COMMUNITY-BASED LIFESTYLE INTERVENTION FOR THE MANAGEMENT AND CONTROL OF METABOLIC SYNDROME AMONG ADULTS ATTENDING ST. MARY'S MISSION HOSPITAL, NAIROBI, KENYA.

OKUBATSION TEKESTE OKUBE

REG.No: H80/52924/2018

A Thesis Submitted in fulfillment of the requirements for the degree of Doctor of Philosophy in Nursing (Community Health Nursing) of the University of Nairobi

May, 2022

DECLARATION

I, Okubatsion Tekeste Okube, declare that this research work is my original and has not, to the best of my knowledge, been submitted for examination or award of degree in this or any other institution of higher learning. Other people's work used in this research has been properly acknowledged and referenced.

Okubatsion Tekeste Okube,

School of Nursing Sciences,

University of Nairobi.

Signature: ________________

Date: 04/05/2022

CERTIFICATE OF APPROVAL

CERTIFICATE OF APPROVAL

We confirm that this thesis titled **"community-based lifestyle intervention for the management and control of metabolic syndrome among adults attending St. Mary's Mission Hospital, Nairobi, Kenya"** has been done by Okubatsion Tekeste Okube under our supervision as the university supervisors.

1. Dr. Samuel T. Kimani, PhD.
   School of Nursing Sciences,
   University of Nairobi,
   Signature:________________
   Date: 04/05/2022

2. Dr. Waithira Mirie, PhD.
   School of Nursing Sciences,
   University of Nairobi,
   Signature:________________
   Date: 04/05/2022

DEDICATION

I humbly dedicate this research work to the Almighty God for giving me the strength, resilience and willingness to finalise my study. With great pride I also dedicate this research work to my wife Mrs. Selam Feshatsion, my parents, Mr. Tekeste Okube and Mrs. Letenguse Tekie, all my siblings and my lovely children; John and Aminadab who provided me with unending love and support throughout this study; you bestowed upon me so many gifts, including a love for learning.

ACKNOWLEDGMENTS

First of all, I would like to thank to the Almighty God, the most gracious and the most merciful, by whose power, I have been able to complete this research work.

My profound gratitude goes to my wife, my parents, my siblings and my cousins for the support they have offered me, without their great contribution to my life and education, I would have not made it this far, I am forever indebted to them.

I would like to thank to my supervisors; Dr. Samuel T. Kimani and Dr. Waithira Mirie for their guidance and encouragement. Their critical eyes and watchful guidance were greatly appreciated.

LIST OF TABLES

LIST OF FIGURES

TABLE OF CONTENTS

LIST OF ABBREVIATIONS

ADA:	American Diabetes Association
AACE:	American Association of Clinical Endocrinologists
ATP:	Adult Treatment Pane
BMI:	Body Mass Index
BP:	Blood pressure
BGL:	Blood glucose level
CI:	Confidence Interval
CVD:	Cardiovascular disease
FBG:	Fasting blood glucose
IDF:	International Diabetes federation
NCDs:	Non-communicable diseases
NAFLD:	Nonalcoholic fatty liver disease
NHSSP:	National Health Sector Strategic Plan
DM:	Diabetes Mellitus
GOK:	Government of Kenya
HC:	Hip circumference
HTN:	Hypertension
KNH:	Kenyatta National Hospital
MoH:	Ministry of Health
MetS	Metabolic Syndrome
NCDs:	Non-communicable diseases
NCEP:	National Cholesterol Education Program

OR:	Odds ratio
PCOS:	Polycystic ovary syndrome
RCT:	Randomized controlled trial
SD:	Standard deviation
SMMH:	St. Mary's Missio Hospital
SPSS:	Statistical Package for Social Sciences
STEPS	Stepwise approach to surveillance on non communicable diseases
UON:	University of Nairobi
WC:	Waist Circumference
WHF:	World heart federation
WHO:	World Health Organization
WHR:	Waist -to- Hip Ratio
WHtR:	Waist –to-Height Ration
HDL-C	High-Density-Lipoprotein-Cholesterol
TGs:	Triglycerides

OPERATIONAL DEFINITIONS

Anthropometrics: Physical measurements including body weight, height, body mass index, waist circumference and hip circumference.

Biomarkers: Blood cholesterol including triglycerides and high-density lipoprotein cholesterol as well as fasting blood glucose.

Community-based approach: An approach in which interventions are implemented at the community level with the aim of controlling or preventing diseases.

Community health workers (CHWs): Members of the community who work either for pay or as volunteers in association with the local health care system.

Dietary patterns: The quantity, variety, or combination of different foods in a diet and the frequency with which they are habitually consumed.

Decisional-balance: The balance between the perceived advantages of adopting a new behavior (the pros), and the perceived disadvantages or barriers to adopting a new behavior (the cons).

Health behaviors: Behaviors that a person engages in to prevent a disease or health problem occurring or to prevent an existing disease or health problem from getting worse.

Hyperglycaemia: A raised level of glucose in the blood. It occurs when the body does not have enough insulin or cannot use the insulin it does have to turn glucose into energy.

Lifestyle interventions: Interventions that include exercise, diet, reducing or avoiding alcohol intake and smoking cessation.

Metabolic syndrome: a cluster of conditions characterized by increased blood pressure, high blood sugar, abdominal obesity and abnormal cholesterol levels that occur together, increasing risk of heart disease, stroke and type-2diabetes.

Nephropathy: Damage or dysfunction of the kidney, which can cause the kidneys to be less efficient or to fail altogether.

Retinopathy: A disease of the retina of the eye which may cause visual impairment and blindness.

Neuropathy: Damage or dysfunction of the peripheral nerves, which can cause numbness or weakness.

Risk factors: Risk factors are conditions that increase chances of developing a disease.

Self-efficacy: A person's belief about his or her ability and capacity to accomplish a task.

Sedentary lifestyle: a type of lifestyle where an individual does not receive regular amounts of **physical activity.**

Type-2 diabetes: A condition when the body cannot effectively use insulin.

ABSTRACT

Background: Metabolic Syndrome (MetS) is a major risk factor for cardiovascular disease and type-2 diabetes. Implementation of interventions targeting risk factors associated with lifestyle could significantly control MetS and reduce the burden of cardiovascular diseases. However, the effectiveness of such interventions implemented through a community-based approach has not been reported in Kenya. **Objective:** Determine the effect of a 15-month community-based lifestyle intervention in adults with metabolic syndrome attending St. Mary's hospital, Nairobi, Kenya.

Methods: A two-arm randomized controlled trial involving 352 adults with MetS was conducted for 15-months. The participants were recruited from the outpatient clinic of St. Mary's Mission Hospital in Nairobi and randomly assigned into control and intervention groups equally. MetS was difined using the International Diabetes Federation diagnostic criteria. The intervention group was exposed to comprehensive lifestyle intervention that entailed detailed verbal and written recommendations focused on the main modifiable cardiovascular risk factors. While the control group received conventional lifestyle advice, which is the routine care provided in the hospital by health care providers according to the usual clinical practice. A questionnaire adopted from the WHO STEPS was used to collect the lifestyle characteristics of the participants. Knowledge of risks and preventive measures of CVDs and behavioural patterns were measured at baseline and end-line. The trans-theoretical model stages of change towards a healthy lifestyle were assessed before and after the intervention. Physical, clinical and biochemical markers were measured at baseline, midline, and end-line. Analysis with chi-square test and binary logistic regression for categorical and independent t-test and paired t-test for continuous variables were employed.

Results: The anthropometric/clinical/biochemical, lifestyle characteristic, knowledge of CVDs were similar across the two groups at baseline. The consumption of recommended dietary intake patterns and physical activity significantly improved in the intervention compared to the control group at the end-line. There was a significant ($p < 0.001$) decline in the proportion of MetS in the intervention (45.5%) relative to the control group (15.9%) at the endline. There was also a significant ($p < 0.05$) improvement in all the components of MetS in the intervention relative to control group at the endline. The level of knowledge of the major risk factors and preventive measures of CVDs significantly ($p < 0.001$) improved in the intervention relative to the control group at the end-line. Additionally, most participants in the intervention group proceeded to the maintenance stage of lifestyle change as per the TTM-based health education intervention.

Conclusions: A community-based lifestyle intervention showed effectiveness in modifying lifestyle goals that resulted in improved metabolic outcomes. One in three adults with MetS exposed to a community-based lifestyle intervention experienced improvement - an indication of the efficacy of the model. The findings have major implications for CVDs prevention through early indentification and management of cardiometabolic abnormalities. Integration of community approach into the health system can greatly improve early identification of those at risk for timely intervention, better outcome and protection against CVDs. The approach could leverage on nurses and CHWs who are the main human resource for health at the primary level health facilities to promote community wellbeing through universal health care model.

CHAPTER ONE: INTRODUCTION

1.1. Background

Metabolic Syndrome (MetS) is a constellation of multiple cardiovascular diseases (CVDs) risk factors including central obesity, high levels of triglycerides (TGs), fasting blood glucose (FBG), blood pressure (BP), and reduced level of high-density lipoprotein cholesterol (HDL-C) (Grundy, 2008). In this study, the International Diabetes Federation (IDF) diagnostic criteria (Alberti *et al.*, 2009) was used to determine MetS. Accordingly, an adult person is deemed to have MetS if he/she has central obesity (waist circumference ≥94 cm for men and ≥80 cm for women) plus at least 2 of the following elements; i) raised TGs level ≥1.7 mmol/L or history of specific treatment for the lipid abnormality, ii) reduced HDL-C <1.03 mmol/L in males and <1.29 mmol/L in females or history of specific treatment for the lipid abnormality, iii) elevated BP: systolic BP ≥130 mm Hg or diastolic BP ≥85 mm Hg or on treatment for previously diagnosed hypertension, iv) raised FBG level of ≥5.6 mmol/L or previously diagnosed with type-2 diabetes.

According to the IDF criteria, the global prevalence of MetS is approximately 25% (IDF, 2015). Using the IDF criteria, the prevalence of MetS in the Sub-Saharan African (SSA) region among the general population was reported at 25.1% in men and 35.4% in women. Among diabetic patients in SSA region, the prevalence of MetS was 80% (Clara *et al.*, 2008). As per the IDF diagnostic criteria, in Kenya, reports have given variable estimates in the prevalence of MetS including 25.6% (Geoffrey *et al.*, 2017) and 34.6% (Lydia *et al.*, 2012). The emergent of a global epidemic for CVDs, notably hypertension and type-2 diabetes points to the need to understand their premorbid states such as MetS. MetS is a known precursor of CVDs which substantially increases risks of morbidity and mortality (Cantiello *et al.*, 2015, Mendonca *et al.*, 2015).

Individuals with MetS are at increased risk of developing coronary heart disease, stroke, and diabetes. Indeed, those with MetS are at about a five-fold increased risk of acquiring type-2 diabetes, three times more likely to experience a heart attack, and two times more likely to die from it relative to people with no MetS (IDF, 2015). For example; in 2012, approximately, one-third (31%) of all global death was attributed to CVDs. Over 75% of deaths caused by CVD occur in the low- and middle-income countries (LMICs) (Giovanna *et al.*, 2017), mostly the SSA region that accounts for much of the global CVD-related deaths (Mensah *et al.,* 2015). In Kenya, it is estimated that NCDs are attributed to half (50%) of all adult admissions in hospitals and 55% of all deaths, with the chief causes being CVDs (WHO, 2012-a). Certainly, CVD-related deaths are expected to increase in SSA countries due to epidemiologic and nutritional transitions in these regions (Mensah et al., 2015, Roth *et al.*, 2015). Moreover, CVDs are significantly contributing to poverty because of huge financial burden associated with medical-surgical treatments. This is specifically heavy in developing countries associated with the use of out-of-pocket, because of lack of affordable and effective insurance. Additionally, the burden of CVDs has a long-term public health impact as it undermines healthcare systems.

The development of MetS is associated with a sedentary lifestyle and behavioural factors including adopting unhealthy dietary patterns, alcohol misuse, cigarette smoking, and lack of physical activity (Mohamed *et al.,* 2016, Mohamed, 2014, Yamaoka and Tango, 2012, Popkin *et al.,* 2012). These factors are common in LMICs dwellers (Giovanna *et al.,* 2017), particularly in the urban environments, predisposing people to MetS and CVDs (Assah *et al.,* 2011, Mensah *et al.,* 2015, Doulougou *et al.*, 2014). Moreover, people in low socio-economic level are at increased risks of MetS and subsequently CVDs because they are more likely to adopt unhealthy lifestyles (unhealthy diet, alcohol, smoking), psycho-social-related stresses, and lack of access to quality healthcare amenities (Van de Vijver *et al.,* 2015).

Compared to the general population, the informal settlements, the target of this study, are disproportionately exposed to risks of CVDs (Sliwa *et al.,* 2016). For example, research conducted among the informal settlements in Nairobi, showed high rates of the major CVDs risk factors such as lack of physical activity, cigarette smoking, and intake of unhealthy diet and excessive alcohol (Haregu et al., 2015, Oti *et al.,* 2013). Hence, informal settlements disproportionately suffer from the main CVD risk factors that include obesity, lipid abnormalities, high BP, and diabetes (Yusuf *et al.,* 2004), all of which are features of MetS. A study carried out in Nairobi's slums reported high rates of central obesity, alcohol intake and smoking, and low vegetables and fruits intake (Hulzebosch *et al.,* 2015). Moreover, across Kenya, community awareness involving CVDs is low.

Most people living with diabetes (WHO, 2014-a) and hypertension (Shukri et al., 2018) are diagnosed late when complications have already set in (WHO, 2014-a).

Considering the heavy burden of CVDs, there is a pressing need for developing countries to implement population-based cost-effective preventive interventions for CVDs. Lifestyle modification intervention focusing on diet and physical activity has been established to be beneficial effect on metabolic and clinical outcomes among type-2 diabetes patients (Mohamed *et al.*, 2016, Mohamed, 2014, Makrilakis *et al.*, 2012). However, achieving lifestyle modification including engaging in adequate physical activity and adopting a healthy diet is quite a challenging issue to prevent and manage CVDs, thereby limiting their impact. This might be due to lack of awareness of people on the recommended lifestyle changes necessary to prevent or manage their health issues. Moreover, people in LMICs do not go for routine screening of diseases; instead, they visit health facilities after they have developed disorders, usually at a late stage, which is impossible to prevent complications of the disease. Further, healthcare professionals in hospitals are busy dealing with acute conditions and thus may not have enough time to provide behavioural modification interventions at an early stage of disease (Pronk and Remington, 2015).

A significant proportion of CVDs-related morbidity and mortality could be prevented through population-based approaches (Boateng *et al.*, 2017). An opportunity to mitigate the impact of CVDs is to address the incidence by focusing on high-risk groups such as those with MetS. Early identification of such individuals can allow for the establishment of cost-effective lifestyle interventions thus mitigating complications and the cost of medical-surgical treatments. Community-based approaches can be promising strategy in addressing the burden of CVDs at the community level through awareness creation and sensitization of the community members about a healthy lifestyle. Community-based lifestyle interventions are interventions that are implemented in the community setting and continuously practiced by the communities in their natural environment to prevent or delay the development of chronic diseases like hypertension and type-2 diabetes (McLaren *et al.*, 2007). A community-based intervention for CVDs prevention and control involves primary and secondary prevention programmes that attempted to reduce the population burden of CVDs by modifying at least one cardiovascular risk factor (ie. blood pressure, smoking, total blood cholesterol, physical activity, diet) (WHO, 2002).

Community-based interventions for primary prevention of CVD offers the opportunity to target those individuals who are likely to develop CVD and those most likely to benefit from prevention and treatment efforts and, thus, could play a role in preventing and control CVDs (Miyares and Davis, 2014). A good example of this group is those individuals diagnosed with MetS. Community-based interventions for secondary prevention of CVD involves identifying, treating and rehabilitating patients with established CVD to reduce their risk of recurrence, to decrease their need for interventional procedures, to improve their quality of life and to extend their overall survival (WHO, 2011-a). According to the number of risk factors to be targeted, community-based programmes can be classified as single cardiovascular risk-management versus comprehensive cardiovascular risk-management. Single cardiovascular risk management approaches address one risk factor of CVD such as high BP control, cholesterol reduction, changes in nutrition, community-based smoking cessation (WHO, 2007). Although single cardiovascular risk management approach can be effective, to achieve the greatest benefits, a comprehensive, community-based intervention approach is required (Miyares and Davis, 2014). A comprehensive, community-based approach for CVD prevention is important because two or more cardiovascular risk factors clustering in one person is very common particularly in obese people, and may act synergistically increasing the risk more than any one single factor acting alone (WHO, 2011-a).

Community-based lifestyle interventions that focus on changing behaviours of people are considered as important and long-lasting interventions to reduce the risk of chronic diseases (Dunkley *et al.,* 2014). Community-based of CVD prevention approaches should target main cardiovascular risk factors, especially four behavioural risk factors (tobacco, unhealthy diet, physical inactivity, alcohol) using comprehensive risk-management strategies (Kolli and Dorairaj, 2007). Such interventions could be appealing, cost-effective, and accessible since they can reach people in their home environment without any financial and social inequalities. Furthermore, a community-based approach to CVD prevention is generalizable, cost-effective, and has the potential for modifying the environment and influencing health policies. We hypothesized that lifestyle modification intervention including changes in diet, alcohol intake, smoking, and physical activity has a role in reducing features of Mets in subjects with MetS. Thus, the study aimed to determine the effectiveness of a community-based lifestyle intervention on MetS among Kenyan adults with MetS.

To achieve the objective of the study and test the hypothesis, we applied the Trans-theoretical Model (TTM). The health education intervention provided to the intervention group was based on the TTM-stage of change. The model has been an instrumental tool in guiding population-based interventions to behaviour change to reduce the seriousness and prevalence of health problems. It describes the method of how individuals try to change behavioural risk factors. It has been successfully applied in several behavioural modifications such as changes in diet, physical activity, smoking, and weight reduction. Further, the model describes why some people fail and others succeed in modifying their behavior. Self-efficacy, stages of change, processes of change, pros and cons of decisional balance are the core constructs of the model. The validity and reliability of the model have also been proved (Fox and Kilvert, 2003). The model classifies the stages of change into five categories including pre-contemplation-where a person is not aware of his/her behaviour and has not considered changing a new behaviour. Contemplation is a stage where a person is aware of his/her behaviour and has considered change. The preparation stage is where a person is intending to modify his/her behaviour in the next month. Action is the stage where a person has started changing a new behaviour for less than six months. Maintenance is the last stage where a person has been practicing the new, beneficial behavior for more than six months (Glanz et al., 2008).

Therefore, the respondents' stages of behavioural change, decisional balance to a healthy lifestyle, and their confidence to adopt a new, healthy lifestyle were assessed at baseline and endline using the TTM. Decisional balance is the second construct of the model, which is described as the advantages (pros) and the disadvantage (cons) of changing behaviour from the person's perception (Koyun and Eroglu, 2014). For beneficial changes to occur, the pros must be much higher than the cons (Yasin *et al.,* 2011). The third construct is self-efficacy, which determines individuals' perceived confidence in performing a behavioural change successfully (Koyun and Eroglu, 2014).

1.2. Statement of the problem

The prevalence of MetS and CVDs is increasingly high in developing countries, where awareness and detection rates remain very low. The syndrome contributes to the spread of CVDs, type-2 diabetes, and other chronic disabilities. Previously, CVDs were considered diseases of the affluent people (Van de Vijver *et al.,* 2015). However, recent data indicates that CVDs now become more common among low-income people due to adoption of unhealthy lifestyles and psychosocial related stresses (Mensah et al., 2015). For example, researches conducted among the informal settlements of Nairobi, showed high rates of the major CVDs risk factors namely: lack of physical activity, cigarette smoking, and intake of unhealthy diet and excessive alcohol (Haregu et al., 2015, Oti *et al.,* 2013). Due to lack of awareness, many people in this class are diagnosed at late stages of the disease and die younger from CVDs and other NCDs (WHO, 2017-d).

Despite the heavy burden of CVDs in Kenya, community awareness around CVDs is low, most people living with diabetes (WHO, 2014-a) and hypertension (Shukri et al., 2018) are diagnosed very late when controlling the disease is impossible (WHO, 2014-a). Among individuals with hypertension, only 15.6% were aware of their elevated BP, of which, only 26.9% were on treatment (Shukri *et al.,* 2018). This is a huge gap in knowledge of CVDs which is an important barrier to effective prevention and early treatment. This indicates that the current management of CVDs is treatment oriented, focusing on individuals who have already developed hypertention and diabetes, depicting inadequate availability of population-based strategies for the prevention of CVDs (WHO, 2014-b). This evidence points to a neglect of awareness creation and diagnosis of premorbid conditions notably MetS relevant to prevention and mitigation of CVDs.

An opportunity to mitigate the impact of CVDs is to address the incidence by focusing on high-risk groups such as those with MetS. However, there is minimal information regarding community-based approaches as a strategy for addressing MetS. Most studies have focused mainly on small hospital-based studies in patients with type-2 diabetes and hypertension. No community-based controlled trials assessing the effects of lifestyle intervention on MetS have been documented. Therefore, the study was aimed to establish the effectiveness of a 12-month community-based lifestyle intervention on MetS in adults with MetS.

1.3. Research Questions

1.3.1 Main Research Question

What is the effect of community-based lifestyle intervention in adults with MetS attending St. Mary's Mission Hospital, Nairobi, Kenya?

1.3.2. Specific-Research Questions

i. What is the effect of a community-based lifestyle intervention on anthropometric measurements among adults with MetS attending SMMH in Nairobi?
ii. What is the effect of a community-based lifestyle intervention on blood pressure among adults with MetS?
iii. What is the effect of a community-based lifestyle intervention on fasting blood sugar level among adults with MetS?
iv. What is the effect of a community-based lifestyle intervention on blood lipid (TGs and HDL-C) levels among adults with MetS?
v. What is the effect of a community-based lifestyle intervention on MetS -related preventive practices among adults with MetS?
vi. What is the effect of a community-based lifestyle intervention on MetS control related knowledge among adults with MetS?
vii. What is the role of the Transtheoretical model as a behavioral changing tool on MetS control related lifestyle changes among adults with MetS?

1.4 Research objectives

1.4.1 Broad objective

To determine the effect of a community-based lifestyle intervention in adults with MetS attending SMMH, Nairobi, Kenya.

1.4.2 Specific Objectives

i. To determine the effect of a community-based lifestyle intervention on anthropometric measurements in adults with MetS attending SMMH, Nairobi, Kenya.

ii. To evaluate the effect of a community-based lifestyle intervention on blood pressure in adults with MetS.

iii. To determine the effect of a community-based lifestyle intervention on fasting blood sugar level in adults with MetS.

iv. To investigate the effect of a community-based lifestyle intervention on blood lipid (TGs and HDL-C) levels in adults with MetS.

v. To establish the effect of a community-based lifestyle intervention on MetS control related knowledge in adults with MetS.

vi. To determine the effect of a community-based lifestyle intervention on MetS-related preventive practices in adults with MetS.

viii. To determine the effect of application of the Transtheoretical model (TTM) as a behavioral changing tool on MetS control related lifestyle changes in adults with MetS attending SMMH, Nairobi, Kenya.

1.5 Hypothesis

- Participants enrolled in the community-based lifestyle intervention program will have favorable metabolic markers compared to those in the control group.

1.6 Justification of the study

The public health importance of MetS lays in its associated risk of CVDs, known for their significant morbidity and mortality. Individuals with MetS are at high risk of CVDs and should be prioritized for early lifestyle modification intervention. The mortality of CVDs is decreasing in developed countries as a result of promotion of population-based prevention measures. However, the burden of CVDs is increasing in LMICs. Because of lack of awareness, early detection and treatment of CVD do not occur in developing countries contributing to the high morbidity and mortality rates. For example, an estimated 75% of Kenyans who live with hypertension do not know they suffer from it, and of those who are aware, only 4% are able to control their BP (Kenya Ministry of Health, 2018). Indeed, poor public knowledge of CVDs is the reflection of unavailability of national programs for NCDs prevention. There is, therefore, a pressing need for LMICs to implement community-oriented affordable approaches for CVD prevention.

The current approach for the management of CVDs is treatment-oriented focusing on individuals who have already developed hypertension, and type-2 diabetes, usually with poor outcomes. This highlights the necessity for designing effective strategies to increase the public's awareness regarding preventive measures of CVDs.To reduce the impact of CVDs and achieve a better outcome, there is a need to shift intervention efforts from treatment-oriented to preventive approaches. Such preventive or proactive approaches require addressing the incidence of CVDs by focusing on high-risk groups such as those diagnosed with MetS. Early identification of individuals with MetS, can allow for the establishment of cost-effective lifestyle interventions, thus mitigating CVD-related complications. Furthermore, community-based approach to CVDs not only reduces CVD-related morbidity and mortality, but also curtails health care costs.

Implementation of lifestyle intervention through a community-based approach can be a promising strategy to control MetS and prevent CVDs in the community. Lifestyle intervention focusing on awareness creation strategy on specific lifestyle recommendations will inform the public to make healthier choices which can lead to better prevention and control of CVDs. Such interventions have a significant public health interest as they reach people in their natural environment. Certainly, people with adequate level of knowledge of CVDs are more likely to identify risks of the disease and adopt healthy lifestyles (Ng et al., 2014, Bergman et al., 2011). Lifestyle intervention involving changes in diet, alcohol intake, physical activity, and smoking is considered cost-effective approach to control MetS and CVD (WHO, 2011-a) as well as prevent or delay progression of pre-diabetes to type-2 diabetes (Muraki, 2013, Tabak *et al.*, 2013). Thus, the community-based lifestyle intervention may be an effective model to create and/or raise awareness of a healthy lifestyle and reduce the burden of CVDs in the community.

There is therefore a strong rationale for considering early detection of CVDs and the establishment of cost-effective lifestyle interventions through community-based approaches as CVDs risk reduction interventions. The study will contribute to the body of knowledge about the effectiveness of community-based lifestyle interventions in the management of MetS and CVDs. The findings will further inform policy development or policy review, and investments on the prevention approaches of CVDs.

1.7. Study variables

1.7.1. Independent variables

1. Participants’ socio-demographic and economic information.
2. Participants’ baseline lifestyle characteristics (diet, physical activity, alcohol and tobacco use).

1.7.2. Dependent variables

1. Change in prevalence of Metabolic Syndrome and its components
2. Changes in anthropometric measurements (Weight, BMI, WHR and WHtR).
3. Changes in lifestyle practices (dietary patterns, alcohol intake, smoking and physical activity).
4. Changes in participants’ level of knowledge on risk factors and preventive measures of CVDs
5. Changes in TTM-based stages of lifestyle change to control MetS.

1.7.3. Outcome variables

1. Reduction in CVDs risk factors
2. Improves patients’ quality of life

1.8 Conceptual frame work

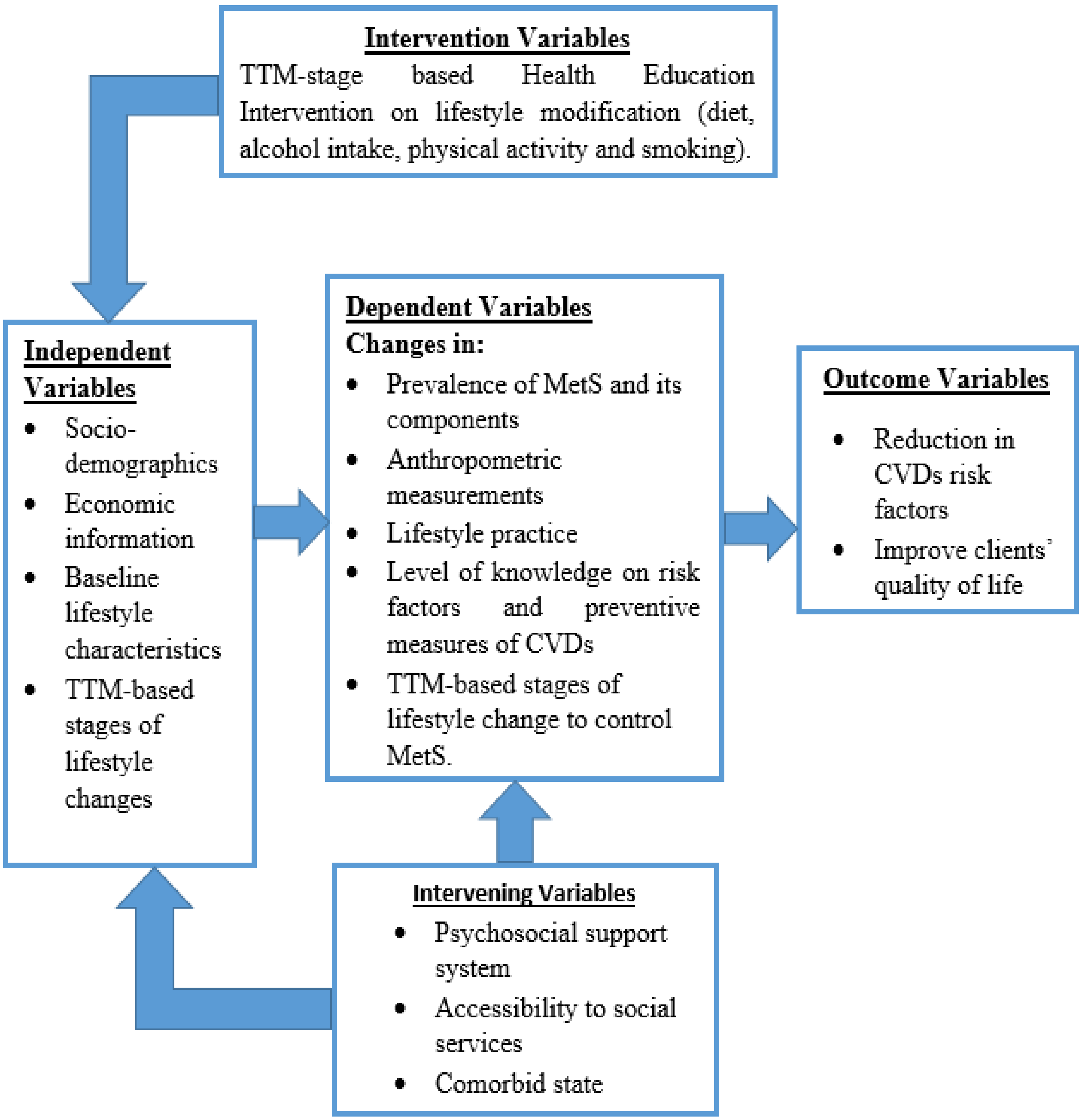
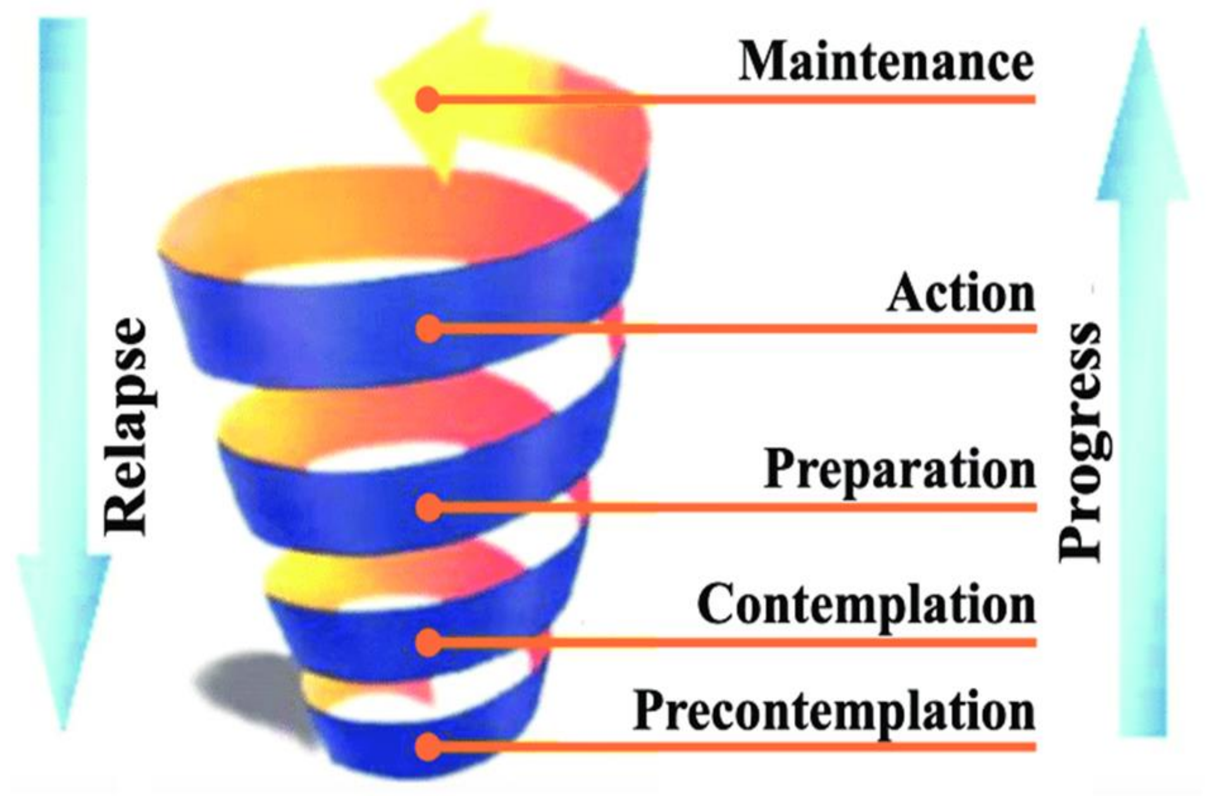


Figure 1: Conceptual framework on community–based lifestyle interventions to control metabolic syndrome.

1.9 Theoretical framework



Adapted from Prochaska and Di Clemente's cycle of change model (1988).

Figure 2: Theoretical framework: stages of the Trans-Theoretical Model

CHAPTER TWO: LITERATURE REVIEW

2.1. Introduction

MetS is a constellation of multiple risk factors namely central obesity, raised FBG, BP and dyslipidemia; that occur together, increasing the risk of CVDs events such as stroke, heart attack and diabetes (Cantiello *et al.,* 2015, Mendonca *et al.*, 2015). MetS has been defined differently by different health organizations as indicated below.

2.1.1 Definition of MetS by the World Health Organization (1999)

According to the WHO, MetS is described as having type-2 diabetes and at least two of the below listed cardiometabolic features: **i**) Hypertension: BP ≥140/90 mmHg, **ii**) High level of blood TGs (≥ 150 mg/Dl) and/or low HDL-C (<35 mg/dL in men, and <39 mg/dl in women), **iii**) Abdominal obesity: Waist/hip ratio >0.9 in men and >0.85 in women and/or BMI >30 kg/m^2, **iv**) Presence of albumin in urine with excretion rate of a minimum of 20 µgm/minute.

2.1.2 Definition of MetS by the European group for study of insulin resistance (EGIR)

According to the EGIR, MetS is defined as raised blood insulin level (>75^{th} percentile) and at least two of the following parameters: **i**) Cental obesity- a large waistline measurement ≥94 cm in men and ≥80 cm in women, **ii**) Raised TGs ≥1.7 mmol/l and/or low HDL-C <39 mg/dL for both sexes), **iii**) BP ≥140/90 mmHg or on treatment for it, **iv**) High serum glucose level, but no enough to be diagnosed with diabetes.

2.1.3 Definition of MetS by the National Cholesterol Education Program Adult Treatment Panel III (2002) (NCEP ATP III)

According to this criterion, a person has MetS, if any 3 or more of the following components are met: **i**) Central obesity- a large waistline: ≥102 cm in male and ≥88 cm in female, **ii**) Raised TGs ≥1.7 mmol/l, **iii**) Reduced HDL-C <1.03 mmol/L in males and <1.29 mmol/L in females, **iv**) Raised fasting blood glucose: >110 mg/dL, **v**) and High BP >130/85 mmHg.

2.1.4 Definition of MetS by the American Association of Clinical Endocrinologists (2003)

According to the American Association of Clinical Endocrinologists (AACE), the main components considered were obesity, high BP, reduced HDL-C and elevated TGs and blood glucose levels. According to the AACE, there is no specific criterial for the diagnosis of MetS, it was left for clinical judgment.

2.1.5 The International Diabetes Federation (2009) Global Consensus definition of MetS

After revising the above different criteria, the IDF framed a global agreement definition for MetS, considering gender and race-specific waistline measurement cutoffs. The IDF considers central obesity as a compulsory element of MetS due to its close association with CVDs. According to the new IDF criteria, for the Sub-Saharan African countries, MetS is defined as one having a large waistline circumference (≥94 cm for men and ≥80 cm women) plus any two of the following factors; i) raised TGs level ≥1.7 mmol/L (≥150 mg/dL) or on treatment for it, ii) low HDL-C <1.03 mmol/L (<40 mg/dL) in males and <1.29 mmol/L (<50mg/dL) in females or on treatment for it, iii) increased systolic BP ≥130 mmHg or diastolic BP ≥85 mmHg or on antihypertensive treatment, iv) increased FBG level ≥5.6 mmol/L (≥100mg/dL) or having type-2 diabetes. The current study, therefore, adopted the IDF diagnostic criteria for MetS definition using gender and race-specific waistline measurement cutoffs for the Sub-Saharan African countries (Alberti *et al.,* 2009).

2.2 Pathophysiology of Metabolic Syndrome

The main causes of MetS are believed to be resistant to insulin and central obesity (Grundy et al., 2005, Roberts *et al.,* 2013). Lack of insulin sensitivity and abdominal obesity cause several clinical-metabolic abnormalities including hypertension, hyperglycemia, and dyslipidemia- elements of MetS and major risk factors of CVDs and type-2 diabetes (Grundy et al., 2005, Sadikot and Hermans, 2010). Insulin resistance is defined as the declined receptiveness of peripheral tissues to insulin secreted by the pancreatic β-cells, contributing to high blood glucose level and subsequently type-2 diabetes (Deedwania, 2011). Lack or decline of responsiveness to insulin is the chief cause of type-2 diabetes (McCracken et al., 2018). Furthermore, IR causes type-2 diabetes by impairing insulin signaling pathways in the liver and decreasing glycogen synthesis and intracellular glucose transport in skeletal muscle (Ferris and Kahn, 2016).

2.3 Components of metabolic syndrome

The components of MetS are central obesity, elevated blood pressure (BP), fasting blood glucose (FBG), high levels of triglycerides (TGs), and reduced level of high-density lipoprotein cholesterol (HDL-C) (Grundy, 2008).

2.3.1 Anthropometric measurements

2. 3.1.1 Waist circumference (WC)

A large WC (≥94 cm for men and ≥80 cm for women) is defined as central obesity. Central obesity is the main component of MetS. The occurrence of MetS is closely linked to abdominal obesity. Hence, WC is a better parameter to determine visceral fat, the harmful internal fat which narrows blood vessels and coats the organs. For this reason, it is considered the best indicator and predictor of MetS, CVDs, and type-2 diabetes. It is a major risk factor for insulin insensitivity, inflammatory process, dyslipidemia, hypertension, type-2 diabetes, and MetS (Hall *et al.,* 2010).

2.3.1.2 Waist-to-Hip Ratio (WHR)

WHR is an important parameter to determine hidden fat in the abdomen. Measurement of waist and hip is obtained and the WHR is determined as the ratio of the waist to the hip measurement. A higher WHR implies a larger waist measure which reflects a high amount of abdominal fat, the dangerous fat. While a lower ratio means larger hip circumference which is good as it indicates a low amount of abdominal fat (WHO, 2011-b). Excess abdominal fat can hinder normal physiologic and metabolic functions of the body contributing to dyslipidemia, raised blood sugar, and increased risks of MetS, heart diseases, and type-2 diabetes. According to WHO, the cutoff point of WHR to determine cardiovascular risk is > 0.90 in men and > 0.85 in women (WHO, 2011-b). In Kenya, the WHO (2011-c) data indicated that 36% and 28% of women and men, respectively, had a higher Waist–hip ratio than recommended.

2.3.1.3 WHO anthropometric cut-off points and risk of metabolic complications

Table one below indicates the cut-off points and risk of metabolic complications (WHO, 2008). Overweight and obesity are known risk factors for MetS and CVDs. A BMI of 25-29.9 kg/m^2 is classified as overweight. While obesity is defined as BMI ≥30 kg/m^2. Globally, in 2016, 40% of women and 39% of men were overweight, of which 13% were obese (WHO, 2016-a). In Kenya, 39% and 18% of women and men, respectively were either overweight or obese (Stepwise Survey for NCDs Risk Factors, 2015), and the percentage was higher in women who reside in urban areas (43%), than rural residents (26%) (Kenya Ministry of Health, 2015).

Components of MetS including dyslipidemia (raised TGs and reduced HDL-C), type-2 diabetes, and hypertension are strongly associated with increased BMI. With excess weight, there is a high concentration of body fat which hinders insulin action resulting in type-2 diabetes. Each year, 2.8 million people die as a result of overweight and obesity (WHO, 2009-b). Unhealthy lifestyle practice is the major risk factor for obesity and its related CVDs. A study conducted in one of the informal settlements of Nairobi indicated a high rate of obesity linked to insufficient vegetables and fruits consumption (Hulzebosch *et al.*, 2015). Lack of awareness of healthy food choices is the main barrier to consume healthy diets in Kenya (Ministry of Health, Kenya, 2015).

Table 1.World Health Organization cut-off points and risk of metabolic complications

Body mass index kg/m^2)	**Obesity class**	**Disease Risk (relative to normal weight and waist line)**
BMI <18.5	Underweight	
BMI =18.5- 24.9	Normal	
BMI = 25-29.9 kg/m2	Overweight	Increased
BMI = 30.0–34.9 kg/m2	Obesity I	High
BMI = 35.0–39.9 kg/m2	Obesity II	Very high
BMI ≥ 40.0 kg/m2	Obesity III	Extremely high
Indicator	**Cut-off points**	**Risk of metabolic complication**
Waist circumference	>94 cm (M);>80 cm (W)	Increased
Waist circumference	>102cm (M);>88 cm (W)	Substantially increased
Waist-hip ratio	≥0.90 cm (M); ≥ 0.85 cm (W)	Substantially increased
M, men; W, women		

2.3.2 Elevated blood pressure

High BP is defined as systolic/diastolic BP ≥ 140/90 mmHg. A person is considered hypertensive if one or both readings are high. As a component of MetS, raised BP is considered when systolic is ≥130 mmHg or diastolic is ≥85 mmHg (Alberti *et al.*, 2009). High BP can be classified as primary (essential) or secondary**.** The exact cause of essential hypertension is not known. It results from several factors, including high blood plasma volume and stimulation of the renin-angiotensin system. It is also influenced by lifestyle factors as well as behaviours such as smoking. While**,** secondary hypertension has specific medical causes such as Cushing syndrome, aldosteronism, pheochromocytoma, and hyperparathyroidism (Troy *et al.,* 2016).

Globally, in 2015, approximately a quarter (24%) of men and one-fifth (20%) of women had hypertension (Zhou *et al.*, 2017). It was highest in the African Region, at 46% (Danaei *et al.*, 2014). The highest rate (54.1%) of hypertension was reported in Soweto, South Africa (Gomez and colleagues, 2017). In Kenya, about a quarter (23.8%) of the adult population is believed to have hypertension (Kenya stepwise survey for NCDs risk factors, 2015). Whereas, in the rural areas of Kenya, the rate of hypertension among adults was reported at 21.4% (Hendriks et al., 2012). Globally, high BP is considered the primary cause of CVDs and related deaths (Danaei et al., 2014, WHO, 2009-b). Worldwide, of the total of all annual deaths, 12.8% is directly related to raised BP (WHO, 2009-c). In Kenya, in 2010, NCDs were responsible for approximately half (45%) of all causes of death, and hypertension was found to be the primary contributor to this trend (Phillips *et al.,* 2014).

2.3.3. Elevated fasting blood glucose (FBG)

Raised FBG as component of MetS level is defined as fasting plasma glucose level of ≥5.6 mmol/L or previously diagnosed with type-2 diabetes (Alberti *et al.,* 2009).

2.3.3.1Trends, prevalence and health consequences of type-2 diabetes

Type-2 diabetes is one of the four major NCDs causing high morbidity and mortality globally. A person should be diagnosed with diabetes if he/she has a fasting blood glucose level ≥ 7.0 mmol/L (≥126 mg/ dl). Whereas, pre-diabetes is defined as fasting plasma glucose level of 6.1-6.9 mmol/L (110-125 mg/ dl). Worldwide, the prevalence of type-2 diabetes among adults was 4.7% in 1980 (WHO, 2016-a). While in 2015, its global prevalence was 8.8% and predicted to be 10.4% in 2040 (IDF, 2015). Type-2 diabetes is more common in low and middle-income countries, accounting for 75% in its prevalence (IDF, 2015). In the Africa region, the rate of diabetes is estimated to be 6% in urban and 2% in rural areas (Mbanya et al., 2010). In Kenya, 3.3% of the adult population is estimated to have type-2 diabetes (WHO, 2009-a, Kenya National Diabetes Strategy, 2010). Another study in Kenya revealed a prevalence of 10.5% in the 45–54-year age category (Richard *et al.,* 2013). However, the Kenya National Diabetes Strategy report (2010), showed that the majority of people with diabetes may be undiagnosed.

Diabetes is significantly associated with several life-threatening complications (WHO, 2016-a, Huang Y, 2016). Of the global NCDs-related deaths, most (63%) is caused by the four major NCDs (CVDs, type-2 diabetes, cancers, and chronic respiratory diseases) (WHO, 2010-c, Forouzanfar, 2016). In 2013, 382 million people died of type-2 diabetes (IDF, 2013). Additionally, the total cost of diabetes-related medical-surgical treatment is extremely huge, contributing to poverty and hindering the country's developmental agendas.

Certainly, the rising trend in type-2 diabetes is closely linked to globalization, urbanization, and adoption of western lifestyles such as consumption of jumble foods and sedentary lack of exercise (Narayan *et al.*, 2011). If there is no modification of lifestyles, the prevalence and its related morbidity and mortality are expected to increase. According to the WHO (2016-b) estimation, the occurrence and magnitude of type-2 diabetes will be greatly increased by 92% in low-income countries. However, adoption of a healthy lifestyle including limiting alcohol intake, engaging in regular physical activity, and taking healthy diets can substantially reduce complications and progression of pre-diabetes to type-2 diabetes (Muraki, 2013). Hence, identification of people with pre-diabetic such as those with MetS is an important strategy to contain it and therefore, prevent type-2 diabetes.

2.3.4 Atherogenic dyslipidemia (raised TGs and reduced HDL-C)

Both HDL-C and TGs are elements of MetS (Alberti *et al.,* 2009). HDL-C is beneficial cholesterol, as it carries excess lipid from several tissues to the liver, which clears from the body, and thus reduces heart disease. If the concentration of HDL-C in the blood is low, the harmful type of lipids remains in tissues causing heart diseases. As an element of MetS, HDL-C level <1.03 mmol/l (<40 mg/dl) in male and <1.29 mmol/l (<50mg/dl) in female is considered low. TGs level of greater or equal to 1.7 mmol/l (≥150 mg/dl) is defined as raised regardless of gender (Alberti *et al.*, 2009). Globally, the principal cause of CVD-related death in adults is atherosclerosis. If there is excess cholesterol in the blood, it is easily trapped in the walls of an artery, and then forms plaque. Once the plaque forms, it narrows the blood vessels and makes them hard or less flexible, causes atherosclerosis. For example, if the arteries that supply blood to the heart are blocked by plaque, the heart muscles are restricted from getting enough oxygen and nutrients. Then the person experiences a heart attack or death in severe cases. Hence, dyslipidemia is considered a primary causes of heart disease (Eyup *et al.*, 2018, Michael *et al.*, 2011).

Unhealthy lifestyles including high carbohydrates consumption, harmful use of alcohol, tobacco use, physical inactivity, and obesity are the main causes of raised TGs and low HDL-C. Evidence shows that the risk of heart attack is greatly increased when the level of TGs reaches 1.7 mmol/l or higher (Michael *et al.,* 2011). Increased body weight is recognized as the chief cause of raised TGs and low HDL levels. Hence, weight reduction by engaging inadequate physical activity, reducing alcohol intake, limiting simple sugars and sugar-sweetened drinks is the measure to be taken to reduce serum TGs and increase HDL-C levels as CVDs prevention strategies (Michael *et al.*, 2011).

2.4. Prevalence, health and economic consequences of metabolic syndrome

According to the IDF diagnostic criterion, approximately a quarter (25%) of the world's adult population suffers from Mets (O'Neill, 2015, Grundy, 2008). However, the occurrence of MetS is more common among obese adults than in the general population. For example, studies carried out among obese adults in Palestine (Basma *et al.*, 2018), Mexico (Salas *et al.*, 2014), and India (Vatakencherry and Saraswathy, 2019) found a high prevalence of MetS at 69.4%, 73.8%, and 76%, respectively. Furthermore, the prevalence of MetS in European countries ranges from 42.7% to 78.2% (Jana *et al.,* 2014).

Like the NCDs, MetS has disproportionately affected the Sub-Saharan Africa countries exerting a heavy burden on health and the economy. For example; studies conducted in South Africa (Maritza and Theo, 2017) and Egypt (Fathi *et al.*, 2014), reported a high prevalence of MetS at 46.3% and 42.1%, respectively. Furthermore, the rates of MetS among adults in Cameroon (Dandji et al., 2018), Nigerian (Sabir *et al.,* 2016), Ghana (Gyakobo *et al.,* 2012), and Morocco (Brini et al., 2014), has been reported at 39.0%, 35.1%, 35.9%, 35.7%, respectively. Whereas, in Kenya, the prevalence of MetS was found to be 34.6% (Lydia *et al.*, 2012). Studies in Nigeria (Oladapo *et al.*, 2013) and Kenya (Kaduka et al., 2012) showed an increased rate of the elements of MetS such as obesity, dyslipidemia, high BP, and type-2 diabetes. Mathenge *et al.* (2010), who carried out a cross-sectional survey in the communities of Nakuru (Kenya), reported a high prevalence of hypertension (52.6%), high cholesterol level (23.9%), obesity (14.5%), and diabetes (7.7%), all are components of MetS.

The health implication of MetS lays in its association with CVDs including type 2 diabetes. People who have Mets are at a higher risk of having CVDs including high BP and type-2 diabetes (O'Neill and O'Driscoll, 2015, Mendonça *et al.*, 2015). Indeed, individuals suffering from MetS are about a 5-fold elevated risk of acquiring diabetes, 3-times more likely to experience heart attacks, and 2-fold elevated risks of death than people free of the syndrome (IDF, 2015). In Kenya, half (50%) of all admissions to hospitals and most (55%) of all mortalities in adults are attributed to NCDs, the chief cause being CVDs (WHO, 2012-a). Moreover, the cost of medical-surgical treatment associated with CVDs is extremely heavy contributing to poverty in the household and society. Certainly, CVDs are substantially associated with poverty due to lack of affordable insurance and, thus use of out-of-pocket. If things remain the same, could result in hampering the achievement of national agendas mainly sustainable development goals and the pillars of Vision 2030.

2.3 MODIFIABLE RISK FACTORS OF METABOLIC SYNDROME

Like NCDs, the major risk factors of MetS are unhealthy diet, alcohol and tobacco use, and insufficient physical activity (Okafor, 2012). Likewise, in the African region, MetS is substantially associated with an unhealthy diet (Kimani et al., 2019) evidenced by intake of junk foods (Okafor, 2012, Popkin *et al.*, 2012), high quantity of carbohydrates (Edyta *et al.*, 2017), lack of variety and quality of foods (WHO, 2017-a, WHO, 2015, Hulzebosch *et al.*, 2015). Traditionally, 2-3 decades back, Kenyan communities used to consume much grains and vegetables as well as engage in physically demanding activities- a characteristic of a healthy lifestyle. However, nowadays, people have adopted a western lifestyle characterized by the consumption of processed foods and sedentary lifestyles especially in urban environments (WHO, 2014-a).

2.3.1. Association between dietary intake patterns and metabolic syndrome

A dietary pattern refers to the quantity, variety, and frequency of certain food intake. A dietary pattern can protect from or predispose a person to MetS and CVDs. MetS and its related CVDs are substantially associated with adopting unhealthy dietary patterns characterized by excessive intake of junk food, and sweetened drinks (Popkin *et al.*, 2012). Consumption of high-calorie diets such as processed sugar, salt, and saturated fats is a known cause of obesity. Evidence shows that a diet rich in carbohydrates, short of high-quality proteins, vegetables and fruits is a precursor for MetS. High-calorie diets contribute to lipid abnormalities, elevated BP, and serum glucose level – the main elements of MetS (James *et al.*, 2016).

Kenya is undergoing a rapid change in nutrition as evidenced by intake of unhealthy foods including refined carbohydrates, junk foods, sugar loaded-beverages, and low vegetables and fruits (Kimani *et al.*, 2019). Adopting unhealthy dietary patterns increased the occurrence of type-2 diabetes, CVDs, and cancers, which are closely related to obesity and significantly hinder the country's development (Ministry of Health, Kenya, 2015). A report from one of Nairobi's slums showed very low vegetables and fruits intake (Hulzebosch *et al.*, 2015). Whereas, a paper published by Kimani and colleagues (2019), showed that regular intake of vegetables and fruits by hypertensive patients had a beneficial effect on body weight, BP, and cholesterol levels- some of the components of MetS. This underscores the importance of adequate fruits and vegetable intake to control MetS and prevent CVDs.

2.3.1. 1 Relationship between carbohydrates and protein intake and metabolic syndrome

Evidence shows that a diet rich in carbohydrates, short of high-quality proteins, vegetables and fruits is a precursor for MetS. High glycemic load foods such as refined carbohydrates are recognized as a driving force for the development of MetS, diabetes, and CVD (Shastun et al., 2016) by increasing glucose and lipid dysregulation (Edyta *et al.*, 2017). Whereas, dietary fiber intake especially from whole grains and cereals (eg. wheat, barley, rice, maize, millet) has been proved to cut down excess weight and properly regulate blood sugar and lipid levels (Papathanasopoulos and Camilleri, 2010). Dietary protein intake has been found to reduce cardio-metabolic risk factors by reducing the concentration of TGs and fat mass while maintaining lean muscle (Leidy *et al.,* 2015). Moreover, intake of protein-rich meals increases metabolic rate and regulates appetite which may reduce body weight and risk of MetS (Leidy *et al.,* 2015, Keller, 2011).

2.3.1. 2 Association between sugar intake and metabolic syndrome

According to the WHO, the daily recommended sugar intake per person is less than 5 teaspoons to prevent NCDs, notably hypertension and diabetes (WHO, 2015). Excessive sugar intake causes obesity- the main precursor for insulin insensitivity, type-2 diabetes, dyslipidemia, and high BP (James *et al.,* 2016), which are all components of MetS. Particularly, added sugars and beverages rich in sugar are closely linked with central obesity (WHO, 2015, Bray and Popkin, 2014), dyslipidemia (Welsh *et al.*, 2011), type-2 diabetes (Kimber, 2016, DiNicolantonio et al., 2015), high BP (DiNicolantonio and Lucan, 2014), MetS (Bray and Popkin, 2014, Denova-Gutierrez *et al.,* 2010). Excessive sugar consumption causes carbohydrate and lipid metabolic dysregulation, resulting in dyslipidemia and insulin insensitivity. Moreover, high sugar intake promotes weight and fat gaining, resulting in carbohydrate and lipid metabolic dysregulation (Kimber, 2016). Both dyslipidemia and insulin insensitivity increase risks of high BP and blood glucose levels- elements of MetS. Moreover, Cox and colleagues (2011) reported, high sugar intake increases the risk of MetS by inducing inflammatory processes (DiNicolantonioa *et al.,* 2017).

