population risk of CAD would therefore be expected. Nuts come from different plant families and are classified as tree nuts (a one -seeded fruit with a hard shell) or pea nuts (a member of the leguminous family). Peanuts are also referred to as ground nuts because they develop in the soil. Despite the diversity of the nuts, tree nut varieties share common nutritional attributes with peanut.

1.2 Problem Statement

Antiretroviral therapy continues to provide a durable clinical benefit to individuals who are living with HIV (Mocroft, et al., 2003). While survival has improved, several conditions associated with an ageing population have emerged as health concerns (Grabar, Weiss & Costagliola, 2006) and in particular, cardiovascular disease which now contributes to more than 10% of deaths among people living with HIV (Lewden, May & Rosenthal, 2007; Weber, Sabin, Friis-Møller,Reiss, El-Sadr,Kirk, Dabis, Law,et al., 2003). Although factors affecting cardiovascular risk in people living with HIV seem to be similarly associated with cardiovascular risk in the general population, exposure to antiretroviral therapy has also been shown to unfavorably affect the risk of cardiovascular disease (Friis-Møller, et al., 2007).

Lipid abnormalities may be aggravated by anti-retroviral therapy and dyslipidemia may develop in up to 70% and 80% of people living with HIV, with hypertriglyceridemia presenting in most of cases (60% to 100%) of treated patients. The hypertriglyceridemia enhances development of small, low density lipoproteins particles (Manfredi, 2005).

Both hypercholesterolemia and hypertriglycedemia constitute well recognized cardiovascular risk factors. This has raised major concerns because CVD has become a chronic disease in most people living with HIV. Episodes of premature vascular events have been reported in individuals on HAART (Henry, et al., 1998).

Managing HIV- associated dyslipidemia should, where possible, include non-drug interventions (Henry et al., 1998). The use of lipid lowering drugs is of concern given their toxicity, intolerance and potential interactions with antiretroviral agents. Non-pharmaceutical interventions that include diet and exercise are always the first line of intervention (Barrios, et al., 2002). Therapeutic interventions in people living with HIV with lipid disorders include; low- fat diet, exercise, smoking cessation, drug therapy and a modification of antiretroviral therapy (Barrios et al., 2002). No specific recommendations for management of dyslipidemia exist for these individuals.

Peanuts are a rich source of magnesium, Folate, fibre, α- tocopherol, copper, arginine and resveratrol. All these compounds have shown to reduce CVD risk in various ways and this suggests that peanut consumption might benefit those at risk of CVD. Epidemiological studies in developed countries have shown that frequent nut consumption decreases the risk of CHD, with adjusted relative risk reductions approaching 50% in subjects who consumed 4-5 servings per week than those who have little or no intake (Kris- Etherton, Zhao & Binkoski, 2001). However these peanut studies have only been performed in either healthy adults or diabetic dyslipidemia subjects in

combination with low fat diet. This study explored the effect of peanut consumption on the lipid profile in patients with normal and dyslipidemia.

1.3 PURPOSE OF THE STUDY

The aim of this study was to investigate cardiovascular risk factors the effect of peanut supplementation and nutrition counseling on biomarkers of CVD in free living adults with dyslipidemia using a randomized crossover at Nyeri-Level 5 Hospital, in Nyeri County, Kenya.

1.4 OBJECTIVES

To achieve the purpose of the study, the specific objectives of this study were to:

1. To examine the demographic and social economic status of the people living with HIV attending Nyeri level-5 hospital CCC
2. To assess the traditional risk factors for cardiovascular diseases in people living with HIV attending Nyeri level-5 hospital CCC
3. To estimate the proximate composition of raw and dry roasted peanut consumed in the study.
4. To establish the dietary intake and practices of the people living with HIV attending Nyeri level-5 hospital CCC.
5. To determine the effect of peanut supplementation on nutritional status markers of the people living with HIV attending Nyeri level-5 hospital CCC.
6. To establish the effect of peanut supplementation on the lipid profile of the people living with HIV attending Nyeri level-5 hospital CCC.

7. To estimate the effect of peanut supplementation on participant's overall 10 year risk of developing coronary heart disease

1.5 Hypothesis

H_{01}. Supplementation with peanuts has no significant effect on nutritional status in HIV-infected subjects with dyslipidemia

H_{02}. Supplementation with peanuts has no significant effect on blood lipid profile in HIV-infected subjects with dyslipidemia.

H_{03}. Supplementation with peanuts has no significant effect on the overall 10 year risk of developing coronary heart disease

H_{04}. There is no significant difference between the effects of treatment 1(80g of peanut and regular diet) and treatment II (80g of peanut and counseling on healthy diet) on lipid profile, hip and waist circumference and BMI.

1.6 Significance of the study

Peanuts are food sources that have a composite of numerous cardio protective nutrients and if they are routinely incorporated in a healthy diet, a remarkable decrease in population risk of coronary artery disease would therefore be expected. The study findings will provide information on the potential of peanut consumption in regulating blood lipids in people living with (PLHIV). The information may be used to formulate policies and programs aimed at lowering risk of CVD in PLHIV and general population. The information obtained will also be useful in persuading the local people to grow peanuts as a source of food and beneficial nutrients. Other stake holders that would benefit from the results are Ministry of Health, National Aids Coordinating Council,

National Aids and STI Control Council as well as county health ministries in the policy making.

1.7 Delimitations

The study sought to investigate the effect of peanut consumption on lipid profile and nutritional status, but not on any other CVD risk factors such as inflammatory factors, lipodystrophy and lipo-hyperatrophy. Outcomes assessed were TC, HDL-C, LDL-C, triglycerides and nutritional status.

1.8 Limitations

Since the participants were free living, control of nut intake from other sources was not possible but was determined or noted from dietary intake assessments.

1.9 Assumptions

The study assumes that the reduction in cardiovascular risk is primarily due to nutrient contents of peanut and not nutrient contents from other food sources**.**

1.10 Conceptual Framework

Figure 1.1 shows the overview of the effects of HIV and its therapies on CVD risk. The contribution of traditional risk factors must be considered, as they may occur with increased prevalence in people with HIV infection (example, smoking). HIV itself and HAART lead to metabolic complications such as dyslipidemia, insulin resistance and adipose tissue redistribution. These complications have been shown to lead to cardiovascular diseases. The link between body composition and CVD indicates that body fat is known to affect mediators such as dyslipidemia and insulin resistance. This

study evaluated the effects of peanuts on lipid profile and various coronary artery disease risk factors such as obesity, elevated serum glucose levels, waist hip ratio, hypertension and body fat. Evidence from other studies has constantly shown the favorable effect of peanut on these CAD risk factors.

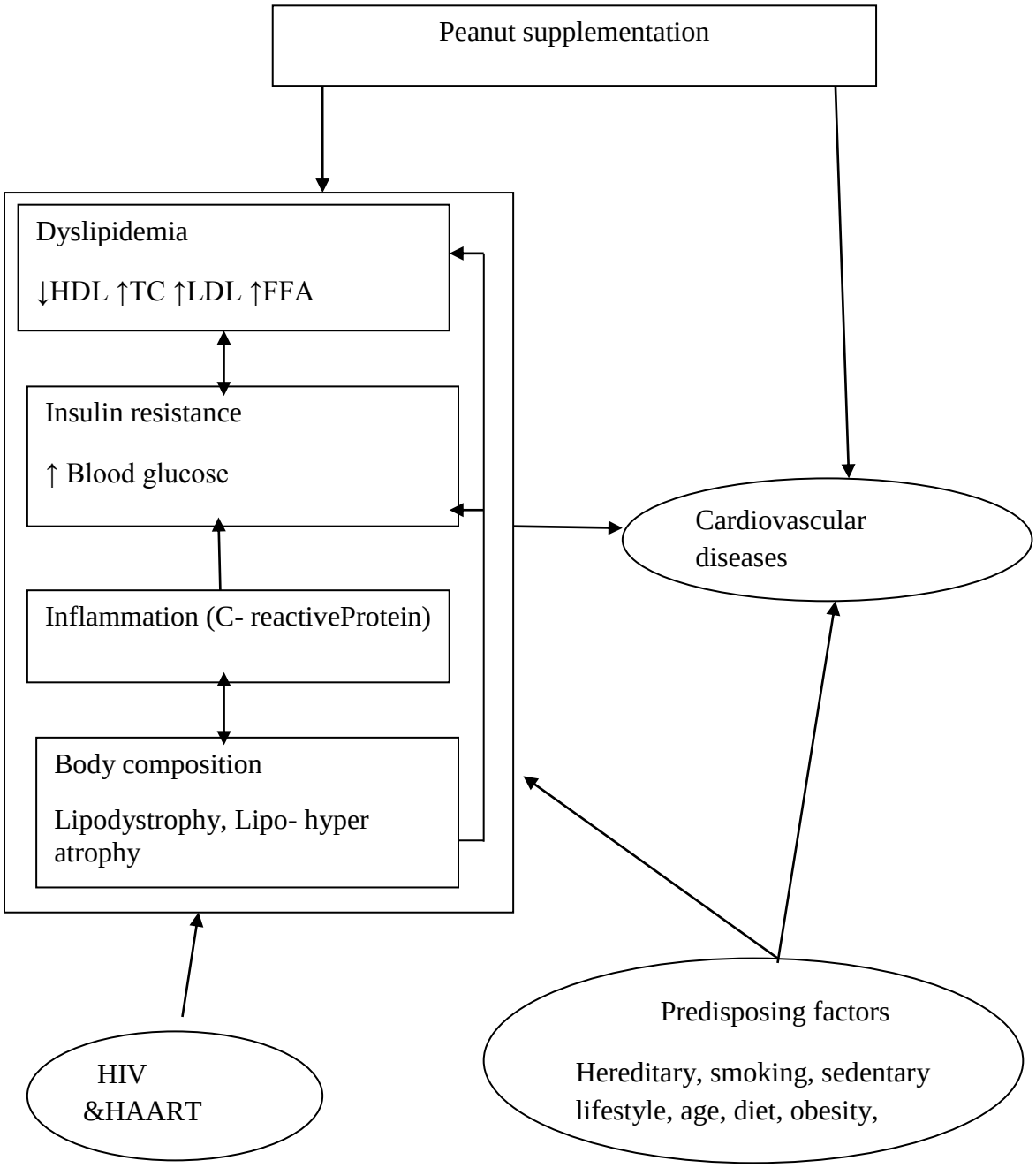

Figure 1.1 Overview of the effects of HIV and its therapies on CVD risk
Source: Adopted from Grunfeld, et al., 2008.

CHAPTER TWO: LITERATURE REVIEW

2.1 HIV and cardiovascular diseases

Cardiovascular diseases (CVD) among HIV-positive subjects in regions of the world where HAART is extensively available are currently the second most frequent cause of death (after cancer) (Effros, et al., 2008). The relationships between HAART and increased cardiovascular events that point to a causal role of the treatment on lipid and glucose metabolism have been reported repeatedly in epidemiologic studies (Worm, De Wit & Weber, 2009). In a healthcare system-based cohort study in Massachusetts General Hospital in USA, using a large data registry with 3851 HIV and 1,044,589 non-HIV patients, the unadjusted acute myocardial infarction (MI) rates per 1000 person-years were higher in HIV versus non-HIV patients (11.13 versus 6.98). The rates of MI were consistently higher for patients in the HIV cohort compared with the non-HIV cohort, across all age groups. In another study, HIV-infected patients on ART had an increased risk of ischemic heart disease compared with the general population.

These studies underscore the increased CVD risk in this population, which is only partially related to traditional risk factors (Friis-Moller,et al., 2003). Retrospective analyses designed to estimate the risk of cardiovascular disease in relation to antiretroviral therapy have yielded variable results (Bozzette, Ake, Tam, Chang & Louis, 2003; Klein, Hurley, Quesenberry & Sidney, 2002; Mary-Krause, Cotte, Simon, Partisani & Costagliola, 2003). From the findings, it is seen that the risk of cardiovascular disease may be greater in younger patients than in older patients. (Currier, Taylor & Boyd, 2003)

The Data Collection on Adverse Events of Anti-HIV Drugs (DAD) Study represents the largest prospective study of cardiovascular risk with antiretroviral therapy (Friis-Moler et al., 2003). Less than one percent of the 23,468 participants had a first myocardial infarction, an incidence of 3.5 per 1000 person. Twenty nine percent of these myocardio infarctions were fatal which represents 6 percent of all the deaths in the study. 77 events were related to ischemia which included ischemic stroke, coronary-artery bypass surgery or angioplasty and carotid endarterectomy among others (d'Arminio, Sabin &Phillips, 2004). The occurrence of any ischemic vascular event or myocardial infarction was observed to increase with increase in exposure to antiretroviral therapy . The study could not determine the relative risk associated with particular antiretroviral drug because the cases of ischemic events were too few. Other factors associated with an increased risk of myocardial infarction were; hypercholesterolemia, older age, smoking, diabetes mellitus, male sex, and a prior history of cardiovascular disease (Friis-Moler et al., 2003).Although the risk of myocardial infarction in relation to the length of use of antiretroviral therapy remained significant, the risk reduced relatively in the analyses that adjusted for increased cholesterol levels. This suggests that metabolic abnormalities that are induced by ART contributed to the observed increased morbidity (Friis-Moler et al., 2003). However for most patients, except those with multiple other cardiovascular risk factors, the absolute risk of cardiovascular disease remained low.

This study sought to establish the cardiovascular risk of the HIV infected patients in Nyeri level -5-hospital and their 10-year risk of coronary heart disease.

2.2.1 Mechanisms of Cardiovascular Disease

Reduced flow-mediated dilation and endothelial dysfunction together with increased atherogenic lipoproteins have been reported among people living with HIV receiving protease inhibitors (Stein, Klein & Bellehumeur, 2001). Hsue, Lo, & Franklin (2004) also reported that there is an increased carotid intima–media thickness with increased rates of HIV progression in people living with HIV with a mean age of 45 years, compared with age and sex-matched uninfected controls. Hsue et al., (2004) reports that, increased carotid intima-media thickness is also associated with traditional risk factors such as hypertension, hypercholesterolemia and smoking. Hypertension has been seen to be more prevalent in people living with HIV on protease inhibitors or non-nucleoside reverse transcriptase inhibitors, or both, than in patients who are not on ART. Hypertension is also associated with increased BMI among people living with HIV (Friis-Moller et al., 2003).

Although the mechanisms of vascular disease in people living with HIV are not known, factors such as diabetes mellitus, dyslipidemia, impaired fibrinolysis, insulin resistance, inflammation, antiretroviral medications specific factors, or combinations of these factors may be related to vascular disease. In people living with HIV there is observed increase in tissue levels of plasminogen-activator inhibitor 1 and plasminogen activator which suggest impairment of fibrinolysis in these patients. The increase of these substances has not only been associated with lipodystrophy, hyperinsulinemia, and protease inhibitor therapy (Koppel, Bratt, Schulman, Bylund & Sandstrom, 2002) but has also not been linked to vascular disease in people living with HIV. Increased levels of PI may cause the formation of lesions that are atherosclerotic by elevating CD36-dependent cholesterol

ester accumulation in macrophages - a scavenger-receptor pathway that is suspected to mediate the formation of these lesions (Dressman, Kincer, Matveev, 2003).

2.3 HIV and cardiovascular risk factors

HIV infection and its treatment with HAART may influence other traditional cardiovascular risk factors thus increasing cardiovascular risk. In a recent study, increased prevalence of traditional risk factors was shown to lead to higher calculated 10 year Framingham risk scores in HIV infected men without known CHD, as compared with non-infected controls (17% versus 11% 10 year risk of ≥25%) (Ho & Hsue, 2009).

Cardio vascular risk factors associated with an increased risk of CVD development are classified into two; modifiable and non-modifiable factors. The modifiable factors include elevated Low density lipoproteins, and non-High Density Lipoprotein cholesterol, smoking, overweight and obesity as well as sedentary lifestyle. Other factors are metabolic, including; elevated waist circumference, insulin resistance, hypertension; elevated triglycerides and low HDL-cholesterol. These consist of the metabolic syndrome and are also modifiable. Those factors that are non-modifiable include; genetic predisposition, gender and age (Grundy, et al., 1999; Hackam, et al., 2003). Furthermore, there are other emerging CVD risk factors that have been identified ; including, elevated levels of C-reactive protein and homocystinuria (Grundy, et al., 1999; Hackam, et al., 2003). HIV infection and components of HAART may unfavorably affect multiple risk factors for CVD according to recent data.

Traditional cardiovascular risk factors also appear fairly prevalent in the HIV infected population in addition to metabolic changes; smoking is also common among HIV patients. In a French cohort study, HIV patients aged 35-44 years on HAART were compared to a general population based cohort. This cohort estimated attributable risks to CHD due to smoking to be 65% for men, and 25% for women. This agrees with the DAD study that found smoking to be the most powerful predictor of CHD in patients living with HIV despite their existing history of CHD (Friis-Moller, et al., 2007). These studies have been done in developed countries and there is little or inadequate information in the study area. The study sought to establish the modifiable and non-modifiable cardiovascular risk factors among the HIV infected patients in Nyeri level -5-hospital.

2.3.1 HIV associated dyslipidemia

High serum triglycerides in association with low HDL and LDL cholesterol levels were a common observation in people living HIV prior to the era of HAART (Grunfeld, Pang, Doerrler, Shigenaga, Jensen & Feingold, 1992). Suggested contributing factors identified by earlier studies included: increased hepatic synthesis of very-low-density lipoprotein, increased apolipoprotein E levels, and decreased clearance of triglycerides. Dyslipidemia may in part be due to the effects of viral infection, circulating cytokines including interferon-α and acute-phase reactants (Christeff, Melchior, de Truchis, Perronne & Gougeon, 2002). It is known that Stavudine-based antiretroviral therapy (except Tenofovir-based) has been associated with early and significant increases in triglyceride and total cholesterol levels (Gallant, Staszewski &Pozniak, 2004). However the specific effects of Thymidine analogues on lipid turnover are not known (Matthews, Moyle, Mandalia, Bower, Nelson & Gazzard, 2000).

Switching from a protease inhibitor based regimen to other regimens has been observed to improve HDL cholesterol levels in patients (Van der Valk, Kastelein & Murphy, 2001). Protease inhibitors increase total cholesterol levels although this effect varies among individual drugs found in this class (Periard, Telenti & Sudre, 1999). Other PIs, most notably Ritonavir, increase hepatic triglyceride synthesis and thus the plasma triglyceride levels (Lenhard, Croom, Weiel &Winegar, 2000). Atazanavir, also a protease inhibitor, does not seem to have a similar effect (Murphy, Sanne & Cahn, 2003). Apolipoprotein B alterations have been observed to occur in patients receiving combination therapy (with a nucleoside analogue and a protease inhibitor) leading to an increase in small, dense LDL 2, elevation of apolipoprotein B levels in blood as well as a shift towards triglyceride-rich VLDL (Schmitz, Michl & Walli, 2001). Additionally, the levels of lipoprotein particles containing apolipoprotein C-III and apolipoprotein E also increase in protease-inhibitor-treated patients (Bonnet, Ruidavets &Tuech, 2001). In addition, proteasomal degradation of nascent lipoprotein B in vitro is decreased by HIV protease inhibitors (Liang, Distler& Cooper, 2001).

Lipid profile changes in HIV infections have been found to occur in two ways. In the early stages of HIV infection before treatment commences the changes include high triglycerides in blood, low high density lipoproteins and high low density lipoproteins values. However, on commencement of HAART, low density lipoproteins and total cholesterol elevate significantly with little change in high density lipoproteins. These changes are particularly associated with the use of PIs (Grinspoon et al., 2005). A Swiss

HIV Cohort study, reported hypercholesterolemia and hypertriglyceridemia to be 1.7-2.3 times more common among patients on Protease Inhibitors (PIs) based HAART than when compared with those without PIs. The overall effect of HIV infection therefore leans towards a significant reduction in HDL, an increase in triglycerides, oxidized LDL and small dense LDL thus an atherogenic lipid profile (Hsue & Waters, 2005). Tungsripipat & Aberg (2005) have also concurred that HIV is, by itself, associated with atherogenic dyslipidemia, which includes hypertriglyceridemia and low levels of high density lipoproteins.

Both HAART and and the adverse metabolic effects of HIV infection are associated with the development of cardiovascular risk factors. HIV infection itself is associated with dyslipidemia and to a lesser extent deranged glucose metabolism. This was seen in people living with HIV before the advent of effective antiretroviral therapy (Grunfeld & Tien, 2003). Hypertriglyceridemia is also associated with antiretroviral therapy. A higher baseline prevalence of triglyceride levels was reported in a study consisting of 113 HIV-infected patients treated with PIs compared with 45 HIV-infected nonusers. The baseline prevalence of triglyceride levels was greater than 177 mg/dL that was 50% versus 22%, respectively; total cholesterol levels were greater than 213 mg/dL that was 58% versus 11%, respectively, and combined hypertriglyceridemia and hypercholesterolemia were at 38% versus 5%, respectively in PI users than in nonusers (Carr, Samarasa & Thorisdottir, 1999).

In the pre-HAART era, researchers documented depressed total cholesterol and low-density lipoprotein levels in patients with early disease while elevated triglycerides were observed predominantly in patients with advanced HIV infection (Constans, Pellegrin & Peuchant, 1994; Grunfeld, et al., 1992). High-density lipoprotein (HDL) cholesterol was found to be lower in patients with lower CD4 lymphocytes and more immune activation (Zangerle, Sarcletti, Gallati, Reibnegger, Wachter& Fuchs 1994). However, few studies examining lipid levels in patients with HIV disease focused on lipid levels as markers of chronic inflammation.

There is an observation by the clinicians about elevated cholesterol and elevated triglyceride levels in people living with HIV who are maintained on HAART. This dyslipidemia was often associated with other metabolic abnormalities, including; lipodystrophy which is often characterized by an enlarged dorsocervical fat pad, accumulation of visceral fat, and atrophy of subcutaneous fat in the face, buttocks, and extremities and insulin resistance (Garg, 2000).

Dyslipidemia has emerged as an important CVD risk factor in SSA even among HIV un-infected people. Norman and colleagues found that hypercholestoremia accounted for above 50% of ischemic heart disease and 29 percent of ischemic stroke burden in adults aged 30 and above. There are regional variations in the prevalence of dyslipidemia, especially cholesterol across SSA. A study performed on healthy workers in Nigeria found 23% of the study population had elevated total serum cholesterol compared to studies in Gambia and Tanzania that reported 25% (Njelekela, Mpembeni, Muhihi,

Mligiliche, Spiegelman, Hertzmark, et al., 2009; Van der Sande, Milligan, Walraven, Dolmans, Newport, Nyan et al., 2001). Similar high dyslipidemia prevalence has been reported among type 2 diabetic patients in Nigerian study and results also agree with a Kenyan study. Same high prevalence of dyslipidemia was reported among diabetics in a Ghanian study (Okafor, Fasanmade & Oke, 2008; Otieno, Mwendwa, Vaghela, Ogola & Amayo, 2006; Eghan & Acheampong, 2003). Another study has shown significant association of triglycerides and HDL-cholesterol with inadequate glycemic control, obesity, female gender, advancing age, physical inactivity (Elnasri & Ahmed, 2008).

Prevalence of elevated cholesterol was higher in urban areas than rural areas in the Gambian study and the same was reported in Cameroon where the prevalence was higher in urban areas that in the rural areas. (Fezeu, Assah, Balkau, Mbanya, Kengne, Awah & Mbanya, 2008) The prevalence of hyperlipidemia is thoroughly researched in patients on protease inhibitors. There is limited information in lipidemia status in HIV infected patients on NRTI and NNRTI. This study will establish the lipidemia status in the patients attending Nyeri level-5-hospital.

2.3.1.1 Pathophysiology of Atherogenic Dyslipidemia

In atherogenic dyslipidemia, there is increased lipolysis (conversion of triglycerides in the adipose cells into free fatty acids) due to impaired insulin signaling. These free fatty acids are then transported through the blood to the liver and muscles (Miranda, DeFronzo, Califf & Guyton, 2005). These free fatty acids are then re-esterified into triglycerides which together with ApoB enhance formation and secretion of Very Low density lipoprotein particles. The production of VLDL is then augmented further by

elevated concentration of plasma glucose. There are two types of VLDL synthesized by the liver; large triglyceride rich VLDL (VLDL-1) and the smaller triglyceride poor VLDL (VLDL-2) (Olofsson, Wiklund & Boren, 2007). Patients with insulin resistance, Type 2 diabetes and atherogenic dyslipidemia have been found to have overproduction of VLDL-1 in the liver. This particle determines the atherogenicity of plasma lipoproteins.

Development of atheroschlerosis is influenced by increased secretion of VLDL-1 which also leads to decrease in HDL and elevated sdLDL production (Adiels, Olofsson, Taskinen & Boren, 2008). Production of VLDL-1 involves three steps; the first step involves limiting the Apo B100 in liver cells by utilizing endoplasmic reticulum microsomal enzyme transfer protein which in turn leads to production of a nascent pre-VLDL particle. The second step involves the lipidation of this nascent pre-VLDL particle in order to produce VLDL-2. The third step involves the lipidation of VLDL-2 to produce VLDL-1. Synthesis of sdLDL from TG-rich VLDL-1involves a two step process; the first step involves the transfer of triglyceride from very low density lipoprotein 1 to low density lipoprotein by a protein called cholesteryl ester transfer protein (CETP). The second step is the conversion of triglyceride rich low density lipoprotein to small dense low density lipoprotein by hepatic lipase. Small dense low density lipoprotein is reportedly more atherogenic than low density lipoproteins and therefore serves as a better predictor of cardiovascular risk (Griffin, Minihane, Furlonger, Chapman, Murphy & Williams, 1999; Musunuru, 2010). This is because of their ability to bind in the arterial proteoglycans hence getting retained in the extracellular matrix as well as their ability to penetrate the arterial intima (Davidson, Carlson, Guthrie, Kelly, Lele, Setze, et al., 2008).

They also have decreased capacity to oxidize (Vinik, 2005). When triglyceride levels are above 1.5 mmol/L, there is a reported increase in generation of sdLDL. These particles have low affinity to LDL receptors in the liver cells thus there is a decreased uptake and clearance resulting in increased presence of the sdLDL in systemic circulation. There are 2 types of LDL patterns, based on LDL-C particles size in the blood there are two types of patterns of LDL. The first one is LDL A whose particle size is >25.5nm and LDL B whose particle size is ≤ 25.5nm.(Musunuru, 2010). One Apo B100 is contained in each LDL, IDL and VLDL particle.