2.3.1. 3 Relationship between salt intake and metabolic syndrome

According to the WHO dietary guidelines, the daily salt intake for an adult person is less than 1 teaspoon to prevent high BP and reduce the risk of heart disease (WHO, 2012-b). Dietary salt has a substantial effect on levels of BP and cardiovascular health status (WHO, 2010-d). Components of MetS including dyslipidemia, hypertension, and type-2 diabetes are directly associated with high salt intake (Baudrand *et al.*, 2014). The WHO data indicates that limiting salt intake reduces levels of BP and CVDs (WHO, 2010-d).

2.3.1. 4 Association between processed foods intake and metabolic syndrome

Development of metabolic disorders including high levels of blood cholesterol, blood sugar, and high Bp is closely associated with processed/fast foods intake that are rich in processed sugars and salt. The high content of fats, processed sugars, and refined carbohydrates of processed/fast food promotes the development of obesity, which in turn causes metabolic abnormalities (WHO, 2017-a, WHO, 2015). Indeed, obesity causes several metabolic abnormalities such as high blood cholesterol, raised BP, insulin resistance, of which all are features of MetS (Popkin *et al.*, 2012, Misra *et al.*, 2011, Paniagua *et al.*, 2011).

2.3.2. Association between level of physical activity and metabolic syndrome

Availability of modern mechanical transportation has contributed to sedentary lifestyle and increased occurrence of chronic diseases notably CVDs. Less than 150 minutes (5 times 30 minutes) of moderate or less than 75 minutes (3 times of 25 minutes) of vigorous-intensity physical activity per week is considered insufficient (WHO, 2011-d). Particularly, urban residents are at a higher risk of MetS due to a sedentary form of lifestyle attributed to the availability and use of automobiles. Lack or insufficient physical activity is identified as one of the fourth chief causes of CVDs-related mortality. An increased amount of sedentary time is linked to an elevated risk of MetS-related components, including reduced HDL-C, increased FBG, and raised TGs (Gennuso *et al.*, 2014, Prasad *et al.*, 2012, Kim *et al.*, 2011).

Lack of enough physical activity contributes to insulin insensitivity and hardening of blood vessels, causes type-2 diabetes and high BP, respectively. Furthermore, physically inactive people are at about a 25% elevated risk of all-cause mortality relative to active individuals (WHO, 2009-

c). Conversely, studies observed the lowest rate of MetS and most of its components among individuals who are involved insufficient physical activity compared to physically inactive people (Junga *et al.,* 2016, Jui-Hua *et al.,* 2017). Evidence indicated that people who were regularly involved in physical activity had lower rates of high BP, blood glucose, heart attack, and stroke events, as well as MetS, compared to physically inactive adults (WHO, 2011-d). Moreover, research revealed that adequate physical activity prevents the occurrence of heart attack and type-2 diabetes by about 30% and 27%, respectively (WHO, 2009-c).

Furthermore, regular physical activity helps with weight loss and proper utilization of glucose and, therefore, improves blood glucose, BP, and cholesterol levels. Regular physical activity also increases vasodilation by increasing vascular nitric oxide concentration, decreases inflammatory mediator release from skeletal muscle and adipose tissue, and regulates autonomic imbalance (Bowles and Laughlin, 2011), hence prevents from hypertension and MetS.

2.3.3. Association between alcohol use and metabolic syndrome

Excessive intake or harmful use of alcohol is defined by the WHO dietary guideline as more than 1 standard drink for women and more than 2 standard drinks per day for men (WHO, 2009-c). Harmful use of alcohol is recognized as one of the four major risk factors of CVDs including type-2 diabetes and CVDs (WHO, 2009-c). Excessive consumption of alcohol substantially increases chances of developing obesity, dyslipidemia (Wakabayashi, 2013, Chen *et al.,* 2012), hyperglycemia (Cullmann *et al.,* 2012), and hypertension (Kimani et al., 2019, Kaur, 2014), all are elements of MetS and cardiovascular risk factors. Studies in the United States showed MetS and all its elements were linked to daily consumption of alcohol that exceeded the WHO dietary guideline recommendations (Sun *et al.,* 2014, Wakabayashi, 2014). Likewise, in Kenya, misuse of alcohol has been identified as the primary risk factor of NCDs, e.g., heart disease, liver cirrhosis, cancer, high BP, and dyslipidemia (Kenya Ministry of health, 2015). Approximately, a third (31.7%) of people in rural western Kenya consume alcohol and the figure is higher in men (54.6%) than in women (8.9%) (Takahashi *et al.,* 2017).

The underlying mechanisms involved in the pathogenesis of alcohol-induced MetS can be explained as follows: Excess alcohol consumption leads to obesity (Sayon-Orea *et al.,* 2011), which leads to other several biochemical and clinical abnormalities, including type-2 diabetes, dyslipidemia, hypertension (Popkin *et al.,* 2012, Misra *et al.,* 2011), all of which are elements of the MetS, main risk factors for CVDs. For alcohol-induced hypertension, several mechanisms are involved including activation of the sympathetic nervous system, causing vasoconstriction, increased cardiac contractility, and impairment of baroreceptor refluxes resulting in aberrant auto-regulation (Rehm *et al.,* 2010). Harmful use of alcohol increases triglycerides synthesis through inhibition of hepatic fatty acid oxidation (Min and Gavin, 2019), resulting in dyslipidemia.

2.3.4. Tobacco smoking and metabolic syndrome

Tobacco use has been recognized as one of the four leading causes of NCDs. Studies have established a positive correlation between tobacco use and MetS (Slagter *et al.,* 2013, Sun K *et al.,* 2012). Tobacco smoking causes insulin insensitivity, a principal causative variable for both type-2 diabetes and CVDs. It may also increase TGs and decrease HDL-C in the blood, raising the risk of a heart attack (Rawan *et al.,* 2018). Insulin resistance raised triglycerides and reduced HDL-C- the two components of MetS. The nicotine released from cigarettes overstimulates the sympathetic nervous system resulting in blood sugar and lipid metabolic dysregulation, which contributes to the development of MetS (Bigazzi and Bianchi, 2007). Nicotine, from cigarettes, induces the release of multiple neurotransmitters such as cortisol (Wilkins *et al.,* 2001). The excessive cortisol level in the blood causes excess accumulation of abdominal fat (Chiolero *et al.,* 2007), a major risk factor for MetS. Hence, cigarette smoking can contribute to the development of MetS (Bigazzi and Bianchi, 2007). Moreover, tobacco smoking induces catecholamine release, resulting in vaso-constriction, coronary spasm, and high BP. Second-hand smokers are also at increased risk of NCDs (WHO, 2010-a).

2.3.5 Level of knowledge of cardiovascular diseases

The prevalence of the major lifestyle risk factors of CVDs such as unhealthy diets, use of alcohol and tobacco (WHO, 2011-a, Roth *et al.,* 2015, Hamid *et al.,* 2019), lack of physical activity (Ofori and Garcia, 2016), and obesity, (Ofori-Asenso *et al.*, 2016) are rising in the SSA region. Gaps in knowledge of CVD risk factors and preventive actions in the general population are important barriers in the effective prevention and treatment of CVDs (Mohd *et al.,* 2017). To reduce the rising burden of CVD in SSA, population-based awareness creation approaches are warranted.

Indeed, a knowledgeable population is more likely to make healthier lifestyle choices, recognize disease risk factors and adopt positive health-seeking behaviours (Bergman *et al.,* 2011).

Most of the SSA population has limited knowledge on these lifestyle risk factors of CVDs. A systematic review in the SSA region showed that the majority had low levels of knowledge on risk factors for CVDs (Boateng *et al.,* 2017). Four studies (Nakibuuka *et al.,* 2014, Yuqiu and Wright, 2008, Wahab *et al.,* 2015, Temu *et al.,* 2015) in the SSA region reported that <30% of study participants cited alcohol consumption as a risk factor for CVDs. A study conducted among staff in Nigerian University reported that 81% had a low level of knowledge of CVDs (Akintunde *et al.,* 2015). Knowledge of physical inactivity or sedentary lifestyle as risk factors for CVD ranged from 0.6% (Wahab *et al.,* 2015) to 57%, (Komolafe *et al.,* 2015) in Nigeria. Furthermore, heavy alcohol consumption as a risk factor for CVD was reported by only 4.5% in a study among patients with hypertension and/or diabetes at specialist medical outpatient clinics in Nigeria (Wahab *et al.,* 2015). Knowledge of smoking as a CVD risk factor was less than one percent among the general populations in Central Uganda (Nakibuuka et al., 2014). Similarly, a study conducted on people living with HIV in Kenya reported a low level of knowledge on CVDs with a mean (SD) score of 1.3 (1.3) out of possible 10 points. Most (77.7 %) could not identify any warning signs for heart attack (Temu *et al.,* 2015). An estimated 75% of Kenyans who live with hypertension do not know they suffer from it, and of those who are aware, only four percent controlled their BP level (Kenya Ministry of Health, 2018).

Without adequate awareness of CVD lifestyle risk factors and preventive measures, it is hard for individuals to modify their lifestyles to reduce the risk of CVD. Community awareness and knowledge of CVDs can lead to better prevention and control of these diseases, as knowledge empowers individuals and communities to prevent or manage these conditions (Fottrell *et al.,* 2019, Aminde *et al.,* 2017). This calls for the development and implementation of cost-effective population-based health education intervention approaches to create and promote public awareness of CVDs prevention measures in the SSA region. Education interventions focusing on specific lifestyle recommendations so that populations have comprehensive information to make informed lifestyle changes will maximize the public health benefits of recommended health practices.

Nurses can significantly play important roles of equipping and empowering communities with CVD prevention knowledge and skills. If nurses are well trained and equipped with adequate knowledge of CVDs prevention, they could be a key resource for improving community awareness on CVD especially in low-income settings through their routine health promotion activities. Indeed, systematic reviews have demonstrated that nurses could be effective in tackling the burden of CVD in both low- and- middle-income countries (Khetan et al., 2017). The effectiveness of nurses could be attributed to their wider reach in many areas, rapport with community members, and their scope of practice to deliver holistic care (Khetan et al., 2017, Hill *et al.,* 2017).

2.4. LIFESTYLE MODIFICATION FOR CONTROLLING METABOLIC SYNDROME

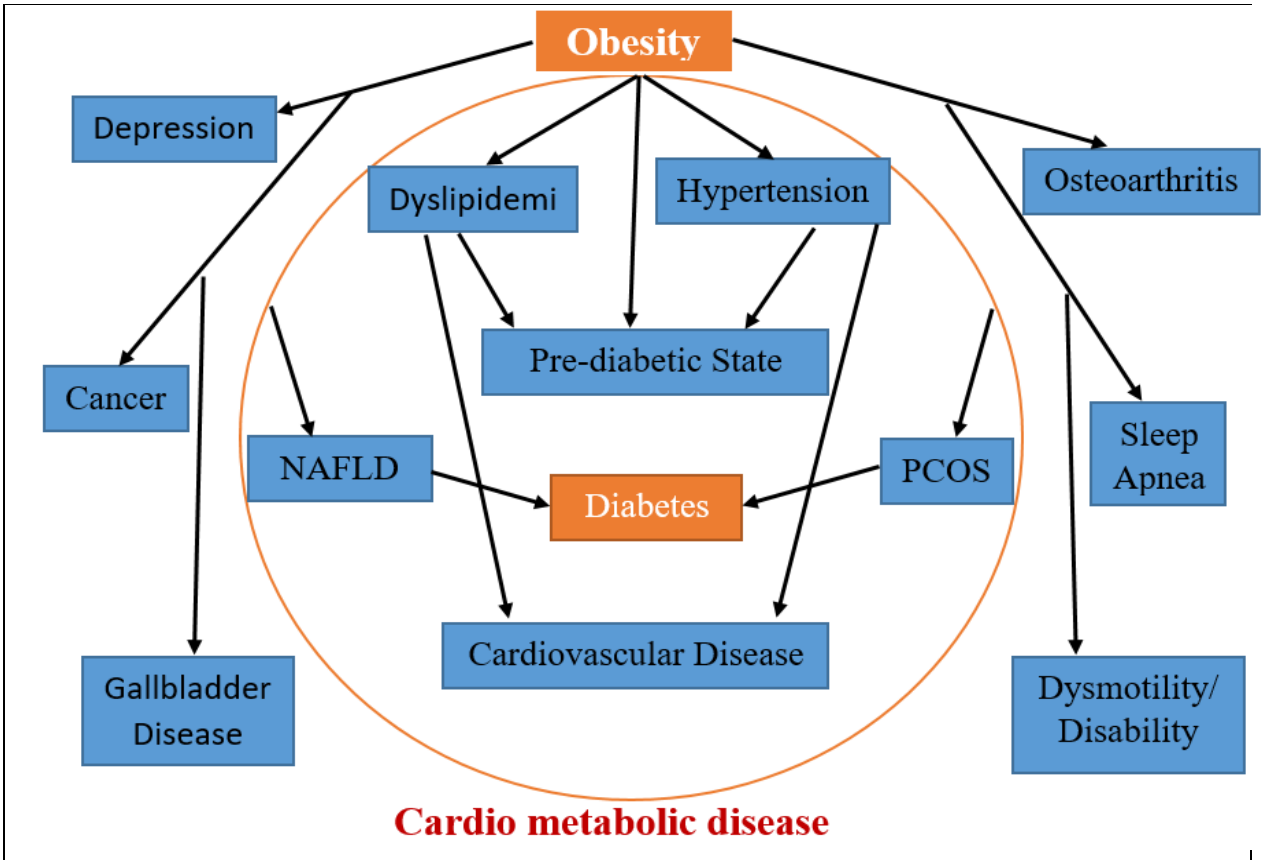
2.4.1 Introduction

Maintaining a healthy weight, consuming a healthy diet, avoiding or limiting alcohol, avoiding smoking, and engaging in an adequate level of physical activity are the lifestyle measures to be taken to control MetS, a known precursor to CVDs. Dietary and physical activity interventions carried out for 6-12 months have reported positive effects on the elements of MetS, including waist circumference and level of triglycerides (Yamaoka and Tango, 2012, Chia-Huei *et al.,* 2014). Participating in an adequate level of physical activity and consuming a diet rich in fibre such as whole-grain cereals, vegetables, and fruits, are found to be effective in reducing risks of type-2 diabetes in adults (Burnet *et al.,* 2011).

2.4. 2. Reducing excess weight to control metabolic syndrome and CVDs

Overweight and obesity are the primary cause of high BP, diabetes, and MetS. Several studies have reported a direct relationship between body weight and MetS (Megan *et al.,* 2016, Moreira et al., 2014, Steffen *et al.,* 2014). The prevalence of all the elements of MetS including high BP, raised fasting blood glucose, and dyslipidemia is substantially linked to increased BMI (WHO, 2009-c). Therefore, losing weight can be considered as an effective strategy to reduce the risks of MetS and CVDs. Adhering to a healthy diet and participating in an adequate level of physical activity can reduce extra weight, and therefore reduce the risk of MetS. A diet rich in fibre such as vegetables, fruits, whole grains, legumes, and nuts is highly recommended to lose weight.

2.4.4.1 Non-communicable diseases associated with central obesity



Source:WT Garvey, 2011. NAFLD; nonalcoholic fatty liver disease, PCOS; polysystic ovary syndrome

Figure 3 Cardiovascular and other chronic diseases associated with central

2.4.3. Eating a healthy diet to control metabolic syndrome and CVDs

Regular intake of fruits, vegetables, whole grain cereals, legumes, and nuts is considered a healthy dietary component. Reduce intake of processed/fast foods, sugar, and salt. The Dietary Approach to Stop Hypertension (DASH) eating plan is a good strategy to control portion of carbohydrate, animal-based proteins, and vegetables and/or fruits consumption (Onvani *et al.*, 2015).

2.4.3.1 Increase fruits and vegetables intake to control MetS and prevent CVDs

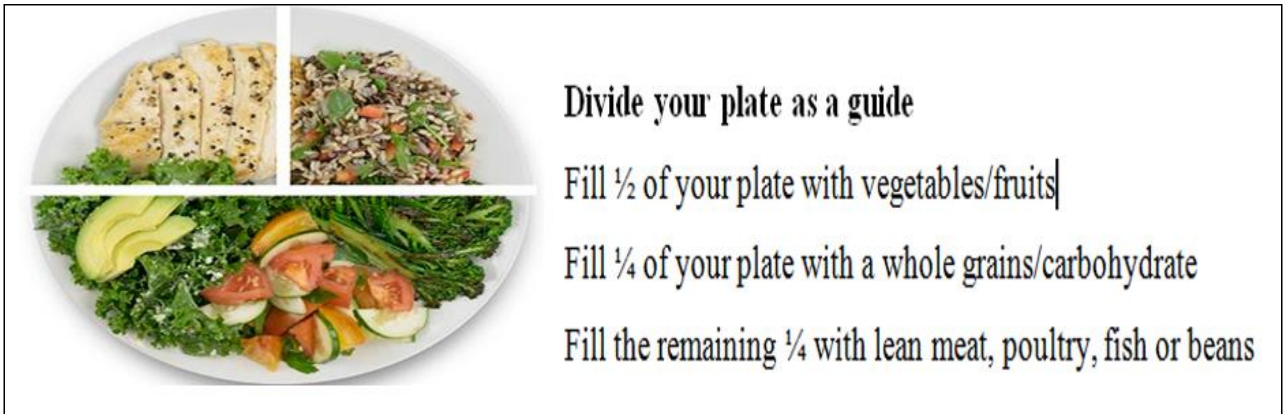
Taking 4-5 servings of fruits and vegetables (3 of vegetables and 2 of fruit) in a day is recommended by the WHO to prevent CVDs (WHO, 2012-c). The beneficial effect of fruits and vegetable consumption on MetS and CVDs is well established. Vegetables and fruits are rich in vitamins, minerals, and fibre, but low in fat and calories. Eating more vegetables and fruits and limiting fat intake is substantially linked to a lower risk of obesity, dyslipidemia, hypertension, type-2 diabetes, MetS, and CVDs (Asemi *et al.*, 2014). Vegetables, fruits, and legumes are rich in soluble fibre (Vrieze *et al.*, 2010), which may offer the best protection against type-2 diabetes, hypertension, and MetS. Fruits and vegetables are full of vitamins, phytochemicals, fibres, potassium, and antioxidants which all have MetS and CVDs protective effects (Steemburgo *et al.*, 2009).

Soluble fibres which are abundantly found in fruits and vegetables decrease intestinal absorption of carbohydrates, cholesterol, and bile salts, and thus improve blood lipid and sugar levels (Visioli, 2011). Moreover, fruits and vegetables have a high concentration of potassium, an important co-factor for BP regulation. With a normal level of potassium in the blood, there is more excretion of salt and water by the kidneys, thus lowering the BP (WHO, 2012-c). However, if there is a low level of potassium in the blood, there is more retention of sodium and water, causing high BP (Rheinschild, 2017, Savica *et al.*, 2010).

2.4.3.2 Essential effects of adherence to the DASH diet on metabolic syndrome and CVDs

The DASH diet is recommended for people either to prevent or treat hypertension. It is rich in vegetables, fruits, whole grains, legumes, and nuts, but restricts animal-based proteins, salt, added sugars, and fat. It encourages no more than 1 teaspoon of sodium per day, which is in line with the WHO dietary guidelines (WHO, 2012-c). According to the DASH eating diet, meal plates should be filled with half of vegetables and/or fruits, one-quarter with proteins, and the remaining one-quarter with carbohydrates to control high BP (Onvani et al., 2015). Several studies have reported a positive impact of adherence to the DASH diet on MetS (Saneei *et al.,* 2015, Babio *et al.,* 2014), type-2 diabetes, and CVDs (Salehi *et al.,* 2013, Levitan et al., 2013). Further, Kim and colleagues (2011) found that the rate of MetS and levels of TGs were inversely associated with regular intake of vegetables and grains.

Recommended dietary portions as per the DASH diet to control and prevent CVDs



Source: *The National Heart, Lung and Blood Institute (NHLBI). (2015). The DASH diet (Dietary Approaches to Stop Hypertension. Agency of the United States Department of Health and Human Services*.

Figure 4 Utilize the DASH diet to control metabolic syndrome and prevent CVDs

Table 2 components and significance of each food group of the DASH eating diet

Food group	Daily servings	Serving sizes
Grains	6-8	• 1 slice of whole-grain bread • 4 tablespoons (28 grams) of dry, whole-grain cereal • 1/2 cup (95 grams) of cooked rice, pasta or cereal
Vegetables	4-5	• 1 cup raw leafy green vegetable like spinach or kale • 1/2 cup of sliced vegetables — raw or cooked — like broccoli, carrots or tomatoes • ½ cup vegetable juice
Fruits	4-5	• 1 medium size fruit • 1/4 cup (50 grams) of dried apricots • 1/2 cup (30 grams) of fresh, frozen or canned
Dairy Products (low in fat-skim milk and low-fat cheese and yogurt).	2-3	• 1 cup (240 ml) of low-fat milk • 1 cup (285 grams) of low-fat yogurt • 6 tablespoons (45 grams) of low-fat cheese
Lean Chicken, Meat and Fish	6 or less	• 28 grams of cooked meat, chicken or fish • 1 egg
Nuts, seeds, and legumes	4-5 per week	• 1/3 cup (50 grams) of nuts • 2 tablespoons (40 grams) of nut butter • 2 tablespoons (16 grams) of seeds • 1/2 cup (40 grams) of cooked legumes
Fats and oils	2-3	• 1 teaspoon (4.5 grams) of soft margarine • 1 teaspoon (5 ml) of vegetable oil • 1 tablespoon (15 grams) of mayonnaise • 2 tablespoons (30 ml) of salad dressing
Sweets and added sugar	5 or less per week	• 1 tablespoon (12.5 grams) of sugar • 1 tablespoon (20 grams) of jelly or jam • 1 cup (240 ml) of lemonade

Source: *The National Heart, Lung and Blood Institute. (NHLBI) (2015). The DASH diet (Dietary Approaches to Stop Hypertension. Agency of the United States Department of Health and Human Services.*

2.4.3.3 Increase legumes and nuts intake to control MetS and prevent CVDs

As recommended in the DASH diet, individuals should take 4-5 servings of legumes and nuts per week to prevent or control MetS and CVDs. There are varieties of legumes/pulses such as black beans, black-eyed peas, kidney beans, etc. They are rich in several elements with bioactive properties, such as fibres, polyphenols, vitamins, iron, magnesium, and zinc (Mozaffarian *et al.*, 2011), which all have MetS and CVDs protective effects.

Studies have shown that adequate intake of legumes reduced the incidence of MetS (Mahan et al., 2016, Hosseinpour *et al.*, 2015) and its components including high BP (Jayalath *et al.*, 2014, Jenkins et al., 2012), blood glucose, and lipid levels (Ley *et al.*, 2014). Likewise, a study conducted in adults with type-2 diabetes reported that regular intake of legumes enhanced insulin sensitivity, reduced blood glucose level (Mahan *et al.*, 2016, Sala-Vila *et al.*, 2015), and risk of CVDs (Maphosa and Jideani, 2017, Messina, 2016). Similarly, interventional studies revealed that regular consumption of nuts markedly reduced body weight (Xiaoran *et al.*, 2019), waist circumference (Hang *et al.*, 2018), BP (Jayalath *et al.*, 2014), and TGs (Sabate et al., 2010)- some elements of MetS. Further, a controlled trial study has reported the beneficial effects of legumes and nuts consumption on type-2 diabetes, high BP, and CVDs (Orlich and Fraser (2014).

The mechanisms involving the link between increased legumes and nut intake and reduced risks of MetS and CVDs can be described as follows. Legumes are full of viscous soluble fibre which decreases absorption of cholesterol, carbohydrates, and bile salts in the intestine. This controls blood glucose (Bouchenak & Lamri-Senhadji, 2013; Hutchins *et al.*, 2012, Sievenpiper *et al.*, 2009) and blood lipid levels (Visioli, 2011). Soluble fibres also have a cholesterol-lowering effect by increasing the excretion of bile salt in feces (Gunness & Gidley, 2010). The protein component found in legumes contributes to its MetS-CVDs protective effect through modulating plasma lipids and displacing saturated fats found in animal-derived proteins with healthy plant-based proteins (Rebello *et al.*, 2014).

Regarding nuts, they are rich in a vegetable type of proteins, fibre, folate, unsaturated fatty acids, minerals, antioxidants, and bioactive phytochemicals. eg. Flavonoids (Jayalath *et al.*, 2014, Ros and Hu, 2013, Mozaffarian *et al.,* 2011), all have MetS-CVDs protective effect by controlling inflammatory process and oxidative stress (Banel and Hu, 2009). These biochemical activities can reduce abdominal obesity and improve BP, blood sugar, lipid levels, and insulin sensitivity (Ros, 2010)- features of MetS. Nuts are also rich in dietary fibres, which have cholesterol-reducing effects (Bouchenak & Lamri-Senhadji, 2013; Hutchins *et al.,* 2012), blood glucose (Visioli, 2011), and excess weight (Salas-Salvado *et al.,* 2006). Moreover, nuts are high in L-arginine, a potent precursor of vasodilator nitric oxide contributes to a reduction in BP (Ros, 2010).

2.4.3.4 Limit or avoid consumption of processed foods to control MetS and prevent CVDs

The development of MetS and CVDs is closely linked to the consumption of processed/fast/junk foods. Studies have reported that consumption of processed/fast or junk foods is substantially related to MetS (Bahadoran *et al.,* 2013, Babio N *et al.,* 2012) and its components (Edyta et al., 2017, Rodriguez *et al.,* 2017, Asghari *et al.,* 2015). Of several dietary patterns, fast food intake has been evaluated as the chief cause of obesity (Garcia *et al.,* 2012, Mozaffarian *et al.,* 2011) and type-2 diabetes (Krishnan *et al.,* 2010, Micha *et al.,* 2010). It is, therefore, highly recommended to limit or avoid processed/fast food consumption.

The mechanism of how processed/fast food intake promotes the development of MetS is as follows. Frequently intake of fast foods, which are rich in saturated fats, cholesterol, and sodium, but poor in fibre, calcium, and antioxidant vitamins, contributes to the development of obesity and MetS (Millen et al., 2006). The content of such foods is mainly processed sugars, salt, refined carbohydrates, cholesterol– MetS friendly food but poor in fibre rich foods such as fruits and vegetables (WHO, 2017-a, WHO, 2015, Paniagua *et al.,* 2011). Additionally, processed/fast foods are rich in saturated fat which increases visceral adiposity and decreases fatty acid and glucose oxidation, resulting in insulin resistance and MetS development (Kennedy *et al.,* 2009). Further, fatty foods increase insulin resistance through activation of serine kinases, which inhibits the insulin phosphorylation cascade, decreases glucose utilization, and subsequently causes type-2 diabetes (Hotamisligil, 2006).

2.4.3. 5 Reduce salt intake to control metabolic syndrome and prevent CVDs

Limiting salt intake to no more than 1 teaspoon (5g) per day to control metabolic syndrome and reduce risks of CVDs. According to the WHO, the daily recommended amount of salt intake is less or equal to 1 teaspoon (5 grams) (WHO, 2012-b). Dietary salt plays a substantial role in determining the status of BP and CVD (WHO, 2010-d). Excess salt intake is the primary cause of elevated BP and CVDs. As CVDs risk reduction intervention, the WHO has recommended salt reduction as a strategy, a cost-effective and feasible approach to implement at the grassroots level (Alwan, 2011). Salt intake can be controlled by avoiding processed/fast foods such as fried foods, chips, and not adding salt after the food has been cooked as well as checking for "sodium" on food labels.

2.4.3. 6 Reduce sugar intake to control metabolic syndrome and prevent CVDs

Limit sugar intake to no more than 5 teaspoons (25g) per day to control metabolic syndrome and prevent CVDs. According to the WHO, the daily recommended sugar intake per person is less or 5 teaspoons to prevent NCDs notably hypertension and type-2 diabetes (WHO, 2015). Excessive sugar intake may lead to obesity, a principal precursor for insulin insensitivity, type-2 diabetes, dyslipidemia, and high BP (James et al., 2016), which are all components of MetS. Specifically, soft drinks like coca-cola and added sugars are determined as the main causative factors of central obesity (WHO, 2015, Bray and Popkin, 2014), dyslipidemia (Welsh *et al.*, 2011), Type-2 diabetes (Kimber, 2016, DiNicolantonio *et al.*, 2015), high BP (DiNicolantonio *et al.*, 2014) and MetS (Bray and Popkin, 2014, Denova-Gutierrez *et al.*, 2010). Therefore, limiting sugar intake to the recommended amount may substantially reduce the risks of MetS-CVDs in the adult population.

2.4.4. Participate in a regular physical activity to control MetS and prevent CVDs

An adequate level of physical activity is a crucial lifestyle factor to control high BP, type-2 diabetes, MetS, and CVDs. To prevent CVDs, the WHO recommends for adults (18-64 years old) to participate for a minimum of 150 minutes of moderate-intensity or 75 minutes of vigorous-intensity physical activity in a week (WHO, 2011-d). The same recommendation applies to individuals with MetS to reduce risks of CVDs (Abelson, 2010). Aerobic physical activity can be divided into three categories according to intensity: light, moderate and vigorous physical activity. Fast swimming, jogging, jumping a rope are some of the vigorous types of physical activity. Brisk walking, climbing stairs, fast cycling, gardening are examples of moderate physical activity. Examples of light physical activity includes walking and cycling slowly.

Studies have shown the protective effects of regular physical activity against MetS and CVDs (Mohamed-Hamad *et al.,* 2016, Junga *et al.,* 2016, Jaspinder, 2014, Huang and Liu, 2014, Park *et al.*, 2014). Studies in Taiwan (Jui-Hua *et al.,* 2017) and Korea (Junga *et al.,* 2016) observed the lowest rate of MetS and most of its elements in adults who regularly participated in moderate-intensity physical activity compared to people who did not engage in such activity. Engaging in regular physical activity enhances body cells to utilize glucose properly and thus reduces blood glucose levels. Participation in an adequate level of physical activity substantially reduces risks of type-2 diabetes, MetS, and CVDs by reducing body weight, improving insulin sensitivity, serum glucose, and BP levels (Kastorini *et al.,* 2011, Cornier *et al.*, 2008). Whereas, insufficient level of physical activity predisposes individuals to MetS-related components, including reduced HDL-C, increased FBG, and raised TGs (Gennuso *et al.*, 2014, Thorp *et al.*, 2013, Prasad *et al.*, 2012) and increases the risk of CVD-related mortality (Li and Siegrist, 2012).

The protective effects of physical activity against MetS can be explained by several facts. Sufficient physical activity reduces body weight, resulting in improvement of insulin sensitivity, blood glucose, BP, and dyslipidemia and therefore, reduces risks of having type-2 diabetes, MetS, and CVDs (Cornier *et al.,* 2008). Whereas, lack of physical activity decreases insulin sensitivity-a principal cause of type-2 diabetes, and also makes blood vessels stiff, increasing the risk of high BP and CVDs (Bassuk and Manson, 2010). Sufficient physical activities, increases the secretion of mitochondria fibres and beneficial hormones like Irisin, resulting in improved insulin sensitivity and reduced postprandial hepatic lipogenesis (Hofmann *et al.,* 2014). Further, regular physical activity increases vasodilation by increasing vascular nitric oxide concentration, resulting in BP reduction. It also decreases inflammatory mediator release from skeletal muscle and adipose tissue and corrects autonomic imbalance (Bowles and Laughlin, 2011), hence prevents metabolic abnormalities.

2.4.5 Limiting or avoiding alcohol consumption to control MetS and prevent CVDs

An excessive amount of alcohol intake is identified as one of the four primary causes of NCDs including MetS and CVDs. Excessive alcohol intake is defined by the WHO dietary guidelines as more than 1 standard drink a day for women and more than 2 standard drinks for men (WHO, 2009-b). Therefore, individuals with MetS should limit or avoid alcohol intake to control MetS and prevent CVDs. The United States-based studies reported that MetS was directly linked to excessive alcohol intake (Sun *et al.,* 2014, Wakabayashi, 2014). Elements of MetS such as dyslipidemia (Wakabayashi, 2013), hyperglycemia (Cullmann *et al.,* 2012), and high BP (Kimani *et al.,* 2019, Kaur, 2014) are closely associated with harmful use of alcohol. Moreover, the risk of CVD-related mortality is strongly linked to harmful use of alcohol (Li and Siegrist, 2012).

The underlying mechanisms involving in the pathogenesis of alcohol-induced MetS can be explained as follows: Excess alcohol consumption causes obesity, the precursor for MetS (Sayon-Orea *et al.,* 2011), which leads to other several clinical-biochemical abnormalities, including dyslipidemia, hypertension, insulin insensitivity, and thus and type-2 diabetes, which are all features of MetS (Popkin *et al.*, 2012, Nguyen *et al.*, 2012, Misra *et al.*, 2011). Further, excessive alcohol consumption activates the sympathetic nervous system, causing vasoconstriction, increasing cardiac contractility, and impairing baroreceptor refluxes leading to abnormal auto-regulation resulting in high BP (Rehm *et al.*, 2010).

2.4.6 Avoiding tobacco smoking to control metabolic syndrome and prevent CVDs

Cigarette smoking is among the four main behavioural risk factors of NCDs. The risk of developing insulin insensitivity, MetS, hypertension, type-2 diabetes, and CVDs is substantially increased with cigarette smoking. It also raises triglycerides and decreases HDL-C in the blood, raising the risk of MetS and CVDs. Furthermore, it causes vasoconstriction and spasms of coronary arteries by inducing catecholamine release, resulting in high BP and heart attack. Globally, each year, cigarette smoking contributes to approximately 6 million people death (WHO, 2009-b). However, evidence shows that one year after stopping smoking the risk of CVDs is lowered by 50%. Three to four years after stopping, the risk of CVDs is almost the same as a person who has never smoked.

2.5 Complications of metabolic syndrome-related cardiovascular diseases

MetS is a major precursor for CVDs notably hypertension and type-2 diabetes, known for their significant morbidity and mortality. The chance of developing heart disease, stroke, kidney failure, and leg amputation is substantially higher among people with MetS than someone without the syndrome. The chance of developing heart attack, stroke, kidney disease, and retinopathy is substantially higher when someone has both high BP and type-2 diabetes, the two main elements of MetS. High BP is one of the most important risk factors of CVD (WHO, 2019). Furthermore, it imposes a huge economic burden on the affected individuals and their families.

In Kenya, it is estimated that half (50%) of hospital admissions and most (55%) causes of death are from NCDs, with the chief causes being CVDs (WHO, 2012-a). Furthermore, diabetes-related complications including foot ulcers, infection, high BP, and dyslipidemia are common in many tertiary clinics in Kenya (Kenya National Diabetes Strategy, 2010). People with diabetes are at substantially high risk of nephropathy and neuropathy**.** Uncontrolled BP and blood glucose levels damage the small blood vessels of the kidneys, resulting in kidneys failure. Men with diabetes complication of neuropathy, suffer from sexual dysfunctions, a major cause of social and emotional stress (IDF, 2015). High BP narrows and damages coronary arteries, limiting blood supply to the heart. When blood cannot flow freely to the heart, the affected individual develops a heart attack. Furthermore, when BP is high, it decreases normal blood flow to the brain resulting in can be ischemic stroke.

2.6 THEORETICAL FRAMEWORK

There are several behavioral changing models aiming at promoting peoples' behavior to adopt a "healthy lifestyle". Examples of such models relevant to the current study are the Health Belief Model (HBM), the Health Promotion Model (HPM) and the Trans-theoretical Model (TTM).

The HBM proposes that people are most likely to take preventative action if they perceive the threat of a health risk to be serious, if they feel they are personally susceptible and if there are fewer costs than benefits to engaging in it (Ashraf and Melvyn, 2017). The central aspect of the HBM is that behavior change interventions are more effective if they address an individual's specific perceptions about susceptibility, benefits, barriers, cues to action and self-efficacy (Mark and Paul, 2021, Vicki, 2015).

1. Perceived susceptibility — refers to beliefs concerning susceptibility to a disease.
2. Perceived severity — refers to beliefs concerning the possible severity of a disease.
3. Perceived benefits — refers to the perceived value or benefit of behavior changes in reducing the risk of a disease.
4. Perceived barriers — refers to any obstacles or barriers to the behavior changes being considered to decrease risk.
5. Cues to action is an event hat spur individuals toward action.
6. Self-efficacy refers to an individual's confidence that he or she can successfully carry out the indicated actions (National Cancer Institute, 2005). The HBM has been applied with significant success to a range of preventive behaviors, such as diet, exercise, smoking cessation, vaccination, and adherence to recommended medical treatments (Mark and Paul, 2021).

The HPM aims to explain the factors underlying motivation to engage in health-promoting behaviors (Pender *et al.,* 2011). The model describes eight behavior-specific beliefs which are believed to determine the health-promoting behavior which are proposed as targets for behavior change interventions. These are: (1) perceived benefits and (2) perceived barriers to action, (3) perceived self-efficacy, (4) activity-related affect, (5) interpersonal influences (including norms, modeling/vicarious learning, and social support), (6) situational influences, (7) commitment to plan of action, and (8) immediate competing demands and preferences (Pender *et al.*, 2011).

The Transtheoretical Model (TTM): Although both HBM and HPM have been applied to promote peoples' behavior, they do not establish the current stage of the person towards the recommended behavioral change. This can be considered as weakness of the models. Because of each person is at a different stage of change, it is more effective when the interventions are matched with the individual's current stage of change.

Considering relevance to the current study, the TTM was applied as a behavioral changing tool to guide the health education intervention. Three major constructs of the model including self-efficacy, stages of change, and decisional balance (pros and cons) were determined at the beginning and end-line of the study towards a healthy lifestyle practice to control MetS. The model has been considered an important approach in lifestyle modification interventions, however, its efficacy in the management of MetS has not been determined. Contemporary psychosocial theories have been applying by health care professionals to examine the lifestyle or behaviour of individuals (Glanz *et al.,* 2008). The TTM of behaviour change is one of the most promising of these theories (Prochaska and DiClemente, 2003). The TTM of behaviour change, originally developed by Dr. James Prochaska and his colleagues in 1988, addresses the psychological mechanisms of changing health behaviours.

The TTM of behaviour change provides supportive information for health education and guides the method of how people attempt to modify health-related risk behaviours. According to the model, all people are not ready at the same time to change their current behaviour to manage their health status (Spellman, 2008). Since each person is at a different stage of change, it is more effective when the interventions are matched with the individual's current stage of change.

The model has been successfully employed to several health behaviours notably smoking cessation, and recovery from drug addiction (Stanton and Grimshaw, 2013, Vilela *et al.,* 2009, Spencer *et al.,* 2006). A study done by Nitzke *et al.* (2007) reported that the intervention group significantly improved vegetables and fruits intake, and greatly progressed to the maintenance stage than the control group after the TTM-based intervention. Another study conducted in Egypt (Abdel-Fatah *et al.,* 2017) regarding dietary changes among pregnant women using the TTM, found that the group who received the intervention significantly improved their self-efficacy, decisional balance, and stage of change than the control group. Mohsen *et al.* (2014), who used the TTM to encourage lifestyle changes, reported a significant improvement in the pros of decisional balance and self-efficacy regarding changes in dietary behavior.

Stages of change (figure 2): This refers to a stage of progress through five sequences of steps (Glanz *et al.,* 2008). The TTM suggests that individuals change behaviour in five stages that integrate current behaviour with their intention to change or maintain behaviour. According to the model, the five stages of motivational readiness to change a specific behaviour are pre-contemplation, contemplation, preparation, action and maintenance (figure 2) (Spellman, 2008, Glanz *et al*., 2008).

Pre-contemplation: This is the phase in which the person does not have any plan to change his/her behaviour in the next 6 months. A healthier lifestyle is not yet considered. For example, an individual who is at high risk for hypertension or diabetes is not thinking about any preventive or control measures of hypertension/diabetes. Individuals at this stage are unaware of their problems because they were never informed or underinformed about the consequences of their behaviour.

Contemplation: (the person is conscious of the problem and considering change). This is the phase where a person is thinking to change his/her behaviour in the next 6 months. At this stage, he/she is aware of the consequences of the current behaviour and is seriously planning to take action. At this point, the individual is aware of preventive measures and is planning to start the new behaviour.

Preparation: (making arrangements and intending to change). At this phase, the person is ready to take action in the next month. The person generally has a plan of action and making necessary preparations to start the new behaviour.

Action: (taking initial steps). At this stage, the person has started practicing the new behaviour for less than 6 months.

Individuals at this stage are practicing the desired behaviour such as changing in dietary patterns, engaging in physical activity, reducing or avoiding alcohol intake, taking medicines regularly. This stage of change requires a commitment by the individual to avoid relapse. To be considered as an action, the person must show substantial changes to reduce the risk of disease. For alcohol abuse, for example, some people believe that controlled drinking an effective action, whereas others consider total abstinence as effective action. For smoking, action is considered if the person has stopped it.

Maintenance: (sustained behaviour change for at least six months). At this stage, the person has been practicing the new behaviours for more than 6 months. This is a stage in which the person is committing to maintain gains attained during the action stage. Such an individual is confident to sustain the changes made and thus less tempted to relapse.

Decisional balance: It is the balance between the person's perception of the advantages (pros) and disadvantages (cons) of adopting a new behaviour (Koyun and Eroglu, 2014). In order for a person to make positive changes, the benefits (pros) must be greater than the costs (cons) (Yasin *et al.*, 2011). The changes are bidirectional, the person can progress or relapse from one stage to another. Hence, to progress from pre-contemplation to the next level of change, the benefits of changing must increase and the disadvantages or cons of changing must decrease.

Self-efficacy: It is the person's perceived self-assurance to make a behaviour change effectively (Koyun and Eroglu, 2014). Both self-efficacy and decisional balance are considered behavioural determinants to make changes. Behaviours risk issues can be greatly reduced or eliminated by self-controlling efforts. Taking unhealthy diet, excessive alcohol, tobacco and lack of physical activity are the four main modifiable risk factors of the four major NCDs including CVDs and type-2 diabetes. Applying behavioural change models in lifestyle modification intervention is more effective in enhancing long-lasting behaviour change than traditional counseling (Elder *et al.*, 1999).

To help an individual move to the next stage of change, first, it is important to determine the persons' current stage of change towards the desired behaviour during the assessment phase. Then based on the identified current stage, stage-matched interventions can be provided. According to the model, people at different stages have different needs for health education intervention (Prochaska and DiClemente, 1984).

CHAPTER THREE: METHODS AND MATERIALS

3.1 Study design

This was a two-arm randomized controlled trial with participants allocated on a 1:1 ratio across the groups. It was conducted between May 2018 and August 2019 among 352 adults with MetS. The total study duration from recruitment to end-line took 15 months, with each participant followed for 12-months. The study was structured into three phases (Figure 5) namely baseline, intervention phase and evaluation phase.

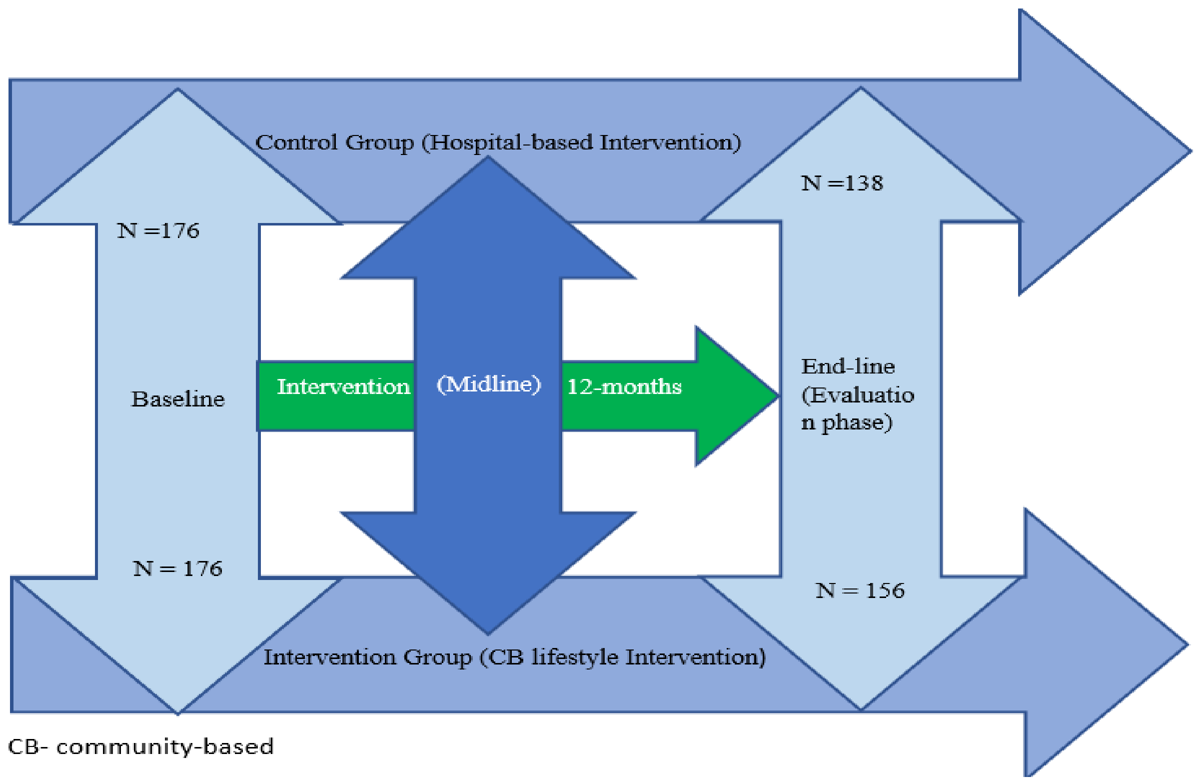


Figure 5. Implementation time frame for the study

Baseline: This involved screening, recruitment, allocation of participants into groups and baseline data collection. During the baseline data collection, socio-demographic information, knowledge on risk factors and prevention measures of CVDs, lifestyle characteristics, anthropometrics, BP and metabolic biomarkers (FBG, TGs, HDL-C) were measured. Using the TTM, the respondents' stage of change towards lifestyle practice was determined as pre-contemplation, contemplation, preparation, action, and maintenance.

Intervention phase: The intervention phase involved provision of TTM-based-stage matched lifestyle intervention to the intervention group (Box 1). The phase involved face-to-face delivery of verbal and written individualized health education and recommendations on risk factors for CVDs in the intervention arm. The control group was exposed to routine care provided in the hospital by health care workers as per the conventional clinical practice. The care included screenings, check-ups, patients counseling and treatment with drugs. The intervention phase ran for 12 months with midline data collection conducted at 9 months. During the midline, anthropometrics, physiologic such as BP and biochemical markers (FBG, TGs, HDL-C) were measured. Additionally, during the midline data collection, health education intervention was reinforced on the intervention group. Furthermore, during the follow-up period (between baseline & midline and between midline and endline), the intervention group received health education intervention through email, SMS, and WhatsApp messages while were in their homes.

Evaluation phase: This was conducted at month 15. This involved collection of data on knowledge and lifestyle practices towards MetS and CVDs prevention measures, anthropometrics, physiologic notably BP, and biomarkers for both the control and intervention groups. Thereafter, the face-to-face health education as well as written health education package messages were provided to both the control and intervention groups at the conclusion of the study.

Follow up: At the beginning of the study, the participants were given follow-up inquiries indicating the next visits or appointments. To follow effectively, participants' phone numbers and email addresses were recorded. At 6 and 12-months post-intervention, the participants were reminded about the appointment days using telephone calls. Those who missed their appointment days were called again and given another day of the appointment.

3.2 Study Site

Recruitment of the study participants was done at St. Mary's Mission Hospital in Nairobi, while follow-up took place in the community for 15-months between May 2018 and August 2019. The hospital is a Christian faith-based health organization dedicated to providing affordable services to the informal settlements of Kibera, Mukuru-Kwa-Njenga, and Kuwinda. Indeed, the Kibera is the largest and most populous informal settlement in Africa. The average monthly income of these inhabitants is USD 39 per person (Amelie and Sophie, 2011). The hospital gives comprehensive inpatient and outpatient services. Hypertension–diabetic clinic is one of the outpatient departments which operates Monday-Friday and serves about 600 hypertensive-diabetic patients in a month. The clinic runs by a multidisciplinary team that includes nurses, doctors, nutritionists, laboratory technicians, social workers, and pharmacists. Daily, the hospital serves about 1, 200 outpatients. Monthly, it conducts about 900 deliveries, and approximately 500 major and 500 minor surgeries. The study is a community-based intervention because after the participants have been recruited at the hospital, they were followed in the community while they were in their homes for 12-months.

3.3 Study population

A total of 352 obese adults aged 18–64 years, attending St. Mary’s Mission Hospital met the diagnostic criteria for MetS and agreed to participate in the study. Individuals who visited the hospital as outpatients and those who escorted them (relatives or friends) were systematically screened for MetS using the IDF criteria (Alberti et al., 2009). Eligible participants were randomly allocated to either normal care control (n = 176) or comprehensive lifestyle intervention groups (n = 176) using a block stratified randomization technique. Of the 352 individuals who initially enrolled in the study, 294 (intervention = 156, control = 138) completed the 15-month study period. This represents a response rate of 83.52% (IG = 88.64%, CG = 78.41%).

Majority of the people who reside in these informal settlements are referred to as the ‘urban poor’ because, most of them live below the poverty line (World Bank, 2006). The study was focused on the urban low-income people because they are disproportionately at increased risk of behavioural and metabolic risk factors of MetS and CDs. Because of lack of awareness and financial constraints, they have less access to health screening and care than other groups. Furthermore, they often consume processed foods and are more sedentary than rural residents (Popkin, 2003), major risk factors for MetS and CVDs. Furthermore, their access to healthcare is limited, partly related to their poor purchasing ability.

The risks for CVDs among individuals from poor social economic status have been shown to be higher. For example, the high proportion of CVDs among slum dwellers was associated with poverty-related stress and adopting unhealthy healthy lifestyles (Van de Vijver *et al.*, 2015). Therefore, the negative impact of CVD is specifically heavy among the urban poor, who may not have adequate knowledge and awareness as well as financial resources to adopt healthier lifestyles (Popkin and Gordon, 2004, Nyaruhucha *et al.*, 2003).

3.3.1 The inclusion criteria at the baseline were:

a. Age 18- 64 years and residence of Nairobi
b. Central obesity (WC ≥94 cm for men and ≥80 cm for women) plus at least 2 of the below-listed elements of MetS (Alberti et al., 2009**).**
   i. Elevated BP (systolic BP ≥130 mm Hg or diastolic BP ≥85 mm Hg) or on treatment for it
   ii. Raised fasting blood glucose (≥5.6 mmol/L) or receiving treatment for diabetes
   iii. Elevated triglycerides (≥1.7 mmol/L or on treatment for elevated TGs
   iv. Low HDL-C (<1.29 mmol/L in females and <1.03 mmol/L in males) or under treatment for it.
c. Willingness to give consent to take part in the study

3.3.2 Exclusion criteria included:

a. Adults aged above 64 years
b. Absence of central obesity (WC ≥94 cm for men and ≥80 cm for women)
c. Absence of metabolic syndrome
d. Pregnant or lactating mothers
e. Any medical contraindication that would limit physical exercise including physical disability, cancer, history of heart attack, mental disorder, end-stage kidney disease were not included in the study.

3.4 Sample size calculation

The below formula by Casagrande *et al.* (1978) was used to established the required sample size

$$n = \frac{\{Z_{1-\alpha/2}\sqrt{[2P(1-P)]} + Z_{1-\beta}\sqrt{[P_1(1-P_1)+P_2(1-P_2)]}\}^2}{(P_1-P_2)^2}$$

Description;

α = 0.05 (Type I error), β = 0.10 (Type II error), at 95% confidence interval, $Z_{1-\alpha/2}$ = 1.96, and at 90% power, $Z_{1-\beta}$ = 1.28.

P_1= Prevalence of MetS at 25.6% in Kenya (Geoffrey et al., 2017) in the control arm.

P_2= Rate of MetS in the intervention arm to be 11.6% (assuming that the lifestyle intervention will reduce the prevalence of MetS by 14%). This was assumed for calculating the sample size. Thus, the effect size was 14%.

$$P = \frac{P_1 + P_2}{2}$$

$$n = \frac{\{Z_{1-\alpha/2}\sqrt{[2P(1-P)]} + Z_{1-\beta}\sqrt{[P_1(1-P_1) + P_2(1-P_2)]}\}^2}{(P_1 - P_2)^2}, \qquad \mathbf{n = 160}$$

To present a 90% power at a significance level of 5%, and to get an absolute effect size of 14% reduction in the proportion of MetS, it was required 160 study subjects in each group using the above formula by Casagrade et al. (1978). Furthermore, to cover attrition, 10% (n = 16) of the initially calculated sample size of each group was added. Therefore, each group had 176 respondents, totaling 352 adults with MetS.

3.5 Sampling technique

The study respondents were selected using a systematic random sampling technique. According to the IDF definition of MetS, central obesity (WC ≥ 94 cm in men and ≥ 80 cm in women) is compulsory to screen for the other components of MetS. The study population was, therefore, considered as a high-risk group for MetS. Evidence has shown that the rate of MetS among obese adults ranging from 65 to 80% (Basma et al., 2018, Vatakencherry and Saraswathy, 2019). Therefore, the prevalence of MetS among this high-risk (obese) population was expected to range from 65 to 80%. Hence, the total population to be screened was estimated to be about 500 (450-550) to get 352 individuals with MetS. We considered including 25% (n = 125) of the study population known hypertensive-diabetic patients and 75% (n = 375) individuals without known chronic diseases.

Since both hypertension and diabetes are components of MetS, to limit the number of known hypertensive –diabetic patients to a maximum of 25% is important in order to determine the effect of the intervention among people without hypertension and diabetes as well as among patients with hypertension and diabetes.

During the baseline survey, monthly, approximately 600 patients with hypertension and/or diabetes attended the hypertensive/diabetic clinic. This translates to 1800 patients in three months, the period required to complete the baseline data collection. To determine the sampling interval, the target population (n = 1800) in three months period was divided by the number of hypertensive-diabetic patients initially calculated to be screened for MetS (n = 125). This gives a sampling interval of 14. Then, every 14th hypertensive-diabetic patient was selected and screened until the required sample size (n = 115) was reached.

Individuals who did not have known chronic diseases including hypertension and diabetes were systematically selected from the laboratory waiting bay of the hospital. These were both outpatients and those who were escorting their relatives/friends. During the baseline survey period, the laboratory of the hospital daily served about 110 adults aged 18-64 years, corresponding to 7, 920 in three months, the period required for the baseline data collection. To determine the sampling interval, the target population (n = 7,920) in three months periods was divided by the number of people calculated to be screened for MetS (n = 375). This produced a sampling interval of 21. Then, every 21st participant was selected and screened until the desired sample size (n = 237) was attained. Therefore, at baseline, the study population comprised known hypertensive–diabetic patients (32.7%, n = 115) and individuals without known chronic diseases (67.3%, n = 237).

3.6 Steps of screening and recruitment process

Step 1: Adults aged 18-64 years who attended the hospital during the baseline survey were screened for abdominal obesity.

Step 2: Individuals with abdominal obesity (WC ≥ 94 cm in men and WC ≥ 80 cm in women were further screened for the other components of MetS.

Step 3: Individuals with three (3) or more MetS components were re-consented and recruited for the study.

3.7 Randomization and allocation of study subjects

At baseline, the eligible participants were randomly allocated to the normal care group (n = 176) and a comprehensive lifestyle intervention group (n = 176) using a block stratified randomization technique. The normal care was the routine care provided in the hospital by health care providers according to the usual clinical practice. The lifestyle intervention entailed detailed verbal and written individualized recommendations focused on the main cardiovascular risk factors which are associated with MetS but considered changeable, as well as with the motivation for changing behaviour, using the TTM model of behavioural change. Further, individuals from the same family and who live together were assigned to the same group. This method reduces variability between the two groups. The participants were divided into 22 blocks, with an average of 16 subjects in each block. Then, subjects from each block were randomly allocated to the groups by picking a paper written "IG" or "CG" for intervention and control groups, respectively.

3.8 Study implementation

Before random allocation to groups, all the participants received general health education about the definition of MetS, type-2 diabetes, and hypertension, their major risk factors, complications, and the benefit of adopting a healthy lifestyle to prevent and control CVDs. Individuals who were found to have hypertension (BP ≥140/90 mmHg) and/or diabetes (FBG ≥ 7.0 mmol/L), but were not aware of their status were referred to the hypertensive-diabetic clinic of the St. Mary's Mission Hospital for further evaluations, treatment, and follow up, while they were part of the study.

3.8.1 Control group

The control group did not receive specific and detailed lifestyle interventions at baseline and mid-line. Moreover, no written recommendations regarding lifestyle interventions were given. However, after the end-line (evaluation phase), they received comprehensive specific lifestyle modification intervention including diet (sugar, salt, saturated fat, fruits and vegetables, nuts, legumes intake), alcohol consumption, and physical activity to control MetS and prevent CVDs. The health education intervention was delivered orally accompanied by printed material (Box 1).

3.8.2 Intervention group

After the standard education, the intervention arm was provided with comprehensive verbal and written individualized and group-based health education interventions at the baseline, 6 and 12-months. The health education intervention package was prepared by the principal investigator after reviewing relevant lifestyle recommendations to control MetS, hypertension, and type-2 diabetes). The health education intervention was targeting the common behavioural risk factors of CVD including unhealthy diet, harmful use of alcohol, tobacco use, and lack of adequate physical activity (WHO, 2011-a). Specifically, the intervention group was educated to adhere to the recommended dietary patterns, standard drinks of alcohol, and physical activity.

The TTM, a behavioral change model, was applied as a teaching and monitoring tool in the lifestyle modification process. At the baseline, the participants were assessed and evaluated their current stage of changes towards nine (9) MetS control-related healthy behaviours namely consumption of fruits, vegetables, processed/fast food, salt, sugar, adherence to the DASH eating diet, alcohol consumption, physical activity, and smoking. Based on their current stages of change towards each variable, they were classified into five stages that included: pre-contemplation, contemplation, preparation, action, and maintenance. Then, stage-matched detailed lifestyle modification intervention was provided via face-to-face interaction at baseline, midline, and endline. During the study period (15 months), there were 3 face-to-face intervention contacts and at least 2 online interventions (email/SMS/WhatsApp or direct telephone call).

Box 1. Health education package on lifestyle modification for clients with MetS

- **Consider the DASH diet** to reduce portion sizes, calorie intake and increase vegetables and fruits intake. Fill half of your meal plate with vegetables and/or fruits, one quarter with carbohydrates and the remaining quarter with plant-based proteins like legumes, soy products, nuts, and seeds proteins.
- **Avoid processed/fast foods**. They are full of processed sugars, salt, refined carbohydrate, saturated fat and low in whole grains. Limit fatty, dairy fat and cooking oil.
- **Reduce fat intake.** Use low-fat dairy products, vegetables oil, avoid fatty foods.
- **Reduce salt intake: Restrict** salt intake to less than 1 tea spoon (5grams) per day. Avoid adding salts on meals, read labels for salt content. Reduce salt when cooking, limit processes and fast foods.
- **Reducing sugar intake**: Restrict sugar intake to less 5 teaspoons a day. Avoid sweetened-beverages**.** Replace soda with water or fresh juice.
- **Choose low glycemic index foods** including: whole grain cereals, brown bread and rice, legumes, fruit, vegetables.
- **Use vegetables or olive oils for cooking,** take monounsaturated from beans, nuts and avocados.
- **Increase consumption of vegetables and fruits**. Consume 4-5 servings of them every day. 1 serving is equivalent to 1 orange or apple or mango or banana or 3 tablespoons of cooked vegetables.
- **Increase fibre intake.** Consume 4-5 servings of legumes (eg. beans, lentils, chickpeas and peas) and nuts a week to increase fibre intake.
- **Increase** time interval between taking dinner and sleeping, at least 2 hours
- **Alcohol:** If you drink, limit it to a max of 1 standard drink for women and 2 for men in a day.
- **Stop smoking!**
- **Increase physical activity**: Reduce periods of inactivity throughout the day. Engage for at least 150 minutes/week of moderate-intensity physical activity (brisk walking, digging, jogging, stairs climbing, cycling and housework) (5 days of 30 minutes a week). OR at least 75 minutes/week of vigorous-intensity physical activity (running, jogging, rope jumping, playing football/valleyball, digging, swimming) (3 days of 25 minutes a week).

3.6 Data collection tools

The data were collected by the principal investigator and two research assistants with a bachelor's degree in Nursing who had prior training on research ethics and data collection tools, while laboratory measurements were obtained by two experienced laboratory technicians. A pretested, structured questionnaire adopted from the WHO STEP-wise approach to NCDs risk factor surveillance (WHO, 2017-c) was used to collect the data. The tool had nine (9) sections: (1) questionnaire on socio-demographic and economic characteristics, (2) Anthropometric, clinical and biochemical measurements, (3) questionnaire on disease profile (history of hypertension, diabetes, dyslipidemia and other chronic diseases), (4) questionnaire on dietary intake patterns, (5) questionnaire on physical activity/exercise, (6) questionnaire on alcohol consumption, (7) questionnaire on tobacco smoking, (8) questionnaire on knowledge of participants towards MetS and CVDs risk factors and preventive measures, (9) questionnaire on stages of change using trans-theoretical model (TTM).

3.6.1 Questionnaire

3.6.1.1 Demographic and economic information

The participants' demographic information (age, gender, marital status, religious denomination, level of education, residence, occupation, ethnicity) and family net monthly income were gathered during the baseline assessment.

3.6.1.2 History of chronic diseases

Disease profiles including history of high BP, diabetes, dyslipidemia and other chronic diseases were collected during the baseline assessment.

3.6.1.3 Anthropometric, clinical and biochemical measurements

3.6.1.3.1 Anthropometric measurements and procedures

Anthropometric parameters including body weight, height, waist circumference (WC), and hip circumference (HC) were measured at baseline, midline, and end-line using standard protocols and techniques. A calibrated, Sohenle mechanical weighing machine was used to measure respondents' weight in light clothes. The participant's height in meters was measured using a standard stadiometer, while they were standing upright on a flat surface without shoes. Then, BMI was determined as a ratio of body weight (kgs) to height in meters squared (WHO, 2008-b). WC was measured at the centre between the lower margin of the last palpable rib and the top of the iliac crest using a flexible measuring tape (WHO, 1995). As a component of MetS, WC measurement ≥ 94 cm in men and ≥80 cm in women was considered as abdominal obesity (Alberti et al., 2009). Further, using the WHO (2008-b) classification, men with WC ≥102, 94-101.9, and < 94 cm were classified as having obesity, overweight and normal weight, respectively. Whereas, women with measurements of WC ≥88, 80-87.9, and <80 cm were considered as having obesity, overweight and normal weight, respectively. Using a flexible tape measure, the participants' HC was measured at the largest circumference of the buttocks (WHO, 2010-e). Then, waist/hip ratio (WHR) was determined as the ratio of WC to HC. WHR ≥1.0, 0.90-0.99, and <0.90 for men were classified as obese, overweight, and normal weight, respectively. While WHR ≥0.85, 0.80-0.84, and <0.80 for women, was classified as obese, overweight, and normal weight, respectively.

3.6.1.3.2 Blood pressure (BP) and heart rate measurements and procedures

Each respondent was invited into a room, allowed to be seated for 5-10 minutes to reduce restlessness and anxiety. The blood pressure was measured on the right arm, while the person was in a sitting position using an OMRON automatic blood pressure device (Model: M3; HEM-141-E, Serial. No: 20170916247VG, Japan). This was measured twice at 5 minutes intervals and the mean of the two readings was recorded. As an element of MetS, systolic BP ≥130 mmHg and/or diastolic BP ≥ 85 mmHg was considered as elevated (Alberti et al., 2009). Subjects whose BP values below 130/80 mmHg, but reported taking antihypertensive drugs for at least two weeks before the baseline survey were considered as having hypertension (Chobanian et al., 2003). Measurement of systolic BP ≥ 140 mmHg and/or diastolic BP ≥ 90 mmHg was considered as hypertension. As regards heart rate, it was measured by the BP monitor.

3.6.1.3.3. Biochemical measurements

A sample of three milliliters of blood was collected after at least 8-hours of overnight fasting or 8 hours after the last meal and analyzed by two qualified laboratory technicians. This blood sample was obtained from the brachial vein of the participants by adhering to infection prevention measures. Each sample of the blood was labeled with a specific participant number and used to establish levels of TGs and HDL-C. The blood sample was centrifuged for 5 minutes to obtain enough serum thereafter analysis was done using the Cobas Integra method. As components of MetS, elevated TGs was considered as ≥ 1.7 mmol/L regardless of gender. While reduced HDL-C was considered as <1.03 mmol/L in males and <1.29 mmol/L in females (Alberti *et al.,* 2009). Additionally, FBG level was extracted from the participants' fingers and determined using HemoCue® B-Glucose photometer (1995). As a component of MetS, raised FBG level was defined as FBG ≥5.6 mmol/L (Alberti *et al.*, 2009). Whereas, FBG levels of 5.6 - 6.9 mmol/L and ≥7mmol/L were considered as pre-diabetes and diabetes, respectively (WHO, 2006).

3.6.1.4 Assessment of dietary intake patterns

The respondents' dietary intake pattern was assessed before and after the intervention using a standard dietary questionnaire adopted from the WHO-steps approach to NCDs risk assessment (WHO, 2017-c). The respondents were asked to estimate the frequency and the quantity they consumed particular food products by making comparisons with specified reference portions including plates, cups, and spoons. Dietary intake patterns included frequency consumption of processed/fast foods (eg. chips, sandwiches, fried chicken, sausages, samosas, etc…), cereals, daily servings of fruit and vegetables, legumes, nuts, quantity of sugars, and salts intake were captured.

Common food measurements such as plates, cups, and spoons were presented to help the participants estimate portion of each type of food. Specifically, the DASH eating plan was used to evaluate the proportion of carbohydrates (e.g. bread, chapatti, rice, ugali, maize, potatoes, pasta), protein (e.g. meat, eggs, whole milk, and beans), and vegetables and/or fruits consumption. Half of the DASH plate should be filled with vegetables and/or fruits. One-quarter of the plate is filled with plant-based proteins like legumes, nuts, and seeds proteins and the remaining one-quarter is filled with carbohydrates.

3.6.1.5 Assessement of alcohol consumption and smoking

Assessment of current alcohol intake included determination of frequency and amount (number of standard drinks per day). First, the respondents were asked whether they drink any type of alcohol. The frequency of alcohol intake was assessed by asking: "in the past, 30 days, 1 week, how often did you drink any alcoholic beverages?" The responses were grouped into four categories (daily, 4-6 days/week, 1-3 days/week, and less than 1 day/week). The amount was assessed by asking: "on those days when you took alcoholic beverages, on average, how many standard drinks (SDs) did you take?" The responses were categorized into three (1, 2, and more than 2 drinks in a day). Men who consumed more than two drinks and women more than one drink per day were classified as drinking in excess relative to WHO (2009-b) dietary recommendation. As regards cigarette smoking, it was determined by considering the past and current smoking history, duration, frequency, and the number of cigarettes smoked per day/week.

3.6.1.6 Assessments of Physical Activity

Pre-and post-intervention level of physical activity was evaluated using the WHO-steps approach to NCDs risk factor assessment questionnaire (WHO, 2017-c). Physical activities including work-related moderate and vigorous-intensity activities, leisure time (e.g. swimming, walking, dancing, etc.), planned exercise, sports, transportation (e.g. walking), activities in the house (e.g. washing) in a typical week were captured. To prevent CVDs, it is recommended by the WHO for adults (18–64 years old) to engage in physical activity for a minimum of 150 minutes a week of moderate-intensity or 75 minutes a week of vigorous-intensity or an equivalent combination of both (WHO, 2011-d).

The respondents were asked frequency (number of days in a week), duration (minutes/day), and intensity (light, moderate or vigorous) of physical activity they engaged in. They were asked the number of days in a week they engaged in moderate-intensity physical activity for at least 30 minutes. The responses in days were: (1) I did not engage in any form of physical activity, (2) 1-2, (3) 3-4, (4) ≥ 5. Furthermore, they were asked, in a week, how many days do you do vigorous-intensity physical activities for at least 25 minutes including work-related activities? The responses in days were: (1) I did not do any vigorous activity, (2) 1-2, (3) 3-4, (4) ≥ 5 days. Using the WHO Global Physical Activity Questionnaire scoring criteria (WHO, 2002-b), the responses were converted to Metabolic Equivalent Task (MET) minutes per week. The total minutes over 7 days spent on vigorous, moderate, and light intensity activity were multiplied by 8.0, 4.0, and 3.3, respectively, to obtain MET scores for each activity level. Hence, the respondents' level of physical activity was expressed as MET-minutes per week by multiplying the duration (minutes) and frequency (days/week) of each activity. The recommended MET-minutes per week is ≥600.

3.6.1.7 Assessment of participants' knowledge of on risks and preventive measures CVDs

Respondents' level of knowledge of the main risk factors and control measures of MetS-CVDs was assessed before and after the intervention. This was collected using both close and open-ended questions. The participants level of knowledge of MetS-CVDs-related risk factors was assessed using seven (7) variables, including knowledge on risk factors of type-2 diabetes, high BP and MetS-CVDs related health consequences of tobacco smoking, excessive alcohol consumption, taking excessive salt and sugar, eating junk foods and being overweight/obese. Additionally, knowledge of the respondents about MetS-CVDs-related preventive measures was assessed using 6 variables. The variables include knowledge on whether hypertension and diabetes are preventable, preventive measures of hypertension and diabetes, the daily recommended amount of salt and sugar intake per person, importance of fruits and vegetables consumption towards MetS-CVDs prevention, and benefits of engaging in physical activities towards MetS-CVDs prevention.

Scoring system of participants' knowledge

Respondents who answered at least three correct responses for each of the MetS-CVDs risk factors and preventive measures, scored "**three**", those respondents who identified two correct responses, scored "**two**" and respondents who identified only one correct response, or incomplete or incorrect answer, scored "**one**". The scores for each response were added to determine the total knowledge score. Then, the total score was expressed in percentages. Scores of ≥ 75%, 50-74%, and < 50% were considered as high, moderate, and low levels of knowledge, respectively, (**appendix IV**).

3.6.1.8 Assessment of respondets' lifestyle modification using the TTM of behaviour change

The three main constructs of the trans-theoretical model (TTM) including self-efficacy, pros and cons of decisional balance and stages of change towards a healthy lifestyle practice were evaluated before and after the intervention.

Assessment of respondents' stage of chang in relation to lifestyle modification

The respondents' stage of change was assessed using five multiple-choice questions adopted from the literature on Fruits and vegetable stage of change developed by Ma *et al.* (2002) and stage of change for exercise by Marcus *et al.* (1992). Respondents' stage of change was determined using nine (9) MetS-CVDs risk factors and preventive measures included consumption of fruits, vegetables, processed/fast food, salt, sugar, adherence to the DASH diet, alcohol, tobacco smoking, and physical activity. The participants were asked to select one of five statements that best represented their current intentions to change their dietary patterns, alcohol intake, tobacco smoking, and physical activity. The scores of the 5-item stages of change were rated as follows: (pre-contemplation=1, contemplation=2, preparation=3, action=4, maintenance=5). Then the first three (pre-contemplation, contemplation, and preparation) were grouped as a pre-action stage.

To determine the overall level of adherence towards a healthy lifestyle, their stage of change was converted into scores. Respondents under the pre-action stage, scored "**1**", those who were under the action stage scored "**2**" and those respondents who were under the maintenance stage, scored "**3**". Hence, the maximum score of the stage of change was 27. After converting the total score into a percentage, respondents' overall level of adherence towards a healthy lifestyle was determined as low, moderate, and high (**appendix iv**).

Assessment of respondents' self-efficacy regarding lifestyle modification

The respondents' self-efficacy regarding dietary intake patterns, alcohol intake, smoking, and physical activity was assessed using nine variables both at baseline and end-line. The respondents were evaluated for each variable using a 3-point Likert scale. For each of the nine variables, they were asked to rate their level of confidence on a scale of 1-3 to practice the recommended healthy lifestyle. They were asked "How confident are you that you can make this change, on a scale of 1 to 3, with 1 will be equal to "not at all confident, 2 for somewhat confident, and 3 being extremely confident?" Therefore, the maximum score of self-efficacy was 27.

Assessment of pros and cons of decisional balance regarding lifestyle modification

The respondents' decisional balance regarding lifestyle modification was evaluated using a standard questionnaire developed by Nigg *et al.* (1998). This was assessed using 26 items, 15 pros to assess perceived advantages, and 11 cons to assess barriers to follow a healthy lifestyle. This was assessed by asking the respondents how important each of the listed pros and cons in their decision to follow a healthy lifestyle. The respondents rated each pros and cons item using a 3-point Likert scale. The scores of pros were grouped into three categories (disagree = 1, somewhat = 2, and agree = 3). Similarly, the scores of cons were 1 for disagree, 2 for somewhat, and 3 for agree. Thus, the maximum score of pros and cons were 45 and 33, respectively.

3. 7 Validity and reliability of the study tool

The WHO-steps approach to NCDs behavioural risk assessment questionnaire (WHO, 2017-c) was utilized to gather the data. The validity of the tools in terms of content was revised by experts in the field of nutrition and CVDs, and their recommendations were included in the questionnaire. The BP device used in this study was frequently counterchecked with another device from the hospital to check its validity. To measure the reliability of the questionnaire, a test-re-test technique was carried out after three weeks. The Cohen's kappa coefficient was employed to determine the degree of agreement between the two results. The repeated questions produced a 0.91 kappa value which was considered reliable.

Pretesting of the study tools: A pilot study was carried out on 5% (n = 18) adults to assess the clarity and objectivity of the tools as well as to estimate the time required to complete the questionnaire. The tools were pre-tested at Mbagathi hospital, which serves a similar population as the St. Mary’s Mission Hospital. Both the institutions provide health services to the low-income residents of Kibera, Mukuru, Njenga, Kuwinda, etc. The pretested data were analyzed and then the necessary amendment of the questionnaire was done.

3.8 Ethical consideration

The study was conducted in line with the principles of Helsinki guidelines (World Medical Association, 2013). Ethically, it was approved by the University of Nairobi-Kenyatta National Hospital Ethical Review Committee (Approval number: P430.07/2017). Further, a permit to conduct this research was obtained from the National Commission for Science, Technology and Innovation (NACOSTI) (Permit No. NACOSTI/P/18/09156/22152). The institutional permission was granted by the administration of the St. Mary's Mission Hospital. Written and verbal consent was obtained from each of the participants before data collection (Appendix I). They were informed that their involvement in the study was wholly voluntary, had the right to ask any question, and terminate their participation in the study at any time. They were also informed about the objective of the research; the time they spend during the interview, benefits and risk of their participation.

Privacy was maintained during the interview, physical, clinical, and biochemical measurements. Apart from the mild pain and discomfort while drawing blood samples, no other risks or distress was expected to be encountered. There may be a very slight risk of infection at the puncture site. However, aseptic techniques were applied during blood collection to prevent any risk of infection. The results were communicated to all the participants. Individuals who had high BP (BP ≥140/90 mmHg) and/or diabetes (FBG ≥ 7.0 mmol/L), but did not aware of their status were referred to the hypertensive-diabetic clinic of the St. Mary hospital for further evaluations and follow up. All the questionnaires were kept in a cabinet that is accessible by the principal investigator only. The data were entered in a password-protected computer. Further, all the gathered data were kept confidential to protect the participants' privacy.

3.9 Data analysis

After coding, the data were analyzed using computer software, (SPSS V. 22). Frequencies and proportions were obtained for categorical variables, whereas, means and standard deviations were expressed for continuous variables. The chi-square test of independence and binary logistic regression analyses were used to establish significant differences between group prevalence rates for categorical variables. For continuous variables, the paired t-test was employed to determine within-group differences (after–before), while, the between-group difference in these variables was performed using the t-test for independent samples, assuming either equal or unequal variances.

Difference-in-difference analysis was performed to establish the effect of the intervention on MetS and its components between the two groups at baseline versus end-line. The changes (baseline to end-line) in the dependent variables in the intervention arm were compared to changes in the control arm. The intervention effect was estimated as the average difference between the two groups by determining an Absolute Risk Reduction. The intervention effect was estimated as the average difference between the two groups by determining an Absolute Risk Reduction. Then, the number needed to treat was calculated to determine the number of MetS cases to successfully treat one case of it. A multiple logistic regression model with *backward conditional* was performed to determine the relationship between changes in MetS as a dependent variable and changes in lifestyle as independent variables, after adjusting for age, sex, and education level. A p-value of <0.05 was considered statistically significant.

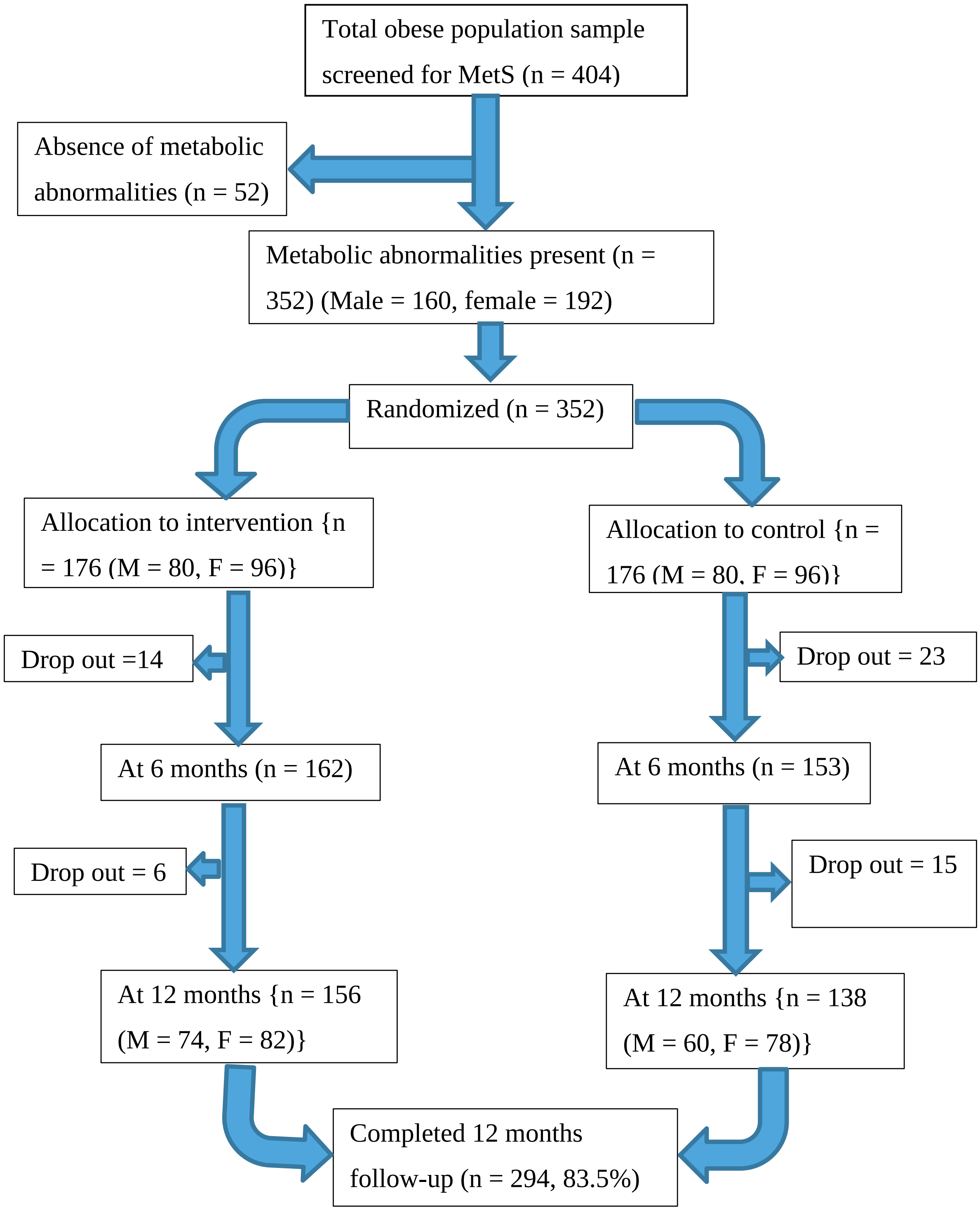


Figure 6. Flow chart showing allocation and enrolment of the subjects to the groups

CHAPTER FOUR: RESULTS

4. 1 Socio-demographic information of the study subjects

A total of 352 (176 in each group) adults with MetS were randomly enrolled in the study, of which 294 (intervention group = 156 and control group = 138) completed the entire 15-months study period, a response rate of 83.5%. Thus, after excluding dropouts, the data for 294 subjects were used for the final analysis (Figure 1). The mean age for the intervention and control groups was 44.2 (±10.6) and 44.5 (±10.7) years, respectively. Most of the respondents were females (54.4%), married (77.9%), and belonged to Christian protestant faith (60.2%). About half (46.6%) of the participants had attained a secondary level of education, were self-employed (52.7%), and reported a family income of 100–500 USD (75.6%) per month. Further analyses revealed that there was no substantial difference between the two groups in terms of the socio-demographic characteristics at the start of the study (Table 3).

Table 3: Baseline socio-demographic information of the respondents by group (n, %)

Characteristic	Intervention	Control	Total	Chi ($\chi 2$)	df	p-value
Mean age (SD)	44.2 (10.6)	44.5 (10.7)	44.3 (10.6)	0.235	292	0.814*
Age group in years				0.008	2	0.996
Less than 30	14 (9.0)	12 (8.7)	26 (8.8)			
30 - 50	99 (63.5)	88 (63.8)	187 (63.6)			
Over 50	43 (27.6)	38 (27.5)	81 (27.6)			
Total	156 (100)	138 (100)	294 (100)			
Sex				0.462	1	0.497
Male	74 (47.4)	60 (43.5)	134 (45.6)			
Female	82 (52.6)	78 (56.5)	160 (54.4)			
Total	156 (100)	138 (100)	294 (100)			
Marital status				0.096	2	0.953
Married	121 (77.6)	108 (78.3)	229 (77.9)			
Single	21 (13.5)	19(13.8)	40(13.6)			
Divorced/separated/widowed	14 (9.0)	11 (8.0)	25 (8.5)			
Total	156(100)	138(100)	294(100)			
Religion				1.104	2	0.576
Protestant	90 (57.7)	87(63.0)	177 (60.2)			
Catholic	52(33.3)	42(30.4)	94 (32.0)			
Muslim	14 (9.0)	9 (6.5)	23 (7.8)			
Total	156(100)	138(100)	294(100)			
Education				0.875	2	0.646
None - primary level	31(19.9)	33(23.9)	64 (21.8)			
Secondary level	76 (48.7)	61(44.2)	137(46.6)			
Tertiary level	49 (31.4)	44 (31.9)	93(31.6)			
Total	156(100)	138(100)	294(100)			
Employment status				1.572	3	0.666
Government employee	7(4.5)	5(3.6)	12(4.1)			
Non- government employee	47 (30.1)	34(24.6)	81(27.6)			
Self- employed	80 (51.3)	75(54.3)	155(52.7)			
Unemployed	22 (14.1)	24(17.4)	46(15.6)			
Total	156(100)	138(100)	294(100)			
Income (USD)				2.226	2	0.329
Less than 100	9 (6.0)	14(10.9)	23(8.2)			
100 - 500	117 (78.0)	94(72.9)	211(75.6)			
Over 500	24 (16.0)	21 (16.3)	45 (16.1)			
Total	150 (100)	129 (100)	279 (100)			

4. 2. Baseline anthropometric, clinical and biochemical data across the groups

Table 4 presents physical, clinical and biochemical measurements of the participants at the baseline. There was no considerable difference between the groups, both for anthropometrics, clinical and biochemical profiles at the baseline. Analysis with a t-test of independence showed that there was no noticeable difference between the two groups for body weight (t = 0.660; p = 0.510), BMI (t = 0.516; p = 0.606), WC (t = 0.668; p = 0.505), HC (t = 0. 749; p = 0.454), WHR (t = 1.327; p = 0.185), WHtR (t = 0.746; p = 0.457), SBP (t = 0.018; p = 0.986), DBP (t = 0.343; p = 0.732), heart rate (t = 1.440; p = 0.151), FBG level (t = 0.600; p = 0.549), TGs (t = 0.308; p = 0.758) and HDL-C (t = 0.172; p = 0.864).

Table 4: Baseline anthropometric, clinical and biochemical data across the groups (mean ± SD)

Measurements	Group	Mean	SD	t-test	df	p
Weight (kg)	Control	85.55	14.04	0.660	292	0.510
	Intervention	86.56	12.13			
Height (cm)	Control	167.48	7.96	0.383	292	0.702
	Intervention	167.83	7.91			
BMI (kg/m2)	Control	30.49	4.53	0.516	292	0.606
	Intervention	30.75	3.92			
WC (cm)	Control	101.45	11.26	0.668	292	0.505
	Intervention	100.63	9.90			
HC (cm)	Control	109.38	9.33	0.749	292	0.454
	Intervention	110.16	8.60			
WHR	Control	0.93	0.09	1.327	292	0.185
	Intervention	0.92	0.09			
WHtR	Control	0.61	0.06	0.746	292	0.457
	Intervention	0.60	0.06			
SBP (mmHg)	Control	135.88	18.60	0.018	292	0.986
	Intervention	135.92	19.04			
DBP (mmHg)	Control	86.35	10.29	0.343	292	0.732
	Intervention	85.89	12.30			
Heart Rate	Control	77.12	11.77	1.440	292	0.151
	Intervention	79.22	13.01			
FBG (mmol/L)	Control	5.05	0.99	0.600	292	0.549
	Intervention	5.12	1.06			
TGs (mmol/L)	Control	2.12	0.92	0.308	292	0.758
	Intervention	2.16	1.06			
HDL-C (mmol/L)	Control	1.03	0.28	0.172	292	0.864
	Intervention	1.02	0.29			

4. 3 Changes in metabolic syndrome over the study period across the groups

Figure 7 indicates changes in the proportion of MetS at different points of time across the groups. Before the intervention, all the respondents had MetS. Generally, there was a decrease in the rate of MetS in both groups at six and twelve months. However, MetS was substantially ($p < 0.001$) less prevalent in the intervention group (IG) both at midline (57.1% vs 78.3%) and endline (54.5% vs 84.1%) relative to the control group (CG). The decline in the proportion of MetS in the IG was 45.5% compared to 15.9% in the CG at the endline, translating to an Absolute Risk Reduction of 29.6%. The number required to treat to control 1 case of MetS was 3.4. This shows that one in three adults with MetS exposed to a community-based lifestyle intervention experienced improvement of their condition (figure 7).

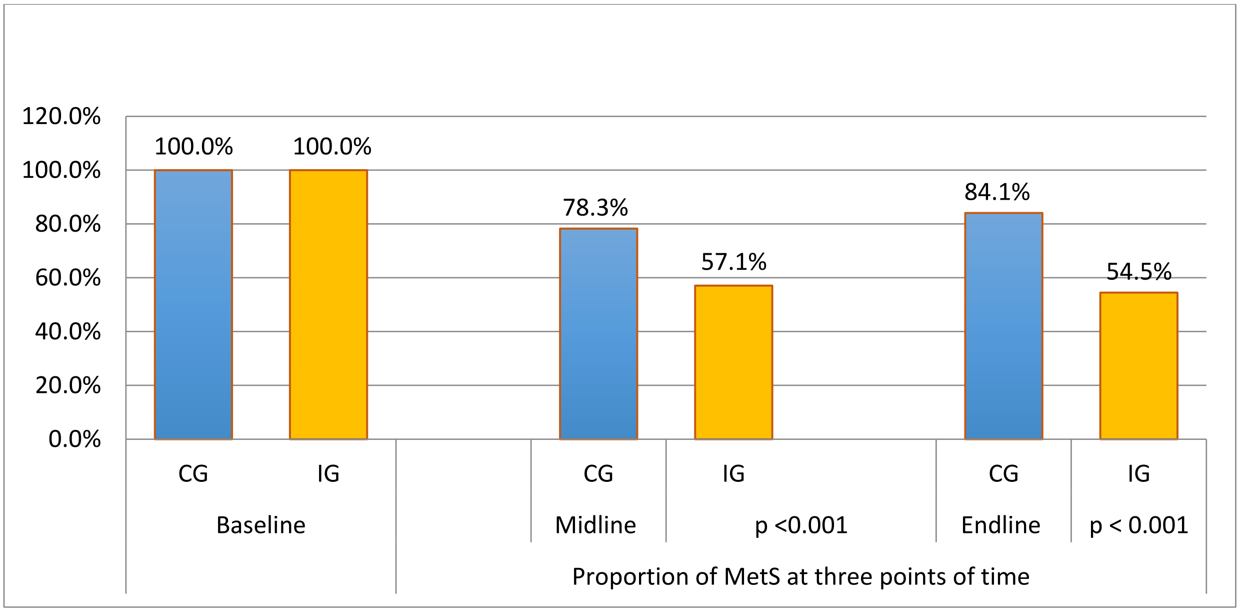


Figure 7. Changes in metabolic syndrome over the study period across the groups

4.4 Changes in central obesity between the groups over the study period

Figure 8 indicates changes in the proportion of central obesity at different points of time across the groups. Before the intervention, all the respondents had central obesity. There was a significant ($p < 0.05$) decrease in the rate of central obesity in the intervention group at midline and endline relative to the control group. The decline in the proportion of central obesity in the intervention group was 7.7% and 9.6% compared to 2.2% and 2.2% in the controls at the midline and end-line, respectively. This translates to an Absolute Risk Reduction of 7.4% in the prevalence of central obesity at the endline.

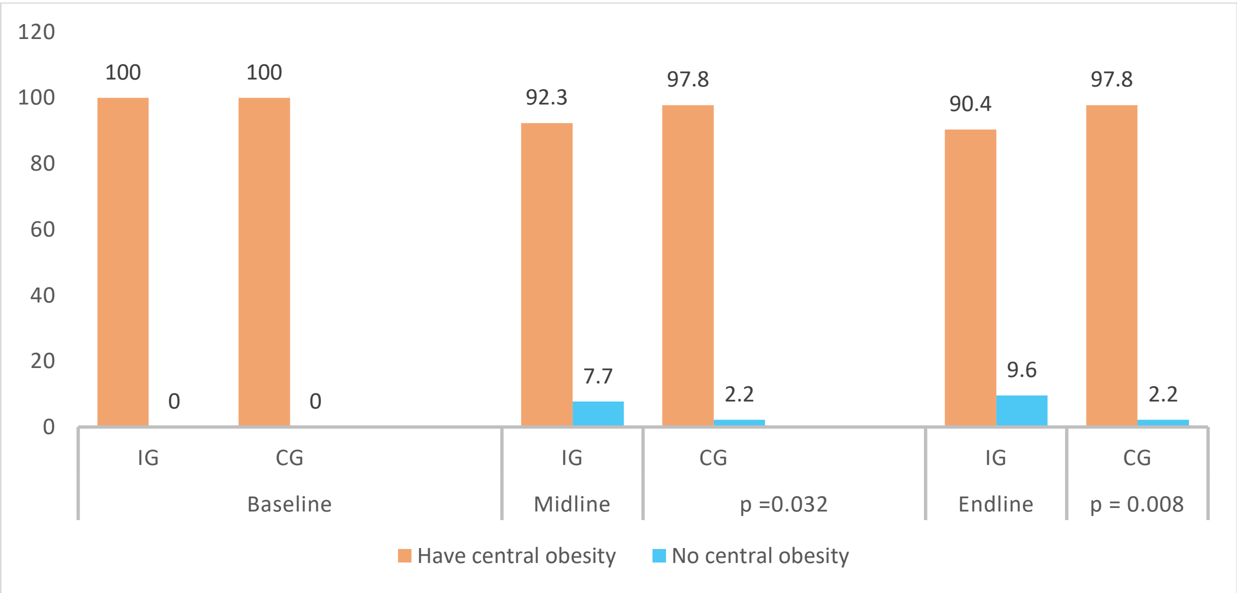


Figure 8. Changes in Central obesity over the study period across the groups (%)

4.5 Changes in blood pressure over the study period across the groups

Figure 9 shows changes in the proportion of elevated BP during the study period across the groups. Before the intervention, most (IG = 69.2%, CG = 76.1%) of the respondents had elevated BP. The proportion of elevated BP in the intervention group markedly ($p < 0.05$) reduced by 15.4% and 16% at the midline and end-line compared to 8.7% and 5.8% in the control group, respectively. This results in an Absolute Risk Reduction of 10.2% in the prevalence of elevated BP at the end-line.

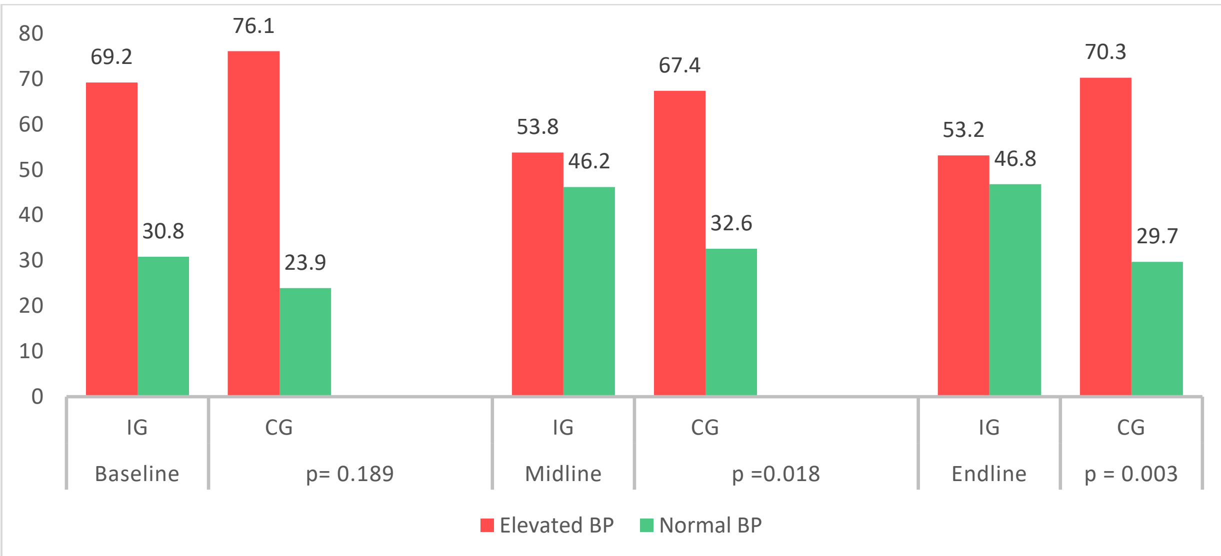


Figure 9. Changes in blood pressure over the study period across the groups (%)

4.6 Changes in raised FBG level over the study period across the groups

The changes in the proportion of raised FBG level during the study period is presents in figure 10. Before the intervention, approximately a quarter (IG = 28.2%, CG = 26.8%) of the respondents had raised FBG levels. The proportion of raised FBG level in the intervention group markedly (p = 0.001) reduced by 10.3%, while in the control group, it worsened by 8.7% at the end-line. This results in an Absolute Risk Reduction of 19% in the prevalence of raised FBG levels at the end-line.

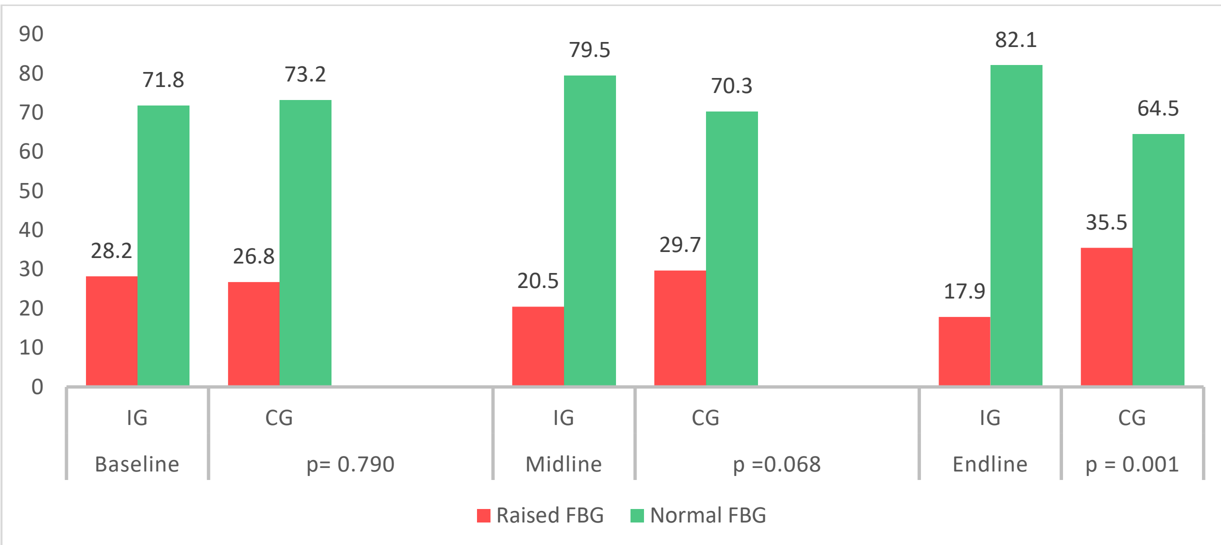


Figure 10. Changes in raised FBG level over the study period across the groups (%)

4.7 Changes in Triglycerides level over the study period across the groups

The changes in the proportion of raised TGs level during the study period is displayed in figure 11. Majority, (IG = 71.8%, CG = 71.7%) of the respondents had raised TGs level at the baseline. There was a substantial ($p < 0.05$) reduction in the proportion of raised TGs at the midline (22.4%) and end-line (26.3%) in the intervention compared to 4.3% and 9.4% in the control group, respectively. This produced an Absolute Risk Reduction of 16.9% in the prevalence of raised TGs levels at the end-line.

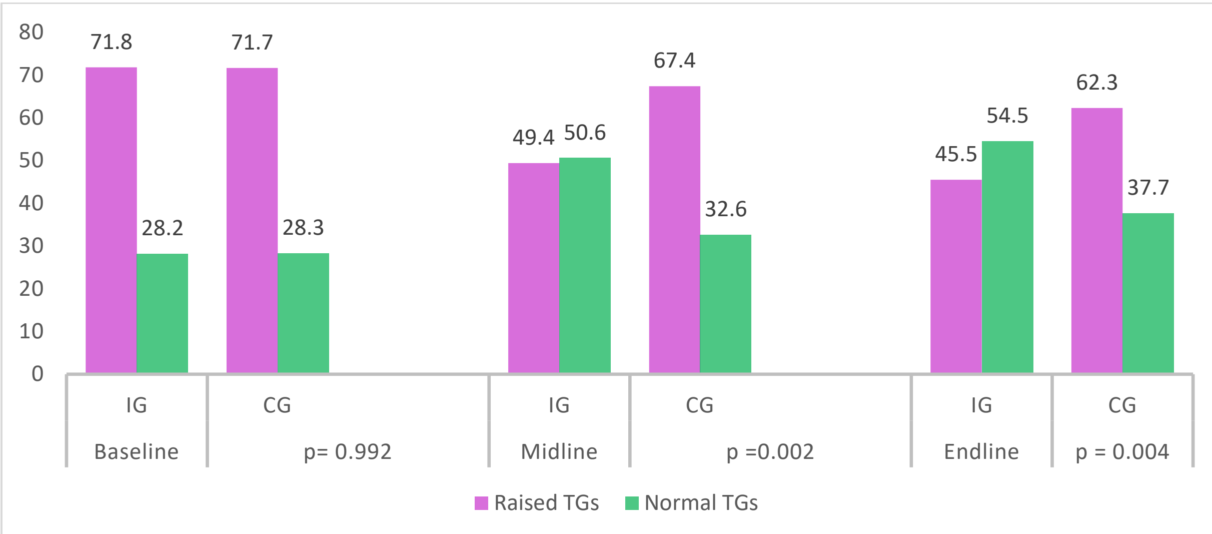


Figure 11. Changes in Triglycerides level over the study period across the groups (%)

4.8 Changes in HDL-C level over the study period across the groups

Figure 12 shows changes in the proportion of reduced high-density lipoprotein cholesterol (HDL-C) during the study period across the groups. Before the intervention, most, (IG = 79.5%, CG = 79%) of the respondents had a low level of HDL-C. There was a significant ($p < 0.05$) decrease in the proportion of reduced HDL-C at the midline (29.7%) and end-line (32.7%) in the intervention compared to 10.9% and 6.5% in the control group, respectively. This results in an absolute risk reduction of 26.2% in the prevalence of reduced HDL-C at the end-line.

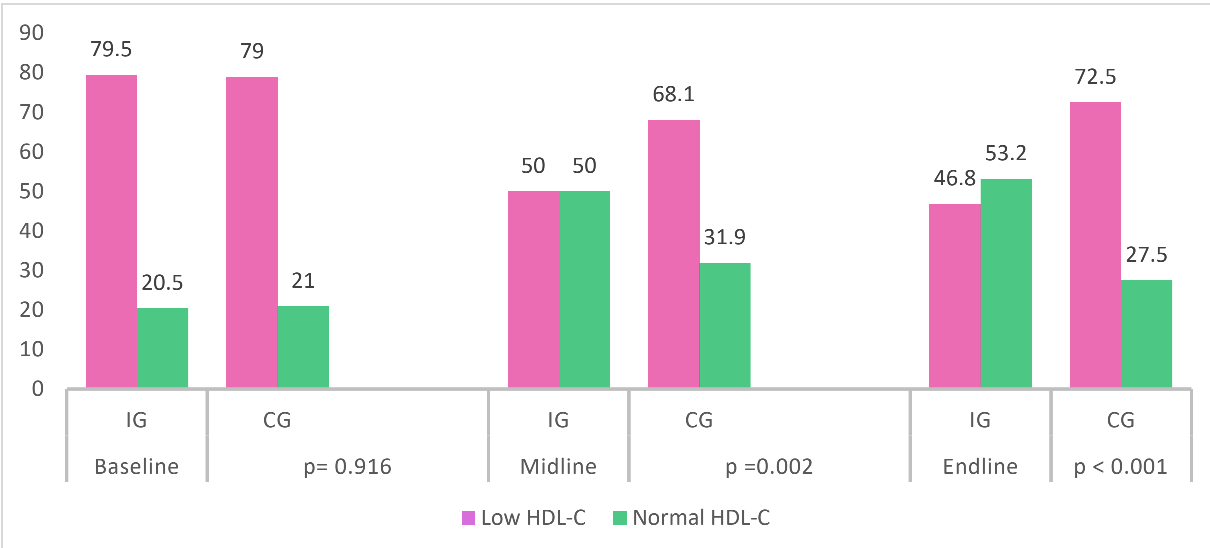


Figure 12. Changes in HDL-C level over the study period across the groups (%)

4.9 Changes in number of the components of MetS during the study period

Figure 13 displays the proportion of respondents with a different number of components of MetS during the study period. At baseline, most (CG = 56.5%, IG = 60.3%) of the respondents had three, while about one-third (CG = 33.3%, IG = 30.8%) had four, and few (10%) had five out of the five components of MetS, respectively. However, at the end-line, the respondents with three components of MetS had significantly (p< 0.001) declined in the IG (25.6%) compared to the CG (42%). Further, the percentage of people who had all the five components for MetS was lower in the IG (8.3% vs 17.4%) compared to the CG.

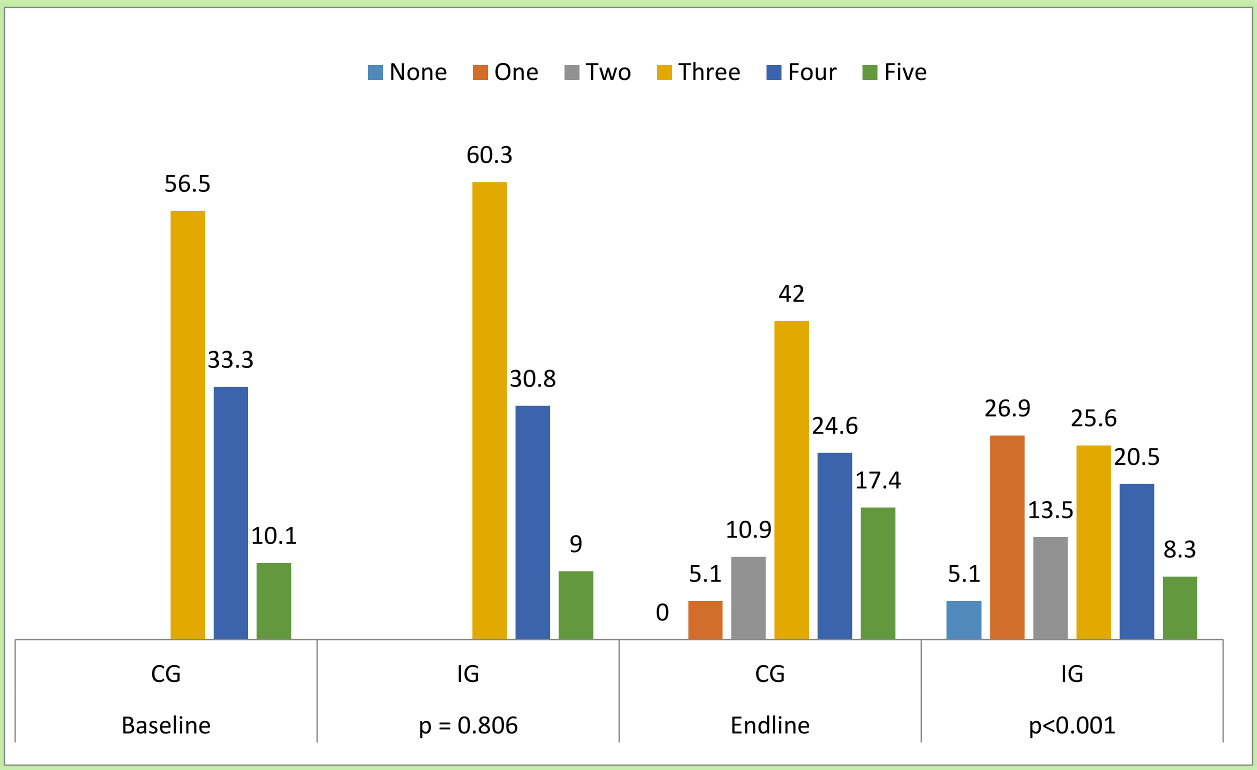


Figure 13. Changes in the number of components for MetS across study groups (%)

4.10 Changes in physical, clinical and biochemical measurements after the intervention across the groups

There was no mean difference between the two groups for anthropometric, clinical, and biochemical profiles at the baseline (Table 5). However, analysis with an independent t-test revealed that most of these measurements were significantly lower by midline and end-line, in the IG compared to CG. For example, at the end-line, measurements of body weight (81.96 vs 85.15 kgs), BMI (29.15 vs 30.35), WC (98.34 vs 101.33 cm), WHR (0.90 vs 0.93), WHtR (0.59 vs 0.61), SBP (131.81 vs 136.12 mm Hg), DBP (83.49 vs 86.12mm Hg), FBG (4.62 vs 5.13 mmol/l) and TGs (1.83 vs 2.11mmol/l) were substantially ($p < 0.05$) lower in the IG relative to the CG. Moreover, the level of HDL-C was substantially ($p < 0.001$ higher in the IG (1.27 mmol/l) when compared to the CG (1.05 mmol/l) at the endline (Table 5).

Table 5. Changes in physical, clinical and biochemical measurements after the intervention across the groups

Measurements	Groups	Midline			End-line		
		Mean (±SD)	t-test	P	Mean (±SD)	t-test	P
Weight (kg)	Control	85.58(13.69)	2.231	0.026	85.15(13.65)	2.230	0.027
	Intervention	82.32(11.30)			81.96(10.880		
BMI (kg/m2)	Control	30.50(4.40)	2.583	0.010	30.35(4.41)	2.532	0.012
	Intervention	29.27(3.82)			29.15(3.76)		
WC (cm)	Control	100. 82(11.28)	1.642	0.102	101.33(11.53)	2.359	0.019
	Intervention	98.77(10.09)			98.34(10.25)		
HC (cm)	Control	109.29(9.27)	0.125	0.901	109.49(9.18)	0.476	0.634
	Intervention	109.16(8.52)			108.99(8.53)		
WHR	Control	0.92(0.09)	1.725	0.086	0.93(0.09)	2.967	0.003
	Intervention	0.91(0.081)			0.90(0.08)		
WHtR	Control	0.60(0.061)	1.732	0.084	0.61(0.061)	2.456	0.015
	Intervention	0.59(0.064)			0.59(0.064)		
SBP (mmHg)	Control	136.04(16.98)	1.936	0.054	136.12(18.03)	2.255	0.025
	Intervention	132.40 (15.26)			131.81(14.71)		
DBP (mmHg)	Control	86.43(10.08)	2.356	0.019	86.12(11.08)	2.138	0.033
	Intervention	83.65(10.16)			83.49(10.05)		
FBG (mmol/L)	Control	5.17(0.83)	3.022	0.003	5.13(0.88)	4.467	0.000
	Intervention	4.86(0.91)			4.62(1.04)		
TGs (mmol/L)	Control	2.16(0.95)	2.596	0.010	2.11(0.98)	2.784	0.006
	Intervention	1.90(0.78)			1.83(0.78)		
HDL-C (mmol/L)	Control	1.08(0.27)	5.570	0.000	1.05(0.28)	6.528	0.000
	Intervention	1.26(0.28)			1.27(0.30)		

WHR = wasit hip ratio, WHtR = waist height ratio, TGs = triglycerides, HDL-C = high density lipoprotein cholesterol

4.11. Comparison of baseline versus endline of the elements of MetS within the groups

Table 6 displays pairwise mean differences between baseline and end-line in the elements of MetS within the groups. The study revealed that the intervention group markedly ($p < 0.001$) reduced mean scores of WC (-2.3 cm), SBP (-4.06 mm Hg), DBP (-2.4 mmHg), heart rate (-2.18 beats per minute), FBG level (-0.5 mmol/l), TGs (-0. 28 mmol/l) and increased HDL-C in male (+0.3 mmol/l) and female (+ 0.2 mmol/l) at the end-line compared to the baseline. However, the control group did not show a significant change in all these parameters at the end-line relative to the baseline. The mean net effects of the intervention of the elements of MetS were: WC (-2.1), SBP (-4.3), DBP (-2.17), HR (-2.13), FBG (-0.58), TGs (-0.23), HDL-C (males +0.27, females +0.18) (Table 6).