The amount of cholesterol carried by a low density lipoprotein particle is reflected by the LDL-C. There is observed significant inter-individual differences in the amount of cholesterol in LDL particles with differences as high as twice between individuals and the changes occurring with lipid altering treatments. The most effective method of measuring LDL levels is total low density lipoprotein particle (LDL-P) or Apo B. Although levels of LDL-P and LDL-c have been reported to be same in many patients, in others both LDL-P and LDL-C levels were discordant due to differences in the amount of cholesterol in LDL particles. Similar discordance of LDL-P and LDL-C has been reported in patients on statin therapy as they lower LDL- C and LDL-P considerably (Witztum & Steinberg, 1991).

Another particle that plays an important role in atherogenic dyslipidemia besides the small dense low density lipoprotein is the oxidized low density lipoprotein. This oxidized LDL particle is generated via sdLDL or LDL. There are three phases in which i*n vitro*

oxidation of LDL by metal ions takes place. The first phase also called the initial lag phase occurs when there is consumption of an endogenous antioxidant. The second phase also called the propagation phase occurs when the unsaturated fatty acids are oxidized rapidly to form lipid hydroperoxides. The third phase also called decomposition phase occurs when there is formation of aldehydes that are reactive. These reactive aldehydes give rise to oxidized LDL by reacting with residues of lysine in apoB-100. The origin of the circulating oxidized LDL is from mild oxidation by cell associated LOX or the myeloperodidase in the arterial wall rather than extensive metal ion-induced oxidation in the blood (Erl, Weber & Weber 1998; Witztum & Steinberg, 2001). Susceptibility to LDL oxidation is varied due to differences in the amount of PUFA and anti oxidants among individuals. The oxidized-LDL continues to interact with scavenger receptors called macrophages present on endothelial cells and smooth muscle cells. There is continued interaction between the oxidized LDL and the macrophages also called the scavenger receptors. This interaction leads to atherosclerosis, which results from endothelial dysfunction that causes accumulation of cholesterol within the blood vessels (Mehta, Yang, Khan, Hendricks, Stephen & Mehta 1995). Oxidized-LDL has the following functions; firstly is the inhibition of expression of endothelial nitric-oxide synthase (eNOS). Secondly it facilitates infiltration and adhesion of monocytes, facilitation of molecule induction adhesion, migration and proliferation of smooth muscle cell as well as the release of growth factor and cytokine from smooth muscle and endothelial cells (Kontush, Chantepie & Chapman 2003: Pryor, 2000).

VLDL-1 produces small HDL besides producing CETP ,sdLDL, and HL. These small HDL that are formed have a high rate of clearance from the circulation, hence a decline in the plasma levels apolipoprotein A-I and HDL-C. The metabolic disturbance that exacerbates atherosclerosis begins with increased formation of VLDL-TG ends in atherogenic reduction of high density lipoprotein, reduced transport of cholesterol from the hepatocytes, macrophages and peripheral tissues to the liver as well as the intravascular remodeling. Pro-atherogenicity of high density lipoprotein was reported among patients with (CAD) according to report by Kontush and colleagues in 2003, and Smith in 2010. In their report, pro atherogenity of HDL was due to alterations in the HDL structure and function. First are the changes in its protein composition, secondly due to the decrease in CETP and Apo A1, thirdly due to the changes in HDL-associated lipids and lastly due to the post-translational modification of Apo A1.The functional and structural heterogeneity of high density lipoprotein particles has led to the pro atherogenic nature of HDL. It was found that increase in density resulted in increase in the antioxidant activity of HDL in the following order HDL2b < HDL2a < HDL3a < HDL3b < HDL3c; HDL3c would have higher antioxidant activity as compared to HDL2b (Smith, 2010).

There have been marked racial differences in atherogenic dyslipidemia. An increased prevalence of atherogenic dyslipidemia has been observed in Asian Indian populations compared to western populations, in a number of studies. This may be attributed to their consumption of low polyunsaturated fatty acid (PUFA) diet that is carbohydrate rich and less physical activity (Mohan, Shanthirani, Deepa, Premalatha, Sastry, Saroja. et al.,

2001; Mulukutla, Venkitachalam, Marroquin, Kip, Aiyer, Edmundowicz, et al., 2008; Ramchandran, Snehalatha, Satyavani, Sivasankari & Vijay, 2003). Compared to western populations, Asian Indians possess significantly lower levels of HDL and LDL-C with hypertriglyceridemia. Besides being more dyslipidemic, Indians were reported to have higher intra-abdominal visceral fat than western populations, which increased their insulin resistance and CVD risk. Compared to the whites, blacks had less likelihood of atherogenic dyslipidemia. This was attributed to the significantly lower levels of total cholesterol, LDL pattern B, LDL-C and triglycerides in blacks than in whites (Mulukutla, et al., 2008). The blacks also have lower hepatic lipase activity, resulting in more buoyant and less atherogenic LDL particles than whites, hence the low prevalence of atherogenic dyslipidemia in their population.

2.3.2 Hypertension

Existing studies have defined hypertension using the World Health Organization criteria for blood pressure as ≥ 160/95 mmHg and also according to the report by Joint National Committee on Prevention, Evaluation and Treatment as blood pressure above 140/90mmHg or those on anti hypertensive medication (Addo, Smeeth & Leon., 2007). Others have referred hypertension a silent killer (Van der Sande, et al., 2001; Van der Sande, et al., 2000; Van der Sande, 2003), owing to the fact that it does not have detectable signs at the early stages even though it is the major cause of heart disease, renal diseases and stroke which are serious health conditions (Duda, Kim, Darko, Adanu, Seffah, Anarfui, & Hill, 2007; Hoel &Howard, 1997). In South Saharan Africa, hypertension has been identified as a major CVD risk factor in and has also emerged as an important public health and medical issue. This is despite of the devastation caused by

HIV, malaria, and tuberculosis (Addo, Amoah & Koram, 2006; Addo, et al., 2007; Agyemang, Bruijnzeels & Owusu-Dabo, 2006; Agyemang, & Owusu-Dabo, 2008).

As in the general population, hypertension is a significant contributor to cardiovascular disease among HIV-infected individuals (Friis-Moller, et al., 2007; Mocroft, Reiss, Gasiorowski, Ledergerber et al., 2010). The prevalence of hypertension in different selected HIV-infected cohorts varies considerably, from 13% to 49% (Lichtenstein, Armon,Buchacz, Chmiel,et al.,2010; Baekken, Sandivik & Oektedalan.,2008; Bergersen, Sandvik, Dunlop, Birkeland & Bruun, 2003). Population differences and methodological issues are likely explanations for the discrepancies in prevalence. Increased blood pressure and development of hypertension have been observed after initiation of combined ART in prospective studies, effects that could be attributed to increased BMI, lipid profile and immunological status (Crane, Van Rompaey & Kitahata., 2006; Palacios, Santos, Garcia, Castells, Gonzales et al., 2006). However, that cumulative exposure to ART leads to development of hypertension could not be confirmed in the large prospective multinational D:A:D (The Data Collection on Adverse Effects of Anti-HIV Drugs) study (Thiebaut, El-Sadar, Friis-moller, Rickenbach, Reiss, et al., 2005). Low-grade inflammation and immune activation persist in HIV-infected individuals despite antiretroviral therapy (Neuhaus Jacobs, Baker, Calmy, Duprez ,et al.,2010), and immune activation and inflammation could contribute to elevated BP (Harrison, Guzik, Lob, Madhur, Marvar Thabet, et al., 2011).

Sander and colleagues (2015) found the prevalence of hypertension among the HIV infected adults in Rakai Uganda, was 8.0% (95% CI: 5.4–10.6%), and that of elevated

blood pressure (one elevated blood pressure reading) was 26.3% (95% CI: 22.1–30.5%). Age ≥50 years and higher BMI were positively associated with elevated blood pressure. This prevalence is consistent with rates reported from rural and urban HIV-positive subjects in Kenya (8.8%) and is lower than rates in urban populations of Uganda (15.1% and 27.9%) (Semeere, Sempa, Lwanga, Parkes & Kambugu,. 2014; Bloomfield, Hogan, Keter, Sang, Carter, Velazquez et al. 2011; Mateen, Kanters, Kalyesubura. Mukasa, Kawuma, Kengne, et al. 2013).

Reported rates of hypertension prevalence vary across and within regions in SSA. An analysis carried out in the 1990s, of all national data in Zimbabwe established that the national crude prevalence of hypertension between 1990 and 1997, increased from 1% to 4% (Mufunda, Mebrahtu, Usman, Nyarango, Kosia, & Ghebbrat 2006

2.3.3 Diabetes mellitus

Hyperinsulinemia which is a surrogate measure of insulin resistance is commonly seen in association with excess truncal fat, loss of fat in the limbs, an increased waist-to-hip ratio and a buffalo hump. When matched for age and BMI, adults living with HIV with fat accumulation or lipoatrophy, diabetes mellitus was seen in 7.0 %, as compared with 0.5 % of otherwise healthy control subjects while impaired glucose tolerance was present in more than 35 % of HIV-infected subjects as compared with 5 % of otherwise healthy control subjects (Hadigan, Jeste & Anderson, 2001).In another longitudinal cohort study, diabetes mellitus was found to be 3.1 times more likely to develop in men living with HIV receiving combination antiretroviral therapy, compared to control subjects over a

three-year period of observation (Brown, Cole & Li, 2004). The rate at which impaired glucose tolerance and insulin resistance in HIV-infected adults' progress to overt diabetes mellitus is not known.

Antiretroviral therapy may lead to altered flux of substrates that include free fatty acids (Hadigan, Borgonha, Rabe, Young & Grinspoon, 2002), accumulation of intramyocellular lipid (Gan, Samaras, Thompson, et al., 2002), alterations in adipokine levels, like low levels of adiponectin (Tong, Sankale, Hadigan, Tan, Rosenberg,et al., 2003) and reduced PPARγ expression in subcutaneous adipocytes (Bastard,Caron, Vidal, Jan, Auclair, et al., 2002). Antiretroviral therapy may also alter glucose homeostasis. Protease inhibitors (Ben-Romano, Rudich, Torok, Vanounou, et al., 2003; Murata, Hruz & Mueckler, 2000; Rudich, Vanounou, Riesenberg, Porat, & Tirosh, et al 2001) have been shown to induce insulin resistance *in vitro* by reducing glucose transport mediated by glucose transporter GLUT 4 (Murata, et al., 2000).This is seen to occur without affecting postreceptor insulin signaling. The results of clinical studies have suggested that Indinavir and Lopinavir have short-term adverse effects on insulin sensitivity (Lee, Seneviratne & Noor, 2004; Noor, Seneviratne & Aweeka, 2002).

Delayed but long-term effects that are possibly related to changes in body composition may also affect insulin sensitivity. Protease inhibitors such as Atazanavir and Saquinavir may have minimal effects on insulin sensitivity (Kurowski, Sternfeld, Sawyer, Hill & Mocklinghoff, 2003; Noor, Grasela & Parker, 2004). Pancreatic beta-cell insulin secretion may be decreased by the protease inhibitors (Woerle, Mariuz &Meyer, 2003),

although insulin resistance is the primary defect. Direct effects of nucleoside analogues on glucose metabolism have not been demonstrated, but such drugs may contribute to insulin resistance indirectly through changes in fat distribution. The prevalence of diabetes in the study population is not known hence the study will establish the prevalence of pre diabetes (impaired glucose tolerance) and diabetes.

Capeau and colleagues, 2012 reported a major role for age, in the adiposity (as evaluated by BMI, WHR and waist circumference) in the development of new-onset diabetes mellitus even stronger than the effect of ARVS drugs. Lipoatrophy and lipohyperatrophy (increased WHR) were also reported as risk factors. Inflammatory factors sTNFR 1 were marginally predictive of subsequent diabetes in patients initiating combined ART which is consistent with data from general population indicating that chronic subclinical inflammation might be involved in the pathogenesis of type 2 diabetes mellitus due to systemic causative factors such as central obesity and insulin resistance (Bonora, Kiechl, Willeit,Oberhollenzer, et al., 2004)

Bhutt and colleagues, 2009 studied 3,327 HIV-infected and 3,240 HIV-uninfected subjects and reported that HIV infected subjects had a lower prevalence of diabetes at baseline (14.9% vs. 21.4%, $P<0.0001$). After adjustment for known risk factors, HIV infected individuals had a lower risk of diabetes (OR 0.84, 95% CI 0.72-0.97). Increasing age, male gender, minority race, and BMI were associated with an increased risk. The odds ratio for diabetes associated with increasing age, minority race and BMI were greater among HIV infected veterans. HCV co infection and nucleoside and non-

nucleoside reverse transcriptase inhibitor therapy were associated with a higher risk of diabetes in HIV infected veterans.

Prior to the 1990s, diabetes in SSA was regarded as a rare disease (Mbanya & Ramiaya, 2006). Since then however, diabetes has been rendered- by demographic and epidemiological transitions and urbanization - as one of the Non-Communicable Disease burdens in SSA. In SSA, there are an estimated 10.4 million individuals living with diabetes which represents 4.2 percent of the population living with diabetes globally (IDF, 2006). This figure is estimated to increase to 18.7 million which represents 80% increase by 2025, with the prevalence being higher in urban areas.(Kengne, Amoah &Mbanya, 2005; IDF, 2006). Rapid urbanization and aging are expected to increase the diabetes prevalence as indicated by some studies (Kengne, et al., 2005).

In SSA, like other parts of the world, the prevalence for Type 2 diabetes is higher than type 1 diabetes (Beran &Yudkin, 2006). Prevalence for type 2 diabetes in Africa is currently estimated to be about 2.8% with countries like Ethiopia and Malawi having prevalence of less than 2% while South Africa and Ghana have prevalence above three percent (Gill, et al., 2009; IDF). In SSA, there are consistent disparities that have been reported in the prevalence of type 2 diabetes in urban and rural settings with rural areas recording lower rates (Gill, et al., 2009; Mbanya, et al., 2006; Motala, Omar & Pirie, 2003). In the rural areas of Cameroon in 1997, Guinea in 2007 and South Africa, the crude prevalence rate of type 2 diabetes was found to be < 1%, 4% and 4.8 % respectively (Balde, Camara, Diallo, Kake & Bah-Sow, 2006; Baldé, Baldé, Barry, Kaba,

Diallo, Kaké, Camara et al., 2007; Mbanya, Ngogang, Salah, Minkoulou & Balkau, 1997; Mollentze, Moore, Steyn, Joubert, Steyn, Oosthuizen & Weich, 2008). However, Elbagir Eltom, Elmahadi, Kadam &Berne (1998) found no rural-urban differences in some cases such as Sudan.

2.3.4 Lipodystophy and obesity

HIV associated lipodystrophy is characterized by uniform subcutaneous and peripheral fat loss, with relative preservation or increase in visceral fat, resulting in relative central adiposity, as well as fat accumulation in the neck and dorsocervical region (Grinspoon et al., 2005). Prospective studies have shown that, these abnormalities are clinically evident in 20-35% of patients 12-24 months after starting HAART. The use of PIs and NRTIs in particular Didanosine and Stavudine are associated with the development of lipoatrophy (Grinspoon et al., 2005). Lipodystrophy in HIV patients is commonly associated with metabolic syndrome, including; insulin resistance, impaired fasting glucose tolerance, elevated triglycerides, low HDL cholesterol, and hypertension. Metabolic syndrome appears to be highly prevalent among HIV patients (Hsue et al., 2005).

McCormic and colleagues,(2014) prospectively reviewed nutritional demographics of clinic attendees at an urban European HIV clinic during four one-month periods at three-yearly intervals (2001, 2004, 2007, and 2010) and in two consecutive whole-year reviews (2010–2011 and 2011–2012). A sub-study of 50 HIV-positive African female patients investigated body size. A dramatic rise in the prevalence of obesity (BMI>30kg/m2), from 8.5 (2001) to 28% (2011–2012) for all clinic attendees, of whom 86% were on antiretroviral treatment was reported. Women of African origin were most affected, 49%

being obese, with a further 32% overweight (BMI 25–30kg/m2) in 2012. Clinical factors strongly associated with obesity included female gender, black African ethnicity, non-smoking, age, and CD4 count (all P <0.001) while greater duration of cART did not predict obesity. Individual weight-time trends mostly showed slow long-term progressive weight gain.

Despite the existence of malnutrition epidemic in SSA the prevalence of obesity and overweight is increasing. Overweight/obesity has been identified as major modifiable CVD risk factor (Agyemang, Owusu-Dabo, de Jonge, Martins, Ogedegbe& Stronk, 2009; Assah, Ekelund, Brage, Corder, Wright, Mbanya, & Wareham, 2009; Kimokoti, et al., 2008; Rotimi, Cooper, Ataman, Osotimehin, Kadiri et al., 1995). In SSA there are concerns that proper anthropomorphic measures and cut off for BMI need to be established. The WHO cut offs that are already established have been used in majority of the studies (Kamadjeu, Edwards, Atanga, Kiawi, Unwin & Mbanya, 2006).

A higher CVD risk has been reported in individuals with increased waist to hip ratio and abdominal obesity. Abdominal obesity was found to be positively associated with metabolic syndrome in a Benin study. In a meta-analysis conducted in West Africa, prevalence of obesity was recorded at 10% (Abubakari, et al., 2008a; Abubakari, et al., 2008b). Adults with high waist to hip ratio were found to be at a higher risk of heart failure while obesity was found to an important risk factor for heart failure in a study conducted in Congo (Longo-Mbenza, et al., 2008).

In many countries across SSA obesity has been linked to both wealth and urban residence, with the wealthier persons having more likelihood to be obese or overweight due to transition in nutrition, energy expenditure and other unknown factors (Ntandou, Delisle, Ague & Fayomi, 2009; Bourne, Lambert & Steyn, 2002; Joubert, Norman, Lambert, Groeneward, Schnider, et al., 2007). A study by Sobngwi, Mbanya, Unwin, Porcher, Kengne, & Fezeu (2004) in urban Cameroon reported a link between urbanization and a decrease in physical acitivity as well as dietary habit changes. Other studies, have reported an increase in obesity. Fezue and colleagues (2008) reported that there has been a significant increase in obesity over a 10 year period. This increase was however not significant in the urban area even though there was an increase in waist circumference. A study conducted among rural and urban residents in Kenya found more overweight and obesity in urban than in rural residents (Christensen, Eis, Hansen, Larsoson, Mwaniki, Kilonzo, et al., 2008).

Throughout, SSA there is existence of gender disparities in overweight and obesity (Kamadjeu,et al., 2006; Siminialayi, Emem-Chioma & Dapper, 2008). Compared to men, obesity and overweight status disproportionately affects women. Over the past decade, West Africa's prevalence of obesity in the urban area has doubled with the increase being almost entirely contributed by increase in prevalence in women (Mbanya, 2006). Dugas and colleagues (2009) performed a study on a sample of young adults living in a peri-urban environment in South Africa and reported that approximately half of the women were overweight or obese. In a Tanzanian study, researchers compared the odds of being obese in males and female and found that women had 4.5 the odds of being obese and

were than three times more likely to have a high waist-to-hip ratio compared to men (Njelekela, et al., 2009). Preferred body image may be a factor contributing to obesity in SSA. Senegalese women have been found to prefer to overweight to normal weight BMI. Those women, who already had adequate knowledge about obesity as a CVD risk factor, still required additional education on the role of fruits and vegetables in reducing weight and BMI (Holdsworth and colleagues, 2004) and Holdsworth and colleagues (2006). However, another study by Duda, Jumah, Hill, Seffah & Biritwum (2006) in Accra, Ghana found that, in order to improve their health status, overweight women were willing to reduce their body size. Studies elsewhere suggest a positive association between food insecurity and overweight in women (Townsend, Peerson, Love, and Achterberg & Murphy 2001).

2.3.6 Alcohol, Tobacco and CVD Risk

Alcohol consumption, which is common among HIV-infected people, may influence the risk of CVD. In non-infected adults, moderate alcohol consumption can reduce the risk of coronary heart disease (CHD), heart attacks, and the most common type of stroke, whereas heavy drinking increases the risk of these cardiovascular events. These relationships can be partially explained by alcohol's effects on various risk factors for CVD, including cholesterol and other lipid levels, diabetes, or blood pressure. In HIV-infected people, both the infection itself and its treatment using combination antiretroviral therapy may contribute to an increased risk of CVD by altering blood lipid levels, inducing inflammation, and impacting blood-clotting processes, all of which can enhance CVD risk (Freiberg & Kraemer, 2010). One of the consequences of HIV infection is a thinning of the intestinal walls. Combined with the depletion of the immune cells

attacked by HIV, this effacement allows bacteria living in the intestine or bacterial products to leak across the gastrointestinal mucosa into the blood stream (Balagopal, Philip, Astemborski, Block, Metha, et al. 2008; Brenchley, Price, Schacker, Asher, Silvestri, et al. 2006). This process is called microbial translocation. It may result in increased activation of the immune system, subsequent inflammation, and increased end organ damage, including acute MIs and death. In these patients, hazardous drinking combined with active chronic HIV infection or HIV/HCV co-infection leads to high levels of microbial translocation, which in turn results in increased immune activation. Increased immune activation then contributes to increased thrombosis and excessive blood clotting (hypercoagulability), which ultimately increase the risk of end organ damage for example CHD and acute MI and death.

Co- morbidity has been seen in substance use disorders and CVD. The risk of developing cardiovascular diseases such as heart disease; heart failure; ischemic stroke and acute myocardial infarction increases with alcohol consumption and tobacco smoking. In their study, Ormel and colleagues (2007), found that patients with alcohol dependency in Nigeria were more than two times more likely to have a co-morbid heart disease compared to those who did not abuse alcohol. Similar findings were reported in Jos, Nigeria where more than 24% heart failure patients seen in the hospital had a regular alcohol intake (Laabes, Thacher &Okeahialam, 2008).

The risk of glucose intolerance and diabetes has been associated with increased alcohol consumption. This concurred with a study conducted by Puepet and colleagues (2008) in Jos, Nigeria which reported 50% prevalence in alcohol consumption among 250

households in a study investigating the risk factors for diabetes. Similar results were reported in a Kenyan study among rural and urban participants which indicated that excess consumption of alcohol increased the likelihood of glucose intolerance by four fold among men although the relationship did not hold for women (Christensen, Friis, Mwaniki, Kilonzo, Tetens, Boit, et al., 2009). In relation to heart failure, gender variations have been found in alcohol consumption. A prospective cohort study among Cameroonians found that alcohol consumption was related to increased probability of cardiovascular death and all-cause death though this was only significant among male participants and not female (Kengne & Awah, 2009).

Cardiovascular incidents have been ranked second after injuries or deaths attributable to alcohol in the South African population (Schneider, Norman, Parry, Bradshaw, Pluddemann, 2000). Additionally, Schneider and colleagues (2009) have observed in South Africa, that tobacco and alcohol use are related to low social economic position and poverty while physical inactivity was reported as a common cardiovascular risk factor among the wealthy population. The most serious risk factor for coronary artery disease prevalence is tobacco use (Verdier, et al., 2007). In SSA the prevalence of smoking is increasing among men and women. Males were found to be more likely to smoke than females in research review on tobacco use and smoking research while older males (age 30-49) are more likely to use tobacco products than younger males. There is a reported increase in smoking prevalence with age in women (Townsend, 2006). A study in Dakar by Seck and collegues (2007) reported that 40% of patients coming to hospital for myocardial infarction treatments were smokers. In another study, approximately 20%

of a hospital-based sample of 202 diabetics in Ethiopia was reported to be male smokers (Seyoum, Abdulkadir, Berhanu, Feleke, Worku & Ayana, 1999). Thus, alcohol and smoking behavior among men and those living in low socio-economic contexts increases the risk of developing CVD and suffering poor CVD outcomes.

2.4 Assessment and stratification of cardiovascular risk

The major risk factors for developing cardiovascular disease that have been widely accepted include; smoking, high blood pressure, age, dyslipidemia, sex, and diabetes (Cupples & D'Agostino, 1987). The clustering of CVD risk factors and multiple interactions in promoting vascular risk is also recognized (Jackson, Lawes, Bennett, Milne & Rodgers, 2005). As a result of this knowledge, multivariable risk prediction algorithms that incorporate these risk factors have been developed, which can be used in assessment of the risk of developing atherosclerotic CVD in individual patients by primary care physicians (Anderson, Wolson, Odell & Kannel, 1991; British Cardiac Society, 2005; De Backer, Ambrosioni, Borch-Johnsen, Brotons, Cifkova, et al., 2004; Conroy, Pyorala, Fitzgerald, Sans, Menotti, et al., 2003; Webcast, 2001; Ridker, Buring, Rifai &Cook, 2007; Woodward, Brindle &Tunstall-Pedoe, 2007). This may also predict specific components of CVD such as stroke, coronary heart disease, peripheral vascular disease, or heart failure (Webcast, 2001; Assmann, Cullen &Schulte, 2002; Anderson, et al., 1991; Ferrario, Chiodini, Chambless, Cesana, Vanusso, et al., 2005; Zhang, Attia, D'Este, Yu & Wu, 2005). In the estimation of absolute CVD risk multivariable assessment has been advocated since it can act as a guide for treatment of risk factors (De Backer,et al., 2004; Jackson, et al., 2005).

The Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults has incorporated the Framingham formulation for predicting coronary heart disease (CHD) (Webcast, 2001). The Framingham CHD risk assessment tool has been validated in both whites and blacks in the United States and is transportable (with calibration) to culturally diverse populations in Europe, the Mediterranean region, and Asia (Webcast, 2001; Ridker, et al., 2007). However the Framingham assessment tool has not been validated in the African population and in particular SSA.

The methods that are used to assess total cardiovascular risk are varied and use different sets of guidelines. There are those that are published jointly by the ESH/ESC (Mancia,et al., 2007) and those from the World Health Organization/International Society of Hypertension (WHO/ISH, 2003), that were written based on criteria from multiple studies, including the Framingham Heart Study (Anderson, et al., 1991). These guidelines have categorized cardiovascular risk according to the presence of other risk factors, target-organ damage and associated clinical conditions, such as a history of cardiovascular disease. Hence, patients can be stratified according to the severity of hypertension and the presence of other risk factors such as having a low (< 10%), medium (15-20%), high (20-30%) or very high (> 30%) level of added risk of cardiovascular morbidity or mortality within the next 10 years.

Moreover, the presence of additional risk factors, target organ damage or associated clinical conditions may expose patients to high or very high risk of cardiovascular disease, even when blood pressure is normal or high-normal (systolic blood pressure

(SBP) 130-139 mmHg and diastolic blood pressure (DBP) 85-89 mmHg). Several studies have also shown an association between increased cardiovascular risk factors and poor prognosis in hypertensive patients. In one such study, 60,343 male patients with hypertension with or with no risk factors associated were followed for fourteen years. Data from a matched group of 29,640 normotensive men without associated risk factors for cardiovascular disease (Thomas, Rudnichi, Bacri, Bean, Guize & Benetos, 2001) was also analyzed. Cardiovascular mortality increased 5-fold in younger hypertensive patients (age < 55 years) with one or two additional risk factors and 15-fold in younger patients with more than two associated risk factors compared with normotensive individuals. A 3-fold increase in cardiovascular mortality was observed in older hypertensive patients (age ≥ 55 years) with up to two associated risk factors, compared with their normotensive counterparts, whereas older hypertensive individuals with more than two risk factors showed a 4.5-fold increase.