Table 6: Comparison of baseline versus endline of the elements of MetS within the groups

Group	Baseline (Mean±SD)	End-line (Mean±SD)	Mean difference	Mean net-effect	Paired t-test	df	p-value
	Waist circumference (cm)						
Intervention	100.63 (9.90)	98.27(10.01)	-2.3	-2.1	9.017	155	0.000
Control	101.45(11.26)	101.33(11.52)	-0.2		0.651	137	0.516
	Systolic blood pressure (mmHg)						
Intervention	135.92 (19.04)	131.86 (14.73)	-4.06	-4.3	4.839	155	0.000
Control	135.88 (18.60)	136.12 (18.03)	+0.24		0.266	137	0.791
	Diastolic blood pressure (mmHg)						
Intervention	85.89 (12.30)	83.49 (10.05)	-2.4	-2.27	4.811	155	0.000
Control	86.35 (10.29)	86.12 (11.08)	-0.23		0.307	137	0.759
	Heart rate (beats per minute)						
Intervention	79.22 (13.01)	77.04 (10.12)	-2.18	-2.13	5.295	155	0.000
Control	77.12 (11.78)	77.07 (9.96)	-0.05		0.091	137	0.928
	Fasting blood glucose (mmol/l)						
Intervention	5.12 (1.05)	4.62 (1.04)	-0.5	-0.58	10.222	155	0.000
Control	5.05 (0.99)	5.13 (0.88)	+0.08		1.514	137	0.132
	Triglycerides (mmol/L)						
Intervention	2.12 (0.92)	1.83 (0.80)	-0.28	-0.23	8.725	155	0.000
Control	2.16 (1.06)	2.11 (0.98)	-0.05		1.013	137	0.313
	HDL-C- Male (mmol/L)						
Intervention	0.98 (0.27)	1.28 (0.29)	+0.3	+0.27	7.402	73	0.000
Control	1.03 (0.33)	1.06 (0.32)	+0.03		1.282	59	0.205
	HDL-C –Female (mmol/L)						
Intervention	1.06 (0.30)	1.26 (0.31)	+0.2	+0.18	7.310	81	0.000
Control	1.02 (0.24)	1.04 (0.24)	+0.02		0.705	77	0.483
Analysis with paired t-test							

4.12 Comparison of baseline vs endline in anthropometric measurements within the groups

Analysis with a paired t-test revealed that there was a significant ($p < 0.001$) reduction in mean scores of body weight (-4.6 kgs), BMI (-1.6), WC in males (-2.85 cm) and females (-1.73 cm), HC in males (-0.62 cm) and female (-0. 66 cm), WHR in male (-0.02) and female (-0.02), WHtR in male (-0.02) and female (-0.01) in the intervention group at the end-line compared to the baseline line. In the control group, there was no significant difference observed in these parameters at the end-line relative to baseline (Table 7).

Table 7: Comparison of baseline vs endline of physical measurements within the groups

Group	Baseline (Mean ± SD)	End-line (Mean ± SD)	Mean-difference	Mean Net-effect	t-test	df	p-value
	Weight (kgs)						
Intervention	86.56 (12.13)	81.96 (10.88)	-4.6	-4.2	13.249	155.000	0.000
Control	85.55 (14.04)	85.15 (13.65)	-0.4		1.521	137.000	0.131
	Body Mass Index						
Intervention	30.75 (3.92)	29.15 (3.76)	-1.6	-1.46	13.380	155.000	0.000
Control	30.49 (4.53)	30.35 (4.41)	-0.14		1.514	137.000	0.132
	Waist circumference (cm) for Male						
Intervention	102.88 (9.99)	100.03 (9.25)	-2.85	-2.69	8.079	73.000	0.000
Control	103.68 (7.87)	103.52 (8.42)	-0.16		0.446	59.000	0.657
	Waist circumference (cm) for female						
Intervention	98.60 (9.43)	96.87 (10.54)	-1.73	-1.64	4.940	81.000	0.000
Control	99.74 (13.09)	99.65 (13.25)	-0.09		0.504	77.000	0.616
	Hip circumference (cm) for Male						
Intervention	107.24 (6.99)	106.62 (6.84)	-0.62	-0.61	7.278	73.000	0.000
Control	106.87 (7.70)	106.88 (7.54)	-0.01		0.145	59.000	0.885
	Hip circumference (cm) for female						
Intervention	112.79 (9.10)	112.13 (9.19)	-0.66	-0.53	6.395	81.000	0.000
Control	111.31 (10.04)	111.18 (9.92)	-0.13		1.043	77.000	0.300
	Waist-Hip ratio for Male						
Intervention	0.96 (0.09)	0.94 (0.09)	-0.02	-0.02	6.541	73.000	0.000
Control	0.97 (0.06)	0.97 (0.06)	0.0		0.143	59.000	0.886
	Waist-Hip ratio for Female						
Intervention	0.88 (0.06)	0.86 (0.07)	-0.02	-0.02	3.876	81.000	0.000
Control	0.90 (0.10)	0.90 (0.10)	0.0		1.303	77.000	0.196
	Waist-Height ratio for Male						
Intervention	0.60 (0.07)	0.58 (0.06)	-0.02	-0.02	7.910	73.000	0.000
Control	0.60 (0.04)	0.60 (0.04)	0.0		0.375	59.000	0.709
	Waist-Height ratio for female						
Intervention	0.60 (0.06)	0.59 (0.06)	-0.01	-0.01	4.936	81.000	0.000
Control	0.61 (0.07)	0.61 (0.07)	0.0		0.548	77.000	0.585

4.13. Utilization of the DASH diet before and after the intervention beween the groups

Adherence to the DASH diet was very low in both groups at the baseline. A majority of the respondents consumed below the recommended portion of a plate as vegetables/fruits, but higher portions as protein and carbohydrate foods, at the baseline. Adherence to the DASH diet was significantly improved in the IG relative to the CG at the endline. The proportion of people who consumed the recommended portions of a plate as vegetables/fruits (42.9% vs 16.7%) was significantly ($p < 0.001$) higher in the intervention relative to the control group (Table 8).

Table 8: Adherence to the DASH diet during the study period between the groups (n, %)

Group	Baseline: proportion of meal plates filled with protein foods				Chi (χ2)	df	p-value
	≤ 25%	26-49%	≥50%	Total			
IG	18(11.5)	85(54.5)	53(34.0)	156(100)	0.652	2	0.722*
CG	12 (8.7)	77(55.8)	49(35.5)	138(100)			
Total	30(10.2)	162(55.1)	102(34.7)	294(100)			
	End-line: proportion of meal plates filled with protein foods						
	≤ 25%	26-49%	≥50%	Total			
IG	57(36.5)	81 (51.9)	18(11.5)	156(100)	22.715	2	0.000*
CG	23(16.7)	74(53.6)	41(29.7)	138(100)			
Total	80(27.2)	155(52.7)	59(20.1)	294(100)			
	Baseline: proportion of meal plates filled with Carbohydrates						
	≤ 25%	26-49%	≥50%	Total			
IG	3(1.9)	41 (26.3)	112(71.8)	156(100)	4.551	2	0.169**
CG	0(0.0)	28(20.3)	110(79.7)	138(100)			
Total	3 (1.0)	69(23.5)	222(75.5)	294(100)			
	End-line: proportion of meal plates filled with Carbohydrates						
	≤ 25%	26-49%	≥50%				
IG	15(9.6)	69(44.2)	72 (46.2)	156(100)	21.242	2	0.000*
CG	5(3.6)	33(23.9)	100(72.5)	138(100)			
Total	20(6.8)	102(34.7)	172(58.5)	294(100)			
	Baseline: proportion of meal plates filled with vegetables-fruits						
	≤ 25%	26-49%	≥50%	Total			
IG	56(35.9)	70(44.9)	30(19.2)	156(100)	3.971	2	0.113*
CG	47(34.1)	75(54.3)	16(11.6)	138(100)			
Total	103(35.0)	145(49.3)	46(15.6)	294(100)			
	End-line: proportion of meal plates filled with vegetables-fruits						
	≤ 25%	26-49%	≥50%				
IG	21(13.5)	68(43.6)	67(42.9)	156(100)	26.770	2	0.000*
CG	40(29.0)	75(54.3)	23(16.7)	138(100)			
Total	61(20.7)	143(48.6)	90(30.6)	294(100)			

**chi-square test of independence, **Fisher's Exact test, IG = intervention group, CG = control group*

4.15 Fruits and vegetables intake across the groups before and after the intervention

Daily consumption of fruits (IG = 16%, CG = 17.4%) and vegetables (IG=42.3%, CG=39.1%) was low in both groups at baseline. The consumption of fruits (44.2% vs 20.2%) and vegetables (70.5% vs 49.3%) significantly ($p < 0.001$) improved in the intervention relative to the control group at the endline. Further analysis with paired t-test revealed that at the end-line, the intervention group significantly increased mean servings of fruits ($t = 3.535$; $p = 0.001$) and vegetables ($t = 3.313$; $p = 0.001$) consumption relative to the baseline line. However, in the control group, no significant change was noticed in terms of servings of fruits and vegetable intake at the end-line compared to baseline (Table 9).

Table 9: Fruits and vegetables intake across the groups before and after the intervention

Group	**Daily (n, %)**	**Not daily (n, %)**	Total	**Chi** ($\chi 2$)	df	p-value
	Baseline fruits consumption					
Intervention	25(16.0)	131(84.0)	156(100)	0.098	1	0.754
Control	24(17.4)	114(82.6)	138(100)			
Total	49(16.7)	245(83.3)	294(100)			
	End-line fruits consumption					
Intervention	69(44.2)	87 (55.8)	156(100)	18.984	1	0.000
Control	28(20.3)	110(79.7)	138(100)			
Total	197(67.0)	97(33.0)	294(100)			
	Baseline vegetables intake					
Intervention	66(42.3)	90(57.7)	156(100)	0.306	1	0.580
Control	54(39.1)	84(60.9)	138(100)			
Total	174(59.2)	120(40.8)	294(100)			
	End-line vegetables intake					
Intervention	110(70.5)	46(29.5)	156(100)	13.825	1	0.000
Control	68(49.3)	70(50.7)	138(100)			
Total	178(60.5)	116(39.5)	294(100)			
Group	Baseline (mean ±SD)	End-line (mean ±SD)		t-test	df	p-value
	Daily servings of fruits intake					
Intervention	1.32 (0.545)	1.42 (0.557)		3.535	155	0.001*
Control	1.38 (0.608)	1.33 (0.584)		1.351	137	0.179*
	Daily servings of vegetables intake					
Intervention	1.72 (0.680)	1.84(0.617)		3.313	155	0.001*
Control	1.62 (0.653)	1.64(0.714)		0.425	137	0.671*
**Analysis with paired t-test*						

4.16 Consumption of legumes, nuts and processed food before and after the intervention

A small proportion of respondents (IG = 21.2%, CG = 18.8%) and (IG = 16.7%, CG = 13%) often (≥ 5 times/week) consumed legumes and nuts, respectively at baseline. Whereas, the proportion of often consumers of legumes (48.1% vs 23.2%) and nuts (31.4% vs 15.9%) had improved and markedly ($p < 0.001$) higher in the IG than the CG at the end-line. Regarding processed/fast foods intake, about one-third of each group often consumed such foods during baseline. However, the rate of often consumers of processed/fast foods significantly ($p = 0.003$) reduced in the IG (19.2%) compared to the CG (30.4%) at the end line (Table 10).

Table 10: Intake of legumes, nuts and processed food before and after the intervention (n,%)

Groups	**Often**	**Sometimes**	**Rarely/never**	Total	**Chi** ($\chi 2$)	df	p-value
	Baseline frequency of legumes/pulses intake						
IG	33 (21.2)	38(24.4)	85 (54.5)	156 (100)	0.396	2	0.820
CG	26 (18.8)	32 (23.2)	80 (58.0)	138 (100)			
Total	59 (20.1)	70 (23.8)	165 (56.1)	294			
	End-line frequency of legumes/pulses intake						
IG	75(48.1)	50 (32.1)	31 (19.9)	156(100)	30.878	2	0.000
CG	32 (23.2)	39(28.3)	67(48.6)	138(100)			
Total	107(36.4)	89(30.3)	98(33.3)	294(100)			
	Baseline frequency of nuts intake						
IG	26(16.7)	38(24.4)	92(59.0)	156(100)	0.773	2	0.679
CG	18 (13.0)	36(26.1)	84(60.9)	138(100)			
Total	44(15.0)	74(25.2)	176(59.9)	294(100)			
	End-line frequency of nuts intake						
IG	49 (31.4)	75(48.1)	32(20.5)	156(100)	37.886	2	0.000
CG	22 (15.9)	40(29.0)	76(55.1)	138(100)			
Total	71(24.1)	115(39.1)	108(36.7)	294(100)			
	Baseline frequency of eating processed foods						
IG	49(31.4)	57(36.5)	50(32.1)	156(100)	0.293	2	0.864
CG	43(31.2)	47(34.1)	48(34.8)	138(100)			
Total	92(31.3)	104(35.4)	98(33.3)	294(100)			
	End-line frequency of eating processed foods						
IG	30(19.2)	39(25.0)	87(55.8)	156(100)	11.510	2	0.003
CG	42(30.4)	46(33.3)	50(36.2)	138(100)			
Total	72(24.5)	85(28.9)	137(46.6)	294(100)			

4.17. Consumption of salt and sugar before and after the intervention across the groups

Only a third of the participants in each group consumed the recommended amount of salt (≤ 1 teaspoon/day) and sugar (≤ 5 teaspoons/day) at baseline. Whereas, at the end-line, intake of the recommended amount of salt (69.6% vs 50.0%) and sugar (64.7% vs 45.7%) was improved and significantly ($p = 0.001$) higher in the intervention relative to the control group (Table 11A). Further analysis with an independent t-test revealed baseline mean salt and sugar consumption between the two groups was not significantly different. However, analysis with a paired t-test revealed a substantial reduction of the total mean of salt ($t = 5.602$; $p < 0.001$) and sugar ($t = 4.166$; $p < 0.001$) intake in the intervention group at the end-line compared to baseline. Whereas, in the control group, there was no considerable change noticed in terms of total mean salt and sugar intake at the end-line relative to the baseline (Table 11B).

Table 11A: Salt and sugar intake before and after the intervention across the groups (n, %)

Group	**Recommended**	**Not recommended**	Total	Chi (χ2)	df	p-value
	Baseline salt consumption status					
Intervention	49 (31.4)	107 (68.6)	156(100)	0.762	1	0.383
Control	50(36.2)	88 (63.8)	138(100)			
Total	99 (33.7)	195 (66.3)	294(100)			
	End-line salt consumption status					
Intervention	109(69.9)	47(30.1)	156(100)	12.105	1	0.001
Control	69(50.0)	69(50.0)	138(100)			
Total	178(60.5)	116(39.5)	294(100)			
	Baseline sugar consumption status					
Intervention	57(36.5)	99(63.5)	156(100)	0.109	1	0.741
Control	53(38.4)	85(61.6)	138(100)			
Total	110(37.4)	184(62.6)	294(100)			
	End-line sugar consumption status					
Intervention	101(64.7)	55(35.3)	156(100)	10.820	1	0.001
Control	63(45.7)	75(54.3)	138(100)			
Total	164(55.8)	130(44.2)	294(100)			

Table 11B: Salt and sugar intake before and after the intervention (Mean tea spoons ±SD)

Group	Baseline salt intake		Total	t-test	df	p-value
Intervention	1.51(0.78)		156(100)	0.703	292	0.483**
Control	1.57(0.74)		138(100)			
All	1.54(0.759)		294(100)			
	Baseline sugar intake					
Intervention	5.54(4.06)		156(100)	0.369	292	0.713**
Control	5.71(3.54)		138(100)			
All	5.62(3.82)		294(100)			
	Salt intake					
Group	Baseline	End-line		t-test	df	p-value
Intervention	1.51(0.78)	1.16(0.58)	156(100)	5.602	155	0.000*
Control	1.57(0.74)	1.51(0.74)	138(100)	0.887	137	0.377*
All	1.54(0.759)	1.33(0.688)	294(100)	5.116	293	0.000*
	Sugar intake					
	Baseline	End-line				
Intervention	5.54(4.06)	4.40(2.61)	156(100)	4.166	155	0.000*
Control	5.71(3.54)	5.49(3.28)	138(100)	1.898	137	0.060*
All	5.62(3.82)	4.93(2.99)	294(100)	4.511	293	0.000*
***Analyzed with independent t-test, *Analyzed with a paired t-test*						

4.18 Alcohol and tobacco use before and after the intervention across the groups

Approximately one-fifth (IG = 22.4%, CG = 21.0%) were alcohol consumers, of which, 97.1% of the IG and all (100%) of the CG drinking above the recommended amount at baseline. However, at the end-line, those who consumed above the recommended amount of alcohol were higher in the CG (72.4%) than the IG (48.5%) with a borderline significant ($p = 0.055$). Further analysis showed that the intervention group significantly ($p < 0.001$) reduced quantity of alcohol intake at the end-line (mean= 2.97 SDs) compared to the baseline (mean = 4.58 SDs). Whereas, there was no significant change observed in terms of quantity of alcohol intake within the CG at the end-line relative to the baseline. Regarding tobacco use, a small number, at the baseline, (intervention = 4.5%, control = 1.4) and end-line (intervention = 3.8%, control = 1.4) were active smokers with no statistical difference between the groups (Table 12).

Table 12: Alcohol and tobacco use before and after the intervention across the groups

	Baseline alcohol consumption status (n, %)				**Chi (χ2)**	**df**	**p-value**
Group	Yes	No		Total			
Intervention	35(22.4)	121(77.6)		156(100)	0.087	1	0.768*
Control	29(21.0)	109(79.0)		138(100)			
All	64(21.8)	230(78.2)		294(100)			
	Baseline quantity of alcohol consumed (n, %)						
	Recommended	Not recommended		Total			
Intervention	1 (2.9)	34 (97.1))		35(100)	0.842		0.547**
Control	0(0.0)	29 (100)		29 (100)			
Total	1(1.6))	63 (98.4))		64 (100)			
	End-line alcohol consumption status (n, %)						
	Yes	No					
Intervention	33(21.2)	123(78.8)		156(100)	0.001	1	0.977*
Control	29(21.0)	109(79.0)		138(100)			
All	62(21.1)	232(78.9)		294(100)			
	End-line quantity of alcohol consumed (n, %)						
	Recommended	Not recommended		Total			
Intervention	17 (51.5)	16 (48.5)		33 (100)	3.673	1	0.055*
Control	8 (27.6)	21 (72.4)		29 (100)			
Total	25 (40.3)	37 (59.7)		62 (100)			
	Monthly occasions of alcohol intake (mean ±SD)				**t-test**	**df**	**p-value**
	Baseline	End-line					
Intervention	11.61 (3.90)	7.09 (3.16)		33	6.232	32	0.000
Control	10.45 (4.63)	9.51 (3.97)		29	1.553	28	0.132
	Standard drinks per drinking occasion (meal ±SD)						
	Baseline	End-line					
Intervention	4.58 (2.40)	2.97 (0.85)		33	4.128	32	0.000
Control	4.34 (1.99)	4.34 (1.88)		29	0.000	28	1.000
	Baseline smoking status				Chi (χ2)	df	p-value
Group	Current smokers	Past smokers	Never smoked	Total			
Intervention	7(4.5)	14(9.0)	135(86.5)	156 (100)	2.872	-	0.250**
Control	2 (1.4)	17(12.3)	119(86.2)	138(100)			
Total	9(3.1)	31(10.5)	254(86.4)	294(100)			
	End-line smoking status						
	Current smokers	Past smokers	Never smoked				
Intervention	6(3.8)	15(9.6)	135(86.5)	156 (100)	1.151	-	0.596**
Control	2(1.4)	17(12.3)	119(86.2)	138(100)			
Total	9(3.1)	32(10.9)	253(86.1)	294(100)			
*Analyzed with chi-square test of independence, **Fisher's Exact Test and paired t-test*							

4. 19. Respondents' level of physical activity before and after the intervention

The pre-and post-intervention physical activity (PA) status across the groups is presented as Metabolic Equivalent Task (MET) in table 13. The MET was determined by multiplying the duration (minutes) and frequency (days/week) of each weekly activity. The recommended level of PA by the WHO is a minimum of 600 MET minutes/week (150 minutes/week). A small proportion (IG = 14.7%, CG = 21.7%) met the recommended level of PA at the baseline. The mean of MET-minutes/week was 273.33 and 329.57 for the IG and CG, respectively, at baseline. However, the proportion of respondents who engaged in the required level of physical activity had increased and significantly ($p < 0.001$) higher in the IG (54.5%) compared to the CG (29.7%) at the end line. Furthermore, the IG significantly ($p < 0.001$) improved (746.79 vs 484.35) (mean of MET-minutes/week) compared to the CG at the end-line (Table 13).

Table 13: Level of physical activity of the respondents before and after the intervention

Group	Baseline mean (±SD) MET- minutes/week		Total	t-test	df	p-value
Intervention	273.33(356.52)		156(100)	-1.313	292	0.190
Control	329.57(377.29)		138(100)			
	End-line mean(±SD) MET- minutes/week					
Intervention	746.79 (482.72)		156(100)	4.399	292	0.000
Control	484.35(540.27)		138(100)			
	Baseline Metabolic equivalent of task (n, %)					
	≥600 MET	<600 MET		Chi(χ2)		p-value
Intervention	23(14.7)	133(85.3)	156(100)	2.425	1	0.119
Control	30(21.7)	108(78.3)	138(100)			
Total	53(18.0)	241 (82.0)	294(100)			
	End-line Metabolic equivalent of task (n, %)					
	≥600 MET	<600 MET		Chi(χ2)		p-value
Intervention	85(54.5)	71(45.5)	156(100)	18.356	1	0.000
Control	41(29.7)	97(70.3)	138(100)			
Total	126(42.9	168(57.1)	294(100)			
	Baseline	End-line				
	Mean (±SD) MET- minutes/week			t-test		p-value
Intervention	273.33(356.52)	746.79(482.72)	156(100)	-11.712	155	0.000
Control	329.57(377.29)	484.35(540.27)	138(100)	-4.108	137	0.000
All	299.73(366.87)	623.61(526.28)	294(100)	-11.066	293	0.000

4.20 Respondents' level of knowledge of CVDs lifestyle risk factors

The respondents' level of knowledge of CVDs risk factors was assessed using seven variables at the baseline and end-line (Table 14). The overall score level of knowledge of CVDs risk factors was determined using a score of responses (appendix iv). The overall level of knowledge was categorized as low, moderate, and high for total mean scores < 50%, 50-74%, and ≥75%, respectively. Knowledge level on risk factors for high BP and type-2 diabetes ($P = 0.092$), tobacco smoking ($p = 0.051$), taking excessive amount of salt ($p = 0.452$) and sugar ($p = 0.701$), eating junk foods ($p = 0.150$) and being overweight/obese ($p = 0.945$) as CVDs risk factors of the two groups was not substantially different at the baseline. The control group, however, significantly (p $=0.046$) scored a higher moderate level of knowledge on excessive alcohol consumption as a CVDs risk factor at baseline. At the baseline, the overall level of knowledge on CVDs-related risk factors was not substantially ($p = 0.785$) different between the two groups.

Whereas, at the end-line, the intervention group significantly scored higher level of knowledge in all the CVDs-related risk factors than the control. The proportions of people who scored high level of knowledge on risk factors of high BP and type-2 diabetes (60.3% vs 13.0%), tobacco smoking (74.4% vs 37.0%), eating junk foods (65.4% vs 33.3%), taking excessive alcohol (70.5% vs47.8%), salt (58.3% vs 21.7%), and sugar (61.5% vs 31.9) as well as being overweight/obese (67.3% vs 35.5%) as CVDs risk factors were substantially ($p < 0.001$)) higher in the intervention compared to the control group at the endline. The overall level of knowledge on risk factors of CVDs was significantly (78.2% vs 30.4%; $p < 0.001$) higher in the intervention relative to the control group at the end-line (Table 14).

Table 14: Respondents' level of knowledge of CVDs lifestyle risk factors before and after the intervention (n, %)

Variables	Baseline (before)		X^2 value	df	p value	End-line (after)		X^2 value	df	p value
	Intervention	Control				Intervention	Control			
Knowledge on risk factors for hypertension and type 2 –diabetes										
High	24(15.4)	10(7.2)	4.76	2.00	0.092	94(60.3)	18(13.0)	73.51	2.00	**0.000**
Moderate	33(21.2)	33(23.9)				27(17.3)	33(23.9)			
Low	99(63.5)	95(68.8)				35(22.4)	87(63.0)			
Knowledge on CVDs-related health consequences of tobacco smoking										
High	45(28.8)	28(20.3)	5.94	2.00	0.051	116(74.4)	51(37.0)	54.66	2.00	**0.000**
Moderate	32(20.5)	44(31.9)				27(17.3)	27(19.6)			
Low	79(50.6)	66(47.8)				13(8.3)	60(43.5)			
Knowledge on CVDs-related health consequences of excessive alcohol consumption										
High	46(29.5)	38(27.5)	6.17	2.00	**0.046**	110(70.5)	66(47.8)	38.67	2.00	**0.000**
Moderate	21(13.5)	34(24.6)				26(16.7)	10(7.2)			
Low	89(57.1)	66(47.8)				20(12.8)	62(44.9)			
Knowledge on CVDs-related health consequences of taking too much salt										
High	16(10.3)	9(6.5)	1.59	2.00	0.452	91(58.3)	30(21.7)	71.88	2.00	**0.000**
Moderate	65(41.7)	64(46.4)				46(29.5)	28(20.3)			
Low	75(48.1)	65(47.1)				19(12.2)	80(58.0)			
Knowledge on CVDs-related health consequences of taking too much sugar										
High	26(16.7)	28(20.3)	0.71	2.00	0.701	96(61.5)	44(31.9)	41.88	2.00	**0.000**
Moderate	37(23.7)	33(23.9)				32(20.5)	20(14.5)			
Low	93(59.6)	77(55.8)				28(17.9)	74(53.6)			
Knowledge on CVDs-related health consequences of eating junk foods										
High	31(19.9)	17(12.3)	3.79	2.00	0.150	102(65.4)	46(33.3)	35.18	2.00	**0.000**
Moderate	42(26.9)	47(34.1)				36(23.1)	43(31.2)			
Low	83(53.2)	74(53.6)				18(11.5)	49(35.5)			
Knowledge on CVDs-related health consequences of being overweight										
High	35(22.4)	32(23.2)	0.11	2.00	0.945	105(67.3)	49(35.5)	65.65	2.00	**0.000**
Moderate	48(30.8)	40(29.0)				32(20.5)	11(8.0)			
Low	73(46.8)	66(47.8)				19(12.2)	78(56.5)			
Overall level of knowledge on CVDs lifestyle risk factors										
High	8(5.1)	5(3.6)	0.48	2.00	0.785	122(78.2)	42(30.4)	76.83	2.00	**0.000**
Moderate	65(41.7)	59(42.8)				22(14.1)	30(21.7)			
Low	83(53.2)	74(53.6)				12(7.7)	66(47.8)			

4. 21: Respondents' level of knowledge of CVDs lifestyle preventive measures

The respondents' level of knowledge on preventive measures of CVDs was assessed using six variables at baseline and end-line. The overall score level of knowledge on CVDs-related preventive measures was determined by using a score of responses (appendix iv). Knowledge on whether hypertension and type-2 diabetes are preventable (p = 0.422), the daily recommended amount of sugar intake (p = 0.060), the importance of fruits and vegetables consumption to prevent CVDs (p = 0.682), and the benefits of engaging in physical activity to prevent CVDs (p = 0.349) of the two groups were not significantly different at baseline. However, knowledge on preventive measures of high BP and type-2 diabetes (p = 0.003) and daily recommended amount of salt intake was noticeably (p = 0.044) higher in the intervention relative to the control arm. The overall level of knowledge on CVDs-related preventive measures was not substantially (p = 0.785) different between the two groups at the baseline. Whereas, at the end-line, the intervention group significantly (p < 0.001) scored a higher level of knowledge on all the CVDs-related preventive measures than the control group. Furthermore, the overall level of knowledge of CVDs-related preventive measures was significantly (74.4% vs 29.05; p< 0.001) higher in the intervention arm relative to the control at the end-line (Table 15).

Table 15: Respondents' level of knowledge of CVDs preventive measures before and after the intervention (n, %)

Variables	Baseline		X^2 value	df	p value	End-line		X^2 value	df	p value
	Intervention	Control				Intervention	Control			
Knowledge on whether hypertension and diabetes are preventable										
Yes	138(88.5)	126(91.3)	0.65	1.00	0.422	152(97.4)	126(91.3)	5.35	1	**0.021**
No	18(11.5)	12(8.7)				4(2.6)	12(8.7)			
Knowledge on preventive measures of CVDs (hypertension and diabetes)										
High	31(19.9)	9(6.5)	11.44	2.00	**0.003**	121(77.6)	25(18.1)	103.54	2	**0.000**
Moderate	29(18.6)	34(24.6)				13(8.3)	44(31.9)			
Low	96(61.5)	95(68.8)				22(14.1)	69(50.0)			
Knowledge on the recommended daily amount of salt intake per person (tea spoons)										
less or equal to 1 tsp	33(21.2)	17(12.3)	4.05	1.00	**0.044**	104(66.7)	18(13.0)	86.73	1	**0.000**
I don't know	123(78.8)	121(87.7)				52(33.3)	120(87.0)			
Knowledge on the recommended daily amount of sugar intake per person (tea spoons)										
≤ 5 tea spoons	2(1.3)	7(5.1)	3.55	1.00	0.060	114(73.1)	25(18.1)	88.74	1	**0.000**
I don't know	154(98.7)	131(94.9)				42(26.9)	113(81.9)			
Knowledge of the importance of fruits and vegetables intake to prevent CVDs										
High	28(17.9)	20(14.5)	0.77	2.00	0.682	114(73.1)	55(39,9)			
Moderate	29(18.6)	29(21.0)				22(14.1)	12(8.7)	51.21	2	**0.000**
Low	99(63.5)	89(64.5)				20(12.8)	71(51.4)			
Knowledge on benefits of physical activity on CVDs prevention										
High	18(11.5)	24(17.4)	2.11	2.00	0.349	96(61.5)	49(35.5)	31.16	2	**0.000**
Moderate	44(28.2)	38(27.5)				38(24.4)	31(22.5)			
Low	94(60.3)	76(55.1)				22(14.1)	58(42.0)			
Overall level of knowledge on CVDs lifestyle preventive measures										
High	10(6.4)	5(3.6)	0.48	2.00	0.785	116(74.4)	40(29.0)	71.83	2	**0.000**
Moderate	62(39.7)	62(44.9)				28(17.9)	32(23.2)			
Low	84(53.8)	71 (51.4)				12(7.7)	66(47.8)			

4.22 Respondents' stage of changes towards a healthy lifestyle practice before and after the intervention

The Trans-Theoretical Model (TTM) five-item stage of change was applied to determine the respondents' baseline and end-line readiness for change towards a healthy lifestyle using 9 variables. The participants selected one of five statements that best represented their current intentions for lifestyles change. The first 3 stages (pre-contemplation, contemplation, preparation) were combined together as a pre-action stage. The overall score level of change (adherence) towards a healthy lifestyle was determined by using a score of responses (appendix iv). At baseline, the most frequently reported stage was the pre-action stage of change for fruits, vegetables, processed food, salt, and sugar intake as well as utilization of the DASH diet, with no noticeable variation between the two groups. Of the alcohol consumers, a majority, (intervention = 57.6%, control = 56.9%) were in the pre-action stage of change for alcohol consumption. Whereas, of the respondents with a history of smoking, most, (intervention = 66.7%, control = 89.5%) were in the maintenance stage of change for tobacco smoking with no significant ($p = 0.133$) difference between the two groups. The overall level of adherence towards a healthy lifestyle was not significantly ($p = 0.456$) different between the two groups at the baseline (Table 16-Appendix V**).**

At the end-line, the proportion of people in the maintenance stage of change for daily fruits intake (44.1% vs 20.3%), adherence to the DASH diet (42.9% vs 16.7%), avoiding/limiting processed/fast foods consumption (55.8% vs 36.2%), daily recommended salt (69.9% vs 50.0%) and sugar intake (64.7% vs 45.7%) as well as engaged to the recommended level of physical activity (54.5% vs 29.7%; $p < 0.001$) was substantially ($p < 0.001$) higher in the group who received the intervention relative to the control. Furthermore, the percentage of individuals in the maintenance stage of change for daily vegetables intake was significantly ($p = 0.001$) higher in the intervention (70.5%) than in the control group (49.3%). Of the alcohol and tobacco users, a majority of them in both groups were in the maintenance stage of change with no considerable variation between the groups. The overall level of change (adherence) towards a healthy lifestyle was significant ($p < 0.001$) higher in the group who received the intervention (68.6%) relative to the control group (25.4%) at the end-line (Table 17-Appendix V).

4.23 Respondents' self-efficacy towards a healthy lifestyle practice before and after the intervention

The individuals' perceived confidence towards a healthy lifestyle practice was assessed using nine (9) lifestyle variables at the baseline and end-line. The responses were categorized as (not at all confident =1, somewhat confident = 2 and extremely confident = 3). At the baseline, the most frequently reported level of confidence was not at all confident to take the recommended frequency of fruits and vegetables, salt, sugar, and adhere to the DASH eating plan with no statistical difference between the two groups. A good number (IG = 39.7%, CG = 40.6%) of the respondents were extremely confident to avoid/limit eating processed/fast foods. Of the alcohol consumers, a majority (IG = 57.6%, CG = 56.9%) were not at all confident to stop/moderate intake of it. Whereas, of the respondents with a history of smoking, most, (IG = 66.7%, CG = 89.5%) were extremely confident to stop it, with no significant ($p = 0.133$) difference between the two groups. Furthermore, a higher proportion (IG = 42.3%, CG = 44.2%) were extremely confident to participate in the recommended level of physical activity at the baseline (Table 18-Appendix V**).**

At the end-line, the percentage of people who were extremely confident to take the recommended frequency of fruits (44.2% vs 20.3%), amount of salt (69.9% vs 50.0%), sugar (65.4% vs 45.7%), utilize the DASH eating diet (42.9 vs 16.7%) and engage to the recommended level of physical activity was markedly ($p < 0.001$) higher in the group who received the intervention (54.5%) relative to the control arm (29.7%). Moreover, the percentage of people who were extremely confident to take the recommended frequency of vegetables (70.5% vs 49.3%; $p = 0.001$) and avoid/limit eating processed/fast foods (55.8% vs 36.2%; $p = 0.004$) was noticeably higher in the intervention arm relative to the control. Of the alcohol and tobacco users, a majority of both groups were extremely confident to stop/moderate alcohol intake and stop smoking (Table 19-Appendix V).

4.24 Respondents' decisional balance (pros) to a healthy lifestyle practice before and after the intervention

Decisional Balance, the balance between the perceived advantages (pros) and disadvantages/barriers (cons) of adopting a new behaviour was assessed using 15 pros and 11 cons. The responses were categorized as (disagree =1, somewhat = 2 and agree = 3). The perceived advantages of adopting a new behaviour (the pros) between the two groups were not significantly different at baseline. At the baseline, a majority of them agreed that regular eating vegetables and fruits could help them live a better, healthier, and longer life. Moreover, most of the respondents agreed that eating five or more servings of vegetables and fruits per day, limiting sugar (≤5 teaspoons per day) and salt (≤1 teaspoon per day) intake to the recommended amount could help prevent hypertension and diabetes. A good number (IG = 44.9%, CG = 37.7%) of them believed that eating vegetables and fruits could help lose/maintain a healthy weight. However, most of the respondents agreed that they cannot find reasonably priced fruits and vegetables in their local markets. Below half, (intervention = 47.4%, control = 42.8%) agreed that limiting/avoiding processed/fast foods could help them prevent hypertension and diabetes. Below a third (IG = 30.8%, CG = 26.8%) believed that limiting/avoiding alcohol consumption could help prevent hypertension and diabetes. Moreover, approximately half (intervention = 47.1%, control = 51.4%) agreed that exercising for a minimum of 30 minutes for 5-7 days per week could help prevent hypertension and diabetes (Table 20-Appendix V).

Generally, the intervention group increased their pros of decisional balance towards a healthy lifestyle practice relative to the control arm at the end-line. The proportions of people who agreed that regular eating vegetables and fruits could help prevent diseases (72.4% vs 58.0%; $p = 0.020$), lose/maintain a healthy weight (74.4% vs 49.3%; $p < 0.001$) and prevent hypertension and diabetes (78.2% vs 59.4%; $p = 0.001$) were markedly higher in intervention relative the control group at the end-line. However, only about one-third (31.4%) of the intervention and one-fifth (20.3%) of the control group agreed that they can find reasonably priced fruits and vegetables in their local markets.

The proportion of people who agreed that limiting to the recommended amount of salt (76.9% vs 53.6%; $p < 0.001$) and sugar (77.6% vs 61.6%; $p = 0.006$) intake, limiting/avoiding processed/fast foods (76.3% vs 58.7%; $p = 0.002$) and alcohol (78.8% vs 55.8%; $p < 0.001$) could help prevent hypertension and diabetes was statistically higher in the intervention arm than the control at the end-line. Moreover, the percentage of people who agreed that engaging in the recommended level of physical activity could help lose or maintain a healthy weight (76.3% vs 60.1%; $p = 0.005$) and prevent hypertension and diabetes (76.3% vs 55.8%; p = 0.001) was substantially higher in the intervention relative to the control arm (table 21-Appendix V).

4.24 Respondents' decisional balance (cons) to a healthy lifestyle during the study period

The respondents' decisional balance on the perceived disadvantages/barriers to adopting a new, healthy lifestyle (cons) was assessed using 11 variables both at the baseline and end-line. At the baseline, there was no noticeable difference between the two groups on the perceived disadvantages to adopting a healthy lifestyle at baseline. A majority of the respondents viewed that buying fruits and vegetables is too expensive and, therefore, eating five or more servings each day is difficult. Moreover, about one-third of each group agreed that they concern about the chemicals used in fruits and vegetables. Approximately, one-third (IG = 37.2%, CG = 35.5%) agreed that it is difficult to control daily salt and sugar intake.

Moreover, a majority (51.3%) of the intervention and close to half (44.9%) of the control group agreed that food with less or no salt or sugar is tasteless. Most (58.7%) of the control and half (50.0%) of the intervention group agreed that processed foods are much available and difficult to avoid or limit. A majority of the alcohol consumers agreed that it is difficult to limit or avoid alcohol intake. Moreover, of those with a history of smoking, one-third (33.3%) of the IG and a small number (10.5%) of the CG agreed that it is hard to stop smoking. A good number (intervention = 31.4%, control = 40.6%) agreed that they feel that they don't have time to participate in physical activity for 5-7 days per week (Table 22-Appendix V).

Overall, the intervention group decreased their cons of decisional balance towards a healthy lifestyle practice relative to the control group at the endline. A majority (IG = 54.5%, CG = 59.4%) of the study subjects agreed that buying fruits and vegetables is too expensive, thus taking five or more servings of vegetables and fruits each day is difficult, with marginal significance (p = 0.051) lower in the intervention (51.9%) relative to the control arm (65.9%). The proportion of people who agreed that they have limited ways to include vegetables and fruits in their daily meals was significantly (p = 0.038) lower the intervention (31.4%) relative to the control group (44.9%).

Further, approximately one-third (IG = 34.6%, CG = 38.4%) agreed that they worry about the safety of chemicals used in fruits and vegetables.

The proportion of respondents who agreed that it is difficult to control daily salt and sugar intake (IG = 25.0%, CG = 37.7%), food with less or no salt/sugar is tasteless (IG =37.8%, CG = 43.5%) and difficult to limit/avoid processed foods (IG =35.9%, CG =52.9%) was substantially ($p < 0.05$) lower in the intervention relative to the control group. Of the alcohol consumers, (IG = 27.1%, CG = 41.2%) agreed that it is difficult to limit or avoid it. Moreover, of those with a history of smoking, approximately a third (28.6%) of the intervention and a small number (10.5%) of the control arm agreed that it is hard to stop smoking. A majority (IG = 64.1%, CG = 50.0%) of the respondents disagreed that they feel they don't have time to do physical activity for 5-7 days per week (table 23-Appendix V).

4 25: Pre-and post intervention mean differences of subtotal TTM construct within the groups

Table 24 displays pairwise baseline and end-line differences in TTM core constructs within the groups. There were significant improvements in all the TTM constructs including the stage of change, self-efficacy, and decisional balance after the intervention relative to the baseline. The mean scores of stages of change ($t = 22.570$; $p < 0.001$), self-efficacy ($t = 9.341$; $p < 0.001$), and pros of decisional balance ($t = 20.332$; $p < 0.001$) significantly increased in the intervention group at the end-line relative to the baseline. Moreover, the mean scores of cons of decisional balance ($t = 8.070$; $p < 0.001$) substantially decreased in the intervention group at the end-line relative to the baseline. In the control group, the mean scores of stages of change ($t = 10.349$; $p < 0.001$) and pros of decisional balance ($t = 10.798$; $p < 0.001$) significantly increased and the cons of decisional balance ($t = 2.677$; $p = 0.008$) significantly decreased at the end-line compared to the baseline. However, there was no noticeable change seen in the self-efficacy construct in the control group at the end-line compared to baseline (Table 24).

Table 24: Mean differences of subtotal TTM construct scores of the study subjects between pre and post intervention (Mean ± SD)

Group	Maximum score	Pre-intervention	Post-intervention	Paired t-test	df	p-value
		TTM constructs score				
	27	Stage of change				
Intervention		15.13(2.42)	22.00(3.36)	22.570	155	0.000
Control		15.38(2.39)	18.57(3.27)	10.349	137	0.000
	27	Self-efficacy				
Intervention		18.66(3.48)	22.03(3.33)	9.341	155	0.000
Control		18.75(3.70)	18.86(3.22)	0.264	137	0.792
	45	Decisional balance (pros)				
Intervention		33.49(4.49)	39.20(4.37)	20.332	155	0.000
Control		32.62(4.69)	35.85(4.47)	10.798	137	0.000
	33	Decisional balance (cons)				
Intervention		19.33(3.03)	17.17(3.63)	8.070	155	0.000
Control		19.79(2.91)	19.13(3.06)	2.677	137	0.008

4.26 Relationship between socio-demographics and MetS in the intervention group

Of the socio-demographics, age, marital status, religion, education and employment status in the intervention group were substantially associated with MetS at the end-line. Respondents aged 50 years and above were at a 10-fold (COR = 10.095, 95% CI = 3.950-25.797; p < 0.001) increased risk of MetS compared to respondents aged below 50 years. Respondents belonged to protestants (COR = 8.595, 95% CI = 1.816-40.685; p = 0.007) and Catholics (COR = 8.182, 95% CI = 1.660-40.316; p = 0.010) were about 8-times at increased risk of MetS relative to Muslims. Respondents with primary (COR = 2.500, 95% CI = 0.993-6.294; p = 0.052) and secondary (COR = 2.559, 95% CI = 1.224-5.352; p = 0.013) level of education were at 2.5 and 2.6-fold, respectively, elevated risk of MetS than respondents who attained tertiary level of education. Whereas, married (COR = 0.175, 95% CI = 0.038-0.816; p = 0.026) and employed (COR = 0.204, 95% CI = 0.068-0.607; p = 0.004) respondents were at 82.5% and 79.6%, respectively, less likely to have MetS compared to divorced/separated/widowed and unemployed respondents (Table 25).

Table 25: Relationship between socio-demographics and MetS in the intervention group (n, %)

Characteristic	MetS status		Total	OR (95%CI)	**p-value**
	Yes	No			
Age					
50 and above	41(87.2)	6(12.8)	47(100)	10.095(3.950-25.797)	0.000
Below 50	44(40.4)	65(59.6)	109(100)	Reference	
Total	85(54.5)	71(45.5)	156(100)		
Sex					
Male	37(50)	37(50)	74(100)	0.708(0.376-1.334)	0.286
Female	48(58.5)	34(41.5)	82(100)	Reference	
Total	85(54.5)	71(45.5)	156(100)		
Marital status					
Married	62(51.2)	59(48.8)	121(100)	0.175(0.038-0.816)	0.026
Single	11(52.4)	10(47.6)	21(100)	0.183(0.033-1.029)	0.054
Divorced/separated/widowed	12(85.7)	2(14.3)	14(100)	Reference	
Total	85(54.5)	71(45.5)	156(100)		
Religion					
Protestant	53(58.9)	37(41.1)	90(100)	8.595 (1.816-40.685)	0.007
Catholic	30(57.7)	22(42.3)	52(100)	8.182(1.660-40.316)	0.010
Muslim	2(14.3)	12(85.7)	14(100)	Reference	
Total	85(54.5)	71(45.5)	156(100)		
Education level					
None - Primary	19(61.3)	12(38.7)	31(100)	2.500(0.993-6.294)	0.052
Secondary	47(61.8)	29(38.2)	76(100)	2.559(1.224-5.352)	0.013
Tertiary level	19(38.8)	30(61.2)	49(100)	Reference	
Total	85(54.5)	71(45.5)	156(100)		
Occupation					
Employed	19(35.2)	35(64.8)	54(100)	0.204(0.068-0.607)	0.004
Self- Employed	50(62.5)	30(37.5)	80(100)	0.625(0.221-1.771)	0.377
Unemployed	16 (72.7)	6(27.3)	22(100)	Reference	
Total	85(54.5)	71(45.5)	156(100)		
Income (USD)					
Less than 100	5(55.6)	4(44.4)	9(100)	1.250(0.268-5.826)	0.776
100 – 500	63(53.8)	54(46.2)	117(100)	1.167(0.484-2.809)	0.731
Over 500	12(50.0)	12(50.0)	24(100)	Reference	
Total	80(53.3)	70(46.7)	150(100)		

4.27 Relationship between socio-demographics and MetS in the control group

Respondents' age and income were the social variables substantially linked to MetS in the control arm at the end-line. The likelihood of having MetS was 5.3 times (OR = 5.263, 95% CI = 1.171-23.662; p = 0.030) more common among respondents aged 50 years and above relative to those aged below 50 years. Respondents whose monthly income between USD 100-500 were at about a 4-fold (OR = 3.778, 95% CI = 1.173-12.169; p = 0.026) elevated riks of MetS compared to respondents who had monthly income of above USD 500 (Table 26).

Table 26: Relationship between socio-demographics and MetS in the control group (n, %)

Characteristic	MetS status		Total	OR (95%CI)	**p-value**
	Yes	No			
Age (years)					
50 and above	40(95.2)	2(4.8)	42(100)	5.263(1.171-23.662)	0.030
Below 50	76(79.2)	20(20.8)	96(100)	Reference	
Total	116(84.1)	22(15.9	138(100)		
Sex					
Male	49(81.7)	11(18.3)	60(100)	0.731(0.293-1.823)	0.502
Female	67(85.9)	11(14.1)	78(100)	Reference	
Total	116(84.1)	22(15.9)	138(100)		
Marital status					
Married	93(86.1)	15(13.9)	108(100)	0.620(0.074-5.200)	0.660
Single	13(68.4)	6(31.6)	19(100)	0.217(0.022-2.101)	0.187
Divorced/separated/widowed	10(90.9)	1(9.1)	11(100)	Reference	
Total	116(84.1)	22(15.9)	138(100)		
Religion					
Protestant	75(86.2)	12(13.8)	87(100)	3.125(0.688-14.202)	0.140
Catholic	35(83.3)	7(16.7)	42(100)	2.500(0.502-12.457)	0.263
Muslim	6(66.7)	3(33.3)	9(100)	Reference	
Total	116(84.1)	22(15.9)	138(100)		
Education level					
None - Primary	31(93.9)	2(6.1)	33(100)	4.559(0.926-22.452)	0.062
Secondary	51(83.6)	10(16.4)	61(100)	1.500(0.564-3.989)	0.416
Tertiary level	34(77.3)	10(22.7)	44(100)	Reference	
Total	116(84.1)	22(15.9)	138(100)		
Occupation					
Employed	32(82.1)	7(17.9)	39(100)	0.653(0.152-2.813)	0.567
Self- Employed	63(84.0)	12(16)	75(100)	0.750(0.194-2.917)	0.678
Unemployed	21(87.5)	3(12.5)	24(100)	Reference	
Total	116(84.1)	22(15.9)	138(100)		
Income (USD)					
Less than 100	9(64.3)	5(35.7)	14(100)	0.720(0.170-3.058)	0.656
100 - 500	85(90.4)	9(9.6)	94(100)	3.778(1.173-12.169)	0.026
Over 500	15(71.4)	6(28.6)	21(100)	Reference	
Total	109(84.5)	20(15.5)	129(100)		

4.28. Association between dietary components and MetS after the intervention

The relationship between diet and MetS at the end-line is presented below (Tables 27 and 28). There was an inverse relationship beween adherence to the DASH diet, vegetables, fruits, legumes and nuts intake and MetS. Whereas, processed food and sugar intake was directly linked to MetS. The rate of MetS was markedly lower in the intervention (COR = 0.288, 95% CI = 0.148-0.559; p < 0.001) and control group (COR = 0.343, 95% CI = 0.121-0.971; p = 0.044) among respondents who adhered to the DASH diet relative to those who did not adhere to it. Individuals in the intervention group who daily consumed fruits (COR = 0.105; 95% CI = 0.051-0.219; p < 0.001) and vegetables (COR = 0.262, 95% CI = 0.121-0.568; p = 0.001) were at 89.5% and 73.8%, respectively, reduced risk of MetS than people who did not daily take fruits and vegetables.

The odds of having MetS was markedly lower in both the IG (COR = 0.304, 95% CI = 0.121-0.766; p = 0.012) and the CG (COR = 0.106, 95% CI = 0.031-0.365; p < 0.001) among respondents who consumed the recommended frequency (≥4 times/week) of legumes compared to those who rarely (≤ once/week) ate legumes. Similarly, individuals in the IG (COR = 0.090, 95% CI = 0.029-0.277; p < 0.001) and CG (COR = 0.151, 95% CI = 0.042-0.540; p = 0.004) who consumed the recommended frequency (≥4 times/week) of nuts had a lower odds ratio in relation to MetS compared to those who rarely (≤ once/week) took nuts (Table 27).

Further, individuals in the intervention group who consumed the daily recommended amount of sugar (≤ 5 tea spoons) (COR = 0. 497, 95% CI = 0.252-0.981; p = 0.044) were at 50.3% reduced risk of MetS compared to those who consumed above the recommended amount. Whereas, participants in the IG (COR = 6.545, 95% CI = 2.423-17.684; P < 0.001) and CG (COR = 7.027, 95% CI = 1.485- 33.253; P = 0.014) who often (≥5 times/week) consumed processed/fast foods were about a seven-fold elevated risk of MetS relative to those who rarely (≤ once/week) took such foods. However, in both groups, no noticeable relationship was observed between salt intake and MetS (Table 28**).**

Table 27: Association between dietary intake patterns and MetS across the groups (n, %)

Group	Dietary status	End-line MetS		Total	COR (95%CI)	p-value
		Yes	No			
	DASH eating plan					
IG	Recommended	25 (37.3)	42 (62.7)	67 (100)	0.288(0.148-0.559)	0.000
	Not Recommended	60 (67.4)	29 (32.6)	89 (100)	Reference	
	Total	85 (54.5)	71 (45.5)	156 (100)		
CG	Recommended	16 (69.6)	7 (30.4)	23 (100)	0.343(0.121-0.971)	0.044
	Not Recommended	100 (87.0)	15 (13.0)	115 (100)	Reference	
	Total	116 (84.1)	22 (15.9)	138 (100)		
	Fruit intake					
IG	Daily	18 (26.1)	51 (73.9)	69 (100)	0.105(0.051-0.219)	0.000
	Not Daily	67 (77.0)	20 (23.0)	87(100)	Reference	
	Total	85 (54.5)	71 (45.5)	156 (100)		
CG	Daily	21 (75.0)	7 (25.0)	28 (100)	0.474(0.172-1.306)	0.149
	Not Daily	95 (86.4)	15 (13.6)	110 (100)	Reference	
	Total	116 (84.1)	22 (15.9)	138 (100)		
	Vegetable intake					
IG	Daily	50 (45.5)	60 (54.5)	110 (100)	0.262(0.121-0.568)	0.001
	Not Daily	35 (76.1)	11 (23.9)	46 (100)	Reference	
	Total	85 (54.5)	71 (45.5)	156 (100)		
CG	Daily	55 (80.9)	13 (19.1)	68 (100)	0.624(0.248-1.574)	0.318
	Not Daily	61 (87.1)	9 (12.9)	70 (100)	Reference	
	Total	116 (84.1)	22 (15.9)	138 (100)		
	Legume intake					
IG	Often	35 (46.7)	40 (53.3)	75 (100)	0.304(0.121-0.766)	0.012
	Sometimes	27 (54)	23 (46)	50 (100)	0.408(0.154-1.086)	0.073
	Rarely	23 (74.2)	8 (25.8)	31 (100)	Reference	
	Total	85 (54.5)	71 (45.5)	156 (100)		
CG	Often	20 (62.5)	12 (37.5)	32 (100)	0.106(0.031-0.365)	0.000
	Sometimes	33 (84.6)	6 (15.4)	39 (100)	0.349(0.092-1.325)	0.122
	Rarely	63 (94)	4 (6)	67 (100)	Reference	
	Total	116 (84.1)	22 (15.9)	138 (100)		
	Nuts intake					
IG	Often	16 (32.7)	33 (67.3)	49 (100)	0.090(0.029-0.277)	0.000
	Sometimes	42 (56.0)	33 (44.0)	75 (100)	0.236(0.082-0.679)	0.007
	Rarely	27 (84.4)	5 (15.6)	32 (100)	Reference	
	Total	85 (54.5)	71 (45.5)	156 (100)		
CG	Often	15 (68.2)	7 (31.8)	22 (100)	0.151(0.042-0.540)	0.004
	Sometimes	30 (75)	10 (25)	40 (100)	0.211(0.067-0.671)	0.008
	Rarely	71 (93.4)	5 (6.6)	76 (100)	Reference	
	Total	116 (84.1)	22 (15.9)	138 (100)		

Table 28: Relationship between processed foods, salt and sugar intake and MetS across the groups (n, %)

Group	Dietary status	End-line MetS		Total	COR (95%CI)	p-value
		Yes	No			
	Fast food intake					
IG	Often	24 (80.0)	6 (20.0)	30 (100)	6.545(2.423-17.684)	0.000
	Sometimes	28 (71.8)	11 (28.2)	39 (100)	4.165(1.833-9.466)	0.001
	Rarely	33 (37.9)	54 (62.1)	87 (100)	Reference	
	Total	85 (54.5)	71 (45.5)	156 (100)		
CG	Often	40 (95.2)	2 (4.8)	42 (100)	7.027(1.485-33.253)	0.014
	Sometimes	39 (84.8)	7 (15.2)	46 (100)	1.958(0.704-5.445)	0.198
	Rarely	37 (74)	13 (26)	50 (100)	Reference	
	Total	116 (84.1)	22 (15.9)	138 (100)		
	Salt intake					
IG	Recommended	57(52.3)	52(47.7)	109(100)	0.744(0.372-1.488)	0.403
	Not recommended	28 (59.6)	19(40.4)	47(100)	**Reference**	
	Total	85 (54.5)	71(45.5)	156(100)		
CG	Recommended	56(81.2)	13(18.8)	69(100)	0.646 (0.256-1.629)	0.355
	Not recommended	60 (87.0)	9(13.0)	69(100)	**Reference**	
	Total	116 (84.1)	22(15.9)	138(100)		
	Sugar intake					
IG	Recommended	49(48.5)	52(51.5)	101(100)	0.497(0.252-0.981)	0.044
	Not recommended	36(65.5)	19(34.5)	55(100)	**Reference**	
	Total	85(54.5)	71(45.5)	156(100)		
CG	Recommended	51 (81.0)	12 (19.0)	63(100)	0.654(0.262-1.634)	0.363
	Not recommended	65(86.7)	10(13.3)	75(100)	**Reference**	
	Total	116(84.1)	22(15.9)	138(100)		

4.29 Alcohol consumption and physical activity in relation to MetS across the groups

Table 29 displays the effect of alcohol intake and physical activity on MetS at the end-line. Respondents in the IG (OR = 16. 500; 95% CI = 2.687-101.331; P = 0.002) and CG (OR = 15. 833; 95% CI = 2.054-122.069; P = 0.008) who consumed above the daily recommended amount of alcohol (>2 drinks in men and > 1 in women) had about a sixteen-fold elevated risk of MetS relative to individuals who took the recommended amount. Moreover, MetS was 14-times (OR = 14.400; 95% CI = 2.289-90.597; P = 0.004) more common among individuals in the IG who consumed alcohol above 8 occasions in a month relative to those who took it less frequently (≤ 8 occasions per month). Whereas, MetS was less prevalent among participants in the IG (COR = 0.044; 95% CI = 0.018-0.107; P < 0.001) and CG (COR = 0.010, 95% CI = 0.001-0.078; P < 0.001) who had participated for adequate level of physical activity (≥ 600 MET-minutes per week) as compared to individuals who did not engage for such level of physical activity (Table 29).

Table 29: Alcohol consumption and physical activity in relation to MetS across the groups

Group	Behavioral pattern	End-line MetS (n, %)		Total	COR (95%CI)	p-value
		Yes	No			
	Alcohol intake					
IG	Not recommended	11 (68.7)	5 (31.3)	16(100)	16.500(2.687-101.331)	0.002
	Recommended	2 (11.8)	15 (88.2)	17 (100)	Reference	
	Total	13 (39.4)	20 (60.6)	33 (100)		
CG	Not recommended	19 (90.5)	2 (9.5)	21 (100)	15.833(2.054-122.069)	0.008
	Recommended	3 (37.5)	5 (62.5)	8 (100)	Reference	
	Total	22 (75.9)	7 (24.1)	29 (100)		
	Monthly Occasions					
IG	Above 8 times	8(80.0)	2(20.0)	10(100)	14.400(2.289-90.597)	0.004
	Below or 8 times	5(21.7)	18(78.3)	23(100)	Reference	
	Total	13 (39.4)	20(60.6)	33 (100)		
CG	Above 8 times	14(87.5)	2(12.5)	16(100)	4.375(0.684-27.983)	0.119
	Below or 8 times	8(61.5)	5(38.5)	13(100)	Reference	
	Total	22(75.9)	7(24.1)	29(100)		
	Physical activity					
IG	Recommended	22 (25.9)	63 (74.1)	85 (100)	0.044 (0.018 - 0.107)	0.000
	Not recommended	63 (88.7)	8 (11.3)	71 (100)	Reference	
	Total	85 (54.5)	71 (45.5)	156 (100)		
CG	Recommended	20 (48.8)	21 (51.2)	41 (100)	0.010 (0.001 - 0.078)	0.000
	Not recommended	96 (99.0)	1 (1.0)	97 (100)	Reference	
	Total	116 (84.1)	22 (15.9)	138 (100)		

4.30 Association between smoking and metabolic syndrome across the group

Analysis with Fisher's Exact Test showed that there was no association between tobacco use and MetS in the intervention (p = 0.934) and control (p = 0.589) groups (Table 30).

Table 30: Relationship between smoking and metabolic syndrome across the group

Group	Smoking status	End-line MetS		Total	Chi (χ2)	df	p-value
		Yes	No				
IG	Current smokers	3 (50.0)	3(50.0)	6 (100)	0.339	-	0.934*
	Past smokers	9 (60.0)	6 (40.0)	15 (100)			
	No history of smoking	73 (54.1)	62(45.9)	135(100)			
	Total	85 (54.5)	71(45.5)	156 (100)			
CG	Current smokers	2 (66.7)	1 (33.3)	3(100)	1.323	-	0.589*
	Past smokers	15 (88.2)	2 (11.8)	17 (100)			
	No history of smoking	99 (83.9)	19(16.1)	118(100)			
	Total	116 (84.1)	22(15.9)	138 (100)			

**Fisher's Exact Test*

4.31 Factors independently associated with MetS in the intervention group at the end-line

A multivariate logistic regression analysis was used to determine the independent factors associated with MetS. At bivariate analysis, respondents' socio-demographics: age, marital status, religion, education, and occupational status and lifestyle factors included: the DASH eating plan, consumption of vegetables, fruits, nuts, legumes, processed/fast foods, alcohol and physical activity were significantly ($p<0.05$) associated with MetS in the intervention (Table 31A).

After subjecting these variables in multivariate analysis by indicating the '*backward conditional'* technique with removal at $p<0.05$; seven (7) variables persisted in the reduced model as independent predictors of MetS (Table 31B). The variables that remained in the final model were age, religion, occupation, consumption of fruits, processed/fast foods, alcohol, and physical activity. According to Hosmer and Lemeshow Test, the fitness model was 0.573, which shows the model fits.

Participants aged 50 years and above were at a 9-fold [AOR = 9.097; 95%CI=3.342 - 24.758; $p < 0.001$] increased risk of MetS compared to those who aged below 50 years. Respondents belonged to Protestants [AOR = 7.292; 95%CI=1.431- 37.147; p = 0.017] and Catholics [AOR = 5.270; 95%CI=0.999 – 27.799; p = 0.050] were 7 and 5 times, respectively, at increased risk of MetS relative to the Muslims. Further, respondents who often (≥ 5 times/week) consumed processed/fast foods were at about a 9-fold [AOR = 8.75; 95%CI= 2.17 – 35.25; p = 0.002] increased risk of MetS compared to those who rarely (≤ once/week) consumed such foods. Moreover, the risk of having MetS was 13-times [AOR = 13.368; 95%CI= 1.901 – 94.002; p = 0.009] higher among respondents who consumed above the recommended amount of alcohol relative to those who consumed the recommended amount.

Whereas, respondents who were employed [AOR = 0.154; 95%CI= 0.042–0.560; p = 0.005] and dialy consumed fruits [AOR = 0.132; 95%CI= 0.037-0.471; p = 0.002] were at 84.6% and 86.8%, less likely to have MetS relative to unemployed and those who did not daily consume fruits, respectively. Moreover, the odds of having MetS was markedly lower [AOR = 0.03; 95%CI= 0.01– 0.11; $p <0.001$] among respondents who had participated for adequate level of physical activity relative to those who did not engage for the same level of PA (Table 31B).

Table 31A: Factors associated with MetS in the intervention group at the end-line

Variables	AOR	95%CI		p value
		Lower	Upper	
Full model				
Age (years)				
50 and above	10.009	3.494	28.667	0.000
Below 50	Reference			
Marital status				
Married	0.238	0.039	1.442	0.118
Single	0.410	0.054	3.103	0.388
Divorced/separated/widowed	Reference			
Religion				
Protestants	8.269	1.520	44.996	0.015
Catholics	5.977	1.069	33.409	0.042
Muslim	Reference			
Education				
None - Primary level	1.697	0.527	5.463	0.375
Secondary level	2.075	0.851	5.064	0.109
Tertiary level	Reference			
Occupation				
Employed	0.213	0.054	0.842	0.027
Self- Employed	0.688	0.193	2.457	0.565
Unemployed	Reference			
DASH plan				
Recommended	0.36	0.10	1.33	0.125
Not Recommended	Reference			
Fruits consumption				
Daily	0.131	0.037	0.471	0.002
Not daily	Reference			
Vegetables consumption				
Recommended	0.65	0.14	2.94	0.576
Not Recommended	Ref			
Legumes consumption				
Often	0.70	0.17	2.80	0.609
Sometimes	0.90	0.13	6.12	0.914
rarely	Reference			
Nuts consumption				
Often	0.63	0.14	2.83	0.549
Sometimes	0.31	0.04	2.76	0.294
rarely	Reference			
Processed food intake				
Often	9.83	2.07	46.69	0.004
Sometimes	6.13	1.59	23.63	0.009

rarely	Reference			
Sugar intake				
Recommended				
Above recommended				
Alcohol consumption				
Above recommended	10.340	1.173	91.116	0.035
Recommended	Reference			
Physical activity				
Recommended	0.03	0.01	0.11	0.000
Not Recommended	Ref			
AOR= Adjusted odds ratio; CI = confidence interval				

Table 31B: Independent variables linked to MetS in the intervention group at the end-line

Variables	AOR	95%CI		p value
		Lower	Upper	
Reduced model				
Age (Years)				
50 and above	9.097	3.342	24.758	**0.000**
Below 50	Reference			
Religion				
Protestant	7.292	1.431	37.147	**0.017**
Catholic	5.270	.999	27.799	**0.050**
Muslim	Reference			
Occupation				
Employed	0.154	0.042	0.560	**0.005**
Self- Employed	0.587	0.175	1.966	**0.388**
Unemployed	Reference			
Fruits intake				
Daily	0.132	0.037	0.471	**0.002**
Not daily	Reference			
Consumption of processed foods				
Often	8.75	2.17	35.25	**0.002**
Sometimes	5.67	1.69	19.00	**0.005**
Rarely	Reference			
Alcohol consumption				
Not Recommended	13.368	1.901	94.002	**0.009**
Recommended	Reference			
Physical activity				
Recommended	0.03	0.01	0.11	**0.000**
Not Recommended	Reference			
AOR= Adjusted odds ratio; CI = /confidence interval				
Goodness of fit: The Hosmer-Lemeshow test was used to assess goodness of fit for the fitted model, a p value = 0.573 indicates that the fitted model is adequate.				

4.32 Factors independently related to MetS in the control group

The factors significantly ($p<0.05$) related to MetS during a bivariate analysis in the control group were age, income, the DASH diet, consumption of legumes, nuts, and processed/fast foods, and physical activity. After subjecting all these factors into a multivariate analysis, four (4) variables including age, income, consumption of legumes, and physical activity remain as independent predictors of MetS.

The risk of having MetS was 5-times [AOR = 5.013; 95%CI=1.022 – 23.831; p = 0.047] higher among aged (≥ 50 years) respondents relative to those who aged below 50 years. Respondents who had monthly income between USD 100 to 500 were at about a 15-fold [AOR = 14.817; 95%CI= 1.640– 133.838; p =0.016] increased risk of MetS relative to those who had less than USD 100. Whereas, the risk of MetS was markedly lower among respondents who often (≥ 5 times/week) consumed legumes [AOR = 0.042; 95%CI= 0.005-0.391; p =0.005] and engaged in an adequate level of physical activity [AOR = 0.003; 95%CI= 0.000– 0.052; p <0.001] relative to those who rarely/never took legumes and did not participate in an adequate level of physical activity, respectively (Table 32).

Table 32: **Factors independently associated with end-line prevalence of MetS in the control group**

Variables	AOR	95%CI		p value
		Lower	Upper	
Full model				
Age				
50 and above	5.180	1.030	26.063	0.046
Below 50	Reference			
Income in USD				
< 100	Reference			
100 to 500	14.817	1.640	133.838	0.016
> 500	0.701	0.052	9.445	0.789
DASH plan				
Recommended	0.590	0.179	1.946	0.386
Not Recommended	Reference			
Legumes consumption				
Often	0.059	0.004	0.889	0.041
Sometimes	0.303	0.027	3.377	0.332
Rarely/never	Ref			
Nuts consumption				
Often	0.756	0.058	9.772	0.830
Sometimes	0.376	0.042	3.392	0.384
Rarely/never	Reference			
Processed/fast foods intake				
Often	3.175	0.252	40.022	0.372
Sometimes	2.357	0.355	15.636	0.374
Rarely/never	Reference			
Physical activity				
Recommended	0.003	0	0.057	0.000
Not Recommended	Ref			
Reduced model				
Age				
50 and above	5.013	1.022	23.831	0.047
Below 50	Reference			
Income in USD				
< 100	Reference			
100 to 500	10.499	1.366	80.667	0.024
> 500	0.669	0.056	8.026	0.751
Legumes consumption				
Often	0.042	0.005	0.391	0.005
Sometimes	0.15	0.019	1.198	0.073
rarely	Reference			
Physical activity				
Recommended	0.003	0.000	0.052	0.000
Not Recommended	Reference			
Fitness model using Hosmer and Lemeshow Test = 0.194, AOR= Adjusted odds ratio; CI = /confidence interval				

CHAPTER FIVE: DISCUSSION

5.1 Introduction

This study aimed to establish the effectiveness of a community-based lifestyle intervention in controlling MetS and its elements in adults using a randomized control trial approach in Kenya. Furthermore, the study has established the effect of lifestyle intervention on knowledge and preventive measures of MetS-CVDs.

5.2 Changes in proportion of MetS and its elements over the study period across the groups

There was a substantial decline in the proportion of MetS and its components associated with the community-based lifestyle intervention approach. There was a 42.9% and 45.5% decline in the proportion of MetS during midline and end-line for the intervention compared to 21.7% and 15.9% in the control group, respectively. This translates to an Absolute Risk Reduction (ARR) of 29.6% in the prevalence of MetS at the end-line. This shows that one in three adults with MetS exposed to a community-based lifestyle intervention had their condition improved. The rates of the components of MetS namely central obesity, elevated BP, raised TGs, and low HDL-C as well as anthropometric parameters including body weight, BMI, WHR, and WHtR improved across the study period in the intervention compared to the control group.

The decline in the proportion of MetS and its components, as well as anthropometric measurements, mirrored improvements in dietary intake patterns and physical activity uptake attributed to the lifestyle intervention in the treatment group. Our findings attributable to the community-based lifestyle intervention is consistent with a 12-month study that reported a 50% decline in the prevalence of MetS with significant improvement of FBG and HDL-C in Switzerland (Gerstel *et al.*, 2013). A nutritional education intervention among Kenyan adults with type-2 diabetes showed a 38% decline in the prevalence of MetS in the treatment group (Thuita et al., 2020). This finding (38%) is much lower than that of our finding (45.5%). However, such a difference is expected, because, in our study, the majory (75%) of the participants did not have diabetes and/or hypertension, while in the other study, all were diabetic patients, a component of MetS.

The 18-month exercise and dietary intervention study conducted in Taiwan among the elderly showed a 30.4% reduction in MetS prevalence (Shu-Hung et al., 2019). The reduction in the prevalence of MetS in this report (30.4%) is much lower than that of our finding. The likely explanation for such difference is that the participants in our study were adults aged 19-65 years, while in the taiwanese study, they were elderly (≥ 65 years old). Aging is significantly linked to several MetS risk factors namely insulin resistance, inflammatory process, reduction of baroreceptors' action, kidneys' buffering process as well as stiffens of blood vessels (Penuela and Penuela, 2015, Guarner-Lans *et al.,* 2011, Howlett, 2010).

A systematic review of lifestyle intervention reported a 39% decline in the prevalence of MetS and substantial improvement in WC, BP, TGs, and FBG levels and in the group who received the intervention relative to controls (Maria *et al.*, 2019), which is much lower than our finding. Again the difference in the decline in the prevalence of MetS between our study (45.5%) and the systematic review (39%) could attributed to differences in study settings, socio-demographic of the studied population and the criteria used to define MetS.

Concerning the components of MetS, similar to our findings, a recently carried out study among obese adults in Saudi Arabia observed a remarkable improvement in body weight, WC, FBG, systolic and diastolic BP, and TGs levels as well as BMI (Mohamed *et al.,* 2020). Furthermore, a 6-month community-based exercise intervention in obese adults with MetS in Taiwan noticed a substantial decrease in body weight, BMI, WC, BP, FBG, and an increase in HDL-C (Shu-Hung *et al.,* 2016). The Tehran Lipid and Glucose longitudinal study proved the beneficial effect of a long-term lifestyle intervention on MetS and all its components in individuals with impaired glucose tolerance (Fereidoun *et al.*, 2013). Furthermore, evidence indicates that adopting a healthy lifestyle can prevent or delay the progression of pre-diabetes to diabetes (Muraki *et al.,* 2013). Moreover, several lifestyle interventions carried out among adults with type-2 diabetes achieved a positive effect on the prevalence of MetS and its metabolic markers (Shehu *et al.,* 2017, Muchiri *et al.*, 2015, Askari *et al.*, 2013).

This piece of evidence confirms the necessity of lifestyle changes to control MetS and therefore prevent CVDs including type-2 diabetes. Indeed, in this study, the changes in diet and physical activity have contributed to the substantial reduction in the prevalence of MetS and its elements as well as anthropometric indices in the intervention relative to the control group. There was a marked increase in the proportion of subjects who improved patterns of dietary intake as well as engaged in optimal physical activity within the intervention group at the end-line. Nutrition-linked education intervention is a crucial part of CVDs management which has been proved to improve dietary behaviour and clinical outcomes in patients with diabetes (Maryam *et al.*, 2019, Muchiri *et al.*, 2016, Liu *et al.*, 2015). Adoption of a healthy diet and participation in an adequate level of physical activity are recommended as the first-line interventions to control MetS (Guzman *et al.*, 2019, Gerstel *et al.*, 2013). Evidence by Saboya *et al.* (2016), Blackford *et al.* (2016), and Gerstel *et al*. (2013) revealed the importance of dietary intervention on MetS and its elements. Hence; early identification of individuals with pre-diabetes such as those diagnosed with MetS is a potential approach to reverse, stop or slow the progression to diabetes by offering a cost-effective intervention model in environments with resource constraints.

On the other hand, it is of interest that the control group showed improvements in MetS prevalence and some of its components including BP, TGs and HDL-C. Thus, it appears that they also changed their behaviour as evidenced by the improvement in diet and physical activity during the follow-up. Such behaviour changes might have resulted from the introduction session of post-randomization; and the regular data collection visits and contacts. Moreover, individuals from the group who received the health education intervention might be in contact with the control group and perhaps have positively influenced their behaviour. This implies that even a minimal information had impact on behavioural changes and reduction in CVDs risks.

5.3 Change in dietary pattern and improvement of MetS

Relative to the control, participants in the intervention group showed a remarkable improvement in their dietary intake patterns at the end of the intervention. This indicates that the community-based health education intervention had a positive effect in improving individuals' dietary behaviour. Indeed, when people are aware of the benefits of a given behaviour, they are more likely to change their behavour. These findings are important because increasing fruits, vegetables, legumes, nuts, and decreasing carbohydrate intake would eventually increase dietary fibre, calcium, and vitamin intake, which is considered a healthy dietary pattern (Ganasegeran et al., 2012, Poddar *et al.,* 2009). After adjusting for socio-demographics, intake of fruits, processed foods, alcohol, and physical activity in the intervention group and legumes and physical activity in the control group were independently associated with MetS. Hence, community-based interventions to address MetS and reduce risks of CVDs should incorporate awareness creation strategies on healthy lifestyle involving diet, exercise and reduction of alcohol.

5.4 Adherence to the DASH diet and improvement of MetS

Adherence to the DASH diet in both groups was very low at baseline; majority of the respondents consumed below the recommended portion of a plate as vegetables and/or fruits. There was a marked increase in the proportion of subjects in the intervention who adhered to the DASH diet compared to the control group at the end-line. The DASH diet has been considered as an affordable and effective intervention to reduce the burden of CVDs in a population (Dori, 2017). In accordance to our findings, a 12-week community-based DASH intervention in the united stated among low-resource urban African-American Communities (Whitt-Glover *et al.,* 2013) and Iran (Mehrabian *et al.,* 2018), observed a significant increase in adherence to the DASH diet in the treatment relative to the control arm.

In our study, individuals in both groups who adhered to the DASH diet had substantially lower rate of MetS at a bivariate analysis. The beneficial effect of adherence to the DASH diet on MetS, type-2 diabetes, and CVDs has been well established (Jeffrey *et al.,* 2017, Babio *et al.,* 2014, Salehi *et al.,* 2013, Levitan *et al.,* 2013). Furthermore, evidence has revealed that the DASH diet reduced the risk of MetS by up to 81% (Asghari *et al.,* 2016, Saneei *et al.,* 2015). According to Asemi *et al.* (2014) DASH diet had a favorable impact on MetS components including obesity, hypertension, and dyslipidemia. However, lack of adherence to DASH diet as characterized by excess consumption of refined carbohydrates (Edyta *et al.,* 2017, Ameyalli *et al.,* 2015) and animal-based proteins (Maowei *et al.,* 2017) increase the risk of MetS. Hence, there is need for community awareness on importance of DASH diet as a CVDs prevention starategy.

5.5 Legumes and nuts intake and improvement of MetS

Legumes and nuts intake was very low, only one-fifth of each group consumed the recommended frequency at the baseline. Consumption of legumes and nuts significantly improved and was doubled in the intervention relative to the control arm at endline. In this study, individuals who consumed the recommended frequency of legumes and nuts had a lower rate of MetS. Similar to our findings, studies have reported the beneficial effect of legumes intake on MetS (Mahan *et al.*, 2016, Hosseinpour *et al.*, 2015) and all its components (Firouzeh *et al.*, 2014). Controlled trial studies have proved the beneficial effects of legumes intake on the elements of MetS namely raised TGs and high BP (Jayalath *et al.*, 2014, Bazzano *et al.*, 2011, Mozaffarian *et al.*, 2011) and raised blood glucose and lipid levels (Ley *et al.*, 2014).

Regarding nuts, several interventional studies proved that regular consumption of nuts reduced waist circumference (Hang *et al.*, 2018, Blanco *et al.*, 2014), BP (Jayalath *et al.*, 2014, Blanco *et al.*, 2014), blood sugar (Afshin *et al.*, 2014, Blanco *et al.*, 2014) and TGs (Sabate *et al.*, 2010, Blanco *et al.*, 2014)- components of MetS. Current evidence showed that consumining both legumes (Sala-Vila *et al.*, 2015) and nuts (Yoona *et al.*, 2019) have an added beneficial effect to control MetS and prevent CVDs including type-2 diabetes. Therefore, it is highly recommended for community awareness programme on the importance of regular intake of legumes and nuts to reduce the burden of CVDs in the community.

Legumes have a high content of viscous soluble fibres which reduce the absorption of bile salts, cholesterol, and carbohydrates in the gut. This improves blood lipid (Ameyalli *et al.*, 2015, Visioli, 2011) and sugar levels (Bouchenak & Lamri-Senhadji, 2013; Hutchins et al., 2012, Sievenpiper *et al.*, 2009), and therefore lower risk of MetS and CVDs. Soluble fibres also reduce blood cholesterol levels by increasing the excretion of bile acid/salt in feces (Gunness & Gidley, 2010). Moreover, the protein component of legumes contributes to its MetS reducing effect through modulating plasma lipids and displacing saturated lipids found in animal-based proteins with healthy plant-based proteins (Rebello *et al.*, 2014).

Nuts are full of fibre; tocopherols with antioxidant properties and bioactive nutrients (phytochemicals), which all have MetS reducing effect by controlling inflammation, oxidative stress, and endothelial function (Ros and Hu, 2013). These activities increase the sensitivity of insulin and, therefore, lower the risk of MetS and its components including obesity, blood lipid levels, type-2 diabetes, and hypertension (Ros, 2010, Bouchenak & Lamri-Senhadji, 2013; Hutchins *et al.*, 2012, Visioli, 2011).

5.6 Vegetables and fruits intake and improvement of MetS

Consumption of vegetables and fruits was low in both groups at baseline. In each group, approximately one-sixth and two-thirds daily consumed fruits and vegetables, respectively, at baseline. In line with our baseline findings, a local study in the Slums of Nairobi found a high prevalence (74%) of insufficient fruit and vegetable intake (Frederick *et al.,* 2020, Hulzebosch *et al.,* 2015). There was a marked increase in the proportion of subjects in the intervention who daily consumed fruits and vegetables compared to the control group at the end-line. Specifically, the proportion of subjects who daily consumed fruits was almost tripled in the intervention group at the end-line relative to the baseline. This shows that when people are properly informed about importance of a a healthy lifestyle, they are more likely to changes.

The improvement in fruit and vegetable consumption in the intervention group is consistent with a randomized controlled trial study in Malaysia that showed fruit consumption had increased almost three times in the treatment group compared to control after 10-week intervention (Mohd *et al.,* 2013). A nutritional intervention conducted in Brazil observed a substantial improvement in vegetable intake in the intervention relative to the control arm at the end of the intervention (Fonseca *et al.,* 2019). Another study by Petrella *et al.* (2014) who evaluated a nutritional intervention in overweight and obese pregnant women, observed the group who received the intervention significantly increased consumption of vegetables and fruits.

In our study, MetS was substantially less prevalent among individuals in the intervention group who daily consumed fruits and vegetables. In accordance to our findings, locally, evidence by Okube *et al.* (2020) showed that individuals who daily consumed fruits and vegetables were less likely to have MetS. Another local study by Kimani and colleagues (2019) indicated that the rates of obesity, high BP, and dyslipidemia- some of the components of MetS, were lower among hypertensive patients who daily consumed fruits and vegetables. Studies in Cameroon (Dabou et al., 2018) and Korea (Choi *et al.,* 2015) reported an indirect association between fruit intake and MetS as well as its elements (Rimkyo *et al.,* 2017). Moreover, a meta-analysis of five studies revealed that regular consumption of vegetables & fruits was strongly correlated with a lower risk of MetS (Li *et al.,* 2017, Mecca *et al.*, 2012), type-2 diabetes (Cooper *et al.,* 2012, Carter *et al.,* 2010) and hypertension (Wang *et al.,* 2014, Nunez-Córdoba *et al.,* 2009). In contrast, a report from one of the informal settlements of Nairobi showed a high rate of obesity linked to a lack of vegetables and fruit intake (Gyakobo *et al.,* 2012). It is, threfere, highly recommended for community-based health education intervention to create public awareness on the benefits of fruits and vegetables consumption to prevent and control CVDs.

The favorable effect of fruits and vegetables intake on MetS is attributed to their richness in bioactive micronutrients such as flavonoids, phytochemicals, antioxidants, fibres, potassium, and vitamins. Phytochemicals and flavonoids are extremely rich in antioxidants and anti-inflammatory properties (Hooper *et al.,* 2008, Erdman *et al.,* 2007) which have MetS and CVDs protective effects (Pamela *et al.,* 2007). Moreover, vegetables and fruits have plenty of soluble fibres which decrease absorption of cholesterol and bile salts in the gut and, therefore, improve blood lipid levels (Ameyalli *et al.,* 2015, Visioli, 2011). Additionally, vegetables and fruits have a high concentration of potassium, a beneficial co-factor to regulate BP. If the potassium level in the blood is normal, the body excretes more sodium and water and, therefore, the BP remains normal. Whereas, if the blood concentration of potassium is low, the body retains more sodium and water causing high BP (Rheinschild, 2017, Savica *et al.,* 2010).

5.7 Processed foods intake and improvement of MetS

Approximately one-third of the respondents in each group often consumed processed/fast foods before the intervention. There was a marked decrease in the proportion of subjects in the intervention group who often consumed processed/fast foods compared to the control group at the end-line. This indicates that when people are properly educated and aware of the benefits and risks of a given behaviour, they are more likely to change their behaviour. In line with this finding, randomized controlled trial studies of nutritional intervention in Brazil (Fonseca *et al.,* 2019) and

Malaysian (Mohd *et al.*, 2013) showed that their intervention group significantly reduced processed/fast food intake compared to the control group.

In our study, changes in processed/fast foods intake had a substantial effect on MetS. MetS was more prevalent among individuals in both groups who often consumed processed/fast foods at the end-line. This underscores the importance of educating the public to properly select healthy foods to control MetS and prevent CVDs. Similar to our findings, interventional studies carried out for 1-year (Babio *et al.*, 2012) and 3-years (Bahadoran *et al.*, 2013) reported a high incidence rate of MetS associated with consumption of processed foods. Furthermore, evidence showed that high intake of fast foods such as fried chicken, sausage, samosas was markedly linked to MetS (Krishnan *et al.*, 2010, Micha *et al.*, 2010) and its components (Edyta *et al.*, 2017, Rodriguez et al., 2017, Rimkyo *et al.*, 2017). Processed/fast food intake was evaluated as the primary cause of obesity, a principal factor for the development of MetS (Garcia *et al.*, 2012, Mozaffarian *et al.*, 2011). Implementation of a community-based approach of dietary awareness is needed to reduce the burden of CVDs.

Several mechanisms are describing the association between processed food intake and MetS. The nutritional content of fast foods is mainly refined carbohydrates, saturated fat, salt and processed or simple sugars- which favours development of obesity and MetS (WHO, 2017-a, WHO, 2015, Paniagua et al., 2011). Saturated fat from processed/fast foods increases visceral adiposity which in turn reduces activation of PGC-1a (PPARg-coactivator). This leads to decrease oxidation of glucose and fatty-acid and, therefore, increase fat accumulation in tissues causing obesity, type-2 diabetes and MetS (Kennedy et al., 2009). Further, rifined carbohydrates and high glycemic index foods may increase fat storage and insulin insensitivity- principal causes of MetS (Finley *et al.*, 2010).

5.8 Changes in salt and sugar intake and improvement of MetS

The WHO strongly recommends a reduction of salt intake to ≤ 1tea spoon per day (WHO, 2012-b) and sugar intake to ≤ 5 tea spoons per day (WHO, 2015) to reduce BP and risks of CVDs in adults. In our study, only one-third of each group consumed the recommended amount of salt and sugar at baseline. There was a marked increase in the proportion of subjects in the intervention group who consumed the recommended amount of salt and sugar relative to the control arm at the end of the intervention. Furthermore, there was a marked decrease in daily intake of salt (from a mean of 1.51 tea spoon to 1.16) and sugar (from a mean of 5.54 tea spoon to 4.40) in the intevention group at the end-line relative to the baseline. Whereas, in the control group, no significant change was observed in terms of daily mean salt and sugar intake at the endline compared to the baseline. This shows that the community-based lifestyle intervention played a significant role in awareness creation about the importance of salt and sugar reduction to control MetS and CVDs. Implementation of a community-based intervention to promote public awareness of dietary changes is feasible and effective approach.

Similar to our findings, community-based dietary education interventions conducted in Ghana (Cappuccio *et al.*, 2006) Japan (Takahashi *et al.*, 2006) reported a substantial reduction in salt intake in the treatment group after 12-months follow-up. Other studies reported a considerable reduction in sugar intake in the group who received the intervention relative to the control group (Elisa and colleagues, 2019, Petrella *et al.*, 2014). As CVDs risk reduction intervention, the WHO has recommended salt reduction as cost-effective and feasible strategy to implement at the community level (Alwan, 2011).

Our findings underscore the need for urgent interventions that promote behaviour change among the public in Kenya to reduce the problem of CVDs. This calls for population-based health education interventions to improve awareness of the public of the dangers of unhealthy diet and beneficial effects of adopting healthy dietary patterns. Although there was a direct association between sugar intake and MetS at bivariate analysis in the intervention group, the relationship did not persist at multivariate analysis. Further, there was no noticeable relationship between salt intake and MetS in both groups.

5.9 Changes in alcohol intake and improvement of MetS

Approximately, a quarter of the participants were alcohol consumers both at the baseline and end-line with no noticeable difference between the two groups. There was a marked reduction in both frequency and quantity of alcohol intake in the intervention group at the endline relative to the baseline. While, in the control group, no substantial change was observed in terms of frequency and amount of alcohol intake at the end-line compared to baseline. This indicates that the community-based lifestyle intervention contributed a significant role in changing individuals' behavior towards alcohol consumption. Our finding was in agreement with reports by Brites and colleagues (2019) and Ettner *et al*. (2014), who found that their intervention group significantly reduced in frequency and amount of alcohol consumption compared to the control group. The WHO evidence shows that behavioural advice is an effective intervention to reduce alcohol intake and its harmful effects in the community (WHO, 2009-d).

In our study, MetS was more prevalent among subjects in the intervention group who consumed above the recommended amount of alcohol. In accordance to our finding, evidence has shown that excessive alcohol intake was substantially linked to MetS (Sun *et al.,* 2014, Wakabayashi, 2014, Slagter *et al.*, 2014) and its components namely abdominal obesity, TGs level (Wakabayashi, 2013, Chen *et al.*, 2012), BP (Kimani *et al.*, 2019, Kaur, 2014, Lee, 2012), and hyperglycemia (Cullmann et al., 2012, Whitfield *et al.*, 2013).

The plausible mechanism involving in the pathogenesis of alcohol-induced MetS can be explained as follows: Excess alcohol consumption causes obesity (Sayon-Orea *et al.*, 2011), which leads to other several biochemical and clinical abnormalities, including dyslipidemia, hypertension, insulin insensitivity and type-2 diabetes- all are elements of MetS (Popkin et al., 2012, Misra *et al.*, 2011). Furthermore, excessive alcohol intake abnormally activates the sympathetic nervous system, causes direct vasoconstriction, increases cardiac contractility and impairs baro-receptor refluxes leading to disruption of auto-regulation mechanism, resulting in high BP (Rehm et al., 2010). Moreover, high dose of alcohol intake induces inflammation, down regulates the insulin signaling cascade and decreases expression of glucose transporters. This causs insulin insensitivity and, therefore, increases risk of MetS and type-2 diabetes (Nguyen *et al.*, 2012, Ronis *et al.*, 2007).

5.10 Level of physical activity and improvement of MetS

In our study, approximately one-fifth of the respondents of each group participated for the recommended level of physical activity at the baseline. Self-reported physical activity level markedly increased in the intervention group at the end-line. Subjects who participated for the recommended level of physical activity increased by 41% in the intervention compared with 8% in the control arm at the end-line. Indeed, the community-based lifestyle intervention increased peoples' awareness of the benefits of physical activity to control MetS and prevent CVDs.

In accordance to our finding, a 12-month interventional studies carried out in Switzerland (Gerstel *et al.,* 2013) Japan (Takashi *et al.,* 2007) revealed that the intervention group significantly increased level of physical activity relative to the control group. Likewise, Sarah and colleagues (2014) reported that 12-weeks lifestyle intervention showed significant improvements in exercise and eating habits in premenopausal women. Meta-Analyses of community-based physical activity intervention among adults showed a substantial increase in physical activity status in the treatment relative to the control arm (Jo-Ana, 2015, Vicki *et al.*, 2011). Evidence by Osborn and colleagues (2010) based on information-motivation-behavioral skills, in adults with diabetes (type-2), found that the intervention arm increased the duration, and frequency of physical activity than the control.

In our study, the change in physical activity had a favorable effect on MetS. MetS was substantially less prevalent among individuals in both groups who participated for the recommended level of physical activity at the endline. It is also interesting to note that there was a marked decrease in mean heart rate in the intervention group at the endline relative to the baseline. The reduced heart rate suggests that the respondents increased their physical activity (Anne *et al.,* 2018). Our findings are in line with prior several observations in Taiwan (Jui-Hua *et al.,* 2017), the United States (Kastorini *et al.,* 2011) and Korea (Junga *et al.,* 2016) which have shown the protective effect of sufficient physical activity against MetS and its elements.

Certainly, physical activity is one of the most crucial modifiable lifestyle factors to prevent and control CVD risk factors including high BP and diabetes and MetS (Anne *et al.*, 2018, Hahn *et al.*, 2009, Strasser, 2013). According to the WHO (2009-b), participation in an adequate level of physical activity is estimated to reduce the risks of MetS by 20% and diabetes by 27%. Conversely, an increased amount of sedentary lifestyle was linked to an elevated risk of MetS and its elements, namely reduced HDL-C, increased FBG, and raised TGs (Gennuso *et al.*, 2014, Thorp *et al.*, 2013, Prasad *et al.*, 2012), main CVDs risk factors. This study showed increased awareness of people on the benefits of physical activity to prevent CVDs. We recommend for urban planners to create a conducive environment for people to participate in physical activities at their convenience.

The beneficial effect of physical activity against MetS can be explained by several facts. Sufficient physical activity burns excessive fat, reduces body weight, BP, and improves insulin sensitivity as well as endothelial function, which all have a positive effect on lipid levels, MetS, type-2 diabetes, and CVDs (Cornier *et al.,* 2008). Regular physical activity promotes vasodilation by increasing vascular nitric oxide concentration, decreases the release of inflammatory mediators from skeletal muscle and adipose tissue, and regulates autonomic imbalance (Bowles and Laughlin, 2011). These mechanisms have remarkable and beneficial effects on MetS and CVDs. Whereas, insufficient physical activity promotes weight gain, insulin resistance and makes blood vessels stiff, causing high BP, type-2 diabetes, and MetS (Bassuk and Manson, 2010).

5.11 Relationship between changes in tobacco use and improvement of MetS

There was no significant change in smoking status in both groups at the endline compatred to the baseline. Further, there was no relationship between tobacco use and MetS at the endline. However, several prospective cohort studies reported a direct correlation between tobacco use and MetS (Sun *et al.,* 2012, Mikael *et al.,* 2013, Slagter *et al.,* 2013). The lack of relationship between smoking and MetS in our study could be due to the small number of smokers.

5.12 Socio-demographic variables associated with Metabolic Syndrome

At multivariate analysis, age, religion, and occupation in the intervention, and age and income in the control groups were significantly associated with MetS at the end-line. Our findings revealed that aged respondents (≥ 50 years), in both groups, were more likely to have MetS. Whereas, MetS was significantly less prevalent among respondents in the intervention group who were employed and Muslims. MetS was more common among respondents in the control group who had a higher monthly income. This finding is consistent with several recently carried out studies (Okubatsion *et al.,* 2020, Iqbal et al., 2020, Leila *et al.,* 2019, Jennifer and Chris, 2010), which have shown a direct association between age and MetS.

Aging is significantly linked to several MetS-CVDs risk factors namely insulin resistance, inflammatory process, reduction of baroreceptors' action, kidneys' buffering process as well as stiffens of blood vessels (Penuela and Penuela, 2015, Guarner-Lans *et al.*, 2011, Howlett, 2010, Banos *et al.,* 2009). Specifically, for women, this can be justified by hormonal changes over time due to decreased estrogen level (Giralt *et al.*, 2012). Additionally, CVDs are positively associated with unemployment status (Zagozdzon *et al.,* 2014, Menetop *et al.,* 2015). Unhealthy lifestyle including smoking, poor diet, alcohol consumption and lack of exercise are positively associated with unemployment (Zagozdzon *et al.,* 2014, Naimi *et al.,* 2009), which are the major risk factors of CVDs. In this case, lack of employment causes financial insecurity and may adversely affects economic status, which may lead to health damaging behaviours (Naimi *et al.,* 2009).

In this study, Muslim respondents in the intervention group displayed a lower prevalence of MetS compared to Protestants and Catholics. The likely explanation for this finding could be lifestyle differences between Muslims and other religions. For example, Muslims do not consume alcohol, a major risk factor of MetS (Wakabayashi, 2014, Briasoulis *et al.*, 2012). Another factor could be the effect of Ramadan Fasting. A study conducted by Amena *et al.* (2011) showed the protective effect of Ramadan Fasting on MetS. However, further studies may be needed to establish whether the same association could be repeated.

In our study, MetS was less prevalent among employed compared to unemployed individuals at the end of the intervention. The possible explanation for this association is that unemployed respondents are most probably aged and retired and therefore, at increased risk for age-related MetS and CVDs. Indeed, aging is substantially associated with insulin resistance, hardening of blood vessels, declining baroreceptor action, and kidneys' buffering activity, which all can contribute to high BP, diabetes, and MetS (Guarner-Lans *et al.,* 2011, Banos *et al.,* 2009).

In the control group, MetS was more common among respondents who had a higher monthly income. This finding is in agreement with several studies in India (Hulzebosch *et al.*, 2015), China (Yiqiang et al., 2012), and Saudi Arabia (Mabry *et al.*, 2010) which have reported a direct association between income and MetS. However, studies in developed countries found an inverse association between income and MetS (Hyunjung *et al.*, 2012, Mackenbach *et al.*, 2008). This discrepancy can be explained by the lifestyle difference between developed and developing countries. High-income earners in developing countries frequently consume unhealthy fatty foods/sugars and have a sedentary form of lifestyle. While those in the developed countries consume quality foods and have an active lifestyle (Micklesfield *et al.*, 2013). These findings indicate that intervention programmes should be planned according to the socio-demographic information of individuals.

5.13 Level of knowledge on risk factors and preventive practices of CVDs

The respondents' level of knowledge on lifestyle risk factors and preventive measures of CVDs was evaluated before and after the intervention. The baseline findings showed that most of the respondents, in both groups, scored a low level of knowledge on risk factors and preventive measures of CVDs. Majority of the respondents in each group scored low levels of knowledge on tobacco smoking, alcohol consumption, excessive salt and sugar intake, consumption of processed/fast foods, and being overweight/obese as CVD risk factors at the baseline. Similarly, most of the respondents, in both groups, scored a low level of knowledge on preventive measures of CVDs, the recommended daily intake of sugar and salt, the importance of vegetables and fruits intake as well as engaging in physical activity on CVDs prevention.

Of the respondents, only, 15.4% of the intervention and 7.2% of the control groups able to list at least 3 risk factors or 3 preventive measures for CVDs at baseline. Overall, majority of the respondents had low level of knowledge towards the major CVDs risk factors and prevention measures at the baseline. The overall lack of CVD risk awareness indicates that there is lack of population-based preventive approaches. This highlights the necessity for designing more targeted educational programmes to increase the public's awareness regarding CVDs.

In line with our baseline findings, a local study among HIV-positive people (Tecla *et al.,* 2015), found a low level of knowledge (mean score of 1.3 out of possible 10 points) on CVDs risk factors. A cross-sectional study conducted in the informal settlements of Korogocho and Viwandani of Nairobi among diabetic/hypertensive patients showed that 41.8% of the respondents did not know any risk factor of diabetes/hypertension (Gladys *et al.*, 2014). Likewise, Oti *et al*. (2013) reported a low level of awareness on the control measures of hypertension and diabetes in low-resourced settings in Nairobi. Studies carried out in the semi-urban community in Uganda (Rawlance et al., 2020), South Africa (Surka *et al.*, 2015), Nigeria (Oladapo *et al.*, 2013), and Benin (Cossi *et al.*, 2012), reported that the majority of their respondents had a low level of knowledge on risk factors and preventive measures of CVDs.

Indeed, lack of adequate knowledge of CVDs risk factors and preventive measures has negative consequences on the outcome of a disease. For example, in Kenya, most people living with diabetes (WHO, 2014-a) and hypertension (Shukri *et al.*, 2018) are diagnosed too late; when complications have already set in (WHO, 2014-a). Indeed, in Kenya, prevention, early detection and management of CVDs is lacking. This calls for community-based intervention programmes to educate people on early prevention and control measures of CVDs.

There was a marked improvement in level of knowledge on the major risk factors and preventive measures of CVDs in the intervention relative to the control group at the end-line. The overall level of knowledge of CVDs risk factors and prevention measures substantially improved in the intervention relative to the control arm at the endline. This indicates that the health education intervention significantly promoted their level of knowledge on CVDs after the intervention. This may indicate the feasibility and effectiveness of health education intervention to create public awareness on prevention and control measures of CVDs. Hence, there is an immediate need to implement population-based health educational interventions to promote public awareness on modifiable risk factors and preventive mreasures of CVD. Implementation of awareness-raising strategies at a grassroots level will have a positive impact on the greater community.

In line with our end-line finding, a community-based health education intervention on nutrition in Portuguese adults with diabetes observed a substantially higher level of knowledge in the treatment group than the controls (Carlos *et al.,* 2019). Similarly, studies carried out in Egypt (Abdel-Fatah *et al.*, 2017) and Brazil (Fonseca *et al.,* 2019) regarding dietary changes in adults found that the intervention group significantly scored a higher level of knowledge than the controls at the endline relative to the baseline. Thus, the community-based health education programme may be an effective model to create and/or raise awareness of a healthy lifestyle and reduce the burden of CVDs in the community. Behavioral risk factors of CVDs can be addressed through nurses and community health workers by educating and encouraging people about lifestyle modifications, such as regular physical activity, eating healthy foods, and utilizing preventive health services.

5.14 Application of the Transtheoretical Model on MetS control-related lifestyle changes

The Transtheoretical Model (TTM) of behaviour change was used to determine baseline and end-line respondents' stage of changes, self-efficacy and decisional balance towards a healthy lifestyle practice to control MetS and prevent CVDs. A majority of the respondents were in the pre-action stage of change for dietary changes (vegetables, fruits, processed foods, salt and sugar intake, utilization of the DASH diet, alcohol use), and physical activity at the baseline. The overall level of adherence to a healthy lifestyle was not substantially different between the groups at the baseline. The proportion of subjects in the maintenance stage of change for dietary changes (fruits and vegetables, salt, sugar, processed/fast foods, adherence to the DASH diet) and physical activity markedly improved in the intervention relative to the control group at the end-line. The overall level of change to a healthy lifestyle was substantially higher in the intervention relative to the control arm.

Regarding self-efficacy towards a healthy lifestyle practice, at baseline, majority of the respondents were either not at all or somewhat confident to take the recommended dietary patterns (fruits and vegetables, salt, sugar, processed/fast foods, adhere to the DASH diet), stop/moderate alcohol intake and participate in the recommended level of physical activity with no significant difference between the two groups. At the end-line, the intervention group substantially increased their level of confidence towards a healthy lifestyle practice than to the control arm. The proportion of people who were extremely confident to take the recommended dietary patterns (fruits and vegetables, salt and sugar, adhere to the DASH diet, avoid/limit processed/fast foods) and participate in an adequate level of physical activity was markedly improved in the intervention relative to the control arm at the end-line.

With regards to the pros of decisional balance, at the baseline, majority of the respondents agreed that regular intake of vegetables and fruits, limiting sugar, salt, and processed foods as well as participating in the recommended level of physical activity could help prevent hypertension, diabetes and lose or maintain a healthy weight. However, most of the respondents in both groups agreed that they cannot find reasonably priced fruits and vegetables in their local markets. Of the respondents, only below one-third, agreed that limiting/avoiding alcohol consumption could help prevent hypertension and diabetes. The pros of decisional balance on adopting a new behaviour to control MetS was significantly improved in the intervention arm relative to the control at the end-line. The proportion of people who agreed that adherence to the recommended dietary intake patterns, alcohol intake, and level of physical activity could help prevent hypertension and diabetes, lose/maintain a healthy weight was substantially improved in the intervention relative to the control group. Compared to baseline, at the end-line, both groups significantly scored a higher mean score for pros of decisional balance.

Concerning cons of decisional balance on adopting a new behaviour, most of the respondents, in both groups, agreed that taking the daily recommended servings of fruits and vegetables is difficult because of high price, consequently, had limited ways to incorporate fruits and vegetables in their daily meals. Moreover, one-third of the respondents in each group agreed that they fear chemicals used in fruits and vegetables. Most of them agreed that it is difficult to avoid or limit processed/fast foods because they are much available than natural foods. Of the alcohol consumers, a majority agreed that it is difficult to limit/avoid it. Approximately one-third of each group agreed that they have a shortage of time to participate in adequate physical activity. In both groups, there was a marked increase in mean scores of pros of decisional balance and decrease in mean scores of cons of decisional balance at the end-line compared to baseline.

In summary, our findings indicate that majority of the intervention group proceeded to the maintenance stage of lifestyle change after the TTM-based health education intervention. Furthermore, the intervention group significantly increased mean scores of stages of change, self-efficacy, pros of decisional balance, and decreased cons of decisional balance concerning lifestyle changes at the end-line relative to the baseline. The control group also significantly increased mean scores of stages of change and pros of decisional balance and decreased cons of decisional balance at the end-line relative to the baseline. These findings show that the TTM-based health education intervention increased individuals' efficacy of perceived benefits (pros), and decreased their negative perceptions (cons).

Our baseline findings concur with a report by Holmen et al (2016), who had evaluated "stages of change for physical activity and dietary habits among patients with type-2 diabetes", which showed that majority of their participants were in pre-action stage for physical activity and dietary change at baseline. Maryam (2013) who had applied the TTM to assess exercise status among Iranian officers found that majority of the subjects were in the pre-action stage of change for physical activity. As the person changes from one stage to another, the cons for the new behaviour are expected to decrease and the pros are expected to increase, hence a positive behavioural change occurs successfully. Our findings revealed that perceived pros have markedly increased at the maintenance stage than pre-action, and perceived cons have decreased at the maintenance stage than pre-action which is in line with reports of other researchers (Salehi *et al.*, 2010, Kang *et al.*, 2012).

Indeed, our end-line findings concur with a study done by Nitzke *et al.* (2007) who had determined the efficacy of a health education intervention to promote vegetables and fruits intake of low-income adults. The study reported that the intervention group significantly improved vegetables and fruits intake, and greatly progressed to the maintenance stage than the control group after the TTM-based intervention. Similar to our findings, a study conducted in Egypt (Abdel-Fatah *et al.*, 2017) regarding dietary changes among pregnant women using the TTM, found that the group who received the intervention significantly improved their self-efficacy, decisional balance, and stage of change than the control group at the endline relative to the baseline. Mohsen *et al.* (2014), who used the TTM to encourage lifestyle changes, reported a significant improvement in the mean scores of pros of decisional balance and self-efficacy regarding changes in dietary behaviour in the intervention relative to the control arm after the intervention.

The application of TTM of behaviour change was instrumental in improving respondents' behaviour towards a healthy lifestyle to control MetS. There was a marked increase in the proportion of subjects who improved patterns of dietary intake as well as engaged in optimal physical activity within the intervention group after the TTM-based health education intervention. The decline in the proportion of MetS and its components mirrored improvements in dietary intake patterns and physical activity uptake attributed to the TTM-based lifestyle intervention in the treatment group. Healthcare providers especially nurses should use the TTM while providing health education regarding lifestyle changes for individuals who are at risk for CVDs such as those diagnosed with MetS.

5.15 Implementation of community-based approach to CVD prevention through Nurses and CHWs involvement

Health education intervention focusing on specific lifestyle recommendations will inform the public to make healthier choices, which can lead to better prevention and control of CVDs. Nurses and Community Health Workers (CHWs) can implement activities contributing to the successful prevention of CVDs at the community level. This approach could involve a facility-based health education approach or community-linked awareness creating strategies such as outreach activity, school or church-based programmes. The effectiveness of nurses and CHWs could be attributed to their wider reach in many areas, rapport with community members, and their scope of practice to deliver holistic care (Khetan *et al.*, 2017, Hill *et al.*, 2017).

Based on our findings and recommendations, we have formulated a potential model for rolling out community-based approach to CVDs prevention through Nurses and CHWs involvement (Fig 14).

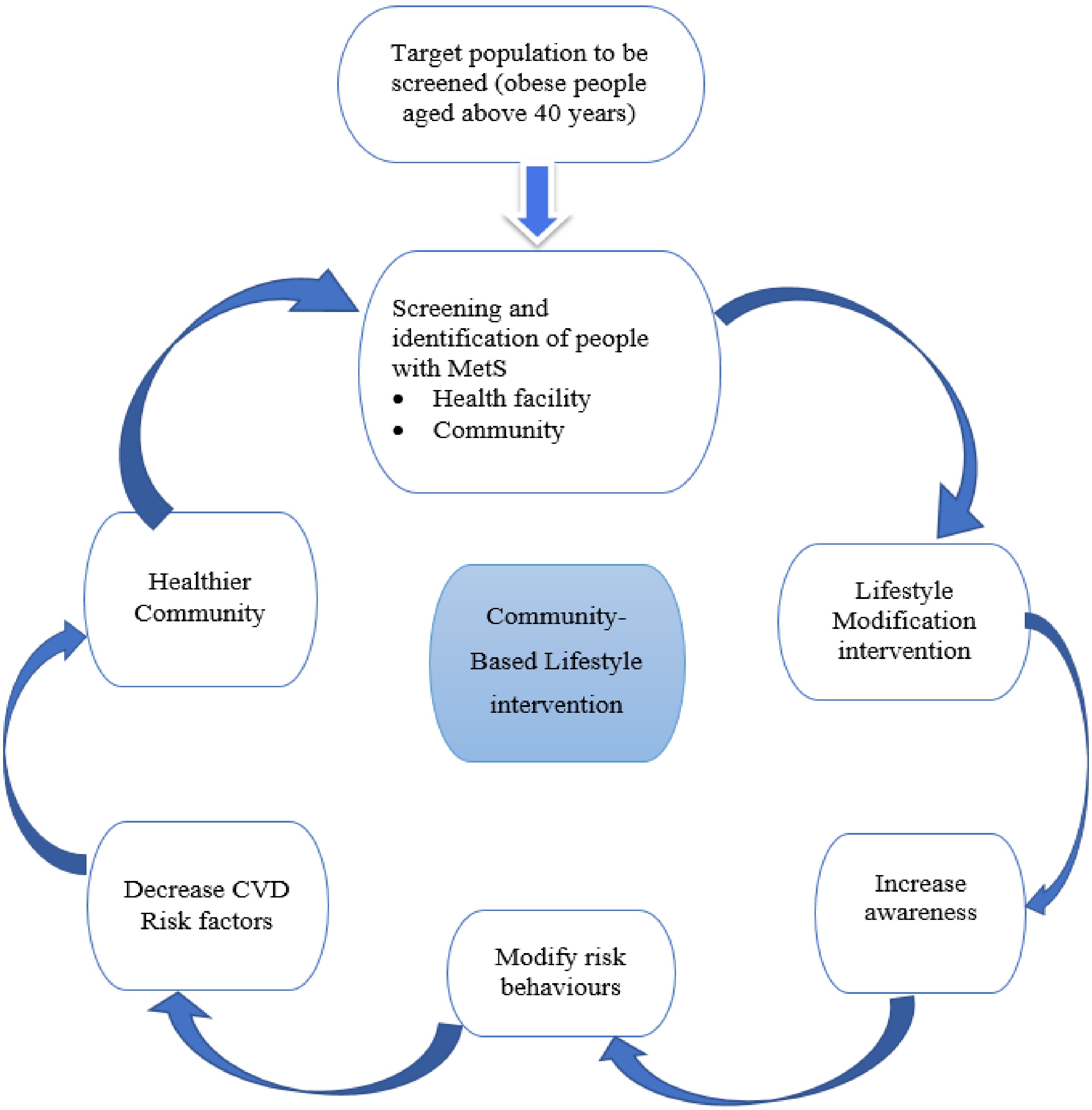


Figure 14: Model of community-based approach to cardiovascular disease (CVD) prevention through Nurses and CHWs involvement.

5.16 STRENGTH AND LIMITATIONS OF THE STUDY

STRENGTH OF THE STUDY

This study is premised on the rigorous randomized controlled trial design in which the findings are attributed to the lifestyle and not by chance. It presents novel findings that can have huge implication on the prevention and management of cardio-metabolic disorders through locally available resources at the community level. Furthermore, this is the first study to determine the effect of a community-based lifestyle intervention on MetS with indications that the model produced better outcomes of improving the proportion and levels of markers for MetS among adults in Kenya.

LIMITATIONS OF THE STUDY

The available data about MetS generally in Kenya and specifically at the study area is limited.In Kenya, no published reports are available regarding the application of TTM in addressing MetS. Moreover, there were no objective measurements of physical activity and food intake. This could probably have introduced some reporting bias because respondents might have overestimated the level of physical activity and underestimated or overestimated dietary intake after the intervention. However, this seems less likely because the reported changes showed consistent variations across laboratory variables, which were collected and analysed blindly.

5.17 SUMMARY KEY FINDINGS.

- The community-based lifestyle intervention is associated with the observed improvement in MetS and the related components including reduced central obesity, blood pressure, blood sugar, triglycerides and cholesterols.
- There was marked decline in the proportion of adults with MetS who were exposed to community-based lifestyle intervention signified the modifying effect of the model.
- The level of knowledge on the major risk factors and preventive measures of CVDs was improved following the community-based health education approach.
- There was increased consumption of vegetables, fruit, legumes and nuts that was linked to the improved metabolic parameters in the adults exposed to the community-based health education approach.
- A reduction in consumption of processed foods, sugar salt as well as alcohol helped improve the MetS markers in adults under the community-based health education intervention.
- The level of physical activity that culminated in the reduction of MetS was improved significantly in adults exposed to the community-based health education intervention.
- The community-based health education intervention helped most adults to proceed to maintenance stage of lifestyle change as per the the Transtheoretical Model (TTM) of behavioural change.