Friis-Moller and colleagues (2010) in a study to predict the cardiovascular disease risk in HIV infected individuals in a D.A.D study conducted in Europe and Australia confirms that the uncalibrated Framingham equation tends to over predict the risk of events, particularly for CHD and CVD endpoints. However, even the recalibrated Framingham equation, which has been forced to sum to the total observed number of events in the DAD cohort, does not predict well in certain subgroups. In particular, the Framingham tended to under predict risk compared with the DAD equations in women (for MI and CHD outcomes), in former smokers and in diabetic patients, but over-predicted in never smokers.

Framingham risk score was developed for a non-HIV-infected and non-ART exposed American population, aged 30–74 years, followed for up to 12 years from a baseline between 1968 and 1975. The HIV-infected population in the DAD Study is slightly younger, with diverse geographical distribution (although predominantly European), and the majority is ART exposed

Estrada & colleagues (2015) in another study to determine cardiovascular risk factors and lifetime risk estimation in HIV-infected patients under antiretroviral treatment in Spain highlighted discrepancy between the high prevalence of cardiovascular risk factors and the relatively low short–term risk estimations using traditional scales, issue that is being increasingly recognized also for HIV negative, general populations (Berry, Liu, Folsom, Lewis, Carr, et al.,2009; Hulten, Villines, Cheezum, et al., 2014). In our study, this figure is even higher, with almost 70% of subjects with a low short-term estimation who have at least one major risk factor and are classified as having a high risk according to lifetime estimation. Even when the Framingham/Regicor scale estimation is low for most of our patients (77% with v5% at 10 years), there is a surprisingly elevated frequency of some relevant CVR factors. Since the influence of tobacco smoking and dyslipidemia is undisputed in CVD risk, this suggests that the relatively low age of patients partially offset the risk. Because of the dependency on age, these relatively young HIV-infected patients would be considered at low short-term risk, but actually they are on risk in the long-term in the case that these factors are not modified.

These discrepancies have been identified in other epidemiological studies conducted in the general population in people under age 50 (Marma & Lloyd-Jones, 2009). Atherosclerosis occurs over decades and is mainly related to long-term and cumulative

exposure to causal, modifiable risk factors (Ferreira-Gonzalez , 2014) Thus, long-term perspective of risk assessment and prevention must be considered, especially among younger individuals

2.5 Dietary intake and Nutrition status of people living with HIV

The complex interaction between Human Immunodeficiency Virus (HIV), infection and immune function exists, with a dominant effect of HIV infection on nutritional status (Macallan, 1999). Immunological deficiencies are as a result of sub optimal nutritional status (Macallan, 1999). Nutrient deficiencies lead to suppressed immunity and increased susceptibility to infections with a resultant loss of immune cell function which then allows intrusion by several different infectious agents. As a result, the body's ability to fight disease is reduced and subsequently opportunistic infections are acquired. If macronutrient intake is insufficient to meet metabolic needs, protein-calorie malnutrition (PCM) and deficiencies of micronutrients result (Baum & Shor-Posner, 1998). These deficiencies may impair both the function of immune-related enzyme systems and the synthesis of molecules necessary for the immune response (Friis, 2006). In HIV and AIDS, the presence of malnutrition strongly predicts patient survival independent of CD4 (cluster of differentiation) T-lymphocyte counts (Friis, 2006). Nutritional quality of the diet improves with consumption of greater food diversity (Hatluy, Torheim & Oshaug, 1998; Shimbo, Kimura & Imai,1994).

However dietary diversity has widely been associated with high socioeconomic status (Slattery, Berry, Potter& Caan, 1997); and this could be explained by the fact that people with high income may have the economic ability to purchase different types of foods from different food groups, as opposed to the low income earners who might stick to the

few cheaper foods available, and this limits diet diversification among the poor people (Friis and Michaelsen, 1998). Monthly income may be a strong and significant predictor of diet diversity among HIV patients. Studies have shown that diet diversity correlates with nutrient adequacy. Hatluy et al. (1998) and Slattery et al. (1997) reported that nutritional quality of the diet improves with consumption of greater food diversity. A study by Stewart (2003) reported that daily servings of the same food from each food source may not be enough, but that one should choose variety within food sources because the characteristic nutrients in each group vary greatly for individual foods.

2.6 Cardio protective nutrients and dietary factors in peanuts

Nuts are from different plant families and are classified as tree nuts (a one-seeded fruit with a hard shell) or peanuts (a member of the leguminous family). Peanuts are also called ground nuts because they develop in the soil. Despite their diversity tree nut varieties share common nutritional characteristics with peanut. Peanuts are nutrient dense foods and also contain a high fat content half of which is unsaturated, which includes monounsaturated fatty acids (oleic) and polyunsaturated fatty acids (Kris-etherton, 1999a).

The fatty fraction of nuts also contains a sizable amount of plant sterols, with anti-oxidant (Vivancos & Moreno, 2005) and cholesterol lowering properties (Segura, Javierre, Lizarraga & Ros, 2006). They are also rich sources of other bioactive macronutrients that include protein and fibre. They contain high amounts of L-arginine which is the amino acid precursor of the endogenous vasodilator nitric acid (Huynh & Chin-Dusting, 2006). There are few studies that have been done on the effect of processing on proximate

composition of peanut. This study sought to determine the effect of dry roasting on proximate composition of Red Valencia which is most commonly consumed variety in Kenyan.

Epidemiologic studies on prevention of diabetes, coronary heart disease and sudden deaths have found that unsaturated fatty acids contribute to the beneficial associations of frequent nut intake and decreases in other CVD risk factors. Nuts form a complex food matrix that are sources of other bioactive compounds which include fiber and protein ; micronutrients, such as potassium, calcium,magnesium, and tocopherols; phytochemicals, such as phytosterols and phenolic compounds; arginine and resveratrol (Kris-Etherton, Yu-Poth , Sabaté, Ratcliffe, Zhao & Etherton 1999). The beneficial effects of nuts on cardiovascular diseases can be explained by the composite and individual cardio protective nutrients in the nuts. Much remains to be explained about the underlying mechanism of action that mediates the multiple effects that have been reported to date. Nuts (tree nuts and peanuts) are nutrient dense foods with complex matrices rich in unsaturated fatty and other bioactive compounds: high-quality vegetable protein, fiber, minerals, tocopherols, phytosterols, and phenolic compounds (Ros, 2010)

Nuts contain sizeable amounts of folate (Segura, et al.,2006), a B-vitamin necessary for normal cellular function that plays an important role in detoxifying homocysteine, a sulfur-containing amino acid with atherothrombotic properties that accumulates in plasma when folate status is subnormal (Welch & Loscalzo,1998). Nuts are also rich sources of antioxidant vitamins (e.g., tocopherols) and phenolic compounds, necessary to

protect the germ from oxidative stress and preserve the reproductive potential of the seed, but also bioavailable after consumption and capable of providing a significant antioxidant load (Blomhoff, Carsen, Andersen & Jacobs., 2006). Remarkably, in all nuts most of the antioxidants are located in the pellicle or outer soft shell, as shown for peanuts (Lou 2004), and 50% or more of them are lost when the skin is removed (Blomhoff,et al., 2006). Nuts are cholesterol-free, but their fatty fraction contains sizeable amounts of chemically related non cholesterol sterols belonging to a heterogeneous group of compounds known as plant sterols or phytosterols (Segura, et al.,2006) . They are non-nutritive components of all plants that play an important structural role in membranes, where they serve to stabilize phospholipids' bilayers just as cholesterol does in animal cell membranes (Hartmann, 1998).Phytosterols interfere with cholesterol absorption and thus help lower blood cholesterol when present in sufficient amounts in the intestinal lumen. The mechanism of action of phytosterols has been linked to their hydrophobicity, which is higher than cholesterol because of a bulkier hydrocarbon molecule and entails a higher affinity for micelles than has cholesterol. Consequently, cholesterol is displaced from micelles and the amount available for absorption is limited (Garrido, Monagas, Gomez-Cordoves, et al., 2008). In all probability the phytosterol content of nuts contributes to their cholesterol-lowering. Compared to other common foods, nuts have an optimal nutritional density with respect to healthy minerals, such as calcium, magnesium, and potassium. Like that of most vegetables, the sodium content of raw or roasted but otherwise unprocessed nuts is very low, ranging from undetectable in hazelnuts to 18 mg/100 g in peanuts (Segura, et al.,2006) . A high intake of calcium, magnesium and potassium, together with a low sodium intake, is associated with protection against bone

demineralization, arterial hypertension, insulin resistance, and overall cardiovascular risk (Cordain, Eaton, Sebastian, Mann, Lindeberg, et al.,2005).

2.7 Effect of peanut in reducing cardiovascular risk

A pooled analysis of four studies in the U.S found that subjects in the highest intake group for nut consumption had an approximately 35% reduced risk of CHD incidence (Albert, Gaziano, Willett & Manson, 2002; Ellsworth, Kushi & Folsom, 2001; Fraser, Sabate, Beeson &Strahan, 1992; Hu, Stampfer, Manson, Rimm, Colditz, et al.,1998). The relative risk for total CHD based on a multivariate analysis was 0.65 (CI: 0.47-0.89). The relative risk of fatal CHD for individuals consuming nuts 5+ times per week was 0.61 (CI: 0.35-1.05) while the relative risk for nonfatal myocardial infarction was 0.68 (CI: 0.47-1.00) (Hu,et al., 1998). Most importantly a dose-response relationship was reported for nut consumption and all relative risks of CHD. One particular study conducted by Albert et al., (2002), an inverse association was noted between consumption of nuts and total coronary heart disease death. That study also found a 47% reduction of risk of sudden cardiac death in men who consumed nuts two or more times in a week.

Hu, et al., (1998) also observed a relative risk of CHD of 0.66 (CI: 0.46-0.94 in subjects who consumed peanuts 2+ times per week compared to the relative risk of 0.79 (CI: 0.50-1.25) in those who consumed tree nuts 2+ times per week. Decreasing inflammatory markers and thus the improved inflammatory status is one of the mechanisms by which peanut and tree nuts consumption may reduce the CHD risk. Inflammation is a key process in atherogenesis, among other diseases. An inverse association between

inflammatory markers and consumption of nuts and seeds was reported Jiang and colleagues (2006) in Multi-Ethnic cross sectional Study of Atherosclerosis. This is significant due to the fact that inflammation contributes to all phases of atherosclerotic disease, ranging from initial recruitment of circulating leukocytes to inducing endothelial dysfunction and to plaque rupture.

Epidemiological studies and clinical trials have demonstrated benefits of nuts and peanut consumption on CAD risk and associated risk factors (Djousse, Rudich & Gaziano, 2009; Ros, 2009). An approximately 35 % reduction in risk of CAD incidence was reported among nut consumers in a pooled analysis of epidemiology studies.

Clinical studies have also evaluated the effects of many different nuts on lipids and, lipoproteins, including oxidation, inflammation and vascular reactivity, and have found evidence that consistently shows a beneficial effect on these CAD risk factors (Djousse et al., 2009; Ros, 2009; Ros, Nunez & Perez-Heras, 2004) Jiang, Jacobs and Mayer-Davis (2006) in a prospective Multi-ethnic study involving 6000 participants, found that the concentration of soluble inflammatory markers decreased in nut consuming participants. The highly sensitive C-reactive protein reduced from 1.97mg/L in subjects not consuming nuts to 1.71mg/L in subjects consuming nuts more than five times a week. A substantial fraction of the fat contained in peanut is made up of monounsaturated fatty acids which are not an oxidation substrate. The fatty acids hence enrich the lipoprotein lipids whose susceptibility to oxidation might decrease with peanut intake (Reaven & Witzum, 1996).

Sabate, Keiji and Ros (2010) investigated the effect of nut consumption on blood lipids levels in 25 pooled intervention metanalysis done in 7 countries among 583 men and women with normalipidemia and hypercholesterolemia, who were not taking lipid lowering medications. It was found that with a mean consumption of 67g of nuts, total cholesterol concentration showed a mean change of 5.1%, and 7.4% LDL-C change. Ratio of LDL to HDL cholesterol concentration changed by 8.3% while ratio of total cholesterol to HDL-C changed by 5.6%. Triglyceride levels were reduced by 10.2% in subjects with TG levels of at least 150mg/dl.

Kris-Etherton et al. (1999b) also showed beneficial effects of peanut consumption on atherogenic index of plasma on blood lipid concentrations when consumption was combined with a high MUFA, low SFA diet in normocholesterolemic subjects. In both studies, MUFA intakes largely substituted SFA intake. Alper and Mattes (2003) reported that a moderate increase in MUFA intake without a concomitant decrease in SFA, did not appear to sufficiently decrease TC and LDL-C in normocholesterolemic individuals, and that peanut consumption may have beneficial effects on diet composition even when the background diet is not controlled.

Clinical studies have also indicated that tree nuts especially walnuts decrease LDL-cholesterol by 3-19% when added to habitual, lower fat and average American diet as reference diets (Griel & Kris-Etherton, 2006). In peanut interventions, up to 11% reductions in total cholesterol and 14% reductions in LDL-C have also been reported

when compared to the same reference diets (Kris-Etherton, et al., 1999a; Lokko, Lartey, Armar-Klemesu & Mattes, 2007). This is consistent with a more general literature (Anderson, Spencer & Hamilton, 1990; Ripsin, Keenan & Jacobs, 1992; Anderson, Johnstone & Cook-Newell, 1995; Romero, Romero &Galaviz, 1998) that report the degree of reduction in plasma cholesterol concentrations in response to peanut consumption to be inversely related to baseline concentrations (Kris-Etherton,et al., 1999a). Nut consumption has shown limited impact on body weight (Mattes, Kris-Etherton & Foster, 2008). Lack of or negative association between nut consumption and body mass index has been reported in some epidemiological studies while some clinical studies have supported a lack of association under several conditions (Mattes, et al., 2008). Through improved dietary compliance, nuts may in fact aid in weight loss (McManus, Antinoro & Sacks, 2001). Reducing weight may lead to a reduced cardiovascular disease risk since central obesity is an independent CVD risk factor (Sowers, 2003). Thus, moderate consumption of nuts may be a functional component in a cardio protective diet (Van Horn, McCoin, Kris-Etherton, 2008). There is limited information available on the effect of peanut on HIV associated dyslipidemia. This study focused on the impact of peanut supplementation with regular diet and peanut supplementation combined with nutritional counseling on healthy diet in HIV infected adults with normal and hyperlipidemia.

2.8 Effect of nuts on weight and BMI.

Development and maintenance of obesity has consistently been associated with intake of dietary fat, although the issue has been considerably controversial. Nuts are a rich source of fat and therefore their potential impact on body weight has raised some concerns that

even the epidemiologic studies have not supported. A large cohort of women followed for 16 years found a slight decrease in the body mass index (BMI) even as the consumption of nut increased. After adjustment for potential confounders, their average weight gain across nut consumption categories was not significantly different (Jiang, Manson, Stampfer, Willet & Hu, 2002).

Other studies have found no net weight gain when nuts were consumed as a replacement food. The same results have been reported even when nuts were been added to diet, even though the intake of total energy was substantially increased (Morgan & Clayshulte, 2000; Morgan, Horton, Reese, Carey, Walker, & Capuzzi, 2002). No weight gain was reported when 48 g of walnuts were added to the diet for six weeks despite increase in energy intake by 1661 kJ/day (Almario Vonghavaravat, Wong & Kasim-Karakas, et al., 2001). Since the nut intervention duration is relatively short in most of these trials, the longterm effect of nut intake may not be indicated. However an isolated intervention study showed a negative effect on body weight. A slight but significant increase in body weight (0.9 kg for men, 0.3 kg for women) was observed when normal weight participants were given 100g of almonds to add to their usual diet for a period of four weeks (Lovejoy, Most, Lefevre ,Greenway & Rood, 2002).

One cross over study specifically evaluated the role of nuts on body weight. In this study, a free daily almond supplement which gave about 15% of the individuals' daily energy intake was given for six months with no nutrition advice. There was no significant change in waist to hip ratio or body weight. Weight gain was only seen in those with

lowest BMI while the most obese participants lost small amounts of weight (Fraser, et al., 2002). In another 18 month trial, McManus and colleagues (2001) compared the impact of a Mediterranean-style diet that is high in nuts and moderate in fat (35% of the daily energy) and that of a standard low-fat diet that consist of 20% of the daily energy) for weight loss in 101 overweight adults. There was a decrease in waist circumference and body weight observed in the moderate-fat group, whereas the low-fat group regained weight progressively. Most of the evidence suggests that although nuts are fatty and high-energy foods, they do not lead to weight gain. This could be explained by the mechanisms such as satiety, dietary compensation, efficiency of energy absorption and increased energy expenditure.

2.8.1 Efficiency of energy absorption

There is evidence of incomplete absorption of energy from nuts. Elevated loss of fecal fat with nut consumption has been consistently reported. An earlier trial by Levine & Silvis (1980) revealed that 17.8% of the lipid load was lost in stool when 10 participants were given peanuts to provided 95% of the energy from fat for 6 days. Fecal fat loss of l7.0% and 4.5% for peanut butter and peanut oil loads, respectively were also reported. A more recent trial indicated that fecal fat content was significantly higher when whole peanuts were consumed relative to other forms such as butter, and flour when customary diet containing 70 g/d of whole peanuts, peanut butter, peanut flour, or peanut oil was given to 93 participants (Traoret, Lokko, Cruz, Oliveira, Costa, et al., 2008). This corresponded to an energy loss of approximately 155 kJ/d or 9.4% of the load or 12% of the peanut lipid. The percentage of fecal fat increased from 2.9 to 8.3%, in a trial where the fat content of the diet of 6 individuals was increased from 30% of energy to 43% of

energy by the addition of peanuts for 4 weeks. A similar significant increase in excretion of percentage fecal fat was reported when subjects were given a four week pecan rich diet. These subjects required more energy to stabilize their body weight (Haddad & Sabate, 2000). This low level of fat absorption could be due to various nut components like fibre or due to the structure of lipid-storing granules in nuts.

2.8.2 Satiety and dietary compensation

The low glycemic index of nuts has been proposed as a mechanism by which they modulate appetite (Rajaram & Sabate, 2006). Nuts have been found to exert some kind of satiating effect as suggested by some authors. This was demonstrated in a 6-month clinical trial by Fraser et al. (2002) who reported a reduction in intake of other foods displaced between 54 and 78% of the extra energy provided by the almond supplement. Components such dietary fibers are suspected to contribute to this effect (Marlett, McBurney & Slavin, 2002).

2.8.3 Increased energy metabolism

The effect of energy metabolism by the composition of the peanuts is due to energy compensation availability. Human and animal studies have demonstrated a higher fat oxidation, higher diet induced thermo genesis and less body fat accumulation with unsaturated fat consumption compared to saturated fat intake (Jones, Ridgen, Phang & Birmingham, 1992; Takeuchi, Matsuo, Tokuyama, Shimomura & Suzuki, 1995). An increase in resting energy expenditure by 11% has been reported after regular peanut consumption for 19 weeks (Alper & Mattes, 2002).

2.9 Effect of nuts on glycemic control

The fatty acid composition of nuts is suspected to play a role in modifying insulin resistance, and therefore, the risk for type-2 diabetes. Specific types of fatty acids have been found to be better predictors of the risk of type-2 diabetes than total dietary fat intake (Hu, van Dam & Liu, 2001). Studies have shown that a higher intake of n-3 PUFA is linked with lower risk of type-2 diabetes, while on the other hand, glycemic control is adversely affected by a higher intake of saturated and trans fatty acid hence increasing the risk of type-2 diabetes (Hu et al., 2001; Vessby et al., 2001).

The mechanisms by which specific fatty acids affect insulin sensitivity are not clear. It has however been shown that the fatty acid composition of the phospholipids in the skeletal muscle cell membranes is directly related to insulin sensitivity in humans. Ion permeability and cell signaling could be influenced by the action of a specific fatty acid in cell membranes, which influences insulin action through altering insulin receptor binding or affinity (Storlien et al., 1996). Further, monounsaturated fatty acids have also been known to improve beta cell efficiency by enhancing secretion of glucagon-like peptide-1 that is known to help in regulating postprandial glucose clearance and insulin sensitivity (Rocca, LaGreca, Kalitsky & Brubaker, 2001). Furthermore, the non-lipid components of nuts may also contribute towards lowering the risk of type-2 diabetes.

Earlier prospective studies (Paolisso, Scheen, D'Onofrio & Lefebvre, 1990; Salmeron, Manson, Stampfer, Colditz, Wing & Willett, 1997) have shown that, with higher intakes of fiber and magnesium and the inclusion of low glycemic index foods, the risk of type-2 diabetes is lowered. Salas-Salvado´ and colleagues (2006) found that by supplementing the diet with nuts it provided a significant amount of fibre (4-12 g/100 g). Nuts also

provide a good source of magnesium (120–300 mg/100 g), and a higher magnesium intake is inversely associated with diabetes risk (Lopez-Ridaura, Willett, Rimm, Liu, et al., 2004). Nuts are a whole food and therefore it is only reasonable to expect synergistic interaction between many of the nutrients and non-nutrients in nuts as they work to influence insulin resistance. There is limited information available on the effect of peanut on weight, BMI fasting blood glucose and blood pressure in HIV infected adults in Kenya. This study focused on the impact of peanut supplementation with regular diet and peanut supplementation combined with nutritional counseling in healthy diet on the nutritional status of HIV infected adults.

2.10 Summary of literature review

Factors affecting cardiovascular risk in HIV-infected individuals seem to be similarly associated with cardiovascular risk in the general population; exposure to antiretroviral therapy has also been shown to unfavorably affect the risk of cardiovascular disease. Factors linked to increased risk of CVD development have been classified as modifiable and non-modifiable. These studies have been done in developed countries and there is little or inadequate information in the study area. There is inadequate information on tools for estimating cardiovascular risk factors in the HIV population and in particular tools that are validated in the African population. Epidemiological studies in developed countries have shown that frequent nut consumption decreases the risk of CHD. However these peanut studies have only been performed in either healthy adults or diabetic dyslipidemic subjects in combination with low fat diet and not in HIV infected subjects.

CHAPTER THREE: METHODOLOGY

3.1 Research design

The research design was a randomized cross-over clinical trial on free living adults living with HIV with normal lipids and hyperlipidemia status. Each patient who participated in the intervention received two treatments sequentially and hence each was considered as his/ her own control. This design helped eliminate patient variation.

3.2 Study variables

The primary outcomes were serum total cholesterol, LDL cholesterol, triglycerides and HDL cholesterol. Independent variables included body weight, BMI, waist circumference, Hip circumference, blood pressure, fasting blood glucose, physical activity level, age, smoking, energy and nutrient intake and Framingham's scores.

3.3 Study location

Nyeri Level - 5 Hospital is a referral hospital situated in Nyeri South Sub county, Nyeri Municipality Division, Mukaro location in Nyeri county, Kenya (-0°25'33"N 36°57'49"E). The hospital has a bed capacity of 384. This hospital serves patients mainly from Nyeri South Sub County. The comprehensive care clinic (CCC) has since inception had a population of 5000 patients of whom 3150 are on HAART.

3.4 Target population

The study population included adult patients attending Comprehensive Care Clinic at Nyeri Level 5 Hospital who are living with HIV with normal lipid and hyperlipidemia.

3.4.1 Inclusion criteria

The study included outpatients of either gender, aged at least 18 years, with normal or high lipidemia status. The patients had to have been on ARVS for at least 2 months and willing to be available for follow up for the six months study period in the CCC.

3.4.2 Exclusion criteria

The study excluded all subjects with known hypersensitivity to peanut and who reacted to peanut after a skin test performed by a clinician, pregnant and breastfeeding. Those on lipid lowering therapy and rigorous exercise were excluded. Also excluded were those with history of irritable bowel disease or diverticulitis that could be exacerbated by daily peanut intake. Habitual peanut or tree nut consumers who were not willing to discontinue the intake of all peanut and/or tree nuts for 6 weeks prior to their first scheduled clinic visit were excluded. Patients with liver disease, renal disease and/or severe dyslipidemia (TG >4.52 mmol/l or TC >7.77 mmol/l) were also excluded.

3.5 Sampling technique

3.5.1 Sampling frame

Nyeri CCC had 564 males and 1150 females who were actively taking HAART. An average of 60 adult patients attended the clinic for 4 days a week. Out of the 60, about 90% were on HAART. Recruitment period was 5 weeks. A total of 125 patients were screened.

3.5.2 Sample size determination

Sample size was determined using the following formula by Chow Shao and Wang, (2003).

$$n = \left(\frac{[Z_{\alpha} + Z_{\beta}]^2 \sigma^2{}_m}{2[\delta]^2} \right)$$

The sample size was of a balanced, cross-over design analyzed by the t-test approach hence

α: The probability of type I error (significance level) is = 1.96.

β: The probability of type II error (1 – power of the test) = 1.282

δ: The true difference between the two mean values at which the power is calculated

$\mu_2 - \mu_1$: Margin of equivalence (δ) = the allowable difference = 0.1

Expected variance for LDL- C **(** σ_m^2 **)** = 0.074 (Sabate, Ros Emilio, 2010)

$$\left(\frac{[1.96+1.282]^2 x[0.074]^2}{2[0.1]^2} \right) = 37.7$$

When these values were substituted in the formulae, the sample size was approximately 38 for each group and an attrition rate of 20% was included giving a total of 91 participants.

3.5.3 Randomization procedure

Out of the 125 screened, 95 patients agreed to sign the consent form. Only 85 patients were followed for the entire 6 months, hence the dropout rate was 10.5%. The recruited participants were then randomized into two groups. Group 1 started with TI and crossed over to TII, while group 2 started with TII and crossed over to TI after the washout period.