5.18 CONCLUSION

Community-based intervention supporting lifestyle changes was highly effective in reducing the major risk factors of CVDs. There was a marked improvement in the proportion of participants in the intervention group who met recommended lifestyle goals which significantly lowered the rate of MetS and its components at the end-line. Nearly one-third of the participants with MetS who received the community-based lifestyle intervention became free of the syndrome. Hence, our present outcome supports the alternative hypothesis that a community-based lifestyle intervention significantly reduced the prevalence of MetS and its related cardiovascular risk factors. Our findings demonstrate the feasibility and effectiveness of community-based lifestyle intervention to control MetS and reduce the risks of CVDs.

The level of knowledge of the major risk factors and preventive measures of CVDs markedly improved in the intervention relative to the control group at the end-line. Moreover, the application of TTM of behaviour change was instrumental in improving individuals' stage of change, self-efficacy, and decisional balance towards lifestyle modification to control MetS and thus prevent CVDs. Most participants in the intervention group proceeded to the maintenance stage of lifestyle change as per the TTM of behavioural change. This highlights the importance of the TTM as a useful framework to promote changes and evaluate respondents' progress towards a healthy lifestyle. The substantial lifestyle changes and reduction in the prevalence of MetS in the intervention group may provide a basis for the effectiveness and feasibility of community-based interventions to reduce CVDs in the community. This calls for environmental and policy changes to support healthy choices of individuals. Effective and sustainable CVDs prevention strategies require government and community support, policy changes, and continuous media campaign to raise public awareness. We have also formulated and provided a potential model for rolling out community-based approach to CVDs prevention through Nurses and CHWs involvement (Figure 14).

5.19 RECOMMENDATIONS

i. There is need for integration of community-based intervention for management of MetS and CVDs in the ongoing health programmes including the universal health care to leverage resources.

ii. Policy makers should develop capacity building strategies to train health care workers to equip them respond to the emerging threat of NCDs. This should be done through review of curriculum to incorporate NCDs in the preservice training, while developing training modules for the in-service health workers through continuous professional development programme.

iii. The community-based intervention for management of MetS and CVDs should include community awareness creation. This approach could involve facility-based health education approach or community-linked awareness strategy such as outreach activity, school or church-based programmes among others.

iv. Multi-sectoral collaboration and partnership with communities to promote production, availability and consumption of healthy locally available foods. This can be implemented through involvement of health care providers, educators, nutritionist, agriculture extension officers and farmers to promote consumption of locally available foods.

v. Awareness creation on the importance of exercises for improved health should be implemented. In regard to the urban population, the urban planners should plan and provide for safe spaces and recreational facilities for people to participate in physical activities at their convenience in cities and towns.

vi. To effectively mainstream community-based CVDs prevention approaches into the universal health care model, policies, standards and tools should be developed to facilitate the implementation of the management approach.

vii. Implementation research is required to test the efficacy if TTM in addressing peoples' behavior that contributed to CVDs.

viii. Large-scale studies are required to address the socio-economic, geographic and ethnic disparities in terms of metabolic outcomes.

REFERENCES

Abelson, R. (2010). New York: The New York Times. An insurer's new approach to diabetes. p. 14.

Abdel-Fatah Ibrahim, Hanan Abd Elwahab El Sayed and Ebtisam Mohamed Abd El-aal. (2017). Diet Behavior Modification of Pregnant Woman with Iron Deficiency Anemia Using Construct of the Trans-Theoretical Model: A Theory-Based Study. Journal of Nursing and Health Science (IOSR-JNHS). 6: (3), PP 72-85

Afshin A, Micha R, Khatibzadeh S, et al. (2014). Consumption of nuts and legumes and risk of incident ischemic heart disease, stroke, and diabetes: a systematic review and meta-analysis. Am J Clin Nutr. 100:278–288.

Akintunde A, Akintunde T, Opadijo O. (2015). Knowledge of heart disease risk factors among workers in a Nigerian University: A call for concern. Niger Med J [Internet]. 2015; 56(2):89. Available from: http://www. nigeriamedj.com/text.asp?2015/56/2/89/150688

Alberti KG, Eckel RH, Grundy SM, et al. (2009). International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; International Association for the Study of Obesity. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. Circulation. 120:1640–1645.

Alwan A. (2011). Global status report on non-communicable diseases 2010.Geneva, Switzerland: The World Health Organization.

Amelie Desgroppes, Sophie Taupin. (2011). Kibera: The Biggest Slum in Africa? Les Cahiers de l'Afriquede l'Est. 44, pp.23-34. halshs-00751833

Amena Sadiya, Solafa Ahmed, Hisham Hussain Siddieg et al. (2011). Effect of Ramadan fasting on metabolic markers, body composition, and dietary intake in Emiratis of Ajman (UAE) with metabolic syndrome. Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy 2011:4 409–416

Ameyalli Rodriguez-Cano, Jennifer Mier-Cabrera, Margie Balas-Nakash et al. (2015). Dietary changes associated with improvement of metabolic syndrome components in postmenopausal women receiving two different nutrition interventions. Menopause. 2015 Jul;22(7):758-64. doi: 10.1097/GME.0000000000000400.

Aminde LN, Takah N, Ngwasiri C, Noubiap JJ, Tindong M, Dzudie A, Veerman JL. (2017). Population awareness of cardiovascular disease and its risk factors in Buea, Cameroon. BMC Public Health. 17(1):545

Anne Kerstin Reimers, Guido Knapp and Carl-Detlev Reimers. (2018). Effects of Exercise on the Resting Heart Rate: A Systematic Review and Meta-Analysis of Interventional Studies. J. Clin. Med.7, 503; doi:10.3390/jcm7120503

Asemi Z, Samimi M, Tabassi Z, et al. (2014). Effects of DASH diet on lipid profiles and biomarkers of oxidative stress in overweight and obese women with polycystic ovary syndrome: a randomized clinical trial. Nutrition. 30 (11): 1287-1293.

Asghari G, Yuzbashian E, Mirmiran P, Hooshmand F, Najafi R1, Azizi F. (2016). Dietary Approaches to Stop Hypertension (DASH) Dietary Pattern Is Associated with Reduced Incidence of Metabolic Syndrome in Children and Adolescents. J Pediatr. 174:178-184.e1. doi: 10.1016/j.jpeds.2016.03.077.

Asghari G, Yuzbashian E, Mirmiran P,Mahmoodi B, Azizi F. (2015). Fast Food Intake Increases the Incidence of Metabolic Syndrome in Children and Adolescents: Tehran Lipid and Glucose Study. PLoS ONE. 10(10): e0139641. doi: 10.1371/journal.pone.0139641

Ashraf Kagee, Melvyn Freeman. (2017). International Encyclopedia of Public Health, Second Edition.

Askari F, Rabiei S, Rastmanesh R. (2013). The Effects of Nutrition Education and Diet Therapy on Glycemic and Lipidemic Control in Iranian Patients with Type 2 Diabetes. 3.

Assah FK, et al. (2011). Urbanization, Physical Activity, and Metabolic Health in Sub-Saharan Africa. Diabetes Care. 34(2): 491. DOI: https://doi.org/10.2337/dc10-0990

Babio N, Toledo E, Estruch R, et al. (2014). Mediterranean diets and metabolic syndrome status in the PREDIMED randomized trial. CMAJ. 186: E649–657

Babio N, Sorlı´M, Bullo´M, Basora J, Ibarrola-Jurado N et al. (2012). Association between red meat consumption and metabolic syndrome in aMediterranean population at high cardiovascular risk: cross-sectional and 1-yearfollow-up assessment. Nutr Metab Cardiovasc Dis. 22: 200–207.

Bahadoran.Z, Mirmiran.P, Hosseini-Esfahani.F and F Azizi.F.(2013). Fast food consumption and the risk of metabolic syndrome after3-years of follow-up: Tehran Lipid and Glucose Study. European Journal of Clinical Nutrition. 67, 1303–1309

Banos G, El Hafidi M, Pérez-Torres I, Guarner V. (2009). Insulin resistance and the metabolic syndrome. Yao EB. Nova Biomedical Books. USA.

Banel DK, Hu FB. (2009). Effects of walnut consumption on blood lipids and other cardiovascular risk factors: a meta-analysis and systematic review. Am J Clin Nutr. 90:56–63.

Basma Damiri, Mohammed S. Abualsoud, Amjad M. Samara and Sakhaa K. Salameh. (2018). Metabolic syndrome among overweight and obese adults in Palestinian refugee camps. Diabetol Metab Syndr. 10:34

Bassuk, S. S. & Manson, J. E. (2010). Physical activity and cardiovascular disease prevention in women: a review of the epidemiologic evidence. Nutr Metab Cardiovasc Dis 20, 467–473.

Bassuk, S., Manson, J. (2005). Epidemiological evidence for the role of physical activity in reducing risk of type 2 diabetes and cardiovascular disease. Journal of Applied Physiology. 99: 1193-1204. Doi:10.1152/japplphysiol.00160.2005. 8750 -7587/05.

Baudrand R, Campino C, Carvaja, C A, Olivieri O, Guidi G, Faccini G & Fardella, C. E. (2014). High sodium intake is associated with increased glucocorticoid production, insulin resistance and metabolic syndrome. Clinical Endocrinology. 80(5),677-684

Bazzano LA, Thompson AM, Tees MT, et al. (2011). Non-soy legume consumption lowers cholesterol levels: a meta-analysis of randomized controlled trials. Nutr Metab Cardiovasc Dis. 21:94–103.

Bergman HE, Reeve BB, Moser RP, Scholl S, Klein WM. (2011). Development of a comprehensive heart disease knowledge questionnaire. Am J Health Educ.2011;42(2):74–87

Bigazzi R, & Bianchi S. (2007). Insulin resistance, metabolic syndrome and endothelial dysfunction. Journal of Nephrology. 20(1),10-14.

Blackford K, Jancey J, Lee AH, James AP, Waddell T, Howat P. (2016). Home-based lifestyle intervention for rural adults improves metabolic syndrome parameters and cardiovascular risk factors: A randomized controlled trial. The Authors. 89:15–22.

Blanco Mejia S, Kendall CW, Viguiliouk E, et al. (2014). Effect of tree nuts on metabolic syndrome criteria: a systematic review and meta-analysis of randomized controlled trials. BMJ Open. 2014; 4: e004660.

Boateng D, Wekesah F, Browne JL, Agyemang C, Agyei-Baffour P et al. (2017) Knowledge and awareness of and perception towards cardiovascular disease risk in sub-Saharan Africa: A systematic review. PLoS ONE 12(12): e0189264. https://doi.org/10.1371/ journal. pone.0189264.

Bouchenak, M., & Lamri-Senhadji, M. (2013). Nutritional quality of legumes, and their role in cardio-metabolic risk prevention: A review. Journal of Medicinal Food. 16(3), 185–198.

Bowles. D.K and Laughlin. M. H. (2011). Mechanism of beneficial effects of physical activity on atherosclerosis and coronary heart disease. J Appl Physiol (1985). 111(1): 308–310

Bray GA, Popkin BM. (2014). Dietary sugar and body weight: have we reached a crisis in the epidemic of obesity and diabetes? health be damned! Pour on the sugar. Diabetes Care. 37(4):950–6. [PubMed: 24652725].

Briasoulis, A., Agarwal, V., & Messerli, F. H. (2012). Alcohol consumption and the risk of hypertension in men and women: a systematic review and meta-analysis. Journal of Clinical Hypertension (Greenwich), 14(11), 792-798.

Brini.O. El, Akhouayri.O, Gamal.A, Mesfioui.A, and Benaz-zouz.B. (2014). "Prevalence of metabolic syndrome and its components based on a harmonious definition among adults in Morocco, Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy. 7; 341–346.

Brites RMR, Abreu AMM, Portela LF. (2019). Reduction of the alcohol consumption among workers using a brief intervention. Texto Contexto Enferm. 28: e20180135. Available from: http://dx.doi.org/10.1590/1980-265X-TCE-2018-0135

Burnet DL, Plaut AJ, Wolf SA, Huo D, Solomon MC, et al. (2011). Reach-out: a family-based diabetes prevention program for African American youth. J Natl Med Assoc 103: 269-277.

Danaei G, Lu Y, Singh GM, Carnahan E, Stevens GA, Cowan MJ, et al. (2014). Cardiovascular disease, chronic kidney disease, and diabetes mortality burden of cardio metabolic risk factors from 1980 to 2010: a comparative risk assessment. Lancet Diabetes Endocrinol. 2(8):634–47.

Desgroppes A, Taupin S. (2011). Kibera: the biggest slum in Africa? Les Cahoers de l'Afriquede l' Est. 44: 23-34 (halshs-00751833).

Cantiello, F. et al. (2015). Association between metabolic syndrome, obesity, diabetes mellitus and oncological outcomes of bladder cancer: a systematic review. Int J Urol 22, 22–32.

Cappuccio FP, Kerry SM, Micah FB, Plange-RhuleJ, Eastwood JB.A.(2006). Community programme to reduce salt intake and blood pressure in Ghana.BMCPublicHealth.6: 13.https://doi.org/10.1186/1471-2458-6-13PMID:16433927

Carlos Vasconcelos, António Almeida, Maria Cabral, Elisabete Ramos and Romeu Mendes. (2019). The Impact of a Community-Based Food Education Program on Nutrition-Related Knowledge in Middle-Aged and Older Patients with Type 2 Diabetes: Results of a Pilot Randomized Controlled Trial. Int. J. Environ. Res. Public Health. 16, 2403; doi:10.3390/ijerph16132403

Carter P, Gray LJ, Troughton J, et al. (2010). Fruit and vegetable intake and incidence of type 2 diabetes mellitus: systematic review and meta-analysis. BMJ. 341:c4229.

Casagrande, J T, Pike M C. (1978). An improved approximate formula for calculating sample sizes for comparing two binomial distributions. Biometrics. 34(3):483-6.

Catherine Nduku Kiama, Joyce Njeri Wamicwe, Elvis Omondi Oyugi, Mark Odhiambo Obonyo et al. (2018). Prevalence and factors associated with metabolic syndrome in an urban population of adults living with HIV in Nairobi, Kenya. The Pan African Medical Journal. 29:90. doi:10.11604/pamj.2018.29.90.13328

Chen.C.C, Lin.W.Y, C.-I. Li.C.I et al. (2012). "The association of alcohol consumption with metabolic syndrome and its individual components: the Taichung community health study," Nutrition Research, vol. 32, no. 1, pp. 24–29.

Chia-Huei Lin, Shang-Lin Chiang, Wen-Chii Tzeng, Li-Chi Chiang. (2014). Systematic Review of Impact of Lifestyle-Modification Programs on Metabolic Risks and Patient-Reported Outcomes in Adults with Metabolic Syndrome. Worldviews on Evidence-Based Nursing 11(6):361-8

Chiolero A, Jacot-Sadowski I, Faeh D, Paccaud F, & Cornuz J. (2007). Association of cigarettes smoked daily with obesity in a general adult population. Obesity. 15(5),1311-1318.

Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. (2003). The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA. 289:2560-72.

Choi J-H, Woo HD, Lee J-H, Kim J. (2015). Dietary patterns and risk for metabolic syndrome in Korean women: a cross-sectional study. Medicine. 94(34): e1424.

Clara Kelliny, Julita William, Walter Riesen, Fred Paccaud and Pascal Bovet. (2008). Metabolic syndrome according to different definitions in a rapidly developing country of the African region. Cardiovascular Diabetology. 7:27 doi: 10.1186/1475-2840-7-2

Cooper AJ, Forouhi NG, Ye Z, et al. (2012). Fruit and vegetable intake and type 2 diabetes: EPIC-InterAct prospective study and meta-analysis. Eur J Clin Nutr. 66:1082–1092.

Cornier MA, Dabelea D, Hernandez TL, Lindstrom RC, Steig AJ, Stob NR, et al. (2008). The Metabolic Syndrome. Endocr Rev. 29(7):777–822.

Cossi M-JJ, Preux P-MM, Chabriat H, Gobron C, Houinato D. (2012). Knowledge of stroke among an urban population in Cotonou (Benin). Neuroepidemiology. 38(3):172-8. https://doi.org/10.1159/ 000336862 PMID: 22472485

Cox CL, Stanhope KL, Schwarz JM, Graham JL, Hatcher B, Griffen SC, et al. (2011). Circulating concentrations of monocyte chemo attractant protein-1, plasminogen activator inhibitor-1, and soluble leukocyte adhesion molecule-1 in overweight/obese men and women consuming fructose- or glucose-sweetened beverages for 10 weeks. J Clin Endocrinol Metab. 96(12): E2034–8. [PubMed: 21956423]

Cullmann.M, Hilding.A, and Ostenson.C.G.(2012). “Alcohol consumption and risk of pre-diabetes and type 2 diabetes development in a Swedish population,” Diabetic Medicine, vol. 29, no. 4, pp. 441–452.

Dabou S, Telefo PB, Leonard Sama F. (2018). Evaluation of Dietary Habits and Lifestyle on the Prevalence of Metabolic Syndrome and Obesity in Undergraduate University Students in Cameroon: A Cross Sectional Study. J Metabolic Synd 7: 236. doi: 10.4172/2167-0943.1000236

Danaei G et al. (2014). National, regional, and global trends in systolic blood pressure since 1980: systematic analysis of health examination surveys and epidemiological studies with 786 country-years and 54 million participants. The Lancet, 2011; 377(9765):568–577.

Dandji.M, Zambou.F, Dangang.D, Nana.F and Tchouanguep.F. (2018). "Prevalence of metabolic syndrome in adult menof the dschang health district in Western-Cameroon" World Journal of Nutrition and Health. 6 (1),1–10

Deedwania P. (2011). Hypertension, dyslipidemia, and insulin resistance in patients with diabetes mellitus or the cardiometabolic syndrome: benefits of vasodilation beta-blockers. J Clin Hypertens (Greenwich).13:52–9.

Denova-Gutierrez E, Talavera JO, Huitron-Bravo G, Mendez-Hernandez P, Salmeron J. (2010). Sweetened beverage consumption and increased risk of metabolic syndrome in Mexican adults. Public Health Nutr. 13(6):835–42. [PubMed] [Google Scholar].

DiNicolantonioa JJ, Varshil Mehtab, Neema Onkaramurthyc, James H. O'Keefes. (2017). Fructose-induced inflammation and increased cortisol: A new mechanism for how sugar induces visceral adiposity. Prog Cardiovasc. https://doi.org/10.1016/j.pcad.2017.12.001

DiNicolantonio, J.J.; O'Keefe, J.H.; Lucan, S.C. (2015). Added fructose: A principal driver of type 2 diabetes mellitus and its consequences. Mayo Clin. Proc. 90, 372–381. [CrossRef] [PubMed]

DiNicolantonio, J.J.; Lucan, S.C. (2014). The wrong white crystals: Not salt but sugar as aetiological in hypertension and cardio-metabolic disease. Open Heart. 2014; 1(1), e000167. [CrossRef] [PubMed].

Dori Steinberg. (2017). The DASH Diet, 20 Years Later. JAMA. 317(15): 1529-1530. doi:10.1001/jama.2017.1628

Doulougou B, et al. (2014). Differences in hypertension between informal and formal areas of Ouagadougou, a sub-Saharan African city. BMC Public Health. 14: 893. DOI: https://doi.org/10.1186/1471- 2458-14-893

Dunkley AJ, Bodicoat DH, Greaves CJ, Russell C, Yates T, Davies MJ, et al. (2014). Diabetes prevention in the real world: effectiveness of pragmatic lifestyle interventions for the prevention of type 2 diabetes and of the impact of adherence to guideline recommendations: a systematic review and meta-analysis. Diabetes Care. 37(4):922–33.

Edyta Suliga, Dorota Kozieł, El˙zbieta Cie´sla, Dorota R˛ebak and Stanisław Głuszek. (2017). Dietary Patterns in Relation to Metabolic Syndrome among Adults in Poland: A Cross-Sectional Study. Nutrients. 2017; 9: 1366; doi: 10.3390/nu9121366

Elder J, Ayala G, Harris S. (1999). Theories and intervention approaches to health-behavior change in primary care. AmJ Prev Med 1999; 17: 275–84.

Elisa Anleu, Marcela Reyes, Marcela Araya B, Marcela Flores et al. (2019). Effectiveness of an Intervention of Dietary Counseling for Overweight and Obese Pregnant Women in the Consumption of Sugars and Energy. Nutrients.2019; 11, 385; doi:10.3390/nu11020385

Erdman JW, Balentine D, Arab L, et al. (2007). Flavonoids and heart health: proceedings of the ILSI North America Flavonoids Workshop, May 31-June 1, 2005, Washington, DC. J Nutr. 137:718S–37.

Ettner SL, Xu H, Duru OK, Ang A, Tseng C-H, Tallen L, Barnes A et al. (2014). The effect of an educational intervention onalcohol consumption, at-risk drinking, and health care utilization in olderadults: the project SHARE study. J Stud Alcohol Drugs. 75(3):447–57.

Eyup Avci, Ahmet Dolapoglu and Didar Elif Akgun. (2018). Role of Cholesterol as a Risk Factor in Cardiovascular Diseases, Cholesterol - Good, Bad and the Heart, Madan L. Nagpal, Intech Open, 2018; DOI: 10.5772/intechopen.76357.

Fathi AH. Maklady, Hanan Mkamal, Azza ZEl-Eraky and Omar H.Hassouna. (2014). Prevalence of metabolic syndrome among adults in Suez Canal area. The Egyptian Heart Journal. 66 (1), Page 15

Fereidoun Azizi, Parvin Mirmiran, Amir Abbas Momenan, Farzad Hadaegh et al. (2013). The Effect of Community-Based Education for Lifestyle Intervention on the Prevalence of Metabolic Syndrome and Its Components: Tehran Lipid and Glucose Study. International Journal of Endocrinology and Metabolism. 11(3): 145-53.

Ferris H. A, & Kahn C. R. (2016). Unraveling the paradox of selective insulin resistance in the liver: the brain–liver connection. Diabetes. 65(6),1481-1483.

Finley CE, Barlow CE, Halton TL, Haskell WL. (2010). Glycemic index, glycemic load, and prevalence of the metabolic syndrome in the cooper center longitudinal study. J Am Diet Assoc. 110: 1820–1829.

Firouzeh Sajjadi, Mojgan Gharipour, Noushin Mohammadifard, Fatemeh Nouri, Maryam Maghroun, Hasan Alikhasi. (2014). Relationship between legumes consumption and metabolic syndrome: Findings of the Isfahan Healthy Heart Program. ARYA Atheroscler. 10 (1)

Fonseca LG, Bertolin MNT, Gubert MB, daSilva EF. (2019). Effects of a nutritional intervention using pictorial representations for promoting knowledge and practices of healthy eating among Brazilian adolescents. PLoSONE 14(3):e0213277.

Forouzanfar MH, et al. (2016). Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2015: a systematic analysis for the global burden of disease study 2015. Lancet. 388:1659–724

Fottrell E, Ahmed N, Morrison J, et al. (2019). Community groups or mobile phone messaging to prevent and control type 2 diabetes and intermediate hyperglycaemia in Bangladesh (DMagic): a cluster-randomised controlled trial. Lancet Diabetes Endocrinol. 7:200–12.

Frederick M. Wekesah, Kerstin Klipstein-Grobusch, Diederick E et al. (2020). Determinants of Mortality from Cardiovascular Disease in the Slums of Nairobi, Kenya. Global Heart. 15(1): 33.

Fox, C. and Kilvert, A. (2003). Intensive education for lifestyle change in diabetes. BMJ; 327: 1120-1121.

Ganasegeran.K, Al-Dubai.S.A, Qureshi.A.M, Al-abed.A.A, Rizal.A, and Aljunid.S.M. (2012). Social and psychological factors affecting eating habits among university students in aMalaysian medical school: a cross-sectional study. Nutrition Journal, vol. 11, no. 1, article 48.

Garcia G, Sunil TS, Hinojosa P. (2012). The fast food and obesity link: consumption patterns and severity of obesity.Obes Surg. 22: 810–818.24

Gennuso KP, Gangnon RE, Thraen-Borowski KM, Colbert LH. (2014). Dose response relationships between sedentary behaviour and the metabolic syndrome and its components. Diabetologia. doi:10.1007/ s00125-014-3453-z

Geoffrey Omuse, Daniel Maina, Mariza Hoffman, Jane Mwangi, Caroline Wambua, Elizabeth Kagotho, Angela Amayo, Peter Ojwang et al. (2017). Metabolic syndrome and its predictors in an urban population in Kenya: A cross sectional study. BMC Endocrine Disorders (2017) 17:37 DOI 10.1186/s12902-017-0188-0.

Gerstel E, Pataky Z, Busnel C, Rutschmann O, Guessous I, Zumwald C, et al. (2013). Impact of lifestyle intervention on body weight and the metabolic syndrome in home-care providers. Diabetes Metab. Elsevier Masson SAS; 39:78–84.

Giovanna Ricci, Isabel Pirillo, Daniele Tomassoni, Ascanio Sirignano & Iolanda Grappasonni. (2017). Metabolic syndrome, hypertension, and nervous system injury: Epidemiological correlates, Clinical and Experimental Hypertension. 2017; DOI: 10.1080/10641963.2016.1210629

Giralt D, Domingues-Montanari S, Mendioroz M, Ortega L, Maisterra O, et al. (2012). The gender gap in stroke: a meta-analysis. Acta Neurol Scand. 125:83–90. doi: 10.1111/j.1600-0404.2011.01514.

Gladys Mugure, Mohamed Karama, Catherine Kyobutungi, Simon Karanja. (2014). Correlates for cardiovascular diseases among diabetic/hypertensive patients attending outreach clinics in two Nairobi slums, Kenya. Pan African Medical Journal (ISSN: 1937-8688).

Glanz, K., Rimer, BK., & Viswanath, K. (2008). Health Behavior and Health Education: Theory, Research, and Practice, (4th edition), Jossey-Bass, San Francisco.

Gomez-Olive FX, Ali SA, Made F, Kyobutungi C, Nonterah E, Micklesfield L, et al. (2017). Stark Regional and Sex Differences in the Prevalence and Awareness of Hypertension: An H3Africa AWI-Gen Study Across 6 Sites in Sub-Saharan Africa. Global Heart. 2017;12(2):81-90.

Grundy S. M, Cleeman J. I, Daniels S. R, Donato K. A, Eckel R. H, Franklin B. A, ... & Spertus J. A. (2005). Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. Circulation. 112(17), 2735-2752.

Grundy, S.M. (2008). Metabolic syndrome pandemic. Arterioscler. Thromb. Vasc. Biol. 28 (4), 629–636.

Guarner-Lans V, Rubio-Ruiz ME, Perez-Torres I, Baños de McCarthy G.(2011). Relation of aging and sex hormones to metabolic syndrome and cardiovascular disease. Exp Gerontol. 46: 517-523.

Gunness, P., & Gidley, M. J. (2010). Mechanisms underlying the cholesterol-lowering properties of soluble dietary fibre polysaccharides. Food & Function. 1(2),149–155.

Guzman.M, Zbella.E, Shah Alvarez.S, Nguyen.J.L, Imperial.E, et al.(2019). Effect of an intensive lifestyle intervention on the prevalence of metabolic syndrome and its components among overweight and obese adults. Journal of Public Health. 2019, Vol. 42, No. 4, pp. 828–838 | doi:10.1093/pubmed/fdz170

Gyakobo.M, Amoah, A.G.B, Martey-Marbell.A, and Snow. R.C. (2012). "Prevalence of the metabolic syndrome in a rural population in Ghana,"BMC Endocrine Disorders. vol.12, article no. 25.

Hahn V, Halle M, Schmidt-Trucksass A, Rathmann W, Meisinger C, & Mielck A.(2009). Physical activity and the metabolic syndrome in elderly German men and women: results from the population-based KORA survey. Diabetes Care.32(3),511-513.

Hall. J.E, da Silva.A.A, do Carmo.J.M et al. (2010). "Obesity-induced hypertension: role of sympathetic nervous system, leptin, and melanocortins," The Journal of Biological Chemistry, vol. 285, no. 23, pp. 17271–17276.

Hamid S, Groot W, Pavlova M. (2019). Trends in cardiovascular diseases and associated risks in sub-Saharan Africa: a review of the evidence for Ghana, Nigeria, South Africa, Sudan and Tanzania. Aging Male. 22(3):169–76.

Hang Li, Xia Li, Sheng Yuan, Yalei Jin and Jinping Lu. (2018). Nut consumption and risk of metabolic syndrome and overweight/obesity: ameta-analysis of prospective cohort studies and randomized trials. Nutrition & Metabolism. 15:46

Haregu TN, et al. (2015). Co-occurrence of behavioral risk factors of common non-communicable diseases among urban slum dwellers in Nairobi, Kenya. Global Health Action. 8: 28697. DOI: https://doi. org/10.3402/gha.v8.28697

Hendriks ME, Wit FWNM, Roos MTL et al. (2012). Hypertension in Sub-Saharan Africa: Cross-Sectional Surveys in Four Rural and Urban Communities. PLoS ONE 2012; 7(3): e32638.

Hill J, Peer N, Oldenburg B, Kengne AP. (2017). Roles, responsibilities and characteristics of lay community health workers involved in diabetes prevention programmes: a systematic review. PLoS One. 12(12): e0189069.

Hofmann T, Elbelt U, Stengel A. (2014). Irisin as a muscle-derived hormone stimulates thermogenesis-a critical update. Peptide. 54: 89–100.

Holmen, H., Wahl, A., Torbjørnsen, A, Jenum, A.K, Småstuen, M.C., & Ribu, L. (2016). Stages of change for physical activity and dietary habits in persons with type 2 diabetes included in a mobile health intervention: the Norwegian study in renewing health. BMJ Open Diabetes Res Care. 4(1): 1-1. e000193. doi: 10.1136/bmjdrc-2016-000193.

Hooper L, Kroon PA, Rimm EB, et al. (2008). Flavonoids, flavonoid-rich foods, and cardiovascular risk: a meta-analysis of randomized controlled trials. Am J Clin Nutr.88:38–50.

Hosseinpour-Niazi S, Mirmiran P, Mirzaei S, et al. (2015). Cereal, fruit and vegetable fibre intake and the risk of the metabolic syndrome: a prospective study in the Tehran Lipid and Glucose Study. J Hum Nutr Diet. 28:236–245.

Hotamisligil GS. (2006). Inflammation and metabolic disorders. Nature. 444:860-867.

Howlett. S.E. (2010). Effects of aging on the cardiovascular system. In: Fillit HM, Rockwood K, Woodhouse K, editors. Brocklehurst's Textbook of Geriatric Medicine and Gerontology, 7th Edition. New York: WB Saunders. 168-70.

Huang Y, et al. (2016). Association between pre-diabetes and risk of cardiovascular disease and all-cause mortality: systematic review and meta-analysis. bmj. 355:i5953.

Huang Y, Liu X. (2014). Leisure-time physical activity and the risk of metabolic syndrome: meta-analysis. Eur J Med Res 19:22. doi:10.1186/2047-783X-19-22.

Huang, C.M., Wu, H.L., Huang, S.H., Chien, L.Y., & Guo, J.L. (2013). Trans-theoretical model based passive smoking prevention programme among pregnant women and mothers of young children. Eur J Public Health. 23(5):777-782.

Hulzebosch Annelieke, Steven van de Vijver, Samuel O. Oti, Thaddaeus Egondi and Catherine Kyobutung. (2015). Profile of people with hypertension in Nairobi's slums: a descriptive study. Globalization and Health. 11:26 DOI 10.1186/s12992-015-0112-1.

Hutchins, A. M., Winham, D. M., & Thompson, S. V. (2012). Phaseolus beans: Impacton glycaemic response and chronic disease risk in human subjects. British Journal of Nutrition. 108 (Suppl 1), S52–S65.

Hwang GY, Cho YJ, Chung RH, Kim SH. (2014). The relationship between smoking level and metabolic syndrome in male health check-up examinees over 40 years of age. Korean J Fam Med. 35:219–226.

Hyunjung Lim, Tuan Nguyen, Ryowon Choue, Youfa Wang. (2012). Socio-demographic Disparities in the Composition of Metabolic Syndrome Components Among Adults in South Korea. Diabetes Care. 35:2028–2035.

International Diabetic Federation (IDF). (2015). Diabetes ATLAS, Seventh Edition.

International Diabetes Federation. (2013). Sixth edition

International Diabetes Federation. (2017). IDF Diabetes Atlas; IDF: Brussels, Belgium.

Inthawong, R. , Khatab, K. , Whitfield, M. , Collins, K. , Raheem, M. and Ismail, M. (2019). Health Care and Hospitalization Costs of Cardiovascular Disease (CVD) in Thailand. *Open Access Library Journal*, **6**, 1-5. doi: 10.4236/oalib.1105320.

Iqbal SP, Ramadas A, Fatt QK, Shin HL, Onn WY, Kadir KA. (2020). Relationship of sociodemographic and lifestyle factors and diet habits with metabolic syndrome (MetS) among three ethnic groups of the Malaysian population. PLoS One. 15(3): e0224054. doi: 10.1371/journal.pone.0224054. eCollection 2020.

James J. DiNicolantonio, Sean C. Lucan and James H. O'Keefe. (2016). The Evidence for Saturated Fat and for Sugar Related to Coronary Heart Disease. Prog Cardiovasc Dis. 58(5): 464–472. doi: 10.1016/j.pcad.2015.11.006

Jana V van Vliet-Ostaptchouk, Marja-Liisa Nuotio, Sandra N Slagter, Dany Doiron, Krista Fischer, Luisa Foco, Amadou Gaye et al. (2014). The prevalence of metabolic syndrome andmetabolically healthy obesity in Europe: a collaborative analysis of ten large cohort studies. BMC Endocrine Disorders. 14:9

Jaspinder Kaur. (2014). Assessment and Screening of the Risk Factors in Metabolic Syndrome. Med. Sci. 2, 140-152; doi:10.3390/medsci2030140

Jayalath VH, de Souza RJ, Sievenpiper JL, et al. (2014). Effect of dietary pulses on blood pressure: a systematic review and meta-analysis of controlled feeding trials. Am J Hypertens. 27:56–64

Jeffrey J. VanWormera, Jackie L. Boucherb, Abbey C. Sidebottomc, Arthur Sillahc, Thomas Knickelbined. (2017). Lifestyle changes and prevention of metabolic syndrome in the Heart of New Ulm Project. Preventive Medicine Reports 6. 242–245.

Jenkins DJA, Kendall CW, Augustin LS, et al. (2012). Effect of legumes as part of a low glycemic index diet on glycemic control and cardiovascular risk factors in type 2 diabetes mellitus. Arch Intern Med. 172:1653–1660

Jennifer L. Kuk, and Chris I. (2010). Age and Sex Differences in the Clustering of Metabolic Syndrome Factors. Diabetes Care. 33(11): 2457–2461.

Jo-Ana D Chase. (2015). Interventions to Increase Physical Activity Among Older Adults: A Meta-Analysis. Gerontologist. 55(4):706-18. doi: 10.1093/geront/gnu090.

Junga Lee, Yoonmyung Kim and Justin Y. Jeon. (2016). Association between physical activity and the prevalence of metabolic syndrome: from the Korean National Health and Nutrition Examination Survey, 1999–2012. SpringerPlus. 5:1870 DOI 10.1186/s40064-016-3514-5.

Jui-Hua Huang, Ren-Hau Li, Shu-Ling Huang, Hon-Ke Sia et al. (2017). Relationships between different types of physical activity and metabolic syndrome among Taiwanese workers. Scientific Reports | 7: 13735 | DOI:10.1038/s41598-017-13872-5

Justin B Echouffo-Tcheugui, Mary Mayige, Anthonia Okeoghene Ogbera, Eugene Sobngwi, Andre P Kengne. (2012). Screening for hyperglycaemia in the developing world: rationale, challenges and opportunities. Diabetes Res Clin Pract. 98(2):199-208. doi: 10.1016/j.diabres.2012.08.003

Kaduka, L. U., Kombe, Y., Kenya, E., Kuria, E., Bore, J. K., Bukania, Z. K., & Mwangi, M. (2012). Prevalence of Metabolic Syndrome among an Urban Population in Kenya. Diabetes Care, 35, 887-893. doi:10.2337/dc11-0537.

Kang SJ, Kim SC, Kim YH. (2012). Korean Older Adult's Physical Activity and Its Related PsychosocialVarieables. Journal of Sports Science and Health. 13 (3): 89-99.

Khetan AK, Purushothaman R, Chami T, Hejjaji V, Madan Mohan SK, Josephson RA, Webel AR.(2017). The Effectiveness of Community Health Workers for CVD Prevention in LMIC. Global Heart. 12(3):233–43 e236.

Kastorini CM, Milionis HJ, Esposito K, Giugliano D, Goudevenos JA et al. (2011). The effect of Mediterranean diet on metabolic syndrome and its components: a meta-analysis of 50 studies and 534,906 individuals. J Am Coll Cardiol. 57(11):1299-313. doi: 10.1016/j.jacc.2010.09.073.

Kaur, J. (2014). A comprehensive review on metabolic syndrome. Cardiol. Res. Pract. 943162.

Keith C. Ferdinand, Kellee P. Patterson, Chery Taylor, Icilma V. Fergus et al. (2012). Community-Based Approaches to Prevention and Management of Hypertension and Cardiovascular Disease. The Journal of Clinical Hypertension Vol 14 | No 5. DOI: 10.1111/j.1751-7176. 00622.x

Keller U. (2011). Dietary proteins in obesity and in diabetes. International Journal for Vitamin and Nutrition Research. 81(2),125.

Kennedy A, Martinez K, Chuang CC, LaPoint K, McIntosh M. (2009). Saturated fatty acid-mediated inflammation and insulin resistance in adipose tissue: mechanisms of action and implications. J Nutr. 139:1-4.

Kenya National Diabetes Strategy. (2010). Republic of Kenya. Ministry of public health and sanitation. First Edition. http://diabetes-communication.org/wordpress/wpcontent/uploads/2012/09/Kenya-National-Diabetes-Strategy-2010-2015.

Khetan AK, Purushothaman R, Chami T, Hejjaji V, Madan Mohan SK et al. (2017). The Effectiveness of Community Health Workers for CVD Prevention in LMIC. Global Heart. 2017;12(3):233–43 e236.

Kimani Samuel, Waithira Mirie, Margaret Chege, Okubatsion Tekeste Okube, Samuel Muniu. (2019). Association of lifestyle modification and pharmacological adherence on blood pressure control among patients with hypertension at Kenyatta National Hospital, Kenya: a cross-sectional study. BMJ Open. 9: e023995. doi:10.1136/bmjopen-2018-023995

Kimber L. Stanhope. (2016). Sugar consumption, metabolic disease and obesity: The state of the controversy. Clin Lab Sci. 53(1): 52–67. doi:10.3109/10408363.2015.1084990.

Kim J, Tanabe K, Yokoyama N, Zempo H, Kuno S. (2011). Association between physical activity and metabolic syndrome in middle-aged Japanese: a cross-sectional study. BMC Public Health. 11:624. doi: 10.1186/1471-2458-11-624.

Kolli Srinath and Dorairaj Prabhakaran. (2007). Treatment Guidelines: The Developing World. in Comprehensive Hypertension.

Komolafe MA, Obembe AO, Olaogun MO, Adebiyi AM et al. (2015). Awareness of stroke risk factors and warning signs in Nigerian adolescents compared with adults. J Stroke Cerebrovasc Dis 24(3):687–93.

Koyun, A. and Eroglu, b.K. (2014). The trans-theoretical model use for smoking cessation, European Journal of Research on Education, Special Issue: Contemporary Studies in Social Science. 130-134. Available Online at http://iassr.org/journal

Koyun, A., & Eroglu, K. (2016). The effect of trans-theoretical model-based individual counseling, training, and a 6-month follow-up on smoking cessation in adult women: a randomized controlled trial. Turk J Med Sci. 46(1):105-11. doi: 10.3906/sag-1407-100.

Krishnan S, Coogan PF, Boggs DA, Rosenberg L, Palmer JR. (2010). Consumption of restaurant foods and incidence of type 2 diabetes in African American women. Am J Clin Nutr. 91: 465–471.

Lee K. (2012). Gender-specific relationships between alcohol drinking patterns and metabolic syndrome: the Korea National Health and Nutrition Examination Survey 2008. Public Health Nutr. 15:1917–24.

Leidy H. J, Clifton P. M, Astrup A, Wycherley T. P, Westerterp-Plantenga M. S, Luscombe- Marsh ND, ... & Mattes RD. (2015). The role of protein in weight loss and maintenance–. The American Journal of Clinical Nutrition. 101(6),1320S-1329S.

Leila Jahangiry, Leila Khosravi-far, Parvin Sarbakhsh et al. (2019). Prevalence of metabolic syndrome and its determinants among Iranian adults: evidence of IraPE N survey on a bi-ethnic population. Scientific Reports. 9:7937 | https://doi.org/10.1038/s41598-019-44486-8

Levitan EB, Lewis CE, Tinker LF, Eaton CB, Ahmed A, Manson JE, et al. (2013). Mediterranean and DASH diet scores and mortality in women with heart failure: The Women's Health Initiative. Circ Heart Fail. 6:1116-23.

Ley SH, Hamdy O, Mohan V, Hu FB. (2014). Prevention and management of type 2 diabetes: dietary components and nutritional strategies. Lancet. 383:1999–2007

Li J and Siegrist J. (2012). Physical Activity and Risk of Cardiovascular Disease—A Meta-Analysis of Prospective Cohort Studies. International Journal of Environmental Research and Public Health. 9(2): 391.DOI: https://doi.org/10.3390/ijerph9020391

Li X-t, Liao W, Yu H-j, Liu M-w, Yuan S, Tang B-w, et al. (2017). Combined effects of fruit and vegetables intake and physical activity on the risk of metabolic syndrome among Chinese adults. PLoS ONE 12(11): e0188533. https://doi.org/10.1371/journal.pone.0188533

Liu Y, Han Y, Shi J, Li R, Li S, Jin N, et al. (2015). Effect of peer education on self-management and psychological status in type-2 diabetes patients with emotional disorders. 2015. vol. 6: p. 479–86.

Luisa Maria Roberta Tedesco, Gabriella Di Giuseppe, Francesco Napolitano, et al. (2015). Cardiovascular Diseases and Women: Knowledge, Attitudes, and Behavior in the General Population in Italy. BioMed Research International Volume Article ID 324692, 7 pages. http://dx.doi.org/10.1155/2015/324692

Lydia U. Kaduka, Yeri Kombe, Eucharia KENYA, Elizabeth Kuria, John K et al. (2012). Prevalence of Metabolic Syndrome Among an Urban Population in Kenya. care. Diabetes journals.org. Diabetes Care 35:887–893, 2012.

Ma, J., Betts, N. M., Horacek, T., Georgiou, C., White, A., & Nitzke, S. (2002). The importance of decisional balance and self-efficacy in relation to stages of change for fruit and vegetable intakes by young adults. Am J Health Promot, 16(3), 157- 166.

Mabry RM, Reeves MM, Eakin EG, Owen N. (2010). Gender differences in prevalence of the metabolic syndrome in Gulf Cooperation Council Countries: a systematic review. Diabet Med. 27: 593-597.

Mackenbach JP, Stirbu I, Roskam AJ, Schaap MM, Menvielle G, Leinsalu M, et al. (2008) Socioeconomic inequalities in health in 22 European countries. N Engl J Med. 358:2468–2481. [PubMed] [Google Scholar].

Makrilakis K, Grammatikou S, Liatis S, Kontogianni M, Perrea D, Dimosthenopoulos C, et al. (2012). The effect of a non-intensive community- based lifestyle intervention on the prevalence of Metabolic Syndrome. The DEPLAN study in Greece. Hormones (Athens). 11:316–24.

Mahan.L, Foster.L., and Dahl.J.W.(2016). Beans, Peas, and Lentils: Health Benefits, Food Science & Human Nutrition.

Maina, W., Ndegwa, A., Njenja, E., Muchemi, E. (2011). Knowledge, attitude, and practices related to diabetes among community members in four provinces in Kenya: a cross sectional study. African journal of diabetes medicine; 19(1):15-18.

Makrilakis K, Grammatikou S, Liatis S, Kontogianni M, Perrea D, Dimosthenopoulos C, et al. (2012). The effect of a non-intensive community- based lifestyle intervention on the prevalence of Metabolic Syndrome. The DEPLAN study in Greece. Hormones (Athens). 11:316–24.

Maowei Cheng, Huijun Wang, Zhihong Wang, Wenwen Du et al. (2017). Relationship between dietary factors and the number of altered metabolic syndrome components in Chinese adults: a cross-sectional study using data from the China Health and Nutrition Survey. BMJ Open. 7(5): e014911.

Maphosa Y, Jideani VA. (2017). The role of legumes in human nutrition. Functional Food Maria Chavarri, Intech Open; https://doi.org/10.5772/ intechopen69127. Available from: https://www.intechopen.com/books/functional-food-improve-health-through-adequate-food/the-role-oflegumes-in-human-nutrition.

Marcus, B. H., Rossi, J. S., Selby, V. C., Niaura, R. S., & Abrams, D. B. (1992). The stages and processes of exercise adoption and maintenance in a worksite sample. Health Psychol, 11(6), 386-395.

Mark Conner and Paul Norman. (2021). Reference Module in Neuroscience and Biobehavioral Psychology.

Maria van Namen, Luke Prendergast, Casey Peiris. (2019). Supervised Lifestyle Intervention for People with Metabolic Syndrome Improves Outcomes and Reduces Individual Risk Factors of Metabolic Syndrome: A Systematic Review and Meta-Analysis. Metabolism. Dec; 101:153988. doi: 10.1016/j.metabol.2019.153988.

Marie Doualla-Bija, Yonathan Lobe Batchama , Jude Moutchia-Suh , Vicky Joceline Ama Moor et al. (2018). Prevalence and characteristics of metabolic syndrome in gout patients in a hospital setting in sub-Saharan Africa. Diabetes Metab Syndr.12(6):1007-1011. doi: 10.1016/j.dsx.2018.06.015.

Maritza J. Kruger and Theo A. Nell. (2017). The prevalence of the metabolic syndrome in a farm worker community in the Bolanddistrict, South Africa. MC Public Health. 17:61

Maryam Shirinzadeh, Babak Afshin-Pour, Ricardo Angeles et al. (2019). The effect of community-based programs on diabetes prevention in low- and middle-income countries: a systematic review and meta-analysis. Globalization and Health. 15:10. https://doi.org/10.1186/s12992-019-0451-4

Maryam Mohammadi. (2013). Application of Transtheoretical Model to Exercise in Office Staff. Electronic physician. 5 (1). http://www.ephysician.ir

Mathenge, W., Foster, A., & Kuper, H. (2010). Urbanization, ethnicity and cardiovascular risk in a population in transition in Nakuru, Kenya: A population-based survey. BMC Public Health. 10, 569. doi:10.1186/1471-2458-10-569

Mbanya, NJC., Ayesha, AM., Sobngwi, E., Assah, FK., Enoru, S. (2010). Diabetes in sub-Saharan Africa. Lancet: 375:2254–2266.

McCracken E, Monaghan M, & Sreenivasan S. (2018). Pathophysiology of the metabolic syndrome. Clinics in Dermatology. 36(1),14-20.

McLaren L., Ghali L. M., Lorenzetti D., Rock M. (2007). Out of context? Translating evidence from the North Karelia Project over place and time. Health Education Research. 22(3):414–424. [PubMed]

Mecca MS, Moreto F, Burini FHP, et al. (2012). Ten-week lifestyle changing program reduces several indicators for metabolic syndrome in overweight adults. Diabetol Metab Syndr . 4: 1-7; http://dx.doi.org/10.1186/1758-5996-4-1

Megan McStea, Kevin McGeechan, Shahrul Bahyah Kamaruzzaman et al. (2016). Defining metabolic syndrome and factors associated with metabolic syndrome in a poly-pharmaceutical population. Journal Postgraduate Medicine.128 (8).

Mehrabian F, Farmanbar R, Mahdavi Roshan M, Omidi S, Aghebati R. (2018). The effect of nutrition education based on DASH diet on blood pressure and dietary adherence among patients with hypertension. Caspian J Health Res. 3(2):48-52. doi: 10.29252/cjhr.3.2.48.

Mendonça FM, de Sousa FR, Barbosa AL, Martins SC, Araújo RL, Soares R, et al.(2015). Metabolic syndrome and risk of cancer: which link? Metabolism; 64:182–9.

Mensah GA, et al. (2015). Mortality from cardiovascular diseases in sub-Saharan Africa, 1990–2013: a systematic analysis of data from the Global Burden of Disease Study 2013.

Cardiovascular Journal of Africa. 26(2 Suppl 1): S6–10. DOI: https://doi.org/10.5830/CVJA-2015-036

Meneton P, Kesse-Guyot E, Méjean C, Fezeu L, Galan P, Hercberg S, Ménard J.(2014). Unemployment is associated with high cardiovascular event rate and increased all-cause mortality in middle-aged socially privileged individuals. Int Arch Occup Environ Health. 2015 Aug; 88(6):707-16. Doi: 10.1007/s00420-014-0997-7. Epub. PMID: 25385250.

Messina M. (2016). Soy and health update: evaluation of the clinical and epidemiologic literature. Nutrients. 8:754

Micha R, Wallace SK, Mozaffarian D. (2010). Red and processed meat consumption and risk of incident coronary heart disease, stroke, and diabetes mellitus: a systematic review and meta-analysis. Circulation. 121: 2271–2283.

Michael Miller, Neil J Stone, Neil J Stone, Christie Ballantyne, Subramanian Pennathur. (2011). Triglycerides and Cardiovascular Disease. A Scientific Statement From the American Heart Association. Circulation 123(20):2292-333

Micklesfield LK, Lambert EV, Hume DJ, et al. (2013). Socio-cultural, environmental and behavioural determinants of obesity in black South African women. Cardiovasc JAfr. 24:369–375

Mikael Rabaeus, Patricia Salen and Michel de Lorgeril. (2013). Is it smoking or related lifestyle variables that increase metabolic syndrome risk? BMC Medicine. 11:196 http://www.biomedcentral.com/1741-7015/11/196

Millen BE, Pencina MJ, Kimokoti RW, Zhu L, Meigs JB et al. (2006). Nutritional risk and the metabolic syndrome in women: opportunities for preventive intervention from the Framingham Nutrition Study.Am J Clin Nutr. 84:434–441

Min You and Gavin E. Arteel. (2019). Effect of ethanol on lipid metabolism. J Hepatol. 70(2): 237–248. doi: 10.1016/j.jhep.2018.10.037

Misra A, Singhal N, Sivakumar B, et al. (2011). Nutrition transition in India: Secular trends in dietary intake and their relationship to diet-related non-communicable diseases. J Diabetes 3: 278-92; http://dx.doi.org/10.1111/j.1753-0407.2011.00139.x

Miyares MA, Davis KA. (2014). Community-Based Interventions to Improve Outcomes for Cardiovascular Disease Prev Chronic.11:140390. DOI: http://dx.doi.org/10.5888/pcd11.140390

Mohamed SA. (2014). Effect of lifestyle intervention on health behaviors, weight and blood glucose level among patients with diabetes mellitus. J. Nurs. Educ. Pract. 4.

Mohamed Hamad Al-Thani Al Anoud Mohammed Al-Thani, Sohaila Cheema et al. (2016). Prevalence and determinants of metabolic syndrome in Qatar: results from a National Health Survey. BMJ Open. 6: e009514. doi:10.1136.

Mohamed Ahmed Said, Mohamed Abdelmoneem, Mohamed Chaab Alibrahim et al. (2020). Effects of diet versus diet plus aerobic and resistance exercise on. metabolic syndrome in obese young men. Journal of Exercise Science & Fitness 18 (2020) 101e108

Mohd Azahar NMZ, Krishnapillai ADS, Zaini NH, Yusoff K. (2017). Risk perception of cardiovascular diseases among individuals with hypertension in rural Malaysia. Heart Asia. 9(2):e010864.

Mohd Razif Shahril,Wan Putri Elena Wan Dali and Pei Lin Lua. (2013). A 10-Week Multimodal Nutrition Education InterventionImproves Dietary Intake among University Students: ClusterRandomised Controlled Trial. Journal of Nutrition and Metabolism. Article ID 658642,11pageshttp://dx.doi.org/10.1155/2013/658642

Mohsen, M.M, Saafan, N.A., Attia, A., & El-Abassy, A. (2014). Lifestyle Behavior Modification of Mothers of Diabetic Children's through Application of Trans-theoretical Model of Change. J Nurs Care. 3(2): 1-10. doi:10.4172/2167-1168.1000153

Moreira GC, Cipullo JP, Ciorlia LAS, Cesarino CB, Vilela-Martin JF. (2014). Prevalence of Metabolic Syndrome: Association with Risk Factors and Cardiovascular Complications in an Urban Population. PLoS ONE 9(9): e105056. https://doi.org/10.1371/journal.pone.0105056

Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. (2011). Changes in diet and lifestyleand long-term weight gain in women and men. N Engl J Med. 364:2392–2404.

Muchiri JW, Gericke GJ, Rheeder P. (2016). Effect of nutrition education on diabetes knowledge and attitudes of adults with type 2 diabetes living in a resource limited setting in South Africa: a randomised controlled trial Impact of nutrition education on diabetes knowledge and attitudes of adul. J Endocrinol Metab Diabetes South Africa Taylor Francis. 21:26–34.

Muchiri JW, Gericke GJ, Rheeder P. (2015). Effect of a nutrition education programme on clinical status and dietary behaviours of adults with type 2 diabetes in a resource-limited setting in South Africa: a randomised controlled trial. Public Health Nutr. 19:1–14.

Muraki I, et al. (2013). Fruit consumption and risk of type 2 diabetes: results from three **prospective longitudinal cohort studies. BMJ. 347.**

Naimi AI, Paquet C, Gauvin L, Daniel M.(2009). Associations between area-level unemployment, body mass index, and risk factors for cardiovascular disease in an urban area. Int J Environ Res Public Health. 6:3082–3096. Google ScholarCrossrefPubMed

Nakibuuka J, Sajatovic M, Katabira E, Ddumba E, Byakika-Tusiime J, Furlan AJ. (2014). Knowledge and Perception of Stroke: A Population-Based Survey in Uganda. ISRN stroke. 2014:1-7.

National Cancer Institute. (2005). Theory at a glance guide for health promotion practice.

Narayan KMV, Chan J, Mohan V. (2011). Early identification of type 2 diabetes: policies should be aligned with health systems strengthening. Diabetes Care. 34(1):244–6.

Neter JE, Stam BE, Kok FJ, Grobbee DE, Gelseijnse JM. (2003). Influence of weight reduction on blood pressure: a meta-analysis of randomized controlled trials. Hypertension. 42:878–884. https://doi.org/10.1161/01. HYP.0000094221.86888.AE

Nguyen KH, Lee JH, Nyomba BL. (2012). Ethanol causes endoplasmic reticulum stress and impairment of insulin secretion in pancreatic beta-cells. Alcohol 46(1), 89–99.

Nigg CR, Rossi JS, Norman GJ, Benisovich SV. (1998). Structure of decisional balance for exercise adoption. Annals of Behavioral Medicine. 20: S211. 1998.

Nitzke, S., Kritsch, K., Boeckner, L., Greene, G., Hoerr, S., Horacek, T. et al. (2007). stage-tailored multi-modal intervention increases fruit and vegetable intakes of low-income young adults. Am J Health Promot. 22(1):6-14.