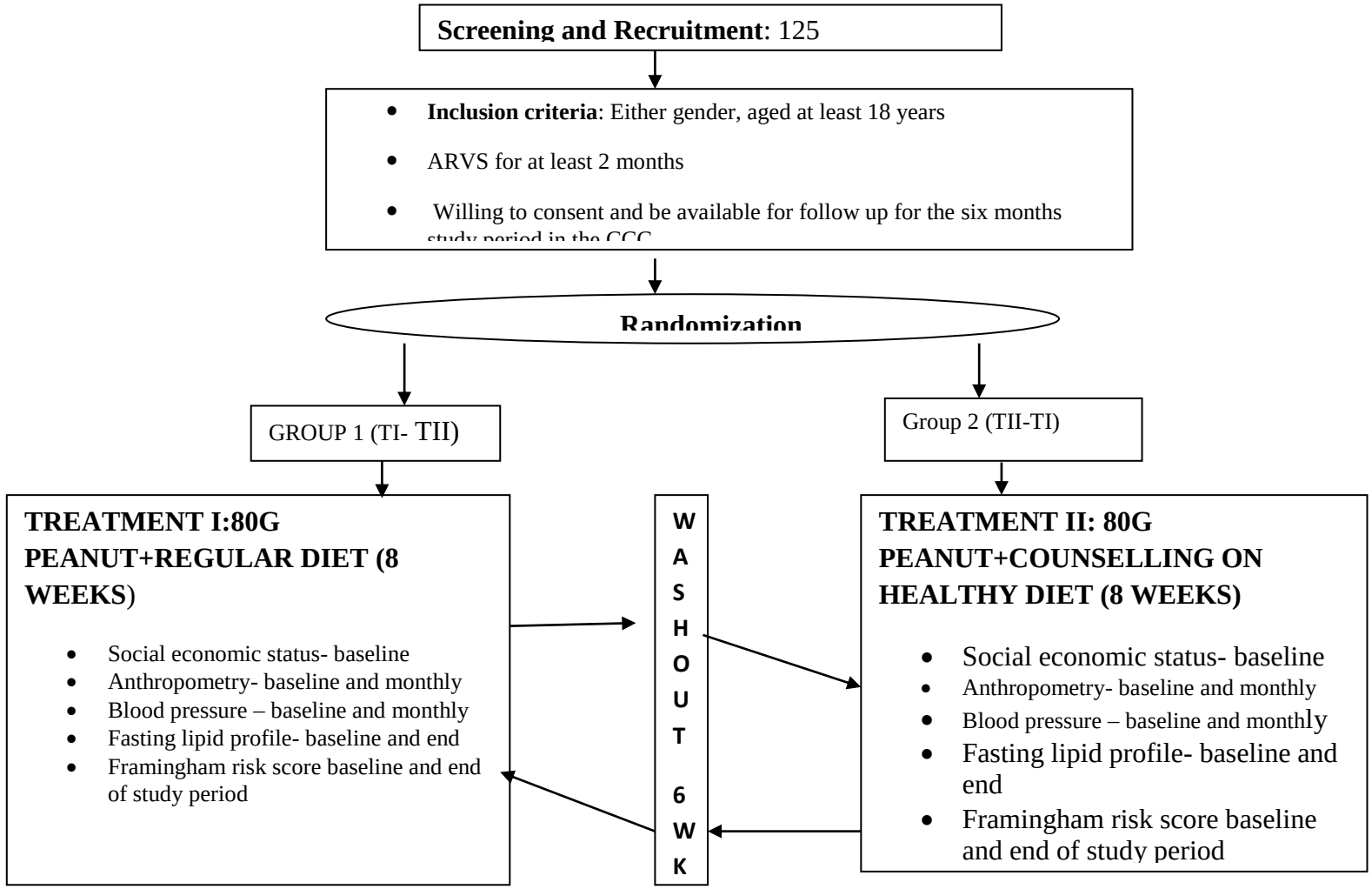

Figure 3.2 Randomization procedure

3.6 Intervention procedure

Five weeks were used for recruitment of the participants. The overall study duration was 27 weeks. Freshly roasted unsalted peanuts were packaged in separate bags of 80g each which was the daily serving. The Red Valencia variety was provided. A package containing thirty days servings was given to the participant to carry home and consume them every day for four weeks after which they would come for another batch for consumption in the following four weeks. The peanuts were consumed as part of participants snack or with the meals.

Before the study began, all participants were given five peanuts each and observed for any allergic reaction. In case of symptoms such as hives, itching, and cramping, acute epigastric pain or respiratory appear, the participants were given anti histamine, Cetrizine 5mg, immediately and withdrawn from the study. Peanuts were sourced from Kenya nut, processed and packed by Nutrinut factories, Thika and assessed for aflatoxin before each batch was released at the Kenya Bureau of Standard (KEBS) laboratory. Participants were also given an extra coded bag of peanuts containing 200g to share with family and friends to improve compliance. Compliance was assessed using 24 hour recall done on randomly selected days.

The participants who met the eligibility criteria and signed consent were randomly assigned to a two arm study. Group 1 started with treatment I and then crossed over to treatment II while group 2 started with treatment II and then crossed over to treatment I. Six weeks washout period Jenkins, Kendall, Marchie, Faulkner, Wong, de Souza, et al., (2005) was included between treatments during which period the subjects were asked to eat their regular diet.

During treatment I (TI) the participants were required to go on consuming their regular diet supplemented with 80g of peanuts daily for eight weeks. Their nutritional status was assessed at baseline and again after fourth and eight weeks. The lipid profile was done at baseline and at end of eight weeks. During the second treatment (TII) the participants were counseled on a healthy diet at baseline and then after four weeks before they picked the next batch of peanut. Counseling covered aspects such as substituting the saturated fat with unsaturated fats, consumption of high fibre foods and inclusion of all food groups in their proper amounts. The participants were also provided 80g of peanuts daily for eight weeks. Their nutritional status was assessed at baseline and again after fourth and eight weeks. The lipid profile was done at baseline and at the end of eight weeks.

3.7 Data collection tools

A structured questionnaire was administered at baseline to collect social economic and demographic information (Appendix B). Socio economic status questionnaire was adopted from KDHS 2008-2009.

Dietary intake was assessed using a 24- hour recall (Appendix C) that was conducted on three randomly selected days during baseline, treatment I and treatment II. This was to help measure

nutrient intake. During baseline, the participants were trained to estimate food portions using diagrams or by weighing. A 7- day food frequency (Appendix D) was used to establish consumption of peanut. This was administered at baseline and during each visit.

Medical history questionnaire (Appendix E) was administered by the clinician at baseline to assess for eligibility of the participant and it included detailed information about medication and nutrient supplement usage.

Framingham risk scores (Appendix F) was used to estimate the 10 year risk of developing myocardial infarction (NCEP ATP 111, 2001). The tool uses the traditional CVD risk factors and the total scores to determine the patient's risk of developing MI. This tool was administered at baseline and at the end of each treatment period.

Anthropometric measurements were taken using different equipment. Body weight and body composition were measured using a bio electric impedance machine, Height was measured using a standiometer while waist and hip circumference were measured using an elastic tape.

Fasting blood glucose was measured using a glucometer while blood pressure was measured using an automatic blood pressure machine.

Data collected at baseline included social demographic, medical history, anthropometry, 24 hour recall, seven day food frequency, blood glucose, blood pressure CD4 count and lipid profile. Anthropometric data and food frequency questionnaire was administered every month while

lipid profile was done at 8th, 14th and 22nd week. CD4 count data was obtained from the patients records.

3.8 Pretesting

Research instruments were pretested on 10% of the sample. Pre testing of the research instruments was done in Thika level 5 Hospital CCC because the patients have similar characteristics

3.9 Reliability and Validity

Test-retest method reliability was used to measure for the reliability of the questionnaires. The questionnaires were administered to the same respondent at two different times. Their scores were then correlated and a test-retest coefficient of reliability was obtained as 0.87. This meant that the study instruments were good and agreed with George and Mallery (2003). Validated questionnaires for Social economic status, 24-hour recall and 7- day food frequency were used for the study. A validated Framingham risk scores questionnaire was adopted from US Department of Health and Human Services. Medical assessment questionnaire was validated for content by a panel of professionals from Kenyatta University Food, Nutrition and Dietetics; Biochemistry departments, and physicians from Nyeri Level Five Hospital.

3.10 Training of the research assistants

The research team included three nutrition officers, a laboratory technologist and a clinician. The research team was trained for 3 days. The training for nutritionists included administration of questionnaires, 24- hour recalls and 7-day food frequency questionnaires as well as on counseling on the healthy diet. They were also trained on anthropometric measurements to

ensure accuracy of data. The clinician was briefed on inclusion criteria in order to help in selection of the patients. The laboratory technician was also briefed on the study procedure and on blood sample collection and storage.

3.11 Data collection procedure

3.11.1 Dietary data

The 24 hour dietary recall was administered to 20% of the participants at three random times. The participants were asked to think back to when they woke up previous morning to the time they went to sleep in the evening. They were then asked to try and remember what they ate or drank from the moment they got up the previous day to the time they when went to sleep at night. The item and time was then recorded as the participant recalled what they ate. They were then asked to describe in detail the food they ate, the amount prepared as well as the preparation method used. Standard household measures and weight were used to determine the amounts of ingredients used. The purchases of already cooked foods from the streets were also included. A description of household portion sizes in terms of cups, spoons, bowls, glasses, matchboxes, manual pictures size or centimeters (using ruler) was then entered. The food code and the portion sizes in grams for the particular items were later entered in the Nutri survey software.

A 7- day food frequency questionnaire (FFQ) was used to assess total dietary fat and different types of dietary fatty acids among HIV adults' patients in Nyeri County. A 50 food item FFQ was adopted and focused on dietary cholesterol, total fat, saturated fat, monounsaturated fat and polyunsaturated fat intake for the past one month was developed and validated against a 24-hour recall. The food list was categorized into 12 different food groups and frequency of intakes was

recorded as times per day and per week. Food pictures were used to assist participants. Common household measurement tools such as bowls, cups, match box, plates and etc. were used to illustrate portion size. The FFQ was administered through a face-to-face interview by the research assistant in Gikuyu language, and the duration of each interview took about 10 to 15 minutes.

3.11.2 Anthropometric data

Anthropometric measurements were taken on monthly basis by the research assistants. Height was measured once at baseline in standing position without shoes using a stadiometer (Seca 213) with shoulders are in normal state. Participants were asked to remove their shoes, heavy outer garments, and hair ornaments. The participant was also asked to stand with his/her back to the height rule. The back of the head, back, buttocks, calves and heels were touching the upright wall and feet were together. The top of the external auditory meatus (ear canal) was level with the inferior margin of the bony orbit (cheek bone). The participant was then asked to look straight ahead. The head piece of the stadiometer or the sliding part of the measuring rod was lowered so that the hair (if present) was pressed flat. Height was recorded to the resolution of the height rule (i.e. nearest 0.5 cm. For the participants who were taller than the research assistant, the research assistant stood on a platform so that he/she could properly read the height rule. All measurements were taken by the same person.

Waist and hip circumference were measured using an inelastic tape at the respective minimum and maximum curvatures. Participants were asked to remove their clothes, except for light underwear. Tight clothing, including the belt, was loosened and pockets emptied. Participants were then asked to stand with their feet fairly close together (about 12-15 cm) with their weight equally distributed to each leg. They were also asked to breathe normally so that the reading of

the measurement was taken at the end of a gentle exhaling. The measuring tape was then held firmly, ensuring its horizontal position around the waist. The tape was held loose enough to allow the observer to place one finger between the tape and the subject's body. Measurements were recorded to the resolution of the tape 0.5 cm. Hip circumference was measured as the maximal circumference over the buttocks and Measurements were recorded to the resolution of the tape 0.5 cm (European Health Risk Monitoring Project, 2002).

Body composition was also assessed using bioelectric impedance analyzer (Beurer GmbH, BF20). Body composition of each participant was measured while a subject was minimally dressed and not wearing shoes and without socks. The scale was adjusted to zero before any measurement was taken. The participant's variable for height, age, sex and level of activity were set before they stepped on the scale. When the analyzer was ready the participant was asked to step on the scale. The measurements for weight, body fat percentage, muscle and muscle percentage would then be automatically displayed on the screen. These measurements were recorded as they were. The measurements were taken twice for reliability and then the average taken.

3.11.3 Clinical data

Blood pressure and heart rate were measured with an automatic blood pressure monitor (Visomat® Comfort 20/40, Roche Diagnostics) during each visit. The participant's blood pressure was taken on the left arm. They were cuffed one inch above the elbow. The participant was then asked to place the cuffed arm on the table and to remain still until the measurements were taken. The systolic readings and the diastolic readings were then recorded. This was done

by the clinician who would also obtain a medical history including detailed information about medication and nutrient supplement usage.

3.11.4 Biochemical data

Fasting blood samples was collected in the morning between 7.00 and 8.00 am. Capillary blood was collected and tested for fasting blood glucose while approximately 5 ml of venous blood was collected for fasting lipid profile and transferred to heparinized collecting tubes. Blood in the tubes was centrifuged at 3000g for 3 min. Separation of serum and plasma was done using an automatic pipette and transferred into specific labeled tubes in a rack ready for analysis. A drop of capillary blood specimen was obtained from a sterilized fingertip area using a lancing device. Lipid profile assays were routinely analyzed on Mindray BSseries auto analyzer (Mindray-Bio Medical GmbH, Hamburg, and Germany) using established techniques. Fasting blood glucose was measured using soft-style Glucometer from Chemlabs.

3.11.5. Methodology for lipid profile analysis

3.11.5.1 Estimation of serum triglyceride by Glycerokinase peroxidase- peroxidase method

Triglycerides are hydrolyzed to glycerol and free fatty acids by the action of lipase. A sequence of three enzymatic steps using glycerol kinase (GK), glyceralphosphate oxidase (GPO), and horsedish peroxide (HPO) causes the oxidative coupling of 3.5-dichloro-3-hydroxybenzenesulfonic acid (DHBS) with 4-aminoantipyrine to form a red quinoneimine dye. The change in the absorbance is directly proportional to the concentration of triglycerides in the sample:

$$\text{Triglycerides} + 3H_2O \xrightarrow{LPL} \text{Glycerol} + 3\text{RCOOH}$$

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

$$\text{Glycerol-3- phosphate} + O_2 \xrightarrow{GO} \text{Dihydoxyacetone phosphate} + H_2O_2$$

$$2H_2O_2 + \text{4-aminoantipyrine} + \text{4-chlorophenol} \xrightarrow{POD} \text{Quinoneimine dye} + HCl + \text{4-chlorophenol} + 4\ H_2O$$

In a clean, dried and labeled test tube**,** 10µLs of sample was taken. Then 1000µLs of reagent mixture (phosphate buffer [50 mmol/L], 4-chlorophenol [5mmol/L], ATP [2 mmol/L], Mg^{2+} [4.5 mmol/L], Glycerokinase [≥ 0.4 U/mL], peroxidase [≥ 0.5 U/mL], lipoprotein lipase [≥ 1.3 U/mL], 4-aminoantipyrine [0.25 mmol/L] and Glycerol-3- phosphate oxidase [≥ 1.5 U/mL)] was added to each tube and contents were mixed immediately on a vortex. The tubes were then incubated at 37^0C for 10 minutes. A coloured complex was developed and the optical density was recorded against the blank at 510nm <0.3 A on a spectrophotometer. The analyzer calculates the triglyceride concentration after calibration. Conversion factor: mg/dl x0.0113 = mmol/L.

3.11.5.2 Estimation of serum total cholesterol by oxidase method

Total cholesterol is determined after enzymatic hydrolysis (where cholesterol enters and is cleaved by the action of cholesterol esterase to yield free cholesterol and fatty acids) and oxidation. The indicator, quinoneimine, is formed from hydrogen peroxide and 4-amino antipyrine in the presence of phenol and peroxidase. The intensity of color produced is directly proportional to the amount of cholesterol present in serum.

$$\text{Cholesterol-ester} + H_2O \xrightarrow{CholesterolOxidase} \text{Cholesterol} + \text{fatty acids}$$

$$\text{Cholesterol} + O_2 \xrightarrow{Cholesteroloxidase} \text{Cholestene-3- one} + H_2O_2$$

$$2H_2O_2 + \text{Phenol} + \text{4-aminoantipyrine} \xrightarrow{Peroxidase} \text{Dye} + H_2O$$

In a clean, dried and labeled test tube**,** 10µL of sample was taken. Then 1000µL of reagent mixture (phosphate buffer [100 mmol/L], phenol [5mmol/L], cholesterol esterase [≥ 150 KU/L],

Peroxidase [5 KU/L], 4-aminoantipyrine [0.3 mmol/L] and Cholesterol oxidase [≥ 100 KU/L)] was added to each tube and contents were mixed immediately on a vortex. The tubes were then incubated at 37^0C for 10 minutes. A coloured complex was developed and the optical density was recorded against the blank at 510nm < 0.3 A on a spectrophotometer. The analyzer calculates the cholesterol concentration after calibration. Conversion factor: mg/dl x0.026= mmol.

3.11.5.3 Estimation of serum HDL-C by direct method

The chylomicrons, VLDL and LDL were precipitated by addition of phosphotungistic acid and magnesium chloride. When centrifuged the supernatant fluid contains the HDL fraction, their cholesterol content was determined enzymatically:

- LDL, VLDL, Chylomicrons $\xleftrightarrow{Cholesterolesterase}$ Cholestenone + H_2O_2

  $2H_2O_2 \xleftrightarrow{Peroxidase} H_2O + O_2$

- HDL $\xleftrightarrow{CHE+CHO}$ Cholestenone + H_2O_2

  H_2O_2 + HDAOS+ 4-Aminoantipyrine $\xleftrightarrow{POD}$ Quinoneimine

In a clean, dried and labeled test tube**,** 12µL of sample was taken. Then 900µL of Reagent 1 mixture (Goods buffer [100 mmol/L], cholesterol esterase [600U/L], Cholesterol oxidase [≥ 380 KU/L], Catalase [600KU/L], HDAOS [0.42 mmol/L)] was added to each tube and contents were mixed immediately on a vortex. The tubes were then incubated at 37^0C for 5 minutes. Then 300 µL of Reagent 2 mixture was added (Goods buffer [100 mmol/L], Peroxidase [> 2.8U/Ml], 4-Aminoantipyrine [1.0 mmol/L] and Surfactant [<2%]). After thorough mixing, the mixture was incubated at 37^0C for 5 minutes. The coloured complex was developed and the optical density

was recorded against the blank at 600nm < 0.08 A on a spectrophotometer. The analyzer calculated the HDL-C concentration after calibration.

3.12 Laboratory Analysis of Peanut Samples

3.12.1 Acquisition and preparation of Peanut Samples

Raw, dried, jumbo size peanuts of red Valencia varieties were purchased from a local grocery store. Raw peanuts were blanched and dry roasted. The roast color of a retail brand was used as the standard for roasted peanuts. The peanuts were diced and a portion of the fresh samples was taken from each variety and used for moisture content determination, while the rest of the samples were dried, pulverized and used for the determination of crude protein, fat content, fibre, ash content and carbohydrate.

3.12.2 Dry-Roasting

Five hundred grams of peanut kernels was spread evenly on a perforated aluminum tray and blanched in a convection oven at 138°C for 20 minutes. The blanched kernels were then dry-roasted in a preheated convection oven at 180 ± 5°C for 22 minutes with periodic shaking for even roasting. The peanuts were then cooled, and their skins were manually removed.

3.13 Proximate composition

Proximate composition includes moisture, crude protein, ether extract for fat content, crude fiber, ash and nitrogen free extract (NFE). The dried peanuts were weighed into various proportions for proximate analysis.

3.13.1 Determination of the moisture content

Moisture was determined by oven drying method (Horwitz, 2000). Peanut powder was well-mixed and 2 g was accurately weighed in clean, dried crucible. The crucible was put in an oven at 100-105°C for 6-12 hours until a constant weight was obtained. Then the crucible was placed in the desiccator for 30 minutes to cool. After cooling it was weighed again, the percent moisture content was calculated by the following formula:

$$\%\, moisture = \left(1 - \frac{Weight_{\,Dry\ Sample}}{Weight_{\,Wet\ Sample}}\right) \times 100$$

3.13.2 Determination of ash content

For the determination of ash, clean empty crucible was placed in a muffle furnace at 600°C for an hour, cooled in desiccator and then weight of empty crucible was noted (W1). One gram of the peanut powder was taken in crucible (W2). This was then ignited over a burner with the help of blowpipe, until it is charred. Then the crucible was placed in muffle furnace at 550°C for 2-4 hours. The appearances of gray white ash indicated complete oxidation of all organic matter in the peanut material. After ashing furnace was switched off. The crucible was cooled and weighed (W3). Percent ash was calculated by following formula:

$$\%\, Ash = \frac{Difference\ in\ weight\ of\ Ash}{Weight\ of\ the\ peanut\ powder} \times 100$$

Difference in weight of ash = W3 - W1

3.13.3 Determination of crude protein content

Protein in the sample was determined by Kjeldahl method. The samples were digested by heating with concentrated sulphuric acid (H_2SO_4) in the presence of digestion mixture. The mixture was then made alkaline. Ammonium sulphate formed, released ammonia which was collected in 2% boric acid solution and titrated against standard HCl. Total protein was calculated by multiplying

the amount of nitrogen with appropriate factor (6.25) and the amount of protein was then calculated. To 1.0g of dried samples in digestion flask, 15ml of concentrated H_2SO_4 was added and 8g of digestion mixture composed of potassium sulphate and copper sulphate in the ration of 8:1.

The flask was swirled in order to mix the contents thoroughly and then placed on the heater to start digestion till the mixture became clear (blue green in color) for 2 hours. The digest was cooled and transferred to 100 ml volumetric flask and volume was made up to mark by the addition of distilled water. Distillation of the digest was performed in Markam Still Distillation Apparatus (Khalil and Manan, 1990). Briefly, 10ml of digest was introduced in the distillation tube then 10ml of 0.5 N NaOH was gradually added through the same way. Distillation was continued for at least 10 min and NH_3 produced was collected as NH_4OH in a conical flask containing 20ml of 4% boric acid solution with few drops of modified methyl red indicator. During distillation yellowish color appears due to NH_4OH. The distillate was then titrated against standard 0.1 N HCl solution till the appearance of pink color. A blank was also run through all steps as above. Percent crude protein content of the sample was calculated by using the following formula:

$\% \, Crude \, Protein = 6.2^{*} \times \% \, N$.

(Whereby * stands for the Correction factor).

$$\% \, \mathrm{N} = \frac{(\mathrm{S}-\mathrm{B}) \times \mathrm{N} \times 0.014 \times \mathrm{D} \times 100}{\text{Weight of the sample} \times \mathrm{V}}$$

Where: S = sample titration reading

B = Blank titration reading

N = Normality of HCl

D = Dilution of the peanut material after digestion

V = Volume taken for distillation

0.014 = Milli equivalent weight of Nitrogen

3.13.4 Determination of crude fat content

Dry extraction method for fat determination was applied. It consisted of extracting dry peanuts with some organic solvent, since all the fat materials like fats, phospholipids, sterols, fatty acids, carotenoids, pigments, chlorophyll, are extracted together, the results are therefore frequently referred to as crude fat. Fats were determined by intermittent Soxhlet extraction apparatus. Crude fat was determined by ether extract method using Soxhlet apparatus.

Approximately 1 g of moisture free sample was wrapped in filter paper, placed in fat free thimble and then introduced in the extraction tube. Weighed, cleaned and dried, the receiving beaker was filled with petroleum ether and fitted into the apparatus. Water and heater were turned on to start extraction. After 4-6 siphoning, ether was allowed to evaporate and beaker was disconnected before last siphoning. Extract was transferred into clean glass dish with ether washing and ether was evaporated on water bath. The dish was then placed in an oven at 105°C for 2 hrs and cooled it in a desiccator. The percent crude fat was determined by using the following formula:

$$\%\ Crude\ fat = \frac{Weight\ of\ ether\ extract}{Weight\ of\ peanut\ material} \times 100$$

3.13.5 Determination of crude fiber content

A moisture free and ether extracted sample of crude fiber made of cellulose was first digested with dilute H_2SO_4 and then with dilute KOH solution. The undigested residue collected after digestion was ignited and loss in weight after ignition was registered as crude fiber. The peanut material was weighed (W0 of 0.153 g) and transferred to a porous crucible. The crucible was then placed into Dosi-fiber unit and the valve kept in "OFF" position. After that 150 ml of preheated H_2SO_4 solution was added and some drops of foam-suppresser to each column.

The cooling circuit was then openned and turned on the heating elements (power at 90%). When it started boiling, the power was reduced to 30% and left for 30 min. Valves were opened for drainage of acid and rinsed with distilled water thrice to completely ensure the removal of acid from sample. The same procedure was used for alkali digestion by using KOH instead of H_2SO_4. Then the sample was dried in an oven at 150°C for 1 hour and then allowed to cool in a desiccator and weighed (W1). The samples were kept in crucibles in muffle furnace at 55°C for 3-4 hours. The samples were cooled in a desiccator and weighed again (W2). The percent crude fiber was calculated as follows:

$$\% \, Crude \; Fiber = \frac{W1 - W2 \times 100}{Wo}$$

3.13.6 Determination of nitrogen free extract

Nitrogen free extract (NFE) was calculated by difference after analysis of all the other items in the proximate analysis. The following equation was applied:

NFE = (100 - % moisture + % crude protein + % crude fat + % crude fiber + % ash).

3.14 Data Analysis and Presentation

The data was entered in MS Excel spread sheet and then exported to the SPSS version 20 software for analysis. Descriptive statistics (frequencies, means, standard deviations, and percentages) were analyzed on the final cleaned data set for all study variables. For continuous variables, mean, median, skewness, standard error of the mean, standard deviation were calculated. Frequencies were run on all categorical variables. All continuous variables were checked for normal distribution by calculating Fisher's measure of skewness. A log transformation was performed on the Framingham risk score variable to achieve a normal distribution.

Student T- tests was used to assess whether there was significant difference on the lipid profile and BMI in both treatment arms from baseline. All the tests were considered significant at $p< 0.05$. Relationships between all and the individual CHD risk factors were analyzed using Spearman's correlation coefficient, single line linkage and Ward's cluster methods. Data analysis for 24-hour dietary recall and 7- day food frequency focused on total energy intake, total fat intake, SFA, MUFA, PUFA, protein and carbohydrate.

Nutrient composition was analyzed using Nutri-survey (2014) program. A BMI calculator was used to analyze the nutritional status. Pearson's correlation (r) was used for non-categorical variable (nutrient and energy intake, CD4 count and weight gain). Chi square was used for categorical variables such as social demographic data. A paired student T-test was applied to compare subject differences in various outcomes at baseline and at the end of each treatment period.

Treatment effects on lipid profile, energy and nutrient intakes were assessed by repeated measures of analysis of variance. Multiple regression analysis was used to determine the effect of peanut supplementation and nutrition counseling on lipid profile. Executive Summary of the Third Report of the National Cholesterol Education Program (ATP III) (2001) guidelines were used to classify risk for lipid profile: Total cholesterol of <5.1mmmol/L – desirable, 5.1-6.1mmol/L- borderline high and above 6.1mmol/L –high; Triglyceride < 1.69mmol/L- normal, 1.69-2.24mmol/L- borderline high and 2.25- 5.6mmol/L- high and above 5.6mmol/L – very high; HDL-C less than 1.03-low while above 1.55mmol/L- high; LDL-C below 2.5mmol/L-optimal, 2.5-3.3mmol/L near optimal, 3.4-4.1mmol/L- borderline high, 4.2-4.9mmol/L high and above 4.91mmol/L- very high.

Diabetes risk classification was adopted from American Diabetes Association (2007): Fasting blood glucose below 100mg//dl- normal, 100-125mg/dl- pre-diabetes and above 126mg/dl-diabetes. Hypertension risk classification was adopted from National high blood pressure education program coordinating committee 7th report (2003): Systolic blood pressure <120mmHg or diastolic blood pressure < 80mmHg-normal; Systolic blood pressure 120-139mmHg or diastolic blood pressure 80-89mmHg-pre hypertension; Systolic blood pressure 140-159mmHg or diastolic blood pressure 90-99mmHg- Stage I hypertension; and Systolic blood pressure ≥160mmHg or diastolic blood pressure ≥100mmHg- Stage II hypertension.

Classification of overweight and obesity and waist hip ratio was adopted from NIH (1998) and WHO (2000): BMI< 18.5kg/m^2 –underweight; 18.5-24.9kg/m^2 – Normal; 25.0- 29.9kg/m^2 – overweight; 30.0-34.9kg/m^2 – obesity class I; 35.0-39.9kg/m^2 – obesity class II; ≥40kg/m^2-

extreme obesity class III. Waist hip ratio above 1 for males and above 0.85 for females was considered high risk.

3.15 Logistical and ethical considerations

Research authorization was obtained from Kenyatta University Graduate School (appendix K). Ethical clearance was sought from Kenyatta University Ethical Review Committee (appendix L) while the research permit was obtained from the National Council of Science and Technology (appendix J). An informed consent (appendix G) was signed by every consenting participant and confidentiality was assured to every participants. All participants were tested for peanut allergy and none of the participant had allergy to the peanut. Participation in the study was voluntary.

CHAPTER FOUR: RESULTS

4.0 Baseline

Preliminary description of the study sample was done at baseline for social economic and demographic characteristics, nutritional status, dietary practices, morbidity patterns and cardiovascular risk**.** The results only included the participants who completed the two treatments. Group 1 had 48 participants while group 2 had 47 participants. Three participants dropped out of the study before the first treatment was over. In group two seven participants dropped out of the study. Three dropped before the treatment II was over because they could not make it to the clinic monthly, three participant dropped after the first treatment while one died in a motor bike accident.

4.1 Socio-economic and demographic characteristics

Table 4.1 shows the description of baseline social economic and demographic characteristics by group. Forty eight percent of participants were aged between 40 to 49 years while over 29% were aged above fifty years. Twenty one percent of the participants were males while 79% were females. Twenty five percent of the participants were single parents while twenty percent were divorced or separated Majority of the participants had completed secondary education while below 5% had no formal education. Majority of the participants were merchants and traders while only 17% were formally employed with a salary. Age, marital status, education level were not significantly different ($p>0.05$) between group 1 that started with treatment I, and group 2 that started the intervention with treatment II. This indicated that the social economic status did not have an effect on the impact of the two treatments offered.

Table 4.1 Description of baseline socio-economic and demographic characteristics by group

	Category	Period of treatment N=85		P value χ
		GROUP1(AB-BA) n=45	GROUP2(BA-AB) n=40	
Age	18-29	0(0.0)	1(1.2)	0.592
	30-39	8(9.4)	10(11.8)	
	40-49	23(27.1)	18(21.2)	
	>50	14(16.5)	11(12.9)	
Sex	Male	10(11.8)	8(9.4)	0.802
	Female	35(41.2)	32(37.6)	
Marital status	Single parent	12(14.1)	10(11.8)	0.405
	Married/Living together	19(22.4)	14(16.5)	
	Divorced/Separated	7(8.2)	10(11.8)	
	Widowed	7(8.2)	4(4.7)	
	Single	0(0.0)	2(2.4)	
Levelof education	Lower primary	3(3.5)	2(2.4)	0.892
	Upper primary	13(15.3)	15(17.6)	
	Secondary	22(25.9)	19(22.4)	
	College	3(3.5)	2(2.4)	
	University	1(1.2)	0(0.0)	
	No formal education	2(2.4)	2(2.4)	
Occupation	Agricultural labor	12(11.1)	15(17.6	0.515
	Employed(Salaried)	7(8.2)	7(8.2)	
	Merchant/ Trader	18(21.2)	11(12.9)	
	Housewife	3(3.5)	2(2.5)	
	Waged labor	5(5.9)	5(5.9)	

Numbers in parenthesis indicate the percentage frequency

4.2 Baseline cardiovascular risk factors

Table 4.2 shows a description of baseline cardiovascular risk factors. The cut offs for lipid profile were based on NCEP (2001) guidelines while obesity was classified using WHO (2000) guidelines. Waist hip ratio and waist circumference were based on WHO (2008) guidelines. Twenty nine percent of the respondents were aged fifty years and above while 48.2% were between 40-49 years. Only 5.9% of the respondents smoked while 8.2% drunk alcohol. Twenty

seven percent (27.1%) had low physical activity while only 11.8% had high physical activity. Twenty four percent (24.7%) had obesity class I (30-34.9), 8.2% had obesity class II (35-39.9) while 1.5% had obesity class III (> 40). Another 31.8% were overweight (25-29.9).

Slightly over 8% of the males had WHR of 0.96-1 (moderate risk) while 3.5% had high risk (WHR >1). Nineteen females (22.4%) had moderate risk (WHR 0.81-0.85) while 42.9% had high risk (WHR >0.85). Systolic blood pressure was used to determine the hypertension status. Over 40% of the respondents were found to be pre hypertensive (120-139) while 28.2% had hypertension stage I (140-159) and another 11.8% had hypertension stage II (>160). Nineteen (22.4%) had high total cholesterol (>6.2), 30.6% had borderline high (5.1-6.1) while 47.1% had desirable levels. Over 21% of the respondents had borderline high triglyceride (1.69-2.24) while 34.1% had high serum triglycerides (2.25-5.6). Only 5.9% had low serum levels of HDL-C (<1.03) while 34.1% had high serum HDL-C (>1.55). Twenty nine percent of the respondents had borderline high serum levels of LDL-C (3.4-4.1) while 7.1% had high (4.2-4.9) and another 4.7% had very high serum LDL-C (>4.91). The two groups were not statistically significantly different at baseline for all the cardiovascular risk factors measures.

Table 4.2: Description of baseline Cardio vascular risk factors

Risk factor	**Category**	**Period of treatment**		**Total**	**P value**
		Group 1 n=45	**Group 2 n=40**		
Age	18-29	0(0.0)	1(1.2)	1(1.2)	0.592
	30-39	8(9.4)	10(11.8)	18(21.2)	
	40-49	23(27.1)	18(21.2)	41(48.3)	
	>50	14(16.5)	11(12.9)	25(39.4)	
Smoking	Yes	2(2.4)	3(3.5)	5(5.9)	0.754
	Never	41(48.2)	26(90.6)	77(90.6)	
	Not in the last 1 year	2(2.4)	1(1.2)	3(3.5)	

Alcohol consumption	Yes	3(3.5)	4(4.7)	7(8.2)	0.577
	No	42(49.4)	36(42.4)	78(91.8)	
Physical activity	Low	12(14.1)	11(12.9)	23(27.1)	0.994
	Moderate	27(31.8)	25(29.4)	52(61.2)	
	High	6(4.1)	4(4.7)	10(11.8)	
BMI(kg/m^2)	Less than 18.5	2(2.4)	0(0.0)	2(2.4)	0.657
	18.5-24.9	13(15.3)	14(16.5)	27(31.8)	
	25-29.9	15(17.6)	12(14.1)	27(31.8)	
	30-34.9	11(12.9)	10(11.8)	21(24.7)	
	35-39.9	4(4.7)	3(3.5)	7(8.2)	
	>40	0(0.0)	1(1.2)	1(1.2)	
WHR	0.95 and below	3(3.5)	5(5.9)	8(9.4)	0.637
	0.96 to 1	5(5.9)	2(2.4)	7(8.2)	
	above 1	2(2.4)	1(1.2)	3(3.5)	
	0.80 and below	2(2.4)	4(4.7)	6(7.1)	
	0.81 to 0.85	9(10.6)	10(11.8)	19(22.4)	
	above 0.85	24(28.2)	18(21.2)	42(49.4)	
SBP (mmHg)	less than 120	7(8.2)	8(9.4)	15(17.6)	0.215
	120 to 139	23(27.1)	13(15.3)	36(42.4)	
	140 to 159	9(10.6)	15(17.6)	24(28.2)	
	160 and above	6(7.1)	4(4.7)	10(11.8)	
TC(mmol/L)	less than 5.1	20(23.5)	20(23.5)	40(47.1)	0.829
	5.1 to 6.1	15(17.6)	11(12.9)	26(30.6)	
	6.2 and above	10(11.8)	9(10.6)	19(22.4)	
TG(mmol/L)	less than 1.69	19(22.4)	21(24.7)	40(47.1)	0.620
	1.69 to 2.24	10(11.8)	8(9.4)	18(21.2)	
	2.25 to 5.6	16(18.8)	18(12.9)	27(31.8)	
HDL-C(mmol/L)	less than	7(8.2)	5(5.9)	12(14.1)	0.350

	1.03				
	1.03 to 1.54	20(23.5)	24(28.2)	44(51.8)	
	1.55 and above	18(21.2)	11(12.9)	29(34.1)	
LDL-C(mmol/L)	<2.5	11(12.9)	5(5.9)	16(18.8)	0.235
	2.5 to 3.3	13(15.3)	18(21.2)	31(36.5)	
	3.4 to 4.1	16(18.8)	9(10.6)	25(29.4)	
	4.2 to 4.9	2(2.4)	4(4.7)	6(7.1)	
	>4.91	3(3.5)	4(4.7)	7(8.2)	
FBG (mg/dl)	less than 100	38(44.7)	33(38.8)	71(83.5)	0.831
	100 to 126	5(5.9)	4(4.7)	9(10.6)	
	126 and above	2(2.4)	3(3.5)	5(5.9)	

4.3. Proximate composition of Red Valencia varieties

Table 4.3 shows the proximate analysis of raw and roasted peanuts Valencia variety. The moisture content of raw and roasted peanut ranged from 4.84-5.11% and 2.02 - 2.17% respectively while fat content of raw and roasted peanut ranged from 43.3-48.3% and 47.3-49.1%, respectively. Protein content in raw peanut ranged from 22.02-28.99% while roasted peanut ranged from 31.4-33.1%. Total ash in raw peanut ranged from 2.37-2.54% while roasted peanut ranged from 4.04-4.13%. Fiber content in raw peanut ranged from 9.8-10.83% while in roasted, it ranged from 5.47-6.56%. Carbohydrate content in raw peanut ranged from 9.49-12.37% while it ranged from 6.63-7.87% in roasted peanut.

Table 4.3 Proximate composition of raw and roasted peanut varieties

	Red Valencia Raw	Red Valencia Roasted	P value (t-test)
Moisture (%)	5.11±0.05	2.17±0.04	0.001
Fat (%)	48.33±0.14	49.13±0.11	0.001
Protein (%)	22.02±0.23	31.45±0.65	0.003
Total ash (%)	2.37±0.04	4.04±0.03	0.001
Fiber (%)	9.80±0.19	6.56±0.07	0.001
Carbohydrate (%)	12.37±0.44	6.63±0.49	0.008

Values are expressed as mean and Standard Deviation, n=3.

4.4. Dietary intake

Dietary intake and practices was assessed using 7 day food frequency questionnaire and 24 hour dietary recall. Table 4.4 describes food consumption patterns from a 7 day food frequency questionnaire. Mean dietary intake for maize and maize products was highest at 8.56±3.51 per week while other cereals were consumed less than two times weekly. Fruits, legumes and vegetables were consumed on average five times per week. Meat and meat products were minimal with frequency of less than once per week being reported on all the products. Majority of those who consumed the vegetables had them fried in oil while those who consumed legumes had them boiled.

Table 4.4 Baseline consumption patterns

Food item	Mean frequency of intake per week	SD
Maize	8.56	3.51
Rice	1.66	1.22
Millet	1.59	2.63
White bread	1.07	1.98
Brown bread	1.09	2.10
Chapati	0.81	1.22
Sorghum	1.55	2.64
Beef roasted or fried with fat	0.69	0.97
Kidney	0.02	0.15
Goat roasted or fried without fat	0.02	0.15
Liver	0.07	0.30
Heart	0.01	0.10
Tripe	0.02	0.15
Egg fried in oil	0.76	1.60
Boiled egg	0.44	0.86
Egg fried in fat	0.36	1.20
Fish deep fried	0.12	0.44
Fish stewed	0.15	0.82
Vegetable boiled	0.27	1.30
Vegetable fried with oil	3.06	3.06
Vegetable fried with fat	2.00	2.85
Fruits	4.42	2.58
Nuts roasted	0.32	0.56
Legumes and pulses fried	1.99	2.43
Legumes and pulses boiled	4.61	1.55
Tubers roots plantain boiled	1.84	2.41

Table 4.5 shows a baseline comparison of the mean nutrient intake from the three random 24 hour dietary recall between the two groups. The mean energy intake for group 1 at baseline was 1850±372.20 while for group 2 were 2060±134.10 which was lower than the recommended dietary intake of 2240Kcals for un- infected adults. The mean intake of monounsaturated fatty acids, polyunsaturated fatty acid and saturated fatty acid for group 1 was 15.29±8.98, 8.72±2.56 and12.49±8.87 respectively while for group 2 were 17.81±6.56, 9.14±4.98 and 17.64±9.80

respectively. The mean percentage intake of fat as a function of energy was 20.50±6.00 for group 1 and 23.71±6.49 for group 2. This was within the recommended intake for fat which is 20- 30% of total energy intake. Mean fiber intake for group 1 and group 2 was 26.71±8.55 and 21.6±8.78 respectively while Vitamin E intake for group 1 and group 2 was 2.08±1.36 and 4.92±3.33 respectively. The intake of Vitamin E was less than RDA which is 5g. Dietary intake of the two groups was not significantly different at baseline.

Table 4.5 Baseline comparison of the estimated nutrient and energy intake between groups

	GROUP 1 (N=10)	GROUP 2 (N=7)	P VALUE (t-test)
Energy (kcal/day)	1850±372.20	2060±134.40	0.176
Fat (%energy)	20.50±6.00	23.71±6.49	0.310
SFA	12.49±8.87	17.64±9.80	0.278
MUFA	15.29±8.98	17.81±6.56	0.537
PUFA	8.72±2.56	9.14±4.98	0.821
Cholesterol (mg)	77.85±114.36	177.57±198.60	0.208
Protein(% energy)	12.50±2.59	11.85±2.67	0.626
Carbohydrate (% energy)	67.10±7.56	64.42±8.38	0.503
Vitamin E	2.08±1.36	4.92±3.33	0.270
Folate (mg/day)	342.21±164.85	273.40±224.39	0.476
Magnesium (mg/day)	469.41±73.53	518.65±135.86	0.348
Carotene	257.37±558.51	740.53±1618.55	0.392
Dietaryfibre (g/day)	26.71±8.55	21.6±8.78	0.250

Values presented as the mean± standard deviation; n=17. Means with different superscript letters are statistically significant at (P< 0.05). PUFA- poly unsaturated fatty acid, MUFA- mono unsaturated fatty acids, SFA – saturated fatty acid.

Table 4.6 shows the mean change in the dietary intake when peanut was added to regular diet (T I) and when counseling in healthy diet plus 80g of peanut were given (T II). There was a statistically significant difference in fat intake ($F(2, 48) = 13.185$, $p < 0.05$) between baseline and the two treatments. The same was found for carbohydrate intake ($F(2, 48) = 11.664$, $p < 0.05$), polyunsaturated fatty acid intake ($F(2, 48) = 55.091$, $p < 0.05$), Vitamin E intake (F (2,

48) = 37.614, p < 0.05) and mono unsaturated fatty acids intake (F (2, 48) = 34.328, p < 0.05). Compared with baseline, energy intake from fat increased significantly during TI and TII (both p< 0.001), MUFA and PUFA increased significantly during TI and TII (all p< 0.001), while SFA remained unchanged. Non-fatty acid lipid materials such as sterols were not measured. There was a significant decrease of carbohydrate intake during TI and TII (both p < 0.001 and p < 0.001), respectively. Dietary intakes of vitamin E (p < 0.001) increased significantly from baseline in Treatment I as well as in treatment II (vitamin E p < 0.001).

All these changes can be attributed to inclusion of peanuts in the diet. However folate and magnesium did not change significantly in both treatments from baseline. There was no significant difference between the dietary intake in treatment I and treatment I.

Table 4.6 Estimated mean daily energy and nutrient intakes from three random-day 24-h recall

	Baseline	T1	T2
Energy (kcal/day)	1937.10±309.98 [a]	2056.02±224.12[a]	2091.99±307.47[a]
Fat (%energy)	21.82±6.22[a]	32.05±7.64[b]	32.76±6.91 [b]
SFA	14.61±9.33[a]	19.39±4.51 [a]	19.39±5.72 [a]
MUFA	16.33±7.95[a]	32.53±6.17 [b]	33.19±5.84 [b]
PUFA	8.89±3.61[a]	17.86±2.66 [b]	19.16±2.96 [b]
Cholesterol (mg)	118.91±157.18[a]	118.12±211.06[a]	103.34±206.23 [a]
Protein(% energy)	12.23±2.56[a]	13.88±3.19[a]	13.35±2.47 [a]
Carbohydrate (% energy)	66.00±7.77[a]	54.23±9.71 [b]	53.64±7.58 [b]
Vitamin E	3.25±2.70[a]	8.87±2.13 [b]	8.80±1.48 [b]
Folate (mg/day)	313.89±188.11[a]	387.40±229.22 [a]	395.12±230.52 [a]
Magnesium (mg/day)	489.68±102.89[a]	592.53±142.34 [a]	618.22±248.03 [a]
Carotene	456.32±1103.60[a]	2149.58±4765.67 [a]	1892.89±4173.92 [a]
Dietaryfibre (g/day)	24.61±8.76[a]	29.57±10.78 [a]	31.87±9.11 [a]

Values presented as the mean± standard deviation; n=17. Means with different superscript letters are statistically significant at (P< 0.05). PUFA- poly unsaturated fatty acid, MUFA- mono unsaturated fatty acids, SFA – saturated fatty acid.

4.5 Nutritional status markers

Table 4.7 compares the nutrition status markers for group 1 and group 2 at baseline. The mean baseline body mass index, weight, body fat, body muscle, waist circumference and hip circumference of the respondents in group 1 was 27.42±4.81, 72.67±13.09, 30.74±7.97, 32.47±4.10, 91.78±10.86, 91.77±11.53 and 101.37±8.63 respectively while for group 2 was 27.36±5.68, 71.50±14.02, 29.69±8.99, 32.03±4.40, 91.80±10.20 and 103.37±12.16 respectively. The mean fasting blood glucose for group 1 and group 2 was 81.80±24.96 and 81.55±33.26 respectively. The baseline mean for total cholesterol, triglycerides, High density lipoproteins and low density lipoprotein cholesterol for group 1 was 5.20±1.16, 1.91±0.79, 1.44±45 and 3.36±0.95 respectively while for group 2 was 5.13±1.22, 1.84±0.91, 1.36±0.38 and 3.36±0.95 respectively. The two groups were not significantly different at baseline for all the nutrition status markers.

Table 4.7 Baseline comparison for nutrition markers according to sequence of treatment

	Group 1n=45	**Group 2 n=40**	**P value (independent t)**
Weight	72.67±13.09	71.50±14.02	0.693
BMI	27.42±4.81	27.36±5.68	0.692
BF	30.74±7.97	29.69±8.99	0.675
LBM	32.47±4.10	32.03±4.40	0.633
WC	91.77±11.53	91.80±10.20	0.993
HC	101.37±8.63	103.37±12.16	0.381
BP	134.82±18.30	135.45±18.61	0.876
FBG	81.80±24.96	81.55±33.26	0.969
TC	5.20±1.16	5.13±1.22	0.762
TG	1.91±0.79	1.84±0.91	0.707
HDL-C	1.44±45	1.36±0.38	0.430
LDL-C	3.36±0.95	3.36±0.95	0.662

Values presented as the mean± standard deviation. BMI- body mass index, BF- body fat, LBM- lean body muscle, W.C- waist circumference, H.C- hip circumference, SBP- systolic blood pressure, DBP- diastolic blood pressure, FBG- fasting blood glucose TC- total cholesterol, TG- triglycerides, HDL-C- high density lipoprotein cholesterol, LDL-C- Low density lipoprotein cholesterol

Table 4.8 describes the monthly trends for the parameters taken monthly. Analysis of variance (ANOVA) was used to find the difference in nutritional status in Treatment I and Treatment II respectively. There was no significance difference in nutritional status markers within the month and between the months. Weight, body fat, waist circumference and fasting blood glucose increased slightly in treatment I but decreased in treatment II. There was no statistically significant change in weight, BMI, waist circumference, hip circumference, body fat, body muscle, systolic and diastolic blood pressure and fasting blood glucose after consumption of peanut with regular diet and consumption of peanut combined with nutritional counseling on healthy diet. There was no statistically significant difference in nutritional status between the two treatments

Table 4.8 Change in nutritional status markers between months and between treatments

	Treatment I			Treatment II		
	Baseline	**Week 4**	**Week 8**	**Baseline**	**Week 4**	**Week 8**
Weight (kg)	72.12±13.47[a]	72.28±13.73 [a]	72.32±13.73 [a]	72.27±13.46 [a]	72.24±13.39 [a]	72.15±13.60 [a]
BMI (kg/m^2)	27.39±5.20 [a]	27.37±5.26 [a]	27.45±5.31 [a]	27.4585±5.22 [a]	27.40±521 [a]	27.39±5.30 [a]
BF (%)	30.37±8.42 [a]	33.01±17.73 [a]	33.96±20.85 [a]	32.62±13.15 [a]	30.96±8.40 [a]	31.12±9.12 [a]
LBM (%)	32.26±4.22 [a]	32.25±4.69 [a]	31.92±4.28 [a]	35.35±29.27 [a]	32.07±3.93 [a]	31.96±4.23 [a]
WC(cm)	91.78±10.86 [a]	93.42±10.27 [a]	92.45±10.58 [a]	92.37±11.17 [a]	90.64±12.53 [a]	91.80±10.69 [a]
HC(cm)	102.31±10.42 [a]	103.20±10.63 [a]	101.85±9.76 [a]	101.79±11.24 [a]	100.09±12.86 [a]	100.50±10.62 [a]
SBP(mmHg)	135.11±18.36 [a]	138.04±19.16 [a]	134.57±16.40 [a]	134.41±17.64 [a]	134.00±18.69 [a]	134.09±20.25 [a]
DBP(mmHg)	81.71±9.83 [a]	81.90±12.07 [a]	81.5882±10.22 [a]	81.48±12.46 [a]	81.65±10.25 [a]	80.82±10.68 [a]
FBG(mg/dl)	81.68±28.99 [a]	85.07±25.88 [a]	86.36±32.84 [a]	88.32±24.36 [a]	91.30±26.82 [a]	88.69±35.84 [a]

Values presented as the mean± standard deviation; n=85. Means with same superscript letters are not statistically significant at ($P< 0.05$). BMI- body mass index, BF- body fat, LBM- lean body muscle, W.C- waist circumference, H.C- hip circumference, SBP- systolic blood pressure, DBP- diastolic blood pressure, FBG- fasting blood glucose

4.5.1 Effect of peanut supplementation on weight, BMI, waist circumference, hip circumference and fasting blood glucose

Table 4.9 shows regression analysis for weight, BMI, waist circumference, hip circumference and fasting blood glucose. The test did not establish relationship between the changes in weight, BMI, waist and fasting blood glucose in treatment I and treatment II and the changes in energy, fat and carbohydrate intake as predicted.

Table 4.9 Effect of peanut supplementation on weight, BMI, waist circumference, hip circumference and fasting blood glucose

		Treatment I			Treatment II		
		β	**t**	**P. value**	**β**	**t**	**P. value**
Change in Weight	Energy	0.282	0.977	0.346	0.451	1.637	0.126
	Fat	1.290	1.217	0.245	-0.578	-0.733	0.476
	Carbohydrate	1.496	1.443	0.173	-0.740	-0.969	0.350
Change in BMI	Energy	0.290	0.997	0.337	0.424	1.523	0.152
	Fat	1.256	1.176	0.261	-0.607	-0.761	0.460
	Carbohydrate	1.442	1.381	0.191	-0.736	-0.953	0.358
Change in WC	Energy	0.440	1.536	0.148	0.398	1.443	0.173
	Fat	1.190	1.132	0.278	0.471	0.597	0.561
	Carbohydrate	0.897	0.872	0.399	-0.058	-0.075	0.941
Change in FBG	Energy	0.262	0.847	0.412	0.066	0.211	0.836
	Fat	0.262	0.230	0.822	-0.416	-0.465	0649
	Carbohydrate	0.149	0.134	0.896	-0.390	-0.450	0.660

BMI- body mass index, WC waist circumference, FBG- fasting blood glucose

4.6. Lipid profile

Table 4.10 shows the change in total cholesterol, triglycerides, high density lipoprotein cholesterol and low density lipoprotein cholesterol. There was a 3.07% decrease in total cholesterol in treatment I while the decrease in treatment II was 5.39% (Figure 4.2). The

decrease was significant in both treatment I and II ($p<0.001$). The mean change between the two treatments was also significant ($p < 0.001$).

There was also a decrease in triglycerides in treatment 1 of 12.81% while in treatment 2 it was 17.01% (Figure 4.2). The mean change between treatments was however not significant ($p=0.121$). There was a significant but slight increase in HDL-C in treatment I and II at 7.38% and 5.1% respectively (Figure 4.2). The mean change between the two treatments was also significant ($p = 0.012$).

The decrease in LDL-C was 5.56% in treatment I and 4.32% in treatment II (Figure 4.2). The change was significant at ($p<0.001$) in both treatments. The mean change between the two treatments was not significant ($p =0.242$). The mean reduction in total cholesterol was found to be higher for participants who had TC >5.1 than those who had TC less than 5.1mmol/L.

The mean reduction in triglycerides was found to be higher for participants who had TAG >2.25 than for those with TAG less than 2.25. The mean reduction in LDL-C was found to be higher for participants who had LDL-C > 4.2mmol/L than for those with less than 4.2mmol/L. Mean increase in HDL-C was higher in participants who had normal levels of HDL-C(1.03-1.55) and it was least in participants who had high HDL-C (> 1.55). Total cholesterol and HDL-C were statistically different ($p= 0.05$) between the two treatments indicating the effect of nutritional counseling.