Nunez-Cordoba JM, Martinez-Gonzalez MA, Bes-Rastrollo M, et al. (2009). Alcohol Consumption and the Incidence of Hypertension in a Mediterranean Cohort: The SUN Study. Revista Española de Cardiología. 62:633–41.

Nyaruhucha CNM, Achen JH, Msuya JM, Shayo NB, Kulwa KBM. (2003). Prevalence and awareness of obesity among people of different age groups ineducational institutions in Morogoro Tanzania. East Afr Med J. 80:68–72.

Ofori-Asenso R, Agyeman AA, Laar A, Boateng D. (2016). Overweight and obesity epidemic in Ghana—a systematic review and meta-analysis. BMC Public Health [Internet]. 2016 Dec; 16(1):1239. Available from: http://bmcpublichealth.biomedcentral.com/articles/10.1186/s12889-016-3901-4 https://doi.org/10.1186/s12889-016-3901-4 PMID: 27938360

Ofori-Asenso R, Garcia D. (2016). Cardiovascular diseases in Ghana within the context of globalization. Cardiovasc Diagn Ther [Internet]. 2016; 6(1):67–77. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=4731582&tool=pmcentrez&rendertype=abstract https://doi.org/10.3978/j.issn.2223-3652.2015.09.02 PMID: 26885494

Okafor CI (2012). "The metabolic syndrome in Africa: Current trends". Indian J Endocrinol Metab. 16: 56–66. PMC 3263198 Freely accessible. PMID 22276253. doi:10.4103/2230-8210.91191.

Okube OT, Kimani S, Waithira M. (2020). Association of dietary patterns and practices on metabolic syndrome in adults with central obesity attending a mission hospital in Kenya: a cross-sectional study. BMJ Open. 10:e039131. doi:10.1136/ bmjopen-2020-039131

Okubatsion Tekeste, Samuel Kimani and Waithira Mirie. (2020). Gender Differences in the Pattern of Socio- Demographics Relevant to MetabolicSyndrome Among Kenyan Adults with Central Obesity at a Mission Hospital in Nairobi, Kenya. High Blood Pressure & Cardiovascular Prevention. https://doi.org/10.1007/s40292-020-00360-7

Oladapo O., Salako L, Sadiq L, Soyinka K, F A.O. (2013). Knowledge of Hypertension and other Risk Factors for Heart Disease among Yoruba Rural South-western Nigerian Population. Br J Med Med Res. 3(4):983-1003.

O'Neill S and O'Driscoll L. (2015). Metabolic syndrome: a closer look at the growing epidemic and its associated pathologies. Obes. Rev. 16(1):1–12. [PubMed] [Google Scholar]

Onvani Sh, Haghighatdoost F, Azadbakht L. (2015). Dietary approach to stop hypertension (DASH): diet components may be related to lower prevalence of different kinds of cancer: A review on the related documents. J Res Med Sci. 20:708-13

Orlich MJ, Fraser GE. (2014). Vegetarian diets in the Adventist Health Study 2: a review of initial published findings. Am J Clin Nutr.100(Suppl. 1):353S–358S

Osborn CY, Amico KR, Cruz N, O'Connell AA, Perez-Escamilla R, et al. (2010). A brief culturally tailored intervention for Puerto Ricans with type 2 diabetes. Health Educ Behav. 37:849–62. [PMC free article] [PubMed] [Google Scholar]

Oti SO, et al. (2013). The magnitude of diabetes and its association with obesity in the slums of Nairobi, Kenya: results from a cross-sectional survey. Tropical Medicine & International Health. 18(12): 1520–1530. DOI: https://doi.org/10.1111/tmi.12200

Pamela J Mink, Carolyn G Scrafford, Leila M Barraj, Lisa Harnack et al. (2007). Flavonoid Intake and Cardiovascular Disease Mortality: A Prospective Study in Postmenopausal Women. Am J Clin Nutr. 85(3):895-909.

Paniagua JA, Pérez-Martinez P, Gjelstad I, et al. (2011). A low-fat high-carbohydrate diet supplemented with long-chain n-3 PUFA reduces the risk of the metabolic syndrome. Atherosclerosis. 2011; 218: 443-50; http://dx.doi.org/10.1016/j.atherosclerosis.07.003

Papathanasopoulos A, & Camilleri M. (2010). Dietary fiber supplements: effects in obesity and metabolic syndrome and relationship to gastrointestinal functions. Gastroenterology. 138(1),65-72.

Park, M. Y., Kim, S. H., Cho, Y. J., Chung, R. H. & Lee, K. T. (2014). Association of Leisure Time Physical Activity and Metabolic Syndrome over 40 Years. Korean J Fam Med 35, 65–73.

Pender, N.J., Murdaugh, C. L., & Parsons, M.A. (2011). Health Promotion in Nursing Practice (6th Edition). Boston, MA: Pearson.

Penuela. R and Penuela.T. (2015). Relationship between blood pressure, age, gender, body mass index and short-term blood pressure varibility in untreated hypertensive patients. Journal of Hypertension. Volume 33, e-Supplement 1.

Petrella, E.; Malavolti, M.; Bertarini, V.; Pignatti, L.; Neri, I.; Battistini, N.C.; Facchinetti, F. (2014). Gestational weight gain in overweight and obese women enrolled in a healthy lifestyle and eating habits program. J. Matern. Fetal Neonatal Med. 27, 1348–1352. [CrossRef] [PubMed]

Phillips-Howard PA, Laserson KF, Amek N, Beynon CM, Angell SY, Khagayi S, et al. (2014). Deaths ascribed to non-communicable diseases among rural Kenyan adults are proportionately increasing: evidence from a health and demographic surveillance system, 2003–2010. PLoS One. 9(11): e114010.

Poddar. K.H, Hosig.K.W, Nickols-Richardson.S.M, Anderson.E.S, Herbert.W.G, and Duncan.S.E. (2009). Low-fat dairy intake and body weight and composition changes in college students. Journal of the American Dietetic Association, vol. 109, no. 8, pp. 1433–1438.

Popkin BM, Adair LS, Ng SW. (2012). Global nutrition transition and the pandemic of obesity in developing countries. Nutr Rev. 70(1):3-21.

Popkin BM, Gordon-Larsen P. (2004). The nutrition transition: worldwide obesity dynamics and their determinants. Int J Obes Relat Metab Disord. 28(3): S2–9.

Popkin BM. (2003). Dynamics of the nutrition transition and its implications for the developing world. Forum Nutr. 56:262–4

Prasad DS, Kabir Z, Dash AK, Das BC. (2012). Prevalence and risk factors for metabolic syndrome in Asian Indians: a community study from urban Eastern India. J Cardiovasc Dis Res. 3(3): 204–11.

Prochaska J, DiClemente C. (2003). Stage and processes of self-change of smoking toward an integrative model, J Consult Clin Psychol. 51(3): 390–395.

Prochaska JO, DiClemente CC. (1984). Stages and processes of self-change of smoking: toward an integrative model of change. J Consulting Clin Psychol. 51(3): 390-5. PubMed PMID: 6863699.

Pronk NP, Remington PL. (2015). Community Preventive Services Task Force. Combined diet and physical activity promotion programs for prevention of diabetes: community preventive services task force recommendation statement. Ann Intern Med. 163(6):437-51.

Rawan h. AL-Qawasmeh and Reema F. Tayyem. (2018). Dietary and Lifestyle Risk Factors and Metabolic Syndrome: Literature Review. Current Research in Nutrition and Food Science. 2018; 6. (3), 594-608

Rawlance Ndejjo, Fred Nuwaha, Hilde Bastiaens, Rhoda K. et al. (2020). Cardiovascular disease prevention knowledge and associated factors among adults in Mukono and Buikwe districts in Uganda. BMC Public Health. 20:1151 https://doi.org/10.1186/s12889-020-09264-6

Rebello, C. J., Greenway, F. L., & Finley, J. W. (2014). Whole grains and pulses: A comparison of the nutritional and health benefits. Journal of Agricultural and Food Chemistry. 62(29), 7029–7049

Rehm J, Baliunas D, Borges GL et al. (2010). The relation between different dimensions of alcohol consumption and burden of disease: an overview. Addiction 105(5), 817–843.

Rheinschild E. (2017). Fruit and veggies rich in potassium may be key to lowering blood pressure. https:// news. usc. edu/ 119637/ how- to lower-your- blood- pressure- eat- more- fruit- and- veggies.

Richard Ayah, Mark D Joshi, Rosemary Wanjiru, Elijah K Njau, C Fredrick Otieno et al. (2013). A population-based survey of prevalence of diabetes and correlates in an urban slum community in Nairobi, Kenya.

Rimkyo Yeo, So Ra Yoon, Oh Yoen Kim. (2017). The Association between Food Group Consumption Patterns and Early Metabolic Syndrome Risk in Non-Diabetic Healthy People. Clin Nutr Res. 2017 Jul;6(3):172-182. https://doi.org/10.7762/cnr.2017.6.3.172 pISSN 2287-3732·eISSN 2287-3740

Roberts C. K, Hevener A. L, & Barnard, R. J. (2013). Metabolic syndrome and insulin resistance: underlying causes and modification by exercise training. Comprehensive Physiology. 3(1),1-58.

Rodriguez-Monforte, M.; Sanchez, E.; Barrio, F.; Costa, B.; Flores-Mateo, G. (2017). Metabolic syndrome and dietary patterns: A systematic review and meta-analysis of observational studies. Eur. J. Nutr. 56, 925–947. [CrossRef] [PubMed].

Ronis MJ, Wands JR, Badger TM, de la Monte SM, Lang CH, Calissendorff J. (2007). Alcohol-induced disruption of endocrine signaling. Alcohol Clin. Exp. Res. 31(8), 1269–1285

Ros E. (2010). Health benefits of nut consumption. Nutrients. 2:652–82.

Rosen, C.S. (2000). Is the sequencing of change processes by stage consistent across health problems? A meta-analysis. Health. Psychol.19(6):593-604.

Ros E, Hu FB. (2013). Consumption of plant seeds and cardiovascular health: epidemiological and clinical trial evidence. Circulation. 128:553–565.

Roth GA, Forouzanfar MH, Moran AE, Barber R, Nguyen G, Feigin VL et al. (2015). Demographic and epidemiologic drivers of global cardiovascular mortality. N Engl J Med. 372(14): 1333–41. DOI: https://doi.org/10.1056/NEJMoa1406656

Sabate J, Oda K, Ros E. (2010). Nut consumption and blood lipid levels: a pooled analysis of 25 intervention trials. Arch Intern Med. 170:821–7.

Sabir.A.A, Jimoh.A and Iwualaetal.S.O. (2016). "Metabolic syndrome in urban city of North-Western Nigeria: Prevalence and determinants. Pan African Medical Journal. vol.23

Saboya PP, Bodanese LC, Zimmermann PR, Gustavo S, Macagnan E, Feoli AP, et al. (2016). Original Article Lifestyle Intervention on Metabolic Syndrome and its Impact on Quality of Life: A Randomized Controlled Trial. 60–9.

Sadikot S, & Hermans M. (2010). Here we go again… The metabolic syndrome revisited!. Diabetes & Metabolic Syndrome: Clinical Research & Reviews. 4(2), 111-120.

Salas R, Bibiloni MdM, Ramos E, Villarreal JZ, Pons A, et al. (2014). Metabolic Syndrome Prevalence among Northern Mexican Adult Population. PLoS ONE. 9(8): e105581. doi: 10.1371/journal.pone.0105581.

Salas-Salvado´J, Bullo M, Perez-Heras A, Ros E. (2006). Dietary fibre, nuts and cardiovascular diseases. Br J Nutr. 96 (suppl 2): S46–51.

Sala-Vila A, Estruch R, Ros E. (2015). New insights into the role of nutrition in CVD prevention. Curr Cardiol Rep. 17:26

Salehi-Abargouei A, Maghsoudi Z, Shirani F, Azadbakht L. (2013). Effects of Dietary Approaches to Stop Hypertension (DASH)-style diet on fatal or nonfatal cardiovascular diseases – incidence: A systematic review and meta-analysis on observational prospective studies. Nutrition. 29:611-8.

Salehi L, Eftekhar H, Mohammad K, Taghdisi MH, Shojaeizadeh D. (2010). Physical Activity among a Sample ofIranians Aged Over 60 Years: An Application of the Trans theoretical Model.Arch Iran Med. 13(6):528-36. PMID: 21039010.

Saneei P, Fallahi E, Barak F, et al. (2015). Adherence to the DASH diet and prevalence of the metabolic syndrome among Iranian women. Eur J Nutr. 54: 421–428.

Sarah L Dunn, Winnie Siu, Judith Freund, Stephen H Boutcher. (2014). The effect of a lifestyle intervention on metabolic health in young women. Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy.7 437–444

Savica V, Bellinghieri G, Kopple JD. (2010). The effect of nutrition on blood pressure. Annual review of nutrition. 30: 365-401.

Sayon-Orea C, Martinez-Gonzalez MA, Bes-Rastrollo M. (2011). Alcohol consumption and body weight: a systematic review. Nutr. Rev. 69(8), 419–431.

Sievenpiper JL, Kendall CW, Esfahani A, et al. (2009). Effect of non-oil-seed pulses on glycaemic control: a systematic review and meta-analysis of randomised controlled experimental trials in people with and without diabetes. Diabetologia. 52: 1479–1495.

Shehu A, Toti F, Carcani M, Lapardhaja A, Tare R. (2017). The prevalence of metabolic syndrome and its relation to metabolic control in patient with diagnosed type 2 Diabetes.

Shukri F. Mohamed, Martin K. Mutua, Richard Wamai, Frederick Wekesah, Tilahun Haregu et al. (2018). Prevalence, awareness, treatment and control of hypertension and their determinants: results from a national survey in Kenya. BMC Public Health.18(Suppl 3):1219 https://doi.org/10.1186/s12889-018-6052-y

Shu-Hung Chang, Nai-Hui Chien, Ching-Yi Yu. (2019). Long-Term Lifestyle Intervention in Elderly with Metabolic Syndrome. Journal of clinical nursing. 28(6):658-675.

Shu-Hung Chang, Miao-Chuan Chen, Nai-Hui Chien, Hsih-Fong Lin. (2016). Effectiveness of community-based exercise intervention programme in obese adults with metabolic syndrome. Journal of clinical nursing. 25 (17-18): 2579-2589

Slagter SN, van Vliet-Ostaptchouk JV, Vonk JM, Boezen HM, Dullaart RPF, et al. (2014) Combined Effects of Smoking and Alcohol on Metabolic Syndrome: The LifeLines Cohort Study. PLoS ONE 9(4): e96406. doi: 10.1371/journal.pone.0096406

Slagter SN, van Vliet-Ostaptchouk JV, Vonk JM, et al. (2013). Associations between smoking, components of metabolic syndrome and lipoprotein particle size. BMC Med. 11:195.

Sliwa K, et al. (2016). Impact of Socioeconomic Status, Ethnicity, and Urbanization on Risk Factor Profiles of Cardiovascular Disease in Africa. Circulation. 133(12): 1199–208. DOI: https://doi.org/10.1161/ CIRCULATIONAHA.114.008730.

Spellman, C.W. (2008). Archieving glycemic control: cornerstone in the treatment of patients with multiple risk factors. Journal of the American Osteopathic Association. 108:S8–S13 (PubMed: 19451256).

Spencer L, Adams TB, Malone S, Roy L, Yost E. (2006). Applying the transtheoretical model to exercise: a systematic and comprehensive review of the literature. Health Promot Pract. 7(4):428e43. http://dx.doi.org/10.1177/1524839905278900.

Stanton A, Grimshaw G. (2013). Tobacco cessation interventions for young people. Cochrane Database Syst Rev. 8:CD003289. http://dx.doi.org/10.1002/14651858.CD003289.pub5

Steffen LM, Van Horn L, Daviglus ML, et al. (2014). A modified Mediterranean diet score is associated with a lower risk of incident metabolic syndrome over 25 years among young adults: the CARDIA (Coronary Artery Risk Development in Young Adults) study. Br J Nutr. 112:1654–1661

Steemburgo T, Dall'Alba V, Almeida JC, Zelmanovitz T, Gross JL, de Azevedo MJ.(2009). Intake of soluble fibers has a protective role for the presence of metabolic syndrome in patients with type 2 diabetes. Eur J Clin Nutr. 63:127-133.

Strasser. B. (2013). Physical activity in obesity and metabolic syndrome. Annals of the New York Academy of Sciences. 1281(1),141-159.

Sun, K., Ren, M., Liu, D., Wang, C., Yang, C., & Yan, L. (2014). Alcohol consumption and risk of metabolic syndrome: a meta-analysis of prospective studies. Clinical Nutrition, 33(4), 596-602.

Sun K, Liu J, Ning G. (2012). Active smoking and risk of metabolic syndrome: a meta-analysis of prospective studies. PLoS One.7: e47791.

Surka S, Steyn K, Everett-Murphy K, Gaziano TA, Levitt N. (2015). Knowledge and perceptions of risk for cardiovascular disease: Findings of a qualitative investigation from a low-income peri-urban community in the Western Cape, South Africa. African J Prim Heal care Fam Med. 7(1):E1-8.

Tabak AG, et al. (2013). Pre-diabetes: a high-risk state for diabetes development. Lancet. 379(9833):2279–90.

Takahashi Risa, Calistus Wilunda, Karani Magutah, Wanja Mwaura-Tenambergen et al. (2017). Correlates of alcohol consumption in rural western Kenya: A cross-sectional study. BMC Psychiatry. 17: 175.DOI 10.1186/s12888-017-1344-9

Takashi Araoa, Yukio Oidaa, Chizuko Maruyamab, Takashi Mutouc et al. (2007). Impact of lifestyle intervention on physical activity and diet of Japanese workers. Preventive Medicine. 45(2–3), 146-152

TakahashiY,SasakiS, OkuboS, HayashiM,TsuganeS. (2006). Blood pressure change in a free-living population-based dietary modification study in Japan. J Hypertension. 24(3):451–8.

Tecla M. Temu1, Nicholas Kirui. Celestine Wanjalla, Alfred M. Ndungu, Jemima H. et al (2015). Cardiovascular health knowledge and preventive practices in people living with HIV in Kenya. BMC Infectious Diseases. 15:421. DOI 10.1186/s12879-015-1157-8

Temu TM, Kirui N, Wanjalla C, Ndungu AM, Kamano JH, Inui TS, et al. (2015). Cardiovascular health knowledge and preventive practices in people living with HIV in Kenya. BMC Infect Dis.15 (1):421.

Thomas R Einarson, Annabel Acs , Craig Ludwig, Ulrik H Panton.(2018). Economic Burden of Cardiovascular Disease in Type 2 Diabetes: A Systematic Review. Value Health. 2018. 21(7):881-890. doi: 10.1016/j.jval.2017.12.019

Thorp AA, McNaughton SA, Owen N, Dunstan DW. (2013). Independent and joint associations of TV viewing time and snack food consumption with the metabolic syndrome and its components; a cross-sectional study in Australian adults. Int J Behav Nutr Phys Act. 10.96. doi: 10.1186/1479-5868-10-96.

Thuita Ann Watetu, Beatrice Nyanchama Kiage, Arnold N. et al. (2020). Effect of a nutrition education programme on the metabolic syndrome in type 2 diabetes mellitus patients at a level 5 Hospital in Kenya: a randomized controlled trial. BMC Nutrition. 6:30 https://doi.org/10.1186/s40795-020-00355-6

Troy Hai Kiat Puar, MBBS, MRCP, Yingjuan Mok, Roy Debajyoti et al. (2016). Secondary hypertension in adults. Singapore Med J 57(5): 228-232

Van de Vijver SJM, Oti S, Addo J, de Graft-Aikins A, Agyemang C. (2015). Review of community-based interventions for prevention of cardiovascular diseases in low- and middle-income countries. Ethnicity & Health. 17(6):651–676.

Vatakencherry RJ and Saraswathy L. (2019). Prevalence of Metabolic syndrome among adults in a teaching hospital in Kochi, Central Kerala: A cross-sectional study. J Family Med Prim Care. 8:2079-83.

Vicki S. Conn, Adam R. Hafdahl, and David R. Mehr. (2011). Interventions to Increase Physical Activity Among Healthy Adults: Meta-Analysis of Outcomes. American Journal of Public Health. 101 (4).

Vicki Simpson. (2015). Models and Theories to Support Health Behavior Intervention and Program Planning. Health and Human Sciences

Vilela FA, Jungerman FS, Laranjeira R, Callaghan R. (2009). The transtheoretical model and substance dependence: theoretical and practical aspects. Revista brasileira de psiquiatria. 31(4):362e8. Epub.11/18. PubMed PMID: 19918675.

Visioli, F. (2011). Nutritional support in the pharmacological treatment of metabolic syndrome. European journal of pharmacology. 668: S43-S49.

Vrieze A, Holleman F, Zoetendal EG, de Vos WM, Hoekstra JB, Nieuwdorp M. (2010). The environment within: How gut microbiota may influence metabolism and body composition. Diabetologia. 53:606–13. [PMC free article] [PubMed].

Wahab KW, Kayode OO, Musa OI. (2015). Knowledge of stroke risk factors among Nigerians at high risk. J Stroke Cerebrovasc Dis. 24(1):125–9. Available from: http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2014.07.053 PMID: 25440355

Wakabayashi, I. (2014). Frequency of heavy alcohol drinking and risk of metabolic syndrome in middle-aged men. Alcoholism: Clinical and Experimental Research, 38(6), 1689-1696.

Wakabayashi.I. (2013). "Associations between heavy alcohol drinking and lipid-related indices in middle-aged men," Alcohol, vol. 47, no. 8, pp. 637–642.

Wang X, Ouyang Y, Liu J, Zhu M, Zhao G, Bao W, Hu FB. (2014). Fruit and vegetable consumption and mortality from all causes, cardiovascular disease, and cancer: systematic review and dose-response meta-analysis of prospective cohort studies. Bmj. 349: g4490.

Welsh JA, Sharma A, Cunningham SA, Vos MB. (2011). Consumption of added sugars and indicators of cardiovascular disease risk among US adolescents. Circulation. 123(3):249–57. [PMC free article] [PubMed] [Google Scholar].

Whitfield JB, Heath AC, Madden PA, et al. (2013). Metabolic and biochemical effects of low-to-moderate alcohol consumption. Alcohol Clin Exp Res. 37:575–586.

Whitt-Glover MC, Hunter JC, Foy CG, Quandt SA, Vitolins MZ, Leng I, et al. (2013). Translating the Dietary Approaches to Stop Hypertension (DASH) Diet for Use in Under resourced, Urban African American Communities, 2010. Prev Chronic Dis. 10:120088. DOI: http://dx.doi.org/10.5888/pcd10.120088

Whiting DR, Guariguata L, Weil C, Shaw J. (2011). IDF diabetes atlas: global estimates of the prevalence of diabetes for 2011 and 2030. Diabetes Res Clin Pract. 94:311–21

Wilkins J. N, Carlson H. E, Van Vunakis H, Hill M. A, Gritz E, & Jarvik M. E. (2001). Nicotine from cigarette smoking increases circulating levels of cortisol, growth hormone. Current Medical Research and Opinion. 17(2),132-141.

World Bank. (2006). Kenya - Inside informality: poverty, jobs, housing and services in Nairobi's slums Washington, DC.

World Health Organization. (2019). Hypertension. Fact Sheet. Geneva, Switzerland.

World Health Organization. (2017-a). Global Strategy on Diet, Physical Activity and Health. Geneva, Switzerland.

World Health Organization. (2017-b). Non-communicable disease education manual for primary health care professionals and patients, Geneva, Switzerland.

World Health Organization. (2017-c). The WHO STEP-wise approach to non-communicable disease risk factor surveillance (STEPS). Geneva, Switzerland.

World Health Organization. (2017-d). Cardiovascular diseases (CVDs). Fact Sheet. Geneva, Switzerland.

World Health Organization. (2016-a). Global report on diabetes. Global Strategy on Diet, Physical Activity and Health. Geneva, Switzerland.

World Health Organization. (2016-b). Global report on diabetes, Geneva, Switzerland.

World Health Organization. (2015). Guideline: Sugars Intake for Adult and Children. Geneva Switzerland.

World Health Organization. (2014-a). Kenya faces rising burden of diabetes. Geneva, Switzerland.

World Health Organization. (2014-b). Global status report on non-communicable diseases. Geneva, Switzerland.

World Health Organization (2012-a). Non-Communicable Diseases: An overview of Africa's New Silent Killers. Geneva, Switzerland.

World Health Organization. (2012-b). Guideline: Salt/sodium intake for adults and children. Geneva, Switzerland.

World Health Organization. (2012-c). Potassium intake for adults and children: guideline. Geneva: Geneva, Switzerland.

World Health Organization. (2011-a). WHO: Global Status Report on Non-communicable diseases. Geneva, Switzerland.

World Health Organization. (2011-b). "Waist Circumference and Waist-Hip Ratio. Report of a WHO Expert Consultation" http://whqlibdoc. who.int/publications/2011/9789241501491_eng.pdf

World Health Organization. (2011-c). Non-communicable disease country profiles, Kenya. 2011; http://www.who.int/nmh/ countries/ken_en.pdf.

World Health Organization. (2011-d). Global Recommendations on Physical Activity for Health 18 - 64 years old. Geneva, Switzerland.

World Health Organization. (2010-a). Global estimate of the burden of disease from second-hand smoke. Geneva, Switzerland.

World Health Organization. (2010-b). Global Recommendations on Physical Activity for Health. Geneva, Switzerland.

World Health Organization. (2010-c). Regional High-level Consultation; Tehran.Background Paper: Non-communicable diseases in low-and middle-income countries.

World Health Organization. (2010-d). Creating an enabling environment for population-based salt reduction strategies: report of a joint technical meeting held by WHO and the Food Standards Agency, United Kingdom. Geneva, Switzerland.

World Health Organization. (2010-e). Physical Status: The Use and Interpretation of Anthropometry.2010x; Available online: http://apps.who.int/iris/ bitstream/10665/37003/1/WHO_TRS_854.pdf.

World Health Organization. (2009-a). Kenya, Country Cooperation Strategy, at a glance. Available at: http://www.who. int/countryfocus/cooperation_strategy/ccsbrief_ken_ en.pdf.

World Health Organization. (2009-b). Global health risks: mortality and burden of disease attributable to selected major risks. Geneva: World Health Organization.

World Health Organization. (2009-c). Global health risks: mortality and burden of disease attributable to selected major risks. Geneva, Switzerland.

World Health Organization. (2008-b). Waist Circumference and Waist-Hip Ratio Report of a WHO Expert Consultation. Geneva, Switzerland.

World Health Organization. (2007). Prevention of cardiovascular disease: guidelines for assessment and management of total cardiovascular risk.

World Health Organization. (2006). International Diabetes Federation: Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia: Report of a WHO/IDF Consultation. 2006: Google Scholar.

World Health Organization. (2002-a). Integrated Management of Cardiovascular Risk. Report of a WHO Meeting. Noncommunicable Diseases and Mental Health, World Health Organization.

World Health Organization. (2002-b). Global Physical Activity Questionnaire (GPAQ) Analysis Guide. Surveillance and Population-Based Prevention of Noncommunicable Diseases. 2002. Geneva, Switzerland

World Health Organization. (1995). The use and interpretation of anthropometry. Report of a WHO Expert Committee. WHO Technical Report Series 854. Geneva, Switzerland.

World Medical Association Declaration of Helsinki. (2013). Ethical Principles for Medical Research Involving Human Subjects.

Xiaoran Liu, Yanping Li, Marta Guasch-Ferré, Walter C Willett et al. (2019). Changes in nut consumption influence long-term weight change in US men and women. bmjnph. 0:1–10. doi:10.1136/bmjnph-2019-000034

Yamaoka K, Tango T. (2012). Effects of lifestyle modification on metabolic syndrome: a systematic review and meta-analysis. BMC Med. 10.

Yasin, S.M., Taib, K.M., and Zaki, R.A. (2011). Reliability and construct validity of the Bahasa Malaysia version of transtheoretical model (TTM) questionnaire for smoking cessation and relapse among Malaysian adult. Asian Pac J Cancer Prev. 212(6): 1439-1443.

Yiqiang Zhan, Jinming Yu, Ruoqing Chen, Junling Gao1, Rongjing Ding, et al. (2012). Socioeconomic status and metabolic syndrome in the general population of China: a cross-sectional study. BMC Public Health. 12:921 http://www.biomedcentral.com/1471-2458/12/921

Yoona Kim, Jennifer B Keogh and Peter M Clifton. (2019). Does Nut Consumption Reduce Mortality and/or Risk of Cardio-metabolic Disease? An Updated Review Based on Meta-Analyses. Int. J. Environ. Res. Public Health. 16, 4957; doi:10.3390/ijerph16244957

Yuqiu L, Wright SCD. (2008). Knowledge and awareness of risk factors for cardiovascular disease in the garankuwa community. Heal SA Gesondheid. 13(3):3–4

Yusuf S, et al. (2004). Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): Case-control study. The Lancet. 364(9438): 937–952. DOI: https://doi.org/10.1016/S0140-6736(04)17018-9

Zagozdzon P, Parszuto J, Wrotkowska M, Dydjow-Bendek D. (2014). Effect of unemployment on cardiovascular risk factors and mental health. Occup Med (Lond). 64(6):436-41. Doi: 10.1093/occmed/kqu044. Epub. PMID: 24727560

Zhou B, Bentham J, Di Cesare M, Bixby H, Danaei G, Cowan MJ, et al. (2017). Worldwide trends in blood pressure from 1975 to 2015: a pooled analysis of 1479 population-based measurement studies with 19·1 million participants. Lancet. 389(10064):37–55.

Time Frame – Gantt chart

The total period planned for the project was 3 years. The implementation stages were as follows

Activity	**2017**		**2018**		**2019**		**2020-2021**	
	March-July	**August-November**	**January - April,**	**May-August**	**January-April**	**August-December**	**January-July, 2020**	**August, 2020-April, 2021**
Proposal development and presentation								
ERC- Review and Approval								
Getting full admission from UoN and Research Permit from NACOSTI								
First phase (baseline) data collection								
Second phase data collection								
Third phase data collection								
Data analysis and writing of manuscripts								
Report writing and submission of thesis for defending								

BUDGET

ITEM	UNIT COST (in Ksh)	QUANTITY	COST	TOTAL COST
FIELD EXPENSES				
Lab expenses	700	321x (4 times)	898,800	898,800
Research assistant	2000	8	16, 000	16,000
Research assistant training	2000	1	2000	2000
Questionnaire and consent form	5 ksh per page	5x22x321	35,310	35,310
For SMS and WhatsApp bundle	5000	5000	5000	5000
Data entry	1000	5x2x1000	10,000	10,000
Data analysis	40,000	40,000	40,000	40,000
Research site fee	6,000	6,000	6,000	6,000
Ethics Committee Fee	5,000	2,000	2,000	5,000
Sub- total				**1,018110**
STATIONARY SUPPLIES				
Pens (2 dozen)	600	600x2	1200	1200
Pencils (2 dozen)	180	180x2	360	360
Folders (10)	100	100x10	1000	1000
Foolscaps	½ ream	400	200	200
Stapler and staples	600	600	600	600
Hard disk	5,000	5,000	5,000	5,000
Sub-total				**19,160**
PROPOSAL AND THESIS				
Proposal development	500	500x4	2000	2000
2 Approval proposals	500	500x2	1000	1000
Photocopying final report	600	600 x7	4,200	4,200
Sub-total				**7,200**
Publications and conferences	35,000	35,000	35,000	**35,000**
GRAND TOTAL				**1, 079470**

APPENDICES

Appendix I: Informed Consent Form (ICF) in English

Serial No. ………………….. Date ……………….

Title of study: Community-based lifestyle interventions for the management and control of metabolic syndrome among adults at St. Mary's hospital, Nairobi, Kenya

Introduction

Investigator:

Mr. Okubatsion Tekeste Okube, PhD student, School of Nursing Sciences, University of Nairobi

Supervisors:

1. Dr. Samuel T. Kimani, Senior Lecturer, School of Nursing Sciences, University of Nairobi.
2. Dr. Waithira Mirie, Senior Lecturer, School of Nursing Sciences, University of Nairobi.

Part I: Investigator's Statement

My name is Okubatsion Tekeste Okube; I am a student at the University of Nairobi, pursuing a Ph.D. in community health nursing. I am carrying out this study to give a community-based lifestyle intervention for the management and control of metabolic syndrome among adults at St. Mary's Hospital, Nairobi. Metabolic syndrome is a cluster of risk factors for cardiovascular and type 2-diabetes. It is characterized by abdominal obesity, raised blood pressure, raised blood glucose level, and lipid level abnormalities.

This study will be carried out for 12 months among 352 participants. During the study period, your blood glucose, lipid level, blood pressure, and physical measurements will be taken three times and you will have face-to-face teaching regarding lifestyle modification to control metabolic syndrome.

Therefore, I kindly request you to participate in this study. This consent form provides you with information that you need to know so that you can decide whether to take part in the study or not. This form gives you information about the purpose, procedure, risks, benefits, confidentiality, and the process that will be expected during the study. Accepting to participate in this study is wholly voluntary.

Purpose of the study

The purpose of this study is to provide lifestyle intervention for the management of metabolic syndrome among adults. This will enhance early detection and timely management of metabolic syndrome thus reduces the risk of developing cardiovascular diseases and type 2 diabetes.

Therefore, you are invited to participate in this study because you are attending the St Mary's hospital where I am conducting the study. You may have the risk factors for cardiovascular disease and you will benefit from early diagnosis. I will measure your abdominal circumference and if it is above normal, you will be screened for the other components of metabolic syndrome (blood pressure, blood glucose, and lipid levels). If you have three or more of the features of metabolic syndrome, you will be invited to participate for 12 months of lifestyle intervention to control the components of metabolic syndrome. In the 12 months study period, you will be required to visit two times (at 6 and 12 months) for follow-up purposes.

Procedure of Study

After the screening, if you are eligible and agree to participate in the study, you will be asked questions about demographic and socio-economic characteristics, lifestyles, and knowledge about lifestyle modifications. Moreover, blood glucose and lipid levels will be measured by taking 3 mL of blood from the brachial vein and your fingertip. Blood pressure and physical measurements will also be measured.

Benefits

The assessment and examinations are free of charge and you will also benefit from knowing your general health status. Early identification of metabolic syndrome will help you prevent the development of chronic diseases like Diabetes and hypertension. Moreover, you will learn more about healthy eating habits associated with lowering the risks of chronic diseases by modifying your lifestyle.

Risks

There are minimal risks to you in participating in this study. The venepuncture may cause some temporary pain at the blood drawing site. However, the procedure is routinely used and presents almost no risk. The amount of blood that is sampled is very small (3mL).

Confidentiality

The information that you will provide and all the results will be strictly kept confidential. The interview will take place in a private room and the information you provide will be coded so that it cannot be identified. Only people working in this research study will have access to the information. Your name or other identifying information will not appear in any research records or reports.

Voluntary participation and withdrawal

Participation in the study is wholly voluntary. You may decline to participate or withdraw your consent to participate at any point during the study. If you withdraw, your care will not be affected in any way. You have a right to ask any questions or clarifications at any time during the study.

Persons to contacts

If you have any concerns or questions regarding to this study or concerning your rights, please contact any of the following persons.

Principal investigator

Okubatsion Tekeste Okube

School of Nursing Sciences, University of Nairobi

P. O. Box 19676 – 00200 NAIROBI

KENYATTA NATIONAL HOSPITAL

Mobile Phone Number: 0708552801

Email: tekeste.ok@gmail.com

First Supervisor

Dr. Dr. Samuel T. Kimani, PhD,

School of Nursing Sciences, University of Nairobi

P. O. Box 19676 – 00200 Nairobi

Mobile phone number: 0722384917

Second supervisor

Dr. Waithira Mirie, PhD,

School of Nursing Sciences, University of Nairobi.

P. O. Box 19676 – 00200 Nairobi

Mobile phone number: 0727142385

KNH/UoN Ethics and Research Committee

The Chairman,

KNH/UoN Ethics and Research Committee

P.O.Box 20723-00202 Nairobi.

Tel: 020-2726300-9 Ext 44102

Email: uonknh_erc@uonbi.ac.ke

Part II: Participants Declaration and Consent Form:

The study above has been explained to me. I have understood its purpose and my rights as a participant in the study. I have been given a chance to ask questions and have been assured that if in the future I have any concerns about the study or my rights as a subject, I can ask the investigator. I understand that I can withdraw from the study at any time. I voluntarily agree to participate in the study.

Signature

Signature of Participant ___________________________ Date: ___________________

Signature of research assistant ______________________ Date: ___________________

Signature of principal investigator ____________________Date: ___________________

Appendix II: Informed Consent Form (ICF)/: in KISWAHILI

Kiambatisho I: Fomu ya Ruhusa ya Kibali (ICF) kwa Kiingereza

Serial No .... Tarehe ....

Kichwa cha kujifunza: Mipango ya maisha ya jamii kwa usimamizi na udhibiti wa ugonjwa wa metaboliki kati ya watu wazima katika hospitali ya St Mary, Nairobi, Kenya

Utangulizi

Mtafiti:

Mheshimiwa Okubatsion Tekeste Okube, mwanafunzi wa PhD, Shule ya Sayansi ya Uuguzi, Chuo Kikuu cha Nairobi

Wasimamizi:

1. Daktari Samuel T. Kimani, Mhadhiri Mkubwa, Shule ya Sayansi ya Uuguzi, Chuo Kikuu cha Nairobi.
2. Daktari Sabina Wakasiaka, Mhadhiri Mkuu, Shule ya Sayansi ya Uuguzi, Chuo Kikuu cha Nairobi.

Sehemu ya I: Taarifa ya Mpelelezi

Jina langu ni Okubatsion Tekeste Okube; Mimi ni mwanafunzi katika Chuo Kikuu cha Nairobi kutafuta PhD katika uuguzi wa afya ya jamii. Ninafanya utafiti huu kutekeleza hatua za jamii za maisha kwa usimamizi na udhibiti wa ugonjwa wa metaboliki kati ya watu wazima katika Hospitali ya St. Mary's, Nairobi. Ugonjwa wa metaboli ni kikundi cha sababu za hatari kwa moyo na mishipa ya aina ya 2. Inajulikana kwa fetma ya tumbo, kukuza shinikizo la damu, kukuza kiwango cha damu ya glucose na kutofautiana kwa kiwango cha lipid.

Utafiti huu utafanyika kwa muda wa miezi 12 kati ya washiriki 320. Wakati wa utafiti, glucose yako ya damu, maelezo ya lipid, shinikizo la damu na vipimo vya kimwili zitachukuliwa mara nne na utakuwa na mafundisho ya uso kwa uso kuhusu muundo wa maisha ili kusimamia na kudhibiti ugonjwa wa metabolic.

Ninakuomba ushiriki kushiriki katika utafiti huu. Fomu hii ya idhini inakupa maelezo ambayo unahitaji kujua ili uweze kuamua kama kushiriki katika utafiti au la. Fomu hii inakupa habari kuhusu madhumuni, utaratibu, hatari, faida, siri na mchakato utakaotarajiwa wakati wa utafiti. Kukubali kushiriki katika utafiti huu ni kikamilifu kwa hiari.

Kusudi la utafiti

Kusudi la utafiti huu ni kutekeleza uingizaji wa maisha kwa ajili ya usimamizi wa ugonjwa wa kimetaboliki kati ya watu wazima. Hii itaongeza kutambua mapema na usimamizi wa wakati wa syndrome ya kimetaboliki na matatizo yake yanayohusiana na moyo na mishipa ya ugonjwa wa kisukari.

Kwa hiyo, umealikwa kushiriki katika utafiti huu kwa sababu unahudhuria hospitali ya St Mary ambapo ninaendesha utafiti. Unaweza kuwa na sababu za hatari kwa ugonjwa wa kimetaboliki na utafaidika kutokana na utambuzi wa mapema. Sasa nitapima mduara wako wa tumbo na ikiwa ni juu ya kawaida, utafuatiliwa kwa vipengele vingine vya ugonjwa wa metaboliki (shinikizo la damu, damu ya sukari na viwango vya lipid). Ikiwa una tatu au zaidi ya vipengele vya ugonjwa wa metabolic, utaalikwa kushiriki katika miezi 12 ya maisha ya kuingilia kati ili kudhibiti vipengele vya ugonjwa wa kimetaboliki. Katika kipindi cha kipindi cha miezi 12, utahitajika kutembelea mara tatu (kila baada ya miezi 4) kwa ajili ya kufuatilia.

Utaratibu wa Utafiti

Baada ya uchunguzi, ikiwa unastahiki na kukubali kushiriki katika utafiti huo, utaulizwa maswali kuhusu sifa za kiuchumi na kijamii na kiuchumi, maisha ya tabia na ujuzi kuhusu marekebisho ya maisha. Aidha, glucose ya damu na wasifu wa lipid utahesabiwa kwa kuchukua mL 3 ya damu kutoka kwenye mshipa wa brachial. Shinikizo la damu na vipimo vya kimwili pia utahesabiwa.

Faida

Tathmini na mitihani hazina malipo na pia utafaidika kutokana na hali yako ya afya ya jumla. Utambuzi wa mapema ya ugonjwa wa metaboliki utakusaidia kuzuia maendeleo ya magonjwa sugu kama kisukari na shinikizo la damu. Aidha, washiriki watajifunza zaidi kuhusu tabia za kula na afya zinazohusiana na kupunguza hatari ya magonjwa sugu kwa kubadilisha tabia zao za maisha.

Hatari

Kuna hatari ndogo kwako katika kushiriki katika utafiti huu. Mazingira yanaweza kusababisha maumivu ya muda mfupi kwenye tovuti ya kuchora damu itachukuliwa. Hata hivyo, utaratibu hutumiwa mara kwa mara na hutoa karibu hakuna hatari. Kiasi cha damu ambacho ni sampuli ni ndogo sana (3mL).

Usiri

Maelezo ambayo utatoa na matokeo yote yatawekwa kwa siri. Mahojiano yatafanyika katika chumba cha faragha na habari unazoyatoa itachukuliwa ili iweze kutambuliwa. Watu tu wanaofanya kazi katika utafiti huu wa utafiti watapata maelezo. Jina lako au habari nyingine za kutambua hazitaonekana katika kumbukumbu yoyote au ripoti.

Ushiriki wa hiari na uondoaji

Kushiriki katika utafiti ni kikamilifu kwa hiari. Unaweza kushuka kushiriki au kuondoa ridhaa yako kushiriki wakati wowote wakati wa utafiti. Ukiondoka, utunzaji wako hautaathirika kwa njia yoyote. Una haki ya kuuliza swali lolote au ufafanuzi wakati wowote wakati wa utafiti.

Watu kuwasiliana na:

Ikiwa una matatizo yoyote au maswali kuhusu utafiti huu au kuhusu haki zako, tafadhali wasiliana na mtu yeyote wafuatayo.

Mtafiti Mkuu:
Viliyoagizwa awali
Okubatsion Tekeste Okube
Shule ya Sayansi ya Uuguzi, Chuo Kikuu ya Nairobi.
Sanduku la P.O.B. 19676 - 00200 NAIROBI
KENYATTA HOSPITAL NATIONAL
Simu ya Mkono: 0708552801
Email: tekeste.ok@gmail.com

Msimamizi Mkuu Kwanza:
Dk. Samuel T. Kimani, PhD, Shule ya Sayansi ya Uuguzi, Chuo Kikuu cha Nairobi
P. Sanduku la O.B. 19676 - 00200 Nairobi
Nambari ya simu ya simu ya mkononi: 0722384917

Msimamizi wa Sekondari:
Dk. Sabina Wakasiaka, PhD, Shule ya Sayansi ya Uuguzi, Chuo Kikuu cha Nairobi.
P. Sanduku la O.B. 19676 - 00200 Nairobi
Nambari ya simu ya simu ya simu: 0727438359
KNH / UoN Kamati ya Maadili na UtafitiWekiti, KNH / UoN Kamati ya Maadili na Utafiti
P.O.Box 20723-00202 Nairobi.
Hii: 020-2726300-9 Ext 44102
Email: uonknh_erc@uonbi.ac .ke

Sehemu ya II:

Azimio la Washiriki na fomu ya kibali: Utafiti ulio juu umeelezewa kwangu. Nimeelewa kusudi lake na haki zangu kama mshiriki katika utafiti. Nimepewa fursa ya kuuliza maswali na nimehakikishiwa kuwa ikiwa katika siku zijazo nina matatizo yoyote kuhusu utafiti au haki zangu kama somo, naweza kumwuliza. Ninaelewa kwamba ninaweza kujiondoa kwenye utafiti wakati wowote. Mimi kwa hiari kukubali kushiriki katika utafiti.

Signature

Signature ya Mshiriki ________________________Tarehe: __________________

Sudio ya msaidizi wa utafiti ___________________Tarehe: __________________

Sudio ya mfuatiliaji mkuu _____________________ Tarehe: __________________

Title: Community-based lifestyle intervention for the management and control of metabolic syndrome among adults at St. Mary's Hospital, Nairobi, Kenya.

Participant's S.No:_______________

Date:________________

Gender:______________

Phone No.________________________, Email address:__________________________

Screening tool for chronic diseases

Conditions	Yes	No	Don't know	Comments
Known Hypertensive (HTN)				
Known Diabetes (DM)				
History of raised HDL				
History of raised TGs				
History of chronic disease(s) other than HTN & DM				

Appendix III: QUESTIONNAIRES

PART ONE: DEMOGRAPHIC INFORMATION OF PARTICIPANTS

1. Age in years:____________
2. What is your gender? [1] Male [2] Female
3. What is your current marital status:
   [1] Married [2] Single [3] Divorced [4] Separated [5] Widowed [6] Cohabitating
4. What is your ethnic group? ___________________________________
5. What is your religion?
   [1] Protestant [2] Catholic [3] Muslim Others (specify): _____________________
6. What is your highest level of education?
   [1] No formal education [2] Primary [3] Secondary [4] Tertiary/University
7. Where do you live? ____
8. What is your occupation: [1] Government employee [2] Non-government employee [3] self-employed [4] unemployed (able to work) [5] Unemployed (unable to work) [6] Doing housework at home [7] Retired/long-term disabled [8] Others (specify): ____
9. What is your/family net monthly income? __________________________ Ksh

PART TWO-A: QUESTIONNAIRE ON HISTORY OF RAISED BLOOD PRESSURE

S. No	**Question**	**Response**
1.HT	Have you ever had your blood pressure measured by a doctor or other health worker?	[1] Yes [2] No, If no, go to 1.DM
2.HT	Have you ever been told by a doctor or other health worker that you have raised blood pressure/hypertension?	[1] Yes [2] No, If no, go to 1.DM
3.HT	When did you know you have raised blood pressure or hypertension?	_________Years back. ________Months back.
4.HT	What was your age when you were told you have raised blood pressure or hypertension?	Age in years:__________
5.HT	Are you under anti-hypertensive medication?	[1] Yes [2] No
6.HT	Are you currently taking medication prescribed by a doctor to lower your blood pressure?	[1] Yes [2] No
6.HT	Is there anyone in your family who is hypertensive?	[1] Yes [2] No

PART TWO-B: QUESTIONNAIRE ON HISTORY OF DIABETES

S.No	Question	Response
1.DM	Have you ever had your blood sugar measured by a doctor or other health worker?	[1] Yes [2] No, If no, go to 1.C
2.DM	Have you ever been told by a doctor or other health worker that you have raised blood sugar or diabetes?	[1] Yes [2] No, If no, go to 1.C
3.DM	When did you know you have raised blood glucose or diabetes?	__________Years back. _________Months back.
4.DM	What was your age in years when you were told you have raised blood glucose or diabetes?	Age in years:___________
5.DM	Are you currently taking medication prescribed by a doctor to lower your blood glucose?	[1] Yes [2] No
6.DM	Is there anyone in your family who is diabetic?	[1] Yes [2] No

PART TWO-C: QUESTIONNAIRE ON HISTORY OF RAISED BLOOD CHOLESTEROL (TGs and HDL-C)

S.No	Question	Response
1.C	Have you ever had your cholesterol (fat levels in your blood) measured by a doctor or other health worker?	[1] Yes [2] No, If no, go to 7.C
2.C	Have you ever been told by a health worker that you have raised cholesterol?	[1] Yes [2] No, If no, go to 7.C
3.C	Have you been told in the past 6 months you have raised cholesterol?	[1] Yes [2] No
4.C	In the past two weeks, have you taken any oral medication for raised total cholesterol prescribed by a health worker?	[1] Yes [2] No
History of Cardiovascular Diseases		
7.C	Have you ever had a heart attack or chest pain from heart disease (angina) or a stroke (cerebrovascular accident)?	[1] Yes [2] No
8.C	Are you currently taking aspirin regularly to prevent or treat heart disease?	[1] Yes [2] No
9.C	Are you currently taking statins (lipid lowering drugs) (Lovastatin/Simvastatin/Atorvastatin or any other statin) regularly to prevent or treat heart disease?	[1] Yes [2] No

PART THREE: ANTHROPOMETRIC, CLINICAL AND BIOCHEMICAL MEASUREMENTS

Anthropometric, Blood Pressure (BP) and Heart Rate (HR) measurements for follow up visits									
Visits	WC (cm)	Hip (cm)	W/H Ratio	Weight (Kg)	Height (cm)	BMI	SBP mmHg	DBP mmHg	HR
1st visit At baseline									
2nd visit At 6 months									
3rd visit At 12months									

Biochemical measurements for follow up visits				
	Blood sugar levels	Blood cholesterol levels		
	FBGL	HDL	TGs	
1st visit At baseline				
2nd visit: At 6 months				
3rd visit: At 12 months				

Key: WC: waist circumference; **BP**: Blood Pressure; **FBG**L: fasting blood glucose level**; RBGL:** Random blood glucose level**; TGs:** Triglycerides**; HLD**: High density lipoprotein**, BMI:** Body mass index.