Table 4.10 Mean change in serum lipid profile

	Treatment I				Treatment II				D1 &D2
	Baseline	**End**	**D1**	**P value(t-test)**	**Baseline**	**end**	**D2**	**P value(t-test)**	**P value(t-test)**
TC (mmol/L)	5.17±1.18	5.01±1.07	-.1589±.38	0.001	5.17±1.13	4.89±1.08	-0.27±0.20	0.001	0.001
TG (mmol/L)	1.88±.85	1.64±.83	-.24±.24	0.001	1.89±.90	1.57±.94	-0.32±0.48	0.001	0.121
HDL-C (mmol/L)	1.40±.41	1.51±.42	.10±.11	0.001	1.42±.42	1.49±.42	-0.07±0.09	0.001	0.012
LDL-C (mmol/L)	3.31±1.01	3.12±.92	-.18±.28	0.001	3.25±1.00	3.11±.99	.-14±25	0001	0.242

Values presented as the mean± standard deviation; n=85. Means are statistically significantly different at (P< 0.05). TC- total cholesterol, TG- triglycerides, HDL-C- high density lipoprotein cholesterol, LDL-C- Low density lipoprotein cholesterol, D1- delta change in treatment I, D2- delta change in treatment II.

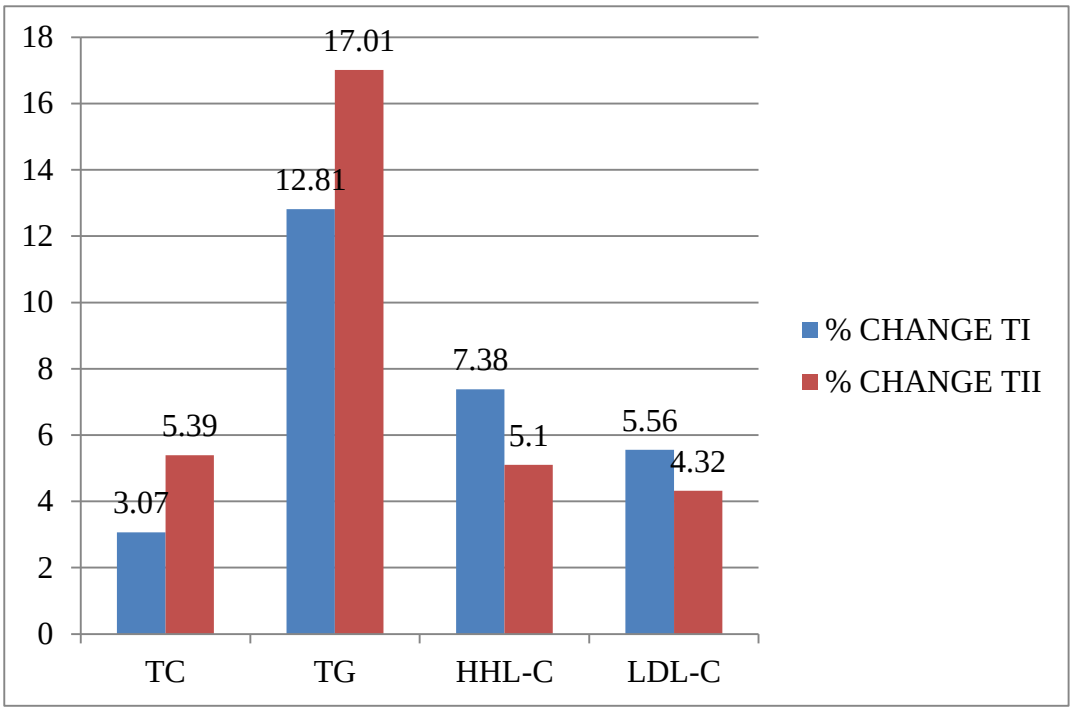

Figure 4.2. **Percentage change in lipid profile in the two treatments**

4.6.1 Effect of peanut supplementation on lipid profile

Table 4.11 shows regression analysis for lipid profile for treatment I and II. The test did not establish relationship between the changes in lipid profile in treatment I and treatment II and the change in poly unsaturated fatty acid, monounsaturated fatty acid and saturated fatty acid intake as predicted**.**

Table 4.11 Effect of peanut supplementation on lipid profile

		Treatment I			Treatment II		
		B	**t**	**P. value**	**β**	**t**	**P. value**
Change in TC	Fat	-0.037	-0.089	0.931	-0.094	-0.271	0.791
	PUFA	-0.239	-0.513	0.618	0.023	0.038	0.970
	Cholesterol	0.558	2.111	0.059	0.346	1.108	0.291
	SAT	0.498	0.772	0.457	-0.278	-0.702	0.497
	MUFA	-0.311	-0.408	0.691	0.248	0.344	0.738
Change in TG	Fat	-0.381	-0.965	0.355	0.569	2.326	0.040
	PUFA	-0.361	-0.815	0.432	0.829	1.973	0.074
	Cholesterol	0.547	2.178	0.052	0.453	2.069	0.063
	SAT	0.021	0.034	0.974	-0.516	-1.858	0.090
	MUFA	0.186	0.256	0.803	-0.773	-1.526	0.155
Change in HDL-C	Fat	0.638	1.430	0.181	-0.491	-1.420	0.183
	PUFA	0.105	0.210	0.837	-0.625	-1.053	0.315
	Cholesterol	0.231	0.812	0.434	0.189	0.610	0.554
	SAT	-0.447	-0.644	0.533	-0.124	-0.316	0.758
	MUFA	-0.334	-0.407	0.691	0.839	1.172	0.266
Change in LDL-C	Fat	0.007	0.014	0.989	-0.426	-1.268	0.231
	PUFA	-0.186	-0.330	0.748	-0.959	-1.661	0.125
	Cholesterol	0.039	0.122	0.905	-0.056	-0.186	0.856
	SAT	0.120	0.153	0.881	0.063	0.164	0.872
	MUFA	-0.045	-0.049	0.962	0.994	1.427	0.181

TC- total cholesterol, TG- triglycerides, HDL-C high density lipoprotein cholesterol, LDL-C- low density lipoprotein cholesterol, PUFA- poly unsaturated fatty acids, MUFA- mono unsaturated fatty acids, SFA- saturated fatty acids

4.6.2 Treatment period effect

Table 4.12 compares the changes in cardiovascular disease risk factors after peanut supplementation versus peanut plus counseling on a healthy diet during the two periods of treatment administration. For period 1(group 1), the changes in CVD risk factors were only significant for waist circumference when treatment I was compared to treatment 11. For period 2 (group 2), a significant difference was observed between treatment I and treatment 11 for waist circumference, total cholesterol and HDL-cholesterol.

Table 4.12 Comparison of peanut supplementation versus peanut plus counseling on a healthy diet on cardiovascular risk factors for the two groups.

	GROUP 1 (AB-BA) N=45			GROUP 2 (BA-AB) N=40		
	TREATMENT 1	**TREATMENT 11**	**t-test**	**TREATMENT 1**	**TREATMENT 11**	**t-test**
BMI	0.01±0.48	0.03±0.49	0.216	-0.12±0.38	0.01±0.41	0.184
WC	-0.71±2.00	0.50±2.46	0.013	-0.62±2.81	0.62±2.77	0.024
SBP	-2.02±12.09	0.142±18.32	0.328	3.42±14.66	-0.92±20.02	0.336
FBG	2.08±30.35	-0.42±27.03	0.707	-12.30±23.66	-0.30±34.25	0.057
TC	0.20±0.37	0.30±0.22	0.085	0.10±0.39	0.25±0.17	0.004
TG	0.26±0.24	0.30±0.45	0.463	0.21±0.33	0.33±0.51	0.164
HDL-C	-0.10±0.11	0.80±0.10	0.265	-0.11±0.11	-0.06±0.87	0.006
LDL-C	0.14±0.31	0.12±0.24	0.736	0.22±0.25	0.15±0.26	0.144

BMI- body mass index, WC waist circumference, FBG- fasting blood glucose, SBP- systolic blood pressure, TC- total cholesterol, TG- triglycerides, HDL-C- high density lipoprotein cholesterol, LDL-C- Low density lipoprotein cholesterol,

Table 4.13 shows the effect of sequencing of treatment I and treatment II in the order in which they were given. There was no observed statistical significant treatment-period effect on the changes in weight, BMI, waist circumference, systolic blood pressure, diastolic blood pressure, serum total cholesterol, serum triglyceride, serum HDL-C and serum LDL-C. There was

however treatment-period effects in fasting blood glucose in treatment I. This would mean that there was a likelihood of a carry- over effect for the two at 95% CI.

Table 4.13 Effect of differential sequencing of peanut versus peanut and healthy diet on cardiovascular disease markers

Category	Treatment	Treatment order		P value (t-test)
		Group 1n=45	**Group 2 n=40**	
WEIGHT	I	-0.327±1.28	-0.340±.98	0.221
	II	0.361±1.32	-.350±1.05	0.135
BMI	I	0.001±0.48	-0.129±0.38	0.179
	II	0.137±0.49	-0.011±0.41	0.122
WC	I	-0.711±2.00	-0.625±2.81	0.870
	II	0.505±2.46	0.625±2.77	0.835
SBP	I	-2.022±12.09	3.42±14.66	0.064
	II	1.422±18.32	-0.925±20.03	0.574
DBP	I	-0.733±8.39	1.100±7.21	0.286
	II	1.17±11.89	0.075±9.14	0.635
FBG	I	2.08±30.25	-12.30±23.60	0.018
	II	-0.422±27.03	-0.30±34.25	0.985
TC	I	0.208±0.37	0.102±0.39	0.204
	II	0.300±0.22	0.255±0.17	0.320
TG	I	0.261±0.24	0.219±0.23	0.429
	II	0.308±0.45	0.339±0.51	0.768
HDL-C	I	-0.101±0.11	-0.106±0.11	0.856
	II	-0.080±0.10	-0.06±0.08	0.454
LDL-C	I	0.146±0.31	0.227±0.25	0.200
	II	0.127±0.24	0.155±0.26	0.610
F.SCORES	I	2.31±5.80	1.77±5.93	0.675
	II	1.91±5.41	1.40±5.18	0.659

BMI- body mass index, LBM- lean body muscle, W.C- waist circumference, H.C- hip circumference, SBP- systolic blood pressure, DBP- diastolic blood pressure, FBG- fasting blood glucose TC- total cholesterol, TG- triglycerides, HDL-C high density lipoprotein cholesterol, LDL-C- low density lipoprotein cholesterol, F-score- Framingham's risk score

4.7 Assessment of cardiovascular risk

Framingham's risk score was used to determine the 10-year risk of developing a coronary heart disease. Table 4.14 shows the change in Framingham's risk score between baseline and end of the study period. Majority of the participants (60% and 64.7%) had low (<10%) 10-year risk of coronary heart disease at the baseline and end of the study respectively while 2. 4% of the participants had medium (20-30%) 10-year risk of coronary heart disease. There was no significant difference in the risk between males and females (p> 0.05) at baseline and at the end of the study period. There was a significant reduction in the 10year risk of coronary heart disease between baseline and end of study (p =0.03).

Table 4.14 Change in Framingham's risk scores

	Baseline			End			P value (baseline& end)
	Male	**Female**	**P value (chi)**	**male**	**female**	**P value (chi)**	
<10%	9(10.6)	42(49.4)	0.08	11(12.9)	44(51.8)	.356	0.03
10-20%	7(8.2)	25(29.4)		5(5.9)	23(27.1)		
20-30%	2(2.4)	0(0.0)		2(2.4)	0(0.0)		
>30%	0(0.0)	0(0.0)		0(0.0)	0(0.0)		

Approximated 10-years added risk of cardiovascular disease: low (< 10%), medium (10-20%), high (20-30%), and very high (> 30%) (Mancia et al., 2007)

4.8 Relationship between risk factors and Framingham's scores

Pearson correlation test was used to test relationship between the risk factors dietary intake and Framingham's risk score. Table 4.15 shows the relationship between these variables. Gender strongly correlated with education status ($r(85) = -0.262$, $p < 0.05$), occupation ($r(85) = -0.217$, $p < 0.05$), body mass index ($r(85) = 0.244$, $p < 0.05$), body fat ($r(85) = 0.335$, $p < 0.01$), serum triglycerides ($r(85) = -0.371$, $p < 0.01$) and Framingham's risk score ($r(85) = -0.605$, $p < 0.01$).

Age correlated with marital status ($r (85) = 0.246$, $p < 0.05$), blood pressure ($r (85) = 0.390$, $p < 0.01$) and Framingham's risk scores ($r (85) = 0.541$, $p < 0.01$). Level of education only correlated with occupation ($r (85) = -0.325$, $p < 0.01$) while smoking only correlated with Framingham's risk score ($r (85) = -0.443$, $p < 0.01$).

Physical activity correlated with blood pressure while BMI correlated with body fat ($r (85) = 0.710$, $p < 0.01$), waist circumference ($r (85) = 0.777$, $p < 0.01$), hip circumference ($r (85) = 0.864$, $p < 0.01$) and blood pressure ($r (85) = 0.262$, $p < 0.05$).

Body fat correlated with waist circumference ($r (85) = .473$, $p < 0.01$), hip circumference ($r (85) = 0.730$, $p < 0.01$) energy intake ($r (85) = 0.484$, $p < 0.05$) and Framingham's risk score ($r (85) = -0.350$, $p < 0.01$). Waist circumference correlated with hip circumference ($r (85) = 0.730$, $p < 0.01$), blood pressure ($r (85) = 0.413$, $p < 0.01$) and fasting blood glucose ($r (85) = 0.239$, $p < 0.05$). Hip circumference correlated with blood pressure ($r (85) =0.279$, $p < 0.01$) while blood pressure correlated with Framingham's risk score ($r (85) = 0.301$, $p < 0.01$).

Fasting blood glucose correlated with poly unsaturated fatty acid intake ($r (85) = 0.554$, $p < 0.05$) while total cholesterol correlated with triglycerides ($r (85) =0.262$, $p < 0.05$), serum LDL-C ($r (85) = 0.777$, $p < 0.01$) and Framingham's risk scores ($r (85) =0.459$, $p < 0.01$). Serum

triglycerides correlated with HDL-C ($r(85) = -0.351$, $p < 0.01$), LDL-C ($r(85) = -0.271$, $p < 0.05$) and Framingham's risk scores ($r(85) = 0.256$, $p < 0.05$). LDL-C correlated with Framingham's score ($r(85) = 0.425$, $p < 0.01$).

Fat intake correlated with energy ($r(17) = 0.571$, $p < 0.05$), PUFA ($r(17) = 0.649$, $p < 0.01$), cholesterol ($r(17) = 0.813$, $p < 0.01$), saturated fat ($r(17) = 0.821$, $p < 0.01$), MUFA ($r(17) = 0.830$, $p < 0.01$) and vitamin E ($r(17) = 0.494$, $p < 0.05$). Energy intake correlated with PUFA ($r(17) = 0.582$, $p < 0.05$), cholesterol ($r(17) = 0.870$, $p < 0.01$) and MUFA ($r(17) = 0.636$, $p < 0.01$). Cholesterol intake correlated with ($r(17) = 0.668$, $p < 0.01$) saturated fatty acids and MUFA ($r(17) = 0.743$, $p < 0.01$) while saturated fatty acids correlated with MUFA ($r(17) = 0.952$, $p < 0.01$).

Table 4.15: Relationship between risk factors, dietary intake and Framingham's risk score

	SEX	AGE	EDU	OCC	MAR	SMOK	ALC	P E	BMI	BF	WC	HC	BP
SEX	1	-.262*	.015	-.217*	.106	.148	.054	.012	.244*	.723**	.010	.335**	.005
AGE		1	.123	-.120	.246*	-.062	.135	-.093	-.113	-.105	.095	-.075	.390**
EDU			1	-.325**	-.097	-.008	.105	.205	-.025	.100	-.025	.027	-.098
OCC				1	-.035	.109	-.127	-.140	-.009	-.173	.108	-.028	-.012
MAR					1	.093	.117	-.101	.027	.148	.158	.043	.176
SMK						1	.117	-.147	.180	.159	.097	.121	-.016
ALC							1	-.005	.003	.056	.096	-.011	.124
P E								1	-.008	-.105	.023	-.017	-.219*
BMI									1	.710**	.777**	.864**	.262*
BF										1	.473**	.702**	.156
WC											1	.730**	.413**
HC												1	.279**
BP													1
FBG													
TC													
TG													
HDLC													
LDLC													
FAT													
FIBRE													
ENERGY													
PUFA													
CHL													
SAT													
MUFA													
VITE													
F RISK													

FBG	TC	TG	HDLC	LDLC	FAT	FIBRE	ENER	PUFA	CHL	SAT	MUFA	VITE	F RISK
.001	-.189	-.371**	.187	-.200	-.294	.222	-.465	-.190	-.143	-.352	-.431	.341	-.605**
.031	.129	.120	.026	.154	-.395	-.303	-.094	-.207	-.221	-.228	-.160	-.636**	.541**
.088	-.011	.106	-.181	.100	.174	-.285	.372	.160	.401	.173	.176	.364	.054
-.092	-.112	.082	-.010	-.092	-.048	.188	-.251	-.232	-.254	-.149	-.196	-.252	-.023
.101	-.177	.006	.078	-.127	.285	-.115	.255	.354	.578*	.278	.259	.227	-.005
.150	-.205	.008	-.048	-.195	.075	-.169	.243	-.003	.157	.124	.121	.099	-.443**
.156	.102	.026	-.032	.077	.050	.252	.112	.160	.190	.099	.156	.282	.055
.028	-.193	-.026	-.146	-.180	-.384	.102	-.195	-.032	-.209	-.386	-.333	-.171	-.038
.170	-.051	.007	-.032	.036	-.426	.352	-.341	-.169	-.391	-.358	-.351	.166	-.187
.129	-.069	-.199	.052	-.026	-.311	.227	-.484*	-.170	-.180	-.357	-.393	.269	-.350**
.239*	-.028	.100	-.065	.009	-.339	.263	-.204	-.108	-.310	-.303	-.296	-.017	.046
.086	-.136	-.113	.032	-.033	-.335	-.064	-.481	-.414	-.429	-.318	-.365	.249	-.205
.013	.131	-.012	.077	.105	-.394	.141	-.477	-.246	-.317	-.363	-.429	-.016	.301**
1	.005	.016	-.111	.003	.155	.064	.057	.554*	.467	-.097	.019	.270	-.076
	1	.252*	.080	.777**	.058	-.188	.388	.153	.190	.308	.349	.097	.459**
		1	-.351**	.271*	-.096	.133	.472	.168	-.038	.080	.181	-.536*	.256*
			1	.067	.061	.028	-.415	-.050	.140	.001	-.093	.371	-.071
				1	.043	-.355	.411	.078	.092	.120	.236	-.055	.425**
					1	-.128	.571*	.649**	.813**	.821**	.830**	.494*	.018
						1	-.133	.134	-.060	-.085	-.103	.195	-.433
							1	.582*	.658**	.677**	.750**	.070	.235
								1	.870**	.466	.636**	.159	-.013
									1	.668**	.743**	.357	-.096
										1	.952**	.443	.176
											1	.300	.234
												1	-.465
													1

Correlation is significant at the 0.05 level (2-tailed). **. Correlation is significant at the 0.01 level (2-tailed).

BMI- body mass index, LBM- lean body muscle, W.C- waist circumference, H.C- hip circumference, SBP- systolic blood pressure, DBP- diastolic blood pressure, FBG- fasting blood glucose TC- total cholesterol, TG- triglycerides, HDL-C high density lipoprotein cholesterol, LDL-C- low density lipoprotein cholesterol, F-risk- Framingham's risk score, PUFA- poly unsaturated fatty acid, MUFA- mono unsaturated fatty acids, CHL- cholesterol, OCC- occupation, SMK –smoking, ACL- alcohol, BF- body fat, MAR- marital status, EDU- education level

CHAPTER FIVE: DISCUSSION

5.1 Social demographic characteristic

Out of the 85 respondents who completed the intervention, 18 (21.2%) were males while 67 (78.8%) were females. The difference in proportion in this study could be due to fewer males attending the CCC. It could also be explained by the results from the 2008-09 KDHS that indicate HIV prevalence in women age 15-49 is 8.0 percent, while for men age 15-49, it is 4.3 percent. This female-to-male ratio of 1.9 to 1 is higher than that found in most population-based studies in Africa.

Majority of the respondents were aged between 40 to 49 years while those who were over 50 years were less than thirty percent. More males were married than the females while majority of the females were single parents. These findings differ with the KDHS (2008-08) report that puts married females at 50% and males at 47%. It also differs in the percentage of divorced people in that the survey reports less than 1% while this study shows 20% divorce rate. This could be because this study had a smaller sample size compared to the survey.

Majority of the respondents had completed their secondary education with more females having completed the secondary education than males. The study results also differs with the KDHS (2008-09) for Central Province as there are more females than males who have completed secondary and post-secondary education. This could be due to difference in proportion of females and males in the study and due to a smaller sample size. Gender strongly correlated with education status ($r(85) = -0.262$, $p < 0.05$), occupation ($r(85) = -0.217$, $p < 0.05$). The difference

in the results of this study could also be attributed to the proportion of males to females in this study.

5.2 Baseline cardiovascular disease risk factors

Waist hip ratio and waist circumference were based on WHO (2008) guidelines. Twenty four percent had obesity class I (30-34.9), 8.2% had obesity class II (35-39.9) while 1.5% had obesity class III (> 40. Another 31.8% were overweight (25-29.9). Thirteen percent of the males and 54% of the females were overweight or obese. These findings are higher than the normal Kenyan population prevalence for obesity for females which is 23% and 35% for central province (KNBS & ICF Macro, 2010). They are also slightly higher than findings on the sero-positive patients in western Kenya where 10.6% men and 22.6% women were overweight or obese (Bloomfield, Hogan, Keter Sang & Carter, 2011). If this population estimate is a reflection of the prevalence of obesity in the whole population, then the study results indicate a higher prevalence of obesity in HIV positive patients in this region. The higher prevalence in obesity in this study could be attributed to the low and moderate activity level (27.1% and 61.2%, respectively). It could also be attributed to higher number of females in the sample as there is a positive correlation between sex and BMI.

Systolic blood pressure was used to determine the hypertension status. Over 40% of the respondents were found to be pre hypertensive (120-139) while 28.2% had hypertension stage I (140-159) and another 11.8% had hypertension stage II (>160). This high prevalence was also reported by Bloomfield et al. (2011) who found the prevalence of pre hypertension and hypertension in western Kenya to be 30.8% and 20%, respectively. Njeru, (2009) found the prevalence of hypertension among HIV positive patients in Thika District Hospital was 18%

(95% Confidence Interval [CI]:12.5-23.5%) who have also used the same classification for hypertension.

Our prevalence estimates may be lower than reported in developed countries due to the low population rate of hypertension in the region. The most recent population based hypertension prevalence data for Kenya are from 1987 and suggest a rate between 1.2% and 2.2% (WHO 2010). If the population estimates reflect the true prevalence in the population, our findings would suggest a higher than expected rate of hypertension among Kenyan HIV+ individuals. This is consistent with observations that HIV can have effects on the vasculature of infected individuals at a relatively young age (Guaraldi, Zona, Alexopoulos, Orlando & Carli, 2009). Findings from developed countries suggest that PI use may be driving both hypertension and obesity (Kaplan, Kingsley, Sharrett, Li & Lazar, 2007). However, in contrast to studies from developed countries, it is unlikely that use of protease inhibitors were a major cause of obesity in our sample since the proportion of people using PIs was low (5.9%). This increase in hypertension prevalence could be explained by high BMI observed as there is a positive correlation between BMI and high blood pressure. High waist and hip circumference seen in this population could also have contributed to the high prevalence of hypertension as there is a positive correlation observed between these two variables. Age is also seen to positively correlate with BMI in this study as majority of the participants were aged forty years and above.

This study found that (22.4%) had high total cholesterol (>6.2mmol/l), 30.6% had borderline high (5.1-6.12mmol/l) while 47.1% had desirable levels. Over 21% of the respondents had borderline high triglyceride (1.69-2.242mmol/l) while 34.1% had high serum triglycerides (2.25-

5.62mmol/l). Only 5.9% had low serum levels of HDL-C (<1.032mmol/l) while 34.1% had high serum HDL-C (>1.552mmol/l). Twenty nine percent of the respondents had borderline high serum levels of LDL-C (3.4-4.12mmol/l) while 7.1% had high (4.2-4.92mmol/l) and another 4.7% had very high serum LDL-C (>4.912mmol/l). These results are consistent with the Manuthu et al (2008) whose study in Kenyatta National Hospital found the prevalence of hypercholesterolemia to be 39.2%, elevated LDL-C to be 40%, low HDL-C to be 14.6% and high triglycerides to be 25.6%. Molla et al. (2014) studied dyslipidemia in HIV positive patients on ART in Addis Ababa Ethiopia and found that 23% had high LDL-C, 46.8% had high triglycerides, 42% had high total cholesterol and 50.8% had low HDLC. Antiretroviral therapy may exacerbate lipid abnormalities and dyslipidemia may develop in up to 70% and 80% of HIV-infected patients with hypertriglyceridemia occurring in majority of cases (60% to 100%) of treated patients. The presence of hypertriglyceridemia enhances development of small, dense low density lipoproteins particles (Manfredi et al., 2005). Norman and colleagues found that high serum cholesterol level (≥3.8 mmol/l) accounted for 59 percent of ischemic heart disease and 29 percent of ischemic stroke burden in adults age 30 and over.

This study found that 4% had pre diabetes (100- 126mg/dl) while 5 % had diabetes (> 126mg/dl). Dysglycemia was also reported by Manuthu et al. (2008) at 20.7% among HIV patients while Molla et al. (2014) reported prevalence of Dysglycemia at 7.5%. The prevalence of diabetes in this study is higher than the national prevalence which is 3.5% (Shaw et al., 2009). This could be due to small sample as compared to the national sample. The results also agree with studies done in developed countries. Among HIV-infected adults with lipoatrophy or fat accumulation, diabetes mellitus was seen in 7.0 %, as compared with 0.5 % of otherwise healthy

control subjects matched for age and body-mass index (Mayere, Hadigan, Jeste, Anderson & Tsay, et al., 2001).

Impaired glucose tolerance was present in more than 35 % of HIV-infected subjects as compared with 5 % of otherwise healthy control subjects matched for age and body-mass index (Mayere et al., 2001). Antiretroviral therapy may also alter glucose homeostasis. Protease inhibitors (including Indinavir, Amprenavir, Nelfinavir, and Ritonavir (Murata, 2000; Rudich, 2001; Ben-Romano, 2003) have been shown to induce insulin resistance in vitro by reducing glucose transport mediated by glucose transporter 4 (Murata, 2000).This is seen to occur without affecting postreceptor insulin signaling. Protease inhibitors may also decrease pancreatic beta-cell insulin secretion (Woerle, et al., 2003) but insulin resistance is seen to be the primary defect. Direct effects of nucleoside analogues on glucose metabolism have not been demonstrated, but such drugs may contribute to insulin resistance indirectly through changes in fat distribution.

Smoking and alcohol consumption were low in this study. The rates of smoking were 1.2% for females and 4.7% for males. This is lower than the estimated national prevalence for smoking which is 1.5% for females and 25.5% for males (World Bank, 2009). This is comparable to studies done elsewhere since men tend to consume more alcohol and smoke more cigarettes than females as found in Kenyan surveys CBS 2004, KNBS and ICF Macro 2008-09. In these surveys, about 2% of women used tobacco in its various forms whereas 1% smoked cigarettes.

This study assessed the cardiovascular disease risk factor and their correlation to Framingham's risk score that predicts 10-year risk of developing coronary heart disease. The study found

correlation between Framingham's risk score with age, sex, body fat blood pressure, total cholesterol, and LDL cholesterol. Similar results have been highlighted by O'Donnell and Elosua (2008) in a review of Cardiovascular Risk Factors: Insights from Framingham Heart Study.

5.3 Proximate composition of Peanut of Red Valencia variety

The study shows crude protein in raw peanut is 22.02-28.99, while in roasted is 31.45-33.17%. This is higher than the results of Musa, Kalejaiye and Ismaila, (2010); Kumar, Shankar, Vasanthi, Vishnuvardhan & Purushotham, (2013); Atasie, Akinhanni & Ojiodu (2009).The results also show that protein content increased when roasted. This does not agree with Atasie, et al., (2009) whose analysis reported that the percentage of crude protein decreases when the groundnut seeds are subjected to heat treatment (sun-dried and roasted). This increase in crude protein levels could be explained by decrease in the water content hence concentrating the proteins.

Fat content ranged between 43.3- 48.3 in raw and 47.3-49.15 in roasted peanuts. This was within the range reported by Shokunbi, Fayomi, Sonuga & Tayo, (2012) who reported fat content as 48.06-50.99, 33.6-54.95 (Asibuo , Akromah ,Safo-Kantanka ,Adu-Dapaah, Ohemeng-Dapaah & Agyeman,2008), 45.09-51.63% (Shad , Perveez , Na Waz, Khan & Amean Ullah ,2009),32.7-53.1% (Musa, et al., 2010).

The fibre content in this study ranges between 9.80-10.83% in raw, and 5.47-6.56% in roasted peanuts. This is higher than 3.7% (Atasie, et al., 2009), 3.3-4.4% (Campos-Mondragon et al., 2009) and 2.76-3.07% (Shokunbi, et al., 2012). Kumar et al., (2013) reported 2.91% crude fibre in raw peanuts and 3.09% in roasted peanut. Carbohydrate content in this study was between 9.49-12.37% in raw peanut and 6.63-7.87% in roasted peanut. This was lower than 19.02-27.16 (Asibuo, et al., 2008), 18.9-23.4% (Campos-Mondragon et al., 2009) and 17.03-18.5%

(Shokunbi, et al., 2012) but higher than 1.81% reported by Atasie, et al., (2009). Kumar, et al., (2013) reported carbohydrate content for raw peanut at 25.3% and 26.5% for roasted peanut. The differences in these results could be attributed to the different varieties that others have analyzed. The results indicated that total ash and crude protein content of raw groundnut was higher than the roasted groundnut seeds. These results are similar to Kumar, et al., (2013) who also found higher crude protein content in roasted groundnut when compared to that of raw groundnut. This could be contributed by low moisture content in roasted groundnuts that results in concentration of this nutrient in dry matter. Crude carbohydrates levels of raw groundnut are lower when compared with that of roasted this also agrees with Kumar, et al., (2013) and Atasie, et al. (2009). Ayoola & Adeyeye (2012) stated that crude carbohydrate content were higher in the roasted and sun-dried than in raw groundnut seeds.

Groundnut seed is a rich source of fat, protein, monounsaturated fat and also it is very low in cholesterol. In this study, raw peanuts had significantly lower crude fat percentage and roasted groundnut seeds in Red Valencia. This is different from Kumar, et al., (2013) who found similar crude fat percentage whereas Ayoola and Adeyeye (2010) have reported that fat content was higher in raw groundnut seeds and seems to be declining in the sun-dried and roasted groundnut seeds depending on the intensity of heat. Atasie, et al., (2009) have also reported that the fat content in groundnut seeds is important for human diet as it facilitates fat soluble vitamin absorption and also high in energy nutrient value.

Crude fiber content is low in roasted groundnuts when compared to raw groundnut. Atasie, et al., (2009) have reported that diet low in crude fiber is undesirable and may cause constipation,

cancer and piles. Results show that the groundnut seeds of this cultivar maintain good crude fiber percentage both in raw and roasted form and roasted groundnut is more advantageous in nutritional value than the raw groundnut.

The moisture content of the raw groundnut seeds sample was higher than that of the roasted groundnut seed. Previous studies have also shown that the moisture content of the raw seeds were reported to be higher than those of the groundnut seeds subjected to heat treatment (Ayoola and Adeyeye 2010; Bhuiyan, Islam & Iji, 2013). The moisture content of the raw groundnut was higher than the roasted groundnut because the raw groundnut is not previously exposed to any heat. The groundnut seed with 10.1% moisture content at 35°C survived for 12 weeks and the survival period increased up to 120 weeks when the moisture content is reduced to 4.4%. Low moisture percentage of groundnut seed also prevents it from the susceptibility to the fungal pathogens (Sastry, Upadhyaya & Gowda, 2007).

5.4 Dietary intake

Average consumption of energy at baseline was below the RDAs for both males and females (3139 ±365 Kcals and 2479 ±312Kcals respectively. The results are similar to Gikonyo, Ndungu, Kuria & Mbithe (2014) in a study in HIV infected adults in Mweiga, Nyeri County. The protein and micronutrient intake was also below the recommendation dietary allowance. This could be attributed poor dietary practices and poor dietary diversity.

This study was an interventional cross over study. Eighty grams (80g) of peanut was given to be taken together with regular diet in treatment I. The same amount was given in treatment II but

the participants were counseled on healthy diet. Energy intake did not change significantly when the peanuts were supplemented to regular diet and healthy diet. These findings agree with Alper & Mattes, 2003 who found similar results in a 30- week crossover intervention where subjects were provided 500 (±136) kcal as peanuts during an eight-week free feeding (FF) diet. Mckiernan, Lokko, Kuevi, Sales, Costa, Bressan, et al, 2010 also did not report significant changes in energy intake in their 4 week randomized clinical trial.

There was also low intake of animal products per week. This is similar to findings by Kuria (2010) in a study done among PLWHA in Thika and Bungoma County.

Compared with baseline, energy intake from fat increased significantly during TI and TII (both $P< 0.001$), MUFA and PUFA increased significantly during TI and TII (all $P< 0.001$), while SFA remained unchanged. Similar finding were also reported by Alper, et al., 2003, Lokko, et al., 2007 and Mckiernan, et al., 2010. There was a significant decrease of carbohydrate intake during TI and TII (both $P= 0.001$) which was similar to Mckiernan, et al.,2010 but different in Alpers, 2003 study who found no decreases in carbohydrate sources when peanut was given with regular diet.. Dietary intakes of vitamin E ($P< 0.001$) increased significantly from baseline in treatment I as well as in treatment II (vitamin E ($P< 0.001$). All these results were estimates because they were based on the 24 hour recall.

These changes can be attributed to inclusion of peanuts in the diet since there was very low intake of poly unsaturated and monounsaturated fatty acids in the diet at baseline.

In this study however, Folate and Magnesium did not change significantly in both treatments from baseline. This however was different in the study by Alper et al., 2003 who reported significant increase in both Magnesium and Folate. This could be due to difference in the peanut

cultiva that was used in their study. Inclusion of peanut in the diet has been found to increase fat, MUFA and PUFA significantly (Alper and Mattes, 2003; Kris-Etherton et al., 1999; Lokko et al., 2007; McKiernan et al., 2010). All these results were estimates because they were based on the 24 hour recall.

5.5 Effect of peanut supplementation and peanut plus healthy diet on nutritional status markers

There were no significant differences between peanut supplementation on healthy diet versus peanut supplementation with regular diet at baseline with respect to nutritional status markers. There was also no significant time period effect respect to change in nutritional status markers following the intervention. This study did not report significant difference in weight, BMI, waist and hip circumference and fasting blood glucose during the two treatments with peanut. This was not expected given that 80g of peanut was expected to contribute an extra approximately 500Kcal/day. A large cohort of women followed for 16 years found a slight decrease in the body mass index (BMI) even as the consumption of nut increased. After adjustment for potential confounders, their average weight gain across nut consumption categories was not significantly different (Jiang, et al., 2002).

Other studies have found no net weight gain when nuts are consumed as a replacement food. The same has been reported even when nuts have been added to diet, even though the intake of total energy was substantially increased (Morgan & Clayshulte, 2000; Morgan, Horton, Reese, Carey, Walker, & Capuzzi, 2002).

No weight gain was reported when 48 g of walnuts was added to the diet for six weeks despite increase in energy intake by 1661 kJ/day (Almario, et al., 2001). Since the nut intervention duration is relatively short in most of these trials, the long-term effect of nut intake may not be

indicated. However an isolated intervention study showed a negative effect on body weight. A slight but significant increase in body weight (0.9 kg for men, 0.3 kg for women) was observed when normal weight participants were given 100g of almonds to add to their usual diet for a period of four weeks (Lovejoy, et al., 2002).

There are several mechanistic explanations why nut intake might protect against weight gain. Nuts are rich in fiber and are energy-dense, high fat foods, with a high content of unsaturated fatty acids. Evidence suggests that monounsaturated and polyunsaturated fatty acids are more readily oxidized (Piers, Walker, Stoney, Soares &O'Dea, 2002) and have a greater thermogenic effect (Casas-Agustench, Lo´pez-Uriarte, Bullo´, Ros, Go´mez- Flores &Salas-Salvado, 2009) than saturated fatty acids, which can lead to less fat accumulation. Nuts are also good sources of plant proteins, which may enhance satiety and suppress subsequent hunger (Garcı´a-Lorda, Megias Rangil, Salas-Salvado, 2003). A high content of dietary fiber, from both vegetables and nuts, is believed to increase satiety and reduce feelings of hunger (Salas-Salvado et al., 2006). Furthermore, fat malabsorption has been reported after nut intake and attributed to the fat being contained within walled cellular structures that are incompletely digested in the gut (Ellis, Kendall, Ren, Parker, Pacy & Waldron, 2004), an effect that can be compounded by incomplete mastication (Cassady, Hollis, Fulford, Considine, & Mattes, 2009). Finally, other mechanisms of protection against adiposity may depend on many other bioactive compounds that are present in nuts (Segura, Javierre, Lizarraga & Ros, 2006).

5.5 Effect of peanut supplementation and peanut plus healthy diet on serum lipid profile

In this study there was a significant reduction in serum total cholesterol in both treatments (3% and 5.3%, respectively). Change in triglycerides was also significant in both treatments (12.8%

and 17.1%, respectively). LDL-C decreased significantly in both treatments (5.5% and 4.3%, respectively).

Epidemiological studies and clinical trials have demonstrated benefits of nuts and peanut consumption on CAD risk and associated risk factors (Ros, 2009; Djousse, et al., 2009). These findings agree with a study by Lokko et al. (2007) who reported a 7.2% decrease in total cholesterol and 20% decrease in triglycerides when 500kcal/day peanut was incorporated with daily diet for eight weeks. A recent study by McKiernan et al. (2010) reported significant reductions in total cholesterol, LDL-C and TAG concentrations were observed when hyperlipidemic individuals consumed 56 g of whole raw, roasted unsalted, roasted salted or honey roasted peanuts, or ground peanut butter daily for 4 weeks. However HDL-C concentrations increased significantly from baseline. Lokko et al (2007) also reported significant decrease in total cholesterol (7.2%) and triacylglycerol (20.0%) after subjects were provided 2,092 kJ/day (500 kcal/day) peanuts to incorporate into their daily diet for 8 weeks at any time and in any form they chose. However, individually, high-density lipoprotein-cholesterol and low-density lipoprotein-cholesterol levels did not change significantly. Recently the findings of a pooled analysis of 1,284 observations contributed by 583 unique participants from 25 clinical studies performed with different nuts, including peanuts, and conducted in seven different countries have been reported Sabate,et al (2010). The results show a dose-response cholesterol lowering effect and indicate that, for an average daily intake of 67 g of nuts (roughly equivalent to 20% of energy), the mean estimated reductions of total cholesterol and LDL-cholesterol were 11 mg/dL (5%) and 10 mg/dL (7%), respectively. Nuts had no significant effect on HDL-

cholesterol or triglycerides, except in participants with serum triglycerides >150 mg/dL, in whom a significant 10.2 mg/dL reduction was observed

In this study the mean decrease in serum total cholesterol, Low density lipoprotein and triglyceride was higher but not significantly higher in participants with high levels compared to those with normal serum levels. This is in contrast to a peanut intervention study that reported a 12% reduction in total cholesterol and a 10% reduction in LDL-C in normocholesterolaemic individuals consuming whole peanuts and peanut butter for 24days (Kris-Etherton,et al., 1999). However, of note, the reductions observed by Kris-Etherton, et al., (1999) among normocholesterolaemic individuals were greater in those with the highest concentrations at baseline.

The study did not find a significant relationship between the changes in dietary PUFA, MUFA, dietary fat and fibre (individually or together) and the changes in the lipid profile after linear regression analysis. This is because there are other component in nuts such as fibre and phytosterols together with unsaturated fatty acid profile that are likely to contribute to the favourable effects nuts have on the plasma lipid (Kris-Etherton ,Hu, & Ros, 2008; Griel et al., 2006). The decrease in the triglycerides in this study may be due to the reduction in carbohydrate intake when the peanuts were added to the diet. Triglyceride concentration decreases with reduction in carbohydrate intake (Appel, Sacks & Carey, 2005), and thus, the decreases in carbohydrate intake reported may have had an independent effect on lipid concentrations. It is estimated that a reduction of total cholesterol and LDL-C by 1 mmol/l may result in 24-28%

decrease in the relative risk of CHD mortality (Gould et al., 2007). Another decrease in triglyceride by 1.0 mmol/l may result in 14-37% decrease in overall CVD risk (Cullen, 2000).

CHAPTER SIX: SUMMARY, CONCLUSIONS AND RECOMMENDATION

6.1 SUMMARY

6.1.1Social economic status

Out of the 85 respondents 18 (21.2%) were males while 67 (78.8%) were females. Age, level of education and occupation did not vary significantly between males and females while marital status differed. Age, marital status, education level and occupation were not significantly different between group 1 and group 2

6.1.2Proximate composition of peanut

Fat, protein and total ash increased while moisture fibre while carbohydrate content decreased after roasting.

6.1.3 Dietary intake

Compared with baseline, energy intake from fat, Vitamin E, MUFA and PUFA increased significantly during TI and TII. There was a significant decrease of carbohydrate intake during TI and TII.There was no significant difference between the dietary intake in treatment I and treatment II.

6.1.4 Nutritional status

There was no significance difference in nutritional status within the month and between the months. There was no significant change in nutritional status after consumption of peanut with regular diet and consumption of peanut combined with nutritional counseling on healthy

diet.There was no statistically significant difference in nutritional status between the two treatments.

6.1.5 Lipid profile

The mean reduction in serum TC, TG,LDL-C while HDL-C increased significantly.Reduction was higher in participants with higher serumTC, TG, LDL-C .Mean increase in HDL-C is higher in participants who had normal levels of HDL-C.Total cholesterol and HDL-C were statistically different between the two treatments indicating the effect of nutritional counseling

6.1.5 Cardiovascular risk factors

Twenty nine percent of the respondents were aged above 50 years. Only 5.9% of the respondents smoked while 8.2% drunk alcohol.Twenty seven percent (27.1%) had low physical activity.Thirty four percent were obesity while for WHR 3.5% of males had high risk while 42.9% of females had high risk. Over 40% of the respondents had pre hypertension while 28.2% had hypertension stage I and another 11.8% had hypertension stage II. Nineteen (19%) had high total cholesterol while 30.6% had borderline high. Over 21% of the respondents had borderline high triglyceride while 34.1% had high serum triglycerides.Only 5.9% had low serum levels of HDL-C.Twenty nine percent of the respondents had borderline high serum levels of LDL-C while 7.1% had high and another 4.7% had very high serum LDL-C. There was a statistically significant difference between males and females for waist hip ratio, smoking, triglyceride and HDL-C.

6.1.7 Assessment of cardiovascular risk

Majority of the participants had low 10- year risk of coronary heart disease at the baseline and end of the study respectively.There was a significant reduction in the 10-year risk of coronary heart disease between baseline and end of study.

6.2 CONCLUSION

- High Prevalence of overweight and obesity, hypercholesterolemia (total and LDLC-C) and hypertriglyceridemia
- Consumption of peanut with regular diet or with counseling on healthy diet improves the lipid profile in people living with HIV and therefore reduces the 10 year risk of developing CHD
- Peanut supplementation had no significant effect on BMI, waist circumference and hip circumference

6.2.1 HYPOTHESIS TESTING

- **H_{01}.** Supplementation with peanuts has no significant affect on nutritional status in HIV-infected patients –NOT REJECTED
- **H_{02}.** Supplementation with peanuts has no significant affect on blood lipid profile in HIV-infected patients- REJECTED
- **H_{03}.** Supplementation with peanuts has no significant effect on the overall 10 year risk of developing coronary heart disease - REJECTED
- **H_{04}.** There is no significant difference between the effects of treatment 1(80g of peanut and regular diet) and treatment II (80g of peanut and counseling on healthy diet) on lipid profile, hip and waist circumference and BMI. NOT REJECTED

6.3 RECOMMENDATION

6.3.1 Policy makers and practice

- Pea nut consumption should be encouraged as it has been found to lower cardiovascular risk significantly due to reduction in total cholesterol, triglyceride and LDL-C.
- Since roasting reduces the fibre content of peanut, the skin should not be peeled while eating as it will reduce the content further.
- The policy makers should promote the incorporation of this food base supplement in order to lower the economic burden cardiovascular diseases are imposing on the economy

6.3.2 For further research

- There are gaps in the information on prevalence of obesity, hypertension and diabetes in the Kenyan population. There is need for a national wide research on the prevalence of these conditions in the HIV positive population.
- The study has also found gaps on the reasons for the high dyslipidemia status among HIV positive patients in Nyeri level five hospitals despite the fact that there is low usage of PI regimen in this population.
- Assessment of cardiovascular risk factor is a huge undertaking. There is need for further research on assessment of risk factors in a longitudinal study in a large sample size in Kenya and Sub Saharan Africa where such data is lacking.
- There is need to to come up with a tool for estimating the 10 year risk for developing CHD that is validated in HIV-infected population in SSA.
- There is need to establish the difference in cardiovascular risk among peanut consuming communities and those that do not consume in Kenya.

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APPENDICES

APPENDIX A:Letter of Introduction.

Dear Sir/ Madam,

I am a staff of Kenyatta University in the Department of Food Nutrition and Dietetics. I am undertaking a research project on cardiovascular risk factors and effect of peanut supplementation on CVD biomarkers in adults with HIV dyslipideamia. The study period will be 24 weeks during which you will be assessed for lipid profile and C reactive protein and other anthropometric measurements. 4 ml of blood will be drawn four times during the study period and the dates will be communicated to you. A summary of your medical history necessary for the study will be accessed through your consent. I will be grateful if would volunteer to assist in this project by giving your consent.

All the information provided will be treated with utmost confidence and none of the participants will be identified by name or CCC Numbers.
Any enquiries may be directed to me through the address and phone number provided below.

Thank you for assistance and participation.

Regina Wangui Kamuhu,
Department of foods Nutrition and Dietetics,
P.O BOX 43844- 00100, NAIROBI.
Tel No: 0717 655 404

APPENDIX B: BASELINE SOCIAL ECONOMIC STATUS QUESTIONAIRRE.

CLIENTS Code	CCC Number	Date of Interview (dd/mm/yy)	Name of Interviewer	Tel. No	Duration of stay in present area: months
		____/____/____			

HOUSEHOLD DATA: Household characteristics (fill in the appropriate codes). Please tell me the number of people living in your household, , sex, age, and marital status, level of education, occupation and whether they earn any income or not.

How many people live in this household together and share meals? (Household☐☐)

1.1 Sex 1= Male 2= Female	1.2 Age	1.3 a)What is the highest level of school you attended (enter code from a list) 1= lower primary 2= Upper Primary 3=Post-Primary 4=Secondary 5= College 6= University 7=adult education 8= No formal education 7=Don't know b)What is the highest (standard/form/year) you completed at that level? IF NONE, WRITE '00'		1.4 Main Occupation of the household head and the respondent or caregiver (enter code from list) *(Ask this question to the respondent/ caregiver. The responses can be more than one)* 1=Agricultural labour 14=Waged labour 2=Livestock herding 15= Brewing 3=Own farm labour 16=Weaving/basketry 4=Employed(salaried) 17= Student 5=Merchant/trader 18= Very old 6=Petty trade 19=Herds boy/girl 7=Unemployed 20=Quarry 8=Firewood/charcoal selling 9=Merchant/trader 10=Mining 11=Housewife 12=Domestic help 13=masonry/carpentry 21=Others (specify)_____	1.5 Marital status of the household head/ respondent or caregiver 1= Single parent 2= Married or living together 3= Divorced/separated 4= Widowed **6= Any other specify _____**
SEX		Level	Yearcompleted	OCCUPATION	MARITAL STATUS

2. SANITATION

2.1. Does your household have access to a toilet/ latrine facility? 1=Yes 2=No IF NO, GO TO QUESTION 6.4	2.2. If yes, what type of toilet facility do you have? 1=Bucket 2=Traditional pit latrines 3=Ventilated improved pit latrine 4=Flush toilet 5= Hanging toilet/hanging latrine 5=Other Specify _____	2.3 a) Do you share this toilet facility with other households? 1=Yes [ ] 2= No [ ] b) If yes, how many households use this toilet facility? _____ households	2.4. If No, where do you go/use? (probe further) 1= Bush 2=Open field 3.=Near the river 4.=Behind the house 5.=Other specify _____	2.5 Do you have hand washing facilities? 1=Yes [ ] 2= No [ ]	2.6. On what occasion (s) do you wash your hands? Record ALL that applies See codes for 2.9 below (Multiple answers possible) Other (specify) _____	2.7 On what occasion (s) do you wash your hands? Record ALL that applies See codes for 2.7 below (Multiple answers possible) Other (specify) _____
	2.9. 1= After using the toilet; 2= After attending to a child who has defecated, 3= Before feeding a child (including before breastfeeding a child), 4=Before eating or preparing a meal; 5=After handling animals, 6= After traveling 7=others specify					

2.8 Do you do anything to raw foods such as fruits before feeding ? (tick appropriately do not read the options)

(01) Nothing [ ]

(02) Wash with water [ ]

(03) Wash with water and soap [ ]

2.10 How do you treat the left-overs from your last meal? (tick appropriately)

(01) Dispose [ ]

(02) Feed without warming [ ]

(03) after 2 hours, warm the food adequately and feed on it [ ]

(04) Kept for the next day's feeding

3 **HOUSEHOLD WATER CONSUMPTION**

3.1. What is your main current water source for household use? *(Probe for the Main source)*	3.2 How long does it take to go to	3.3 On avera ge, how many	3.4. How much do you pay for a 20lt jerrican (enter zero	3.5. What is your main source of drinking water? *(Probe for the Main source of drinking water)*	3.6 How do you store drinking water?	3.7. Do you do anything to the water before

1=River 2=Lake 3=Water tap 4=Borehole 5=Unprotected well 6=Protected well	7=Public tap 8=Water tanks 9=Dam 10= Irrigation channel 11=Springs 12=Other ____	the main source of water and come back (in minutes) To _____ At water point_____ From _____	LITRES (20 litre jerricans) of water does the household use per day?	if water is free). Enter in Kenya shillings 1.Per 20lt jerrican _____ 2. Per month_____ 3. Free_____	1=River 2=Lake 3=Water tap 4=Borehole 5=Unprotected well	6=Protected well 7=Public pan 8=Tanker 9=Dam 10= Cart with containers/drum 11=Other ____	1=No storage 2=Drums/ Jerricans 3=Tank 4=Any other specify	drinking it? 1=Boiling 2=Use traditional herbs 3=Use chemicals 4=Filters/Sieves 5=Decant 6= Solar disinfection 7=Nothing 7.8 How often do you treat the water? 1=Always 2= Sometimes 3= Don't know
	Total							

4. Socio-economic characteristics

4.1	What is the major material of the roof? |__| 1= Concrete 2= Tiles 3= Straw (grass, papyrus, banana fibres) 4= Wood	4.5	How many sleeping rooms/structures do you have?