PART FOUR-A: QUESTIONNAIRE ON GENERAL DIETARY INTAKE PATTERNS

S.No	Question	Response
1.D	How many meals do you usually eat **per day** (breakfast, lunch, dinner, snacks)? Consider every fruit, every yoghurt, or a glass of milk etc. as a single snack.	[1] 1 meal [2] 2 meals [3] 3 meals [4] 4 meals [5] ≥ 5 meals
2.D	Among the meals you take daily, which one is the highest in quantity?	[1] Breakfast [2] Lunch [4] Dinner [4] snacks
3.D	How often do you eat **processed/fast foods**? (such as chips, sandwiches, hamburgers, fried chicken, french fries, sausages, samosas, pizza, hot dogs, ice cream etc…)	[1] Always [2] Often [3] Sometimes [4] Rarely [5] Never
4.D	Which type of bread do you regularly choose?	[1] Whole meal bread/brown [2] White bread [3] Both brown and white [4] I don't take bread
5.D	How often do you include **legumes/pulses** (beans, peas, chick peas, lentils) in your diet?	[1] Always [2] Often [3] Sometimes [4] Rarely [5] Never
6.D	How often do you include **nuts** (peanuts, groundnuts) in your diet?	[1] Always [2] Often [3] Sometimes [4] Rarely [5] Never
7.D	If you are working, do you carry your own food for lunch?	[1] Yes [2] No [3] N/A
8.D	How many times **per day** do you include as your meals the following food items**?**	
a.	**Starch** (bread, ugali, maize, cereal, pasta, rice, potato and grains …)	[1] Never [2] Once [3] twice [4] trice [5] 4 or more times
b.	Dairy products (milk, yogurt, Cheese, butter…)	[1] Never [2] Once [3] twice [4] trice [5] ≥ 4 times
c.	Meat, fish, poultry, eggs, cheese	[1] Never [2] Once [3] twice [4] trice [5] ≥4 times
d.	Fat (butter, margarine, oil, salad dressing, sour cream, cream cheese)	[1] Never [2] Once [3] twice [4] trice [5] ≥4 times
e.	Sweets (candies, cake, chocolate, table sugar, soda, honey, jam, juice with added sugar).	[1] Never [2] Once [3] twice [4] trice [5] ≥4 times
12.D	What percentage of your meal plate is filled by **proteins** (meat, chicken, fish, eggs, cheese, , beans…)?	[1] < 25% [2] 25% [3] 50% [3] ≥75%
13.D	What percentage of your meal plate is filled by **carbohydrates** (Ugali, rice, potato, chapatti, maize..)?	[1] < 25% [2] 25% [3] 50% [3] ≥75%
14.D	What percentage of your meal plate is filled by **vegetables/fruits**?	[1] < 25% [2] 25% [3] 50% [3] ≥75%

15.D	At what time do you usually take you dinner?	[1] Before 8 pm [2] 8- 9 pm [3] 9 - 10pm [4] After 10 pm
16.D	At what time do you usually sleep?	[1] 8- 9 pm [2] 9 - 10pm [3] 10 -11 pm [4] 11- mid night [5] After mid-night
17.D	Calculated time interval between dinner and sleep	_________hrs________mns

PART FOUR-B: QUESTIONNAIRE ON FRUITS AND VEGETABLES CONSUMPTION

1.F	How often do you eat **fruits**? (such as apple, banana, orange, tangerine, melon…)	[1] Daily [2] 5-6 days per week [3] 3-4 days per week [4] 1-2 days per week [5] Less than once per week [6] I don't eat fruits
2.F	In a typical **week**, on how many **days** do you eat fruits?	Number of days:___________ If Zero days, go to D4
3.F	How many times do you eat **Fruits** on one of those days?	__________
4.F	How many **servings** of fruits do you eat on one of those days?	Number of servings:__________
5.F	In a typical week, on how many **days** do you drink fruit juice?	Number of days:___________
1.V	How often do you eat **vegetables**? (salad vegetables (e.g.tomato, Carrot, cucumber) or cooked vegetables).	[1] Daily [2] 5-6 days per week [3] 3-4 days per week [4] 1-2 days per week [5] Less than once per week [6] I don't eat vegetables
2.V	In a typical week, on how many **days** do you eat vegetables?	Number of days:___________
3.V	How many times do you eat **Vegetables** on one of those days?	_____________
4.V	How many servings of vegetables do you eat on one of those days?	Number of servings:________

PART FOUR-C: QUESTIONNAIRE ON SALT INTAKE

1.D	Do you take salt?	[1] Yes [2] No
2.D	Do you regularly add salt to foods during **cooking**?	[1] Yes [2] No
3.D	How often do you add salt or a salty sauce such as soya sauce to your food right before you eat it or as you are eating it (**on table**)?	[1] Always [2] Often [3] Sometimes [4] Rarely [5] Never
5.D	Approximately, how many teaspoons of salt do you take per day?	Number of teaspoons:__________
6.D	How often do you eat **<u>processed foods</u>** high in salt? (such as packaged salty snacks, canned salty foods, sausage, ham salty food prepared at a fast food restaurant, cheese, bacon and processed meat).	[1] Always [2] Often [3] Sometimes [4] Rarely [5] Never
7.D	How much salt or salty sauce do you think you consume?	[1] Far too much [2] Too much [3] Just the right amount [4] Too little
8.D	How important to you is lowering salt in your diet?	[1] Very important [2] Somewhat important [3] Not at all important [4] Don't know
9.D	Do you know the recommended **salt** intake per day per person?	[1] Yes [2] No
10.D	If yes, how much is the recommended salt intake per person per day?	Number of tea spoons of salt per day:_________
11.D	**Do you do any of the following on a regular basis to control your salt intake?**	
a.	Limit consumption of processed foods	[1] Yes [2] No
b.	Look at the salt or sodium content on food labels	[1] Yes [2] No
c.	Buy low salt/sodium alternatives	[1] Yes [2] No
d.	Use spices other than salt when cooking	[1] Yes [2] No
e.	Avoid eating foods prepared outside of a home	[1] Yes [2] No
The next questions ask about the **oil or fat** that is most often use for meal preparation in your household, and about meals that you eat outside a home.		
12. D	What type of **oil** or fat do you most often use for meal preparation in your household?	[1] Vegetable oil [2] Butter or ghee [3] Margarine [4] None in particular [5] None used [77] Don't know

PART FOUR-D: QUESTIONNAIRE ON SUGAR INTAKE

S. No	Question	Response
1.S	Do you take sugar?	[1] Yes [2] No
2.S	How often do you take sugar?	[1] Always [2] Often [3] Sometimes [4] Rarely [5] Never
3.S	Approximately, how many teaspoons of sugar do you take per day?	Number of teaspoons: __________
4.S	Do you take coffee/tea/chocolate?	[1] Yes [2] No
5.S	If yes, how many cups of coffee/tea/chocolate do you take per day?	Number of cups of coffee/ tea/chocalate per day:________
6.S	How many tea spoons of sugar do you add in a cup of coffee/tea/chocalate?	Number of tea spoons of sugar per cup:_________
7.S	**Actual intake** of number of tea spoons of sugar per day	________________
8.S	Do you know the recommended sugar intake per person per day?	[1] Yes [2] No
9.S	If yes, how much is the recommended sugar intake per day?	Number of tea spoons of sugar per day:_________
10.S	How often do you take sugar contained beverages?	[1] Always [2] Often [3] Sometimes [4] Rarely [5] Never
11.S	How often do you eat cakes, sweets, chocolate or biscuits?	[1] Always [2] Often [3] Sometimes [4] Rarely [5] Never
12.S	How much sugar do you think you consume?	[1] Far too much [2] Too much [3] Just the right amount [4] Too little [5] Far too little
13.D	How important to you is reducing sugar intake?	[1] Very important [2] Somewhat important [3] Not at all important [4] Don't know
14.D	Do you think that too much sugar could cause a health problem?	[1] Yes [2] No [3] I don’t know
15.D	**Do you do any of the following on a regular basis to control your sugar intake?**	
a.	Limit consumption of sugar contained beverages	[1] Yes [2] No
b.	Look at the sugar content on food labels and drinks	[1] Yes [2] No
c.	Buy low sugar alternatives	[1] Yes [2] No
d.	Avoid drinking any sugar contained beverages	[1] Yes [2] No

PART FIVE: QUESTIONNAIRE ON ALCOHOL CONSUMPTION

S.No	Question	Response
1.A	Have you ever consumed any alcohol such as beer, wine, spirits etc… or local drinks (Chang'aa, Busaa…)?	[1] Yes [2] No If No, go to 1 D
2.A	**Do** you drink formal alcohol (beer, wine, spirits etc…) or informal/local drinks (Chang'aa, Busaa…)?	[1] formal drinks [2] informal drinks [3] both formal and local drinks
3.A	If formal, which type (s) of formal alcohol do you take?	[1] Beer [2] Wine [3] Spirits [4] Vodka [5] Gin [6] whisky [6] others:___________________
4.A	If informal, which type (s) of local drinks do you take?	[1] Chang'aa [2] Busaa [3] Muratina Others:_______
5.A	For how long have you been taking alcohol?	_______Years. ______,Months
6.A	Have you consumed any alcohol within the past 12 months?	[1] Yes , [2] No
7.A	Have you consumed any alcohol within the past **6** months?	[1] Yes , [2] No
8.A	Have you consumed any alcohol within the past **30** days?	[1] Yes [2] No
9.A	During the past **30 days**, on how many occasions did you have at least one standard alcoholic drink?	Number of occasions:_________
10.A	During the past 30 days, how many standard drinks on average did you have during one drinking occasion?	Number of SDs:____________
11.A	In a typical week, average, on how many days **do** you drink alcohol?	[1] Daily [2] 5-6 days per week [3] 3-4 days per week [4] 1-2 days per week
12.A	How many standard drinks **do** you take on those days?	[1] < 1 standard drink [2] 1-2 standard drinks [3] 3-4 standard drinks [4] ≥ 5 standard drinks
14.A	While you **are** drinking, do you also smoke?	[1] Yes [2] No
15.A	When you **are** drinking, do you accompany with food bites?	[1] Yes [2] No
16.A	If yes, which type of foods do you usually take?	[1] Namachoma alone [2] Namachoma with Ugali [3] Fried meat [4] Chicken [5] Chips [6] others:___________

PART SIX: QUESTIONNAIRE ON TOBACCO SMOKING

1.T	How would you describe your smoking experience at present?	[1] I smoke every day [2] I smoke but not every day [3] I don't smoke but I used to smoke every day [4] I don't smoke but I have smoked occasionally [5] I have never smoked.
2.T	Which products do you smoke?	[1] Manufactured cigarettes [2] Hand-rolled cigarettes [3] Pipes full of tobacco [4] Cigars [5] Shisha [6] others:___________________
3.T	How often do you smoke?	[1] Daily [2] 5-6 days per week [3] 3-4 days per week [4] 1-2 days per week [5] 1-3 days per month [6] Less than once a month
4.T	How many sticks of cigarettes do you smoke per **<u>day</u>**?	No. of sticks of cigarettes:_______
5.T	How many sticks of cigarettes do you smoke per **<u>week</u>**?	No. of sticks of cigarettes:_______
6.T	How many sticks of cigarettes do you smoke per **<u>month</u>**?	No. of sticks of cigarettes:_______
7.T	At what age did you first start smoking tobacco?	Age in years:____________
8.T	For how long have you been smoking?	_______Years,_______Months
9.T	Do you currently use any smokeless tobacco products such as (snuff, chewing tobacco, betel)?	[1] Yes [2] No
10.T	In the past, did you ever use smokeless tobacco products such as (snuff, chewing tobacco, or betel)?	[1] Yes [2] No

PART SEVEN: QUESTIONNAIRE ON PHYSICAL ACTIVITY/EXERCISE

S.No	Question	Response
1.P	Do you exercise regularly (i.e. 3o minutes of moderately intense activity for 5-7 days in a week, or vigorous physical activity **3 times** a week for **≥ 20** minutes each time)?	[1] Yes [2] No
2.P	What type (s) of exercises do you do?	[1] Walking [2] Running [3] Rugby [4] Jumping rope [5] Playing basketball/volleyball [6] Washing clothes/car… [7] Others, specify:_____________
3.P	On average, how many **days in a week** do you exercise for at least 30 minutes?	[1] None [2] 1-2 days [3] 3-4 days [4] 5 or more days
4.P	Approximately, how many **minutes** do you exercise on those days?	Number of minutes per day:________
5.P	Does your **work** involve **vigorous-intensity** activity that causes large increases in breathing or heart rate like (carrying or lifting heavy loads, digging or construction work) for at least 10 minutes continuously?	[1] Yes [2] No, If no, go to 9.P
6.P	In a typical week, on how many days do you do vigorous intensity activities as part of your work?	Number of days:_____________________
7.P	How much **time** do you spend doing vigorous-intensity activities at work on a typical day?	Hours :_________ Minutes:_________
8.P	Does your **work** involve **moderate-intensity** activity that causes small increases in breathing or heart rate such as brisk walking (or carrying light loads) for at least 10 minutes continuously?	[1] Yes [2] No, If no, go to 13.P
9.P	In a typical week, on how many days do you do moderate intensity activities as part of your work?	Number of days:_____
10.P	How much time do you spend doing moderate-intensity activities at work on a typical day?	Hours :_________ Minutes:_________
Travel to and from places		
The next questions **exclude** the physical activities at work that you have already mentioned. Now, I would like to ask you about the usual way you travel to and from places. For example to work, for shopping, to market, to place of worship.		
11.P	Do you walk for at least 10 minutes continuously to get to and from places?	[1] Yes [2] No

12.P	In a typical week, on how many days do you walk for at least 10 minutes continuously to get to and from places?	Number of days:_____
13.P	How much time do you spend walking for travel on a typical day?	Hours :_________ Minutes:_________
14. P	Which mode of transportation do you usually use?	[1] drive personal care [2] use public bus/matatu [3] use motor bike [4] walk
Recreational activities		
The next questions **exclude** the work and transport activities that you have already mentioned. Now, I would like to ask you about sports, fitness and recreational activities (leisure).		
15.P	Do you do any **<u>vigorous-intensity</u>** sports, fitness or recreational (leisure) activities that cause large increases in breathing or heart rate like [running, rugby, football] for at least 10 minutes continuously?	[1] Yes [2] No, If no, go to 18.P
16.P	In a typical week, on how many days do you do vigorous intensity sports, fitness or recreational (leisure) activities?	Number of days:________
17.P	How much time do you spend doing vigorous-intensity sports, fitness or recreational activities on a typical day?	Hours :_________ Minutes:_________
18.P	Do you do any **<u>moderate-intensity</u>** sports, fitness or recreational (leisure) activities that cause a small increase in breathing or heart rate such as brisk walking, [cycling, swimming, volleyball] for at least 10 minutes continuously?	[1] Yes [2] No, If no, go to 21.P
19.P	In a typical week, on how many days do you do moderate intensity sports, fitness or recreational (leisure) activities?	Number of days:_____
20.P	How much time do you spend doing moderate-intensity sports, fitness or recreational (leisure) activities on a typical day?	Hours :_________ ______ Minutes:______________
Sedentary behavior		
The following question is about sitting or reclining at work, at home, getting to and from places, or with friends including time spent sitting at a desk, sitting with friends, traveling in car, bus, train, reading, playing cards or watching television, but do not include time spent sleeping.		
21.P	How much time do you usually spend sitting or reclining on a typical day?	Hours :_________ Minutes:_________

PART EIGHT: QUESTIONNAIRE ON KNOWLEDGE OF PARTICIPANTS OF CVDs RISK FACTORS AND PREVENTIVE MEASURES

Part I: General knowledge on diabetes, hypertension and related cardiovascular diseases

1. Why do you think many people in Kenya are suffering from diabetes, hypertension and related cardiovascular diseases like heart attack, stroke…)?
   ____
   ____
2. Do you think the problem of type-2 diabetes, hypertension and their related cardiovascular diseases can be prevented/controlled? [1] Yes [2] No [3] I don't know
3. If yes, what preventive measures can be done?
   ____
   ____
   ____

Part II: Smoking related questions

1. Do you smoke any tobacco products? [1] Yes [2] No
2. If yes, have you ever been advised to stop tobacco smoking? [1] Yes [2] No
3. If yes, why do you think, you were advised to stop smoking?
   ____
   ____
4. Do you think tobacco smoking causes health problem? [1] Yes [2] No [3] I don't know
5. If yes, what health problems/conditions can be caused by tobacco smoking?
   ____
   ____

Part III: Alcohol related questions

1. Do/did you take alcohol? [1] Yes [2] No
2. If you were/are drinking alcohol, have you ever been advised to stop drinking?
   [1] Yes [2] No
3. If yes, why do you think, you were advised to stop drinking alcohol?
   ____
4. Do you think excessive alcohol consumption causes health problem? [1] Yes [2] No
   [3] I don't know
5. If yes, what health problems/conditions can be caused by excessive alcohol consumption?
   ____

Part IV: Diet related questions (Salt)

1. Do you take salt? [1] Yes [2] No
2. Have you ever been advised to stop or reduce taking salt? [1] Yes [2] No
3. If yes, why do you think, you were advised to **stop or reduce** taking salt?
   ____
4. What is the recommended salt intake per person per day?________________________

5. Do you think taking too much salt causes health problem? [1] Yes [2] No [3] I don't know
6. If yes, what health problems/conditions can be caused by taking too much salt?

   ____
7. Do you take sugar? [1] Yes [2] No
8. What is the recommended **sugar** intake per person per day?____________________
9. Do you think taking too much sugar causes health problem? [1] Yes [2] No
   [3] I don't know
10. If yes, what health problems/conditions can be caused by taking too much sugar?

    ____
11. What do you think the health consequences of eating jack foods (chips, sandwiches, hamburgers, fried chicken, French fries, sausages, samosas, pizza, hot dogs, ice cream)?

    ____

12. What do you know about the importance of fruits and vegetables consumption?

    ____

Part V: Physical activity related questions

1. Do you exercise? [1] Yes [2] No
2. Have you ever been advised to exercise? [1] Yes [2] No
3. If yes, why do you think, you were advised to be active?

   ____
4. Have you started exercising since then? [1] Yes [2] No
5. Can you mention some of the benefits of doing exercise?

   ____

Part VI: Body weight related questions

1. Do you know your weight? [1] Yes [2] No
2. Do you think your weight is within normal range? [1] Yes [2] No [3] don't know
3. If no, what do you think the risks of being overweight?

   ____
4. Have you ever been advised to reduce weight? [1] Yes [2] No
5. If yes, why do you think, you were advised to cut on your weight?

   ____
6. Have you tried reducing your weight? [1] Yes [2] No
7. What methods have you employed to help you reduce weight?

   ____

PART NINE-A: QUESTIONNAIRE ON STAGE OF CHANGE USING TRANS-THEORETICAL MODEL (TTM)

Participants were asked to choose on the statement that best describes their status.		
Description	**#**	**Stage of change**
1. Fruits consumption		
Please circle the number corresponding to the best description of your current stage of fruits intake.		
I am not currently consuming 5 or more servings of fruits in a day and I am not thinking of doing so in the coming 6 months.	1	Pre-contemplation stage
I am not currently consuming 5 or more servings of fruits in a day but I have thought about that.	2	Contemplation stage
I am not currently consuming 5 or more servings of fruits in a day but I plan to do so within the next 30 days.	3	Preparation stage
I am currently consuming 5 or more servings of fruits in a day but I have only been doing so for less than 6 months.	4	Action stage
I am currently consuming 5 or more servings of fruits in a day and I have been doing so for 6 months or more.	5	Maintenance stage
2. Vegetables consumption		
Please circle the number corresponding to the best description of your current stage of fruit and vegetable consumption.		
I am not currently consuming 5 or more servings of vegetables in a day and I am not thinking of doing so in the coming 6 months.	1	Pre-contemplation stage
I am not currently consuming 5 or more servings of vegetables in a day but I have thought about that.	2	Contemplation stage
I am not currently consuming 5 or more servings of in vegetables a day but I plan to do so within the next 30 days.	3	Preparation stage
I am currently consuming 5 or more servings of in vegetables a day but I have only been doing so for less than 6 months.	4	Action stage
I am currently consuming 5 or more servings of in vegetables a day and I have been doing so for 6 months or more.	5	Maintenance stage
3. Stage of change on adherence to the DASH eating plan		
Please circle the number corresponding to the best description of your current stage of adherence to the DASH eating plan		
I am not currently adhering to the DASH eating plan and I am not thinking of doing so in the coming 6 months.		Pre-contemplation stage
I am not currently adhering to the DASH eating plan but I have thought about that.		Contemplation stage
I am not currently adhering to the DASH eating plan but I plan to do so within the next 30 days.		Preparation stage

I am currently adhering to the DASH eating plan but I have only been doing so for less than 6 months.		Action stage
I am currently adhering to the DASH eating plan and I have been doing so for 6 months or more.		Maintenance stage
4. **Consumption of processed/fast foods**		
Please circle the number corresponding to the best description of your current stage of processed/fast foods consumption.		
I am currently often eating processed/fast foods and I am not thinking of stopping or reducing in the coming 6 months.		Pre-contemplation stage
I am currently often eating processed/fast foods but I have thought about avoiding or limiting those foods.		Contemplation stage
I am currently often eating processed/fast foods but I plan to avoid or limit those foods.		Preparation stage
I am currently avoided or limited often eating processed/fast foods but I have only been doing so for less than 6 months.		Action stage
I am currently avoided or limited often eating processed/fast foods and I have been doing so for 6 months or more.		Maintenance stage
5. Salt Intake (World Health Organization Guideline, 2012s)		
I am currently taking 1 teaspoon (5 g) or more salt a day and I am not thinking of stopping or reducing in the coming 6 months.	1.	Pre-contemplation stage
I am currently taking 1 teaspoon (5 g) or more salt a day but I intend to stop or reduce in the next 6 months.	2.	Contemplation stage
I am currently taking 1 teaspoon (5 g) or more salt a day but I intend to stop in the next 30 days.	3.	Preparation stage
I have stopped or reduced salt intake to less than 1 teaspoon for less than 6 months.	4.	Action stage
I have stopped or reduced salt intake to less than 1 teaspoon for 6 months or more.	5.	Maintenance stage
6. Sugar Intake (World Health Organization recommendation)		
I am currently taking more than 5 teaspoons of sugar and I am not thinking of stopping in the coming 6 months.	1.	Pre-contemplation stage
I am currently taking more than 5 teaspoons of sugar but I intend to stop in the next 6 months.	2.	Contemplation stage
I am currently taking more than 5 teaspoons of sugar but I intend to stop in the next 30 days.	3.	Preparation stage
I have stopped or reduced sugar intake to ≤ 5 teaspoons for less than 6 months.	4.	Action stage
I have stopped or reduced sugar intake to ≤ 5 teaspoons for 6 months or more.	5.	Maintenance stage
7. Alcohol Cessation (for people with history of alcohol intake)		
Please circle the number corresponding to the best description of your current stage of alcohol intake		

Currently, I am taking alcohols and I am not thinking of stopping in the coming 6 months.	1.	Pre-contemplation stage
I am currently taking alcohols but I intend to stop in the next 6 months.	2.	Contemplation stage
Currently, I am taking alcohols, but I intend to stop in the next 30 days.	3.	Preparation stage
I have stopped taking alcohols but not yet 6 months since then.	4.	Action stage
I have stopped taking alcohols for 6 months or more/never taken it	5.	Maintenance stage
8. Smoking Cessation (for people with history of smoking)		
Please circle the number corresponding to the best description of your current stage of smoking		
Have you stopped smoking? OR you have never smoked		
NO, and I am not thinking of stopping in the coming 6 months.		Pre-contemplation stage
NO, but I intend to stop in the next 6 months.		Contemplation stage
NO, but I intend to stop in the next 30 days		Preparation stage
YES, I have stopped smoking but not yet 6 months since then.		Action stage
YES, I have stopped smoking for 6 months or more or Never smoked		Maintenance stage
9. Physical Activity/Exercise		
Please circle the number corresponding to the best description of your current stage of Exercise.		
Do you exercise regularly (i.e. 3o minutes of moderately intense activity over the course of the day every day, or almost, or one vigorous physical activity three times a week for 20 minutes each time)?		
NO, and I do NOT plan to do so within the next 6 month		Pre-contemplation stage
NO, but I plan to do so in the next 6 months.		Contemplation stage
NO, but I plan to start doing so within the next 30 days.		Preparation stage
YES, I have been doing so for less than 6 months.		Action stage
YES, I have been doing so for over 6 months.		Maintenance stage

PART NINE-B: ASSESSMENT OF SELF-EFFICACY REGARDING LIFESTYLE MODIFICATION

S.No	**How confident are you that you can make this change?** (not at all confident =1, somewhat confident = 2 and extremely confident = 3).	**1.**	**2.**	**3.**
1.	Eat 5 or more servings of fruits a day			
2.	Eat 5 or more servings of vegetables a day			
3.	Observe and adhere to the DASH eating plan			
4.	Avoid or limit eating processed/fast foods			
5.	Take ≤ 1 teaspoon of salt per day			
6.	Take ≤ 5 teaspoons of sugar per day			
7.	Stop or moderate alcohol consumption			
8.	Cessation of smoking			
9.	Exercise for at least 30 minutes for 5-7 days per week			

PART NINE-C: ASSESSMENT OF DECISIONAL BALANCE REGARDING LIFESTYLE MODIFICATION

Decisional balance on dietary patterns	Scale		
Please indicate whether you agree or disagree with the following items when you are deciding whether or not to consume Fruits and Vegetables on daily basis. (disagree =1, somewhat = 2 and agree = 3).	1	2	3
Decisional balance- Pros			
1. I feel I am doing something good for my body if I eat more fruits and vegetables.			
2. Fruits and vegetables are low in fat			
3. Eating fruits and vegetables could help me live a better, healthier, and longer life			
4. Eating fruits and vegetables regularly could help me prevent diseases			
5. Eating fruits and vegetables could help me lose or maintain my weight			
6. Fruits and vegetables are a good substitute for junk food			
7. Eating five or more servings of fruits and vegetables per day could help me prevent hypertension and diabetes.			
8. I can find reasonably priced fruits and vegetables in my local markets			
9. Limiting salt intake to 1 tea spoon or less per day could help me prevent hypertension and diabetes.			
10. Limiting sugar intake to 5 tea spoons or less per day could help me prevent hypertension and diabetes.			
11. Limiting or avoiding processed/fast foods could help me prevent hypertension and diabetes.			
12. Limiting or avoiding alcohol consumption could help me prevent hypertension and diabetes.			
13. It is important for me to do exercise that makes me feel good			
14. Exercising for at least 30 minutes for 5-7 days per week			
15. Exercising for at least 30 minutes for 5-7 days per week could help me lose or maintain my weight			
16. Exercising for at least 30 minutes for 5-7 days per week could help me prevent hypertension and diabetes.			
Decisional balance - Cons			
1. Fruits and vegetables are too expensive to buy			
2. It is too difficult to eat five or more servings of fruits and vegetables each day			
3. I worry about the safety of chemicals used in fruits and vegetables.			
4. I have limited ways to incorporate fruits and vegetables in my meals.			
5. It is difficult to control daily salt and sugar intake			
6. Food with less salt or sugar do not tasty well			
7. Processed/fast foods are much available and it is difficult to limiting or avoiding them			
8. Processed/fast foods are cheap and it is difficult to limiting or avoiding them			
9. It is difficult to limit or avoid alcohol consumption			
10. It is hard for me to stop smoking			
11. I feel I don't have the time to engage in physical activity			
12. I don't have access to exercise equipment			

Appendix IV: Overall score of knowledge on risks preventive measures of CVDs

Scoring system of knowledge: respondents who gave at **least three** correct responses for each of the risk factors and preventive measures of CVDs, scored "**three**", those respondents who gave two correct responses, scored **"two"** and respondents who gave only one correct response, or incomplete answer or incorrect answer, scored "**one**". The total knowledge score was calculated by adding the scores for each response. The higher scores reflect higher levels of knowledge about CVDs. Then, the total score was expressed in percentages. Accordingly, score of < 50% was classified as low level of knowledge, between 50 and 74% as moderate level of knowledge score ≥ 75% as high level of knowledge.

Overall score of knowledge on modifiable risk factors of cardiovascular diseases (CVDs)

The following 7 elements were used to assess the overall score of knowledge on modifiable risk factors of CVDs. The scoring system was calculated as showing below.

- Knowledge on risk factors of hypertension and type 2 diabetes (≤ l correct response =1; 2 correct responses = 2; ≥ 3 correct responses = 3)
- CVDs related health consequences of tobacco smoking (≤ l correct response =1; 2 correct responses = 2; ≥ 3 correct responses = 3)
- CVDs related health consequences of excessive alcohol consumption (≤ l correct response =1; 2 correct responses = 2; ≥ 3 correct responses = 3)
- CVDs related health consequences of taking excessive salt (≤ l correct response =1; 2 correct responses = 2; ≥ 3 correct responses = 3)
- CVDs related health consequences of taking excessive sugar (≤ l correct response =1; 2 correct responses = 2; ≥ 3 correct responses = 3)
- CVDs related health consequences of eating junk foods (≤ l correct response =1; 2 correct responses = 2; ≥ 3 correct responses = 3)
- CVDs related health consequences of being overweight/obese (≤ l correct response =1; 2 correct responses = 2; ≥ 3 correct responses = 3)

The maximum attainable total score was 21 and minimum score was 7. A percentage score was generated and classified as low (<50%), moderate (50-74%), high (≥75%).

Overall score of knowledge on preventive measures of cardiovascular diseases (CVDs)

Knowledge of the respondents towards CVDs related preventive measures was assessed using 6 variables.

- knowledge on whether hypertension and diabetes are preventable (incorrect response = 1; correct response = 2)
- Knowledge on preventive measures of hypertension and diabetes (≤ 1 correct response =1; 2 correct responses = 2; ≥ 3 correct responses = 3)
- Knowledge on daily recommended amount of salt intake (incorrect response = 1; correct response = 2)
- Knowledge on daily recommended amount of sugar intake (incorrect response = 1; correct response = 2)
- Knowledge on importance of fruits and vegetables consumption towards CVDs prevention (≤ 1 correct response =1; 2 correct responses = 2; ≥ 3 correct responses = 3)
- Knowledge on benefits of engaging in physical activities towards CVDs prevention (≤ 1 correct response =1; 2 correct responses = 2; ≥ 3 correct responses = 3)

The maximum attainable total score was 15 and minimum score was 6. A percentage score was generated and classified as low (<50%), moderate (50-74%), high (≥75%).

Appendix V: The transtheoretical model (TTM) baseline and end-line results

Table16: Respondents' stage of changes towards a healthy lifestyle practice at baseline (n, %)

Group	Baseline TTM Stage of change construct			Total	Chi	df	p-value
	Pre-action	Action	Maintenance				
	Stage of fruits consumption						
Intervention	124 (79.5)	25 (16.0)	7 (4.5)	156 (100)	1.287	2	0.526
Control	112 (81.2)	17 (12.3)	9 (6.5)	138 (100)			
Total	236 (80.3)	42 (14.3)	16 (5.4)	294 (100)			
	Stage of vegetables consumption						
Intervention	128 (82.1)	22 (14.1)	6(3.8)	156 (100)	2.535	2	0.282
Control	111 (80.4)	16(11.6)	11(8.0)	138(100)			
Total	239 (81.3)	38(12.9)	17(5.8)	294(100)			
	Stage of DASH eating plan						
Intervention	130 (83.3)	22(14.1)	4(2.6)	156(100)	1.448		0.517**
Control	119 (86.2)	18(13.0)	1(0.7)	138(100)			
Total	249 (84.7)	40(13.6)	5 (1.7)	294 (100)			
	Stage of processed food consumption						
Intervention	106 (67.9)	8(5.1)	42 (26.9)	156 (100)	0.998	2	0.607
Control	90 (65.2)	11(8.0	37 (26.8)	138 (100)			
Total	196 (66.7)	19 (6.5)	79 (26.9)	294(100)			
	Stage of salt consumption						
Intervention	107 (68.6)	10 (6.4)	39 (25.0)	156 (100)	1.542	2	0.463
Control	88 (63.8)	14 (10.1)	36 (26.1)	138 (100)			
Total	195 (66.3)	24(8.2)	75(25.5)	294(100)			
	Stage of sugar consumption						
Intervention	99 (63.5)	18(11.5)	39(25.0)	156(100)	1.282	2	0.527
Control	85 (61.6)	22(15.9)	31(22.5)	138(100)			
Total	184 (62.6)	40 (13.6)	70 (23.8)	294(100)			
	Stage of alcohol consumption (n = 110)						
Intervention	34(57.6)	0(0.0)	25(42.4)	59(100)		1	
Control	29(56.9)	0(0.0)	22(43.1)	51(100)			
Total	63(57.3)	0(0.0)	47(42.7)	110(100)			
	Stage of tobacco smoking (n = 40)						
Intervention	7(33.3)	0(0.0)	14(66.7)	21(100)	2.976	1	0.133**
Control	2(10.5)	0(0.0)	17(89.5)	19(100)			
Total	9(22.5)	0(0.0)	31(77.5)	40(100)			
	Stage of physical activity						
Intervention	133 (85.3)	3(1.9)	20(12.8)	156 (100)	5.280	2	0.071*
Control	108 (78.3)	10(7.2)	20(14.5)	138 (100)			
Total	241 (82.0)	13(4.4)	40 (13.6)	294 (100)			
	Overall level of adherence to healthy lifestyle/behavioral practice						
	Low	Moderate	High				

Intervention	46(29.5)	107(68.6)	3(2.9)	156 (100)	1.568		0.456**
Control	32(23.2)	103(74.6)	3(2.2)	138 (100)			
Total	78(26.5)	210(71.4)	6(2.0)	294 (100)			
*Pearson Chi-Square, **Fisher's Exact Test*							

Table 17: Respondents' stage of changes towards a healthy lifestyle at the end-line (n, %)

Group	End-line TTM Stage of change construct			Total	Chi	df	p-value
	Pre-action	Action	Maintenance				
	Daily fruits consumption status						
Intervention	58(37.2)	29(18.6)	69(44.1)	156 (100)	22.227	2	0.000
Control	86(62.3)	24(17.4)	28(20.3)	138 (100)			
Total	144(49.0)	53(18.0)	97(33.0)	294 (100)			
	Daily vegetables consumption status						
Intervention	42(26.9)	4(2.6)	110(70.5)	156 (100)	14.290	2	0.001
Control	61(44.2)	9(6.5)	68(49.3)	138(100)			
Total	103(35.0)	13(4.4)	178(60.5)	294(100)			
	Use of the DASH eating plan status						
Intervention	43 (27.6)	46(29.5)	67(42.9)	156(100)	65.242	2	0.000
Control	103 (74.6)	12(8.7)	23(16.7)	138(100)			
Total	146 (49.7)	58(19.7)	90(30.6)	294 (100)			
	Processed food consumption status						
Intervention	48(30.8)	21(13.5)	87(55.8)	156 (100)	24.424	2	0.000
Control	81(58.7)	7(5.1)	50(36.2)	138 (100)			
Total	129(43.9)	28(9.5)	137(46.6)	294(100)			
	Salt consumption status						
Intervention	7(4.5)	40(25.6)	109(69.9)	156 (100)	22.280	2	0.000
Control	30(21.7)	39(28.3)	69(50.0)	138 (100)			
Total	37(12.6)	79(26.9)	178(60.5)	294(100)			
	Sugar consumption status						
Intervention	13(8.3)	42(26.9)	101(64.7)	156(100)	35.307	2	0.000
Control	51(37.0)	24(17.4)	63(45.7)	138(100)			
Total	64(21.8)	66(22.4)	164(55.8)	294(100)			
	Alcohol consumption status						
Intervention	16(27.1)	0(0.0)	43(72.9)	59(100)	2.422	1	0.120
Control	21(41.2)	0(0.0)	30(58.8)	51(100)			
Total	37(33.6)	0(0.0)	73(66.4)	110(100)			
	Tobacco smoking status						
Intervention	6(28.6)	0(0.0)	15(71.4)	21(100)	2.030[a]	1	0.241**
Control	2(10.5)	0(0.0)	17(89.5)	19(100)			
Total	8(20.0)	0(0.0)	32(80.0)	40(100)			
	Physical activity status						
Intervention	61(39.1)	10(6.4)	85(54.5)	156 (100)	18.416	2	0.000
Control	82(59.4)	15(10.9)	41(29.7)	138 (100)			
Total	143(48.6)	25(8.5)	126(42.9)	294 (100)			
	End-line overall level of adherence to a healthy lifestyle practice						
	Low	Moderate	High				
Intervention	2 (1.3)	47(30.1)	107(68.6)	156 (100)	56.290		0.000**
Control	5(3.6)	98(71.0)	35(25.4)	138 (100)			
Total	7(2.4)	145(49.3)	142 (48.3)	294 (100)			

**Pearson Chi-Square, **Fisher's Exact Test*

Table 18 Respondents' self-efficacy towards a healthy lifestyle before the intervention (n, %)

Groups	Baseline TTM self-efficacy construct			Total	Chi	df	p-value
	Not at all confident	Somewhat confident	Extremely confident				
	I Eat 5 or more servings of fruits a day						
Intervention	66 (42.3)	52(33.3)	38(24.4)	156(100)	3.905	2	0.142
Control	64 (46.4)	32 (23.2)	42(30.4)	138 (100)			
Total	130(44.2)	84(28.6)	80(27.2)	294(100)			
	I Eat 5 or more servings of vegetables a day						
Intervention	67 (42.9)	51(32.7)	38(24.4)	156(100)	3.811	2	0.149
Control	68 (49.3)	31(22.5)	39(28.3)	138 (100)			
Total	135 (45.9)	82(27.9)	77(26.2)	294(100)			
	I observe and adhere to the DASH eating plan						
Intervention	70(44.9)	50(32.1)	36(23.1)	156(100)	2.403	2	0.301
Control	70(50.7)	33(23.9)	35(25.4)	138 (100)			
Total	140(47.6)	83(28.2)	71(24.1)	294(100)			
	I avoid or limit eating processed/fast foods						
Intervention	59(37.8)	35(22.4)	62(39.7)	156(100)	2.185	2	0.335
Control	60(43.5)	22(15.9)	56(40.6)	138 (100)			
Total	119(40.5)	57(19.4)	118(40.1)	294(100)			
	I take ≤ 1 teaspoon of salt per day						
Intervention	92(59.0)	25(16.0)	39(25.0)	156(100)	1.144	2	0.564
Control	74(53.6)	28(20.3)	36(26.1)	138 (100)			
Total	166(56.5)	53(18.0)	75(25.5)	294(100)			
	I Take ≤ 5 teaspoons of sugar per day						
Intervention	63(40.4)	36(23.1)	57(36.5)	156(100)	2.878	2	0.237
Control	50(36.2)	44(31.9)	44(31.9)	138 (100)			
Total	113(38.4)	80(27.2)	101(34.4)	294(100)			
	I stop or moderate alcohol consumption						
Intervention	34(57.6)	0(0.0)	25(42.4)	59(100)	0.007	1	0.936
Control	29(56.9)	0(0.0)	22(43.1)	51(100)			
Total	63(57.3)	0(0.0)	47(42.7)	110(100)			
	I stop tobacco smoking						
Intervention	7(33.3)	0(0.0)	14(66.7)	21(100)	**2.976**	1	0.133**
Control	2(10.5)	0(0.0)	17(89.5)	19(100)			
Total	9(22.5)	0(0.0)	31(77.5)	40(100)			
	I do exercise for at least 30 minutes for 5-7 days per week						
Intervention	56(35.9)	34(21.8)	66(42.3)	156(100)	1.895	2	0.388
Control	40(29.0)	37(26.8)	61(44.2)	138 (100)			
Total	96(32.7)	71(24.1)	127(43.2)	294(100)			
**Fisher's Exact Test*							

Table 19 Respondents' self-efficacy towards a healthy lifestyle practice at the end-line (n, %)

Groups	End-line TTM self-efficacy construct			Total	Chi	df	p-value
	Not at all confident	Somewhat confident	Extremely confident				
	I eat 5 or more servings of fruits a day						
Intervention	58(37.2)	29(18.6)	69(44.2)	156(100)	22.227	2	0.000
Control	86(62.3)	24(17.4)	28(20.3)	138 (100)			
Total	144(49.0)	53(18.0)	97(33.0)	294(100)			
	I eat 5 or more servings of vegetables a day						
Intervention	39(25.0)	7(4.5)	110(70.5)	156(100)	14.036	2	0.001
Control	57(41.3)	13(9.4)	68(49.3)	138 (100)			
Total	96(32.7)	20(6.8)	178(60.5)	294(100)			
	I adherence to the DASH eating plan						
Intervention	43(27.6)	46(29.5)	67(42.9)	156(100)	37.700	2	0.000
Control	85(61.6)	30(21.7)	23(16.7)	138 (100)			
Total	128(43.5)	76(25.9)	90(30.6)	294(100)			
	I avoid or limit eating processed/fast foods						
Intervention	48(30.8)	21(13.5)	87(55.8)	156(100)	11.308	2	0.004
Control	63(45.7)	25(18.1)	50(36.2)	138 (100)			
Total	111(37.8)	46(15.6)	137(46.6)	294(100)			
	Take ≤ 1 teaspoon of salt per						
Intervention	7(4.5)	40(25.6)	109(69.9)	156(100)	22.280	2	0.000
Control	30(21.7)	39(28.3)	69(50.0)	138 (100)			
Total	37(12.6)	79(26.9)	178(60.5)	294(100)			
	I take ≤ 5 teaspoons of sugar per day						
Intervention	12(7.7)	42(26.9)	102(65.4)	156(100)	37.308	2	0.000
Control	51(37.0)	24(17.4)	63(45.7)	138 (100)			
Total	63(21.4)	66(22.4)	165(56.1)	294(100)			
	I stop/moderate alcohol consumption						
Intervention	16(27.1)	0(0.0)	43(72.9)	59(100)	2.422	1	0.120
Control	21(41.2)	0(0.0)	30(58.8)	51(100)			
Total	37(33.6)	0(0.0)	73(66.4)	110(100)			
	I stop tobacco smoking						
Intervention	6(28.6)	0(0.0)	15(71.4)	21(100)	2.030	1	0.241**
Control	2(10.5)	0(0.0)	17(89.5)	19(100)			
Total	8(20.0)	0(0.0)	32(80.0)	40(100)			
	I do exercise for at least 30 minutes for 5-7 days per week						
Intervention	61(39.1)	10(6.4)	85(54.5)	156(100)	18.416	2	0.000
Control	82(59.4)	15(10.9)	41(29.7)	138 (100)			
Total	143(48.6)	25(8.5)	126(42.9)	294(100)			

*Fisher's Exact Test

Table 20 Respondents' decisional balance (pros) to healthy lifestyle practice at baseline (n %)

Groups	Baseline TTM decisional balance construct(pros)			Total	Chi	df	p-value
	Disagree	Somewhat	Agree				
	I feel I am doing something good for my body if I eat more fruits and vegetables.						
Intervention	33(21.2)	43(27.6)	80(51.3)	156 (100)	1.057	2	0.589
Control	36(26.1)	34(24.6)	68(49.3)	138 (100)			
Total	69(23.5)	77(26.2)	148(50.3)	294 (100)			
	Fruits and vegetables are low in fat						
Intervention	12(7.7)	9(5.8)	135(86.5)	156 (100)	0.940	2	0.625
Control	9(6.5)	5(3.6)	124(89.9)	138 (100)			
Total	21(7.1)	14(4.8)	259(88.1)	294 (100)			
	Fruits and vegetables are a good substitute for junk food						
Intervention	63(40.4)	36(23.1)	57(36.5)	156 (100)	2.878	2	0.237
Control	50(36.2)	44(31.9)	44(31.9)	138 (100)			
Total	113(38.4)	80(27.2)	101(34.4)	294 (100)			
	I can find reasonably priced fruits and vegetables in my local markets						
	86(55.1)	32(20.5)	38(24.4)	156 (100)	4.327	2	0.115
	90(65.2)	27(19.6)	21(15.2)	138 (100)			
	176(59.9)	59(20.1)	59(20.1)	294 (100)			
	Eating fruits and vegetables could help me live a better, healthier, and longer life						
Intervention	30(19.2)	38(24.4)	88(56.4)	156 (100)	4.687	2	0.096
Control	41(29.7)	26(18.8)	71(51.4)	138 (100)			
Total	71(24.1)	64(21.8)	159(54.1)	294 (100)			
	Eating fruits and vegetables regularly could help me prevent diseases						
Intervention	33(21.2)	28(17.9)	95(60.9)	156 (100)	2.885	2	0.236
Control	41(29.7)	21(15.2)	76(55.1)	138 (100)			
Total	74(25.2)	49(16.7)	171(58.2)	294 (100)			
	Eating fruits and vegetables could help me lose or maintain my weight						
Intervention	60(38.5)	26(16.7)	70(44.9)	156 (100)	1.588	2	0.452
Control	61(44.2)	25(18.1)	52(37.7)	138 (100)			
Total	121(41.2)	51(17.3)	122(41.5)	294 (100)			
	Eating five or more servings of fruits and vegetables per day could help me prevent hypertension and diabetes.						
Intervention	23(14.7)	40(25.6)	93(59.6)	156 (100)	5.669	2	0.059
Control	5(18.1)	20(14.5)	93(67.4)	138 (100)			
Total	48(16.3)	60(20.4)	186(63.3)	294 (100)			
	I can find reasonably priced fruits and vegetables in my local markets						
Intervention	86(55.1)	32(20.5)	38(24.4)	156 (100)	4.327	2	0.115
Control	90(65.2)	27(19.6)	21(15.2)	138 (100)			

Total	176(59.9)	59(20.1)	59(20.1)	294 (100)			
	Limiting salt intake to 1 tea spoon or less per day could help me prevent hypertension and diabetes.						
Intervention	55(35.3)	32(20.5)	69(44.2)	156 (100)	4.477	2	0.107
Control	40(29.0)	43(31.2)	55(39.9)	138 (100)			
Total	95(32.3)	75(25.5)	124(42.2)	294 (100)			
	Limiting sugar intake to ≤ 5 tea spoons per day could help me prevent hypertension and diabetes.						
Intervention	43(27.6)	22(14.1)	91(58.3)	156 (100)	0.477	2	0.788
Control	42(30.4)	21(15.2)	75(54.3)	138 (100)			
Total	85(28.9)	43(14.6)	166(56.5)	294 (100)			
	Limiting or avoiding processed/fast foods could help me prevent hypertension and diabetes.						
Intervention	54(34.6)	28(17.9)	74(47.4)	156 (100)	4.498	2	0.106
Control	40(29.0)	41(28.3)	56(42.8)	138 (100)			
Total	94(32.0)	67(22.8)	133(45.2)	294 (100)			
	Limiting or avoiding alcohol consumption could help me prevent hypertension and diabetes.						
Intervention	51(32.7)	57(36.5)	48(30.8)	156 (100)	1.653	2	0.437
Control	55(39.9)	46(33.3)	37(26.8)	138 (100)			
Total	106(36.1)	103(35.0)	85(28.9)	294 (100)			
	Exercising for at least 30 minutes for 5-7 days per week						
Intervention	17(10.9)	62(39.7)	77(49.4)	156 (100)	5.476	2	0.065
Control	27(19.6)	42(30.4)	69(50.0)	138 (100)			
Total	44(15.0)	104(35.4)	146(49.7)	294 (100)			
	Exercising for at least 30 minutes for 5-7 days per week could help me lose or maintain my weight						
Intervention	38(24.4)	37(23.7)	81(51.9)	156 (100)	2.460	2	0.292
Control	45(32.6)	29(21.0)	64(46.4)	138 (100)			
Total	83(28.2)	66(22.4)	145(49.3)	294 (100)			
	Exercising for at least 30 minutes for 5-7 days per week could help me prevent hypertension and diabetes.						
Intervention	29(18.7)	53(34.2)	73(47.1)	156 (100)	1.779	2	0.411
Control	18(13.0)	49(35.5)	71(51.4)	138 (100)			
Total	47(16.0)	102(34.8)	144(49.1)	294 (100)			

Table 21 Respondents decisional balance (pros) to healthy lifestyle practice at endline (n, %)

Groups	End-line TTM decisional balance construct (pros)			Total	Chi	df	p-value
	Disagree	Somewhat	Agree				
	I feel I am doing something good for my body if I eat more fruits and vegetables						
Intervention	30(19.2)	25(16.0)	101(64.7)	156 (100)	7.336	2	0.026
Control	32(23.2)	37(26.8)	69 (50.0)	138 (100)			
Total	62(21.1)	62(21.1)	170 (57.8)	294 (100)			
	Fruits and vegetables are low in fat						
Intervention	5(3.2)	1(0.6)	150(96.2)	156 (100)	3.327		0.184*
Control	8(5.8)	4(2.9)	126(91.3)	138 (100)			
Total	13(4.4)	5(1.7)	276(93.9)	294 (100)			
	Eating fruits and vegetables could help me live a better, healthier, and longer life						
Intervention	22(14.1)	31(19.9)	103(66.0)	156 (100)	5.257	2	0.072
Control	33(23.9)	29(21.0)	76(55.1)	138 (100)			
Total	55(18.7)	60(20.4)	179(60.9)	294 (100)			
	Eating fruits and vegetables regularly could help me prevent diseases						
Intervention	10(6.4)	33(21.2)	113(72.4)	156 (100)	7.863	2	0.020
Control	19(13.8)	39(28.3)	80(58.0)	138 (100)			
Total	29(9.9)	72(24.5)	193(65.6)	294 (100)			
	Eating fruits and vegetables could help me lose or maintain my weight						
Intervention	19(12.2)	21(13.5)	116(74.4)	156 (100)	21.410	2	0.000
Control	24(17.4)	46(33.3)	68(49.3)	138 (100)			
Total	43(14.6)	67(22.8)	184(62.6)	294 (100)			
	Fruits and vegetables are a good substitute for junk food						
Intervention	15(9.6)	27(17.3)	114(73.1)	156 (100)	15.748	2	0.000
Control	22(15.9)	46(33.3)	70(50.7)	138 (100)			
Total	37(12.6)	73(24.8)	184(62.6)	294 (100)			
	Eating five or more servings of fruits and vegetables per day could help me prevent hypertension and diabetes.						
Intervention	10(6.4)	24(15.4)	122(78.2)	156 (100)	14.113	2	0.001
Control	25(18.1)	31(22.5)	82(59.4)	138 (100)			
Total	35(11.9)	55(18.7)	204(69.4)	294 (100)			
	I can find reasonably priced fruits and vegetables in my local markets						
Intervention	69(44.2)	38(24.4)	49(31.4)	156 (100)	4.757	2	0.093
Control	69(50.0)	41(29.7)	28(20.3)	138 (100)			
Total	138(46.9)	79(26.9)	77(26.2)	294 (100)			

	Limiting salt intake to ≤1 tea spoon per day could help me prevent hypertension and diabetes.						
Intervention	19(12.2)	17(10.9)	120(76.9)	156 (100)	20.695	2	0.000
Control	22(15.9)	42(30.4)	74(53.6)	138 (100)			
Total	41(13.9)	59(20.1)	194(66.0)	294 (100)			
	Limiting sugar intake to 5 tea spoons or less per day could help me prevent hypertension and diabetes.						
Intervention	7(4.5)	28(17.9)	121(77.6)	156 (100)	10.395	2	0.006
Control	17(12.3)	36(26.1)	85(61.6)	138 (100)			
Total	24(8.2)	64(21.8)	206(70.1)	294 (100)			
	Limiting/avoiding processed/fast foods could help me prevent hypertension and diabetes.						
Intervention	19(12.2)	18(11.5)	119(76.3)	156 (100)	12.264	2	0.002
Control	21(15.2)	36(26.1)	81(58.7)	138 (100)			
Total	40(13.6)	54(18.4)	200(68.0)	294 (100)			
	Limiting/avoiding alcohol consumption could help me prevent hypertension and diabetes						
Intervention	6(3.8)	27(17.3)	123(78.8)	156 (100)	18.057	2	0.000
Control	9(6.5)	52(37.7)	77(55.8)	138 (100)			
Total	15(5.1)	79(26.9)	200(68.0)	294 (100)			
	Exercising for at least 30 minutes for 5-7 days per week						
Intervention	7(4.5)	39(25.0)	110(70.5)	156 (100)	9.793	2	0.007
Control	18(13.0)	43(31.2)	77(55.8)	138 (100)			
Total	25(8.5)	82(27.9)	187(63.6)	294 (100)			
	Exercising for at least 30 minutes for 5-7 days per week could help me lose or maintain my weight						
Intervention	16(10.3)	21(13.5)	119(76.3)	156 (100)	10.754	2	0.005
Control	16(11.6)	39(28.3)	83(60.1)	138 (100)			
Total	32(10.9)	60(20.4)	202(68.7)	294 (100)			
	Exercising for at least 30 minutes for 5-7 days per week could help me prevent hypertension and diabetes.						
Intervention	10(6.4)	27(17.3)	119(76.3)	156 (100)	14.871	2	0.001
Control	11(8.0)	50(36.2)	77(55.8)	138 (100)			
Total	21(7.1)	77(26.2)	196(66.7)	294 (100)			
*Fisher's Exact Test							

Table 22 Respondents' decisional balance (cons) to healthy lifestyle practice at baseline (n, %)

Groups	Baseline TTM decisional balance construct (cons)			Total	Chi	df	p-value
	Disagree	Somewhat	Agree				
	Fruits and vegetables are too expensive to buy						
Intervention	32(20.5)	35(22.4)	89(57.1)	156 (100)	3.436	2	0.179
Control	23(16.7)	22(15.9)	93(67.4)	138 (100)			
Total	55(18.7)	57(19.4)	182(61.9)	294 (100)			
	It is too difficult to eat five or more servings of fruits and vegetables each day						
Intervention	42(26.9)	32(20.5)	82(52.6)	156 (100)	5.072	2	0.079
Control	42(30.4)	15(10.9)	81(58.7)	138 (100)			
Total	84(28.6)	47(16.0)	163(55.4)	294 (100)			
	I worry about the safety of chemicals used in fruits and vegetables.						
Intervention	36(23.1)	75(48.1)	45(28.8)	156 (100)	4.934	2	0.085
Control	18(13.0)	74(53.6)	46(33.3)	138 (100)			
Total	54(18.4)	149(50.7)	91(31.0)	294 (100)			
	I have limited ways to incorporate fruits and vegetables in my daily meals.						
Intervention	49(31.4)	39(25.0)	68(43.6)	156 (100)	1.285	2	0.526
Control	45(32.6)	27(19.6)	66(47.8)	138 (100)			
Total	94(32.0)	66(22.4)	134(45.6)	294 (100)			
	It is difficult to control daily salt and sugar intake						
Intervention	40(25.6)	58(37.2)	58(37.2)	156 (100)	0.859	2	0.651
Control	42(30.4)	47(34.1)	49(35.5)	138 (100)			
Total	82(27.9)	105(35.7)	107(36.4)	294 (100)			
	Food with less or no salt or sugar are tasteless						
Intervention	21(13.5)	55(35.3)	80(51.3)	156 (100)	5.336	2	0.069
Control	33(23.9)	43(31.2)	62(44.9)	138 (100)			
Total	54(18.4)	98(33.3)	142(48.3)	294 (100)			
	Processed/fast foods are much available and thus difficult to avoid or limit them						
Intervention	41(26.3)	37(23.7)	78(50.0)	156 (100)	2.273	2	0.321
Control	31(22.5)	26(18.8)	81(58.7)	138 (100)			
Total	72(24.5)	63(21.4)	159(54.1)	294 (100)			
	Processed/fast foods are cheaper than natural foods						
Intervention	59(37.8)	61(39.1)	36(23.1)	156 (100)	3.457	2	0.178
Control	48(34.8)	45(32.6)	45(32.6)	138 (100)			
Total	107(36.4)	106(36.1)	81(27.6)	294 (100)			
	It is/was difficult for me to limit/avoid alcohol consumption						
Intervention	25(42.4)	0(0.0)	34(57.6)	59(100)	0.007	1	0.936
Control	22(43.1)	0(0.0)	29(56.9)	51(100)			
Total	47(42.7)	0(0.0)	63(57.3)	110(100)			
	It is/was hard for me to stop smoking						
Intervention	9(42.9)	5(23.8)	7(33.3)	19(100.0)	2.982		0.250*
Control	10(52.6)	7(36.8)	2(10.5)	21(100.0)			
Total	19(47.5)	12(30.0)	9(22.5)	40(100.0)			

	I feel I don't have the time to do physical activity on 5-7 days per week						
Intervention	58(37.2)	49(31.4)	49(31.4)	156 (100)	3.538	2	0.170
Control	50(36.2)	32(23.2)	56(40.6)	138 (100)			
Total	108(36.7)	81(27.6)	105(35.7)	294 (100)			
*Fisher's Exact Test							

Table 23 Respondents decisional balance (cons) to healthy lifestyle practice at the end-line (n, %)

	End-line TTM decisional balance construct (cons)			Total	Chi	df	p-value
Groups	Disagree	Somewhat	Agree				
Groups	Fruits and vegetables are too expensive to buy						
Intervention	33(21.2)	38(24.4)	85(54.5)	156 (100)	3.246	2	0.197
Control	34(24.6)	22(15.9)	82(59.4)	138 (100)			
Total	67(22.8)	60(20.4)	167(56.8)	294 (100)			
	It is too difficult to eat five or more servings of fruits and vegetables each day						
Intervention	50(32.1)	25(16.0)	81(51.9)	156 (100)	5.953	2	0.051
Control	32(23.2)	15(10.9)	91(65.9)	138 (100)			
Total	82(27.9)	40(13.6)	172(58.5)	294 (100)			
	I worry about the safety of chemicals used in fruits and vegetables.						
Intervention	37(23.7)	65(41.7)	54(34.6)	156 (100)	0.474	2	0.789
Control	30(21.7)	55(39.9)	53(38.4)	138 (100)			
Total	67(22.8)	120(40.8)	107(36.4)	294 (100)			
	I have limited ways to incorporate fruits and vegetables in my meals.						
Intervention	72 (46.2)	35(22.4)	49(31.4)	156 (100)	6.558	2	0.038
Control	46(33.3)	30(21.7)	62(44.9)	138 (100)			
Total	118(40.1)	65(22.1)	111(37.8)	294 (100)			
	It is difficult to control daily salt and sugar intake						
Intervention	100 (64.1)	17(10.9)	39(25.0)	156 (100)	15.722	2	0.000
Control	57(41.3)	29(21.0)	52(37.7)	138 (100)			
Total	157(53.4)	46(15.6)	91(31.0)	294 (100)			
	Food with less or no salt or sugar are tasteless						
Intervention	61(39.1)	36(23.1)	59(37.8)	156 (100)	12.319	2	0.002
Control	29(21.0)	49(35.5)	60(43.5)	138 (100)			
Total	90(30.6)	85(28.9)	119(40.5)	294 (100)			
	Processed/fast foods are much available and it is difficult to limiting or avoiding them						
Intervention	71(45.5)	29(18.6)	56(35.9)	156 (100)	13.432	2	0.001
Control	35(25.4)	30(21.7)	73(52.9)	138 (100)			
Total	106(36.1)	59(20.1)	129(43.9)	294 (100)			
	Processed/fast foods are cheaper than natural foods						
Intervention	78(50.0)	43(27.6)	35(22.4)	156 (100)	3.115	2	0.211
Control	55(39.9)	44(31.9)	39(28.3)	138 (100)			
Total	133(45.2)	87(29.6)	74(25.2)	294 (100)			
	It is difficult to limit or avoid alcohol consumption						
Intervention	43(72.9)	0(0.0)	16(27.1)	59(100)	2.422	1	0.120

Control	30(58.8)	0(0.0)	21(41.2)	51(100)			
Total	73(66.4)	0(0.0)	37(33.6)	110(100)			
	It is hard for me to stop smoking						
Intervention	15(71.4)	0(0.0)	6(28.6)	21(100)	2.030[a]	1	0.241**
Control	17(89.5)	0(0.0)	2(10.5)	19(100)			
Total	32(80.0)	0(0.0)	8(20.0)	40(100)			
	I feel I don’t have the time to do physical activity on 5-7 days per week						
Intervention	100(64.1)	41(26.3)	15(9.6)	156 (100)	8.055	2	0.018
Control	69(50.0)	42(30.4)	27(19.6)	138 (100)			
Total	169(57.5)	83(28.2)	42(14.3)	294 (100)			
*Fisher's Exact Test							

Appendix VI: List of publications

1. **Okube, O.T., Kimani, S. & Mirie, W. (2022).** Community-based lifestyle intervention improves metabolic syndrome and related markers among Kenyan adults. J Diabetes Metab Disord. https://doi.org/10.1007/s40200-022-01023-1.

2. **Okube OT, Kimani S, Waithira M. (2020).** Association of dietary patterns and practices on metabolic syndrome in adults with central obesity attending a mission hospital in Kenya: a cross-sectional study. BMJ Open. 10(10):e039131. doi: 10.1136/bmjopen-2020-039131. PMID: 33046471; PMCID: PMC7552860.

3. **Okube OT, Kimani ST, Mirie W. (2020).** Gender Differences in the Pattern of Socio-Demographics Relevant to Metabolic Syndrome Among Kenyan Adults with Central Obesity at a Mission Hospital in Nairobi, Kenya. High Blood Press Cardiovasc Prev.(1):61-82. doi: 10.1007/s40292-020-00360-7. Epub 2020 Jan 25. PMID: 31981085.