	5= Plastic shelter 6= Galvanized iron 7= Mud 8= Tin can 9=Other, specify _____		Structures|__|__| Rooms |__|__|
4.2	What is the major material of the Walls? 1= Concrete 2= 3= Straw (grass, papyrus, banana fibres) 4= Wood 5= Plastic shelter |__| 6= Galvanized iron 7= Mud 8= Tin can 9= Cardboard 10= Bricks 11=Other, specify _____	4.6	How many people usually sleep in this dwelling/compound? People |__|__|
4.3	What is the major material of the floor? |__| 1= Concrete 2= Mud 3= Straw/bamboo 4= Wood 5= Vinyl 6= Tiles 7= Natural floor 8= Dung 9= Parquet 10= Carpet 11=Other, specify _____	4.7	What is the **MAIN** source of cooking fuel? |__| 1=Kerosene 2=Electricity 3=Firewood 4=Charcoal 5=Gas 6= Others Specify_____
4.4	What is the **MAIN source of lighting?** 1= Kerosene |__| 2= Electricity 3=Solar 4=Candle 5=Others Specify _____		

4.8	Does your household own any of following items?	Asset	Tick Box/number	Asset	Tick Box	Asset	Tick Box

		A clock or watch	___/___	Refrigerator		Sofa set	___/___
		Electricity	___/___	Solar panel		Gas cooker(any kind)	
		Radio	___/___	Paraffin Stove		Cupboard	
		Television	___/___	Pressure Lamp		Bank account	
		Mobile telephone	___/___	Beds	___/___	Roofing (any kind)	
		Pressing iron	___/___	Sponge mattress		Water Tank	
		Bicycle	___/___	Video cassette		Carpet	
		Car	___/___	Motor cycle		Land	

4.9	Does your household own this structure? 1=Yes 2=No If no, do you rent it, or do you live here without pay? 1=Pays rent/lease 2= No rent, with consent of owner 3= No rent, squatting	4.10 Does your household own the land on which the structure sits? 1=Owns 2= Pays rent/lease 3= No rent, with consent of owner 4=No rent, squatting	4.11	Source of income What were your sources of income the last three months (please indicate the three most important in order of priority) 1=sell of live stock, 2=sale of livestock product, 3= sale of fish 4=sale of ration food, 5=sale of own crop, 6=wage labour, 7=Remittance/kupewa 8=charcoal/firewood, 9=basket weaving, 10=petty trade, 11=Salary 12=Casual labour 13= No source of income 14= Others (specify)_____

	Ownership _____ rent _____			

5. Food sources and food production

5.1	What are the main sources of food? 1=Own production 2=Purchased from the market 3=Donations and gifts 4= Any other specify_____	5.2 Do you produce any food in your current location? Yes [ ] No [ ] 9.2.1 If yes, is the land legally or officially allocated? Yes [ ] No [ ] Where is the land located? 1=Within the yard 2= Open spaces in the area/neighbourhood 3=Open spaces outside the area	5.3. If no, how do you produce food? 1= Roof garden 2= No space technologies (sack, mound, others) 3= No cultivation by household 4= Any other specify_____	Indicate if they have the following animals: Codes: 1=<5, 2=5-10, 3= 10-20, 4=20-50 5=50-100 6=100- 200 7=>200		5.4. Have you received FOOD AID (general food distribution) in the last three (3) months? (Please circle) 1 = Yes 2 = No If, YES, how many times in the last 3 months? 1= Once 2= Two times 3= Three times
				Cattle		

				Rabbits		
				Goats		
				Sheep		
				Chicken		
				Donkeys		
	5.5 How many acres of land do your Household Cultivate? ____ acres (Estimate based on 4 gorogoro = 10kg that is usually used to cultivate 1 acre of land)					

APPENDIX C:24-Hour dietary recall

TICK THE DAY OF THE WEEK WHICH YOU ARE RECALLING **(IT SHOULD BE THE DAY BEFORE THE INTERVIEW) FOR THE CHILD**

Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday

Step 1: Please think back to when you woke up yesterday morning to the time you went to sleep in the evening. Now, I want you to try and remember what [Name] ate or drank yesterday from the moment you got up until you went to sleep again last night. Run through the whole day in your mind and try to remember everything that [Name] ate or drank. (THE INTERVIEWER MUST GIVE THE RESPONDENT A LITTLE TIME TO DO THIS). Now I would like to you tell me what [Name] ate and drank in the morning after [Name] got up. AFTER THE PARTICIPANT MENTIONS AN ITEM, THE INTERVIEWER SHOULD PROMPT THE RESPONDENT BY SAYING "AND THEN?"
ENTER THE INFORMATION IN COLUMN 1)

STEP 1: Food/drink eaten/drank during the day	**STEP 2:** Forgotten foods (PROMPTED)

STEP 2: NOW ASK THE FOLLOWING QUESTIONS ON FORGOTTEN FOODS AND ENTER THEM IN COLUMN 2.

Did (name) have any cold drinks or soda yesterday?

Did (name) have any sweets and or chocolate yesterday?

Did (name) have any cake and or cookies yesterday?

Did (name) have any snacks like chips, samosa, yesterday

Did (name) have any (other) fruit yesterday?

Did (name) have any (other) vegetable yesterday?

Did (name) have any bread and or rolls yesterday?

Did (name) have any mandazi yesterday?

Did (name) have anything else yesterday?

Q. What [NAME] ate/ drank yesterday; was it same as, more than or less than usual? (MARK **X** WHERE APPROPRIATE)

	Same as usual		More than usual		Less than usual

If more or less than usual, explain why (tick one)

1. **Celebration** □
2. **Religious activity** □
3. **Little food in the household** □
4. **Sickness** □

5. Other (specify) □ _____

Step 3: ***"Now I am going to ask you more about each food or drink that*** [Name] ***ate/drank yesterday"***.

START WITH THE 1ST ITEM REPORTED IN TABLE 1. TRANSFER THIS ITEM TO THE COLUMN 3 IN THE TABLE BELOW.

ASK "At what time was the item 1 eaten?"REPORT THE TIME IN COLUMN 1. DO NOT SPEND MUCH TIME IN GETTING THE EXACT HOUR.

ASK "for what meal was the item 1 eaten? INDICATE FOR WHAT MEAL ITEM 1 WAS EATEN AND REPORT IT IN COLUMN 2.'

Step 4 "Now I want you to tell me more about this food item…."

THIS INCLUDES A DESCRIPTION OF THE FOOD AS WELL AS THE PREPARATION. **ENTER THIS INFORMATION IN COLUMN 4).** *"Now I want you to tell me more about this food item……..*

THIS INCLUDES A DETAILED DESCRIPTION OF THE FOOD (BRAND NAME, IF UNPROCESSED, SEMI-PROCESSED OR FULLY PROCESSED SIZE, ETC), THE AMOUNT PREPARED AND THE METHOD OF PREPARATION. ENTER THIS INFORMATION IN COLUMN 4.

USE STANDARD HOUSEHOLD MEASURES AND WEIGHTS TO DETERMINE AMOUNTS OF INGREDIENTS USED. INDICATE IF FOOD WAS PURCHASED ALREADY COOKED FROM THE STREETS BY INCLUDING THE FOLLOWING TEXT "STREET FOOD"NEXT TO THE ITEM

Step 5. ***"Now we are going to find out how much of this item was eaten/drank"***

INTERVIEWER AND RESPONDENT USE HOUSEHOLD MEASURES AND WEIGHING EQUIPMENT TO DETERMINE HOUSEHOLD PORTION SIZES.

A DESCRIPTION OF HOUSEHOLD PORTION SIZES IN TERMS OF CUPS, SPOONS, BOWLS, GLASSES, MATCHBOXES, MANUAL PICTURES SIZE OR CENTIMETERS (USING RULER) IS THEN ENTERED IN COLUMN 5.

INFORMATION SHOULD BE OBTAINED FOR TOTAL AMOUNT OF COOKED FOOD AND AMOUNTS CONSUMED SHOULD BE ENTERED

IF THE FOOD CODE AND THE PORTION SIZE IN GRAMS OF THIS PARTICULAR ITEM IS EASY TO FIND, IT CAN BE **ENTERED COLUMN 6).** IF IT IS NOT CLEAR OR EASY, THE CODE AND GRAM WEIGHT CAN BE LEFT OUT TO BE COMPLETED AFTER THE INTERVIEW. THIS PROCESS IS REPEATED FOR EACH FOOD ITEM THAT WAS ENTERED ON FORM 1).

STEP 4: RECORDING SHEET FOR INFORMATION COLLECTED IN STEP 3 OF THE 24-HOUR RECALL

1	2	3	4		
Item carried from step 1 and 2	Time	Meal 1=breakfast 2=lunch 3=dinner 4=snack 5=other	description of ingredients (*Include* •*brand name,* • *if unprocessed, semi-processed and fully processed,* •*large, medium, small*	Detailed description of the portion size	*Weight (grams)*

APPENDIX D:7- Day Food Frequency Questionnaire

Food	Description	Times eaten		Source		
		Per day	Per week	purchase	production	Gifts/donations
Maize						
Rice						
Wheat						
White bread						
Brown bread						
Chapatti						
Other types						
chicken	boiled wt skin					
	Boiled without skin					
	Fried wt skin					
	Fried witout skin					
	Offals					
	Any other preparation method					
Beef	Roasted/ fried wt fat					
	Roasted/fried wtout fat					
	Any other					
Goat	Roasted/ fried wt fat					
	Roasted/fried wtout fat					
	Any other					
Offals	Kidney					
	Liver					
	Heart					
	Tripe					
Eggs	Pouched/ boiled					
	Fried in oil					
	Fried in fat					
Fish	Deep fried					
	Stewed					
Vegetables: cabbage, spinach,	Boiled					

traditional vegetables						
	Fried wt oil					
	Fried wt fat					
Fruits						
Legumes and pulses	Boiled					
	Fried					
Nuts	Roasted					
	Fried					
	Boiled					
Tubers/ roots And plantain	Boiled					
	Roasted					
	Any other method					

APPENDIX E: Patient Assessment Form

Identification number……date……

Identification number…… Age …… Sex

……..

Risk factors

Do you smoke?

Yes…….Never……....

Not in the last one year……..

If YES,

How many sticks per day?

Do you take alcohol?

Yes…… ….No……

Physical activity low…… (Walking slowly, household tasks)

Medium …… (Walking fast, cycling, gardening)

High…… (Chopping wood, swimming, jogging, skipping rope, cycling fast,)

Medical history

What medicines are you taking?

ARVS being taken

1

2

3

4

Other medicines

1

2

3

4

Reasons for the other medication

Are you on any micronutrient supplement?

1) Yes [ ] **2)** No [ ]

2) If yes specify…….

Nutritional status

Anthropometric, Biochemical And Dietary Assessment

last weight…….kgs (6 months)

Current Weight……kg

Weight change……...kg

Height …….m

BMI……Kgs/ m^2

Waist circumference…… cm

Hip circumference ……cm

Waist/hip ratio ……

Blood pressure……..mmHg

Glucose…….mg/L

Total Cholesterol ……mg/L

Triglycerides…… mg/L

Oxidized LDl-C…… mg/L

C reactive protein…….. mg/ L

Body composition

Lean body mass……..%

Fat..... %

Water .....%

APPENDIX F: Framingham point scores

Estimate of 10- year coronary heart disease risk for women: Framingham point scores

Age	Points
20-34	-7
35-39	-3
40-44	0
45-49	3
50-54	6
55-59	8
60-64	10
65-69	12
70-74	14
75-79	16

HDL	points
≥60	-1
50-59	0
40-49	1
<40	2

Total cholesterol	Points at age 20-39	Points at age 40-49	Points at age 50-59	Points at age 60-69	Points at age 70-79
< 160	0	0	0	0	0
160-199	4	3	2	1	1
200-239	8	6	4	2	1
240-279	11	8	5	3	2
≥ 280	13	10	7	4	2

	Points at age 20-39	**Points at age 40-49**	**Points at age 50-59**	**Points at age 60-69**	**Points at age 70-79**
Non smoker	0	0	0	0	0
Smoker	9	7	4	2	1

Systolic BP	**If untreated**	**If treated**
<120	0	0
120-129	1	3
130-139	2	4
140-159	3	5
≥ 160	4	6

Point total	**10-year risk (%)**	**Point total**	**10 year risk (10%)**
<9	< 1	19	8
9	1	20	11
10	1	21	14
11	1	22	17
12	1	23	22
13	2	24	27
14	2	≥ 25	≥ 30
15	3		
16	4		

17	5		
18	6		

(National, Heart ,Lung and Blood Institute: Detection, evaluation, and treatment of high blood cholesterol in adults. *(Adult Treatment panel 111). Final report. U.S. Department of Health and Human Services, National Institute of Health Publication No. 02- 5215, Bethsenda, Md, September 2002)*

Estimate of 10- year coronary heart disease risk for men: Framingham point scores

Age	points
20-34	-9
35-39	-4
40-44	0
45-49	3
50-54	6
55-59	8
60-64	10
65-69	11
70-74	12
75-79	13

HDL	Points
≥60	-1
50-59	0
40-49	1
<40	2

Total cholesterol	Points at age 20-39	Points at age 40-49	Points at age 50-59	Points at age 60-69	Points at age 70-79
< 160	0	0	0	0	0
160-199	4	3	2	1	0
200-239	7	5	3	1	0
240-279	9	6	4	2	1
≥ 280	11	8	5	3	1

	Points at age 20-39	Points at age 40-49	Points at age 50-59	Points at age 60-69	Points at age 70-79
Non smoker	0	0	0	0	0
Smoker	8	5	3	1	1

Systolic BP	If untreated	If treated

<120	0	0
120-129	0	1
130-139	1	2
140-159	1	2
≥ 160	2	3

Point total	**10-year risk (%)**	**Point total**	**10 year risk (10%)**
<0	< 1	11	8
0	1	12	10
1	1	13	12
2	1	14	16
3	1	15	20
5	2	16	25
6	2	≥ 17	≥ 30
7	3		
8	4		
9	5		
10	6		

APPENDIX G: CONSENT LETTER

This Informed Consent Form is for men and women who attend Nyeri comprehensive care clinic, and who we are inviting to participate in research on patients. The title of our research project is "impact of peanut supplementation on the cardiovascular disease biomarkers in adults with HIV dyslipideamia attending Nyeri Level 5 Hospital.

I am Regina Ph.D student at Kenyatta University. I am doing a research on reducing cardiovascular disease, which is very common in this country. I am going to give you information and invite you to be part of this research. You do not have to decide today whether or not you will participate in the research. Before you decide, you can talk to anyone you feel comfortable with about the research. There may be some words that you do not understand. Please ask me to stop as we go through the information and I will take time to explain. If you have questions later, you can ask me, the study doctor or the staff.

Cardiovascular diseases (CVD) is currently the second most frequent cause of death (after cancer) among HIV-positive subjects in areas of the world where Highly active anti retroviral therapy (HAART) is widely available. Dyslipidaemia is a major modifiable cardiovascular risk factor that is a common clinical feature of HIV-infected patients in the current era of HAART. The purpose of this study is to investigate the impact of peanut in reducing Cardiovascular disease risk factors in adults with HIV.

This research will involve blood tests, anthropometric measurements and peanut consumption as well as four follow-up visits to the clinic. We are inviting all adults with HIV who fit into the criteria and who attend Nyeri CCC to participate in the research. Your participation in this research is entirely voluntary. It is your choice whether to participate or not. Whether you choose to participate or not, all the services you receive at this clinic will continue and nothing will change. If you choose not to participate in this research project, you will be offered the treatment that is routinely offered in this clinic. You may change your mind later and stop participating even if you agreed earlier.

The peanut that you will be consuming will be sourced from, processed and packaged under sanitary conditions by Kenya Nut, Thika. They will test for aflatoxin before being process and certificate of analysis will be issued to ensure safety of the peanut. 80g of peanut will be given to every participant to take it as a snack. One group will start taking peanut with regular diet and then shift to diet low in fat while the other group will start by taking diet low in fat with peanut and then shift to regular diet.

The healthcare workers will be looking after you and the other participants very carefully during the study. If there is anything you are concerned about or that is bothering you about the research please talk to me or one of the other health worker in the CCC.

We will take blood from your arm using a syringe and needle. Each time we will take about 4 ml of blood. In total, we will take about 16ml of blood in 6 months. At the end of the research, 6 months, any left over blood sample will be destroyed.

During the research you will make four visits to the clinic.

- In the first visit, a small amount of blood, equal to about a teaspoon, will be taken from your arm with a syringe. This blood will be tested for the presence of substances that help us know the cardiovascular risk factor in your body. We will also ask you a few questions about your general health and measure how tall you are and how much you weigh. You will be given peanut packaged in small sachets to take daily for the 8 weeks
- At the next visit, which will be eight weeks later, you will again be asked some questions about your health and then you blood will be taken for further tests. Peanut will not be given for another 4 weeks.
- After one month you will come back to the clinic for a blood test, body measurements and then peanut for another 8 weeks
- At the end of the 8 weeks the last bloods tests and body measurements will be taken.

The research takes place over 20 weeks in total. During that time, it will be necessary for you to come to the clinic/hospital/health facility 4 days , for 3 hours each day.

In total, you will be asked to come 4 times to the clinic in 6 months. At the end of six months, the research will be finished.

With this research, something out of the ordinary is being done in your community. It is possible that if others in the community are aware that you are participating, they may ask you questions. We will not be sharing the identity of those participating in the research.

The information that we collect from this research project will be kept confidential. Information about you that will be collected during the research will be put away and no-one but the researchers will be able to see it. Any information about you will have a number on it instead of your name. Only the researchers will know what your number is and we will lock that information up with a lock and key. It will not be shared with or given to anyone except your clinician.

The knowledge that we get from doing this research will be shared with you through community meetings before it is made widely available to the public. Confidential information will not be shared. There will be small meetings in the community and these will be announced. After these meetings, we will publish the results in order that other interested people may learn from our research.

You do not have to take part in this research if you do not wish to do so and refusing to participate will not affect your treatment at this clinic in any way. You will still have all the benefits that you would otherwise have at this clinic. You may stop participating in the research

at any time that you wish without losing any of your rights as a patient here. Your treatment at this clinic will not be affected in any way.

If you have any questions you may ask them now or later, even after the study has started. If you wish to ask questions later, you may contact using any of the following:

Regina Wangui Kamuhu

Kenyatta University.

P.O BOX, 43844-00100, NRB

Tel no: 0717 655 404

Email: rkamuhu@yahoo.com

APPENDIX H: CERTIFICATE OF CONSENT

I ……. have on this day……..month……..year……..consented to participate in the study voluntarily. The investigator has thoroughly explained to me the purpose, objectives and duration of the study. I have understood that the study will be investigating the impact of peanut supplementation on the cardiovascular disease biomarkers in adults with HIV dyslipideamia attending Nyeri level 5 hospital. I also understand that 4mls of blood will be drawn four times during the the six months study period and that I will be interviewed several times and that I will be required to consume 80gm of peanut daily during the two treatment periods. I have been assured that confidentiality will be maintained and I have therefore consented to the use of my medical records for the purpose of the study.

Signature of the respondent……..Date……..

Signature of the investigator ……..Date……..

APPENDIX I: CERTIFICATE OF CONSENT (KISWAHILI)

Mimi ……katika hii siku……..mwezi……..mwaka…….. nimekubali kushiriki katika huu utaifiti kwa hiari yangu. Nimeelewa kuwa huu utafiti unahusu umuhimu wa matumizi ya njugu karanga katika kwa kupunguza madhara yanayotokana na maradhi ya moyo kwa watu watu ambao wana virusi vya HIV ambao wahashida ya wingi wa mafuta katika damu wanaotibiwa hapa hospitali ya Nyeri level 5. Nimeelewa pia kuwa damu kiasi cha mililita tano itatolewa mara nne katika mda wa huu uchunguzi na kuwa nutaulizwa maswali katika huu wakati. Tena nimeelezwa kuwa mambo nitakayoyasema yatewekwa kisiri na kwa hivyo nimekubalisha recordi zangu za hospitali zitumike katika uchunguzi huu.

Sahihi ya mshiriki…..……..Tarehe……..

Sahihi ya mtafiti……..Tarehe……..

APPENDIX J: RESEARCH AUTHORIZATION-NACOSTI

REPUBLIC OF KENYA

NATIONAL COUNCIL FOR SCIENCE AND TECHNOLOGY

Telephone: 254-020-2213471, 2241349, 254-020-2673550
Mobile: 0713 788 787 , 0735 404 245
Fax: 254-020-2213215
When replying please quote
secretary@ncst.go.ke

P.O. Box 30623-00100
NAIROBI-KENYA
Website: www.ncst.go.ke

Our Ref: NCST/RCD/12A/013/4

Date: 23rd January, 2013

Regina Wangui Kamuhu
Kenyatta University
P.O.Box 43844-00100
Nairobi.

RE: RESEARCH AUTHORIZATION

Following your application dated *10th January, 2013* for authority to carry out research on "***Impact of peanut supplementation on the cardiovascular disease markers in adults with HIV Dyslipideamia attending Nyeri Level 5 Hospital, Kenya,***" I am pleased to inform you that you have been authorized to undertake research in **Nyeri Municipality** for a period ending **31st December, *2013.***

You are advised to report to **the District Commissioner, the District Education Officer and the District Medical Officer of Health, Nyeri District** before embarking on the research project.

On completion of the research, you are expected to submit **two hard copies and one soft copy in pdf** of the research report/thesis to our office.

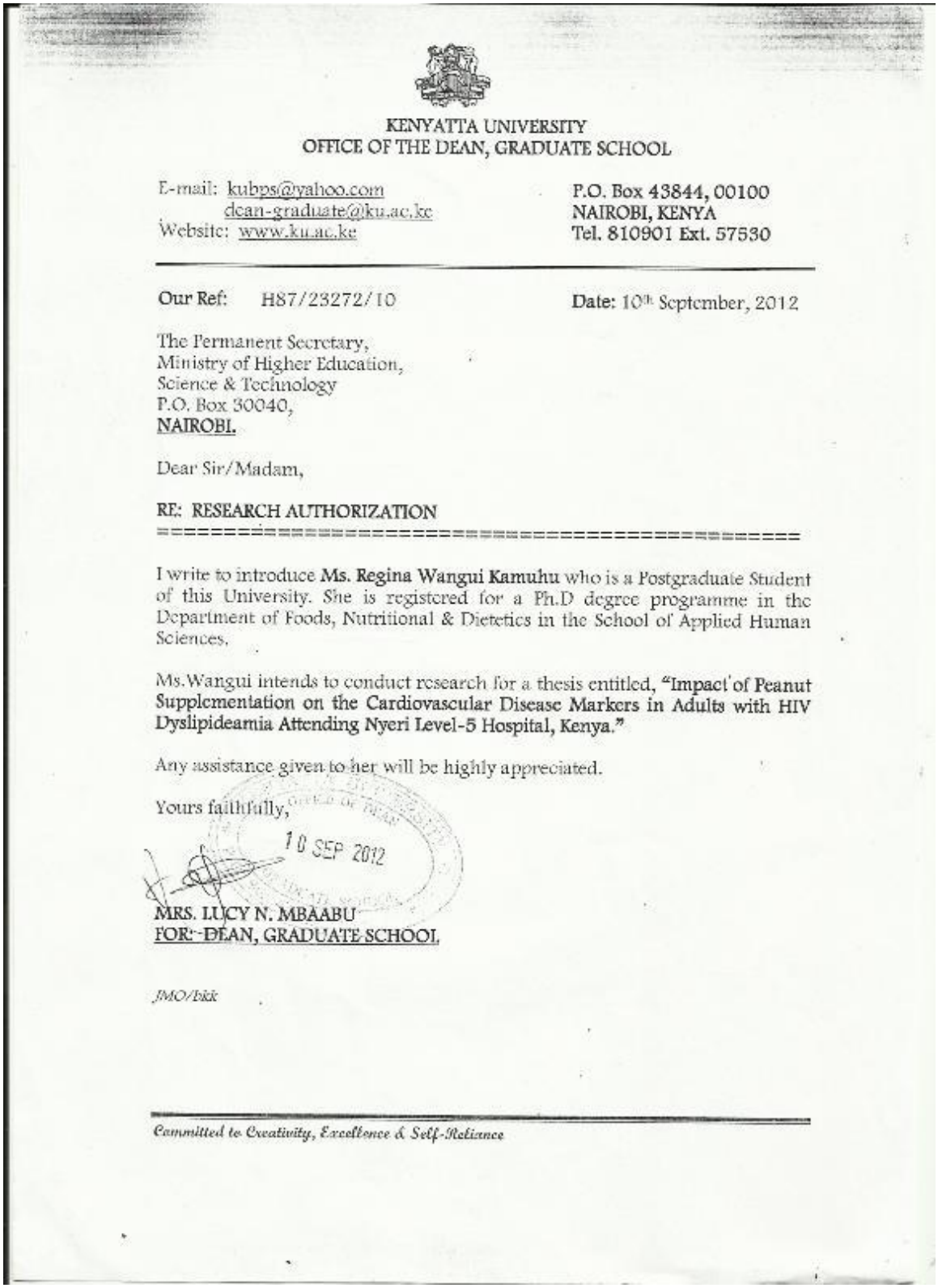
DR M.K. RUGUTT, PhD, HSC.
DEPUTY COUNCIL SECRETARY

Copy to:

The District Commissioner
The District Education Officer
The District Medical Officer of Health
Nyeri District.

"The National Council for Science and Technology is Committed to the Promotion of Science and Technology for National Development".

APPENDIX K: RESEARCH AUTHORIZATION-GRADUATE SCHOOL



KENYATTA UNIVERSITY
OFFICE OF THE DEAN, GRADUATE SCHOOL

E-mail: kubps@yahoo.com
dean-graduate@ku.ac.ke
Website: www.ku.ac.ke

P.O. Box 43844, 00100
NAIROBI, KENYA
Tel. 810901 Ext. 57530

Our Ref: H87/23272/10

Date: 10th September, 2012

The Permanent Secretary,
Ministry of Higher Education,
Science & Technology
P.O. Box 30040,
NAIROBI.

Dear Sir/Madam,

RE: RESEARCH AUTHORIZATION

I write to introduce **Ms. Regina Wangui Kamuhu** who is a Postgraduate Student of this University. She is registered for a Ph.D degree programme in the Department of Foods, Nutritional & Dietetics in the School of Applied Human Sciences.

Ms.Wangui intends to conduct research for a thesis entitled, **"Impact of Peanut Supplementation on the Cardiovascular Disease Markers in Adults with HIV Dyslipideamia Attending Nyeri Level-5 Hospital, Kenya."**

Any assistance given to her will be highly appreciated.

Yours faithfully,

10 SEP 2012

MRS. LUCY N. MBAABU
FOR: DEAN, GRADUATE SCHOOL

JMO/bkk

Committed to Creativity, Excellence & Self-Reliance

APPENDIX L: ETHICAL CLEARANCE

KENYATTA UNIVERSITY
ETHICS REVIEW COMMITTEE

Fax: 8711242/8711575
Email: kuerc.chairman@ku.ac.ke
kuerc.secretary@ku.ac.ke
Website: www.ku.ac.ke

P. O. Box 43844
Nairobi, 00100
Tel: 8710901/12

Our Ref: KU/R/COMM/51/273

Date: 23rd December, 2013

Regina Wangui Kamuhu
Dept of Foods, Nutrition and Dietetics
Kenyatta University
P. O. Box 43844, Nairobi.

Dear Ms. Kamuhu,

RENEWAL OF APPROVAL FOR PKU/071/1 63 of 2012 – "IMPACT OF PEANUT SUPPLEMENTATION ON THE CARDIOVASCULAR DISEASE MARKERS IN ADULTS WITH HIV DYSLIPIDEAMIA ATTENDING NYERI LEVEL 5 HOSPITAL, KENYA"

1. IDENTIFICATION OF PROTOCOL

The application for renewal of ethical approval of proposal is with a research topic, "Impact of Peanut supplementation on the cardiovascular Disease markers in adults with HIV Dyslipideamia attending Nyeri Level 5 Hospital, Kenya" dated 16th December, 2013.

2. DECISION

Kenyatta University Ethics Review Committee has **RENEWED THE APPROVAL** and that the research may proceed for a period of another ONE YEAR from 23rd December, 2013 to end on 22nd December, 2014.

3. ADVICE/CONDITIONS

i. Progress reports are submitted to the Kenyatta University Ethics Review Committee (KU-ERC) every six months and a full report is submitted at the end of the study.

ii. Serious and unexpected adverse events related to the conduct of the study are reported to this board immediately they occur.

iii. Notify the KU-ERC of any amendments to the protocol.

When replying, kindly quote the application number above.

KENYATTA UNIVERSITY OFFICE OF THE CHAIRMAN 23 DEC 2013 ETHICS REVIEW COMMITTEE P.O. BOX 43844 - 00100 NAIROBI

for PROF. NICHOLAS K. GIKONYO
CHAIRMAN: KENYATTA UNIVERSITY ETHICS REVIEW COMMITTEE

cc. Vice-Chancellor
Director: Institute for Research Science and Technology